Final Heavy Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results

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1 Introduction

As part of EPA's comprehensive national control program to regulate the heavy-duty vehicle and its fuel as a single system program, new emission standards to be applied to heavy-duty highway engines and vehicles will begin to take effect in 2007. These standards are based on the use of high-efficiency catalytic exhaust emission control devices or comparably effective advanced technologies. Because these devices are damaged by sulfur, EPA is also significantly reducing the level of sulfur in highway diesel fuel by mid-2006. This program will result in emission levels of particulate matter (PM) and oxides of nitrogen that are 90% and 95%, respectively, below current standards levels. In order to meet these more stringent standards for diesel engines, the program calls for a 97% reduction in the sulfur content of diesel fuel. This analysis presents estimates of the potential benefits from the Heavy Duty (HD) Engine/Diesel Fuel rule occurring in 2030.

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

This document has three appendices. Appendix A presents the physical and monetary benefits associated with sensitivity and alternative calculations for the HD Engine/Diesel Fuel Rule 2030 control scenario not considered in the primary analysis. Appendix B presents the ozone C-R functions used in this analysis, and Appendix C presents the PM C-R functions.

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

This chapter describes the methods used to forecast changes in ozone and PM. We use two types of air quality models to make these forecasts. The following sub-sections summarize how we use air quality model results in conjunction with the Criteria Air Pollutant Modeling System (CAPMS) to estimate ozone and PM exposure.

CAPMS is a population-based system for modeling exposures of populations to ambient levels of criteria air pollutants that we use to estimate health benefits.[1] CAPMS divides the United States into eight kilometer by eight kilometer grid cells, and estimates the changes in incidence of adverse health and welfare effects associated with given changes in air quality in each grid cell. We then calculate the national incidence change as the sum of grid-cell-specific changes.

2.1 Ozone Air Quality

To develop baseline and control forecasts for ozone, we use the results of the variable-grid Urban Airshed Model (UAM-V) and observed ozone season data for 1995. The modeling data are used to generate "adjustment factors" that quantify the relationship between modeled levels of ozone in the Eastern U.S. for the base year, 1995, and the future year, 2030. We combine the adjustment factors with actual monitoring data to generate estimates of the future-year levels of ozone. Note that we do not use the modeling data directly to estimate future-year ozone levels. Instead, we use them in a relative sense to simply adjust actual monitor levels. We use the modeling results in a relative sense because it provides a better estimate than the UAM-V modeling data alone. In particular, UAM-V has difficulty modeling night-time hours.

The modeling domain is bounded by longitude -99° to -67° and latitude 26° to 47°.[2] This corresponds to the area that is east of a line running from central South Dakota through central Texas; small portions of the Eastern U.S. are not covered by the UAM-V modeling, such as in northern Maine. In areas outside the modeling domain, we assume that ozone levels in the control scenario are identical with those in the baseline scenario. The three simulation periods for the eastern U.S. are based on meteorology for June 15-24, July 8-15, and August 10-21, 1995, and are based on an emission inventory for 1996.

We collected ozone monitoring data for the ozone season, defined for this analysis as May through September.[3] An ozone monitor record was considered complete if data were available for 50 percent of days in a given season. Each of these days in turn had to have at least nine hourly observations between 8:00am and 7:59pm.

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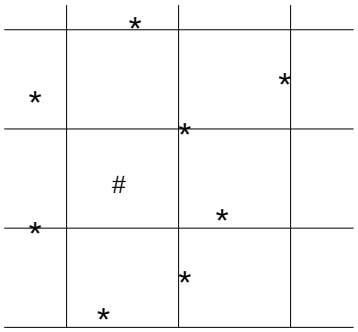
[1] CAPMS does not model individual exposures to these pollutants.

[2] The edge of the modeling domain has missing data, so the domain with actual observations extends from longitude -98.5° to -67.5° and latitude 26.33° to 46.67°.

[3] EPA has a direct link to the AIRS database: http://www.epa.gov/airs/; however, the data used in this analysis were downloaded from the (password-protected) mainframe version of AIRS, available at: epaibm.rtpnc.epa.gov. Both sets of data are identical; the mainframe allows larger data queries.

In calculating adjustment factors, the UAM-V modeled hourly values from 8:00 am to 7:59 pm are sorted by concentration level for the base-year and the future-year.[4] For each set of modeled data, we split evenly the ordered hourly values into the ten rank-ordered deciles,[5] selecting the average of hourly values in each decile as the representative value for that decile. This means that the first decile's representative ozone level equals the average of values within that decile, and so on for the other deciles. We then calculate the decile adjustment factors as the ratio of the UAM-V future-year scenario's decile to the corresponding UAM-V base-year's decile. We do similar calculations when determining the decile adjustment factors for the future baseline and for the control scenarios.

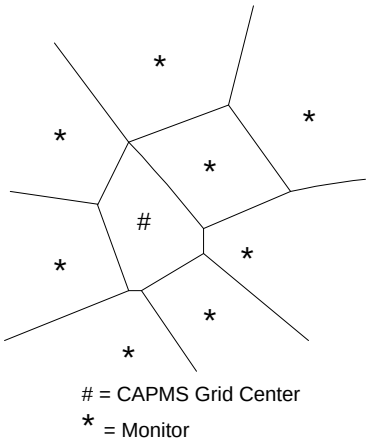
We use enhanced Voronoi Neighbor Averaging (eVNA) to interpolate air quality at every population grid cell by first identifying the set of monitors that best "surround" the center of the grid cell. We consider each CAPMS grid-cell separately, and identify the monitors that are close to the center of the grid-cell.



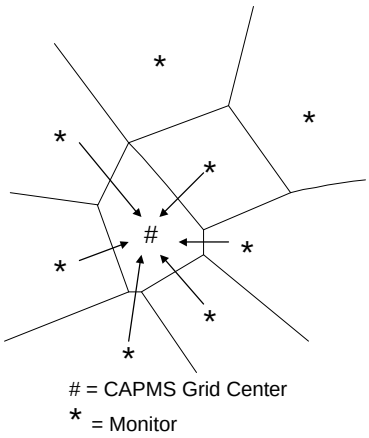
In particular, we identify the nearest monitors, or "neighbors," by drawing a polygon, or "Voronoi" cell, around the center of each CAPMS grid cell. The polygons have the special property that the boundaries of the polygon are as close as possible to the center of the CAPMS grid cell and that all of the points on each boundary are the same distance from the center of the cell and the monitor sharing this boundary.

[4] The data format of Eastern UAM-V modeled hourly output presents all grid cell data starting at 12:00 am.. In processing of data, a correction was encoded to ensure that calculations were based on 8:00 am to 7:59 pm of the appropriate local time zone of the grid cell.

[5] The use of more adjustment factors is generally considered desirable because it provides flexibility; however, it can lead to unreasonably large adjustment factors for lower ozone values, unless a threshold is used (e.g., one ppb as used in this analysis).



We calculate an inverse-distance weight for each of these monitors, including those monitors that share a boundary with a CAPMS grid cell in the subset of monitors that best surround the center of the grid cell.



