Chapter 5: Benefits Analysis Approach and Results

Synopsis

EPA estimated the monetized human health benefits of reducing cases of morbidity and premature mortality among populations exposed to SO_2 and $PM_{2.5}$ in 2020 for each of the alternative standard levels in 2006$. For an SO_2 standard at 50 ppb (99th percentile, daily 1-hour maximum), the total monetized benefits would be $41 to $100 billion at a 3% discount rate and $37 to $90 billion at a 7% discount rate. For an SO_2 standard at 75 ppb, the total monetized benefits would be $22 to $53 billion at a 3% discount rate and $20 to $48 billion at a 7% discount rate. For an SO_2 standard at 100 ppb, the total monetized benefits would be $16 to $38 billion at a 3% discount rate and $14 to $35 billion at a 7% discount rate. For an SO_2 standard at 150 ppb, the total monetized benefits would be $6.4 to $16 billion at a 3% discount rate and $5.8 to $14 billion at a 7% discount rate.

These estimates reflect EPA's most current interpretation of the scientific literature and include three key changes: (1) a no-threshold model for PM2.5 that calculates incremental benefits down to the lowest modeled air quality levels; (2) a different Value of Statistical Life (VSL); (3) two technical updates to the population dataset and aggregation method.[1] These benefits are incremental to an air quality baseline that reflects attainment with the 2008 ozone and 2006 $PM_{2.5}$ National Ambient Air Quality Standards (NAAQS). More than 99% of the total dollar benefits are attributable to reductions in $PM_{2.5}$ exposure resulting from SOx emission controls. Higher or lower estimates of benefits are possible using other assumptions; examples of this are provided in Figures 5.1 and 5.2 for the proposal standard range of 50 ppb to 100 ppb. Methodological limitations prevented EPA from quantifying the impacts to, or monetizing the benefits from several important benefit categories, including ecosystem effects from sulfur deposition, improvements in visibility, and materials damage. Other direct benefits from reduced SO_2 exposure have not been quantified, including reductions in premature mortality.

[1] Using the previous methodology (i.e., a threshold model at 10 $\mu g/m^3$ without two technical updates), the total monetized benefits would be $27 to $58 billion (2006$, 3 percent discount rate) for the 50 ppb standard alternative, $14 to $31 billion for the 75 ppb standard alternative, $10 to $22 billion for the 100 ppb standard alternative, and $4.2 to $9.0 billion for the 150 ppb standard alternative in 2020.

Figure 5.1: Total Monetized Benefits (SO_2 and $PM_{2.5}$) of Attaining 50 ppb in 2020*

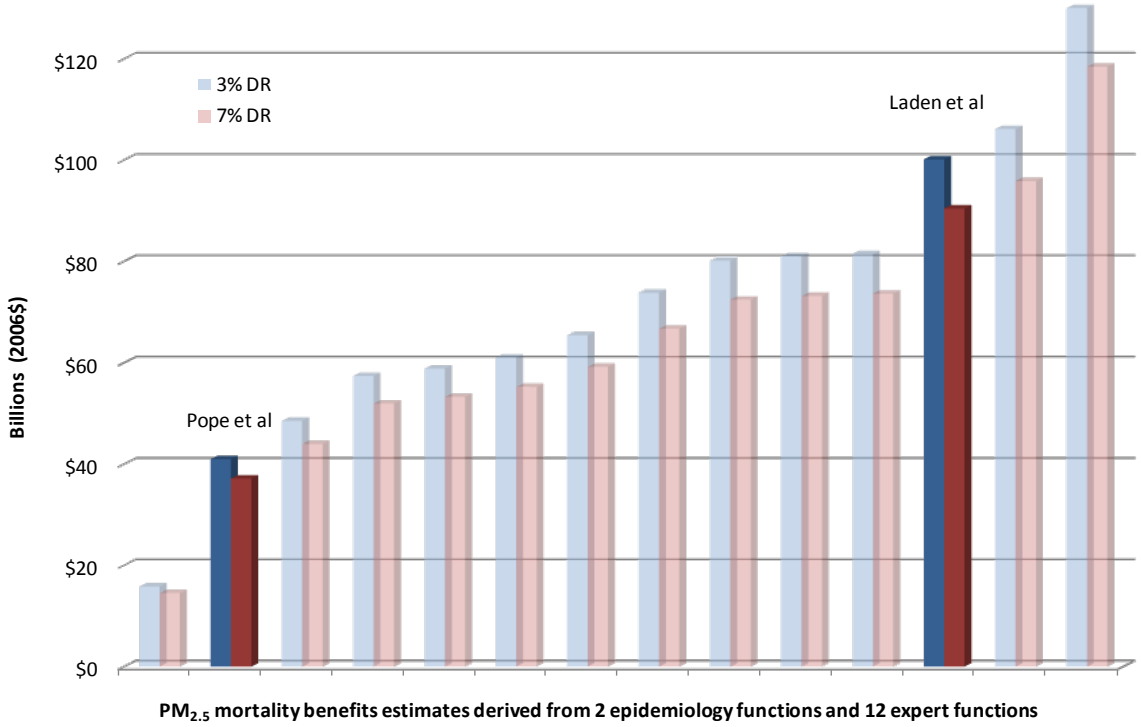
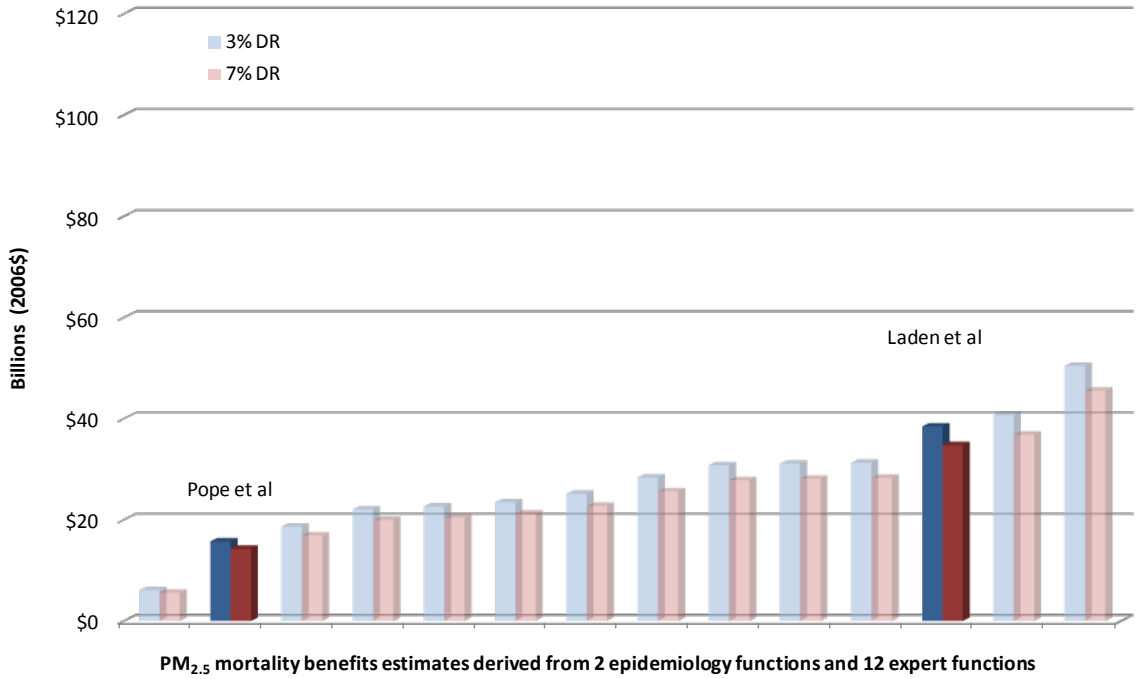


Figure 5.2: Total Monetized Benefits (SO_2 and $PM_{2.5}$) of Attaining 100 ppb in 2020*



*These graphs shows the estimated total monetized benefits in 2020 for the proposed standard range of 50 ppb and 100 ppb using the no-threshold model at discount rates of 3% and 7% using effect coefficients derived from the Pope et al. study and the Laden et al study, as well as 12 effect coefficients derived from EPA's expert elicitation on PM mortality. The results shown are not the direct results from the studies or expert elicitation; rather, the estimates are based in part on the concentration-response function provided in those studies. Graphs for alternative standards at 75 ppb and 150 ppb would show a similar pattern.

5.1 Introduction

This chapter documents our analysis of health benefits expected to result from achieving alternative levels of the SO_2 NAAQS in 2020, relative to baseline ambient concentrations that represent attainment with the 2008 ozone and 2006 $PM_{2.5}$ NAAQS. We first describe our approach for estimating and monetizing the health benefits associated with reductions of SO_2. Next, we provide a summary of our results, including an analysis of the sensitivity of several assumptions in our model. We then estimate the $PM_{2.5}$ co-benefits from controlling SO_2 emissions. Finally, we discuss the key results of the benefits analysis and indicate limitations and areas of uncertainty in our approach.

5.2 Primary Benefits Approach

This section presents our approach for estimating avoided adverse health effects due to SO_2 exposure in humans resulting from achieving alternative levels of the SO_2 NAAQS, relative to a baseline concentration of ambient SO_2. First, we summarize the scientific evidence concerning potential health effects of SO_2 exposure, and then we present the health endpoints we selected for our primary benefits estimate. Next, we describe our benefits model, including the key input data and assumptions. Finally, we describe our approach for assigning an economic value to the SO_2 health benefits. The approach for estimating the benefits associated with exposure to PM is described in section 5.7.

We estimated the economic benefits from annual avoided health effects expected to result from achieving alternative levels of the SO_2 NAAQS (the "control scenarios") in the year 2020. We estimated benefits in the control scenarios relative to the incidence of health effects consistent with the ambient SO_2 concentration expected in 2020 (the "baseline"). Note that this "baseline" reflects emissions reductions and ambient air quality improvements that we anticipate will result from implementation of other air quality rules, including compliance with all relevant rules up to the recently revised NAAQS for ozone in March 2008 (U.S. EPA, 2008a).

We compare benefits across four alternative SO_2 NAAQS levels: 50 ppb, 75 ppb, 100 ppb, and 150 ppb (99^{th} percentile). Consistent with EPA's approach for RIA benefits assessments, we estimate the health effects associated with an incremental difference in ambient concentrations between a baseline scenario and a pollution control strategy. As indicated in Chapter 4, several areas of the country may not be able to attain the alternative standard levels using known pollution control methods. For this reason, we provide an estimate of the benefits associated with partially attaining the standard using known controls

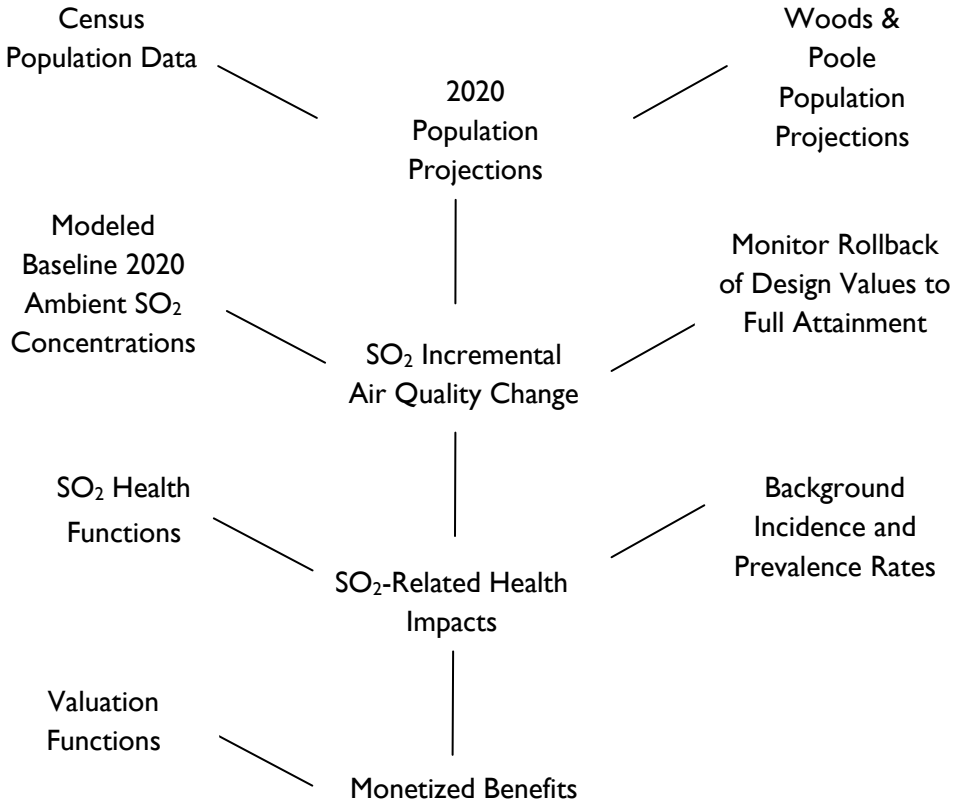
as well as the full attainment results in Table 5.10. Because some areas require substantial emission reductions from unknown sources to attain the various standards, the results are very sensitive to assuming full attainment. All of the other results tables in this chapter assume full attainment with the various alternative standards.

5.3 Overview of analytical framework for benefits analysis

5.3.1 Benefits Model

For the primary benefits analysis, we use the Environmental Benefits Mapping and Analysis Program (BenMAP) (Abt Associates, 2008) to estimate the health benefits occurring as a result of implementing alternative SO_2 NAAQS levels. Although EPA has used BenMAP extensively to estimate the health benefits of reducing exposure to $PM_{2.5}$ and ozone in previous RIAs, this is the first RIA in which EPA has used BenMAP to estimate the health benefits directly attributable to reducing exposure to SO_2. Figure 5.3 below shows the major components of, and data inputs to, the BenMAP model.

Figure 5.3: Diagram of Inputs to BenMAP model for SO_2 Analysis



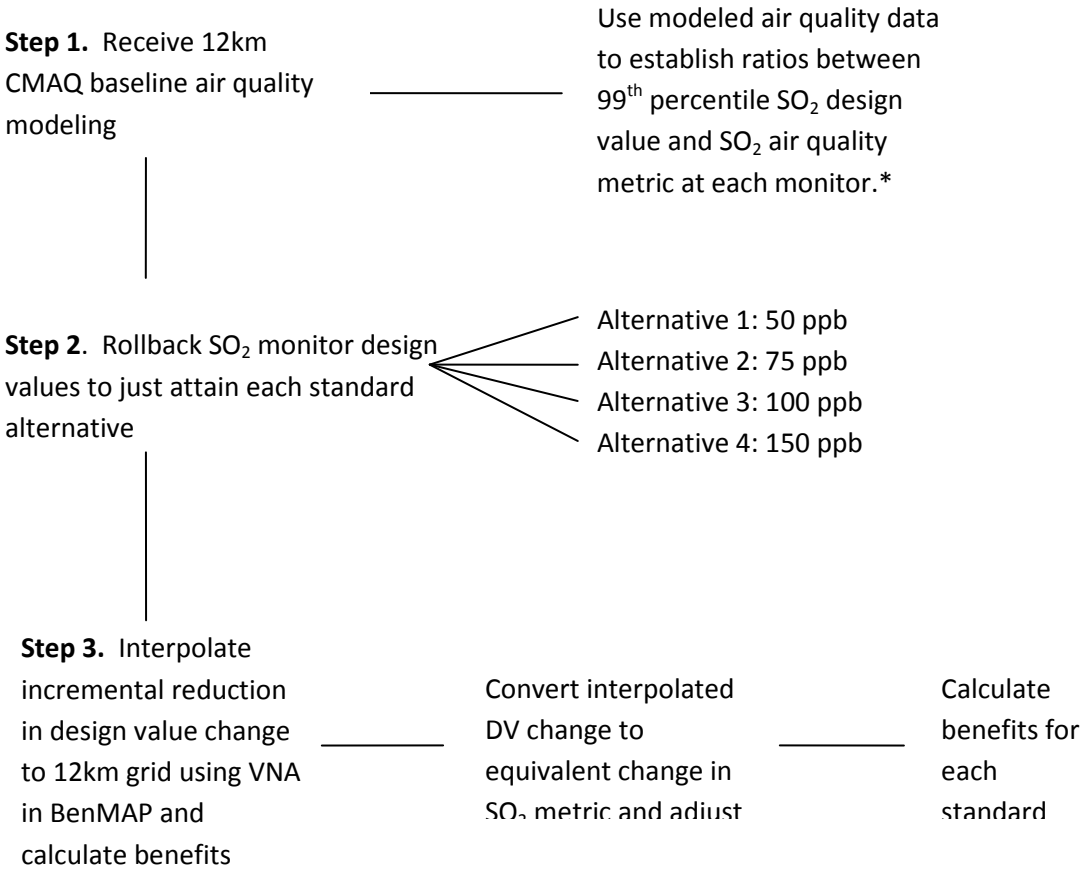
5.3.2 Air Quality Estimates

As Figure 5.3 shows, the primary input to any benefits assessment is the estimated changes in ambient air quality expected to result from a simulated control strategy or attainment of a particular standard. EPA typically relies upon air quality modeling to generate these data, but time and technical limitations described in Chapter 3 prevented us from generating new air quality modeling to simulate the changes in ambient SO_2 resulting from each control strategy. Instead, we utilize the ambient SO_2 concentrations modeled by CMAQ as part of the 2008 Ozone RIA as our baseline.[2]

The CMAQ air quality model provides projects both design values at SO_2 monitors and air quality concentrations at 12km by 12km grid cells nationwide. To estimate the benefits of fully attaining the standards in all areas, EPA employed the "monitor rollback" approach to approximate the air quality change resulting from just attaining alternative SO_2 NAAQS at each design value monitor. Figure 5.4 depicts the rollback process, which differs from the technique described in Chapter 3. The emission control strategy estimated the level of emission reductions necessary to attain each alternate NAAQS standard, whereas the approach described here aims to estimate the change in population exposure associated with attaining an alternate NAAQS. This approach relies on data from the existing SO_2 monitoring network and the inverse distance squared variant of the Veronoi Neighborhood Averaging (VNA) interpolation method to adjust the CMAQ-modeled SO_2 concentrations such that each area just attains the standard alternatives. We believe that the interpolation method using inverse distance squared most appropriately reflects the exposure gradient for SO_2 around each monitor (EPA, 2008c).[3]

[2] See Chapter 3 for more detail regarding the air quality data used in this analysis.
[3] A sensitivity analysis of alternate VNA interpolation methods for the NO_2 NAAQS proposal RIA showed that the results were not sensitive to the interpolation method (U.S. EPA, 2009b).

Figure 5.4: Diagram of Rollback Method



*Metrics used in the epidemiology studies include the 24hr mean, 3hr mean, 8hr max, and 1hr

Because the VNA rollback approach interpolates monitor values, it is most reliable in areas with a denser monitoring network. In areas with a sparser monitoring network, there is less observed monitoring data to support the VNA interpolation and we have less confidence in the predicted air quality values further away from the monitors. For this reason, we interpolated air quality values—and estimated health impacts—within the CMAQ grid cells that are located within 50 km of the monitor, assuming that emission changes within this radius would affect the SO_2 concentration at each monitor. Limiting the interpolation to this radius attempts to account for the limitations of the VNA approach, the air quality data limitations identified in Chapter 3 and ensures that the benefits and costs analyses consider a consistent geographic area.[4] Therefore, the primary benefits analysis assesses health impacts occurring to populations living in the CMAQ grid cells located within the 50km buffer for the specific geographic areas assumed to not attain the alternate standard levels. We test the sensitivity of this assumption relative to other exposure buffers in Table 5.12.

[4] Please see Chapter 3 for more information regarding the technical basis for the 30 km assumption.

5.4 Estimating Avoided Health Effects from SO_2 Exposure

Following an extensive evaluation of health evidence from epidemiologic and laboratory studies, the U.S. EPA has concluded that there is a causal relationship between respiratory health effects and short-term exposure to SO_2 (U.S. EPA, 2008c). The immediate effect of SO_2 on the respiratory system in humans is bronchoconstriction. This response is mediated by chemosensitive receptors in the tracheobronchial tree, which trigger reflexes at the central nervous system level resulting in bronchoconstriction, mucus secretion, mucosal vasodilation, cough, and apnea followed by rapid shallow breathing. In some cases, local nervous system reflexes also may be involved. Asthmatics are more sensitive to the effects of SO_2 likely resulting from preexisting inflammation associated with this disease. This inflammation may lead to enhanced release of mediators, alterations in the autonomic nervous system and/or sensitization of the chemosensitive receptors. These biological processes are likely to underlie the bronchoconstriction and decreased lung function observed in response to SO_2 exposure. A clear concentration-response relationship has been demonstrated in laboratory studies following exposures to SO_2 at concentrations between 20 and 100 ppb, both in terms of increasing severity of effect and percentage of asthmatics adversely affected.

