

Diesel Emissions: Particulate Matter-Related Health Damages

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Project Manager:
Conrad Schneider

Prepared by
Abt Associates Inc.
4800 Montgomery Lane
Bethesda, MD 20814-5341

with
Computer Sciences Corporation
and
E.H. Pechan Associates, Inc.

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1. Introduction

Over the past decade and more, hundreds of studies worldwide have linked particulate matter to a wide range of adverse health effects in people of all ages, including premature death, chronic bronchitis, hospital admissions and asthma. While this large body of research has yet to establish a conclusive cause-and-effect relationship between PM and adverse health effects, additional recent research identifying plausible biological mechanisms provides strong evidence that reducing ambient PM concentrations will lead to improvements in human health.

The US EPA developed analytical methods that draw on this health research, combined with estimates of future air pollution emissions and air quality models, to prepare quantified estimates of the avoidable health effects from improving ambient PM levels. The EPA used these analytical methods to estimate the health benefits of a wide variety of actual or proposed individual federal air programs, including programs that reduce emissions from power plants, cars, and both on-road and off-road diesel engines. We used these same analytical methods that the U.S. Environmental Protection Agency used in 2003 to prepare an analysis of the potential health effects of the proposed Clear Skies Act (U.S. EPA, 2003).

This report estimates the avoidable health effects of *eliminating* diesel emissions in 2010, focusing on the adverse human health effects due to exposure to fine particulate matter (PM_{2.5}, which are particles less than 2.5 microns in diameter). This “No Diesel” analysis is clearly not a policy option, but rather helps gain a better understanding of the total magnitude of the health effects associated with this major source of PM.

Chapter 2 describes the emissions inventory estimates. Chapter 3 describes the methods used to estimate changes in particulate matter concentrations. Chapter 4 describes general issues arising in estimating and valuing changes in adverse health effects associated with changes in particulate matter. Chapter 5 describes in some detail the methods used for estimating and valuing adverse health effects, and in Chapter 6 we present the results of these analyses. Finally, Appendix A presents a derivation of the particulate matter concentration-response functions used in this report.

2. Emissions Inventory

The detailed estimates of the future emissions inventory used in this analysis is the same inventory EPA(2003) used in their analysis of the Clear Skies Act. In order to conduct an analysis of changes in the levels of ambient PM_{2.5} in the atmosphere from changes in emissions from on-road and non-road diesel sources, it is necessary to have an estimate of the complete inventory from all sources of precursor emissions, not just the emissions from the source categories. EPA prepared the complete estimated emissions inventory for 2010 necessary to conduct a PM air quality analysis.

This inventory includes emissions from not only on-road and non-road diesel sources, but also other large industrial sources, all mobile sources, smaller “Area” emission sources ranging from gasoline stations to household emissions, agricultural emissions, and naturally occurring emissions from forests, grasslands, etc. The location and timing of emissions have an important impact on PM formation, so the emissions inventory includes extensive detail on the location and timing of the estimated emissions. Canadian and Gulf of Mexico sources are included in the inventory as well, as these pollutants effect PM levels in the continental US.

2.1 Summary of the National Emissions Inventory

There are six air pollutant emissions that are used to model PM concentrations: oxides of nitrogen (NO_x), volatile organic compounds (VOC), ammonia (NH₃), sulfur dioxide (SO₂), direct fine particle emissions (PM_{2.5}), direct coarse particle emissions (PM₁₀), and primary elemental carbon (PMC). Exhibit 2-1 summarizes the estimated total emissions in the continental United States in 2010 for the six precursor air pollutants.

Exhibit 2-1. 2010 Baseline Emissions Inventory (Tons/Year)

Source	NOx	VOC	NH3	SO2	PM10	PM2.5	PMC
EGU	3,943,438	32,660	1,783	9,856,926	217,623	109,983	107,640
Other Industrial	3,221,605	1,707,062	284,824	3,799,164	1,015,052	605,692	409,359
On Road	4,931,951	2,824,715	322,961	29,780	178,649	113,771	64,879
Non Road	3,409,824	2,016,276	49,964	252,924	286,189	243,085	43,104
Area	2,225,898	7,221,877	4,341,905	1,367,643	7,693,802	2,285,814	5,407,988
Total US	17,732,716	13,802,589	5,001,437	15,306,437	9,391,315	3,358,345	6,032,971
Canada & Gulf of Mexico	1,972,010	2,550,200	555,496	1,901,396	1,887,887	419,719	1,468,168
Total Modeled	19,704,726	16,352,789	5,556,933	17,207,833	11,279,202	3,778,064	7,501,139

3. Air Quality Modeling

The analysis used results from the Regulatory Modeling System for Aerosols and Acid Deposition (REMSAD, ver 7.06) to forecast changes in the ambient concentration of both PM₁₀ and PM_{2.5} at the REMSAD grid cell level. Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is useful for evaluating the air-quality effects of emission control scenarios.

Modeling future air quality anticipated to result from policy-driven emissions changes is extremely difficult and inherently uncertain. Alternative air quality models inevitably produce differing results. Scientific understanding of the complex atmospheric processes involved with PM formation and transport is increasing rapidly. The new PM_{2.5} monitoring data now being collected nationwide, and improvements in the estimates of emissions from all sources, will help calibrate and verify the performance of air quality models. Existing air quality models are being improved constantly, and the next generation of PM air quality models are under development.

3.1 Particulate Matter Formation

Ambient concentrations of PM are composed of directly emitted particles and of secondary aerosols of sulfate, nitrate, and organics. Particulate matter is the generic term for the mixture of microscopic solid particles and liquid droplets found in the air. The particles are either emitted directly from these combustion sources or are formed in the atmosphere through reactions involving gases, such as SO₂ and NO_x.

3.1.1 REMSAD Air Quality Model

REMSAD was used to simulate estimates of particulate matter concentration for three future-year scenarios. Computer Sciences Corporation (CSC) performed the REMSAD modeling for this report. Subsequently we used the modeling results to estimate the health-related costs for each of the scenarios in the primary analysis.

The REMSAD model is designed to simulate the effects of changes in emissions on PM concentrations and deposition. REMSAD calculates concentrations of pollutants by simulating the physical and chemical processes in the atmosphere. The basis for REMSAD is the atmospheric diffusion or species continuity equation. This equation represents a mass balance that includes all of the relevant emissions, transport, diffusion, chemical reactions, and removal processes in mathematical terms.

Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD can evaluate the air-quality effects of specific emission control scenarios. This is achieved by first replicating a historical episode to establish a base-case simulation. CSC prepared model inputs from observed meteorological, emissions, and air quality data for selected episode days using various input preparation techniques. They applied the REMSAD model with these inputs, and the results are evaluated to determine model performance. Once the model results have been evaluated and determined to perform within prescribed levels, they combined the same base-case meteorological inputs with *modified* or *projected* emission inventories to simulate possible alternative/future emission scenarios.

The PM levels estimated by REMSAD were not directly used in EPA's (2003) health analysis of the Clear Skies Act, nor are they directly used here. Instead of using the REMSAD results directly, we

use the REMSAD results to estimate the relative change in PM levels. We combined the REMSAD results with actual PM_{2.5} monitor readings from 2001 to estimate the PM_{2.5} levels actually used in the health analysis. This same procedure was used in the EPA Clear Skies Act health analysis.

At the location of each PM_{2.5} monitor, we quantified the relationship between REMSAD estimated levels of PM_{2.5} at the monitor for the base year (2001) and the future year (2010). These REMSAD-based adjustment ratios are applied to the actual monitoring data to generate estimates of PM_{2.5} levels at each monitor for the future scenario.

