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Technical Support Document for the Powerplant Impact Estimator Software Tool

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Prepared for
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1. Introduction

The purpose of this report is to describe a software application, the Powerplant Impact Estimator (PIE), developed to estimate the health and economic of electric generating units (EGUs) in the United States. In particular, we focus on the impacts in the years 2010, 2015, and 2020 of reducing ambient concentrations of particulate matter less than 2.5 microns in aerodynamic diameter ($PM_{2.5}$) – an air pollutant that has been linked to a variety of serious health effects, including asthma attacks, chronic bronchitis, hospital admissions, and premature mortality.

To estimate the $PM_{2.5}$-related benefits associated with reducing emissions from EGUs, the PIE model first calculates the impact on ambient air quality, and then using the results from epidemiological studies, it estimates the number of adverse health impacts (*e.g.*, avoided deaths), and then finally it estimates the associated economic benefits. This three-step process is the standard approach for evaluating the health and economic benefits of reduced air pollution. EPA used this approach when evaluating the National Ambient Air Quality Standards (U.S. EPA, 2006), the Clean Air Act (U.S. EPA, 1999b), the benefits of reducing greenhouse gases (Abt Associates Inc., 1999), the health effects of motor vehicles (U.S. EPA, 2000; 2004), and other major regulations.

This report describes the algorithms used to calculate population exposure, adverse health impacts, and the economic benefit of reducing these emissions. Chapter 2 provides an overview of PIE, and Chapter 3 describes the emissions reduction and modeling of the subsequent air quality change. Chapters 4 and 5 describe the estimation of health impacts and their valuation. The various appendices describe in further detail the assumptions and calculations underlying the analysis, and they also present health impact estimates for existing and new EGUs.

2. Overview of Powerplant Impact Estimator (PIE)

Abt Associates developed the Powerplant Impact Estimator, or PIE, to support assessments of the human health benefits of air pollution reductions and their associated economic benefits. PIE is the result of years of research and development, and reflects methods that are based on the peer-reviewed health and benefits analysis literature.

2.1 Damage Function Approach

PIE is based on a damage function approach, which involves modeling changes in ambient air pollution levels, calculating the associated change in adverse health effects, such as premature mortality, and then assigning an economic value to these effects. For changes in the concentrations of particulate matter and ozone, this is typically done by translating a change in pollutant levels into associated changes in human health effects. These health effects are then translated into economic values.

The first step in this process involves *health impact functions*, which are derived from concentration-response functions reported in the peer-reviewed epidemiological literature. A typical health impact function has four components:
(1) an effect estimate, which quantifies the change in health effects per unit of change in a pollutant, and is derived from a particular concentration-response function from an epidemiology study;
(2) a baseline incidence rate for the health effect;
(3) the affected population; and
(4) the estimated change in the concentration of the pollutant.

The health impact function might look like:

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

where y_0 is the baseline incidence (the baseline incidence rate times the affected population), β is the effect estimate, and Δx is the estimated change in pollutant concentration. Health impact functions come in many forms (as seen in Appendix B), but the basic elements remain the same.

The result of these functions, Δy, is an estimated change in the incidence of a particular health effect for a given change in air pollution. Examples of health effects that have been associated with changes in air pollution levels include premature mortality, hospital admissions for respiratory and cardiovascular illnesses, and asthma exacerbation.

The second step in the damage function approach involves estimated *unit values* that give the estimated economic value of avoiding a single case of a particular endpoint – a single death, for example, or a single hospital admission. These unit values are derived from the economics literature, and come in several varieties.

- For some endpoints, such as hospital admissions, we use *cost of illness* (COI) unit values, which estimate the cost of treating or mitigating the effect. COI unit values generally underestimate the

true value of reductions in risk of a health effect, since they include hospital costs and lost wages, but do not include any estimate of the value of avoided pain and suffering.

- For other endpoints, such as asthma exacerbation, we use *willingness to pay* (WTP) unit values, which are estimates of willingness to pay to avoid an asthma exacerbation.
- Typically *value of statistical life* (VSL) unit values are used for reductions in risk of premature mortality.

Returning to the previous equation, estimating the economic benefit of the estimated change in health incidence is a simple matter of multiplying by the associated unit value:

$$\$Benefits = \Delta y \cdot Unit\ Value$$

Finally, the calculation of total benefits involves summing estimated benefits across all non-overlapping health effects, such as hospital admissions for pneumonia, chronic lung disease, and cardiovascular-related problems.

2.2 PIE Analysis

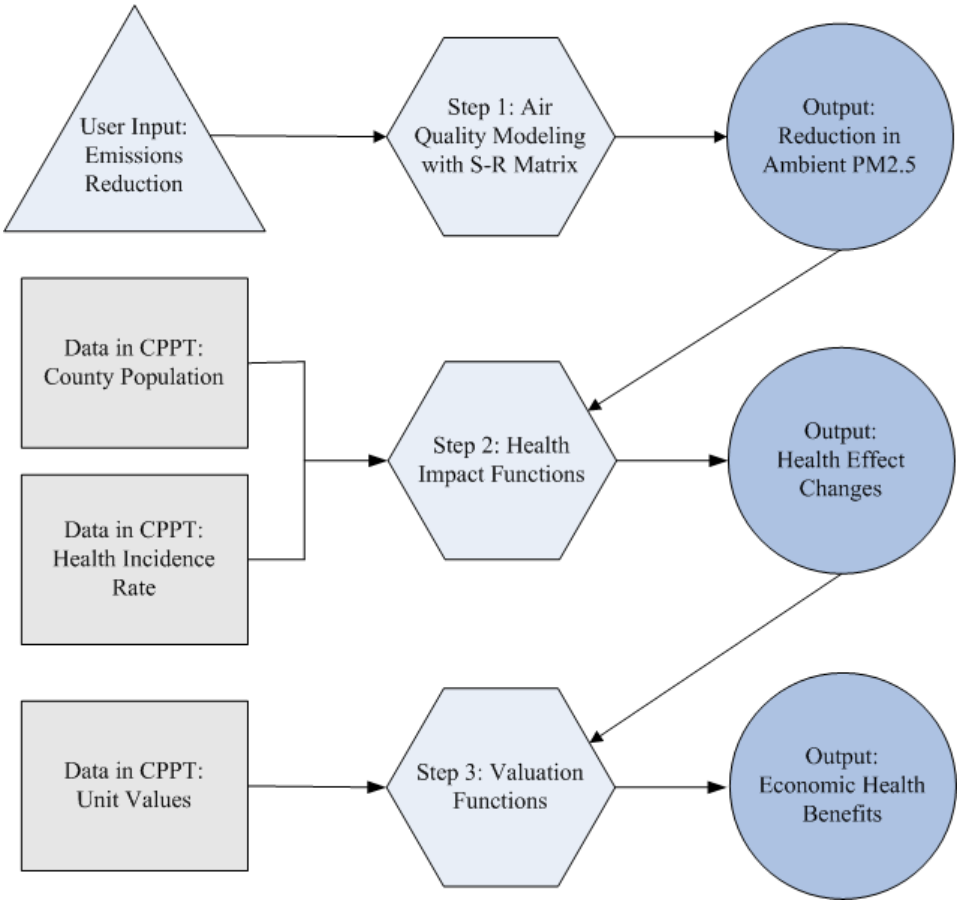
A PIE analysis relies on first estimating a reduction in air pollution emissions. The determination fo the emission reduction occurs outside of PIE and is used as input to the PIE analysis. After the user enters this information into PIE, the model then estimates:

(1) the reduction in ambient $PM_{2.5}$ levels in each county in the continental United States;
(2) the associated reduction in the incidence of various adverse health effects; and
(3) the associated economic benefit of these reductions in adverse health effects.

Chapter 3 discusses how PIE calculates the change in ambient $PM_{2.5}$ levels (Step 1). Chapter 4 discusses the calculation of the reduction in the incidence of adverse health effects (Step2). Chapter 5 discusses the calculation of the economic benefit (Step 5). The appendices to this report provide additional details for each of the steps.

Figure 1 summarizes these steps, and illustrates the flow of data through PIE.

Figure 1. PIE Data Flow



3. Emissions and Impacts on Ambient $PM_{2.5}$

This chapter examines how reductions in sulfur dioxide (SO_2), nitrogen oxides (NO_x), fine particulate matter less than 2.5 microns in aerodynamic diameter ($PM_{2.5}$), volatile organic carbon (VOC), and ammonia (NH_3) emissions can affect population exposure to particulate matter air pollution. As noted in Chapter 1, the focus of our analysis is on $PM_{2.5}$ – both emitted directly from EGUs and formed in secondary reactions in the atmosphere – because the most serious adverse health impacts, such as premature mortality and chronic bronchitis, are related to $PM_{2.5}$.

3.1 Modeling Reductions in Ambient $PM_{2.5}$ Using the S-R Matrix

The great majority of the impact of power plants is on $PM_{2.5}$ formation, with little change expected in more coarse particles. To estimate the change in $PM_{2.5}$, the PIE uses the S-R Matrix, which provides an estimate of the annual level of $PM_{2.5}$ in each county in the continental United States.

The S-R Matrix has two basic building blocks: an emissions inventory and transfer coefficients.[1] The emission inventory has the following pollutants that are relevant to $PM_{2.5}$ formation: SO_2, NO_x, direct emissions of $PM_{2.5}$, NH_3, and volatile organic carbons (VOCs). Emissions are estimated for several thousand identifiable point sources in the United States, and the inventory includes more diffuse "area" emissions, such as from motor vehicles and small point sources. The S-R matrix has a transfer coefficient from each source of emissions (point or area) to each county in the continental United States for four pollutants. There are four matrices: (1) directly emitted, or primary particulate matter, and organics; (2) SO_2; (3) NO_x; and (4) NH_3. The first stage of the modeling process is essentially matrix multiplication between a matrix of emissions and a matrix of transfer coefficients.

The result of this first stage is an estimate for each county in the United States of the micrograms per meter cubed ($\mu g/m^3$) of the pollutants from each of the point and area sources that can subsequently react to form $PM_{2.5}$: sulfate (from SO_2) , nitrate (from NO_x), ammonium (NH_3), organic aerosols (from VOC), and direct $PM_{2.5}$. Note that at this stage, we have a very large data set that provides for each county what pollutants can from *every* area and point source.

In the second stage, the S-R Matrix modeling process has a fairly simple air chemistry. First we sum up the ingredients (sulfate, nitrate, ammonium, organic aerosols, and direct $PM_{2.5}$) to get a total for each county. We then assume that all sulfate forms ammonium sulfate ($SO_4(NH_4)_2$). To get $\mu g/m^3$ of ammonium sulfate, we just multiply the micrograms of sulfate (SO_4) with the ratio of the molecular weight of $SO_4(NH_4)_2$ to the molecular weight of SO_4.

To estimate ammonium nitrate (NO_3NH_4), we calculate the moles of SO_4, the available NH_4 (before and after reacting with SO_4), and the available moles of nitrate (NO_3). To calculate the moles of SO_4, we divide the micrograms of SO_4 by the molecular weight of SO_4. Similarly, we estimate the moles of NH_4 before reacting with SO_4 by dividing the micrograms of NH_4 by the molecular weight of NH_4. Then, for every mole of SO_4 we will have two moles of NH_4 react with it. If there is not enough NH_4, then we assume SO_4 forms ammonium bisulfate ($NH4HSO_4$) or stays as particulate SO_4, and no ammonium nitrate would form in this case.

[1] Appendix A provides some background on the S-R Matrix.

If there is sufficient NH_4 to completely react with the SO_4, then any remaining NH_4 would be free to react with NO_3. To calculate the moles of NO_3, we divide the micrograms of NO_3 by its molecular weight, and then divide this number by four. (This is a seasonal adjustment incorporated in the S-R modeling approach.) The available moles of NH_4 can then react with the available moles of NO_3: one mole of NH_4 goes with one mole of NO_3 to produce ammonium nitrate (NO_3NH_4). To calculate $\mu g/m^3$ of NO_3NH_4, we multiply the moles of NO_3 that have reacted with NH_4 with the ratio of the molecular weight of NO_3NH_4 to the molecular weight of NO_3.

Now with $\mu g/m^3$ of ammonium sulfate, ammonium bisulfate, SO_4, and ammonium nitrate, to calculate overall $\mu g/m^3$ of $PM_{2.5}$, we added the $\mu g/m^3$ of organic aerosols and direct $PM_{2.5}$. Although this reduced form version of air chemistry is simple, peer-reviewed published studies of this approach nevertheless suggest that it is reasonably consistent with more complicated air quality models (see: Levy, et al., 2003).

4. Calculating Reductions in Adverse Health Impacts

A reduction in ambient $PM_{2.5}$ levels is associated with reductions in a number of adverse health effects, or "endpoints." This chapter discusses the calculation of these reductions in health impacts. The first section covers the choice of epidemiological studies and the development of health impact functions. The second section presents the health impact functions that we use. Appendix C provides additional details on the specific form of the health impact functions, and Appendices D and E describe the health incidence rate and population data used in these functions.

4.1 Issues in Selecting Epidemiological Studies and Health Impact Functions

This section reviews the steps we performed in selecting concentration-response (C-R) functions and developing health impact functions from them. The first section describes how studies were chosen from the epidemiological literature for use in the present analysis. The second section describes how we chose the specific estimated C-R relationships, or models, from among the potentially large number available in any given study. (In any given study, there are often a large number of estimated relationships between air pollution and adverse health effects, because the estimated relationship can depend on the number and types of pollutants included in the model, among other reasons.) In the third section, we briefly discuss the issue of thresholds in health impact functions.

Study Selection

The health impact functions in the PIE model were prepared by Abt Associates in close consultation with EPA and rely on an up-to-date assessment of the published scientific literature to ascertain the relationship between particulate matter and adverse human health effects. We evaluated studies using a variety of selection criteria, including: study location and design, the characteristics of the study population, and whether the study was peer-reviewed (Table 1).

Table 1. Summary of Considerations Used in Selecting Studies

Consideration	Comments
Peer reviewed research	Peer reviewed research is preferred to research that has not undergone the peer review process.
Study type	Among studies that consider chronic exposure (e.g., over a year or longer) prospective cohort studies are preferred over cross-sectional studies because they control for important individual-level confounding variables that cannot be controlled for in cross-sectional studies.
Study period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and life style over time.
Study size	Studies examining a relatively large sample are preferred because they generally have more statistical power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population, or through repeated observations on a smaller population, e.g. through a symptom diary recorded for a panel of asthmatic children.
Study location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior and life style.
Measure of PM	For this analysis, C-R functions based on $PM_{2.5}$ are preferred to those based on PM_{10} (particulate matter less than 10 microns in aerodynamic diameter) because reductions in emissions from diesel engines are expected to reduce fine particles and not have much impact on coarse particles.
Economically valuable health effects	Some health effects, such as changes in forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are therefore not quantified in this analysis.
Non-overlapping endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double counting of benefits. Including emergency room visits in a benefits analysis that already considers hospital admissions, for example, will result in double counting of some benefits if the category "hospital admissions" includes emergency room visits.

Model Selection

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

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

cases, professional judgment and an understanding of the study context are necessary as well to select the most appropriate models. Table 2 summarizes the selection criteria that we used.

Table 2. Description of Selection Criteria

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

Distributed Lag Effect

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

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

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

Thresholds

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

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

Moreover, USEPA recently completed an "expert elicitation" analysis in which it elicited opinions from 12 experts (in epidemiology, toxicology, and medicine) on the nature of this relationship (see: IEc, 2006). The experts were asked how likely they thought it is that the relationship between $PM_{2.5}$ and mortality is causal, and if it is causal, what is the functional form of the C-R relationship, including whether there is a threshold. Eleven of the twelve experts thought that, although each individual may have a threshold, there is insufficient empirical evidence for a threshold for the *population*, which is the entity of interest in the C-R function. One expert did include the possibility of a population threshold, assigning a probability of 50 percent to there being a threshold and, if there is a threshold, an 80 percent chance that it is less than or equal to 5 $\mu g/m^3$, and a 20 percent chance that it is between 5 and 10 $\mu g/m^3$.

While we find the evidence for thresholds to be limited, we nevertheless provide results both with and without a threshold assumption. To be consistent with some recent EPA analyses (e.g., U.S. EPA, 2008), we assume a threshold of 10 ug/m^3, and adjust the health impact coefficients to reflect this threshold. Appendix C discusses this adjustment process.

4.2 Summary of Health Impact Functions Used in this Analysis

This Chapter describes individual health effects associated with $PM_{2.5}$ and the functions used to quantify the expected number of cases of various health effects avoided as a result of eliminating emissions from new EGUs. Table 3 presents the PM-related health endpoints included in our analysis.

Table 3. Epidemiological Studies Used to Estimate Adverse Health Impacts of $PM_{2.5}$

Endpoint	Author	Age
Mortality, All Cause	Pope et al. (2002)	30-99
Mortality, All Cause	Laden et al (2006)	25-99
Mortality, All Cause	Woodruff et al. (1997)	Infant
Chronic Bronchitis	Abbey et al. (1995c)	27-99
Acute Myocardial Infarction, Nonfatal	Peters et al. (2001)	18-99
HA, All Cardiovascular (less Myocardial Infarctions)	Moolgavkar (2000b)	18-64
HA, All Cardiovascular (less Myocardial Infarctions)	Moolgavkar (2003)	65-99
HA, Congestive Heart Failure	Ito (2003)	65-99
HA, Dysrhythmia	Ito (2003)	65-99
HA, Ischemic Heart Disease (less Myocardial Infarctions)	Ito (2003)	65-99
HA, Pneumonia	Ito (2003)	65-99
HA, Chronic Lung Disease (less Asthma)	Moolgavkar (2000a)	18-64
HA, Chronic Lung Disease	Ito (2003)	65-99
HA, Chronic Lung Disease	Moolgavkar (2003)	65-99
HA, Asthma	Sheppard (2003)	0-64
Emergency Room Visits, Asthma	Norris et al. (1999)	0-17
Minor Restricted Activity Days	Ostro and Rothschild (1989)	18-64
Acute Bronchitis	Dockery et al. (1996)	8-12
Work Loss Days	Ostro (1987)	18-64
Lower Respiratory Symptoms	Schwartz and Neas (2000)	7-14
Asthma Exacerbation, Cough	Ostro et al. (2001)	6-18
Asthma Exacerbation, Shortness of Breath	Ostro et al. (2001)	6-18
Asthma Exacerbation, Wheeze	Ostro et al. (2001)	6-18
Upper Respiratory Symptoms	Pope et al. (1991)	9-11
Asthma Exacerbation, Cough	Vedal et al. (1998)	6-18

Premature Mortality

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

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

Particulate matter has been linked with premature mortality in adults (Laden, et al., 2006;Jerrett, et al., 2005;Pope, et al., 2002;Katsouyanni, et al., 2001;Samet, et al., 2000b) as well as infants (Bobak and Leon, 1999;Conceicao, et al., 2001;Loomis, et al., 1999;Woodruff, et al., 2008;Woodruff, et al., 1997) in multiple studies throughout the world. To estimate premature mortality in adults, we used an epidemiological analysis of the American Cancer Society cohort by Pope et al. (2002) and analysis of the

Six-City cohort by Laden et al (2006). To estimate premature mortality in infants, we used a study by Woodruff et al. (1997).

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 consecutive years, and affects roughly five percent of the U.S. population (American Lung Association, 2002b, Table 4). There are a limited number of studies that have estimated the impact of air pollution on new incidences of chronic bronchitis. Schwartz (1993) and Abbey et al. (1995c) provide evidence that long-term PM exposure can give rise to the development of chronic bronchitis in the U.S. A reduction in power plant emissions primarily reduces $PM_{2.5}$, so this analysis uses the Abbey et al study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of chronic bronchitis.

