Draft Regulatory Impact Analysis: Control of Emissions of Air Pollution from Locomotive Engines and Marine Compression-Ignition Engines Less than 30 Liters per Cylinder

Chapter 6
Cost-Benefit Analysis

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6.1 Overview

This chapter presents our analysis of the health and environmental benefits that can be expected to occur as a result of the proposed locomotive and marine engine standards throughout the period from initial implementation through 2030. Nationwide, the engines subject to the proposed emission standards in this rule are a significant source of mobile source air pollution. The proposed standards would reduce exposure to direct $PM_{2.5}$, NO_x 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 major new pollution control regulations. Accordingly, the analysis presented here attempts to answer three questions: (1) what are the physical health and welfare effects of changes in ambient air quality resulting from particulate matter (PM) and ozone precursor emission reductions (direct PM and NO_x)? (2) what is the monetary value of the changes in these effects attributable to the proposed rule? and (3) how do the monetized benefits compare to the costs? It constitutes one part of EPA's thorough examination of the relative merits of this regulation.

All of the benefit estimates for the proposed control options in this analysis are based on an analytical structure and sequence similar to that used in the final PM NAAQS analysis.[1] The benefits analysis relies on three major components:

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

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

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

A wide range of human health and welfare effects are linked to the emissions of direct PM and NO_x and the resulting impact on ambient concentrations of ozone and $PM_{2.5}$. Recent studies have linked short-term ozone exposures with premature mortality. Exposure to ozone has also been linked to a variety of respiratory effects including hospital admissions and illnesses resulting in school absences. Potential human health effects associated with $PM_{2.5}$ range from premature mortality to morbidity effects linked to long-term (chronic) and shorter-term (acute) exposures (e.g., respiratory and cardiovascular symptoms resulting in hospital admissions, asthma exacerbations, and acute and chronic bronchitis). Welfare effects potentially linked to PM include materials damage and visibility impacts, while ozone can adversely affect the agricultural and forestry sectors by decreasing yields of crops and forests.

EPA typically quantifies PM- and ozone-related benefits in its regulatory impact analyses (RIAs) when possible. In the analysis of past air quality regulations, ozone-related benefits have

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

Since the NAS effort is not expected to conclude until 2008, the agency is currently deliberating how best to characterize ozone-related mortality benefits in its rulemaking analyses in the interim. For the analysis of the proposed locomotive and marine standards, we do not quantify an ozone mortality benefit. So that we do not provide an incomplete picture of all of the benefits associated with reductions in emissions of ozone precursors, we have chosen not to include an estimate of total ozone benefits in the proposed RIA. By omitting ozone benefits in this proposal, we acknowledge that this analysis underestimates the benefits associated with the proposed standards. Our analysis, however, indicates that the rule's monetized PM2.5 benefits alone substantially exceed our estimate of the costs.

Table 6.1-1 summarizes the annual monetized health and welfare benefits associated with the proposed standards for two years, 2020 and 2030. There are a number of items to note about these benefits:

- 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 emission control program being proposed. The differences reflect further refinements of the regulatory program since we performed the air quality modeling for this rule. Section 3.6 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 proposed regulatory scenario.

- Consistent with the approach used in the recent RIA for the PM NAAQS, rather than presenting both a "primary" estimate of the benefits and a separate characterization of the uncertainty associated with that estimate, the current analysis follows the recommendation of the National Research Council's (NRC) 2002 report "Estimating the Public Health Benefits of Proposed Air Pollution Regulations" to begin moving the assessment of uncertainties from

its ancillary analyses into its main benefits presentation through the conduct of probabilistic analyses.

- Since the publication of CAIR, we have completed a full-scale expert elicitation designed to more fully characterize the state of our understanding of the concentration-response function for PM-related premature mortality. Consistent with the approach used in the recent RIA for the PM NAAQS, the elicitation results form a major component of the current effort to use probabilistic assessment techniques to integrate uncertainty into the main benefits analysis.

- Since the publication of CAIR, a follow-up to the Harvard Six-Cities study on premature mortality was published (Laden et al., 2006 based on Dockery et al., 1993),[2,3] which 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. Consistent with the approach used in the recent RIA for the PM NAAQS, we further characterize uncertainty by presenting a range of PM-related premature mortality estimates derived from both the American Cancer Society (ACS) cohort study (Pope et al.,2002),[4] used as the primary estimate of PM-related mortality in previous Regulatory Impact Analyses (RIAs), and the Six-Cities study (Laden et al., 2006).

- Consistent with the approach used in the recent RIA for the PM NAAQS, we have updated our projections of mortality incidence rates to be consistent with the U.S. Census population projections that form the basis of our future population estimates. Compared to the methodology used in the CAIR analysis, this change will result in a reduction in mortality impacts in future years, as overall mortality rates are projected to decline for most age groups.

- Consistent with the approach used in the recent RIA for the PM NAAQS, we provide additional characterizations of the impacts of assuming alternative thresholds in the concentration-response functions derived from the epidemiology literature. Unless specifically noted, our base PM-related premature mortality benefits estimates are based on an assumed cutpoint in the long-term mortality concentration-response function at 10 µg/m3, and an assumed cutpoint in the short-term morbidity concentration-response functions at 10 µg/m3. We also show the results of a sensitivity analysis for PM-related premature mortality, with 4 alternative cutpoints, at 3 µg/m3, 7.5 µg/m3, 12 µg/m3, and 14 µg/m3.

Table 6.1-1. Estimated Monetized PM-Related Health Benefits of the Proposed Locomotive and Marine Engine Standards

	Total Benefits[a,b,c,d] (billions 2005$)	
	2020	2030
PM mortality derived from the ACS cohort study;Morbidity functions from epidemiology literature		
Using a 3% discount rate	$4.4+B	$12+B
Confidence Intervals (5th - 95th %ile)	($1.0 - $10)	($2.1 - $27)
Using a 7% discount rate	$4.0+B	$11+B
Confidence Intervals (5th - 95th %ile)	($1.0 - $9.2)	($1.8 - $25)
PM mortality derived from lower bound and upper bound expert-based result;[e] Morbidity functions from epidemiology literature		
Using a 3% discount rate	$1.7+B - $12+B	$4.6+B - $33+B
Confidence Intervals (5th - 95th %ile)	($0.2 - $8.5) – ($2.0 - $27)	($1.0 - $23) – ($5.4 - $72)
Using a 7% discount rate	$1.6+B - $11+B	$4.3+B - $30+B
Confidence Intervals (5th - 95th %ile)	($0.2 - $7.8) – ($1.8 - $24)	($1.0 - $21) – ($4.9 - $65)

[a] Benefits include avoided cases of mortality, chronic illness, and other morbidity health endpoints.
[b] PM-related mortality benefits estimated using an assumed PM threshold of 10 μg/m3. There is uncertainty about which threshold to use and this may impact the magnitude of the total benefits estimate. For a more detailed discussion of this issue, please refer to Section 6.6.1.3 of the RIA.
[c] For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. A detailed listing of unquantified health and welfare effects is provided in Table 6.1-2.
[d] Results reflect the use of two different discount rates: 3 and 7 percent, which are recommended by EPA's Guidelines for Preparing Economic Analyses[5] and OMB Circular A-4. Results are rounded to two significant digits for ease of presentation and computation.
[e] The effect estimates of nine of the twelve experts included in the elicitation panel fall within the empirically-derived range provided by ACS and Six-Cities studies. One of the experts fall below this range and two 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. Likewise the 5th and 95th percentiles for these highest and lowest judgments of the effect estimate do not imply any particular distribution within those bounds. The distribution of benefits estimates associated with each of the twelve expert responses can be found in tables 6.4-3 and 6.4-4.

Table 6.1-2 lists the full complement of human health and welfare effects associated with PM, ozone and air toxics, and identifies those effects that are quantified for the primary estimate and those that remain unquantified because of current limitations in methods or available data.

Table 6.1-2. Human Health and Welfare Effects of Pollutants Affected by the Proposed Standards

Pollutant/Effect	Quantified and Monetized in Base Estimates[a]	Unquantified Effects - Changes in:
PM/Health[b]	Premature mortality based on 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 Cardiovascular emergency room visits Acute respiratory symptoms Chronic respiratory damage Premature aging of the lungs Nonasthma respiratory emergency room visits UVb exposure (+/-)[e]
Ozone/Welfare		Decreased outdoor worker productivity Yields for commercial crops Yields for commercial forests and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions UVb exposure (+/-)[e]
CO Health		Behavioral effects
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

Pollutant/Effect	Quantified and Monetized in Base Estimates[a]	Unquantified Effects - Changes in:
		nitrogen deposition Recreation in estuarine ecosystems due to nitrogen deposition Ecosystem functions Passive fertilization
NO_x/Health		Lung irritation Lowered resistance to respiratory infection Hospital admissions for respiratory and cardiac diseases
HC/Toxics Health[g]		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[g]		Direct toxic effects to animals Bioaccumulation in the food chain Damage to ecosystem function Odor

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

[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).[6]

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

[e] May result in benefits or disbenefits. See Section 6.5.3 for more details.

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

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

The general benefits analysis framework is as follows:

- Given baseline and post-control emissions inventories for the emission species expected to affect ambient air quality, we use sophisticated photochemical air quality models to estimate baseline and post-control ambient concentrations of PM and visibility for each year.

- The estimated changes in ambient concentrations are then combined with monitoring data to estimate population-level potential exposures to changes in ambient concentrations for use in estimating health effects. Modeled changes in ambient data are also used to estimate changes in visibility.

- Changes in population exposure to ambient air pollution are then input to impact functions[A] to generate changes in the incidence of health effects, or changes in other exposure metrics are input to dose-response functions to generate changes in welfare effects. Because these estimates contain uncertainty, we characterize the benefits estimates probabilistically when appropriate information is available.

- The resulting effects changes are then assigned monetary values, taking into account adjustments to values for growth in real income out to the year of analysis (values for health and welfare effects are in general positively related to real income levels).

- Finally, values for individual health and welfare effects are summed to obtain an estimate of the total monetary value of the benefits resulting from the changes in emissions.

EPA is currently developing a comprehensive integrated strategy for characterizing the impact of uncertainty in key elements of the benefits modeling process (e.g., emissions modeling, air quality modeling, health effects incidence estimation, valuation) on the benefits estimates. A recently completed component of this effort is an expert elicitation designed to characterize more fully our understanding of PM-related mortality resulting from both short-term and long-term exposure.[B] We include the results of the formal expert elicitation among the sources of information used in developing health impact functions for this benefits analysis. The results of the 'pilot' for this expert elicitation were presented in RIAs for both the Nonroad Diesel and Clean Air Interstate Rules.[7,8] The results of these elicitation projects, including peer review comments, are available on EPA's Web site, at http://www.epa.gov/ttn/ecas/. In addition,

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

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

similar to our approach in the Nonroad Diesel and CAIR RIAs, we present a distribution of benefits estimates based on a more limited set of uncertainties, those characterized by the sampling error and variability in the underlying health and economic valuation studies used in the benefits modeling framework. We note that incorporating only the uncertainty from random sampling error omits important sources of uncertainty (e.g., in the functional form of the model, as discussed below). 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 that are summarized later in this chapter.

The benefits estimates generated for the proposed standards are subject to a number of assumptions and uncertainties, which are discussed throughout this document. For example, key assumptions underlying the data-derived concentration-response functions for the $PM_{2.5}$-related mortality category include the following:

1. Inhalation of fine particles is causally associated with premature death at concentrations near those experienced by most Americans on a daily basis. Although biological mechanisms for this effect have not yet been specifically identified, the weight of the available epidemiological, toxicological, and experimental evidence supports an assumption of causality. The impacts of including a probabilistic representation of causality are explored using the results of the expert elicitation.
2. All fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because the composition of PM produced via transported precursors emitted from locomotive and marine engines may differ significantly from direct PM released from electric generating units (EGUs) and other industrial sources.[C] In accordance with advice from the CASAC, EPA has determined that no clear scientific grounds exist for supporting differential effects estimates by particle type, based on information in the most recent Criteria Document. We provide a decomposition of benefits by PM component species to provide additional insights into the makeup of the benefits associated with reductions in overall $PM_{2.5}$ mass (See Tables 5-32 and 5-33).
3. The C-R function for fine particles is approximately linear within the range of ambient concentrations under consideration (above the assumed threshold of 10 $\mu g/m^3$). Thus, we assume that the CR functions are applicable to estimates of health benefits associated with reducing fine particles in areas with varied concentrations of PM, including both regions that are in attainment with $PM_{2.5}$ standards and those that do not meet the standards. However, we examine the impact of this assumption by looking at alternative thresholds in a sensitivity analysis.

The first and third of these assumptions are directly addressed in the expert elicitation, providing probabilistic characterizations of the likelihood of causality and the shape of the concentration-response function. The second of these is not directly addressed by the expert

[C] Even within certain components such as directly emitted PM, there may be significant differences in toxicity of component particles such as trace metals and specific carbonaceous species.

elicitation, and remains a significant source of uncertainty in the state of knowledge about the health benefits associated with various emission reduction strategies.

In addition, a key assumption underlying the entire analysis is that the forecasts for future emissions and associated air quality modeling are valid. Because we are projecting emissions and air quality out to 2030, there are inherent uncertainties in all of the factors that underlie the future state of emissions and air quality levels. While it is important to keep in mind the difficulties, assumptions, and inherent uncertainties in the overall enterprise, these analyses are based on peer-reviewed scientific literature and up-to-date assessment tools, and we believe the results are highly useful in assessing the impacts of this rule.

In addition to the quantified and monetized benefits summarized above, a number of additional categories associated with ozone and $PM_{2.5}$ and its precursor emissions are not currently amenable to quantification or valuation. These include reduced acid and particulate deposition damage to cultural monuments and other materials, and environmental benefits due to reductions of impacts of acidification in lakes and streams and eutrophication in coastal areas. Additionally, we have not quantified a number of known or suspected health effects linked with ozone and PM for which appropriate health impact functions are not available or which do not provide easily interpretable outcomes (i.e., changes in heart rate variability). As a result, monetized benefits generated for the primary estimate may underestimate the total benefits attributable to attainment of alternative standards.

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

This chapter is organized as follows. In Section 6.2, we provide an overview of the air quality impacts modeled for the proposed standards that are used as inputs to the benefits analysis. In Section 6.3, we document the key methods and inputs used in the benefits analysis. In Section 6.4, we report the results of the analysis for human health and welfare effects. In Section 6.5, we present a comparison of the costs and benefits associated with the proposed standards.

6.2 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 proposed 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 $PM_{2.5}$ related to emission reductions estimated to occur due to the proposed 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 proposed 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 emission control program being proposed. 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 3.6 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 proposed regulatory scenario.

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

This analysis extracted hourly, surface-layer PM concentrations for each grid cell from the standard CMAQ output files. To estimate PM-related health and welfare effects for the contiguous United States, we use 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 36 km grid.[D]

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 proposed emissions controls. Full documentation of the revised SMAT methodology is contained in the Air Quality Modeling TSD.

6.2.2 $PM_{2.5}$ Air Quality Results

This section provides a summary of the predicted ambient $PM_{2.5}$ concentrations from the CMAQ model for the 2020 and 2030 base cases and changes associated with the proposed rule. Table 6.2-1 provides those $PM_{2.5}$ 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, therefore, better reflects the potential benefits of these predicted changes through exposure changes to these populations.

[D]The 36-km grid squares contain the population data used in the health benefits analysis model, BenMAP.

Table 6.2-1. Summary of CMAQ-Derived Population-Weighted $PM_{2.5}$ Air Quality Metrics for Health Benefits Endpoints Due to the Locomotive and Marine Engine Proposed Standards

	2020	**2030**
Statistic[a]	**Change**[b]	**Change**[b]
$PM_{2.5}$ Metrics: National Population-Weighted Average (ug/m^3)		
Annual Average Concentration	0.05	0.10
[a] $PM_{2.5}$ 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.		
[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.		

6.3 Benefits Analysis – Data and Methods

Given changes in environmental quality (ambient air quality, visibility, nitrogen, and sulfate deposition), the next step is to determine the economic value of those changes. We follow a "damage-function" approach in calculating total benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare endpoints (specific effects that can be associated with changes in air quality) and assigns values to those changes assuming independence of the individual values. Total benefits are calculated simply as the sum of the values for all nonoverlapping health and welfare endpoints. This imposes no overall preference structure and does not account for potential income or substitution effects (i.e., adding a new endpoint will not reduce the value of changes in other endpoints). The "damage-function" approach is the standard approach for most benefit-cost analyses of environmental quality programs and has been used in several recent published analyses (Banzhaf, Burtraw, and Palmer, 2002; Hubbell et al., 2004; Levy et al., 2001; Levy et al., 1999; Ostro and Chestnut, 1998).

To assess economic value in a damage-function framework, the changes in environmental quality must be translated into effects on people or on the things that people value. In some cases, the changes in environmental quality can be directly valued, as is the case for changes in visibility. In other cases, such as for changes in PM, a health and welfare impact analysis must first be conducted to convert air quality changes into effects that can be assigned dollar values. Inherent in each of these steps is a high degree of uncertainty, due both to the randomness of environmental factors such as meteorology, and the difficulty in measuring and predicting model inputs such as pollutant emissions. As such, where possible, we incorporate probabilistic representations of model inputs and outputs. However, in many cases, probabilistic representations are not available. In these cases, we use the best available science and models, and characterize uncertainty using sensitivity analyses.

For the purposes of this RIA, the health impacts analysis is limited to those health effects that are directly linked to ambient levels of air pollution and specifically to those linked to $PM_{2.5}$. These impacts may be positive or negative, but in general, for the proposed standards, they are expected to be small relative to the direct air pollution-related impacts.

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

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

6.3.1 Valuation Concepts

In valuing health impacts, we note that reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects by a fairly small amount for a large population. The appropriate economic measure is willingness to pay[E] (WTP) for changes in risk prior to the regulation (Freeman, 2003).[F] Adoption of WTP as the measure of value implies that the value of environmental quality improvements depends on the individual preferences of the affected population and that the existing distribution of income (ability to pay) is appropriate. For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as the measure of benefits. These cost of illness (COI) estimates generally (although not in every case) understate the true value of reductions in risk of a health effect, because they do not include the value of avoided pain and suffering from the health effect (Harrington and Portney, 1987; Berger et al., 1987).

[E] For many goods, WTP can be observed by examining actual market transactions. For example, if a gallon of bottled drinking water sells for $1, it can be observed that at least some people are willing to pay $1 for such water. For goods not exchanged in the market, such as most environmental "goods," valuation is not as straightforward. Nevertheless, a value may be inferred from observed behavior, such as sales and prices of products that result in similar effects or risk reductions (e.g., nontoxic cleaners or bike helmets). Alternatively, surveys can be used in an attempt to directly elicit WTP for an environmental improvement.

[F] In general, economists tend to view an individual's WTP for an improvement in environmental quality as the appropriate measure of the value of a risk reduction. An individual's willingness to accept (WTA) compensation for not receiving the improvement is also a valid measure. However, WTP is generally considered to be a more readily available and conservative measure of benefits. In some cases, such as the value of fatal risk reductions, we use WTA measures due to the difficulty in obtaining WTP estimates. For cases where the changes in the good are small WTP and WTA are approximately equal.

One distinction in environmental benefits estimation is between use values and nonuse values. Although no general agreement exists among economists on a precise distinction between the two (see Freeman [2003]), the general nature of the difference is clear. Use values are those aspects of environmental quality that affect an individual's welfare directly. These effects include changes in product prices, quality, and availability; changes in the quality of outdoor recreation and outdoor aesthetics; changes in health or life expectancy; and the costs of actions taken to avoid negative effects of environmental quality changes.

Nonuse values are those for which an individual is willing to pay for reasons that do not relate to the direct use or enjoyment of any environmental benefit but might relate to existence values and bequest values. Nonuse values are not traded, directly or indirectly, in markets. For this reason, measuring nonuse values has proven to be significantly more difficult than measuring use values. The air quality changes produced by the proposed standards would cause changes in both use and nonuse values, but the monetary benefits estimates are almost exclusively for use values.

More frequently than not, the economic benefits from environmental quality changes are not traded in markets, so direct measurement techniques cannot be used. There are three main nonmarket valuation methods used to develop values for endpoints considered in this analysis: stated preference (including contingent valuation [CV]), indirect market (e.g., hedonic wage), and avoided cost methods.

The stated preference method values endpoints by using carefully structured surveys to ask a sample of people what amount of compensation is equivalent to an improvement in environmental quality. There is an extensive scientific literature and body of practice on both the theory and technique of stated preference-based valuation. Well-designed and well-executed stated preference studies are valid for estimating the benefits of air quality regulations.[G] Stated preference valuation studies form the complete or partial basis for valuing a number of health and welfare endpoints, including the value of mortality risk reductions, chronic bronchitis (CB) risk reductions, minor illness risk reductions, and visibility improvements.

Indirect market methods can also be used to infer the benefits of pollution reduction. The most important application of this technique for our analysis is the calculation of the VSL for use in estimating benefits from mortality risk reductions. No market exists where changes in the probability of death are directly exchanged. However, people make decisions about occupation, precautionary behavior, and other activities associated with changes in the risk of death. By examining these risk changes and the other characteristics of people's choices, it is possible to

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

infer information about the monetary values associated with changes in mortality risk (see Section 6.3.5).

