Preliminary Nonroad Landbased Diesel Engine Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results

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1 Introduction

This Technical Support Document details the models and methods used to estimate the benefits of the Preliminary Nonroad Landbased Diesel Engine Rule. For the preliminary control options analyzed here, it is assumed that implementation will occur in two stages. First there would be a reduction in sulfur content of nonroad diesel fuel, then followed by the gradual adoption of engine controls on new vehicles over several decades. Based on the projected time paths for emissions reductions, EPA chose to focus detailed emissions and air quality modeling on two future years, 2020 and 2030, which reflect partial and close to complete turnover of the fleet of nonroad land-based diesel engines to models meeting the preliminary control options.[1]

This analysis presents estimates of the benefits from the preliminary control options occurring in 2020 and 2030. We used methods generally consistent with the benefits analyses performed for the recent analysis of the Heavy Duty Engines/Diesel Fuel rulemaking (U.S. EPA, 2000c) and the proposed Clear Skies Act (U.S. EPA, 2002b). As described in the Regulatory Impact Assessment (U.S. EPA, 2003), the benefits analysis relies on three major modeling components:

➢Calculation of the impact that a set of preliminary fuel and engine standards would have on the nationwide inventories for NO_x, non-methane hydrocarbons (NMHC), SO2, and PM emissions in 2020 and 2030;

➢Air quality modeling for 2020 and 2030 to determine changes in ambient concentrations of ozone and particulate matter, reflecting baseline and post-control emissions inventories.

➢A benefits analysis to determine the changes in human health and welfare, both in terms of physical effects and monetary value, that result from the projected changes in ambient concentrations of various pollutants for the modeled standards.

This document focuses on the third component, detailing the steps we use to estimate health and welfare benefits, such as, reductions in premature mortality, visibility improvement, reduction in crop losses, and reduction in household soiling. The approaches to each of these benefits estimates can differ in detail, such as for visibility, as we describe later in this document, but the general approach is as follows:

➢We combined the results of sophisticated photochemical air quality models with air quality monitoring data, to estimate baseline and post-control ambient concentrations of ozone and PM.

➢We calculated the change between baseline and post-control estimates, and then combined this change with population forecasts, to give the change in population-level exposures due to the preliminary policy.

➢We used the changes in population exposure as an input to concentration-response functions, which allowed us to estimate the associated change in the incidence of health and welfare effects for each individual concentration-response (C-R) function.

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[1] The emissions changes modeled for these two years are similar to those in the preliminary standards, differing only in the treatment of smaller engines and the timing of reductions in the sulfur content of nonroad diesel fuel.

➢Where multiple C-R functions are available for an endpoint, we pooled the estimated change in incidence using fixed or random effects pooling methods.

➢We then assigned monetary values to the changes in health and welfare effects, taking into account adjustments to values for growth in real income out to the year of analysis. Finally, we summed these monetary values to obtain an estimate of the total monetary value of the changes in emissions.

Exhibit 1-1 summarizes the results of the Base Estimate, and an Alternative Estimate of the benefits of the control options of the Nonroad Landbased Diesel Engine Rule. As described in more detail in this analysis, the difference between the two is that the Alternative Estimate quantifies changes in premature mortality using time series studies in place of long-term cohort studies, and uses different valuation methods for mortality and chronic bronchitis risk reductions.

In addition to these quantified and monetized benefits, there are a number of additional categories that are not currently amenable to quantification or valuation (Exhibit 1-2). These include reduced effects on forested ecosystems, yields for fruits, vegetables, and other commercial and non-commercial crops, and reduced premature aging of the lungs. Additionally, we have not quantified a number of known or suspected health effects linked with PM and ozone for which we cannot as yet quantify, or easily value (e.g., decreased lung functioning). As a result, both the Base and Alternative monetized benefits estimates incorporate an underestimation bias in the total benefits attributable to the preliminary control options.

Exhibit 1-1. Summary of Estimated Benefits of the Modeled Preliminary Control Option

Estimation Method [a]	Total Benefits [b] (Billions 2000$)	
	2020	*2030*
Base Estimate [c]		
Using a 3% discount rate	$52,000	$92,000
Using a 7% discount rate	$49,000	$87,000
Alternative Estimate [d]		
Using a 3% discount rate	$11,000	$19,000
Using a 7% discount rate	$11,000	$20,000

[a] Results reflect the use of two different discount rates; a 3% rate which is recommended by EPA's Guidelines for Preparing Economic Analyses (US EPA, 2000a), and 7% which is recommended by OMB Circular A-94 (OMB, 1992).

[b] Benefits of carbon monoxide and hazardous air pollution emission reductions are not quantified in this analysis and, therefore, are not presented in this table. The quantifiable benefits are from emission reductions of NOx, NMHC, SO_2 and PM only. Exhibit 1-2 presents a detailed listing of unquantified health and welfare effects.

[c] Base Estimate reflects premature mortality based on application of concentration-response function derived from long-term exposure to $PM_{2.5}$, valuation using the value of statistical lives saved approach, and a willingness-to-pay approach for valuing chronic bronchitis incidence.

[d] Alternative Estimate reflects premature mortality based on application of concentration-response function derived from short-term exposures to $PM_{2.5}$, valuation using the value of statistical live years saved approach, assumption of 0.5 life years saved for each COPD related premature mortality avoided and 5 years for all other causes of death, and a cost-of-illness approach for valuing chronic bronchitis incidence.

Chapter 2 describes the methods used to estimate population-level exposures to changes in ambient ozone and particulate matter (PM) concentrations and changes in visibility, based on modeled and monitored air quality inputs. Chapter 3 describes general issues arising in estimating and valuing changes in adverse health and welfare effects associated with changes in ozone, PM, and visibility. Chapter 4 describes in some detail the methods used for estimating and valuing adverse health effects, while Chapter 5 describes the methods used for welfare effects: crop damage, visibility, and household soiling. The results of these analyses follow in Chapter 6.

This document has eight appendices. Appendix A presents the physical and monetary benefits associated with the sensitivity calculations for the Preliminary Nonroad Landbased Diesel Engine Rule. Appendix B details the forecasting of population levels. Appendix C provides statistical summaries of the forecasted air quality exposure, and maps of the underlying monitor data, modeling data, and resulting 2020 and 2030 population-level ambient exposure estimates. Appendix D derives the general form of the concentration-functions (C-R) that we use to estimate the change in adverse health effects. Appendix E details the calculation of incidence and prevalence rates for use in the C-R functions. Appendix F presents the particulate matter C-R functions used in this analysis, and Appendix G presents the ozone C-R functions. Finally, Appendix H presents a discussion of the pooling approaches used in this analysis.

Exhibit 1-2. Human Health and Welfare Effects of Pollutants Affected by the Preliminary Nonroad Landbased Diesel Engine Rule

Pollutant/Effect	Quantified and Monetized in Base and Alternative Estimates	Quantified and/or Monetized Effects in Sensitivity Analyses [a]	Unquantified Effects
Ozone/Health	Hospital admissions - respiratory Emergency room visits for asthma Minor restricted activity days School loss days	Chronic Asthma [b] Asthma attacks Cardiovascular emergency room visits Premature mortality – acute exposures [b] Acute respiratory symptoms	Increased airway responsiveness to stimuli Inflammation in the lung Chronic respiratory damage Premature aging of the lungs Acute inflammation and respiratory cell damage Increased susceptibility to respiratory infection Non-asthma respiratory emergency room visits
Ozone/Welfare	Decreased outdoor worker productivity Decreased yields for commercial crops (selected species) Decreased Eastern commercial forest productivity (selected species)		Decreased commercial forest productivity Decreased yields for fruits, vegetables, and other commercial and non-commercial crops Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Damage to ecosystem functions
PM/Health	Premature mortality Bronchitis - chronic and acute Hospital admissions - respiratory and cardiovascular Emergency room visits for asthma Non-fatal heart attacks Lower and upper respiratory illness Minor restricted activity days Work loss days	Premature mortality – short term exposures Asthma attacks (asthmatic population) Respiratory symptoms (asthmatic population) Infant mortality	Low birth weight Changes in pulmonary function Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Cancer Non-asthma respiratory emergency room visits
PM/Welfare	Visibility in California, Southwestern, and Southeastern Class I areas	Visibility in Northeastern, Northwestern, and Midwestern Class I areas Visibility in residential and non-Class I areas Household soiling	

[a] Note that while no causal mechanism has been identified linking new incidences of chronic asthma to ozone exposure, two epidemiological studies shows a statistical association between long-term exposure to ozone and incidences of chronic asthma in exercising children and some non-smoking men (McDonnell et al., 1999; McConnell et al., 2002). Premature mortality associated with ozone is not separately included in the primary analysis. It is assumed that the American Cancer Society (ACS)/ Krewski, et al. (2000) C-R function captures both PM mortality benefits and any mortality benefits due to other air pollutants.

2 Forecasting Changes in Population-Level Exposure to Ambient Ozone And Particulate Matter for Use in the Benefits Analysis

We estimated population exposure using the Criteria Air Pollutant Modeling System (CAPMS), a population-based geographic information system for modeling changes in population exposure and estimating the associated health and welfare benefits. To forecast population-level exposure to ambient ozone and particulate matter, CAPMS uses both air quality modeling and monitoring data files, as well as demographic data from the Census and population projections.

This chapter describes how we use these different data sources to forecast changes in ambient levels of ozone and particulate matter (PM) for use in the benefits analysis. We used two basic approaches. Most commonly, we used air quality modeling combined with actual monitoring values to estimate the exposure of people and crops to air pollution. This has the advantage of linking the modeling with actual observations. We also used modeling results directly, in the case of estimating visibility, because we do not have readily available monitoring data for visibility. Estimating population exposure is somewhat more complicated, so we have focused this chapter on how we combine population forecasts with modeling results and monitoring data.

CAPMS has grouped individuals into what we refer to as "population grid-cells," where the grid-cells typically conform to some type of grid structure used in an air quality model. For each type of grid, we calculate the population in each grid-cell by aggregating census block data. In the next step, CAPMS estimates the air pollution exposure for each grid-cell, with the assumption that people living within a particular grid-cell experience the same ambient air pollution levels.

In the first section of this chapter, we provide an overview of how we estimate the population exposure in each grid-cell. We discuss how we forecast population levels in each grid-cell, and how we forecast air pollution levels, by combining air quality modeling results and air quality monitoring data with an algorithm called Voronoi Neighbor Averaging (VNA). In the second and third sections, we detail the exposure calculations for ozone and PM, and provide tables summarizing the estimated changes in exposure for each.

2.1 Forecasting Population-Level Exposure to Air Pollution with CAPMS

To forecast future population-level exposure to air pollution, CAPMS uses projections of future air quality and population levels. These are estimates of what air quality and population modelers predict will occur in the future. In addition, CAPMS uses predictions from these same models to "predict" what air quality and population levels should be currently. Finally, CAPMS uses the actual measured current air pollution and population levels from the EPA air monitoring network and the U.S. Census, respectively. In this section, we discuss how CAPMS uses the two sets of *model* values (future-year and current-year) in combination with the actual current-year measurements.

CAPMS has a variety of approaches to estimate the exposure to air pollution for the people living within a given population grid-cell. Perhaps the simplest approach is to use modeled air quality data directly, and to assume that the people living within a particular model grid-cell experience the level estimated by the model. An alternative approach is to use air pollution monitoring data, where the analyst may choose the closest monitor data to the center of a grid-cell, or take an average of data from nearby

monitors. In a third general approach, the user may combine both modeling and monitoring data to estimate exposure. We use this latter approach in the current analysis.

When combining modeling and monitoring data, CAPMS scales or adjusts the monitoring data with modeling data. The advantage of modeling data is that they can provide predictions for years in which monitoring data are not available, as well as to provide predictions in areas of the country for which monitoring data are not available.

The goal of estimating exposure is to provide the necessary input for concentration-response functions, so that CAPMS can estimate the impact of air pollution on adverse health effects, as well as various welfare effects, such as crop losses and household soiling. Exhibit 2-1 lists the types of metrics commonly used in concentration-response functions.

Exhibit 2-1. Metrics Typically Used in Concentration-Response Functions for Criteria Air Pollutants

Measurement Frequency	Metric Name	Metric Description
Daily (e.g., $PM_{2.5}$, PM_{10})	Daily Average	Daily average
	Annual Average	Average of four quarterly averages. The four quarters are defined as: Jan-Mar, April-June, Jul-Sep, Oct-Dec.
	Annual Median	Median of values through out the year.
Hourly (e.g., Ozone)	1-hour Daily Max	Highest hourly value from 12:00 A.M. through 11:59 P.M.
	5-hour Daily Average	Average of hourly values from 10:00 A.M. through 2:59 P.M.
	8-hour Daily Average	Average of hourly values from 9:00 A.M. through 4:59 P.M. [a]
	12-hour Daily Average	Average of hourly values from 8:00 A.M. through 7:59 P.M.
	24-hour Daily Average	Average of hours from 12:00 A.M. through 11:59 P.M.
	SUM06	SUM06 index is the sum of the ozone concentrations (measured in ppm) that exceed 0.06 ppm between 8:00 am and 7:59 pm.

[a] Note that the 8-hour daily average differs from the maximum 8-hour *moving average* described in the Federal Register (6 FR / Vol. 62, No. 138 / Friday, July 18, 1997 / Prepublication).

2.1.1 Woods and Poole County-Level Population Forecasts

In the current analysis, we use more sophisticated projections based on economic forecasting models developed by Woods and Poole (2001). The Woods and Poole database contains county level projections of population by age, sex, and race out to 2025. Projections in each county are determined simultaneously with every other county in the U.S. to take into account patterns of economic growth and migration. The sum of growth in county level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates (Hollman et al., 2000). According to Woods and Poole, linking county level growth projections together and constraining to a national level total growth avoids potential errors introduced by forecasting each county independently.

Woods and Poole (2001) developed county projections in a four stage process. First, national level variables such as income, employment, populations, etc. are forecasted. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an

"export-base" approach, which relies on linking industrial sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-base approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector. Third, population is projected for each economic area based on net migration rates derived from employment opportunities, and following a cohort-component method based on fertility and mortality in each area. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2025 based on historical rates of mortality, fertility, and migration.

The Woods and Poole (2001) projections of county level population are based on historical population data from 1969-1999, and do not include the 2000 Census results. Given the availability of detailed 2000 Census data, we constructed adjusted county level population projections for each future year using a two stage process. First, we constructed ratios of the projected Woods and Poole populations in a future year to the projected Woods and Poole population in 2000 for each future year by age, sex, and race. Second, we multiplied the block level 2000 Census population data (GeoLytics Inc., 2002a) by the appropriate age, sex, and race specific Woods and Poole ratio for the county containing the census block, for each future year. This results in a set of future population projections that is consistent with the most recent detailed census data. The total U.S. population is projected to increase from 281 million in 2000 to 345 million in 2025, while the percent of the population 18 and under is expected to decrease slightly, from 27 to 25 percent, and the percent of the population over 65 is expected to increase from 12 percent to 18 percent.

Since the Woods and Poole (2001) projections only extend through 2025, we used the existing projections and constant growth factors to provide additional projections. To calculate populations for 2030, we applied the growth rate from 2024 to 2025 to each year between 2025 and 2030. This is described in more detail below.

2.1.2 Population Projections for REMSAD and CAMx Population Grid-Cells

The benefits for this analysis are based on predicted air quality changes for the years 2020 and 2030, so population projections are needed for these years. The underlying data used to create these 2020 and 2030 population projections are based on: (1) 2000 block data from the U.S. Census (GeoLytics Inc., 2002a), and (2) county-level population forecasts provided by Woods and Poole (2001).

First, we aggregate the 2000 block data to the level of the air quality modeling data, in order to have these two key sources of information at the same level of aggregation. The 2000 block data are the most disaggregated data provided by the Census Bureau, and typically cover a much smaller geographic area than the air quality models. In the current analysis, we use the Regulatory Model System for Aerosols and Deposition (REMSAD) and Comprehensive Air Quality Model with Extensions (CAMx) models, which have an average grid size of about 36 by 36 kilometers and 12 by 12 kilometers, respectively. After the aggregation, we have two sets of population grid-cells with year 2000 population data, one matching REMSAD and the other matching CAMx.

Second, we scale population grid-cells with the ratio of the matching county-level populations for the future year of interest and the base-year 2000. In the simplest case, where one is forecasting a single population variable, say, children ages 4 to 9 in the year 2020, CAPMS calculates:

$$age_{4-9,\,g,\,2020} = age_{4-9,\,g,\,2000} \cdot \frac{age_{4-9,\,county,\,2020}}{age_{4-9,\,county,\,2000}}$$

where the gth population grid-cell is wholly located within a given county.

In the case, where the gth grid-cell includes more than one county in its boundary, the situation is somewhat more complicated. CAPMS first estimates the fraction of individuals in a given age group (e.g., ages 4 to 9) that reside in the part of each county within the gth grid-cell. CAPMS calculates this fraction by simply dividing the all-age population of a given county within the gth grid-cell by the total population in the gth grid-cell:

$$fraction\ of\ age_{4-9,\,g\ in\ county_c} = \frac{age_{all,\,g\ in\ county_c}}{age_{all,\,g}}$$

We multiply this fraction with the number of individuals ages 4 to 9 in the year 2000, to give an estimate of the number of individuals ages 4 to 9 that reside in the fraction of the county within the gth grid-cell in the year 2000:

$$age_{4-9,\,g\ in\ county_c,\,2000} = age_{4-9,\,g,\,2000} \cdot fraction\ age_{4-9,\,g\ in\ county_c}$$

To then forecast the population in 2020, we scale the 2000 estimate with the ratio of the county projection for 2020 to the county projection for 2000:

$$age_{4-9,\,g\ in\ county_c,\,2020} = age_{4-9,\,g\ in\ county_c,\,2000} \cdot \frac{age_{4-9,\,county_c,\,2020}}{age_{4-9,\,county_c,\,2000}} \quad .$$

Combining all these steps for "c" counties within the gth grid-cell, we forecast the population of persons ages 4 to 9 in the year 2020 as follows:

$$age_{4-9,\,g,\,2020} = \sum_{c=1}^{n} age_{4-9,\,g,\,2000} \cdot \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \cdot \frac{age_{4-9,\,county_c,\,2020}}{age_{4-9,\,county_c,\,2000}}$$

In the case where there are multiple age groups and multiple counties, CAPMS first calculates the forecasted population level for individual age groups, and then combines the forecasted age groups. In calculating the number of children ages 4 to 12, CAPMS calculates:

$$age_{4-9,\,g,\,2020} = \sum_{c=1}^{n} age_{4-9,\,g,\,2000} \cdot \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \cdot \frac{age_{4-9,\,county_c,\,2020}}{age_{4-9,\,county_c,\,2000}}$$

$$age_{10-14,\,g,\,2020} = \sum_{c=1}^{n} age_{10-14,\,g,\,2000} \cdot \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \cdot \frac{age_{10-14,\,county_c,\,2020}}{age_{10-14,\,county_c,\,2000}}$$

$$age_{4-12,\,g,\,2020} = age_{4-9,\,g,\,2020} + \frac{3}{5} \cdot age_{10-14,\,g,\,2020} \quad .$$

The Woods and Poole county-level forecasts extend to 2025. To estimate population levels in 2030, CAPMS linearly extrapolates from the final two years of data. For example, to forecast population in 2030, CAPMS calculates:

$$age_{4-9,\,2030} = age_{4-9,\,2025} + 5 \cdot \left(age_{4-9,\,2025} - age_{4-9,\,2024} \right) \ .$$

Appendix B provides more details on the underlying data and the steps used to calculate population forecasts.

2.1.3 Voronoi Neighbor Averaging (VNA)

Given a population estimate at each grid-cell, we then need to estimate the change in population exposure to PM and ozone. The approach we use to estimate exposure is based on the Voronoi Neighbor Averaging (VNA) algorithm, and a scaling of air quality monitor data with air quality modeling data.

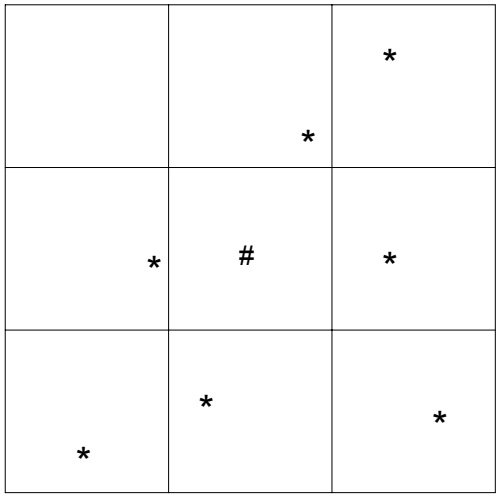
The VNA algorithm works at every population grid-cell. It first identifies the set of air quality monitors that best "surround" the center of the population grid-cell, and given the best set of monitors, it scales the monitor data with modeling data, to generate air quality forecasts. Thus, rather than use modeling data directly or "absolutely," it uses modeling data in a relative sense to scale actual monitor levels. This has the advantage of linking model results with observed air pollution levels.

We use this approach to estimate population exposure to both ozone and $PM_{2.5}$. Note that in the case of air pollution metrics calculated on a daily basis, such as the one-hour maximum and the 24-hour average, it is often the case that there are missing days of data, because air quality monitors often miss a number of observations through out the year. To account for these missing days, CAPMS represents the distribution of air quality levels with a discrete number of points or "bins," where each bin represents a certain range of the distribution, with the underlying assumption that missing days have the same distribution as the available data. The number of bins equals the number of days analyzed, so that for ozone we use 153 bins (representing the summer ozone season), and for PM we use 365 bins. However, note that for annual metrics, such as the annual average, there is only a single value, so there is no need for bins.

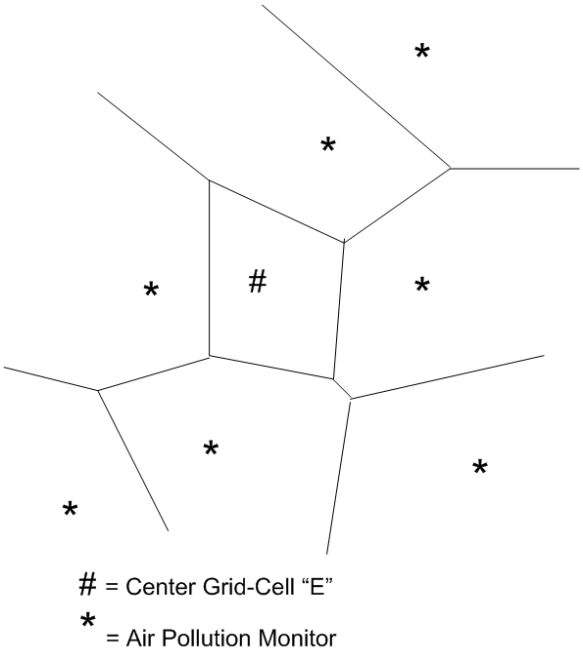
As described below, there are three different ways to scale the monitoring data – (1) temporal scaling, (2) spatial scaling, and (3) temporal and spatial scaling. For the present analysis, we used VNA with both temporal and spatial scaling. To explain our approach, we start with an example describing VNA, and then we turn to each of the scaling approaches.

Numerical Example of VNA

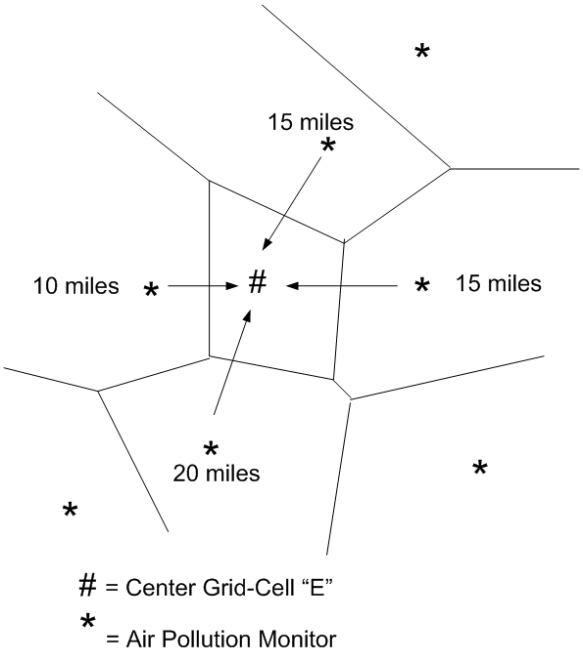
The first step in VNA is to identify the set of nearest monitors for each of the population grid cells in the modeling domain. The figure below presents nine population grid-cells and three monitors, with the focus on identifying the set of nearest neighbors to grid-cell "E."



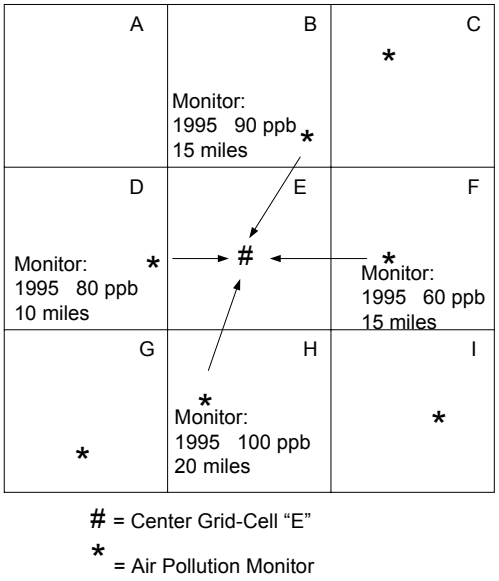
In particular, CAPMS identifies the nearest monitors, or "neighbors," by drawing a polygon, or "Voronoi" cell, around the center of each CAPMS grid cell. The polygons have the special property that the boundaries are the same distance from the two closest points.



We then choose those monitors that share a boundary with the center of grid-cell "E." These are the nearest neighbors, we use these monitors to estimate the air pollution level for this grid-cell.



To estimate the air pollution level in each grid-cell, CAPMS calculates the annual and the binned daily metrics for each of the neighboring monitors, and then calculates an inverse-distance weighted average of the metrics. The further the monitor is from the CAPMS grid-cell, the smaller the weight.



The weight for the monitor 10 miles from the center of grid-cell E is calculated as follows:

$$d_{i,1} = \frac{\frac{1}{10}}{\left(\frac{1}{10}+\frac{1}{15}+\frac{1}{15}+\frac{1}{20}\right)} = 0.35 \ .$$

The weights for the other monitors would be calculated in a similar fashion. CAPMS would then calculate an inverse-distance weighted average for 1995 air pollution levels in grid-cell E as follows:

Forecast $_{1995}$ = 0.35*80 ppb + 0.24*90 ppb+ 0.24*60 ppb + 0.18*100 ppb = 81.2 ppb .

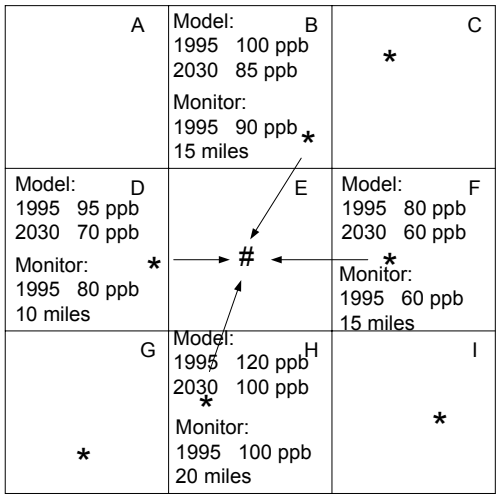
Numerical Example of VNA with Temporal Scaling

In combining VNA with temporal scaling, CAPMS temporally scales all of the neighboring monitors, calculates the metrics of interest, and then calculates an inverse distance-weighted average of the metrics. Consider the example in the figure below.

To forecast air pollution levels for 2030, CAPMS would multiply the 1995 monitor value by the ratio of the 2030 model value to the 1995 model value:

$$Forecast_{2030} = \sum_{i=1}^{4} Weight_i * Monitor_i * \frac{Model_{i,\,2030}}{Model_{i,\,1995}}$$

$$Forecast_{2030} = \left(0.35*80*\frac{70}{95}\right) + \left(0.24*90*\frac{100}{85}\right) + \left(0.24*60*\frac{80}{60}\right) + \left(0.18*100*\frac{120}{100}\right) = 64.1ppb$$



= Center Grid-Cell "E"

* = Air Pollution Monitor

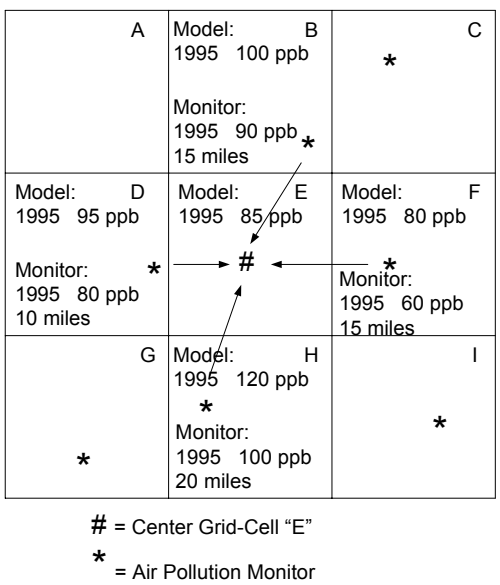
Numerical Example of VNA with Spatial Scaling

CAPMS can also combine VNA with spatial scaling. For each of the neighbor monitors, CAPMS multiplies the monitoring data with the ratio of the base-year modeling data for the destination grid-cell to the base-year modeling data for grid-cell containing the monitor. The spatial scaling occurs in the same fashion as with temporal scaling. In the case of pollutants typically measured hourly, such as ozone, CAPMS scales the hourly monitor values, calculates the annual and daily metrics of interest, bins the daily metrics, and then calculates an inverse-distance weighted average of the metrics. In the case of pollutants typically measured daily, CAPMS scales the daily values, calculates the annual metrics of interest, bins the daily metric, and then calculates an inverse-distance weighted average of the metrics.

Consider the example in the figure below. To forecast air pollution levels for 1995, CAPMS would multiply the 1995 monitor value by the ratio of the 1995 model value to the 1995 model value:

$$Forecast_{1995} = \sum_{i=1}^{4} Weight_i * Monitor_i * \frac{Model_{E,1995}}{Model_{i,1995}}$$

$$Forecast_{1995} = \left(0.35*80*\frac{85}{95}\right) + \left(0.24*90*\frac{85}{100}\right) + \left(0.24*60*\frac{85}{80}\right) + \left(0.18*100*\frac{85}{120}\right) = 70.8\,ppb$$



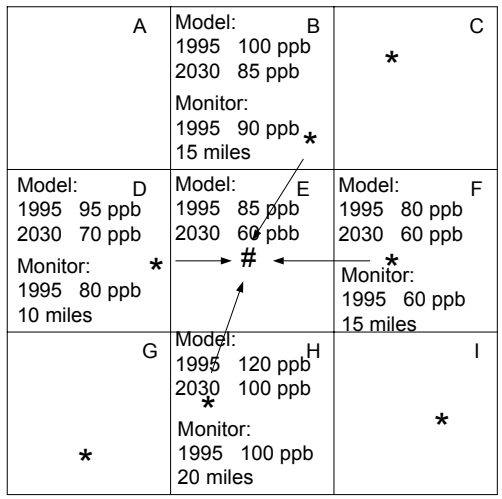
Numerical Example of VNA with Temporal & Spatial Scaling

Finally, as done in the present analysis, CAPMS can combine temporal and spatial scaling. In this approach, CAPMS multiplies monitoring data with the ratio of the future-year to base-year modeling data, where the future-year modeling data are from the destination grid-cell and the base-year modeling data are from the grid-cell containing the monitor. Once the hourly and daily monitoring data are scaled, CAPMS generates the metrics of interest, bins the daily metrics, and then calculates an inverse-distance weighted average of the metrics.

The figure below gives an example of combining temporal and spatial scaling.

$$Forecast_{2030} = \sum_{i=1}^{4} Weight_i * Monitor_i * \frac{Model_{E,2030}}{Model_{i,1995}}$$

$$Forecast_{2030} = \left(0.35*80*\frac{60}{95}\right) + \left(0.24*90*\frac{60}{100}\right) + \left(0.24*60*\frac{60}{80}\right) + \left(0.18*100*\frac{60}{120}\right) = 50.0$$



= Center Grid-Cell "E"

* = Air Pollution Monitor

2.2 Ozone Exposure Forecasts

To develop baseline and control forecasts for ozone, we used spatial and temporal VNA, combining air quality modeling data from the Comprehensive Air Quality Model with Extensions (CAMx) and air quality monitoring data from a nationwide network of monitors.

2.2.1 Ozone Model Data

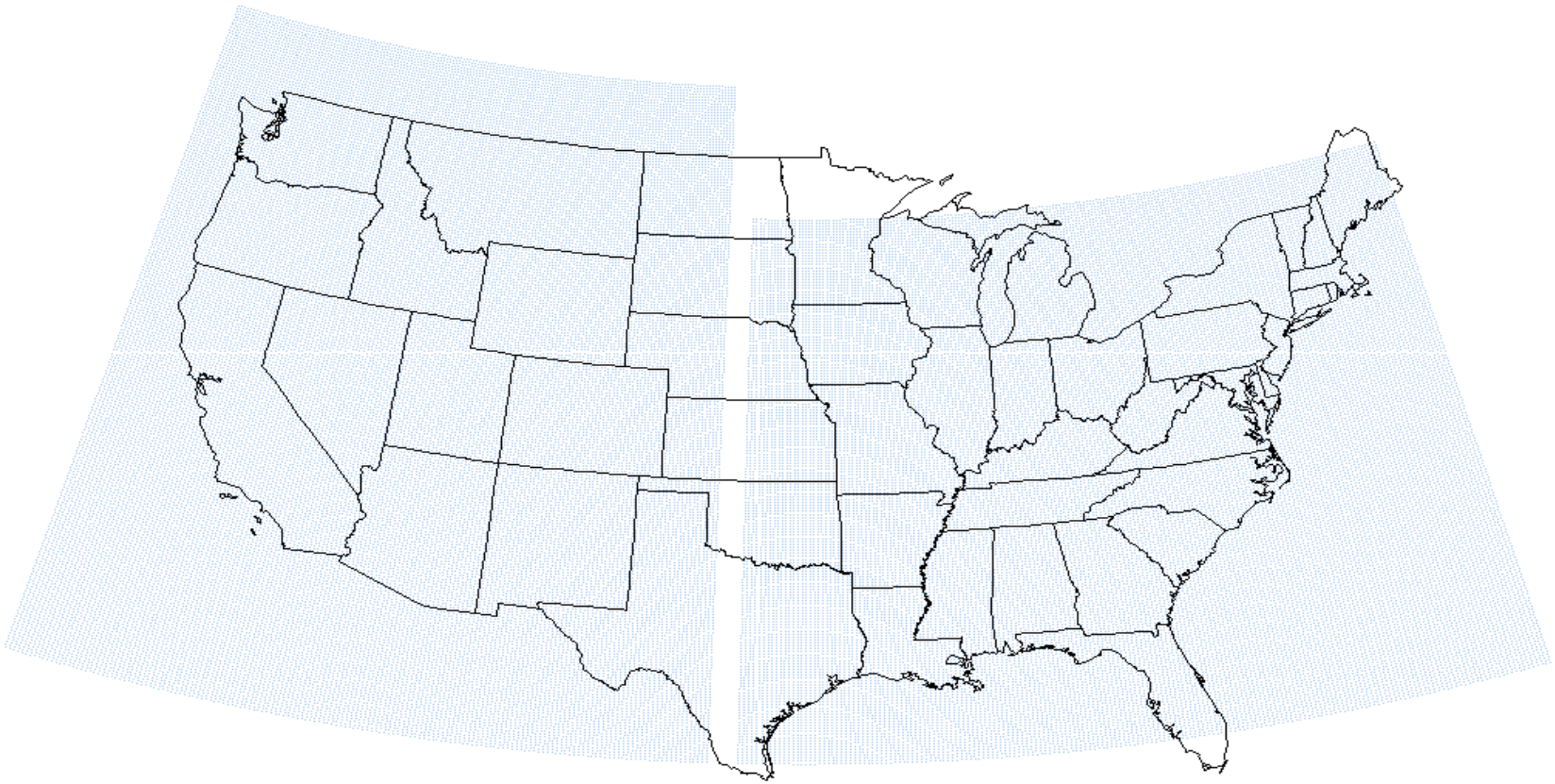
To estimate ozone air quality in the Eastern and Western U.S., this analysis relies on a regional-scale version of CAMx, which has a relatively fine grid-cell structure of about 12 kilometers by 12 kilometers. CAMx is a numerical multi-scale three-dimensional grid photochemical air quality model, designed to calculate the concentrations of both inert and chemically reactive pollutants by simulating the physical and chemical processes in the atmosphere that affect ozone formation. Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, the CAMx is useful for evaluating the impacts of reductions in emissions of NO_x and NMHC from nonroad diesel engines on U.S. ozone concentrations.

The base-year for the modeling is determined by the available historical meteorological data – 1995 for the Eastern U.S., and 1996 for the Western U.S.[2] And the future-year is determined by the years of interest for this analysis – 2020 and 2030. Taking the ratio of future year to base year modeling values, we create adjustment factors.

The Eastern modeling domain is bounded by longitude -99° to -67° and latitude 26° to 47°, and the Western modeling domain is bounded by -127° to -99° and latitude 26° to 52°. This covers most of the United States (see Exhibit 2-2). Each grid cell in the two modeling domains is 1/6 of a degree longitude wide and 1/9 of a degree latitude high. At the edge of each of the modeling domains, there is a border of three grid cells with missing data, so some populated areas of the U.S. are not modeled. The actual modeled area in the Eastern domain extends from longitude -98.5° to -67.5° and latitude 26.33° to 46.67°, and the Western domain extends from longitude -126.5° to -99.5° and latitude 26.33° to 51.67°.

[2] The three simulation periods for the eastern U.S. are based on meteorology for June 15-24, July 8-15, and August 10-21, 1995, and are based on an emission inventory for 1996. The three simulation periods for the Western U.S. are based on meteorology for July 8-15 and July 21-31, 1996, and are based on an emission inventory for 1996.

Exhibit 2-2. Eastern and Western Modeling Domains for Ozone Exposure Forecasts



For populated areas outside of the modeling domain, we assume that ozone levels in the control scenario are identical with those in the baseline scenario. As a result, we assume that people outside of the modeling domain will not be affected by the preliminary regulation. Exhibit 2-3 summarizes the number of people outside the modeling domain.

Exhibit 2-3. Summary by State of the Number of People Outside the Eastern and Western CAMx Modeling Domains

State	2020		2030	
	# of people [a]	*% of total*	*# of people*	*% of total*
Florida	5,323,490	25.3%	5,900,976	25.0%
Kansas	112,283	3.7%	110,876	3.4%
Louisiana	67,681	4.8%	67,104	4.5%
Michigan	54,039	0.5%	55,096	0.5%
Minnesota	616,073	10.5%	642,214	10.1%
Nebraska	109,463	5.6%	112,767	5.4%
North Dakota	340,889	47.7%	363,403	48.2%
Oklahoma	152,057	3.9%	153,626	3.7%
South Dakota	43,578	5.0%	43,185	4.7%
Texas	3,203,407	11.9%	3,615,301	12.0%
Wisconsin	36,454	0.6%	37,955	0.6%
Continental U.S.	10,059,414	3.1%	11,102,503	3.1%

[a] The column "# of people" is a count of the number of people outside the CAMx modeling domain. The column "% of total" is the percentage of the total population in a given state that is outside the CAMx modeling domain.

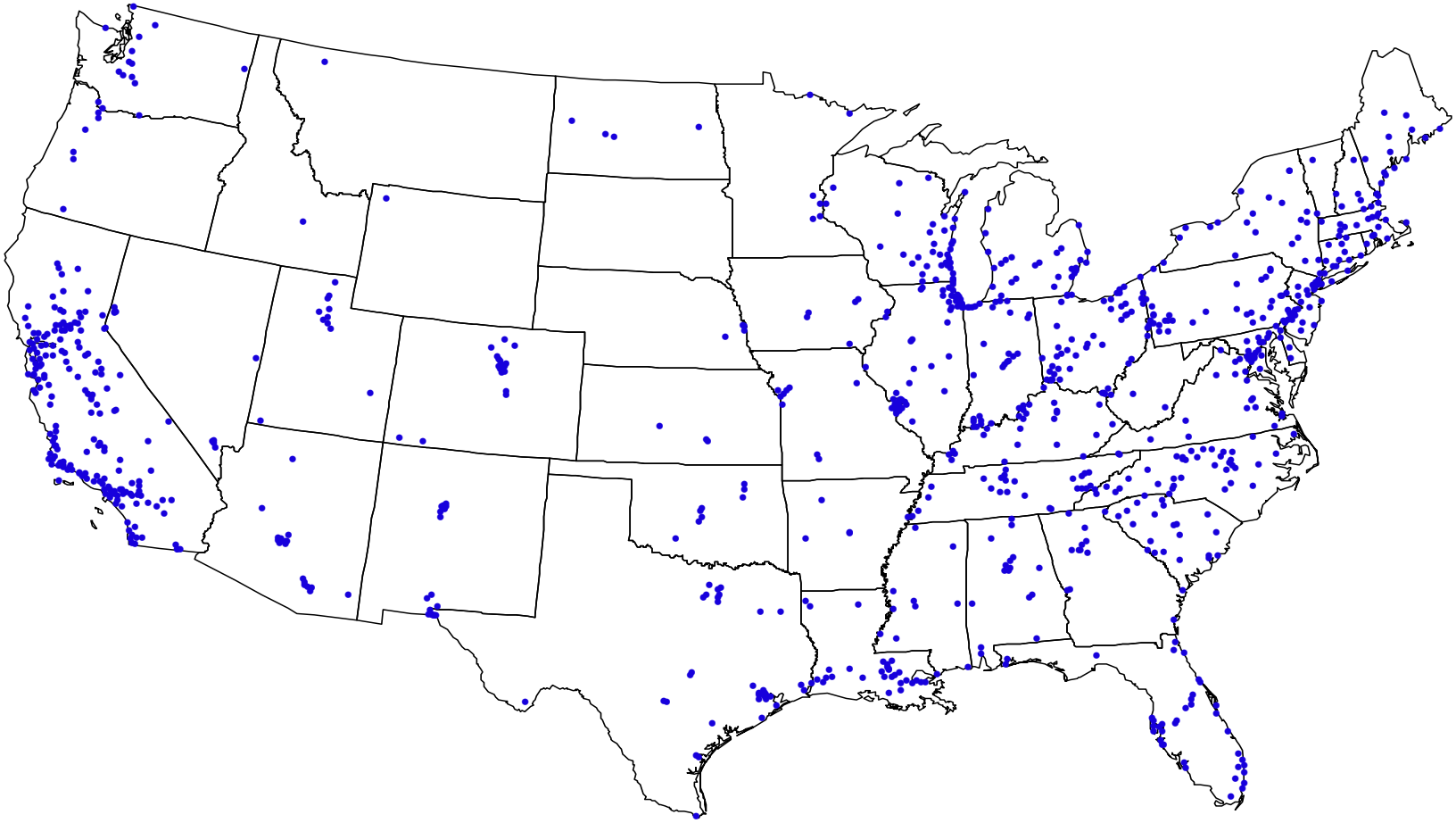
2.2.2 Ozone Monitoring Data

Since the monitoring data should match the base-year modeling, we used monitoring data from 1995 for the Eastern domain, and monitoring data from 1996 for the Western domain.[3] We dropped any monitors with a POC code greater than four, and further dropped any monitors that did not have a sufficient number of valid observation days.[4] We defined a valid observation day as one with at least nine hourly observations between 8:00am and 7:59pm, and we kept only those ozone monitors with 50 percent or more valid days during the ozone season (defined as May 1 through September 30). Finally, for those locations with more than one ozone monitor, we selected the monitor with the lowest POC code (e.g., we chose POC 1 rather than 2), and dropped any others. This resulted in a set of 961 monitors for use in this analysis. Exhibit 2-4 presents the locations of the monitors used in this analysis.

[3] The data used in this analysis were downloaded as AMP350 files from the (password-protected) mainframe version of AIRS, originally available at: epaibm.rtpnc.epa.gov.

[4] The Parameter Occurence Code (POC) is used to distinguish between two or more instruments or monitoring techniques measuring the same parameter at the same site.

Exhibit 2-4. Ozone Monitor Locations



2.2.3 Calculating Spatial and Temporal VNA for Ozone

When using spatial and temporal VNA to forecast ozone levels, we first adjusted the ozone monitor data with the representative model values. Second, given the adjusted monitor values, we calculated the metrics of interest at each monitor, and then calculated an inverse-distance weighted average of the metrics.

To calculate the representative model values, we first sorted the CAMX modeled hourly values by concentration level for the base-year and the future-year, and then split evenly the ordered hourly values into the ten rank-ordered deciles.[5] We then calculated the average of hourly values in each decile, and used this average as the representative value for that decile. This means that the first decile's representative ozone level equals the average of values within that decile, and so on for the other deciles.

To prepare the monitor data, we sorted all of the hourly monitor values by concentration level, and assigned the monitor data to one of ten groups – thus providing the link between the monitor and model data. The monitor values in the lowest group were paired with the lowest of the model deciles, and so on through each of the ten groups.

After preparing the model and monitor data, we used VNA to identify the set of neighbor monitors for each population grid cell, and then combined the representative model values and the ozone monitoring data. In the case of ozone, we calculated the following:

$$adjusted\ monitor_{i,j,future} = monitor_{i,j,base} \cdot \frac{CAMX_{j,k,future}}{CAMX_{j,l,base}}$$

where:

adjusted monitor	= predicted hourly ozone level, after adjustment by model data (ppb)
monitor	= observed hourly ozone monitor level (ppb)
i	= hour identifier
j	= model decile group (1 to 10)
k	= grid cell identifier for population grid cell
l	= grid cell identifier for grid cell containing monitor
base	= base-year (Eastern U.S. = 1995; Western U.S. = 1996)
future	= future-year (2020, 2030)
CAMX	= representative model decile value (ppb)

After adjusting the monitor values to reflect air quality modeling, we calculated for each monitor the ozone metrics needed to estimate adverse health effects. Using the neighbors identified for each population grid cell, we then calculated an inverse distance-weighted average of the metrics from these neighboring monitors.

$$population\ grid\ cell_{future} = \sum_{m=1}^{n} adjusted\ monitor_{m,future} \cdot weight_m \cdot$$

[5] The use of more adjustment factors is generally considered desirable because it provides flexibility; however, it can lead to unreasonably large adjustment factors for lower ozone values, unless a threshold is used. If the average in any of the ten deciles fell below ten ppb, then we set the average to ten ppb.

where:

population grid cell	= inverse distance-weighted ozone metric at population grid cell (ppb)
adjusted monitor	= predicted ozone metric, after adjustment by model data (ppb)
m	= monitor identifier
future	= future-year (2020, 2030)
weight	= inverse-distance weight for monitor

After generating the bins for both the baseline and control scenarios, we take the difference between these two values at each bin. We subtract the baseline value in the first bin from the control value in the first bin, and so on for each of the 153 bins that we created for the daily ozone metrics. For each population grid cell, we then get 153 values representing the difference between the baseline and control, and we use these to estimate the change in adverse effects associated with the implementation of the policy.

Exhibit 2-5 summarizes the population-weighted ozone values that we calculated. Note that the results show that ozone levels are generally increased in the control scenario in the Western region. This occurs because NO_x emissions reductions from the preliminary modeled standards result in less "nitrogen-scavenging" and higher ozone concentrations for certain hours during the year, especially in urban, NO_x-limited areas.

Exhibit 2-5. Population-Weighted Average of Ozone Metrics Used in Concentration-Response Functions for Criteria Air Pollutants

		2020 [b]			2030 [b]		
Region	**Metric [a]**	***Base Case***	***Change***	***% Change***	***Base Case***	***Change***	***% Change***
Eastern U.S.	1-Hour Maximum	51.34	-0.67	-1.31%	51.47	-1.16	-2.25%
	5-Hour Average	42.77	-0.47	-1.10%	42.90	-0.84	-1.96%
	8-Hour Average	42.07	-0.46	-1.08%	42.19	-0.82	-1.93%
	12-Hour Average	39.65	-0.40	-1.00	39.75	-0.72	-1.80%
	24-Hour Average	30.24	-0.18	-0.60%	30.29	-0.37	-1.23%
Western U.S.	1-Hour Maximum	63.80	0.34	0.54%	64.23	0.38	0.58%
	5-Hour Average	53.56	0.45	0.84%	53.89	0.55	1.03%
	8-Hour Average	51.96	0.46	0.88%	52.29	0.57	1.10%
	12-Hour Average	47.68	0.49	1.02%	47.99	0.63	1.32%
	24-Hour Average	35.53	0.47	1.31%	35.74	0.63	1.77%

[a] These ozone metrics are calculated at the CAPMS grid-cell level for use in health effects estimates based on the results of spatial and temporal Voronoi Neighbor Averaging. We calculated the population-weighted average by summing the product of the projected grid-cell population and the estimated PM concentration over all grid-cells, and then dividing by the total population.

[b] The base-case is the population-weighted average over the course of the 153 days in the ozone season (defined as May 1 through September 30). The change is defined as the population-weighted average baseline minus the population-weighted average control. The percent change is the "Change" divided by the "Base Case," and then multiplied by 100 to convert the value to a percentage.

2.3 $PM_{2.5}$ Exposure Forecasts

To develop baseline and control forecasts for $PM_{2.5}$, we used spatial and temporal VNA, combining air quality modeling data from the Regulatory Model System for Aerosols and Deposition (REMSAD) and air quality monitoring data from a nationwide network of monitors.

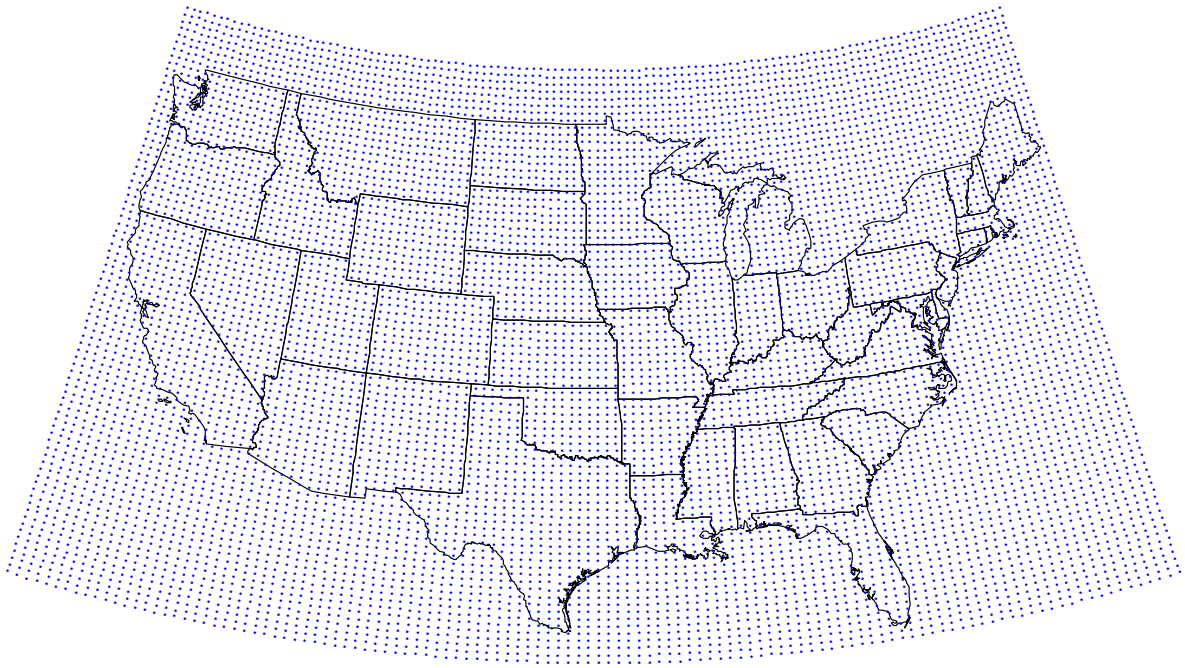
2.3.1 $PM_{2.5}$ Model Data

The $PM_{2.5}$ modeling data are based on the Agency's application of a national-scale version of REMSAD, which is a three-dimensional grid-based Eulerian air quality model designed to estimate PM concentrations and deposition over large spatial scales (e.g., continental U.S.). REMSAD is useful for evaluating the impacts of the preliminary rule on U.S. PM concentrations, because it can assess the preliminary rule's effect on both primary and secondary PM, as well as account for spatial and temporal variations in emissions, and the reactivity of emissions.

The base-year for the REMSAD modeling is 1996, and the modeling for future-years is based on emission forecasts for 2020 and 2030. As with the CAMx modeling, we use REMSAD in a relative sense, by taking the ratio of future-year to base-year model values and scaling monitor values.

The REMSAD modeling domain completely encompasses the contiguous 48 States (see Exhibit 2-6). The domain extends from 126 degrees west longitude to 66 degrees west longitude, and from 24 degrees north latitude to 52 degrees north latitude. The model contains horizontal grid-cells across the model domain of roughly 36 km by 36 km. There are 8 vertical layers of atmospheric conditions with the top of the modeling domain at roughly 16,000 meters. The 36 by 36 km horizontal grid results in a 120 by 84 grid (or 10,080 grid-cells) for each vertical layer.

Exhibit 2-6. REMSAD Modeling Domain for $PM_{2.5}$ Exposure Forecasts



2.3.2 $PM_{2.5}$ Monitoring Data

Since $PM_{2.5}$ monitoring data are not widely available for 1996, the base-year of the modeling, we used monitoring data from 1999 through 2001. We combined two types of monitoring data sets that we received from EPA[6] – AMP500 and AMP501. The AMP500 contains a description of each monitor, such as the latitude and longitude, and the AMP501 has the actual $PM_{2.5}$ observations. We started with data from 1999, 2000 and 2001, and then trimmed it in the following fashion. We only used data from federal reference monitors, which are denoted by "method" codes 116, 117, 118, 119, 120, and 123; we only used data from "local" PM2.5 (designated by pollutant code 88101); we included only those monitors located in the continental U.S., between latitude 24 to 55 and longitude -126 to -66; and we dropped monitors with a POC code greater than four.

The data we received from EPA were either averaged every hour, or every 24 hours. We are interested in 24-hour averages, since the concentration-response functions used in the current analysis are based on 24-hour (and annual averages), but never on hourly averages. In calculating 24-hour averages from the hourly observations, we kept the observations for any given day, only if there are 18 or more

[6] Text files with AMP500 and AMP501 files received from Virginia Ambrose (ambrose.virginia@epa.gov).

over the course of the day. For days with 18 hourly values, we averaged the available observations, even the occasional negative observation. (In no cases are the resulting 24-hour averages ever negative.)

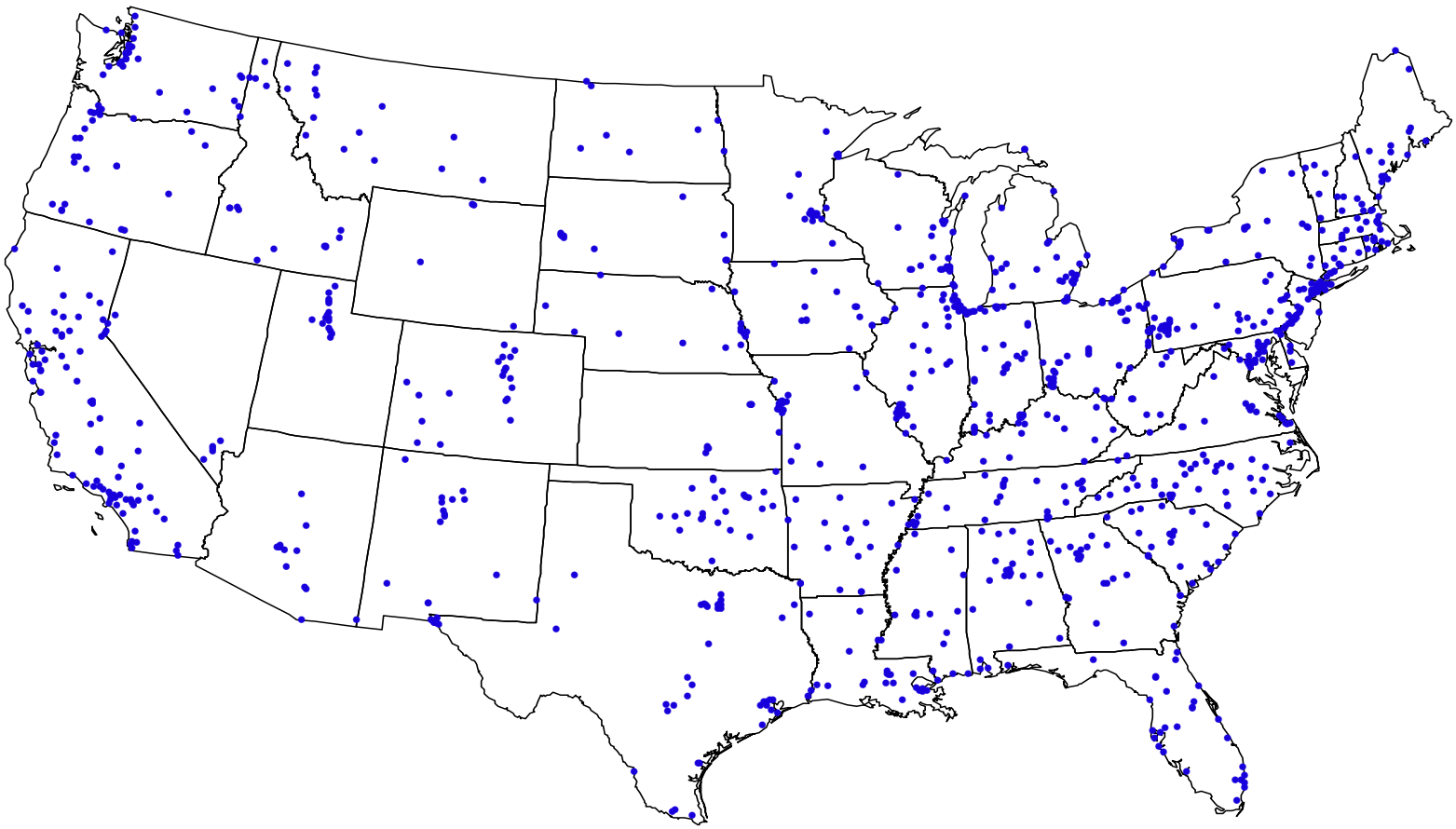
For each of the three years of monitoring, we divided the data into four seasons: Winter (January 1 - March 31), Spring (April 1 - June 30), Summer (July 1 - September 30), and Fall (October 1 - December 31). If a season has fewer than 11 observations, we dropped the values for that season. Alternatively, if a season has 11 or more observations, we create a binned data set for that season – that is, we use the available observations for the season, and assume that any missing values have the same distribution as the available ones.

Specifically, to create a binned data set, we sort the available observations in each season from low to high, and assign a percentage weight to each based on its relative frequency. For example, if there are 30 available observations in the Winter (which has 90 days in a non-leap year), and three of them have a value of 17 $\mu g/m^3$, then we assume that 10 percent (= 3/30) of Winter-time days have a value of 17 $\mu g/m^3$. So, when developing 90 days of data for Winter, we would assign nine of the days (= 0.10*90) a value of 17 $\mu g/m^3$. Similarly, if six of the 30 available observations have a value of 23 $\mu g/m^3$, then we would assign 20 percent (= 6/30), or 18 of the 90 Winter days, a value of 23 $\mu g/m^3$.

In this fashion, to the extent possible, we created complete data sets for winter, spring, summer and fall, for each monitor for each year. The completed seasons had 90, 91, 92, and 92 binned values, respectively, and together comprised 365 daily binned values over the course of a year. We then calculated an average over the three years of data. If, say, Winter values were available for 1999, 2000 and 2001, then we averaged the binned values. If, say, Winter values were available for only one year, then we used that single year of data. If a given monitor did not have a valid season (i.e., in all three years, there were fewer than 11 observations) then we dropped the monitor. Finally, in those cases where there is more than one valid monitor at a given location, we selected the monitor with the lowest POC code (e.g., we chose POC 1 rather than 2), and dropped any others.

These various steps resulted in a set of 1073 $PM_{2.5}$ monitors for use in this analysis. Exhibit 2-7 presents the locations of the monitors used in this analysis.

Exhibit 2-7. PM2.5 Monitor Locations



2.3.3 Calculating Spatial and Temporal VNA for $PM_{2.5}$

When using spatial and temporal VNA to forecast $PM_{2.5}$ levels, we first adjusted the $PM_{2.5}$ monitor data with the representative model values. Second, given the adjusted monitor values, we calculated the metrics of interest at each monitor, and then calculated an inverse-distance weighted average of the metrics.

To calculate the representative model values, we first divided the REMSAD modeled daily values by the four seasons of interest, sorted each season by concentration level, and then split evenly the ordered daily values into the five rank-ordered quintiles.[7] For each season, we then calculated the average of daily values in each quintile, and used this average as the representative value for that season's quintile. This means that the first quintiles's representative $PM_{2.5}$ level equals the average of the values within that season's quintile, and so on for the other quintiles.

To prepare the monitor data, we divided the monitor data into the four seasons, we then sorted all of the daily monitor values by concentration level for each season, and then assigned the monitor data to one of twenty season/quintile groups – thus providing the link between the monitor and model data. The monitor values in the lowest group for a given season, were paired with the lowest of the model quintiles for that season, and so on through each of the twenty season/quintile groups.

After preparing the model and monitor data, we used VNA to identify the set of neighbor monitors for each population grid cell, and then combined the representative model values and the $PM_{2.5}$ monitoring data. In the case of $PM_{2.5}$, we calculated the following:

$$adjusted\ monitor_{i,j,future} = monitor_{i,j,base} \cdot \frac{REMSAD_{j,k,future}}{REMSAD_{j,l,base}}$$

where:

adjusted monitor	= predicted daily $PM_{2.5}$ level, after adjustment by model data ($\mu g/m^3$)
monitor	= observed daily $PM_{2.5}$ monitor level ($\mu g/m^3$)
i	= day identifier
j	= model season/quintile group (1 to 20)
k	= grid cell identifier for population grid cell
l	= grid cell identifier for grid cell containing monitor
base	= base-year (1996)
future	= future-year (2020, 2030)
REMSAD	= representative model season/quintile value ($\mu g/m^3$)

After adjusting the monitor values to reflect air quality modeling, we calculated for each monitor the $PM_{2.5}$ metrics needed to estimate adverse health effects. Using the neighbors identified for each population grid cell, we then calculated an inverse distance-weighted average of the metrics from these neighboring monitors.

$$population\ grid\ cell_{future} = \sum_{m=1}^{n} adjusted\ monitor_{m,future} \cdot weight_m \cdot$$

[7] Unlike the CAMx modeling data, where we used a minimum value of 10 ppb because of some unusually large adjustment factors, we did not assume any minimum level for the REMSAD particulate matter modeling data, as we did not see any results that caused concern.

where:

population grid cell	= inverse distance-weighted $PM_{2.5}$ metric at population grid cell ($\mu g/m^3$)
adjusted monitor	= predicted $PM_{2.5}$ metric, after adjustment by model data ($\mu g/m^3$)
m	= monitor identifier
future	= future-year (2020, 2030)
weight	= inverse-distance weight for monitor

After generating the bins for both the baseline and control scenarios, we take the difference between these two values at each bin. We subtract the baseline value in the first bin from the control value in the first bin, and so on for each of the 365 bins that we created for the daily $PM_{2.5}$ average. For each population grid cell, we then get 365 values representing the difference between the baseline and control, and we use these to estimate the change in adverse effects associated with the implementation of the policy. Exhibits 2-8 and 2-9 summarize the forecasted $PM_{2.5}$ values that we calculated.

Exhibit 2-8. Summary of Forecasted $PM_{2.5}$ Levels Due to Nonroad Engine/Diesel Fuel Standards: 2020 and 2030

Summary of Forecasted of Annual Mean $PM_{2.5}$ Levels [a]	2020 [b]			2030 [b]		
	Base Case	*Change*	*% Change*	*Base Case*	*Change*	*% Change*
Average	8.10	-0.20	-2.5%	8.37	-0.28	-3.4%
Median	7.50	-0.18	-2.7%	7.71	-0.22	-2.8%
Population-Weighted Average	12.42	-0.42	-3.3%	13.07	-0.59	-4.5%

[a] All of the statistics are for the annual mean of the REMSAD grid-cells, and are based on the results of spatial and temporal Voronoi Neighbor Averaging. The annual mean is based on average of 365 daily averages. The "average annual mean" is the simple average of all REMSAD grid-cell mean. The "median annual mean" is the median of the REMSAD grid-cell means. We calculate the "population-weighted average annual mean" by summing the product of the projected grid-cell population and the estimated PM concentration over all grid-cells, and then dividing by the total population.

[b] The change is defined as the control-case value minus the base-case value. The percent change is the "Change" divided by the "Base Case," and then multiplied by 100 to convert the value to a percentage.

Exhibit 2-9. Distribution of Forecasted PM2.5 Air Quality Improvements Over Population Due to Nonroad Engine/Diesel Fuel Standards: 2020 and 2030

Change in Annual Mean $PM_{2.5}$ Concentrations ($\mu g/m^3$) [a]	2020 Population		2030 Population	
	Number (millions)	*Percent (%)*	*Number (millions)*	*Percent (%)*
$0 > \Delta PM_{2.5}$ Conc ≤ 0.25	65.11	19.8%	28.60	8.0%
$0.25 > \Delta PM_{2.5}$ Conc ≤ 0.5	184.52	56.0%	147.09	41.3%
$0.5 > \Delta PM_{2.5}$ Conc ≤ 0.75	56.66	17.2%	107.47	30.2%
$0.75 > \Delta PM_{2.5}$ Conc ≤ 1.0	14.60	4.4%	38.50	10.8%
$1.0 > \Delta PM_{2.5}$ Conc ≤ 1.25	5.29	1.6%	8.82	2.5%
$1.25 > \Delta PM_{2.5}$ Conc ≤ 1.5	3.51	1.1%	15.52	4.4%
$1.5 > \Delta PM_{2.5}$ Conc ≤ 1.75	0	0.0%	5.70	1.6%
$\Delta PM_{2.5}$ Conc > 1.75	0	0.0%	4.19	1.2%

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

3 General Issues in Estimating Health and Welfare Benefits

Changes in ozone, PM, and visibility levels result in changes in a number of health and welfare effects, or "endpoints," that society values. This chapter discusses key issues in the estimation of adverse health effects and in the valuation of health and welfare benefits. The first section discusses the development of C-R functions, based on the results from epidemiological studies. The second section briefly describes how CAPMS calculates adverse health effects, and the third section discusses some general issues that arise with C-R functions. The fourth describes general issues in valuing health and welfare changes, and the final section discusses how uncertainty is characterized in this analysis.

3.1 Choosing Epidemiological Studies and Developing Concentration-Response Functions

This section reviews the steps we performed in selecting and developing C-R functions. The first section of this appendix describes how we chose studies from the epidemiological literature for use in the present analysis. In any given study, there are often a large number of estimated relationships between air pollution and adverse health effects, because the estimated relationship can depend on the number and types of pollutants included in the model, among other reasons. The second section describes how we chose the specific estimated relationships, or models, from among the potentially large number available in any given study. In the third section, we briefly discuss how we convert the estimated model into C-R functions, which then allow us to quantify the change in adverse health effects due to a change in air pollution exposure. Appendix B provides more details on the derivation of C-R functions.

3.1.1 Study Selection

We relied on an up-to-date assessment of the published scientific literature to ascertain the relationship between particulate matter and ozone exposure and adverse human health effects. We evaluated studies using a variety of selection criteria, including: its location and design, the characteristics of the study population, and whether the study was peer-reviewed (Exhibit 3-1).

In selecting studies for use in this analysis, priority was given to studies that focused on $PM_{2.5}$ and ozone, given that the emissions reductions from nonroad sources are likely to result primarily in reduced ambient $PM_{2.5}$ and ozone levels. For a given health effect, if sufficient $PM_{2.5}$ studies were available, we selected them rather than PM_{10} studies in the base analysis. In addition, results from several recent studies allowed for the inclusion of new health effects, such as myocardial infarction for $PM_{2.5}$ and school loss days for ozone.

While a broad range of serious health effects have been associated with exposure to elevated ozone and PM levels, we include only a subset of health effects in this quantified benefit analysis. Health effects are excluded from this analysis for three reasons: (i) the possibility of double counting (such as hospital admissions for specific respiratory diseases); (ii) uncertainties in applying effect relationships based on clinical studies to the affected population; or (iii) a lack of an established C-R relationship.

A more detailed description of the studies and health effects included in this analysis are presented in Appendices F and G.

