CHAPTER 9: Cost-Benefit Analysis

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 final Nonroad Diesel Engines Tier 4 Standards. EPA is required by Executive Order 12866 to estimate the costs and 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 (NOx), sulfur dioxide (SO_2), non-methane hydrocarbons (NMHC), carbon monoxide (CO) and direct diesel particulate matter ($PM_{2.5}$)[A] emissions?; (2) how much are the changes in these effects attributable to the final rule 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 regulation. In Chapter 12, of the Draft RIA, we provided an analysis of the benefits of several alternatives to the selected standards to examine their relative benefits and costs for public comment.

For the final rulemaking, we rely on the air quality modeling conducted for the proposed rule, documented in the Regulatory Impact Analysis (U.S. EPA, 2003a), available at http://www.epa.gov/nonroad.[B] To estimate the benefits of the final rule, we use a set of scaling factors which separately estimate a set of emission reduction profiles for NOx, SO_2, and directly emitted diesel $PM_{2.5}$. For this analysis of the final rule, we conduct a benefits transfer analysis using those same scaling factors, applied to the updated results of the modeled preliminary control option which accounts for changes in the health benefits methodology adopted during the recent proposed Interstate Air Quality Rule (IAQR) analysis.[C] These methodological changes are reflected both in the detailed estimates for 2020 and 2030 and in the time stream of total monetized benefits. The methodological changes are summarized in this chapter and described in detail in Appendix 9A.

EPA has used the best available information and tools of analysis to quantify the expected changes in public health, environmental and economic benefits for the modeled option. We

[A]Emissions from nonroad diesel engines include directly emitted fine particles (carbon and sulfates) as well as gaseous pollutants that react in the atmosphere to form fine particles. This final rule will results in reductions in ambient PM particle levels due to reductions in both directly emitted particles as well as reductions in PM precursor emissions, including NOx and SO_2.

[B]As discussed in Chapter 2, because of the long lead times to conduct complex photochemical air quality modeling at the national scale, decisions must be made early in the process about the scenarios to be modeled. Based on updated information and public comment, EPA has made changes to the final control program, which results in changes in emissions as detailed in Chapter 3, section 3.6.

[C]Note that the methodology for estimating visibility benefits is unchanged from proposal. The documents related to the IAQR can be found at OAR Docket number 2003-0053.

summarize the results of that analysis in section 9.3, and present details in Appendix 9A, directly following this chapter. The standards we are finalizing in this rulemaking are slightly different in the amount of emission reductions expected to be achieved in 2020 and 2030 relative to both the proposed standards and the preliminary modeled option. As such, we determined that benefits would need to be scaled to reflect the differences in emission reductions between the modeled and final standards. The results of that scaling analysis are the focus of this chapter.

In order to characterize the benefits attributable to the 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 final rule is similar to that used to estimate benefits in the recent analysis of the Large SI/Recreational Vehicles standards (see U.S. EPA 2002, Docket A-2000-01, Document V-B-4). A similar method has also been used in recent benefits analyses for the proposed Clean Air Act Section 112 Utility Mercury Emission Reduction rule, the proposed Industrial Boilers and Process Heaters National Emissions Standards for Hazardous Air Pollutants (NESHAP) standards (Docket numbers OAR-2003-A-96-47) and the Reciprocating Internal Combustion Engines NESHAP standards (Docket numbers OAR-2002-0059 and A-95-35). 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 NOx and VOC. Coupled with the potential for NOx reductions to either increase or decrease ambient ozone levels, this prevents us from scaling the benefits associated with a particular combination of VOC and NOx 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 standards. 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. We quantify benefits for the contiguous 48 states. Note that this table categorizes ozone-related benefits as unquantified effects. Furthermore, we quantify benefits for the contiguous 48 states. We have quantified ozone-related benefits in our modeling of the preliminary control option, summarized in Section 9.3 and detailed in Appendix 9A. However, as noted above, we are unable to quantify ozone-related benefits for the final standards. 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 input into a national benefits analysis. Although these pollutants have been linked to numerous adverse health effects, we are unable to quantify the CO- or air toxics-related health or welfare benefits of the final rule at this time. We also omitted the significant SO2 reductions from lower sulfur in home heating oil in the Northeast.

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

1. Identification of final standards and calculation of the impact that the standards will have on the nationwide inventories for NOx, non-methane hydrocarbons (NMHC), SO_2, and PM emissions throughout the rule implementation period;
2. Calculation of scaling factors relating emissions changes resulting from the final standards to emissions changes from a set of preliminary control options that were used to model air quality and benefits (see Appendix 9A for full details).
3. Apportionment of modeled benefits of preliminary control options to NOx, SO_2, and diesel PM emissions (see Appendix 9A for a complete discussion of the modeling of the benefits for the preliminary set of standards, including updates in the benefits methodology since the time of proposal).
4. Application of scaling factors to apportioned modeled benefits associated with NOx, $_{SO2}$, 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 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 final 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 seven steps laid out above. In Section 1, we identify the potential standard to analyze, establish the timeframe over which benefits are estimated, and summarize emissions impacts. In Section 2, we summarize the changes in emissions that were used in the preliminary modeled benefits analysis and develop the ratios of the emissions reductions under the final standards to preliminary emissions reductions 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 (NOx, SO_2, and $PM_{2.5}$). In Section 4, we estimate the benefits in 2020 and 2030 for the final standards, based on scaling of the modeled benefits of the preliminary control options. In Section 5, we develop intertermporal scaling factors based on the ratios of yearly emission changes to the emission changes in 2020 and 2030 and estimate yearly benefits of the final 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 final standards.

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

Pollutant/Effect	Quantified and Monetized Effects in Primary Analysis	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
PM/Health	Premature mortality in adults Infant mortality Bronchitis - chronic and acute Hospital admissions - respiratory and cardiovascular Emergency room visits for asthma Non-fatal heart attacks (myocardial infarction) Asthma exacerbations (asthmatic population) Lower and upper respiratory illness Respiratory symptoms (asthmatic population) Minor restricted activity days Work loss days		Low birth weight Changes in pulmonary function Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Non-asthma respiratory emergency room visits PM reductions associated with reductions in sulfur in home heating oil
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 Sulfate PM reductions associated with reductions in sulfur in home heating oil

Pollutant/Effect	Quantified and Monetized Effects in Primary Analysis	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
Ozone/Health			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
Ozone/Welfare			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
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

Pollutant/Effect	Quantified and Monetized Effects in Primary Analysis	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
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
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 Bioaccumlation in the food chain Reduced odors

[a] While no causal mechanism has been identified linking new development of chronic asthma to ozone exposure, two epidemiological studies shows a statistical association between long-term exposure to ozone and development 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.

[c] All non-methane hydrocarbons (NMHCs) listed in the table are also hazardous air pollutants listed in Section 112(b) of the Clean Air Act.

9.1 Time Path of Emission Changes for the Final Standards

The final 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 nonroad diesel engine standards, implementation will occur in stages: reductions in sulfur content of nonroad diesel fuel and then adoption of controls on most new nonroad engines. Because full turnover of the fleet of nonroad diesel engines will not occur for many years, the emission reduction benefits of the 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 2007 and extending 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 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 standards 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. NOx and VOC contribute to ambient ozone formation, while NOx, SO_2, NMHC/VOC, and directly emitted $PM_{2.5}$ emissions are precursors to ambient $PM_{2.5}$ and PM_{10} 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 for Air Quality Modeling

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 summarizes the expected changes in emissions of key species. SO_2 emissions are expected to be reduced by over 84 percent within the first two years of implementation. Emissions of $PM_{2.5}$, NOx, and NMHC are expected to be reduced significantly over the period of implementation from 2007 to 2030. Table 9-4 breaks out the expected changes in emissions of key species for the components the fuel portion of the program.

Table 9-3
Summary of Reduction in 48-State Emissions[a]
Attributable to Final Nonroad Diesel Engine Standards and Fuel Programs

	Tons Reduced (Percent of baseline from this category)[a]			
	Direct $PM_{2.5}$	NOx	SO_2	VOC
2010	21,692 13%	149 0%	256,447 91%	525 0%
2015	53,072 33%	193,431 17%	297,513 99%	8,318 8%
2020	85,808 52%	442,061 39%	323,378 99%	18,141 19%
2025	110,043 64%	613,629 54%	349,312 99%	25,002 26%
2030	128,350 72%	734,184 62%	375,354 99%	30,030 31%

[a] NOx, VOC, and CO inventories are for land-based diesel engines only; PM and SO_2 inventories include land-based, recreational marine, commercial marine, and locomotive diesel engines.

Table 9-4
Summary of Reduction in 48-State Emissions
Attributable to Final Fuel Programs of the Nonroad Diesel Standards

	Tons Direct $PM_{2.5}$ and SO_2 Reduced					
	Fuel Only Program		500 ppm NRLM Fuel Program		15 ppm LM Fuel Program (no home heating oil)	
	Direct $PM_{2.5}$	SO_2	Direct $PM_{2.5}$	SO_2	Direct $PM_{2.5}$	SO_2
2010	20,051	256,447	19,156	245,007	0	0
2015	23,241	297,389	20,876	267,118	428	5,318
2020	25,248	323,137	22,674	290,192	433	5,382
2025	27,265	348,994	24,482	313,367	427	5,308
2030	29,293	374,982	26,300	336,665	426	5,294

9.2 Development of Benefits Scaling Factors Based on Differences in Emission Impacts Between the Final Standards 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 engines. The emissions changes modeled for these two years are similar to those in the final standards, differing in the treatment of smaller engines and fuel requirements.[D] Table 9-5 summarizes the reductions in emissions of NOx, SO_2, and $PM_{2.5}$ from baseline for the preliminary and final standards, the difference between the two, and the ratio of emissions reductions from the final 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.

[D]As discussed in Chapter 2, emissions 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 considered public comment. As a result, we have revised the control scenario as described in detail in previous chapters of this document. Section 3.6 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 final baseline and control scenarios.

Table 9-5
Comparison of 48-state Emission Reductions[a, b]
in 2020 and 2030 Between Preliminary and Final Standards

Emissions Species	Reduction from Baseline		Difference in Reductions (Final minus Preliminary)	Ratio of Reductions (Final/ Preliminary)
	Preliminary	Final		
2020				
NOx	663,618	442,061	221,557	0.67
SO_2	414,692	323,378	91,314	0.78
$PM_{2.5}$	98,121	85,808	12,313	0.87
2030				
NOx	1,009,744	734,184	275,560	0.73
SO_2	483,401	375,354	108,047	0.78
$PM_{2.5}$	138,208	128,350	9,858	0.93

[a] Includes all affected nonroad sources: land-based, recreational marine, commercial marine, and locomotives.
[b] We note that the magnitude of NOx reductions determined in the final rule analysis is somewhat less than what was reported in the proposal's draft RIA, especially in the later years when the fleet has mostly turned over to Tier 4 designs. The greater part of this is due to the fact that we have deferred setting a long-term NOx standard for mobile machinery over 750 hp to a later action. When this future action is completed, we would expect roughly equivalent reductions between the proposal and the overall final program, though there are some other effects reflected in the differing NOx reductions as well, due to updated modeling assumptions and the adjusted NOx standards levels for engines over 750 hp. Preamble Section II.A.4 contains a detailed discussion of the NOx standards we are adopting for engines over 750 hp, and the basis for those standards.

9.3 Summary of Modeled Benefits and Apportionment Method

As a second step in the analysis, we calculated scaling factors relating emissions changes resulting from the final standards to emissions changes from a set of preliminary control options that were used to model air quality and benefits (see Appendix 9A for full details). Based on the emissions inventories developed at the time of the proposal 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 NOx, SO_2, 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 PM and ozone concentrations. However, health improvements may also come from reductions in exposure to CO and air toxics. The full analysis is available in Appendix 9A and the benefits Technical Support Document (TSD) (Abt Associates, 2003).

The reductions in emissions of NOx, SO_2, and $PM_{2.5}$ from nonroad engines in the United States are expected to result in wide-spread overall reductions in ambient concentrations of

ozone and $PM_{2.5}$[E]. 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.

Since the publication of the RIA for the proposed rule, EPA has received new technical guidance and input regarding its methodology for conducting PM- and ozone-related benefits analysis from the Health Effects Subgroup (HES) of the Science Advisory Board (SAB) Council reviewing the 812 blueprint (SAB-HES, 2003) and the Office of Management and Budget (OMB) through ongoing discussions regarding methods used in conducting regulatory impact analyses (RIAs) (e.g., see OMB Circular A-4). The SAB HES recommendations include the following (SAB-HES, 2003):

- use of the updated ACS Pope et al. (2002) study rather than the ACS Krewski et al. study to estimate premature mortality for the primary analysis;
- dropping the alternative estimate used in earlier RIAs and instead including a primary estimate that incorporates consideration of uncertainly in key effects categories such as premature mortality directly into the estimates (e.g., use of the standard errors from the Pope et al. (2002) study in deriving confidence bounds for the adult mortality estimates);
- addition of infant mortality (children under the age of one) into the primary estimate, based on supporting evidence from the World Health Organization Global Burden of Disease study (World Health Organization, 2002) and other published studies that strengthen the evidence for a relationship between PM exposure and respiratory inflamation and infection in children leading to death;
- inclusion of asthma exacerbations for children in the primary estimate;
- expansion of the age groups evaluated for a range of morbidity effects beyond the narrow band of the studies to the broader (total) age group (e.g., expanding a study population for 7 to 11 year olds to cover the entire child age range of 6 to 18 years).

[E] Reductions in NOx are expected to result in some localized increases in ozone concentrations, especially in NOx-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 NOx are expected to reduce national levels of health impacts associated with ozone.

- inclusion of new endpoints (school absences [ozone], nonfatal heart attacks in adults [PM], hospital admissions for children under two [ozone]), and suggestion of a new meta-analysis of hospital admissions (PM_{10}) rather than using a few $PM_{2.5}$ studies;[F] and

- updating of populations and baseline incidences.

Recommendations from the Office of Management and Budget (OMB) regarding EPA's methods have focused on the approach used to characterize uncertainty in the benefits estimates generated for RIAs, as well as the approach used to value premature mortality estimates. The EPA is currently in the process of developing a comprehensive, integrated strategy for characterizing the impact of uncertainty in key elements of the benefits modeling process (e.g., emissions modeling, air quality modeling, health effects incidence estimation, valuation) on the results that are generated. A subset of this effort involved an expert elicitation designed to characterize uncertainty in the estimation of PM-related mortality resulting from both short-term and longer-term exposure. In its 2002 report, the NAS provides a number of recommendations on how EPA might improve the characterization of uncertainty in its benefits analyses. One recommendation was that "EPA should begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabalistic, multiple-source uncertainty analyses. This shift will require specification of probability distributions for major sources of uncertainty. These distributions should be based on available data and expert judgement." The NAS elaborated on this recommendation by suggesting a program of methodological development involving review and critique of existing protocols for selection and elicitation of experts by decision analysts, biostatisticians, and psychologists. They recommended the use of formally elicited expert judgements, but noted that a number of issues must be addressed, and that sensitivity analyses would be needed for distributions that are based on expert judgment. They also recommended that EPA clearly distinguish between data-derived components of an uncertainty assessment and those based on expert opinions. As a first step in addressing the NAS recommendations regarding expert elicitation, EPA, in collaboration with OMB, conducted a pilot expert elicitation to characterize uncertainties in the relationship between ambient $PM_{2.5}$ concentrations and premature mortality. While it is premature to include the results of the pilot in the primary analysis for this rulemaking, EPA and OMB believe this pilot moves toward the goal of incorporating additional uncertainty analyses in its future primary benefits analyses. The pilot expert elicitation is described in Appendix 9B and the full report is placed in the public docket.

We have also modified the analysis to reflect new information in the academic literature on the appropriate characterization of the value of reducing the risk of premature mortality (value of

[F]Note that the SAB-HES comments were made in the context of a review of the methods for the Section 812 analysis of the costs and benefits of the Clean Air Act. This context is pertinent to our interpretation of the SAB-HES comments on the selection of effect estimates for hospital admissions associated with PM (SAB-HES, 2003). The Section 812 analysis is focused on a broad set of air quality changes, including both the coarse and fine fractions of PM_{10}. As such, impact functions that focus on the full impact of PM_{10} are appropriate. However, for the Nonroad Diesel Engines rule, which is expected to affect primarily the fine fraction ($PM_{2.5}$) of PM_{10}, impact functions that focus primarily on $PM_{2.5}$ are more appropriate.

statistical life (VSL)). In previous analyses, we used a distribution based on 26 VSL estimates from the economics literature. For this analysis, we are characterizing the VSL distribution in a more general fashion, based on two recent meta-analyses of the wage-risk-based VSL literature (Mrozek and Taylor, 2000 and Viscusi and Aldy, 2003). The new distribution is assumed to be normal, with a mean of $5.5 million and a 95 percent confidence interval between $1 and $10 million. The $1 million lower confidence limit represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis.[G] The $10 million upper confidence limit represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis.

The EPA has addressed many of the comments received from the SAB-HES and OMB in developing the analytical approach for the final rule. We use an approach consistent with the methods used in the benefits analysis of the recently proposed Interstate Air Quality rule (IAQR). We have also reflected advances in data and methods in air quality modeling, epidemiology, and economics in developing this analysis. Updates to the assumptions and methods used in estimating $PM_{2.5}$-related and ozone-related benefits since completion of the Proposed Nonroad Diesel Rule include the following:

Health Endpoints

- We incorporated updated impact functions to reflect updated time-series studies of hospital admissions to correct for errors in application of the generalized additive model (GAM) functions in S-plus. More information on this issue is available at http://www.healtheffects.org.
- The primary analysis used an all-cause mortality effect estimate based on the Pope et al. (2002) reanalysis of the ACS study data.
- Infant mortality was included in the primary analysis.
- Asthma exacerbations were incorporated into the primary analysis. Although the analysis of the proposed rule included asthma exacerbations as a separate endpoint outside of the base case analysis, for the final rule, we will include asthma exacerbations in children 6 to 18 years of age as part of the primary analysis.
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Valuation

- In generating the monetized benefits for premature mortality in the primary analysis, the VSL will be entered as a mean (best estimate) of $5.5 million. Unlike the analysis of the proposed rule, the final rule analysis will not include a value of

[G]An alternative rationale for the low end of the range could be found in some recent stated preference studies suggesting VSL of between $1 and $5 million (Alberini et al., forthcoming).

statistical life year (VSLY) estimate. This reflects the advice of the SAB-Council and concerns raised by commentors on the proposed rule.

The proposed Nonroad Diesel rule included an alternative estimate in addition to the primary estimate that was intended to evaluate the impact of several key assumptions on the estimated reductions in premature mortality and chronic bronchitis. However, reflecting comments from the SAB-HES, rather than including an alternative estimate in the analysis of the final rule, the EPA will investigate the impact of key assumptions on mortality and morbidity estimates through a series of sensitivity analyses. This advice is consistent with the NAS recommendations as well.

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 NOx, NMHC, SO_2, and direct PM emissions in 2020 and 2030; 2) Air quality modeling for 2020 and 2030 to determine changes in ambient concentrations of ozone and PM, 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 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 cannot 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 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 Documents (U.S. EPA

1996a, U.S. EPA, 2004) and the EPA Diesel HAD (U.S. EPA 2002). A discussion of the state of the science as of the last NAAQS review of ozone-related effects can be found in the Ozone Criteria Document (U.S. EPA 1996b). 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 result in an underestimate of the total health benefits of the pollution controls.

9.3.2 Air Quality Modeling

As described in Chapter 2 and the technical support documents (TSDs), 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,[H] 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 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 NO_3 and N_2O_5 species and the addition of several reactions to better account for the wide ranges in temperature, pressure, and concentrations that are encountered for regional and national applications.

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

[H]In 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).

3. Aqueous phase chemistry updates to incorporate the oxidation of $_{SO2}$ by O_3 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.[I]

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 nonroad diesel engine 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., Ozone Transport Assessment Group (OTAG), On-highway Tier-2 Passenger Vehicles, and Heavy Duty Engine/Diesel Fuel 2007 program).

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.

[I] Comments from industry have stated that EPA's methodology form computing benefits over time is based on unsupportable assumptions related to air quality modeling. Specifically, they state that EPA assumes that there will be no interactions between precursors and directly emitted PM in the formation of secondary PM and that EPA excludes consideration of non-linearities in its air quality modeling. The commentor is partially incorrect in the statement that "EPA assumes no interactions between NOx, SO_2, and direct PM in the formation of $PM_{2.5}$." In order to estimate benefits in years other than 2020 and 2030, it was necessary to interpolate values from 2020 and 2030. We used sophisticated air quality modeling (using the REMSAD model) to predict changes in ambient $PM_{2.5}$ in 2020 and 2030. This air quality modeling for 2020 and 2030 does incorporate the nonlinear interactions between NOx, SO_2, and direct PM. However, in order to develop the intertemporal scaling factors, we had to make some simplifying assumptions. We assumed that the interactions between SO_2 and NOx were linear over time, rather than assuming that there was no interaction. In other words, we assumed that the rate of change in the sulfate to SO_2, nitrate to NOx, and primary PM to direct PM ratios was a linear function of time. The rate of change is driven by differences in the baseline emissions between 2020 and 2030 and by differences in the ratio of NOx to SO_2 reductions from the nonroad sector. We verified the interpolation approach by predicting 2020 benefits using scaling factors for sulfate, nitrate, and direct PM based on the modeled 2030 benefits. Scaled benefits were within 4 percent of the actual modeled benefits for 2020.

As discussed in more detail in Chapter 2.3, our ozone air quality modeling showed that the NOx 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, NOx-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 Impact Functions

Health impact functions are derived from the epidemiology literature. A standard health impact function has four components: an effect estimate from a particular epidemiological study, a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics like the Centers for Disease Control), the affected population, and the estimated change in the relevant PM or ozone summary measure.

A typical health impact function might look like:

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

where y_0 is the baseline incidence, equal to the baseline incidence rate times the potentially affected population, β is the effect estimate, and Δx is the estimated change in the summary $PM_{2.5}$ or ozone measure. There are other functional forms, but the basic elements remain the same.

Integral to the estimation of the impact functions are reasonable estimates of future population projections. The underlying data used to create county-level 2010 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 (WP), which account for patterns of economic growth 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.

Specific populations matching the study populations in each epidemiological study are constructed by accessing the appropriate age-specific projections from the overall population database. For some endpoints, such as asthma attacks, we further limit the population by

applying prevalence rates to the overall population. We do not have sufficient information to quantitatively characterize uncertainty in the population estimates.

Fundamental to the estimation of health benefits was our utilization of the PM epidemiology literature. We rely upon effect estimates derived from published, peer reviewed epidemiological studies that relate health effects to ambient concentrations of PM. The specific studies from which effect estimates are drawn are listed in Table 9-5. While a broad range of serious health effects have been associated with exposure to elevated PM levels, we include only a subset of health effects in this benefit analysis due to limitations in available effect estimates and concerns about double-counting of overlapping effects (U.S. EPA, 1996). For the most part, we use the same set of effect estimates as we used in the analysis of the proposed Nonroad Diesel Engines rule. However, based on recent advice from the SAB, we use an updated effect estimate for premature mortality and include two additional health effects, infant mortality and asthma exacerbations. Because of their significance in the analysis, we provide a more detailed discussion of premature mortality and chronic illness endpoints below.

To generate health outcomes, projected changes in ambient PM concentrations were entered into BenMAP, a customized geographic information system based program. BenMAP aggregates populations to air quality model grids and calculates changes in air pollution metrics (e.g., daily averages) for input into health impact functions. BenMAP uses grid cell level population data and changes in pollutant concentrations to estimate changes in health outcomes for each grid cell. Details on the BenMAP program can be found in the BenMAP User's Manual (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 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 effect estimates and the application of those estimates in formulating health impact functions. In general, we selected effect estimates that 1) most closely match the pollutants of interest, i.e. $PM_{2.5}$) cover the broadest potentially exposed population (i.e. all ages functions would be preferred to adults 27 to 35), 3) have appropriate model specification (e.g. control for confounding pollutants), 4) have been peer-reviewed, and 5) are biologically plausible. Other factors may also affect our selection of effect estimates for specific endpoints, such as premature mortality. Some of the more important of these relating to premature mortality and chronic illness are discussed below and are discussed in detail in Appendix 9A. Alternative assumptions about these judgements may lead to substantially different results and they are explored using appropriate sensitivity analyses provided in Appendix 9B.

While there is a consistent body of evidence supporting a relationship between a number of adverse health effects and ambient PM levels, there is often only a single study of a specific endpoint covering a specific age group. There may be multiple estimates examining subgroups (i.e. asthmatic children). However, for the purposes of assessing national population level benefits, we chose the most broadly applicable effect estimate to more completely capture health benefits in the general population. Estimates for subpopulations are provided in Appendix 9A.

There is no consensus on whether or not there is a threshold for the health effects of PM, and if so, what the possible threshold might be. Consistent with recent literature (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 health impact functions are continuous and differentiable down to natural background levels. Our assumptions regarding thresholds are considered reasonable 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 PM_{10} or $PM_{2.5}$, nor any indication of a threshold. (NRC, 2002). 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. We explore this important assumption in a sensitivity analysis described in Appendix 9C.

Premature Mortality

As receommended by the NAS (2002) and the SAB-HES, and demonstrated in the Kunzli et al. (2000) health impact assessment, we focus on the prospective cohort long-term exposure studies in deriving the health impact 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 an effect estimate from the extended analysis of the American Cancer Society (ACS) cohort (Pope et al., 2002) because it represents the most comprehensive cohort analysis with the longest period of followup. In addition, this study has been recommended for impact assessments by the SAB-HES (SAB-HES, 2003). This effect estimate quantifies the relationship between annual mean $PM_{2.5}$ levels and all-cause mortality in adults 30 and older. We selected the effect estimate estimated using the measure of PM representing average exposure over the follow-up period, calculated as the average of 1979-1984 and 1999-2000 $PM_{2.5}$ levels.

In previous analyses, infant mortality has not been evaluated as part of the primary analysis due to uncertainty in the strength of the association between exposure to PM and postneonatal mortality. Instead, benefits estimates related to reduced infant mortality have been included as part of the sensitivity analyses. However recently published studies have strengthened the case for an association between PM exposure and respiratory inflamation and infection leading to premature mortality in infants under five years of age. Specifically, the SAB's HES noted the release of the World Health Organization Global Burden of Disease Study focusing on ambient air which cites several recently-published time-series studies relating daily PM exposure to mortality in children. The HES also cites the study by Belanger et al., (2003) as corroborating

findings linking PM exposure to increased respiratory inflamation and infections in children. With regard to the cohort study conducted by Woodruff et al. (1997), the HES notes several strengths of the study including the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control for a variety of individual risk factors in children (e.g., maternal educational level, maternal ethnicity, parental marital status and maternal smoking status). We follow the HES recommendation to include infant mortality in the primary benefits estimate using the effect estimate from the Woodruff et al. (1997) study.

Chronic Illness

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 in adults aged 25 and older, 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 in adults. 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 are consistent with an increased risk of heart attacks.

9.3.4 Economic Values for Health Outcomes

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a fairly small amount for a large population. The appropriate

economic measure is therefore willingness-to-pay (WTP) for changes in risk prior to the regulation (Freeman, 1993). For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. These costs of illness (COI) estimates generally understate the true value of reductions in risk of a health effect, reflecting the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect (Harrington and Portney, 1987; Berger, 1987). Unit values for health endpoints are provided in Table 9-7. All values are in constant year 2000 dollars.

The length of the delay between reduction in chronic PM exposures and reduction in mortality rates is unknown and yet an important parameter in the benefits analysis. The size 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 the size 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 premature mortality studies suggest that all incidences of premature mortality reduction associated with a given incremental change in PM exposure would not occur in the same year as the exposure reduction. This literature implies that lags of a few years or longer are plausible. For our current analysis, based on previous advice from the SAB (EPA-SAB-COUNCIL-ADV-00-001, 1999), 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.

A more recent SAB-HES report confirmed the NAS (2002) conclusion that there is little justification for the 5-year time course used by EPA in its past assessments, and suggested that future assessments more fully and explicitly account for the uncertainty. The SAB-HES suggests that appropriate lag structures may be developed based on the distribution of cause specific deaths within the overall all-cause estimate. The SAB-HES specifically noted understanding mechanisms of damage and developing models for different cause of death categories may be the key to characterizing more appropriate cessation lag functions. They note that our current understanding of mechanisms suggests there are likely short-term (e.g., less than six months for some cardiovascular effects), medium term (e.g., 2-5 years for COPD), and longer term (e.g., 15 to 25 years for lung cancer). They noted that there is a current lack of direct data to specify a lag function and recommended that information on the lag function be considered in future expert elicitations and/or sensitivity analyses. While we are working to develop the underlying data to support a more appropriate segmented lag structure, for this analysis we maintain the 5-year lag structure used in the benefits analysis for the proposed rule. We have added an additional sensitivity analysis to Appendix 9C examining the impact of assuming a segmented lag of the type suggested by the SAB-HES. The overall impact of moving from the 5-year distributed lag to this version of a segmented lag is relatively modest, reducing benefits by approximately 8 percent when a three percent discount rate is used and 22 percent when a seven percent discount rate is used.

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 1.08 for minor health effects, 1.27 for severe and chronic health effects, 1.23 for premature mortality, and 1.61 for recreational visibility. Adjustment factors for 2020 are 1.07 for minor health effects, 1.23 for severe and chronic health effects, 1.20 for premature mortality, and 1.52 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.

9.3.5 Welfare Effects

Our analysis of the preliminary control option examined 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.

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. as discussed in Chapter 2. Visibility directly affects people's enjoyment of a variety of daily activities. Individuals value visibility both in the places they live, work, and recreate, 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.[J]

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

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 an 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 for the proposed rule (Abt Associates, 2003). Major sources of uncertainty for the visibility benefit estimate include the quality of the underlying study and 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. EPA is considering next steps in improving its visibility benefits estimates.

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.

9.3.5.3 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.

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.

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 9C.

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 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. Likewise, EPA is unable to quantify climate-change related impacts.

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.

We present a primary estimate of the total benefits, based on our interpretation of the best available scientific literature and methods and supported by the SAB-HES and the NAS (NRC, 2002). The benefits estimates generated for the final Nonroad Diesel Engine rule are subject to a number of assumptions and uncertainties, which are discussed throughout the document. For example, key assumptions underlying the primary estimate for the premature mortality which accounts for 90 percent of the total benefits we were able to quantify 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. Although 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 PM produced via transported precursors emitted from EGUs may differ significantly from direct PM released from diesel engines and other industrial sources, but no clear scientific grounds exist for supporting differential effects estimates by particle type.
(3) The impact 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 PM, including both regions that are in attainment with fine particle standard and those that do not meet the standard.
(4) The forecasts for future emissions and associated air quality modeling are valid. Although recognizing the difficulties, assumptions, and inherent uncertainties in the overall enterprise, these analyses are based on peer-reviewed scientific literature and up-to-date assessment tools, and we believe the results are highly useful in assessing this rule.

In addition, we 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 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.

In Appendix 9B, we present two types of probabilistic approaches designed to illustrate how some aspects of the uncertainty in the C-R function could be handled in a PM benefits analysis. The first approach generates a probabilistic estimate of statistical uncertainty based on standard errors reported in the underlying studies used in the benefit modeling framework. In the second illustrative approach, EPA, in collaboration with OMB, conducted a pilot expert elicitation to characterize uncertainties in the relationship between ambient $PM_{2.5}$ and premature mortality (IEc 2004). This pilot was designed to improve our understanding of the design and application of expert elicitation methods to economic benefits analysis. For instance, the pilot was designed to provide feedback on the efficacy of the protocol developed and the analytic challenges, as well as to provide insight regarding potential implications of the results on the degree of uncertainty surrounding the C-R function for $PM_{2.5}$ mortality. The scope of the pilot was limited in that we focused the elicitation on the C-R function of PM mass rather than on individual issues surrounding an estimate of the change in premature mortality due to PM exposure. In Appendix 9B we present sensitivity analyses for illustrative purposes.

9.3.7 Model Results

We summarize our preliminary control option modeling as background for calculating the scaling factors. The scaling factors are then used to estimate the PM-related benefits of the final rule. Insights into ozone impacts can also be discerned. As discussed in Table 9-5 above and Table 9A-4 below, full implementation of the modeled preliminary control options is projected in 2020 to reduce 48-state emissions of land-based nonroad NOx by 663,600 tons (58 percent of base case), SO_2 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 option is expected to reduce 48-state emissions of NOx by 1 million tons (82 percent), SO_2 by 359,800 tons (99.7 percent), VOC by 34,000 tons (35 percent) and direct PM by 138,000 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 $\mu g/m^3$) and 3.4 percent (or 0.28 $\mu g/m^3$) in 2020 and 2030, respectively. The population-weighted average mean concentration declined by 3.3 percent (or 0.42 $\mu g/m^3$) in 2020 and 4.5 percent (or 0.59 $\mu g/m^3$) 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. By 2030, slightly over 50 percent of U.S. populations will live in areas with reductions of greater than 0.5 $\mu g/m^3$. This information indicates how widespread the improvements in PM air quality are expected to be.

Applying the health impact functions described in Table 9-5 to the estimated changes in $PM_{2.5}$ and ozone from the preliminary modeling yields estimates of the number of avoided incidences for each health outcome. These estimates are presented in Appendix A Table 9A-30 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 9A-30, 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 for the preliminary modeled scenario are also presented in Appendix A in Table 9A-31.

The largest monetized health benefit is associated with reductions in the risk of premature mortality, which accounts for 90 percent of total monetized health benefits. The next largest benefit 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 arising from this rule 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 based on the preliminary modeling). However, by 2030, there is a net decrease in ozone-related MRAD consistent with widespread reductions in ozone concentrations from the increased NOx 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.

Monetized and quantified welfare benefits are far outweighed by health benefits. However, we have not been able to quantify some important welfare categories, including the value of changes in ecosystems from reduced deposition of nitrogen and sulfur and climate impacts. 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 $1.6 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.

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				
Adults – Long-term exposure	$PM_{2.5}$	>29 years	Pope, et al. (2002)	CDC Wonder (1996-1998)
Infants	$PM_{2.5}$	<1	Woodruff et al. (1997)	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 (2003) - ICD 490-496 (COPD) Ito (2003) - 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	Ito (2003) - ICD 480-486 (pneumonia)	1999 NHDS public use data files
	$PM_{2.5}$	< 65 years	Sheppard, et al. (2003) - ICD 493 (asthma)	1999 NHDS public use data files
Cardiovascular	$PM_{2.5}$	> 64 years	Pooled estimate:	1999 NHDS public use

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
	$PM_{2.5}$	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)
Asthma Exacerbations	$PM_{2.5}$	6-18 years[A]	Pooled estimate: Ostro et al. (2001) Cough Ostro et al. (2001) Wheeze Ostro et al. (2001) Shortness of breath Vedal et al. (1998) Cough	Ostro et al. (2001) Vedal et al. (1998)
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)

[A] The original study populations were 8-13 for the Ostro et al. (2001) study and 6-13 for the Vedal et al. (1998) study. Based on advice from the SAB-HES and NRC, we have extended the applied population to 6-18, reflecting the common biological basis for the effect in children in the broader age group.

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 (Value of a Statistical Life)	$5,500,000	$6,600,000	$6,800,000	Point estimate is the mean of a normal distribution with a 95 percent confidence interval between $1 and $10 million. Confidence interval is based on two meta-analyses of the wage-risk VSL literature. $1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. $10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population.
Chronic Bronchitis (CB)	$340,000	$420,000	$430,000	Point estimate 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.
Nonfatal Myocardial Infarction (heart attack)				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).
3% discount rate				
Age 0-24	$66,902	$66,902	$66,902	
Age 25-44	$74,676	$74,676	$74,676	
Age 45-54	$78,834	$78,834	$78,834	Lost earnings:
Age 55-65	$140,649	$140,649	$140,649	Cropper and Krupnick (1990). Present discounted value of 5 yrs of lost earnings:
Age 66 and over	$66,902	$66,902	$66,902	
7% discount rate				age of onset: at 3% at 7% 25-44 $8,774 $7,855
Age 0-24	$65,293	$65,293	$65,293	45-54 $12,932 $11,578
Age 25-44	$73,149	$73,149	$73,149	55-65 $74,746 $66,920
Age 45-54	$76,871	$76,871	$76,871	
Age 55-65	$132,214	$132,214	$132,214	Direct medical expenses: An average of:
Age 66 and over	$65,293	$65,293	$65,293	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)

(continued)

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

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.

