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

Final Regulatory Impact Analysis

Chapter 8
Cost-Benefit Analysis

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CHAPTER 8: Cost-Benefit Analysis

8.1 Overview

This chapter presents our analysis of the health and environmental benefits that are estimated to occur as a result of the final Small SI and Marine SI engine standards throughout the period from initial implementation through 2030. Nationwide, the engines subject to the final emission standards in this rule are a significant source of nonroad mobile source air pollution. The final standards will reduce exposure to direct PM2.5, NOx, VOCs and air toxics emissions and help avoid a range of adverse health effects associated with ambient ozone and $PM_{2.5}$ levels.

EPA is required by Executive Order (E.O.) 12866 to estimate the benefits and costs of regulations with estimated annual impacts of over 100 million dollars. Such regulations tend to include major new pollution control regulations. To estimate these benefits and costs, the analysis presented here attempts to answer three questions: (1) what are the physical health and welfare effects projected to result from particulate matter (PM) and ozone precursors (direct PM, VOCs and NOx)? (2) what is the monetary value of the projected changes in health and welfare attributable to the final rule? and (3) how do the projected monetized benefits compare to the projected costs? This analysis constitutes one part of EPA's thorough examination of the relative merits of this regulation.

The benefits analysis relies on three major components to answer these questions:

- Calculation of the projected impact of the final rule on the national nonroad emissions inventory of precursors to ozone and $PM_{2.5}$, specifically NOx, VOCs and direct PM, for two future years (2020 and 2030).

- Air quality modeling for 2020 and 2030 to determine projected changes in ambient concentrations of ozone and $PM_{2.5}$, reflecting baseline and post-control emissions inventories.

- A benefits analysis to determine the projected changes in human health and welfare, both in terms of physical effects and monetary value, that result from the projected changes in ambient concentrations of ozone and $PM_{2.5}$ for the modeled standards.

A wide range of human health and welfare effects are linked with exposure to PM, VOCs and NOx. 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.

The benefits modeling is based on peer-reviewed studies of air quality and health and welfare effects associated with improvements in air quality and peer-reviewed studies of the dollar values of those public health and welfare effects. All of the benefit estimates for the control options in this analysis are based on an analytical structure and sequence consistent with benefits analyses performed for the recent analysis of the final Ozone NAAQS and the final PM NAAQS analysis.[1,2] For a more detailed discussion of the principles of benefits analysis used here, we refer the reader to those documents, as well as to the EPA Guidelines for Economic Analysis.

Table 8.1-1 summarizes the annual monetized health and welfare benefits associated with the final standards for two years, 2020 and 2030. The estimates in Table 8.1-1, and all monetized benefits presented in this chapter, are in year 2005 dollars. There are a few items to note about these benefits:

- Using a conservative benefits estimate, the 2020 benefits outweigh the costs by a factor of 5. Using the upper end of the benefits range, the benefits could outweigh the costs by a factor of 19. Likewise, in 2030 benefits outweigh the costs by at least a factor of 8 and could be as much as a factor of 34. Thus, even taking the most conservative benefits assumptions, benefits of the final standards clearly outweigh the costs.

- Emissions and air quality modeling decisions are made early in the analytical process. For this reason, the emission control scenarios used in the air quality and benefits modeling are slightly different than the final emission control program. The differences reflect further refinements of the regulatory program since we performed the air quality modeling for this rule. Chapter 3 of the RIA describes the changes in the inputs and resulting emission inventories between the preliminary assumptions used for the air quality modeling and the final regulatory scenario.

- The RIA for the proposal for this rulemaking only quantified benefits from PM; in the current RIA we quantify and monetize the ozone-related health impacts associated with the final rule. The science underlying the analysis is based on the current ozone criteria document.[3] The analytic approach to characterizing uncertainty is consistent with the analysis used in the RIA for the final Ozone NAAQS.

- In a recent report on the estimation of ozone-related premature mortality published by the National Research Council (NRC),[4] a panel of experts and reviewers concluded that ozone-related mortality should be included in estimates of the health benefits of reducing ozone exposure. The report also recommended that the estimation of ozone-related premature mortality be accompanied by broad uncertainty analyses while giving little or no weight to the assumption that there is no causal association between ozone exposure and premature mortality. Because EPA has yet to develop a coordinated response to the NRC report's findings and recommendations, however, we have retained the approach to

estimating ozone-related premature mortality used in RIA for the final Ozone NAAQS. EPA will specifically address the report's findings and recommendations in future rulemakings.

Table 8.1-1. Estimated Monetized PM- and Ozone-Related Health Benefits of the Small SI and Marine SI Engine Standards

2030 Total Ozone and PM Benefits – PM Mortality Derived from American Cancer Society Analysis[a]			
Premature Ozone Mortality Function or Assumption	Reference	Mean Total Benefits (Billions, 2005$, 3% Discount Rate)[c,d]	Mean Total Benefits (Billions, 2005$, 7% Discount Rate)[c,d]
NMMAPS	Bell et al., 2004	$2.4	$2.2
Meta-analysis	Bell et al., 2005	$3.7	$3.5
	Ito et al., 2005	$4.4	$4.2
	Levy et al., 2005	$4.4	$4.3
Assumption that association is not causal[e]		$1.8	$1.6
2030 Total Ozone and PM Benefits – PM Mortality Derived from Expert Elicitation[b]			
Premature Ozone Mortality Function or Assumption	Reference	Mean Total Benefits (Billions, 2005$, 3% Discount Rate)[c,d]	Mean Total Benefits (Billions, 2005$, 7% Discount Rate)[c,d]
NMMAPS	Bell et al., 2004	$1.7 - $9.7	$1.6 - $8.8
Meta-analysis	Bell et al., 2005	$3.0 - $11	$2.9 - $10
	Ito et al., 2005	$3.7 - $12	$3.6 - $11
	Levy et al., 2005	$3.7 - $12	$3.7 - $11
Assumption that association is not causal[e]		$1.1 to $9.1	$1.0 - $8.2

[a] Total includes ozone and $PM_{2.5}$ benefits. Range was developed by adding the estimate from the ozone premature mortality function to the estimate of $PM_{2.5}$-related premature mortality derived from the American Cancer Society analysis (Pope et al., 2002).

[b] Total includes ozone and $PM_{2.5}$ benefits. Range was developed by adding the estimate from the ozone premature mortality function to both the lower and upper ends of the range of the $PM_{2.5}$ premature mortality functions characterized in the expert elicitation. The effect estimates of five of the twelve experts included in the elicitation panel fall within the empirically-derived range provided by the ACS and Six-Cities studies. One of the experts fall below this range and six of the experts are above this range. Although the overall range across experts is summarized in this table, the full uncertainty in the estimates is reflected by the results for the full set of 12 experts. The twelve experts' judgments as to the likely mean effect estimate are not evenly distributed across the range illustrated by arraying the highest and lowest expert means.

[c] Note that total benefits presented here do not include a number of unquantified benefits categories. A detailed listing of unquantified health and welfare effects is provided in Table 8.4-1.

[d] Results reflect the use of both a 3 and 7 percent discount rate, as recommended by EPA's Guidelines for Preparing Economic Analyses and OMB Circular A-4. Results are rounded to two significant digits for ease of presentation and computation.

[e] A recent report published by the National Research Council (NRC, 2008) recommended that EPA "give little or no weight to the assumption that there is no causal association between estimated reductions in premature mortality and reduced ozone exposure."

Table 8.1-1 reflects those human health and welfare effects we are able to quantify and monetize. However, the full complement of known or suspected human health and welfare effects associated with PM, ozone and air toxics remain unquantified because of current limitations in methods or available data. We have not quantified potential health and welfare effects of ozone and PM because impact functions are not available or do not provide easily interpretable outcomes (e.g., changes in heart rate variability, acid and particulate deposition

damage to cultural monuments and other materials, and reductions in acidification of lakes and streams and eutrophication in coastal areas). As a result, we may underestimate the total benefits attributable to the implementation of the final standards.

This chapter is organized as follows. In Section 8.2, we provide an overview of the air quality impacts modeled for the final standards that are used as inputs to the benefits analysis. In Section 8.3, we discuss how uncertainty is characterized in this analysis. Section 8.4 discusses the literature on ozone- and PM-related health effects and describes the specific set of health impact functions we used in the benefits analysis. Section 8.5 describes the economic values selected to estimate the dollar value of ozone- and PM-related health impacts. In Section 8.6, we report the results of the analysis for human health and welfare effects. Finally, Section 8.7 presents a comparison of the costs and benefits associated with the final standards. There are also two appendices associated with this chapter. The first, Appendix 8A, presents the results of the health-based cost effectiveness analysis. The second, Appendix 8A, presents the results of sensitivity analyses of key parameters in the benefits analysis.

8.2 Air Quality Impacts for Benefits Analysis

In Chapter 2, we summarize the methods for and results of estimating air quality for the 2020 and 2030 base case and final control scenario. These air quality results are in turn associated with human populations and ecosystems to estimate changes in health and welfare effects. For the purposes of the benefits analysis, we focus on the health effects that have been linked to ambient changes in ozone and $PM_{2.5}$ related to emission reductions estimated to occur due to the final standards. We estimate ambient $PM_{2.5}$ and ozone concentrations using the Community Multiscale Air Quality model (CMAQ). The air quality modeling Technical Support Document (TSD), which can be found in the docket for this rule, contains detailed information about the modeling conducted for this rule. In this section, we describe how the modeled air quality results were used for the benefits analysis.

We remind the reader that the emission control scenarios used in the air quality and benefits modeling are slightly different than the final emission control program. The differences reflect further refinements of the regulatory program since we performed the air quality modeling for this rule. Emissions and air quality modeling decisions are made early in the analytical process. Chapter 3 of the RIA describes the changes in the inputs and resulting emission inventories between the preliminary assumptions used for the air quality modeling and the final regulatory scenario.

8.2.1 Converting CMAQ Outputs to Full-Season Profiles for Benefits Analysis

This analysis extracted hourly, surface-layer PM and ozone concentrations for each grid cell from the standard CMAQ output files. For ozone, these model predictions are used

in conjunction with the observed concentrations obtained from the Aerometric Information Retrieval System (AIRS) to generate ozone concentrations for the entire ozone season.[A,B] The predicted changes in ozone concentrations from the future-year base case to future-year control scenario serve as inputs to the health and welfare impact functions of the benefits analysis (i.e., the Environmental Benefits Mapping and Analysis Program [BenMAP]).

To estimate ozone-related health and welfare effects for the contiguous United States, full-season ozone data are required for every BenMAP grid-cell. Given available ozone monitoring data, we generated full-season ozone profiles for each location in two steps: (1) we combined monitored observations and modeled ozone predictions to interpolate hourly ozone concentrations to a grid of 12-km by 12-km population grid cells for the contiguous 48 states, and (2) we converted these full-season hourly ozone profiles to an ozone measure of interest, such as the daily 8-hour maximum.[C,D]

For $PM_{2.5}$, we also use the model predictions in conjunction with observed monitor data. CMAQ generates predictions of hourly PM species concentrations for every grid. The species include a primary coarse fraction (corresponding to PM in the 2.5 to 10 micron size range), a primary fine fraction (corresponding to PM less than 2.5 microns in diameter), and several secondary particles (e.g., sulfates, nitrates, and organics). $PM_{2.5}$ is calculated as the sum of the primary fine fraction and all of the secondarily formed particles. Future-year estimates of $PM_{2.5}$ were calculated using relative reduction factors (RRFs) applied to 2002 ambient $PM_{2.5}$ and $PM_{2.5}$ species concentrations. A gridded field of $PM_{2.5}$ concentrations was created by interpolating Federal Reference Monitor ambient data and IMPROVE ambient data. Gridded fields of $PM_{2.5}$ species concentrations were created by interpolating EPA speciation network (ESPN) ambient data and IMPROVE data. The ambient data were interpolated to the CMAQ 12 km grid.

The procedures for determining the RRFs are similar to those in EPA's draft guidance for modeling the $PM_{2.5}$ standard (EPA, 1999). The guidance recommends that model predictions be used in a relative sense to estimate changes expected to occur in each major $PM_{2.5}$ species. The procedure for calculating future-year $PM_{2.5}$ design values is called the "Speciated Modeled Attainment Test (SMAT)." EPA used this procedure to estimate the ambient impacts of the final emissions controls. Full documentation of the revised SMAT methodology is contained in the Air Quality Modeling TSD.

[A] The ozone season for this analysis is defined as the 5-month period from May to September.

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

[C] The 12-km grid squares contain the population data used in the health benefits analysis model, BenMAP.

[D] This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (EVNA) spatial interpolation. See the BenMAP manual for technical details, available for download at http://www.epa.gov/air/benmap.

8.2.2 Ozone and $PM_{2.5}$ Air Quality Results

This section provides a summary of the predicted ambient $PM_{2.5}$ and ozone concentrations from the CMAQ model for the 2020 and 2030 base cases and changes associated with the final rule. Table 8.2-1 provides those ozone and $PM_{2.5}$ metrics for grid cells in the modeled domain that enter the health impact functions for health benefits endpoints. The population-weighted average reflects the baseline levels and predicted changes for more populated areas of the nation. This measure better reflects the potential benefits through exposure changes to these populations.

Table 8.2-1. Summary of CMAQ-Derived Population-Weighted Ozone and $PM_{2.5}$ Air Quality Metrics for Health Benefits Endpoints Due to the Final Small SI and Marine SI Engine Standards

	2020		2030	
Statistic[a]	Baseline	Change[b]	Baseline	Change[b]
Ozone Metrics: National Population-Weighted Average (ppb)[c]				
Daily 1-Hour Maximum Concentration	47.60	0.078	46.91	0.108
Daily 8-Hour Maximum Concentration	44.07	0.066	43.47	0.093
Daily 8-Hour Average Concentration	42.63	0.062	42.06	0.088
Daily 24-Hour Average Concentration	35.39	0.047	35.02	0.068
$PM_{2.5}$ Metrics: National Population-Weighted Average (ug/m^3)				
Annual Average Concentration	9.41	0.015	9.38	0.021

[a] Ozone and $PM_{2.5}$ metrics are calculated at the CMAQ grid-cell level for use in health effects estimates based on the results of spatial and temporal Voronoi Neighbor Averaging. Ozone metrics are calculated over relevant time periods during the daylight hours of the "ozone season" (i.e., May through September). For the 8-hour average, for example, the relevant time period is 9 am to 5 pm.

[b] The change is defined as the base-case value minus the control-case value.

[c] Calculated by summing the product of the projected CMAQ grid-cell population and the estimated CMAQ grid cell seasonal ozone concentration and then dividing by the total population.

8.3 Characterizing Uncertainty: Moving Toward a Probabilistic Framework for Benefits Assessment

The National Research Council (NRC)[5] highlighted the need for EPA to conduct rigorous quantitative analysis of uncertainty in its benefits estimates and to present these estimates to decision makers in ways that foster an appropriate appreciation of their inherent uncertainty. In response to these comments, EPA's Office of Air and Radiation (OAR) is developing a comprehensive strategy for characterizing the aggregate impact of uncertainty in key modeling elements on both health incidence and benefits estimates. Components of that process include emissions modeling, air quality modeling, health effects incidence estimation, and valuation.

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, it is particularly important to characterize the uncertainties associated with reductions in premature mortality. The health impact functions used to estimate avoided premature deaths associated with reductions in ozone have associated standard errors that represent the statistical errors around the effect estimates in the underlying epidemiological studies.[E] In our results, we report credible intervals based on these standard errors, reflecting the uncertainty in the estimated change in incidence of avoided premature deaths. We also provide multiple estimates, to reflect model uncertainty between alternative study designs. In addition, we characterize the uncertainty introduced by the inability of existing empirical studies to discern whether the relationship between ozone and pre-mature mortality is causal by providing an effect estimate preconditioned on an assumption that the effect estimate for pre-mature mortality from ozone is zero.

For premature mortality associated with exposure to PM, we follow the same approach that has been used in several recent RIAs.[F,G,H] First, we use Monte Carlo methods for estimating random sampling error associated with the concentration response functions from epidemiological studies and economic valuation functions. Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables, such as incidence of premature mortality. Specifically, we used Monte Carlo methods to generate confidence intervals around the estimated health impact and dollar benefits. Distributions for individual effect estimates are based on the reported standard errors in the epidemiological studies. Distributions for unit values are described in Table 8.5-1.

Second, we use the results of our expert elicitation of the concentration response function describing the relationship between premature mortality and ambient $PM_{2.5}$ concentration.[I, J] Incorporating only the uncertainty from random sampling error omits important sources of uncertainty (e.g., in the functional form of the model; whether or not a

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[E] Health impact functions measure the change in a health endpoint of interest, such as hospital admissions, for a given change in ambient ozone or PM concentration.

[F] U.S. Environmental Protection Agency, 2004a. Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines. EPA420-R-04-007. Prepared by Office of Air and Radiation. Available at http://www.epa.gov/nonroad-diesel/2004fr/420r04007.pdf

[G] U.S. Environmental Protection Agency, 2005. Regulatory Impact Analysis for the Clean Air Interstate Rule. EPA 452/-03-001. Prepared by Office of Air and Radiation. Available at: http://www.epa.gov/interstateairquality/tsd0175.pdf

[H] U.S. Environmental Protection Agency, 2006. Regulatory Impact Analysis for the PM NAAQS. EPA Prepared by Office of Air and Radiation. Available at: http://www.epa.gov/ttn/ecas/regdata/RIAs/Chapter%205--Benefits.pdf

[I] Expert elicitation is a formal, highly structured and well documented process whereby expert judgments, usually of multiple experts, are obtained (Ayyb, 2002).

[J] Industrial Economics, Inc. 2006. Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between PM2.5 Exposure and Mortality. Prepared for EPA Office of Air Quality Planning and Standards, September. Available at: http://www.epa.gov/ttn/ecas/regdata/Uncertainty/pm_ee_report.pdf

threshold may exist). This second approach attempts to incorporate these other sources of uncertainty.

Use of the expert elicitation and incorporation of the standard errors approaches provide insights into the likelihood of different outcomes and about the state of knowledge regarding the benefits estimates. Both approaches have different strengths and weaknesses, which are fully described in Chapter 5 of the PM NAAQS RIA.

These multiple characterizations, including confidence intervals, omit the contribution to overall uncertainty of uncertainty in air quality changes, baseline incidence rates, populations exposed and transferability of the effect estimate to diverse locations. Furthermore, the approach presented here does not yet include methods for addressing correlation between input parameters and the identification of reasonable upper and lower bounds for input distributions characterizing uncertainty in additional model elements. As a result, the reported confidence intervals and range of estimates give an incomplete picture about the overall uncertainty in the estimates. This information should be interpreted within the context of the larger uncertainty surrounding the entire analysis.

8.4 Health Impact Functions

Health impact functions measure the change in a health endpoint of interest, such as hospital admissions, for a given change in ambient ozone or PM concentration. Health impact functions are derived from primary epidemiology studies, meta-analyses of multiple epidemiology studies, or expert elicitations. A standard health impact function has four components: 1) an effect estimate from a particular study; 2) a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics such as the Centers for Disease Control); 3) the size of the potentially affected population; and 4) the estimated change in the relevant ozone or PM summary measures.

A typical health impact function might look like:

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

where y_0 is the baseline incidence (the product of the baseline incidence rate times the potentially affected population), β is the effect estimate, and Δx is the estimated change in the summary pollutant measure. There are other functional forms, but the basic elements remain the same. Section 6.2 described the ozone and PM air quality inputs to the health impact functions. The following subsections describe the sources for each of the other elements: size of potentially affected populations; effect estimates; and baseline incidence rates.

8.4.1 Potentially Affected Populations

The starting point for estimating the size of potentially affected populations is the 2000 U.S. Census block level dataset.[6] Benefits Modeling and Analysis Program (BenMAP) incorporates 250 age/gender/race categories to match specific populations potentially affected

by ozone and other air pollutants. The software constructs specific populations matching the populations in each epidemiological study by accessing the appropriate age-specific populations from the overall population database. BenMAP projects populations to 2020 using growth factors based on economic projections.[7]

8.4.2 Effect Estimate Sources

The most significant monetized benefits of reducing ambient concentrations of ozone and PM are attributable to reductions in human health risks. EPA's Ozone and PM Criteria Documents[8,9] and the World Health Organization's 2003 and 2004[10,11] reports outline numerous health effects known or suspected to be linked to exposure to ambient ozone and PM. EPA recently evaluated the PM literature for use in the benefits analysis for the 2006 PM NAAQS RIA. Because we used the same literature for the PM benefits analysis in this RIA, and also in the RIA for the proposed rule, we do not provide a detailed discussion of individual effect estimates for PM in this section. Instead, we refer the reader to the 2006 PM NAAQS RIA and the proposed Small SI and Marine SI RIA for details.[K]

The RIA for the proposal for this rulemaking only quantified benefits from PM; in the current RIA we quantify and monetize the ozone-related health and environmental impacts associated with the final rule using an approach consistent with the final ozone NAAQS RIA. More than one thousand new ozone health and welfare studies have been published since EPA issued the 8-hour ozone standard in 1997. Many of these studies investigated the impact of ozone exposure on health effects such as: changes in lung structure and biochemistry; lung inflammation; asthma exacerbation and causation; respiratory illness-related school absence; hospital and emergency room visits for asthma and other respiratory causes; and premature death. We provide a discussion of those ozone-related impacts in this section. For a more detailed discussion of the health effects of ozone exposure, we point the reader to EPA's ozone Criteria Document.[12]

It is important to note that we were not able to separately quantify all of the PM and ozone health effects that have been reported in the ozone and PM criteria documents in this analysis for four reasons: (1) the possibility of double counting (such as hospital admissions for specific respiratory diseases); (2) uncertainties in applying effect relationships that are based on clinical studies to the potentially affected population; (3) the lack of an established concentration-response relationship; or 4) the inability to appropriately value the effect (for example, changes in forced expiratory volume) in economic terms. Table 8.4-1 lists the human health and welfare effects of pollutants affected by the final standards. Table 8.4-2 lists the health endpoints included in this analysis.

[K] U.S. Environmental Protection Agency, 2005. Regulatory Impact Analysis for the PM NAAQS. EPA Prepared by Office of Air and Radiation. Available at: http://www.epa.gov/ttn/ecas/regdata/RIAs/Chapter%205--Benefits.pdf pp. 5-29.

Table 8.4-1 Human Health and Welfare Effects of Pollutants Affected by the Final Standards

Pollutant/Effect	*Quantified and Monetized in Base Estimates*[a]	*Unquantified Effects - Changes in:*
PM/Health[b]	Premature mortality based on both cohort study estimates and on expert elicitation[c,d] Bronchitis: chronic and acute Hospital admissions: respiratory and cardiovascular Emergency room visits for asthma Nonfatal heart attacks (myocardial infarction) Lower and upper respiratory illness Minor restricted-activity days Work loss days Asthma exacerbations (asthmatic population) Respiratory symptoms (asthmatic population) Infant mortality	Subchronic bronchitis cases Low birth weight Pulmonary function Chronic respiratory diseases other than chronic bronchitis Nonasthma respiratory emergency room visits UVb exposure (+/-)[e]
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 Hospital admissions: respiratory Emergency room visits for asthma Minor restricted-activity days School loss days Asthma attacks Acute respiratory symptoms	Cardiovascular emergency room visits Chronic respiratory damage[g] Premature aging of the lungs[g] Nonasthma respiratory emergency room visits UVb exposure (+/-)[e]
Ozone/Welfare	Decreased outdoor worker productivity	Yields for commercial crops Yields for commercial forests and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions UVb exposure (+/-)[e]

Pollutant/Effect	*Quantified and Monetized in Base Estimates*[a]	*Unquantified Effects - Changes in:*
Nitrogen Deposition/ Welfare		Commercial forests due to acidic sulfate and nitrate deposition Commercial freshwater fishing due to acidic deposition Recreation in terrestrial ecosystems due to acidic deposition Commercial fishing, agriculture, and forests due to nitrogen deposition Recreation in estuarine ecosystems due to nitrogen deposition Ecosystem functions Passive fertilization
NOx/Health		Lung irritation Lowered resistance to respiratory infection Hospital admissions for respiratory and cardiac diseases
HC/Toxics Health[h]		Cancer, including lung (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)
HC/Toxics Welfare[h]		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 final standards.

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

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

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

[e] May result in benefits or disbenefits.

[f] The public health impact of biological responses such as increased airway responsiveness to stimuli, inflammation in the lung, acute inflammation and respiratory cell damage, and increased susceptibility to respiratory infection are likely partially represented by our quantified endpoints.

[g] The public health impact of effects such as chronic respiratory damage and premature aging of the lungs may be partially represented by quantified endpoints such as hospital admissions or premature mortality, but a number of other related health impacts, such as doctor visits and decreased athletic performance, remain unquantified.

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

Table 8.4-2. Ozone- and PM-Related Health Endpoints

Endpoint	*Pollutant*	*Study*	*Study Population*
Premature Mortality			
Premature mortality – daily time series, non-accidental	ozone	Bell et al (2004) (NMMAPS study)[13] Meta-analyses: Bell et al (2005)[14] Ito et al (2005)[15] Levy et al (2005)[16]	All ages
Premature mortality —cohort study, all-cause	$PM_{2.5}$	Pope et al. (2002)[17] Laden et al. (2006)[18]	>29 years >25 years
Premature mortality, total exposures	$PM_{2.5}$	Expert Elicitation (IEc, 2006)[19]	>24 years
Premature mortality — all-cause	$PM_{2.5}$	Woodruff et al. (1997)[20]	Infant (<1 year)
Chronic Illness			
Chronic bronchitis	$PM_{2.5}$	Abbey et al. (1995)[21]	>26 years
Nonfatal heart attacks	$PM_{2.5}$	Peters et al. (2001)[22]	Adults (>18 years)
Hospital Admissions			
Respiratory	ozone	Pooled estimate: Schwartz (1995) - ICD 460-519 (all resp)[23] Schwartz (1994a; 1994b) - ICD 480-486 (pneumonia)[24,25] Moolgavkar et al. (1997) - ICD 480-487 (pneumonia)[26] Schwartz (1994b) - ICD 491-492, 494-496 (COPD) Moolgavkar et al. (1997) – ICD 490-496 (COPD)	>64 years
		Burnett et al. (2001)[27]	<2 years
	$PM_{2.5}$	Pooled estimate: Moolgavkar (2003)—ICD 490-496 (COPD)[28] Ito (2003)—ICD 490-496 (COPD)[29]	>64 years
	$PM_{2.5}$	Moolgavkar (2000)—ICD 490-496 (COPD)[30]	20–64 years
	$PM_{2.5}$	Ito (2003)—ICD 480-486 (pneumonia)	>64 years
	$PM_{2.5}$	Sheppard (2003)—ICD 493 (asthma)[31]	<65 years
Cardiovascular	$PM_{2.5}$	Pooled estimate: Moolgavkar (2003)—ICD 390-429 (all cardiovascular) Ito (2003)—ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	>64 years
	$PM_{2.5}$	Moolgavkar (2000)—ICD 390-429 (all cardiovascular)	20–64 years
Asthma-related ER visits	ozone	Pooled estimate: Jaffe et al (2003)[32] Peel et al (2005)[33] Wilson et al (2005)[34]	5–34 years All ages All ages

Endpoint	*Pollutant*	*Study*	*Study Population*
Asthma-related ER visits (con't)	$PM_{2.5}$	Norris et al. (1999)[35]	0–18 years
Other Health Endpoints			
Acute bronchitis	$PM_{2.5}$	Dockery et al. (1996)[36]	8–12 years
Upper respiratory symptoms	$PM_{2.5}$	Pope et al. (1991)[37]	Asthmatics, 9–11 years
Lower respiratory symptoms	$PM_{2.5}$	Schwartz and Neas (2000)[38]	7–14 years
Asthma exacerbations	$PM_{2.5}$	Pooled estimate: Ostro et al. (2001)[39] (cough, wheeze and shortness of breath) Vedal et al. (1998)[40] (cough)	6–18 years[a]
Work loss days	$PM_{2.5}$	Ostro (1987)[41]	18–65 years
School absence days	ozone	Pooled estimate: Gilliland et al. (2001)[42] Chen et al. (2000)[43]	5–17 years[b]
Minor Restricted Activity Days (MRADs)	ozone	Ostro and Rothschild (1989)[44]	18–65 years
	$PM_{2.5}$	Ostro and Rothschild (1989)	18–65 years

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

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

In selecting epidemiological studies as sources of effect estimates, we applied several criteria to develop a set of studies that is likely to provide the best estimates of impacts in the U.S. To account for the potential impacts of different health care systems or underlying health status of populations, we give preference to U.S. studies over non-U.S. studies. In addition, due to the potential for confounding by co-pollutants, we give preference to effect estimates from models including both ozone and PM over effect estimates from single-pollutant models.[45,46]

A number of endpoints that are not health-related also may significantly contribute to monetized benefits. Potential welfare benefits associated with ozone exposure include: increased outdoor worker productivity; increased yields for commercial and non-commercial crops; increased commercial forest productivity; reduced damage to urban ornamental plants; increased recreational demand for undamaged forest aesthetics; and reduced damage to ecosystem functions.[47,48] While we include estimates of the value of increased outdoor worker productivity, estimation of other welfare impacts is beyond the scope of this analysis.

