Benefit Analyses of Alternative SAMI Strategies: Selected Health and Welfare Methods and Analysis Results

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Disclaimer

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Executive Summary

The Southern Appalachian Mountains Initiative (SAMI) commissioned an "integrated assessment" to assess the environmental effects and selected socioeconomic costs and benefits of SAMI-designed reduction strategies. Though four socioeconomic topics were considered in the assessment,[1] the topics were very narrow in scope in terms of the possible set of avoided health and welfare effects achievable under each control strategy. In fact, human mortality was the only health effect considered in the integrated assessment. This analysis is meant to provide an alternative evaluation of the same SAMI control strategies through the estimation of health benefits associated with SAMI-related pollution reductions.

The SAMI-designed emission reduction strategies proposed progressively more stringent emission reduction controls in each of five major source categories (utility, industrial, highway vehicle, non-road engines, and area sources) for 2010 and 2040. However, because human mortality was the only health effect considered, and because the relationship between mortality and exposures to air pollution is based on an annual average measure of PM2.5, SAMI did not estimate the full impact of each control strategy on the range of potential air pollutants. Instead, when converting these strategies into input data for the socioeconomic analyses, the SAMI atmospheric modeling contractor only provided Abt Associates with annual average PM2.5 profiles for each of three future control strategies. The first, and least stringent, strategy is referred to as the "A2" scenario, which serves as the base level of future-year air quality from which changes in air quality conditions are calculated. The remaining two scenarios are named the "B1" scenario and the "B3" scenario. These strategies serve as the control scenarios, where B1 is more stringent than A2, and B3 is more stringent than B1.

We base our analysis on the assumptions and models that have been approved by the EPA Science Advisory Board and are typically utilized by EPA to assess national regulatory programs. Specifically, this analysis relies upon the methods used in the analysis of EPA's Heavy Duty Engine/Diesel Fuel Rule and described in detail in the Heavy Duty Diesel Technical Support Document (Abt Associates, 2000). We estimate not only human mortality, but other health effects associated with exposures to annual measures of PM2.5. To account for unquantifiable benefits associated with the range of potential SAMI-related air quality improvements, we consider, qualitatively, the benefits associated with exposures to daily measures of PM2.5, PM10 and particulate matter between 2.5 and 10 microns (coarse PM10), NOx, SO2, ozone, and others.

Estimation of Health Effects

We estimated PM2.5-related health effects using the Criteria Air Pollutant Modeling System (CAPMS). CAPMS is a population-based system for modeling exposures of populations to ambient levels of criteria pollutants that we use to estimate health benefits. CAPMS divides the United States into eight kilometer by eight kilometer grid cells and estimates the changes in incidence of adverse health effects associated with given changes in air quality in each grid cell. Total incidence changes are the sum of grid cell-specific changes.

The SAMI annual average PM2.5 data came to Abt Associates at the modeled grid cell level; a nested grid structure comprised of an inner set of 12x12 km grid cells and an outer set of coarser 24x24 km grid cells. The data represented predicted annual average values at the center point of each grid cell. The SAMI modeling domain covered a geographical range that extended beyond the eight state SAMI

[1] The topics covered by the assessment include fishing, hiking/enjoying scenery, stewardship sense of place, and lifestyle changes.

region while not entirely covering the extent of the eight SAMI states. Coverage of the 8 state SAMI region was about 85%. This analysis limited the consideration of annual PM2.5 data to those grid cells whose centers fell within the eight state region. We then assigned each 8x8 km CAPMS grid cell to the nearest SAMI grid cell by calculating the shortest distance between the center of the CAPMS grid cell to the center of a SAMI grid cell.

Using the suite of health effect studies considered in the Heavy Duty Diesel analysis as our guide, we identified three annual average PM2.5-related endpoints for inclusion in the analysis; mortality associated with long-term PM2.5 exposures, chronic bronchitis, and acute bronchitis. Exhibit ES-1 contains details about each health effect, the study upon which the concentration-response function is based, and its associated valuation.

Exhibit ES-1 Annual Average PM2.5-Related Health Endpoints

Endpoint	Population	Study	Mean Estimate [a]	Uncertainty Distribution [a]
Mortality				
Associated with long-term exposure	Ages 30+	Krewski et al. (2000), reanalysis of Pope et al. (1995) using the annual mean and all-cause mortality	$6.324 million per statistical life	Weibull distribution, mean = $6.324 million; std. dev. = 4.27 million.
Chronic Illness				
Chronic Bronchitis	>26	Abbey et al. (1995b)	$340,568 per case	A Monte Carlo-generated distribution, based on three underlying distributions.
Respiratory Symptoms/Illnesses Not Requiring Hospitalization				
Acute bronchitis	Ages 8-12	Dockery et al. (1989)	$59.29 per case	Continuous uniform distribution over [$17.13, $101.45].

[a] The derivation of each of the estimates is discussed in the main text. All WTP-based dollar values were obtained by multiplying rounded 1990 $ values used in the §812 Prospective Analysis by 1.318 to adjust to 2000 $.

Health Effect Results: Incidence and Valuation

The total dollar benefit associated with a given endpoint depends on how much the endpoint will change (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth). Exhibit ES-2 summarizes the mean changes in incidence associated with each SAMI control scenario in each future year. Exhibit ES-3 summarizes the mean valuation (in 2000$) associated with the changes in incidence across all endpoints (mortality, chronic bronchitis, and acute bronchitis) for each SAMI control scenario in each future year.

We note that the benefits presented in Exhibit ES-3 include an adjustment for the impact of expected growth in real income on future year benefit estimates. The factors were calculated by EPA for use in the Heavy Duty Standards RIA (U.S. EPA, 2000), and are discussed fully in the main text.

Exhibit ES-2 Estimated Annual Average PM2.5-Related Health Effects Associated with Air Quality Changes Resulting from the SAMI Control Scenarios

Endpoint	Mean Avoided Incidence (cases/year)			
	2010 B1	2040 B1	2010 B3	2040 B3
Mortality	1,662	4,273	6,155	8,007
Chronic Bronchitis	1,258	3,303	4,531	6,051
Acute Bronchitis	3,464	8,952	12,192	16,177

Exhibit ES-3 Estimated Annual Average PM2.5-Related Health Benefits Associated with Air Quality Changes Resulting from the SAMI Control Scenarios

Endpoint	Mean Monetary Benefits (millions 2000$)			
	2010 B1	2040 B1	2010 B3	2040 B3
Mortality [a]	$11,114	$33,332	$41,163	$62,457
Chronic Bronchitis	$483	$1,508	$1,740	$2,763
Acute Bronchitis	$0.2	$0.6	$0.8	$1.1
Total	$11,597 + B [b]	$34,841 + B	$42,904 + B	$65,221 + B

[a] Calculated using a 3% discount rate in the mortality lag adjustment. See main text for the discussion on mortality lags.

[b] B represents benefits associated with the SAMI control scenarios but not captured by health effects estimated using the available annual average PM2.5 data. These benefits include avoided health effects associated with reductions in daily PM2.5, PM10 and coarse PM10, ozone, NO2, SO2, and CO, hazardous air pollutants, and nitrogen deposition. B also represents benefits associated with improvements in visibility and recreational fishing, calculated for the SAMI Integrated Assessment.

Uncertainty

As with any complex analysis such as this one, there are a wide variety of sources for uncertainty. Some key sources of uncertainty in each stage of the benefits include:

- gaps in scientific data and inquiry;
- variability in estimated relationships, such as C-R functions introduced through differences in study design and statistical modeling;
- errors in measurement and projections for variables such as population growth rates;
- errors due to misspecification of model structures, excluded variables, and simplification of complex functions;
- biases due to omissions or other research limitations.

The above benefits are considered primary estimates for this analysis, based on the best available scientific literature and methods. Where possible, we attempt to provide estimates of the effects of uncertainty about key analytical assumptions. In the main text, we address uncertainty by presenting alternative calculations, sensitivity analyses, and probabilistic assessments associated with the annual average PM2.5-related health effects. They include:

- Alternative Calculations - Estimates of mortality based on alternative studies; Valuation of avoided premature mortality incidence based on statistical life years; Age-based adjustments to the value of a statistical life lost; Estimation and valuation of reversals in chronic bronchitis.
- Sensitivity Analyses - Calculation of the impact varying threshold assumptions have on the estimation of mortality incidence; Calculation of the impact different lag structures have on the estimation of benefits associated with avoided mortality incidence.
- Statistical Uncertainty Bounds - The total dollar benefit associated with a given endpoint depends on how much the endpoint will change due to the assumptions in the control scenarios (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth). Based on these distributions, we use Monte Carlo methods to provide estimates of the 5^{th} and 95^{th} percentile values of the distribution of estimated health effect endpoint incidence and valuation.

Unquantified Benefits From Other Pollutant Reductions

One significant limitation of the SAMI health benefits analyses is the inability to quantify many of the adverse effects associated with exposures to pollutants other than annual average PM2.5. Though estimates of PM2.5-related mortality and chronic and acute bronchitis may have captured the bulk of the economic benefits associated with reducing emissions in the SAMI region, we still miss a variety of potential benefits because there are a limited number of epidemiological studies based on annual PM2.5. Benefits missed in the SAMI analysis likely include:

- Other PM Effects (daily PM2.5, PM10 and coarse PM10) - In analyses conducted for the EPA, benefit estimates related to hospital admissions, emergency room visits, lower and upper respiratory symptoms, work loss days, MRADs, and recreational visibility improvements have equaled between 3 to 5% of benefits related to annual average PM2.5 effects.
- Ozone Effects - Across the same EPA analyses, benefits of ozone related hospital admissions, emergency room visits, MRADs, decreased worker productivity and agricultural crop losses have equaled between 2 to 24% of benefits related to annual average PM2.5 effects.
- NO2, SO2, and CO Effects - These pollutants are generally related to a small subset of effects; the most serious of which is perhaps hospitalization for heart-related problems. There have been studies finding some evidence that NO2 and CO are linked to mortality but it is difficult to determine if these effects are in addition to effects associated with PM and ozone.
- Effects of Air Toxics - Air toxics encompass a broad range of harmful chemical compounds that are either released directly into the air or formed in secondary reactions in the air, water, and soil. Exposure to air toxics can result in cancer, noncancer health effects, and ecological damage. The large number of air toxics, and the difficulties associated with estimating the impact of changes in emissions of air toxics, make these effects extremely hard to quantify.
- Nitrogen Deposition Effects - Excess nutrient loads, especially that of nitrogen, are responsible for a variety of adverse consequences to the health of estuarine and coastal waters. These effects include: toxic and/or noxious algal blooms such as brown and red tides; low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters; the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats; and fundamental shifts in phytoplankton community structure.

1. Introduction

In 1992, the Southern Appalachian Mountains Initiative (SAMI) was created to "identify and recommend reasonable measures to remedy existing and prevent future adverse effects from human-induced air pollution on the air quality related values of the Southern Appalachians, primarily those of Class I parks and wilderness areas, weighing the environmental and socioeconomic implications of any recommendations"(SAMI 2001). To do this, SAMI commissioned an "integrated assessment" to estimate the environmental effects and selected socioeconomic costs and benefits of SAMI-designed emissions reduction strategies. Four socioeconomic topics are covered by the integrated assessment, including: 1) fishing; 2) hiking/enjoying scenery; 3) stewardship/sense of place; and 4) lifestyle changes.

The SAMI emission strategies were designed to propose progressively more stringent emissions reduction controls in each of five major source categories (utility, industrial, highway vehicle, non-road engines, and area sources) for 2010 and 2040. Converting these strategies into input data for the socioeconomic analyses, the SAMI atmospheric modeling contractor generated air quality profiles for each of three future control strategies. Each strategy represented a series of different assumptions, including different applied control technologies, implementation of regulations and incentives, and demand for goods and services. The first, and least stringent, strategy is referred to as the "A2" scenario, or the reference strategy. This scenario served as the base level of future-year visual air quality from which changes in air quality conditions are calculated.[2] The remaining two scenarios are named the "B1" scenario and the "B3" scenario. These strategies served as the control scenarios, where B1 was more stringent (i.e., lower emissions) than A2, and B3 was more stringent than B1. A complete description of the assumptions present within each of these emission reduction strategies can be found in the 2001 SAMI Interim Report.

Though four socioeconomic topics were considered in the integrated assessment, the topics were very narrow in scope in terms of the possible set of avoided health and welfare effects achievable under each control strategy.[3] In fact, human mortality was the only health effect considered until it was removed from the SAMI integrated assessment. Before it was removed, however, the SAMI air modeling contractor provided air quality inputs for the mortality analysis. Yet, because the relationship between mortality and exposures to air pollution is based on an annual average measure of $PM_{2.5}$, the atmospheric modelers did not estimate the full impact of each control strategy on the range of potential air pollutants. Instead, only annual $PM_{2.5}$ was provided, though improvements in ozone, sulfur dioxide (SO_2), nitrogen oxide (NOx), PM_{10} and its associated coarse fraction (coarse PM_{10}) were likely to occur.

This analysis is meant to provide an alternative evaluation of the same SAMI control strategies through the estimation of the health benefits associated with reductions in annual $PM_{2.5}$. This includes not only human mortality, but other health effects associated with exposures to annual measures of $PM_{2.5}$. We base our analysis on the assumptions and models that have been approved by the EPA Science Advisory Board and are typically utilized by EPA to assess national regulatory programs. Specifically, this analysis relies upon the methods used in the analysis of EPA's Heavy Duty Engine/Diesel Fuel Rule

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[2] Benefits are calculated based on measured changes in air quality between a future-year base-case scenario and a future-year control scenario.

[3] The SAMI Integrated Assessment estimated recreational visibility benefits related to visual air quality improvements associated with the SAMI emission control scenarios. The Integrated Assessment also estimated recreational fishing benefits related to water quality improvements associated with the SAMI emission control scenarios. Both of these benefit categories are added to the health benefits we calculate in this analysis to capture the magnitude of total benefits associated with the SAMI emission control scenarios. Section 5 presents total benefit results.

and described in detail in the Heavy Duty Diesel Technical Support Document (Abt Associates, 2000). To account for unquantifiable benefits associated with the potential reduction, we also present a qualitative discussion of benefits associated with exposures to daily measures of $PM_{2.5}$, PM_{10} and particulate matter between 2.5 and 10 microns (coarse PM_{10}), NOx, SO_2, ozone, and others.

Section 2 describes the method used to develop the PM air quality inputs for use in the benefits analysis. Section 3 describes general issues arising in estimating and valuing changes in adverse health effects associated with changes in PM. Section 4 describes in some detail the methods used for estimating and valuing adverse health effects, while Section 5 presents the results of these analyses. Section 6 presents a discussion of the unquantified benefits from other pollutant reductions that are likely associated with the SAMI emission control scenarios.

This document also has two appendices. Appendix A presents the physical and monetary benefits associated with sensitivity calculations for the SAMI emission control scenarios not considered in the primary analysis. Appendix B presents the PM C-R functions used in this analysis.

2. Development of PM Air Quality Inputs For Use in the Benefits Analysis

The following section summarizes how we use the SAMI annual average $PM_{2.5}$ air quality model results in conjunction with the Criteria Air Pollutant Modeling System (CAPMS) to estimate $PM_{2.5}$ exposure.

CAPMS is a population-based system for modeling exposures of populations to ambient levels of criteria air pollutants that we use to estimate health benefits.[4] CAPMS divides the United States into eight kilometer by eight kilometer grid cells, and estimates the changes in incidence of adverse health and welfare effects associated with given changes in air quality in each grid cell. We then calculate the total incidence change as the sum of grid-cell-specific changes.

Contractors for the SAMI integrated assessment forecasted annual average $PM_{2.5}$ data associated with each of the future-year (2010 and 2040) emission control scenarios; EPA provided this data, in spreadsheet format, to Abt Associates. Data was provided at the modeled grid cell level; a nested grid structure comprised of an inner set of 12x12 km grid cells and an outer set of coarser 24x24 km grid cells. Location information (i.e., latitude and longitude) was also provided. The data represented predicted annual average values at the center point of each grid cell.

The modeled air quality data predicted for the SAMI analysis, and used here, covers a geographical range that extends beyond the eight state SAMI region while not entirely covering the extent of the eight SAMI states. In fact, coverage of the 8 State SAMI region is about 85%. Some counties did not appear in the database because they were outside the 12x12 or 24x24 grid cell areas. These excluded areas include: southern Alabama and Georgia and western Tennessee and Kentucky. This analysis limited the consideration of annual $PM_{2.5}$ data to those grid cells whose centers fell within the eight state SAMI region. We then assigned each 8x8 km CAPMS grid cell to the nearest SAMI grid cell by calculating the shortest distance between the center of the CAPMS grid cell to the center of a SAMI grid cell.

Exhibit 2-1 presents the population-weighted average change in annual mean PM2.5 between each of the SAMI emission control scenarios and their respective baselines. For the sake of comparison, we have also included the same measure of PM2.5 from a recent analysis, the Heavy Duty Engine/Diesel Fuel Rule Analysis.

Exhibit 2-1 Changes in Annual Mean PM2.5 Due to SAMI Emission Control Scenarios

Statistic	Annual Mean PM2.5 by Scenario (ug/m3)				
	2010 B1	2040 B1	2010 B3	2040 B3	2030 HDD Analysis
Population-Weighted Average Change from Baseline	1.14	2.43	4.22	4.54	0.65

[4] CAPMS does not model individual exposures to these pollutants. For a complete description of CAPMS, please refer to the Heavy Duty Diesel Technical Support Document (Abt Associates, 2000).

3. General Issues in Estimating Health and Welfare Benefits

Changes in PM result in changes in a number of health effects, or "endpoints," that society values. The Heavy Duty Diesel Analysis Technical Support Document (HDD TSD) (Abt Associates, 2000) discussed key issues in the estimation of adverse health effects and in the valuation of health and welfare benefits. For common issues between the Heavy Duty Diesel Analysis and the SAMI analysis, we refer the reader to the HDD TSD. Exhibit 3-1 lists these common issues. For issues specific to the estimation of benefits for the SAMI analysis, we include their discussion below.

Exhibit 3-1 General Issues in Estimating Health and Welfare Benefits

1. Estimating Adverse Health Effects
-The basic concentration-response model.
-Calculation of adverse health effects with CAPMS.
-Overlapping health effects.
-Baseline incidences.
-Thresholds.
-Application of a single C-R function everywhere.
-Estimating pollutant-specific benefits using single pollutant vs. multi-pollutant models.
2. Valuing Changes in Health and Welfare Effects
-Willingness-to-Pay estimation.
-Aggregation of monetized benefits.

Because this analysis is limited to the evaluation of effects related to PM2.5, we have also tailored the characterization of uncertainty to reflect uncertainties associated with PM2.5-related health effects. We again refer the reader to the HDD TSD for the discussion of uncertainty characterization associated with daily average PM2.5- and PM10-related endpoints.

3.1 Population Projections

Benefits for the SAMI analysis are based on health effect incidence changes due to predicted air quality improvements in the years 2010 and 2040. Integral to the estimation of such benefits is an accurate estimate of future population projections.

The underlying data used to create county-level 2040 population projections is based on: (1) 1990 county-level population statistics for all U.S. counties collected by the U.S. Census (Wessex, 1994), and (2) future-year state and metropolitan area population estimates provided by the Bureau of Economic Analysis (1995). Growth factors are calculated using the BEA data and are applied to the 1990 county-level populations.

A growth factor is calculated by taking the ratio of an estimated region's 2030 population divided by the1990 population for that same area. Population estimates for the years 1990-93, 2000, 2005, 2010, 2015, 2025 and 2045 were collected by the BEA. A 2040 population estimate was not provided. Instead, 2040 state and metropolitan area populations were interpolated linearly using estimates from the years 2025 and 2045.

Growth factors are calculated for both urban areas and rural areas. An urban area is defined as a county that falls within a metropolitan area. This includes metropolitan statistical areas (MSAs), primary metropolitan statistical areas (PMSAs), consolidated metropolitan statistical areas (CMSAs), and New England county metropolitan areas (NECMAs), as defined by U.S. Census Bureau.[5] In this section, however, all metropolitan areas are referred to as MAs. A rural area is defined as a county that falls outside the defined metropolitan areas.

Urban areas grow according to the growth rate calculated for the particular metropolitan area within which they are located. This adjustment is very straightforward, simply taking the ratio of future year to base year metropolitan area population and multiplying that factor by the base year county population. The equation is:

$$FutureCountyPop_i = 1990CountyPop_i \cdot \frac{FutureMAPop_i}{1990\,MAPop_i}$$

where:

$FutureCountyPop_i$ = projected 2010 or 2040 population in urban county i
$1990CountyPop_i$ = actual 1990 population for county i
$FutureMAPop_i$ = projected 2010 or 2040 population in metropolitan area for county i
$1990MAPop_i$ = actual 1990 population for metropolitan area for county i.

