

DRAFT to OMB:

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CHAPTER 9: Cost-Benefit Analysis

This chapter reports EPA's analysis of the public health and welfare impacts and associated monetized benefits to society of the proposed Nonroad Diesel Engines Standards. EPA is required by Executive Order 12866 to estimate the benefits of major new pollution control regulations. Accordingly, the analysis presented here attempts to answer three questions: 1) what are the physical health and welfare effects of changes in ambient air quality resulting from reductions in nitrogen oxides (NO_x), sulfur dioxide (SO₂), non-methane hydrocarbons (NMHC), carbon monoxide (CO) and direct diesel particulate matter (PM) emissions?; 2) how much are the changes in these effects worth to U.S. citizens as a whole in monetary terms?; and 3) how do the monetized benefits compare to the costs over time? It constitutes one part of EPA's thorough examination of the relative merits of this proposed regulation. In Chapter 12, we provide an analysis of the benefits of several alternatives to the proposed standards to examine their relative benefits and costs.

Due to the time requirements for running the sophisticated emissions and air quality models needed to obtain estimates of the changes in air quality expected to result from implementation of emission controls, it is often necessary to select a set of preliminary control options for the purposes of emissions and air quality modeling. The standards we are proposing in this rulemaking are slightly different in the amount of emission reductions expected to be achieved in 2020 and 2030 relative to the preliminary control options that we modeled. EPA has used the best available information and tools of analysis to quantify the expected changes in public health, environmental and economic benefits of the preliminary control options, and these are presented in Appendix 9A, directly following this chapter. However, we determined that additional analysis was necessary to reflect the differences in emission reductions between the modeled and proposed standards. The results of that additional analysis are the focus of this chapter.

In order to characterize the benefits attributable to the proposed Nonroad Diesel Engines standards, given the constraints on time and resources available for the analysis, we use a benefits transfer method to scale the benefits of the modeled preliminary control options to reflect the differences in emission reductions. We also apply intertemporal scaling factors to examine the stream of benefits over the rule implementation period. The benefits transfer method used to estimate benefits for the proposed standards is similar to that used to estimate benefits in the recent analysis of the Large SI/Recreational Vehicles standards (see RIA, Docket A-2000-01). A similar method has also been used in recent benefits analyses for the proposed Industrial Boilers and Process Heaters MACT standards and the Reciprocating Internal Combustion Engines MACT standards. One significant limitation to this method is the inability to scale ozone-related benefits. Because ozone is a homogeneous gaseous pollutant formed through complex atmospheric photochemical processes, it is not possible to apportion ozone benefits to the precursor emissions of NO_x and VOC. Coupled with the potential for NO_x reductions to either increase or decrease ambient ozone levels, this prevents us from scaling the benefits associated with a particular combination of VOC and NO_x emissions reductions to another. A more detailed discussion is provided below. Because of our inability to scale ozone benefits, we provide the ozone benefits results for the modeled preliminary control options as a referent, but do not include ozone benefits as part of the monetized benefits of the proposed standards.

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For the most part, quantifiable ozone benefits do not contribute significantly to the monetized benefits. Thus, their omission will not materially affect the conclusions of the benefits analysis.

Table 9-1 lists the known quantifiable and unquantifiable effects considered for this analysis. It is important to note that there are significant categories of benefits which can not be monetized (or in many cases even quantified), resulting in a significant limitation to this analysis. Also, EPA currently does not have appropriate tools for modeling changes in ambient concentrations of CO or air toxics for input into a national benefits analysis. They have been linked to numerous health effects; however, we are unable to quantify the CO- or air toxics-related health or welfare benefits of the Nonroad Diesel Engine rule at this time.

The benefit analysis that we performed for our proposed rule can be thought of as having seven parts, each of which will be discussed separately in the Sections that follow. These seven steps are:

1. Identification of proposed standards and calculation of the impact that the proposed standards will have on the nationwide inventories for NO_x, non-methane hydrocarbons (NMHC), SO₂, and PM emissions throughout the rule implementation period;
2. Calculation of scaling factors relating emissions changes resulting from the proposed standards to emissions changes from a set of preliminary control options that were used to develop modeled air quality and benefits (see Appendix 9A for full details).
3. Apportionment of modeled benefits of preliminary control options to NO_x, SO₂, and diesel PM emissions (see Appendix 9A for a complete discussion of the modeling of the benefits for the preliminary set of standards).
4. Application of scaling factors to apportioned modeled benefits associated with NO_x, SO₂, and PM in 2020 and 2030.
5. Development of intertemporal scaling factors based on 2020 and 2030 modeled air quality and benefits results.
6. Application of intertemporal scaling factors to the yearly emission changes expected to result from the proposed standards from 2010 through 2030 to obtain yearly monetized benefits.
7. Calculation of present value of stream of benefits.

This analysis presents estimates of the potential benefits from the proposed Nonroad Diesel Engine rule occurring in future years. The predicted emissions reductions that will result from the rule have yet to occur, and therefore the actual changes in human health and welfare outcomes to which economic values are ascribed are predictions. These predictions are based on the best available scientific evidence and judgment, but there is unavoidable uncertainty associated with each step in the complex process between regulation and specific health and welfare outcomes. Uncertainties associated with projecting input and parameter values into the future may contribute significantly to the overall uncertainty in the benefits estimates. However, we make these projections to more completely examine the impact of the program as the equipment fleet turns over.

In general, the chapter is organized around the steps laid out above. In section 1, we identify the potential standard to analyze, establish the timeframe of the analysis, and summarize emissions impacts. In section 2, we summarize the changes in emissions that were used in the preliminary modeled benefits analysis and develop ratios of proposed to preliminary emissions that are used to scale modeled benefits. In section 3, we summarize the modeled benefits associated with the emissions changes for the preliminary control options and apportion those benefits to the individual emission species (NO_x, SO₂, and PM). In Section 4, we estimate the benefits in 2020 and 2030 for the proposed standards, based on scaling of the modeled benefits of the preliminary control options. In section 5, we develop intertemporal scaling factors based on the ratios of yearly emission changes to the emission changes in 2020 and 2030 and estimate yearly benefits of the proposed standards, based on scaling of the benefits in 2020 and 2030. Finally, in Section 6, we compare the estimated streams of benefits and costs over the full implementation period, 2007 to 2030, to calculate the present value of net benefits for the proposed standards.

**Table 9-1
Health and Welfare Effects of Pollutants Affected by the Proposed Nonroad Diesel Engine Rule**

Pollutant/Effect	Quantified and Monetized in Base and Alternative Estimates	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
PM/Health	Premature mortality – long 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	Premature mortality – short term exposures Asthma attacks (asthmatic population) 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)
PM/Welfare	Visibility in California, Southwestern, and Southeastern Class I areas	Visibility in Northeastern, Northwestern, and Midwestern Class I areas Visibility in residential and non-Class I areas Household soiling	

Pollutant/Effect	Quantified and Monetized in Base and Alternative Estimates	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
Ozone/Health			<p>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 Hospital admissions - respiratory Emergency room visits for asthma Minor restricted activity days School loss days Chronic Asthma^a Asthma attacks Cardiovascular emergency room visits Premature mortality – acute exposures^b Acute respiratory symptoms</p>
Ozone/Welfare			<p>Decreased commercial forest productivity Decreased yields for fruits and vegetables Decreased yields for commercial and non-commercial crops Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Damage to ecosystem functions Decreased outdoor worker productivity</p>

Pollutant/Effect	Quantified and Monetized in Base and Alternative Estimates	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
Nitrogen and Sulfate Deposition/ Welfare		Costs of nitrogen controls to reduce eutrophication in selected eastern estuaries	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
SO ₂ /Health			Hospital admissions for respiratory and cardiac diseases Respiratory symptoms in asthmatics
NO _x /Health			Lung irritation Lowered resistance to respiratory infection Hospital Admissions for respiratory and cardiac diseases
CO/Health			Premature mortality Behavioral effects Hospital admissions - respiratory, cardiovascular, and other Other cardiovascular effects Developmental effects Decreased time to onset of angina Non-asthma respiratory ER visits

Pollutant/Effect	Quantified and Monetized in Base and Alternative Estimates	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
NMHCs ^c Health			Cancer (diesel PM, benzene, 1,3-butadiene, formaldehyde, acetaldehyde) Anemia (benzene) Disruption of production of blood components (benzene) Reduction in the number of blood platelets (benzene) Excessive bone marrow formation (benzene) Depression of lymphocyte counts (benzene) Reproductive and developmental effects (1,3-butadiene) Irritation of eyes and mucous membranes (formaldehyde) Respiratory and respiratory tract Asthma attacks in asthmatics (formaldehyde) Asthma-like symptoms in non-asthmatics (formaldehyde) Irritation of the eyes, skin, and respiratory tract (acetaldehyde) Upper respiratory tract irritation & congestion (acrolein)
NMHCs ^c Welfare			Direct toxic effects to animals Bioaccumulation in the food chain Reduced odors

^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).

^b Premature mortality associated with ozone is not separately included in the calculation of total monetized benefits. It is assumed that the American Cancer Society (ACS)/ Krewski, et al., 2000 C-R function we use for premature mortality captures both PM mortality benefits and any mortality benefits associated with other air pollutants (ACS/ Krewski, et al., 2000).

^c All non-methane hydrocarbons (NMHCs) listed in the table are also hazardous air pollutants listed in the Clean Air Act.

9.1 Time Path of Emission Changes for the Proposed Standards

The proposed standards have various cost and emission related components, as described earlier in this RIA. These components would begin at various times and in some cases would phase in over time. This means that during the early years of the program there would not be a consistent match between cost and benefits. This is especially true for the equipment control portions and initial fuel changes required by the program, where the full equipment cost would be incurred at the time of equipment purchase, while the fuel and maintenance costs, along with the emission reductions and benefits resulting from all these costs would occur throughout the lifetime of the equipment. Because of this inconsistency and our desire to more appropriately match the costs and emission reductions of our program, our analysis examines costs and benefits throughout the period of program implementation. This chapter focuses on estimating the stream of benefits over time and comparing streams of benefits and costs. Detailed information on cost estimates can be found in chapters 6, 7 and 8 of this RIA.

For the proposed standards, implementation will occur in two stages: reduction in sulfur content of nonroad diesel fuel and adoption of controls on new engines. Because full turnover of the fleet of nonroad diesel engines will not occur for many years, the emission reduction benefits of the proposed standards will not be fully realized until several decades after the reduction in fuel sulfur content. The timeframe for the analysis reflects this turnover, beginning in 2010 and extended through 2030.

Chapter 3 discussed the development of the 1996, 2020 and 2030 baseline emissions inventories for the nonroad sector and for the sectors not affected by this proposed rule. The emission sources and the basis for current and future-year inventories are listed in Table 9-2. Using these modeled inventories, emissions with and without the proposed regulations are interpolated to provide streams of emissions from the rule implementation date through full implementation in 2030. These streams of emissions are presented in Chapter 3 and summarized in Table 9-3 for the species that form the inputs to the benefits modeling. NO_x and VOC contribute to ambient ozone formation, while NO_x, SO₂, NMHC/VOC, and directly emitted PM_{2.5} emissions are precursors to ambient PM_{2.5} and PM₁₀ concentrations. Although the rule is expected to reduced CO and air toxics emissions as well, we do not include benefits related to these reductions in the benefits analysis due to a lack of appropriate air quality and exposure models.

Table 9-2
Emissions Sources and Basis for Current and Future-Year Inventories

Emissions Source	1996 Base year	Future-year Base Case Projections
Utilities	1996 NEI Version 3.12 (CEM data)	Integrated Planning Model (IPM)
Non-Utility Point and Area sources	1996 NEI Version 3.12 (point) Version 3.11 (area)	BEA growth projections
Highway vehicles	MOBILE5b model with MOBILE6 adjustment factors for VOC and NOx; PART5 model for PM	VMT projection data
Nonroad engines (except locomotives, commercial marine vessels, and aircraft)	NONROAD2002 model	BEA and Nonroad equipment growth projections

Note: Full description of data, models, and methods applied for emissions inventory development and modeling are provided in the Emissions Inventory TSD (U.S. EPA, 2003a).

Table 9-3.
Summary of 48-State Baseline Emissions for Nonroad Diesel Engines for Key Emission Species^A

	Annual Tons			
	NO _x	SO ₂	VOC	PM _{2.5}
2000	1,591,801	243,333	191,136	218,311
2005	1,509,081	273,331	155,943	194,554
2010	1,319,917	288,617	122,996	179,213
2015	1,199,235	315,367	101,641	178,559
2020	1,175,544	341,941	93,241	183,250
2025	1,211,002	369,475	91,709	191,976
2030	1,273,245	397,109	93,899	201,567

^A Excludes Alaska and Hawaii

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Table 9-4 summarizes the expected changes in emissions of key species. SO₂ emissions are expected to be reduced by over 90 percent within the first two years of implementation. Emissions of NO_x, NMHC, and PM_{2.5} are expected to be reduced gradually over the period of implementation from 2007 to 2030. Overall, NO_x, SO₂, NMHC, and PM_{2.5} emissions are expected to decline by 65, 97, 30, and 63 percent, respectively, over the 2007 to 2030 implementation period.

Table 9-4.
Summary of Reduction in 48-State Emissions Attributable to Proposed Nonroad Diesel Engine Standards

	Tons Reduced (% of baseline)			
	NO _x	SO ₂	VOC	PM _{2.5}
2010	1,007 (0.1%)	270,977 (93.9%)	90 (0.1%)	21,864 (12.2%)
2015	217,575 (18.1%)	305,639 (96.9%)	8,788 (8.6%)	52,476 (29.4%)
2020	503,701 (42.8%)	331,840 (97.0%)	18,033 (19.3%)	85,254 (46.5%)
2025	693,857 (57.3%)	358,863 (97.1%)	24,624 (26.9%)	109,325 (56.9%)
2030	821,911 (64.6%)	385,932 (97.2%)	29,487 (31.4%)	126,910 (63.0%)

9.2 Development of Benefits Scaling Factors Based on Differences in Emission Impacts Between Proposed and Modeled Preliminary Control Options

Based on the projected time paths for emissions reductions, we focused our detailed emissions and air quality modeling on two future years, 2020 and 2030, which reflect partial and close to complete turnover of the fleet of nonroad diesel engines to rule compliant models. The emissions changes modeled for these two years are similar to those in the proposed standards, differing in the

treatment of smaller engines and fuel requirements^A. Table 9-5 summarizes the reductions in emissions of NO_x, SO₂, and PM_{2.5} from baseline for the preliminary and proposed standards, the difference between the two, and the ratio of emissions reductions from the proposed standards to the preliminary control options. The ratios presented in the last column of Table 9-5 are the basis for the benefits scaling approach discussed below.

**Table 9-5.
Comparison of 48-state Emission Reductions in 2020 and 2030 Between Preliminary and Proposed Standards**

Emissions Species	Reduction from Baseline		Difference in Reductions (Proposed-Preliminary)	Ratio of Reductions (Proposed/Preliminary)
	Preliminary	Proposed		
2020				
NO _x	663,618	503,701	-159,917	0.759
SO ₂	414,692	331,840	-82,852	0.800
PM _{2.5}	98,121	85,254	-12,867	0.869
2030				
NO _x	1,009,744	821,911	-187,833	0.814
SO ₂	483,401	385,932	-97,469	0.798
PM _{2.5}	138,208	126,910	-11,298	0.918

9.3 Summary of Modeled Benefits and Apportionment Method

Based on the emissions inventories developed for the preliminary control option, we conducted a benefits analysis to determine the air quality and associated human health and welfare benefits resulting from the reductions in emissions of NO_x, SO₂, NMHC/VOC, and PM_{2.5}. Based on the availability of air quality and exposure models, this summary focuses on reporting the health and welfare benefits of reductions in ambient particulate matter (PM) and ozone concentrations. However, health improvements may also come from modest reductions in exposure to CO and air toxics. The

^AEmissions and air quality modeling decisions are made early in EPA’s analytical process. Since the preliminary control scenario was developed, EPA has gathered more information regarding the technical feasibility of the standards, and has revised the control scenario. For the reasons discussed in the preamble, EPA has decided not to propose standards based on aftertreatment for certain of the smallest engine sizes. Section 3.6 of the RIA describes the changes in the inputs and resulting emission inventories between the preliminary baseline and control scenarios used for the air quality modeling and the proposed baseline and control scenarios.

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full analysis is available in Appendix 9A and the benefits Technical Support Document (TSD) (Abt Associates, 2003).

The reductions in emissions of NO_x, SO₂, and PM from nonroad engines in the United States are expected to result in wide-spread overall reductions in ambient concentrations of ozone and PM_{2.5}^B. These improvements in air quality are expected to result in substantial health benefits, based on the body of epidemiological evidence linking PM and ozone with health effects such as premature mortality, chronic lung disease, hospital admissions, and acute respiratory symptoms. Based on modeled changes in ambient concentrations of PM_{2.5} and ozone, we estimate changes in the incidence of each health effect using concentration-response (C-R) functions derived from the epidemiological literature with appropriate baseline populations and incidence rates. We then apply estimates of the dollar value of each health effect to obtain a monetary estimate of the total PM- and ozone-related health benefits of the rule. Welfare effects are estimated using economic models which link changes in physical damages (e.g., light extinction or agricultural yields) with economic values.

9.3.1 Overview of Analytical Approach

This section summarizes the three steps involved in our analysis of the modeled preliminary control options: 1) Calculation of the impact that a set of preliminary fuel and engine standards would have on the nationwide inventories for NO_x, NMHC, SO₂, and PM emissions in 2020 and 2030; 2) Air quality modeling for 2020 and 2030 to determine changes in ambient concentrations of ozone and particulate matter, reflecting baseline and post-control emissions inventories; and 3) A benefits analysis to determine the changes in human health and welfare, both in terms of physical effects and monetary value, that result from the projected changes in ambient concentrations of various pollutants for the modeled standards.

We follow a “damage-function” approach in calculating total benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare endpoints (specific effects that can be associated with changes in air quality) and assigns values to those changes assuming independence of the individual values. Total benefits are calculated simply as the sum of the values for all non-overlapping health and welfare endpoints. This imposes no overall preference structure, and does not account for potential income or substitution effects, i.e. adding a new endpoint will not reduce the value of changes in other endpoints. The “damage-function” approach is the standard approach for most cost-benefit analyses of regulations affecting environmental quality, and it has been used in several recent published analyses (Banzhaf et al., 2002; Levy et al, 2001; Kunzli et al, 2000; Levy et al, 1999; Ostro and Chestnut, 1998). Time and resource constraints prevented us from

^B Reductions in NO_x are expected to result in some localized increases in ozone concentrations, especially in NO_x-limited large urban areas, such as Los Angeles, New York, and Chicago. A fuller discussion of this phenomenon is provided in Chapter 2.3. While localized increases in ozone will result in some increases in health impacts from ozone exposure in these areas, on net, the reductions in NO_x are expected to reduce national levels of health impacts associated with ozone.

performing extensive new research to measure either the health outcomes or their values for this analysis. Thus, similar to these studies, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary research from similar contexts to obtain the most accurate measure of benefits available for the environmental quality change under analysis.

There are significant categories of benefits that can not be monetized (or in many cases even quantified), and thus they are not included in our accounting of health and welfare benefits. These unquantified effects include infant mortality, low birth weight, changes in pulmonary function, chronic respiratory diseases other than chronic bronchitis, morphological changes, altered host defense mechanisms, non-fatal cancers, and non-asthma respiratory emergency room visits. A complete discussion of PM related health effects can be found in the PM Criteria Document (U.S. EPA, 1996). Since many health effects overlap, such as minor restricted activity days and asthma symptoms, we made assumptions intended to reduce the chances of “double-counting” health benefits, which may have lead to an underestimate of the total health benefits of the pollution controls.

9.3.2 Air Quality Modeling

We used a national-scale version of the REgional Modeling System for Aerosols and Deposition (REMSAD version 7) to estimate PM air quality in the contiguous United States. We used the Comprehensive Air Quality Model with Extensions (CAMx) to estimate ambient ozone concentrations^C, using two domains representing the Eastern and Western U.S. These models are discussed in the air quality TSD for this rule.

9.3.2.1 PM Air Quality Modeling with REMSAD

REMSAD is appropriate for evaluating the impacts of emissions reductions from nonroad sources, because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions. The annual county level emission inventory data described in Chapter 3 was speciated, temporally allocated and gridded to the REMSAD modeling domain to simulate PM concentrations for the 1996 base year and the 2020 and 2030 base and control scenarios. Peer-reviewed for the EPA, REMSAD is a three-dimensional grid-based Eulerian air quality model designed to estimate annual particulate concentrations and deposition over large spatial scales (Seigneur et al., 1999). Each of the future scenarios was simulated using 1996 meteorological data to provide daily averages and annual mean PM concentrations required for input to the concentration-response functions of the benefits analysis. Details regarding the application of REMSAD Version 7 for this analysis are provided in the

^CIn the benefits analysis of the recent Heavy Duty Engine/Diesel Fuel rule, we used the Urban Airshed Model Variable-Grid (UAM-V) to estimate ozone concentrations in the Eastern U.S. CAMx has a number of improvements relative to UAM and has improved model performance in the Western U.S. Details on the performance of CAMx can be found in Chapter 2 as well as the Air Quality Modeling TSD (U.S. EPA, 2003b).

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Air Quality Modeling TSD (U.S. EPA, 2003b). This version 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 NO₃ and N₂O₅ 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 SO₂ by O₃ and O₂ and to include the cloud and rain liquid water content from MM5 meteorological data directly in sulfate production and deposition calculations.

As discussed earlier in Chapter 2, the model tends to underestimate observed PM_{2.5} concentrations nationwide, especially over the western U.S.

9.3.2.2 Ozone Air Quality Modeling with CAMx

We use the emissions inputs described in Chapter 3 with a regional-scale version of CAMx to estimate ozone air quality in the Eastern and Western U.S. CAMx is an Eulerian three-dimensional photochemical grid air quality model designed to calculate the concentrations of both inert and chemically reactive pollutants by simulating the physical and chemical processes in the atmosphere that affect 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 proposed rule on U.S. ozone concentrations. As discussed earlier in Chapter 2, 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, On-highway Tier-2, and HD Engine/Diesel Fuel).

Our analysis applies the modeling system separately to the Eastern and Western U.S. for five emissions scenarios: a 1996 baseline projection, a 2020 baseline projection and a 2020 projection with nonroad controls, a 2030 baseline projection and a 2030 projection with nonroad controls. As discussed in detail in the technical support document, a 1996 base year assessment is necessary because the relative model predictions are used with ambient air quality observations from 1996 to determine the expected changes in 2020 and 2030 ozone concentrations due to the modeled emission changes (Abt Associates, 2003). These results are used solely in the benefits analysis.

As discussed in more detail in Chapter 2.3, our ozone air quality modeling showed that the NO_x emissions reductions from the preliminary modeled standards are projected to result in increases in ozone concentrations for certain hours during the year, especially in urban, NO_x-limited areas. Most of these increases are expected to occur during hours where ozone levels are low (and often below the one-hour ozone standard). However, most of the country experiences decreases in ozone concentrations for most hours in the year.

9.3.3 Health Effect Concentration-Response Functions

Health benefits for this analysis are based on health effect incidence changes due to predicted air quality changes in the years 2020 and 2030. Integral to the estimation of such benefits is a reasonable estimate of future population projections. The underlying data used to create county-level 2020 and 2030 population projections is 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. Growth factors are calculated using the Woods and Poole data and are applied to 2000 U.S. Census data.

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 Table 9-5. 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 (U.S. EPA, 1996).

To generate health outcomes, projected changes in ambient PM and ozone concentrations were put into the Criteria Air Pollutant Modeling System (CAPMS), a customized GIS-based program. CAPMS aggregates population to air quality model grids and calculates changes in air pollution metrics (e.g., daily averages) for input into C-R functions. CAPMS uses grid cell level population data and changes in pollutant concentrations to estimate changes in health outcomes for each grid cell. Details on the application of CAPMS for this analysis are provided in a separate report (Abt Associates, 2003).

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 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

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population is assumed to represent typical incidence at the national level. Sources of baseline incidence rates are reported in Table 9-6.

In this assessment we made analytical judgements affecting both the selection of C-R functions and the application of those functions in estimating impacts on health outcomes. Some of the more important of these are discussed below. Alternative assumptions about these judgements may lead to substantially different results and they are explored using appropriate sensitivity analyses provided in Appendix 9B.

Premature Mortality

As in the Kunzli et al. (2000) analysis, we focus on the prospective cohort long-term exposure studies in deriving the C-R function for our base estimate of premature mortality. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli, 2001; NRC, 2002). We selected a C-R function from the re-analysis of the American Cancer Society (ACS) study conducted for the Health Effects Institute (Pope et al., 1995; Krewski et al; 2000)^D. The selected C-R function relates premature mortality and mean PM_{2.5} levels rather than median levels as used in the original ACS analysis. For policy analysis purposes, functions based on the mean air quality levels may be preferable to functions based on the median air quality levels because changes in the mean more accurately reflect the changes in peak values targeted by many policies than do changes in the median.

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 for premature mortality 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. The Alternative Estimate assumes 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. This will clearly provide a lower bound to the mortality impacts of fine particle exposure, as it omits any additional mortality impacts from longer term exposures. 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).

Chronic Illness

^DA recent analysis (Pope et al, 2002) reexamines the ACS cohort using a longer follow-up period. We have examined how using alternative C-R functions derived from this new study impact our results in a sensitivity analysis presented in Appendix 9B.

Although there are several studies examining the relationship between PM of different size fractions and incidence of chronic bronchitis, we use a study by Abbey et al (1995) to obtain our estimate of avoided incidences of chronic bronchitis, because Abbey et al (1995) is the only available estimate of the relationship between PM_{2.5} and chronic bronchitis. Based on the Abbey et al study, we estimate the number of new chronic bronchitis cases that will “reverse” over time and subtract these reversals from the estimate of avoided chronic bronchitis incidences. Reversals refer to those cases of chronic bronchitis that were reported at the start of the Abbey et al. survey, but were subsequently not reported at the end of the survey. Since we assume that chronic bronchitis is a permanent condition, we subtract these reversals. Given the relatively high value assigned to chronic bronchitis, this ensures that we do not overstate the economic value of this health effect.

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 (2000) 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 the U.S. These studies provide a weight of evidence for this type of effect. Several epidemiologic 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 is consistent with an increased risk of heart attacks.

Hospital Admissions and Respiratory Illnesses

Most emergency room (ER) visits do not result in an admission to the hospital. Our estimates of hospital admission costs do not include the costs of preadmission to the ER. Therefore we estimate both hospital admissions and ER visits and treat them as additive effects. Because we are estimating the incidence of non-fatal heart attacks separately, and the economic values assigned to heart attacks includes hospital costs, we subtract baseline heart attack admissions from the cardiovascular hospital admission baseline incidence rate to avoid doublecounting benefits associated with reducing incidences of non-fatal heart attacks.

For respiratory symptom related endpoints, we use a variety of C-R functions covering different symptoms and age groups. While there is a consistent body of evidence supporting a relationship between respiratory symptoms and PM and ozone exposure, there is often only a single

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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 Appendix 9A.

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, 2000), 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 Appendix 9B.

9.3.4 Economic Values for Health Outcomes

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects 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 Portnoy, 1987; Berger, 1987). Unit values for health endpoints are provided in Table 9-7. All values are in constant year 2000 dollars.

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 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 (2020 or 2030) 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.

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. Adjustment factors used to account for projected growth in real income from 1990 to 2030 are 1.09 for minor health effects, 1.33 for severe and chronic health effects, 1.29 for premature mortality, and 1.79 for recreational visibility. Adjustment factors for 2020 are 1.08 for minor health effects, 1.30 for severe and chronic health effects, 1.26 for premature mortality, and 1.70 for recreational visibility. Note that due to a lack of reliable projections of income growth past 2024, we assume constant WTP from 2024 through 2030. This will result in an underestimate of benefits occurring between 2024 and 2030. Details of the calculation of the income adjustment factors are provided in Appendix 9A.

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 EEAC of the SAB, The base estimate uses the 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 \$6.3 million in constant 2000 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) 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, 2) the relationship between age and willingness-to-pay for fatal risk reductions, and 3) the degree of prematurity in mortalities from air pollution. [THIS ALTERNATIVE VALUATION APPROACH IS BEING REVISED PER COMMENTS RECEIVED FROM OMB AND WILL BE UPDATED IN A FUTURE DRAFT]

9.3.5 Welfare Effects

Our analysis examines two categories of welfare effects: visibility in a subset of national parks and changes in consumer and producer surplus associated with changes in agricultural yields. There are a number of other environmental effects which may affect human welfare, but due to a lack of appropriate physical effects or valuation methods, we are unable to quantify or monetize these effects for our analysis of the nonroad standards.

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9.3.5.1 Visibility Benefits

Changes in the level of ambient particulate matter caused by the reduction in emissions from the preliminary control options will 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.

For the purposes of this analysis, visibility improvements were valued only for a limited set of mandatory federal Class I areas. Benefits of improved visibility in the places people live, work, and recreate outside of these limited set of Class I areas were not included in our estimate of total benefits, although they are examined in a sensitivity analysis presented in Appendix 9B. All households in the U.S. are assumed to derive some benefit from improvements in Class I areas, given their national importance and high visitation rates from populations throughout the U.S. However, values are assumed to be higher if the Class I area is located close to their home.^E We use the results of a 1988 contingent valuation survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b) to derive values for visibility improvements. 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. 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. However, these values are less certain and are thus presented only as a sensitivity estimate in Appendix 9B.

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 Nonroad Diesel Engine rule. The method for developing calibrated WTP functions is based on the approach developed by Smith, et al. (2002), and is described in detail in the benefits technical support document (Abt Associates, 2003). 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.

^E For details of the visibility estimates discussed in this section, please refer to the benefits technical support document for this RIA (Abt Associates 2003).

9.3.5.2 Agricultural Benefits

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 economic value associated with varying levels of yield loss for ozone-sensitive commodity crops is analyzed 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 employs 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. For some crops there are multiple C-R functions, some more sensitive to ozone and some less. Our base estimate assumes that crops are evenly mixed between relatively sensitive and relatively insensitive varieties.

The measure of benefits calculated by the AGSIM[®] model is the net change in consumer and producer surplus from baseline ozone concentrations to the ozone concentrations resulting from emission reductions. Using the baseline and post-control equilibria, the model calculates the change in net consumer and producer surplus on a crop-by-crop basis. Dollar values are aggregated across crops for each standard. The total dollar value represents a measure of the change in social welfare associated with changes in ambient ozone.

9.3.5.2 Other Welfare Benefits

Ozone also has been shown conclusively to cause discernible injury to forest trees (US EPA, 1996; Fox and Mickler, 1996). In our previous analysis of the HD Engine/Diesel Fuel rule, we were able to quantify the effects of changes in ozone concentrations on tree growth for a limited set of species. Due to data limitations, we were not able to quantify such impacts for this analysis. We plan to assess both physical impacts on tree growth and the economic value of those physical impacts in our analysis of the final rule. We will use econometric models of forest product supply and demand to estimate changes in prices, producer profits and consumer surplus.

An additional welfare benefit expected to accrue as a result of reductions in ambient ozone concentrations in the U.S. is the economic value the public receives from reduced aesthetic injury to forests. There is sufficient scientific information available to reliably establish that ambient ozone levels cause visible injury to foliage and impair the growth of some sensitive plant species (US EPA, 1996c, p. 5-521). However, present analytic tools and resources preclude EPA from quantifying the benefits of improved forest aesthetics.

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Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to impact large economic sectors. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative economic benefits analysis has been conducted.

The nonroad diesel standards, by reducing NO_x emissions, will also reduce nitrogen deposition on agricultural land and forests. There is some evidence that nitrogen deposition may have positive effects on agricultural output through passive fertilization. Holding all other factors constant, farmers' use of purchased fertilizers or manure may increase as deposited nitrogen is reduced. Estimates of the potential value of this possible increase in the use of purchased fertilizers are not available, but it is likely that the overall value is very small relative to other health and welfare effects.

The nonroad diesel standards are also expected to produce economic benefits in the form of reduced materials damage. There are two important categories of these benefits. Household soiling refers to the accumulation of dirt, dust, and ash on exposed surfaces. Criteria pollutants also have corrosive effects on commercial/industrial buildings and structures of cultural and historical significance. The effects on historic buildings and outdoor works of art are of particular concern because of the uniqueness and irreplaceability of many of these objects.

Previous EPA benefit analyses have been able to provide quantitative estimates of household soiling damage. Consistent with SAB advice, we determined that the existing data (based on consumer expenditures from the early 1970's) are too out of date to provide a reliable enough estimate of current household soiling damages (EPA-SAB-Council-ADV-003, 1998) to include in our base estimate. We calculate household soiling damages in a sensitivity estimate provided in Appendix 9B.

EPA is unable to estimate any benefits to commercial and industrial entities from reduced materials damage. Nor is EPA able to estimate the benefits of reductions in PM-related damage to historic buildings and outdoor works of art. Existing studies of damage to this latter category in Sweden (Grosclaude and Soguel, 1994) indicate that these benefits could be an order of magnitude larger than household soiling benefits.

Reductions in emissions of diesel hydrocarbons that result in unpleasant odors may also lead to improvements in public welfare. The magnitude of this benefit is very uncertain, however, Lareau and Rae (1989) found a significant and positive WTP to reduce the number of exposures to diesel odors. They found that households were on average willing to pay around \$20 to \$27 (2000\$) per year for a reduction of one exposure to intense diesel odors per week (translating this to a national level, for the approximately 125 million households in 2020, the total WTP would be between \$2.5 and \$3.4 billion annually). Their results are not in a form that can be transferred to the context of this analysis, but the general magnitude of their results suggests this could be a significant welfare benefit of the rule.

The effects of air pollution on the health and stability of ecosystems are potentially very important, but are at present poorly understood and difficult to measure. The reductions in NO_x caused by the proposed rule could produce significant benefits. Excess nutrient loads, especially of nitrogen, cause a variety of adverse consequences to the health of estuarine and coastal waters. These effects include toxic and/or noxious algal blooms such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure (Bricker et al., 1999).

Direct C-R functions relating changes in nitrogen loadings to changes in estuarine benefits are not available. The preferred WTP based measure of benefits depends on the availability of these C-R functions and on estimates of the value of environmental responses. Because neither appropriate C-R functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, calculation of a WTP measure is not possible.

If better models of ecological effects can be defined, EPA believes that progress can be made in estimating WTP measures for ecosystem functions. For example, if nitrogen or sulfate loadings can be linked to measurable and definable changes in fish populations or definable indexes of biodiversity, then CV studies can be designed to elicit individuals' WTP for changes in these effects. This is an important area for further research and analysis, and will require close collaboration among air quality modelers, natural scientists, and economists.

9.3.6 Treatment of Uncertainty

In any complex analysis, there are likely to be many sources of uncertainty. This analysis is no exception. Many inputs are used to derive the final estimate of economic benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological estimates of C-R functions, estimates of values, population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Some of the key uncertainties in the benefits analysis are presented in Table 9-8. For some parameters or inputs it may be possible to provide a statistical representation of the underlying uncertainty distribution. For other parameters or inputs, the necessary information is not available.

In addition to uncertainty, the annual benefit estimates presented in this analysis are also inherently variable due to the truly random processes that govern pollutant emissions and ambient air quality in a given year. Factors such as hours of equipment use and weather display constant variability regardless of our ability to accurately measure them. As such, the estimates of annual benefits should be viewed as representative of the magnitude of benefits expected, rather than the actual benefits that would occur every year.

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We present a base estimate of the total benefits, based on the best available scientific literature and methods, an alternative estimate based on several important alternative assumptions about the estimation and valuation of reductions in premature mortality and chronic bronchitis. We also provide sensitivity analyses to illustrate the effects of uncertainty about key analytical assumptions. Our analysis of the preliminary control options did not include formal integrated probabilistic 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 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. We are working to implement these recommendations, however, for this proposal we do not attempt to assign probabilities to sensitivity estimates due to a lack of peer-reviewed methods. We plan to better characterize some of this uncertainty, especially regarding mortality-related benefits in the RIA to accompany the final rule.

9.3.7 Model Results

Full implementation of the modeled preliminary control options is projected in 2020 to reduce 48-state emissions of NO_x by 663,600 tons (58 percent of landbased nonroad emissions), SO₂ by 305,000 tons (98.9 percent), VOC by 23,200 tons (24 percent) and directly emitted PM_{2.5} by 91,300 tons (71 percent). In 2030, the modeled preliminary control options are expected to reduce 48-state emissions of NO_x by 1 million tons (82 percent), SO₂ by 359,800 tons (99.7 percent), VOC by 34,000 tons (35 percent) and direct PM by 129,000 tons (90 percent).

Based on these projected emission changes, REMSAD modeling results indicate the pollution controls generate greater absolute air quality improvements in more populated, urban areas. The rule will reduce average annual mean concentrations of PM_{2.5} across the U.S. by roughly 2.5 percent (or 0.2 μg/m³) and 3.4 percent (or 0.28 μg/m³) in 2020 and 2030, respectively. The population-weighted average mean concentration declined by 3.3 percent (or 0.42 μg/m³) in 2020 and 4.5 percent (or 0.59 μg/m³) in 2030, which is much larger in absolute terms than the spatial average for both years. Table 9-9 presents information on the distribution of modeled reductions in ambient PM concentrations across populations in the U.S. Significant populations live in areas with meaningful reductions in annual mean PM_{2.5} concentrations resulting from the pollution controls. As shown, slightly over 50 percent will live in areas with reductions of greater than 0.5 μg/m³. This information indicates how widespread the improvements in PM air quality are expected to be.

Applying the C-R functions described in Table 9-5 to the estimated changes in PM_{2.5} and ozone yields estimates of the number of avoided incidences for each health outcome. These estimates are presented in Table 9-10 for the 2020 and 2030 model analysis years. To provide estimates of the monetized benefits of the reductions in PM-related health outcomes described in Table 9-10, we multiply the point estimates of avoided incidences by unit values. Values for welfare effects are based

on application of the economic models described above. The estimated total monetized health and welfare benefits are presented in Table 9-11.

The largest monetized health benefit is associated with reductions in the risk of premature mortality, which accounts for over \$80 billion, or over 90 percent of total monetized health benefits. The next is for chronic illness reductions (chronic bronchitis and nonfatal heart attacks), although this value is more than an order of magnitude lower than for premature mortality. Minor restricted activity days, work loss days, and hospital admissions account for the majority of the remaining benefits. While the other categories account for less than \$100 million each, they represent a large number of avoided incidences affecting many individuals.

Ozone benefits are in aggregate positive for the nation. However, due to ozone increases occurring during certain hours of the day in some urban areas, in 2020 the net effect is an increase in ozone-related minor restricted activity days (MRAD), which are related to changes in daily average ozone (which includes hours during which ozone levels are low, but are increased relative to the baseline). However, by 2030, there is a net decrease in ozone-related MRAD consistent with widespread reductions in ozone concentrations from the increased NO_x emissions reductions. Note that in both years, the overall impact of changes in both PM and ozone is a large decrease in the number of MRAD. Overall, ozone benefits are low relative to PM benefits for similar endpoint categories because of the increases in ozone concentrations during some hours of some days in certain urban areas. For a more complete discussion of this issue, see Chapter 2.

Welfare benefits are far outweighed by health benefits, partly due to the incomplete coverage of important welfare categories, including the value of changes in ecosystems from reduced deposition of nitrogen and sulfur. The welfare benefits we are able to quantify are dominated by the value of improved visibility. Visibility benefits just in the limited set of parks included in the monetized total benefit estimate are over \$2 billion in 2030. Agricultural benefits, while small relative to visibility benefits, are significant relative to ozone-related health benefits, representing the largest single benefit category for ozone.