Non-Fatal Myocardial Infarction (Heart Attack)

Non-fatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the U.S. (Peters, et al., 2001) and other countries (Poloniecki, et al., 1997). We used the C-R function reported in Peters et al. (2001), the only available U.S. study to provide an estimate specifically for $PM_{2.5}$-related heart attacks. Other studies, such as Samet et al. (2000a) and Moolgavkar et al. (2000b), 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, it is useful 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 epidemiological studies (Gold, et al., 2000;Liao, et al., 1999;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 (Dekker, et al., 2000;Liao, et al., 1997;Tsuji, et al., 1996). As such, the reduction in heart rate variability due to PM is consistent with an increased risk of heart attacks.

Cardiovascular and Respiratory Hospital Admissions

Respiratory and cardiovascular hospital admissions are the two broad categories of hospital admissions that have been related to PM exposure. 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.

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 from a number of studies. We used a fixed/random effects approach, such as was used in the recent NonRoad Diesel Analysis (Abt Associates Inc., 2003). However, there is no single correct pooling procedure.

To estimate avoided cardiovascular hospital admissions associated with reduced $PM_{2.5}$, we use studies by Moolgavkar (2000b; 2003) and Ito (2003). 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 C-R functions for populations over 65, allowing us to pool the C-R functions for this age group. Only Moolgavkar (2000b) estimated a separate C-R function for populations age 20 to 64. Total cardiovascular hospital admissions are thus estimated as the sum of the pooled estimate for populations over 65 and the single study estimate for populations age 20 to 64.

Cardiovascular hospital admissions include admissions for myocardial infarctions (MIs). 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 respiratory hospital admissions, we use C-R functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, there are additional published studies showing a statistically significant relationship between PM_{10} and respiratory hospital admissions. However, we use only those focusing on $PM_{2.5}$. Both Moolgavkar (2000b; 2003) and Ito (2003) estimated C-R functions for COPD in populations over 65, allowing us to pool the C-R functions for this group. Only Moolgavkar (2000a) 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 age 20 to 64. In addition, Sheppard et al (1999) estimated a C-R function for asthma hospital admissions for populations under age 65. Total avoided PM-related respiratory hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

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). Another study, Schwartz et al. (1993), examined a broader age group (under 65) but focused on PM_{10} rather than $PM_{2.5}$. 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 but over 18.

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.

Acute Bronchitis

Around five percent of U.S. children between ages five and seventeen experience episodes of acute bronchitis annually (Adams and Marano, 1995). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the

MedlinePlus medical encyclopedia[1], 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 of 8 and 12 using a C-R function reported in Dockery et al. (1996).

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

Upper Respiratory Symptoms (URS)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary, and the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS), as defined below, 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.

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 LRS, we used a study by Schwartz and Neas (2000). Schwartz and Neas used logistic regression to link LRS in children with a variety of pollutants, including $PM_{2.5}$, 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. A total of 1,844 children were enrolled in 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.

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

Minor Restricted Activity Days (MRADs)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.

Work-Loss Days (WLDs)

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 surveys 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 health impact function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

Asthma Exacerbations

We pool the results of studies by Ostro et al. (2001) and Vedal et al. (1998) to derive an estimate of lower respiratory symptoms in asthmatics. In addition to the lower respiratory estimate, we include an upper respiratory estimate based on a study by Pope et al. (1991).

5. Economic Value of Reducing Adverse Health Impacts

This Chapter discusses some issues that arise in valuing avoided adverse health effects and then provides a summary table of the values that we use. Appendix F provides additional details on the individual effects and the methods we used.

5.1 Issues in Valuing Avoided Adverse Health Effects

This section discusses a number of issues that arise in valuing changes in health effects. We first discuss the use of ex-ante economic values. Second, we discuss updating our benefit estimates to account for inflation. Third, we discuss the possibility that as income changes, willingness-to-pay (WTP) would also change. Finally, we describe the derivation of the present discounted value of future benefits, such as in the case of premature mortality that may occur at some point in the future, relative to a reduction in emissions.

Ex-Ante Economic Values

The appropriate economic value for a change in a health effect depends on whether the health effect is viewed *ex ante* (before the effect has occurred) or *ex post* (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a small amount for a large population. The appropriate economic measure is therefore *ex ante* WTP for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk.

For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is $100, then the WTP for an avoided statistical premature mortality amounts to $1 million ($100/0.0001 change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect. For example, for the valuation of hospital admissions EPA used the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These COI estimates generally understate the true value of reductions in risk of a health effect, because, while they reflect the direct expenditures related to treatment, they omit the value of avoiding the pain and suffering from the health effect itself.

Updating Values for Inflation

The valuation functions were originally developed based on year 2000 $. To allow for the effect of inflation, we have adjusted these values to reflect prices in 2006 $. Because some functions are based on willingness to pay to avoid illness, while others are based on cost of illness and/or lost wages, three different inflation indices are used. These are the All Goods Index, the Medical Cost Index, and the Wage Index, respectively. Table 4 summarizes the values we used.

Table 4. Inflators and Health Effects Endpoints for Each Inflation Index

Index	Inflator from 2000 $ to 2006 $	Health Effects Endpoints
All Goods Index	1.171	Acute Bronchitis Asthma Exacerbation Chronic Bronchitis Lower Respiratory Symptoms Mortality Minor Restricted Activity Days Upper Respiratory Symptoms
Medical Cost Index	1.289	Emergency Room Visits Hospital Admissions
Wage Index	1.191	Acute Myocardial Infarction Hospital Admissions School Loss Days Work Loss Days

Growth in Unit Values Reflecting Growth in National Income

The unit value estimates reflect expected growth in real income over time. This is consistent with economic theory, which argues that WTP for most goods (such as health risk reductions) will increase if real incomes increase. There is substantial empirical evidence that the income elasticity of WTP for health risk reductions is positive, although there is uncertainty about its exact value (and it may vary by health effect). Although one might assume that the income elasticity of WTP is unit elastic (e.g., a 10 percent higher real income level implies a 10 percent higher WTP to reduce health risks), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefits estimates in two ways: through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and through differences in income between study populations and the affected populations at a particular time. Following the analysis in the CAIR regulatory impact assessment, we have focused on the former.

The income adjustment in PIE follows the approach used by EPA (2005b, p. 4-17), who adjusted the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, EPA applied estimates derived from cross-sectional studies.[1] The available income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income

[1] Details of the procedure can be found in Kleckner and Neumann (1999).

and changes in WTP. As a result, EPA (2005b, p. 4-18) used different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality (Table 5).

In addition to elasticity estimates, projections of real gross domestic product (GDP) and populations from 1990 to 200 are needed to adjust benefits to reflect real per capita income growth. For consistency with the emissions and benefits modeling, EPA (2005b, p. 4-17) used national population estimates for the years 1990 to 1999 based on U.S. Census Bureau estimates (Hollman, et al., 2000). These population estimates are based on an application of a cohort-component model applied to 1990 U.S. Census data projections (U.S. Bureau of the Census, 2000). For the years between 2000 and 2010, EPA applied growth rates based on the U.S. Census Bureau projections to the U.S. Census estimate of national population in 2000. EPA used projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010, and projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's (2000) for the years 2010 to 2020.

Using the method outlined in Kleckner and Neumann (1999) and the population and income data described above, EPA (2005b, p. 4-18) calculated WTP adjustment factors for each of the elasticity estimates. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) are adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. Table 5 lists the estimated adjustment factors.

Table 5. Elasticity Values and Adjustment Factors Used to Account for National Income Growth

Benefit Category	Central Elasticity Estimate	Adjustment Factor for 2010	Adjustment Factor for 2015	Adjustment Factor for 2020
Minor Health Effect	0.14	1.034	1.052	1.066
Severe & Chronic Health Effects	0.45	1.113	1.176	1.229
Premature Mortality	0.40	1.100	1.155	1.201

Note that because of a lack of data on the dependence of COI on income, and a lack of data on projected growth in average wages, no adjustments are made to benefits estimates based on the COI approach or to work loss days and worker productivity benefits estimates. This lack of adjustment would tend to result in an under-prediction of benefits in future years, because it is likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

Present Discounted Value of Avoiding Future Mortality

The delay, or lag, between changes in PM exposures and changes in mortality rates is not precisely known. The 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 estimated in chronic exposure studies versus daily mortality studies, suggests that it is likely that not all cases of avoided premature mortality associated with a given incremental reduction in PM exposure would occur in the same year as the exposure reduction.

Following EPA analyses (U.S. EPA, 2006, p. 5-21), we assume a 20-year lag structure, with 30 percent of premature deaths occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 20 percent occurring evenly over years 6 to 20 after the reduction in $PM_{2.5}$. It should be noted that the selection of a 20-year lag structure is not directly supported by any PM-specific literature. Rather, it is intended to be a best guess at the appropriate time distribution of avoided cases of PM-related mortality. As noted by EPA, the distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. Finally, it is important to keep in mind that changes in the lag assumptions do *not* change the total number of estimated deaths but rather the timing of those deaths.

Specifying the lag is important because people are generally willing to pay more for something now than for the same thing later. They would, for example, be willing to pay more for a reduction in the risk of premature death in the same year as exposure is reduced than for that same risk reduction to be received the following year. This time preference for receiving benefits now rather than later is expressed by discounting benefits received later. The exact discount rate that is appropriate (i.e., that represents people's time preference) is a topic of much debate. EPA has typically used a discount rate of three percent, and we use a three percent rate for this analysis in conjunction with the 20-year lag structure described above.

5.2 Summary of Valuation Functions Used in this Analysis

Table 6 presents a summary of the economic values that we use to estimate the benefits of reducing adverse health impacts. Appendix F presents details on the derivations of these values.

Table 6. Unit Values for Economic Valuation of Health Endpoints by Income Year (2006 $)

Health Endpoint	Age Range	Unit Value *2010*	*2015*	*2020*
Mortality	0 - 99	$7,300,000	$7,700,000	$8,000,000
Chronic Bronchitis	27 - 99	$440,000	$470,000	$490,000
Acute Myocardial Infarction, Nonfatal	0 - 24	$85,000	$85,000	$85,000
Acute Myocardial Infarction, Nonfatal	25 - 44	$96,000	$96,000	$96,000
Acute Myocardial Infarction, Nonfatal	45 - 54	$100,000	$100,000	$100,000
Acute Myocardial Infarction, Nonfatal	55 - 64	$180,000	$180,000	$180,000
Acute Myocardial Infarction, Nonfatal	65 - 99	$85,000	$85,000	$85,000
HA, All Cardiovascular (less AMI)	18 - 64	$29,000	$29,000	$29,000
HA, All Cardiovascular (less AMI)	65 - 99	$27,000	$27,000	$27,000
HA, Asthma	0 - 64	$10,000	$10,000	$10,000
HA, Chronic Lung Disease	65 - 99	$17,000	$17,000	$17,000
HA, Chronic Lung Disease (less Asthma)	18 - 64	$16,000	$16,000	$16,000
HA, Congestive Heart Failure	65 - 99	$20,000	$20,000	$20,000
HA, Dysrhythmia	65 - 99	$20,000	$20,000	$20,000
HA, Ischemic Heart Disease (less AMI)	65 - 99	$33,000	$33,000	$33,000
HA, Pneumonia	65 - 99	$23,000	$23,000	$23,000
Asthma ER Visits	0 - 17	$370	$370	$370
Acute Bronchitis	7 - 14	$430	$440	$440
Lower Resp. Symptoms	9 - 11	$19	$19	$19
Upper Resp. Symptoms	18 - 64	$30	$30	$31
MRAD	18 - 64	$61	$62	$63
Work Loss Days	18 - 99	**	**	**
Asthma Exacerbation, Cough	6 - 18	$52	$53	$53
Asthma Exacerbation, Shortness of Breath	6 - 18	$52	$53	$53
Asthma Exacerbation, Wheeze	6 - 18	$52	$53	$53

NOTE: Numbers rounded to two significant digits.
* Mortality value after adjustment for 20-year lag.
** County-specific median daily wage.

Appendix A: Description of Source-Receptor Matrix and Emissions Data

PIE estimates particulate matter levels using the Phase II source-receptor (S-R) matrix. The model is desirable because it is flexible, and can be used to quickly estimate the impact of emission changes on ambient $PM_{2.5}$ levels. The S-R matrix consists of fixed transfer coefficients that reflect the relationship between annual average $PM_{2.5}$ concentration values at a single receptor in each county (a hypothetical monitor located at the county centroid) and the contribution by $PM_{2.5}$ species to this concentration from each emission source (E.H. Pechan & Associates Inc., 1994).

Levy et al (Levy, et al., 2003) found that an earlier version of the S-R Matrix compared relatively well with the performance of CALPUFF, a comparatively sophisticated model often used in risk assessments. Using the emission impacts from seven power plants in northern Georgia, Levy et al reported that the two models yielded generally similar results for sulfates or primary PM2.5, with somewhat greater differences for nitrates. However, they carefully noted that this result may differ depending on the location of the emissions, as temperature and humidity are important considerations in the formation of ambient particles.[1]

The following sections summarize the development of the S-R matrix and the steps taken to apply the matrix to derive changes in air quality resulting from changes in emissions.

A.1 Development of S-R Matrix Transfer Coefficients

The S-R matrix is based on the Climatological Regional Dispersion Model (CRDM), which uses assumptions similar to the Industrial Source Complex Short Term model (ISCST3), an EPA-recommended short range Gaussian dispersion model (U.S. EPA, 1995). The CRDM incorporates terms for wet and dry deposition of primary and secondary species that constitute $PM_{2.5}$ and uses meteorological summaries (annual average mixing heights and joint frequency distributions of wind speed and direction) from 100 upper air meteorological sites throughout North America. This analysis employs meteorological data collected in 1990.

Relative to more sophisticated and resource-intensive three-dimensional modeling approaches, the CRDM does not fully account for all the complex chemical interactions that take place in the atmosphere in the secondary formation of PM. Instead it relies on more simplistic species dispersion–transport mechanisms supplemented with chemical conversion at the receptor location.

The CRDM uses Turner's sector-average approach (Turner, 1970), a probabilistic method in which relative frequencies of occurrence of combinations of wind and stability conditions at the emissions source are used to calculate the relative frequencies of transport in various sectors. This method is recommended for the estimation of long-term average pollutant concentrations (E.H. Pechan & Associates Inc., 1997).

The pollutant concentration in a destination sector is estimated as follows:

[1] Note that the version of the S-R Matrix used in PIE differs in some respects from the version tested by Levy et al (2003).

$$C_j(r) = \frac{2Q(r)}{y\sqrt{2\pi}} \sum_{i,k} \frac{f_{i,j,k}}{u_i \sigma_{z,k}} \exp\left[-\frac{1}{2}\left(\frac{H}{\sigma_{z,k}} \right)^2 \right]$$

where:

$C_j(r)$ = atmospheric concentration in destination sector *j* at distance *r*

$Q(r)$ = pollutant mass flux at distance *r*

y = sector width at distance *r*

$f_{i,j,k}$ = joint frequency of wind speed class *i*, wind direction *j*, and stability category *k*

$\sigma_{z,k}$ = vertical diffusion coefficient for stability category *k*

u_i = wind speed for wind class *i*

H = effective stack height of emissions source (= 0 for ground-level sources)

The sector width is calculated as:

$$y = \left(\frac{2\pi}{16} \right) r$$

Primary emissions from a county are assumed to always impact the county source county itself and are evenly distributed over a square with the same area as the county. A simple box model is used for each combination of wind speed and stability category. The vertical diffusion coefficient, σ_z, is then calculated at a downwind distance corresponding to the side of the square.[2] These assumptions are necessary since the spatial variation of emissions within a county cannot be provided for a national scale model.[3]

Additional adjustments are made to ensure a consistent distribution of pollutant species among areas in close proximity to the emissions source. Receptors at a distance less than the square root of the source area are assumed to receive the same concentration of pollutants as the source area. In addition, the destination sector width is constrained to be at least equal to the square root of the source area.

Equation (1) is applicable to both point and area sources, either ground-level or elevated, and results in a Gaussian distribution of pollutant mass in the vertical dimension. However, for long-range transport, emissions are distributed uniformly in the vertical between the top of the mixed layer and the ground.

[2] The vertical diffusion coefficient σ_z was calculated using a subroutine from EPA's ISC3 model. Atmospheric stabilities were assumed to be C class (slightly unstable) during the day and E class (slightly stable) at night. However, for wind speeds in excess of 6 m/s, stability was assumed to be neutral (class D).

[3] Actual measured concentrations would be expected to be higher than those modeled with these assumptions for a monitor located in, or generally downwind from, a portion of the county with emission densities much higher than the county average. On the other hand, concentrations would be expected to be lower if a monitor is located at the prevailing upwind edge of the county or in an area of relatively low emission density.

This occurs when the vertical diffusion parameter, σ_z , is equal to the height of the mixed layer, h_m. For such long-range situations, the sector–average limited mixing model of Turner (1970) estimates pollutant concentrations at a downward distance *r* from the source as:

$$C_j(r) = \frac{Q(r)}{h_m y} \sum_{i,k} \frac{f_{i,j,k}}{u_i}$$

The mass flux of a directly emitted primary species at distance *r* from the source is a function of the material initially emitted, the amount chemically converted to a secondary pollutant, and the amount deposited by wet and dry processes during the period of transport (time *t*) from the emission point to the receptor. This is calculated by solving the relevant differential equation(Latimer, 1993):

$$Q_p(t) = Q_0 e^{-(k_c + k_p)t}$$

where:

$Q_p(t)$ = primary pollutant mass flux at transport time *t*

Q_0 = initial emission rate

k_c = pseudo-first-order rate constant for chemical conversion of the primary species to the secondary species

k_p = pseudo-first-order rate constant for deposition of primary species, equal to the sum of the dry and wet deposition rate constants ($k_{pd} + k_{pw}$)

t = transport time

The mass flux of secondary pollutants is dependent upon the fraction of the primary species that is chemically converted in the atmosphere to the secondary species and the amount of the secondary species that is deposited by wet and dry deposition processes during the transport time *t* from the stack to the downwind receptor point at distance *r*. This is also calculated by solving the relevant differential equation(Latimer, 1993):

$$Q_s(t) = \frac{k_c Q_0}{k_c + k_p - k_s} \left(e^{-k_s t} - e^{-(k_c + k_p)t} \right)$$

where:

$Q_s(t)$ = mass flux of the secondary species at transport time *t*

Q_0 = initial emission rate

k_c = pseudo-first-order rate constant for chemical conversion of the primary species to the secondary species

k_p = pseudo-first-order rate constant for deposition of primary species, equal to the sum of the dry and wet deposition rate constants ($k_{pd} + k_{pw}$)

k_s = pseudo-first-order rate constant for deposition of secondary species, equal to the sum of the dry and wet deposition rate constants ($k_{sd} + k_{sw}$)

t = transport time

The model parameters used to estimate mass flux are detailed in Table 7. Note that the pseudo-first-order rate constant for deposition, k_p, is estimated from the dry and wet deposition velocities by dividing them by the mixing height (h_m).