Exhibit 3-1. Summary of Considerations Used in Selecting C-R Functions

Consideration	Comments
Peer reviewed research	Peer reviewed research is preferred to research that has not undergone the peer review process.
Study type	Among studies that consider chronic exposure (e.g., over a year or longer) prospective cohort studies are preferred over cross-sectional studies because they control for important individual-level confounding variables that cannot be controlled for in cross-sectional studies.
Study period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and life style over time. However, when there are only a few studies available, studies from all years will be included.
Population attributes	The most technically appropriate measures of benefits would be based on C-R functions that cover the entire sensitive population, but allow for heterogeneity across age or other relevant demographic factors. In the absence of C-R functions specific to age, sex, preexisting condition status, or other relevant factors, it may be appropriate to select C-R functions that cover the broadest population, to match with the desired outcome of the analysis, which is total national-level health impacts.
Study size	Studies examining a relatively large sample are preferred because they generally have more power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population, or through repeated observations on a smaller population, i.e. through a symptom diary recorded for a panel of asthmatic children.
Study location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior and life style.
Pollutants included in model	When modeling the effects of ozone and PM (or other pollutant combinations) jointly, it is important to use properly specified C-R functions that include both pollutants. Use of single pollutant models in cases where both pollutants are expected to affect a health outcome can lead to double-counting when pollutants are correlated.
Measure of PM	For this analysis, C-R functions based on $PM_{2.5}$ are preferred to PM_{10} because reductions in emissions from diesel engines are expected to reduce fine particles and not have much impact on coarse particles. Where $PM_{2.5}$ functions are not available, PM_{10} functions are used as surrogates, recognizing that there will be potential downward (upward) biases if the fine fraction of PM_{10} is more (less) toxic than the coarse fraction.
Economically valuable health effects	Some health effects, such as forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are not quantified in this analysis.
Non-overlapping endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double counting of benefits. Including emergency room visits in a benefits analysis that already considers hospital admissions, for example, will result in double counting of some benefits if the category "hospital admissions" includes emergency room visits.

3.1.2 Model Selection

For any given study selected for use in this analysis, there are often multiple models quantifying the relationship between air pollution exposure and adverse health. For each model, we needed to identify the specific models that we would use to develop C-R functions.

Single Pollutant versus Multipollutant Models

Many of the epidemiological studies present results both for the case where only one pollutant is entered into the health effects model, or single-pollutant models, and where two or more pollutants are entered into the health effects model, or multi-pollutant models. When attempting to quantify the impact of a single pollutant, such as $PM_{2.5}$, on adverse health effects, the use of single-pollutant models may result in biased estimates. For example, to the extent that any of the co-pollutants present in the ambient air may have contributed to the health effects attributed to $PM_{2.5}$ in single pollutant models, risks attributed to $PM_{2.5}$ might be overestimated where C-R functions are based on single-pollutant models.

In multi-pollutant models, it may be difficult to sort out which pollutants are exerting an independent effect when pollutants in a given location are highly correlated. As discussed in the 2002 draft PM CD (U.S. EPA, 2002a), inclusion of pollutants that are highly correlated with one another can lead to misleading conclusions in identifying a specific causal pollutant. When collinearity exists, multi-pollutant models would be expected to produce unstable and statistically insignificant effects estimates for both PM and the co-pollutants (U.S. EPA, 2002a, p.9-130).

Single- and multi-pollutant models each have potential advantages and disadvantages, with neither type clearly preferable over the other, however, the regulatory focus of this analysis is on PM and ozone. For regulatory analyses which consider two pollutants together, adding incidence changes for a given health endpoint, based on a single-pollutant PM model, to the incidence changes based on a single-pollutant ozone model could result in an overestimate of incidence change, if both have an effect on the health endpoint and there is some collinearity between the two pollutants.

As a result, our first choice for this analysis is to use multi-pollutant models with both PM and ozone, rather than single-pollutant models and multi-pollutant models with other pollutants. If multi-pollutant models with both PM and ozone were not available, then models with other co-pollutants were preferred to single-pollutant models. In the absence of multi-pollutant models from a given study, single pollutant models were selected for use in the analysis.

Model Selection Criteria

In many epidemiological studies of air pollution and health, researchers estimate and present numerous single pollutant and multi-pollutant models for the same pollutant and health endpoint. These models may differ from each other in a number of characteristics, including: the functional form of the model, the covariates included in the model, the pollutant exposure metric, the lag structure, and the study population.

For the purposes of estimating health benefits associated with pollutant changes, it is neither realistic nor advantageous to include every model presented in each study. However, it is important that a relatively objective process be used to select among models. Described below are the criteria that were used as guidance in the selection of a particular model from among several models presented in a study. It is not possible in all cases to select a model using a completely objective and mechanical process. In many cases, professional judgement and an understanding of the study context are necessary as well to select the most appropriate models. Exhibit 3-2 summarizes the selection criteria that we used.

Exhibit 3-2. Description of Selection Criteria

Selection Criteria	Description
Goodness-of-fit statistics	If an appropriate measure for model selection is reported for each of several models in a study, then this measure may be used as the basis on which to select a model.
Best captures distributed lag	Select the model that appears to best capture a distributed lag effect: If multiple single-lag models and/or moving average models are specified, select the model with the largest effect estimate, all else equal.
Best set of control variables	Select the model which includes temporal variables (i.e. season, weather patterns, day of the week) and other known non-pollutant confounders, all else equal. Select the model which uses the most sophisticated methods of capturing the relationship between these variables and the dependent variable (e.g., affords the most flexibility in fitting possible nonlinear trends).
Useful for health effects modeling	The model must be in a form that is useful for health effects modeling (e.g., the pollutant variable should be a continuous variable rather than a categorical variable).
Biologically plausible	Select only those models that are biologically plausible.
Sample size	Select the model with the larger sample size, all else equal.

Goodness-of-Fit Statistics

Model specification (or mis-specification) is one of the most important issues confronting researchers – and those who apply the results of their research. The goal is to select the "right model" – i.e., the model that has included all the variables that should be in the model (i.e., are relevant) and has not included any variables that should not be in the model (i.e., that are irrelevant). However, is not often known which model is the "right model." There are several ways of selecting one model from among several. One way is to use a goodness of fit measure, which provides a measure of how well a model fits the data. There are a variety of goodness of fit measures available, but use of such measures can at times be misleading. In order to select models based on a goodness of fit criterion, it is important to understand the meaning behind typical goodness of fit measures.

One of the most common goodness of fit measures is R^2, often called the "explained variance" or the "coefficient of multiple determination." R^2 measures the proportion of the total variability in the dependent variable (e.g., the daily incidence of a health effect) that is explained by the linear regression. The closer R^2 is to 1.0, the greater this proportion. The problem with R^2, however, is that it can be increased simply by adding more independent variables to the model, regardless of the variables' relevance to predicting the dependent variable. In the extreme, if there are N observations in the dataset, a model with N explanatory variables will result in an R^2 of 1, but it would be a meaningless model with respect to predictive value. If several models are reported in a study, all with the same number of variables, this drawback is avoided. In that case, if R^2 is reported for each estimated model, this may be a reasonable measure of the relative goodness of fit of the models and an acceptable way to select a single model from among several.

In many cases, however, R^2 is a problematic measure of goodness of fit, for the reason stated above. In view of the drawback of R^2, several alternative measures of goodness of fit have been developed which essentially penalize the model for additional variables – or, equivalently, give "points" for parsimony. Two of the more commonly used of these measures are the adjusted R^2 and Akaike's Information Criterion (AIC). The selection criterion, using the adjusted R^2, is to select the model with the largest adjusted R^2; the selection criterion, using the AIC, is to select the model with the smallest AIC. Both of these measures offset the incremental "fit" gained by including variables in the model with a

"penalty" for increasing the number of explanatory (independent) variables. It should be noted, however, that several such measures have been suggested, and there is no clear way to determine which of these measures to select the "best" model is itself the "best" measure. Nevertheless, if a measure of goodness of fit (particularly, one of the measures that consider parsimony) is presented in a paper, this provides a reasonable means by which to select one model out of several.

Often, however, no goodness of fit measure is presented in a paper. A common approach for deciding the appropriate set of independent variables to be included in a model is to include a variable if its t-value exceeds the critical value for testing whether the variable's coefficient is significantly different from zero at the 5 percent level. Several variables, or an entire model, can similarly be tested with an F-test. (If all the coefficients are being tested jointly, the null hypothesis being tested is that all the coefficients are zero, in which case the model has no more predictive value than the mean of the dependent variable.) In some cases, a comparison of F-statistics (or their corresponding p-values) can be used to select from among several models – in particular, if the F-test for one model suggests that one cannot reject the null hypothesis at the five percent level whereas the F-test for another model suggests that one should reject the null hypothesis.[8]

For example, Stieb et al. (1996) estimated the association between ozone and ER visit rates using both a linear model (in which ER visit rate was a linear function of ozone level) and a quadratic model (in which ER visit rate was a linear function of ozone level squared). No goodness of fit measure was reported in the paper. However, model p-values were reported. The linear model was not statistically significant at the 5% level, whereas the quadratic model was highly significant. This suggests that the linear model does no better in predicting ER visits than the mean of ER visits, whereas the quadratic model has predictive value. The authors of the study themselves noted that "only ozone appeared to have a nonlinear relationship with visit rates" (Stieb et al., 1996, p. 1356) and that "quadratic, linear-quadratic, and indicator models consistently fit the data better than the linear model ..." (Stieb et al., 1996, p. 1358). Based on the relative model p-values presented in the paper, corroborated by the authors' observations, the quadratic model was selected for inclusion in this analysis.

Best Captures a Distributed Lag Effect.

The question of lags and the problems of correctly specifying the lag structure in a model has been discussed extensively (U.S. EPA, 2002a, Section 8.4.4). In many time-series studies, after the basic model is fit (before considering the pollutant of interest), several different lags are typically fit in separate single-lag models and the most significant lag is chosen. The 2002 draft PM CD notes that "while this practice may bias the chance of finding a significant association, without a firm biological reason to establish a fixed pre-determined lag, it appears reasonable" (U.S. EPA, 2002a, p. 8-237).

There is recent evidence (Schwartz, 2000b) that the relationship between PM and health effects may best be described by a distributed lag (i.e., the incidence of the health effect on day n is influenced by PM concentrations on day n, day n-1, day n-2 and so on). If this is the case, a model that includes only a single lag (e.g., a 0-day lag or a 1-day lag) is likely to understate the total impact of PM. The 2002 draft PM CD makes this point, noting that "if one chooses the most significant single lag day only, and if more than one lag day shows positive (significant or otherwise) associations with mortality, then reporting a RR [relative risk] for only one lag would also underestimate the pollution effects" (U.S. EPA, 2002a, p. 8-241). The same may hold true for other pollutants that have been associated with various health effects.

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[8] If F-statistics are both (or all) greater than the critical value, it is less clear that a comparison of these F-statistics would be a good way to select a model.

Several studies report similar models with different lag structures. For example, Moolgavkar (2000b) studied the relationship between air pollution and respiratory hospital admissions in three U.S. metropolitan areas. The author reports models with PM lagged from zero to five days. Since the lagging of PM was the only difference in the models and the relationship is probably best described using a distributed lag model, any of single-lag effect estimates are likely to underestimate the full effect. Therefore, we selected the model with the largest effect estimate.

Most Sophisticated Model That Includes Temporal Variables

A correctly specified model for evaluating air pollution and health would include all variables that are relevant independent predictors of the health outcome and none that are not. If there are variables that are known from prior literature to be associated with both air pollution and the health endpoint (e.g. temperature or season), then omitting these variables is likely to result in biased effect estimates – the C-R function would attribute too much or too little of the health effect to the pollutant. Since temporal and weather patterns are known to confound the relationship between air pollution and health, we selected the models which, all else equal, adjusted for these factors over those that did not.

Useful for Health Effects Modeling

In order for a model to be selected for use, the pollutant must be a continuous variable, so that changes in incidence of the health effect can be predicted to result from any change in pollutant concentration. Those models which examine the effects of being above or below a pollutant threshold or those that look at changes in health associated with categories of pollutant levels are not useful for this purpose.

For example, in a study of the association between air pollution and emergency room (ER) visits, Stieb et al. (1996) estimated several different models. One of these models relates ER visit rates to being above or below the 95th percentile value of ozone (that is, it essentially estimates an average ER visit rate for days above the 95th percentile value of ozone and a different average ER visit rate for days below it). In another study, Peters et al. (2001) estimated a model using quintiles of PM levels. None of these models is appropriate for use in CAPMS. Instead, we selected models which associate the incidence (rate) of a health effect with the pollutant concentration.

Biologically Plausible.

If a model includes a relationship that simply doesn't make biological sense, it is probably mis-specified and should not be used for predictive purposes. It is sometimes not clear, however, what is biologically plausible and what is not. For example, Stieb et al. (1996) estimated a linear-quadratic model – i.e., a model which included both ozone and the square of ozone as independent variables – in a study of air pollution and ER visits. The coefficient of the linear term in this model was negative, while the coefficient of the quadratic term was positive. A graph of the model showed a curve which "dips" at low levels of ozone – i.e. at low ozone levels, increases in ozone are associated with *decreases* in risk. Since ozone is not likely to be beneficial at any levels, this model is not considered to be biologically plausible.

Sample Size

Several studies report the results of an analysis using different population subsets. All else equal, the model based on the larger population, which results in more statistical power, is selected. For example, Pope et al. (1991) studied the association between PM and respiratory health in children. The authors report results for a school-based sample of 34 children and a patient-based sample of 21 residents. Since there are many more observations in the school-based sample (3,096 versus 1,912) and no other significant differences between the models, the model estimated from the school-based sample is used.

Chen et al. (2000) examined the association between air pollution and school absenteeism. The authors reported results by elementary school grade and for all grades combined. With all else equal in the models, the C-R function with the larger number of observations was selected (i.e., the model based on all grades combined).

As discussed above, these criteria are used as general guidance when it is not obvious which model should be chosen from a particular study. The purpose of this process is to provide as objective a protocol as possible for selecting C-R functions. However, model selection can never be a completely mechanical and objective process because it often depends on the specific context of the particular study. In some cases, consideration of several of the aforementioned criteria must be weighed before selecting C-R functions for use in the analysis. The C-R functions selected for use in this analysis are described below with study summaries and a description of which model was selected, when multiple models are available.

3.1.3 Basic Concentration-Response Model

The methods discussed in this sub-section apply to the estimation of both ozone-related and PM-related changes in adverse health effects. For expository simplicity, the discussion focuses primarily on PM-related changes. The methods, however, are equally applicable to ozone-related changes in effects. Similarly, while several health endpoints have been associated with ozone and PM, the discussion below refers only to a generic "health endpoint," denoted as y.

Different epidemiological studies may have estimated the relationship between PM and a particular health endpoint in different locations. The C-R functions estimated by these different studies may differ from each other in several ways. They may have different functional forms; they may have measured PM concentrations in different ways; they may have characterized the health endpoint, y, in slightly different ways; or they may have considered different types of populations. For example, some studies of the relationship between ambient PM concentrations and mortality have excluded accidental deaths from their mortality counts; others have included all deaths. One study may have measured daily (24-hour) average PM concentrations while another study may have used two-day averages. Some studies have assumed that the relationship between y and PM is best described by a linear form (i.e., the relationship between y and PM is estimated by a linear regression in which y is the dependent variable and PM is one of several independent variables). Other studies have assumed that the relationship is best described by a log-linear form (i.e., the relationship between the natural logarithm of y and PM is estimated by a linear regression).[9] Finally, one study may have considered changes in the health endpoint

[9] The log-linear form used in the epidemiological literature on PM-related health effects is often referred to as "Poisson regression" because the underlying dependent variable is a count (e.g., number of deaths), believed to be Poisson distributed. The model may be estimated by regression techniques but is often estimated by maximum likelihood techniques. The form of the model, however, is still log-linear.

only among members of a particular subgroup of the population (e.g., individuals 65 and older), while other studies may have considered the entire population in the study location.

The estimated relationship between PM and a health endpoint in a study location is specific to the type of population studied, the measure of PM used, and the characterization of the health endpoint considered. For example, a study may have estimated the relationship between daily average PM concentrations and daily hospital admissions for "respiratory illness," among individuals age 65 and older, where "respiratory illness" includes International Classification of Disease (ICD) codes A, B, and C.[10] If any of the inputs had been different (for example, if the entire population had been considered, or if "respiratory illness" had consisted of a different set of ICD codes), the estimated C-R function would have been different. When using a C-R function estimated in an epidemiological study to estimate changes in the incidence of a health endpoint corresponding to a particular change in PM in a population cell, then, it is important that the inputs be appropriate for the C-R function being used -- i.e., that the measure of PM, the type of population, and the characterization of the health endpoint be the same as (or as close as possible to) those used in the study that estimated the C-R function.

Estimating the relationship between PM and a health endpoint, y, consists of (1) choosing a functional form of the relationship and (2) estimating the values of the parameters in the function assumed. The two most common functional forms in the epidemiological literature on PM (and ozone) and health effects are the log-linear and the linear relationship. The log-linear relationship is of the form:

$$y = Be^{\beta \cdot PM} \quad ,$$

or, equivalently,

$$\ln(y) = \alpha + \beta \cdot PM \quad ,$$

where the parameter B is the incidence of y when the concentration of PM is zero, the parameter β is the coefficient of PM, ln(y) is the natural logarithm of y, and $\alpha = \ln(B)$.[11] If the functional form of the C-R relationship is log-linear, the relationship between ΔPM and Δy is:

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

where y is the baseline incidence of the health effect (i.e., the incidence before the change in PM). For a log-linear C-R function, the relative risk (RR) associated with the change ΔPM is:

$$RR_{\Delta PM} = e^{\beta \cdot \Delta PM} \quad .$$

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[10] The International Classification Codes are described at the website of the Medical Center Information Systems: Duke University Health Systems (1999).

[11] Other covariates besides pollution clearly affect mortality. The parameter B might be thought of as containing these other covariates, for example, evaluated at their means. That is, $B = B_o \exp\{\beta_1 x_1 + ... + \beta_n x_n\}$, where B_o is the incidence of y when all covariates in the model are zero, and $x_1, ... , x_n$ are the other covariates evaluated at their mean values. The parameter B drops out of the model, however, when changes in incidences are calculated, and is therefore not important.

Epidemiological studies often report a relative risk for a given ΔPM, rather than the coefficient, β, in the C-R function. The coefficient can be derived from the reported relative risk and ΔPM, however, by solving for β:

$$\beta = \frac{\ln(RR)}{\Delta PM} \ .$$

The linear relationship is of the form:

$$y = \alpha + \beta \cdot PM \ ,$$

where α incorporates all the other independent variables in the regression (evaluated at their mean values, for example) times their respective coefficients. When the C-R function is linear, the relationship between a relative risk and the coefficient, β, is not quite as straightforward as it is when the function is log-linear. Studies using linear functions usually report the coefficient directly.

If the functional form of the C-R relationship is linear, the relationship between ΔPM and Δy is simply:

$$\Delta y = \beta \cdot \Delta PM \ .$$

A few epidemiological studies, estimating the relationship between certain morbidity endpoints and PM, have used functional forms other than linear or log-linear forms. Of these, logistic regressions are the most common. Appendix B provides further details on the derivation of C-R functions.

3.2 Calculating Adverse Health Effects with CAPMS

CAPMS is a population-based system for modeling population exposure to ambient levels of criteria air pollutants and estimating the adverse health effects associated with this exposure. CAPMS divides the United States into grid cells that correspond to either the REMSAD or the CAMX air quality models. CAPMS estimates the changes in incidence of adverse health and welfare effects associated with given changes in air quality in each grid cell, and then sums the results to obtain county, state or national results.

To calculate point estimates of the changes in incidence of a given selection of adverse health and welfare effects associated with a given set of air quality changes, CAPMS performs a series of calculations at each population grid-cell. First, CAPMS accesses the C-R functions needed for the analysis, and then accesses any data needed by the C-R functions. Typically, these includes the grid-cell population, the change in population exposure at the grid-cell, and the appropriate incidence rate. CAPMS then calculates the change in incidence of each adverse health effect for which a C-R function has been accessed.

In estimating the change in population exposure, CAPMS estimates the population exposure in the baseline and control scenarios, as described in Chapter 2, and then takes the difference between the two. In the case of binned daily values (365 bins for PM and 153 for ozone), CAPMS calculates the change in the nth bin concentration as the difference between the baseline n^{th} bin concentration and the control scenario n^{th} bin concentration.

For functions based on changes in daily average pollutant concentrations, estimated incidence changes corresponding to air quality changes on each day are summed. This procedure is applied to each grid cell in CAPMS. The resulting incidence change is stored, and CAPMS proceeds to the next grid cell, where the above process is repeated. The national change (or the change in any designated geographical area) is calculated at the end of the process by summing the grid cell-specific changes.

To reflect the uncertainty surrounding predicted incidence changes resulting from the sampling uncertainty surrounding the pollutant coefficients in the C-R functions used, CAPMS produces a *distribution* of possible incidence changes for each adverse health effect, rather than a single point estimate. To do this, it uses both the point estimate of the pollutant coefficient (β) in the C-R function and the standard error of the estimate to produce a normal distribution with mean equal to the estimate, and standard deviation equal to the standard error of the estimate.

Using a Latin Hypercube method,[12] CAPMS takes the nth percentile value of β from this normal distribution, for n = 2.5, 7.5, ..., 97.5, and produces an estimate of the incidence change, given the β selected. Repeating the procedure for each value of β selected results in a distribution of incidence changes in the population grid-cell. This distribution is stored, and CAPMS proceeds to the next population grid-cell, where the process is repeated. A distribution of the national change (or change in a designated geographical area) is calculated by summing the nth percentile grid cell-specific changes, for n = 2.5, 7.5, ..., 97.5.

3.3 Issues in Using Concentration-Response Functions

This section briefly summarizes some of the issues that arise when using C-R functions.

3.3.1 S-Plus Issue

Recently, the Health Effects Institute (HEI) reported findings by health researchers at Johns Hopkins University and others that have raised concerns about aspects of the statistical methods used in a number of recent time-series studies of short-term exposures to air pollution and health effects (Greenbaum, 2002). The estimates derived from the long-term exposure studies, which account for a major share of the economic benefits described in Chapter 9, are not affected. Similarly, the time-series studies employing generalized linear models (GLMs) or other parametric methods, as well as case-crossover studies, are not affected. As discussed in HEI materials provided to EPA and to CASAC (Greenbaum, 2002), researchers working on the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) found problems in the default "convergence criteria" used in Generalized Additive Models (GAM) and a separate issue first identified by Canadian investigators about the potential to underestimate standard errors in the same statistical package. These and other scientists have begun to reanalyze the results of several important time series studies with alternative approaches that address these issues and have found a downward revision of some results. For example, the mortality risk estimates for short-term exposure to PM_{10} from NMMAPS were overestimated (this study was not used in this benefits analysis of fine particle effects). However, both the relative magnitude and the direction of bias introduced by the convergence issue is case-specific. In most cases, the concentration-response relationship may be

[12] The Latin Hypercube method is used to enhance computer processing efficiency. It is a sampling method that divides a probability distribution into intervals of equal probability, with an assumption value for each interval assigned according to the interval's probability distribution. Compared with conventional Monte Carlo sampling, the Latin Hypercube approach is more precise over a fewer number of trials because the distribution is sampled in a more even, consistent manner (Decisioneering, 1996, pp. 104-105).

overestimated; in other cases, it may be underestimated. The preliminary reanalyses of the mortality and morbidity components of NMMAPS suggest that analyses reporting the lowest relative risks appear to be affected more greatly by this error than studies reporting higher relative risks (Dominici et al., 2002).

During the compilation of the draft Air Quality Criteria Document, examination of the original studies used in our benefits analysis found that the health endpoints that are potentially affected by the GAM issues include: reduced hospital admissions in both the Base and Alternative Estimates, reduced lower respiratory symptoms in both the Base and Alternative Estimates, and reduced premature mortality due to short-term PM exposures in the Alternative Estimate. While resolution of these issues is likely to take some time, the preliminary results from ongoing reanalyses of some of the studies used in our analyses (e.g., Dominici et al., 2002) suggest a more modest effect of the S-plus error than reported for the NMMAPS PM_{10} mortality study. In December 2002, a number of researchers submitted reanalysis reports, and the HEI is currently coordinating review of these reports by a peer review panel. The final report on these reanalyses is expected by the end of April 2003, and the results will be incorporated in the fourth external review draft of the Criteria Document that will be released in summer 2003. While we wait for further clarification from the scientific community, we have chosen not to remove these results from the Nonroad Diesel benefits estimates, nor have we elected to apply any interim adjustment factor based on the preliminary reanalyses EPA will continue to monitor the progress of this concern, and make appropriate adjustments as further information is made available.

3.3.2 Composition of PM

Inhalation of fine particles is interpreted as being causally associated with premature death at concentrations near those experienced by most Americans on a daily basis. While biological mechanisms for this effect have not yet been definitively established, the weight of the available epidemiological evidence supports an assumption of causality.

All fine particles, regardless of their chemical composition, are assumed to be equally potent in causing premature mortality. This is an important assumption, because fine particles directly emitted from diesel engines are chemically different from fine particles resulting from both utility sources and industrial facilities, but no clear scientific grounds exist for supporting differential effects estimates by particle type. The major epidemiological studies used to support this benefits analysis have used indices of particles that do not differentiate by particle type. For example, the American Cancer Society (Pope et al., 1995) and Harvard six-city (Dockery et al., 1993) analyses use total $PM_{2.5}$ mass as the index of particle exposure for most of their analyses.

3.3.3 Thresholds

When conducting clinical (chamber) and epidemiological studies, C-R functions may be estimated with or without explicit thresholds. Air pollution levels below the threshold are assumed to have no associated adverse health effects. When a threshold is not assumed, as is often the case in epidemiological studies, any exposure level is assumed to pose a non-zero risk of response to at least one segment of the population.

The possible existence of an effect threshold is a very important scientific question and issue for policy analyses such as this one. The EPA Science Advisory Board Advisory Council for Clean Air Compliance, which provides advice and review of EPA's methods for assessing the benefits and costs of the Clean Air Act under Section 812 of the Clean Air Act, has advised EPA that there is currently no scientific basis for selecting a threshold of 15 $\mu g/m^3$ or any other specific threshold for the PM-related

health effects considered in typical benefits analyses (U.S. EPA, 1999e). This is supported by the recent literature on health effects of PM exposure (Rossi et al., 1999; Daniels et al., 2000; Pope, 2000; Schwartz, 2000c) which finds in most cases no evidence of a non-linear concentration-response relationship and certainly does not find a distinct threshold for health effects. The most recent draft of the EPA Air Quality Criteria for Particulate Matter (U.S. EPA, 2002a) reports only one study, analyzing data from Phoenix, AZ, that reported even limited evidence suggestive of a possible threshold for $PM_{2.5}$ (Smith et al., 2000).

Recent cohort analyses by the Health Effects Institute (Krewski et al., 2000) and Pope et al. (Pope et al., 2002) provide additional evidence of a quasi-linear concentration-response relationship between long-term exposures to $PM_{2.5}$ and mortality. According to the latest draft PM criteria document, Krewski et al. "found a visually near-linear relationship between all-cause and cardiopulmonary mortality residuals and mean sulfate concentrations, near-linear between cardiopulmonary mortality and mean $PM_{2.5}$, but a somewhat nonlinear relationship between all-cause mortality residuals and mean $PM_{2.5}$ concentrations that flattens above about 20 $\mu g/m^3$. The confidence bands around the fitted curves are very wide, however, neither requiring a linear relationship nor precluding a nonlinear relationship if suggested by reanalyses." The Pope et al. analysis, which represented an extension to the Krewski et al. analysis, found that the concentration-response relationships relating $PM_{2.5}$ and mortality "were not significantly different from linear associations."

Daniels et al. (2000) examined the presence of threshold in PM_{10} concentration-response relationships for daily mortality using the largest 20 U.S. cities for 1987-1994. The results of their models suggest that the linear model was preferred over spline and threshold models. Thus, these results suggest that linear models without a threshold may well be appropriate for estimating the effects of PM_{10} on the types of mortality of main interest. Schwartz and Zanobetti (2000) investigated the presence of threshold by simulation and actual data analysis of 10 U.S. cities. In the analysis of real data from 10 cities, the combined concentration-response curve did not show evidence of a threshold in the PM_{10}-mortality associations. Schwartz et al. (2002) investigated thresholds by combining data on the $PM_{2.5}$-mortality relationships for six cities and found an essentially linear relationship down to 2 $\mu g/m^3$, which is at or below anthropogenic background in most areas. They also examined just traffic related particles and again found no evidence of a threshold. The Smith et al. (2000) study of associations between daily total mortality and $PM_{2.5}$ and $PM_{10-2.5}$ in Phoenix, AZ (during 1995-1997) also investigated the possibility of a threshold using a piecewise linear model and a cubic spline model. For both the piecewise linear and cubic spline models, the analysis suggested a threshold of around 20 to 25 $\mu g/m^3$. However, the concentration-response curve for $PM_{2.5}$ presented in this publication suggests more of a U- or V-shaped relationship than the usual "hockey stick" threshold relationship.

Based on the recent literature and advice from the SAB, we assume there are no thresholds for modeling health effects. Although not included in the Base Estimate, the potential impact of a health effects threshold on avoided incidences of PM-related premature mortality is explored as a key sensitivity analysis.

Our assumptions regarding thresholds are supported by the National Research Council (2002)in its recent review of methods for estimating the public health benefits of air pollution regulations. In their review, the National Research Council concluded that there is no evidence for any departure from linearity in the observed range of exposure to PM_{10} or $PM_{2.5}$, nor any indication of a threshold. They cite the weight of evidence available from both short and long term exposure models and the similar effects found in cities with low and high ambient concentrations of PM.

3.3.4 Application of a Single C-R Function Everywhere

Whether the C-R relationship between a pollutant and a given health endpoint is estimated by a single function from a single study or by a pooled function of C-R functions from several studies, that same C-R relationship is applied everywhere in the benefits analysis. Although the C-R relationship may in fact vary somewhat from one location to another (for example, due to differences in population susceptibilities or differences in the composition of PM), location-specific C-R functions are available only for those locations in which studies were conducted.

While a single function applied everywhere may result in overestimates of incidence changes in some locations and underestimates of incidence changes in other locations, these location-specific biases will to some extent cancel each other out when the total incidence change is calculated. It is not possible to know the extent or direction of the bias in the total incidence change based on application of a single C-R function everywhere.

3.3.5 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), Schwartz (2000a), and Zanobetti et al. (2002) that focused exclusively on this issue, reported that short-term harvesting does not play a major role in the PM-mortality relationship.[13]

However, it is not likely 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.

3.3.6 Estimating Effects for Multiple Age Groups

This analysis focused on two future years, 2020 and 2030, and the population age distribution is expected to change over time, with a greater percentage of the population moving into older age categories. Because baseline incidence rates for older populations tend to exceed those for younger populations for several health endpoints (most importantly, for mortality), this demographic shift has important implications for the estimation of future-year incidence change. If we were to apply a C-R

[13] 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."

function to an entire population, using one average baseline incidence, this demographic shift would be missed, and the future-year incidence change would be significantly underestimated.

To take into account projected demographic shifts and the corresponding implications for predicted incidence change, we applied C-R functions to separate age groups within the entire population to which a C-R function is applicable, using projected populations in each age group. Projected baseline incidences (incidence rates times populations) used in the calculation of future-year pollutant-related incidence change therefore better reflect the expected demographic shifts. We note that, because we did not attempt to estimate changes in baseline incidence rates (which may decline slightly over time for younger age groups and increase for the oldest groups), we may be mis-estimating incidence change for particular age groups to the extent that baseline incidence rates change over time.

3.3.7 Pooling Study Results

When only a single study has estimated the C-R relationship between a pollutant and a given health endpoint, the estimation of grid cell-specific incidence change, Δy, is straightforward, as noted above. When several studies have estimated C-R relationships between a pollutant and a given health endpoint, the results of the studies can be pooled to derive a single estimate of the function. If the functional forms, pollutant averaging times, and study populations are all the same (or very similar), a pooled, "central tendency" C-R function can be derived from multiple study-specific C-R functions.

However, even if there are differences among the studies that make a pooled C-R function infeasible, a pooled estimate of the incidence change, Δy, and/or the monetary benefit of the incidence change can be obtained by incorporating the appropriate air quality and baseline incidence data into the study-specific C-R functions and pooling the resulting study-specific predictions of incidence change (or monetary benefit) at a national level. This is the approach that was used here.

As with estimates based on only a single study, we want to characterize the uncertainty surrounding pooled estimates of incidence change and/or monetary benefit. To do this, the pooling procedure incorporates both within-study uncertainty and between-study variability to derive a pooled *distribution* of incidence changes (or monetary benefit). This distribution incorporates information from all the studies used in the pooling procedure. The mean of the distribution is used as a point estimate of incidence change (or monetary benefit), and the 5th and 95th percentiles of the distribution are used to characterize the uncertainty surrounding that point estimate.

The relative contribution of any one study in the pooling process depends on the weight assigned to that study. There are various methods that can be used to assign weights to studies. We used a standard weighting procedure, described in detail in Appendix H. The pooling of study results to form a distribution of incidence change (or monetary benefit) proceeded through the following steps:

1. <u>Generate a 20-point national-level Latin Hypercube distribution for each C-R function</u>: For each study-specific C-R function, CAPMS generates 20 Latin Hypercube percentile points representing the distribution of incidence changes that are estimated using that C-R function in a given population grid-cell, as described in Section 3.2 above. This distribution is stored, and CAPMS proceeds to the next population grid-cell, where the process is repeated. A distribution of the national change (or change in a designated geographical area) is calculated by summing the nth percentile grid cell-specific changes, for n = 2.5, 7.5, ..., 97.5. If there is more than one C-R function for the same health endpoint-pollutant combination, there will be a national-level 20-point Latin Hypercube distribution of incidence changes corresponding to each one.

2. An additional step for ozone-related respiratory hospital admissions: At least one study estimated separate C-R functions for the two major components of ozone-related respiratory hospital admissions, COPD and pneumonia, while other studies estimate a C-R function for "all respiratory admissions" as one category. In order to pool the results of these studies, we first develop a national level distribution of "respiratory hospital admissions" from the two separate distributions for COPD and pneumonia hospital admissions from the same study as follows: On each iteration of a Monte Carlo procedure, we

- randomly select one of the 20 Latin Hypercube percentile points for COPD hospital admissions change for the study;
- multiply the selected incidence change estimate by the unit value for COPD hospital admissions;[14]
- randomly select one of the 20 Latin Hypercube percentile points for pneumonia hospital admission change for the study;
- multiply the selected incidence change estimate by the unit value for pneumonia hospital admissions;
- add the incidence change estimates together; and
- add the monetary benefits estimates together.

In the same Monte Carlo procedure, we also create similar distributions of 5,000 estimates, one for incidence change and the other for monetary value, for any other ozone-related respiratory hospital admissions study with which the first study is to be pooled. On each iteration of the Monte Carlo procedure, in addition to the above steps, for each study we

- randomly select one of the 20 Latin Hypercube percentile points from the study's 20-point distribution of respiratory hospital admissions change; and
- multiply the selected incidence change estimate by the unit value for respiratory hospital admissions.

The result is that for each study there are now two national-level distributions of 5,000 estimates each – one for ozone-related "all respiratory hospital admissions" and the other for the monetary benefit of those admissions avoided.

3. Calculate study-specific weights for pooling: Using the study-specific national distributions of incidence change calculated on the previous steps, we calculate a weight for each study – that is, a probability that, on any iteration of the Monte Carlo procedure used for pooling (see next steps), the study will be selected.

   3a. For all endpoints except ozone-related respiratory hospital admissions, study weights are based on the variances of the national-level 20-point Latin Hypercube distributions of incidence changes, generated in step 1. We use weights based on a fixed effects model, unless the fixed effects hypothesis is rejected, in which case we use weights based on a random effects model. The fixed and random effects models, the calculation of fixed and random effects weights, and the hypothesis test to determine which model is appropriate are all described in detail in Appendix H.

   3b. For ozone-related respiratory hospital admissions, we have two distributions of 5,000 estimates each, for each study – one for incidence change and the other for monetary

[14] There are no uncertainty distributions for hospital admissions unit values (see Chapter 4).

value, generated in step 2. The means and variances of the incidence change distributions will be different from those for the corresponding monetary value distributions. Note that for all other endpoints, the same unit value is used for all studies; the variances of the distributions of monetary value are therefore just a multiple of the variances of the distributions of incidence change, so the weights derived from the incidence change distributions will be the same as those derived from the distributions of monetary value. This is not the case for ozone-related respiratory hospital admissions. Therefore, we calculate one set of weights, based on the incidence change distributions and another set of weights based on the monetary value distributions, and we use each separate set of weights in the separate poolings of incidence change and monetary values. As with the other endpoints, we use either fixed effects weights or random effects weights (see Appendix H).

4. Generate a pooled incidence change distribution and a pooled monetary benefit distribution for an endpoint (*not* ozone-related respiratory hospital admissions): To derive pooled distributions of national-level incidence changes and monetary benefits for a health endpoint-pollutant combination, we use a Monte Carlo procedure. On each of 5,000 iterations, we

   - randomly select a study-specific set of 20 Latin Hypercube incidence changes, using the weights generated in step 3a;
   - randomly select one of the 20 points from the national-level distribution for the selected study;
   - randomly select a value from the corresponding unit value distribution; and
   - multiply the two together.[15]

   The result of the first two steps is a distribution of 5,000 estimates of national-level incidence change; the result of all four steps is a distribution of 5,000 estimates of national monetary benefits for the health endpoint. The mean of each distribution is used as the point estimate for the endpoint. The 5th and 95th percentile points are used to characterize the uncertainty surrounding that point estimate.

5. Generate a pooled incidence change distribution for ozone-related respiratory hospital admissions: To derive a pooled distribution of national-level incidence change for ozone-related respiratory hospital admissions, we use a Monte Carlo procedure. On each of 5,000 iterations, we

   - select a study-specific national-level distribution of 5,000 estimates of incidence change, using the incidence change weights generated on step 3b; and
   - randomly select one of the 5,000 estimates from the national level incidence change distribution for the selected study.

   The mean of the distribution is used as the point estimate of incidence change for ozone-related respiratory hospital admissions. The 5th and 95th percentile points are used to characterize the uncertainty surrounding that point estimate.

7. Generate a pooled monetary benefit distribution for ozone-related respiratory hospital admissions: To derive a pooled distribution of national level monetary benefits for ozone-related respiratory hospital admissions, we use a Monte Carlo procedure. On each of 5,000 iterations, we

[15] This appropriately assumes stochastic independence between the incidence change estimate and the unit value estimate.

- select a study-specific national level distribution of 5,000 estimates of monetary value, using the monetary value weights generated on step 3b; and
- randomly select one of the 5,000 estimates from the national level monetary value distribution for the selected study.

The mean of the distribution is used as the point estimate of monetary value for ozone-related respiratory hospital admissions. The 5th and 95th percentile points are used to characterize the uncertainty surrounding that point estimate.

3.4 Issues in Valuing Changes in Health And Welfare Effects

This section discusses a number of issues that arise in valuing changes in health and welfare effects. The first section provides some background on willingness to pay (WTP). The second section discusses the possibility that as income changes then WTP would also change. The third section describes how WTP estimates, that were originally calculated in 1990 dollars, are corrected for inflation to get estimates in 2000 dollars. In the last section, we briefly review how we aggregate benefits estimates.

3.4.1 Willingness-to-Pay Estimation

WTP is a measure of value an individual places on gaining an outcome viewed as desirable, be it something that can be purchased in a market or not. The WTP measure, therefore, is the amount of money such that the individual would be indifferent between having the good (or service) and having the money. An alternative measure of economic value is willingness to accept (WTA) a monetary compensation to offset a deterioration in welfare, such that the individual would be indifferent between having the money and not having the deterioration. Whether WTP or WTA is the appropriate measure depends on how property rights are assigned. Consider an increase in air pollution. If society has assigned property rights so that people have a right to clean air, then they must be compensated for an increase in the level of air pollution. The appropriate measure of the value of avoiding an increase in air pollution, in this case, would be the amount people would be willing to accept in compensation for the more polluted air. If, on the other hand, society has not assigned people the right to clean air, then the appropriate measure of the value of avoiding an increase in air pollution would be what people are willing to pay to avoid it. The assignment of property rights in our society is unclear. WTP is by far the more common measure used in benefits analyses, however, reflecting the fact that this is a much more common measure in the empirical valuation literature. In this analysis, wherever possible, the valuation measures are in terms of WTP. Where such estimates are not available, alternative measures are used, such as cost-of-illness and wage-risk studies. These are discussed for each endpoint where applicable.

For both market and non-market goods, WTP reflects individuals' preferences. Because preferences are likely to vary from one individual to another, WTP for both market (e.g., the purchase of a new automobile) and non-market goods (e.g., health-related improvements in environmental quality) is likely to vary from one individual to another. In contrast to market goods, however, non-market goods, such as environmental quality improvements, are public goods whose benefits are shared by many individuals. The individuals who benefit from the environmental quality improvement may have different WTPs for this non-market good. The total social value of the good is the sum of the WTPs of all individuals who "consume" (i.e., benefit from) the good.

In the case of health improvements related to pollution reduction, it is not certain specifically who will receive particular benefits of reduced pollution. For example, the analysis may predict 100 hospital admissions for respiratory illnesses avoided, but the analysis does not estimate which individuals will be spared those cases of respiratory illness that would have required hospitalization. The health benefits conferred on individuals by a reduction in pollution concentrations are, then, actually *reductions in the risk* of having to endure certain health problems. These benefits (reductions in risk) may not be the same for all individuals (and could be zero for some individuals). Likewise, the WTP for a given benefit is likely to vary from one individual to another. In theory, the total social value associated with the decrease in risk of a given health problem resulting from a given reduction in pollution concentrations is:

$$\sum_{i=1}^{N} WTP_i\left(B_i\right) \ ,$$

where B_i is the benefit (i.e., the reduction in risk of having to endure the health problem) conferred on the ith individual (out of a total of N) by the reduction in pollution concentrations, and $WTP_i(B_i)$ is the ith individual's WTP for that benefit.

If a reduction in pollution concentrations affects the risks of several health endpoints, the total health-related social value of the reduction in pollution concentrations is:

$$\sum_{i=1}^{N} \sum_{j=1}^{J} WTP_i\left(B_{i,j}\right) \ ,$$

where B_{ij} is the benefit related to the jth health endpoint (i.e., the reduction in risk of having to endure the jth health problem) conferred on the ith individual by the reduction in pollution concentrations, and $WTP_i(B_{ij})$ is the ith individual's WTP for that benefit.

The reduction in risk of each health problem for each individual is not known, nor is each individual's WTP for each possible benefit he or she might receive known. Therefore, in practice, benefits analysis estimates the value of a *statistical* health problem avoided. For example, although a reduction in pollutant concentrations may save actual lives (i.e., avoid premature mortality), whose lives will be saved cannot be known *ex ante*. What is known is that the reduction in air pollutant concentrations results in a reduction in mortality risk. It is this reduction in mortality risk that is valued in a monetized benefit analysis. Individual WTPs for small reductions in mortality risk are summed over enough individuals to infer the value of a *statistical* life saved. This is different from the value of a particular, identified life saved. Rather than "WTP to avoid a death," then, it is more accurate to use the term "the value of a statistical life."

Suppose, for example, that a given reduction in PM concentrations results in a decrease in mortality risk of 1/10,000. Then for every 10,000 individuals, one individual would be expected to die in the absence of the reduction in PM concentrations (who would not die in the presence of the reduction in PM concentrations). If WTP for this 1/10,000 decrease in mortality risk is $500 (assuming, for now, that all individuals' WTPs are the same), then the value of a statistical life is 10,000 x $500, or $5 million.

A given reduction in PM concentrations is unlikely, however, to confer the same risk reduction (e.g., mortality risk reduction) on all exposed individuals in the population. (In terms of the expressions above, B_i is not necessarily equal to B_j , for $i \neq j$). In addition, different individuals may not be willing to pay the same amount for the same risk reduction. The above expression for the total social value

associated with the decrease in risk of a given health problem resulting from a given reduction in pollution concentrations may be rewritten to more accurately convey this. Using mortality risk as an example, for a given unit risk reduction (e.g., 1/1,000,000), the total mortality-related benefit of a given pollution reduction can be written as:

$$\sum_{i=1}^{N} \int_{0}^{n_i} marginal\ WTP_i(x)dx \ ,$$

where marginal $WTP_i(x)$ is the ith individual's marginal willingness to pay curve, n_i is the number of units of risk reduction conferred on the ith exposed individual as a result of the pollution reduction, and N is the total number of exposed individuals.

The values of a statistical life implied by the value-of-life studies were derived from specific risk reductions. Implicit in applying these values to a situation involving possibly different risk reductions is the assumption that the marginal willingness to pay curve is horizontal – that is, that WTP for n units of risk reduction is n times WTP for one unit of risk reduction. If the marginal willingness to pay curve is horizontal, the integral in the above expression becomes a simple product of the number of units of risk reduction times the WTP per unit. The total mortality-related benefit (the expression above) then becomes:

If different subgroups of the population have substantially different WTPs for a unit risk

$$\sum_{i=1}^{N} \left(number\ of\ units\ of\ risk\ reduction\right)_i \cdot \left(\frac{WTP_i}{unit\ of\ risk\ reduction}\right) .$$

reduction and substantially different numbers of units of risk reduction conferred on them, then estimating the total social benefit by multiplying the population mean WTP to save a statistical life times the predicted number of statistical lives saved could yield a biased result. Suppose, for example, that older individuals' WTP per unit risk reduction is less than that of younger individuals (e.g., because they have fewer years of expected life to lose). Then the total benefit will be less than it would be if everyone's WTP were the same. In addition, if each older individual has a larger number of units of risk reduction conferred on him (because a given pollution reduction results in a greater absolute reduction in risk for older individuals than for younger individuals), this, in combination with smaller WTPs of older individuals, would further reduce the total benefit.

While the estimation of WTP for a market good (i.e., the estimation of a demand schedule) is not a simple matter, the estimation of WTP for a non-market good, such as a decrease in the risk of having a particular health problem, is substantially more difficult. Estimation of WTP for decreases in very specific health risks (e.g., WTP to decrease the risk of a day of coughing or WTP to decrease the risk of admission to the hospital for respiratory illness) is further limited by a paucity of information.[16] Derivation of the dollar value estimates discussed below was often limited by available information.

[16] Some health effects, such as technical measures of pulmonary functioning (e.g., forced expiratory volume in one second) are frequently studied by epidemiologists, but there has been very little work by economists on valuing these changes (e.g., Ostro et al., 1989).

3.4.2 Change Over Time in WTP in Real Dollars

The WTP for health-related environmental improvements (in real dollars) could change between now and the year 2020 or 2030. If real income increases between now and 2020 or 2030, 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).

We did not adjust the monetary benefits in this analysis to account for changes over time in real income. However, in Chapter 9 of the corresponding Regulatory Impact Analysis (RIA) of the Preliminary Nonroad Land-based Diesel Engines Standards, EPA (2003) made such adjustments to the dollar benefits presented in this analysis. The RIA describes these adjustments in detail.

3.4.3 Adjusting Benefits Estimates to 2000 Dollars

This section describes the methods used to convert benefits estimates into constant dollars. In past RIA analyses, 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 constant dollars in one year (e.g., 1990) to constant dollars in another year (e.g., 2000), the method of adjustment depends on the basis of the benefits estimates. These methods are presented below. Four different bases of estimates are delineated in Exhibit 3-3, including that for agricultural benefits.[17]

Exhibit 3-3. Bases of Benefits Estimation

Basis of Benefit Estimation	Benefit Endpoints
Cost of illness	Hospital admissions Myocardial Infarction Asthma-related ER visits Chronic bronchitis (Alternative Estimate)
Direct estimates of WTP	Statistical lives; statistical life-years Chronic bronchitis (Base Estimate) Chronic asthma Morbidity endpoints using WTP Visibility -- residential Visibility -- recreational Consumer cleaning cost savings
Earnings	Work loss days (WLDs) Increased worker productivity School absences avoided
Changes in yields and prices of market commodities	Agricultural benefits

Benefits estimates based on cost-of-illness have been adjusted by using the consumer price indexes (CPI-Us) for medical care. Because increases in medical costs have been significantly greater

[17] Chapter 5 discusses agricultural benefits.

than the general rate of inflation, using a general inflator (the CPI-U for "all items" or some other general inflator) to adjust from 1990 to 2000 dollars would downward bias cost-of-illness estimates in 2000 dollars.

Benefits 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). The derivation of adjustment factors to convert from 1990 and from 1997 dollars to 2000 dollars for WTP-based and cost-of-illness based valuations is given for purposes of illustration in Exhibit 3-4.

Exhibit 3-4. Consumer Price Indexes Used to Adjust WTP-Based, Wage-Based, and Cost-of-Illness-Based Benefits Estimates to 2000 Dollars

	1990 (1)	1997 (2)	2000 (3)	Adjustment Factor [a] (3)/(1)	Adjustment Factor [a] (3)/(2)	Relevant Endpoints
CPI-U for "All Items" [b]	130.7	160.5	172.2	1.3175	1.0729	WTP-based valuation: 1. Statistical lives saved [c] 2. Chronic bronchitis (Base Estimate) 3. Chronic bronchitis (Alternative Estimate) – opportunity cost component 4. Chronic asthma 5. Morbidity endpoints using WTP [d] 6. Visibility -- residential 7. Visibility -- recreational 8. Consumer cleaning cost savings Wage-based valuation: 1. Work loss days (WLDs) [e] 2. Increased worker productivity [e] 3. School absences avoided [e] 4. Hospital admissions – opportunity cost component 5. Myocardial infarction – opportunity cost component
CPI-U for Medical Care [b]	162.8	234.6	260.8	1.6020	1.1117	Cost-of-illness based valuation: 1. Hospital admissions – hospital charge component [f] 2. Myocardial Infarction – medical cost component 3. Asthma-related ER visits 4. Chronic bronchitis (Alternative Estimate) – medical expenses component

[a] Benefits estimates in 1990 or 1997 dollars are multiplied by the appropriate 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 $ 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 $ were originally made by Industrial Economics Inc. (1993) using the CPI-U for "all items."

[e] Data for these wage-based unit values were already in year 2000 $ and therefore did not require any conversion.

[f] Hospital charges from the Agency for Healthcare Research and Quality's Healthcare Utilization Project (2000) were already in 2000 $ and therefore did not require any conversion.

Finally, agricultural benefits (changes in farm income and consumer welfare) predicted to result in a future year have been adjusted to 2000 dollars from 2010 using a GDP price deflator. In this analysis, 2010 benefits were adjusted to 2000 dollars by multiplying by 0.6735, the ratio of the 2000 GDP price deflator (of 112.3 from:Council of Economic Advisers, 1997, Table B-3) to a projected 2010 GDP price index (of 167.16) forecasted from the trend between 1997 and 2007, obtained from the USDA baseline projections (U.S. Department of Agriculture, 1988b, electronic file Tab01.wk1).

3.4.4 Derivation of the Present Discounted Value of Future Benefits

Some health benefits are not expected to occur until some time after exposure is reduced. The reduction in expected premature mortalities, for example, is expected to be distributed over several years, with some statistical lives saved in the same year as exposure is reduced, some in the next year, and so forth. People generally are willing to pay more for something now than for the same thing later. They would, for example, be willing to pay more for a reduction in the risk of premature death in the same year as exposure is reduced than for that same risk reduction to be received the following year. This time preference for receiving benefits now rather than later is expressed by discounting benefits received later. The exact discount rate that is appropriate (i.e., that represents people's time preference) is a topic of much debate. EPA has typically used a discount rate of 3 percent; OMB has recommended a discount rate of 7 percent. Both are used in this analysis. If d denotes the discount rate, and WTP denotes what an individual would be willing to pay now to receive a benefit now, then that individual's willingness to pay now to receive that same benefit in n years – the present discounted value of that future benefit – is calculated as

$$pdv = \frac{WTP}{(1+d)^n} .$$

Cost-of-illness estimates of the value of avoiding adverse health effects that are chronic effects, such as chronic bronchitis, also involve discounting, for the same reason – many of the avoided costs would have been incurred in future years, over the course of the individual's lifetime. The value of avoiding the stream of costs over a number of years is the present discounted value of those avoided costs – what the individual would pay now to avoid having to pay those costs in the future. Suppose, for example, that an individual with chronic bronchitis incurs medical costs of $200 for each of the first 10 years of having the illness (roughly what has been estimated for someone who gets chronic bronchitis at age 30). The $200 incurred throughout the first year, discounted back to the beginning of the first year, using a 3 percent discount rate, would be ($200)/(1.03) = $194. That is, an individual whose time preference is expressed by a 3 percent discount rate would be indifferent between paying $194 now and paying $200 spread out over the course of the year. The $200 incurred during the second year, discounted back to the beginning of the first year would be $(\$200)/(1.03)^2 = \$189$, and so forth. The present discounted value of the stream of costs over N years is calculated as

$$pdv = \sum_{n=1}^{N} \frac{\$200}{(1+d)^n} .$$

3.4.5 Aggregation of Monetized Benefits

The total monetized benefit associated with attaining a given set of pollution changes in a given location is just the sum of the non-overlapping benefits associated with these changes. In theory, the total health-related social value of the reduction in pollution concentrations is:

$$\sum_{i=1}^{N} \sum_{j=1}^{J} WTP_i\left(B_{i,j}\right) ,$$

where B_{ij} is the benefit related to the jth health endpoint (i.e., the reduction in probability of having to endure the jth health problem) conferred on the ith individual by the reduction in pollution concentrations, and $WTP_i(B_{ij})$ is the ith individual's WTP for that benefit.

However, the reduction in probability of each health problem for each individual is not known, nor do we know each individual's WTP for each possible benefit he or she might receive. Therefore, in practice, benefits analysis estimates the value of a *statistical* health problem avoided. The benefit in the kth location associated with the jth health endpoint is just the change in incidence of the jth health endpoint in the kth location, Δy_{jk}, times the value of an avoided occurrence of the jth health endpoint.

Assuming that WTP to avoid the risk of a health effect varies from one individual to another, there is a *distribution* of WTPs to avoid the risk of that health effect. This population distribution has a mean. It is this population mean of WTPs to avoid or reduce the risk of the jth health effect, $MWTP_j$, that is the appropriate value in the benefit analysis.[18] The monetized benefit associated with the jth health endpoint resulting from attainment of standard(s) in the kth location, then, is:

$$benefit_{jk} = \Delta y_{jk} \cdot MWTP_j$$

and total monetized benefit in the kth location (TMB_k) may be written as the sum of the monetized benefits associated with all non-overlapping endpoints:

$$TMB_k = \sum_{j=1}^{N} \Delta y_{jk} \cdot MWTP_j \ .$$

The location- and health endpoint-specific incidence change, Δy_{jk}, is modeled as the population response to the change in pollutant concentrations in the kth location. The discussion below uses particulate matter as an example but is equally applicable to any other pollutant, such as ozone. Assuming a log-linear C-R function, the change in incidence of the jth health endpoint in the kth location corresponding to a change in PM, ΔPM_k, in the kth location is:

$$\Delta y_{jk} = y_{jk} \cdot \left(e^{\beta_{jk} \cdot \Delta PM_k} - 1\right) ,$$

[18] The population of interest has not been defined. In a location-specific analysis, the population of interest is the population in that location. The MWTP is ideally the mean of the WTPs of all individuals in the location. There is insufficient information, however, to estimate the MWTP for any risk reduction in any particular location. Instead, estimates of MWTP for each type of risk reduction will be taken to be estimates of the MWTP in the United States as a whole, and it will be assumed that $MWTP_i$, i=1, ..., N in each location is approximately the same as in the United States as a whole.

where y_{jk} is the baseline incidence of the jth health endpoint in the kth location and β_{jk} is the value of β_j, the coefficient of PM in the C-R relationship between PM and the jth health endpoint, in the kth location.

This approach assumes that there is a *distribution* of β_j's across the United States, that is, that the value of β_j in one location may not be the same as the value of β_j in another location. The value of β_j in the kth location is denoted as β_{jk}.

The total PM-related monetized benefit for the kth location can now be rewritten as:

$$TMB_k = \sum_{j=1}^{N} y_{jk} \cdot \left(e^{\beta_{jk} \cdot \Delta PM_k} - 1\right) \cdot MWTP_j \ ,$$

The total monetized PM-related benefit to be estimated for a location is thus a function of 2N parameters: the coefficient of PM, β_{jk}, in the C-R function for the jth health (or welfare) endpoint, for j=1, ..., N, specific to the kth location, and the population mean WTP to reduce the risk of the jth health endpoint, $MWTP_j$, j=1, ..., N.

The above model assumes that total monetized benefit is the sum of the monetized benefits from all non-overlapping endpoints. If two or more endpoints were overlapping, or if one was contained within the other (as, for example, hospital admissions for Chronic Obstructive Pulmonary Disease - COPD - is contained within hospital admissions for "all respiratory illnesses"), then adding the monetized benefits associated with those endpoints would result in double (or multiple) counting of monetized benefits. If some endpoints that are not contained within endpoints included in the analysis are omitted, then the aggregated monetized benefits will be less than the total monetized benefits.

The total monetized benefit (TMB) is the sum of the total monetized benefits achieved in each location:

$$TMB = \sum_{k=1}^{K} TMB_k$$

where TMB_k denotes the total monetized benefit achieved in the kth location, and K is the number of locations.

Theoretically, the nation-wide analysis could use location-specific C-R functions to estimate location-specific benefits. Total monetized benefits (TMB), then, would just be the sum of these location-specific benefits:

$$TMB = \sum_{k=1}^{K} TMB_k = \sum_{k=1}^{K} \sum_{j=1}^{N} y_{jk} \cdot \left(e^{\beta_{jk} \cdot \Delta PM_k} - 1\right) \cdot MWTP_j \ ,$$

There are many locations in the United States, however, and the individual location-specific values of β_j (the β_{jk}'s) are not known.[19] Since the national incidence of the jth health endpoint attributed to PM, I_j, is a continuous function of the set of β_{jk}'s, that is, since:

$$I_j = \sum_{k=1}^{K} \Delta y_{jk} = \sum_{k=1}^{K} y_{jk} \cdot \left(e^{\beta_{jk} \cdot \Delta PM_k} - 1 \right) ,$$

is a continuous function of the set of β_{jk}'s, there is some value of β_j , which can be denoted β_j*, that, if applied in *all* locations, would yield the same result as the proper set of location-specific β_{jk}'s. This follows from the Intermediate Value Theorem. While β_j* will result in overestimates of incidence in some locations, it will result in underestimates in others. If β_j* is applied in all locations, however, the *total regional* change in incidence will be correct. That is,

$$I_j = \sum_{k=1}^{K} \Delta y_{jk} = \sum_{k=1}^{K} y_{jk} \cdot \left(e^{\beta_j^* \cdot \Delta PM_k} - 1 \right) ,$$

$$= \sum_{k=1}^{K} y_{jk} \cdot \left(e^{\beta_{jk} \cdot \Delta PM_k} - 1 \right) .$$

The total regional monetized PM-related benefit can now be rewritten as:

$$TMB_k = \sum_{j=1}^{N} \sum_{k=1}^{K} y_{jk} \cdot \left(e^{\beta_j^* \cdot \Delta PM_k} - 1 \right) \cdot MWTP_j .$$

The total regional monetized (PM-related) benefit is thus a function of 2N population means: the β* for the jth health (or welfare) endpoint (β_j* , for j=1, ..., N) and the population mean WTP to reduce the risk of the jth health endpoint ($MWTP_j$, j=1, ..., N).

The above formulation of the total monetized benefits associated with a given set of changes in PM across K locations is applied to ozone as well. The set of health and welfare endpoints may be different for ozone, but the calculation of benefits is the same, with $\Delta ozone_k$ substituted for ΔPM_k everywhere.

Both the endpoint-specific coefficients (the β_j's) and the endpoint-specific mean WTPs (the $MWTP_j$'s) are uncertain, as are the incidence rates and air quality changes. One approach to estimating the total monetized benefit is to simply use the mean values of the endpoint-specific coefficients and mean WTPs in the above formula. It is preferable, however, to characterize not only the mean total monetized benefit but the distribution of possible values of total monetized benefit, using a Monte Carlo approach. The Monte Carlo approach has three steps. First, in each of many (e.g., 5000) iterations, we randomly select a value from the distribution of (national) incidence change of the health or welfare

[19] This may also be true of the y_{ij}'s. It may be desirable to apply the uncertainty analysis used for the β's to these population parameters as well. In the current discussion, however, it is assumed that the location-specific incidences are known and therefore have no uncertainty associated with them. It is also assumed that $MWTP_i$ is the same in all locations.

effect. Second, we randomly select a value from the distribution of unit dollar values for that health or welfare effect. And third, we multiply the two values. The result is a distribution of (e.g., 5000) monetized benefits associated with the given health or welfare effect. From this distribution, we present the mean as well as the 5th and 95th percentiles. In the limit, the mean of the Monte Carlo based distribution of benefits would be equal to the product of the mean values of the endpoint specific coefficients and mean WTPs. However, due to finite sampling, there may be slight deviations between the estimated Monte Carlo mean and the simple mean.

3.5 Characterization of Uncertainty

In any complex analysis, there are likely to be many sources of uncertainty. This analysis is no exception. Many inputs are used to derive the final estimate of economic 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.

Exhibit 3-5 catalogs some more of the selected sources of uncertainties in the benefits 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, including the use of surrogate variables, such as using PM_{10} when $PM_{2.5}$ is not available, excluded variables, and simplification of complex functions
- biases due to omissions or other research limitations.

Exhibit 3-5. Key Sources of Uncertainty in the Benefit Analysis

Uncertainties associated with concentration-response functions	• The value of the ozone- or 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 ozone or PM concentrations observed in the study. • Application of C-R relationships only to those subpopulations matching the original study population.
Uncertainties associated with ozone and 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. • Lack of ozone monitors in rural areas requires extrapolation of observed ozone data from urban to rural areas. • Use of separate air quality models for ozone and PM does not allow for a fully integrated analysis of pollutants and their interactions. • Full ozone season air quality distributions are extrapolated from a limited number of simulation days. • Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern US and underpredicts nitrate in parts of the Western US.
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.
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.
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 2020 and 2030. • Projected population and demographics may not represent well future-year population and demographics.
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. • Future markets for agricultural and forestry products are uncertain.
Uncertainties associated with aggregation of monetized benefits	• Health and welfare benefits estimates are limited to the available C-R functions. Thus, unquantified or unmonetized benefits are not included.

3.5.1 Approach for Addressing Uncertainty

Given the wide variety of sources for uncertainty and the potentially large degree of uncertainty about any estimate, it is necessary for us to address uncertainty in several ways, based on the following types of uncertainty:

Quantifiable Uncertainty in Benefits Estimates. For some parameters or inputs it may be possible to provide a statistical representation of the underlying uncertainty distribution. Quantitative

uncertainty may include measurement uncertainty or variation in estimates across or within studies. For example, the variation in VSL results across the 26 studies that underlie the Base Estimate represent a quantifiable uncertainty.

Uncertainty in the Basis for Quantified Estimates. Often it is possible to identify a source of uncertainty (for example, an ongoing debate over the proper method to estimate premature mortality) that is not readily addressed through traditional uncertainty analysis. In these cases, it is possible to characterize the potential impact of this uncertainty on the overall benefits estimates through sensitivity analyses.

Nonquantifiable Uncertainty. Uncertainties may also result from omissions of known effects from the benefits calculation, perhaps owing to a lack of data or modeling capability. For example, in this analysis we were unable to quantify the benefits of avoided airborne nitrogen deposition on aquatic and terrestrial ecosystems, or avoided health and environmental effects associated with reductions in CO emissions.

It should be noted that even for individual endpoints, there is usually more than one source of uncertainty. This makes it difficult to provide an overall quantified uncertainty estimate for individual endpoints or for total benefits. For example, the C-R function used to estimate avoided premature mortality has an associated standard error which represents the sampling error around the pollution coefficient in the estimated C-R function. It is possible to report a confidence interval around the estimated incidences of avoided premature mortality based on this standard error. However, this would omit the contribution of air quality changes, baseline population incidences, projected populations exposed, and transferability of the C-R function to diverse locations to uncertainty about premature mortality. Thus, a confidence interval based on the standard error gives a misleading picture about the overall uncertainty in the estimates. In this analysis, we present information on the uncertainty surrounding particular C-R and valuation functions. However, this information should be interpreted within the context of the larger uncertainty surrounding the entire analysis.

Our approach to characterizing model uncertainty is to present a base estimate of the benefits, based on the best available scientific literature and methods, and to then provide sensitivity analyses to illustrate the effects of uncertainty about key analytical assumptions. In addition, we provide an estimate of the 90 percent confidence interval surrounding the total benefits estimate, based solely on the statistical uncertainty surrounding the estimated C-R functions and the assumed distributions around the unit values. Our analysis of the preliminary control options has not included formal integrated uncertainty analyses, although we have conducted several sensitivity tests and have analyzed a full Alternative Estimate based on changes to several key model parameters.

The recent report by the National Research Council (2002) on estimating public health benefits of air pollution regulations recommended that EPA begin to move the assessment of uncertainties from its ancillary analyses into its base analyses by conducting probabilistic, multiple-source uncertainty analyses. However, for this proposal we did not attempt to assign probabilities to sensitivity estimates due to a lack of peer-reviewed methods. At this time, we simply demonstrated the sensitivity of our benefits results to key uncertain parameters.

Our estimate of total benefits should be viewed as an approximate result because of the wide range of sources of uncertainty that we have not incorporated. The 90 percent confidence interval based on statistical error and cross-study variability provides some insight into how uncertain our estimate is with regards to those sources of uncertainty. However, it does not capture other sources of uncertainty regarding other inputs to the model, including emissions, air quality, and aspects of the health science not

captured in the studies, such as the likelihood that PM is causally related to premature mortality and other serious health effects.

3.5.2 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 final standard (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).[20] However, as we have noted, 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.[21] 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. Credible ranges do not have to be 90 percent ranges, however. We are using 20-point Latin Hypercubes to represent incidence change distributions, which represent a distribution by 20 percentile points, spaced 5 percentile points apart, starting from the 2.5th and ending with the 97.5th percentile point. Because of this, we do not know either the 5th or the 95th percentile points of the incidence change distributions. Therefore, instead of characterizing uncertainty by 90 percent credible intervals, we use the 2.5th and 97.5th percentiles of the distribution to characterize uncertainty by a 95 percent credible range.

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

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[20] 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 β^*.

[21] 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).

randomly drawn from the unit dollar value distribution, and the total dollar benefit for that iteration is the product of the two.[22] 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.

3.5.3 Base and Alternative Estimates

We calculated the Base Estimate, using our current interpretation of the scientific and economic literature; regarding the best available data, modeling methods; and assumptions most appropriate to adopt in the face of important uncertainties. The majority of the analytical assumptions used to develop the Base Estimate have been reviewed and approved by EPA's Science Advisory Board (SAB). However, we recognize that data and modeling limitations as well as simplifying assumptions can introduce significant uncertainty into the benefit results and that reasonable alternative assumptions exist for some inputs to the analysis, such as the mortality C-R functions.

To address these concerns, we supplemented our Base Estimate of benefits with an Alternative Estimate that shows the impact of changing several important assumptions about the estimation and valuation of reductions in premature mortality. This approach provides an alternative estimate of health benefits using the time series studies in place of cohort studies, as well as alternative valuation methods for mortality and chronic bronchitis.

3.5.4 Impacts on Sensitive Subpopulations

EPA is currently evaluating how air pollution related symptoms in the asthmatic population should be incorporated into the overall benefits analysis. Clearly, studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double-counting. In one specific case, upper respiratory symptoms in children, the only study available was limited to asthmatic children, so this endpoint is included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms, are estimated for the total population of children.

Given the increased susceptibility of the asthmatic population, it is of interest to understand better the specific impacts on asthmatics. We provided a separate set of estimated health impacts for asthmatic populations with the results in Chapter 6. However, the reader should carefully note that these are not additive, nor can they be easily combined with other endpoints to derive total benefits. They are provided only to highlight the potential impacts on a susceptible population.

Several epidemiological studies have estimated concentration-response functions relating pollution to various symptoms (wheeze, cough, dyspnea, chest tightness, shortness of breath) among asthmatics. One or all of these symptoms may be considered indicators of an asthma episode when it

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

occurs in an asthmatic. We currently have the following asthma exacerbation health endpoints in this analysis: asthma attack, moderate or worse, shortness of breath, wheeze, cough, and one or more symptoms.

To gain further understanding into the public health impact of the modeled change in air quality associated with the preliminary control options, we examined the incidence of health effects occurring in three age groups: children (0-17), adults (18-64), and elderly adults (65 and older). Certain endpoints occur only in a subset of age groups, so not all endpoints are reported for all age groups. Two sets of age group estimates were calculated. The first is based on the specific age ranges examined in the epidemiological studies, for example, the Dockery et al (1996) acute bronchitis study focused on a sample population aged 8 to 12. These are the estimates that were used in deriving total incidences as reported in Chapter 6, Exhibit 6-2. In many cases however, the study populations were defined as a matter of convenience or due to data availability, rather than due to any biological factor that would restrict the effect to the specific age group. In order to gain a more complete understanding of the potential magnitude of the health impact in the entire population, we calculate a separate estimate including the health impact on all population within an age group. The two sets of age specific incidence estimates are provided in Chapter 6, Exhibit 6-5.

We also estimated respiratory symptoms and attacks occurring in the asthmatic population, based on the studies defined in Chapter 4, Table 4-23. As with the age group specific estimates, we provide two sets of calculations, one based on applying the C-R function only to the specific population subgroup included in a study's sample population, and another based on applying the C-R function to all populations within a broader population. The two sets of asthma symptom incidences are provided in Chapter 6, Exhibit 6-6. Note that the asthma symptom estimates provided in Exhibit 6-6 are not additive to total benefits. They are provided to show the specific impacts on an especially susceptible subpopulation. Also note that the estimates are not additive even within the exhibit. We have grouped the estimates based on the type of symptoms measured, but there is the potential for considerable overlap. However, these estimates provide an illustration of the consistency of the effects across studies and populations of asthmatics.

Sensitivity Calculations

In addition to the Base and Alternative estimates of benefits, we present a series of sensitivity calculations that make use of other sources of concentration-response and valuation data for key benefits categories, as well as examining key analytical parameters, such as the form of the lag between changes in PM exposure and realization of changes in health outcomes. These sensitivity calculations are conducted relative to the Base Estimate and not for the Alternative Estimate.