In order to provide estimates of ambient PM_{2.5} levels everywhere in the country, and not just at the monitors, an additional analytical step is required. To calculate population exposure to PM, each REMSAD grid cell was assigned a distance-weighted average of adjusted PM levels from a set of monitors that best surrounds the cell. This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (eVNA) spatial interpolation.¹

¹ See Abt Associates (2003) for a more detailed description of eVNA.

4. Issues in Estimating Health Benefits

Changes in PM levels result in changes in a number of health effects, or “endpoints,” that society values. Section 1 of this Chapter summarizes the form of the C-R functions that we use to estimate adverse health effects. Section 2 describes general issues in valuing health changes. And Section 3 discusses how uncertainty is characterized in this analysis.

4.1 Estimating Adverse Health Effects

While several health endpoints have been associated with exposure to ambient PM, the discussion below refers only to a generic “health endpoint,” denoted as y . The discussion refers to estimation of changes in the incidence of the health endpoint at a single location (the population cell, which is equivalent to the REMSAD gridcell). Region-wide changes are estimated by summing the estimated changes over all population cells in the region.

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).² Finally, one study may have considered changes in the health endpoint 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.³ 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

² 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), assumed 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.

³ The International Classification Codes are described at the website of the Medical Center Information Systems: Duke University Health Systems.

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 health effects are the log-linear and the linear relationship. The log-linear relationship is of the form:

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

or, equivalently,

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

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 $\alpha = \ln(B)$.⁴ If the functional form of the C-R relationship is log-linear, the relationship between ΔPM and Δy is:

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

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:

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

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

⁴ 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, $B = B_0 \exp\{\beta_1 x_1 + \dots + \beta_n x_n\}$, where B_0 is the incidence of y when all covariates in the model are zero, and x_1, \dots, 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.

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. Abt Associates (1999, Appendix A) provides further details on the derivation of dose-response functions.

4.1.1 Calculation of Adverse Health Effects with BenMAP

The health effects analysis in this report was prepared using BenMAP (ver 1.0), which was developed by Abt Associates Inc. for the US EPA.⁵ Although BenMAP is still being revised and expanded, the same version of BenMAP was used in this analysis as was used for EPA's analysis in 2003 of the Clear Skies Act. BenMAP is a population-based system for modeling exposure to ambient levels of criteria air pollutants and estimating the adverse health effects associated with this exposure. BenMAP uses the same grid cell configuration as REMSAD ver 7.06 (36km x 36km), and estimates the changes in incidence of adverse health effects associated with given changes in air quality in each grid cell. The national incidence change (or the changes within individual states or counties) is then calculated as the sum of grid-cell-specific changes.

To reflect the uncertainty surrounding predicted incidence changes resulting from the uncertainty surrounding the pollutant coefficients in the C-R functions used, BenMAP produces a *distribution* of possible incidence changes for each adverse health, rather than a single point estimate. To do this, it uses both the point estimate of the pollutant coefficient (β in the above equation) and the standard error of the estimate to produce a normal distribution with mean equal to the estimate of β and standard deviation equal to the standard error of the estimate. Using a Latin Hypercube method,⁶ we take the n^{th} percentile value of β from this normal distribution, for $n = 0.5, 1.5, \dots, 99.5$, and follow the procedure outlined in the section above to produce 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 BenMAP grid cell. This distribution is stored, and BenMAP proceeds to the next grid cell, where the process is repeated. We calculate the distribution of the national change (or change in a designated geographical area) by summing the n^{th} percentile grid cell-specific changes, for $n = 0.5, 1.5, \dots, 99.5$.

4.2 Valuing Changes in Health Effects

This section discusses two of the issues that arise in valuing changes in health effects. The first sub-section discusses the possibility that as income changes then WTP would also change. The second

⁵ See the following website in order to download BenMAP (ver 1.0): <http://www.epa.gov/ttn/ecas/benmapdownload.html>.

⁶ 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, ppl 104-105).

sub-section describes how we adjust the original WTP estimates dollars to correct for inflation to get estimates in 1999 dollars. The WTP estimates were originally calculated in a variety of different years, and hence reflect values in values expressed in the a variety of different inflation amounts.

4.2.1 Change Over Time in WTP in Real Dollars

The WTP for health-related environmental improvements (in real dollars) could change between now and 2010. If real income increases between now and the year 2010, for example, it is reasonable to expect that WTP, in real dollars, would also increase. We follow EPA’s (2003) approach for adjusting for income growth.

4.2.2 Adjusting Benefits Estimates to Year 1999 Dollars

This section describes the methods used to convert benefits estimates to constant 1999 dollars. This is necessary because some of the WTP estimates that we use are measured in dollars from different years. The method that we use depends on the basis of the benefits estimates. Exhibit 4-1 delineates these bases.

Exhibit 4-1. Bases of Benefits Estimation

Basis of Benefit Estimation	Benefit Endpoints
Cost of illness	Hospital admissions avoided
Direct estimates of WTP	Statistical lives saved Chronic bronchitis Morbidity endpoints using WTP
Earnings	Work loss days (WLDs) avoided

Benefits estimates based on cost-of-illness have been adjusted by using the consumer price indexes (CPI-U) for medical care. Because increases in medical costs have been significantly greater than the general rate of inflation, using a general inflator (the CPI-U for “all items” or some other general inflator) to adjust from previous year dollars to 1999 dollars would downward bias cost-of-illness estimates in 1999 dollars.

Benefits estimates based directly on estimates of WTP have been adjusted using the CPI-U for “all items.” The CPI-U, published by the U.S. Dept. of Labor, Bureau of Labor Statistics, can be found in Council of Economic Advisers (2000). An overview of the adjustments from 1990 to 1999 dollars for WTP-based and cost-of-illness based valuations is given in Exhibit 4-2.

Exhibit 4-2. Consumer Price Indexes Used to Adjust WTP-Based and Cost-of-Illness-Based Benefits Estimates from 1990 Dollars to 1999 Dollars

	1990 (1)	1999 (2)	Adjustment Factor ^a (2)/(1)	Relevant Endpoints
CPI-U for "All Items" ^b	130.7	166.6	1.275	<u>WTP-based valuation:</u> 1. Statistical lives saved ^c 2. Chronic bronchitis 3. Morbidity endpoints using WTP ^d
CPI-U for Medical Care ^b	162.8	250.6	1.539	<u>Cost-of-illness based valuation:</u> Hospital admissions avoided ^e

^a Benefits estimates in 1990 dollars are multiplied by the adjustment factor to derive benefits estimates in 1999 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. (1992) using the CPI-U for "all items."

^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 Adjustments of cost-of-illness based estimates of all hospital admissions avoided to 1990 \$ were made by Abt Associates Inc. in previous analyses, such as the NAAQS RIA (U.S. EPA, 1997a).

Benefit estimates for work loss days (WLDs) avoided have in past analyses been based on either the mean or median daily wage. For this analysis, the valuation of the benefit of avoiding a work loss day used the median daily income rather than the mean, consistent with economic welfare theory. The income distribution in the United States is highly skewed, so that the mean income is substantially larger than the median income. However, the incomes of those individuals who lose work days due to pollution are not likely to be a random sample from this income distribution. In particular, the probability of being drawn from the upper tail of the distribution is likely to be substantially less than the probability mass in that tail. To reflect this likelihood, we used the median income rather than the mean income as the value of a work loss day. (This is explained more fully below in the section on valuing work loss days.) To adjust the 2000 county-level estimates, we adjust with the employment cost index from the U.S. Department of Labor, Bureau of Labor Statistics.

4.3 Characterization of Uncertainty

In any complex analysis using estimated parameters and inputs from numerous different models, there are likely to be many sources of uncertainty. This analysis is no exception. There are many inputs that are used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological estimates of C-R functions, estimates of values (both from WTP and cost-of-illness studies), population estimates, income estimates, and estimates of the future state of the world, i.e. regulations, technology, and human behavior. Each of these inputs may be uncertain, and depending on 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.

