Control of Emissions from Marine SI and Small SI Engines, Vessels, and Equipment

Draft Regulatory Impact Analysis

Chapter 8
Cost-Benefit Analysis

Assessment and Standards Division Office of Transportation and Air Quality U.S. Environmental Protection Agency

CHAPTER 8: Cost-Benefit Analysis	1
8.1 Overview	1
8.2 Air Quality Impacts	8
8.2.1 PM Air Quality Impact Estimation	8
8.3 PM-Related Health Benefits Estimation - Methods and Inputs	10
8.4 Benefits Analysis Results for the Proposed Standards	17
8.5 Unquantified Health and Welfare Effects	
8.5.1 Human Health Impact Assessment	21
8.5.2 Welfare Impact Assessment	
8.5.3 UVb Exposure	23
8.6 Methods for Describing Uncertainty	23
8.6.1 Analysis of Statistical Uncertainty	25
8.6.2 Additional Approaches to Characterizing Uncertainty Related to PM-Mortality	
8.7 Health-Based Cost Effectiveness Analysis	
8.8 Comparison of Costs and Benefits	

CHAPTER 8: Cost-Benefit Analysis

8.1 Overview

Mobile sources are significant contributors to air pollutant emissions across the country and into the future. The Agency has determined that these emissions cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare, and is therefore establishing standards to control these emissions. The health- and environmentally-related effects associated with these emissions are a classic example of an externality-related market failure. An externality occurs when one party's actions impose uncompensated costs on another party. The proposed Small SI and Marine SI engine standards will help correct this market failure.

EPA is required by Executive Order (E.O.) 12866 to estimate the benefits and costs 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 particulate matter (PM) and ozone precursor emission reductions (direct PM, NO_x and VOC)? (2) what is the monetary value of the changes in these effects attributable to the proposed rule? and (3) how do the monetized benefits compare to the costs? It constitutes one part of EPA's thorough examination of the relative merits of this regulation.

This chapter presents our analysis of the health and environmental benefits that can be expected to occur as a result of the proposed standards throughout the period from initial implementation through 2030. Nationwide, the engines subject to the proposed emission standards in this rule are a significant source of mobile source air pollution. The proposed standards would reduce exposure to VOC, direct PM_{2.5}, NOx and CO emissions and help avoid a range of adverse health effects associated with ambient ozone and PM_{2.5} levels. In addition, the proposed standards would help reduce exposure to CO, air toxics, and PM_{2.5} for persons who operate or who work with or are otherwise active in close proximity to these engines.

The analysis presented in this chapter uses a methodology generally consistent with benefits analyses performed for the recent analysis of the the Clean Air Nonroad Diesel Rule (CAND) and the Mobile Source Air Toxics Rule (MSAT). To the extent possible, we also incorporate benefits analysis methods consistent with the approach used in the recent RIA for the PM NAAQS. For this reason, the current chapter avoids repeating this information and refers to the appropriate sections of each RIA. The benefits analysis relies on two major components:

- 1) Calculation of the impact of the proposed standards on the national direct PM and NOx emissions inventories for two future years (2020 and 2030).
- 2) A benefits analysis to determine the changes in human health, both in terms of physical effects and monetary value, based on a PM benefits transfer approach that scales CAND results (see Section 8.2).

It should be noted that since the CAND rule, EPA's Office of Air and Radiation (OAR) has adopted a different format for its benefits analysis in which characterization of uncertainty is integrated into the main benefits analysis. The benefits scaling approach used in the analysis of the proposed standards limits our ability to integrate uncertainty into the main analysis. For the benefits analysis of the final standards, we will adopt this integrated uncertainty approach. Please see the PM NAAQS RIA for an indication of the uncertainty present in the base estimate of benefits and the sensitivity of our results to the use of alternative concentration-response functions.

A wide range of human health and welfare effects are linked to the emissions of VOCs, direct PM and NOx and the resulting impact on ambient concentrations of ozone and PM_{2.5}. Recent studies have linked short-term ozone exposures with premature mortality. Exposure to ozone has also been linked to a variety of respiratory effects including hospital admissions and illnesses resulting in school absences. Potential human health effects associated with PM_{2.5} range from premature mortality to 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). 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.

EPA typically quantifies PM- and ozone-related benefits in its regulatory impact analyses (RIAs) when possible. In the analysis of past air quality regulations, ozone-related benefits have included morbidity endpoints and welfare effects such as damage to commercial crops. EPA has not recently included a separate and additive mortality effect for ozone, independent of the effect associated with fine particulate matter. For a number of reasons, including 1) advice from the Science Advisory Board (SAB) Health and Ecological Effects Subcommittee (HEES) that EPA consider the plausibility and viability of including an estimate of premature mortality associated with short-term ozone exposure in its benefits analyses and 2) conclusions regarding the scientific support for such relationships in EPA's 2006 Air Quality Criteria for Ozone and Related Photochemical Oxidants (the CD), EPA is in the process of determining how to appropriately characterize ozone-related mortality benefits within the context of benefits analyses for air quality regulations. As part of this process, we are seeking advice from the National Academy of Sciences (NAS) regarding how the ozone-mortality literature should be used to quantify the reduction in premature mortality due to diminished exposure to ozone, the amount of life expectancy to be added and the monetary value of this increased life expectancy in the context of health benefits analyses associated with regulatory assessments. In addition, the Agency has sought advice on characterizing and communicating the uncertainty associated with each of these aspects in health benefit analyses.

Since the NAS effort is not expected to conclude until 2008, the agency is currently deliberating how best to characterize ozone-related mortality benefits in its rulemaking analyses in the interim. For the analysis of the proposed locomotive and marine standards, we do not quantify an ozone mortality benefit. So that we do not provide an incomplete picture of all of the benefits associated with reductions in emissions of ozone precursors, we have chosen not to

include an estimate of total ozone benefits in the proposed RIA. By omitting ozone benefits in this proposal, we acknowledge that this analysis underestimates the benefits associated with the proposed standards. Our analysis, however, indicates that the rule's monetized PM_{2.5} benefits alone substantially exceed our estimate of the costs.

Table 8.1-1 summarizes the annual monetized health and welfare benefits associated with the proposed standards for two years, 2020 and 2030. The PM_{2.5} benefits are scaled based on relative changes in direct PM and NOx emissions between this rule and the proposed Clean Air Nonroad Diesel (CAND) rule. As explained in Section 8.2.1 of this chapter, the PM_{2.5} benefits scaling approach is limited to those studies, health impacts, and assumptions that were used in the proposed CAND analysis. As a result, PM-related premature mortality is based on the updated analysis of the American Cancer Society cohort (ACS; Pope et al., 2002). However, it is important to note that since the CAND rule, EPA's Office of Air and Radiation (OAR) has adopted a different format for its benefits analysis in which characterization of the uncertainty in the concentration-response function is integrated into the main benefits analysis. Within this context, additional data sources are available, including a recent expert elicitation and updated analysis of the Six-Cities Study cohort (Laden et al., 2006). Please see the PM NAAQS RIA for an indication of the sensitivity of our results to use of alternative concentration-response functions.

The analysis presented here assumes a PM threshold of $3 \mu g/m^3$, equivalent to background. Through the RIA for CAIR, EPA's consistent approach had been to model premature mortality associated with PM exposure as a nonthreshold effect; that is, with harmful effects to exposed populations modeled regardless of the absolute level of ambient PM concentrations. This approach had been supported by advice from EPA's technical peer review panel, the Science Advisory Board's Health Effects Subcommittee (SAB-HES). However, EPA's most recent PM_{2.5} Criteria Document concludes that "the available evidence does not either support or refute the existence of thresholds for the effects of PM on mortality across the range of concentrations in the studies," (p. 9-44). Furthermore, in the RIA for the PM NAAQS we used a threshold of $10 \mu g/m^3$ based on recommendations by CASAC for the Staff Paper analysis. We consider the impact of a potential, assumed threshold in the PM-mortality concentration response function in Section 8.6.2.2 of the RIA.

-

^A Due to time and resource constraints, EPA scaled the final CAND benefits estimates from the benefits estimated for the CAND proposal. The scaling approach used in that analysis, and applied here, is described in the RIA for the final CAND rule.²

Table 8.1-1. Estimated Monetized PM-Related Health Benefits of the Proposed Standards

	Total Benefits ^{a, b, c} (billions 2003\$) 2020 2030			
Using a 3% discount rate	\$2.1 + B	\$3.4 + B		
Using a 7% discount rate	\$1.9 + B	\$3.1 + B		

Benefits include avoided cases of mortality, chronic illness, and other morbidity health endpoints. PM-related mortality benefits estimated using an assumed PM threshold at background levels (3 μg/m³). There is uncertainty about which assumed threshold to use and this may impact the magnitude of the total benefits estimate. For a more detailed discussion of this issue, please refer to Section 8.6.2.2 of the RIA.

Table 8.1-2 lists the full complement of human health and welfare effects associated with PM, ozone and air toxics, and identifies those effects that are quantified for the primary estimate and those that remain unquantified because of current limitations in methods or available data.

Table 8.1-2. Human Health and Welfare Effects of Pollutants Affected by the Proposed Standards

Pollutant/Effect	Quantified and Monetized in Base Estimates ^a	Unquantified Effects - Changes in:
PM/Health ^b	Premature mortality based on cohort study estimates ^c Bronchitis: chronic and acute Hospital admissions: respiratory and cardiovascular Emergency room visits for asthma Nonfatal heart attacks (myocardial infarction) Lower and upper respiratory illness Minor restricted-activity days Work loss days Asthma exacerbations (asthmatic population) Respiratory symptoms (asthmatic population) Infant mortality	Premature mortality: short-term exposures ^d Subchronic bronchitis cases Low birth weight Pulmonary function Chronic respiratory diseases other than chronic bronchitis Nonasthma respiratory emergency room visits UVb exposure (+/-) ^e

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 8.1-2 of the RIA.

Results reflect the use of two different discount rates: 3 and 7 percent, which are recommended by EPA's *Guidelines for Preparing Economic Analyses*⁵ and OMB Circular A-4.⁶ Results are rounded to two significant digits for ease of presentation and computation.