8.4.2.1 Ozone Exposure Metric

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

This practice is consistent both with the available exposure modeling and with the form of the current ozone standard. This conversion also does not affect the relative magnitude of the health impact function. An equivalent change in the 24-hour average, maximum1-hour average, and maximum 8-hour average will provide the same overall change in incidence of a health effect. The conversion ratios are based on observed relationships between the 24-hour average and maximum 8-hour average ozone values. For example, in the Bell et al., 2004 analysis of ozone-related premature mortality, the authors found that the relationship between the 24-hour average, the maximum 8-hour average, and the maximum 1-hour average was 2:1.5:1, so that the derived health impact effect estimate based on the maximum 1-hour average should be half that of the effect estimate based on the 24-hour values (and the maximum 8-hour average three-quarters of the 24-hour effect estimate).

8.4.2.2 Premature Mortality Effect Estimates

While particulate matter is the criteria pollutant most clearly associated with premature mortality, recent research suggests that short-term repeated ozone exposure likely contributes to premature death. The 2006 Ozone Criteria Document states: "Consistent with observed ozone-related increases in respiratory- and cardiovascular-related morbidity, several newer multi-city studies, single-city studies, and several meta-analyses of these studies have provided relatively strong epidemiologic evidence for associations between short-term ozone exposure and all-cause mortality, even after adjustment for the influence of season and PM" (EPA, 2006: E-17).[49] The epidemiologic data are also supported by newly available experimental data from both animal and human studies which provide evidence suggestive of

[L] An exposure metric is a measure of air quality calculated as the average or maximum of modeled ambient concentrations over a relevant time period, such as during the daylight hours of the "ozone season" (which is May through September for this analysis). The 24-hour average is therefore calculated as the average of all hourly ozone concentrations throughout the day (from 12am to 11:59pm). The 8-hour maximum is the maximum hourly value observed between 9am and 5pm each day. The 1-hour maximum is the maximum hourly value observed throughout an entire day.

plausible pathways by which risk of respiratory or cardiovascular morbidity and mortality could be increased by ambient ozone. With respect to short-term exposure, the ozone Criteria Document concludes: "This overall body of evidence is highly suggestive that ozone directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality, but additional research is needed to more fully establish underlying mechanisms by which such effects occur" (pg. E-18).

With respect to the time-series studies, the conclusion regarding the relationship between short-term exposure and premature mortality is based, in part, upon recent city-specific time-series studies such as the Schwartz (2004) analysis in Houston and the Huang et al. (2004) analysis in Los Angeles.[M] This conclusion is also based on recent meta-analyses by Bell et al. (2005), Ito et al. (2005), and Levy et al. (2005), and a new analysis of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data set by Bell et al. (2004), which specifically sought to disentangle the roles of ozone, PM, weather-related variables, and seasonality. The 2006 Criteria Document states that "the results from these meta-analyses, as well as several single- and multiple-city studies, indicate that co-pollutants generally do not appear to substantially confound the association between ozone and mortality" (p. 7-103). However, CASAC raised questions about the implications of these time-series results in a policy context. Specifically, CASAC emphasized that "…while the time-series study design is a powerful tool to detect very small effects that could not be detected using other designs, it is also a blunt tool" (Henderson, 2006: 3). They point to findings (e.g., Stieb et al., 2002, 2003) that indicated associations between premature mortality and all of the criteria pollutants, indicating that "findings of time-series studies do not seem to allow us to confidently attribute observed effects to individual pollutants" (id.). They note that "not only is the interpretation of these associations complicated by the fact that the day-to-day variation in concentrations of these pollutants is, to a varying degree, determined by meteorology, the pollutants are often part of a large and highly correlated mix of pollutants, only a very few of which are measured" (id.). Even with these uncertainties, the CASAC Ozone Panel, in its review of EPA's Staff Paper, found "…premature total non-accidental and cardiorespiratory mortality for inclusion in the quantitative risk assessment to be appropriate."

Consistent with the methodology used in the ozone risk assessment found in the Characterization of Health Risks found in the Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, we included ozone mortality in the primary health effects analysis, with the recognition that the exact magnitude of the effects estimate is subject to continuing uncertainty. We used effect estimates from the Bell et al. (2004) NMMAPS analysis, as well as effect estimates from the three meta-analyses.

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

In a recent report on the estimation of ozone-related premature mortality published by the National Research Council (NRC),[50] a panel of experts and reviewers concluded that ozone-related mortality should be included in estimates of the health benefits of reducing ozone exposure. The report also recommended that the estimation of ozone-related premature mortality be accompanied by broad uncertainty analyses while giving little or no weight to the assumption that there is no causal association between ozone exposure and premature mortality. Because EPA has yet to develop a coordinated response to the NRC report's findings and recommendations, however, we have retained the approach to estimating ozone-related premature mortality used in RIA for the final Ozone NAAQS. EPA will specifically address the report's findings and recommendations in future rulemakings.

We estimate the change in mortality incidence and estimated credible interval[N] resulting from application of the effect estimate from each study and present them separately to reflect differences in the study designs and assumptions about causality. However, it is important to note that this procedure only captures the uncertainty in the underlying epidemiological work, and does not capture other sources of uncertainty, such as uncertainty in the estimation of changes in air pollution exposure (Levy et al., 2000).

8.4.2.3 Respiratory Hospital Admissions Effect Estimates

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

Because the number of hospital admission studies we considered is so large, we used results from a number of studies to pool some hospital admission endpoints. Pooling is the process by which multiple study results may be combined in order to produce better estimates of the effect estimate, or β. For a complete discussion of the pooling process, see Abt (2005).[O] To estimate total respiratory hospital admissions associated with changes in ambient ozone concentrations for adults over 65, we first estimated the change in hospital admissions for each of the different effects categories that each study provided for each city. These cities included Minneapolis, Detroit, Tacoma and New Haven. To estimate total respiratory hospital admissions for Detroit, we added the pneumonia and COPD estimates, based on the effect estimates in the Schwartz study (1994). Similarly, we summed the estimated hospital admissions based on the effect estimates the Moolgavkar study reported for Minneapolis (Moolgavkar et al., 1997). To estimate total respiratory hospital admissions for Minneapolis using the Schwartz study (1994), we simply estimated pneumonia hospital admissions based on the effect estimate. Making this assumption that pneumonia admissions represent the total

[N] A credible interval is a posterior probability interval used in Bayesian statistics, which is similar to a confidence interval used in frequentist statistics.

[O] Abt Associates, Incorporated. Environmental Benefits Mapping and Analysis Program, Technical Appendices. May 2005. pp. I-3

impact of ozone on hospital admissions in this city will give some weight to the possibility that there is no relationship between ozone and COPD, reflecting the equivocal evidence represented by the different studies. We then used a fixed-effects pooling procedure to combine the two total respiratory hospital admission estimates for Minneapolis. Finally, we used random effects pooling to combine the results for Minneapolis and Detroit with results from studies in Tacoma and New Haven from Schwartz (1995). As noted above, this pooling approach incorporates both the precision of the individual effect estimates and between-study variability characterizing differences across study locations.

8.4.2.4 Asthma-Related Emergency Room Visits Effect Estimates

We used three studies as the source of the concentration-response functions we used to estimate the effects of ozone exposure on asthma-related emergency room (ER) visits: Peel et al. (2005); Wilson et al. (2005); and Jaffe et al. (2003). We estimated the change in ER visits using the effect estimate(s) from each study and then pooled the results using the random effects pooling technique (see Abt, 2005). The study by Jaffe et al. (2003) examined the relationship between ER visits and air pollution for populations aged five to 34 in the Ohio cities of Cleveland, Columbus and Cincinnati from 1991 through 1996. In single-pollutant Poisson regression models, ozone was linked to asthma visits. We use the pooled estimate across all three cities as reported in the study. The Peel et al. study (2005) estimated asthma-related ER visits for all ages in Atlanta, using air quality data from 1993 to 2000. Using Poisson generalized estimating equations, the authors found a marginal association between the maximum daily 8-hour average ozone level and ER visits for asthma over a 3-day moving average (lags of 0, 1, and 2 days) in a single pollutant model. Wilson et al. (2005) examined the relationship between ER visits for respiratory illnesses and asthma and air pollution for all people residing in Portland, Maine from 1998-2000 and Manchester, New Hampshire from 1996-2000. For all models used in the analysis, the authors restricted the ozone data incorporated into the model to the months ozone levels are usually measured, the spring-summer months (April through September). Using the generalized additive model, Wilson et al. (2005) found a significant association between the maximum daily 8-hour average ozone level and ER visits for asthma in Portland, but found no significant association for Manchester. Similar to the approach used to generate effect estimates for hospital admissions, we used random effects pooling to combine the results across the individual study estimates for ER visits for asthma. The Peel et al. (2005) and Wilson et al. (2005) Manchester estimates were not significant at the 95 percent level, and thus, the confidence interval for the pooled incidence estimate based on these studies includes negative values. This is an artifact of the statistical power of the studies, and the negative values in the tails of the estimated effect distributions do not represent improvements in health as ozone concentrations are increased. Instead these should be viewed as a measure of uncertainty due to limitations in the statistical power of the study. Note that we included both hospital admissions and ER visits as separate endpoints associated with ozone exposure, because our estimates of hospital admission costs do not include the costs of ER visits, and because most asthma ER visits do not result in a hospital admission.

8.4.2.5 Minor Restricted Activity Days Effects Estimate

Minor restricted activity days (MRADs) occur when individuals reduce most usual daily activities and replace them with less-strenuous activities or rest, but do not miss work or school. We estimated the effect of ozone exposure on MRADs using a concentration-response function derived from Ostro and Rothschild (1989). These researchers estimated the impact of ozone and $PM_{2.5}$ on MRAD incidence in a national sample of the adult working population (ages 18 to 65) living in metropolitan areas. We developed separate coefficients for each year of the Ostro and Rothschild analysis (1976-1981), which we then combined for use in EPA's analysis. The effect estimate used in the impact function is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4), using the inverse of the variance as the weight.

8.4.2.6 School Absences Effect Estimate

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

Following recent advice from the National Research Council (2002), we calculated reductions in school absences for the full population of school age children, ages five to 17. This is consistent with recent peer-reviewed literature on estimating the impact of ozone exposure on school absences (Hall et al. 2003). We estimated the change in school absences using both Chen et al. (2000) and Gilliland et al. (2001) and then, similar to hospital admissions and ER visits, pooled the results using the random effects pooling procedure.

8.4.2.7 Worker Productivity

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst (1981). Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts. Worker productivity is measuring the value of the loss in productivity for a worker who is at work on a particular day, but due to ozone, cannot work as hard. It only applies to outdoor workers, like fruit and vegetable pickers, or construction workers. Here, productivity impacts are measured as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration. The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income. Given the national median daily income for

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

8.4.2.8 Unquantified Effects

8.4.2.8.1 Direct Ozone Effects on Vegetation

The Ozone Criteria Document notes that "current ambient concentrations in many areas of the country are sufficient to impair growth of numerous common and economically valuable plant and tree species." (U.S. EPA, 2006, page 9-1). Changes in ground-level ozone resulting from the implementation of alternative ozone standards are expected to affect crop and forest yields throughout the affected area. Recent scientific studies have also found the ozone negatively impacts the quality or nutritive value of crops (U.S. EPA, 2006, page 9-16).

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 the supply of and demand for agricultural products. 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 benefits estimates for the final rule.

An additional welfare benefit expected to accrue as a result of reductions in ambient ozone concentrations in the United States is the economic value the public receives from reduced aesthetic injury to forests. There is sufficient scientific information available to reliably establish that ambient ozone levels cause visible injury to foliage and impair the growth of some sensitive plant species (U.S. EPA, 2006, page 9-19). However, present analytic tools and resources preclude EPA from quantifying the benefits of improved forest aesthetics.

Urban ornamentals (floriculture and nursery crops) represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to affect large economic sectors. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative economic benefits analysis has been conducted. The farm production value of ornamental crops was estimated at over $14 billion in 2003 (USDA, 2004). This is therefore a potentially important welfare effects category. However, information and valuation methods are not available to allow for

plausible estimates of the percentage of these expenditures that may be related to impacts associated with ozone exposure.

8.4.2.8.2 Nitrogen Deposition

Deposition to Estuarine and Coastal Waters

Excess nutrient loads, especially of nitrogen, cause a variety of adverse consequences to the health of estuarine and coastal waters. These effects include toxic and/or noxious algal blooms such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure (Bricker et al., 1999). A recent study found that for the period 1990-2002, atmospheric deposition accounted for 17 percent of nitrate loadings in the Gulf of Mexico, where severe hypoxic zones have been existed over the last two decades (Booth and Campbell, 2007)[P].

Reductions in atmospheric deposition of NOx are expected to reduce the adverse impacts associated with nitrogen deposition to estuarine and coastal waters. However, direct functions relating changes in nitrogen loadings to changes in estuarine benefits are not available. The preferred WTP-based measure of benefits depends on the availability of these functions and on estimates of the value of environmental responses. Because neither appropriate functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, calculation of a WTP measure is not possible.

Deposition to Agricultural and Forested Land

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

[P] Booth, M.S., and C. Campbell. 2007. Spring Nitrate Flux in the Mississippi River Basin: A Landscape Model with Conservation Applications. Environ. Sci. Technol.; 2007; ASAP Web Release Date: 20-Jun-2007; (Article) DOI: 10.1021/es070179e

Information on the effects of changes in passive nitrogen deposition on forests and other terrestrial ecosystems is very limited. The multiplicity of factors affecting forests, including other potential stressors such as ozone, and limiting factors such as moisture and other nutrients, confound assessments of marginal changes in any one stressor or nutrient in forest ecosystems. However, reductions in deposition of nitrogen could have negative effects on forest and vegetation growth in ecosystems where nitrogen is a limiting factor (US EPA, 1993). Moreover, any positive effect that nitrogen deposition has on forest productivity would enhance the level of carbon dioxide sequestration as well.[Q,R,S]

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

8.4.2.8.3 Ultraviolet Radiation

Atmospheric ozone absorbs a harmful band of ultraviolet radiation from the sun called UV-B, providing a protective shield to the Earth's surface. The majority of this protection occurs in the stratosphere where 90% of atmospheric ozone is located. The remaining 10% of the Earth's ozone is present at ground level (referred to as tropospheric ozone) (NAS, 1991; NASA). Only a portion of the tropospheric fraction of UV-B shielding is from anthropogenic sources (e.g., power plants, byproducts of combustion). The portion of ground level ozone associated with anthropogenic sources varies by locality and over time. Even so, it is reasonable to assume that reductions in ground level ozone would lead to increases in the same health effects linked to in UV-B exposures. These effects include fatal and nonfatal melanoma and non-melanoma skin cancers and cataracts. The values of $15,000 per case for non-fatal melanoma skin cancer, $5,000 per case for non-fatal non-melanoma skin cancer, and $15,000 per case of cataracts have been used in analyses of stratospheric ozone depletion (U.S. EPA, 1999). Fatal cancers are valued using the standard VSL estimate, which for 2020 is $6.6 million (1999$). UV-B has also been linked to ecological effects including damage to crops and forest. For a more complete listing of quantified and unquantified UV-B radiation effects, see Table G-4 and G-7 in the Benefits and Costs of the Clean Air Act, 1990-2010 (U.S. EPA, 1999. UV-B related health effects are also discussed in the context of stratospheric ozone in a 2006 report by ICF Consulting, prepared for the U.S. EPA.

[Q] Peter M. Vitousek et. al., "Human Alteration of the Global Nitrogen Cycle: Causes and Consequences" *Issues in Ecology* No. 1 (Spring) 1997.

[R] Knute J. Nadelhoffer et. al., "Nitrogen deposition makes a minor contribution to carbon sequestration in temperate forests" *Nature* 398, 145-148 (11 March 1999)

[S] Martin Köchy and Scott D. Wilson, "Nitrogen deposition and forest expansion in the northern Great Plains Journal of Ecology *Journal of Ecology* 89 (5), 807–817

There are many factors that influence UV-B radiation penetration to the earth's surface, including latitude, altitude, cloud cover, surface albedo, PM concentration and composition, and gas phase pollution. Of these, only latitude and altitude can be defined with small uncertainty in any effort to assess the changes in UV-B flux that may be attributable to any changes in tropospheric ozone as a result of any revision to the ozone NAAQS. Such an assessment of UV-B related health effects would also need to take into account human habits, such as outdoor activities (including age- and occupation-related exposure patterns), dress and skin care to adequately estimate UV-B exposure levels. However, little is known about the impact of these factors on individual exposure to UV-B.

Moreover, detailed information does not exist regarding other factors that are relevant to assessing changes in disease incidence, including: type (e.g., peak or cumulative) and time period (e.g., childhood, lifetime, current) of exposures related to various adverse health outcomes (e.g., damage to the skin, including skin cancer; damage to the eye, such as cataracts; and immune system suppression); wavelength dependency of biological responses; and interindividual variability in UV-B resistance to such health outcomes. Beyond these well recognized adverse health effects associated with various wavelengths of UV radiation, the Criteria Document (section 10.2.3.6) also discusses protective effects of UV-B radiation. Recent reports indicate the necessity of UV-B in producing vitamin D, and that vitamin D deficiency can cause metabolic bone disease among children and adults, and may also increase the risk of many common chronic diseases (e.g., type I diabetes and rheumatoid arthritis) as well as the risk of various types of cancers. Thus, the Criteria Document concludes that any assessment that attempts to quantify the consequences of increased UV-B exposure on humans due to reduced ground-level ozone must include consideration of both negative and positive effects. However, as with other impacts of UVB on human health, this beneficial effect of UVB radiation has not previously been studied in sufficient detail.

The Agency is currently evaluating the feasibility of estimating the effects of increased UVB exposures resulting from reductions in tropospheric ozone. Please refer to the final Ozone NAAQS RIA for a sensitivity analysis that explores the quantification of UV-B-related health effects.[51]

8.4.2.8.4 Climate Implications of Tropospheric Ozone

Although climate and air quality are generally treated as separate issues, they are closely coupled through atmospheric processes. Ozone, itself, is a major greenhouse gas and climate directly influences ambient concentrations of ozone.

The concentration of tropospheric ozone has increased substantially since the pre-industrial era and has contributed to warming. Tropospheric ozone is (after CO2 and CH4) the third most important contributor to greenhouse gas warming. The National Academy of Sciences recently stated[T] that regulations targeting ozone precursors would have combined

[T] National Academy of Sciences, "Radiative Forcing of Climate Change: Expanding the Concept and Addressing Uncertainties," October 2005.

benefits for public health and climate. As noted in the OAQPS Staff Paper, the overall body of scientific evidence suggests that high concentrations of ozone on a regional scale could have a discernible influence on climate. However, the Staff Paper concludes that insufficient information is available at this time to quantitatively inform the secondary NAAQS process with regard to this aspect of the ozone-climate interaction.

Climate change can affect tropospheric ozone by modifying emissions of precursors, chemistry, transport and removal.[U] Climate change affects the sources of ozone precursors through physical response (lightning), biological response (soils, vegetation, and biomass burning) and human response (energy generation, land use, and agriculture). Increases in regional ozone pollution are expected due to higher temperatures and weaker circulation. Simulations with global climate models for the 21st century indicate a decrease in the lifetime of tropospheric ozone due to increasing water vapor which could decrease global background ozone concentrations.

The Intergovernmental Panel on Climate Change (IPCC) recently released a report[V] which projects, with "virtual certainty," declining air quality in cities due to warmer and fewer cold days and nights and/or warmer/more frequent hot days and nights over most land areas. The report states that projected climate change-related exposures are likely to affect the health status of millions of people, in part, due to higher concentrations of ground level ozone related to climate change.

The IPCC also reports[W] that the current generation of tropospheric ozone models is generally successful in describing the principal features of the present-day global ozone distribution. However, there is much less confidence in the ability to reproduce the changes in ozone associated with perturbations of emissions or climate. There are major discrepancies with observed long-term trends in ozone concentrations over the 20th century, including after 1970 when the reliability of observed ozone trends is high. Resolving these discrepancies is needed to establish confidence in the models.

[U]Denman, K.L., G. Brasseur, A. Chidthaisong, P. Ciais, P.M. Cox, R.E. Dickinson, D. Hauglustaine, C. Heinze, E. Holland, D. Jacob, U. Lohmann, S Ramachandran, P.L. da Silva Dias, S.C. Wofsy and X. Zhang, 2007: Couplings Between Changes in the Climate System and Biogeochemistry. In: *Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment*

Report of the Intergovernmental Panel on Climate Change [Solomon, S., D. Qin, M. Manning, Z. Chen, M. Marquis, K.B. Averyt, M.Tignor and H.L. Miller (eds.)]. Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA.

[V] IPCC, Climate Change 2007: Climate Change Impacts, Adaptation and Vulnerability,

Summary for Policymakers

[W] Denman, et al, 2007: Couplings Between Changes in the Climate System and Biogeochemistry. In: *Climate Change 2007: The Physical Science Basis.*

The EPA is currently leading a research effort with the goal of identifying changes in regional US air quality that may occur in a future (2050) climate, focusing on fine particles and ozone. The research builds first on an assessment of changes in US air quality due to climate change, which includes direct meteorological impacts on atmospheric chemistry and transport and the effect of temperature changes on air pollution emissions. Further research will result in an assessment that adds the emission impacts from technology, land use, demographic changes, and air quality regulations to construct plausible scenarios of US air quality 50 years into the future. As noted in the Staff Paper, results from these efforts are expected to be available for consideration in the next review of the ozone NAAQS.

8.4.3 Baseline Incidence Rates

Epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the *relative risk* of a health effect, rather than estimating the absolute number of avoided cases. For example, a typical result might be that a 100 ppb decrease in daily ozone levels might, in turn, decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. A baseline incidence rate is the estimate of the number of cases of the health effect per year in the assessment location, as it corresponds to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number. For example, if the baseline incidence rate is the number of cases per year per 100,000 people, that number must be multiplied by the number of 100,000s in the population.

Table 8.4-3 summarizes the sources of baseline incidence rates and provides average incidence rates for the endpoints included in the analysis. For both baseline incidence and prevalence data, we used age-specific rates where available. We applied concentration-response functions to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits. In most cases, we used a single national incidence rate, due to a lack of more spatially disaggregated data. Whenever possible, the national rates used are national averages, because these data are most applicable to a national assessment of benefits. For some studies, however, the only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level. Regional incidence rates are available for hospital admissions, and county-level data are available for premature mortality. We have projected mortality rates such that future mortality rates are consistent with our projections of population growth (Abt Associates, 2005).

Table 8.4-3. National Average Baseline Incidence Rates[a]

Endpoint	Source	Notes	Rate per 100 people per year[d] by Age Group						
			<18	*18-24*	*25-34*	*35-44*	*45-54*	*55-64*	*65+*
Mortality	CDC Compressed Mortality File, accessed through CDC Wonder (1996-1998)	non-accidental	0.025	0.022	0.057	0.150	0.383	1.006	4.937
Respiratory Hospital Admissions.	1999 NHDS public use data files[b]	incidence	0.043	0.084	0.206	0.678	1.926	4.389	11.629
Asthma ER visits	2000 NHAMCS public use data files[c]; 1999 NHDS public use data files[b]	incidence	1.011	1.087	0.751	0.438	0.352	0.425	0.232
Minor Restricted Activity Days (MRADs)	Ostro and Rothschild (1989, p. 243)	incidence	–	780	780	780	780	780	–
School Loss Days	National Center for Education Statistics (1996) and 1996 HIS (Adams et al., 1999, Table 47); estimate of 180 school days per year	all-cause	990.0	–	–	–	–	–	–
Endpoint	Source	Notes			Rate per 100 people per year				
Asthma Exacerbations	Ostro et al. (2001)	Incidence (and prevalence) among asthmatic African-American children		Daily wheeze Daily cough Daily dyspnea	0.076 (0.173) 0.067 (0.145) 0.037 (0.074)				
	Vedal et al. (1998)	Incidence among asthmatic children		Daily wheeze Daily cough Daily dyspnea	0.038 0.086 0.045				

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

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

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

[d] All of the rates reported here are population-weighted incidence rates per 100 people per year. Additional details on the incidence and prevalence rates, as well as the sources for these rates are available upon request.

8.5 Economic Values for Health Outcomes

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects for a large population. Therefore, the appropriate economic measure is willingness-to-pay (WTP) for changes in risk of a health effect rather than WTP for a health effect that would occur with certainty (Freeman, 1993). Epidemiological studies generally provide estimates of the relative risks of a particular health effect that is avoided because of a reduction in air pollution. We converted those to units of avoided statistical incidence for ease of presentation. We calculated the value of avoided statistical incidences by dividing

individual WTP for a risk reduction by the related observed change in risk. For example, suppose a pollution-reduction regulation is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is $100, then the WTP for an avoided statistical premature death is $1 million ($100/0.0001 change in risk).

WTP estimates generally are not available for some health effects, such as hospital admissions. In these cases, we used the cost of treating or mitigating the effect as a primary estimate. These cost-of-illness (COI) estimates generally understate the true value of reducing the risk of a health effect, because they reflect the direct expenditures related to treatment, but not the value of avoided pain and suffering (Harrington and Portney, 1987; Berger, 1987). We provide unit values for health endpoints (along with information on the distribution of the unit value) in Table 8.5-1. All values are in constant year 2000 dollars, adjusted for growth in real income out to 2020 using projections provided by Standard and Poor's. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real income increases. Many of the valuation studies used in this analysis were conducted in the late 1980s and early 1990s. Because real income has grown since the studies were conducted, people's willingness to pay for reductions in the risk of premature death and disease likely has grown as well. We did not adjust cost of illness-based values because they are based on current costs. Similarly, we did not adjust the value of school absences, because that value is based on current wage rates. Table 8.5-1 presents the values for individual endpoints adjusted to year 2020 income levels. The discussion below provides additional details on ozone related endpoints not previously included in the proposal for this rule. For details on valuation estimates for PM-related endpoints, see the 2006 PM NAAQS RIA and the proposed Small SI and Marine SI RIA.

8.5.1 Mortality Valuation

To estimate the monetary benefit of reducing the risk of premature death, we used the "value of statistical lives" saved (VSL) approach, which is a summary measure for the value of small changes in mortality risk for a large number of people. The VSL approach applies information from several published value-of-life studies to determine a reasonable monetary value of preventing premature mortality. The mean value of avoiding one statistical death is estimated to be roughly $6.2 million at 1990 income levels (2005$), and $7.5 million at 2020 income levels. This represents an intermediate value from a variety of estimates in the economics literature (see the 2006 PM NAAQS RIA for more details on the calculation of VSL).

8.5.2 Hospital Admissions Valuation

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

The International Classification of Diseases (ICD-9, 1979) code-specific COI estimates used in this analysis consist of estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). We based all estimates of hospital charges and length of stays on statistics provided by the Agency for Healthcare Research and Quality (AHRQ 2000). We estimated the opportunity cost of a day spent in the hospital as the value of the lost daily wage, regardless of whether the hospitalized individual is in the workforce. To estimate the lost daily wage, we divided the 1990 median weekly wage by five and inflated the result to year 2005$ using the CPI-U "all items." The resulting estimate is $135.59. The total cost-of-illness estimate for an ICD code-specific hospital stay lasting *n* days, then, was the mean hospital charge plus $136 • *n.*

8.5.3 Asthma-Related Emergency Room Visits Valuation

To value asthma emergency room visits, we used a simple average of two estimates from the health economics literature. The first estimate comes from Smith et al. (1997), who reported approximately 1.2 million asthma-related emergency room visits in 1987, at a total cost of $186.5 million (1987$). The average cost per visit that year was $155; in 2005$, that cost was $386.32 (using the CPI-U for medical care to adjust to 2005$). The second estimate comes from Stanford et al. (1999), who reported the cost of an average asthma-related emergency room visit at $323.23 (in 2005$), based on 1996-1997 data. A simple average of the two estimates yields a (rounded) unit value of $355.

8.5.4 Minor Restricted Activity Days Valuation

No studies are reported to have estimated WTP to avoid a minor restricted activity day. However, one of EPA's contractors, IEc (1993) has derived an estimate of willingness to pay to avoid a minor *respiratory* restricted activity day, using estimates from Tolley et al. (1986) of WTP for avoiding a combination of coughing, throat congestion and sinusitis. The IEc estimate of WTP to avoid a minor respiratory restricted activity day is $38.37 (1990$), or about $59 (2005$).

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

8.5.5 School Absences

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

lost wages.

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

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

Our estimate of daily lost wage (wages lost if a mother must stay at home with a sick child) is based on the year 2000 median weekly wage among women ages 25 and older (U.S. Census Bureau, 2001). This median weekly wage is $551. Dividing by five gives an estimated median daily wage of $103. To estimate the expected lost wages on a day when a mother has to stay home with a school-age child, we first estimated the probability that the mother is in the workforce then multiplied that estimate by the daily wage she would lose by missing a work day: 72.85 percent times $103, for a total loss of $75. Using the CPI-U for all items to adjust to 2005$, the value equals approximately $85. This valuation approach is similar to that used by Hall et al. (2003).