Table 7. Pollutant-specific Model Parameters

	PM2.5, SOA	SO_2 *	NO_2	NH_3
Chemical Conversion Rate, k_c (%/hr) [RH = relative humidity (%)]	0	0.5 if RH < 40 1.5 if RH > 70 ((RH - 40)/30) + 0.5 otherwise	2	0
Dry Deposition Velocities (cm/s)	0.1	0.5	1	1
Wet Deposition Velocities (cm/s) [P = annual precipitation rate (in.)]	0.01 * P	0.003 * P	0.0003 * P	0.0003 * P

*** The chemical conversion rate for SO_2 was parameterized as a function of relative humidity to account for greater atmospheric conversion rates in areas of the country with higher humidity.**

**** Wet deposition velocities are from (Yamartino, 1985)**

Meteorological Data

Meteorological variables were calculated from rawinsonde data on the NAMER-WINDTEMP tapes[4] obtained from the National Climatic Data Center. Winds for each of 100 sites throughout North America were averaged for the following layers: the surface to 250 meters above ground level (m agl), 250-500 m agl, 500-1000 m agl, 1000-2000 m agl, and 2000-4000 m agl. For each of these levels and for each of the 100 meteorological sites, a joint frequency distribution of wind direction (16 cardinal directions) and wind speeds (11 speeds in 1 m/s increments) was calculated for 1990.

These distributions were calculated separately for the twice-daily soundings. The early morning soundings were assumed to be associated with the E stability category, and the late afternoon soundings were assumed to be associated with the C stability category. Mixing heights were determined from each

[4] Refers to North America wind and temperature. These are standard data tapes for upper-air (rawinsonde) data collected twice daily throughout North America. Rawinsondes are radar-tracked wind balloons.

sounding by calculating the virtual potential temperature. The annual average afternoon mixing heights were calculated for each of the 100 meteorological sites and were used to calculate the upper limit of vertical diffusion (h_m). The appropriate wind layer for concentration calculations was determined using the centroid of the diffusing plume: σ_z for a ground-based plume that has not yet mixed uniformly in the vertical, H for an elevated source, and $h_m/2$ for a uniformly mixed plume (E.H. Pechan & Associates Inc., 1994).

S-R Transfer Coefficients

The S-R matrix used in PIE estimated the transport of the following emissions species: (1) directly emitted $PM_{2.5}$ and secondary organic aerosols (SOA), (2) sulfur dioxide (SO_2), (3) nitrogen oxides (NO_x), and (4) ammonia (NH_3). These species were then used in the calculation of ambient concentrations of $PM_{2.5}$.

A matrix of source-receptor coefficients (in units of sec/m^3) spanning the entire contiguous U.S. was developed for each of the four pollutants using the CRDM. For a unique combination of source and receptor sites, a S-R transfer coefficient represents the incremental ambient air quality impact in μg/m^3 at the receptor resulting from a 1 μg/s unit emission from the source. The S-R matrix therefore provides a link between emission reductions and resulting air quality concentrations. Concentration reductions that occur in proportion to a decrease in emissions at a source are determined by the S-R coefficients for a given source and all receptors.

The pollutant concentration at a destination county is given by:

$$D_j^s = \sum_i \sum_c E_{c,i}^s T_{c,i,j}^s \cdot F^s \cdot F_{unit}$$

where:

D_j^s = Concentration of pollutant s at destination county j (μg/m^3)

$E_{i,c}^s$ = Emission of pollutant s from emissions category c in source county i (tons/year)

$T_{i,j}^s$ = Transfer coefficient for pollutant s from source county i to destination county j for emissions category c (sec/m^3)

F^s = Ionic conversion factor for pollutant s

F_{unit} = Unit conversion factor (28,778 μg-year/ton-sec)

The ionic conversion factors are molecular weight ratios used to adjust the transfer coefficients to reflect the concentration of precursors to secondarily-formed particulate species. Standard molecular weights along with the ionic conversion factors used in this analysis are given in Table 8 and Table 9.

Table 8. Standard Molecular Weights

Specie	Symbol	Standard molecular weight [5]
Nitrate ion	NO_3^-	62.0049
Sulfate ion	SO_4^{2-}	96.0626
Ammonium ion	NH_4^+	18.03846
Ammonium Nitrate	NH_4NO_3	80.04336
Ammonium Bisulfate	$NH_4\ SO_4$	114.1011
Ammonium Sulfate	$(NH_4)_2SO_4$	132.13952

Table 9. Ionic Conversion Factors

Species	Ionic conversion factor, F^s
PM_{25}, SOA	1
$SO_2 \rightarrow SO_4^{2-}$	96.0626 / 64.0638
$NO_2 \rightarrow NO_3^-$	62.0049 / 46.0055
$NH_3 \rightarrow NH_4^+$	18.03846 / 17.03052

A.2 Air Pollution Emissions Data

We use emissions data from the baseline scenario of the EPA Multipollutant Analyses[6] and the control scenario of the Clean Air Interstate Rule (CAIR) to forecast ambient 2010, 2015, and 2020 $PM_{2.5}$ levels.[7] The assumptions underlying the non-EGU emission inventories are detailed in the CAIR Emissions Inventory Technical Support Document (U.S. EPA, 2005a).

The development of the EGU emissions data is somewhat more involved. An initial set of EGU emission files were provided by EPA.[8] Working with David Schoengold, we then updated the EPA-supplied EGU emission data to reflect the latest information on forecasted EGU construction.[9]

In addition to the 2010, 2015, and 2020 emission inventories, we used a 2001 emissions inventory (initially developed for the Clean Air Interstate Rule) to help develop calibration factors (discussed briefly here and in more detail in a later section). Table 10 through Table 13 summarize the 2001, 2010, 2015, and 2020 emissions data for the continental U.S. that we used.

[5] Standard atomic weights from http://physics.nist.gov/PhysRefData/Compositions

[6] The Multipollutant Analyses (including CAIR) are described at: http://www.epa.gov/airmarkets/progsregs/cair/multi.html.

[7] Note that 2002 county-level natural emissions (from plants and soil) and were estimated using the BEIS 3.12 model (U.S. EPA, http://www.epa.gov/asmdnerl/biogen.html).

[8] EGU emission files were provided by Michael Cohen (cohen.michael@epa.gov) of the EPA Clean Air Markets Division via email to Donald McCubbin of Abt Associates on August 8, 2008.

[9] The file version of the EGU emissions file was provided by David Schoengold (schoengo@msbnrg.com) of MSB Energy Associates, Inc via email to Donald McCubbin of Abt Associates on December 22, 2008.

Table 10. 2001 Emissions Inventory Summary, by Tier 1 (tons/year)

Tier 1	NO_x	SO_2	$PM_{2.5}$	VOC	NH_3
Fuel Combustion Electric Utilities	4,905,369	10,832,338	582,708	60,164	10,819
Fuel Combustion Industrial	2,726,986	2,222,662	261,204	172,431	31,362
Fuel Combustion Other	775,970	639,339	446,932	943,664	8,182
Chemical & Allied Product Manuf.	103,659	342,204	47,217	261,839	26,835
Metals Processing	94,369	331,758	126,899	71,217	2,401
Petroleum & Related Industries	123,728	316,864	27,442	440,794	9,733
Other Industrial Processes	500,264	428,376	396,602	427,901	51,927
Solvent Utilization	4,442	1,177	17,393	5,012,183	405
Storage & Transport	14,432	5,518	36,276	1,191,651	5,011
Waste Disposal & Recycling	129,801	34,553	332,882	418,710	84,986
Highway Vehicles	8,064,067	271,033	161,373	4,709,818	277,378
Off-Highway Vehicles	4,050,800	433,252	307,540	2,584,530	1,753
Natural Sources	1,054,501			41,842,833	
Miscellaneous	202,056	49,277	2,499,120	569,908	3,178,800
Total	**22,750,444**	**15,908,352**	**5,243,587**	**58,707,645**	**3,689,592**

Table 11. 2010 Emissions Inventory Summary, by Tier 1 (tons/year)

Tier 1	NO_x	SO_2	$PM_{2.5}$	VOC	NH_3
Fuel Combustion Electric Utilities (CAIR)	2,587,285	6,440,630	542,138	54,807	3,239
Fuel Combustion Electric Utilities (Base)	3,844,746	10,078,349	690,592	55,618	3,262
Fuel Combustion Industrial	2,594,238	2,173,852	252,223	180,161	35,170
Fuel Combustion Other	835,509	678,813	353,487	511,233	8,703
Chemical & Allied Product Manuf.	106,680	337,520	52,351	235,982	31,321
Metals Processing	101,050	403,466	127,652	74,797	2,393
Petroleum & Related Industries	138,528	339,890	30,765	388,168	10,517
Other Industrial Processes	537,557	481,608	435,298	415,616	53,443
Solvent Utilization	5,111	1,331	20,149	5,060,918	476
Storage & Transport	15,187	5,354	32,503	954,741	6,441
Waste Disposal & Recycling	113,217	31,616	351,319	389,357	95,852
Highway Vehicles	4,682,898	27,439	91,719	2,593,284	341,532
Off-Highway Vehicles	3,282,481	219,034	250,625	1,903,532	2,069
Natural Sources	1,054,501	0	0	41,842,833	0
Miscellaneous	202,046	49,329	2,541,927	570,631	3,258,245
Total (CAIR)	**16,256,288**	**11,189,883**	**5,082,157**	**55,176,061**	**3,849,401**
Total (Base)	**17,493,369**	**14,798,311**	**5,225,722**	**55,175,899**	**3,849,424**

Table 12. 2015 Emissions Inventory Summary, by Tier 1 (tons/year)

Tier 1	NO_x	SO_2	$PM_{2.5}$	VOC	NH_3
Fuel Combustion Electric Utilities (CAIR)	2,410,242	5,404,793	515,273	62,128	5,062
Fuel Combustion Electric Utilities (Base)	3,982,613	9,427,003	735,320	64,725	5,122
Fuel Combustion Industrial	2,851,065	2,468,790	284,275	200,528	38,709
Fuel Combustion Other	887,989	724,593	334,047	427,218	9,069
Chemical & Allied Product Manuf.	120,137	377,936	59,585	273,896	34,685
Metals Processing	119,210	440,615	147,647	86,712	2,821
Petroleum & Related Industries	147,168	352,666	33,210	408,229	10,913
Other Industrial Processes	607,606	546,881	484,328	463,583	55,564
Solvent Utilization	6,317	1,492	25,283	5,556,458	547
Storage & Transport	17,241	5,928	36,950	934,336	7,427
Waste Disposal & Recycling	120,048	35,091	364,997	411,163	108,123
Highway Vehicles	3,152,447	30,823	70,696	2,031,629	379,364
Off-Highway Vehicles	2,912,633	232,632	217,787	1,648,429	2,264
Natural Sources	1,054,501	0	0	41,842,833	0
Miscellaneous	202,047	49,353	2,552,712	571,212	3,338,009
Total (CAIR)	**14,608,650**	**10,671,593**	**5,126,790**	**54,918,355**	**3,992,558**
Total (Base)	**16,061,807**	**14,515,027**	**5,316,378**	**54,914,328**	**3,992,618**

Table 13. 2020 Emissions Inventory Summary, by Tier 1 (tons/year)

Tier 1	NO_x	SO_2	$PM_{2.5}$	VOC	NH_3
Fuel Combustion Electric Utilities (CAIR)	2,469,975	4,667,340	568,571	69,343	4,374
Fuel Combustion Electric Utilities (Base)	4,065,227	9,206,392	806,964	71,993	4,389
Fuel Combustion Industrial	2,922,582	2,318,894	288,132	206,136	39,019
Fuel Combustion Other	901,103	713,617	308,266	339,333	8,844
Chemical & Allied Product Manuf.	135,498	424,914	67,336	316,015	38,372
Metals Processing	139,437	540,307	171,021	99,430	3,312
Petroleum & Related Industries	157,185	369,602	36,134	439,705	11,398
Other Industrial Processes	684,907	619,487	536,368	514,079	57,000
Solvent Utilization	7,083	1,662	28,387	5,847,730	616
Storage & Transport	19,525	6,600	41,427	920,863	8,376
Waste Disposal & Recycling	126,739	38,507	379,661	434,112	119,907
Highway Vehicles	3,054,847	30,826	70,700	2,002,251	385,189
Off-Highway Vehicles	2,666,512	279,202	193,001	1,527,842	2,457
Natural Sources	1,054,501	0	0	41,842,833	0
Miscellaneous	202,048	49,375	2,509,136	571,760	3,417,741
Total (CAIR)	**14,541,943**	**10,060,333**	**5,198,141**	**55,131,433**	**4,096,604**
Total (Base)	**16,019,260**	**14,420,874**	**5,406,230**	**55,127,486**	**4,096,619**

When modeling emission sources, we categorized them into *elevated point sources* and *area/mobile sources*. For each, we calculate an "effective stack" height, which takes into account the actual stack height, gas temperature and velocity, stack diameter, and other factors. The effective stack height is important as it significantly affects the ability of emissions to disperse – generally the taller the effective stack the further the emissions might go. In calculating effective stack height, we assume an average wind speed of 5 meters per second using the plume rise algorithm from ISCST3(U.S. EPA, 1995).

We group stationary point source emissions for each county into three groups based on effective stack height: (1) less than 250 meters, (2) 250 to 500 meters, and (3) greater than 500 meters. We assume that emissions from the two groups less than 500 meters originate from the center of the county in which they are located. For point sources with effective stack heights greater than 500 meters, we use their true latitude and longitude coordinates when modeling the dispersion of emissions.

Emissions from both ground-level mobile and area sources in the contiguous U.S.are combined at the county-level and modeled as emissions from stacks with an effective stack height of zero located at the source county centroid. Table 14 summarizes these emission categories.

Table 14. Emissions Categories for the S-R Matrix

Emissions Category	Effective Stack Height	Modeled Location
U.S. area and mobile emissions	0m	County center
U.S. elevated point emissions	0-250m	County center
U.S. elevated point emissions	250-500m	County center
U.S. elevated point emissions	>500m	True location

A.3 Atmospheric Chemistry

The S-R Matrix tracks the movement of both directly emitted particles as well as gases that react with each other to form "secondary" $PM_{2.5}$, ammonium sulfate, ammonium nitrate, and secondary organic aerosols (SOA). We should note that the air chemistry is greatly simplified, relative to state-of-the-art air quality models. Calibrating the modeling results to actual $PM_{2.5}$ monitor (as discussed in the next section) helps to make some of these simplifications less problematic. Nevertheless some uncertainty remains. Below we describe these secondary reactions.

Ammonium Sulfate & Ammonium Nitrate

In the presence of sulfate and nitrate ions, ammonium reacts preferentially with sulphate to form ammonium sulfate; ammonium nitrate is formed under conditions of excess ammonium and low temperatures. For each destination county, the ammonium sulfate – ammonium nitrate equilibrium is subject to the following simplifying assumptions regarding atmospheric chemistry that form these particulate compounds:

1a. We first compare the moles of ammonium and sulfate. If the mole ratio of ammonium to sulfate is **less than one**, then we assume that only a portion of sulfate converts to ammonium bisulfate and the rest remains as sulfate.

$$NH_4HSO_4 = (115.1088 / 96.0626)\ SO_4$$

1b. If the mole ratio of ammonium to sulfate is between **one and two**, then we assume that all sulfate converts to ammonium bisulfate, and a portion of the ammonium bisulfate converts to ammonium sulfate.

$$(NH_4)_2SO_4 = (132.1392 / 115.1088) \cdot NH_4HSO_4$$

1c. If the mole ratio of ammonium to sulfate is **greater than two**, then we assume that all sulfate converts to ammonium nitrate.

$$(NH_4)_2SO_4 = (132.1392 / 96.0626) \cdot SO_4$$

2. Any ammonium remaining after the sulfate neutralization process is estimated as:

$$(NH_4)_{remaining} = (NH_4)_{total} - (2 \cdot 18.0383 / 96.0626) \cdot SO_4$$

3. Ammonium nitrate formation is limited by the relative concentrations of nitrate and ammonium remaining after the sulfate neutralization process. The amount of nitrate that is neutralized by ammonium is estimated as:

if $(NH_4)_{remaining} > 0$ then $(NO_3)_{neutralized} = 62.0049 \cdot \min[(NO_3 / 62.0049) \cdot (62.0049 / 18.0383) \cdot (NH_4)_{remaining}]$ otherwise $(NO_3)_{neutralized} = 0$

4. Particulate ammonium nitrate is stable at relatively low temperatures. Following prior usage of the S-R Matrix (e.g., NOx SIP Call) we assume that nitrate converts to ammonium nitrate only a quarter of the time. The annual average concentration of ammonium nitrate formed by the neutralization process is therefore:

$$NH_4NO_3 = 0.25 \cdot (80.0432 / 62.0049) \cdot (NO_3)_{neutralized}$$

5. The concentration of $PM_{2.5}$ at the destination county is estimated as the sum of direct particulate emissions (direct PM2.5 and SOA) and secondary ammonium nitrate and sulfate.

Secondary Organic Aerosols (SOA)

We calculate the formation of SOA using a fixed relationship between SOA and VOC for each Tier 3 emission category.[10] The inventory for the CAIR rule (U.S. EPA, 2005a) estimated VOC but did not estimate SOA, so we developed a simple approach to estimate the conversion of VOC to SOA. Ideally the conversion depends upon a number of factors including climate and the type of VOC. To account for these factors, we used the 2010 inventory of SOA and VOC emissions generated for the Clear Skies Rule.[11] For each state and Tier 3 emission category in this inventory, we calculated the ratio of SOA to VOC. We then used these state- and Tier 3 category-specific ratios to estimate SOA in the CAIR emission inventory:

$$SOA_{CAIR,\,State,\,Tier\,3} = VOC_{CAIR,\,State,\,Tier\,3} \cdot \left(\frac{SOA_{Clear\ Skies,\,State,\,Tier\,3}}{VOC_{Clear\ Skies,\,State,\,Tier\,3}} \right)$$

[10] The emissions inventory in PIE has fourteen broad Tier 1 categories (e.g., on-road motor vehicles), and within each of these larger categories there are Tier 2 (e.g., diesels), and Tier 3 (e.g., heavy duty diesels) categories.

[11] U.S. EPA. 2010 emissions projections developed for the 2003 Technical Analysis of the Clear Skies Act. Online at ftp://ftp.epa.gov/modelingcenter/Clear_skies/CSA2003/Emissions/ [downloaded on 12/08/2003]

A.4 Calibration of S-R Matrix to Monitoring Data

To ensure the modeling estimates are as accurate as possible, we calibrated the S-R Matrix model estimates to actual monitoring data. We did this on a county-by-county basis, estimating the calibration factors using a 2001 emission inventory developed for the CAIR rule (U.S. EPA, 2005a) and data from Federal Reference Method (FRM) and EPA's Speciation Network (STN) monitor sites for 2002 obtained from the EPA.

First, we used the S-R Matrix with the 2001 inventory to estimate PM2.5 levels at the center of each county. Second, we used Voronoi Neighbor Averaging (VNA)[12] to spatially interpolate the $PM_{2.5}$ monitor data to generate a monitor-based estimate for each county center using the following algorithm:

- For cells with an FRM monitor within 50km, use only neighboring FRM monitors and inverse distance (1/d) weighting
- For cells with an FRM monitor within 50km to 100km, use neighboring FRM and ESPN monitors and 1/d weighting
- For cells with no FRM monitors within 100km, use only neighboring ESPN monitors and inverse distance squared ($1/d^2$) weighting

This method allows for a regional background of $PM_{2.5}$ (determined primarily by ESPN monitors) with higher concentrations in urban areas characterized by FRM monitors.

We calculated a "calibration factor" for each county by dividing our monitor estimate by the model estimate. For each state, Table 15 gives the average of the county-level monitor and model values as well as the ratio of the two (the ratio being the average of the calibration factors).