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 PM₁₀ when PM_{2.5} is not available, excluded variables, and simplification of complex functions
- biases due to omissions or other research limitations.

A final approach to measuring uncertainty is through probabilistic assessments where statistical uncertainty bounds are calculated for each endpoint. We discuss statistical uncertainty bounds in the following section.

4.3.1 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 incidence changes (deriving from uncertainty about 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).⁷ Based on these distributions, we provide estimates of the 5th and 95th percentile values of the distribution of estimated benefits. However, we hasten to add that this omits important sources of uncertainty, such as the contribution of air quality changes, baseline population incidences, projected populations exposed, transferability of the C-R function to diverse locations, and uncertainty about premature mortality. Thus, a confidence interval based on the standard error would provide a misleading picture about the overall uncertainty in the estimates. The empirical evidence about uncertainty is presented where it is available.

Both the uncertainty about the incidence changes and uncertainty about unit dollar values can be characterized by *distributions*. Each “uncertainty distribution” characterizes our beliefs about what the true value of an unknown (e.g., the true change in incidence of a given health effect) is likely to be, based on the available information from relevant studies.⁸ 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.

⁷ 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^* .

⁸ 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).

The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from these two sources, and is estimated with a Monte Carlo method. In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution and a value is randomly drawn from the unit dollar value distribution, and the total dollar benefit for that iteration is the product of the two.⁹ 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.

⁹ This method assumes that the incidence change and the unit dollar value for an endpoint are stochastically independent.

5. Health Benefits

The most significant monetized benefits of reducing ambient concentrations of PM are attributable to reductions in health risks. 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 PM-based C-R functions. The changes in incidence of PM-related adverse health effects and corresponding monetized benefits associated with these changes are estimated separately. Exhibit 5-1 presents the PM-related health endpoints included in this analysis, and Exhibit 5-2 presents the unit monetary values for each of these endpoints and associated uncertainty distributions. Appendix A presents the functional forms for each C-R function and their derivation.

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.

Exhibit 5-1 PM-Related Health Endpoints

Endpoint	Population	PM Measure	Study
Premature Mortality associated with long-term exposure	Ages 30+	PM _{2.5}	Pope et al (2002)
Chronic Bronchitis	Ages 27+	PM _{2.5}	Abbey et al. (1995c)
Acute Myocardial Infarction (Non-fatal)	Ages 18+	PM _{2.5}	Peters et al. (2001)
Hospl Admis., Chronic Lung Disease Less Asthma (ICD codes 490-492, 494-496)	Ages 18-64	PM _{2.5}	Moolgavkar (2000b)
Hospl Admis., Asthma (ICD code 493)	< 65	PM _{2.5}	Sheppard et al. (1999)
Hospl Admis., Pneumonia (ICD-9 codes 480-487)	Ages 65+	PM _{2.5}	Lippman et al. (2000)
Hospl Admis., Chronic Lung Disease (ICD codes 490-496)	Ages 65+	PM _{2.5}	Pooled Estimate: Lippman et al. (2000), Moolgavkar (2000b)
Hospl Admis., Cardiovascular (ICD codes 390-409, 411-429)	Ages 20-64	PM _{2.5}	Moolgavkar (2000a)
Hospl Admis., Cardiovascular ((ICD codes 390-409, 411-429)	age 65+	PM _{2.5}	Pooled Estimate: Moolgavkar (2000a), Lippman et al. (2000)
Asthma-related ER visits (ICD code 493)	< 18	PM _{2.5}	Norris et al. (1999)
Acute bronchitis	Ages 8-12	PM _{2.5}	Dockery et al. (1996)
Lower respiratory symptoms (LRS)	Ages 7-14	PM _{2.5}	Schwartz (1994)
Upper respiratory symptoms (URS)	Asthmatics, ages 9-11	PM ₁₀	Pope et al. (1991)
Minor restricted activity day (MRAD)	Ages 18-64	PM _{2.5}	Ostro and Rothschild (1989)
Work loss days (WLDs)	Ages 18-64	PM _{2.5}	Ostro (1987)

Exhibit 5-2. Unit Values for Economic Valuation of Health Endpoints (1999 \$)

Health Endpoint	Mean Estimate ^a	Uncertainty Distribution ^a												
Mortality														
Value of a statistical life	\$6.12 million per statistical life ^b	Weibull distribution, mean = \$6.12 million; std. dev. = \$4.13 million.												
Chronic Bronchitis														
WTP approach	\$331,000 per case	A Monte Carlo-generated distribution, based on three underlying distributions.												
Heart Attacks														
Acute Myocardial Infarction (Non-fatal)	<table style="margin-left: 20px; border-collapse: collapse;"> <thead> <tr> <th style="text-align: left; border-bottom: 1px solid black;">Age</th> <th style="text-align: left; border-bottom: 1px solid black;">Per Case</th> </tr> </thead> <tbody> <tr> <td>18-24</td> <td>\$63,325</td> </tr> <tr> <td>25-44</td> <td>\$71,755</td> </tr> <tr> <td>45-54</td> <td>\$75,751</td> </tr> <tr> <td>55-64</td> <td>\$135,148</td> </tr> <tr> <td>65+</td> <td>\$63,325</td> </tr> </tbody> </table>	Age	Per Case	18-24	\$63,325	25-44	\$71,755	45-54	\$75,751	55-64	\$135,148	65+	\$63,325	None available
Age	Per Case													
18-24	\$63,325													
25-44	\$71,755													
45-54	\$75,751													
55-64	\$135,148													
65+	\$63,325													
Hospital Admissions														
Chronic Lung Disease Less Asthma(ICD codes 490-492, 494-496) (Ages 20-64)	\$11,333 per admission	None available. ^c												
Asthma (ICD code 493)	\$7,467 per admission	None available. ^c												
Pneumonia (ICD codes 480-487) (Ages 65+)	\$17,106 per admission	None available. ^c												
Chronic Lung Disease (ICD codes 490-496) (Ages 65+)	\$13,083 per admission	None available. ^c												
Cardiovascular (ICD codes 390-429)	<table style="margin-left: 20px; border-collapse: collapse;"> <thead> <tr> <th style="text-align: left; border-bottom: 1px solid black;">Age</th> <th style="text-align: left; border-bottom: 1px solid black;">Per Case</th> </tr> </thead> <tbody> <tr> <td>65+</td> <td>\$20,344</td> </tr> <tr> <td>20-64</td> <td>\$21,864</td> </tr> </tbody> </table>	Age	Per Case	65+	\$20,344	20-64	\$21,864	None available. ^c						
Age	Per Case													
65+	\$20,344													
20-64	\$21,864													
Asthma-related ER visits	\$275 per visit	None available.												
Respiratory Ailments Not Requiring Hospitalization														
Acute bronchitis	\$344 per case	None available.												
Lower resp. Symptoms	\$15.30 per symptom-day	Continuous uniform distribution over [\$6.37, \$24.22].												
Upper resp. Symptoms	\$24.23 per symptom-day	Continuous uniform distribution over [\$8.93,\$42.06].												
Minor respiratory activity day (MRAD)	\$48.43 per day	Triangular distribution centered at \$48.43 over [\$20.34, \$77.76].												
Work loss days	\$106 per day	None available.												

^a The derivation of each of the estimates is discussed in the text.

^b An adjustment for lagged mortality, discussed in the text, is used in this analysis. The lag-adjusted value of a statistical life is approximately 95% of the full value presented here, assuming a three percent discount rate.

^c Standard errors were not available for hospital admission costs. However, the sample sizes on which these estimates (from the Agency for Healthcare Research and Policy's Healthcare Cost and Utilization Project) are very large and the standard errors are therefore negligible.

5.1 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, a substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates.