Table 8.5-1. Unit Values Used for Economic Valuation of Health Endpoints (2005$)[a]

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level[b]	2030 Income Level[b]	
Premature Mortality (Value of a Statistical Life): $PM_{2.5}$- and Ozone-related	$6,200,000	$7,500,000	$7,700,000	Point estimate is the mean of a normal distribution with a 95 percent confidence interval between $1 and $10 million (in 2000$). 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)[52] meta-analysis and $10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003)[53] meta-analysis. Adjusted for 2005$, the mean equals approximately $6.2 million. The VSL represents the value of a small change in mortality risk aggregated over the affected population.
Chronic Bronchitis (CB)	$380,000	$470,000	$490,000	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][54]) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB.
Nonfatal Myocardial Infarction (heart attack)				Age-specific cost-of-illness values 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).[55] Direct medical costs are based on simple average of estimates from Russell et al. (1998)[56] and Wittels et al. (1990).[57]
3% discount rate				Lost earnings:
Age 0–24	$82,958	$82,958	$82,958	Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings:
Age 25–44	$92,598	$92,598	$92,598	
Age 45–54	$97,754	$97,754	$97,754	
Age 55–65	$174,405	$174,405	$174,405	
Age 66 and over	$82,958	$82,958	$82,958	
7% discount rate				
Age 0–24	$80,963	$80,963	$80,963	
Age 25–44	$90,705	$90,705	$90,705	
Age 45–54	$95,320	$95,320	$95,320	
Age 55–65	$163,945	$163,945	$163,945	
Age 66 and over	$80,963	$80,963	$80,963	

age of onset:	at 3%	at 7%
25-44	$10,880	$9,740
45-54	$16,036	$14,357
55-65	$92,685	$82,958

Direct medical expenses: An average of:
1. Wittels et al. (1990) ($127,296—no discounting)
2. Russell et al. (1998), 5-year period ($27,690 at 3% discount rate; $26,180 at 7% discount rate)

(continued)

Table 8.5-1. Unit Values Used for Economic Valuation of Health Endpoints (2005$)[a] (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level[b]	2030 Income Level[b]	
Hospital Admissions				
Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)	$15,345	$15,345	$15,345	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)[58] (www.ahrq.gov).
Pneumonia (ICD codes 480-487)	$18,219	$18,219	$18,219	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	$8,226	$8,226	$8,226	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)	$22,800	$22,800	$22,800	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	$355	$355	$355	Simple average of two unit COI values: (1) $386.32, from Smith et al. (1997)[59] and (2) $323.23, from Stanford et al. (1999).[60]

(continued)

Table 8.5-1. Unit Values Used for Economic Valuation of Health Endpoints (2005$)[a] (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level[b]	2030 Income Level[b]	
Respiratory Ailments Not Requiring Hospitalization				
Upper Respiratory Symptoms (URS)	$28	$30	$30	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)[61] 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)	$18	$19	$19	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS.
Asthma Exacerbations	$47	$51	$51	Asthma exacerbations are valued at $47 per incidence (2005$), based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986).[62] 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	$407	$434	$438	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).[63]

(continued)

Table 8.5-1. Unit Values Used for Economic Valuation of Health Endpoints (2005$)[a] (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income Level	2020 Income Level[b]	2030 Income Level[b]	
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.
School Absence Days	$85	$85	$85	Based on expected lost wages from parent staying home with child. Estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This median wage is $551. Dividing by 5 gives an estimated median daily wage of $103.. The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of $103, or $75 ($85 in 2005$)
Worker Productivity	$1.07 per worker per 10% change in ozone per day	$1.07 per worker per 10% change in ozone per day	$1.07 per worker per 10% change in ozone per day	Based on $68 ($77 in 2005$) – median daily earnings of workers in farming, forestry and fishing – from Table 621, Statistical Abstract of the United States ("Full-Time Wage and Salary Workers – Number and Earnings: 1985 to 2000") (Source of data in table: U.S. Bureau of Labor Statistics, Bulletin 2307 and Employment and Earnings, monthly).
Minor Restricted Activity Days (MRADs)	$58	$61	$62	Median WTP estimate to avoid one MRAD from Tolley et al. (1986).[64]

[a] All annual benefit estimates associated with the final 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.[65] 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 the PM NAAQS regulatory impact analysis. 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.

8.6 Benefits Analysis Results for the Final Standards

Applying the impact and valuation functions described previously in this chapter to the estimated changes in $PM_{2.5}$ and ozone associated with the final standards results in estimates of the changes in health damages (e.g., premature mortalities, cases, admissions) and the associated monetary values for those changes. Estimates of physical health impacts are presented in Table 8.6-1. Monetized values for those health endpoints are presented in Table 8.6-2. Total aggregate monetized benefits are presented in Table 8.6-3 and Table 8.6-4 using either a 3 percent or 7 percent discount rate, respectively. All of the monetary benefits are in constant-year 2005 dollars. For each endpoint presented in Tables 8.6-1 and 8.6-2, we provide both the mean estimate and the 90% confidence interval.

In addition to omitted benefits categories such as air toxics and various welfare effects, not all known $PM_{2.5}$- and ozone-related health and welfare effects could be quantified or monetized. The estimate of total monetized health benefits of the final standards is thus equal to the subset of monetized $PM_{2.5}$- and ozone-related health benefits we are able to quantify plus the sum of the nonmonetized health and welfare benefits. We believe the total benefits are therefore likely underestimated.

Total monetized benefits are dominated by benefits of mortality risk reductions. We provide results for particulate matter based on $PM_{2.5}$ concentration response functions from the American Cancer Society Study (ACS), Six Cities, and Expert Elicitation to give an indication of the sensitivity of the benefits estimates to alternative assumptions. Following the recommendations of the NRC report (NRC, 2002), we identify those estimates which are based on empirical data, and those which are based on expert judgments. EPA recently asked its Science Advisory Board (SAB) to evaluate how EPA has incorporated expert elicitation results into the benefits analysis, and the extent to which they find the presentation in this RIA responsive to the NRC (2002) guidance to incorporate uncertainty into the main analysis and further, whether the agency should move toward presenting a central estimate with uncertainty bounds or continue to provide separate estimates for each of the 12 experts as well as from the ACS and Six Cities studies. EPA has not yet had a chance to incorporate the results of the SAB's July 11, 2008 report (EPA-COUNCIL-08-002).

Using the ACS and Six-Cities results, we estimate that the final standards would result in between 150 and 340 cases of avoided $PM_{2.5}$-related premature deaths annually in 2020 and between 230 and 510 avoided premature deaths annually in 2030. When the range of expert opinion is used, we estimate between 80 and 840 fewer premature mortalities in 2020 and between 120 and 1,300 fewer premature mortalities in 2030. Note that in the case of the premature mortality estimates derived from the expert elicitation, we report the 90% credible interval, which encompasses a broader representation of uncertainty relative to the statistical confidence intervals provided for the effect estimates derived from the epidemiology literature.

The range of ozone benefits associated with the final standards is based on risk reductions estimated using several sources of ozone-related mortality effect estimates. This

analysis presents four alternative estimates for the association based upon different functions reported in the scientific literature, derived from both the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) (Bell et al., 2004) and from a series of recent meta-analyses (Bell et al., 2005, Ito et al., 2005, and Levy et al., 2005). This approach is not inconsistent with recommendations provided by the NRC in their recent report (NRC, 2008) on the estimation of ozone-related mortality risk reductions, "The committee recommends that the greatest emphasis be placed on estimates from new systematic multicity analyses that use national databases of air pollution and mortality, such as in the NMMAPS, without excluding consideration of meta-analyses of previously published studies."

Prior to the publication of the NRC ozone mortality report, EPA considered the possibility that the observed associations between ozone and mortality may not be causal in nature. The report, however, recommended that EPA give "little or no weight to the assumption that there is no causal association between ozone exposure and premature mortality." Because EPA has yet to develop a coordinated response to the NRC report's findings and recommendations, we have retained the approach to estimating ozone-related premature mortality used in RIA for the final Ozone NAAQS. EPA will specifically address the report's findings and recommendations in future rulemakings.

For ozone-related premature mortality, we estimate a range of between 46 to 210 fewer premature mortalities as a result of the final rule in 2020 and between 77 to 350 in 2030, assuming that there is a causal relationship between ozone exposure and mortality. The increase in annual benefits from 2020 to 2030 reflects additional emission reductions from the final 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 final standards, using the ACS and Six-Cities PM mortality studies and the range of ozone mortality assumptions, is between $1.2 billion and $4.0 billion, assuming a 3 percent discount rate, or between $1.1 billion and $3.8 billion, assuming a 7 percent discount rate. In 2030, we estimate the monetized benefits to be between $1.8 billion and $6.4 billion, assuming a 3 percent discount rate, or between $1.6 billion and $6.1 billion, assuming a 7 percent discount rate. The monetized benefit associated with reductions in the risk of both ozone- and $PM_{2.5}$-related premature mortality ranges between 90 to 98 percent of total monetized health benefits, in part because we are unable to quantify a number benefits categories (see Table 8.4-1). These unquantified benefits may be substantial, although their magnitude is highly uncertain.

The next largest benefit is for reductions in chronic illness (chronic bronchitis 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 PM-related premature mortalities (based on the ACS study), 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). As such, the true value of these effects may be higher than that reported in Table 8.6-2.

Following these tables, we also provide a more comprehensive presentation of the distributions of incidence generated using the available information from empirical studies and expert elicitation. Tables 8.6-5 and 8.6-6 present the distributions of the reduction in $PM_{2.5}$-related premature mortality based on the C-R distributions provided by each expert, as well as that from the data-derived health impact functions, based on the statistical error associated with the ACS study (Pope et al., 2002) and the Six-cities study (Laden et al., 2006). The 90% confidence interval for each separate estimate of PM-related mortality is also provided.

The effect estimates of five of the twelve experts included in the elicitation panel fall within the empirically-derived range provided by the ACS and Six-Cities studies. One of the experts fall below this range and six of the experts are above this range. Although the overall range across experts is summarized in these tables, the full uncertainty in the estimates is reflected by the results for the full set of 12 experts. The twelve experts' judgments as to the likely mean effect estimate are not evenly distributed across the range illustrated by arraying the highest and lowest expert means.

Table 8.6-1. Estimated Reduction in Incidence of Adverse Health Effects Related to the Final Standards[a]

		2020	2030
Health Effect		Mean Incidence Reduction (5^{th} – 95^{th} %ile)	
PM-Related Endpoints			
Premature Mortality – Derived from Epidemiology Literature	Adult, age 30+ - ACS cohort study (Pope et al., 2002)	150 (60 - 240)	230 (88 – 360)
	Adult, age 25+ - Six-Cities study (Laden et al., 2006)	340 (190 – 500)	510 (280 – 740)
	Infant, age <1 year – Woodruff et al. 1997	0 (0 – 1)	1 (0 – 1)
Premature Mortality – Derived from Expert Elicitation[b]	Adult, age 25+ - Lower Bound (Expert K)	81 (0 – 380)	120 (0 – 580)
	Adult, age 25+ - Upper Bound (Expert E)	840 (420 – 1,300)	1,300 (650 – 1,900)
Chronic bronchitis (adult, age 26 and over)		150 (28 – 270)	220 (40 – 400)
Acute myocardial infarction (adults, age 18 and older)		330 (180 – 480)	530 (280 – 770)
Hospital admissions—respiratory (all ages)[c]		40 (20 – 59)	61 (30 – 88)
Hospital admissions—cardiovascular (adults, age >18)[d]		81 (50 – 110)	130 (82 – 180)
Emergency room visits for asthma (age 18 years and younger)		150 (85 – 210)	210 (120 – 300)
Acute bronchitis (children, age 8–12)		400 (-14 – 810)	580 (-20 – 1,200)
Lower respiratory symptoms (children, age 7–14)		2,700 (1,300 – 4,000)	3,800 (1,800 – 5,800)
Upper respiratory symptoms (asthmatic children, age 9–18)		1,900 (610 – 3,300)	2,800 (880 – 4,700)
Asthma exacerbation (asthmatic children, age 6–18)		2,400 (270 – 7,000)	3,500 (380 – 10,000)
Work loss days (adults, age 18–65)		17,000 (15,000 – 19,000)	23,000 (20,000 – 26,000)
Minor restricted-activity days (adults, age 18–65)		100,000 (86,000 – 120,000)	140,000 (120,000 – 160,000)
Ozone-Related Endpoints			
Premature Mortality, All ages – Derived from NMMAPS	Bell et al., 2004	46 (20 – 72)	77 (34 – 120)
Premature Mortality, All ages – Derived from Meta-analyses	Bell et al., 2005	150 (84 – 210)	250 (140 – 360)
	Ito et al., 2005	200 (140 – 270)	340 (230 – 450)
	Levy et al., 2005	210 (160 – 260)	350 (260 - 440)
Premature Mortality – Assumption that association between ozone and mortality is not causal[e]		0	0
Hospital admissions- respiratory causes (children, under 2;		540	1,000

adult, 65 and older)[f]	(170 – 900)	(290 – 1,700)
Emergency room visit for asthma (all ages)	200 (0 – 510)	320 (0 - 810)
Minor restricted activity days (adults, age 18-65)	310,000 (160,000 – 460,000)	450,000 (230,000 – 670,000)
School absence days	110,000 (40,000 – 200,000)	180,000 (62,000 – 320,000)

[a] Incidence is rounded to two significant digits. PM and ozone estimates represent impacts from the final standards nationwide.

[b] Based on effect estimates derived from the full-scale expert elicitation assessing the uncertainty in the concentration-response function for PM-related premature mortality (IEc, 2006).[66] The effect estimates of five of the twelve experts included in the elicitation panel fall within the empirically-derived range provided by the ACS and Six-Cities studies. One of the experts fall below this range and six of the experts are above this range. Although the overall range across experts is summarized in this table, the full uncertainty in the estimates is reflected by the results for the full set of 12 experts. The twelve experts' judgments as to the likely mean effect estimate are not evenly distributed across the range illustrated by arraying the highest and lowest expert means.

[c] Respiratory hospital admissions for PM include admissions for chronic obstructive pulmonary disease (COPD), pneumonia, and asthma.

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

[e] A recent report published by the National Research Council (NRC, 2008) recommended that EPA "give little or no weight to the assumption that there is no causal association between estimated reductions in premature mortality and reduced ozone exposure."

[f] Respiratory hospital admissions for ozone include admissions for all respiratory causes and subcategories for COPD and pneumonia.

Table 8.6-2. Estimated Monetary Value in Reductions in Incidence of Health and Welfare Effects (in millions of 2005$)[a,b]

		2020	2030
$PM_{2.5}$-Related Health Effect		Estimated Mean Value of Reductions (5^{th} and 95^{th} %ile)	
Premature Mortality – Derived from Epidemiology Studies[c,d,]	Adult, age 30+ - ACS study (Pope et al., 2002)		
	3% discount rate	$1,000 ($240 - $2,100)	$1,600 ($370 - $3,200)
	7% discount rate	$910 ($220 - $1,900)	$1,400 ($330 - $2,800)
	Adult, age 25+ - Six-cities study (Laden et al., 2006)		
	3% discount rate	$2,300 ($630 - $4,400)	$3,500 ($970 - $6,700)
	7% discount rate	$2,100 ($570 - $3,900)	$3,200 ($870 - $6,000)
	Infant Mortality, <1 year – (Woodruff et al. 1997)		
	3% discount rate	$3.2 ($0.8 - $6.2)	$3.9 ($1.0 - $7.7)
	7% discount rate	$2.9 ($0.8 - $5.6)	$3.5 ($0.9 - $6.9)
Premature mortality – Derived from Expert Elicitation[c,d,e]	Adult, age 25+ - Lower bound (Expert K)		
	3% discount rate	$540 ($0 - $2,600)	$850 ($0 - $4,100)
	7% discount rate	$490 ($0 - $2,400)	$760 ($0 - $3,700)
	Adult, age 25+ - Upper bound (Expert E)		
	3% discount rate	$5,600 ($1,500 - $11,000)	$8,800 ($2,400 - $17,000)
	7% discount rate	$5,100 ($1,400 - $10,000)	$8,000 ($2,100 - $16,000)
Chronic bronchitis (adults, 26 and over)		$70 ($5.7 - $230)	$110 ($8.6 - $350)
Non-fatal acute myocardial infarctions			
3% discount rate		$34 ($10 - $72)	$52 ($15 - $110)
7% discount rate		$33 ($10 - $70)	$51 ($14 - $110)
Hospital admissions for respiratory causes		$0.8 ($0.4 - $1.2)	$1.3 ($0.6 - $1.8)
Hospital admissions for cardiovascular causes		$2.2 ($1.3 - $2.9)	$3.5 ($2.2 - $4.7)
Emergency room visits for asthma		$0.05 ($0.03 - $0.08)	$0.07 ($0.04 - $0.1)
Acute bronchitis (children, age 8–12)		$0.2 ($0 - $0.4)	$0.2 ($0 - $0.6)
Lower respiratory symptoms (children, 7–14)		$0.05 ($0.02 - $0.09)	$0.07 ($0.03 - $0.1)
Upper respiratory symptoms (asthma, 9–11)		$0.06	$0.08

		($0.02 - $0.1)	($0.02 - $0.2)
Asthma exacerbations		$0.1 ($0.01 - $0.4)	$0.2 ($0.02 - $0.5)
Work loss days		$2.5 ($2.2 - $2.8)	$3.4 ($3.0 - $3.8)
Minor restricted-activity days (MRADs)		$2.9 ($0.3 – $5.7)	$4.0 ($0.4 - $7.7)
Recreational Visibility, 86 Class I areas		$17 (na)[f]	$7 (na)
Ozone-related Health Effect			
Premature Mortality, All ages – Derived from NMMAPS	Bell et al., 2004	$340 ($86 - $680)	$590 ($150 - $1,200)
Premature Mortality, All ages – Derived from Meta-analyses	Bell et al., 2005	$1,100 ($310 - $2,100)	$1,900 ($530 - $3,600)
	Ito et al., 2005	$1,500 ($450 - $2,800)	$2,600 ($760 - $4,700)
	Levy et al., 2005	$1,600 ($470 - $2,700)	$2,600 ($800 - $4,700)
Premature Mortality – Assumption that association between ozone and mortality is not causal[f]		$0	$0
Hospital admissions- respiratory causes (children, under 2; adult, 65 and older)		$8.7 ($2.1 - $15)	$17 ($3.8 - $31)
Emergency room visit for asthma (all ages)		$0.07 ($0 - $0.2)	$0.1 ($0 - $0.3)
Minor restricted activity days (adults, age 18-65)		$19 ($8.5 - $31)	$27 ($13 - $46)
School absence days		$9.7 ($3.4 - $17)	$15 ($5.4 - $27)
Worker Productivity		$3.1 (na)[g]	$5.1 (na)[g]

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

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

[c] Valuation assumes discounting over the SAB recommended 20 year segmented lag structure. 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).

[d] The valuation of adult premature mortality, derived either from the epidemiology literature or the expert elicitation, is not additive. Rather, the valuations represent a range of possible mortality benefits.

[e] Based on effect estimates derived from the full-scale expert elicitation assessing the uncertainty in the concentration-response function for PM-related premature mortality (IEc, 2006). The effect estimates of five of the twelve experts included in the elicitation panel fall within the empirically-derived range provided by the ACS and Six-Cities studies. One of the experts fall below this range and six of the experts are above this range. Although the overall range across experts is summarized in this table, the full uncertainty in the estimates is reflected by the results for the full set of 12 experts. The twelve experts' judgments as to the likely mean effect estimate are not evenly distributed across the range illustrated by arraying the highest and lowest expert means.

[f] A recent report published by the National Research Council (NRC, 2008) recommended that EPA "give little or no weight to the assumption that there is no causal association between estimated reductions in premature mortality and reduced ozone exposure."

[g] We are unable at this time to characterize the uncertainty in the estimate of benefits of worker productivity and improvements in visibility at Class I areas. As such, we treat these benefits as fixed and add them to all percentiles of the health benefits distribution.

Table 8.6-3 Total Monetized Benefits of the Final Small SI and Marine SI Engine Rule – 3% Discount Rate

Total Ozone and PM Benefits (billions, 2005$) – PM Mortality Derived from the ACS and Six Cities Studies					
2020			**2030**		
Ozone Mortality Function	Reference	Mean Total Benefits	Ozone Mortality Function	Reference	Mean Total Benefits
NMMAPS	Bell et al., 2004	$1.5 - $2.8	NMMAPS	Bell et al., 2004	$2.4 - $4.3
Meta-analysis	Bell et al., 2005	$2.3 - $3.6	Meta-analysis	Bell et al., 2005	$3.7 - $5.6
	Ito et al., 2005	$2.7 - $4.0		Ito et al., 2005	$4.4 - $6.4
	Levy et al., 2005	$2.7 - $4.0		Levy et al., 2005	$4.4 - $6.4
Assumption that association is not causal[a]		$1.2 - $2.5	Assumption that association is not causal[a]		$1.8 - $3.8
Total Ozone and PM Benefits (billions, 2005$) – PM Mortality Derived from Expert Elicitation (Lowest and Highest Estimate)					
2020			**2030**		
Ozone Mortality Function	Reference	Mean Total Benefits	Ozone Mortality Function	Reference	Mean Total Benefits
NMMAPS	Bell et al., 2004	$1.1 - $6.1	NMMAPS	Bell et al., 2004	$1.7 - $9.7
Meta-analysis	Bell et al., 2005	$1.8 - $6.9	Meta-analysis	Bell et al., 2005	$3.0 - $11
	Ito et al., 2005	$2.2 - $7.3		Ito et al., 2005	$3.7 - $12
	Levy et al., 2005	$2.3 - $7.4		Levy et al., 2005	$3.7 - $12
Assumption that association is not causal[a]		$0.7 - $5.8	Assumption that association is not causal[a]		$1.1 - $9.1

[a] A recent report published by the National Research Council (NRC, 2008) recommended that EPA "give little or no weight to the assumption that there is no causal association between estimated reductions in premature mortality and reduced ozone exposure."

Table 8.6-4 Total Monetized Benefits of the Final Small SI and Marine SI Engine Rule – 7% Discount Rate

Total Ozone and PM Benefits (billions, 2005$) – PM Mortality Derived from the ACS and Six Cities Studies					
2020			**2030**		
Ozone Mortality Function	Reference	Mean Total Benefits	Ozone Mortality Function	Reference	Mean Total Benefits
NMMAPS	Bell et al., 2004	$1.4 - $2.6	NMMAPS	Bell et al., 2004	$2.2 - $4.0
Meta-analysis	Bell et al., 2005	$2.2 - $3.4	Meta-analysis	Bell et al., 2005	$3.5 - $5.3
	Ito et al., 2005	$2.6 - $3.7		Ito et al., 2005	$4.2 - $6.0
	Levy et al., 2005	$2.6 - $3.8		Levy et al., 2005	$4.3 - $6.1
Assumption that association is not causal[a]		$1.1 - $2.2	Assumption that association is not causal[a]		$1.6 - $3.4
Total Ozone and PM Benefits (billions, 2005$) – PM Mortality Derived from Expert Elicitation (Lowest and Highest Estimate)					
2020			**2030**		
Ozone Mortality Function	Reference	Mean Total Benefits	Ozone Mortality Function	Reference	Mean Total Benefits
NMMAPS	Bell et al., 2004	$1.0 - $5.6	NMMAPS	Bell et al., 2004	$1.6 - $8.8
Meta-analysis	Bell et al., 2005	$1.8 - $6.4	Meta-analysis	Bell et al., 2005	$2.9 - $10
	Ito et al., 2005	$2.2 - $6.8		Ito et al., 2005	$3.6 - $11
	Levy et al., 2005	$2.2 - $6.8		Levy et al., 2005	$3.7 - $11
Assumption that association is not causal[a]		$0.7 - $5.2	Assumption that association is not causal[a]		$1.0 - $8.2

[a] A recent report published by the National Research Council (NRC, 2008) recommended that EPA "give little or no weight to the assumption that there is no causal association between estimated reductions in premature mortality and reduced ozone exposure."

Table 8.6-5. Results of Application of Expert Elicitation: Annual Reductions in Premature Mortality in 2020 Associated with the Final Standards

Source of Mortality Estimate	2020 Primary Option		
	5th Percentile	Mean	95th Percentile
Pope et al. (2002)	59	150	240
Laden et al. (2006)	190	340	500
Expert A	120	670	1,200
Expert B	64	510	1,100
Expert C	92	510	1,100
Expert D	74	350	580
Expert E	420	840	1,300
Expert F	320	460	670
Expert G	0	300	550
Expert H	1	380	870
Expert I	80	500	900
Expert J	120	410	900
Expert K	0	81	380
Expert L	45	350	690

Table 8.6-6. Results of Application of Expert Elicitation: Annual Reductions in Premature Mortality in 2030 Associated with the Final Standards

Source of Mortality Estimate	2030 Primary Option		
	5th Percentile	Mean	95th Percentile
Pope et al. (2002)	88	230	360
Laden et al. (2006)	280	510	740
Expert A	190	1,000	1,900
Expert B	97	780	1,700
Expert C	140	780	1,700
Expert D	110	540	890
Expert E	650	1,300	1,900
Expert F	490	700	1,000
Expert G	0	450	840
Expert H	2	580	1,300
Expert I	120	770	1,400
Expert J	190	620	1,400
Expert K	0	120	580
Expert L	67	530	1,100

8.7 Comparison of Costs and Benefits

In estimating the net benefits of the final standards, the appropriate cost measure is 'social costs.' Social costs represent the welfare costs of a rule to society. These costs do not consider transfer payments (such as taxes) that are simply redistributions of wealth. Table 8.7-1 contains the estimates of monetized benefits and estimated social welfare costs for the final rule and each of the final control programs. The annual social welfare costs of all provisions of this final rule are described more fully in Chapter 9 of this RIA.

The results in Table 8.7-1 suggest that the 2020 monetized benefits of the final standards are greater than the expected social welfare costs. Specifically, the annual benefits of the total program will range between $1.2 to $4.0 billion annually in 2020 using a three percent discount rate, or between $1.1 to $3.8 billion assuming a 7 percent discount rate, compared to estimated social costs of approximately $210 million in that same year. These benefits are expected to increase to between $1.8 and $6.4 billion annually in 2030 using a three percent discount rate, or between $1.6 and $6.1 billion assuming a 7 percent discount rate, while the social costs are estimated to be approximately $190 million. Though there are a number of health and environmental effects associated with the final standards that we are unable to quantify or monetize (see Table 8.4-1), the benefits of the final standards far outweigh the projected costs. When we examine the benefit-to-cost comparison for the rule standards separately, we also find that the benefits of the specific engine standards outweigh their projected costs.

Using a conservative benefits estimate, the 2020 benefits outweigh the costs by a factor of 5. Using the upper end of the benefits range, the benefits could outweigh the costs by a factor of 19. Likewise, in 2030 benefits outweigh the costs by at least a factor of 8 and could be as much as a factor of 34. Thus, even taking the most conservative benefits assumptions, benefits of the final standards clearly outweigh the costs.

Table 8.7-1. Summary of Annual Benefits and Costs of the Final Standards[a] (Millions of 2005 dollars)

Description	2020	2030
Estimated Social Costs[b]		
Small SI	$163	$185
Marine SI	$44	$0.8
Total Social Costs	$210	$190
Estimated Health Benefits of the Final Standards[c,d,e,f]		
Small SI		
3 percent discount rate	$860 to $2,600	$820 to $2,900
7 percent discount rate	$790 to $2,500	$710 to $2,800
Marine SI		
3 percent discount rate	$340 to $1,400	$980 to $3,500
7 percent discount rate	$310 to $1,300	$890 to $3,300
Total Benefits		
3 percent discount rate	$1,200 to $4,000	$1,800 to $6,400
7 percent discount rate	$1,100 to $3,800	$1,600 to $6,100
Annual Net Benefits (Total Benefits – Total Costs)		
3 percent discount rate	$990 to $3,800	$1,600 to $6,200
7 percent discount rate	$890 to $3,600	$1,400 to $5,900

[a] All estimates represent annualized benefits and costs anticipated for the years 2020 and 2030. Totals may not sum due to rounding.

[b] The calculation of annual costs does not require amortization of costs over time. Therefore, the estimates of annual cost do not include a discount rate or rate of return assumption (see Chapter 9 of the RIA). 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).

[c] Total includes ozone and $PM_{2.5}$ benefits. Range was developed by adding the estimate from the ozone premature mortality function, including an assumption that the association is not causal, to $PM_{2.5}$-related premature mortality derived from the ACS (Pope et al., 2002) and Six Cities (Laden et al., 2006) studies.

[d] Annual benefits analysis results reflect the use of a 3 percent and 7 percent discount rate in the valuation of premature mortality and nonfatal myocardial infarctions, consistent with EPA and OMB guidelines for preparing economic analyses (US EPA, 2000 and OMB, 2003).