Pollutant/Effect	Quantified and Monetized in Base Estimates ^a	Unquantified Effects - Changes in:
PM/Welfare		Visibility in Southeastern Class I areas Visibility in northeastern and Midwestern Class I areas Household soiling Visibility in western U.S. Class I areas Visibility in residential and non-Class I areas UVb exposure (+/-)e
Ozone/Health ^f		Premature mortality: short-term exposures ^g Hospital admissions: respiratory Emergency room visits for asthma Minor restricted-activity days School loss days Asthma attacks Cardiovascular emergency room visits Acute respiratory symptoms Chronic respiratory damage Premature aging of the lungs Nonasthma respiratory emergency room visits UVb exposure (+/-) ^e
Ozone/Welfare		Decreased outdoor worker productivity Yields for: - Commercial forests - Fruits and vegetables, and - Other commercial and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions UVb exposure (+/-)e
MSAT Health ^h		Cancer (benzene, 1,3-butadiene, formaldehyde, acetaldehyde, naphthalene) 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 mucus membranes (formaldehyde) Respiratory irritation (formaldehyde) 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 and congestion (acrolein) Neurotoxicity (n-hexane, toluene, xylenes)
MSAT Welfareh		Direct toxic effects to animals Bioaccumulation in the food chain Damage to ecosystem function Odor

^a Primary quantified and monetized effects are those included when determining the primary estimate of total monetized benefits of the proposed standards.

Figure 8.1-1 illustrates the major steps in the PM benefits analysis. Given the change in direct PM and NOx emissions modeled for the proposed standards, we use a benefits transfer approach to scale PM benefits estimated for the CAND analysis (see Section 8.2 for a description of the scaling approach). For the CAND analysis, EPA ran a sophisticated photochemical air quality model, the Regional Modeling System for Aerosols and Deposition (REMSAD), to estimate baseline and post-control ambient concentrations of PM for each future year (2020 and 2030). The estimated changes in ambient concentrations were then combined with population projections to estimate population-level potential exposures to changes in ambient concentrations. Changes in population exposure to ambient air pollution were then input to impact functions^B to generate changes in the incidence of health effects. The resulting changes in incidence were 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). Values for individual health and welfare effects were summed to obtain an estimate of the total monetary value of the changes in emissions. Finally, we scale the CAND results to reflect the magnitude of the direct PM and NOx emissions changes we estimate will occur as a result of the proposed standards.

Benefits estimates calculated for the CAND analysis, and scaled for the proposed

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

^c Cohort estimates are designed to examine the effects of long term exposures to ambient pollution, but relative risk estimates may also incorporate some effects due to shorter-term exposures (see Kunzli, 2001 for a discussion of this issue).⁷

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

^e May result in benefits or disbenefits. See Section 8.5.3. for more details.

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

^g EPA sponsored a series of meta-analyses of the ozone mortality epidemiology literature, published in the July 2005 volume of the journal Epidemiology, which found that short-term exposures to ozone may have a significant effect on daily mortality rates, independent of exposure to PM. EPA is currently considering how to include an estimate of ozone mortality in its benefits analyses.

^h The categorization of unquantified toxic health and welfare effects is not exhaustive.

^B The term "impact function" as used here refers to the combination of a) an effect estimate obtained from the epidemiological literature, b) the baseline incidence estimate for the health effect of interest in the modeled population, c) the size of that modeled population, and d) the change in the ambient air pollution metric of interest. These elements are combined in the impact function to generate estimates of changes in incidence of the health effect. The impact function is distinct from the C-R function, which strictly refers to the estimated equation from the epidemiological study relating incidence of the health effect and ambient pollution. We refer to the specific value of the relative risk or estimated coefficients in the epidemiological study as the "effect estimate." In referencing the functions used to generate changes in incidence of health effects for this RIA, we use the term "impact function" rather than C-R function because "impact function" includes all key input parameters used in the incidence calculation.

standards, were generated using the Environmental Benefits Mapping and Analysis Program (BenMAP). BenMAP is a computer program developed by EPA that integrates a number of the modeling elements used in previous RIA's (e.g., interpolation functions, population projections, health impact functions, valuation functions, analysis and pooling methods) to translate modeled air concentration estimates into health effect incidence estimates and monetized benefit estimates. Interested parties may wish to consult the webpage http://www.epa.gov/ttn/ecas/benmodels.html for more information.

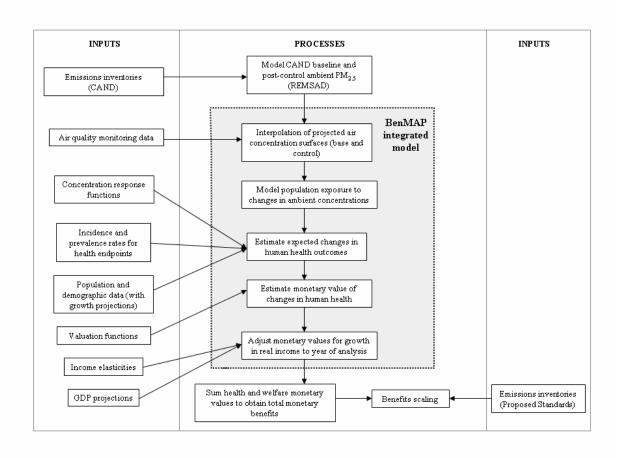


Figure 8.1-1. Key Steps in Air Quality Modeling Based Benefits Analysis

All of the benefit estimates for the proposed control options in this analysis are based on an analytical structure and sequence similar to that used in the benefits analyses for the CAND final rule, the CAIR rule, and, when feasible, the final PM NAAQS analysis. By adopting the major design elements, models, and assumptions developed in recent RIAs, we rely on methods that have already received extensive review by the independent Science Advisory Board (SAB),

^C See: Clean Air Nonroad Diesel final rule (69 FR 38958, June 29, 2004); Clean Air Interstate final rule (70 FR 25162, May 12, 2005); PM NAAQS (71 FR 61144, Oct. 17, 2006).

by the public, and by other federal agencies. In addition, we will be working through the next section 812 prospective study to enhance our methods.^D

This chapter is organized as follows. In Section 8.2, we provide an overview of the air quality impacts modeled for the proposed standards that are used as inputs to the benefits analysis. In Section 8.3, we document key differences between this benefits analysis and the benefits analysis completed for the final CAIR and CAND rules. This section also presents and discusses the key inputs and methods used in the benefits analysis. In Section 8.4, we report the results of the analysis for human health and welfare effects. Section 8.5 qualitatively describes benefits categories that are omitted from this analysis, due either to inadequate methods or resources. Section 8.6 discusses how we incorporate uncertainty into our analysis. Section 8.7 discusses the health-based cost-effectiveness analysis for the proposed standards. Finally, in Section 8.8, we present a comparison of the costs and benefits associated with the proposed standards.

8.2 Air Quality Impacts

This section summarizes the methods for and results of estimating air quality for the 2020 and 2030 base case and proposed control scenario for the purposes of the benefits analysis. EPA has focused on the health, welfare, and ecological effects that have been linked to ambient changes in PM_{2.5} related to direct PM and NOx emission reductions estimated to occur due to the proposed standards. We do this by scaling the modeled relationship between emissions and ambient PM concentrations observed for the CAND analysis.⁸

8.2.1 PM Air Quality Impact Estimation

To estimate PM_{2.5} benefits from the proposed standards, we rely on a benefits transfer technique. The benefits transfer approach uses as its foundation the relationship between emission reductions and ambient PM_{2.5} concentrations modeled for the Clean Air Nonroad Diesel (CAND) proposal. E For a given future year, we first calculate the ratio between CAND PM_{2.5} precursor emission reductions (direct PM and NOx) and PM_{2.5} precursor emission reductions associated with the proposed standards (proposed emission reductions/CAND emission reductions, displayed in Table 8.2-1). We multiply these ratios by the percent that each PM_{2.5} precursor contributes towards population-weighted reductions in total PM_{2.5} due to the CAND standards (displayed in Table 8.2-2). This calculation results in a "benefits apportionment factor" for the relationship between direct PM emissions and ambient PM_{2.5} and NOx emissions and ambient PM_{2.5} (displayed in Table 8.2-3). The benefits apportionment factors are then applied to the BenMAP-based incidence and monetized benefits from the CAND proposal. In this way, we apportion the results of the proposed CAND analysis to its underlying PM_{2.5} precursor emission reductions and scale the apportioned benefits to reflect differences in

^D Interested parties may want to consult the webpage: http://www.epa.gov/science1 regarding components of the 812 prospective analytical blueprint.

^E See 68 FR 28327, May 23, 2003.

emission reductions between the two rules.^F This benefits transfer method is consistent with the approach used in other recent mobile and stationary source rules.^G We refer the reader to the final CAND RIA for more details on this benefits transfer approach.⁹

Table 8.2-1. Comparison of 48-state Emission Reductions in 2020 and 2030 Between the CAND Rule and Proposed Standards

Emissions Species	Reduction from	Reduction from Baseline (tons)		
	CAND Modeling Inputs ^a	Small SI/Marine SI Emissions Changes ^b	(Proposal/ CAND)	
2020				
NOx	663,618	72,257	0.11	
Direct PM _{2.5}	98,121	4,896	0.05	
2030				
NOx	1,009,774	98,146	0.10	
Direct PM _{2.5}	138,208	6,299	0.05	

^a Includes all affected nonroad sources: land-based, recreational marine, commercial marine, and locomotives. See the CAND RIA for more information regarding the CAND emission inventories.

^b Includes changes to the small spark ignition engine inventory (lawn and garden equipment) and recreational marine spark ignition engine inventory.

F Note that while the proposed regulations also control VOCs, which contribute to PM formation, the benefits transfer scaling approach only scales benefits based on NOx, SO2, and direct PM emission reductions. PM benefits will likely be underestimated as a result, though we are unable to estimate the magnitude of the underestimation.

G See: Clean Air Nonroad Diesel final rule (69 FR 38958, June 29, 2004); Nonroad Large Spark-Ignition Engines and Recreational Engines standards (67 FR 68241, November 8, 2002); Final Industrial Boilers and Process Heaters NESHAP (69 FR 55217, September 13, 2004); Final Reciprocating Internal Combustion Engines NESHAP (69 FR 33473, June 15, 2004); Final Clean Air Visibility Rule (EPA-452/R-05-004, June 15, 2005); Ozone Implementation Rule (70 FR 71611, November 29, 2005).

Table 8.2-2. Apportionment of Modeled CAND Preliminary Control Option Populationweighted Change in Ambient PM_{2.5} to Nitrate, Sulfate, and Primary Particles

	202	0	203	30
	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%

Source: CAND RIA, Chapter 9.

Table 8.2-3. Calculation of PM_{2.5} Benefits Apportionment Factors for the Proposed Emission Reductions

		2020		2030			
	Ratio of	% of Total	Benefits	Ratio of	% of Total	Benefits	
	Emission	Ambient	Apportionment	Emission	Ambient	Apportionment	
	Reductions ^a	Change ^b	Factor	Reductions ^a	Change ^b	Factor	
	(1)	(2)	(1*2)	(3)	(4)	(3*4)	
NOx	0.11	0.131	0.014	0.10	0.168	0.016	
Emissions	0.11	0.131	0.014	0.10	0.100	0.010	
Direct PM	0.05	0.644	0.032	0.05	0.627	0.029	
Emissions	0.03	0.044	0.032	0.03	0.027	0.027	

^a Calculated by dividing the small SI and marine SI engine emission reductions by CAND emission reductions. See Table 8.2-1.