Avoided cost methods are ways to estimate the costs of pollution by using the expenditures made necessary by pollution damage. For example, if buildings must be cleaned or painted more frequently as levels of PM increase, then the appropriately calculated increment of these costs is a reasonable lower-bound estimate (under most, although not all, conditions) of true economic benefits when PM levels are reduced. Avoided costs methods are also used to estimate some of the health-related benefits related to morbidity, such as hospital admissions (see Section 6.3.5). In general, avoided cost methods should be used only if there is no information available using other valuation methods (OMB Circular A-4 offers some additional caution on the use of avoided cost methods).

6.3.2 Growth in WTP Reflecting National Income Growth Over Time

Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. There is substantial empirical evidence that the income elasticity[H] of WTP for health risk reductions is positive, although there is uncertainty about its exact value. Thus, as real income increases, the WTP for environmental improvements also increases. Although many analyses assume that the income elasticity of WTP is unit elastic (i.e., a 10% higher real income level implies a 10% higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefits estimates in two different ways: through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the Science Advisory Board (SAB) advised EPA to adjust WTP for increases in real income over time but not to adjust WTP to account for cross-sectional income differences "because of the sensitivity of making such distinctions, and because of insufficient evidence available at present" (U.S. EPA-SAB, 2000a). A recent advisory by another committee associated with the SAB, the Advisory Council on Clean Air Compliance Analysis, has provided conflicting advice. While agreeing with "the general principle that the willingness to pay to reduce mortality risks is likely to increase with growth in real income (U.S. EPA-SAB, 2004a, p. 52)" and that "The same increase should be assumed for the WTP for serious nonfatal health effects (U.S. EPA-SAB, 2004a, p. 52)," they note that "given the limitations and uncertainties in the available empirical evidence, the Council does not support the use of the proposed adjustments for aggregate income growth as part of the primary analysis (U.S. EPA-SAB, 2004a, p. 53)." Until these conflicting advisories have been

[H] Income elasticity is a common economic measure equal to the percentage change in WTP for a 1% change in income.

reconciled, EPA will continue to adjust valuation estimates to reflect income growth using the methods described below, while providing sensitivity analyses for alternative income growth adjustment factors.

Based on a review of the available income elasticity literature, we adjusted the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, we applied estimates derived from cross-sectional studies in our analysis. Details of the procedure can be found in Kleckner and Neumann (1999). An abbreviated description of the procedure we used to account for WTP for real income growth between 1990 and 2020 is presented below.

Reported income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP. As such, we use different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. Note that because of the variety of empirical sources used in deriving the income elasticities, there may appear to be inconsistencies in the magnitudes of the income elasticities relative to the severity of the effects (*a priori* one might expect that more severe outcomes would show less income elasticity of WTP). We have not imposed any additional restrictions on the empirical estimates of income elasticity. One explanation for the seeming inconsistency is the difference in timing of conditions. WTP for minor illnesses is often expressed as a short term payment to avoid a single epidsode. WTP for major illnesses and mortality risk reductions are based on longer term measures of payment (such as wages or annual income). Economic theory suggests that relationships become more elastic as the length of time grows, reflecting the ability to adjust spending over a longer time period. Based on this theory, it would be expected that WTP for reducing long term risks would be more elastic than WTP for reducing short term risks. We also expect that the WTP for improved visibility in Class I areas would increase with growth in real income. The relative magnitude of the income elasticity of WTP for visibility compared with those for health effects suggests that visibility is not as much of a necessity as health, thus, WTP is more elastic with respect to income. The elasticity values used to adjust estimates of benefits in 2020 and 2030 are presented in Table 6.3-1.

Table 6.3-1. Elasticity Values Used to Account for Projected Real Income Growth[a]

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

[a] Derivation of estimates can be found in Kleckner and Neumann (1999) and Chestnut (1997). COI estimates are assigned an adjustment factor of 1.0.

In addition to elasticity estimates, projections of real gross domestic product (GDP) and populations from 1990 to 2020 and 2030 are needed to adjust benefits to reflect real per capita income growth. For consistency with the emissions and benefits modeling, we used national population estimates for the years 1990 to 1999 based on U.S. Census Bureau estimates (Hollman, Mulder, and Kallan, 2000). These population estimates are based on application of a cohort-component model applied to 1990 U.S. Census data projections (U.S. Bureau of Census, 2000). For the years between 2000 and 2030, we applied growth rates based on the U.S. Census

Bureau projections to the U.S. Census estimate of national population in 2000. We used projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010.[I] We used projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's (2000) for the years 2010 to 2024.[J] We were unable to find reliable projections of GDP past 2024. As such, we assume that per capita GDP remains constant between 2024 and 2030.

Using the method outlined in Kleckner and Neumann (1999) and the population and income data described above, we calculated WTP adjustment factors for each of the elasticity estimates listed in Table 6.3-1. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) are adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. Table 6.3-2 lists the estimated adjustment factors. Note that, for premature mortality, we applied the income adjustment factor to the present discounted value of the stream of avoided mortalities occurring over the lag period. Also note that because of a lack of data on the dependence of COI and income, and a lack of data on projected growth in average wages, no adjustments are made to benefits based on the COI approach or to work loss days and worker productivity. This assumption leads us to underpredict benefits in future years because it is likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

Table 6.3-2. Adjustment Factors Used to Account for Projected Real Income Growth[a]

Benefit Category	2020	2030
Minor Health Effect	1.066	1.076
Severe and Chronic Health Effects	1.229	1.266
Premature Mortality	1.201	1.233
Visibility	1.517	1.613

[a] Based on elasticity values reported in Table 6.3-1, U.S. Census population projections, and projections of real GDP per capita.

6.3.3 Demographic Projections

Quantified and monetized human health impacts depend on the demographic characteristics of the population, including age, location, and income. We use projections based on economic forecasting models developed by Woods and Poole, Inc. The Woods and Poole (WP) database contains county-level projections of population by age, sex, and race out to 2025. Projections in each county are determined simultaneously with every other county in the United States to take into account patterns of economic growth and migration. The sum of growth in

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

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

county-level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates (Hollman, Mulder, and Kallan, 2000). According to WP, linking county-level growth projections together and constraining to a national-level total growth avoids potential errors introduced by forecasting each county independently. County projections are developed in a four-stage process. First, national-level variables such as income, employment, and populations are forecasted. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an "export-base" approach, which relies on linking industrial-sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-based approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector. Third, population is projected for each economic area based on net migration rates derived from employment opportunities and following a cohort-component method based on fertility and mortality in each area. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2030 based on historical rates of mortality, fertility, and migration.

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

As noted above, values for environmental quality improvements are expected to increase with growth in real per capita income. Accounting for real income growth over time requires projections of both real GDP and total U.S. populations. For consistency with the emissions and benefits modeling, we used national population estimates based on the U.S. Census Bureau projections.

6.3.4 Methods for Describing Uncertainty

The NRC (2002) highlighted the need for EPA to conduct rigorous quantitative analysis of uncertainty in its benefits estimates as well as the need for presenting these estimates to decision makers in ways that foster an appropriate appreciation of their inherent uncertainty. In response to these comments, EPA has initiated the development of a comprehensive methodology for characterizing the aggregate impact of uncertainty in key modeling elements on both health incidence and benefits estimates

In the current analysis, consistent with the approach used in the RIA for the recent PM NAAQS, EPA continues to move forward on one of the key recommendations of the NRC – moving the assessment of uncertainties from its ancillary analyses into its main benefits presentation through the conduct of probabilistic analyses. In this proposed rule, EPA addressed

key sources of uncertainty by Monte Carlo propagation of uncertainty in the concentration-response (C-R) functions and economic valuation functions through its base estimates as well as by continuing its practice of conducting a series of ancillary sensitivity analyses examining the impact of alternate assumptions on the benefits estimates. It should be noted that the Monte Carlo-generated distributions of benefits reflect only some of the uncertainties in the input parameters. Uncertainties associated with emissions, air quality modeling, populations, and baseline health effect incidence rates are not represented in the distributions of benefits of attaining alternative standards. Issues such as correlation between input parameters and the identification of reasonable upper and lower bounds for input distributions characterizing uncertainty in additional model elements will be addressed in future versions of the uncertainty framework.

In benefit analyses of air pollution regulations conducted to date, the estimated impact of reductions in premature mortality has accounted for 85% to 95% of total benefits. Therefore, in characterizing the uncertainty related to the estimates of total benefits it is particularly important to attempt to characterize the uncertainties associated with this endpoint. As such the analysis for this rule incorporates the results of our recent expert elicitation to characterize uncertainty in the effect estimates used to estimate premature mortality resulting from exposures to PM into the main analysis. In collaboration with OMB, EPA completed a pilot expert elicitation in 2004, which was used to characterize uncertainty in the PM mortality C-R function in the Nonroad Diesel and CAIR RIAs. EPA has recently completed a full-scale expert elicitation that incorporated peer-review comments on the pilot application, and that provides a more robust characterization of the uncertainty in the premature mortality function. This expert elicitation was designed to evaluate uncertainty in the underlying causal relationship, the form of the mortality impact function (e.g., threshold versus linear models) and the fit of a specific model to the data (e.g., confidence bounds for specific percentiles of the mortality effect estimates). Additional issues, such as the ability of long-term cohort studies to capture premature mortality resulting from short-term peak PM exposures, were also addressed in the expert elicitation.

For this proposal, consistent with the approach used in the RIA for the recent PM NAAQS, EPA addressed key sources of uncertainty through Monte Carlo propagation of uncertainty in the C-R functions and economic valuation functions and through a series of sensitivity analyses examining the impact of alternate assumptions on the benefits estimates that are generated. It should be noted that the Monte Carlo-generated distributions of benefits reflect only some of the uncertainties in the input parameters. Uncertainties associated with emissions, air quality modeling, populations, and baseline health effect incidence rates are not represented in the distributions of benefits of attaining alternative standards.

Our distributions of total benefits do not completely represent full uncertainty because of the uncertainty in model elements discussed below (see Table 6.3-3). Uncertainty about specific aspects of the health and welfare estimation models is discussed in greater detail in the following sections. The estimated distributions of total benefits may not completely capture the shape and location of the actual distribution of total benefits.

6.3.4.1 Sources of Uncertainty

In any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty. This analysis is no exception. As outlined both in this and preceding chapters, many inputs were used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, estimates of values (both from WTP and COI studies), population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Each of these inputs may be uncertain and, depending on its role in the benefits analysis, may have a disproportionately large impact on final estimates of total benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emission levels can lead to large impacts on total benefits.

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

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

Some of the key uncertainties in the benefits analysis are presented in Table 6.3-3.

More specifically, there are key uncertainties in many aspects of the health impact functions used in our analyses. These are discussed in detail in the following section.

Table 6.3-3. Primary Sources of Uncertainty in the Benefits Analysis

1. Uncertainties Associated with Impact Functions
● The value of the PM effect estimate in each impact function.
● Application of a single impact function to pollutant changes and populations in all locations.
● Similarity of future-year impact functions to current impact functions.
● Correct functional form of each impact function.
● Extrapolation of effect estimates beyond the range of PM concentrations observed in the source epidemiological study.
● Application of some impact functions only to those subpopulations matching the original study population.
2. Uncertainties Associated with PM Concentrations
● Responsiveness of the models to changes in precursor emissions resulting from the control policy.
● Projections of future levels of precursor emissions, especially organic carbonaceous particle emissions.
● Model chemistry for the formation of ambient nitrate concentrations.
● Lack of speciation monitors in some areas requires extrapolation of observed speciation data.
● CMAQ model performance in the Western U.S., especially California indicates significant underprediction of $PM_{2.5}$.
3. Uncertainties Associated with PM Mortality Risk
● Differential toxicity of specific component species within the complex mixture of PM has not been determined.
● The extent to which adverse health effects are associated with low-level exposures that occur many times in the year versus peak exposures.
● The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study.
● Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated with Possible Lagged Effects
● The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels that would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
5. Uncertainties Associated with Baseline Incidence Rates
● Some baseline incidence rates are not location specific (e.g., those taken from studies) and therefore may not accurately represent the actual location-specific rates.
● Current baseline incidence rates may not approximate well baseline incidence rates in 2020 and 2030.
● Projected population and demographics may not represent well future-year population and demographics.
6. Uncertainties Associated with Economic Valuation
● Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them.
● Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates because of differences in income or other factors.
7. Uncertainties Associated with Aggregation of Monetized Benefits
● Health and welfare benefits estimates are limited to the available impact functions. Thus, unquantified or unmonetized benefits are not included.

6.3.4.2 Uncertainties Associated with Health Impact Functions based on Reported Effect Estimates from the Epidemiological Literature

Within-Study Variation. Within-study variation refers to the precision with which a given study estimates the relationship between air quality changes and health effects. Health effects studies provide both a "best estimate" of this relationship plus a measure of the statistical uncertainty of the relationship. The size of this uncertainty depends on factors such as the number of subjects studied and the size of the effect being measured. The results of even the most well-designed epidemiological studies are characterized by this type of uncertainty, though

well-designed studies typically report narrower uncertainty bounds around the best estimate than do studies of lesser quality. In selecting health endpoints, we generally focus on endpoints where a statistically significant relationship has been observed in at least some studies, although we may pool together results from studies with both statistically significant and insignificant estimates to avoid selection bias.

Across-Study Variation. Across-study variation refers to the fact that different published studies of the same pollutant/health effect relationship typically do not report identical findings; in some instances the differences are substantial. These differences can exist even between equally well designed and executed studies and may result in health effect estimates that vary considerably. Across-study variation can result from a variety of possible causes. Such differences might simply be associated with different measurement techniques. Sources of variation can be introduced by the air quality monitoring technique, measurement averaging times, health endpoint data sources (differences in the way medical records are kept at different institutions or questionnaire wording). One possibility is that estimates of the single true relationship between a given pollutant and a health effect differ across studies because of differences in study design, random chance, or other factors. For example, a hypothetical study conducted in New York and one conducted in Seattle may report different C-R functions for the relationship between PM and mortality, in part because of differences between these two study populations (e.g., demographics, activity patterns). Alternatively, study results may differ because these two studies are in fact estimating different relationships; that is, the same reduction in PM in New York and Seattle may result in different reductions in premature mortality. This may result differences in the relative sensitivity of these two populations to PM pollution and differences in the composition of PM in these two locations, as well as other factors. In either case, where we identified multiple studies that are appropriate for estimating a given health effect, we generated a pooled estimate of results from each of those studies.

Application of C-R Relationship Nationwide. Regardless of the use of impact functions based on effect estimates from a single epidemiological study or multiple studies, each impact function was applied uniformly throughout the United States to generate health benefit estimates. However, to the extent that pollutant/health effect relationships are region specific, applying a location-specific impact function at all locations in the United States may result in overestimates of health effect changes in some locations and underestimates of health effect changes in other locations. It is not possible, however, to know the extent or direction of the overall effect on health benefit estimates introduced by applying a single impact function to the entire United States. This may be a significant uncertainty in the analysis, but the current state of the scientific literature does not allow for a region-specific estimation of health benefits for most health outcomes.[K]

Extrapolation of Impact Functions Across Populations. Epidemiological studies often focus on specific age ranges, either due to data availability limitations (e.g., most hospital admission data come from Medicare records, which are limited to populations 65 and older), or to simplify

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

data collection (e.g., some asthma symptom studies focus on children at summer camps, which usually have a limited age range). We have assumed for the primary analysis that most impact functions should be applied only to those populations with ages that strictly match the populations in the underlying epidemiological studies. However, in many cases, there is no biological reason why the observed health effect would not also occur in other populations within a reasonable range of the studied population. For example, Dockery et al. (1996) examined acute bronchitis in children aged 8 to 12. There is no biological reason to expect a very different response in children aged 6 or 14. By excluding populations outside the range in the studies, we may be underestimating the health impact in the overall population. In response to recommendations from the SAB-HES, where there appears to be a reasonable physiological basis for expanding the age group associated with a specific effect estimate beyond the study population to cover the full age group (e.g., expanding from a study population of 7 to 11 year olds to the full 6- to 18-year child age group), we have done so and used those expanded incidence estimates in the primary analysis.

Uncertainties in Concentration-Response Functions. The following uncertainties exist in almost all concentration-response functions for PM-related health effects. For expository purposes, and because of the importance of mortality, we focus the discussion on how these uncertainties affect the PM mortality concentration-response functions.

Causality: Epidemiological studies are not designed to definitively prove causation. For the analysis of the proposed standards, we assumed a causal relationship between exposure to elevated PM and premature mortality, based on the consistent evidence of a correlation between PM and mortality reported in the substantial body of published scientific literature (CASAC, 2005). As with all health effects included in our analysis, a weight of evidence process is used to evaluate endpoints before including them in the analysis.

Other Pollutants: PM concentrations are correlated with the concentrations of other criteria pollutants, such as ozone and CO. To the extent that there is correlation, this analysis may be assigning mortality effects to PM exposure that are actually the result of exposure to other pollutants. Recent studies suggest that ozone may have mortality effects independent of PM.

Shape of the C-R Function: The shape of the true PM mortality C-R function is uncertain, but this analysis assumes the C-R function has a non-threshold log-linear form throughout the relevant range of exposures. If this is not the correct form of the C-R function, or if certain scenarios predict concentrations well above the range of values for which the C-R function was fitted, avoided mortality may be misestimated.

In addition, there is ongoing debate as to whether there exists a threshold below which there would be no benefit to further reductions in $PM_{2.5}$. Some researchers have hypothesized the presence of a threshold relationship. The nature of the hypothesized relationship is the possibility that there exists a PM concentration level below which further reductions no longer yield premature mortality reduction benefits. EPA's most recent $PM_{2.5}$ Criteria Document concludes that "the available evidence does not either support or refute the existence of

thresholds for the effects of PM on mortality across the range of concentrations in the studies" (U.S. EPA, 2004b, p. 9-44). EPA's Science Advisory Board (SAB) that provides advice on benefits analysis methods[L] has recommended modeling premature mortality associated with PM exposure as a non-threshold effect, that is, with harmful effects to exposed populations regardless of the absolute level of ambient PM concentrations.

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

Relative Toxicity of PM Component Species: In this analysis, all fine particles, regardless of their chemical composition, are assumed to be equally potent in causing premature mortality. This is an important assumption, because there may be significant differences between PM produced via transported precursors, direct PM released from automotive engines, and direct PM from other industrial sources. The analysis also assumes that all components of fine particles have equal toxicity (because the available epidemiological effect estimates are based on total $PM_{2.5}$ mass rather than the mass of individual component species). While it is reasonable to expect that the potency of components may vary across the numerous effect categories associated with particulate matter, EPA's interpretation of scientific information considered to date is that such information does not yet provide a basis for quantification beyond using fine particle mass. However, to provide information that may be useful as additional studies become available, we are providing estimates of the proportions of benefits that are attributable to specific components of $PM_{2.5}$, e.g., ammonium sulfate, ammonium nitrate, elemental carbon, organic carbon, and crustal material (which includes metals). This apportionment does not make any assumptions about the relative toxicity of the different species; rather, it divides total benefits based on the contribution of reductions in individual component species to the overall reduction in $PM_{2.5}$ mass.

Lag Time Between Change in Exposure and Health Impact: There is a time lag between changes in PM exposures and the total realization of changes in health effects. Within the context of benefits analyses, this term is often referred to as "cessation lag." For the chronic PM/mortality relationship, the length of the cessation lag is unknown. The existence of such a lag is important for the valuation of premature mortality incidence because economic theory suggests that benefits occurring in the future should be discounted. There is no specific scientific evidence of the existence or structure of a health effects cessation lag for reductions in exposures to fine PM.

[L] The advice from the 2004 SAB-HES (U.S. EPA-SAB, 2004b) is characterized by the following: "For the studies of long-term exposure, the HES notes that Krewski et al. (2000) have conducted the most careful work on this issue. They report that the associations between $PM_{2.5}$ and both all-cause and cardiopulmonary mortality were near linear within the relevant ranges, with no apparent threshold. Graphical analyses of these studies (Dockery et al., 1993, Figure 3, and Krewski et al., 2000, page 162) also suggest a continuum of effects down to lower levels. Therefore, it is reasonable for EPA to assume a no threshold model down to, at least, the low end of the concentrations reported in the studies."

Information about latency (the amount of time between exposure and onset of a health effect) may inform our understanding of cessation lags.

Scientific literature on adverse health effects similar to those associated with PM (e.g., smoking-related disease) and the difference in the effect size between chronic exposure studies and daily mortality studies suggests that all incidences of premature mortality reduction associated with a given incremental change in PM exposure probably would not occur in the same year as the exposure reduction. The smoking-related literature also implies that lags of up to a few years or longer are plausible, although it is worth noting here that in the case of ambient air pollution we are predicting the effects of reduced exposure rather than complete cessation. The SAB-HES suggests that appropriate lag structures may be developed based on the distribution of cause-specific deaths within the overall all-cause estimate. Diseases with longer progressions should be characterized by long-term lag structures, while impacts occurring in populations with existing disease may be characterized by short-term lags.

A key question is the distribution of causes of death within the relatively broad categories analyzed in the cohort studies used. While we may be more certain about the appropriate length of cessation lag for lung cancer deaths, it is not clear what the appropriate lag structure should be for different types of cardiopulmonary deaths, which include both respiratory and cardiovascular causes. Some respiratory diseases may have a long period of progression, while others, such as pneumonia, have a very short duration. In the case of cardiovascular disease, there is an important question of whether air pollution is causing the disease, which would imply a relatively long cessation lag, or whether air pollution is causing premature death in individuals with preexisting heart disease, which would imply very short cessation lags.