We then combine the weights with the decile adjustment factors and the ozone monitoring data, to calculate hourly ozone values at each CAPMS grid cell in the Eastern U.S.:

$$CAPMS\,cell_{i,j,k,2030} = \left(UAMV_{i,j,2030}\right) \cdot \left(\sum_{h=1}^{N} \frac{mon_{h,j,k,1995}}{UAMV_{h,j,1995}} \cdot d_{h,i} \right)$$

where:

$CAPMS\ cell_{i,j,k,2030}$ = predicted concentration at CAPMS cell *i*, decile group j, hourly observation k

$UAMV_{i,,j,2030}$ = average UAMV modeled 2030 concentration in decile group j of model gridcell closest to CAPMS cell *i*

N = number of neighboring monitors for CAPMS gridcell i

$mon_{h,j,k,1995}$ = observed 1995 ozone level at monitor h, decile group j, hourly observation k

$UAMV_{h,j,1995}$ = average UAMV modeled 1995 concentration in decile group j of model gridcell closest to monitor h

$d_{h,i}$ = inverse-distance weight for cell *i* to monitor h .

The hourly ozone value assigned to each CAPMS gridcell is basically a distance-weighted average of adjusted ozone levels from nearby ozone monitors, where we adjust each of the monitor values with the ratio of UAM-V predictions at the CAPMS cell location and UAM-V predictions at the monitor.

For example consider the following situation where we have three monitors that are the nearest neighbor to a CAPMS grid-cell. We focus on one CAPMS grid-cell whose centered we marked with "#." Around this are nine UAM-V cells labeled A through I, with values in parts per billion (ppb); UAM-V cell "E" contains the CAPMS grid-cell of interest. And there are three monitors labeled 1 through 3; we mark each with a "*" and include the distance to the CAPMS grid-cell.

UAM-V values: 1995 90 ppb 2030 75 ppb A	UAM-V values: 1995 95 ppb 2030 80 ppb B	UAM-V values: 1995 100 ppb 2030 80 ppb C * 1995 Monitor 1=100ppb (20 miles)
UAM-V values: 1995 95 ppb 2030 70 ppb D * 1995 Monitor 2=80ppb (16 miles)	UAM-V values: 1995 40 ppb 2030 30 ppb E # CAPMS Cell	UAM-V values: 1995 60 ppb 2030 40 ppb F
UAM-V values: 1995 70 ppb 2030 50 ppb G	UAM-V values: 1995 80 ppb 2030 60 ppb H	UAM-V values: 1995 100 ppb 2030 90 ppb I * 1995 Monitor 3=120 ppb (14 miles)

In the enhanced VNA (eVNA) method, we incorporate UAM-V modeling information by including adjustment factors (i.e., E/C; E/D, and E/I) based on the direct air quality model results:

$$CAPMS\ gridcell_i = mon_1 \cdot \frac{E_{2030}}{C_{1995}} \cdot d_{i,1} + mon_2 \cdot \frac{E_{2030}}{D_{1995}} \cdot d_{i,2} + mon_3 \cdot \frac{E_{2030}}{I_{1995}} \cdot d_{i,3}\ .$$

Using the numbers in the table, the estimated air quality at the CAPMS grid-cell would be:

$$CAPMS\ gridcell_i = 100 \cdot \frac{30}{100} \cdot d_{i,1} + 80 \cdot \frac{30}{95} \cdot d_{i,2} + 120 \cdot \frac{30}{110} \cdot d_{i,3}.$$

The final step in the calculation is to include the inverse-distance weights $d_{i,1}$, $d_{i,2}$, and $d_{i,3}$. The further the monitor is from the CAPMS grid-cell, the smaller the weight. In this example, we calculate $d_{i,1}$ as follows:

$$d_{i,1} = \frac{\frac{1}{20}}{\left(\frac{1}{20} + \frac{1}{16} + \frac{1}{14}\right)} = 0.27\ .$$

Using the same type of calculation, we find the weights for $d_{i,2} = 0.34$ and $d_{i,3} = 0.39$, and as expected, the weights sum to one. We can then finish the calculation:

$$CAPMS\ gridcell_i = 100 \cdot \frac{30}{100} \cdot 0.27 + 80 \cdot \frac{30}{95} \cdot 0.34 + 120 \cdot \frac{30}{110} \cdot 0.39 = 29.5\ ppb.$$

After calculating both baseline and control hourly ozone levels at each CAPMS gridcell, we then calculate the ozone measures that are needed to estimate adverse health effects. For example, a number of studies use the 24-hour daily average ozone level, so for each CAPMS gridcell we get 2030 baseline and control estimates for the 24-hour daily average.

To reduce computational time when estimating the change in health effects associated with daily ozone levels, CAPMS approximates a season's worth of daily ozone measures at each CAPMS gridcell by 20 "bins." Each bin represents five percent of the daily ozone concentrations, and the value for each bin is set at the midpoint of the percentile range it represents. The first bin represents the first (lowest) five percent of the distribution of daily ozone values, and is set at the 2.5th percentile value; the second bin represents the next five percent of the distribution of daily values, and is set at the 7.5th percentile value, and so on. Each of the twenty bins therefore represents 7.65 (=153/20) days, since there are 153 days between May and September.

After generating 20 bins for both the baseline and control scenarios, we take the difference between these two values at each bin. We subtract the baseline value in the first bin from the control value in the first bin, and so on for each of the 20 bins. For each CAPMS gridcell, we then get 20 values representing the difference between the baseline and control, and we use these to estimate the change in adverse effects associated with the implementation of the policy. Note that since each value represents 7.65 days, we then multiply each of the 20 incidence change estimates by 7.65 to reconstruct an entire season's worth of incidence changes in the CAPMS grid cell.

2.2 PM Air Quality

We estimated the reduction in PM-related adverse effects based on the Agency's application of a national-scale version of the Regulatory Model System for Aerosols and Deposition (REMSAD). REMSAD was developed as an extension of the episodic UAM-V regional model. Like UAM-V,

REMSAD is a three-dimensional grid-based Eulerian air quality model designed to estimate annual particulate concentrations and deposition over large spatial scales (e.g., over the contiguous U.S.). Consideration of the different processes that affect primary (directly emitted) and secondary (formed by atmospheric processes) PM at the regional scale in different locations is fundamental to understanding and assessing the effects of proposed pollution control measures that affect ozone, PM and deposition of pollutants to the surface.[6] Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is useful for evaluating the impacts of the HD Engine/Diesel Fuel rule on U.S. PM concentrations.

For use in this benefits analysis, the Agency applied the modeling system to the entire U.S. for two future-year scenarios: a 2030 base case and a 2030 HD Engine/Diesel Fuel control scenario. The modeling domain encompasses the contiguous 48 States. The domain extends from 126 degrees west longitude to 66 degrees west longitude, and from 24 degrees north latitude to 52 degrees north latitude. The model contains horizontal grid-cells across the model domain of roughly 36 km by 36 km. There are 8 vertical layers of atmospheric conditions with the top of the modeling domain at roughly 16,000 meters. The 36 by 36 km horizontal grid results in a 120 by 92 grid (or 10,080 grid-cells) for each vertical layer.

We assigned each CAPMS grid cell to the nearest REMSAD grid cell, by calculating the shortest distance between the center of the CAPMS grid cell to the center of a REMSAD grid cell. Note that we use REMSAD data in an absolute sense, unlike the case with UAM-V where we use the modeling results in a relative sense and scale ozone monitoring data. We use the REMSAD modeling directly because there are no clear biases in the modeling results, and perhaps most importantly, there is not a widespread network of $PM_{2.5}$ monitors that we could use in conjunction with the REMSAD modeling data.