8.3 PM-Related Health Benefits Estimation - Methods and Inputs

The analytical approach used in this benefits analysis is largely the same approach used in the Final CAND benefits analysis and the reader is referred to that RIA for details on the benefits methods and inputs. This analysis, however, also reflects some advances in data and methods in epidemiology, economics, and health impact estimation consistent with the approach used in the recent RIA for the PM NAAQS. Updates to the assumptions and methods used in estimating PM_{2.5}-related benefits since the analysis for the CAND rule include the following:

• Consistent with the approach used in the recent RIA for the PM NAAQS, we have updated our projections of mortality incidence rates to be consistent with the U.S. Census population projections that form the basis of our future population estimates. Compared to the methodology used in the CAIR analysis, this change will result in a reduction in mortality impacts in future years, as overall mortality rates are projected to decline for most age groups. A memorandum drafted by Abt Associates (Abt Associates, 2005) contains complete details regarding the derivation of mortality rate

^b See Table 8.2-2.

- adjustment factors, and estimation of future-year mortality rates used in the analysis. ¹⁰ The scaled mortality benefits for the proposed standards have been updated accordingly.
- Consistent with the approach used in the recent RIA for the PM NAAQS, we use a revised mortality lag assumption. In the Final CAND, we used a five-year segmented lag. Since that analysis, upon which the PM benefits transfer scaling approach is based, the SAB Health Effects Subcommittee (HES) recommended that until additional research has been completed, EPA should assume a segmented lag structure characterized by 30 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in PM_{2.5}, and 20 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 long-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. This approach is different than the 5-year segmented lag used in the CAND analysis, and the scaled benefits analysis of the proposed standards has been updated accordingly.

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 PM. The specific studies from which effect estimates for the primary analysis are drawn are included in Table 8.3-1. The specific unit values used for economic valuation of health endpoints are included in Table 8.3-2.

Table 8.3-1. Endpoints and Studies Used to Calculate Total Monetized Health Benefits^a

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature mortality — ACS cohort study,	PM _{2.5}	Pope et al. (2002) ¹¹	>29 years
all-cause Premature mortality — all-cause	PM _{2.5}	Woodruff et al. (1997) ¹²	Infant (<1 year)
Chronic Illness			
Chronic bronchitis	PM _{2.5}	Abbey et al. (1995) ¹³	>26 years
Nonfatal heart attacks	PM _{2.5}	Peters et al. (2001) ¹⁴	Adults
Hospital Admissions			
Respiratory	PM _{2.5}	Pooled estimate: Moolgavkar (2003) ¹⁵ —ICD 490-496 (COPD) Ito (2003) ¹⁶ —ICD 490-496 (COPD)	>64 years
Cardiovascular	$\begin{array}{c} PM_{2.5} \\ PM_{2.5} \\ PM_{2.5} \\ PM_{2.5} \\ PM_{2.5} \end{array}$	Moolgavkar (2000) ¹⁷ —ICD 490-496 (COPD) Ito (2003)—ICD 480-486 (pneumonia) Sheppard (2003) ¹⁸ —ICD 493 (asthma) Pooled estimate: Moolgavkar (2003)—ICD 390-429 (all cardiovascular) Ito (2003)—ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	20–64 years >64 years <65 years >64 years
Asthma-related ER visits	PM _{2.5} PM _{2.5}	Moolgavkar (2000)—ICD 390-429 (all cardiovascular) Norris et al. (1999) ¹⁹	20–64 years 0–18 years
Other Health Endpoints			
Acute bronchitis	PM _{2.5}	Dockery et al. (1996) ²⁰	8–12 years
Upper respiratory symptoms	PM _{2.5}	Pope et al. (1991) ²¹	Asthmatics, 9– 11 years
Lower respiratory symptoms	PM _{2.5}	Schwartz and Neas (2000) ²²	7–14 years
Asthma exacerbations	PM _{2.5}	Pooled estimate: Ostro et al. (2001) ²³ (cough, wheeze and shortness of breath) Vedal et al. (1998) ²⁴ (cough)	6–18 years ^b
Work loss days	PM _{2.5}	Ostro (1987) ²⁵	18–65 years
MRADs	PM _{2.5}	Ostro and Rothschild (1989) ²⁶	18–65 years

The endpoints and studies used for the primary estimate of benefits associated with the proposed rule have been subject to external technical guidance and review, including the Health Effects Subgroup (HES) of the EPA's Science Advisory Board (SAB) and the Office of Management and Budget (OMB).

The original study populations were 8 to 12 feet to 0 to 12 feet t

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

Table 8.3-2. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)^a

	Central Estimate of Value Per Statistical Incidence			
Health Endpoint	1990 Income Level	2020 Income Level ^b	2030 Income Level ^b	Derivation of Estimates
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 $(2002)^{27}$ meta-analysis and \$10 million represents the upper end of the interquartile range from the Viscusi and Aldy $(2003)^{28}$ 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] ²⁹) 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) 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 25–44 Age 45–54	\$66,902 \$74,676 \$78,834 \$140,649 \$66,902 \$65,293 \$73,149 \$76,871	\$66,902 \$74,676 \$78,834 \$140,649 \$66,902 \$65,293 \$73,149 \$76,871	\$66,902 \$74,676 \$78,834 \$140,649 \$66,902 \$65,293 \$73,149 \$76,871	Age-specific cost-of-illness values reflect lost earnings and direct medical costs over a 5-year period following a nonfatal MI. Lost earnings estimates are based on Cropper and Krupnick (1990). Direct medical costs are based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990). Lost earnings: Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings: age of onset: at 3% at 7% at 7% at 7% at 37,855 at 5-54 at 2,932 at 11,578 at 55-65 at 374,746 at 366,920 at 11,578 before the discounted expenses: An average of:
Age 55–65 Age 66 and over	\$132,214 \$65,293	\$132,214 \$65,293	\$132,214 \$65,293	1. Wittels et al. (1990) (\$102,658—no discounting) 2. Russell et al. (1998), 5-year period (\$22,331 at 3% discount rate; \$21,113 at 7% discount rate)

(continued)

Table 8.3-2. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)^a (continued)

	Central Estimate of Value Per Statistical Incidence			
Health Endpoint	1990 Income Level	2020 Income Level ^b	2030 Income Level ^b	Derivation of Estimates
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) ³³ (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) ³⁴ and (2) \$260.67, from Stanford et al. (1999). ³⁵

(continued)

Table 8.3-2. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)^a (continued)

	Central Estimate of Value Per Statistical Incidence			
Health Endpoint	1990 Income Level	2020 Income Level ^b	2030 Income Level ^b	Derivation of Estimates
Respiratory Ailments Not Requiring I	Hospitalization			
Upper Respiratory Symptoms (URS)	\$25	\$27	\$27	Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) ³⁶ 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 seven different types of URS.
Lower Respiratory Symptoms (LRS)	\$16	\$17	\$17	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS.
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). ³⁷ 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). ³⁸

(continued)

Table 8.3-2. Unit Values Used for Economic Valuation of Health Endpoints (2000\$)^a (continued)

	Central Estimate	e of Value Per Sta	tistical Incidence		
Health Endpoint	1990 Income Level	2020 Income Level ^b	2030 Income Level ^b	Derivation of Estimates	
Restricted Activity and Work/School	Loss Days				
Work Loss Days (WLDs)	Variable (national median =)			County-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.	
Minor Restricted Activity Days (MRADs)	\$51	\$54	\$55	Median WTP estimate to avoid one MRAD from Tolley et al. (1986). ³⁹	

^a Although the unit values presented in this table are in year 2000 dollars, all monetized annual benefit estimates associated with the proposed standards have been inflated to reflect values in year 2005 dollars. We use the Consumer Price Indexes to adjust both WTP- and COI-based benefits estimates to 2005 dollars from 2000 dollars. ⁴⁰ For WTP-based estimates, we use an inflation factor of 1.13 based on the CPI-U for "all items." For COI-based estimates, we use an inflation factor of 1.24 based on the CPI-U for medical care.

^b 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. Benefits are therefore adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor to account for income growth over time. For a complete discussion of how these adjustment factors were derived, we refer the reader to Chapter 9 of the CAND regulatory impact analysis (EPA, 2004). Note that similar adjustments do not exist for cost-of-illness-based unit values. For these, we apply the same unit value regardless of the future year of analysis.

EPA typically estimates the welfare impacts of effects such as changes in recreational visibility (related to reductions in ambient PM) and agricultural productivity (related to reductions in ambient ozone) in its RIAs of air quality policy. For the analysis of the proposed standards, however, we are unable to quantitatively characterize these impacts because of limited data availability; we are not quantifying ozone benefits related to the proposed standards and the PM scaling approach does not provide the spatial detail necessary to attribute specific air quality improvements to specific areas of visual interest (Class I areas). Instead, we discuss these welfare effects qualitatively in Section 8.5 of this chapter. We also qualitatively describe the impacts of other environmental and ecological effects for which we do not have an economic value.

8.4 Benefits Analysis Results for the Proposed Standards

Applying the impact and valuation functions described previously in this chapter to the estimated changes in PM_{2.5} associated with the proposed standards results in estimates of the changes in physical damages (e.g., premature mortalities, cases, admissions) and the associated monetary values for those changes. Estimates of physical health impacts are presented in Table 8.4-1. Monetized values for those health endpoints are presented in Table 8.4-2, along with total aggregate monetized benefits. All of the monetary benefits are in constant-year 2005 dollars.

Table 8.4-1. Estimated Reduction in Incidence of Adverse Health Effects Related to the Proposed Standards^a

	2020	2030
Health Effect	Incidence	Reduction
PM-Related Endpoints		
Premature Mortality ^{b,c} Adult, age 30+ and Infant, age <1 year	290	450
Chronic bronchitis (adult, age 26 and over)	200	290
Nonfatal myocardial infarction (adults, age 18 and older)	490	800
Hospital admissions—respiratory (all ages) ^d	160	270
Hospital admissions—cardiovascular (adults, age >18) ^e	130	200
Emergency room visits for asthma (age 18 years and younger)	210	310
Acute bronchitis (children, age 8–12)	470	700
Lower respiratory symptoms (children, age 7–14)	5,600	8,300
Upper respiratory symptoms (asthmatic children, age 9-18)	4,300	6,300
Asthma exacerbation (asthmatic children, age 6-18)	7,000	10,000
Work loss days (adults, age 18–65)	38,000	52,000
Minor restricted-activity days (adults, age 18–65)	220,000	310,000

^a Incidences are rounded to two significant digits. PM estimates are nationwide.