(continued)

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

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.
Asthma Exacerbations	$42	$45	$45	Asthma exacerbations are valued at $42 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study.
Acute Bronchitis	$360	$380	$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.

(continued)

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

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Restricted Activity and Work/School Loss Days				
Work Loss Days (WLDs)	Variable	Variable	Variable	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.
School Absence Days	$75	$75	$75	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. 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.
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	$54	$55	Median WTP estimate to avoid one MRAD from Tolley, et al. (1986) .

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

1. Uncertainties Associated With Health Impact Functions
– The value of the ozone or PM effect estimate in each health impact function. – Application of a single effect estimate to pollutant changes and populations in all locations. – Similarity of future year effect estimates to current effect estimates. – Correct functional form of each impact function. – Extrapolation of effect estimates beyond the range of ozone or PM concentrations observed in the study. – Application of effect estimates only to those subpopulations matching the original study population.
2. Uncertainties Associated With Ozone and PM Concentrations
– Responsiveness of the models to changes in precursor emissions resulting from the control policy. – Projections of future levels of precursor emissions, especially ammonia and crustal materials. – Model chemistry for the formation of ambient nitrate concentrations. – Lack of ozone monitors in rural areas requires extrapolation of observed ozone data from urban to rural areas. – Use of separate air quality models for ozone and PM does not allow for a fully integrated analysis of pollutants and their interactions. – Full ozone season air quality distributions are extrapolated from a limited number of simulation days. – Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern US and underpredicts nitrate in parts of the Western US.
3. Uncertainties Associated with PM Premature mortality Risk
– No scientific literature supporting a direct biological mechanism for observed epidemiological evidence. – Direct causal agents within the complex mixture of PM have not been identified. – The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures. – The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study. – Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated With Possible Lagged Effects
– The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
5. Uncertainties Associated With Baseline Incidence Rates
– Some baseline incidence rates are not location-specific (e.g., those taken from studies) and may therefore not accurately represent the actual location-specific rates. – Current baseline incidence rates may not approximate well baseline incidence rates in 2030. – Projected population and demographics may not represent well future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
– Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them. – Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors. – Future markets for agricultural products are uncertain.
7. Uncertainties Associated With Aggregation of Monetized Benefits
– Health and welfare benefits estimates are limited to the available effect estimates. 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 [a] in 2020 and 2030

Change in Annual Mean $PM_{2.5}$ Concentrations ($\mu g/m^3$)	*2020 Population*		*2030 Population*	
	Number (millions)	*Percent (%)*	*Number (millions)*	*Percent (%)*
$0 < \Delta PM_{2.5}$ Conc ≤ 0.25	65.11	19.75%	28.60	8.04%
$0.25 < \Delta PM_{2.5}$ Conc ≤ 0.5	184.52	55.97%	147.09	41.33%
$0.5 < \Delta PM_{2.5}$ Conc ≤ 0.75	56.66	17.19%	107.47	30.20%
$0.75 < \Delta PM_{2.5}$ Conc ≤ 1.0	14.60	4.43%	38.50	10.82%
$1.0 < \Delta PM_{2.5}$ Conc ≤ 1.25	5.29	1.60%	8.82	2.48%
$1.25 < \Delta PM_{2.5}$ Conc ≤ 1.5	3.51	1.06%	15.52	4.36%
$1.5 < \Delta PM_{2.5}$ Conc ≤ 1.75	0	0.00%	5.70	1.60%
$\Delta PM_{2.5}$ 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. The results reflect the modeling for the preliminary control option, not the final rule.

9.3.8 Apportionment of Benefits to NOx, SO_2, and Direct PM Emissions Reductions

As noted in the introduction to this chapter, the standards we are finalizing in this rule 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 final and preliminary modeled standards. In order to do so, however, we must first apportion total benefits to the NOx, SO_2, 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 NOx and NMHC/VOC reductions to the change in ozone concentrations.

As discussed in detail in Chapter 2, 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 NOx, $_{SO2}$, 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 NOx contributes to total ambient PM mass differently than a ton of SO_2 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 particulate species. For this exercise, we assume that changes in sulfate particles are attributable to changes in SO_2 emissions, changes in nitrate particles are attributable to changes in NOx emissions, and changes in primary PM are attributable to changes in direct PM emissions. These assumptions essentially assume independence between SO_2, NOx, 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, SO_2 and NOx emissions interact with other compounds in the atmosphere to form $PM_{2.5}$. For example, ammonia reacts with SO_2 first to form ammonium sulfate. If there is remaining ammonia, it reacts with NOx to form ammonium nitrate. When $_{SO2}$ alone is reduced, ammonia is freed to react with any NOx that has not been used in forming ammonium nitrate. If NOx is also reduced, then there will be less available NOx to form ammonium nitrate from the newly available ammonia. Thus, reducing SO_2 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 NOx 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 NOx, then NOx reductions will only have an impact if they reduce emissions to the point where ammonium nitrate formation will be affected. NOx reductions will not result in any offsetting increases in ambient PM under most conditions. The implications of this for apportioning benefits between NOx, SO_2, and direct PM is that some of the sulfate-related benefits will be offset by reductions in nitrate benefits, so benefits from SO_2 reductions will be overstated, while NOx benefits will be understated. It is not immediately apparent the size of this bias.

The measure of change in ambient particulate mass that is most related to health benefits is the population-weighted change in $PM_{2.5}$ $\mu g/m^3$, 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 9-10. The sulfate percentage of total change is used to represent the $_{SO2}$ contribution to health benefits, the nitrate percentage is used to represent the NOx 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 Appendix A in Tables 9A-30 and 9A-31 and combined with the emission scaling factors developed in section 9.2 to estimate benefits for the final set of standards.

Table 9-10. Apportionment of Modeled Preliminary Control Option Population-weighted Change in Ambient $PM_{2.5}$ to Nitrate, Sulfate, and Primary Particles

	2020		2030	
	Population-weighted Change (μg/m3)	Percent of Total Change	Population-weighted Change (μg/m3)	Percent of Total Change
Total $PM_{2.5}$	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 (Sisler, 1996):

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

where rh is relative humidity, ΔTSO4 is the change in particulate sulfate, ΔPNO3 is the change in particulate nitrate, ΔPEC is the change in primary elemental carbon, ΔTOA is the change in total organic aerosols, ΔPMFINE is the change in primary fine particles, and ΔPMCOARSE is the change in primary coarse particles.

The proportion of the total change in light extinction associated with changes in sulfate particles is $[3F(rh) * 1.375 * \Delta TSO4] / \Delta\beta_{EXT}$. The proportion of the total change in light extinction associated with changes in nitrate particles is $[3F(rh) * 1.29 * \Delta PNO3] / \Delta\beta_{EXT}$. Finally, the proportion of the total change in light extinction associated with the change in directly emitted particles is $[10 * \Delta PEC + 4 * \Delta TOA + \Delta PMFINE + 0.6 * \Delta PMCOARSE] / \Delta\beta_{EXT}$.

We calculate these proportions for each park to apportion park specific benefits between $_{SO2}$, NOx, 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 9D.

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

To estimate the benefits of the NOx, SO_2, and direct PM emission reductions from the nonroad diesel engine 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 9A-30 and 9A-31. 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 final 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:

$$Scaled\ Incidence = Modeled\ Incidence * \sum_i R_i A_i ,$$

where Ri 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 9-10. 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:

Scaled Premature Mortality Incidence = 7,821 * (0.759*0.131 + 0.800*0.225 + 0.869*0.644) = 6,562 (rounded to 6,600)

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 final rule based on application of the weighted scaling factors are presented in Table 9-11. The estimated monetized benefits for both PM health and visibility benefits are presented in Table 9-12. 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.

Table 9-11.
Reductions in Incidence of PM-related Adverse Health Effects Associated with the Final Full Program of Nonroad Diesel Engine and Fuel Standards

Endpoint	Avoided Incidence[A] (cases/year)	
	2020	2030
Premature mortality[B]: Long-term exposure (adults, 30 and over)	6,400	12,000
Infant mortality (infants under one year)	15	22
Chronic bronchitis (adults, 26 and over)	3,500	5,600
Non-fatal myocardial infarctions (adults, 18 and older)	8,700	15,000
Hospital admissions – Respiratory (adults, 20 and older)[C]	2,800	5,100
Hospital admissions – Cardiovascular (adults, 20 and older)[D]	2,300	3,800
Emergency Room Visits for Asthma (18 and younger)	3,800	6,000
Acute bronchitis (children, 8-12)	8,400	13,000
Asthma exacerbations (asthmatic children, 6-18)	120,000	200,000
Lower respiratory symptoms (children, 7-14)	99,000	160,000
Upper respiratory symptoms (asthmatic children, 9-11)	76,000	120,000
Work loss days (adults, 18-65)	670,000	1,000,000
Minor restricted activity days (adults, age 18-65)	3,900,000	5,900,000

[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 9-12. Results of PM Human Health and Welfare Benefits Valuation for the Final Full Program of Nonroad Diesel Engine and Fuel Standards

Endpoint	Monetary Benefits[A,B] (millions 2000$, Adjusted for Income Growth)	
	2020	2030
Premature mortality[C]: (adults, 30 and over)		
3% discount rate	$40,000	$77,000
7% discount rate	$38,000	$72,000
Infant mortality (infants under one year)	$960	$150
Chronic bronchitis (adults, 26 and over)	$1,500	$2,400
Non-fatal myocardial infarctions[D]		
3% discount rate	$740	$1,200
7% discount rate	$720	$1,200
Hospital Admissions from Respiratory Causes[E]	$49	$92
Hospital Admissions from Cardiovascular Causes[F]	$50	$83
Emergency Room Visits for Asthma	$1.0	$1.7
Acute bronchitis (children, 8-12)	$3.2	$5.1
Asthma exacerbations (asthmatic children, 6-18)	$5.7	$9.2
Lower respiratory symptoms (children, 7-14)	$1.7	$2.7
Upper respiratory symptoms (asthmatic children, 9-11)	$2.0	$3.2
Work loss days (adults, 18-65)	$91	$130
Minor restricted activity days (adults, age 18-65)	$210	$320
Recreational visibility (86 Class I Areas)	$1,000	$1,700
Monetized Total[G]		
3% discount rate	$44,000+B	$83,000+B
7% discount rate	$42,000+B	$78,000+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 distributed lag structure described earlier. Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4).
[D] Estimates assume costs of illness and lost earnings in later life years are discounted using either 3 or 7 percent
[E] Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.
[F] Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.
[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. These estimates do not include the benefits of reduced sulfur in home heating oil or benefits in Alaska or Hawaii.

We also evaluated the benefits of the NOx, SO_2, and direct PM emission reductions from the nonroad diesel engine standards in 2020 and 2030 of the fuel-only portions of the program. Accordingly, we applied the benefits transfer methods to calculate similar results for the fuel only portion of the program and the 500 ppm NRLM program. Because there would be no NOx or NMHC reductions for the fuel-only components of the rule, the benefits transfer technique may have more uncertainty in this application compared to the full program. As discussed above, we apply scaling and apportionment factors only to PM health and visibility related endpoints. Toxics and ozone-related health and welfare benefits are not estimated for the emissions reductions associated with the final standards for reasons noted in the introduction to this chapter.

The estimated changes in incidence of health effects in 2020 and 2030 for the fuel-only components of the final 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. As described above, 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.

Table 9-13.
Reductions in Incidence of PM-related Adverse Health Effects Associated with the Final Fuel-Related Components of Nonroad Diesel Standards

Endpoint	Avoided Incidence[A] (cases/year)			
	Fuel Only Program		500 ppm NRLM Fuel	
	2020	2030	2020	2030
Premature mortality[B]: Long-term exposure (adults, 30 and over)	2,700	4,000	2,400	3,600
Infant mortality (infants under one year)	<10	<10	<10	<10
Chronic bronchitis (adults, 26 and over)	1,500	1,900	1,300	1,700
Non-fatal myocardial infarctions (adults, 18 and older)	3,600	5,200	3,200	4,700
Hospital admissions – Respiratory (adults, 20 and older)[C]	1,200	1,700	1,000	1,600
Hospital admissions – Cardiovascular (adults, 20 and older)[D]	900	1,300	900	1,100
Emergency Room Visits for Asthma (18 and younger)	1,600	2,000	1,400	1,800
Acute bronchitis (children, 8-12)	3,500	4,600	3,100	4,100
Asthma exacerbations (asthmatic children, 6-18)	51,000	68,000	46,000	61,000
Lower respiratory symptoms (children, 7-14)	41,000	54,000	37,000	49,000
Upper respiratory symptoms (asthmatic children, 9-11)	31,000	41,000	28,000	37,000
Work loss days (adults, 18-65)	280,000	340,000	250,000	300,000
Minor restricted activity days (adults, age 18-65)	1,600,000	2,000,000	1,500,000	1,800,000

[A] Incidences are rounded to two significant digits or nearest ten. The estimates do not include the benefits of reduced sulfur in home heating oil or benefits in Alaska or Hawaii.
[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 PM Human Health and Welfare Benefits Valuation for the Final Fuel-Related Components of the Nonroad Diesel Standards

Endpoint	Monetary Benefits[A,B] (millions 2000$, Adjusted for Income Growth)			
	Fuel Only Program		500 ppm NRLM Fuel	
	2020	2030	2020	2030
Premature mortality[C]: (adults, 30 and over)				
3% discount rate	$17,000	$26,000	$15,000	$23,000
7% discount rate	$16,000	$24,000	$14,000	$22,000
Infant mortality (infants under one year)	$40	$52	$36	$47
Chronic bronchitis (adults, 26 and over)	$610	$820	$550	$740
Non-fatal myocardial infarctions[D]				
3% discount rate	$310	$420	$280	$380
7% discount rate	$300	$410	$270	$370
Hospital Admissions from Respiratory Causes[E]	$20	$31	$18	$28
Hospital Admissions from Cardiovascular Causes[F]	$21	$28	$19	$25
Emergency Room Visits for Asthma	$0.4	$0.6	$0.4	$0.5
Acute bronchitis (children, 8-12)	$1.3	$1.7	$1.2	$1.6
Asthma exacerbations (asthmatic children, 6-18)	$2.3	$3.1	$2.1	$2.8
Lower respiratory symptoms (children, 7-14)	$0.7	$0.9	$0.6	$0.8
Upper respiratory symptoms (asthmatic children, 9-11)	$0.8	$1.1	$0.7	$1.0
Work loss days (adults, 18-65)	$38	$43	$34	$39
Minor restricted activity days (adults, age 18-65)	$90	$110	$80	$100
Recreational visibility (86 Class I Areas)	$400	$550	$360	$500
Monetized Total[G]				
3% discount rate	$18,000+B	$28,000+B	$16,000+B	$25,000+B
7% discount rate	$17,000+B	$26,000+B	$15,000+B	$24,000+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 distributed lag structure described earlier. Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4).
[D] Estimates assume costs of illness and lost earnings in later life years are discounted using either 3 or 7 percent
[E] Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.
[F] Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.
[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. The estimates do not include the benefits of reduced sulfur in home heating oil or benefits in Alaska or Hawaii.

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

To estimate the health and visibility benefits of the NOx, SO_2, and direct PM emission reductions from the final 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 NOx, SO_2, 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 SO_2 emission reductions, nitrate changes are primarily associated with NOx emission reductions, and primary PM changes are associated with direct PM emission reductions.

Using the linear relationship, we estimate the marginal contribution of SO_2 to sulfate, NOx 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 NOx, SO_2, 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 NOx, SO_2, and PM, as well as ammonia and VOCs are changing between years, the relative effectiveness of NOx 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. However, without these assumptions, it is not possible to develop year by year benefits estimates.

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 $PM_{2.5}$

	Change in $PM_{2.5}$ species (population-weighted $\mu g/m^3$ per million tons reduced)		
Year	Sulfate/SO_2	Nitrate/NOx	Primary PM/direct PM
2007	0.153	0.049	2.130
2008	0.154	0.050	2.123
2009	0.156	0.051	2.117
2010	0.157	0.052	2.111
2011	0.159	0.053	2.105
2012	0.160	0.054	2.098
2013	0.161	0.055	2.092
2014	0.163	0.056	2.086
2015	0.164	0.057	2.080
2016	0.166	0.058	2.073
2017	0.167	0.059	2.067
2018	0.169	0.060	2.061
2019	0.170	0.061	2.054
2020	0.171	0.062	2.048
2021	0.173	0.063	2.042
2022	0.174	0.064	2.036
2023	0.176	0.065	2.029
2024	0.177	0.066	2.023
2025	0.179	0.067	2.017
2026	0.180	0.069	2.011
2027	0.181	0.070	2.004
2028	0.183	0.071	1.998
2029	0.184	0.072	1.992
2030	0.186	0.073	1.985

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 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 2015, there are 193,431 tons of NOx reductions, 297,513 tons of SO_2 reductions, and 53,072 tons of direct $PM_{2.5}$ reductions. These are associated with a

populated weighted change in total $PM_{2.5}$ of 0.17, calculated from Table 9-15. The ratio of this change to the change in the 2030 base year is 0.392. The age group apportionment factors (based on using a 3% discount rate for 2030) are 0.2% for 0 to 18, 19.2% for 19 to 64, and 80.6% for 65 and older. The age group population growth ratios for 2015 relative to 2030 are 0.891 for 0 to 18, 0.986 for 19 to 64, and 0.639 for 65 and older. The income growth adjustment ratios for 2015 are 0.936 for premature mortality endpoints and 0.928 for morbidity endpoints. Premature mortality accounts for 93 percent of total health benefits and morbidity accounts for 7 percent of health benefits. Combining these elements with the total estimate of PM health benefits in 2030 of $94.2 billion , total PM health benefits in 2015 for the final standards are calculated as:

Total PM health benefits (2015) =

[$94.2 billion * 0.392*(0.002*0.891+0.192*0.986+0.806*0.639)*(0.93*0.936+0.07*0.928)]

= $24.2 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 NOx 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 SO_2, NOx, 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 final standards using 3 and 7 percent concurrent discount rates.[K] Figure 9-1 shows the undiscounted time stream of benefits 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 NOx emission reduction, so that benefits are dominated by SO_2 and direct $PM_{2.5}$ reductions.

Using a 3 percent intertemporal discount rate, the present value in 2004 of the benefits of the final standards is approximately $805 billion for the time period 2007 to 2036, using a matching 3 percent concurrent discount rate. Using a 7 percent intertemporal discount rate, the present value in 2004 of the benefits of the final standards for the base estimate is approximately $352 billion using a matching 7 percent concurrent discount rate.

Annualized benefits using 3 percent intertemporal and concurrent discount rates are approximately $39 billion. Annualized benefits using 7 percent intertemporal and concurrent discount rates are approximately $28 billion.

[K]We 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.

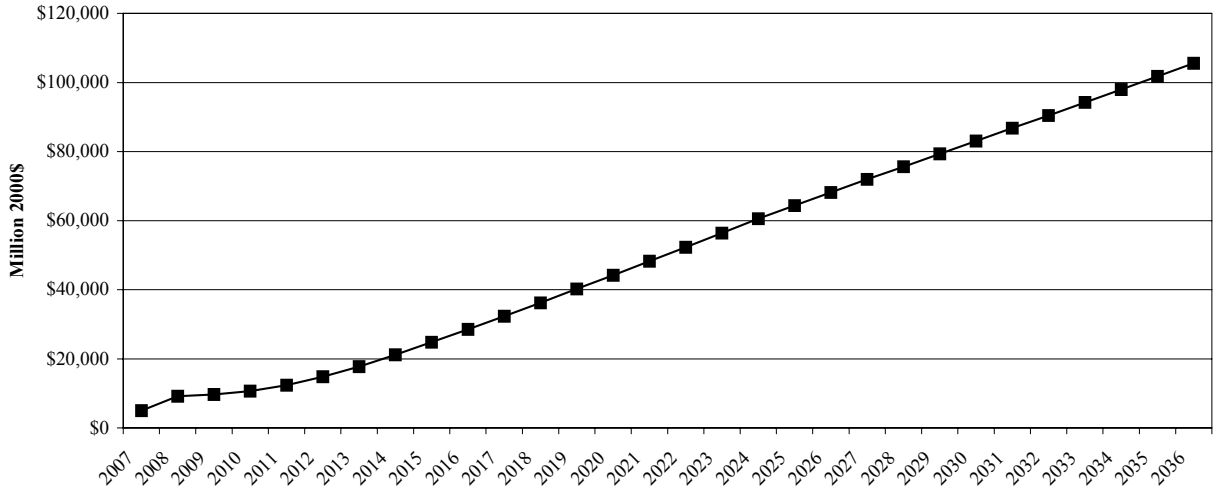
Table 9-16. Time Stream of Benefits for Final Nonroad Diesel Engine Standards[A,B]

Year	Monetized PM-Health and Visbility Benefits (Million 2000$)	
	3% Concurrent Discount Rate	7% Concurrent Discount Rate
2007	$5,000	$4,700
2008	$9,100	$8,600
2009	$9,700	$9,100
2010	$11,000	$10,000
2011	$12,000	$12,000
2012	$15,000	$14,000
2013	$18,000	$17,000
2014	$21,000	$20,000
2015	$25,000	$23,000
2016	$28,000	$27,000
2017	$32,000	$31,000
2018	$36,000	$34,000
2019	$40,000	$38,000
2020	$44,000	$42,000
2021	$48,000	$46,000
2022	$52,000	$49,000
2023	$56,000	$53,000
2024	$61,000	$57,000
2025	$64,000	$61,000
2026	$68,000	$64,000
2027	$72,000	$68,000
2028	$76,000	$71,000
2029	$79,000	$75,000
2030	$83,000	$78,000
2031	$87,000	$82,000
2032	$90,000	$85,000
2033	$94,000	$89,000
2034	$98,000	$92,000
2035	$100,000	$96,000
2036	$110,000	$100,000
Present Value in 2004		
3% Intertemporal Discount Rate	$805,000	--
7% Intertemporal Discount Rate	--	$350,000

[A] All dollar estimates rounded to two significant digits.

[B] Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4).

Figure 9-1.
Base Estimate of the Stream of Annual Benefits for the Final Nonroad Diesel Engine Standards: 2007 to 2036



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 $2.0 billion (here, converted to 2000$). Thus, the net benefit (social benefits minus social costs) of the program at full implementation is approximately $81 + B billion, where B represents the sum of all unquantified benefits and disbenefits. In 2020, partial implementation of the program yields net benefits of $42 + B billion. Therefore, implementation of the final rule is expected, based purely on economic efficiency criteria, to provide society with a significant net gain in social welfare. Table 9-17 presents a summary of the benefits, costs, and net benefits of the final rule. Figure 9-2 displays the stream of benefits, costs, and net benefits of the Nonroad Diesel Engine and Fuel Standards from 2007 to 2036. In addition, Table 9-18 presents the present value of the stream of benefits, costs, and net benefits associated with the rule for this 30 year period. The total present value of the stream of monetized net benefits (benefits minus costs) is $750 billion (using a three percent discount rate).

Table 9-17.
Summary of Monetized Benefits, Costs, and Net Benefits of the Final Full Program Nonroad Diesel Engine and Fuel Standards[A]

	Base Estimate[B]	
	2020 (Billions of 2000 dollars)	**2030** (Billions of 2000 dollars)
Social Costs[C]	$1.8	$2.0
Social Benefits[D, E]:		
CO, VOC, Air Toxic-related benefits	Not monetized	Not monetized
Ozone-related benefits	Not monetized	Not monetized
PM-related Welfare benefits	$1.0	$1.7
PM-related Health benefits (3% discount rate)	$43	$81
PM-related Health benefits (7% discount rate)	$41	$78
Net Benefits (Benefits-Costs)[D,E] (3% discount rate)	$42 +B	$81 +B
Net Benefits (Benefits-Costs)[D,E] (7% discount rate)	$41 +B	$78 +B

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

[B] Base Estimate reflects premature mortality based on application of concentration-response function derived from long-term exposure to $PM_{2.5}$, valuation using the value of statistical lives saved apporach, and a willingness-to-pay approach for valuing chronic bronchitis incidence.

[C] 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. These estimates do not include the benefits of reduced sulfur in home heating oil or benefits in Alaska or Hawaii. Costs are converted from 2002$ to 2000$ in this table using the PPI for Total Manufacturing Industries.

[D] 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. These estimates do not include the benefits of reduced sulfur in home heating oil or benefits in Alaska or Hawaii. B is the sum of all unquantified benefits and disbenefits.

[E] Monetized benefits are presented using two different discount rates. Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4).

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

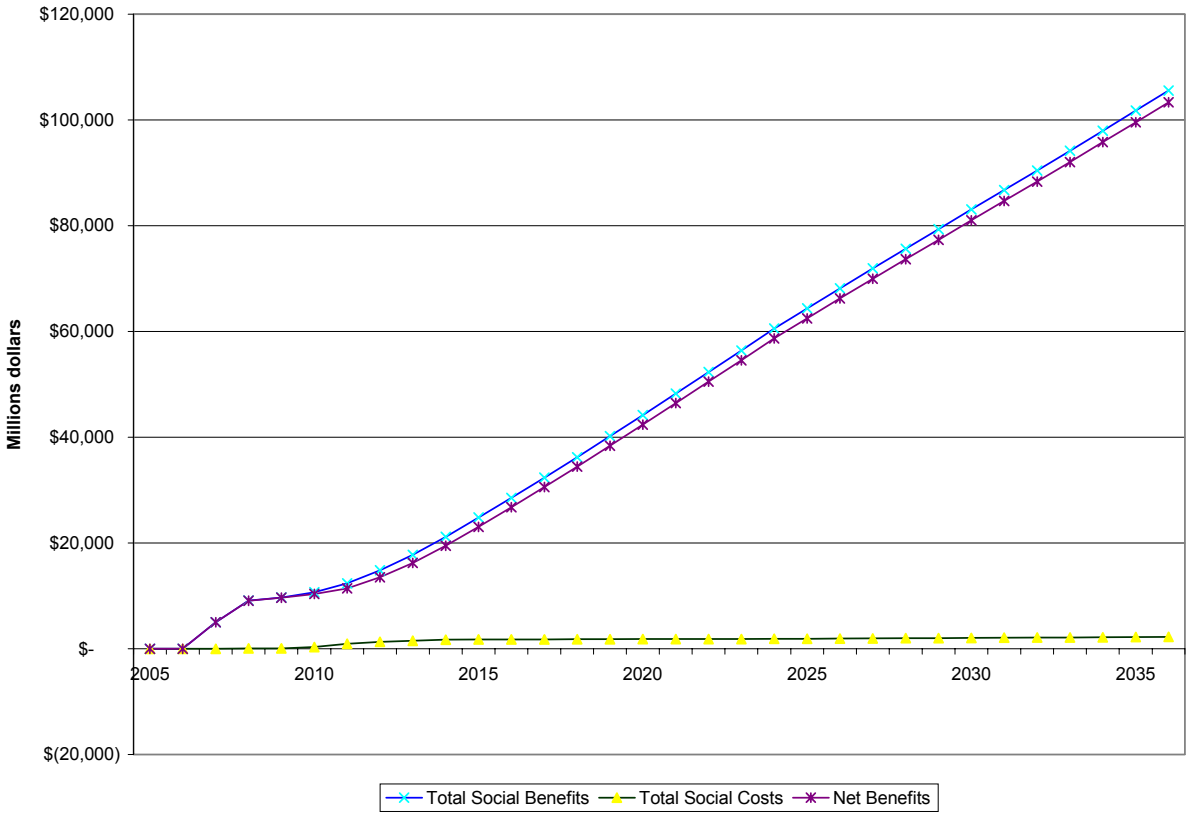


Table 9-18.
Present Value in 2004 of the Stream of 30 Years of Benefits, Costs, and Net Benefits for the Final Full Program Nonroad Diesel Engine and Fuel Standards
(Billions of 2000$)[a, b]

	Billions of 2000$ 3% Discount Rate	**Billions of 2000$** 7% Discount Rate
Social Costs	$ 27	$ 14
Social Benefits	$805	$352
Net Benefits [a]	$780	$340

[a] Rounded to two significant digits

b Benefits represent 48-state benefits and exclude home heating oil sulfur reduction benefits, whereas costs include 50-state estimates. Costs were converted from 2002$ to 2000$ using the PPI for Total Manufacturing Industries.

Table 9-19.
Summary of Monetized Benefits, Costs, and Net Benefits of the Final Fuel Only Components of the Nonroad Diesel Standards (Billions of 2000 dollars) [A]

	Fuel Only Program		500 ppm NRLM Fuel	
	2020	**2030**	**2020**	**2030**
Costs [B, C]	$0.62	$0.72	($0.28)	($0.36)
Social Benefits[C, D, E]:				
CO, VOC, Air Toxic-related benefits	Not monetized	Not monetized	Not monetized	Not monetized
Ozone-related benefits	Not monetized	Not monetized	Not monetized	Not monetized
PM-related Welfare benefits	$0.4	$0.6	$0.4	$0.5
PM-related Health benefits (3 % discount rate)	$18	$28	$16	$25
PM-related Health benefits (7% discount rate)	$17	$26	$15	$23
Net Benefits (3% discount rate) = (Benefits-Costs)[C, D, E]	$ 18 + B	$ 28 + B	$ 16 + B	$ 25 + B
Net Benefits (7% discount rate) = (Benefits-Costs)[C, D, E]	$ 17 + B	$ 26 + B	$ 16 + B	$ 24 + B

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

[B] Engineering costs are presented instead of social costs. As discussed in previous chapters, total engineering costs include fuel costs (refining, distribution, lubricity) and other operating costs (oil change maintenance savings). All engine and equipment fixed cost expenditures are amortized using a seven percent capital cost to reflect the time value of money. The annual costs presented here are the costs in the indicated year and are not the net present values.

[C] 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 SO_2 reductions. The estimates do not include the benefits of reduced sulfur in home heating oil or benefits in Alaska or Hawaii. Costs were converted from 2002$ to 2000$ using the PPI for Total Manufacturing Industries.

[D] 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.

[E] Monetized costs and benefits are presented using two different discount rates. Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4).

Table 9-20.
Present Value in 2004 of the Stream of 30 Years of Benefits, Costs, and Net Benefits for the Final Fuel Only Components of the Nonroad Diesel Standards (Billions of 2000$)[A, B, C, D]

	Fuel Only Program	500 ppm NRLM Fuel
3 % discount rate		
Costs	$9.2	($0.54)
Social Benefits	$340	$310
Net Benefits	**$330**	**$310**
7 % discount rate		
Costs	$4.6	($0.3)
Social Benefits	$160	$140
Net Benefits	**$160**	**$140**

[A] Results are rounded to two significant digits. Sums may differ because of rounding.

[B] Engineering costs are presented instead of social costs. As discussed in previous chapters, total engineering costs include fuel costs (refining, distribution, lubricity) and other operating costs (oil change maintenance savings).

[C] 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 SO_2 reductions. The estimates do not include the benefits of reduced sulfur in home heating oil or benefits in Alaska or Hawaii.

[D] 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.

[E] Monetized costs and benefits are presented using two different discount rates. Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4).

A key input to our benefit-cost analysis is the social costs and emission reductions associated with the final 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 unforseen technical hurdles that may act to increase control system costs.

There is also uncertainty in our 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 estimated to be very small, with a maximum impact of less than one percent. These results are not surprising given the small share of total production costs of diesel engines, equipment, and fuel affected by the rule. See Chapter 10 for a more detailed discussion.

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.[L] 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 9-21 presents the results for some of the fuel and vehicle rules reviewed in the paper.

Table 9-21.
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 9-21 would lead us to believe that cost uncertainty is largely a risk of overestimation by EPA. However, given the uncertainty in estimating costs, we believe it is appropriate to consider the potential for both overestimation and underestimation. 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 -22. This uncertainty has virtually no impact on

[L]For this analysis, 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 (Euromot 2002, Docket A-2001-28 Document number II-B-12). 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 insufficient information that could be used in trying to estimate cost uncertainty for nonroad diesel engine technologies.

our estimates of the net benefits of the final rule, given the large magnitude by which benefits exceed costs.

Table 9-22.
Estimated Uncertainty for Cost of Final Full Program

Year	Engineering Cost Estimate	Uncertainty Range (-20 to +20 percent)
2010	$0.30 billion	$0.24 - $0.36 billion
2020	$1.8 billion	$1.5 - $2.2 billion
2030	$2.1 billion	$1.7 - $2.6 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 final rule, 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 final rule 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.

As discussed in Chapter 3 and our summary and analysis of comments, 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 Chapters 3 and Appendix 8A 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.

Chapter 9 References

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APPENDIX 9A: Benefits Analysis of Modeled Preliminary Control Option

This appendix details the models and methods used to generate the benefits estimates from which the benefits of the final 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 Interstate Air Quality Rule (U.S. EPA, 2004). 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 NOx, non-methane hydrocarbons (NMHC), SO_2, 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.

Potential human health effects linked to $PM_{2.5}$ range from premature mortality linked to long-term exposure to PM, to a range of morbidity effects linked to long-term (chronic) and shorter-term (acute) exposures (e.g., respiratory and cardiovascular symptoms resulting in hospital admissions, asthma exacerbations, and acute and chronic bronchitis). Exposure to ozone has also been linked to a variety of respiratory effects including hospital admissions and illnesses resulting in school absences.[a] Welfare effects potentially linked to PM include materials damage and visibility impacts, while ozone can adversely affect the agricultural and forestry sectors by decreasing yields of crops and forests. Although methods exist for quantifying the benefits associated with many of these human health and welfare categories, not all can be evaluated at this time due to limitations in methods and/or data. Table 4-1 lists the full complement of human health and welfare effects associated with PM and ozone and identifies those effects that are quantified for the primary estimate, are quantified as part of the sensitivity analysis (to be completed for the supplemental analysis), and remain unquantified because of to current limitations in methods or available data.

[A]Short-term exposure to ambient ozone has also been linked to premature death. The EPA is currently evaluating the epidemiological literature examining the relationship between ozone and premature mortality, sponsoring three independent meta-analyses of the literature. Once this evaluation has been completed and peer-reviewed, the EPA will consider including ozone-related premature mortality in the primary benefits analysis for future rules.

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 the EPA's approach for estimating the health benefits of regulations designed to reduce concentrations of airborne PM.

In its report, the NAS said that the 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 5 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 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; and
- 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 the EPA's approach, it found that the studies selected by the Agency for use in its benefits analysis were generally reasonable choices. In particular, the NAS agreed with the 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 the Agency should evaluate. Since the publication of the NAS report, the EPA has reviewed new cohort studies, including reanalyses of the ACS study data and has carefully considered these new study data in developing the analytical approach for the final rule (see below).

In addition to the NAS report, the EPA has also received technical guidance and input regarding its methodology for conducting PM- and ozone-related benefits analysis from two additional sources, including the Health Effects Subgroup (HES) of the SAB Council reviewing the 812 blueprint (SAB-HES, 2003) and the Office of Management and Budget (OMB) through ongoing discussions regarding methods used in conducting regulatory impact analyses (RIAs) (e.g., see OMB Circular A-4). The SAB HES recommendations include the following (SAB-HES, 2003):

- use of the updated ACS Pope et al. (2002) study rather than the ACS Krewski et al. study to estimate premature mortality for the primary analysis;
- dropping the alternative estimate used in the proposal RIA and instead including a primary estimate that incorporates consideration of uncertainly in key effects categories such as premature mortality directly into the estimates (e.g., use of the standard errors from the Pope et al. [2002] study in deriving confidence bounds for the adult mortality estimates);
- addition of infant mortality (children under the age of one) into the primary estimate, based on supporting evidence from the World Health Organization Global Burden of Disease study and other published studies that strengthen the evidence for a relationship between PM exposure and respiratory inflamation and infection in children leading to death;
- inclusion of asthma exacerbations for children in the primary estimate;
- expansion of the age groups evaluated for a range of morbidity effects beyond the narrow band of the studies to the broader (total) age group (e.g., expanding a study population for 7 to 11 year olds to cover the entire child age range of 6 to 18 years).
- inclusion of new endpoints (school absences [ozone], nonfatal heart attacks in adults [PM], hospital admissions for children under two [ozone]), and suggestion of a new

meta-analysis of hospital admissions (PM_{10}) rather than using a few $PM_{2.5}$ studies;[b] and

- updating of populations and baseline incidences.