[6] Given the potential impact of the HD Engine/Diesel Fuel rule on secondarily formed particles it is important to employ a Eulerian model such as REMSAD. The impact of secondarily formed pollutants typically involves primary precursor emissions from a multitude of widely dispersed sources, and chemical and physical processes of pollutants that are best addressed using an air quality model that employs an Eulerian grid model design.

3 General Issues in Estimating Health and Welfare Benefits

Changes in ozone, PM, and visibility levels result in changes in a number of health and welfare effects, or "endpoints," that society values. This chapter discusses key issues in the estimation of adverse health effects and in the valuation of health and welfare benefits. Section 1 describes general issues that particularly affect the estimation of changes in health effects. Section 2 describes general issues in valuing health and welfare changes. Finally, Section 3 discusses how uncertainty is characterized in this analysis.

3.1 Estimating Adverse Health Effects

This section reviews issues that arise in the estimation of adverse health effects. It reviews the derivation of C-R functions, and it reviews how CAPMS combines air quality data and C-R functions. In addition, we discuss how we handle overlapping health effects, thresholds, estimating the baseline incidence rates for the C-R functions, and other issues.

3.1.1 Basic Concentration-Response Model

The methods discussed in this sub-section apply to the estimation of both ozone-related and PM-related changes in adverse health effects. For expository simplicity, the discussion focuses primarily on PM-related changes. The methods, however, are equally applicable to ozone-related changes in effects. Similarly, while several health endpoints have been associated with ozone and PM, the discussion below refers only to a generic "health endpoint," denoted as y. Finally, the discussion refers to estimation of changes in the incidence of the health endpoint at a single location (the population cell, which is equivalent to the CAPMS gridcell). Region-wide changes are estimated by summing the estimated changes over all population cells in the region.

Different epidemiological studies may have estimated the relationship between PM and a particular health endpoint in different locations. The C-R functions estimated by these different studies may differ from each other in several ways. They may have different functional forms; they may have measured PM concentrations in different ways; they may have characterized the health endpoint, y, in slightly different ways; or they may have considered different types of populations. For example, some studies of the relationship between ambient PM concentrations and mortality have excluded accidental deaths from their mortality counts; others have included all deaths. One study may have measured daily (24-hour) average PM concentrations while another study may have used two-day averages. Some studies have assumed that the relationship between y and PM is best described by a linear form (i.e., the relationship between y and PM is estimated by a linear regression in which y is the dependent variable and PM is one of several independent variables). Other studies have assumed that the relationship is best described by a log-linear form (i.e., the relationship between the natural logarithm of y and PM is estimated by a linear regression).[7] Finally, one study may have considered changes in the health endpoint only among members of a particular

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

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

The estimated relationship between PM and a health endpoint in a study location is specific to the type of population studied, the measure of PM used, and the characterization of the health endpoint considered. For example, a study may have estimated the relationship between daily average PM concentrations and daily hospital admissions for "respiratory illness," among individuals age 65 and older, where "respiratory illness" includes International Classification of Disease (ICD) codes A, B, and C.[8] If any of the inputs had been different (for example, if the entire population had been considered, or if "respiratory illness" had consisted of a different set of ICD codes), the estimated C-R function would have been different. When using a C-R function estimated in an epidemiological study to estimate changes in the incidence of a health endpoint corresponding to a particular change in PM in a population cell, then, it is important that the inputs be appropriate for the C-R function being used -- i.e., that the measure of PM, the type of population, and the characterization of the health endpoint be the same as (or as close as possible to) those used in the study that estimated the C-R function.

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

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

or, equivalently,

$$ln(y) = a + b \cdot PM \ ,$$

where the parameter B is the incidence of y when the concentration of PM is zero, the parameter β is the coefficient of PM, ln(y) is the natural logarithm of y, and $\alpha = \ln(B)$.[9] If the functional form of the C-R relationship is log-linear, the relationship between ΔPM and Δy is:

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

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

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

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

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

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

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

The linear relationship is of the form:

$$y = a + b \cdot PM \quad ,$$

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

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

$$\Delta y = b \cdot \Delta PM \quad .$$

A few epidemiological studies, estimating the relationship between certain morbidity endpoints and PM, have used functional forms other than linear or log-linear forms. Of these, logistic regressions are the most common. Abt Associates (1999a, Appendix A) provides further details on the derivation of dose-response functions.

3.1.2 Calculation of Adverse Health Effects with CAPMS

CAPMS is a population-based system for modeling population exposure to ambient levels of criteria air pollutants and estimating the adverse health effects associated with this exposure. CAPMS divides the United States into multiple grid cells, and estimates the changes in incidence of adverse health and welfare effects associated with given changes in air quality in each grid cell. The national incidence change (or the changes within individual states or counties) is then calculated as the sum of grid-cell-specific changes.

To calculate point estimates of the changes in incidence of a given selection of adverse health and welfare effects associated with a given set of air quality changes, CAPMS goes through the following steps at each CAPMS grid cell:

- Interpolate the air quality in the baseline scenario and in the control scenario at the CAPMS grid cell center, as described in Chapter 2. If the daily values have been binned at the monitors from

which the interpolation is carried out, the resulting baseline and control scenario air quality data at the CAPMS grid cell center is also binned.

- Calculate the changes in air quality from baseline to control scenario in the CAPMS grid cell. The changes in air quality are calculated as the differences between the baseline (daily, annual, or bin) values and the corresponding control scenario (daily, annual, or bin) values. The change in the n^{th} daily or bin concentration is the difference between the baseline n^{th} daily or bin concentration and the control scenario n^{th} daily or bin concentration.

- Access the selected C-R functions being used, and the required baseline incidence rates and grid cell population.

- Using the above inputs, calculate the change in incidence of each adverse health effect for which a C-R function has been accessed.

For functions based on changes in daily average pollutant concentrations, estimated incidence changes corresponding to air quality changes on each of 365 days or in each of the 20 bins are summed. When binning is used, this summed incidence is the result of 20 representative air quality changes (one for each bin). Recall that each bin represents 18.25 days for PM (to represent a year's worth of exposure) and 7.65 days for ozone (to represent an ozone season's worth of exposure). To adjust the summed incidence estimate, it is multiplied by either 18.25 to produce an annual change, or by 7.65 to produce a seasonal change. This procedure is applied to each grid cell in CAPMS. The resulting incidence change is stored, and CAPMS proceeds to the next grid cell, where the above process is repeated. The national change (or the change in any designated geographical area) is calculated at the end of the process by summing the grid cell-specific changes.

To reflect the uncertainty surrounding predicted incidence changes resulting from the sampling uncertainty surrounding the pollutant coefficients in the C-R functions used, CAPMS produces a *distribution* of possible incidence changes for each adverse health, rather than a single point estimate. To do this, it uses both the point estimate of the pollutant coefficient (β in the above equation) and the standard error of the estimate to produce a normal distribution with mean equal to the estimate of β and standard deviation equal to the standard error of the estimate. Using a Latin Hypercube method,[10] we take the n^{th} percentile value of β from this normal distribution, for n = 0.5, 1.5, ..., 99.5, and follow the procedure outlined in the section above to produce an estimate of the incidence change, given the β selected. Repeating the procedure for each value of β selected results in a distribution of incidence changes in the CAPMS grid cell. This distribution is stored, and CAPMS proceeds to the next grid cell, where the process is repeated. A distribution of the national change (or change in a designated geographical area) is calculated by summing the n^{th} percentile grid cell-specific changes, for n = 0.5, 1.5, ..., 99.5.