^b PM premature mortality impacts for adults are based on application of the effect estimate derived from the ACS cohort study (Pope et al., 2002). ⁴¹ Infant premature mortality based upon studies by Woodruff, et al 1997. ⁴²

^c PM-related mortality benefits estimated using an assumed PM threshold at background levels (3 μg/m³). There is uncertainty about which threshold to use and this may impact the magnitude of the total benefits estimate. For a more detailed discussion of this issue, please refer to Section 8.6.2.2 of the RIA.

d Respiratory hospital admissions for PM include admissions for COPD, pneumonia, and asthma.

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

Table 8.4-2. Estimated Monetary Value in Reductions in Incidence of Health and Welfare Effects (in millions of 2005\$)^{a,b}

	2020	2030
PM-Related Health Effect	Estimated Value of Reductions	
Premature mortality ^{c,d,e}		
Adult, age 30+ and Infant, < 1 year		
3% discount rate	\$2,000	\$3,100
7% discount rate	\$1,800	\$2,800
Chronic bronchitis (adults, 26 and over)	\$94	\$140
Non-fatal acute myocardial infarctions		
3% discount rate	\$50	\$77
7% discount rate	\$48	\$75
Hospital admissions for respiratory causes	\$2.9	\$5.0
Hospital admissions for cardiovascular causes	\$3.1	\$4.7
Emergency room visits for asthma	\$0.07	\$0.11
Acute bronchitis (children, age 8–12)	\$0.20	\$0.30
Lower respiratory symptoms (children, 7–14)	\$0.11	\$0.16
Upper respiratory symptoms (asthma, 9–11)	\$0.13	\$0.19
Asthma exacerbations	\$0.36	\$0.54
Work loss days	\$5.8	\$7.0
Minor restricted-activity days (MRADs)	\$14	\$19
Monetized Total ^f		
Base Estimate:		
3% discount rate	\$2,100+ B	\$3,400+ B
7% discount rate	\$1,900+ B	\$3,100+ B

Monetary benefits are rounded to two significant digits for ease of presentation and computation. PM benefits are nationwide.

In addition to omitted benefits categories such as air toxics, ozone, and various welfare effects, not all known PM-related health and welfare effects could be quantified or monetized. Furthermore, we did not quantify reductions in secondary PM_{2.5} and the associated health and welfare effects. The monetized value of all of these unquantified effects is represented by adding an unknown "B" to the aggregate total. The estimate of total monetized health benefits of the proposed control package is thus equal to the subset of monetized PM-related health benefits plus B, the sum of the nonmonetized health and welfare benefits.

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

^c PM-related mortality benefits estimated using an assumed PM threshold at background levels (3 μg/m³). There is uncertainty about which threshold to use and this may impact the magnitude of the total benefits estimate. For a more detailed discussion of this issue, please refer to Section 8.6.2.2 of the RIA.

Valuation assumes discounting over the SAB recommended 20-year segmented lag structure described earlier. Results reflect the use of 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003). 43,44

^e Adult premature mortality estimates based upon the ACS cohort study (Pope et al., 2002). ⁴⁵ Infant premature mortality based upon Woodruff et al 1997. ⁴⁶

B represents the monetary value of health and welfare benefits and disbenefits not monetized. A detailed listing is provided in Table 8.1-2.

Total monetized benefits are dominated by benefits of mortality risk reductions. The primary estimate projects that the proposed standards will result in 290 avoided premature deaths annually in 2020 and 450 avoided premature deaths annually in 2030. The increase in annual benefits from 2020 to 2030 reflects additional emission reductions from the proposed standards, as well as increases in total population and the average age (and thus baseline mortality risk) of the population.

Our estimate of total monetized benefits in 2020 for the proposed standards is \$2.1 billion using a three percent discount rate and \$1.9 billion using a seven percent discount rate. In 2030, the monetized benefits are estimated at \$3.4 billion using a three percent discount rate and \$3.1 billion using a seven percent discount rate. The monetized benefit associated with reductions in the risk of premature mortality, which accounts for \$2.0 billion in 2020 and \$3.1 billion in 2030 (assuming a three percent discount rate), is over 90 percent of total monetized health benefits. The next largest benefit is for reductions in chronic illness (CB and nonfatal heart attacks), although this value is more than an order of magnitude lower than for premature mortality. Hospital admissions for respiratory and cardiovascular causes, minor restricted activity days, and work loss days account for the majority of the remaining benefits. The remaining categories each account for a small percentage of total benefit; 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 over 100 times more work loss days than premature mortalities, yet work loss days account for only a very small fraction of total monetized benefits. This reflects the fact that many of the less severe health effects, while more common, are valued at a lower level than the more severe health effects. Also, some effects, such as hospital admissions, are valued using a proxy measure of willingness-to-pay (e.g., cost-of-illness). H As such, the full value of these effects may be higher than that reported in Table 8.4-2.

8.5 Unquantified Health and Welfare Effects

In considering the monetized benefits estimates, the reader should remain aware of the many limitations of conducting the analyses mentioned throughout this RIA. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the effects listed in Table 8.1-2. For many health and welfare effects, such as changes in health effects due to reductions in air toxics exposure, changes in ecosystem functions and PM-related materials damage, reliable impact functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefit categories, the benefits estimates presented in this analysis would increase, although the magnitude of such an increase is highly uncertain.

Other welfare effects that EPA has monetized in past RIAs, such as recreational visibility, are omitted from the current analysis. Due to time and resource constraints, we did not

-

^H See Table 12.3-2 for a description of how each particular endpoint is valued.

run the full-scale PM air quality modeling needed to estimate this benefit category. Instead, we relied on the PM scaling benefits transfer approach that provides analytical efficiency but sacrifices the full range of outputs typically generated when models such as the Community Multiscale Air Quality (CMAQ) model or the Regional Modeling System for Aerosols and Deposition (REMSAD) are run.

Unquantified benefits are qualitatively discussed in the following health and welfare effects sections. In addition to unquantified benefits, there may also be environmental costs (disbenefits) that we are unable to quantify, which we qualitatively discuss 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. Although we are not currently able to estimate the magnitude of these unquantified and unmonetized benefits, specific categories merit further discussion. EPA believes, however, the unquantified benefits associated with health and non-health benefit categories are likely significant and that their omission lends a downward bias to the monetized benefits presented in this analysis.

8.5.1 Human Health Impact Assessment

In addition to the $PM_{2.5}$ health effects discussed above, there is emerging evidence that human exposure to PM may be associated a number of health effects not quantified in this analysis (see Table 8.1-2). An improvement in ambient $PM_{2.5}$ concentrations may reduce the number of incidences within each of these unquantified effect categories that the U.S. population would experience. Although these health effects are believed to be PM-induced, effect estimates are not available for quantifying the benefits associated with reducing these effects. Furthermore, the health effects associated with reductions in air toxics are not quantified in this analysis.

The proposed standards will also reduce the national emissions inventory of precursors to ozone, such as VOCs. Exposure to ozone has been linked to a variety of respiratory effects including hospital admissions, emergency room visits, minor restricted activity days, worker productivity and illnesses resulting in school absences. Emerging evidence has also shown that human exposure to ozone may be associated with a number of other health effects not quantified in this analysis (see Table 8.1-2). Ozone can also adversely affect the agricultural and forestry sectors by decreasing yields of crops and forests. Although ozone benefits are typically quantified in regulatory impact analyses, we have chosen not to evaluate them for this analysis. As discussed in Chapter 2, the ozone modeling conducted for the proposed standards results in a net reduction in ambient concentrations of ozone in 2020 and 2030. By omitting ozone benefits in this proposal, we acknowledge that this analysis underestimates the benefits associated with the proposed standards.

8.5.2 Welfare Impact Assessment

For many welfare effects, such as changes in ecosystem functions and PM-related materials damage, reliable impact functions and/or valuation functions are not currently

available. In general, if it were possible to monetize these benefit categories, the benefits estimates presented in this analysis would increase, although the magnitude of such an increase is highly uncertain.

8.5.2.1 Visibility Benefits

Changes in the level of ambient $PM_{2.5}$ caused by the proposed standards will change the level of visibility in much of the United States. 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 Great Smoky Mountains National Park. Though not quantified in this analysis, the value of improvements in visibility monetized for regulatory analyses such as the final CAIR are significant. We refer the reader to that analysis for a complete description of the methods used to value visibility.⁴⁷

8.5.2.2 gricultural and Forestry 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" (EPA, 1996, page 5-11). ⁴⁸ Though we do not quantify the potential improvements in ambient ozone concentrations associated with the proposed standards, it is possible that yields will improve in areas of agricultural or forestry production impacted by the standards.

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. Because of resource limitations, we are unable to provide agricultural or forestry benefits estimates for the proposed standards.

8.5.2.2.1 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 United States." In addition, economic studies have shown a relationship between observed ozone levels and crop yields. 49

8.5.2.2.2 Forestry Benefits

Ozone also has been shown conclusively to cause discernible injury to forest trees (EPA,

1996; Fox and Mickler, 1996).^{54,50} In our previous analysis of the Heavy-Duty 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.

8.5.2.3 Benefits from Reductions in Materials Damage

The proposed standards 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. PM also has 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 benefits 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 1970s) are too out of date to provide a reliable estimate of current household soiling damages (EPA-SAB-COUNCIL-ADV-98-003, 1998). 51

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)⁵² indicate that these benefits could be an order of magnitude larger than household soiling benefits.

8.5.3 UVb Exposure

In contrast to the unquantified benefits of the proposed standards discussed above, it is also possible that this rule will result in disbenefits in some areas of the United States. The effects of ozone and PM on radiative transfer in the atmosphere can lead to effects of uncertain magnitude and direction on the penetration of ultraviolet light and climate. Ground level ozone makes up a small percentage of total atmospheric ozone (including the stratospheric layer) that attenuates penetration of ultraviolet - b (UVb) radiation to the ground. EPA's past evaluation of the information indicates that potential disbenefits would be small, variable, and with too many uncertainties to attempt quantification of relatively small changes in average ozone levels over the course of a year. EPA's most recent provisional assessment of the currently available information indicates that potential but unquantifiable benefits may also arise from ozone-related attenuation of UVb radiation. EPA believes that we are unable to quantify any net climate-related disbenefit or benefit associated with the combined ozone and PM reductions in this rule.

8.6 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. This analysis is no exception. As outlined both in this and preceding chapters, many inputs were used to derive the benefits estimate, including

emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, estimates of values (both from WTP and COI 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 its role in the benefits analysis, may have a disproportionately large impact on 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. Some of the key uncertainties in the quantified benefits analysis are presented in Table 8.6-1.