5.4.1 Selection of Health Endpoints for SO_2

Epidemiological researchers have associated SO_2 exposure with adverse health effects in numerous toxicological, clinical and epidemiological studies, as described in the Integrated Science Assessment for Oxides of Sulfur - Health Criteria (Final Report) (U.S. EPA, 2008c); hereafter, "SO_2 ISA"). The SO_2 ISA provides a comprehensive review of the current evidence of health and environmental effects of SO_2.

Previous reviews of the SO_2 primary NAAQS, most recently in 1996, did not include a quantitative benefits assessment for SO_2 exposure. As the first health benefits assessment for SO_2 exposure, we build on the methodology and lessons learned from the SO_2 risk and exposure assessment (U.S. EPA, 2009c) and the benefits assessments for the recent $PM_{2.5,}$ $O_{3,}$ and proposed NO_2 NAAQS (U.S. EPA, 2006a; U.S. EPA, 2008a; U.S. EPA, 2009b).

We selected the health endpoints to be consistent with the conclusions of the SO_2 ISA. 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)). The differing evidence and associated strength of the evidence for these different effects is described in detail in the SO_2 ISA.

Although a number of adverse health effects have been found to be associated with SO_2 exposure, this benefits analysis only includes a subset due to limitations in understanding and quantifying the dose-response relationship for some of these health endpoints. In this analysis, we only estimated the benefits for those endpoints with sufficient evidence to support a quantified concentration-response relationship using the information presented in the SO_2 ISA, which contains an extensive literature review for several health endpoints related to SO_2 exposure. Because the ISA only included studies published or accepted for publication through April 2008, we also performed supplemental literature searches in the online search engine PubMed® to identify relevant studies published between January 2008, and the present.[5] Based on our review of this information, we quantified four short-term respiratory morbidity endpoints that the SO_2 ISA identified as a "causal relationship": acute respiratory symptoms, asthma exacerbation, respiratory-related emergency department visits, and respiratory-related hospitalizations.

Table 5.1 presents the health effects related to SO_2 exposure quantified in this benefits analysis. In addition, the table includes other endpoints potentially linked to SO_2 exposure, but which we are not yet ready to quantify with dose-response functions. For a list of the health effects related to $PM_{2.5}$ exposure that we quantify in this analysis, please see Table 5.6 in section 5.7.

The SO_2 ISA concluded that the relationship between short-term SO_2 exposure and premature mortality was "suggestive of a causal relationship" because it is difficult to attribute the mortality risk effects to SO_2 alone. Therefore, we decided not to quantify premature mortality from SO_2 exposure in this analysis despite evidence suggesting a positive association (U.S. EPA, 2008c). Although the SO_2 ISA stated that studies are generally consistent in reporting a relationship between SO_2 exposure and mortality, there was a lack of robustness of the observed associations to adjustment for co-pollutants. As the literature continues to evolve, we may revisit this decision in future benefits assessment for SO_2.

As noted in Table 5.1, we are not able to quantify several welfare benefit categories in this analysis because we are limited by the available data or resources. Although we cannot

[5] The O'Conner et al. study (2008) is the only study included in this analysis that was published after the cut-off date for inclusion in the SO2 ISA.

quantify the ecosystem benefits of reducing sulfur deposition or visibility improvements in this analysis, we provide a qualitative analysis in section 5.9.

Table 5.1: Human Health and Welfare Effects of SO_2

Pollutant / Effect	Quantified and Monetized in Primary Estimates [a]	Unquantified Effects [b, c] Changes in:
SO_2/Health	Respiratory Hospital Admissions	Premature mortality
	Asthma ER visits	Pulmonary function
	Asthma exacerbation	Other respiratory emergency department visits
	Acute Respiratory symptoms	Other respiratory hospital admissions
SO_2/Welfare		Visibility improvements
		Commercial fishing and forestry from acidic deposition
		Recreation in terrestrial and aquatic ecosystems from acid deposition
		Increased mercury methylation

[a] Primary quantified and monetized effects are those included when determining the primary estimate of total monetized benefits of the alternative standards.
[b] The categorization of unquantified toxic health and welfare effects is not exhaustive.
[c] Health endpoints in the unquantified benefits column include both a) those for which there is not consensus on causality and those for which causality has been determined but empirical data are not available to allow calculation of benefits.

5.4.2 Selection of Concentration-Response Functions

After identifying the health endpoints to quantify in this analysis, we then selected concentration-response functions drawn from the epidemiological literature identified in the SO_2 ISA. We considered several factors, in the order below, in selecting the appropriate epidemiological studies and concentration-response functions for this benefits assessment.

1. We considered ambient SO_2 studies that were identified as key studies in the SO_2 ISA (or a more recent study), excluding those affected by the general additive model (GAM) S-Plus issue.[6]
2. We judged that studies conducted in the United States are preferable to those conducted outside the United States, given the potential for effect estimates to be affected by factors such as the ambient pollutant mix, the placement of monitors, activity patterns of the population, and characteristics of the healthcare system especially for hospital admissions and emergency department visits. We include Canadian studies in sensitivity analyses, when available.

[6] The S-Plus statistical software is widely used for nonlinear regression analysis in time-series research of health effects. However, in 2002, a problem was discovered with the software's default conversion criteria in the general additive model (GAM), which resulted in biased relative risk estimates in many studies. This analysis does not include any studies that encountered this problem. For more information on this issue, please see U.S. EPA (2002).

3. We only incorporated concentration-response functions for which there was a corresponding valuation function. Currently, we only have a valuation function for asthma-related emergency department visits, but we do not have a valuation function for all-respiratory-related emergency department visits.
4. We preferred concentration-response functions that correspond to the age ranges most relevant to the specific health endpoint, with non-overlapping ICD-9 codes. We preferred completeness when selecting functions that correspond to particular age ranges and ICD codes. Age ranges and ICD codes associated with the selected functions are identified in Table 5.2.
5. We preferred multi-city studies or combined multiple single city studies, when available.
6. When available, we judged that effect estimates with distributed or cumulative lag structures were most appropriate for this analysis.
7. When available, we selected SO_2 concentration-response functions based on multi-pollutant models. Studies with multi-pollutant models are identified in Table 5.2.

These criteria reflect our preferences for study selection, and it was possible to satisfy many of these, but not all. There are trade-offs inherent in selecting among a range of studies, as not all studies met all criteria outlined above. At minimum, we ensured that none of the studies were GAM affected, we selected only U.S. based studies, and we quantified health endpoints for which there was a corresponding valuation function.

We believe that U.S.-based studies are most appropriate studies to use in this analysis to estimate the number of hospital admissions associated with SO_2 exposure because of the characteristics of the ambient air, population, and healthcare system. Using only U.S.-based studies, we are limited to one epidemiology study for hospital admissions (Schwartz, 1996). However, there are several Canada-based epidemiology studies that also estimate respiratory hospital admissions (Fung, 2006; Luginaah, 2005; Yang, 2003). Table 5.12 provides the sensitivity of the SO_2 benefits using the effect estimates from the Canadian studies. Compared to the U.S. based study, the Canadian studies produce a substantially larger estimate of hospital admissions associated with SO_2 exposure.

When selecting concentration-response functions to use in this analysis, we reviewed the scientific evidence regarding the presence of thresholds in the concentration-response functions for SO_2 -related health effects to determine whether the function is approximately linear across the relevant concentration range. The SO_2 ISA concluded that, "The overall limited evidence from epidemiologic studies examining the concentration-response function of SO_2

health effects is inconclusive regarding the presence of an effect threshold at current ambient levels." For this reason, we have not incorporated thresholds in the concentration-response functions for SO_2 -related health effects in this analysis.

Table 5.2 shows the studies and health endpoints that we selected for this analysis. Table 5.3 shows the baseline health data used in combination with these health functions. Following these tables is a description of each of the epidemiology studies used in this analysis.

Table 5.2: SO_2 -Related Health Endpoints Quantified, Studies Used to Develop Health Impact Functions and Sub-Populations to which They Apply

Endpoint	Study	Study Population
Hospital Admissions		
All respiratory	Schwartz et al., 1996 – ICD-9 460-519	65 - 99
Emergency Department Visits		
Asthma	Pooled Estimate: Ito et al. (2007)—ICD-9 493 Michaud (2004) – ICD-9 493 NYDOH (2006)[b]—ICD-9 493 Peel et al. (2005)—ICD-9 493 Wilson (2005) – ICD-9 493	All ages
Other Health Endpoints		
Asthma exacerbations	Pooled estimate: Mortimer et al. (2002) (one or more symptoms)[a] O'Connor et al. (2008) (slow play, missed school days[c], nighttime asthma)[a, b] Schildcrout et al. (2006) (one or more symptoms)[a]	4 - 12
Acute Respiratory Symptoms	Schwartz et al. (1994)[b]	7 - 14

[a] The original study populations were 4 to 9 for the Mortimer et al. (2002) study and 5 to 12 for the O'Conner et al. (2008) study and the Schildcrout et al. (2006) study. We extended the applied population to facilitate the pooling process, recognizing the common biological basis for the effect in children in the broader age group. See: National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press, pg 117.

[b] Study specifies a multipollutant model.

[c] The form of this one function is uncertain and that we initially assumed that it was log-linear, but have subsequently determined that it is logistic. This will be fixed in the RIA for the final SO_2 NAAQS.

Table 5.3: National Average Baseline Incidence Rates used to Calculate SO_2 -Related Health Impacts [a]

Endpoint	Source	Notes	Rate per 100 people per year by Age Group						
			<18	18–24	25–34	35–44	45–54	55–64	65+
Respiratory Hospital Admissions	1999 NHDS public use data files [b]	incidence	0.043	0.084	0.206	0.678	1.926	4.389	11.629
Asthma ER visits	2000 NHAMCS public use data files [c]; 1999 NHDS public use data files [b]	incidence	1.011	1.087	0.751	0.438	0.352	0.425	0.232
Minor Restricted Activity Days (MRADs)	Schwartz (1994, table 2)	incidence	0.416	—	—	—	—	—	—
Asthma Exacerbations	Mortimer	Incidence (and prevalence) among asthmatic children	Any morning symptom				0.116 (0.0567) [d]		
	O'Connor et al. (2008)	Incidence (and prevalence) among asthmatic children	Missed school				0.057 (0.0567) [d]		
			One or more symptoms				0.207 (0.0567) [d]		
			Slow play				0.157 (0.0567) [d]		
			Nighttime asthma				0.121 (0.0567) [d]		
	Schildcrout et al. (2006)	Incidence (and prevalence) among asthmatic children	One or more symptoms				0.52 (0.0567) [d]		

[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] We assume that this prevalence rate for ages 5 to 9 is also applicable down to age 4.

Schwartz et al. (1996)

Schwartz et al.(1996) is a review paper with an example drawn from hospital admissions of the elderly in Cleveland, Ohio from 1988-1990. The authors argued that the central issue is control for seasonality. They illustrated the use of categorical variables for weather and sinusoidal terms for filtering season in the Cleveland example. After controlling for season, weather, and day of the week effects, hospital admissions of persons aged 65 and older in Cleveland for respiratory illness was associated with ozone (RR = 1.09, 95% CI 1.02, 1.16) and PM_{10} (RR = 1.12, 95% CI 1.01, 1.24), and marginally associated with SO_2 (RR = 1.03, 95% CI = 0.99, 1.06). All of the relative risks are for a 100 micrograms/m^3 increase in the pollutant.

Fung et al. (2006) – Sensitivity Analysis

Fung et al. (2006) assessed the impact of ambient gaseous pollutants (SO_2, NO_2, CO, and O_3) and particulate matters (PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$) as well as the coefficient of haze (COH) on recurrent respiratory hospital admissions (ICD-9 codes 460-519) among the elderly in Vancouver, Canada, for the period of June 1, 1995, to March 31, 1999, using a new method proposed by Dewanji and Moolgavkar(2000; 2002). The authors found significant associations between respiratory hospital admissions and 3-day, 5-day, and 7-day moving averages of the ambient SO_2 concentrations, with the strongest association observed at the 7-day lag (RR = 1.044, 95% CI: 1.018-1.070). The authors also found $PM_{10-2.5}$ for 3-day and 5-day lag to be significant, with the strongest association at 5-day lag (RR = 1.020, 95% CI: 1.001-1.039). No significant associations with admission were found with current day exposure.

Luginaah et al. (2005) – Sensitivity analysis

Luginaah et al. (2005) assessed the association between air pollution and daily respiratory hospitalization (ICD-9 codes 460-519) for different age and sex groups from 1995 to 2000. The pollutants included were NO_2, SO_2, CO, O_3, PM_{10}, coefficient of haze (COH), and total reduced sulfur (TRS). The authors estimated relative risks (RR) using both time-series and case-crossover methods after controlling for appropriate confounders (temperature, humidity, and change in barometric pressure). The results of both analyses were consistent. They found associations between NO_2, SO_2, CO, COH, or PM_{10} and daily hospital admission of respiratory diseases especially among females. For females 0-14 years of age, there was 1-day delayed effect of NO_2 (RR = 1.19, case-crossover method), a current-day SO_2 (RR = 1.11, time series), and current-day and 1- and 2-day delayed effects for CO by case crossover (RR = 1.15, 1.19, 1.22, respectively). Time-series analysis showed that 1-day delayed effect of PM_{10} on respiratory admissions of adult males (15-64 years of age), with an RR of 1.18. COH had significant effects on female respiratory hospitalization, especially for 2-day delayed effects on adult females, with RRs of 1.15 and 1.29 using time-series and case-crossover analysis, respectively. There were no significant associations between O_3 and TRS with respiratory admissions.

Yang et al. (2003) – Sensitivity analysis

Yang et al. (2003) examined the impact of ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide, and coefficient of haze on daily respiratory admissions (ICD-9 codes 460-519) in both young children (<3 years of age) and the elderly (65-99 years of age) in greater Vancouver, British Columbia during the 13-yr period 1986-1998. Bidirectional case-crossover

analysis was used to investigate associations and odds ratios were reported for single-pollutant, two-pollutant and multiple-pollutant models. Sulfur dioxide was found marginally significant in all models for elderly.

Ito et al. (2007)

Ito et al. (2007) assessed associations between air pollution and asthma emergency department visits in New York City for all ages. Specifically they examined the temporal relationships among air pollution and weather variables in the context of air pollution health effects models. The authors compiled daily data for $PM_{2.5}$, O_3, NO_2, SO_2, CO, temperature, dew point, relative humidity, wind speed, and barometric pressure for New York City for the years 1999-2002.The authors evaluated the relationship between the various pollutants' risk estimates and their respective concurvities, and discuss the limitations that the results imply about the interpretability of multi-pollutant health effects models.

Michaud et al. (2004)

Michaud et al. (2004) examined the association of emergency department (ED) visits in Hilo, Hawai'i, from January 1997 to May 2001 with volcanic fog, or "vog", measured as sulfur dioxide (SO_2) and submicrometer particulate matter (PM_1). Log-linear regression models were used with robust standard errors. The authors studied four diagnostic groups: asthma/COPD; cardiac; flu, cold, and pneumonia; and gastroenteritis. Before adjustments, highly significant associations with vog-related air quality were seen for all diagnostic groups except gastroenteritis. After adjusting for month, year, and day of the week, only asthma/COPD had consistently positive associations with air quality. They found that the strongest associations were for SO_2 with a 3-day lag (6.8% per 10 ppb; P=0.001) and PM_1, with a 1-day lag (13.8% per 10 $\mu g/m^3$; P=0.011).

NYDOH (2006)

New York State Department of Health (NYDOH) investigated whether day-to-day variations in air pollution were associated with asthma emergency department (ED) visits in Manhattan and Bronx, NYC and compared the magnitude of the air pollution effect between the two communities. NYDOH (2006) used Poisson regression to test for effects of 14 key air contaminants on daily ED visits, with control for temporal cycles, temperature, and day-of-week effects. The core analysis utilized the average exposure for the 0- to 4-day lags. Mean daily SO_2 was found significantly associated with asthma ED visits in Bronx but not Manhattan. Their findings of more significant air pollution effects in the Bronx are likely to relate in part to

greater statistical power for identifying effects in the Bronx where baseline ED visits were greater, but they may also reflect greater sensitivity to air pollution effects in the Bronx.

Peel et al. (2005)

Peel et al. (2005) examined the associations between air pollution and respiratory emergency department visits (i.e., asthma (ICD-9 code 493, 786.09), COPD (491,492,496), URI (460-466, 477), pneumonia (480-486), and an all respiratory-disease group) in Atlanta, GA from 1 January 1993 to 31 August 2000. They used 3-Day Moving Average (Lags of 0, 1, and 2 Days) and unconstrained distributed lag (Lags of 0 to 13 Days) in the Poisson regression analyses. In single-pollutant models, positive associations persisted beyond 3 days for several outcomes, and over a week for asthma. The effects of NO_2, CO or PM_{10} on asthma ED visits were found significant but SO_2 or O_3 were not significantly associated with asthma ED visits.

Wilson et al. (2005)

Daily emergency room (ER) visits for all respiratory (ICD-9 codes 460-519) and asthma (ICD-9 code 493) were compared with daily SO_2, O_3, and weather variables over the period 1998-2000 in Portland, Maine and 1996-2000 in Manchester, New Hampshire. Seasonal variability was removed from all variables using nonparametric smoothed function (LOESS). Wilson et al.(2005) used generalized additive models to estimate the effect of elevated levels of pollutants on ER visits. Relative risks of pollutants were reported over their inter-quartile range (IQR, the 75^{th} -25^{th} percentile pollutant values). In Portland, an IQR increase in SO_2 was associated with a 5% (95% CI 2-7%) increase in all respiratory ER visits and a 6% (95% CI 1-12%) increase in asthma visits. An IQR increase in O_3 was associated with a 5% (95% CI 1-10%) increase in Portland asthmatic ER visits. No significant associations were found in Manchester, New Hampshire, possibly due to statistical limitations of analyzing a smaller population. The absence of statistical evidence for a relationship should not be used as evidence of no relationship. This analysis reveals that, on a daily basis, elevated SO_2 and O_3 have a significant impact on public health in Portland, Maine.

Villeneuve et al. (2007) – Sensitivity Analysis

Villeneuve et al. (2007) examined the associations between air pollution and emergency department (ED) visits for asthma among individuals two years of age and older in the census metropolitan area of Edmonton, Canada between April 1, 1992 and March 31, 2002 using a time stratified case-crossover design. Daily air pollution levels for the entire region were estimated from three fixed-site monitoring stations. Odds ratios and their corresponding 95%

confidence intervals were estimated using conditional logistic regression with adjustment for temperature, relative humidity and seasonal epidemic of viral related respiratory disease. Villeneuve et al.(2007) found positive associations for asthma ED visits with outdoor air pollution levels between April and September, but such associations were absent during the remainder of the year. Effects were strongest among young children (2-4 years of age) and elderly (>75 years of age). Air pollution risk estimates were largely unchanged after adjustment for aeroallergen levels. This study is not included in the SO_2 ISA only because it was published after the cut-off date, but it met all of the other criteria for inclusion in this analysis.

Mortimer et al. (2002)

Mortimer et al. (2002) examined the effect of daily ambient air pollution within a cohort of 846 asthmatic children residing in eight urban areas of the USA between June 1 to August 31, 1993, using data from the National Cooperative Inner-City Asthma Study. Daily air pollution concentrations were extracted from the Aerometric Information Retrieval System database from the Environment Protection Agency in the USA. Logistic models were used to evaluate the effects of several air pollutants (O_3, NO_2, SO_2 and PM_{10}) on peak expiratory flow rate (PEFR) and symptoms in 846 children (ages 4-9 yrs) with a history of asthma. In single pollutant models, each pollutant was associated with an increased incidence of morning symptoms: (odds ratio (OR) = 1.16 (95% CI 1.02-1.30) per IQR increase in 4-day average O_3, OR = 1.32 (95% CI 1.03-1.70) per IQR increase in 2-day average SO_2, OR = 1.48 (95% CI 1.02-2.16) per IQR increase in 6-day average NO_2 and OR = 1.26 (95% CI 1.0-1.59) per IQR increase in 2-day average PM_{10}. This longitudinal analysis supports previous time-series findings that at levels below current USA air-quality standards, summer-air pollution is significantly related to symptoms and decreased pulmonary function among children with asthma.