[e] Valuation of premature mortality based on long-term PM exposure assumes discounting over the SAB recommended 20-year segmented lag structure described in the Regulatory Impact Analysis for the Final Clean Air Interstate Rule (March, 2005).

[f] Not all possible benefits or disbenefits are quantified and monetized in this analysis. Potential benefit categories that have not been quantified and monetized are listed in Table 8.4-1.

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Appendix 8A: Sensitivity Analyses of Key Parameters in the Benefits Analysis

The primary analysis presented in Chapter 8 is based on our current interpretation of the scientific and economic literature. That interpretation requires judgments regarding the best available data, models, and modeling methodologies and the assumptions that are most appropriate to adopt in the face of important uncertainties and resource limitations. The majority of the analytical assumptions used to develop the primary estimates of benefits have been used to support similar rulemakings and approved by EPA's Science Advisory Board (SAB). Both EPA and the SAB recognize that data and modeling limitations as well as simplifying assumptions can introduce significant uncertainty into the benefit results and that alternative choices exist for some inputs to the analysis, such as the mortality C-R functions. This appendix supplements our primary estimates of benefits with a series of sensitivity calculations that use other sources of health effect estimates and valuation data for key benefits categories. The supplemental estimates examine sensitivity to both valuation issues and for physical effects issues. These supplemental estimates are not meant to be comprehensive. Rather, they reflect some of the key issues identified by EPA or commenters as likely to have a significant impact on total benefits. The individual adjustments in the tables should not simply be added together because: 1) there may be overlap among the alternative assumptions; and 2) the joint probability among certain sets of alternative assumptions may be low.

8.A.1 Premature Mortality – Alternative Threshold Analysis

To consider the impact of a threshold in the response function for the chronic mortality endpoint, we have 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, we have adjusted the mortality function slopes accordingly.[A] Five cutpoints (including the base case assumption) were included in the sensitivity analysis: (a) 14 µg/m3 (assumes no impacts below the alternative annual NAAQS), (b) 12 µg/m3 (c) 10 µg/m3 (reflects comments from CASAC, 2005) [1], (d) 7.5 µg/m3 (reflects recommendations from SAB-HES to consider estimating mortality benefits down to the lowest exposure levels considered in the Pope 2002 study used as the basis for modeling chronic mortality) [2] and (e) background or 3 µg/m3 (reflects NRC recommendation to consider effects all the way to background).[3] We repeat this sensitivity analysis for the RIA of the final standards, the results of which can be found in Table 8A-1.

[A] Note that this analysis only adjusted the mortality slopes for the 10 $\mu g/m^3$, 12 $\mu g/m^3$ and 14 $\mu g/m^3$ cutpoints since the 7.5 $\mu g/m^3$ 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.

Table 8A-1. PM-Related Mortality Benefits of the Final Standards: Cutpoint Sensitivity Analysis Using the ACS Study (Pope et al., 2002)[a]

Certainty that Benefits are At Least Specified Value	Level of Assumed Threshold	PM Mortality Incidence	
		2020	2030
More Certain that Benefits Are at Least as Large	14 µg/m3 [b]	6	7
	12 µg/m3	29	40
	10 µg/m3 [c]	150	230
	7.5 µg/m3 [d]	220	340
Less Certain that Benefits Are at Least as Large	3 µg/m3 [e]	250	380

[a] Note that this table only presents the effects of a cutpoint on PM-related mortality incidence.
[b] Alternative annual PM NAAQS.
[c] Primary threshold assumption based on CASAC (2005).[85]
[d] SAB-HES (2004)[86]
[e] NAS (2002)[87]

8.A.2 Premature Mortality - Alternative Lag Structures

Over the last ten years, there has been a continuing discussion and evolving advice regarding the timing of changes in health effects following changes in ambient air pollution. It has been hypothesized that some reductions in premature mortality from exposure to ambient $PM_{2.5}$ will occur over short periods of time in individuals with compromised health status, but other effects are likely to occur among individuals who, at baseline, have reasonably good health that will deteriorate because of continued exposure. No animal models have yet been developed to quantify these cumulative effects, nor are there epidemiologic studies bearing on this question.

The SAB-HES has recognized this lack of direct evidence. However, in early advice, they also note that "although there is substantial evidence that a portion of the mortality effect of PM is manifest within a short period of time, i.e., less than one year, it can be argued that, if no lag assumption is made, the entire mortality excess observed in the cohort studies will be analyzed as immediate effects, and this will result in an overestimate of the health benefits of improved air quality. Thus some time lag is appropriate for distributing the cumulative mortality effect of PM in the population," (EPA-SAB-COUNCIL-ADV-00-001, 1999, p. 9).[4] In recent advice, the SAB-HES suggests that appropriate lag structures may be developed based on the distribution of cause-specific deaths within the overall all-cause estimate (EPA-SAB-COUNCIL-ADV-04-002, 2004). They suggest that diseases with longer progressions should be characterized by longer-term lag structures, while air pollution impacts occurring in populations with existing disease may be characterized by shorter-term lags.

A key question is the distribution of causes of death within the relatively broad categories analyzed in the long-term cohort studies. Although it may be reasonable to assume the

cessation lag for lung cancer deaths mirrors the long latency of the disease, it is not at all clear what the appropriate lag structure should be for cardiopulmonary deaths, which include both respiratory and cardiovascular causes. Some respiratory diseases may have a long period of progression, while others, such as pneumonia, have a very short duration. In the case of cardiovascular disease, there is an important question of whether air pollution is causing the disease, which would imply a relatively long cessation lag, or whether air pollution is causing premature death in individuals with preexisting heart disease, which would imply very short cessation lags.

The SAB-HES provides several recommendations for future research that could support the development of defensible lag structures, including using disease-specific lag models and constructing a segmented lag distribution to combine differential lags across causes of death (EPA-SAB-COUNCIL-ADV-04-002, 2004). The SAB-HES indicated support for using "a Weibull distribution or a simpler distributional form made up of several segments to cover the response mechanisms outlined above, given our lack of knowledge on the specific form of the distributions," (EPA-SAB-COUNCIL-ADV-04-002, 2004, p. 24). However, they noted that "an important question to be resolved is what the relative magnitudes of these segments should be, and how many of the acute effects are assumed to be included in the cohort effect estimate," (EPA-SAB-COUNCIL-ADV-04-002, 2004, p. 24-25). Since the publication of that report in March 2004, EPA has sought additional clarification from this committee. In its follow-up advice provided in December 2004, the SAB suggested 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 $PM_{2.5}$ (EPA-COUNCIL-LTR-05-001, 2004).[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. Furthermore, in their advisory letter, the SAB-HES recommended that EPA include sensitivity analyses on other possible lag structures. In this appendix, we investigate the sensitivity of premature mortality-reduction related benefits to alternative cessation lag structures, noting that ongoing and future research may result in changes to the lag structure used for the primary analysis.

In previous advice from the SAB-HES, they recommended an analysis of 0-, 8-, and 15-year lags, as well as variations on the proportions of mortality allocated to each segment in the segmented lag structure (EPA-SAB-COUNCIL-ADV-00-001, 1999, (EPA-COUNCIL-LTR-05-001, 2004). The 0-year lag is representative of EPA's assumption in previous RIAs. The 8- and 15-year lags are based on the study periods from the Pope et al. (1995)[6] and Dockery et al. (1993)[7] studies, respectively.[B] However, neither the Pope et al. nor Dockery et al. studies assumed any lag structure when estimating the relative risks from PM exposure. In fact, the Pope et al. and Dockery et al. analyses do not support or refute the existence of a lag. Therefore, any lag structure applied to the avoided incidences estimated from either of these studies will be an assumed structure. The 8- and 15-year lags implicitly

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

assume that all premature mortalities occur at the end of the study periods (i.e., at 8 and 15 years).

In addition to the simple 8- and 15-year lags, we have added two additional sensitivity analyses examining the impact of assuming different allocations of mortality to the segmented lag of the type suggested by the SAB-HES. The first sensitivity analysis assumes that more of the mortality impact is associated with chronic lung diseases or lung cancer and less with acute cardiopulmonary causes. This illustrative lag structure is characterized by 20 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 30 percent occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$. The second sensitivity analysis assumes the 5-year distributed lag structure used in previous analyses, which is equivalent to a three-segment lag structure with 50 percent in the first 2-year segment, 50 percent in the second 3-year segment, and 0 percent in the 6- to 20-year segment.

The estimated impacts of alternative lag structures on the monetary benefits associated with reductions in PM-related premature mortality (estimated with the Pope et al. ACS impact function) are presented in Table 8A-2. These estimates are based on the value of statistical lives saved approach (i.e., $5.5 million per incidence) and are presented using both a 3 percent and 7 percent discount rate over the lag period.

Table 8A-2. Sensitivity of Benefits of Premature Mortality Reductions to Alternative Lag Assumptions (Relative to Primary Benefits Estimates of the Final Standards)

		Avoided Incidences (ACS; Pope et al., 2002)[a]		Value (million 2006$)[b]	
Description of Sensitivity Analysis		2020	2030	2020	2030
Alternative Lag Structures for PM-Related Premature Mortality					
Primary	30 percent of incidences occur in 1st year, 50 percent in years 2 to 5, and 20 percent in years 6 to 20				
	3% Discount Rate	150	230	$1,000	$1,600
	7% Discount Rate	150	230	$900	$1,400
None	Incidences all occur in the first year	150	230	$1,100	$1,700
8-year	Incidences all occur in the 8th year				
	3% Discount Rate	150	230	$910	$1,400
	7% Discount Rate	150	230	$690	$1,100
15-year	Incidences all occur in the 15th year				
	3% Discount Rate	150	230	$740	$1,100
	7% Discount Rate	150	230	$430	$660
Alternative Segmented	20 percent of incidences occur in 1st year, 50 percent in years 2 to 5, and 30 percent in years 6 to 20				
	3% Discount Rate	150	230	$1,100	$1,500
	7% Discount Rate	150	230	$1,000	$1,300
5-Year Distributed	50 percent of incidences occur in years 1 and 2 and 50 percent in years 2 to 5				
	3% Discount Rate	150	230	$980	$1,600
	7% Discount Rate	150	230	$850	$1,500

[a] Incidences rounded to two significant digits.

[b] Dollar values rounded to two significant digits. The alternative lag structure analysis presents benefits calculated using both a 3 percent and 7 percent discount rate.

The results of the scaled alternative lag sensitivity analysis demonstrate that choice of lag structure can have a large impact on benefits. Because of discounting of delayed benefits, the lag structure may have a large downward impact on monetized benefits if an extreme assumption that no effects occur until after 15 years is applied. However, for most reasonable distributed lag structures, differences in the specific shape of the lag function have relatively small impacts on overall benefits.

8.A.3 Visibility Benefits in Additional Class I Areas

The Chestnut and Rowe (1990)[viii] study from which the primary visibility valuation estimates are derived only examined WTP for visibility changes in Class I areas (national parks and wilderness areas) in the southeast, southwest, and California. To obtain estimates of WTP for visibility changes at national parks and wilderness areas in the northeast, northwest, and central regions of the U.S., we have to transfer WTP values from the studied regions. This introduces additional uncertainty into the estimates. However, we have taken steps to adjust the WTP values to account for the possibility that a visibility improvement in parks in one region is not necessarily the same environmental quality good as the same visibility improvement at parks in a different region. This may be due to differences in the scenic vistas at different parks, uniqueness of the

parks, or other factors, such as public familiarity with the park resource. To take this potential difference into account, we adjusted the WTP being transferred by the ratio of visitor days in the two regions.

Based on this benefits transfer methodology (implemented within the preference calibration framework discussed in Chapter 5 and Appendix I of the final PM NAAQS RIA), estimated additional visibility benefits in the northwest, central, and northeastern U.S. are provided in Table 8.A-3.

Table 8.A-3: Monetary Benefits Associated with Improvements in Visibility in Additional Federal Class I Areas in 2020 and 2030 (in millions of 2006$)[a]

Year	*Northwest*[b]	*Central*[c]	*Northeast*[d]	*Total*
2020	$3.9	$1.7	$9.2	$15
2030	$15	$17	$12	$44

[a] All estimates are rounded to 2 significant digits. All rounding occurs after final summing of unrounded estimates. As such, totals will not sum across columns

[b] Northwest Class I areas include Crater Lake, Mount Rainier, North Cascades, and Olympic national parks, and Alpine Lakes, Diamond Peak, Eagle Cap, Gearhart Mountain, Glacier Peak, Goat Rocks, Hells Canyon, Kalmiopsis, Mount Adams, Mount Hood, Mount Jefferson, Mount Washington, Mountain Lakes, Pasayten, Strawberry Mountain, and Three Sisters wilderness areas.

[c] Central Class I areas include Craters of the Moon, Glacier, Grand Teton, Theodore Roosevelt, Badlands, Wind Cave, and Yellowstone national parks, and Anaconda-Pintlar, Bob Marshall, Bridger, Cabinet Mountains, Fitzpatrick, Gates of the Mountain, Lostwood, Medicine Lake, Mission Mountain, North Absaroka, Red Rock Lakes, Sawtooth, Scapegoat, Selway-Bitterroot, Teton, U.L. Bend, and Washakie wilderness areas.

[d] Northeast Class I areas include Acadia, Big Bend, Guadalupe Mountains, Isle Royale, Voyageurs, and Boundary Waters Canoe national parks, and Brigantine, Caney Creek, Great Gulf, Hercules-Glades, Lye Brook, Mingo, Moosehorn, Presidential Range-Dry Roosevelt Campobello, Seney, Upper Buffalo, and Wichita Mountains wilderness areas.

Appendix 8B: Health-Based Cost-Effectiveness of Reductions in Ambient O_3 and $PM_{2.5}$ Associated with the Final Small SI and Recreational Marine Engine Rule

8B.1 Introduction

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. Analyses of environmental regulations have typically used benefit-cost analysis to characterize impacts on social welfare. Benefit-cost analyses allow for aggregation of the benefits of reducing mortality risks with other monetized benefits of reducing air pollution, including reduced risk of acute and chronic morbidity, and non-health benefits. One of the great advantages of the benefit-cost paradigm is that a wide range of quantifiable benefits can be compared to costs to evaluate the economic efficiency of particular actions. However, alternative paradigms such as CEA and CUA analyses may also provide useful insights. CEA involves estimation of the costs per unit of benefit (e.g., lives or life years saved). CUA is a special type of CEA using preference-based measures of effectiveness, such as quality-adjusted life years (QALYs).

QALYs were developed to evaluate the effectiveness of individual medical treatments, and EPA is still evaluating the appropriate methods for CEA for 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 non-health benefits.

The Office of Management and Budget (OMB) recently 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, however, making application of CEA more difficult and less straightforward.

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.

CEA and CUA are most useful for comparing programs that have similar goals, for example, alternative medical interventions or treatments that can save a life or cure a disease. They are less readily applicable to programs with multiple categories of benefits, such as those reducing ambient air pollution, because the cost-effectiveness calculation is based on the quantity of a single benefit category. In other words, we cannot readily convert non-health benefits, such as visibility improvements associated with reductions in $PM_{2.5}$ or increases in worker productivity associated with reductions in O_3, to a health metric such as life years saved. For these reasons, environmental economists prefer to present results in terms of monetary benefits and net benefits.

However, QALY-based CUA has been widely adopted within the health economics literature (Neumann, 2003; Gold et al., 1996) and in the analysis of public health interventions (US FDA, 2004). QALY-based analyses have not been as accepted in the environmental economics literature because of concerns about the theoretical consistency of QALYs with individual preferences (Hammitt, 2002), treatment of nonhuman health benefits, and a number of other factors (Freeman, Hammitt, and De Civita, 2002). For environmental regulations, benefit-cost analysis has been the preferred method of choosing among regulatory alternatives in terms of economic efficiency. Recently several academic analyses have proposed the use of life years-based benefit-cost or CEAs of air pollution regulations (Cohen, Hammitt, and Levy, 2003; Coyle et al., 2003; Rabl, 2003; Carrothers, Evans, and Graham, 2002). In addition, the World Health Organization has adopted the use of disability-adjusted life years, a variant on QALYs, to assess the global burden of disease due to different causes, including environmental pollution (Murray et al., 2002; de Hollander et al., 1999).

One of the ongoing controversies in health impact assessment regards whether reductions in mortality risk should be reported and valued in terms of statistical lives saved or in terms of statistical life years saved. Life years saved measures differentiate among premature mortalities based on the remaining life expectancy of affected individuals. In general, under the life years approach, older individuals will gain fewer life years than younger individuals for the same reduction in mortality risk during a given time period, making interventions that benefit older individuals seem less beneficial relative to similar interventions benefiting younger individuals. A further complication in the debate is whether to apply quality adjustments to life years lost. Under this approach, individuals with preexisting health conditions would have fewer QALYs lost relative to healthy individuals for the same loss in life expectancy, making interventions that primarily benefit individuals with poor health seem less beneficial than similar interventions affecting primarily healthy individuals.

In this CEA, based largely on a report prepared under contract with Abt Associates,[3] we calculated both life years saved and statistical lives saved. Following the methodology used in the CEAs for the PM and O_3 NAAQS RIAs, we did not assign QALY weights to the life years saved – i.e., we calculated life years saved, rather than QALYs gained from mortality avoided. Put another way, we assumed weights of 1.0 for all life years saved. Life years saved in the future, however, were discounted to reflect people's time preference (i.e., a benefit received now

[3] The full report prepared by Abt Associates is included in the docket for the Final Small SI and Recreational Marine Engine Rule (EPA-HQ-OAR-2004-0008).

is worth more than the same benefit received in the future). We used discount rates of 3 percent and 7 percent.

Where possible, benefits that could not be quantified in the denominator of our cost-effectiveness ratios were monetized and subtracted from the cost of the regulation in the numerator. For example, developing QALYs for acute health effects is problematic (Bala and Zarkin, 2000). Therefore, rather than try to derive QALYs for the acute morbidity endpoints, we instead applied valuation estimates and subtracted the total monetized value of all avoided acute morbidity effects from the cost of the regulation, in the numerator of the cost-effectiveness ratios. The monetized benefits of non-health improvements, where they were estimated, were similarly subtracted from the cost of the regulation. Finally, although QALY estimates were derived for the ($PM_{2.5}$-related) chronic morbidity endpoints, the medical and opportunity costs associated with these chronic illnesses were also subtracted from the cost of the regulation.

$PM_{2.5}$-related benefits derive not only from avoided cases of premature mortality and acute morbidity, but from avoided cases of chronic morbidity (chronic bronchitis and non-fatal myocardial infarction) as well. In the CEAs for the PM and O_3 NAAQS RIAs, EPA derived QALYs for these two chronic morbidity endpoints (see, for example, Appendix G of the PM NAAQS RIA, http://www.epa.gov/ttn/ecas/regdata/RIAs/Appendix%20G--Health%20Based%20Cost%20Effectiveness%20Analysis.pdf) and used an alternative aggregate effectiveness metric, Morbidity Inclusive Life Years (MILYs), to address some of the concerns about aggregation of life extension and quality-of-life impacts. MILYs represent the sum of life years gained due to reductions in premature mortality and the QALYs gained due to reductions in chronic morbidity. This measure may be preferred to existing QALY aggregation approaches because it does not devalue life extensions in individuals with preexisting illnesses that reduce quality of life. However, the MILY measure is still based on life years and thus still inherently gives more weight to interventions that reduce mortality and morbidity impacts for younger populations with higher remaining life expectancy.

For this analysis, we present several metrics: lives saved, life years saved, cost of the regulation (net of the monetized benefits not included in the denominator) per life saved and per life year saved, and MILYs gained and the cost of the regulation (net of the monetized benefits not included in the denominator) per MILY gained.

Note that, like future life years saved, future QALYs gained from avoided cases of chronic bronchitis and myocardial infarction are discounted. All costs and monetized benefits are in 2005 dollars.

Monte Carlo simulation methods as implemented in the Crystal Ball™ software program were used to propagate uncertainty in several of the model parameters throughout the analysis. In particular, we incorporated uncertainty surrounding the coefficients in the concentration-response (C-R) functions, the unit values for the various morbidity endpoints included in the analysis, and the quality of life weights for the two chronic morbidity endpoints for which we developed QALYs.

We characterized overall uncertainty in the results with 95 percent credible or confidence intervals based on the Monte Carlo simulations. In addition, we examined the impacts on the cost effectiveness metrics of changing key parameters and/or assumptions, including

- the discount rate (for the cost of the regulation in the numerator and future lives or life years saved and QALYs gained in the denominator);
- the C-R functions for O_3-related and $PM_{2.5}$-related mortality ; and
- the life expectancies (and therefore years of potential life lost) of individuals who die as a result of exposure to O_3 (as explained in Section 8B.4 below).

The methodology presented in this appendix is not intended to stand as precedent either for future air pollution regulations or for other EPA regulations where it may be inappropriate. It is intended solely to demonstrate one particular approach to estimating the cost-effectiveness of reductions in ambient $PM_{2.5}$ and O_3 in achieving improvements in public health. Reductions in ambient $PM_{2.5}$ and O_3 are estimated to have other health and environmental benefits that will not be reflected in this CEA. Other EPA regulations affecting other aspects of environmental quality and public health may require additional data and models that may preclude the development of similar health-based CEAs. A number of additional methodological issues must be considered when conducting CEAs for environmental policies, including treatment of non-health effects, aggregation of acute and long-term health impacts, and aggregation of life extensions and quality-of-life improvements in different populations. The appropriateness of health-based CEA should be evaluated on a case-by-case basis subject to the availability of appropriate data and models, among other factors.

The remainder of this appendix provides an overview of the methods used to derive the cost effectiveness metrics developed for this CEA and presents the resulting metrics. Section 8B.2 provides an overview of effectiveness measures. Section 8B.3 discusses general issues in constructing cost-effectiveness ratios. Section 8B.4 presents methods and results. Finally, Section 8B.5 presents concluding remarks.

8B.2 Effectiveness Measures

For the purposes of CEA, we focus the effectiveness measures on the quantifiable health impacts of the reductions in $PM_{2.5}$ and O_3 estimated to occur as a result of this rule. If the main impact of interest is reductions in mortality risk from air pollution, the effectiveness measures are relatively straightforward to develop. Mortality impacts can be characterized similar to the benefits analysis, by counting the number of premature deaths avoided, or can be characterized in terms of increases in life expectancy or life years.[4] Estimates of premature mortality have the benefit

[4] Life expectancy is an *ex ante* concept, indicating the impact on an entire population's expectation of the number of life years they have remaining, before knowing which individuals will be affected. Life expectancy thus incorporates both the probability of an effect and the impact of the effect if realized. Life years is an *ex post* concept, indicating the impact on individuals who actually die from exposure to air pollution. Changes in population life expectancy will always be substantially smaller than changes in life years per premature mortality avoided, although the total life years gained in the population will be the same. This is

of being relatively simple to calculate, are consistent with the benefit-cost analysis, and do not impose additional assumptions on the degree of life shortening. However, some have argued that counts of premature deaths avoided are problematic because a gain in life of only a few months would be considered equivalent to a gain of many life years, and the true effectiveness of an intervention is the gain in life expectancy or life years (Rabl, 2003; Miller and Hurley, 2003).

Calculations of changes in life years and life expectancy can be accomplished using standard life table methods (Miller and Hurley, 2003). However, the calculations require assumptions about the baseline mortality risks for each age cohort affected by air pollution. A general assumption may be that air pollution mortality risks affect the general mortality risk of the population in a proportional manner. However, some concerns have been raised that air pollution affects mainly those individuals with preexisting cardiovascular and respiratory disease, who may have reduced life expectancy relative to the general population. This issue is explored in more detail below.

Air pollution is also associated with a number of significant chronic and acute morbidity endpoints. Failure to consider these morbidity effects may understate the cost-effectiveness of air pollution regulations or give too little weight to reductions in particular pollutants that have large morbidity impacts but no effect on life expectancy. The QALY approach explicitly incorporates morbidity impacts into measures of life years gained and is often used in health economics to assess the cost-effectiveness of medical spending programs (Gold et al., 1996). Using a QALY rating system, health quality ranges from 0 to 1, where 1 may represent full health, 0 death, and some number in between (e.g., 0.8) an impaired condition. QALYs thus measure morbidity as a reduction in quality of life over a period of life. QALYs assume that duration and quality of life are equivalent, so that 1 year spent in perfect health is equivalent to 2 years spent with quality of life half that of perfect health. QALYs can be used to evaluate environmental rules under certain circumstances, although some very strong assumptions (detailed below) are associated with QALYs. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommended using QALYs when evaluating medical and public health programs that primarily reduce both mortality and morbidity (Gold et al., 1996). Although there are significant non-health benefits associated with air pollution regulations, over 90 percent of quantifiable monetized benefits are health-related. Thus, it can be argued that QALYs are more applicable for these types of regulations than for other environmental policies. However, the value of non-health benefits should not be ignored. As discussed below, we have chosen to subtract the value of non-health benefits from the costs in the numerator of the cost-effectiveness ratio.

The use of QALYs is predicated on the assumptions embedded in the QALY analytical framework. As noted in the QALY literature, QALYs are consistent with the utility theory that underlies most of economics only if one imposes several restrictive assumptions, including independence between longevity and quality of life in the utility function, risk neutrality with respect to years of life (which implies that the utility function is linear), and constant proportionality in trade-offs between quality and quantity of life (Pliskin, Shepard, and Weinstein, 1980; Bleichrodt, Wakker, and Johannesson, 1996). To the extent that these assumptions do not represent actual preferences, the QALY approach will not provide results

because life expectancy gains average expected life years gained over the entire population, while life years gained measures life years gained only for those experiencing the life extension.

that are consistent with a benefit-cost analysis based on the Kaldor-Hicks criterion.[5] Even if the assumptions are reasonably consistent with reality, because QALYs represent an average valuation of health states rather than the sum of societal WTP, there are no guarantees that the option with the highest QALY per dollar of cost will satisfy the Kaldor-Hicks criterion (i.e., generate a potential Pareto improvement [Garber and Phelps, 1997]).

Benefit-cost analysis based on WTP is not without potentially troubling underlying structures as well, incorporating ability to pay (and thus the potential for equity concerns) and the notion of consumer sovereignty (which emphasizes wealth effects). Table 8B-1 compares the two approaches across a number of parameters. For the most part, WTP allows parameters to be determined empirically, while the QALY approach imposes some conditions *a priori*.

Table 8B-1. Comparison of QALY and WTP Approaches

Parameter	*QALY*	*WTP*
Risk aversion	Risk neutral	Empirically determined
Relation of duration and quality	Independent	Empirically determined
Proportionality of duration/ quality trade-off	Constant	Variable
Treatment of time/age in utility function	Utility linear in time	Empirically determined
Preferences	Community/Individual	Individual
Source of preference data	Stated	Revealed and stated
Treatment of income and prices	Not explicitly considered	Constrains choices

8B.3 Construction of Cost-Effectiveness Ratios: General Issues

8B.3.1 Dealing with Morbidity Health Effects and Non-health Effects

Health effects from exposure to $PM_{2.5}$ and O_3 air pollution encompass a wide array of chronic and acute conditions in addition to premature mortality. EPA's Ozone and PM Criteria Documents outline numerous health effects known or suspected to be linked to exposure to ambient ozone and PM (US EPA, 2006; US EPA, 2005; Anderson et al., 2004). Although chronic conditions and premature mortality generally account for the majority of monetized benefits, acute symptoms can affect a broad population or sensitive populations (e.g., asthma-related emergency room visits among asthmatics). In addition, reductions in air pollution may result in a broad set of non-health environmental benefits, including improved worker productivity, improved visibility in national parks, increased agricultural and forestry yields, reduced acid damage to buildings, and a host of other impacts. Lives saved, life years saved, and

[5] The Kaldor-Hicks efficiency criterion requires that the "winners" in a particular case be potentially able to compensate the "losers" such that total societal welfare improves. In this case, it is sufficient that total benefits exceed total costs of the regulation. This is also known as a potential Pareto improvement, because gains could be allocated such that at least one person in society would be better off while no one would be worse off.

QALYs gained address only health impacts, and the OMB guidance notes that "where regulation may yield several different beneficial outcomes, a cost-effectiveness comparison becomes more difficult to interpret because there is more than one measure of effectiveness to incorporate in the analysis."

With regard to acute health impacts, Bala and Zarkin (2000) suggest that QALYs are not appropriate for valuing acute symptoms, because of problems with both measuring utility for acute health states and applying QALYs in a linear fashion to very short duration health states. Johnson and Lievense (2000) suggest using conjoint analysis to get healthy-utility time equivalences that can be compared across acute effects, but it is not clear how these can be combined with QALYs for chronic effects and loss of life expectancy. There is also a class of effects that EPA has traditionally treated as acute, such as hospital admissions, which may also result in a loss of quality of life for a period of time following the effect. For example, life after asthma hospitalization has been estimated with a utility weight of 0.93 (Bell et al., 2001; Kerridge, Glasziou, and Hillman, 1995).