The SAB-HES provides several recommendations for future research that could support the development of defensible lag structures, including the use of disease-specific lag models, and the construction of a segmented lag distribution to combine differential lags across causes of death. The SAB-HES recommended that until additional research has been completed, EPA should assume a segmented lag structure characterized by 30% of mortality reductions occurring in the first year, 50% occurring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 20% occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$ (EPA-COUNCIL-LTR-05-001, 2004). The distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. For future analyses, the specific distribution of deaths over time will need to be determined through research on causes of death and progression of diseases associated with air pollution. It is important to keep in mind that changes in the lag assumptions do not change the total number of estimated deaths but rather the timing of those deaths.

Cumulative Effects: We attribute the PM-mortality relationship in the underlying epidemiological studies to cumulative exposure to PM. However, the relative roles of PM exposure duration and PM exposure level in inducing premature mortality are still uncertain at this time.

6.3.5 Health Benefits Assessment Methods

The largest monetized benefits of reducing ambient concentrations of PM and ozone are attributable to reductions in health risks associated with air pollution. EPA's Criteria Documents for ozone and PM list numerous health effects known to be linked to ambient concentrations of these pollutants (EPA, 2006; 2006). As discussed above, quantification of health impacts requires several inputs, including epidemiological effect estimates (concentration-response functions), baseline incidence and prevalence rates, potentially affected populations, and estimates of changes in ambient concentrations of air pollution. Previous sections have described the population and air quality inputs. This section describes the effect estimates and baseline incidence and prevalence inputs and the methods used to quantify and monetize changes in the expected number of incidences of various health effects. These include premature mortality, nonfatal heart attacks, chronic bronchitis, acute bronchitis, hospital admissions, emergency room visits for asthma, upper and lower respiratory symptoms, asthma exacerbations, minor restricted activity days and days of work lost.

As discussed above, we have chosen to not include estimates of ozone-related health effects in this analysis, though the proposed standards are expected to reduce ambient levels of ozone. Some health effects are excluded from this analysis for three reasons: the possibility of double-counting, uncertainties in applying effect relationships based on clinical studies to the affected population, or a lack of an established relationship between the health effect and pollutant in the published epidemiological literature. Unquantifed effects are listed in Table 6.1-2. An improvement in ambient $PM_{2.5}$ and ozone air quality may reduce the number of incidences within each unquantified effect category that the U.S. population would experience. Although these health effects are believed to be PM and ozone induced, effect estimates are not available for quantifying the benefits associated with reducing these effects. The inability to quantify these effects lends a downward bias to the monetized benefits presented in this analysis.

6.3.5.1 Selection of Health Endpoints

We base our selection of health endpoints on consistency with EPA Criteria Documents and Staff Papers, with input and advice from the EPA Science Advisory Board Health Effects Subcommittee, a scientific review panel specifically established to provide advice on the use of the scientific literature in developing benefits analyses for air pollution regulations (http://www.epa.gov/sab/). In general, we follow a weight of evidence approach, based on the biological plausibility of effects, availability of concentration-response functions from well-conducted peer-reviewed epidemiological studies, cohesiveness of results across studies, and a focus on endpoints reflecting public health impacts (like hospital admissions) rather than physiological responses (such as changes in clinical measures like Forced Expiratory Volume (FEV1)).

6.3.5.2 Sources of Information for Effect Estimates

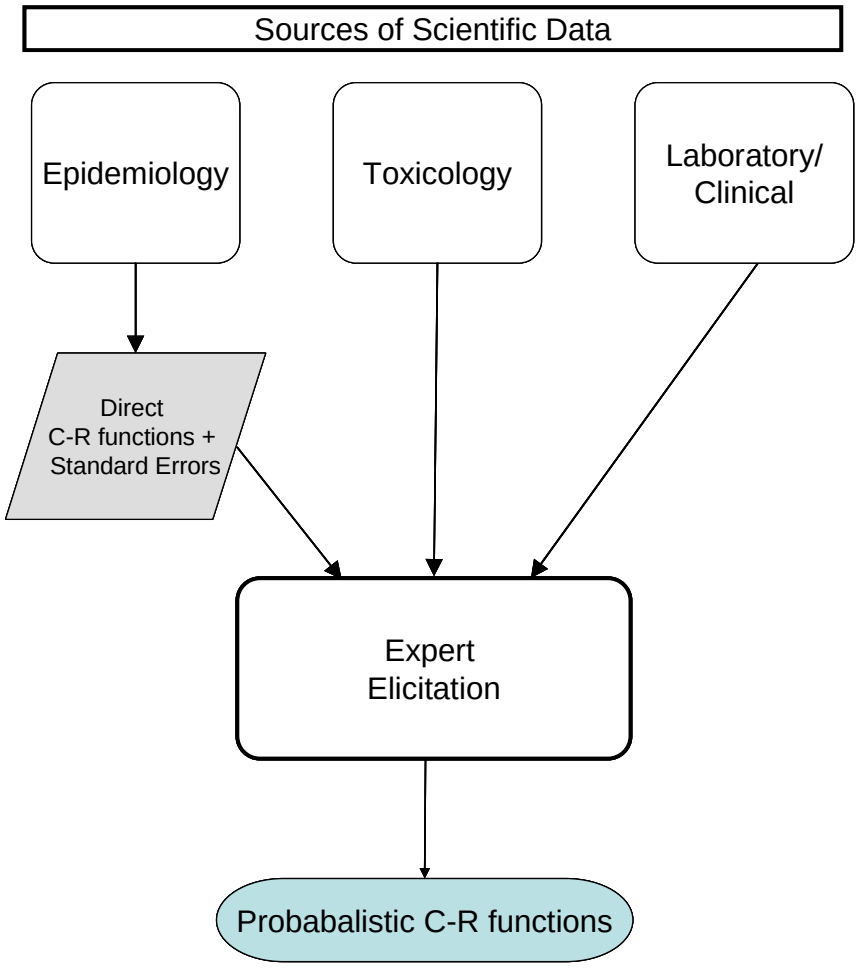
There are several types of data that can support the determination of types and magnitude of health effects associated with air pollution exposures. These sources of data include toxicological studies (including animal and cellular studies), human clinical trials, and observational epidemiology studies. All of these data sources provide important contributions to

the weight of evidence surrounding a particular health impact, however, only epidemiology studies provide direct concentration-response relationships which can be used to evaluate population-level impacts of reductions in ambient pollution levels.

However, standard environmental epidemiology studies provide only a limited representation of the uncertainty associated with a specific C-R function, measuring only the statistical error in the estimates, and usually relating more to the power of the underlying study (driven largely by population size and the frequency of the outcome measure). There are many other sources of uncertainty in the relationships between ambient pollution and population level health outcomes, including many sources of model uncertainty, such as model specification, potential confounding between factors that are both correlated with the health outcome and each other, and many other factors. As such, in recent years, EPA has begun investigating how expert elicitation methods can be used to integrate across various sources of data in developing C-R functions for regulatory benefits analyses.

Expert elicitation is useful in integrating the many sources of information about uncertainty in the C-R function, because it allows experts to synthesize these data sources using their own mental models, and provide a probabilistic representation of their synthesis of the data in the form of a probability distribution of the C-R function. Figure 6.3-1 shows how expert elicitation builds on both the direct empirical data on C-R relationships and other less direct evidence to develop probabilistic distributions of C-R functions. EPA has used expert elicitation to inform the regulatory process in the past (see for example the previous staff paper for the lead NAAQS, U.S. EPA, 1990). In the current analysis, we have only used expert elicitation to characterize the C-R function for the relationship between fine PM and premature mortality. However, similar methods could be used to characterize C-R functions for other health outcomes.

Figure 6.3-1. Sources and Integration of Scientific Data in Informing Development of Health Impact Functions



6.3.5.3 Information Used in Quantifying C-R Functions

For the data-derived estimates, we relied on the published scientific literature to ascertain the relationship between PM and adverse human health effects. We evaluated epidemiological studies using the selection criteria summarized in Table 6.3-4. These criteria include consideration of whether the study was peer-reviewed, the match between the pollutant studied and the pollutant of interest, the study design and location, and characteristics of the study population, among other considerations. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility.

In general, the use of results from more than a single study can provide a more robust estimate of the relationship between a pollutant and a given health effect. However, there are often differences between studies examining the same endpoint, making it difficult to pool the results in a consistent manner. For example, studies may examine different pollutants or different age groups. For this reason, we consider very carefully the set of studies available

examining each endpoint and select a consistent subset that provides a good balance of population coverage and match with the pollutant of interest. In many cases, either because of a lack of multiple studies, consistency problems, or clear superiority in the quality or comprehensiveness of one study over others, a single published study is selected as the basis of the effect estimate.

When several effect estimates for a pollutant and a given health endpoint have been selected, they are quantitatively combined or pooled to derive a more robust estimate of the relationship. The BenMAP Technical Appendices provides details of the procedures used to combine multiple impact functions (Abt Associates, 2005). In general, we used fixed or random effects models to pool estimates from different studies of the same endpoint. Fixed effects pooling simply weights each study's estimate by the inverse variance, giving more weight to studies with greater statistical power (lower variance). Random effects pooling accounts for both within-study variance and between-study variability, due, for example, to differences in population susceptibility. We used the fixed effects model as our null hypothesis and then determined whether the data suggest that we should reject this null hypothesis, in which case we would use the random effects model.[M] Pooled impact functions are used to estimate hospital admissions and asthma exacerbations. For more details on methods used to pool incidence estimates, see the BenMAP Technical Appendices (Abt Associates, 2005), which are available with the BenMAP software at http://www.epa.gov/ttn/ecas/benmodels.html.

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

The specific studies from which effect estimates for the primary analysis are drawn are included in Table 6.3-5. In all cases where effect estimates are drawn directly from epidemiological studies, standard errors are used as a partial representation of the uncertainty in the size of the effect estimate. Detailed information about the form and parameters of each impact function used in this analysis can be found in the BenMAP users manual.[N] For those functions not included in the BenMAP users manual, we include a technical memo to the docket for this rule. Below we provide the basis for selecting these studies.

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

[N] Interested parties may wish to consult the webpage http://www.epa.gov/ttn/ecas/benmodels.html to download the BenMAP users manual. The users manual is also included in the docket for this rule.

Table 6.3-4 Summary of Considerations Used in Selecting C-R Functions

Consideration	Comments
Peer-Reviewed Research	Peer-reviewed research is preferred to research that has not undergone the peer-review process.
Study Type	Among studies that consider chronic exposure (e.g., over a year or longer), prospective cohort studies are preferred over ecological studies because they control for important individual-level confounding variables that cannot be controlled for in ecological studies.
Study Period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and lifestyle over time. However, when there are only a few studies available, studies from all years will be included.
Population Attributes	The most technically appropriate measures of benefits would be based on impact functions that cover the entire sensitive population but allow for heterogeneity across age or other relevant demographic factors. In the absence of effect estimates specific to age, sex, preexisting condition status, or other relevant factors, it may be appropriate to select effect estimates that cover the broadest population to match with the desired outcome of the analysis, which is total national-level health impacts. When available, multi-city studies are preferred to single city studies because they provide a more generalizable representation of the C-R function.
Study Size	Studies examining a relatively large sample are preferred because they generally have more power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population or through repeated observations on a smaller population (e.g., through a symptom diary recorded for a panel of asthmatic children).
Study Location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior, and lifestyle.
Pollutants Included in Model	When modeling the effects of ozone and PM (or other pollutant combinations) jointly, it is important to use properly specified impact functions that include both pollutants. Using single-pollutant models in cases where both pollutants are expected to affect a health outcome can lead to double-counting when pollutants are correlated.
Measure of PM	For this analysis, impact functions based on $PM_{2.5}$ are preferred to PM_{10} because of the focus on reducing emissions of $PM_{2.5}$ precursors, and because air quality modeling was conducted for this size fraction of PM. Where $PM_{2.5}$ functions are not available, PM_{10} functions are used as surrogates, recognizing that there will be potential downward (upward) biases if the fine fraction of PM_{10} is more (less) toxic than the coarse fraction.
Economically Valuable Health Effects	Some health effects, such as forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are not quantified in this analysis.
Nonoverlapping Endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double-counting of benefits.

Table 6.3-5. Endpoints and Studies Used to Calculate Total Monetized Health Benefits

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature mortality — cohort study, all-cause	$PM_{2.5}$	Pope et al. (2002)[9]	>29 years
		Laden et al. (2006)[10]	>25 years
Premature mortality, total exposures	$PM_{2.5}$	Expert Elicitation (IEc, 2006)[11]	>24 years
Premature mortality — all-cause	$PM_{2.5}$	Woodruff et al. (1997)[12]	Infant (<1 year)
Chronic Illness			
Chronic bronchitis	$PM_{2.5}$	Abbey et al. (1995)[13]	>26 years
Nonfatal heart attacks	$PM_{2.5}$	Peters et al. (2001)[14]	Adults
Hospital Admissions			
Respiratory		Pooled estimate:	>64 years
	$PM_{2.5}$	Moolgavkar (2003)—ICD 490-496 (COPD)[15]	
		Ito (2003)—ICD 490-496 (COPD)[16]	
	$PM_{2.5}$	Moolgavkar (2000)—ICD 490-496 (COPD)[17]	20–64 years
	$PM_{2.5}$	Ito (2003)—ICD 480-486 (pneumonia)	>64 years
	$PM_{2.5}$	Sheppard (2003)—ICD 493 (asthma)[18]	<65 years
Cardiovascular	$PM_{2.5}$	Pooled estimate:	>64 years
		Moolgavkar (2003)—ICD 390-429 (all cardiovascular)	
		Ito (2003)—ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	
	$PM_{2.5}$	Moolgavkar (2000)—ICD 390-429 (all cardiovascular)	20–64 years
Asthma-related ER visits	$PM_{2.5}$	Norris et al. (1999)[19]	0–18 years
Other Health Endpoints			
Acute bronchitis	$PM_{2.5}$	Dockery et al. (1996)[20]	8–12 years
Upper respiratory symptoms	$PM_{2.5}$	Pope et al. (1991)[21]	Asthmatics, 9–11 years
Lower respiratory symptoms	$PM_{2.5}$	Schwartz and Neas (2000)[22]	7–14 years
Asthma exacerbations	$PM_{2.5}$	Pooled estimate:	6–18 yearsa
		Ostro et al. (2001)[23] (cough, wheeze and shortness of breath)	
		Vedal et al. (1998)[24] (cough)	
Work loss days	$PM_{2.5}$	Ostro (1987)[25]	18–65 years
MRADs	$PM_{2.5}$	Ostro and Rothschild (1989)[26]	18–65 years

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

$PM_{2.5}$-Related Adult Premature Mortality – Epidemiological Basis.

Both long- and short-term exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates

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

Although a number of uncertainties remain to be addressed by continued research (NRC, 1998), a substantial body of published scientific literature documents the correlation between elevated PM concentrations and increased mortality rates (US EPA, 2004). Time-series methods have been used to relate short-term (often day-to-day) changes in PM concentrations and changes in daily mortality rates up to several days after a period of elevated PM concentrations. Cohort methods have been used to examine the potential relationship between community-level PM exposures over multiple years (i.e., long-term exposures) and community-level annual mortality rates. Researchers have found statistically significant associations between PM and premature mortality using both types of studies. In general, the risk estimates based on the cohort studies are larger than those derived from time-series studies. Cohort analyses are thought to better capture the full public health impact of exposure to air pollution over time, because they capture the effects of long-term exposures and possibly some component of short-term exposures (Kunzli et al., 2001; NRC, 2002). This section discusses some of the issues surrounding the estimation of premature mortality. To demonstrate the sensitivity of the benefits estimates to the specific sources of information regarding the impact of PM2.5 exposures on the risk of premature death, we are providing estimates in our results tables based on studies derived from the epidemiological literature and from the recent EPA sponsored expert elicitation. The epidemiological studies from which these estimates are drawn are described below. The expert elicitation project and the derivation of effect estimates from the expert elicitation results are described in the next section.

Over a dozen studies have found significant associations between various measures of long-term exposure to PM and elevated rates of annual mortality, beginning with Lave and Seskin (1977). Most of the published studies found positive (but not always statistically significant) associations with available PM indices such as total suspended particles (TSP). However, exploration of alternative model specifications sometimes raised questions about causal relationships (e.g., Lipfert, Morris, and Wyzga [1989]). These early "ecological cross-sectional" studies (e.g., Lave and Seskin [1977]; Ozkaynak and Thurston [1987]) were criticized for a number of methodological limitations, particularly for inadequate control at the individual level for variables that are potentially important in causing mortality, such as wealth, smoking, and diet. Over the last 10 years, several studies using "prospective cohort" designs have been published that appear to be consistent with the earlier body of literature. These new "prospective cohort" studies reflect a significant improvement over the earlier work because they include individual-level information with respect to health status and residence. The most extensive analyses have been based on data from two prospective cohort groups, often referred to as the Harvard "Six-Cities Study" (Dockery et al., 1993; Laden et al, 2006) and the "American Cancer Society or ACS study" (Pope et al., 1995; Pope et al, 2002; Pope et al, 2004); these studies have found consistent relationships between fine particle indicators and premature mortality across multiple locations in the United States. A third major data set comes from the California-based 7th Day Adventist Study (e.g., Abbey et al., 1999), which reported associations between long-term PM exposure and mortality in men. Results from this cohort, however, have been inconsistent, and the air quality results are not geographically representative of most of the United States, and the lifestyle of the population is not reflective of much of the U.S. population.

Analysis is also available for a cohort of adult male veterans diagnosed with hypertension has been examined (Lipfert et al., 2000; Lipfert et al, 2003, 2006). The characteristics of this group differ from the cohorts in the Six-Cities, ACS, and 7th Day Adventist studies with respect to income, race, health status, and smoking status. Unlike previous long-term analyses, this study found some associations between mortality and ozone but found inconsistent results for PM indicators. Because of the selective nature of the population in the veteran's cohort, we have chosen not to include any effect estimates from the Lipfert et al. (2000) study in our benefits assessment.[O]

Given their consistent results and broad geographic coverage, and importance in informing the NAAQS development process, the Six-Cities and ACS data have been particularly important in benefits analyses. The credibility of these two studies is further enhanced by the fact that the initial published studies (Pope et al, 1995 and Dockery et al 1993) were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by HEI (Krewski et al., 2000). The final results of the reanalysis were then independently peer reviewed by a Special Panel of the HEI Health Review Committee. The results of these reanalyses confirmed and expanded those of the original investigators. While the HEI reexamination lends credibility to the original studies, it also highlights sensitivities concerning the relative impact of various pollutants, such as SO2, the potential role of education in mediating the association between pollution and mortality, and the influence of spatial correlation modeling.

Further confirmation and extension of the findings of the 1993 Six City Study and the 1995 ACS study were recently completed using more recent air quality and a longer follow-up period for the ACS cohort was recently published (Pope et al, 2002, 2004; Laden et al, 2006). The follow up to the Harvard Six City Study both confirmed the effect size from the first analysis and provided additional confirmation that reductions in $PM_{2.5}$ are likely to 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 extended analyses of the ACS cohort data (Pope et al., 2002, 2004) 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)

[O] EPA recognizes that the ACS cohort also is not representative of the demographic mix in the general population. The ACS cohort is almost entirely white and has higher income and education levels relative to the general population. EPA's approach to this problem is to match populations based on the potential for demographic characteristics to modify the effect of air pollution on mortality risk. Thus, for the various ACS-based models, we are careful to apply the effect estimate only to ages matching those in the original studies, because age has a potentially large modifying impact on the effect estimate, especially when younger individuals are excluded from the study population. For the Lipfert analysis, the applied population should be limited to that matching the sample used in the analysis. This sample was all male, veterans, and diagnosed hypertensive. There are also a number of differences between the composition of the sample and the general population, including a higher percentage of African Americans (35%) and a much higher percentage of smokers (81% former smokers, 57% current smokers) than the general population (12% African American, 24% current smokers).

substantially increasing exposure data, including additional measurement of cohort exposure to $PM_{2.5}$ following implementation of the $PM_{2.5}$ 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 NRC (2002) also recommended that EPA review the database of cohort studies and consider developing a weighted mean estimate based on selected studies. Because of the differences in the study designs and populations considered in the ACS and Harvard Six-cities studies, we have elected to not pool the results of the studies, instead presenting a range of estimates reflecting the different sources of impact estimates.

In developing and improving the methods for estimating and valuing the potential reductions in mortality risk over the years, EPA consulted with the SAB-HES. That panel recommended using long-term prospective cohort studies in estimating mortality risk reduction (U.S. EPA,1999b). This recommendation has been confirmed by a recent report from the National Research Council, which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure" (NRC, 2002, p. 108). More specifically, the SAB recommended emphasis on the ACS study because it includes a much larger sample size and longer exposure interval and covers more locations (e.g., 50 cities compared to the Six-Cities Study) than other studies of its kind. Because of the refinements in the extended follow-up analysis, the SAB-HES recommends using the Pope et al. (2002) study as the basis for the primary mortality estimate for adults and suggests that alternate estimates of mortality generated using other cohort and time-series studies could be included as part of the sensitivity analysis (U.S. EPA-SAB, 2004b).

The SAB-HES also recommended using the specific estimated relative risks from the Pope et al. (2002) study based on the average exposure to $PM_{2.5}$, measured by the average of two $PM_{2.5}$ measurements, over the periods 1979–1983 and 1999–2000. In addition to relative risks for all-cause mortality, the Pope et al. (2002) study provides relative risks for cardiopulmonary, lung cancer, and all-other cause mortality. Because of concerns regarding the statistical reliability of the all-other cause mortality relative risk estimates, we calculated mortality impacts for the primary analysis based on the all-cause relative risk. Based on our most recently available SAB guidance, we provide mortality impacts based on the ACS study as the best estimate for comparing across the current and previous RIAs. This provides historical continuity with past analyses and serves as one point of reference in interpreting the results of the expert elicitation (see discussion below).