O'Connor et al. (2008)

O'Connor et al. (2008) investigated the association between fluctuations in outdoor air pollution and asthma exacerbation (wheeze-cough, nighttime asthma, slow play and school absence) among 861 inner-city children (5-12 years of age) with asthma in seven US urban communities. Asthma symptom data were collected every 2 months during the 2-year study period. Daily pollution measurements were obtained from the Aerometric Information Retrieval System between August 1998 and July 2001. The relationship of symptoms to fluctuations in pollutant concentrations was examined by using logistic models. In single-pollutant models, significant or nearly significant positive associations were observed between higher NO_2 concentrations and each of the health outcomes. The O_3, $PM_{2.5}$, and SO_2 concentrations did not appear significantly associated with symptoms or school absence except

for a significant association between $PM_{2.5}$ and school absence. This study is not included in the SO_2 ISA only because it was published after the cut-off date, but it met all of the other criteria for inclusion in this analysis.

Schildcrout et al. (2006)

Schildcrout et al. (2006) investigated the relation between ambient concentrations of the five criteria pollutants (PM_{10}, O_3, NO_2, SO_2, and CO) and asthma exacerbations (daily symptoms and use of rescue inhalers) among 990 children in eight North American cities during the 22-month prerandomization phase (November 1993-September 1995) of the Childhood Asthma Management Program. Short-term effects of CO, NO_2, PM_{10}, SO_2, and warm-season O_3 were examined in both one-pollutant and two-pollutant models, using lags of up to 2 days in logistic and Poisson regressions. Lags in CO and NO_2 were positively associated with both measures of asthma exacerbation, and the 3-day moving sum of SO_2 levels was marginally related to asthma symptoms. PM_{10} and O_3 were unrelated to exacerbations. The strongest effects tended to be seen with 2-day lags, where a 1-parts-per-million change in CO and a 20-parts-per-billion change in NO_2 were associated with symptom odds ratios of 1.08 (95% confidence interval (CI): 1.02, 1.15) and 1.09 (95% CI: 1.03, 1.15), respectively.

Schwartz et al. (1994)

Schwartz et al. (1994) studied the association between ambient air pollution exposures and respiratory illness among 1,844 school children (7-14 years of age) in six U.S. cities during five warm season months between April and August. Daily measurements of ambient sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), inhalable particles (PM_{10}), respirable particles ($PM_{2.5}$), light scattering, and sulfate particles were made, along with integrated 24-h measures of aerosol strong acidity. Significant associations in single pollutant models were found between SO_2, NO_2, or $PM_{2.5}$ and incidence of cough, and between sulfur dioxide and incidence of lower respiratory symptoms. Significant associations were also found between incidence of coughing symptoms and incidence of lower respiratory symptoms and PM_{10}, and a marginally significant association between upper respiratory symptoms and PM_{10}.

Delfino et al. (2003) – Sensitivity Analysis

Delfino et al. (2003) conducted a panel study of 22 Hispanic children with asthma who were 10-16 years old and living in a Los Angeles community with high traffic density. Subjects filled out symptom diaries daily for up to 3 months (November 1999 through January 2000). Pollutants included ambient hourly values of ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO) and 24-hr values of volatile organic compounds (VOCs), particulate matter with aerodynamic diameter < 10 micro (PM_{10}), and elemental carbon (EC) and organic carbon (OC) PM_{10} fractions. Asthma symptom severity was regressed on pollutants using logistic models. The authors found positive associations of symptoms with criteria air pollutants (O_3, NO_2, SO_2, and PM_{10}). Selected adjusted odds ratio for more severe asthma symptoms from interquartile range increases in pollutants was, for 2.5 ppb 8-hr max SO_2, 1.36 [95% confidence interval (CI), 1.08-1.71]. Their findings support the view that air toxins in the pollutant mix from traffic and industrial sources may have adverse effects on asthma in children.

5.4.3 Pooling Multiple Health Studies

After selecting which health endpoints to analyze and which epidemiology studies provide appropriate effect estimates, we then selected a method to combine the multiple health studies to provide a single benefits estimate for each health endpoint. The purpose of pooling multiple studies together is to generate a more robust estimate by combining the evidence across multiple studies and cities. Because we used a single study for acute respiratory symptoms and a single study for hospital admission for asthma, there was no pooling necessary for those endpoints.

See Table 5.2 for more information on how the asthma studies were adjusted. Because asthma represents the largest benefits category in this analysis, we tested the sensitivity of the SO_2 benefits to alternate pooling choices in Table 5.12.

5.5 Valuation of Avoided Health Effects from SO_2 Exposure

The selection of valuation functions very similar to the NO_2 proposed NAAQS RIA (U.S. EPA, 2009b) and the $PM_{2.5}$ NAAQS RIA (U.S. EPA, 2006a) with a couple exceptions. First, in this analysis, we estimated changes in all respiratory hospital admissions. This is consistent with the $PM_{2.5}$ NAAQS RIA, but inconsistent with the NO_2 NAAQS RIA, which estimated changes for only a subset of respiratory hospital admissions (i.e., chronic lung disease and asthma) because concentration-response functions were only available for the subset. Second, in this analysis,

we used the any-of-19 symptoms valuation function for acute respiratory symptoms. This is consistent with the NO_2 NAAQS RIA, but inconsistent with the $PM_{2.5}$ NAAQS RIA, which used the valuation function for "minor-restricted activity day" (MRADs). The valuation for any-of-19-symptoms is approximately 50% of the valuation for MRADs. Consistent with economic theory, these valuation functions include adjustments for inflation (2006$) and income growth over time (2020 income levels). Table 5.4 provides the unit values used to monetize the benefits of reduced exposure to SO_2.

Table 5.4: Central Unit Values SO_2 Health Endpoints (2006$)*

Health Endpoint	Central Unit Value Per Statistical Incidence (2020 income level)	Derivation of Distributions of Estimates
Hospital Admissions and ER Visits		
Respiratory Hospital Admissions	$24,000	No distributional information available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Asthma Emergency Room Visits	$370	No distributional information available. Simple average of two unit COI values: (1) $400 (2006$), from Smith et al. (1997) and (2) $340 (2006$), from Stanford et al. (1999).
Respiratory Ailments Not Requiring Hospitalization		
Asthma Exacerbation	$53	Asthma exacerbations are valued at $49 (2006$) per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between $19 and $83 (2006$).
Acute Respiratory Symptoms	$30	The valuation estimate for "any of 19 acute respiratory symptoms" is derived from Krupnick et al. (1990) assuming that this health endpoint consists either of upper respiratory symptoms (URS) or lower respiratory symptoms (LRS), or both. We assumed the following probabilities for a day of "any of 19 acute respiratory symptoms": URS with 40 percent probability, LRS with 40 percent probability, and both with 20 percent probability. The point estimate of WTP to avoid a day of "the presence of any of 19 acute respiratory symptoms" is $28 (2006$). The value is assumed have a uniform distribution between $0 and $56 (2006$).

*All estimates rounded to two significant figures. All values have been inflated to reflect values in 2006 dollars and income levels in 2020.

5.6 Health Benefits of SO_2 Reduction Results

EPA estimated the monetized human health benefits of reducing cases of morbidity among populations exposed to SO_2 in 2020 for each of the alternative standard levels in 2006$. For an SO_2 standard at 50 ppb, the monetized benefits from reduced SO_2 exposure would be $12 million. For an SO_2 standard at 75 ppb, the monetized benefits from reduced SO_2 exposure would be $4.5 million. For an SO_2 standard at 100 ppb, the monetized benefits from reduced SO_2 exposure would be $1.9 million. For an SO_2 standard at 150 ppb, the monetized benefits from reduced SO_2 exposure would be $0.58 million. Figure 5.5 shows the breakdown of the monetized SO_2 benefits by health endpoint. Table 5.5 shows the incidences of health effects and monetized benefits of attaining the alternative standard levels by health endpoint. Because all health effects from SO_2 exposure are expected to occur within the analysis year, the monetized benefits for SO_2 do not need to be discounted. Please note that these benefits do not include any of the benefits listed as "unquantified" in Table 5.1, nor do they include the PM co-benefits, which are presented in the section 5.7.

Figure 5.5: Breakdown of Monetized SO_2 Health Benefits by Endpoint

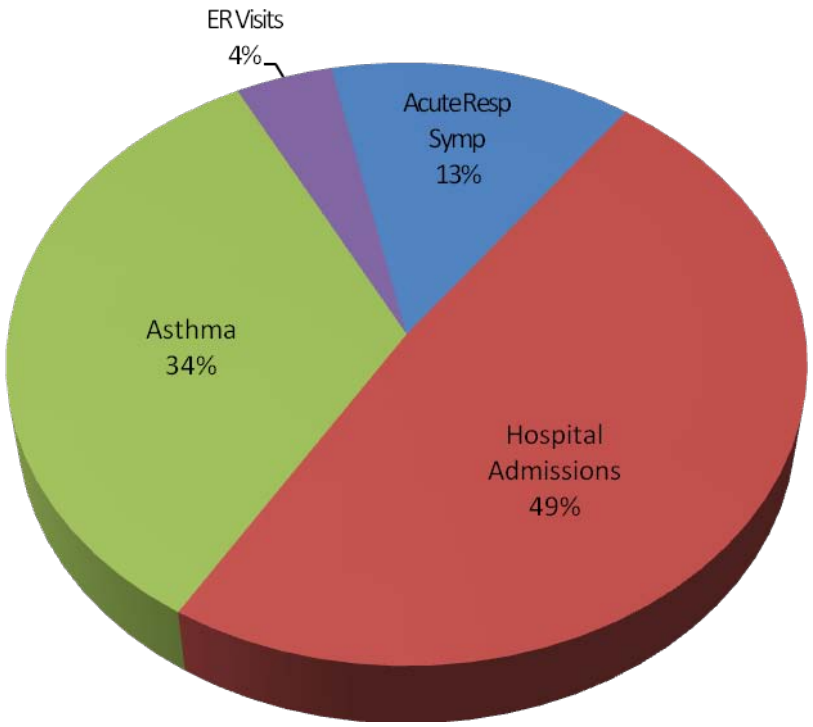


Table 5.5: SO_2 Health Benefits of Attaining Alternate Standard Levels in 2020 in 2006$ (95th percentile confidence interval)

		Incidence			Valuation
50 ppb	Acute Respiratory Symptoms	53,000	(-29,000 -- 130,000)	$1,600,000	(-$1,000,000 -- $5,900,000)
	Hospital Admissions, Respiratory	240	(-15 -- 500)	$5,800,000	($170,000 -- $11,000,000)
	Asthma Exacerbation	74,000	(11,000 -- 180,000)	$4,000,000	($610,000 -- $12,000,000)
	Emergency Room Visits, Respiratory	1,400	(-340 -- 3,900)	$510,000	(-$77,000 -- $1,400,000)
			Total	**$12,000,000**	**(-$300,000 -- $31,000,000)**
75 ppb	Acute Respiratory Symptoms	20,000	(-11,000 -- 50,000)	$590,000	(-$370,000 -- $2,200,000)
	Hospital Admissions, Respiratory	97	(-6 -- 200)	$2,300,000	($69,000 -- $4,500,000)
	Asthma Exacerbation	28,000	(4,100 -- 69,000)	$1,500,000	($230,000 -- $4,500,000)
	Emergency Room Visits, Respiratory	530	(-130 -- 1,500)	$200,000	(-$30,000 -- $540,000)
			Total	**$4,600,000**	**(-$110,000 -- $12,000,000)**
100 ppb	Acute Respiratory Symptoms	8,200	(-4,500 -- 21,000)	$1,600,000	(-$160,000 -- $910,000)
	Hospital Admissions, Respiratory	42	(-3 -- 86)	$5,800,000	($30,000 -- $1,900,000)
	Asthma Exacerbation	12,000	(1,700 -- 29,000)	$4,000,000	($94,000 -- $1,900,000)
	Emergency Room Visits, Respiratory	220	(-55 -- 620)	$510,000	(-$12,000 -- $230,000)
			Total	**$1,900,000**	**(-$44,000 -- $5,000,000)**
150 ppb	Acute Respiratory Symptoms	2,400	(-1,300 -- 6,100)	$72,000	(-$46,000 -- $270,000)
	Hospital Admissions, Respiratory	13	(-1 -- 26)	$300,000	($9,100 -- $590,000)
	Asthma Exacerbation	3,500	(480 -- 8,400)	$180,000	($28,000 -- $550,000)
	Emergency Room Visits, Respiratory	68	(-17 -- 190)	$25,000	(-$3,900 -- $69,000)
			Total	**$580,000**	**(-$13,000 -- $1,500,000)**

*All estimates are rounded to two significant figures. The negative 5th percentile incidence estimates for acute respiratory symptoms are a result of the weak statistical power of the study and should not be inferred to indicate that decreased SO_2 exposure may cause an increase in this health endpoint.

In Table 5.6, we present the results of sensitivity analyses for the SO_2 benefits. We indicate each input parameter, the value used as the default, and the values for the sensitivity analyses, and then we provide the total monetary benefits for each input and the percent change from the default value.

Table 5.6 Sensitivity Analyses for SO_2 Health Benefits to Fully Attain 50 ppb Standard

		Total SO_2 Benefits (millions of 2006$)	% Change from Default
Exposure Estimation Method	50km radius	$12	N/A
	25km radius	$9.3	-21%
	100km radius	$15	26%
	Unconstrained	$22	89%
Location of Hospital Admission Studies	w/US-based studies only	$12	N/A
	w/Canada-based studies only	$62	424%
Asthma Pooling Method	Pool all endpoints together	$12	N/A
	One or more symptoms only	$12	-0.2%

5.7 $PM_{2.5}$ Co-Benefits

Because SO_2 is also a precursor to $PM_{2.5,}$ reducing SO_2 emissions in the projected non-attainment areas will also reduce $PM_{2.5}$ formation, human exposure and the incidence of $PM_{2.5}$-related health effects. In this analysis, we estimated the co-benefits of reducing $PM_{2.5}$ exposure for the alternative standards. Due to analytical limitations, it was not possible to provide a comprehensive estimate of $PM_{2.5}$-related benefits. Instead, we used the "benefit-per-ton" method to estimate these benefits (Fann et al, 2009). Please see Chapter 4 for more information on the tons of emission reductions calculated for the control strategy.[7,8]

The $PM_{2.5}$ benefit-per-ton methodology incorporates key assumptions described in detail below. These $PM_{2.5}$ benefit-per-ton estimates provide the total monetized human health benefits (the sum of premature mortality and premature morbidity) of reducing one ton of $PM_{2.5}$ from a specified source. EPA has used the benefit per-ton technique in previous RIAs, including the recent Ozone NAAQS RIA (U.S. EPA, 2008a) and NO_2 NAAQS RIA (U.S. EPA, 2009b). Table 5.7 shows the quantified and unquantified benefits captured in those benefit-per-ton estimates.

Table 5.7: Human Health and Welfare Effects of $PM_{2.5}$

Pollutant / Effect	Quantified and Monetized in Primary Estimates	Unquantified Effects Changes in:
$PM_{2.5}$	Adult premature mortality	Subchronic bronchitis cases
	Bronchitis: chronic and acute	Low birth weight
	Hospital admissions: respiratory and cardiovascular	Pulmonary function
		Chronic respiratory diseases other than chronic bronchitis
	Emergency room visits for asthma	Non-asthma respiratory emergency room visits
	Nonfatal heart attacks (myocardial infarction)	Visibility
	Lower and upper respiratory illness	Household soiling
	Minor restricted-activity days	
	Work loss days	
	Asthma exacerbations (asthmatic population)	
	Infant mortality	

[7] In addition to reducing SO_2 emissions, the control strategy also reduces direct $PM_{2.5}$ emissions. Please see Table 5.7 for the total estimate of emission reductions used to calculate $PM_{2.5}$ co-benefits.

[8] Pollution controls installed to comply with this proposed standard would also reduce ambient $PM_{2.5}$ concentrations. This illustrative analysis is incremental to the 2006 PM NAAQS, so these benefits are in addition to those estimates for that rule. Furthermore, the controls installed to comply with this proposed standard might also help states attain a more stringent PM NAAQS if one is promulgated in 2011.

Consistent with the Portland Cement NESHAP, the benefits estimates utilize the concentration-response functions as reported in the epidemiology literature, as well as the 12 functions obtained in EPA's expert elicitation study as a sensitivity analysis.

- One estimate is based on the concentration-response (C-R) function developed from the extended analysis of American Cancer Society (ACS) cohort, as reported in Pope et al. (2002), a study that EPA has previously used to generate its primary benefits estimate. When calculating the estimate, EPA applied the effect coefficient as reported in the study without an adjustment for assumed concentration threshold of 10 $\mu g/m^3$ as was done in recent (post-2006) Office of Air and Radiation RIAs.
- One estimate is based on the C-R function developed from the extended analysis of the Harvard Six Cities cohort, as reported by Laden et al (2006). This study, published after the completion of the Staff Paper for the 2006 $PM_{2.5}$ NAAQS, has been used as an alternative estimate in the $PM_{2.5}$ NAAQS RIA and $PM_{2.5}$ co-benefits estimates in RIAs completed since the $PM_{2.5}$ NAAQS. When calculating the estimate, EPA applied the effect coefficient as reported in the study without an adjustment for assumed concentration threshold of 10 $\mu g/m^3$ as was done in recent (post 2006) RIAs.
- Twelve estimates are based on the C-R functions from EPA's expert elicitation study[9,10] on the $PM_{2.5}$-mortality relationship and interpreted for benefits analysis in EPA's final RIA for the $PM_{2.5}$ NAAQS. For that study, twelve experts (labeled A through L) provided independent estimates of the $PM_{2.5}$-mortality concentration-response function. EPA practice has been to develop independent estimates of $PM_{2.5}$-mortality estimates corresponding to the concentration-response function provided by each of the twelve experts, to better characterize the degree of variability in the expert responses.

The effect coefficients are drawn from epidemiology studies examining two large population cohorts: the American Cancer Society cohort (Pope et al., 2002) and the Harvard Six Cities cohort (Laden et al., 2006).[11] These are logical choices for anchor points in our presentation because, while both studies are well designed and peer reviewed, there are strengths and weaknesses inherent in each, which we believe argues for using both studies to generate benefits estimates. Previously, EPA had calculated benefits based on these two empirical studies, but derived the range of benefits, including the minimum and maximum results, from an expert elicitation of the relationship between exposure to $PM_{2.5}$ and premature

[9] Industrial Economics, Inc., 2006. *Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between $PM_{2.5}$ Exposure and Mortality*. Prepared for the U.S. EPA, Office of Air Quality Planning and Standards, September. Available on the Internet at http://www.epa.gov/ttn/ecas/regdata/Uncertainty/pm_ee_report.pdf.

[10] Roman et al., 2008. *Expert Judgment Assessment of the Mortality Impact of Changes in Ambient Fine Particulate Matter in the U.S*. Environ. Sci. Technol., 42, 7, 2268–2274.

[11] These two studies specify multi-pollutant models that control for SO_2, among other co-pollutants.

mortality (Roman et al., 2008). Within this assessment, we include the benefits estimates derived from the concentration-response function provided by each of the twelve experts to better characterize the uncertainty in the concentration-response function for mortality and the degree of variability in the expert responses. Because the experts used these cohort studies to inform their concentration-response functions, benefits estimates using these functions generally fall between results using these epidemiology studies (see Figure 5.9). In general, the expert elicitation results support the conclusion that the benefits of $PM_{2.5}$ control are very likely to be substantial.

Readers interested in reviewing the methodology for creating the benefit-per-ton estimates used in this analysis can consult the Technical Support Document (TSD) accompanying the recent final ozone NAAQS RIA (USEPA 2008a).[12] As described in the documentation for the benefit per-ton estimates cited above, national per-ton estimates are developed for selected pollutant/source category combinations. The per-ton values calculated therefore apply only to tons reduced from those specific pollutant/source combinations (e.g., SO_2 emitted from electric generating units; SO_2 emitted from mobile sources). Our estimate of $PM_{2.5}$ co-control benefits is therefore based on the total $PM_{2.5}$ emissions controlled by sector and multiplied by this per-ton value.

The benefit-per-ton coefficients in this analysis were derived using modified versions of the health impact functions used in the PM NAAQS Regulatory Impact Analysis. Specifically, this analysis uses the benefit-per-ton estimates first applied in the Portland Cement NESHAP RIA (U.S. EPA, 2009a), which incorporated three updates: a new population dataset, an expanded geographic scope of the benefit-per-ton calculation, and the functions directly from the epidemiology studies without an adjustment for an assumed threshold.[13] Removing the threshold assumption is a key difference between the method used in this analysis of PM-co benefits and the methods used in RIAs prior to Portland Cement, and we now calculate incremental benefits down to the lowest modeled $PM_{2.5}$ air quality levels.

EPA strives to use the best available science to support our benefits analyses, and we recognize that interpretation of the science regarding air pollution and health is dynamic and evolving. Based on our review of the body of scientific literature, EPA applied the no-threshold model in this analysis. EPA's draft Integrated Science Assessment (2008e; 2009d), which was recently reviewed by EPA's Clean Air Scientific Advisory Committee (U.S. EPA-SAB, 2009a; U.S. EPA-SAB, 2009b), concluded that the scientific literature consistently finds that a no-threshold

[12] The Technical Support Document (U.S. EPA, 2008b), entitled: Calculating Benefit Per-Ton Estimates, can be found in EPA Docket EPA-HQ-OAR-2007-0225-0284.