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Table 9-6. Endpoints and Studies Used to Calculate Total Monetized Health Benefits

Endpoint	Pollutant	Applied Population	Source of Effect Estimate(s)	Source of Baseline Incidence
Premature Mortality				
Base – Long-term exposure	PM _{2.5}	>29 years	Krewski, et al. (2000)	CDC Wonder (1996-1998)
Alternative – Short-term exposure	PM _{2.5}	all ages	Schwartz et al. (1996) adjusted using ratio of distributed lag to single day coefficients from Schwartz et al. (2000)	CDC Wonder (1996-1998)
Chronic Illness				
Chronic Bronchitis	PM _{2.5}	> 26 years	Abbey, et al. (1995)	1999 HIS (American Lung Association, 2002b, Table 4); Abbey et al. (1993, Table 3)
Non-fatal Heart Attacks	PM _{2.5}	Adults	Peters et al. (2001)	1999 NHDS public use data files; adjusted by 0.93 for prob. of surviving after 28 days (Rosamond et al., 1999)
Hospital Admissions				
Respiratory	O ₃	> 64 years	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
	O ₃	< 2 years	Burnett et al. (2001)	1999 NHDS public use data files
	PM _{2.5}	>64 years	Pooled estimate: Moolgavkar (2000) - ICD 490-496 (COPD) Lippman et al. (2000) - ICD 490-496 (COPD)	1999 NHDS public use data files
	PM _{2.5}	20-64 years	Moolgavkar (2000) - ICD 490-496 (COPD)	1999 NHDS public use data files
	PM _{2.5}	> 64 years	Lippman et al. (2000) - ICD 480-486	1999 NHDS public use data

Table 9-6. Endpoints and Studies Used to Calculate Total Monetized Health Benefits

Endpoint	Pollutant	Applied Population	Source of Effect Estimate(s)	Source of Baseline Incidence
	PM _{2.5}	< 65 years	Sheppard, et al. (1999) - ICD 493 (asthma)	1999 NHDS public use data files
Cardiovascular	PM _{2.5}	> 64 years	Pooled estimate: Moolgavkar (2000) - ICD 390-429 (all cardiovascular) Lippman et al. (2000) - ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	1999 NHDS public use data files
	PM _{2.5}	20-64 years	Moolgavkar (2000) - ICD 390-429 (all cardiovascular)	1999 NHDS public use data files
Asthma-Related ER Visits	O ₃	All ages	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
	PM _{2.5}	0-18 years	Norris et al. (1999)	2000 NHAMCS public use data files; 1999 NHDS public use data files
Other Health Endpoints				
Acute Bronchitis	PM _{2.5}	8-12 years	Dockery et al. (1996)	American Lung Association (2002a, Table 11)
Upper Respiratory Symptoms	PM ₁₀	Asthmatics, 9-11 years	Pope et al. (1991)	Pope et al. (1991, Table 2)
Lower Respiratory Symptoms	PM _{2.5}	7-14 years	Schwartz and Neas (2000)	Schwartz (1994, Table 2)
Work Loss Days	PM _{2.5}	18-65 years	Ostro (1987)	1996 HIS (Adams et al., 1999, Table 41); U.S. Bureau of the Census (2000)
School Absence Days	O ₃	9-10 years 6-11 years	Pooled estimate: Gilliland et al (2001) Chen et al (2000)	National Center for Education Statistics (1996)
Worker Productivity	O ₃	Outdoor workers, 18-65	Crocker and Horst (1981) and U.S. EPA (1984)	NA
Minor Restricted Activity Days	PM _{2.5} , O ₃	18-65 years	Ostro and Rothschild (1989)	Ostro and Rothschild (1989, p. 243)

**Table 9-7.
Unit Values Used for Economic Valuation of Health Endpoints (2000\$)**

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Premature Mortality				Base value is the mean of VSL estimates from 26 studies (5 contingent valuation and 21 labor market studies) reviewed for the Section 812 Costs and Benefits of the Clean Air Act, 1990-2010 (US EPA, 1999). Alternative values are based on adjustments to the mean of VSL estimates from the 5 contingent valuation studies referenced above. Adjustments are made for age and expected number of life years remaining based on cause of death and assumed health status at time of death.
Base Estimate	\$6,300,000	\$8,000,000	\$8,100,000	
Alternative Estimate				
<u>3% discount rate</u>				
COPD deaths (under 65)	\$84,000	\$110,000	\$110,000	
COPD deaths (65 and older)	\$136,000	\$170,000	\$170,000	
Other causes (under 65)	\$790,000	\$1,000,000	\$1,000,000	
Other causes (65 and older)	\$1,200,000	\$1,600,000	\$1,600,000	
<u>7% discount rate</u>				
COPD deaths (under 65)	\$140,000	\$170,000	\$170,000	
COPD deaths (65 and older)	\$160,000	\$200,000	\$200,000	
Other causes (under 65)	\$1,200,000	\$1,500,000	\$1,500,000	
Other causes (65 and older)	\$1,400,000	\$1,700,000	\$1,700,000	

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates												
	1990 Income Level	2020 Income Level	2030 Income Level													
Chronic Bronchitis (CB) Base Estimate Alternative Estimate <u>3% discount rate</u> Age 27-44 Age 45-64 Age 65 and older <u>7% discount rate</u> Age 27-44 Age 45-64 Age 65 and older	\$340,000 \$150,542 \$97,610 \$11,088 \$86,026 \$72,261 \$9,030	\$430,000 \$150,542 \$97,610 \$11,088 \$86,026 \$72,261 \$9,030	\$440,000 \$150,542 \$97,610 \$11,088 \$86,026 \$72,261 \$9,030	<p>Base value is the mean of a generated distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP (as described in Viscusi et al., 1991) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB.</p> <p>Alternative value is a cost of illness (COI) estimate based on Cropper and Krupnick (1990). Includes both medical costs and opportunity cost from age of onset to expected age of death (assumes that chronic bronchitis does not change life expectancy).</p>												
Non-fatal Myocardial Infarction (heart attack) <u>3% discount rate</u> Age 0-24 Age 25-44 Age 45-54 Age 55-65 Age 66 and over <u>7% discount rate</u> Age 0-24 Age 25-44 Age 45-54 Age 55-65 Age 66 and over	\$66,902 \$66,902 \$74,676 \$78,834 \$140,649 \$66,902 \$65,293 \$73,149 \$76,871 \$132,214 \$65,293	\$66,902 \$66,902 \$74,676 \$78,834 \$140,649 \$66,902 \$65,293 \$73,149 \$76,871 \$132,214 \$65,293	\$66,902 \$66,902 \$74,676 \$78,834 \$140,649 \$66,902 \$65,293 \$73,149 \$76,871 \$132,214 \$65,293	<p>Age specific cost-of-illness values reflecting lost earnings and direct medical costs over a 5 year period following a non-fatal MI. Lost earnings estimates based on Cropper and Krupnick (1990). Direct medical costs based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990).</p> <p><u>Lost earnings:</u> Cropper and Krupnick (1990). Present discounted value of 5 yrs of lost earnings:</p> <table border="0"> <tr> <td><u>age of onset:</u></td> <td><u>at 3%</u></td> <td><u>at 7%</u></td> </tr> <tr> <td>25-44</td> <td>\$8,774</td> <td>\$7,855</td> </tr> <tr> <td>45-54</td> <td>\$12,932</td> <td>\$11,578</td> </tr> <tr> <td>55-65</td> <td>\$74,746</td> <td>\$66,920</td> </tr> </table> <p><u>Direct medical expenses:</u> An average of: 1. Wittels et al., 1990 (\$102,658 – no discounting) 2. Russell et al., 1998, 5-yr period. (\$22,331 at 3% discount rate; \$21,113 at 7% discount rate)</p>	<u>age of onset:</u>	<u>at 3%</u>	<u>at 7%</u>	25-44	\$8,774	\$7,855	45-54	\$12,932	\$11,578	55-65	\$74,746	\$66,920
<u>age of onset:</u>	<u>at 3%</u>	<u>at 7%</u>														
25-44	\$8,774	\$7,855														
45-54	\$12,932	\$11,578														
55-65	\$74,746	\$66,920														

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Hospital Admissions				
Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)	\$12,378	\$12,378	\$12,378	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Pneumonia (ICD codes 480-487)	\$14,693	\$14,693	\$14,693	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total pneumonia category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Asthma admissions	\$6,634	\$6,634	\$6,634	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
All Cardiovascular (ICD codes 390-429)	\$18,387	\$18,387	\$18,387	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Emergency room visits for asthma	\$286	\$286	\$286	Simple average of two unit COI values: (1) \$311.55, from Smith et al., 1997, and (2) \$260.67, from Stanford et al., 1999.
Respiratory Ailments Not Requiring Hospitalization				

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Upper Respiratory Symptoms (URS)	\$25	\$27	\$27	Combinations of the 3 symptoms for which WTP estimates are available that closely match those listed by Pope, et al. result in 7 different “symptom clusters,” each describing a “type” of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for URS is the average of the dollar values for the 7 different types of URS.
Lower Respiratory Symptoms (LRS)	\$16	\$17	\$17	Combinations of the 4 symptoms for which WTP estimates are available that closely match those listed by Schwartz, et al. result in 11 different “symptom clusters,” each describing a “type” of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS.
Acute Bronchitis	\$360	\$390	\$390	Assumes a 6 day episode, with daily value equal to the average of low and high values for related respiratory symptoms recommended in Neumann, et al. 1994.
Restricted Activity and Work/School Loss Days				
Work Loss Days (WLDs)	Variable (national median = \$115)			County-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5 – to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.

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Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
School Absence Days	\$75	\$75	\$75	<p>Based on expected lost wages from parent staying home with child. Estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This median wage is \$551. Dividing by 5 gives an estimated median daily wage of \$103.</p> <p>The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of \$103, or \$75.</p>
Worker Productivity	\$0.95 per worker per 10% change in ozone per day	\$0.95 per worker per 10% change in ozone per day	\$0.95 per worker per 10% change in ozone per day	Based on \$68 – median daily earnings of workers in farming, forestry and fishing – from Table 621, Statistical Abstract of the United States (“Full-Time Wage and Salary Workers – Number and Earnings: 1985 to 2000”) (Source of data in table: U.S. Bureau of Labor Statistics, Bulletin 2307 and Employment and Earnings, monthly).
Minor Restricted Activity Days (MRADs)	\$51	\$55	\$56	Median WTP estimate to avoid one MRAD from Tolley, et al. (1986).

Table 9-8.
Primary Sources of Uncertainty in the Benefit Analysis

<i>1. Uncertainties Associated With Concentration-Response Functions</i>	
§	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.
<i>2. Uncertainties Associated With Ozone and PM Concentrations</i>	
§	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.
<i>3. Uncertainties Associated with PM Mortality Risk</i>	
§	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.
<i>4. Uncertainties Associated With Possible Lagged Effects</i>	
§	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.
<i>5. Uncertainties Associated With Baseline Incidence Rates</i>	
§	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.
<i>6. Uncertainties Associated With Economic Valuation</i>	
§	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.
<i>7. Uncertainties Associated With Aggregation of Monetized Benefits</i>	

§ Health and welfare benefits estimates are limited to the available C-R functions. Thus, unquantified or unmonetized benefits are not included.

Table 9-9.
Distribution of PM_{2.5} Air Quality Improvements Over Population
Due to Nonroad Engine/Diesel Fuel Standards: 2020 and 2030

Change in Annual Mean PM _{2.5} Concentrations (µg/m ³)	2020 Population		2030 Population	
	Number (millions)	Percent (%)	Number (millions)	Percent (%)
0 >) PM _{2.5} Conc # 0.25	65.11	19.75%	28.60	8.04%
0.25 >) PM _{2.5} Conc # 0.5	184.52	55.97%	147.09	41.33%
0.5 >) PM _{2.5} Conc # 0.75	56.66	17.19%	107.47	30.20%
0.75 >) PM _{2.5} Conc # 1.0	14.60	4.43%	38.50	10.82%
1.0 >) PM _{2.5} Conc # 1.25	5.29	1.60%	88.22	2.48%
1.25 >) PM _{2.5} Conc # 1.5	3.51	1.06%	15.52	4.36%
1.5 >) PM _{2.5} Conc # 1.75	0	0.00%	5.70	1.60%
) PM _{2.5} Conc > 1.75	0	0.00%	4.19	1.18%

^a The change is defined as the control case value minus the base case value.

Table 9-10.
Reductions in Incidence of Adverse Health Effects Associated with Reductions in Particulate Matter and Ozone Due to the Modeled Preliminary Nonroad Engine Standards

Endpoint	Avoided Incidence ^A (cases/year)	
	2020	2030
PM-related Endpoints		
Premature mortality ^B -		
Base estimate: Long-term exposure (adults, 30 and over)	6,200	11,000
Alternative estimate: Short-term exposure (all ages)	3,700	6,600
Chronic bronchitis (adults, 26 and over)	4,300	6,500
Non-fatal myocardial infarctions (adults, 18 and older)	11,000	18,000
Hospital admissions – Respiratory (all ages) ^C	3,100	5,500
Hospital admissions – Cardiovascular (adults, 20 and older) ^D	3,300	5,700
Emergency Room Visits for Asthma (18 and younger)	4,300	6,500
Acute bronchitis (children, 8-12)	10,000	16,000
Lower respiratory symptoms (children, 7-14)	110,000	170,000
Upper respiratory symptoms (asthmatic children, 9-11)	92,000	120,000
Work loss days (adults, 18-65)	780,000	1,100,000
Minor restricted activity days (adults, age 18-65)	4,600,000	6,500,000
Ozone-related Endpoints		
Hospital Admissions – Respiratory Causes (adults, 65 and older) ^E	370	1,100
Hospital Admissions - Respiratory Causes (children, under 2 years)	150	280
Emergency Room Visits for Asthma (all ages)	93	200
Minor restricted activity days (adults, age 18-65)	(2,400)	96,000
School absence days (children, age 6-11)	65,000	96,000

^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.

^E Respiratory hospital admissions for ozone includes admissions for all respiratory causes and subcategories for COPD and pneumonia.

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Table 9-11
Results of Human Health and Welfare Benefits
Valuation for the Modeled Preliminary Nonroad Diesel Engine Standards

Endpoint	Pollutant	Monetary Benefits ^{A,B} (millions 2000\$, Adjusted for Income Growth)	
		2020	2030
Premature mortality ^C	PM		
Base estimate: Long-term exposure, (adults, 30 and over)			
3% discount rate		\$47,000	\$85,000
7% discount rate		\$44,000	\$80,000
Alternative estimate: Short-term exposure, (all ages)			
3% discount rate		\$5,000	\$9,100
7% discount rate	\$5,700	\$10,000	
Chronic bronchitis (adults, 26 and over)	PM		
Base estimate: Willingness-to-pay		\$1,900	\$3,000
Alternative estimate: Cost-of-illness			
3% discount rate		\$420	\$600
7% discount rate	\$270	\$390	
Non-fatal myocardial infarctions	PM		
3% discount rate		\$900	\$1,400
7% discount rate		\$870	\$1,400
Hospital Admissions from Respiratory Causes	O ₃ and PM	\$55	\$110
Hospital Admissions from Cardiovascular Causes	PM	\$72	\$120
Emergency Room Visits for Asthma	O ₃ and PM	\$1	\$2
Acute bronchitis (children, 8-12)	PM	\$4	\$6
Lower respiratory symptoms (children, 7-14)	PM	\$2	\$3
Upper respiratory symptoms (asthmatic children, 9-11)	PM	\$2	\$3
Work loss days (adults, 18-65)	PM	\$110	\$150
Minor restricted activity days (adults, age 18-65)	O ₃ and PM	\$250	\$370
School absence days (children, age 6-11)	O ₃	\$5	\$10
Worker productivity (outdoor workers, age 18-65)	O ₃	\$4	\$7
Recreational visibility (86 Class I Areas)	PM	\$1,400	\$2,200
Agricultural crop damage (6 crops)	O ₃	\$89	\$140
Monetized Total ^H	O ₃ and PM		
Base estimate			
3% discount rate		\$52,000+B	\$92,000+B
7% discount rate		\$49,000+B	\$87,000+B
Alternative estimate			
3% discount rate	\$8,300+B	\$14,000+B	

Cost-Benefit Analysis

Premature mortality ^c	PM		
Base estimate: Long-term exposure, (adults, 30 and over)			
3% discount rate		\$47,000	\$85,000
7% discount rate		\$44,000	\$80,000
Alternative estimate: Short-term exposure, (all ages)			
3% discount rate		\$5,000	\$9,100
7% discount rate		\$5,700	\$10,000
Chronic bronchitis (adults, 26 and over)	PM		
Base estimate: Willingness-to-pay		\$1,900	\$3,000
Alternative estimate: Cost-of-illness			
3% discount rate		\$420	\$600
7% discount rate		\$270	\$390
Non-fatal myocardial infarctions	PM		
3% discount rate		\$900	\$1,400
7% discount rate		\$870	\$1,400
Hospital Admissions from Respiratory Causes	O ₃ and PM	\$55	\$110
Hospital Admissions from Cardiovascular Causes	PM	\$72	\$120
Emergency Room Visits for Asthma	O ₃ and PM	\$1	\$2
Acute bronchitis (children, 8-12)	PM	\$4	\$6
Lower respiratory symptoms (children, 7-14)	PM	\$2	\$3
Upper respiratory symptoms (asthmatic children, 9-11)	PM	\$2	\$3
Work loss days (adults, 18-65)	PM	\$110	\$150
Minor restricted activity days (adults, age 18-65)	O ₃ and PM	\$250	\$370
School absence days (children, age 6-11)	O ₃	\$5	\$10
Worker productivity (outdoor workers, age 18-65)	O ₃	\$4	\$7
7% discount rate		\$8,800+B	\$15,000+B

^A Monetary benefits are rounded to two significant digits.

^B Monetary benefits are adjusted to account for growth in real GDP per capita between 1990 and the analysis year (2020 or 2030).

^C Premature mortality associated with ozone is not separately included in this analysis. It is assumed that the C-R function for premature mortality captures both PM mortality benefits and any mortality benefits associated with other air pollutants. Also note that the valuation assumes the 5 year distributed lag structure described earlier. Results reflect the use of two different discount rates; a 3% rate which is recommended by EPA's Guidelines for Preparing Economic Analyses (US EPA, 2000c), and 7% which is recommended by OMB Circular A-94 (OMB, 1992).

^D Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.

^E Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

^F Respiratory hospital admissions for ozone includes admissions for all respiratory causes and subcategories for COPD and pneumonia.

^G B represents the monetary value of the unmonetized health and welfare benefits. A detailed listing of unquantified PM, ozone, CO, and NMHC related health effects is provided in Table 9.1.

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9.3.8 Apportionment of Benefits to NO_x, SO₂, and PM Emissions Reductions

As noted in the introduction to this chapter, the proposed standards differ from those that we used in modeling air quality and economic benefits. As such, it is necessary for us to scale the modeled benefits to reflect the difference in emissions reductions between the proposed and preliminary modeled standards. In order to do so, however, we must first apportion total benefits to the NO_x, SO₂, and direct PM reductions for the modeled preliminary control options. This apportionment is necessary due to the differential contribution of each emission species to the total change in ambient PM and total benefits. We do not attempt to develop scaling factors for ozone benefits because of the difficulty in separating the contribution of NO_x and NMHC/VOC reductions to the change in ozone concentrations.

PM is a complex mixture of particles of varying species, including nitrates, sulfates, and primary particles, including organic and elemental carbon. These particles are formed in complex chemical reactions from emissions of precursor pollutants, including NO_x, SO₂, ammonia, hydrocarbons, and directly emitted particles. Different emissions species contribute to the formation of PM in different amounts, so that a ton of emissions of NO_x contributes to total ambient PM mass differently than a ton of SO₂ or directly emitted PM. As such, it is inappropriate to scale benefits by simply scaling the sum of all precursor emissions. A more appropriate scaling method is to first apportion total PM benefits to the changes in underlying emission species and then scale the apportioned benefits.

PM formation relative to any particular reduction in an emission species is a highly nonlinear process, depending on meteorological conditions and baseline conditions, including the amount of available ammonia to form ammonium nitrate and ammonium sulfate. Given the limited air quality modeling conducted for this analysis, we make several simplifying assumptions about the contributions of emissions reductions for specific species to changes in particle species. For this exercise, we assume that changes in sulfate particles are attributable to changes in SO₂ emissions, changes in nitrate particles are attributable to changes in NO_x emissions, and changes in primary PM are attributable to changes in direct PM emissions. These assumptions essentially assume independence between SO₂, NO_x, and direct PM in the formation of ambient PM. This is a reasonable assumption for direct PM, as it is generally not reactive in the atmosphere. However, SO₂ and NO_x emissions interact with other compounds in the atmosphere to form PM_{2.5}. For example, ammonia reacts with SO₂ first to form ammonium sulfate. If there is remaining ammonia, it reacts with NO_x to form ammonium nitrate. When SO₂ alone is reduced, ammonia is freed to react with any NO_x that has not been used in forming ammonium nitrate. If NO_x is also reduced, then there will be less available NO_x to form ammonium nitrate from the newly available ammonia. Thus, reducing SO₂ can potentially lead to decreased ammonium sulfate and increased nitrate, so that overall ambient PM benefits are less than the reduction in sulfate particles. If NO_x alone is reduced, there will be a direct reduction in ammonium nitrate, although the amount of reduction depends on whether an area is ammonia limited. If there is not enough ammonia in an area to use up all of the available NO_x, then NO_x reductions will only have an impact if they reduce emissions to the point where ammonium nitrate formation will be affected. NO_x

reductions will not result in any offsetting increases in ambient PM under most conditions. The implications of this for apportioning benefits between NO_x, SO₂, and direct PM is that some of the sulfate related benefits will be offset by reductions in nitrate benefits, so benefits from SO₂ reductions will be overstated, while NO_x benefits will be understated. It is not immediately apparent the size of this bias.

The measure of change in ambient particle mass that is most related to health benefits is the population-weighted change in PM_{2.5} : g/m³, because health benefits are driven both by the size of the change in PM_{2.5} and the populations exposed to that change. We calculate the proportional share of total change in mass accounted for by nitrate, sulfate, and primary particles. Results of these calculations for the 2020 and 2030 REMSAD modeling analysis are presented in Table 9-12. The sulfate percentage of total change is used to represent the SO₂ contribution to health benefits, the nitrate percentage is used to represent the NO_x contribution to health benefits, and the primary PM percentage is used to represent the direct PM contribution to health benefits. These percentages will be applied to the PM-related health benefits estimates in Table 9-10 and 9-11 and combined with the emission scaling factors developed in section 9.2 to estimate benefits for the proposed standards.

Table 9-12. Apportionment of Population Weighted Change in Ambient PM2.5 to Nitrate, Sulfate, and Primary Particles

	2020		2030	
	Population-weighted Change (: g/m3)	Percent of Total Change	Population-weighted Change (: g/m3)	Percent of Total Change
Total PM2.5	0.316		0.438	
Sulfate	0.071	22.5%	0.090	20.5%
Nitrate	0.041	13.1%	0.073	16.8%
Primary PM	0.203	64.4%	0.274	62.7%

Visibility benefits are highly specific to the parks at which visibility improvement occur, rather than where populations live. As such, it is necessary to scale benefits at each individual park and then aggregate to total scaled visibility benefits. We apportion benefits at each park using the contribution of changes in sulfates, nitrates, and primary particles to changes in light extinction. The change in light extinction at each park is determined by the following equation:

$$\Delta \mathbf{b}_{EXT} = [3F(rh) * 1.375 * \Delta TSO4] + [3F(rh) * 1.29 * \Delta PNO3] + 10 * \Delta PEC + 4 * \Delta TOA + \Delta PMFINE + 0.6 * \Delta PMCOARSE$$

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where rh is relative humidity, ΔTSO_4 is the change in particulate sulfate, ΔPNO_3 is the change in particulate nitrate, ΔPEC is the change in primary elemental carbon, ΔTOA is the change in total organic aerosols, $\Delta PMFINE$ is the change in primary fine particles, and $\Delta PMCOARSE$ is the change in primary coarse particles.

The proportion of the total change in light extinction associated with changes in sulfate particles is $[3F(rh) * 1.375 * \Delta TSO_4] / \Delta b_{EXT}$. The proportion of the total change in light extinction associated with changes in nitrate particles is $[3F(rh) * 1.29 * \Delta PNO_3] / \Delta b_{EXT}$. Finally, the proportion of the total change in light extinction associated with the change in directly emitted particles is $[10 * \Delta PEC + 4 * \Delta TOA + \Delta PMFINE + 0.6 * \Delta PMCOARSE] / \Delta b_{EXT}$.

We calculate these proportions for each park to apportion park specific benefits between SO_2 , NO_x , and PM. The apportioned benefits are then scaled using the emission ratios in Table 9-5. Park specific apportionment of benefits is detailed in Appendix 9C.

9.4 Estimated Benefits of Proposed Nonroad Diesel Engine Standards in 2020 and 2030

To estimate the benefits of the NO_x , SO_2 , and direct PM emission reductions from the proposed standards in 2020 and 2030, we apply the emissions scaling factors derived in section 9.2 and the apportionment factors described in section 9.3 to the benefits estimates for 2020 and 2030 listed in Tables 9-10 and 9-11. Note that we apply scaling and apportionment factors only to PM and visibility related endpoints. Ozone related health and welfare benefits are not estimated for the emissions reductions associated with the proposed standards for reasons noted in the introduction to this chapter.

The scaled avoided incidence estimate for any particular health endpoint is calculated using the following equation:

$$\text{Scaled Incidence} = \text{Modeled Incidence} * \sum_i R_i A_i,$$

where R_i is the emissions ratio for emission species i from Table 9-4, and A_i is the health benefits apportionment factor for emission species i , from Table 9-12. Essentially, benefits are scaled using a weighted average of the species specific emissions ratios. For example, the calculation of the avoided incidence of premature mortality for the base estimate in 2020 is:

$$\text{Scaled Premature Mortality Incidence} = 6,200 * (0.761 * 0.129 + 0.777 * 0.224 + 0.903 * 0.647) = 5,620$$

The monetized value for each endpoint is then obtained simply by multiplying the scaled incidence estimate by the appropriate unit value in Table 9-6. The estimated changes in incidence of health effects in 2020 and 2030 for the proposed rule based on application of the weighted scaling factors are presented in Table 9-13. The estimated monetized benefits for both PM health and visibility benefits are presented in Table 9-14. The visibility benefits are based on application of the weighted scaling factors for visibility at each Class I area in the Chestnut and Rowe study regions, aggregated to a national total for each year.

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**Table 9-13.
Reductions in Incidence of PM-related Adverse Health Effects Associated with the Proposed
Nonroad Diesel Engine Standards**

Endpoint	Avoided Incidence ^A (cases/year)	
	2020	2030
Premature mortality ^B -		
Base estimate: Long-term exposure (adults, 30 and over)	5,200	9,600
Alternative estimate: Short-term exposure (all ages)	3,100	5,800
Chronic bronchitis (adults, 26 and over)	3,600	5,700
Non-fatal myocardial infarctions (adults, 18 and older)	9,200	16,000
Hospital admissions – Respiratory (adults, 20 and older) ^C	2,400	4,500
Hospital admissions – Cardiovascular (adults, 20 and older) ^D	1,900	3,800
Emergency Room Visits for Asthma (18 and younger)	3,600	5,700
Acute bronchitis (children, 8-12)	8,400	14,000
Lower respiratory symptoms (children, 7-14)	92,000	150,000
Upper respiratory symptoms (asthmatic children, 9-11)	77,000	110,000
Work loss days (adults, 18-65)	650,000	960,000
Minor restricted activity days (adults, age 18-65)	3,900,000	5,700,000

^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.

**Table 9-14.
Results of Human Health and Welfare Benefits Valuation for the Proposed Nonroad Diesel
Engine Standards**

Endpoint	Monetary Benefits ^{A,B} (millions 2000\$, Adjusted for Income Growth)	
	2020	2030
Premature mortality ^C		
Base estimate: Long-term exposure, (adults, 30 and over)		
3% discount rate (over 5 year cessation lag)	\$39,000	\$74,000
7% discount rate (over 5 year cessation lag)	\$37,000	\$70,000
Alternative estimate: Short-term exposure, (all ages)		
3% discount rate (amortization of VSL)	\$4,200	\$8,000
7% discount rate (amortization of VSL)	\$4,800	\$9,100
Chronic bronchitis (adults, 26 and over) ^D		
Base estimate: Willingness-to-pay	\$1,600	\$2,600
Alternative estimate: Cost-of-illness		
3% discount rate (over lifetime with disease)	\$350	\$530
7% discount rate (over lifetime with disease)	\$220	\$340
Non-fatal myocardial infarctions ^E		
3% discount rate (over 5 year follow up)	\$750	\$1,300
7% discount rate (over 5 year follow up)	\$730	\$1,200
Hospital Admissions from Respiratory Causes ^F	\$38	\$74
Hospital Admissions from Cardiovascular Causes ^G	\$40	\$80
Emergency Room Visits for Asthma	\$1	\$2
Acute bronchitis (children, 8-12)	\$3	\$5
Lower respiratory symptoms (children, 7-14)	\$2	\$3
Upper respiratory symptoms (asthmatic children, 9-11)	\$2	\$3
Work loss days (adults, 18-65)	\$90	\$130
Minor restricted activity days (adults, age 18-65)	\$210	\$320
Recreational visibility (86 Class I Areas)	\$1,200	\$1,900
Monetized Total ^H		
Base estimate		
3% discount rate	\$43,000+B	\$81,000+B
7% discount rate	\$41,000+B	\$76,000+B
Alternative estimate		
3% discount rate	\$6,800+B	\$12,000+B
7% discount rate	\$7,300+B	\$13,000+B

^A Monetary benefits are rounded to two significant digits.

^B Monetary benefits are adjusted to account for growth in real GDP per capita between 1990 and the analysis year (2020 or 2030).

^C Valuation of base estimate assumes discounting over the 5 year distributed lag structure described earlier. Valuation of alternative estimate assumes value of a statistical life year derived from amortization of age specific value of statistical life over remaining life expectancy. Results reflect the use of two different discount rates; a 3% rate which is recommended by EPA's Guidelines for Preparing Economic Analyses (US EPA, 2000c), and 7% which is recommended by OMB Circular A-94 (OMB, 1992).

^D Alternative estimate assumes costs of illness and lost earnings in later life years are discounted using either 3 or 7 percent.

^E Estimates assume costs of illness and lost earnings in later life years are discounted using either 3 or 7 percent

^F Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.

^G Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

^H B represents the monetary value of the unmonetized health and welfare benefits. A detailed listing of unquantified PM, ozone, CO, and NMHC related health effects is provided in Table 9-1.

9.5 Development of Intertemporal Scaling Factors and Calculation of Benefits Over Time

To estimate the health and visibility benefits of the NO_x, SO₂, and direct PM emission reductions from the proposed standards occurring in years other than 2020 and 2030, it is necessary to develop factors to scale the modeled benefits in 2020 and 2030. In addition to scaling based on the relative reductions in NO_x, SO₂, and direct PM, intertemporal scaling requires additional adjustments to reflect population growth, changes in the age composition of the population, and per capita income levels.

Two separate sets of scaling factors are required, one for PM related health benefits, and one for visibility benefits. For the first of these, PM health benefits, we need scaling factors based on ambient PM_{2.5}. Because of the nonproportional relationship between precursor emissions and ambient concentrations of PM_{2.5}, it is necessary to first develop estimates of the marginal contribution of reductions in each emission species to reductions in PM_{2.5} in each year. Because we have only two points (2020 and 2030), we assume a very simple linear function for each species over time (assuming that the marginal contribution of each emission species to PM_{2.5} is independent of the other emission species) again assuming that sulfate changes are primarily associated with SO₂ emission reductions, nitrate changes are primarily associated with NO_x emission reductions, and primary PM changes are associated with direct PM emission reductions.

Using the linear relationship, we estimate the marginal contribution of SO₂ to sulfate, NO_x to nitrate, and direct PM to primary PM in each year. These marginal contribution estimates are presented in Table 9-15. Note that these projections do not take into account differences in overall baseline proportions of NO_x, SO₂, and PM. They assume that the change in the relative effectiveness of each emission species in reducing ambient PM that is observed between 2020 and 2030 can be extrapolated to other years. Because baseline emissions of NO_x, SO₂, and PM, as well as ammonia and VOCs are changing between years, the relative effectiveness of NO_x and SO₂ emission reductions may change in a non-linear fashion. It is not clear what overall biases these nonlinearities will introduce into the scaling exercise.

Multiplying the year specific marginal contribution estimates by the appropriate emissions reductions in each year yields estimates of the population weighted changes in PM_{2.5} constituent species, which are summed to obtain year specific population weighted changes in total PM_{2.5}. Total benefits in each specific year are then developed by scaling total benefits in a base year using the ratio of the change in PM_{2.5} in the target year to the base year, with additional scaling factors to account for growth in total population, age composition of the population, and growth in per capita income.

Table 9-15.
Projected Marginal Contribution of Reductions in Emission Species to Reductions in Ambient PM2.5

Change in PM2.5 species (population weighted : g/m ³ per million tons reduced)			
Year	Sulfate/SO ₂	Nitrate/NOx	Primary PM/direct PM
2007	0.233	0.048	1.988
2008	0.235	0.049	1.982
2009	0.237	0.050	1.976
2010	0.238	0.051	1.970
2011	0.240	0.052	1.964
2012	0.242	0.054	1.957
2013	0.244	0.055	1.951
2014	0.246	0.056	1.945
2015	0.247	0.057	1.939
2016	0.249	0.058	1.933
2017	0.251	0.059	1.927
2018	0.253	0.060	1.921
2019	0.254	0.061	1.915
2020	0.256	0.062	1.909
2021	0.258	0.063	1.903
2022	0.260	0.064	1.897
2023	0.262	0.065	1.891
2024	0.263	0.066	1.885
2025	0.265	0.067	1.879
2026	0.267	0.068	1.873
2027	0.269	0.069	1.867
2028	0.271	0.070	1.861
2029	0.272	0.071	1.854
2030	0.274	0.072	1.848

Growth in population and changes in age composition are accounted for by apportioning total benefits into benefits accruing to three different age groups, 0 to 18, 19 to 64, and 65 and older. Benefits for each age group are then adjusted by the ratio of the age group population in the target year to the age group population in the base year. Age composition adjusted estimates are then reaggregated to obtain total population and age composition adjusted benefits for each year. Growth in

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per capita income is accounted for by multiplying the target year estimate by the ratio of the income adjustment factors in the target year to those in the base year.

For example, for the target year of 2010, there are 2,281 tons of NO_x reductions, 293,124 tons of SO₂ reductions, and 22,967 tons of PM reductions. These are associated with a populated weighted change in total PM_{2.5} of 0.119. The ratio of this change to the change in the 2030 base year is 0.272. The age group apportionment factors (based on the Base estimate using a 3% discount rate for 2030) are 0.02% for 0 to 18, 19.4% for 19 to 64, and 80.6% for 65 and older. The age group population growth ratios for 2010 relative to 2030 are 0.88 for 0 to 18, 0.96 for 19 to 64, and 0.55 for 65 and older. The income growth adjustment ratios for 2015 are 0.85 for mortality endpoints and 0.84 for morbidity endpoints. Mortality accounts for 93 percent of total health benefits and morbidity accounts for 7 percent of health benefits. Combining these elements with the total Base estimate of PM health benefits in 2030 of \$89.8 billion, total PM health benefits in 2010 for the proposed standards are calculated as:

Total PM health benefits (2020) =

$$\$89.8 \text{ billion} * 0.272 * (0.0002 * 0.88 + 0.194 * 0.96 + 0.806 * 0.55) * (0.93 * .85 + 0.07 * .84) = \$13.1 \text{ billion}$$

In order to develop the time stream of visibility benefits, we need to develop scaling factors based on the contribution of each emission species to light extinction. Similar to ambient PM_{2.5}, because we have only two estimates of the change in light extinction (2020 and 2030), we assume a very simple linear function for each species over time (assuming that the marginal contribution of each emission species to light extinction is independent of the other emission species) assuming that changes in the sulfate component of light extinction are associated with SO₂ emission reductions, changes in the nitrate component of light extinction are primarily associated with NO_x emission reductions, and changes in the primary PM components of light extinction are associated with direct PM emission reductions. Linear relationships (slope and intercept) are calculated for each Class I area.

Using the linear relationships, we estimate the marginal contribution of SO₂, NO_x, and direct PM to the change in light extinction at each Class I area in each year. Again, note that these estimates assume that the change in the relative effectiveness of each emission species in reducing light extinction that is observed between 2020 and 2030 can be extrapolated to other years.

Multiplying the year specific marginal contribution estimates by the appropriate emissions reductions in each year yields estimates of the changes in light extinction components, which are summed to obtain year specific changes in total light extinction. Benefits for each park in each specific year are then developed by scaling total benefits in a base year using the ratio of the change in light extinction in the target year to the base year, with additional scaling factors to account for growth in total population, and growth in per capita income. Total national visibility benefits for each year are obtained by summing the scaled benefits across Class I areas.

Table 9-16 provides undiscounted estimates of the time stream of benefits for the proposed standards for the Base and Alternative estimates using 3 and 7 percent concurrent discount rates^F. Figure 9-1 shows the undiscounted time stream for the Base estimate using a 3 percent concurrent discount rate. Because of the assumptions we made about the linearity of benefits for each emission species, overall benefits are also linear, reflecting the relatively linear emissions reductions over time for each emission type. The exception is during the early years of the program, where there is little NO_x emission reduction, so that benefits are dominated by SO₂ and direct PM_{2.5} reductions.

Using a 3 percent intertemporal discount rate, the present value in 2004 of the benefits of the proposed standards for the base estimate is approximately \$550 billion for the time period 2007 to 2030, using either a 3 percent concurrent discount rate or \$520 billion using a 7 percent concurrent discount rate. For the alternative estimate, the present value using a 3 percent intertemporal discount rate is approximately \$90 billion using either a 3 or 7 percent concurrent discount rate. Annualized benefits using a 3 percent intertemporal discount rate for the base estimate are approximately \$30 billion using either a 3 or 7 percent concurrent discount rate. Annualized benefits using a 3 percent intertemporal discount rate for the alternative estimate are approximately \$5 billion using either a 3 or 7 percent concurrent discount rate.

Using a 7 percent intertemporal discount rate, the present value in 2004 of the benefits of the proposed standards for the base estimate is approximately \$290 billion for the time period 2007 to 2030, using a 3 percent concurrent discount rate or \$270 billion using a 7 percent concurrent discount rate. For the alternative estimate, the present value using a 7 percent intertemporal discount rate is approximately \$45 billion using a 3 percent concurrent discount rate or \$48 billion using a 7 percent concurrent discount rate.

^FWe refer to discounting that occurs during the calculation of benefits for individual years as concurrent discounting. This is distinct from discounting that occurs over the time stream of benefits, which is referred to as intertemporal discounting.

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Table 9-16. Time Stream of Benefits for Proposed Nonroad Diesel Engine Standards^{A,B}

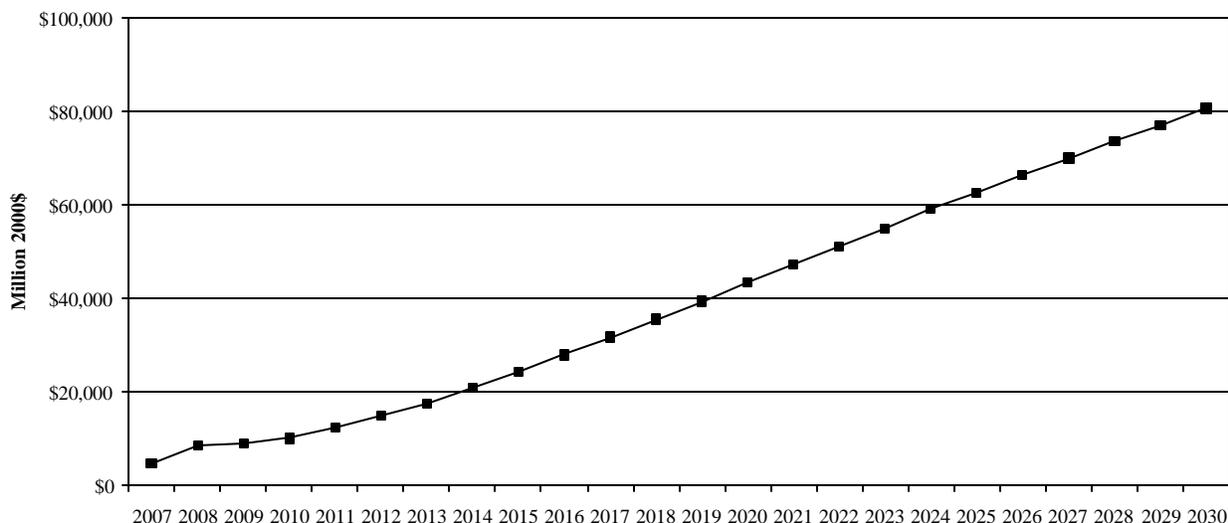
Year	Base Estimate (Million 2000\$)		Alternative Estimate (Million 2000\$)	
	3% Concurrent Discount Rate	7% Concurrent Discount Rate	3% Concurrent Discount Rate	7% Concurrent Discount Rate
2007	\$4,700	\$4,500	\$730	\$780
2008	\$8,600	\$8,100	\$1,300	\$1,400
2009	\$9,100	\$8,600	\$1,400	\$1,500
2010	\$10,000	\$9,500	\$1,500	\$1,600
2011	\$12,000	\$12,000	\$2,000	\$2,100
2012	\$15,000	\$14,000	\$2,400	\$2,500
2013	\$18,000	\$17,000	\$2,800	\$3,000
2014	\$21,000	\$20,000	\$3,300	\$3,600
2015	\$24,000	\$23,000	\$3,800	\$4,100
2016	\$28,000	\$26,000	\$4,400	\$4,700
2017	\$32,000	\$30,000	\$5,000	\$5,300
2018	\$35,000	\$34,000	\$5,500	\$5,900
2019	\$39,000	\$37,000	\$6,100	\$6,600
2020	\$43,000	\$41,000	\$6,700	\$7,200
2021	\$47,000	\$45,000	\$7,300	\$7,900
2022	\$51,000	\$48,000	\$7,900	\$8,500
2023	\$55,000	\$52,000	\$8,500	\$9,100
2024	\$59,000	\$56,000	\$9,200	\$9,800
2025	\$63,000	\$59,000	\$9,700	\$10,000
2026	\$66,000	\$63,000	\$10,000	\$11,000
2027	\$70,000	\$66,000	\$11,000	\$12,000
2028	\$74,000	\$70,000	\$11,000	\$12,000
2029	\$77,000	\$73,000	\$12,000	\$13,000
2030	\$81,000	\$76,000	\$12,000	\$13,000
Present Value in 2004				
3% Intertemporal Discount Rate	\$550,000	\$520,000	\$85,000	\$91,000
7% Intertemporal Discount Rate	\$290,000	\$270,000	\$45,000	\$48,000

^A All dollar estimates rounded to two significant digits.

^B Results reflect the use of two different discount rates; a 3% rate which is recommended by EPA's Guidelines for Preparing Economic Analyses (US EPA, 2000c), and 7% which is recommended by OMB Circular A-94 (OMB, 1992).

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Figure 9-1.
Base Estimate of the Stream of Annual Benefits for the Proposed Nonroad Diesel Engine Standards: 2007 to 2030



Engine Standards: 2007 to 2030

9.6 Comparison of Costs and Benefits

The estimated social cost (measured as changes in consumer and producer surplus) in 2030 to implement the final rule, as described in Chapter 8 is \$1.2 billion (2000\$). Thus, the net benefit (social benefits minus social costs) of the program at full implementation is approximately \$80 + B billion. In 2020, partial implementation of the program yields net benefits of \$41 + B billion. Therefore, implementation of the final rule is expected to provide society with a net gain in social welfare based on economic efficiency criteria. Table 9-17 presents a summary of the benefits, costs, and net benefits of the proposed rule. Figure 9-2 displays the stream of benefits, costs, and net benefits of the Nonroad Diesel Engine and Fuel Standards from 2007 to 2030. In addition, Table 9-18 presents the present value of the stream of benefits, costs, and net benefits associated with the rule for this 23 year period (using a three percent discount rate). The total present value of the stream of net benefits (benefits minus costs) is \$530 billion.

Table 9-17.
Summary of Benefits, Costs, and Net Benefits of the
Proposed Nonroad Diesel Engine and Fuel Standards

	2020^A (Billions of 2000 dollars)	2030^A (Billions of 2000 dollars)
Social Costs^B	\$1.1	\$1.2
Social Benefits^{B, C, D:}		
CO, VOC, Air Toxic-related benefits	Not monetized	Not monetized
Ozone-related benefits	Not monetized	Not monetized
PM-related Welfare benefits	\$1.2	\$1.9
PM-related Health benefits	\$42 + B	\$79 + B
Net Benefits (Benefits-Costs)^{C, D}	\$41 + B	\$80 + B

^A All costs and benefits are rounded to two significant digits.

^B Note that costs are the total costs of reducing all pollutants, including CO, VOCs and air toxics, as well as NOx and PM. Benefits in this table are associated only with PM, NOx and SO₂ reductions.

^C Not all possible benefits or disbenefits are quantified and monetized in this analysis. Potential benefit categories that have not been quantified and monetized are listed in Table 9-1. B is the sum of all unquantified benefits and disbenefits.