5.1.1 Estimating PM-Related Premature Mortality

Pope et al (2002) extends previously published results based on the American Cancer Society cohort tracking data. EPA's (2003) Clear Skies Analysis did not include this study because of its recent publication, however EPA's (2004) analysis of reductions in non-road emissions included this new work by Pope et al. For this analysis, we have used the PM_{2.5}-related all-cause mortality estimate from this new work by Pope et al. In particular, we used the effects estimate based on the estimated average exposure in the study period.

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 (EPA-SAB-COUNCIL-ADV-00-001, 1999). 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 distribution of avoided incidences of PM-related mortality.

5.1.2 Valuing Premature Mortality

The "statistical lives lost" approach to valuing premature mortality estimates the value of a statistical death to be \$6.12 million (in 1999 \$). We assume for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the five years following exposure (the five-year mortality lag). To take this into account in the valuation of reductions in premature mortalities, we apply an annual three percent discount rate to the value of premature mortalities occurring in future years.

The "statistical lives lost" value of \$6.12 million represents an intermediate value from a variety of estimates that appear in the economics literature, and is a value that EPA has frequently used. This estimate is the mean of a distribution fitted to the estimates from 26 value-of-life studies identified in the §812 study as "applicable to policy analysis." The approach and set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria used by Viscusi in his review of value-of-life studies. The \$6.12 million estimate is consistent with Viscusi's conclusion (updated to 1999 \$) that "most of the reasonable estimates of the value of life are clustered in the \$3.84 to \$8.93 million range." Uncertainty associated with the valuation of premature mortality is expressed through a Weibull distribution with a standard deviation of \$4.13 million (IEc 1992, p. 2).

5.2 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 has 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). 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).

5.2.1 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, 2002a, 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.

We estimated the changes in the number of new cases of PM-related chronic bronchitis using the study by Abbey et al (1995c) which is based on a sample of California residents. The study by Abbey et al. examined the relationship between estimated PM_{2.5} (annual mean from 1966 to 1977), PM₁₀ (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 airway obstructive disease (AOD) or asthma; PM₁₀ 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.

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 the Krupnick and Cropper (1992), provide estimates of WTP to avoid a case of chronic bronchitis.

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

The study by Viscusi et al. (1991) uses a sample that is larger and more representative of the general population than the study by Krupnick and Cropper (which selects people who have a relative with the disease). Thus, the valuation for the high-end estimate is based on the distribution of WTP responses from Viscusi et al. The WTP to avoid a case of pollution-related chronic bronchitis (CB) is derived by starting with the WTP to avoid a severe case of chronic bronchitis, as described by Viscusi et al, 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, which was about \$319,000 (\$331,000 in 1999\$), 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 \$1,275,000 (1999 \$)¹⁰; the median response implied a WTP of about \$676,000 (1999 \$). 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. The mean of this distribution is about \$918,000 (1999 \$).

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 $\ln(\text{WTP})$ 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 $\ln(\text{WTP})$ on severity, discussed above. This estimated constant (regression coefficient) is normally distributed with mean = 0.18 and standard deviation = 0.0669 (obtained from Krupnick and Cropper).

The distribution of WTP to avoid a case of pollution-related CB was generated by Monte Carlo methods, drawing from the three distributions described above. On each of 16,000 iterations (1) a value was selected from each distribution, and (2) a value for WTP was generated by the iterative procedure described above, in which the severity level was decreased by one percent on each iteration, and the corresponding WTP was derived. The mean of the resulting distribution of WTP to avoid a case of pollution-related CB was \$331,000 (1999\$).

This WTP estimate is reasonably consistent with full COI estimates derived for chronic bronchitis, using average annual lost earnings and average annual medical expenditures reported by Cropper and Krupnick (1990). Using a 5 percent discount rate and assuming that (1) lost earnings continue until age 65, (2) medical expenditures are incurred until death, and (3) life expectancy is unchanged by chronic bronchitis, the present discounted value of the stream of medical expenditures and lost earnings associated with an average case of chronic bronchitis is estimated to be about \$113,000 for a 30 year old, about \$109,000 for a 40 year old, about \$100,000 for a 50 year old, and about \$57,000 for a 60 year old. A WTP estimate would be expected to be greater than a full COI estimate, reflecting the willingness to pay to avoid the pain and suffering associated with the illness. The WTP estimate of \$331,000 is from 2.9 times the full COI estimate (for 30 year olds) to 5.8 times the full COI estimate (for 60 year olds).

5.3 Non-Fatal Myocardial Infarction (Heart Attacks)

Non-fatal heart attacks have been linked with short term exposures to $\text{PM}_{2.5}$ 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

¹⁰ 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 1999 dollars.

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.

5.3.1 Valuing Non-Fatal Myocardial Infarction (Heart Attack)

EPA has only recently 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 5-3, 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. We currently do not have adequate information to characterize the uncertainty surrounding any of these estimates. Exhibit 5-4 gives the resulting estimates adjusted to 1999\$. Note that we assumed a three percent discount rate for this analysis.

Exhibit 5-3. 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 5-4. Estimated Costs Over a 5-Year Period of a Non-Fatal Myocardial Infarction

Age Group	Opportunity Cost ^a (1999\$)		Medical Cost ^b (1999\$)		Total Cost (1999\$)	
	3% Discount Rate	7% Discount Rate	3% Discount Rate	7% Discount Rate	3% Discount Rate	7% Discount Rate
0 - 24	\$0	\$0	\$65,466	\$64,881	\$65,466	\$64,881
25-44	\$8,774	\$7,855	\$65,466	\$64,881	\$74,240	\$72,736
45 - 54	\$12,932	\$11,578	\$65,466	\$64,881	\$78,398	\$76,459
55 - 65	\$74,746	\$66,920	\$65,466	\$64,881	\$140,212	\$131,801
> 65	\$0	\$0	\$65,466	\$64,881	\$65,466	\$64,881

^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". Note that we assumed a three percent discount rate for this analysis.

^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 (\$2000); 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.

5.4 Hospital Admissions

We estimate the impact of PM on both respiratory and cardiovascular hospital admissions. In addition, we estimate the impact of these pollutants on emergency room visits for asthma. The respiratory and cardiovascular hospital admissions studies used in the analysis are listed in Exhibits 5-5 and 5-6, respectively. Appendix A provides details on each study.

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. 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. The two categories of hospital admissions are therefore discussed together in this section.

Exhibit 5-5. Respiratory Hospital Admission Studies

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
Los Angeles, CA	Moolgavkar (2000b)	Chronic Lung Disease Less Asthma(ICD codes 490-492, 494-496)	PM _{2.5}	Ages 18-64
Seattle, WA	Sheppard, et al. (1999)	asthma (493)	PM _{2.5}	<65
Detroit, MI	Lippman et al. (2000)	Pneumonia (ICD-9 codes 480-487)	PM _{2.5}	Ages 65+
Detroit, (Lippman) Chicago, Los Angeles, and Phoenix (Moolgavkar)	Lippman et al. (2000), Moolgavkar (2000b)	Chronic Lung Disease (ICD codes 490-496)	PM _{2.5}	Ages 65+
Seattle, WA	Norris et al (1999)	Asthma-related ER visits (ICD code 493)	PM _{2.5}	< 18

Exhibit 5-6. Cardiovascular Hospital Admission Studies

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
Los Angeles, CA	Moolgavkar (2000a)	Cardiovascular (ICD codes 390-409, 411-429) ¹¹	PM _{2.5}	Ages 20-64
Los Angeles (Moolgavkar), Detroit (Lippman)	Moolgavkar (2000a), Lippman et al. (2000)	Cardiovascular ((ICD codes 390-409, 411-429) ¹²	PM _{2.5}	age 65+

¹¹ 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.

¹² 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.