Rural areas grow according to the growth rate calculated for the particular state within which they are located, adjusted to subtract out metropolitan area populations. Before the ratio of future year to base year state population is calculated, the population attributed to all metropolitan areas located within that state is subtracted from the future year and base year population totals. Once this metropolitan area adjustment has been made, the rural growth factor is multiplied by the base-year population in all non-MA counties to get future-year population projections.

To calculate the future year population, we use the following equation:

$$FutureCountyPop_i = 1990CountyPop_i \cdot \frac{(FutureStatePop_i - \sum FutureMAPop_i)}{(1990StatePop_i - \sum 1990\,MAPop_i)}$$

where:

$FutureCountyPop_i$ = projected 2010 or 2040 population in rural county i
$1990CountyPop_i$ = actual 1990 population for county i
$FutureStatePop_i$ = projected 2010 or 2040 population in state where county i is located
$1990State\ Pop_i$ = actual 1990 population for state where county i is located
$\Sigma FutureMAPop_i$ = projected 2010 or 2040 population in metropolitan areas located in state with county i
$\Sigma 1990MAPop_i$ = actual 1990 population for metropolitan areas located in state with county i .

One problem that exists with this method is that many metropolitan areas cross state boundaries. To accurately subtract urban populations from state populations, we need to know the urban county populations for both 1990 and the future year. Using the county populations for 1990, we can estimate the portion of a particular metropolitan area's population that belongs to a given state. However, we do

[5] The Census Bureau definitions are available at: http://www.census.gov/population/www/estimates/aboutmetro.html .

not have future year county population projections with which to apportion future year metropolitan area populations. To remedy this, we apply the same percent of the population a given county contributes to a metropolitan area in 1990 to 2010 and 2040 metropolitan areas when apportioning populations between states.

The above procedure refers to population estimates at the county level. CAPMS, however, apportions population estimates to the CAPMS grid cell level. To do this, CAPMS uses census-derived 1990 block group population estimates. Each block group has a centroid. For each centroid that is located within a CAPMS grid cell, the grid cell is assigned that population. To inflate 1990 population estimates to a future year estimation of population within a CAPMS grid cell, county level ratios, calculated using the county level estimates described above, are applied to CAPMS grid cells that fall within a particular county. There are a few inaccuracies with this procedure. CAPMS grid cells and census block groups do not share similar borders. When a block group centroid is assigned to a CAPMS grid cell, there may be some overlap with other grid cells. The total block group population, however, is assigned only to the CAPMS grid cell in which it is located. A similar issue exists when assigning county-level ratios to CAPMS grid cells. The county in which a grid cell is located is determined by the grid cell center. However, the grid cell center may overlap with other counties. Both issues may lead to the assignment of populations or adjustment factors to the wrong area. The overall magnitude of the discrepancy, however, is slight because of the small area each of the block groups and grid cells represent.

3.2 Change Over Time in Benefit Value in Real Dollars

The value placed on benefits, or willingness to pay (WTP), for health-related environmental improvements (in real dollars) could change between now and the years 2010 and 2040. If real income increases between now and the year 2040, for example, it is reasonable to expect that WTP, in real dollars, would also increase. Based on historical trends, the U.S. Bureau of Economic Analysis projects that, for the United States as a whole as well as for regions and states within the U.S., mean per capita real income will increase. For the U.S. as a whole, for example, mean per capita personal income is projected to increase by about 16 percent from 1993 to 2005 (U.S. Bureau of Economic Analysis, 1995).

The monetary benefits presented in this Technical Support Document (TSD) have been adjusted to account for changes over time in real income. A complete description of the theory behind the income growth adjustment and its application to the benefits analysis can be found in Chapter 7 of the Heavy Duty Diesel Regulatory Impact Analysis (EPA 2000b). Exhibit 3-2 displays the adjustment factors used in this analysis.

Exhibit 3-2 Income Growth Adjustment Factors

Year	Endpoint		
	Minor Illness	Severe/Chronic Illness	Mortality
2010	1.0380	1.1274	1.1124
2040	1.0949	1.3407	1.2972

3.3 Adjusting Benefit Estimates from 1990 Dollars to 2000 Dollars

This section describes the method used to convert benefits estimates into constant dollars. In past RIA analyses performed for the EPA, cost and benefit estimates have been presented in constant 1990 dollars. Benefits estimates in this analysis, however, are presented in constant 2000 dollars. To adjust benefits estimates from 1990 dollars to 2000 dollars, the method of adjustment depends on the basis of the benefits estimates.[6] For the SAMI analysis, all of the benefit estimates are based on direct estimates of WTP.

Benefit estimates based directly on estimates of WTP have been adjusted using the CPI-U for "all items." The CPI-Us, published by the U.S. Dept. of Labor, Bureau of Labor Statistics, can be found in Council of Economic Advisers (2000, Table B-58). An overview of the adjustments from 1990 to 2000 dollars for WTP-based valuations is given in Exhibit 3-3.

Exhibit 3-3 Consumer Price Indexes Used to Adjust WTP-Based and Cost-of-Illness-Based Benefits Estimates from 1990 Dollars to 2000 Dollars

	1990 (1)	2000 (2)	Adjustment Factor [a] (2)/(1)	Relevant Endpoints
CPI-U for "All Items" [b]	130.7	172.2	1.3175	WTP-based valuation: 1. Statistical lives saved [c] 2. Chronic bronchitis 3. Morbidity endpoints using WTP [d]

[a] Benefits estimates in 1990 dollars are multiplied by the adjustment factor to derive benefits estimates in 2000 dollars.

[b] Source: Dept. of Labor, Bureau of Labor Statistics; reported in Council of Economic Advisers (2000, Table B-58)

[c] Adjustments to 1990 S were originally made by Industrial Economics Inc. using the CPI-U for "all items" (IEc1992).

[d] Adjustments of WTP-based benefits for morbidity endpoints to 1990 S were originally made by Industrial Economics Inc. (1993) using the CPI-U for "all items."

3.4 Characterization of Uncertainty

In any complex analysis using estimated parameters and inputs from numerous different models, there are likely to be many sources of uncertainty. This analysis is no exception. There are many inputs that are used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological estimates of C-R functions, estimates of values, population estimates, income estimates, and estimates of the future state of the world, i.e. regulations, technology, and human behavior. Each of these inputs may be uncertain, and depending on their location in the benefits analysis, may have a disproportionately large impact on final estimates of total benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emissions can lead to much larger impacts on total benefits.

[6] For example, benefit analyses in the past have included estimates based on direct estimates of WTP, cost of illness, and earnings. For each, the adjustment from 1990 to 2000 dollars is different.

Exhibit 3-4 summarizes the wide variety of sources for uncertainty in this analysis. Some key sources of uncertainty in each stage of the benefits analysis are:

- gaps in scientific data and inquiry
- variability in estimated relationships, such as C-R functions, introduced through differences in study design and statistical modeling
- errors in measurement and projection for variables such as population growth rates
- errors due to misspecification of model structures, excluded variables, and simplification of complex functions
- biases due to omissions or other research limitations.

Our approach to characterizing model uncertainty in the estimate of total benefits is to present a primary estimate, based on the best available scientific literature and methods, and to provide estimates of the effects of uncertainty about key analytical assumptions. However, in some cases, it was not possible to quantify uncertainty. For example, many benefits categories, while known to exist, do not have enough information available to provide a quantified or monetized estimate. The uncertainty regarding these endpoints is such that we could determine neither a primary estimate nor a plausible range of values.

Another source of uncertainty related to the SAMI analysis is the limited air quality data available with which to conduct the benefit analysis. Only those health effects associated with annual mean $PM_{2.5}$, and for which a C-R function was available, were included in the analysis. However, the SAMI control scenarios, "B1" and "B3", project that there will also be reductions in PM_{10}, ozone, NOx and SO_2. To the extent that health effects are related to these other pollutant reductions, the current SAMI analysis will underestimate the total benefits associated with the alternative SAMI pollution control scenarios. The extent to which benefits are underestimated, however, is impossible to determine, however Section 6 discusses in detail the health effects associated with exposures to these additional pollutants and their relative contribution to total benefits in other policy analyses.

This report also addresses uncertainty by presenting alternative calculations, sensitivity analyses, and probabilistic assessments associated with the annual average $PM_{2.5}$-related health effects. We discuss each approach in turn.

Exhibit 3-4 Key Sources of Uncertainty in the Benefit Analysis

1. Uncertainties Associated With Concentration-Response Functions
-The value of the PM-coefficient in each C-R function.
-Application of a single C-R function to pollutant changes and populations in all locations.
-Similarity of future year C-R relationships to current C-R relationships.
-Correct functional form of each C-R relationship.
-Extrapolation of C-R relationships beyond the range of PM concentrations observed in the study.
-Application of C-R relationships only to those subpopulations matching the original study population.
2. Uncertainties Associated With PM Concentrations
-Responsiveness of the models to changes in precursor emissions resulting from the control policy.
-Projections of future levels of precursor emissions, especially ammonia and crustal materials.
-Model chemistry for the formation of ambient nitrate concentrations.
3. Uncertainties Associated with PM Mortality Risk
-No scientific literature supporting a direct biological mechanism for observed epidemiological evidence. -Direct causal agents within the complex mixture of PM have not been identified. -The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures. -Possible confounding in the epidemiological studies of $PM_{2.5}$ effects with other factors (e.g., other air pollutants, weather, indoor/outdoor air, etc.). -The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study. -Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated With Possible Lagged Effects
-The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
5. Uncertainties Associated With Baseline Incidence Rates
-Some baseline incidence rates are not location-specific (e.g., those taken from studies) and may therefore not accurately represent the actual location-specific rates.
-Current baseline incidence rates may not approximate well baseline incidence rates in 2010 or 2040.
-Projected population and demographics may not represent well future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
-Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them.
-Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors.
7. Uncertainties Associated With Aggregation of Monetized Benefits
-Health benefit estimates are limited to the available C-R functions. Thus, unquantified or unmonetized benefits are not included.
-Health benefit estimates are limited to the available air quality data. Though only annual mean PM2.5 is considered in this analysis, the SAMI control scenarios will also bring reductions in other pollutants, such as PM10, ozone, NOx and SO2. The avoided health effects associated with these reductions, though potentially significant, are not quantified in this analysis.

3.4.1 Alternative Calculations

The alternative calculations included in this analysis are based on relatively plausible alternatives to the assumptions used in deriving the primary benefit estimates. We do not attempt to assign probabilities to these alternative calculations, as we believe this would only add to the uncertainty of the

analysis or present a false picture about the precision of the results.[7] Instead, the reader is invited to examine the impact of applying the different assumptions on the estimate of total benefits. While it is possible to combine all of the alternative calculations with a positive impact on benefits to form a "high" estimate or all of the alternative calculations with a negative impact on benefits to form a "low" estimate, we do not recommend this because the probability of all of these alternative assumptions occurring simultaneously is likely to be very low. Instead, the alternative calculations are intended to demonstrate the sensitivity of our benefits results to key parameters which may be uncertain. Exhibit 3-5 summarizes the alternative calculations included in this analysis.

Studies Used for Alternative Calculations

A number of studies that estimate plausible alternative relationships between PM exposure and premature mortality are presented as alternative calculations to the mortality study included in the primary analysis (Krewski et al., 2000, mean all-cause mortality). These alternative mortality functions are discussed in more detail in Section 4.

The value of statistical life years alternative calculation recognizes that individuals who die from air pollution related causes tend to be older than the average age of individuals in the VSL studies used to develop the $5.9 million value. To employ the value of statistical life-year (VSLY) approach, we first estimated the age distribution of those lives projected to be saved by reducing air pollution. Based on life expectancy tables, we calculate the life-years saved from each statistical life saved within each age and gender cohort. To value these statistical life-years, we hypothesized a conceptual model which depicted the relationship between the value of life and the value of life-years. The average number of life-years saved across all age groups for which data were available is 14 for PM-related mortality. The average for PM, in particular, differs from the 35-year expected remaining lifespan derived from existing wage-risk studies. Using the same distribution of value of life estimates used above, we estimated a distribution for the value of a life-year and combined it with the total number of estimated life-years lost.

An alternative to the calculation of life-years lost is age-based adjustments to the value of a statistical life lost based on empirical estimates of WTP by age. Several studies conducted by Jones-Lee, et al. (1985; 1989; 1993) found a significant effect of age on the value of mortality risk reductions expressed by citizens in the United Kingdom. We used the results of the Jones-Lee et al. analysis to calculate age-specific values of a statistical life. As described below, we started with the value of a statistical life lost by an individual of about age 40, and then adjusted it with age-specific factors. We use 40 as the base because we use wage risk studies in developing the value of a statistical life, and the average age in the wage-risk studies is about 40.

We apportioned the number of lives saved in each of the age groups used in the statistical life-years-lost alternative calculation to the age groups used by Jones-Lee et al. (1989; 1993). We then multiplied the number of lives saved in an age group by the age-adjusted value of a statistical life saved for that age group. To calculate the value of a statistical life saved in an age group, we multiplied $6.12 million by the ratio of the WTP for mortality risk reduction in that age group to the WTP for mortality risk reduction in the age 40-59 group, as reported by Jones-Lee et al. (1989; 1993). The five-year lag structure used in the primary method is also applied under two alternative discount rate assumptions of

[7] Some recent benefit-cost analyses in Canada and Europe (Lang et al., 1995; Holland et al., 1999) have estimated ranges of benefits by assigning *ad hoc* probabilities to ranges of parameter values for different endpoints. Although this does generate a quantitative estimate of an uncertainty range, the estimated points on these distributions are themselves highly uncertain and very sensitive to the subjective judgements of the analyst. To avoid these subjective judgements, we choose to allow the reader to determine the weights they would assign to alternative estimates.

three percent and seven percent. Because the two Jones-Lee studies reported different ratios, this alternative calculation is carried out separately using each of the two Jones-Lee studies.

Reversals in chronic bronchitis incidences are defined as those cases where an individual reported having chronic bronchitis at the beginning of the study period but reported not having chronic bronchitis in follow-up interviews at a later point in the study period. Since, by definition, chronic diseases are long-lasting or permanent, if the disease goes away it is not chronic. In the primary analysis, these reversals are given a value of zero. As an alternative calculation, we estimate reversals and value each as a case of the mildest form of chronic bronchitis.

Exhibit 3-5 Alternative Benefits Calculations for the SAMI Benefit Analyses

Alternative Calculations	Description
PM-related premature mortality	A number of studies provide an alternative estimate of the relationship between chronic PM exposure and mortality.
Value of avoided premature mortality incidences based on statistical life years.	Calculate the incremental number of life-years lost from exposure to changes in ambient PM and use the value of a statistical life year based on a $5.9 million value of a statistical life.
Age-based adjustments to the value of a statistical life lost	Results of the Jones-Lee et al. (1985; 1989; 1993) analysis were used to calculate age-based adjustment factors to adjust the value of a statistical life lost by an individual of about age 40 to age-specific values.
Reversals in chronic bronchitis treated as lowest severity cases	Instead of omitting those cases of chronic bronchitis that reverse after a period of time, they are treated as being cases with the lowest severity rating.

3.4.2 Sensitivity Analyses

In addition to alternative calculations, we perform sensitivity analyses, briefly described in Exhibit 3-6. Sensitivity analyses, as opposed to alternative calculations, examine the sensitivity of estimated benefits results to less plausible alternatives to the assumptions used in the primary analysis. Sensitivity calculations also demonstrate the sensitivity of our benefits results to key analytical parameters. The sensitivity analyses calculated for this analysis includes an examination of how a PM concentration threshold could influence mortality incidence estimates, and alternative lag structures when valuing mortality. Results from the sensitivity analyses are presented in Appendix A.

Exhibit 3-6 Sensitivity Analyses for the SAMI Benefit Analyses

Sensitivity Analysis	Description
Threshold assumptions	Calculate the impact varying threshold assumptions have on the estimation of mortality incidence based on the Krewski et al. (2000) study.
Alternative mortality lag structures	Calculate the impact different lag structures have on the estimation of benefits associated with avoided mortality incidence.

3.4.3 Statistical Uncertainty Bounds

Although there are several sources of uncertainty affecting estimates of endpoint-specific benefits, the sources of uncertainty that are most readily quantifiable in this analysis are the C-R

relationships and uncertainty about unit dollar values. The total dollar benefit associated with a given endpoint depends on how much the endpoint will change due to the SAMI emission control scenarios (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth).[8] Based on these distributions, we provide estimates of the 5th and 95th percentile values of the distribution of estimated benefits. However, we hasten to add that this omits important sources of uncertainty, such as the contribution of air quality changes, baseline population incidences, projected populations exposed, transferability of the C-R function to diverse locations, and uncertainty about premature mortality. Thus, a confidence interval based on the standard error would provide a misleading picture about the overall uncertainty in the estimates. The empirical evidence about uncertainty is presented where it is available.

Both the uncertainty about the incidence changes and uncertainty about unit dollar values can be characterized by *distributions*. Each "uncertainty distribution" characterizes our beliefs about what the true value of an unknown (e.g., the true change in incidence of a given health effect) is likely to be, based on the available information from relevant studies.[9] Unlike a sampling distribution (which describes the possible values that an *estimator* of an unknown value might take on), this uncertainty distribution describes our beliefs about what values the unknown value itself might be. Such uncertainty distributions can be constructed for each underlying unknown (such as a particular pollutant coefficient for a particular location) or for a function of several underlying unknowns (such as the total dollar benefit of a regulation). In either case, an uncertainty distribution is a characterization of our beliefs about what the unknown (or the function of unknowns) is likely to be, based on all the available relevant information. Uncertainty statements based on such distributions are typically expressed as 90 percent credible intervals. This is the interval from the fifth percentile point of the uncertainty distribution to the ninety-fifth percentile point. The 90 percent credible interval is a "credible range" within which, according to the available information (embodied in the uncertainty distribution of possible values), we believe the true value to lie with 90 percent probability.

The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from these two sources, and is estimated with a Monte Carlo method. In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution and a value is randomly drawn from the unit dollar value distribution, and the total dollar benefit for that iteration is the product of the two.[10] If this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated.

Using this Monte Carlo procedure, a distribution of dollar benefits may be generated for each endpoint. The mean and median of this Monte Carlo-generated distribution are good candidates for a point estimate of total monetary benefits for the endpoint. As the number of Monte Carlo draws gets larger and larger, the Monte Carlo-generated distribution becomes a better and better approximation to the underlying uncertainty distribution of total monetary benefits for the endpoint. In the limit, it is identical to the underlying distribution.

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

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

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

3.4.4 Unquantified Benefits

In considering the monetized benefits estimates, the reader should remain aware of the limitations. One significant limitation of the SAMI health benefits analyses is the inability to quantify many of the adverse effects associated with exposures to pollutants other than PM2.5. Section 6 discusses these unquantified benefits in detail. Another limitation of the SAMI analyses is that for many additional health and welfare effects associated with exposures to PM, such as PM-related materials damage, reliable C-R functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefits categories, the benefits estimates presented in this RIA would increase. In addition to unquantified benefits, there may also be environmental costs that we are unable to quantify. Several of these environmental cost categories are related to nitrogen deposition, while one category is related to the issue of ultraviolet light. The net effect of excluding benefit and disbenefit categories from the estimate of total benefits depends on the relative magnitude of the effects.

4. Health Benefits

Typically, in benefit analyses of air policies, the most significant monetized benefits of reducing ambient concentrations of PM are associated with reductions in health risks associated with the fine particulate portion of PM, $PM_{2.5}$. This Section describes individual effects and the methods used to quantify and monetize changes in the expected number of incidences of various health effects related to changes in $PM_{2.5}$. Though only effects associated with annual mean $PM_{2.5}$ are valued in this analysis, many additional benefits are associated with incidence changes associated with reductions in other pollutants like ozone, PM_{10}, NOx, and SO_2. Yet, because we only have data for $PM_{2.5}$, these other benefits can not be quantified. We discuss these endpoints qualitatively in Section 6.

We estimate the incidence of adverse health effects using C-R functions based on $PM_{2.5}$. The changes in incidence of PM-related adverse health effects and corresponding monetized benefits associated with these changes are estimated separately. The PM-related health endpoints for which C-R functions are estimated are shown in Exhibit 4-1. The unit monetary values for each of these endpoints, and associated uncertainty distributions, are presented in Exhibit 4-2. Issues relating to the calculation of changes in incidence and the monetization of these changes are discussed below for each endpoint.

Note also that in some cases there are alternative endpoints, studies, or unit dollar values that could be used in calculating the benefits of a change in pollution. Again, this analysis follows the methodology the EPA's Heavy Duty Diesel Analysis and the Section 812 Analysis, which have undergone extensive review and critique. In following this methodology, these alternatives are presented where appropriate in Exhibits 4-1 and 4-2 in italics to indicate that they are not used in the primary analysis but may be used in alternative analyses. Appendix B presents the functional forms for each C-R function and how they were derived.