How should these effects be combined with QALYs for chronic and mortality effects? One method would be to convert the acute effects to QALYs; however, as noted above, there are problems with the linearity assumption (i.e., if a year with asthma symptoms is equivalent to 0.7 year without asthma symptoms, then 1 day without asthma symptoms is equivalent to 0.0019 QALY gained). This is troubling from both a conceptual basis and a presentation basis. An alternative approach is simply to treat acute health effects like non-health benefits and subtract the dollar value (based on WTP or COI) from compliance costs in the CEA.

To address the issues of incorporating acute morbidity and non-health benefits, OMB suggests that agencies "subtract the monetary estimate of the ancillary benefits from the gross cost estimate to yield an estimated net cost." As with benefit-cost analysis, any unquantified benefits and/or costs should be noted and an indication of how they might affect the cost-effectiveness ratio should be described. We followed this recommended "net cost" approach, specifically in netting out the benefits of health improvements other than reduced mortality and improved quality of life from avoided chronic illness – in particular, the monetized benefits of acute morbidity avoided, the medical and opportunity costs ("cost of illness") of avoided chronic illness, and the benefits of non-health improvements, including increases in worker productivity associated with reductions in O_3 and visibility improvements at national parks associated with reductions in $PM_{2.5}$ (see Chapter 8 for more details on these benefit categories).

8B.3.2 Should Life Years Gained Be Adjusted for Initial Health Status?

The methods outlined below in Section 8B.4 provide estimates of the total number of life years gained in a population, regardless of the quality of those life years, or equivalently, assuming that all life years gained are in perfect health. In some CEAs (Cohen, Hammitt, and Levy, 2003; Coyle et al., 2003), analysts have adjusted the number of life years gained to reflect the fact that 1) the general public is not in perfect health and thus "healthy" life years are less than total life years gained and 2) those affected by air pollution may be in a worse health state than the general population and therefore will not gain as many "healthy" life years adjusted for quality, from an air pollution reduction. This adjustment, which converts life years gained into QALYs, raises a number of serious ethical issues. Proponents of QALYs have promoted the nondiscriminatory

nature of QALYs in evaluating improvements in quality of life (e.g., an improvement from a score of 0.2 to 0.4 is equivalent to an improvement from 0.8 to 1.0), so the starting health status does not affect the evaluation of interventions that improve quality of life. However, for life-extending interventions, the gains in QALYs will be directly proportional to the baseline health state (e.g., an individual with a 30-year life expectancy and a starting health status of 0.5 will gain exactly half the QALYs of an individual with the same life expectancy and a starting health status of 1.0 for a similar life-extending intervention). This is troubling because it imposes an additional penalty for those already suffering from disabling conditions. Brock (2002) notes that "the problem of disability discrimination represents a deep and unresolved problem for resource prioritization."

OMB (2003) has recognized this issue in their Circular A-4 guidance, which includes the following statement:

> *When CEA is performed in specific rulemaking contexts, you should be prepared to make appropriate adjustments to ensure fair treatment of all segments of the population. Fairness is important in the choice and execution of effectiveness measures. For example, if QALYs are used to evaluate a lifesaving rule aimed at a population that happens to experience a high rate of disability (i.e., where the rule is not designed to affect the disability), the number of life years saved should not necessarily be diminished simply because the rule saves the lives of people with life-shortening disabilities. Both analytic simplicity and fairness suggest that the estimated number of life years saved for the disabled population should be based on average life expectancy information for the relevant age cohorts. More generally, when numeric adjustments are made for life expectancy or quality of life, analysts should prefer use of population averages rather than information derived from subgroups dominated by a particular demographic or income group. (p. 13)*

This suggests two adjustments to the standard QALY methodology: one adjusting the relevant life expectancy of the affected population, and the other affecting the baseline quality of life for the affected population.

In addition to the issue of fairness, potential measurement issues are specific to the air pollution context that might argue for caution in applying quality-of-life adjustments to life years gained due to air pollution reductions. A number of epidemiological and toxicological studies link exposure to air pollution with chronic diseases, such as CB and atherosclerosis (Abbey et al., 1995; Schwartz, 1993; Suwa et al., 2002). If these same individuals with chronic disease caused by exposure to air pollution are then at increased risk of premature death from air pollution, there is an important dimension of "double jeopardy" involved in determining the correct baseline for assessing QALYs lost to air pollution (see Singer et al. [1995] for a broader discussion of the double-jeopardy argument).

Analyses estimating mortality from acute exposures that ignore the effects of long-term exposure on morbidity may understate the health impacts of reducing air pollution. Individuals exposed to chronically elevated levels of air pollution may realize an increased risk of death and chronic disease throughout life. If at some age they contract heart (or some other chronic) disease as a result of the exposure to air pollution, they will from that point forward have both reduced life

expectancy and reduced quality of life. The benefit to that individual from reducing lifetime exposure to air pollution would be the increase in life expectancy plus the increase in quality of life over the full period of increased life expectancy. If the QALY loss is determined based on the underlying chronic condition and life expectancy without regard to the fact that the person would never have been in that state without long-term exposure to elevated air pollution, then the person is placed in double jeopardy. In other words, air pollution has placed more people in the susceptible pool, but then we penalize those people in evaluating policies by treating their subsequent deaths as less valuable, adding insult to injury, and potentially downplaying the importance of life expectancy losses due to air pollution. If the risk of chronic disease and risk of death are considered together, then there is no conceptual problem with measuring QALYs, but this has not been the case in recent applications of QALYs to air pollution (Carrothers, Evans, and Graham, 2002; Coyle et al., 2003). The use of QALYs thus highlights the need for a better understanding of the relationship between chronic disease and long-term exposure and suggests that analyses need to consider morbidity and mortality jointly, rather than treating each as a separate endpoint (this is an issue for current benefit-cost approaches as well).

Because of the fairness and measurement concerns discussed above, for the purposes of this analysis, we do not reduce the number of life years gained to reflect any differences in underlying health status that might reduce quality of life in remaining years. Thus, we maintain the assumption that all direct gains in life years resulting from mortality risk reductions will be assigned a weight of 1.0. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" (Gold et al., 1996). However, for the purposes of this analysis, we propose an alternative to the traditional aggregate QALY metric that keeps separate quality adjustments to life expectancy and gains in life expectancy. As such, we do not make any adjustments to life years gained to reflect the less than perfect health of the general population. Gains in quality of life will be addressed as they accrue because of reductions in the incidence of chronic diseases. This is an explicit equity choice in the treatment of issues associated with quality-of-life adjustments for increases in life expectancy that still capitalizes on the ability of QALYs to capture both morbidity and mortality impacts in a single effectiveness measure.

8B.3.3 Constructing Cost-Effectiveness Ratios

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. The estimate of costs in the numerator should include both the direct costs of the controls necessary to achieve the reduction in ambient concentrations of the air pollutant and the avoided costs (cost savings) associated with the reductions in morbidity (Gold et al., 1996). In general, because reductions in air pollution do not require direct actions by the affected populations, there are no specific costs to affected individuals (aside from the overall increases in prices that might be expected to occur as control costs are passed on by affected industries). Likewise, because individuals do not engage in any specific actions to realize the health benefit of the pollution reduction, there are no decreases in utility (as might occur from a medical intervention) that need to be adjusted for in the denominator. Thus, the elements of the numerator are direct costs of controls minus the avoided costs of illness (COI) associated with chronic illnesses. In addition, as noted above, to account for the value of reductions in acute

health impacts and non-health benefits, we netted out the monetized value of these benefits from the numerator to yield a "net cost" estimate.

The denominators of the cost-effectiveness ratios we calculated are either lives saved, life years saved, or MILYs gained. For the MILY aggregate effectiveness measure, the denominator is simply the sum of life years gained from increased life expectancy and QALYs gained from the reductions in incidence of chronic illnesses associated with $PM_{2.5}$ – chronic bronchitis (CB) and nonfatal acute myocardial infarction (AMI).

8B.4 Cost Effectiveness Metrics

In this section we describe the development of cost effectiveness metrics. To generate health outcomes, we used the same framework as for the benefit-cost analysis described in Chapter 8. For convenience, we summarize the basic methodologies here. For more details, see Chapter 8 and the Environmental Benefits Mapping and Analysis Program (BenMAP) user's manual (http://www.epa.gov/ttn/ecas/benmodels.html).

BenMAP uses health impact functions to generate changes in the incidence of health effects. Health impact functions are derived from the C-R functions reported in the epidemiology literature. A standard health impact function has four components: an effect estimate from a particular epidemiological study, a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics, such as CDC), the affected population, and the estimated change in the relevant pollutant summary measure.

A typical health impact function might look like this:

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

where y_0 is the baseline incidence, equal to the baseline incidence rate times the potentially affected population; β is the effect estimate; Δx is the estimated change in the pollutant (e.g., $PM_{2.5}$ or O_3) and Δy is the estimated change in incidence of the health effect (e.g., the number of deaths avoided) associated with the change in the pollutant, Δx. There are other functional forms, but the basic elements remain the same.

8B.4.1 Reductions in O_3-Related Premature Deaths

To calculate O_3-related life years saved under the Final Small SI and Recreational Marine Engine Rule (hereafter, Final SSI & RME Rule), we first calculated the numbers of O_3-related statistical lives saved within 5-year age groups, using BenMAP. (For more details on the calculation of statistical lives saved using BenMAP, see Chapter 8 or the BenMAP user's manual (http://www.epa.gov/ttn/ecas/benmodels.html). We used two studies used in the benefit analysis for the Final SSI & RME Rule RIA – Bell et al. (2004) and Levy et al. (2005). Both studies report estimated C-R functions of the association between premature mortality and short-term exposures to ambient O_3. Bell et al. (2004) is a multi-city study of 95 cities, and as such may avoid the potential for publication bias that may be inherent in single-city studies or meta-

analyses of single-city studies. This study provides the lowest estimate of O_3-related premature deaths among the mortality studies included in the Final SSI & RME Rule RIA benefit analysis. An upper bound estimate of O_3-related premature deaths in the Final SSI & RME Rule RIA benefit analysis was provided by Levy et al. (2005). More extensive discussions of these studies are given in Chapter 8.

We checked to confirm that the total number of O_3-related statistical lives saved, summed across all age groups, equals the corresponding number calculated in the Final SSI & RME Rule RIA benefit analysis. Age group-specific O_3-related premature deaths avoided under the Final SSI & RME Rule in 2020 and in 2030 are given in Table 8B-2.

Table 8B-2. Estimated Reduction in Incidence of O_3-Related Premature Mortality Under the Final SSI & RME Rule in 2020 and 2030

Age Interval	Reduction in O_3-Related Premature Mortality (95% CI)*			
	2020		2030	
	Bell et al. (2004)	Levy et al. (2005)	Bell et al. (2004)	Levy et al. (2005)
0 - 4	0 (0 - 0)	1 (1 - 1)	0 (0 - 0)	1 (1 - 2)
5 - 9	0 (0 - 0)	0 (0 - 1)	0 (0 - 0)	1 (0 - 1)
10 - 14	0 (0 - 0)	0 (0 - 1)	0 (0 - 0)	1 (0 - 1)
15 - 19	0 (0 - 0)	1 (0 - 1)	0 (0 - 0)	1 (1 - 1)
20 - 24	0 (0 - 0)	1 (1 - 2)	0 (0 - 0)	2 (1 - 2)
25 - 29	0 (0 - 0)	2 (1 - 2)	0 (0 - 0)	2 (2 - 3)
30 - 34	0 (0 - 0)	2 (1 - 2)	0 (0 - 0)	2 (1 - 3)
35 - 39	0 (0 - 1)	3 (2 - 3)	1 (0 - 1)	4 (3 - 5)
40 - 44	0 (0 - 1)	2 (2 - 3)	1 (0 - 1)	3 (2 - 4)
45 - 49	1 (0 - 2)	5 (3 - 6)	1 (0 - 2)	7 (5 - 9)
50 - 54	1 (0 - 2)	5 (4 - 7)	1 (0 - 2)	5 (4 - 7)
55 - 59	3 (1 - 5)	13 (9 - 18)	3 (1 - 6)	16 (11 - 20)
60 - 64	3 (1 - 5)	13 (9 - 17)	4 (1 - 6)	16 (11 - 21)
65 - 69	6 (2 - 9)	25 (17 - 33)	9 (3 - 16)	42 (29 - 54)
70 - 74	4 (1 - 7)	20 (14 - 26)	8 (3 - 13)	35 (24 - 46)
75 - 79	7 (2 - 12)	31 (22 - 41)	15 (5 - 26)	67 (46 - 88)
80 - 84	5 (2 - 8)	20 (14 - 26)	9 (3 - 15)	39 (27 - 51)
85+	15 (5 - 25)	65 (45 - 85)	24 (8 - 40)	100 (72 - 140)
Total:	46 (15 - 77)	210 (140 - 270)	77 (25 - 130)	350 (240 - 460)

*95 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

8B.4.2 Life Years Saved as a Result of Reductions in O_3-Related Mortality Risk

The number of life years saved depends not only on the number of statistical lives saved, but also on the life expectancies associated with those statistical lives. As was pointed out in the CEAs for the PM and O_3 NAAQS RIAs, age-specific life expectancies for the general population are calculated from mortality rates for the general population, and these reflect the prevalence of chronic disease, which shortens life expectancies. The only reason one might use lower life expectancies than those for the general population in the CEA for the Final SSI & RME Rule RIA is if the population at risk from exposure to O_3 was limited solely or disproportionately to individuals with preexisting chronic illness, whose life expectancies were, on average, shorter than those of the general population (unless all of those individuals had preexisting chronic illness because of long-term exposure to O_3).

It is reasonable to assume that someone who dies from exposure to an air pollutant is already in a compromised state. However, there are both acute and chronic compromised states. If an individual has an acute illness (e.g., pneumonia) that puts him at risk of mortality when exposed to a high concentration of an air pollutant, then in the absence of that high concentration he could be expected to recover from the illness and go on to live the expected number of years for someone his age – i.e., he would have the age-specific life expectancy of the general population.

If an individual has a chronic illness that makes him vulnerable to a high concentration of an air pollutant, then an important question is whether or not he would have had that chronic illness if he had not been exposed over the long term to high levels of the air pollutant.

We can categorize individuals who are at risk of dying because of exposure to an air pollutant into three groups:

- those who are vulnerable because of a preexisting acute condition;
- those who are vulnerable because of a preexisting chronic condition that they would *not* have had, had they not been exposed over the long term to high levels of the air pollutant; and
- those who are vulnerable because of a preexisting chronic condition that they would have had even in the absence of long term exposure to high levels of the air pollutant.

The age-specific life expectancies of the general population should apply to the first two groups, and the age-specific life expectancies of the subpopulation with the relevant chronic condition(s) should apply to the third group. If we knew the proportions of people who die from exposure to O_3 who are in each group, and the life expectancies of people in the third group, we could calculate the number of life years saved as follows:

$$Total\ life\ years\ saved = \sum_i M_i * (p_{1i} * LE_i + p_{2i} * LE_i + p_{3i} * LE_i^*)$$

where

M_i denotes the number of O_3-related deaths of individuals age i,

LE_i denotes the general population life expectancy for age i,

LE_i^* denotes the life expectancy for age *i* of the subpopulation with the relevant chronic condition(s) – i.e., the third group;

p_{1i} denotes the proportion of the M_i O_3-related deaths that are in the first group;

p_{2i} denotes the proportion of the M_i O_3-related deaths that are in the second group; and

p_{3i} denotes the proportion of the M_i O_3-related deaths that are in the third group.

Unlike for $PM_{2.5}$ (discussed below), we currently lack information that would allow us to estimate the relevant proportions necessary to estimate the set of life expectancies that would be appropriate to apply to O_3-related deaths. Although there is substantial evidence linking premature mortality to short-term exposures to O_3, there is currently not similar evidence for long-term exposures. We therefore do not know if the second group above is relevant in the case of O_3-related mortality. Nor do we know what proportion of O_3-related deaths can be attributed to preexisting acute conditions (the first group) versus preexisting chronic conditions that these individuals would have had even in the absence of long term exposure to O_3 (the third group).

Because we currently lack the necessary information to determine the appropriate set of life expectancies to use in calculating life years saved associated with O_3-related premature mortality avoided, we calculated life years saved based on four different underlying assumptions:

- A lower bound assumption of zero life years saved, based on the hypothesis that the observed statistical association between premature mortality and short-term exposures to O_3 is not actually a causal relationship;
- An upper bound assumption that an O_3-related premature death of an individual of a given age will result in a loss of life years equal to the life expectancy in the general population of that age;
- Two intermediate assumptions: That the proportions of O_3-related premature deaths in the three groups delineated above (p_{1i}, p_{2i}, and p_{3i}) are such that, on average, the age-specific life expectancies among people who die O_3-related premature deaths are those of
    - people with severe preexisting chronic conditions, whose life expectancies are substantially shorter than those of the general population; and
    - people with preexisting chronic conditions of a range of severities, whose life expectancies are somewhat shorter than those of the general population.

Life years saved based on the upper bound assumption were calculated from age-specific mortality probabilities for the general population taken from the Centers for Disease Control (CDC) National Vital Statistics Reports, Vol. 56, No. 9, December 28, 2007, Table 1. Life table for the total population: United States, 2004.[6] We used a simplified method of calculating life expectancies from these age-specific mortality probabilities that yielded life expectancies that were close to the life expectancies derived using the more complicated method employed by the

[6] http://www.cdc.gov/nchs/data/nvsr/nvsr56/nvsr56_09.pdf

CDC.[7] In particular, starting with a cohort of size 1,000,000 at birth, we calculated the life-years lived between ages x and (x+1), for x = 0, 1, 2, …, 99, using the age-specific mortality probabilities taken from the CDC Vital Statistics Report (see above) and assuming that all deaths that occurred between ages x and (x+1) occurred midway through the year (i.e., we assigned 0.5 life-year to each year of death). The life expectancy at age n was then calculated as the sum of the life-years lived from age n through age 100 divided by the cohort size at age n. The life expectancy at age n is the number of life years lost due to an O_3-related premature mortality of an individual age n.

To estimate life years saved under the two intermediate assumptions about the life years lost as a result of O_3-related premature mortality, we turned to the epidemiological evidence of a statistically significant association between short-term exposures to O_3 and respiratory hospital admissions. This evidence suggests that these short-term exposures may exacerbate respiratory conditions that were preexisting. It is reasonable to suppose that some of these hospitalizations for respiratory illnesses on days of relatively high O_3 concentrations might result in death. It may also be the case that some individuals who did not go to the hospital might also die. We therefore looked for information on life expectancies of people with chronic respiratory conditions.

While there is information readily available in vital statistics sources on rates of death *from* chronic respiratory diseases, there is not similarly available information on rates of death *among that subpopulation who suffer from those diseases*. It is the latter rate – the rate of death among that subpopulation who suffers from those diseases – that is of interest.

A recent study of people with and without chronic obstructive pulmonary disease (COPD) provided data from which we were able to construct estimates of the mortality rates of interest. Mannino et al. (2006) followed a cohort of 15,440 subjects ages 43 to 66 for up to 11 years. The cohort subjects were selected from the larger cohort of the Atherosclerosis Risk in Communities (ARIC) study, which selected its subjects from the population of four U.S. communities by probability sampling.[8] The subjects in the Mannino study were limited to the ARIC participants who provided baseline information on respiratory symptoms and diagnoses, who underwent pulmonary function testing, and for whom follow-up data were available.

Using a modification of the criteria developed by the Global Initiative on Obstructive Lung Disease (GOLD), Mannino et al. (2006) classified the study subjects into COPD severity groups (or stages), with GOLD stage 0 (presence of respiratory symptoms in the absence of any lung function abnormality) being the least severe COPD group, and GOLD stages 3 and 4 being the most severe. The unadjusted death rates of the study participants (taken from Table 1 of Mannino et al., 2006), ratios of (unadjusted) death rates, and hazard ratios, based on Cox

[7] We calculated life expectancies from the mortality probabilities rather than using the life expectancies given in the CDC table because we were going to also calculate life expectancies for the subpopulations with severe COPD and with "average" COPD by adjusting the age-specific mortality probabilities and then calculating life expectancies using these adjusted probabilities.

[8] In one of the four communities probability sampling was used to select African-Americans only.

proportional hazard regressions, which took into account several covariates (including, among others, age, sex, race, smoking status, and education level) are shown in the table below. In addition, the right-most column of the table below shows the proportion of COPD subjects in the study in each GOLD category.

Table 8B-3. Death Rates and Hazard Ratios for Subjects with Varying Degrees of Severity of COPD (from Mannino et al., 2006)

GOLD* Category	N	Deaths	(%)	Person-Years	Death Rate per 1,000 Person-Years	Ratio of Death Rate to Death Rate for Normal Population	Hazard Ratio**	Proportion of COPD Subjects in GOLD Category
GOLD 3 or 4	271	92	33.9%	2,143	42.9	7.97	5.7	4.77%
GOLD 2	1,484	232	15.6%	12,852	18.1	3.35	2.4	26.14%
GOLD 1	1,679	137	8.2%	15,031	9.1	1.69	1.4	29.57%
GOLD 0	2,244	204	9.1%	20,191	10.1	1.88	1.5	39.52%
Restricted	1,101	150	13.6%	9,644	15.6	2.89	2.3	
Normal	8,661	427	4.9%	79,317	5.4	1.00	1.0	
Total	15,440	1,242	8.0%	139,178	8.9			

*Global Inititative on Obstructive Lung Disease (GOLD) guidelines for the staging of COPD severity.
**See Mannino et al. (2006), p. 117.

The ratios of unadjusted death rates are somewhat larger than the corresponding hazard ratios because these ratios were not adjusted for age. COPD is a progressive disease, so it would be expected that the proportion of older individuals would increase as the stages (and severity) increased, and this was indeed the case in the Mannino study. The hazard ratios, being based on regressions that took age into account, avoid this problem. We therefore used the hazard ratios to derive age-specific mortality rates for individuals with (1) severe COPD and (2) COPD of "average" severity. In particular, to derive age-specific mortality probabilities for the subpopulation with severe COPD, we multiplied each age-specific mortality probability for the general population by 5.7 (the hazard ratio for GOLD 3 or 4); to derive age-specific mortality probabilities for the subpopulation with "average" COPD, we multiplied each age-specific mortality probability for the general population by a weighted average of the GOLD category-specific hazard ratios, where the weight for a GOLD category was the proportion of COPD subjects in that GOLD category (given in the right-most column of Table 1 above). The weighted average hazard ratio was 1.906. Age-specific life expectancies were then derived for the severe COPD and "average" COPD subpopulations using these adjusted mortality probabilities and the method for calculating life expectancies described above.

Once an appropriate set of life expectancies has been determined (e.g., life expectancies for the general population or life expectancies for a subpopulation with severe COPD), these then provide the number of life years lost for an individual who dies at a given age. This information can then be combined with the estimated number of O_3-related premature deaths at each age calculated with BenMAP (see previous subsection). Because BenMAP calculates numbers of premature deaths avoided within age intervals, we can either allocate the premature deaths avoided within an age interval uniformly to the ages within the interval or, alternatively, we can calculate average life expectancies for the age intervals. We illustrate the first approach in

calculating O_3-related life years saved and the second approach in calculating $PM_{2.5}$-related life years saved (see Section 8B.4.4).

Total O_3-related life years gained was calculated as the sum of life years gained at each age:

$$Total\ life\ years\ gained = = \sum_{i=0}^{N} LE_i \times M_i$$

where LE_i is the remaining life expectancy for age i, M_i is the number of premature deaths avoided among individuals age i, and N is the oldest age considered.

For the purposes of determining cost effectiveness, it is also necessary to consider the time-dependent nature of the gains in life years. Standard economic theory suggests that benefits occurring in future years should be discounted relative to benefits occurring in the present. OMB and EPA guidance suggest discount rates of three and seven percent. Selection of a 3 percent discount rate is also consistent with recommendations from the U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine (Gold et al., 1996).

Discounted total life years gained is calculated as follows:

$$Discounted\ LY = \int_0^{LE} e^{-rt} dt,$$

where r is the discount rate, t indicates time, and LE is the life expectancy at the time when the premature death would have occurred. Because O_3-related premature mortality is associated only with short-term exposures, all O_3-related premature deaths are assumed to occur in the year of exposure. We therefore did not discount O_3-related premature deaths avoided.

Undiscounted age-specific life expectancies, and age-specific life expectancies using discount rates of 3 percent and 7 percent are given for the general population, the subpopulation of individuals with severe COPD, and the subpopulation of individuals with COPD of average severity in Tables 8B-4, 8B-5, and 8B-6, respectively. The O_3-related (discounted) life years saved, based on each of the two O_3-mortality studies and each of the assumptions about relevant life expectancies, are given, using 3 percent and 7 percent discount rates, in Tables 8B-7 and 8B-8, respectively. The O_3-related (discounted) life years saved, under the first assumption – that the observed statistical association between premature mortality and short-term exposures to O_3 is not actually a causal relationship – is zero in all cases (i.e., regardless of the mortality study used and the scenario considered), and is therefore not shown in these Tables.