5.4.1 Respiratory and Cardiovascular Hospital Admissions

To estimate avoided incidences of cardiovascular hospital admissions associated with $PM_{2.5}$, we use studies by Moolgavkar (2000a) and Lippman 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 used 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 used only those focusing on $PM_{2.5}$. Both Moolgavkar (2000a) and Lippman 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.

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), and the pain and suffering of the affected individual as well as of that of relatives, friends, and associated care-givers.¹³

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.

¹³ 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.

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 available for use in BenMAP 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. Based on Science Advisory Board (SAB) advice, the COI values currently available are not adjusted.

Unit values are based on ICD-code-specific estimated hospital charges and 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.

For all hospital admissions endpoints, estimates of hospital charges and lengths of hospital stays were based on discharge 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 is estimated as the mean hospital charge plus n times the daily lost wage. Year 2000 county-specific median annual wages¹⁴ divided by (52*5) were used to estimate county-specific median daily wages, and then were adjusted to 1999\$ with the employment cost index. Because the wage data used in this analysis are county-specific, the unit value for a hospital admission varies from one county to another.

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, with negligible standard errors. Exhibits 5-7 and 5-8 present the estimates that we use.

Exhibit 5-7. Unit Values for Respiratory Hospital Admissions

Endpoints Estimated (ICD code)	Age of Study Population	COI ^a (1999 \$)
Chronic Lung Disease Less Asthma(ICD codes 490-492, 494-496)	Ages 18-64	\$11,333
Asthma (493)	<65	\$7,467
Pneumonia (ICD-9 codes 480-487)	Ages 65+	\$17,106
Chronic Lung Disease (ICD codes 490-496)	Ages 65+	\$13,083
Asthma-related ER visits (ICD code 493)	< 18	\$275

^a Source of hospital charges and lengths of stay: Agency for Healthcare Research and Quality. 2000. HCUPnet, Healthcare Cost and Utilization Project. <http://www.agrq.gov/data/hcup/hcupnet.htm> .

¹⁴ Source: U.S. Year 2000 Census, compiled by Geolytics.

Exhibit 5-8. Unit Values for Cardiovascular Hospital Admissions

Endpoints Estimated (ICD code)	Age of Study Population	COI ^a (1999 \$)
Cardiovascular (ICD codes 390-409, 411-429)	Ages 20-64	\$21,864 (ICD codes 390-429)
Cardiovascular (ICD codes 390-409, 411-429)	age 65+	\$20,334 (ICD codes 390-429)

* Source of hospital charges and lengths of stay: Agency for Healthcare Research and Quality. 2000. HCUPnet, Healthcare Cost and Utilization Project. <http://www.agrq.gov/data/hcup/hcupnet.htm> .

5.4.2 Asthma-Related Emergency Room (ER) 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 PM₁₀ 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.

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.

5.5 Acute Illnesses and Symptoms Not Requiring Hospitalization

We consider in this section a number of acute symptoms that do not require hospitalization, such as acute bronchitis, and upper and lower respiratory symptoms (Exhibit 5-9). Several of these illnesses and symptoms were considered in the §812 Prospective analysis as well. The unit values and the uncertainty distributions for those acute illnesses and symptoms that were also considered in the §812 Prospective analysis were obtained by adjusting the unit values used in that analysis from 1990 \$ to 1999 \$ by multiplying by the CPI-U for “all items.”

For several of the acute symptoms and illnesses for which more than one unit value is available, one of these is the value that EPA used in several recent benefits analyses. These “original” unit values were all based on a set of three CV studies, in which respondents were asked their WTP to avoid a day of specific symptoms. These study- and symptom-specific WTP estimates, along with the recommended midrange estimates derived by IEC (1993) on which the original unit values were based, are presented in Exhibit 5-10 below.

Exhibit 5-9. Studies of Symptoms/Illnesses not Requiring Hospitalization

Endpoint	Study	Pollutants	Study Population
Acute bronchitis	Dockery et al. (1996)	PM _{2.5}	Ages 8-12
Upper respiratory symptoms (URS)	Pope et al. (1991)	PM ₁₀	Asthmatics, ages 9-11
Lower respiratory symptoms (LRS)	Schwartz et al. (1994)	PM _{2.5}	Ages 7-14
Minor restricted activity day (MRAD)	Ostro and Rothschild (1989)	PM _{2.5}	Ages 18-65
Work loss days (WLDs)	Ostro (1987)	PM _{2.5}	Ages 18-65

Exhibit 5-10. Median WTP Estimates and Derived Midrange Estimates (in 1999 \$)

Symptom ^a	Dickie et al. (1987)	Tolley et al. (1986)	Loehman et al. (1979)	Mid-Range Estimate
Throat congestion	4.81	20.84	-	12.75
Head/sinus congestion	5.61	22.45	10.45	12.75
Coughing	1.61	17.65	6.35	8.93
Eye irritation	-	20.03	-	20.03
Headache	1.61	32.07	-	12.75
Shortness of breath	0.00	-	13.47	6.37
Pain upon deep inhalation	5.63	-	-	5.63
Wheeze	3.21	-	-	3.21
Coughing up phlegm	3.51 ^b	-	-	3.51
Chest tightness	8.03	-	-	8.03

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

^b 10% trimmed mean.

5.5.1 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 encyclopedia¹⁵, with the exception of cough, most acute bronchitis 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 PM₁₀ 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.

¹⁵ See <http://www.nlm.nih.gov/medlineplus/ency/article/000124.htm>, accessed January 2002

In previous benefits analyses, EPA used a unit value of \$59.31. 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 estimate was taken to be twice the value of a minor respiratory restricted activity day.¹⁶

5.5.2 Upper Respiratory Symptoms (URS)

Using logistic regression, Pope et al. (1991) estimated the impact of PM₁₀ 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 PM₁₀ 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, NO₂, and SO₂ 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 PM₁₀ significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM₁₀ effect. The results from the school-based sample are used here.

Valuing URS

Willingness to pay to avoid a day of URS is based on symptom-specific WTPs to avoid those symptoms identified by Pope et al. as part of the URS complex of symptoms. Three contingent valuation (CV) studies have estimated WTP to avoid various morbidity symptoms that are either within the URS symptom complex defined by Pope et al. (1991) or are similar to those symptoms identified by Pope et al. 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 5-10.

The three individual symptoms listed in Exhibit 5-10 that were identified as most closely matching those listed by Pope, et al. for URS 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 URS could consist of any one of the seven possible “symptom complexes” consisting of at least one of these three symptoms. There are seven possible symptom complexes. It is assumed that each of the seven URS complexes is equally likely.¹⁷ The point estimate of MWTP to avoid an occurrence of URS is just an average of the seven estimates of MWTP for the different URS complexes – \$18.70, or about \$19 in 1990 \$. This is \$24.23 (= \$19*1.275) in 1999 \$. In the absence of information surrounding

¹⁶ For a more complete description of the derivation of this estimate, see Abt Associates (2000, p. 4-30).

¹⁷ 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.

the frequency with which each of the seven types of URS occurs within the URS symptom complex, an uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution, with a range of [\$7, \$33], or [\$8.93, \$42.08] in 1999 \$.

5.5.3 Lower Respiratory Symptoms (LRS)

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 SO₂, NO₂, ozone, PM₁₀, 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 SO₂, NO₂, PM_{2.5}, and PM₁₀ were significantly linked to cough. In two-pollutant models, PM₁₀ had the most consistent relationship with cough; ozone was marginally significant, controlling for PM₁₀. In models for upper respiratory symptoms, they reported a marginally significant association for PM₁₀. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO₂, O₃, PM_{2.5}, PM₁₀, SO₄, 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 LRS

The method for deriving a point estimate of mean WTP to avoid a day of LRS is the same as for URS. Schwartz et al. (1994, p. 1235) define LRS as at least two of the following symptoms: cough, chest pain, phlegm, and wheeze. The symptoms for which WTP estimates are available that reasonably match those listed by Schwartz et al. for LRS are cough, chest tightness, coughing up phlegm, and wheeze. A day of LRS, as defined by Schwartz et al., could consist of any one of the 11 combinations of at least two of these four symptoms.