Recommendations from OMB regarding RIA methods have focused on the approach used to characterize uncertainty in the benefits estimates generated for RIAs, as well as the approach used to value premature mortality estimates. The EPA is currently in the process of developing a comprehensive integrated strategy for characterizing the impact of uncertainty in key elements of the benefits modeling process (e.g., emissions modeling, air quality modeling, health effects incidence estimation, valuation) on the results that are generated.

We are also altering the value of a statistical life (VSL) used in the analysis to reflect new information in the ongoing academic debate over the appropriate characterization of the value of reducing the risk of premature mortality. In previous analyses, we used a distribution of VSL based on 26 VSL estimates from the economics literature. For this analysis, we are characterizing the VSL distribution in a more general fashion, based on two recent meta-analyses of the wage-risk-based VSL literature. The new distribution is assumed to be normal, with a mean of $5.5 million and a 95 percent confidence interval between $1 and $10 million. The EPA welcomes public comment on the appropriate methodology for valuing reductions in the risk of premature death.

The EPA has addressed the comments received from the public, the NAS, the SAB-HES, and OMB in developing the analytical approach for the final rule. We have also reflected advances in data and methods in air quality modeling, epidemiology, and economics that have occurred since the proposal analysis. Updates to the assumptions and methods used in estimating $PM_{2.5}$-related and ozone-related benefits since completion of the Proposed Nonroad Diesel Rule include the following:

Health Endpoints

- The primary analysis incorporates updated impact functions to reflect updated time-series studies of hospital admissions to correct for errors in application of the

[B]Note that the SAB-HES comments were made in the context of a review of the methods for the Section 812 analysis of the costs and benefits of the Clean Air Act. This context is pertinent to our interpretation of the SAB-HES comments on the selection of effect estimates for hospital admissions associated with PM (SAB-HES, 2003). The Section 812 analysis is focused on a broad set of air quality changes, including both the coarse and fine fractions of PM_{10}. As such, impact functions that focus on the full impact of PM_{10} are appropriate. However, for the Nonroad Diesel Engines rule, which is expected to affect primarily the fine fraction ($PM_{2.5}$) of PM_{10}, impact functions that focus primarily on $PM_{2.5}$ are more appropriate.

generalized additive model (GAM) functions in S-plus. More information on this issue is available at http://www.healtheffects.org.

- The primary analysis uses an all cause mortality effect estimate based on the Pope et al. (2002) reanalysis of the ACS study data. In addition, we provide a breakout for two major cause of death categories—cardiopulmonary and lung cancer.
- Infant mortality is included in the primary analysis (infants age 0-1 years).
- Asthma exacerbations are incorporated into the primary analysis. Although the analysis of the proposed rule included asthma exacerbations as a separate endpoint outside of the base case analysis, for the final rule, we will include asthma exacerbations in children 6 to 18 years of age as part of the primary analysis.

Valuation

- In generating the monetized benefits for premature mortality in the primary analysis, the VSL is entered as a mean (best estimate) of 5.5 million. Unlike the analysis of the proposed rule, the analysis of the final rule does not include a value of statistical life year (VSLY) estimate.

The analysis of the proposed rule included an alternative estimate in addition to the primary estimate that was intended to evaluate the impact of several key assumptions on the estimated reductions in premature premature mortality and chronic bronchitis. However, reflecting comments from the public, the SAB-HES as well as the NAS panel, rather than including an alternative estimate in the analysis, the EPA will investigate the impact of key assumptions on mortality and morbidity estimates through a series of sensitivity analyses.

The benefits estimates generated for the final Nonroad Diesel Engine rule are subject to a number of assumptions and uncertainties, which are discussed throughout the document. For example, key assumptions underlying the primary estimate for the premature 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. Although 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 PM produced via transported precursors emitted from EGUs may differ significantly from direct PM released from diesel engines and other industrial sources, but no clear scientific grounds exist for supporting differential effects estimates by particle type.

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

In addition to the quantified and monetized benefits summarized above, 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 odor, 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 health impact functions are not available or which do not provide easily interpretable outcomes (i.e., changes in forced expiratory volume [FEV1]). As a result, monetized benefits generated for the primary estimate may underestimate the total benefits attributable to the final regulatory option.

Benefits estimates for the final Nonroad Diesel Engines rule were generated using BenMAP, which is a computer program developed by the EPA that integrates a number of the modeling elements used in previous RIAs (e.g., interpolation functions, population projections, health impact functions, valuation functions, analysis and pooling methods) to translate modeled air concentration estimates into health effects incidence estimates and monetized benefits estimates. BenMAP provides estimates of both the mean impacts and the distribution of impacts.

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 NOx, VOC, SO_2, 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 and 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 and 9C.

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

Discount Rate	Total Benefits[A, B] (Billions 2000$)	
	2020	2030
3% discount rate	$52+B	$92+B
7% discount rate	$49+B	$87+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, SO_2 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 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4). Results are rounded to two significant digits.

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

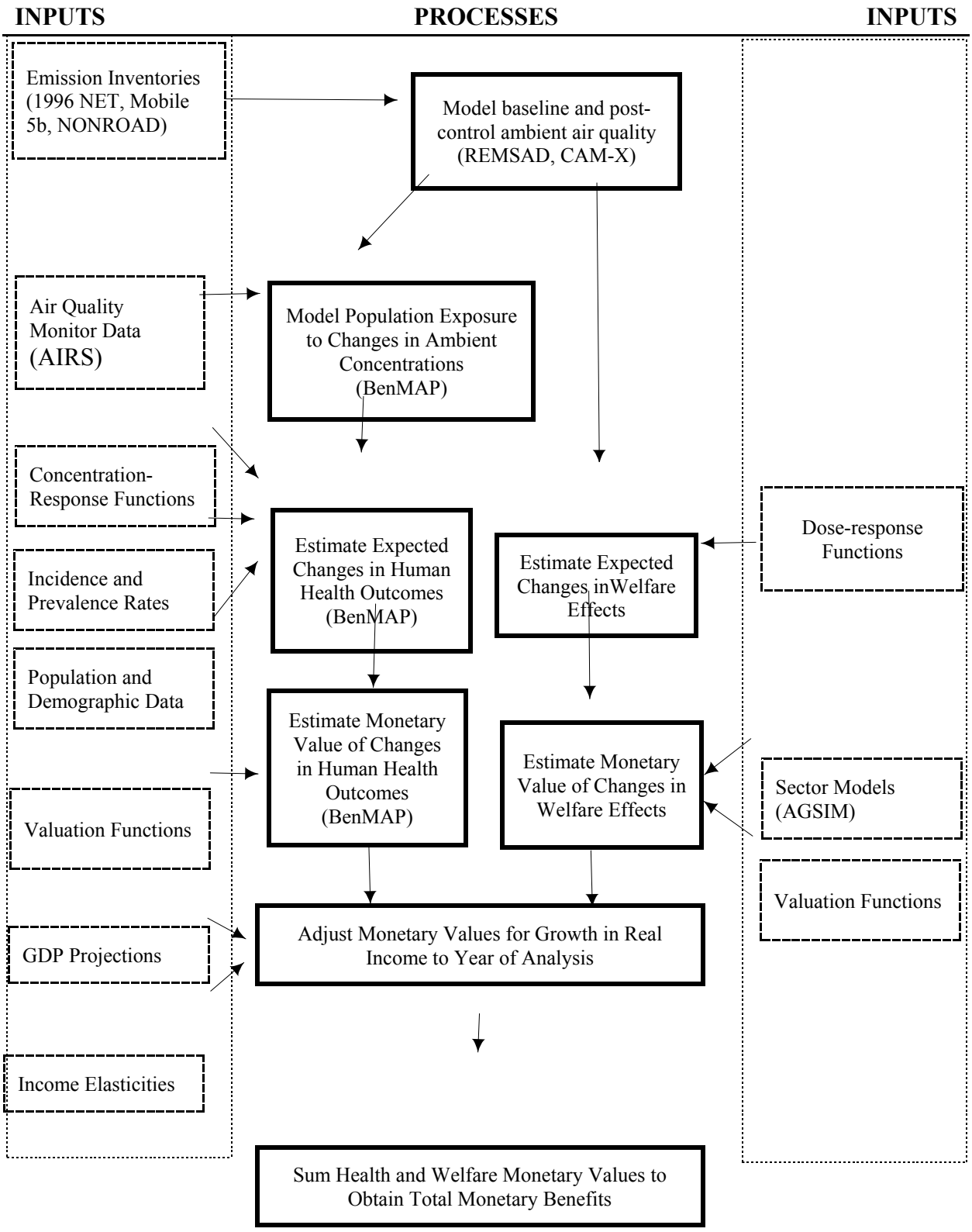


Table 9A.2.
Human Health and Welfare Effects of Pollutants Affected by the Final Nonroad Diesel Engine Rule

Pollutant/Effect	Quantified and Monetized Effects in Primary Analysis	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
PM/Health	Premature mortality in adults – long term exposures Infant mortality Bronchitis - chronic and acute Hospital admissions - respiratory and cardiovascular Emergency room visits for asthma Non-fatal heart attacks (myocardial infarction) Asthma exacerbations Lower and upper respiratory illness Minor restricted activity days Work loss days		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 Effects in Primary Analysis	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
Ozone/Health			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
Ozone/Welfare			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
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

Pollutant/Effect	Quantified and Monetized Effects in Primary Analysis	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
SO_2/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
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 Bioaccumlation 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.

[c] All non-methane hydrocarbons (NMHCs) listed in the table are also hazardous air pollutants listed in Section 112(b) of 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 ways: 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 final 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, SO_2, 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 and received public comment regarding the technical feasibility of the standards, and EPA has revised the control scenario accordingly. 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 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 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

	Pollutant Emissions (tons)			
Source	**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

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

	Pollutant			
Item	**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_2 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 (PM_{10} 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 rule nor have the associated benefits been estimated.

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 pollution control measures that affect ozone, PM and deposition of pollutants to the surface.[c] 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 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

[c] 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.

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.

- Aqueous phase chemistry updates to incorporate the oxidation of SO_2 by O_3 and O_2 and to include the cloud and rain liquid water content from MM5 meteorological data 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.

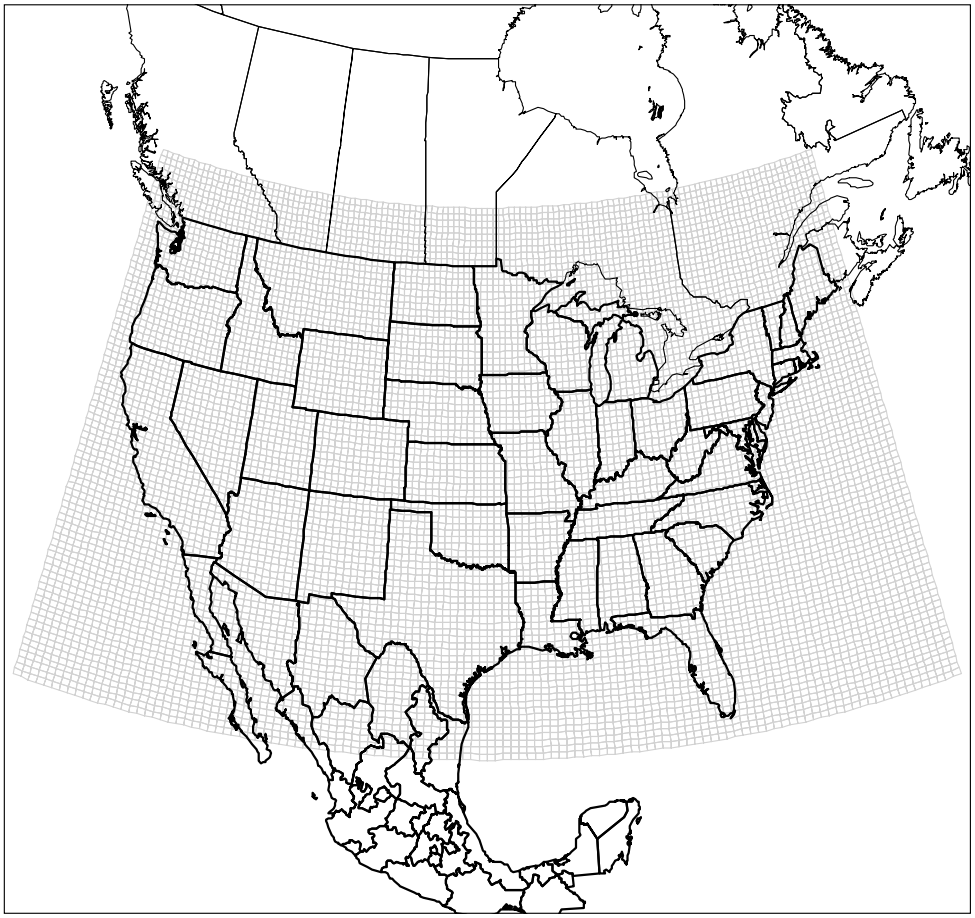
9A.2.1.1 Modeling Domain

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.

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.

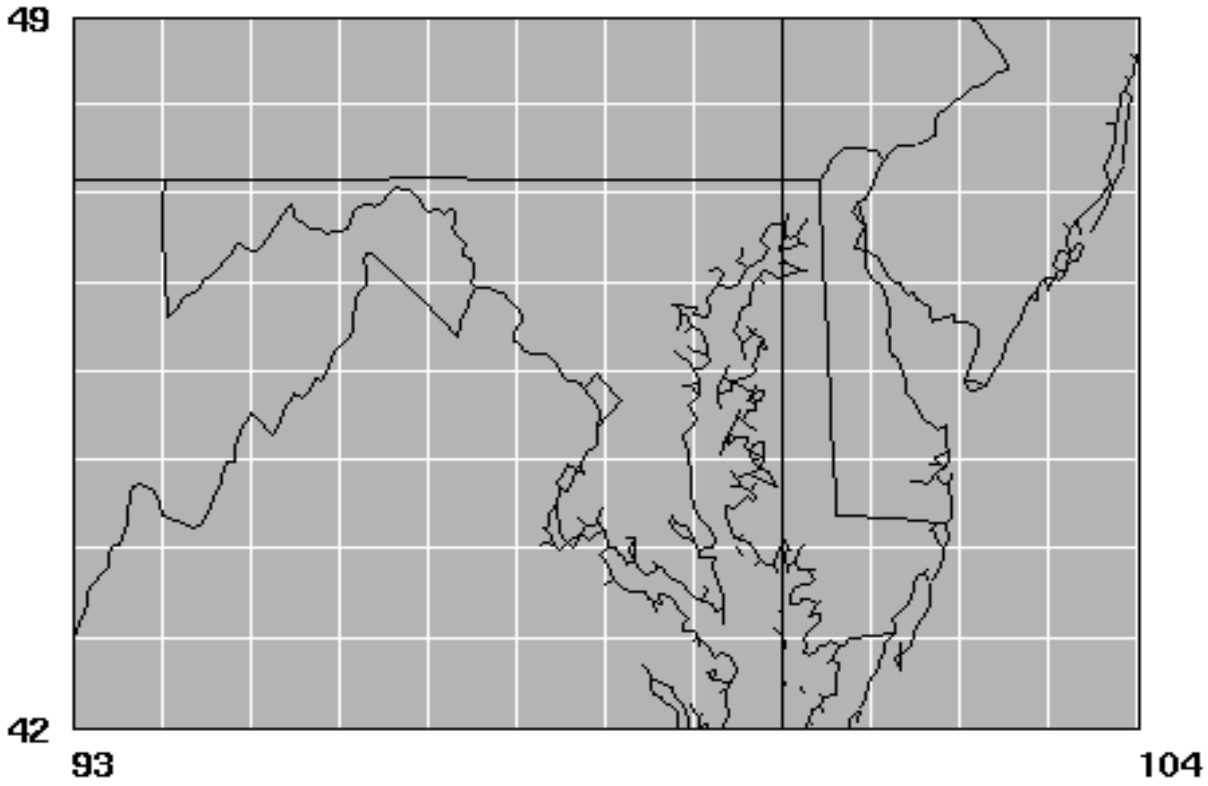


Figure 9A-3. Example of REMSAD 36 x 36km Grid-cells for Maryland Area

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.[d] In addition, 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. [e] 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 (PM_{10} less $PM_{2.5}$). Therefore, the observed changes in PM_{10} 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 $\mu g/m^3$) and 3.4 percent (or 0.28 $\mu g/m^3$) in 2020 and 2030, respectively. The population-weighted average mean concentration declined by 3.3 percent (or 0.42 $\mu g/m^3$) in 2020 and 4.5 percent (or 0.59 $\mu g/m^3$) in 2030, which is much larger in absolute terms than the spatial average for both years. This indicates the rule may generate greater absolute air quality improvements in more populated, urban areas.

[D]Based on AIRS, there were 1,071 FRM PM monitors with valid data as defined as more than 11 observations per season.

[E]This 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 Preliminary Control Option for Nonroad Diesel Standards: 2020 and 2030

Statistic	*2020*			*2030*		
	Base Case	*Change*[a]	*Percent Change*	*Base Case*	*Change*[a]	*Percent Change*
$PM_{2.5}$ ($\mu g/m^3$)						
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 potential reductions in annual mean $PM_{2.5}$ concentrations resulting from the rule. As shown, almost 10 percent of the 2030 U.S. population are predicted to experience reductions of greater than 1 $\mu g/m^3$. 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 $\mu g/m^3$ and slightly over 50 percent will live in areas with reductions of greater than 0.5 $\mu g/m^3$. 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.

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

Change in Annual Mean $PM_{2.5}$ Concentrations ($\mu g/m^3$)	*2020 Population*		*2030 Population*	
	Number (millions)	*Percent (%)*	*Number (millions)*	*Percent (%)*
$0 < \Delta\ PM_{2.5}$ Conc ≤ 0.25	65.11	19.75%	28.60	8.04%
$0.25 < \Delta\ PM_{2.5}$ Conc ≤ 0.5	184.52	55.97%	147.09	41.33%
$0.5 < \Delta\ PM_{2.5}$ Conc ≤ 0.75	56.66	17.19%	107.47	30.20%
$0.75 < \Delta\ PM_{2.5}$ Conc ≤ 1.0	14.60	4.43%	38.50	10.82%
$1.0 < \Delta\ PM_{2.5}$ Conc ≤ 1.25	5.29	1.60%	88.22	2.48%
$1.25 < \Delta\ PM_{2.5}$ Conc ≤ 1.5	3.51	1.06%	15.52	4.36%
$1.5 < \Delta\ PM_{2.5}$ Conc ≤ 1.75	0	0.00%	5.70	1.60%
$\Delta\ PM_{2.5}$ 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 potential changes in PM air quality resulting from the 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., $\mu g/m^3$) and relative terms (i.e., percent) across individual REMSAD grid-cells. Therefore, it provide more information on the range of predicted changes associated with the rule. As shown for 2020, the absolute reduction in annual mean $PM_{2.5}$ concentration ranged from a low of 0.02 $\mu g/m^3$ to a high of 1.36 $\mu g/m^3$, 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 $\mu g/m^3$, 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 Preliminary Control Option for Nonroad Engine/Diesel Fuel Standards: 2020 and 2030

Statistic	*2020* $PM_{2.5}$ *Annual Mean*	*2030* $PM_{2.5}$ *Annual Mean*
Absolute Change from Base Case (μg/m³)[a]		
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
Relative Change from Base Case (%)[b]		
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 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., Ozone Transport Assessment Group (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 preliminary nonroad controls, a 2030 baseline projection and a 2030 projection with preliminary 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.

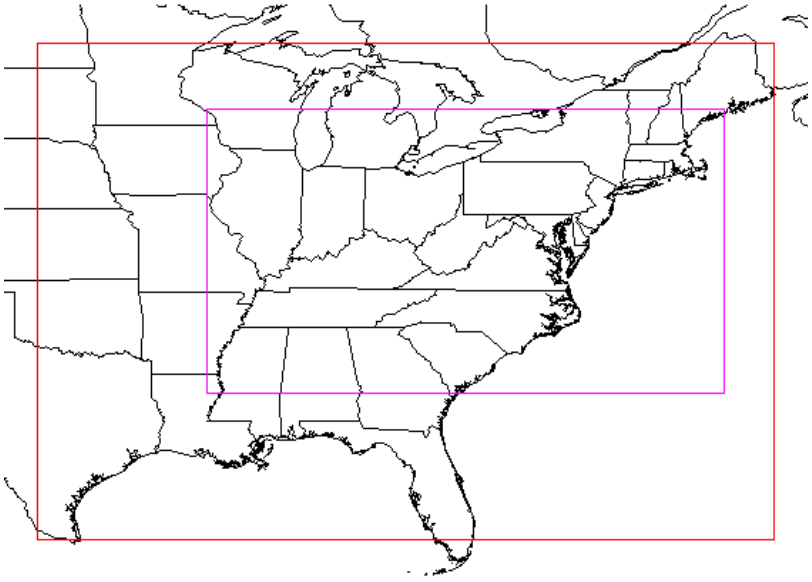
9A.2.2.1 Modeling Domain

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,

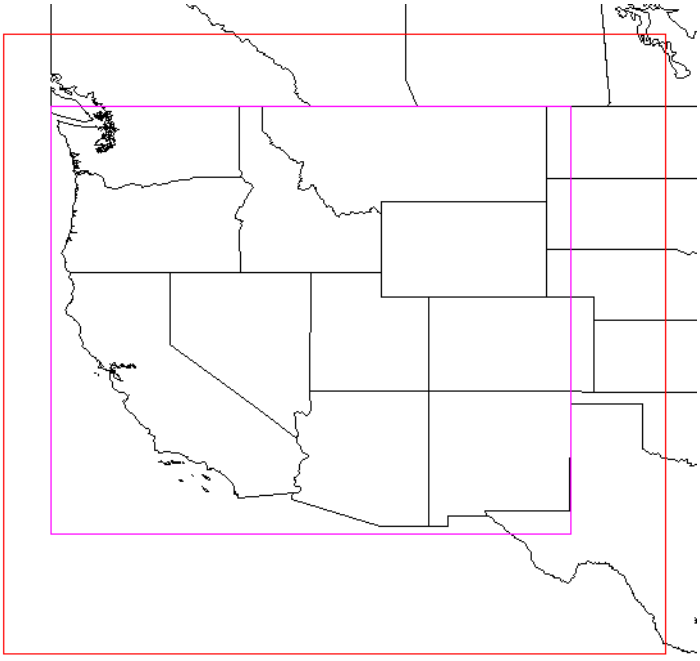
Kansas, Oklahoma, and Texas) and consists of two grids with differing resolutions. The domain extends from 127 degrees to 99 degrees west longitude and from 26 degrees to 52 degrees north latitude. The inner portion of the modeling domain shown in Figure 9A-5 uses a relatively fine grid of 12 km consisting 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.[f,g] 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., BENMAP.

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

[F] 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.

[G] Based 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.

hourly ozone concentrations to a grid of 8 km by 8 km population grid-cells, and (2) we converted these full-season hourly ozone profiles to an ozone measure of interest, such as the daily average. [h,i] 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 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 NOx 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 rule results in average reductions of roughly 1.5 percent, or between 0.57 to 0.52 ppb, in the daily average ozone concentration

[H]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.

[I]This 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).

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.

As discussed in more detail in Chapter 2, our ozone air quality modeling showed that the NOx 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, NOx 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 NOx 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 Preliminary Control Option for 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]
Daily 1-Hour Maximum Concentration (ppb)						
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%
Daily 5-Hour Average Concentration (ppb)						
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%
Daily 8-Hour Average Concentration (ppb)						
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%
Daily 12-Hour Average Concentration (ppb)						
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%
Daily 24-Hour Average Concentration (ppb)						
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 Preliminary Control Option for Nonroad Engine/Diesel Fuel Standards for Health Benefits EndPoints: Western U.S.

Statistic [a]	*2020*			*2030*		
	Base Case	*Change* [b]	*Percent Change* [b]	*Base Case*	*Change* [b]	*Percent Change* [b]
Daily 1-Hour Maximum Concentration (ppb)						
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%
Daily 5-Hour Average Concentration (ppb)						
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%
Daily 8-Hour Average Concentration (ppb)						
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%
Daily 12-Hour Average Concentration (ppb)						
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%
Daily 24-Hour Average Concentration (ppb)						
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, 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 Preliminary Control Option for 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 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 Preliminary Control Option for Nonroad Diesel Engine Standards: 2020 and 2030

Improvements in Visibility [a] *(annual average deciviews)*	*2020 Population*		*2030 Population*	
	Number (millions)	*Percent (%)*	*Number (millions)*	*Percent (%)*
$0 < \Delta$ Deciview ≤ 0.2	52.0	15.8%	11.6	3.3%
$0.2 < \Delta$ Deciview ≤ 0.4	115.5	35.0%	179.7	50.5%
$0.4 < \Delta$ Deciview ≤ 0.6	81.3	24.7%	90.5	25.4%
$0.6 < \Delta$ Deciview ≤ 0.8	62.0	18.8%	49.1	13.8%
$0.8 < \Delta$ Deciview ≤ 1.0	13.2	4.0%	16.4	4.6%
Δ 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 2020 and 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%
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%

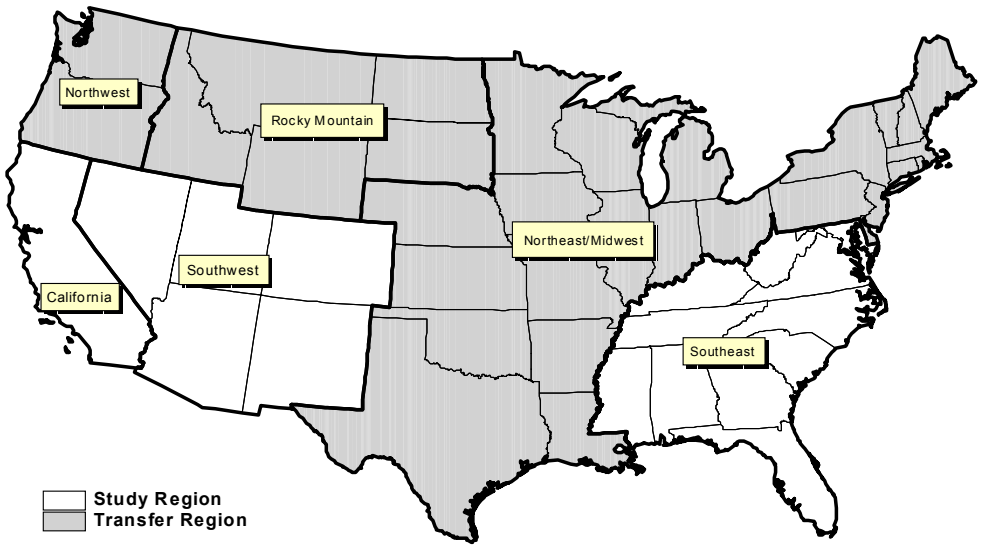
[a] Eastern and Western regions are separated by 100 degrees north longitude. Background visibility conditions differ by region.

[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.

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



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.

Table 9A-14.
Summary of Baseline Recreational Visibility and Changes by Region: 2020 and 2030
(Annual Average Deciviews)

Class I Visibility Regions[a]	*2020*			*2030*		
	Base Case	*Change*[b]	*Percent Change*	*Base Case*	*Change*[b]	*Percent Change*
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.

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, odor), 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-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, SO_2, and air toxics such as benzene.[j] 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 and odor, 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

[J] Several commentators from the public and from public interest groups noted that occupational studies have shown diesel exhaust, as a mixture, to be carcinogenic. In addition, several of these commentors also noted that diesel exhaust contains carcinogenic hazardous air pollutants (HAPs). For these reasons, it was suggested that EPA should include modeling of cancer incidence associated with exposure to the carcinogenic components of diesel exhaust. Diesel particles producing lung cancer mortality may be included in the lung cancer mortality estimates for $PM_{2.5}$. We also acknowledge both that diesel exhaust as a mixture is likely to be carcinogenic and that it contains specific carcinogenic HAPs which represent a cancer risk. However, at this time, as discussed in Chapter 2, we do not believe that the data support the determination of a unit risk for diesel exhaust as a mixture and therefore, lifetime mortality attributable to diesel exhaust exposure cannot be quantified for purposes of benefits analysis.

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 a 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 (Harrrington 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.

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.[k] Stated preference valuation studies form the basis for valuing a number of health and welfare endpoints, including the value of premature 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 premature 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 premature 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

[K]Concerns 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.

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).

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 elasticity[l] 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

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

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.[m]

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.

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[n]. 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

[M] Industry commentors suggest that the income elasticity values used to adjust willingness to pay (WTP) values for avoidance of adverse health effects are based on incorrect methodology. Specifically, they assert that EPA values are based on cross-sectional data when they should be based on time series data. The method we used to derive income adjustment factors, which is detailed here, is consistent with advice from the SAB-EEAC and reflect modest increases in WTP over time. Some recent evidence from published meta-analyses (see Viscusi and Aldy, 2003) suggest that we should be using a larger income adjustment factor for premature mortality.

[N] U.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)

Neumann (1999) for the years 1990 to 2010[o]. We use projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's[p] for the years 2010 to 2024[q]. 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 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.

[O]U.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.

[P]Standard and Poor's. 2000. "The U.S. Economy: The 25 Year Focus." Winter.

[Q]In previous analyses, we used the Standard and Poor's projections of GDP directly. This led to an apparent discontinuity in the adjustment factors between 2010 and 2011. We refined the method by applying the relative growth rates for GDP derived from the Standard and Poor's projections to the 2010 projected GDP based on the Bureau of Economic Analysis projections.

Table 9A-16. Adjustment Factors Used to Account for Projected Real Income Growth[A,B]

Benefit Category	2020	2030[C]
Minor Health Effect	1.066	1.076
Severe and Chronic Health Effects	1.229	1.266
Premature Mortality	1.201	1.233
Visibility	1.516	1.613

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

[B] Note that these factors have been modified from the proposal analysis to refelect relative growth rates for GDP derived from the Standard and Poor's projections rather than absolute growth rates.

[C] 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.[r] This analysis is no exception. As outlined both in this and preceding chapters, many inputs are used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, 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).

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

- Gaps in scientific data and inquiry;

[R] 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.

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

Some of the key uncertainties in the benefits analysis are presented in Table 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 available meta-analyses provides a quantifiable basis for representing some uncertainty that can be calculated for monetized benefits. Methods typically used to evaluate the impact of these quantifiable sources of uncertainty on benefits and incidence estimates center on Monte Carlo-based probabilistic simulation. This technique allows uncertainty in key inputs to be propagated through the model to generate a single distribution of results reflecting the combined impact of multiple sources of uncertainty. Variability can also be considered along with uncertainty using nested two-stage Monte Carlo simulation.

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, diesel odor, 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, without conducting a comprehensive uncertainty

analysis that considers the aggregate impact of multiple sources of uncertainty on benefits estimates.

The NAS report on the EPA's benefits analysis methodology highlighted the need for the EPA to conduct rigorous quantitative analysis of uncertainty in its benefits estimates. In response to these comments, the EPA has initiated the development of a comprehensive methodology for characterizing the aggregate impact of uncertainty in key modeling elements on both health incidence and benefits estimates. This methodology will begin by identifying those modeling elements that have a significant impact on benefits due to either the magnitude of their uncertainty or other factors such as nonlinearity within the modeling framework. A combination of influence analysis and sensitivity analysis methods may be used to focus the analysis of uncertainty on these key sources of uncertainty. A probabilistic simulation approach based on Monte Carlo methods will be developed for propagating the impact of these sources of uncertainty through the modeling framework. Issues such as correlation between input parameters and the identification of reasonable upper and lower bounds for input distributions characterizing uncertainty will be addressed in developing the approach.

For this analysis of the final rule, EPA has addressed key sources of uncertainty through a series of sensitivity analyses examining the impact of alternate assumptions on the benefits estimates that are generated. Sensitivity estimates are presented in Appendix 9C. We also present information related to an expert elicitation pilot 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.

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

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

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.

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.[s] We use projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's for the years 2010 to 2024.[t] 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.

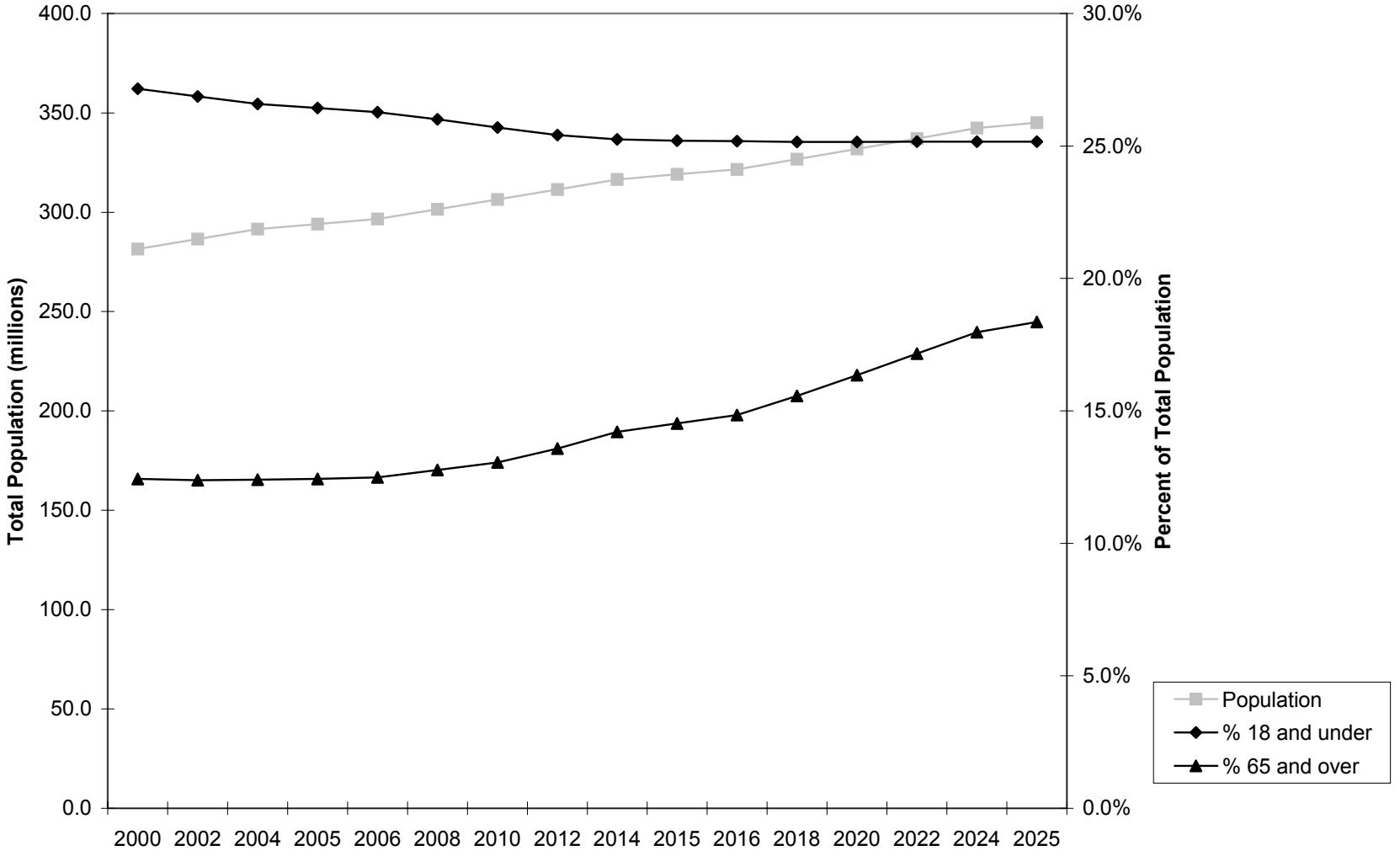
9A.3.5 Health Benefits Assessment Methods

The most significant monetized benefits of reducing ambient concentrations of PM and ozone are attributable to reductions in health risks associated with air pollution. The EPA's Criteria Documents for ozone and PM list numerous health effects known to be linked to ambient concentrations of these pollutants (EPA, 1996a and 1996b). As illustrated in Figure 9A-1, quantification of health impacts requires several inputs, including epidemiological effect estimates, 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 effect estimates 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.