When calculating future year $PM_{2.5}$ levels, we used our calibration factor to adjust our model estimate for each county in the following way:

$$Calibrated\ \ Model\ \ PM2.5_{2010,\,County} = Model\ \ PM2.5_{2010,\,County} \cdot \left(\frac{Interpolated\ \ PM2.5\ \ Monitor_{2001,\,County}}{Model\ \ PM2.5_{2001,\,County}} \right)$$

[12] All spatial interpolations were conducted using the EPA's Benefits Mapping and Analysis Program (BenMAP), available online at http://www.epa.gov/ttn/ecas/benmodels.html. For details about the VNA algorithm, consult the BenMAP User Manual, available online at http://www.epa.gov/ttn/ecas/models/modeldoc.pdf.

Table 15. Monitor & Model Averages (ug/m3) and Average of Monitor to Model Ratios by State

State	Monitor	Model	Ratio	State	Monitor	Model	Ratio
AL	13.4	25.0	0.55	NE	7.1	10.1	0.70
AZ	5.8	65.1	0.15	NV	4.5	30.2	0.24
AR	11.3	19.9	0.57	NH	8.6	17.0	0.51
CA	11.9	44.4	0.34	NJ	12.2	26.2	0.47
CO	6.1	12.6	0.51	NM	4.9	23.9	0.24
CT	11.8	22.5	0.53	NY	11.2	21.0	0.59
DC	15.4	33.4	0.46	NC	12.9	19.6	0.67
DE	13.0	26.3	0.50	ND	4.9	8.6	0.58
FL	9.7	24.9	0.41	OH	15.3	20.4	0.76
GA	13.5	22.1	0.62	OK	9.5	15.1	0.65
ID	7.7	20.4	0.44	OR	7.9	24.9	0.43
IL	13.5	18.4	0.76	PA	13.6	21.3	0.67
IN	14.8	20.6	0.73	RI	9.5	14.6	0.65
IA	10.2	11.8	0.88	SC	12.6	23.3	0.55
KS	7.7	11.2	0.67	SD	6.0	8.8	0.69
KY	13.8	18.3	0.76	TN	13.2	19.8	0.68
LA	10.8	25.3	0.46	TX	8.6	14.7	0.64
ME	8.4	17.4	0.52	UT	6.7	17.2	0.46
MD	13.7	26.0	0.55	VT	9.8	13.7	0.72
MA	10.9	21.5	0.57	VA	13.3	19.7	0.69
MI	10.6	15.6	0.68	WA	7.3	12.7	0.69
MN	7.7	13.9	0.57	WV	14.4	18.0	0.81
MS	12.0	22.8	0.53	WI	9.7	13.9	0.70
MO	11.8	17.0	0.73	WY	4.6	19.4	0.25
MT	5.3	10.7	0.51				

Appendix B: Derivation of Health Impact Functions

This appendix reviews the steps we performed in taking models from the epidemiological study and converting them into health impact functions, which we then use to quantify the change in adverse health effects due to a change in air pollution exposure. The most common functional forms the log-linear and logistic, with a linear model used in some cases. All three are discussed below.

Note that the log-linear and logistic generally produce comparable results, so the fact that some health impacts are estimated with a logistic function and others with a log-linear function is not a cause for concern. Indeed, in some circumstances, such as for small changes in air pollution, the logistic and log-linear produce essentially the same result.

B.1 The Linear Model

A linear model between the adverse health effect, y, and the pollutant concentration, x, is of the form

$$y = \alpha + \beta \cdot x$$

A linear model includes the factors that are believed to affect the incidence of the health effect, of which the pollutant would be one. So, the variable "α" in the linear function consists of all the other independent variables in the regression, typically evaluated at their mean values, times their respective coefficients.

The function describing the relationship between a *change* in x and the corresponding *change* in incidence (rate) of the health effect from the baseline level (y_0) to the post-control level (y_c) is then:

$$\Delta y = y_c - y_0 = \beta \cdot (x_c - x_0) = \beta \cdot \Delta x \, .$$

If y denotes an incidence rate, then Δy denotes the change in the incidence rate. The expected number of cases avoided would then be calculated by multiplying this Δy by the relevant population. If y denotes an incidence count, then the β is first divided the baseline study population to generate an incidence rate. The expected number of cases avoided can then be calculated by multiplying Δy by the relevant population of interest:

$$CasesAvoided = \beta \cdot \Delta x \cdot pop.$$

The coefficient, β, and standard error of β (σ_β) are reported directly in studies presenting results from linear regression models.

B.2 The Log-linear Model

The most commonly used functional form for criteria air pollutant concentration-response functions is the log-linear model. It defines the relationship between x and y to be of the form:

$$y = B \cdot e^{\beta \cdot x}$$

or, equivalently,

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

where the parameter B is the incidence (rate), y, when the pollutant concentration, x, is zero; the parameter β is the coefficient of x; ln(y) is the natural logarithm of y; and α = ln(B).[1]

Estimating Avoided Cases

The relationship between Δx and Δy is:

$$\Delta y = y_c - y_0 = Be^{\beta \cdot x_c} - Be^{\beta \cdot x_0} \,.$$

This may be rewritten as:

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

where y_0 is the baseline incidence (rate) of the health effect -- i.e., the incidence (rate) before the change in x. If y is incidence rate rather than incidence, then the change in incidence rate, Δy, must be multiplied by the relevant population to get the expected number of cases avoided. For example, if y denotes the annual number of cases of the adverse health effect per 100,000 population, and pop denotes the population, then the expected number of cases avoided is calculated as

$$CasesAvoided = \Delta y \cdot (\frac{pop}{100{,}000}).$$

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

Estimating the Coefficient (β)

Epidemiological studies that estimate log-linear concentration-response functions often report a relative risk for a specific Δx, rather than the coefficient, β, in the function itself. The relative risk (RR) is simply the ratio of two risks corresponding to two levels of pollutant concentration – the "high" risk (corresponding to the higher pollutant level, $x = x_{high}$) and the lower risk (corresponding to the lower pollutant level, $x = x_{low}$):

$$RR = \frac{y_{high}}{y_{low}} .$$

Using the original log-linear function above, it can be shown that the relative risk associated with a specific change in pollutant concentration of Δx* = xhigh - xlow can be written as

$$RR_{\Delta x^*} = \frac{y_{high}}{y_{low}} = e^{\beta \cdot \Delta x^*} .$$

Taking the natural log of both sides, the coefficient in the function underlying the relative risk can be derived as:

$$\beta = \frac{\ln(RR)}{\Delta x^*} .$$

Once the pollutant coefficient, β, has been calculated, the change in incidence (rate), Δy, corresponding to any change in pollutant concentration, Δx, can be calculated, using the relationship between Δx and Δy given above, the baseline incidence (rate) and assessment population.

Estimating the Standard Error of β (σ_β)

The standard error of β (σ_β) is not often directly reported in studies presenting results from log-linear regression models. Results are most commonly presented as a relative risk and 95% confidence interval. The 95% confidence interval is defined as follows:

$$95\%CI = e^{(\beta \cdot \Delta x \pm 1.96 \cdot \sigma_\beta \Delta x)}$$

Based on this equation, the standard error of β (σ_β) can be estimated from the relative risk (RR), upper limit of the 95% confidence interval (UL), and lower limit of the 95% confidence interval (LL), as follows:

$$\sigma_{\beta,\,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(UL)}{\Delta x} - \frac{\ln(RR)}{\Delta x}\right)}{1.96}$$

$$\sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(RR)}{\Delta x} - \frac{\ln(LL)}{\Delta x}\right)}{1.96}$$

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

Some studies report only a central effect estimate and t-statistic. The t-statistic describes the strength of the observed pollutant-health effect association. It is defined as the ratio of the coefficient, β, to the standard error of β (σ_{β}). The standard error of β (σ_{β}) can, therefore, be estimated from the t-statistic as follows:

$$\sigma_{\beta} = \frac{\beta}{t}.$$

The Log-Linear Model: An Example

Lippmann et al. (2000) reported a relative risk (RR) of 1.045 for premature (non-accidental) mortality associated with an increase in daily $PM_{2.5}$ of 36 μg/m^3 in Detroit, MI. The $PM_{2.5}$ coefficient in the C-R function from which the RR was derived was back-calculated to be:

$$\beta = \frac{\ln(1.045)}{36} = 0.001223.$$

Suppose we use the C-R function from Lippmann et al. (2000) to estimate the change in incidence of premature deaths in Wayne County, MI (which contains Detroit) in the year 2000 resulting from a decrease in $PM_{2.5}$ concentration of 15 μg/m^3 per day. The baseline incidence of non-accidental mortality in Detroit is estimated to be 891.12 per year per 100,000 general population, or 2.441 per day per 100,000 general population. The population of Wayne County, MI in the year 2000 is estimated to be 2,061,162. The inputs to this calculation are:

Δx = -15

y_0 = 2.441 per day per 100,000 general population

β = 0.001223

general population of Wayne County, MI = 2,061,162.

The number of avoided premature deaths per day is estimated to be:

$$\Delta y = y_0 (e^{\beta * \Delta x} - 1) * (pop / 100{,}000)$$

$$= 2.441 * (e^{-0.001223 * 15} - 1) * 20.61162$$

$$= -0.914503325$$

That is, a decrease in $PM_{2.5}$ of 15 μg/m^3 per day is predicted to result in 0.914503325 premature deaths avoided per day in Wayne County, MI. Over the year (the year 2000 was a leap year, and so had 366 days), that's

0.914503325*366

= 334.7 premature deaths avoided.

B.3 The Logistic Model

In some epidemiological studies, a logistic model is used to estimate the probability of an occurrence of an adverse health effect. Given a pollutant level, x, and a vector of other explanatory variables, Z, the logistic model assumes the probability of an occurrence is:

$$y = prob\left(occurrence|\beta x, \alpha Z\right) = \left(\frac{e^{\beta x} e^{\alpha Z}}{1 + e^{\beta x} e^{\alpha Z}}\right),$$

where β is the coefficient of the pollutant concentration, x, and α is a vector of coefficients of the variables in the vector Z.[2]

Estimating Avoided Cases

The *change* in the probability of an occurrence (Δy) corresponding to a change in the level of the pollutant from x_c to x_0 (= Δx), all other covariates held constant, may be derived from the original C-R function above:

$$\Delta y = y_c - y_0 = \frac{y_0}{\left(1 - y_0\right) \cdot e^{-\Delta x \cdot \beta} + y_0} - y_0 .$$

Once again, to calculate the expected number of avoided cases of the adverse effect, it is necessary to multiply by the population:[3]

$$CasesAvoided = \Delta y \cdot pop.$$

[2] Greene (1997, Chapter 19) presents models with discrete dependent variables; in particular, page 874 presents the logit model. See also Judge et al.(1985, p. 763).

[3] Note that because Δy here is a change in probability of occurrence (rather than a change in the rate per 100,000 population), it is necessary to multiply by the population rather than by the population/100,000.

Estimating the Coefficient (β)

The estimated pollutant coefficient, β, in the original function is typically not reported in studies that use the logistic model. Instead, the odds ratio corresponding to a specific change in x is reported.

The odds of an occurrence is defined as:

$$odds = \frac{y}{1-y}.$$

It can be shown that

$$odds = \frac{y}{1-y} = e^{\beta x} e^{\alpha Z}$$

The *odds ratio* is just the ratio of the odds when the pollutant is at a specified higher level, x_{high}, to the odds when the pollutant is at a specified lower level, x_{low}:

$$oddsratio = \frac{odds_{high}}{odds_{low}} = \frac{e^{\beta x_{high}}}{e^{\beta x_{low}}} = e^{\beta(x_{high} - x_{low})}.$$

Often the odds ratio corresponding to a specified change in x, call it Δx*, is the only measure of the effect of x reported from a study using a logistic model (just as the relative risk corresponding to a specified change in x is often the only measure of the effect of x reported from a study using a log-linear model). However, it is easy to calculate the underlying pollutant coefficient, β, from the odds ratio as follows:

$$odds\,ratio = e^{\beta \Delta x^*}$$

$$\ln(odds\ ratio) = \beta \Delta x^* \Rightarrow \beta = \frac{\ln(oddsratio)}{\Delta x^*}.$$

Given the pollutant coefficient, β, and the baseline probability of occurrence, y_0, the change in the probability, Δy, associated with any change in pollutant concentration, Δx, can be derived using the equation for Δy above. The expected number of avoided cases of the adverse effect is then obtained by multiplying by the population.

Estimating the Standard Error of β (σ_β)

The standard error of β (σ_β) is not often directly reported in studies presenting results from logistic regression models. Results are most commonly presented as an odds ratio and 95% confidence interval. The 95% confidence interval is defined as follows:

$$95\%\ CI = e^{(\beta\Delta x \pm 1.96\sigma_\beta)}$$

Based on this equation, the standard error of β (σ_β) can be estimated from the odds ratio (OR), upper limit of the 95% confidence interval (UL), and lower limit of the 95% confidence interval (LL), as follows:

$$\sigma_{\beta,\,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{\left(\frac{\ln(UL)}{\Delta x} - \frac{\ln(OR)}{\Delta x}\right)}{1.96}$$

$$\sigma_{\beta,\,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(\frac{\ln(OR)}{\Delta x} - \frac{\ln(LL)}{\Delta x}\right)}{1.96}$$

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

Some studies report only a central effect estimate and t-statistic. The t-statistic describes the strength of the observed pollutant-health effect association. It is defined as the ratio of the coefficient, β, to the standard error of β (σ_β). The standard error of β (σ_β) can, therefore, be estimated from the t-statistic as follows:

$$\sigma_\beta = \frac{\beta}{t}.$$

The Logistic Model: An Example

Schwartz and Neas (2000) reported an odds ratio of 1.33 for lower respiratory symptoms (LRS) among school children, ages 7 - 14, corresponding to an increase in daily $PM_{2.5}$ concentration of 15 μg/m^3. The $PM_{2.5}$ coefficient in the logistic C-R function from which the odds ratio was derived is back-calculated as

$$\beta = \frac{\ln(1.33)}{15} = 0.019012\ .$$

The baseline incidence rate, y_0, (the probability per child per day of lower respiratory symptoms) was estimated in the study to be 0.0012.

Suppose we use the logistic C-R function from Schwartz and Neas (2000) to estimate the number of days with LRS avoided among schoolchildren, ages 7-14, in St. Louis during the warm months of April through August (the months used in the study) if $PM_{2.5}$ concentrations were reduced by 10 μg/m^3 each day. The inputs to this calculation are:

$\Delta x = x_c - x_0 = -10$

$y_0 = 0.0012$

$\beta = 0.019012$

the number of days in April through August = 153

the number of children, ages 7 - 14, in St. Louis area (9 counties) = 307,170.

The number of avoided LRS days among children ages 7-14 in the St. Louis area resulting from a decrease of 10 μg/m^3 $PM_{2.5}$ per day is estimated to be

$$\Delta y = \left[\frac{y_0}{(1 - y_0) * e^{-\beta * \Delta x} + y_0} - y_0 \right] * pop$$

$$= \left[\frac{0.0012}{(1 - 0.0012) * e^{-0.019012 * (-10)} + 0.0012} - 0.0012 \right] * 307{,}170$$

$$= -0.0002076 * 307{,}170 = -63.7568 \text{ per day.}$$

There are 153 days in April through August, for a total of -63.7568*153 =

-9754.79, or

9,754.79 LRS days avoided.

Appendix C: Health Impact Functions

In this Appendix, we present the health impact functions used to estimate PM-related adverse health effects. Each sub-section has an Exhibit with a brief description of the Health impact function and the underlying parameters. Following each Exhibit, we present a brief summary of each of the studies and any items that are unique to the study.

Note that Appendix B mathematically derives the standard types of health impact functions that we encountered in the epidemiological literature, such as, log-linear, logistic and linear, so we simply note here the type of functional form. Finally, Appendix D presents a description of the sources for the incidence and prevalence data used in these health impact functions.

Exhibit C-1. Health Impact Functions for Particulate Matter and All-Cause Mortality

Author	Year	Location	Age	Metric	Beta	Std Err	Form	Notes
Laden et al.	2006	6 cities	25-99	Annual	0.014842	0.004170	Log-linear	
Pope et al.	2002	51 cities	30-99	Annual	0.005827	0.002157	Log-linear	
Woodruff et al.	1997	86 cities	0-0	Annual	0.003922	0.001221	Logistic	

C.1 Mortality

Both long- and short-term exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most significant health endpoint quantified in this analysis. We include mortality in adults, as well as infants.

Mortality, All Cause (Pope, et al., 2002)

The Pope et al. (2002) analysis is a longitudinal cohort tracking study that uses the same American Cancer Society 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.[1] 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_4 (as well as SO_2), and all-cause, cardiopulmonary, lung cancer and “all other” mortality.

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 (1.06) and 95% confidence interval (1.02-1.11) associated with a change in annual mean exposure of 10.0 $\mu g/m^3$ (Pope, et al., 2002, Table 2).

Functional Form: Log-linear
Coefficient: 0.005827

[1] 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.

Standard Error: 0.002157
Incidence Rate: county-specific annual all-cause mortality rate per person ages 30 and older
Population: population of ages 30 and older.

Mortality, All Cause – Laden, et al. (2006)

Laden et al (2006) performed an extended mortality follow-up for eight years in a period of reduced air pollution concentrations using data from the Harvard Six Cities adult cohort study. They used annual city-specific $PM_{2.5}$ concentrations measured from1979-1988, and estimated the air quality data for the subsequent eight years using publicly available data. The authors used a Cox proportional hazards regression controlling for individual risk factors to examine the relationship between long-term exposure to $PM_{2.5}$ and mortality. Laden et al found a significant increase in the overall mean mortality associated with a 10-$\mu g/m^3$ increase in $PM_{2.5}$.

The coefficient and standard error are estimated from the relative risk (1.16) and 95% confidence interval (1.07-1.26) associated with a 10-$\mu g/m^3$ increase in $PM_{2.5}$ (Laden, et al., 2006, p. 667).

Functional Form: Log-linear
Coefficient: 0.01484
Standard Error: 0.00417
Incidence Rate: county-specific annual all cause mortality rate per person ages 25 and older
Population: population of ages 25 and older.

Infant Mortality (Woodruff, et al., 1997)

In a study of four million infants in 86 U.S. metropolitan areas conducted from 1989 to 1991, Woodruff et al. (1997) found a significant link between PM_{10} exposure in the first two months of an infant's life with the probability of dying between the ages of 28 days and 364 days. PM_{10} exposure was significant for all-cause mortality. PM_{10} was also significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants.

The coefficient and standard error are based on the odds ratio (1.04) and 95% confidence interval (1.02-1.07) associated with a 10 $\mu g/m^3$ change in PM_{10} (Woodruff, et al., 1997, Table 3).

Functional Form: Logistic
Coefficient: 0.003922
Standard Error: 0.001221
Incidence Rate: county-specific annual post-neonatal[13] infant deaths per infant under the age of one
Population: population of infants under one year old.

[13] Post-neonatal refers to infants that are 28 days to 364 days old.

Table 16. Health Impact Functions for Particulate Matter and Chronic Illness

Endpoint Name	Author	Year	Location	Age	Metric	Beta	Std Error	Functional Form
Chronic Bronchitis	Abbey et al.	1995	California	27-99	Annual	0.013185	0.006796	Logistic
Acute Myocardial Infarction, Nonfatal	Peters et al.	2001	Boston, MA	18-99	D24HourMean	0.024121	0.009285	Logistic

C.2 Chronic Illness

We include two types of chronic illness: chronic bronchitis and non-fatal heart attacks. Non-fatal heart attacks are considered "chronic" because the impact is long-lasting and this is reflected in its valuation (discussed in Appendix F).