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

3.1.3 Population Projections

Benefits for the HD Engine/Diesel Fuel rule analysis are based on health and welfare effect incidence changes due to predicted air quality improvements in the year 2030. Integral to the estimation of such benefits is an accurate estimate of future population projections. This section describes the method used to estimate county-level 2030 populations.

The underlying data used to create county-level 2030 population projections is based on: (1) 1990 county-level population statistics for all U.S. counties collected by the U.S. Census (Wessex, 1994), and (2) future-year state and metropolitan area population estimates provided by the Bureau of Economic Analysis (1995). Growth factors are calculated using the BEA data and are applied to the 1990 county-level populations.

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

Growth factors are calculated for both urban areas and rural areas. An urban area is defined as a county that falls within a metropolitan area. This includes metropolitan statistical areas (MSAs), primary metropolitan statistical areas (PMSAs), consolidated metropolitan statistical areas (CMSAs), and New England county metropolitan areas (NECMAs), as defined by U.S. Census Bureau.[11] In this section, however, all metropolitan areas are referred to as MAs. A rural area is defined as a county that falls outside the defined metropolitan areas.

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

$$2030CountyPop_i = 1990CountyPop_i \cdot \frac{2030\,MAPop_i}{1990\,MAPop_i}$$

where:

$2030CountyPop_i$ = projected 2030 population in urban county i
$1990CountyPop_i$ = actual 1990 population for county i
$2030MAPop_i$ = projected 2030 population in metropolitan area for county i
$1990MAPop_i$ = actual 1990 population for metropolitan area for county i.

Rural areas grow according to the growth rate calculated for the particular state within which they are located, adjusted to subtract out metropolitan area populations. Before the ratio of future year to base year state population is calculated, the population attributed to all metropolitan areas located within that state is subtracted from the future year and base year population totals. Once this metropolitan area

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

adjustment has been made, the rural growth factor is multiplied by the base-year population in all non-MA counties to get future-year population projections.

To calculate 2030 population, we use the following equaiton:

$$2030CountyPop_i = 1990CountyPop_i \cdot \frac{(2030StatePop_i - \sum 2030MAPop_i)}{(1990StatePop_i - \sum 1990MAPop_i)}$$

where:

$2030CountyPop_i$ = projected 2030 population in rural county i
$1990CountyPop_i$ = actual 1990 population for county i
$2030StatePop_i$ = projected 2030 population in state where county i is located
$1990State\ Pop_i$ = actual 1990 population for state where county i is located
$\Sigma 2030MAPop_i$ = projected 2030 population in metropolitan areas located in state with county i
$\Sigma 1990MAPop_i$ = actual 1990 population for metropolitan areas located in state with county i .

One problem that exists with this method is that many metropolitan areas cross state boundaries. To accurately subtract urban populations from state populations, we need to know the urban county populations for both 1990 and 2030. Using the county populations for 1990, we can estimate the portion of a particular metropolitan area's population that belongs to a given state. However, we do not have 2030 county population projections with which to apportion 2030 metropolitan area populations. To remedy this, we apply the same percent of the population a given county contributes to a metropolitan area in 1990 to 2030 metropolitan areas when apportioning populations between states.

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

3.1.4 Overlapping Health Effects

Several endpoints reported in the health effects literature overlap with each other. Hospital admissions for single respiratory ailments (e.g. pneumonia) overlap with estimates of hospital admissions

for "all respiratory" ailments.[12] Similarly, several studies quantify the occurrence of respiratory symptoms where the definitions of symptoms are not unique (e.g., shortness of breath or upper respiratory symptoms). In choosing studies to include in the aggregated benefits estimate (discussed below), this analysis carefully avoids double-counting benefits that might arise from overlapping health effects. Specific methods for avoiding double-counting of benefits are described in detail in the sections discussing health effects for which this is an issue.

3.1.5 Baseline Incidences

As noted above, most of the relevant C-R functions are log-linear, and the estimation of incidence changes based on a log-linear C-R function requires a baseline incidence. The baseline incidence for a given CAPMS population cell is the baseline incidence rate in that location multiplied by the relevant population. County mortality rates are used in the estimation of air pollution-related mortality, and all CAPMS population cells in the county are assumed to have the same mortality rate. Hospital admissions are only available at the national level, so all areas are assumed to have the same incidence rate for a given population age group. For some endpoints, such as respiratory symptoms and illnesses and restricted activity days, baseline incidence rates are not available even at the national level. The only sources of estimates of baseline incidence rates in such cases are the studies reporting the C-R functions for those health endpoints. The baseline incidence rate and its source are given for each C-R function in Appendices B and C.

3.1.6 Thresholds

A very important issue in applied modeling of changes in PM is whether to apply the C-R functions to all predicted changes in ambient concentrations, even small changes occurring at levels approaching the concentration in which they exist in the natural environment (without interference from humans), referred to as "anthropogenic background." Different assumptions about whether to model thresholds, and if so, at what levels, can have a major effect on the resulting benefits estimates.[13]

In this analysis, we do not use thresholds in any of the epidemiological functions relating PM or ozone to various health and welfare endpoints. We assume that all of these functions are continuous and differentiable down to zero pollutant levels.

There is some evidence that, at least for particulate matter, not only is there no threshold, but the PM coefficient may actually be larger at lower levels of PM and smaller at higher levels. Examining the relationship between particulate matter (measured as TSP) and mortality in Milan, Italy during the ten year period 1980-1989, Rossi et al. (1999) fitted a model with one slope across the entire range of TSP and an additional slope for TSP greater than 200 $\mu g/m^3$. The second slope was statistically significant ($p<0.0001$) and negative, indicating a lower slope at higher TSP levels.

[12]Pneumonia is often classified with the International Classification of Diseases (ICD) codes of 480-486, while all respiratory admissions are classified with ICD codes 460-519.

[13]Thresholds may also apply to ozone, however, recent RIAs have not explicitly modeled ozone thresholds.

Schwartz (2000b, p. 566) examined the relationship between PM_{10} and mortality in ten U.S. cities and reported similar results. When restricting his model to days with PM_{10} levels below 50 $\mu g/m^3$, Schwartz found a larger effect for PM_{10}, in comparison to a model that included all days.

If desired, a threshold may be imposed on these models in several ways, and there are various points at which the threshold could be set. Some points are obvious candidates, such as the background level of the pollutant or a relevant standard for the pollutant. Whatever the threshold, the implication is that there are no effects below the threshold.

A threshold model can be constructed in more than one way. One method is to simply truncate the C-R function at the threshold (i.e., to not include any physical effect changes associated with PM concentrations below the designated threshold). This method uses the original C-R function, but calculates the change in PM as [max(T,baseline PM) - max(T, regulatory alternative PM)], where T denotes the designated threshold. This threshold model will predict a smaller incidence of the health effect than the original model without a threshold. Clearly, as T increases, the predicted incidence of the health effect will decrease.