[S] 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.

[T] Standard and Poor's. 2000. "The U.S. Economy: The 25 Year Focus." Winter 2000.

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



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.

9A.3.5.1 Selecting Health Endpoints and Epidemiological Effect Estimates

Quantifiable health benefits of the rule 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, nonfatal heart attacks, chronic bronchitis, acute bronchitis, upper and lower respiratory symptoms, asthma exacerbations, and work loss days.[u] Health effects related to both PM and ozone include hospital admissions, emergency room visits for asthma, and minor restricted activity days.

We relied on the available published scientific literature to ascertain the relationship between PM 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.

The Health Effects Institute (HEI) reported findings by health researchers 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, 2002). The estimates derived from the long-term exposure studies, which account for a major share of the economic benefits described in this chapter, are not affected. Similarly, the time-series studies employing generalized linear models (GLMs) or other parametric methods, as well as case-crossover studies, are not affected. As discussed in HEI materials provided to the EPA and to CASAC (Greenbaum, 2002), researchers working on the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) found problems in the default

[U]Evidence has been found linking ozone exposures with premature mortality independent of PM exposures. 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 exposure mortality benefits of the ozone NAAQS (EPA, 1997) may have been underestimated by up to a factor of two, even when PM is controlled for in the models. 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). In addition, a recent World Health Organization (WHO) report found that "recent epidemiological studies have strengthened the evidence that there are short-term O3 effects on premature mortality and respiratory morbidity and provided further information on exposure-response relationships and effect modification." (WHO, 2003). Based on these new analyses and recommendations, the EPA is currently reevaluating ozone-related mortality for inclusion in the primary benefits analysis. The EPA is sponsoring three independent meta-analyses of the ozone-mortality epidemiology literature to inform a determination on inclusion of this important health endpoint. Upon completion and peer review of the meta-analyses, the EPA will make its determination on whether benefits of reductions in ozone-related mortality will be included in the future benefits analyses.

"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. Following identification of the GAM issue, a number of time-series studies were reanalyzed using alternative methods, typically GAM with more stringent convergence criteria and an alternative model such as generalized linear models (GLM) with natural smoothing splines, and the results of the reanalyses have been compiled and reviewed in a recent HEI publication (HEI, 2003a). In most, but not all, of the reanalyzed studies, it was found that risk estimates were reduced and confidence intervals increased with the use of GAM with more stringent convergence criteria or GLM analyses; however, the reanalyses generally did not substantially change the findings of the original studies, and the changes in risk estimates with alternative analysis methods were much smaller than the variation in effects across studies. The HEI review committee concluded the following:

- Although the number of studies showing an association of PM with premature mortality was slightly smaller, the PM association persisted in the majority of studies.
- In some of the large number of studies in which the PM association persisted, the estimates of PM effect were substantially smaller.
- In the few studies in which investigators performed further sensitivity analyses, some showed marked sensitivity of the PM effect estimate to the degree of smoothing and/or the specification of weather (HEI, 2003b, p. 269)

Examination of the original studies used in our benefits analysis found that the health endpoints that are potentially affected by the GAM issues include reduced hospital admissions and reduced lower respiratory symptoms. For the analysis of the final rule, we have incorporated a number of studies that have been updated to correct for the GAM issue, including Ito et al. (2003) for respiratory-related hospital admissions (COPD and pneumonia), Shepard et al. (2003) for respiratory-related hospital admissions (asthma), Moolgavkar (2003) for cardiovascular-related hospital admissions (ICD codes 390-429), and Ito et al. (2003) for cardiovascular-related hospital admissions (ischemic heart disease, dysrhythmia, and heart failure). Several additional hospital admissions-related studies have not yet been formally updated to correct for the GAM issue. These include the lower respiratory symptoms study and hospital admissions for respiratory and cardiovascular causes in populations aged 20 to 64. However, as discussed above, available evidence suggests that the errors introduced into effect estimates due to the GAM issue should not significantly affect incidence results.

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 because they control for important individual-level confounding variables that cannot be controlled for in cross-sectional studies.
Study period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and life style over time. However, when there are only a few studies available, studies from all years will be included.
Population attributes	The most technically appropriate measures of benefits would be based on impact functions that cover the entire sensitive population, but allow for heterogeneity across age or other relevant demographic factors. In the absence of effect estimates specific to age, sex, preexisting condition status, or other relevant factors, it may be appropriate to select effect estimates that cover the broadest population, to match with the desired outcome of the analysis, which is total national-level health impacts.
Study size	Studies examining a relatively large sample are preferred because they generally have more power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population, or through repeated observations on a smaller population, i.e. through a symptom diary recorded for a panel of asthmatic children.
Study location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior and life style.
Pollutants included in model	When modeling the effects of ozone and PM (or other pollutant combinations) jointly, it is important to use properly specified impact functions that include both pollutants. Use of single pollutant models in cases where both pollutants are expected to affect a health outcome can lead to double-counting when pollutants are correlated.
Measure of pollutant	For this analysis for PM-related effects, impact functions based on $PM_{2.5}$ are preferred to PM_{10} because the Nonroad Diesel Engine rule will regulate emissions of $PM_{2.5}$ precursors and air quality modeling was conducted for this size fraction of PM. 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. Adequacy of ozone exposure metrics in studies was also considered.
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.

It is important to reiterate that the estimates derived from the long-term exposure studies, which account for a major share of the economic benefits described in this chapter, are not affected by the GAM issue. Similarly, the time-series studies employing GLMs or other parametric methods, as well as case-crossover studies, are not affected.

Although a broad range of serious health effects has 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 (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: the possibility of double counting (such as hospital admissions for specific respiratory diseases); uncertainties in applying effect relationships based on clinical studies to the affected population; or a lack of an established relationship between the health effect and pollutant in the published epidemiological literature.

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, making it difficult to pool the results in a consistent manner. For example, studies may examine different pollutants or different age groups. For this reason, we consider very carefully the set of studies available examining each endpoint and select a consistent subset that provides a good balance of population coverage and match with the pollutant of interest. In many cases, either because of 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 effect estimate.

When several effect estimates for 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 Technical Support Document (TSD) completed for the nonroad diesel rulemaking provides details of the procedures used to combine multiple impact 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 study's 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.[v] Pooled impact functions are

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

used to estimate hospital admissions (PM), school absence days (ozone), lower respiratory symptoms (PM), asthma exacerbations (PM), and asthma-related emergency room visits (ozone). For more details on methods used to pool incidence estimates, see the benefits TSD for the nonroad diesel rulemaking (Abt Associates, 2003).

Effect estimates for a pollutant and a given health endpoint are applied consistently across all locations nationwide. This applies to both impact functions defined by a single effect estimate and those defined by a pooling of multiple effect estimates. Although the effect estimate may, in fact, vary from one location to another (e.g., due to differences in population susceptibilities or differences in the composition of PM), location-specific effect estimates are generally not available.

The specific studies from which effect estimates for the primary analysis are drawn are included in Table 9A-19.

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 premature mortality risk estimates from these epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make premature mortality risk reduction the most important health endpoint quantified in this analysis.

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

Researchers have found statistically significant associations between PM and premature mortality using both types of studies. In general, the risk estimates based on the long-term exposure studies are larger than those derived from short-term studies. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli, 2001; NRC, 2002). This section discusses some of the issues surrounding the estimation of premature mortality.

Over a dozen studies have found significant associations between various measures of long-term exposure to PM and elevated rates of annual mortality, beginning with Lave and Seskin (1977). Most of the published studies found positive (but not always statistically significant) associations with available PM indices such as total suspended particles (TSP). Particles of different fine particles components (i.e., sulfates), and fine particles, as well as exploration of alternative model specifications sometimes found inconsistencies (e.g., Lipfert, [1989]). These early "cross-sectional" studies (e.g., Lave and Seskin [1977]; Ozkaynak and Thurston [1987]) 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 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 individual-level information with respect 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 and Pope et al., 2002); these studies have found consistent relationships between fine particle indicators and premature mortality across multiple locations in the United States. 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 premature mortality in men. Results from this cohort, however, have been inconsistent and the air quality results are not geographically representative of most of the United States. The Veterans Study was originally designed as a means of assessing the efficacy of anti-hypertensive drugs in reducing morbidity and mortality in a population with pre-existing high blood pressure (in this case, male veterans) (Lipfert et al., 2000). Unlike previous long-term analyses, this study found some associations between premature mortality and ozone but found inconsistent results for PM indicators. A variety of issues associated with the study design, including sample representativeness and loss to follow up, make this cohort a poor choice for extrapolating to the general public. Furthermore, because of the selective nature of the population in the veteran's cohort and methodological weaknesses, which may have resulted in estimates of relative risk that are biased relative to a relative risk for the general population, we have chosen not to include any effect estimates from the Lipfert et al. (2000) study in our benefits assessment. We note that, while the PM analyses considering segmented (shorter) time periods such as Lipfert et al. gave differing results (including significantly negative mortality coefficients for some PM metrics), when methods consistent with the past studies were used (i.e., many- year average PM concentrations), similar results were reported: the authors found that "(t)he single-mortality-period responses without ecological variables are qualitatively similar to what has been reported before (SO_4 > $PM_{2.5}$ > PM_{15})."

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

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature Mortality— Long-term exposure, all-cause	$PM_{2.5}$	Pope et al. (2002)	>29 years
Premature Mortality— Long-term exposure, all-cause	$PM_{2.5}$	Woodruff et al., 1997	Infant (<1 yr)
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 (2003) - ICD 490-496 (COPD) Ito (2003) - ICD 490-496 (COPD)	> 64 years
	$PM_{2.5}$	Moolgavkar (2000) - ICD 490-496 (COPD)	20-64 years
	$PM_{2.5}$	Ito (2003) - ICD 480-486 (pneumonia)	> 64 years
	$PM_{2.5}$	Sheppard, et al. (2003) - ICD 493 (asthma)	< 65 years
Cardiovascular	$PM_{2.5}$	Pooled estimate: Moolgavkar (2003) - ICD 390-429 (all cardiovascular) Ito (2003) - 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

(continued)

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

Endpoint	Pollutant	Study	Study Population
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	$PM_{2.5}$	Schwartz and Neas (2000)	7-14 years
Asthma Exacerbations	$PM_{2.5}$	Pooled estimate: Ostro et al. (2001) (cough, wheeze and shortness of breath) Vedal et al. (1998) Cough	6-18 years[a]
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)	Outdoor workers, 18-65
Minor Restricted Activity Days	$PM_{2.5}$, Ozone	Ostro and Rothschild (1989)	18-65 years

[a] The original study populations were 8 to 13 for the Ostro et al. (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the SAB-HES, we have extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group.

Given their consistent results and broad geographic coverage, the Six-City and ACS data have been particularly important 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 team of scientific experts commissioned by HEI (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 and highlights sensitivities concerning the relative impact of various pollutants, the potential role of education in mediating the association between pollution and premature mortality, and the influence of spatial

correlation modeling.[w] Further confirmation and extension of the overall findings using more recent air quality and a longer follow-up period for the ACS cohort was recently published in the *Journal of the American Medical Association* (Pope et al., 2002).

In developing and improving the methods for estimating and valuing the potential reductions in premature mortality risk over the years, the EPA has consulted with the SAB-HES. That panel recommended use of long-term prospective cohort studies in estimating premature 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" (NAS, 2002, p. 108). In the NRC's view, compared with the time-series studies, cohort studies give a more complete assessment of the long-term, cumulative effects of air pollution. The overall effect estimates may be a combination of effects from long-term exposure plus some fraction from short-term exposure, but the amount of overlap is unknown. Additionally, the SAB recommended emphasis on the ACS study because it includes a much larger sample size and longer exposure interval and covers more locations (e.g., 50 cities compared to the Six Cities Study) than other studies of its kind. As explained in the regulatory impact analysis for the Heavy-Duty Engine/Diesel Fuel rule (EPA, 2000a), more recent EPA benefits analyses have relied on an improved specification of the ACS cohort data that was developed in the HEI reanalysis (Krewski et al., 2000). The latest reanalysis of the ACS cohort data (Pope et al., 2002), provides additional refinements to the analysis of PM-related mortality by (a) extending the follow-up period for the ACS study subjects to 16 years, which triples the size of the mortality data set; (b) substantially increasing exposure data, including consideration

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[w] Regarding potential confounding by copollutants, commentors noted that the HEI reanalysis of the ACS study data for long-term exposure mortality found an association between SO_2 and premature mortality and did not find a strong association between $PM_{2.5}$ and premature mortality. These commentors suggest that these findings regarding potential confounding compromise the accuracy of the ACS study. While recognizing the need for research into the issue of copollutants, including SO_2, we disagree with the commentor's interpretation of the HEI reanalysis. While this study did find an association between premature mortality and SO_2, such an association was also reported for fine particles and sulfate. In addition, the HEI reanalysis, as well as other studies examining the copollutant issue (Samet et al., 2000, 2001) have suggested that SO_2 might represent a surrogate for ambient $PM_{2.5}$ concentrations and is likely associated with sulfate concentrations since it is a precursor. This could partially explain the association between SO_2 and premature mortality found in the HEI reanalysis. Finally, we have updated our methods for characterizing premature mortality and are now using the Pope et al. 2002 reanalysis of the ACS study data. While this study continues to find and association between SO_2 and cardiovascular mortality, it also finds the strongest association yet between long term $PM_{2.5}$ exposure and premature mortality.

Commentors have also suggested that both the ACS and Six Cities studies provide evidence for confounding by socio-economic factors in the chronic exposure mortality endpoint. Following recommendations by the SAB-HES, we have updated our analytical framework to use the Pope et al. 2002 reanalysis of the ACS study data in estimating long-term exposure mortality. This study incorporates consideration for a variety of potential risk factors including smoking, educational status and age. With the exception of smoking status, none of the socio-economic factors examined in the Pope et al. 2002 reanalysis had a significant effect on the association between premature mortality and $PM_{2.5}$ exposure. Rather than representing confounders, several of these socio-economic factors, including educational status, were identified as potential effects modifiers.

for cohort exposure to $PM_{2.5}$ following implementation of $PM_{2.5}$ standard in 1999; (c) controlling for a variety of personal risk factors including occupational exposure and diet; and (d) using advanced statistical methods to evaluate specific issues that can adversely affect risk estimates including the possibility of spatial autocorrelation of survival times in communities located near each other. Because of these refinements, the SAB- HES recommends using the Pope et al. (2002) study as the basis for the primary mortality estimate for adults and suggests that alternate estimates of premature mortality generated using other cohort and time series studies could be included as part of the sensitivity analysis (SAB-HES, 2003).

The SAB-HES also recommended using the estimated relative risks from the Pope et al. (2002) study based on the average exposure to $PM_{2.5}$, measured by the average of two $PM_{2.5}$ measurements, over the periods 1979-1983, and 1999-2000. In addition to relative risks for all-cause mortality, the Pope et al. (2002) study provides relative risks for cardiopulmonary, lung cancer, and all other cause mortality.[X] Because of concerns regarding the statistical reliability of the all-other cause mortality relative risk estimates, we calculate premature mortality impacts for the primary analysis based on the all-cause relative risk. However, we provide separate estimates of cardiopulmonary and lung cancer deaths to show how these important causes of death are affected by reductions in $PM_{2.5}$.

In previous RIAs, infant mortality has not been evaluated as part of the primary analysis because of uncertainty in the strength of the association between exposure to PM and postneonatal mortality. Instead, benefits estimates related to reduced infant mortality have been included as part of the sensitivity analysis for RIAs. However, recently published studies have strengthened the case for an association between PM exposure and respiratory inflamation and infection leading to premature mortality in children under 5 years of age. Specifically, the SAB-HES noted the release of the World Health Organization Global Burden of Disease Study focusing on ambient air, which cites several recently published time-series studies relating daily PM exposure to mortality in children (SAB-HES, 2003). The SAB-HES also cites the study by Belanger et al. (2003) as corroborating findings linking PM exposure to increased respiratory

[X] Commentors pointed out that both cardiovascular disease and cancer have latency periods of from 15 to 20 years. Therefore, given that PM concentrations were four times higher in the 1960's compared with the 1980's, we may be overestimating mortality incidence by using effects estimates, based on the original ACS study data, that do not sufficiently correct for these higher PM concentrations during earlier segments of the exposure period for target populations. We recognize that uncertainty is introduced into benefits estimates as a result of both latency and lag issues. As the SAB-HES pointed out, the lack of detailed temporal exposure data for long term prospective cohort studies makes it difficult to characterize latency and lag periods and evaluate the importance of temporal variation in exposure levels. The Pope et al. 2002 reanalysis of the ACS study data, which includes additional years of follow-up data for the original study population, does suggest that lung cancer may have a longer latency period. However, inclusion of additional years of exposure data, in the case of lung cancer has served to strengthen, rather than weaken the association between $PM_{2.5}$ and premature mortality. By contrast, inclusion of additional follow-on data for cardiovascular effects has suggested that this endpoint may have a shorter latency/lag period in that the effects estimate has been reduced and not strengthened with the inclusion of the additional data.

inflamation and infections in children. Recently, a study by Chay and Greenstone (2003) found that reductions in TSP caused by the recession of 1981-1982 were related to reductions in infant mortality at the county level. With regard to the cohort study conducted by Woodruff et al. (1997), the SAB- HES notes several strengths of the study, including the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control for a variety of individual risk factors in infants (e.g., maternal educational level, maternal ethnicity, parental marital status, and maternal smoking status). Based on these findings, the SAB-HES recommends that the EPA incorporate infant mortality into the primary benefits estimate and that infant mortality be evaluated using a impact function developed from the Woodruff et al. (1997) study (SAB-HES, 2003).

Chronic Bronchitis. Chronic bronchitis is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. Chronic bronchitis affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). A limited number of studies have estimated the impact of air pollution on new incidences of chronic bronchitis. Schwartz (1993) and Abbey et al.(1995) provide evidence that long-term PM exposure gives rise to the development of chronic bronchitis in the United States. Because the Nonroad Diesel regulations are expected to reduce primarily $PM_{2.5}$, this analysis uses only the Abbey et al. (1995) study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of chronic bronchitis.

Nonfatal Myocardial Infarctions (heart attacks). Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001) and other countries (Poloniecki et al. ,1997). We use a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between $PM_{2.5}$ and nonfatal 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 nonfatal 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 nonfatal heart attacks based on the single available U.S. effect estimate. 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 United States. These studies provide a weight of evidence for this type of effect, as discussed in the Criteria Document. 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 a.l, 2002; Dekker et al., 2000; Liao et al., 1997, Tsuji et al., 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

Hospital and Emergency Room Admissions. Because of the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, many of the hospital admission endpoints use pooled impact functions based on the results of a number of studies. 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 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 United States are asthma-related visits.

To estimate avoided incidences of cardiovascular hospital admissions associated with $PM_{2.5}$, we use studies by Moolgavkar (2003) and Ito et al. (2003). There are additional published studies showing a statistically significant relationship between PM_{10} 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 provide an effect estimate for populations over 65, allowing us to pool the impact functions for this age group. Only Moolgavkar (2000) provided a separate effect estimate for populations 20 to 64.[y] 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. To avoid double counting benefits from reductions in myocardial infarctions when applying the impact function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarctions.

To estimate total avoided incidences of respiratory hospital admissions, we use impact 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 PM_{10} and respiratory

[Y]Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we have chosen to use the existing study. Given the very small (<5 percent) difference in the effect estimates for 65 and older cardiovascular hospital admissions between the original and reanalyzed results, we do not expect there to be much bias introduced by this choice.

hospital admissions. We use only those focusing on $PM_{2.5}$. Both Moolgavkar (2000) and Ito et al. (2003) provide effect estimates for COPD in populations over 65, allowing us to pool the impact functions for this group. Only Moolgavkar (2000) provided a separate effect estimate for populations 20 to 64.[z] 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 Ito et al. (2003) estimated pneumonia, and only for the population 65 and older. In addition, Sheppard et al. (2003) provided an effect estimate 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 effect estimate from 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 PM_{10} rather than $PM_{2.5}$. We selected the Norris et al. (1999) effect estimate 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.

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 effect estimates for two different cities relating ozone and hospital admissions for all respiratory causes (defined as ICD codes 460-519). Impact functions based on these studies 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 impact functions for Minneapolis are pooled together first, and the resulting impact function is then pooled with the impact 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. 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 an effect estimate for respiratory hospital admissions in children under 2.

[z]Again, given the very small (<10 percent) difference in the effect estimates for 65 and older COPD hospital admissions between the original and reanalyzed results, we do not expect there to be much bias introduced by this choice.

Acute Health Events and School/Work Loss Days. As indicated in Table 9A-1, in addition to mortality, chronic illness, and hospital admissions, a number of acute health effects not requiring hospitalization are associated with exposure to ambient levels of ozone and PM. The sources for the effect estimates used to quantify these effects are described below.

Around 4 percent of U.S. children between ages 5 and 17 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,[aa] 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 5 and 17 are estimated using an effect estimate developed from Dockery et al. (1996).

Incidences of lower respiratory symptoms (e.g., wheezing, deep cough) in children aged 7 to 14 are estimated using an effect estimate from Schwartz and Neas (2000).

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (e.g., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms has thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged 9 to 11 are estimated using an effect estimate 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 an effect estimate 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.

[AA]See http://www.nlm.nih.gov/medlineplus/ency/article/000124.htm, accessed January 2002.

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 an effect estimate derived from Ostro and Rothschild (1989).

In previous RIAs, we have not included estimates of asthma exacerbations in the asthmatic population in the primary analysis because of concerns over double counting of benefits and difficulties in differentiating asthma symptoms for purposes of first developing impact functions that cover distinct endpoints and then establishing the baseline incidence estimates required for predicting incidence reductions. Concerns over double counting stem from the fact that studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double counting. In one specific case (upper respiratory symptoms in children), the only study available was limited to asthmatic children, so this endpoint can be readily included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms and MRADs, are estimated for the total population that includes asthmatics. Therefore, to simply add predictions of asthma-related symptoms generated for the population of asthmatics to these total population-based estimates could result in double counting, especially if they evaluate similar endpoints.

The SAB-HES, in commenting on the analytical blueprint for 812 acknowledged these challenges in evaluating asthmatic symptoms and appropriately adding them into the primary analysis (SAB-HES, 2003). However, despite these challenges, the SAB-HES recommends the addition of asthma-related symptoms (i.e., asthma exacerbations) to the primary analysis, provided that the studies use the panel study approach and that they have comparable design and baseline frequencies in both asthma prevalence and exacerbation rates. Note also, that the SAB-HES, while supporting the incorporation of asthma exacerbation estimates, does not believe that the association between ambient air pollution, including ozone and PM, and the new onset of asthma is sufficiently strong to support inclusion of this asthma-related endpoint in the primary estimate. For this analysis, we have followed the SAB-HES recommendations regarding asthma exacerbations in developing the primary estimate. To prevent double counting, we are focusing the estimation on asthma exacerbations occurring in children and are excluding adults from the calculation. Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, we would likely double count incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double counting.

To characterize asthma exacerbations in children, we selected two studies (Ostro et al., 2001 and Vedal et al., 1998) that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between $PM_{2.5}$, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates (see below). Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM_{10} and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations. We employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al. study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled with the cough-related estimate generated using the Vedal study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

Additional epidemiological studies are available for characterizing asthma-related health endpoints (the full list of epidemiological studies considered for modeling asthma-related incidence are presented in Table 9A-20). However, based on recommendations from the SAB-HES, we decided not to use these additional studies in generating the primary estimate. In particular, the Yu et al. (2000) estimates show a much higher baseline incidence rate than other studies, which may lead to an overstatement of the expected impacts in the overall asthmatic population. The Whittemore and Korn (1980) study did not use a well-defined endpoint, instead focusing on a respondent-defined "asthma attack." Other studies looked at respiratory symptoms in asthmatics but did not focus on specific exacerbations of asthma.

9A.3.5.2 Uncertainties Associated with Health Impact 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 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.

Table 9A-20. Studies Examining Health Impacts in the Asthmatic Population Evaluated for Use in the Benefits Analysis

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

Application of C-R Relationship Nationwide. Regardless of the use of impact functions based on effect estimates from a single epidemiological study or multiple studies, each impact function was applied uniformly throughout the United States to generate health benefit estimates. However, to the extent that pollutant/health effect relationships are region-specific, applying a location-specific impact function at all locations in the United States 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 impact function to the entire United States. 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.[bb]

Extrapolation of Impact Functions Across Populations. Epidemiological studies often focus on specific age ranges, either due to data availability limitations (e.g., most hospital admission data come from Medicare records, which are limited to populations 65 and older), or to simplify data collection (e.g., 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 most impact functions should be applied only to those populations with ages that strictly match the populations in the underlying epidemiological studies. However, 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. In response to recommendations from the SAB-HES, where there appears to be a reasonable physiological basis for expanding the age group associated with a specific effect estimate beyond the study population to cover the full age group (e.g., expanding from a study population of 7 to 11 year olds to the full 6to 18 year child age group), we have done so and used those expanded incidence estimates in the primary analysis.

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 premature mortality rates. However, much about this relationship is still uncertain. These uncertainties include the following:

[BB]Although we are not able to use region-specific effect estimates, 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.

- Causality: A substantial number of published epidemiological studies recognize an association between elevated PM concentrations and increased premature mortality rates; however, these epidemiological studies are not designed to and cannot definitively prove causation. For the analysis of the final Nonroad Diesel Engines 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 premature 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 premature mortality effects independent of PM, but we do not view the evidence as conclusive at this time. The EPA is currently evaluating the epidemiological literature on the relationship between ozone and premature mortality and in future regulatory analyses may include ozone mortality as a separate impact in the primary analysis. To the extent that the effect estimates we use to evaluate the preliminary control options in fact capture premature mortality effects of other criteria pollutants besides PM, we may be overestimating the benefits of reductions in PM. However, we are not providing separate estimates of the premature mortality benefits from the ozone and CO reductions likely to occur due to the preliminary control options.

- Shape of the C-R Function: The shape of the true PM premature 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 premature mortality may be mis-estimated.

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

- Exposure/Mortality Lags: There is a potential time lag between changes in PM exposures and changes in premature mortality rates. For the chronic PM/mortality relationship, the length of the lag is unknown and may be dependent on the kind of exposure. The existence of such a lag is important for the valuation of premature mortality incidence because economic theory suggests that benefits occurring in the future should be discounted. There is no specific scientific evidence of the existence or structure of a PM effects lag. However, current scientific literature on adverse health effects similar to those associated with PM (e.g., smoking-related disease) and the difference in the effect size between chronic exposure studies and daily mortality

studies suggests that all incidences of premature mortality reduction associated with a given incremental change in PM exposure probably would not occur in the same year as the exposure reduction. The smoking-related literature also implies that lags of up to a few years or longer are plausible. Adopting the lag structure used in the Tier 2/Gasoline Sulfur and Heavy-Duty Engine/Diesel Fuel RIAs and endorsed by the SAB (EPA-SAB-COUNCIL-ADV-00-001, 1999), we assume a 5-year lag structure. This approach assumes that 25 percent of PM-related premature deaths occur in each of the first 2 years after the exposure and the rest occur in equal parts (approximately 17 percent) in each of the ensuing 3 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 $\mu g/m^3$ decrease in daily $PM_{2.5}$ levels might decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. 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 (e.g., asthma). For both baseline incidence and prevalence data, we use age-specific rates where available. Impact 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, because of 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 are 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 final rule, we will continue to use current incidence rates.

Table 9A-21 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 3 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- to 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. Note that we have not projected any changes in mortality rates over time. We are aware that the U.S. Census projections of total and age-specific mortality rates used in our population projections are based on projections of declines in national level mortality rates for younger populations and increases in mortality rates for older populations over time. We are evaluating the most appropriate way to incorporate these projections of changes in overall national mortality rates into our database of county-level cause-specific mortality rates. In the interim, we have not attempted to adjust future mortality rates. This will lead to an overestimate of premature mortality benefits in future years, with the overestimation bias increasing the further benefits are projected into the future. We do not at this time have a quantified estimate of the magnitude of the potential bias in the years analyzed for this rule (2010 and 2015).

Table 9A-21. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population (continued)

Endpoint	Parameter	Rates	
		Value	Source[a]
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children[d]	0.0012	Schwartz (1994, Table 2)
Hospitalizations	Daily hospitalization rate	Age, cause, and county-specific rate	CDC Wonder (1996-1998)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children	0.3419	Pope et al. (1991, Table 2)
Asthma ER visits	Daily asthma ER visit rate	Age, Region specific visit rate	2000 NHAMCS public use data files[c]; 1999 NHDS public use data files[b] (Adams et al., 1999, Table 47); U.S. Bureau of the Census (2000)
Work Loss Days	Daily WLD incidence rate per person (18-65)	0.00540	1996 HIS (American Lung Association, 2002b, Table 4)
Chronic Bronchitis	Annual prevalence rate per person	Age 18-24: 0.00678 Age 25-44: 0.00492 Age 45-64: 0.0367 Age 65 and older: 0.0505	
Minor Restricted Activity Days	Daily MRAD incidence rate per person	0.02137	Ostro and Rothschild (1989, p. 243)
School Loss Days[e]	Annual incidence rate per person	0.0587	Abbey et al. (1993, Table 3)
	Daily school absence rate per person	0.033	National Center for Education Statistics (1996)
Nonfatal MI (heart attacks)	Daily illness-related school absence rate per person	0.00378	1996 HIS (Adams et al., 1999, Table 47); estimate of 180 school days per year
	Daily nonfatal myocardial infarction incidence rate per person, 18+	Northeast: 0.0136 Midwest: 0.0159 South: 0.0146 West: 0.0135	1999 NHDS public use data files[b]; adjusted by 0.9318 prob. of surviving after 28 days (Rosamond et al., 1999)
Asthma Exacerbations	Daily *respiratory* illness related school absence rate per person	0.0411	1996 HIS (Adams et al., 1999, Table 47); estimate of 180 school days per year
	Incidence (and prevalence) among asthmatic African American children	0.0206	Ostro et al. (2001)
	- daily wheeze	Northeast: 0.0073 Midwest: 0.0092 South: 0.076 (0.273) West: 0.0124	Vedal et al. (1998)
	- daily cough	0.067 (0.145)	
	- daily dyspnea	0.057 (0.074)	
	Prevalence among asthmatic children		
	- daily wheeze	0.038	
	- daily cough	0.086	
	- daily dyspnea	0.045	
Acute Bronchitis[f]	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002a, Table 11)

The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: HIS refers to the National Health Interview Survey; NHDS—National Hospital Discharge Survey; NHAMCS—National Hospital Ambulatory Medical Care Survey.

[b] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/

[c] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/

[d] Lower Respiratory Symptoms are defined as ≥2 of the following: cough, chest pain, phlegm, wheeze

[e] 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.

(continued)

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-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.

9A.3.5.4 Accounting for Potential Health Effect Thresholds

When conducting clinical (chamber) and epidemiological studies, 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 nonzero 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 SAB Advisory Council for Clean Air Compliance, which provides advice and review of the EPA's methods for assessing the benefits and costs of the Clean Air Act under Section 812 of the Clean Air Act, has advised the EPA that there is currently no scientific basis for selecting a threshold of 15 $\mu g/m^3$ 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) that finds in most cases no evidence of a nonlinear relationship between PM and health effects and certainly does not find a distinct threshold. The most recent draft of the EPA Air Quality Criteria for Particulate Matter (EPA, 2004) 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).

Table 9A-22. Asthma Prevalence Rates Used to Estimate Asthmatic Populations in Impact 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[a]
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

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

Recent cohort analyses by HEI (Krewski et al., 2000) and Pope et al. (2002) provide additional evidence of a quasi-linear relationship between long-term exposures to $PM_{2.5}$ and premature 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 $\mu g/m^3$. 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 functions relating $PM_{2.5}$ and premature mortality "were not significantly different from linear associations."

Daniels et al. (2000) examined the presence of thresholds in PM_{10} C-R 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 PM_{10} on the types of premature 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 data from 10 cities, the combined C-R curve did not show evidence of a threshold in the PM_{10}-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 $\mu g/m^3$, 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 $\mu g/m^3$. However, the C-R 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 PM_{10} 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 affects 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 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-23 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, 2010 and 2015. 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. A discussion of the valuation methods for premature mortality and chronic bronchitis is provided here because of the relative importance of these effects. Discussions of the methods used to value nonfatal myocardial infarctions (heart attacks) and school absence days are provided because these endpoints have only recently been added to the analysis 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-23.

Table 9A-23. Unit Values Used for Economic Valuation of Health Endpoints (1999$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Premature Mortality (Value of a Statistical Life)	$5,500,000	$6,600,000	$6,800,000	Point estimate is the mean of a normal distribution with a 95 percent confidence interval between $1 and $10 million. Confidence interval is based on two meta-analyses of the wage-risk VSL literature. $1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. $10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population.
Chronic Bronchitis (CB)	$340,000	$420,000	$430,000	Point estimate 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.
Nonfatal Myocardial Infarction (heart attack)				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).
3% discount rate				
Age 0-24	$66,902	$66,902	$66,902	
Age 25-44	$74,676	$74,676	$74,676	
Age 45-54	$78,834	$78,834	$78,834	
Age 55-65	$140,649	$140,649	$140,649	
Age 66 and over	$66,902	$66,902	$66,902	
7% discount rate				
Age 0-24	$65,293	$65,293	$65,293	
Age 25-44	$73,149	$73,149	$73,149	
Age 45-54	$76,871	$76,871	$76,871	
Age 55-65	$132,214	$132,214	$132,214	
Age 66 and over	$65,293	$65,293	$65,293	

Lost earnings:
Cropper and Krupnick (1990). Present discounted value of 5 yrs of lost earnings:

age of onset:	at 3%	at 7%
25-44	$8,774	$7,855
45-54	$12,932	$11,578
55-65	$74,746	$66,920

Direct medical expenses: 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)

(continued)

Table 9A-23. Unit Values Used for Economic Valuation of Health Endpoints (1999$) (continued)

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.

(continued)

Table 9A-23. Unit Values Used for Economic Valuation of Health Endpoints (1999$) (continued)

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.
Asthma Exacerbations	$42	$45	$45	Asthma exacerbations are valued at $42 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study.
Acute Bronchitis	$360	$380	$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.

(continued)

Table 9A-23. Unit Values Used for Economic Valuation of Health Endpoints (1999$) (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level	2030 Income Level	
Restricted Activity and Work/School Loss Days				
Work Loss Days (WLDs)	Variable	Variable	Variable	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.
School Absence Days	$75	$75	$75	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. 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.
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	$54	$55	Median WTP estimate to avoid one MRAD from Tolley, et al. (1986) .

9A.3.5.5.1 Valuing Reductions in Premature Mortality Risk.

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 premature 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 assumed to be $5.5 million in 1999 dollars. This represents a central value consistent with the range of values suggested by recent meta-analyses of the wage-risk VSL literature. The distribution of VSL is characterized by a confidence interval from $1 to $10 million, based on two meta-analyses of the wage-risk VSL literature. The $1 million lower confidence limit represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. The $10 million upper confidence limit represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis.