^D Monetized benefits are presented using two different discount rates. Results calculated using 3 percent discount rate are recommended by EPA's *Guidelines for Preparing Economic Analyses* (U.S. EPA, 2000c). Results calculated using 7 percent discount rate are recommended by OMB Circular A-94 (OMB, 1992).

Figure 9-2.
Stream of Benefits, Costs, and Net Benefits of the
Proposed Nonroad Diesel Engine and Fuel Standards

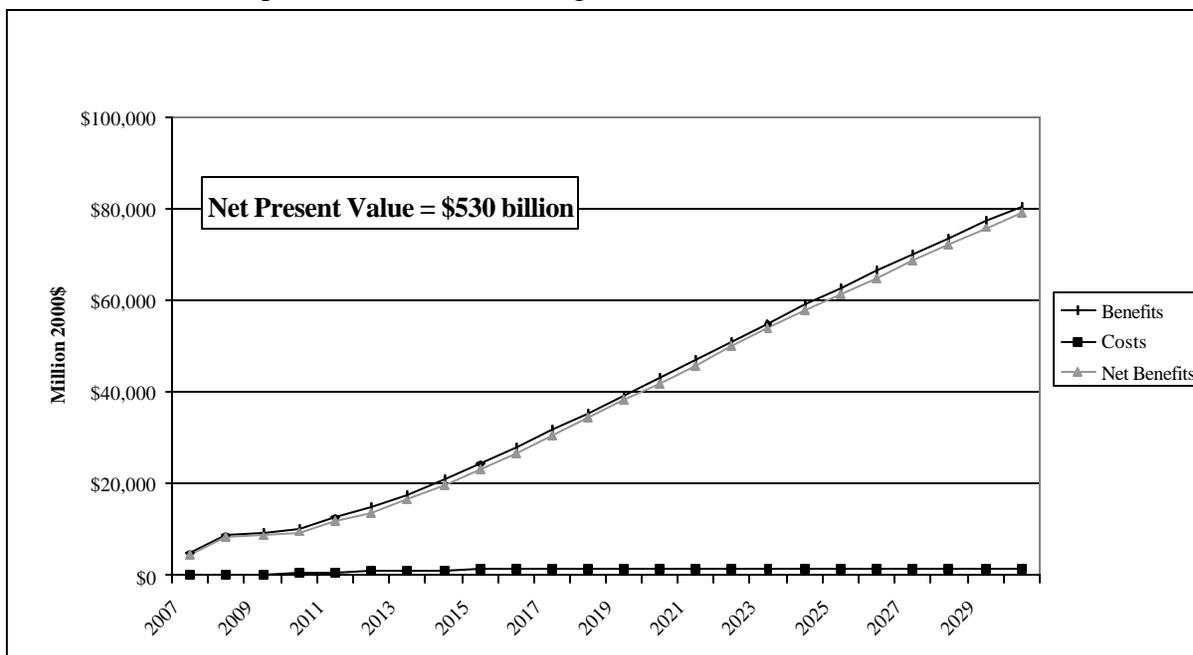


Table 9-18.
Present Value in 2004 of the Stream of
Benefits, Costs, and Net Benefits for the
Proposed Nonroad Diesel Engine and Fuel Standards
(Billions of 2000\$)^a

Social Costs	\$14
Social Benefits	\$550
Net Benefits	\$530

^a Rounded to two significant digits

9.6.1 Potential Impacts of Cost and Emissions Uncertainty

Two key inputs to our benefit-cost analysis are the social costs and emission reductions associated with the proposed program. Each of these elements also has associated uncertainty which contributes to the overall uncertainty in our analysis of benefit-cost.

EPA engineering cost estimates are based upon considerable expertise and experience within the Agency. At the same time, any estimate of the future cost of control technology for engines or the cost of removing sulfur from diesel fuel is inherently uncertain to some degree. At the start is the question of what technology will actually be used to meet future standards, and what such technology will cost at the time of implementation. Our estimates of control costs are based upon current technology plus newer technology already “in the pipeline.” New technology not currently anticipated is by its nature not specifically included. Potential new production techniques which might lower costs are also not included in these estimates (although they are partially included among factors contributing to learning curve effects). On the other side of the equation are unforeseen technical hurdles that may act to increase control system costs.

Some uncertainty is also introduced when translating engineering cost into social cost estimates. Our Economic Impact Assessment presented in Chapter 10 includes sensitivity analyses examining the effect of varying assumptions surrounding the following key factors (Chapter 10, Appendix 10-I):

- market supply and demand elasticity parameters
- alternative assumptions about the fuel market supply shifts and fuel maintenance savings
- alternative assumptions about the engine and equipment market supply shifts

For all of these factors, the change in social cost was projected to be very small, with a maximum impact of less than one percent.

Overall, we have limited means available to develop quantitative estimates of total uncertainty in costs. Some of the factors identified above can act to either increase or decrease actual cost compared to our estimates. Some, such as new technology developments and new production techniques, will act to lower costs compared to our estimates.

One source of a useful information about the overall uncertainty we might expect to see in cost is literature comparing historical rulemaking cost estimates with actual price increases when new standards went into effect.^G Perhaps the most relevant of such studies is the paper by Anderson and Sherwood analyzing these effects for those mobile source rules adopted since the Clean Air Act Amendments of 1990. That paper reviewed six fuel quality rules and ten light-duty vehicle control rules that had been required by those amendments. It found that EPA estimates of the costs for future standards tended to be similar to or higher than actual price changes observed in the market place. Table 9-19 presents a summary of results for the fuel and vehicle rules reviewed in the paper.

^GFor this proposal, we based our cost estimates on information received from industry and technical reports relevant to the US market. We are also aware of two studies done to support nonroad standards development in Europe, namely the VTT report and the EMA/Euromot report. We are not utilizing the cost information in these reports because neither one has sufficient information to allow us to understand or derive the relevant cost figures and therefore provide us information that could be used in trying to estimate cost uncertainty for nonroad diesel engine technologies.

**Table 9-19.
Comparison of Historical EPA Cost Estimates with Actual Price Changes**

EPA Rule	EPA Mid-point Estimate	Actual Price Change	Percent Difference for Price vs EPA
Phase 2 RVP control	1.1 c/gal	0.5 c/gal	-54%
Reformulated Gasoline Phase 1	4.1 c/gal	2.2 c/gal	-46%
Reformulated Gasoline Phase 2	5.7 c/gal	5.1 c/gal	-10%
500ppm Sulfur Highway Diesel Fuel	2.2 c/gal	2.2 c/gal	0%
1994-2001 LDV Regulations	\$446/vehicle	\$347	-22%

The data in Table 9-19 would lead us to believe that cost uncertainty is largely a risk of overestimation by EPA. However, given the uncertainty in constructing the comparison in Anderson and Sherwood plus the increasing sophistication of our cost analyses as time goes on, we believe that a more conservative approach is appropriate. As a sensitivity factor for social cost variability we have chosen to evaluate a range of possible errors in social cost of from twenty percent higher to twenty percent lower than the EPA estimate. The resulting social cost range is shown in Table 9 -20. This uncertainty has virtually no impact on our estimates of the net benefits of the proposed rule, given the large magnitude by which benefits exceed costs.

**Table 9-20.
Estimated Uncertainty for Social Cost of Proposal**

Year	Social Cost Estimate	Uncertainty Range (-20 to +20 percent)
2010	\$0.25 billion	\$0.20 - \$0.30 billion
2020	\$1.1 billion	\$.86 - \$1.3 billion
2030	\$1.2 billion	\$.95 - \$1.4 billion

Turning to the question of emissions uncertainty, the Agency does not at this time have useful quantitative information to bring to bear on this question. For our estimates, we rely on the best information that is available to us. However, there is uncertainty involved in many aspects of emissions estimations. Uncertainty exists in the estimates of emissions from the nonroad sources affected by this proposal, as well as in the universe of other sources included in the emission inventories used for our air quality modeling. To the extent that these other sources are unchanged between our baseline and control case, the impact of uncertainty in those estimates is lessened. Similarly, since the key driver of the benefits of our proposal is the changes produced by the new standards, the effect of uncertainty in the overall estimates of nonroad emissions on our benefits estimates may be lessened.

The main sources of uncertainty in our estimates of nonroad emissions fall in the three areas of population size estimates, equipment usage rates (activity) and engine emission factors. Since nonroad equipment is not subject to state registration and licensing requirements like those applying to highway vehicles, it is difficult to develop precise equipment counts for in-use nonroad equipment. Our modeled equipment populations are derived from related data about sales and scrappage rates. Similarly, annual amount of usage and related load factor information is estimated with some degree of uncertainty. We have access to extensive bodies of data on these areas, but are also aware of the need for improvement. Finally, the emission rates of engines in actual field operation cannot readily be measured at the present time, but are estimated from laboratory testing under a variety of typical operating cycles. While laboratory estimates are a reliable source of emissions data, they cannot fully capture all of the impacts of real in-use operation on emissions, leading to some uncertainty about the results. For further details on our modeling of nonroad emissions, please refer to the discussions in Chapter 3 of this RIA.

We have ongoing efforts in all three of these areas designed to improve their accuracy. Since the opportunity to gather better data exists, we have chosen to focus our main efforts on developing improved estimates rather than on developing elaborate techniques to estimate the uncertainty of current estimates. In the long run, better estimates are the most desired outcome.

One of the most important new tools we are developing is the use of portable emission measurement devices to gather detailed data on actual engines and equipment in daily use. These devices have recently become practical due to advances in computing and sensor technology, and will allow us to generate intensive data defining both activity-related factors (e.g., hours of use, load factors, patterns of use) and in-use emissions data specific to the measured activity and including effects from such things as age and emissions related deterioration. The Agency is pursuing this equipment for improving both its highway and nonroad engine emissions models.

Because of the multiplicity of factors involved, we cannot make a quantitative estimate of the uncertainty in our emissions estimates. In an attempt to estimate the effect of a reasonable amount of uncertainty, we have performed an analysis of the effect of a plus or minus five percent change in the

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amount of emission reduction produced by our proposal. Table 9-21 presents the results of this analysis for 2030 (where the largest effect would be seen).

Table 9-21.
Estimated Effect of Emissions Uncertainty on 2030 Benefits Estimates

Case Examined	Range of 2030 Benefit
-5% - +5% for NO _x	\$78 - \$79 billion
-5% to + 5% for SO ₂	\$78 - \$79 billion
-5% to +5% for PM	\$76 - \$81 billion
-5% to +5% for all emissions	\$75 - \$82 billion

The effect of this analysis shows the final benefit value changing a maximum of the full five percent sensitivity to a value of less than one percent, depending on which pollutant or pollutants were affected. In the real world, each of these three pollutants would not necessarily have the same uncertainty or see errors in the same direction at the same time.

Chapter 9 References

Anderson,J; Sherwood,T; *Comparison of EPA and Other Estimates of Mobile Source Rule Costs to Actual Price Changes*; Society of Automotive Engineers; SAE 2002-01-1980; May 14, 2002.

APPENDIX 9A: Benefits Analysis of Modeled Preliminary Control Option

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9A.4 Benefits Analysis—Results 9-150

This appendix details the models and methods used to generate the benefits estimates from which the benefits of the proposed standards presented in Chapter IX are derived. This analysis uses a methodology generally consistent with benefits analyses performed for the recent analysis of the Heavy Duty Engines/Diesel Fuel rulemaking (U.S. EPA, 2000a) and the proposed Clear Skies Act (U.S. EPA, 2002). The benefits analysis relies on three major modeling components:

- 1) Calculation of the impact that a set of preliminary fuel and engine standards would have on the nationwide inventories for NO_x, non-methane hydrocarbons (NMHC), SO₂, and PM emissions in 2020 and 2030;
- 2) Air quality modeling for 2020 and 2030 to determine changes in ambient concentrations of ozone and particulate matter, reflecting baseline and post-control emissions inventories.
- 3) A benefits analysis to determine the changes in human health and welfare, both in terms of physical effects and monetary value, that result from the projected changes in ambient concentrations of various pollutants for the modeled standards.

Figure 9A.1 illustrates the major steps in the analysis. Given baseline and post-control emissions inventories for the emission species expected to impact ambient air quality, we use sophisticated photochemical air quality models to estimate baseline and post-control ambient concentrations of ozone and PM, and deposition of nitrogen and sulfur for each year. The estimated changes in ambient concentrations are then combined with monitoring data to estimate population level exposures to changes in ambient concentrations for use in estimating health effects. Modeled changes in ambient data are also used to estimate changes in visibility, and changes in other air quality statistics that are necessary to estimate welfare effects. Changes in population exposure to ambient air pollution are then input to concentration-response functions to generate changes in incidence of health effects, or, changes in other exposure metrics are input to dose-response functions to generate changes in welfare effects. The resulting effects changes are then assigned monetary values, taking into account adjustments to values for growth in real income out to the year of analysis (values for health and welfare effects are in general positively related to real income levels). Finally, values for individual health and welfare effects are summed to obtain an estimate of the total monetary value of the changes in emissions.

On September 26, 2002, the National Academy of Sciences (NAS) released a report on its review of the Agency's methodology for analyzing the health benefits of measures taken to reduce air pollution. The report focused on EPA's approach for estimating the health benefits of regulations designed to reduce concentrations of airborne particulate matter (PM).

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In its report, the NAS said that EPA has generally used a reasonable framework for analyzing the health benefits of PM-control measures. It recommended, however, that the Agency take a number of steps to improve its benefits analysis. In particular, the NAS stated that the Agency should:

- include benefits estimates for a range of regulatory options;
- estimate benefits for intervals, such as every five years, rather than a single year;
- clearly state the projected baseline statistics used in estimating health benefits, including those for air emissions, air quality, and health outcomes;
- examine whether implementation of proposed regulations might cause unintended impacts on human health or the environment;
- when appropriate, use data from non-U.S. studies to broaden age ranges to which current estimates apply and to include more types of relevant health outcomes;
- begin to move the assessment of uncertainties from its ancillary analyses into its base analyses by conducting probabilistic, multiple-source uncertainty analyses. This assessment should be based on available data and expert judgment.

Although the NAS made a number of recommendations for improvement in EPA's approach, it found that the studies selected by EPA for use in its benefits analysis were generally reasonable choices. In particular, the NAS agreed with EPA's decision to use cohort studies to derive benefits estimates. It also concluded that the Agency's selection of the American Cancer Society (ACS) study for the evaluation of PM-related premature mortality was reasonable, although it noted the publication of new cohort studies that should be evaluated by the Agency.

EPA has addressed many of the NAS comments in our analysis of the proposed rule. We provide benefits estimates for each year over the rule implementation period for a wide range of regulatory alternatives, in addition to our proposed emission control program. We use the estimated time path of benefits and costs to calculate the net present value of benefits of the rule. In the RIA, we provide baseline statistics for air emissions, air quality, population, and health outcomes. We have examined how our benefits estimates might be impacted by expanding the age ranges to which epidemiological studies are applied, and we have added several new health endpoints, including non-fatal heart attacks, which are supported by both U.S. studies and studies conducted in Europe. We have also improved the documentation of our methods and provided additional details about model assumptions.

Several of the NAS recommendations addressed the issue of uncertainty and how the Agency can better analyze and communicate the uncertainties associated with its benefits assessments. In particular, the Committee expressed concern about the Agency's reliance on a single value from its analysis and suggested that EPA develop a probabilistic approach for analyzing the health benefits of proposed regulatory actions. The Agency agrees with this suggestion and is working to develop such an approach for use in future rulemakings. EPA plans to hold a meeting of its Science Advisory Board (SAB) in early Summer 2003 to review its plans for addressing uncertainty in its analyses. Our likely

approach will incorporate short-term elements intended to provide interim methods in time for the final Nonroad rule to address uncertainty in important analytical parameters such as the concentration-response relationship for PM-related premature mortality. Our approach will also include longer-term elements intended to provide scientifically sound, peer-reviewed characterizations of the uncertainty surrounding a broader set of analytical parameters and assumptions, including but not limited to emissions and air quality modeling, demographic projections, population health status, concentration-response functions, and valuation estimates.

Our primary approach, generating our Base Estimate is a peer-reviewed method developed for previous risk and benefit-cost assessments carried out by the Environmental Protection Agency. It is the method used in the regulatory assessments of the Heavy Duty Diesel and Tier II (light duty engine) Rules and the Section 812 Report to Congress. Following the approach of these earlier assessments, along with the results of the Base Estimate, we present various sensitivity analyses on the Base Estimate that alter select subsets of variables, such as the concentration-response function for premature mortality.

Many of the techniques applied in analyzing the benefits of the proposed rule have also been reviewed by EPA's independent Science Advisory Board (SAB). We have relied heavily on the advice of the SAB in determining the health and welfare effects considered in the benefits analysis and in establishing the most scientifically valid measurement and valuation techniques. Since the publication of the final HD Engine/Diesel Fuel RIA, we have updated some of the assumptions and methods used in our analysis to reflect SAB and NAS recommendations, as well as advances in data and methods in air quality modeling, epidemiology, and economics. Changes to the methodology are described fully in the following sections and in the benefits technical support document (Abt Associates, 2003) and include the following:

- Demographic/population data:
 - We have updated our base population data from 1990 to Census 2000 block level data
 - We have developed future year population projections based on Woods and Poole Economics, Inc. 2001 Regional Projections of county population.
- Health effects incidence/prevalence data:
 - We have updated county-level mortality rates (all-cause, non-accidental, cardiopulmonary, lung cancer, COPD) from 1994-1996 to 1996-1998 using the CDC Wonder database.
 - We have updated hospitalization rates from 1994 to 1999 and switched from national rates to regional rates using 1999 National Hospital Discharge Survey results.
 - We have developed regional emergency room visit rates using results of the 2000 National Hospital Ambulatory Medical Care Survey.
 - We have updated prevalence of asthma and chronic bronchitis to 1999 using results of the National Health Interview Survey (HIS), as reported by the American Lung Association (ALA), 2002

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- We have developed non-fatal heart attack incidence rates based on National Hospital Discharge Survey results.
- We have updated the national acute bronchitis incidence rate using HIS data as reported in ALA, 2002, Table 11.
- We have updated the work loss days rate using the 1996 HIS data, as reported in Adams, et al. 1999, Table 41
- We have developed school absence rates using data from the National Center for Education Statistics and the 1996 HIS, as reported in Adams, et al., 1999, Table 46.
- We have developed baseline incidence rates for respiratory symptoms in asthmatics, 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
 - We have added several new endpoints to the analysis, including
 - > hospital admissions for all cardiovascular causes in adults 20-64, PM (Moolgavkar et al., 2000)
 - > 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)
 - > hospital admissions for all respiratory causes in children under 2, Ozone (Burnett et al., 2001)
 - We have changed the sources for concentration-response functions for hospital admission for pneumonia, COPD, and total cardiovascular from Samet et al, 2000 (a PM₁₀ study), to Lippmann et al, 2000 and Moolgavkar, 2000 (PM_{2.5} studies)
 - We have added a separate table with incidence estimates for the asthmatic subpopulation, 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.
 - We have added 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, i.e. extending lower respiratory symptoms to all children, rather than only children aged 7-14.

- Valuation of Changes in Health Outcomes:
 - We have developed a value for school absence days 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.
 - We have developed age-specific values for non-fatal heart attacks 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.

- We have corrected a previous error in the valuation of acute bronchitis episodes. 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:
 - PM air quality modeling results are used to develop adjustment factors which will be 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.
 - We have changed the ozone air quality model from the Urban Airshed Model to CAM-X, modeled using 30 episode days in 1995 for the Eastern U.S. and 19 episode days in 1996 for the Western U.S. (note that in the HD Engine/Diesel Fuel analysis, we did not use ozone modeling results for the Western U.S.). For both Eastern and Western domains, a nested grid structure was used, with a 36 km outer resolution, and a 12 km inner resolution over urban areas.
 - We have updated the PM air quality model, REMSAD, to version 7.3, run at 36 km grid resolution.

In addition to the above changes, for the proposed rule, the Agency has used an interim approach that shows the impact of several important alternative assumptions about the estimation and valuation of reductions in premature mortality and chronic bronchitis. This approach, which was developed in the context of the Agency's Clear Skies analysis, provides an alternative estimate of health benefits using the time series studies in place of cohort studies, as well as alternative valuation methods for mortality and chronic bronchitis risk reductions.

All such benefit estimates are subject to a number of assumptions and uncertainties, which are discussed throughout the appendix. 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 directly emitted from diesel engines are chemically different from fine particles resulting from both utility sources and industrial facilities, but no clear scientific grounds exist for supporting differential effects estimates by particle type. (3) The concentration-response 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

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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 are not currently amenable to quantification or valuation. These include reduced acid and particulate deposition damage to cultural monuments and other materials; reduced ozone effects on forested ecosystems; and environmental benefits due to reductions of impacts of acidification in lakes and streams and 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 forced expiratory volume (FEV1)). As a result, both the Base and Alternative monetized benefits estimates underestimate the total benefits attributable to the preliminary control options.

In general, the chapter is organized around the steps illustrated in Figure 9A.1. In section A, we describe and summarize the emissions inventories and modeled reductions in emissions of NO_x, VOC, SO₂, and directly emitted diesel PM for the set of preliminary control options. In section B, we describe and summarize the air quality models and results, including both baseline and post-control conditions, and discuss the way modeled air quality changes are used in the benefits analysis. In Section C, we provide an overview of the data and methods that are used to quantify and value health and welfare endpoints, and provide a discussion of how we incorporate uncertainty into our analysis. In Section D, we report the results of the analysis for human health and welfare effects. Additional sensitivity analyses are provided in Appendix 9B.

**Table 9A.1. Summary of Results: Estimated Benefits
of the Modeled Preliminary Control Option**

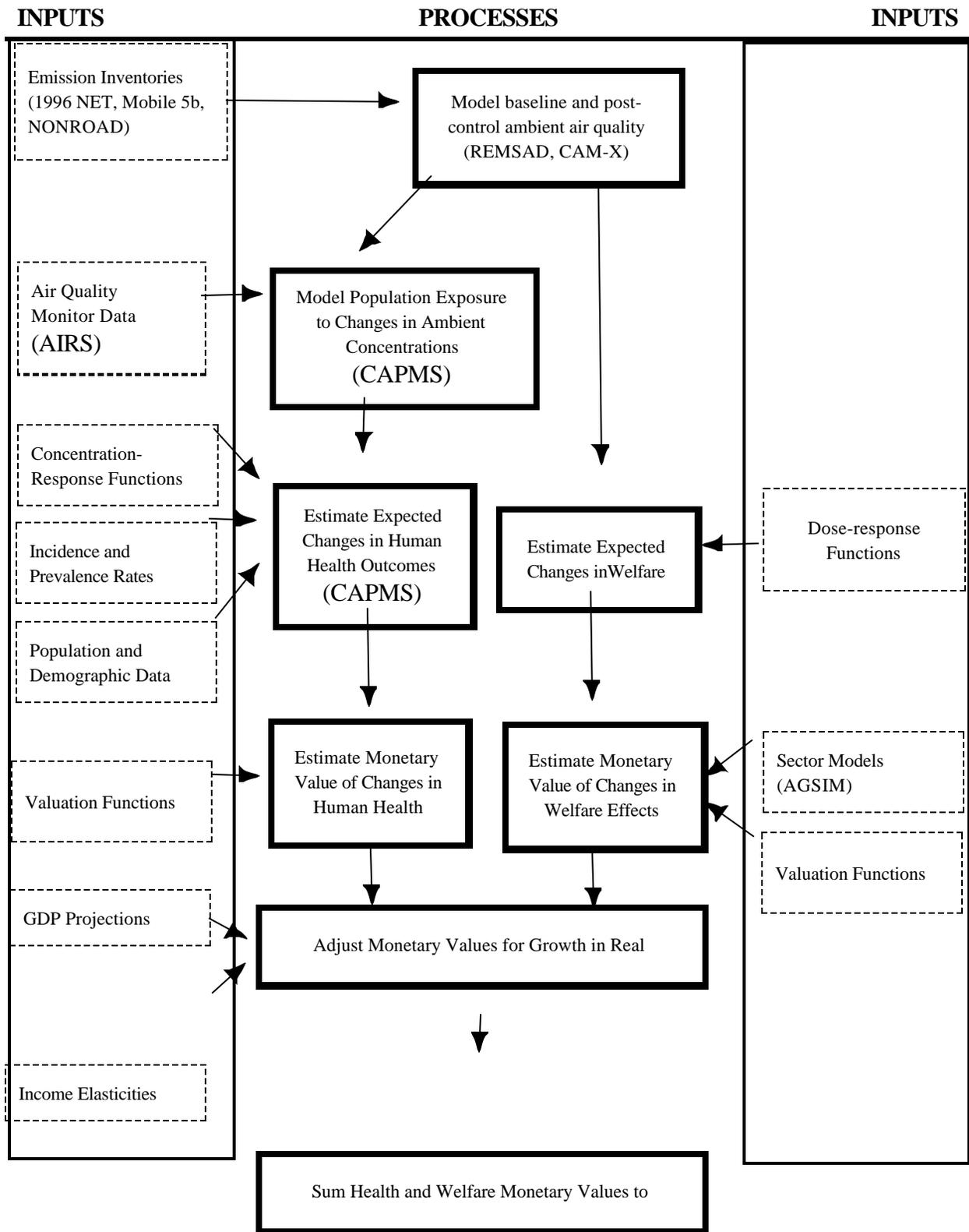
Estimation Method	Total Benefits ^{A, B} (Billions 2000\$)	
	2020	2030
Base Estimate:		
Using a 3% discount rate	\$52+B	\$92+B
Using a 7% discount rate	\$49+B	\$87+B
Alternative Estimate:		
Using a 3% discount rate	\$8.3+B	\$14+B
Using a 7% discount rate	\$8.8+B	\$15+B

^A Benefits of CO and HAP emission reductions are not quantified in this analysis and, therefore, are not presented in this table. The quantifiable benefits are from emission reductions of NOX, NMHC, SO₂ and PM only. For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. A detailed listing of unquantified health and welfare effects is provided in Table 9A-2.

^B Results reflect the use of two different discount rates; a 3% rate which is recommended by EPA's Guidelines for Preparing Economic Analyses (US EPA, 2000c), and 7% which is recommended by OMB Circular A-94 (OMB, 1992). Results are rounded to two significant digits.

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Figure 9A.1. Key Steps in Air Quality Modeling Based Benefits Analysis



**Table 9A.2.
Human Health and Welfare Effects of Pollutants Affected by the Proposed Nonroad Diesel Engine Rule**

Pollutant/Effect	Quantified and Monetized in Base and Alternative Estimates^A	Quantified and/or Monetized Effects in Sensitivity Analyses^B	Unquantified Effects
Ozone/Health	Hospital admissions - respiratory Emergency room visits for asthma Minor restricted activity days School loss days	Chronic Asthma ^C Asthma attacks Cardiovascular emergency room visits Premature mortality – acute exposures ^D Acute respiratory symptoms	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
Ozone/Welfare	Decreased outdoor worker productivity Decreased yields for commercial crops (selected species) Decreased Eastern commercial forest productivity (selected species)		Decreased Western commercial forest productivity Decreased Eastern commercial forest productivity (other species) Decreased yields for fruits and vegetables Decreased yields for other commercial and non-commercial crops Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Damage to ecosystem functions
PM/Health	Premature mortality – long 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	Premature mortality – short term exposures Asthma attacks (asthmatic population) 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

Pollutant/Effect	Quantified and Monetized in Base and Alternative Estimates ^A	Quantified and/or Monetized Effects in Sensitivity Analyses ^B	Unquantified Effects
PM/Welfare	Visibility in California, Southwestern, and Southeastern Class I areas	Visibility in Northeastern, Northwestern, and Midwestern Class I areas Visibility in residential and non-Class I areas Household soiling	
Nitrogen and Sulfate Deposition/Welfare		Costs of nitrogen controls to reduce eutrophication in selected eastern estuaries	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
SO₂/Health			Hospital admissions for respiratory and cardiac diseases Respiratory symptoms in asthmatics
NOX/Health			Lung irritation Lowered resistance to respiratory infection Hospital Admissions for respiratory and cardiac diseases
CO/Health			Premature mortality Behavioral effects Hospital admissions - respiratory, cardiovascular, and other Other cardiovascular effects Developmental effects Decreased time to onset of angina Non-asthma respiratory ER visits

Pollutant/Effect	Quantified and Monetized in Base and Alternative Estimates ^A	Quantified and/or Monetized Effects in Sensitivity Analyses ^B	Unquantified Effects
NMHCs^E Health			Cancer (diesel PM, benzene, 1,3-butadiene, formaldehyde, acetaldehyde) Anemia (benzene) Disruption of production of blood components (benzene) Reduction in the number of blood platelets (benzene) Excessive bone marrow formation (benzene) Depression of lymphocyte counts (benzene) Reproductive and developmental effects (1,3-butadiene) Irritation of eyes and mucous membranes (formaldehyde) Respiratory and respiratory tract Asthma attacks in asthmatics (formaldehyde) Asthma-like symptoms in non-asthmatics (formaldehyde) Irritation of the eyes, skin, and respiratory tract (acetaldehyde) Upper respiratory tract irritation & congestion (acrolein)
NMHCs^E Welfare			Direct toxic effects to animals Bioaccumulation in the food chain Reduction in odors

^A Primary quantified and monetized effects are those included when determining the primary estimate of total monetized benefits of the Noroad Diesel Engine rule. See Section C-2 for a more complete discussion of presentation of benefits estimates.

^B Alternative quantified and/or monetized effects are those presented as alternatives to the primary estimates or in addition to the primary estimates, but not included in the primary estimate of total monetized benefits.

^C 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).

^D Premature mortality associated with ozone is not separately included in the primary analysis. It is assumed that the American Cancer Society (ACS)/ Krewski, et al., 2000 C-R function we use for premature mortality captures both PM mortality benefits and any mortality benefits associated with other air pollutants (ACS/ Krewski, et al., 2000).

^E All non-methane hydrocarbons (NMHCs) listed in the table are also hazardous air pollutants listed in the Clean Air Act.

9A.1 Summary of Emissions Inventories and Modeled Changes in Emissions from Nonroad Engines

For the preliminary control options we modeled, implementation will occur in two stages: reduction in sulfur content of nonroad diesel fuel and adoption of controls on new engines. Because full turnover of the fleet of nonroad diesel engines will not occur for many years, the emission reduction benefits of the proposed standards will not be fully realized until decades after the initial reduction in fuel sulfur content. Based on the projected time paths for emissions reductions, EPA chose to focus detailed emissions and air quality modeling on two future years, 2020 and 2030, which reflect partial and close to complete turnover of the fleet of nonroad diesel engines to models meeting the preliminary control options. Tables 9A-3 and 9A-4 summarize the baseline emissions of NO_x, SO₂, VOC, and direct diesel PM_{2.5} and the change in the emissions from nonroad engines used in modeling air quality changes.

Emissions and air quality modeling decisions are made early in the analytical process. Since the preliminary control scenario was developed, EPA has gathered more information regarding the technical feasibility of the standards, and has revised the control scenario. Section 3.6 of the RIA describes the changes in the inputs and resulting emission inventories between the preliminary baseline and control scenarios used for the air quality modeling and the proposed baseline and control scenarios.

Chapter 3 discussed the development of the 1996, 2020 and 2030 baseline emissions inventories for the nonroad sector and for the sectors not affected by this proposed rule. The emission sources and the basis for current and future-year inventories are listed in Table 9A-5.

**Table 9A-3
Summary of Baseline Emissions for Preliminary Nonroad Engine Control Options**

Source	Pollutant Emissions (tons)			
	NO _x	SO ₂	VOC	PM _{2.5}
1996 Baseline				
Nonroad Engines	1,583,641	172,175	221,398	178,500
All Other Sources	22,974,945	18,251,679	18,377,795	2,038,726
Total, All Sources	24,558,586	18,423,854	18,599,193	2,217,226
2020 Base Case				
Nonroad Engines	1,144,686	308,075	97,113	127,755
All Other Sources	14,394,399	14,882,962	13,812,619	1,940,307
Total, All Sources	15,539,085	15,191,037	13,909,732	2,068,062
2030 Base Case				
Nonroad Engines	1,231,981	360,933	97,345	143,185
All Other Sources	14,316,841	15,190,439	15,310,670	2,066,918
Total, All Sources	15,548,822	15,551,372	15,408,015	2,210,103

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**Table 9A-4
Summary of Emissions Changes for the Preliminary Nonroad Control Options***

Item	Pollutant			
	NO _x	SO ₂	VOC	PM _{2.5}
2020 Nationwide Emission Changes				
Absolute Tons	663,618	304,735	23,172	91,278
Percent Reduction from Landbased Nonroad Emissions	58.0%	98.9%	23.9%	71.4%
Percentage Reduction from All Manmade Sources	4.5%	2.1%	0.2%	4.6%
2030 Emission Changes				
Absolute Tons	1,009,744	359,774	34,060	129,073
Percent Reduction from Landbased Nonroad Emissions	82.0%	99.7%	35.0%	90.0%
Percentage Reduction from All Manmade Sources	6.3%	2.1%	0.2%	5.5%

* Does not include SO_x and PM_{2.5} reductions from recreational marine diesel engines, commercial marine diesel engines, and locomotives due to control of diesel fuel sulfur levels.

Table 9A-5
Emissions Sources and Basis for Current and Future-Year Inventories

Emissions Source	1996 Base year	Future-year Base Case Projections
Utilities	1996 NEI Version 3.12 (CEM data)	Integrated Planning Model (IPM)
Non-Utility Point and Area sources	1996 NEI Version 3.12 (point) Version 3.11 (area)	BEA growth projections
Highway vehicles	MOBILE5b model with MOBILE6 adjustment factors for VOC and NOX; PART5 model for PM	VMT projection data
Nonroad engines (except locomotives, commercial marine vessels, and aircraft)	NONROAD2002 model	BEA and Nonroad equipment growth projections

Note: Full description of data, models, and methods applied for emissions inventory development and modeling are provided in Emissions Inventory TSD (EPA, 2003a).

9A.2 Air Quality Impacts

This section summarizes the methods for and results of estimating air quality for the 2020 and 2030 base cases and control scenarios for the purposes of benefit-cost analyses. EPA has focused on the health, welfare, and ecological effects that have been linked to air quality changes. These air quality changes include the following:

- Ambient particulate matter (PM₁₀ and PM_{2.5})—as estimated using a national-scale version of the REgional Modeling System for Aerosols and Deposition (REMSAD);
- Ambient ozone—as estimated using regional-scale applications of the Comprehensive Air Quality Model with Extensions (CAMx); and
- Visibility degradation (i.e., regional haze), as developed using empirical estimates of light extinction coefficients and efficiencies in combination with REMSAD modeled reductions in pollutant concentrations.

Although we expect reductions in airborne sulfur and nitrogen deposition, these air quality impacts have not been quantified for this proposed rule nor have the associated benefits been estimated.

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The air quality estimates in this section are based on the emission changes for the modeled preliminary control program discussed in Chapter 3. These air quality results are in turn associated with human populations and ecosystems to estimate changes in health and welfare effects. In Section B-1, we describe the estimation of PM air quality using REMSAD, and in Section B-2, we cover the estimation of ozone air quality using CAMx. Lastly, in Section B-3, we discuss the estimation of visibility degradation.

9A.2.1 PM Air Quality Estimates

We use the emissions inputs summarized above with a national-scale version of the Regional Model System for Aerosols and Deposition (REMSAD) to estimate PM air quality in the contiguous U.S. REMSAD is a three-dimensional grid-based Eulerian air quality model designed to estimate annual particulate concentrations and deposition over large spatial scales (e.g., over the contiguous U.S.). Consideration of the different processes that affect primary (directly emitted) and secondary (formed by atmospheric processes) PM at the regional scale in different locations is fundamental to understanding and assessing the effects of proposed pollution control measures that affect ozone, PM and deposition of pollutants to the surface.^a Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is useful for evaluating the impacts of the proposed rule on U.S. PM concentrations.

REMSAD was peer-reviewed in 1999 for EPA as reported in “*Scientific Peer-Review of the Regulatory Modeling System for Aerosols and Deposition*” (Seigneur et al., 1999). Earlier versions of REMSAD have been employed for the EPA’s Prospective 812 Report to Congress, EPA’s HD Engine/Diesel Fuel rule, and EPA’s air quality assessment of the Clear Skies Initiative. Version 7 of REMSAD was employed for this analysis and is fully described in the air quality modeling technical support document (US EPA, 2003b). This version reflects updates in the following areas to improve performance and address comments from the 1999 peer-review:

- Gas phase chemistry updates to “micro-CB4” mechanism including new treatment for the NO₃ and N₂O₅ 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.
- 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.

^A Given the potential impact of the Nonroad Engine/Diesel Fuel rule on secondarily formed particles it is important to employ a Eulerian model such as REMSAD. The impact of secondarily formed pollutants typically involves primary precursor emissions from a multitude of widely dispersed sources, and chemical and physical processes of pollutants that are best addressed using an air quality model that employs an Eulerian grid model design.

- Aqueous phase chemistry updates to incorporate the oxidation of SO₂ by O₃ and O₂ and to include the cloud and rain liquid water content from MM5 meteorological data directly in sulfate production and deposition calculations.

As discussed earlier in Chapter 2, the model tends to underestimate observed PM_{2.5} concentrations nationwide, especially over the western U.S.

Our analysis applies the modeling system to the entire U.S. for the five emissions scenarios: a 1996 baseline projection, a 2020 baseline projection and a 2020 projection with nonroad controls, a 2030 baseline projection and a 2030 projection with nonroad controls. As discussed in the Benefits Analysis TSD, we use the relative predictions from the model by combining the 1996 base-year and each future-year scenario with ambient air quality observations to determine the expected change in 2020 or 2030 ozone concentrations due to the rule (Abt Associates, 2003). These results are used solely in the benefits analysis.

REMSAD simulates every hour of every day of the year and, thus, requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, 1-hour average emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. As applied to the contiguous U.S., the model segments the area within the region into square blocks called grids (roughly equal in size to counties), each of which has several layers of air conditions. Using this data, REMSAD generates predictions of 1-hour average PM concentrations for every grid. We then calibrate the modeling results to develop 2020 and 2030 PM estimates at monitor sites by normalizing the observations to the observed 1996 concentrations at each monitor site. For areas (grids) without PM monitoring data, we interpolated concentration values using data from monitors surrounding the area. After completing this process, we then calculated daily and seasonal PM air quality metrics as inputs to the health and welfare C-R functions of the benefits analysis. The following sections provide a more detailed discussion of each of the steps in this evaluation and a summary of the results.

9A.2.1.1 Modeling Domain

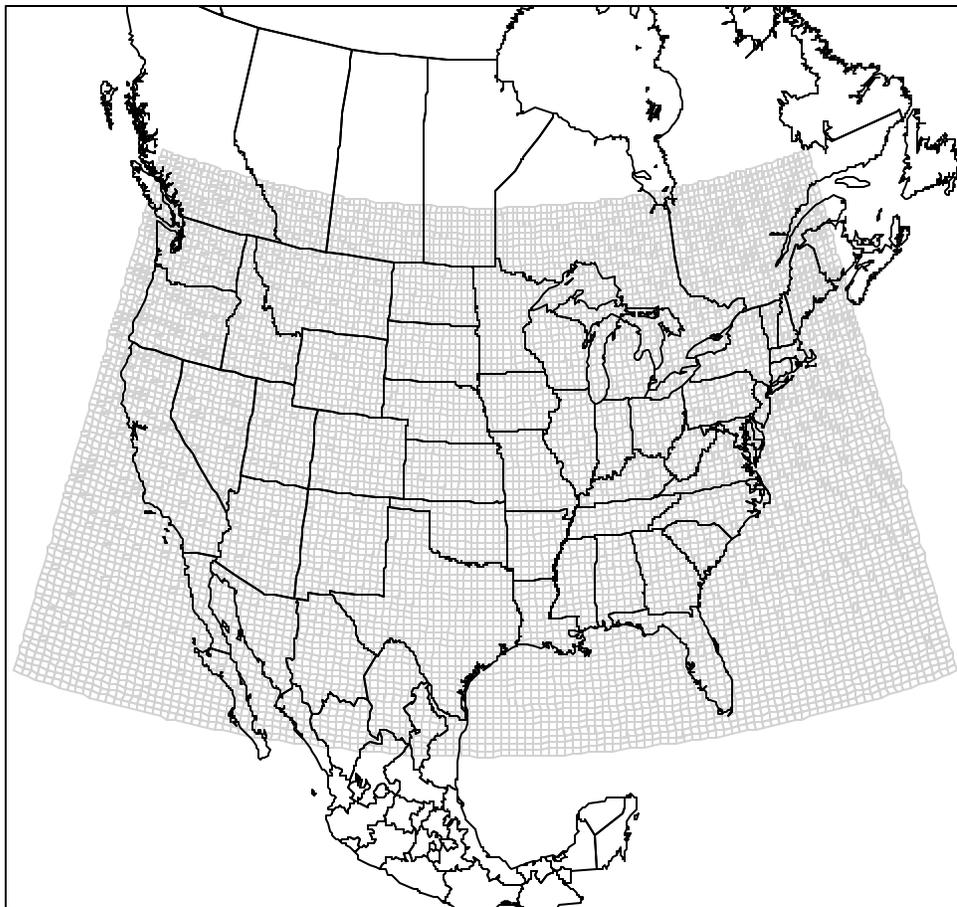
The PM air quality analyses employed the modeling domain used previously in support of Clear Skies air quality assessment. As shown in Figure 9A-2, the modeling domain encompasses the lower 48 States and extends from 126 degrees to 66 degrees west longitude and from 24 degrees north latitude to 52 degrees north latitude. The model contains horizontal grid-cells across the model domain of roughly 36 km by 36 km. There are 12 vertical layers of atmospheric conditions with the top of the modeling domain at 16,200 meters. The 36 by 36 km horizontal grid results in a 120 by 84 grid (or 10,080 grid-cells) for each vertical layer. Figure 9A-3 illustrates the horizontal grid-cells for Maryland and surrounding areas.

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9A.2.1.2 Simulation Periods

For use in this benefits analysis, the simulation periods modeled by REMSAD included separate full-year application for each of the five emissions scenarios as described in Chapter 3, i.e., 1996 baseline and the 2020 and 2030 base cases and control scenarios.

Figure 9A-2
REMSAD Modeling Domain for Continental United States



Note: Gray markings define individual grid-cells in the REMSAD model.