The RIAs for the CAIR and Clean Air Nonroad Diesel rules included an estimate of mortality impacts based on application of the C-R function derived from the Harvard Six-cities study. In those analyses, the Six-cities estimate was included as a sensitivity analysis in an appendix to the RIA. Following the NAS advice to begin moving sensitivity and uncertainty analyses into the main body of the RIA, we are including a separate estimate based on the Six-cities study to complement the estimate based on the ACS study. This also reflects 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.

As noted above, since the most recent SAB review, an extended follow-up of the Harvard Six-cities study has been published (Laden et al., 2006). We use this specific estimate to represent the Six-cities study because it reflects the most up-to-date science and because it was cited by many of the experts in their elicitation responses. We note that because of the recent publication date of the Laden et al (2006) study, it has not undergone the CASAC and SAB-HES review received by the Pope et al (2002) and earlier Six-cities publications (see Dockery et al, 1993). However, it is clear from the expert elicitation that the results published in Laden et al (2006) are potentially influential, and in fact, the expert elicitation results encompass within their range the estimates from both the Pope et al (2002) and Laden et al (2006) studies. As part of the NAAQS review process, EPA conducted a provisional assessment of "new" science published since the closing date for the PM Criteria Document. The provisional assessment found that "new" studies generally strengthen the evidence that acute and chronic exposures to fine particles are associated with health effects. The provisional assessment found that the results reported in the studies do not dramatically diverge from previous findings, and, taken in context with the findings of the Criteria Document, the new information and findings do not materially change any of the broad scientific conclusions regarding the health effects of PM exposure made in the Criteria Document. The Laden et al (2006) study was included in this provisional assessment and therefore can be considered to be covered under the broad findings of the provisional assessment.

A number of additional analyses have been conducted on the ACS cohort data (Jarrett et al., 2005; Krewski et al., 2005; Pope et al., 2004). These studies have continued to find a strong significant relationship between $PM_{2.5}$ and mortality outcomes. Specifically, much of the recent research has suggested a stronger relationship between cardiovascular mortality and lung cancer mortality with $PM_{2.5}$, and a less significant relationship between respiratory-related mortality and $PM_{2.5}$.

EPA's is committed to seeking the advice of its Science Advisory Board to review 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, and if so, the appropriateness of using Laden et al 2006, the most recently published update, as the estimate for the Six Cities based model.

$PM_{2.5}$-Related Adult Premature Mortality – Expert Elicitation Study

Among the recommendations made by the National Research Council (NRC) in its 2002 review of EPA's method for assessing health benefits of air pollution regulations was a recommendation for EPA to consider the use of formally elicited expert judgments as a means of characterizing uncertainty in inputs to health benefits analyses. As part of its efforts to improve the characterization of uncertainties in its benefits estimates, EPA has conducted a study of the concentration-response (C-R) relationship between changes in $PM_{2.5}$ exposures and mortality using formally elicited expert judgments. The goal of the study was to elicit from a sample of health experts probabilistic distributions describing uncertainty in estimates of the reduction in mortality among the adult U.S. population resulting from reductions in ambient annual average

$PM_{2.5}$ levels. These distributions were obtained through a formal interview protocol using methods designed to elicit subjective expert judgments.

In 2003 and 2004, EPA conducted a pilot-scale elicitation study with five experts to explore the effectiveness of expert judgment techniques for characterizing uncertainty and to explore the use of the expert judgment results in the context of economic benefits analysis (Industrial Economics, 2004). EPA previously applied the results of the pilot-scale study as part of its uncertainty analysis in the regulatory analysis accompanying the Clean Air Interstate Rule (CAIR) (U.S. EPA, 2005). EPA has recently completed a full-scale expert elicitation analysis of the PM2.5-mortality relationship that included numerous refinements based on insights from conducting the pilot study and on comments from peer reviewers of the pilot (Industrial Economics, 2006). This analysis applies the results of the full-scale study.

The full-scale study involved personal interviews with twelve health experts who have conducted research on the relationship between $PM_{2.5}$ exposures and mortality. These experts were selected through a peer-nomination process and included experts in epidemiology, toxicology, and medicine. The elicitation interview consisted of a protocol of carefully structured questions, both qualitative and quantitative, about the nature of the $PM_{2.5}$-mortality relationship.[P] The questions requiring qualitative responses probed experts' beliefs concerning key evidence and critical sources of uncertainty and enabled them to establish a conceptual basis supporting their quantitative judgments. Questions covered topics such as potential biological mechanisms linking $PM_{2.5}$ exposures with mortality; the role of study design in capturing PM/mortality effects; key scientific evidence on the magnitude of the PM/mortality relationship; sources of potential error or bias in epidemiological results; the likelihood of a causal relationship between $PM_{2.5}$ and mortality, and the shape of the C-R function. The main quantitative question in the protocol asked experts to provide the 5th, 25th, 50th, 75th, and 95th percentiles of a probabilistic distribution for the percent change in U.S. annual, adult all-cause mortality resulting from a 1 $\mu g/m^3$ change in annual average $PM_{2.5}$ exposure, assuming a range of baseline PM2.5 levels between 4 and 30 $\mu g/m^3$. This quantitative question was designed to yield results appropriate for application in EPA's quantitative health benefit analyses.

[P] In addition to the elicitation interviews, the twelve experts participated in pre- and post-elicitation workshops. The pre-elicitation workshop was designed to prepare the experts by familiarizing them with the protocol, providing them information about probabilistic judgments, and allowing them to discuss key issues and relevant evidence. At this workshop, the experts were also provided with "briefing book" materials, including a CD containing relevant studies and background information pages with data on air quality in the US, population demographics, health status, summaries of published effect estimates, and data on other factors potentially useful to experts in developing their judgments (air conditioning use, housing stock, PM composition, educational attainment). The post-elicitation workshop was designed to anonymously share and discuss results of the expert interviews; discuss key areas where expert opinion varied; and clarify any questions that may have arisen during the interviews. Experts were given the opportunity to revise their judgments in response to discussions at this workshop; however, experts were not encouraged to reach a consensus opinion.

The results of the full-scale study consist of twelve individual distributions for the coefficient or slope of the C-R function relating changes in annual average $PM_{2.5}$ exposures to annual, adult all-cause mortality. The results have not been combined in order to preserve the breadth and diversity of opinion on the expert panel. In applying these results in a benefits analysis context, EPA incorporates information about each expert's judgments concerning the shape of the C-R function (including the potential for a population threshold $PM_{2.5}$ concentration below which there is no effect on mortality), the distribution of the slope of the C-R function, and the likelihood that the $PM_{2.5}$-mortality relationship is or is not causal (unless the expert incorporated this last element directly in his slope distribution - see Industrial Economics, 2006).

Consistent with the approach used in the RIA for the recent PM NAAQS, we constructed a corresponding set of 12 health impact functions for premature mortality based on the responses of the 12 experts (designated A through L). For those experts providing log-linear non-threshold functions, construction of a health impact function was straightforward, and directly matched the construction of health impact functions based on the epidemiology literature.[Q] In these cases, the expert's function can be translated into a health impact function of the form:

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

Where y_0 is the baseline incidence, equal to the baseline incidence rate time the potentially affected population, β is the effect estimate provided by the expert, and ΔPM is the change in $PM_{2.5}$.

Some experts specified a piecewise log-linear function, in which case we developed health impact functions that incorporate ambient concentration levels. For example, Expert B specified a piecewise function with two segments, representing the concentration-response function for ambient concentrations between 4 and 10 $\mu g/m^3$ and between 10 and 30 $\mu g/m^3$. In this case, the expert's function can be translated into a health impact function of the form:

$$\Delta y = \begin{cases} y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) if\ Q_0 < 10 \\ y_{02} \cdot \left(e^{\beta_2 \cdot \Delta PM} - 1\right) if\ Q_0 \geq 10 \end{cases},$$

Where Q_0 is the baseline concentration of $PM_{2.5}$, y_{01} is the baseline incidence for populations living in areas with baseline concentrations of $PM_{2.5}$ less than 10 $\mu g/m^3$, y_{02} is the baseline incidence for populations living in areas with baseline concentrations of $PM_{2.5}$ greater than or equal to10 $\mu g/m^3$, and β_1 and β_2 are the effect estimates corresponding to the segments of the C-R function relating to ambient concentrations between 4 and 10 $\mu g/m^3$ and 10 and 30 $\mu g/m^3$, respectively.

[Q] Note that in the expert elicitation protocol, we specified the relevant range of exposure as between 4 and 30 $\mu g/m^3$. As such, when applying the expert elicitation based functions, benefits are only estimated for starting concentrations greater than 4 $\mu g/m^3$.

A third form specified by one expert (Expert K) included both a piecewise log-linear function and a probabilistic threshold. Expert K did not provide a full set of information about the shape of the distribution of the threshold, providing only the probability that a threshold existed between 0 and 5 μg/m^3 (equal to 0.4) and the probability that a threshold existed between 5 and 10 μg/m^3 (equal to 0.1). The probability that a threshold above 10 existed was set to zero, and the probability that there was no threshold was specified as 0.50. We assumed that the probability distribution across the range 0 to 5 was uniform, such that the probability of a threshold between 0 and 1, 1 and 2, etc. was equal. Likewise, we assumed that the probability distribution across the range 5 to 10 was uniform. Expert K also provided a two segment piecewise log-linear function, with the segments defined over the ranges 4 to 16 μg/m^3, and 16 to 30 μg/m^3. Using this information, we translated Expert K's responses into the following three conditional health impact functions:

$$\text{(K1)} \quad \Delta y = \begin{cases} y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) if\ Q_0 < 16 \\ y_{02} \cdot \left(e^{\beta_2 \cdot \Delta PM} - 1\right) if\ Q_0 \geq 16 \end{cases}$$

$$\text{(K2)} \quad \Delta y = \begin{cases} y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.0\, if\ 0 \leq Q_0 < 1 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.2\ if\ 1 \leq Q_0 < 2 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.4\ if\ 2 \leq Q_0 < 3 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.6\ if\ 3 \leq Q_0 < 4 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.8 if\ 4 \leq Q_0 < 5 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) if\ 5 \leq Q_0 < 16 \\ y_{02} \cdot \left(e^{\beta_2 \cdot \Delta PM} - 1\right) if\ Q_0 \geq 16 \end{cases}$$

$$\text{(K3)} \quad \Delta y = \begin{cases} y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.0\, if\ 0 \leq Q_0 < 6 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.2\ if\ 6 \leq Q_0 < 7 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.4\ if\ 7 \leq Q_0 < 8 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.6\ if\ 8 \leq Q_0 < 9 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 0.8 if\ 9 \leq Q_0 < 10 \\ y_{01} \cdot \left(e^{\beta_1 \cdot \Delta PM} - 1\right) \times 1.0 if\ 10 \leq Q_0 < 16 \\ y_{02} \cdot \left(e^{\beta_2 \cdot \Delta PM} - 1\right) if\ Q_0 \geq 16 \end{cases}$$

Function K1 is associated with a no threshold segmented log-linear specification with a knot at 16 μg/m^3. Function K2 represents the segmented log-linear function with a threshold between 0 and 5 μg/m^3, with the cumulative probability of a threshold at or below the initial concentration Q_0 increasing as Q_0 decreases (this will result in a declining expected value of the impact at lower initial concentrations). Likewise, function K3 represented the segmented log-linear function with a threshold between 5 and 10 μg/m^3. The results of applying the three conditional functions are then combined using Monte Carlo analysis with weights of 0.5, 0.4, and 0.1 assigned to conditional functions K1, K2, and K3, respectively.

In addition to specifying a function form, each expert provided a representation of the distribution (or distributions for those who specified piecewise functions) of the effect size (in terms of the percent change in premature mortality associated with a one microgram change in annual mean $PM_{2.5}$). Six of the experts simply chose a normal distribution, which is completely specified with two parameters, the mean and standard deviation (see Figure 6.3-2 for example). In one case, the expert specified a triangular distribution, which is represented by a minimum, maximum, and most likely value (see Figure 6.3-3). In another case, the expert specified a Weibull distribution, which has three parameters representing scale, location, and shape (see Figure 6.3-4). Four of the experts did not choose a parametric distribution, preferring instead to provide only effect estimates at particular percentiles of their distributions. In these cases, we constructed custom distributions to represent their percentiles. For these custom distributions, we assume a continuous and smooth transition of the distribution between the reported percentiles (see Figure 6.3-5 for example).

Figure 6.3-2. Example Normal Distribution for Expert A

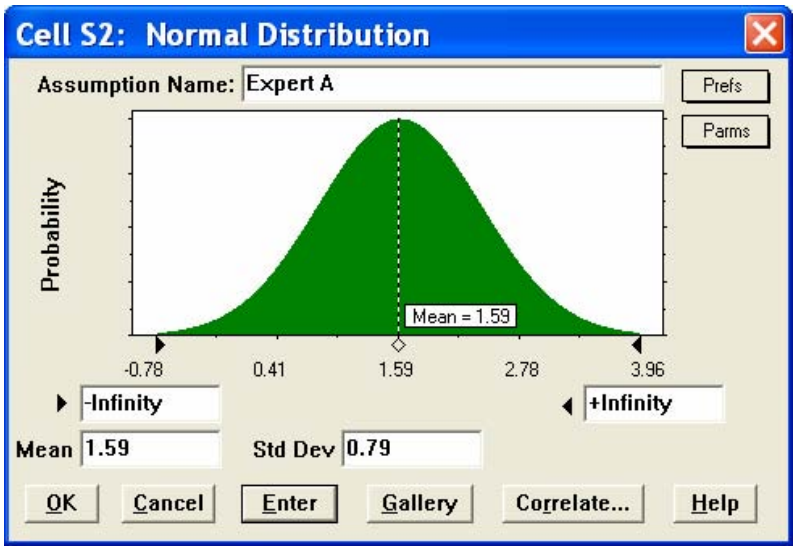


Figure 6.3-3. Example Triangular Distribution for Expert D

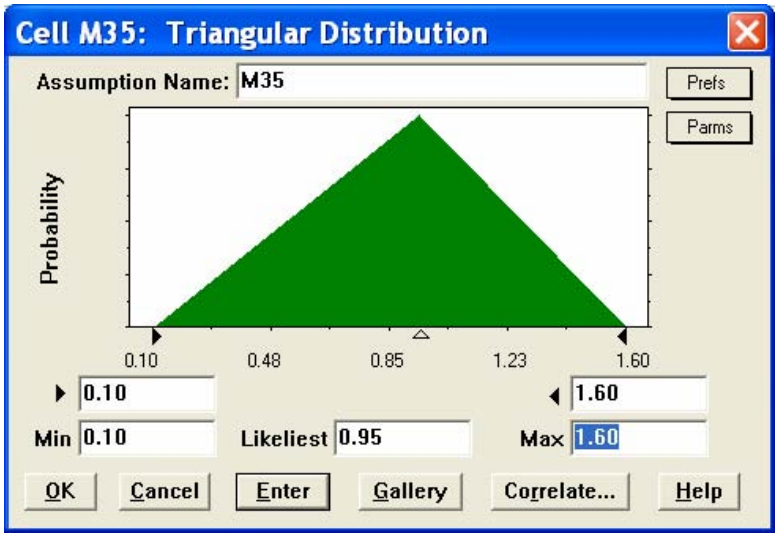


Figure 6.3-4. Example Weibull Distribution for Expert J

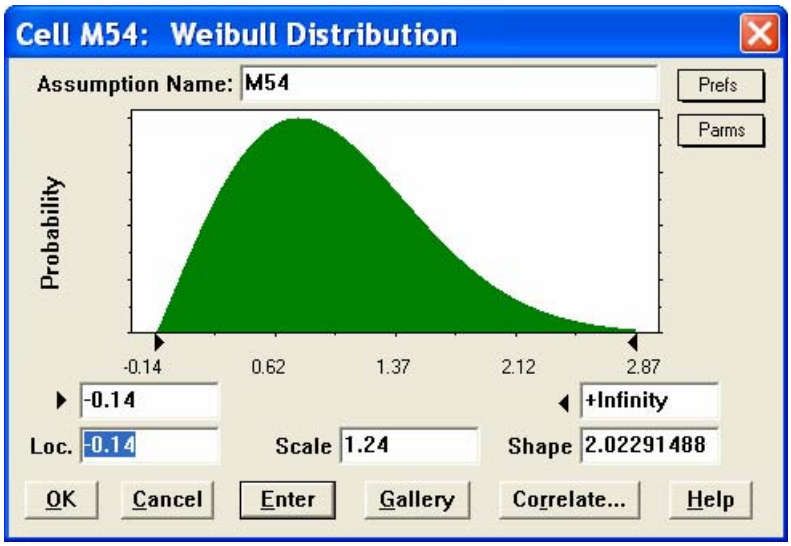
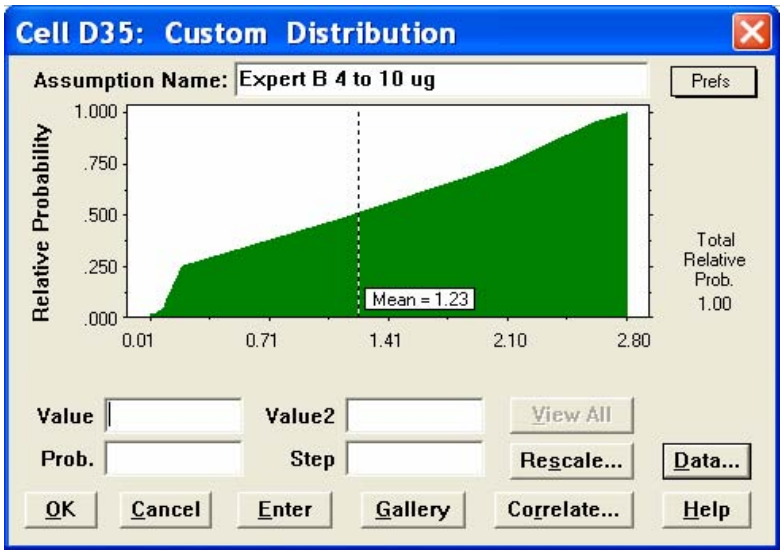
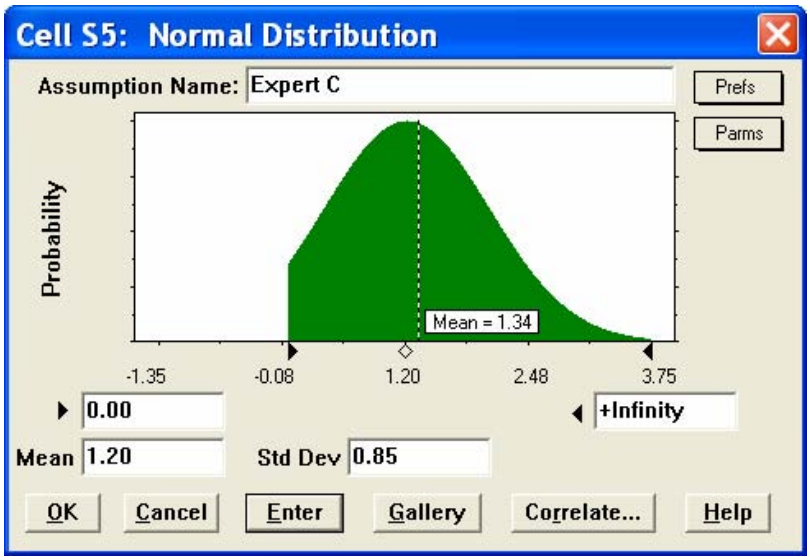


Figure 6.3-5. Example Custom Distribution for Expert B



In one special case, Expert E provided a normal distribution that implied a negative tail at the 2.5th percentile (the lower bound of a typical 95 percent confidence interval), but also specified a minimum value at zero. In this case, we treated the distribution as a truncated normal. In the case, the mean of the resulting incidence distribution will be shifted upwards relative to a full normal, to adjust for the mass of the distribution that would have been below zero (see Figure 6.3-6). Note that in the figure, the mean of the normal distribution specified by Expert C is 1.2, while the mean of the implied truncated normal will be 1.34.