Exhibit 4-1 PM-Related Health Endpoints

Endpoint	Population	PM	Study
Mortality			
Associated with long-term exposure	Ages 30+	$PM_{2.5}$	Krewski et al. (2000), reanalysis of Pope et al. (1995) using the annual mean and all-cause mortality
Associated with long-term exposure[a]	*Ages 30+*	*$PM_{2.5}$*	*Krewski et al. (2000), reanalysis of Dockery et al. (1993)*
Associated with long-term exposure	*Ages 27+*	*$PM_{2.5}$*	*Dockery et al. (1993)*
Chronic Illness			
Chronic Bronchitis	>26	$PM_{2.5}$	Abbey et al. (1995b)
Respiratory Symptoms/Illnesses Not Requiring Hospitalization			
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1989)

[a] Italicized entries are either alternative or supplemental calculations to the endpoints and/or studies used in the primary analysis.

Exhibit 4-2 Unit Values for Economic Valuation of Health Endpoints (2000 $)

Health Endpoint	Mean Estimate [a]	Assumed Uncertainty Distribution [a]
Mortality		
Value of a statistical life	$6.324 million per statistical life	Weibull distribution, mean = $6.324 million; std. dev. = 4.27 million.
Chronic Bronchitis		
WTP approach	$340,568 per case	A Monte Carlo-generated distribution, based on three underlying distributions.
Respiratory Ailments Not Requiring Hospitalization		
Acute bronchitis	$59.29 per case	Continuous uniform distribution over [$17.13, $101.45].

[a] The derivation of each of the estimates is discussed in the text. All WTP-based dollar values were obtained by multiplying rounded 1990 $ values used in the §812 Prospective Analysis by 1.318 to adjust to 2000 $. Entries in italics are not used in the primary benefits analysis.

4.1 Premature Mortality

Health researchers have consistently linked air pollution, especially PM, with increases in premature mortality. A substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates. For instance, studies have found associations between day-to-day particulate air pollution and increased risk of various adverse health outcomes, including cardiopulmonary mortality (Pope et al., 2000). Fine particles, $PM_{2.5}$, are the largest health concern because they can be breathed most deeply into the lung. Much of this literature is summarized in 1996 PM Criteria Document (US EPA, 1996) and the Heavy Duty Diesel Regulatory Impact Analysis (US EPA, 2000).

These epidemiological studies typically estimate the relationship between air quality changes and the *relative risk* of a health effect, rather than estimate the absolute number of avoided cases of premature mortality. For example, the risk of mortality at ambient PM level x_0 relative to the risk of mortality at ambient PM level x may be characterized by the ratio of the two mortality rates: the mortality rate among individuals when the ambient PM level is x_0 and the mortality rate among (otherwise identical) individuals when the ambient PM level is x. We incorporate the relative risk into a concentration response function so that the effects of changes in PM concentrations on mortality can be estimated by a count of the expected number of deaths avoided due to a given reduction in PM concentrations. An alternative measure is to infer the number of years of life that are saved by a given reduction in PM concentrations: years of life that each individual was expected to live and that would have been lost had the reduction in PM concentrations not occurred. To provide a range of the possible cost of premature mortality, we estimate both measures of mortality in this analysis.

There are two types of exposure to elevated levels of air pollution that may result in premature mortality. Acute (short-term) exposure (e.g., exposure on a given day) to peak pollutant concentrations may result in excess mortality on the same day or within a few days of the elevated exposure. Chronic (long-term) exposure (e.g., exposure over a period of a year or more) to levels of pollution that are generally higher may result in mortality in excess of what it would be if pollution levels were generally lower. The excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels.

4.1.1 Short-Term Versus Long-Term Studies

There are two types of epidemiological studies that examine the relationship between mortality and exposure. Long-term studies (e.g., Pope et al., 1995) estimate the association between long-term (chronic) exposure to air pollution and the survival of members of a large study population over an extended period of time. Such studies examine the health endpoint of concern in relation to the general long-term level of the pollutant of concern, for example, relating annual mortality to some measure of annual pollutant level. Daily peak concentrations would impact the results only insofar as they affect the measure of long-term (e.g., annual) pollutant concentration. In contrast, short-term studies relate daily levels of the pollutant to daily mortality. By their basic design, daily studies can detect acute effects but cannot detect the effects of long-term exposures. A chronic exposure study design (a prospective cohort study, such as the Pope study) is best able to identify the long-term exposure effects, and may detect some of the short-term exposure effects as well. Because a long-term exposure study may detect some of the same short-term exposure effects detected by short-term studies, including both types of study in a benefit analysis would likely result in some degree of double counting of benefits. While the long-term study design is preferred, these types of studies are expensive to conduct and consequently there are relatively few well designed long-term studies.

4.1.2 Degree of Prematurity of Mortality

It is possible that the short-term studies are detecting an association between PM and mortality that is primarily occurring among terminally ill people. Critics of the use of short-term studies for policy analysis purposes correctly point out that an added risk factor that results in terminally ill people dying a few days or weeks earlier than they otherwise would have (referred to as "short-term harvesting") is potentially included in the measured PM mortality "signal" detected in such a study. While some of the detected excess deaths may have resulted in a substantial reduction in lifespan, others may have resulted in a relatively small decrease in lifespan. Studies by Spix et al (1993) and Pope et al. (1992) yield conflicting evidence, suggesting that harvesting may represent anywhere from zero to 50 percent of the deaths estimated in short-term studies. However, recent work by Zeger et al. (1999) and Schwartz (2000) that focused exclusively on this issue, reported that short-term harvesting does not play a major role in the PM-mortality relationship.[11]

It is not likely, however, that the excess mortality reported in a long-term prospective cohort study like Pope et al. (1995) contains any significant amount of this short-term harvesting. The Cox proportional hazard statistical model used in the Pope study examines the question of survivability throughout the study period (ten years). Deaths that are premature by only a few days or weeks within the ten-year study period (for example, the deaths of terminally ill patients, triggered by a short duration PM episode) are likely to have little impact on the calculation of the average probability of surviving the entire ten-year interval.

4.1.3 Estimating PM-Related Premature Mortality

The benefits analysis estimates $PM_{2.5}$ -related mortality using the C-R function estimated by Krewski et al. (2000). This study is a reanalysis of Pope et al. (1995), which estimated the association between long-term (chronic) exposure to $PM_{2.5}$ and the survival of members of a large study population. Our decision to use Pope et al. in previous benefits analyses reflected the EPA Science Advisory

[11]Zeger et al. (1999, p. 171) reported that: "The TSP-mortality association in Philadelphia is inconsistent with the harvesting-only hypothesis, and the harvesting-resistant estimates of the TSP relative risk are actually larger – not smaller – than the ordinary estimates."

Board's[12] explicit recommendation for modeling the mortality effects of PM in both the Clean Air Act §812 Retrospective Report to Congress and the Clean Air Act §812 Prospective study (U.S. EPA, 1999a, p. 12). An advantage of Krewski et al. over Pope et al. is that Krewski et al.'s reanalysis uses the annual mean $PM_{2.5}$ concentration rather than the annual median. Because the mean is more readily affected by high PM values than is the median, if high PM days are actually important in causing premature mortality, the annual mean may be a preferable measure of long-term exposure than the median.

The Krewski et al. (2000) long-term study is selected for use in the benefits analysis instead of short-term (daily pollution) studies for a number of reasons. It is used alone– rather than considering the total effect to be the sum of estimated short-term and long-term effects– because summing creates the possibility of double-counting a portion of PM-related mortality. The Krewski et al. study and the Pope study it reanalyzes are considered preferable to other available long-term studies because they use better statistical methods, have a much larger sample size, the longest exposure interval, and more locations (51 cities) in the United States, than other studies.

It is currently unknown whether there is a time lag (a delay between changes in PM exposures and changes in mortality rates) in the chronic PM/premature mortality relationship. The existence of such a lag is important for the valuation of premature mortality incidences because economic theory suggests that benefits occurring in the future should be discounted. Although there is no specific scientific evidence of the existence or structure of a PM effects lag, current scientific literature on adverse health effects, such as those associated with PM (e.g., smoking related disease) and the difference in the effect size between chronic exposure studies and daily mortality studies suggest that it is likely that not all incidences of premature mortality reduction associated with a given incremental change in PM exposure would occur in the same year as the exposure reduction. This same smoking-related literature implies that lags of up to a few years are plausible. Following explicit advice from the SAB, we assume a five-year lag structure, with 25 percent of premature deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years (U.S. EPA, 1999c, p. 9). It should be noted that the selection of a five-year lag structure is not directly supported by any PM-specific literature. Rather, it is intended to be a best guess at the appropriate time distribution of avoided incidences of PM-related mortality.

Alternative Calculations: PM-Related Premature Mortality

Although we use the Krewski, et al. (2000) mean-based ("PM2.5(DC), All Causes") model exclusively to derive our primary estimates of avoided premature mortality, we also examine the impacts of selecting alternative C-R functions for premature mortality. There are several candidates for alternative C-R functions, some from the Krewski, et al. study, and others from the original ACS study by Pope et al. (1995) or from the "Harvard Six-City Study" by Dockery et al. (1993), however, for this analysis, we are limited to examining those based on annual mean $PM_{2.5}$.

The Krewski et al. (2000) reanalysis provides results for several models that control for spatial correlations in the data. Krewski et al. pointed out that "if not identified and modeled correctly, spatial correlation could cause substantial errors in both the regression coefficients and their standard errors,"

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[12] "The EPA Science Advisory Board was established by Congress to provide independent scientific and engineering advice to the EPA Administrator on the technical basis for EPA regulations. Expressed in terms of the current parlance of the risk assessment/risk management paradigm of decision making (National Research Council, Managing Risk in the Federal Government. 1983), the SAB deals with risk assessment issues (hazard identification, dose-response assessment, exposure assessment and risk characterization) and only that portion of risk management that deals strictly with the technical issues associated with various control options." Source: http://www.epa.gov/sab/overview.htm, April 5, 2002.

(Krewski et al., 2000). These models are based on the original ACS air quality dataset, which contained only median $PM_{2.5}$ concentrations. Ideally, our primary C-R function for premature mortality would be both based on the mean and adjusted for regional variability. Unfortunately, Krewski et al. do not provide such an estimate. As such, we have chosen to use the mean-based relative risk in our primary analysis; because the SAMI analysis only generated annual mean $PM_{2.5}$, we are unable to use the median-based regionally adjusted relative risks to provide alternative estimates exploring the impact of adjustments for spatial correlations.

Though we are unable to estimate median-based mortality incidence based on relative risks adjusted for spatial correlation, we can compare the relative risks between our primary mortality model and the alternative Krewski model that is based on the annual median and adjusted for regional variability. The primary model we use has a reported relative risk of 1.12; the alternative model has a reported relative risk of 1.16. If the two models shared the same PM metric (average or median) and all else was equal, the model with the adjustment for spatial correlation would yield the larger estimate of avoided mortality incidence. However, the two models are not based on the same PM metric. As we explained above, a mean-based estimate is preferred to the median because changes in the mean more accurately reflect changes in peak values than do changes in the median. For emission control scenarios that affect peak PM days more than average PM days, larger changes in the mean will be observed compared to median changes. Because of the competing effects of the spatial correlation adjustment and the different PM metrics, it is difficult to say how the adjustment for spatial correlation impacts the overall estimate of avoided incidence in a given policy analysis. We can, however, compare the impact using each relative risk had on the estimate of mortality incidence in the Heavy Duty Diesel Analysis. The median-based, spatial correlation adjusted estimate of avoided mortality incidence was approximately 13% greater than the primary estimate of avoided mortality incidence (Abt Associates, 2000).

Krewski, et al. (2000) also reanalyzed the data from another prospective cohort study (the Harvard "Six Cities Study") authored by Dockery et al. (1993). The Dockery et al. study used a smaller sample of individuals from fewer cities than the study by Pope et al. (1995); however, it features improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. The SAB has noted that "the [Harvard Six Cities] study had better monitoring with less measurement error than did most other studies" (U.S. EPA, 1999d, p. 10).

The Dockery et al. (1993) study finds a larger effect of PM on premature mortality relative to the Pope et al. (1995) study. To provide a more complete picture of the range of possible premature mortality risks that may be associated with long-term exposures to fine particles, we also present alternative estimates based on the Krewski et al. (2000) reanalysis of the Dockery et al. data and the original study estimates. The Health Review Committee (2000, p. 270) commentary noted the "inherent limitations of using only six cities, understood by the original investigators, should be taken into account when interpreting the results of the Six Cities Study." We emphasize, that based on our understanding of the relative merits of the two datasets, the Krewski et al. ACS model based on mean $PM_{2.5}$ levels in 63 cities is the most appropriate model for analyzing the premature mortality impacts of the HD Engine/Diesel Fuel rule. It is thus used for our primary estimate of this important health effect.

Some of the functions are based on changes in mean $PM_{2.5}$ concentrations while others are based on median $PM_{2.5}$ concentrations. Given the available SAMI air quality data, we have only considered those based on annual mean changes. Estimated reductions in premature mortality will depend on both the size of the C-R coefficient and the change in the annual mean $PM_{2.5}$ metric. We also estimated alternative premature mortality incidence using both non-accidental and all-cause mortality rates. In previous benefit analyses conducted for the EPA, premature mortality was calculated using non-accidental mortality rates. For the sake of comparability to previous analyses, we included estimates of premature mortality based on both rates.

Sensitivity Calculation: Mortality Lag Structure

Just when PM-related mortality occurs in relation to exposure to PM is uncertain. We do not know what percentage of PM-related mortality occurs in the same year as exposure, in the following year, and so forth. To account for the uncertainty about possible lags in PM-related mortality, we examine the sensitivity of mortality-related benefits to alternative lag structures. Exhibit 4-3 presents the lags that are used in these sensitivity calculations. As stated earlier, the primary analysis uses a five-year lag structure in the valuation of mortality and chronic bronchitis, with incidence apportioned as follows: 25 percent in the first year, 25 percent in the second year, and 16.67 percent in each of the last three years.

To examine the effect alternate lag-structures have on the estimation of both mortality and chronic bronchitis valuation, the mortality benefits will be calculated using five alternative lag structures. Lag 1 will apportion the occurrence of all incidence to the first year. Valuation of these cases will not be discounted. In lag 2, based on the length of the study period for the Dockery et al. (1993) study, 100 percent of mortality incidence occurs in fifteen years from the modeled future-year. Lag 3, based on the length of the study period for the Pope et al. (1995) study, assigns 100 percent of the occurrence of mortality incidence to the eighth year out from the modeled future-year. Lag 4 front loads the occurrence of mortality incidence. Incidence is apportioned in decreasing amounts out to fifteen years. Lag 5 apportions incidence over fifteen years, assigning a lesser percentage of incidence in the beginning years, with the percentage of incidence increasing over time out to fifteen years. The latter two lag structures are intended to show how the distribution of incidences within a lag period affects benefit estimates.

Sensitivity Calculation: Threshold Analysis

To examine the effect an implied PM threshold has on the estimation of health effects in this analysis, we applied an increasingly stringent threshold to the Krewski et al. (2000) mortality function in one ug/m^3 increments. The results of this sensitivity analysis can be found in Appendix A.

Exhibit 4-3 Mortality Lag Structures Examined in Sensitivity Analyses

Year	Primary	Sensitivity 1	Sensitivity 2	Sensitivity 3	Sensitivity 4	Sensitivity 5
1	25	100	0	0	30	1
2	25	0	0	0	25	1
3	16.67	0	0	0	15	1
4	16.67	0	0	0	6	2
5	16.67	0	0	0	4	2
6	0	0	0	0	3	2
7	0	0	0	0	3	2
8	0	0	0	100	3	3
9	0	0	0	0	2	3
10	0	0	0	0	2	3
11	0	0	0	0	2	4
12	0	0	0	0	2	6
13	0	0	0	0	1	15
14	0	0	0	0	1	25
15	0	0	100	0	1	30

See Appendix A for results of the sensitivity analysis and its implications on the avoided mortality incidence benefits total.

4.1.4 Valuing Premature Mortality

Three methods for valuing avoided premature mortality are presented in this analysis. The first and primary one is the "statistical lives lost" approach, which derives the value of a "statistical life" lost from information about what people are willing to pay for mortal risk reduction. In contrast to the "statistical lives lost" approach, the second and third valuation approaches try to take into account that an individual's willingness to pay for mortal risk reduction may depend on his age. Using these approaches, the value of an avoided premature death depends on the age at which the individual dies. In all three methods, we assume for this analysis that PM-related premature mortality is distributed over the five years following exposure (the five-year mortality lag). To take this into account in the valuation of reductions in premature deaths, we apply an annual three percent discount rate to the value of avoided premature deaths occurring in future years.

Statistical Lives Lost

We estimate the monetary benefit of reducing premature mortality risk using the value of a "statistical life lost" approach, even though the actual valuation is of small changes in mortality risk experienced by a large number of people. The estimated value of a "statistical life lost" is an intermediate value from a variety of estimates in the economics literature, and is a value that EPA has frequently used in RIAs for other rules. This estimate is the mean of a distribution fitted to the estimates from 26 value-of-life studies identified in the §812 study as "applicable to policy analysis." The approach and set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria used by Viscusi in his review of value-of-life studies. The estimate is consistent with Viscusi's conclusion (updated to 2000 $) that "most of the reasonable estimates of the value of life are clustered in the $4 to $9.2 million range." Uncertainty associated with the valuation of premature mortality avoided is expressed through a Weibull distribution (see Exhibit 4-3) (IEc 1992, p. 2).

Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. The 26 studies are listed in Exhibit 4-4. The references for all but Gegax et al. (1985) and V.K. Smith (1983) may be found in Viscusi (1992). Although each of the studies estimated the mean WTP ($\bar{M}$WTP) for a given reduction in mortality risk, the amounts of reduction in risk being valued were not necessarily the same across studies, nor were they necessarily the same as the amounts of reduction in mortality risk that would actually be conferred by a given reduction in ambient pollutant concentrations.

The transferability of estimates of the value of a statistical life from the 26 studies to this analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction, or equivalently, that the marginal willingness to pay curve is horizontal within a reasonable range. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is $30. Suppose, however, that the actual mortality risk reduction resulting from a given air quality improvement is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $30 for a reduction of 1/100,000 implies a WTP of $300 for a risk reduction of 1/10,000 (which is ten times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the value of a statistical life does not depend on the particular amount of risk reduction being valued.

Exhibit 4-4 Summary of Mortality Valuation Estimates

Study	Type of Estimate	Valuation (millions 2000 $)
Kneisner and Leeth (1991) (US)	Labor Market	0.8
Smith and Gilbert (1984)	Labor Market	0.9
Dillingham (1985)	Labor Market	1.2
Butler (1983)	Labor Market	1.5
Miller and Guria (1991)	Contingent Valuation	1.6
Moore and Viscusi (1988)	Labor Market	3.3
Viscusi et al. (1991)	Contingent Valuation	3.6
Gegax et al. (1985; 1991)	Contingent Valuation	4.3
Marin and Psacharopoulos (1982)	Labor Market	3.7
Kneisner and Leeth (1991) (Australia)	Labor Market	4.3
Gerking et al. (1988)	Contingent Valuation	4.5
Cousineau et al. (1988; 1992)	Labor Market	4.7
Jones-Lee (1989)	Contingent Valuation	5.0
Dillingham (1985)	Labor Market	5.1
Viscusi (1978; 1979)	Labor Market	5.4
R.S. Smith (1976)	Labor Market	6.1
V.K. Smith (1983)	Labor Market	6.2
Olson (1981)	Labor Market	6.9
Viscusi (1981)	Labor Market	8.6
R.S. Smith (1974)	Labor Market	9.5
Moore and Viscusi (1988)	Labor Market	9.6
Kneisner and Leeth (1991) (Japan)	Labor Market	10.0
Herzog and Schlottman (1987; 1990)	Labor Market	12.0
Leigh and Folson (1984)	Labor Market	12.8
Leigh (1987)	Labor Market	13.7
Garen (1988)	Labor Market	17.8

Source: Viscusi (1992, Table 4.1).

Although the particular amount of mortality risk reduction being valued in a study may not affect the transferability of the WTP estimate from the study to this analysis, the characteristics of the study subjects and the nature of the mortality risk being valued in the study could be important. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the MWTP to reduce the risk. The appropriateness of the MWTP estimates from the 26 studies for valuing the mortality-related benefits of reductions in ambient air concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on (1) the extent to which the subjects in the studies are similar to the population affected by changes in ambient air concentrations and (2) the extent to which the risks being valued are similar.

Focusing on the wage-risk studies, which make up the substantial majority of the 26 studies relied upon, the likely differences between (1) the subjects in these studies and the population affected by changes in air concentrations and (2) the nature of the mortality risks being valued in these studies and the

nature of air pollution-related mortality risk are considered. The direction of bias in which each difference is likely to result is also considered.

Compared with the subjects in wage-risk studies, the population believed to be most affected by air pollution (i.e., the population that would receive the greatest mortality risk reduction associated with a given reduction in air concentrations) is, on average, older and probably more risk averse. For example, citing Schwartz and Dockery (1992) and Ostro et al. (1996), Chestnut (1995) estimated that approximately 85 percent of those who die prematurely from ambient air pollution-related causes are over 65. The average age of subjects in wage-risk studies, in contrast, is well under 65.