In previous analyses, we used an estimate of mean VSL equal to $6.3 million, based on a distribution fitted to the estimates from 26 value-of-life studies identified in the Section 812 reports as "applicable to policy analysis." [cc]

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 5 years following exposure. To take this into account in the valuation of reductions in premature mortality, we apply 3 percent and 7 percent discount rates to the value of premature mortality occurring in future years.[dd]

[CC] Commentors have suggested that the VSL used in the Draft RIA may not be appropriate for populations impacted by the rule in that it may not reflect the risk preference of the of the target population. We recognize the large amount of uncertainty in the VSL for application to environmental policy. Following SAB-EEAC guidance, we used a wage-risk-based VSL in valuing premature mortality for the primary estimate in the final rule. In response to concerns about the range of estimates included in the VSL distribution, we modified the value of life distribution used for the final rule. As described above, the new mean value of avoiding one statistical death ($5.5 million in 1999 dollars) represents a central value consistent with the range of values suggested by recent meta-analyses of the wage-risk VSL literature. The distribution of VSL used in this RIA is characterized by a confidence interval from $1 to $10 million, based on two meta-analyses of the wage-risk VSL literature. Following SAB-EEAC guidance, we discount over the lag period between exposure and premature mortality in valuing reductions in mortality incidence (see Section 9.A.3.5.2).

[DD] The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the federal government. The 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 the EPA's *Guidelines for Preparing Economic Analyses* (EPA 2000c).

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 economics and public policy analysis community. Regardless of the theoretical economic considerations, the 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.

Following the advice of the EEAC of the SAB, the EPA currently uses the VSL approach in calculating the primary estimate of premature mortality benefits, because we believe this calculation provides the most reasonable single estimate of an individual's willingness to trade off money for reductions in premature mortality risk (EPA-SAB-EEAC-00-013). Although there are several differences between the labor market studies the EPA uses to derive a VSL estimate and the PM 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-24 lists some of these differences and the expected effect on the VSL estimate for air pollution-related premature mortality. In the absence of a comprehensive and balanced set of adjustment factors, the EPA believes it is reasonable to continue to use the $5.5 million value while acknowledging the significant limitations and uncertainties in the available literature.

Table 9A-24. 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

Some economists emphasize that the VSL is not a single number relevant for all situations. Indeed, the VSL estimate of $5.5 million (1999 dollars) is itself the central tendency of a number of estimates of the VSL for some rather narrowly defined populations. When there are significant differences between the population affected by a particular health risk and the

populations used in the labor market studies, as is the case here, some economists prefer to adjust the VSL estimate to reflect those differences.

The SAB-EEAC has advised 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, EPA, 2000b). In developing our primary estimate of the benefits of premature mortality reductions, we have followed this advice and discounted over the lag period between exposure and premature mortality.

Uncertainties Specific to Premature Mortality Valuation. The economic benefits associated with premature mortality are the largest category of monetized benefits of this rule. In addition, in prior analyses, the EPA has identified valuation of premature mortality benefits as the largest contributor to the range of uncertainty in monetized benefits (see 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 premature 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 premature mortality or reflect differences in the nature of the risk presented by air pollution relative to the risks studied in the relevant economics 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. An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's 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 depend on such characteristics as age, health state, and the current age to which the individual is likely to survive.

Although a survival curve approach provides a theoretically preferred method for valuing the benefits of reduced risk of premature mortality associated with reducing air pollution, the

approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value avoided premature mortality risk using the VSL approach.

Other uncertainties specific to premature mortality valuation include the following:

- Across-study variation: There is considerable uncertainty as to whether the available literature on VSL provides adequate estimates of the VSL saved by air pollution reduction. Although there is considerable variation in the analytical designs and data used in the existing literature, 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 WTP to reduce the risk. The appropriateness of a distribution of WTP based on the current VSL literature for valuing the premature 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 the extent to which the risks being valued are similar and 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 VSL from the wage-risk studies to the context of the this 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 10 times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the VSL 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 job-related mortality risks may differ in several ways 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. Some evidence suggests 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.

- Self-selection and skill in avoiding risk. Recent research (Shogren et al., 2002) suggests that VSL estimates based on hedonic wage studies may overstate the average value of a risk reduction. This is based on the fact that the risk-wage tradeoff revealed in hedonic studies reflects the preferences of the marginal worker (i.e., that worker who demands the highest compensation for his risk reduction). This worker must have either higher risk, lower risk tolerance, or both. However, the risk estimate used in hedonic studies is generally based on average risk, so the VSL may be upwardly biased because the wage differential and risk measures do not match.

For more discussion, see Appendix 9B.

9A.3.5.5.2 Valuing Reductions in the Risk of Chronic Bronchitis.

The best available estimate of WTP to avoid a case of chronic bronchitis comes from Viscusi et al. (1991). The Viscusi et al. study, however, describes a severe case of chronic bronchitis to the survey respondents. We therefore employ an estimate of WTP to avoid a pollution-related case of chronic bronchitis, 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 chronic bronchitis is not as severe. The adjustment is made by applying the elasticity of WTP with respect to severity reported in the Krupnick and Cropper (1992) study. Details of this adjustment procedure are provided in the benefits TSD for the nonroad diesel rulemaking (Abt Associates, 2003).

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of chronic bronchitis in this analysis. The distribution incorporates uncertainty from three sources: the WTP to avoid a case of severe chronic bronchitis, as described by Viscusi et al.; the severity level of an average pollution-related case of chronic bronchitis (relative to that of the case described by Viscusi et al.); and the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these three uncertain components, we derive a distribution of WTP to avoid a pollution-related case of chronic bronchitis 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 chronic bronchitis.

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

The Agency has recently incorporated into its analyses the impact of air pollution on the expected number of nonfatal heart attacks, although it has examined the impact of reductions in other related cardiovascular endpoints. We were not able to identify a suitable WTP value for reductions in the risk of nonfatal heart attacks. Instead, we propose a COI unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with an myocardial infarction 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 3 percent discount rate, we estimated a present discounted value in lost earnings (in 2000$) over 5 years due to an myocardial infarction 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 7 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 found three possible sources in the literature of estimates of the direct medical costs of myocardial infarction:

- Wittels et al. (1990) estimated expected total medical costs of myocardial infarction 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 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 myocardial infarction 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 for myocardial infarction may have decreased from 1983 to the present. The average length of stay for ICD code 410 (myocardial infarction) 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 nonfatal myocardial infarction cases), whose length of stay was therefore substantially shorter than it would be if they had not died.

- Eisenstein et al. (2001) estimated 10-year costs of $44,663 in 1997$, or $49,651 in 2000$ for myocardial infarction 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 myocardial infarction 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 10-year period.

In summary, the three different studies provided significantly different values (see Table 9A-25).

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 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 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 Table 9A-26.

Table 9A-25. 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[a]	5
Russell et al. (1998)	$22,331[b]	5
Eisenstein et al. (2001)	$49,651[b]	10
Russell et al. (1998)	$27,242[b]	10

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

Table 9A-26. Estimated Costs Over a 5-Year Period (in 2000$) of a Nonfatal Myocardial Infarction

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

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

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: 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 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 three significant problems with this method, however. First, it omits WTP to avoid the symptoms/illness that 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). Third, it makes an assumption about the gender of the parent that would miss work. 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, lower 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 Table 9A-27. We do not take into account that many single and "other" women with children may lose their jobs if they are repeatedly absent due to their children's illnesses.

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.

Table 9A-27. Women with Children: Number and Percent in the Labor Force, 2000, and Weighted Average Participation Rate[a]

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

[a] 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.

[b] Widowed, divorced, or separated.

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.[ee]

9A.3.5.6 Unquantified Health Effects

In addition to the health effects discussed above, there is emerging evidence that human exposure to ozone may be associated with premature mortality (Ito and Thurston, 1996; Samet, et al. 1997, Ito and Thurston, 2001), PM and ozone with increased emergency room visits for non-asthma respiratory causes (US EPA, 1996a; 1996b), ozone with impaired airway responsiveness (US EPA, 1996a), ozone with increased susceptibility to respiratory infection (US EPA, 1996a), ozone with acute inflammation and respiratory cell damage (US EPA, 1996a), ozone and PM with premature aging of the lungs and chronic respiratory damage (US EPA, 1996a; 1996b), ozone with onset of asthma in exercising children (McConnell et al. 2002), and PM with reduced heart rate variability and other changes in cardiac function. 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

[EE] In a very recent article, Hall, Brajer, and Lurmann (2003) use a similar methodology to derive a mid-estimate value per school absence day for California of between $70 and $81, depending on differences in incomes between three counties in California. Our national average estimate of $75 per absence is consistent with these published values.

to be PM or ozone-induced, effect estimates 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.

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.[ff]

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.[gg] 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.[hh]

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

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

[GG] The Clean Air Act designates 156 national parks and wilderness areas as Class I areas for visibility protection.

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

basis for monetary estimates of the benefits of visibility changes in recreational areas.[ii] 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.

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

[II] 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).

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.

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.[jj]

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.[kk] Dollar values are aggregated across 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.[ll] 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.

[JJ] 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.

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

[LL] The total value for these crops in 1998 was $47 billion.

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 phyisical 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 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 and Odor

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 9C.

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.

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, 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.

The total monetized estimates are dominated by benefits of premature mortality risk reductions. Our benefits analysis projects that the modeled preliminary control options will result in 7,800 avoided premature deaths in 2020 and 14,000 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 (and thus baseline mortality risk) of the population.

Our primary estimate of total monetized benefits (including PM health, ozone health and welfare, and visibility) in 2030 for the modeled nonroad preliminary control options is $96 billion using a 3 percent discount rate and $91 billion using a 7 percent discount rate. In 2020, the monetized benefits are estimated at $54 billion using a 3 percent discount rate and $51 billion using a 7 percent discount rate. Health benefits account for 97 percent of total benefits. The monetized benefit associated with reductions in the risk of premature mortality, which accounts for $89 billion in 2030 and $49 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. Visibility, 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.

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 known to

underestimate 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.

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: Long-term exposure (adults, 30 and over)[B]	7,800	13,800
Infant mortality (infants under one year)	18	26
Chronic bronchitis (adults, 26 and over)	4,300	6,500
Non-fatal myocardial infarctions (adults, 18 and older)	10,600	17,700
Hospital admissions — Respiratory (adults, 20 and older)[C]	3,400	6,000
Hospital admissions — Cardiovascular (adults, 20 and older)[D]	2,800	4,400
Emergency Room Visits for Asthma (18 and younger)	4,600	6,900
Acute bronchitis (children, 8-12)	10,000	16,000
Asthma exacerbations (asthmatic children, 6-18)	150,000	230,000
Lower respiratory symptoms (children, 7-14)	120,000	190,000
Upper respiratory symptoms (asthmatic children, 9-11)	92,000	141,000
Work loss days (adults, 18-65)	810,000	1,160,000
Minor restricted activity days (adults, age 18-65)	4,800,000	6,800,000
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]: (adults, 30 and over)	PM		
3% discount rate		$49,000	$89,000
7% discount rate		$46,000	$84,000
Infant mortality (infants under one year)	PM	$120	$180
Chronic bronchitis (adults, 26 and over)	PM	$1,800	$2,800
Non-fatal myocardial infarctions	PM		
3% discount rate		$910	$1,440
7% discount rate		$880	$1,400
Hospital Admissions from Respiratory Causes[D,F]	O_3	$7.4	$21
	PM	$60	$110
Hospital Admissions from Cardiovascular Causes[E]	PM	$61	$96
Emergency Room Visits for Asthma	O_3	$0.03	$0.06
	PM	$1.3	$2.0
Acute bronchitis (children, 8-12)	PM	$3.9	$6.0
Asthma exacerbations (asthmatic children, 6-18)	PM	$6.9	$10.7
Lower respiratory symptoms (children, 7-14)	PM	$2.0	$3.1
Upper respiratory symptoms (asthmatic children, 9-11)	PM	$2.4	$3.7
Work loss days (adults, 18-65)	PM	$110	$150
Minor restricted activity days (adults, age 18-65)	O_3	($0.1)	$4.9
	PM	$260	$370
School absence days (children, age 6-11)	O_3	$4.8	$10
Worker productivity (outdoor workers, age 18-65)	O_3	$4.2	$6.9
Recreational visibility (86 Class I Areas)	PM	$1,300	$2,100
Agricultural crop damage (6 crops)	O_3	$88	$137
Monetized Total[H]	O_3 and PM		
3% discount rate		$54,000+B	$96,000+B
7% discount rate		$51,000+B	$91,000+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 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4).

[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.

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 NOx, SO_2, 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 14,000 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 9C 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; Ibald-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.

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APPENDIX 9B: Supplemental Analyses Addressing Uncertainties in the Concentration-Response and Valuation Functions for Particulate Matter Health Effects

9B.1 Introduction

In this appendix, we describe our progress toward improving our approach to characterizing the uncertainties in our economic benefits estimates, with particular emphasis on the concentration-response (C-R) function. We present two types of probabilistic approaches designed to illustrate how some aspects of the uncertainty in the C-R function might be handled in a PM benefits analysis. The first approach generates a probabilistic estimate of statistical uncertainty based on standard errors reported in the underlying studies used in the benefit modeling framework. The second approach uses the results from a pilot expert elicitation designed to characterize certain aspects of uncertainty in the ambient PM2.5/mortality relationship. For the reasons discussed earlier in Chapter 9, neither the primary benefit estimate nor these approaches have been used to inform any regulatory decisions in this rulemaking.

In any benefit analyses of air pollution regulations, estimation of pre-mature mortality accounts for 85 to 95 percent of total benefits. Therefore, it is an endpoint that will be an important focus for characterizing the uncertainty related to the estimates of total benefits. As part of a collaboration with the EPA's Office of Air and Radiation (OAR) and the Office of Management and Budget (OMB) on the Non-Road Diesel Rule, EPA extended it's collaboration with OMB in 2003 to conduct a pilot expert elicitation intended to more fully characterize uncertainty in the effect estimates used to estimate mortality resulting from exposure to PM.

It should be recognized that in addition to uncertainty, the annual benefit estimates for the Final Non-Road Diesel Rule also are inherently variable, due to the truly random processes that govern pollutant emissions and ambient air quality in a given year. Factors such as hourly use of engines and daily weather display constant variability regardless of our ability to accurately measure them. As such, the primary estimates of annual benefits presented in this chapter and the sensitivity analysis estimates presented in this and other appendices should be viewed as representative of the types of benefits that will be realized, rather than the actual benefits that would occur every year. As such, the distributions of the estimate 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.

9B.2 Monte Carlo Based Uncertainty Analysis Using Classical Statistical Sources of Uncertainty

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.

However, for this proposal we did not attempt to assign probabilities to all of the uncertain parameters in the model due to a lack of resources and reliable methods. At this time, we simply generate estimates of the distributions of dollar benefits for PM health effects and for total dollar benefits including visibility. We provide a likelihood distribution for the total benefits estimate, based solely on the statistical uncertainty surrounding the estimated C-R functions and the assumed distributions around the unit values.

Our estimate of the likelihood distribution for total benefits should be viewed as an approximate result because of the wide range of sources of uncertainty that we have not incorporated. The 5th and 95th percentile points of our estimate are based on statistical error and cross-study variability provides some insight into how uncertain our estimate is with regards to those sources of uncertainty. However, it does not capture other sources of uncertainty regarding other inputs to the model, including emissions, air quality, and aspects of the health science not captured in the studies, such as the likelihood that PM is causally related to premature mortality and other serious health effects..

Although there are several sources of uncertainty affecting estimates of endpoint-specific benefits, the sources of uncertainty that are most readily quantifiable in this analysis are the C-R relationships and uncertainty about unit dollar values. The total dollar benefit associated with a given endpoint depends on how much reducing risk of the endpoint will change due to the final standard (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth).[mm] However, as we have noted, this omits important sources of uncertainty, such as the contribution of air quality changes, baseline population incidences, projected populations exposed, transferability of the C-R function to diverse locations, and uncertainty about the C-R relationship for premature mortality. Thus, a likelihood description based on the standard error would provide a misleading picture about the overall uncertainty in the estimates. The empirical evidence about uncertainty is presented where it is available.

Both the uncertainty about the incidence changes and uncertainty about unit dollar values can be characterized by *distributions*. Each " likelihood distribution" characterizes our beliefs about what the true value of an unknown variable (e.g., the true change in incidence of a given health effect in relation to PM exposure) is likely to be, based on the available information from relevant studies.[nn] Unlike a sampling distribution (which describes the possible values that an

[MM] Because this is a national analysis in which, for each endpoint, a single C-R function is applied everywhere, there are two sources of uncertainty about incidence: (1) statistical uncertainty (due to sampling error) about the true value of the pollutant coefficient in the location where the C-R function was estimated, and (2) uncertainty about how well any given pollutant coefficient approximates β*.

[NN] Although such a "likelihood distribution" is not formally a Bayesian posterior distribution, it is very similar in concept and function (see, for example, the discussion of the Bayesian approach in Kennedy 1990, pp. 168-172).

estimator of an unknown variable might take on), this likelihood distribution describes our beliefs about what values the unknown variable itself might be. Such likelihood distributions can be constructed for each underlying unknown variable (such as a particular pollutant coefficient for a particular location) or for a function of several underlying unknown variables (such as the total dollar benefit of a regulation). In either case, a likelihood distribution is a characterization of our beliefs about what the unknown variable (or the function of unknown variables) is likely to be, based on all the available relevant information. A likelihood description based on such distributions are typically expressed as the interval from the fifth percentile point of the likelihood distribution to the ninety-fifth percentile point. If all uncertainty had been included, this range would be the "credible range" within which we believe the true value is likely to lie with 90 percent probability.

The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from these two sources (the C-R relationship and the valuation), and is estimated with a Monte Carlo method. In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution and a value is randomly drawn from the unit dollar value distribution, and the total dollar benefit for that iteration is the product of the two.[oo] If this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated.

Using this Monte Carlo procedure, a distribution of dollar benefits may be generated for each endpoint. As the number of Monte Carlo draws gets larger and larger, the Monte Carlo-generated distribution becomes a better and better approximation of a joint likelihood distribution for the considered likelihood distributions making up the overall model of total monetary benefits for the endpoint.

After endpoint-specific distributions are generated, the same Monte Carlo procedure can then be used to combine the dollar benefits from different (non-overlapping) endpoints to generate a distribution of total dollar benefits.

The estimate of total benefits may be thought of as the end result of a sequential process in which, at each step, the estimate of benefits from an additional source is added. Each time an estimate of dollar benefits from a new source (e.g., a new health endpoint) is added to the previous estimate of total dollar benefits, the estimated total dollar benefits increases. However, our bounding or likelihood description of where the true total value lies also increases as we add more sources.

[oo] This method assumes that the incidence change and the unit dollar value for an endpoint are stochastically independent.

As an example, consider the benefits from reductions in PM-related hospital admissions for cardiovascular disease. Because the actual dollar value is unknown, it may be described using a variable, with a distribution describing the possible values it might have. If this variable is denoted as X_1 , then the mean of the distribution, $E(X_1)$ and the variance of X_1, denoted $Var(X_1)$, and the 5th and 95th percentile points of the distribution (related to $Var(X_1)$), are ways to describe the likelihood for the true but unknown value for the benefits reduction.

Now suppose the benefits from reductions in PM-related hospital admissions for respiratory diseases are added. Like the benefits from reductions in PM-related hospital admissions for cardiovascular disease, the likelihood distribution for where we expect the true value to be may be considered a variable, with a distribution. Denoting this variable as X_2, the benefits from reductions in the incidence of *both* types of hospital admissions is $X_1 + X_2$. This variable has a distribution with mean $E(X_1 + X_2) = E(X_1) + E(X_2)$, and a variance of $Var(X_1 + X_2) = Var(X_1) + Var(X_2) + 2Cov(X_1,X_2)$; if X_1 and X_2 are stochastically independent, then it has a variance of $Var(X_1 + X_2) = Var(X_1) + Var(X_2)$, and the covariance term is zero.

The benefits from reductions in all non-overlapping PM-related health and welfare endpoints (X_{m+1}, ..., X_n) is $X = X_1 + ... + X_n$. The mean of the distribution of total benefits, X, is:

$$E(X) = E(X_1) + E(X_2) + ... + E(X_n) \quad \textbf{(1)}$$

and the variance of the distribution of total benefits -- assuming that the components are stochastically independent of each other (i.e., no covariance between variables) -- is:

$$Var(X) = Var(X_1) + Var(X_2) + ... + Var(X_n) \quad . \quad \textbf{(2)}$$

If all the means are positive, then each additional source of benefits increases the point estimate (mean) of total benefits. However, with the addition of each new source of benefits, the

$$E(X_1) < E(X_1 + X_2) < E(X_1 + X_2 + X_3) < ...< E(X_1 + ... + X_n) = E(X) \quad \textbf{(3)}$$

variance of the estimate of total benefits also increases. That is,
but:

$$Var(X_1) < Var(X_1 + X_2) < Var(X_1 + X_2 + X_3) < ...< Var(X_1 + ... + X_n) = Var(X) \ .$$

That is, the addition of each new source of benefits results in a larger mean estimate of total benefits (as more and more sources of benefits are included in the total) about which there is less certainty. This phenomenon occurs whenever estimates of benefits are added.

Calculated with a Monte Carlo procedure, the distribution of X is composed of random draws from the components of X. In the first draw, a value is drawn from each of the distributions, X_1, X_2, through X_n, these values are summed, and the procedure is repeated again, with the number of repetitions set at a high enough value (e.g., 5,000) to reasonably trace out the distribution of X. The fifth percentile point of the distribution of X will be composed of points pulled from all points along the distributions of the individual components, and not simply from the fifth percentile. While the sum of the fifth percentiles of the components would be represented in the distribution of X generated by the Monte Carlo, it is likely that this value would occur at a significantly lower percentile. For a similar reason, the 95th percentile of X will be *less* than the sum of the 95th percentiles of the components, and instead the 95th percentile of X will be composed of component values that are significantly lower than the 95th percentiles.

The physical effects estimated in this analysis are assumed to occur independently. It is possible that, for any given pollution level, there is some correlation between the occurrence of physical effects, due to say avoidance behavior or common causal pathways and treatments (e.g., stroke, some kidney disease, and heart attack are related to treatable blood pressure). Estimating accurately any such correlation, however, is beyond the scope of this analysis, and instead it is simply assumed that the physical effects occur independently.

We conduct two different Monte Carlo analyses, one based on the distribution of reductions in premature mortality characterized by the mean effect estimate and standard error from the epidemiology study of PM-associated mortality associated with long-term exposure used in the primary estimate in Chapter 9 (Pope et al., 2002), and one based on the results from a pilot expert elicitation project (Industrial Economics, 2004). In both analyses, the distributions of all other health endpoints are characterized by the reported mean and standard deviations from the epidemiology literature. Distributions for unit dollar values are based on reported ranges or distributions of values in the economic literature and are summarized in Table 9B-1. We are unable at this time to characterize the uncertainty in the estimate of benefits of improvements in visibility at Class I areas. As such, we treat the visibility benefits as fixed and add them to all percentiles of the PM health benefits distribution. Results of the Monte Carlo analysis based on the Pope et al. (2002) distribution are presented in the next section. Results of the Monte Carlo analysis based on the pilot expert elicitation are presented in section 9B.3.

9B.2.1 Monte Carlo Analysis Using Pope et al. (2002) to Characterize the Distribution of Reductions in Premature Mortality

Based on the Monte Carlo techniques described earlier, we generated likelihood distributions for the dollar value of reductions in PM-related health endpoints and a similar distribution for total annual PM-related benefits including PM health and visibility benefits for the nonroad diesel modeled preliminary control option. For this analysis, the likelihood descriptions for the

true value for each of the PM health endpoint incidence measures, including premature mortality, were based on classical statistical uncertainty measures, including the mean and standard deviation for the C-R relationships in the epidemiological literature, and assumption of particular likelihood distribution shapes for the valuation for each health endpoint values based on reported values in the economic literature. Table 9B-1 summarizes the chosen parameters for likelihood distributions for unit values for each of the PM health effects included in the Monte Carlo simulation. The distributions for the value used to represent incidence of a health effect in the total benefits valuation represent both the simple statistical uncertainty surrounding individual effect estimates and, for those health endpoints with multiple effects from different epidemiology studies, interstudy variability. Visibility benefits are also included in the distribution of total benefits, however, we were unable to characterize a distribution for visibility benefits. As such, they are simply added to each percentile of the distribution of PM health benefits.

Table 9B-1. Distributions for Unit Values of Health Endpoints

Health Endpoint	Mean Value, Adjusted for Income Growth to 2030	Derivation of Distribution
Premature Mortality (Value of a Statistical Life)	$5,500,000	Normal distribution anchored at 2.5th and 97.5th percentiles of $1 and $10 million, respectively. Confidence interval is based on two meta-analyses of the wage-risk VSL literature. $1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. $10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population. Normal distribution chosen through best professional judgment.
Chronic Bronchitis (CB)	$430,000	The distribution of WTP to avoid a case of pollution-related CB was generated by Monte Carlo methods, drawing from each of three distributions: (1) WTP to avoid a severe case of CB is assigned a 1/9 probability of being each of the first nine deciles of the distribution of WTP responses in Viscusi et al., 1991; (2) the severity of a pollution related case of CB (relative to the case described in the Viscusi study) is assumed to have a triangular distribution, centered at severity level 6.5 with endpoints at 1.0 and 12.0 (see text for further explanation); and (3) the constant in the elasticity of WTP with respect to severity is normally distributed with mean = 0.18 and standard deviation = 0.0669 (from Krupnick and Cropper, 1992). This process and the rationale for choosing it is described in detail in the Costs and Benefits of the Clean Air Act, 1990 to 2010 (U.S. EPA, 1999)
Nonfatal Myocardial Infarction (heart attack) 3% discount rate Age 0-24 Age 25-44 Age 45-54 Age 55-65 Age 66 and over 7% discount rate Age 0-24 Age 25-44 Age 45-54 Age 55-65 Age 66 and over	 $66,902 $74,676 $78,834 $140,649 $66,902 $65,293 $73,149 $76,871 $132,214 $65,293	No distribution available. 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). Lost earnings: Cropper and Krupnick (1990). Present discounted value of 5 yrs of lost earnings: age of onset: at 3% at 7% 25-44 $8,774 $7,855 45-54 $12,932 $11,578 55-65 $74,746 $66,920 Direct medical expenses: 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)
Hospital Admissions		
Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)	$12,378	No distribution available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, 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).

Health Endpoint	Mean Value, Adjusted for Income Growth to 2030	Derivation of Distribution
Pneumonia (ICD codes 480-487)	$14,693	No distribution available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, 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	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	No distribution available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, 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	No distribution available. The COI point estimate is the 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		
Upper Respiratory Symptoms (URS)	$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. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, we assume a uniform distribution between $10 and $45.
Lower Respiratory Symptoms (LRS)	$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. In the absence of information surrounding the frequency with which each of the eleven types of LRS occurs within the LRS symptom complex, we assume a uniform distribution between $8 and $25.
Asthma Exacerbations	$45	Asthma exacerbations are valued at $45 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between $17 and $73.

Health Endpoint	Mean Value, Adjusted for Income Growth to 2030	Derivation of Distribution
Acute Bronchitis	$390	Assumes a 6 day episode, with the distribution of the daily value specified as uniform with the low and high values based on those recommended for related respiratory symptoms in Neumann, et al. 1994. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day.
Restricted Activity and Work Loss Days		
Work Loss Days (WLDs)	Variable	No distribution available. Point estimate is based on county-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5 – to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.
Minor Restricted Activity Days (MRADs)	$55	Median WTP estimate to avoid one MRAD from Tolley, et al. (1986) . Distribution is assumed to be triangular with a minimum of $22 and a maximum of $83. Range is based on assumption that value should exceed WTP for a single mild symptom (the highest estimate for a single symptom--for eye irritation--is $16.00) and be less than that for a WLD. The triangular distribution acknowledges that the actual value is likely to be closer to the point estimate than either extreme.

Results of the Monte Carlo simulations are presented in Table 9B-2. The table provides the estimated means of the distributions and the estimated 5th and 95th percentiles of the distributions. The contribution of mortality to the mean benefits and to both the 5th and 95th percentiles of total benefits is substantial, with mortality accounting for over 90 percent of the mean estimate, and even the 5th percentile of mortality benefits dominating the 95th percentile of all other benefit categories. Thus, the choice of value and the shape for likelihood distribution for VSL should be examined closely and is key information to provide to decision makers for any decision involving this variable. The 95th percentile of total benefits is approximately twice the mean, while the 5th percentile is approximately one fourth of the mean. The overall range from 5th to 95th represents about one order of magnitude.

Table 9B-2.
Distribution of Value of Annual Human Health and Welfare Benefits in 2030 for the Modeled Preliminary Control Option of the Non-Road Diesel Rule[A]

Endpoint	Monetary Benefits[B, C] (Millions 2000$, Adjusted for Income Growth)		
	5th Percentile	Mean	95th Percentile
Premature mortality[D]			
Long-term exposure, (adults, >30yrs)	$20,000	$89,000	$180,000
Long-term exposure (child <1yr)	$40	$180	$350
Chronic bronchitis (adults, 26 and over)	$200	$2,800	$9,400
Non-fatal myocardial infarctions (adults, 18 and over)	$300	$1,400	$3,300
Hospital Admissions from Respiratory Causes[E]	$17	$36	$54
Hospital Admissions from Cardiovascular Causes[F]	$59	$96	$130
Emergency Room Visits for Asthma (children, <18)	$1.3	$2.2	$3.4
Acute bronchitis (children, 8-12)	($0.2)	$5.9	$15
Lower respiratory symptoms (children, 7-14)	$1.1	$2.9	$5.4
Upper respiratory symptoms (asthmatic children, 9-11)	$0.9	$3.7	$7.7
Work loss days (adults, 18-65)	$140	$160	$180
Asthma exacerbations (asthmatic children, 6-18)	$0.2	$11	$29
Minor restricted activity days (adults, age 18-65)	$200	$340	$500
Recreational visibility (86 Class I Areas)	$1,700	$1,700	$1,700
Unquantified Benefits	B	B	B
Monetized Total[G]	$23,000+B	$96,000+B	$200,000+B

[A] The benefit estimates provided in this table are based on the modeled air quality data for the preliminary control option used in the Non-Road Diesel proposal analysis and do not reflect the predicted emission reductions of the final rule's stringency levels. In the primary estimate in Chapter 9, the modeled benefits were scaled to the level necessary to reflect the predicted emission reductions of the final rule. The estimates provided in this table have not been scaled to the rule's stringency level, as the scaling methodology adds a new element of uncertainty that cannot be appropriately characterized here. These estimates should not be compared with the primary estimate provided in the chapter, but could be compared to results presented in Apprendix 9A.

[B] Monetary benefits are rounded to two significant digits.

[C] Monetary benefits are adjusted to account for growth in real GDP per capita between 1990 and 2030.

[D] The valuation of mortality assumes the 5 year distributed lag structure described earlier. Impacts of alternative lag structures are provided in a sensitivity analysis in Appendix 9C. Results reflect the use of 3% and 7% discount rates consistent with EPA and OMB's guidelines for preparing economic analyses (US EPA, 2000c, OMB Circular A-4).

[E] Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.

[F] Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

[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.

9B.3 Expert Elicitation of PM Mortality

In its 2002 report, the NAS provides a number of recommendations on how EPA might improve the characterization of uncertainty in its benefits analyses. One recommendation was that "EPA should begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple-source uncertainty analyses. This shift will require specification of probability distributions for major sources of uncertainty. These distributions should be based on available data and expert judgment."(NAS, 2002: 14) The NAS elaborated on this recommendation by saying "although the specific methods for selection and elicitation of experts may need to be modified somewhat, the protocols that have been developed and tested by OAQPS [in prior EPA projects -- see below] provide a solid foundation for future work in the area. EPA may also consider having its approaches reviewed and critiqued by decision analysts, biostatisticians, and psychologists from other fields where expert judgment is applied." (NAS, 2002: 140). They recommended the use of formally elicited expert judgments, but noted that a number of issues must be addressed, and that sensitivity analyses would be needed for distributions that are based on expert judgment. They also recommended that EPA clearly distinguish between data-derived components of an uncertainty assessment and those based on expert opinions. As a first step in addressing the NAS recommendations regarding expert elicitation, EPA, in collaboration with OMB, conducted a pilot expert elicitation to characterize uncertainties in the relationship between ambient PM2.5 and mortality. While it is premature to include the results of the pilot in the primary analysis for this rulemaking, EPA and OMB believe this pilot is an important step in moving toward the goal of incorporating additional uncertainty analyses in its future primary benefits analyses.

This pilot was designed to provide EPA with an opportunity to improve its understanding of the design and application of expert elicitation methods to economic benefits analysis and lay the groundwork for a more comprehensive elicitation. For instance, the pilot was designed to provide feedback on the efficacy of the protocol developed and the analytic challenges, as well as to provide insight regarding potential implications of the results on the degree of uncertainty surrounding the C-R function for $PM_{2.5}$ mortality. The scope of the pilot was limited in that we focused the elicitation on the C-R function of PM mass rather than on individual issues surrounding an estimate of the change in mortality due to PM exposure. Also, to meet time constraints placed on the pilot, we selected expert s for participation from two previously established expert panels of the NAS, and chose not to conduct a workshop with the experts prior to the elicitation. The limited scope of the pilot meant that a full expert elicitation process was truncated and many aspects of the uncertainty surrounding the $PM_{2.5}$-mortality relationship could not be quantitatively characterized. Recognizing this, the results of the pilot are only used in this benefits estimation for illustrative purposes. A full description of the pilot is contained in a report titled, "An Expert Judgment Assessment of the Concentration-Response Relationship between PM2.5 Exposure and Mortality," (IEc, 2004) available in the public docket for this rule.

The analytic plan for the pilot was developed based on established elicitation methods as suggestedby the NAS and published in the peer-reviewed literature. The plan was internally reviewed by EPA and OMB scientists with experience using expert elicitation methods. The Health Effect Subcommittee (HES) of the Council on Clean Air Compliance Analysis (the "Council") then provided additional suggestions, which led to further changes in the elicitation protocol. However, it should be noted that the Council did not provide a complete peer review of the elicitation methods or interpretation of results. Finally, the protocol was tested on PM scientists from within EPA and external to the Agency, who would not be part of the final elicitation process. The project team that implemented the pilot consisted of individuals with experience in expert elicitation and individuals with expertise in PM health effects and health benefits.

As a final step in this carefully designed pilot, the EPA and OMB will sponsor an external peer review of the methods used in this pilot expert elicitation as well as the approaches to presenting the results (particularly with respect to combining results across experts), in accordance with EPA's peer review guidelines. Until the peer review is complete andthe comments of the reviewers addressed, we do not recommend use of these results for other regulatory analysis.

9B.3.1 Elicitation Method

Expert elicitation is a formal, highly structured and well documented process whereby expert judgments, usually of multiple experts, are obtained (U.S. NRC, 1996). Formal expert elicitation usually involves experts with training and expertise in statistics, decision analysis, and probability encoding who work with subject matter experts to structure questions about uncertain relationships or parameters and who design and implement the process used to obtain probability and other judgments from subject matter experts. Several academic traditions – judgment and decision-making, human factors, cognitive sciences, expert systems, management science, to name a few – have sought to understand how to successfully elicit probabilistic judgments from both lay people and experts (Morgan and Henrion 1990, Cooke 1991,; Wright and Ayton *1994,* Ayyub 2002). Over the past two decades, there has been an increasing number of studies that have used expert judgment techniques to characterize uncertainty in quantities of interest to environmental risk analysis and decision-making. North and Merkhofer (1976) considered the use of expert judgment in evaluating emission control strategies. As referred to by the NAS, the EPA's Office of Air Quality Planning and Standards (OAQPS) successfully used expert judgment to characterize uncertainty in the health effects of exposure to lead (McCurdy and Richmond, 1983; Whitfield and Wallsten, 1989) and to ozone (Whitfield et al. 1991; Winkler et al., 1995). Amaral (1983) and Morgan et al. (1984) used expert judgment in the evaluation of the transport and impacts of sulfur air pollution. Several studies have been done in the area of climate change (Manne and Richels, 1994; Nordhaus, 1994; Morgan and

Keith, 1995; Reilly et al, 2001). Hawkins and Evans (1989) used industrial hygienists to predict toluene exposures to workers involved in a batch chemical process. In a more recent use of expert judgment in exposure analysis, Walker et al. (2001, 2003) asked experts to estimate ambient, indoor and personal air concentrations of benzene. A few studies have used expert judgment to characterize uncertainty in chemical dose response: Hawkins and Graham (1988) and Evans et al. (1994) for formaldehyde and Evans et al. (1994b) for risk of exposure to chloroform in drinking water. Expert judgment has also been used in the characterization of residential radon risks (Krewski et al., 1999).