Table 8.6-1. Primary Sources of Uncertainty in the Quantified Benefits Analysis

- 1. Uncertainties Associated with Impact Functions
- The value of the PM effect estimate in each impact function.
- Application of a single impact function to pollutant changes and populations in all locations.
- Similarity of future-year impact functions to current impact functions.
- Correct functional form of each impact function.
- Extrapolation of effect estimates beyond the range of PM concentrations observed in the source epidemiological study.
- Application of some impact functions only to those subpopulations matching the original study population.
- 2. Uncertainties Associated with PM Concentrations
- Responsiveness of the models to changes in precursor emissions resulting from the control policy.
- Projections of future levels of precursor emissions, especially organic carbonaceous particle emissions.
- Model chemistry for the formation of ambient nitrate concentrations.
- Lack of speciation monitors in some areas requires extrapolation of observed speciation data.
- CMAQ model performance in the Western U.S., especially California indicates significant underprediction of PM_{2.5}.
- 3. Uncertainties Associated with PM Mortality Risk
- Differential toxicity of specific component species within the complex mixture of PM has not been determined.
- 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.
- 5. Uncertainties Associated with Possible Lagged Effects
- The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels that would occur in a single year is uncertain as well as the portion that might occur in subsequent years.

- 6. Uncertainties Associated with Baseline Incidence Rates
- Some baseline incidence rates are not location specific (e.g., those taken from studies) and therefore may not accurately represent the actual location-specific rates.
- Current baseline incidence rates may not approximate well baseline incidence rates in 2020 and 2030.
- Projected population and demographics may not represent well future-year population and demographics.
- 7. 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 because of differences in income or other factors.
- 8. Uncertainties Associated with Aggregation of Monetized Benefits
- Health and welfare benefits estimates are limited to the available impact functions. Thus, unquantified or unmonetized benefits are not included.

As part of EPA's approach to characterizing uncertainties in the benefits assessment, we generate a probabilistic estimate of statistical uncertainty based on standard errors reported in the underlying studies used in the benefits modeling framework, with particular emphasis on the health impact functions. Using a Monte Carlo procedure, the distribution of each health endpoint and its unit dollar value is characterized by the reported mean and standard error derived from the epidemiology and valuation literature. Details on the distributions used to value individual health endpoints are provided in Section 8.6.1, as well as in the CAIR RIA (Appendix B; EPA, 2005). It should be noted that the Monte Carlo-generated distributions of benefits reflect only some of the uncertainties in the input parameters (described in Table 8.6-1). Uncertainties associated with emissions, air quality modeling, populations, and baseline health effect incidence rates are not represented in the distributions of benefits of attaining alternative standards. Issues such as correlation between input parameters and the identification of reasonable upper and lower bounds for input distributions characterizing uncertainty in additional model elements will be addressed in future versions of the uncertainty framework.

In benefit analyses of air pollution regulations conducted to date, the estimated impact of reductions in premature mortality has accounted for 85% to 95% of total benefits. Therefore, in characterizing the uncertainty related to the estimates of total benefits it is particularly important to attempt to characterize the uncertainties associated with this endpoint. As such, we specifically discuss the uncertainty related to PM-related premature mortality in Section 8.6.2.

8.6.1 Analysis of Statistical Uncertainty

For the proposed standards, we did not attempt to assign probabilities to all of the uncertain parameters in the model because of 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. For all quantified PM endpoints, we scaled the likelihood distributions of the benefit estimates from the CAND uncertainty analysis, based on the same

¹ U.S. Environmental Protection Agency. May 2004. *Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/nonroad-diesel/2004fr.htm#documents. Accessed December 15, 2005.

benefits transfer approach we used to estimate the benefits of the standards presented in Section 8.2. The CAND likelihood distributions were based solely on the statistical uncertainty surrounding the estimated C-R functions and the assumed distributions around the unit values. We use the benefits transfer approach to scale those distributions to reflect the predicted PM precursor emission reductions of the proposed standards. Though the scaling approach adds another element of uncertainty that we cannot characterize in the distributions, we believe the scaled uncertainty is a reasonable approximation of the statistical uncertainty based on standard errors reported in the underlying epidemiological and valuation studies.

Our scaled estimates of the likelihood distributions for health-related PM benefits should be viewed as incomplete because of the wide range of sources of uncertainty that we have not incorporated. The 5th and 95th percentile points of our scaled estimate are based on statistical error, and cross-study variability provides some insight into how uncertain our estimate is with regard to those sources of uncertainty. However, it does not capture other sources of uncertainty regarding the benefits transfer scaling approach or the inputs to the CAND modeling upon which the scaling is based, including emissions, air quality, baseline population incidence, and projected exposures. It also does not account for 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. Thus, a likelihood description based on the standard error would provide a misleading picture about the overall uncertainty in the estimates.

Both the uncertainty about incidence changes¹ 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. Unlike a sampling distribution (which describes the possible values that an 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 is typically expressed as the interval from the 5th percentile point of the likelihood distribution to the 95th 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.

_

^J 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: 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 uncertainty about how well any given pollutant coefficient approximates β^* .

^K 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. A Guide to Econometrics. 2nd ed. MIT Press: Cambridge, MA., pp. 168-172).

8.6.1.1 Monte Carlo Approach

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, another value is randomly drawn from the unit dollar value distribution; the total dollar benefit for that iteration is the product of the two. When 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 can be generated for each endpoint. As the number of Monte Carlo draws gets larger and larger, the Monte Carlogenerated distribution becomes a better and better approximation of a joint likelihood distribution (for the considered parameters) making up the 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 (nonoverlapping) 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.

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 X1, then the mean of the distribution, E(X1) and the variance of X1, denoted Var(X1), and the 5th and 95th percentile points of the distribution (related to Var(X1)), 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 X2, the benefits from reductions in the incidence of both types of hospital admissions is X1 + X2. This variable has a distribution with mean E(X1 + X2) = E(X1) + E(X2), and a variance of Var(X1 + X2) = E(X1) + E(X2).

-

^L This method assumes that the incidence change and the unit dollar value for an endpoint are stochastically independent.

X2) = Var(X1) + Var(X2) + 2Cov(X1,X2); if X1 and X2 are stochastically independent, then it has a variance of Var(X1 + X2) = Var(X1) + Var(X2), and the covariance term is zero.

The benefits from reductions in all nonoverlapping PM-related health and welfare endpoints are (Xm+1, ..., Xn) is X = X1 + ... + Xn. The mean of the distribution of total benefits, X, is

$$E(X) = E(X1) + E(X2) + ... + E(Xn)$$

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

$$Var(X) = Var(X1) + Var(X2) + ... + Var(Xn)$$

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

$$E(X1) < E(X1 + X2) < E(X1 + X2 + X3) < ... < E(X1 + ... + Xn) = E(X)$$

$$Var(X1) < Var(X1 + X2) < Var(X1 + X2 + X3) < ... < Var(X1 + ... + Xn) = Var(X)$$

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, X1, X2, through Xn; 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 5th 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 5th percentile. Although the sum of the 5th 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.

8.6.1.2 Monte Carlo Results

Based on the Monte Carlo techniques and benefits transfer methods described above, we scaled the CAND likelihood distributions for the dollar value of total PM health-related benefits for the proposed standards. For this analysis, the likelihood descriptions for the true value of each of the health endpoint incidence estimates, including premature mortality, were based on classical statistical uncertainty measures. The measures include the mean and standard deviation of the C-R relationships in the epidemiological literature, and assumptions of particular likelihood distribution shapes for the valuation of each health endpoint value based on reported values in the economic literature. 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. Distributions for unit dollar values are summarized in Table 8.3-2.

Results of the scaled Monte Carlo simulations are presented in Table 8.6-2. The table provides the scaled 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 close to 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 8.6-2. Distribution of Value of Annual PM-Related Human Health Benefits in 2030 for the Proposed Standards $^{\rm a}$

Endpoint	Monetary Benefits ^{b, c} (Millions 2003\$, Adjusted for Income Growth)		
	5 th Percentile	Mean	95 th Percentile
Premature mortality ^c , Long-term exposure			
Adults, 30+ yrs and Infants, <1yr			
3% Discount Rate	\$750	\$3,100	\$6,200
7% Discount Rate	\$680	\$2,800	\$5,600
Chronic bronchitis (adults, 26 and over)	\$7.0	\$140	\$480
Nonfatal myocardial infarctions			
3% Discount Rate	\$18	\$77	\$180
7% Discount Rate	\$17	\$75	\$180
Hospital admissions from respiratory causes	\$1.6	\$5.0	\$8.0
Hospital admissions from cardiovascular causes	\$2.8	\$4.7	\$7.0
Emergency room visits for asthma	\$0.07	\$0.11	\$0.16
Acute bronchitis (children, aged 8-12)	\$0	\$0.30	\$0.70
Lower respiratory symptoms (children, aged 7-14)	\$0.06	\$0.16	\$0.29
Upper respiratory symptoms (asthmatic children,			
aged 9–11)	\$0.05	\$0.19	\$0.42
Asthma exacerbations	\$0.01	\$0.54	\$1.5
Work loss days (adults, aged 18-65)	\$6	\$7	\$8
Minor restricted-activity days (adults, aged 18-65)	\$11	\$19	\$27
Monetized Total ^d			
3% Discount Rate	800 + B	\$3,400 + B	\$7,000 + B
7% Discount Rate	720 + B	\$3,100 + B	\$6,300 + B

^a Monetary benefits are rounded to two significant digits.

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

Results show 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).

B represents the monetary value of the nonmonetized health and welfare benefits. A detailed listing of unquantified PM-, ozone-, and air toxics-related health effects is provided in Table 8.1-2.

8.6.2 Additional Approaches to Characterizing Uncertainty Related to PM-Mortality

As part of an overall program to improve the Agency's characterization of uncertainties in health benefits analyses, we attempt to address uncertainties associated with the $PM_{2.5}$ mortality health impact function relationship and valuation. Use of the ACS cohort (Pope et al., 2002) mortality function to support this analysis does not address uncertainty associated with: (a) potential of the study to incompletely capture short-term exposure-related mortality effects, (b) potential mis-match between study and analysis populations which introduces various forms of bias into the results, (c) failure to identify all key confounders and effects modifiers, which could result in incorrect effects estimates relating mortality to $PM_{2.5}$ exposure, and (d) model uncertainty. EPA is researching methods to characterize all elements of uncertainty in the doseresponse function for mortality.

As is discussed in detail in the final PM NAAQS RIA, EPA uses three methods to quantify uncertainties in the mortality function, including: the statistical uncertainty derived from the standard errors reported in the ACS cohort study, the presentation of additional estimates of mortality based upon the peer-reviewed literature, and the use of results of an expert elicitation conducted to explore a more thorough characterization of uncertainties in the mortality estimate. Because this analysis utilizes the PM scaling benefits transfer approach to estimate mortality incidence for the proposed standards, we cannot quantify the PM mortality uncertainty to the same extent as was done for the CAIR or PM NAAQS analyses. However, in a similar fashion to the analysis conducted for the Clean Air Visibility Rule (CAVR), ⁵⁶ we can scale the results of the CAND mortality uncertainty analysis to the PM precursor emission changes modeled for the proposed standards.