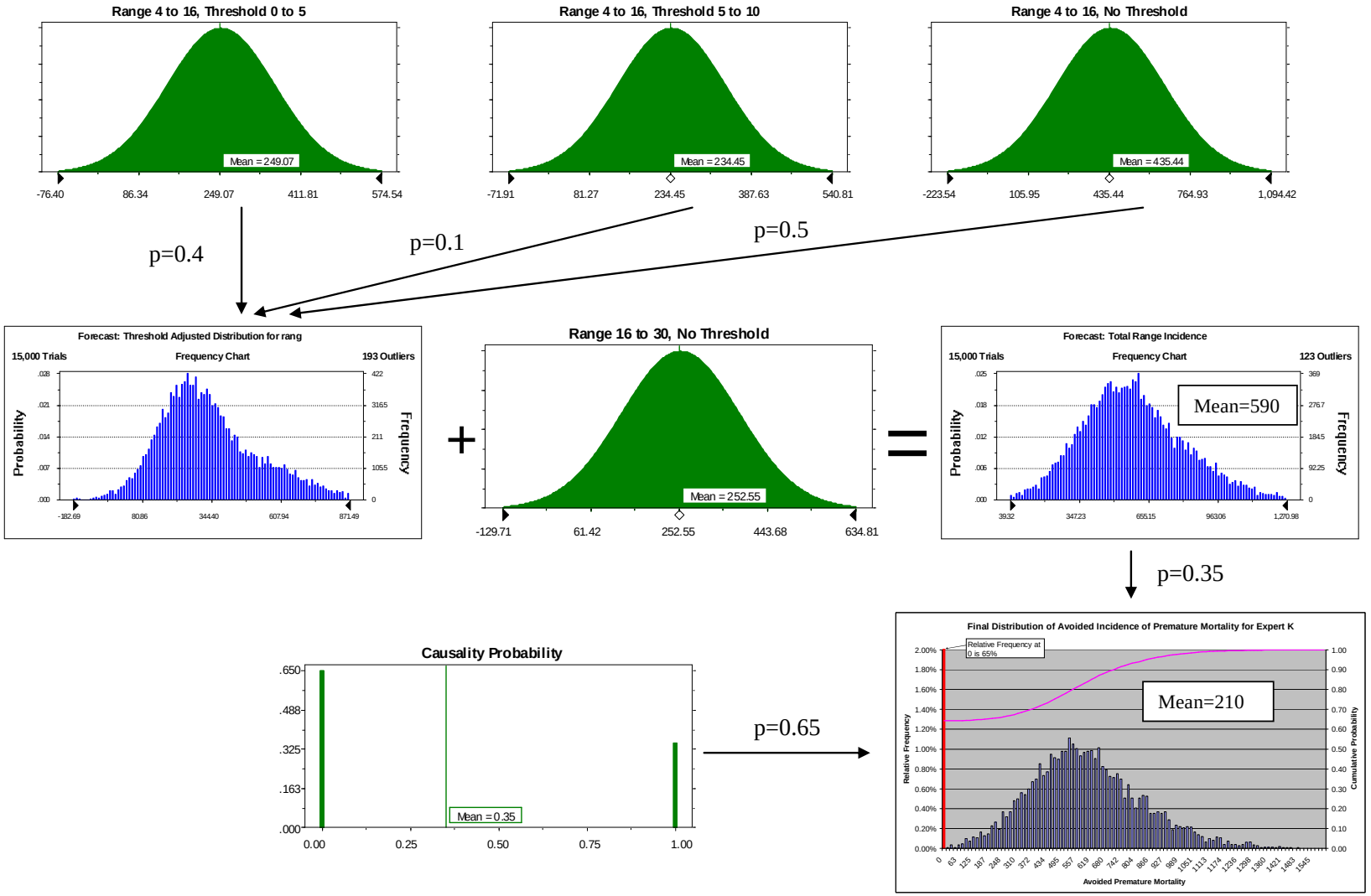
Figure 6.3-6. Truncated Normal Distribution for Expert C



In some cases, experts included in their reported distributions the likelihood that the relationship between $PM_{2.5}$ and mortality was not causal, e.g., that reducing $PM_{2.5}$ would not actually reduce the risk of premature death. In these cases, the distributions are unconditional, and included zero with some probability to reflect views on less than certain causality. In most cases, the experts chose to specify a conditional distribution, such that the distribution of the effect estimate is conditional on there being a causal relationship. In these cases, the final estimated distribution of avoided incidence of premature mortality will be the expected value of the unconditional distribution. In practice, we implement this by estimating each expert's conditional distribution and then, using Monte Carlo sampling, construct an unconditional distribution using the expert's reported probability of a causal relationship. To illustrate how these various components of an expert's results are combined to produce an estimate of the distribution of reduced mortality associated with a reduction in ambient $PM_{2.5}$, we provide an example calculation for Expert K. This example calculation is graphically displayed in Figure 6.3-7. In Figure 6.3-7, the initial application of Expert K's conditional concentration-response functions provides 3 distributions associated with reductions in $PM_{2.5}$ concentrations in the range of starting concentrations from 4 to 16 $\mu g/m^3$. These distributions are assigned weights based on the expert's judgments about the likelihood of a threshold existing in the ranges 0 to 5, 5 to 10, or not at all. These weights are used to develop a new distribution for the change in mortality for starting concentrations between 4 and 16. These are then added to the distribution of the change in mortality associated with reductions in $PM_{2.5}$ in the range of starting concentrations from 16 to 30 $\mu g/m^3$. This gives an overall distribution of reductions in mortality for the full range of starting concentrations, conditional on the existence of a causal relationship. This conditional distribution is then combined with the expert's judgment about causality (35 percent likelihood of a causal relationship), to derive the unconditional distribution of changes in mortality, which, as can be seen in the figure, is composed of a mass of probability at zero (reflecting the likelihood of no causal relationship), and a probability density function (PDF) over the remaining 35 percent of probability characterized by the conditional distribution. As expected, the unconditional

Figure 6.3-7. Example Calculations Expert K

Initial Distributions:



distribution has a mean change in mortality that is 35 percent of the mean of the conditional distribution.

$PM_{2.5}$-Related Infant Mortality. Recently published studies have strengthened the case for an association between PM exposure and respiratory inflammation and infection leading to premature mortality in children under 5 years of age. Specifically, the SAB-HES noted the release of the WHO Global Burden of Disease Study focusing on ambient air, which cites several recently published time-series studies relating daily PM exposure to mortality in children (U.S. EPA-SAB, 2004b). The SAB-HES also cites the study by Belanger et al. (2003) as corroborating findings linking PM exposure to increased respiratory inflammation and infections in children. Recently, a study by Chay and Greenstone (2003) found that reductions in TSP caused by the recession of 1981–1982 were related to reductions in infant mortality at the county level. With regard to the cohort study conducted by Woodruff et al. (1997), the SAB-HES notes several strengths of the study, including the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control for a variety of individual risk factors in infants (e.g., maternal educational level, maternal ethnicity, parental marital status, and maternal smoking status). Based on these findings, the SAB-HES recommends that EPA incorporate infant mortality into the primary benefits estimate and that infant mortality be evaluated using an impact function developed from the Woodruff et al. (1997) study (U.S. EPA-SAB, 2004b). A more recent study by Woodruff et al. (2006) continues to find associations between $PM_{2.5}$ and infant mortality. The study also found the most significant relationships with respiratory-related causes of death. We have not yet sought comment from the SAB on this more recent study and as such continue to rely on the earlier 1997 analysis.

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% of the U.S. population (American Lung Association, 1999). A limited number of studies have estimated the impact of air pollution on new incidences of 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. Because the proposed standards are expected to reduce primarily $PM_{2.5}$, this analysis uses only the Abbey et al. (1995) study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of CB.

Nonfatal Myocardial Infarctions (heart attacks). Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We 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. A more recent study by Zanobetti and Schwartz (2005) used a similar method to Peters et al. (2001), but focused on adults 65 and older, and used PM10 as the PM indicator. They found a significant relationship between nonfatal heart attacks and PM10, although the magnitude of the effect was much lower than Peters et al. This may reflect the use of PM10, the more limited age range, or the less precise diagnosis of heart attack used in defining the outcome measure. Other studies, such as Domenici et al. (2006), Samet et al. (2000), and Moolgavkar (2000), show a consistent relationship between all cardiovascular hospital admissions, including those for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on long-term health costs and earnings, we provide a separate estimate for nonfatal heart attacks. The estimate used in the analysis of the proposed standards is based on the single available U.S. $PM_{2.5}$ effect estimate

from Peters et al. (2001). 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. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other coronary heart diseases (Carthenon et al., 2002; Dekker et al., 2000; Liao et al., 1997; Tsuji et al., 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

Hospital and Emergency Room Admissions. Because of the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, many of the hospital admission endpoints use pooled impact functions based on the results of a number of studies. In addition, some studies have examined the relationship between air pollution and emergency room visits. Since most emergency room visits do not result in an admission to the hospital (the majority of people going to the emergency room are treated and return home), we treat hospital admissions and emergency room visits separately, taking account of the fraction of emergency room visits that are admitted to the hospital.

The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking PM with other types of hospital admissions. The only type of emergency room visits that have been consistently linked to PM in the United States are asthma-related visits.

To estimate avoided incidences of $PM_{2.5}$ related cardiovascular hospital admissions in populations aged 65 and older, we use effect estimates from studies by Moolgavkar (2003) and Ito (2003). However, only Moolgavkar (2000) provided a separate effect estimate for populations 20 to 64.[R] Total cardiovascular hospital admissions are thus the sum of the pooled estimates from Moolgavkar (2003) and Ito (2003) for populations over 65 and the Moolgavkar (2000) based impacts for populations aged 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. To avoid double-counting benefits from reductions in myocardial infarctions when applying the impact function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarctions.

To estimate total avoided incidences of respiratory hospital admissions, we used impact functions for several respiratory causes, including chronic obstructive pulmonary disease

[R] Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we chose to use the existing study. Updates have been provided for the 65 and older population, and showed little difference. Given the very small (<5%) difference in the effect estimates for people 65 and older with cardiovascular hospital admissions between the original and reanalyzed results, we do not expect the difference in the effect estimates for the 20 to 64 population to differ significantly. As such, the choice to use the earlier, uncorrected analysis will likely not introduce much bias.

(COPD), pneumonia, and asthma. As with cardiovascular admissions, additional published studies show a statistically significant relationship between PM_{10} and respiratory hospital admissions. We used only those focusing on $PM_{2.5}$. Both Moolgavkar (2000) and Ito (2003) provide effect estimates for COPD in populations over 65, allowing us to pool the impact functions for this group. Only Moolgavkar (2000) provides a separate effect estimate for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Ito (2003) estimated pneumonia and only for the population 65 and older. In addition, Sheppard (2003) provided an effect estimate for asthma hospital admissions for populations under age 65. Total avoided incidence of PM-related respiratory-related hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the effect estimate from a study of children 18 and under by Norris et al. (1999). As noted earlier, there is another study by Schwartz examining a broader age group (less than 65), but the Schwartz study focused on PM_{10} rather than $PM_{2.5}$. We selected the Norris et al. (1999) effect estimate because it better matched the pollutant of interest. Because children tend to have higher rates of hospitalization for asthma relative to adults under 65, we will likely capture the majority of the impact of $PM_{2.5}$ on asthma emergency room visits in populations under 65, although there may still be significant impacts in the adult population under 65.

Acute Health Events and Work Loss Days. As indicated in Table 6.1-2, in addition to mortality, chronic illness, and hospital admissions, a number of acute health effects not requiring hospitalization are associated with exposure to ambient levels of PM. The sources for the effect estimates used to quantify these effects are described below.

Around 4 percent of U.S. children between the ages of 5 and 17 experience episodes of acute bronchitis annually (American Lung Association, 2002c). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia,[S] with the exception of cough, most acute bronchitis symptoms abate within 7 to 10 days. Incidence of episodes of acute bronchitis in children between the ages of 5 and 17 were estimated using an effect estimate developed from Dockery et al. (1996).

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

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (e.g., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms has thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged 9 to 11 are estimated using an effect estimate developed from Pope et al. (1991).

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

Health effects from air pollution can also result in missed days of work (either from personal symptoms or from caring for a sick family member). Days of work lost due to $PM_{2.5}$ were estimated using an effect estimate developed from Ostro (1987).

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

In analyzing the proposed standards, we have followed the SAB-HES recommendations regarding asthma exacerbations in developing the primary estimate. To prevent double-counting, we focused the estimation on asthma exacerbations occurring in children and excluded adults from the calculation.[T] Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, we would likely double-count incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double-counting.

To characterize asthma exacerbations in children, we selected two studies (Ostro et al., 2001; Vedal et al., 1998) that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between $PM_{2.5}$, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this

[T] Estimating asthma exacerbations associated with air pollution exposures is difficult, due to concerns about double-counting of benefits. Concerns over double-counting stem from the fact that studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double-counting. In one specific case (upper respiratory symptoms in children), the only study available is limited to asthmatic children, so this endpoint can be readily included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms and MRADs, are estimated for the total population that includes asthmatics. Therefore, to simply add predictions of asthma-related symptoms generated for the population of asthmatics to these total population-based estimates could result in double-counting, especially if they evaluate similar endpoints. The SAB-HES, in commenting on the analytical blueprint for 812, acknowledged these challenges in evaluating asthmatic symptoms and appropriately adding them into the primary analysis (U.S. EPA-SAB, 2004b). However, despite these challenges, the SAB-HES recommends the addition of asthma-related symptoms (i.e., asthma exacerbations) to the primary analysis, provided that the studies use the panel study approach and that they have comparable design and baseline frequencies in both asthma prevalence and exacerbation rates. Note also, that the SAB-HES, while supporting the incorporation of asthma exacerbation estimates, does not believe that the association between ambient air pollution, including ozone and PM, and the new onset of asthma is sufficiently strong to support inclusion of this asthma-related endpoint in the primary estimate.

endpoint, along with shortness of breath and wheeze, in generating incidence estimates (see below). Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM_{10} and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations. We employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al. study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled with the cough-related estimate generated using the Vedal study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

Additional epidemiological studies are available for characterizing asthma-related health endpoints (the full list of epidemiological studies considered for modeling asthma-related incidence is presented in Table 6.3-6). However, based on recommendations from the SAB-HES, we decided not to use these additional studies in generating the primary estimate. In particular, the Yu et al. (2000) estimates show a much higher baseline incidence rate than other studies, which may lead to an overstatement of the expected impacts in the overall asthmatic population. The Whittemore and Korn (1980) study did not use a well-defined endpoint, instead focusing on a respondent-defined "asthma attack." Other studies looked at respiratory symptoms in asthmatics but did not focus on specific exacerbations of asthma.

6.3.5.4 Treatment of Potential Thresholds in Health Impact Functions

Unless specifically noted, our premature mortality benefits estimates are based on an assumed cutpoint in the premature mortality concentration-response function at 10 $\mu g/m^3$, and an assumed cutpoint of 10 $\mu g/m^3$ for the concentration-response functions for morbidity associated with short term exposure to PM2.5. The 10 $\mu g/m^3$ threshold reflects comments from CASAC (U.S. EPA Science Advisory Board, 2005). To consider the impact of a threshold in the response function for the chronic mortality endpoint on the primary benefits estimates, we also 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 adjusted the mortality function slopes accordingly.[U] This sensitivity analysis allows us to determine the change (reduction) in avoided mortality cases and associated monetary benefits associated with

[U] Note, that the adjustment to the mortality slopes was only done 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 are at or below the lowest measured exposure levels reported in the ACS cohort study, for the combined exposure dataset. See Appendix H for a complete discussion of the slope adjustment procedure.

alternative cutpoints. Five cutpoints (including the base case assumption) were included in this sensitivity analysis: (a) 14 $\mu g/m^3$ (assumes no impacts below the alternative annual NAAQS), (b) 12 $\mu g/m^3$ (c) 10 $\mu g/m^3$ (reflects comments from CASAC - 2005), (d) 7.5 $\mu g/m^3$ (reflects recommendations from SAB-HES to consider estimating mortality benefits down to the lowest exposure levels considered in the ACS cohort study used as the basis for modeling chronic mortality) and (e) background or 3 $\mu g/m^3$ (reflects NRC recommendation to consider effects all the way to background).

Table 6.3-6. Studies Examining Health Impacts in the Asthmatic Population Evaluated for Use in the Benefits Analysis

Endpoint	Definition	Pollutant	Study	Study Population
Asthma Attack Indicators				
Shortness of breath	Prevalence of shortness of breath; incidence of shortness of breath	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8–13
Cough	Prevalence of cough; incidence of cough	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8–13
Wheeze	Prevalence of wheeze; incidence of wheeze	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8–13
Asthma exacerbation	>= 1 mild asthma symptom: wheeze, cough, chest tightness, shortness of breath	PM_{10}, $PM_{1.0}$	Yu et al. (2000)	Asthmatics, 5–13
Cough	Prevalence of cough	PM_{10}	Vedal et al. (1998)	Asthmatics, 6–13
Other Symptoms/Illness Endpoints				
Upper respiratory symptoms	>= 1 of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes	PM_{10}	Pope et al. (1991)	Asthmatics, 9–11
Moderate or worse asthma	Probability of moderate (or worse) rating of overall asthma status	$PM_{2.5}$	Ostro et al. (1991)	Asthmatics, all ages
Acute bronchitis	>= 1 episodes of bronchitis in the past 12 months	$PM_{2.5}$	McConnell et al. (1999)	Asthmatics, 9–15
Phlegm	"Other than with colds, does this child usually seem congested in the chest or bring up phlegm?"	$PM_{2.5}$	McConnell et al. (1999)	Asthmatics, 9–15
Asthma attacks	Respondent-defined asthma attack	$PM_{2.5}$	Whittemore and Korn (1980)	Asthmatics, all ages

6.3.5.5 Baseline Health Effect Incidence Rates

The epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the relative risk of a health effect, rather than an estimate of the absolute number of avoided cases. For example, a typical result might be that a 10 $\mu g/m^3$ decrease in daily $PM_{2.5}$ levels might decrease hospital admissions by 3%. To then to convert this relative change into a number of cases, the baseline incidence of the health effect is necessary. The baseline incidence rate provides an estimate of the incidence rate (number of cases of the health effect per year, usually

per 10,000 or 100,000 general population) in the assessment location corresponding to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number (e.g., if the baseline incidence rate is number of cases per year per 100,000 population, it must be multiplied by the number of 100,000s in the population).

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

In most cases, because of a lack of data or methods, we have not attempted to project incidence rates to future years, instead assuming that the most recent data on incidence rates is the best prediction of future incidence rates. In recent years, better data on trends in incidence and prevalence rates for some endpoints, such as asthma, have become available. We are working to develop methods to use these data to project future incidence rates. However, for our primary benefits analysis, we continue to use current incidence rates. The one exception is in the case of premature mortality. In this case, we have projected mortality rates such that future mortality rates are consistent with our projections of population growth (Abt Associates, 2005). Compared with previous analyses, this will result in a reduction in the mortality related impacts of air pollution in future years.

Table 6.3-7 summarizes the baseline incidence data and sources used in the benefits analysis. We use the most geographically disaggregated data available. For premature mortality, county-level data are available. For hospital admissions, regional rates are available. However, for all other endpoints, a single national incidence rate is used, due to a lack of more spatially disaggregated data. In these cases, we used national incidence rates whenever possible, because these data are most applicable to a national assessment of benefits. However, for some studies, the only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level.

Table 6.3-7: Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population

Endpoint	Parameter	Rates	
		Value	Source[a]
Mortality	Daily or annual mortality rate	Age-, cause-, and county-specific rate	CDC Wonder (1996–1998)
Hospitalizations	Daily hospitalization rate	Age-, region-, and cause-specific rate	1999 NHDS public use data files[b]
Asthma ER Visits	Daily asthma ER visit rate	Age- and region-specific visit rate	2000 NHAMCS public use data files[c]; 1999 NHDS public use data files[b]
Chronic Bronchitis	Annual prevalence rate per person - Aged 18–44 - Aged 45–64 - Aged 65 and older	 0.0367 0.0505 0.0587	1999 NHIS (American Lung Association, 2002b, Table 4)
	Annual incidence rate per person	0.00378	Abbey et al. (1993, Table 3)
Nonfatal Myocardial Infarction (heart attacks)	Daily nonfatal myocardial infarction incidence rate per person, 18+ - Northeast - Midwest - South - West	 0.0000159 0.0000135 0.0000111 0.0000100	1999 NHDS public use data files[b]; adjusted by 0.93 for probability of surviving after 28 days (Rosamond et al., 1999)
Asthma Exacerbations	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)	Ostro et al. (2001)
	Prevalence among asthmatic children - daily wheeze - daily cough - daily dyspnea	 0.038 0.086 0.045	Vedal et al. (1998)
Acute Bronchitis	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002c, Table 11)
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children[d]	0.0012	Schwartz et al. (1994, Table 2)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children	0.3419	Pope et al. (1991, Table 2)
Work Loss Days	Daily WLD incidence rate per person (18–65) - Aged 18–24 - Aged 25–44 - Aged 45–64	 0.00540 0.00678 0.00492	1996 HIS (Adams, Hendershot, and Marano, 1999, Table 41); U.S. Bureau of the Census (2000)
Minor Restricted-Activity Days	Daily MRAD incidence rate per person	0.02137	Ostro and Rothschild (1989, p. 243)

[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] Lower respiratory symptoms are defined as two or more of the following: cough, chest pain, phlegm, and wheeze.

Baseline age, cause, and county-specific mortality rates were obtained from the U.S. Centers for Disease Control and Prevention (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.

To estimate age- and county-specific mortality rates in years 2000 through 2020, we calculated adjustment factors, based on a series of Census Bureau projected national mortality rates, to adjust the CDC Wonder age- and county-specific mortality rates in 1996-1998 to corresponding rates for each future year. For the analysis year 2020, these adjustment factors ranged across age categories from 0.76 to 0.86

For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Table 6.3-7 lists the baseline incidence rates and their sources for asthma symptom endpoints. Table 6.3-8 lists the prevalence rates used to determine the applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years. As noted above, we are investigating methods for projecting asthma prevalence rates in future years. However, it should be noted that current trends in asthma prevalence do not lead us to expect that asthma prevalence rates will be more than 4% overall in 2020, or that large changes will occur in asthma prevalence rates for individual age categories (Mansfield et al., 2005).

Table 6.3-8 Asthma Prevalence Rates Used to Estimate Asthmatic Populations in Impact Functions

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

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

6.3.5.6 Selecting Unit Values for Monetizing Health Endpoints

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

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These COI estimates generally (although not in every case) understate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect. Table 6.3-9 summarizes the value estimates per health effect that we used in this analysis. Values are presented both for a 1990 base income level and adjusted for income growth out to 2020 and 2030. Note that the unit values for hospital admissions are the weighted averages of the ICD-9 code-specific values for the group of ICD-9 codes included in the hospital admission categories. A discussion of the valuation methods for premature mortality and CB is provided here because of the relative importance of these effects. Discussions of the methods used to value nonfatal myocardial infarctions (heart attacks) and school absence days are provided because these endpoints have only recently been added to the analysis and the valuation methods are still under development. In the following discussions, unit values are presented at 1990 levels of income for consistency with previous analyses. Equivalent future-year values can be obtained from Table 6.3-9. COI estimates are converted to constant 1999 dollar equivalents using the medical CPI.