[13] The benefit-per-ton estimates have also been updated since the Cement RIA to incorporate a revised VSL, as discussed on the next page.

log-linear model most adequately portrays the PM-mortality concentration-response relationship while recognizing potential uncertainty about the exact shape of the concentration-response function. Although this document does not represent final agency policy that has undergone the full agency scientific review process, it provides a basis for reconsidering the application of thresholds in $PM_{2.5}$ concentration-response functions used in EPA's RIAs. It is important to note that while CASAC provides advice regarding the science associated with setting the National Ambient Air Quality Standards, typically other scientific advisory bodies provide specific advice regarding benefits analysis.[14]

Because the benefits are sensitive to the assumption of a threshold, we also provide a sensitivity analysis using the previous methodology (i.e., a threshold model at 10 $\mu g/m^3$ without the two technical updates) as a historical reference. Table 5.12 shows the sensitivity of an assumed threshold on the monetized results, with and without an assumed threshold at 10 $\mu g/m^3$. Using the threshold model at 10 $\mu g/m^3$ without the two technical updates, we estimate the monetized benefits $27 to $58 billion (2006$, 3 percent discount rate) for the 50 ppb standard alternative, $14 to $31 billion for the 75 ppb standard alternative, $10 to $22 billion for the 100 ppb standard alternative, and $4.2 to $9.0 billion for the 150 ppb standard alternative.[15]

As is the nature of Regulatory Impact Analyses (RIAs), the assumptions and methods used to estimate air quality benefits evolve over time to reflect the Agency's most current interpretation of the scientific and economic literature. For a period of time (2004-2008), the Office of Air and Radiation (OAR) valued mortality risk reductions using a value of statistical life (VSL) estimate derived from a limited analysis of some of the available studies. OAR arrived at a VSL using a range of $1 million to $10 million (2000$) consistent with two meta-analyses of the wage-risk literature. The $1 million value represented the lower end of the interquartile range from the Mrozek and Taylor (2002) meta-analysis of 33 studies. The $10 million value represented the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis of 43 studies. The mean estimate of $5.5 million (2000$)[16] was also consistent with the mean VSL of $5.4 million estimated in the Kochi et al. (2006) meta-analysis. However, the Agency neither changed its official guidance on the use of VSL in rule-makings nor subjected the

[14] In the Portland Cement RIA (U.S. EPA, 2009a), we solicited comment on the use of the no-threshold model for benefits analysis within the preamble of that proposed rule. The comment period for the Portland Cement proposed NESHAP closed on September 4, 2009 (Docket ID No. EPA–HQ–OAR–2002–0051 available at http://www.regulations.gov). EPA is currently reviewing those comments.

[15] Using a 7% discount rate, these results would be approximately 9% lower.

[16] In this analysis, we adjust the VSL to account for a different currency year ($2006) and to account for income growth to 2020. After applying these adjustments to the $5.5 million value, the VSL is $7.7m.

interim estimate to a scientific peer-review process through the Science Advisory Board (SAB) or other peer-review group.

During this time, the Agency continued work to update its guidance on valuing mortality risk reductions, including commissioning a report from meta-analytic experts to evaluate methodological questions raised by EPA and the SAB on combining estimates from the various data sources. In addition, the Agency consulted several times with the Science Advisory Board Environmental Economics Advisory Committee (SAB-EEAC) on the issue. With input from the meta-analytic experts, the SAB-EEAC advised the Agency to update its guidance using specific, appropriate meta-analytic techniques to combine estimates from unique data sources and different studies, including those using different methodologies (i.e., wage-risk and stated preference) (U.S. EPA-SAB, 2007).

Until updated guidance is available, the Agency determined that a single, peer-reviewed estimate applied consistently best reflects the SAB-EEAC advice it has received. Therefore, the Agency has decided to apply the VSL that was vetted and endorsed by the SAB in the Guidelines for Preparing Economic Analyses (U.S. EPA, 2000)[17] while the Agency continues its efforts to update its guidance on this issue. This approach calculates a mean value across VSL estimates derived from 26 labor market and contingent valuation studies published between 1974 and 1991. The mean VSL across these studies is $6.3 million (2000$).[18] The Agency is committed to using scientifically sound, appropriately reviewed evidence in valuing mortality risk reductions and has made significant progress in responding to the SAB-EEAC's specific recommendations. The Agency anticipates presenting results from this effort to the SAB-EEAC in Spring 2010 and that draft guidance will be available shortly thereafter.

Table 5.8 provides the unit values used to monetize the benefits of reduced exposure to $PM_{2.5}$. Figure 5.6 illustrates the relative breakdown of the monetized $PM_{2.5}$ health benefits.

[17] In the (draft) update of the Economic Guidelines (U.S. EPA, 2008), EPA retained the VSL endorsed by the SAB with the understanding that further updates to the mortality risk valuation guidance would be forthcoming in the near future. Therefore, this report does not represent final agency policy.

[18] In this analysis, we adjust the VSL to account for a different currency year ($2006) and to account for income growth to 2020. After applying these adjustments to the $6.3 million value, the VSL is $8.9m.

Table 5.8: Unit Values used for Economic Valuation of $PM_{2.5}$ Health Endpoints (2006$)*

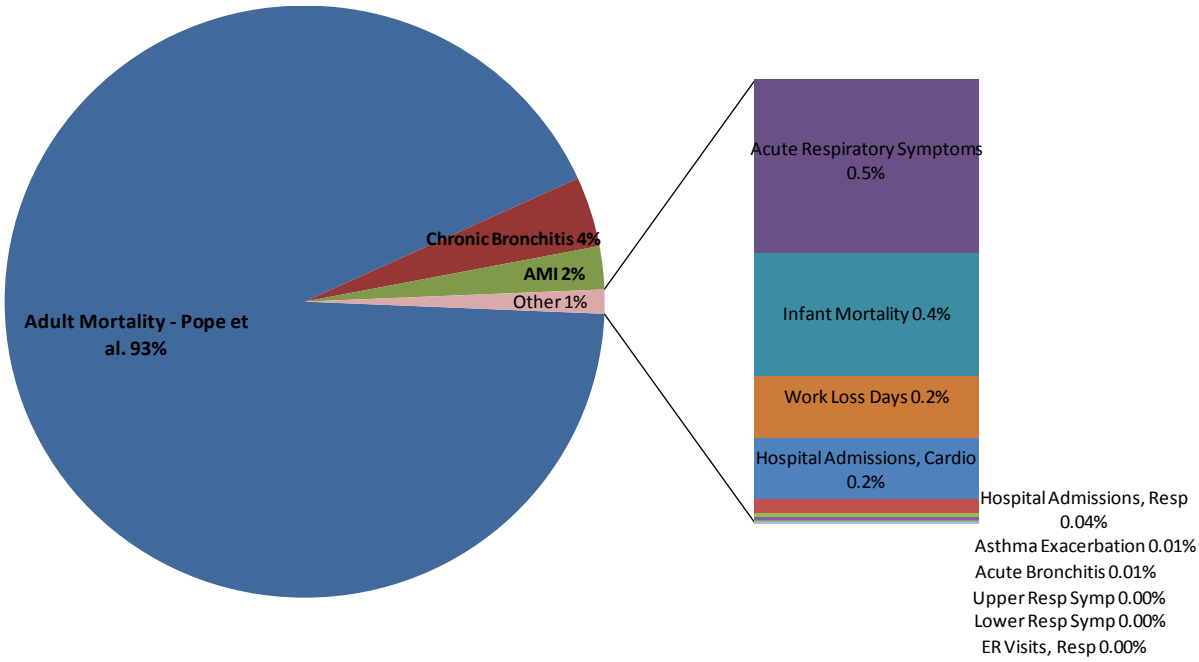
Health Endpoint	Central Estimate of Value Per Statistical Incidence (2020 income level)	Derivation of Distributions of Estimates
Premature Mortality (Value of a Statistical Life)	$8,900,000	EPA currently recommends a central VSL of $6.3m (2000$) based on a Weibull distribution fitted to 26 published VSL estimates (5 contingent valuation and 21 labor market studies). The underlying studies, the distribution parameters, and other useful information are available in Appendix B of EPA's current Guidelines for Preparing Economic Analyses (U.S. EPA, 2000).
Chronic Bronchitis (CB)	$490,000	The WTP to avoid a case of pollution-related CB is calculated as $WTP_x = WTP_{13} * e^{-\beta*(13-x)}$, where x is the severity of an average CB case, WTP13 is the WTP for a severe case of CB, and $ is the parameter relating WTP to severity, based on the regression results reported in Krupnick and Cropper (1992). The distribution of WTP for an average severity-level case of CB was generated by Monte Carlo methods, drawing from each of three distributions: (1) WTP to avoid a severe case of CB is assigned a 1/9 probability of being each of the first nine deciles of the distribution of WTP responses in Viscusi et al. (1991); (2) the severity of a pollution-related case of CB (relative to the case described in the Viscusi study) is assumed to have a triangular distribution, with the most likely value at severity level 6.5 and endpoints at 1.0 and 12.0; and (3) the constant in the elasticity of WTP with respect to severity is normally distributed with mean = 0.18 and standard deviation = 0.0669 (from Krupnick and Cropper [1992]). This process and the rationale for choosing it is described in detail in the Costs and Benefits of the Clean Air Act, 1990 to 2010 (U.S. EPA, 1999).
Nonfatal Myocardial Infarction (heart attack)		No distributional information available. Age-specific cost-of-illness values reflect lost earnings and direct medical costs over a 5-year on period following a nonfatal MI. Lost earnings estimates are based Cropper and Krupnick (1990). Direct medical costs are based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990). Lost earnings: Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings in (2006$): age of onset: at 3%, at 7% 25–44: $11,000, $10,000 45–54: $17,000, $15,000 55–65: $96,000, $86,000 Direct medical expenses: An average of: 1. Wittels et al. (1990) ($130,000—no discounting) 2. Russell et al. (1998), 5-year period ($29,000 at 3%, $27,000 at 7%)
<u>3% discount rate</u>		
Age 0–24	$80,000	
Age 25–44	$96,000	
Age 45–54	$100,000	
Age 55–65	$180,000	
Age 66 and over	$80,000	
<u>7% discount rate</u>		
Age 0–24	$80,000	
Age 25–44	$88,000	
Age 45–54	$92,000	
Age 55–65	$160,000	
Age 66 and over	$78,000	

Hospital Admissions and ER Visits		
Chronic Obstructive Pulmonary Disease (COPD)	$17,000	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
Asthma Admissions	$8,900	No distributional information available. 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	$25,000	No distributional information available. 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).
All respiratory (ages 65+)	$25,000	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
All respiratory (ages 0–2)	$10,000	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Emergency Room Visits for Asthma	$370	No distributional information available. Simple average of two unit COI values: (1) $400 (2006$), from Smith et al. (1997) and (2) $340 (2006$), from Stanford et al. (1999).
Respiratory Ailments Not Requiring Hospitalization		
Upper Respiratory Symptoms (URS)	$31	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) to avoid each symptom in the cluster and assuming additivity of WTPs. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, we assumed a uniform distribution between $11 and $50 (2006$).

Lower Respiratory Symptoms (LRS)	$19	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS. In the absence of information surrounding the frequency with which each of the 11 types of LRS occurs within the LRS symptom complex, we assumed a uniform distribution between $8 and $29 (2006$).
Asthma Exacerbations	$53	Asthma exacerbations are valued at $49 (2006$) per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between $19 and $83 (2006$).
Acute Bronchitis	$440	Assumes a 6-day episode, with the distribution of the daily value specified as uniform with the low and high values based on those recommended for related respiratory symptoms in Neumann et al. (1994). The low daily estimate of $12 (2006$) is the sum of the mid-range values recommended by IEc for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high daily estimate was taken to be twice the value of a minor respiratory restricted-activity day, or $130 (2006$).
Work Loss Days (WLDs)	Variable	No distribution available. Point estimate is based on county-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.
Minor Restricted Activity Days (MRADs)	$63	Median WTP estimate to avoid one MRAD from Tolley et al. (1986). Distribution is assumed to be triangular with a minimum of $26 and a maximum of $97 (2006$). Range is based on assumption that value should exceed WTP for a single mild symptom (the highest estimate for a single symptom—for eye irritation—is $19 (2006$)) and be less than that for a WLD. The triangular distribution acknowledges that the actual value is likely to be closer to the point estimate than either extreme.

*All estimates rounded to two significant figures. All values have been inflated to reflect values in 2006 dollars.

Figure 5.6: Breakdown of Monetized $PM_{2.5}$ Health Benefits using Mortality Function from Pope et al.*



*This pie chart is an illustrative breakdown of the monetized PM co-benefits, using the results based on Pope et al. (2002) as an example. Using the Laden et al. (2006) function for premature mortality, the percentage of total monetized benefits due to adult mortality would be 97%. This chart shows the breakdown using a 3% discount rate, and the results would be similar if a 7% discount rate was used.

Because epidemiology studies have indicated that there is a lag between exposure to $PM_{2.5}$ and premature mortality, the discount rate has a substantial effect on the final monetized benefits. We provide the PM co-benefit results using both discount rates in Table 5.11 and the total monetized benefits (i.e., SO_2 and PM) results using both discount rates in Table 5.13. We test the sensitivity of the PM results to discount rates of 3% and 7% in Table 5.12.

The benefit-per-ton estimates are provided in Table 5.9 and the health incidences are provided in Table 5.10. Higher or lower estimates of benefits are possible using other assumptions; examples of this are provided for the proposed standard range of 50 ppb and 100 ppb in Figures 5.10 and 5.11. Table 5.11 shows the monetized results using the two epidemiology-based estimates as well as the 12 expert-based estimates. Figure 5.8 provides a graphical breakdown of the $PM_{2.5}$ co-benefits by sector. Figure 5.9 provides a graphical representation of all 14 of the $PM_{2.5}$ co-benefits, at both a 3 percent and 7 percent discount rate.

Table 5.9: $PM_{2.5}$ Co-benefits associated with reducing SO_2 emissions (2006$)*

$PM_{2.5}$ Precursor	Benefit per Ton Estimate (Pope)	Benefit per Ton Estimate (Laden)
SO_2 EGU:	$42,000	$100,000
SO_2 non-EGU:	$30,000	$74,000
SO_2 area:	$19,000	$47,000
NO_2 EGU	$7,600	$19,000
NO_2 non-EGU	$5,000	$12,000
Direct $PM_{2.5}$:	$230,000	$570,000

*Estimates have been rounded to two significant figures. This table includes extrapolated tons, spread across the sectors in proportion to the emissions in the county. Confidence intervals are not available for benefit per-ton estimates. Estimates shown use a 3% discount rate. Estimates at a 7% discount rate would be approximately 9% lower.

Table 5.10. Summary of Reductions in Health Incidences from $PM_{2.5}$ Co-Benefits to Attain Alternate Standard Levels in 2020*

	50 ppb	75 ppb	100 ppb	150 ppb
Avoided Premature Mortality				
Pope	4,700	2,500	1,800	740
Laden	12,000	6,400	4,600	1,900
Woodruff (Infant Mortality)	18	10	7	3
Avoided Morbidity	3,200	1,700	1,200	490
Chronic Bronchitis	7,900	4,200	3,000	1,200
Acute Myocardial Infarction	1,200	640	460	190
Hospital Admissions, Respiratory	2,600	1,400	1,000	410
Hospital Admissions, Cardiovascular	4,600	2,500	1,800	720
Emergency Room Visits, Respiratory	7,400	3,900	2,800	1,200
Acute Bronchitis	590,000	310,000	230,000	92,000
Work Loss Days	81,000	43,000	31,000	13,000
Asthma Exacerbation	3,500,000	1,900,000	1,300,000	540,000
Acute Respiratory Symptoms	88,000	47,000	34,000	14,000
Lower Respiratory Symptoms	67,000	36,000	26,000	10,000
Upper Respiratory Symptoms	13,000	6,800	4,900	2,000

*All estimates are for the analysis year (2020) and are rounded to two significant figures. All fine particles are assumed to have equivalent health effects, but each $PM_{2.5}$ precursor pollutant has a different propensity to form $PM_{2.5}$.

Table 5.11: All $PM_{2.5}$ Co-Benefits Estimates to Attain Alternate Standard Levels in 2020 at discount rates of 3% and 7% (in millions of 2006$)*

	50 ppb		75 ppb		100 ppb		150 ppb	
	3%	7%	3%	7%	3%	7%	3%	7%
Benefit-per-ton Coefficients Derived from Epidemiology Literature								
Pope et al.	$41,000	$37,000	$22,000	$20,000	$16,000	$14,000	$6,400	$5,800
Laden et al.	$100,000	$90,000	$53,000	$48,000	$38,000	$35,000	$16,000	$14,000
Benefit-per-ton Coefficients Derived from Expert Elicitation								
Expert A	$110,000	$96,000	$57,000	$51,000	$41,000	$37,000	$17,000	$15,000
Expert B	$81,000	$74,000	$43,000	$39,000	$31,000	$28,000	$13,000	$11,000
Expert C	$81,000	$73,000	$43,000	$39,000	$31,000	$28,000	$13,000	$11,000
Expert D	$57,000	$52,000	$31,000	$28,000	$22,000	$20,000	$9,000	$8,100
Expert E	$130,000	$120,000	$70,000	$63,000	$50,000	$45,000	$20,000	$18,000
Expert F	$74,000	$67,000	$39,000	$36,000	$28,000	$26,000	$12,000	$10,000
Expert G	$49,000	$44,000	$26,000	$23,000	$19,000	$17,000	$7,600	$6,900
Expert H	$61,000	$55,000	$33,000	$29,000	$23,000	$21,000	$9,500	$8,600
Expert I	$80,000	$72,000	$43,000	$39,000	$31,000	$28,000	$13,000	$11,000
Expert J	$65,000	$59,000	$35,000	$32,000	$25,000	$23,000	$10,000	$9,200
Expert K	$16,000	$15,000	$8,400	$7,700	$6,100	$5,600	$2,500	$2,300
Expert L	$59,000	$53,000	$31,000	$28,000	$23,000	$20,000	$9,200	$8,300

*All estimates are rounded to two significant figures. Estimates do not include confidence intervals because they were derived through the benefit-per-ton technique described above. The benefits estimates from the Expert Elicitation are provided as a reasonable characterization of the uncertainty in the mortality estimates associated with the concentration-response function.

In Table 5.12, we present the results of sensitivity analyses for the PM co-benefits. We indicate each input parameter, the value used as the default, and the values for the sensitivity analyses, and then we provide the total monetary benefits for each input and the percent change from the default value.

Table 5.12: Sensitivity Analyses for $PM_{2.5}$ Health Co-Benefits for an Alternative Standard SO_2 at 50 ppb

		Total $PM_{2.5}$ Benefits (billions of 2006$)	% Change from Default
Threshold Assumption (with Epidemiology Study)	No Threshold (Pope)	$38	N/A
	No Threshold (Laden)	$93	N/A
	Threshold (Pope)*	$25	-34%
	Threshold (Laden)*	$54	-42%
Discount Rate (with Epidemiology Study)	3% (Pope)	$38	N/A
	3% (Laden)	$93	N/A
	7% (Pope)	$35	-9%
	7% (Laden)	$84	-10%
Simulated Attainment (using Pope)	Full attainment	$38	N/A
	Partial Attainment	$29	-29%

* The threshold model is not directly comparable to the no-threshold model. The threshold estimates do not include two technical updates, and they are based on data for 2015, instead of 2020. Directly comparable estimates are not available.