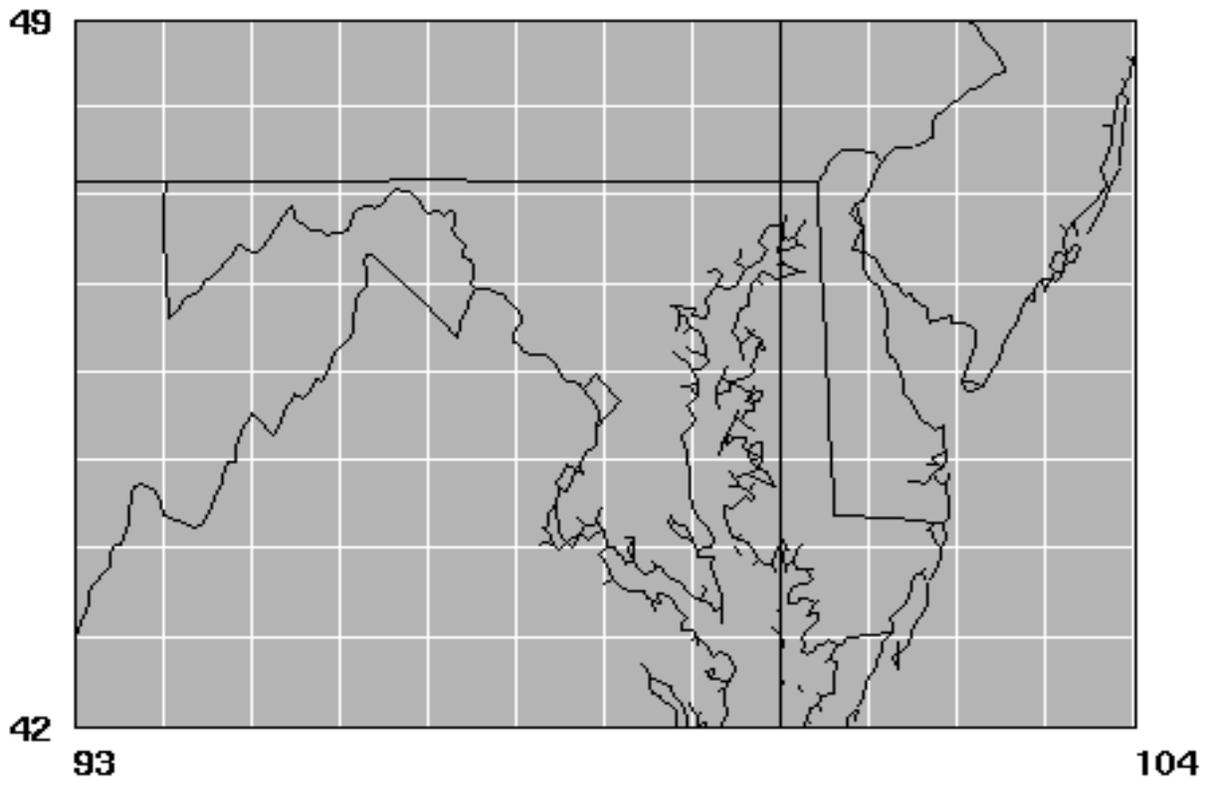


Figure 9A-3. Example of REMSAD 36 x 36km Grid-cells for Maryland Area

9A.2.1.3 Model Inputs

REMSAD requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, 1-hour average emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. Separate emissions inventories were prepared for the 1996 baseline and each of the future-year base cases and control scenarios. All other inputs were specified for the 1996 baseline model application and remained unchanged for each future-year modeling scenario.

Similar to CAMx, REMSAD requires detailed emissions inventories containing temporally allocated emissions for each grid-cell in the modeling domain for each species being simulated. The previously described annual emission inventories were preprocessed into model-ready inputs through the SMOKE emissions preprocessing system. Details of the preprocessing of emissions through SMOKE as provided in the emissions modeling TSD. Meteorological inputs reflecting 1996 conditions across the contiguous U.S. were derived from Version 5 of the Mesoscale Model (MM5). These inputs included horizontal wind components (i.e., speed and direction), temperature, moisture, vertical diffusion rates, and rainfall rates for each grid cell in each vertical layer. Details of the annual 1996 MM5 modeling are provided in Olerud (2000).

Initial species concentrations and lateral boundary conditions were specified to approximate background concentrations of the species; for the lateral boundaries the concentrations varied (decreased parabolically) with height. These background concentrations are provided in the air quality modeling TSD (U.S. EPA, 2003b). Land use information was obtained from the U.S. Geological Survey database at 10 km resolution and aggregated to the ~36 KM horizontal resolution used for this REMSAD application.

9A.2.1.4 Converting REMSAD Outputs to Benefits Inputs

REMSAD generates predictions of hourly PM concentrations for every grid. The particulate matter species modeled by REMSAD include a primary coarse fraction (corresponding to PM in the 2.5 to 10 micron size range), a primary fine fraction (corresponding to PM less than 2.5 microns in diameter), and several secondary particles (e.g., sulfates, nitrates, and organics). $PM_{2.5}$ is calculated as the sum of the primary fine fraction and all of the secondarily-formed particles. These hourly predictions for each REMSAD grid-cell are aggregated to daily averages and used in conjunction with observed PM concentrations from AIRS to generate the predicted changes in the daily and annual PM air quality metrics (i.e., annual mean PM concentration) from the future-year base case to future-year control scenario as inputs to the health and welfare C-R functions of the benefits analysis.^b In addition,

^bBased on AIRS, there were 1,071 FRM PM monitors with valid data as defined as more than 11 observations per season.

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the speciated predictions from REMSAD are employed as inputs to a post-processing module that estimates atmospheric visibility, as discussed later in Section 9A.3.

In order to estimate PM-related health and welfare effects for the contiguous U.S., daily and annual average PM concentrations are required for every location. Given available PM monitoring data, we generated an annual profile for each location in the contiguous 48 States in two steps: (1) we combine monitored observations and modeled PM predictions to interpolate forecasted daily PM concentrations for each REMSAD grid-cell, and (2) we compute the daily and annual PM measures of interest based on the annual PM profiles.^c These methods are described in detail in the benefits analysis technical support document (Abt Associates, 2003).

9A.2.1.5 PM Air Quality Results

Table 9A-5 provides a summary of the predicted ambient PM_{2.5} concentrations for the 2020 and 2030 base cases and changes associated with Nonroad Engine/Diesel Fuel control scenarios. The REMSAD results indicate that the predicted change in PM concentrations is composed almost entirely of reductions in fine particulates (PM_{2.5}) with little or no reduction in coarse particles (PM₁₀ less PM_{2.5}). Therefore, the observed changes in PM₁₀ are composed primarily of changes in PM_{2.5}. In addition to the standard frequency statistics (e.g., minimum, maximum, average, median), Table 9A-5 provides the population-weighted average which better reflects the baseline levels and predicted changes for more populated areas of the nation. This measure, therefore, will better reflect the potential benefits of these predicted changes through exposure changes to these populations. As shown, the average annual mean concentrations of PM_{2.5} across all U.S. grid-cells declines by roughly 2.5 percent (or 0.2 µg/m³) and 3.4 percent (or 0.28 µg/m³) in 2020 and 2030, respectively. The population-weighted average mean concentration declined by 3.3 percent (or 0.42 µg/m³) in 2020 and 4.5 percent (or 0.59 µg/m³) in 2030, which is much larger in absolute terms than the spatial average for both years. This indicates the proposed rule generates greater absolute air quality improvements in more populated, urban areas.

^cThis approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (EVNA) spatial interpolation (See Abt Associates (2003) for a more detailed description).

**Table 9A-6.
Summary of Base Case PM Air Quality
and Changes Due to Nonroad Engine/Diesel Fuel Standards: 2020 and 2030**

<i>Statistic</i>	<i>2020</i>			<i>2030</i>		
	<i>Base Case</i>	<i>Change^a</i>	<i>Percent Change</i>	<i>Base Case</i>	<i>Change^a</i>	<i>Percent Change</i>
PM_{2.5} (µg/m³)						
Minimum Annual Mean ^b	2.18	-0.02	-0.78%	2.33	-0.02	-1.01%
Maximum Annual Mean ^b	29.85	-1.36	-4.56%	32.85	-2.03	-6.18%
Average Annual Mean	8.10	-0.20	-2.49%	8.37	-0.28	-3.38%
Median Annual Mean	7.50	-0.18	-2.68%	7.71	-0.22	-2.80%
Pop-Weighted Average Annual Mean ^c	12.42	-0.42	-3.34%	13.07	-0.59	-4.48%

^a The change is defined as the control case value minus the base case value.

^b The base case minimum (maximum) is the value for the populated grid-cell with the lowest (highest) annual average. The change relative to the base case is the observed change for the populated grid-cell with the lowest (highest) annual average in the base case.

^c Calculated by summing the product of the projected REMSAD grid-cell population and the estimated PM concentration, for that grid-cell and then dividing by the total population in the 48 contiguous States.

Table 9A-6 provides information on the populations in 2020 and 2030 that will experience improved PM air quality. There are significant populations that live in areas with meaningful reductions in annual mean PM_{2.5} concentrations resulting from the proposed rule. As shown, almost 10 percent of the 2030 U.S. population are predicted to experience reductions of greater than 1 µg/m³. This is an increase from the 2.7 percent of the U.S. population that are expected to experience such reductions in 2020. Furthermore, just over 20 percent of the 2030 U.S. population will benefit from reductions in annual mean PM_{2.5} concentrations of greater than 0.75 µg/m³ and slightly over 50 percent will live in areas with reductions of greater than 0.5 µg/m³. This information indicates how widespread the improvements in PM air quality are expected to be and the large populations that will benefit from these improvements.

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Table 9A-7
Distribution of PM_{2.5} Air Quality Improvements Over Population Due to Nonroad Engine/Diesel Fuel Standards: 2020 and 2030

Change in Annual Mean PM _{2.5} Concentrations (µg/m ³)	2020 Population		2030 Population	
	Number (millions)	Percent (%)	Number (millions)	Percent (%)
0 >) PM _{2.5} Conc # 0.25	65.11	19.75%	28.60	8.04%
0.25 >) PM _{2.5} Conc # 0.5	184.52	55.97%	147.09	41.33%
0.5 >) PM _{2.5} Conc # 0.75	56.66	17.19%	107.47	30.20%
0.75 >) PM _{2.5} Conc # 1.0	14.60	4.43%	38.50	10.82%
1.0 >) PM _{2.5} Conc # 1.25	5.29	1.60%	88.22	2.48%
1.25 >) PM _{2.5} Conc # 1.5	3.51	1.06%	15.52	4.36%
1.5 >) PM _{2.5} Conc # 1.75	0	0.00%	5.70	1.60%
) PM _{2.5} Conc > 1.75	0	0.00%	4.19	1.18%

^a The change is defined as the control case value minus the base case value.

Table 9A-7 provides additional insights on the changes in PM air quality resulting from the proposed standards. The information presented previously in Table 9A-5 illustrated the absolute and relative changes for different points along the distribution of baseline 2020 and 2030 PM_{2.5} concentration levels, e.g., the change reflects the lowering of the minimum predicted baseline concentration rather than the minimum predicted change for 2020 and 2030. The latter is the focus of Table 9A-7 as it presents the distribution of predicted changes in both absolute terms (i.e., µg/m³) and relative terms (i.e., percent) across individual REMSAD grid-cells. Therefore, it provides more information on the range of predicted changes associated with the proposed rule. As shown for 2020, the absolute reduction in annual mean PM_{2.5} concentration ranged from a low of 0.02 µg/m³ to a high of 1.36 µg/m³, while the relative reduction ranged from a low of 0.3 percent to a high of 12.2 percent. Alternatively, for 2030, the absolute reduction ranged from 0.02 to 2.03 µg/m³, while the relative reduction ranged from 0.4 to 15.5 percent.

Table 9A-8.
Summary of Absolute and Relative Changes in PM Air Quality Due to Nonroad Engine/Diesel Fuel Standards: 2020 and 2030

Statistic	2020	2030
	<i>PM_{2.5} Annual Mean</i>	<i>PM_{2.5} Annual Mean</i>
<i>Absolute Change from Base Case (µg/m³)^a</i>		
Minimum	-0.02	-0.02
Maximum	-1.36	-2.03
Average	-0.20	-0.28
Median	-0.19	-0.26
Population-Weighted Average ^c	-0.42	-0.59
<i>Relative Change from Base Case (%)^b</i>		
Minimum	-0.33%	-0.44%
Maximum	-12.24%	-15.52%
Average	-2.44%	-3.32%
Median	-2.33%	-3.13%
Population-Weighted Average ^c	-3.28%	-4.38%

^a The absolute change is defined as the control case value minus the base case value for each REMSAD grid-cell.

^b The relative change is defined as the absolute change divided by the base case value, or the percentage change, for each gridcell. The information reported in this section does not necessarily reflect the same gridcell as is portrayed in the absolute change section.

^c Calculated by summing the product of the projected gridcell population and the estimated gridcell PM absolute/relative measure of change, and then dividing by the total population in the 48 contiguous states.

9A.2.2 Ozone Air Quality Estimates

We use the emissions inputs summarized in Section 9A.1 with a regional-scale version of CAMx to estimate ozone air quality in the Eastern and Western U.S. CAMx is an Eulerian three-dimensional photochemical grid air quality model designed to calculate the concentrations of both inert and chemically reactive pollutants by simulating the physical and chemical processes in the atmosphere that affect 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 proposed rule on U.S. ozone concentrations. As discussed earlier in Chapter 2, 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, On-highway Tier-2, and HD Engine/Diesel Fuel).

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Our analysis applies the modeling system separately to the Eastern and Western U.S. for five emissions scenarios: a 1996 baseline projection, a 2020 baseline projection and a 2020 projection with nonroad controls, a 2030 baseline projection and a 2030 projection with nonroad controls. As discussed in the Benefits Analysis TSD, we use the relative predictions from the model by combining the 1996 base-year and each future-year scenario with ambient air quality observations to determine the expected change in 2020 or 2030 ozone concentrations due to the rule (Abt Associates, 2003). These results are used solely in the benefits analysis.

The CAMx modeling system requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, day-specific emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. The model divides the continental United States into two regions: East and West. As applied to each region, the model segments the area within the subject region into square blocks called grids (roughly equal in size to counties), each of which has several layers of air conditions that are considered in the analysis. Using this data, the CAMx model generates predictions of hourly ozone concentrations for every grid. We then calibrate the results of this process to develop 2020 and 2030 ozone profiles at monitor sites by normalizing the observations to the observed ozone concentrations at each monitor site. For areas (grids) without ozone monitoring data, we interpolated ozone values using data from monitors surrounding the area. After completing this process, we calculated daily and seasonal ozone metrics to be used as inputs to the health and welfare C-R functions of the benefits analysis. The following sections provide a more detailed discussion of each of the steps in this evaluation and a summary of the results.

9A.2.2.1 Modeling Domain

The modeling domain representing the Eastern U.S. is the same as that used previously for OTAG and the On-highway Tier-2 rulemaking. As shown in Figure 9A-4, this domain encompasses most of the Eastern U.S. from the East coast to mid-Texas and consists of two grids with differing resolutions. The modeling domain extends from 99 degrees to 67 degrees west longitude and from 26 degrees to 47 degrees north latitude. The inner portion of the modeling domain shown in Figure 9A-4 uses a relatively fine grid of 12 km consisting of nine vertical layers. The outer area has less horizontal resolution, as it uses a 36 km grid with the same nine vertical layers. The vertical height of the modeling domain is 4,000 meters above ground level for both areas.

The modeling domain representing the Western U.S. is the same as that used previously for the On-highway Tier-2 rulemaking. As shown in Figure 9A-5, this domain encompasses the area west of the 99th degree longitude (which runs through North and South Dakota, Nebraska, Kansas, Oklahoma, and Texas) and consists of two grids with differing resolutions. The domain extends from 127 degrees to 99 degrees west longitude and from 26 degrees to 52 degrees north latitude. The inner portion of the modeling domain shown in Figure 9A-5 uses a relatively fine grid of 12 km consisting of eleven vertical layers. The outer area has less horizontal resolution, as it uses a 36 km grid with the same

eleven vertical layers. The vertical height of the modeling domain is 4,800 meters above ground level.

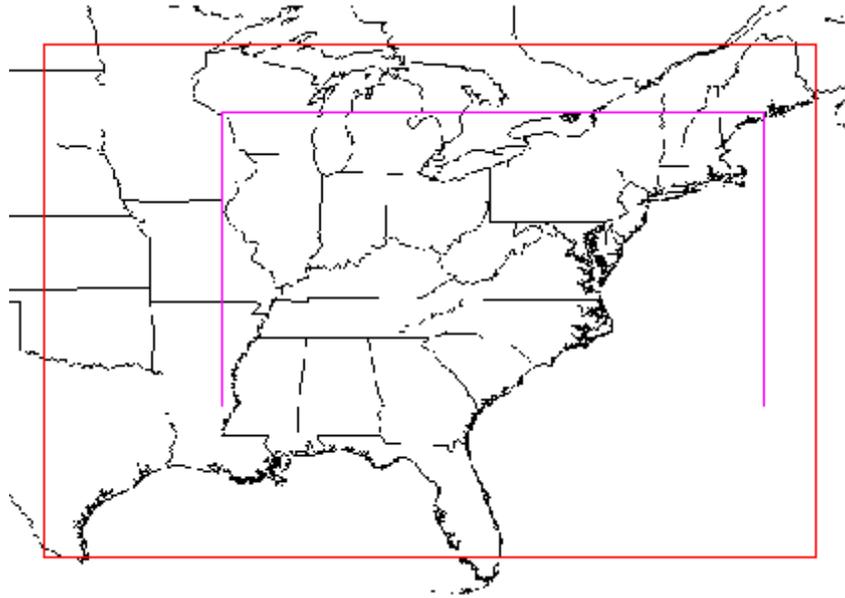


Figure 9A-4. CAMx Eastern U.S. Modeling Domain.

Note: The inner area represents fine grid modeling at 12 km resolution, while the outer area represents the coarse grid modeling at 36 km resolution.

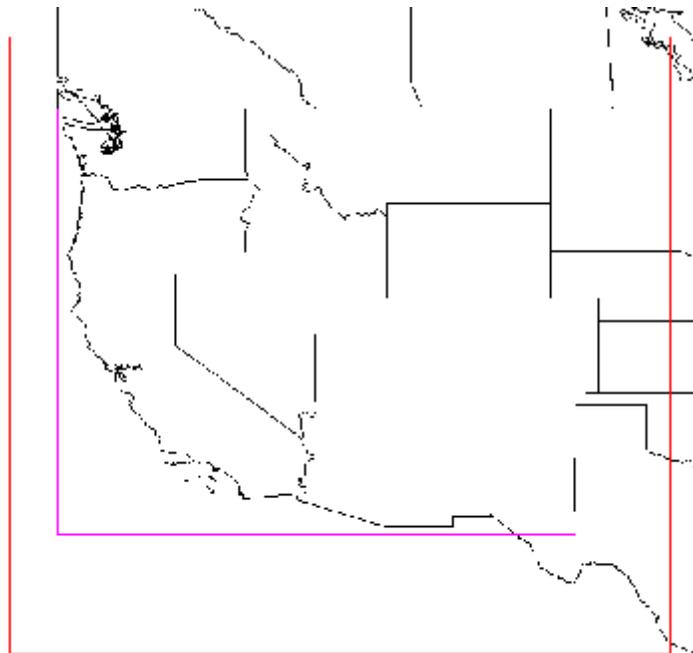


Figure 9A-5. CAMx Western U.S. Modeling Domain.

Note: The inner area represents fine grid modeling at 12 km resolution, while the outer area represents the coarse grid modeling at 36 km resolution.

9A.2.2.2 Simulation Periods

For use in this benefits analysis, the simulation periods modeled by CAMx included several multi-day periods when ambient measurements recorded high ozone concentrations. A simulation period, or episode, consists of meteorological data characterized over a block of days that are used as inputs to the air quality model. A simulation period is selected to characterize a variety of ozone conditions including some days with high ozone concentrations in one or more portions of the U.S. and observed exceedances of the 1-hour NAAQS for ozone being recorded at monitors. We focused on the summer of 1995 for selecting the episodes to model in the East and the summer of 1996 for selecting the episodes to model in the West because each is a recent time period for which we had model-ready meteorological inputs and this timeframe contained several periods of elevated ozone over the Eastern and Western U.S., respectively. As detailed in the air quality modeling TSD, this analysis used three multi-day meteorological scenarios during the summer of 1995 for the model simulations over the eastern U.S.: June 12-24, July 5-15, and August 7-21. Two multi-day meteorological scenarios during the summer of 1996 were used in the model simulations over the western U.S.: July 5-15 and July 18-31. Each of the five emissions scenarios (1996 base year, 2020 base, 2020 control, 2030 baseline, 2030 control) were simulated for the selected episodes. These episodes include a three day “ramp-up” period to initialize the model, but the results for these days are not used in this analysis.

9A.2.2.3 Converting CAMx Outputs to Full-Season Profiles for Benefits Analysis

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.^{d,e} The predicted changes in ozone concentrations from the future-year base case to future-year control scenario serve as inputs to the health and welfare C-R functions of the benefits analysis, i.e., the Criteria Air Pollutant Modeling System (CAPMS).

In order to estimate ozone-related health and welfare effects for the contiguous U.S., full-season ozone data are required for every CAPMS grid-cell. Given available ozone monitoring data, we generated full-season ozone profiles for each location in the contiguous 48 States in two steps: (1) we combine monitored observations and modeled ozone predictions to interpolate hourly ozone concentrations to a grid of 8 km by 8 km population grid-cells, and (2) we converted these full-season

^D 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.

^EBased on AIRS, there were 961 ozone monitors with sufficient data, i.e., 50 percent or more days reporting at least 9 hourly observations per day (8 am to 8 pm) during the ozone season.

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hourly ozone profiles to an ozone measure of interest, such as the daily average.^{f§} For the analysis of ozone impacts on agriculture and commercial forestry, we use a similar approach except air quality is interpolated to county centroids as opposed to population grid-cells. We report ozone 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 benefits analysis technical support document (Abt Associates, 2003).

9A.2.2.4 Ozone Air Quality Results

This section provides a summary the predicted ambient ozone concentrations from the CAMx model for the 2020 and 2030 base cases and changes associated with the Nonroad Engine/Diesel Fuel control scenario. In Tables 9A-8 and 9A-9, we provide those ozone metrics for grid-cells in the Eastern and Western U.S. respectively, that enter the concentration response functions for health benefits endpoints. In addition to the standard frequency statistics (e.g., minimum, maximum, average, median), we provide the population-weighted average which better reflects the baseline levels and predicted changes for more populated areas of the nation. This measure, therefore, will better reflect the potential benefits of these predicted changes through exposure changes to these populations.

As shown in Table 9A-8, for the 2020 ozone season, the proposed rule results in average reductions of roughly 2 percent, or between 0.57 to 0.85 ppb, in the daily average ozone concentration metrics across the Eastern U.S. population grid-cells. For the 2030 ozone season, the average reductions in the daily average ozone concentration are between 3 and 3.5 percent, or between 0.91 to 1.35 ppb. A slightly lower relative decline is predicted for the population-weighted average, which reflects the observed increases in ozone concentrations for certain hours during the year in highly populated urban areas associated with NO_x emissions reductions (see more detailed discussion in Chapter 2). Additionally, the daily 1-hour maximum ozone concentrations are predicted to decline between 2.3 and 3.6 percent in 2020 and 2030 respectively, i.e., between 1.05 and 1.66 ppb.

As shown in Table 9A-9, for the 2020 ozone season, the proposed rule results in average reductions of roughly 1.5 percent, or between 0.57 to 0.52 ppb, in the daily average ozone concentration metrics across the Western U.S. population grid-cells. For the 2030 ozone season, the average reductions in the daily average ozone concentration are roughly 2 percent, or between 0.61 to 0.82 ppb. Additionally, the daily 1-hour maximum ozone concentrations are predicted to decline between 1.3 and 2.1 percent in 2020 and 2030 respectively, i.e., between 0.62 and 0.97 ppb.

^{f§}The 8 km grid squares contain the population data used in the health benefits analysis model, CAPMS. See Section C of this appendix for a discussion of this model.

^gThis approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (EVNA) spatial interpolation (See Abt Associates (2003) for a more detailed description).

As discussed in more detail in Chapter 2, our ozone air quality modeling showed that the NO_x emissions reductions from the preliminary modeled standards are projected to result in increases in ozone concentrations for certain hours during the year, especially in urban, NO_x limited areas. These increases are often observed within the highly populated urban areas in California. As a result, the population-weighted metrics for ozone shown in Table 9A-9 indicate increases in concentrations. Most of these increases are expected to occur during hours where ozone levels are low (and often below the one-hour ozone standard). These increase are accounted for in the benefits analysis because it relies on the changes in ozone concentrations across the entire distribution of baseline levels. However, as detailed in Chapter 2 and illustrated by the results from Tables 9A-8 and 9A-9, most of the country experiences decreases in ozone concentrations for most hours in the year.

In Table 9A-10, we provide the seasonal SUM06 ozone metric for counties in the Eastern and Western U.S. that enters the concentration response function for agriculture benefit end-points. This metric is a cumulative threshold measure so that the increase in baseline NO_x emissions from Tier 2 post-control to this rulemaking have resulted in a larger number of rural counties exceeding the hourly 0.06 ppm threshold. As a result, changes in ozone concentrations for these counties are contributing to greater impacts of the Nonroad Diesel Engine rule on the seasonal SUM06 ozone metric. As shown, the average across all Eastern U.S. counties declined by 78 percent, or almost 17 ppb. Similarly high percentage reductions are observed across the other points on the distribution with the maximum declining by almost 30 ppb, or 55 percent, and the median declining by almost 20 ppb, or 83 percent.

Table 9A-9.
Summary of CAMx Derived Ozone Air Quality Metrics Due to Nonroad Engine/Diesel Fuel Standards
for Health Benefits EndPoints: Eastern U.S.

Statistic ^a	2020			2030		
	Base Case	Change ^b	Percent Change ^b	Base Case	Change ^b	Percent Change ^b
<i>Daily 1-Hour Maximum Concentration (ppb)</i>						
Minimum ^c	28.85	-0.81	-2.80%	28.81	-1.24	-4.31%
Maximum ^c	93.94	-0.85	-0.90%	94.70	-1.61	-1.70%
Average	45.54	-1.05	-2.30%	45.65	-1.66	-3.64%
Median	45.45	-1.23	-2.71%	45.52	-1.73	-3.80%
Population-Weighted Average ^d	51.34	-0.67	-1.31%	51.47	-1.16	-2.25%
<i>Daily 5-Hour Average Concentration (ppb)</i>						
Minimum ^c	24.90	-0.67	-2.68%	24.87	-1.03	-4.13%
Maximum ^c	68.69	-0.20	-0.29%	69.11	-0.44	-0.64%
Average	38.99	-0.85	-2.17%	39.08	-1.35	-3.45%
Median	38.94	-0.92	-2.39%	39.00	-1.40	-3.58%
Population-Weighted Average ^d	42.77	-0.47	-1.10%	42.90	-0.84	-1.96%
<i>Daily 8-Hour Average Concentration (ppb)</i>						
Minimum ^c	24.15	-0.64	-2.64%	24.12	-0.98	-4.07%
Maximum ^c	68.30	-0.21	-0.31%	68.72	-0.46	-0.67%
Average	38.46	-0.83	-2.16%	38.55	-1.33	-3.44%
Median	38.44	-0.89	-2.33%	38.50	-1.45	-3.76%
Population-Weighted Average ^d	42.07	-0.46	-1.08%	42.19	-0.82	-1.93%
<i>Daily 12-Hour Average Concentration (ppb)</i>						
Minimum ^c	22.42	-0.58	-2.57%	22.40	-0.89	-3.96%
Maximum ^c	66.06	-0.17	-0.25%	66.46	-0.38	-0.58%
Average	36.59	-0.78	-2.13%	36.66	-1.25	-3.40%
Median	36.61	-0.84	-2.30%	36.66	-1.43	-3.89%
Population-Weighted Average ^d	39.65	-0.40	-1.00%	39.75	-0.72	-1.80%
<i>Daily 24-Hour Average Concentration (ppb)</i>						
Minimum ^c	15.20	-0.35	-2.28%	15.19	-0.54	-3.52%
Maximum ^c	55.95	0.10	0.18%	56.23	0.04	0.07%
Average	28.93	-0.57	-1.96%	28.98	-0.91	-3.14%
Median	28.92	-0.63	-2.15%	28.98	-1.01	-3.48%
Population-Weighted Average ^d	30.24	-0.18	-0.60%	30.29	-0.37	-1.23%

^a These ozone metrics are calculated at the CAMx grid-cell level for use in health effects estimates based on the results of spatial and temporal Voronoi Neighbor Averaging. Except for the daily 24-hour average, these ozone metrics are calculated over relevant time periods during the daylight hours of the "ozone season," i.e., May through September. For the 5-hour average, the relevant time period is 10 am to 3 pm; for the 8-hr average, it is 9 am to 5 pm; and, for the 12-hr average it is 8 am to 8 pm.

^b The change is defined as the control case value minus the base case value. The percent change is the "Change" divided by the "Base Case," and then multiplied by 100 to convert the value to a percentage.

^c The base case minimum (maximum) is the value for the CAMx grid cell with the lowest (highest) value.

^d Calculated by summing the product of the projected CAMx grid-cell population and the estimated CAMx grid-cell seasonal ozone concentration, and then dividing by the total population.

Table 9A-10.
 Summary of CAMx Derived Ozone Air Quality Metrics Due to Nonroad Engine/Diesel Fuel Standards
 for Health Benefits EndPoints: Western U.S.

Statistic ^a	2020			2030		
	Base Case	Change ^b	Percent Change	Base Case	Change ^b	Percent Change ^b
<i>Daily 1-Hour Maximum Concentration (ppb)</i>						
Minimum ^c	27.48	-0.01	-0.03%	27.48	-0.01	-0.05%
Maximum ^c	201.28	4.87	2.42%	208.02	6.26	3.01%
Average	47.02	-0.62	-1.31%	47.04	-0.97	-2.07%
Median	46.10	-0.56	-1.19%	46.06	-0.66	-1.43%
Population-Weighted Average ^d	63.80	0.34	0.54%	64.23	0.38	0.58%
<i>Daily 5-Hour Average Concentration (ppb)</i>						
Minimum ^c	24.20	-0.01	-0.04%	24.21	-0.01	-0.05%
Maximum ^c	163.41	2.55	1.56%	168.89	6.04	3.57%
Average	41.11	-0.52	-1.26%	41.13	-0.82	-2.00%
Median	40.48	-0.40	-1.04%	40.46	-0.69	-1.70%
Population-Weighted Average ^d	53.56	0.45	0.84%	53.89	0.55	1.03%
<i>Daily 8-Hour Average Concentration (ppb)</i>						
Minimum ^c	23.77	-0.01	-0.04%	23.77	-0.01	-0.05%
Maximum ^c	157.49	1.33	0.84%	161.92	5.94	3.67%
Average	40.68	-0.51	-1.25%	40.69	-0.81	-1.99%
Median	40.11	-0.36	-1.03%	40.09	-0.72	-1.79%
Population-Weighted Average ^d	51.96	0.46	0.88%	52.29	0.57	1.10%
<i>Daily 12-Hour Average Concentration (ppb)</i>						
Minimum ^c	22.13	0.31	1.39%	22.09	0.44	2.01%
Maximum ^c	140.48	1.65	1.18%	143.59	1.78	1.24%
Average	39.30	-0.48	-1.23%	39.31	-0.77	-1.95%
Median	38.85	-0.38	-0.97%	38.82	-0.58	-1.50%
Population-Weighted Average ^d	47.68	0.49	1.02%	47.99	0.63	1.32%
<i>Daily 24-Hour Average Concentration (ppb)</i>						
Minimum ^c	14.08	0.22	1.60%	14.03	0.32	2.30%
Maximum ^c	95.27	0.41	0.43%	96.59	0.29	0.30%
Average	33.42	-0.38	-1.14%	33.42	-0.61	-1.82%
Median	32.97	-0.30	-0.89%	32.95	-0.61	-1.85%
Population-Weighted Average ^d	35.53	0.47	1.31%	35.74	0.63	1.77%

^a These ozone metrics are calculated at the CAMX grid-cell level for use in health effects estimates based on the results of spatial and temporal Voronoi Neighbor Averaging. Except for the daily 24-hour average, these ozone metrics are calculated over relevant time periods during the daylight hours of the "ozone season," i.e., May through September. For the 5-hour average, the relevant time period is 10 am to 3 pm; for the 8-hr average, it is 9 am to 5 pm; and,

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for the 12-hr average it is 8 am to 8 pm.

^b The change is defined as the control case value minus the base case value. The percent change is the “Change” divided by the “Base Case,” and then multiplied by 100 to convert the value to a percentage.

^c The base case minimum (maximum) is the value for the CAMX grid cell with the lowest (highest) value.

^d Calculated by summing the product of the projected CAMX grid-cell population and the estimated CAMX grid-cell seasonal ozone concentration, and then dividing by the total population.

Table 9A-11.
Summary of CAMx Derived Ozone Air Quality Metrics Due to Nonroad Engine/Diesel Fuel Standards
for Welfare Benefits Endpoints: 2020 and 2030

Statistic ^a	2020			2030		
	Base Case	Change ^b	Percent Change ^b	Base Case	Change ^b	Percent Change ^b
Eastern U.S.						
Sum06 (ppm)						
Minimum ^c	0.00	0.00	-	0.00	0.00	-
Maximum ^c	67.24	-3.30	-4.91	68.63	-5.54	-8.07%
Average	4.74	-0.72	-15.10	4.88	-1.09	-22.43%
Median	2.18	-0.76	-35.02	2.21	-0.77	-34.84%
Western U.S.						
Sum06 (ppm)						
Minimum ^c	0.00	0.00	-	0.00	0.00	-
Maximum ^c	132.73	6.09	4.59	137.71	8.45	6.14%
Average	2.78	-0.22	-7.85	2.83	-0.33	-11.72%
Median	0.00	0.00	-	0.00	0.00	-

^a SUM06 is defined as the cumulative sum of hourly ozone concentrations over 0.06 ppm (or 60 ppb) that occur during daylight hours (from 8am to 8pm) in the months of May through September. It is calculated at the county level for use in agricultural benefits based on the results of temporal and spatial Voronoi Neighbor Averaging.

^b The change is defined as the control case value minus the base case value. The percent change is the “Change” divided by the “Base Case,” which is then multiplied by 100 to convert the value to a percentage.

^c The base case minimum (maximum) is the value for the county level observation with the lowest (highest) concentration.

9A.2.3 Visibility Degradation Estimates

Visibility degradation is often directly proportional to decreases in light transmittal in the atmosphere. Scattering and absorption by both gases and particles decrease light transmittance. To quantify changes in visibility, our analysis computes a light-extinction coefficient, based on the work of Sisler (1996), which shows the total fraction of light that is decreased per unit distance. This coefficient accounts for the scattering and absorption of light by both particles and gases, and accounts for the higher extinction efficiency of fine particles compared to coarse particles. Fine particles with significant light-extinction efficiencies include sulfates, nitrates, organic carbon, elemental carbon (soot), and soil (Sisler, 1996).

Based upon the light-extinction coefficient, we also calculated a unitless visibility index, called a “deciview,” which is used in the valuation of visibility. The deciview metric provides a linear scale for perceived visual changes over the entire range of conditions, from clear to hazy. Under many scenic conditions, the average person can generally perceive a change of one deciview. The higher the deciview value, the worse the visibility. Thus, an improvement in visibility is a decrease in deciview value.

Table 9A-11 provides the distribution of visibility improvements across 2020 and 2030 populations resulting from the Nonroad Engine/Diesel Fuel rule. The majority of the 2030 U.S. population live in areas with predicted improvement in annual average visibility of between 0.4 to 0.6 deciviews resulting from the proposed rule. As shown, almost 20 percent of the 2030 U.S. population are predicted to experience improved annual average visibility of greater than 0.6 deciviews. Furthermore, roughly 70 percent of the 2030 U.S. population will benefit from reductions in annual average visibility of greater than 0.4 deciviews. The information provided in Table 9A-11 indicates how widespread the improvements in visibility are expected to be and the share of populations that will benefit from these improvements.

Because the visibility benefits analysis distinguishes between general regional visibility degradation and that particular to Federally-designated Class I areas (i.e., national parks, forests, recreation areas, wilderness areas, etc.), we separated estimates of visibility degradation into “residential” and “recreational” categories. The estimates of visibility degradation for the “recreational” category apply to Federally-designated Class I areas, while estimates for the “residential” category apply to non-Class I areas. Deciview estimates are estimated using outputs from REMSAD for the 2020 and 2030 base cases and control scenarios.

Table 9A-12.
Distribution of Populations Experiencing Visibility Improvements Due to Nonroad Diesel Engine Standards: 2020 and 2030

<i>Improvements in Visibility^a</i> <i>(annual average deciviews)</i>	<i>2020 Population</i>		<i>2030 Population</i>	
	<i>Number</i> <i>(millions)</i>	<i>Percent (%)</i>	<i>Number (millions)</i>	<i>Percent (%)</i>

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0 >) Deciview # 0.2	52.0	15.8%	11.6	3.3%
0.2 >) Deciview # 0.4	115.5	35.0%	179.7	50.5%
0.4 >) Deciview # 0.6	81.3	24.7%	90.5	25.4%
0.6 >) Deciview # 0.8	62.0	18.8%	49.1	13.8%
0.8 >) Deciview # 1.0	13.2	4.0%	16.4	4.6%
) Deciview > 1.0	5.6	1.7%	8.5	2.4%

^a The change is defined as the control case deciview level minus the base case deciview level.

9A.2.3.1 Residential Visibility Improvements

Air quality modeling results predict that the Nonroad Engine/Diesel Fuel rule will create improvements in visibility through the country. In Table 9A-12, we summarize residential visibility improvements across the Eastern and Western U.S. in 2020 and 2030. The baseline annual average visibility for all U.S. counties is 14.8 deciviews. The mean improvement across all U.S. counties is 0.28 deciviews, or almost 2 percent. In urban areas with a population of 250,000 or more (i.e., 1,209 out of 5,147 counties), the mean improvement in annual visibility was 0.39 deciviews and ranged from 0.05 to 1.08 deciviews. In rural areas (i.e., 3,938 counties), the mean improvement in visibility was 0.25 deciviews in 2030 and ranged from 0.02 to 0.94 deciviews.

On average, the Eastern U.S. experienced slightly larger absolute but smaller relative improvements in visibility than the Western U.S. from the Nonroad Engine/Diesel Fuel reductions. In Eastern U.S., the mean improvement was 0.34 deciviews from an average baseline of 19.32 deciviews. Western counties experienced a mean improvement of 0.21 deciviews from an average baseline of 9.75 deciviews projected in 2030. Overall, the data suggest that the Nonroad Engine/Diesel Fuel rule has the potential to provide widespread improvements in visibility for 2030.

Table 9A-13.
Summary of Baseline Residential Visibility and Changes by Region: 2020 and 2030
(Annual Average Deciviews)

Regions ^a	2020			2030		
	Base Case	Change ^b	Percent Change	Base Case	Change ^b	Percent Change
Eastern U.S.	20.27	0.24	1.3%	20.54	0.33	1.7%
Urban	21.61	0.24	1.2%	21.94	0.33	1.6%
Rural	19.73	0.24	1.3%	19.98	0.33	1.8%
Western U.S.	8.69	0.18	2.1%	8.83	0.25	2.8%

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Urban	9.55	0.25	2.7%	9.78	0.35	3.6%
Rural	8.50	0.17	2.0%	8.61	0.23	2.7%
National, all counties	14.77	0.21	1.7%	14.98	0.29	2.3%
Urban	17.21	0.24	1.7%	17.51	0.34	2.3%
Rural	14.02	0.20	1.6%	14.20	0.28	2.2%

^a Eastern and Western regions are separated by 100 degrees north longitude. Background visibility conditions differ by region.

^b An improvement in visibility is a decrease in deciview value. The change is defined as the Nonroad Engine/Diesel Fuel control case deciview level minus the basecase deciview level.

9A.2.3.2 Recreational Visibility Improvements

In Table 9A-13, we summarize recreational visibility improvements by region in 2020 and 2030 in Federal Class I areas. These recreational visibility regions are shown in Figure 9A-6. As shown, the national improvement in visibility for these areas increases from 1.5 percent, or 0.18 deciviews, in 2020 to 2.1 percent, or 0.24 deciviews, in 2030. Predicted relative visibility improvements are the largest in the Western U.S. as shown for California (3.2% in 2030), and the Southwest (2.9%) and the Rocky Mountain (2.5%). Federal Class I areas in the Eastern U.S. are predicted to have an absolute improvement of 0.24 deciviews in 2030, which reflects a 1.1 to 1.3 percent change from 2030 baseline visibility of 20.01 deciviews.

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Table 9A-14.

Summary of Baseline Recreational Visibility and Changes by Region: 2020 and 2030
(Annual Average Deciviews)

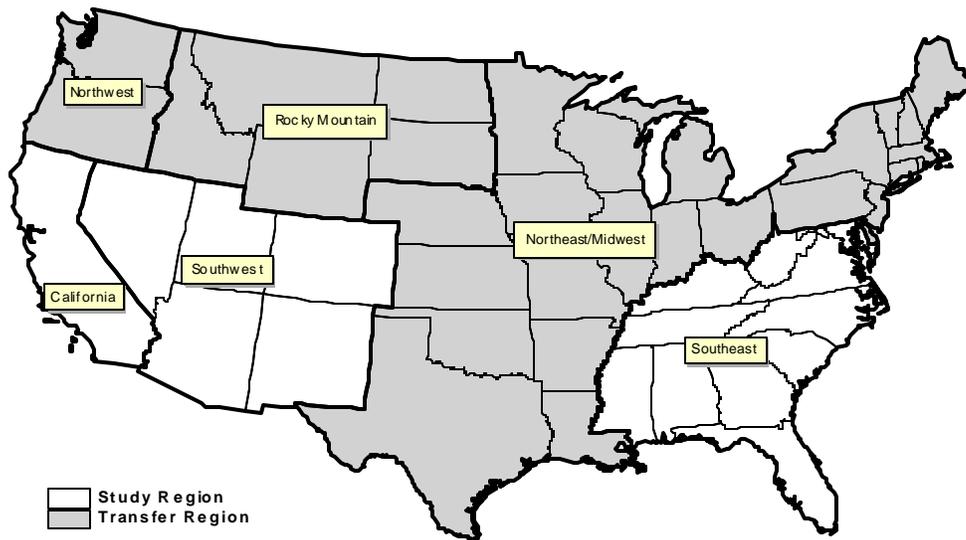
<i>Class I Visibility Regions^a</i>	<i>2020</i>			<i>2030</i>		
	<i>Base Case</i>	<i>Change^b</i>	<i>Percent Change</i>	<i>Base Case</i>	<i>Change^b</i>	<i>Percent Change</i>
Eastern U.S.	19.72	0.18	0.9%	20.01	0.24	1.2%
Southeast	21.31	0.18	0.9%	21.62	0.24	1.1%
Northeast/Midwest	18.30	0.18	1.0%	18.56	0.24	1.3%
Western U.S.	8.80	0.17	2.0%	8.96	0.24	2.7%
California	9.33	0.21	2.3%	9.56	0.30	3.2%
Southwest	6.87	0.16	2.3%	7.03	0.21	2.9%
Rocky Mountain	8.46	0.15	1.8%	8.55	0.21	2.5%
Northwest	12.05	0.18	1.5%	12.18	0.24	2.0%
National Average (unweighted)	11.61	0.18	1.5%	11.80	0.24	2.1%

^a Regions are pictured in Figure VI-5 and are defined in the technical support document (see Abt Associates, 2003).