The sensitivity estimates can be used to answer questions like "What would total benefits be if we were to value avoided incidences of premature mortality using the a statistical life years approach rather than the VSL approach?" These estimates examine sensitivity to both valuation issues (e.g. the correct value for a statistical life saved) and for physical effects issues (e.g., possible recovery from chronic illnesses). These estimates are not meant to be comprehensive. Rather, they reflect some of the key issues likely to have a significant impact on total benefits..

In our sensitivity calculations, we consider different assumptions regarding key parameters in the Base Estimate. They can be broken down into three broad groups. First, we consider different assumptions regarding the estimation of benefits associated with reductions in premature mortality from long-term exposure, including selection of the C-R function, structure of the lag between reduced exposure and reduced mortality risk, the relationship between age and VSL, and effect thresholds.

Second, we consider additional studies that potentially overlap with studies already included in the Base Estimate. Finally, we consider different assumptions about specific elements of the benefits analysis – infant mortality associated with exposure to PM, treatment of reversals in chronic bronchitis as lowest severity cases, effects of ozone on new incidences of chronic asthma, alternative C-R functions for PM hospital admissions, valuation of residential visibility, valuation of recreational visibility at Class I areas outside of the study regions examined in the Chestnut and Rowe (1990a; 1990b) study, and valuation of household soiling damages. Exhibit 3-6 summarizes the sensitivity calculations.

Exhibit 3-6. Sensitivity Calculations

Category	Description
Long-term Mortality	
Alternative studies for premature mortality	A number of studies provide an alternative estimate of the relationship between chronic PM exposure and mortality.
Alternative mortality lag structures	Calculate the impact different lag structures have on the estimation of benefits associated with avoided mortality incidence.
Thresholds	Calculate the impact varying threshold assumptions have on the estimation of mortality incidence based on the Krewski et al. (2000) study.
Overlapping endpoints	
Ozone-related mortality	Ozone-related mortality benefits estimated using a pooled analysis based on four U.S. studies.
Any-of-19 symptoms.	Due to the potential for overlap with health effects covered in the estimate of MRADs and both PM- and ozone-related asthma attacks, we present Any-of-19 Respiratory Symptoms separately.
Alternative and Supplementary Estimates	
Infant mortality	The Woodruff et al. (1997) study provides an estimate of the relationship between chronic exposure and infant mortality.
Chronic asthma	Avoided incidences of chronic asthma are estimated using the McDonnell et al. (1999) C-R function.
PM10 hospital admissions	Samet et al. (2000) is used to estimate respiratory and cardiovascular hospital admissions.
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.
Value of visibility changes in all Class I areas	Values of visibility changes at Class I areas in California, the Southwest, and the Southeast are transferred to visibility changes in Class I areas in other regions of the country.
Value of visibility changes in Eastern U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Eastern U.S. based on the reported values for Chicago and Atlanta derived from McClelland et al. (1991).
Value of visibility changes in Western U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Western U.S. based on the reported values for Chicago and Atlanta from McClelland et al. (1990).
Household soiling damage	Value of decreases in expenditures on cleaning are estimated using values derived from Manuel et al. (1982).

Sensitivity Calculations for Long-term Mortality

In examining the sensitivity of the mortality estimate, we considered three key assumptions. First, we considered the impact of alternative models examining the relationship between long-term PM

exposure and premature mortality. Second, we considered alternative lag structures. And third, we considered the potential impact of an effects threshold.

Alternative Long-term PM Studies. 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 base analysis (Krewski et al., 2000, mean all-cause mortality). These alternative mortality functions are discussed in more detail in Section 4.

Alternative Lag Structures. As noted by the SAB (U.S. EPA, 1999d, p. 9), "some of the mortality effects of cumulative exposures will occur over short periods of time in individuals with compromised health status, but other effects are likely to occur among individuals who, at baseline, have reasonably good health that will deteriorate because of continued exposure. No animal models have yet been developed to quantify these cumulative effects, nor are there epidemiologic studies bearing on this question." However, they also note that "Although there is substantial evidence that a portion of the mortality effect of PM is manifest within a short period of time, i.e., less than one year, it can be argued that, if no lag assumption is made, the entire mortality excess observed in the cohort studies will be analyzed as immediate effects, and this will result in an overestimate of the health benefits of improved air quality. Thus some time lag is appropriate for distributing the cumulative mortality effect of PM in the population."

In the base analysis, based on SAB advice, we assume that mortality occurs over a five year period, with 25 percent of the deaths occurring in the first year, 25 percent in the second year, and 16.7 percent in each of the third, fourth, and fifth years. Note that the selection of a 5 year lag is not supported by any scientific literature on PM-related mortality. Rather it is intended to be a reasonable guess at the appropriate distribution of avoided incidences of PM-related mortality.

The SAB recommended the five-year distributed lag be used for the base analysis, and also recommended that alternative lag structures be explored as a sensitivity analysis (U.S. EPA, 1999d, p. 9). Specifically, they recommended an analysis of 0, 8, and 15 year lags. The 0 year lag is representative of EPA's assumption in previous RIAs, and the 8 and 15 year lags are based on the study periods from the Pope, et al. (1995) and Dockery, et al. (1993) studies.[23] However, it should be kept in mind that neither study assumed any lag structure when estimating the relative risks from PM exposure, and do not contain any data supporting or refuting the existence of a lag. Any lag structure applied to the avoided incidences estimated from either of these studies is an assumed structure. Moreover, the imposition of a lag structure does not change the total number of estimated deaths, but rather the timing of those deaths.

Thresholds. A number of studies (Rossi et al., 1999; Daniels et al., 2000; Pope, 2000; Schwartz, 2000c) have found little evidence for the existence of a threshold – a point below which further reductions no longer yield premature mortality reduction benefits. Nevertheless, to examine the impact of this possibility, we estimated how the number of premature mortalities in the Base Estimate might change under a range of alternative assumptions for a PM mortality threshold.

One important assumption that we adopted for the threshold sensitivity analysis is that no adjustments are made to the shape of the C-R function above the assumed threshold. Instead, thresholds were applied by simply assuming that any changes in ambient concentrations below the assumed threshold have no impacts on the incidence of premature mortality. If there were actually a threshold,

[23] The 8 and 15 year lags implicitly assume that all premature mortalities occur at the end of the study periods, i.e. at 8 and 15 years.

then the shape of the C-R function would likely change and there would be no health benefits to reductions in PM below the threshold.

Overlapping Endpoints

In order to provide the reader with a fuller understanding of the health effects associated with reductions in air pollution associated with the preliminary control options, this set of sensitivity estimates examines those health effects which, if included in the base estimate, could result in double-counting of benefits. Note that these supplemental estimates should not be considered as additive to the total estimate of benefits, but illustrative of these issues and uncertainties.

➢First, we present an alternative estimate of minor respiratory symptoms, termed Any-of-19 Respiratory Symptoms (Krupnick et al., 1990). We had excluded Any-of-19 Respiratory Symptoms from the base analysis because of potential for overlap with health effects covered in the estimate of MRADs (Ostro and Rothschild, 1989).

➢Second, we present ozone-related mortality. We had excluded ozone-related mortality from the base analysis because of concerns about double-counting the impact of PM and ozone on premature mortality. Additional research is needed to provide separate estimates of the effects for PM and ozone, so to be conservative we included only the effect of PM on premature mortality in the base analysis.

Ozone-Related Mortality. There has been a great deal of research recently on the potential effect of ozone on premature mortality. A recent analysis by Thurston and Ito (2001) reviewed previously published time series studies of the effect of daily ozone levels on daily mortality and found that previous EPA estimates of the short-term mortality benefits of the ozone NAAQS (U.S. EPA, 1997a) may have been underestimated by up to a factor of two. Thurston and Ito hypothesized that much of the variability in published estimates of the ozone/mortality effect could be explained by how well each model controlled for the influence of weather, an important confounder of the ozone/mortality effect, and that earlier studies using less sophisticated approaches to controlling for weather consistently under-predicted the ozone/mortality effect.

Thurston and Ito (2001) found that models incorporating a non-linear temperature specification appropriate for the "U-shaped" nature of the temperature/mortality relationship (i.e., increased deaths at both very low and very high temperatures) produced ozone/mortality effect estimates that were both more strongly positive (a two percent increase in relative risk over the pooled estimate for all studies evaluated) and consistently statistically significant. Further accounting for the interaction effects between temperature and relative humidity produced even more strongly positive results. Inclusion of a PM index to control for PM/mortality effects had little effect on these results, suggesting an ozone/mortality relationship independent of that for PM. However, most of the studies examined by Thurston and Ito only controlled for PM_{10} or broader measures of particles and did not directly control for $PM_{2.5}$. As such, there may still be potential for confounding of $PM_{2.5}$ and ozone mortality effects, as ozone and $PM_{2.5}$ are highly correlated during summer months in some areas.

There are a number of studies examining the relationship between ambient ozone levels and daily mortality levels. We calculated the potential impact of ozone on mortality, using results from four U.S. studies (Kinney et al., 1995; Moolgavkar et al., 1995; Ito and Thurston, 1996; Samet et al., 1997), assuming that demographic and environmental conditions on average would be more similar between these studies and the conditions prevailing with the implementation of the Preliminary Nonroad Landbased Diesel Engine Rule.

Alternative and Supplementary Estimates

We also examined how the value for individual endpoints or total benefits would change if we were to make a different assumption about specific elements of the benefits analysis. Specifically, we examined the impact of alternative assumptions regarding infant mortality associated with exposure to PM, effects of ozone on new incidences of chronic asthma, treatment of reversals in chronic bronchitis as lowest severity cases, alternative C-R functions for PM hospital admissions, valuation of residential visibility, valuation of recreational visibility at Class I areas outside of the study regions examined in the Chestnut and Rowe (1990a; 1990b) study, and valuation of household soiling damages.

Infant Mortality. The estimated effect of PM exposure on premature mortality in infants (post-neonatal) is based on a single U.S. study (Woodruff et al., 1997) that, on recommendation of the EPA Science Advisory Board, was deemed too uncertain to include in the base analysis. Adding this endpoint to the base benefits estimate would result in an increase in total benefits.

Chronic Asthma. Chronic asthma is characterized by repeated incidences of inflammation of the lungs that causes restriction in the airways and results in shortness of breath, wheezing, and coughing. Asthma is also characterized by airway hyper responsiveness to stimuli. We estimate the development of chronic asthma using a recent study by McDonnell, et al. (1999) that found a statistical association between ozone and the development of asthma in adult white, non-Hispanic males. However, questions have been raised regarding the statistical validity of the associations found in this study, and the appropriateness of transferring the estimated C-R function from the study populations (white, non-Hispanic males) to other male populations (e.g., African-American males). Moreover, other studies have not identified an association between air quality and the onset of asthma. We therefore include the results of this study as a sensitivity calculation.

Chronic Bronchitis Reversals. 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 base 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.

Alternative C-R Functions for PM Hospital Admissions. A recent study by the Health Effects Institute (HEI) (Samet et al., 2000) estimated separate models for PM_{10} and pneumonia, COPD and cardiovascular diseases in each of fourteen cities in the United States, as well as pooled estimates across these cities. The pooled estimates from this study are desirable for several reasons. First, the HEI models are distributed lag models that are designed to capture not only same-day effects of PM but the effects of PM on a series of days subsequent to exposure. A second advantage of the HEI models is that they represent the PM effect across a range of cities in the United States. However, we chose not to use this study in the base analysis because it is based on PM_{10}, and most of the estimated change in air quality will affect $PM_{2.5}$ levels, and not coarse PM_{10} levels.

Recreational Visibility. The alternative calculation for recreational visibility is an estimate of the full value of visibility in the entire region affected by the preliminary NonRoad Standard. The Chestnut and Rowe (1990a) study from which the base valuation estimates are derived only examined WTP for visibility changes in the southeastern portion of the affected region. In order to obtain estimates of WTP for visibility changes in the northeastern and central portion of the affected region, we have to transfer the southeastern WTP values. This introduces additional uncertainty into the estimates. However, we have taken steps to adjust the WTP values to account for the possibility that a visibility

improvement in parks in one region, is not necessarily the same environmental quality good as the same visibility improvement at parks in a different region. This may be due to differences in the scenic vistas at different parks, uniqueness of the parks, or other factors, such as public familiarity with the park resource. To take this potential difference into account, we adjusted the WTP being transferred by the ratio of visitor days in the two regions.

Residential Visibility. The alternative calculations for residential visibility are based on the McClelland et al. (1991) study of WTP for visibility changes in Chicago and Atlanta. The residential visibility estimates from the available literature have been determined by the SAB to be inadequate for use in a base estimate in a benefit-cost analysis, because they have not undergone rigorous peer review (U.S. EPA, 1999c, p. 13). However, residential visibility is likely to have some value and the McClelland et al. study is probably the best in estimating the likely magnitude of the benefits of residential visibility improvements.

Household Soiling. The alternative calculation for household soiling is based on the Manuel et al. (1982) study of consumer expenditures on cleaning and household maintenance. However, the data used to estimate household soiling damages in the Manuel et al. study is from a 1972 consumer expenditure survey and as such may not accurately represent consumer preferences in the future. Despite this limitation, we believe that the Manuel et al. estimates are still useful in providing an estimate of the likely magnitude of the benefits of reduced PM household soiling.

3.5.5 Unquantified Benefits

In considering the monetized benefits estimates, the reader should remain aware of the limitations. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the PM and ozone-induced adverse health and welfare effects. Unquantified welfare effects include reduced acid and particulate deposition damage to cultural monuments and other materials; reduced ozone effects on forested ecosystems; and environmental benefits due to reductions of impacts of acidification in lakes and streams and eutrophication in coastal areas (see Exhibit 1-2).

Additionally, we have not quantified a number of known or suspected adverse health effects linked with PM and ozone for which appropriate concentration-response functions are not available or which do not provide easily interpretable outcomes. These include low birth weight, changes in pulmonary function, morphological changes, altered host defense mechanisms, non-fatal cancers, and non-asthma respiratory emergency room visits. These omissions tend to lead to lower Base and Alternative estimates of the monetized benefits attributable to the preliminary control options.

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

The most significant monetized benefits of reducing ambient concentrations of PM and ozone are attributable to reductions in health risks associated with air pollution. This Chapter describes individual effects and the methods used to quantify and monetize changes in the expected number of incidences of various health effects.

We estimate the incidence of adverse health effects using C-R functions based on PM and ozone. The changes in incidence of PM-related and ozone-related adverse health effects and corresponding monetized benefits associated with these changes are estimated separately. Exhibits 4-1 and 4-2 present the PM- and ozone-related health endpoints included in the Base and Alternative Estimates. Exhibit 4-3 presents the incidence rates used for these C-R functions.[24] And Exhibit 4-4 presents the unit monetary values for each of these endpoints.

Note also, that in some cases there are alternative and/or sensitivity endpoints, studies, or unit dollar values that could be used in calculating the benefits of a change in pollution. As appropriate, we included these alternatives in Exhibits 4-1, 4-2, and 4-4.

Below, we discuss for each endpoint issues relating to the calculation of changes in incidence, the monetization of these changes, and the characterization of the uncertainty surrounding our estimates. For some of the endpoint-pollutant combinations, there are several epidemiological studies that have estimated C-R functions. In these cases, we pooled the information from the multiple studies. That is, we based the estimation of the change in incidence and the corresponding monetized value of that change on a synthesis of the information from the available studies. Appendix H gives a detailed description of the pooling methods used in this analysis.

[24] Appendix D presents the general derivation of C-R functions from epidemiological models. Appendices F and G present additional detail on specific C-R functions and the underlying studies. Appendix E provides additional detail on the incidence and prevalence rates used in the C-R functions.

Exhibit 4-1. PM-Related Health Endpoints Used in Base and Alternative Estimates

Health Effect	Applied Ages	Pollutant	Description
Mortality			
Long-term – Base Estimate	30+	$PM_{2.5}$	Krewski et al. (2000), reanalysis of Pope et al. (1995) using the annual mean and all-cause mortality.
Short-term – Alternative Estimate	All	$PM_{2.5}$	Schwartz et al. (1996) adjusted using ratio of distributed lag to single day coefficients from Schwartz et al. (2000b).
Chronic Illness			
Chronic bronchitis	27+	$PM_{2.5}$	Abbey et al (1995c)
Non-fatal heart attacks		$PM_{2.5}$	Peters et al. (2001)
Hospital Admissions			
Respiratory	65+	$PM_{2.5}$	Pooled estimate: Moolgavkar (2000b) - ICD 490-496 (COPD) Lippman et al. (2000) - ICD 490-496 (COPD)
	20-64	$PM_{2.5}$	Moolgavkar (2000b) - ICD 490-496 (COPD)
	65+	$PM_{2.5}$	Lippman et al. (2000) - ICD 480-486 (pneumonia)
	<65	$PM_{2.5}$	Sheppard, et al. (1999) - ICD 493 (asthma)
Cardiovascular	65+	$PM_{2.5}$	Pooled estimate: Moolgavkar (2000a) - ICD 390-429 (all cardiovascular) Lippman et al. (2000) - ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)
	20-64	$PM_{2.5}$	Moolgavkar (2000a) - ICD 390-429 (all cardiovascular)
Emergency Room Visits			
Asthma ER Visits	0-18	$PM_{2.5}$	Norris et al. (1999)
Other Health Effects			
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1996)
Upper respiratory symptoms	Asthmatics, ages 9-11	PM_{10}	Pope et al. (1991)
Lower respiratory symptoms	7-14	$PM_{2.5}$	Schwartz (1994)
Work loss days	18-64	$PM_{2.5}$	Ostro (1987)
Minor restricted activity day	18-64	$PM_{2.5}$	Ostro and Rothschild (1989),

Exhibit 4-2. Ozone-Related Health Endpoints in Base and Alternative Estimates

Health Effect	Applied Ages	Pollutant	Description
Hospital Admissions			
Respiratory	65+	O_3	Pooled estimate: Schwartz (1995) - ICD 460-519 (all resp) Schwartz (1994a; 1994b) - ICD 480-486 (pneumonia) Moolgavkar et al. (1997) - ICD 480-487 (pneumonia) Schwartz (1994b) - ICD 491-492, 494-496 (COPD) Moolgavkar et al (1997) - ICD 490-496 (COPD)
	<2	O_3	Burnett et al. (2001)
Asthma-Related ER Visits	All	O_3	Pooled estimate: Weisel et al. (1995) Cody et al. (1992) Stieb et al. (1996)
Other Health Effects			
School Loss Days		O_3	Pooled estimate:
	9-10		Gilliland et al (2001)
	6-11		Chen et al (2000)
Worker productivity	18-65	O_3	Crocker and Horst (1981) and EPA (1994). Applied to outdoor workers, defined as those engaged in the farming, fishing, and forestry occupations.

Exhibit 4-3. Baseline Incidence Rates and Population Prevalence Rates in Base and Alternative Estimates

Endpoint	Notes	Rate per 100 people per year [a] by Age Group						
		<18	*18-24*	*25-34*	*35-44*	*45-54*	*55-64*	*65+*
Mortality	all-cause	0.045	0.093	0.119	0.211	0.437	1.056	5.050
	chronic-lung	0.000	0.001	0.001	0.002	0.009	0.046	0.278
Chronic Bronchitis [c]		--	--	0.378	0.378	0.378	0.378	0.378
	prevalence [b]	--	3.70%	3.70%	3.70%	5.10%	5.10%	5.90%
Nonfatal MI (heart attacks)		0.000	0.002	0.012	0.089	0.305	0.654	1.442
Hospitalizations	respiratory	0.043	0.084	0.206	0.678	1.926	4.389	11.629
	cardiovascular	1.066	0.271	0.318	0.446	0.763	1.632	5.200
Asthma ER visits		1.011	1.087	0.751	0.438	0.352	0.425	0.232
Acute Bronchitis [d]		4.300	--	--	--	--	--	--
Lower Respiratory Symptoms [e]		43.80	--	--	--	--	--	--
Upper Respiratory Symptoms among asthmatic children [f]		12479	--	--	--	--	--	--
Asthma [g]	prevalence [b]	5.30%	3.70%	3.70%	3.70%	3.30%	3.30%	2.20%
Work Loss Days [h]		--	197.1	247.5	247.5	179.6	179.6	--
Minor Restricted Activity Days [i]		--	780.0	780.0	780.0	780.0	780.0	--
School Loss Days [j]	all-cause	990.0	--	--	--	--	--	--
	illness-related	281.1	--	--	--	--	--	--
	respiratory illness-related	152.1	--	--	--	--	--	--

[a] All of the rates reported here are population-weighted incidence rates per 100 people per year. Appendix E provides further details on the incidence and prevalence rates, as well as the sources for these rates.

[b] "Prevalence" refers to the percentage of the population that has a given adverse health effect (e.g., chronic bronchitis).

[c] The chronic bronchitis incidence rate is calculated for ages 27 and up. The three chronic bronchitis prevalence rates are calculated for ages 18 to 44, 44 to 64, and 65 up.

[d] The acute bronchitis incidence rate is calculated for ages 8 to 12.

[e] The lower respiratory symptoms incidence rate is calculated for ages 7 to 14.

[f] The upper respiratory symptoms incidence rate is calculated for ages 9 to 11.

[g] The four asthma prevalence rates are calculated for ages under 18, 18 to 44, 45 to 64, and 65 up.

[h] The three work loss days incidence rates are calculated for ages 18 to 24, 25 to 44, and 45 to 64.

[i] The minor restricted activity days incidence rate is calculated for ages 18 to 64.

[j] We estimated 180 school days in a year to calculate school absence rates per year.

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

Health Endpoint	Description	Mean Estimate [a]
Mortality [b]	Base Estimate – VSL based on 26 studies	$6.3 million per statistical life
	Alternative Estimates – VSLY	Age / at 3% / at 7% <65 / $172,000 / $286,000 65+ / $434,000 / $527,000
Chronic Bronchitis	Base Estimate – WTP	$340,482 per case
	Alternative Estimate – COI	Age / at 3% / at 7% 27-44 / $150,542 / $86,026 per case 45-64 / $97,610 / $72,261 per case 65+ / $11,088 / $9,030 per case
Non-Fatal Heart Attacks	--	Age / at 3% / at 7% 0-24 / $65,902 / $65,293 per case 25-44 / $74,676 / $73,149 per case 45-54 / $78,834 / $76,871 per case 55-64 / $140,649 / $132,214 per case 65+ / $65,902 / $65,293 per case
Hospital Admissions	all respiratory, ages 65+	$18,353 per admission
	all respiratory, ages 0-2	$7,741 per admission
	pneumonia, ages 65+	$17,802 per admission
	COPD, ages 65+	$13,615 per admission
	COPD , ages 20-64	$11,794 per admission
	asthma, ages < 65	$7,771 per admission
	all cardiovascular ages 65+	$21,162 per admission
	all cardiovascular, ages 20-64	$22,754 per admission
	ischemic heart disease, ages 65+	$25,848 per admission
	dysrhythmias, ages 65+	$15,698 per admission
	congestive heart failure, ages 65+	$15,185 per admission
Emergency Room Visits	Asthma-related	$286 per visit
Minor Effects	Acute bronchitis	$356 per case
	Lower resp. Symptoms	$15.57 per symptom-day
	Upper resp. Symptoms	$24.64 per symptom-day
	Minor restricted activity day (MRAD)	$50.55 per day
	School loss days	$75 per day
	Work loss days	County-specific median daily wage
	Worker productivity	Change in daily wages adjusted by regional variations in income

[a] The derivation of each of the estimates is discussed in the text. Entries in italics are not used in the base benefits analysis.

[b] Long-term exposure mortality was modeled as lagged, both in the base analysis and in the sensitivity analyses, as shown in Exhibit 4-10. The unit values shown here were adjusted to take this into account. For example, the base analysis assumes that 25 percent of premature deaths occur in the first year, 25 percent occur in the second year, and 16.7 percent occur in each of the three subsequent years after exposure. The expected value of an individual's avoided premature death is therefore (0.25 x the unit value) + (0.25 x the unit value discounted back one year) + (16.7 x the unit value discounted back two years) + (16.7 x the unit value discounted back three years) + (16.7 x the unit value discounted back four years). This is true only for long-term exposure mortality, regardless of which of the possible unit values listed above is used.

4.1 Premature Mortality

Health researchers have consistently linked air pollution, especially PM, with excess mortality. Although a number of uncertainties remain to be addressed by continued research (National Research Council, 1998), a substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates.

Both long and short-term exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. It is clearly an important health endpoint because of the size of the mortality risk estimates, the serious nature of the effect itself, and the high monetary value ascribed to avoiding mortality risk. Because of the importance of this endpoint and the considerable uncertainty among economists and policymakers as to the appropriate way to estimate mortality risks, this section discusses some of the issues surrounding the estimation of premature mortality.

4.1.1 Calculating Incidence of Premature Mortality

Both long and short-term exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from these epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most important health endpoint quantified in this analysis. Because of the importance of this endpoint and the considerable uncertainty among economists and policymakers as to the appropriate way to value reductions in mortality risks, both a base and an alternative estimate are provided. As in the Kunzli et al. (2000) analysis, we focus on the prospective cohort long-term exposure studies in deriving the C-R function for our base estimate of premature mortality.

Epidemiological analyses have consistently linked air pollution, especially PM, with excess mortality. Although a number of uncertainties remain to be addressed by continued research (National Research Council, 1998), a substantial body of published scientific literature documents the correlation between elevated PM concentrations and increased mortality rates. Community epidemiological studies that have used both short-term and long-term exposures and response have been used to estimate PM/ mortality relationships. Short-term studies use a time-series approach to relate short-term (often day-to-day) changes in PM concentrations and changes in daily mortality rates up to several days after a period of elevated PM concentrations. Long-term studies examine the potential relationship between community-level PM exposures over multiple years and community-level annual mortality rates. Researchers have found statistically significant associations between PM and premature mortality using both types of studies. In general, the risk estimates based on the long-term exposure studies are larger than those derived from short-term studies. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli et al., 2001; National Research Council, 2002). The alternative estimate is based on time-series studies demonstrating the effect of short-term exposures. This section discusses some of the issues surrounding the estimation of premature mortality.

Base Estimate: Long-term Studies

Over a dozen studies have found significant associations between various measures of long-term exposure to PM and elevated rates of annual mortality, beginning with Lave and Seskin (1977). Most of the published studies found positive (but not always statistically significant) associations with available PM indices such as total suspended particles (TSP). Particles of different fine particles components (i.e.

sulfates), and fine particles, as well as exploration of alternative model specifications sometimes found inconsistencies (e.g., Lipfert et al., 1989). These early "cross-sectional" studies (e.g., Lave and Seskin, 1977; Ozkaynak and Thurston, 1987) were criticized for a number of methodological limitations, particularly for inadequate control at the individual level for variables that are potentially important in causing mortality, such as wealth, smoking, and diet. More recently, several long-term studies have been published that use improved approaches and appear to be consistent with the earlier body of literature. These new "prospective cohort" studies reflect a significant improvement over the earlier work because they include individual-level information with respect to health status and residence.

The most extensive study and analyses has been based on data from two prospective cohort groups, often referred to as the Harvard "Six-City study" (Dockery et al., 1993) and the "American Cancer Society or ACS study" (Pope et al., 1995); these studies have found consistent relationships between fine particle indicators and premature mortality across multiple locations in the U.S. A third major data set comes from the California based 7th Day Adventist Study (e.g., Abbey et al., 1999), which reported associations between long-term PM exposure and mortality in men. Results from this cohort, however, have been inconsistent and the air quality results are not geographically representative of most of the US. More recently, a cohort of adult male veterans diagnosed with hypertension has been examined (Lipfert et al., 2000). The characteristics of this group differ from the cohorts in the ACS, Six-Cities, and 7th Day Adventist studies with respect to income, race, and smoking status. Unlike previous long-term analyses, this study found some associations between mortality and ozone but found inconsistent results for PM indicators.

Given their consistent results and broad geographic coverage, the Six-City and ACS data have been of particular importance in benefits analyses. The credibility of these two studies is further enhanced by the fact that they were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by the Health Effects Institute (Krewski et al., 2000). The final results of the reanalysis were then independently peer reviewed by a Special Panel of the HEI Health Review Committee. The results of these reanalyses confirmed and expanded those of the original investigators. This intensive independent reanalysis effort was occasioned both by the importance of the original findings as well as concerns that the underlying individual health effects information has never been made publicly available.

The HEI re-examination lends credibility to the original studies as well as highlighting sensitivities concerning (a) the relative impact of various pollutants, (b) the potential role of education in mediating the association between pollution and mortality, and (c) the influence of spatial correlation modeling. Further confirmation and extension of the overall findings using more recent air quality and a longer follow up period for the ACS cohort was recently published in the Journal of the American Medical Association (Pope et al., 2002).

In developing and improving the methods for estimating and valuing the potential reductions in mortality risk over the years, EPA has consulted with a panel of the Science Advisory Board. That panel recommended use of long-term prospective cohort studies in estimating mortality risk reduction (U.S. EPA, 1999a). This recommendation has been confirmed by a recent report from the National Research Council, which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure (National Research Council, 2002, p. 108)." More specifically, the SAB recommended emphasis on the ACS study because it includes a much larger sample size and longer exposure interval, and covers more locations (e.g. 50 cities compared to the Six Cities Study) than other studies of its kind. As explained in the regulatory impact analysis for the Heavy-Duty Engine/Diesel Fuel rule (U.S. EPA, 2000c), more recent EPA benefits analyses have relied on an improved specification of the ACS cohort data that was developed in the HEI reanalysis (Krewski et al., 2000). The particular specification yielded a relative risk based on changes in mean levels of $PM_{2.5}$, as

opposed to the specification in the original study, which reported a relative risk based on median levels[25]. The Krewski et al analysis also includes a broader geographic scope than the original study (63 cities versus 50). Specifically, the relative risk from which the Base estimate is derived is 1.12 per 24.5 $\mu g/m^3$ for all-cause mortality (see: Part II page 173 Table 31 in Krewski et al., 2000). The SAB has recently agreed with EPA's selection of this specification for use in analyzing mortality benefits of PM reductions (U.S. EPA, 2001).

Alternative Estimate: Short-term Studies

To reflect concerns about the more limited number of cohort studies that examine the association between long-term exposure and mortality and the inherent limitations for drawing conclusions regarding causality from these studies, especially the ecological measure of exposure used, a plausible alternative to the base benefit estimate is provided. This estimate was derived from the larger number of time-series studies, the body of which have established a likely causal relationship between short-term measures of PM and daily mortality statistics. A particular strength of the design of these studies for drawing conclusions about causality is the fact that potential confounding variables such as socio-economic status, occupation, and smoking do not vary on a day-to-day basis in an individual area. A number of multi-city and other types of studies strongly suggest that these short term PM exposure-premature mortality relationships cannot be explained by weather, statistical approaches, or other pollutants.

The fact that the PM-mortality coefficients from the cohort studies are far larger than the coefficients derived from the daily time-series studies provides some evidence for an independent chronic effect of PM pollution on health. Indeed, the Base Estimate presumes that the larger coefficients represent a more complete accounting of mortality effects, including both the cumulative total of short-term mortality as well as an additional chronic effect. This is, however, not the only possible interpretation of the disparity. Various reviewers have argued that 1) the long-term estimates may be biased high and/or 2) the short-term estimates may be biased low. In this view, the two study types could be measuring the same underlying relationship.

With respect to possible sources of upward bias in the long-term studies, HEI reviewers have noted that the less robust estimates based on the Six-Cities Study are significantly higher than those based on the more broadly distributed ACS data sets. Some reviewers have also noted that the observed mortality associations from the 1980's and 90's may reflect higher pollution exposures from the 1950's to 1960's. Such an argument is consistent with the dramatic decrease in PM levels over the last 50 years, as long as the relative differences in PM among the cities did not change. Indeed, Pope et al. (2002) demonstrated that the relative differences in pollution levels among the cities was similar between the years 1979-1980 and the years 1999-1980. If the lower PM exposures today pose disproportionately less risk than the exposures of the 1950's-1960's, then the base mortality estimate may be biased upwards. While this would bias estimates based on more recent pollution levels upwards, it also would imply a truly long-term chronic effect of pollution, at least for the higher exposure levels.

With regard to possible sources of downward bias, it is of note that the recent studies suggest that the single day time series studies may understate the short-term effect on the order of a factor of two (Zanobetti et al., 2002). Previous daily mortality studies (Schwartz et al., 1996) examined the impact of

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[25] For policy analysis purposes, functions based on the mean air quality levels may be preferable to functions based on the median air quality levels because changes in the mean more accurately reflect changes in peak values than do changes in the median. Policies which affect peak PM days more than average PM days will result in a larger change in the mean than in the median. In these cases, all else being equal, C-R functions based on median $PM_{2.5}$ will lead to lower estimates of avoided incidences of premature mortality than C-R functions based on mean $PM_{2.5}$.

$PM_{2.5}$ on mortality on a single day or over the average of two or more days. Although the risk estimates from the vast majority of the short-term studies include the effects of only one or two-day exposure to air pollution, more recently, several studies have found that the practice of examining the effects on a single day basis may significantly understate the risk of short-term exposures (Schwartz, 2000a; Zanobetti et al., 2002). These studies suggest that the short-term risk can double when the single-day effects are combined with the cumulative impact of exposures over multiple days to weeks prior to a mortality event. Multi-day models are often referred to as "distributed lag" models because they assume that mortality following a PM event will be distributed over a number of days following or "lagging" the PM event. The size of the effect estimates from these models suggests consistency between the findings of studies that examine premature mortality impacts of short-term and long-term exposures. Additional research may be necessary to confirm this trend.

The United Kingdom's Committee on the Medical Effects of Air Pollution (2001) evaluated the various models proposed by Krewski et al. (2000). In the judgment of the COMEAP, as published in it's "Statement on the Long-term Effects of Particles on Mortality," it is more appropriate to develop a "range of estimates along with comments on their confidence in them" than selecting a single estimate of possible effects. The inclusion of an Alternative Estimate, as well as the sensitivity analyses presented in Appendix 9B is an appropriate response to this suggestion.

These considerations provide a basis for considering an Alternative Estimate using the most recent estimates from the wealth of time-series studies, in addition to the Base Estimate based on the long-term cohort studies. In essence, the Alternative Estimate offers an approach to characterizing some of the uncertainties in the relationship between premature mortality and exposures to ambient levels of fine particles by assuming that there is no mortality effect of chronic exposures to fine particles. Instead, it assumes that the full impact of fine particles on premature mortality is captured using a concentration-response function relating daily mortality to short-term fine particle levels. This will clearly provide a lower bound to the mortality impacts of fine particle exposure, as it omits any additional mortality impacts from longer term exposures.

There are no $PM_{2.5}$ daily mortality studies which report numeric estimates of relative risks from distributed lag models; only PM_{10} studies are available. Daily mortality C-R functions for PM_{10} are consistently lower in magnitude than $PM_{2.5}$-mortality C-R functions, because fine particles are believed to be more closely associated with mortality than the coarse fraction of PM. Given that the emissions reductions under the Nonroad Diesel Engine program result primarily in reduced ambient concentrations of $PM_{2.5}$, use of a PM_{10} based C-R function results in a significant downward bias in the estimated reductions in mortality.

The Alternative Estimate is based on a concentration- response function derived from Schwartz et al. (1996), with an adjustment to account for recent evidence that daily mortality is associated with particle levels from a number of previous days (Schwartz, 2000b). Specifically, to account for the full potential multi-day mortality impact of acute $PM_{2.5}$ events, we use the distributed lag model for PM_{10} reported in Schwartz (2000b) develop an adjustment factor which we then apply to the $PM_{2.5}$ based C-R function reported in Schwartz et al. (1996).

If most of the increase in mortality is expected to be associated with the fine fraction of PM_{10}, then it is reasonable to assume that the same proportional increase in risk would be observed if a distributed lag model were applied to the $PM_{2.5}$ data. The distributed lag adjustment factor is constructed as the ratio of the estimated coefficient from the unconstrained distributed lag model to the estimated coefficient from the single-lag model reported in Schwartz (2000b). The unconstrained distributed lag model coefficient estimate is 0.0012818 and the single-lag model coefficient estimate is 0.0006479. The ratio of these estimates is 1.9784. This adjustment factor is then multiplied by the estimated coefficients

from the Schwartz et al. (1996) study. There are two relevant coefficients from the Schwartz et al. study, one corresponding to all-cause mortality, and one corresponding to chronic obstructive pulmonary disease (COPD) mortality (separation by cause is necessary to implement the life years lost approach detailed below). The adjusted estimates for these two C-R functions are:

All cause mortality = 0.001489 * 1.9784 = 0.002946

COPD mortality = 0.003246 * 1.9784 = 0.006422

Note that these estimates, while approximating the full impact of daily pollution levels on daily death counts, do not capture any impacts of long-term exposure to air pollution. As discussed earlier, EPA's Science Advisory Board, while acknowledging the uncertainties in estimation of a PM-mortality relationship, has repeatedly recommended the use of a study that does reflect the impacts of long-term exposure. This recommendation has been confirmed by the recent NRC (2002) report on estimating health benefits of air pollution regulations. The omission of long-term impacts accounts for approximately a 40 percent reduction in the estimate of avoided premature mortality in the Alternative Estimate relative to the Base Estimate. For comparison, an estimate calculated using the lower confidence interval of the Base estimate C-R function coefficient would fall between these two estimates (i.e., the lower confidence interval on the RR of 1.12 used in the Base Estimate is 1.06, translating to a coefficient estimate of 0.002).[26] In summary, the alternative estimate has a technical foundation in both a plausible interpretation of the cohort studies and the time-series studies that incorporate the longer lag periods.

4.1.2 Valuing Premature Mortality

We used two methods to value avoided premature mortality. In the Base Estimate we used 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 the Alternative Estimate, we take into account that an individual's willingness to pay for mortal risk reduction may depend on his age and remaining life expectancy, using a "statistical life years lost" approach.

Base Estimate: Statistical Lives Lost

Following the advice of the EPA Science Advisory Board (2000a), we calculated the monetary benefits of reducing premature mortality risk using the "value of statistical lives" (VSL) approach, which is a summary measure for the value of small changes in mortality risk experienced by a large number of people. The VSL approach applies information from several published value-of-life studies to determine a reasonable benefit of preventing premature mortality. The unit value of avoiding one statistical death is estimated to be $6.3 million (in 2000 $). This represents an intermediate value from 26 estimates that

[26] The 40% smaller estimate is also consistent with a judgment offered by COMEAP based on its review of the use of U.S. cohort studies in a European context. The alternative estimate of premature mortality is similar in magnitude to their judgement of a "likely" estimate, based on a sensitivity analysis included in the Krewski et al (2000) reanalysis of the ACS cohort data. That sensitivity analysis was based on a much smaller set of cities and included SO2 and other variables as controls. In addition to PM2.5, this specification found a significant impact of SO2 on mortality. SO2 and PM2.5 levels are at least somewhat correlated, so it is not clear in a multi-pollutant specification how much of the SO2 effect is capturing some of the PM2.5 signal and vice versa. An appropriate comparison would be between the COMEAP estimate and the total mortality impact of the nonroad rule predicted using both the PM2.5 and SO2 changes. Comparing PM2.5 related mortality generated from a single pollutant mortality function with a multi-pollutant specification ignores the implied benefits of SO2 reductions under the rule. Thus it is likely that the COMEAP estimate would understate the total mortality impacts likely to be associated with the proposed nonroad rule.

appear in the economics literature, and it is a value EPA has frequently used in RIAs for other rules (e.g., U.S. EPA, 2000c) and in the Section 812 Reports to Congress (U.S. EPA, 1999b).

This estimate is the mean of a Weibull distribution fitted to the estimates from 26 value-of-life studies identified in the Section 812 reports as "applicable to policy analysis." The approach and set of selected studies mirrors that of Viscusi (1992) in his review of value-of-life studies, and uses his same criteria. The $6.3 million estimate is consistent with Viscusi's conclusion (updated to 2000$) that "most of the reasonable estimates of the value of life are clustered in the $3.8 to $8.9 million range."

Exhibit 4-5 presents the 26 studies that we used. 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. As indicated in the previous section on calculating the incidence of avoided premature mortality, we assumed that the incidences of premature mortality related to PM exposures occur in a distributed fashion over the five years following exposure. To take this into account in the valuation of reductions in premature mortality, we applied an annual discount rate to the value of premature mortality occurring in future years.[27] And to allow for some uncertainty regarding the appropriate discount rate, we used rates of three percent and seven percent.

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

Exhibit 4-5. 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	1.4
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: The references for all but Gegax et al. (1985) and V.K. Smith (1983) may be found in Viscusi (1992).

The uncertainty surrounding the estimate of $6.3 million per statistical life is modeled by the Weibull distribution of which this is the mean. As discussed in detail below, there has been considerable discussion about the appropriateness of the 26 value-of-life studies from which the base estimate is derived to the air pollution context. Some of the underlying uncertainty surrounding this estimate is therefore not captured by the Weibull distribution fitted to these 26 values. We have attempted to address this, to some degree, in the alternative estimate.

Alternative Estimates: Age-Adjusted Life Years Lost

The Alternative Estimate reflects the impact of changes to key assumptions associated with the valuation of mortality. These include: 1) the impact of using wage-risk and contingent valuation-based

value of statistical life estimates in valuing risk reductions from air pollution as opposed to contingent valuation-based estimates alone, 2) the use of a value of statistical life years approach as opposed to a VSL approach, and 3) the degree of prematurity (number of life years lost) for mortalities from air pollution.

The Alternative Estimate addresses the first issue by using an estimate of the value of statistical life that is based only on the set of five contingent valuation studies included in the larger set of 26 studies recommended by Viscusi (1992) as applicable to policy analysis. The mean of the five contingent valuation based VSL estimates is $3.7 million (2000$), which is approximately 60 percent of the mean value of the full set of 26 studies. Note that because these are deaths associated with short-term exposures to $PM_{2.5}$, it is assumed that there is no lag between reduced exposure and reduced risk of mortality. Sensitivity analyses exploring the implications for the alternative estimate of using different starting VSL estimates are presented in Appendix 9B.

While the base estimate is based on a VSL approach, the alternative estimate is based on the number of years of life saved and economic value of saving a statistical life year (VSLY). The VSLY approach has been developed in the peer-reviewed economics literature (e.g., Viscusi and Moore, 1989) and has been applied for many years by the U.S. Food and Drug Administration. Some recent analyses, however, have raised concerns about the use of this method to value reductions in premature mortality in an environmental context (e.g., Krupnick, 2002). The VSLY approach applied in this RIA recognizes that each year late in the life span may have a higher monetary value than the average life year saved in the middle of the life span. The non-constant VSLY, rising later in the lifespan, is qualitatively compatible with theoretical economic models of an individual's demand for lifesaving as a function of age (Shepard and Zeckhauser, 1984). The conceptional rationale for a premium on VSLY among the elderly is that they have saved through their working lifetimes and accumulated assets that can be devoted to health protection, and have rising baseline risks, which increase the marginal value of risk reductions (Pratt and Zeckhauser, 1996).

Under the alternative approach, the value of a life year for younger individuals is calculated as if they had an average life expectancy. However, instead of attempting to estimate the remaining life expectancy for different age groups, we have assumed that everyone who dies from exposure to air pollution loses five years of life. Because we assume that younger individuals do not have the accumulated assets or do not adjust the value of life years to reflect reductions in life expectancy, this approach implies that the total value of a five-year loss in life years is greater for the elderly than for younger individuals. An additional limitation of this approach is the discontinuity at age 65. A more complex approach would produce a continuous VSLY curve; however, the empirical data required to specify these models are not available.

There is no latency period assumed in the alternative analysis since the premature deaths are assumed to occur primarily among persons with chronic disease who experience short-term elevations in daily air pollution levels. Even the latency periods associated with the distributed lag models are too short to be of significance in the valuation process.

In order to implement the non-constant VSLY approach, we begin by using a VSL of $3.7 million based on five contingent valuation studies which were also considered as part of the base estimate. This smaller VSL is also consistent with an alternative interpretation of the wage-risk literature (Mrozek and Taylor, 2002). For persons under age 65, the $3.7 million VSL is assumed to reflect an average loss of 35 years. The VSLY associated with $3.7 million VSL is $172,000, annualized using a 3 percent discount rate, or $286,000, annualized using a 7 percent discount rate. Note that the larger discount rate increases the VSLY because at a higher discount rate, a larger stream of VSLY is required to yield a VSL of $3.7 million. For those over age 65, the VSLY is derived from a $3.7 million VSL and an

assumed 10-year life expectancy. This gives a VSLY of $434,000 at a 3 % discount rate of a $527,000 at a 7% discount rate.

The alternative estimate also assumes that deaths from chronic obstructive pulmonary disease (COPD) are advanced by 6 months, and deaths from all other causes are advanced by 5 years. As a first approximation, these reductions in life years lost are applied regardless of the age at death. Actuarial evidence suggests that individuals with serious preexisting cardiovascular conditions have an average remaining life expectancy of around 5 years. While many deaths from daily exposure to PM may occur in individuals with cardiovascular disease, studies have shown relationships between all cause mortality and PM, and between PM and mortality from pneumonia (Schwartz, 2000a). In addition, recent studies have shown a relationship between PM and non-fatal heart attacks, which suggests that some of the deaths due to PM may be due to fatal heart attacks (Peters et al., 2001). And, a recent meta-analysis has shown little effect of age on the relative risk from PM exposure (Stieb et al., 2002). The alternative estimate suggests that the number of deaths in non-elderly populations that 21 percent of non-COPD premature deaths avoided are in populations under 65 (with the possibility for greater loss of life years in this age group). Thus, while the assumption of 5 years of life lost may be appropriate for a subset of total avoided premature mortalities, it may overestimate or underestimate the degree of life shortening attributable to PM for the remaining deaths.

Monetized estimates of the benefits of a reduction in premature mortality are calculated as follows. First, the expected reduction in premature mortality by age category (over 65 and under 65) is multiplied by the assumed gain in additional discounted life years for the two disease categories (COPD and non-COPD) to obtain an estimate of the additional discounted life-years associated with the reduction in PM exposures. No adjustment is made for the quality of life-years saved. The monetized benefit estimate for a reduction in premature mortality, then, is the product of the additional discounted life-years times the calculated VSLY.

Uncertainties Specific to Premature Mortality Valuation

The economic benefits associated with premature mortality are typically the largest category of monetized benefits, as well as the largest contributor to the range of uncertainty in monetized benefits (see U.S. EPA, 1999b, p.107). Because of the uncertainty in estimates of the value of premature mortality avoidance, it is important to adequately characterize and understand the various types of economic approaches available for mortality valuation. Such an assessment also requires an understanding of how alternative valuation approaches reflect the fact that some individuals may be more susceptible to air pollution-induced mortality, or reflect differences in the nature of the risk presented by air pollution relative to the risks studied in the relevant economic literature.

The health science literature on air pollution indicates that several human characteristics affect mortality risks. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. At-risk individuals include those who have suffered strokes or are suffering from cardiovascular disease and angina (Rowlatt et al., 1998).

An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to individuals' WTP to improve their own chances of survival plus WTP to improve other individuals' survival rates. The ideal measure would also take into account the specific nature of the risk reduction, as well as the context in which risk is reduced. To measure the value of a risk reduction, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward, and how individuals value these changes. Each individual's survival curve, or the

probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. Changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This survival curve shift will differ across individuals because survival curves are dependent on such characteristics as age, health state, and the current age to which the individual is likely to survive.

Although a survival curve approach may be the theoretically preferred method for valuing the benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we valued avoided premature mortality risk using the value of statistical life approach in the Base Estimate, supplemented by valuation based on an age-adjusted value of statistical life estimates in the Alternative Estimate.

Some economists emphasize that the value of a statistical life is not a single number relevant for all situations. Indeed, the VSL estimate of $6.3 million (2000 dollars), used in the Base Estimate, is itself the central tendency of a number of estimates of the VSL for some rather narrowly defined populations. Because there are significant differences between the population affected by a particular health risk and the populations used in the labor market studies, as is the case here, some economists prefer to adjust the VSL estimate to reflect those differences. Exhibit 4-7 discusses some of the approaches that have been proposed for valuing reductions in mortality risk.

Exhibit 4-7. Alternative Approaches for Assessing the Value of Reduced Mortality Risk

Stated preference studies – These studies use survey responses to estimate WTP to avoid risks. *Strengths:* flexible approach allowing for appropriate risk context, good data on WTP for individuals. *Weaknesses:* risk information may not be well-understood by respondents and questions may be unfamiliar.

Consumer market studies – These studies use consumer purchases and risk data (e.g., smoke detectors) to estimate WTP to avoid risks. *Strengths:* uses revealed preferences and is a flexible approach. *Weaknesses:* very difficult to estimate both risk and purchase variables.

Value of statistical life year (VSLY) – Provides an annual equivalent to value of statistical life estimates. *Strengths:* provides financially accurate adjustment for age at death. *Weaknesses:* adjustment may not reflect how individuals consider life-years; assumes equal value for all remaining life-years.

Quality adjusted life year – Applies quality of life adjustment to life-extension data, uses cost-effectiveness data to value. *Strengths:* widely used in public health literature to assess private medical interventions. *Weaknesses:* lack of data on health state indices and life quality adjustments that are applicable to an air pollution context. Similar to VSLY, adjustment may not reflect how individuals consider life-years, and typically assumes an equal value for all remaining life-years despite evidence to the contrary.

WTP for a change in survival curve – Reflects WTP for change in risk, potentially incorporates age-specific nature of risk reduction. *Strengths:* theoretically preferred approach that most accurately reflects risk reductions from air pollution control. *Weaknesses:* almost no empirical literature available; difficulty in obtaining reliable values.

WTP for a change in longevity – Uses stated preference approach to generate WTP for longevity or longer life expectancy. *Strengths:* life expectancy is a familiar term to most individuals. *Weaknesses:* does not incorporate age-specific risk information; problems in adapting to air pollution context.

Cost-effectiveness – Determines the implicit cost of saving a life or life-year. *Strengths:* widely used in public health contexts. *Weaknesses:* health context is for private goods, dollar values do not necessarily reflect individual preferences.

In the Base Estimate we estimate the monetary benefits of avoided premature mortality using the results of 26 studies of the value of mortal risks. However, there is considerable uncertainty as to whether the 26 studies on the value of a statistical life provide adequate estimates of the value of a statistical life saved by air pollution reduction. Most of the studies examined differences in wages of risky occupations, using a wage-hedonic approach. The characteristics of the typical hedonic study population, and the risks they face, may cause their average WTP to reduce risk to differ from the population affected by air pollution. The appropriateness of using these 26 studies depends on at least two key factors: the extent to which the risks being valued are similar to the risks of air pollution; and, the extent to which the study population is similar to the population affected by changes in pollution concentrations.

Level of Risk Reduction. 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. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is $50, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $50 for a reduction of 1/100,000 implies a WTP of $500 for a risk reduction of 1/10,000 (which is 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. This assumption has been shown to be reasonable provided the change in the risk being valued is within the range of risks evaluated in the underlying studies (Rowlatt et al., 1998).

Age. Compared with the subjects in wage-risk studies, the population believed to receive the greatest mortality risk reduction associated in this analysis is, on average, older and probably more risk averse. 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. However, the average age of subjects in wage-risk studies, in contrast, is well under 65.

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. However, the direction of bias resulting from the age difference is unclear, particularly because age is confounded by risk aversion (relative to the general population).

The relationship between age and willingness to pay for mortality risk reductions has been the subject of much research over the past several years. Recent research in the U.S. has not found a significant reduction in WTP for risk reductions in older populations (Alberini et al., 2002; Smith et al., forthcoming). Studies outside of the U.S. have found a significant reduction in WTP for older individuals, ranging from 10 percent (Jones-Lee et al., 1993) to around 35 percent (Alberini et al., 2002) for a 70 year old, relative to a 40 year old. Around 80 percent of the deaths projected to be avoided from reduced exposure to PM in 2020 and 2030 are in populations over 65. As such, the assumption that populations of all ages have the same VSL can have a significant impact on the total benefits.

The age-WTP relationship based on Jones-Lee (1989), as summarized in U.S. EPA (U.S. EPA, 2000d), suggests a steep inverted U shape between age and VSL, with the VSL for a 70 year old at 63 percent of that for a 40 year old, and the VSL for an 85 year old at 7 percent of that for 40 year old. The relationship based on Jones-Lee (1993) and summarized in U.S. EPA (2000d), suggests a much flatter inverted U shape, with the VSL for a 70 year old at 92 percent of that for a 40 year old, and the VSL for an 85 year old at 82 percent of that for a 40 year old. Recent analyses conducted in Canada and the U.S. by Krupnick et al. (Krupnick et al., 2002) found mixed results. The Canadian analysis found around a 35

percent reduction in VSL for respondents over age 70, but the U.S. analysis found no significant differences in VSL across ages. The wide range of age-adjustment ratios, especially at older ages demonstrates the difficulty in making these kinds of adjustments. We note that our Base estimate is the most consistent with current evidence on U.S. preferences for risk reduction in older populations.

Risk Aversion. There is 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.

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.

Income. 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.

Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. 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.

As real income increases the WTP for environmental improvements also increases. While many analyses assume that the income elasticity of WTP is unit elastic (i.e., ten percent higher real income level implies a ten percent higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income. 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.

The effects of real income changes on WTP estimates can influence benefit estimates in two different ways: (1) through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and (2) through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the SAB advised EPA to adjust WTP for increases in real income over time, but not to adjust WTP to account for cross-sectional income differences "because of the sensitivity of making such distinctions, and because of insufficient evidence available at present" (U.S. EPA, 2000a).

Voluntary versus Involuntary Risk. 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.

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

The potential sources of bias in an estimate of mean WTP to reduce the risk of air pollution related mortality based on wage-risk studies are summarized in Exhibit 4-8. Although most of the individual factors tend to have a downward bias, the overall effect of these biases is unclear.

Exhibit 4-8. 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
Level of Risk	None
Age	Uncertain, perhaps overestimate
Attitudes toward risk	Underestimate
Income	Uncertain
Voluntary versus Involuntary	Uncertain, perhaps underestimate
Catastrophic versus Protracted Death	Uncertain, perhaps underestimate

4.1.3 Sensitivity Calculations for Premature Mortality

We conducted a variety of sensitivity calculations regarding premature mortality. We considered the impact of alternative C-R examining the relationship between long-term $PM_{2.5}$ exposure and premature mortality. We considered alternative lag structures. And, we considered the potential impact of an effects threshold. In addition, we estimated ozone-related mortality, and the impact of PM on infant mortality.

Sensitivity Calculation: Alternative C-R Functions for Long-Term $PM_{2.5}$-Related Premature Mortality

Although we use the Krewski et al. (2000) mean-based all-cause model exclusively to derive our Base Estimate of avoided premature mortality, we also examined the impacts of selecting alternative C-R functions for premature mortality. Exhibit 4-9 summarizes the alternative C-R functions that we used from the original ACS study by Pope et al. (1995) and from the "Harvard Six-City Study" by Dockery et al. (1993), as well as the recent reanalyses by Krewski et al. (2000) and Pope et al. (2002).

Exhibit 4-9. Alternative C-R Functions for Long-Term $PM_{2.5}$-Related Premature Mortality

Study	Mortality Category	Age	Pollutant	Metric
Pope et al. (2002)	All-cause	30+	$PM_{2.5}$	Annual Mean
Krewski et al. (2000) reanalysis of Pope et al. (1995)	All-cause	30+	$PM_{2.5}$	Annual Median
Krewski et al. (2000) reanalysis of Pope et al. (1995). Random effects, independent cities	All-cause	30+	$PM_{2.5}$	Annual Median
Krewski et al. (2000) reanalysis of Pope et al. (1995). Random effects, regional adjustment	All-cause	30+	$PM_{2.5}$	Annual Median
Pope et al. (2002)	All-cause	30+	$PM_{2.5}$	Annual Median
Krewski et al. (2000) reanalysis of Dockery et al. (1993)	All-cause	25+	$PM_{2.5}$	Annual Mean
Dockery et al. (1993)	All-cause	25+	$PM_{2.5}$	Annual Mean
Pope et al. (2002)	Cardiopulmonary	30+	$PM_{2.5}$	Annual Mean
Pope et al. (2002)	Lung Cancer	30+	$PM_{2.5}$	Annual Mean

The Krewski et al. (2000) reanalysis provides results for several models which control for spatial correlations in the data. These models are based on the original ACS air quality dataset, which contained only median $PM_{2.5}$ concentrations. Ideally, the 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 base analysis and to use the median-based regionally adjusted relative risks to provide alternative estimates exploring the impact of adjustments for spatial correlations.

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, 1999e, p. 10).

Some of the functions are based on changes in mean $PM_{2.5}$ concentrations while others are based on median $PM_{2.5}$ concentrations. Estimated reductions in premature mortality will depend on both the size of the C-R coefficient and the change in the relevant $PM_{2.5}$ metric (mean or median). 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.

In addition, we included a recent study by Pope et al. (2002) that extends the original analysis by Pope et al. (1995) in a number of significant ways. Pope et al. (2002) had fifteen years of cohort data, as opposed to the eight years of data in the original work, and they used three different sets of years to measure mean $PM_{2.5}$ levels, as opposed to a single measure. The new set of results confirm the results of the earlier studies. In addition, the new set of results includes relative risk estimates for lung cancer and cardiopulmonary causes of death, in addition to all cause mortality.

Sensitivity Calculation: Mortality Lag Structure

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, 1999d, 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.

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 three alternative lag structures. Lag 1 will apportion the occurrence of all incidence to the first year. Valuation of these cases will not be discounted. Lag 2, 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. And, lag 3, based on the length of the study period for the Dockery et al. (1993) study, assigns 100 percent of the occurrence to the fifteenth year out from the modeled future-year. Exhibit 4-10 summarizes these lag structures.

Exhibit 4-10. Mortality Lag Structures in Base and Sensitivity Analyses

Structure	Year														
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*
Base	25	25	16.7	16.7	16.7	0	0	0	0	0	0	0	0	0	0
Sensitivity 1	100	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Sensitivity 2	0	0	0	0	0	0	0	100	0	0	0	0	0	0	0
Sensitivity 3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	100

Sensitivity Calculation: $PM_{2.5}$ Effects Thresholds

The consistent advice from EPA's Science Advisory Board has been to model premature mortality associated with PM exposure as a non-threshold effect, with harmful effects to exposed populations regardless of the absolute level of ambient PM concentrations. Nevertheless, some analysts (Smith et al., 2000) have hypothesized the presence of a threshold relationship. The nature of the hypothesized relationship is that there might exist a PM concentration level below which further reductions no longer yield premature mortality reduction benefits. EPA does not necessarily endorse any particular threshold. Nonetheless, the sensitivity analysis illustrates how our estimates of the number of premature mortalities in the Base Estimate might change under a range of alternative assumptions for a PM mortality threshold.

One important assumption that we adopted for the threshold sensitivity analysis is that no adjustments are made to the shape of the C-R function above the assumed threshold. Instead, thresholds were applied by simply assuming that any changes in ambient concentrations below the assumed threshold have no impacts on the incidence of premature mortality. If there were actually a threshold, then the shape of the C-R function would likely change and there would be no health benefits to reductions in PM below the threshold.

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 five $\mu g/m^3$ increments from no threshold to a 25 $\mu g/m^3$. Appendix A presents the results of this sensitivity analysis.

Sensitivity Calculation: Ozone-Related Mortality

Both ozone and PM have been associated with increased risk of premature mortality, which is an important health endpoint in this economic analysis due to the high monetary value associated with risks to life. However, to avoid any double-counting of estimated effects, we included only PM-related mortality in the base analysis, and estimated ozone-related mortality as a sensitivity analysis. Using a random/fixed-effects weighting procedure, we pooled the incidence estimates of four U.S. ozone-related mortality studies -- Ito and Thurston (1996), Kinney et al. (1995), Moolgavkar et al. (1995), and Samet et al. (1997). Appendix A presents the results of this analysis.

Sensitivity Calculation: Infant Mortality

Woodruff et al. (1997) associated changes in annual PM_{10} levels with changes in post-neonatal mortality of infants aged 28 to 364 days. Conceptually, any additional mortality from this function could be added to the premature mortality predicted by Krewski et al. (2000), because the Krewski et al. reanalysis of the Pope et al. (1995) function covers only the population over 29 years old. The EPA Clean Air Council, in an advisory issued for the §812 Prospective Analysis, recommended that this endpoint not be included because it was a new endpoint that had not been replicated in other studies in the U.S. (U.S. EPA, 1999a, p. 12). The Council deemed that the coherence and consistency arguments which support the use of other studies are not present with this study. For this reason, the results for this endpoint are presented as a supplemental calculation to the base analysis.

4.2 Chronic Illness

Researchers have linked air pollution with a variety of adverse health effects that have long-term, or chronic implications. The onset of bronchitis and asthma have both been associated with exposure to air pollutants. Studies have linked the onset of chronic bronchitis in adults to particulate matter (Schwartz, 1993; Abbey et al., 1995b); one study has linked the onset of chronic asthma in adults to ozone (McDonnell et al., 1999); and, recent work has linked ozone and outdoor exercise with the development of asthma in children (McConnell et al., 2002). 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 addition, recent work by Peters et al. (2001) suggests that short-term levels of $PM_{2.5}$ can trigger the onset of acute myocardial infarction. The impacts can be long lasting, and in a sense chronic. Work by Wittels et al. (1990), Eisenstein et al. (2001), and Russell et al. (1998) indicates that the health costs of myocardial infarction can stretch over several years, as periodic treatment is necessary.

4.2.1 Chronic Bronchitis

Chronic bronchitis is characterized by mucus in the lungs and a persistent wet cough for at least three months a year for several years in a row, and affects roughly five percent of the U.S. population (American Lung Association, 2002b, Table 4). There are a limited number of studies that have estimated the impact of air pollution on new incidences of chronic bronchitis. Schwartz (1993) and Abbey et al.(1995c) provide evidence that long-term PM exposure gives rise to the development of chronic bronchitis in the U.S. Because the nonroad standards are expected to reduce primarily $PM_{2.5}$, this analysis uses only the Abbey et al (1995c) study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of chronic bronchitis.

Base Estimate: 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, the SAB (U.S. EPA, 1999c) has indicated that the severity-adjusted values from this study provide reasonable estimates of the WTP for avoidance of 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 chronic bronchitis 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 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:

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

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 $)[28]; the median response implied a WTP of about $699,000 (2000 $). However, the extreme tails of distributions of WTP responses are usually considered 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 (1992)).

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 $340,482. This WTP estimate is reasonably consistent with full COI estimates derived for chronic bronchitis (see below).

Alternative Estimate: Valuing Chronic Bronchitis

For the Alternative Estimate, we used cost-of illness values based on average annual lost earnings and average annual medical expenditures reported by Cropper and Krupnick (1990). This study estimated annual lost earnings resulting from chronic bronchitis as a function of age at onset of the illness, for the following age categories: 25-43, 35-44, 45-54, and 55-65 (see Cropper and Krupnick (1990), Table 8). Annual medical expenses were estimated for 10-years age groups (0-9, 10-19, 20-29, ..., 80-89). We derived estimates of the present discounted value of the stream of medical and opportunity costs for people whose age of onset is 30, 40, 50, 60, 70, and 80. Medical costs (which are in 1977$ in the Cropper and Krupnick study) were inflated to 2000$ using the CPI-U for medical care; lost earnings (opportunity costs) were inflated to 2000$ using the CPI-U "all items." Life expectancies were assumed to be unaffected by the illness.[29] For example, an individual at age 70 has a life expectancy of 14.3 more years, and we assumed that someone whose age of onset of chronic bronchitis is 70 will also live for 14.3 more

[28] 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.

[29] Source of life expectancies: National Vital Statistics Reports, Volume 47, No. 19, June 30, 1999. Table 5: "Life expectancy at selected ages by race and sex: United States, 1997.

years. We also assumed that opportunity costs at ages 66 and over were zero. Present discounted values were calculated using three and seven percent discount rates.

For each of the two discount rates, we derived cost of illness unit values for chronic bronchitis for the following age categories: 27-44, 45-64, and 65+, corresponding to the age categories that were used in the epidemiological study that estimated a concentration-response function for chronic bronchitis. The estimate for the 27-44 age group is an average of the present discounted values calculated for ages 30 and 40; the estimate for the 45-64 age category is an average of the present discounted values calculated for ages 50 and 60; and the estimate for the 65+ age category is an average of the present discounted values calculated for ages 70 and 80. Exhibit 4-11 presents the resulting cost of illness estimates.

Exhibit 4-11. Alternative Estimate: Cost-of-Illness for Chronic Bronchitis

Age	Discount Rate	Value [a] (2000 $)	Description
27-44	3%	$150,542	Cropper and Krupnick (1990). Including both medical costs and opportunity cost from age of onset to expected age of death (assuming that chronic bronchitis does not change life expectancy). Opportunity cost adjusted from 1977$ to 2000$ using CPI-U "all items." Medical cost adjusted from 1977$ to 2000$ using CPI-U for medical care.
	7%	$86,026	
45-64	3%	$97,610	
	7%	$72,261	
65+	3%	$11,088	
	7%	$9,030	

[a] This table gives present values of future-year costs. The greater the discount rate applied to a given future-year cost, the smaller the present discounted value of that future-year cost. Therefore, present values derived using a 3% discount rate are larger than present values derived using a 7% discount rate, when both discount rates are applied to the same future-year cost.

Sensitivity Calculation: Chronic Bronchitis Reversals

In developing the C-R function 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.[30] 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 Reversals

In valuing chronic bronchitis reversals, we use the same method used to value cases of chronic bronchitis. However, instead of allowing the severity level to range from one to 12, we value all reversals at a severity level of one.

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

4.2.2 Non-Fatal Myocardial Infarction (Heart Attack)

Non-fatal heart attacks have been linked with short term exposures to $PM_{2.5}$ in the U.S. (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We used a recent study by Peters et al. as the basis for the C-R function estimating the relationship between $PM_{2.5}$ and non-fatal heart attacks. It is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar et al. (2000a) reported a consistent relationship between all cardiovascular hospital admissions, including for non-fatal heart attacks, and PM. However, they did not focus specifically on heart attacks. Given the lasting impact of a heart attack on longer-term health costs and earnings, we chose to provide a separate estimate for non-fatal heart attacks based on the single available U.S. C-R function.

The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the U.S. These studies provide a weight of evidence for this type of effect. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Lack of heart rate variability is a risk factor for heart attacks and other coronary heart diseases (Tsuji et al., 1996; Liao et al., 1997; Dekker et al., 2000). As such, the reduction in heart rate variability due to PM is consistent with an increased risk of heart attacks.

Valuing Non-Fatal Myocardial Infarction (Heart Attack)

EPA has not previously estimated the impact of its programs on reductions in the expected number of non-fatal heart attacks, although it has examined the impact of reductions in other related cardiovascular endpoints. We were not able to identify a suitable WTP value for reductions in the risk of non-fatal heart attacks. Instead, we have used a cost-of-illness unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with a heart attack extend beyond the initial event itself, we considered costs incurred over several years. For opportunity costs, we used values derived from Cropper and Krupnick (1990), originally used in the 812 Retrospective Analysis of the Clean Air Act (U.S. EPA, 1997b). For the direct medical costs, we found three possible sources in the literature.

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

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

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

As seen in Exhibit 4-12, the three different studies provided significantly different values. We have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used a simple average of the two estimates for medical costs that similarly cover a 5-year period, or $62,495. We added this to the 5-year opportunity cost estimate. Exhibit 4-13 gives the resulting estimates. We currently do not have adequate information to characterize the uncertainty surrounding any of these estimates.

Exhibit 4-12. Summary of Studies Valuing Reduced Incidences of Myocardial Infarction

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

[a] Wittels et al. did not appear to discount costs incurred in future years. The values for the other two studies are based on a three percent discount rate.

Exhibit 4-13. Estimated Costs Over a 5-Year Period of a Non-Fatal Myocardial Infarction

Age Group	Opportunity Cost [a]		Medical Cost [b]		Total Cost	
	3% Discount Rate	*7% Discount Rate*	*3% Discount Rate*	*7% Discount Rate*	*3% Discount Rate*	*7% Discount Rate*
0 - 24	$0	$0	$65,902	$65,293	$65,902	$65,293
25-44	$8,774	$7,855	$65,902	$65,293	$74,676	$73,149
45 - 54	$12,932	$11,578	$65,902	$65,293	$78,834	$76,871
55 - 65	$74,746	$66,920	$65,902	$65,293	$140,649	$132,214
> 65	$0	$0	$65,902	$65,293	$65,902	$65,293

[a] From Cropper and Krupnick (1990). Present discounted value of 5 yrs of lost earnings, at 3% and 7% discount rate, adjusted from 1977$ to 2000$ using CPI-U "all items".

[b] An average of the 5-year costs estimated by Wittels et al. (1990) and Russell et al. (1998). Note that Wittels et al. appears not to have used discounting in deriving a 5-year cost of $109,474; Russell et al. estimated first-year direct medical costs and annual costs thereafter. The resulting 5-year cost is $22,331, using a 3% discount rate, and $21,113, using a 7% discount rate. Medical costs were inflated to 2000$ using CPI-U for medical care.

4.2.3 Sensitivity Calculation: Chronic Asthma

In a number of studies ozone, PM, and even CO have been linked to acute asthmatic complaints (e.g., Whittemore and Korn, 1980; Ostro et al., 1995; Sheppard et al., 1999), however there is more limited evidence regarding the link between air pollution and the development of asthma. The best evidence points to ozone. Abbey et al. (1991; 1993) reported a significant link between ozone and the development of asthma, and Portney and Mullahy (1990) found ozone linked to sinusitis and hay fever, and McConnell et al. (2002) reported that ozone exposure and outdoor exercise is associated is with the development of asthma in children. A review of research data by the EPA (1996b, p. 9-35) concluded that prolonged ozone exposure causes structural changes in several regions of the respiratory tract, and the available epidemiological studies are suggestive of a link between chronic health effects in humans and long-term ozone exposure. A cohort study by McDonnell et al. (1999) carefully measured ozone exposure over 15 years, and found ozone exposure was linked to the onset of asthma in adult males.