An alternative method is to replace the original C-R function with a "hockey stick" model that best approximates the original function that was estimated using actual data. The hockey stick model is horizontal up to a designated threshold PM level, T, and is linear with a positive slope for PM concentrations greater than T. Recall the log-linear C-R function:

$$y = a + b \cdot PM \ .$$

Assuming that the value of the coefficient, β, depends on the level of PM, we get:

$$\ln(y) = a' \ , \quad for\ PM \le T \ , and$$
$$\ln(y) = a' + b' \cdot PM \ \ , \ for\ PM > T \ .$$

Ideally, the coefficients would be estimated based on the data in the original study – that is, a hockey stick model would be fit to the original data, so that the threshold model that is most consistent with the available information would be chosen. If a threshold model could be estimated from the original data, it is unlikely that α' would equal α or that β' would equal β, because such a hockey stick model would be consistently below the original model, except at PM=0 (where the two models would coincide). If that were the hockey stick model that best fit the data, then it is unlikely that the best fitting linear model would be consistently above it. Instead, the hockey stick model that best fits the same data would most likely have $\alpha'>\alpha$ and $\beta'>\beta$. A graph of this model would therefore cross the graph of the linear model at two points. Whether such a hockey stick threshold model predicted a greater or smaller incidence of the health effect than the linear model would depend on the distribution of PM levels. It is worth noting that the graph of the first type of threshold model, in which the C-R function is simply truncated at the threshold, would be discontinuous at the threshold. This is highly unlikely to be a good model of the actual relationship between PM and any health endpoint.

3.1.7 Application of a Single C-R Function Everywhere

Whether the C-R relationship between a pollutant and a given health endpoint is estimated by a single function from a single study or by a pooled function of C-R functions from several studies, that same C-R relationship is applied everywhere in the benefits analysis. Although the C-R relationship may in fact vary somewhat from one location to another (for example, due to differences in population susceptibilities or differences in the composition of PM), location-specific C-R functions are available only for those locations in which studies were conducted. While a single function applied everywhere may result in overestimates of incidence changes in some locations and underestimates of incidence changes in other locations, these location-specific biases will to some extent cancel each other out when the total incidence change is calculated. It is not possible to know the extent or direction of the bias in the total incidence change based on application of a single C-R function everywhere.

3.1.8 Estimating Pollutant-Specific Benefits Using Single Pollutant vs. Multi-Pollutant Models

Many studies include both ozone and particulate matter in their final models. It is often difficult to separate out the effect of a single pollutant from the effects of other pollutants in the mix. Multi-pollutant models have the advantage that the coefficient for a single pollutant in such a model will be unbiased (so that the effects of other pollutants will not be attributed falsely to the single pollutant). However, the variance of the estimator of the coefficient of the pollutant of interest will increase as the correlations between the other pollutants in the model and that pollutant increase. If the other pollutants in the model are highly correlated with the pollutant of interest, we would have an unbiased but unstable (high variance) estimator. However, while single pollutant models have the advantage of more stable estimators, the coefficient estimate in a single pollutant model could be biased in such a model. We could consider the single pollutant as an "indicator pollutant" – i.e., an indicator of a pollution mix – if we use single pollutant models. However, there is no guarantee that the composition of the pollution mix will remain the same under a control scenario that targets only a single pollutant.

This analysis uses both single pollutant and multi-pollutant models to derive pollutant-specific benefits estimates. When more than one study has estimated the relationship between a given endpoint and a given pollutant, information from both single-pollutant and multi-pollutant models may be pooled to derive pollutant-specific benefits estimates. For example, the benefits predicted by a model with only PM may be pooled with the benefits predicted by a model with both PM and ozone to derive an estimate of the PM-related benefits associated with a given endpoint. If the benefits of PM-related and ozone-related incidence changes are both being calculated and added together, there is the possibility of overestimating benefits if some of the studies used are single pollutant models.

If ozone is actually associated with a given endpoint, but PM appears to be associated only because it is correlated with ozone, then there is the potential for problems. In this case, the benefits predicted by a single pollutant PM model would actually reflect the benefits of reducing ozone, to the extent that PM and ozone are correlated. If those "PM-related" benefits were then added to the ozone-related benefits calculated from other models, a likely result would be the overstatement of benefits of reducing ozone. To avoid this problem, we prefer to use models that include both ozone and PM.

3.1.9 Pooling Study Results

When only a single study has estimated the C-R relationship between a pollutant and a given health endpoint, the estimation of a population cell-specific incidence change, Δy, is straightforward, as noted above. When several studies have estimated C-R relationships between a pollutant and a given health

endpoint, the results of the studies can be pooled to derive a single estimate of the function. If the functional forms, pollutant averaging times, and study populations are all the same (or very similar), a pooled, "central tendency" C-R function can be derived from multiple study-specific C-R functions. Even if there are differences among the studies, however, that make a pooled C-R function infeasible, a pooled estimate of the incidence change, Δy, and/or the monetary benefit of the incidence change can be obtained by incorporating the appropriate air quality data into the study-specific C-R functions and pooling the resulting study-specific predictions of incidence change. Similarly, study-specific predictions of incidence change can be combined with unit dollar values to produce study-specific predictions of benefits.

Whether the pooling is done in "coefficient space," "incidence change space," or "dollar space," the question of the relative weights assigned to the estimates (of coefficients, incidence changes, or dollar benefits) from each input study must be addressed. One possibility is simply averaging the estimates from all the studies. This has the advantage of simplicity, but the disadvantage of not taking into account the measured uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty.

An alternative approach to pooling incidence estimates from different studies is to give more weight to studies with little estimated variance than to studies with a great deal of estimated variance. The exact way in which weights are assigned to estimates from different studies in a pooled analysis depends on the underlying assumption about how the different estimates are related to each other. Under the assumption that there is actually a distribution of true effect coefficients, or β's, that differ by location and/or study (referred to as the random effects model), the different coefficients reported by different studies may be estimates of *different* underlying coefficients, rather than just different estimates of the same coefficient. In contrast to the "fixed-effects" model (which assumes that there is only one β everywhere), the random-effects model allows the possibility that different studies are estimating different parameters.[14] Note that both methods tend to bias towards smaller estimates.

A third approach to pooling studies is to apply subjective weights to the studies, rather than conducting a random effects pooling analysis. If the analyst is aware of specific strengths and weaknesses of the studies involved, this prior information may be used as input to the calculation of weights which reflect the relative reliability of the estimates from the studies.

In those cases in which pooling of information from multiple studies was an option in this analysis, pooling was done in both "incidence change space" and "dollar benefit space." The hypothesis of fixed effects was tested. If this hypothesis was rejected, an underlying random effects model was used as the basis for weighting of studies. A more detailed description of the pooling procedure used is given below in the section on hospital admissions.

[14] In studies of the effects of PM_{10} on mortality, for example, if the composition of PM_{10} varies among study locations the underlying relationship between mortality and PM_{10} may be different from one study location to another. For example, fine particles make up a greater fraction of PM_{10} in Philadelphia County than in Southeast Los Angeles County. If fine particles are disproportionately responsible for mortality relative to coarse particles, then one would expect the true value of β for PM_{10} in Philadelphia County to be greater than the true value of β for PM_{10} in Southeast Los Angeles County. This would violate the assumption of the "fixed effects" model. However, applying a random effects model assumes that the observed set of coefficients in the policy region.

3.2 Valuing Changes in Health And Welfare Effects

This section discusses a number of issues that arise in valuing changes in health and welfare effects. The first section provides some background on willingness to pay (WTP). The second section discusses the possibility that as income changes then WTP would also change. The third section describes how WTP estimates, that were originally calculated in 1990 dollars, are corrected for inflation to get estimates in 1999 dollars. In the last section, we briefly review how we aggregate benefits estimates.