^b An improvement in visibility is a decrease in deciview value. The change is defined as the Nonroad Engine/Diesel Fuel control case deciview level minus the basecase deciview level.

Note: Study regions were represented in the Chestnut and Rowe (1990a, 1990b) studies used in evaluating the benefits of visibility improvements, while transfer regions used extrapolated study results.

Figure 9A-6. Recreational Visibility Regions for Continental U.S.



9A.3 Benefit Analysis- Data and Methods

Environmental and health economists have a number of methods for estimating the economic value of improvements in (or deterioration of) environmental quality. The method used in any given situation depends on the nature of the effect and the kinds of data, time, and resources that are available for investigation and analysis. This section provides an overview of the methods we selected to quantify and monetize the benefits included in this RIA.

Given changes in environmental quality (ambient air quality, visibility, nitrogen and sulfate deposition), the next step is to determine the economic value of those changes. We follow a “damage-function” approach in calculating total benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare endpoints (specific effects that can be associated with changes in air quality) and assigns values to those changes assuming independence of the individual values. Total benefits are calculated simply as the sum of the values for all non-

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overlapping health and welfare endpoints. This imposes no overall preference structure, and does not account for potential income or substitution effects, i.e. adding a new endpoint will not reduce the value of changes in other endpoints. The “damage-function” approach is the standard approach for most cost-benefit analyses of environmental quality programs, and has been used in several recent published analyses (Banzhaf et al., 2002; Levy et al, 2001; Levy et al, 1999; Ostro and Chestnut, 1998).

In order to assess economic value in a damage-function framework, the changes in environmental quality must be translated into effects on people or on the things that people value. In some cases, the changes in environmental quality can be directly valued, as is the case for changes in visibility. In other cases, such as for changes in ozone and PM, a health and welfare impact analysis must first be conducted to convert air quality changes into effects that can be assigned dollar values.

For the purposes of this RIA, the health impacts analysis is limited to those health effects that are directly linked to ambient levels of air pollution, and specifically to those linked to ozone and particulate matter. There are known health effects associated with other emissions expected to be reduced by these standards, however, due to limitations in air quality models, we are unable to quantify the changes in the ambient levels of CO, SO₂, and air toxics such as benzene. There may be other, indirect health impacts associated with implementation of controls to meet the preliminary control options, such as occupational health impacts for equipment operators. These impacts may be positive or negative, but in general, for this set of preliminary control options, are expected to be small relative to the direct air pollution related impacts.

The welfare impacts analysis is limited to changes in the environment that have a direct impact on human welfare. For this analysis, we are limited by the available data to examining impacts of changes in visibility and agricultural yields. We also provide qualitative discussions of the impact of changes in other environmental and ecological effects, for example, changes in deposition of nitrogen and sulfur to terrestrial and aquatic ecosystems, but we are unable to place an economic value on these changes.

We note at the outset that EPA rarely has the time or resources to perform extensive new research to measure either the health outcomes or their values for this analysis. Thus, similar to Kunzli et al (2000) and other recent health impact analyses, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Where appropriate, adjustments are made 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.

9A.3.1 Valuation Concepts

In valuing health impacts, we note that 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 for changes in risk prior to the regulation (Freeman, 1993). In general, economists tend to view an individual's willingness-to-pay (WTP) for an improvement in environmental quality as the appropriate measure of the value of a risk reduction. An individual's willingness-to-accept (WTA) compensation for not receiving the improvement is also a valid measure. However, WTP is generally considered to be a more readily available and conservative measure of benefits. Adoption of WTP as the measure of value implies that the value of environmental quality improvements is dependent on the individual preferences of the affected population and that the existing distribution of income (ability to pay) is appropriate. 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 Portnoy, 1987; Berger, 1987).

For many goods, WTP can be observed by examining actual market transactions. For example, if a gallon of bottled drinking water sells for one dollar, it can be observed that at least some persons are willing to pay one dollar for such water. For goods not exchanged in the market, such as most environmental "goods," valuation is not as straightforward. Nevertheless, a value may be inferred from observed behavior, such as sales and prices of products that result in similar effects or risk reductions, (e.g., non-toxic cleaners or bike helmets). Alternatively, surveys may be used in an attempt to directly elicit WTP for an environmental improvement.

One distinction in environmental benefits estimation is between use values and non-use values. Although no general agreement exists among economists on a precise distinction between the two (see Freeman, 1993), the general nature of the difference is clear. Use values are those aspects of environmental quality that affect an individual's welfare more or less directly. These effects include changes in product prices, quality, and availability, changes in the quality of outdoor recreation and outdoor aesthetics, changes in health or life expectancy, and the costs of actions taken to avoid negative effects of environmental quality changes.

Non-use values are those for which an individual is willing to pay for reasons that do not relate to the direct use or enjoyment of any environmental benefit, but might relate to existence values and bequest values. Non-use values are not traded, directly or indirectly, in markets. For this reason, the measurement of non-use values has proved to be significantly more difficult than the measurement of use values. The air quality changes produced by the Nonroad Diesel Engine rule cause changes in both use and non-use values, but the monetary benefit estimates are almost exclusively for use values.

More frequently than not, the economic benefits from environmental quality changes are not traded in markets, so direct measurement techniques can not be used. There are three main non-market valuation methods used to develop values for endpoints considered in this analysis. These include stated preference (or contingent valuation), indirect market (e.g. hedonic wage), and avoided cost methods.

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The stated preference or CV method values endpoints by using carefully structured surveys to ask a sample of people what amount of compensation is equivalent to a given change in environmental quality. There is an extensive scientific literature and body of practice on both the theory and technique of stated preference based valuation. EPA believes that well-designed and well-executed stated preference studies are valid for estimating the benefits of air quality regulation.^h Stated preference valuation studies form the basis for valuing a number of health and welfare endpoints, including the value of mortality risk reductions, chronic bronchitis risk reductions, minor illness risk reductions, and visibility improvements.

Indirect market methods can also be used to infer the benefits of pollution reduction. The most important application of this technique for our analysis is the calculation of the value of a statistical life for use in the estimate of benefits from mortality risk reductions. There exists no market where changes in the probability of death are directly exchanged. However, people make decisions about occupation, precautionary behavior, and other activities associated with changes in the risk of death. By examining these risk changes and the other characteristics of people's choices, it is possible to infer information about the monetary values associated with changes in mortality risk (see Section 9A.3.5.5.1).

Avoided cost methods are ways to estimate the costs of pollution by using the expenditures made necessary by pollution damage. For example, if buildings must be cleaned or painted more frequently as levels of PM increase, then the appropriately calculated increment of these costs is a reasonable lower bound estimate (under most conditions) of true economic benefits when PM levels are reduced. Avoided costs methods are also used to estimate some of the health-related benefits related to morbidity, such as hospital admissions (see section 9A.3.5).

The most direct way to measure the economic value of air quality changes is in cases where the endpoints have market prices. For the final rule, this can only be done for effects on commercial agriculture. Well-established economic modeling approaches are used to predict price changes that result from predicted changes in agricultural outputs. Consumer and producer surplus measures can then be developed to give reliable indications of the benefits of changes in ambient air quality for this category (see Section 9A.3.6.2).

^HConcerns about the reliability of value estimates from CV studies arose because research has shown that bias can be introduced easily into these studies if they are not carefully conducted. Accurately measuring WTP for avoided health and welfare losses depends on the reliability and validity of the data collected. There are several issues to consider when evaluating study quality, including but not limited to 1) whether the sample estimates of WTP are representative of the population WTP; 2) whether the good to be valued is comprehended and accepted by the respondent; 3) whether the WTP elicitation format is designed to minimize strategic responses; 4) whether WTP is sensitive to respondent familiarity with the good, to the size of the change in the good, and to income; 5) whether the estimates of WTP are broadly consistent with other estimates of WTP for similar goods; and 6) the extent to which WTP responses are consistent with established economic principles.

9A.3.2 Growth in WTP Reflecting National Income Growth 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. There is substantial empirical evidence that the income elasticity¹ of WTP for health risk reductions is positive, although there is uncertainty about its exact value. Thus, as real income increases the WTP for environmental improvements also increases. While many analyses assume that the income elasticity of WTP is unit elastic (i.e., ten percent higher real income level implies a ten percent higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefit estimates in two different ways: (1) through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and (2) through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the SAB advised EPA to adjust WTP for increases in real income over time, but not to adjust WTP to account for cross-sectional income differences “because of the sensitivity of making such distinctions, and because of insufficient evidence available at present” (EPA-SAB-EEAC-00-013).

Based on a review of the available income elasticity literature, we adjust the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, we applied estimates derived from cross-sectional studies in our analysis. Details of the procedure can be found in Kleckner and Neumann (1999). An abbreviated description of the procedure we used to account for WTP for real income growth between 1990 and 2030 is presented below.

Reported income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP. As such, we use different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. We also expect that the WTP for improved visibility in Class I areas would increase with growth in real income. The elasticity values used to adjust estimates of benefits in 2020 and 2030 are presented in Table 9A-11.

¹Income elasticity is a common economic measure equal to the percentage change in WTP for a one percent change in income.

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Table 9A-15. Elasticity Values Used to Account for Projected Real Income Growth^A

Benefit Category	Central Elasticity Estimate
Minor Health Effect	0.14
Severe and Chronic Health Effects	0.45
Premature Mortality	0.40
Visibility ^B	0.90

^A Derivation of estimates can be found in Kleckner and Neumann (1999) and Chestnut (1997). Cost of Illness (COI) estimates are assigned an adjustment factor of 1.0.

^B No range was applied for visibility because no ranges were available in the current published literature.

In addition to elasticity estimates, projections of real GDP and populations from 1990 to 2020 and 2030 are needed to adjust benefits to reflect real per capita income growth. For consistency with the emissions and benefits modeling, we use national population estimates for the years 1990 to 1999 based on U.S. Census Bureau estimates (Hollman, Mulder and Kallan, 2000). These population estimates are based on application of a cohort-component model applied to 1990 U.S. Census data projections^j. For the years between 2000 and 2030, we applied growth rates based on the U.S. Census Bureau projections to the U.S. Census estimate of national population in 2000. We use projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010^k. We use projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's^l for the years 2010 to 2024. The Standard and Poor's database only provides estimates of real GDP between 1990 and 2024. We were unable to find reliable projections of GDP past 2024. As such, we assume that per capita GDP remains constant between 2024 and 2030.

Using the method outlined in Kleckner and Neumann (1999), and the population and income data described above, we calculate WTP adjustment factors for each of the elasticity estimates listed in Table 1. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) will be adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. Table 2 lists the estimated adjustment factors. Note that for premature mortality, we apply the income adjustment factor ex post to the present discounted value of the stream of avoided mortalities occurring over the lag period. Also note that no adjustments will be made to benefits based on the cost-of-illness approach or to work loss days and worker productivity. This

^jU.S. Bureau of Census. Annual Projections of the Total Resident Population, Middle Series, 1999-2100. (Available on the internet at <http://www.census.gov/population/www/projections/natsum-T1.html>)

^kU.S. Bureau of Economic Analysis, Table 2A (1992\$). (Available on the internet at <http://www.bea.doc.gov/bea/dn/0897nip2/tab2a.htm>) and U.S. Bureau of Economic Analysis, Economics and Budget Outlook. Note that projections for 2007 to 2010 are based on average GDP growth rates between 1999 and 2007.

^lStandard and Poor's. 2000. "The U.S. Economy: The 25 Year Focus." Winter.

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assumption will also lead us to under predict benefits in future years 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 and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher). No adjustments are needed for agricultural benefits, as the model is based on projections of supply and demand in future years and should already incorporate future changes in real income.

Table 9A-16. Adjustment Factors Used to Account for Projected Real Income Growth^A

Benefit Category	2020	2030 ^B
Minor Health Effect	1.084	1.092
Severe and Chronic Health Effects	1.299	1.329
Premature Mortality	1.262	1.287
Visibility	1.704	1.787

^A Based on elasticity values reported in Table 9A-11, US Census population projections, and projections of real gross domestic product per capita.

^B Income growth adjustment factor for 2030 is based on an assumption that there is no growth in per capita income between 2024 and 2030, based on a lack of available GDP projections beyond 2024.

9A.3.3 Methods for Describing Uncertainty

In any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty.^m This analysis is no exception. As outlined both in this and preceding chapters, 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 concentration-response (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) (Abt Associates, 2003).

^M It should be recognized that in addition to uncertainty, the annual benefit estimates for the Nonroad Diesel Engines rulemaking presented in this analysis are also inherently variable, due to the truly random processes that govern pollutant emissions and ambient air quality in a given year. Factors such as engine hours and weather display constant variability regardless of our ability to accurately measure them. As such, the estimates of annual benefits should be viewed as representative of the types of benefits that will be realized, rather than the actual benefits that would occur every year.

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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 PM₁₀ 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 9A-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:

- a. *Quantifiable uncertainty in benefits estimates.* For some parameters or inputs it may be possible to provide a statistical representation of the underlying uncertainty distribution. 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.
- b. *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.
- c. *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 CO 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 this RIA (Abt Associates, 2003). But, this information should be interpreted within the context of the larger uncertainty surrounding the entire analysis.

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 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. We are working to implement these recommendations, however, for this proposal we do not attempt to assign probabilities to sensitivity estimates due to a lack of peer-reviewed methods. At this time, we simply demonstrate the sensitivity of our benefits results to key parameters which may be uncertain. Sensitivity estimates are presented in Appendix 9B.

Our estimate of total benefits should be viewed as an approximate result because of the sources of uncertainty discussed above (see Table 9A-13). Uncertainty about specific aspects of the health and welfare estimation models are discussed in greater detail in the following sections and in the benefits TSD (Abt Associates, 2003). The total benefits estimate may understate or overstate actual benefits of the rule.

In considering the monetized benefits estimates, the reader should remain aware of the many limitations of conducting these analyses mentioned throughout this RIA. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the serious effects listed in Table 9A-1. 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 the health and welfare effects sections. 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 health and welfare effects sections as well. The net effect of excluding benefit and disbenefit categories from the estimate of total benefits depends on the relative magnitude of the effects.

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Table 9A-17. Primary Sources of Uncertainty in the Benefit Analysis

<i>1. Uncertainties Associated With Concentration-Response Functions</i>	
	<ul style="list-style-type: none"> 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.
<i>2. Uncertainties Associated With Ozone and PM Concentrations</i>	
	<ul style="list-style-type: none"> 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.
<i>3. Uncertainties Associated with PM Mortality Risk</i>	
	<ul style="list-style-type: none"> 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.
<i>4. Uncertainties Associated With Possible Lagged Effects</i>	
	<ul style="list-style-type: none"> 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.
<i>5. Uncertainties Associated With Baseline Incidence Rates</i>	
	<ul style="list-style-type: none"> 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.
<i>6. Uncertainties Associated With Economic Valuation</i>	
	<ul style="list-style-type: none"> 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 and forestry products are uncertain.
<i>7. Uncertainties Associated With Aggregation of Monetized Benefits</i>	
	<ul style="list-style-type: none"> Health and welfare benefits estimates are limited to the available C-R functions. Thus, unquantified or unmonetized benefits are not included.

9A.3.4 Demographic Projections

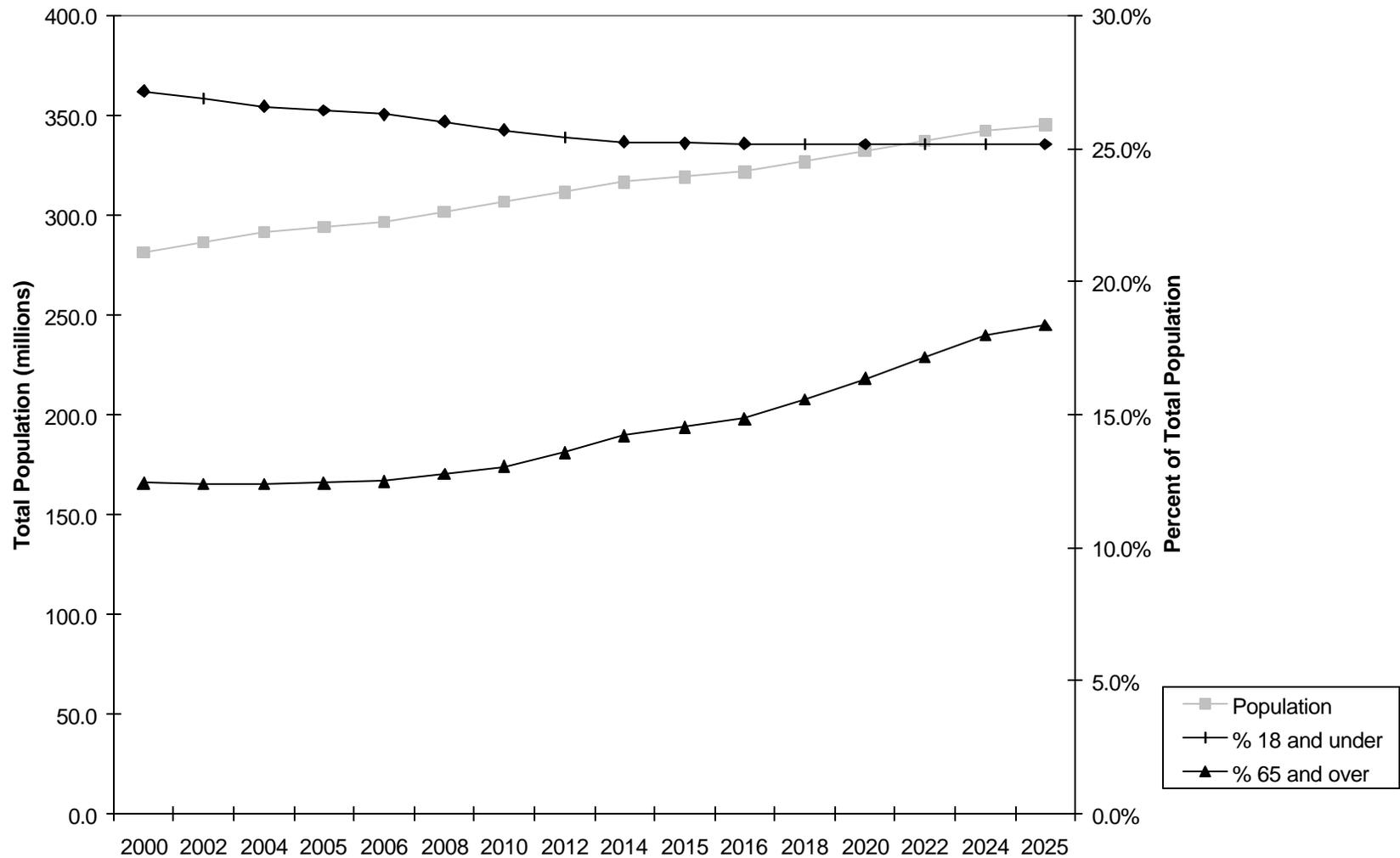
Quantified and monetized human health impacts depend critically on the demographic characteristics of the population, including age, location, and income. In previous analyses, we have used simple projections of total population that did not take into account changes in demographic composition over time. In the current analysis, we use more sophisticated projections based on economic forecasting models developed by Woods and Poole, Inc. The Woods and Poole (WP) database contains county level projections of population by age, sex, and race out to 2025. Projections in each county are determined simultaneously with every other county in the U.S. to take into account patterns of economic growth and migration. The sum of growth in county level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates (Hollman, Mulder and Kallan, 2000). According to WP, linking county level growth projections together and constraining to a national level total growth avoids potential errors introduced by forecasting each county independently. County projections are developed in a four stage process. First, national level variables such as income, employment, populations, etc. are forecasted. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an “export-base” approach, which relies on linking industrial sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-base approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector. Third, population is projected for each economic area based on net migration rates derived from employment opportunities, and following a cohort-component method based on fertility and mortality in each area. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2025 based on historical rates of mortality, fertility, and migration.

The WP projections of county level population are based on historical population data from 1969-1999, and do not include the 2000 Census results. Given the availability of detailed 2000 Census data, we constructed adjusted county level population projections for each future year using a two stage process. First, we constructed ratios of the projected WP populations in a future year to the projected WP population in 2000 for each future year by age, sex, and race. Second, we multiplied the block level 2000 Census population data by the appropriate age, sex, and race specific WP ratio for the county containing the census block, for each future year. This results in a set of future population projections that is consistent with the most recent detailed census data. The WP projections extend only through 2025. To calculate populations for 2030, we applied the growth rate from 2024 to 2025 to each year between 2025 and 2030.

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Figure 9A-7 shows the projected trends in total U.S. population and the percentage of total population aged zero to eighteen and over 65. This figure illustrates that total populations are projected increase from 281 million in 2000 to 345 million in 2025. The percent of the population 18 and under is expected to decrease slightly, from 27 to 25 percent, and the percent of the population over 65 is expected to increase from 12 percent to 18 percent.

**Figure 9A-7.
Projections of U.S. Population, 2000-2025**



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As noted above, values for environmental quality improvements are expected to increase with growth in real per capita income. Accounting for real income growth over time requires projections of both real gross domestic product (GDP) and total U.S. populations. For consistency with the emissions and benefits modeling, we use national population estimates based on the U.S. Census Bureau projections. We use projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010.ⁿ We use projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's for the years 2010 to 2024.^o The Standard and Poor's database only provides estimates of real GDP between 1990 and 2024. We were unable to find reliable projections of GDP beyond 2024. As such, we assume that per capita GDP remains constant between 2024 and 2030. This assumption will lead us to under-predict benefits because at least some level of income growth would be projected to occur between the years 2024 and 2030.

9A.3.5 Health Benefits Assessment Methods

The most significant monetized benefits of reducing ambient concentrations of PM and ozone are attributable to reductions in health risks associated with air pollution. EPA's Criteria Documents for ozone and PM list numerous health effects known to be linked to ambient concentrations of these pollutants (US EPA, 1996a and 1996b). As illustrated in Figure 9A.1, quantification of health impacts requires several inputs, including concentration-response functions, baseline incidence and prevalence rates, potentially affected populations, and estimates of changes in ambient concentrations of air pollution. Previous sections have described the population and air quality inputs. This section describes the C-R functions and baseline incidence and prevalence inputs, and the methods used to quantify and monetize changes in the expected number of incidences of various health effects.

9A.3.5.1 Selecting Concentration-Response Functions

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,

ⁿ US Bureau of Economic Analysis, Table 2A (1992\$). (Available on the internet at <http://www.bea.doc.gov/bea/dn/0897nip2/tab2a.htm>) and US Bureau of Economic Analysis, Economics and Budget Outlook. Note that projections for 2007 to 2010 are based on average GDP growth rates between 1999 and 2007.

^o Standard and Poor's. 2000. "The U.S. Economy: The 25 Year Focus." Winter 2000.

acute bronchitis, upper and lower respiratory symptoms, and work loss days.^P Health effects related to both PM and ozone include hospital admissions and minor restricted activity days.

We relied on the most recently available, published scientific literature to ascertain the relationship between particulate matter and ozone exposure and adverse human health effects. We evaluated studies using the selection criteria summarized in Table 9A-18. These criteria include consideration of whether the study was peer-reviewed, the match between the pollutant studied and the pollutant of interest, the study design and location, and characteristics of the study population, among other considerations. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility.

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. As discussed in HEI materials provided to sponsors and to the Clean Air Scientific Advisory Committee (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 statistical 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 PM₁₀ from NMMAPS were overestimated (this study was not used in this benefits analysis of fine particle effects). 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

^P 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).

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 (ACS/Krewski, et al, 2000) of mortality are included in the total primary benefits estimate. The benefits associated with ozone reductions are presented as a sensitivity analysis in Appendix 9-B but are not included in the estimate of total benefits.

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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. While resolution of these issues is likely to take some time, the preliminary results from ongoing reanalyses of some of the studies used in our benefits analysis (Dominici et al, 2002; Schwartz and Zanobetti, 2002; Schwartz, personal communication 2002) suggest a more modest effect of the S-plus error than reported for the NMMAPS PM₁₀ mortality study. While we wait for further clarification from the scientific community, we have chosen not to remove these results from the Nonroad Diesel 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 9A-1 and described more fully in the ozone and PM Criteria Documents (US EPA, 1996a, 1996b), 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, there are two studies that examine the relationship between PM and hospital admissions for asthma. One study examined the relationship between PM₁₀ and admissions for populations under the age of 65, and one examined the relationship between PM_{2.5} and populations 18 and younger. Each study provides a C-R function from which the total asthma ER visits avoided can be estimated. However, they are not compatible estimates for two reasons. First, one study is based on PM₁₀ and another on PM_{2.5}. For many health effects, PM_{2.5} is more toxic than PM₁₀ (which is composed of both PM_{2.5} and coarse PM). As such, for a given change in PM_{2.5}, a PM₁₀ function will yield a lower estimate of avoided admissions relative to a PM_{2.5} function. Pooling the two estimates will yield a downwardly biased estimate. Second, based on the evidence that most ER visits for asthma occur in the 18 and younger population, it would be expected that most of the impact measured in the 65 and younger population would actually be due to increases in admissions for the 18 and younger population. However, the C-R function would assume that the increase in risk occurs evenly throughout the population, understating the impact on the 18 and younger population. Thus, pooling the two estimates would result in a downwardly biased estimate of the avoided asthma ER visits in the 18 and younger population. Conversely, if we were to pool the two estimates to obtain an estimate of the avoided ER visits in the under 65 population, we would bias that estimate downward, because the 18 and younger study omits a potentially relevant population⁹. For this reason, we consider very carefully

⁹ One could apply the C-R function from the 18 and younger study to the full population under 65, but this would likely result in an upwardly biased estimate, given that most asthma ER visits occur in the 18 and younger

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 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.^r Pooled C-R functions are used to estimate hospital admissions related to PM and asthma-related emergency room visits related to ozone.

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.

The specific studies from which C-R functions for calculating the Base and Alternative estimates are drawn are included in Table 9A-14. A complete discussion of the C-R functions used for this analysis and information about each endpoint are contained in the benefits TSD for this RIA (Abt Associates, 2003). Basic information on each endpoint is presented below.

population.

^rThe 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.

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Table 9A-18.

Summary of Considerations Used in Selecting C-R Functions

Consideration	Comments
Peer reviewed research	Peer reviewed research is preferred to research that has not undergone the peer review process.
Study type	Among studies that consider chronic exposure (e.g., over a year or longer) prospective cohort studies are preferred over cross-sectional studies (a.k.a. "ecological studies") because they control for important confounding variables that cannot be controlled for in cross-sectional studies. If the chronic effects of a pollutant are considered more important than its acute effects, prospective cohort studies may also be preferable to longitudinal time series studies because the latter type of study is typically designed to detect the effects of short-term (e.g. daily) exposures, rather than chronic exposures. If short-term effects are considered more important, distributed lag approaches, which assume that mortality following a PM event will be distributed over a number of days following the event, are preferred over daily mortality studies. (Daily mortality studies examine the impact of PM _{2.5} on mortality on a single day or over the average of several days).
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.
Study population	Studies examining a relatively large sample are preferred. Studies of narrow population groups are generally disfavored, although this does not exclude the possibility of studying populations that are potentially more sensitive to pollutants (e.g., asthmatics, children, elderly). However, there are tradeoffs to comprehensiveness of study population. Selecting a C-R function from a study that considered all ages will avoid omitting the benefits associated with any population age category. However, if the age distribution of a study population from an "all population" study is different from the age distribution in the assessment population, and if pollutant effects vary by age, then bias can be introduced into the benefits analysis.
Study location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior and life style.
Pollutants included in model	Models with more pollutants are generally preferred to models with fewer pollutants, though careful attention must be paid to potential colinearity between pollutants. Because PM has been acknowledged to be an important and pervasive pollutant, models that include some measure of PM are highly preferred to those that do not.
Measure of PM	PM _{2.5} and PM ₁₀ are preferred to other measures of particulate matter, such as total suspended particulate matter (TSP), coefficient of haze (COH), or black smoke (BS) based on evidence that PM _{2.5} and PM ₁₀ are more directly correlated with adverse health effects than are these other measures of PM. For this analysis, PM _{2.5} is preferred to PM ₁₀ because reductions in emissions from diesel engines 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.

Cost-Benefit Analysis

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.

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Table 9A-19.
Endpoints and Studies Used to Calculate Total Monetized Health Benefits

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Base – Long-term exposure	PM _{2.5}	Krewski, et al. (2000) ^A	>29 years
Alternative – Short-term exposure ^B	PM _{2.5}	Schwartz et al. (1996) adjusted using ratio of distributed lag to single day coefficients from Schwartz et al. (2000)	all ages
Chronic Illness			
Chronic Bronchitis	PM _{2.5}	Abbey, et al. (1995)	> 26 years
Non-fatal Heart Attacks	PM _{2.5}	Peters et al. (2001)	Adults
Hospital Admissions			
Respiratory	Ozone	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)	> 64 years
	Ozone	Burnett et al. (2001)	< 2 years
	PM _{2.5}	Pooled estimate: Moolgavkar (2000) - ICD 490-496 (COPD) Lippman et al. (2000) - ICD 490-496 (COPD)	> 64 years
	PM _{2.5}	Moolgavkar (2000) - ICD 490-496 (COPD)	20-64 years
	PM _{2.5}	Lippman et al. (2000) - ICD 480-486 (pneumonia)	> 64 years
	PM _{2.5}	Sheppard, et al. (1999) - ICD 493 (asthma)	< 65 years
Cardiovascular	PM _{2.5}	Pooled estimate: Moolgavkar (2000) - ICD 390-429 (all cardiovascular) Lippman et al. (2000) - ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	> 64 years
	PM _{2.5}	Moolgavkar (2000) - ICD 390-429 (all cardiovascular)	20-64 years
Asthma-Related ER Visits	Ozone	Pooled estimate: Weisel et al. (1995), Cody et al. (1992), Stieb et al. (1996)	All ages
	PM _{2.5}	Norris et al. (1999)	0-18 years
Other Health Endpoints			
Acute Bronchitis	PM _{2.5}	Dockery et al. (1996)	8-12 years
Upper Respiratory Symptoms	PM ₁₀	Pope et al. (1991)	Asthmatics, 9-11 years
Lower Respiratory Symptoms	PM _{2.5}	Pooled estimate: Schwartz et al. (1994); Schwartz and Neas (2000)	7-14 years
Work Loss Days	PM _{2.5}	Ostro (1987)	18-65 years
School Absence Days	Ozone	Pooled estimate: Gilliland et al (2001) Chen et al (2000)	9-10 years 6-11 years
Worker Productivity	Ozone	Crocker and Horst (1981) and U.S. EPA (1984)	Outdoor workers, 18-65
Minor Restricted Activity Days	PM _{2.5} , Ozone	Ostro and Rothschild (1989)	18-65 years

^A Estimate derived from Table 31, PM_{2.5}(DC), All Causes Model (Relative Risk = 1.12 for a 24.5 : g/m³ increase in mean PM_{2.5}).

- Premature Mortality

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.

Health researchers 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 recognizes a correlation between elevated PM concentrations and increased mortality rates. Two types of community epidemiological studies (involving measures of short-term and long-term exposures and response) have been used to estimate PM/ mortality relationships. Short-term studies 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 longer-term (e.g., one or more years) exposure to PM and annual mortality rates. Researchers have found statistically significant associations using both types of studies.

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 statistically 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 (mostly current or past smokers) diagnosed with hypertension has been examined (Lipfert et al., 2000). Unlike previous long-term

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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. 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.” 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 regulatory impact analysis for the Heavy-Duty Engine/Diesel Fuel rule (U.S. EPA, 2000a), 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). 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). For these reasons, the present analysis uses the same C-R function in developing the Base Estimate of mortality benefits.

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. This will clearly provide a lower bound to the mortality impacts of fine particle exposure, as it omits any additional mortality impacts from longer term exposures. 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

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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.

There are no PM_{2.5} daily mortality studies which report numeric estimates of relative risks from distributed lag models; only PM₁₀ studies are available. Daily mortality C-R functions for PM₁₀ 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 Nonroad Diesel Engine program result primarily in reduced ambient concentrations of PM_{2.5}, use of a PM₁₀ 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 PM₁₀ 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 PM₁₀, 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:

$$\text{All cause mortality} = 0.001489 * 1.9784 = 0.002946$$

$$\text{COPD mortality} = 0.003246 * 1.9784 = 0.006422$$

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. This recommendation has been confirmed by the recent NRC report on estimating health benefits of air pollution regulations. The omission of long-term impacts accounts for approximately a 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). There are a limited number of studies that 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 gives rise to the development of chronic bronchitis in the U.S. Because the nonroad 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.

- Non-fatal 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. (2000) 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 the U.S. These studies provide a weight of evidence for this type of effect. Several epidemiologic 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 is consistent with an increased risk of heart attacks.

- Hospital and emergency room admissions

Due to the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, many of the hospital admission endpoints will use pooled C-R functions based on the results of a number of studies. In addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most ER visits do not result in an admission to the hospital (the majority of people going to the ER are treated and return home) we treat hospital

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admissions and ER visits separately, taking account of the fraction of ER visits that are admitted to the hospital.

Hospital admissions require the patient to be examined by a physician, and on average may represent more serious incidents than ER visits. The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking ozone or PM with other types of hospital admissions. The only type of ER visits that have been consistently linked to ozone and PM in the U.S. are asthma-related visits.

To estimate avoided incidences of cardiovascular hospital admissions associated with PM_{2.5}, we use studies by Moolgavkar (2000) and Lippmann et al (2000). There are additional published studies showing a statistically significant relationship between PM₁₀ and cardiovascular hospital admissions. However, given that the preliminary control options we are analyzing are expected to reduce primarily PM_{2.5}, we have chosen to focus on the two studies focusing on PM_{2.5}. Both of these studies estimated a C-R function for populations over 65, allowing us to pool the C-R functions for this age group. Only Moolgavkar (2000) estimated a separate C-R function for populations 20 to 64. Total cardiovascular hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. In order to avoid double counting benefits from reductions in MI when applying the C-R function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for MI.

To estimate total avoided incidences of respiratory hospital admissions, we use C-R functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, there are additional published studies showing a statistically significant relationship between PM₁₀ and respiratory hospital admissions. We use only those focusing on PM_{2.5}. Both Moolgavkar (2000) and Lippmann et al (2000) estimated C-R functions for COPD in populations over 65, allowing us to pool the C-R functions for this group. Only Moolgavkar (2000) estimated a separate C-R function for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Lippmann et al (2000) estimated pneumonia, and only for the population 65 and older. In addition, Sheppard, et al. (1999) estimated a C-R function for asthma hospital admissions for populations under age 65. Total avoided incidences of PM-related respiratory-related hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the C-R function based on a study of children 18 and under by Norris et al. (1999). As noted earlier, there is another study by Schwartz examining a broader age group (less than 65), but the Schwartz study focused on PM₁₀ rather than PM_{2.5}. We selected the Norris et al. (1999) C-R function because it better matched the pollutant of interest. Because children tend to have higher rates of hospitalization for asthma relative to adults under 65, we will likely capture the majority of the impact of PM_{2.5} on asthma ER visits in populations under 65, although there may still be significant impacts in the adult population

under 65. Because we are estimating ER visits as well as hospital admissions for asthma, we must avoid counting twice the ER visits for asthma that are subsequently admitted to the hospital. To avoid double-counting, the baseline incidence rate for ER visits is adjusted by subtracting the percentage of patients that are admitted into the hospital.

To estimate avoided incidences of respiratory hospital admissions associated with ozone, we use a number of studies examining hospital admissions for a range of respiratory illnesses, including pneumonia and COPD. Two age groups, adults over 65 and children under 2, are examined. For adults over 65, Schwartz (1995) provides C-R functions for 2 different cities relating ozone and hospital admissions for all respiratory causes (defined as ICD codes 460-519). These C-R functions are pooled first before being pooled with other studies. Two studies (Moolgavkar et al., 1997; Schwartz, 1994a) examined ozone and pneumonia hospital admissions in Minneapolis. One additional study (Schwartz, 1994b) examined ozone and pneumonia hospital admissions in Detroit. The C-R functions for Minneapolis are pooled together first, and the resulting C-R function is then pooled with the C-R function for Detroit. This avoids assigning too much weight to the information coming from one city. For COPD hospital admissions, there are two available studies, Moolgavkar et al. (1997), conducted in Minneapolis, and Schwartz (1994b), conducted in Detroit. These two studies are pooled together. In order to estimate total respiratory hospital admissions for adults over 65, COPD admissions are added to pneumonia admissions, and the result is pooled with the Schwartz (1995) estimate of total respiratory admissions. Burnett et al. (2001), is the only study providing a C-R function for respiratory hospital admissions in children under two.

- Minor Illnesses, Restricted Activity Days, and School/Work Loss Days

As indicated in Table 9A-1, in addition to mortality, chronic illness, and hospital admissions, there are a number of acute health effects not requiring hospitalization that are associated with exposure to ambient levels of ozone and PM. The sources for the C-R functions used to quantify these effects are described below.

Around four percent of U.S. children between ages five and seventeen experience episodes of acute bronchitis annually (American Lung Association, 2002). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia^s, with the exception of cough, most acute bronchitis symptoms abate within 7 to 10 days. Incidence of episodes of acute bronchitis in children between the ages of five and seventeen are estimated using a C-R function developed from Dockery, et al. (1996).

Incidences of lower respiratory symptoms (i.e., wheezing, deep cough) in children aged seven to fourteen are estimated using a C-R function developed from Schwartz, et al. (1994).

^s See <http://www.nlm.nih.gov/medlineplus/ency/article/000124.htm>, accessed January 2002

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

Health effects from air pollution can also result in missed days of work (either from personal symptoms or from caring for a sick family member). Work loss days due to PM_{2.5} are estimated using a C-R function developed from Ostro (1987). Children may also be absent from school due to respiratory or other diseases caused by exposure to air pollution. Most studies examining school absence rates have found little or no association with PM_{2.5}, but several studies have found a significant association between ozone levels and school absence rates. We use two recent studies, Gilliland et al. (2001) and Chen et al. (2000) to estimate changes in absences (school loss days) due to changes in ozone levels. The Gilliland et al. study estimated the incidence of new periods of absence, while the Chen et al. study examined absence on a given day. We convert the Gilliland estimate to days of absence by multiplying the absence periods by the average duration of an absence. We estimate an average duration of school absence of 1.6 days by dividing the average daily school absence rate from Chen et al. (2000) and Ransom and Pope (1992) by the episodic absence rate from Gilliland et al. (2001). This provides estimates from Chen et al. (2000) and Gilliland et al. (2000) which can be pooled to provide an overall estimate.

Minor restricted activity days (MRAD) result when individuals reduce most usual daily activities and replace them with less strenuous activities or rest, yet not to the point of missing work or school. For example, a mechanic who would usually be doing physical work most of the day, will instead spend the day at a desk doing paper and phone work due to difficulty breathing or chest pain. The effect of PM_{2.5} and ozone on MRAD is estimated using a C-R function derived from Ostro and Rothschild (1989).

The Agency is currently evaluating how air pollution related symptoms in the asthmatic population should be incorporated into the overall benefits analysis. Clearly, studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double-counting. In one specific case, upper respiratory symptoms in children, the only study available was limited to asthmatic children, so this endpoint is included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms, are estimated for the total population of children. Given the increased susceptibility of the asthmatic population, it is of interest to understand better the specific impacts on asthmatics. We are providing a separate set of estimated health impacts for asthmatic populations, listed in Table 9A-20, with the caveat that these are not additive, nor can they be easily combined with other endpoints to derive total benefits. They are provided only to highlight the potential impacts on a susceptible population.

Table 9A.20.
Studies Examining Health Impacts in the Asthmatic Population

Endpoint	Definition	Pollutant	Study	Study Population
Asthma Attack Indicators ¹				
Shortness of Breath	prevalence of shortness of breath; incidence of shortness of breath	PM _{2.5}	Ostro et al. (2001)	African American asthmatics, 8-13
Cough	prevalence of cough; incidence of cough	PM _{2.5}	Ostro et al. (2001)	African American asthmatics, 8-13
Wheeze	prevalence of wheeze; incidence of wheeze	PM _{2.5}	Ostro et al. (2001)	African American asthmatics, 8-13
Asthma Exacerbation	\$1 mild asthma symptom: wheeze, cough, chest tightness, shortness of breath)	PM ₁₀ , PM _{1.0}	Yu et al. (2000)	Asthmatics, 5-13
Cough	prevalence of cough	PM ₁₀	Vedal et al. (1998)	Asthmatics, 6-13
Other symptoms/illness endpoints				
Upper Respiratory Symptoms	\$1 of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes	PM ₁₀	Pope et al. (1991)	Asthmatics 9-11
Moderate or Worse Asthma	probability of moderate (or worse) rating of overall asthma status	PM _{2.5}	Ostro et al. (1991)	Asthmatics, all ages
Acute Bronchitis	\$1 episodes of bronchitis in the past 12 months	PM _{2.5}	McConnell et al. (1999)	Asthmatics, 9-15*
Phlegm	"other than with colds, does this child usually seem congested in the chest or bring up phlegm?"	PM _{2.5}	McConnell et al. (1999)	Asthmatics, 9-15*
Asthma Attacks	respondent-defined asthma attack	PM _{2.5} , ozone	Whittemore and Korn (1980)	Asthmatics, all ages

9A.3.5.2 Uncertainties Associated with Concentration-Response Functions

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

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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.

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.

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^t.

^tAlthough 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.

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 population 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.