Chronic Bronchitis (Abbey, et al., 1995b)

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

The estimated coefficient (0.0137) is presented for a one $\mu g/m^3$ change in $PM_{2.5}$ (Abbey, et al., 1995b, Table 2). The standard error is calculated from the reported relative risk (1.81) and 95% confidence interval (0.98-3.25) for a 45 $\mu g/m^3$ change in $PM_{2.5}$ (Abbey, et al., 1995b, Table 2).

Functional Form: Logistic
Coefficient: 0.0137
Standard Error: 0.00680
Incidence Rate: annual bronchitis incidence rate per person (Abbey, et al., 1993b, Table 3) = 0.00378
Population: population of ages 27 and older[1] without chronic bronchitis = 95.57%[2] of population 27+.

Acute Myocardial Infarction (Heart Attacks), Nonfatal (Peters, et al., 2001)

Peters et al. (2001) studied the relationship between increased particulate air pollution and onset of heart attacks in the Boston area from 1995 to 1996. The authors used air quality data for PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$,"black carbon", O_3, CO, NO_2, and SO_2 in a case-crossover analysis. For each subject, the case period was matched to three control periods, each 24 hours apart. In univariate analyses, the authors observed a positive association between heart attack occurrence and $PM_{2.5}$ levels hours before and days before onset. The authors estimated multivariate conditional logistic models including two-hour and twenty-four hour pollutant concentrations for each pollutant. They found significant and independent associations between heart attack occurrence and both two-hour and twenty-four hour $PM_{2.5}$ concentrations before onset. Significant associations were observed for PM_{10} as well. None of the other

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

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

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 $\mu g/m^3$ increase in twenty-four hour average $PM_{2.5}$ (Peters, et al., 2001, Table 4, p. 2813).

Functional Form: Logistic
Coefficient: 0.024121
Standard Error: 0.009285
Incidence Rate: region-specific daily nonfatal heart attack rate per person 18+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410) [3]
Population: population of ages 18 and older.

[3]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.

Table 17. Health Impact Functions for Particulate Matter and Hospital Admissions

Endpoint Name	Author	Year	Location	Age	Beta	Std Error	Functional Form
Congestive Heart Failure	Ito	2003	Detroit, MI	65-99	0.003074	0.001292	Log-linear
Dysrhythmia	Ito	2003	Detroit, MI	65-99	0.001249	0.002033	Log-linear
Ischemic Heart Disease (less AMI)	Ito	2003	Detroit, MI	65-99	0.001435	0.001156	Log-linear
Chronic Lung Disease	Ito	2003	Detroit, MI	65-99	0.001169	0.002064	Log-linear
Pneumonia	Ito	2003	Detroit, MI	65-99	0.003979	0.001659	Log-linear
All Cardiovascular (less AMI)	Moolgavkar	2000	Los Angeles, CA	18-64	0.001400	0.000341	Log-linear
Chronic Lung Disease (less Asthma)	Moolgavkar	2000	Los Angeles, CA	18-64	0.002200	0.000733	Log-linear
All Cardiovascular (less AMI)	Moolgavkar	2003	Los Angeles, CA	65-99	0.001580	0.000344	Log-linear
Chronic Lung Disease	Moolgavkar	2003	Los Angeles, CA	65-99	0.001850	0.000524	Log-linear
Asthma	Sheppard	2003	Seattle, WA	0-64	0.003324	0.001045	Log-linear

C.3 Hospitalizations

We include two main types of hospital admissions – respiratory (pneumonia, COPD, and ashtma) and cardiovascular (all types, including ischemic heart disease, dysrhythmia, and heart failure).

Hospital Admissions for Asthma (Sheppard, et al., 1999;2003)

Sheppard et al. (1999) studied the relation between air pollution in Seattle and nonelderly (<65) hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM10_{10\text{-}2.5}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects.[14] They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, $PM_{10\text{-}2.5}$, CO, and ozone. They did not observe an association for SO_2. They found PM and CO to be jointly associated with asthma admissions. The best fitting co-pollutant models were found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. For the remaining pollutants, the best fitting models included $PM_{2.5}$ and CO. Results for other co-pollutant models were not reported.

In response to concerns that the work by Sheppard et al. (1999) may be biased because of concerns about the (S-plus) software used in the original analysis, Sheppard (2003) reanalyzed some of this work, in particular Sheppard reanalyzed the original study's $PM_{2.5}$ single pollutant model.

The coefficient and standard error are based on the relative risk (1.04) and 95% confidence interval (1.01-1.06) for a 11.8 $\mu g/m^3$ increase in $PM_{2.5}$ in the 1-day lag GAM stringent model (Sheppard, 2003, pp. 228-299).

Functional Form: Log-linear
Coefficient: 0.003324
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 Chronic Lung Disease (Ito, 2003)

Lippmann et al. (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10\text{-}2.5}$ 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), $PM_{10\text{-}2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not

[14] $PM_{2.5}$ levels were estimated from light scattering data.

statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R functions are based on results of the single pollutant model and co-pollutant model with ozone.

In response to concerns about the (S-plus) software used in the original analysis, Ito (2003) reanalyzed the study by Lippmann et al. (2000). The reanalysis by Ito reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates.

The coefficient and standard error are based on the relative risk (1.043) and 95% confidence interval (0.902-1.207) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ in the 3-day lag GAM stringent model(Ito, 2003, Table 8).

Functional Form: Log-linear
Coefficient: 0.001169
Standard Error: 0.002064
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 for Chronic Lung Disease (Moolgavkar, 2003; 2000a)

Moolgavkar (2000a) examined the association between air pollution and COPD hospital admissions (ICD 490-496) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO_2, NO_2, CO, and PM_{10} in all three areas. $PM_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group in Chicago and Phoenix, weak associations were observed between the gaseous pollutants and admissions. No consistent associations were observed for PM_{10}. In Los Angeles, marginally significant associations were observed for $PM_{2.5}$, which were generally lower than for the gases. In co-pollutant models with CO, the $PM_{2.5}$ effect was reduced. Similar results were observed in the 0-19 and 20-64 year old age groups.

In response to concerns about the (S-plus) software used in the original analysis, Moolgavkar (2003) reanalyzed his earlier study. In the reanalysis, he reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates.

The $PM_{2.5}$ C-R functions for the 65+ age group are based on the reanalysis in Moolgavkar (2003) of the single-pollutant model. The $PM_{2.5}$ C-R functions for the 20-64 age group are based on the original study's single-pollutant model. Since the true PM effect is most likely best represented by a distributed lag model, then any single lag model should underestimate the total PM effect. As a result, we selected the lag models with the greatest effect estimates for use in the C-R functions.

Ages 18 to 64 (Moolgavkar, 2000a)[15]

The single pollutant coefficient and standard error are calculated from an estimated percent change of 2.2and t-statistic of 3.0 for a 10 μg/m^3 increase in $PM_{2.5}$ in the two-day lag model (Moolgavkar, 2000a, Table 4).[16]

Functional Form: Log-linear
Coefficient: 0.0022
Standard Error: 0.000733
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 18-64 (ICD codes 490-492, 494-496)[17]
Population: population of ages 18 to 64.

Ages 65 and older (Moolgavkar, 2003)

The coefficient and standard error are calculated from an estimated percentage change of 1.85 and t-statistic of 3.53 for a 10 μg/m^3 increase in $PM_{2.5}$ in the 2-day lag GAM-30df stringent (10^{-8}) model (Moolgavkar, 2003, Table 17).[18]

Functional Form: Log-linear
Coefficient: 0.001833
Standard Error: 0.000519
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 for Pneumonia (Ito, 2003)

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,

[15] Although Moolgavkar (2000a) reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64.

[16] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 2.2 would result in a relative risk of 1.022 and coefficient of 0.002176. The "estimated" percent change, as reported by Moolgavkar, of 2.2 results in a relative risk of 1.022244 and coefficient of 0.0022.

[17] Moolgavkar (2000a) reports results for ICD codes 490-496. In order to avoid double counting non-elderly asthma hospitalizations (ICD code 493) in a total benefits estimation, we have excluded ICD code 493 from the baseline incidence rate used in this function.

[18] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 2.0 would result in a relative risk of 1.020 and coefficient of 0.001980. An "estimated" percent change of 2.0 results in a relative risk of 1.020201 and coefficient of 0.002.

1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable.

In response to concerns about the (S-plus) software used in the original analysis, Ito (2003) reanalyzed the study by Lippmann et al.(2000). The reanalysis by Ito reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates. The $PM_{2.5}$ C-R function is based on the results of the single pollutant model.

The estimated $PM_{2.5}$ coefficient and standard error are based on a relative risk of 1.154 (95% CI -1.027, 1.298) due to a $PM_{2.5}$ change of 36 $\mu g/m^3$ in the 1-day lag GAM stringent model (Ito, 2003, Table 7).

Functional Form: Log-linear
Coefficient: 0.003979
Standard Error: 0.001659
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 All Cardiovascular (Moolgavkar, 2000b; 2003)

Moolgavkar (2000b) examined the association between air pollution and cardiovascular hospital admissions (ICD 390-448) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO_2, NO_2, CO, and PM_{10} in all three areas. $PM_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group, the gaseous pollutants generally exhibited stronger effects than PM_{10} or $PM_{2.5}$. The strongest overall effects were observed for SO_2 and CO. In a single pollutant model, $PM_{2.5}$ was statistically significant for lag 0 and lag 1. In co-pollutant models with CO, the $PM_{2.5}$ effect dropped out and CO remained significant. For ages 20-64, SO_2 and CO exhibited the strongest effect and any $PM_{2.5}$ effect dropped out in co-pollutant models with CO.

In response to concerns about the (S-plus) software used in the original analysis, Moolgavkar (2003) reanalyzed his earlier study. In the reanalysis, he reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates. Not all of the original results were replicated, so we present here a mix of C-R functions from the reanalysis and from the original study (when the reanalyzed results were not available). The $PM_{2.5}$ C-R functions are based on single pollutant and co-pollutant ($PM_{2.5}$ and CO) models.

We use the single-pollutant results for ages 65 and older from Moolgavkar(2003). Since he did not reanalyze the results for ages 20-64, we use the single-pollutant results from his earlier study.

Note that Moolgavkar (2000b) reported 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.

Ages 18 to 64[19] (Moolgavkar, 2000a)

The single pollutant coefficient and standard error are calculated from an estimated percent change of 1.4 and t-statistic of 4.1 for a 10 μg/m^3 increase in $PM_{2.5}$ in the zero lag model (Moolgavkar, 2000b, Table 4).[20]

Functional Form: Log-linear
Coefficient: 0.0014
Standard Error: 0.000341
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person ages 18 to 64 (ICD codes 390-409, 411-429)
Population: population of ages 18 to 64.

Ages 65 and older (Moolgavkar, 2003)

The single pollutant coefficient and standard error are calculated from an estimated percent change of 1.58 and t-statistic of 4.59 for a 10 μg/m^3 increase in $PM_{2.5}$ in the 0-day lag GAM-30df stringent (10^{-8}) model (Moolgavkar, 2003, Table 12).[21]

Functional Form: Log-linear
Coefficient: 0.001568
Standard Error: 0.000342
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person 65+ (ICD codes 390-409, 411-429)
Population: population of ages 65 and older.

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[19] Although Moolgavkar (2000a) reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64.

[20] 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 1.4 would result in a relative risk of 1.014 and coefficient of 0.00139. Assuming that the 1.4 is the "estimated" percent change described previously would result in a relative risk of 1.014098 and coefficient of 0.0014. We assume that the "estimated" percent changes reported in this study reflect the definition from (Moolgavkar, 2000b).

[21] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 2.2 would result in a relative risk of 1.022 and coefficient of 0.002176. An "estimated" percent change of 2.2 results in a relative risk of 1.022244 and coefficient of 0.0022.

Hospital Admissions for Dysrhythmia, Ischemic Heart Disease, and Congestive Heart Failure (Ito, 2003)

Lippmann et al. (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable.

In response to concerns about the (S-plus) software used in the original analysis, Ito (2003) reanalyzed the study by Lippmann et al.(2000). The reanalysis by Ito reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates. We use the single-pollutant model results from this reanalysis.

Dysrhythmia

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.046 (95% CI 0.906-1.207) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ in the 1-day lag GAM stringent model (Ito, 2003, Table 10).

Functional Form: Log-linear
Coefficient: 0.001249
Standard Error: 0.002033
Incidence Rate: region-specific daily hospital admission rate for dysrhythmia admissions per person 65+ (ICD code 427)
Population: population of ages 65 and older.

Congestive Heart Failure

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.117 (95% CI 1.020-1.224) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ in the 1-day lag GAM stringent model (Ito, 2003, Table 11).

Functional Form: Log-linear
Coefficient: 0.003074
Standard Error: 0.001292
Incidence Rate: region-specific daily hospital admission rate for congestive heart failure admissions per person 65+ (ICD code 428)
Population: population of ages 65 and older.

Ischemic Heart Disease

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.053 (95% CI 0.971-1.143) for a 36 μg/m^3 increase in $PM_{2.5}$ in the 1-day lag GAM stringent model (Ito, 2003, Table 9).

Functional Form: Log-linear
Coefficient: 0.001435
Standard Error: 0.001156
Incidence Rate: region-specific daily hospital admission rate for ischemic heart disease admissions per person 65+ (ICD codes 411-414)[9]
Population: population of ages 65 and old.

[9] 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.

Table 18. Health Impact Functions for Particulate Matter and Emergency Room Visits

Endpoint Name	Author	Year	Location	Age	Other Pollutants in Model	Metric	Beta	Std Error	Functional Form
Emergency Room Visits, Asthma	Norris et al.	1999	Seattle, WA	0-17	NO2, SO2	D24HourMean	0.016527	0.004139	Log-linear

C.4 Emergency Room Visits

Emergency Room Visits for Asthma (Norris, et al., 1999)

Norris et al. (1999) examined the relation between air pollution in Seattle and childhood (<18) hospital admissions for asthma from 1995 to 1996. The authors used air quality data for PM_{10}, light scattering (used to estimate fine PM), CO, SO_2, NO_2, and O_3 in a Poisson regression model with adjustments for day of the week, time trends, temperature, and dew point. They found significant associations between asthma ER visits and light scattering (converted to $PM_{2.5}$), PM_{10}, and CO. No association was found between O_3, NO_2, or SO_2 and asthma ER visits, although O_3 had a significant amount of missing data. In multipollutant models with either PM metric (light scattering or PM_{10}) and NO_2 and SO_2, the PM coefficients remained significant while the gaseous pollutants were not associated with increased asthma ER visits.

In a model with NO_2 and SO_2, the $PM_{2.5}$ coefficient and standard error are calculated from a relative risk of 1.17 (95% CI 1.08-1.26) for a 9.5 $\mu g/m^3$ increase in $PM_{2.5}$ (Norris, et al., 1999, p. 491).

Functional Form: Log-linear
Coefficient: 0.016527
Standard Error: 0.004139
Incidence Rate: region-specific daily emergency room rate for asthma admissions per person <18 (ICD code 493)
Population: population of ages under 18.

Table 19. Health Impact Functions for Particulate Matter and Acute Effects

Endpoint Name	Author	Year	Location	Age	Other Pollutants in Model	Metric	Beta	Std Error	Functional Form
Minor Restricted Activity Days	Ostro & Rothschild	1989	Nationwide	18-64	Ozone	24-hr avg	0.007410	0.000700	Log-linear
Acute Bronchitis	Dockery et al.	1996	24 communities	8-12		Annual	0.027212	0.017096	Logistic
Work Loss Days	Ostro	1987	Nationwide	18-64		24-hr avg	0.004600	0.000360	Log-linear
Lower Respiratory Symptoms	Schwartz and Neas	2000	6 U.S. cities	7-14		24-hr avg	0.019012	0.006005	Logistic

C.5 Minor Effects

We include functions to estimate acute bronchitis, lower respiratory symptoms, minor restricted days, and work loss days.

Acute Bronchitis (Dockery, et al., 1996)

Dockery et al (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.1}$ and PM_{10} were marginally significantly related to bronchitis.[1] They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes. Earlier work, by Dockery et al.(1989), based on six U.S. cities, found acute bronchitis and chronic cough significantly related to PM_{15}. Because it is based on a larger sample, the Dockery et al (1996) study is the better study to develop a C-R function linking $PM_{2.5}$ with bronchitis.

Bronchitis was counted in the study only if there were "reports of symptoms in the past 12 months" (Dockery, et al., 1996, p. 501). It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. Dockery et al. found no relationship between PM and chronic cough and chronic phlegm, which are important indicators of chronic bronchitis. For this analysis, we assumed that the health impact function based on Dockery et al. is measuring acute bronchitis. The health impact function is based on results of the single pollutant model reported in Table 1.

The estimated logistic coefficient and standard error are based on the odds ratio (1.50) and 95% confidence interval (0.91-2.47) associated with being in the most polluted city ($PM_{2.1}$ = 20.7 $\mu g/m^3$) versus the least polluted city ($PM_{2.1}$ = 5.8 $\mu g/m^3$)(Dockery, et al., 1996, Tables 1 and 4). The original study used $PM_{2.1}$, however, we use the $PM_{2.1}$ coefficient and apply it to $PM_{2.5}$ data.

Functional Form: Logistic
Coefficient: 0.027212
Standard Error: 0.017096
Incidence Rate: annual bronchitis incidence rate per person = 0.043 (American Lung Association, 2002c, Table 11)
Population: population of ages 8-12.

[1] 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.

Lower Respiratory Symptoms (Schwartz and Neas, 2000)

Schwartz and Neas (2000) used logistic regression to link lower respiratory symptoms and cough in children with coarse PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

The coefficient and standard error are calculated from the reported odds ratio (1.33) and 95% confidence interval (1.11-1.58) associated with a 15 $\mu g/m^3$ change in $PM_{2.5}$ (Schwartz and Neas, 2000, Table 2).

Functional Form: Logistic
Coefficient: 0.01901
Standard Error: 0.006005
Incidence Rate: daily lower respiratory symptom incidence rate per person = 0.0012(Schwartz, et al., 1994, Table 2).
Population: population of ages 7 to 14.

Minor Restricted Activity Days (Ostro, 1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.[2] The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone has highly variable association with RRADs and MRADs. Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.[3] The health impact function for PM is based on this co-pollutant model.

The study is based on a "convenience" sample of non-elderly individuals. Applying the health impact function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65.

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight:

[2] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the health impact function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

[3] The study used a two-week average pollution concentration; the health impact function uses a daily average, which is assumed to be a reasonable approximation.

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

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent: This reduces down to:

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

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

Functional Form: Log-linear
Coefficient: 0.00741
Standard Error: 0.00070
Incidence Rate: daily incidence rate for minor restricted activity days (MRAD) = 0.02137 (Ostro and Rothschild, 1989, p. 243)
Population: adult population ages 18 to 64.

Work Loss Days (Ostro, 1987)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.[4] The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels[5] were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function presented here is a weighted average of the coefficients in Ostro (1987, Table 3) using the inverse of the variance as the weight.

[4] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the health impact function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

[5] The study used a two-week average pollution concentration; the health impact function uses a daily average, which is assumed to be a reasonable approximation.

The study is based on a "convenience" sample of non-elderly individuals. Applying the health impact function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65. On the other hand, the number of workers over the age of 65 is relatively small; it was approximately 3% of the total workforce in 2001(U.S. Bureau of the Census, 2002).