There is also reason to believe that those over 65 are, in general, more risk averse than the general population while workers in wage-risk studies are likely to be less risk averse than the general population. Although Viscusi's (1992) list of recommended studies excludes studies that consider only much-higher-than-average occupational risks, there is nevertheless likely to be some selection bias in the remaining studies -- that is, these studies are likely to be based on samples of workers who are, on average, more risk-loving than the general population. In contrast, older people as a group exhibit more risk averse behavior.

In addition, it might be argued that because the elderly have greater average wealth than those younger, the affected population is also wealthier, on average, than wage-risk study subjects, who tend to be blue collar workers. It is possible, however, that among the elderly it is largely the poor elderly who are most vulnerable to air pollution-related mortality risk (e.g., because of generally poorer health care). If this is the case, the average wealth of those affected by a reduction in air concentrations relative to that of subjects in wage-risk studies is uncertain.

The direction of bias resulting from the age difference is unclear, particularly because age is confounded by risk aversion (relative to the general population). It could be argued that, because an older person has fewer expected years left to lose, his WTP to reduce mortality risk would be less than that of a younger person. This hypothesis is supported by one empirical study, Jones-Lee et al.(1985), that found the value of a statistical life at age 65 to be about 90 percent of what it is at age 40. Citing the evidence provided by Jones-Lee et al., Chestnut (1995) assumed that the value of a statistical life for those 65 and over is 75 percent of what it is for those under 65.

The greater risk aversion of older people, however, implies just the opposite. Citing Ehrlich and Chuma (1990), Industrial Economics Inc. (1992) noted that "older persons, who as a group tend to avoid health risks associated with drinking, smoking, and reckless driving, reveal a greater demand for reducing mortality risks and hence have a greater implicit value of a life year." That is, the more risk averse behavior of older individuals suggests a greater WTP to reduce mortality risk.

There is substantial evidence that the income elasticity of WTP for health risk reductions is positive (Loehman and De, 1982; Jones-Lee et al., 1985; Mitchell and Carson, 1986; Gerking et al., 1988; Alberini et al., 1997). However, there is uncertainty about the exact value of this elasticity. Individuals with higher incomes (or greater wealth) should, then, be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. Whether the average income or level of wealth of the population affected by ambient air pollution reductions is likely to be significantly different from that of subjects in wage-risk studies, however, is unclear.

Finally, although there may be several ways in which job-related mortality risks differ from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily whereas air pollution-related risks are incurred involuntarily.

There is some evidence that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily (e.g., Violette and Chestnut, 1983). Job-related risks are incurred voluntarily whereas air pollution-related risks are incurred involuntarily. If this is the case, WTP estimates based on wage-risk studies may be downward biased estimates of WTP to reduce involuntarily incurred ambient air pollution-related mortality risks.

The potential sources of bias in an estimate of MWTP to reduce the risk of air pollution related mortality based on wage-risk studies are summarized in Exhibit 4-5. Although most of the individual factors tend to have a downward bias (i.e. the given WTP estimate is understated), the overall effect of these biases is unclear.

Exhibit 4-5 Potential Sources of Bias in Estimates of Mean WTP to Reduce the Risk of PM Related Mortality Based on Wage-Risk Studies

Factor	Likely Direction of Bias in Mean WTP Estimate
Age	Uncertain
Degree of Risk Aversion	Downward
Income	Downward, if the elderly affected are a random sample of the elderly. It is unclear, if the elderly affected are the poor elderly.
Risk Perception: Voluntary vs. Involuntary risk	Downward

Alternative Calculation: Statistical Life-Years Lost

In an alternative calculation, we value statistical life-years, rather than valuing statistical lives. Moore and Viscusi (1988) value a statistical life-year lost, by assuming that the WTP to save a statistical life is the value of a single year of life times the expected number of years of life remaining for an individual. They suggest that a typical respondent in a mortal risk study has a life expectancy of an additional 35 years. Using a mean estimate of $4.8 million (1990 $) to save a statistical life, their approach yields an estimate of $137,000 per life-year lost or saved, assuming no discounting. If an individual discounts future additional years using a standard discounting procedure, the value of each life-year lost must be greater than the value assuming no discounting. Using a 35 year life expectancy, a $6.324 million value of a statistical life, and a three percent discount rate, the implied value of each life-year lost is $293,807 in 2000 dollars.

In addition, the "statistical life-years lost" analysis must accommodate the five-year lag structure. For each person dying at a given age, using the expected number of years remaining for that age, based on 1997 life expectancy tables (National Center for Health Statistics, 1999, Table 5), and a VSLY of $293,807, we calculate the present discounted value (discounted back to the beginning of the year of death) for that person. All values are then discounted back to the beginning of the scenario year (2010 or 2040), whether the individual dies in that year or in a subsequent year. The present discounted value (discounted back to the beginning of the scenario year) of an avoided premature mortality will vary from one individual to another, depending on the age of the individual at death and on the extent of lag between exposure and death. The age at death determines the expected number of life-years lost, while the extent of lag between exposure and death determines the amount of discounting needed.

Alternative Calculation: Age-Based Adjustments of the Value of a Statistical Life Lost

There are drawbacks to the "statistical life-years lost" approach, however. In a recent report, the Scientific Advisory Board (SAB) notes that "inferring the value of a statistical life year ... requires assumptions about the discount rate and about the time path of expected utility of consumption" (U.S. EPA, 2000a, p. 8). In considering the merits of age-based adjustments, the SAB also notes that "the theoretically appropriate method is to calculate WTP for individuals whose ages correspond to those of the affected population, and that it is preferable to base these calculations on empirical estimates of WTP by age." Several studies conducted by Jones-Lee, et al. (1985; 1989; 1993) found a significant effect of age on the value of mortality risk reductions expressed by citizens in the United Kingdom. The Jones-Lee-based analysis suggests a U-shaped relationship between age and VSL, peaking around age 40, and declining to between 60 and 90 percent of the mean VSL value for individuals over the age of 70, and declining further as individuals age. This finding has been supported by two recent analyses conducted by Krupnick, et al. (2000; 2000), which asked samples of Canadian and U.S. residents their values for reductions in mortality risk.

The results of the Jones-Lee et al. analysis were used to calculate age-based adjustment factors to adjust the value of a statistical life lost by an individual of about age 40 (the average age in the wage-risk studies on which the value of a statistical life is based in the primary method), to age-specific values. For example, Jones-Lee et al. (1989) found that people ages 30-39 were willing to pay 89 percent as much as people ages 40-59 for the same mortality risk reduction. If the value of a statistical life saved of someone 40 years old is $6.12 million, then the value of a statistical life saved of someone age 30-39 would be 89 percent of that, or $5.45 million. Numbers of lives saved in each of the age groups used in the statistical life-years-lost alternative calculation were apportioned to the age groups used by Jones-Lee et al. (1989; 1993). The number of lives saved in an age group was then multiplied by the age-adjusted value of a statistical life saved for that age group. The value of a statistical life saved in an age group was calculated as $6.12 million times the ratio of the WTP for mortality risk reduction in that age group to the WTP for mortality risk reduction in the age 40-59 group, as reported by Jones-Lee et al. (1989; 1993). The five-year lag structure used in the primary method was applied under two alternative discount rate assumptions of three percent and seven percent. Because the two Jones-Lee studies reported different ratios, this alternative calculation was carried out separately using each of the two Jones-Lee studies.

4.2 Chronic Illness

The only benefit category evaluated in this analysis associated with chronic (long-term) illness is bronchitis. Onset of bronchitis has been associated with exposure to air pollutants. Three studies have, in fact, linked the onset of chronic bronchitis in adults to particulate matter. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998). For more information, see the Heavy Duty Diesel Technical Support Document (Abt Associates, 2000).

4.2.1 Chronic Bronchitis

In past analyses, we have estimated the changes in the number of new cases of PM-related chronic bronchitis using studies by Schwartz (1993) and Abbey et al. (1995b). The Schwartz study examined the relationship between exposure to PM_{10} and prevalence of chronic bronchitis. The Abbey et al. study examined the relationship between $PM_{2.5}$ and new incidences of chronic bronchitis. Both studies have strengths and weaknesses which suggest that, if both measures of PM were available, pooling the effect estimates from each study may provide a better estimate of the expected change in incidences of chronic bronchitis than using either study alone. However, since the SAMI analysis is based solely upon

changes in annual mean PM2.5, we use the Abbey et al. study to predict changes in chronic bronchitis incidence.

It should be noted, however, that reliance on the Abbey et al. (1995b) estimate will result in an underestimate of the change in chronic bronchitis incidences if both the fine and coarse fractions of PM_{10} are associated with chronic bronchitis. The SAMI control scenarios would likely result in reductions in both the fine and coarse fractions of PM_{10}.

Alternative Calculation: Chronic Bronchitis Reversals

In developing the C-R functions for chronic bronchitis, it is necessary to estimate its annual incidence rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate.[13] Reversals refer to those cases of chronic bronchitis that were reported at the start of the Abbey et al. survey, but were subsequently not reported at the end of the survey. Since we assume that chronic bronchitis is a permanent condition, we subtract these reversals. Nevertheless, reversals may likely represent a real effect that should be included in the analysis. To allow for this possibility, we present an estimate of reversals in an alternative calculation in which reversals are considered to be chronic bronchitis cases of the lowest severity level, as described below.

Valuing Chronic Bronchitis

PM-related chronic bronchitis is expected to last from the initial onset of the illness throughout the rest of the individual's life. WTP to avoid chronic bronchitis would therefore be expected to incorporate the present discounted value of a potentially long stream of costs (e.g., medical expenditures and lost earnings) and pain and suffering associated with the illness. Two studies, Viscusi et al. (1991) and Krupnick and Cropper (1992), provide estimates of WTP to avoid a case of chronic bronchitis.

The Viscusi et al. (1991) and the Krupnick and Cropper (1992) studies were experimental studies intended to examine new methodologies for eliciting values for morbidity endpoints. Although these studies were not specifically designed for policy analysis, we believe the studies provide reasonable estimates of the WTP for chronic bronchitis. As with other contingent valuation studies, the reliability of the WTP estimates depends on the methods used to obtain the WTP values. The Viscusi et al. and the Krupnick and Cropper studies are broadly consistent with current contingent valuation practices, although specific attributes of the studies may not be.

The study by Viscusi et al. (1991) uses a sample that is larger and more representative of the general population than the study by Krupnick and Cropper (1992), which selects people who have a relative with the disease. Thus, the valuation for the high-end estimate is based on the distribution of WTP responses from Viscusi et al. The WTP to avoid a case of pollution-related chronic bronchitis (CB) is derived by starting with the WTP to avoid a severe case of chronic bronchitis, as described by Viscusi et al. (1991), and adjusting it downward to reflect (1) the decrease in severity of a case of pollution-related CB relative to the severe case described in the Viscusi et al. study, and (2) the elasticity of WTP with respect to severity reported in the Krupnick and Cropper study. Because elasticity is a marginal concept and because it is a function of severity (as estimated from Krupnick and Cropper), WTP adjustments were made incrementally, in one percent steps. A severe case of CB was assigned a severity

[13]The percentage of reversals is estimated to be 46.6% based on Abbey et al. (1995a, Table 1).

level of 13 (following Krupnick and Cropper). The WTP for a one percent decrease in severity is given by:

$$WTP_{0.99sev} = WTP_{sev} \cdot (1 - 0.01 \cdot e) \ ,$$

where sev is the original severity level (which, at the start, is 13) and e is the elasticity of WTP with respect to severity. Based on the regression in Krupnick and Cropper (1992) (see below), the estimate of e is 0.18*sev. At the mean value of sev (6.47), e = 1.16. As severity decreases, however, the elasticity decreases. Using the regression coefficient of 0.18, the above equation can be rewritten as:

$$WTP_{0.99sev} = WTP_{sev} \cdot (1 - 0.01 \cdot 0.18 sev)$$

For a given WTP_{sev} and a given coefficient of sev (0.18), the WTP for a 50 percent reduction in severity can be obtained iteratively, starting with sev =13, as follows:

$$WTP_{12.87} = WTP_{0.99\cdot 13} = WTP_{13} \cdot (1 - 0.01 \cdot 0.18 \cdot 13)$$

$$WTP_{12.74} = WTP_{0.99\cdot 12.87} = WTP_{12.87} \cdot (1 - 0.01 \cdot 0.18 \cdot 12.87)$$

$$WTP_{12.61} = WTP_{0.99\cdot 12.74} = WTP_{12.74} \cdot (1 - 0.01 \cdot 0.18 \cdot 12.74)$$

and so forth. This iterative procedure eventually yields $WTP_{6.5}$, or WTP to avoid a case of chronic bronchitis that is of "average" severity.

The derivation of the WTP to avoid a case of pollution-related chronic bronchitis is based on three components, each of which is uncertain: (1) the WTP to avoid a case of severe CB, as described in the Viscusi et al. (1991) study, (2) the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi et al.), and (3) the elasticity of WTP with respect to severity of the illness. Because of these three sources of uncertainty, the WTP is uncertain. Based on assumptions about the distributions of each of the three uncertain components, a distribution of WTP to avoid a pollution-related case of CB was derived by Monte Carlo methods. The mean of this distribution is taken as the central tendency estimate of WTP to avoid a pollution-related case of CB. Each of the three underlying distributions is described briefly below.

1. The distribution of WTP to avoid a severe case of CB was based on the distribution of WTP responses in the Viscusi et al. (1991) study. Viscusi et al. derived respondents' implicit WTP to avoid a statistical case of chronic bronchitis from their WTP for a specified reduction in risk. The mean response implied a WTP of about $1,318,000 (2000 $)[14]; the median response implied a WTP of about $700,000 (2000 $). However, the extreme tails of distributions of WTP responses are usually considered

[14]There is an indication in the Viscusi et al. (1991) paper that the dollar values in the paper are in 1987 dollars. Under this assumption, the dollar values were converted to 2000 dollars.

unreliable. Because the mean is much more sensitive to extreme values, the median of WTP responses is often used rather than the mean. Viscusi et al. report not only the mean and median of their distribution of WTP responses, however, but the decile points as well. The distribution of reliable WTP responses from the Viscusi et al. study could therefore be approximated by a discrete uniform distribution giving a probability of 1/9 to each of the first nine decile points. This omits the first five and the last five percent of the responses (the extreme tails, considered unreliable). This trimmed distribution of WTP responses from the Viscusi et al. study was assumed to be the distribution of WTPs to avoid a severe case of CB.

2. The distribution of the severity level of an average case of pollution-related CB was modeled as a triangular distribution centered at 6.5, with endpoints at 1.0 and 12.0. These severity levels are based on the severity levels used in Krupnick and Cropper (1992), which estimated the relationship between ln(WTP) and severity level, from which the elasticity is derived. The most severe case of CB in that study is assigned a severity level of 13. The mean of the triangular distribution is 6.5. This represents a 50 percent reduction in severity from a severe case.

3. The elasticity of WTP to avoid a case of CB with respect to the severity of that case of CB is a constant times the severity level. This constant was estimated by Krupnick and Cropper (1992) in the regression of ln(WTP) on severity, discussed above. This estimated constant (regression coefficient) is normally distributed with mean = 0.18 and standard deviation = 0.0669 (obtained from Krupnick and Cropper).

The distribution of WTP to avoid a case of pollution-related CB was generated by Monte Carlo methods, drawing from the three distributions described above. On each of 16,000 iterations (1) a value was selected from each distribution, and (2) a value for WTP was generated by the iterative procedure described above, in which the severity level was decreased by one percent on each iteration, and the corresponding WTP was derived. The mean of the resulting distribution of WTP to avoid a case of pollution-related CB was $340,568.

This WTP estimate is reasonably consistent with full COI estimates derived for chronic bronchitis, using average annual lost earnings and average annual medical expenditures reported by Cropper and Krupnick (1990) Using a 5 percent discount rate and assuming that (1) lost earnings continue until age 65, (2) medical expenditures are incurred until death, and (3) life expectancy is unchanged by chronic bronchitis, the present discounted value of the stream of medical expenditures and lost earnings associated with an average case of chronic bronchitis is estimated to be about $117,000 for a 30 year old, about $113,000 for a 40 year old, about $103,000 for a 50 year old, and about $59,000 for a 60 year old. A WTP estimate would be expected to be greater than a full COI estimate, reflecting the willingness to pay to avoid the pain and suffering associated with the illness. The WTP estimate of $340,568 is from 2.9 times the full COI estimate (for 30 year olds) to 5.8 times the full COI estimate (for 60 year olds).

Alternative Calculation: Valuing Chronic Bronchitis Reversals

In an alternative calculation, we estimate chronic bronchitis reversals and value them using the same method used to value cases of chronic bronchitis. However, instead of allowing the severity level to range from one to 13, we value all reversals at a severity level of one.

4.3 Acute Illnesses and Symptoms Not Requiring Hospitalization

We consider in this section the only acute symptom based upon a measure of annual mean PM2.5, acute bronchitis. Note that there are several additional types of acute illnesses and symptoms

associated with daily average PM2.5 and PM10, as well. These have been considered in a number of previous analyses, such as the Heavy Duty Diesel analysis (see Abt Associates, 2000). The extent of the acute illness endpoints that are not evaluated in this analysis is discussed in Section 6.

4.3.1 Acute Bronchitis

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in the U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.5}$ and PM_{10} were marginally significantly related to bronchitis.

Valuing Acute Bronchitis

Estimating WTP to avoid a case of acute bronchitis is difficult for several reasons. First, WTP to avoid acute bronchitis itself has not been estimated. Estimation of WTP to avoid this health endpoint therefore must be based on estimates of WTP to avoid symptoms that occur with this illness. Second, a case of acute bronchitis may last more than one day, whereas it is a day of avoided symptoms that is typically valued. Finally, the C-R function used in the benefit analysis for acute bronchitis was estimated for children, whereas WTP estimates for those symptoms associated with acute bronchitis were obtained from adults.

With these caveats in mind, the values used for acute bronchitis in this analysis were obtained by adjusting the values used in the CAA §812 Prospective analysis from 1990 dollars to 2000 dollars by multiplying by 1.318. WTP to avoid a case of acute bronchitis was estimated as the midpoint between a low estimate and a high estimate. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day. The unit value is the midpoint between the low and high estimates. The low, high, and midpoint estimates used in the §812 Prospective analysis were $13, $77, and $45, respectively (1990$). The corresponding values in 2000 dollars are $17.13, $101.45, and $59.29, respectively.

5. Results

This Section provides estimates of the magnitude and value of changes in selected health endpoints associated with each of the SAMI emission control scenario-related changes in annual average $PM_{2.5}$ concentrations. The total dollar benefits associated with a given endpoint depend on how much the endpoint will change (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth).

Before presenting the benefits associated with each of the SAMI emission control scenarios, however, we provide a list of benefit categories that were not quantified or monetized in Exhibit 5-1. Each of these unquantified endpoints are likely to be associated with the ambient pollution reductions achieved under the emission control assumptions each SAMI emission control scenario makes. Many of these endpoints have been quantified in previous analyses, where modeled air pollutant data was available. This table is meant to illustrate the extent of health and welfare effects that are associated with pollution reductions beyond those considered in this analysis.

We also note that annual average PM2.5-related health effects are not the only benefits that have been estimated in association with the SAMI control scenarios. The SAMI integrated assessment also estimated benefits associated with improved recreational visibility and improved recreational fishing opportunities due to improvements in SAMI-region visual air quality and water quality. We have added these benefits, as well as a symbolic representation of all other unmonetized benefits, "B", to the benefits displayed in the subsequent primary analysis tables to capture the magnitude of total benefits associated with the SAMI emission control strategies.

To place SAMI estimated incidence changes into context with predicted baseline incidence throughout the eight state SAMI region, Exhibit 5-2 displays the baseline incidence figures for $PM_{2.5}$ endpoints. Both the mean estimated incidence change and corresponding percent change between post-control incidence reductions and the predicted incidence baseline is presented. Note that these baseline incidences include all incidences, not just those associated with air pollution.

Exhibits 5-3 through 5-6 present results of our primary analysis; incidence and benefit estimates associated with each of the scenarios in each future year. Note that Scenario A2 is considered the baseline – the expected state of the world in 2010 and 2040 before SAMI controls are applied. These tables present results for Scenario B1 and Scenario B3 in 2010 and 2040, respectively. A 5^{th} percentile, mean, and 95^{th} percentile estimate for both incidence and benefits is presented for each endpoint, as well as the simple mean benefit (calculated by multiplying the mean estimate of incidence by the corresponding mean valuation). Total benefits are also displayed, calculated by summing the mean of each endpoint and adding to it the point estimates of recreational visibility and recreational fishing benefits.