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. 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 Nonroad Diesel Engine rulemaking, 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 preliminary control options in fact capture mortality effects of other criteria pollutants besides PM, we may be overestimating the benefits of reductions in PM. However, since we are not providing separate estimates of the mortality benefits from the ozone and CO reductions likely to occur due to the preliminary control options, this approach represents a reasonable surrogate for the mortality effects of all criteria pollutant reductions.

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- 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. It is currently unknown whether there is a time lag -- a delay between changes in PM exposures and changes in mortality rates -- in the chronic PM/mortality relationship. The existence of such a lag 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 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.

9A.3.5.3 Baseline Health Effect Incidence Rates

The epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the relative risk of a health effect, rather than an estimate of the absolute number of avoided cases. For example, a typical

result might be that a 10 : g/m^3 decrease in daily $\text{PM}_{2.5}$ levels might decrease hospital admissions by three percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. The baseline incidence rate provides an estimate of the incidence rate (number of cases of the health effect per year, usually per 10,000 or 100,000 general population) in the assessment location corresponding to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number (e.g., if the baseline incidence rate is number of cases per year per 100,000 population, it must be multiplied by the number of 100,000s in the population).

Some epidemiological studies examine the association between pollution levels and adverse health effects in a specific subpopulation, such as asthmatics or diabetics. In these cases, it is necessary to develop not only baseline incidence rates, but also prevalence rates for the defining condition, i.e. asthma. For both baseline incidence and prevalence data, we use age-specific rates where available. Concentration-response functions are applied to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits.

In most cases, due to a lack of data or methods, we have not attempted to project incidence rates to future years, instead assuming that the most recent data on incidence rates is the best prediction of future incidence rates. In recent years, better data on trends in incidence and prevalence rates for some endpoints, such as asthma, have become available. We are working to develop methods to use these data to project future incidence rates. However, for our primary benefits analysis of the proposed nonroad rule, we will continue to use current incidence rates. We will examine the impact of using projected mortality rates and asthma prevalence in sensitivity analyses.

Table 9A-2 summarizes the baseline incidence data and sources used in the benefits analysis. In most cases, a single national incidence rate is used, due to a lack of more spatially disaggregated data. We used national 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. However, for hospital admissions, regional rates are available, and for premature mortality, county level data are available.

Age, cause, and county-specific mortality rates were obtained from the U.S. Centers for Disease Control (CDC) for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at <http://wonder.cdc.gov/>. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across three years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25-34 year old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups.

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For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Table 9A-21 lists the baseline incidence rates and their sources for asthma symptom endpoints. Table 9A-22 lists the prevalence rates used to determine the applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years. As noted above, we are investigating methods for projecting asthma prevalence rates in future years.

Table 9A-21.
Baseline Incidence Rates and Population Prevalence Rates for Use in C-R Functions, General Population

Endpoint	Parameter	Rates		
		Value	Source ¹	
Mortality	Daily or annual mortality rate	Age, cause, and county-specific rate	CDC Wonder (1996-1998)	
Hospitalizations	Daily hospitalization rate	Age, region, cause-specific rate	1999 NHDS public use data files ²	
Asthma ER visits	Daily asthma ER visit rate	Age, Region specific visit rate	2000 NHAMCS public use data files ³ ; 1999 NHDS public use data files ²	
Chronic Bronchitis	Annual prevalence rate per person	Age 18-44 Age 45-64 Age 65 and older	0.0367 0.0505 0.0587	1999 HIS (American Lung Association, 2002b, Table 4)
	Annual incidence rate per person		0.00378	Abbey et al. (1993, Table 3)
Nonfatal MI (heart attacks)	Daily nonfatal myocardial infarction incidence rate per person, 18+	Northeast	0.0000159	1999 NHDS public use data files ² ; adjusted by 0.93 for prob. of surviving after 28 days (Rosamond et al., 1999)
		Midwest	0.0000135	
		South	0.0000111	
		West	0.0000100	
Acute Bronchitis	Annual bronchitis incidence rate, children		0.043	American Lung Association (2002a, Table 11)
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children ⁴		0.0012	Schwartz (1994, Table 2)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children		0.3419	Pope et al. (1991, Table 2)
Work Loss Days	Daily WLD incidence rate per person (18-65)	Age 18-24	0.00540	1996 HIS (Adams et al., 1999, Table 41); U.S. Bureau of the Census (2000)
		Age 25-44	0.00678	
		Age 45-64	0.00492	
Minor Restricted Activity Days	Daily MRAD incidence rate per person		0.02137	Ostro and Rothschild (1989, p. 243)

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Endpoint	Parameter	Rates	
		Value	Source ¹
School Loss Days ⁵	Daily school absence rate per person	0.055	National Center for Education Statistics (1996)
	Daily illness-related school absence rate per person ⁵		1996 HIS (Adams et al., 1999, Table 47); estimate of 180 school days per year
	Northeast	0.0136	
	Midwest	0.0146	
	South	0.0142	
	Southwest	0.0206	
	Daily <i>respiratory</i> illness-related school absence rate per person		1996 HIS (Adams et al., 1999, Table 47); estimate of 180 school days per year
	Northeast	0.0073	
	Midwest	0.0092	
	South	0.0061	
	West	0.0124	

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 ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/

3. See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/

4. Lower Respiratory Symptoms are defined as \$2 of the following: cough, chest pain, phlegm, wheeze

5. 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.

**Table 9A-22.
Baseline Incidence Rates and Population Prevalence Rates of Asthma Symptoms for use in C-
R Functions, Asthmatic Population.**

Endpoint	Parameter	Rates	
		Value	Source ¹
Asthma Exacerbation, wheeze	Daily wheeze incidence among asthmatic children (African-American)	0.076	Ostro et al. (2001, p. 202)
	Daily wheeze prevalence among asthmatic children (African-American)	0.173	Ostro et al. (2001, p. 202)
	Daily wheeze prevalence among asthmatic children	0.038	Vedal et al. (1998, Table 1)
Asthma Exacerbation, cough	Daily cough incidence among asthmatic children (African-American)	0.067	Ostro et al. (2001, p. 202)
	Daily cough prevalence among asthmatic children (African-American)	0.145	Ostro et al. (2001, p. 202)
	Daily cough prevalence among asthmatic children	0.086	Vedal et al. (1998, Table 1)
Asthma Exacerbation, dyspnea	Daily dyspnea incidence among asthmatic children (African-American)	0.037	Ostro et al. (2001, p. 202)
	Daily dyspnea prevalence among asthmatic children (African-American)	0.074	Ostro et al. (2001, p. 202)
	Daily dyspnea prevalence among asthmatic children	0.045	Vedal et al. (1998, Table 1)
Asthma Exacerbation, one or more	Daily prevalence among asthmatic children of at least one of the following symptoms: wheeze, cough, chest tightness, shortness of breath.	0.60	Yu et al. (2000, Table 2)
Asthma Attacks	Daily incidence of asthma attacks	0.055	HIS 1999
Acute/Chronic Bronchitis	Annual bronchitis incidence rate among asthmatic children	0.326	McConnell et al.(1999, Table 2)
Chronic Phlegm	Annual phlegm incidence rate among asthmatic children	0.257	McConnell et al.(1999, Table 2)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children*	0.3419	Pope et al. (1991, Table 2)

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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.

* Upper Respiratory Symptoms are defined as \$1 of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes.

Table 9A-24.
Asthma Prevalence Rates Used to Estimate Asthmatic Populations in C-R Functions

Population Group	Asthma Prevalence Rates	
	Value	Source
All Ages	0.0386	American Lung Association (2002c, Table 7)-based on 1999 HIS
<18	0.0527	American Lung Association (2002c, Table 7)-based on 1999 HIS
5-17	0.0567	American Lung Association (2002c, Table 7)-based on 1999 HIS
18-44	0.0371	American Lung Association (2002c, Table 7)-based on 1999 HIS
45-64	0.0333	American Lung Association (2002c, Table 7)-based on 1999 HIS
65+	0.0221	American Lung Association (2002c, Table 7)-based on 1999 HIS
Male, 27+	0.021	2000 HIS public use data files ¹
African-American, 5 to 17	0.0726	American Lung Association (2002c, Table 9)-based on 1999 HIS
African-American, <18	0.0735	American Lung Association (2002c, Table 9)-based on 1999 HIS

1. See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/HIS/2000/

9A.3.5.4 Accounting for Potential Health Effect Thresholds

When conducting clinical (chamber) and epidemiological studies, C-R functions may be estimated with or without explicit thresholds. Air pollution levels below the threshold are assumed to have no associated adverse health effects. When a threshold is not assumed, as is often the case in epidemiological studies, any exposure level is assumed to pose a non-zero risk of response to at least one segment of the population.

The possible existence of an effect threshold is a very important scientific question and issue for policy analyses such as this one. The EPA Science Advisory Board Advisory Council for Clean Air Compliance, which provides advice and review of EPA’s methods for assessing the benefits and costs of the Clean Air Act under Section 812 of the Clean Air Act, has advised EPA that there is currently no scientific basis for selecting a threshold of 15 : g/m³ or any other specific threshold for the PM-related health effects considered in typical benefits analyses (EPA-SAB-Council-ADV-99-012, 1999). This is supported by the recent literature on health effects of PM exposure (Daniels et al., 2000; Pope, 2000; Rossi et al., 1999; Schwartz, 2000) which finds in most cases no evidence of a non-linear

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concentration-response relationship and certainly does not find a distinct threshold for health effects. The most recent draft of the EPA Air Quality Criteria for Particulate Matter (U.S. EPA, 2002) reports only one study, analyzing data from Phoenix, AZ, that reported even limited evidence suggestive of a possible threshold for PM_{2.5} (Smith et al., 2000).

Recent cohort analyses by the Health Effects Institute (Krewski et al., 2000) and Pope et al. (2002) provide additional evidence of a quasi-linear concentration-response relationship between long-term exposures to PM_{2.5} and mortality. According to the latest draft PM criteria document, Krewski et al. (2000) “found a visually near-linear relationship between all-cause and cardiopulmonary mortality residuals and mean sulfate concentrations, near-linear between cardiopulmonary mortality and mean PM_{2.5}, but a somewhat nonlinear relationship between all-cause mortality residuals and mean PM_{2.5} concentrations that flattens above about 20 : g/m³. The confidence bands around the fitted curves are very wide, however, neither requiring a linear relationship nor precluding a nonlinear relationship if suggested by reanalyses.” The Pope et al. (2002) analysis, which represented an extension to the Krewski et al. analysis, found that the concentration-response relationships relating PM_{2.5} and mortality “were not significantly different from linear associations.”

Daniels et al. (2000) examined the presence of threshold in PM₁₀ concentration-response relationships for daily mortality using the largest 20 U.S. cities for 1987-1994. The results of their models suggest that the linear model was preferred over spline and threshold models. Thus, these results suggest that linear models without a threshold may well be appropriate for estimating the effects of PM₁₀ on the types of mortality of main interest. Schwartz and Zanobetti (2000) investigated the presence of threshold by simulation and actual data analysis of 10 U.S. cities. In the analysis of real data from 10 cities, the combined concentration-response curve did not show evidence of a threshold in the PM₁₀-mortality associations. Schwartz, Laden, and Zanobetti (2002) investigated thresholds by combining data on the PM_{2.5}-mortality relationships for six cities and found an essentially linear relationship down to 2 : g/m³, which is at or below anthropogenic background in most areas. They also examined just traffic related particles and again found no evidence of a threshold. The Smith et al. (2000) study of associations between daily total mortality and PM_{2.5} and PM_{10-2.5} in Phoenix, AZ (during 1995-1997) also investigated the possibility of a threshold using a piecewise linear model and a cubic spline model. For both the piecewise linear and cubic spline models, the analysis suggested a threshold of around 20 to 25 : g/m³. However, the concentration-response curve for PM_{2.5} presented in this publication suggests more of a U- or V-shaped relationship than the usual “hockey stick” threshold relationship.

Based on the recent literature and advice from the SAB, we assume there are no thresholds for modeling health effects. Although not included in the primary analysis, the potential impact of a health effects threshold on avoided incidences of PM-related premature mortality is explored as a key sensitivity analysis and is presented in Appendix 9-B.

Our assumptions regarding thresholds are supported by the National Research Council in its recent review of methods for estimating the public health benefits of air pollution regulations. In their

review, the National Research Council concluded that there is no evidence for any departure from linearity in the observed range of exposure to PM₁₀ or PM_{2.5}, nor any indication of a threshold. They cite the weight of evidence available from both short and long term exposure models and the similar effects found in cities with low and high ambient concentrations of PM.

9A.3.5.5 Selecting Unit Values for Monetizing Health Endpoints

The appropriate economic value of a change in a health effect depends on whether the health effect is viewed *ex ante* (before the effect has occurred) or *ex post* (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects by a fairly small amount for a large population. The appropriate economic measure is therefore *ex ante* WTP for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is \$100, then the WTP for an avoided statistical premature mortality amounts to \$1 million (\$100/0.0001 change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

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. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These costs of illness (COI) estimates generally understate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect. Table 9A-15 summarizes the value estimates per health effect that we used in this analysis. Values are presented both for a 1990 base income level and adjusted for income growth in the two future analysis years, 2020 and 2030. Note that the unit values for hospital admissions are the weighted averages of the ICD-9 code-specific values for the group of ICD-9 codes included in the hospital admission categories. Details of the derivation of values for hospital admissions and other endpoints can be found in the benefits TSD for this RIA (Abt Associates, 2003). A discussion of the valuation methods for premature mortality and chronic bronchitis is provided here due to the relative importance of these effects. Discussions of the methods used to value non-fatal myocardial infarctions (heart attacks) and school absence days are provided because these endpoints have not been included in previous analyses and the valuation methods are still under development. In the following discussions, unit values are presented at 1990 levels of income for consistency with previous analyses. Equivalent future year values can be obtained from Table 9A-15.

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Table 9A-25. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Premature Mortality				Base value is the mean of VSL estimates from 26 studies (5 contingent valuation and 21 labor market studies) reviewed for the Section 812 Costs and Benefits of the Clean Air Act, 1990-2010 (US EPA, 1999). Alternative values are based on adjustments to the mean of VSL estimates from the 5 contingent valuation studies referenced above. Adjustments are made for age and expected number of life years remaining based on cause of death and assumed health status at time of death.
Base Estimate	\$6,300,000	\$8,000,000	\$8,100,000	
Alternative Estimate				
<u>3% discount rate</u>				
COPD deaths (under 65)	\$84,000	\$110,000	\$110,000	
COPD deaths (65 and older)	\$136,000	\$170,000	\$170,000	
Other causes (under 65)	\$790,000	\$1,000,000	\$1,000,000	
Other causes (65 and older)	\$1,200,000	\$1,600,000	\$1,600,000	
<u>7% discount rate</u>				
COPD deaths (under 65)	\$140,000	\$170,000	\$170,000	
COPD deaths (65 and older)	\$160,000	\$200,000	\$200,000	
Other causes (under 65)	\$1,200,000	\$1,500,000	\$1,500,000	
Other causes (65 and older)	\$1,400,000	\$1,700,000	\$1,700,000	

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Table 9A-25. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Chronic Bronchitis (CB)				<p>Base value is the mean of a generated distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP (as described in Viscusi et al., 1991) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB.</p> <p>Alternative value is a cost of illness (COI) estimate based on Cropper and Krupnick (1990). Includes both medical costs and opportunity cost from age of onset to expected age of death (assumes that chronic bronchitis does not change life expectancy).</p>
Base Estimate	\$340,000	\$430,000	\$440,000	
Alternative Estimate				
<u>3% discount rate</u>				
Age 27-44	\$150,542	\$150,542	\$150,542	
Age 45-64	\$97,610	\$97,610	\$97,610	
Age 65+	\$11,088	\$11,088	\$11,088	
<u>7% discount rate</u>				
Age 27-44	\$86,026	\$86,026	\$86,026	
Age 45-64	\$72,261	\$72,261	\$72,261	
Age 65+	\$9,030	\$9,030	\$9,030	

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Table 9A-25. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates												
	1990 Income Level	2020 Income Level	2030 Income Level													
<p>Non-fatal Myocardial Infarction (heart attack)</p> <p><u>3% discount rate</u></p> <p>Age 0-24 \$66,902 \$66,902 \$66,902</p> <p>Age 25-44 \$74,676 \$74,676 \$74,676</p> <p>Age 45-54 \$78,834 \$78,834 \$78,834</p> <p>Age 55-65 \$140,649 \$140,649 \$140,649</p> <p>Age 66 and over \$66,902 \$66,902 \$66,902</p> <p><u>7% discount rate</u></p> <p>Age 0-24 \$65,293 \$65,293 \$65,293</p> <p>Age 25-44 \$73,149 \$73,149 \$73,149</p> <p>Age 45-54 \$76,871 \$76,871 \$76,871</p> <p>Age 55-65 \$132,214 \$132,214 \$132,214</p> <p>Age 66 and over \$65,293 \$65,293 \$65,293</p>				<p>Age specific cost-of-illness values reflecting lost earnings and direct medical costs over a 5 year period following a non-fatal MI. Lost earnings estimates based on Cropper and Krupnick (1990). Direct medical costs based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990).</p> <p><u>Lost earnings:</u> Cropper and Krupnick (1990). Present discounted value of 5 yrs of lost earnings:</p> <table style="margin-left: 20px; border-collapse: collapse;"> <tr> <td style="text-align: left;"><u>age of onset:</u></td> <td style="text-align: center;"><u>at 3%</u></td> <td style="text-align: center;"><u>at 7%</u></td> </tr> <tr> <td>25-44</td> <td style="text-align: center;">\$8,774</td> <td style="text-align: center;">\$7,855</td> </tr> <tr> <td>45-54</td> <td style="text-align: center;">\$12,932</td> <td style="text-align: center;">\$11,578</td> </tr> <tr> <td>55-65</td> <td style="text-align: center;">\$74,746</td> <td style="text-align: center;">\$66,920</td> </tr> </table> <p><u>Direct medical expenses:</u> An average of: 1. Wittels et al., 1990 (\$102,658 – no discounting) 2. Russell et al., 1998, 5-yr period. (\$22,331 at 3% discount rate; \$21,113 at 7% discount rate)</p>	<u>age of onset:</u>	<u>at 3%</u>	<u>at 7%</u>	25-44	\$8,774	\$7,855	45-54	\$12,932	\$11,578	55-65	\$74,746	\$66,920
<u>age of onset:</u>	<u>at 3%</u>	<u>at 7%</u>														
25-44	\$8,774	\$7,855														
45-54	\$12,932	\$11,578														
55-65	\$74,746	\$66,920														
Hospital Admissions																
<p>Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)</p>	\$12,378	\$12,378	\$12,378	<p>The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).</p>												

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Table 9A-25. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Pneumonia (ICD codes 480-487)	\$14,693	\$14,693	\$14,693	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total pneumonia category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Asthma admissions	\$6,634	\$6,634	\$6,634	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
All Cardiovascular (ICD codes 390-429)	\$18,387	\$18,387	\$18,387	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Emergency room visits for asthma	\$286	\$286	\$286	Simple average of two unit COI values: (1) \$311.55, from Smith et al., 1997, and (2) \$260.67, from Stanford et al., 1999.

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Table 9A-25. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Respiratory Ailments Not Requiring Hospitalization				
Upper Respiratory Symptoms (URS)	\$25	\$27	\$27	Combinations of the 3 symptoms for which WTP estimates are available that closely match those listed by Pope, et al. result in 7 different “symptom clusters,” each describing a “type” of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for URS is the average of the dollar values for the 7 different types of URS.
Lower Respiratory Symptoms (LRS)	\$16	\$17	\$17	Combinations of the 4 symptoms for which WTP estimates are available that closely match those listed by Schwartz, et al. result in 11 different “symptom clusters,” each describing a “type” of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS.
Acute Bronchitis	\$360	\$390	\$390	Assumes a 6 day episode, with daily value equal to the average of low and high values for related respiratory symptoms recommended in Neumann, et al. 1994.
Restricted Activity and Work/School Loss Days				
Work Loss Days (WLDs)	Variable (national median =)			County-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5 – to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.

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Table 9A-25. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
School Absence Days	\$75	\$75	\$75	<p>Based on expected lost wages from parent staying home with child. Estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This median wage is \$551. Dividing by 5 gives an estimated median daily wage of \$103.</p> <p>The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of \$103, or \$75.</p>
Worker Productivity	\$0.95 per worker per 10% change in ozone per day	\$0.95 per worker per 10% change in ozone per day	\$0.95 per worker per 10% change in ozone per day	<p>Based on \$68 – median daily earnings of workers in farming, forestry and fishing – from Table 621, Statistical Abstract of the United States (“Full-Time Wage and Salary Workers – Number and Earnings: 1985 to 2000”) (Source of data in table: U.S. Bureau of Labor Statistics, Bulletin 2307 and Employment and Earnings, monthly).</p>
Minor Restricted Activity Days (MRADs)	\$51	\$55	\$56	<p>Median WTP estimate to avoid one MRAD from Tolley, et al. (1986) .</p>

*9A.3.5.5.1 Valuing Reductions in Premature Mortality Risk*Base Estimate

We estimate the monetary benefit of reducing premature mortality risk using the “value of statistical lives saved” (VSL) approach, which is a summary measure for the value 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 to determine a reasonable benefit of preventing premature mortality. The mean value of avoiding one statistical death is estimated to be \$6 million in 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.

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 \$6.3 million estimate is consistent with Viscusi’s conclusion (updated to 2000\$) that “most of the reasonable estimates of the value of life are clustered in the \$3.8 to \$8.9 million range.” Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. 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.^u

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, EPA prefers not to draw distinctions in the monetary value assigned to the lives saved even if they differ in age, health status, socioeconomic status, gender or other characteristic of the adult population.

^u The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the federal government. EPA adopted a 3 percent discount rate for its base estimate in this case to reflect reliance on a “social rate of time preference” discounting concept. We have also calculated benefits and costs using a 7 percent rate consistent with an “opportunity cost of capital” concept to reflect the time value of resources directed to meet regulatory requirements. In this case, the benefit and cost estimates were not significantly affected by the choice of discount rate. Further discussion of this topic appears in EPA’s *Guidelines for Preparing Economic Analyses* (in press).

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Following the advice of the EEAC of the SAB, EPA currently uses the 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). While there are several differences between the labor market studies EPA uses 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. Table 9A-17 lists some of these differences and the expected effect on the VSL estimate for air pollution-related mortality. For example, adjusting for age differences may imply the need to adjust the \$6.3 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 \$6.3 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 \$6.3 million (2000 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. Some of the alternative approaches that have been proposed for valuing reductions in mortality risk are discussed in Figure 9A-6.

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 \$6.3 million VSL for many of these factors does not yet exist, a thorough discussion of these factors is contained in the benefits TSD for this RIA (Abt Associates, 2003). EPA recognizes the need for investigation by the scientific community to develop additional empirical support for adjustments to VSL for the factors mentioned above.

The SAB-EEAC advised in their recent report that the EPA "continue to use a wage-risk-based VSL as its primary 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, U.S. EPA, 2000b). In developing our primary 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 our primary estimate and present sensitivity estimates reflecting age-specific VSL.

Table 9A-26. Expected Impact on Estimated Benefits of Premature Mortality Reductions of Differences Between Factors Used in Developing Applied VSL and Theoretically Appropriate VSL

Attribute	Expected Direction of Bias
Age	Uncertain, perhaps overestimate
Life expectancy/health status	Uncertain, perhaps overestimate
Attitudes toward risk	Underestimate
Income	Uncertain
Voluntary vs. Involuntary	Uncertain, perhaps underestimate
Catastrophic vs. Protracted Death	Uncertain, perhaps underestimate

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, 2) the relationship between age and willingness-to-pay for fatal risk reductions, and 3) the degree of prematurity in mortalities from air pollution.

The Alternative Estimate addresses the first 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 (2000\$), which is approximately 60 percent of the mean value of the full set of 26 studies. The second issue is addressed by assuming that the relationship between age and willingness-to-pay for fatal risk reductions can be approximated using an adjustment factor derived from Jones-Lee (1989). The SAB has advised the EPA that the appropriate way to account for age differences is to obtain the values for risk reductions from the age groups affected by the risk reduction. Several studies have found a significant effect of age on the value of mortality risk reductions expressed by citizens in the United Kingdom (Jones-Lee et al., 1985; Jones-Lee, 1989; Jones-Lee, 1993).

Two of these studies provide the basis to form ratios of the WTP of different age cohorts to a base age cohort of 40 years. These ratios can be used to provide Alternative age-adjusted estimates of the value of avoided premature mortalities. One problem with both of the Jones-Lee studies is that they examine VSL for a limited age range. They then fit VSL as a function of age and extrapolate outside the range of the data to obtain ratios for the very old. Unfortunately, because VSL is specified as quadratic in age, extrapolation beyond the range of the data can lead to a very severe decline in VSL at ages beyond 75.

Figure 9A-8. Alternative Approaches for Assessing the Value of Reduced Mortality Risk

Stated preference studies – These studies use survey responses to estimate WTP to avoid risks. *Strengths:* flexible approach allowing for appropriate risk context, good data on WTP for individuals. *Weaknesses:* risk information may not be well-understood by respondents and questions may be unfamiliar.

Consumer market studies – These studies use consumer purchases and risk data (e.g., smoke detectors) to estimate WTP to avoid risks. *Strengths:* uses revealed preferences and is a flexible approach. *Weaknesses:* very difficult to estimate both risk and purchase variables.

Value of statistical life year (VSLY) – Provides an annual equivalent to value of statistical life estimates. *Strengths:* provides financially accurate adjustment for age at death. *Weaknesses:* adjustment may not reflect how individuals consider life-years; assumes equal value for all remaining life-years.

Quality adjusted life year – Applies quality of life adjustment to life-extension data, uses cost-effectiveness data to value. *Strengths:* widely used in public health literature to assess private medical interventions. *Weaknesses:* lack of data on health state indices and life quality adjustments that are applicable to an air pollution context. Similar to VSLY, adjustment may not reflect how individuals consider life-years, and typically assumes an equal value for all remaining life-years despite evidence to the contrary.

WTP for a change in survival curve – Reflects WTP for change in risk, potentially incorporates age-specific nature of risk reduction. *Strengths:* theoretically preferred approach that most accurately reflects risk reductions from air pollution control. *Weaknesses:* almost no empirical literature available; difficulty in obtaining reliable values.

WTP for a change in longevity – Uses stated preference approach to generate WTP for longevity or longer life expectancy. *Strengths:* life expectancy is a familiar term to most individuals. *Weaknesses:* does not incorporate age-specific risk information; problems in adapting to air pollution context.

Cost-effectiveness – Determines the implicit cost of saving a life or life-year. *Strengths:* widely used in public health contexts. *Weaknesses:* health context is for private goods, dollar values do not necessarily reflect individual preferences.

A simpler and potentially less biased approach is to simply apply a single age adjustment based on whether the individual was over or under 65 years of age at the time of death. This is consistent with the range of observed ages in the Jones-Lee studies and also agrees with the findings of more recent studies by Krupnick et al. (2000) that the only significant difference in WTP is between the over 70 and under 70 age groups. To correct for the potential extrapolation error for ages beyond 70, the adjustment factor is selected as the ratio of a 70 year old individual's WTP to a 40 year old individual's WTP, which is 0.63, based on the Jones-Lee (1989) results and 0.92 based on the Jones-Lee (1993) results. To show the maximum impact of the age adjustment, the Alternative Estimate is based on the Jones-Lee (1989) adjustment factor of 0.63, which yields a VSL of \$2.3 million for populations over the age of 70. Deaths of individuals under the age of 70 are valued using the unadjusted mean VSL value of \$3.7 million (2000\$). Since these are acute mortalities, it is assumed that there is no lag between reduced exposure and reduced risk of mortality.

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Jones-Lee and Krupnick may understate the effect of age because they only control for income and do not control for wealth. While there is no empirical evidence to support or reject hypotheses regarding wealth and observed WTP, WTP for additional life years by the elderly may in part reflect their wealth position vis a vis middle age respondents.

The third 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.

In order to value the expected life years lost for COPD and non-COPD deaths, we need to construct estimates of the value of a statistical life year. The value of a life year varies based on the age at death, due to the differences in the base VSL between the 65 and older population and the under 65 population. The valuation approach used is a value of statistical life years (VSLY) approach, based on amortizing the base VSL for each age cohort. Previous applications have arrived at a single value per life year based on the discounted stream of values that correspond to the VSL for a 40 year old worker (U.S. EPA, 1999). This assumes 35 years of life lost is the base value associated with the mean VSL value of \$3.7 million (2000\$). The VSLY associated with the \$3.7 million VSL is \$172,000, annualized assuming EPA's guideline value of a 3 percent discount rate, or \$286,000, annualized assuming OMB's guideline value of a 7 percent discount rate. The VSL applied in this analysis is then built up from that VSLY by taking the present value of the stream of life years, again assuming a 3% discount rate. Thus, if you assume that a 40 year-old dying from pneumonia would lose 5 years of life, the VSL applied to that death would be \$0.79 million. For populations over age 65, we then develop a VSLY from the age-adjusted base VSL of \$2.3 million. Given an assumed remaining life expectancy of 10 years, this gives a VSLY of \$273,000, assuming a 3 percent discount rate, or \$332,000, assuming a 7 percent discount rate. Again, the VSL is built based on the present value of 5 years of lost life, so in this case, we have a 70 year old individual dying from pneumonia losing 5 years of life, implying an estimated VSL of \$1.25 million. As a final step, these estimated VSL values are multiplied by the appropriate adjustment factors to account for changes in WTP over time, as outlined above.

Applying the VSLY approach to the four categories of acute mortality results in four separate sets of values for an avoided premature mortality based on age and cause of death. After adjusting for income growth, non-COPD deaths for populations aged 65 and older are valued at around \$1.6 million per incidence in both 2010 and 2020 using a 3% discount rate and \$1.7 million per incidence using a 7% discount rate. Non-COPD deaths for populations aged 64 and younger are valued at \$1.0 million per incidence in 2020 and 2030 using a 3% discount rate and \$1.5 million using a 7% discount rate. COPD deaths for populations aged 65 and older are valued at \$0.17 million per incidence in 2020 and 2030 using a 3% discount rate and \$0.20 million using a 7% discount rate. Finally, COPD deaths for populations aged 64 and younger are valued at \$0.11 million per incidence in 2020 and 2030 using a 3% discount rate and \$0.17 million using a 7% discount rate. The implied VSL for younger populations is less than that for older populations because the value per life year is higher for older populations. Since we assume that there is a 5 year loss in life years for a PM related mortality, regardless of the age of person dying, this necessarily leads to a lower VSL for younger populations.

Uncertainties Specific to Premature Mortality Valuation

The economic benefits associated with premature mortality are the largest category of monetized benefits of the Nonroad Diesel Engine rule. 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 U.S. EPA, 1999). 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. 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.

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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 mortal risks. There is considerable uncertainty as to whether the 26 studies on the value of a statistical life provide adequate estimates of the value of a statistical life saved by air pollution reduction. Although there is considerable variation in the analytical designs and data used in the 26 underlying studies, the majority of the studies involve the value of risks to a middle-aged working population. Most of the studies examine differences in wages of risky occupations, using a wage-hedonic approach. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the average willingness to pay (WTP) to reduce the risk. The appropriateness of a distribution of WTP estimates from the 26 studies for valuing the mortality-related benefits of reductions in air pollution concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on (1) the extent to which the risks being valued are similar, and (2) the extent to which the subjects in the studies are similar to the population affected by changes in pollution concentrations.
- **Level of risk reduction.** The transferability of estimates of the value of a statistical life from the 26 studies to the Nonroad Diesel Engine rulemaking 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. 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.

9A.3.5.5.2 Valuing Reductions in the Risk 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 are provided in the benefits TSD for this 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 about \$331,000 (2000\$), 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

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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 this study 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 2000\$) due to chronic bronchitis of \$150,542 for someone between the ages of 27 and 44; \$97,610 for someone between the ages of 45 and 64; and \$11,088 for someone over 65. The corresponding age-specific estimates of lifetime present discounted value (in 2000\$) using a seven percent discount rate are \$86,026, \$72,261, and assuming \$9,030, 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.

9A.3.5.5.3 Valuing Reductions in Non-Fatal Myocardial Infarctions (Heart Attacks)

The Agency has not previously estimated the impact of its programs on reductions in the expected number of non-fatal heart attacks, although it has examined the impact of reductions in other related cardiovascular endpoints^v. We were not able to identify a suitable WTP value for reductions in the risk of non-fatal heart attacks. Instead, we propose a cost-of-illness unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with an MI 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, we estimated a present discounted value in lost earnings (in 2000\$) over 5 years due to an MI of \$8,774 for someone between the ages of 25 and 44, \$12,932 for someone between the ages of 45 and 54, and \$74,746 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 \$7,855, \$11,578, and \$66,920, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. As such we do not include lost earnings in the cost estimates for these age groups.

We have found three possible sources in the literature of estimates of the direct medical costs of MI:

- Wittels et al. (1990) estimated expected total medical costs of MI over 5 years to be \$51,211 (in 1986\$) for people who were admitted to the hospital and survived hospitalization. (There does not appear to be any discounting used.) Wittels et al. was used to value coronary heart disease in the 812 Retrospective

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Analysis of the Clean Air Act. Using the CPI-U for medical care, the Wittels estimate is \$109,474 in year 2000\$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes and prices (using “knowledgeable cardiologists” as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The authors note that the average length of hospitalization for acute MI has decreased over time (from an average of 12.9 days in 1980 to an average of 11 days in 1983). Wittels et al. used 10 days as the average in their study. It is unclear how much further the length of stay (LOS) for MI may have decreased from 1983 to the present. The average LOS for ICD code 410 (MI) in the year-2000 AHQR HCUP database is 5.5 days. However, this may include patients who died in the hospital (not included among our non-fatal MI cases), whose LOS was therefore substantially shorter than it would be if they hadn’t died.

- Eisenstein et al. (2001) estimated 10-year costs of \$44,663, in 1997\$, or \$49,651 in 2000\$ for MI patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included.
- Russell et al. (1998) estimated first-year direct medical costs of treating nonfatal MI of \$15,540 (in 1995\$), and \$1,051 annually thereafter. Converting to year 2000\$, that would be \$23,353 for a 5-year period (without discounting), or \$29,568 for a ten-year period.

In summary, the three different studies provided significantly different values:

Table 9A-27.
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.

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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, we will use estimates for medical costs that similarly cover a 5-year period – i.e., estimates from Wittels et al., 1990, and Russell et al., 1998. We will use 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 the table below.

Table 9A-28.
Estimated Costs Over a 5-Year Period (in 2000\$) of a Non-Fatal Myocardial Infarction

Age Group	Opportunity Cost	Medical Cost**	Total Cost
0 - 24	\$0	\$65,902	\$65,902
25-44	\$8,774*	\$65,902	\$74,676
45 - 54	\$12,253*	\$65,902	\$78,834
55 - 65	\$70,619*	\$65,902	\$140,649
> 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.

9A.3.5.5.4 Valuing Reductions in School Absence Days

School absences associated with exposure to ozone are likely to be due to respiratory-related symptoms and illnesses. Because the respiratory symptom and illness endpoints we are including are all PM-related rather than ozone-related, we do not have to be concerned about double counting of benefits if we aggregate the benefits of avoiding ozone-related school absences with the benefits of avoiding PM-related respiratory symptoms and illnesses.

One possible approach to valuing a school absence is using a parental opportunity cost approach. This method requires two steps: (1) estimate the probability that, if a school child stays home from school, a parent will have to stay home from work to care for the child, and (2) value the lost productivity at the person's wage. Using this method, we would estimate the proportion of families with school-age children in which both parents work, and value a school loss day as the probability of a work loss day resulting from a school loss day (i.e., the proportion of households with school-age children in which both parents work) times some measure of lost wages (whatever measure we use to value work loss days). There are two significant problems with this method, however. First, it omits WTP to avoid the symptoms/illness which resulted in the school absence. Second, it effectively gives zero value to school absences which do not result in a work loss day (unless we derive an alternative estimate of the value of the parent's time for those cases in which the parent is not in the labor force).

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We are investigating approaches using WTP for avoid the symptoms/illnesses causing the absence. In the interim, we will use the parental opportunity cost approach.

For the parental opportunity cost approach, we make an explicit, conservative assumption that in married households with two working parents, the female parent will stay home with a sick child. From the U.S. Census Bureau, Statistical Abstract of the United States: 2001, we obtained (1) the numbers of single, married, and “other” (i.e., widowed, divorced, or separated) women with children in the workforce, and (2) the rates of participation in the workforce of single, married, and “other” women with children. From these two sets of statistics, we inferred the numbers of single, married, and “other” women with children, and the corresponding percentages. These percentages were used to calculate a weighted average participation rate, as shown in the table below.

Table 9A-29.
Women with Children: Number and Percent
in the Labor Force, 2000, and Weighted Average Participation Rate*

	Number (in millions) in Labor Force	Participation Rate	Implied Total Number in Population (in millions)	Implied Percent in Population	Weighted Average Participation Rate [=sum (2)*(4) over rows]
	(1)	(2)	(3) = (1)/(2)	(4)	
Single	3.1	73.9%	4.19	11.84%	
Married	18.2	70.6%	25.78	72.79%	
Other**	4.5	82.7%	5.44	15.36%	
Total:			35.42		
					72.85%

*Data in columns (1) and (2) are from U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 577.

**Widowed, divorced, or separated.

Our estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This median wage is \$551. Dividing by 5 gives an estimated median daily wage of \$103.

The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of \$103, or \$75.

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9A.3.5.6 Unquantified Health Effects

In addition to the health effects discussed above, human exposure to PM and ozone is believed to be linked to health effects such as ozone-related premature mortality (Ito and Thurston, 1996; Samet, et al. 1997), PM-related infant mortality (Woodruff, et al., 1997), cancer (US EPA, 1996b), increased emergency room visits for non-asthma respiratory causes (US EPA, 1996a; 1996b), impaired airway responsiveness (US EPA, 1996a), increased susceptibility to respiratory infection (US EPA, 1996a), acute inflammation and respiratory cell damage (US EPA, 1996a), premature aging of the lungs and chronic respiratory damage (US EPA, 1996a; 1996b). An improvement in ambient PM and ozone air quality may reduce the number of incidences within each effect category that the U.S. population would experience. Although these health effects are believed to be PM or ozone-induced, C-R data are not available for quantifying the benefits associated with reducing these effects. The inability to quantify these effects lends a downward bias to the monetized benefits presented in this analysis.

Another category of potential effects that may change in response to ozone reduction strategies results from the shielding provided by ozone against the harmful effects of ultraviolet radiation (UV-B) derived from the sun. The great majority of this shielding results from naturally occurring ozone in the stratosphere, but the 10 percent of total “column” ozone present in the troposphere also contributes (NAS, 1991). A variable portion of this tropospheric fraction of UV-B shielding is derived from ground level or “smog” ozone related to anthropogenic air pollution. Therefore, strategies that reduce ground level ozone will, in some small measure, increase exposure to UV-B from the sun.

While it is possible to provide quantitative estimates of benefits associated with globally based strategies to restore the far larger and more spatially uniform stratospheric ozone layer, the changes in UV-B exposures associated with ground level ozone reduction strategies are much more complicated and uncertain. Smog ozone strategies, such as mobile source controls, are focused on decreasing peak ground level ozone concentrations, and it is reasonable to conclude that they produce a far more complex and heterogeneous spatial and temporal pattern of ozone concentration and UV-B exposure changes than do stratospheric ozone protection programs. In addition, the changes in long-term total column ozone concentrations are far smaller from ground-level programs. To properly estimate the change in exposure and impacts, it would be necessary to match the spatial and temporal distribution of the changes in ground-level ozone to the spatial and temporal distribution of exposure to ground level ozone and sunlight. More importantly, it is long-term exposure to UV-B that is associated with effects. Intermittent, short-term, and relatively small changes in ground-level ozone and UV-B are not likely to measurably change long-term risks of these adverse effects.

For all of these reasons, we were unable to provide reliable estimates of the changes in UV-B shielding associated with ground-level ozone changes. This inability lends an upward bias to the net monetized benefits presented in this analysis. It is likely that the adverse health effects associated with increases in UV-B exposure from decreased tropospheric ozone will, however, be relatively small because 1) the expected long-term ozone change resulting from this rule is small relative to total anthropogenic tropospheric ozone, which in turn is small in comparison to total column natural

stratospheric and tropospheric ozone; 2) air quality management strategies are focused on decreasing peak ozone concentrations and thus may change exposures over limited areas for limited times; 3) people often receive peak exposures to UV-B in coastal areas where sea or lake breezes reduce ground level pollution concentrations regardless of strategy; and 4) ozone concentration changes are greatest in urban areas and areas immediately downwind of urban areas. In these areas, people are more likely to spend most of their time indoors or in the shade of buildings, trees or vehicles.

9A.3.6 Human Welfare Impact Assessment

PM and ozone have numerous documented effects on environmental quality that affect human welfare. These welfare effects include direct damages to property, either through impacts on material structures or by soiling of surfaces, direct economic damages in the form of lost productivity of crops and trees, indirect damages through alteration of ecosystem functions, and indirect economic damages through the loss in value of recreational experiences or the existence value of important resources. EPA's Criteria Documents for PM and ozone list numerous physical and ecological effects known to be linked to ambient concentrations of these pollutants (US EPA, 1996a; 1996b). This section describes individual effects and how we quantify and monetize them. These effects include changes in commercial crop and forest yields, visibility, and nitrogen deposition to estuaries.

9A.3.6.1 Visibility Benefits

Changes in the level of ambient particulate matter caused by the reduction in emissions from the preliminary control options will 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.^w

^w A change of less than 10 percent in the light extinction budget represents a measurable improvement in visibility, but may not be perceptible to the eye in many cases. Some of the average regional changes in visibility are less than one deciview (i.e. less than 10 percent of the light extinction budget), and thus less than perceptible. However, this does not mean that these changes are not real or significant. Our assumption is then that individuals can place values on changes in visibility that may not be perceptible. This is quite plausible if individuals are aware

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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.^x 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.^y

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.^z 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). Residential visibility benefits are therefore only included as a sensitivity estimate in Appendix 9-B.

that many regulations lead to small improvements in visibility which when considered together amount to perceptible changes in visibility.

^x The Clean Air Act designates 156 national parks and wilderness areas as Class I areas for visibility protection.

^y For details of the visibility estimates discussed in this chapter, please refer to the benefits technical support document for this RIA (Abt Associates 2003).

^z 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).

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 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. However, these values are not as defensible and are thus presented only as an alternative calculation in Table 9A-25. 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 this 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 Nonroad Diesel Engines rule. 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 Nonroad Diesel Engines rule. 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.