Figure 5.8: Monetized $PM_{2.5}$ Co-Benefits of Fully Attaining 50 ppb by $PM_{2.5}$ Precursor

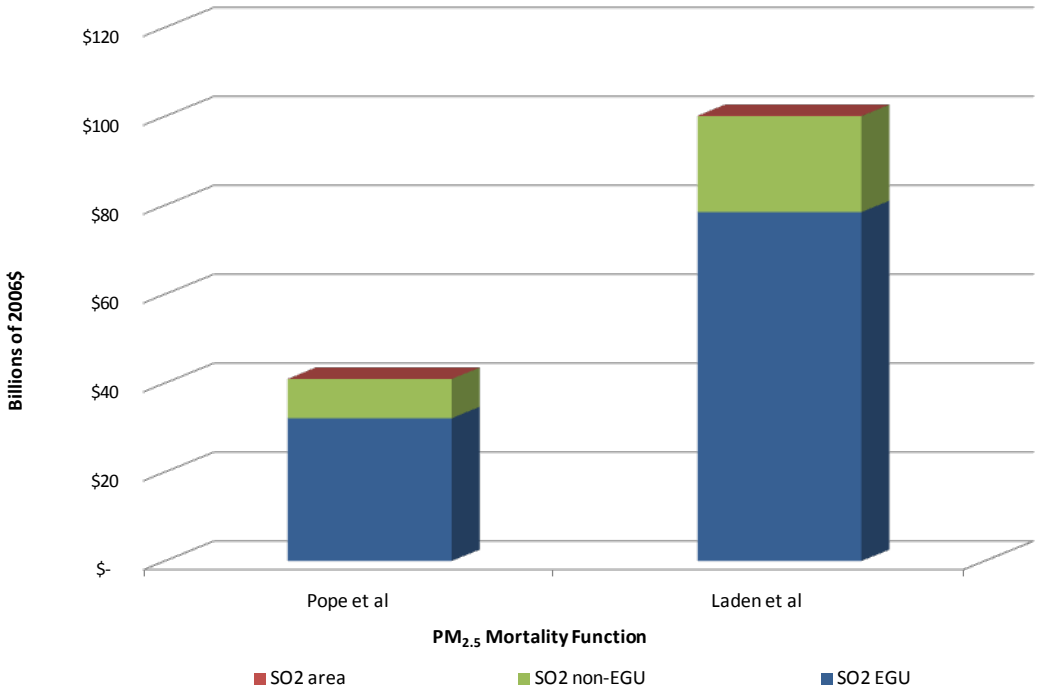
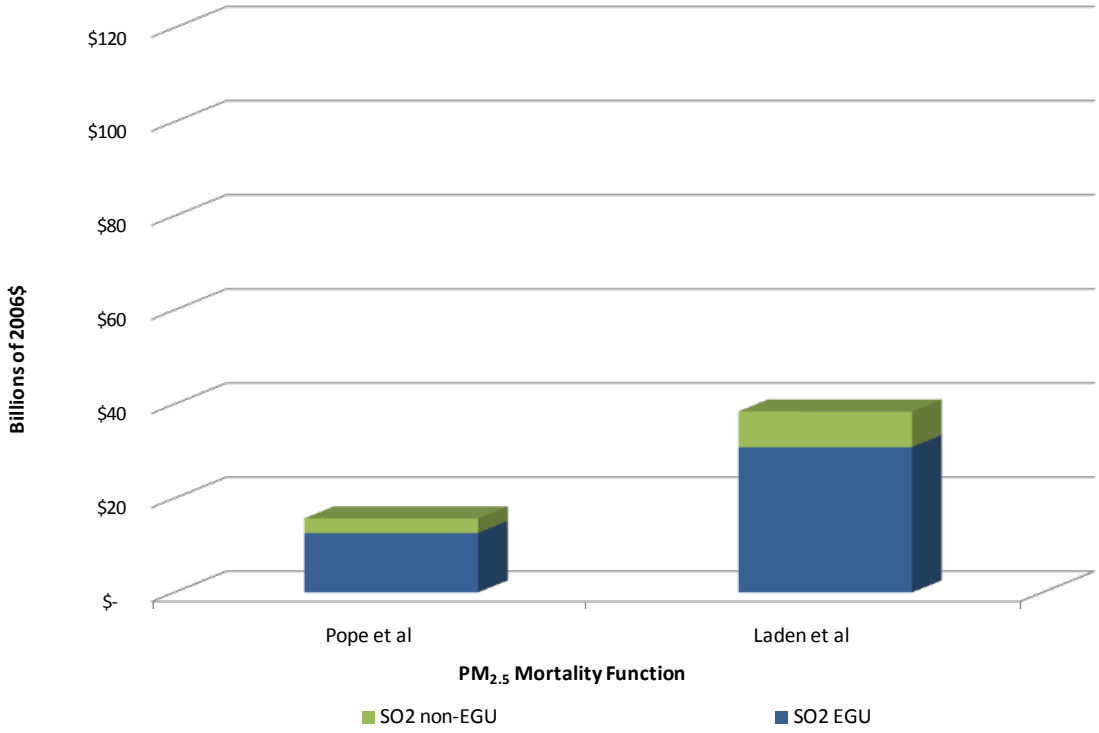


Figure 5.9: Monetized $PM_{2.5}$ Co-Benefits of Fully Attaining 100 ppb by $PM_{2.5}$ Precursor



* All estimates are for the analysis year (2020). All fine particles are assumed to have equivalent health effects, but each $PM_{2.5}$ precursor pollutant has a different propensity to form $PM_{2.5}$. Results using a 7% discount rate would show a similar breakdown.

Figure 5.10: Monetized $PM_{2.5}$ Co-Benefits of Attaining 50 ppb*

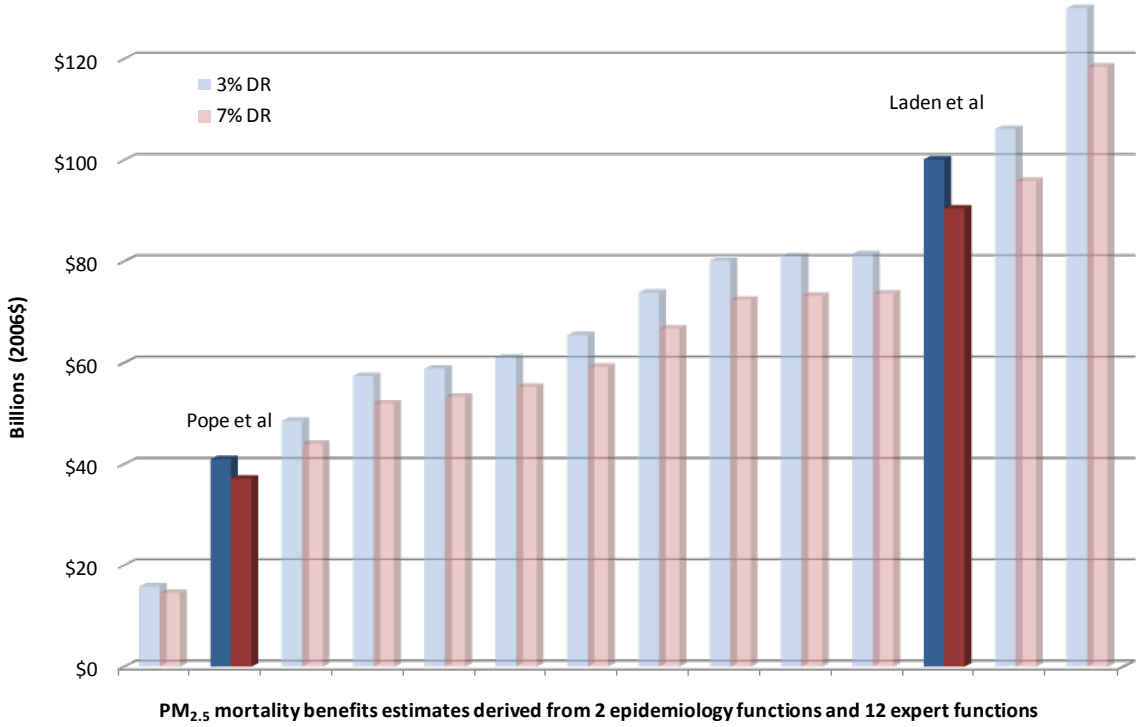
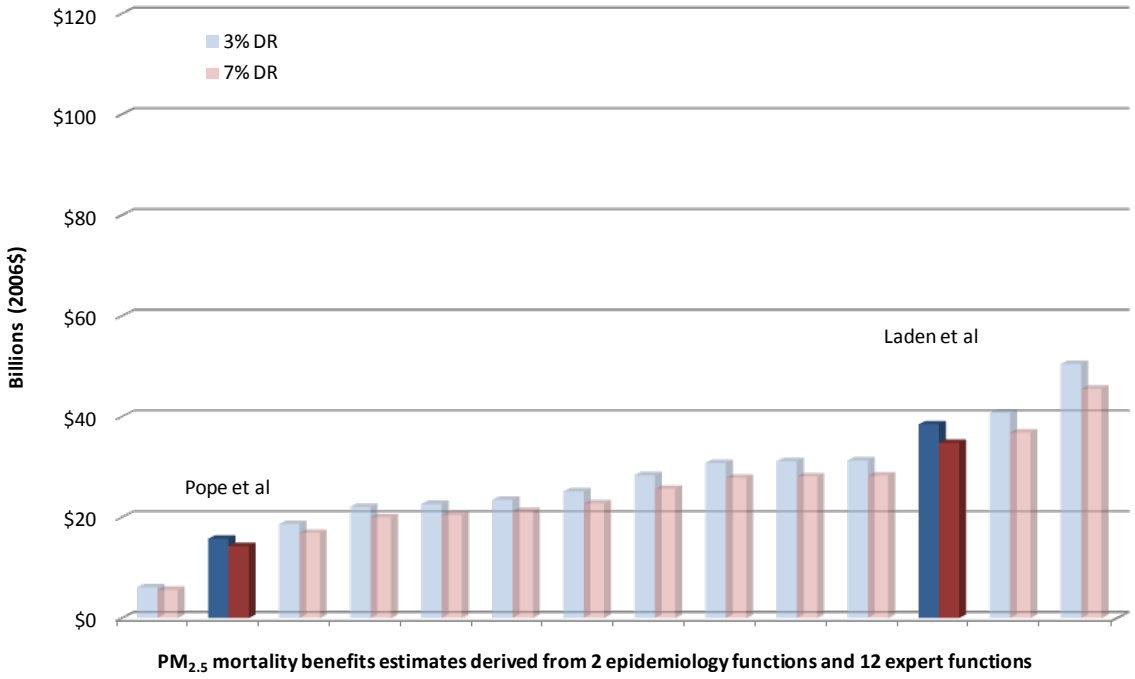


Figure 5.11: Monetized $PM_{2.5}$ Co-Benefits of Attaining 100 ppb*



* These graphs shows the estimated co- benefits in 2020 for the proposed standard range of 50 ppb and 100 ppb using the no-threshold model at discount rates of 3% and 7% using effect coefficients derived from the Pope et al. study and the Laden et al study, as well as 12 effect coefficients derived from EPA's expert elicitation on PM mortality. The results shown are not the direct results from the studies or expert elicitation; rather, the estimates are based in part on the concentration-response function provided in those studies. Graphs for alternative standards at 75 ppb and 150 ppb would show a similar pattern.

5.8 Summary of Total Monetized Benefits (SO_2 and $PM_{2.5}$)

EPA estimated the monetized human health benefits of reducing cases of morbidity and premature mortality among populations exposed to SO_2 and $PM_{2.5}$ in 2020 for each of the alternative standard levels in 2006$. For an SO_2 standard at 50 ppb, the total monetized benefits would be $41 to $100 billion at a 3% discount rate and $37 to $90 billion at a 7% discount rate. For an SO_2 standard at 75 ppb, the total monetized benefits would be $22 to $53 billion at a 3% discount rate and $20 to $48 billion at a 7% discount rate. For an SO_2 standard at 100 ppb, the total monetized benefits would be $16 to $38 billion at a 3% discount rate and $14 to $35 billion at a 7% discount rate. For an SO_2 standard at 150 ppb, the total monetized benefits would be $6.4 to $16 billion at a 3% discount rate and $5.8 to $14 billion at a 7% discount rate.

All of the results in this chapter present benefits estimates that assume full attainment with the alternative standard levels. Partial attainment only incorporates the emission reductions from identified controls without the extrapolated emission reductions.[19] These results are shown in Table 5.13 along with the full attainment at discount rates of 3% and 7%. Table 5.14 shows the total incidences of avoided health effects. Figures 5.12 and 5.13 provides a graphical representation of all 14 total monetized benefits estimates, at both a 3 percent and 7 percent discount rate, for the proposed standard range of 50 ppb to 100 ppb, respectively. Figures for alternative standards at 75 ppb and 150 ppb would show a similar pattern.

[19] See Chapter 4 for more information regarding the control strategy, including the identified and extrapolated emission reductions.

Table 5.13: Total Monetized Benefits to attain Alternate Standard Levels at Discount Rates of 3% and 7% for Full and Partial Attainment (millions of 2006$)*

		SO_2	$PM_{2.5}$ (Pope et al)	$PM_{2.5}$ (Laden et al)	TOTAL (with Pope)	TOTAL (with Laden)
50 ppb	3% Full Attainment	$12	$41,000	$100,000	$41,000	$100,000
	7% Full Attainment	$12	$37,000	$90,000	$37,000	$90,000
	3% Partial Attainment	$12	$29,000	$76,000	$29,000	$76,000
	7% Partial Attainment	$12	$27,000	$69,000	$27,000	$69,000
75 ppb	3% Full Attainment	$4.6	$22,000	$53,000	$22,000	$53,000
	7% Full Attainment	$4.6	$20,000	$48,000	$20,000	$48,000
	3% Partial Attainment	$4.6	$17,000	$41,000	$17,000	$41,000
	7% Partial Attainment	$4.6	$15,000	$37,000	$15,000	$37,000
100 ppb	3% Full Attainment	$1.9	$16,000	$38,000	$16,000	$38,000
	7% Full Attainment	$1.9	$14,000	$35,000	$14,000	$35,000
	3% Partial Attainment	$1.9	$13,000	$33,000	$13,000	$33,000
	7% Partial Attainment	$1.9	$12,000	$29,000	$12,000	$29,000
150 ppb	3% Full Attainment	$0.6	$6,400	$16,000	$6,400	$16,000
	7% Full Attainment	$0.6	$5,800	$14,000	$5,800	$14,000
	3% Partial Attainment	$0.6	$6,300	$15,000	$6,300	$15,000
	7% Partial Attainment	$0.6	$5,700	$14,000	$5,700	$14,000

*Estimates have been rounded to two significant figures and therefore summation may not match table estimates.

Table 5.14: Summary of Reductions in Health Incidences from SO_2 and $PM_{2.5}$ to attain Alternate Standard Levels*

	50 ppb	75 ppb	100 ppb	150 ppb
Avoided Premature Mortality				
Pope	4,700	2,500	1,800	740
Laden	12,000	6,400	4,600	1,900
Woodruff (Infant Mortality)	18	10	7	3
Avoided Morbidity				
Chronic Bronchitis	7,900	4,200	3,000	1,200
Acute Myocardial Infarction	1,200	640	460	190
Hospital Admissions, Respiratory	2,900	1,500	1,000	410
Hospital Admissions, Cardiovascular	4,600	2,500	1,800	720
Emergency Room Visits, Respiratory	590,000	310,000	230,000	92,000
Acute Bronchitis	81,000	43,000	31,000	13,000
Work Loss Days	3,500,000	1,900,000	1,300,000	540,000
Asthma Exacerbation	3,600,000	1,900,000	1,300,000	540,000
Acute Respiratory Symptoms	140,000	67,000	42,000	14,000
Lower Respiratory Symptoms	67,000	36,000	26,000	10,000
Upper Respiratory Symptoms	13,000	6,800	4,900	2,000

*All estimates are for the analysis year (2020) and are rounded to two significant figures.

Figure 5.12: Total Monetized Benefits (SO_2 and $PM_{2.5}$) of Attaining 50 ppb in 2020*

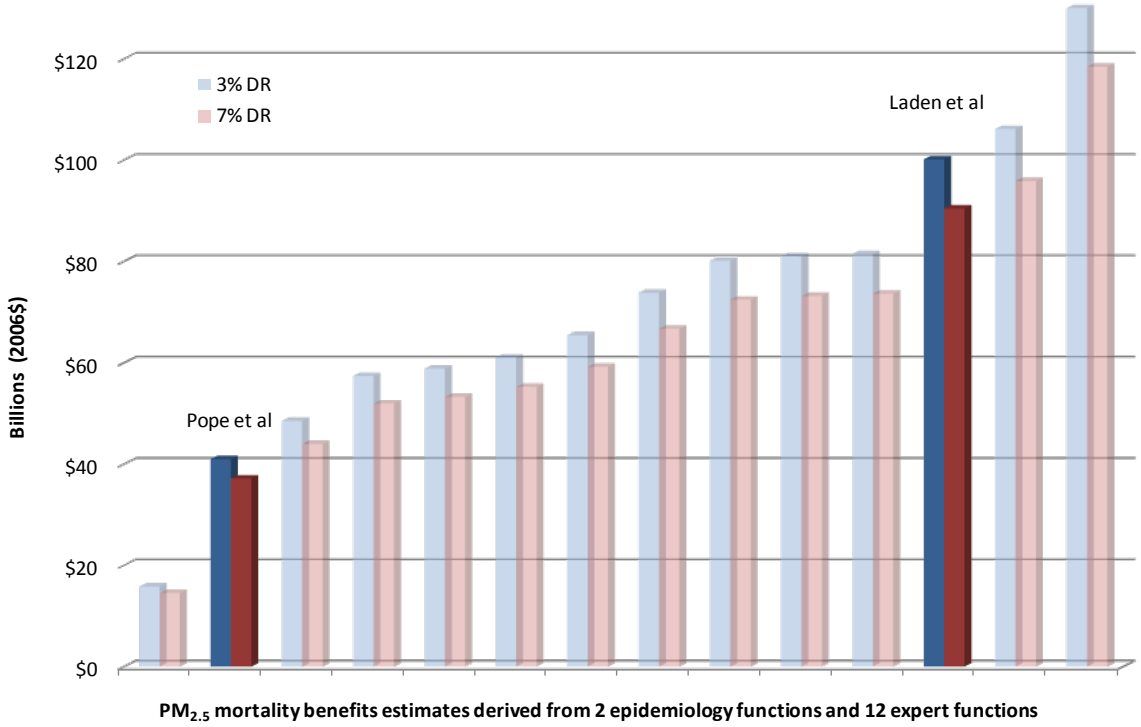
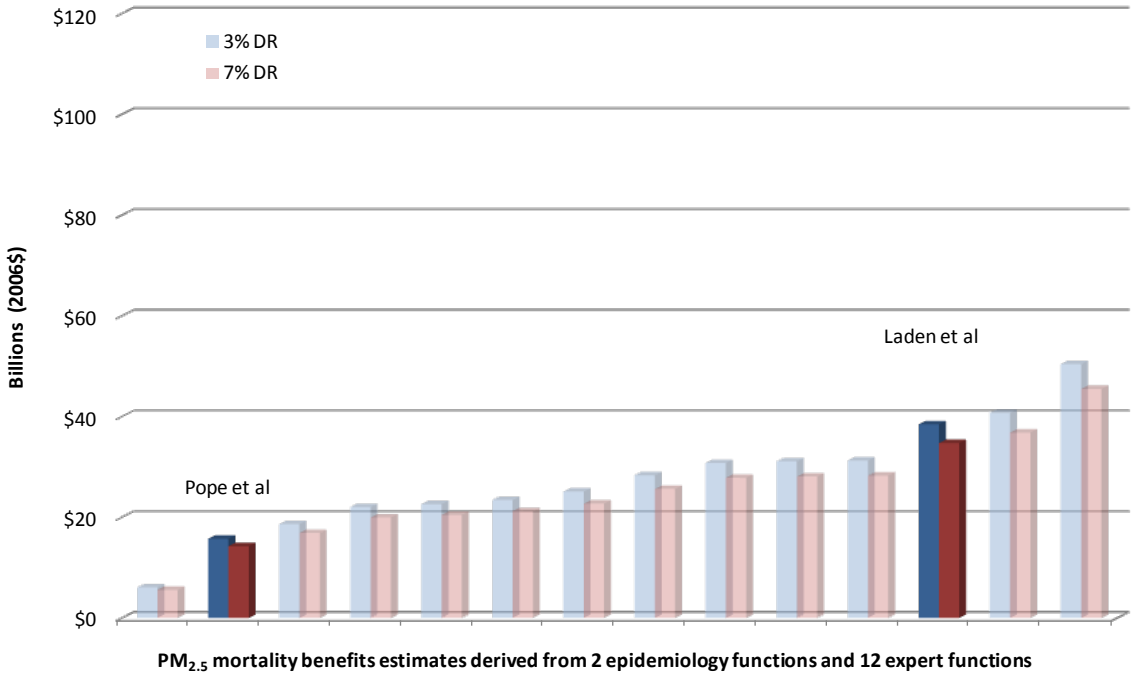


Figure 5.13: Total Monetized Benefits (SO_2 and $PM_{2.5}$) of Attaining 100 ppb in 2020*



* These graphs shows the estimated total monetized benefits in 2020 for the proposed standard range of 50 ppb and 100 ppb using the no-threshold model at discount rates of 3% and 7% using effect coefficients derived from the Pope et al. study and the Laden et al study, as well as 12 effect coefficients derived from EPA's expert elicitation on PM mortality. The results shown are not the direct results from the studies or expert elicitation; rather, the estimates are based in part on the concentration-response function provided in those studies. Graphs for alternative standards at 75 ppb and 150 ppb would show a similar pattern.