The benefits associated with the SAMI emission control scenarios are substantial. Under scenario B1, total benefits are approximately \$12 billion + B in 2010 and grow to over \$36 billion + B by 2040. These benefits are reflected by the number of incidences avoided by each scenario; by 2040, premature mortality is reduced by 4,200 cases, there are 3,300 fewer cases of chronic bronchitis, and cases of acute bronchitis are reduced by 9,000. Under scenario B3, total benefits are approximately \$45 billion + B in 2010 and grow to approximately \$68 billion + B by 2040. By 2040, premature mortality is reduced by 8,000 cases, there are 6,000 fewer cases of chronic bronchitis, and cases of acute bronchitis are reduced by 16,000.

We present total health-related benefits by SAMI state in Exhibit 5-7 for the 2040 B1 and B3 scenarios. State-level benefits only include benefits associated with annual average PM2.5-related

endpoints; other benefits, such as recreational visibility and recreational fishing are not included. Of the states included in the 8-state SAMI region, North Carolina receives the largest percentage share of total health-related benefits. In 2040, North Carolina received 24% and 23% of total benefits for scenarios B1 and B3. Georgia receives the next largest percentage share, equal to 21% of total benefits for scenarios B1 B1 and B3 in 2040. The state receiving the smallest share is West Virginia with 2% and 3% of total benefits for scenarios B1 and B3 in 2040.

Exhibits 5-8 through 5-11 display the alternative incidence and benefit calculations to those included in the primary analysis. Where possible, a 5th percentile, mean, and 95th percentile estimate for incidence and/or benefits is presented for each alternative endpoint. Exhibits 5-12 through 5-15 present the alternative mortality functions. A 5th percentile, mean, and 95th percentile estimate for incidence and benefits is presented for each alternative mortality function. Note that the valuation of alternative mortality functions uses a 3% discount rate in the application of a mortality lag adjustment factor.

We provide quantified estimates of the 90 percent confidence intervals around these estimates based solely on the standard errors of the C-R coefficients. These intervals do not account for any uncertainties in the change in air quality, population projections, baseline incidence rates, or model uncertainties. We also provide estimates of the 90 percent confidence intervals around the dollar value estimates for each endpoint, using Monte Carlo techniques to combine the distributions of the health effect estimates and the valuation estimates. Again, these intervals do not account for any uncertainties in other factors. We do not calculate a confidence interval for the total economic value of all health outcomes, as this would imply a precision which is not warranted based on the gaps in information about the impact of unquantified sources of uncertainty.

Exhibit 5-1 Unquantified Endpoints Associated with Pollution Reductions Associated with the SAMI Emission Control Scenarios

Unquantified Endpoints	Pollutant
PM-Related Endpoints - Health Effects [a]	
Hospital Admissions - Pneumonia (adults, over 64)	Daily Average PM10
Hospital Admissions - COPD (adults, 64 and over)	Daily Average PM10
Hospital Admissions - Asthma (65 and younger)	Daily Average PM2.5
Hospital Admissions - Cardiovascular (adults, over 64)	Daily Average PM10
Emergency Room Visits for Asthma (65 and younger)	Daily Average PM10
Asthma Attacks (asthmatics, all ages)	Daily Average PM10
Lower Respiratory Symptoms (children, 7-14)	Daily Average PM2.5
Upper Respiratory Symptoms (asthmatic children, 9-11)	Daily Average PM10
Work Loss Days (adults, 18-65)	Daily Average PM2.5
Minor Restricted Activity Days (adults, age 18-65)	Daily Average PM2.5
Other PM-Related Health Effects	Varies
PM-Related Endpoints - Welfare Effects [a]	
Residential Visibility	Annual Average Extinction
Household Soiling	Annual Average PM10
Materials Damage	Varies
Estuary Eutrophication	Varies
Other PM-related Welfare Effects	Varies
Ozone-Related Endpoints - Health Effects [a]	
Hospital Admissions - Respiratory Causes (all ages)	Daily 12-Hour Average Ozone
Hospital Admissions - Cardiac Dysrhythmias (all ages)	Daily Average Ozone
Emergency Room Visits for Asthma (all ages)	Varies
Asthma Attacks (asthmatics, all ages)	Daily One-Hour Max Ozone
Minor Restricted Activity Days (adults, age 18-65)	Daily One-Hour Max Ozone
Decreased Worker Productivity (adult working population)	Daily Average Ozone
Other Ozone-Related Health Effects	Varies
Ozone-Related Endpoints - Welfare Effects [a]	
Commercial Agricultural Benefits (6 major crops)	Sum 06
Commercial Forestry Benefits	Varies
Other Ozone-Related Welfare Effects	Varies
CO-Related Health and Welfare Effects	Varies
SO_x-Related Health and Welfare Effects	Varies
NOx-Related Health and Welfare Effects	Varies
Hazardous Air Pollutant-Related Health and Welfare Effects	Varies
Total Unmonetized Health- and Welfare-Related Benefits	**B** [b]

[a] For a complete discussion of the nature, estimation, and valuation of PM- and Ozone-related endpoints see the Heavy Duty Diesel Regulatory Impact Analysis (U.S. EPA, 2000b) and the associated Technical Support Document (Abt Associates, 2000)

[b] "B" represents the sum of all unmonetized benefit categories.

Exhibit 5-2 Baseline Percentages

Endpoint	Reference	Baseline Scenarios		2010 B1 Scenario		2040 B1 Scenario		2010 B3 Scenario		2040 B3 Scenario	
		2010	2040	Mean	% of Baseline	Mean	% of Baseline	Mean	% of Baseline	Mean	% of Baseline
Ages 30+	Krewski et al. (2000)	325,741	399,728	1,662	0.51%	4,273	1.07%	6,155	1.89%	8,007	2.00%
Chronic Bronchitis	Abbey et al. (1995b)	76,832	95,580	1,258	1.64%	3,303	3.46%	4,531	5.90%	6,051	6.33%
Acute Bronchitis	Dockery et al. (1996)	120,528	149,809	3,464	2.87%	8,952	5.98%	12,192	10.12%	16,177	10.80%

Exhibit 5-3 Total Quantified Benefits of SAMI: B1 Scenario in 2010

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Mortality							
Ages 30+, 3% Discount Rate	Krewski et al. (2000), Table 31	931	1,662	2,355	$1,568	$11,114	$26,902
Ages 30+, 7% Discount Rate	Krewski et al. (2000)	931	1,662	2,355	$1,473	$10,439	$25,269
Chronic Illness							
Chronic Bronchitis	Abbey et al. (1995b)	222	1,258	2,269	$25	$483	$1,569
Minor Illness							
Acute Bronchitis	Dockery et al. (1996)	0	3,464	6,805	$0.0	$0.2	$0.5
Welfare Effects							
Recreational Visibility Benefits	Abt Associates Inc. (2002a)	Direct Economic Valuation			--	$796	--
Recreational Fishing Benefits	Abt Associates Inc. (2002b)	Direct Economic Valuation			--	$0.6	--
Total Primary Benefits (3% discount rate)		--	--	--	--	**$12,394 + B**	--
Total Primary Benefits (7% discount rate)		--	--	--	--	**$11,719 + B**	--

Exhibit 5-4 Total Quantified Benefits of SAMI: B1 Scenario in 2040

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Mortality							
Ages 30+, 3% Discount Rate	Krewski et al. (2000), Table 31	2,399	4,273	6,049	$4,710	$33,332	$80,666
Ages 30+, 7% Discount Rate	Krewski et al. (2000)	2,399	4,273	6,049	$4,424	$31,308	$75,767
Chronic Illness							
Chronic Bronchitis	Abbey et al. (1995b)	590	3,303	5,924	$94	$1,508	$5,456
Minor Illness							
Acute Bronchitis	Dockery et al. (1996)	0	8,952	17,362	$0.0	$0.6	$1.4
Welfare Effects							
Recreational Visibility Benefits	Abt Associates Inc. (2002a)	Direct Economic Valuation			--	$1,474	--
Recreational Fishing Benefits	Abt Associates Inc. (2002b)	Direct Economic Valuation			--	$1.4	--
Total Primary Benefits (3% discount rate)		--	--	--	--	**$36,316 + B**	--
Total Primary Benefits (7% discount rate)		--	--	--	--	**$34,292 + B**	--

Exhibit 5-5 Total Quantified Benefits of SAMI: B3 Scenario in 2010

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Mortality							
Ages 30+, 3% Discount Rate	Krewski et al. (2000), Table 31	3,463	6,155	8,700	$5,828	$41,163	$99,672
Ages 30+, 7% Discount Rate	Krewski et al. (2000)	3,463	6,155	8,700	$5,474	$38,664	$93,619
Chronic Illness							
Chronic Bronchitis	Abbey et al. (1995b)	820	4,531	8,072	$92	$1,740	$5,630
Minor Illness							
Acute Bronchitis	Dockery et al. (1996)	0	12,192	23,340	$0.0	$0.8	$1.8
Welfare Effects							
Recreational Visibility Benefits	Abt Associates Inc. (2002a)	Direct Economic Valuation			--	$2,502	--
Recreational Fishing Benefits	Abt Associates Inc. (2002b)	Direct Economic Valuation			--	$1.4	--
Total Primary Benefits (3% discount rate)		--	--	--	--	**$45,407 + B**	--
Total Primary Benefits (7% discount rate)		--	--	--	--	**$42,907 + B**	--

Exhibit 5-6 Total Quantified Benefits of SAMI: B3 Scenario in 2040

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Mortality							
Ages 30+, 3% Discount Rate	Krewski et al. (2000), Table 31	4,507	8,007	11,314	$8,845	$62,457	$151,219
Ages 30+, 7% Discount Rate	Krewski et al. (2000)	4,507	8,007	11,314	$8,307	$58,665	$142,037
Chronic Illness							
Chronic Bronchitis	Abbey et al. (1995b)	1,099	6,051	10,759	$174	$2,763	$10,019
***Minor* Illness**							
Acute Bronchitis	Dockery et al. (1996)	0	16,177	30,844	$0.0	$1.1	$2.5
Welfare Effects							
Recreational Visibility Benefits	Abt Associates Inc. (2002a)	Direct Economic Valuation			--	$2,705	--
Recreational Fishing Benefits	Abt Associates Inc. (2002b)	Direct Economic Valuation			--	$4.9	--
Total Primary Benefits (3% discount rate)		--	--	--	--	**$67,931 + B**	--
Total Primary Benefits (7% discount rate)		--	--	--	--	**$64,138 + B**	--

Exhibit 5-7 Total Health Related Benefits in 2040 by State

State	2040 B1		2040 B3	
	Monetary Benefits (millions 2000$)	Percent of Total	Monetary Benefits (millions 2000$)	Percent of Total
SAMI-Region Total	$34,841	100%	$65,221	100%
Alabama	$4,149	12%	$7,209	11%
Georgia	$7,285	21%	$13,948	21%
Kentucky	$1,196	3%	$3,555	5%
North Carolina	$8,404	24%	$14,992	23%
South Carolina	$2,827	8%	$5,986	9%
Tennessee	$5,772	17%	$10,251	16%
Virginia	$4,345	12%	$7,469	11%
West Virginia	$863	2%	$1,811	3%

Exhibit 5-8 Alternative Benefit Calculations for the 2010 SAMI "B1" Scenario

Endpoint	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
PM-related Alternative Calculations							
Life Years Lost, by age:	Krewski et al. (2000), Table 31						
30-34		707	1,268	1,798	-	-	-
35-44		1,703	3,055	4,331	-	-	-
45-54		1,817	3,260	4,622	-	-	-
55-64		2,604	4,671	6,623	-	-	-
65-74		3,104	5,568	7,895	-	-	-
75-84		2,343	4,202	5,957	-	-	-
85+		1,166	2,091	2,965	-	-	-
Life years lost	3% discount rate	-	-	-	$790	$5,346	$12,398
Life years lost	7% discount rate	-	-	-	$914	$6,207	$14,337
Age-Adjusted Value of Statistical Lives Lost	Jones-Lee et al. (1989) 3% discount rate	-	-	-	$3,546	$6,361	$9,018
	Jones-Lee et al. (1989) 7% discount rate	-	-	-	$3,331	$5,974	$8,470
	Jones-Lee et al. (1993) 3% discount rate	-	-	-	$5,617	$10,075	$14,284
	Jones-Lee et al. (1993) 7% discount rate	-	-	-	$5,276	$9,463	$13,417
Chronic Bronchitis	Reversals	194	1,095	1,980	$8	$183	$675

Alternative benefit estimates are presented to display the impact alternative assumptions have on total benefits. We caution, however, that multiple alternative calculations cannot be added together and substituted into the primary benefit estimate because the likelihood of all alternative assumptions occurring simultaneously is low. See the Heavy Duty Diesel Regulatory Impact Analysis for a more detailed discussion on the use of alternative estimates (U.S. EPA, 2000b).

Exhibit 5-9 Alternative Benefit Calculations for the 2040 SAMI "B1" Scenario

Endpoint	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
PM-related Alternative Calculations							
Life Years Lost, by age:	Krewski et al. (2000), Table 31						
30-34		1,924	3,458	4,910	-	-	-
35-44		4,611	8,288	11,767	-	-	-
45-54		4,795	8,619	12,237	-	-	-
55-64		6,740	12,113	17,198	-	-	-
65-74		7,983	14,347	20,368	-	-	-
75-84		6,012	10,805	15,340	-	-	-
85+		3,007	5,403	7,671	-	-	-
Life years lost	3% discount rate	-	-	-	$2,396	$16,253	$37,741
Life years lost	7% discount rate	-	-	-	$2,775	$18,830	$43,514
Age-Adjusted Value of Statistical Lives Lost	Jones-Lee et al. (1989) 3% discount rate	-	-	-	$10,736	$19,295	$27,395
	Jones-Lee et al. (1989) 7% discount rate	-	-	-	$10,084	$18,124	$25,731
	Jones-Lee et al. (1993) 3% discount rate	-	-	-	$16,955	$30,472	$43,262
	Jones-Lee et al. (1993) 7% discount rate	-	-	-	$15,925	$28,621	$40,634
Chronic Bronchitis	Reversals	515	2,877	5,169	$24	$573	$2,105

Exhibit 5-10 Alternative Benefit Calculations for the 2010 SAMI "B3" Scenario

Endpoint	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
PM-related Alternative Calculations							
Life Years Lost, by age:	Krewski et al. (2000), Table 31						
30-34		2,632	4,742	6,743	-	-	-
35-44		6,343	11,426	16,247	-	-	-
45-54		6,805	12,257	17,428	-	-	-
55-64		9,779	17,612	25,041	-	-	-
65-74		11,678	21,032	29,902	-	-	-
75-84		8,799	15,846	22,529	-	-	-
85+		4,367	7,864	11,181	-	-	-
Life years lost	3% discount rate	-	-	-	$2,979	$20,131	$46,724
Life years lost	7% discount rate	-	-	-	$3,439	$23,379	$53,960
Age-Adjusted Value of Statistical Lives Lost	Jones-Lee et al. (1989) 3% discount rate	-	-	-	$13,307	$23,966	$34,075
	Jones-Lee et al. (1989) 7% discount rate	-	-	-	$12,500	$22,511	$32,005
	Jones-Lee et al. (1993) 3% discount rate	-	-	-	$21,078	$37,960	$53,970
	Jones-Lee et al. (1993) 7% discount rate	-	-	-	$19,798	$35,654	$50,693
Chronic Bronchitis	Reversals	716	3,946	7,044	$28	$661	$2,423

Exhibit 5-11 Alternative Benefit Calculations for the 2040 SAMI "B3" Scenario

Endpoint	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
PM-related Alternative Calculations							
Life Years Lost, by age:	Krewski et al. (2000), Table 31						
30-34		3,600	6,489	9,233	-	-	-
35-44		8,630	15,556	22,131	-	-	-
45-54		9,030	16,274	23,150	-	-	-
55-64		12,741	22,961	32,658	-	-	-
65-74		15,133	27,268	38,784	-	-	-
75-84		11,389	20,522	29,188	-	-	-
85+		5,682	10,238	14,561	-	-	-
Life years lost	3% discount rate	-	-	-	$4,542	$30,781	$71,480
Life years lost	7% discount rate	-	-	-	$5,255	$35,677	$82,481
Age-Adjusted Value of Statistical Lives Lost	Jones-Lee et al. (1989) 3% discount rate	-	-	-	$20,291	$36,566	$52,010
	Jones-Lee et al. (1989) 7% discount rate	-	-	-	$19,059	$34,345	$48,851
	Jones-Lee et al. (1993) 3% discount rate	-	-	-	$32,057	$57,767	$82,165
	Jones-Lee et al. (1993) 7% discount rate	-	-	-	$30,110	$54,260	$77,176
Chronic Bronchitis	Reversals	959	5,270	9,389	$44	$1,050	$3,845

Exhibit 5-12 Alternative Mortality Calculations for the 2010 SAMI "B1" Scenario

Age Group	Statistic	Mortality	Reference	Mortality Incidence (cases/year)			Monetary Benefits (millions 2000$)		
				5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Age 30+	Mean	Non-Accidental	Krewski et al. (2000), Table 31	922	1,583	2,271	$1,609	$10,805	$26,256
Age 25+	Mean	Non-Accidental	Dockery et al. (1993)	1,821	4,280	6,615	$3,751	$28,628	$70,855
Age 25+	Mean	All-Cause	Dockery et al. (1993)	2,086	4,547	7,175	$3,768	$30,260	$77,049
Age 25+	Mean	Non-Accidental	Krewski et al. (2000)	2,355	4,584	6,960	$4,129	$31,255	$78,750
Age 25+	Mean	All-Cause	Krewski et al. (2000)	2,356	4,864	7,249	$4,430	$32,382	$82,749

Exhibit 5-13 Alternative Mortality Calculations for the 2040 SAMI "B1" Scenario

Age Group	Statistic	Mortality	Reference	Mortality Incidence (cases/year)			Monetary Benefits (millions 2000$)		
				5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Age 30+	Mean	Non-Accidental	Krewski et al. (2000), Table 31	2,374	4,068	5,829	$4,825	$32,383	$78,642
Age 25+	Mean	Non-Accidental	Dockery et al. (1993)	4,685	10,932	16,823	$11,225	$85,270	$211,237
Age 25+	Mean	All-Cause	Dockery et al. (1993)	5,368	11,622	18,252	$11,303	$90,186	$229,584
Age 25+	Mean	Non-Accidental	Krewski et al. (2000)	6,050	11,701	17,685	$12,306	$93,037	$234,447
Age 25+	Mean	All-Cause	Krewski et al. (2000)	6,058	12,424	18,438	$13,224	$96,451	$245,821

Exhibit 5-14 Alternative Mortality Calculations for the 2010 SAMI "B3" Scenario

Age Group	Statistic	Mortality	Reference	Mortality Incidence (cases/year)			Monetary Benefits (millions 2000$)		
				5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Age 30+	Mean	Non-Accidental	Krewski et al. (2000), Table 31	3,429	5,862	8,388	$5,968	$40,021	$97,280
Age 25+	Mean	Non-Accidental	Dockery et al. (1993)	6,742	15,617	23,919	$13,818	$104,460	$258,452
Age 25+	Mean	All-Cause	Dockery et al. (1993)	7,716	16,589	25,923	$13,895	$110,394	$281,087
Age 25+	Mean	Non-Accidental	Krewski et al. (2000)	8,696	16,704	25,123	$15,140	$113,900	$286,111
Age 25+	Mean	All-Cause	Krewski et al. (2000)	8,703	17,724	26,181	$16,183	$117,987	$300,270

Exhibit 5-15 Alternative Mortality Calculations for the 2040 SAMI "B3" Scenario

Age Group	Statistic	Mortality	Reference	Mortality Incidence (cases/year)			Monetary Benefits (millions 2000$)		
				5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Age 30+	Mean	Non-Accidental	Krewski et al. (2000), Table 31	4,461	7,623	10,904	$9,052	$60,688	$147,523
Age 25+	Mean	Non-Accidental	Dockery et al. (1993)	8,776	20,286	31,033	$20,959	$158,237	$391,388
Age 25+	Mean	All-Cause	Dockery et al. (1993)	10,048	21,561	33,647	$21,072	$167,317	$426,021
Age 25+	Mean	Non-Accidental	Krewski et al. (2000)	11,314	21,696	32,588	$22,992	$172,507	$432,478
Age 25+	Mean	All-Cause	Krewski et al. (2000)	11,331	23,033	33,982	$24,531	$178,794	$454,594

6. Unquantified Benefits From Other Pollutant Reductions

The SAMI integrated assessment generated changes in annual $PM_{2.5}$ concentrations associated with future year scenarios that control emissions from utility, industrial, highway vehicle, non-road engines, and area sources. Using these annual $PM_{2.5}$ concentrations, we then estimated the change in premature mortality, chronic bronchitis, and other measures. While this may have captured the bulk of the economic benefits associated with reducing these emission sources, we still miss a variety of potential benefits. In addition to reductions in $PM_{2.5}$, the emission reductions likely to be achieved under each of the SAMI emission control scenarios are expected to also reduce exposure to ambient concentrations of PM_{10} and particulate matter between 2.5 and 10 microns (coarse PM_{10}), as well as NO_2, SO_2, CO, hazardous air pollutants (HAPs) and ozone.