3.2.1 WTP Estimation

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

While the estimation of WTP for a market good (i.e., the estimation of a demand schedule) is not a simple matter, the estimation of WTP for a non-market good, such as a decrease in the risk of having a particular health problem, is substantially more difficult. Estimation of WTP for decreases in very specific health risks (e.g., WTP to decrease the risk of a day of coughing or WTP to decrease the risk of admission to the hospital for respiratory illness) is further limited by a paucity of information.[15] Derivation of the dollar value estimates discussed below was often limited by available information.

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

3.2.2 Change Over Time in WTP in Real Dollars

The WTP for health-related environmental improvements (in real dollars) could change between now and the year 2030. If real income increases between now and the year 2030, for example, it is reasonable to expect that WTP, in real dollars, would also increase. Based on historical trends, the U.S. Bureau of Economic Analysis projects that, for the United States as a whole as well as for regions and states within the U.S., mean per capita real income will increase. For the U.S. as a whole, for example, mean per capita personal income is projected to increase by about 16 percent from 1993 to 2005 (U.S. Bureau of Economic Analysis, 1995).

Although the monetary benefits presented in this Technical Support Document (TSD) have not been adjusted to account for changes over time in real income[16], such adjustments were made to the dollar benefits presented in this TSD, as described in Chapter VII of the corresponding Regulatory Impact Analysis (RIA) for the Final HD Engine/Diesel Fuel rule.

3.2.3 Adjusting Benefits Estimates from 1990 Dollars to 1999 Dollars

This section describes the methods used to convert benefits estimates into constant dollars. In past RIA analyses, cost and benefit estimates have been presented in constant 1990 dollars. Benefits estimates in this analysis, however, are presented in constant 1999 dollars. To adjust benefits estimates from 1990 dollars to 1999 dollars, the method of adjustment depends on the basis of the benefits estimates. These methods are presented below. Four different bases of estimates are delineated in Exhibit 3-1, including that for agricultural benefits.[17]

Exhibit 3-1 Bases of Benefits Estimation

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

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

[16] An exception to this is the aggregate benefits, presented in Exhibit 6-5.

[17] Agricultural benefits are discussed in Chapter 5.

general rate of inflation, using a general inflator (the CPI-U for "all items" or some other general inflator) to adjust from 1990 to 1999 dollars would downward bias cost-of-illness estimates in 1999 dollars.

Benefits estimates based directly on estimates of WTP have been adjusted using the CPI-U for "all items." The CPI-Us, published by the U.S. Dept. of Labor, Bureau of Labor Statistics, can be found in Council of Economic Advisers (2000, Table B-58). An overview of the adjustments from 1990 to 1999 dollars for WTP-based and cost-of-illness based valuations is given in Exhibit 3-2.

Exhibit 3-2 Consumer Price Indexes Used to Adjust WTP-Based and Cost-of-Illness-Based Benefits Estimates from 1990 Dollars to 1999 Dollars

	1990 (1)	1997 (2)	1999 (3)	Adjustment Factor [a] (3)/(1)	Adjustment Factor [a] (3)/(2)	Relevant Endpoints
CPI-U for "All Items" [b]	130.7	160.5	166.6	1.275	1.038	WTP-based valuation: 1. Statistical lives saved [c] 2. Chronic bronchitis 3. Chronic asthma 4. Morbidity endpoints using WTP [d]
CPI-U for Medical Care [b]	162.8	234.6	250.6	1.539	1.0682	Cost-of-illness based valuation: Hospital admissions avoided [e]

[a] Benefits estimates in 1990 dollars are multiplied by the adjustment factor to derive benefits estimates in 1999 dollars.

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

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

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

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

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

The benefits estimates for WLDs avoided can be put into 1999 dollars in several ways. One approach is to obtain the 1998 median weekly earnings (the most up-to-date measure of earnings available), divide by five to derive the median daily earnings, and adjust the median earnings from 1998 to 1999 dollars. This is an alternative to relying on adjustments from 1990 to 1999 dollars. The median weekly earnings of full-time wage and salary workers in 1998 was $523 (U.S. Bureau of the Census 1998, Table 696). This implies a median daily earnings of $104.6, or rounded to the nearest dollar, $105. Alternatively, we can adjust the median daily wage for 1990 to 1999 dollars, using the CPI-U for "all

items." The result turns out to be the same. The adjustment factor (the ratio of the 1999 CPI-U to the 1990 CPI-U) is 1.275. Applied to the median daily earnings of $82.4 in 1990, the median daily earnings in 1999 would be $105.1, or rounded to the nearest dollar, $105.

Consistent with economic welfare theory, the valuation of benefits associated with increased worker productivity resulting from improved ozone air quality used the average daily income for outdoor workers engaged in strenuous activity, reported by the 1990 U.S. Census ($73 per day, in 1990). Using the CPI-U for "all items," the adjustment factor to adjust from 1990 to 1999 dollars (the ratio of the 1999 CPI-U to the 1990 CPI-U) is 1.275. Applied to the average daily earnings of $73 in 1990, the average daily earnings in 1999 would be $93.08, or rounded to the nearest dollar, $93.

Finally, agricultural benefits (changes in farm income and consumer welfare) predicted to result in a future year have been adjusted to 1999 dollars from 2010 using a GDP price deflator. In this analysis, 2010 benefits were adjusted to 1999 dollars by multiplying by 0.6735, the ratio of the 1999 GDP price deflator (of 112.3 from:Council of Economic Advisers, 1997, Table B-3) to a projected 2010 GDP price index (of 167.16) forecasted from the trend between 1997 and 2007, obtained from the USDA baseline projections (U.S. Department of Agriculture, 1988b, electronic file Tab01.wk1).

3.2.4 Aggregation of Monetized Benefits

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

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

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

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

Assuming that WTP to avoid the risk of a health effect varies from one individual to another, there is a *distribution* of WTPs to avoid the risk of that health effect. This population distribution has a mean. It is this population mean of WTPs to avoid or reduce the risk of the j^{th} health effect, $MWTP_j$, that is the

appropriate value in the benefit analysis.[18] The monetized benefit associated with the j^{th} health endpoint resulting from attainment of standard(s) in the k^{th} location, then, is:

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

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

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

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

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

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

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

The total PM-related monetized benefit for the k^{th} location can now be rewritten as:

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

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

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

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

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

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

where TMB_k denotes the total monetized benefit achieved in the k^{th} location, and K is the number of locations.

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

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

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

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

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

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

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

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

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

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

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

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

Both the endpoint-specific coefficients (the $\ddot{y}_j$'s) and the endpoint-specific mean WTPs (the $MWTP_j$'s) are uncertain, as are the incidence rates and air quality changes. One approach to estimating the total monetized benefit is to simply use the mean values of the endpoint-specific coefficients and mean WTPs in the above formula. We term this approach the "simple mean." Alternatively, we can characterize not only the mean total monetized benefit but the distribution of possible values of total monetized benefit, using a Monte Carlo approach. The Monte Carlo approach has three steps. First, in each of 5000 iterations, we randomly select a value from the distribution of (national) incidence change of the health or welfare effect. Second, we randomly select a value from the distribution of unit dollar values for that health or welfare effect. And third, we multiply the two values. The result is a distribution of (5000) monetized benefits associated with the given health or welfare effect. From this distribution, we present the mean as well as the 5^{th} and 95^{th} percentiles. We discuss the background of the Monte Carlo in the following sub-section.