We assumed that each of the eleven types of LRS is equally likely.¹⁸ The mean WTP to avoid a day of LRS as defined by Schwartz et al. (1994) is therefore the average of the mean WTPs to avoid each type of LRS, – \$11.82. This is \$15.07 (=1.275*\$11.82) in 1999 \$. This is the point estimate used in the benefit analysis for the dollar value for LRS as defined by Schwartz et al. 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, WTP to avoid a day of LRS should be a conservative estimate of WTP to avoid a case of LRS.

In the absence of information about the frequency of each of the seven types of LRS among all occurrences of LRS, the uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution, with a range of [\$5, \$19], or [\$6.37, \$24.22] in 1999 \$. This is the same procedure as that used in the URS uncertainty analysis.

¹⁸ 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.

5.5.4 Minor Restricted Activity Days (MRADs)

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 (MRADs)

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 1999 \$ by multiplying by 1.275. 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 1999 \$, this is a triangular distribution centered at \$48.43, ranging from \$20.34 to \$77.76.

5.5.5 Work Loss Days (WLD)

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 WLDs

Willingness to pay to avoid the loss of one day of work was estimated by dividing the county median annual wage for 2000 by an assumed 50 week work-year and 5 days of work per week (to get the median daily wage). This values the loss of a day of work at the county median wage for the day lost. 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.

6. Results

This chapter provides estimates of the magnitude and value of changes in adverse health effects associated with reducing on-road and non-road diesel admissions. Exhibit 6-1 presents the number of adverse health cases avoided, and Exhibit 6-2 presents the monetary value of the avoidable health effects.

Along with using additional years of follow-on data than was previously available, Pope et al (2002) also found a statistically significant relationship between $PM_{2.5}$ levels and a specific cause of death: lung cancer. Exhibit 6-3 presents estimates of the number of PM-related premature deaths from lung cancer, as well as the total mortality estimates previously presented. Note that the lung cancer mortality estimates are not additional deaths beyond the estimates from the all-cause mortality results presented in Table 6-1. The mortality estimates from lung cancer are included in the all-cause premature mortality estimates; the remaining cases of premature mortality (approximately 88 percent of the total) are from other causes, including both respiratory and cardiovascular diseases.

Another health effect associated with exposure to PM are asthma attacks. Because of possible double counting with endpoints that are included (such as emergency room visits for asthma and upper respiratory symptom days), EPA does not quantify the number of asthma attacks. Exhibit 6-3 presents the results of this alternative analysis.

Exhibit 6-1. Health Benefits Estimates: Numbers of Cases Reduced

Effect (age group)	5%	Mean	95%
Mortality (30+)	8,000	21,000	34,000
Chronic Bronchitis (27+)	2,200	12,000	22,000
Heart Attacks (18+)	9,900	27,000	43,000
<u>Hospital Admissions & Visits</u>			
Chronic Lung, less Asthma (20-64)	180	730	1,300
Asthma (0-64)	440	1,400	2,300
Pneumonia (65+)	1,500	5,300	9,000
Chronic Lung (65+)	-810	640	2,200
<i>Total Hospital Admissions-Respiratory</i>		<i>8,000</i>	
All Cardiovascular (20-64)	170	2,000	3,800
All Cardiovascular (65+)	-930	4,500	14,000
<i>Total Hospital Admissions-Cardiovascular</i>		<i>6,500</i>	
Emergency Room Visits for Asthma (<18)	9,000	15,000	22,000
<u>Acute Symptoms Not Requiring Hospitalization</u>			
Acute Bronchitis (8-12)	-1,000	29,000	58,000
Lower Respiratory Symptoms (7-14)	160,000	330,000	510,000
Upper Respiratory Symptoms (9-11)	85,000	270,000	450,000
Work Loss Days (18-64)	2,100,000	2,400,000	2,700,000
Minor Restricted Activity Days (18-64)	12,000,000	14,000,000	17,000,000

Exhibit 6-2. Value of Health Benefits (in millions of \$1999)

Effect (age group)	5%	Mean	95%
Mortality (30+)	\$15,000	\$130,000	\$340,000
Chronic Bronchitis (27+)	\$270	\$4,400	\$15,000
Heart Attacks (18+)	\$530	\$2,200	\$4,900
<u>Hospital Admissions & Visits</u>			
Chronic Lung, less Asthma (20-64)	\$2	\$9	\$15
Asthma (0-64)	\$3	\$10	\$18
Pneumonia (65+)	\$27	\$91	\$160
Chronic Lung (65+)	-\$11	\$8	\$29
<i>Total Hospital Admissions-Respiratory</i>		<i>\$120</i>	
All Cardiovascular (20-64)	\$4	\$43	\$83
All Cardiovascular (65+)	-\$19	\$92	\$290
<i>Total Hospital Admissions-Cardiovascular</i>		<i>\$140</i>	
Emergency Room Visits for Asthma (<18)	\$2	\$4	\$6
<u>Acute Symptoms Not Requiring Hospitalization</u>			
Acute Bronchitis (8-12)	\$0	\$10	\$26
Lower Respiratory Symptoms (7-14)	\$2	\$5	\$10
Upper Respiratory Symptoms (9-11)	\$2	\$7	\$15
Work Loss Days (18-64)	\$290	\$330	\$370
Minor Restricted Activity Days (18-64)	\$420	\$730	\$1,000

Exhibit 6-3. Alternative Health Benefits Estimates: Numbers of Cases Reduced

	Mean
Mortality (all-cause) ^a	21,000
<i>Mortality (lung cancer)</i>	<i>3,000</i>
ER visits for asthma ^a	15,000
Upper respiratory symptoms ^a	270,000
<i>Asthma Attacks</i>	<i>410,000</i>

^aThe estimates for all-cause mortality, ER visits for asthma, and upper respiratory symptoms are taken from Exhibit 6-1 for purposes of comparison. Note that the estimates of lung cancer mortality and asthma attacks are excluded from the primary analysis because they overlap with these other estimates. The asthma attacks are calculated for asthmatics of all ages (based on Whittemore & Korn, 1980).

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Appendix A: Particulate Matter C-R Functions

Appendix A describes the concentration-response functions that we use in this analysis. Note that for all of the concentration-response functions we define ΔPM as $PM_{\text{baseline}} - PM_{\text{control}}$, and we define the change in incidence as: $-(\text{incidence}_{\text{control}} - \text{incidence}_{\text{baseline}})$.

A.1 Mortality

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

All-Cause Mortality

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.¹⁹ 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.

Average of '79-'83 and '99-'00 Exposure

¹⁹ 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.

The coefficient and standard error for PM_{2.5} using the average of '79-'83 and '99-'00 PM data are estimated from the relative risk (1.062) and 95% confidence interval (1.016-1.110) associated with a change in *annual mean* exposure of 10.0 µg/m³. Pope et al. (2002, Table 2).²⁰

Functional Form: Log-linear

Coefficient: 0.006015

Standard Error: 0.002257

Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older

Population: population of ages 30 and older

A.2 Chronic Illness

Schwartz (1993) and Abbey et al. (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).²¹

Chronic Bronchitis

Abbey et al. (1995c) examined the relationship between estimated PM_{2.5} (annual mean from 1966 to 1977), PM₁₀ (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; PM₁₀ 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.

The estimated coefficient (0.0137) is presented for a one µg/m³ 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 µg/m³ 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

²⁰ 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.

²¹ 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.