8.6.2.1 Uncertainty Associated with the Concentration-Response Function

In the benefit analysis of the CAND 2030 emission control standards, the statistical uncertainty represented by the standard error of the American Cancer Society cohort study (Pope et al, 2002) was one and one-half times the mean benefit estimate at the 95th percentile and less than one-half of the mean at the 5th percentile. The CAND analysis also derived mortality from the reanalysis of the Harvard Six-Cities study (Krewski et al., 2000).⁵⁷ At the time of the CAND analysis, EPA's Science Advisory Board provided guidance stating, "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).⁵⁸ In the CAND analysis, the Harvard Six-Cities mean benefits estimate was over twice the size of the mean estimate of mortality benefits derived from the ACS study.

Recently, a new peer-reviewed extension of the Six-Cities study has been published (Laden et al., 2006). This follow-up to the Harvard Six-Cities study both confirmed the effect size from the first analysis and provided additional evidence that reductions in $PM_{2.5}$ are likely associations with reductions in the risk of premature death. This additional evidence stems from

the observed reductions in PM_{2.5} in each city during the extended follow-up period. Laden et al. (2006) found that mortality rates consistently went down at a rate proportionate to the observed reductions in PM_{2.5}. In the recently finalized PM NAAQS RIA, results from this study were presented as an additional estimate of premature mortality benefits along with the benefits derived from the ACS study. The mean benefits estimate derived from the Six-Cities study was more than twice the size of the mean estimate of mortality benefits derived from the ACS study. Because this study was not available during the CAND analysis, from which the benefits of the proposed standards are scaled, we are unable to provide an estimate of mortality benefits based on the Six-Cities study for this proposed analysis. However, based on the relationship between the Six-Cities study and the ACS cohort study observed in the final PM NAAQS RIA, we can surmise that the mean estimate of PM-related mortality associated with the proposed standards could be approximately twice as large. For a full discussion of the epidemiological basis of EPA's premature mortality estimates, we refer the reader to Chapter 5.1 of the final PM NAAQS RIA.

EPA recently completed a full-scale expert elicitation that incorporated peer-review comments on the pilot application used in CAND, and that provides a more robust characterization of the uncertainty in the premature mortality function. This expert elicitation was designed to evaluate uncertainty in the underlying causal relationship, the form of the mortality impact function (e.g., threshold versus linear models) and the fit of a specific model to the data (e.g., confidence bounds for specific percentiles of the mortality effect estimates). Additional issues, such as the ability of long-term cohort studies to capture premature mortality resulting from short-term peak PM exposures, were also addressed in the expert elicitation. The recently published RIA supporting the Particulate Matter National Ambient Air Quality Standards (PM NAAQS) used the results of this expert elicitation to quantitatively characterize uncertainty.

Due to the analytical constraints associated with the PM benefits scaling approach, we are unable to assess the premature mortality health impacts derived from the formally elicited expert judgments. Compared to the final PM NAAQS estimate of mean premature mortality derived from the ACS cohort study, however, expert-based mortality incidence ranged from approximately 50 percent of the mean ACS estimate to approximately five times the size of the mean ACS estimate. In total, PM-related premature mortality derived from eleven of the experts was greater than the ACS estimate, while one expert-based estimate fell below the ACS result.

8.6.2.2 PM_{2.5}-Mortality Cutpoint/Threshold Analysis

Another source of uncertainty that has received recent attention from several scientific review panels is the shape of the concentration-response function for PM-related mortality, and specifically whether there exists a threshold below which there would be no benefit to further reductions in PM_{2.5}. The consistent advice from EPA's SAB^M has been to model premature

_

^M The advice from the 2004 SAB-HES (EPA-SAB-COUNCIL-ADV-04-002)⁶⁹ is characterized by the following:

[&]quot;For the studies of long-term exposure, the HES notes that Krewski et al. (2000) have conducted the most careful

mortality associated with PM exposure as a nonthreshold effect, that is, with harmful effects to exposed populations regardless of the absolute level of ambient PM concentrations. However, EPA's most recent PM_{2.5} Criteria Document concludes that "the available evidence does not either support or refute the existence of thresholds for the effects of PM on mortality across the range of concentrations in the studies." Some researchers have hypothesized the presence of a threshold relationship. That is, the hypothesized relationship includes the possibility that there exists a PM concentration level below which further reductions no longer yield premature mortality reduction benefits.

To consider the impact of a threshold in the response function for the chronic mortality endpoint, the final PM NAAQS RIA⁶¹ constructed a sensitivity analysis by assigning different cutpoints below which changes in PM_{2.5} are assumed to have no impact on premature mortality. In applying the cutpoints, the PM NAAOS analysis adjusted the mortality function slopes accordingly. Five cutpoints (including the base case assumption) were included in the sensitivity analysis: (a) 14 µg/m³ (assumes no impacts below a level being considered at the time for the annual PM_{2.5} NAAQS), (b) 12 μg/m³ (c) 10 μg/m³ (reflects comments from CASAC, 2005), ⁶² (d) 7.5 µg/m³ (reflects recommendations from SAB-HES to consider estimating mortality benefits down to the lowest exposure levels considered in the ACS cohort study (Pope et al., 2002) used as the basis for modeling chronic mortality) ⁶³ and (e) background or $3 \mu g/m^3$ (reflects NRC recommendation to consider effects all the way to background). 64 The results of the sensitivity analysis displayed the change in avoided mortality cases and associated monetary benefits associated with the alternative cutpoints (see the final PM NAAQS RIA, Chapter 5.1 and Tables 5-28 to 5-31).

A sensitivity analysis such as this can be difficult to interpret, because when a threshold above the lowest observed level of PM_{2.5} in the underlying ACS cohort study (Pope et al., 2002) is assumed, the slope of the concentration-response function above that level must be adjusted upwards to account for the assumed threshold. Depending on the amount of slope adjustment and the proportion of the population exposed above the assumed threshold, the estimated mortality impact can either be lower (if most of the exposures occur below the threshold) or higher (if most of the exposures occur above the threshold). To demonstrate this, we present an example from the proposed PM NAAQS RIA. In its examination of the benefits of attaining alternative PM NAAQS in Chicago, the analysis found that, because annual mean levels are generally higher in Chicago, there was a two-part pattern to the relationship between assumed

work on this issue. They report that the associations between PM_{2.5} 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."

^N Note that the PM NAAOS analysis only adjusted the mortality slopes for the 10 μg/m³, 12 μg/m³ and 14 μg/m³ cutpoints since the 7.5 µg/m³ and background cutpoints were at or below the lowest measured exposure levels reported in the Pope et al. (2002) study for the combined exposure dataset.

O See NAS (2002)⁷¹ and CASAC (2005)⁶⁸ for discussions of this issue.
P See the proposed PM NAAQS RIA (2005),⁶⁷ Appendix A, pp. A63-A64.

threshold and mortality impacts. As the threshold increased from background to $7.5 \,\mu\text{g/m}^3$, the mortality impact fell (because there is no slope adjustment). However, at an assumed threshold of $10 \,\mu\text{g/m}^3$, estimated mortality impacts actually increased, because the populations exposed above $10 \,\mu\text{g/m}^3$ were assumed to have a larger response to particulate matter reductions (due to the increased slope above the assumed threshold). And finally, mortality impacts again fell to zero if a $15 \,\mu\text{g/m}^3$ threshold was assumed, because these impacts were measured incremental to attainment of the current standard.

We are unable to do this type of sensitivity analysis for the analysis of the proposed standards because of the analytical limitations of the PM benefits scaling procedure. When EPA conducted the CAND analysis (from which the primary estimates of benefits for the proposed standards are based), there were no PM mortality concentration-response functions with the slope adjusted upwards to account for an assumed threshold. Instead, our primary PM benefits estimate for the proposed standards reflects a background threshold assumption of 3 μ g/m³. We present in Table 8.6-3 the results of our scaled PM-related mortality benefits in the context of its relationship to other cutpoints.

Table 8.6-3. PM-Related Mortality Benefits of the Proposed Standards: Cutpoint Sensitivity Analysis^a

Certainty that Benefits are At Least Specified Value	Level of Assumed Threshold	Discount Rate	PM Mortality Benefits (Billion 2003\$)	
			2020	2030
More Certain that Benefits Are at Least as Large	14 μg/m ^{3 c}	3% 7%	N/A	A^b
	$12 \mu\text{g/m}^3$	3% 7%	N/	A
	$10~\mu g/m^{3~d}$	3% 7%	N/	A
	$7.5 \mu g/m^3 e$	3% 7%	N/	A
Less Certain that Benefits Are at Least as Large	$3 \mu g/m^3 ^{\mathrm{f}}$	3% 7%	\$3.3 \$3.0	\$6.3 \$5.7

^a Note that this table only presents the effects of a cutpoint on PM-related mortality incidence and valuation estimates.

^b Not Available. We are unable to provide cutpoint analysis results for the proposed standards because of the analytical limitations of the PM benefits scaling procedure.

^c EPA intends to analyze a cutpoint between $12 \mu g/m^3$ and $15 \mu g/m^3$ for the final RIA.

^d CASAC (2005)⁶⁸

e SAB-HES (2004)⁶⁹

f NAS (2002)⁷¹

8.7 Health-Based Cost Effectiveness Analysis

Health-based cost-effectiveness analysis (CEA) and cost-utility analysis (CUA) have been used to analyze numerous health interventions but have not been widely adopted as tools to analyze environmental policies. The Office of Management and Budget (OMB) issued Circular A-4 guidance on regulatory analyses, requiring Federal agencies to "prepare a CEA for all major rulemakings for which the primary benefits are improved public health and safety to the extent that a valid effectiveness measure can be developed to represent expected health and safety outcomes." Environmental quality improvements may have multiple health and ecological benefits, making application of CEA more difficult and less straightforward. For the CAIR analysis, the first to incorporate an analysis of this kind, CEA provided a useful framework for evaluation: nonhealth benefits were substantial, but the majority of quantified benefits came from health effects. EPA included in the CAIR RIA a preliminary and experimental application of one type of CEA—a modified quality-adjusted life-years (QALYs) approach. For CAIR, EPA concluded that the direct usefulness of cost-effectiveness analysis is mitigated by the lack of rule alternatives to compare relative effectiveness, but that comparisons could still be made to other benchmarks bearing in mind methodological differences.

QALYs were developed to evaluate the effectiveness of individual medical treatments, and EPA is still evaluating the appropriate methods for CEA of environmental regulations. Agency concerns with the standard QALY methodology include the treatment of people with fewer years to live (the elderly); fairness to people with preexisting conditions that may lead to reduced life expectancy and reduced quality of life; and how the analysis should best account for nonhealth benefits, such as improved visibility.