The coefficient used in the health impact function is a weighted average of the coefficients in Ostro (1987, Table 3) using the inverse of the variance as the weight:

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

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

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

This eventually reduces down to:

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

Functional Form: Log-linear
Coefficient: 0.0046
Standard Error: 0.00036
Incidence Rate: daily work-loss-day incidence rate per person ages 18 to 64 = 0.00595 (Adams, et al., 1999, Table 41;U.S. Bureau of the Census, 1997, No. 22)
Population: adult population ages 18 to 64.

Table 20. Health Impact Functions for Particulate Matter and Asthma-Related Effects

Endpoint Name	Author	Year	Location	Age	Averaging Time[1]	Beta	Std Error	Functional Form
Asthma Exacerbation, Cough	Ostro et al.	2001	Los Angeles, CA	6-18	24-hr avg	0.000985	0.000747	Logistic
Asthma Exacerbation, Shortness of Breath	Ostro et al.	2001	Los Angeles, CA	6-18	24-hr avg	0.002565	0.001335	Logistic
Asthma Exacerbation, Wheeze	Ostro et al.	2001	Los Angeles, CA	6-18	24-hr avg	0.001942	0.000803	Logistic
Asthma Exacerbation, Cough	Vedal et al.	1998	Vancouver, CAN	6-18	24-hr avg	0.007696	0.003786	Logistic
Upper Respiratory Symptoms	Pope et al.	1991	Utah Valley	9-11	24-hr avg	0.0036	0.0015	Logistic

C.6 Asthma-Related Effects

We pool the results of studies by Ostro et al. (2001) and Vedal et al. (1998) to get an estimate of lower respiratory symptoms in asthmatics. We pool results using a fixed/random-effects approach, similar to the analysis performed for Clean Air Interstate Rule (U.S. EPA, 2005b, Table 4-7). In addition to the lower respiratory estimate, we include an upper respiratory estimate based on a study by Pope et al. (1991).

Pooling Ostro et al. (2001) and Vedal et al. (1998)

To characterize asthma exacerbations in children, we use two studies that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between PM2.5, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates.

Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM10 and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations.

We employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled (with a fixed/random effects approach) the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al. (2001) study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled with the cough-related estimate generated using the Vedal study (again using a fixed/random effects approach). The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

To prevent double-counting, we followed EPA (2005b, p. 4-38) and focused the estimation on asthma exacerbations occurring in children and excluded adults from the calculation. Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, this would likely have double-counted incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double-counting.

Asthma Exacerbation: Cough, Wheeze, and Shortness of Breath (Ostro, et al., 2001)

Ostro et al. (2001) studied the relation between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects.[1] Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found cough prevalence associated with PM_{10} and $PM_{2.5}$ and cough incidence associated with $PM_{2.5}$, PM_{10}, and NO_2. Ozone was not significantly associated with cough among asthmatics.

Note that the study focused on African-American children ages 8 to 13 years old. We apply the function based on this study to the general population ages 6 to 18 years old.

Asthma Exacerbation, Cough

The coefficient and standard error are based on an odds ratio of 1.03 (95% CI 0.98-1.07) for a 30 μg/m^3 increase in 12-hour average $PM_{2.5}$ concentration(Ostro, et al., 2001, Table 4, p. 204).

Functional Form: Logistic
Coefficient: 0.000985
Standard Error: 0.000747
Incidence Rate: daily cough rate per person (Ostro, et al., 2001, p. 202) = 0.145
Population: asthmatic population ages 6 to 18 = 5.67%.[2]

Asthma Exacerbation, Shortness of Breath

The coefficient and standard error are based on an odds ratio of 1.08 (95% CI 1.00-1.17) for a 30 μg/m^3 increase in 12-hour average $PM_{2.5}$ concentration (Ostro, et al., 2001, Table 4, p. 204).

Functional Form: Logistic
Coefficient: 0.002565
Standard Error: 0.001335
Incidence Rate: daily shortness of breath rate per person (Ostro, et al., 2001, p. 202) = 0.074
Population: asthmatic population ages 6 to 18 = 5.67%.

[1] The authors note that there were 26 days in which $PM_{2.5}$ concentrations were reported higher than PM_{10} concentrations. The majority of results the authors reported were based on the full dataset. These results were used for the basis for the C-R functions.

[2] The American Lung Association (2002a, Table 7) estimates asthma prevalence for children 5-17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Asthma Exacerbation, Wheeze

The coefficient and standard error are based on an odds ratio of 1.06 (95% CI 1.01-1.11) for a 30 μg/m^3 increase in 12-hour average $PM_{2.5}$ concentration (Ostro, et al., 2001, Table 4, p. 204).

Functional Form: Logistic
Coefficient: 0.001942
Standard Error: 0.000803
Incidence Rate: daily wheeze rate per person (Ostro, et al., 2001, p. 202) = 0.173
Population: asthmatic population ages 6 to 18 = 5.67%.

Asthma Exacerbation, Cough (Vedal, et al., 1998)

Vedal et al. (1998) studied the relationship between air pollution and respiratory symptoms among asthmatics and non-asthmatic children (ages 6 to 13) in Port Alberni, British Columbia, Canada. Four groups of elementary school children were sampled from a prior cross-sectional study: (1) all children with current asthma, (2) children without doctor diagnosed asthma who experienced a drop in FEV after exercise, (3) children not in groups 1 or 2 who had evidence of airway obstruction, and (4) a control group of children with matched by classroom.

The authors used logistic regression and generalized estimating equations to examine the association between daily PM_{10} levels and daily increases in various respiratory symptoms among these groups. In the entire sample of children, PM_{10} was significantly associated with cough, phlegm, nose symptoms, and throat soreness. Among children with diagnosed asthma, the authors report a significant association between PM_{10} and cough symptoms, while no consistent effects were observed in the other groups. Since the study population has an over-representation of asthmatics, due to the sampling strategy, the results from the full sample of children are not generalizeable to the entire population. The health impact function presented below is based on results among asthmatics ages 6 to 18.

The PM_{10} coefficient and standard error are based on an increase in odds of 8% (95% CI 0-16%) reported in the abstract for a 10 μg/m^3 increase in daily average PM_{10}.

Functional Form: Logistic
Coefficient: 0.007696
Standard Error: 0.003786
Incidence Rate: daily cough rate per person (Vedal, et al., 1998, Table 1, p. 1038) = 0.086
Population: asthmatic population ages 6 to 18 = 5.67%.[3]

[3] The American Lung Association (American Lung Association, 2002a) estimates asthma prevalence for children 5-17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Upper Respiratory Symptoms (Pope, 1991)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes.

Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma'(Pope, 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, 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, 1991, Table 2)
Population: asthmatic population ages 9 to 11 = 5.67%[4] of population ages 9 to 11.

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

C.7 Calculating Threshold-Adjusted Health Impact Functions[22]

Following the approach taken in OAQPS' June 2005 particulate matter (PM) risk assessment (U.S. EPA, 2005c), we used a 10 ug/m^3 cutpoint for health impact functions. The risk assessment noted that while there are likely biological thresholds in individuals for specific health responses, the available epidemiological studies do not support or refute the existence of thresholds at the population level for either long-term or short-term $PM_{2.5}$ exposures within the range of air quality observed in the studies. It may therefore be appropriate to consider health risks estimated not only with the reported log-linear or logistic C-R functions, but also with modified functions that approximate non-linear, sigmoidal-shaped functions that would better reflect possible population thresholds.

We approximated hypothetical sigmoidal $PM_{2.5}$ C-R functions by "hockeystick" functions based on the reported log-linear or logistic functions. This approximation consisted of (1) imposing a cutpoint (i.e., an assumed threshold) on the original C-R function, that is intended to reflect an inflection point in a typical sigmoidal shaped function, below which there is little or no population response, and (2) adjusting the slope of the original C-R function above the cutpoint.

If the researchers in the original study fit a log-linear, linear, or logistic model through data that actually better support a sigmoidal or "hockeystick" form, the slope of the fitted curve would be smaller than the slope of the upward-sloping portion of the "true" hockeystick relationship, as shown in Figure 2 and Figure 3. The horizontal portion of the data below the cutpoint would essentially cause the estimated slope to be biased downward relative to the "true" slope of the upward-sloping portion of the hockeystick. The slope of the upward-sloping portion of the hockeystick model should therefore be adjusted upward (from the slope of the reported C-R function), as shown in Figure 2. If the data used in a study do not extend down below the cutpoint or extend only slightly below it, then the extent of the downward bias of the reported $PM_{2.5}$ coefficient will be minimal, as illustrated in Figure 3.

[22] The discussion in this section on the approach taken to adjust the PM2.5 coefficient in a threshold model is based on the discussion in Section 2.5.3 of: Abt Associates Inc., 2005. "Particulate Matter Health Risk Assessment for Selected Urban Areas." Prepared by Abt Associates for Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC. June 2005.

Figure 2. Relationship Between Estimated Log-Linear Concentration-Response Function and Hockeystick Model With Threshold C – General Case

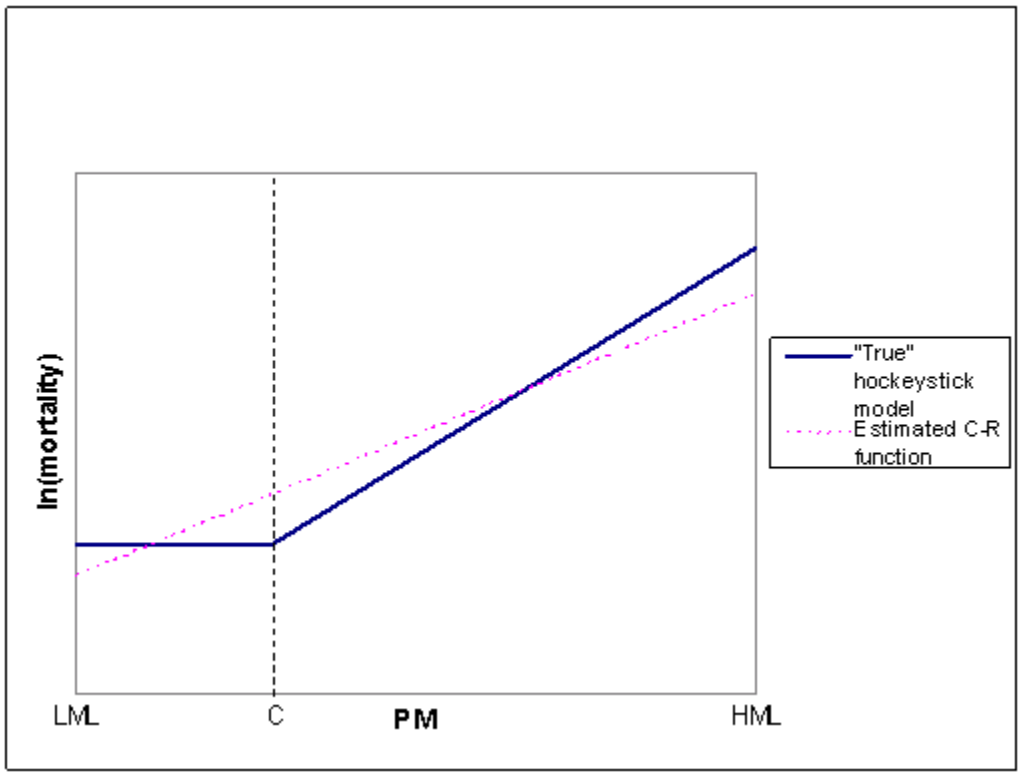
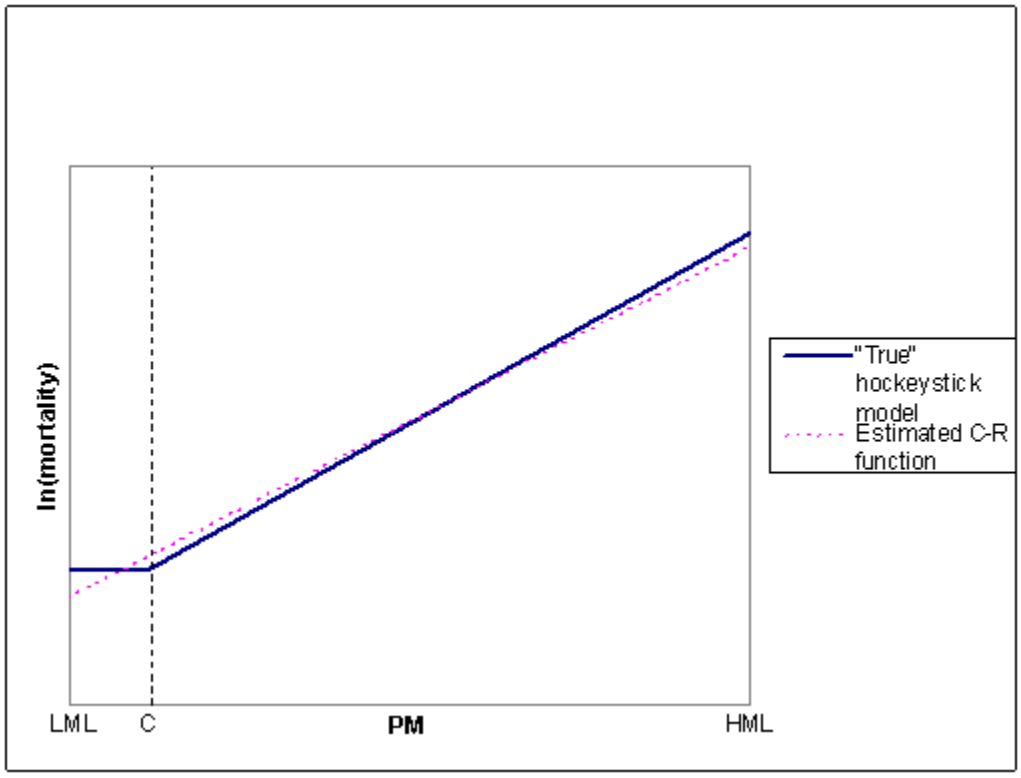


Figure 3. Relationship Between Estimated Log-Linear Concentration-Response Function and Hockeystick Model With Threshold C – Lowest Measured Level (LML) Close to Hypothetical Threshold



We used a simple slope adjustment method based on the idea discussed above – that, if the data in the study were best described by a hockeystick model with a cutpoint at c, then the slope estimated in the study using a log-linear or logistic model would be approximately a weighted average of the two slopes of the hockeystick – namely, zero and the slope of the upward-sloping portion of the hockeystick. If we let

- LML denote the lowest measured PM level in the study,
- c denote the cutpoint (for c > LML),[2]
- HML denote the highest measured PM level in the study,
- β^{est} denote the slope (the PM coefficient) estimated in the study (using a log-linear or logistic model), and
- β^{T} denote the "true" slope of the upward-sloping portion of the hockeystick,

then, assuming the estimated coefficient reported by the study is (approximately) a weighted average of the slope below the cutpoint (0) and the slope above the cutpoint,

[2] If c< LML, no slope adjustment is needed.

$$\beta^{est} = 0 * \frac{(c - LML)}{(HML - LML)} + \beta^{T} * \frac{(HML - c)}{(HML - LML)}.$$

Solving for β^{T},

$$\beta^{T} = \beta^{est} * \frac{(HML - LML)}{(HML - c)}.$$

That is, the "true" slope of the upward-sloping portion of the hockeystick would be the slope estimated in the study (using a log-linear or logistic model rather than a hockeystick model) adjusted by the inverse of the proportion of the range of PM levels observed in the study that was above the cutpoint. Note that if the LML was below the estimated PRB (or if it was not available for the study), the estimated PRB was substituted for LML in the above equation.

Table 21 presents the threshold adjustments that were used to multiply with both the mean coefficient estimate and its standard error.

Table 21. Threshold Adjustment Factors Based on Assumed Threshold of 10 ug/m^3

Endpoint	Author	Min	Max	Adj	Note
Mortality, All Cause	Pope et al. (2002)	7.5	30	1.125	
Mortality, All Cause	Laden et al (2006)	10.8	25.5	1.000	
Mortality, All Cause	Woodruff et al. (1997)	11.9	68.8	1.000	
Chronic Bronchitis	Abbey et al. (1995c)	--	--	1.000	Min and max not reported.
AMI, Nonfatal	Peters et al. (2001)	4.6	24.3	1.378	Min and max based on 5% and 95%.
HA, various types	Moolgavkar (2000b)	4	86	1.079	
HA, various types	Moolgavkar (2003)	4	86	1.079	
HA, various types	Ito (2003)	6	42	1.125	Min and max based on 5% and 95%.
HA, Asthma	Sheppard (2003)	6	32	1.182	Min and max based on 5% and 95%.
ER Visits, Asthma	Norris et al. (1999)	9	18.2	1.122	
MRAD	Ostro and Rothschild (1989)	--	--	1.000	Study gave mean and std dev. We estimate min is above threshold of 10.
Acute Bronchitis	Dockery et al. (1996)	5.8	20.7	1.393	
Work Loss Days	Ostro (1987)	--	--	1.000	Study did not provide a mean, SD, or pollutant range.
LRS	Schwartz and Neas (2000)	7.2	86	1.037	
Asthma Exacerbation,	Ostro et al. (2001)	4.5	208.7	1.028	
URS	Pope et al. (1991)	11	195	1.000	
Asthma Exacerbation	Vedal et al. (1998)	3	159	1.047	Min = policy-relevant background. Actual min was 0.2 in North and 0.5 in South.

Appendix D: Baseline Incidence Rates for Adverse Health Effects

Health impact functions developed from log-linear or logistic models estimate the percent change in an adverse health effect associated with a given pollutant change. In order to estimate the absolute change in incidence using these functions, we need the baseline incidence rate of the adverse health effect. This appendix describes the data used to estimate baseline incidence rates for the health effects considered in this analysis.

Note that the level of geographic aggregation varies with the type of health effect, due to data limitations. The mortality data are available at the county-level, and would seem appropriate for PIE's county-level results. For hospital admissions, in which we have data for four broad regions, the level of aggregation is greater than the county-level, and as a result, the health impacts estimates for any given county are more uncertain. Similarly, for chronic bronchitis, lower respiratory symptoms, and minor restricted activity days – health effects with national incidence rates – we introduce additional uncertainty to the estimates. In some instances we will likely over estimate, and in others under estimate, however, on the whole, we hope to have a reasonably unbiased estimate.

D.1 Mortality

Age, cause, and county-specific mortality rates were originally obtained from the U.S. Centers for Disease Control (CDC) for the years 1996 through 1998. However, since mortality rates are projected to change significantly over time due to the general increase in life-expectancy, we calibrated our county-specific rates with U.S. Census forecasts of national, all-cause mortality rates for 2010, 2015, and 2020. Table 22 presents population-weighted national mortality rates by year and age group.

Table 22. National All-Cause Mortality Rates for Selected Conditions, by Year and Age Group

Year	Mortality Rate by Age Group (deaths per 100 people per year)							
	Infants	25-34	35-44	45-54	55-64	65-74	75-84	85+
1996-1998	0.246	0.119	0.211	0.437	1.056	2.518	5.765	15.160
2010	0.217	0.107	0.181	0.377	0.908	2.094	5.087	13.850
2015	0.204	0.103	0.170	0.357	0.858	1.966	4.742	13.473
2020	0.192	0.099	0.158	0.335	0.819	1.890	4.418	13.067

Source: We obtained county-level 1996-1998 mortality rates from the CDC Wonder (http://wonder.cdc.gov/). Year 2010 and 2015 forecasted rates were estimated based on the U.S. Census Burean projected life tables (http://www.census.gov/population/www/projections/natdet-D5.html) and population forecasts (http://www.census.gov/ipc/www/usinterimproj/). Note that county-specific mortality rates are used in PIE's health impact functions. Also note that the rates presented here are population-weighted by the population for the year specific to the rate estimate.