The literature (Granger and Morgan, 1990) suggest there are several steps involved in the design and implementation of an expert elicitation, including:

- developing a protocol that contains the specific content of the elicitation and the questions that will be asked of the experts,
- selection of experts,
- compiling a briefing book of materials that can be used by the experts as background information to respond to the elicitation,
- pilot testing the protocol,
- conducting the elicitation and summarizing the findings.

The pilot expert elicitation consisted of a series of structured questions, both quantitative and qualitative, about the nature of the $PM_{2.5}$/mortality relationship. The objective was to obtain experts' quantitative, probabilistic judgments about the average expected decrease in mortality rates associated with decreases in $PM_{2.5}$ exposures in the United States. These judgments were expressed in terms of median estimates and associated percentile values of an uncertainty distribution. The quantitative questions in the protocol asked experts to provide judgments about changes in mortality due exposure to $PM_{2.5}$. Specifically, they were asked to estimate:1) the percent change in annual non-accidental mortality associated with a 1 $\mu g/m^3$ change in annual average $PM_{2.5}$ (long-term exposure); and 2) the percent change in daily non-accidental mortality associated with a 10 $\mu g/m^3$ change in daily 24-hour average $PM_{2.5}$ (short-term exposure). For each type of exposure, each expert provided minimum, maximum, and median estimates, plus 5^{th}, 25^{th}, 75^{th}, and 95^{th} percentile values for the distribution describing his uncertainty in the mortality effect of the specified change in $PM_{2.5}$.

The pilot focused on eliciting judgments about the C-R function for PM2.5 mass (without regard to source) and their solicited opinions about the key factors influencing the uncertainty in estimating the PM2.5/mortality relationship. As a warm-up to answering the quantitative question, experts were asked their views on several key issues including: cause of death, mechanisms, thresholds, lag/cessation period, the relative effect of PM components and their sources, confounding, and effect modification. This discussion allowed the experts to articulate

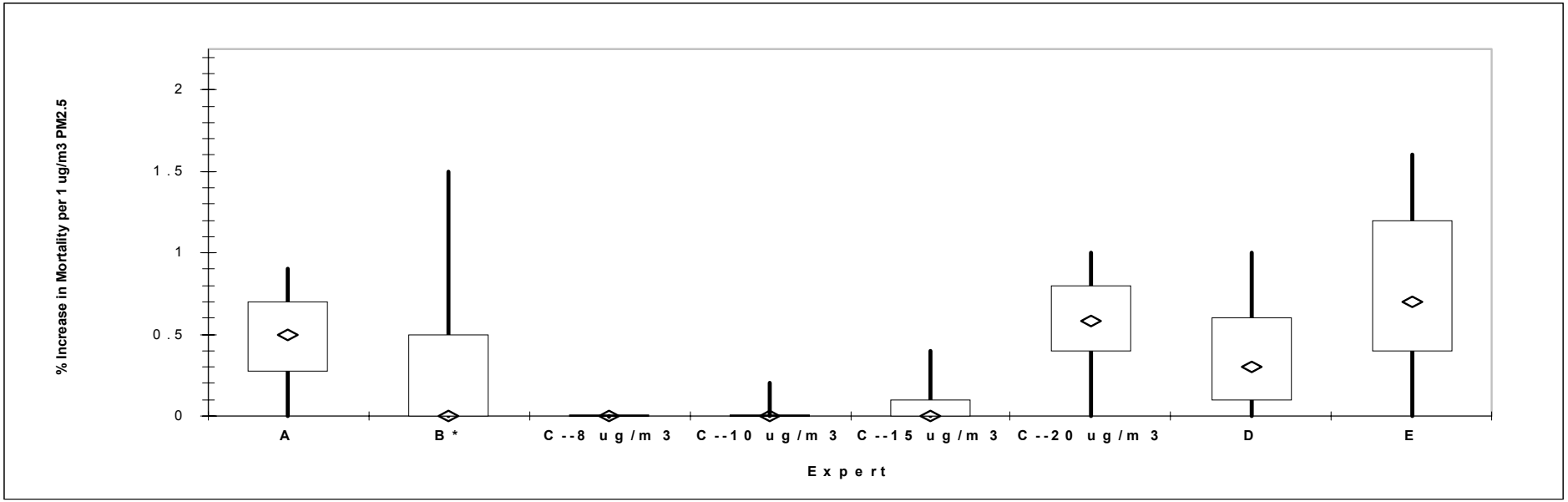
the way they interpreted the underlying issues, thus what would form the conceptual framework of their quantitative judgments. Their responses also provided EPA with information that would be useful for designing a more comprehensive and disaggregated elicitation assessment in the future.

The pilot elicitation consisted of personal interviews with five experts. The five experts were selected from an initial pool defined by the membership on two PM-related NAS committees. The rosters of both NRC committees included recognized experts in pertinent fields such as epidemiology and toxicology who had already undergone extensive review of their qualifications by the NRC, producing a reasonable initial list of experts likely to meet our expert selection criteria. The five experts selected for participation in the elicitation include: Dr. Roger McClellan, Dr. Bart Ostro, Dr. Jonathan Samet, Dr. Mark Utell, and Dr. Scott Zeger. The specific process used to select experts is detailed in the technical report of the elicitation (IEc, 2004) along with additional information about the experts' affiliations and fields of expertise. The size of the final expert panel was dictated by time and resource constraints, and the decision to restrict the initial expert pool to the NRC committees was made to help expedite the expert selection process. The experts were provided a briefing book of reference materials and a copy of the elicitation protocol prior to the interviews. Each interview lasts 6-8 hours.

9.B.3.2 Elicitation Results

Figure 9B-1 displays the responses of the experts to the quantitative elicitation question for the mortality effects of changes in long-term $PM_{2.5}$ exposures. The distributions provided by each expert, identified by the letters A through E, are depicted as box plots with the diamond symbol showing the median (50th percentile), a circle symbol showing the mean estimate, the box defining the interquartile range (bounded by the 25th and 75th percentiles), and the whiskers defining each expert's 90 percent confidence interval (bounded by the 5th and 95th percentiles of the distribution).

Figure 9B-1. Summary of Experts' Judgments About the Percent Increase in Annual Average Non-Accidental Mortality Associated with a 1 μg/m3 Increase in Annual Average Exposures to PM2.5



*Expert B specified this distribution for the PM/mortality coefficient above an uncertain threshold which he characterized as ranging between 4 and 15 with a modal value of 12 μg/m^3.As illustrated here, considerable variation exists in both the median values and the spread of uncertainty provided by the experts. The median value of the percent change in annual non-accidental mortality per unit change in annual $PM_{2.5}$ concentration (within a range of $PM_{2.5}$ concentrations from 8 to 20 μg/m^3) ranged from values at or near zero to a value of 0.7 percent. The variation in the responses largely reflects differences in the amount of uncertainty each expert considered inherent in the key epidemiological results from long-term cohort studies, the likelihood of a causal relationship, and the shape of the C-R function. The technical report (IEc, 2004) provides detailed descriptions of the experts' judgments about these factors, but we present a few brief observations relative to their responses below.

** Expert C specified a non-linear model and provided distributions for the slope of the curve at four discrete concentrations within the range.

As illustrated by the figure, the experts exhibited considerable variation in both the median values they reported and in the spread of uncertainty about the median. In response to the question concerning the effects of changes in long-term exposures to $PM_{2.5}$, the median value ranged from values at or near zero to a 0.7 percent increase in annual non-accidental mortality per 1 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentration (within a range of $PM_{2.5}$ concentrations from 8 to 20 $\mu g/m^3$). The variation in the responses for the effects of long-term exposures largely reflects differences of opinion among the experts on a number of factors such as key epidemiological results from long-term cohort studies, the likelihood of a causal relationship, and the shape of the C-R function. Some observations concerning the outcome of the individual expert judgments are provided below:

Key Cohort Studies. The experts' non-zero responses for the percent change in annual mortality were mostly influenced by the Krewski et al., (2000) reanalysis of the original American Cancer Society (ACS) cohort study and by the later Pope et al. (2002) update of the ACS study that included additional years of follow-up. None of the experts placed substantial weight on the mortality estimates from the Six-Cities study (Dockery et al., 1993) in composing their quantitative responses, despite citing numerous strengths of that analysis. Concern about sample size and representativeness of the Six Cities study for the entire U.S. appeared to be a major reason for de-emphasizing those results.

Causality for Long-Term Effects. Three of the five experts gave distributions more heavily weighted towards zero. Those experts were also the ones who gave the lowest probability of a causal effect of long-term exposure to $PM_{2.5}$ in the preliminary questions. All of the experts placed at least a 5 percent probability on the possibility that there is no causal relationship between fine PM exposure and mortality; as a result, all experts gave a fifth percentile value for the C-R coefficient of zero. For most of the experts, this was based primarily on residual concerns about the strength of the mechanistic link between the exposures and mortality.

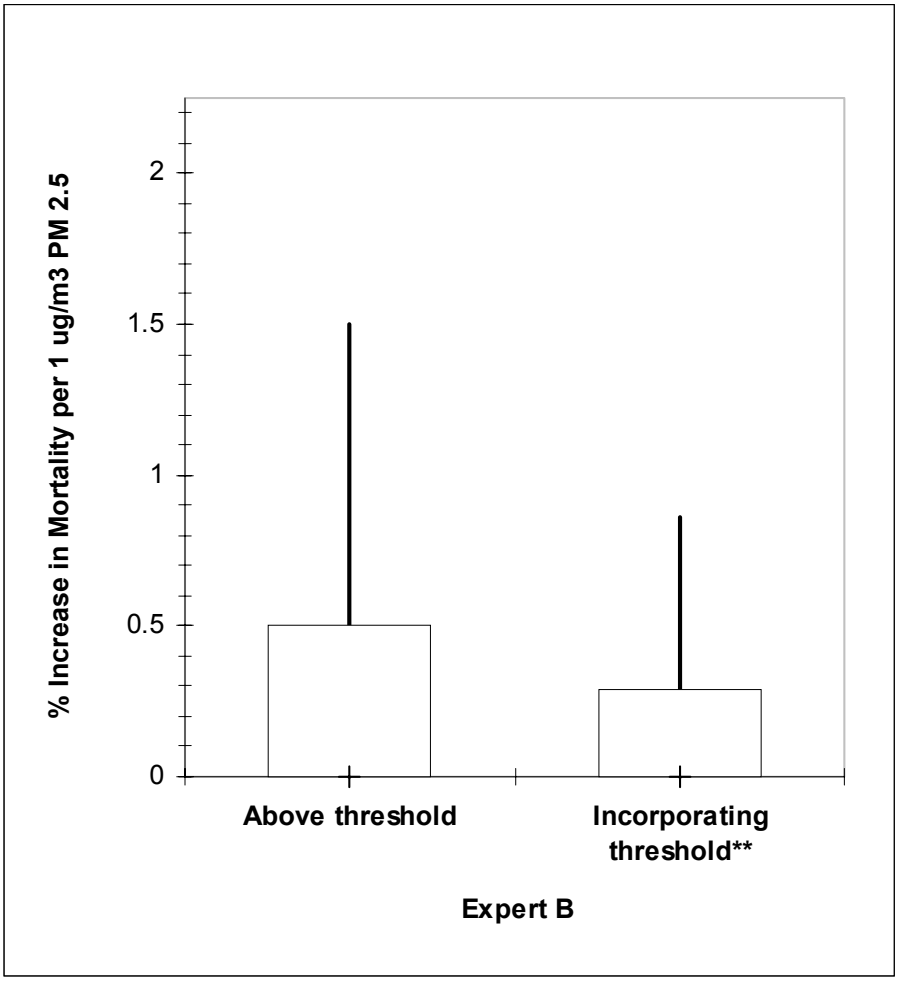
Shape of the C-R Function for Long-Term Effects. The other key determinant of each expert's responses for long-term effects was his assumption about the nature of the C-R function across the range of baseline annual average $PM_{2.5}$ concentrations assumed in the pilot (8 to 20 $\mu g/m^3$). Three experts (A, D, and E) assumed that the function relating mortality with PM concentrations would be log-linear with constant slope over the specified range. They therefore gave a single estimate of the distribution of the slope describing that log-linear function. The other two experts provided more complex responses.

Expert B assumed a population threshold in his model, below which there would be no effect of increased $PM_{2.5}$ exposure and above which the relationship would be log-linear. He characterized his estimate of a possible threshold as uncertain, ranging between 4 $\mu g/m^3$ and 15 $\mu g/m^3$, with a modal value of 12 $\mu g/m^3$. He then described a distribution for the slope for the log-linear function

that might exist above the threshold; this distribution is depicted in Figure 9B-2. The effect of incorporating the uncertain threshold is essentially to shift his entire distribution downward.

Expert C believed that the increased relative risks for mortality observed in the cohort studies were likely to be the result of exposures at the higher end of the exposure range, and he expected there to be a declining effect on mortality with decreasing levels of $PM_{2.5}$. He also argued that some practical concentration threshold was likely to exist below which we would not observe any increase in mortality. He reflected these beliefs by developing a non-linear model within the range from 8 to 20 $\mu g/m^3$; he described the model by providing distributions for the slope of the curve at four discrete concentrations within the range.

Figure 9B-2.
Expert B's Distributions for the Percent Increase in Annual Non-Accidental Mortality Associated with a 1 μg/m3 Increase in Long-term Exposures to PM2.5: Comparison of His Distribution Above a Threshold to His Expected Distribution* for the Range 8-20 μg/m3



* Expert B specified the threshold as uncertain between 4 and 15 μg/m^3 with a modal value at 12 μg/m3. He assumed the percent increase in mortality to increase linearly with concentration above the threshold. His effective distribution was simulated using Monte Carlo techniques assuming an underlying distribution of population-weighted annual average $PM_{2.5}$ concentrations for the U.S. generated from the BenMAP model (see the technical report (IEc, 2004) for details).

9B.3.3 Experts' Views of Sources of Uncertainty

The experts were asked at several points during the interview to discuss the key sources of potential bias and uncertainty in current evidence on which they relied for their judgments. In the context of the quantitative discussion they were asked to list the top five issues. They were encouraged to think about how these issues would affect the uncertainty surrounding their best estimate of the potential impact on total mortality of a small change in long-term exposure to $PM_{2.5}$. The tables summarizing the factors identified by each expert may be found in Appendix E of the technical report (IEc, 2004).

Many of the same factors appeared in the list of the five experts. However, the experts often differed on whether a particular factor was a source of potential bias or uncertainty. Some of the common concerns raised as either sources of bias or uncertainty, include:

- Residual confounding by smoking,
- Residual confounding by "life-style" or other personal factors or "stressors,"
- Exposure errors/misclassification,
- The role of co-pollutants as confounders or effect modifiers,
- Impact of the relative toxicity of PM components,
- Representativeness of the cohort populations with respect to the general U.S. population, and
- Investigator/publication biases.

Despite the many qualitative discussions about sources of uncertainty, because the pilot study did not elicit quantitative judgments about the size and nature of impacts of each source of uncertainty and bias, we were unable to systematically evaluate the nature of the influence of these factors on the quantitative results provided by each expert unless an expert explicitly adjusted his estimates by a particular factor.

9B.3.4 Advisory Council Comments on the Preliminary Design of the Elicitation

As part of a review of the analytical blueprint of the EPA's Second Prospective Analysis of the Costs and Benefits of the Clean Air Act under section 812 of the Act, a panel of outside experts - the Health Effects Subcommittee (HES) of the Advisory Council on Clean Air Compliance Analysis (Council)[pp] - provided a limited[qq] and preliminary review of the

[PP] The Council is an advisory committee with an independent statutory charter that is organized and supported under the EPA's Science Advisory Board.

[QQ] Council/HES report: "**...**in view of the fact that the pilot project is well-underway, the experts have already been selected, and many (if not all) of the interviews have been conducted, the HES sees little potential benefit in providing detailed suggestions about the design or conduct of the pilot study." (EPA-SAB-COUNCIL-ADV-04-002,

methodology and design of the expert elicitation. In an Advisory issued by the Council to the EPA (EPA-SAB-COUNCIL-ADV-04-002, March 2004), the Council-HES provided the following comments with regard to the elicitation:

- "We applaud the Agency's interest in exploring the use of formal expert judgment as a tool for improving uncertainty analysis and believe that the proposed pilot study has great potential to yield important insights. The pilot is well designed to inform subsequent and more comprehensive expert elicitation projects, but relies on the opinions of a relatively small group of experts. It may provide preliminary information about the general magnitude of the mortality effects, and may yield a sense of both the uncertainty inherent in these estimates and the factors largely responsible for such uncertainty. However, until the pilot study methods and results have been subjected to peer review, it may be unwise for the Agency to rely directly on these preliminary results in key policy decisions."

- In presenting results of the pilot elicitation, "the HES advises the EPA to present the entire collection of individual judgments; to carefully examine the collection of individual judgments noting the extent of agreement or disagreement; to thoughtfully assess the reasons for any disagreement; and to consider formal combinations of judgments only after such deliberation and with full awareness of the context ..."

- "The HES recognizes that in order to make the pilot tractable it was necessary to limit participation, and is aware of the many factors which must be balanced in the selections of expert panels (Hawkins and Graham, 1988), but is concerned about whether the judgments of such a limited group can reasonably be interpreted as representing a fair and balanced view of the current state of knowledge."

9B.3.5 Limitations in Pilot Elicitation Design

The pilot elicitation has afforded many opportunities for learning about expert elicitation in the context of economic benefits analysis. However, because this was an initial assessment that was limited in scope (as is discussed in section 9B.1), this section briefly discusses some of the limitations in the design of the pilot. Additional detail on the strengths and weaknesses of the pilot are provided in the technical report (IEc, 2004).

- Short time-period to design and conduct the elicitation - The scope of the pilot was limited in order to complete the assessment and present our findings as part of the Final Nonroad Diesel Rule. Thus, there was a one-year time period in which were

March 2004, page 34).

designed the elicitation, conducted the interviews, and provided an interpretation of the results in this RIA and the technical report (IEc, 2004). In addition to designing the elicitation with specific limitations as are discussed below, the experts we given short notice of the elicitation (some experts were interested but not available in our time frame), and we were required to process the results rapidly to meet the rulemaking schedule.

- The design and implementation of the elicitation has not undergone a complete external peer review. While EPA is planning to conduct a peer review of the elicitation process, we were not able to complete the review prior to the promulgation of the final rule. The results of the pilot should be viewed tentatively until the full peer review is complete.

- Small panel of experts - Due to resource constraints we limited the pilot to a panel of five experts. As noted above, the SAB-HES expressed their concern "about whether the judgements of such a limited group can reasonably be interpreted as representing a fair and balanced view of the current state of knowledge." They point to the many factors which must be balanced in the selection of expert panels (Hawkins and Graham, 1988) and there are numerous opinions among a large set of experts.

  Little analytical research has been conducted on the more difficult question of how to determine the ideal number of experts for a particular application. We have not found any analyses of the effect of expert panel size based on comparisons of empirical results of expert judgment studies. A theoretical analysis by Clemen and Winkler (1985) suggests that where data sources are moderately positively dependent there are diminishing marginal returns to the value of information associated with each additional data source. In the context of expert judgment studies, such a result implies that when dealing with experts of similar backgrounds who rely on the same models and studies, a larger expert panel may not provide significantly higher quality results than a smaller one. However, the addition of an expert expected to provide a more independent assessment, such as an expert from a different, but pertinent field, would be expected to exhibit a much greater value of information. Clemen and Winkler (1999) note that "heterogeneity among experts is highly desirable." These findings would appear to support addressing complex issues using a panel comprised of relatively small subgroups (perhaps three to five experts each) from multiple disciplines. Although the decision analysis field tends to use relatively small sample sizes (i.e., typically 5-10 experts), some are not comfortable with obtaining a combined distribution from such small numbers in the absence of an apriori assessment of the degree to which the expert panel is likely to be representative of the

overall population of relevant experts on the question of interest. The panel we used may not have captured the full range of reasonable opinions.

- Use of an aggregate elicitation question - The expert judgment literature discusses two broad approaches to elicitation of judgments; an aggregated and a disaggregated approach. As the term implies, an aggregated approach asks the expert to estimate the quantity of interest directly; for example, the numbers of newspapers sold in the U.S. in a particular year. In a disaggregated approach, the expert (or group of experts) would be asked to construct a model for estimating the quantity of interest and would be asked directly about the inputs to that model (e.g. population in each state, percentage of the population that reads newspapers, etc.) The intuition is that it is easier for experts to answer questions about the intermediate quantities than about the total quantity.

  The project team carefully considered the relative advantages and disadvantages of the two approaches. A major advantage of the disaggregated approach is a more structured and transparent characterization of the key inputs and sources of uncertainty in the final quantity of interest. However, the method does require additional time and resources to develop a model structure (or in some cases, multiple models) and set of inputs on which the experts can agree prior to the individual elicitations.

  The limited time frame available to complete this assessment drove the decision to undertake an aggregate approach to elicit the C-R coefficient for the $PM_{2.5}$/mortality relationship.[rr] Nonetheless, a major goal of the preliminary and follow-up questions in the protocol was to identify critical issues that could be addressed through the development of a more disaggregated approach in a future assessment.

  Thus, the design of the pilot limits our ability to determine the influence of any one key factor over others in a large list of issues that the experts were to consider prior to answering the quantitative question. It also limited the ability of the experts to express their views about the difference in the C-R function based on the location in the U.S. (i.e., the demographics of the exposed population, the air concentration of PM and/or PM mixture).

[RR] While the Project Team initially considered using a highly aggregated approach that would have asked experts to characterize a single overall PM / mortality effect due to both short- and long-term exposures to PM2.5. However, based on advice from the SAB-HES, we opted to disaggregate effects due to long- and short-term exposures. The Project Team felt that separate questions to address effects of long- and short-term exposures, though still at a high level of aggregation, would prove to be easier for experts to address than a question that "rolled up" all the effects into a single estimate. This level of disaggregation also enabled the elicitation team to explore with experts possible overlap in reported mortality effects detected using long-term and short-term epidemiological studies.

- No workshop was conducted - It is customary to conduct a workshop prior to the elicitation interview with the experts. This allows the experts to become familiar with the protocol, the background materials contained in the briefing book, and to discuss methods to limit bias during the interview. Due to time constraints for the pilot, we did not conduct a pre-elicitation workshop.

- No calibration of experts - We do not have calibration measures that could be used to assess the results of this pilot. At this point, we can only assess the process – did the pilot assessment employ a structure, supporting materials, and a process that enabled experts to make judgments that would be likely to be well calibrated? The peer review for this aspect is still underway. Nevertheless, without calibration measures, we cannot weight experts based on their performance on calibration tasks.

- Full-day elicitation - The elicitation interview with each expert took a full-day to complete. Again, experts were given short notice of the elicitation and found time in their schedules to participate, yet not all of the experts were available for the full-day interview. The length of the interview could lead to response fatigue that could affect the outcome of the experts' response.

9B.3.6 Combining the Expert Judgments for Application to Economic Benefit Analyses

Analysts must give careful thought to whether and how to combine the results individual expert judgments into a single distribution. When dealing with a small sample number of experts, the analyst must be particularly careful to identify the influence of each expert's response on the combined distribution. Therefore, we considered four alternative methods for combining the pilot results. However, the Project Team identified significant issues associated with each of the methods. In this section, we discuss the issues we considered in combining the results of the pilot and how we came to the conclusion that for the illustrative benefits analysis presented in Section 9B.5 below, we would present both the individual quantitative distributions of the C-R coefficient elicited from the five experts interviewed as well as results based on a probabilistic estimate that represents the combined results of the pilot based on an equal weighting of the calculated change in mortality incidence based on the individual judgments.

9B.3.6.1 Background

Combination of expert judgments is not strictly necessary; some investigators (e.g., Hawkins and Graham, 1990; Winkler and Wallsten, 1995; and Morgan et al., 1984) have preferred to keep expert opinions separate in order to preserve the diversity of opinion on the

issues of interest. In such situations, the range of values expressed by the experts can help decision-makers to understand the sensitivity of their analyses to the analytical model chosen, thereby bounding possible outcomes. Individual judgments can also illustrate varying opinions arising from different disciplinary perspectives or from the rational selection of alternative theoretical models or data sets (Morgan and Henrion, 1990). Nonetheless, analysts are often interested in developing a single distribution of values that reflects a synthesis of the judgments elicited from a group of experts.

There are also some advantages to combining the results across experts. An extensive literature exists concerning methods for combining expert judgments. These methods can be broadly classified as either mathematical or behavioral (Clemen and Winkler, 1999). Mathematical approaches range from simple averaging of responses to much more complex models incorporating information about the quality of expert responses, potential dependence among expert judgments, or (in the case of Bayesian methods) prior probability distributions about the variable of interest. Behavioral approaches require the interaction of experts in an effort to encourage them to achieve consensus, either through face-to-face meetings or through the exchange of information about judgments among experts. As noted in the technical report (IEc, 2004) , there are both methodological and practical issues arguing against a behavioral approach. Therefore, we used a mathematical combination process to derive a single distribution.

One advantage of mathematical combination over behavioral approaches is the ability to be completely transparent about how weights have been assigned to the judgments of specific experts and about what assumptions have been made concerning the degree of correlation between experts. Several approaches can be used to assign weights to individual experts. Weights can be assigned based on the analyst's opinion of the relative expertise of each expert; on a quantitative assessment of the calibration and informativeness (i.e., precision) of each expert based on their responses to a set of calibration questions (as described in Cooke, 1991); or on weights assigned by each expert, either to him or herself or to the other experts on the panel (see Evans et al., 1994 for an example of this approach). Ideally, such a weighting system would address problems of uneven calibration and informativeness across experts, as well as potential motivational biases (Cooke, 1991).[a] In practice, appropriate weights can be difficult to determine, though Cooke and others have conducted considerable research on this issue.

At the design stages of the pilot, we decided that the resulting expert judgments would be combined using equal weights, essentially calculating the arithmetic mean of the expert responses, for simplicity and transparency. The reasons for choosing equal weights were both practical and

[A] "Motivational bias" refers to the willful distortion of an expert's true judgments. The origins of this bias can vary, but could include, for example, a reluctance to contradict views expressed by one's employer or a deliberate attempt to skew the outcome of the study for political gain.

methodological. Development of defensible differential weights was not possible given the expedited schedule for this project. Although we did conduct a sample calibration exercise with each expert, the purpose of the exercise was to train the experts in providing quantitative responses, not to develop calibration scores that would be used to weight experts. Some empirical evidence suggests that the simple combination rules, like equal weighting, perform equally well when compared to more complex methods in terms of calibration scores for the combined results (Clemen and Winkler, 1999). The methods to combine the expert judgments will be explicitly addressed during the peer review of the pilot assessment.

9B.3.6.2 Alternative Combination Methods

While a combination method using equal weights for the results of each expert is straightforward in principle, applying it in this context of the results of the pilot was complicated by the fact that the elicitation protocol gave the experts freedom to specify different forms for the C-R function. If all the experts had chosen the same form of the C-R function,(e.g., if each expert had specified a log-linear C-R function with a constant, but uncertain, C-R coefficient (i.e., slope) over the PM range specified in the protocol) the combination of their distributions for the C-R coefficient would require a simple averaging across experts at each elicited percentile. However, in this assessment, three experts specified log-linear functions with constant C-R coefficients over the specified range of $PM_{2.5}$ concentrations, and two of the experts specified the C-R coefficient as likely to vary over the range of specified $PM_{2.5}$ concentrations (as discussed in Section 9B.4.2 above). These more complex C-R functions necessitated some additional steps in the calculation of the combined results.

As discussed in the technical report for the pilot (IEc, 2004), individual response either can be combined before application of the benefits model or during the application of the model, allowing each expert's C-R function to be estimated in the benefits model independently. Specifically, we derive the total mortality incidence for each expert, and combine (or pool) the estimates into an aggregate value before taking an average of the mortality incidence. This is referred to as a "pooled" approach and is used in our modeling framework for other benefit endpoints that have multiple C-R function (due to multiple studies). We prefer the pooled approach because it seems to reduce the amount of alteration of the actual step-function responses provided by Experts B and C (although some adjustments must still be made)[b]. Details of the illustration are provided in Section 9B.6.

[B] Expert B specified a distribution for the C-R coefficient for $PM_{2.5}$ concentrations above a threshold and assigned the coefficient a value of zero for all PM concentrations below the threshold. He then specified a probability distribution to describe the uncertainty about the threshold value. Expert C specified separate distributions for the C-R coefficient at four discrete points within the concentration ranges defined in the protocol, to represent a continuous C-R function whose slope varied with the $PM_{2.5}$ concentration. Expert C indicated that the coefficient value between these points was best modeled as a continuous function, rather than a step function. Both experts assumed the same functional forms in responding to elicitation question.

The alternative would be to combine the individual expert judgments into a single C-R function before applying the results to the benefits model. Below, we present three approaches we considered for combining the expert judgments before applying the benefits model. Among the three approaches to combining expert judgments before the benefits analysis, the primary difference is how they account for the underlying particulate air pollution levels. The first option assumes a uniform distribution and equal weighting, which involves taking a simple average of responses across experts for each percentile. In a second combination method, we combined the results using a normal distribution describing population-weighted annual average $PM_{2.5}$ concentration data generated from EPA's Environmental Benefits and Mapping Analysis Program (BenMAP), the model EPA currently uses for economic benefit analyses of air quality regulations affecting PM and other criteria pollutants.[c]

As discussed above, for the two of the experts that specified a C-R function that varied over the range of PM concentrations., their estimated C-R function necessitated some additional steps in the calculation of the combined results. To derive a single distribution across all experts for a particular range of exposures (e.g. 8-20 $\mu g/m^3$ annual average $PM_{2.5}$), we first needed to estimate an "effective" distribution of uncertainty about the C-R coefficient for both Experts B and C across that range by using Monte Carlo simulation (Crystal Ball® software) to estimate the expected value of each percentile elicited across the full $PM_{2.5}$ range specified. Specifically, the additional steps we took for this combination method are as follows:

- Expert B specified a distribution for the C-R coefficient fo rPM2.5 concentrations above a stated threshold and assigned the coefficient a value of zero for all PM concentrations below the threshold. He then specified a probability distribution to describe the uncertainty about the threshold value. Thus, we conducted Monte Carlo sampling using two distributions: his uncertainty distribution for the threshold, and an assumed distribution of baseline $PM_{2.5}$ concentrations for the $PM_{2.5}$ range specified in the elicitation protocol. On each iteration, we selected a value from each of these two distributions and compared them. If the selected baseline concentration was less than or equal to the selected threshold value, each of the percentiles of Expert B's uncertainty distribution was assigned a zero value (no mortality effect); if the concentration was greater than the threshold, we assigned each percentile the "above-the-threshold" value specified by Expert B in his interview.[d] We repeated this

[C] To facilitate Monte Carlo sampling, we evaluated the fit of the BENMAP data to several distributional forms, ultimately selecting a normal distribution, truncated at zero, with a mean of 11.04 $\mu g/m^3$ and a standard deviation of 2.32 $\mu g/m^3$.

[D] An example for mortality effects from long-term exposures helps illustrate this approach. Expert B estimated that he was 75 percent sure (i.e., his 75th percentile) that the percent increase in mortality would be less than or equal to 0.5 percent per 1 $\mu g/m^3$ change in $PM_{2.5}$ concentration if the baseline concentration were above the threshold, but zero

process for thousands of iterations and then took the average value for each of the percentiles to obtain Expert B's "effective" distribution of uncertainty about the C-R coefficient across each range of exposures.

- Expert C specified separate distributions for the C-R coefficient at four discrete points within the concentration ranges defined in the protocol, to represent an continuous function whose slope varied with the PM concentration. Thus, we first randomly sampled from the assumed distribution of baseline PM concentrations. We then linearly interpolated between Expert C's responses at the two points nearest to the sampled PM concentration, to estimate his uncertainty distribution for the C-R coefficient at the sampled concentration. For example, Expert C provided slope values at $PM_{2.5}$ concentrations of 8, 10, 15 and 20 for mortality effects of long-term exposure. If, on a given iteration we selected a $PM_{2.5}$ concentration of 12 $\mu g/m^3$, we would generate a slope at each percentile of his uncertainty distribution by interpolating between Expert C's responses at 10 and 15 $\mu g/m^3$. We repeated this process for thousands of iterations and then took the average value for each of the percentiles to obtain the "effective" distribution of the average slope of Expert C's C-R function.

While the uniform distribution is the simplest method of combining the expert judgments, it required us to alter the true responses of Experts B and C. It is also based on a uniform distribution, which does not match the observed $PM_{2.5}$ concentrations that tend to be skewed toward the lower concentration values. The estimates of Expert B and C's "effective" distributions, and thus the combined expert distribution, are all sensitive to the probability density function chosen to describe the U.S. baseline $PM_{2.5}$ concentrations in the simulations. This sensitivity arises because both Experts B and C assume that the effect of an increase in $PM_{2.5}$ concentration on mortality depends on the initial $PM_{2.5}$ concentration. Table 9B-3 presents the resulting values of the distribution for these two methods of combining the results of the pilot.

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percent if it were below the threshold. If on a given iteration, the program selects a baseline concentration of 12 $\mu g/m^3$ and a threshold level of 10 $\mu g/m^3$, we assign his 75th percentile the value of 0.5. If the threshold level selected were 15 $\mu g/m^3$, the 75th percentile would be assigned a value of zero.

Table 9B-3. Methods for Combining Expert Judgments: Combined C-R function with Uniform Distribution and a Population-Weighted Distribution

Percentiles	Combined Expert Judgments using a Uniform Distribution of Baseline Annual Mean $PM_{2.5}$ Concentrations	Combined Expert Judgments Based on Population-Weighted Distribution of Baseline Annual Mean $PM_{2.5}$ Concentrations in U.S.
95th %ile	1.05	0.93
75th %ile	0.65	0.59
50th %ile	0.33	0.3
25th %ile	0.17	0.16
5th %ile	0.00	0
Minimum	0.00	0
Maximum	1.71	1.5

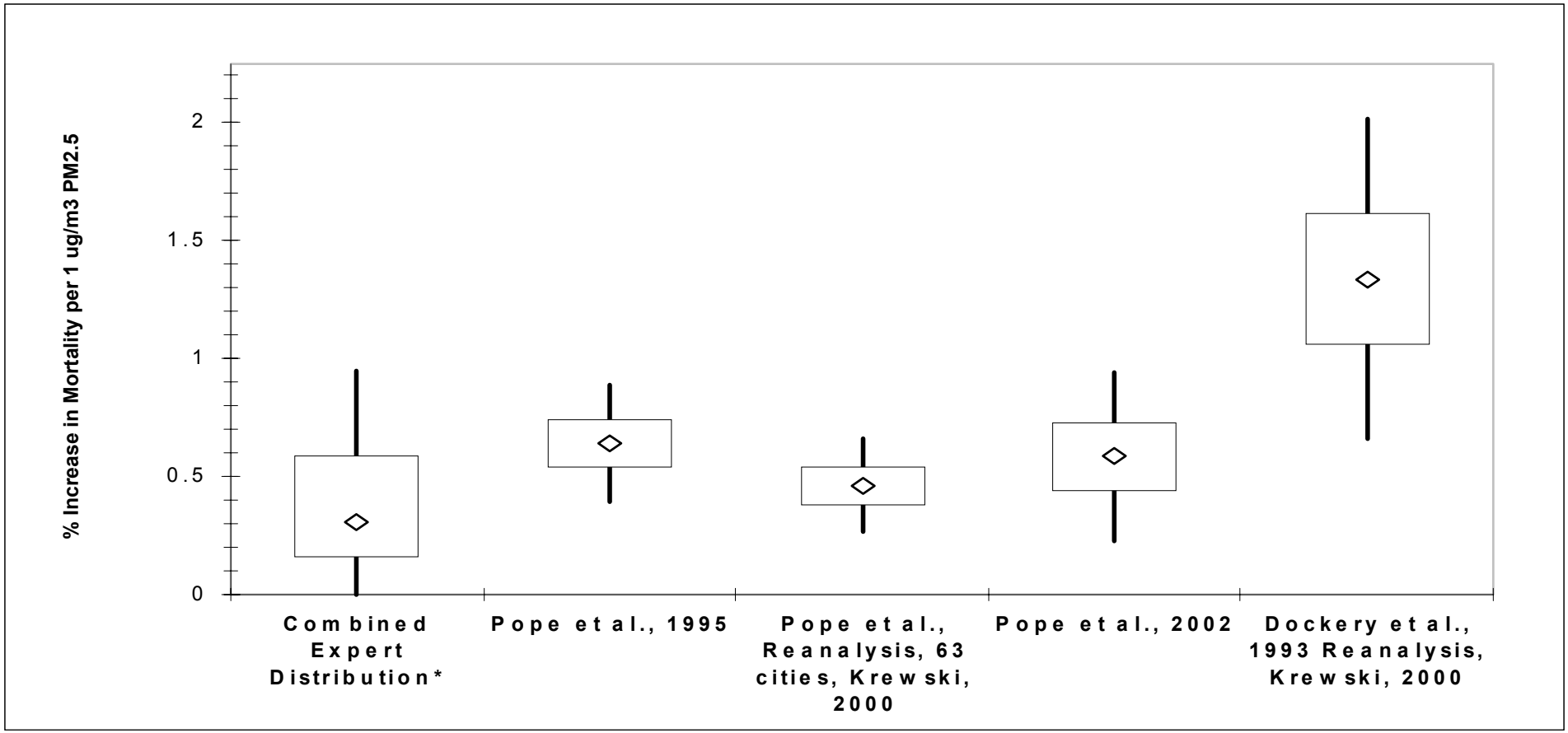
Given the differences in the responses given by Experts B and C at various levels of PM concentrations (i.e., a conditional C-R function), we considered a third combination method in which we calculate combined expert distributions at four different $PM_{2.5}$ baseline concentrations. Using the methods described above, we first calculated Expert B's and C's distributions at the four concentration points and then averaged them with the distributions of the other three experts (which remain constant over the concentration range) using equal weights. This method reduces the level of adjustments that are made to Expert B's and C's response function in that we estimate four C-R function for each individual, rather than one smoothed function. The functions for the three other experts remain log-linear. Results of this combination method are provided in Table 9B-4.