5.9 Unquantified Welfare Benefits

This analysis is limited by the available data and resources. As such, we are not able to quantify several welfare benefit categories in this analysis because we are limited by the available data or resources. In this section, we provide a qualitative assessment of the two largest welfare benefit categories: ecosystem benefits of reducing sulfur deposition and visibility improvements.

5.9.1 Ecosystem Benefits of Reduced Sulfur Deposition

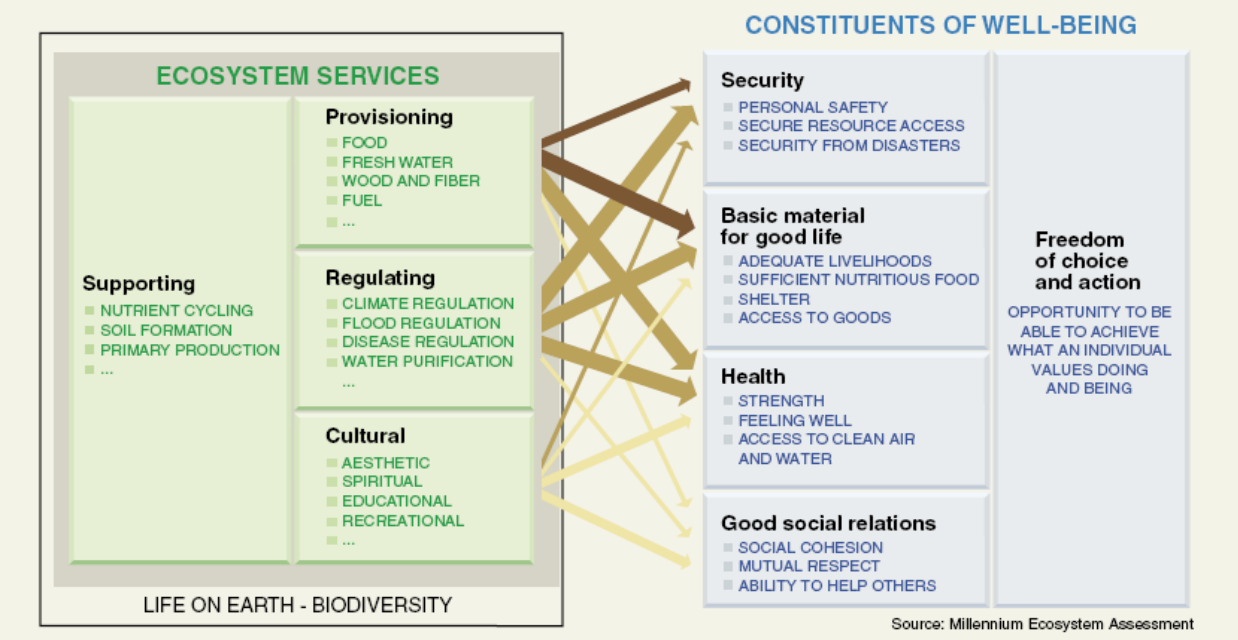
Ecosystem Services

Ecosystem services can be generally defined as the benefits that individuals and organizations obtain from ecosystems. EPA has defined ecological goods and services as the "outputs of ecological functions or processes that directly or indirectly contribute to social welfare or have the potential to do so in the future. Some outputs may be bought and sold, but most are not marketed" (U.S. EPA, 2006c). Figure 5.14 provides the World Resources Institute's schematic demonstrating the connections between the categories of ecosystem services and human well-being. The interrelatedness of these categories means that any one ecosystem may provide multiple services. Changes in these services can affect human well-being by affecting security, health, social relationships, and access to basic material goods (MEA, 2005).

In the Millennium Ecosystem Assessment (MEA, 2005), ecosystem services are classified into four main categories:

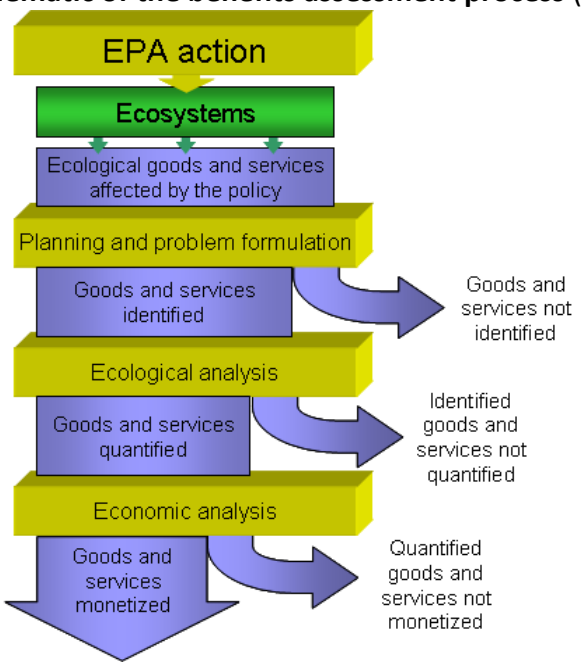
1. Provisioning: Products obtained from ecosystems, such as the production of food and water
2. Regulating: Benefits obtained from the regulation of ecosystem processes, such as the control of climate and disease
3. Cultural: Nonmaterial benefits that people obtain from ecosystems through spiritual enrichment, cognitive development, reflection, recreation, and aesthetic experiences
4. Supporting: Services necessary for the production of all other ecosystem services, such as nutrient cycles and crop pollination

Figure 5.14. Linkages between categories of ecosystem services and components of human well-being from Millennium Ecosystem Assessment (MEA, 2005)



The monetization of ecosystem services generally involves estimating the value of ecological goods and services based on what people are willing to pay (WTP) to increase ecological services or by what people are willing to accept (WTA) in compensation for reductions in them (U.S. EPA, 2006c). There are three primary approaches for estimating the monetary value of ecosystem services: market-based approaches, revealed preference methods, and stated preference methods (U.S. EPA, 2006c). Because economic valuation of ecosystem services can be difficult, nonmonetary valuation using biophysical measurements and concepts also can be used. An example of a nonmonetary valuation method is the use of relative-value indicators (e.g., a flow chart indicating uses of a water body, such as boatable, fishable, swimmable, etc.). It is necessary to recognize that in the analysis of the environmental responses associated with any particular policy or environmental management action, only a subset of the ecosystem services likely to be affected are readily identified. Of those ecosystem services that are identified, only a subset of the changes can be quantified. Within those services whose changes can be quantified, only a few will likely be monetized, and many will remain nonmonetized. The stepwise concept leading up to the valuation of ecosystems services is graphically depicted in Figure 5.15.

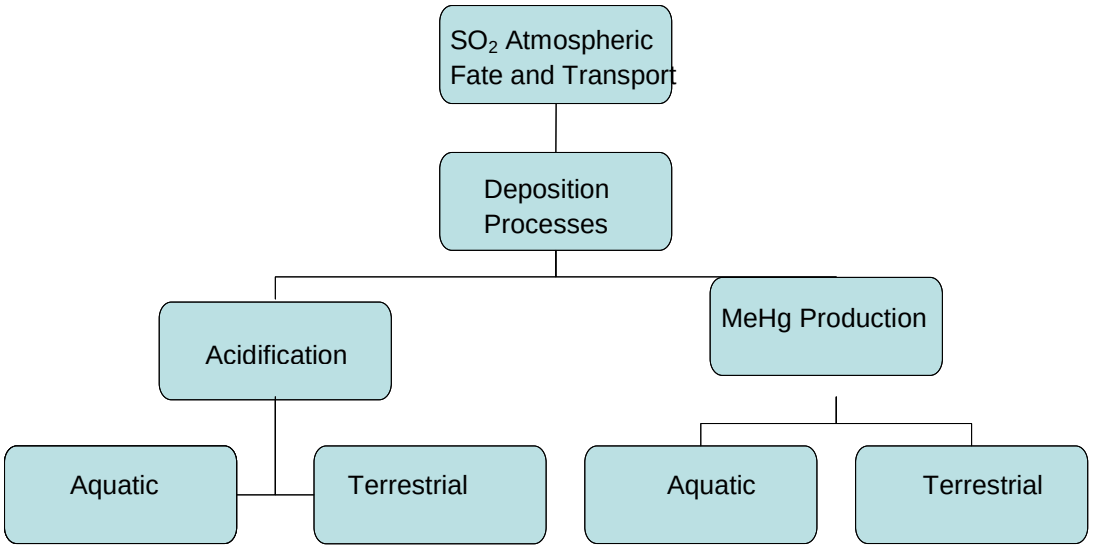
Figure 5.15: Schematic of the benefits assessment process (U.S. EPA, 2006c)



Science of Sulfur Deposition

Sulfur emissions occur over a wide area and depending on prevailing winds and other meteorological conditions, these emissions may be transported hundreds and even thousands of kilometers across North America. Sulfur is primarily emitted as SO_2, and secondary particles are formed from SO_X gaseous emissions and associated chemical reactions in the atmosphere. Deposition of sulfur can occur in either a wet (i.e., rain, snow, sleet, hail, clouds, or fog) or dry form (i.e., gases, dust, and minute particulate matters). Together these emissions are deposited onto terrestrial and aquatic ecosystems across the U.S., contributing to the problems of acidification and methyl mercury production as represented in Figure 5-16.

Figure 5-16: Schematic of Ecological Effects of Sulfur Deposition



The lifetimes of particles vary with particle size. Accumulation-mode particles such as sulfates are kept in suspension by normal air motions and have a lower deposition velocity than coarse-mode particles; they can be transported thousands of kilometers and remain in the atmosphere for a number of days. They are removed from the atmosphere primarily by cloud processes. Particulates affect acid deposition by serving as cloud condensation nuclei and contribute directly to the acidification of rain. In addition, the gas-phase species that lead to the dry deposition of acidity are also precursors of particles. Therefore, reductions in SO_2 emissions will decrease both acid deposition and PM concentrations, but not necessarily in a linear fashion. Sulfuric acid is also deposited on surfaces by dry deposition and can contribute to environmental effects (U.S. EPA, 2008f).

Ecological Effects of Acidification

Deposition of sulfur causes acidification, which alters biogeochemistry and affects animal and plant life in terrestrial and aquatic ecosystems across the U.S. Major effects include a decline in sensitive tree species, such as red spruce (*Picea rubens*) and sugar maple (*Acer saccharum*); and a loss of biodiversity of fishes, zooplankton, and macro invertebrates. The sensitivity of terrestrial and aquatic ecosystems to acidification from sulfur deposition is predominantly governed by geological characteristics (bedrock, weathering rates, etc.). Biological effects of acidification in terrestrial ecosystems are generally linked to aluminum toxicity and decreased ability of plant roots to take up base cations. Decreases in the acid neutralizing capacity and increases in inorganic aluminum concentration contribute to declines in zooplankton, macro invertebrates, and fish species richness in aquatic ecosystems.

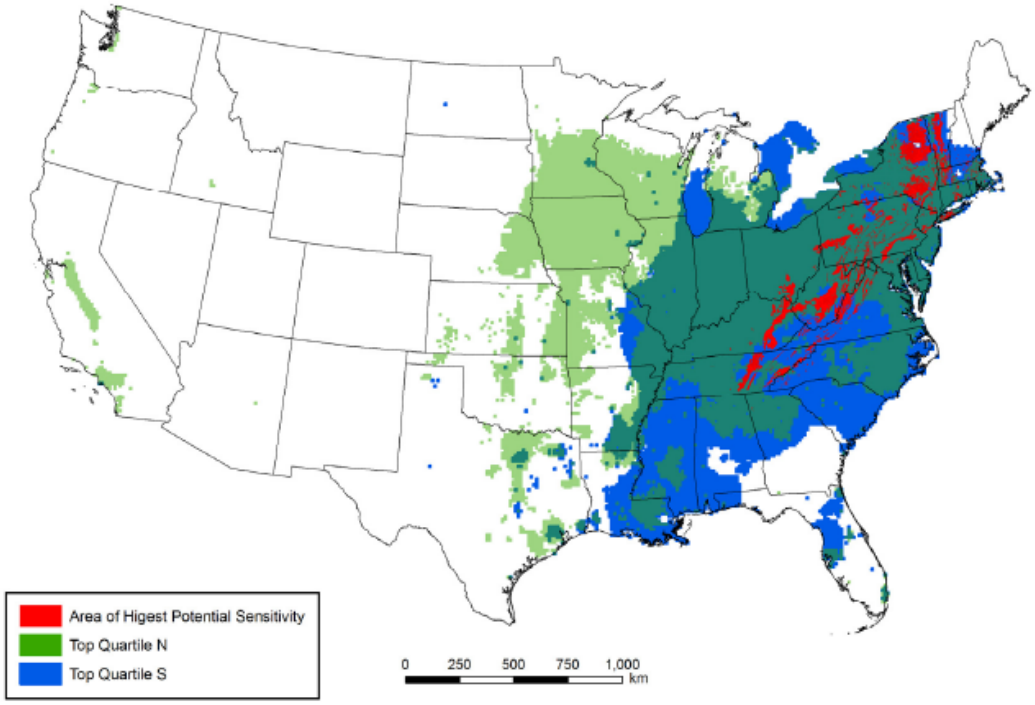
Geology (particularly surficial geology) the principal factor governing the sensitivity of terrestrial and aquatic ecosystems to acidification from nitrogen and sulfur deposition is (U.S. EPA, 2008f). Geologic formations having low base cation supply generally underlie the watersheds of acid-sensitive lakes and streams. Other factors contribute to the sensitivity of soils and surface waters to acidifying deposition, including topography, soil chemistry, land use, and hydrologic flow path.

Terrestrial

Acidifying deposition has altered major biogeochemical processes in the U.S. by increasing the nitrogen and sulfur content of soils, accelerating nitrate and sulfate leaching from soil to drainage waters, depleting base cations (especially calcium and magnesium) from soils, and increasing the mobility of aluminum. Inorganic aluminum is toxic to some tree roots. Plants affected by high levels of aluminum from the soil often have reduced root growth, which restricts the ability of the plant to take up water and nutrients, especially calcium (U. S. EPA, 2008f). These direct effects can, in turn, influence the response of these plants to climatic stresses such as droughts and cold temperatures. They can also influence the sensitivity of plants to other stresses, including insect pests and disease (Joslin et al., 1992) leading to increased mortality of canopy trees. In the U.S., terrestrial effects of acidification are best described for forested ecosystems (especially red spruce and sugar maple ecosystems) with additional information on other plant communities, including shrubs and lichen (U.S. EPA, 2008f).

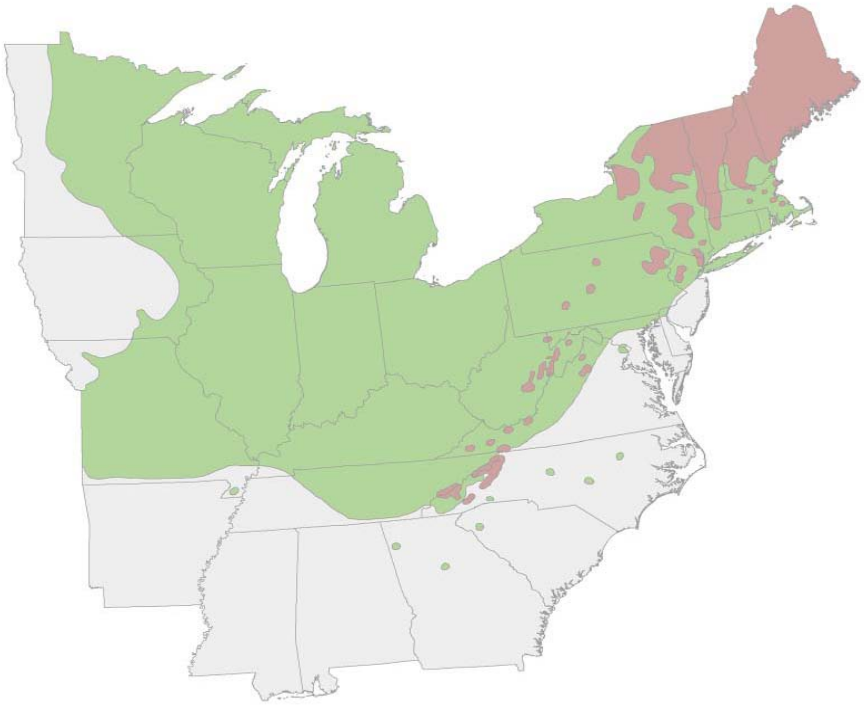
Certain ecosystems in the continental U.S. are potentially sensitive to terrestrial acidification, which is the greatest concern regarding sulfur deposition U.S. EPA (2008b). Figure 5-17 depicts the areas across the U.S. that are potentially sensitive to terrestrial acidification.

Figure 5-17: Areas Potentially Sensitive to Terrestrial Acidification (U.S. EPA, 2008f)



Both coniferous and deciduous forests throughout the eastern U.S. are experiencing gradual losses of base cation nutrients from the soil due to accelerated leaching for acidifying deposition. This change in nutrient availability may reduce the quality of forest nutrition over the long term. Evidence suggests that red spruce and sugar maple in some areas in the eastern U.S. have experienced declining health because of this deposition. For red spruce, (*Picea rubens*) dieback or decline has been observed across high elevation landscapes of the northeastern U.S., and to a lesser extent, the southeastern U.S., and acidifying deposition has been implicated as a causal factor (DeHayes et al., 1999). Figure 5-18 shows the distribution of red spruce (brown) and sugar maple (green) in the eastern U.S.

Figure 5-18: Distribution of Red Spruce (pink) and Sugar Maple (green) in the Eastern U.S. (U.S. EPA, 2008f)



Ecosystem Services

Terrestrial acidification affects several important ecological endpoints, including declines in habitat for threatened and endangered species (cultural), declines in forest aesthetics (cultural), declines in forest productivity (provisioning), and increases in forest soil erosion and reductions in water retention (cultural and regulating).

Forests in the northeastern United States provide several important and valuable provisioning services in the form of tree products. Sugar maples are a particularly important commercial hardwood tree species, providing timber and maple syrup. In the United States, sugar maple saw timber was nearly 900 million board feet in 2006 (U.S. F.S., 2006), and annual production of maple syrup was nearly 1.4 million gallons, accounting for approximately 19% of worldwide production. The total annual value of U.S. production in these years was approximately $160 million (NASS, 2008). Red spruce is also used in a variety of products including lumber, pulpwood, poles, plywood, and musical instruments. The total removal of red spruce saw timber from timberland in the United States was over 300 million board feet in 2006 (U.S. F.S., 2006).

Forests in the northeastern United States are also an important source of cultural ecosystem services—nonuse (i.e., existence value for threatened and endangered species), recreational, and aesthetic services. Red spruce forests are home to two federally listed species and one delisted species:

1. Spruce-fir moss spider (Microhexura montivaga)—endangered
2. Rock gnome lichen (Gymnoderma lineare)—endangered
3. Virginia northern flying squirrel (Glaucomys sabrinus fuscus)—delisted, but important

Forestlands support a wide variety of outdoor recreational activities, including fishing, hiking, camping, off-road driving, hunting, and wildlife viewing. Regional statistics on recreational activities that are specifically forest based are not available; however, more general data on outdoor recreation provide some insights into the overall level of recreational services provided by forests. More than 30% of the U.S. adult population visited a wilderness or primitive area during the previous year and engaged in day hiking (Cordell et al., 2008). From 1999 to 2004, 16% of adults in the northeastern United States participated in off-road vehicle recreation, for an average of 27 days per year (Cordell et al., 2005). The average consumer surplus value per day of off-road driving in the United States was $25.25 (in 2007 dollars), and the implied total annual value of off-road driving recreation in the northeastern United States was more than $9 billion (Kaval and Loomis, 2003). More than 5% of adults in the northeastern United States participated in nearly 84 million hunting days (U.S. FWS and U.S. Census Bureau, 2007). Ten percent of adults in northeastern states participated in wildlife viewing away from home on 122 million days in 2006. For these recreational activities in the northeastern United States, Kaval and Loomis (2003) estimated average consumer surplus values per day of $52.36 for hunting and $34.46 for wildlife viewing (in 2007 dollars). The implied total annual value of hunting and wildlife viewing in the northeastern United States was, therefore, $4.4 billion and $4.2 billion, respectively, in 2006.

As previously mentioned, it is difficult to estimate the portion of these recreational services that are specifically attributable to forests and to the health of specific tree species. However, one recreational activity that is directly dependent on forest conditions is fall color viewing. Sugar maple trees, in particular, are known for their bright colors and are, therefore, an essential aesthetic component of most fall color landscapes. A survey of residents in the Great Lakes area found that roughly 30% of residents reported at least one trip in the previous year involving fall color viewing (Spencer and Holecek, 2007). In a separate study conducted in Vermont, Brown (2002) reported that more than 22% of households visiting Vermont in 2001 made the trip primarily for viewing fall colors.