The Institute of Medicine (a member institution of the National Academies of Science) established the Committee to Evaluate Measures of Health Benefits for Environmental, Health, and Safety Regulation to assess the scientific validity, ethical implications, and practical utility of a wide range of effectiveness measures used or proposed in CEA. This committee prepared a report titled "Valuing Health for Regulatory Cost-Effectiveness Analysis," which concluded that CEA is a useful tool for assessing regulatory interventions to promote human health and safety, although not sufficient for informed regulatory decisions (Miller, Robinson, and Lawrence, 2006). They emphasized the need for additional data and methodological improvements for CEA analyses, and urged greater consistency in the reporting of assumptions, data elements, and analytic methods. They also provided a number of recommendations for the conduct of regulatory CEA analyses. EPA is evaluating these recommendations and will determine a response for upcoming analyses.

In Appendix G of the RIA for the CAIR, ⁶³ EPA conducted an extensive cost-effectiveness analysis using morbidity inclusive life years (MILY). That analysis concluded that reductions in PM_{2.5} associated with CAIR were expected to be cost-saving (because the value of expenditures on illnesses and non-health benefits exceeded costs), and that costs of the CAIR could have been significantly higher and still result in cost-effective improvements in public

health. Because the current analysis relies on a benefits transfer approach to estimate PM-related benefits, scaling PM benefits from the CAND rule, we do not have the necessary inputs to develop a valid cost-effectiveness measure for the proposed standards. Furthermore, the CAND analysis did not include a health-based CEA, the results of which might have been scaled in a similar fashion to the benefits.

For the CAVR rule, EPA was able to draw inferences from the CAIR CEA by scaling the relative magnitude of the costs and health impacts between the two rules. ⁶⁶ While the CAVR was not expected to be cost-saving like CAIR, EPA expected that CAVR was likely to have a relatively low cost per MILY. For the proposed standards, however, it is difficult to draw similar inferences with CAIR because the geographic distribution of emission changes, the distribution of those changes over time, and the age distribution of the mortality and chronic disease reductions are all expected to differ between the two rules. For these reasons, we do not scale the CAIR health-based cost-effectiveness analysis for the proposed standards.

8.8 Comparison of Costs and Benefits

The proposed rule establishes separate standards that reduce the evaporative and exhaust emissions from small SI and marine SI engines. A full appreciation of the overall economic consequences of these provisions requires consideration of the benefits and costs expected to result from each standard. Due to limitations in data availability and analytical methods, however, we are only able to present the benefits of the entire proposed rule in the aggregate for both PM_{2.5} and ozone. There are also a number of health and environmental effects associated with the proposed standards that we were unable to quantify or monetize (see Table 8.1-2).

Table 8.8-1 contains the estimates of monetized benefits of the proposed standards and estimated social welfare costs for each of the proposed control programs. The annual social welfare costs of all provisions of this proposed rule are described more fully in Chapter 9. The results in Table 8.8-1 suggest that the 2020 and 2030 monetized benefits of the proposed standards are much greater than the expected social welfare costs. Specifically, the annual benefits of the program would be approximately \$2.1 + B billion annually in 2020 using a three percent discount rate (or \$1.9 + B billion using a seven percent discount rate), compared to estimated social welfare costs of approximately \$252 million in that same year. The net benefits are expected to increase to \$3.4 + B billion annually in 2030 using a three percent discount rate (or \$3.1 + B billion using a seven percent discount rate), even as the social welfare costs of that program fall to \$241 million.

In Table 8.8-1, we present the costs and PM-related benefits related to each of the two broad engine classes regulated by the proposed standards: Small SI and Marine SI engines. Table 8.8-1 also presents the costs and PM-related benefits related to the specific engine classes regulated by the proposed standards: Small SI – Class I, Class II, and Handheld (HH); Marine SI

-

^Q Social costs represent the welfare costs of the rule to society. These social costs do not consider transfer payments (such as taxes) that are simply redistributions of wealth.

– Sterndrive/Inboard (SD/I), and Outboard/Personal Water Craft (OB/PWC). Using the same PM scaling approach described in Section 8.2.2., we are able to split out the estimated PM benefits related to the different Small SI and Marine SI engine classes. One can see that in all cases, the PM benefits accrued by the engine classes are greater than the costs, even when fuel savings is not factored into the cost estimate. The benefit-to-cost ratio would be even greater if we estimated the ozone benefits related to the proposed standards.

Table 8.8-1. Summary of Annual Benefits and Costs of the Proposed Standards^a (Millions of 2005 dollars)

	(Millions of 2005 dollars) 2020 2030				
Description	(Millions of 2005	(Millions of 2005			
Description	dollars)	dollars)			
Estimated Social Welfare Costs ^{b,c}	uonars)	uonars)			
Small SI	\$351	\$404			
Class I	\$145	\$167			
Class II	\$143 \$199	\$107 \$229			
Class II HH ^d	\$199 \$7	\$229 \$8			
	•	·			
Marine SI	\$154	\$164			
SD/I	\$41	\$44			
OB/PWC	\$113	\$120			
Total	\$505	\$569			
Fuel Savings	\$(253)	\$(327)			
Total Social Welfare Costs	\$252	\$241			
Estimated Benefits ^{e,f}					
PM-Only Small SI Benefits					
3 percent discount rate	\$861	\$1,280			
7 percent discount rate	\$782	\$1,160			
Class I					
3 percent discount rate	\$478	\$647			
7 percent discount rate	\$434	\$587			
Class II					
3 percent discount rate	\$383	\$627			
7 percent discount rate	\$348	\$570			
PM-Only Marine SI Benefits					
3 percent discount rate	\$1,280	\$2,110			
7 percent discount rate	\$1,160	\$1,190			
SD/I	. ,	. ,			
3 percent discount rate	\$209	\$487			
7 percent discount rate	\$190	\$442			
OB/PWC	,				
3 percent discount rate	\$1,070	\$1,620			
7 percent discount rate	\$969	\$1,470			
, personi discount fuic	Ψ, σ,	Ψ1,			
Total Benefits ^g					
3 percent discount rate	\$2,140+B	\$3,380+B			
7 percent discount rate	\$1,940+B	\$3,070+B			
Annual Net Benefits (Total Benefits-Total Cost		. ,			
3 percent discount rate	\$1,890+B	\$3,140+B			
7 percent discount rate	\$1,690+B	\$2,830+B			

^b Note that costs are the annual total costs of reducing all pollutants associated with each provision of the proposed control package, while the benefits reflect the value of reductions in $PM_{2.5}$ only.

economic analyses (US EPA, 2000 and OMB, 2003). R.S d Handheld emission reductions associated with the proposed standards, volatile organic hydrocarbons, are not accounted for in the PM benefits scaling approach. The PM benefit scaling approach is based upon changes in NOx and direct PM_{2.5} (see section 8.2). We therefore do not estimate any PM-related benefits associated with emission reductions in the handheld engine class. e PM-related benefits in this table are nationwide.

^g Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Potential benefit categories that have not been quantified and monetized are listed in Table 8.1-2.

^a All estimates are rounded to three significant digits and represent annualized benefits and costs anticipated for the years 2020 and 2030. Columnar totals may not sum due to rounding.

^c To calculate annual fixed costs, we use a 7 percent average before-tax rate of return on private capital (see Chapter 9). We do not present annual costs using an alternative rate of return. In Chapter 9, however, we use both a 3 percent and 7 percent social discount rate to calculate the net present value of total social costs consistent with EPA and OMB guidelines for preparing economic analyses (US EPA, 2000 and OMB, 2003). ^{R,S}

^f Valuation of premature mortality based on long-term PM exposure assumes discounting over the SAB recommended 20-year segmented lag structure described in section 8.3. Valuation of non-fatal myocardial infarctions is based on the cost-of-illness over a 5-year period after the incident. The valuation of both endpoints therefore requires the use of a discount rate. We present the PM-related benefits results using a 3 percent and 7 percent social discount rate consistent with EPA and OMB guidelines for preparing economic analyses (US EPA, 2000 and OMB, 2003).

^RU.S. Environmental Protection Agency, 2000. Guidelines for Preparing Economic Analyses. www.yosemitel.epa.gov/ee/epa/eed/hsf/pages/Guideline.html.

S Office of Management and Budget, The Executive Office of the President, 2003. Circular A-4. http://www.whitehouse.gov/omb/circulars.

References for Chapter 8

- ¹ U.S. Environmental Protection Agency. May 2004. *Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/nonroad-diesel/2004fr.htm#documents. Accessed December 15, 2005.
- ² U.S. Environmental Protection Agency. February 2007. *Control of Hazardous Air Pollutants from Mobile Sources: Regulatory Impact Analysis*. Prepared by: Office of Air and Radiation. EPA420-R-07-002. Available at http://www.epa.gov/otaq/regs/toxics/420r07002contents.pdf. Accessed March 29, 2007.
- ³ U.S. Environmental Protection Agency. October 2006. Final Regulatory Impact Analysis (RIA) for the Proposed National Ambient Air Quality Standards for Particulate Matter. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/ttn/ecas/ria.html Accessed October 18, 2006.
- ⁴ U.S. Environmental Protection Agency. 2004. Air Quality Criteria for Particulate Matter Volume II of II. National Center for Environmental Assessment, Office of Research and Development. Research Triangle Park, NC. EPA/600/P-99/002bF.
- ⁵ U.S. Environmental Protection Agency. September 2000. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003.
- ⁶ U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance for Federal Agencies Preparing Regulatory Analyses, Available at: http://www/whitehouse.gov/omb/inforeg/iraguide.html. Accessed December 15, 2005.
- ⁷ Kunzli, N., S. Medina, R. Kaiser, P. Quenel, F. Horak Jr, and M. Studnicka. 2001. "Assessment of Deaths Attributable to Air Pollution: Should We Use Risk Estimates Based on Time Series or on Cohort Studies?" *American Journal of Epidemiology* 153(11):1050-55.
- ⁸ U.S. Environmental Protection Agency. May 2004. *Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/nonroad-diesel/2004fr.htm#documents. Accessed December 15, 2005.
- ⁹ U.S. Environmental Protection Agency. May 2004. *Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/nonroad-diesel/2004fr.htm#documents. Accessed December 15, 2005.
- ¹⁰ Abt Associates. 2005. Methodology for County-Level Mortality Rate Projections. Memorandum from Ellen Post and Don McCubbin (Abt Associates) to Bryan Hubbell and Zach Pekar (EPA). Sent October 25, 2005.
- ¹¹ Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- ¹² Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Air Pollution in the United States." *Environmental Health Perspectives* 105(6):608-612.
- ¹³ Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren, and P.K. Mills. 1995. "Estimated Long-Term Ambient Concentrations of PM(10) and Development of Respiratory Symptoms in a Nonsmoking Population." *Archives of Environmental Health* 50(2): 139-152.
- ¹⁴ Peters, A., D.W. Dockery, J.E. Muller, and M.A. Mittleman. 2001. "Increased Particulate Air Pollution and the Triggering of Myocardial Infarction." *Circulation* 103:2810-2815.
- ¹⁵ Moolgavkar, S.H. 2003. "Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties." In *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Special Report. Boston, MA: Health Effects Institute.
- ¹⁶ Ito, K. 2003. "Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan." In *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Special Report. Health Effects Institute, Boston, MA.
- ¹⁷ Moolgavkar, S.H. 2000. "Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas." *Journal of the Air and Waste Management Association* 50:1199-1206.
- ¹⁸ Sheppard, L. 2003. "Ambient Air Pollution and Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994." In *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Special Report. Boston, MA: Health Effects Institute.