The McDonnell et al. (1999) study used the same cohort of Seventh-Day Adventists as Abbey et al. (1991; 1993), and examined the association between air pollution and the onset of asthma in adults between 1977 and 1992. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures, controlling for other covariates; no significant effect was found between ozone exposure and asthma in females. No significant effect was reported for females or males due to exposure to PM, NO_2, SO_2, or SO_4.

Some questions have been raised about the statistical validity of the associations found in this study and the appropriateness of transferring the estimated C-R function from the study populations (white, non-Hispanic males) to other male populations (i.e. African-American males). Some of these concerns include the following: 1) no significant association was observed for female study participants also exposed to ozone; 2) the estimated C-R function is based on a cross-sectional comparison of ozone levels, rather than incorporating information on ozone levels over time; 3) information on the accuracy of self-reported incidence of chronic asthma was collected but not used in estimating the C-R function; 4) the study may not be representative of the general population because it included only those individuals living 10 years or longer within 5 miles of their residence at the time of the study; and 5) the study had a significant number of study participants drop out, either through death, loss of contact, or failure to provide complete or consistent information. While these issues may result in increased uncertainty about this effect, however, none can be identified with a specific directional bias in the estimates. In addition, the SAB reviewed the study and deemed it appropriate for quantification of changes in ozone concentrations in benefits analyses (U.S. EPA, 1999d, p. 6). However, because of the sources of uncertainty associated with this study, we include it only as a sensitivity calculation.

Valuing Chronic Asthma

Two studies have estimated WTP to avoid chronic asthma in adults. Blumenschein and Johannesson (1998) used two different contingent valuation (CV) methods, the dichotomous choice method and a bidding game, to estimate mean willingness to pay for a cure for asthma. The mean WTP elicited from the bidding game was $189 per month, or $2,268 per year (in 1996$). The mean WTP elicited from the dichotomous choice approach was $343 per month, or $4,116 per year (in 1996$). Using $2,268 per year, a five percent discount rate, and 1997 life expectancies for males in the United States (National Center for Health Statistics, 1999, Table 5), the present discounted value of the stream of annual WTPs is $37,735 (in 2000 $).

O'Conor and Blomquist (1997) estimated WTP to avoid chronic asthma from estimates of risk-risk tradeoffs. Combining the risk-risk tradeoffs with a statistical value of life, the annual value of

avoiding asthma can be derived. Assuming a value of a statistical life of $6 million, they derived an annual WTP to avoid asthma of $1500 (O'Connor and Blomquist, 1997, p. 677). For a value of a statistical life of $5,894,400 (in 1997 $), the corresponding implied annual value of avoiding chronic asthma, based on O'Conor and Blomquist would be $1,474. Assuming a five percent discount rate and 1997 life expectancies for males in the United States, the present discounted value of the stream of annual WTPs would be about $23,968 (in 2000 $).[31]

We estimated the value of avoiding a case of chronic asthma to be $30,852, the average of the two study-specific values. The uncertainty surrounding the WTP to avoid a case of chronic asthma among adult males was characterized by a triangular distribution on the range determined by the two study-specific WTP estimates, [$23,968, $37,735], centered at $30,852.

4.3 Hospital Admissions and Emergency Room Visits

Respiratory and cardiovascular hospital admissions are the two broad categories of hospital admissions that have been related to exposure to both PM and ozone. We estimated the impact of ozone and PM on respiratory and cardiovascular hospital admissions. Although the benefits associated with respiratory and cardiovascular hospital admissions are estimated separately in the analysis, the methods used to estimate changes in incidence and to value those changes are the same for both broad categories of hospital admissions. In addition to respiratory- and cardiovascular-related hospital admissions, we estimated asthma-related emergency room (ER) visits associated with PM and ozone.

4.3.1 Respiratory and Cardiovascular Hospital Admissions

Due to the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, we pooled some of the hospital admission endpoints, using the results of a number of studies. Exhibit 4-14 summarizes the studies that we used.

[31] Because chronic asthma is simply an alternative calculation, we present a single estimate based on a five percent discount rate, rather than present separate estimates based on three percent and seven percent discount rates.

Exhibit 4-14. Ozone and PM-Related Hospital Admission Studies

Hospitalization Type	Location	ICD-9 Codes	Study	Age of Study Population
$PM_{2.5}$-Related Hospital Admissions				
COPD, 65+	Cook, Los Angeles, and Mariposa counties	490-496	Moolgavkar (2000b)	65+
	Urban U.S.	490-496	Lippman et al. (2000)	65+
COPD, 20-64	Cook, Los Angeles, and Mariposa counties	490-496	Moolgavkar (2000b)	20-64
Pneumonia, 65+	Urban U.S.	480-487	Lippman et al. (2000)	65+
Asthma, <65	Seattle, WA	493	Sheppard, et al. (1999)	<65
Cardiovascular, 65+	Cook, Los Angeles, and Mariposa counties	390-459	Moolgavkar (2000a)	65+
	Urban U.S.	410-414, 427-428	Lippman et al. (2000)	65+
Cardiovascular, 20-64	Cook, Los Angeles, and Mariposa counties	390-459	Moolgavkar (2000a)	20-64
Ozone-Related Hospital Admissions				
All-Respiratory, 65+	New Haven, CT; and Tacoma, WA	460-519	Schwartz (1995)	65+
	Detroit, MI	480-487, 491-492, 494-496	Schwartz (1994b)	65+
	Minneapolis, MN	480-487	Schwartz (1994a)	65+
	Minneapolis, MN	480-487, 490-496	Moolgavkar et al (1997)	65+
	Toronto, CAN	464, 466, 480-487, 490-496	Burnett et al. (2001)	<2

To estimate avoided incidences of cardiovascular hospital admissions associated with $PM_{2.5}$, we use studies by Moolgavkar (2000a) and Lippmann et al. (2000). There are additional published studies showing a statistically significant relationship between PM_{10} and cardiovascular hospital admissions. However, given that the control option we are analyzing is expected to reduce primarily $PM_{2.5}$, we have chosen to focus on the two studies focusing on $PM_{2.5}$. Both of these studies estimated a C-R function for populations over 65, allowing us to pool the C-R functions for this age group. Only Moolgavkar estimated a separate C-R function for populations 20 to 64. Total cardiovascular hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. In order to avoid double counting benefits from reductions in MI when applying the C-R function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarction.

To estimate total avoided incidences of respiratory hospital admissions, we use C-R functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, there are additional published studies showing a statistically significant relationship between PM_{10} and respiratory hospital admissions. We use only those focusing on $PM_{2.5}$. Both Moolgavkar (2000a) and Lippmann et al (2000) estimated C-R functions for COPD in populations over 65, allowing us to pool the C-R functions for this group. Only Moolgavkar estimated a

separate C-R function for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Lippmann et al estimated pneumonia, and only for the population 65 and older. In addition, Sheppard et al (1999) estimated a C-R function for asthma hospital admissions for populations under age 65. Total avoided incidences of PM-related respiratory-related hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

To estimate avoided incidences of respiratory hospital admissions associated with ozone, we use a number of studies examining hospital admissions for a range of respiratory illnesses, including pneumonia and COPD. Two age groups, adults over 65 and children under 2, are examined. For adults over 65, Schwartz (1995) provides C-R functions for 2 different cities relating ozone and hospital admissions for all respiratory causes (defined as ICD codes 460-519). Two studies (Schwartz, 1994a; Moolgavkar et al., 1997) examined the link between ozone and both pneumonia and COPD hospital admissions in Minneapolis. However, Schwartz (1994a) found no significant relationship between ozone and COPD, and did not report a coefficient. One additional study (Schwartz, 1994b) examined ozone and both pneumonia and COPD hospital admissions in Detroit. Burnett et al. (2001), is the only study providing a C-R function for respiratory hospital admissions in children under two.

To estimate total respiratory hospital admissions due to ozone for adults over 65, we first summed the separate effects categories calculated by each study for each city. We summed together the pneumonia and COPD estimates reported for Detroit by Schwartz (1994b) in order to get an estimate of all-respiratory hospital admissions. Similarly, we summed the Minneapolis from the C-R functions based on the work by Moolgavkar et al (1997). And, we simply used the pneumonia estimate reported by Schwartz (1994a) as an estimate all-respiratory hospital admissions. We then used a fixed/random effects pooling procedure to combine the two all-respiratory hospital admission estimates for Minneapolis. Finally, we used fixed/random effects pooling to combine the results for Minneapolis, Detroit, Tacoma, and New Haven.

4.3.2 Valuing Respiratory and Cardiovascular Hospital Admissions

Society's WTP to avoid a hospital admission includes medical expenses, lost work productivity, the non-market costs of treating illness (i.e., air, water and solid waste pollution from hospitals and the pharmaceutical industry), as well as WTP of the affected individual, as well as of that of relatives, friends, and associated caregivers, to avoid the pain and suffering.[32]

Because medical expenditures are to a significant extent shared by society, via medical insurance, Medicare, etc., the medical expenditures actually incurred by the individual are likely to be less than the total medical cost to society. The total value to society of an individual's avoidance of hospital admission, then, might be thought of as having two components: (1) the cost of illness (COI) to society, including the total medical costs plus the value of the lost productivity, as well as (2) the WTP of the individual, as well as that of others, to avoid the pain and suffering resulting from the illness.

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[32] Some people take action to avert the negative impacts of pollution. While the costs of successful averting behavior should be added to the sum of the health-endpoint-specific costs when estimating the total costs of pollution, these costs are not associated with any single health endpoint It is possible that in some cases the averting action was not successful, in which case it might be argued that the cost of the averting behavior should be added to the other costs listed (for example, it might be the case that an individual incurs the costs of averting behavior and in addition incurs the costs of the illness that the averting behavior was intended to avoid). Because averting behavior is generally not taken to avoid a particular health problem (such as a hospital admission for respiratory illness), but instead is taken to avoid the entire collection of adverse effects of pollution, it does not seem reasonable to ascribe the entire costs of averting behavior to any single health endpoint. However, omission of these averting behavior costs will tend to bias the estimates downward.

In the absence of estimates of social WTP to avoid hospital admissions for specific illnesses (components 1 plus 2 above), estimates of total COI (component 1) are typically used as conservative (lower bound) estimates. Because these estimates do not include the value of avoiding the pain and suffering resulting from the illness (component 2), they are biased downward. Some analyses adjust COI estimates upward by multiplying by an estimate of the ratio of WTP to COI, to better approximate total WTP. Other analyses have avoided making this adjustment because of the possibility of over-adjusting -- that is, possibly replacing a known downward bias with an upward bias. The COI values used in this benefits analysis will not be adjusted to better reflect the total WTP.

Following the method used in the §812 analysis (U.S. EPA, 1999b), ICD-code-specific COI estimates used in our analysis consist of two components: estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). The opportunity cost of a day spent in the hospital is estimated as the value of the lost daily wage, regardless of whether or not the individual is in the workforce. This was estimated as the median weekly wage in 1990, divided by 5, and inflated to year 2000$ using the CPI-U "all items." The resulting estimate is $109.35.

For all hospital admissions included in this analysis, estimates of hospital charges and lengths of hospital stays were based on statistics provided by the Agency for Healthcare Research and Quality's Healthcare Utilization Project (2000). The total COI for an ICD-code-specific hospital stay lasting n days, then, would be estimated as the mean hospital charge plus $109.35*n. Most respiratory hospital admissions categories considered in epidemiological studies consisted of sets of ICD codes. The unit dollar value for the set of ICD codes was estimated as the weighted average of the ICD-code-specific values (mean hospital charges plus opportunity costs, based on length of stay) of each ICD code in the set. The weights were the relative frequencies of the ICD codes among hospital discharges in the United States, as estimated by the National Hospital Discharge Survey (Owings and Lawrence, 1999, Table 1). Exhibit 4-15 shows the unit values thus derived for valuing respiratory and cardiovascular hospital admissions.

The mean hospital charges and mean lengths of stay provided by (AHRQ 2000) are based on a very large nationally representative sample of about seven million hospital discharges, and are therefore the best estimates of mean hospital charges and mean lengths of stay available. We were not able to estimate the uncertainty surrounding cost-of-illness estimates for hospital admissions because 1993 was the last year for which standard errors of estimates of mean hospital charges were reported . However, the standard errors reported in 1993 were very small because estimates of mean hospital charges were based on large sample sizes, and the overall sample size in 1997 was about ten times as large as that in 1993 (at about seven million hospital discharges in all). The standard errors of the current estimates of mean hospital charges will therefore be negligible. Therefore, although we cannot include the uncertainty surrounding these cost-of-illness estimates in our overall uncertainty analysis, the omission of this component of uncertainty will have virtually no impact on the overall characterization of uncertainty.

Because of distortions in the market for medical services, the hospital charge may exceed "the cost of a hospital stay." We use the example of a hospital visit to illustrate the problem. Suppose a patient is admitted to the hospital to be treated for an asthma episode. The patient's stay in the hospital (including the treatments received) costs the hospital a certain amount. This is the hospital cost – i.e., the short-term expenditures of the hospital to provide the medical services that were provided to the patient during his hospital stay. The hospital then charges the payer a certain amount – the hospital charge. If the hospital wants to make a profit, is trying to cover costs that are not associated with any one particular patient admission (e.g., uninsured patient services), and/or has capital expenses (building expansion or renovation) or other long term costs, it may charge an amount that exceeds the patient-specific short term costs of providing services. The payer (e.g., the health maintenance organization or other health insurer)

pays the hospital a certain amount – the payment – for the services provided to the patient. The less incentive the payer has to keep costs down, the closer the payment will be to the charge. If, however, the payer has an incentive to keep costs down, the payment may be substantially less than the charge; it may still, however, exceed the short-term cost for services to the individual patient.

Although the hospital charge may exceed the short-term cost to the hospital of providing the medical services required during a patient's hospital stay, cost of illness estimates based on hospital charges are still likely to understate the total social WTP to avoid the hospitalization in the first place, because the omitted WTP to avoid the pain and suffering is likely to be quite large.

Exhibit 4-15. Unit Values for Respiratory and Cardiovascular Hospital Admissions

Hospital Admission Category	ICD-9 Codes	Age Range	COI [a] (2000 $)
all respiratory	460-519	65+	$18,353
		0 - 2	$7,741
pneumonia	480-487	65+	$17,802
COPD	490-492, 494-496	65+	$13,615
		20-64	$11,794
asthma	493	<65	$7,771
all cardiovascular	390-429	65+	$21,162
		20-64	$22,754
ischemic heart disease	410-414	65+	$25,848
dysrhythmias	427	65+	$15,698
congestive heart failure	428	65+	$15,185

[a] The unit value for a group of ICD-9 codes is the weighted average of ICD-9 code-specific values, from AHRQ (2000). The weights are the relative frequencies of hospital discharges for each ICD-9 code in the group (Owings and Lawrence, 1999, Table 1).

4.3.3 Asthma-Related Emergency Room Visits

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

Initially we were concerned about double-counting the benefits from reducing both hospital admissions and ER visits. However, our estimates of hospital admission costs do not include the costs of admission to the ER, so we can safely estimate both hospital admissions and ER visits.

Exhibit 4-16 lists the four C-R functions that we used to estimate the effects of exposure to PM

and ozone on asthma-related ER visits. For ozone-related asthma ER visits, we use three epidemiological studies: Cody et al. (1992), Weisel et al. (1995), and Stieb et al. (1996). Working in central New Jersey, Cody et al. examined asthma-related ER visits over a 16 month period between May, 1988 and August, 1989, and found that ozone was linked to asthma-related ER visits. No significant effect was seen for PM_{10} or SO_2. Using a one-pollutant model, Weisel et al. also found ozone linked to asthma-related ER visits in an all-age 1990 population for eight New Jersey counties. Stieb et al. (1996) examined the relationship between ER visits and air pollution for persons of all ages in St. John, New Brunswick, Canada, from May through September in 1984-1992. Ozone was significantly linked to ER visits, especially when ozone levels exceeded 75 ppb. Schwartz et al. (1993) failed to find a significant relationship between asthma-related ER visits and ozone. In this study of older Seattle residents, Schwartz et al. instead found PM_{10} to be significantly related to asthma-related ER visits.

Exhibit 4-16. Asthma-Related Emergency Room Visit Studies

Location	Study	Pollutants Used in Final Model	Study Population
central and northern NJ	Cody et al. (1992)	O_3	all ages
central and northern NJ	Weisel et al. (1995)	O_3	all ages
New Brunswick, Canada	Stieb et al. (1996)	O_3	all ages
Seattle, WA	Schwartz et al. (1993)	PM_{10}	<65

Valuing Asthma-Related Emergency Room (ER) Visits

To value asthma emergency room (ER) visits, we used a simple average of two estimates from the literature. The first estimate comes from Smith et al. (1997), who reported that there were approximately 1.2 million asthma-related ER visits made in 1987, at a total cost of $186.5 million, in 1987$. The average cost per visit was therefore $155 in 1987$, or $311.55 in 2000 $ (using the CPI-U for medical care to adjust to 2000 $). The second is from Stanford et al. (1999), who examined data from asthmatics from 1996-1997, and reported an average cost of $260.67. A simple average of the two estimates yields a (rounded) unit value of $286.

In comparing their study to Smith et al. (1997), Stanford et al. (1999) noted that the data used by Smith et al., "may not reflect changes in treatment patterns during the 1990s." In addition, its costs are the costs to the hospital (or ER) for treating asthma rather than charges or payments by the patient and/or third party payer. Costs to the ER are probably a better measure of the value of the medical resources used up on an asthma ER visit. (See above for a discussion of costs versus charges.).

Each of the unit values offered by the two studies has uncertainty surrounding it, and this uncertainty can be characterized by a distribution. The uncertainty surrounding the unit value of $311.55 from Smith et al. is characterized by a triangular distribution, centered at $311.55, on the range [$231, $431]. The uncertainty surrounding the unit value of $260.67 from Stanford et al. can be characterized as a normal distribution with mean equal to $260.67 and standard deviation equal to 5.22 (the reported standard error of the estimate. The uncertainty distribution for asthma-related ER visits is a pooled distribution obtained via Monte Carlo methods. On each of many iterations, one of the two distributions is selected (with each distribution having a 50 percent chance of selection), and then a value is randomly selected from that distribution.

4.3.4 Sensitivity Calculation: PM_{10}-Related Hospital Admissions

A recent study by the Health Effects Institute (HEI) (Samet et al., 2000) estimated separate models for PM_{10} and pneumonia, COPD and cardiovascular diseases in each of fourteen cities in the United States, as well as pooled estimates across these cities. The fourteen cities included in the HEI hospital admissions study are Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis/St. Paul, Minnesota; Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo/Orem, Utah; Seattle, Washington; Spokane, Washington; and Youngstown, Ohio.

The Samet et al. (2000) pooled estimates are desirable for several reasons. First, the HEI models are distributed lag models that are designed to capture not only same-day effects of PM but the effects of PM on a series of days subsequent to exposure. This type of model therefore captures the full impact of PM on hospital admissions. Samet et al. (2000) note that because of serial correlation, the coefficients of the PM lags tend to be unstable (i.e., have large variances) in single-city models; however, the pooled estimates, based on all fourteen cities are more stable because they are based on much larger sample sizes. A second advantage of the HEI models is that they represent the PM effect across a range of cities in the United States. Although other studies have estimated C-R functions in various cities in the United States, many of these cities (e.g., Minneapolis/St. Paul, Birmingham, Detroit, Spokane, New Haven, and Seattle) are included in the HEI study, which is a more recent analysis of the PM-hospital admissions relationships in these cities.

Although the HEI models do not include other pollutants, they do investigate the impact of omitting other pollutants on the estimated PM effects on hospital admissions. The results of this investigation are shown graphically in Figures 33 and 34 of Samet et al. (2000). The study authors conclude that the omission of SO_2 and O_3 from the models had virtually no effect on the estimated PM effect in any of the three pooled estimates (for cardiovascular diseases, COPD, and pneumonia). While Figure 34 suggests that this is the case for CV diseases and pneumonia, the omission of ozone from the model appears to have resulted in a downward-biased estimate of the PM effect on hospital admissions for COPD. This suggests that using the HEI pooled estimate for COPD will tend to understate the PM effect.

In valuing PM_{10}-related hospital admissions, we use the same cost-of-illness approach that we used for the Base Estimate. Appendix A summarizes these results.

4.4 Acute Illnesses and Symptoms Not Requiring Hospitalization

As indicated in Exhibit 4-17, in addition to mortality, chronic illness, and hospital admissions, there are a number of acute health effects not requiring hospitalization that are associated with exposure to ambient levels of ozone and PM. The sources for the C-R functions used to quantify these effects are described below.

Exhibit 4-17. Studies of Symptoms/Illnesses Not Requiring Hospitalization

Endpoint [a]	Applied Ages	Pollutant	Study
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1996)
Lower respiratory symptoms	Ages 7-14	$PM_{2.5}$	Schwartz et al. (1994)
Upper respiratory symptoms	Asthmatics, ages 9-11	PM_{10}	Pope et al. (1991)
Minor restricted activity day	Ages 18-65	$PM_{2.5}$	Ostro and Rothschild (1989),
Work loss days	Ages 18-65	$PM_{2.5}$	Ostro (1987)
School Loss Days	9-10 6-11	O_3	Pooled estimate: Gilliland et al (2001) Chen et al (2000)
Worker productivity	Outdoor workers	O_3	Crocker and Horst (1981) and EPA (1994).

4.4.1 Acute Bronchitis

Around five percent of U.S. children between ages five and seventeen experience episodes of acute bronchitis annually (Adams and Marano, 1995). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia[33], with the exception of cough, most acute bronchtis symptoms abate within 7 to 10 days. We estimated the incidence of episodes of acute bronchitis in children between the ages 8-12 using a C-R function developed from 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 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.

In previous benefits analyses, such as in the §812 Prospective analysis (U.S. EPA, 1999b), acute bronchitis was valued at $59.31 (in 2000 $). This is 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

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

estimate was taken to be twice the value of a minor respiratory restricted activity day. For a more complete description of the derivation of this estimate, see Abt Associates (2000, p. 4-30).

A unit value of $59.31 assumes that an episode of acute bronchitis lasts only one day. However, this is generally not the case. More typically, it can last for 6 or 7 days. We therefore made a simple adjustment, multiplying the original unit value of $59.31 by 6. The unit value thus derived and used was $356 (=$59.31 x 6). The uncertainty surround this unit value was modeled as a uniform distribution on the range [$105, $607]. The endpoints of this range are 6 times the low and the high estimates used in the §812 Prospective analysis, inflated to 2000$.

4.4.2 Upper Respiratory Symptoms (URS)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary, and the daily occurrences of URS and LRS, as defined above, were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis.

The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

Valuing Upper Respiratory Symptoms

Willingness to pay to avoid a day of upper respiratory symptoms is based on symptom-specific WTPs to avoid those symptoms identified by Pope et al. (1991) as part of the complex of upper respiratory symptoms. Three contingent valuation studies have estimated WTP to avoid various morbidity symptoms that are either within the complex defined by Pope et al. (1991), or are similar to those symptoms. In each CV study, participants were asked their WTP to avoid a day of each of several symptoms. The WTP estimates corresponding to the morbidity symptoms valued in each study are presented in Exhibit 4-18.

The three individual symptoms listed in Exhibit 4-18 that were identified as most closely matching those listed by Pope, et al. (1991) for upper respiratory symptoms are cough, head/sinus congestion, and eye irritation, corresponding to "wet cough," "runny or stuffy nose," and "burning, aching or red eyes," respectively. A day of upper respiratory symptoms could consist of any one of the seven possible "symptom complexes" consisting of at least one of these three symptoms. These seven possible symptom complexes are presented in Exhibit 4-19. We assumed that each of these seven

complexes is equally likely.[34] The point estimate of WTP is just an average of the seven estimates of WTP for the different complexes. In the absence of information surrounding the frequency with which each of the seven occurs, we assumed a continuous uniform distribution between the lowest and highest values in Exhibit 4-19.

Exhibit 4-18. Median WTP Estimates and Derived Midrange Estimates (in 2000 $)

Symptom [a]	Dickie et al. (1987)	Tolley et al. (1986)	Loehman et al. (1979)	Mid-Range Estimate
Throat congestion	4.97	21.54	-	13.18
Head/sinus congestion	5.80	23.20	10.80	13.18
Coughing	1.66	18.24	6.56	9.23
Eye irritation	-	20.70	-	20.70
Headache	1.66	33.15	-	13.18
Shortness of breath	0.00	-	13.92	6.58
Pain upon deep inhalation (PDI)	5.82	-	-	5.82
Wheeze	3.32	-	-	3.32
Coughing up phlegm	3.63 [b]	-	-	3.63
Chest tightness	8.30	-	-	8.30

[a] All estimates are WTP to avoid one day of symptom. Midrange estimates were derived by IEc (1993).

[b] 10% trimmed mean.

Exhibit 4-19. Estimates of WTP to Avoid Upper Respiratory Symptoms (2000 $)

Symptom Combinations Identified as URS by Pope et al. (1991)	WTP to Avoid Symptom(s)
Coughing	$9.23
Head/Sinus Congestion	$13.18
Eye Irritation	$20.70
Coughing, Head/Sinus Congestion	$22.40
Coughing, Eye Irritation	$29.93
Head/Sinus Congestion, Eye Irritation	$33.88
Coughing, Head/Sinus Congestion, Eye Irritation	$43.11
	Average: $24.63

Based on values reported in Exhibit 4-18.

4.4.3 Lower Respiratory Symptoms

[34] With empirical evidence, we could presumably improve the accuracy of the probabilities of occurrence of each type of URS. Lacking empirical evidence, however, a uniform distribution seems the most reasonable "default" assumption.

Lower respiratory symptoms include symptoms such as cough, chest pain, phlegm, and wheeze. To estimate the link between $PM_{2.5}$ and lower respiratory symptoms, we used a study by Schwartz et al. (1994). Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+. The $PM_{2.5}$ C-R function is based on the single pollutant model reported in Schwartz et al. (1994, Table 5).

Valuing Lower Respiratory Symptoms

Schwartz et al. (1994, p. 1235) defined lower respiratory symptoms as at least two of the following symptoms: cough, chest pain, phlegm, and wheeze. To value this combination of symptoms, we used the same method as we did for upper respiratory symptoms. We chose those individual health effects that seem most consistent with lower respiratory symptoms, we derived all of the possible combinations of these symptoms, and then we valued these combinations.

The symptoms for which WTP estimates are available that reasonably match lower respiratory symptoms are: cough (C), chest tightness (CT), coughing up phlegm (CP), and wheeze (W). A day of lower respiratory symptoms could consist of any one of the 11 combinations of at least two of these four symptoms, as displayed in Exhibit 4-20.[35] We assumed that each of the eleven types of lower respiratory symptoms is equally likely,[36] and the mean WTP is the average of the WTPs over all combinations. Finally, in the absence of information about the frequency of each of the eleven types of lower respiratory symptoms, the uncertainty analysis for LRS assumes a continuous uniform distribution on the range from the lowest to the highest value in Exhibit 4-20.

Note that the WTP estimates are based on studies which considered the value of a *day* of avoided symptoms, whereas the Schwartz et al. study used as its measure a *case* of LRS. Because a case of LRS usually lasts at least one day, and often more, our estimate is a conservative one.

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[35] Because cough is a symptom in some of the upper respiratory symptom clusters as well as some of the lower respiratory symptom clusters, there is the possibility of a very small amount of double counting – if the same individual were to have an occurrence of upper respiratory symptoms which included cough and an occurrence of lower respiratory symptoms which included cough *both on exactly the same day.* Because this is probably a very small probability occurrence, the degree of double counting is likely to be very minor. Moreover, because upper respiratory symptoms is applied only to asthmatics ages 9-11 (a very small population), the amount of potential double counting should be truly negligible.

[36] As with URS, if we had empirical evidence we could improve the accuracy of the probabilities of occurrence of each type of LRS. Lacking empirical evidence, however, a uniform distribution seems the most reasonable "default" assumption.

Exhibit 4-20. Estimates of WTP to Avoid Lower Respiratory Symptoms (2000 $)

Symptom Combinations Identified as LRS by Schwartz et al. (1994)	WTP to Avoid Symptoms
Coughing, Chest Tightness	$17.52
Coughing, Coughing Up Phlegm	$12.84
Coughing, Wheeze	$12.54
Chest Tightness, Coughing Up Phlegm	$11.92
Chest Tightness, Wheeze	$11.62
Coughing Up Phlegm, Wheeze	$6.95
Coughing, Chest Tightness, Coughing Up Phlegm	$21.15
Coughing, Chest Tightness, Wheeze	$20.85
Coughing, Coughing Up Phlegm, Wheeze	$16.17
Chest Tightness, Coughing Up Phlegm, Wheeze	$15.25
Coughing, Chest Tightness, Coughing Up Phlegm, Wheeze	$24.47
	Average: $15.57

Based on values reported in Exhibit 4-18.

Issues in the Valuation of URS and LRS

The point estimates derived for mean WTP to avoid a day of URS and a case of LRS are based on the assumption that WTPs are additive. For example, if WTP to avoid a day of cough is $9.23, and WTP to avoid a day of shortness of breath is $6.58, then WTP to avoid a day of both cough and shortness of breath is $15.81. If there are no synergistic effects among symptoms, then it is likely that the marginal utility of avoiding symptoms decreases with the number of symptoms being avoided. If this is the case, adding WTPs would tend to overestimate WTP for avoidance of multiple symptoms. However, there may be synergistic effects– that is, the discomfort from two or more simultaneous symptoms may exceed the sum of the discomforts associated with each of the individual symptoms. If this is the case, adding WTPs would tend to underestimate WTP for avoidance of multiple symptoms. It is also possible that people may experience additional symptoms for which WTPs are not available, again leading to an underestimate of the correct WTP. However, for small numbers of symptoms, the assumption of additivity of WTPs is unlikely to result in substantive bias.

There are also three sources of uncertainty in the valuation of both URS and LRS: (1) an occurrence of URS or of LRS may be comprised of one or more of a variety of symptoms (i.e., URS and LRS are each potentially a "complex of symptoms"), so that what is being valued may vary from one occurrence to another; (2) for a given symptom, there is uncertainty about the mean WTP to avoid the symptom; and (3) the WTP to avoid an occurrence of multiple symptoms may be greater or less than the sum of the WTPs to avoid the individual symptoms.

Information about the degree of uncertainty from either the second or the third source is not available. The first source of uncertainty, however, is addressed because an occurrence of URS or LRS may vary in symptoms. For example, seven different symptom complexes that qualify as URS, as defined by Pope et al. (1991), were identified above. The estimates of WTP to avoid these seven different kinds of URS range from $9.23 (to avoid an occurrence of URS that consists of only coughing) to $43.11 (to avoid an occurrence of URS that consists of coughing plus head/sinus congestion plus eye irritation).

There is no information, however, about the frequency of each of the seven types of URS among all occurrences of URS.

Because of insufficient information to adequately estimate the distributions of the estimators of WTP for URS and LRS, as a rough approximation, a continuous uniform distribution over the interval from the smallest point estimate to the largest is used, as noted above.

Alternatively, a discrete distribution of the seven unit dollar values associated with each of the seven types of URS identified could be used. This would provide a distribution whose mean is the same as the point estimate of WTP. A continuous uniform distribution, however, is probably more reasonable than a discrete uniform distribution. The differences between the means of the discrete uniform distributions (the point estimates) and the means of the continuous uniform distributions are relatively small, as shown in Exhibit 4-21.

Exhibit 4-21. Comparison of the Means of Discrete and Continuous Uniform Distributions of WTP Associated with URS and LRS (2000 $)

Health Endpoint	Mean of Discrete Uniform Distribution (Point Est.)	Mean of Continuous Uniform Distribution
Upper Respiratory Symptoms	$24.64	$26.17
Lower Respiratory Symptoms	$15.57	$15.70

4.4.4 Minor Restricted Activity Days

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRAD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. We developed separate coefficients for each year in the analysis (1976-1981), which were then combined for use in this analysis. The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild (1989), Table 4, using the inverse of the variance as the weight.

Valuing Minor Restricted Activity Days

The unit value and uncertainty distribution for MRADs for this analysis were obtained by adjusting the (rounded) values in 1990 $ used in the §812 Prospective analysis to 2000 $ by multiplying by 1.3175. No studies are reported to have estimated WTP to avoid a minor restricted activity day (MRAD). However, IEc (1993) has derived an estimate of WTP to avoid a minor respiratory restricted activity day (MRRAD), using WTP estimates from Tolley et al. (1986) for avoiding a three-symptom combination of coughing, throat congestion, and sinusitis. This estimate of WTP to avoid a MRRAD, so defined, is $38.37 (1990 $), or about $38. Although Ostro and Rothschild (1989) estimated the relationship between $PM_{2.5}$ and MRADs, rather than MRRADs (a component of MRADs), it is likely that most of the MRADs associated with exposure to $PM_{2.5}$ are in fact MRRADs. For the purpose of valuing this health endpoint, then, we assumed that MRADs associated with PM exposure may be more specifically defined as MRRADs, and therefore used the estimate of mean WTP to avoid a MRRAD.

Any estimate of mean WTP to avoid a MRRAD (or any other type of restricted activity day other

than WLD) will be somewhat arbitrary because the endpoint itself is not precisely defined. Many different combinations of symptoms could presumably result in some minor or less minor restriction in activity. Krupnick and Kopp (1988) argued that mild symptoms will not be sufficient to result in a MRRAD, so that WTP to avoid a MRRAD should exceed WTP to avoid any single mild symptom. A single severe symptom or a combination of symptoms could, however, be sufficient to restrict activity. Therefore WTP to avoid a MRRAD should, these authors argue, not necessarily exceed WTP to avoid a single severe symptom or a combination of symptoms. The "severity" of a symptom, however, is similarly not precisely defined; moreover, one level of severity of a symptom could induce restriction of activity for one individual while not doing so for another. The same is true for any particular combination of symptoms.

Given that there is inherently a substantial degree of arbitrariness in any point estimate of WTP to avoid a MRRAD (or other kinds of restricted activity days), the reasonable bounds on such an estimate must be considered. By definition, a MRRAD does not result in loss of work. WTP to avoid a MRRAD should therefore be less than WTP to avoid a WLD. At the other extreme, WTP to avoid a MRRAD should exceed WTP to avoid a single mild symptom. The highest IEc midrange estimate of WTP to avoid a single symptom is $15.72 (1990 $), or about $16, for eye irritation. The point estimate of WTP to avoid a WLD in the benefit analysis is $83 (1990 $). If all the single symptoms evaluated by the studies are not severe, then the estimate of WTP to avoid a MRRAD should be somewhere between $16 and $83. Because the IEc estimate of $38 falls within this range (and acknowledging the degree of arbitrariness associated with any estimate within this range), the IEc estimate is used as the mean of a triangular distribution centered at $38, ranging from $16 to $61. Adjusting to 2000 $, this is a triangular distribution centered at $50.55, ranging from $21 to $80.

4.4.5 Work Loss Days

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

Valuing Work Loss Days

Willingness to pay to avoid the loss of one day of work was estimated by dividing county-specific median annual wages (GeoLytics Inc., 2002b) by 50 (assuming 2 weeks of vacation) and then by 5, to get county-specific median daily wages. Valuing the loss of a day's work at the wages lost is consistent with economic theory, which assumes that an individual is paid exactly the value of his labor.

The use of the median rather than the mean, however, requires some comment. If all individuals in society were equally likely to be affected by air pollution to the extent that they lose a day of work because of it, then the appropriate measure of the value of a work loss day would be the mean daily wage. It is highly likely, however, that the loss of work days due to pollution exposure does not occur with equal probability among all individuals, but instead is more likely to occur among lower income individuals than among high income individuals. It is probable, for example, that individuals who are

vulnerable enough to the negative effects of air pollution to lose a day of work as a result of exposure tend to be those with generally poorer health care. Individuals with poorer health care have, on average, lower incomes.

To estimate the average lost wages of individuals who lose a day of work because of exposure to PM pollution, then, would require a weighted average of all daily wages, with higher weights on the low end of the wage scale and lower weights on the high end of the wage scale. Because the appropriate weights are not known, however, the median wage was used rather than the mean wage. The median is more likely to approximate the correct value than the mean because means are highly susceptible to the influence of large values in the tail of a distribution (in this case, the small percentage of very large incomes in the United States), whereas the median is not susceptible to these large values. We currently have insufficient information to characterize the uncertainty surrounding any county-specific estimate for WLD.

4.4.6 School Loss Days

Children may also be absent from school due to respiratory or other diseases caused by exposure to air pollution. Most studies examining school absence rates have found little or no association with PM2.5, but several studies have found a significant association between ozone levels and school absence rates. We use two recent studies, Gilliland et al. (2001) and Chen et al. (2000) to estimate changes in absences (school loss days) due to changes in ozone levels. The Gilliland et al. study estimated the incidence of new periods of absence, while the Chen et al. study examined absence on a given day. We convert the Gilliland estimate to days of absence by multiplying the absence periods by the average duration of an absence. We estimate an average duration of school absence of 1.6 days by dividing the average daily school absence rate from Chen et al. (2000) and Ransom and Pope (1992) by the episodic absence rate from Gilliland et al. (2001). This provides estimates from Chen et al. (2000) and Gilliland et al. (2000) which can be pooled to provide an overall estimate.

Valuing School Loss Days

To value a school absence, (1) we estimated the probability that, if a school child stays home from school, a parent will have to stay home from work to care for the child, and (2) we valued the lost productivity at the person's wage. To do this, we first estimated the proportion of families with school-age children in which both parents work, and then valued a school loss day as the probability of a work loss day resulting from a school loss day (i.e., the proportion of households with school-age children in which both parents work) times a measure of lost wages. This approach is likely to understate the value of a school loss day in two ways. First, it omits WTP to avoid the symptoms/illness which resulted in the school absence. Second, it effectively gives zero value to school absences which do not result in a work loss day. We used this method, however, in the absence of a preferable method.

From the U.S. Census Bureau, Statistical Abstract of the United States: 2001, we obtained (1) the numbers of single, married, and "other" (i.e., widowed, divorced, or separated) women with children in the workforce, and (2) the rates of participation in the workforce of single, married, and "other" women with children. From these two sets of statistics, we calculated a weighted average participation rate of 72.85 percent (Exhibit 4-22).

Our estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This

median weekly wage is $551. Dividing by 5 gives an estimated median daily wage of $103. The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of $103, or $75. We currently have insufficient information to characterize the uncertainty surrounding this estimate.

Exhibit 4-22. Women with Children: Number and Percent in the Labor Force, 2000, and Weighted Average Participation Rate

Category	Women in Labor Force (millions) [a]	Participation Rate (%) [a]	Implied Total Number in Population (in millions)	Implied Percent in Population	Population-Weighted Average Participation Rate
	(1)	*(2)*	*(3) = (1)/(2)*	*(4)*	*[=sum (2)*(4) over rows]*
Single	3.1	73.9%	4.19	11.84%	
Married	18.2	70.6%	25.78	72.79%	
Other [b]	4.5	82.7%	5.44	15.36%	
Total			35.42		**72.85%**

[a] Source: U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 577.

[b] Widowed, divorced, or separated.

4.4.7 Worker Productivity

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst (1981) and summarized in EPA (1994). Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration (-0.1427).[37] The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income.

Previous EPA analyses used a unit value of $1.00 per worker per 10% change in ozone per day, based on a U.S. Census Bureau estimate of "average daily income of outdoor workers engaged in strenuous activity" of $96.00 (in 2000$). We were unable to find the same category in tables of more recent U.S. Census Bureau estimates. The most similar category we were able to find was "median daily earnings of workers in farming, forestry and fishing."[38] Using this category, the value of a day of work for outdoor workers is $68 (in 2000$). From this we derived a unit value of $0.95 per worker per 10% change in ozone per day. We applied this unit value to county-specific numbers of workers in farming, forestry, and fishing, obtained from the 2000 U.S. Census. The median daily income for outdoor workers is a national estimate, however. We adjust this estimate to reflect regional variations in income using a

[37] The relationship estimated by Crocker and Horst (1981) between wages and ozone is a log-log relationship. Therefore the elasticity of wages with respect to ozone is a constant, equal to the coefficient of log ozone in the model.

[38] Statistical Abstract of the United States, Table 621, "Full-Time Wage and Salary Workers – Number and Earnings: 1985 to 2000" (Source of data in table: U.S. Bureau of Labor Statistics, Bulletin 2307 and Employment and Earnings, monthly).

factor based on the ratio of national median household income divided by a county's median household income. No information was available for quantifying the uncertainty associated with the central valuation estimate. Therefore, no uncertainty analysis was conducted for this endpoint.

4.4.8 Sensitivity Calculation: Asthmatic Studies

The base analysis does not include C-R functions to estimate asthma-related symptoms. EPA is currently evaluating how air pollution related symptoms in the asthmatic population should be incorporated into the overall benefits analysis. Clearly, studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double-counting. In one specific case, upper respiratory symptoms in children, the only study available was limited to asthmatic children, so this endpoint is included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms, are estimated for the total population of children.

Given the increased susceptibility of the asthmatic population, it is of interest to understand better the specific impacts on asthmatics. We are providing a separate set of estimated health impacts for asthmatic populations, listed it Exhibit 4-23, with the caveat that these are not additive, nor can they be easily combined with other endpoints to derive total benefits. They are provided only to highlight the potential impacts on a susceptible population.

Exhibit 4-23. Studies Examining Health Impacts in the Asthmatic Population

Endpoint	Definition	Pollutant	Study	Study Population
Asthma Attack Indicators				
Shortness of Breath	prevalence of shortness of breath; incidence of shortness of breath	$PM_{2.5}$	Ostro et al. (2001)	African American asthmatics, 8-13
Cough	prevalence of cough; incidence of cough	$PM_{2.5}$	Ostro et al. (2001)	African American asthmatics, 8-13
Wheeze	prevalence of wheeze; incidence of wheeze	$PM_{2.5}$	Ostro et al. (2001)	African American asthmatics, 8-13
Asthma Exacerbation	≥1 mild asthma symptom: wheeze, cough, chest tightness, shortness of breath)	PM_{10}, $PM_{1.0}$	Yu et al. (2000)	Asthmatics, 5-13
Cough	prevalence of cough	PM_{10}	Vedal et al. (1998)	Asthmatics, 6-13
Other symptoms/illness endpoints				
Upper Respiratory Symptoms	≥1 of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes	PM_{10}	Pope et al. (1991)	Asthmatics 9-11
Moderate or Worse Asthma	probability of moderate (or worse) rating of overall asthma status	$PM_{2.5}$	Ostro et al. (1991)	Asthmatics, all ages
Acute Bronchitis	≥1 episodes of bronchitis in the past 12 months	$PM_{2.5}$	McConnell et al. (1999)	Asthmatics, 9-15*
Phlegm	"other than with colds, does this child usually seem congested in the chest or bring up phlegm?"	$PM_{2.5}$	McConnell et al. (1999)	Asthmatics, 9-15*
Asthma Attacks	respondent-defined asthma attack	PM2.5, ozone	Whittemore and Korn (1980)	Asthmatics, all ages

4.4.9 Sensitivity Calculations: "Any-of-19 Respiratory Symptoms"

Krupnick et al. (1990) estimated the impact of coefficient of haze (COH, a measure of particulate matter concentrations), ozone and SO_2 on the incidence of any of 19 symptoms or conditions in the adult population, ages 18 to 65.[39] They used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model is that the model they used includes a lagged value of the dependent variable. This makes the derivation of a C-R function somewhat more complicated than the usual logistic regression.[40]

The presence of "any of 19 acute respiratory symptoms" is a somewhat subjective health effect used by Krupnick et al. (1990). Moreover, not all 19 symptoms are listed in the Krupnick et al. study. It is therefore not clear exactly what symptoms were included in the study. Even if all 19 symptoms were

[39] Krupnick et al. (1990) list 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

[40] Details of the derivation of the C-R function based on the model used by Krupnick et al. (1990) are presented in Abt Associates (1999, p. A-40).

known, it is unlikely that WTP estimates could be obtained for all of the symptoms. Finally, even if all 19 symptoms were known and WTP estimates could be obtained for all 19 symptoms, the assumption of additivity of WTPs becomes tenuous with such a large number of symptoms. The likelihood that all 19 symptoms would occur simultaneously, moreover, is very small.

Valuing "Any of 19 Respiratory Symptoms"

The unit value and uncertainty distribution for "any of 19 respiratory symptoms" for this analysis were obtained by adjusting the values in 1990 $ used in the §812 Prospective analysis to 2000 $ by multiplying by 1.3175. Acute respiratory symptoms must be either upper respiratory symptoms or lower respiratory symptoms. In the absence of further knowledge about which of the two types of symptoms is more likely to occur among the "any of 19 acute respiratory symptoms," we assumed that they occur with equal probability. Because this health endpoint may also consist of combinations of symptoms, it was also assumed that there is some (smaller) probability that upper and lower respiratory symptoms occur together. To value avoidance of a day of "the presence of any of 19 acute respiratory symptoms" we therefore assumed that this health endpoint consists either of URS, or LRS, or both. We also assumed that it is as likely to be URS as LRS and that it is half as likely to be both together. That is, it was assumed that "the presence of any of 19 acute respiratory symptoms" is a day of URS with 40 percent probability, a day of LRS with 40 percent probability, and a day of both URS and LRS with 20 percent probability. Using the point estimates of WTP to avoid a day of URS and LRS derived above, the point estimate of WTP to avoid a day of "the presence of any of 19 acute respiratory symptoms" is:

$$(0.40)(\$24.64) + (0.40)(\$15.57) + (0.20)(\$24.64 + \$15.57) = \$24.12 \text{ (2000 \$)}.$$

Because this health endpoint is only vaguely defined, and because of the lack of information on the relative frequencies of the different combinations of acute respiratory symptoms that might qualify as "any of 19 acute respiratory symptoms," the unit dollar value derived for this health endpoint must be considered only a rough approximation.

The sources of uncertainty in the valuation of LRS and URS described above similarly exist in the valuation of this health endpoint. In particular, (1) "the presence of any of 19 acute respiratory symptoms" may be comprised of one or more of a variety of symptoms, so that what is being valued may vary from one occurrence to another; (2) for a given symptom, there is uncertainty about the mean WTP to avoid the symptom; and (3) the WTP to avoid an occurrence of multiple symptoms may be greater or less than the sum of the WTPs to avoid the individual symptoms.

To characterize the uncertainty surrounding the estimated value of avoiding "any of 19 acute respiratory symptoms," we used the distributions described above for the input components, URS and LRS. On each iteration of a Monte Carlo procedure, URS was chosen with 40 percent probability, LRS was chosen with 40 percent probability and URS+LRS was chosen with 20 percent probability. Given the choice, a dollar value was randomly selected from the appropriate distribution. For example, if URS was selected, a dollar value was selected from the continuous uniform distribution for URS.

5 Welfare Benefits

This analysis considers three types of benefits that are loosely termed "welfare" benefits. These include visibility improvements, reductions in agricultural crop damage, and reduced household soiling. We consider each in turn.

5.1 Visibility Benefits

Visibility degradation estimates used in this analysis are generated by the REMSAD model. To conduct the visibility benefits analysis, however, we need visibility data at the county level. To convert REMSAD visibility data from the square grid to the county level, we use the following rule: if a county center falls within a given REMSAD grid cell, we assign that REMSAD grid cell's visibility values to that county. Because the modeled air quality-related changes in visibility are directly used in the benefits analysis, the methodology for predicting visibility changes is not discussed here. The visibility estimation procedure is described in detail in EPA (2000b), and is based on the methods in Sisler (1996).

Economic benefits may result from two broad categories of visibility changes: (1) changes in "residential" visibility – i.e., the visibility in and around the locations where people live; and (2) changes in "recreational" visibility at Class I areas – i.e., visibility at Class I national parks and wilderness areas.[41] In this analysis, only those recreational benefits in Class I areas that have been directly studied (in California, the Southeast, and the Southwest) are included in the base presentation of benefits; residential benefits and recreational benefits in all U.S. Class I areas are presented as alternative calculations of visibility benefits.

Within the category of recreational visibility, further distinctions have been made. There is evidence (Chestnut and Rowe, 1990a) that an individual's WTP for improvements in visibility at a Class I area is influenced by whether it is in the region in which the individual lives, or whether it is somewhere else. In general people appear to be willing to pay more for visibility improvements at parks and wilderness areas that are "in-region" than at those that are "out-of-region." This is plausible, because people are more likely to visit, be familiar with, and care about parks and wilderness areas in their own part of the country.

To value estimated visibility changes, we are using an approach consistent with economic theory. Below we discuss an application of the Constant Elasticity of Substitution (CES) utility function approach[42] to value both residential visibility improvements and visibility improvements at Class I areas in the United States. This approach is based on the preference calibration method developed by Smith et al. (2002). The presentation of this methodology is organized as follows. The basic utility model is presented in Section 5.1.1. In Section 5.1.2 we discuss the measurement of visibility, and the mapping from environmental "bads" to environmental "goods." In Sections 5.1.3 and 5.1.4 we summarize the information that is available to estimate the parameters of the model corresponding to visibility at in-

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[41] Hereafter referred to as Class I areas, which are defined as areas of the country such as national parks, national wilderness areas, and national monuments that have been set aside under Section 162(a) of the Clean Air Act to receive the most stringent degree of air quality protection. Class I federal lands fall under the jurisdiction of three federal agencies, the National Park Service, the Fish and Wildlife Service, and the Forest Service.

[42] The Constant Elasticity of Substitution utility function has been chosen for use in this analysis due to its flexibility when illustrating the degree of substitutability present in various economic relationships (in this case, the tradeoff between income and improvements in visibility).

region and out-of-region Class I areas, and visibility in residential areas, respectively, and we describe the methods used to estimate these parameters. Section 5.1.5 synthesizes the results.

5.1.1 Basic Utility Model

We begin with a CES utility function in which a household derives utility from

(1) "all consumption goods," X,
(2) visibility in the residential area in which the household is located ("residential visibility"),[43]
(3) visibility at Class I areas in the same region as the household ("in-region recreational visibility"), and
(4) visibility at Class I areas outside the household's region ("out-of-region recreational visibility").

There are a total of six regions being considered, so there are 5 regions for which any household is out-of-region. The utility function of a household in the nth residential area and the ith region of the country is:

$$U_{ni} = (X^{\rho} + \theta Z_n^{\rho} + \sum_{k=1}^{N_i} \gamma_{ik} Q_{ik}^{\rho} + \sum_{j \neq i} \sum_{k=1}^{N_j} \delta_{jk} Q_{jk}^{\rho})^{1/\rho} \quad ,$$

$$\theta > 0, \gamma_{ik} > 0, \forall i,k, \delta_{jk} > 0, \forall j,k, \ \rho \leq 1.$$

where

Z_n = the level of visibility in the nth residential area;
Q_{ik} = the level of visibility at the kth in-region park (i.e., the kth park in the ith region);
Q_{jk} = the level of visibility at the kth park in the jth region (for which the household is out-of-region), $j \neq i$;
N_i = the number of Class I areas in the ith region;
N_j = the number of Class I areas in the jth region (for which the household is out-of-region), $j \neq i$; and

θ, the γ's and δ's are parameters of the utility function corresponding to the visibility levels at residential areas, and at in-region and out-of-region Class I areas, respectively. In particular, the γ_{ik}'s are the parameters corresponding to visibility at in-region Class I areas; the δ_1's are the parameters corresponding to visibility at Class I areas in region 1 (California), if $i \neq 1$; the δ_2's are the parameters corresponding to visibility at Class I areas in region 2 (Colorado Plateau), if $i \neq 2$, and so forth. Because the model assumes that the relationship between residential visibility and utility is the same everywhere, there is only one θ. The parameter ρ in this CES utility function is an important determinant of the slope of the marginal WTP curve associated with any of the environmental quality variables. When ρ=1, the marginal WTP curve is horizontal. When ρ<1, it is downward sloping.

The household's budget constraint is:

$$m - p \cdot X \leq 0 \ ,$$

[43] We remind the reader that, although residential and recreational visibility benefits estimation is discussed simultaneously in this section, benefits are calculated and presented separately for each visibility category.

where m is income, and p is the price of X. Without loss of generality, set p = 1. The only choice variable is X. The household maximizes its utility by choosing X=m. The indirect utility function for a household in the nth residential area and the ith region is therefore

$$V_{ni}(m, Z_n, Q; \theta, \gamma, \delta, \rho) = (m^\rho + \theta Z_n^\rho + \sum_{k=1}^{N_i} \gamma_{ik} Q_{ik}^\rho + \sum_{j \neq i} \sum_{k=1}^{N_j} \delta_{jk} Q_{jk}^\rho)^{1/\rho} \quad ,$$

where Q denotes the vector of vectors, Q_1, Q_2, Q_3, Q_4, Q_5, and Q_6, and the unsubscripted γ and δ denote vectors as well.

Given estimates of ρ, θ, the γ's and the δ's, the household's utility function and the corresponding WTP functions are fully specified. The household's WTP for any set of changes in the levels of visibility at in-region Class I areas, out-of-region Class I areas, and the household's residential area can be shown to be:

$$WTP_{ni}(\Delta Z, \Delta Q) = m - [m^\rho + \theta(Z_{0n}^\rho - Z_{1n}^\rho) + \sum_{k=1}^{N_i} \gamma_{ik}(Q_{0ik}^\rho - Q_{1ik}^\rho) + \sum_{j \neq i} \sum_{k=1}^{N_j} \delta_{jk}(Q_{0jk}^\rho - Q_{1jk}^\rho)]^{1/\rho} \quad .$$

The household's WTP for a single visibility improvement will depend on its order in the series of visibility improvements the household is valuing. If it is the first visibility improvement to be valued, the household's WTP for it follows directly from the previous equation. For example, the household's WTP for an improvement in visibility at the first in-region park, from $Q_{i1} = Q_{0i1}$ to $Q_{i1} = Q_{1i1}$, is

$$WTP(\Delta Q_{i1}) = m - [m^\rho + \gamma_{i1}(Q_{0i1}^\rho - Q_{1i1}^\rho)]^{1/\rho} \quad ,$$

if this is the first (or only) visibility change the household values.

5.1.2 Measure of Visibility: Environmental "Goods" Versus "Bads"

In the above model, Q and Z are environmental "goods." As the level of visibility increases, utility increases. The utility function and the corresponding WTP function both have reasonable properties. The first derivative of the indirect utility function with respect to Q (or Z) is positive; the second derivative is negative. WTP for a change from Q_0 to a higher (improved) level of visibility, Q_1, is therefore a concave function of Q_1, with decreasing marginal WTP.

The measure of visibility that is currently preferred by air quality scientists is the deciview, which increases as visibility *decreases*. Deciview, in effect, is a measure of the *lack* of visibility. As deciviews increase, visibility, and therefore utility, decreases. The deciview, then, is a measure of an environmental "bad." There are many examples of environmental "bads" – all types of pollution are environmental "bads." Utility decreases, for example, as the concentration of particulate matter in the atmosphere increases.

One way to value decreases in environmental bads is to consider the "goods" with which they are associated, and to incorporate those goods into the utility function. In particular, if B denotes an environmental "bad," such that:

$$\frac{\partial V}{\partial B} < 0 \ ,$$

and the environmental "good," Q, is a function of B,

$$Q = F(B) \ ,$$

then the environmental "bad" can be related to utility via the corresponding environmental "good":[44]

$$V = V(m, Q) = V(m, F(B)) \ .$$

The relationship between Q and B, F(B), is an empirical relationship that must be estimated.

There is a potential problem with this approach, however. If the function relating B and Q is not the same everywhere (i.e., if for a given value of B, the value of Q depends on other factors as well), then there can be more than one value of the environmental good corresponding to any given value of the environmental bad, and it is not clear which value to use. This has been identified as a problem with translating deciviews (an environmental "bad") into visual range (an environmental "good"). It has been noted that, for a given deciview value, there can be many different visual ranges, depending on the other factors that affect visual range – such as light angle and altitude. We note here, however, that this problem is not unique to visibility, but is a general problem when trying to translate environmental "bads" into "goods."[45]

In order to translate deciviews (a "bad") into visual range (a "good"), we use a relationship derived by Malm and Pitchford (1994) in which

$$DV = 10 * ln(\frac{391}{VR}) \ ,$$

where DV denotes deciview and VR denotes visual range (in kilometers). Solving for VR as a function of DV yields

$$VR = 391 * e^{-0.1DV} \ .$$

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[44] There may be more than one "good" related to a given environmental "bad." To simplify the discussion, however, we assume only a single "good."

[45] Another example of an environmental "bad" is particulate matter air pollution (PM). The relationship between survival probability (Q) and the ambient PM level is generally taken to be of the form

$$Q = 1 - \alpha e^{\beta PM} \ .$$

where α denotes the mortality rate (or level) when there is no ambient PM (i.e., when PM=0). However, α is implicitly a function of all the factors other than PM that affect mortality. As these factors change (e.g., from one location to another), α will change (just as visual range changes as light angle changes). It is therefore possible to have many values of Q corresponding to a given value of PM, as the values of α vary.

This conversion is based on specific assumptions characterizing the "average" conditions of those factors, such as light angle, that affect visual range. To the extent that specific locations depart from the average conditions, the relationship will be an imperfect approximation.[46]

5.1.3 Estimating the Parameters for Visibility at Class I Areas: the γ's and δ's

As noted in Section 2, if we consider a particular visibility change as the first or the only visibility change valued by the household, the household's WTP for that change in visibility can be calculated, given income (m), the "shape" parameter, ρ, and the corresponding recreational visibility parameter. For example, a Southeast household's WTP for a change in visibility at in-region parks (collectively) from $Q_1 = Q_{01}$ to $Q_1 = Q_{11}$ is:

$$WTP(DQ_1) = m - [m^r + g_1(Q_{01}^r - Q_{11}^r)]^{1/r}$$

if this is the first (or only) visibility change the household values.

Alternatively, if we have estimates of m as well as WTP_1^{in} and WTP_1^{out} of in-region and out-of-region households, respectively, for a given change in visibility from Q_{01} to Q_{11} in Southeast parks, we can solve for γ_1 and δ_1 as a function of our estimates of m, WTP_1^{in} and WTP_1^{out}, for any given value of ρ. Generalizing, we can derive the values of γ and δ for the jth region as follows:

$$\gamma_j = \frac{(m - WTP_j^{in})^\rho - m^\rho}{(Q_{0j}^\rho - Q_{1j}^\rho)}$$

and

$$\delta_j = \frac{(m - WTP_j^{out})^\rho - m^\rho}{(Q_{0j}^\rho - Q_{1j}^\rho)} \quad .$$

Chestnut and Rowe (1990a) and Chestnut (1997) estimated WTP (per household) for specific visibility changes at national parks in three regions of the United States – both for households that are in-region (in the same region as the park) and for households that are out-of-region. The Chestnut and Rowe study asked study subjects what they would be willing to pay for each of three visibility improvements in the national parks in a given region. Study subjects were shown a map of the region, with dots indicating the locations of the parks in question. The WTP questions referred to the three visibility improvements in all the parks collectively; the survey did not ask subjects' WTP for these improvements in specific parks individually. Responses were categorized according to whether the respondents lived in the same region as the parks in question ("in-region" respondents) or in a different region ("out-of-region" respondents). The areas for which in-region and out-of-region WTP estimates are available from Chestnut and Rowe (1990a), and the sources of benefits transfer-based estimates that we employ in the absence of estimates, are summarized in Exhibit 5-1. In all cases, WTP refers to WTP per household.

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[46] Ideally, we would want the location- , time-, and meteorological condition-specific relationships between deciviews and visual range, which could be applied as appropriate. This is probably not feasible, however.

Exhibit 5-1. Available Information on WTP for Visibility Improvements in National Parks

Region of Park	Region of Household	
	In-Region[a]	Out-of-Region[b]
1. California	WTP estimate from study	WTP estimate from study
2. Colorado Plateau	WTP estimate from study	WTP estimate from study
3. Southeast United States	WTP estimate from study	WTP estimate from study
4. Northwest United States	(based on benefits transfer from California)	
5. Northern Rockies	(based on benefits transfer from Colorado Plateau)	
6. Rest of United States	(based on benefits transfer from Southeast U.S.)	

[a] In-region" WTP is WTP for a visibility improvement in a park in the same region as that in which the household is located. For example, in-region WTP in the "Southeast" row is the estimate of the average Southeast household's WTP for a visibility improvement in a Southeast park.

[b] Out-of-region" WTP is WTP for a visibility improvement in a park that is not in the same region in which the household is located. For example, out-of-region WTP in the "Southeast" row is the estimate of WTP for a visibility improvement in a park in the Southeast by a household outside of the Southeast.

In the base calculation of visibility benefits for this analysis, only visibility changes at parks within visibility regions for which a WTP estimate was available from Chestnut and Rowe (1990a) are considered (for both in- and out-of-region benefits). Base estimates will not include visibility benefits calculated by transferring WTP values to visibility changes at parks not included in the Chestnut and Rowe study. Transferred benefits at parks located outside of the Chestnut and Rowe visibility regions will, however, be included as an alternative calculation.

The values of the parameters in a household's utility function will depend on where the household is located. The region-specific parameters associated with visibility at Class I areas (that is, all parameters except the residential visibility parameter) are arrayed in Exhibit 5-2. The parameters in columns 1-3 can be directly estimated using WTP estimates from Chestnut and Rowe (1990a) (the columns labeled "Region 1," "Region 2," and "Region 3").

Exhibit 5-2. Summary of Region-Specific Recreational Visibility Parameters to be Estimated in Household Utility Functions

Region of Household	Region of Park					
	Region 1	Region 2	Region 3	Region 4	Region 5	Region 6
Region 1	γ_1[a]	δ_2	δ_3	δ_4	δ_5	δ_6
Region 2	δ_1	γ_2	δ_3	δ_4	δ_5	δ_6
Region 3	δ_1	δ_2	γ_3	δ_4	δ_5	δ_6
Region 4	δ_1	δ_2	δ_3	γ_4	δ_5	δ_6
Region 5	δ_1	δ_2	δ_3	δ_4	γ_5	δ_6
Region 6	δ_1	δ_2	δ_3	δ_4	δ_5	γ_6

[a] The parameters arrayed in this table are region-specific rather than park-specific or wilderness area-specific. For example, δ_1 is the parameter associated with visibility at " Class I areas in region 1" for a household in any region other than region 1. The benefits analysis must derive Class I area-specific parameters – e.g., δ_{1k}, for the kth Class I area in the first region.

For the three regions covered in Chestnut and Rowe (1990a) (California, the Colorado Plateau, and the Southeast United States), we can directly use the in-region WTP estimates from the study to estimate the parameters in the utility functions corresponding to visibility at in-region parks (γ_1); similarly, we can directly use the out-of-region WTP estimates from the study to estimate the parameters for out-of-region parks (δ_1). For the other three regions not covered in the study, however, we must rely on benefits transfer to estimate the necessary parameters.

While Chestnut and Rowe (1990a) provide useful information on households' WTP for visibility improvements in national parks, there are several significant gaps remaining between the information provided in that study and the information necessary for the benefits analysis. First, as noted above, the WTP responses were not park-specific, but only region-specific. Because visibility improvements vary from one park in a region to another, the benefits analysis must value park-specific visibility changes. Second, not all Class I areas in each of the three regions considered in the study were included on the maps shown to study subjects. Because the focus of the study was primarily national parks, most Class I wilderness areas were not included. Third, only three regions of the United States were included, leaving the three remaining regions without direct WTP estimates.

In addition, Chestnut and Rowe (1990a) elicited WTP responses for *three different* visibility changes, rather than a single change. In theory, if the CES utility function accurately describes household preferences, and if all households in a region have the same preference structure, then households' three WTP responses corresponding to the three different visibility changes should all produce the same value of the associated recreational visibility parameter, given a value of ρ and an income, m. In practice, of course, this is not the case.

In addressing these issues, we take a three-phase approach:

(1) We estimate region-specific parameters for the region in the modeled domain covered by Chestnut and Rowe (1990a) (California, the Colorado Plateau, and the Southeast) – γ_1, γ_2, and γ_3 and δ_1, δ_2, and δ_3. **(2)** We infer region-specific parameters for those regions not covered by the Chestnut and Rowe study (the Northwest United States, the Northern Rockies, and the rest of the U.S.) – γ_4, γ_5, and γ_6

and δ_4, δ_5, and δ_6. **(3)** We derive park- and wilderness area-specific parameters within each region (γ_{1k} and δ_{1k}, for k=1, ..., N_1; γ_{2k} and δ_{2k}, for k=1, ..., N_2; and so forth).

The question that must be addressed in the first phase is how to estimate a single region-specific in-region parameter and a single region-specific out-of-region parameter for each of the three regions covered in Chestnut and Rowe (1990a) from study respondents' WTPs for *three different* visibility changes in each region. All parks in a region are treated collectively as if they were a single "regional park" in this first phase. In the second phase, we infer region-specific recreational visibility parameters for regions not covered in the Chestnut and Rowe study (the Northwest United States, the Northern Rockies, and the rest of the U.S.). As in the first phase, we ignore the necessity to derive park-specific parameters at this phase. Finally, in the third phase, we derive park- and wilderness area-specific parameters for each region.

Estimating Region-Specific Recreational Visibility Parameters for the Region Covered in the Chestnut and Rowe Study (Regions 1, 2, and 3)

Given a value of ρ and estimates of m and in-region and out-of-region WTPs for a change from Q_0 to Q_1 in a given region, the in-region parameter, γ, and the out-of-region parameter, δ, for that region can be solved for. Chestnut and Rowe (1990a), however, considered not just one, but three visibility changes in each region, each of which results in a different calibrated γ and a different calibrated δ, even though in theory all the γ's should be the same and similarly, all the δ's should be the same. For each region, however, we must have only a single γ and a single δ.

Denoting $\hat{\gamma}_j$ as our estimate of γ for the jth region, based on all three visibility changes, we chose $\hat{\gamma}_j$ to best predict the three WTPs observed in the study for the three visibility improvements in the jth region. First, we calculated $\hat{\gamma}_{ji}$, i=1, 2, 3, corresponding to each of the three visibility improvements considered in the study. Then, using a grid search method beginning at the average of the three $\hat{\gamma}_{ji}$'s, we chose $\hat{\gamma}_j$ to minimize the sum of the squared differences between the WTPs we predict using $\hat{\gamma}_j$ and the three region-specific WTPs observed in the study. That is, we selected $\hat{\gamma}_j$ to minimize:

$$\sum_{i=1}^{3} (WTP_{ij}(\hat{\gamma}_j) - WTP_{ij})^2$$

where WTP_{ij} and $WTP_{ij}(\hat{\gamma}_j)$ are the observed and the predicted WTPs for a change in visibility in the jth region from $Q_0 = Q_{0i}$ to $Q_1 = Q_{1i}$, i=1, ..., 3. An analogous procedure was used to select an optimal δ, for each of the three regions in the Chestnut and Rowe study.

Inferring Region-Specific Recreational Visibility Parameters for Regions Not Covered in the Chestnut and Rowe Study (Regions 4, 5, and 6)

One possible approach to estimating region-specific parameters for regions not covered by Chestnut and Rowe (1990a) (γ_4, γ_5, and γ_6 and δ_4, δ_5, and δ_6) is to simply assume that households' utility functions are the same everywhere, and that the environmental goods being valued are the same – e.g., that a change in visibility at national parks in California is the same environmental good to a Californian as a change in visibility at national parks in Minnesota is to a Minnesotan.

For example, to estimate δ_4 in the utility function of a California household, corresponding to visibility at national parks in the Northwest United States, we might assume that out-of-region WTP for a given visibility change at national parks in the Northwest United States is the same as out-of-region WTP for the same visibility change at national parks in California (income held constant). Suppose, for example, that we have an estimated mean WTP of out-of-region households for a visibility change from Q_{01} to Q_{11} at national parks in Califonia (region 1), denoted WTP_1^{out}. Suppose the mean income of the out-of-region subjects in the study was m. We might assume that, for the same change in visibility at national parks in the Northwest United States, $WTP_4^{out} = WTP_1^{out}$ among out-of-region individuals with income m.

We could then derive the value of δ_4, given a value of ρ as follows:

$$\delta_4 = \frac{(m - WTP_4^{out})^\rho - m^\rho}{Q_{04}^\rho - Q_{14}^\rho}$$

where $Q_{04} = Q_{01}$ and $Q_{14} = Q_{11}$, (i.e., where it is *the same* visibility change in parks in region 4 that was valued at parks in the region 1).

This benefits transfer method assumes that (1) all households have the same preference structures and (2) what is being valued in the Northwest United States (by a California household) is the same as what is being valued in the California (by all out-of-region households). While we cannot know the extent to which the first assumption approximates reality, the second assumption is clearly problematic. National parks in one region are likely to differ from national parks in another region in both quality and quantity (i.e., number of parks).

One statistic which is likely to reflect both the quality and quantity of national parks in a region is the average annual visitation rate to the parks in that region. A reasonable way to gauge the extent to which out-of-region people would be willing to pay for visibility changes in parks in the Northwest United States versus in California might be to compare visitation rates in the two regions.[47] Suppose, for example, that twice as many visitor-days are spent in California parks per year as in parks in the Northwest United States per year. This could be an indication that the parks in California are in some way more desirable than those in the Northwest United States and/or that there are more of them -- i.e., that the environmental goods being valued in the two regions ("visibility at national parks") are not the same.