Population: population of ages 27 and older²² without chronic bronchitis = 95.57%²³ of population 27+

A.3 Heart Attacks

Acute Myocardial Infarction (Heart Attacks), Nonfatal

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 PM10, PM10-2.5, PM2.5, “black carbon”, O3, CO, NO2, and SO2 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 PM2.5 levels hours before and days before onset. The authors estimated multivariate conditional logistic models including two-hour and 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 PM2.5 concentrations before onset. Significant associations were observed for PM10 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).

The coefficient and standard error are calculated from an odds ratio of 1.62 (95% CI 1.13-2.34) for a 20 µg/m³ 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)²⁴

Population: population of ages 18 and older

²² Using the same data set, Abbey et al. (1995a, p. 140) reported that the respondents in 1977 ranged in age from 27 to 95.

²³ The American Lung Association (2002a, Table 4) reports a chronic bronchitis prevalence rate for ages 18 and over of 4.43% (American Lung Association, 2002a, Table 4).

²⁴ 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.

A.4 Hospital Admissions

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most emergency room visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

Hospital admissions require the patient to be examined by a physician, and on average may represent more serious incidents than ER visits (Lipfert, 1993, p. 230). The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking air pollution with other types of hospital admissions. The only types of ER visits that have been linked to air pollution in the U.S. or Canada are asthma-related visits.

Hospital Admissions for Chronic Lung Disease Less Asthma (Ages 18-64)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 2.0²⁵ and t-statistic of 2.2 for a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ 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)²⁶

Population: population of ages 18 to 64

Hospital Admissions for Asthma

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} , $\text{PM}_{2.5}$, coarse $\text{PM}_{10-2.5}$, SO_2 , ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects.²⁷ They found asthma hospital admissions associated with PM_{10} , $\text{PM}_{2.5}$, $\text{PM}_{10-2.5}$, 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 $\text{PM}_{2.5}$ and CO. Results for

²⁵ In a log-linear model, the percent change is equal to $(\text{RR} - 1) * 100$. In this study, Moolgavkar defines and reports the "estimated" percent change as $(\log \text{RR} * 100)$. Because the relative risk is close to 1, $\text{RR}-1$ and $\log \text{RR}$ 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.

²⁶ 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.

²⁷ $\text{PM}_{2.5}$ levels were estimated from light scattering data.

other co-pollutant models were not reported. The PM_{2.5} C-R function is based on the multipollutant model.

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 µg/m³ increase²⁸ 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

Hospital Admissions for Pneumonia

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 PM₁₀, 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 PM₁₀ were significant for ischemic heart disease (ICD code 410-414), and PM_{2.5} and PM₁₀ 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, SO₂, NO₂, 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.

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/m³ 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

Hospital Admissions for Chronic Lung Disease (Ages 65+)

The following two studies, Lippmann et al (2000) and Moolgavkar (2000b), were combined together using a random/fixed effects pooling method. The pertinent information for the individual studies has been included below.

²⁸ The reported Inter Quartile Range(11.8 µg/m³) 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.

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 PM₁₀, 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 PM₁₀ were significant for ischemic heart disease (ICD code 410-414), and PM_{2.5} and PM₁₀ 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, SO₂, NO₂, 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.

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/m³ 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

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, SO₂, NO₂, CO, and PM₁₀ 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 PM₁₀. 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.

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.8²⁹ and t-statistic of 0.8 for a 10 µg/m³ 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

Hospital Admissions, All Cardiovascular (Ages 20-64)

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, SO₂, NO₂, CO, and PM₁₀ 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. 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, SO₂ 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.

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.9³⁰ and t-statistic of 1.8 for a 10 µg/m³ 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)³¹

Population: population of ages 18 to 64

²⁹ 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.

³⁰ 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).

³¹ 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.

Hospital Admissions for All Cardiovascular (Ages 65+)

We use studies by Moolgavkar (2000a) and Lippmann et al (2000) to estimate the impact of PM on cardiovascular hospital admissions for persons aged 65 and older.

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, SO₂, NO₂, CO, and PM₁₀ 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 PM₁₀ or PM_{2.5}. The strongest overall effects were observed for SO₂ 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.

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.5³² and t-statistic of 0.9 for a 10 µg/m³ increase in PM_{2.5} in the one day lag model (Moolgavkar, 2000a, Table 3, p. 1202).

Functional Form: Log-linear

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)³³

Population: population of ages 65 and older

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 PM₁₀, 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 PM₁₀ were significant for ischemic heart disease (ICD code 410-414),

³² 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).

³³ 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.

and PM_{2.5} and PM₁₀ 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, SO₂, NO₂, or CO, the results were generally comparable. The PM_{2.5} C-R function is based on the co-pollutant model with ozone.

Dysrhythmia Hospital Admissions

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 µg/m³ 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

Heart Failure Hospital Admissions

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 µg/m³ 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

Ischemic Heart Disease Hospital Admissions

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 µg/m³ 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)³⁴

Population: population of ages 65 and older

³⁴ 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.

A .5 Emergency Room Visits

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and ER visits. Because most ER visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

The only types of ER visit that have been explicitly linked to ozone in U.S. and Canadian epidemiological studies are asthma visits. However, it seems likely that ozone may be linked to other types of respiratory-related ER visits.

Emergency Room Visits for Asthma

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 PM₁₀, light scattering (used to estimate fine PM), CO, SO₂, NO₂, and O₃ 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}), PM₁₀, and CO. No association was found between O₃, NO₂, or SO₂ and asthma ER visits, although O₃ had a significant amount of missing data. In multipollutant models with either PM metric (light scattering or PM₁₀) and NO₂ and SO₂, 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.

In a model with NO₂ and SO₂, 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 µg/m³ 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

A .6 Acute Morbidity

In addition to chronic illnesses and hospital admissions, there is a considerable body of scientific research that has estimated significant relationships between elevated air pollution levels and other morbidity health effects. Chamber study research has established relationships between specific air pollution chemicals and symptoms such as coughing, pain on deep inspiration, wheezing, eye irritation and headaches. In addition, epidemiological research has found air pollution relationships with acute infectious diseases (e.g., bronchitis, sinusitis) and a variety of "symptom-day" categories. Some "symptom-day" studies examine excess incidences of days with identified symptoms such as wheezing, coughing, or other specific upper or lower respiratory symptoms. Other studies estimate relationships for days with a more general description of days with adverse health impacts, such as "respiratory restricted activity days" or work loss days.

A challenge in preparing an analysis of the minor morbidity effects is identifying a set of effect estimates that reflects the full range of identified adverse health effects but avoids double counting. From the definitions of the specific health effects examined in each research project, it is possible to identify a set of effects that are non-overlapping, and can be ultimately treated as additive in a benefits analysis.

Acute Bronchitis

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in 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.³⁵ They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes.

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

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

where:

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

Incidence Rate. Bronchitis was counted in the study only if there were “reports of symptoms in the past 12 months” (Dockery et al., 1996, p. 501). It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. Dockery et al. found no relationship between PM and chronic cough and chronic phlegm, which are important indicators of chronic bronchitis. For this analysis, we assumed that the C-R function based on Dockery et al. is measuring acute bronchitis.

In 1994, 2,115,000 children ages 5-17 experienced acute conditions (Adams and Marano, 1995, Table 6) out of population of 48.110 million children ages 5-17 (U.S. Bureau of the Census, 1998, Table 14), or 4.4 percent of this population. This figure is somewhat lower than the 5.34 percent of children under the age of 18 reported to have chronic bronchitis in 1990-1992 (Collins, 1997, Table 8). Dockery et al. (1996, p. 503) reported that in the 24 study cities the bronchitis rate varied from three to ten percent. Finally a weighted average of the incidence rates in the six cities in the Dockery et al. (1989) study is 6.34 percent, where the sample size from each city is used to weight the respective incidence rate (Dockery et

³⁵ 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.

al., 1989, Tables 1 and 4).³⁶ This analysis assumes a 4.4 percent prevalence rate is the most representative of the national population. Note that this measure reflects the fraction of children that have a chest ailment diagnosed as bronchitis in the past year, not the number of days that children are adversely affected by acute bronchitis.³⁷

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

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

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

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

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

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

Lower Respiratory Symptoms

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 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

³⁶ The unweighted average of the six city rates is 0.0647.