In developing our county mortality incidence projections, we multiplied the county-specific all-cause mortality rates for 1996-1998 with the ratio of the future year (e.g., 2010) national all-cause rate to the 1996-1998 national all-cause rate.

$$Mort\ Rate_{County\ i,\ 2010} = Mort\ Rate_{County\ i,\ 1996-1998} \cdot \left(\frac{Mort\ Rate_{U.S.,\ 2010}}{Mort\ Rate_{U.S.,\ 1996-1998}} \right)$$

CDC maintains an online data repository of health statistics, CDC Wonder, accessible at http://wonder.cdc.gov/. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau post-censal population estimates. We averaged mortality rates across three years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group.

D.2 Hospitalizations

Regional hospitalization counts were obtained from the National Center for Health Statistics' (NCHS) National Hospital Discharge Survey (NHDS). NHDS is a sample-based survey of non-Federal, short-stay hospitals (<30 days)[1], and is the principal source of nationwide hospitalization data. The survey collects data on patient characteristics, diagnoses, and medical procedures.

Public use data files for the year 1999 survey were downloaded[2] and processed to estimate hospitalization counts by region. NCHS groups states into four regions using the following groupings defined by the U.S. Bureau of the Census:

· Northeast - Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Pennsylvania

· Midwest - Ohio, Indiana, Illinois, Michigan, Wisconsin, Minnesota, Iowa, Missouri, North Dakota, South Dakota, Nebraska, Kansas

· South - Delaware, Maryland, District of Columbia, Virginia, West Virginia, North Carolina, South Carolina, Georgia, Florida, Kentucky, Tennessee, Alabama, Mississippi, Arkansas, Louisiana, Oklahoma, Texas

· West - Montana, Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada, Washington, Oregon, California, Alaska, Hawaii

[1]The following hospital types are excluded from the survey: hospitals with an average patient length of stay of greater than 30 days, federal, military, Department of Veterans Affairs hospitals, institutional hospitals (e.g. prisons), and hospitals with fewer than six beds.

[2] Data are available at ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/

We calculated per capita hospitalization rates, by dividing these counts by the estimated regional population estimates for 1999 that we derived from the U.S. Bureau of the Census and the population projections used by NHDS to generate the counts. Note that NHDS started with hospital admission counts, based on a sample of admissions, and then they used population estimates to generate population-weighted hospital admission counts that are representative of each region. This weighting used forecasts of 1999 population data. Ideally, we would use these same forecasts to generate our admission rates. However, while NHDS presented counts of hospital admissions with a high degree of age specificity, it presented regional population data for only four age groups: 0-14, 15-44, 45-64, and 65+. Using only the NHDS data, we would be limited to calculating regional admission rates for four groups. Because we are interested in a broader range of age groups, we turned to 2000 Census.

We used the 2000 Census to obtain more age specificity, and then corrected the 2000 Census figures so that the total population equaled the total for 1999 forecasted by NHDS. That is, we sued the following procedure: (1) we calculated the count of hospital admissions by region in 1999 for the age groups of interest, (2) we calculated the 2000 regional populations corresponding to these age groups, (3) calculated regional correction factors, that equal the regional total population in 1999 divided by the regional total population in 2000 by region, (4) multiplied the 2000 population estimates by these correction factors, and (5) divided the 1999 regional count of hospital admissions by the estimated 1999 population.

The endpoints in hospitalization studies are defined using different combinations of ICD codes. Rather than generating a unique baseline incidence rate for each ICD code combination, for the purposes of this analysis, we identified a core group of hospitalization rates from the studies and applied the appropriate combinations of these rates in the C-R functions:

- all respiratory (ICD-9 460-519)
- chronic lung disease (ICD-9 490-496)
- asthma (ICD-9 493)
- pneumonia (ICD-9 480-487)
- acute bronchitis (ICD-9 466)
- acute laryngitis (ICD-9 464)
- all cardiovascular (ICD-9 390-459)
- ischemic heart disease (ICD-9 410-414)
- dysrhythmia (ICD-9 427)
- congestive heart failure (ICD-9 428)

For each C-R function, we selected the baseline rate or combination of rates that most closely matches to the study endpoint definition. For studies that define chronic lung disease as ICD 490-492, 494-496, we subtracted the incidence rate for asthma (ICD 493) from the chronic lung disease rate (ICD 490-496). In some cases, the baseline rate will not match exactly to the endpoint definition in the study. For example, Burnett et al. (2001)studied the following respiratory conditions in infants <2 years of age: ICD 464.4, 466, 480-486, 493. For this C-R function we apply an aggregate of the following rates: ICD 464, 466, 480-487, 493. Although they do not match exactly, we assume that relationship observed between the pollutant and study-defined endpoint is applicable for the additional codes. Table 23 presents a summary of the national hospitalization rates for 1999 from NHDS.

Table 23. Hospitalization Rates, by Region and Age Group

Hospitalization Category	ICD-9 Codes	Hospitalization Rate by Age Group (admissions per 100 people per year)									
		Under 2	2-17	18-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Respiratory											
all respiratory	460-519	5.447	0.545	0.271	0.318	0.446	0.763	1.632	3.506	6.276	9.746
acute laryngitis	464	0.285	0.029	0.002	0.001	0.002	0.008	0.000	0.001	0.009	0.005
acute bronchitis	466	2.428	0.028	0.017	0.014	0.017	0.027	0.040	0.090	0.192	0.364
pneumonia	480-487	1.498	0.168	0.069	0.103	0.155	0.256	0.561	1.344	2.781	5.597
asthma	493	0.730	0.226	0.081	0.109	0.098	0.144	0.161	0.182	0.231	0.258
chronic lung disease	490-496	0.769	0.232	0.089	0.124	0.148	0.301	0.711	1.383	1.907	1.574
Cardiovascular											
all cardiovascular	390-429	0.089	0.023	0.052	0.146	0.534	1.552	3.384	6.611	10.032	13.192
ischemic heart disease	410-414	0.026	0.002	0.008	0.031	0.231	0.902	2.021	3.345	4.193	4.099
dysrhythmia	427	0.015	0.010	0.017	0.027	0.076	0.158	0.392	1.014	1.709	2.203
congestive heart failure	428	0.016	0.001	0.005	0.011	0.055	0.160	0.469	1.226	2.677	4.948

Source: As described in the text, we obtained the regional count of hospital admissions from National Hospital Discharge Survey (NHDS), and we obtained the population data from the 2000 U.S. Census and NHDS.

D.3 Emergency Room Visits for Asthma

Regional asthma emergency room visit counts were obtained from the National Hospital Ambulatory Medical Care Survey (NHAMCS). NHAMCS is a sample-based survey, conducted by NCHS, designed to collect national data on ambulatory care utilization in hospital emergency and outpatient departments of non-Federal, short-stay hospitals (<30 days).[1]

Public use data files for the year 2000 survey were downloaded[2] and processed to estimate hospitalization counts by region. We obtained population estimates from the 2000 U.S. Census. The NCHS regional groupings described above were used to estimate regional emergency room visit rates. Table 24 presents the estimated asthma emergency room rates by region.

[1] The target universe of the NHAMCS is in-person visits made in the United States to emergency and outpatient departments of non-Federal, short-stay hospitals (hospitals with an average stay of less than 30 days) or those whose specialty is general (medical or surgical) or children's general.

[2] Data are available at ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/

Table 24. Emergency Room Visit Rates for Asthma, by Region and Age Group

ER Category	ICD-9 Code	Region	ER Visit Rate (visits per 100 people per year)		
			0-18	18-64	65+
asthma	493	Northeast	0.761	0.802	0.300
		Midwest	1.476	0.877	0.334
		South	1.243	0.420	0.192
		West	0.381	0.381	0.137

Source: We obtained ER visit counts for the year 2000 from the National Hospital Ambulatory Medical Care Survey (NHAMCS) and population data were obtained from the 2000 U.S. Census.

D.4 Nonfatal Heart Attacks

The relationship between short-term particulate matter exposure and heart attacks was quantified in a case-crossover analysis by Peters et al (2001). The study population was selected from heart attack survivors in a medical clinic. Therefore, the applicable population to apply to the C-R function is all individuals surviving a heart attack in a given year. Several data sources are available to estimate the number of heart attacks per year. For example, several cohort studies have reported estimates of heart attack incidence rates in the specific populations under study. However, these rates depend on the specific characteristics of the populations under study and may not be the best data to extrapolate nationally. The American Heart Association reports approximately 540,000 new heart attacks per year using data from a multi-center study (Haase, 2002). Exclusion of heart attack deaths reported by CDC Wonder yields approximately 330,000 nonfatal cases per year.[3]

An alternative approach to the estimation of heart attack rates is to use data from the National Hospital Discharge Survey, assuming that all heart attacks that are not instantly fatal will result in a hospitalization. According to the National Hospital Discharge Survey, in 1999 there were approximately 829,000 hospitalizations due to heart attacks (acute myocardial infarction: ICD-9 410) (Popovic, 2001, Table 8). We used regional hospitalization rates over estimates extrapolated from cohort studies because the former is part of a nationally representative survey with a larger sample size, which is intended to provide reliable national estimates. As additional information is provided regarding the American Heart Association methodology, we will evaluate the usefulness of this estimate of heart attack incidence.

Rosamond et al. (1999) reported that approximately six percent of male and eight percent of female hospitalized heart attack patients die within 28 days (either in or outside of the hospital). We, therefore, applied a factor of 0.93 to the count of hospitalizations to estimate the number of nonfatal heart attacks per year. To estimate the *rate* of nonfatal heart attack, we divided the count by the population estimate for 2000 from the U.S. Census. Table 25 presents the regional nonfatal heart attack incidence rates.

[3] Note that we excluded fatal heart attacks to avoid double-counting mortality, as well as to be consistent with prior EPA regulatory impact assessments (e.g., Clean Air Interstate Rule).

Table 25. Nonfatal Heart Attack Rates, by Region and Age Group

Endpoint (ICD codes)	Region	Nonfatal Heart Attack Rate (cases per 100 people per year) [a]		
		0-18	18-64	65+
Nonfatal heart attacks (ICD-9 410)	Northeast	0.0000	0.2167	1.6359
	Midwest	0.0003	0.1772	1.4898
	South	0.0006	0.1620	1.1797
	West	0.0000	0.1391	1.1971

[a] Rates are based on data from the 1999 National Hospital Discharge Survey (NHDS) and an estimate from Rosamond et al. (1999) that approximately 7% of individuals hospitalized for a heart attack die within 28 days.

D.5 Other Acute and Chronic Effects

For many of the minor effect studies, baseline rates from a single study are often the only source of information, and we assume that these rates hold for locations in the U.S. The use of study-specific estimates are likely to increase the uncertainty around the estimate because they are often estimated from a single location using a relatively small sample. These endpoints include: acute bronchitis, chronic bronchitis, upper respiratory symptoms, lower respiratory symptoms. Table 26 presents a summary of these baseline rates.

Table 26. Selected Acute and Chronic Effects Rates

Endpoint	Age	Parameter [a]	Rate	Source
Acute Bronchitis	8-12	Incidence	4.300	(American Lung Association, 2002c, Table 11)
Chronic Bronchitis	27+	Incidence	0.378	(Abbey, et al., 1993b, Table 3)
	18-44	Prevalence	3.67%	(American Lung Association, 2002b, Table 4)
	45-64		5.05%	
	65+		5.87%	
Lower Respiratory Symptoms (LRS)	7-14	Incidence	43.8	(Schwartz, et al., 1994, Table 2)
Minor Restricted Activity Days (MRAD)	18-64	Incidence	780.0	(Ostro and Rothschild, 1989, p. 243)
Work Loss Day (WLD)	18-64	Incidence	217.2	(Adams, et al., 1999, Table 41); (U.S. Bureau of the Census, 1997)
	18-24		197.1	
	25-44		247.5	
	45-64		179.6	

a The incidence rate is the number of cases per 100 people per year. Prevalence refers to the fraction of people that have a particular illness during a particular time period.

Acute Bronchitis

The annual rate of acute bronchitis for children ages 5 to 17 was obtained from the American Lung Association (2002c). The authors reported an annual incidence rate per person of 0.043, derived from the 1996 National Health Interview Survey.

Chronic Bronchitis

The annual incidence rate for chronic bronchitis is estimated from data reported by Abbey et al.(1993a). The rate is calculated by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (estimated to be 46.6% based on Abbey et al. (1995c, Table 1)). We then multiplied this result by 100 to calculate an annual incidence rate per 100 people of 0.378.

Age-specific incidence rates are not available. Abbey et al. (1995c, Table 1) did report the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis. However, they did not report an overall incidence rate for bronchitis by age-group. Since, the cough and sputum types of bronchitis overlap to an unknown extent, we did not attempt to generate age-specific incidence rates for the over-all rate of bronchitis.

We obtained the annual prevalence rate for chronic bronchitis from the American Lung Association,(American Lung Association, , Table 4). Based on an analysis of 1999 National Health

Interview Survey data, they estimated a rate of 0.0443 for persons 18 and older, they also reported the following prevalence rates for people in the age groups 18-44, 45-64, and 65+: 0.0367, 0.0505, and 0.0587, respectively.

Lower Respiratory Symptoms

Lower respiratory symptoms (LRS) are defined as two or more of the following: cough, chest pain, phlegm, wheeze. The proposed yearly incidence rate for 100 people, 43.8, is based on the percentiles in Schwartz et al (1994, Table 2). The authors did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated per person per day values are 10^{th} = 0 percent, 25^{th} = 0 percent, 50^{th} = 0 percent, 75^{th} = 0.29 percent, and 90^{th} = 0.34 percent. The most conservative estimate consistent with the data are to assume the incidence per person per day is zero up to the 75^{th} percentile, a constant 0.29 percent between the 75^{th} and 90^{th} percentiles, and a constant 0.34 percent between the 90^{th} and 100^{th} percentiles. Alternatively, assuming a linear slope between the 50^{th} and 75^{th}, 75^{th} and 90^{th}, and 90^{th} to 100^{th} percentiles, the estimated mean incidence rate per person per day is 0.12 percent.[23] We used the latter approach in this analysis, and then multiplied by 100 and by 365 to calculate the incidence rate per 100 people per year.

Minor Restricted Activity Days (MRAD)

Ostro and Rothschild (1989, p. 243) provide an estimate of the annual incidence rate of MRADs (7.8). We multiplied this estimate by 100 to get an annual rate per 100 people.

Work Loss Days

The yearly work-loss-day incidence rate per 100 people is based on estimates from the 1996 National Health Interview Survey (Adams, et al., 1999, Table 41). They reported a total annual work loss days of 352 million for individuals ages 18 to 65. The total population of individuals of this age group in 1996 (162 million) was obtained from (U.S. Bureau of the Census, 1997). The average annual rate of work loss days per individual (2.17) was multiplied by 100 to obtain the average yearly work-loss-day rate of 217 per 100 people. Using a similar approach, we calculated work-loss-day rates for ages 18-24, 25-44, and 45-64, respectively.

D.6 Asthma-Related Health Effects

Several studies have examined the impact of air pollution on asthma development or exacerbation in the asthmatic population. Many of the baseline incidence rates used in the C-R functions are based on study-specific estimates. The baseline rates for the various endpoints are described below and summarized in Table 27.

[23] For example, the 62.5^{th} percentile would have an estimated incidence rate per person per day of 0.145 percent.

Table 27. Asthma-Related Health Effects Rates

Endpoint	Age	Parameter [a]	Rate	Source
Cough	6-18	Incidence	3,139.0	(Vedal, et al., 1998, Table 1, p. 1038)
Asthma Exacerbation, Cough	6-18	Incidence	5,292.5	(Ostro, et al., 2001, p. 202)
Asthma Exacerbation, Shortness of Breath	6-18	Incidence	2,701.0	(Ostro, et al., 2001, p. 202)
Asthma Exacerbation, Wheeze	6-18	Incidence	6,314.5	(Ostro, et al., 2001, p. 202)
Asthma	6-18	Prevalence	5.67%	(American Lung Association, 2002a, Table 7)
Upper Respiratory Symptoms (URS)2	9-11	Incidence	12,479.4	(Pope, et al., 1991, Table 2)

[a] The incidence rate is the number of cases per 100 people per year. Prevalence refers to the fraction of people that have a particular illness during a particular time period.

Appendix E: Population Forecasts

To estimate the change in population exposure to air pollution, we use projections based on economic forecasting models developed by Woods & Poole (2001) . The Woods and Poole (WP) database contains county-level projections of population by age, sex, and race out to 2030. Projections in each county are determined simultaneously with every other county in the United States to take into account patterns of economic growth and migration. The sum of growth in county-level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates. The projection years used for this particular analysis are 2010, 2015, and 2020.

According to WP, linking county-level growth projections together and constraining to a national-level total growth avoids potential errors introduced by forecasting each county independently. County projections are developed in a four-stage process. First, national-level variables such as income, employment, and populations are forecasted. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an "export-base" approach, which relies on linking industrial sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy.

The export-based approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector. Third, population is projected for each economic area based on net migration rates derived from employment opportunities and following a cohort component method based on fertility and mortality in each area. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2020 based on historical rates of mortality, fertility, and migration.

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

The unit of analysis in PIE is the county, in the years 2010, 2015, and 2020. To forecast population levels for these years, we started with county-level data from the 2000 U.S. Census (GeoLytics Inc., 2002b), and then scaled these data with the ratio of the county-level forecast for the future year (e.g., 2010) over the 2000 county-level population level. In developing the population scaling ratios, we used the county-level forecasts from WP. For any given county "c," we use the following forecasting procedure:

$$age_{4-9,\,c,\,2010,\,COBRA} = age_{4-9,\,c,\,2000,\,Census} \cdot \frac{age_{4-9,\,c,\,2010,\,Woods\ \&\ Poole}}{age_{4-9,\,c,\,2000,\,Woods\ \&\ Poole}} \quad .$$

Appendix F: Economic Value of Health Effects

This appendix presents the mean estimate of the unit values used in this analysis. Table 28 lists these unit values. Note that because of an assumed rise in income between 2010, 2015, and 2020, some are progressively higher in the latter two years.

Table 28. Unit Values for Economic Valuation of Health Endpoints by Income Year (2006 $)

Health Endpoint	Age Range	Unit Value *2010*	*2015*	*2020*
Mortality	0 - 99	$7,300,000	$7,700,000	$8,000,000
Chronic Bronchitis	27 - 99	$440,000	$470,000	$490,000
Acute Myocardial Infarction, Nonfatal	0 - 24	$85,000	$85,000	$85,000
Acute Myocardial Infarction, Nonfatal	25 - 44	$96,000	$96,000	$96,000
Acute Myocardial Infarction, Nonfatal	45 - 54	$100,000	$100,000	$100,000
Acute Myocardial Infarction, Nonfatal	55 - 64	$180,000	$180,000	$180,000
Acute Myocardial Infarction, Nonfatal	65 - 99	$85,000	$85,000	$85,000
HA, All Cardiovascular (less AMI)	18 - 64	$29,000	$29,000	$29,000
HA, All Cardiovascular (less AMI)	65 - 99	$27,000	$27,000	$27,000
HA, Asthma	0 - 64	$10,000	$10,000	$10,000
HA, Chronic Lung Disease	65 - 99	$17,000	$17,000	$17,000
HA, Chronic Lung Disease (less Asthma)	18 - 64	$16,000	$16,000	$16,000
HA, Congestive Heart Failure	65 - 99	$20,000	$20,000	$20,000
HA, Dysrhythmia	65 - 99	$20,000	$20,000	$20,000
HA, Ischemic Heart Disease (less AMI)	65 - 99	$33,000	$33,000	$33,000
HA, Pneumonia	65 - 99	$23,000	$23,000	$23,000
Asthma ER Visits	0 - 17	$370	$370	$370
Acute Bronchitis	7 - 14	$430	$440	$440
Lower Resp. Symptoms	9 - 11	$19	$19	$19
Upper Resp. Symptoms	18 - 64	$30	$30	$31
MRAD	18 - 64	$61	$62	$63
Work Loss Days	18 - 99	**	**	**
Asthma Exacerbation, Cough	6 - 18	$52	$53	$53
Asthma Exacerbation, Shortness of Breath	6 - 18	$52	$53	$53
Asthma Exacerbation, Wheeze	6 - 18	$52	$53	$53

NOTE: Numbers rounded to two significant digits.
* Mortality value after adjustment for 20-year lag.
** County-specific median daily wage.