Table 9B-4. Combined Concentration-Response Function Conditional to PM Concentrations

Percentiles	8 ug/m3	12 ug/m3	15 ug/m3	20 ug/m3
95th percentile	0.82	0.99	1.08	1.20
75th percentile	0.56	0.61	0.64	0.76
50th percentile	**0.30**	**0.30**	**0.30**	**0.42**
25th percentile	0.16	0.16	0.16	0.24
5th percentile	0	0	0	0

Overall, the combination methods considered result in fairly similar results at the median and mean relative risk estimate. However, slight differences occur in the tails of the distribution in their characterization of uncertainty. In figure 9B-2, the C-R function for the population-weighted combination method was compared to the existing cohort epidemiological studies of the long-term $PM_{2.5}$/mortality relationship. We observe that the results of the pilot elicitation are generally within the range of findings from these epidemiological studies. However, as expected, the elicitation results in a larger spread of uncertainty than is given by the standard errors of the individual studies.

Figure 9B-2. Comparison of Combined Expert Judgment Distribution to Selected Published Studies



9B.3.5 Limitations of Combining Expert Judgments

Although we present several methods for combining the results of the pilot, there are several limitations in interpreting the pilot results that should be considered.

- The conditional functions of Experts B and C required us to estimate some values on the C-R function between the points that were elicited, which requires an extrapolation from the response provided in the pilot to create continuous distributions.

- There are many methods available to combine the responses from the experts. Each method has advantages and disadvantages from a statistical viewpoint. The project team is not aware of any rule-of-thumb in statistics that would provide guidance for combining linear and non-linear functions. Therefore, we present four alternative methods for combining the results as an illustration of potential combinations of the results, and have asked for a peer review of these methods.

- In designing the pilot, there was a decision to combine the results of the individual experts using an equal weighting. In some elicitation studies, the authors use a calibration measure to weight the experts appropriately. Because we did not conduct a calibration exercise, we present only an equal weighting of the responses.

- We have used a normal distribution to characterize the pilot results, but the distribution could potentially be skewed due to the bounding at zero. The C-R functions are bounded by zero, and anchored to one data source. There is a concern that the upper-end of the distribution resulting from the pilot may not fully reflect the available data and knowledge on the PM/mortality relationship. There may have been some anchoring to the study results from the ACS cohort, and less use of the Six-Cities study in the characterization of uncertainty upper-bounds. However, the experts were provided the Six Cities results in their briefing books as background material.

9B.4. Illustrative Application of Pilot Expert Elicitation Results

In this section, we apply the pilot expert elicitation results, using the pooled approach discussed above for combining results across participants to the VSL distribution discussed in Chapter 9 (section 9.3.4), thereby providing an illustrative example of how one might translate the results from the pilot elicitation into quantified estimates of economic benefits. The analysis is based on the modeled air quality changes conducted for the preliminary nonroad diesel control option in 2030. As such, the results are comparable to the point estimates provided in Appendix

9A, but not to those in Chapter 9. The values generated below do not reflect the Agency's estimates of the benefits of the emissions reductions expected from the Final Non-Road Diesel rule and are included solely as an illustration of the impacts of using expert elicitation based distributions for premature mortality associated with long-term exposure to $PM_{2.5}$ rather than a data-derived distribution.

9B.5.1 Method

9B.5.1.1 Concentration-Response Distribution Based on Combined Results Across Experts

As discussed in Section 9B.4.5, we converted each expert's percentile responses about mortality associated with long-term exposure into a custom distribution such that each percentile is correctly represented and percentiles in between are represented as continuous functions (custom distributions were generated using Crystal Ball and are represented as 15,000 equally probable points).

For experts A, D, and E, we used a standard log-linear functional form:

$$\Delta y = y_0 \cdot (e^{\beta \cdot \Delta x} - 1), \qquad (4)$$

where we set β equal to ln(1+B/100), where B is the percent change in all cause mortality associated with a one μg reduction in $PM_{2.5}$. BenMAP then represents the distribution of Δy based on the custom distribution of β.

Expert C provided a set of conditional C-R functions for different baseline levels of $PM_{2.5}$. Expert C provided four conditional responses, one for 8 μg/m3, one for 10 μg/m3, one for 15 μg/m3, and one for 20 μg/m3. In order to "fill-in" the C-R function for intermediate baseline $PM_{2.5}$ values, we linearly interpolated between the responses for each pair of points, e.g. 10 to 15 or 15 to 20. We calculated interpolated values for 13 points, ranging from 8 μg to 20 μg. For baseline values less than 8 μg, we assigned a value of zero (essentially assuming a threshold at 8 μg). For baseline values greater than 20, we assigned the values provided by Expert C for 20 μg. This may result in an underestimate of the incidence of mortality for Expert C. For each of the conditional functions, we used a log-linear specification, similar to A, D, and E. Total incidence of mortality for Expert C is the sum of the conditional estimates over the range of baseline air concentrations.

Expert B provided a log-linear C-R function, conditional on an unknown threshold characterized by a triangular distribution bounded by 4 μg and 15 μg, with a mode at 12 μg. We discretized the triangular distribution into 12 ranges of unit length (e.g. 4 to 5, 5 to 6, etc.) and

calculated the expected value of the response at each population gridcell based on the observed baseline $PM_{2.5}$ and the probability of that baseline value exceeding the potential threshold. We assume that if a grid cell has a baseline value above the threshold, then the full value of the reduction in $PM_{2.5}$ at that grid cell is associated with a reduction in mortality. This may result in an overestimate of the mortality impact for Expert B because for grid cells where the baseline level is only marginally above the threshold, a benefit might only accrue to the change in $PM_{2.5}$ down to the threshold. The rest of the change would not result in any mortality reduction. Because most of the changes in air quality are relatively small (population weighted change in annual mean $PM_{2.5}$ is -0.59 μg), this should not be a large issue.

To put these estimates in perspective, it is useful to summarize the projected baseline (pre-nonroad diesel regulations) air quality in 2030. Table 9B-5 lists the population distribution of baseline concentrations of $PM_{2.5}$ in 2030:

Table 9B-5. Population Distribution of Baseline Ambient $PM_{2.5}$

Baseline $PM_{2.5}$ ($\mu g/m^3$)	2030 Population (millions)	Percent of Total 2030 Population
$PM_{2.5}<5$	3.5	1.0%
$5 \le PM_{2.5}<10$	68.8	19.5%
$10 \le PM_{2.5}<15$	198.1	56.2%
$15 \le PM_{2.5}<20$	66.1	18.8%
$20 \le PM_{2.5}<25$	12.1	3.4%
$25 \le PM_{2.5}<30$	4	1.1%

9B.5.1.2 Estimated Reduction in Premature Morality and Valuation

Based on the air quality modeling conducted for the Nonroad Diesel preliminary control option, we calculated the reduction in incidence of premature mortality associated with $PM_{2.5}$ and the value of that reduction. We used Monte Carlo simulations to derive the distributions of the dollar values of estimated reductions in premature mortality. For each expert, the Monte Carlo simulation generates a dollar value by randomly sampling from the distribution of the reduction in mortality incidence and the distribution of VSL (normally distributed with a mean of $5.5 and a 95 percent confidence interval between $1 and $10 million) and multiplying the values together. This yields an estimate of the dollar value of the mortality reductions. This process is repeated 5,000 times to generate a distribution of dollar

values. The Monte Carlo process was conducted using the estimated distribution for each expert individually and for the combined (pooled) distribution, as well as for the distribution derived from the Pope et al. (2002) study.

9B.5.2 Results

Figure 9B-4 presents box plots that display the distribution of the reduction in $PM_{2.5}$ related premature mortality based on the concentration response distributions provided by each expert, as well as that based on the pooled response.[a] For comparison, the figure also displays the distribution derived from the statistical error associated with Pope et al (2002). The figure shows that the average annual number of premature deaths avoided for the "modeled preliminarily control option" ranges from approximately 4000 to 19,000, depending on the concentration response function used. The medians span zero to 16,000, with the zero value due to the low threshold associated with one of the expert's distributions. Specifically, because less than a quarter of the population is expected to live in areas with PM2.5 levels above the threshold specified by expert C, and much of the decrease in PM2.5 predicted by the preliminary control option occurs below that threshold, a much smaller decrease in premature morality is predicted for expert C than those experts who provided continuos C-R functions down to zero (PM2.5) as well as for expert B who provided an uncertain threshold. Furthermore, note that at the 50th and 75th percentiles, the C-R functions provided by all of the experts predict positive benefits from the modeled control option.

The boxplots displayed in Figure 9B-4 are derived by applying the C-R distributions specified by each expert (as presented in Figure 9B-1) to the change in air quality predicted by the preliminary non-road diesel control option. Although the figures 9B-3 and 9B-1 show similar patterns, there are important differences. Specifically, the ratio of 75th percentiles of the C-R functions specified by experts A and B (as denoted in Figure 9B-1) is 0.4, whereas the ratio of the predicted change in incidence of premature mortality associated with the modeled preliminary control option is 0.5. This 25% increase in the ratio suggests a larger effective difference in the distributions between the experts than was evident before applying the expert's C-R functions to a predicted change in air quality and highlights the impact of the air quality change predicted on the choice of C-R function used in the benefits analysis.

The combined expert distribution depicted in Figure 9B-4 provides additional insights. The combined (average) distribution has a 90 percent credible interval between zero and 24,000. When compared with results derived from the Pope et al. (2002) study, it is clear that the combined expert distribution reflects greater uncertainty about the estimated reduction in

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[A] As discussed above, the elicitation results were combined assuming equal weight for each expert's distribution. We assumed complete dependence of the expert's distributions for this illustrative analysis, so that each percentile of the pooled distribution is simply the average of the corresponding percentiles of the 5 experts.

premature mortality, as well as placing more weight on the lower end of the distribution. The mean estimate from the combined expert distribution is almost 30 percent lower than the mean derived from the Pope et al. (2002) distribution. However, the 90 percent confidence interval based on the standard error from Pope et al. (2002) is completely contained within the 90 percent credible interval of the combined expert distribution.

Figure 9B-5 shows the same data using cumulative distribution functions (CDFs). This figure is valuable for demonstrating differences in degree of certainty in achieving specific reductions in premature mortality. For instance, the Pope et al. 2002 concentration response distribution predicts a 20% chance that there will be at least 10,000 fewer premature deaths, whereas the pooled distribution predicts a 60% chance of the same reduction in premature deaths. The probabilities associated with the individual experts for avoiding 10,000 premature deaths range from about 28% to 98%, demonstrating once again the sensitivity of the estimate to assumptions regarding the concentration response function. The CDFs of the estimated reductions in premature mortality shows that for several experts, there is a small probability of a substantially higher estimate. For example, the 75th percentile of the distribution based on Expert B's responses is at 8,800, while the 99th percentile for that distribution is almost 4 times higher, at 34,000. The CDF also shows that while most of the experts provided fairly wide distributions, reflecting a lack of confidence in the precision of the empirical data, the CDF based on Expert C's responses is much narrower, reflecting the high degree of confidence he placed on the existence of a threshold below 15 μg.

Figures 9B-6 and 9B-7 use box plots and CDFs to display the estimated dollar value of these annual reductions in premature mortality. Whereas the average based on the Pope et al 2002 distribution is $94 billion, the average based on the pooled estimate is $67 billion, a difference of approximately one-third. Once the concentration response distributions are combined with the VSL distributions, not only are the mean values closer to one another, but the distributions show considerably more overlap.

Because these distributions are the result of a Monte Carlo simulation combining the non-normal distributions for reductions in mortality with a normal distribution for VSL, the resulting distributions will also be non-normal, but the shape depends on the skewness of the input distribution of mortality reductions. For example, the ratio of the 95th to 75th percentile of mortality reductions for Expert B is 3.1, while the same ratio for the value of mortality reductions is 4.2, indicating the value distribution is more skewed than the reductions distribution. In general, combining normal or left skewed distributions in a mulitplicative fashion will result in left skewed distributions with greater skewness than the input distributions. So even for the normally distributed estimates based on Pope et al. (2002), the value distribution is somewhat skewed, because it is the result of multiplying two normally distributed random variables.

The shapes of the two distributions are more similar in this case because both reflect the same additional information in the VSL distribution. This demonstrates that as additional sources of uncertainty are added to the analysis, the influence of any one source of uncertainty will fall. Because VSL is a large source of uncertainty, the influence on overall uncertainty relative to the distribution of the mortality reduction is also large. All of the distributions of the value of mortality reductions have a small negative tail, this time due to propagation of the normally distributed VSL, which has a small amount of the distribution below zero. Again, we interpret this as a statistical artifact rather than a true probability that the value of a statistical life is negative (implying that individuals would pay to increase the risk of death).

We used additional Monte Carlo simulations to combine the expert-based distributions for the dollar benefits of mortality with the distributions of dollar benefits for the remaining health and welfare endpoints to derive estimates of the overall distribution of total dollar benefits[b]. The box plots for these distributions of overall dollar benefits associated with the modeled nonroad diesel preliminary control options are presented in Figure B-8. Because mortality accounts for over 90 percent of the benefits, the addition of other endpoints has little impact on the overall distributions. The overall mean annual total dollar benefits in 2030 for the distribution incorporating the combined expert distribution for reductions in premature mortality is $70 billion, compared to $96 billion for the results derived from the Pope et al. (2002) study for the nonroad diesel modeled preliminary control option.

For clarity of presentation, in Figure 9B-9, we present CDFs for total dollar benefits only for the combined expert distribution and results derived from the Pope et al. (2002) study. These again suggest that the use of the expert elicitation based representation of uncertainty in the relationship between $PM_{2.5}$ and premature mortality has a large impact on the shape and range of the distribution of total benefits. The Pope et al. (2002) derived results have an approximately Weibull shaped distribution with a range from 5th to 95th percentiles of $23 billion to $190 billion, or about one order of magnitude. The distribution of total dollar benefits incorporating the combined expert distribution for reductions in premature mortality has a much more skewed shape with an elongated positive tail above the 75th percentile with a range from 5th to 95th percentiles of $3 billion to $240 billion, or about two orders of magnitude.

[B]Note that visibility benefits are treated as fixed for this illustrative analysis. We are working on methods to characterize the uncertainty in visibility and other non-health benefits.

Figure 9B-4 Results of Illustrative Application of Pilot Expert Elicitation: Annual Reductions in Premature Mortality in 2030 Associated with the Modeled Preliminary Control Option for the Nonroad Diesel Rule

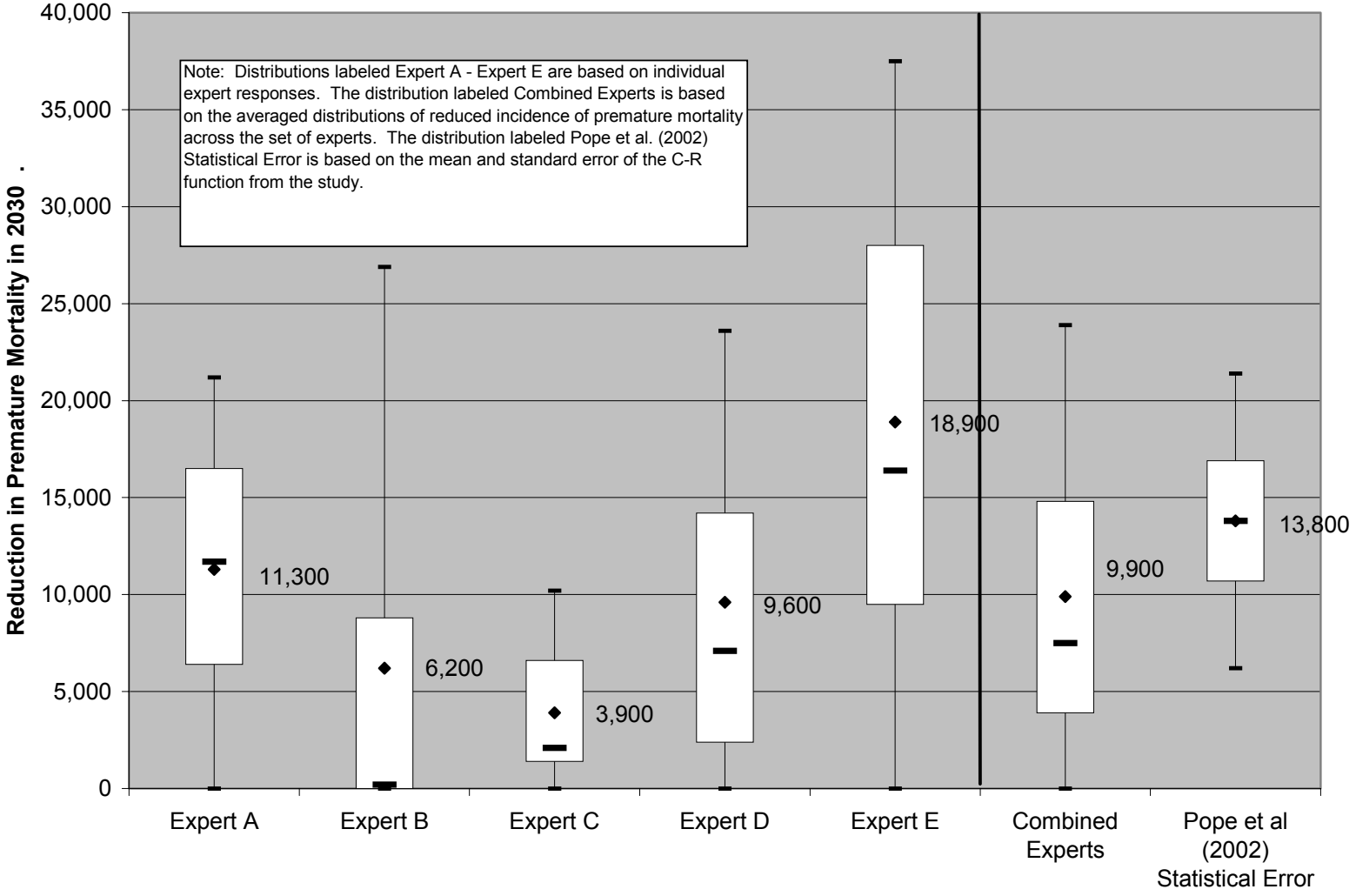


Figure 9B-5. Cumulative Distribution Functions for Annual Reductions in Premature Mortality in 2030 Associated with the Nonroad Diesel Modeled Preliminary Control Option

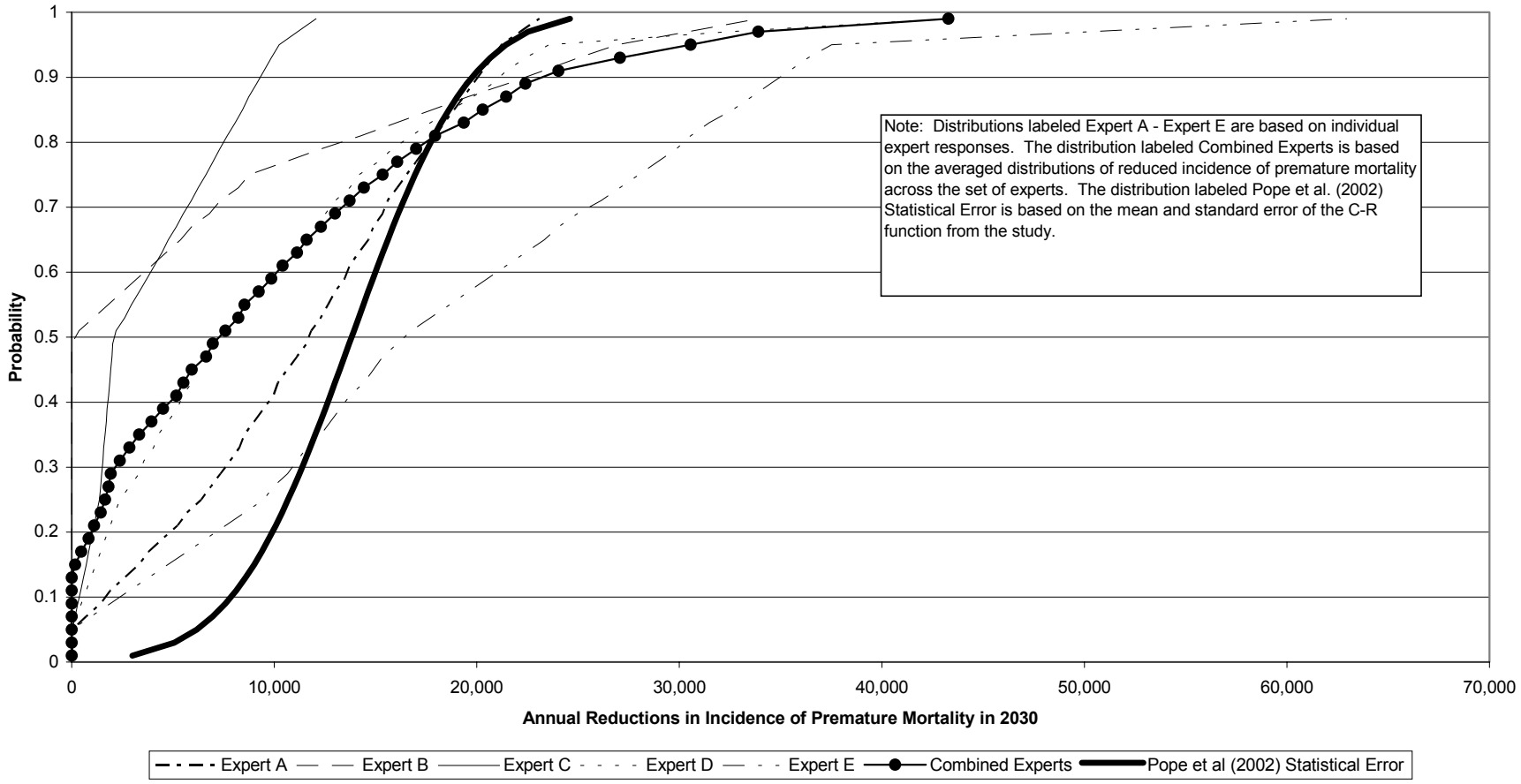


Figure 9B-6. Results of Illustrative Application of Pilot Expert Elicitation: Dollar Value of Annual Reductions in Premature Mortality in 2030 Associated with the Modeled Preliminary Control Option for the Nonroad Diesel Rule

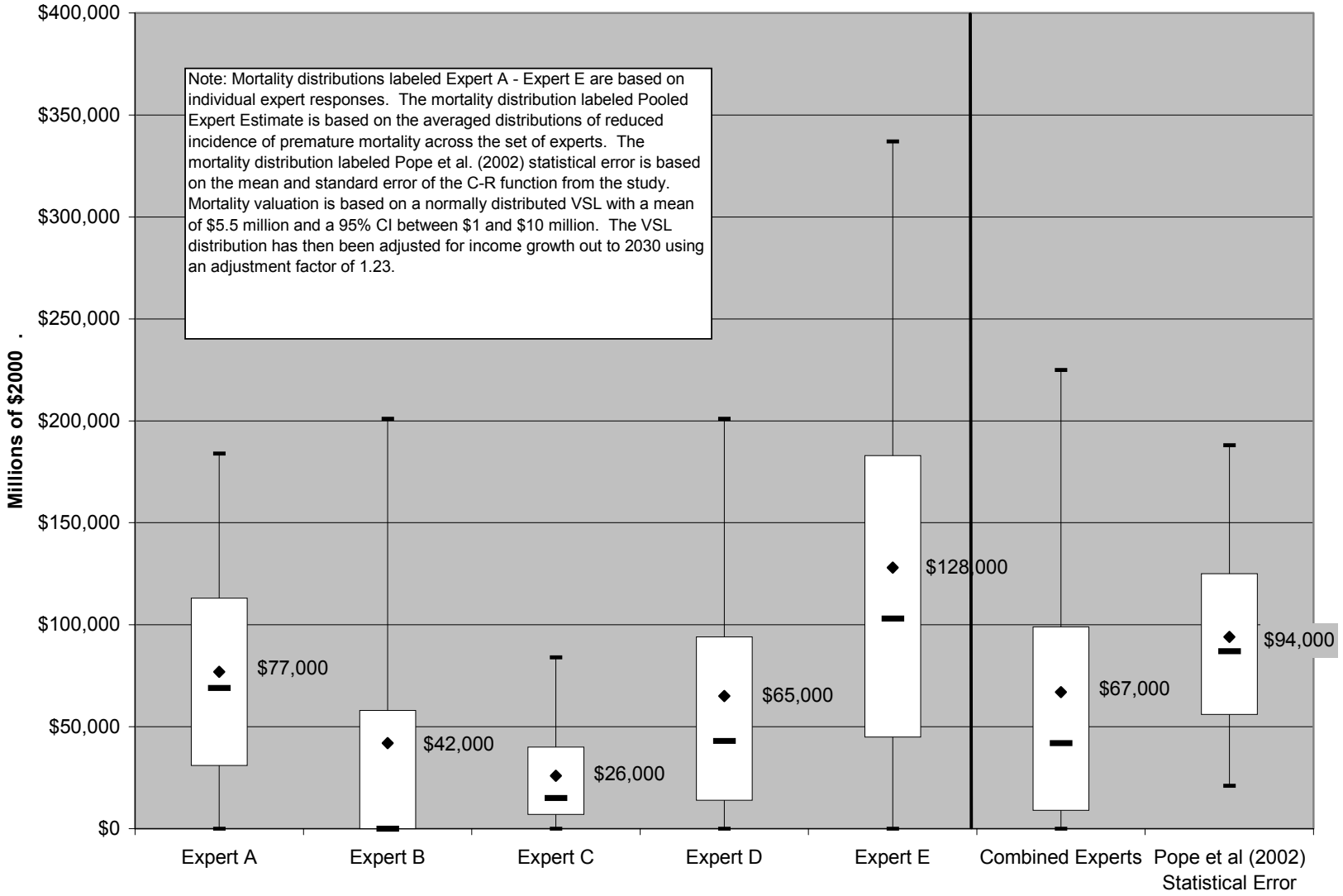


Figure 9B-7. Cumulative Distribution Functions for Dollar Value of Annual Reductions in Premature Mortality in 2030 Associated with the Nonroad Diesel Modeled Preliminary Control Option

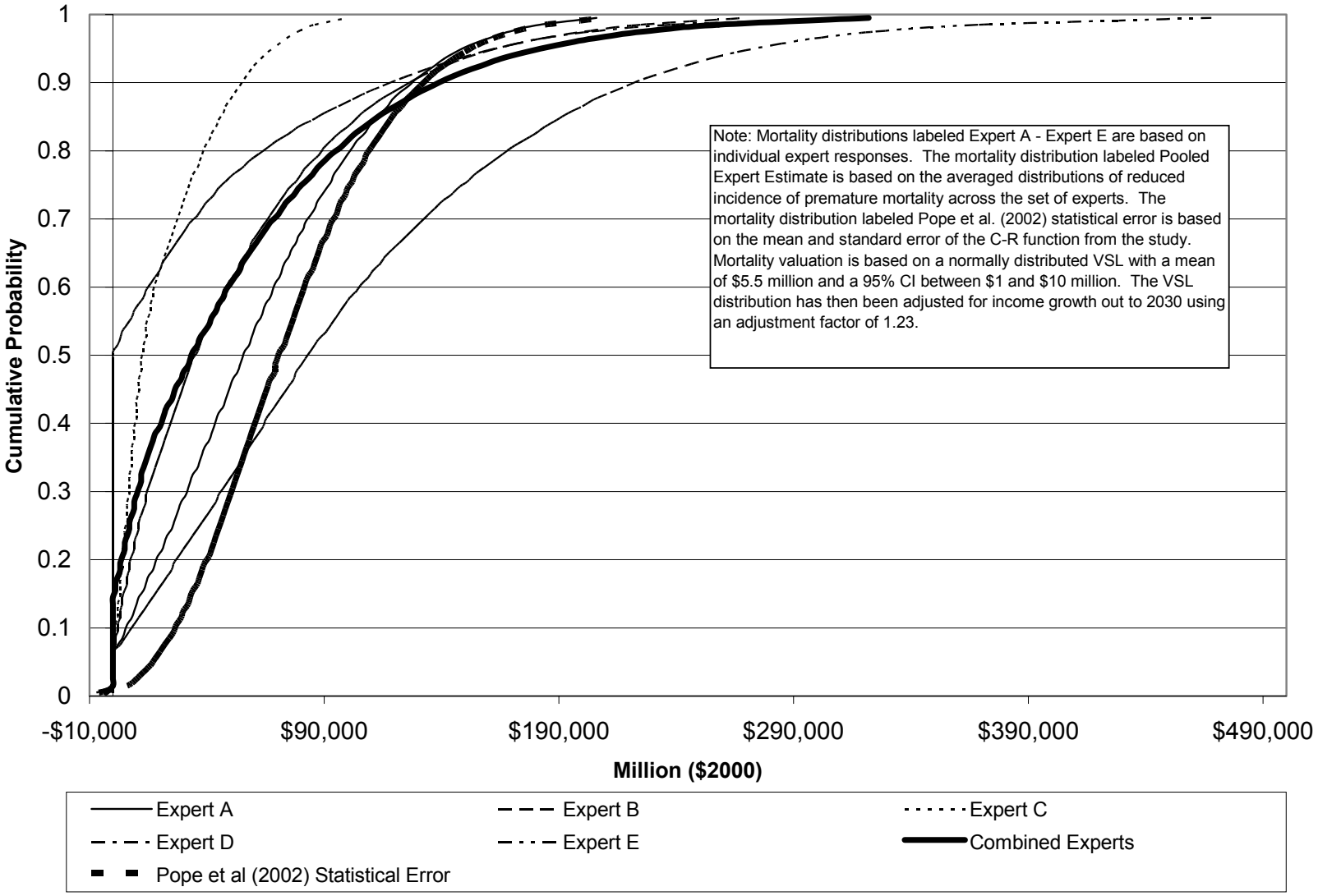


Figure 9B-8. Results of Illustrative Application of Pilot Expert Elicitation: Dollar Value of Total Annual PM-related Health and Visibility Benefits in 2030 Associated with the Modeled Preliminary Control Option for the Tier 4 Rule

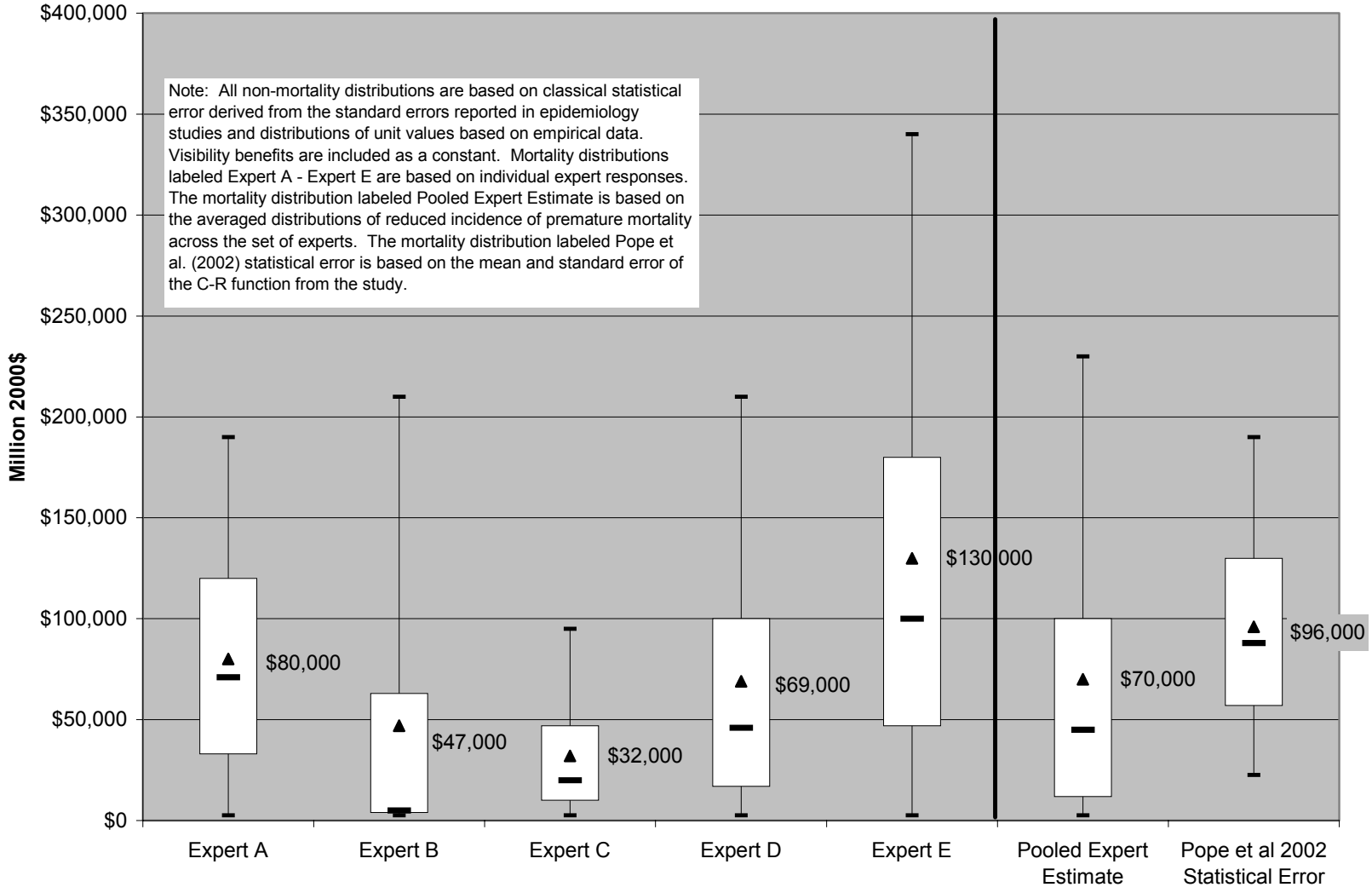
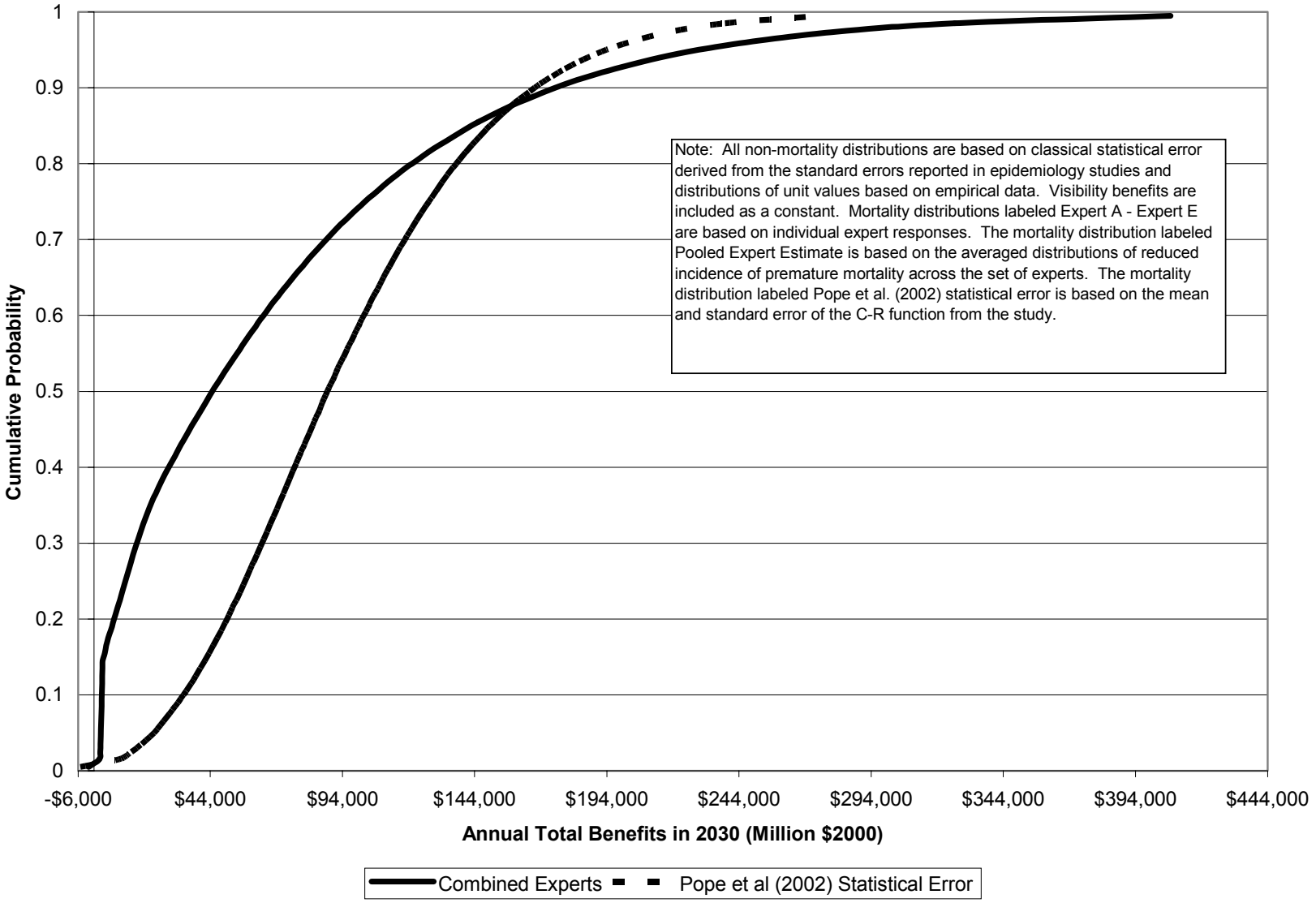


Figure 9B-9. Cumulative Distribution Functions of Dollar Value of Total Annual PM-related Health and Visibility Benefits in 2030 Associated with the Nonroad Diesel Modeled Preliminary Control Option



9B.5.3 Limitations of the Application of the Pilot Elicitation Results to the Nonroad Scenario

The results presented in this section should be viewed cautiously given the limited scope of the pilot, and the limitations of the elicitation design and methods used to combine the expert judgments discussed above. Therefore, the results presented above should be considered "illustrative" until both the peer review of the pilot is complete and the methods used to interpret and apply the results of the pilot have been peer-reviewed and accepted. Until this occurs, we do not recommend applying this method in other regulatory analyses.