Table 8B-4. Undiscounted and Discounted Age-Specific Life Expectancies for the General Population

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
0	0.006799	1,000,000	6,799	996,600	77.8	30.9	15.2
1	0.000483	993,201	480	992,961	77.3	30.8	15.2
2	0.000297	992,721	295	992,574	76.4	30.7	15.2
3	0.000224	992,427	222	992,315	75.4	30.6	15.2
4	0.000188	992,204	187	992,111	74.4	30.5	15.2
5	0.000171	992,017	170	991,932	73.4	30.4	15.2
6	0.000161	991,847	159	991,768	72.4	30.3	15.2
7	0.000151	991,688	149	991,613	71.4	30.2	15.2
8	0.000136	991,538	135	991,471	70.4	30.1	15.2
9	0.000119	991,403	118	991,345	69.5	29.9	15.1
10	0.000106	991,286	105	991,233	68.5	29.8	15.1
11	0.000112	991,180	111	991,125	67.5	29.7	15.1
12	0.000149	991,070	148	990,996	66.5	29.5	15.1
13	0.000227	990,922	225	990,809	65.5	29.4	15.1
14	0.000337	990,697	333	990,530	64.5	29.2	15.1
15	0.000460	990,363	456	990,135	63.5	29.1	15.1
16	0.000579	989,907	573	989,621	62.5	28.9	15.1
17	0.000684	989,334	677	988,996	61.6	28.8	15.0
18	0.000763	988,657	755	988,280	60.6	28.6	15.0
19	0.000819	987,902	809	987,498	59.7	28.4	15.0
20	0.000873	987,093	862	986,662	58.7	28.3	15.0
21	0.000926	986,231	913	985,775	57.8	28.1	15.0
22	0.000960	985,318	946	984,845	56.8	27.9	15.0
23	0.000972	984,372	957	983,893	55.9	27.8	14.9
24	0.000969	983,415	953	982,939	54.9	27.6	14.9
25	0.000960	982,462	943	981,991	54.0	27.4	14.9
26	0.000954	981,519	936	981,051	53.0	27.2	14.9
27	0.000952	980,583	933	980,117	52.1	27.0	14.8
28	0.000958	979,650	939	979,181	51.1	26.8	14.8
29	0.000973	978,712	952	978,235	50.2	26.5	14.8
30	0.000994	977,759	972	977,273	49.2	26.3	14.7
31	0.001023	976,787	999	976,287	48.3	26.1	14.7
32	0.001063	975,788	1,038	975,269	47.3	25.9	14.7
33	0.001119	974,750	1,091	974,205	46.4	25.6	14.6
34	0.001192	973,659	1,160	973,079	45.4	25.4	14.6
35	0.001275	972,499	1,240	971,879	44.5	25.1	14.5
36	0.001373	971,259	1,334	970,592	43.5	24.9	14.5
37	0.001493	969,925	1,448	969,201	42.6	24.6	14.4
38	0.001634	968,477	1,582	967,686	41.7	24.3	14.4
39	0.001788	966,895	1,729	966,031	40.7	24.0	14.3
40	0.001945	965,166	1,877	964,228	39.8	23.7	14.3
41	0.002107	963,290	2,029	962,275	38.9	23.5	14.2
42	0.002287	961,260	2,198	960,161	38.0	23.2	14.1
43	0.002494	959,062	2,392	957,866	37.0	22.8	14.0
44	0.002727	956,670	2,609	955,366	36.1	22.5	14.0
45	0.002982	954,061	2,845	952,639	35.2	22.2	13.9
46	0.003246	951,216	3,088	949,672	34.3	21.9	13.8

Table 8B-4. Undiscounted and Discounted Age-Specific Life Expectancies for the General Population (cont'd)

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
47	0.003520	948,129	3,337	946,460	33.5	21.6	13.7
48	0.003799	944,792	3,589	942,997	32.6	21.2	13.6
49	0.004088	941,203	3,848	939,279	31.7	20.9	13.5
50	0.004404	937,355	4,128	935,291	30.8	20.5	13.4
51	0.004750	933,227	4,433	931,010	30.0	20.2	13.3
52	0.005113	928,794	4,749	926,419	29.1	19.8	13.2
53	0.005488	924,045	5,071	921,510	28.2	19.4	13.0
54	0.005879	918,974	5,403	916,273	27.4	19.1	12.9
55	0.006295	913,571	5,751	910,696	26.6	18.7	12.7
56	0.006754	907,820	6,131	904,755	25.7	18.3	12.6
57	0.007280	901,689	6,564	898,407	24.9	17.9	12.4
58	0.007903	895,125	7,074	891,588	24.1	17.5	12.3
59	0.008633	888,051	7,667	884,217	23.3	17.1	12.1
60	0.009493	880,384	8,357	876,205	22.5	16.7	11.9
61	0.010449	872,027	9,112	867,471	21.7	16.2	11.8
62	0.011447	862,915	9,878	857,976	20.9	15.8	11.6
63	0.012428	853,037	10,601	847,736	20.1	15.4	11.4
64	0.013408	842,435	11,295	836,788	19.4	15.0	11.2
65	0.014473	831,140	12,029	825,126	18.6	14.5	11.0
66	0.015703	819,111	12,863	812,680	17.9	14.1	10.7
67	0.017081	806,249	13,771	799,363	17.2	13.7	10.5
68	0.018623	792,477	14,758	785,098	16.5	13.2	10.3
69	0.020322	777,719	15,805	769,817	15.8	12.8	10.0
70	0.022104	761,915	16,841	753,494	15.1	12.3	9.8
71	0.024023	745,073	17,899	736,124	14.4	11.9	9.5
72	0.026216	727,174	19,064	717,642	13.7	11.5	9.3
73	0.028745	708,110	20,355	697,933	13.1	11.0	9.0
74	0.031561	687,756	21,706	676,903	12.5	10.6	8.7
75	0.034427	666,050	22,930	654,585	11.9	10.2	8.4
76	0.037379	643,120	24,039	631,100	11.3	9.7	8.2
77	0.040756	619,080	25,231	606,465	10.7	9.3	7.9
78	0.044764	593,849	26,583	580,558	10.1	8.9	7.6
79	0.049395	567,266	28,020	553,256	9.6	8.5	7.3
80	0.054471	539,246	29,373	524,560	9.0	8.1	7.0
81	0.059772	509,873	30,476	494,635	8.5	7.7	6.7
82	0.065438	479,397	31,371	463,712	8.1	7.3	6.4
83	0.071598	448,026	32,078	431,987	7.6	6.9	6.1
84	0.078516	415,949	32,659	399,619	7.1	6.5	5.8
85	0.085898	383,290	32,924	366,828	6.7	6.2	5.6
86	0.093895	350,366	32,897	333,917	6.3	5.8	5.3
87	0.102542	317,468	32,554	301,192	5.9	5.5	5.0
88	0.111875	284,915	31,875	268,977	5.5	5.1	4.7
89	0.121928	253,040	30,853	237,613	5.1	4.8	4.5
90	0.132733	222,187	29,492	207,441	4.8	4.5	4.2
91	0.144318	192,695	27,809	178,791	4.4	4.2	3.9
92	0.156707	164,886	25,839	151,967	4.1	3.9	3.7
93	0.169922	139,047	23,627	127,234	3.7	3.6	3.4
94	0.183975	115,420	21,234	104,803	3.4	3.3	3.1
95	0.198875	94,186	18,731	84,820	3.0	3.0	2.8
96	0.214620	75,454	16,194	67,357	2.7	2.6	2.5
97	0.231201	59,260	13,701	52,410	2.3	2.2	2.2
98	0.248600	45,559	11,326	39,896	1.8	1.8	1.8
99	0.266786	34,233	9,133	29,667	1.2	1.2	1.2
100	1.000000	25,100	25,100	12,550	0.5	0.5	0.5

*Mortality probabilities for the general population taken from Table 1. Life table for the total population: United States, 2004. CDC National Vital Statistics Reports, Vol. 56, No. 9, December 28, 2007
http://www.cdc.gov/nchs/data/nvsr/nvsr56/nvsr56_09.pdf

Table 8B-5. Undiscounted and Discounted Age-Specific Life Expectancies for the Subpopulation with Severe COPD

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
0	0.038755	1,000,000	38,755	980,622	54.5	27.5	14.9
1	0.002752	961,245	2,646	959,922	55.7	27.7	14.9
2	0.001692	958,599	1,622	957,788	54.9	27.5	14.9
3	0.001277	956,977	1,222	956,366	53.9	27.4	14.9
4	0.001074	955,755	1,026	955,242	53.0	27.2	14.9
5	0.000978	954,729	933	954,263	52.1	27.0	14.8
6	0.000916	953,796	873	953,359	51.1	26.8	14.8
7	0.000859	952,923	819	952,513	50.2	26.5	14.8
8	0.000777	952,104	739	951,734	49.2	26.3	14.7
9	0.000677	951,365	644	951,043	48.2	26.1	14.7
10	0.000606	950,721	576	950,433	47.3	25.8	14.7
11	0.000636	950,145	605	949,842	46.3	25.6	14.6
12	0.000850	949,540	807	949,137	45.3	25.3	14.6
13	0.001295	948,733	1,229	948,119	44.4	25.1	14.5
14	0.001918	947,505	1,818	946,596	43.4	24.8	14.5
15	0.002625	945,687	2,482	944,446	42.5	24.6	14.4
16	0.003301	943,205	3,113	941,648	41.6	24.3	14.4
17	0.003901	940,092	3,667	938,258	40.8	24.0	14.3
18	0.004351	936,424	4,075	934,387	39.9	23.8	14.3
19	0.004671	932,350	4,355	930,172	39.1	23.5	14.2
20	0.004976	927,995	4,618	925,686	38.3	23.3	14.1
21	0.005278	923,377	4,873	920,941	37.5	23.0	14.1
22	0.005472	918,504	5,026	915,991	36.7	22.7	14.0
23	0.005542	913,478	5,063	910,947	35.9	22.4	13.9
24	0.005522	908,415	5,016	905,907	35.1	22.2	13.9
25	0.005470	903,399	4,942	900,928	34.2	21.9	13.8
26	0.005436	898,458	4,884	896,016	33.4	21.6	13.7
27	0.005425	893,573	4,847	891,150	32.6	21.2	13.6
28	0.005461	888,726	4,853	886,300	31.8	20.9	13.5
29	0.005547	883,873	4,903	881,422	31.0	20.6	13.4
30	0.005668	878,970	4,982	876,479	30.1	20.2	13.3
31	0.005830	873,988	5,095	871,440	29.3	19.9	13.2
32	0.006061	868,893	5,266	866,260	28.5	19.5	13.1
33	0.006380	863,626	5,510	860,872	27.6	19.2	12.9
34	0.006792	858,117	5,828	855,203	26.8	18.8	12.8
35	0.007269	852,289	6,195	849,191	26.0	18.4	12.7
36	0.007827	846,094	6,622	842,783	25.2	18.0	12.5
37	0.008510	839,472	7,144	835,900	24.4	17.6	12.3
38	0.009312	832,328	7,750	828,452	23.6	17.2	12.2
39	0.010191	824,577	8,403	820,376	22.8	16.8	12.0
40	0.011084	816,174	9,047	811,651	22.0	16.4	11.8
41	0.012008	807,128	9,692	802,282	21.3	16.0	11.7
42	0.013035	797,436	10,395	792,238	20.5	15.6	11.5
43	0.014215	787,041	11,187	781,447	19.8	15.2	11.3
44	0.015546	775,854	12,061	769,823	19.1	14.8	11.1
45	0.016996	763,792	12,981	757,301	18.4	14.4	10.9
46	0.018503	750,811	13,892	743,865	17.7	14.0	10.7

Table 8B-5. Undiscounted and Discounted Age-Specific Life Expectancies for the Subpopulation with Severe COPD (cont'd)

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
47	0.020061	736,919	14,784	729,527	17.0	13.6	10.4
48	0.021652	722,135	15,636	714,317	16.3	13.1	10.2
49	0.023303	706,500	16,464	698,268	15.7	12.7	10.0
50	0.025103	690,036	17,322	681,375	15.0	12.3	9.8
51	0.027075	672,714	18,214	663,607	14.4	11.9	9.5
52	0.029144	654,500	19,075	644,963	13.8	11.5	9.3
53	0.031280	635,425	19,876	625,487	13.2	11.1	9.0
54	0.033512	615,549	20,628	605,235	12.6	10.7	8.8
55	0.035880	594,921	21,346	584,248	12.0	10.3	8.5
56	0.038497	573,575	22,081	562,535	11.5	9.9	8.2
57	0.041497	551,494	22,885	540,052	10.9	9.5	8.0
58	0.045046	528,609	23,812	516,703	10.3	9.0	7.7
59	0.049211	504,797	24,842	492,376	9.8	8.6	7.4
60	0.054108	479,956	25,969	466,971	9.3	8.2	7.1
61	0.059560	453,986	27,040	440,467	8.8	7.9	6.9
62	0.065249	426,947	27,858	413,018	8.3	7.5	6.6
63	0.070839	399,089	28,271	384,953	7.9	7.1	6.3
64	0.076425	370,818	28,340	356,648	7.4	6.8	6.0
65	0.082495	342,478	28,253	328,352	7.0	6.4	5.8
66	0.089507	314,225	28,125	300,163	6.6	6.1	5.5
67	0.097361	286,100	27,855	272,173	6.2	5.7	5.2
68	0.106149	258,245	27,413	244,539	5.8	5.4	5.0
69	0.115833	230,833	26,738	217,463	5.4	5.1	4.7
70	0.125993	204,094	25,714	191,237	5.1	4.8	4.4
71	0.136933	178,380	24,426	166,167	4.7	4.5	4.2
72	0.149433	153,954	23,006	142,451	4.4	4.2	3.9
73	0.163847	130,948	21,455	120,220	4.1	3.9	3.7
74	0.179896	109,493	19,697	99,644	3.8	3.6	3.5
75	0.196231	89,795	17,621	80,985	3.5	3.4	3.2
76	0.213062	72,175	15,378	64,486	3.2	3.1	3.0
77	0.232309	56,797	13,194	50,200	3.0	2.9	2.8
78	0.255152	43,603	11,125	38,040	2.7	2.7	2.6
79	0.281552	32,477	9,144	27,905	2.5	2.4	2.4
80	0.310486	23,333	7,245	19,711	2.3	2.2	2.2
81	0.340699	16,089	5,481	13,348	2.1	2.0	2.0
82	0.372994	10,607	3,956	8,629	1.9	1.9	1.8
83	0.408108	6,651	2,714	5,294	1.7	1.7	1.7
84	0.447543	3,937	1,762	3,056	1.5	1.5	1.5
85	0.489619	2,175	1,065	1,642	1.4	1.4	1.4
86	0.535199	1,110	594	813	1.3	1.3	1.2
87	0.584489	516	302	365	1.1	1.1	1.1
88	0.637689	214	137	146	1.0	1.0	1.0
89	0.694992	78	54	51	0.9	0.9	0.9
90	0.756579	24	18	15	0.8	0.8	0.8
91	0.822612	6	5	3	0.6	0.6	0.6
92	0.893232	1	0	0	0.0	0.0	0.0

*Mortality probabilities derived from mortality probabilities for the general population by multiplying by the hazard ratio (5.7) for GOLD 3 or 4, from Mannino et al. (2006).

Table 8B-6. Undiscounted and Discounted Age-Specific Life Expectancies for the Subpopulation with COPD of Average Severity

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
0	0.012960	1,000,000	12,960	993,520	69.6	29.9	15.1
1	0.000920	987,040	908	986,586	69.5	29.9	15.1
2	0.000566	986,132	558	985,853	68.6	29.8	15.1
3	0.000427	985,574	421	985,363	67.6	29.7	15.1
4	0.000359	985,153	354	984,976	66.7	29.5	15.1
5	0.000327	984,799	322	984,638	65.7	29.4	15.1
6	0.000306	984,477	301	984,326	64.7	29.3	15.1
7	0.000287	984,176	283	984,034	63.7	29.1	15.1
8	0.000260	983,893	256	983,765	62.7	29.0	15.1
9	0.000226	983,638	223	983,526	61.8	28.8	15.1
10	0.000203	983,415	199	983,315	60.8	28.6	15.0
11	0.000213	983,216	209	983,111	59.8	28.5	15.0
12	0.000284	983,006	279	982,867	58.8	28.3	15.0
13	0.000433	982,727	426	982,514	57.8	28.1	15.0
14	0.000642	982,302	630	981,986	56.8	27.9	15.0
15	0.000878	981,671	862	981,241	55.9	27.8	14.9
16	0.001104	980,810	1,083	980,268	54.9	27.6	14.9
17	0.001304	979,727	1,278	979,088	54.0	27.4	14.9
18	0.001455	978,449	1,424	977,737	53.1	27.2	14.9
19	0.001562	977,025	1,526	976,262	52.1	27.0	14.8
20	0.001664	975,499	1,623	974,688	51.2	26.8	14.8
21	0.001765	973,876	1,719	973,017	50.3	26.6	14.8
22	0.001830	972,157	1,779	971,268	49.4	26.4	14.7
23	0.001853	970,378	1,798	969,479	48.5	26.1	14.7
24	0.001846	968,580	1,788	967,686	47.6	25.9	14.7
25	0.001829	966,792	1,769	965,907	46.7	25.7	14.6
26	0.001818	965,023	1,754	964,146	45.7	25.5	14.6
27	0.001814	963,269	1,747	962,395	44.8	25.2	14.5
28	0.001826	961,521	1,756	960,643	43.9	25.0	14.5
29	0.001855	959,766	1,780	958,875	43.0	24.7	14.5
30	0.001896	957,985	1,816	957,077	42.1	24.4	14.4
31	0.001949	956,169	1,864	955,237	41.1	24.2	14.3
32	0.002027	954,305	1,934	953,338	40.2	23.9	14.3
33	0.002133	952,371	2,032	951,355	39.3	23.6	14.2
34	0.002271	950,339	2,158	949,260	38.4	23.3	14.1
35	0.002431	948,181	2,305	947,028	37.5	23.0	14.1
36	0.002617	945,876	2,476	944,638	36.6	22.7	14.0
37	0.002846	943,400	2,685	942,058	35.7	22.4	13.9
38	0.003114	940,716	2,929	939,251	34.8	22.0	13.8
39	0.003408	937,786	3,196	936,189	33.9	21.7	13.7
40	0.003707	934,591	3,464	932,859	33.0	21.4	13.6
41	0.004016	931,127	3,739	929,257	32.1	21.0	13.5
42	0.004359	927,388	4,042	925,366	31.2	20.7	13.4
43	0.004753	923,345	4,389	921,151	30.4	20.3	13.3
44	0.005199	918,956	4,777	916,567	29.5	20.0	13.2
45	0.005683	914,179	5,196	911,581	28.7	19.6	13.1
46	0.006187	908,983	5,624	906,171	27.8	19.2	13.0

Table 8B-6. Undiscounted and Discounted Age-Specific Life Expectancies for the Subpopulation with COPD of Average Severity (cont'd)

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
47	0.006709	903,359	6,060	900,329	27.0	18.9	12.8
48	0.007241	897,298	6,497	894,050	26.2	18.5	12.7
49	0.007793	890,801	6,942	887,331	25.3	18.1	12.5
50	0.008395	883,860	7,420	880,150	24.5	17.7	12.4
51	0.009054	876,440	7,935	872,472	23.7	17.3	12.2
52	0.009746	868,505	8,464	864,273	23.0	16.9	12.1
53	0.010460	860,040	8,996	855,542	22.2	16.5	11.9
54	0.011207	851,044	9,537	846,276	21.4	16.1	11.7
55	0.011999	841,507	10,097	836,458	20.6	15.7	11.5
56	0.012874	831,410	10,703	826,058	19.9	15.3	11.3
57	0.013877	820,707	11,389	815,012	19.1	14.8	11.1
58	0.015064	809,318	12,191	803,222	18.4	14.4	10.9
59	0.016456	797,127	13,118	790,568	17.7	14.0	10.7
60	0.018094	784,009	14,186	776,916	17.0	13.5	10.4
61	0.019917	769,823	15,333	762,157	16.3	13.1	10.2
62	0.021820	754,490	16,463	746,259	15.6	12.7	10.0
63	0.023689	738,028	17,483	729,286	14.9	12.3	9.7
64	0.025557	720,545	18,415	711,337	14.3	11.8	9.5
65	0.027587	702,130	19,370	692,445	13.6	11.4	9.2
66	0.029932	682,760	20,436	672,542	13.0	11.0	8.9
67	0.032558	662,324	21,564	651,542	12.4	10.5	8.7
68	0.035497	640,760	22,745	629,388	11.8	10.1	8.4
69	0.038735	618,015	23,939	606,046	11.2	9.7	8.1
70	0.042133	594,076	25,030	581,561	10.6	9.3	7.8
71	0.045791	569,046	26,057	556,017	10.1	8.9	7.6
72	0.049971	542,989	27,134	529,422	9.6	8.4	7.3
73	0.054791	515,855	28,264	501,723	9.0	8.0	7.0
74	0.060158	487,591	29,333	472,924	8.5	7.6	6.7
75	0.065621	458,258	30,071	443,223	8.0	7.3	6.4
76	0.071249	428,187	30,508	412,933	7.6	6.9	6.1
77	0.077685	397,679	30,894	382,232	7.1	6.5	5.8
78	0.085324	366,785	31,296	351,137	6.7	6.1	5.6
79	0.094152	335,489	31,587	319,696	6.2	5.8	5.3
80	0.103828	303,902	31,554	288,125	5.8	5.4	5.0
81	0.113932	272,349	31,029	256,834	5.5	5.1	4.7
82	0.124731	241,319	30,100	226,269	5.1	4.8	4.5
83	0.136473	211,219	28,826	196,806	4.8	4.5	4.2
84	0.149661	182,394	27,297	168,745	4.4	4.2	4.0
85	0.163731	155,096	25,394	142,399	4.1	3.9	3.7
86	0.178974	129,702	23,213	118,096	3.8	3.7	3.5
87	0.195456	106,489	20,814	96,082	3.5	3.4	3.3
88	0.213247	85,675	18,270	76,540	3.3	3.2	3.1
89	0.232409	67,405	15,666	59,572	3.0	3.0	2.8
90	0.253004	51,740	13,090	45,194	2.8	2.7	2.7
91	0.275086	38,649	10,632	33,333	2.6	2.5	2.5
92	0.298702	28,017	8,369	23,833	2.4	2.4	2.3
93	0.323890	19,649	6,364	16,467	2.2	2.2	2.1
94	0.350677	13,285	4,659	10,955	2.0	2.0	2.0
95	0.379078	8,626	3,270	6,991	1.9	1.8	1.8
96	0.409089	5,356	2,191	4,261	1.7	1.7	1.6
97	0.440695	3,165	1,395	2,468	1.5	1.5	1.5
98	0.473858	1,770	839	1,351	1.3	1.3	1.3
99	0.508523	931	474	695	1.0	1.0	1.0
100	1.000000	458	458	229	0.5	0.5	0.5

*Mortality probabilities derived from mortality probabilities for the general population (see Table 2) by multiplying by the weighted average of hazard ratios for the GOLD severity categories (1.906) from Mannino et al. (2006).

Table 8B-7. Estimated O_3-Related Life Years Saved in 2020 and in 2030 Under the Final SSI & RME Rule, Using a 3 Percent Discount Rate

Estimated O_3-Related Life Years Saved (95% CI)*				
	2020		2030	
Mortality Study:	Bell et al (2004)	Levy et al. (2005)	Bell et al (2004)	Levy et al. (2005)
Assuming Life Expectancies of the General Population	500 (150 - 800)	2,200 (1,500 - 2,900)	700 (250 - 1,200)	3,500 (2,400 - 4,600)
Assuming Life Expectancies of the Sub-Population with COPD of Average Severity	360 (120 - 600)	1,700 (1,200 - 2,200)	560 (180 - 900)	2,700 (1,800 - 3,500)
Assuming Life Expectancies of the Sub-Population with Severe COPD	190 (60 - 320)	1,000 (700 - 1,300)	290 (100 - 490)	1,500 (1,000 - 1,900)

*95 percent confidence or credible intervals are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

Table 8B-8. Estimated O_3-Related Life Years Saved in 2020 and in 2030 Under the Final SSI & RME Rule, Using a 7 Percent Discount Rate

Estimated O_3-Related Life Years Saved (95% CI)*				
	2020		2030	
Mortality Study:	Bell et al (2004)	Levy et al. (2005)	Bell et al (2004)	Levy et al. (2005)
Assuming Life Expectancies of the General Population	360 (120 - 600)	1,700 (1,200 - 2,200)	590 (190 - 1,000)	2,700 (1,900 - 3,500)
Assuming Life Expectancies of the Sub-Population with COPD of Average Severity	290 (90 - 500)	1,400 (900 - 1,800)	460 (150 - 800)	2,100 (1,500 - 2,800)
Assuming Life Expectancies of the Sub-Population with Severe COPD	170 (50 - 280)	800 (600 - 1,100)	250 (80 - 430)	1,200 (800 - 1,600)

*95 percent confidence or credible intervals are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

8B.4.3 Reductions in $PM_{2.5}$-Related Premature Deaths

To generate $PM_{2.5}$-related health outcomes, we used the same framework as for the benefit-cost analysis described in Chapter 8 and briefly summarized above in the introductory portion of Section 8B.4.

As in several recent air pollution health impact assessments (e.g., Kunzli et al., 2000; EPA, 2004), we focused on the prospective cohort long-term exposure studies in deriving the health impact function for the estimate of premature mortality. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli et al., 2001; NRC, 2002). We selected an effect estimate from the extended analysis of the ACS cohort (Pope et al., 2002) as well as from the Harvard Six City Study (Laden et al., 2006). Given the focus in

this analysis on developing a broader expression of uncertainties in the benefits estimates, and the weight that was placed on both the ACS and Harvard Six-city studies by experts participating in the $PM_{2.5}$ mortality expert elicitation, we elected to provide estimates derived from both Pope et al. (2002) and Laden et al. (2006).

This latest re-analysis of the ACS cohort data (Pope et al, 2002) provides additional refinements to the analysis of PM-related mortality by (a) extending the follow-up period for the ACS study subjects to 16 years, which triples the size of the mortality data set; (b) substantially increasing exposure data, including consideration for cohort exposure to $PM_{2.5}$ following implementation of $PM_{2.5}$ standard in 1999; (c) controlling for a variety of personal risk factors including occupational exposure and diet; and (d) using advanced statistical methods to evaluate specific issues that can adversely affect risk estimates, including the possibility of spatial autocorrelation of survival times in communities located near each other. The effect estimate from Pope et al. (2002) quantifies the relationship between annual mean $PM_{2.5}$ levels and all-cause mortality in adults 30 and older. We selected the effect estimate estimated using the measure of PM representing average exposure over the follow-up period, calculated as the average of 1979–1984 and 1999–2000 $PM_{2.5}$ levels. The effect estimate from this study is 0.0058, which is equivalent to a relative risk of 1.06 for a 10 μg change in $PM_{2.5}$.

A recent follow up to the Harvard 6-city study (Laden et al., 2006) both confirmed the effect size from the first study and provided additional confirmation that reductions in $PM_{2.5}$ directly result in 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 $PM_{2.5}$. The effect estimate obtained from Laden et al. (2006) is 0.0148, which is equivalent to a relative risk of 1.16 for a 10 $\mu g/m^3$ change in $PM_{2.5}$.

Age, cause, and county-specific mortality rates were obtained from CDC for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at http://wonder.cdc.gov/. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across 3 years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25- to 34-year old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups.

The reductions in incidence of $PM_{2.5}$-related premature mortality within each age group associated with the Final SSI & RME Rule in 2020 and 2030 are summarized in Table 8B-9.

Table 8B-9: Estimated Reduction in Incidence of $PM_{2.5}$-Related All-Cause Premature Mortality Under the Final SSI & RME Rule in 2020 and 2030

Age Interval	Reduction in $PM_{2.5}$-Related Premature Mortality (90% CI)*			
	2020		2030	
	Pope et al. (2002)	Laden et al. (2006)	Pope et al. (2002)	Laden et al. (2006)
25 - 29	---	3 (2 - 5)	---	4 (2 - 5)
30 - 34	1 (0 - 2)	3 (2 - 4)	2 (1 - 3)	4 (2 - 5)
35 - 44	3 (1 - 5)	7 (4 - 10)	5 (2 - 8)	11 (6 - 17)
45 - 54	5 (2 - 9)	12 (7 - 18)	9 (3 - 14)	20 (11 - 29)
55 - 64	13 (5 - 21)	29 (16 - 43)	22 (9 - 35)	50 (27 - 72)
65 - 74	19 (7 - 30)	42 (23 - 61)	51 (20 - 81)	110 (62 - 170)
75 - 84	31 (12 - 49)	69 (38 - 100)	69 (27 - 110)	160 (85 - 230)
85+	47 (18 - 75)	110 (57 - 150)	68 (27 - 110)	150 (84 - 220)
Total:	120 (47 - 190)	270 (150 - 390)	230 (88 - 360)	510 (280 - 750)

*90 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

8B.4.4 Life Years Saved as a Result of Reductions in $PM_{2.5}$-Related Mortality Risk

To calculate life years saved associated with a given change in air pollution, we used a life table approach coupled with age-specific estimates of reductions in premature mortality. We began with the complete unabridged life table for the United States in 2000, obtained from CDC (CDC, 2002). For each 1-year age interval (e.g., zero to one, one to two) the life table provides estimates of the baseline probability of dying during the interval, person years lived in the interval, and remaining life expectancy. From this unabridged life table, we constructed an abridged life table to match the age intervals for which we have predictions of changes in incidence of premature mortality. We used the abridgement method described in CDC (2002). Table 8B-10 presents the abridged life table for 10-year age intervals for adults over 30 (to match the Pope et al. [2002] study population). Note that the abridgement actually includes one 5-year interval, covering adults 30 to 34, with the remaining age intervals covering 10 years each. This is to provide conformity with the age intervals available for mortality rates.

From the abridged life table (Table 8B-10), we obtained the remaining life expectancy for each age cohort, conditional on surviving to that age. This is then the number of life years lost for an individual in the general population dying during that age interval. This information can then be combined with the estimated number of premature deaths in each age interval calculated with

BenMAP (see previous subsection). Total life years gained will then be the sum of life years gained in each age interval:

$$Total Life Years = \sum_{i=1}^{N} LE_i \times M_i ,$$

where LE_i is the remaining life expectancy for age interval i, M_i is the change in incidence of mortality in age interval i, and N is the number of age intervals.

As noted above, for the purposes of determining cost-effectiveness, it is also necessary to consider the time-dependent nature of the gains in life years. Standard economic theory suggests that benefits occurring in future years should be discounted relative to benefits occurring in the present. OMB and EPA guidance suggest discount rates of three and seven percent. Selection of a 3 percent discount rate is also consistent with recommendations from the U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine (Gold et al., 1996).

Table 8B-10. Abridged Life Table for the Total Population, United States, 2000

Age Interval		*Probability of Dying Between Ages x to x+1*	*Number Surviving to Age x*	*Number Dying Between Ages x to x+1*	*Person Years Lived Between Ages x to x+1*	*Total Number of Person Years Lived Above Age x*	*Expectation of Life at Age x*
Start Age	End Age	q_x	l_x	d_x	L_x	T_x	e_x
30	35	0.00577	97,696	564	487,130	4,723,539	48.3
35	45	0.01979	97,132	1,922	962,882	4,236,409	43.6
45	55	0.04303	95,210	4,097	934,026	3,273,527	34.4
55	65	0.09858	91,113	8,982	872,003	2,339,501	25.7
65	75	0.21779	82,131	17,887	740,927	1,467,498	17.9
75	85	0.45584	64,244	29,285	505,278	726,571	11.3
85	95	0.79256	34,959	27,707	196,269	221,293	6.3
95	100	0.75441	7,252	5,471	20,388	25,024	3.5
100+		1.00000	1,781	1,781	4,636	4,636	2.6

Unlike O_3-related premature deaths, $PM_{2.5}$-related premature deaths are associated with long-term exposures. We therefore did not assume that these deaths all occur in 2020 or 2030. The $PM_{2.5}$-related premature deaths avoided and associated life years saved are thus further discounted to account for the lag between the reduction in ambient $PM_{2.5}$ and the corresponding reduction in mortality risk. We used the same 20-year segmented lag structure that is used in the benefit-cost analysis (see Chapter 8).

The most complete estimate of the impacts of $PM_{2.5}$ on life years is calculated using the Pope et al. (2002) C-R function relating all-cause mortality in adults 30 and over with ambient $PM_{2.5}$ concentrations averaged over the periods 1979–1983 and 1999–2000. Use of all-cause mortality is appropriate if there are no differences in the life expectancy of individuals dying from air pollution-related causes and those dying from other causes. The argument that long-term exposure to $PM_{2.5}$ may affect mainly individuals with serious preexisting illnesses is not supported by current empirical studies. For example, the Krewski et al. (2000) ACS reanalysis suggests that the mortality risk is no greater for those with preexisting illness at time of enrollment in the study. Life expectancy for the general population in fact includes individuals with serious chronic illness. Mortality rates for the general population then reflect prevalence of chronic disease, and as populations age the prevalence of chronic disease increases.