Valuing Reductions in Premature Mortality Risk. Following the advice of the EEAC of the SAB, EPA currently uses the VSL approach when calculating mortality benefits, because we believe this calculation provides the most reasonable single estimate of an individual's willingness to trade off money for reductions in mortality risk (EPA, 2000a). The VSL approach is a summary measure for the value of small changes in mortality risk experienced by a large number of people. The mean value of avoiding one statistical death is assumed to be $5.5 million in 1999 dollars. This represents a central value consistent with the range of values suggested by recent meta-analyses of the wage-risk VSL literature. The distribution of VSL is characterized by a confidence interval from $1 to $10 million, based on two meta-analyses of the

wage-risk VSL literature. The $1 million lower confidence limit represents the lower end of the interquartile range from the Mrozek and Taylor (2002) meta-analysis. The $10 million upper confidence limit represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The mean estimate of $5.5 million is consistent with the mean VSL of $5.4 million estimated in the Kochi et al. (2006) meta-analysis. Because the majority of the studies in these meta-analyses are based on datasets from the early 1990s or previous decades, we continue to assume that the VSL estimates provided by those meta-analyses are in 1990 income equivalents. Future research might provide income-adjusted VSL values for individual studies that can be incorporated into the meta-analyses. This would allow for a more reliable base-year estimate for use in adjusting VSL for aggregate changes in income over time.

As indicated in the previous section on quantification of premature mortality benefits, we assumed for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the 20 years following exposure. To take this into account in the valuation of reductions in premature mortality, we applied an annual 3% discount rate to the value of premature mortality occurring in future years.[V]

[V] The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the federal government. EPA adopted a 3% discount rate for its base estimate in this case to reflect reliance on a "social rate of time preference" discounting concept. We have also calculated benefits and costs using a 7% rate consistent with an "opportunity cost of capital" concept to reflect the time value of resources directed to meet regulatory requirements. In this case, the benefit and cost estimates were not significantly affected by the choice of discount rate. Further discussion of this topic appears in EPA's *Guidelines for Preparing Economic Analyses* (EPA, 2000b).

Table 6.3-9. Unit Values Used for Economic Valuation of Health Endpoints (2000$)[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): $PM_{2.5}$-related	$5,500,000	$6,600,000	$6,800,000	Point estimate is the mean of a normal distribution with a 95 percent confidence interval between $1 and $10 million. Confidence interval is based on two meta-analyses of the wage-risk VSL literature: $1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2002)[27] meta-analysis and $10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003)[28] meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population.
Chronic Bronchitis (CB)	$340,000	$420,000	$430,000	Point estimate is the mean of a generated distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP (as described in Viscusi et al., [1991][29]) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB.
Nonfatal Myocardial Infarction (heart attack) 3% discount rate				Age-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).[30]
Age 0–24	$66,902	$66,902	$66,902	Direct medical costs are based on simple average of estimates from
Age 25–44	$74,676	$74,676	$74,676	Russell et al. (1998)[31] and Wittels et al. (1990).[32]
Age 45–54	$78,834	$78,834	$78,834	Lost earnings:
Age 55–65	$140,649	$140,649	$140,649	Cropper and Krupnick (1990). Present discounted value of 5 years of
Age 66 and over	$66,902	$66,902	$66,902	lost earnings:
				age of onset: at 3% at 7%
7% discount rate				25-44 $8,774 $7,855
Age 0–24	$65,293	$65,293	$65,293	45-54 $12,932 $11,578
Age 25–44	$73,149	$73,149	$73,149	55-65 $74,746 $66,920
Age 45–54	$76,871	$76,871	$76,871	Direct medical expenses: An average of:
Age 55–65	$132,214	$132,214	$132,214	1. Wittels et al. (1990) ($102,658—no discounting)
Age 66 and over	$65,293	$65,293	$65,293	2. Russell et al. (1998), 5-year period ($22,331 at 3% discount rate; $21,113 at 7% discount rate)

(continued)

Table 6.3-9. Unit Values Used for Economic Valuation of Health Endpoints (2000$)[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)	$12,378	$12,378	$12,378	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality (2000)[33] (www.ahrq.gov).
Pneumonia (ICD codes 480-487)	$14,693	$14,693	$14,693	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total pneumonia category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
Asthma Admissions	$6,634	$6,634	$6,634	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
All Cardiovascular (ICD codes 390-429)	$18,387	$18,387	$18,387	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
Emergency Room Visits for Asthma	$286	$286	$286	Simple average of two unit COI values: (1) $311.55, from Smith et al. (1997)[34] and (2) $260.67, from Stanford et al. (1999).[35]

(continued)

Table 6.3-9. Unit Values Used for Economic Valuation of Health Endpoints (2000$)[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)	$25	$27	$27	Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994)[36] to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for URS is the average of the dollar values for the seven different types of URS.
Lower Respiratory Symptoms (LRS)	$16	$17	$17	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS.
Asthma Exacerbations	$42	$45	$45	Asthma exacerbations are valued at $42 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986).[37] This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study.
Acute Bronchitis	$360	$380	$390	Assumes a 6-day episode, with daily value equal to the average of low and high values for related respiratory symptoms recommended in Neumann et al. (1994).[38]

(continued)

Table 6.3-9. Unit Values Used for Economic Valuation of Health Endpoints (2000$)[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.
Minor Restricted Activity Days (MRADs)	$51	$54	$55	Median WTP estimate to avoid one MRAD from Tolley et al. (1986).[39]

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

[b] Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. Benefits are therefore adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor to account for income growth over time. For a complete discussion of how these adjustment factors were derived, we refer the reader to Chapter 9 of the CAND regulatory impact analysis (EPA, 2004). Note that similar adjustments do not exist for cost-of-illness-based unit values. For these, we apply the same unit value regardless of the future year of analysis.

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The adoption of a value for the projected reduction in the risk of premature mortality is the subject of continuing discussion within the economics and public policy analysis community. EPA strives to use the best economic science in its analyses. Given the mixed theoretical finding and empirical evidence regarding adjustments to VSL for risk and population characteristics, we use a single VSL for all reductions in mortality risk.

Although there are several differences between the labor market studies EPA uses to derive a VSL estimate and the PM air pollution context addressed here, those differences in the affected populations and the nature of the risks imply both upward and downward adjustments. Table 6.3-10 lists some of these differences and the expected effect on the VSL estimate for air pollution-related mortality. In the absence of a comprehensive and balanced set of adjustment factors, EPA believes it is reasonable to continue to use the $5.5 million value while acknowledging the significant limitations and uncertainties in the available literature.

Table 6.3-10. Expected Impact on Estimated Benefits of Premature Mortality Reductions of Differences Between Factors Used in Developing Applied VSL and Theoretically Appropriate VSL

Attribute	Expected Direction of Bias
Age	Uncertain, perhaps overestimate
Life Expectancy/Health Status	Uncertain, perhaps overestimate
Attitudes Toward Risk	Underestimate
Income	Uncertain
Voluntary vs. Involuntary	Uncertain, perhaps underestimate
Catastrophic vs. Protracted Death	Uncertain, perhaps underestimate

The SAB-EEAC has reviewed many potential VSL adjustments and the state of the economics literature. The SAB-EEAC advised EPA to "continue to use a wage-risk-based VSL as its primary estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which adjustments to the VSL can be made is the timing of the risk" (U.S. EPA, 2000a). In developing our primary estimate of the benefits of premature mortality reductions, we have followed this advice and discounted over the lag period between exposure and premature mortality.

Uncertainties Specific to Premature Mortality Valuation. The economic benefits associated with $PM_{2.5}$-related premature mortality are the largest category of monetized benefits associated with the proposed standards. In addition, in prior analyses, EPA has identified valuation of mortality benefits as the largest contributor to the range of uncertainty in monetized benefits (see U.S. EPA, 1999).[W] Because of the uncertainty in estimates of the value of premature mortality

[W] This conclusion was based on a assessment of uncertainty based on statistical error in epidemiological effect estimates and economic valuation estimates. Additional sources of model error such as those examined in the PM

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

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

Although a survival curve approach provides a theoretically preferred method for valuing the benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value avoided premature mortality risk using the VSL approach.

Other uncertainties specific to premature mortality valuation include the following:

- *Across-study variation:* There is considerable uncertainty as to whether the available literature on VSL provides adequate estimates of the VSL saved by air pollution reduction. Although there is considerable variation in the analytical designs and data used in the existing literature, the majority of the studies involve the value of risks to a middle-aged working population. Most of the studies examine differences in wages of risky occupations, using a hedonic wage approach. Certain characteristics of both the

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mortality expert elicitation may result in different conclusions about the relative contribution of sources of uncertainty.

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

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

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

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

- *Self-selection and skill in avoiding risk:* Recent research (Shogren and Stamland, 2002) suggests that VSL estimates based on hedonic wage studies may overstate the average

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

- *Baseline risk and age:* Recent research (Smith, Pattanayak, and Van Houtven, 2006) finds that because individuals reevaluate their baseline risk of death as they age, the marginal value of risk reductions does not decline with age as predicted by some lifetime consumption models. This research supports findings in recent stated preference studies that suggest only small reductions in the value of mortality risk reductions with increasing age.

Valuing Reductions in the Risk of Chronic Bronchitis. The best available estimate of WTP to avoid a case of CB comes from Viscusi, Magat, and Huber (1991). The Viscusi, Magat, and Huber study, however, describes a severe case of CB to the survey respondents. We therefore employ an estimate of WTP to avoid a pollution-related case of CB, based on adjusting the Viscusi, Magat, and Huber (1991) estimate of the WTP to avoid a severe case. This is done to account for the likelihood that an average case of pollution-related CB is not as severe. The adjustment is made by applying the elasticity of WTP with respect to severity reported in the Krupnick and Cropper (1992) study. Details of this adjustment procedure are provided in the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of CB in this analysis. The distribution incorporates uncertainty from three sources: the WTP to avoid a case of severe CB, as described by Viscusi, Magat, and Huber; the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi, Magat, and Huber); and the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these three uncertain components, we derive a distribution of WTP to avoid a pollution-related case of CB by statistical uncertainty analysis techniques. The expected value (i.e., mean) of this distribution, which is about $331,000 (2000$), is taken as the central tendency estimate of WTP to avoid a PM-related case of CB.

Valuing Reductions in Nonfatal Myocardial Infarctions (Heart Attacks). The Agency has recently incorporated into its analyses the impact of air pollution on the expected number of nonfatal heart attacks, although it has examined the impact of reductions in other related cardiovascular endpoints. We were not able to identify a suitable WTP value for reductions in the risk of nonfatal heart attacks. Instead, we use a COI unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with 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% discount rate, we estimated a present discounted value in lost earnings (in 2000$) over 5 years due to a myocardial infarction of $8,774 for someone between the ages of 25 and 44, $12,932 for someone between the ages of 45 and 54, and $74,746 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2000$) using a 7% discount rate are $7,855, $11,578, and $66,920, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. As such, we do not include lost earnings in the cost estimates for these age groups.

We found three possible sources in the literature of estimates of the direct medical costs of myocardial infarction:

- Wittels et al. (1990) estimated expected total medical costs of myocardial infarction over 5 years to be $51,211 (in 1986$) for people who were admitted to the hospital and survived hospitalization. (There does not appear to be any discounting used.) Wittels et al. was used to value coronary heart disease in the 812 Retrospective Analysis of the Clean Air Act. Using the CPI-U for medical care, the Wittels estimate is $109,474 in year 2000$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes, and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The authors note that the average length of hospitalization for acute myocardial infarction has decreased over time (from an average of 12.9 days in 1980 to an average of 11 days in 1983). Wittels et al. used 10 days as the average in their study. It is unclear how much further the length of stay for myocardial infarction may have decreased from 1983 to the present. The average length of stay for ICD code 410 (myocardial infarction) in the year-2000 Agency for Healthcare Research and Quality (AHRQ) HCUP database is 5.5 days. However, this may include patients who died in the hospital (not included among our nonfatal myocardial infarction cases), whose length of stay was therefore substantially shorter than it would be if they had not died.

- Eisenstein et al. (2001) estimated 10-year costs of $44,663 in 1997$, or $49,651 in 2000$ for myocardial infarction patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included.

- Russell et al. (1998) estimated first-year direct medical costs of treating nonfatal myocardial infarction of $15,540 (in 1995$) and $1,051 annually thereafter. Converting to year 2000$, that would be $23,353 for a 5-year period (without discounting) or $29,568 for a 10-year period.

In summary, the three different studies provided significantly different values (see Table 6.3-11).

Table 6.3-11. Alternative Direct Medical Cost of Illness Estimates for Nonfatal Heart Attacks

Study	Direct Medical Costs (2000$)	Over an x-Year Period, for x =
Wittels et al. (1990)	$109,474[a]	5
Russell et al. (1998)	$22,331[b]	5
Eisenstein et al. (2001)	$49,651[b]	10
Russell et al. (1998)	$27,242[b]	10

[a] Wittels et al. (1990) did not appear to discount costs incurred in future years.

[b] Using a 3% discount rate. Discounted values as reported in the study.

As noted above, the estimates from these three studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used estimates for medical costs that similarly cover a 5-year period (i.e., estimates from Wittels et al. (1990) and Russell et al. (1998). We used a simple average of the two 5-year estimates, or $65,902, and added it to the 5-year opportunity cost estimate. The resulting estimates are given in Table 6.3-12.

Table 6.3-12. Estimated Costs Over a 5-Year Period (in 2000$) of a Nonfatal Myocardial Infarction

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

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

[b] From Cropper and Krupnick (1990), using a 3% discount rate.

6.3.6 Human Welfare Impact Assessment

Ozone, PM and their precursor emissions have numerous documented effects on environmental quality that affect human welfare. These welfare effects include direct damages to property, either through impacts on material structures or by soiling of surfaces, direct economic damages in the form of lost productivity of crops and trees, indirect damages through alteration of ecosystem functions, and indirect economic damages through the loss in value of recreational experiences or the existence value of important resources. EPA's Criteria Documents for ozone, PM, NO_x, and SO_2 list numerous physical and ecological effects known to be linked to ambient concentrations of these pollutants (U.S. EPA, 2005; 1993). This section describes individual effects and how we quantify and monetize them. These effects include changes in nitrogen and sulfate deposition, and visibility.

6.3.6.1 Visibility Benefits

Changes in the level of ambient $PM_{2.5}$ caused by the reduction in emissions associated with the proposed standards will change the level of visibility throughout the United States. Visibility directly affects people's enjoyment of a variety of daily activities. Individuals value visibility both in the places they live and work, in the places they travel to for recreational purposes, and at sites of unique public value, such as the Great Smoky Mountains National Park. This section discusses the measurement of the economic benefits of improved visibility.

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

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

Residential visibility benefits are those that occur from visibility changes in urban, suburban, and rural areas and also in recreational areas not listed as federal Class I areas.[Y] For the purposes of this analysis, recreational visibility improvements are defined as those that occur specifically in federal Class I areas. A key distinction between recreational and residential benefits is that only those people living in residential areas are assumed to receive benefits from

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

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

residential visibility, while all households in the United States are assumed to derive some benefit from improvements in Class I areas. Values are assumed to be higher if the Class I area is located close to their home.[Z]

Only two existing studies provide defensible monetary estimates of the value of visibility changes. One is a study on residential visibility conducted in 1990 (McClelland et al., 1993) and the other is a 1988 survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b). Although there are a number of other studies in the literature, they were conducted in the early 1980s and did not use methods that are considered defensible by current standards. Both the Chestnut and Rowe and McClelland et al. studies use the CV method. There has been a great deal of controversy and significant development of both theoretical and empirical knowledge about how to conduct CV surveys in the past decade. In EPA's judgment, the Chestnut and Rowe study contains many of the elements of a valid CV study and is sufficiently reliable to serve as the basis for monetary estimates of the benefits of visibility changes in recreational areas.[AA] This study serves as an essential input to our estimates of the benefits of recreational visibility improvements in the primary benefits estimates. Consistent with SAB advice, EPA has designated the McClelland et al. study as significantly less reliable for regulatory benefit-cost analysis, although it does provide useful estimates on the order of magnitude of residential visibility benefits (U.S. EPA-SAB, 1999). Residential visibility benefits are not calculated for this analysis.

The Chestnut and Rowe study measured the demand for visibility in Class I areas managed by the National Park Service (NPS) in three broad regions of the country: California, the Southwest, and the Southeast. Respondents in five states were asked about their WTP to protect national parks or NPS-managed wilderness areas within a particular region. The survey used photographs reflecting different visibility levels in the specified recreational areas. The visibility levels in these photographs were later converted to deciviews for the current analysis. The survey data collected were used to estimate a WTP equation for improved visibility. In addition to the visibility change variable, the estimating equation also included household income as an explanatory variable.

The Chestnut and Rowe study did not measure values for visibility improvement in Class I areas outside the three regions. Their study covered 86 of the 156 Class I areas in the United States. We can infer the value of visibility changes in the other Class I areas by transferring

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[Z] For details of the visibility estimates discussed in this chapter, please refer to the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

[AA] An SAB advisory letter indicates that "many members of the Council believe that the Chestnut and Rowe study is the best available" (EPA-SAB-COUNCIL-ADV-00-002, 1999, p. 13). However, the committee did not formally approve use of these estimates because of concerns about the peer-reviewed status of the study. EPA believes the study has received adequate review and has been cited in numerous peer-reviewed publications (Chestnut and Dennis, 1997).

values of visibility changes at Class I areas in the study regions. A complete description of the benefits transfer method used to infer values for visibility changes in Class I areas outside the study regions is provided in the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

The Chestnut and Rowe study, although representing the best available estimates, has a number of limitations. These include the following:

- The age of the study (late 1980s) will increase the uncertainty about the correspondence of the estimated values to those that might be provided by current or future populations.
- The survey focused only on populations in five states, so the application of the estimated values to populations outside those states requires that preferences of populations in the five surveyed states be similar to those of nonsurveyed states.
- There is an inherent difficulty in separating values expressed for visibility improvements from an overall value for improved air quality. The Chestnut and Rowe study attempted to control for this by informing respondents that "other households are being asked about visibility, human health, and vegetation protections in urban areas and at national parks in other regions." However, most of the respondents did not feel that they were able to segregate visibility at national parks entirely from residential visibility and health effects.
- It is not clear exactly what visibility improvements the respondents to the Chestnut and Rowe survey were valuing. For the purpose of the benefits analysis for this rule, EPA assumed that respondents provided values for changes in annual average visibility. Because most policies will result in a shift in the distribution of visibility (usually affecting the worst days more than the best days), the annual average may not be the most relevant metric for policy analysis.
- The WTP question asked about changes in average visibility. However, the survey respondents were shown photographs of only summertime conditions, when visibility is generally at its worst. It is possible that the respondents believed those visibility conditions held year-round, in which case they would have been valuing much larger overall improvements in visibility than what otherwise would be the case.
- The survey did not include reminders of possible substitutes (e.g., visibility at other parks) or budget constraints. These reminders are considered to be best practice for stated preference surveys.
- The Chestnut and Rowe survey focused on visibility improvements in and around national parks and wilderness areas. The survey also focused on visibility improvements of national parks in the southwest United States. Given that national parks and wilderness areas exhibit unique characteristics, it is not clear whether the WTP estimate obtained from Chestnut and Rowe can be transferred to other national parks and wilderness areas, without introducing additional uncertainty.

In general, the survey design and implementation reflect the period in which the survey was conducted. Since that time, many improvements to the stated preference methodology have been developed. As future survey efforts are completed, EPA will incorporate values for visibility improvements reflecting the improved survey designs.

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

Using the methodology outlined above, EPA estimates that the total WTP for the visibility improvements in California, Southwestern, and Southeastern Class I areas associated with the proposed standards would be $150 million in 2020 and $400 million in 2030. These values includes the value to households living in the same states as the Class I areas as well as values for all households in the United States living outside the states containing the Class I areas, and the value accounts for growth in real income.

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

6.3.6.2 Agricultural, Forestry, and Other Vegetation-Related Benefits

The Ozone Criteria Document notes that "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant" (EPA, 2006).[41] Changes in ground-level ozone are expected to improve crop and forest yields throughout the country as a result of the proposed standards.

Well-developed techniques exist to provide monetary estimates of these benefits to agricultural producers and to consumers. These techniques use models of planting decisions, yield response functions, and agricultural products' supply and demand. The resulting welfare

measures are based on predicted changes in market prices and production costs. Models also exist to measure benefits to silvicultural producers and consumers. However, these models have not been adapted for use in analyzing ozone-related forest impacts. Because of resource limitations, we are unable to provide agricultural or forestry benefits estimates for the proposed standards.

Laboratory and field experiments have shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN), examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the United States."[42] In addition, economic studies have shown a relationship between observed ozone levels and crop yields.[43]

Ozone also has been shown conclusively to cause discernible injury to forest trees (EPA, 1996; Fox and Mickler, 1996).[68,44] In our previous analysis of the HD Engine/Diesel Fuel rule, we were able to quantify the effects of changes in ozone concentrations on tree growth for a limited set of species. Because of resource limitations, we were not able to quantify such impacts for this analysis.

NO_x emission reductions will 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 oversaturation due to an abundance of on-farm nitrogen production, primarily from animal manure. In these areas, reductions in atmospheric deposition of nitrogen represent additional agricultural benefits.