F.1 Valuing Premature Mortality

EPA has estimated the monetary benefit of reducing premature mortality risk using the VSL approach, which is a summary measure for the value of small changes in mortality risk experienced by a large number of people. The mean value of avoiding one statistical death is assumed to be $4.8 million in 1990 dollars and 1990 income levels. The rationale for this choice is discussed in EPA's 2010 Final NO_2 NAAQS RIA.[24]

EPA (2005b, p. 4-56) assumed that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the 20 years following exposure. To take this into account in the valuation of reductions in premature mortality, we applied an annual 3 percent discount rate to the value of premature mortality occurring in future years.

There are a number of uncertainties in this estimate. The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's WTP to improve one's own chances of survival plus WTP to improve other individuals' survival rates.

The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ across individuals because survival curves depend on such characteristics as age, health state, and the current age to which the individual is likely to survive.

F.2 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) as well as WTP to avoid the pain and suffering associated with the illness. Both WTP and COI estimates are currently available in BenMAP.

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

[24] *See*: page 4-7 at: http://www.epa.gov/ttnecas1/regdata/RIAs/FinalNO2RIAfulldocument.pdf.

The study by Viscusi et al. (1991)used a sample that is larger and more representative of the general population than the study by Krupnick and Cropper(1992), which selected people who have a relative with the disease. However, the chronic bronchitis described to study subjects in the Viscusi study is severe, whereas a pollution-related case may be less severe.

The relationship between the severity of a case of chronic bronchitis and WTP to avoid it was estimated by Krupnick and Cropper(1992). We used that estimated relationship to derive a relationship between WTP to avoid a severe case of chronic bronchitis, as described in the Viscusis study, and WTP to avoid a less severe case. The estimated relationship (see Table 4 in Krupnick and Cropper) can be written as:

$$\ln(WTP) = \alpha + \beta * sev$$

where α denotes all the other variables in the regression model and their coefficients, β is the coefficient of sev, estimated to be 0.18, and sev denotes the severity level (a number from 1 to 13). Let x (< 13) denote the severity level of a pollution-related case of chronic bronchitis, and 13 denote the highest severity level (as described in Viscusi, et al., 1991). Then

$$\ln(WTP_{13}) = \alpha + \beta * 13$$

and

$$\ln(WTP_x) = \alpha + \beta * x \ .$$

Subtracting one equation from the other,

$$\ln(WTP_{13}) - \ln(WTP_x) = \beta * (13 - x)$$

or

$$\ln\left(\frac{WTP_{13}}{WTP_x}\right) = \beta * (13 - x) \ .$$

Exponentiating and rearranging terms,

$$WTP_x = WTP_{13} * e^{-\beta * (13 - x)} \ .$$

Because this function is non-linear, the expected value of WTP for a pollution-related case of CB cannot be obtained by using the expected values of the three uncertain inputs in the function (doing that will substantially understate mean WTP).

F.3 Valuing Non-Fatal Myocardial Infarction

We are 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(Cropper and Sussman, 1990), originally used in the 812 Retrospective Analysis of the Clean Air Act(U.S. EPA, 1997). 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 Table 29, 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. Table 30 gives the resulting estimates.

Table 29. 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.

Table 30. Estimated Costs Over a 5-Year Period of a Non-Fatal Myocardial Infarction

Age Group	Opportunity Cost (2000 $) [a]	Medical Cost (2000 $) [b]	Total Cost (2000 $)
0 - 24	$0	$65,902	$65,902
25-44	$8,774	$65,902	$74,676
45 - 54	$12,932	$65,902	$78,834
55 - 65	$74,746	$65,902	$140,649
> 65	$0	$65,902	$65,902

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

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

F.4 Valuing Hospital Admissions

Society's WTP to avoid a hospital admission includes medical expenses, lost work productivity, the non-market costs of treating illness (i.e., air, water and solid waste pollution from hospitals and the pharmaceutical industry), as well as WTP of the affected individual, as well as of that of relatives, friends, and associated caregivers, to avoid the pain and suffering.[1]

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.

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

Following the method used in the §812 analysis(U.S. EPA, 1999a), ICD-code-specific COI estimates used in our analysis consist of two components: estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). The opportunity cost of a day spent in the hospital is estimated as the value of the lost daily wage, regardless of whether or not the individual is in the workforce. This was estimated as the county median weekly wage in 2000 divided by 5.[25]

For all hospital admissions included in this analysis, estimates of hospital charges and lengths of hospital stays were based on statistics provided by the Agency for Healthcare Research and Quality's Healthcare Utilization Project(2000). The total COI for an ICD-code-specific hospital stay lasting n days, then, would be estimated as the mean hospital charge plus lost wages. Most respiratory hospital admissions categories considered in epidemiological studies consisted of sets of ICD codes. The unit dollar value for

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

[25] The median daily wage was calculated by dividing the median weekly wage ($576 in 2000$) by 5. The median daily wage was obtained from U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12, Table 621: "Full-Time Wage and Salary Workers – Numbers and Earnings: 1985 to 2000."

the set of ICD codes was estimated as the weighted average of the ICD-code-specific values (mean hospital charges plus opportunity costs, based on length of stay) of each ICD code in the set. The weights were the relative frequencies of the ICD codes among hospital discharges in the United States, as estimated by the National Hospital Discharge Survey(Owings and Lawrence, 1999, Table 1). Table 31 shows the unit values thus derived for valuing respiratory and cardiovascular hospital admissions.

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

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

Table 31. Unit Values for Respiratory and Cardiovascular Hospital Admissions

Hospital Admission Category	ICD-9 Codes	Age Range	Medical Cost (2000 $)	Days	COI [a] (2000 $)
Pneumonia	480-487	65+	$17,030	7.07	$17,844
COPD	490-492, 494-496	65+	$12,993	5.69	$13,648
		20-64	$11,820	4.48	$11,820
Asthma	493	<65	$7,448	2.95	$7,788
All cardiovascular	390-429	65+	$20,607	5.07	$21,191
		20-64	$22,300	4.15	$22,778

[a] The unit value for a group of ICD-9 codes is the weighted average of ICD-9 code-specific values, from AHRQ(2000). The weights are the relative frequencies of hospital discharges for each ICD-9 code in the group (Owings and Lawrence, 1999, Table 1). Note that when estimating the cost of lost wages due to days in the hospital, we have used the national median for this table. The actual calculation in COBRA uses each county's median income.

F.5 Valuing Emergency Room Visits for Asthma

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. We use a simple average of the two estimates, which yields a unit value of about $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.

F.6 Valuing Acute Symptoms and Illness Not Requiring Hospitalization

Several acute symptoms and illnesses have been associated with air pollution, including acute bronchitis in children, upper and lower respiratory symptoms, and exacerbation of asthma (as indicated by one of several symptoms whose occurrence in an asthmatic generally suggests the onset of an asthma episode). In addition, several more general health endpoints which are associated with one or more of these acute symptoms and illnesses, such as minor restricted activity days and work loss days, have also been associated with air pollution.

Valuing Acute Bronchitis in Children

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

In previous benefits analyses, such as in the §812 Prospective analysis(U.S. EPA, 1999a), acute bronchitis was valued at $59.31 (in 2000 $). This is the midpoint between a low estimate and a high estimate. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day. For a more complete description of the derivation of this estimate, see Abt Associates(2000, p. 4-30).

A unit value of $59.31 assumes that an episode of acute bronchitis lasts only one day. However, this is generally not the case. More typically, it can last for 6 or 7 days. We therefore made a simple adjustment, multiplying the original unit value of $59.31 by 6. The unit value thus derived and used was $356 (=$59.31 x 6).

Valuing Upper Respiratory Symptoms (URS) in Children

Willingness to pay to avoid a day of upper respiratory symptoms is based on symptom-specific WTPs to avoid those symptoms identified by Pope et al. (1991)as part of the complex of upper respiratory symptoms. Three contingent valuation studies have estimated WTP to avoid various morbidity symptoms that are either within the complex defined by Pope et al.(1991), or are similar to those symptoms. In each CV study, participants were asked their WTP to avoid a day of each of several symptoms. The WTP estimates corresponding to the morbidity symptoms valued in each study are presented in Table 32.

The three individual symptoms listed in Table 32 that were identified as most closely matching those listed by Pope, et al. (1991)for upper respiratory symptoms are cough, head/sinus congestion, and eye irritation, corresponding to "wet cough," "runny or stuffy nose," and "burning, aching or red eyes," respectively. A day of upper respiratory symptoms could consist of any one of the seven possible "symptom complexes" consisting of at least one of these three symptoms. These seven possible symptom complexes are presented in Table 33. We assumed that each of these seven complexes is equally likely.[1] The point estimate of WTP is just an average of the seven estimates of WTP for the different complexes.

Table 32. Median WTP Estimates and Derived Midrange Estimates (2000 $)

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

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

[b] 10% trimmed mean.

[1] 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.

Table 33. Estimates of WTP to Avoid Upper Respiratory Symptoms (2000 $)

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

Valuing Lower Respiratory Symptoms (LRS) in Children

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

The symptoms for which WTP estimates are available that reasonably match lower respiratory symptoms are: cough (C), chest tightness (CT), coughing up phlegm (CP), and wheeze (W). A day of lower respiratory symptoms could consist of any one of the 11 combinations of at least two of these four symptoms.[1] We assumed that each of the eleven types of lower respiratory symptoms is equally likely,[2] and the mean WTP is the average of the WTPs over all combinations. Table 34 presents resulting estimate.

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

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

[2] 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.

Table 34. Estimates of WTP to Avoid Lower Respiratory Symptoms (2000 $)

Symptom Combinations Identified as LRS by Schwartz et al. (1994, p. 1235)	WTP to Avoid Symptoms
Coughing, Chest Tightness	$17.52
Coughing, Coughing Up Phlegm	$12.84
Coughing, Wheeze	$12.54
Chest Tightness, Coughing Up Phlegm	$11.92
Chest Tightness, Wheeze	$11.62
Coughing Up Phlegm, Wheeze	$6.95
Coughing, Chest Tightness, Coughing Up Phlegm	$21.15
Coughing, Chest Tightness, Wheeze	$20.85
Coughing, Coughing Up Phlegm, Wheeze	$16.17
Chest Tightness, Coughing Up Phlegm, Wheeze	$15.25
Coughing, Chest Tightness, Coughing Up Phlegm, Wheeze	$24.47
	Average: $15.57

Valuing Work Loss Days (WLDs)

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

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

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

Valuing Minor Restricted Activity Days (MRADs)

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 after adjusting for inflation $50.55 (2000 $). 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.

Valuing Asthma Exacerbations

Asthma exacerbations are valued at $42 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut(1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut study.

Appendix G: Health & Economic Impacts of Existing EGUs in 2010

This appendix presents the national and state-level results due to reducing emissions from *existing* power plants in 2010. Note that some EGUs (termed "mixed" EGUs in PIE) will comprise both existing and new units operating in 2010. Table 35 presents the percentage of emissions due to existing units at the "mixed" EGUs. Table 36 presents the national impacts of existing plants in 2010, and Table 37 presents the state-level health impacts of existing plants in 2010.

Table 35. Percent of "Mixed" EGU Emissions due to Existing Units in 2010

		Percent of "Mixed" EGU Emissions due to Existing Units				
Plant Name	**ORIS**	***VOC***	***NO_x***	***SO_2***	***$PM_{2.5}$***	***NH_3***
PUBLIC SERVICE CO COMANCHE PLT	470	27%	58%	69%	17%	100%
COUNCIL BLUFFS	1082	44%	83%	84%	45%	100%
CLECO CORP/RODEMACHER POWER STATION	6190	43%	78%	86%	54%	100%
TXU ELECTRIC CO	6648	59%	72%	70%	82%	100%
CITY PUBLICSERVICE B	7097	35%	56%	68%	38%	100%
TUCSON ELECTRIC POWER CO-SPRINGERVILLE	8223	55%	90%	92%	87%	100%

Table 36. Estimated Health Impacts & Economic Damages of Existing EGUs in 2010 – National Summary

Effect	***Health Effects (Cases)***	***Monetary Cost (million 2006 $)***
Adult Mortality (Pope)	13,000	$97,000
Adult Mortality (Laden)	34,000	$250,000
Infant Mortality	32	$230
Chronic Bronchitis	8,100	$3,600
AMI	20,000	$2,200
Cardio. Hosp. Adm.	6,700	$190
Resp. Hosp. Adm.	3,200	$44
Asthma ER Visits	12,000	$4.5
Asthma Exacerbation	220,000	$11
Acute Bronchitis	19,000	$8
URS	170,000	$5.2
LRS	230,000	$4.3
WLD	1,600,000	$150
MRAD	9,800,000	$600
Mortality (Pope) + Morbidity	--	$100,000
Mortality (Laden) + Morbidity	--	$260,000

Note: Results rounded to two digits.

Table 37. Estimated Health Impacts of Existing EGUs in 2010 -- State-Level Results (Cases)

State	Mort. (Pope)	Mort. (Laden)	Infant Mort.	Chron. Bron.	AMI	HA, Cardio	HA, Resp.	Asthma ER	Acute Bron.	URS	LRS	Asthma Exac.	WLD	MRAD
Alabama	300	770	0.9	170	380	140	62	280	400	3,700	4,800	4,700	35,000	210,000
Arizona	8	20	0.0	5	11	3	1	3	14	130	170	160	1,100	6,400
Arkansas	140	360	0.5	76	170	65	28	130	190	1,700	2,200	2,100	15,000	90,000
California	44	110	0.1	33	66	18	9	18	85	760	1,000	980	7,100	42,000
Colorado	54	140	0.2	47	86	23	11	25	120	1,100	1,400	1,300	10,000	59,000
Connecticut	200	510	0.3	120	370	110	55	110	270	2,500	3,200	3,200	24,000	140,000
DC	47	120	0.2	28	53	20	9	38	54	490	640	640	5,900	34,000
Delaware	75	190	0.2	44	97	37	16	69	99	900	1,200	1,200	9,000	54,000
Florida	320	830	0.7	180	450	170	68	260	370	3,400	4,400	4,300	34,000	200,000
Georgia	550	1,400	2.1	390	730	270	130	710	1,000	9,200	12,000	12,000	84,000	490,000
Idaho	6	15	0.0	4	8	2	1	2	11	95	130	120	800	4,700
Illinois	640	1,600	1.8	410	1,000	310	160	810	1,000	9,100	12,000	12,000	85,000	500,000
Indiana	560	1,400	1.6	340	870	260	130	710	870	8,000	10,000	10,000	71,000	420,000
Iowa	160	400	0.3	93	260	79	37	170	200	1,900	2,400	2,400	18,000	110,000
Kansas	94	240	0.3	59	150	46	23	120	150	1,300	1,700	1,700	12,000	72,000
Kentucky	440	1,100	1.0	260	560	210	93	410	610	5,600	7,200	7,100	54,000	320,000
Louisiana	92	240	0.3	55	120	43	20	97	140	1,300	1,700	1,600	11,000	68,000
Maine	47	120	0.0	27	87	26	12	21	51	460	610	600	5,100	31,000
Maryland	400	1,000	1.0	260	560	210	92	430	630	5,800	7,500	7,400	55,000	330,000
Massachusetts	260	660	0.3	160	480	150	72	140	350	3,100	4,100	4,000	32,000	190,000
Michigan	700	1,800	1.8	430	1,100	330	170	840	1,000	9,500	12,000	12,000	89,000	530,000
Minnesota	110	290	0.3	83	210	62	31	160	200	1,800	2,300	2,200	17,000	100,000
Mississippi	140	360	0.6	81	170	65	29	140	210	1,900	2,500	2,400	17,000	100,000
Missouri	310	780	0.8	180	470	140	70	340	420	3,800	5,000	4,900	37,000	220,000
Montana	7	18	0.0	4	10	3	1	2	8	75	100	98	820	4,900
Nebraska	49	120	0.1	31	81	24	12	63	78	700	920	880	6,300	38,000
Nevada	3	8	0.0	2	4	1	1	1	5	41	55	52	400	2,400
New Hampshire	50	130	0.1	32	96	29	14	28	67	610	800	800	6,400	38,000
New Jersey	560	1,400	1.0	340	1,000	310	160	340	820	7,500	9,800	9,500	69,000	410,000
New Mexico	10	26	0.0	7	15	4	2	3	17	150	200	190	1,400	8,400
New York	980	2,500	1.8	610	1,800	550	280	560	1,400	12,000	16,000	16,000	120,000	740,000
North Carolina	700	1,800	2.2	430	920	350	150	740	1,000	9,500	13,000	12,000	89,000	530,000
North Dakota	9	23	0.0	5	15	4	2	8	10	91	120	120	990	5,900

State	Mort. (Pope)	Mort. (Laden)	Infant Mort.	Chron. Bron.	AMI	HA, Cardio	HA, Resp.	Asthma ER	Acute Bron.	URS	LRS	Asthma Exac.	WLD	MRAD
Ohio	1,300	3,200	2.7	730	1,900	580	290	1,400	1,700	16,000	20,000	20,000	150,000	880,000
Oklahoma	120	310	0.4	71	160	59	26	120	170	1,600	2,100	2,000	14,000	86,000
Oregon	7	17	0.0	4	9	3	1	2	10	89	120	110	870	5,200
Pennsylvania	1,400	3,600	2.2	720	2,400	730	340	620	1,500	14,000	18,000	18,000	140,000	840,000
Rhode Island	56	140	0.1	33	100	31	15	29	68	620	820	830	6,700	40,000
South Carolina	290	740	0.8	170	370	140	60	270	400	3,600	4,700	4,600	34,000	200,000
South Dakota	18	47	0.1	11	31	9	5	21	27	240	320	300	2,200	13,000
Tennessee	510	1,300	1.3	300	640	240	110	450	660	6,000	7,900	7,700	60,000	360,000
Texas	330	840	1.2	240	470	170	82	440	640	5,800	7,700	7,300	51,000	300,000
Utah	21	54	0.1	20	33	9	5	15	72	640	850	790	4,700	27,000
Vermont	42	110	0.1	25	80	24	11	19	45	410	540	550	4,900	29,000
Virginia	690	1,700	1.9	440	930	350	160	710	1,000	9,300	12,000	12,000	92,000	550,000
Washington	18	47	0.0	13	26	7	3	6	29	260	340	330	2,600	16,000
West Virginia	230	590	0.5	120	280	110	44	160	240	2,200	2,800	2,800	23,000	140,000
Wisconsin	280	710	0.6	180	470	140	68	310	390	3,500	4,600	4,600	36,000	210,000
Wyoming	3	8	0.0	2	4	1	0	1	4	35	47	45	370	2,200
Total	13,000	34,000	32	8,100	20,000	6,700	3,200	12,000	19,000	170,000	230,000	220,000	1,600,000	9,800,000

Note: Results rounded to two digits.

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