A preferable way to estimate δ_4, then, might be to assume the following relationship:

$$\frac{WTP_4^{out}}{WTP_1^{out}} = \frac{n_4}{n_1}$$

(income held constant), where n_1 = the average annual number of visitor-days to California parks and n_4 = the average annual number of visitor-days to parks in the Northwest United States. This implies that

[47] We acknowledge that reliance on visitation rates does not get at nonuse value.

$$WTP_4^{out} = \frac{n_4}{n_1} * WTP_1^{out}$$

for the same change in visibility in region 4 parks among out-of-region individuals with income m. If, for example, $n_1 = 2n_4$, WTP_4^{out} would be half of WTP_1^{out}. The interpretation would be the following: California national parks have twice as many visitor-days per year as national parks in the Northwest United States; therefore they must be twice as desirable/plentiful; therefore, out-of-region people would be willing to pay twice as much for visibility changes in California parks as in parks in the Northwest United States; therefore a Californian would be willing to pay only half as much for a visibility change in national parks in the Northwest United States as an out-of-region individual would be willing to pay for the same visibility change in national parks in California. This adjustment, then, is based on the premise that the environmental goods being valued (by people out-of-region) are not the same in all regions.

The parameter δ_4 is estimated as shown above, using this adjusted WTP_4^{out}. The same procedure is used to estimate δ_5 and δ_6. We estimate γ_4, γ_5, and γ_6 in an analogous way, using the in-region WTP estimates from the transfer regions, e.g.,

$$WTP_4^{in} = \frac{n_4}{n_1} * WTP_1^{in} \quad .$$

Estimating Park- and Wilderness Area-Specific Parameters

As noted above, Chestnut and Rowe (1990a) estimated WTP for a region's national parks collectively, rather than providing park-specific WTP estimates. The γ's and δ's are therefore the parameters that would be in household utility functions if there were only a single park in each region, or if the many parks in a region were effectively indistinguishable from one another. Also noted above is the fact that the Chestnut and Rowe study did not include all Class I areas in the regions it covered, focusing primarily on national parks rather than wilderness areas. Most Class I wilderness areas were not represented on the maps shown to study subjects. In California, for example, there are 31 Class I areas, including 6 national parks and 25 wilderness areas. The Chestnut and Rowe study map of California included only 10 of these Class I areas, including all six of the national parks. It is unclear whether subjects had in mind "all parks and wilderness areas" when they offered their WTPs for visibility improvements, or whether they had in mind the specific number of (mostly) parks that were shown on the maps. The derivation of park- and wilderness area-specific parameters depends on this.

Derivation of Region-specific WTP for National Parks and Wilderness Areas

If study subjects were lumping all Class I areas together in their minds when giving their WTP responses, then it would be reasonable to allocate that WTP among the specific parks and wilderness areas in the region to derive park- and wilderness area-specific γ's and δ's for the region. If, on the other hand, study subjects were thinking only of the (mostly) parks shown on the map when they gave their WTP response, then there are two possible approaches that could be taken. One approach assumes that households would be willing to pay some additional amount for the same visibility improvement in additional Class I areas that were not shown, and that this additional amount can be estimated using the same benefits transfer approach used to estimate region-specific WTPs in regions not covered by Chestnut and Rowe (1990a).

However, even if we believe that households would be willing to pay some additional amount for the same visibility improvement in additional Class I areas that were not shown, it is open to question whether this additional amount can be estimated using benefits transfer methods. A third possibility, then, is to simply omit wilderness areas from the benefits analysis. For this analysis we calculate visibility benefits assuming that study subjects lumped all Class I areas together when stating their WTP, even if these Class I areas were not present on the map.

Derivation of park- and wilderness area-specific WTPs, given region-specific WTPs for national parks and wilderness areas

The first step in deriving park- and wilderness area-specific parameters is the estimation of park- and wilderness area-specific WTPs. To derive park and wilderness area-specific WTPs, we apportion the region-specific WTP to the specific Class I areas in the region according to each area's share of the region's visitor-days. For example, if WTP_1^{in} and WTP_1^{out} denote the mean household WTPs in the Chestnut and Rowe (1990a) study among respondents who were in-region-1 and out-of-region-1, respectively, n_{1k} denotes the annual average number of visitor-days to the kth Class I area in California, and n_1 denotes the annual average number of visitor-days to all Class I areas in California (that are included in the benefits analysis), then we assume that

$$WTP_{1k}^{in} = \frac{n_{1k}}{n_1} * WTP_1^{in} ,$$

and

$$WTP_{1k}^{out} = \frac{n_{1k}}{n_1} * WTP_1^{out} .$$

Using WTP_j^{in} and WTP_j^{out}, either from the Chestnut and Rowe study (for j = 1, 2, and 3) or derived by the benefits transfer method (for j = 4, 5, and 6), the same method is used to derive Class I area-specific WTPs in each of the six regions.

While this is not a perfect allocation scheme, it is a reasonable scheme, given the limitations of data. Visitors to national parks in the United States are not all from the United States, and certainly not all from the region in which the park is located. A very large proportion of the visitors to Yosemite National Park in California, for example, may come from outside the U.S. The above allocation scheme implicitly assumes that the relative frequencies of visits to the parks in a region *from everyone in the world* is a reasonable index of the relative WTP of an average household in that region (WTP_j^{in}) or out of that region (but in the U.S.) (WTP_j^{out}) for visibility improvements at these parks.[48]

A possible problem with this allocation scheme is that the relative frequency of visits is an indicator of use value but not necessarily of nonuse value, which may be a substantial component of the household's total WTP for a visibility improvement at Class I areas. If park A is twice as popular (i.e., has twice as many visitors per year) as park B, this does not necessarily imply that a household's WTP for

[48] This might be thought of as two assumptions: (1) that the relative frequencies of visits to the parks in a region *from everyone in the world* is a reasonable representation of the relative frequency of visits *from people in the United States* – i.e., that the parks that are most popular (receive the most visitors per year) in general are also the most popular among Americans; and (2) that the relative frequency with which Americans visit each of their parks is a good index of their relative WTPs for visibility improvements at these parks.

an improvement in visibility at park A is twice its WTP for the same improvement at park B. Although an allocation scheme based on relative visitation frequencies has some obvious problems, however, it is still probably the best way to allocate a collective WTP.

Derivation of park- and wilderness area-specific parameters, given park- and wilderness area-specific WTPs

Once the Class I area-specific WTPs have been estimated, we could derive the park- and wilderness area-specific γ's and δ's using the method used to derive region-specific γ's and δ's. Recall that that method involved (1) calibrating γ and δ to each of the three visibility improvements in the Chestnut and Rowe study (producing three γ's and three δ's), (2) averaging the three γ's and averaging the three δ's, and finally, (3) using these average γ and δ as starting points for a grid search to find the optimal γ and the optimal δ – i.e., the γ and δ that would allow us to reproduce, as closely as possible, the three in-region and three out-of-region WTPs in the study for the three visibility changes being valued.

Going through this procedure for each national park and each wilderness area separately would be very time consuming, however. We therefore used a simpler approach, which produces very close approximations to the γ's and δ's produced using the above approach. If:

WTP_j^{in} =	the in-region WTP for the change in visibility from Q_0 to Q_1 in the jth region;
WTP_{jk}^{in}=	the in-region WTP for the same visibility change (from Q_0 to Q_1) in the kth Class I area in the jth region (= s_{jk}*WTP_j^{in}, where s_{jk} is the kth area's share of visitor-days in the jth region);
m =	income;
γ_j* =	the optimal value of γ for the jth region; and
γ_{jk} =	the value of γ_{jk} calibrated to WTP_{jk}^{in} and the change from Q_0 to Q_1;

then[49]:

$$\gamma_j^* \approx \frac{(m - WTP_j^{in})^\rho - m^\rho}{(Q_0^\rho - Q_1^\rho)}$$

and

$$\gamma_{jk} = \frac{(m - WTP_{jk}^{in})^\rho - m^\rho}{(Q_0^\rho - Q_1^\rho)}$$

which implies that:

$$\gamma_{jk} \approx a_{jk} * \gamma_j^* ,$$

[49] γ_j* is only approximately equal to the right-hand side because, although it is the optimal value designed to reproduce as closely as possible all three of the WTPs corresponding to the three visibility changes in the Chestnut and Rowe study, γ_j* will not exactly reproduce any of these WTPs.

where:

$$a_{jk} = \frac{(m - WTP_{jk}^{in})^{\rho} - m^{\rho}}{(m - WTP_{j}^{in})^{\rho} - m^{\rho}} .$$

We use the adjustment factor, a_{jk}, to derive γ_{jk} from γ_j*, for the kth Class I area in the jth region. We use an analogous procedure to derive δ_{jk} from δ_j* for the kth Class I area in the jth region (where, in this case, we use WTP_j^{out} and WTP_{jk}^{out} instead of WTP_j^{in} and WTP_{jk}^{in}).[50]

5.1.4 Estimating the Parameter for Visibility in Residential Areas: θ

The estimate of θ is based on McClelland et al. (1991), in which household WTP for improvements in residential visibility was elicited from respondents in Chicago and Atlanta. A notable difference between the Chestnut and Rowe study and the McClelland study is that, while the former elicited WTP responses for three different visibility changes, the latter considered only one visibility change. The estimation of θ was therefore a much simpler procedure, involving a straightforward calibration to the single income and WTP in the study:

$$\theta = \frac{(m - WTP)^{\rho} - m^{\rho}}{(Z_0^{\rho} - Z_1^{\rho})} .$$

5.1.5 Putting it All Together: the Household Utility and WTP Functions

Given an estimate of θ, derived as shown in Section 5, and estimates of the γ's and δ's, derived as shown in Section 4, based on an assumed or estimated value of ρ, the utility and WTP functions for a household in any region are fully specified. We can therefore estimate the value to that household of visibility changes from any baseline level to any alternative level in the household's residential area and/or at any or all of the Class I areas in the United States, in a way that is consistent with economic theory. In particular, the WTP of a household in the ith region and the nth residential area for any set of changes in the levels of visibility at in-region Class I areas, out-of-region Class I areas, and the household's residential area (given by equation (24)) is:

$$WTP_{ni}(\Delta Z, \Delta Q) = m - [m^{\rho} + \theta(Z_{0n}^{\rho} - Z_{1n}^{\rho}) + \sum_{k=1}^{N_i} \gamma_{ik}(Q_{0ik}^{\rho} - Q_{1ik}^{\rho}) + \sum_{j \neq i} \sum_{k=1}^{N_j} \delta_{jk}(Q_{0jk}^{\rho} - Q_{1jk}^{\rho})]^{1/\rho} .$$

The national benefits associated with any suite of visibility changes is properly calculated as the sum of these household WTPs for those changes. The benefit of any subset of visibility changes (e.g., changes in visibility only at Class I areas in California) can be calculated by setting all the other components of the WTP function to zero (that is, by assuming that all other visibility changes that are not of interest are zero). This is effectively the same as assuming that the subset of visibility changes of interest is the first or the only set of changes being valued by households. Estimating benefit components

[50] This method uses a single in-region WTP and a single out-of-region WTP per region. Although the choice of WTP will affect the resulting adjustment factors (the a_{jk}'s) and therefore the resulting γ_{jk}'s and δ_{jk}'s, the effect is negligible. We confirmed this by using each of the three in-region WTPs in California and comparing the resulting three sets of γ_{jk}'s and δ_{jk}'s, which were different from each other by about one one-hundredth of a percent.

in this way will yield slightly upward biased estimates of benefits, because disposable income, m, is not being reduced by the WTPs for any prior visibility improvements. That is, each visibility improvement (e.g., visibility at Class I areas in the California) is assumed to be the first, and they cannot all be the first. The upward bias should be extremely small, however, because all of the WTPs for visibility changes are likely to be very small relative to income.

5.2 Agricultural Benefits

Changes in ozone concentrations are known to affect agricultural production, affecting agricultural crops to different degrees depending on their sensitivity. Estimating the economic benefits associated with these changes in production requires several steps. Estimated changes in ozone concentrations are combined with experimental dose-response functions to estimate crop yield changes. The effect of yield changes on agricultural cropping decisions and resulting production and prices are then evaluated using a model of the agricultural sector, resulting in estimates of changes in farm income and consumer welfare. Each of the steps involved in this analysis is described in more detail in the following sections. Section 5.2.1 describes the source of exposure-response functions and the selection of an index of ozone exposure. Section 5.2.2 describes the derivation of estimated ozone concentrations under alternative regulatory profiles. The method for estimating yield changes is described in Section 5.2.3, and the agricultural model used to estimate the impact of changes in yield is discussed in Section 5.2.4. The results are presented in Chapter 6.

5.2.1 Exposure-Response Functions

Experimental data to evaluate the response of crops to ozone has been collected for a limited number of crops under the National Crop Loss Assessment Network (NCLAN) program. The objective of this program was to employ a consistent experimental methodology to provide comparable results across crops. The crops included in the NCLAN experiments are corn, cotton, peanuts, sorghum, soybeans, winter wheat, potatoes, lettuce, kidney beans, tomatoes, and hay. For many crops, the NCLAN program evaluated the effects of ozone on several different cultivars. Although not necessarily representative of the full range of variability in crop response, the results for different cultivars do permit identification of a range of responsiveness. The most tolerant and responsive functions are used to represent minimum and maximum impacts, *within the limits of available data*.

In its analysis of the welfare benefits associated with ozone National Ambient Air Quality Standards (NAAQS), U.S. EPA elected to represent crop exposure to ozone as a cumulative index (U.S. EPA, 1996a). The index selected is the SUM06 index, which sums the ozone concentration for every hour that exceeds 0.06 ppm, within a 12-hour period from 8:00 A.M. to 8:00 P.M.

Use of cumulative exposure-response functions is relatively recent, and few experiments have been designed or reported in terms of the SUM06 index. Because the NCLAN program used a consistent protocol and developed a database of experimental conditions and results for all of its studies, U.S. EPA's Environmental Research Laboratory (ERL) was able to use original data from NCLAN studies to develop SUM06 exposure response functions for most NCLAN crops[51] (Lee and Hogsett, 1996). In addition, the agricultural model used in this analysis does not reflect non-commodity crops such as lettuce and kidney beans (described below). Exhibit 5-3 presents the exposure-response functions used in this analysis.

[51] Data were not sufficient to develop functions for tomatoes or hay.

Exhibit 5-3. Ozone Exposure-Response Functions for Selected Crops (SUM06)

Ozone Index	Quantity	Crop	Function	Median Experimental Duration (Days)	Median Duration (Months)
SUM06	Max	Cotton	1-exp(-(index/78)^1.311)	119	4
SUM06	Max	Field Corn	1-exp(-(index/92.4)^2.816)	83	3
SUM06	Max	Grain Sorghum	1-exp(-(index/177.8)^2.329)	85	3
SUM06	Max	Peanut	1-exp(-(index/99.8)^2.219)	112	4
SUM06	Max	Soybean	1-exp(-(index/131.4)^1)	104	3
SUM06	Max	Winter Wheat	1-exp(-(index/27.2)^1.0)	58	2
SUM06	Min	Cotton	1-exp(-(index/116.8)^1.523)	119	4
SUM06	Min	Field Corn	1-exp(-(index/94.2)^4.307)	83	3
SUM06	Min	Grain Sorghum	1-exp(-(index/177.8)^2.329)	85	3
SUM06	Min	Peanut	1-exp(-(index/99.8)^2.219)	112	4
SUM06	Min	Soybean	1-exp(-(index/299.7)^1.547)	104	3
SUM06	Min	Winter Wheat	1-exp(-(index/72.1)^2.353)	58	2

Source: Lee and Hogsett (1996)

The form of these functions is a Weibull specification transformed to predict a yield loss relative to conditions of "clean air", or a zero SUM06 value. The resulting equation is in the form of:

$$Y = 1 - e^{[-(SUM06/B)^C]},$$

where:

Y	=	predicted relative yield loss (PRYL), expressed as a decimal value (i.e., not multiplied by 100 to report as a percent loss), and relative to a zero SUM06 (or clean air) condition
SUM06	=	cumulative SUM06 ozone statistic at a specified level of spatial representation, in ppm
B, C	=	statistically estimated parameters, unitless.

Application of Exposure Response Functions to a Non-Zero Baseline

There is an issue associated with applying the yield loss functions to analysis of alternative regulatory profiles. The functions provide a predicted yield loss relative to "clean" air, while regulatory analysis needs to compare regulatory options to a baseline, non-zero ozone condition. Therefore, the yield change resulting from the regulatory scenario is evaluated as the yield loss relative to clean air under the regulatory scenario being evaluated compared to the yield loss under baseline conditions.

To address this issue, the change in yield under clean air conditions can be divided by the baseline yield. If yield under clean conditions is 100 percent of possible yield, then baseline yield in this context is 1 minus baseline yield loss. Thus the change in yields relative to the baseline can be given as:

$$(PRYL_{baseline} - PRYL_{control})/(1-PRYL_{baseline}).$$

Ozone Index Computation

In order to accurately reflect changes in yields using exposure response functions, they must be applied in a way that is consistent with the experimental conditions used to generate the functions. Specifically, the ozone index, in this case the SUM06 index, needs to be consistent with ozone exposure used in the experimental derivation of the function. For example, if the function is a 12-hour exposure function, then the index used must be a 12-hour index. Another component of the experimental exposure is the duration of the experiment. A precise reflection of experimental conditions would require that the ozone index should be calculated for the same number of days as used in the experiment for each crop. However, in the benefits analysis for the 1997 ozone NAAQS RIA, it was determined that the median duration of all NCLAN experiments for a given crop provided a statistically sound reflection of duration for the purposes of estimating SUM06 indices for estimating agricultural benefits (Mathtech1997). The median durations for each crop are reported in Exhibit 5-3 in both days and months. The ozone NAAQS analysis constructed the ozone index based on the nearest number of months; this analysis constructed the index based on the number of days.

Finally, because growing seasons vary throughout the U.S., the exposure needs to reflect the months in which a crop would be grown in a given location. To calculate the SUM06 index for the appropriate growing season, state-level data on planting and harvesting dates was used in this analysis[52] (U.S. Department of Agriculture, 1984; U.S. EPA, 1993). To calculate the cumulative SUM06 index, the experimental duration for each crop was anchored on that crop's harvest date in each state in order to most closely approximate the relevant period of exposure for yield analysis. The harvest date was assumed to be the first day in the month of harvest, so that the SUM06 index includes the months up to but not including the harvest month.

The baseline and control ozone data for this analysis were developed from monthly SUM06 values, requiring several steps in the calculation of a duration-based index. First, starting at the month before the harvest month, each full month of SUM06 data was summed. The ozone value for the first month of the duration period was calculated as the fraction of the remaining days in the duration period to the number of days in the month. For example, soybeans have a 104-day duration, translating to 3 full months plus a fraction of the first month in the growing season. If soybeans are harvested in October in a given state, three full months of data starting in September are summed (91 days), along with 13 days of June, or 0.43 of the June SUM06 data, to obtain the 104-day SUM06 index. This approach implicitly assumes an equal average daily SUM06 within each bi-monthly period. The index was calculated on a county level assuming all counties reflect the state-level growing seasons.

While the ozone data in this analysis were modeled from May through September, the growing season for some crops includes April, October, and November. To estimate SUM06 values for these unmodeled months, base-year ozone values were used.

5.2.2 Estimation of Yield Changes

In this analysis, use of a single exposure response function to estimate changes in yields implies that all producers are using a single cultivar of a given crop. This, combined with the limited number of

[52] Peanut emergence and harvest dates were taken from the U.S. EPA PRZM-2 Model data.

cultivars evaluated in the NCLAN program, introduces an unquantifiable uncertainty into the estimation of yield changes. The most sensitive cultivar was used to represent the upper bound of the range that could be estimated, and the least sensitive cultivar was used to represent the lower bound of that range.

Using the exposure response functions and the SUM06 ozone indices, county-level yield changes were estimated between each regulatory profile and the baseline. County level yield changes were then aggregated to the state level using 1997 data on county level production as weights (U.S. Department of Agriculture, 1988a): the resulting state-level yield changes were used for quality control purposes. The model used to estimate changes in the agricultural sector resulting from yield changes (described below) requires a national level yield change; this was calculated in the same manner as was the change in state-level yields.

5.2.3 AGSIM© Model

AGSIM© is an econometric-simulation model that is based on a large set of statistically estimated demand and supply equations for agricultural commodities produced in the United States. This model has been peer-reviewed and utilized in many pesticide and other major agricultural policy evaluations (Taylor et al., 1993).

The model is capable of analyzing the effects of changes in policies that affect crop yields or production costs. This is achieved by estimating how farmers will adjust crop acreage between commodities when relative profitability changes as a result of policy-induced crop yield and/or production cost changes.[53] Acreage and yield changes from various scenarios will affect total production of crops, which simultaneously affects both commodity prices and consumption. Commodity price changes, in turn, affect profitability and cropping patterns in subsequent years. Federal farm program and conservation reserve effects are also incorporated into the model. The model has been adapted to reflect the projections to 2010 from the last future year for which baseline forecasts are available: 2007. Although ozone impacts will be experienced far in the future, it was not possible to forecast the AGSIM© model far beyond USDA baseline forecasts that extend to 2007. Therefore, the 2030 ozone conditions were modeled using the 2010 version of the model.

Model Specification

AGSIM© is based on a set of dynamic supply and demand equations for major crops. Commodities are generally linked on both the supply side and demand side of markets. Crops included in the model are corn, grain sorghum, barley, oats, wheat, soybeans, cotton, hay, peanuts and rice. The simulation component of the model finds the set of prices for all commodities endogenous to the model that simultaneously clear all markets in each year over the simulation period. Dynamics are incorporated into the econometric specification and thus incorporated into the simulation model. All equations in the model were econometrically estimated, except a few policy equations that were based on legislated formula.

Supply Components

[53] To the extent that the Rule increases diesel prices, shipping prices for some agricultural products may increase, and may cause some farmers to change their production decisions. The magnitude of such an impact is likely to be small. Time and resources did not permit modeling this possible impact on their decision-making.

The crop supply component of AGSIM© is based on a set of supply equations for the major field crops produced in the United States. Effects of farm programs, specifically the 1985 Food Security Act (FSA), the 1990 Food Agricultural Conservation and Trade Act (FACTA), and the 1996 Federal Agricultural Improvement and Reform Act (FAIR), are reflected in the econometric specification of the supply component of the model, and thus are included in the simulation model.

Ex ante simulation of environmental policy will likely involve an assumption of continuation of the 1996 FAIR Act indefinitely. However, since most of the historical observations on which supply equations were econometrically estimated occurred under different programs, it is important to consider how historical equations reflect the 1996 FAIR Act. The basic philosophy that guided inclusion of farm program features into the supply component of the model follow. First, beginning with the 1985 FSA, continuing with the 1990 FACTA, and now with the 1996 FAIR Act, North American Free Trade Agreement (NAFTA) and the General Agreement on Tariffs and Trade (GATT), farm and international trade policy has moved U.S. agriculture to a market orientation. Although the 1985 FSA and the 1990 FACTA had price support and acreage diversion features, they embodied a strong market orientation. For all major program crops (in AGSIM©), the acreage devoted to the crop exceeded the acreage under government programs. Thus, *at the margin,* market prices (and not support prices) influenced crop acreage. Another way of looking at this is that farm programs have influenced crops at the intra-margin, while the market has influenced crops at the margin. Thus, after accounting for acreage diverted under farm programs, expected prices determine acreage. For these reasons, AGSIM© should be valid for simulating agricultural markets under the market conditions established under the 1996 FAIR Act.

Sets of equations that comprise the supply component of the current version of the model include: (1) acreage planted to each crop, (2) acreage harvested of each crop, (3) acreage in annual set-aside or acreage reduction programs (ARP) by crop, (4) acreage in cultivated summer fallow, (5) crop yields per harvested acre, (6) rate of participation in Federal farm programs by crop, and (7) annual set-aside rates by crop under past farm programs, as related to stock levels (historically legislated) and thus related to market price. Identities in the model are: (a) production is the product of acreage harvested and yield per harvested acre, and (b) the quantity supplied equals the quantity demanded for each commodity (market clearing). Specification of each of these sets of equations follows.

Acreage Planted Equations. Acreage planted is the key behavioral relationship in the supply component of the model. Acreage planted of a particular crop depends on expected per-acre net returns for that crop, expected per-acre net returns for competing crops, and farm program variables. In algebraic (and Fortran) form, the acreage planted equation is:

(1) acresp(ic,it,irun) = bc(ic) + bap(ic)*acresp(ic,it-1,irun) + bcrp(ic)*acrp(ic,it,irun) + bdiv(ic)*acrediv + brm(ic)*rerntm(ic,it,irun) + ber(ic)*rerentnp(it,irun) + byr(ic)*time(it) + bd83(ic)*dumb83(it)

where:

acresp(ic,it,irun)	=	acreage planted to the icth crop in the itth year and in simulation "irun",
acrp(ic,it,irun)	=	acreage of crop "ic" that was placed in the conservation reserve program,
acrediv	=	acreage diverted under annual set-aside programs,
rerentm(ic,it,irun)	=	real expected per acre returns over variable costs for the icth crop,

rerentnp(it,irun)	=	real expected per acre returns over variables costs computed as a weighted average[54] of rerentm(ic,it,irun) over all endogenous crops,
time(it)	=	a time-trend variable, and
dumb83(it)	=	a binary dummy variable to account for the PIK program in crop year 1983.

The remaining variables in equation (1) represent estimated coefficients. A single run of AGSIM involves two simulations, one for the baseline (irun=0) and one for the policy scenario (irun=1). These two simulations are then compared to estimate the economic impacts of the policy scenario.

Expected returns over variable costs, rerentm(ic,it,irun), is defined as:

(1a) rerntm(ic,it,irun) = rp(ic,it-1,irun)*ey(ic,it,irun) - rcost(ic,it,irun)

where:

rp(ic,it-1,irun)	=	real price the previous crop year (actual or simulated, depending on the time period),
ey(ic,it,irun)	=	expected crop yield, and
rcost(ic,it,irun)	=	real variable production cost.

Expected yield is based on trend-line regressions:

(1b) ey(ic,it,irun) = [cint(ic) + by(ic)*time(it)]

where:

cint(ic) and by(ic) are estimated coefficients.

In the policy run, expected yield is adjusted for exogenously specified percentage yield changes ("dyld"):

(1c) ey(ic,it,irun) = [cint(ic) + by(ic)*time(it)]*(1.0 + dyld(ic,it)/100.)

Changes in real variable costs of production can also be exogenously specified for the policy simulation run. Thus, yield and cost changes directly impact acreage planted through equation (1), and indirectly impact acreage planted because of the resulting impact on prices in equation (1a) and thus in equation (1).

Given signs and magnitudes of estimated coefficients in equation (1), an increase in expected returns of the icth crop will increase acreage planted of that crop, while an increase in expected returns of other endogenous crops will decrease acreage of the icth crop. The estimated coefficient on lagged acreage planted in equation (1) is positive and less than one in value for all crops, which means that acreage planted is dynamically stable. The estimated coefficient on the set-aside acreage is negative and less than one in absolute value for all crops except oats, which reflects acreage slippage in the ARP program. Oats were typically planted to set-aside acreage, thus the estimated coefficient on set-aside acreage is positive in the oats equation, as expected. Further comments will be made on the acreage diverted effects on planted acreage after participation rate and acreage diverted equations, which are endogenous, are presented below.

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[54] Weights used in computing a composite expected return variable were the acreage harvested of each crop the previous year divided by total acreage harvested the previous year.

Acreage Harvested Equations. Acreage harvested depends primarily on acreage planted:

(2) acresh(ic,it,irun) = bch(ic) + baph(ic)*acresp(ic,it,irun) + byrh(ic)*time(it) + bdvh(ic)*acrediv

where:

acresh(ic,it,irun) = the acreage harvested of the icth crop in the itth year and in simulation "irun",

and other variables are as defined previously.

The estimated coefficient baph(ic) is positive and less than one, indicating that not all planted acreage is harvested, as expected. The coefficient bdvh(ic) on the acreage diverted variable is non-zero for oats only, in which case it is negative. This adjusts oat acreage harvested for the complexity of oats being planted (but not harvested) on ARP acreage. A time-trend variable for corn and grain sorghum, but not other crops shows how harvested acreage as a percentage of planted acreage has been increasing slightly over time.

Participation Rate in Farm Programs. Participation rates in the annual set-aside programs under the 1985 FSA and the 1990 FACTA were endogenized in the model with the set of equations:

(3) part(ic,it,irun) = bcp(ic) + brmp(ic)*rerntm(ic,it,irun) + brpp(ic)*rerntp(ic,it,irun) + byr(ic)*time(ic) + bpart(ic)*part(ic,it-1,irun) + bedpp(ic)*redp(ic,it,irun) + bd83p(ic)*dumb83(it)

where:

part(ic,it,irun) = the participation rate in the farm program for the icth crop in the itth year and in simulation "irun",

rerntp(ic,it,irun)= real expected returns over variable costs based on the support (target) price for that crop,

redp(ic,it,irun) = real effective acreage diversion payment rate,

and other variables are as defined previously.

Estimated coefficients brpp(ic) are non-negative, indicating that an increase in expected returns based on support price will increase participation, while estimated coefficients brmp(ic) are non-positive, indicating that an increase in expected returns based on expected market price will decrease participation. Lagged participation rate in equation (3) shows strong dynamics with respect to farm program participation.

Acreage Diverted under Farm Programs. Acreage diverted under annual set-aside (or ARP) programs is modeled as:

(4) adiv(ic,it,irun) = bcd(ic) + bd83d(ic)*dumb83(it) + bedpd(ic)*redp(ic,it,irun) + byrd(ic)*time(it) + bpsa(ic)*sa(ic,it,irun)*part(ic,it,irun)

where:

adiv(ic,it,irun) = acreage diverted under annual diversion programs for the icth crop in the itth year and in simulation "irun",

sa(ic,it,irun) = the set-aside rate specified by the Secretary of Agriculture under 1985 FSA and 1990 FACTA,

and other variables are as defined previously.

Acreage slippage (with respect to historical set-aside) in farm programs is implicit in the model specification, and results from the complex simultaneity of farm program variables in sets of equations (1), (3), and (4).

Acreage in Cultivated Summer Fallow. Acreage in cultivated summer fallow is modeled by the equation:

(5) afl(it,irun) = bcfl + bafl*afl(it-1,irun) + berfl*rerentnp(it,irun) + byrfl*time(it) + bd83fl*dumb83(it)

where:

afl(it,irun) = acreage fallowed in year it in simulation run "irun".

Although the acreage in cultivated summer fallow is highly inelastic, this equation shows that an increase in expected returns based on expected market price results in a small decrease in acreage fallowed.

Demand Components

The crop demand component of AGSIM© is based on a set of demand equations for each crop for utilization categories of (a) imports, (b) exports,(c) livestock feed, (d) food, fiber, ethanol production and other domestic uses, (e) ending stocks, and (f) residual use. Each demand component depends on current market price for that commodity and, where relevant, prices of other commodities. The model specification of each utilization category follows.

Imports. Imports of agricultural commodities are modeled by the set of equations:

(6) qd(ic,it,irun,1) = bim(1,ic) + bim(2,ic)*rp(ic,it,irun)*xrate(ic,it-1,irun) + bim(3,ic)*qd(ic,it-1,irun,1) + bim(4,ic)*time(it) + bim(5,ic)*uspop(it,irun)

where:

qd(ic,it,irun,1) = the quantity of crop ic imported in year it in simulation run "irun",
rp(ic,it,irun) = real market price,
xrate(ic,it-1,irun) = the real trade-weighted exchange rate,
uspop(it,irun) = the United States population,

and bim(j,ic) are estimated coefficients. Lagged imports in equation (6) reflects dynamic adjustments.

Exports. Exports of agricultural commodities are modeled by the set of equations:

(7) qd(ic,it,irun,2) = bex(1,ic) + bex(2,ic)*rp(ic,it,irun)*xrate(ic,it-1,irun) + bex(3,ic)* qd(ic,it-1,irun,2) + bex(4,ic)*time(it) + bex(5,ic)*wpop(it,irun)

where:

qd(ic,it,irun,2) = the quantity of crop ic exported in year it in simulation run "irun", and
wpop(it,irun) = world population.

Feed, Fiber and Crushing Use. Domestic utilization of crops for feed, fiber or crushing (depending on the crop) is modeled by the set of equations:

(8) $qd(ic,it,irun,3) = bfd(1,ic) + \sum_{jc} bfdcross(ic,jc)*rp(jc,it,irun) + bfd(2,ic)*qd(ic,it-1,irun,3) + bfd(3,ic)*time(it)$

where:

$qd(ic,it,irun,3)$ = utilization for feed, fiber or crushing.

Note that cross-price effects are incorporated into this set of equations through the set of estimated coefficients bfdcross(ic,jc). Symmetry of cross-price effects, consistent with microeconomic theory, was imposed on estimation so that bfdcross(ic,jc) = bfdcross(jc,ic) for $ic \neq jc$. Own-price effects are all negative, as expected.

Domestic Food Use. The set of equations to represent domestic food use is:

(9) $qd(ic,it,irun,4) = bfo(1,ic) + bfo(2,ic)*rp(ic,it,irun) + bfo(3,ic)*qd(ic,it-1,irun,4) + bfo(4,ic)*time(it) + bfo(5,ic)*uspop(it,irun) + bfo(6,ic)*rdincome(it,irun)$

where:

rdincome(it,irun) = real per-capita disposable income in the United States,

and other variables are as defined previously. In the case of peanuts, the real market price is replaced by the fixed quota price that applies to all domestically consumed peanuts. This quota price for peanuts applies to the 1985 FSA, the 1990 FACTA, and continues with the 1996 FAIR Act.

Ending Stocks. Ending stocks are viewed as another component of demand. Although commodities are often held to maintain pipeline inventories, commodities are also held for speculative purposes. Thus, stock levels respond strongly to prices, so the stock relationships were specified and estimated as

(10) $qd(ic,it,irun,5) = bst(1,ic) + bst(2,ic)*rp(ic,it,irun) + bst(3,ic)*qd(ic,it-1,irun,5) + bst(4,ic)*time(it)$

where qd(ic,it,irun,5) is ending stocks in year t.

Residual Use. For some crops (rice, peanuts, and cottonseed), supply and utilization data show a residual category, which is modeled as,

(11) $qd(ic,it,irun,6) = brs(1,ic) + brs(2,ic)*rp(ic,it,irun) + brs(3,ic)*time(it)$

where:

$qd(ic,it,irun,6)$ = residual use.

Although quantities in this residual use category are never used, the level of the residual does respond negatively to the real price, and is thus viewed as another utilization (demand) category.

Market Clearing Identities

In supply and demand specification outlined above, supply generally depends on past prices, while demand depends on current prices. In simulating these econometrically estimated equations into the future, simulated prices are solved by simultaneously solving the market clearing identities

(12) qs(ic,it,irun) +qd(ic,it-1,irun,5) = qd(ic,it,irun,1) + qd(ic,it,irun,2) + qd(ic,it,irun,3) + qd(ic,it,irun,4) + qd(ic,it,irun,5) + qd(ic,it,irun,6)

where:

qs(ic,it,irun) = the quantity produced of crop ic in year it in simulation "irun".

Production is defined to be qs(ic,it,irun) = acresh(ic,it,irun)*ey(ic,it,irun). The left hand side of the equal sign in (12) gives total supply (production plus beginning stocks), while the right-hand side of (12) gives total utilization, including ending stocks.

In the simulation model this set of simultaneous equations are numerically solved to get the market clearing prices in a given year. This process is continued, considering the dynamics of the model, indefinitely into the future.

Historical Observation Period

Many econometric relationships in the model were estimated with data for the 1975-1995 time period. However, where structural change was apparent, such as with stock holding behavior and international trade, some of the early years were dropped from statistical analysis so that the simulation model would better reflect the future.

Alternative Specifications Considered

Many different specifications of how farm programs influence crop acreage have been considered in the evolution of AGSIM©, including: (a) acreage depends on support price, (b) acreage depends on the maximum of expected market price and support price, (c) acreage depends on a weighted average of support and expected market prices, with weights based on program and non-program acreage of the crop, and (d) acreage depends on expected market price. Models for expected market price have considered complex distributed lags that go back several years in time, to a simple model that expected market price is actual price the previous year. Acreage equations have also been specified to depend on expected returns of: (1) all competing individual crops with no parameter restrictions, (2) all competing individual crops with full symmetry of cross-effects imposed on estimation, (3) major competing individual crops, and (4) a weighted average of all expected returns for all other crops. Many different ways of incorporating participation rates and acreage diverted into the model have also been considered. Several alternative functional forms (linear, log-linear, nonlinear share equations, asymptotic) have also been considered. Theoretical specifications considered have ranged from *ad hoc* models to very tightly specified and detailed theoretical economic models based on complex assumptions. The present model draws from economic theory (e.g. symmetry of cross-price effects in demand and homogeneity of degree zero of all supply and demand equations with respect to prices), but does not specify the model so tightly with untested assumptions and functional forms that empirical data has almost no role in the resulting estimates. Alternative estimation techniques, ranging from simultaneous equations techniques, to Zellner's seemingly unrelated regressions, to ordinary least squares regression have been used. The current version of AGSIM© reflects a degree of subjective judgement of what best reflects supply and demand of agricultural commodities based on microeconomic theory, traditional statistical criteria, and substantive direct contact with farmers and ranchers in most regions of the United States.

Baseline

The current version of AGSIM© is designed to estimate *changes* in the agricultural sector resulting from pesticide or other policy. Changes in economic variables are computed by comparing a policy simulation of the model with a baseline simulation of the model. For *ex post* (retrospective) evaluations, the baseline reflects actual farm programs, prices, acreages, etc. However, for *ex ante* evaluations, AGSIM© is calibrated to an external baseline. The calibration is done by comparing an internally generated baseline to the external baseline and computing adjusted intercepts for all of the relevant demand and supply relationships in AGSIM©**.**

For the 1999 version of AGSIM© the externally specified year 2010 baseline is forecasted from the 2007 baseline reported by USDA (1988b). A few endogenous variables in AGSIM© were not included in the USDA baseline. In those cases, the 1997 FAPRI baseline was used (FAPRI, 1997).

It should be noted that the baseline is not especially critical to estimates of *changes* in the agricultural sector, except for the case of price support policy, which is not relevant here. That is, sensitivity analyses with previous versions of AGSIM© have shown that estimates of *changes* in variables are not very sensitive to baseline absolute values of variables. Use of the USDA baseline to the extent possible assures consistency with other governmental mandated agricultural policy analyses.

Regional Effects Sub-Model

AGSIM© subroutines are also available to combine AGSIM© output with production cost information to estimate net farm income impacts for the policy scenario at the regional level (or farm, representative farm, area or state level). Required information for this type of evaluation includes for each farm or area: (a) yield and cost changes (which often differ from the national yield and cost changes for the policy scenario), (b) baseline production costs, and(c) acreages of each crop. This information is combined with price impacts estimated with AGSIM©, and regional supply elasticities from a prior version of AGSIM© (or from other sources) to estimate net farm income changes for the farms or areas considered.

The conceptual foundation for regional evaluation in this version of AGSIM© begins with a net farm income formula,

(13) $$\Pi_{ir} = \sum_{ic} A_{ic,ir} R_{ic,ir}$$

where:

Π_{ir}	=	net farm income in region ir,
Aic,ir	=	acreage harvested of the icth crop in that region, and
$R_{ic,ir}$	=	per-acre net return in that region.

Based on equation (13), it can be shown that the theoretically appropriate formula for computing net farm income *changes* for different regional situations is:

(14) $$\frac{\Delta \Pi_{ir}}{\Delta Z} \cong \sum_{ic} R_{ic,ir} \sum_{jc} \frac{\Delta A_{ic,ir}}{\Delta R_{jc,ir}} \frac{\Delta R_{jc,ir}}{\Delta Z} + \sum_{ic} A_{ic,ir} \sum_{jc} \frac{\Delta R_{ic,ir}}{\Delta Z}$$

where:

Δ represents a discrete change,
ΔZ represents the discrete policy change,
ic and jc are crop indices,

and other variables are as previously defined.

Equation (14) can be expressed in acreage elasticity (with respect to per-acre income) form,

$$\text{(15)} \qquad \frac{\Delta \Pi_{ir}}{\Delta Z} \cong \sum_{ic} R_{ic,ir} \sum_{jc} \varepsilon_{ic,ij,ir} \frac{R_{ic,ir}}{A_{jc,ir}} \frac{\Delta R_{jc,ir}}{\Delta Z} + \sum_{ic} A_{ic,ir} \sum_{jc} \frac{\Delta R_{ic,ir}}{\Delta Z}$$

where:

$\epsilon_{ic,ij,ir}$ = elasticity of acreage of the icth crop in the irth region with respect to per-acre income of the jcth crop in that region.

The term $\Delta R_{ic,ij}/\Delta Z$ in equations (14) and (15) can be further expanded to give

$$\text{(16)} \qquad \frac{\Delta R_{ic,ir}}{\Delta Z} \cong P_{ic} \Delta Y_{ic,ir} + Y_{ic,ir} \Delta P_{ic} - \Delta C_{ic,ir}$$

Formula (15) along with (16) can be empirically implemented to estimate the change in regional (or farm, representative farm, area or state level) farm income with the following information for each region: (a) crop budgets, (b) the change in yield and cost associated with the policy in question, price impacts estimated with AGSIM©, and externally specified (from an older version of AGSIM©, from subjective estimates, or from the literature) elasticities.

The first term on the right-hand side of (14) and (15) represents the change in net income resulting from increased or decreased acreage, while the last term on the right-hand side of (14) and (15) represents the change in net farm income on existing acreage of crops in the region. Since acreage response is generally inelastic, the last term on the right-hand side of (14) and (15) dominates the change in net farm income in a region; thus, elasticities generally will not have a major impact on regional net farm income changes estimated with the above approach.

AGSIM© Output

The major outputs from AGSIM© are changes in crop acreage, production, price, income, foreign consumer benefits, domestic consumer benefits, and farm program costs. The traditional method of economic welfare analysis (which is based on the concept of economic surplus) of policy changes is used to compute the sum of changes in producer surplus (net farm income) plus changes to all consumers (changes in consumers surplus) plus any changes in farm program payments (zero under 1996 FAIR). To avoid the possibility of inappropriately comparing a baseline with a policy scenario that was actually based on another baseline, a single run of AGSIM© produces both the baseline tables and the policy scenario tables, then computes economic surplus and price changes based on these two runs of the model.

Output from each run of the model includes two sets of tables for each crop; one set of tables for supply variables and another set of tables for supply and utilization variables. Each table includes historical statistics as well as simulations into the future. These tables are constructed for the baseline and the policy scenario.

5.3 Consumer Cleaning Cost Savings

Particulate matter air pollution has been shown to result in dirtier clothes, which in turn results in higher annual cleaning costs for consumers. One benefit of reduced particulate matter, then, is the consequent reduction in cleaning costs for consumers. Several studies have provided estimates of the cost to households of PM soiling. The study that is cited by ESEERCO (1994) as one of the most sophisticated and is relied upon by EPA in its 1988 Regulatory Impact Analysis for SO_2 is Manuel et al. (1982). Using a household production function approach and household expenditure data from the 1972-73 Bureau of Labor Statistics Consumer Expenditure Survey for over twenty cities in the United States, Manuel et al. estimated the annual cost of cleaning per $\mu g/m^3$ PM per household as $1.55 ($0.59 per person times 2.63 persons per household). This estimate is low compared with others (e.g., estimates provided by Cummings et al. (1985) and Watson and Jaksch (1982) are about eight times and five times greater, respectively). The ESEERCO report notes, however, that the Manuel estimate is probably downward biased because it does not include the time cost of do-it-yourselfers. Estimating that these costs may comprise at least half the cost of PM-related cleaning costs, they double the Manuel estimate to obtain a point estimate of $3.10 (reported by ESEERCO in 1992 dollars as $2.70).

The Manuel et al. (1982) study measured particulate matter as TSP rather than PM_{10} or $PM_{2.5}$. If a one $\mu g/m^3$ increase in TSP causes $1.55 worth of cleaning expenses per household, the same unit dollar value can be used for PM_{10} (or $PM_{2.5}$) only if particle size doesn't matter -- i.e., only if particles of all sizes are equally soiling. Suppose, for example, that PM_{10} is 75% of TSP and that all particles are equally soiling. Then 75% of the damage caused by a one $\mu g/m^3$ increase in TSP is due to PM_{10}. This is (0.75)($1.55) = $1.16. However, this corresponds to a 0.75 $\mu g/m^3$ increase in PM_{10}. A one $\mu g/m^3$ increase in PM_{10} would therefore yield a dollar soiling damage of $1.16/0.75 = $1.55.

Suppose, however, that only PM_{10} matters. Then the $1.55 underestimates the impact of a one $\mu g/m^3$ increase in PM_{10}, because it corresponds to a less than one $\mu g/m^3$ increase in PM_{10} (e.g., a 0.75 $\mu g/m^3$ increase in PM_{10}). In this case, the correct unit value per unit of PM_{10} would be ($1.55)/0.75 = $2.07. If only PM_{10} matters, then either (1) the dollar value can be adjusted by dividing it by the percentage of TSP that is PM_{10} and PM_{10} can be used in the soiling damage function, or (2) the dollar value can be left unadjusted and TSP, rather than PM_{10}, can be used in the soiling damage function.

Finally, it is possible that, while both PM_{10} and $PM_{2.5}$ are components of TSP that cause consumer cleaning costs, the remaining portion of TSP has a greater soiling capability than either the PM_{10} or $PM_{2.5}$ component. In this case, using either PM_{10} or $PM_{2.5}$ air quality data with a household soiling function based on TSP would yield overestimates of the PM_{10}- or $PM_{2.5}$-related consumer cleaning costs avoided by reductions in concentration of these pollutants.

There is, however, insufficient information on the relative soiling capabilities of the different components of TSP. This analysis assumes that all components of TSP have an equivalent soiling capacity.

6 Results

This chapter provides estimates of the magnitude and value of changes in selected health and welfare endpoints associated with Nonroad Diesel-related changes in ambient ozone and PM concentrations in 2020 and 2030. 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).

To place estimated incidence changes into context with predicted baseline incidence, Exhibit 6-1 displays the reductions in PM- and ozone-related adverse health effects as a percent of total baseline incidence. Due to the nature of the endpoints, baseline incidence can be calculated only for certain ozone- and PM-related health effects; there is no baseline incidence associated with welfare effects or endpoints that are direct economic valuations. In addition to the percent change between post-control incidence reductions and the predicted incidence baseline, we present the mean incidence change for each health effect. Note that the percent of total baseline incidences are calculated by dividing the number of health effects avoided due to the Nonroad Diesel rule by the total number of baseline incidences.

Exhibit 6-2 presents the base and alternative incidence estimates associated with the Nonroad Diesel rule in 2020 and 2030. A 2.5th percentile, mean, and 97.5th percentile estimate for incidence is presented for each endpoint. It should be noted that premature mortality associated with ozone is not separately included in this analysis. It is assumed that the C-R function for premature mortality captures both PM mortality benefits and any mortality benefits associated with other air pollutants.

Exhibits 6-3 and 6-4 present, respectively, the 2020 and 2030 base and alternative valuation estimates associated with the incidence change estimates presented in Exhibit 6-2. Exhibits 6-3 and 6-4 also present endpoints that were valued directly (ozone-related worker productivity) and the value of changes in visibility and agricultural benefits. Where possible, a 2.5th percentile, mean, and 97.5th percentile estimate for incidence is presented for each endpoint, representing uncertainty associated with both the estimate of incidence and the valuation. The valuation results also reflect the use of two different discount rates; a 3% rate which is recommended by EPA's Guidelines for Preparing Economic Analyses (2000e), and 7% which is recommended by OMB Circular A-94 (1992). Also note that the valuation for long-term mortality assumes the 5 year distributed lag structure described earlier.

Exhibit 6-5 presents the incidence of health effects occurring in three age groups: children (0-17), adults (18-64), and elderly adults (65 and older). Certain endpoints occur only in a subset of age groups, so not all endpoints are reported for all age groups. The exhibit presents two sets of age group estimates. The first is based on the specific age ranges examined in the epidemiological studies, for example, the Dockery et al. (1996) acute bronchitis study focused on a sample population aged 8 to 12. These are the estimates that we used to derive total incidences in Exhibit 6-2. In many cases, however, the study populations were defined as a matter of convenience or due to data availability, rather than due to any biological factor that would restrict the effect to the specific age group. In order to gain a more complete understanding of the potential magnitude of the health impact in the entire population, we calculate a separate estimate including the health impact on all population within an age group. Exhibit 6-5 also presents these results.

Exhibit 6-6 presents the mean incidence results associated with respiratory symptoms and attacks occurring in the asthmatic population, based on the studies defined in Chapter 4, Table 4-23. As with the age group specific estimates, Exhibit 6-6 provides two sets of calculations, one based on applying the C-R function only to the specific population subgroup included in a study's sample population, and another

based on applying the C-R function to all populations within a broader population. Note that the asthma symptom estimates provided in Exhibit 6-6 are not additive to total benefits. They are provided to show the specific impacts on an especially susceptible subpopulation. Also note that the estimates are not additive even within the exhibit. We have grouped the estimates based on the type of symptoms measured, but there is the potential for considerable overlap. However, these estimates provide an illustration of the consistency of the effects across studies and populations of asthmatics.

Exhibit 6-1. Reductions in PM- and Ozone-related Adverse Health Effects as Percent of Baseline Incidence

Endpoint	Avoided Incidence (cases/year)			
	2020	*% Baseline*	*2030*	*% Baseline*
PM-related Endpoints				
Premature mortality				
Base estimate: Long-term exposure (adults, 30 and over)	6,211	0.18%	10,989	0.26%
Alternative estimate: Short-term exposure (all ages)	3,706	0.11%	6,571	0.15%
Chronic bronchitis (adults, 27 and over)	4,282	0.47%	6,548	0.67%
Non-fatal myocardial infarctions (adults, 18 and older)	10,567	0.52%	17,768	0.91%
Hospital admissions - Chronic Lung (adults, 20-64)	257	0.02%	340	0.02%
Hospital admissions - Asthma (0-64)	426	0.03%	613	0.05%
Hospital admissions - Pneumonia (adults, 65+)	2,136	1.10%	4,050	1.42%
Hospital admissions - Chronic Lung (adults, 65+)	263	0.19%	499	0.25%
Total hospital admissions – Respiratory	3,082	0.05%	5,502	0.09%
Hospital admissions - Cardiovascular (adults, 20-64)	921	0.01%	1,236	0.01%
Hospital admissions - Cardiovascular (adults, 65+)	2,086	0.21%	3,954	0.27%
Total hospital admissions – Cardiovascular	3,007	0.02%	5,191	0.04%
Emergency Room Visits for Asthma (18 and younger)	4,324	0.55%	6,510	0.76%
Acute bronchitis (children, 8-12)	10,172	1.34%	15,604	1.90%
Lower respiratory symptoms (children, 7-14)	116,055	0.75%	178,206	1.06%
Upper respiratory symptoms (asthmatic children, 9-11)	91,900	0.15%	123,976	0.18%
Work loss days (adults, 18-64)	777,566	0.18%	1,112,905	0.26%
Minor restricted activity days (adults, 18-64)	4,577,570	0.30%	6,507,255	0.42%
Ozone-related Endpoints				
Hospital Admissions – Respiratory (adults, 65+)	372	0.08%	1,131	0.17%
Hospital Admissions - Respiratory (children, under 2)	432	0.08%	863	0.16%
Emergency Room Visits for Asthma (all ages)	93	0.005%	199	0.01%
Minor restricted activity days (adults, 18-64)	-2,416	-0.0002%	95,858	0.01%
School absence days (children, 6-11)	64,595	0.10%	137,654	0.20%

Exhibit 6-2. Reductions in Incidence of PM- and Ozone-related Adverse Health Effects Associated with the Preliminary Nonroad Landbased Diesel Engine Standards

Endpoint [a]	Avoided Incidence [b] (cases/year)					
	2020			*2030*		
	2.5%	*mean*	*97.5%*	*2.5%*	*mean*	*97.5%*
PM-related Endpoints						
Premature mortality						
Base Estimate: Long-term exposure (30+)	3,043	6,211	9,376	5,386	10,989	16,585
Alternative Estimate, Short-term exposure:						
Non-COPD (65+)	–	2,551	–	–	4798	–
Non-COPD Related (0-64)	–	724	–	–	978	–
COPD-Related (65+)	–	369	–	–	712	–
COPD-Related (0-64)	–	62	–	–	83	–
Chronic bronchitis (27+)	119	4,282	8,416	182	6,548	12,848
Non-fatal myocardial infarctions (18+)	2,610	10,567	18,431	4,400	17,768	30,913
Hospital admissions - Chronic Lung (20-64)	28	257	485	37	340	642
Hospital admissions - Asthma (0-64)	78	426	775	112	613	1,118
Hospital admissions - Pneumonia (65+)	343	2,136	3,934	650	4,050	7,463
Hospital admissions - Chronic Lung (65+)	-569	263	1,083	-1,080	499	2,057
Total hospital admissions – Respiratory	n/a	3,082	n/a	n/a	5,502	n/a
Hospital admissions - Cardiovascular (20-64)	-82	921	1,924	-110	1,236	2,584
Hospital admissions - Cardiovascular (65+)	-916	2,086	5,074	-1,736	3,954	9,619
Total hospital admissions – Cardiovascular	n/a	3,007	n/a	n/a	5,191	n/a
Emergency Room Visits for Asthma (0-18)	2,196	4,324	6,463	3,303	6,510	9,740
Acute bronchitis (8-12)	-2,373	10,172	22,508	-3,653	15,604	34,403
Lower respiratory symptoms (7-14)	43,173	116,055	188,361	66,413	178,206	288,722
Upper respiratory symptoms (asthmatics 9-11)	16,855	91,900	166,896	25,990	123,976	257,267
Work loss days (18-64)	658,458	777,566	896,616	942,534	1,112,905	1,283,152
Minor restricted activity days (18-64)	3,731,795	4,577,570	5,422,544	5,306,090	6,507,255	7,706,762
Ozone-related Endpoints						
Hospital Admissions – Respiratory (65+)	76	372	749	230	1,131	2,241
Hospital Admissions - Respiratory (under 2)	182	432	689	363	863	1,382
Emergency Room Visits for Asthma (all ages)	26	93	225	56	199	347
Minor restricted activity days (18-64)	-5,573	-2,416	-547	40,668	95,858	148,194
School absence days (6-11)	-7,666	64,595	138,066	-16,622	137,654	271,781

[a] Ozone-related mortality is not included in the estimate of premature mortality. Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma, and ozone-related respiratory admissions includes all respiratory causes and subcategories for COPD and pneumonia. Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

Exhibit 6-3. Results of Human Health and Welfare Benefits Valuation for the Modeled Preliminary Nonroad Landbased Diesel Engine Standards: 2020

Endpoint [a]	2020 Monetary Benefits [b] (millions 2000$)		
	2.5%	*mean*	*97.5%*
PM-related Endpoints			
Premature mortality			
Base estimate: Long-term exposure, (adults, 30 and over)			
3% discount rate	$2,439	$37,202	$81,622
7% discount rate	$2,291	$34,943	$76,666
Alternative estimate: Short-term exposure, 3% discount rate (all ages)			
Non-COPD Related (65 and over)	–	$5,067	–
Non-COPD Related (0-64)	–	$571	–
COPD Related (65 and over)	–	$78	–
COPD Related (0-64)	–	$5	–
Sum	–	$5,722	–
Alternative estimate: Short-term exposure, 7% discount rate (all ages)			
Non-COPD Related (65 and over)	–	$5510	–
Non-COPD Related (0-64)	–	$848	–
COPD Related (65 and over)	–	$92	–
COPD Related (0-64)	–	$8	–
Sum	–	$6,459	–
Chronic bronchitis (adults, 26 and over)			
Base estimate: Willingness-to-pay	$27	$1,458	$6,705
Alternative estimate: Cost-of-illness			
3% discount rate	$12	$416	$817
7% discount rate	$7	$267	$525
Non-fatal myocardial infarctions			
3% discount rate	$222	$899	$1,569
7% discount rate	$215	$872	$1,521
Hospital admissions - Chronic Lung Disease (adults, 20-64)	$0.3	$3	$6
Hospital admissions - Asthma (0-64)	$1	$3	$6
Hospital admissions - Pneumonia (adults, 65 and over)	$6	$38	$70
Hospital admissions - Chronic Lung Disease (adults, 65 and over)	-$8	$4	$15
Total hospital admissions – Respiratory	–	$48	–
Hospital admissions - Cardiovascular (adults, 20-64)	-$2	$21	$44
Hospital admissions - Cardiovascular (adults, 65 and over	-$19	$44	$107
Total hospital admissions – Cardiovascular	–	$65	–
Emergency Room Visits for Asthma	$1	$1	$2
Acute bronchitis (children, 8-12)	-$1	$4	$10
Lower respiratory symptoms (children, 7-14)	$1	$2	$4
Upper respiratory symptoms (asthmatic children, 9-11)	$0	$2	$6
Work loss days (adults, 18-65)	$90	$107	$123
Minor restricted activity days (adults, age 18-65)	$122	$233	$352
Ozone-related Endpoints			
Hospital Admissions – Respiratory Causes (adults, 65 and older)	$1	$6	$13
Hospital Admissions - Respiratory Causes (children, under 2 years)	$1	$3	$5

Exhibit 6-3. Results of Human Health and Welfare Benefits Valuation for the Modeled Preliminary Nonroad Landbased Diesel Engine Standards: 2020 (Continued)

Endpoint [a]	2020 Monetary Benefits [b] (millions 2000$)		
	2.5%	*mean*	*97.5%*
Emergency Room Visits for Asthma (all ages)	$0.00	$0.03	$0.06
Minor restricted activity days (adults, age 18-64)	-$0.30	-$0.12	-$0.03
School absence days (children, age 6-11)	-$1	$5	$10
Worker productivity (outdoor workers, age 18-65)	–	$14	–
Welfare Endpoints			
Recreational visibility (86 Class I Areas)	–	$826	–
Agricultural crop damage (6 crops)[c]	$41	$88	$134
Monetized Total			
Base estimate			
3% discount rate	–	$40,963	–
7% discount rate	–	$38,677	–
Alternative estimate			
3% discount rate	–	$8,441	–
7% discount rate	–	$9,002	–

[a] Monetary benefits are rounded to two significant digits.

[b] Ozone-related mortality is not included in the estimate of premature mortality. Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma, and ozone-related respiratory admissions includes all respiratory causes and subcategories for COPD and pneumonia. Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

[c] The credible range for agricultural crop damage does not represent the 2.5th and 97.5th percentiles. Instead, the range represents the minimum and maximum of the range of benefits associated with avoided agricultural crop damage.

Exhibit 6-4. Results of Human Health and Welfare Benefits Valuation for the Modeled Preliminary Nonroad Landbased Diesel Engine Standards: 2030

Endpoint [a]	2030 Monetary Benefits [b] (millions 2000$)		
	2.5%	*mean*	*97.5%*
PM-related Endpoints			
Premature mortality			
Base estimate: Long-term exposure, (adults, 30 and over)			
3% discount rate	$4,314	$65,817	$144,398
7% discount rate	$4,052	$61,820	$135,630
Alternative estimate: Short-term exposure, 3% discount rate (all ages)			
Non-COPD Related (65 and over)	–	$9,531	–
Non-COPD Related (0-64)	–	$771	–
COPD Related (65 and over)	–	$151	–
COPD Related (0-64)	–	$7	–
Sum	–	$10,460	–
Alternative estimate: Short-term exposure, 7% discount rate (all ages)			
Non-COPD Related (65 and over)	–	$10,364	–
Non-COPD Related (0-64)	–	$1,146	–
COPD Related (65 and over)	–	$178	–
COPD Related (0-64)	–	$11	–
Sum	–	$11,699	–
Chronic bronchitis (adults, 26 and over)			
Base estimate: Willingness-to-pay	$42	$2,229	$10,245
Alternative estimate: Cost-of-illness			
3% discount rate	$17	$601	$1,180
7% discount rate	$11	$385	$755
Non-fatal myocardial infarctions			
3% discount rate	$355	$1,435	$2,496
7% discount rate	$346	$1,396	$2,429
Hospital admissions - Chronic Lung Disease (adults, 20-64)	$0	$4	$8
Hospital admissions - Asthma (0-64)	$1	$5	$9
Hospital admissions - Pneumonia (adults, 65 and over)	$12	$72	$133
Hospital admissions - Chronic Lung Disease (adults, 65 and over)	-$15	$7	$28
Total hospital admissions – Respiratory		$88	
Hospital admissions - Cardiovascular (adults, 20-64)	-$2	$28	$59
Hospital admissions - Cardiovascular (adults, 65 and over	-$37	$84	$204
Total hospital admissions – Cardiovascular	–	$112	–
Emergency Room Visits for Asthma	$1	$2	$3
Acute bronchitis (children, 8-12)	-$1	$6	$16
Lower respiratory symptoms (children, 7-14)	$1	$3	$6
Upper respiratory symptoms (asthmatic children, 9-11)	$1	$3	$9
Work loss days (adults, 18-65)	$129	$153	$176
Minor restricted activity days (adults, age 18-65)	$173	$332	$500
Ozone-related Endpoints			
Hospital Admissions – Respiratory Causes (adults, 65 and older)	$4	$19	$38
Hospital Admissions - Respiratory Causes (children, under 2 years)	$3	$7	$11

Exhibit 6-4. Results of Human Health and Welfare Benefits Valuation for the Modeled Preliminary Nonroad Landbased Diesel Engine Standards: 2030 (Continued)

Endpoint [a]	2030 Monetary Benefits [b] (millions 2000$)		
	2.5%	*mean*	*97.5%*
Emergency Room Visits for Asthma (all ages)	$0.02	$0.06	$0.1
Minor restricted activity days (adults, age 18-64)	$2	$5	$9
School absence days (children, age 6-11)	-$1	$10	$20
Worker productivity (outdoor workers, age 18-64)	–	$23	–
Welfare Endpoints			
Recreational visibility (86 Class I Areas)	–	$1,250	–
Agricultural crop damage (6 crops)[c]	$68	$137	$205
Monetized Total			
Base estimate			
3% discount rate	–	$71,631	–
7% discount rate	–	$67,595	–
Alternative estimate			
3% discount rate	–	$14,646	–
7% discount rate	–	$15,630	–

[a] Monetary benefits are rounded to two significant digits.

[b] Ozone-related mortality is not included in the estimate of premature mortality. Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma, and ozone-related respiratory admissions includes all respiratory causes and subcategories for COPD and pneumonia. Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

[c] The credible range for agricultural crop damage does not represent the 2.5th and 97.5th percentiles. Instead, the range represents the minimum and maximum of the range of benefits associated with avoided agricultural crop damage.

Exhibit 6-5. Reductions in Incidence of Health Endpoints by Age Group

Pollutant	Endpoint/Age Group [a]	Age of Study Population	Avoided Incidence (cases/year)			
			Study Population Only		Total Age Group Population	
			2020	*2030*	*2020*	*2030*
Children, 0-17						
PM	Premature mortality - Alternative estimate: Short-term exposure	0-17	20	30	20	30
	Hospital Admissions - Asthma	0-17	192	290	192	290
	Emergency Room Visits for Asthma	0-17	4,324	6,510	4,324	6,510
	Acute bronchitis	8-12	10,172	15,604	30,898	46,739
	Lower respiratory symptoms	7-14	116,055	178,206	220,500	333,589
	Upper respiratory symptoms in asthmatics	9-11	91,900	123,976	433,317	657,319
O_3	Hospital Admissions - Respiratory Causes	<2	432	863	432	863
	School absence days (two studies pooled)	9-10, 6-11	64,595	137,654	161,671	321,654
Adults, 18-64						
PM	Premature mortality -					
	Base estimate: Long-term exposure	30-64	1,360	1,841	1,453	1,971
	Alternative estimate: Short-term exposure	18-64	766	1,031	766	1,031
	Chronic bronchitis	27-64	3,256	4,618	4,943	5,567
	Non-fatal myocardial infarctions	18-64	3,942	5,269	3,942	5,269
	Hospital admissions - Chronic Lung Disease	20-64	257	340	257	340
	Hospital admissions - Asthma	18-64	234	323	234	323
	Hospital admissions - Cardiovascular Causes	20-64	921	1,236	921	1,236
	Work loss days	18-64	777,566	1,112,905	777,566	1,112,905
	Minor restricted activity days	18-64	4,577,570	6,507,255	4,577,570	6,507,255
O_3	Minor restricted activity days	18-64	-2,416	95,858	-2,416	95,858
Adults, 65 and older						
PM	Premature mortality -					
	Base estimate: Long-term exposure	65+	4,852	9,148	4,852	9,148
	Alternative estimate: Short-term exposure	65+	2,920	5,510	2,920	5,510
	Chronic Bronchitis	65+	1,026	1,930	1,026	1,930
	Non-fatal Myocardial Infarctions	65+	6,625	12,499	6,625	12,499
	Hospital Admissions - Pneumonia	65+	2,136	4,050	2,136	4,050
	Hospital Admissions - Chronic Lung Disease	65+	263	499	263	499
	Hospital Admissions - Cardiovascular Causes	65+	2,086	3,954	2,086	3,954
O_3	Hospital Admissions – Respiratory Causes	65+	372	1,131	372	1,131

[a] Ozone-related mortality is not included in the estimate of premature mortality.

Exhibit 6-6. Reductions in Incidence of Respiratory Symptoms in the Asthmatic Population

PM-Related Endpoint	Description of Study Population	Avoided Incidence (cases/year)			
		Study Population Only		Total Age Group Population	
		2020	*2030*	*2020*	*2030*
Asthma Attack Indicators [a]					
Shortness of Breath	African American asthmatics, 8-13	9,970	14,836	30,261	45,250
Cough	African American asthmatics, 8-13	20,779	30,922	63,069	94,312
Wheeze	African American asthmatics, 8-13	16,300	24,258	49,473	73,986
Asthma Exacerbation – one or more symptoms	Asthmatics, 5-13	401,458	614,782	626,871	951,249
Cough	Asthmatics, 6-13	183,586	281,306	323,021	489,599
Other symptoms/illness endpoints					
Upper Respiratory Symptoms	Asthmatics 9-11	91,900	141,683	433,317	657,319
Moderate or Worse Asthma	Asthmatics, all ages	85,532	121,408	85,532	121,408
Acute Bronchitis	Asthmatics, 9-15	3,039	4,707	7,201	10,914
Chronic Phlegm	Asthmatics, 9-15	7,453	11,510	17,656	26,686
Asthma Attacks	Asthmatics, all ages	129,502	194,158	129,502	194,158

[a] Note that these symptoms of asthma are not necessarily independent. Combinations of these symptoms may occur in the same individuals, so that the sum of the avoided incidences is not necessarily equal to the sum of the affected populations. Also, some asthma studies cover the same or similar endpoints in overlapping populations. For example, the Vedal et al (1998) and Ostro et al (2000) studies both examined cough. However, the Ostro et al (2000) estimate examined a more restricted population than Vedal et al (1998), so estimates should be combined with caution.

7 References

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Appendix A: Individual Study Results for Base and Alternative Estimates, Pooling Weights, and Sensitivity Results

This Appendix provides additional details underlying the results presented in the Base and Alternative Estimates. This includes the results from the individual studies included in the pooled estimates, as well as the weights for these studies. In addition, in those cases where we calculated results for individual age groups and then summed these age groups to provide our estimate, we have provided the individual age-specific results. In addition, this appendix provides the results of the sensitivity analyses.

Exhibit A-1. Supplemental Benefit Estimates for the 2030 Control Scenario

Endpoint	Year	<18	18-24	25-34/30-34	35-44	45-54	55-64	65-74	75-84	85+	Total
Krewski et al. (2000) - Mortality, Long-Term, All Cause, 30+	2020	n/a	n/a	51	164	323	822	1,425	1,606	1,821	6,211
	2030	n/a	n/a	73	258	447	1,063	2,527	3,470	3,151	10,989
Schwartz et al. (1996) - Mortality, Short-Term, Non-COPD Related, All Ages	2020	19	5	31	70	166	433	723	824	1,004	3,274
	2030	29	7	42	110	229	561	1,282	1,778	1,738	5,776
Schwartz et al. (1996) - Mortality, Short-Term, COPD Related, All Ages	2020	1	0	1	2	9	49	131	143	95	431
	2030	1	1	2	4	12	63	234	312	166	795

Exhibit A-2. Sensitivity Calculation: Alternative Threshold and Alternative Lag Structures for Long-term Mortality

Analysis	Description	Incidence (Cases/Year)		Benefits (millions 2000$) [a]	
		2020	*2030*	*2020*	*2030*
Alternative Threshold					
Mortality, Long-Term, 30+' Krewski et al. (2000) Annual Mean PM2.5	No Threshold	6,211	10,989	$37,202	$65,817
	5 ug/m3	6,194	10,962	$37,097	$65,657
	10 ug/m3	5,035	9,402	$30,154	$56,315
	15 ug/m3	1,345	3,197	$8,055	$19,147
	20 ug/m3	498	1,032	$2,985	$6,183
	25 ug/m3	150	428	$900	$2,566
Alternative Lag					
Mortality, Long-Term, 30+ Krewski et al. (2000) Annual Mean PM2.5	No lag	6,211	10,989	$39,132	$69,231
	Base (5 Year) Distributed Lag: 25%, 25%, 17%, 17%, 16%	6,211	10,989	$37,202	$65,817
	8 Year Lag: Incidence Occurs 8th Year	6,211	10,989	$31,818	$56,291
	15 Year Lag: Incidence Occurs 15th Year	6,211	10,989	$25,871	$45,770

[a] When calculating benefits for the alternative threshold, we assume a 5-year distributed lag adjustment calculated, and we use a three percent discount rate. For the alternative lag benefits, we also use a three percent discount rate.