Previous analyses have found that these pollutants are associated with adverse health effects, as well as crop and forestry loss, nitrogen deposition and resulting algal blooms, and other effects (e.g., U.S. EPA, 1999b). Since this analysis did not model the change in the ambient concentrations of these other pollutants, there is no direct method to estimate their effects. One might try to indirectly estimate these benefits, but, as discussed below, such approaches have significant problems that preclude quantifying these potential health and welfare effects.

One indirect method is to estimate the health and welfare effects of changing the ambient concentrations of these other pollutants by looking at the relative level of benefits in previous studies and extrapolating them to the current study. For example, if a study found annual $PM_{2.5}$ studies had ten times the benefits of ozone, then one might assume – under certain conditions -- that the ozone benefits for the present study are a tenth of the annual $PM_{2.5}$ benefits. Alternatively, one might rely on previous estimates of the benefits of reducing a ton of emissions, and apply these dollar per ton estimates to the present study. Unfortunately the conditions under which one may make these assumptions are difficult to meet. The result is that indirect measures to estimate the impact changing concentrations of NO_2, SO_2, ozone, and CO are highly uncertain. The problem is compounded when considering toxic pollutants. Even if one were able to estimate the change in ambient concentrations, significant difficulties remain in estimating benefits.

The first section briefly outlines the health and welfare effects associated with pollutants such as NO_2, SO_2, CO, and hazardous air pollutants (HAPs). The second section briefly outlines methods to extrapolate the results from previous studies to the current study, and it presents a table summarizing previous estimates of the relative impact of PM and ozone.

6.1 Other Pollutants (NO_2, SO_2, CO, and HAPs)

Benefits of Reduced Nitrous Oxide Emissions

Emissions of NOx produce a wide variety of health and welfare effects (EPA,1999b). Nitrogen dioxide can irritate the lungs and lower resistance to respiratory infection (such as influenza). NOx emissions are an important precursor to acid rain and may affect both terrestrial and aquatic ecosystems. Atmospheric deposition of nitrogen leads to excess nutrient enrichment problems ("eutrophication") in the Chesapeake Bay and several nationally important estuaries along the East and Gulf Coasts. Eutrophication can produce multiple adverse effects on water quality and the aquatic environment, including increased algal blooms, excessive phytoplankton growth, and low or no dissolved oxygen in bottom waters. Eutrophication also reduces sunlight, causing losses in submerged aquatic vegetation critical for healthy estuarine ecosystems. Deposition of nitrogen-containing compounds also affects terrestrial ecosystems. Nitrogen fertilization can alter growth patterns and change the balance of species

in an ecosystem. Nitrogen dioxide and airborne nitrate also contribute to pollutant haze, which impairs visibility and can reduce residential property values and the value placed on scenic views.

NOx in combination with volatile organic compounds (VOC) also serve as precursors to ozone. PM can also be formed from NOx emissions; secondary PM is formed in the atmosphere through a number of physical and chemical processes that transform gases, such as NOx, into particles. The effects of secondary PM and ozone exposures due to NOx emissions are the same as those of directly emitted PM and ozone.

Benefits of Sulfur Dioxide Reductions

High concentrations of sulfur dioxide (SO2) affect breathing and may aggravate existing respiratory and cardiovascular disease. Sensitive populations include asthmatics, individuals with bronchitis or emphysema, children and the elderly. SO2 is also a primary contributor to acid deposition, or acid rain, which causes acidification of lakes and streams and can damage trees, crops, historic buildings and statues. In addition, sulfur compounds in the air contribute to visibility impairment in large parts of the country. This is especially noticeable in national parks.

PM can also be formed from SO2 emissions. Secondary PM is formed in the atmosphere through a number of physical and chemical processes that transform gases, such as SO2, into particles. The effects of secondary PM exposures due to SO2 emissions are the same as those of directly emitted PM.

Benefits of Reduction in Carbon Monoxide Emissions

Human health effects associated with exposure to CO include cardiovascular system and central nervous system (CNS) effects. Cardiovascular effects of CO are directly related to reduced oxygen content of blood, resulting in tissue hypoxia (i.e., oxygen starvation). Most healthy individuals have mechanisms (e.g. increased blood flow, blood vessel dilation) which compensate for this reduction in tissue oxygen, although the effect of reduced maximal exercise capacity has been reported in some healthy persons. Several other medical conditions such as occlusive vascular disease, chronic obstructive lung disease, and anemia can increase susceptibility to potential adverse effects of CO during exercise. Effects of CO on the CNS involve both behavioral and physiological changes. These include modification of visual perception, hearing, motor and sensorimotor performance, vigilance, and cognitive ability.

Although acute poisoning induced by CO can be lethal and is probably the best known health endpoint of CO, this only occurs at very high concentrations of CO (greater than 100 ppm, hourly average). In the ambient air, exposures to lower-levels of CO predominate and at these levels the best documented adverse health endpoint in human subjects is the decrease in time to onset of reproducible exercise-induced chest pain. Results of some human exposure studies and reports of workers routinely exposed to combustion products provide support for recent epidemiology research suggesting day-to-day variations in ambient CO concentrations are related to cardiovascular hospital admissions and daily mortality, especially for individuals over 65 years of age. Uncertainties about the association between these health endpoints and ambient CO and the relative influence of indoor vs. outdoor CO have not been resolved and will require further research.

There are certain people who are more "at risk" to CO exposures. Individuals with preexisting illness or cardiovascular diseases which limit oxygen absorption or oxygen transport to body tissues would be somewhat more susceptible to the effects of CO. Very little data are available demonstrating human health effects in healthy individuals caused by or associated with exposures to low CO concentrations. Decrements in maximal exercise duration and performance in healthy individuals have been reported, however, these decrements are small and likely to affect only athletes in competition. No

effects were seen in healthy individuals during submaximal exercise, representing more typical daily activities. Most recent evidence of CNS effects induced by exposure to CO indicates that behavioral impairments in healthy individuals should not be expected until CO levels are well above what would be caused by typical ambient air levels of CO. Also, evidence of CO-induced fetal toxicity or of interactions with high altitudes, drugs, other pollutants, or other environmental stresses remains uncertain or suggests that effects of concern will occur in healthy individuals only with exposure to very high levels of CO.

Benefits from Reductions in Hazardous Air Pollutants (HAPs)

Human exposure to HAPs may occur directly through inhalation or indirectly through ingestion of food or water contaminated by HAPs or through exposure to the skin. HAPs may also enter terrestrial and aquatic ecosystems through atmospheric deposition. HAPs can be deposited on vegetation and soil through wet or dry deposition. HAPs may also enter the aquatic environment from the atmosphere via gas exchange between surface water and the ambient air, wet or dry deposition of particulate HAPs and particles to which HAPs adsorb, and wet or dry deposition to watersheds with subsequent leaching or runoff to bodies of water (EPA,1992a). This analysis is focused only on the air quality benefits of HAP reduction.

Health Benefits of HAP Reductions

The HAP emission reductions likely to be achieved under each of the SAMI emission control scenarios are expected to reduce exposure to ambient concentrations of arsenic, cadmium, chromium, hydrogen chloride, hydrogen flouride, lead, manganese, mercury, and nickel, which will reduce a variety of adverse health effects considering both cancer and noncancer endpoints. These adverse health effects include chronic health disorders (e.g., irritation of the lung, skin, and mucus membranes and effects on the blood, digestive tract, kidneys, and central nervous system), and acute health disorders (e.g., lung irritation and congestion, alimentary effects such as nausea and vomiting, and effects on the central nervous system). EPA has classified several of these HAPs as known or probable human carcinogens.

Noncancer health effects can be generally grouped into the following broad categories: genotoxicity, developmental toxicity, reproductive toxicity, systemic toxicity, and irritation. Genotoxicity is a broad term that usually refers to a chemical that has the ability to damage DNA or the chromosomes. Developmental toxicity refers to adverse effects on a developing organism that may result from exposure prior to conception, during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism. Reproductive toxicity refers to the harmful effects of HAP exposure on fertility, gestation, or offspring, caused by exposure of either parent to a substance. Systemic toxicity affects a portion of the body other than the site of entry. Irritation, for the purpose of this document, refers to any effect which results in irritation of the eyes, skin, and respiratory tract. Though we do not know the extent to which the adverse health effects described above occur in the SAMI region population, to the extent the adverse effects do occur, the emission control scenarios will likely reduce emissions and subsequent exposures.

Welfare Benefits of HAP Reductions

The welfare effects of exposure to HAPs have received less attention from analysts than the health effects. However, this situation is changing, especially with respect to the effects of toxic substances on ecosystems. Over the past ten years, ecotoxicologists have started to build models of ecological systems which focus on interrelationships in function, the dynamics of stress, and the adaptive potential for recovery. Chronic sub-lethal exposures may affect the normal functioning of individual species in ways that make it less than competitive and therefore more susceptible to a variety of factors including disease, insect attack, and decreases in habitat quality. All of these factors may contribute to an overall change in the structure (i.e., composition) and function of the ecosystem.

The adverse, non-human biological effects of HAP emissions include ecosystem and recreational and commercial fishery impacts. Atmospheric deposition of HAPs directly to land may affect terrestrial ecosystems. Atmospheric deposition of HAPs also contributes to adverse aquatic ecosystem effects. This not only has adverse implications for individual wildlife species and ecosystems as a whole, but also the humans who may ingest contaminated fish and waterfowl. In general, HAP emission reductions achieved through the Industrial Boilers and Process Heaters NESHAP should reduce the associated adverse environmental impacts.

6.2 Extrapolation of Benefits from Other Studies

Previous studies estimating the impact of PM and other pollutants have generally found that monetary benefits associated with reducing PM outweigh the benefits achieved from reducing other pollutants, such as NO_2, SO_2, ozone, and CO.[15] To get a rough idea of the non-PM benefits achieved by reducing automotive and power plant emissions, one might extrapolate past results to the present study.

Two ways might be considered:

- Estimate the dollar benefits per ton of emission reduction from previous studies, where the dollar per ton estimates is calculated by dividing estimates of benefits by the emissions associated with these benefits.

- Estimate the relative damages of PM to other pollutants from previous studies, and assume that these relative damages apply to the current analysis. For example, if the health and welfare benefits of PM associated with PM reductions are ten times the health and welfare benefits associated with other pollutants, then one might assume that a similar ratio holds for the present study.

With the exception of using impacts associated with annual $PM_{2.5}$ to estimate other impacts associated with particulate matter, these two approaches fail to provide reliable estimates for other pollutants. Previous analyses deviate from the current analysis in one or more ways that significantly reduce their comparability with the present study. They differ in terms of: baseline assumptions, emissions reductions, geographic area, choice of health and welfare effects, valuation of effects, and so on. A pollutant like ozone results from a complex, temperature-dependent interaction between NO_x and volatile organic carbon (VOC); in some cases ozone levels will *increase* when the concentrations of one of its precursors *decreases* (U.S. EPA, 1996, Figure 3-25). It is difficult – without an air quality model – to determine if the relevant changes in previous studies would be similar to the relative changes in the present study.

Exhibit 6-1 summarizes the dollar benefits associated with the annual $PM_{2.5}$ C-R functions versus daily $PM_{2.5}$ and PM_{10} functions. The dollar benefits of these other PM measures are no more than five percent of the dollar benefits associated with annual $PM_{2.5}$. However, these other measures of PM capture a range of effects categories that we have not captured thus far in the analysis, such as hospital admissions (Samet et al., 2000), emergency room visits (Schwartz et al., 1993), lower respiratory symptoms (Schwartz et al., 1994), upper respiratory symptoms (Pope et al., 1991), work loss day (Ostro, 1987), MRAD (Ostro and Rothschild, 1989), and recreational visibility reduction. The number of persons affected by these excluded categories greatly exceeds those affected by the C-R functions linked to annual $PM_{2.5}$.

[15] Lead is a criteria pollutant, but it is discussed in the next section on toxic pollutants.

Exhibit 6-1 Comparison of Annual $PM_{2.5}$ and Other PM-related Benefits From Previous Analyses

Study	Benefits by Category ($ million)	Total Benefits ($ million)
Heavy Duty Diesel (Abt Associates Inc., 2000, Exhibits 6-2 & 6-3). Location: 48 contiguous U.S. States for PM, and 37 Eastern U.S. States for ozone.	*Annual $PM_{2.5}$ effects*: mortality ($48,129), chronic bronchitis ($1,803), acute bronchitis ($1)	$49,933
	Other PM effects: hospital admissions ($82), emergency room visits ($1), lower respiratory symptoms ($3), upper respiratory symptoms ($5), work loss day ($178), MRAD ($391), recreational visibility reduction ($1,789)	2,449
	Other PM effects as percent of annual $PM_{2.5}$ effects	5%
Tier 2 (Abt Associates Inc., 1999, Exhibit 6-2 & 6-3). Location: 48 contiguous U.S. States.	*Annual $PM_{2.5}$ effects*: mortality ($23,370), chronic bronchitis ($727), acute bronchitis ($0.4)	$24,097
	Other PM effects: hospital admissions ($18), emergency room visits ($0.3), lower respiratory symptoms ($1), upper respiratory symptoms ($2), work loss day ($70), MRAD ($173), recreational visibility reduction ($371)	635
	Other PM effects as percent of annual $PM_{2.5}$ effects	3%
"NO_x SIP Call" (Abt Associates Inc., 1998a, Exhibits 4.b.1 & 4.b.2; Abt Associates Inc., 1998b, Exhibit 13). Location: 37 Eastern U.S. States.	*Annual $PM_{2.5}$ effects*: mortality ($1,123), chronic bronchitis ($187), acute bronchitis (< $0.1)	$1,309
	Other PM effects: hospital admissions ($2), lower respiratory symptoms ($0.1), upper respiratory symptoms (< $0.1), work loss day ($6), MRAD ($22), household soiling ($9), recreational visibility reduction ($30)	68
	Other PM effects as percent of annual $PM_{2.5}$ effects	5%

[a] Average of RADM and S-R. results for 37 Eastern U.S. states from the 0.15 Trading Alternative. Numbers may not sum to the total due to rounding.

Exhibit 6-2 compares the estimated benefits associated with annual $PM_{2.5}$ and ozone benefits from previous studies. For the Heavy Duty Diesel and Tier 2 analyses, both transportation-related analyses, the annual $PM_{2.5}$ benefits dominate the benefits associated with ozone, with ozone representing three percent or less of annual $PM_{2.5}$ benefits. For the NO_x SIP Call, an analysis of NO_x power plant emission reductions, the ozone benefits are about 24 percent of the annual $PM_{2.5}$ benefits, due in large part to ozone-related agricultural crop losses. Since the SAMI program affects both automotive and power plant emission sources, the relative contribution of ozone is likely to be somewhere between these two prior estimates. However, we guess that it is likely to tend toward the lower end of this range, because the annual $PM_{2.5}$ benefits in the current analysis are an order of magnitude greater than that found in the NO_x SIP Call, and the impact of ozone on agricultural sources in the eight SAMI states is unlikely to be as large as that found in the 37 state NO_x SIP Call analysis.

Including the estimated benefits of NO_2, SO_2, and CO probably would not have changed the results in Exhibit 6-2 greatly. Changes in NO_2, SO_2, and CO are generally related to a relatively small subset of effects; the most serious of which is perhaps hospitalization for heart-related problems (e.g., Schwartz and Morris, 1995). There have been studies finding some evidence that NO_2 and CO are linked to mortality but it is difficult to determine if these effects are in addition to any effects associated with PM

and ozone (e.g., Kinney and Ozkayank, 1991; Kinney et al., 1995).[16] To the extent that PM and ozone are acting as indicators for NO_2, SO_2, and CO, then it is reasonable to simply look at the relative effects of PM and ozone.

Exhibit 6-2 Comparison of Annual $PM_{2.5}$ and Ozone Benefits From Previous Analyses

Study	Benefits by Category ($ million)	Total Benefits ($ million)
Heavy Duty Diesel (Abt Associates Inc., 2000, Exhibits 6-2 & 6-3). Location: 48 contiguous U.S. States for PM, and 37 Eastern U.S. States for ozone.	*Annual $PM_{2.5}$ effects*: mortality ($48,129), chronic bronchitis ($1,803), acute bronchitis ($1)	$49,933
	Ozone effects: hospital admissions ($21), emergency room visits ($0.1), MRAD ($90), decreased worker productivity ($142), agricultural crop losses ($1,078)	$1,331
	Ozone effects as percent of annual $PM_{2.5}$ effects	3%
Tier 2 (Abt Associates Inc., 1999, Exhibit 6-2 & 6-3). Location: 48 contiguous U.S. States.	*Annual $PM_{2.5}$ effects*: mortality ($23,370), chronic bronchitis ($727), acute bronchitis ($0.4)	$24,097
	Ozone effects: hospital admissions ($13), emergency room visits ($0.1), MRAD ($101), decreased worker productivity ($142), agricultural crop losses ($217)	$473
	Ozone effects as percent of annual $PM_{2.5}$ effects	2%
"NO_x SIP Call" (Abt Associates Inc., 1998b, Exhibit 13). Location: 37 Eastern U.S. States.	*Annual $PM_{2.5}$ effects*: mortality ($1,123), chronic bronchitis ($187), acute bronchitis (< $0.1)	$1,309 [a]
	Ozone effects: hospital admissions ($4), any-of-19 symptoms ($1), decreased worker productivity ($22), agricultural crop losses ($283 million)	$310
	Ozone effects as percent of annual $PM_{2.5}$ effects	24%

[a] Average of RADM and S-R. results for 37 Eastern U.S. states from the 0.15 Trading Alternative.

[16] Some fraction of persons hospitalized subsequently die at the hospital. CO may thus be related to mortality, although the evidence for this relationship is not well developed.

7. References

Abbey, D.E., F. Petersen, P.K. Mills and W.L. Beeson. 1993. Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. Vol. 48(1): 33-46.

Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren and P.K. Mills. 1995a. Estimated Long-Term Ambient Concentrations of Pm(10) and Development of Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. Vol. 50(2): 139-152.

Abbey, D.E., B.E. Ostro, F. Petersen and R.J. Burchette. 1995b. Chronic Respiratory Symptoms Associated with Estimated Long-Term Ambient Concentrations of Fine Particulates Less Than 2.5 Microns in Aerodynamic Diameter (PM2.5) and Other Air Pollutants. J Expo Anal Environ Epidemiol. Vol. 5(2): 137-159.

Abbey, D.E., R.J. Burchette, S.F. Knutsen, W.F. McDonnell, M.D. Lebowitz and P.L. Enright. 1998. Long-term particulate and other air pollutants and lung function in nonsmokers. American Journal of Respiratory and Critical Care Medicine. Vol. 158(1): 289-298.

Abt Associates Inc. 1998a. Agricultural Benefits Using AGSIM for the NOx SIP Call. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, under contract no. 68-D-98-001. Research Triangle Park, NC. September.

Abt Associates Inc. 1998b. Benefit Analysis Results of Selected Health and Welfare Endpoints for the NOx SIP Call RIA. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, under contract no. 68-D-98-001. Research Triangle Park, NC. September.

Abt Associates Inc. 1999. Final Tier 2 Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC. Bethesda, MD. December.

Abt Associates Inc. 2000. Final Heavy Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Slelected Health and Welfare Benefits Methods, and Benefit Analysis Results. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC. Bethesda, MD. December.

Abt Associates Inc. 2002a. The Value of Visibility Improvements in the Southern Appalachian Mountains Region. Prepared for Southern Appalachian Mountains Initiative, Asheville, NC. Bethesda, MD. April.

Abt Associates Inc. 2002b. The Value of Improved Recreational Fishing from Reduced Acid Deposition in the Southern Appalachian Mountains Region. Prepared for Southern Appalachian Mountains Initiative, Asheville, NC. Bethesda, MD. March.

Ackermann-Liebrich, U., P. Leuenberger, J. Schwartz, C. Schindler, C. Monn, C. Bolognini, J.P. Bongard, O. Brandli, G. Domenighetti, S. Elsasser, L. Grize, W. Karrer, R. Keller, H. KellerWossidlo, N. Kunzli, B.W. Martin, T.C. Medici, A.P. Perruchoud, M.H. Schoni, J.M. Tschopp, B. Villiger, B. Wuthrich, J.P. Zellweger and E. Zemp. 1997. Lung function and long term exposure to air pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. Am J Respir Crit Care Med. Vol. 155(1): 122-129.

Adams, P.F. and M.A. Marano. 1995. Current Estimates from the National Health Interview Survey, 1994. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10, No. 193. December.

Alberini, A., M. Cropper, T.T. Fu, A. Krupnick, J.T. Liu, D. Shaw and W. Harrington. 1997. Valuing health effects of air pollution in developing countries: The case of Taiwan. Journal of Environmental Economics and Management. Vol. 34(2): 107-126.