Specific limitations of the illustrative application include:

- Extrapolation of percentile responses provided by individual experts. Each expert provided minimum and maximum values, as well as the 5th, 25th, 50th, 75th, and 95th percentiles. In order to generate the continuous distributions of mortality impacts, we had to make assumptions about the continuity of the distributions between the reported percentiles. This adds uncertainty to the results.

- Interpolation of C-R relationship across $PM_{2.5}$ levels. Expert C provided a set of conditional distributions of the C-R relationship conditioned on the baseline level of $PM_{2.5}$. Because he only provided functions for a limited number of baseline levels, we had to interpolate the values between levels, introducing additional uncertainty. In addition, Expert C provided no information on the C-R function for baseline $PM_{2.5}$ levels below 8 μg/m3 or above 20 μg/m3. We assumed no mortality impacts for baseline levels lower than 8 and no increase in the C-R function above 20. This likely biased our results downward.

- Interpretation of Expert B results. Expert B provided a conditional distribution for the C-R function, conditioned on an uncertain threshold. Expert B provided additional information about the shape of the distribution for the threshold. To develop an applied function, we assumed that the uncertain threshold could be incorporated into the C-R function through the construction of an expected value function. The specific functions may lead to a slight overestimate of mortality impacts.

- Use of simple averaging of expert results. To develop the combined expert distribution, we used equal weights for each expert. Given the lack of calibration questions in the pilot elicitation, this is the most defensible approach. However, many expert elicitation applications have use more complex weighting schemes based on how well experts are calibrated.

- Ranges based on individual experts should be viewed with caution as they represent only a single individual's interpretation of the state of knowledge about PM and mortality. Results for individual experts should not be extracted and presented without reference to the full range of results across the five experts.

- Any range of results presented based on this application should be presented along with their relative likelihood (i.e., the percentile represented in the distribution).

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

The primary analysis is based on our current interpretation of the scientific and economic literature. That interpretation requires judgments regarding the best available data, models, and modeling methodologies; and assumptions we consider 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 alternative choices exist for some inputs to the analysis, such as the mortality C-R functions.

We supplement our primary estimates of benefits with a series of sensitivity calculations that make use of other sources of health effect estimates and valuation data for key benefits categories. These estimates examine sensitivity to both valuation issues (e.g. the appropriate income elasticity) 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 tables should not be added together without addressing potential issues of overlap and low joint probability among the endpoints.

9C.1 Premature Mortality—Long term exposure

Given current evidence regarding their value, reduction in the risk of premature mortality is the most important PM-related health outcome in terms of contribution to dollar benefits. There are at least three 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, and effect thresholds. Results of this set of sensitivity analyses are presented in Table 9C.1.

9C.1.1 Alternative C-R Functions

Following the advice of the EPA Science Advisory Board Health Effects Subcommittee (SAB-HES), we used the Pope, et al. (2002) all-cause mortality model exclusively to derive our primary estimate of avoided premature mortality. While the SAB-HES "recommends that the base case rely on the Pope et al. (2002) study and that EPA use total mortality concentration-response functions (C-R), rather than separate cause-specific C-R functions, to calculate total PM mortality cases," they also suggested that "the cause-specific estimates can be used to communicate the relative contribution of the main air pollution related causes of death." As such, we provide the estimates of cardiopulmonary and lung cancer deaths based on the Pope et al. (2002).

In addition, the SAB-HES has noted that the American Cancer Society cohort used in Pope et al. (2002) "has some inherent deficiencies, in particular the imprecise exposure data, and the non-representative (albeit very large) population. Thus, ACS is not necessarily "the better

study," but, at this point in time, is a prudent choice for the base case estimates in the Second Prospective Analysis. The Harvard Six-Cities C-R functions are valid estimates on a more representative, although geographically selected, population, and its updated analysis has not yet been published. The Six Cities estimates may be used in a sensitivity analysis to demonstrate that with different but also plausible selection criteria for C-R functions, benefits may be considerably larger than suggested by the ACS study." (EPA-SAB-COUNCIL-ADV-04-002). In previous advice, the SAB has noted that "the [Harvard Six Cities] study had better monitoring with less measurement error than did most other studies" (EPA-SAB-COUNCIL-ADV-99-012, 1999). 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 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). We emphasize, that based on our understanding of the relative merits of the two datasets, the Pope, et al. (2002) 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 base estimate of this important health effect.

9C.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 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 previous 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 (NRC 2002). Rather it is intended to be a reasonable guess at the appropriate distribution of avoided incidences of PM-related mortality. The SAB-HES has recently noted that "empirical evidence is lacking to inform the choice of the lag distribution directly and agrees with the NAS report that there is little empirical justification for the 5-year cessation lag structure used in the previous analyses." The SAB-HES suggests that appropriate lag structures may be developed based on the distribution of cause specific deaths within the overall all-cause estimate. Diseases with longer progressions should be characterized by longer term lag structures, while air pollution impacts occurring in populations with existing disease may be characterized by shorter term lags.

A key question is the distribution of causes of death within the relatively broad categories analyzed in the long-term cohort studies. While we may be more certain about the appropriate length of cessation lag for lung cancer deaths, it is not at all clear what the appropriate lag structure should be for cardiopulmonary deaths, which include both respiratory and cardiovascular causes. Some respiratory diseases may have a long period of progression, while others, such as pneumonia, have a very short duration. In the case of cardiovascular disease, there is an important question of whether air pollution is causing the disease, which would imply a relatively long cessation lag, or whether air pollution is causing premature death in individuals with preexisting heart disease, which would imply very short cessation lags. The SAB-HES provides several recommendations for future research that could support the development of defensible lag structures, including the use of disease specific lag models, and the construction of a segmented lag distribution to combine differential lags across causes of death. The SAB-HES indicated support for using "a Weibull distribution or a simpler distributional form made up of several segments to cover the response mechanisms outlined above, given our lack of knowledge on the specific form of the distributions." However, they noted that "an important question to be resolved is what the relative magnitudes of these segments should be, and how many of the acute effects are assumed to be included in the cohort effect estimate." They conclude their discussion of cessation lags by stating that "given the current lack of direct data upon which to specify the lag function, the HES recommends that this question be considered for inclusion in future expert elicitation efforts and/or sensitivity analyses." (EPA-SAB-COUNCIL-ADV-04-002) EPA will continue to investigate this important issue for future benefits analyses and in the upcoming 2nd Prospective Analysis of the Costs and Benefits of the Clean Air Act. For this RIA, we investigate alternative cessation lag structures as senstivity analyses, noting that these might be as likely as the previous 5-year distributed lag in the base analysis.

Although the prior 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[c]. 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.

In addition to the simple 8 and 15 year lags, we have added an additional senstivity analysis examining the impact of assuming a segmented lag of the type suggested by the SAB-

[c]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.

HES. This illustrative lag structure is characterized by 20 percent of mortality reductions occuring in the first year, 50 percent occuring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 30 percent occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$. The distribution of deaths over the latency period is intended to reflect the contribution of short term exposures in the first year, cardiopulmonary deaths in the 2 to 5 year period, and longer term lung disease and lung cancer in the 6 to 20 year period. For future analyses, the specific distribution of deaths over time will need to be determined through research on causes of death and progression of diseases associated with air pollution. 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 Pope et al. ACS impact function) are presented in Table 9C.2. These estimates are based on the value of statistical lives saved approach, i.e. $5.5 million per incidence, and are presented for both a 3 and 7 percent discount rate over the lag period.

9C.1.3 Thresholds

Although the consistent advice from EPA's Science Advisory Board has been to model premature mortality associated with PM exposure as a non-threshold effect, that is, with harmful effects to exposed populations regardless of the absolute level of ambient PM concentrations, some analysts have hypothesized the presence of a threshold relationship[d]. 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.[e] EPA does not necessarily endorse any particular threshold and, as discussed in Appendix 9A, virtually every study to consider the issue indicates absence of a threshold.

We construct a senstivity analysis by assigning different cutpoints below which changes in $PM_{2.5}$ are assumed to have no impact on premature mortality. The sensitivity analysis illustrates how our estimates of the number of premature mortalities in the Base Estimate might change under a range of alternative assumptions for a PM mortality threshold. If, for example, there were no benefits of reducing PM concentrations below the $PM_{2.5}$ standard of 15 μg/m3, our estimate of the total number of avoided PM-related premature mortalities in 2030 from the preliminary modeling would be reduced by approximately 70 percent, from approximately

[D]The most recent advice from the SAB-HES is characterized by the following: "For the studies of long-term exposure, the HES notes that Krewski et al. (2000) have conducted the most careful work on this issue. They report that the associations between PM2.5 and both all-cause and cardiopulmonary mortality were near linear within the relevant ranges, with no apparent threshold. Graphical analyses of these studies (Dockery et al., 1993, Figure 3 and Krewski et al., 2000, page 162) also suggest a continuum of effects down to lower levels. Therefore, it is reasonable for EPA to assume a no threshold model down to, at least, the low end of the concentrations reported in the studies."

[E]The illustrative example in Appendix 9B presents the potential implications of assuming some probability of a threshold on the benefits estimate.

14,000 annually to approximately 4,000 annually. However, this type of cutoff is unlikely, as supported by the recent NRC report, which stated that "for pollutants such as PM_{10} and $PM_{2.5}$, there is no evidence for any departure of linearity in the observed range of exposure, nor any indiciation of a threshold. (NRC, 2002)" Another possible senstivity analysis which we have not conducted at this time might examine the potential for a nonlinear relationship at lower exposure levels.[f]

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.

The results of these sensitivity analyses demonstrate that choice of effect estimate can have a large impact on benefits, potentially doubling benefits if the effect estimate is derived from the HEI 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. The overall impact of moving from the 5-year distributed lag to a segmented lag is relatively modest, reducing benefits by approximately 8 percent when a three percent discount rate is used and 22 percent when a seven percent discount rate is used. If no lag is assumed, benefits are increased by around five percent. The threshold analysis indicates that approximately 85 percent of the premature mortality related benefits are due to changes in $PM_{2.5}$ concentrations occurring above 10 $\mu g/m^3$, and around 30 percent are due to changes above 15 $\mu g/m^3$, the current $PM_{2.5}$ standard.

[F]The pilot expert elicitation discussed in Appendix 9B provides some information on the impact of applying nonlinear and threshold based C-R functions.

Table 9C-1.
Sensitivity of Benefits of Premature Mortality Reductions to Alternative Assumptions (Relative to Base Case Benefits of Modeled Preliminary Control Option)

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)[C]					
Lung Cancer		*1,200*	*2,100*	*$7,700*	*$13,000*
Cardiopulmonary		*6,000*	*11,000*	*$37,000*	*$67,000*
Krewski/Harvard Six-city Study		17,000	30,000	$110,000	$190,000
Alternative Lag Structures for PM-related Premature Mortality					
None	Incidences all occur in the first year	7,800	14,000	$52,000	$94,000
8-year	Incidences all occur in the 8th year				
	3% Discount Rate	7,800	14,000	$42,000	$76,000
	7% Discount Rate	7,800	14,000	$32,000	$62,000
15-year	Incidences all occur in the 15th year				
	3% Discount Rate	7,800	14,000	$34,000	$62,000
	7% Discount Rate	7,800	14,000	$20,000	$36,000
Segmented	20 percent of incidences occur in 1st year, 50 percent in years 2 to 5, and 30 percent in years 6 to 20				
	3% Discount Rate	7,800	14,000	$45,000	$82,000
	7% Discount Rate	7,	14	$3	$6

Alternative Thresholds				
No Threshold (base estimate)	7,800	14,000	$49,000	$89,000
5	7,800	14,000	$49,000	$89,000
10	6,300	12,000	$40,000	$77,000
15	1,700	4,000	$11,000	$26,000
20	630	1,300	$4,000	$8,400
25	190	520	$1,200	$3,400

[A] Incidences rounded to two significant digits.

[B] Dollar values rounded to two significant digits.

[C] Note that the sum of lung cancer and cardiopulmonary deaths will not be equal to the total all cause death estimate. There is some residual mortality associated with long term exposures to $PM_{2.5}$ that is not captured by the caridopulmonary and lung cancer categories.

9C.2 Other Health Endpoint Sensitivity Analyses

9C.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 health impact function that linked a larger set of effects to a change in pollution, rather than using health impact functions for individual effects. For example, for premature mortality, we selected an impact 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 impact 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 9.C-3.

There has been a great deal of research recently on the potential effect of ozone on premature mortality. While the air pollutant most clearly associated with premature mortality is particulate matter, with dozens of studies reporting such an association, repeated ozone exposure is a likely contributing factor for premature mortality, causing an inflammatory response in the lungs which may predispose elderly and other sensitive individuals to become more susceptible. The findings of three recent analyses provide consistent data suggesting that ozone exposure is associated with increased mortality. Although the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) did not find an effect of ozone on total mortality across the full year, Samet et al. (2000), who conducted the NMMAPS study, did observe an effect after limiting the analysis to summer when ozone levels are highest. Similarly, Thurston and Ito (1999) have shown associations between ozone and mortality. Toulomi et al. (1997) found that 1-hour maximum ozone levels were associated with daily numbers of deaths in 4 cities (London, Athens, Barcelona, and Paris), and a quantitatively similar effect was found in a group of 4 additional cities (Amsterdam, Basel, Geneva, and Zurich). Fairly et al. (2003) also found a relatively strong association between maximum 8-hour average ozone concentrations and mortality in Santa Clara County, CA, even after controlling for $PM_{2.5}$ exposure.

While not as extensive as the data base for particulate matter, these recent studies provide supporting evidence for inclusion of mortality in the ozone health benefits analysis. 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. Thurston and Ito 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.

Thurston and Ito (2001) 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 Thurston and Ito only controlled for PM_{10} 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.

A recent World Health Organization (WHO) report found that "recent epidemiological studies have strengthened the evidence that there are short-term O_3 effects on

mortality and respiratory morbidity and provided further information on exposure-response relationships and effect modification." (WHO, 2003). Based on a preliminary meta-analysis, the WHO report suggests an effect estimate of between 0.2 and 0.4 percent increase in premature death per 10 $\mu g/m^3$ increase in 1 hour maximum ozone and between 0.4 and 0.6 percent increase in premature death per 10 $\mu g/m^3$ increase in daily average. This is equivalent to a relative risk of between 1.04 and 1.08 per 100 ppb increase in 1 hour maximum and between 1.08 and 1.12 per 100 ppb increase in daily average. The WHO report provides effect estimates for both all seasons and summer seasons. Because our analysis is limited to the summer ozone season, the most appropriate effect estimate is for the summer season. The WHO summer season relative risk estimate is 1.08 per 100 ppb increase in 1 hour maximum ozone and 1.12 per 100 ppb increase in daily average ozone.

Levy et al. (2001) assessed the epidemiological evidence examining the link between short term exposures to ozone and premature mortality. Based on four U.S. studies (Kellsall et al., 1997; Moolgavkar et al., 1995; Ito and Thurston, 1996; and Moolgavkar, 2000), they conclude that an appropriate pooled effect estimate is a 0.5 percent increase in premature deaths per 10 $\mu g/m^3$ increase in 24-hour average ozone concentrations, with a 95 percent confidence interval between 0.3 percent and 0.7 percent. This is equivalent to a relative risk of 1.10 per 100 ppb increase in daily average, which falls in the middle of the range of relative risks from the WHO report. Levy et al. also note that there are a number of studies which did not report a quantitative effect estimate but did indicate that ozone was insignificant. They suggest that the uncertainty surrounding the ozone-mortality effect estimate is greater than that reflected in the confidence interval around their pooled estimate.

In its September 2001 advisory on the draft analytical blueprint for the second Section 812 prospective analysis, the SAB Health Effects Subcommittee (HES) 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). Based on these new analyses and recommendations, EPA is sponsoring three independent meta-analyses of the ozone-mortality epidemiology literature to inform a determination on inclusion of this important health endpoint. Publication of these meta-analyses will significantly enhance the scientific defensibility of benefits estimates for ozone which include the benefits of premature mortality reductions. In its 2003 review of the analysis plans for the second Prospective Analysis, the HES indicated support for EPAs new meta-analyses of the ozone mortality literature and EPA's plans to consider adding ozone mortality to the base case analysis, subsequent to review of the results of the meta-analyses. Thus, recent evidence suggests that by not including an estimate of reductions in short-term mortality due to changes in ambient ozone, the Base Estimate may underestimate the benefits of implementation of the Nonroad Diesel Engine rule.

The ozone mortality sensitivity estimate is calculated using results from four U.S. studies (Ito and Thurston, 1996; Kinney et al., 1995; Moolgavkar et al., 1995; and Samet et al., 1997), 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. We include the Kinney et al., 1995 estimate for completeness, even

though Levy et al. (2001) reject the results because the study only included a linear term for temperature. Because the Kinney et al. (1995) study found no significant effect of ozone, this has the effect of reducing the estimated mortality impacts and increasing the uncertainty surrounding the estimated mortality reductions. 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.

Table 9C-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	$2,900	$6,800
Kinney et al. (1995)	0	0	$0	$0
Moolgavkar et al. (1995)	77	240	$510	$1,600
Samet et al. (1997)	120	360	$790	$2,400
Pooled estimate (random effects weights)	**940**	**2800**	**$6200**	**$19,000**
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 $5.5 million per premature statistical death, adjusted for income growth.

9C.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 9C.3, we show the impact of alternative assumptions about other parameters, including treatment of reversals in chronic bronchitis as lowest severity cases, alternative impact 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 9C-3.
Additional Parameter Sensitivity Analyses

Alternative Calculation		Description of Estimate	Impact on Base Benefit Estimate (million 2000$)	
			2020	2030
1	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%)
2	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%)
3	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%)
4	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%)
5	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%)

An important issue related to chronic conditions is the possible reversal in chronic bronchitis incidences (row 1 of Table 9C-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 2 of Table 9C-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 3 and 4 of Table 9C-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.

The alternative calculation for household soiling (row 5 of Table 9C-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.

9C.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 9C-4 lists the ranges elasticity values used to calculate the income adjustement factors, while Table 9C-5 lists the ranges of corresponding adjustement factors. The results of this sensitivity analysis, giving the monetized benefit subtotals for the four benefit categories, are presented in Table 9C-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 9C-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 9C-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	1.018	1.021	1.147	1.170
Severe and Chronic Health Effects	1.121	1.139	1.317	1.371
Premature Mortality	1.037	1.043	1.591	1.705
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 9C-6.
Sensitivity Analysis of Alternative Income Elasticities[A]

Benefit Category	Lower Sensitivity Bound		Upper Sensitivity Bound	
	2020	2030	2020	2030
Minor Health Effect	$510	$760	$540	$810
Severe and Chronic Health Effects	$2,500	$3,900	$2,800	$4,400
Premature Mortality	$42,000	$75,000	$65,000	$123,000
Visibility and Other Welfare Effects[A]	$1,400	$2,200	$1,400	$2,200
Total Benefits	$47,000	$82,000	$70,000	$131,000

[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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APPENDIX 9D: Visibility Benefits Estimates for Individual Class I Areas

Table 9D-1
Apportionment Factors for 2020 Park Specific Visibility Benefits

PARK	COUNTY	STATE	Percent of 2020 Visibility Benefit Due to Changes in:		
			SO_2	NOx	direct PM
Shenandoah	Lawrence	AL	0.428	0.234	0.338
Anaconda-Pintlar W	Cochise Co	AZ	0.337	0.061	0.602
Boundary Waters	Gila Co	AZ	0.396	0.054	0.550
Breton W	Gila Co	AZ	0.396	0.054	0.550
Isle Royale	Coconino	AZ	0.336	0.053	0.612
Jarbidge W	Apache Co	AZ	0.469	0.049	0.481
Medicine Lake W	Apache Co	AZ	0.469	0.049	0.481
Red Rock Lakes W	Graham Co	AZ	0.302	0.038	0.660
Roosevelt Campobello	Pima Co	AZ	0.224	0.061	0.715
Selway-Bitterroot W	Maricopa	AZ	0.061	0.014	0.924
Seney W	Coconino	AZ	0.336	0.053	0.612
Wolf Island W	Yavapai Co	AZ	0.216	0.140	0.644
Agua Tibia W	Tuolumne	CA	0.090	0.580	0.330
Black Canyon of the	San	CA	0.074	0.158	0.768
Caribou W	Calaveras	CA	0.049	0.520	0.432
Chiricahua	Trinity Co	CA	0.367	0.239	0.394
Cucamonga W	Fresno Co	CA	0.051	0.101	0.848
Dome Land W	Mono Co	CA	0.195	0.302	0.504
Flat Tops W	Inyo Co	CA	0.145	0.098	0.757
Grand Canyon	Marin Co	CA	0.060	0.577	0.363
Hoover W	Los	CA	0.099	0.143	0.758
John Muir W	Monterey	CA	0.071	0.563	0.366
Kaiser W	San Benito	CA	0.057	0.633	0.310
La Garita W	Riverside	CA	0.040	0.314	0.646
Mazatzal W	Siskiyou	CA	0.469	0.220	0.311
Mesa Verde	San	CA	0.074	0.158	0.768
Petrified Forest	Del Norte	CA	0.518	0.097	0.385
Pine Mountain W	Shasta Co	CA	0.146	0.469	0.385
Pinnacles	Fresno Co	CA	0.051	0.101	0.848
Point Reyes	Lassen Co	CA	0.285	0.347	0.368
Rawah W	Riverside	CA	0.040	0.314	0.646
Rocky Mountain	San Diego	CA	0.068	0.497	0.435
Saguaro	Shasta Co	CA	0.146	0.469	0.385
San Gabriel W	El Dorado	CA	0.050	0.487	0.463
San Gorgino W	Mariposa	CA	0.085	0.374	0.541
San Jacinto W	Fresno Co	CA	0.051	0.101	0.848
San Rafael W	Tuolumne	CA	0.090	0.580	0.330
Sequoia-Kings	Tulare Co	CA	0.052	0.478	0.470
Sycamore Canyon W	Siskiyou	CA	0.469	0.220	0.311
Ventana W	Santa	CA	0.111	0.156	0.733
Yolla-Bolly-Middle-	Tulare Co	CA	0.052	0.478	0.470

PARK	COUNTY	STATE	Percent of 2020 Visibility Benefit Due to Changes in:		
			SO_2	NOx	direct PM
Yosemite	Modoc Co	CA	0.277	0.407	0.316
Carlsbad Caverns	San Juan	CO	0.522	0.114	0.364
Gila W	Garfield Co	CO	0.335	0.246	0.420
Joyce Kilmer-Slickrock	Routt Co	CO	0.420	0.140	0.440
Kalmiopsis W	Larimer Co	CO	0.449	0.120	0.431
Linville Gorge W	Pitkin Co	CO	0.425	0.098	0.477
Lostwood W	Alamosa	CO	0.458	0.097	0.445
Pecos W	Gunnison	CO	0.437	0.152	0.411
Presidential Range-Dry	Montezuma	CO	0.353	0.077	0.570
Salt Creek W	Montrose	CO	0.355	0.175	0.470
Shining Rock W	Summit Co	CO	0.525	0.042	0.433
Wheeler Peak W	Mineral Co	CO	0.589	0.048	0.364
Wichita Mountains W	Larimer Co	CO	0.449	0.120	0.431
Fitzpatrick W	Monroe Co	FL	0.546	0.020	0.434
Glacier Peak W	Wakulla Co	FL	0.535	0.048	0.417
Mount Adams W	Citrus Co	FL	0.416	0.148	0.436
Dolly Sods W	Charlton	GA	0.543	0.058	0.399
North Absaroka W	McIntosh	GA	0.500	0.052	0.448
Olympic	Edmonson	KY	0.415	0.246	0.338
Lye Brook W	Stone Co	MS	0.539	0.112	0.349
Bridger W	Hyde Co	NC	0.344	0.327	0.329
Goat Rocks W	Haywood	NC	0.476	0.191	0.333
Otter Creek W	Avery Co	NC	0.516	0.184	0.300
Pasayten W	Graham Co	NC	0.564	0.138	0.298
Bandelier	Sandoval	NM	0.426	0.034	0.540
Bosque del Apache W	Rio Arriba	NM	0.512	0.047	0.441
Brigantine W	Grant Co	NM	0.414	0.017	0.569
Crater Lake	Chaves Co	NM	0.471	0.094	0.434
Mount Hood W	Mora Co	NM	0.568	0.081	0.352
Mount Washington W	Eddy Co	NM	0.417	0.052	0.531
San Pedro Parks W	Socorro Co	NM	0.409	0.025	0.565
Swanguarter W	Taos Co	NM	0.538	0.057	0.405
Theodore Roosevelt	Lincoln Co	NM	0.603	0.056	0.341
Maroon Bells-	Elko Co	NV	0.311	0.301	0.388
Mount Rainier	Polk Co	TN	0.405	0.237	0.358
North Cascades	Blount Co	TN	0.384	0.184	0.432
Bob Marshall W	San Juan	UT	0.373	0.048	0.579
Gates of the Mountain	Grand Co	UT	0.354	0.038	0.608
Glacier	San Juan	UT	0.373	0.048	0.579
St. Marks W	Washington	UT	0.219	0.096	0.685
Voyageurs	Garfield Co	UT	0.295	0.052	0.652
Teton W	Botetourt	VA	0.485	0.151	0.364
Yellowstone	Madison	VA	0.385	0.316	0.300
Grand Teton NP	Grant Co	WV	0.533	0.190	0.278
Washakie W	Tucker Co	WV	0.568	0.118	0.314

Table 9D-2
Apportionment Factors for 2030 Park Specific Visibility Benefits

PARK	COUNTY	STATE	Percent of 2030 Visibility Benefit Due to Changes in: SO_2	NOx	direct PM
Shenandoah	Lawrence	AL	0.376	0.297	0.327
Anaconda-Pintlar W	Cochise	AZ	0.313	0.075	0.612
Boundary Waters	Gila Co	AZ	0.277	0.048	0.675
Breton W	Gila Co	AZ	0.293	0.089	0.619
Isle Royale	Coconino	AZ	0.342	0.107	0.551
Jarbidge W	Apache	AZ	0.429	0.069	0.503
Medicine Lake W	Apache	AZ	0.429	0.069	0.503
Red Rock Lakes W	Graham	AZ	0.188	0.173	0.639
Roosevelt Campobello	Pima Co	AZ	0.207	0.072	0.721
Selway-Bitterroot W	Maricopa	AZ	0.342	0.107	0.551
Seney W	Coconino	AZ	0.057	0.019	0.924
Wolf Island W	Yavapai	AZ	0.293	0.089	0.619
Agua Tibia W	Tuolumne	CA	0.055	0.571	0.375
Black Canyon of the	San	CA	0.226	0.407	0.368
Caribou W	Calaveras	CA	0.065	0.191	0.745
Chiricahua	Trinity Co	CA	0.129	0.111	0.759
Cucamonga W	Fresno Co	CA	0.039	0.520	0.441
Dome Land W	Mono Co	CA	0.046	0.493	0.461
Flat Tops W	Inyo Co	CA	0.070	0.616	0.314
Grand Canyon	Marin Co	CA	0.070	0.616	0.314
Hoover W	Los	CA	0.049	0.109	0.842
John Muir W	Monterey	CA	0.033	0.376	0.591
Kaiser W	San Benito	CA	0.049	0.109	0.842
La Garita W	Riverside	CA	0.049	0.109	0.842
Mazatzal W	Siskiyou	CA	0.116	0.518	0.366
Mesa Verde	San	CA	0.411	0.270	0.320
Petrified Forest	Del Norte	CA	0.411	0.270	0.320
Pine Mountain W	Shasta Co	CA	0.158	0.344	0.498
Pinnacles	Fresno Co	CA	0.043	0.535	0.422
Point Reyes	Lassen Co	CA	0.047	0.663	0.289
Rawah W	Riverside	CA	0.053	0.588	0.360
Rocky Mountain	San Diego	CA	0.468	0.133	0.399
Saguaro	Shasta Co	CA	0.090	0.175	0.735
San Gabriel W	El Dorado	CA	0.065	0.191	0.745
San Gorgino W	Mariposa	CA	0.033	0.376	0.591
San Jacinto W	Fresno Co	CA	0.099	0.179	0.722
San Rafael W	Tuolumne	CA	0.046	0.493	0.461
Sequoia-Kings	Tulare Co	CA	0.225	0.452	0.323
Sycamore Canyon W	Siskiyou	CA	0.116	0.518	0.366
Ventana W	Santa	CA	0.059	0.593	0.348
Yolla-Bolly-Middle-	Tulare Co	CA	0.321	0.292	0.386
Yosemite	Modoc Co	CA	0.073	0.400	0.527
Carlsbad Caverns	San Juan	CO	0.312	0.203	0.485
Gila W	Garfield	CO	0.464	0.087	0.449

PARK	COUNTY	STATE	Percent of 2030 Visibility Benefit Due to Changes in: SO_2	NOx	direct PM
Joyce Kilmer-Slickrock	Routt Co	CO	0.289	0.286	0.425
Kalmiopsis W	Larimer	CO	0.407	0.123	0.470
Linville Gorge W	Pitkin Co	CO	0.537	0.074	0.389
Lostwood W	Alamosa	CO	0.391	0.103	0.505
Pecos W	Gunnison	CO	0.320	0.091	0.589
Presidential Range-Dry	Montezum	CO	0.367	0.180	0.452
Salt Creek W	Montrose	CO	0.397	0.156	0.447
Shining Rock W	Summit	CO	0.397	0.156	0.447
Wheeler Peak W	Mineral	CO	0.471	0.140	0.389
Wichita Mountains W	Larimer	CO	0.385	0.188	0.428
Fitzpatrick W	Monroe	FL	0.365	0.204	0.431
Glacier Peak W	Wakulla	FL	0.503	0.033	0.464
Mount Adams W	Citrus Co	FL	0.497	0.070	0.433
Dolly Sods W	Charlton	GA	0.503	0.085	0.412
North Absaroka W	McIntosh	GA	0.463	0.082	0.456
Olympic	Edmonson	KY	0.365	0.304	0.332
Lye Brook W	Stone Co	MS	0.486	0.166	0.348
Bridger W	Hyde Co	NC	0.515	0.183	0.302
Goat Rocks W	Haywood	NC	0.455	0.252	0.293
Otter Creek W	Avery Co	NC	0.436	0.232	0.332
Pasayten W	Graham	NC	0.309	0.371	0.320
Bandelier	Sandoval	NM	0.389	0.051	0.560
Bosque del Apache W	Rio Arriba	NM	0.374	0.037	0.589
Brigantine W	Grant Co	NM	0.378	0.069	0.553
Crater Lake	Chaves Co	NM	0.387	0.021	0.592
Mount Hood W	Mora Co	NM	0.525	0.100	0.375
Mount Washington W	Eddy Co	NM	0.421	0.124	0.455
San Pedro Parks W	Socorro	NM	0.472	0.059	0.469
Swanguarter W	Taos Co	NM	0.481	0.092	0.427
Theodore Roosevelt	Lincoln	NM	0.553	0.078	0.369
Maroon Bells-	Elko Co	NV	0.261	0.345	0.394
Mount Rainier	Polk Co	TN	0.359	0.295	0.346
North Cascades	Blount Co	TN	0.345	0.232	0.423
Bob Marshall W	San Juan	UT	0.322	0.046	0.632
Gates of the Mountain	Grand Co	UT	0.265	0.065	0.671
Glacier	San Juan	UT	0.337	0.064	0.600
St. Marks W	Washingto	UT	0.337	0.064	0.600
Voyageurs	Garfield	UT	0.190	0.129	0.680
Teton W	Botetourt	VA	0.445	0.193	0.361
Yellowstone	Madison	VA	0.331	0.387	0.282
Grand Teton NP	Grant Co	WV	0.455	0.275	0.270
Washakie W	Tucker Co	WV	0.487	0.200	0.313