- ¹⁹ Norris, G., S.N. YoungPong, J.Q. Koenig, T.V. Larson, L. Sheppard, and J.W. Stout. 1999. "An Association between Fine Particles and Asthma Emergency Department Visits for Children in Seattle." *Environmental Health Perspectives* 107(6):489-493.
- ²⁰ Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne, and F.E. Speizer. 1996. "Health Effects of Acid Aerosols On North American Children-Respiratory Symptoms." *Environmental Health Perspectives* 104(5):500-505.
- ²¹ Pope, C.A., III, D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. "Respiratory Health and PM₁₀ Pollution: A Daily Time Series Analysis." *American Review of Respiratory Diseases* 144:668-674.
- ²² Schwartz, J., and L.M. Neas. 2000. "Fine Particles are More Strongly Associated than Coarse Particles with Acute Respiratory Health Effects in Schoolchildren." *Epidemiology* 11:6-10.
- ²³ Ostro, B., M. Lipsett, J. Mann, H. Braxton-Owens, and M. White. 2001. "Air Pollution and Exacerbation of Asthma in African-American Children in Los Angeles." *Epidemiology* 12(2):200-208.
- ²⁴ Vedal, S., J. Petkau, R. White, and J. Blair. 1998. "Acute Effects of Ambient Inhalable Particles in Asthmatic and Nonasthmatic Children." *American Journal of Respiratory and Critical Care Medicine* 157(4):1034-1043.
- ²⁵ Ostro, B.D. 1987. "Air Pollution and Morbidity Revisited: A Specification Test." *Journal of Environmental Economics Management* 14:87-98.
- ²⁶ Ostro, B.D. and S. Rothschild. 1989. "Air Pollution and Acute Respiratory Morbidity: An Observational Study of Multiple Pollutants." *Environmental Research* 50:238-247.
- ²⁷ Mrozek, J.R., and L.O. Taylor. 2002. "What Determines the Value of Life? A Meta-Analysis." *Journal of Policy Analysis and Management* 21(2):253-270.
- ²⁸ Viscusi, V.K., and J.E. Aldy. 2003. "The Value of a Statistical Life: A Critical Review of Market Estimates Throughout the World." *Journal of Risk and Uncertainty* 27(1):5-76.
- ²⁹ Viscusi, W.K., W.A. Magat, and J. Huber. 1991. "Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-Dollar Trade-Offs for Chronic Bronchitis." *Journal of Environmental Economics and Management* 21:32-51.
- ³⁰ Cropper, M.L., and A.J. Krupnick. 1990. "The Social Costs of Chronic Heart and Lung Disease." Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.
- ³¹ Russell, M.W., D.M. Huse, S. Drowns, E.C. Hamel, and S.C. Hartz. 1998. "Direct Medical Costs of Coronary Artery Disease in the United States." *American Journal of Cardiology* 81(9):1110-1115.
- ³² Wittels, E.H., J.W. Hay, and A.M. Gotto, Jr. 1990. "Medical Costs of Coronary Artery Disease in the United States." *American Journal of Cardiology* 65(7):432-440.
- ³³ Agency for Healthcare Research and Quality (AHRQ). 2000. HCUPnet, Healthcare Cost and Utilization Project. Rockville, MD. http://www.ahrq.gov/HCUPnet/.
- ³⁴ Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista, and W.B. Saunders. 1997. "A National Estimate of the Economic Costs of Asthma." *American Journal of Respiratory and Critical Care Medicine* 156(3 Pt 1):787-793.
- ³⁵ Stanford, R., T. McLaughlin, and L.J. Okamoto. 1999. "The Cost of Asthma in the Emergency Department and Hospital." *American Journal of Respiratory and Critical Care Medicine* 160(1):211-215.
- ³⁶ Industrial Economics, Incorporated (IEc). March 31, 1994. Memorandum to Jim DeMocker, Office of Air and Radiation, Office of Policy Analysis and Review, U.S. Environmental Protection Agency.
- ³⁷ Rowe, R.D., and L.G. Chestnut. 1986. "Oxidants and Asthmatics in Los Angeles: A Benefits Analysis— Executive Summary." Prepared by Energy and Resource Consultants, Inc. Report to the U.S. Environmental Protection Agency, Office of Policy Analysis. EPA-230-09-86-018. Washington, DC.
- ³⁸ Neumann, J.E., M.T. Dickie, and R.E. Unsworth. March 31, 1994. "Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis—Draft Valuation Document." Industrial Economics Incorporated (IEc) Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review.
- ³⁹ Tolley, G.S. et al. January 1986. *Valuation of Reductions in Human Health Symptoms and Risks. University of Chicago*. Final Report for the U.S. Environmental Protection Agency.
- ⁴⁰ Council of Economic Advisors. 2005. The Annual Report of the Council of Economic Advisors. In: Economic Report of the President. Table B-60. U.S. Government Printing Office: Washington, DC.

- ⁴¹ Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- ⁴² Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Air Pollution in the United States." *Environmental Health Perspectives* 105(6):608-612.
- ⁴³ Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- ⁴⁴ Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Air Pollution in the United States." *Environmental Health Perspectives* 105(6):608-612.
- ⁴⁵ U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance for Federal Agencies Preparing Regulatory Analyses, Available at: http://www/whitehouse.gov/omb/inforeg/iraguide.html. Accessed December 15, 2005.
- ⁴⁶ U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance for Federal Agencies Preparing Regulatory Analyses, Available at: http://www/whitehouse.gov/omb/inforeg/iraguide.html. Accessed December 15, 2005.
- ⁴⁷ U.S. Environmental Protection Agency. March 2005. *Regulatory Impact Analysis for the Final Clean Air Interstate Rule*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/cair. Accessed December 15, 2005.
- ⁴⁸ U.S. Environmental Protection Agency (EPA). 1996. *Review of the National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information*. Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA report no. EPA/4521R-96-007.
- ⁴⁹ Garcia, P., B. Dixon, and J. Mjelde. 1986. "Measuring the Benefits of Environmental Change Using a Duality Approach: The Case of Ozone and Illinois Cash Grain Farms." *Journal of Environmental Economics and Management* 13:69-80.
- ⁵⁰ Fox, S., and R.A. Mickler. 1996. "Impact of Air Pollutants on Southern Pine Forests." *Ecological Studies* 118. New York: Springer Verlag.
- ⁵¹ EPA-SAB-COUNCIL-ADV-98-003. 1998. "Advisory Council on Clean Air Compliance Analysis Advisory on the Clean Air Act Amendments (CAAA) of 1990 Section 812 Prospective Study: Overview of Air Quality and Emissions Estimates: Modeling, Health and Ecological Valuation Issues Initial Studies."
- ⁵² Grosclaude, P., and N.C. Soguel. 1994. "Valuing Damage to Historic Buildings Using a Contingent Market: A Case Study of Road Traffic Externalities." *Journal of Environmental Planning and Management* 37: 279-287.
- ⁵³ U.S. Environmental Protection Agency. 2005. Air Quality Criteria for Ozone and Related Photochemical Oxidants (First External Review Draft). January. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=114523
- ⁵⁴ EPA, 2005. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Second External Review Draft). August. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=137307
- ⁵⁵ U.S. Environmental Protection Agency. March 2005. *Regulatory Impact Analysis for the Final Clean Air Interstate Rule*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/cair. Accessed December 15, 2005.
- ⁵⁶ U.S. Environmental Protection Agency. June 2005. Regulatory Impact Analysis for the Final Clean Air Visibility Rule or the Guidelines for Best Available Retrofit Technology (BART) Determinations Under the Regional Haze Regulations. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/visibility/pdfs/bart_ria 2005 6 15.pdf. Accessed December 15, 2005.
- ⁵⁷ Krewski D., R.T. Burnett, M.S. Goldbert, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz, and W.H. White. July 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute, Cambridge MA.
- ⁵⁸ EPA-SAB-COUNCIL_ADV_04-002. March 2004. Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis Benefits and Costs of the Clean Air Act, 1990-2020: Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis.

⁶² Clean Air Science Advisory Committee. June 2005. EPA's Review of the National Ambient Air Quality Standards for Particulate Matter (Second Draft PM Staff Paper, January 2005). A Review by the PM Review Panel of the EPA Clean Air Science Advisory Committee. EPASAB-CASAC-05-007.

63 EPA-SAB-COUNCIL_ADV_04-002. March 2004. Advisory on Plans for Health Effects Analysis in the

Analytical Plan for EPA's Second Prospective Analysis – Benefits and Costs of the Clean Air Act, 1990-2020: Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis. ⁶⁴ National Research Council (NRC). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. Washington, DC: The National Academies Press.

⁶⁵ Miller W, Robinson LA, Lawrence RS, eds. Valuing Health: Cost Effectiveness Analysis for Regulation. Committee to Evaluate Measures of Health Benefits for Environmental, Health, and Safety Regulation (Lawrence RS, chair), Board on Health Care Services, Institute of Medicine, National Academy Press, Washington D.C., 2006. ⁶⁶ U.S. Environmental Protection Agency. June 2005. Regulatory Impact Analysis for the Final Clean Air Visibility Rule or the Guidelines for Best Available Retrofit Technology (BART) Determinations Under the Regional Haze Regulations. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/visibility/pdfs/bart ria 2005 6 15.pdf. Accessed December 15, 2005.

⁵⁹ Laden, F., J. Schwartz, F.E. Speizer, and D.W. Dockery. 2006. Reduction in Fine Particulate Air Pollution and Mortality. American Journal of Respiratory and Critical Care Medicine. 173: 667-672.

⁶⁰ U.S. EPA. 2004. Air Quality Criteria for Particulate Matter, Volume II. Office of Research and Development. EPA/600/P-99/002bF, October.

⁶¹ U.S. Environmental Protection Agency. October 2006. Final Regulatory Impact Analysis (RIA) for the Proposed National Ambient Air Quality Standards for Particulate Matter. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/ttn/ecas/ria.html Accessed October 18, 2006.