Butler, R.J. 1983. Wage and Injury Rate Responses to Shifting Levels of Workers' Compensation. In: Safety and the Work Force. Worrall, J.D., Ed. Cornell University, ILR Press: Ithaca, NY.

Chestnut, L.G. 1995. Dollars and Cents: The Economic and Health Benefits of Potential Particulate Matter Reductions in the United States. Prepared for American Lung Association.

Collins, J.G. 1997. Prevalence of Selected Chronic Conditions: United States 1990-1992. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10. No. 194.

Council of Economic Advisers. 2000. The Annual Report of the Council of Economic Advisers. In: Economic Report of the President. U.S. Government Printing Office: Washington, DC.

Cousineau, J., R. Lacroix and A. Girard. 1988. Occupational Hazard and Wage Compensating Differentials. University of Montreal Working Paper.

Cousineau, J., R. Lacroix and A. Girard. 1992. Occupational Hazard and Wage Compensating Differentials. The Review of Economics and Statistics. Vol. 74: 166-169.

Cropper, M.L. and A.J. Krupnick. 1990. The Social Costs of Chronic Heart and Lung Disease. Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.

Detels, R., D.P. Tashkin, J.W. Sayre, S.N. Rokaw, F.J. Massey, A.H. Coulson and D.H. Wegman. 1991. The Ucla Population Studies of Cord .10. a Cohort Study of Changes in Respiratory Function Associated With Chronic Exposure to Sox, Nox, and Hydrocarbons. American Journal of Public Health. Vol. 81(3): 350-359.

Dillingham, A. 1985. The Influence of Risk Variable Definition on Value of Life Estimates. Economic Inquiry. Vol. 24: 277-294.

Dockery, D.W., F.E. Speizer, D.O. Stram, J.H. Ware, J.D. Spengler and B.G. Ferris, Jr. 1989. Effects of Inhalable Particles on Respiratory Health of Children. Am Rev Respir Dis. Vol. 139: 587-594.

Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris and F.E. Speizer. 1993. An association between air pollution and mortality in six U.S. cities. N Engl J Med. Vol. 329(24): 1753-1759.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer. 1996. Health Effects of Acid Aerosols On North American Children - Respiratory Symptoms. Environmental Health Perspectives. Vol. 104(5): 500-505.

Ehrlich, I. and H. Chuma. 1990. A Model of the Demand For Longevity and the Value of Life Extension. Journal of Political Economy. Vol. 98(4): 761-782.

Garen, J. 1988. Compensating Wage Differentials and the Endogeneity of Job Riskiness. The Review of Economics and Statistics. Vol. 70(1): 9-16.

Gegax, D., S. Gerking and W. Shulze. 1985. Perceived Risk and the Marginal Value of Safety. Working paper prepared for the U. S. Environmental Protection Agency.

Gegax, D., S. Gerking and W. Shulze. 1991. Perceived Risk and the Marginal Value of Safety. The Review of Economics and Statistics. Vol. 73(4): 589-596.

Gerking, S., M. DeHaan and W. Schulze. 1988. The Marginal Value of Job Safety: A Contingent Valuation Study. Journal of Risk and Uncertainty. Vol. 1: 185-199.

Health Review Committee. 2000. Commentary: Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Health Effects Institute. Cambridge. July.

Herzog, H.W., Jr., and A.M. Schlottmann. 1987. Valuing Risk in the Workplace: Market Price, Willingness to Pay, and the Optimal Provision of Safety. University of Tennessee Working Paper.

Herzog, H.W., Jr., and A.M. Schlottmann. 1990. Valuing Risk in the Workplace: Market Price, Willingness to Pay, and the Optimal Provision of Safety. The Review of Economics and Statistics. Vol. 72(3): 463-470.

Holland, M., D. Forster and M. Wenborn. 1999. Economic Valuation of Proposals Under the UNECE Multi-Effects and Multi-Pollutant Protocol. Prepared for: European Commission, DGXI. Brussels and Luxembourg. AEAT-4587. January.

Industrial Economics Incorporated (IEc). 1992. Review of Existing Value of Life Estimates: Valuation Document. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. November 6.

Industrial Economics Incorporated (IEc). 1993. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. September 30.

Industrial Economics Incorporated (IEc). 1994. Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis -- Draft Valuation Document. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. Prepared by J.E. Neumann, M.T. Dickie, and R.E. Unsworth. March 31.

Jones-Lee, M.W., M. Hammerton and P.R. Philips. 1985. The Value of Safety: Result of a National Sample Survey. Economic Journal. Vol. 95(March): 49-72.

Jones-Lee, M.W. 1989. The Economics of Safety and Physical Risk. Basil Blackwell: Oxford.

Jones-Lee, M.W., G. Loomes, D. O'Reilly and P.R. Phillips. 1993. The Value of Preventing Non-fatal Road Injuries: Findings of a Willingness-to-pay National Sample Survey. TRY Working Paper, WP SRC2.

Kennedy. 1990. A Guide to Econometrics. 2nd ed. MIT Press: Cambridge, MA.

Kinney, P.L. and H. Ozkayank. 1991. Associations of Daily Mortality and Air Pollution in Los Angeles County. Environmental Research. Vol. 54: 99-120.

Kinney, P.L., K. Ito and G.D. Thurston. 1995. A Sensitivity Analysis of Mortality Pm-10 Associations in Los Angeles. Inhalation Toxicology. Vol. 7(1): 59-69.

Kniesner, T.J. and J.D. Leeth. 1991. Compensating Wage Differentials for Fatal Injury Risk in Australia, Japan, and the United States. Journal of Risk and Uncertainty. Vol. 4(1): 75-90.

Krewski, D., R. Burnett, M. Goldberg, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz and M. White. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Health Effects Institute. Cambridge. July.

Krupnick, A.J. and M.L. Cropper. 1992. The Effect of Information On Health Risk Valuations. Journal of Risk and Uncertainty. Vol. 5(1): 29-48.

Krupnick, A.J. 2000. Resources for the Future. Personal communication with Lisa Akeson. December 5.

Krupnick, A.J., A. Alberini, M. Cropper, N. Simon, B. O'Brien, R. Goeree and M. Heintzelman. 2000. Age, Health, and the Willingness to Pay for Mortality Risk Reductions: A Contingent Valuation Survey of Ontario Residents. Resources for the Future. Washington, DC. Discussion Paper 00-37. September.

Lang, C., G. Yarwood, F. Lalonde and R. Bloxam. 1995. Environmental and Health Benefits of Cleaner Vehicles and Fuels. Prepared for: Canadian Council of Ministers of the Environment Task Force on Cleaner Vehicles and Fuels. Winnipeg, Manitoba. October.

Leigh, J.P. and R.N. Folsom. 1984. Estimates of the Value of Accident Avoidance at the Job Depend on Concavity of the Equalizing Differences Curve. The Quarterly Review of Economics and Business. Vol. 24(1): 56-66.

Leigh, J.P. 1987. Gender, Firm Size, Industry and Estimates of the Value-of-Life. Journal of Health Economics. Vol. 6: 255-273.

Loehman, E.T. and V.H. De. 1982. Application of Stochastic Choice Modeling to Policy Analysis of Public Goods. The Review of Economics and Statistics. Vol. 64(3): 474-480.

Marin, A. and G. Psacharopoulos. 1982. The Reward for Risk in the Labor Market: Evidence from the United Kingdom and a Reconciliation with Other Studies. Journal of Political Economy. Vol. 90(4): 827-853.

Miller, T. and J. Guria. 1991. The Value of Statistical Life in New Zealand. Report to the New Zealand Ministry of Transport, Land Transport Division.

Mitchell, R.C. and R.T. Carson. 1986. The Use of Contingent Valuation Data for Benefit/Cost Analysis in Water Pollution Control. Draft report submitted by Resources for the Future to Environmental Protection Agency, Office of Policy Analysis. Washington, DC. September.

Moore, M.J. and W.K. Viscusi. 1988. Doubling the Estimated Value of Life: Results Using New Occupational Fatality Data. Journal of Policy Analysis and Management. Vol. 7(3): 476-490.

National Center for Health Statistics. 1999. National Vital Statistics Reports. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. Washington, DC. Volume 47, Number 19. June 30.

Olson, C.A. 1981. An Analysis of Wage Differentials Received by Workers on Dangerous Jobs. Journal of Human Resources. Vol. 16: 167-185.

Ostro, B.D. 1987. Air Pollution and Morbidity Revisited: A Specification Test. Journal of Environmental Economics and Management. Vol. 14: 87-98.

Ostro, B.D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity - an Observational Study of Multiple Pollutants. Environ Res. Vol. 50(2): 238-247.

Ostro, B.D., J.M. Sanchez, C. Aranda and G.S. Eskeland. 1996. Air Pollution and Mortality - Results From a Study of Santiago, Chile. J Expo Anal Environ Epidemiol. Vol. 6(1): 97-114.

Pope, C.A., D.W. Dockery, J.D. Spengler and M.E. Raizenne. 1991. Respiratory Health and Pm10 Pollution - a Daily Time Series Analysis. American Review of Respiratory Disease. Vol. 144(3): 668-674.

Pope, C.A., J. Schwartz and M.R. Ransom. 1992. Daily Mortality and PM10 Pollution in Utah Valley. Archives of Environmental Health. Vol. 47(3): 211-217.

Pope, C.A., M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer and C.W. Heath. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med. Vol. 151(3): 669-674.

Samet, J., S. Zeger, F. Dominici, F. Curriero, I. Coursac, D. Dockery, J. Schwartz and A. Zanobetti. 2000. The National Morbidity, Mortality, and Air Pollution Study. Health Effects Institute. Cambridge, MA. Report No. 94. May.

Schwartz, J. and D.W. Dockery. 1992. Particulate Air Pollution and Daily Mortality in Steubenville, Ohio. American Journal of Epidemiology. Vol. 135(1): 12-19.

Schwartz, J. 1993. Particulate Air Pollution and Chronic Respiratory Disease. Environ Res. Vol. 62: 7-13.

Schwartz, J., D. Slater, T.V. Larson, W.E. Pierson and J.Q. Koenig. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis. Vol. 147(4): 826-31.

Schwartz, J., D.W. Dockery, L.M. Neas, D. Wypij, J.H. Ware, J.D. Spengler, P. Koutrakis, F.E. Speizer and B.G. Ferris. 1994. Acute Effects of Summer Air Pollution On Respiratory Symptom Reporting in Children. Am J Respir Crit Care Med. Vol. 150(5): 1234-1242.

Schwartz, J. and R. Morris. 1995. Air Pollution and Hospital Admissions For Cardiovascular Disease in Detroit, Michigan. American Journal of Epidemiology. Vol. 142(1): 23-35.

Schwartz, J. 2000. Harvesting and long term exposure effects in the relation between air pollution and mortality [see comments]. Am J Epidemiol. Vol. 151(5): 440-8.

Smith, R.S. 1974. The Feasibility of an 'Injury Tax' Approach to Occupational Safety. Law and Contemporary Problems. Vol. 38(4): 730-744.

Smith, R.S. 1976. The Occupational Safety and Health Act: Its Goals and Achievements. American Enterprise Institute. Washington, DC.

Smith, V.K. 1983. The Role of Site and Job Characteristics in Hedonic Wage Models. Journal of Urban Economics. Vol. 13: 296-321.

Smith, V.K. and C. Gilbert. 1984. The Implicit Risks to Life: A Comparative Analysis. Economics Letters. Vol. 16: 393-399.

Southern Appalachian Mountains Initiative. 2001. 2001 Interim Report. Asheville, North Carolina. May.

Spix, C., J. Heinrich, D. Dockery, J. Schwartz, G. Volksch, K. Schwinkowski, C. Collen and H.E. Wichmann. 1993. Air pollution and daily mortality in Erfurt, east Germany, 1980-1989. Environ Health Perspect. Vol. 101(6): 518-26.

U.S. Bureau of Economic Analysis. 1995. BEA Regional Projections to 2045: Volume 1, States. U.S. Department of Commerce. Washington, DC. July.

U.S. Bureau of the Census. 1998. Statistical Abstract of the United States: 1998. 118 ed. Washington, DC.

U.S. Centers for Disease Control. 1999. CDC Wonder.

U.S. EPA. 1996. Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. OAQPS Staff Paper. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA-452\R-96-007. June.

U.S. EPA. 1999a. An SAB Advisory: The Clean Air Act Section 812 Prospective Study Health and Ecological Initial Studies. Prepared by the Health and Ecological Effects SubCommittee (HEES) of the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-Council-ADV-99-005. February.

U.S. EPA. 1999b. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Advisory Council on Clean Air Compliance Analysis: Costs and Benefits of the CAAA. Prepared by the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-COUNCIL-ADV-00-002. October 29.

U.S. EPA. 1999c. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on initial assessments of Health and Ecological Effects; Part 2. Prepared by the Health and Ecological Effects Subcommittee, Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-COUNCIL-ADV-00-001. October 29.

U.S. EPA. 1999d. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects; Part 1. Prepared by the Health and Ecological Effects SubCommittee (HEES) of the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-Council-ADV-99-012. July 28.

U.S. EPA. 2000a. An SAB Report on EPA's White Paper Valuing the Benefits of Fatal Cancer Risk Reduction. Prepared by the Environmental Economics Advisory Committee (EEAC) of the Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-EEAC-00-013. July 27,.

U.S. EPA. 2000b. Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements. U.S. EPA, Office of Air and Radiation. Washington, DC. EPA 420-R-00-026. December.

Violette, D.M. and L.G. Chestnut. 1983. Valuing Reductions in Risks: A Review of the Empirical Estimates. Prepared for U.S. Environmental Protection Agency. Washington DC. EPA-230-05-83-002.

Viscusi, W.K. 1978. Labor Market Valuations of Life and Limb: Empirical Estimates and Policy Implications. Public Policy. Vol. 26(3): 359-386.

Viscusi, W.K. 1979. Employment Hazards: An Investigation of Market Performance. Harvard University Press: Cambridge.

Viscusi, W.K. 1981. Occupational Safety and Health Regulation: Its Impact and Policy Alternatives. In: Research in Public Policy Analysis and Management. Crecine, J., Ed. JAI Press: Greenwich, CT. Vol: 2. p. 281-299.

Viscusi, W.K., W.A. Magat and J. Huber. 1991. Pricing Environmental Health Risks - Survey Assessments of Risk - Risk and Risk - Dollar Trade-Offs For Chronic Bronchitis. Journal of Environmental Economics and Management. Vol. 21(1): 32-51.

Viscusi, W.K. 1992. Fatal Tradeoffs: Public and Private Responsibilities for Risk. Oxford University Press: New York.

Wessex, I. 1994. PRO/FILER, US Demographics and ZIPS: Summary Tape File 1A. Wessex, Inc.

Zeger, S.L., F. Dominici and J. Samet. 1999. Harvesting-resistant estimates of air pollution effects on mortality. Epidemiology. Vol. 10(2): 171-5.

Appendix A: Results for Sensitivity Analyses

Exhibit A-1 Sensitivity Analysis Results for the 2010 SAMI "B1" Scenario

Mortality Lag	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	r = 3%	r = 5%	r = 7%
No Lag		-	1,662	-	$11,690	$11,690	$11,690
5 Year	25%, 25%, 17%, 17%, 16%	-	1,662	-	$11,114	$10,764	$10,439
8 Year	Incidence Occurs 8th Year	-	1,662	-	$9,505	$8,308	$7,280
15 Year	Incidence Occurs 15th Year	-	1,662	-	$7,729	$5,904	$4,534
15 Year	Incidence Skewed Early	-	1,662	-	$10,882	$10,433	$10,041
15 Year	Incidence Skewed Late	-	1,662	-	$8,384	$6,794	$5,556

Exhibit A-2 Sensitivity Analysis Results for the 2010 SAMI "B3" Scenario

Mortality Lag	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	r = 3%	r = 5%	r = 7%
No Lag		-	6,155	-	$43,298	$43,298	$43,298
5 Year	25%, 25%, 17%, 17%, 16%	-	6,155	-	$41,163	$39,868	$38,664
8 Year	Incidence Occurs 8th Year	-	6,155	-	$35,205	$30,771	$26,964
15 Year	Incidence Occurs 15th Year	-	6,155	-	$28,625	$21,868	$16,792
15 Year	Incidence Skewed Early	-	6,155	-	$40,303	$38,640	$37,188
15 Year	Incidence Skewed Late	-	6,155	-	$31,052	$25,163	$20,577

Exhibit A-3 Sensitivity Analysis Results for the 2040 SAMI "B1" Scenario

Mortality Lag	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	r = 3%	r = 5%	r = 7%
No Lag		-	4,273	-	$35,054	$35,054	$35,054
5 Year	25%, 25%, 17%, 17%, 16%	-	4,273	-	$33,326	$32,277	$31,302
8 Year	Incidence Occurs 8th Year	-	4,273	-	$28,502	$24,912	$21,830
15 Year	Incidence Occurs 15th Year	-	4,273	-	$23,175	$17,705	$13,595
15 Year	Incidence Skewed Early	-	4,273	-	$32,630	$31,283	$30,107
15 Year	Incidence Skewed Late	-	4,273	-	$25,140	$20,372	$16,659

Exhibit A-4 Sensitivity Analysis Results for the 2040 SAMI "B3" Scenario

Mortality Lag	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 2000$)		
		5th %ile	Mean	95th %ile	r = 3%	r = 5%	r = 7%
No Lag		-	8,007	-	$65,685	$65,685	$65,685
5 Year	25%, 25%, 17%, 17%, 16%	-	8,007	-	$62,446	$60,481	$58,654
8 Year	Incidence Occurs 8th Year	-	8,007	-	$53,408	$46,681	$40,905
15 Year	Incidence Occurs 15th Year	-	8,007	-	$43,425	$33,175	$25,474
15 Year	Incidence Skewed Early	-	8,007	-	$61,141	$58,618	$56,415
15 Year	Incidence Skewed Late	-	8,007	-	$47,107	$38,172	$31,216

Exhibit A-5 Sensitivity Analysis: Effect of Thresholds on Estimated PM-Related Mortality Based on Krewski et al. (2000) - Mean, All-Cause for the 2010 SAMI "B1" Scenario

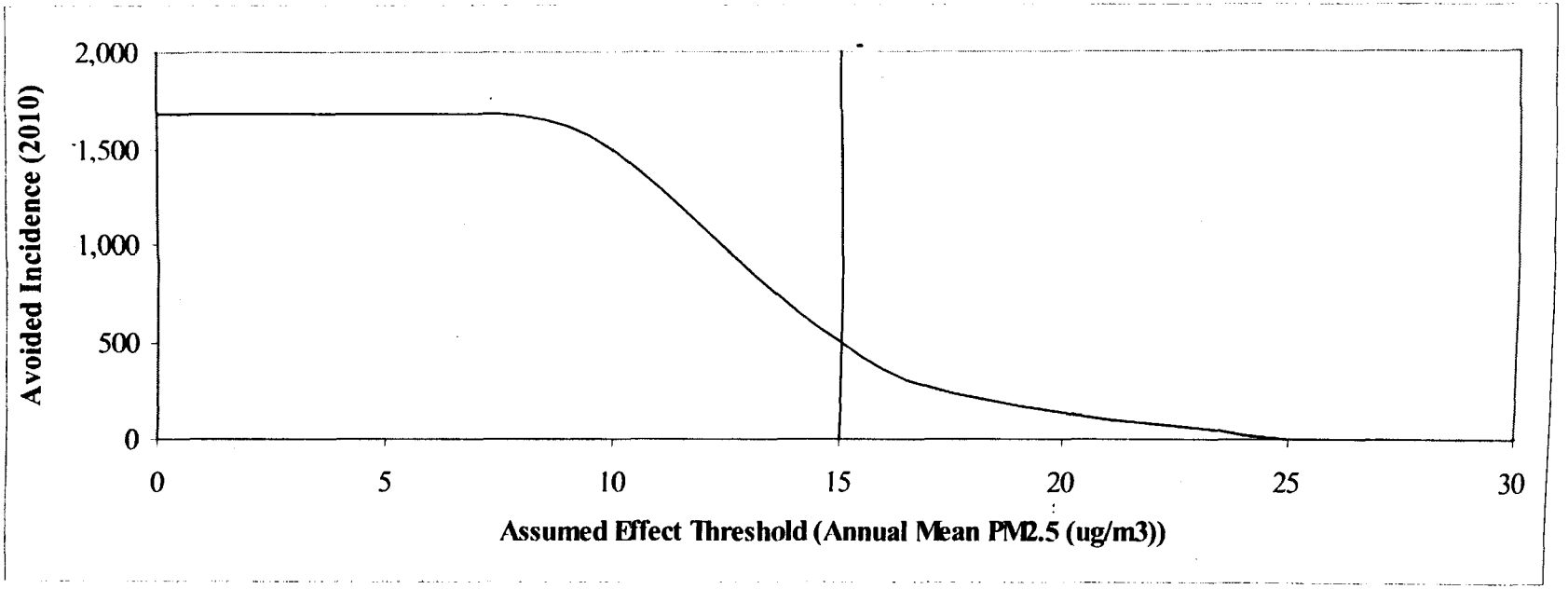


Exhibit A-6 Sensitivity Analysis: Effect of Thresholds on Estimated PM-Related Mortality Based on Krewski et al. (2000) - Mean, All-Cause for the 2010 SAMI "B3" Scenario