Two studies estimated values for protecting high-elevation spruce forests in the southern Appalachian Mountains. Kramer et al., (2003) conducted a contingent valuation study estimating households' willingness to pay (WTP) for programs to protect remaining high-elevation spruce forests from damages associated with air pollution and insect infestation. Median household WTP was estimated to be roughly $29 (in 2007 dollars) for a smaller program, and $44 for the more extensive program. Jenkins et al. (2002) conducted a very similar study in seven Southern Appalachian states on a potential program to maintain forest conditions at status quo levels. The overall mean annual WTP for the forest protection programs was $208 (in 2007 dollars). Multiplying the average WTP estimate from this study by the total number of households in the seven-state Appalachian region results in an aggregate annual value of $3.4 billion for avoiding a significant decline in the health of high-elevation spruce forests in the Southern Appalachian region.

Forests in the northeastern United States also support and provide a wide variety of valuable regulating services, including soil stabilization and erosion control, water regulation, and climate regulation. The total value of these ecosystem services is very difficult to quantify in a meaningful way, as is the reduction in the value of these services associated with total nitrogen and sulfur deposition. As terrestrial acidification contributes to root damages, reduced biomass growth, and tree mortality, all of these services are likely to be affected; however, the magnitude of these impacts is currently very uncertain.

Aquatic Ecosystems

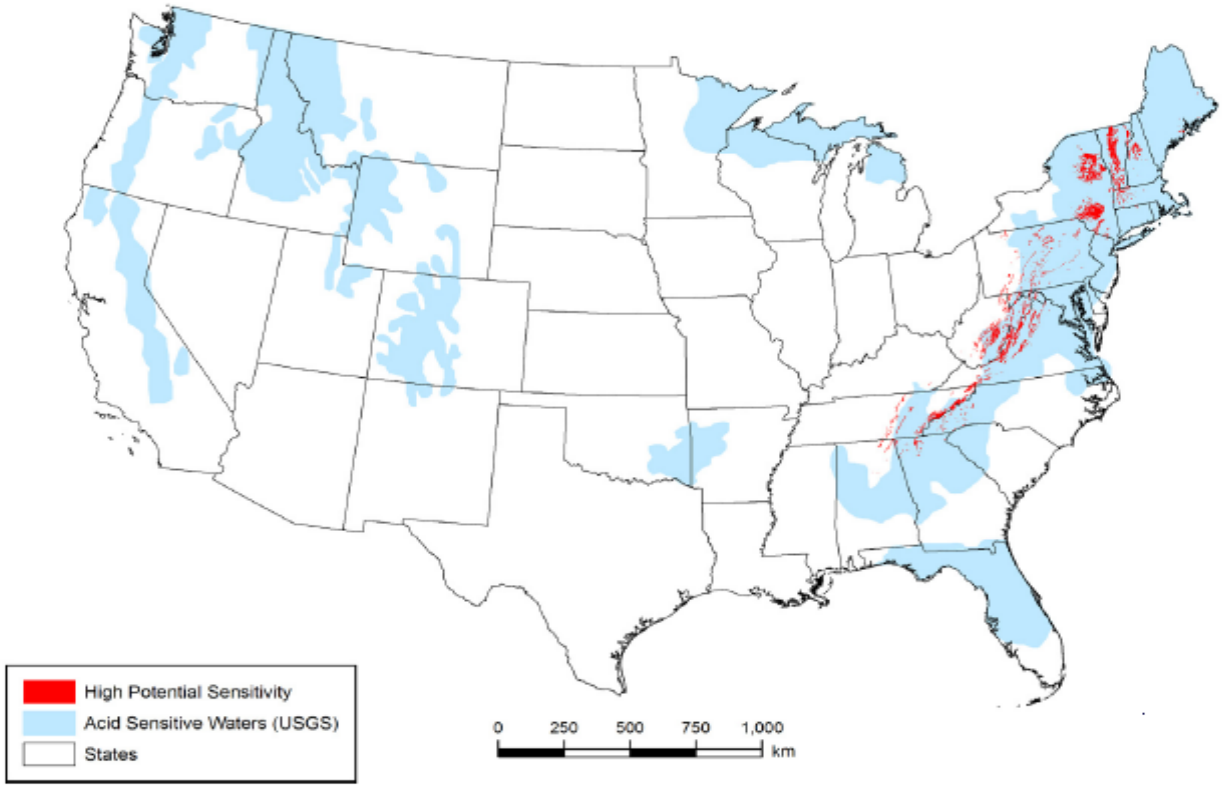
Aquatic effects of acidification have been well studied in the U.S. and elsewhere at various trophic levels. These studies indicate that aquatic biota have been affected by acidification at virtually all levels of the food web in acid sensitive aquatic ecosystems. Effects have been most clearly documented for fish, aquatic insects, other invertebrates, and algae. Biological effects are primarily attributable to a combination of low pH and high inorganic aluminum concentrations. Such conditions occur more frequently during rainfall and snowmelt that cause high flows of water and less commonly during low-flow conditions, except where chronic acidity conditions are severe. Biological effects of episodes include reduced fish condition factor, changes in species composition and declines in aquatic species richness across multiple taxa, ecosystems and regions. These conditions may also result in direct fish mortality (Van Sickle et al., 1996). Biological effects in aquatic ecosystems can be divided into two major categories: effects on health, vigor, and reproductive success; and effects on biodiversity. Several studies have shown that surface water with ANC values greater than 50 µeq/L tend to protect most fish (i.e., brook trout, others) and other aquatic organisms (see Table 5-15).

Table 5-15: Aquatic Status Categories

Category Label	ANC Levels	Expected Ecological Effects
Acute Concern	<0 micro equivalent per Liter (µeq/L)	Near complete loss of fish populations is expected. Planktonic communities have extremely low diversity and are dominated by acidophilic forms. The number of individuals in plankton species that are present is greatly reduced.
Severe Concern	0–20 µeq/L	Highly sensitive to episodic acidification. During episodes of high acidifying deposition, brook trout populations may experience lethal effects. Diversity and distribution of zooplankton communities decline sharply.
Elevated Concern	20–50 µeq/L	Fish species richness is greatly reduced (i.e., more than half of expected species can be missing). On average, brook trout populations experience sublethal effects, including loss of health, reproduction capacity, and fitness. Diversity and distribution of zooplankton communities decline.
Moderate Concern	50–100 µeq/L	Fish species richness begins to decline (i.e., sensitive species are lost from lakes). Brook trout populations are sensitive and variable, with possible sublethal effects. Diversity and distribution of zooplankton communities also begin to decline as species that are sensitive to acidifying deposition are affected.
Low Concern	>100 µeq/L	Fish species richness may be unaffected. Reproducing brook trout populations are expected where habitat is suitable. Zooplankton communities are unaffected and exhibit expected diversity and distribution.

A number of national and regional assessments have been conducted to estimate the distribution and extent of surface water acidity in the U.S (U.S. EPA, 2008f). As a result, several regions of the U.S. have been identified as containing a large number of lakes and streams that are seriously impacted by acidification. Figure 5-19 illustrates those areas of the U.S. where aquatic ecosystems are at risk from acidification.

Figure 5-19: Areas Potentially Sensitive to Aquatic Acidification (U.S. EPA, 2008b)



Ecosystem Services

Because acidification primarily affects the diversity and abundance of aquatic biota, it also affects the ecosystem services that are derived from the fish and other aquatic life found in these surface waters.

While acidification is unlikely to have serious negative effects on, for example, water supplies, it can limit the productivity of surface waters as a source of food (i.e., fish). In the northeastern United States, the surface waters affected by acidification are not a major source of commercially raised or caught fish; however, they are a source of food for some recreational and subsistence fishermen and for other consumers. For example, although there is evidence that certain population subgroups in the northeastern United States, such as the Hmong and Chippewa ethnic groups, have particularly high rates of self-caught fish consumption (Hutchison and Kraft, 1994; Peterson et al., 1994), it is not known if and how their consumption patterns are affected by the reductions in available fish populations caused by surface water acidification.

Inland surface waters support several cultural services, including aesthetic and educational services and recreational fishing. Recreational fishing in lakes and streams is among the most popular outdoor recreational activities in the northeastern United States. Based on studies conducted in the northeastern United States, Kaval and Loomis (2003) estimated average consumer surplus values per day of $35.91 for recreational fishing (in 2007 dollars); therefore, the implied total annual value of freshwater fishing in the northeastern United States was $5.06 billion in 2006. For recreation days, consumer surplus value is most commonly measured using recreation demand, travel cost models.

In addition, inland surface waters provide a number of regulating services associated with hydrological and climate regulation by providing environments that sustain aquatic food webs. These services are disrupted by the toxic effects of acidification on fish and other aquatic life. Although it is difficult to quantify these services and how they are affected by acidification, some of these services may be captured through measures of provisioning and cultural services.

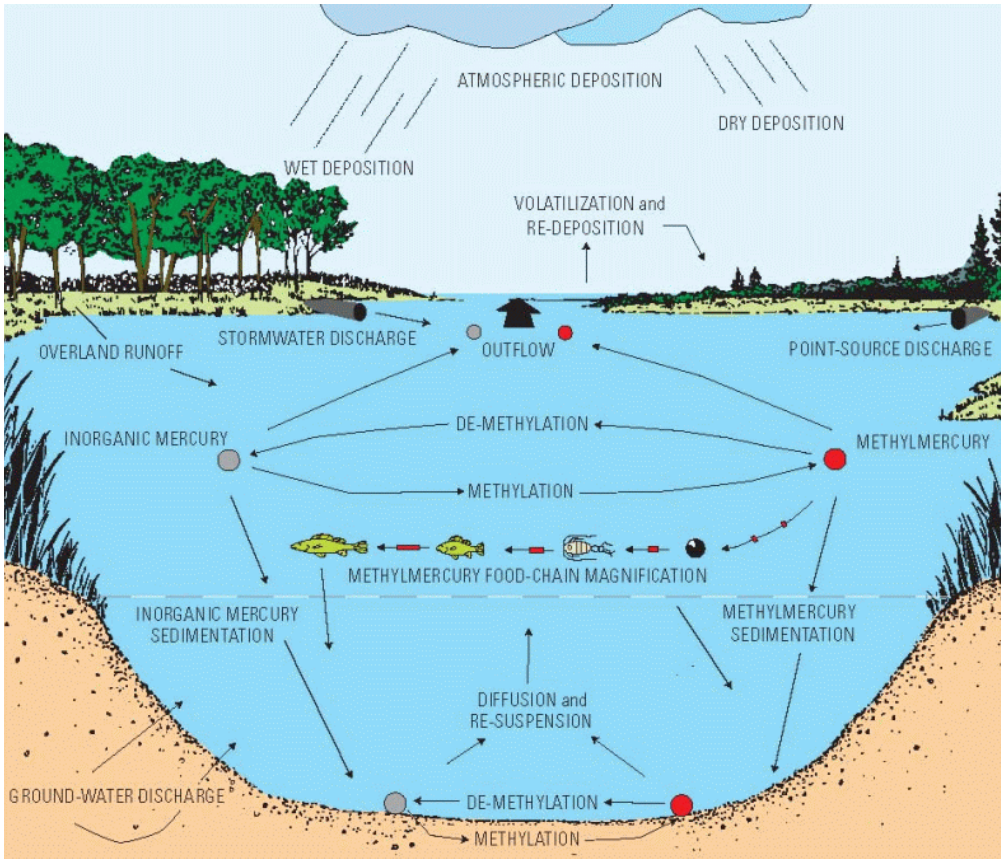
Ecological Effects of Associated with Mercury Methylation

Mercury is a highly neurotoxic contaminant that enters the food web as a methylated compound, methylmercury (U.S. EPA, 2008f). The contaminant is concentrated in higher trophic levels, including fish eaten by humans. Experimental evidence has established that only inconsequential amounts of methylmercury can be produced in the absence of sulfate. Many variables influence how much mercury accumulates in fish, but elevated mercury levels in fish can only occur where substantial amounts of methylmercury are present. Current evidence indicates that in watersheds where mercury is present, increased SO_x deposition very likely results in methylmercury accumulation in fish (Drevnick et al., 2007; Munthe et al, 2007). The ISA concluded that evidence is sufficient to infer a casual relationship between sulfur deposition and increased mercury methylation in wetlands and aquatic environments.

Establishing the quantitative relationship between sulfate and mercury methylation in natural settings is difficult because of the presence of multiple interacting factors in aquatic and terrestrial environments, including wetlands, aquatic environments where sulfate, sulfur-reducing bacteria (SRB), and mercury are present. The presence of sulfate, inorganic mercury, and sulfate reducing bacteria (SRB) are the primary requirements for bacterially mediated sulfate-reducing mercury conversion. Additional factors affecting conversion include the presence of anoxic conditions, temperature, the presence and types of organic matter, the presence and types of mercury-binding species, and watershed effects (e.g., watershed type, land cover, water body limnology, and runoff loading). With regard to methylmercury, the highest concentrations in the environment generally occur at or near the sedimentary surface,

below the oxic–anoxic boundary. Although mercury methylation can occur within the water column, there is generally a far greater contribution of mercury methylation from sediments because of anoxia and of greater concentrations of SRB, substrate, and sulfate. Figure 5-20 depicts the mercury cycle.

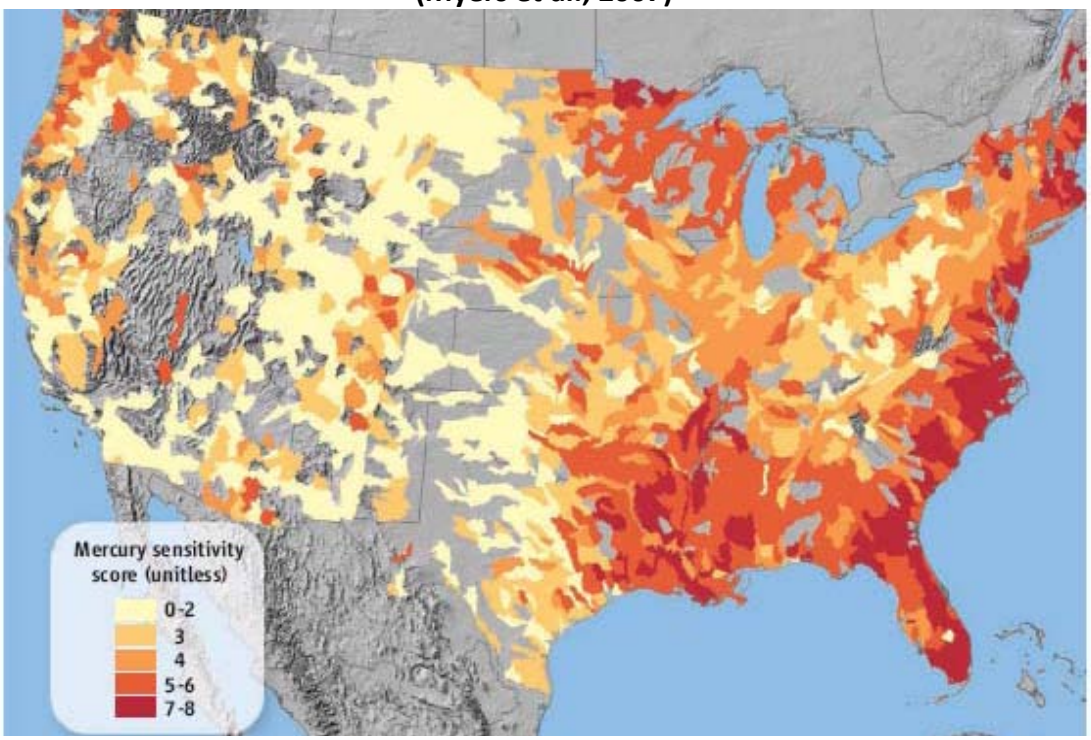
Figure 5-20: The mercury cycle in an ecosystem (USGS, 2006)



State-level fish consumption advisories for mercury are based on state criteria, many of which are based on EPA's fish tissue criterion for methylmercury or on U.S. Food and Drug Administration's action limits. In 2008, there were 3,361 fish advisories issued at least in part for mercury contamination (80% of all fish advisories), covering 16.8 million lake acres (40% of total lake acreage) and 1.3 million river miles (35% of total river miles) over all 50 states, one U.S. territory, and 3 tribes (U.S. EPA, 2009f). Recently, the U.S. Geological Survey (USGS) examined mercury levels in top-predator fish, bed sediment, and water from 291 streams across the U.S. (Scudder et al., 2009). USGS detected mercury contamination in every fish sampled, and the concentration of mercury in fish exceeded EPA's criterion in 27% of the sites sampled. Figure 5.21 illustrates a map of mercury-sensitive watersheds based on sulfate concentrations, acid neutralizing capacity (ANC), levels of dissolved organic carbon and pH,

mercury species concentrations, and soil types to gauge the methylation sensitivity (Myers et al., 2007).

Figure 5.21: Preliminary USGS map of mercury methylation–sensitive watersheds (Myers et al., 2007)



Decreases in sulfate deposition/emissions have already shown promising reductions in methylmercury (U.S. EPA, 2009e). Observed decreases in methylmercury fish tissue concentrations have been linked to decreased acidification and declining sulfate and mercury deposition (Hrabik and Watras, 2002; Drevnick et al., 2007).

The ecosystem service most directly affected by sulfate mediated mercury methylation is the provision of fish for consumption as a food source. This service is of particular importance to groups engaged in subsistence fishing, pregnant women and young children. While it is not possible to quantify the reduction in fish consumption due to the presence of methyl mercury in fish from sulfur deposition, it is likely, given the number of state advisories and the EPA/FDA guidelines (EPA/FDA, 2004) on consumption for pregnant women and young children, that this service is negatively affected.

Ecological Effects Associated with Gaseous Sulfur Dioxide

Uptake of gaseous sulfur dioxide in a plant canopy is a complex process involving adsorption to surfaces (leaves, stems, and soil) and absorption into leaves. SO_2 penetrates into leaves through to the stomata, although there is evidence for limited pathways via the cuticle. Pollutants must be transported from the bulk air to the leaf boundary layer in order to get to the stomata. When the stomata are closed, as occurs under dark or drought conditions, resistance to gas uptake is very high and the plant has a very low degree of susceptibility to injury. In contrast, mosses and lichens do not have a protective cuticle barrier to gaseous pollutants or stomates and are generally more sensitive to gaseous sulfur and nitrogen than vascular plants (U.S. EPA, 2008f). Acute foliar injury usually happens within hours of exposure, involves a rapid absorption of a toxic dose, and involves collapse or necrosis of plant tissues. Another type of visible injury is termed chronic injury and is usually a result of variable SO_2 exposures over the growing season. Besides foliar injury, chronic exposure to low SO_2 concentrations can result in reduced photosynthesis, growth, and yield of plants. These effects are cumulative over the season and are often not associated with visible foliar injury. As with foliar injury, these effects vary among species and growing environment. SO_2 is also considered the primary factor causing the death of lichens in many urban and industrial areas (Hutchinson et al., 1996).