Exhibit A-3. Sensitivity Calculation: Alternative C-R Functions for Mortality

Reference	Age	PM Metric	Incidence (Cases/Year)		Benefits (millions 2000$)	
			2020	*2030*	*2020*	*2030*
Pope et al. (2002)	30+	Annual Mean $PM_{2.5}$	5,397	9,549	$32,322	$57,192
Krewski et al. (2000) - Pope et al. (1995) Reanalysis	30+	Annual Median PM2.5	6,518	11,560	$39,041	$69,235
Krewski et al. (2000) - Pope et al. (1995) Reanalysis - Random Effects, Independent Cities	30+	Annual Median PM2.5	12,653	22,428	$75,784	$134,327
Krewski et al. (2000) - Pope et al. (1995) Reanalysis - Random Effects, Regional Adjustment	30+	Annual Median PM2.5	7,382	13,091	$44,216	$78,407
Pope et al. (1995)	30+	Annual Median PM2.5	7,809	13,846	$46,769	$82,931
Krewski et al. (2000) - Dockery et al. (1993) Reanalysis	25+	Annual Mean PM2.5	17,940	31,647	$107,447	$189,542
Dockery et al. (1993)	25+	Annual Mean PM2.5	16,799	29,637	$100,614	$177,505
Pope et al. (2002)	30+	Annual Mean PM2.5	3,978	7,163	$23,824	$42,899
Pope et al. (2002)	30+	Annual Mean PM2.5	742	1,279	$4,444	$7,658
Woodruff et al. (1997)	Infant	Annual Mean PM10	35	52	$211	$312

Exhibit A-4. Sensitivity Calculations: Miscellaneous Ozone- and PM-related

			Incidence (Cases/Year)		Benefits (millions 2000$)	
Sensitivity	**Reference**	**Pollutant**	***2020***	***2030***	***2020***	***2030***
Chronic Bronchitis Reversals	Based on Abbey et al. (1995c)	Annual Mean $PM_{2.5}$	3,725	5,697	$560	$856
Alternative Hospital Admissions						
COPD	Samet et al. (2000)	Daily Mean PM_{10}	791	2,217	$11	$30
Pneumonia	Samet et al. (2000)	Daily Mean PM_{10}	976	1,851	$17	$33
All Cardiovascular	Samet et al. (2000)	Daily Mean PM_{10}	2,879	5,398	$61	$114
PM-Based Alternative Minor Illness						
Any-of-19 Respiratory Symptoms	Krupnick et al. (1990)	Daily Mean PM_{10}	13,360,641	19,015,146	$317	$451
Alternative Welfare Benefits						
Household Soiling Damage	ESEERCO (1994)	Annual Mean PM_{10}	–	–	$170	$258
Ozone-Based Alternative Analyses						
Chronic Asthma	McDonnell et al. (1999)	Annual Mean 8-hr ozone	1,167	2,412	$36	$74
Any-of-19 Respiratory Symptoms	Krupnick et al. (1990)	Daily 1-hr Max ozone	1,488,798	2,739,641	$35	$65
Ozone Mortality	4 U.S. Studies	Daily Mean and 1-hr Max ozone	94	281	$561	$1,685

Exhibit A-5. Pooling Weights Used in Base Analysis and Sensitivity Calculations

		2020 Pooling			2030 Pooling		
Endpoint	**Study**	***Mean***	***Std. Dev.***	***Weights***	***Mean***	***Std. Dev.***	***Weights***
PM-Related COPD 65+							
	Lippmann et al. (2000)	361	762	0.15	687	1,447	0.15
	Moolgavkar (2000)	255	322	0.85	485	612	0.85
PM-Related All Cardiovascular 65+							
	Lippmann et al. (2000)	3,155	1,157	0.50	5,980	2,193	0.50
	Moolgavkar (2000)	1,019	1,162	0.50	1,932	2,204	0.50
Ozone-Related All Respiratory 65+ – Incidence							
	Schwartz (1995), New Haven	339	171	0.11	1,051	530	0.10
	Schwartz (1995), Tacoma	924	331	0.03	2,858	1,022	0.03
	Schwartz (1994), Detroit	534	113	0.24	1,604	339	0.25
	Moolgavkar et al. (1997), Minneapolis	336	94	0.36	1,020	284	0.35
	Schwartz (1994), Minneapolis	235	108	0.27	713	327	0.27
Ozone-Related All Respiratory 65+ – Valuation							
	Schwartz (1995), New Haven	6.23	3.14	0.08	19.28	9.73	0.08
	Schwartz (1995), Tacoma	16.97	6.08	0.02	52.45	18.75	0.02
	Schwartz (1994), Detroit	8.57	1.78	0.27	25.79	5.33	0.27
	Moolgavkar et al. (1997), Minneapolis	5.49	1.15	0.40	16.68	4.40	0.40
	Schwartz (1994), Minneapolis	4.18	1.92	0.23	12.69	5.82	0.23
Ozone-Related Asthma ER Visits All Ages							
	Weisel et al. (1995)	115	18	0.48	248	39	0.49
	Cody et al. (1992)	53	18	0.49	114	39	0.49
	Stieb et al. (1996)	362	175	0.03	817	396	0.02
Ozone-Related School Loss Days							
	Gilliland et al. (2001)	46,614	27,809	0.58	101,807	60,909	0.53
	Chen et al. (2000)	89,552	32,649	0.42	176,282	64,270	0.47
Ozone-Related Mortality							
	Moolgavkar et al. (1995)	77	27	0.65	235	83	0.63
	Samet et al. (1997)	118	39	0.31	360	120	0.30
	Ito and Thurston (1996)	444	175	0.02	993	392	0.03
	Kinney et al. (1995)	0	149	0.02	0	333	0.04

Exhibit A-6. Age-Specific Incidence Estimates: PM-related Hospital Admissions

Endpoint	Year	Age Group									
		<18	*18-24*	*25-34*	*35-44*	*45-54*	*55-64*	*65-74*	*75-84*	*85+*	*Total*
Lippmann et al. (2000) - Pneumonia, 65+	2020	–	–	–	–	–	–	738	750	649	2,136
	2030	–	–	–	–	–	–	1,311	1,619	1,120	4,050
Lippmann et al. (2000) - COPD, 65+	2020	–	–	–	–	–	–	182	123	44	349
	2030	–	–	–	–	–	–	323	263	77	663
Moolgavkar (2000b) - COPD, 65+	2020	–	–	–	–	–	–	134	90	33	256
	2030	–	–	–	–	–	–	237	193	57	487
Moolgavkar (2000b) - COPD, 20-64	2020	–	1	6	16	49	184	–	–	–	257
	2030	–	2	8	26	67	237	–	–	–	340
Sheppard et al. (1999) - Asthma, <65	2020	192	17	52	40	57	67	–	–	–	426
	2030	290	25	71	63	78	86	–	–	–	613
Lippmann et al. (2000) - Ischemic, 65+	2020	–	–	–	–	–	–	303	170	53	526
	2030	–	–	–	–	–	–	536	368	91	995
Lippmann et al. (2000) - Dysrhythmia, 65+	2020	–	–	–	–	–	–	259	220	124	603
	2030	–	–	–	–	–	–	456	473	215	1,144
Lippmann et al. (2000) - Heart Failure, 65+	2020	–	–	–	–	–	–	682	740	596	2,019
	2030	–	–	–	–	–	–	1,207	1,591	1,027	3,825
Moolgavkar (2000a) - Cardio., 65+	2020	–	–	–	–	–	–	462	366	208	1,036
	2030	–	–	–	–	–	–	818	789	358	1,965
Moolgavkar (2000a) - Cardio., 20-64	2020	–	7	34	87	229	564	–	–	–	921
	2030	–	10	47	137	314	727	–	–	–	1,236
Peters et al. (2001) - Heart Attack (Non-Fatal), 18+	2020	–	3	45	330	1,077	2,487	3,076	2,269	1,280	10,567
	2030	–	5	62	521	1,479	3,202	5,435	4,875	2,189	17,768

Exhibit A-7. Age-Specific Incidence Estimates: Ozone-related Hospital Admissions for Persons 65+

Endpoint	Year	Age Group			
		65-74	*75-84*	*85+*	*Total*
Schwartz (1995) - All Resp., New Haven	2020	150	133	53	336
	2030	415	465	161	1,040
Schwartz (1995) - All Resp., Tacoma	2020	412	365	147	924
	2030	1,138	1,273	445	2,855
Schwartz (1994) - COPD, Detroit	2020	126	86	11	223
	2030	331	288	39	658
Moolgavkar et al. (1997) - COPD, Minneapolis	2020	62	48	7	117
	2030	172	161	23	356
Schwartz (1994) - Pneumonia, Detroit	2020	120	121	69	310
	2030	326	417	198	941
Moolgavkar et al. (1997) - Pneumonia, Minneapolis	2020	85	85	49	219
	2030	230	294	140	664
Schwartz (1994) - Pneumonia, 65+, Minneapolis	2020	91	92	52	235
	2030	247	317	150	715

Exhibit A-8. Age-Specific Incidence Estimates: PM-related Chronic Bronchitis and Work Loss Days

Endpoint	Year	Age Group				
		18-24	*25-44/27-44*	*45-64*	*65+*	*Total*
Abbey et al. (1995c) - Chronic Bronchitis, 27+	2020	–	1,631	1,625	1,026	4,282
	2030	–	2,438	2,180	1,930	6,548
Ostro (1987) - Work Loss Days, 18-64	2020	83,413	420,206	273,946	–	777,566
	2030	123,122	621,900	367,883	–	1,112,905

Exhibit A-9. Age-Specific Incidence Estimates: PM-related Asthma Attacks and Moderate-or-Worse Asthma

Endpoint	Year	Age Group									
		<18	*18-24*	*25-34*	*35-44*	*45-54*	*55-64*	*65-74*	*75-84*	*85+*	*Total*
Whittemore and Korn (1980) - Asthma Attacks, All Ages	2020	39,945	9,314	20,132	17,239	14,658	15,474	7,475	3,677	1,588	129,502
	2030	51,683	12,254	24,287	26,118	21,173	14,096	6,117	4,278	1,537	161,543
Ostro et al. (1991) - Moderate-or-Worse Asthma	2020	26,211	6,107	13,127	11,364	9,653	10,367	5,106	2,512	1,084	85,532
	2030	37,657	8,520	17,266	17,038	12,790	12,594	8,584	5,175	1,785	121,408

Exhibit A-10. Age-Specific Incidence Estimates: Ozone-related Mortality

Endpoint	Year	Age Group									
		<18	*18-24*	*25-34*	*35-44*	*45-54*	*55-64*	*65-74*	*75-84*	*85+*	*Total*
Ito and Thurston, (1996)	2020	2	0	1	4	16	57	114	126	124	444
	2030	3	1	2	9	28	89	249	343	268	993
Kinney et al., (1995)	2020	0	0	0	0	0	0	0	0	0	0
	2030	0	0	0	0	0	0	0	0	0	0
Moolgavkar et al., (1995)	2020	0	0	-1	-1	1	10	25	25	17	77
	2030	0	0	-1	0	4	21	68	90	53	235
Samet et al., (1997)	2020	0	0	-2	-1	1	15	39	39	26	118
	2030	1	0	-1	-1	7	32	104	138	82	360

Appendix B: Population Forecasts

This appendix summarizes the steps used to estimate 2020 and 2030 population. In addition, we include a table with age-specific population estimates by state for the years 2000, 2020, and 2030.

B .1 Population Grid-Cells

CAPMS calculates health impacts at the level of U.S. counties as well as for a variety of grid structures used in air quality modeling (e.g.,, REMSAD, and CAMx). In this description, we use the term "population grid-cells" to refer to counties or the cells within an air quality modeling grid. The foundation for calculating the population level in the population grid-cells is the 2000 Census block data.[55] A separate application developed by Abt Associates, called "PopGrid," combines the Census block data with any user-specified set of population grid-cells, so long as they are defined by a GIS shape file.

If the center of a Census block falls within a population grid-cell, PopGrid assigns the block population to this particular population grid-cell. Note that the grid-cells in air quality model, such as REMSAD and CAMx, may cross multiple county boundaries. To account for this, PopGrid keeps track of the total number of people by county within a particular populatin grid-cell. Keeping track of the total number of people in a county is useful in the estimation of adverse health effects, where the calculation of premature mortality depends on county-level mortality rates. It is also useful in the presentation of health benefits, when users may want estimates at the state- and county-level, as opposed to estimates by, say, the area covered by an air quality model.

Within any given population grid-cell, CAPMS has 256 demographic variables, including 180 unique racial-gender-age groups: 19 age groups by gender by 5 racial groups (19*2*5=180). In addition there is an Hispanic ethnicity variable, which includes a number of different racial groups, as well as a number of variables that aggregate the population by race and gender. Exhibit B-1 presents the 256 population variables available in CAPMS. As discussed below, these variables are available for use in developing age estimates in whatever grouping desired by the user.

Exhibit B-1. Demographic Groups and Variables Available in CAPMS

Racial/Ethnic Group	Gender	Age	# Variables
White, African American, Asian, American Indian, Other, Hispanic	Female, Male	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+	228
All	–	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+	19
All	Female, Male	–	2
White, African American, Asian, American Indian, Other, Hispanic	–	–	6
All	–	–	1

[55] Geolytics (2001; 2002a) provided the 1990 and 2000 census data.

B .2 Census Data 2000

In addition to forecasting post-2000 population levels based on the 2000 Census, CAPMS also allows the user to estimate the impacts for 1991-1999 by interpolating between the results of the 1990 and 2000 Census. As a result, we have developed a consistent set of demographic variables, based on the 1990 Census, which provides somewhat less detail than the 2000 Census.

The 2000 Census allows respondents to choose more than one racial category, unlike the 1990 Census, which allowed only one choice. As a result there are seven racial categories in the 2000 Census versus five in the 1990 Census (Exhibit B-2). To make the 2000 Census data consistent with the 1990 Census, we reduced the seven racial groups to the five used in the 1990 Census.

The initial data set at the block level includes 368 demographic groups: seven racial groups and Hispanic ethnicity, by 23 pre-defined age groups by gender (Exhibit B-2). Because the 2000 Census includes somewhat different age groupings than that for the final set generated for the 1990 Census. Age variables 15-17 and 18-19 are combined, 20, 21, and 22-24 are combined, 60-61 and 62-24 are combined, and 65-66 and 67-69 are combined at the block level. One variable, under 5 years, must be split into two variables (Under 1 and 1-4 years). Assuming that the population is uniformly distributed within age groups, we apply a factor of 1/5 to create the 0-1 age group and 4/5 to create the 1-4 age group.

Exhibit B-2. Race, Ethnicity and Age Variables in 2000 Census Block Data

	Race / Ethnicity	Gender	Age
Initial Variables	White Alone, Black Alone, Native American Alone, Asian Alone, Pacific Islander / Hawaiian Alone, Other Alone, Two or More Alone, Hispanic (Non-Exclusive)	Male, Female	0-5, 5-10, 10-14, 15-17, 18-19, 20, 21, 22-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-61, 62-64, 65-66, 67-69, 70-74, 75-79, 80-84 85+
Final Variables (identical to 1990 variables)	White, African American, Asian & Pacific Islander, American Indian, Other, Hispanic	Female, Male	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+

Source: Geolytics (2002a). Note: Some population values were errors in the original Census data (e.g., values of a billion or more). Following personal communication with Geolytics, these were set to zero.

B .1.1 Matching Racial Categories in the 1990 and 2000 Censuses

Unlike the 1990 Census, respondents in the 2000 Census respondents could check more than one box for race, so the reported results included a grouping of individuals that had checked two or more racial categories. In addition, the 2000 Census separately reported the categories "Pacific Islander / Hawaiian Along" and "Asian Alone." To make the racial groupings comparable with the 1990 Census, we first combined Pacific Islander / Hawaiian Alone with the Asian Alone category to form the category Asian and Pacific Islander category. Then we divided the category Two-or-More between the remaining five racial categories.

Exhibit B-3 presents the estimated percentage of the national population by five racial groups: (1) American Indian or Alaska Native, (2) Asian or Pacific Islander, (3) Black, (4) White, and (5) Other, as well as for four combinations: (1) American Indian or Alaska Native (AIAN)/White, (2) Asian or Pacific

Islander (API)/White, (3) Black/White, and (4) Other combinations. Slightly over 98 percent of individuals chose a single racial category, with 1.45 percent choosing three AIAN/White, API/White, and Black/White, and 0.30 choosing other combinations (e.g., Black/Asian). Exhibit B-3 also presents the estimated primary racial affiliation of individuals in these subcategories if they were to choose a single racial affiliation.

Exhibit B-3. Distribution of Racial Groups

Racial Category	% of Total U.S. Population [a]	% of Population in Sub-Groups by Primary Racial Affiliation [b]					
		AIAN	API	Black	White	Other	All
American Indian or Alaska Native (AIAN)	0.85	100	–	–	–	–	100
Asian or Pacific Islander (API)	3.35	–	100	–	–	–	100
Black	12.07	–	–	100	–	–	100
White	79.72	–	–	–	100	–	100
Other race	2.25	–	–	–	–	100	100
AIAN/White	0.89	12.4	–	–	80.9	6.7	100
API/White	0.30	–	34.6	–	46.9	18.4	100
Black/White	0.26	–	–	48.2	25.2	26.6	100
Other combinations [c]	0.30	–	–	–	–	100.0	100
Two-or-More Sub-Total [d]	1.75	6.3	5.9	7.2	52.9	27.7	100

[a] All percentages weighted to be nationally representative. Percentages taken from Parker and Makuc (2001, Table 2), who cited the National Health Interview Survey 1993-1995, APPENDIX: Percent Distribution (Standard Error) of Primary Racial Identification for Selected Detailed Race Groups.

[b] Primary racial affiliation based on survey results from Parker and Makuc (2001, Appendix).

[c] Parker and Makuc (2001) did not provide an estimate of the primary racial affiliation for "Other combinations, so we assume that it belongs to the "Other" category. Note that they did provide the primary racial affiliation for a fourth group "Black/AIAN:" 85.4% Black, 7.0% AIAN, and 7.6% Other. However, we do not have an estimate of the relative abundance of Black/AIAN in the general population, so we have dropped it from further consideration.

[d] As described in the text below, we calculated the percentages in this row from the percentages in the previous four rows for AIAN/White, API/White, Black/White, and Other combinations.

To estimate how to assign a single racial group for individuals that chose two or more racial groups, we used the results of Exhibit B-3 for the three main categories for which we an estimate of the primary racial affiliation: AIAN/White, API/White, and Black/White. To account for the 0.30 percent of the population in other combinations, we For each Census block, we assume that .89 / (.89+.30+.26+.30) = 50.8% of respondents in the Two or More category will fall into the AIAN / White category, and of these, 80.9% would primarily identify themselves as White if they were to choose a single racial category, 12.4% would primarily identify themselves as American Indian or Alaska Native, and 6.7% would primarily identify themselves as Other. Thus 0.508 * .809 = 41% of Two or More we will call White, 10% we identify as Native American, and 5% as Other.

We did not attempt to predict what respondents in the 'Other Combinations' category would have selected if they were to choose a single racial category, so we assume they are part of the "Other" category. To estimate the number of individuals in each of the five races, we performed the following calculations:

$$NativeAmerican = NativeAmericanAlone_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot AIAN\%_{AIAN/White} \right)$$

$$Asian = AsianAlone_{Pop} + PacificIslander/Hawaiian_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot API\%_{API/White} \right)$$

$$Black = BlackAlone_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Black\%_{Black/White} \right)$$

$$White = White\ Alone_{Pop} + Two\ \ or\ More_{Pop} \cdot \left(\begin{array}{l} \frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{AIAN/White} + \\ \frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{API/White} + \\ \frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{Black/White} \end{array} \right)$$

$$Other = Other\ Alone_{Pop} + Two\ \ or\ More_{Pop} \cdot \left(\begin{array}{l} \frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{AIAN/White} + \\ \frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{API/White} + \\ \frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{Black/White} + \\ \frac{Other\ \ Combinations_{Nat\%}}{MultipleRace_{Nat\%}} \end{array} \right)$$

This then reduces to:

$$Native\ American_{Pop} = Native\ American\ Alone_{Pop} + (0.063) Two\ or\ More_{Pop}$$

$$Asian_{Pop} = Asian\ Alone_{Pop} + Pacific\ Islander / Hawaiian_{Pop} + (0.059) Two\ or\ More_{Pop}$$

$$Black_{Pop} = Black\ Alone_{Pop} + (0.072) Two\ or\ More_{Pop}$$

$$White_{Pop} = White\ Alone_{Pop} + (0.530) Two\ or\ More_{Pop}$$

$$Other_{Pop} = White\ Alone_{Pop} + (0.276) Two\ or\ More_{Pop} \quad .$$

B .1.2 Estimating Population Levels in Alternative Age Groups

In calculating the population in age groups that may include a portion of one of the pre-specified demographic groups in Exhibit B-1, CAPMS assumes the population is uniformly distributed in the age group. For example, to calculate the number of children ages 3 through 12, CAPMS calculates:

$$age_{3-12} = \frac{1}{4} \cdot age_{1-4} + age_{5-9} + \frac{3}{5} \cdot age_{10-14} \quad .$$

B .2 Estimating Population Levels in Non-Census Years

To forecast population levels beyond 2000, CAPMS scales the 2000 Census-based estimate with the ratio of the county-level forecast for the future year of interest over the 2000 county-level population level. Woods & Poole (2001) provides the county-level population forecasts used to calculate the scaling ratios.

In the simplest case, where one is forecasting a single population variable, say, children ages 4 to 9 in the year 2010, CAMPS calculates:

$$age_{4-9,\, g,\, 2010} = age_{4-9,\, g,\, 2000} \cdot \frac{age_{4-9,\, county,\, 2010}}{age_{4-9,\, county,\, 2000}}$$

where the gth population grid-cell is wholly located within a given county.

In the case, where the gth grid-cell includes "n" counties in its boundary, the situation is somewhat more complicated. CAPMS first estimates the fraction of individuals in a given age group (e.g., ages 4 to 9) that reside in the part of each county within the gth grid-cell. CAPMS calculates this fraction by simply dividing the population all ages of a given county within the gth grid-cell by the total population in the gth grid-cell:

$$fraction\ of\ age_{4-9,\, g\ in\ county_c} = \frac{age_{all,\, g\ in\ county_c}}{age_{all,\, g}}$$

Multiplying this fraction with the number of individuals ages 4 to 9 in the year 2000 gives an estimate of the number of individuals ages 4 to 9 that reside in the fraction of the county within the gth grid-cell in the year 2000:

$$age_{4-9,\, g\ in\ county_c,\, 2000} = age_{4-9,\, g,\, 2000} \cdot fraction\ age_{4-9,\, g\ in\ county_c}$$

To then forecast the population in 2010, we scale the 2000 estimate with the ratio of the county projection for 2010 to the county projection for 2000:

$$age_{4-9,\, g\ in\ county_c,\, 2010} = age_{4-9,\, g\ in\ county_c,\, 2000} \cdot \frac{age_{4-9,\, county_c,\, 2010}}{age_{4-9,\, county_c,\, 2000}} \quad .$$

Combining all these steps for "c" counties within the gth grid-cell, we forecast the population of persons ages 4 to 9 in the year 2010 as follows:

$$age_{4-9,\, g,\, 2010} = \sum_{c=1}^{n} age_{4-9,\, g,\, 2000} \cdot \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \cdot \frac{age_{4-9,\, county_c,\, 2010}}{age_{4-9,\, county_c,\, 2000}}$$

In the case where there are multiple age groups and multiple counties, CAPMS first calculates the forecasted population level for individual age groups, and then combines the forecasted age groups. In calculating the number of children ages 4 to 12, CAPMS calculates:

$$age_{4-9,\, g,\, 2010} = \sum_{c=1}^{n} age_{4-9,\, g,\, 2000} \cdot \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \cdot \frac{age_{4-9,\, county_c,\, 2010}}{age_{4-9,\, county_c,\, 2000}}$$

$$age_{10-14,\, g,\, 2010} = \sum_{c=1}^{n} age_{10-14,\, g,\, 2000} \cdot \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \cdot \frac{age_{10-14,\, county_c,\, 2010}}{age_{10-14,\, county_c,\, 2000}}$$

$$age_{4-12,\, g,\, 2010} = age_{4-9,\, g,\, 2010} + \frac{3}{5} \cdot age_{10-14,\, g,\, 2010} \quad .$$

Since the Woods and Poole (2001) projections only extend through 2025, we used the existing projections and constant growth factors to provide additional projections. To estimate population levels beyond 2025, CAPMS linearly extrapolates from the final two years of data. For example, to forecast population in 2030, CAPMS calculates:

$$age_{4-9,\, 2030} = age_{4-9,\, 2025} + 5 \cdot \left(age_{4-9,\, 2025} - age_{4-9,\, 2024} \right) \quad .$$

Exhibit B-4 summarizes the forecasted age-stratified, state-level populations for 2020 and 2030. In addition, to provide a point of comparison, it includes population levels for year 2000.

Exhibit B-4. State-Level Population Estimates by Age Group

	2000			2020			2030		
State	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***
AL	1,126,337	2,740,965	579,798	1,212,702	3,057,540	909,065	1,284,828	3,069,518	1,195,921
AZ	1,371,099	3,091,693	667,839	1,832,737	4,351,254	1,309,943	2,117,749	4,708,843	1,812,966
AR	681,003	1,618,378	374,019	769,208	1,821,876	557,233	832,285	1,861,954	698,259
CA	9,254,212	21,021,768	3,595,658	10,105,474	25,308,061	5,717,329	10,895,067	26,084,324	7,681,464
CO	1,101,772	2,783,415	416,073	1,321,930	3,359,731	954,691	1,498,688	3,453,808	1,370,557
CT	839,051	2,096,330	470,183	821,773	2,110,504	588,222	845,210	1,991,695	728,973
DE	195,997	485,877	101,726	213,375	571,224	149,015	227,024	574,497	204,731
DC	120,659	381,502	69,898	95,389	337,146	103,401	93,833	305,565	123,728
FL	3,643,004	9,531,774	2,807,597	4,466,384	12,098,406	4,472,647	5,026,785	12,483,019	5,933,620
GA	2,176,259	5,224,918	785,275	2,600,100	6,296,967	1,328,722	2,851,139	6,620,751	1,778,194
ID	369,522	778,515	145,916	451,473	980,346	282,616	507,776	1,045,592	374,826
IL	3,247,904	7,671,362	1,500,025	3,286,653	8,148,579	2,069,429	3,425,612	7,962,757	2,669,430
IN	1,581,993	3,745,661	752,831	1,691,800	4,113,510	1,087,932	1,800,717	4,096,828	1,421,006
IA	737,415	1,752,696	436,213	734,433	1,820,333	593,034	766,374	1,750,358	755,945
KS	714,371	1,617,818	356,229	760,573	1,795,227	499,065	814,382	1,778,859	653,139
KY	998,042	2,538,933	504,793	1,077,101	2,762,379	801,696	1,154,120	2,750,564	1,052,988
LA	1,221,651	2,730,396	516,929	1,247,161	2,952,038	850,018	1,318,748	2,917,899	1,116,293
ME	299,691	791,830	183,402	284,880	852,466	289,399	297,507	807,626	394,873
MD	1,350,517	3,346,661	599,307	1,453,726	3,868,715	926,465	1,559,338	3,877,266	1,256,566
MA	1,508,818	3,980,116	860,162	1,533,618	4,071,543	1,144,857	1,604,543	3,871,104	1,469,089
MI	2,596,118	6,123,307	1,219,018	2,587,563	6,590,540	1,798,905	2,703,858	6,417,627	2,366,125
MN	1,285,100	3,040,113	594,266	1,413,120	3,525,458	948,035	1,547,597	3,524,435	1,298,319
MS	779,939	1,721,196	343,523	826,142	1,912,067	513,412	867,469	1,926,497	667,632
MO	1,428,853	3,410,978	755,379	1,526,846	3,830,433	1,062,471	1,631,969	3,798,554	1,404,065
MT	228,916	552,330	120,949	234,129	612,736	241,971	258,376	604,179	322,696
NE	450,372	1,028,696	232,195	483,340	1,148,129	329,112	522,703	1,142,368	426,556
NV	510,633	1,268,694	218,929	757,488	1,921,749	477,249	904,840	2,150,250	654,589
NH	309,490	778,326	147,970	321,958	906,776	236,489	346,320	897,774	329,510

Exhibit B-4. State-Level Population Estimates by Age Group (Continued)

	2000			2020			2030		
State	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***
NJ	2,074,020	5,227,192	1,113,136	2,126,538	5,560,594	1,507,553	2,222,228	5,402,892	1,949,786
NM	506,558	1,100,262	212,225	583,389	1,372,898	412,394	644,935	1,420,580	572,907
NY	4,696,232	11,831,869	2,448,352	4,487,417	11,815,310	3,179,326	4,540,245	11,246,710	3,953,934
NC	1,977,387	5,102,877	969,048	2,399,345	6,081,807	1,588,246	2,646,039	6,337,401	2,079,430
ND	162,017	385,705	94,478	152,979	407,052	152,185	160,056	387,072	203,240
OH	2,889,207	6,956,175	1,507,757	2,894,902	7,316,549	2,031,922	3,021,197	7,084,923	2,597,112
OK	894,531	2,100,173	455,950	968,204	2,255,616	685,395	1,037,634	2,249,445	865,166
OR	846,233	2,136,988	438,177	1,028,841	2,593,792	824,166	1,161,142	2,731,400	1,065,084
PA	2,930,189	7,431,699	1,919,165	2,807,320	7,589,422	2,473,482	2,879,828	7,112,827	3,152,928
RI	252,438	643,479	152,402	248,650	664,840	185,270	253,697	626,436	236,588
SC	1,017,627	2,509,052	485,333	1,125,147	2,936,359	879,310	1,217,702	2,989,589	1,188,398
SD	202,496	444,217	108,131	209,379	498,258	159,468	222,092	487,168	215,460
TN	1,402,958	3,583,013	703,311	1,614,405	4,118,556	1,147,546	1,759,007	4,213,846	1,513,183
TX	5,891,741	12,887,542	2,072,532	7,108,830	15,994,222	3,802,007	7,929,363	16,840,990	5,236,651
UT	724,466	1,318,481	190,222	989,440	1,826,327	368,454	1,120,100	2,046,412	497,421
VT	147,949	383,368	77,510	137,590	414,505	138,315	144,053	397,442	190,941
VA	1,743,459	4,542,721	792,333	1,955,331	5,201,333	1,308,689	2,132,729	5,295,036	1,734,954
WA	1,511,831	3,720,140	662,148	1,785,937	4,644,371	1,174,213	2,006,978	4,857,761	1,581,410
WV	404,484	1,126,965	276,895	388,379	1,094,529	403,851	404,280	1,038,496	488,364
WI	1,369,215	3,291,907	702,553	1,413,693	3,680,062	1,070,942	1,511,982	3,612,218	1,450,806
WY	128,585	307,504	57,693	124,005	314,574	117,862	132,595	301,356	154,460

B .3 Woods & Poole Data

Woods & Poole (2001) developed county-level forecasts for each year from 2000 through 2025, for three racial groups "Black," "White," and "Other," and by age and by gender. For the Hispanic ethnic group, Woods and Poole developed forecasts just for the total population, and not by age and gender. As discussed in the section on population forecasts, CAPMS uses these forecasts to simply scale the 2000 Census block data, in order to estimate the population in the population grid-cells for any given year after 2000.

B .3.1 Aligning Woods & Poole FIPS Codes with CAPMS FIPS Codes

The county geographic boundaries used by Woods & Poole are somewhat more aggregated than the county definitions used in the 2000 Census (and CAPMS), and the FIPS codes used by Woods and Poole are not always the standard codes used in the Census. To make the Woods and Poole data consistent with the county definitions in CAPMS, we disaggregated the Woods and Poole data and changed some of the FIPS codes. Exhibit B-5 lists the discrepancies in the county definitions between Woods & Poole and those used in CAPMS.

To assign the population in the more aggregated Woods & Poole county definitions to the more disaggregated definitions used in CAPMS (and the U.S. Census), we used the total county population from the 2000 U.S. Census. We then assumed that the age and racial groups were distributed uniformly across the CAPMS counties contained within a Woods & Poole county definition. For example, in estimating the population of children ages 4-9 in county "c" contained within a more broadly defined Woods & Poole county, we would do the following:

$$age_{4-9,\, county_c} = age_{4-9,\, W\&P\ county} \cdot \frac{age_{all,\, county_c}}{age_{all,\, W\&P\ county}}$$

After this factor was applied, we rounded the estimates to the nearest integer so as to avoid having data with "partial people."

Exhibit B-5. Linkage Between Woods & Poole County Definitions and CAPMS County Definitions

Woods and Poole Counties (FIPS)	Counties in CAPMS (FIPS)
Northwest Arctic Borough, AK (02188)	Kobuk, AK (02140)
Remainder of Alaska, AK (02999)	Aleutian Islands, AK (02010), Aleutian Islands East Borough, AK (02013), Aleutian Islands West Census Area, AK (02016), Bethel Census Area, AK (02050), Denali Borough, AK (02068), Dillingham Census Area, AK (02070), Haines Borough, AK (02100), Kenai Peninsula Borough, AK (02122), Lake and Peninsula Borough, AK (02164), North Slope Borough, AK (02185), Prince of Wales-Outer Ketchikan, AK (02201), Sitka Borough, AK (02220), Skagway-Yukatat-Angoon, AK (02231), Skagway-Hoonah-Angoon Census Area, AK (02232), Southeast Fairbanks Census Area, AK (02240), Valdez-Cordova Census Area, AK (02261), Wrangell-Petersburg Census Area, AK (02280), Yakutat Borough, AK (02282), Yukon-Koyukuk, AK (02290)
Yuma + La Paz, AZ (04027)	La Paz, AZ (04012), Yuma, AZ (04027)
Miami-Dade, FL (12086)	Dade, FL (12025)
Maui + Kalawao, HI (15901)	Kalawao, HI (15005), Maui, HI (15009)

Exhibit B-5. Linkage Between Woods & Poole County Definitions and CAPMS County Definitions (Continued)

Woods and Poole Counties (FIPS)	Counties in CAPMS (FIPS)
Fremont, ID (16043)	Fremont, ID (16043), Yellowstone Park, ID
Park, MT (30067)	Park, MT (30067), Yellowstone Park, MT (30113)
Valencia + Cibola, NM (35061)	Cibola, NM (35006), Valencia, NM (35061)
Halifax, VA (51083)	Halifax, VA (51083), South Boston City, VA (51780)
Albemarle + Charlottesville, VA (51901)	Albemarle, VA (51003), Charlottesville City, VA (51540)
Alleghany + Clifton Forge + Covington, VA (51903)	Alleghany, VA (51005), Clifton Forge City, VA (51560), Covington City, VA (51580)
Augusta + Staunton + Waynesboro, VA (51907)	Augusta, VA (51015), Staunton City, VA (51790), Waynesboro City, VA (51820)
Bedford + Bedford City, VA (51909)	Bedford, VA (51019), Bedford City, VA (51515)
Campbell + Lynchburg, VA (51911)	Campbell, VA (51031), Lynchburg City, VA (51680)
Carroll + Galax, VA (51913)	Carroll, VA (51035), Galax City, VA (51640)
Dinwiddie + Colonial Heights + Petersburg, VA (51918)	Dinwiddie, VA (51053), Colonial Heights City, VA (51570), Petersburg City, VA (51730)
Fairfax + Fairfax City + Falls Church City, VA (51919)	Fairfax, VA (51059), Fairfax City, VA (51600), Falls Church City, VA (51610)
Frederick + Winchester, VA (51921)	Frederick, VA (51069), Winchester City, VA (51840)
Greensville + Emporia, VA (51923)	Greensville, VA (51081), Emporia City, VA (51595)
Henry + Martinsville, VA (51929)	Henry, VA (51089), Martinsville City, VA (51690)
James City + Williamsburg, VA (51931)	James City County, VA (51095), Williamsburg City, VA (51830)
Montgomery + Radford, VA (51933)	Montgomery, VA (51121), Radford City, VA (51750)
Pittsylvania + Danville, VA (51939)	Pittsylvania, VA (51143), Danville City, VA (51590)
Prince George + Hopewell, VA (51941)	Prince George, VA (51149), Hopewell City, VA (51670)
Prince William + Manassas + Manassas Park, VA (51942)	Prince William, VA (51153), Manassas City, VA (51683), Manassas Park City, VA (51685)
Roanoke + Salem, VA (51944)	Roanoke, VA (51161), Salem City, VA (51775)
Rockbridge + Buena Vista + Lexington, VA (51945)	Rockbridge, VA (51163), Buena Vista City, VA (51530), Lexington City, VA (51678)
Rockingham + Harrisonburg, VA (51947)	Rockingham, VA (51165), Harrisonburg City, VA (51660)
Southampton + Franklin, VA (51949)	Southampton, VA (51175), Franklin City, VA (51620)
Spotsylvania + Fredericksburg, VA (51951)	Spotsylvania, VA (51177), Fredericksburg City, VA (51630)
Washington + Bristol, VA (51953)	Washington, VA (51191), Bristol City, VA (51520)
Wise + Norton, VA (51955)	Wise, VA (51195), Norton City, VA (51720)
York + Poquoson, VA (51958)	York, VA (51199), Poquoson City, VA (51735)
Shawano (includes Menominee), WI (55901)	Menominee, WI (55078), Shawano, WI (55115)

B .3.2 Age, Gender, Race, and Ethnicity

We generated the same 38 age and gender categories developed from the 1990 and 2000 Census data. Since these projections are available for every year of age, it is a simple matter to sum the individual years to get the same age categories used by CAPMS.

However, the only racial categories available are "White," "Black," and "Other." Since we do not have an Asian or Native American group, or an Other group which is consistent with the definition used by the 1990 and 2000 Census data, we assume that projection data's Other category is representative of all 3 groups, and that they move together over time.

The county projections only forecast the Hispanic population of all ages, and does not have separate gender and age forecasts. Lacking further information, we use the ratio of future-year all age population to the year 2000 all age population when forecasting any particular age group of Hispanics. In effect, we assume for all forecast years the same distribution of age and gender as found in the 2000 Census.

B .3.3 Creating Growth Ratios from Absolute Population Values

For each year from 2000 through 2025 and for each of the 256 demographic groups listed in Exhibit B-1, CAPMS stores the ratio of the future-year to year 2000 county-level population projections. As described below, these ratios are used to forecast population levels in the population grid-cells used by CAPMS to health effects.

Note that there are a small number of cases were the 2000 county population for a specific demographic group is zero, so the ratio of any future year to the year 2000 data is undefined. In these relatively rare cases, we set the year 2000 ratio and all subsequent ratios to 1, assuming no growth.

There are an even smaller number of cases where a total population variable dwindles from some non-zero number to zero, creating ratios of zero. Variables which represent a subpopulation of the first variable may not be zero, however. In these cases, we set all subset population variables for that year to zero.

For instance, if a county only had one person in it for the year 2000 - a 79 year old black male - we set all variables (excluding total variables and BlackMale75to79) to a ratio of 1, because their 2000 values of 0 produce undefined ratios. If the man dies at age 82, the total black population variable for years 2003 and beyond is calculated as 0/1 = 0. Thus for each of those years where the total black population is listed as zero, we go back and set all black population variables to zero, to reflect the knowledge that the block is empty. For all variables except the BlackMale75to79 age group (already zero), 1 becomes 0.

Appendix C: Air Quality Forecasts

This appendix summarizes the air pollution measures used in this analysis for PM and ozone. Exhibits C-1 through C-5 presents summaries of the air quality metrics used in this analysis.

Exhibit C-1. Summary of Ozone Air Quality Metrics Used in Benefits Analysis for Nonroad Engine/Diesel Fuel Standards: Eastern U.S.

		2020 [c]			2030 [c]		
Metric [a]	**Statistic [b]**	***Base Case***	***Change***	***% Change***	***Base Case***	***Change***	***% Change***
1-Hour Maximum	Minimum	28.85	-0.81	-2.80%	28.81	-1.24	-4.31%
	Maximum	93.94	-0.85	-0.90%	94.70	-1.61	-1.70%
	Average	45.54	-1.05	-2.30%	45.65	-1.66	-3.64%
	Median	45.45	-1.23	-2.71%	45.52	-1.73	-3.80%
	Pop-Weighted Average	51.34	-0.67	-1.31%	51.47	-1.16	-2.25%
5-Hour Average	Minimum	24.90	-0.67	-2.68%	24.87	-1.03	-4.13%
	Maximum	68.69	-0.20	-0.29%	69.11	-0.44	-0.64%
	Average	38.99	-0.85	-2.17%	39.08	-1.35	-3.45%
	Median	38.94	-0.92	-2.39%	39.00	-1.40	-3.58%
	Pop-Weighted Average	42.77	-0.47	-1.10%	42.90	-0.84	-1.96%
8-Hour Average	Minimum	24.15	-0.64	-2.64%	24.12	-0.98	-4.07%
	Maximum	68.30	-0.21	-0.31%	68.72	-0.46	-0.67%
	Average	38.46	-0.83	-2.16%	38.55	-1.33	-3.44%
	Median	38.44	-0.89	-2.33%	38.50	-1.45	-3.76%
	Pop-Weighted Average	42.07	-0.46	-1.08%	42.19	-0.82	-1.93%
12-Hour Average	Minimum	22.42	-0.58	-2.57%	22.40	-0.89	-3.96%
	Maximum	66.06	-0.17	-0.25%	66.46	-0.38	-0.58%
	Average	36.59	-0.78	-2.13%	36.66	-1.25	-3.40%
	Median	36.61	-0.84	-2.30%	36.66	-1.43	-3.89%
	Pop-Weighted Average	39.65	-0.40	-1.00	39.75	-0.72	-1.80%
24-Hour Average	Minimum	15.20	-0.35	-2.28%	15.19	-0.54	-3.52%
	Maximum	55.95	0.10	0.18%	56.23	0.04	0.07%
	Average	28.93	-0.57	-1.96%	28.98	-0.91	-3.14%
	Median	28.92	-0.63	-2.15%	28.98	-1.01	-3.48%
	Pop-Weighted Average	30.24	-0.18	-0.60%	30.29	-0.37	-1.23%

[a] These ozone metrics are calculated at the CAMX grid-cell level and based on the results of spatial and temporal Voronoi Neighbor Averaging. Except for the daily 24-hour average, these ozone metrics are calculated over relevant time periods during the daylight hours of the "ozone season," i.e., May through September. For the 5-hour average, the relevant time period is 10 am to 3 pm; for the 8-hour average, it is 9 am to 5 pm; and, for the 12-hour average it is 8:00 am to 8 pm.

[b] The base case minimum (maximum) is the value for the CAMX grid cell with the lowest (highest) value. We calculated the population-weighted average by summing the product of the projected CAMX grid-cell population and the estimated CAMX grid-cell seasonal ozone concentration over all grid-cells, and then dividing by the total population.

[c] The change is defined as the control case value minus the base case value. The percent change is the "Change" divided by the "Base Case," and then multiplied by 100 to convert the value to a percentage.

Exhibit C-2. Summary of Ozone Air Quality Metrics Used in Benefits Analysis for Nonroad Engine/Diesel Fuel Standards: Western U.S.

		2020 [c]			2030 [c]		
Metric [a]	**Statistic [b]**	***Base Case***	***Change***	***% Change***	***Base Case***	***Change***	***% Change***
1-Hour Maximum	Minimum	27.48	-0.01	-0.03%	27.48	-0.01	-0.05%
	Maximum	201.28	4.87	2.42%	208.02	6.26	3.01%
	Average	47.02	-0.62	-1.31%	47.04	-0.97	-2.07%
	Median	46.10	-0.56	-1.19%	46.06	-0.66	-1.43%
	Pop-Weighted Average	63.80	0.34	0.54%	64.23	0.38	0.58%
5-Hour Average	Minimum	24.20	-0.01	-0.04%	24.21	-0.01	-0.05%
	Maximum	163.41	2.55	1.56%	168.89	6.04	3.57%
	Average	41.11	-0.52	-1.26%	41.13	-0.82	-2.00%
	Median	40.48	-0.40	-1.04%	40.46	-0.69	-1.70%
	Pop-Weighted Average	53.56	0.45	0.84%	53.89	0.55	1.03%
8-Hour Average	Minimum	23.77	-0.01	-0.04%	23.77	-0.01	-0.05%
	Maximum	157.49	1.33	0.84%	161.92	5.94	3.67%
	Average	40.68	-0.51	-1.25%	40.69	-0.81	-1.99%
	Median	40.11	-0.36	-1.03%	40.09	-0.72	-1.79%
	Pop-Weighted Average	51.96	0.46	0.88%	52.29	0.57	1.10%
12-Hour Average	Minimum	22.13	0.31	1.39%	22.09	0.44	2.01%
	Maximum	140.48	1.65	1.18%	143.59	1.78	1.24%
	Average	39.30	-0.48	-1.23%	39.31	-0.77	-1.95%
	Median	38.85	-0.38	-0.97%	38.82	-0.58	-1.50%
	Pop-Weighted Average	47.68	0.49	1.02%	47.99	0.63	1.32%
24-Hour Average	Minimum	14.08	0.22	1.60%	14.03	0.32	2.30%
	Maximum	95.27	0.41	0.43%	96.59	0.29	0.30%
	Average	33.42	-0.38	-1.14%	33.42	-0.61	-1.82%
	Median	32.97	-0.30	-0.89%	32.95	-0.61	-1.85%
	Pop-Weighted Average	35.53	0.47	1.31%	35.74	0.63	1.77%

[a] These ozone metrics are calculated at the CAMX grid-cell level and based on the results of spatial and temporal Voronoi Neighbor Averaging. Except for the daily 24-hour average, these ozone metrics are calculated over relevant time periods during the daylight hours of the "ozone season," i.e., May through September. For the 5-hour average, the relevant time period is 10 am to 3 pm; for the 8-hour average, it is 9 am to 5 pm; and, for the 12-hour average it is 8:00 am to 8 pm.

[b] The base case minimum (maximum) is the value for the CAMX grid cell with the lowest (highest) value. We calculated the population-weighted average by summing the product of the projected CAMX grid-cell population and the estimated CAMX grid-cell seasonal ozone concentration over all grid-cells, and then dividing by the total population.

[c] The change is defined as the control case value minus the base case value. The percent change is the "Change" divided by the "Base Case," and then multiplied by 100 to convert the value to a percentage.

Exhibit C-3. Forecasted SUM06 in 2020 and 2030

		2020 [c]			2030 [c]		
Region [a]	**Statistic [b]**	***Base Case***	***Change***	***% Change***	***Base Case***	***Change***	***% Change***
Eastern U.S.	Minimum	0.00	0.00	-	0.00	0.00	-
	Maximum	67.24	-3.30	-4.91%	68.63	-5.54	-8.07%
	Average	4.74	-0.72	-15.10%	4.88	-1.09	-22.43%
	Median	2.18	-0.76	-35.02%	2.21	-0.77	-34.84%
	Population-Weighted Average	12.81	-0.71	-5.57%	13.23	-1.17	-8.84%
Western U.S.	Minimum	0.00	0.00	-	0.00	0.00	-
	Maximum	132.73	6.09	4.59%	137.71	8.45	6.14%
	Average	2.78	-0.22	-7.85%	2.83	-0.33	-11.72%
	Median	0.00	0.00	-	0.00	0.00	-
	Population-Weighted Average	33.54	0.39	1.17%	34.63	0.43	1.24%

[a] SUM06 is defined as the cumulative sum of hourly ozone concentrations over 0.06 ppm (or 60 ppb) that occur during daylight hours (from 8am to 8pm) in the months of May through September. It is calculated at the county level for use in agricultural benefits based on the results of temporal and spatial Voronoi Neighbor Averaging.

[b] The base case minimum (maximum) is the value for the county level observation with the lowest (highest) concentration. We calculated the population-weighted average by summing the product of the year 2000 county population and the estimated county level ozone concentration over all counties, and then dividing by the total population of all counties.

[c] The change is defined as the control case value minus the base case value. The percent change is the "Change" divided by the "Base Case," which is then multiplied by 100 to convert the value to a percentage.

Exhibit C-4. Summary of Base Case PM Air Quality and Changes Due to Nonroad Engine/Diesel Fuel Standards: 2020 and 2030

	2020 [b]			2030 [b]		
Statistic for Annual Means [a]	***Base Case***	***Change***	***% Change***	***Base Case***	***Change***	***% Change***
Minimum for a	2.18	-0.02	-0.78%	2.33	-0.02	-1.01%
Maximum	29.85	-1.36	-4.56%	32.85	-2.03	-6.18%
Average	8.10	-0.20	-2.49%	8.37	-0.28	-3.38%
Median	7.50	-0.18	-2.68%	7.71	-0.22	-2.80%
Population-Weighted Average	12.42	-0.42	-3.34%	13.07	-0.59	-4.48%

[a] These statistics are for the annual mean $PM_{2.5}$ levels for REMSAD grid-cells, and are based on the results of spatial and temporal Voronoi Neighbor Averaging. The base case minimum (maximum) is the value for the populated grid-cell with the lowest (highest) annual mean. The "average" is the simple average of all REMSAD grid-cell means. The "median" is the median of the REMSAD grid-cell means. We calculated the "population-weighted average" by summing the product of the projected grid-cell population and the estimated PM concentration over all grid-cells, and then dividing by the total population of all grid-cells.

[b] The change is defined as the control case value minus the base case value. The percent change is the "Change" divided by the "Base Case," which is then multiplied by 100 to convert the value to a percentage.

Table C-5 Summary of Absolute and Relative Changes in PM Air Quality Due to Nonroad Engine/Diesel Fuel Standards: 2020 and 2030

Type of Change [a]	Statistic [b]	2020 $PM_{2.5}$ Annual Mean	2030 $PM_{2.5}$ Annual Mean
Absolute Change from Base Case ($\mu g/m^3$)	Minimum	-1.36	-2.03
	Maximum	-0.02	-0.02
	Average	-0.20	-0.28
	Median	-0.19	-0.26
	Population-Weighted Average	-0.42	-0.59
Relative Change from Base Case (%)	Minimum	-12.24%	-15.52%
	Maximum	-0.33%	-0.44%
	Average	-2.44%	-3.32%
	Median	-2.33%	-3.13%
	Population-Weighted Average	-3.28%	-4.38%

[a] The changes are based on the results of spatial and temporal Voronoi Neighbor Averaging. The absolute change is defined as the control case value minus the base case value for each REMSAD grid-cell. The relative change is defined as the absolute change divided by the base case value, or the percentage change, for each gridcell. The information reported for the relative changes does not necessarily reflect the same gridcells as those portrayed for absolute changes.

[b] We calculated the population-weighted average by summing the product of the projected REMSAD grid-cell population and the estimated gridcell PM absolute/relative measure of change over all grid-cells, and then dividing by the total population of all grid-cells.

Appendix D: Derivation of Concentration-Response Functions

This appendix reviews the steps we performed in taking models from the epidemiological study and converting them into concentration-response (C-R) functions, when we then use to quantify the change in adverse health effects due to a change in air pollution exposure. The most common functional forms are linear, log-linear, and logistic, with the log-linear model by far the most widely used. All three are discussed below.

D.1 The Linear Model

A linear model between the adverse health effect, y, and the pollutant concentration, x, is of the form

$$y = \alpha + \beta \cdot x$$

A linear model includes the factors that are believed to affect the incidence of the health effect, of which the pollutant would be one. So, the variable "α" in the linear C-R function consists of all the other independent variables in the regression, typically evaluated at their mean values, times their respective coefficients.

The function describing the relationship between a *change* in x and the corresponding *change* in incidence (rate) of the health effect from the baseline level (y_0) to the post-control level (y_c) is then:

$$\Delta y = y_c - y_0 = \beta \cdot (x_c - x_0) = \beta \cdot \Delta x .$$

If y denotes an incidence rate, then Δy denotes the change in the incidence rate. The expected number of cases avoided would then be calculated by multiplying this Δy by the relevant population. If y denotes an incidence count, then the β is first divided the baseline study population to generate an incidence rate. The expected number of cases avoided can then be calculated by multiplying Δy by the relevant population of interest:

$$CasesAvoided = \beta \cdot \Delta x \cdot pop.$$

The coefficient, β, and standard error of β (σ_β) are reported directly in studies presenting results from linear regression models.

D.2 The Log-linear Model

The most commonly used functional form for criteria air pollutant C-R functions is the log-linear model. It defines the relationship between x and y to be of the form

$$y = B \cdot e^{\beta \cdot x}$$

or, equivalently,

$$\ln(y) = \alpha + \beta \cdot x ,$$

where the parameter B is the incidence (rate), y, when the pollutant concentration, x, is zero; the parameter β is the coefficient of x; ln(y) is the natural logarithm of y; and α = ln(B).[56]

Estimating Avoided Cases

The relationship between Δx and Δy is:

$$\Delta y = y_c - y_0 = Be^{\beta \cdot x_c} - Be^{\beta \cdot x_0}.$$

This may be rewritten as:

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

where y_0 is the baseline incidence (rate) of the health effect -- i.e., the incidence (rate) before the change in x. If y is incidence rate rather than incidence, then the change in incidence rate, Δy, must be multiplied by the relevant population to get the expected number of cases avoided. For example, if y denotes the annual number of cases of the adverse health effect per 100,000 population, and pop denotes the population, then the expected number of cases avoided is calculated as

$$CasesAvoided = \Delta y \cdot (\frac{pop}{100{,}000}).$$

Estimating the Coefficient (β)

Epidemiological studies that estimate log-linear C-R functions often report a relative risk for a specific Δx, rather than the coefficient, β, in the C-R function itself. The relative risk (RR) is simply the ratio of two risks corresponding to two levels of pollutant concentration – the "high" risk (corresponding to the higher pollutant level, $x = x_{high}$) and the lower risk (corresponding to the lower pollutant level, $x = x_{low}$):

$$RR = \frac{y_{high}}{y_{low}}.$$

Using the original log-linear C-R function above, it can be shown that the relative risk associated with a specific change in pollutant concentration of $\Delta x^* = x_{high} - x_{low}$ can be written as

$$RR_{\Delta x^*} = \frac{y_{high}}{y_{low}} = e^{\beta \cdot \Delta x^*}.$$

Taking the natural log of both sides, the coefficient in the C-R function underlying the relative risk can be derived as:

$$\beta = \frac{\ln(RR)}{\Delta x^*}.$$

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[56] Other covariates besides pollution clearly affect mortality. The parameter B might be thought of as containing these other covariates, for example, evaluated at their means. That is, $B = B_o \exp\{\beta_1 x_1 + ... + \beta_n x_n\}$, where B_o is the incidence of y when all covariates in the model are zero, and $x_1, ..., x_n$ are the other covariates evaluated at their mean values. The parameter B drops out of the model, however, when changes in y are calculated, and is therefore not important.

Once the pollutant coefficient, β, has been calculated, the change in incidence (rate), Δy, corresponding to any change in pollutant concentration, Δx, can be calculated, using the relationship between Δx and Δy given above, the baseline incidence (rate) and assessment population.

Estimating the Standard Error of β (σ_β)

The standard error of β (σ_β) is not often directly reported in studies presenting results from log-linear regression models. Results are most commonly presented as a relative risk and 95% confidence interval. The 95% confidence interval is defined as follows:

$$95\%CI = e^{(\beta \cdot \Delta x \pm 1.96 \cdot \sigma_\beta \Delta x)}$$

Based on this equation, the standard error of β (σ_β) can be estimated from the relative risk (RR), upper limit of the 95% confidence interval (UL), and lower limit of the 95% confidence interval (LL), as follows:

$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(UL)}{\Delta x} - \frac{\ln(RR)}{\Delta x}\right)}{1.96}$$

$$\sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(RR)}{\Delta x} - \frac{\ln(LL)}{\Delta x}\right)}{1.96}$$

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

Some studies report only a central effect estimate and t-statistic. The t-statistic describes the strength of the observed pollutant-health effect association. It is defined as the ratio of the coefficient, β, to the standard error of β (σ_β). The standard error of β (σ_β) can, therefore, be estimated from the t-statistic as follows:

$$\sigma_\beta = \frac{\beta}{t}.$$

The Log-Linear Model: An Example

Lippmann et al. (2000) reported a relative risk (RR) of 1.045 for premature (non-accidental) mortality associated with an increase in daily $PM_{2.5}$ of 36 μg/m^3 in Detroit, MI. The $PM_{2.5}$ coefficient in the C-R function from which the RR was derived was back-calculated to be:

$$\beta = \frac{\ln(1.045)}{36} = 0.001223 .$$

Suppose we use the C-R function from Lippmann et al. (2000) to estimate the change in incidence of premature deaths in Wayne County, MI (which contains Detroit) in the year 2000 resulting from a decrease in $PM_{2.5}$ concentration of 15 μg/m^3 per day. The baseline incidence of non-accidental mortality in Detroit is estimated to be 891.12 per year per 100,000 general population (for 1998)*, or 2.441 per day per 100,000 general population. The population of Wayne County, MI in the year 2000 is estimated to be 2,061,162. The inputs to this calculation are:

$\Delta x = -15$
$y_0 = 2.441$ per day per 100,000 general population
$\beta = 0.001223$
general population of Wayne County, MI = 2,061,162.

The number of avoided premature deaths per day is estimated to be:

$$\Delta y = y_0 (e^{\beta * \Delta x} - 1) * (pop / 100{,}000)$$

$$= 2.441 * (e^{-0.001223*15} - 1) * 20.61162$$

$$= -0.914503325 \quad .$$

That is, a decrease in $PM_{2.5}$ of 15 μg/m^3 per day is predicted to result in 0.914503325 premature deaths avoided per day in Wayne County, MI. Over the year (the year 2000 was a leap year, and so had 366 days), that's

0.914503325*366
= 334.7 premature deaths avoided.

*Data obtained from the Centers for Disease Control (CDC Wonder, an online database).

D.3 The Logistic Model

In some epidemiological studies, a logistic model is used to estimate the probability of an occurrence of an adverse health effect. Given a pollutant level, x, and a vector of other explanatory variables, Z, the logistic model assumes the probability of an occurrence is:

$$y = prob\left(occurrence | \beta x, \alpha Z\right) = \left(\frac{e^{\beta x} e^{\alpha Z}}{1 + e^{\beta x} e^{\alpha Z}} \right) ,$$

where β is the coefficient of the pollutant concentration, x, and α is a vector of coefficients of the variables in the vector Z.[57]

Estimating Avoided Cases

The *change* in the probability of an occurrence (Δy) corresponding to a change in the level of the pollutant from x_c to x_0 (= Δx), all other covariates held constant, may be derived from the original C-R function above:
Once again, to calculate the expected number of avoided cases of the adverse effect, it is necessary to

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

multiply by the population:[58]

$$CasesAvoided = \Delta y \cdot pop.$$

Estimating the Coefficient (β)

The estimated pollutant coefficient, β, in the original C-R function is typically not reported in studies that use the logistic model. Instead, the odds ratio corresponding to a specific change in x is reported.

The odds of an occurrence is defined as:

$$odds = \frac{y}{1 - y} \, .$$

It can be shown that

$$odds = \frac{y}{1 - y} = e^{\beta x} e^{\alpha Z}$$

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[57] Greene (1997, Chapter 19) presents models with discrete dependent variables; in particular, page 874 presents the logit model. (See also Judge et al. (1985, p. 763).)

[58] Note that because Δy here is a change in probability of occurrence (rather than a change in the rate per 100,000 population), it is necessary to multiply by the population rather than by the population/100,000.

The *odds ratio* is just the ratio of the odds when the pollutant is at a specified higher level, x_{high}, to the odds when the pollutant is at a specified lower level, x_{low}:

$$odds\,ratio = \frac{odds_{high}}{odds_{low}} = \frac{e^{\beta x_{high}}}{e^{\beta x_{low}}} = e^{\beta(x_{high} - x_{low})}.$$

Often the odds ratio corresponding to a specified change in x, call it Δx*, is the only measure of the effect of x reported from a study using a logistic model (just as the relative risk corresponding to a specified change in x is often the only measure of the effect of x reported from a study using a log-linear model). However, it is easy to calculate the underlying pollutant coefficient, β, from the odds ratio as follows:

$$odds\ ratio = e^{\beta \Delta x^*}$$

$$\ln(odds\ ratio) = \beta \Delta x^*$$

$$\Rightarrow \beta = \frac{\ln(odds\,ratio)}{\Delta x^*}.$$

Given the pollutant coefficient, β, and the baseline probability of occurrence, y_0, the change in the probability, Δy, associated with any change in pollutant concentration, Δx, can be derived using the equation for Δy above. The expected number of avoided cases of the adverse effect is then obtained by multiplying by the population.

Estimating the Standard Error of β (σ_β)

The standard error of β (σ_β) is not often directly reported in studies presenting results from logistic regression models. Results are most commonly presented as an odds ratio and 95% confidence interval. The 95% confidence interval is defined as follows:

$$95\%CI = e^{(\beta \Delta x \pm 1.96\sigma_\beta)}$$

Based on this equation, the standard error of β (σ_β) can be estimated from the odds ratio (OR), upper limit of the 95% confidence interval (UL), and lower limit of the 95% confidence interval (LL), as follows:

$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(UL)}{\Delta x} - \frac{\ln(OR)}{\Delta x}\right)}{1.96}$$

$$\sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(OR)}{\Delta x} - \frac{\ln(LL)}{\Delta x}\right)}{1.96}$$

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

Some studies report only a central effect estimate and t-statistic. The t-statistic describes the strength of the observed pollutant-health effect association. It is defined as the ratio of the coefficient, β, to the standard error of β (σ_β). The standard error of β (σ_β) can, therefore, be estimated from the t-statistic as follows:

$$\sigma_\beta = \frac{\beta}{t}.$$

The Logistic Model: An Example

Schwartz and Neas (2000) reported an odds ratio of 1.33 for lower respiratory symptoms (LRS) among school children, ages 7 - 14, corresponding to an increase in daily $PM_{2.5}$ concentration of 15 μg/m^3. The $PM_{2.5}$ coefficient in the logistic C-R function from which the odds ratio was derived is back-calculated as

$$\beta = \frac{\ln(1.33)}{15} = 0.019012 .$$

The baseline incidence rate, y_0, (the probability per child per day of lower respiratory symptoms) was estimated in the study to be 0.0012.

Suppose we use the logistic C-R function from Schwartz and Neas (2000) to estimate the number of days with LRS avoided among schoolchildren, ages 7-14, in St. Louis during the warm months of April through August (the months used in the study) if $PM_{2.5}$ concentrations were reduced by 10 μg/m^3 each day. The inputs to this calculation are:

$\Delta x = x_c - x_0 = -10$
$y_0 = 0.0012$
$\beta = 0.019012$
the number of days in April through August = 153
the number of children, ages 7 - 14, in St. Louis area (9 counties) = 307,170.

The number of avoided LRS days among children ages 7-14 in the St. Louis area resulting from a decrease of 10 μg/m^3 $PM_{2.5}$ per day is estimated to be

$$\Delta y = \left[\frac{y_0}{(1 - y_0) * e^{-\beta * \Delta x} + y_0} - y_0 \right] * pop$$

$$= \left[\frac{0.0012}{(1 - 0.0012) * e^{-0.019012*(-10)} + 0.0012} - 0.0012 \right] * 307{,}170$$

= -0.0002076*307,170 = –63.7568 per day.

There are 153 days in April through August, for a total of -63.7568*153 =

-9754.79, or
9,754.79 LRS days avoided.

Appendix E: Baseline Incidence Rates for Adverse Health Effects

Concentration-response (C-R) functions developed from log-linear or logistic models estimate the percent change in an adverse health effect associated with a given pollutant change. In order to estimate the absolute change in incidence using these functions, we need the baseline incidence rate of the adverse health effect. This appendix describes the data used to estimate baseline incidence rates for the health effects considered in this analysis.

E.1 Mortality

Age, cause, and county-specific mortality rates were obtained from the U.S. Centers for Disease Control (CDC) for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at http://wonder.cdc.gov/. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across three years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25-34 year old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups. Population-weighted national mortality rates are presented in Exhibit E-1.

Exhibit E-1. National Mortality Rates for Selected Conditions, by Age Group

Mortality Category (ICD codes)	Mortality Rate by Age Group (deaths per 100 people per year)									
	25+	30+	25-29	30-34	35-44	45-54	55-64	65-74	75-84	85+
All-Cause	1.288	1.436	0.119	0.119	0.211	0.437	1.056	2.518	5.765	15.160
Non-Accidental (ICD <800)	1.219	1.366	0.057	0.057	0.150	0.383	1.006	2.453	5.637	14.859
Chronic Lung Disease (ICD 490-496)	0.062	0.070	0.001	0.001	0.002	0.009	0.046	0.166	0.367	0.561
Cardio-Pulmonary	0.650	0.731	0.013	0.013	0.044	0.143	0.420	1.163	3.179	9.846

Source: We obtained data from 1996-1998 from the CDC Wonder (http://wonder.cdc.gov/). County-specific rates are used in the C-R functions.

E.2 Hospitalizations

Regional hospitalization counts were obtained from the National Center for Health Statistics' (NCHS) National Hospital Discharge Survey (NHDS). NHDS is a sample-based survey of non-Federal, short-stay

hospitals (<30 days)[59], and is the principal source of nationwide hospitalization data. The survey collects data on patient characteristics, diagnoses, and medical procedures.

Public use data files for the year 1999 survey were downloaded[60] and processed to estimate hospitalization counts by region. NCHS groups states into four regions using the following groupings defined by the U.S. Bureau of the Census:

- Northeast - Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Pennsylvania
- Midwest - Ohio, Indiana, Illinois, Michigan, Wisconsin, Minnesota, Iowa, Missouri, North Dakota, South Dakota, Nebraska, Kansas
- South - Delaware, Maryland, District of Columbia, Virginia, West Virginia, North Carolina, South Carolina, Georgia, Florida, Kentucky, Tennessee, Alabama, Mississippi, Arkansas, Louisiana, Oklahoma, Texas
- West - Montana, Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada, Washington, Oregon, California, Alaska, Hawaii

We calculated per capita hospitalization rates, by dividing these counts by the estimated regional population estimates for 1999 that we derived from the U.S. Bureau of the Census and the population projections used by NHDS to generate the counts. Note that NHDS started with hospital admission counts, based on a sample of admissions, and then they used population estimates to generate population-weighted hospital admission counts that are representative of each region. This weighting used forecasts of 1999 population data. Ideally, we would use these same forecasts to generate our admission rates. However, while NHDS presented counts of hospital admissions with a high degree of age specificity, it presented regional population data for only four age groups: 0-14, 15-44, 45-64, and 65+.[61] Using only the NHDS data, we would be limited to calculating regional admission rates for four groups. Because we are interested in a broader range of age groups, we turned to 2000 Census.

We used the 2000 Census to obtain more age specificity, and then corrected the 2000 Census figures so that the total population equaled the total for 1999 forecasted by NHDS. That is, we sued the following procedure: (1) we calculated the count of hospital admissions by region in 1999 for the age groups of interest, (2) we calculated the 2000 regional populations corresponding to these age groups, (3) calculated regional correction factors, that equal the regional total population in 1999 divided by the regional total population in 2000 by region, (4) multiplied the 2000 population estimates by these correction factors, and (5) divided the 1999 regional count of hospital admissions by the estimated 1999 population.

The endpoints in hospitalization studies are defined using different combinations of ICD codes. Rather than generating a unique baseline incidence rate for each ICD code combination, for the purposes of this analysis, we identified a core group of hospitalization rates from the studies and applied the appropriate combinations of these rates in the C-R functions:

- all respiratory (ICD-9 460-519)

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[59] The following hospital types are excluded from the survey: hospitals with an average patient length of stay of greater than 30 days, federal, military, Department of Veterans Affairs hospitals, institutional hospitals (e.g. prisons), and hospitals with fewer than six beds.

[60] Data are available at ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/

[61] See: 1999nhds_summary.pdf (p. 187) for published regional population estimates for 1999.

- chronic lung disease (ICD-9 490-496)
- asthma (ICD-9 493)
- pneumonia (ICD-9 480-487)
- acute bronchitis (ICD-9 466)
- acute laryngitis (ICD-9 464)
- all cardiovascular (ICD-9 390-459)
- ischemic heart disease (ICD-9 410-414)
- dysrhythmia (ICD-9 427)
- congestive heart failure (ICD-9 428)

For each C-R function, we selected the baseline rate or combination of rates that most closely matches to the study endpoint definition. For studies that define chronic lung disease as ICD 490-492, 494-496, we subtracted the incidence rate for asthma (ICD 493) from the chronic lung disease rate (ICD 490-496). In some cases, the baseline rate will not match exactly to the endpoint definition in the study. For example, Burnett et al. (2001) studied the following respiratory conditions in infants <2 years of age: ICD 464.4, 466, 480-486, 493. For this C-R function we apply an aggregate of the following rates: ICD 464, 466, 480-487, 493. Although they do not match exactly, we assume that relationship observed between the pollutant and study-defined endpoint is applicable for the additional codes. Exhibit E-2 presents a summary of the national hospitalization rates for 1999 from NHDS.

Exhibit E-2. Hospitalization Rates, by Region and Age Group

Hospitalization Category		ICD-9 Codes	Hospitalization Rate by Age Group (admissions per 100 people per year)						
			0-18	18-24	25-34	35-44	45-54	55-64	65+
Respiratory	all respiratory	460-519	1.066	0.271	0.318	0.446	0.763	1.632	5.200
	acute laryngitis	464	0.055	0.002	0.001	0.002	0.008	0.000	0.005
	acute bronchitis	466	0.283	0.017	0.014	0.017	0.027	0.040	0.156
	pneumonia	480-487	0.308	0.069	0.103	0.155	0.256	0.561	2.355
	asthma	493	0.281	0.081	0.110	0.099	0.144	0.161	0.205
	chronic lung disease	490-496	0.291	0.089	0.124	0.148	0.301	0.711	1.573
Cardiovascular	all cardiovascular	390-459	0.043	0.084	0.206	0.678	1.926	4.389	11.629
	ischemic heart disease	410-414	0.004	0.008	0.031	0.231	0.902	2.021	3.708
	dysrhythmia	427	0.011	0.017	0.027	0.076	0.158	0.392	1.387
	congestive heart failure	428	0.003	0.005	0.011	0.011	0.160	0.469	2.167

Source: As described in the text, we obtained the regional count of hospital admissions from National Hospital Discharge Survey (NHDS), and we obtained the population data from the 2000 U.S. Census and NHDS.