³⁷ In 1994, there were 13,707,000 restricted activity days associated with acute bronchitis, and 2,115,000 children (ages 5-17) experienced acute conditions (Adams and Marano, 1995, Tables 6 and 21). On average, then, each child with acute bronchitis suffered 6.48 days.

significant association for PM₁₀. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO₂, O₃, PM_{2.5}, PM₁₀, SO₄, and H⁺.

The C-R function used to estimate the change in lower respiratory symptoms is:

$$\Delta \text{Lower Respiratory Symptoms} = - \left[\frac{y_0}{(1 - y_0) \cdot e^{\Delta PM_{2.5} \cdot \beta} + y_0} - y_0 \right] \cdot \text{pop}.$$

where:

y_0 = daily lower respiratory symptom incidence rate per person = 0.0012

β = estimated PM_{2.5} logistic regression coefficient = 0.01823

$\Delta PM_{2.5}$ = change in daily average PM_{2.5} concentration

pop = population of ages 7-14

σ_β = standard error of β = 0.00586

Incidence Rate. The proposed incidence rate, 0.12 percent, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). They did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated 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 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 is 0.12 percent,³⁸ which is used in this analysis.

Coefficient Estimate (β). The coefficient β is calculated from the reported odds ratio (= 1.44) in a single-pollutant model associated with a 20 $\mu\text{g}/\text{m}^3$ change in PM_{2.5} (Schwartz et al., 1994, Table 5):

$$\beta = \frac{\ln(1.44)}{20} = 0.01823.$$

Standard Error (σ_β). The standard error for the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (Schwartz et al., 1994, Table 5):

$$\sigma_{\beta, \text{high}} = \frac{\beta_{\text{high}} - \beta}{1.96} = \frac{\left(\frac{\ln(1.82)}{20} - \frac{\ln(1.44)}{20} \right)}{1.96} = 0.00597$$

$$\sigma_{\beta, \text{low}} = \frac{\beta - \beta_{\text{low}}}{1.96} = \frac{\left(\frac{\ln(1.44)}{20} - \frac{\ln(1.15)}{20} \right)}{1.96} = 0.00574$$

$$\sigma_\beta = \frac{\sigma_{\beta, \text{high}} + \sigma_{\beta, \text{low}}}{2} = 0.00586.$$

³⁸ For example, the 62.5th percentile would have an estimated incidence rate of 0.145 percent.

Population. Schwartz et al. (1994, Table 5 and p. 1235) enrolled 1,844 children into a year-long study conducted in different years in different cities; the students were in grades two through five and lived in six U.S. cities. All study participants were enrolled in September 1984; the actual study was conducted in Watertown, MA in 1984/85; Kingston-Harriman, TN, and St. Louis, MO in 1985/86; Steubenville, OH, and Portage, WI in 1986/87; and Topeka, KS in 1987/88. The study does not publish the age range of the children when they participated. As a result, the study is somewhat unclear about the appropriate age range for the resulting C-R function. If all the children were in second grade in 1984 (ages 7-8) then the Topeka cohort would be in fifth grade (ages 10-11) when they participated in the study. It appears from the published description, however, that the students were in grades two through five in 1984.³⁹ By the completion of the study, some students in the Topeka cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

Upper Respiratory Symptoms

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.

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 population⁴⁰ ages 9 to 11 = 5.67% of population ages 9 to 11

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

³⁹ Neas et al. (1994, p. 1091) used the same data set; their description suggests that grades two to five were represented initially.

⁴⁰ The American Lung Association (2002b, Table 7) estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

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.

The study is based on a “convenience” sample of individuals ages 18-65. 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 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b). On the other hand, the number of workers over the age of 65 is relatively small; it was under 3% of the total workforce in 1996 (U.S. Bureau of the Census, 1997, Table 633).

The C-R function to estimate the change in the number of work-loss days is:

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

where:

- y_0 = daily work-loss-day incidence rate per person = 0.00648
- β = inverse-variance weighted $PM_{2.5}$ coefficient = 0.0046
- $\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration⁴¹
- pop = population of ages 18 to 65
- σ_β = standard error of β = 0.00036

Incidence Rate. The estimated 1994 annual incidence rate is the annual number (376,844,000) of WLD per person in the age 18-64 population divided by the number of people in 18-64 population (159,361,000). The 1994 daily incidence rate is calculated as the annual rate divided by 365.⁴² Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

Coefficient Estimate (β). 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:

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.0046.$$

Standard Error (σ_β). The standard error of the coefficient (σ_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

⁴¹ The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

⁴² Ostro (1987) analyzed a sample aged 18 to 65. It is assumed that the age 18-64 rate is a reasonably good approximation to the rate for individuals 18-65. Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

$$\sigma_{\beta}^2 = \text{var} \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1976}^{1981} \text{var} \left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right).$$

This eventually reduces down to:

$$\sigma_{\beta}^2 = \frac{1}{\gamma} \Rightarrow \sigma_{\beta} = \sqrt{\frac{1}{\gamma}} = 0.00036.$$

Minor Restricted Activity Days

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ 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. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average O_3 has highly variable association with RRADs and MRADs. Controlling for O_3 , two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.

The study is based on a “convenience” sample of individuals ages 18-65. 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 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b).

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 used in this analysis is a weighted average of the coefficients (Ostro, 1987, Table IV) using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) is:

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

where:

- y_0 = daily MRAD daily incidence rate per person = 0.02137
- β = inverse-variance weighted $PM_{2.5}$ coefficient = 0.00741
- $\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration⁴³
- pop = adult population ages 18 to 65
- σ_{β} = standard error of β = 0.0007

Incidence Rate. The annual incidence rate (7.8) provided by Ostro and Rothschild (1989, p. 243) was divided by 365 to get a daily rate of 0.02137.

Coefficient Estimate (β). 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:

⁴³ The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

$$\beta = \frac{\left(\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2} \right)}{\left(\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2} \right)} = 0.00741.$$

Standard Error (σ_{β}). The standard error of the coefficient (σ_{β}) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_{\beta}^2 = \text{var} \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = \frac{\left(\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2} \right)^2}{\gamma} = \sum_{i=1976}^{1981} \text{var} \left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right).$$

This reduces down to:

$$\sigma_{\beta}^2 = \frac{1}{\gamma} \Rightarrow \sigma_{\beta} = \sqrt{\frac{1}{\gamma}} = 0.00070.$$

A.7 Supplemental Concentration Response Functions

Mortality, Lung Cancer

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.⁴⁴ 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.

Average of '79-'83 and '99-'00 Exposure

The coefficient and standard error for $PM_{2.5}$ using the average of the '79-'83 and 99-'00 PM data are estimated from the relative risk (1.135) and 95% confidence interval (1.044-1.234) associated with a change in *annual mean* exposure of $10.0 \mu\text{g}/\text{m}^3$. Pope et al. (2002, Table 2).⁴⁵

Functional Form: Log-linear

Coefficient: 0.0126633

Standard Error: 0.0042653

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

⁴⁴ 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.

⁴⁵ 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.

Asthma Attacks

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 (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 TSP⁴⁶ 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 = 0.0550⁴⁷

Population: population of asthmatics of all ages = 3.86% of the population of all ages (American Lung Association, 2002b, Table 7)

⁴⁶ 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).

⁴⁷ 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.