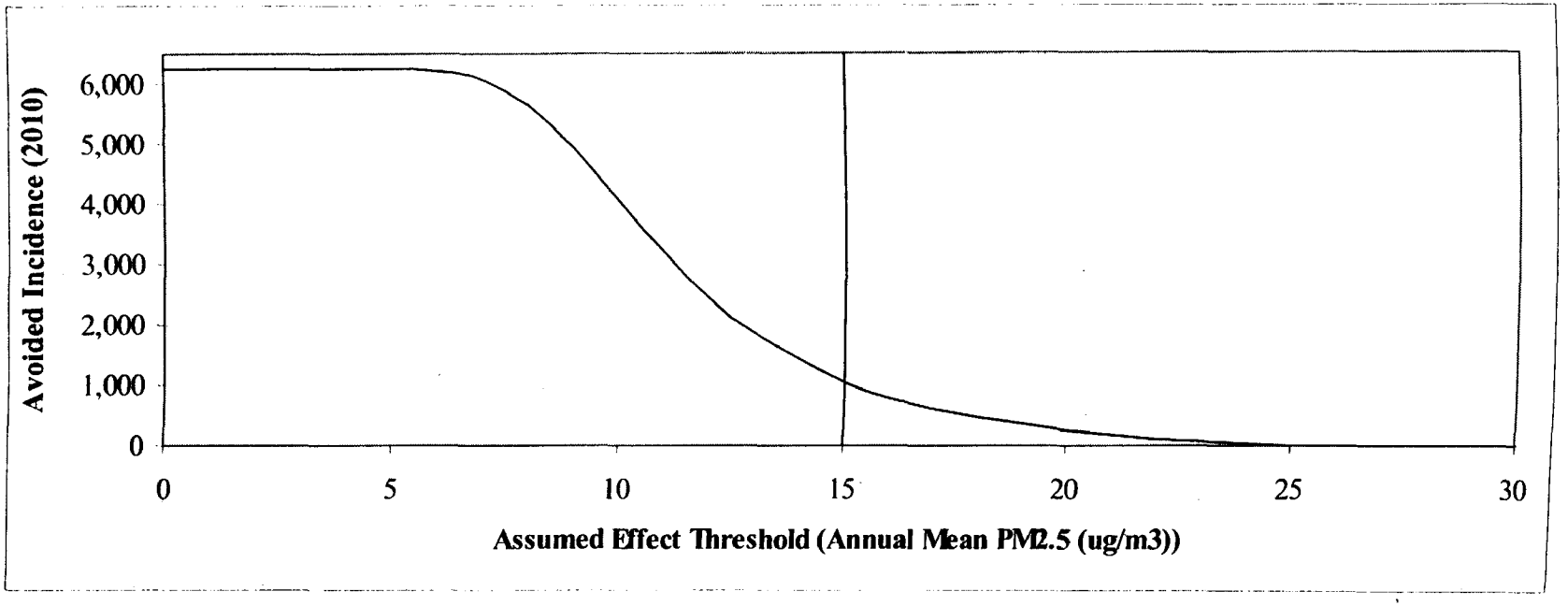


Exhibit A-7 Sensitivity Analysis: Effect of Thresholds on Estimated PM-Related Mortality Based on Krewski et al. (2000) - Mean, All-Cause for the 2040 SAMI "B1" Scenario

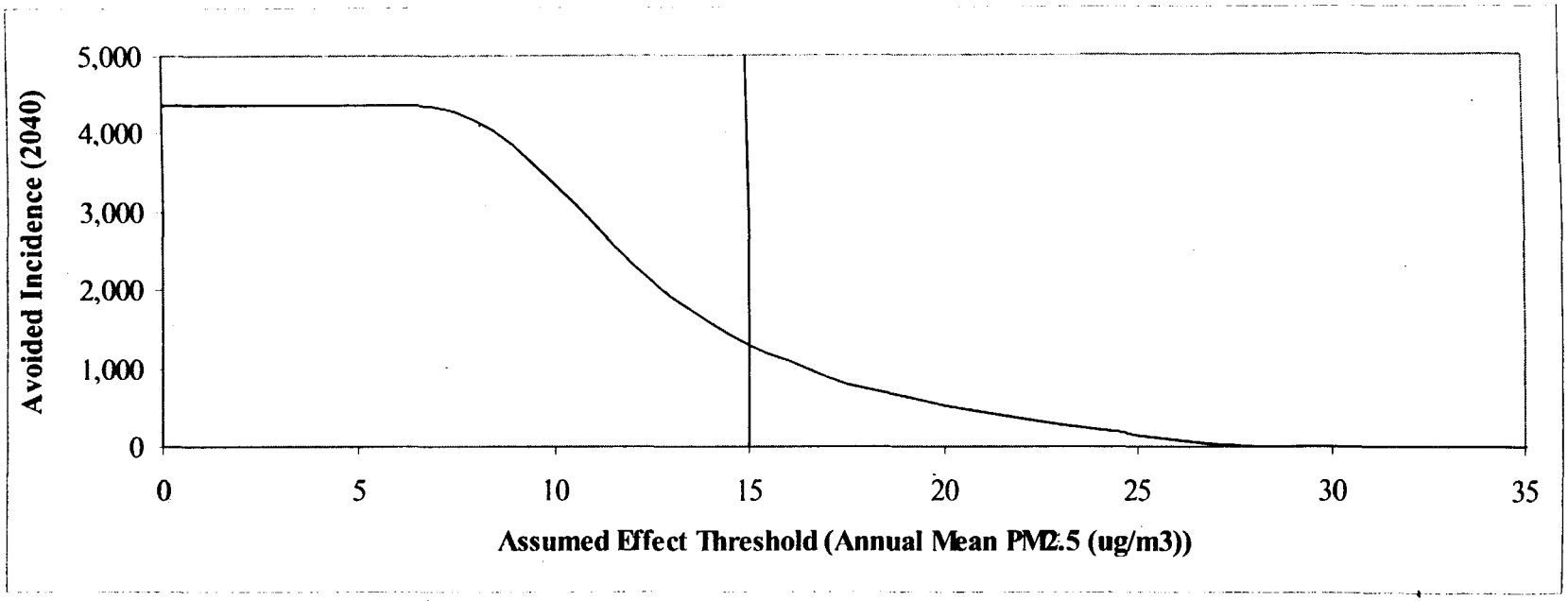
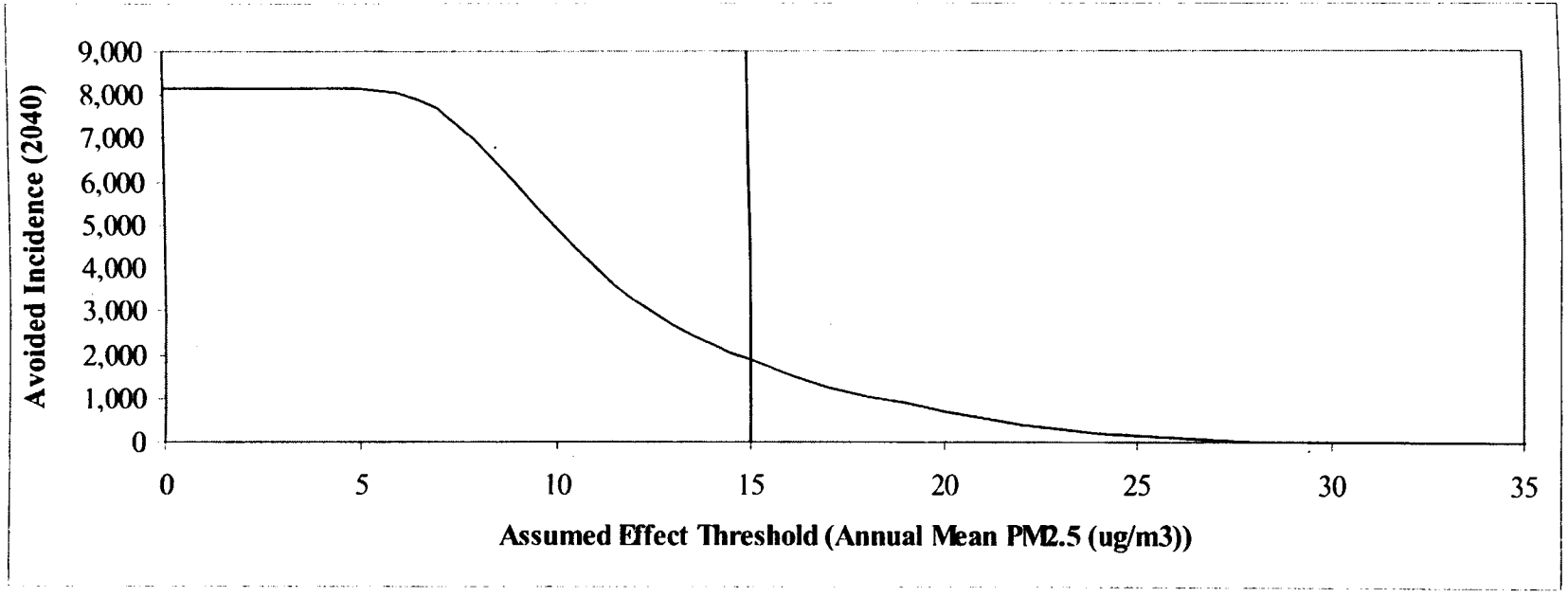


Exhibit A-8 Sensitivity Analysis: Effect of Thresholds on Estimated PM-Related Mortality Based on Krewski et al. (2000) - Mean, All-Cause for the 2040 SAMI "B3" Scenario



Appendix B: Particulate Matter C-R Functions

Note that ΔPM is defined -- for all of the concentration-response (C-R) functions -- as $PM_{baseline}$ - $PM_{control}$, and that the change is defined to be: - ($incidence_{control}$ - $incidence_{baseline}$).

B .1 Mortality

There are two types of exposure to PM that may result in premature mortality. Short-term exposure may result in excess mortality on the same day or within a few days of exposure. Long-term exposure over, say, a year or more, may result in mortality in excess of what it would be if PM levels were generally lower, although the excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels. In other words, long-term exposure may capture a facet of the association between PM and mortality that is not captured by short-term exposure.

B .1.1 Mortality (Krewski et al., 2000) Based on ACS Cohort: Mean $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.0046257
$\Delta PM_{2.5}$ = change in annual mean $PM_{2.5}$ concentration
pop = population of ages 30 and older
σ_β = standard error of β = 0.0012046

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual all-cause county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Note that the Krewski et al. (2000) replication of Pope et al. (1995) used the same all-cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.12) associated with a change in mean exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) (Krewski et al., 2000, Part II - Table 31).

$$\beta = \frac{\ln(1.12)}{(24.5)} = 0.0046257.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II - Table 31).

$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(1.19)}{24.5} - \frac{\ln(1.12)}{24.5}\right)}{1.96} = 0.0012625$$

$$\sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(1.12)}{24.5} - \frac{\ln(1.06)}{24.5}\right)}{1.96} = 0.0011466$$

$$\sigma_\beta = \frac{\sigma_{high} + \sigma_{low}}{2} = 0.0012046$$

B .1.2 Mortality (Krewski et al., 2000), Based on Six-City Cohort: Mean $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level all-cause annual death rate per person ages 25 and older
β = $PM_{2.5}$ coefficient = 0.013272
$\Delta PM_{2.5}$ = change in annual mean $PM_{2.5}$ concentration
pop = population of ages 25 and older
σ_β = standard error of β = 0.004070

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 25 and over, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). The Krewski et al. (2000) reanalysis of Dockery et al. (1993, p. 1754) appears to have used all-cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.28) associated with a change in mean exposure going from 11.0 $\mu g/m^3$ to 29.6 $\mu g/m^3$ (Krewski et al., 2000, Part I - Table 19c):

$$\beta = \frac{\ln(1.28)}{(29.6 - 11)} = 0.013272.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part I - Table 19c):

$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(1.48)}{18.6} - \frac{\ln(1.28)}{18.6}\right)}{1.96} = 0.003982$$

$$\sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(1.28)}{18.6} - \frac{\ln(1.10)}{18.6}\right)}{1.96} = 0.004157$$

$$\sigma_{\beta} = \frac{\sigma_{high} + \sigma_{low}}{2} = 0.004070$$

B .1.3 Mortality (Dockery et al., 1993), Based on Six-City Cohort: Mean $PM_{2.5}$

Dockery et al. (1993) examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. (1995). Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

The C-R function to estimate the change in long-term mortality is:

$$\Delta\ Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level all-cause annual death rate per person ages 25 and older
β = $PM_{2.5}$ coefficient = 0.0124
$\Delta PM_{2.5}$ = change in annual mean $PM_{2.5}$ concentration
pop = population of ages 25 and older
σ_β = standard error of β = 0.00423

Incidence Rate. Dockery et al. (1993, p. 1754) appear to have used all-cause mortality when estimating the impact of PM. To estimate county-specific baseline mortality incidence among individuals ages 25 and over, this analysis used the average all-cause annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.26) associated with a change in mean exposure going from 11.0 $\mu g/m^3$ to 29.6 $\mu g/m^3$ (Dockery et al., 1993, Tables 1 and 5):

$$\beta = \frac{\ln(1.26)}{(29.6 - 11)} = 0.0124.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Dockery et al., 1993, Table 5):

$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(1.47)}{18.6} - \frac{\ln(1.26)}{18.6}\right)}{1.96} = 0.00423$$

$$\sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(1.26)}{18.6} - \frac{\ln(1.08)}{18.6}\right)}{1.96} = 0.00423$$

$$\sigma_{\beta} = \frac{\sigma_{high} + \sigma_{low}}{2} = 0.00423.$$

B .2 Chronic Morbidity

Onset of bronchitis has been associated with exposure to air pollutants. Three studies have, in fact, linked the onset of chronic bronchitis in adults to particulate matter. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998).

In past analyses, we have estimated the changes in the number of new cases of PM-related chronic bronchitis using studies by Schwartz (1993) and Abbey et al. (1995b). The Schwartz study examined the relationship between exposure to PM_{10} and prevalence of chronic bronchitis. The Abbey et al. study examined the relationship between $PM_{2.5}$ and new incidences of chronic bronchitis. Both studies have strengths and weaknesses which suggest that, if both measures of PM were available, pooling the effect estimates from each study may provide a better estimate of the expected change in incidences of chronic bronchitis than using either study alone. However, since the SAMI analysis is based solely upon changes in annual mean PM2.5, we use the Abbey et al. study to predict changes in chronic bronchitis incidence.

B .2.1 Chronic Bronchitis (Abbey et al., 1995b, California)

Abbey et al. (1995b) examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for AOD or asthma; PM_{10} was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined.

The C-R function to estimate the change in chronic bronchitis is:

$$\Delta Chronic\ Bronchitis = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0132
$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration
pop = population of ages 27 and older without chronic bronchitis[17] = 0.9465*population 27+
σ_β = standard error of β = 0.00680

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (estimated to be 46.6% based on Abbey et al. (1995a, Table 1)). Using the same data base, Abbey et al. (1995a, Table 1) reported the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis, but they did not report an overall incidence rate for bronchitis.

Coefficient Estimate (β). The estimated coefficient (β) is based on the relative risk (= 1.81) associated with 45 $\mu g/m^3$ change in $PM_{2.5}$ (Abbey et al., 1995b, Table 2). The coefficient is calculated as follows:

$$\beta = \frac{\ln(1.81)}{45} = 0.0132.$$

Standard Error (σ_β). The standard error for the coefficient (σ_β) is calculated from the reported lower and upper bounds of the relative risk (0.98 to 3.25) (Abbey et al., 1995b, Table 2):

$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(3.25)}{45} - \frac{\ln(1.81)}{45}\right)}{1.96} = 0.00664$$

$$\sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(1.81)}{45} - \frac{\ln(0.98)}{45}\right)}{1.96} = 0.00696$$

$$\sigma_\beta = \frac{\sigma_{high} + \sigma_{low}}{2} = 0.00680.$$

B .3 Acute Morbidity

There is a considerable body of scientific research that has estimated significant relationships between elevated air pollution levels and other morbidity health effects. Chamber study research has

[17] Using the same data set, Abbey et al. (1995a, p. 140) reported that the respondents in 1977 ranged in age from 27 to 95. Chronic bronchitis prevalence from Adams and Marano (1995, Tables 62 and 78).

established relationships between specific air pollution chemicals and symptoms such as coughing, pain on deep inspiration, wheezing, eye irritation and headaches. In addition, epidemiological research has found air pollution relationships with acute infectious diseases (e.g., bronchitis, sinusitis) and a variety of "symptom-day" categories. Some "symptom-day" studies examine excess incidences of days with identified symptoms such as wheezing, coughing, or other specific upper or lower respiratory symptoms. Other studies estimate relationships for days with a more general description of days with adverse health impacts, such as "respiratory restricted activity days" or work loss days.

A challenge in preparing an analysis of the minor morbidity effects is identifying a set of effect estimates that reflects the full range of identified adverse health effects but avoids double counting. However, because only annual mean PM2.5 was available for the analysis, only one acute morbidity endpoint was available for evaluation, acute bronchitis.

B .3.1 Acute Bronchitis C-R Function (Dockery et al., 1996)

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.1}$ and PM_{10} were marginally significantly related to bronchitis.[18] They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes.

Earlier work, by Dockery et al. (1989), based on six U.S. cities, found acute bronchitis and chronic cough significantly related to PM_{15}. Because it is based on a larger sample, the Dockery et al. (1996) study is the better study to develop a C-R function linking $PM_{2.5}$ with bronchitis. The C-R function to estimate the change in acute bronchitis is:

$$\Delta Acute\ Bronchitis = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{2.5}\cdot\beta} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = annual bronchitis incidence rate per person = 0.044
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0272
$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration
pop = population of ages 8-12
σ_β = standard error of β = 0.0171

Incidence Rate. Bronchitis was counted in the study only if there were "reports of symptoms in the past 12 months" (Dockery et al., 1996, p. 501). It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. Dockery et al. found no relationship between PM

[18] The original study measured $PM_{2.1}$, however when using the study's results we use $PM_{2.5}$. This makes only a negligible difference, assuming that the adverse effects of $PM_{2.1}$ and $PM_{2.5}$ are comparable.

and chronic cough and chronic phlegm, which are important indicators of chronic bronchitis. For this analysis, we assumed that the C-R function based on Dockery et al. is measuring acute bronchitis.

In 1994, 2,115,000 children ages 5-17 experienced acute conditions (Adams and Marano, 1995, Table 6) out of population of 48.110 million children ages 5-17 (U.S. Bureau of the Census, 1998, Table 14), or 4.4 percent of this population. This figure is somewhat lower than the 5.34 percent of children under the age of 18 reported to have chronic bronchitis in 1990-1992 (Collins, 1997, Table 8). Dockery et al. (1996, p. 503) reported that in the 24 study cities the bronchitis rate varied from three to ten percent. Finally a weighted average of the incidence rates in the six cities in the Dockery et al. (1989) study is 6.34 percent , where the sample size from each city is used to weight the respective incidence rate (Dockery et al., 1989, Tables 1 and 4).[19] This analysis assumes a 4.4 percent prevalence rate is the most representative of the national population. Note that this measure reflects the fraction of children that have a chest ailment diagnosed as bronchitis in the past year, not the number of days that children are adversely affected by acute bronchitis.[20]

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.50) associated with being in the most polluted city ($PM_{2.1}$ = 20.7 μg/m^3) versus the least polluted city ($PM_{2.1}$ = 5.8 μg/m^3) (Dockery et al., 1996, Tables 1 and 4). The original study used $PM_{2.1}$, however, we use the $PM_{2.1}$ coefficient and apply it to $PM_{2.5}$ data.

$$\beta_{PM_{2.5}} = \frac{\ln(1.50)}{(20.7 - 5.8)} = 0.0272 .$$

Standard Error (σ_β). The standard error of the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (Dockery et al., 1996, Table 4):

$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(2.47)}{14.9} - \frac{\ln(1.50)}{14.9}\right)}{1.96} = 0.0171$$

$$\sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(1.50)}{14.9} - \frac{\ln(0.91)}{14.9}\right)}{1.96} = 0.0171$$

$$\sigma_\beta = \frac{\sigma_{\beta,high} + \sigma_{\beta,low}}{2} = 0.0171 .$$

[19]The unweighted average of the six city rates is 0.0647.

[20]In 1994, there were 13,707,000 restricted activity days associated with acute bronchitis, and 2,115,000 children (ages 5-17) experienced acute conditions (Adams and Marano, 1995, Tables 6 and 21). On average, then, each child with acute bronchitis suffered 6.48 days.