The only reason one might use a lower life expectancy is if the population at risk from air pollution was limited solely to those with preexisting disease. Also, note that the OMB Circular A-4 notes that "if QALYs are used to evaluate a lifesaving rule aimed at a population that happens to experience a high rate of disability (i.e., where the rule is not designed to affect the disability), the number of life years saved should not necessarily be diminished simply because the rule saves lives of people with life-shortening disabilities. Both analytic simplicity and fairness suggest that the estimate number of life years saved for the disabled population should be based on average life expectancy information for the relevant age cohorts." As such, use of a general population life expectancy is preferred over disability-specific life expectancies. Our primary life years calculations are thus consistent with the concept of not penalizing individuals with disabling chronic health conditions by assessing them reduced benefits of mortality risk reductions. $PM_{2.5}$-Related life years saved under the Final SSI & RME Rule in 2020 and 2030 are given in Table 8B-11.

Table 8B-11. Estimated $PM_{2.5}$-Related Life Years Saved Under the Final SSI & RME Rule in 2020 and 2030

Estimated $PM_{2.5}$-Related Life Years Saved (95% CI)*				
	2020		2030	
	Pope et al (2002)	Laden et al. (2006)	Pope et al (2002)	Laden et al. (2006)
Discounted back to 2020 or 2030, using a 3 percent discount rate:	1,100 (400 - 1,800)	2,600 (1,400 - 4,000)	2,200 (900 - 3,500)	5,000 (2,700 - 7,000)
Discounted back to 2020 or 2030, using a 7 percent discount rate:	800 (300 - 1,200)	1,800 (1,000 - 2,500)	1,500 (600 - 2,400)	3,500 (1,900 - 5,100)

*95 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

For this analysis, direct impacts on life expectancy are measured only through the estimated change in mortality risk based on the Pope et al. (2002) C-R function. The SAB-HES has advised against including additional gains in life expectancy due to reductions in incidence of chronic disease or nonfatal heart attacks (EPA-SAB-COUNCIL-ADV-04-002). Although reductions in these endpoints are likely to result in increased life expectancy, the HES has suggested that the cohort design and relatively long follow-up period in the Pope et al. study

should capture any life-prolonging impacts associated with those endpoints. Impacts of CB and nonfatal heart attacks on quality of life will be captured separately in the QALY calculation as years lived with improved quality of life. The methods for calculating this benefit are discussed below.

8B.4.5 Calculating Changes in the Quality of Life Years ($PM_{2.5}$-Related Chronic Morbidity)

In addition to directly measuring the quantity of life gained, measured by life years, it may also be informative to measure gains in the quality of life. The indirect reductions in levels of $PM_{2.5}$ also lead to reductions in serious illnesses that affect quality of life. These include chronic bronchitis (CB) and cardiovascular disease, for which we are able to quantify changes in the incidence of nonfatal heart attacks. To capture these important benefits in the measure of effectiveness, they must first be converted into a life-year equivalent so that they can be combined with the direct gains in life expectancy.

For the cost effectiveness analyses for the PM and O_3 NAAQS RIAs, we developed estimates of the QALYs gained from reductions in the incidence of CB and nonfatal heart attacks associated with reductions in ambient $PM_{2.5}$. In general, QALY calculations require four elements:

1. the estimated change in incidence of the health condition,
2. the duration of the health condition,
3. the quality-of-life weight with the health condition, and
4. the quality-of-life weight without the health condition (i.e., the baseline health state).

The first element is derived using the health impact function approach. The second element is based on the medical literature for each health condition. The third and fourth elements are derived from the medical cost-effectiveness and cost-utility literature. In the following two subsections, we discuss the choices of elements for CB and nonfatal heart attacks.

The preferred source of quality-of-life weights are those based on community preferences, rather than patient or clinician ratings (Gold et al., 1996). Several methods are used to estimate quality-of-life weights. These include rating scale, standard gamble, time trade-off, and person trade-off approaches (Gold, Stevenson, and Fryback, 2002). Only the standard gamble approach is completely consistent with utility theory. However, the time trade-off method has also been widely applied in eliciting community preferences (Gold, Stevenson, and Fryback, 2002).

Quality-of-life weights can be directly elicited for individual specific health states or for a more general set of activity restrictions and health states that can then be used to construct QALY weights for specific conditions (Horsman et al., 2003; Kind, 1996). For this analysis, we used weights based on community-based preferences, using time trade-off or standard gamble when available. In some cases, we used patient or clinician ratings when no community preference-based weights were available. Sources for weights are discussed in more detail below. Table 8B-12 summarizes the key inputs for calculating QALYs associated with chronic health endpoints.

Table 8B-12. Summary of Key Parameters Used in QALY Calculations for Chronic Disease Endpoints

Parameter	*Value(s)*	*Source(s)*
Discount rate	0.03 (0.07 sensitivity analysis)	Gold et al. (1996), U.S. EPA (2000), U.S. OMB (2003)
Quality of life preference score for chronic bronchitis	0.5 – 0.7	Triangular distribution centered at 0.7 with upper bound at 0.9 (Vos, 1999a) (slightly better than a mild/moderate case) and a lower bound at 0.5 (average weight for a severe case based on Vos [1999a] and Smith and Peske [1994])
Duration of acute phase of acute myocardial infarction (AMI)	5.5 days – 22 days	Uniform distribution with lower bound based on average length of stay for an AMI (AHRQ, 2000) and upper bound based on Vos (1999b).
Probability of CHF post AMI	0.2	Vos, 1999a (WHO Burden of Disease Study, based on Cowie et al., 1997)
Probability of angina post AMI	0.51	American Heart Association, 2003 (Calculated as the population with angina divided by the total population with heart disease)
Quality-of-life preference score for post-AMI with CHF (no angina)	0.80 – 0.89	Uniform distribution with lower bound at 0.80 (Stinnett et al., 1996) and upper bound at 0.89 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with CHF and angina	0.76 – 0.85	Uniform distribution with lower bound at 0.76 (Stinnett et al., 1996, adjusted for severity) and upper bound at 0.85 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with angina (no CHF)	0.7 – 0.89	Uniform distribution with lower bound at 0.7, based on the standard gamble elicitation method (Pliskin, Stason, and Weinstein, 1981) and upper bound at 0.89, based on the time trade-off method (Kuntz et al., 1996).
Quality-of-life preference score for post-AMI (no angina, no CHF)	0.93	Only one value available from the literature. Thus, no distribution is specified. Source of value is Kuntz et al. (1996).

8B.4.5.1 Calculating QALYs Associated with Reductions in the Incidence of Chronic Bronchitis

CB is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). For gains in quality of life resulting from reduced incidences of PM-induced CB, discounted QALYs are calculated as

$$DISCOUNTED\ QALY\ GAINED = \sum_i \Delta CB_i \times D_i^* \times \left(w_i - w_i^{CB}\right)$$

where ΔCB_i is the number of incidences of CB avoided in age interval i, w_i is the average QALY weight for the *i*th age interval, w_i^{CB} is the QALY weight associated with CB in the *i*th age interval, and D_i^* is the discounted duration of life with CB for individuals with onset of disease in the *i*th age interval, equal to $\int_0^{D_i} e^{-rt} dt$, where D_i is the duration of life with CB for individuals with onset of disease the *i*th age interval.

A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993) and Abbey et al. (1995) provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Only the Abbey et al. (1995) study was used, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of CB. The number of cases of CB in each age interval was derived by applying the impact function from Abbey et al. (1995) to the population in each age interval with the appropriate baseline incidence rate.[9] The effect estimate from the Abbey et al. (1995) study is 0.0137, which, based on the logistic specification of the model, is equivalent to a relative risk of 1.15 for a 10 μg change in $PM_{2.5}$. Table 8B-13 presents the estimated reduction in new incidences of CB associated with the Final SSI & RME Rule in 2020 and 2030.

CB is assumed to persist for the remainder of an affected individual's lifespan. Duration of CB will thus equal life expectancy conditioned on having CB. CDC has estimated that COPD (of which CB is one element) results in an average loss of life years equal to 4.26 per COPD death, relative to a reference life expectancy of 75 years (CDC, 2003). Thus, we subtracted 4.26 from the remaining life expectancy for each age group, up to age 75. For age groups over 75, we applied the ratio of 4.26 to the life expectancy for the 65 to 74 year group (0.237) to the life expectancy for the 75 to 84 and 85 and up age groups to estimate potential life years lost and then subtracted that value from the base life expectancy.

[9] Prevalence rates for CB were obtained from the 1999 National Health Interview Survey (American Lung Association, 2002). Prevalence rates were available for three age groups: 18–44, 45–64, and 65 and older. Prevalence rates per person for these groups were 0.0367 for 18–44, 0.0505 for 45–64, and 0.0587 for 65 and older. The incidence rate for new cases of CB (0.00378 per person) was taken directly from Abbey et al. (1995).

Table 8B-13. Estimated Reduction in Incidence of Chronic Bronchitis Under the Final SSI & RME Rule in 2020 and 2030

	Reduction in $PM_{2.5}$-Related Chronic Bronchitis (90% CI)*	
Age Interval	2020	2030
27 - 34	18 (3 - 33)	22 (4 - 40)
35 - 44	15 (3 - 28)	26 (5 - 48)
45 - 54	14 (3 - 26)	22 (4 - 40)
55 - 64	16 (3 - 28)	22 (4 - 40)
65 - 74	11 (2 - 19)	21 (4 - 38)
75 - 84	6 (1 - 11)	12 (2 - 22)
85+	3 (1 - 5)	4 (1 - 8)
Total:	84 (16 - 150)	130 (24 - 240)

*90 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

Quality of life with chronic lung diseases has been examined in several studies. In an analysis of the impacts of environmental exposures to contaminants, de Hollander et al. (1999) assigned a weight of 0.69 to years lived with CB. This weight was based on physicians' evaluations of health states similar to CB. Salomon and Murray (2003) estimated a pooled weight of 0.77 based on visual analogue scale, time trade-off, standard gamble, and person trade-off techniques applied to a convenience sample of health professionals. The Harvard Center for Risk Analysis catalog of preference scores reports a weight of 0.40 for severe COPD, with a range from 0.2 to 0.8, based on the judgments of the study's authors (Bell et al., 2001). The Victoria Burden of Disease (BoD) study used a weight of 0.47 for severe COPD and 0.83 for mild to moderate COPD, based on an analysis by Stouthard et al. (1997) of chronic diseases in Dutch populations (Vos, 1999a). Based on the recommendations of Gold et al. (1996), quality-of-life weights based on community preferences are preferred for CEA of interventions affecting broad populations. Use of weights based on health professionals is not recommended. It is not clear from the Victoria BoD study whether the weights used for COPD are based on community preferences or judgments of health professionals. The Harvard catalog score is clearly identified as based on author judgment. Given the lack of a clear preferred weight, we selected a triangular distribution centered at 0.7 with an upper bound at 0.9 (slightly better than a mild/moderate case defined by the Victoria BoD study) and a lower bound at 0.5 based on the Victoria BoD study. We will need additional empirical data on quality of life with chronic respiratory diseases based on community preferences to improve our estimates.

Selection of a reference weight for the general population without CB is somewhat uncertain. It is clear that the general population is not in perfect health; however, there is some uncertainty as to whether individuals' ratings of health states are in reference to a perfect health state or to a generally achievable "normal" health state given age and general health status. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" (Gold et al., 1996). Following Carrothers, Evans, and Graham (2002), we assumed that the reference weight for the general population without CB is 0.95. To allow for uncertainty in this parameter, we assigned a triangular distribution around this weight, bounded by 0.9 and 1.0. Note that the reference weight for the general population is used solely to determine the incremental quality-of-life improvement applied to the duration of life that would have been lived with the chronic disease. For example, if CB has a quality-of-life weight of 0.7 relative to a reference quality-of-life weight of 0.9, then the incremental quality-of-life improvement in 0.2. If the reference quality-of-life weight is 0.95, then the incremental quality-of-life improvement is 0.25. As noted above, the population is assumed to have a reference weight of 1.0 for all life years gained due to mortality risk reductions.

We present discounted QALYs over the duration of the lifespan with CB using a 3 percent discount rate. Based on the assumptions defined above, we used Monte Carlo simulation methods as implemented in the Crystal Ball™ software program to develop the distribution of QALYs gained per incidence of CB for each age interval.[10] Based on the assumptions defined above, the mean 3 percent discounted QALY gained per incidence of CB for each age interval along with the 95 percent confidence interval resulting from the Monte Carlo simulation is presented in Table 8B-14. Table 8B-14 presents both the undiscounted and discounted QALYs gained per incidence, using a 3 percent discount rate.

[10] Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables. For more details, see Gentile (1998).

Table 8B-14. QALYs Gained per Avoided Incidence of CB

Age Interval		*QALYs Gained per Incidence*[a]	
Start Age	End Age	Undiscounted	Discounted (3%)
25	34	12.15 (4.40-19.95)	6.52 (2.36-10.71)
35	44	9.91 (3.54-16.10)	5.94 (2.12-9.66)
45	54	7.49 (2.71-12.34)	5.03 (1.82-8.29)
55	64	5.36 (1.95-8.80)	4.03 (1.47-6.61)
65	74	3.40 (1.22-5.64)	2.84 (1.02-4.71)
75	84	2.15 (0.77-3.49)	1.92 (0.69-3.13)
85+		0.79 (0.27-1.29)	0.77 (0.26-1.25)

[a] Mean of Monte Carlo generated distribution; 95% confidence interval presented in parentheses.

8B.4.5.2 Calculating QALYs Associated with Reductions in the Incidence of Nonfatal Myocardial Infarctions

Nonfatal heart attacks, or acute myocardial infarctions, require more complicated calculations to derive estimates of QALY impacts. The actual heart attack, which results when an area of the heart muscle dies or is permanently damaged because of oxygen deprivation, and subsequent emergency care are of relatively short duration. Many heart attacks result in sudden death. However, for survivors, the long-term impacts of advanced coronary heart disease (CHD) are potentially of long duration and can result in significant losses in quality of life and life expectancy.

In this phase of the analysis, we did not independently estimate the gains in life expectancy associated with reductions in nonfatal heart attacks. Based on recommendations from the SAB-HES, we assumed that all gains in life expectancy are captured in the estimates of reduced mortality risk provided by the Pope et al. (2002) analysis. We estimated only the change in quality of life over the period of life affected by the occurrence of a heart attack. This may understate the QALY impacts of nonfatal heart attacks but ensures that the overall QALY impact estimates across endpoints do not double-count potential life-year gains.

Our approach adapts a CHD model developed for the Victoria Burden of Disease study (Vos, 1999b). This model accounts for the lost quality of life during the heart attack and the possible health states following the heart attack. Figure 8B-1 shows the heart attack QALY model in diagrammatic form.

The total gain in QALYs is calculated as:

$$DISCOUNTED\ AMI\ QALY\ GAINED =$$

$$\sum_i \Delta AMI_i \times D_i^{*AMI} \times \left(w_i - w_i^{AMI}\right) + \sum_i \sum_{j=1}^{4} \Delta AMI_i \times p_j D_{ij}^{*PostAMI} \times \left(w_i - w_{ij}^{postAMI}\right)$$

where ΔAMI_i is the number of nonfatal acute myocardial infarctions avoided in age interval *i*, w_i^{AMI} is the QALY weight associated with the acute phase of the AMI, p_j is the probability of being in the *j*th post-AMI status, $w_{ij}^{postAMI}$ is the QALY weight associated with post-AMI health status *j*, w_i is the average QALY weight for age interval i, $D_i^{*AMI} = \int_{t=1}^{D_i^{AMI}} e^{-rt} dt$, the discounted value of D_i^{AMI}, the duration of the acute phase of the AMI, and $D_i^{*postAMI} = \int_{t=1}^{D_i^{postAMI}} e^{-rt} dt$, is the discounted value of $D_{ij}^{PostAMI}$, the duration of post-AMI health status *j*.

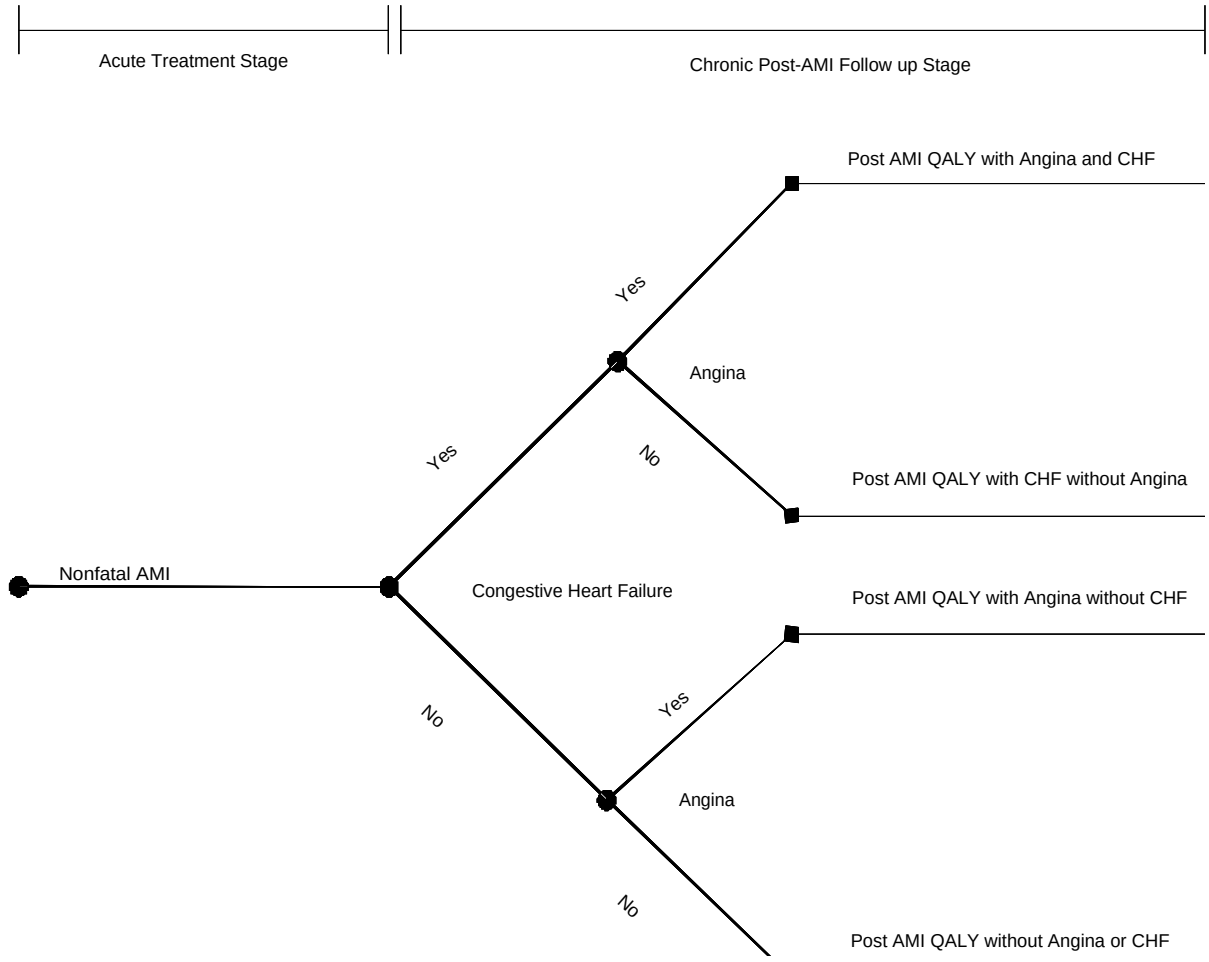

Figure 8B-1. Decision Tree Used in Modeling Gains in QALYs from Reduced Incidence of Nonfatal Acute Myocardial Infarctions

Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We used a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between $PM_{2.5}$ and nonfatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar (2000), show a consistent relationship between all cardiovascular hospital admissions, including for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on longer-term health costs and earnings, we chose to provide a separate estimate for nonfatal heart attacks based on the single available U.S. effect estimate. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the United States. These studies provide a weight of evidence for this type of effect. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much

the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other CHDs (Carthenon et al., 2002; Dekker et al., 2000; Liao et al., 1997, Tsuji et al., 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

The number of avoided nonfatal AMI in each age interval was derived by applying the impact function from Peters et al. (2001) to the population in each age interval with the appropriate baseline incidence rate.[11] The effect estimate from the Peters et al. (2001) study is 0.0241, which, based on the logistic specification of the model, is equivalent to a relative risk of 1.27 for a 10 μg change in $PM_{2.5}$. Table 8B-15 presents the estimated reduction in nonfatal AMI associated with the Final SSI & RME Rule in 2020 and 2030.

Table 8B-15. Estimated Reduction in Nonfatal Acute Myocardial Infarctions Under the Final SSI & RME Rule in 2020 and 2030

	Reduction in $PM_{2.5}$-Related Acute Myocardial Infarction (90% CI)*	
Age Interval	2020	2030
18 - 24	0 (0 - 0)	0 (0 - 0)
25 - 29	1 (1 - 2)	2 (1 - 3)
35 - 44	10 (5 - 14)	16 (9 - 23)
45 - 54	29 (16 - 42)	43 (23 - 63)
55 - 64	68 (37 - 98)	99 (53 - 140)
65 - 74	94 (51 - 140)	160 (84 - 230)
75 - 84	48 (26 - 69)	140 (76 - 210)
85+	42 (23 - 62)	67 (36 - 98)
Total:	290 (160 - 420)	530 (280 - 770)

*90 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

[11] Daily nonfatal myocardial infarction incidence rates per person were obtained from the 1999 National Hospital Discharge Survey (assuming all diagnosed nonfatal AMI visit the hospital). Age-specific rates for four regions are used in the analysis. Regional averages for populations 18 and older are 0.0000159 for the Northeast, 0.0000135 for the Midwest, 0.0000111 for the South, and 0.0000100 for the West.

Acute myocardial infarction results in significant loss of quality of life for a relatively short duration. The WHO Global Burden of Disease study, as reported in Vos (1999b), assumes that the acute phase of an acute myocardial infarction lasts for 0.06 years, or around 22 days. An alternative assumption is the acute phase is characterized by the average length of hospital stay for an AMI in the United States, which is 5.5 days, based on data from the Agency for Healthcare Research and Quality's Healthcare Cost and Utilization Project (HCUP).[12] We assumed a distribution of acute phase duration characterized by a uniform distribution between 5.5 and 22 days, noting that due to earlier discharges and in-home therapy available in the United States, duration of reduced quality of life may continue after discharge from the hospital. In the period during and directly following an AMI (the acute phase), we assigned a quality of life weight equal to 0.605, consistent with the weight for the period in treatment during and immediately after an attack (Vos, 1999b).

During the post-AMI period, a number of different health states can determine the loss in quality of life. We chose to classify post-AMI health status into four states defined by the presence or absence of angina and congestive heart failure (CHF). This makes a very explicit assumption that without the occurrence of an AMI, individuals would not experience either angina or CHF. If in fact individuals already have CHF or angina, then the quality of life gained will be overstated. We do not have information about the percentage of the population have been diagnosed with angina or CHF with no occurrence of an AMI. Nor do we have information on what proportion of the heart attacks occurring due to PM exposure are first heart attacks versus repeat attacks. Probabilities for the four post-AMI health states sum to one.

Given the occurrence of a nonfatal AMI, the probability of congestive heart failure is set at 0.2, following the heart disease model developed by Vos (1999b). The probability is based on a study by Cowie et al. (1997), which estimated that 20 percent of those surviving AMI develop heart failure, based on an analysis of the results of the Framingham Heart Study.

The probability of angina is based on the prevalence rate of angina in the U.S. population. Using data from the American Heart Association, we calculated the prevalence rate for angina by dividing the estimated number of people with angina (6.6 million) by the estimated number of people with CHD of all types (12.9 million). We then assumed that the prevalence of angina in the population surviving an AMI is similar to the prevalence of angina in the total population with CHD. The estimated prevalence rate is 51 percent, so the probability of angina is 0.51.

Combining these factors leads to the probabilities for each of the four health states as follows:

I. Post AMI with CHF and angina = 0.102

II. Post AMI with CHF without angina = 0.098

III. Post AMI with angina without CHF = 0.408

IV. Post AMI without angina or CHF = 0.392

[12] Average length of stay estimated from the HCUP data includes all discharges, including those due to death. As such, the 5.5-day average length of stay is likely an underestimate of the average length of stay for AMI admissions where the patient is discharged alive.

Duration of post-AMI health states varies, based in part on assumptions regarding life expectancy with post-AMI complicating health conditions. Based on the model used for established market economies (EME) in the WHO Global Burden of Disease study, as reported in Vos (1999b), we assumed that individuals with CHF have a relatively short remaining life expectancy and thus a relatively short period with reduced quality of life (recall that gains in life expectancy are assumed to be captured by the cohort estimates of reduced mortality risk). Table 8B-16 provides the duration (both discounted and undiscounted) of CHF assumed for post-AMI cases by age interval.

Table 8B-16. Assumed Duration of Congestive Heart Failure

Age Interval		*Duration of Heart Failure (years)*	
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	7.11	6.51
25	34	6.98	6.40
35	44	6.49	6.00
45	54	5.31	4.99
55	64	1.96	1.93
65	74	1.71	1.69
75	84	1.52	1.50
85+		1.52	1.50

Duration of health states without CHF is assumed to be equal to the life expectancy of individuals conditional on surviving an AMI. Ganz et al. (2000) note that "Because patients with a history of myocardial infarction have a higher chance of dying of CHD that is unrelated to recurrent myocardial infarction (for example, arrhythmia), this cohort has a higher risk for death from causes other than myocardial infarction or stroke than does an unselected population." They go on to specify a mortality risk ratio of 1.52 for mortality from other causes for the cohort of individuals with a previous (nonfatal) AMI. The risk ratio is relative to all-cause mortality for an age-matched unselected population (i.e., general population). We adopted the same ratios and applied them to each age-specific all-cause mortality rate to derive life expectancies (both discounted and undiscounted) for each age group after an AMI, presented in Table 8B-17. These life expectancies were then used to represent the duration of non-CHF post-AMI health states (III and IV).

Table 8B-17. Assumed Duration of Non-CHF Post-AMI Health States

Age Interval		Post-AMI Years of Life Expectancy (non-CHF)	
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	55.5	27.68
25	34	46.1	25.54
35	44	36.8	22.76
45	54	27.9	19.28
55	64	19.8	15.21
65	74	12.8	10.82
75	84	7.4	6.75
85+		3.6	3.47

For the four post-AMI health states, we used QALY weights based on preferences for the combined conditions characterizing each health state. A number of estimates of QALY weights are available for post-AMI health conditions.

The first two health states are characterized by the presence of CHF, with or without angina. The Harvard Center for Risk Analysis catalog of preference scores provides several specific weights for CHF with and without mild or severe angina and one set specific to post-AMI CHF. Following the Victoria Burden of Disease model, we assumed that most cases of angina will be treated and thus kept at a mild to moderate state. We thus focused our selection on QALY weights for mild to moderate angina. The Harvard database includes two sets of community preference-based scores for CHF (Stinnett et al., 1996; Kuntz et al., 1996). The scores for CHF with angina range from 0.736 to 0.85. The lower of the two scores is based on angina in general with no delineation by severity. Based on the range of the scores for mild to severe cases of angina in the second study, one can infer that an average case of angina has a score around 0.96 of the score for a mild case. Applying this adjustment raises the lower end of the range of preference scores for a mild case of angina to 0.76. We selected a uniform distribution over the range 0.76 to 0.85 for CHF with mild angina, with a midpoint of 0.81. The same two studies in the Harvard catalog also provide weights for CHF without angina. These scores range from 0.801 to 0.89. We selected a uniform distribution over this range, with a midpoint of 0.85.

The third health state is characterized by angina, without the presence of CHF. The Harvard catalog includes five sets of community preference-based scores for angina, one that specifies scores for both mild and severe angina (Kuntz et al., 1996), one that specifies mild angina only (Pliskin, Stason, and Weinstein, 1981), one that specifies severe angina only (Cohen, Breall, and Ho, 1994), and two that specify angina with no severity classification (Salkeld, Phongsavan, and Oldenburg, 1997; Stinnett et al., 1996). With the exception of the Pliskin, Stason, and Weinstein score, all of the angina scores are based on the time trade-off method of elicitation. The Pliskin, Stason, and Weinstein score is based on the standard gamble elicitation method. The scores for the nonspecific severity angina fall within the range of the two scores for mild angina specifically. Thus, we used the range of mild angina scores as the endpoints of a uniform distribution. The range of mild angina scores is from 0.7 to 0.89, with a midpoint of 0.80.

For the fourth health state, characterized by the absence of CHF and/or angina, there is only one relevant community preference score available from the Harvard catalog. This score is 0.93, derived from a time trade-off elicitation (Kuntz et al., 1996). Insufficient information is available to provide a distribution for this weight; therefore, it is treated as a fixed value.