E.3 Emergency Room Visits for Asthma

Regional asthma emergency room visit counts were obtained from the National Hospital Ambulatory Medical Care Survey (NHAMCS). NHAMCS is a sample-based survey, conducted by NCHS, designed

to collect national data on ambulatory care utilization in hospital emergency and outpatient departments of non-Federal, short-stay hospitals (<30 days).[62]

Public use data files for the year 2000 survey were downloaded[63] and processed to estimate hospitalization counts by region. We obtained population estimates from the 2000 U.S. Census. The NCHS regional groupings described above were used to estimate regional emergency room visit rates. Exhibit E-3 presents the estimated asthma emergency room rates by region.

Exhibit E-3. Emergency Room Visit Rates for Asthma, by Region and Age Group

ER Category	ICD-9 Code	Region	ER Visit Rate (visits per 100 people per year)		
			0-18	18-64	65+
asthma	493	Northeast	0.761	0.802	0.300
		Midwest	1.476	0.877	0.334
		South	1.243	0.420	0.192
		West	0.381	0.381	0.137

Source: We obtained ER visit counts for the year 2000 from the National Hospital Ambulatory Medical Care Survey (NHAMCS) and population data were obtained from the 2000 U.S. Census.

E.4 Nonfatal Heart Attacks

The relationship between short-term particulate matter exposure and heart attacks was quantified in a case-crossover analysis by Peters et al. (2001). The study population was selected from heart attack survivors in a medical clinic. Therefore, the applicable population to apply to the C-R function is all individuals surviving a heart attack in a given year. Several data sources are available to estimate the number of heart attacks per year. For example, several cohort studies have reported estimates of heart attack incidence rates in the specific populations under study. However, these rates depend on the specific characteristics of the populations under study and may not be the best data to extrapolate nationally. The American Heart Association reports approximately 540,000 new heart attacks per year using data from a multi-center study (Haase, 2002, to be published in the American Heart Association's 2003 Statistical Handbook). Exclusion of heart attack deaths reported by CDC Wonder yields approximately 330,000 nonfatal cases per year.

An alternative approach to the estimation of heart attack rates is to use data from the National Hospital Discharge Survey, assuming that all heart attacks that are not instantly fatal will result in a hospitalization. According to the National Hospital Discharge Survey, in 1999 there were approximately 829,000 hospitalizations due to heart attacks (acute myocardial infarction: ICD-9 410) (Popovic, 2001, Table 8). We used regional hospitalization rates over estimates extrapolated from cohort studies because the former is part of a nationally representative survey with a larger sample size, which is intended to

[62] The target universe of the NHAMCS is in-person visits made in the United States to emergency and outpatient departments of non-Federal, short-stay hospitals (hospitals with an average stay of less than 30 days) or those whose specialty is general (medical or surgical) or children's general.

[63] Data are available at ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/

provide reliable national estimates. As additional information is provided regarding the American Heart Association methodology, we will evaluate the usefulness of this estimate of heart attack incidence.

Rosamond et al. (1999) reported that approximately six percent of male and eight percent of female hospitalized heart attack patients die within 28 days (either in or outside of the hospital). We, therefore, applied a factor of 0.93 to the count of hospitalizations to estimate the number of nonfatal heart attacks per year. To estimate the *rate* of nonfatal heart attack, we divided the count by the population estimate for 2000 from the U.S. Census. Exhibit E-4 presents the regional nonfatal heart attack incidence rates.

Exhibit E-4. Nonfatal Heart Attack Rates, by Region and Age Group

Endpoint (ICD codes)	Region	Nonfatal Heart Attack Rate (cases per 100 people per year) [a]		
		0-18	18-64	65+
nonfatal heart attacks (ICD-9 410)	Northeast	0.0000	0.2167	1.6359
	Midwest	0.0003	0.1772	1.4898
	South	0.0006	0.1620	1.1797
	West	0.0000	0.1391	1.1971

[a] Rates are based on data from the 1999 National Hospital Discharge Survey (NHDS) and an estimate from Rosamond et al. (1999) that approximately 7% of individuals hospitalized for a heart attack die within 28 days.

E.5 School Loss Days

Epidemiological studies have examined the relationship between air pollution and a variety of measures of school absence. These measures include: school loss days for all causes, illness-related, and respiratory illness-related. We have two sources of information. The first is the National Center for Education Statistics, which provided an estimate of all-cause school loss days, and the other is the National Health Interview Survey (Adams et al., 1999, Table 47), which has data on different categories of acute school loss days. Exhibit E-5 presents the illness-related rates used in this analysis.

E.5.1 All-Cause School Loss Rates

Based on data from the U.S. Department of Education (1996, Table 42-1), the National Center for Education Statistics estimates that for the 1993-1994 school year, 5.5 percent of students are absent from school on a given day. This estimate is comparable to study-specific estimates from Chen et al. (2000) and Ransom and Pope (1992), which ranged from 4.5 to 5.1 percent.

We use the total or all-cause school absence rate in C-R functions based on studies by Chen et al. (2000), Gilliland et al. (2001) and Ransom et al. (1992). We also use the all-cause school absence rate as a population adjustment in C-R functions derived from Gilliland et al. (2001), for which it is necessary to estimate the average proportion of children attending school on a given day. This is described in more detail in the specific C-R function summaries.

E.5.2 Illness-Related School Loss Rates

The National Health Interview Survey (NHIS) has regional estimates of school loss days due to a variety of acute conditions (Adams et al., 1999). NHIS is a nationwide sample-based survey of the health of the noninstitutionalized, civilian population, conducted by NCHS. The survey collects data on acute conditions, prevalence of chronic conditions, episodes of injury, activity limitations, and self-reported health status. However, it does not provide an estimate of all-cause school loss days.

In estimating illness-related school loss days, we started with school loss days due to acute problems (Adams et al., 1999, Table 47) and subtracted lost days due to injuries, in order to match the definition of the study used in the C-R function to estimate illness-related school absences (Gilliland et al., 2001). We then divided by 180 school days per to estimate *illness*-related school absence rates per school day. Similarly, when estimating respiratory illness-related school loss days, we use data from Adams et al. (1999, Table 47). Note that we estimated 180 school days in a year to calculate respiratory illness-related school absence rates per year.

Exhibit E-5. School Loss Day Rates

Type of School Loss Day [a]	Absence Rate by Region (cases per 100 students per year)			
	Northeast	Midwest	South	West
Respiratory illness-related absences	131.4	165.6	109.8	223.2
Illness-related absences	244.8	262.8	255.6	370.8
All-cause	990.0	990.0	990.0	990.0

[a] We based illness-related school loss day rates on data from the 1996 NHIS (Adams et al., 1999, Table 47) and an estimate of 180 school days per year. This excludes school loss days due to injuries. We based the all-cause school loss day rate on data from the National Center for Education Statistics (U.S. Department of Education, 1996, Table 42-1).

E.6 Other Acute and Chronic Effects

For many of the minor effect studies, baseline rates from a single study are often the only source of information, and we assume that these rates hold for locations in the U.S. The use of study-specific estimates are likely to increase the uncertainty around the estimate because they are often estimated from a single location using a relatively small sample. These endpoints include: acute bronchitis, chronic bronchitis, upper respiratory symptoms, lower respiratory symptoms. Exhibit E-6 presents a summary of these baseline rates.

Exhibit E-6. Selected Acute and Chronic Effects Rates

Endpoint	Age	Parameter [a]	Rate	Source
Acute Bronchitis	8-12	Incidence	4.300	(American Lung Association, 2002a, Table 11)
Chronic Bronchitis	27+	Incidence	0.378	(Abbey et al., 1993, Table 3)
Chronic Bronchitis	18+	Prevalence	4.43%	
	18-44		3.67%	(American Lung Association, 2002b, Table 4)
	45-64		5.05%	
	65+		5.87%	
Lower Respiratory Symptoms (LRS)	7-14	Incidence	43.8	(Schwartz et al., 1994, Table 2)
Minor Restricted Activity Days (MRAD)	18-64	Incidence	780.0	(Ostro and Rothschild, 1989, p. 243)
Work Loss Day (WLD)	18-64	Incidence	217.2	
	18-24		197.1	(Adams et al., 1999, Table 41); (U.S. Bureau of the Census, 1997, No. 22)
	25-44		247.5	
	45-64		179.6	

[a] The incidence rate is the number of cases per 100 people per year. Prevalence refers to the fraction of people that have a particular illness during a particular time period.

E.6.1 Acute Bronchitis

The annual rate of acute bronchitis for children ages 5 to 17 was obtained from the American Lung Association (2002a, Table 11). The authors reported an annual incidence rate per person of 0.043, derived from the 1996 National Health Interview Survey.

E.6.2 Chronic Bronchitis Incidence Rate

The annual incidence rate for chronic bronchitis is estimated from data reported by Abbey et al.(1993, Table 3). The rate is calculated by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), 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)). We then multiplied this result by 100 to calculate an annual incidence rate per 100 people of 0.378.

Age-specific incidence rates are not available. Abbey et al. (1995a, Table 1) did report the incidences by three age groups (25-54, 55-74, and 75+) for “cough type” and “sputum type” bronchitis. However, they did not report an overall incidence rate for bronchitis by age-group. Since, the cough and sputum types of bronchitis overlap to an unknown extent, we did not attempt to generate age-specific incidence rates for the over-all rate of bronchitis.

E.6.3 Chronic Bronchitis Prevalence Rate

We obtained the annual prevalence rate for chronic bronchitis from the American Lung Association (2002b, Table 4). Based on an analysis of 1999 National Health Interview Survey data, they estimated a rate of 0.0443 for persons 18 and older, they also reported the following prevalence rates for people in the age groups 18-44, 45-64, and 65+: 0.0367, 0.0505, and 0.0587, respectively.

E.6.4 Lower Respiratory Symptoms

Lower respiratory symptoms (LRS) are defined as two or more of the following: cough, chest pain, phlegm, wheeze. The proposed yearly incidence rate for 100 people, 43.8, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). The authors did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated per person per day values are 10th = 0 percent, 25th = 0 percent, 50th = 0 percent, 75th = 0.29 percent, and 90th = 0.34 percent. The most conservative estimate consistent with the data are to assume the incidence per person per day is zero up to the 75th percentile, a constant 0.29 percent between the 75th and 90th percentiles, and a constant 0.34 percent between the 90th and 100th percentiles. Alternatively, assuming a linear slope between the 50th and 75th, 75th and 90th, and 90th to 100th percentiles, the estimated mean incidence rate per person per day is 0.12 percent.[64] We used the latter approach in this analysis, and then multiplied by 100 and by 365 to calculate the incidence rate per 100 people per year.

E.6.5 Minor Restricted Activity Days (MRAD)

Ostro and Rothschild (1989, p. 243) provide an estimate of the annual incidence rate of MRADs (7.8). We multiplied this estimate by 100 to get an annual rate per 100 people.

E.6.6 Work Loss Days

The yearly work-loss-day incidence rate per 100 people is based on estimates from the 1996 National Health Interview Survey (Adams et al., 1999, Table 41). They reported a total annual work loss days of 352 million for individuals ages 18 to 65. The total population of individuals of this age group in 1996 (162 million) was obtained from (U.S. Bureau of the Census, 1997, No. 22). The average annual rate of work loss days per individual (2.17) was multiplied by 100 to obtain the average yearly work-loss-day rate of 217 per 100 people. Using a similar approach, we calculated work-loss-day rates for ages 18-24, 25-44, and 45-64, respectively.

E.7 Asthma-Related Health Effects

Several studies have examined the impact of air pollution on asthma development or exacerbation. Many of the baseline incidence rates used in the C-R functions are based on study-specific estimates. The baseline rates for the various endpoints are described below and summarized in Exhibit E-7.

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[64] For example, the 62.5^{th} percentile would have an estimated incidence rate per person per day of 0.145 percent.

Exhibit E-7. Asthma-Related Health Effects Rates

Endpoint	Age	Parameter [a]	Rate	Source
Acute Bronchitis	9-15	Incidence	32.6	(McConnell et al., 1999, Table 2)
Asthma Attacks	18+	Incidence	2008	1999 National Health Interview Survey
Asthma Exacerbation, Shortness of Breath, African American	8-13	Incidence	1351	(Ostro et al., 2001, p.202)
	8-13	Prevalence	7.40%	
Asthma Exacerbation, Wheeze, African American	8-13	Incidence	2774	(Ostro et al., 2001, p.202)
	8-13	Prevalence	17.30%	
Asthma Exacerbation, Cough, African American	8-13	Incidence	2446	(Ostro et al., 2001, p.202)
	8-13	Prevalence	14.50%	
Asthma Exacerbation, Cough	6-13	Incidence	3139	(Vedal et al., 1998, Table 1 p. 1038)
Asthma Exacerbation, One or more symptoms	5-13	Incidence	21900	(Yu et al., 2000, Table 2 p. 1212)
Chronic Asthma, Male	27+	Incidence	0.219	(McDonnell et al., 1999, Table 4)
Phlegm	9-15	Incidence	25.7	(McConnell et al., 1999, Table 2)
Upper Respiratory Symptoms (URS)[2]	9-11	Incidence	12479	(Pope et al., 1991, Table 2)

[a] The incidence rate is the number of cases per 100 people per year. Prevalence refers to the fraction of people that have a particular illness during a particular time period.

E.7.1 Asthma Attacks

The annual rate of asthma attacks among asthmatics is estimated from the 1999 National Health Interview Survey. Individuals with asthma were asked about the number of wheezing attacks per year. The average number of wheezing attacks per year was multiplied by 100 to obtain a wheezing attack rate per year per 100 people for individuals 18 and older. We assume that this rate of wheezing attacks can be used as a surrogate for asthma attacks.

Note that the same survey examined wheezing attacks for children. However, the number of wheezing attacks per year were censored at 12 (compared to censoring at 95 for adults). Due to the potential for underestimation of the number of children's wheezing attacks, we used the adult rate for all individuals.

E.7.2 Asthma Exacerbation

There are a variety of types of symptoms for asthma exacerbation. We calculated rates for shortness of breath, wheeze, cough, and other asthma related effects.

E.7.3 Shortness of Breath

To estimate the annual rate of new shortness of breath episodes among African-American asthmatics, ages 8-13, we used the rate reported by Ostro et al. (2001, p.202). We estimated the daily prevalence of shortness of breath episodes among African-American asthmatics, ages 8-13, by taking a weighted average of the reported rates in Ostro et al. (2001, p.202).

E.7.4 Wheeze

The daily rate of new wheeze episodes among African-American asthmatics, ages 8-13, is reported by Ostro et al. (2001, p.202) as 0.076. We multiplied this value by 100 and by 365 to get the annual incidence rate per 100 people. The daily rate of prevalent wheeze episodes (0.173) among African-American asthmatics, ages 8-13, is estimated by taking a weighted average of the reported rates in Ostro et al. (2001, p.202).

E.7.5 Cough

The daily rate of new cough episodes among African-American asthmatics, ages 8-13, is reported by Ostro et al. (2001, p.202) as 0.067. We multiplied this value by 100 and by 365 to get the annual incidence rate per 100 people. The daily rate of prevalent cough episodes (0.145) among African-American asthmatics, ages 8-13, is estimated by taking a weighted average of the reported rates in Ostro et al. (2001, p.202).

E.7.6 One or More Symptoms

Yu et al. (2000, Table 2, p. 1212) reported a daily rate of at least one asthma episode per asthmatic child ages 5-13. An asthma episode is defined as at least one of the following asthma symptoms: wheezing, coughing, chest tightness, or shortness of breath.

E.7.7 Chronic Asthma

We derived the annual incidence rate per 100 people by taking the number of new cases (32), dividing by the number of individuals in the sample (972), as reported by (McDonnell et al., 1999, Table 4), and then dividing by the 15 years in the sample. We then multiplied by 100 to get the annual incidence rate per 100 people.

E.7.8 Upper Respiratory Symptoms

Upper Respiratory Symptoms are defined as one or more of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes. Using the incidence rates for upper respiratory symptoms among asthmatics, published in Pope et al. (1991, Table 2), we calculated a sample size-weighted average incidence rate.

E.7.9 Asthma Population Estimates

In studies examining the association between air pollution and the development or exacerbation of asthma, often times an estimate of the percent of the population with asthma is required. Asthma percentages were obtained either directly from the National Health Interview Survey (NHIS) or an American Lung Association (2002c) report summarizing data from NHIS. Exhibit E-8 presents asthma prevalence rates used define asthmatic populations in the C-R functions.

Exhibit E-8. Asthma Prevalence Rates Used to Estimate Asthmatic Populations

Population Group	Prevalence	Source
All Ages	3.86%	American Lung Association (2002c, Table 7) [a]
<18	5.27%	
5-17	5.67%	
18-44	3.71%	
45-64	3.33%	
65+	2.21%	
African-American, 5 to 17	7.26%	American Lung Association (2002c, Table 9) [a]
African-American, <18	7.35%	
Male, 27+	2.10%	2000 NHIS public use data files [b]

[a] The work by the American Lung Association is based on the 1999 National Health Interview Survey.

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

Appendix F: Particulate Matter Concentration-Response Functions

In this Appendix, we present the concentration-response (C-R) functions used to estimate PM-related adverse health effects. Each sub-section has an Exhibit with a brief description of the C-R function and the underlying parameters. Following each Exhibit, we present a brief summary of each of the studies and any items that are unique to the study.

Note that the main text describes the methods that we used to choose these C-R functions from the wide range available in the literature. In addition, Appendix D mathematically derives the standard types of C-R functions that we encountered in the epidemiological literature, such as, log-linear, logistic and linear, so we simply note here the type of functional form. Finally, Appendix E presents a description of the sources for the incidence and prevalence data used in these C-R functions.

Exhibit F-1. Concentration-Response (C-R) Functions for Particulate Matter and Long-Term Mortality

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time	Beta	Std Error	Functional Form	Notes
All Cause	$PM_{2.5}$	Krewski et al.	2000	63 cities	30+	All	All	None	Annual Avg	0.004626	0.001205	Log-linear	ACS reanalysis
All Cause	$PM_{2.5}$	Krewski et al.	2000	50 cities	30+	All	All	None	Annual Median	0.005348	0.001464	Log-linear	ACS reanalysis
All Cause	$PM_{2.5}$	Krewski et al.	2000	nationwide	30+	All	All	None	Annual Median	0.010394	0.002902	Log-linear	ACS reanalysis; RE Ind Cities
All Cause	$PM_{2.5}$	Krewski et al.	2000	nationwide	30+	All	All	None	Annual Median	0.006058	0.003383	Log-linear	ACS reanalysis; RE Reg Adj
All Cause	$PM_{2.5}$	Krewski et al.	2000	6 cities	25+	All	All	None	Annual Avg	0.013272	0.004070	Log-linear	Six Cities reanalysis
All Cause	$PM_{2.5}$	Pope et al.	1995	50 cities	30+	All	All	None	Annual Median	0.006408	0.001509	Log-linear	
All Cause	$PM_{2.5}$	Dockery et al.	1993	6 cities	25+	All	All	None	Annual Avg	0.012425	0.004228	Log-linear	
All Cause	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.004018	0.001642	Log-linear	'79-'83 exposure
Cardiopulmonary	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.005733	0.002167	Log-linear	'79-'83 exposure
Lung Cancer	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.007881	0.003463	Log-linear	'79-'83 exposure
Infant	PM_{10}	Woodruff et al.	1997	86 cities	<1	All	All	None	Annual Avg	0.003922	0.001221	Logistic	

F.1 Long-term 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.

F.1.1 Mortality - Mean, All Cause (Krewski et al., 2000) - Reanalysis of Pope et al. (1995)

The Krewski et al. (2000) reanalysis of Pope et al. (1995) used a Cox proportional hazard model to estimate the impact of long-term PM exposure. The original investigation followed 295,223 individuals[65] ages 30 and over in 50 cities from September 1, 1982 to December 31, 1989, and related their survival to median $PM_{2.5}$ concentrations for 1979 to 1983. Krewski et al. (2000) independently estimated city-specific annual *mean* values from EPA's Inhalable Particle Monitoring Network (IPMN) for the same years (1979-1983). Krewski et al. (2000) followed Pope et al. (1995, Table 2) and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths,[66] and found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. Krewski et al. included only PM, so it is unclear to what extent it may be including the impacts of ozone or other gaseous pollutants.

Pope et al. (1995) is the better of the two published prospective cohort studies: it has a larger population and includes more cities than the prospective cohort study by Dockery et al. (1993). Pope et al.'s study has several further advantages. The population followed in this study was largely Caucasian and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in race, socioeconomic status, or related factors. In addition, the PM coefficient in Pope et al. is likely to be biased downward, counteracting a possible upward bias associated with historical air quality trends discussed earlier. One source of this downward bias is the generally healthier and study population, in comparison to poorer minority populations. Krewski et al. (2000, Part II - Table 52) found that educational status was a strong effect modifier of the PM - mortality relationship in both studies, with the strongest effect seen among the less educated. In fact, much of the differences in magnitude of effect between the studies was made up when assessing risk across comparable levels of educational attainment.

Another source of downward bias is that intercity movement of cohort members was not considered in the original study and therefore could not be evaluated in the reanalysis. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, that are proxied by differences in city-specific annual average PM levels, will be exaggerated, and will result in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

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[65] The total study population was 552,138 in 151 cities, however, only 295,223 individuals resided in 50 cities with fine particle data.

[66] All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.12) and 95% confidence interval (1.06-1.19) associated with a change in *annual mean* $PM_{2.5}$ exposure of 24.5 μg/m^3 (based on the range from the original ACS study) (Krewski et al., 2000, Part II - Table 31).

Functional Form: Log-linear
Coefficient: 0.004626
Standard Error: 0.001205
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

F.1.2 Mortality - Median, All Cause (Krewski et al., 2000) - Reanalysis of Pope et al. (1995)

Krewski et al. (2000) performed an analysis of Pope et al. (2000) using independently estimated city-specific annual *median* values as well. Fine particle estimates were obtained from EPA's Inhalable Particle Monitoring Network (IPMN) for the years 1979-1983 for the same 50 cities. Overall, the estimates showed good agreement with the median values used in the original investigation with one exception. The median fine particle concentration for Denver dropped from 16.1 to 7.8 μg/m^3, resulting in a larger range between the least and most polluted cities and a reduced relative risk. Since the original estimate could not be audited, Denver is included in the subsequent C-R function as there is no reason to believe that the monitoring data is invalid.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.14) and 95% confidence interval (1.06-1.22) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 μg/m^3 (based on the range from the original ACS study) (Krewski et al., 2000, Part II - Table 31).

Functional Form: Log-linear
Coefficient: 0.005348
Standard Error: 0.001464
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

F.1.3 Mortality - Median, Random Effects with Regional Adjustment (Krewski et al., 2000) - Reanalysis of Pope et al. (1995)

Krewski et al. (2000) also performed an analysis of Pope et al. (2000) using a random effects model to estimate a regionally-adjusted relative risk. The authors used an indicator variable representing seven regions of the U.S. The regionally-adjusted estimate was comparable with the results from the standard Cox Proportional Hazards Model, which assumes that all observations are statistically independent.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.16) and 95% confidence interval (0.99-1.37) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 μg/m^3 (based on the range from the original ACS study) (Krewski et al., 2000, Part II - Table 46).

Functional Form: Log-linear
Coefficient: 0.006058
Standard Error: 0.003383
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

F.1.4 Mortality - Median, Random Effects with Independent Cities (Krewski et al., 2000) - Reanalysis of Pope et al. (1995)

Krewski et al. (2000) also performed an analysis of Pope et al. (2000) using a random effects approach to estimate an independent cities model. This approach incorporates between-city variation into second-stage modeling weights, thereby avoiding the assumption of independent observations. However, potential regional patterns in mortality may be overlooked, because the approach assumes that city-specific mortality rates are statistically independent. The independent cities estimate is considerably larger than the standard Cox Proportional Hazards Model, which assumes that all observations are statistically independent.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.29) and 95% confidence interval (1.12-1.48) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) (Krewski et al., 2000, Part II - Table 46).

Functional Form: Log-linear
Coefficient: 0.010394
Standard Error: 0.002902
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

F.1.5 Mortality (Krewski et al., 2000) - Reanalysis of Dockery et al. (1993)

Krewski et al. (2000) performed a validation and replication analysis of Dockery et al. (1993). The original investigators 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; a higher proportion without a high school education), and a follow-up period nearly twice as long as that of Pope et al. (1995). Krewski et al. (2000, Part II - Table 52) found that educational status was a strong effect modifier of the PM - mortality relationship in both studies, with the strongest effect seen among the less educated. 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.

After an audit of the air pollution data, demographic variables, and cohort selection process, Krewski et al. (2000) noted that a small portion of study participants were mistakenly censored early. The following C-R function is based on the risk estimate from the audited data, with the inclusion of those person-years mistakenly censored early.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.28) and 95% confidence interval (1.10-1.48) associated with a change in *annual mean* $PM_{2.5}$ exposure of 18.6 μg/m^3 to 29.6 μg/m^3 (Krewski et al., 2000, Part I - Table 19c).

Functional Form: Log-linear
Coefficient: 0.013272
Standard Error: 0.004070
Incidence Rate: county-specific annual all cause mortality rate per person ages 25 and older
Population: population of ages 25 and older

F.1.6 Mortality, All Cause (Pope et al., 1995)

Pope et al. (1995) used a Cox proportional hazard model to estimate the impact of long-term PM exposure. They followed 295,223 individuals[67] ages 30 and over in 50 cities from September 1, 1982 to December 31, 1989, and related their survival to median $PM_{2.5}$ concentrations for 1979 to 1983. Pope et al. (1995, Table 2) reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths,[68] and found that median $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. Pope et al. included only PM, so it is unclear to what extent it may be including the impacts of ozone or other gaseous pollutants.

Pope et al. (1995) is the better of the two published prospective cohort studies: it has a larger population and includes more cities than the prospective cohort study by Dockery et al. (1993). Pope et al.'s study has several further advantages. The population followed in this study was largely Caucasian and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in race, socioeconomic status, or related factors. In addition, the PM coefficient in Pope et al. is likely to be biased downward, counteracting a possible upward bias associated with historical air quality trends discussed earlier. One source of this downward bias is the generally healthier study population, in comparison to poorer minority populations. Another source of downward bias is that intercity movement of cohort members was not considered in this study. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, that are proxied by differences in city-specific annual average PM levels, will be exaggerated, and will result in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.17) and 95% confidence interval (1.09-1.26) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 μg/m^3 (Pope et al., 1995, Table 2).

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[67] The total study population was 552,138 in 151 cities, however, only 295,223 individuals resided in 50 cities with fine particle data.

[68] All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

Functional Form: Log-linear
Coefficient: 0.006408
Standard Error: 0.001509
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

F.1.7 Mortality, All Cause (Dockery et al., 1993)

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.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.26) and 95% confidence interval associated (1.08-1.47) with a change in *annual mean* $PM_{2.5}$ exposure of 18.6 $\mu g/m^3$ (Dockery et al., 1993, Tables 1 and 5).

Functional Form: Log-linear
Coefficient: 0.012425
Standard Error: 0.004228
Incidence Rate: county-specific annual all cause mortality rate per person ages 25 and older
Population: population of ages 25 and older

F.1.8 Mortality, All Cause (Pope et al., 2002) - Based on ACS Cohort: Mean $PM_{2.5}$

The Pope et al. (2002) analysis is a longitudinal cohort tracking study that uses the same American Cancer Society (ACS) cohort as the original Pope et al. (1995) study, and the Krewski et al. (2000) reanalysis. Pope et al. (2002) analyzed survival data for the cohort from 1982 through 1998, 9 years longer than the original Pope study. Pope et al. (2002) also obtained $PM_{2.5}$ data in 116 metropolitan areas collected in 1999, and the first three quarters of 2000. This is more metropolitan areas with $PM_{2.5}$ data than was available in the Krewski reanalysis (61 areas), or the original Pope study (50 areas), providing a larger size cohort.

They used a Cox proportional hazard model to estimate the impact of long-term PM exposure using three alternative measures of $PM_{2.5}$ exposure; metropolitan area-wide annual mean PM levels from the beginning of tracking period ('79-'83 PM data, conducted for 61 metropolitan areas with 359,000 individuals), annual mean PM from the end of the tracking period ('99-'00, for 116 areas with 500,000 individuals), and the average annual mean PM levels of the two periods (for 51 metropolitan areas, with 319,000 individuals). PM levels were lower in '99-00 than in '79 - '83 in most cities, with the largest improvements occurring in cities with the highest original levels.

Pope et al. (2002) followed Krewski et al. (2000) and Pope et al. (1995, Table 2) and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and

460-519), and "all other" deaths.[69] Like the earlier studies, Pope et al. (2002) found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. (2002) found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with "all other" deaths.

Pope et al. (2002) obtained ambient data on gaseous pollutants routinely monitored by EPA during the 1982-1998 observation period, including SO_2, NO_2, CO, and ozone. They did not find significant relationships between NO_2, CO, and ozone and premature mortality, but there were significant relationships between SO_2, and all-cause, cardiopulmonary, lung cancer and "all other" mortality.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.041) and 95% confidence interval (1.008-1.075) associated with a change in *annual mean* exposure of 10.0 $\mu g/m^3$. Pope et al. (2002, Table 2).[70]

Functional Form: Log-linear
Coefficient: 0.004018
Standard Error: 0.001642
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

F.1.9 Mortality, Cardiopulmonary (Pope et al., 2002) - Based on ACS Cohort: Mean $PM_{2.5}$

Pope et al. (2002) followed Krewski et al. (2000) and Pope et al. (1995, Table 2) and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths.[71] Like the earlier studies, Pope et al. (2002) found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. (2002) found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with "all other" deaths.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.059) and 95% confidence interval (1.015-1.105) associated with a change in *annual mean* exposure of 10.0 $\mu g/m^3$. Pope et al. (2002, Table 2).[72]

Functional Form: Log-linear

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[69] All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

[70] Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

[71] All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

[72] Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

Coefficient: 0.005733
Standard Error: 0.002167
Incidence Rate: county-specific annual cardiopulmonary mortality rate (ICD codes 401-440, 460-519) per person ages 30 and older
Population: population of ages 30 and older

F.1.10 Mortality, Lung Cancer (Pope et al., 2002) - Based on ACS Cohort: Mean $PM_{2.5}$

Pope et al. (2002) followed Krewski et al. (2000) and Pope et al. (1995, Table 2) and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths.[73] Like the earlier studies, Pope et al. (2002) found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. (2002) found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with "all other" deaths.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.082) and 95% confidence interval (1.011-1.158) associated with a change in *annual mean* exposure of 10.0 $\mu g/m^3$. Pope et al. (2002, Table 2).[74]

Functional Form: Log-linear
Coefficient: 0.007881
Standard Error: 0.003463
Incidence Rate: county-specific annual lung cancer mortality rate (ICD code 162) per person ages 30 and older
Population: population of ages 30 and older

F.1.11 Infant Mortality (Woodruff et al., 1997)

In a study of four million infants in 86 U.S. metropolitan areas conducted from 1989 to 1991, Woodruff et al. (1997) found a significant link between PM_{10} exposure in the first two months of an infant's life with the probability of dying between the ages of 28 days and 364 days. PM_{10} exposure was significant for all-cause mortality. PM_{10} was also significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants.

In addition to the work by Woodruff et al., work in Mexico City (Loomis et al., 1999), the Czech Republic (Bobak and Leon, 1992), Sao Paulo (Saldiva et al., 1994; Pereira et al., 1998), and Beijing (Wang et al., 1997) provides additional evidence that particulate levels are significantly related to infant or child mortality, low birth weight or intrauterine mortality.

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[73] All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

[74] Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

Conceptually, neonatal or child mortality could be added to the premature mortality predicted by Pope et al. (1995), because the Pope function covers only the population over 30 years old.[75] However, the EPA Science Advisory Board recently advised the Agency not to include post-neonatal mortality in this analysis because the study is of a new endpoint and the results have not been replicated in other studies (U.S. EPA, 1999a, p. 12). The estimated avoided incidences of neonatal mortality are estimated and presented as a sensitivity analysis, and are not included in the base analysis.

Single Pollutant Model

The coefficient and standard error are based on the odds ratio (1.04) and 95% confidence interval (1.02-1.07) associated with a 10 $\mu g/m^3$ change in PM_{10} (Woodruff et al., 1997, Table 3).

Functional Form: Logistic
Coefficient: 0.003922
Standard Error: 0.001221
Incidence Rate: county-specific annual postneonatal[76] infant deaths per infant under the age of one
Population: population of infants under one year old

[75] Predicted neonatal mortality could not be added to the premature mortality predicted by the daily (short-term exposure) mortality studies, however, because these studies cover all ages.

[76] Post-neonatal refers to infants that are 28 days to 364 days old.

Exhibit F-2. Concentration-Response (C-R) Functions for Particulate Matter and Short-Term Mortality

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form	Notes
Non-Accidental	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.001433	0.00013	Log-linear	
Non-Accidental	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.002835	--	Log-linear	Lag Adjusted[2]
Chronic Lung	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.006423	--	Log-linear	Lag Adjusted[2]

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.
2. Refer to the study summaries below for a discussion of the lag adjustment used for these functions.

F.2 Short-term Mortality

Short-term mortality studies are those that typically link daily air pollution levels with daily changes in mortality rates.

F.2.1 Short-Term Mortality, Non-Accidental (Schwartz et al., 1996)

Schwartz et al. (1996) pooled the results from six cities in the U.S. and found a significant relationship between daily $PM_{2.5}$ concentration and non-accidental mortality.[77] Abt Associates Inc. (1996b, p. 52) used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to reestimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error (Abt Associates Inc., 1996a, p. 65).

Single Pollutant Model

Abt Associates Inc. (1996b, p. 52) used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient (Abt Associates Inc., 1996a, Exhibit 7.2) and its standard error (Abt Associates Inc., 1996a, Exhibit 7.2).

Functional Form: Log-linear
Coefficient: 0.001433
Standard Error: 0.000129
Incidence Rate: county-specific annual daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

F.2.2 Short-Term Mortality, Non-Accidental - Lag Adjusted (Schwartz et al., 1996)

Schwartz et al. (1996) pooled the results from six cities in the U.S. and found a significant relationship between daily $PM_{2.5}$ concentration and non-accidental mortality.[78] Abt Associates Inc. (1996b, p. 52) used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to reestimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error (Abt Associates Inc., 1996a, p. 65). In order to estimate the impact of daily $PM_{2.5}$ levels on daily mortality if a distributed lag model had been fit, the $PM_{2.5}$ coefficient is adjusted as described below.

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[77] Schwartz et al. (1996, p. 929) defined non-accidental mortality as all-cause mortality less deaths due to accidents and other external causes (ICD-9 codes: 800-999). Other external causes includes suicide, homicide, and legal intervention (National Center for Health Statistics, 1994).

[78] Schwartz et al. (1996, p. 929) defined non-accidental mortality as all-cause mortality less deaths due to accidents and other external causes (ICD-9 codes: 800-999). Other external causes includes suicide, homicide, and legal intervention (National Center for Health Statistics, 1994).

Recent studies have found that an increase in PM levels on a given day can elevate mortality for several days following the exposure (Samet et al., 2000; Schwartz, 2000b). These studies have reported the results of distributed lag models for the relationship between PM_{10} and daily mortality. Schwartz (2000b) examined the relationship between PM_{10} and daily mortality and reported results both for a single day lag model and an unconstrained distributed lag model. The unconstrained distributed lag model coefficient estimate is 0.0012818 and the single-lag model coefficient estimate is 0.0006479. A distributed lag adjustment factor can be constructed as the ratio of the estimated coefficient from the unconstrained distributed lag model to the estimated coefficient from the single-lag model reported in Schwartz (2000). The ratio of these estimates is 1.9784. In order to estimate the full impact of daily PM levels on daily mortality, we applied this ratio to the coefficient obtained from Schwartz et al. (1996) for the association between $PM_{2.5}$ and daily mortality.

In applying the ratio derived from a PM_{10} study to $PM_{2.5}$, we assume that the same relationship between the distributed lag and single day estimates would hold for $PM_{2.5}$. Effect estimates for the PM_{10}-daily mortality relationship tend to be lower in magnitude than for $PM_{2.5}$, because fine particles are believed to be more closely associated with mortality than the coarse fraction of PM. If most of the increase in mortality is expected to be associated with the fine fraction of PM_{10}, then it is reasonable to assume that the same proportional increase in risk would be observed if a distributed lag model were applied to the $PM_{2.5}$ data.

Single Pollutant Model

The distributed lag model coefficient is estimated by applying the distributed lag adjustment factor of 1.9784 to the pooled $PM_{2.5}$ coefficient (0.001433) estimated by Abt Associates Inc. (1996a, Exhibit 7.2) from the six $PM_{2.5}$ relative risks reported by Schwartz et al. (1996).[79]

Functional Form: Log-linear
Coefficient: 0.002835
Incidence Rate: county-specific annual daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

F.2.3 Short-Term Mortality, Chronic Lung Disease - Lag Adjusted (Schwartz et al., 1996)

Schwartz et al. (1996) evaluated the relationship between daily $PM_{2.5}$ levels and short-term mortality in six U.S. cities. Schwartz pooled results across the six cities and found statistically significant associations between daily $PM_{2.5}$ levels and non-accidental mortality (ICD codes <800), along with mortality for ischemic heart disease (ICD codes 410-414), COPD (ICD codes 490-496), and pneumonia (ICD codes 480-486). A smaller association was found for PM_{10} and no significant associations were reported for $PM_{10-2.5}$. The C-R function for chronic lung disease mortality is based on the results of a single pollutant model using a two-day average of $PM_{2.5}$ (Schwartz et al., 1996, Table 7). In order to estimate the impact of daily $PM_{2.5}$ levels on daily mortality if a distributed lag model had been fit, the $PM_{2.5}$ coefficient is adjusted as described below.

Recent studies have found that an increase in PM levels on a given day can elevate mortality for several days following the exposure (Samet et al., 2000; Schwartz, 2000b). These studies have reported the results of distributed lag models for the relationship between PM_{10} and daily mortality. Schwartz

[79] The distributed lag adjustment C-R function is only run for the point estimate. The standard error of this coefficient has not been estimated.

(2000b) examined the relationship between PM_{10} and daily mortality and reported results both for a single day lag model and an unconstrained distributed lag model. The unconstrained distributed lag model coefficient estimate is 0.0012818 and the single-lag model coefficient estimate is 0.0006479. A distributed lag adjustment factor can be constructed as the ratio of the estimated coefficient from the unconstrained distributed lag model to the estimated coefficient from the single-lag model reported in Schwartz (2000). The ratio of these estimates is 1.9784. In order to estimate the full impact of daily PM levels on daily mortality, we applied this ratio to the coefficient obtained from Schwartz et al. (1996) for the association between $PM_{2.5}$ and daily mortality.

In applying the ratio derived from a PM_{10} study to $PM_{2.5}$, we assume that the same relationship between the distributed lag and single day estimates would hold for $PM_{2.5}$. Effect estimates for the PM_{10}-daily mortality relationship tend to be lower in magnitude than for $PM_{2.5}$, because fine particles are believed to be more closely associated with mortality than the coarse fraction of PM. If most of the increase in mortality is expected to be associated with the fine fraction of PM_{10}, then it is reasonable to assume that the same proportional increase in risk would be observed if a distributed lag model were applied to the $PM_{2.5}$ data.

Single Pollutant Model

The $PM_{2.5}$ coefficient is based on a reported 3.3% increase in COPD mortality associated with a 10 $\mu g/m^3$ change in two-day average $PM_{2.5}$ levels (Schwartz et al., 1996, Table 7). This coefficient (0.003247) is then multiplied by the distributed lag adjustment factor of 1.9784 to estimate a distributed lag model coefficient.

Functional Form: Log-linear
Coefficient: 0.006423
Incidence Rate: county-specific annual daily chronic lung disease mortality rate (ICD codes 490-496)
Population: population of all ages

Exhibit F-3. Concentration-Response (C-R) Functions for Particulate Matter and Chronic Illness

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time	Beta	Std Error	Functional Form
Chronic Bronchitis	$PM_{2.5}$	Abbey et al.	1995	SF, SD, South Coast Air Basin	27+	All	All	None	Annual Avg	0.0137	0.00680	Logistic
Chronic Bronchitis	PM_{10}	Schwartz	1993	53 cities	30+	All	All	None	Annual Avg	0.0123	0.00434	Logistic

F.3 Chronic Illness

Schwartz (1993) and Abbey et al. (1993; 1995c) provide evidence that PM exposure over a number of years gives rise to the development of chronic bronchitis in the U.S., and a recent study by McDonnell et al. (1999) provides evidence that ozone exposure is linked to the development of asthma in adults. 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).[80]

F.3.1 Chronic Bronchitis (Abbey et al., 1995c, California)

Abbey et al. (1995c) 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 is based on the results of the single pollutant model presented in Table 2.

Single Pollutant Model

The estimated coefficient (0.0137) is presented for a one $\mu g/m^3$ change in $PM_{2.5}$ (Abbey et al., 1995c, Table 2). The standard error is calculated from the reported relative risk (1.81) and 95% confidence interval (0.98-3.25) for a 45 $\mu g/m^3$ change in $PM_{2.5}$ (Abbey et al., 1995c, Table 2).

Functional Form: Logistic
Coefficient: 0.0137
Standard Error: 0.00680
Incidence Rate: annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378
Population: population of ages 27 and older[81] without chronic bronchitis = 95.57%[82] of population 27+

F.3.2 Chronic Bronchitis (Schwartz, 1993)

Schwartz (1993) examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1974 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz (1993, Table 3) reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the

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[80] There are a limited number of studies that have estimated the impact of air pollution on chronic bronchitis. An important hindrance is the lack of health data and the associated air pollution levels over a number of years.

[81] Using the same data set, Abbey et al. (1995a, p. 140) reported that the respondents in 1977 ranged in age from 27 to 95.

[82] The American Lung Association (2002b, Table 4) reports a chronic bronchitis prevalence rate for ages 18 and over of 4.43% (American Lung Association, 2002b, Table 4).

prevalence of reported chronic bronchitis, asthma, shortness of breath (dyspnea) and respiratory illness[83], and the annual levels of TSP, collected in the year prior to the survey (TSP was the only pollutant examined in this study). TSP was significantly related to the prevalence of chronic bronchitis, and marginally significant for respiratory illness. No effect was found for asthma or dyspnea. The C-R function for PM_{10} is estimated from the results of the single pollutant model reported for TSP.

Single Pollutant Model

The estimated coefficient is based on the odds ratio (1.07) associated with 10 $\mu g/m^3$ change in TSP (Schwartz, 1993, p. 9). Assuming that PM_{10} is 55 percent of TSP[84] and that particulates greater than ten micrometers are harmless, the coefficient is calculated by dividing the TSP coefficient by 0.55. The standard error for the coefficient is calculated from the 95% confidence interval for the odds ratio (1.02 to 1.12) (Schwartz, 1993, p. 9).

Schwartz (1993) examined the *prevalence* of chronic bronchitis, not its *incidence*. To use Schwartz's study and still estimate the change in incidence, there are at least two possible approaches. The first is to simply assume that it is appropriate to use the baseline *incidence* of chronic bronchitis in a C-R function with the estimated coefficient from Schwartz's study, to directly estimate the change in incidence. The second is to estimate the percentage change in the prevalence rate for chronic bronchitis using the estimated coefficient from Schwartz's study in a C-R function, and then to assume that this percentage change applies to a baseline incidence rate obtained from another source. (That is, if the prevalence declines by 25 percent with a drop in PM, then baseline incidence drops by 25 percent with the same drop in PM.) This analysis is using the latter approach, and estimates a percentage change in prevalence which is then applied to a baseline incidence rate. The scaling factor used in the C-R function is the ratio of chronic bronchitis incidence rate (estimated from Abbey et al. (1993)) to chronic bronchitis prevalence rate (estimated from American Lung Association (2002b, Table 4)).

Functional Form: Logistic
Coefficient: 0.0123
Standard Error: 0.00434
Incidence Rate: annual chronic bronchitis prevalence rate per person (American Lung Association, 2002b, Table 4) = 0.0443
Population: population of ages 30 and older without chronic bronchitis = 95.57%[85] of population 30+
Adjustment Factor: ratio of chronic bronchitis incidence to chronic bronchitis prevalence = 0.00378/0.0443 = 0.085 (Abbey et al., 1993, Table 3; American Lung Association, 2002b, Table 4)

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[83] Respiratory illness defined as a significant condition, coded by an examining physician as ICD-8 code 460-519.

[84] The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).

[85] The American Lung Association (2002b, Table 4) reports a chronic bronchitis prevalence rate for ages 18 and over of 4.43% (American Lung Association, 2002b, Table 4).

Exhibit F-4. Concentration-Response (C-R) Functions for Particulate Matter and Hospital Admissions

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Asthma	$PM_{2.5}$	Sheppard et al.	1999	Seattle, WA	<65	All	All	CO	24-hr avg	0.002505	0.001045	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.001089	0.002420	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	65+	All	All	CO	24-hr avg	0.0008	0.001000	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	18-64	All	All	CO	24-hr avg	0.0020	0.000909	Log-linear
Chronic Lung Disease (less Asthma)	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.002839	0.001351	Log-linear
Pneumonia	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.004480	0.001918	Log-linear
Pneumonia	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.002049	0.000570	Log-linear
All Cardiovascular	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	65+	All	All	CO	24-hr avg	0.0005	0.000556	Log-linear
All Cardiovascular	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	18-64	All	All	CO	24-hr avg	0.0009	0.000500	Log-linear
All Cardiovascular	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.001183	0.000111	Log-linear
Dysrhythmia	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.002138	0.002525	Log-linear
Heart Failure	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.004668	0.001650	Log-linear
Ischemic Heart Disease	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.001116	0.001339	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

F.4 Hospitalizations

F.4.1 Hospital Admissions for Asthma (Sheppard et al., 1999, Seattle)

Sheppard et al. (1999) studied the relation between air pollution in Seattle and nonelderly (<65) hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM10_{10-2.5}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects.[86] They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, CO, and ozone. They did not observe an association for SO_2. They found PM and CO to be jointly associated with asthma admissions. The best fitting co-pollutant models were found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. For the remaining pollutants, the best fitting models included $PM_{2.5}$ and CO. Results for other co-pollutant models were not reported. The $PM_{2.5}$ C-R function is based on the multipollutant model.

Multipollutant Model ($PM_{2.5}$ and CO)

The coefficient and standard error for the co-pollutant model with CO are calculated from a relative risk of 1.03 (95% CI 1.01-1.06) for an 11.8 $\mu g/m^3$ increase[87] in $PM_{2.5}$ (Sheppard et al., 1999, p. 28).

Functional Form: Log-linear
Coefficient: 0.002505
Standard Error: 0.001045
Incidence Rate: region-specific daily hospital admission rate for asthma admissions per person <65 (ICD code 493)
Population: population of ages 65 and under

F.4.2 Hospital Admissions for Chronic Lung Disease (Lippmann et al., 2000, Detroit)

Lippmann et al. (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on results of the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

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[86] $PM_{2.5}$ levels were estimated from light scattering data.

[87] The reported IQR change in the abstract and text is smaller than reported in Table 3. We assume the change reported in the abstract and text to be correct because greater number of significant figures are reported.

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.040 (95% CI 0.877-1.234) for a 36 μg/m^3 increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 26).

Functional Form: Log-linear
Coefficient: 0.001089
Standard Error: 0.002420
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 65+ (ICD codes 490-496)
Population: population of ages 65 and older

F.4.3 Hospital Admissions for Chronic Lung Disease (Moolgavkar, 2000b)

Moolgavkar (2000b) examined the association between air pollution and COPD hospital admissions (ICD 490-496) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO_2, NO_2, CO, and PM_{10} in all three areas. $PM_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group in Chicago and Phoenix, weak associations were observed between the gaseous pollutants and admissions. No consistent associations were observed for PM_{10}. In Los Angeles, marginally significant associations were observed for $PM_{2.5}$, which were generally lower than for the gases. In co-pollutant models with CO, the $PM_{2.5}$ effect was reduced. Similar results were observed in the 0-19 and 20-64 year old age groups.

The $PM_{2.5}$ C-R functions are based on the co-pollutant models ($PM_{2.5}$ and CO) reported for the 20-64 and 65+ age groups. Since the true PM effect is most likely best represented by a distributed lag model, then any single lag model should underestimate the total PM effect. As a result, we selected the lag models with the greatest effect estimates for use in the C-R functions.

Ages 65 and older

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.8[88] and t-statistic of 0.8 for a 10 μg/m^3 increase in $PM_{2.5}$ in the two-day lag model (Moolgavkar, 2000b, Table 3, p. 80).

Functional Form: Log-linear
Coefficient: 0.0008
Standard Error: 0.001000
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 65+ (ICD codes 490-496)
Population: population of ages 65 and older

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[88] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.8 would result in a relative risk of 1.008 and coefficient of 0.000797. The "estimated" percent change, as reported by Moolgavkar, of 0.8 results in a relative risk of 1.008032 and coefficient of 0.0008.

Ages 18 to 64[89]

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 2.0[90] and t-statistic of 2.2 for a 10 μg/m^3 increase in $PM_{2.5}$ in the two-day lag model (Moolgavkar, 2000b, Table 4, p. 81).

Functional Form: Log-linear
Coefficient: 0.0020
Standard Error: 0.000909
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 18-64 (ICD codes 490-492, 494-496)[91]
Population: population of ages 18 to 64

F.4.4 Hospital Admissions for Chronic Lung Disease (less Asthma) (Samet et al., 2000, 14 Cities)

Samet et al. (2000) examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[92] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by

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[89] Although Moolgavkar (2000b) reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64.

[90] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 2.0 would result in a relative risk of 1.020 and coefficient of 0.001980. The "estimated" percent change, as reported by Moolgavkar, of 2.0 results in a relative risk of 1.020201 and coefficient of 0.002.

[91] Moolgavkar (2000b) reports results for ICD codes 490-496. In order to avoid double counting non-elderly asthma hospitalizations (ICD code 493) with Sheppard et al. (1999) in a total benefits estimation, we have excluded ICD code 493 from the baseline incidence rate used in this function.

[92] The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[93] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[94] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 2.88 percent increase (RR = 1.0288) in admissions due to a PM_{10} change of 10.0 $\mu g/m^3$ (Samet et al., 2000, Part II - Table 14)[95]. The standard error is estimated from the reported lower (0.19 percent) and upper bounds (5.64 percent) of the percent increase (Samet et al., 2000, Part II - Table 14).

Functional Form: Log-linear
Coefficient: 0.002839
Standard Error: 0.001351
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-492, 494-496)
Population: population of ages 65 and older

F.4.5 Hospital Admissions for Pneumonia (Lippmann et al., 2000, Detroit)

Lippmann et al. (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on the results of the co-pollutant model with ozone.

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[93] Joel Schwartz (co-author), personal communication.

[94] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

[95] The random effects estimate of the unconstrained distributed lag model was chosen for COPD admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.175 (95% CI 1.026-1.345) for a 36 μg/m^3 increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 26).

Functional Form: Log-linear
Coefficient: 0.004480
Standard Error: 0.001918
Incidence Rate: region-specific daily hospital admission rate for pneumonia admissions per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

F.4.6 Hospital Admissions for Pneumonia (Samet et al., 2000, 14 Cities)

Samet et al. (2000) examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[96] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[97] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[98] Finally, for the case of COPD, adjustment led to an increased PM_{10}

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[96] The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

[97] Joel Schwartz (co-author), personal communication.

[98] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "while the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 2.07 percent increase (RR = 1.0207) in admissions due to a PM_{10} change of 10.0 μg/m^3 (Samet et al., 2000, Part II - Table 14)[99]. The standard error is estimated from the reported lower (0.94 percent) and upper bounds (3.22 percent) of the percent increase (Samet et al., 2000, Part II - Table 14).

Functional Form: Log-linear
Coefficient: 0.002049
Standard Error: 0.000570
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

F.4.7 Hospital Admissions for All Cardiovascular (Moolgavkar, 2000a, Los Angeles)

Moolgavkar (2000a) examined the association between air pollution and cardiovascular hospital admissions (ICD 390-448) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO_2, NO_2, CO, and PM_{10} in all three areas. $PM_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group, the gaseous pollutants generally exhibited stronger effects than PM_{10} or $PM_{2.5}$. The strongest overall effects were observed for SO_2 and CO. In a single pollutant model, $PM_{2.5}$ was statistically significant for lag 0 and lag 1. In co-pollutant models with CO, the $PM_{2.5}$ effect dropped out and CO remained significant. For ages 20-64, SO_2 and CO exhibited the strongest effect and any $PM_{2.5}$ effect dropped out in co-pollutant models with CO. The $PM_{2.5}$ C-R functions are based on co-pollutant ($PM_{2.5}$ and CO) models.

Ages 65 and older

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.5[100] and t-statistic of 0.9 for a 10 μg/m^3 increase in $PM_{2.5}$ in the one day lag model (Moolgavkar, 2000a, Table 3, p. 1202).

Functional Form: Log-linear

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[99] The random effects estimate of the unconstrained distributed lag model was chosen for pneumonia admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

[100] In a log-linear model, the percent change is equal to (RR - 1) * 100. In a similar hospitalization study by Moolgavkar (2000b), he defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.5 would result in a relative risk of 1.005 and coefficient of 0.000499. Assuming that the 0.5 is the "estimated" percent change described previously would result in a relative risk of 1.005013 and coefficient of 0.0005. We assume that the "estimated" percent changes reported in this study reflect the definition from (Moolgavkar, 2000b).

Coefficient: 0.0005
Standard Error: 0.000556
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person 65+ (ICD codes 390-409, 411-459)[101]
Population: population of ages 65 and older

Ages 18 to 64[102]

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.9[103] and t-statistic of 1.8 for a 10 μg/m^3 increase in $PM_{2.5}$ in the zero lag model (Moolgavkar, 2000a, Table 4, p. 1203).

Functional Form: Log-linear
Coefficient: 0.0009
Standard Error: 0.000500
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person ages 18 to 64 (ICD codes 390-409, 411-459)[104]
Population: population of ages 18 to 64

F.4.8 Hospital Admissions for All Cardiovascular (Samet et al., 2000, 14 Cities)

Samet et al. (2000) examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[105] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility

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[101] Moolgavkar (2000a) reports results for ICD codes 390-429. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

[102] Although Moolgavkar (2000a) reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64.

[103] In a log-linear model, the percent change is equal to (RR - 1) * 100. In a similar hospitalization study by Moolgavkar (2000b), he defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.9 would result in a relative risk of 1.009 and coefficient of 0.000896. Assuming that the 0.9 is the "estimated" percent change described previously would result in a relative risk of 1.009041 and coefficient of 0.0009. We assume that the "estimated" percent changes reported in this study reflect the definition from (Moolgavkar, 2000b).

[104] Moolgavkar (2000a) reports results that include ICD code 410 (heart attack). In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

[105] The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[106] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[107] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 1.19 percent increase (RR = 1.0119) in admissions due to a PM_{10} change of 10.0 $\mu g/m^3$ (Samet et al., 2000, Part II - Table 14)[108]. The standard error is estimated from the reported lower (0.97 percent) and upper bounds (1.41 percent) of the percent increase (Samet et al., 2000, Part II - Table 14).

Functional Form: Log-linear
Coefficient: 0.001183
Standard Error: 0.000111
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular disease per person 65+ (ICD codes 390-459)
Population: population of ages 65 and older

F.4.9 Hospital Admissions for Dysrhythmia (Lippmann et al., 2000, Detroit)

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[106] Joel Schwartz (co-author), personal communication.

[107] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

[108] The fixed effects estimate of the unconstrained distributed lag model was chosen for CVD admissions since the chi-square test of heterogeneity was non-significant (see Samet et al., 2000, Part II - Table 15).

Lippmann et al. (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.080 (95% CI 0.904-1.291) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 27).

Functional Form: Log-linear
Coefficient: 0.002138
Standard Error: 0.002525
Incidence Rate: region-specific daily hospital admission rate for dysrhythmia admissions per person 65+ (ICD code 427)
Population: population of ages 65 and older

F.4.10 Hospital Admissions for Heart Failure (Lippmann et al., 2000, Detroit)

Lippmann et al. (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.183 (95% CI 1.053-1.329) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 27).

Functional Form: Log-linear
Coefficient: 0.004668
Standard Error: 0.001650

Incidence Rate: region-specific daily hospital admission rate for heart failure admissions per person 65+ (ICD code 428)
Population: population of ages 65 and older

F.4.11 Hospital Admissions for Ischemic Heart Disease (Lippmann et al., 2000, Detroit)

Lippmann et al. (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.041 (95% CI 0.947-1.144) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 27).

Functional Form: Log-linear
Coefficient: 0.001116
Standard Error: 0.001339
Incidence Rate: region-specific daily hospital admission rate for ischemic heart disease admissions per person 65+ (ICD codes 411-414)[109]
Population: population of ages 65 and older

[109] Lippmann et al. (2000) reports results for ICD codes 410-414. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

Exhibit F-5. Concentration-Response (C-R) Functions for Particulate Matter and Emergency Room Visits

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Asthma	$PM_{2.5}$	Norris et al.	1999	Seattle, WA	<18	All	All	NO_2, SO_2	24-hr avg	0.016527	0.004139	Log-linear
Asthma	PM_{10}	Schwartz et al.	1993	Seattle, WA	<65	All	All	None	24-hr avg	0.00367	0.00126	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

F.5 Emergency Room Visits

F.5.1 Emergency Room Visits for Asthma (Norris et al., 1999)

Norris et al. (1999) examined the relation between air pollution in Seattle and childhood (<18) hospital admissions for asthma from 1995 to 1996. The authors used air quality data for PM_{10}, light scattering (used to estimate fine PM), CO, SO_2, NO_2, and O_3 in a Poisson regression model with adjustments for day of the week, time trends, temperature, and dew point. They found significant associations between asthma ER visits and light scattering (converted to $PM_{2.5}$), PM_{10}, and CO. No association was found between O_3, NO_2, or SO_2 and asthma ER visits, although O_3 had a significant amount of missing data. In multipollutant models with either PM metric (light scattering or PM_{10}) and NO_2 and SO_2, the PM coefficients remained significant while the gaseous pollutants were not associated with increased asthma ER visits. The $PM_{2.5}$ C-R function is on the multipollutant model reported.

Multipollutant Model ($PM_{2.5}$, NO_2, and SO_2)

In a model with NO_2 and SO_2, the $PM_{2.5}$ coefficient and standard error are calculated from a relative risk of 1.17 (95% CI 1.08-1.26) for a 9.5 $\mu g/m^3$ increase in $PM_{2.5}$ (Norris et al., 1999, p. 491).

Functional Form: Log-linear
Coefficient: 0.016527
Standard Error: 0.004139
Incidence Rate: region-specific daily emergency room rate for asthma admissions per person <18 (ICD code 493)
Population: population of ages under 18

F.5.2 Emergency Room Visits for Asthma (Schwartz et al., 1993, Seattle)

Schwartz et al. (1993) examined the relationship between air quality and emergency room visits for asthma (ICD codes 493,493.01,493.10,493.90,493.91) in persons under 65 and 65 and over, living in Seattle from September 1989 to September 1990. Using single-pollutant models they found daily levels of PM_{10} linked to ER visits in individuals ages under 65, and they found no effect in individuals ages 65 and over. They did not find a significant effect for SO_2 and ozone in either age group. The results of the single pollutant model for PM_{10} are used in this analysis.

Single Pollutant Model

The PM_{10} coefficient and standard error are reported by Schwartz et al. (1993, p. 829) for a unit $\mu g/m^3$ increase in four-day average PM_{10} levels.

Functional Form: Log-linear
Coefficient: 0.00367
Standard Error: 0.00126
Incidence Rate: region-specific daily emergency room rate for asthma admissions per person <65 (ICD code 493)
Population: population of ages under 65

Exhibit F-6. Concentration-Response (C-R) Functions for Particulate Matter and Acute Effects

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Acute Bronchitis	$PM_{2.5}$	Dockery et al.	1996	24 communities	8-12	All	All	None	Annual Avg	0.027212	0.017096	Logistic
Acute Myocardial Infarction, Nonfatal	$PM_{2.5}$	Peters et al.	2001	Boston, MA	18+	All	All	None	24-hr avg	0.024121	0.009285	Logistic
Any of 19 Respiratory Symptoms	PM_{10}	Krupnick	1990	Los Angeles, CA	18-64	All	All	O_3	24-hr avg	0.000461	0.000239	Linear
Lower Respiratory Symptoms	$PM_{2.5}$	Schwartz et al.	1994	6 cities	7-14	All	All	None	24-hr avg	0.018232	0.005856	Logistic
Minor Restricted Activity Days	$PM_{2.5}$	Ostro and Rothschild	1989	nationwide	18-64	All	All	O_3	24-hr avg	0.00741	0.00070	Log-linear
Work Loss Days	$PM_{2.5}$	Ostro	1987	nationwide	18-64	All	All	None	24-hr avg	0.0046	0.00036	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

F.6 Acute Effects

F.6.1 Acute Bronchitis (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.[110] 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.

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 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. The C-R function is based on results of the single pollutant model reported in Table 1.

Single Pollutant Model

The estimated logistic coefficient and standard error are based on the odds ratio (1.50) and 95% confidence interval (0.91-2.47) 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.

Functional Form: Logistic
Coefficient: 0.027212
Standard Error: 0.017096
Incidence Rate: annual bronchitis incidence rate per person = 0.043 (American Lung Association, 2002a, Table 11)
Population: population of ages 8-12

F.6.2 Acute Myocardial Infarction (Heart Attacks), Nonfatal (Peters et al., 2001)

Peters et al. (2001) studied the relationship between increased particulate air pollution and onset of heart attacks in the Boston area from 1995 to 1996. The authors used air quality data for PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$,"black carbon", O_3, CO, NO_2, and SO_2 in a case-crossover analysis. For each subject, the case period was matched to three control periods, each 24 hours apart. In univariate analyses, the authors observed a positive association between heart attack occurrence and $PM_{2.5}$ levels hours before and days before onset. The authors estimated multivariate conditional logistic models including two-hour and

[110] 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.

twenty-four hour pollutant concentrations for each pollutant. They found significant and independent associations between heart attack occurrence and both two-hour and twenty-four hour $PM_{2.5}$ concentrations before onset. Significant associations were observed for PM_{10} as well. None of the other particle measures or gaseous pollutants were significantly associated with acute myocardial infarction for the two hour or twenty-four hour period before onset.

The patient population for this study was selected from health centers across the United States. The mean age of participants was 62 years old, with 21% of the study population under the age of 50. In order to capture the full magnitude of heart attack occurrence potentially associated with air pollution and because age was not listed as an inclusion criteria for sample selection, we apply an age range of 18 and over in the C-R function. According to the National Hospital Discharge Survey, there were no hospitalizations for heart attacks among children <15 years of age in 1999 and only 5.5% of all hospitalizations occurred in 15-44 year olds (Popovic, 2001, Table 10).

Single Pollutant Model

The coefficient and standard error are calculated from an odds ratio of 1.62 (95% CI 1.13-2.34) for a 20 $\mu g/m^3$ increase in twenty-four hour average $PM_{2.5}$ (Peters et al., 2001, Table 4, p. 2813).

Functional Form: Logistic
Coefficient: 0.024121
Standard Error: 0.009285
Incidence Rate: region-specific daily nonfatal heart attack rate per person 18+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410) [111]
Population: population of ages 18 and older

F.6.3 Any of 19 Respiratory Symptoms (Krupnick et al., 1990)

Krupnick et al. (1990) estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. (1990) listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included COH, ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily O_3, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

Multipollutant Model (PM_{10} and ozone)

[111] This estimate assumes that all heart attacks that are not instantly fatal will result in a hospitalization. In addition, Rosamond et al. (1999) report that approximately six percent of male and eight percent of female hospitalized heart attack patients die within 28 days (either in or outside of the hospital). We applied a factor of 0.93 to the number of hospitalizations to estimate the number of nonfatal heart attacks per year.

The C-R function used to estimate the change in ARD2 associated with a change in daily average PM_{10} concentration is based on Krupnick et al. (1990, p. 12):[112]

$$\Delta ARD2 \cong \beta^*_{PM_{10}} \cdot \Delta PM_{10} \cdot pop \,,$$

Functional Form: Linear
Coefficient: first derivative of the stationary probability = 0.000461
Standard Error: 0.000239
Population: population of ages 18-64 years[113]

The logistic regression model used by Krupnick et al. (1990) takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$probability(ARD2 | sickness\, or\, not_{t-1}) = p_i = \frac{1}{1 - e^{\beta_0 + \beta_1 \cdot ARD2_{t-1} + X \cdot \beta}} \,, for\ i = 0,1 \,.$$

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of COH (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in COH and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before} \,.$$

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[112] Krupnick and Kopp (1988, p. 2-24) and ESEERCO (1994, p. V-32) used the same C-R functional form as that used here.

[113] Krupnick et al. (1990, Table 1) reported the age distribution in their complete data, but they did not report the ages of individuals that were considered "adult." This analysis assumes that individuals 18 and older were considered adult. Only a small percentage (0.6%) of the study population is above the age of 60, so the C-R function was limited to the adult population. up through the age of 65.

However the full suite of coefficient estimates are not available.[114] Rather than use the full suite of coefficient values, the impact of COH on the probability of probability of ARD2 may be approximated by the derivative of ARD2 with respect to COH:

$$\frac{\partial probability(ARD2)}{\partial COH} = \frac{p_0 \cdot (1 - p_1) \cdot \beta_{COH} \cdot [p_1 + (1 - p_0)]}{(1 - p_1 + p_0)^2} = \beta^*_{COH} ,$$

where β_{COH} is the reported logistic regression coefficient for COH. Since COH data are not available for the benefits analysis, an estimated PM_{10} logistic regression coefficient is used based on the following assumed relationship between PM_{10}, COH, and TSP:

$$COH = 0.116 \cdot TSP$$

$$PM_{10} = 0.55 \cdot TSP$$

$$\Rightarrow COH = 0.2109 \cdot PM_{10}$$

$$\Rightarrow \beta_{PM_{10}} = 0.2109 \cdot \beta_{COH} = 0.2109 \cdot 0.0088 = 0.001856 .$$

This analysis uses $\beta_{COH} = 0.0088$ (Krupnick et al., 1990, Table V equation 3). The conversion from COH to TSP is based on study-specific information provided to ESEERCO (1994, p. V-32). The conversion of TSP to PM_{10} is from also from ESEERCO (1994, p. V-5), which cited studies by EPA (1986) and the California Air Resources Board (1982).

The change in the incidence of ARD2 associated with a given change in COH is then estimated by:

$$\frac{\partial ARD2}{\partial PM_{10}} \cong \frac{\Delta ARD2}{\Delta PM_{10}}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta PM_{10}} \cong \beta^*_{PM_{10}}$$

$$\Rightarrow \Delta ARD2 \cong \beta^*_{PM_{10}} \cdot \Delta PM_{10} .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO (1994, p. V-32), for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

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[114] The model without NO_2 (Krupnick et al., 1990, Table V equation 3) was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (1990, Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., 1990, Table IV). Both the ozone and COH coefficients dropped by about a factor of two or more.

$$\beta^*_{PM_{10}} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.001856 \cdot \left[0.7775_1 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = 0.000461.$$

The *standard error* for the coefficient is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. (1990, Table V):

$$\Rightarrow \beta_{PM_{10},high} = 0.2109 \cdot \beta_{COH,high} = 0.2109 \cdot \left(0.0088 + (1.96 \cdot 0.0046)\right) = 0.003757$$

$$\Rightarrow \beta^*_{PM_{10},high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.003757 \cdot \left[0.7775 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = 0.000934$$

$$\sigma_{\beta,high} = \frac{\beta_{PM_{10},high} - \beta_{PM_{10}}}{1.96} = \frac{(0.000934 - 0.000461)}{1.96} = 0.000236$$

$$\beta_{PM_{10},low} = 0.2109 \cdot \beta_{COH,low} = 0.2109 \cdot \left(0.0088 - (1.96 \cdot 0.0046)\right) = -4.555 \cdot 10^{-5}$$

$$\Rightarrow \beta^*_{PM_{10},low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot (-4.555 \cdot 10^{-5}) \cdot \left[0.7775 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = -1.132 \cdot 10^{-5}$$

$$\Rightarrow \sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(0.000461 + 1.132 \cdot 10^{-5}\right)}{1.96} = 0.000241$$

$$\sigma_\beta = \frac{\sigma_{\beta,high} + \sigma_{\beta,low}}{2} = 0.000239.$$

F.6.4 Lower Respiratory Symptoms (Schwartz et al., 1994)

Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+. The $PM_{2.5}$ C-R function is based on the single pollutant model reported in Table 5.

Single Pollutant Model

The coefficient and standard error are calculated from the reported odds ratio (1.44) and 95% confidence interval (1.15-1.82) associated with a 20 $\mu g/m^3$ change in $PM_{2.5}$ (Schwartz et al., 1994, Table 5).

Functional Form: Logistic
Coefficient: 0.018232
Standard Error: 0.005856
Incidence Rate: daily lower respiratory symptom incidence rate per person = 0.0012 (Schwartz et al., 1994, Table 2)
Population: population of ages 7 to 14

F.6.5 Minor Restricted Activity Days: Ostro and Rothschild (1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.[115] The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone has highly variable association with RRADs and MRADs. Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years. The C-R function for PM is based on this co-pollutant model.