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

On the other hand, there is evidence that forest ecosystems in some areas of the United States are nitrogen saturated (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.

6.3.6.3 Benefits from Reductions in Materials Damage

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

Previous EPA benefits analyses have been able to provide quantitative estimates of household soiling damage. Consistent with SAB advice, we determined that the existing data (based on consumer expenditures from the early 1970s) are too out of date to provide a reliable estimate of current household soiling damages (U.S. EPA, 1998).

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

6.3.6.4 Benefits from Reduced Ecosystem Damage

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

Direct 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.

If better models of ecological effects can be defined, EPA believes that progress can be made in estimating WTP measures for ecosystem functions. These estimates would be superior to avoided cost estimates in placing economic values on the welfare changes associated with air

pollution damage to ecosystem health. For example, if nitrogen or sulfate loadings can be linked to measurable and definable changes in fish populations or definable indexes of biodiversity, then stated preference studies can be designed to elicit individuals' WTP for changes in these effects. This is an important area for further research and analysis and will require close collaboration among air quality modelers, natural scientists, and economists.

6.4 Benefits Analysis Results for the Proposed Standards

Applying the impact and valuation functions described previously in this chapter to the estimated changes in $PM_{2.5}$ associated with the proposed standards results in estimates of the changes in health effects (e.g., premature mortalities, cases, admissions) and the associated monetary values for those changes. Estimates of physical health impacts are presented in Table 6.4-1. Monetized values for those health endpoints are presented in Table 6.4-2, along with total aggregate monetized benefits. All of the monetary benefits are in constant-year 2005 dollars. For each endpoint and total benefits, we provide both the mean estimate and the 95% confidence interval.

In addition to omitted benefits categories such as air toxics and various welfare effects, not all known $PM_{2.5}$-related health and welfare effects could be quantified or monetized. The monetized value of all of these unquantified effects is represented by adding an unknown "B" to the aggregate total. The estimate of total monetized health benefits of the proposed standards is thus equal to the subset of monetized $PM_{2.5}$- related health benefits plus B, the sum of the nonmonetized health and welfare benefits.

Total monetized benefits are dominated by benefits of mortality risk reductions. We provide results based on 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 intends to ask its Science Advisory Board to evaluation 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, and if so, the appropriateness of using Laden et al 2006, the most recently published update, as the estimate for the Six Cities based model.

Using the ACS and Six-Cities results, we estimate that the proposed standards would result in between 570 and 1,300 cases of avoided $PM_{2.5}$-related premature deaths annually in 2020 and between 1,500 and 3,400 avoided premature deaths annually in 2030. Note that in the case of the premature mortality estimates derived from the expert elicitation, we report the 95%

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.

As noted above, we provide two approaches to estimating avoided premature mortality associated with $PM_{2.5}$ exposure. Our estimate of total monetized benefits in 2020 for the proposed standards, using the ACS and Six-Cities PM mortality studies, is between $4.4 billion and $9.2 billion, assuming a 3 percent discount rate (or $4.0 and $8.3 billion assuming a 7 percent discount rate). In 2030, the monetized benefits are estimated to be between $12 billion and $25 billion (or $11 and $23 billion assuming a 7 percent discount rate). The monetized benefit associated with reductions in the risk of $PM_{2.5}$-related premature mortality is over 90 percent of total monetized health benefits, in part because we are unable to quantify a number benefits categories (see Table 6.1-2). These unquantified benefits may be substantial, although their magnitude is highly uncertain. Our estimate of total monetized benefits based on the expert elicitation is between $1.7 billion and $12 billion, assuming a 3 percent discount rate (or $1.6 and $11 billion assuming a 7 percent discount rate). In 2030, the monetized benefits are estimated to be between $4.6 billion and $33 billion (or $4.3 and $30 billion assuming a 7 percent discount rate).

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 6.4-2.

Following these tables, we also provide a more comprehensive graphical presentation of the distributions of incidence generated using the available information from empirical studies and expert elicitation. Figures 6.4-1 and 6.4-2 present box plots of 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 distributions are depicted as box plots with the diamond symbol (◆) showing the mean, the

dash (–) showing the median (50th percentile), the box defining the interquartile range (bounded by the 25th and 75th percentiles), and the whiskers defining the 90% confidence interval (bounded by the 5th and 95th percentiles of the distribution). The mean and 90% confidence interval for each separate estimate of mortality is also provided in Tables 6.4-3 and 6.4-4.

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.[BB] 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) [45], (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) [46] and (e) background or 3 µg/m3 (reflects NRC recommendation to consider effects all the way to background).[47] We repeat this sensitivity analysis for the RIA of the proposed standards, the results of which can be found in Table 6.4-5.

A sensitivity analysis such as this can be difficult to interpret, because when a threshold above the lowest observed level of $PM_{2.5}$ in the underlying epidemiology study (Pope et al., 2002) is assumed, the slope of the concentration-response function above that level must be adjusted upwards to account for the assumed threshold.[CC] Depending on the amount of slope adjustment and the proportion of the population exposed above the assumed threshold, the estimated mortality impact can either be lower (if most of the exposures occur below the threshold) or higher (if most of the exposures occur above the threshold). To demonstrate this possibility, we present an example from the proposed PM NAAQS RIA. In its examination of the benefits of attaining alternative PM NAAQS in Chicago,[DD] the analysis found that, because annual mean levels are generally higher in Chicago, there was a two-part pattern to the relationship between assumed threshold and mortality impacts. As the threshold increased from background to 7.5 $\mu g/m^3$, the mortality impact fell (because there is no slope adjustment). However, at an assumed threshold of 10 $\mu g/m^3$, estimated mortality impacts actually increased, because the populations exposed above 10 $\mu g/m^3$ were assumed to have a larger response to particulate matter reductions (due to the increased slope above the assumed threshold). And finally, mortality impacts again fell to zero if a 15 $\mu g/m^3$ threshold was assumed, because these impacts were measured incremental to attainment of the current standard.

[BB] 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.

[CC] See NAS (2002)[87] and CASAC (2005)[85] for discussions of this issue.

[DD] See the proposed PM NAAQS RIA (2005),[67] Appendix A, pp. A63-A64.

Our analysis of the proposed standards also demonstrates this possibility. In Table 6.4-5, we can see that there is a two-part pattern to the relationship between assumed threshold and mortality impacts. As the threshold increases from background to 7.5 μg/m3, we see no difference in mortality impact (because all changes in PM appear to occur above a 7.5 μg/m3 threshold and there is no slope adjustment). At a threshold of 10 μg/m3, however, estimated mortality impacts actually increase, because the populations exposed above 10 μg/m3 are assumed to have a larger response to particulate matter reductions (due to the increased slope above the assumed threshold). Finally, like the PM NAAQS example, mortality impacts again fall as the threshold is increased.

Table 6.4-1. Estimated Reduction in Incidence of Adverse Health Effects Related to the Proposed Standards[a]

	2020	2030
Health Effect	Mean Incidence Reduction (5^{th} – 95^{th} %ile)	
PM-Related Endpoints		
Premature Mortality – Derived from Epidemiology Studies[b,c] Adult, age 30+ - Range based on Pope et al. 2002 and Laden et al.2006, Respectively Infant, age <1 year – Woodruff et al. 1997	570 - 1,300 (220-920)–(710-1,900) 1 (1-2)	1,500 - 3,400 (590-2,400)-(1,900-5,000) 2 (1-4)
Premature Mortality – Derived from Expert Elicitation[c,d] Adult, age 25+ - Lower and Upper Bound EE Results, Respectively	180 - 1,700 (0-830) – (870-2,600)	460 - 4,600 (0-2,200) – (2,300-6,900)
Chronic bronchitis (adult, age 26 and over)	370 (68 – 670)	940 (170 – 1,700)
Acute myocardial infarction (adults, age 18 and older)	1,200 (640 – 1,700)	3,300 (1,800 – 4,800)
Hospital admissions—respiratory (all ages)[e]	130 (65 – 200)	350 (170 – 510)
Hospital admissions—cardiovascular (adults, age >18)[f]	270 (170 – 380)	770 (490 – 1,100)
Emergency room visits for asthma (age 18 years and younger)	460 (270 – 650)	1,000 (620 – 1,500)
Acute bronchitis (children, age 8–12)	1,000 (0 – 2,100)	2,600 (0 – 5,300)
Lower respiratory symptoms (children, age 7–14)	11,000 (5,400 – 17,000)	28,000 (14,000 – 43,000)
Upper respiratory symptoms (asthmatic children, age 9–18)	8,300 (2,600 – 14,000)	21,000 (6,600 – 35,000)
Asthma exacerbation (asthmatic children, age 6–18)	10,000 (1,100 – 29,000)	26,000 (2,800 – 74,000)
Work loss days (adults, age 18–65)	71,000 (62,000 – 81,000)	170,000 (150,000 – 190,000)
Minor restricted-activity days (adults, age 18–65)	420,000 (360,000 – 490,000)	1,000,000 (850,000 – 1,200,000)

[a] Incidence is rounded to two significant digits. PM estimates represent benefits from the proposed standards nationwide.

[b] Based on application of the effect estimate derived from the Pope et al (2002) cohort study and the Laden et al (2006) study.[48,49] Note that these two estimates are not additive; instead, they provide a range of mortality incidence derived from the epidemiology literature. Infant premature mortality based upon studies by Woodruff, et al. 1997.[50]

[c] PM-related mortality benefits estimated using an assumed PM threshold at 10 μg/m3. There is uncertainty about which threshold to use and this may impact the magnitude of the total benefits estimate.

[d] 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).[51] The lower bound result reflects the function derived from the expert with the most conservative effect estimate. The upper bound result reflects the

function derived from the expert with the least conservative effect estimate. It should be noted, however, that the weight of expert-based opinion on the risk of premature death is skewed towards the range reflected by the published scientific studies. The effect estimates of nine of the twelve experts included in the elicitation panel falls within the scientific study-based range provided by Pope and Laden. One of the experts fall below this range and two of the experts are above this range.

[e] Respiratory hospital admissions for PM include admissions for COPD, pneumonia, and asthma.

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

Table 6.4-2. 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,e]		
Adult, age 30+ - ACS study (Pope et al. 2002)		
3% discount rate	$3,900 ($500 - $8,800)	$10,000 ($1,500 - $24,000)
7% discount rate	$3,700 ($500 - $7,900)	$9,400 ($1,300 - $21,000)
Adult, age 30+ - Six-Cities study (Laden et al. 2006)		
3% discount rate	$8,700 ($1,400 - $18,000)	$24,000 ($3,800 - $50,000)
7% discount rate	$7,800 ($1,300 - $17,000)	$21,000 ($3,400 - $45,000)
Infant Mortality,<1 year – Woodruff et al. 1997		
3% discount rate	$8 ($1 - $18)	$17 ($3 - $37)
7% discount rate	$7 ($1 - $16)	$15 ($2 - $$33)
Premature mortality – Derived from Expert Elicitation[c,d,e,f]		
Adult, age 25+ - Lower bound EE result		
3% discount rate	$1,200 ($0 - $7,200)	$3,300 ($0 - $20,000)
7% discount rate	$1,100 ($0 - $6,500)	$3,000 ($0 - $18,000)
Adult, age 25+ - Upper bound EE result		
3% discount rate	$12,000 ($1,800 - $25,000)	$31,000 ($4,800 - $68,000)
7% discount rate	$11,000 ($1,600 - $23,000)	$28,000 ($4,400 - $62,000)
Chronic bronchitis (adults, 26 and over)	$200 ($10 - $800)	$500 ($26 - $2,100)
Non-fatal acute myocardial infarctions		
3% discount rate	$123 ($32 - $270)	$330 ($80 - $730)
7% discount rate	$119 ($30 - $270)	$320 ($76 - $720)
Hospital admissions for respiratory causes	$2.7 ($1.3 - $4.0)	$7.2 ($3.6 - $11)
Hospital admissions for cardiovascular causes	$7.3 ($4.6 - $10)	$21 ($13 - $28)
Emergency room visits for asthma	$0.16	$0.37

	($0.09 - $0.26)	($0.20 - $0.60)
Acute bronchitis (children, age 8–12)	$0.44 ($0 - $1.2)	$1.1 ($0 - $3.1)
Lower respiratory symptoms (children, 7–14)	$0.21 ($0.07 - $0.43)	$0.53 ($0.18 - $1.1)
Upper respiratory symptoms (asthma, 9–11)	$0.24 ($0.05 - $0.59)	$0.62 ($0.14 - $1.5)
Asthma exacerbations	$0.53 ($0.04 - $2.0)	$1.4 ($0.10 - $5.1)
Work loss days	$11 ($9.6 - $12)	$27 ($23 - $30)
Minor restricted-activity days (MRADs)	$12 ($0.61 – $25)	$29 ($1.5 - $60)
Recreational Visibility, 86 Class I areas	$150 (na)[f]	$400 (na)
Monetized Total – PM-Mortality Derived from Epi. Studies; Morbidity Functions 3% discount rate 7% discount rate	 $4.4 - $9.2 Billion ($1.0 - $10) – ($1.6 - $20) $4.0 - $8.3 Billion ($1.0 - $9.2) – ($1.5 - $18)	 $12 - $25 Billion ($2.1 - $27) – ($4.4 - $53) $11 - $23 Billion ($1.8 - $25) – ($3.9 - $48)
Monetized Total – PM-Mortality Derived from Expert Elicitation[g]; Morbidity Functions 3% discount rate 7% discount rate	$1.7 - $12 Billion($0.2 - $8.5) – ($2.0 - $27)$1.6 - $11 Billion($0.2 - $7.8) – ($1.8 - $24)	$4.6 - $33 Billion($1.0 - $23) – ($5.4 - $72)$4.3 - $30 Billion($1.0 - $21) – ($4.9 - $65)

[a] Monetary benefits are rounded to two significant digits for ease of presentation and computation. PM 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] PM-related mortality benefits estimated using an assumed PM threshold of 10 μg/m3. There is uncertainty about which threshold to use and this may impact the magnitude of the total benefits estimate.

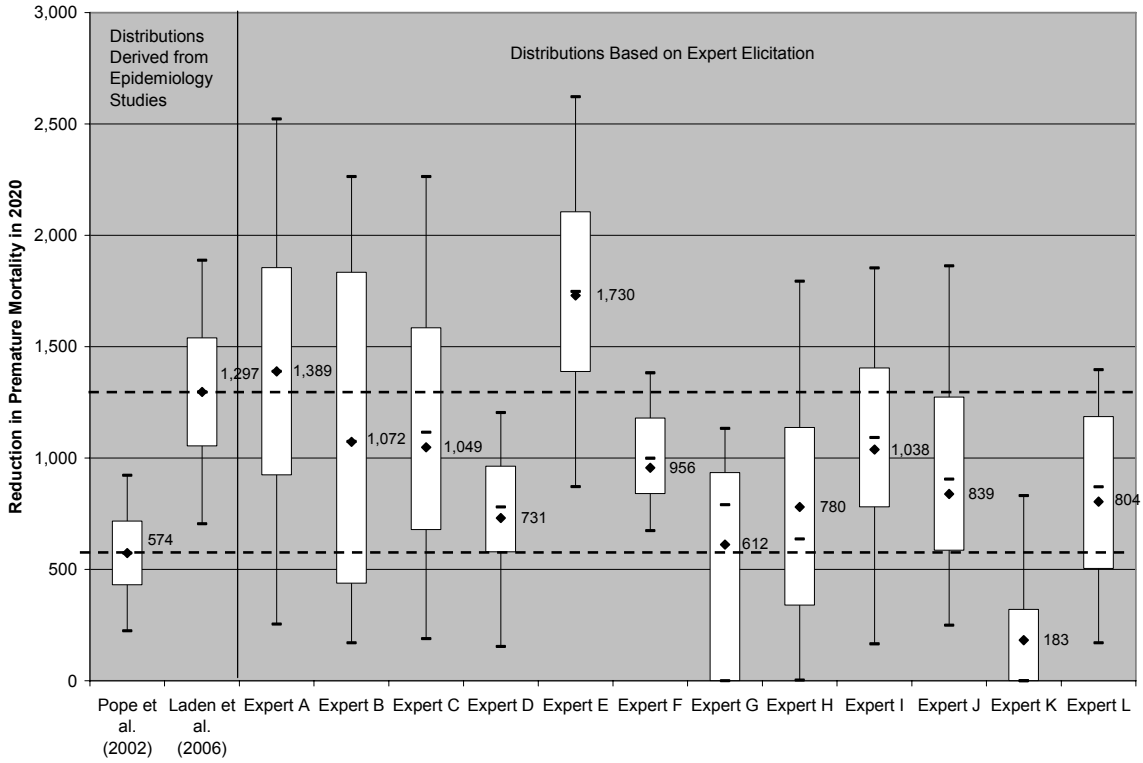
[d] Valuation assumes discounting over the SAB recommended 20 year segmented lag structure. Results reflect the use of 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).[i,ii]

[e] 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.

[f] 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.

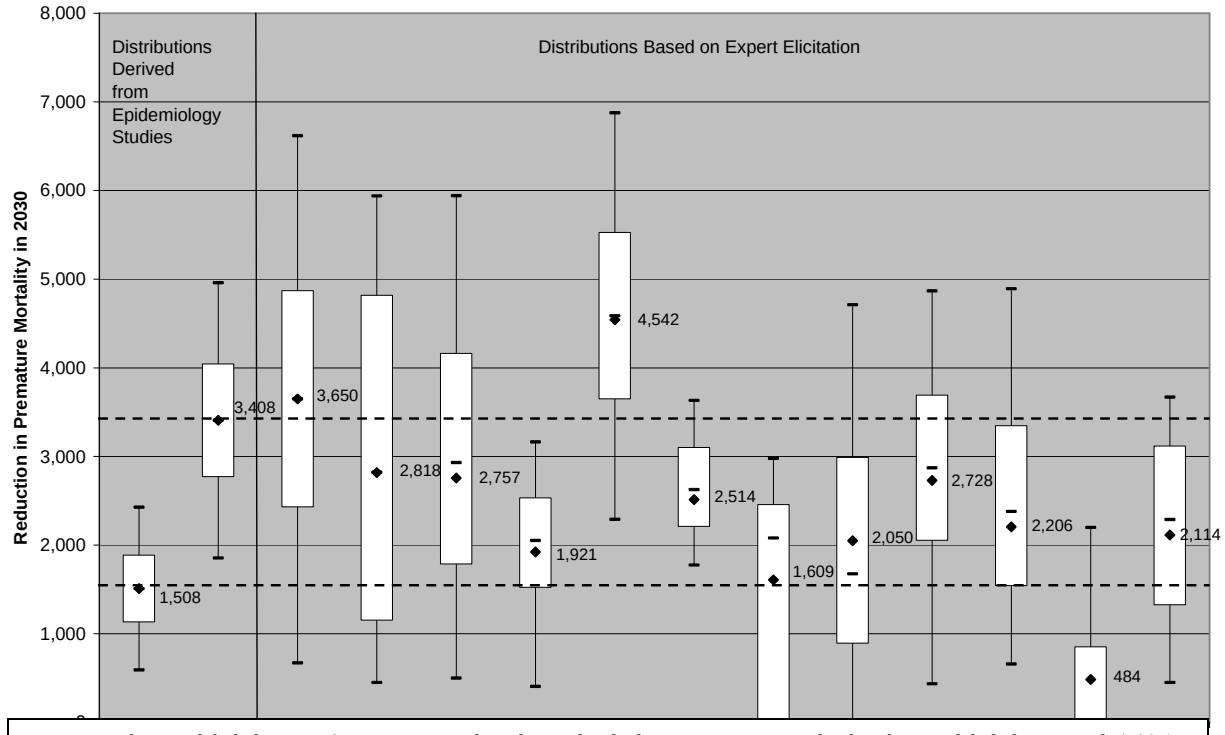
[g] It should be noted that the effect estimates of nine of the twelve experts included in the elicitation panel falls within the scientific study-based range provided by Pope and Laden. One of the experts fall below this range and two of the experts are above this range.

Figure 6.4-1. Results of Application of Expert Elicitation: Annual Reductions in Premature Mortality in 2020 Associated with the Proposed Standards



Note: Distributions labeled Expert A – Expert L are based on individual expert responses. The distributions labeled Pope et al. (2002) and Laden et al. (2006) are based on the means and standard errors of the C-R functions from the studies. The dotted lines enclose a range bounded by the means of the two data-derived distributions.

Figure 6.4-2. Results of Application of Expert Elicitation: Annual Reductions in Premature Mortality in 2030 Associated with the Proposed Standards



Note: Distributions labeled Expert A – Expert L are based on individual expert responses. The distributions labeled Pope et al. (2002) and Laden et al. (2006) are based on the means and standard errors of the C-R functions from the studies. The dotted lines enclose a range bounded by the means of the two data-derived distributions.