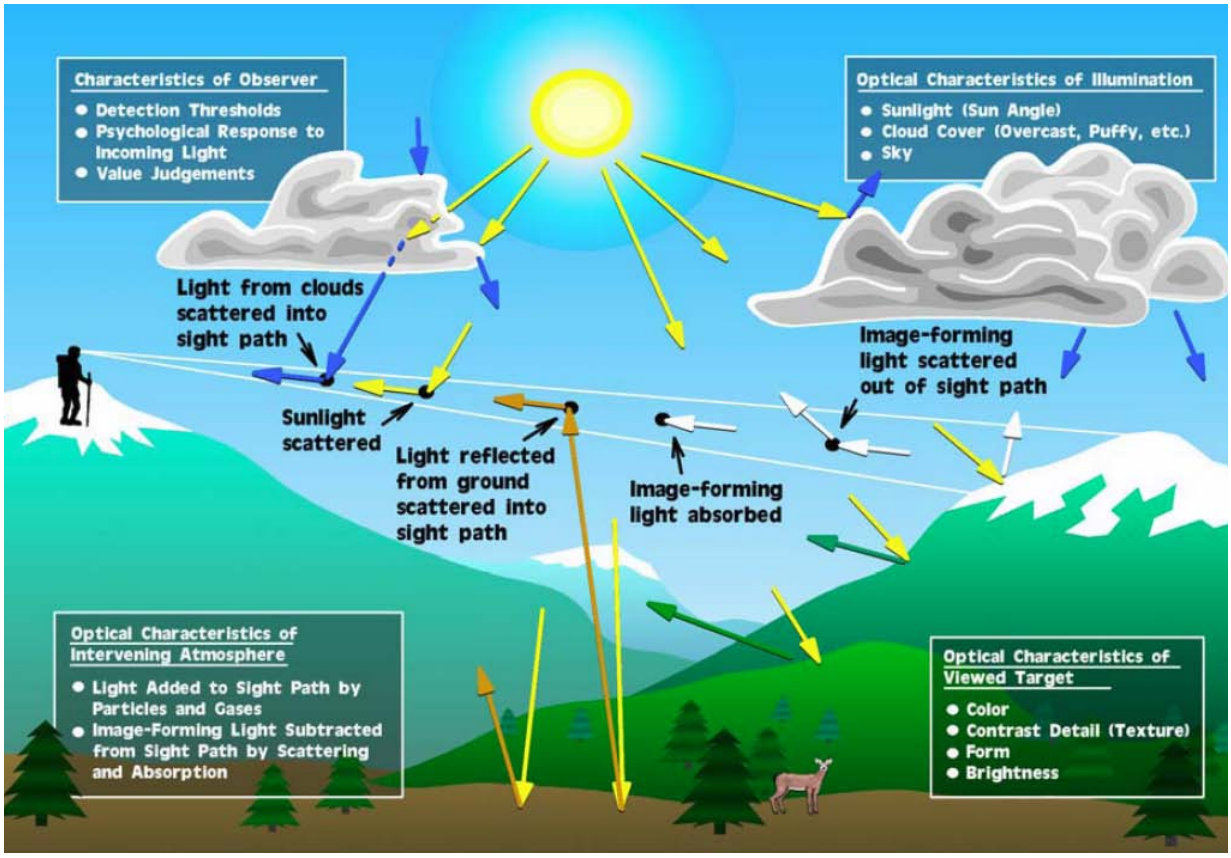
5.9.2 Visibility Improvements

Reductions in SO_2 emissions and secondary formation of $PM_{2.5}$ due to the alternative standards will improve the level of visibility throughout the United States. These suspended particles and gases degrade visibility by scattering and absorbing light. 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 Smokey Mountains National Park. Without the necessary air quality data, we were unable to calculate the predicted change in visibility due to control strategy to attain various alternate standard levels. However, in this section, we describe the process by which SO_2 emissions impair visibility and how this impairment affects the public.

Visual air quality (VAQ) is commonly measured as either light extinction, which is defined as the loss of light per unit of distance in terms of inverse megameters (Mm^{-1}) or the deciview (dv) metric (Pitchford and Malm, 1993), which is a logarithmic function of extinction. Extinction and deciviews are physical measures of the amount of visibility impairment (e.g., the amount of "haze"), with both extinction and deciview increasing as the amount of haze increases. Light extinction is the optical characteristic of the atmosphere that occurs when light is either

scattered or absorbed, which converts the light to heat. Particulate matter and gases can both scatter and absorb light. Fine particles with significant light-extinction efficiencies include sulfates, nitrates, organic carbon, elemental carbon, and soil (Sisler, 1996). The extent to which any amount of light extinction affects a person's ability to view a scene depends on both scene and light characteristics. For example, the appearance of a nearby object (i.e. a building) is generally less sensitive to a change in light extinction than the appearance of a similar object at a greater distance. See Figure 5-22 for an illustration of the important factors affecting visibility.

Figure 5-22: Important factors involved in seeing a scenic vista (Malm, 1999)



In conjunction with the U.S. National Park Service, the U.S. Forest Service, other Federal land managers, and State organizations in the U.S., the U.S. EPA has supported visibility monitoring in national parks and wilderness areas since 1988. The monitoring network known as IMPROVE (Interagency Monitoring of Protected Visual Environments) now includes 150 sites that represent almost all of the Class I areas across the country (see figure 5-23) (U.S. EPA, 2009d).

Figure 5-23: Mandatory Class I Areas in the U.S.

Annual average visibility conditions (reflecting light extinction due to both anthropogenic and non-anthropogenic sources) vary regionally across the U.S. The rural East generally has higher levels of impairment than remote sites in the West, with the exception of urban-influenced sites such as San Gorgonio Wilderness (CA) and Point Reyes National Seashore (CA), which have annual average levels comparable to certain sites in the Northeast (U.S. EPA, 2004). Higher visibility impairment levels in the East are due to generally higher concentrations of fine particles, particularly sulfates, and higher average relative humidity levels. In fact, particulate sulfate is the largest contributor to regional haze in the eastern U.S. (i.e., 40% or more annually and 75% during summer). In the western U.S., particulate sulfate contributes to 20-50% of regional haze (U.S. EPA, 2009d). While visibility trends have improved in most Class I areas, the recent data show that these areas continue to suffer from visibility impairment. In eastern parks, average visual range has decreased from 90 miles to 15-25 miles, and in the West, visual range has decreased from 140 miles to 35-90 miles (U.S. EPA, 2004; U.S. EPA, 1999).

Visibility has direct significance to people's enjoyment of daily activities and their overall sense of wellbeing (U.S. EPA, 2009d). Good visibility increases the quality of life where individuals live and work, and where they engage in recreational activities. When the necessary AQ data is available, EPA generally considers benefits from these two categories of visibility

changes: residential visibility (i.e., the visibility in and around the locations where people live) and recreational visibility (i.e., visibility at Class I national parks and wilderness areas.) 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 bird watching. 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. In addition, evidence suggests that an individual's WTP for improvements in visibility at a Class I area is influenced by whether it is in the region in which the individual lives, or whether it is somewhere else (Chestnut and Rowe, 1990). In general, people appear to be willing to pay more for visibility improvements at parks and wilderness areas that are "in-region" than at those that are "out-of-region." This is plausible, because people are more likely to visit, be familiar with, and care about parks and wilderness areas in their own part of the country. EPA generally uses a contingent valuation study as the basis for monetary estimates of the benefits of visibility changes in recreational areas (Chestnut and Rowe, 1990). To estimate the monetized value of visibility changes, an analyst would multiply the willingness-to-pay estimates by the amount of visibility impairment, but this information in unavailable for this analysis.

5.10 Limitations and Uncertainties

The National Research Council (NRC) (2002) concluded that EPA's general methodology for calculating the benefits of reducing air pollution is reasonable and informative in spite of inherent uncertainties. To address these inherent uncertainties, NRC highlighted the need to conduct rigorous quantitative analysis of uncertainty and to present benefits estimates to decisionmakers in ways that foster an appropriate appreciation of their inherent uncertainty. In response to these comments, EPA's Office of Air and Radiation (OAR) is developing a comprehensive strategy for characterizing the aggregate impact of uncertainty in key modeling elements on both health incidence and benefits estimates. Components of that strategy include emissions modeling, air quality modeling, health effects incidence estimation, and valuation.

In this analysis, we use three methods to assess uncertainty quantitatively: Monte Carlo analysis, sensitivity analysis, and alternate concentration-response functions for PM mortality. We also provide a qualitative assessment for those aspects that we are unable to address quantitatively in this analysis. Each of these analyses is described in detail in the following sections.

This analysis includes many data sources as inputs, including emission inventories, air quality data from models (with their associated parameters and inputs), population data, health effect estimates from epidemiology studies, and economic data for monetizing benefits. Each of these inputs may be uncertain and would affect the benefits estimate. When the uncertainties from each stage of the analysis are compounded, small uncertainties can have large effects on the total quantified benefits. In this analysis, we are unable to quantify the cumulative effect of all of these uncertainties, but we provide the following analyses to characterize many of the largest sources of uncertainty.

5.9.1 Monte Carlo analysis

Similar to other recent RIAs, we used Monte Carlo methods for estimating characterizing random sampling error associated with the concentration response functions and economic valuation functions. Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables, such as incidence of morbidity. Specifically, we used Monte Carlo methods to generate confidence intervals around the estimated health impact and dollar benefits. The reported standard errors in the epidemiological studies determined the distributions for individual effect estimates, as shown in Table 5.5 for SO_2 benefits. Unfortunately, the associated confidence intervals are not available for the $PM_{2.5}$ co-benefits due to limitations in the benefit-per-ton methodology.

5.9.2 Sensitivity analyses

We performed a variety of sensitivity analyses on the benefits results to assess the sensitivity of the primary results to various data inputs and assumptions. We then changed each default input one at a time and recalculated the total monetized benefits to assess the percent change from the default. In Tables 5.16 and 5.17, we repeat the results of this sensitivity analysis already presented in previous section for comparison purposes. We indicate each input parameter, the value used as the default, and the values for the sensitivity analyses, and then we provide the total monetary benefits for each input and the percent change from the default value. This sensitivity analysis indicates that the results are most sensitive to assumptions regarding the attainment status and the threshold assumption in the PM-mortality relationship, and the results are less sensitive to alternate assumptions regarding the interpolation method, discount rate, and various assumptions regarding SO_2 exposure. To account for the large difference in magnitude between benefits from reduced SO_2 exposure and $PM_{2.5}$ exposure, we provide separate sensitivity analyses. We show the sensitivity analysis for the most stringent alternative analyzed (50 ppb), but other standard levels would show similar

sensitivity to these perturbations, albeit with smaller magnitudes. Descriptions of the sensitivity analyses are provided in the relevant sections of this chapter.

Table 5.16: Sensitivity Analyses for SO_2 Health Benefits to Fully Attain 50 ppb Standard

		Total SO_2 Benefits (millions of 2006$)	% Change from Default
Exposure Estimation Method	50km radius	$12	N/A
	25km radius	$9.3	-21%
	100km radius	$15	26%
	Unconstrained	$22	89%
Location of Hospital Admission Studies	w/US-based studies only	$12	N/A
	w/Canada-based studies only	$62	424%
Asthma Pooling Method	Pool all endpoints together	$12	N/A
	One or more symptoms only	$12	-0.2%

Table 5.17: Sensitivity Analyses for $PM_{2.5}$ Health Co-Benefits for an Alternative Standard SO_2 at 50 ppb

		Total $PM_{2.5}$ Benefits (billions of 2006$)	% Change from Default
Threshold Assumption (with Epidemiology Study)	No Threshold (Pope)	$38	N/A
	No Threshold (Laden)	$93	N/A
	Threshold (Pope)*	$25	-34%
	Threshold (Laden)*	$54	-42%
Discount Rate (with Epidemiology Study)	3% (Pope)	$38	N/A
	3% (Laden)	$93	N/A
	7% (Pope)	$35	-9%
	7% (Laden)	$84	-10%
Simulated Attainment (using Pope)	Full attainment	$38	N/A
	Partial Attainment	$29	-29%

* The threshold model is not directly comparable to the no-threshold model. The threshold estimates do not include two technical updates, and they are based on data for 2015, instead of 2020. Directly comparable estimates are not available.

5.9.3 Alternate concentration-response functions for PM mortality

$PM_{2.5}$ mortality co-benefits are the largest benefit category that we monetized in this analysis. To better understand the concentration-response relationship between $PM_{2.5}$ exposure and premature mortality, EPA conducted an expert elicitation in 2006 (Roman et al., 2008; IEc, 2006). In general, the results of the expert elicitation support the conclusion that the benefits of $PM_{2.5}$ control are very likely to be substantial. In previous RIAs, EPA presented benefits estimates using concentration response functions derived from the $PM_{2.5}$ Expert Elicitation as a range from the lowest expert value (Expert K) to the highest expert value (Expert E). However, this approach did not indicate the agency's judgment on what the best estimate of PM benefits may be, and EPA's Science Advisory Board described this presentation as

misleading. Therefore, we began to present the cohort-based studies (Pope et al, 2002; and Laden et al., 2006) as our core estimates in the Portland Cement RIA (U.S. EPA, 2009a). Using alternate relationships between $PM_{2.5}$ and premature mortality supplied by experts, higher and lower benefits estimates are plausible, but most of the expert-based estimates fall between the two epidemiology-based estimates (Roman et al., 2008).

In this analysis, we present the results derived from the expert elicitation as indicative of the uncertainty associated with a major component of the health impact functions, and we provide the independent estimates derived from each of the twelve experts to better characterize the degree of variability in the expert responses. In this chapter, we provide the results using the concentration-response functions derived from the expert elicitation in both tabular (Table 5.11) and graphical form (Figure 5.9). Please note that these results are not the direct results from the studies or expert elicitation; rather, the estimates are based in part on the concentration-response function provided in those studies. Because in this RIA we estimate PM co-benefits using benefit-per-ton estimates, technical limitations prevent us from providing the associated credible intervals with the expert functions.

5.9.4 Qualitative assessment of uncertainty and other analysis limitations

Although we strive to incorporate as many quantitative assessments of uncertainty, there are several aspects for which we are only able to address qualitatively. These aspects are important factors to consider when evaluating the relative benefits of the attainment strategies for each of the alternative standards:

1. The gradient of ambient SO_2 concentrations is difficult to estimate due to the sparsity of the monitoring network in some areas. The 12km CMAQ grid, which is the air quality modeling resolution, may be too coarse to accurately estimate the potential near-field health benefits of reducing SO_2 emissions. These uncertainties may under- or over-estimate benefits.
2. The interpolation techniques used to estimate the full attainment benefits of the alternative standards contributed some uncertainty to the analysis. The great majority of benefits estimated for the various standard alternatives were derived through interpolation. As noted previously in this chapter, these benefits are likely to be more uncertain than if we had modeled the air quality scenario for both SO_2 and $PM_{2.5}$. In general, the VNA interpolation approach will under-estimate benefits because it does not account for the broader spatial distribution of air quality changes that may occur due to the implementation of a regional emission control program.

3. There are many uncertainties associated with the health impact functions used in this modeling effort. These include: within study variability (the precision with which a given study estimates the relationship between air quality changes and health effects); across study variation (different published studies of the same pollutant/health effect relationship typically do not report identical findings and in some instances the differences are substantial); the application of C-R functions nationwide (does not account for any relationship between region and health effect, to the extent that such a relationship exists); extrapolation of impact functions across population (we assumed that certain health impact functions applied to age ranges broader than that considered in the original epidemiological study); and various uncertainties in the C-R function, including causality and thresholds. These uncertainties may under- or over-estimate benefits.

4. Co-pollutants present in the ambient air may have contributed to the health effects attributed to SO_2 in single pollutant models. Risks attributed to SO_2 might be overestimated where concentration-response functions are based on single pollutant models. If co-pollutants are highly correlated with SO_2, their inclusion in an SO_2 health effects model can lead to misleading conclusions in identifying a specific causal pollutant. Because this collinearity exists, many of the studies reported statistically insignificant effect estimates for both SO_2 and the co-pollutants; this is due in part to the loss of statistical power as these models control for co-pollutants. Where available, we have selected multipollutant effect estimates to control for the potential confounding effects of co-pollutants; these include NYDOH (2006), Schwartz et al. (1994) and O'Conner et al. (2007). The remaining studies include single pollutant models.

5. This analysis is for the year 2020, and projecting key variables introduces uncertainty. Inherent in any analysis of future regulatory programs are uncertainties in projecting atmospheric conditions and source level emissions, as well as population, health baselines, incomes, technology, and other factors.

6. This analysis omits certain unquantified effects due to lack of data, time and resources. These unquantified endpoints include other health effects, ecosystem effects, and visibility. EPA will continue to evaluate new methods and models and select those most appropriate for estimating the benefits of reductions in air pollution. Enhanced collaboration between air quality modelers, epidemiologists, toxicologists, ecologists, and economists should result in a more tightly integrated analytical framework for measuring benefits of air pollution policies.

7. $PM_{2.5}$ co-benefits represent a substantial proportion of total monetized benefits (over 99% of total monetized benefits), and these estimates are subject to a number of assumptions and uncertainties.

a. $PM_{2.5}$ co-benefits were derived through benefit per-ton estimates, which do not reflect local variability in population density, meteorology, exposure, baseline health incidence rates, or other local factors that might lead to an over-estimate or under-estimate of the actual benefits of controlling directly emitted fine particulates.

b. We assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because $PM_{2.5}$ produced via transported precursors emitted from EGUs may differ significantly from direct $PM_{2.5}$ released from diesel engines and other industrial sources, but no clear scientific grounds exist for supporting differential effects estimates by particle type.

c. We assume that the health impact function for fine particles is linear down to the lowest air quality levels modeled in this analysis. Thus, the estimates include health benefits from reducing fine particles in areas with varied concentrations of $PM_{2.5,}$ including both regions that are in attainment with fine particle standard and those that do not meet the standard down to the lowest modeled concentrations.

d. To characterize the uncertainty in the relationship between $PM_{2.5}$ and premature mortality (which typically accounts for 85% to 95% of total monetized benefits), we include a set of twelve estimates based on results of the expert elicitation study in addition to our core estimates. Even these multiple characterizations omit the uncertainty in air quality estimates, baseline incidence rates, populations exposed and transferability of the effect estimate to diverse locations. As a result, the reported confidence intervals and range of estimates give an incomplete picture about the overall uncertainty in the $PM_{2.5}$ estimates. This information should be interpreted within the context of the larger uncertainty surrounding the entire analysis. For more information on the uncertainties associated with $PM_{2.5}$ co-benefits, please consult the $PM_{2.5}$ NAAQS RIA (Table 5.5).

5.11 Discussion

The results of this benefits analysis suggest that attaining any of the SO_2 alternative standards would produce substantial health benefits in the form of fewer respiratory hospitalizations, respiratory emergency department visits and cases of acute respiratory symptoms from reduced SO_2 exposure. In addition, attaining any of the SO_2 alternative

standards would also produce substantial health co-benefits from reducing $PM_{2.5}$ exposure in the form of avoided premature mortality and other morbidity effects.

This analysis is the first time that EPA has estimated the monetized human health benefits of reducing exposure to SO_2 to support a proposed change in the NAAQS. In contrast to recent $PM_{2.5}$ and ozone-related benefits assessments, there was far less analytical precedent on which to base this assessment. For this reason, we developed entirely new components of the health impact analysis, including the identification of health endpoints to be quantified and the selection of relevant effect estimates within the epidemiology literature. As the SO_2 health literature continues to evolve, EPA will reassess the health endpoints and risk estimates used in this analysis.

While the monetized benefits of reduced SO_2 exposure appear small when compared to the monetized benefits of reduced $PM_{2.5}$ exposure, readers should not necessarily infer that the total monetized benefits of attaining a new SO_2 standard are minimal. As shown in Table 5.13, the $PM_{2.5}$ co-benefits represent over 99% of the total monetized benefits. This result is consistent with recent RIAs, where the $PM_{2.5}$ co-benefits represent a large proportion of total monetized benefits. This is primarily due to the decision not to quantify SO_2 -related premature mortality and other morbidity endpoints due to the uncertainties associated with estimating those endpoints. Studies have shown that there is a relationship between SO_2 exposure and premature mortality, but that relationship is limited by potential confounding. Because premature mortality generally comprises over 90% of the total monetized benefits, this decision may underestimate the monetized health benefits of reduced SO_2 exposure.

We were unable to quantify the benefits from several welfare benefit categories. We lacked the necessary air quality data to quantify the benefits from improvements in visibility from reducing light-scattering particles. Previous RIAs for ozone (U.S. EPA, 2008a) and $PM_{2.5}$ (U.S. EPA, 2006a) indicate that visibility is an important benefit category, and previous efforts to monetize those benefits have only included a subset of visibility benefits, excluding benefits in urban areas and many national and state parks. Even this subset accounted for up to 5% of total monetized benefits in the Ozone NAAQS RIA (U.S. EPA, 2008a).

We were also unable to quantify the ecosystem benefits of reduced sulfur deposition because we lacked the necessary air quality data, and the methodology to estimate ecosystem benefits is still being developed. Previous assessments (U.S. EPA, 1999; U.S. EPA, 2005; U.S. EPA, 2009e) indicate that ecosystem benefits are also an important benefits category, but those efforts were only able to monetize a tiny subset of ecosystem benefits in specific geographic locations, such as recreational fishing effects from lake acidification in the Adirondacks.

In section 5.7 of this RIA, we discuss the revised presentation using benefits based on Pope et al. and Laden et al. as anchor points instead of the low and high end of the expert elicitation. This change was incorporated in direct response to recommendations from EPA's Science Advisory Board (U.S.EPA-SAB, 2008). Although using benefit-per-ton estimates limited our ability to incorporate all of their suggestions fully, we have incorporated the following recommendations into this analysis:

- Added "bottom line" statements where appropriate
- Clarified that the benefits results shown are not the actual judgments of the experts
- Acknowledged uncertainties exist at each stage of the analytic process, although difficult to quantify when using benefit-per-ton estimates
- Did not use the expert elicitation range to characterize the uncertainty as it focuses on the most extreme judgments with zero weight to all the others,
- Described the rationale for using expert elicitation in the context of the regulatory process (to characterize uncertainty)
- Identified results based on epidemiology studies and expert elicitation separately
- Showed central mass of expert opinion using graphs
- Presented the quantitative results using diverse tables and more graphics

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