The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994d, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994b; Schwartz, 1994c).

Multipollutant Model ($PM_{2.5}$ and ozone)

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma^2_{\beta_i}}}{\sum_{i=1976}^{1981} \frac{1}{\sigma^2_{\beta_i}}} \right) = 0.00741.$$

[115] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma}\right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma}\right).$$

This reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00070.$$

Functional Form: Log-linear
Coefficient: 0.00741
Standard Error: 0.00070
Incidence Rate: daily incidence rate for minor restricted activity days (MRAD) = 0.02137 (Ostro and Rothschild, 1989, p. 243)
Population: adult population ages 18 to 64

F.6.6 Work Loss Days (Ostro, 1987)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.[116] The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels[117] were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function presented here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994d, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994b; Schwartz, 1994c). On the other hand, the number of workers over the age of 65 is relatively small; it was approximately 3% of the total workforce in 2001 (U.S. Bureau of the Census, 2002, Table 561).

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[116] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

[117] The study used a two-week average pollution concentration; the C-R function uses a daily average, which is assumed to be a reasonable approximation.

Single Pollutant Model

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.0046 .$$

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \mathrm{var}\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1976}^{1981} \mathrm{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right) .$$

This eventually reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00036 .$$

Functional Form: Log-linear
Coefficient: 0.0046
Standard Error: 0.00036
Incidence Rate: daily work-loss-day incidence rate per person ages 18 to 64 = 0.00595 (U.S. Bureau of the Census, 1997, No. 22; Adams et al., 1999, Table 41)
Population: adult population ages 18 to 64

Exhibit F-7. Concentration-Response (C-R) Functions for Particulate Matter and Asthma-Related Effects

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form	Notes
Acute Bronchitis	$PM_{2.5}$	McConnell et al.	1999	Southern California	9-15	All	All	None	Annual Avg	0.022431	0.015957	Logistic	
Asthma Exacerbation, Asthma Attacks	PM_{10}	Whittemore and Korn	1980	Los Angeles, CA	All	All	All	O_3	24-hr avg	0.001436	0.000558	Logistic	
Asthma Exacerbation, Cough	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.003177	0.001156	Logistic	New onset of symptoms
Asthma Exacerbation, Cough	PM_{10}	Vedal et al.	1998	Vancouver, CAN	6-13	All	All	None	24-hr avg	0.007696	0.003786	Logistic	
Asthma Exacerbation, Moderate or Worse	$PM_{2.5}$	Ostro et al.	1991	Denver, CO	All	All	All	None	24-hr avg	0.0006	0.0003	Linear (log of pollutant)	
Asthma Exacerbation, One or More Symptoms	PM_{10}	Yu et al.	2000	Seattle, WA	5-13	All	All	CO, SO_2	24-hr avg	0.004879	0.005095	Logistic	
Asthma Exacerbation, Shortness of Breath	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.003177	0.001550	Logistic	New onset of symptoms
Asthma Exacerbation, Wheeze	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.002565	0.001030	Logistic	New onset of symptoms
Chronic Phlegm	$PM_{2.5}$	McConnell et al.	1999	Southern California	9-15	All	All	None	Annual Avg	0.063701	0.025580	Logistic	
Upper Respiratory Symptoms	PM_{10}	Pope et al.	1991	Utah Valley	9-11	All	All	None	24-hr avg	0.0036	0.0015	Logistic	

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

F.7 Asthma-Related Effects

F.7.1 Acute Bronchitis (McConnell et al., 1999)

McConnell et al. (1999) examined the relationship between air pollution and bronchitic symptoms among asthmatic 4th, 7th, and 10th grade children in southern California.[118] The authors collected information on the prevalence of bronchitis, chronic cough, and chronic phlegm among children with and without a history of asthma and/or wheeze. They used annual measurements of ozone, PM_{10}, $PM_{2.5}$, NO_2, and acids in a logistic regression model with adjustments for personal covariates. Neither bronchitis, cough, or phlegm were associated with any of the pollutants among children with no history of wheeze or asthma or a history of wheeze without diagnosed asthma. Among asthmatics, PM_{10} was significantly associated with bronchitis and phlegm; $PM_{2.5}$ was significantly associated with phlegm and marginally associated with bronchitis; NO_2 and acids were both significantly associated with phlegm; and ozone was not significantly associated with any of the endpoints.

Bronchitis was defined in the study by the question: "How many times in the past 12 months did your child have bronchitis?" (McConnell et al., 1999, p. 757). It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. McConnell et al. found a relationship between PM and chronic phlegm but none with chronic cough, each of which may be indicators of chronic bronchitis. For this analysis, we assumed that the C-R function based on McConnell et al. is measuring acute bronchitis. The $PM_{2.5}$ C-R function for bronchitis among asthmatics is based on the results of the single pollutant model reported in Table 3.

Single Pollutant Model

The estimated logistic coefficient and standard error are based on the odds ratio (1.4) and 95% confidence interval (0.9-2.3) associated with an increase in yearly mean 2-week average $PM_{2.5}$ of 15 $\mu g/m^3$. (McConnell et al., 1999, Table 3)

Functional Form: Logistic
Coefficient: 0.022431
Standard Error: 0.015957
Incidence Rate: annual incidence rate of one or more episodes of bronchitis per asthmatic = 0.326 (McConnell et al., 1999, Table 2)
Population: population of asthmatics ages 9 to 15 = 5.67%[119] of population ages 9 to 15

F.7.2 Asthma Attacks (Whittemore and Korn, 1980)

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants (O_x). Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks. The results from this model were used, and the oxidant result was adjusted so it may be used with ozone data.

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[118] Assuming that a child enters kindergarten at age 5, 4th grade corresponds to age 9 and 10th grade corresponds to age 15. We therefore applied the results of this study to children ages 9 to 15.

[119] The American Lung Association (2002c, Table 7) estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Multipollutant Model (PM_{10} and ozone)

The PM_{10} C-R function is based on the results of a co-pollutant model of TSP and ozone (Whittemore and Korn, 1980, Table 5). Assuming that PM_{10} is 55 percent of TSP[120] and that particulates greater than ten micrometers are harmless, the coefficient is calculated by dividing the TSP coefficient (0.00079) by 0.55. The standard error is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

Functional Form: Logistic
Coefficient: 0.001436
Standard Error: 0.000558
Incidence Rate: daily incidence of asthma attacks = 0.0550[121]
Population: population of asthmatics of all ages = 3.86% of the population of all ages (American Lung Association, 2002c, Table 7)

F.7.3 Asthma Exacerbation, Cough (Ostro et al., 2001)

Ostro et al. (2001) studied the relation between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects.[122] Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found cough prevalence associated with PM_{10} and $PM_{2.5}$ and cough incidence associated with $PM_{2.5}$ PM_{10}, and NO_2. Ozone was not significantly associated with cough among asthmatics. The $PM_{2.5}$ C-R function is based on the results of the single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.10 (95% CI 1.03-1.18) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration.

The C-R function based on this model will estimate the number of new onset episodes of cough avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. (2001) using the ratio of the probability of a symptom episode to the probability of a new onset episode. For cough, this ratio is 2.2 (14.5% divided by 6.7%) (Ostro et al., 2001, p.202).

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[120] The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).

[121] Based on an analysis of the 1999 National Health Interview Survey, the daily incidence of wheezing attacks for adult asthmatics is estimated to be 0.0550. In the same survey, wheezing attacks for children were examined, however, the number of wheezing attacks per year were censored at 12 (compared to censoring at 95 for adults). Due to the potential for underestimation of the number of children's wheezing attacks, we used the adult rate for all individuals.

[122] The authors note that there were 26 days in which $PM_{2.5}$ concentrations were reported higher than PM_{10} concentrations. The majority of results the authors reported were based on the full dataset. These results were used for the basis for the C-R functions.

In addition, not all children are at-risk for a new onset of cough, as defined by the study. On average, 14.5% of African-American asthmatics have cough on a given day (Ostro et al., 2001, p.202). Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.145 = 85.5%). As a result, a factor of 85.5% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new cough episode.

Functional Form: Logistic
Coefficient: 0.003177
Standard Error: 0.001156
Incidence Rate: daily new onset cough (incidence) rate per person (Ostro et al., 2001, p.202) = 0.067
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of cough = 6.21% of African-American population ages 8 to 13 multiplied (85.5% at-risk[123] times 7.26% asthmatic[124])
Adjustment Factor: average number of consecutive days with a cough episode (days) = 2.2

F.7.4 Asthma Exacerbation, Cough (Vedal et al., 1998)

Vedal et al. (1998) studied the relationship between air pollution and respiratory symptoms among asthmatics and non-asthmatic children (ages 6 to 13) in Port Alberni, British Columbia, Canada. Four groups of elementary school children were sampled from a prior cross-sectional study: (1) all children with current asthma, (2) children without doctor diagnosed asthma who experienced a drop in FEV after exercise, (3) children not in groups 1 or 2 who had evidence of airway obstruction, and (4) a control group of children with matched by classroom. The authors used logistic regression and generalized estimating equations to examine the association between daily PM_{10} levels and daily increases in various respiratory symptoms among these groups. In the entire sample of children, PM_{10} was significantly associated with cough, phlegm, nose symptoms, and throat soreness. Among children with diagnosed asthma, the authors report a significant association between PM_{10} and cough symptoms, while no consistent effects were observed in the other groups. Since the study population has an over-representation of asthmatics, due to the sampling strategy, the results from the full sample of children are not generalizeable to the entire population. The C-R function presented below is based on results among asthmatics only.

Single Pollutant Model

The PM_{10} coefficient and standard error are based on an increase in odds of 8% (95% CI 0-16%) reported in the abstract for a 10 $\mu g/m^3$ increase in daily average PM_{10}.

Functional Form: Logistic
Coefficient: 0.007696
Standard Error: 0.003786
Incidence Rate: daily cough rate per person (Vedal et al., 1998, Table 1, p. 1038) = 0.086

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[123] On average, 17.3% of African-American asthmatics have cough episodes on a given day (Ostro et al., 2001, p.202). Only those who are symptom-free on the previous day (1-0.145 = 85.5%) are at-risk for a new onset episode.

[124] The American Lung Association (2002c, Table 9) estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

Population: asthmatic population ages 6 to 13 = 5.67%[125] of population ages 6 to 13

F.7.5 Asthma Exacerbation, Moderate or Worse (Ostro et al., 1991)

Ostro et al. (1991) examined the effect of air pollution on asthmatics, ages 18 to 70, living in Denver, Colorado from December 1987 to February 1988. The respondents in this study were asked to record daily a subjective rating of their overall asthma status each day (0=none, 1=mild, 2=moderate, 3=severe, 4=incapacitating). Ostro et al. then examined the relationship between moderate (or worse) asthma and H^+, sulfate, SO_2, $PM_{2.5}$, estimated $PM_{2.5}$, PM_{10}, nitrate, and nitric acid. Daily levels of H^+ were linked to cough, asthma, and shortness of breath. $PM_{2.5}$ was linked to asthma. Sulfate was linked to shortness of breath. No effects seen for other pollutants. The C-R function is based on a single-pollutant linear regression model where the log of the pollutant is used.

Single Pollutant Model

Two $PM_{2.5}$ coefficients are presented, both equal 0.0006, however only one is significant. The coefficient based on data that does not include estimates of missing $PM_{2.5}$ values is not significant (std error = 0.0053); the coefficient that includes estimates of missing $PM_{2.5}$ values (estimated using a function of sulfate and nitrate) is significant at $p < 0.5$ (std error = 0.0003). The latter coefficient is used here.

The C-R function to estimate the change in the number of days with moderate (or worse) asthma

$$\Delta Days\ Moderate\ /\ Worse\ Asthma = -\beta \cdot \ln\left(\frac{PM_{2.5,\,after}}{PM_{2.5,\,before}}\right) \cdot pop\ ,$$

Functional Form: Linear (using log of the pollutant)
Coefficient: 0.0006
Standard Error: 0.0003
Population: population of asthmatics of all ages[126] = 3.86% of the population of all ages (American Lung Association, 2002c, Table 7)

F.7.6 Asthma Exacerbation, One or More Symptoms (Yu et al., 2000)

Yu et al. (2000) examined the association between air pollution and asthmatic symptoms among mild to moderate asthmatic children ages 5-13 in Seattle. They collected air quality data for CO, SO_2, PM_{10}, and $PM_{1.0}$ and asked study subjects to record symptoms daily. They used logistic regression models with generalized estimating equations in two different approaches. A "marginal approach" was used to estimate the impact of air pollution on asthma symptoms and a "transition approach" was used to estimate

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[125] The American Lung Association (2002c, Table 7) estimates asthma prevalence for children 5-17 at 5.67% (based on data from the 1999 National Health Interview Survey).

[126] The C-R function is applied to asthmatics of all ages, although the study population consists of asthmatics between the ages of 18 and 70. It seems reasonable to assume that individuals over the age of 70 are at least as susceptible as individuals in the study population. It also seems reasonable to assume that individuals under the age of 18 are also susceptible. For example, controlling for oxidant levels, Whittemore and Korn (1980) found TSP significantly related to asthma attacks in a study population comprised primarily (59 percent) of individuals less than 16 years of age.

the association conditioned on the previous day's outcome. The primary endpoint, odds of at least one asthma symptom, was significantly associated with CO, PM_{10}, and $PM_{1.0}$ in single pollutant models. In multipollutant models, CO remained significant while PM effects declined slightly. The magnitude of the effects were similar between the "marginal" and "transition" approaches. The C-R function is based on the results of the "transition approach," where the previous day's symptoms is an explanatory variable.

Multipollutant Model (PM_{10}, CO, SO_2)

The C-R function is based on the results of the "transition approach," where the previous day's symptoms is an explanatory variable. The multipollutant PM_{10} coefficient and standard error are based on the odds ratio (1.05) and 95% confidence interval (0.95-1.16) for a 10 $\mu g/m^3$ increase in one-day lagged daily average PM_{10} (Yu et al., 2000, Table 4, p. 1212).

Functional Form: Logistic
Coefficient: 0.004879
Standard Error: 0.005095
Incidence Rate: daily rate of at least one asthma episode per person (Yu et al., 2000, Table 2, p. 1212) = 0.60
Population: asthmatic population ages 5 to 13 = 5.67%[127] of population ages 5 to 13

F.7.7 Asthma Exacerbation, Shortness of Breath (Ostro et al., 2001)

Ostro et al. (2001) studied the relationship between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM_{10}, $PM_{2.5}$, NO_2, and ozone in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "new onset of a symptom episode". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found that both the prevalent and incident episodes of shortness of breath were associated with $PM_{2.5}$ and PM_{10}. Neither ozone nor NO_2 were significantly associated with shortness of breath among asthmatics. The $PM_{2.5}$ C-R function is based on the results of a single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.10 (95% CI 1.00-1.20) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration (Ostro et al., 2001, Table 5, p.204).

The C-R function based on this model will estimate the number of new onset episodes of shortness of breath avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. (2001) using the ratio of the probability of a symptom episode to the probability of a new onset episode. For shortness of breath, this ratio is 2.0 (7.4% divided by 3.7%) (Ostro et al., 2001, p.202).

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[127] The American Lung Association (2002c, Table 7) estimates asthma prevalence for children 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

In addition, not all children are at-risk for a new onset of shortness of breath, as defined by the study. On average, 7.4% of African-American asthmatics have shortness of breath episodes on a given day (Ostro et al., 2001, p.202). Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.074 = 92.6%). As a result, a factor of 92.6% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new shortness of breath episode.

Functional Form: Logistic
Coefficient: 0.003177
Standard Error: 0.001550
Incidence Rate: daily new onset shortness of breath (incidence) rate per person (Ostro et al., 2001, p.202) = 0.037
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of shortness of breath = 6.72% of African-American population ages 8 to 13 multiplied (92.6% at-risk[128] times 7.26% asthmatic[129])
Adjustment Factor: average number of consecutive days with a shortness of breath episode (days) = 2.0

F.7.8 Asthma Exacerbation, Wheeze (Ostro et al., 2001)

Ostro et al. (2001) studied the relation between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found both the prevalence and incidence of wheeze associated with $PM_{2.5}$ PM_{10}, and NO_2. Ozone was not significantly associated with wheeze among asthmatics. The $PM_{2.5}$ C-R function is based on the results of a single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.08 (95% CI 1.01-1.14) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration (Ostro et al., 2001, Table 5, p.204).

The C-R function based on this model will estimate the number of new onset episodes of wheeze avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. (2001) using the ratio of the probability of a symptom episode to the probability of a new onset episode. For wheeze, this ratio is 2.3 (17.3% divided by 7.6%) (Ostro et al., 2001, p.202).

In addition, not all children are at-risk for a new onset of wheeze, as defined by the study. On average, 17.3% of African-American asthmatics have wheeze on a given day (Ostro et al., 2001, p.202). Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.173 = 82.7%). As a result, a factor of 82.7% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new wheeze episode.

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[128] On average, 7.4% of African-American asthmatics have shortness of breath episodes on a given day (Ostro et al., 2001, p.202). Only those who are symptom-free on the previous day (1-0.074 = 92.6%) are at-risk for a new onset episode.

[129] The American Lung Association (2002c, Table 9) estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

Functional Form: Logistic
Coefficient: 0.002565
Standard Error: 0.001030
Incidence Rate: daily new onset wheeze (incidence) rate per person (Ostro et al., 2001, p.202) = 0.076
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of wheeze = 6.00% of African-American population ages 8 to 13 multiplied (82.7% at-risk[130] times 7.26% asthmatic[131])
Adjustment Factor: average number of consecutive days with a wheeze episode (days) = 2.3

F.7.9 Chronic Phlegm (McConnell et al., 1999)

McConnell et al. (1999) examined the relationship between air pollution and bronchitic symptoms among asthmatic 4th, 7th, and 10th grade children in southern California.[132] The authors collected information on the prevalence of bronchitis, chronic cough, and chronic phlegm among children with and without a history of asthma and/or wheeze. They used annual measurements of ozone, PM_{10}, $PM_{2.5}$, NO_2, and acids in a logistic regression model with adjustments for personal covariates. Neither bronchitis, cough, or phlegm were associated with any of the pollutants among children with no history of wheeze or asthma or a history of wheeze without diagnosed asthma. Among asthmatics, PM_{10} was significantly associated with bronchitis and phlegm; $PM_{2.5}$ was significantly associated with phlegm and marginally associated with bronchitis; NO_2 and acids were both significantly associated with phlegm; and ozone was not significantly associated with any of the endpoints.

Phlegm was defined in the study by the question: "Other than with colds, does this child usually seem congested in the chest or bring up phlegm?" (McConnell et al., 1999, p. 757). The authors refer to this definition as "chronic phlegm" and we also assume that the term "usually" refers to chronic, rather than acute, phlegm. The PM C-R functions for chronic phlegm among asthmatics are based on the results of the single pollutant model reported in Table 3.

Single Pollutant Model

The estimated logistic coefficient and standard error are based on the odds ratio (2.6) and 95% confidence interval (1.2-5.4) associated with an increase in yearly mean 2-week average $PM_{2.5}$ of 15 $\mu g/m^3$. (McConnell et al., 1999, Table 3)

Functional Form: Logistic
Coefficient: 0.063701
Standard Error: 0.025580
Incidence Rate: annual incidence rate of phlegm per asthmatic = 0.257 (McConnell et al., 1999, Table 2)

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130 On average, 17.3% of African-American asthmatics have wheeze episodes on a given day (Ostro et al., 2001, p.202). Only those who are symptom-free on the previous day (1-0.173 = 82.7%) are at-risk for a new onset episode.

131 The American Lung Association (2002c, Table 9) estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

132 Assuming that a child enters kindergarten at age 5, 4th grade corresponds to age 9 and 10th grade corresponds to age 15. We therefore applied the results of this study to children ages 9 to 15.

Population: population of asthmatics ages 9 to 15 = 5.67%[133] of population ages 9 to 15

F.7.10 Upper Respiratory Symptoms (Pope et al., 1991)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

Single Pollutant Model

The coefficient and standard error for a one $\mu g/m^3$ change in PM_{10} is reported in Table 5.

Functional Form: Logistic
Coefficient: 0.0036
Standard Error: 0.0015
Incidence Rate: daily upper respiratory symptom incidence rate per person = 0.3419 (Pope et al., 1991, Table 2)
Population: asthmatic population ages 9 to 11 = 5.67%[134] of population ages 9 to 11

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[133] The American Lung Association (2002c, Table 7) estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

[134] The American Lung Association (2002c, Table 7) estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Appendix G: Ozone Concentration-Response Functions

In this Appendix, we present the concentration-response (C-R) functions used to estimate ozone-related adverse health effects. Each sub-section has an Exhibit with a brief description of the C-R function and the underlying parameters. Following each Exhibit, we present a brief summary of each of the studies and any items that are unique to the study.

Note that the main text describes the methods that we used to choose these C-R functions from the wide range available in the literature. In addition, Appendix D mathematically derives the standard types of C-R functions that we encountered in the epidemiological literature, such as, log-linear, logistic and linear, so we simply note here the type of functional form. Finally, Appendix E presents a detailed description of the sources for the incidence and prevalence data used in these C-R functions.

Exhibit G-1. Concentration-Response (C-R) Functions for Ozone and Short-Term Mortality

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Non-Accidental	Ito and Thurston	1996	Chicago, IL	All	All	All	PM_{10}	1-hr max	Log-linear	0.000634	0.000251
Non-Accidental	Kinney et al.	1995	Los Angeles, CA	All	All	All	PM_{10}	1-hr max	Log-linear	0	0.000214
Non-Accidental	Moolgavkar et al.	1995	Philadelphia, PA	All	All	All	SO_2, TSP	24-hr avg	Log-linear	0.000611	0.000216
Non-Accidental	Samet et al.	1997	Philadelphia, PA	All	All	All	CO, NO_2, SO_2, TSP	24-hr avg	Log-linear	0.000936	0.000312

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

G .1 Short-term Mortality

Exhibit 1 summarizes the C-R functions used to estimate the relationship between ozone and short-term mortality. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

G .1.1 Short-Term Mortality, Non-Accidental (Ito and Thurston, 1996, Chicago)

Ito and Thurston (1996) examined the relationship between daily non-accidental mortality and air pollution levels in Cook County, Illinois from 1985 to 1990. They examined daily levels of ozone, PM_{10}, SO_2, and CO, and found a significant relationship for ozone and PM_{10} with both pollutants in the model; no significant effects were found for SO_2 and CO. In single pollutant models the effects were slightly larger. The C-R function for ozone is based on the results of the co-pollutant model.

Multipollutant Model (ozone and PM_{10})

In a co-pollutant model with PM_{10}, the coefficient (0.000634) and standard error (0.000251) were obtained directly from the author because the published paper reported incorrect information.

Functional Form: Log-linear
Coefficient: 0.000634
Standard Error: 0.000251
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

G .1.2 Short-Term Mortality, Non-Accidental (Kinney et al., 1995, Los Angeles)

Kinney et al. (1995) examined the relationship between daily non-accidental mortality and air pollution levels in Los Angeles, California from 1985 to 1990. They examined ozone, PM_{10}, and CO, and found a significant relationship for each pollutant in single pollutant models. The effect for ozone dropped to zero with the inclusion of PM_{10} in the model, while the effect for CO and PM_{10} appeared co-pollutant ozone models. The C-R function for ozone is based on the results of the co-pollutant model.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are based on the relative risk (1.00) and 95% confidence interval (0.94-1.06) reported for a 143 ppb increase in daily one-hour maximum ozone concentration (Kinney et al., 1995, Table 2, p. 64).

Functional Form: Log-linear
Coefficient: 0
Standard Error: 0.000214
Incidence: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

G .1.3 Short-Term Mortality, Non-Accidental (Moolgavkar et al., 1995, Philadelphia)

Moolgavkar et al. (1995) examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1973 to 1988. They examined ozone, TSP, and SO_2 in a three-pollutant model, and found a significant relationship for ozone and SO_2; TSP was not significant. In season-specific models, ozone was significantly associated with mortality only in the summer months. The C-R function for ozone is based on the full-year three-pollutant model reported in Table 5 (Moolgavkar et al., 1995, p. 482).

Multipollutant Model (ozone, SO_2, TSP)

The coefficient and standard error are based on the relative risk (1.063) and 95% confidence interval (1.018-1.108) associated with a 100 ppb increase in daily average ozone (Moolgavkar et al., 1995, p. 482, Table 5).

Functional Form: Log-linear
Coefficient: 0.000611
Standard Error: 0.000216
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

G .1.4 Short-Term Mortality, Non-Accidental (Samet et al., 1997, Philadelphia)

Samet et al. (1997) examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1974 to 1988. They examined ozone, TSP, SO_2, NO_2, and CO in a Poisson regression model. In single pollutant models, ozone, SO_2, TSP, and CO were significantly associated with mortality. In a five-pollutant model, they found a positive statistically significant relationship for each pollutant except NO_2. The C-R function for ozone is based on the five-pollutant model (ozone, CO, NO_2, SO_2, and TSP) reported in Table 9 (Samet et al., 1997, p. 20).

Multipollutant Model (ozone, CO, NO_2, SO_2, and TSP)

In a model with CO, NO_2, SO_2, and TSP, the ozone coefficient and standard error are based on the percent increase (1.91) and t-statistic (3) associated with a 20.219 ppb increase in two-day average ozone (Samet et al., 1997, p. 20, Table 9).

Functional Form: Log-linear
Coefficient: 0.000936
Standard Error: 0.000312
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Exhibit G-2. Concentration-Response (C-R) Functions for Ozone and Chronic Illness

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Chronic Asthma	McDonnell et al.	1999	SF, SD, South Coast Air Basin	27+	All	Male	None	annual avg 8-hr avg	Logistic	0.0277	0.0135

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

G .2 Chronic Illness

Exhibit 2 summarizes the C-R function ((McDonnell et al., 1999)) used to estimate the relationship between ozone and chronic asthma. A more detailed summary of McDonnell et al. (1999), and the parameters used in the function, is described below.

G .2.1 Chronic Asthma (McDonnell et al., 1999)

McDonnell et al. (1999) used the same cohort of Seventh-Day Adventists as Abbey et al. (1993), and examined the association between air pollution and the onset of asthma in adults between 1977 and 1992. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures, controlling for other covariates; no significant effect was found between ozone exposure and asthma in females. No significant effect was reported for females or males due to exposure to PM, NO_2, SO_2, or SO_4. The C-R function for ozone is based on the single pollutant model for males reported in Table 5 (McDonnell et al., 1999, 1999, p. 117).

Single Pollutant Model

The coefficient and standard error for males is reported in Table 5 for a unit increase in annual average eight-hour ozone concentrations.[135]

Functional Form: Logistic
Coefficient: 0.0277
Standard Error: 0.0135
Incidence Rate: annual asthma incidence rate per person = 0.00219 (McDonnell et al., 1999, 1999, Table 4)
Population: non-asthmatic males age 27 and over = 97.9%[136] of males 27+

[135] The eight-hour ozone concentration is defined as 9:00 A.M. to 4:59 P.M. The study used the 1973-1992 mean 8-hour average ambient ozone concentration (McDonnell et al., 1999, p. 113).

[136] The prevalence of asthma among males 27 and older (2.10 percent) was estimated from the 2000 National Health Interview Survey (NHIS) public use data, available at ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000.

Exhibit G-3. Concentration-Response (C-R) Functions for Ozone and Hospital Admissions

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
All Respiratory	Burnett et al.	2001	Toronto, CAN	<2	All	All	$PM_{2.5}$	1-hr max	Log-linear	0.006309	0.001834
All Respiratory	Schwartz	1995	New Haven, CT	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.002652	0.001398
All Respiratory	Schwartz	1995	Tacoma, WA	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.007147	0.002565
Chronic Lung Disease	Moolgavkar et al.	1997	Minneapolis, MN	65+	All	All	CO, PM_{10}	24-hr avg	Log-linear	0.002743	0.001699
Chronic Lung Disease (less Asthma)	Schwartz	1994	Detroit, MI	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.00549	0.00205
Pneumonia	Moolgavkar et al.	1997	Minneapolis, MN	65+	All	All	NO_2, PM_{10}, SO_2	24-hr avg	Log-linear	0.003696	0.001030
Pneumonia	Schwartz	1994	Detroit, MI	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.00521	0.0013
Pneumonia	Schwartz	1994	Minneapolis, MN	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.003977	0.001865

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

G .3 Hospital Admissions

Exhibit 3 summarizes the C-R functions used to estimate the relationship between ozone and hospital admissions. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

G .3.1 Hospital Admissions for All Respiratory (Burnett et al., 2001, Toronto)

Burnett et al. (2001) studied the association between air pollution and acute respiratory hospital admissions (ICD codes 493, 466, 464.4, 480-486) in Toronto from 1980-1994, among children less than 2 years of age. They collected hourly concentrations of the gaseous pollutants, CO, NO_2, SO_2, and ozone. Daily measures of particulate matter were estimated for the May to August period of 1992-1994 using TSP, sulfates, and coefficient of haze data. The authors report a positive association between ozone in the May through August months and respiratory hospital admissions, for several single days after elevated ozone levels.

The strongest association was found using a five-day moving average of ozone. No association was found in the September through April months. In co-pollutant models with a particulate matter or another gaseous pollutant, the ozone effect was only slightly diminished. The effects for PM and gaseous pollutants were generally significant in single pollutant models but diminished in co-pollutant models with ozone, with the exception of CO. The C-R function for ozone is based on a co-pollutant model with $PM_{2.5}$, using the five-day moving average of one-hour max ozone.

Multipollutant Model (ozone and $PM_{2.5}$)

In a model with $PM_{2.5}$, the coefficient and standard error are based on the percent increase (33.0) and t-statistic (3.44) associated with a 45.2 ppb increase in the five-day moving average of one-hour max ozone (Burnett et al., 2001, Table 3).

Functional Form: Log-linear
Coefficient: 0.006309
Standard Error: 0.001834
Incidence Rate: region-specific daily hospital admission rate for all respiratory admissions per person less than 2 years of age (ICD codes 464, 466, 480-487, 493)
Population: population less than 2 years of age

G .3.2 Hospital Admissions for All Respiratory (Schwartz, 1995, New Haven)

Schwartz (1995) examined the relationship between air pollution and respiratory hospital admissions (ICD codes 460-519) for individuals 65 and older in New Haven, Connecticut, from January 1988 to December 1990. In single-pollutant models, PM_{10} and SO_2 were significant, while ozone was marginally significant. In a co-pollutant model with ozone and PM_{10}, both pollutants were significant. PM_{10} remained significant in a model with SO_2, while ozone was marginally significant when adjusted for SO_2. SO_2 was significant in a co-pollutant model with PM_{10} but not with ozone. The ozone C-R function is based on results from the co-pollutant model with PM_{10}.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are estimated from the relative risk (1.07) and 95% confidence interval (1.00-1.15) for a 50 μg/m^3 increase in average daily ozone levels (Schwartz, 1995, Table 3, p. 534).[137]

Functional Form: Log-linear
Coefficient: 0.002652
Standard Error: 0.001398
Incidence Rate: region-specific daily hospital admission rate for respiratory admissions per person 65+ (ICD codes 460-519)
Population: population of ages 65 and older

G .3.3 Hospital Admissions for All Respiratory (Schwartz, 1995, Tacoma)

Schwartz (1995) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Tacoma, Washington, from January 1988 to December 1990. In single-pollutant models, PM_{10}, ozone, and SO_2 were all significant. Ozone remained significant in separate co-pollutant models with PM_{10} and SO_2. PM_{10} remained significant in a co-pollutant model with SO_2, but not in a co-pollutant model with ozone. SO_2 was not significant in either of the co-pollutant models. The ozone C-R function is based on results from the co-pollutant model with PM_{10}.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are estimated from the relative risk (1.20) and 95% confidence interval (1.06-1.37) for a 50 μg/m^3 increase in average daily ozone levels (Schwartz, 1995, Table 6, p. 535).[138]

Functional Form: Log-linear
Coefficient: 0.007147
Standard Error: 0.002565
Incidence Rate: region-specific daily hospital admission rate for respiratory admissions per person 65+ (ICD codes 460-519)
Population: population of ages 65 and older

G .3.4 Hospital Admissions for Chronic Lung Disease (Moolgavkar et al., 1997, Minneapolis)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions (ICD codes 490-496) for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a Poisson regression, they found no significant effect for any of the pollutants (PM_{10}, ozone, or CO). The effect for ozone was marginally significant. The model with a 100 df

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[137] To calculate the coefficient, a conversion of 1.96 μg/m^3 per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million μg in a gram, this density means that there are 1.96 billion μg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 μg of ozone (i.e., one ppb = 1.96 μg/m^3).

[138] To calculate the coefficient, a conversion of 1.96 μg/m^3 per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million μg in a gram, this density means that there are 1.96 billion μg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 μg of ozone (i.e., one ppb = 1.96 μg/m^3).

smoother was reported to be optimal (p. 368). The C-R function is based on the results from a three-pollutant model (ozone, CO, PM_{10}) using the 100 df smoother.

Multipollutant Model (ozone, CO, PM_{10})

In a model with CO and PM_{10}, the estimated coefficient and standard error are based on the percent increase (4.2) and 95% confidence interval of the percent increase (-1.0-9.4) associated with a change in daily average ozone levels of 15 ppb (Moolgavkar et al., 1997, Table 4 and p. 366).

Functional Form: Log-linear
Coefficient: 0.002743
Standard Error: 0.001699
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-496)
Population: population of ages 65 and older

G .3.5 Hospital Admissions for Chronic Lung Disease (less Asthma) (Schwartz, 1994b, Detroit)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions (ICD codes 491-492, 494-496) for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant Poisson regression model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD. The authors state that effect estimates were relatively unchanged compared to the unreported single pollutant models. No significant associations were found between either pollutant and asthma admissions. The C-R function for chronic lung disease incidence is based on the results of the "basic" co-pollutant model (ozone and PM_{10}) presented in Table 4 (p. 651).[139]

Multipollutant Model (ozone and PM_{10})

The coefficient and standard error for the "basic" model are reported in Table 4 (Schwartz, 1994b, p.651) for a one ppb change in daily average ozone.

Functional Form: Log-linear
Coefficient: 0.00549
Standard Error: 0.00205
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-492, 494-496)
Population: population of ages 65 and older

G .3.6 Hospital Admissions for Pneumonia (Moolgavkar et al., 1997, Minneapolis)

Moolgavkar et al. (1997) examined the relationship between air pollution and pneumonia hospital admissions (ICD 480-487) for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a four pollutant Poisson model examining pneumonia admissions in Minneapolis, ozone was significant, while NO_2, SO_2, and PM_{10} were not significant. The model with a

[139] Schwartz (1994b) also reports results using generalized additive models to fit time and temperature variables, however no standard error or confidence intervals were reported.

130 df smoother was reported to be optimal (p. 368). The ozone C-R function is based on the results from the four-pollutant model with a 130 df smoother.

Multipollutant Model (ozone, NO_2, PM_{10}, SO_2)

In a model with NO_2, PM_{10},and SO_2, the estimated coefficient and standard error are based on the percent increase (5.7) and 95% confidence interval of the percent increase (2.5-8.9) associated with an increase in daily average ozone levels of 15 ppb (Moolgavkar et al., 1997, Table 4 and p. 366).

Functional Form: Log-linear
Coefficient: 0.003696
Standard Error: 0.00103
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

G .3.7 Hospital Admissions for Pneumonia (Schwartz, 1994b, Detroit)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant Poisson regression model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD. The authors state that effect estimates were relatively unchanged compared to the unreported single pollutant models. No significant associations were found between either pollutant and asthma admissions. The PM_{10} C-R function for pneumonia incidence is based on results of the "basic" co-pollutant model (ozone and PM_{10}).[140]

Multipollutant Model (ozone and PM_{10})

The ozone C-R function for pneumonia incidence is based on the coefficient and standard error for the "basic" co-pollutant model presented in Table 4 (Schwartz, 1994b, p. 651).

Functional Form: Log-linear
Coefficient: 0.00521
Standard Error: 0.0013
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

G .3.8 Hospital Admissions for Pneumonia (Schwartz, 1994a, Minneapolis)

Schwartz (1994a) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1989. In single-pollutant Poisson regression models, both ozone and PM_{10} were significantly associated with pneumonia admissions. In a two-pollutant model, Schwartz found PM_{10} significantly related to pneumonia; ozone was weakly linked to pneumonia. The results were not sensitive to the methods used

[140] Schwartz (1994b) also reports results using generalized additive models to fit time and temperature variables, however no standard error or confidence intervals were reported.

to control for seasonal patterns and weather. The ozone C-R function is based on the results of the two-pollutant model (PM_{10} and ozone) with spline smoothing for temporal patterns and weather.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10} and spline functions to adjust for time and weather, the coefficient and standard error are based on the relative risk (1.22) and 95% confidence interval (1.02, 1.47) for a 50 ppb increase in daily average ozone levels (Schwartz, 1994a, Table 4).

Functional Form: Log-linear
Coefficient: 0.003977
Standard Error: 0.001865
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

Exhibit G-4. Concentration-Response (C-R) Functions for Ozone and Emergency Room Visits

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Asthma	Cody et al.	1992	New Jersey (Northern)	All	All	All	SO_2	5-hr avg	Linear	0.0203	0.00717
Asthma	Stieb et al.	1996	New Brunswick, CAN	All	All	All	None	1-hr max	Quadratic	0.00004	0.00002
Asthma	Weisel et al.	1995	New Jersey (Northern and Central)	All	All	All	None	5-hr avg	Linear	0.0443	0.00723

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

G .4 Emergency Room Visits

Exhibit 4 summarizes the C-R functions used to estimate the relationship between ozone and emergency room visits. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

G .4.1 Emergency Room Visits for Asthma (Cody et al., 1992, Northern NJ)

Cody et al. (1992) examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1988-1989. In a two pollutant multiple linear regression model, ozone was linked to asthma visits, and no effect was seen for SO_2. They modeled PM_{10} in separate analysis because of limited (every sixth day) sampling. No significant effect was seen for PM_{10}. The C-R function for ozone is based on results of a co-pollutant model with SO_2 (Cody et al., 1992, Table 6, p. 191).

Multipollutant Model (ozone and SO_2)

The ozone coefficient and standard error are reported per 1 ppm increment of five-hour ozone levels, which are converted to a 1 ppb increment by dividing by 1,000 (Cody et al., 1992, Table 6, p. 191).

Functional Form: Linear
Coefficient: 0.0203
Standard Error: 0.00717
Baseline Population: baseline population of Northern New Jersey[141] = 4,436,976
Population: population of all ages

G .4.2 Emergency Room Visits for Asthma (Stieb et al., 1996, New Brunswick)

Stieb et al. (1996) examined the relationship between ER visits and air pollution for persons of all ages in St. John, New Brunswick, Canada, from May through September in 1984-1992. Ozone was significantly linked to ER visits, especially when ozone levels exceeded 75 ppb. The authors reported results from a linear model, quadratic model, and linear-quadratic model using daily average and 1-hour maximum ozone. In the linear model, ozone was borderline significant. In the quadratic and linear-quadratic models, ozone was highly significant. This is consistent with the author's conclusion that "only ozone appeared to have a nonlinear relationship with visit rates" (p. 1356) and that "quadratic, linear-quadratic, and indicator models consistently fit the data better than the linear model ..." (p. 1358). The linear term in the linear-quadratic model is negative, implying that at low ozone levels, increases in ozone are associated with decreases in risk. Since this does not seem biologically plausible, the ozone C-R function described here is based on the results of the quadratic regression model presented in Table 2 (Stieb et al., 1996, p. 1356), for a change in one-hour maximum ozone levels.

Single Pollutant Model

[141] The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. (1992, Figure 1) presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

The coefficient and standard error of the quadratic model are reported in Table 2 (Stieb et al., 1996, p. 1356) for a 1 ppb increase in 1-hour daily maximum ozone levels. The C-R function to estimate avoided emergency visits derived from a quadratic regression model is shown below:

$$\Delta\ Asthma\ ERVisits = \frac{\beta}{BasePop} \cdot [(O_{3,baseline})^2 - (O_{3,control})^2] \cdot pop,$$

Functional Form: Quadratic
Coefficient: 0.00004
Standard Error: 0.00002
Baseline Population: baseline population of St. John, New Brunswick (Stieb et al., 1996, p. 1354) = 125,000
Population: population of all ages

G .4.3 Emergency Room Visits for Asthma (Weisel et al., 1995, Northern NJ)

Weisel et al. (1995) examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1986-1990. A significant relationship was reported for ozone. The C-R function is based on the results of the single pollutant models reported by Weisel et al. (1995, Table 2).

Single Pollutant Model

The coefficient (β) used in the C-R function is a weighted average of the coefficients in Weisel et al. (1995, Table 2) using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma^2_{\beta_i}}}{\sum_{i=1986}^{1990} \frac{1}{\sigma^2_{\beta_i}}} \right) = 0.0443.$$

The standard error of the coefficient (σ_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma^2_\beta = \mathrm{var}\left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma^2_{\beta_i}}}{\sum_{i=1986}^{1990} \frac{1}{\sigma^2_{\beta_i}}} \right) = \left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma^2_{\beta_i}}}{\gamma} \right) = \sum_{i=1986}^{1990} \mathrm{var}\left(\frac{\beta_i}{\sigma^2_{\beta_i} \cdot \gamma} \right).$$

This eventually reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00723.$$

Functional Form: Linear
Coefficient: 0.0443
Standard Error: 0.00723
Baseline Population: baseline population of Northern New Jersey[142] = 4,436,976
Population: population of all ages

[142] The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. (1992, Figure 1) presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

Exhibit G-5. Concentration-Response (C-R) Functions for Ozone and Acute Effects

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Any of 19 Respiratory Symptoms	Krupnick	1990	Los Angeles, CA	18-64	All	All	COH	1-hr max	Linear	0.000137	0.000070
Minor Restricted Activity Days	Ostro and Rothschild	1989	nationwide	18-64	All	All	$PM_{2.5}$	24-hr avg	Log-linear	0.0022	0.000658
School Loss Days, All Cause	Chen et al.	2000	Washoe Co, NV	6-11	All	All	CO, PM_{10}	1-hr max	Linear	0.013247	0.004985
School Loss Days, All Cause	Gilliland et al.	2001	Southern California	9-10	All	All	None	8-hr avg	Log-linear	0.00755	0.004527
Worker Productivity	Crocker and Horst	1981	nationwide	18-64	All	All	None	24-hr avg	Linear	0.14	–

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

G .5 Acute Morbidity

Exhibit 5 summarizes the C-R functions used to estimate the relationship between ozone and acute morbidity. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

G .5.1 Any of 19 Respiratory Symptoms: Krupnick (1990)

Krupnick et al. (1990) estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. (1990) listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included coefficient of haze (COH, a measure of particulate matter concentrations), ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily ozone, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

Multipollutant Model (ozone and coefficient of haze)

The C-R function used to estimate the change in ARD2 associated with a change in daily one-hour maximum ozone[143] is based on Krupnick et al. (1990, p. 12):[144]

$$\Delta ARD2 \cong \beta^* \cdot \Delta O_3 \cdot pop \,,$$

Functional Form: Linear
Coefficient: first derivative of the stationary probability = 0.000137
Standard Error: 0.0000697
Population: population of ages 18-64 years[145]

The logistic regression model used by Krupnick et al. (1990) takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

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[143] Krupnick et al. (1990) used parts per hundred million (pphm) to measure ozone; the coefficient used here is based on ppb.

[144] Krupnick and Kopp (1988, p. 2-24) and ESEERCO (1994, p. V-32) used the same C-R functional form as that used here.

[145] The coefficient estimates are based on the sample of "adults," and assumes that individuals 18 and older were considered adult. According to Krupnick et al. (1990, Table 1), about 0.6 percent of the study sample was over the age of 60. This is a relatively small fraction, so it is further assumed that the results do not apply to individuals 65 years of age and older.

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$p_i = probability(ARD2 | sickness\, or\, not_{t-1}) = \frac{1}{1 - e^{\beta_0 + \beta_1 \cdot ARD2_{t-1} + X \cdot \beta}}, for\ i = 0,1.$$

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of ozone (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in ozone and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before}.$$

However the full suite of coefficient estimates are not available.[146] Rather than use the full suite of coefficient values, the impact of ozone on the probability of ARD2 may be approximated by the derivative of ARD2 with respect to ozone:[147]

$$\frac{\partial probability(ARD2)}{\partial O_3} = \frac{p_0 \cdot (1 - p_1) \cdot \beta \cdot [p_1 + (1 - p_0)]}{(1 - p_1 + p_0)^2} = \beta^*,$$

where β is the reported logistic regression coefficient for ozone. The change in the incidence of ARD2 associated with a given change in ozone is then estimated by:

$$\frac{\partial ARD2}{\partial O_3} \cong \frac{\Delta ARD2}{\Delta O_3}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta O_3} \cong \beta^*$$

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[146] The model without NO_2 (Krupnick et al., 1990, Table V equation 3) was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., Table V). Both the ozone and COH coefficients dropped by about a factor of two or more.

[147] The derivative result is reported by Krupnick et al. (1990, p. 12).

$$\Rightarrow \Delta ARD2 \cong \beta^* \cdot \Delta O_3 \,.$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO (1994, p. V-32) for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$\beta^* = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00055 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000137 \,.$$

The *standard error* for the coefficient is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. (1990, Table V):

$$\beta_{high} = 0.00055 + (1.96 \cdot 0.00027) = 0.00108$$

$$\Rightarrow \beta^*_{high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00108 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000268$$

$$\sigma_{\beta,\,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{(0.000268 - 0.000137)}{1.96} = 0.0000668$$

$$\beta_{low} = 0.00055 - (1.96 \cdot 0.00027) = 0.0000208$$

$$\Rightarrow \beta^*_{low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.0000208 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 5.17 \cdot 10^{-6}$$

$$\Rightarrow \sigma_{\beta,\,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{(0.000137 + 5.17 \cdot 10^{-6})}{1.96} = 0.0000725$$

$$\sigma_\beta = \frac{\sigma_{\beta,\,high} + \sigma_{\beta,\,low}}{2} = 0.0000697 \,.$$

G .5.2 Minor Restricted Activity Days: Ostro and Rothschild (1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national

sample of the adult working population, ages 18 to 65, living in metropolitan areas.[148] The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone had a highly variable association with RRADs and MRADs. Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years. The C-R function for ozone is based on the co-pollutant model with $PM_{2.5}$.

The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to ozone as individuals under 65. A number of studies have found that hospital admissions for the elderly are related to ozone exposures (e.g., Schwartz, 1994b; Schwartz, 1995).

Multipollutant Model (ozone and $PM_{2.5}$)

The coefficient and standard error used in the C-R function are based on a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4). The derivation of these estimates is described below.

Functional Form: Log-linear
Coefficient: 0.00220
Standard Error: 0.000658
Incidence Rate: daily incidence rate for minor restricted activity days (MRAD) = 0.02137 (Ostro and Rothschild, 1989, p. 243)
Population: adult population ages 18 to 64

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight:[149]

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.00220 .$$

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \mathrm{var}\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1976}^{1981} \mathrm{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right) .$$

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[148] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

[149] The calculation of the MRAD coefficient and its standard error is exactly analogous to the calculation done for the work-loss days coefficient based on Ostro (1987).

This reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.000658 .$$

G .5.3 School Loss Days, All Cause (Chen et al., 2000)

Chen et al. (2000) studied the association between air pollution and elementary school absenteeism (grades 1-6)[150] in Washoe County, Nevada. Daily absence data were available for all elementary schools in the Washoe Country School District. The authors regressed daily total absence rate on the three air pollutants, meteorological variables, and indicators for day of the week, month, and holidays. They reported statistically significant associations between both ozone and CO and daily total absence rate for grades one through six. PM_{10} was negatively associated with absence rate, after adjustment for ozone, CO, and meteorological and temporal variables. The C-R function for ozone is based on the results from a multiple linear regression model with CO, ozone, and PM_{10}.

Multipollutant Model (ozone, CO, and PM_{10})

The coefficient and standard error are presented in Table 3 (Chen et al., 2000, p. 1008) for a unit ppm increase in the two-week average of daily one-hour maximum ozone concentration. This is converted to unit ppb increase by dividing by 1,000.

The reported coefficient represents an *absolute* increase in absenteeism rate for a unit increase in ozone. If we apply this study to other locations, we assume that the same absolute increase will occur for a unit increase in ozone, regardless of the baseline rate. If the study location has a particularly high baseline rate, we may be overestimating decreases in absenteeism nationally, and vice-versa. As an example, consider if the baseline absenteeism rate were 10% in the study and 5% nationally. An absolute increase in absence rate of 2% associated with a given increase in ozone reflects a relative increase in absence rate of 20% for the study population. However, in the national estimate, we would assume the same absolute increase of 2%, but this would reflect a relative increase in the absenteeism rate of 40%.

An alternative approach is to estimate apply the *relative* increase in absenteeism rate in the C-R function by adjusting the results by the ratio of the national absenteeism rate to the study-specific rate. As a result, the percent increase in absenteeism rate associated with an increase in ozone is extrapolated nationally rather than the absolute increase in absenteeism rate. The incidence derivation section above describes the data used to estimate national and study-specific absence rates.

In addition to this scaling factor, there are two other scaling factors which are applied to the function. A scaling factor of 0.01 is used to convert the beta from a percentage (x 100) per unit increase of ozone to a proportion per unit increase of ozone. As a result it can be applied directly to the national population of school children ages 6 through 11 to estimate the number of absences avoided.

The final scaling factor adjusts for the number of school days in the ozone season. In the modeling program, the function is applied to every day in the ozone season (May 1 - September 30), however, in

[150] Assuming that most children start kindergarten at age 5, the corresponding ages for grades 1 through 6 would be 6 through 11.

reality, school absences will be avoided only on school days. We assume that children are in school during weekdays for all of May, two weeks in June, one week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7). The C-R function parameters are shown below.

Functional Form: Linear
Coefficient: 0.013247
Standard Error: 0.004985
Population: population of children ages 6-11
Scaling Factor 1: Ratio of national school absence rate to study-specific school absence rate[151] = 1.081
Scaling Factor 2: Convert beta in percentage terms to a proportion = 0.01
Scaling Factor 3: Proportion of days that are school days in the ozone season[152] = 0.393

G .5.4 School Loss Days, All Cause (Gilliland et al., 2001)

Gilliland et al. (2001) examined the association between air pollution and school absenteeism among 4th grade school children (ages 9-10) in 12 southern Californian communities. The study was conducted from January through June 1996. The authors used school records to collect daily absence data and parental telephone interviews to identify causes. They defined illness-related absences as respiratory or non-respiratory. A respiratory illness was defined as an illness that included at least one of the following: runny nose/sneezing, sore throat, cough, earache, wheezing, or asthma attack. The authors used 15 and 30 day distributed lag models to quantify the association between ozone, PM_{10}, and NO_2 and incident school absences. Ozone levels were positively associated with all school absence measures and significantly associated with all illness-related school absences (non-respiratory illness, respiratory illness, URI and LRI). Neither PM_{10} nor NO_2 was significantly associated with illness-related school absences, but PM_{10} was associated with non-illness related absences. The C-R function for ozone is based on the results of the single pollutant model.

Gilliland et al. (2001) defines an incident absence as an absence that followed attendance on the previous day and the incidence rate as the number of incident absences on a given day over the population at risk for an absence on a given day (i.e. those children who were not absent on the previous day). Since school absences due to air pollution may last longer than one day, an estimate of the average duration of school absences could be used to calculated the total avoided school loss days from an estimate of avoided new absences. A simple ratio of the total absence rate divided by the new absence rate would provide an estimate of the average duration of school absences, which could be applied to the estimate of avoided new absences as follows:

$$Duration = \frac{totalAbsences}{newAbsences}$$

$$\Delta\, TotalAbsences = -\left[incidence \cdot \left(e^{-\beta \cdot \Delta O_3} - 1\right)\right] \cdot duration \cdot pop$$

[151] National school absence rate of 5.5% obtained from the U.S. Department of Education (1996, Table 42-1). Study-specific school absence rate of 5.09% obtained from Chen et al. (2000, Table 1).

[152] Ozone is modeled for the 5 months from May 1 through September 30. We assume that children are in school during weekdays for all of May, 2 weeks in June, 1 week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

Since the function is log-linear, the baseline incidence rate (in this case, the rate of new absences) is multiplied by duration, which reduces to the total school absence rate. Therefore, the same result would be obtained by using a single estimate of the total school absence rate in the C-R function. Using this approach, we assume that the same relationship observed between pollutant and new school absences in the study would be observed for total absences on a given day. As a result, the total school absence rate is used in the function below. The derivation of this rate is described in the section on baseline incidence rate estimation.

Single Pollutant Model

For all absences, the coefficient and standard error are based on a percent increase of 16.3 percent (95% CI -2.6 percent, 38.9 percent) associated with a 20 ppb increase in 8-hour average ozone concentration (2001, Table 6, p. 52).

A scaling factor is used to adjust for the number of school days in the ozone season. In the modeling program, the function is applied to every day in the ozone season (May 1 - September 30), however, in reality, school absences will be avoided only on school days. We assume that children are in school during weekdays for all of May, two weeks in June, one week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

In addition, not all children are at-risk for a new school absence, as defined by the study. On average, 5.5% of school children are absent from school on a given day (U.S. Department of Education, 1996, Table 42-1). Only those who are in school on the previous day are at risk for a new absence (1-0.055 = 94.5%). As a result, a factor of 94.5% is used in the function to estimate the population of school children at-risk for a new absence.

Functional Form: Log-linear
Coefficient: 0.007550
Standard Error: 0.004527
Incidence Rate: daily school absence rate = 0.055 (U.S. Department of Education, 1996, Table 42-1)
Population: population of children ages 9-10 not absent from school on a given day[153] = 94.5% of children ages 9-10
Scaling Factor: Proportion of days that are school days in the ozone season[154] = 0.393

G .5.5 Worker Productivity: Crocker and Horst (1981)

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst (1981) and summarized in EPA (1994). Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts as the change in income associated with a change in ozone

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[153] The proportion of children not absent from school on a given day (5.5%) is based on 1996 data from the U.S. Department of Education (1996, Table 42-1).

[154] Ozone is modeled for the 5 months from May 1 through September 30. We assume that children are in school during weekdays for all of May, 2 weeks in June, 1 week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

exposure, given as the elasticity of income with respect to ozone concentration (-0.1427).[155] The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income. Given the national median daily income for outdoor workers engaged in strenuous activity reported by the U.S. Census Bureau (2002), $68 per day (2000$),[156] a ten percent reduction in ozone yields about $0.97 in increased daily wages. We adjust the national median daily income estimate to reflect regional variations in income using a factor based on the ratio of county median household income to national median household income. No information was available for quantifying the uncertainty associated with the central valuation estimate. Therefore, no uncertainty analysis was conducted for this endpoint.

Single Pollutant Model

The C-R function for estimating changes in worker productivity is shown below:

$$\Delta productivity = \beta \cdot \frac{Q_1 - Q_0}{Q_1} \cdot dailyincome \cdot pop \cdot,$$

Functional Form: Linear
Coefficient: 0.1427
Daily Income: median daily income for outdoor workers[157]
Population: population of adults 18 to 64 employed as farm workers

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[155] The relationship estimated by Crocker and Horst between wages and ozone is a log-log relationship. Therefore the elasticity of wages with respect to ozone is a constant, equal to the coefficient of the log of ozone in the model.

[156] The national median daily income for workers engaged in "farming, forestry, and fishing" from the U.S. Census Bureau (2002, Table 621, p. 403) is used as a surrogate for outdoor workers engaged in strenuous activity.

[157] The national median daily income for workers engaged in "farming, forestry, and fishing" was obtained from the U.S. Census Bureau (2002, Table 621, p. 403) and is used as a surrogate for outdoor workers engaged in strenuous activity. This national median daily income ($68) is then scaled by the ratio of national median income to county median income to estimate county median daily income for outdoor workers.

Exhibit G-6. Concentration-Response (C-R) Functions for Ozone and Asthma-Related Effects

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Asthma Exacerbation, Asthma Attacks	Whittemore and Korn	1980	Los Angeles, CA	All	All	All	TSP	1-hr max	Logistic	0.001843	0.000715

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

G .6 Asthma-Related Effects

Exhibit 6 summarizes the C-R functions used to estimate the relationship between ozone and asthma-related effects. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

G .6.1 Asthma Attacks (Whittemore and Korn, 1980)

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants (O_x). Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks. The results from this model were used, and the oxidant result was adjusted so it may be used with ozone data.

Multipollutant Model (ozone and PM_{10})

The daily one-hour ozone coefficient is based on an oxidant coefficient (1.66) estimated from data expressed in ppm. The coefficient is converted to ppb by dividing by 1,000 and to ozone by multiplying by 1.11.[158] The standard error is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

Functional Form: Logistic
Coefficient: 0.001843
Standard Error: 0.000715
Incidence Rate: daily incidence of asthma attacks = 0.0550[159]
Population: population of asthmatics of all ages = 3.86% of the population of all ages (American Lung Association, 2002c, Table 7)

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[158] The study used oxidant measurements in ppm (Whittemore and Korn, 1980, p. 688); these have been converted to ozone measurements in ppb, assuming ozone comprises 90% of oxidants (i.e., 1.11*ozone=oxidant). It is assumed that the harm of oxidants is caused by ozone. The view expressed in the Ozone Staff Paper (U.S. EPA, 1996a, p.164) is consistent with assuming that ozone is the oxidant of concern at normal ambient concentrations: "Further, among the photochemical oxidants, the acute-exposure chamber, field, and epidemiological human health data base raises concern only for ozone at levels of photochemical oxidants commonly reported in ambient air. Thus, the staff recommends that ozone remain as the pollutant indicator for protection of public health from exposure to all photochemical oxidants found in the ambient air."

[159] Based on an analysis of the 1999 National Health Interview Survey, the daily incidence of wheezing attacks for adult asthmatics is estimated to be 0.0550. In the same survey, wheezing attacks for children were examined, however, the number of wheezing attacks per year were censored at 12 (compared to censoring at 95 for adults). Due to the potential for underestimation of the number of children's wheezing attacks, we used the adult rate for all individuals.

Appendix H: Pooling

This Appendix presents a discussion part of which has been published previously (Abt Associates Inc., 1996a; Abt Associates Inc., 1996b). It is repeated here to provide a complete documentation of all the issues involved in the derivation of C-R functions used in benefits analysis.

Many studies have attempted to determine the influence of particulate matter and/or ozone pollution on human health. Usually this involves estimation of a parameter β in a concentration-response function. Each study provides an estimate of β, along with a measure of the uncertainty of the estimate. Because uncertainty decreases as sample size increases, combining data sets is expected to yield more reliable estimates of β. Combining data from several comparable studies in order to analyze them together is often referred to as meta-analysis.

For a number of reasons, including data confidentiality, it is often impractical or impossible to combine the original data sets. Combining the *results* of studies in order to produce better estimates of β provides a second-best but still valuable way to synthesize information (DerSimonian and Laird, 1986). This is referred to as pooling results in this report. Pooling β's requires that all of the studies contributing estimates of β use the same functional form for the concentration-response function. That is, the β's must be measuring the same thing.

It is also possible to pool the study-specific estimates of incidence change derived from the C-R functions, instead of pooling the underlying β's themselves. For a variety of reasons, this is often possible when it is not feasible to pool the underlying β's. For example, if one study is log-linear and another is linear, we could not pool the β's because they are not different estimates of a coefficient in the same C-R function, but are instead estimates of coefficients in different C-R functions. We can, however, calculate the incidence change predicted by each C-R function (for a given change in pollutant concentration and, for the log-linear function, a given baseline incidence rate), and pool these incidence changes.

Whether we are pooling the β's in the C-R functions, the incidence changes predicted by the C-R functions, or the monetary values of the incidence changes, a key component of the pooling process is the determination of the weight given to each study. Below we discuss possible weighting schemes, with particular emphasis on the fixed effects/random effects models, used in the current analysis. The discussion is first presented in terms of pooling the β's, because that most closely matches the discussion of the method for pooling study results as it was originally presented by DerSimonian and Laird (1986). We then give a brief overview of the analogous weighting process if study-specific incidence changes are being pooled rather than β's. The discussion refers to PM for ease of exposition. It is applicable to any pollutant, however.

H.1 Background Considerations: Underlying Assumptions

It is possible that the concentration-response relationship between PM and an adverse health effect is the same everywhere. If this is the case, different estimates of β reported by different epidemiological studies are all estimates of the same underlying parameter and differ from each other only because of sampling error. A more general and a more plausible model, however, is that there is not just a single concentration-response relationship between PM and the health effect, but that this relationship varies from one location to another, depending on such factors as the composition of the PM and the composition of the exposed population.

If there is an underlying distribution of β's, then differences in reported estimates of β among studies carried out on a *single* population in a single location (using identical averaging times, methodology, etc.) would reflect only sampling error, because all such studies are estimating the same β. Differences in reported estimates among studies carried out on different populations in different locations, however, may also reflect differences in the β's being estimated. There are, then, two potential sources of variability among concentration-response estimates:

(1) within-location variability, or sampling error (so that even two studies estimating the same β in the same location are likely to report different estimates), and

(2) between-location variability derived from the fact that studies may be estimating different underlying parameter values in different locations,

and associated with these two sources of variability is uncertainty about the correct concentration-response function for a given location. The basic model assumption – that there is a single β in all locations versus a distribution of β's across locations – underlies the difference between the fixed and random effects models, discussed below.

H.2 Alternative Methods to Pool Estimates of β

One method of pooling study results is simply averaging all reported β's. This has the advantage of simplicity, but the disadvantage of not taking into account the uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty.

For example, consider the three studies whose results are presented in Exhibit H-1.

Exhibit H-1. Three Sample Studies

Study	Estimate of β	Standard Deviation	Variance
Study 1	0.75	0.35	0.1225
Study 2	1.25	0.05	0.0025
Study 3	1.00	0.10	0.0100

The average of the three estimates is 1.0. However, the study 2 estimate has much less uncertainty associated with it (variance = 0.0025) than either the study 1 or study 3 estimates. It seems reasonable that a pooled estimate which combines the estimates from all three studies should therefore give more weight to the estimate from the second study than to the estimates from the first and third studies.

H.2.1 Inverse Variance Weighting

A common method for weighting estimates involves using their variances. Variance takes into account both the consistency of data and the sample size used to obtain the estimate, two key factors that influence the reliability of results. The exact way in which variances are used to weight the estimates from different studies in a pooled estimate depends on the underlying model assumed.

H.2.2 Fixed Effects Model

The fixed effects model assumes that there is a single true concentration-response relationship and therefore a single true value for the parameter β. Differences among β's reported by different studies are therefore simply the result of sampling error. That is, each reported β is an estimate of the *same underlying parameter.* The certainty of an estimate is reflected in its variance (the larger the variance, the less certain the estimate). Pooling that assumes a fixed effects model therefore weights each estimate under consideration in proportion to the *inverse* of its variance.

Suppose there are n studies, with the ith study providing an estimate β_i with variance v_i (I = 1, ..., n). Let

$$S = \Sigma \frac{1}{v_i},$$

denote the sum of the inverse variances. Then the weight, w_i, given to the ith estimate, β_i, is

$$w_i = \frac{1/v_i}{S}.$$

This means that estimates with small variances (i.e., estimates with relatively little uncertainty surrounding them) receive large weights, and those with large variances receive small weights.

The estimate produced by pooling based on a fixed effects model, then, is just a weighted average of the estimates from the studies being considered, with the weights as defined above. That is,

$$\beta_{fe} = \Sigma\, w_i * \beta_i .$$

The variance associated with this pooled estimate is the inverse of the sum of the inverse variances:

$$v_{fe} = \frac{1}{\Sigma\, 1/v_i}.$$

Exhibit H-2 shows the relevant calculations for this pooling for the three sample studies summarized in Exhibit H-1.

Exhibit H-2. Fixed Effects Model Calculations

Study	β_i	v_i	$1/v_i$	w_i	$w_i*\beta_i$
1	0.75	0.1225	8.16	0.016	0.012
2	1.25	0.0025	400	0.787	0.984
3	1.00	0.0100	100	0.197	0.197
Sum			∑ = 508.16	∑ = 1.000	∑ = 1.193

The sum of weighted contributions in the last column is the pooled estimate of β based on the fixed effects model. This estimate (1.193) is considerably closer to the estimate from study 2 (1.25) than is the estimate (1.0) that simply averages the study estimates. This reflects the fact that the estimate from study 2 has a much smaller variance than the estimates from the other two studies and is therefore more heavily weighted in the pooling.

The variance of the pooled estimate, v_{fe}, is the inverse of the sum of the variances, or 0.00197. (The sums of the β_i and v_i are not shown, since they are of no importance. The sum of the $1/v_i$ is S, used to calculate the weights. The sum of the weights, w_i , I=1, ..., n, is 1.0, as expected.)

H.2.3 Random Effects Model

An alternative to the fixed effects model is the random effects model, which allows the possibility that the estimates β_i from the different studies may in fact be estimates of *different* parameters, rather than just different estimates of a single underlying parameter. In studies of the effects of PM_{10} on mortality, for example, if the composition of PM_{10} varies among study locations the underlying relationship between mortality and PM_{10} may be different from one study location to another. For example, fine particles make up a greater fraction of PM_{10} in Philadelphia than in El Paso. If fine particles are disproportionately responsible for mortality relative to coarse particles, then one would expect the true value of β in Philadelphia to be greater than the true value of β in El Paso. This would violate the assumption of the fixed effects model.

The following procedure can test whether it is appropriate to base the pooling on the random effects model (vs. the fixed effects model):

A test statistic, Q_w , the weighted sum of squared differences of the separate study estimates from the pooled estimate based on the fixed effects model, is calculated as:

$$Q_w = \Sigma_i \frac{1}{v_i} (\beta_{fe} - \beta_i)^2.$$

Under the null hypothesis that there is a single underlying parameter, β, of which all the β_i 's are estimates, Q_w has a chi-squared distribution with n-1 degrees of freedom. (Recall that n is the number of studies in the meta-analysis.) If Q_w is greater than the critical value corresponding to the desired confidence level, the null hypothesis is rejected. That is, in this case the evidence does not support the fixed effects model, and the random effects model is assumed, allowing the possibility that each study is estimating a different β.

The weights used in a pooling based on the random effects model must take into account not only the within-study variances (used in a meta-analysis based on the fixed effects model) but the between-study variance as well. These weights are calculated as follows:

Using Q_w , the between-study variance, η^2, is:

$$\eta^2 = \frac{Q_w - (n-1)}{\Sigma 1/v_i - \frac{\Sigma 1/v_i^2}{\Sigma 1/v_i}} .$$

It can be shown that the denominator is always positive. Therefore, if the numerator is negative (i.e., if $Q_w < n-1$), then η^2 is a negative number, and it is not possible to calculate a random effects estimate. In this case, however, the small value of Q_w would presumably have led to accepting the null hypothesis described above, and the meta-analysis would be based on the fixed effects model. The remaining discussion therefore assumes that η^2 is positive.

Given a value for η^2 , the random effects estimate is calculated in almost the same way as the fixed effects estimate. However, the weights now incorporate both the within-study variance (v_i) and the between-study variance (η^2). Whereas the weights implied by the fixed effects model used only v_i, the within-study variance, the weights implied by the random effects model use $v_i + \eta^2$.

Let $v_i^* = v_i + \eta^2$. Then

$$S^* = \Sigma \frac{1}{v_i^*} ,$$

and

$$w_i^* = \frac{1/v_i^*}{S^*} .$$

The estimate produced by pooling based on the random effects model, then, is just a weighted average of the estimates from the studies being considered, with the weights as defined above. That is,

$$\beta_{rand} = \Sigma\, w_i^* * \beta_i .$$

The variance associated with this random effects pooled estimate is, as it was for the fixed effects pooled estimate, the inverse of the sum of the inverse variances:

$$v_{rand} = \frac{1}{\Sigma\, 1/v_i^*} .$$

The weighting scheme used in a pooling based on the random effects model is basically the same as that used if a fixed effects model is assumed, but the variances used in the calculations are different. This is because a fixed effects model assumes that the variability among the estimates from different studies is due only to sampling error (i.e., each study is thought of as representing just another sample from the same underlying population), while the random effects model assumes that there is not only sampling error associated with each study, but that there is also *between-study* variability -- each study is estimating a different underlying β. Therefore, the sum of the within-study variance and the between-study variance yields an overall variance estimate.

H.2.4 Fixed Effects and Random Effects Weighting to Pool Study-Specific Incidence Changes

Weights can be derived for pooling incidence changes predicted by different studies, using either the fixed effects or the random effects model, in a way that is analogous to the derivation of weights for

pooling the β's in the C-R functions. For a given change in pollutant level and a given baseline incidence rate, corresponding to every possible value of β, there is an incidence change. Corresponding to β_i, with variance v_i (calculated from the reported standard error of β_i,) from the ith study, there is therefore an estimate of incidence change, I_i, with variance $v(I)_i$. In practice, we generate a sample mean and a sample variance of incidence changes by calculating an incidence change for each of many β's pulled from the distribution of β's for the study.

This can be done either using Monte Carlo methods (making many random pulls) or by a Latin Hypercube approach, in which we pull the nth percentile β from the distribution of β's, for, e.g., n = 2.5, 7.5, ..., 97.5. Either way, the result is a corresponding sample distribution of incidence changes that would be predicted by the study, from which we calculate the sample mean and the sample variance. The sample means of incidence change from the studies to be pooled are used in exactly the same way as the reported β's are used in the discussion of fixed effects and random effects models above. The sample variances of incidence change are used in the same way as the variances of the β's. The formulas above for calculating fixed effects weights, for testing the fixed effects hypothesis, and for calculating random effects weights can all be used by substituting the sample mean incidence change for the ith study for β_i and the sample variance of incidence change for the ith study for v_i.