Using the methodology outlined above, EPA estimates that the total WTP for the visibility improvements in California, Southwestern, and Southeastern Class I areas brought about by the Nonroad Diesel Engines rule is \$2.2 billion. 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, and the value accounts for growth in real income. We examine the impact of expanding the visibility benefits analysis to other areas of the country in a sensitivity analysis presented in Appendix 9-B.

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.

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9A.3.6.2 Agricultural, Forestry and other Vegetation Related Benefits

The 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, 1996). Changes in ground level ozone resulting from the preliminary control options are expected to impact crop and forest yields throughout the affected area.

Well-developed techniques exist to provide monetary estimates of these benefits to agricultural producers and to consumers. These techniques use models of planting decisions, yield response functions, and agricultural products supply and demand. The resulting welfare measures are based on predicted changes in market prices and production costs. Models also exist to measure benefits to silvicultural producers and consumers. However, these models have not been adapted for use in analyzing ozone related forest impacts. As such, our analysis provides monetized estimates of agricultural benefits, and a discussion of the impact of ozone changes on forest productivity, but does not monetize commercial forest related benefits.

9A.3.6.2.1 Agricultural Benefits

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, 1996). In addition, economic studies have shown a relationship between observed ozone levels and crop yields (Garcia, et al., 1986). The economic value associated with varying levels of yield loss for ozone-sensitive commodity crops is analyzed 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 (in this case, the implementation of the Nonroad Diesel Engines rule) that affect commodity crop yields or production costs.^{aa}

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.^{bb} Dollar values are aggregated across

^{aa}AGSIM[®] is designed to forecast agricultural supply and demand out to 2010. We were not able to adapt the model to forecast out to 2030. Instead, we apply percentage increases in yields from decreased ambient ozone levels in 2030 to 2010 yield levels, and input these into an agricultural sector model held at 2010 levels of demand and supply. It is uncertain what impact this assumption will have on net changes in surplus.

^{bb} 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

crops for each standard. The total dollar value represents a measure of the change in social welfare associated with the Nonroad Diesel Engines rule.

The model employs 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.^{cc} For some crops there are multiple C-R functions, some more sensitive to ozone and some less. Our base estimate assumes that crops are evenly mixed between relatively sensitive and relatively insensitive varieties. Sensitivity to this assumption is tested in Appendix 9-B.

9A.3.6.2.2 Forestry Benefits

Ozone also has been shown conclusively to cause discernible injury to forest trees (US EPA, 1996; Fox and Mickler, 1996). In our previous analysis of the HD Engine/Diesel Fuel rule, we were able to quantify the effects of changes in ozone concentrations on tree growth for a limited set of species. Due to data limitations, we were not able to quantify such impacts for this analysis. We plan to assess both physical impacts on tree growth and the economic value of those physical impacts in our analysis of the final rule. We will use econometric models of forest product supply and demand to estimate changes in prices, producer profits and consumer surplus.

9A.3.6.2.3 Other Vegetation Effects

An additional welfare benefit expected to accrue as a result of reductions in ambient ozone concentrations in the U.S. is the economic value the public receives from reduced aesthetic injury to forests. There is sufficient scientific information available to reliably establish that ambient ozone levels cause visible injury to foliage and impair the growth of some sensitive plant species (US EPA, 1996c, p. 5-521). However, present analytic tools and resources preclude EPA from quantifying the benefits of improved forest aesthetics.

Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to impact large economic sectors. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative economic benefits analysis has been conducted. It is estimated that more than \$20 billion (1990 dollars) are spent annually on landscaping using ornamentals (Abt Associates, 1995), both by private property owners/tenants and by governmental units responsible for public areas. This is therefore a

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.

^{cc} The total value for these crops in 1998 was \$47 billion.

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potentially important welfare effects category. However, information and valuation methods are not available to allow for plausible estimates of the percentage of these expenditures that may be related to impacts associated with ozone exposure.

The nonroad diesel standards, by reducing NO_x emissions, will also reduce nitrogen deposition on agricultural land and forests. There is some evidence that nitrogen deposition may have positive effects on agricultural output through passive fertilization. Holding all other factors constant, farmers' use of purchased fertilizers or manure may increase as deposited nitrogen is reduced. Estimates of the potential value of this possible increase in the use of purchased fertilizers are not available, but it is likely that the overall value is very small relative to other health and welfare effects. The share of nitrogen requirements provided by this deposition is small, and the marginal cost of providing this nitrogen from alternative sources is quite low. In some areas, agricultural lands suffer from nitrogen over-saturation due to an abundance of on-farm nitrogen production, primarily from animal manure. In these areas, reductions in atmospheric deposition of nitrogen from PM represent additional agricultural benefits.

Information on the effects of changes in passive nitrogen deposition on forests and other terrestrial ecosystems is very limited. The multiplicity of factors affecting forests, including other potential stressors such as ozone, and limiting factors such as moisture and other nutrients, confound assessments of marginal changes in any one stressor or nutrient in forest ecosystems. However, reductions in deposition of nitrogen could have negative effects on forest and vegetation growth in ecosystems where nitrogen is a limiting factor (US EPA, 1993).

On the other hand, there is evidence that forest ecosystems in some areas of the United States are nitrogen saturated (US EPA, 1993). Once saturation is reached, adverse effects of additional nitrogen begin to occur such as soil acidification which can lead to leaching of nutrients needed for plant growth and mobilization of harmful elements such as aluminum. Increased soil acidification is also linked to higher amounts of acidic runoff to streams and lakes and leaching of harmful elements into aquatic ecosystems.

9A.3.6.3 Benefits from Reductions in Materials Damage

The preliminary control options that we modeled are expected to produce economic benefits in the form of reduced materials damage. There are two important categories of these benefits. Household soiling refers to the accumulation of dirt, dust, and ash on exposed surfaces. Criteria pollutants also have corrosive effects on commercial/industrial buildings and structures of cultural and historical significance. The effects on historic buildings and outdoor works of art are of particular concern because of the uniqueness and irreplaceability of many of these objects.

Previous EPA benefit analyses have been able to provide quantitative estimates of household soiling damage. Consistent with SAB advice, we determined that the existing data (based on consumer expenditures from the early 1970's) are too out of date to provide a reliable enough estimate of current

household soiling damages (EPA-SAB-Council-ADV-003, 1998) to include in our base estimate. We calculate household soiling damages in a sensitivity estimate provided in Appendix 9B.

EPA is unable to estimate any benefits to commercial and industrial entities from reduced materials damage. Nor is EPA able to estimate the benefits of reductions in PM-related damage to historic buildings and outdoor works of art. Existing studies of damage to this latter category in Sweden (Grosclaude and Soguel, 1994) indicate that these benefits could be an order of magnitude larger than household soiling benefits.

9A.3.6.4 Benefits from Reduced Ecosystem Damage

The effects of air pollution on the health and stability of ecosystems are potentially very important, but are at present poorly understood and difficult to measure. The reductions in NO_x caused by the final rule could produce significant benefits. Excess nutrient loads, especially of nitrogen, cause a variety of adverse consequences to the health of estuarine and coastal waters. These effects include toxic and/or noxious algal blooms such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure (Bricker et al., 1999).

Direct C-R functions relating changes in nitrogen loadings to changes in estuarine benefits are not available. The preferred WTP based measure of benefits depends on the availability of these C-R functions and on estimates of the value of environmental responses. Because neither appropriate C-R functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, calculation of a WTP measure is not possible.

If better models of ecological effects can be defined, EPA believes that progress can be made in estimating WTP measures for ecosystem functions. These estimates would be superior to avoided cost estimates in placing economic values on the welfare changes associated with air pollution damage to ecosystem health. For example, if nitrogen or sulfate loadings can be linked to measurable and definable changes in fish populations or definable indexes of biodiversity, then CV studies can be designed to elicit individuals' WTP for changes in these effects. This is an important area for further research and analysis, and will require close collaboration among air quality modelers, natural scientists, and economists.

9A.4 Benefits Analysis—Results

Applying the C-R and valuation functions described in Section C to the estimated changes in ozone and PM described in Section B yields estimates of the changes in physical damages (i.e. premature mortalities, cases, admissions, change in deciviews, increased crop yields, etc.) and the associated monetary values for those changes. Estimates of physical health impacts are presented in Table 9A.9. Monetized values for both health and welfare endpoints are presented in Table 9A.10,

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along with total aggregate monetized benefits. All of the monetary benefits are in constant year 2000 dollars.

Not all known PM- and ozone-related health and welfare effects could be quantified or monetized. The monetized value of these unquantified effects is represented by adding an unknown “B” to the aggregate total. The estimate of total monetized health benefits is thus equal to the subset of monetized PM- and ozone-related health and welfare benefits plus B, the sum of the unmonetized health and welfare benefits.

Both the Base and Alternative estimates are dominated by benefits of mortality risk reductions. The Base estimate projects that the modeled preliminary control options will result in 6,200 avoided premature deaths in 2020 and 11,000 avoided premature deaths in 2030. The Alternative estimate projects that reductions in short-term PM_{2.5} exposures alone will result in 3,700 avoided premature deaths in 2020 and 6,600 avoided premature deaths in 2030. The increase in benefits from 2020 to 2030 reflects additional emission reductions from the standards, as well as increases in total population and the average age of the population. The omission of long-term impacts of particulate matter on mortality accounts for approximately 40 percent reduction in the estimate of avoided premature mortality in the Alternative Estimate relative to the Base Estimate.

Our base estimate of total monetized benefits in 2030 for the modeled preliminary control options rule is \$92 billion using a 3 percent discount rate and \$87 billion using a 7 percent discount rate. In 2020, the base monetized benefits are estimated at \$52 billion using a 3 percent discount rate and \$47 billion using a 7 percent discount rate. Health benefits account for 94 percent of total benefits. The monetized benefit associated with reductions in the risk of premature mortality, which accounts for \$85 billion in 2030 and \$47 billion in 2020, is over 90 percent of total monetized health benefits. The next largest benefit is for reductions in chronic illness (chronic bronchitis and non-fatal heart attacks), although this value is more than an order of magnitude lower than for premature mortality. Minor restricted activity days, work loss days, school absence days, and worker productivity account for the majority of the remaining benefits. The remaining categories account for less than \$10 million each, however, they represent a large number of avoided incidences affecting many individuals.

The alternative estimate of total monetized benefits in 2030 for the modeled preliminary control option is \$14 billion using a 3 percent discount rate and \$15 billion using a 7 percent discount rate. In 2020, the alternative monetized benefits are estimated at \$8 billion using a 3 percent discount rate and \$9 billion using a 7 percent discount rate. Health benefits account for around 80 percent of the total alternative benefits estimates. The 40 percent reduction in mortality under the Alternative Estimate and the difference in valuation of premature mortality and chronic bronchitis explain the difference in benefits between these two approaches.

A comparison of the incidence table to the monetary benefits table reveals that there is not always a close correspondence between the number of incidences avoided for a given endpoint and the monetary value associated with that endpoint. For example, there are 100 times more work loss days

than premature mortalities, yet work loss days account for only a very small fraction of total monetized benefits. This reflects the fact that many of the less severe health effects, while more common, are valued at a lower level than the more severe health effects. Also, some effects, such as hospital admissions, are valued using a proxy measure of WTP. As such the true value of these effects may be higher than that reported in Table 9A.9.

Ozone benefits are in aggregate positive for the nation. However, due to ozone increases occurring during certain hours of the day in some urban areas, in 2020 the net effect is an increase in minor restricted activity days, which are related to changes in daily average ozone (which includes hours during which ozone levels are low, but are increased relative to the baseline). However, by 2030, there is a net decrease in MRAD consistent with widespread reductions in ozone concentrations from the increased NOX emissions reductions. Overall, ozone benefits are low relative to PM benefits for similar endpoint categories because of the increases in ozone concentrations during some hours of some days in certain urban areas. For a more complete discussion of this issue, see Chapter 3.

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Table 9A.30.

Reductions in Incidence of Adverse Health Effects Associated with Reductions in Particulate Matter and Ozone Associated with the Modeled Preliminary Control Option

Endpoint	Avoided Incidence ^A (cases/year)	
	2020	2030
PM-related Endpoints		
Premature mortality ^B -		
Base estimate: Long-term exposure (adults, 30 and over)	6,200	11,000
Alternative estimate: Short-term exposure (all ages)	3,700	6,600
Chronic bronchitis (adults, 26 and over)	4,300	6,500
Non-fatal myocardial infarctions (adults, 18 and older)	11,000	18,000
Hospital admissions – Respiratory (all ages) ^C	3,100	5,500
Hospital admissions – Cardiovascular (adults, 20 and older) ^D	3,300	5,700
Emergency Room Visits for Asthma (18 and younger)	4,300	6,500
Acute bronchitis (children, 8-12)	10,000	16,000
Lower respiratory symptoms (children, 7-14)	110,000	170,000
Upper respiratory symptoms (asthmatic children, 9-11)	92,000	120,000
Work loss days (adults, 18-65)	780,000	1,100,000
Minor restricted activity days (adults, age 18-65)	4,600,000	6,500,000
Ozone-related Endpoints		
Hospital Admissions – Respiratory Causes (adults, 65 and older) ^E	370	1,100
Hospital Admissions - Respiratory Causes (children, under 2 years)	150	280
Emergency Room Visits for Asthma (all ages)	93	200
Minor restricted activity days (adults, age 18-65)	(2,400)	96,000
School absence days (children, age 6-11)	65,000	96,000

^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.

^E Respiratory hospital admissions for ozone includes admissions for all respiratory causes and subcategories for COPD and pneumonia.

Table 9A.31
Results of Human Health and Welfare Benefits Valuation for the Modeled Preliminary
Nonroad Diesel Engine Standards

Endpoint	Pollutant	Monetary Benefits ^{A,B} (millions 2000\$, Adjusted for Income Growth)	
		2020	2030
Premature mortality ^C	PM		
Base estimate: Long-term exposure, (adults, 30 and over)			
3% discount rate		\$47,000	\$85,000
7% discount rate		\$44,000	\$80,000
Alternative estimate: Short-term exposure, (all ages)			
3% discount rate		\$5,000	\$9,100
7% discount rate	\$5,700	\$10,000	
Chronic bronchitis (adults, 26 and over)	PM		
Base estimate: Willingness-to-pay		\$1,900	\$3,000
Alternative estimate: Cost-of-illness			
3% discount rate		\$420	\$600
7% discount rate	\$270	\$390	
Non-fatal myocardial infarctions	PM		
3% discount rate		\$900	\$1,400
7% discount rate		\$870	\$1,400
Hospital Admissions from Respiratory Causes	O ₃ and PM	\$55	\$110
Hospital Admissions from Cardiovascular Causes	PM	\$72	\$120
Emergency Room Visits for Asthma	O ₃ and PM	\$1	\$2
Acute bronchitis (children, 8-12)	PM	\$4	\$6
Lower respiratory symptoms (children, 7-14)	PM	\$2	\$3
Upper respiratory symptoms (asthmatic children, 9-11)	PM	\$2	\$3
Work loss days (adults, 18-65)	PM	\$110	\$150
Minor restricted activity days (adults, age 18-65)	O ₃ and PM	\$250	\$370
School absence days (children, age 6-11)	O ₃	\$5	\$10
Worker productivity (outdoor workers, age 18-65)	O ₃	\$4	\$7
Recreational visibility (86 Class I Areas)	PM	\$1,400	\$2,200
Agricultural crop damage (6 crops)	O ₃	\$89	\$140
Monetized Total ^H	O ₃ and PM		
Base estimate			
3% discount rate		\$52,000+B	\$92,000+B
7% discount rate		\$49,000+B	\$87,000+B
Alternative estimate			
3% discount rate		\$8,300+B	\$14,000+B
7% discount rate	\$8,800+B	\$15,000+B	

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^A Monetary benefits are rounded to two significant digits.

^B Monetary benefits are adjusted to account for growth in real GDP per capita between 1990 and the analysis year (2020 or 2030).

^C Premature mortality associated with ozone is not separately included in this analysis. It is assumed that the C-R function for premature mortality captures both PM mortality benefits and any mortality benefits associated with other air pollutants. Also note that the valuation assumes the 5 year distributed lag structure described earlier. Results reflect the use of two different discount rates; a 3% rate which is recommended by EPA's Guidelines for Preparing Economic Analyses (US EPA, 2000c), and 7% which is recommended by OMB Circular A-94 (OMB, 1992).

^D Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.

^E Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

^F Respiratory hospital admissions for ozone includes admissions for all respiratory causes and subcategories for COPD and pneumonia.

^G B represents the monetary value of the unmonetized health and welfare benefits. A detailed listing of unquantified PM, ozone, CO, and NMHC related health effects is provided in Table XI-B.1.

To gain further understanding into the public health impact of the modeled change in air quality associated with the preliminary control options, we examined the incidence of health effects occurring in three age groups: children (0-17), adults (18-64), and elderly adults (65 and older). Certain endpoints occur only in a subset of age groups, so not all endpoints are reported for all age groups. Two sets of age group estimates were calculated. The first is based on the specific age ranges examined in the epidemiological studies, for example, the Dockery et al (1996) acute bronchitis study focused on a sample population aged 8 to 12. These are the estimates that were used in deriving total incidences as reported in Table 9A.9. In many cases however, the study populations were defined as a matter of convenience or due to data availability, rather than due to any biological factor that would restrict the effect to the specific age group. In order to gain a more complete understanding of the potential magnitude of the health impact in the entire population, we calculate a separate estimate including the health impact on all population within an age group. The two sets of age specific incidence estimates are provided in Table 9A-32. Note that for premature mortality, we chose not to extend the estimates based on long-term exposure to children, even though there is some evidence that PM exposure has mortality impacts in this age group (see Woodruff et al., 1997). The short-term exposure studies used in the alternative estimate include all ages, and thus provide an estimate of mortality benefits occurring in children.

We also estimated respiratory symptoms and attacks occurring the asthmatic population, based on the studies defined in Table 9A-22. As with the age group specific estimates, we provide two sets of calculations, one based on applying the C-R function only to the specific population subgroup included in a study's sample population, and another based on applying the C-R function to all populations within a broader population. The two sets of asthma symptom incidences are provided in Table 9A-33. As noted earlier in this appendix, the asthma symptom estimates provided in Table 9A-33 are not additive to the total benefits presented in Table 9A-31. They are provided to show the specific impacts on an especially susceptible Subpopulations. Also note that the estimates are not additive even within the table. We have grouped the estimates based on the type of symptoms measured, but there is the potential for considerable overlap. However, these estimates provide an illustration of the consistency of the effects across studies and populations of asthmatics.

Table 9A-32.
Reductions in Incidence of Health Endpoints by Age Group^A

Endpoint/Age Group	Pollutants	Avoided Incidence - Study Population Only (cases/year)		Avoided Incidence - Total Age Group Population (cases/year)	
		2020	2030	2020	2030
Children, 0-17					
Premature mortality ^B - Alternative estimate: Short-term exposure	PM	20	30	20	30
Hospital Admissions - Respiratory Causes ^C	O ₃ and PM	240	570	240	570
Emergency Room Visits for Asthma	O ₃ and PM	4,300	6,500	4,300	6,500
Acute bronchitis	PM	10,000	16,000	31,000	47,000
Lower respiratory symptoms	PM	110,000	170,000	220,000	330,000
Upper respiratory symptoms in asthmatic children	PM	92,000	120,000	430,000	660,000
School absence days (children, age 6-11)	O ₃				
Adults, 18-64					
Premature mortality ^B - Base estimate: Long-term exposure	PM	1,400	1,800	1,500	1,900
Alternative estimate: Short-term exposure	PM	770	1,000	770	1,000
Chronic bronchitis	PM	7,600	11,000	8,300	12,000
Non-fatal myocardial infarctions	PM	3,900	5,300	3,900	5,300
Hospital admissions – Cardiovascular ^D	PM	1,100	1,450	1,100	1,450
Hospital admissions – Respiratory ^E	PM	490	660	490	660
Work loss days	PM	780,000	1,100,000	780,000	1,100,000
Minor restricted activity days	O ₃ and PM	4,600,000	6,600,000	4,600,000	6,600,000
Adults, 65 and older					
Premature mortality ^B - Base estimate: Long-term exposure	PM	4,900	9,100	4,900	9,100
Alternative estimate: Short-term exposure	PM	2,900	5,500	2,900	5,500
Chronic Bronchitis	PM	1,000	1,900	1,000	1,900
Non-fatal Myocardial Infarctions	PM	6,600	12,000	6,600	12,000
Hospital Admissions - Cardiovascular Causes	PM	2,300	4,300	2,300	4,300
Hospital Admissions – Respiratory Causes	O ₃ and PM	2,700	5,700	2,700	5,700

^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 children include ICD codes 493, 464.4, 466, and 480-486).

^D Cardiovascular hospital admissions for adults includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

^E Respiratory hospital admissions for adults include admissions for all respiratory causes and subcategories for COPD and pneumonia, and asthma.

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Table 9A-33.

Reductions in Incidence of Respiratory Symptoms in the Asthmatic Population

Endpoint (Study population)	Study	Pollutant	Avoided Incidence - Study Population Only (cases/year)		Avoided Incidence - Total Age Group Population (cases/year)	
			2020	2030	2020	2030
Asthma Attack Indicators ^A						
Shortness of Breath (African American asthmatics, 8-13)	Ostro et al. (2001)	PM	10,000	15,000	30,000	45,000
Cough (African American asthmatics, 8-13)	Ostro et al. (2001)	PM	21,000	31,000	63,000	94,000
Wheeze (African American asthmatics, 8-13)	Ostro et al. (2001)	PM	16,000	24,000	49,000	74,000
Asthma Exacerbation – one or more symptoms (Asthmatics, 5-13)	Yu et al. (2000)	PM	400,000	530,000	630,000	950,000
Cough (Asthmatics, 6-13)	Veal et al. (1998)	PM	180,000	240,000	320,000	490,000
Other symptoms/illness endpoints						
Upper Respiratory Symptoms (Asthmatics 9-11)	Pope et al. (1991)	PM	92,000	120,000	430,000	660,000
Moderate or Worse Asthma (Asthmatics, all ages)	Ostro et al. (1991)	PM	86,000	121,000	86,000	121,000
Acute Bronchitis (Asthmatics, 9-15)	McConnell et al. (1999)	PM	3,000	4,700	7,000	11,000
Chronic Phlegm (Asthmatics, 9-15)	McConnell et al. (1999)	PM	7,500	12,000	18,000	27,000
Asthma Attacks (Asthmatics, all ages)	Whittemore and Korn (1980)	PM	130,000	160,000	130,000	160,000

^A Note that these are not necessarily independent symptoms. 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 studies cover the same or similar endpoints in overlapping populations. For example, the Veal et al (1998) and Ostro et al (2000) studies both examine cough. The Ostro et al (2000) estimate examines a more restricted population than Veal et al (1998), so estimates should be combined with caution.

9A.5 Discussion

This analysis has estimated the health and welfare benefits of reductions in ambient concentrations of particulate matter resulting from reduced emissions of NO_X, SO₂, VOC, and diesel PM from nonroad diesel engines. The result suggests there will be significant health and welfare benefits arising from the regulation of emissions from nonroad engines in the U.S. Our estimate that 11,000 premature mortalities would be avoided in 2030, when emission reductions from the regulation are fully realized, provides additional evidence of the important role that pollution from the nonroad sector plays in the public health impacts of air pollution.

We provide sensitivity analyses in Appendix 9B to examine key modeling assumptions. In addition, there are other uncertainties that we could not quantify, such as the importance of unquantified effects and uncertainties in the modeling of ambient air quality. Inherent in any analysis of future regulatory programs are uncertainties in projecting atmospheric conditions, source-level emissions, and engine use hours, as well as population, health baselines, incomes, technology, and other factors. The assumptions used to capture these elements are reasonable based on the available evidence. However, data limitations prevent an overall quantitative estimate of the uncertainty associated with estimates of total economic benefits. If one is mindful of these limitations, the magnitude of the benefit estimates presented here can be useful information in expanding the understanding of the public health impacts of reducing air pollution from nonroad engines.

The U.S. EPA will continue to evaluate new methods and models and select those most appropriate for the estimation the health benefits of reductions in air pollution. It is important to continue improving benefits transfer methods in terms of transferring economic values and transferring estimated C-R functions. The development of both better models of current health outcomes and new models for additional health effects such as asthma and high blood pressure will be essential to future improvements in the accuracy and reliability of benefits analyses (Guo et al., 1999; Ibal-Mulli et al., 2001). Enhanced collaboration between air quality modelers, epidemiologists, and economists should result in a more tightly integrated analytical framework for measuring health benefits of air pollution policies. The Agency welcomes comments on how we can improve the quantification and monetization of health and welfare effects and on methods for characterizing uncertainty in our estimates.

Appendix 9A References

- Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren, and P.K. Mills. 1995. Estimated Long-Term Ambient Concentrations of PM(10) and Development of Respiratory Symptoms in a Nonsmoking Population. *Archives of Environmental Health* 50(2): 139-152.
- Abbey, D.E., F. Petersen, P. K. Mills, and W. L. Beeson. 1993. Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population. *Archives of Environmental Health* 48(1): 33-46.
- Abbey, D.E., S.D. Colome, P.K. Mills, R. Burchette, W.L. Beeson and Y. Tian. 1993. Chronic Disease Associated With Long-Term Concentrations of Nitrogen Dioxide. *Journal of Exposure Analysis and Environmental Epidemiology*. Vol. 3(2): 181-202.
- Abbey, D.E., N. Nishino, W.F. McDonnell, R.J. Burchette, S.F. Knutsen, W. Lawrence Beeson and J.X. Yang. 1999. Long-term inhalable particles and other air pollutants related to mortality in nonsmokers [see comments]. *Am J Respir Crit Care Med*. Vol. 159(2): 373-82.
- Abt Associates, Inc. 2003. *Proposed Nonroad Landbased Diesel Engine Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results*. Prepared for Office of Air Quality Planning and Standards, U.S. EPA. April, 2003.
- Agency for Healthcare Research and Quality. 2000. HCUPnet, Healthcare Cost and Utilization Project.
- American Lung Association, 1999. Chronic Bronchitis. Web site available at: <http://www.lungusa.org/diseases/lungchronic.html>.
- Adams, P.F., G.E. Hendershot and M.A. Marano. 1999. Current Estimates from the National Health Interview Survey, 1996. *Vital Health Stat*. Vol. 10(200): 1-212.
- Alberini, A., M. Cropper, T.Fu, A. Krupnick, J. Liu, D. Shaw, and W. Harrington. 1997. Valuing Health Effects of Air Pollution in Developing Countries: The Case of Taiwan. *Journal of Environmental Economics and Management*. 34: 107-126.
- American Lung Association. 2002a. Trends in Morbidity and Mortality: Pneumonia, Influenza, and Acute Respiratory Conditions. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.
- American Lung Association. 2002b. Trends in Chronic Bronchitis and Emphysema: Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.
- American Lung Association. 2002c. Trends in Asthma Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.
- Banzhaf, S., D. Burtraw, and K. Palmer. 2002. Efficient Emission Fees in the U.S. Electricity Sector. Resources for the Future Discussion Paper 02-45, October.
- Berger, M.C., G.C. Blomquist, D. Kenkel, and G.S. Tolley. 1987. Valuing Changes in Health Risks: A Comparison of Alternative Measures. *The Southern Economic Journal* 53: 977-984.
- Bricker, S. B., C. G. Clement, D. E. Pirhalla, S. P. Orlando and D. R. G. Farrow. 1999. National Estuarine Eutrophication Assessment: Effects of Nutrient Enrichment in the Nation's Estuaries.

- National Oceanic and Atmospheric Administration, National Ocean Service, Special Projects Office and the National Centers for Coastal Ocean Science. Silver Spring, Maryland. 71p
- Burnett RT, Smith-Doiron M, Stieb D, Raizenne ME, Brook JR, Dales RE, Leech JA, Cakmak S, Krewski D. 2001. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol* 153:444-52
- Carnethon MR, Liao D, Evans GW, Cascio WE, Chambless LE, Rosamond WD, Heiss G. 2002. Does the cardiac autonomic response to postural change predict incident coronary heart disease and mortality? The Atherosclerosis Risk in Communities Study. *American Journal of Epidemiology*, 155(1):48-56
- Chen, L., B.L. Jennison, W. Yang and S.T. Omaye. 2000. Elementary school absenteeism and air pollution. *Inhal Toxicol*. Vol. 12(11): 997-1016.
- Chestnut, L.G. 1997. Draft Memorandum: *Methodology for Estimating Values for Changes in Visibility at National Parks*. April 15.
- Chestnut, L.G. and R.L. Dennis. 1997. Economic Benefits of Improvements in Visibility: Acid Rain Provisions of the 1990 Clean Air Act Amendments. *Journal of Air and Waste Management Association* 47:395-402.
- Chestnut, L.G. and R.D. Rowe. 1990a. *Preservation Values for Visibility Protection at the National Parks: Draft Final Report*. Prepared for Office of Air Quality Planning and Standards, US Environmental Protection Agency, Research Triangle Park, NC and Air Quality Management Division, National Park Service, Denver, CO.
- Chestnut, L.G., and R.D. Rowe. 1990b. A New National Park Visibility Value Estimates. In *Visibility and Fine Particles*, Transactions of an AWMA/EPA International Specialty Conference, C.V. Mathai, ed. Air and Waste Management Association, Pittsburgh.
- Cody, R.P., C.P. Weisel, G. Birnbaum and P.J. Liroy. 1992. The effect of ozone associated with summertime photochemical smog on the frequency of asthma visits to hospital emergency departments. *Environ Res*. Vol. 58(2): 184-94.
- Crocker, T.D. and R.L. Horst, Jr. 1981. Hours of Work, Labor Productivity, and Environmental Conditions: A Case Study. *The Review of Economics and Statistics*. Vol. 63: 361-368.
- Cropper, M.L. and A.J. Krupnick. 1990. *The Social Costs of Chronic Heart and Lung Disease. Resources for the Future*. Washington, DC. Discussion Paper QE 89-16-REV.
- Daniels MJ, Dominici F, Samet JM, Zeger SL. 2000. Estimating particulate matter-mortality dose-response curves and threshold levels: an analysis of daily time-series for the 20 largest US cities. *Am J Epidemiol* 152(5):397-406
- Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris and F.E. Speizer. 1993. An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine* 329(24): 1753-1759.
- Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer. 1996. "Health Effects of Acid Aerosols On North American Children-Respiratory Symptoms." *Environmental Health Perspectives*. 104(5): 500-505.
- Dominici F, McDermott A, Zeger SL, Samet JM. 2002. On the use of generalized additive models in time-series studies of air pollution and health. *Am J Epidemiol* 156(3):193-203
- Dekker J.M., R.S. Crow, A.R. Folsom, P.J. Hannan, D. Liao, C.A. Swenne, and E. G. Schouten. 2000. Low Heart Rate Variability in a 2-Minute Rhythm Strip Predicts Risk of Coronary

Draft Regulatory Impact Analysis

- Heart Disease and Mortality From Several Causes : The ARIC Study. *Circulation* 2000 102: 1239-1244.
- Eisenstein, E.L., L.K. Shaw, K.J. Anstrom, C.L. Nelson, Z. Hakim, V. Hasselblad and D.B. Mark. 2001. Assessing the clinical and economic burden of coronary artery disease: 1986-1998. *Med Care*. Vol. 39(8): 824-35.
- EPA-SAB-COUNCIL-ADV-99-05, 1999. An SAB Advisory on the Health and Ecological Effects Initial Studies of the Section 812 Prospective Study: Report to Congress: Advisory by the Health and Ecological Effects Subcommittee, February.
- EPA-SAB-COUNCIL-ADV-98-003, 1998. Advisory Council on Clean Air Compliance Analysis Advisory on the Clean Air Act Amendments (CAAA) of 1990 Section 812 Prospective Study: Overview of Air Quality and Emissions Estimates: Modeling, Health and Ecological Valuation Issues Initial Studies.
- EPA-SAB-COUNCIL-ADV-99-012, 1999. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects: Part 1. July.
- EPA-SAB-COUNCIL-ADV-00-001, 1999. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects: Part 2. October, 1999.
- EPA-SAB-COUNCIL-ADV-00-002, 1999. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Advisory Council on Clean Air Compliance Analysis: Costs and Benefits of the CAAA. Effects Subcommittee on Initial Assessments of Health and Ecological Effects: Part 2. October, 1999.
- EPA-SAB-EEAC-00-013, 2000. An SAB Report on EPA's White Paper Valuing the Benefits of Fatal Cancer Risk Reduction. July.
- EPA-SAB-COUNCIL-ADV-01-004. 2001. Review of the Draft Analytical Plan for EPA's Second Prospective Analysis - Benefits and Costs of the Clean Air Act 1990-2020: An Advisory by a Special Panel of the Advisory Council on Clean Air Compliance Analysis. September.
- Evans, William N., and W. Kip Viscusi. 1993. Income Effects and the Value of Health. *Journal of Human Resources* 28(3):497-518.
- Fox, S., and R.A. Mickler, 1995. Impact of Air Pollutants on Southern Pine Forests *Ecological Studies* 118. Springer Verlag: New York.
- Freeman, A. M. III. 1993. *The Measurement of Environmental and Resource Values: Theory and Methods*. Resources for the Future, Washington, D.C.
- Garcia, P., Dixon, B. and Mjelde, J. (1986): Measuring the benefits of environmental change using a duality approach: The case of Ozone and Illinois cash grain farms. *Journal of Environmental Economics and Management*.
- Gilliland, F.D., K. Berhane, E.B. Rappaport, D.C. Thomas, E. Avol, W.J. Gauderman, S.J. London, H.G. Margolis, R. McConnell, K.T. Islam and J.M. Peters. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology*. Vol. 12(1): 43-54.
- Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B, Allen G, Verrier M, Cherry R, Verrier R. 2000. Ambient pollution and heart rate variability. *Circulation* 101(11):1267-73

- Greenbaum, D. 2002a. Letter. Health Effects Institute. May 30. Available online at : <http://www.healtheffects.org/Pubs/NMMAPSletter.pdf> . Accessed March 20, 2003.
- Grosclaude, P. and N.C. Soguel. 1994. "Valuing Damage to Historic Buildings Using a Contingent Market: A Case Study of Road Traffic Externalities." *Journal of Environmental Planning and Management* 37: 279-287.
- Guo, Y.L., Y.C. Lin, F.C. Sung, S.L. Huang, Y.C. Ko, J.S. Lai, H.J. Su, C.K. Shaw, R.S. Lin, D.W. Dockery. 1999. Climate, Traffic-Related Air Pollutants, and Asthma Prevalence in Middle-School Children in Taiwan. *Environmental Health Perspectives* 107: 1001-1006.
- Harrington, W. and P. R. Portney. 1987. Valuing the Benefits of Health and Safety Regulation. *Journal of Urban Economics* 22:101-112.
- Hollman, F.W., T.J. Mulder, and J.E. Kallan. 2000. Methodology and Assumptions for the Population Projections of the United States: 1999 to 2100. Population Division Working Paper No. 38, Population Projections Branch, Population Division, U.S. Census Bureau, Department of Commerce. January.
- Ibald-Mulli, A., J. Stieber, H.-E. Wichmann, W. Koenig, and A. Peters. 2001. Effects of Air Pollution on Blood Pressure: A Population-Based Approach. *American Journal of Public Health*. 91: 571-577.
- Industrial Economics, Incorporated (IEc). 1994. Memorandum to Jim DeMocker, Office of Air and Radiation, Office of Policy Analysis and Review, US Environmental Protection Agency, March 31.
- Ito, K. and G.D. Thurston. 1996. Daily PM10/mortality associations: an investigations of at-risk subpopulations. *Journal of Exposure Analysis and Environmental Epidemiology*. Vol. 6(1): 79-95.
- Jones-Lee, M.W., M. Hammerton and P.R. Philips. 1985. The Value of Safety: Result of a National Sample Survey. *Economic Journal*. 95(March): 49-72.
- Jones-Lee, M.W. 1989. *The Economics of Safety and Physical Risk*. Oxford: Basil Blackwell.
- Jones-Lee, M.W., G. Loomes, D. O'Reilly, and P.R. Phillips. 1993. The Value of Preventing Non-fatal Road Injuries: Findings of a Willingness-to-pay National Sample Survey. TRY Working Paper, WP SRC2.
- Kleckner, N. and J. Neumann. 1999. "Recommended Approach to Adjusting WTP Estimates to Reflect Changes in Real Income. Memorandum to Jim Democker, US EPA/OPAR, June 3.
- Krewski D, Burnett RT, Goldbert MS, Hoover K, Siemiatycki J, Jerrett M, Abrahamowicz M, White WH. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute, Cambridge MA, July 2000
- Krupnick, A.J. and M.L. Cropper. 1992. "The Effect of Information on Health Risk Valuations." *Journal of Risk and Uncertainty* 5(2): 29-48.
- Krupnick, A.J., A. Alberini, M. Cropper, N. Simon, B. O'Brien, R. Goeree, and M. Heintzelman. 2000. Age, Health, and the Willingness to Pay for Mortality Risk Reductions: A Contingent Valuation Survey of Ontario Residents. Resources for the Future Discussion Paper 00-37.
- Kunzli, N., R. Kaiser, S. Medina, M. Studnicka, O. Chanel, P. Filliger, M. Herry, F. Horak Jr., V. Puybonnieux-Texier, P. Quenel, J. Schneider, R. Seethaler, J-C Vergnaud, and H. Sommer.

Draft Regulatory Impact Analysis

2000. Public-health Impact of Outdoor and Traffic-related Air Pollution: A European Assessment. *The Lancet*, 356: 795-801.
- Kunzli N, Medina S, Kaiser R, Quenel P, Horak F Jr, Studnicka M. 2001. Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies? *Am J Epidemiol* 153(11):1050-5
- Lareau, T.J. and D.A. Rae. 1989. Valuing WTP for Diesel Odor Reductions: An Application of Contingent Ranking Techniques, *Southern Economic Journal*, 55: 728- 742.
- Lave, L.B. and E.P. Seskin. 1977. *Air Pollution and Human Health*. Johns Hopkins University Press for Resources for the Future: Baltimore.
- Levy, J.I., J.K. Hammitt, Y. Yanagisawa, and J.D. Spengler. 1999. Development of a New Damage Function Model for Power Plants: Methodology and Applications. *Environmental Science and Technology*, 33: 4364-4372.
- Levy, J.I., T.J. Carrothers, J.T. Tuomisto, J.K. Hammitt, and J.S. Evans. 2001. Assessing the Public Health Benefits of Reduced Ozone Concentrations. *Environmental Health Perspectives*. 109: 1215-1226.
- Liao D, Cai J, Rosamond WD, Barnes RW, Hutchinson RG, Whitsel EA, Rautaharju P, Heiss G. 1997. Cardiac autonomic function and incident coronary heart disease: a population-based case-cohort study. The ARIC Study. Atherosclerosis Risk in Communities Study. *American Journal of Epidemiology*, 145(8):696-706.
- Liao D, Creason J, Shy C, Williams R, Watts R, Zweidinger R. 1999. Daily variation of particulate air pollution and poor cardiac autonomic control in the elderly. *Environ Health Perspect* 107:521-5
- Lipfert, F.W., S.C. Morris and R.E. Wyzga. 1989. Acid Aerosols - the Next Criteria Air Pollutant. *Environmental Science & Technology*. Vol. 23(11): 1316-1322.
- Lipfert, F.W. ; H. Mitchell Perry Jr ; J. Philip Miller ; Jack D. Baty ; Ronald E. Wyzga ; Sharon E. Carmody 2000. The Washington University-EPRI Veterans' Cohort Mortality Study: Preliminary Results, *Inhalation Toxicology*, 12: 41-74
- Lippmann, M., K. Ito, A. Nádas, and R.T. Burnett. 2000. Association of Particulate Matter Components with Daily Mortality and Morbidity in Urban Populations. Health Effects Institute Research Report Number 95, August.
- Magari SR, Hauser R, Schwartz J, Williams PL, Smith TJ, Christiani DC. 2001. Association of heart rate variability with occupational and environmental exposure to particulate air pollution. *Circulation* 104(9):986-91
- McClelland, G., W. Schulze, D. Waldman, J. Irwin, D. Schenk, T. Stewart, L. Deck, and M. Thayer. 1993. *Valuing Eastern Visibility: A Field Test of the Contingent Valuation Method*. Prepared for Office of Policy, Planning and Evaluation, US Environmental Protection Agency. September.
- McConnell, R., K. Berhane, F. Gilliland, S.J. London, H. Vora, E. Avol, W.J. Gauderman, H.G. Margolis, F. Lurmann, D.C. Thomas, and J.M. Peters. 1999. Air Pollution and Bronchitic Symptoms in Southern California Children with Asthma. *Environmental Health Perspectives*, 107(9): 757-760.

- McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. 2002. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359(9309):896.
- McDonnell, W.F., D.E. Abbey, N. Nishino and M.D. Lebowitz. 1999. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the ahsmog study. *Environmental Research*. 80(2 Pt 1): 110-21.
- Miller, T.R. 2000. Variations between Countries in Values of Statistical Life. *Journal of Transport Economics and Policy*. 34: 169-188.
- Moolgavkar SH, Luebeck EG, Anderson EL. 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis-St. Paul and Birmingham. *Epidemiology* 8:364-70
- Moolgavkar, S.H. 2000. Air pollution and hospital admissions for diseases of the circulatory system in three U.S. metropolitan areas. *J Air Waste Manag Assoc* 50:1199-206
- National Center for Education Statistics. 1996 The Condition of Education 1996, Indicator 42: Student Absenteeism and Tardiness. U.S. Department of Education National Center for Education Statistics. Washington DC.
- National Research Council (NRC). 1998. Research Priorities for Airborne Particulate Matter: I. Immediate Priorities and a Long-Range Research Portfolio. The National Academies Press: Washington, D.C.
- National Research Council (NRC). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. The National Academies Press: Washington, D.C.
- NCLAN. 1988. Assessment of Crop Loss from Air Pollutants. (Eds. Walter W. Heck, O. Clifton Taylor and David T. Tingey) Elsevier Science Publishing Co.: New York, Pp. 1-5. (ERL,GB 639).
- Neumann, J.E., M.T. Dickie, and R.E. Unsworth. 1994. Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis -- Draft Valuation Document. Industrial Economics Incorporated (IEc) Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. March 31.
- Norris, G., S.N. YoungPong, J.Q. Koenig, T.V. Larson, L. Sheppard and J.W. Stout. 1999. An association between fine particles and asthma emergency department visits for children in Seattle. *Environ Health Perspect*. Vol. 107(6): 489-93.
- Ostro, B.D. 1987. Air Pollution and Morbidity Revisited: a Specification Test. *Journal of Environmental Economics Management*. 14: 87-98.
- Ostro, B. and L. Chestnut. 1998. Assessing the Health Benefits of Reducing Particulate Matter Air Pollution in the United States. *Environmental Research, Section A*, 76: 94-106.
- Ostro B.D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity: An Observational Study of Multiple Pollutants. *Environmental Research* 50:238-247.
- Ostro, B.D., M.J. Lipsett, M.B. Wiener and J.C. Selner. 1991. Asthmatic Responses to Airborne Acid Aerosols. *Am J Public Health*. Vol. 81(6): 694-702.
- Ostro, B., M. Lipsett, J. Mann, H. Braxton-Owens and M. White. 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiology*. Vol. 12(2): 200-8.