3.3 Characterization of Uncertainty

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

analysis. When compounded with uncertainty in later stages, small uncertainties in emissions can lead to much larger impacts on total benefits.

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

- gaps in scientific data and inquiry
- variability in estimated relationships, such as C-R functions, introduced through differences in study design and statistical modeling
- errors in measurement and projection for variables such as population growth rates
- errors due to misspecification of model structures, including the use of surrogate variables, such as using PM_{10} when $PM_{2.5}$ is not available, excluded variables, and simplification of complex functions
- biases due to omissions or other research limitations.

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

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

1. Uncertainties Associated With Concentration-Response Functions
-The value of the ozone- or PM-coefficient in each C-R function.
-Application of a single C-R function to pollutant changes and populations in all locations.
-Similarity of future year C-R relationships to current C-R relationships.
-Correct functional form of each C-R relationship.
-Extrapolation of C-R relationships beyond the range of ozone or PM concentrations observed in the study.
-Application of C-R relationships only to those subpopulations matching the original study population.
2. Uncertainties Associated With Ozone and PM Concentrations
-Responsiveness of the models to changes in precursor emissions resulting from the control policy.
-Projections of future levels of precursor emissions, especially ammonia and crustal materials.
-Model chemistry for the formation of ambient nitrate concentrations.
-Lack of ozone monitors in rural areas requires extrapolation of observed ozone data from urban to rural areas.
-Use of separate air quality models for ozone and PM does not allow for a fully integrated analysis of pollutants and their interactions.
-Full ozone season air quality distributions are extrapolated from a limited number of simulation days.
-Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern US and underpredicts nitrate in parts of the Western US.
3. Uncertainties Associated with PM Mortality Risk
-No scientific literature supporting a direct biological mechanism for observed epidemiological evidence. -Direct causal agents within the complex mixture of PM have not been identified. -The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures. -Possible confounding in the epidemiological studies of $PM_{2.5}$, effects with other factors (e.g., other air pollutants, weather, indoor/outdoor air, etc.). -The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study. -Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated With Possible Lagged Effects
-The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
5. Uncertainties Associated With Baseline Incidence Rates
-Some baseline incidence rates are not location-specific (e.g., those taken from studies) and may therefore not accurately represent the actual location-specific rates.
-Current baseline incidence rates may not approximate well baseline incidence rates in 2030.
-Projected population and demographics may not represent well future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
-Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them.
-Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors.
-Future markets for agricultural and forestry products are uncertain.
7. Uncertainties Associated With Aggregation of Monetized Benefits
-Health and welfare benefits estimates are limited to the available C-R functions. Thus, unquantified or unmonetized benefits are not included.

3.3.1 Alternative and Supplementary Calculations

The alternative calculations included in this analysis are based on relatively plausible alternatives to the assumptions used in deriving the primary benefit estimates. We do not attempt to assign probabilities to these alternative calculations, as we believe this would only add to the uncertainty of the analysis or present a false picture about the precision of the results[20]. Instead, the reader is invited to examine the impact of applying the different assumptions on the estimate of total benefits. While it is possible to combine all of the alternative calculations with a positive impact on benefits to form a "high" estimate or all of the alternative calculations with a negative impact on benefits to form a "low" estimate, we do not recommend this because the probability of all of these alternative assumptions occurring simultaneously is likely to be very low. Instead, the alternative calculations are intended to demonstrate the sensitivity of our benefits results to key parameters which may be uncertain. Exhibit 3-4 summarizes the alternative calculations included in this analysis.

Exhibit 3-4 also summarizes supplemental calculations prepared for this analysis. Supplemental calculations are intended to provide additional information about specific health effects, but are not suitable for inclusion in the primary or alternative estimates due to concerns about double-counting of benefits or the high degree of uncertainty about the estimates. Results from the supplemental calculations can be found in Appendix A.

Alternative Calculations

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

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

An alternative to the calculation of life-years lost is age-based adjustments to the value of a statistical life lost based on empirical estimates of WTP by age. Several studies conducted by Jones-Lee, et al. (1985; 1989; 1993) found a significant effect of age on the value of mortality risk reductions expressed

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

by citizens in the United Kingdom. We used the results of the Jones-Lee et al. analysis to calculate age-specific values of a statistical life. As described below, we started with the value of a statistical life lost by an individual of about age 40, and then adjusted it with age-specific factors. We use 40 as the base because we use wage risk studies in developing the value of a statistical life, and the average age in the wage-risk studies is about 40.

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

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

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

For this benefits analysis, we have adopted the C-R function for COPD and pneumonia hospital admissions from Samet et al. (2000a). This estimate, while representing the state of the art in epidemiological studies, is a good deal larger than the estimate from Moolgavkar et al. (1997). We explore the impact of using the Moolgavkar et al. estimate instead of the Samet et al. in the alternative calculations.

In the primary analysis we present an estimate of the number of avoided asthma attacks due to PM and ozone. However, due to uncertainty over the magnitude of the estimate, we present the value of avoided asthma attacks as an alternative calculation.

The alternative calculation for recreational visibility is an estimate of the full value of visibility in the entire region affected by the final HD Engine/Diesel Fuel rule. The Chestnut and Rowe (1990) study from which the primary valuation estimates are derived only examined WTP for visibility changes in the southeastern portion of the affected region. In order to obtain estimates of WTP for visibility changes in the northeastern and central portion of the affected region, we have to transfer the southeastern WTP values. This introduces additional uncertainty into the estimates. However, we have taken steps to adjust

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

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

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

Uncertainty bounds are provided as an alternative calculation for aggregate totals of benefits. The 5^{th} and 95^{th} percentile alternative calculations are estimated by holding air quality changes, population estimates, and other factors constant and determining the distribution of total benefits that would be generated by a large number of random draws from the distributions of C-R functions and economic valuation functions. These alternative calculations thus show how the primary estimate of benefits changes in response to uncertainty in the measurement of C-R and valuation functions.

Supplemental Calculations

Studies examining the relationship between short-term exposures and premature mortality can reveal what proportion of premature mortality is due to immediate response to daily variations in PM. There is only one short-term study (presenting results from 6 separate U.S. cities) that uses $PM_{2.5}$ as the metric of PM (Schwartz et al., 1996). As such, the supplemental estimate for premature mortality related to short-term PM exposures is based on the pooled city-specific, short-term $PM_{2.5}$ results from Schwartz et al.

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

In previous regulatory analyses, estimated incidences of ozone-related premature mortality have been estimated as a primary endpoint. However, based on recent advice from the EPA Science Advisory Board (1999e, p. 6), we have converted this endpoint to a supplemental estimate to avoid potential double-counting of benefits captured by the Pope et al. PM premature mortality endpoint. There are many studies of the relationship between ambient ozone levels and daily mortality levels. However, we chose four U.S. studies because we assume that demographic and environmental conditions on average would be more similar between these studies and the conditions prevailing when this regulation is implemented. The four

studies are by Ito and Thurston (1996), Kinney et al. (1995), Moolgavkar et al. (1995), and Samet et al. (1997).

In perhaps one of the most detailed studies to date, Samet et al.(2000b) recently examined data from 20 U.S. cities. They reported that ozone exposure during summer months may lead to premature mortality, after controlling for PM_{10} exposure. They did not find a significant effect when examining the full year, perhaps because ozone levels are higher during summer months and there is a threshold in the ozone-mortality relationship. We did not attempt to include this study in our analysis because it was published after we completed our calculations.