Table 6.4-3. Results of Application of Expert Elicitation: Annual Reductions in Premature Mortality in 2020 Associated with the Proposed Standards

Source of Mortality Estimate	2020 Primary Option		
	5th Percentile	Mean	95th Percentile
Pope et al. (2002)	220	570	920
Laden et al. (2006)	710	1,300	1,900
Expert A	250	1,400	2,500
Expert B	170	1,100	2,300
Expert C	190	1,000	2,300
Expert D	150	730	1,200
Expert E	870	1,700	2,600
Expert F	670	960	1,400
Expert G	0	610	1,100
Expert H	3	780	1,800
Expert I	170	1,000	1,900
Expert J	250	840	1,900
Expert K	0	180	830
Expert L	170	800	1,400

Table 6.4-4. Results of Application of Expert Elicitation: Annual Reductions in Premature Mortality in 2030 Associated with the Proposed Standards

Source of Mortality Estimate	2020 Primary Option		
	5th Percentile	Mean	95th Percentile
Pope et al. (2002)	590	1,500	2,400
Laden et al. (2006)	1,900	3,400	5,000
Expert A	670	3,700	6,600
Expert B	450	2,800	5,900
Expert C	500	2,800	5,900
Expert D	400	1,900	3,200
Expert E	2,300	4,500	6,900
Expert F	1,800	2,500	3,600
Expert G	0	1,600	3,000
Expert H	8	2,100	4,700
Expert I	440	2,700	4,900
Expert J	660	2,200	4,900
Expert K	0	480	2,200
Expert L	450	2,100	3,700

Table 6.4-5. PM-Related Mortality Benefits of the Proposed 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	Discount Rate	PM Mortality Benefits (Billion 2005$)	
			2020	2030
More Certain that Benefits Are at Least as Large	14 µg/m3 [b]	3%	$1.8	$5.4
		7%	$1.6	$4.8
	12 µg/m3	3%	$2.5	$7.2
		7%	$2.3	$6.5
	10 µg/m3 [c]	3%	$3.9	$10.4
		7%	$3.5	$9.4
	7.5 µg/m3 [d]	3%	$3.4	$9.2
		7%	$3.1	$8.3
Less Certain that Benefits Are at Least as Large	3 µg/m3 [e]	3%	$3.4	$9.2
		7%	$3.1	$8.3

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

[b] Alternative annual PM NAAQS.

[c] CASAC (2005)[85]

[d] SAB-HES (2004)[86]

[e] NAS (2002)[87]

6.5 Comparison of Costs and Benefits

In estimating the net benefits of the proposed 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 6.5-1 contains the estimates of monetized benefits and estimated social welfare costs for the proposed rule and each of the proposed control programs. The annual social welfare costs of all provisions of this proposed rule are described more fully in Chapter 7 of this RIA.[EE]

[EE] The estimated 2030 social welfare cost of 267.3 million is based on an earlier version of the engineering costs of the rule which estimated $568.3 million engineering costs in 2030 (see table 5-17). The current engineering cost estimate for 2030 is $605 million. See Section V.C.5 for an explanation of the difference. The estimated social costs of the program will be updated for the final rule.

The results in Table 6.5-1 suggest that the 2020 monetized benefits of the proposed standards are greater than the expected social welfare costs. Specifically, the annual benefits of the total program would be between $4.4 + B billion and $9.2 + B billion annually in 2020 using a three percent discount rate (or $4.0 and $8.3 billion assuming a 7 percent discount rate), compared to estimated social costs of approximately $250 million in that same year. These benefits are expected to increase to between $12 + B billion and $25 + B billion annually in 2030 using a three percent discount rate (or $11 and $23 billion assuming a 7 percent discount rate), while the social costs are estimated to be approximately $600 million. Though there are a number of health and environmental effects associated with the proposed standards that we are unable to quantify or monetize (represented by "+B"; see Table 6.1-2), the benefits of the proposed 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 class standards far outweigh their projected costs.

Table 6.5-1. Summary of Annual Benefits and Costs of the Proposed Standards[a]
(Millions of 2005 dollars)

Description	2020 (Millions of 2005 dollars)	2030 (Millions of 2005 dollars)
Estimated Social Costs[b]		
Locomotive	$147	$383
Marine	$103	$222
Total Social Costs	$250	$605
Estimated Health Benefits of the Proposed Standards[c,d]		
Locomotive		
3 percent discount rate	$2,300+B - $4,800+B	$4,700+B - $9,800+B
7 percent discount rate	$2,100+B - $4,400+B	$4,300+B - $8,900+B
Marine		
3 percent discount rate	$2,100+B - $4,400+B	$7,100+B - $15,000+B
7 percent discount rate	$1,900+B - $3,900+B	$6,400+B - $14,000+B
Total Benefits		
3 percent discount rate	$4,400+B - $9,200+B	$12,000+B - $25,000+B
7 percent discount rate	$4,000+B - $8,300+B	$11,000+B - $23,000+B
Annual Net Benefits (Total Benefits – Total Costs)		
3 percent discount rate	$4,150+B - $8,950+B	$11,400+B - $24,400+B
7 percent discount rate	$3,750+B - $8,050+B	$10,400+B - $22,400+B

[a] All estimates are rounded to three significant digits and 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 7 of the RIA).In Chapter 7, however, we

do 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. [FF]

[c] 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. 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). Note that the benefits in this table reflect PM mortality derived from the ACS (Pope et al., 2002) and Six-Cities (Laden et al., 2006) studies. Valuation of nonfatal myocardial infarctions (MI) assumes discounting over a 5-year period, reflecting lost earnings and direct medical costs following a nonfatal MI. Note that we do not calculate a net present value of benefits associated with the proposed standards.

[d] Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Potential benefit categories that have not been quantified and monetized are listed in Table 6.1-2.

[FF] U.S. Environmental Protection Agency, 2000. Guidelines for Preparing Economic Analyses. www.yosemite1.epa.gov/ee/epa/eed/hsf/pages/Guideline.html.

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

Appendix 6.A Health-Based Cost Effectiveness Analysis

Health-based cost-effectiveness analysis (CEA) and cost-utility analysis (CUA) have been used to analyze numerous health interventions but have not been widely adopted as tools to analyze environmental policies. The Office of Management and Budget (OMB) 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, making application of CEA more difficult and less straightforward. For the recently finalized PM NAAQS analysis, CEA provided a useful framework for evaluation: non-health benefits were substantial, but the majority of quantified benefits came from health effects. EPA included in the PM NAAQS RIA a preliminary and experimental application of one type of CEA—a modified quality-adjusted life-years (QALYs) approach. A detailed description of this QALY approach is provided in Appendix G of the final PM NAAQS RIA. For the analysis presented here, we use the same modified QALY approach to characterize the health-based cost effectiveness of the proposed standards.

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

The Institute of Medicine (a member institution of the National Academies of Science) established the Committee to Evaluate Measures of Health Benefits for Environmental, Health, and Safety Regulation to assess the scientific validity, ethical implications, and practical utility of a wide range of effectiveness measures used or proposed in CEA. This committee prepared a report titled "Valuing Health for Regulatory Cost-Effectiveness Analysis," which concluded that CEA is a useful tool for assessing regulatory interventions to promote human health and safety, although not sufficient for informed regulatory decisions (Miller, Robinson, and Lawrence, 2006).[54] 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.

The methodology derived from the final PM NAAQS analysis 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 $PM_{2.5}$ in achieving improvements in public health. Reductions in ambient $PM_{2.5}$ likely will 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 nonhealth 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 proposed standards are expected to result in substantial reductions in potential population exposure to ambient concentrations of PM by 2030. The benefit-cost analysis presented in this chapter shows that the proposed standards would achieve substantial health benefits whose monetized value far exceeds costs (net benefits are between $12 and $28 billion in 2030, based on empirically derived estimates of PM mortality and using a 3 percent discount rate). Despite the risk of oversimplifying benefits, cautiously-interpreted cost-effectiveness calculations may provide further evidence of whether the costs associated with the proposed standards are a reasonable health investment for the nation.

This analysis provides estimates of commonly used health-based effectiveness measures, including lives saved, life years saved (from reductions in mortality risk), and QALYs saved (from reductions in morbidity risk) associated with the reduction of ambient $PM_{2.5}$ due to the proposed standards. In addition, we use an alternative aggregate effectiveness metric, Morbidity Inclusive Life Years (MILY) to address some of the concerns about aggregation of life extension and quality-of-life impacts. It represents the sum of life years gained due to reductions in premature mortality and the QALY 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. This analysis focuses on life extensions and improvements in quality of life through reductions in two diseases with chronic impacts: chronic bronchitis (CB) and nonfatal acute myocardial infarctions. Monte Carlo simulations are used to propagate uncertainty in several analytical parameters and characterize the distribution of estimated impacts. While the benefit-cost analysis presented in the RIA characterizes mortality impacts using a number of different sources for the PM mortality effect estimate, for this analysis, we focus on the mortality results generated using the effect estimate derived from the Pope et al. (2002) study.

Presented in three different metrics, the analysis suggests the following:

- In 2020, the proposed standards will result in:
  - 570 (95% CI: 180 – 960) premature PM-related deaths avoided, or
  - 5,900 (95% CI: 4,100 – 7,600) PM-related life years gained (discounted at 3 percent), or
  - 11,000 (95% CI: 7,900 – 18,000) MILYs gained (discounted at 3 percent).
- In 2030, the proposed standards will result in:
  - 1,500 (95% CI: 590 – 2,400) premature PM-related deaths avoided, or
  - 15,000 (95% CI: 10,000 – 20,000) PM-related life years gained (discounted at 3 percent), or
  - 23,000 (95% CI: 16,000 – 34,000) MILYs gained (discounted at 3 percent).
- Using a 7 percent discount rate, mean discounted life years gained are 3,700 for the proposed standards in 2020 and 9,500 in 2030; mean MILYs gained are 7,300 in 2020 and 15,000 in 2030. (The estimates of premature deaths avoided are not affected by the discount rate.)
- The associated reductions in CB and nonfatal acute myocardial infarctions will reduce medical costs by approximately $180 million in 2020 and $550 million in 2030 based on a 3 percent discount rate, or $120 million in 2020 and $440 million in 2030 based on a 7 percent discount rate.
- Other health and visibility benefits are valued at $200 million in 2020 and $510 million in 2030.

Direct private compliance costs for the proposed standards are $240 million in 2020 and $600 in 2030 (see Chapter 7 of this RIA for more discussion of the cost estimates). Therefore, the net costs (private compliance costs minus avoided cost of illness minus other benefits) are negative, indicating that the proposed standards result in cost savings. As such, traditional cost-effectiveness ratios are not informative. However, it is possible to calculate the maximum costs for the rule that would still result in cost-effective improvements in public health compared with standard benchmarks of $50,000 and $100,000 per MILY:

- Taking into account avoided medical costs and other benefits, annual costs of the proposed standards would need to exceed $920 million (95% CI: $700 million – $1,400 million) in 2020 and $2.2 billion (95% CI: $1.6 billion – $3.0 billion) in 2030 to have a cost per MILY that exceeds a benchmark of $50,000, based on a 3 percent discount rate.

- Annual costs of the proposed standards would need to exceed $1.5 billion (95% CI: $1.1 billion – $2.3 billion) in 2020 and $3.4 billion (95% CI: $2.4 billion – $4.7 billion) in 2030 to have a cost per MILY that exceeds a benchmark of $100,000, based on a 3 percent discount rate.

- Using a 7 percent discount rate, annual costs of the proposed standards would need to exceed $680 million in 2020 and $1.7 billion in 2030 to have a cost per MILY that exceeds a benchmark of $50,000, and would need to exceed $1.0 billion in 2020 and $2.5 billion in 2030 to have a cost per MILY that exceeds a benchmark of $100,000.

Given costs of $240 million and $600 million in 2020 and 2030, respectively, the proposed standards are clearly a very cost-effective way to achieve improvements in public health.

Tables 6.A-1 through 6.A-9 present the intermediate and summary results of the health-based CEA of the proposed standards. Note that the methods used to generate these estimates follow the same methods as those explained in Appendix G of the final PM NAAQS RIA. We refer the reader to that document for more details about this modified QALY approach to health-based CEA.

Table 6.A-1: Estimated Reduction in Incidence of All-cause Premature Mortality Associated with the Proposed Standards in 2020 and 2030

	Reduction in All-Cause Premature Mortality (95% CI)	
Age Interval	2020	2030
30 – 34	5 (2-9)	11 (3-18)
35 – 44	15 (5-26)	35 (11-59)
45 – 54	31 (10-52)	64 (20-110)
55 – 64	78 (25-130)	150 (49-260)
65 – 74	130 (40-210)	340 (110-570)
75 – 84	140 (46-240)	460 (150-780)
85+	180 (56-300)	450 (140-750)
Total	570 (180-960)	1,510 (480-2,500)

Table 6.A-2: Estimated Life Years Gained from All-cause Premature Mortality Risk Reductions Associated with the Proposed Standards in 2020 and 2030

	Life Years Gained from Mortality Risk Reduction, 3% Discount Rate (95% CI)	
Age Interval	2020	2030
25 – 34	120 (27-210)	250 (77-450)
35 – 44	350 (120-560)	800 (250-1,300)
45 – 54	610 (190-1,000)	1,300 (420-2,100)
55 – 64	1,300 (420-2,200)	2,500 (800-4,200)
65 – 74	1,600 (500-2,700)	4,300 (1,300-7,200)
75 – 84	1,300 (400-2,100)	4,100 (1,400-6,800)
85+	630 (200-1,100)	1,600 (520-2,700)
Total	5,900 (4,100-7,600)	15,000 (10,000-20,000)

Table 6.A-3: Estimated Reduction in Incidence of Chronic Bronchitis Associated with the Proposed Standards in 2020 and 2030

	Reduction in Incidence (95% Confidence Interval)	
Age Interval	2020	2030
25 – 34	88 (8-170)	200 (18-380)
35 – 44	99 (9-190)	250 (22-480)
45 – 54	91 (8-170)	210 (19-400)
55 – 64	93 (8-180)	200 (18-380)
65 – 74	66 (6-130)	190 (17-360)
75 – 84	32 (3-61)	110 (10-210)
85+	13 (1-26)	37 (3-70)
Total	480 (43-920)	1,200 (110-2,300)

Table 6.A-4: QALYs Gained per Avoided Incidence of CB

Age Interval		QALYs Gained per Incidence	
Start Age	End Age	Undiscounted	Discounted (3%)
25	34	12.21	6.56
35	44	9.84	5.90
45	54	7.54	5.06
55	64	5.36	4.03
65	74	3.41	2.85
75	84	2.15	1.93
85+		0.79	0.76

Table 6.A-5: Estimated Reduction in Nonfatal Acute Myocardial Infarctions Associated with the Proposed Standards in 2020 and 2030

	Reduction in Incidence (95% Confidence Interval)	
Age Interval	2020	2030
18 – 24	1 (0-1)	1 (1-2)
25 – 34	4 (2-5)	8 (4-11)
35 – 44	38 (20-55)	97 (52-140)
45 – 54	121 (65-177)	280 (150-410)
55 – 64	290 (160-420)	630 (340-920)
65 – 74	340 (190-500)	1,000 (550-1,500)
75 – 84	250 (140-370)	870 (470-1,300)
85+	130 (70-190)	360 (190-520)
Total	1,200 (640-1,700)	3,270 (1,800-4,800)

Table 6.A-6: QALYs Gained per Avoided Nonfatal Myocardial Infarction

Age Interval		QALYs Gained per Incidence	
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	4.04	2.10
25	34	3.38	1.95
35	44	2.73	1.74
45	54	2.08	1.48
55	64	1.44	1.12
65	74	0.95	0.81
75	84	0.57	0.52
85+		0.31	0.30

Table 6.A-7. Estimated Gains in 3 Percent Discounted MILYs Associated with the Proposed Standards in 2020[a]

Age	Life Years Gained from Mortality Risk Reductions (95% CI)	QALY Gained from Reductions in Chronic Bronchitis (95% CI)	QALY Gained from Reductions in Acute Myocardial Infarctions (95% CI)	Total Gain in MILYs (95% CI)
18–24	-	-	3 (0-5)	3 (0-5)
25–34	120 (27-210)	560 (38-1,400)	15 (4-32)	710 (160-1,500)
35–44	350 (120-560)	590 (42-1,400)	170 (73-600)	1,100 (550-2,100)
45–54	610 (190-1,000)	460 (34-1,100)	420 (200-1,600)	1,500 (960-2,900)
55–64	1,300 (420-2,200)	380 (31-890)	710 (340-2,900)	2,400 (1,600-4,900)
65–74	1,600 (500-2.700)	190 (12-440)	820 (300-2,600)	2,600 (1,500-4,700)
75–84	1,300 (400-2,100)	62 (4-150)	460 (130-1,000)	1,800 (870-2,800)
85+	630 (200-1,100)	10 (1-23)	110 (43-300)	750 (340-1,300)
Total	5,900 (4,100-7,600)	2,300 (173-5,300)	2,700 (1,100-9,100)	11,000 (7,900-18,000)

[a] Note that all estimates have been rounded to two significant digits.

Table 6.A-8: Estimated Gains in 3 Percent Discounted MILYs Associated with the Proposed Standards in 2030[a]

Age	Life Years Gained from Mortality Risk Reductions (95% CI)	QALY Gained from Reductions in Chronic Bronchitis (95% CI)	QALY Gained from Reductions in Acute Myocardial Infarctions (95% CI)	Total Gain in MILYs (95% CI)
18–24	-	-	3 (0-5)	3 (0-5)
25–34	250 (77-450)	1,300 (79-3,100)	15 (4-32)	1,600 (340-3,400)
35–44	800 (250-1,300)	1,500 (85-3,500)	170 (76-590)	2,400 (1,000-4,600)
45–54	1,300 (420-2,100)	1,100 (75-2,500)	420 (200-1,600)	2,700 (1,600-4,900)
55–64	2,500 (800-4,200)	790 (61-1,800)	710 (360-2,900)	4,000 (2,500-7,100)
65–74	4,300 (1,300-7,200)	530 (38-1,200)	820 (310-2,500)	5,600 (2,900-9,400)
75–84	4,100 (1,400-6,800)	210 (14-500)	460 (140-1,000)	4,800 (2,100-7,600)
85+	1,600 (520-2,700)	28 (2-66)	110 (44-300)	1,700 (700-2,900)
Total	15,000 (10,000-20,000)	5,400 (390-13,000)	2,700 (1,100-9,000)	23,000 (16,000-34,000)

[a] Note that all estimates have been rounded to two significant digits.

Table 6.A-9: Summary of Health-Based Cost Effectiveness Results for the Proposed Standards in 2020 and 2030[a]

	Result Using 3% Discount Rate (95% Confidence Interval)	
	2020	2030
Life years gained from mortality risk reductions	5,900 (4,100-7,600)	15,000 (10,000-20,000)
QALY gained from reductions in chronic bronchitis	2,300 (173-5,300)	5,400 (390-13,000)
QALY gained from reductions in acute myocardial infarctions	2,700 (1,100-9,100)	2,700 (1,100-9,000)
Total gain in MILYs	11,000 (7,900-18,000)	23,000 (16,000-34,000)
Avoided cost of illness		
Chronic bronchitis	$57 Million ($37 - $89 Million)	$130 Million ($86 - $210 Million)
Nonfatal AMI	$120 Million ($67 - $200 Million)	$420 Million ($170 - $550 Million)
Other benefits (based on COI and WTP estimates)	$200 Million ($180 - $210 Million)	$510 Million ($480 – $540 Million)
Implementation strategy costs[b]	$240 Million	$600 Million
Net cost per MILY	Cost Savings	Cost Savings

[a] All summary results are reported at a precision level of two significant digits to reflect limits in the precision of the underlying elements.

[b] Costs are the private firm costs of control, as discussed in Chapter 7, and reflect discounting using firm specific costs of capital.

Appendix 6.B Sensitivity Analyses of Key Parameters in the Benefits Analysis

The primary analysis presented in Chapter 6 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 commentors 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.

6.B.1 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).[55] 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).[56] 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)[57] and Dockery et al. (1993)[58] studies, respectively.[GG] 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 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 (scaled from the CAND analysis) 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 6B-1. 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.

GG 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.

Table 6B-1. Sensitivity of Benefits of Premature Mortality Reductions to Alternative Lag Assumptions (Relative to Primary Benefits Estimates of the Proposed Standards)

		Avoided Incidences[a]		Value (million 2005$)[b]	
Description of Sensitivity Analysis		2020	2030	2020	2030
Alternative Lag Structures for PM-Related Premature Mortality					
None	Incidences all occur in the first year	570	1,500	$4,300	$11,500
8-year	Incidences all occur in the 8th year				
	3% Discount Rate	570	1,500	$3,500	$9,300
	7% Discount Rate	570	1,500	$2,600	$7,100
15-year	Incidences all occur in the 15th year				
	3% Discount Rate	570	1,500	$2,800	$7,600
	7% Discount Rate	570	1,500	$1,600	$4,400
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	570	1,500	$3,700	$10,100
	7% Discount Rate	570	1,500	$3,200	$8,700
5-Year Distributed	50 percent of incidences occur in years 1 and 2 and 50 percent in years 2 to 5				
	3% Discount Rate	570	1,500	$4,000	$10,900
	7% Discount Rate	570	1,500	$3,800	$10,200

[a] Incidences rounded to two significant digits.

[b] Dollar values rounded to two significant digits. Note that dollar values reflect the use of a 3 percent discount rate in the primary lag adjustment for valuation of alternative mortality C-R functions. 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. For example, the overall impact of moving from the previous 5-year distributed lag to the segmented lag recommended by the SAB-HES in 2004 in the 2030 primary estimate is relatively modest, reducing PM-related mortality benefits by approximately 5 percent when a 3 percent discount rate is used and approximately 10 percent when a 7 percent discount rate is used. If no lag is assumed, benefits increase by around 10 percent relative to the segmented lag with a 3 percent discount rate and 23 percent with a 7 percent discount rate.

6.B.2 Visibility Benefits in Additional Class I Areas

The Chestnut and Rowe (1990) 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 6B-2.

Table 6.B-2: Monetary Benefits Associated with Improvements in Visibility in Additional Federal Class I Areas in 2020 and 2030 (in millions of 2005$)[a]

Year	Northwest[b]	Central[c]	Northeast[d]	Total
2020	$11	$55	$10	$75
2030	$30	$130	$20	$180

[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.

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