Similar to CB, we assumed that the reference weight for the general population without AMI is 0.95. To allow for uncertainty in this parameter, we assigned a triangular distribution around this weight, bounded by 0.9 and 1.0.

Based on the assumptions defined above, we used Monte Carlo simulation methods as implemented in the Crystal Ball™ software program to develop the distribution of QALYs gained per incidence of nonfatal AMI for each age interval. For the Monte Carlo simulation, all distributions were assumed to be independent. The mean QALYs gained per incidence of nonfatal AMI for each age interval is presented in Table 8B-18, along with the 95 percent confidence interval resulting from the Monte Carlo simulation. Table 8B-18 presents both the undiscounted and discounted QALYs gained per incidence.

Table 8B-18. QALYs Gained per Avoided Nonfatal Myocardial Infarction

Age Interval		QALYs Gained per Incidence[a]	
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	4.18 (1.24-7.09)	2.17 (0.70-3.62)
25	34	3.48 (1.09-5.87)	2.00 (0.68-3.33)
35	44	2.81 (0.88-4.74)	1.79 (0.60-2.99)
45	54	2.14 (0.67-3.61)	1.52 (0.51-2.53)
55	64	1.49 (0.42-2.52)	1.16 (0.34-1.95)
65	74	0.97 (0.30-1.64)	0.83 (0.26-1.39)
75	84	0.59 (0.20-0.97)	0.54 (0.19-0.89)
85+		0.32 (0.13-0.50)	0.31 (0.13-0.49)

[a] Mean of Monte Carlo generated distribution; 95% confidence interval presented in parentheses.

8B.4.6 Aggregating Life Expectancy and Quality-of-Life Gains

Given the estimates of changes in life expectancy and quality of life, the next step is to aggregate life expectancy and quality-of-life gains to form an effectiveness measure that can be compared to costs to develop cost-effectiveness ratios. This section discusses the proper characterization of the combined effectiveness measure for the denominator of the cost-effectiveness ratio.

To develop an integrated measure of changes in health, we simply sum together the gains in life years from reduced mortality risk in each age interval with the gains in QALYs from reductions in incidence of chronic morbidity endpoints (CB and acute myocardial infarctions). The resulting measure of effectiveness then forms the denominator in the cost-effectiveness ratio. This combined measure of effectiveness is not a QALY measure in a strict sense, because we have not adjusted life-expectancy gains for preexisting health status (quality of life). It is however, an effectiveness measure that adds a scaled morbidity equivalent to the standard life years calculation. Thus, we term the aggregate measure morbidity inclusive life years, or MILYs. Alternatively, the combined measure could be considered as QALYs with an assumption that the community preference weight for all life-expectancy gains is 1.0. If one considers that this weight might be considered to be a "fair" treatment of those with preexisting disabilities, the effectiveness measure might be termed "fair QALY" gained. However, this implies that all aspects of fairness have been addressed, and there are clearly other issues with the fairness of QALYs (or other effectiveness measures) that are not addressed in this simple adjustment. The MILY measure violates some of the properties used in deriving QALY weights, such as linear substitution between quality of life and quantity of life. However, in aggregating life expectancy and quality-of-life gains, it merely represents an alternative social weighting that is consistent with the spirit of the recent OMB guidance on CEA. The guidance notes that "fairness is important in the choice and execution of effectiveness measures" (OMB, 2003). The resulting aggregate measure of effectiveness will not be consistent with a strict utility interpretation of QALYs; however, it may still be a useful index of effectiveness.

Applying the life expectancies and distributions of QALYs per incidence for CB and AMI to estimated distributions of incidences yields distributions of life expectancy and QALYs gained under the Final SSI & RME Rule. These distributions reflect both the quantified uncertainty in estimates of avoided incidence and the quantified uncertainty in QALYs gained per incidence avoided.

Tables 8B-19 and 8B-20 present the discounted life years, QALYs, and MILYs gained, based on each combination of O_3-mortality study, $PM_{2.5}$-mortality study, and life expectancy assumption for O_3-related life years saved used for the analysis, using a 3 percent discount rate, for 2020 and 2030, respectively. Tables 8B-21 and 8B-22 present the corresponding results using a 7 percent discount rate.

Table 8B-19. Estimated Gains in Discounted MILYs Under the Final SSI & RME Rule in 2020, Using a 3 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	O3-Related Life Years Gained from Mortality Risk Reductions (95% CI)	PM2.5-Related Life Years Gained from Mortality Risk Reductions (95% CI)	QALYs Gained from Reductions in PM2.5-Related Chronic Bronchitis (95% CI)	QALYs Gained from Reductions in PM2.5-Related Non-Fatal Myocardial Infarction (95% CI)	Total MILYs Gained (95% CI)
Bell et al. (2004)	Pope et al. (2002)	General Population	500 (200 - 800)	1,100 (400 - 1,800)	390 (50 - 900)	250 (70 - 510)	5,500 (2,600 - 8,000)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	400 (100 - 600)				5,400 (2,500 - 8,000)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	200 (100 - 300)				5,200 (2,400 - 8,000)
Levy et al. (2005)	Pope et al. (2002)	General Population	2,200 (1,500 - 2,900)				7,000 (4,300 - 10,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	1,700 (1,200 - 2,200)				7,000 (3,800 - 10,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	1,000 (700 - 1,300)				6,000 (3,100 - 9,000)
Bell et al. (2004)	Laden et al. (2006)	General Population	500 (200 - 800)	2,600 (1,400 - 4,000)			11,000 (6,300 - 16,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	400 (100 - 600)				11,000 (6,100 - 16,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	200 (100 - 300)				11,000 (6,000 - 15,000)
Levy et al. (2005)	Laden et al. (2006)	General Population	2,200 (1,500 - 2,900)				13,000 (7,900 - 17,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	1,700 (1,200 - 2,200)				12,000 (7,000 - 17,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	1,000 (700 - 1,300)				11,000 (6,800 - 16,000)

*Life years, QALYs, and MILYs are discounted back to 2020. 95% confidence or credible intervals (CIs) around the point estimates are based on the uncertainty surrounding the effect estimates (coefficients) in the C-R functions and, for QALYs and MILYs, the uncertainty surrounding the quality of life weights. All estimates rounded to two significant figures.

Table 8B-20. Estimated Gains in Discounted MILYs Under the Final SSI & RME Rule in 2030, Using a 3 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	O3-Related Life Years Gained from Mortality Risk Reductions (95% CI)	PM2.5-Related Life Years Gained from Mortality Risk Reductions (95% CI)	QALYs Gained from Reductions in PM2.5-Related Chronic Bronchitis (95% CI)	QALYs Gained from Reductions in PM2.5-Related Non-Fatal Myocardial Infarction (95% CI)	Total MILYs Gained (95% CI)
Bell et al. (2004)	Pope et al. (2002)	General Population	700 (200 - 1,200)	2,200 (900 - 3,500)	590 (80 - 1,400)	430 (110 - 880)	6,100 (3,100 - 9,000)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	600 (200 - 900)				6,000 (3,000 - 9,000)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	300 (100 - 500)				5,700 (2,700 - 9,000)
Levy et al. (2005)	Pope et al. (2002)	General Population	3,500 (2,400 - 4,600)				9,000 (5,800 - 12,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	2,700 (1,800 - 3,500)				8,000 (5,000 - 11,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	1,500 (1,000 - 1,900)				6,800 (3,900 - 10,000)
Bell et al. (2004)	Laden et al. (2006)	General Population	700 (200 - 1,200)	5,000 (2,700 - 7,000)			11,600 (6,800 - 16,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	600 (200 - 900)				11,000 (6,600 - 16,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	300 (100 - 500)				11,000 (6,400 - 16,000)
Levy et al. (2005)	Laden et al. (2006)	General Population	3,500 (2,400 - 4,600)				14,000 (9,400 - 19,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	2,700 (1,800 - 3,500)				14,000 (9,000 - 18,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	1,500 (1,000 - 1,900)				12,000 (7,500 - 17,000)

*Life years, QALYs, and MILYs are discounted back to 2030. 95% confidence or credible intervals (CIs) around the point estimates are based on the uncertainty surrounding the effect estimates (coefficients) in the C-R functions and, for QALYs and MILYs, the uncertainty surrounding the quality of life weights. All estimates rounded to two significant figures.

Table 8B-21. Estimated Gains in Discounted MILYs Under the Final SSI & RME Rule in 2020, Using a 7 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	O3-Related Life Years Gained from Mortality Risk Reductions (95% CI)	PM2.5-Related Life Years Gained from Mortality Risk Reductions (95% CI)	QALYs Gained from Reductions in PM2.5-Related Chronic Bronchitis (95% CI)	QALYs Gained from Reductions in PM2.5-Related Non-Fatal Myocardial Infarction (95% CI)	Total MILYs Gained (95% CI)
Bell et al. (2004)	Pope et al. (2002)	General Population	360 (120 - 600)	800 (300 - 1,200)	300 (30 - 600)	200 (50 - 400)	3,800 (1,800 - 5,700)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	290 (90 - 500)				3,700 (1,800 - 5,700)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	170 (50 - 280)				3,600 (1,600 - 5,500)
Levy et al. (2005)	Pope et al. (2002)	General Population	1,700 (1,200 - 2,200)				5,100 (3,100 - 7,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	1,400 (900 - 1,800)				4,800 (2,700 - 7,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	800 (600 - 1,100)				4,200 (2,300 - 6,200)
Bell et al. (2004)	Laden et al. (2006)	General Population	360 (120 - 600)	1,800 (1,000 - 2,500)			7,500 (4,300 - 11,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	290 (90 - 500)				7,500 (4,200 - 11,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	170 (50 - 280)				7,300 (4,100 - 11,000)
Levy et al. (2005)	Laden et al. (2006)	General Population	1,700 (1,200 - 2,200)				9,000 (5,600 - 12,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	1,400 (900 - 1,800)				9,000 (5,300 - 12,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	800 (600 - 1,100)				8,000 (4,800 - 11,000)

*Life years, QALYs, and MILYs are discounted back to 2020. 95% confidence or credible intervals (CIs) around the point estimates are based on the uncertainty surrounding the effect estimates (coefficients) in the C-R functions and, for QALYs and MILYs, the uncertainty surrounding the quality of life weights. All estimates rounded to two significant figures.

Table 8B-22. Estimated Gains in Discounted MILYs Under the Final SSI & RME Rule in 2030, Using a 7 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	O3-Related Life Years Gained from Mortality Risk Reductions (95% CI)	PM2.5-Related Life Years Gained from Mortality Risk Reductions (95% CI)	QALYs Gained from Reductions in PM2.5-Related Chronic Bronchitis (95% CI)	QALYs Gained from Reductions in PM2.5-Related Non-Fatal Myocardial Infarction (95% CI)	Total MILYs Gained (95% CI)
Bell et al. (2004)	Pope et al. (2002)	General Population	590 (190 - 1,000)	800 (300 - 1,200)	400 (50 - 900)	340 (90 - 700)	4,300 (2,200 - 6,300)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	460 (150 - 800)				4,100 (2,100 - 6,200)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	250 (80 - 430)				3,900 (1,900 - 5,900)
Levy et al. (2005)	Pope et al. (2002)	General Population	2,700 (1,900 - 3,500)				6,400 (4,200 - 9,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	2,100 (1,500 - 2,800)				5,800 (3,700 - 8,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	1,200 (800 - 1,600)				4,900 (2,900 - 7,000)
Bell et al. (2004)	Laden et al. (2006)	General Population	590 (190 - 1,000)	1,800 (1,000 - 2,500)			8,100 (4,800 - 11,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	460 (150 - 800)				8,000 (4,600 - 11,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	250 (80 - 430)				7,800 (4,500 - 11,000)
Levy et al. (2005)	Laden et al. (2006)	General Population	2,700 (1,900 - 3,500)				10,000 (6,800 - 14,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	2,100 (1,500 - 2,800)				10,000 (6,300 - 13,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	1,200 (800 - 1,600)				9,000 (5,400 - 12,000)

*Life years, QALYs, and MILYs are discounted back to 2030. 95% confidence or credible intervals (CIs) around the point estimates are based on the uncertainty surrounding the effect estimates (coefficients) in the C-R functions and, for QALYs and MILYs, the uncertainty surrounding the quality of life weights. All estimates rounded to two significant figures.

8B.4.7 Estimating the Avoided Costs of Chronic Illness

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. As noted above (see Section 8B.3.1), our estimate of costs in the numerator is net of the avoided costs (cost savings) associated with the reductions in morbidity (Gold et al., 1996). Among the morbidity costs subtracted from the direct costs of controls in the numerator are the avoided costs of illness (COI) associated with $PM_{2.5}$-related CB and nonfatal AMI.

Avoided costs for CB and nonfatal AMI are based on estimates of lost earnings and medical costs.[13] Using age-specific annual lost earnings and medical costs estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a lifetime present discounted value (in 2005$) due to CB of $179,305 for someone between the ages of 27 and 44; $116,892 for someone between the ages of 45 and 64; and $13,741 for someone over 65. The corresponding age-specific estimates of lifetime present discounted value (in 2005$) using a 7 percent discount rate are $102,300, $86,359, and $11,190, respectively. These estimates assumed that 1) lost earnings continue only until age 65, 2) medical expenditures are incurred until death, and 3) life expectancy is unchanged by CB.

Because the costs associated with a myocardial infarction extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a present discounted value in lost earnings (in 2005$) over 5 years due to a myocardial infarction of $10,389 for someone between the ages of 25 and 44, $15,313 for someone between the ages of 45 and 54, and $88,508 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2005$) using a 7 percent discount rate are $9,301, $13,709, and $79,241, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. Thus, we do not include lost earnings in the cost estimates for these age groups.

Two estimates of the direct medical costs of myocardial infarction are used. The first estimate is from Wittels, Hay, and Gotto (1990), which estimated expected total medical costs of MI over 5 years to be $51,211 (in 1986$) for people who were admitted to the hospital and survived hospitalization (there does not appear to be any discounting used). Using the CPI-U for medical care, the Wittels estimate is $135,667 in year 2005$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes, and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical

[13] Gold et al. (1996) recommend not including lost earnings in the cost-of-illness estimates, suggesting that in some cases, they may be already be counted in the effectiveness measures. However, this requires that individuals fully incorporate the value of lost earnings and reduced labor force participation opportunities into their responses to time-tradeoff or standard-gamble questions. For the purposes of this analysis and for consistency with the way costs-of-illness are calculated for the benefit-cost analysis, we have assumed that individuals do not incorporate lost earnings in responses to these questions. This assumption can be relaxed in future analyses with improved understanding of how lost earnings are treated in preference elicitations.

decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The second estimate is from Russell et al. (1998), which estimated first-year direct medical costs of treating nonfatal myocardial infarction of $15,540 (in 1995$), and $1,051 annually thereafter. Converting to year 2005$, that would be $27,674 for a 5-year period (using a 3 percent discount rate).

The two estimates from these studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used estimates for medical costs that similarly cover a 5-year period. We used a simple average of the two 5-year estimates, or $81,671, and add it to the 5-year opportunity cost estimate. The resulting estimates are given in Table 8B-23.

Table 8B-23. Estimated Costs Over a 5-Year Period (in 2005$) of a Nonfatal Myocardial Infarction

Age of Onset	Opportunity Cost[1]	Medical Cost[2]	Total Cost*
0 - 24	$0	$81,671	$81,671
25 - 44	$10,389	$81,671	$92,060
45 - 54	$15,313	$81,671	$96,984
55 - 65	$88,508	$81,671	$170,179
> 65	$0	$81,671	$81,671

[1] Positive opportunity costs are based on Cropper and Krupnick (1990), using a 3 percent discount rate.

[2] An average of the 5-year costs estimated by Wittels, Hay, and Gotto (1990) and Russell et al. (1998).

The total avoided COI by age group associated with the reductions in CB and nonfatal acute myocardial infarctions (using a 3 percent discount rate) is provided in Table 8B-24. The total avoided COI associated with the Final SSI & RME Rule (using a 3 percent discount rate) is about $42 million in 2020 and about $71 million in 2030. Note that these estimates do not include any direct avoided medical costs associated with premature mortality. Nor do they include any medical costs that occur more than 5 years from the onset of a nonfatal AMI. Therefore, they are likely underestimates of the true avoided COI associated with the Final SSI & RME Rule in 2020 and 2030.

Table 8B-24. Avoided Costs of Illness Associated with Reductions in Chronic Bronchitis and Nonfatal Acute Myocardial Infarctions Under the Final SSI & RME Rule in 2020 and 2030

	Avoided Cost of Illness (in millions of 2005$)*			
Age Interval	2020		2030	
	Chronic Bronchitis	Nonfatal Acute Myocardial Infarction	Chronic Bronchitis	Nonfatal Acute Myocardial Infarction
18 - 24	---	$0.0	---	$0.0
25 - 29	$4.1	$0.1	$4.9	$0.2
35 - 44	$3.4	$0.9	$6.0	$1.5
45 - 54	$2.1	$2.8	$3.2	$4.2
55 - 64	$2.2	$11.5	$3.3	$16.8
65 - 74	$0.2	$7.7	$0.4	$12.8
75 - 84	$0.1	$3.9	$0.2	$11.6
85+	$0.1	$3.5	$0.1	$5.5
Total:	$12.1	$30.4	$18.1	$52.5

*Discounted using a 3 percent discount rate.

8B.4.8 Cost-Effectiveness Ratios

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. As noted above (see Section 8B.3.1), the estimate of costs in the numerator should include both the direct costs of the controls necessary to achieve the reduction in ambient $PM_{2.5}$ and O_3 and the avoided costs (cost savings) associated with the reductions in morbidity (Gold et al., 1996). In general, because reductions in air pollution do not require direct actions by the affected populations, there are no specific costs to affected individuals (aside from the overall increases in prices that might be expected to occur as control costs are passed on by affected industries). Likewise, because individuals do not engage in any specific actions to realize the health benefit of the pollution reduction, there are no decreases in utility (as might occur from a medical intervention) that need to be adjusted for in the denominator. Thus, the elements of the numerator are direct costs of controls minus the avoided COI associated with CB and nonfatal AMI. In addition, to account for the value of reductions in O_3- and $PM_{2.5}$-related acute health impacts and non-health benefits, we netted out the monetized value of these benefits from the numerator to yield a "net cost" estimate. For the MILY aggregate effectiveness measure, the denominator is simply the sum of (O_3- and $PM_{2.5}$-related) life years gained from increased life expectancy and QALYs gained from the ($PM_{2.5}$-related) reductions in CB and nonfatal AMI. The separate O_3- and $PM_{2.5}$-related inputs to the denominators of the cost-effectiveness ratios are summarized above in Tables 8B-19 through 8B-22. The cost-effectiveness ratios and 95 percent confidence (credible) intervals resulting from all of the sources of uncertainty considered, using Monte Carlo procedures as implemented in the Crystal Ball™ software program and incorporating both the O_3- and $PM_{2.5}$-related benefits are shown in the tables below. Tables 8B-25 and 8B-26 show cost per life saved, using a 3 percent and 7 percent discount rate, respectively. Tables 8B-27 and 8B-28 show cost per life year saved at the two discount rates; and Tables 8B-29 and 8B-30 show cost per MILY gained.

Table 8B-25. Estimated Net Cost (2005$) per O_3- and $PM_{2.5}$-Related Life Saved Under the Final SSI & RME Rule in 2020 and 2030, Using a 3 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Cost Effectiveness Ratio: Net Cost (in Thousand $) per Life Saved* (95% CI)**	
		2020	2030
Bell et al. (2004)	Pope et al. (2002)	$260 ($110 - $580)	$74 ($-99 - $280)
Bell et al. (2004)	Laden et al. (2006)	$110 ($54 - $220)	$34 ($-44 - $120)
Levy et al. (2005)	Pope et al. (2002)	$180 ($85 - $320)	$44 ($-58 - $140)
Levy et al. (2005)	Laden et al. (2006)	$96 ($48 - $170)	$26 ($-34 - $83)

*The cost of the regulation is estimated to be $207.4 million in 2020 and $185.5 million in 2030. $PM_{2.5}$-related avoided deaths are discounted back to 2020 or 2030. O_3-related deaths are assumed to occur in 2020 or 2030.

**95 percent confidence or credible intervals incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 8B-26. Estimated Net Cost (2005$) per O_3- and $PM_{2.5}$-Related Life Saved Under the Final SSI & RME Rule in 2020 and 2030, Using a 7 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Cost Effectiveness Ratio: Net Cost (in Thousand $) per Life Saved* (95% CI)**	
		2020	2030
Bell et al. (2004)	Pope et al. (2002)	$300 ($130 - $660)	$99 ($-87 - $330)
Bell et al. (2004)	Laden et al. (2006)	$130 ($67 - $250)	$47 ($-39 - $140)
Levy et al. (2005)	Pope et al. (2002)	$200 ($100 - $350)	$57 ($-49 - $160)
Levy et al. (2005)	Laden et al. (2006)	$110 ($58 - $190)	$35 ($-30 - $95)

*The cost of the regulation is estimated to be $207.4 million in 2020 and $185.5 million in 2030. $PM_{2.5}$-related avoided deaths are discounted back to 2020 or 2030. O_3-related deaths are assumed to occur in 2020 or 2030.

**95 percent confidence or credible intervals incorporate uncertainty surrounding the O3 and PM2.5 coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 8B-27. Estimated Net Cost (2005$) per O_3- and $PM_{2.5}$-Related Life Year Saved Under the Final SSI & RME Rule in 2020 and 2030, Using a 3 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	Cost Effectiveness Ratio: Net Cost (in Thousand $) per Life Year Saved* (95% CI)**	
			2020	2030
Bell et al. (2004)	Pope et al. (2002)	General Population	$23 ($9.9 - $54)	$6.8 ($-9 - $26)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	$24 ($10 - $56)	$7.1 ($-9.5 - $27)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	$25 ($10 - $61)	$7.6 ($-10 - $30)
Levy et al. (2005)	Pope et al. (2002)	General Population	$16 ($7.8 - $30)	$4.1 ($-5.5 - $13)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	$18 ($8.3 - $34)	$4.7 ($-6.2 - $15)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	$21 ($9.2 - $44)	$5.8 ($-7.6 - $20)
Bell et al. (2004)	Laden et al. (2006)	General Population	$10 ($5 - $20)	$3.1 ($-4.2 - $11)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	$11 ($5 - $21)	$3.2 ($-4.2 - $11)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	$11 ($5.1 - $21)	$3.3 ($-4.4 - $11)
Levy et al. (2005)	Laden et al. (2006)	General Population	$8.8 ($4.4 - $16)	$2.4 ($-3.2 - $7.7)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	$9.2 ($4.5 - $17)	$2.6 ($-3.4 - $8.3)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	$9.9 ($4.8 - $19)	$2.9 ($-3.9 - $9.5)

*The cost of the regulation is estimated to be $207.4 million in 2020 and $185.5 million in 2030. All life years are discounted back to the year of death. $PM_{2.5}$-related avoided deaths are discounted back to 2020 or 2030. O_3-related deaths are assumed to occur in 2020 or 2030.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 8B-28. Estimated Net Cost (2005$) per O_3- and $PM_{2.5}$-Related Life Year Saved Under the Final SSI & RME Rule in 2020 and 2030, Using a 7 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	Cost Effectiveness Ratio: Net Cost (in Thousand $) per Life Year Saved* (95% CI)**	
			2020	2030
Bell et al. (2004)	Pope et al. (2002)	General Population	$36 ($16 - $81)	$12 ($-11 - $42)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	$37 ($16 - $85)	$13 ($-11 - $44)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	$39 ($17 - $93)	$14 ($-12 - $50)
Levy et al. (2005)	Pope et al. (2002)	General Population	$24 ($12 - $44)	$7 ($-6 - $20)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	$26 ($13 - $49)	$8.0 ($-6.7 - $23)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	$31 ($15 - $62)	$10 ($-8.3 - $31)
Bell et al. (2004)	Laden et al. (2006)	General Population	$16 ($7.8 - $30)	$5.6 ($-4.7 - $17)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	$16 ($7.9 - $31)	$5.7 ($-4.8 - $17)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	$17 ($8 - $32)	$5.9 ($-5 - $18)
Levy et al. (2005)	Laden et al. (2006)	General Population	$13 ($6.8 - $23)	$4.3 ($-3.5 - $12)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	$14 ($7.1 - $24)	$4.6 ($-3.9 - $13)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	$15 ($7.5 - $28)	$5.1 ($-4.4 - $15)

*The cost of the regulation is estimated to be $207.4 million in 2020 and $185.5 million in 2030. All life years are discounted back to the year of death. $PM_{2.5}$-related avoided deaths are discounted back to 2020 or 2030. O_3-related deaths are assumed to occur in 2020 or 2030.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 8B-29. Estimated Net Cost (2005$) per O_3- and $PM_{2.5}$-Related MILY Gained Under the Final SSI & RME Rule in 2020 and 2030, Using a 3 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	Cost Effectiveness Ratio: Net Cost (in Thousand $) per MILY Gained* (95% CI)**	
			2020	2030
Bell et al. (2004)	Pope et al. (2002)	General Population	$20 ($9 - $42)	$5.5 ($-7.4 - $19)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	$21 ($9.3 - $44)	$5.6 ($-7.6 - $20)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	$21 ($9.4 - $46)	$6.0 ($-8.1 - $21)
Levy et al. (2005)	Pope et al. (2002)	General Population	$15 ($7.2 - $26)	$3.6 ($-4.9 - $11)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	$16 ($7.6 - $29)	$4.0 ($-5.4 - $13)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	$18 ($8.4 - $36)	$4.8 ($-6.4 - $16)
Bell et al. (2004)	Laden et al. (2006)	General Population	$9.8 ($4.7 - $18)	$2.8 ($-3.8 - $9.4)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	$9.9 ($4.8 - $19)	$2.9 ($-3.8 - $9.6)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	$10 ($4.8 - $19)	$3.0 ($-3.9 - $10)
Levy et al. (2005)	Laden et al. (2006)	General Population	$8.3 ($4.2 - $14)	$2.2 ($-2.9 - $7)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	$8.7 ($4.3 - $16)	$2.4 ($-3.2 - $7.5)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	$9.3 ($4.5 - $17)	$2.7 ($-3.5 - $8.6)

*The cost of the regulation is estimated to be $207.4 million in 2020 and $185.5 million in 2030. $PM_{2.5}$-related avoided deaths are discounted back to 2020 or 2030. O_3-related deaths are assumed to occur in 2020 or 2030.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 8B-30. Estimated Net Cost (2005$) per O_3- and $PM_{2.5}$-Related MILY Gained Under the Final SSI & RME Rule in 2020 and 2030, Using a 7 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	Cost Effectiveness Ratio: Net Cost (in Thousand $) per MILY Gained* (95% CI)**	
			2020	2030
Bell et al. (2004)	Pope et al. (2002)	General Population	$110 ($58 - $190)	$31 ($15 - $64)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	$33 ($15 - $70)	$31 ($15 - $66)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	$31 ($15 - $64)	$33 ($15 - $70)
Levy et al. (2005)	Pope et al. (2002)	General Population	$31 ($15 - $66)	$22 ($12 - $38)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	$27 ($13 - $51)	$24 ($12 - $42)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	$22 ($12 - $38)	$27 ($13 - $51)
Bell et al. (2004)	Laden et al. (2006)	General Population	$23 ($12 - $42)	$15 ($7.4 - $27)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	$15 ($7.6 - $29)	$15 ($7.5 - $28)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	$15 ($7.4 - $27)	$15 ($7.6 - $29)
Levy et al. (2005)	Laden et al. (2006)	General Population	$15 ($7.5 - $28)	$13 ($6.5 - $21)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	$14 ($7.1 - $25)	$13 ($6.8 - $22)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	$13 ($6.5 - $21)	$14 ($7.1 - $25)

*The cost of the regulation is estimated to be $207.4 million in 2020 and $185.5 million in 2030. $PM_{2.5}$-related avoided deaths are discounted back to 2020 or 2030. O_3-related deaths are assumed to occur in 2020 or 2030.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

8B.5 Conclusions

We estimated the cost effectiveness of attaining the Final Small SI and Recreational Marine Engine Rule in 2020 and in 2030, based on reductions in premature deaths and incidence of chronic disease. We measured effectiveness using several different metrics, including lives saved, life years saved, and QALYs gained (for improvements in quality of life due to reductions in incidence of chronic disease). We suggested a new metric for aggregating life years saved and improvements in quality of life, morbidity inclusive life years (MILY) which assumes that society assigns a weight of one to years of life extended regardless of preexisting disabilities or chronic health conditions.

CEA of environmental regulations that have substantial public health impacts may be informative in identifying programs that have achieved cost-effective reductions in health impacts and can suggest areas where additional controls may be justified. However, the overall efficiency of a regulatory action can only be judged through a complete benefit-cost analysis that takes into account all benefits and costs, including both health and non-health effects. The benefit-cost analysis for the Final Small SI and Recreational Marine Engine Rule, provided in Chapter 8, shows that the rule has potentially large net benefits, indicating that implementation of the Final Small SI and Recreational Marine Engine Rule will likely result in improvements in overall public welfare.

8B.6 References

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