Draft Regulatory Impact Analysis

- Ozkaynak, H. and G.D. Thurston. 1987. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. *Risk Anal.* Vol. 7(4): 449-61.
- Peters A, Dockery DW, Muller JE, Mittleman MA. 2001. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation.* 103:2810-2815.
- Poloniecki JD, Atkinson RW, de Leon AP, Anderson HR. 1997. Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occup Environ Med* 54(8):535-40.
- Pope, C.A. 2000. Invited Commentary: Particulate Matter-Mortality Exposure-Response Relations and Thresholds. *American Journal of Epidemiology*, 152: 407-412.
- Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, G.D. Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. *Journal of the American Medical Association.* 287: 1132-1141.
- Pope, C.A., III, M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer, and C.W. Heath, Jr. 1995. Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults. *American Journal of Respiratory Critical Care Medicine* 151: 669-674.
- Pope, C.A., III, D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. Respiratory Health and PM₁₀ Pollution: a Daily Time Series Analysis *American Review of Respiratory Diseases* 144: 668-674.
- Ransom, M.R. and C.A. Pope. 1992. Elementary School Absences and PM(10) Pollution in Utah Valley. *Environmental Research.* Vol. 58(2): 204-219.
- Rosamond, W., G. Broda, E. Kawalec, S. Rywik, A. Pajak, L. Cooper and L. Chambless. 1999. Comparison of medical care and survival of hospitalized patients with acute myocardial infarction in Poland and the United States. *American Journal of Cardiology.* 83: 1180-5.
- Rossi G, Vigotti MA, Zanobetti A, Repetto F, Gianelle V, Schwartz J. 1999. Air pollution and cause-specific mortality in Milan, Italy, 1980-1989. *Arch Environ Health* 54(3):158-64
- Rowlatt et al. 1998. Valuation of Deaths from Air Pollution. NERA and CASPAR for DETR.
- Russell, M.W., D.M. Huse, S. Drowns, E.C. Hamel and S.C. Hartz. 1998. Direct medical costs of coronary artery disease in the United States. *Am J Cardiol.* Vol. 81(9): 1110-5.
- Samet, J.M., S.L. Zeger, J.E. Kelsall, J. Xu and L.S. Kalkstein. 1997. Air Pollution, Weather, and Mortality in Philadelphia 1973-1988. Health Effects Institute. Cambridge, MA. March.
- Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW, Schwartz J, Zanobetti A. 2000. The National Morbidity, Mortality and Air Pollution Study: Part II: Morbidity, Mortality and Air Pollution in the United States. Research Report No. 94, Part II. Health Effects Institute, Cambridge MA, June 2000.
- Schwartz, J., Dockery, D.W., Neas, L.M., Wypij, D., Ware, J.H., Spengler, J.D., Koutrakis, P., Speizer, F.E., and Ferris, Jr., B.G. 1994. Acute Effects of Summer Air Pollution on Respiratory Symptom Reporting in Children *American Journal of Respiratory Critical Care Medicine* 150: 1234-1242.
- Schwartz J, Laden F, Zanobetti A. 2002. The concentration-response relation between PM(2.5) and daily deaths. *Environmental Health Perspectives* 110:1025-9
- Schwartz J. 2000. The distributed lag between air pollution and daily deaths. *Epidemiology.* 2000 May;11(3):320-6.

- Schwartz, J. 2000. Assessing confounding, effect modification, and thresholds in the association between ambient particles and daily deaths. *Environmental Health Perspectives* 108(6): 563-8.
- Schwartz, J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. *Thorax* 50(5):531-8
- Schwartz, J. 1993. Particulate Air Pollution and Chronic Respiratory Disease *Environmental Research* 62: 7-13.
- Schwartz J, Dockery DW, Neas LM. 1996. Is daily mortality associated specifically with fine particles? *J Air Waste Manag Assoc.* 46:927-39.
- Schwartz J and Zanobetti A. 2000. Using meta-smoothing to estimate dose-response trends across multiple studies, with application to air pollution and daily death. *Epidemiology.* 11:666-72.
- Schwartz J, Neas LM. 2000. Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. *Epidemiology* 11:6-10.
- Seigneur, C., G. Hidy, I. Tombach, J. Vimont, and P. Amar. 1999. Scientific Peer Review of the Regulatory Modeling System for Aerosols and Deposition (REMSAD). Prepared for the KEVRIC Company, Inc.
- Sheppard, L., D. Levy, G. Norris, T.V. Larson and J.Q. Koenig. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. *Epidemiology.* Vol. 10: 23-30.
- Sisler, J.F. 1996. Spatial and Seasonal Patterns and Long Term Variability of the Composition of the Haze in the United States: An Analysis of Data from the IMPROVE Network. Cooperative Institute for Research in the Atmosphere, Colorado State University; Fort Collins, CO July.
- Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista and W.B. Saunders. 1997. A national estimate of the economic costs of asthma. *Am J Respir Crit Care Med.* 156(3 Pt 1): 787-93.
- Smith, V. K., G. Van Houtven, and S.K. Pattanayak. 2002. Benefit Transfer via Preference Calibration. *Land Economics.* 78: 132-152.
- Stanford, R., T. McLaughlin and L.J. Okamoto. 1999. The cost of asthma in the emergency department and hospital. *Am J Respir Crit Care Med.* Vol. 160(1): 211-5.
- Stieb, D.M., R.T. Burnett, R.C. Beveridge and J.R. Brook. 1996. Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. *Environmental Health Perspectives.* Vol. 104(12): 1354-1360.
- Stieb DM, Judek S, Burnett RT. 2002. Meta-analysis of time-series studies of air pollution and mortality: effects of gases and particles and the influence of cause of death, age, and season. *J Air Waste Manag Assoc* 52(4):470-84
- Taylor, C.R., K.H. Reichelderfer, and S.R. Johnson. 1993. *Agricultural Sector Models for the United States: Descriptions and Selected Policy Applications.* Iowa State University Press: Ames, IA.
- Thurston, G.D. and K. Ito. 2001. Epidemiological studies of acute ozone exposures and mortality. *J Expo Anal Environ Epidemiol.* Vol. 11(4): 286-94.
- Tolley, G.S. et al. 1986. Valuation of Reductions in Human Health Symptoms and Risks. University of Chicago. Final Report for the US Environmental Protection Agency. January.

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- Tsuji H, Larson MG, Venditti FJ Jr, Manders ES, Evans JC, Feldman CL, Levy D. 1996. Impact of reduced heart rate variability on risk for cardiac events. The Framingham Heart Study. *Circulation* 94(11):2850-5
- US Bureau of the Census. 2002. Statistical Abstract of the United States: 2001. Washington DC.
- US Department of Commerce, Bureau of Economic Analysis. BEA Regional Projections to 2045: Vol. 1, States. Washington, DC US Govt. Printing Office, July 1995.
- US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. 1999. National Vital Statistics Reports, 47(19).
- US Environmental Protection Agency. 2002. Third External Review Draft of Air Quality Criteria for Particulate Matter (April, 2002): Volume II. EPA/600/P-99/002aC
- US Environmental Protection Agency. 2003a. Emissions Inventory Technical Support Document for the Proposed Nonroad Diesel Engines Rule.
- US Environmental Protection Agency. 2003b. Air Quality Technical Support Document for the Proposed Nonroad Diesel Engines Rule.
- US Environmental Protection Agency, 1996a. *Review of the National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information*. Office of Air Quality Planning and Standards, Research Triangle Park, NC EPA report no. EPA/4521R-96-007.
- US Environmental Protection Agency, 1996b. *Review of the National Ambient Air Quality Standards for Particulate Matter: Assessment of Scientific and Technical Information*. Office of Air Quality Planning and Standards, Research Triangle Park, NC EPA report no. EPA/4521R-96-013.
- US Environmental Protection Agency, 1999. *The Benefits and Costs of the Clean Air Act, 1990-2010*. Prepared for US Congress by US EPA, Office of Air and Radiation/Office of Policy Analysis and Review, Washington, DC, November; EPA report no. EPA-410-R-99-001.
- US Environmental Protection Agency, 1993. External Draft, Air Quality Criteria for Ozone and Related Photochemical Oxidants. Volume II. US EPA, Office of Health and Environmental Assessment. Research Triangle Park, NC, EPA/600/AP-93/004b.3v.
- US Environmental Protection Agency, 2000a. *Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements*. Prepared by: Office of Air and Radiation. Available at <http://www.epa.gov/otaq/diesel.htm> Accessed March 20, 2003.
- US Environmental Protection Agency, 2000b. *Valuing Fatal Cancer Risk Reductions*. White Paper for Review by the EPA Science Advisory Board.
- US Environmental Protection Agency 2000c. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003. September.
- US Environmental Protection Agency, 1997. *The Benefits and Costs of the Clean Air Act, 1970 to 1990*. Prepared for US Congress by US EPA, Office of Air and Radiation/Office of Policy Analysis and Review, Washington, DC
- US Environmental Protection Agency, 2002. Technical Addendum: Methodologies for the Benefit Analysis of the Clear Skies Initiative. September. Available online at http://www.epa.gov/air/clearskies/tech_adden.pdf. Accessed March 20, 2003.

- US Office of Management and Budget. 1992. Guidelines and Discount Rates for Benefit-Cost Analysis of Federal Programs. Circular No. A-94. October.
- Vedal, S., J. Petkau, R. White and J. Blair. 1998. Acute effects of ambient inhalable particles in asthmatic and nonasthmatic children. *American Journal of Respiratory and Critical Care Medicine*. Vol. 157(4): 1034-1043.
- Viscusi, W.K. 1992. *Fatal Tradeoffs: Public and Private Responsibilities for Risk*. (New York: Oxford University Press).
- Viscusi, W.K., W.A. Magat, and J. Huber. 1991. "Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-Dollar Trade-Offs for Chronic Bronchitis" *Journal of Environmental Economics and Management*, 21: 32-51.
- Weisel, C.P., R.P. Cody and P.J. Lioy. 1995. Relationship between summertime ambient ozone levels and emergency department visits for asthma in central New Jersey. *Environ Health Perspect*. Vol. 103 Suppl 2: 97-102.
- Whittemore, A.S. and E.L. Korn. 1980. Asthma and Air Pollution in the Los Angeles Area. *American Journal of Public Health*. 70: 687-696.
- Wittels, E.H., J.W. Hay and A.M. Gotto, Jr. 1990. Medical costs of coronary artery disease in the United States. *Am J Cardiol*. Vol. 65(7): 432-40.
- Woodruff, T.J., J. Grillo and K.C. Schoendorf. 1997. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environmental Health Perspectives*. Vol. 105(6): 608-612.
- Woods & Poole Economics Inc. 2001. Population by Single Year of Age CD. Woods & Poole Economics, Inc.
- Yu, O., L. Sheppard, T. Lumley, J.Q. Koenig and G.G. Shapiro. 2000. Effects of Ambient Air Pollution on Symptoms of Asthma in Seattle-Area Children Enrolled in the CAMP Study. *Environ Health Perspect*. Vol. 108(12): 1209-1214.
- Zanobetti, A., J. Schwartz, E. Samoli, A. Gryparis, G. Touloumi, R. Atkinson, A. Le Tertre, J. Bobros, M. Celko, A. Goren, B. Forsberg, P. Michelozzi, D. Rabczenko, E. Aranguiz Ruiz and K. Katsouyanni. 2002. The temporal pattern of mortality responses to air pollution: a multicity assessment of mortality displacement. *Epidemiology*. Vol. 13(1): 87-93.

**APPENDIX 9B:
Sensitivity Analyses of Key Parameters in the Benefits Analysis**

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The Base Estimate is based on our 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, we recognize that 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, we supplement our Base Estimate of benefits with a series of sensitivity calculations that make use of other sources of concentration-response and valuation data for key benefits categories. These sensitivity calculations are conducted relative to the Base Estimate and not for the Alternative Estimate. The sensitivity estimates can be used to answer questions like "What would total benefits be if we were to value avoided incidences of premature mortality using the age-dependent VSL rather than the age-independent VSL approach?" These estimates examine sensitivity to both valuation issues (e.g. the correct value for a statistical life saved) and for physical effects issues (e.g., possible recovery from chronic illnesses). 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 table should not be added together without addressing potential issues of overlap and low joint probability among the endpoints. Additional sensitivity estimates are provided in the benefits TSD (Abt Associates, 2003).

9B.1 Premature Mortality—Long term exposure

Arguably, reduction in the risk of premature mortality is the most important PM-related health outcome in terms of public health significance and 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 9B.1.

9B.1.1 Alternative C-R Functions

Although we used the Krewski, et al. (2000) mean-based ("PM2.5(DC), All Causes") model 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. We used two sources of alternative C-R functions for this sensitivity analysis: (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]

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study had better monitoring with less measurement error than did most other studies" (EPA-SAB-COUNCIL-ADV-99-012, 1999). However, the Krewski-Harvard Six Cities study had a more limited geographic scope (and a smaller study population) than the Krewski-ACS study. The demographics of the ACS study population, i.e., largely white and middle-class, may also produce a downward bias in the estimated PM mortality coefficient, because short-term studies indicate that the effects of PM tend to be significantly greater among groups of lower socioeconomic status. The Krewski-Harvard Six Cities study also covered a broader age category (25 and older compared to 30 and older in the ACS study) and followed the cohort for a longer period (15 years compared to 8 years in the ACS study). The HEI commentary notes that "the inherent limitations of using only six cities, understood by the original investigators, should be taken into account when interpreting the results of the Six Cities Study." We emphasize, that based on our understanding of the relative merits of the two datasets, the Krewski, et al. (2000) ACS model based on mean PM_{2.5} levels in 63 cities is the most appropriate model for analyzing the premature mortality impacts of the nonroad standards. It is thus used for our primary estimate of this important health effect. In addition to these alternative C-R functions, a broader set of alternative mortality C-R functions is examined in the benefits TSD (Abt Associates, 2003).

9B.1.2 Alternative Lag Structures

As noted by the SAB (EPA-SAB-COUNCIL-ADV-00-001, 1999), "some of the mortality effects of cumulative exposures will occur over short periods of time in individuals with compromised health status, but other effects are likely to occur among individuals who, at baseline, have reasonably good health that will deteriorate because of continued exposure. No animal models have yet been developed to quantify these cumulative effects, nor are there epidemiologic studies bearing on this question." However, they also note that "Although there is substantial evidence that a portion of the mortality effect of PM is manifest within a short period of time, i.e., less than one year, it can be argued that, if no a lag assumption is made, the entire mortality excess observed in the cohort studies will be analyzed as immediate effects, and this will result in an overestimate of the health benefits of improved air quality. Thus some time lag is appropriate for distributing the cumulative mortality effect of PM in the population." In the primary analysis, based on SAB advice, we assume that mortality occurs over a five year period, with 25 percent of the deaths occurring in the first year, 25 percent in the second year, and 16.7 percent in each of the third, fourth, and fifth years. Readers should note that the selection of a 5 year lag is not supported by any scientific literature on PM-related mortality. 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. The 0 year lag is representative of EPA's assumption in previous RIAs. The 8 and 15 year lags are based on the study periods from the Pope, et al. (1995) and Dockery, et al. (1993) studies,

respectively^{dd}. However, neither the Pope, et al. or Dockery, et al studies assumed any lag structure when estimating the relative risks from PM exposure. In fact, the Pope, et al. and Dockery, et al. studies do not contain any data either supporting or refuting the existence of a lag. Therefore, any lag structure applied to the avoided incidences estimated from either of these studies will be an assumed structure. The 8 and 15 year lags implicitly assume that all premature mortalities occur at the end of the study periods, i.e. at 8 and 15 years. 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 9B.2. These estimates are based on the value of statistical lives saved approach, i.e. \$6 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.

9B.1.3 Age and VSL

The relationship between age and willingness to pay for mortality risk reductions has been the subject of much research over the past several years. Recent research in the U.S. has not found a significant reduction in WTP for risk reductions in older populations (Smith et al. 2002; Alberini et al., 2002; Schultze, 2002). Studies outside of the U.S. have found a significant reduction in WTP for older individuals, ranging from 10 percent (Jones-Lee, 1993) to around 35 percent (Alberini et al. 2002) for a 70 year old, relative to a 40 year old. Around 80 percent of the deaths projected to be avoided from reduced exposure to PM in 2020 and 2030 are in populations over 65. As such, the assumption that populations of all ages have the same VSL can have a significant impact on the total benefits. For this sensitivity analysis, the method we use to account for age differences is to adjust the base \$6.1 million VSL based on ratios of VSL's for specific ages to the VSL for a 40 year old individual. There are several potential sources for these ratios. Two Jones-Lee studies to provide evidence of strong and weak age effects on WTP for mortality risk reductions. The ratios based on Jones-Lee (1989), as summarized in U.S. EPA (2000), suggest a steep inverted U shape between age and VSL, with the VSL for a 70 year old at 63 percent of that for a 40 year old, and the VSL for an 85 year old at 7 percent of that for 40 year old. The ratios based on Jones-Lee (1993) and summarized in U.S. EPA (2000), suggest a much flatter inverted U shape, with the VSL for a 70 year old at 92 percent of that for a 40 year old, and the VSL for an 85 year old at 82 percent of that for a 40 year old. Recent analyses conducted in Canada and the U.S. by Krupnick et al. (2000a, 2000b) found mixed results. The Canadian analysis found around a 35 percent reduction in VSL for respondents over age 70, but the U.S. analysis found no significant differences in VSL across ages. The wide range of age-

^{DD} Although these studies were conducted for 8 and 15 years, respectively, the choice of the duration of the study by the authors was not likely due to observations of a lag in effects, but is more likely due to the expense of conducting long-term exposure studies or the amount of satisfactory data that could be collected during this time period.

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adjustment ratios, especially at older ages demonstrates the difficulty in making these kinds of adjustments. We select the recent Krupnick et al results for Canada as the basis for calculating age-specific VSL, because it uses state of the art stated preference methods and reflects more current preferences. We note that our Base estimate is the most consistent with current evidence on U.S. preferences for risk reduction in older populations. To calculate benefits using the age-adjusted VSL, we first calculate the number of avoided premature mortalities in each age category, and then apply the age adjusted VSL to the appropriate incidences in each age category^{ee}.

9B.1.4 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, as discussed in section 9A, virtually every study to consider the issue indicates absence of a threshold. Nonetheless, 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. If, for example, there were no benefits of reducing PM concentrations below the PM_{2.5} standard of 15 : g/m³, our estimate of the total number of avoided PM-related premature mortalities in 2030 would be reduced by approximately 70 percent, from approximately 11,000 annually to approximately 3,200 annually. However, this type of cutoff is unlikely, as supported by the recent NRC report, which stated that “for pollutants such as PM₁₀ 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)”

One important assumption that we 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 NRC, “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 NRC suggested that the treatment of thresholds should be evaluated in a formal uncertainty analysis. As noted in earlier sections, EPA is developing a formal

^{EE} The age categories and lower and upper end estimated age-adjustment ratios are: 30-39 (0.89, 0.98), 40-59 (1.0, 1.0), 60-69 (0.86, 0.97), 70-79 (0.63, 0.92), 80-84 (0.28, 0.85), 85+ (0.07, 0.82).

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uncertainty analysis processes which we intend to at least partially implement for the analysis of the final rule.

Table 9B-1.
Sensitivity of Estimates to Alternative Assumptions Regarding Quantification of Mortality Benefits

Description of Sensitivity Analysis	Avoided Incidences ^A		Value (million 2000\$) ^B		
	2020	2030	2020	2030	
Alternative Concentration-Response Functions for PM-related Premature Mortality					
Pope/ACS Study (2002)					
All Cause	5,400	9,500	\$41,000	\$74,000	
<i>Lung Cancer</i>	740	1,300	\$5,600	\$9,900	
<i>Cardiopulmonary</i>	4,000	7,200	\$30,000	\$55,000	
Krewski/Harvard Six-city Study	18,000	32,000	\$140,000	\$240,000	
Alternative Lag Structures for PM-related Premature Mortality (3% discount rate)					
None	Incidences all occur in the first year	6,200	11,000	\$49,000	\$89,000
8-year	Incidences all occur in the 8 th year	6,200	11,000	\$40,000	\$72,000
15-year	Incidences all occur in the 15 th year	6,200	11,000	\$33,000	\$59,000
Alternative Mortality Risk Valuation Based on Age Specific VSL					
VSL applied to statistical deaths avoided in populations 70 and over equal to 65% of VSL for avoided deaths in populations under 70	6,200	11,000	\$36,000	\$63,000	
Alternative Thresholds					
No Threshold (base estimate)	6,200	11,000	\$47,000	\$85,000	
5	6,200	11,000	\$47,000	\$85,000	
10	5,000	9,400	\$38,000	\$72,000	
15	1,300	3,200	\$10,000	\$25,000	
20	500	1,000	\$3,800	\$8,000	
25	150	430	\$1,100	\$3,300	

^A Incidences rounded to two significant digits.

^B Dollar values rounded to two significant digits.

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

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reanalysis of the Harvard Six-cities data (Krewski et al., 2000). 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. 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 : g/m³, and around 20 percent are due to changes above 15 : g/m³, the current PM_{2.5} standard.

9B.2 Other Health Endpoint Sensitivity Analyses

9B.2.1 Overlapping Endpoints

In Appendix 9A, 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 concentration-response (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 in this appendix 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 Table 9B.2.

The benefit estimates presented in the Alternative estimate in Tables 9A-30 and 9A-31 of Appendix 9A do not capture any additional short-term mortality impacts related to changes in exposure to ambient ozone. A recent analysis by Thurston and Ito (2001) reviewed previously published time series studies of the effect of daily ozone levels on daily mortality and found that previous EPA estimates of the short-term mortality benefits of the ozone NAAQS (U.S. EPA, 1997) may have been underestimated by up to a factor of two. The authors hypothesized that much of the variability in published estimates of the ozone/mortality effect could be explained by how well each model controlled for the influence of weather, an important confounder of the ozone/mortality effect, and that earlier studies using less sophisticated approaches to controlling for weather consistently under-predicted the ozone/mortality effect. They found that models incorporating a non-linear temperature specification appropriate for the "U-shaped" nature of the temperature/mortality relationship (i.e., increased deaths at

both very low and very high temperatures) produced ozone/mortality effect estimates that were both more strongly positive (a two percent increase in relative risk over the pooled estimate for all studies evaluated) and consistently statistically significant. Further accounting for the interaction effects between temperature and relative humidity produced even more strongly positive results. Inclusion of a PM index to control for PM/mortality effects had little effect on these results, suggesting an ozone/mortality relationship independent of that for PM. However, most of the studies examined by Ito and Thurston only controlled for PM₁₀ or broader measures of particles and did not directly control for PM_{2.5}. As such, there may still be potential for confounding of PM_{2.5} and ozone mortality effects, as ozone and PM_{2.5} are highly correlated during summer months in some areas. In its September 2001 advisory on the draft analytical blueprint for the second Section 812 prospective analysis, the SAB cited the Thurston and Ito study as a significant advance in understanding the effects of ozone on daily mortality and recommended re-evaluation of the ozone mortality endpoint for inclusion in the next prospective study (EPA-SAB-COUNCIL-ADV-01-004, 2001). Thus, recent evidence suggests that by not including an estimate of reductions in short-term mortality due to changes in ambient ozone, both the Base and Alternative Estimates may underestimate the benefits of implementation of the Nonroad Diesel Engine rule.

There are many studies of the relationship between ambient ozone levels and daily mortality levels. The ozone mortality sensitivity estimate is calculated using results from only four U.S. studies (Ito and Thurston, 1996; Kinney et al., 1995; Moolgavkar et al., 1995; and Samet et al., 1997), based on the assumption that demographic and environmental conditions on average would be more similar between these studies and the conditions prevailing when the nonroad standards are implemented. However, the full body of peer-reviewed ozone mortality studies should be considered when evaluating the weight of evidence regarding the presence of an association between ambient ozone concentrations and premature mortality. We combined these studies using probabilistic sampling methods to estimate the impact of ozone on mortality incidence. The technical support document for this analysis provides additional details of this approach (Abt Associates, 2003). The estimated incidences of short-term premature mortality are valued using the value of statistical lives saved method, as described in Appendix 9A.

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**Table 9B-2.
Sensitivity Estimates for Potentially Overlapping Endpoints^A**

Description of Sensitivity Analysis	Avoided Incidences		Monetized Value (Million 2000\$)	
	2020	2030	2020	2030
Mortality from Short-term Ozone Exposure^B				
Ito and Thurston (1996)	440	1,000	\$3,500	\$8,100
Kinney et al. (1995)	0	0	\$0	\$0
Moolgavkar et al. (1995)	77	240	\$620	\$1,900
Samet et al. (1997)	120	360	\$960	\$2,900
Pooled estimate (random effects weights)	94	280	\$750	\$2,300
Any of 19 Acute Respiratory Symptoms, Adults 18-64 (Krupnick et al. 1990)				
Ozone	1,500,000	2,800,000	\$38	\$71
PM	14,000,000	19,000,000	\$340	\$490

^A All estimates rounded to two significant digits.

^B Mortality valued using Base estimate of \$6.3 million per premature statistical death, adjusted for income growth.

9B.2.2 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 9B.3, 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.

**Table 9B-3.
Additional Parameter Sensitivity Analyses**

Alternative Calculation		Description of Estimate	Impact on Base Benefit Estimate (million 2000\$)	
			2020	2030
1	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 35 in 2020 and 52 in 2030	+\$270 (+0.5%)	+\$400 (+0.4%)
2	Chronic Asthma	Avoided incidences of chronic asthma are estimated using the McDonnell, et al. (1999) C-R function. The number of avoided incidences of chronic asthma is 1,200 in 2020 and 2,400 in 2030	+\$36 (+0.1%)	+\$74 (+0.1%)
3	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 in 2020 increases from 4,300 to 8,000 (87%). The increase in 2030 is from 6,500 to 12,000 (87%).	+\$730 (+1.4%)	+\$1,100 (+1.2%)
4	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.	+\$640 (+1.2%)	+\$970 (+1.1%)
5	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).	+\$700 (+1.3%)	+\$1,100 (+1.1%)
6	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).	+\$530 (+1.0%)	+\$830 (+0.9%)
7	Household soiling damage	Value of decreases in expenditures on cleaning are estimated using values derived from Manuel, et al. (1983).	+\$170 (+0.3%)	+\$260 (+0.3%)

The estimated effect of PM exposure on premature mortality in post neo-natal infants (row 1 of Table 9B.3) is based on a single U.S. study (Woodruff et al.,1997) which, on SAB advice, was

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deemed too uncertain to include in the primary analysis. Adding this endpoint to the primary benefits estimate would result in an increase in total benefits. The infant mortality estimate indicates that exclusion of this endpoint does not have a large relative impact, either in terms of incidences (35 in 2020 and 52 in 2030) or monetary value (approximately \$270 million in 2020 and \$400 million in 2030).

The alternative calculation for the development of chronic asthma (row 2 of Table 9B.3) is estimated using a recent study by McDonnell, et al. (1999) which found a statistical association between ozone and the development of asthma in adult white, non-Hispanic males. Other studies have not identified an association between air quality and the onset of asthma. Chronic asthma is characterized by repeated incidences of inflammation of the lungs. This causes restriction in the airways and results in shortness of breath, wheezing, and coughing. Asthma is also characterized by airway hyper responsiveness to stimuli.

The McDonnell, et al. study is a prospective cohort analysis, measuring the association between long-term exposure to ambient concentrations of ozone and development of chronic asthma in adults. The study found a statistically significant effect for adult males, but none for adult females. EPA also believes it to be appropriate to apply the C-R function to all adult males over age 27 because no evidence exists to suggest that non-white adult males have a lower responsiveness to air-pollution. For other health effects such as shortness of breath, where the study population was limited to a specific group potentially more sensitive to air pollution than the general population (Ostro et al., 1991), EPA has applied the C-R function only to the limited population.

Some commentors have raised questions about the statistical validity of the associations found in this study and the appropriateness of transferring the estimated C-R function from the study populations (white, non-Hispanic males) to other male populations (i.e. African-American males). Some of these concerns include the following: 1) no significant association was observed for female study participants also exposed to ozone; 2) the estimated C-R function is based on a cross-sectional comparison of ozone levels, rather than incorporating information on ozone levels over time; 3) information on the accuracy of self-reported incidence of chronic asthma was collected but not used in estimating the C-R function; 4) the study may not be representative of the general population because it included only those individuals living 10 years or longer within 5 miles of their residence at the time of the study; and 5) the study had a significant number of study participants drop out, either through death, loss of contact, or failure to provide complete or consistent information. EPA believes that while these issues may result in increased uncertainty about this effect, none can be identified with a specific directional bias in the estimates. In addition, the SAB reviewed the study and deemed it appropriate for quantification of changes in ozone concentrations in benefits analyses (EPA-SAB-COUNCIL-ADV-00-001, 1999). EPA recognizes the need for further investigation by the scientific community to confirm the statistical association identified in the McDonnell, et al. study.

Following SAB advice (EPA-SAB-COUNCIL-ADV-00-001, 1999) and consistent with the Section 812 Prospective Report, we quantify this endpoint for the RIA. However, it should be noted

that it is not clear that the intermittent, short-term, and relatively small changes in annual average ozone concentrations resulting from this rule alone are likely to measurably change long-term risks of asthma.

Similar to the valuation of chronic bronchitis, WTP to avoid chronic asthma is presented as the net present value of what would potentially be a stream of costs and lower well-being incurred over a lifetime. Estimates of WTP to avoid asthma are provided in two studies, one by Blumenschein and Johannesson (1998) and one by O’Conor and Blomquist (1997). Both studies use the contingent valuation method to solicit annual WTP estimates from individuals who have been diagnosed as asthmatics. The central estimate of lifetime WTP to avoid a case of chronic asthma among adult males, approximately \$25,000, is the average of the present discounted value from the two studies. Details of the derivation of this central estimate from the two studies is provided in the benefits TSD for this RIA (Abt Associates, 2003).

Another important issue related to chronic conditions is the possible reversal in chronic bronchitis incidences (row 3 of Table 9B.3). Reversals are defined as those cases where an individual reported having chronic bronchitis at the beginning of the study period but reported not having chronic bronchitis in follow-up interviews at a later point in the study period. Since, by definition, chronic diseases are long-lasting or permanent, if the disease goes away it is not chronic. However, we have not captured the benefits of reducing incidences of bronchitis that are somewhere in-between acute and chronic. One way to address this is to treat reversals as cases of chronic bronchitis that are at the lowest severity level. These cases thus get the lowest value for chronic bronchitis.

The alternative calculation for recreational visibility (row 4 of Table 9B.3) is an estimate of the full value of visibility in the entire region affected by the nonroad emission reductions. The Chestnut and Rowe study from which the primary valuation estimates are derived only examined WTP for visibility changes in the southeastern portion of the affected region. In order to obtain estimates of WTP for visibility changes in the northeastern and central portion of the affected region, we have to transfer the southeastern WTP values. This introduces additional uncertainty into the estimates. However, we have taken steps to adjust the WTP values to account for the possibility that a visibility improvement in parks in one region, is not necessarily the same environmental quality good as the same visibility improvement at parks in a different region. This may be due to differences in the scenic vistas at different parks, uniqueness of the parks, or other factors, such as public familiarity with the park resource. To take this potential difference into account, we adjusted the WTP being transferred by the ratio of visitor days in the two regions.

The alternative calculations for residential visibility (rows 5 and 6 of Table 9B.3) are based on the McClelland, et al. study of WTP for visibility changes in Chicago and Atlanta. As discussed in Appendix 9A, SAB advised EPA that the residential visibility estimates from the available literature are inadequate for use in a primary estimate in a benefit-cost analysis. However, EPA recognizes that residential visibility is likely to have some value and the McClelland, et al. estimates are the most useful in providing an estimate of the likely magnitude of the benefits of residential visibility improvements.

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The alternative calculation for household soiling (row 7 of Table 9B.3) is based on the Manuel, et al. study of consumer expenditures on cleaning and household maintenance. This study has been cited as being “the only study that measures welfare benefits in a manner consistent with economic principals (Desvouges et al., 1998).” However, the data used to estimate household soiling damages in the Manuel, et al. study are from a 1972 consumer expenditure survey and as such may not accurately represent consumer preferences in 2030. EPA recognizes this limitation, but believes the Manuel, et al. estimates are still useful in providing an estimate of the likely magnitude of the benefits of reduced PM household soiling.

9B.3 Income Elasticity of Willingness to Pay

As discussed in Appendix 9A, our estimate of monetized benefits accounts for growth in real GDP per capita by adjusting the WTP for individual endpoints based on the central estimate of the adjustment factor for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility). We examine how sensitive the estimate of total benefits is to alternative estimates of the income elasticities. Table 9B.4 lists the ranges elasticity values used to calculate the income adjustment factors, while Table 9B.5 lists the ranges of corresponding adjustment factors. The results of this sensitivity analysis, giving the monetized benefit subtotals for the four benefit categories, are presented in Table 9B.6.

Consistent with the impact of mortality on total benefits, the adjustment factor for mortality has the largest impact on total benefits. The value of mortality ranges from 81 percent to 150 percent of the primary estimate based on the lower and upper sensitivity bounds on the income adjustment factor. The effect on the value of minor and chronic health effects is much less pronounced, ranging from 93 percent to 111 percent of the primary estimate for minor effects and from 88 percent to 110 percent for chronic effects.

Table 9B-4.
Ranges of Elasticity Values Used to Account for Projected Real Income Growth^A

Benefit Category	Lower Sensitivity Bound	Upper Sensitivity Bound
Minor Health Effect	0.04	0.30
Severe and Chronic Health Effects	0.25	0.60
Premature Mortality	0.08	1.00
Visibility ^B	--	--

^A Derivation of these ranges can be found in Kleckner and Neumann (1999) and Chestnut (1997). Cost of Illness (COI) estimates are assigned an adjustment factor of 1.0.

^B No range was applied for visibility because no ranges were available in the current published literature.

Table 9B-5.
Ranges of Adjustment Factors Used to Account for Projected Real Income Growth^A

Benefit Category	Lower Sensitivity Bound		Upper Sensitivity Bound	
	2020	2030	2020	2030
Minor Health Effect	1.023	1.025	1.190	1.208
Severe and Chronic Health Effects	1.156	1.170	1.420	1.464
Premature Mortality	1.047	1.052	1.814	1.914
Visibility ^B	--	--	--	--

^A Based on elasticity values reported in Table 9A-11, US Census population projections, and projections of real gross domestic product per capita.

^B No range was applied for visibility because no ranges were available in the current published literature.

Table 9B-6.
Sensitivity Analysis of Alternative Income Elasticities^A

Benefit Category	Lower Sensitivity Bound		Upper Sensitivity Bound	
	2020	2030	2020	2030
Minor Health Effect	\$1,400	\$2,200	\$1,400	\$2,200
Severe and Chronic Health Effects (base estimate)	\$1,700	\$2,600	\$2,100	\$3,300
Premature Mortality (base estimate)	\$38,000	\$67,000	\$67,000	\$130,000
Visibility and Other Welfare Effects ^A	\$1,500	\$2,400	\$1,500	\$2,400
Total Benefits	\$43,000	\$75,000	\$72,000	\$130,000

^A All estimates rounded to two significant digits.

^B No range was applied for visibility because no ranges were available in the current published literature.

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- Abt Associates, Inc. 2003. *Proposed Nonroad Landbased Diesel Engine Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results*. Prepared for Office of Air Quality Planning and Standards, U.S. EPA. April, 2003.
- Alberini, A., M. Cropper, A. Krupnick, and N.B. Simon. 2002. Does the Value of a Statistical Life Vary with Age and Health Status? Evidence from the United States and Canada. Resources for the Future Discussion Paper 02-19. April.
- Blumenschein, K. and M. Johannesson. 1998. "Relationship Between Quality of Life Instruments, Health State Utilities, and Willingness to Pay in Patients with Asthma." *Annals of Allergy, Asthma, and Immunology* 80:189-194.
- Chestnut, L.G. 1997. Draft Memorandum: *Methodology for Estimating Values for Changes in Visibility at National Parks*. April 15.
- Chestnut, L.G. and R.D. Rowe. 1990a. *Preservation Values for Visibility Protection at the National Parks: Draft Final Report*. Prepared for Office of Air Quality Planning and Standards, US Environmental Protection Agency, Research Triangle Park, NC and Air Quality Management Division, National Park Service, Denver, CO.
- Chestnut, L.G., and R.D. Rowe. 1990b. A New National Park Visibility Value Estimates. In *Visibility and Fine Particles*, Transactions of an AWMA/EPA International Specialty Conference, C.V. Mathai, ed. Air and Waste Management Association, Pittsburgh.
- Desvousges, W.H., F. R. Johnson, H.S. Banzhaf. 1998. *Environmental Policy Analysis With Limited Information: Principles and Applications of the Transfer Method (New Horizons in Environmental Economics)*. Edward Elgar Pub: London.
- Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris and F.E. Speizer. 1993. "An association between air pollution and mortality in six U.S. cities." *New England Journal of Medicine*. 329(24): 1753-1759.
- EPA-SAB-COUNCIL-ADV-00-001, 1999. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects; Part 2. October.
- EPA-SAB-COUNCIL-ADV-99-012, 1999. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects; Part 1. July.
- EPA-SAB-COUNCIL-ADV-01-004. 2001. Review of the Draft Analytical Plan for EPA's Second Prospective Analysis - Benefits and Costs of the Clean Air Act 1990-2020: An Advisory by a Special Panel of the Advisory Council on Clean Air Compliance Analysis. September.
- Ito, K. and G.D. Thurston. 1996. "Daily PM10/mortality associations: an investigations of at-risk subpopulations." *Journal of Exposure Analysis and Environmental Epidemiology* 6(1): 79-95.
- Jones-Lee, M.W. 1989. *The Economics of Safety and Physical Risk*. Oxford: Basil Blackwell.
- Jones-Lee, M.W., G. Loomes, D. O'Reilly, and P.R. Phillips. 1993. The Value of Preventing Non-fatal Road Injuries: Findings of a Willingness-to-pay National Sample Survey. TRY Working Paper, WP SRC2.

- Kinney, P.L., K. Ito and G.D. Thurston. 1995. A Sensitivity Analysis of Mortality Pm-10 Associations in Los Angeles. *Inhalation Toxicology* 7(1): 59-69.
- Kleckner, N. and J. Neumann. 1999. Recommended Approach to Adjusting WTP Estimates to Reflect Changes in Real Income. Memorandum to Jim Democker, US EPA/OPAR, June 3.
- Krewski D, Burnett RT, Goldbert MS, Hoover K, Siemiatycki J, Jerrett M, Abrahamowicz M, White WH. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute, Cambridge MA, July 2000.
- Krupnick, A., M. Cropper., A. Alberini, N. Simon, B. O'Brien, R. Goeree, and M. Heintzelman. 2002. Age, Health and the Willingness to Pay for Mortality Risk Reductions: A Contingent Valuation Study of Ontario Residents, *Journal of Risk and Uncertainty*, 24, 161-186.
- Manuel, E.H., R.L. Horst, K.M. Brennan, W.N. Lanen, M.C. Duff and J.K. Tapiero. 1982. Benefits Analysis of Alternative Secondary National Ambient Air Quality Standards for Sulfur Dioxide and Total Suspended Particulates, Volumes I-IV. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Research Triangle Park, NC.
- McClelland, G., W. Schulze, D. Waldman, J. Irwin, D. Schenk, T. Stewart, L. Deck and M. Thayer. 1991. Valuing Eastern Visibility: A Field Test of the Contingent Valuation Method. Prepared for U.S. Environmental Protection Agency, Office of Policy, Planning and Evaluation. June.
- McDonnell, W.F., D.E. Abbey, N. Nishino and M.D. Lebowitz. 1999. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the ahsmog study. *Environmental Research*. 80(2 Pt 1): 110-21.
- Moolgavkar, S.H., E.G. Luebeck, T.A. Hall and E.L. Anderson. 1995. Air Pollution and Daily Mortality in Philadelphia. *Epidemiology* 6(5): 476-484.
- National Research Council (NRC). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. The National Academies Press: Washington, D.C.
- O'Connor, R.M. and G.C. Blomquist. 1997. Measurement of Consumer-Patient Preferences Using a Hybrid Contingent Valuation Method. *Journal of Health Economics*. Vol. 16: 667-683.
- Ostro, B.D., M.J. Lipsett, M.B. Wiener and J.C. Selner. 1991. Asthmatic Responses to Airborne Acid Aerosols. *American Journal of Public Health* 81(6): 694-702.
- Pope, C.A., M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer and C.W. Heath. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *American Journal of Respiratory Critical Care Medicine* 151(3): 669-674.
- Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, G.D. Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. *Journal of the American Medical Association*. 287: 1132-1141.
- Samet, J.M., S.L. Zeger, J.E. Kelsall, J. Xu and L.S. Kalkstein. 1997. Air Pollution, Weather, and Mortality in Philadelphia 1973-1988. Health Effects Institute. Cambridge, MA. March.
- Scultze, W. 2003. Personal Communication. January.
- Smith, V.K., M.F. Evans, H. Kim, and D.H. Taylor, Jr. 2003. Do the "Near" Elderly Value Mortality Risks Differently? Review of Economics and Statistics (forthcoming).
- Thurston, G.D. and K. Ito. 2001. Epidemiological studies of acute ozone exposures and mortality. *J Expo Anal Environ Epidemiol*. Vol. 11(4): 286-94.

Draft Regulatory Impact Analysis

- U.S. EPA. 1997. Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. July.
- US Environmental Protection Agency, 2000. *Valuing Fatal Cancer Risk Reductions*. White Paper for Review by the EPA Science Advisory Board.
- Woodruff, T.J., J. Grillo and K.C. Schoendorf. 1997. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environmental Health Perspectives*. 105(6): 608-612.