Exhibit 3-4 Alternative and Supplemental Benefits Calculations for the HD Engine/Diesel Fuel Rule 2030 Control Scenario

Alternative/Supplemental Calculations	Description
Alternative Calculations	
PM-related premature mortality	A number of studies provide an alternative estimate of the relationship between chronic PM exposure and mortality.
Value of avoided premature mortality incidences based on statistical life years	Calculate the incremental number of life-years lost from exposure to changes in ambient PM and use the value of a statistical life year based on a $5.9 million value of a statistical life.
Age-based adjustments to the value of a statistical life lost	Results of the Jones-Lee et al. (1985; 1989; 1993) analysis were used to calculate age-based adjustment factors to adjust the value of a statistical life lost by an individual of about age 40 to age-specific values.
Chronic asthma	Avoided incidences of chronic asthma are estimated using the McDonnell et al. (1999) C-R function.
Reversals in chronic bronchitis treated as lowest severity cases	Instead of omitting those cases of chronic bronchitis that reverse after a period of time, they are treated as being cases with the lowest severity rating.
COPD and pneumonia hospital admissions	Hospital admissions for pneumonia and COPD estimated using the Moolgavkar et al. (1997) C-R function instead of the Samet et al. (2000a) pooled C-R function.
Value of avoided asthma attacks	Due to uncertainty regarding the number of avoided asthma attacks, we present the value of avoided asthma attacks separately.
Value of visibility changes in all Class I areas	Values of visibility changes at Class I areas in California, the Southwest, and the Southeast are transferred to visibility changes in Class I areas in other regions of the country.
Value of visibility changes in Eastern U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Eastern U.S. based on the reported values for Chicago and Atlanta derived from McClelland et al. (1991).
Value of visibility changes in Western U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Western U.S. based on the reported values for Chicago and Atlanta from McClelland et al. (1990).
Household soiling damage	Value of decreases in expenditures on cleaning are estimated using values derived from Manuel et al. (1982).
Uncertainty bounds of aggregate benefit totals	5^{th} and 95^{th} percentile values of the distribution of total estimated benefits for ozone, PM, and ozone + PM.
Supplemental Calculations	
Short-term mortality	The Schwartz et al. (1996) study provides an estimate of the relationship between acute PM exposure and mortality.
Post-neonatal mortality	The Woodruff et al. (1997) study provides an estimate of the relationship between chronic exposure and infant mortality.
Ozone mortality	Ozone-related mortality benefits estimated using a pooled analysis based on four U.S. studies.
Any-of-19 respiratory symptoms	Due to the potential for overlap with health effects covered in the estimate of MRADs and both PM- and ozone-related asthma attacks, we present Any-of-19 Respiratory Symptoms separately.
Moderate or worse asthma	Due to the potential for overlap with health effects covered in the estimate of MRADs and PM-related asthma attacks, we present cases of moderate or worse asthma separately.
Shortness of breath	Due to the potential for overlap with health effects covered in the estimate of MRADs and PM-related asthma attacks, we present cases of shortness of breath separately.

Due to the potential for overlap with health effects covered in the pooled estimate of MRADs and both PM- and ozone-related asthma attacks(Whittemore and Korn, 1980), cases of Any-of-19 Respiratory Symptoms (Krupnick et al., 1990), cases of PM-related moderate or worse asthma (Ostro et al., 1991), and cases of PM-related shortness of breath (Ostro et al., 1995) are presented separately as supplemental calculations. To include them would lead to a potential double-counting of benefits related to the avoidance of asthma-related health effects.

3.3.2 Sensitivity Analyses

In addition to alternative calculations and supplementary calculations, we will perform sensitivity analyses, briefly described in Exhibit 3-5. Sensitivity analyses, as opposed to alternative calculations, examine the sensitivity of estimated benefits results to less plausible alternatives to the assumptions used in the primary analysis. Sensitivity calculations also demonstrate the sensitivity of our benefits results to key analytical parameters. The sensitivity analyses calculated for this analysis will include the impact of a threshold assumption on the Krewski et al. (2000) mortality function, and alternative lag structures when valuing mortality. Results from the sensitivity analyses are presented in Appendix A.

Exhibit 3-5 Sensitivity Analyses for the HD Engine/Diesel Fuel Rule 2030 Control Scenario

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

3.3.3 Statistical Uncertainty Bounds

Although there are several sources of uncertainty affecting estimates of endpoint-specific benefits, the sources of uncertainty that are most readily quantifiable in this analysis are the C-R relationships and uncertainty about unit dollar values. The total dollar benefit associated with a given endpoint depends on how much the endpoint will change due to the final standard (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth).[21] Based on these distributions, we provide estimates of the 5^{th} and 95^{th} percentile values of the distribution of estimated benefits. However, we hasten to add that this omits important sources of uncertainty, such as the contribution of air quality changes, baseline population incidences, projected populations exposed, transferability of the C-R function to diverse locations, and uncertainty about premature mortality. Thus, a confidence interval based on the standard error would provide a misleading

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

picture about the overall uncertainty in the estimates. The empirical evidence about uncertainty is presented where it is available.

Both the uncertainty about the incidence changes and uncertainty about unit dollar values can be characterized by *distributions*. Each "uncertainty distribution" characterizes our beliefs about what the true value of an unknown (e.g., the true change in incidence of a given health effect) is likely to be, based on the available information from relevant studies.[22] Unlike a sampling distribution (which describes the possible values that an *estimator* of an unknown value might take on), this uncertainty distribution describes our beliefs about what values the unknown value itself might be. Such uncertainty distributions can be constructed for each underlying unknown (such as a particular pollutant coefficient for a particular location) or for a function of several underlying unknowns (such as the total dollar benefit of a regulation). In either case, an uncertainty distribution is a characterization of our beliefs about what the unknown (or the function of unknowns) is likely to be, based on all the available relevant information. Uncertainty statements based on such distributions are typically expressed as 90 percent credible intervals. This is the interval from the fifth percentile point of the uncertainty distribution to the ninety-fifth percentile point. The 90 percent credible interval is a "credible range" within which, according to the available information (embodied in the uncertainty distribution of possible values), we believe the true value to lie with 90 percent probability.

The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from these two sources, and is estimated with a Monte Carlo method. In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution and a value is randomly drawn from the unit dollar value distribution, and the total dollar benefit for that iteration is the product of the two.[23] If this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated.

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

3.3.4 Unquantified Benefits

In considering the monetized benefits estimates, the reader should remain aware of the limitations. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the PM and ozone-induced adverse effects. For many health and welfare effects, such as PM-related materials damage, reliable C-R functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefits categories, the benefits estimates presented in this RIA would increase. In addition to unquantified benefits, there may also be environmental costs that we are unable to quantify. Several of these environmental cost categories are related to nitrogen deposition, while one

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

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

category is related to the issue of ultraviolet light. The net effect of excluding benefit and disbenefit categories from the estimate of total benefits depends on the relative magnitude of the effects.

4 Health Benefits

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

We estimate the incidence of adverse health effects using C-R functions based on PM and ozone. The changes in incidence of PM-related and ozone-related adverse health effects and corresponding monetized benefits associated with these changes are estimated separately. The PM- and ozone-related health endpoints for which C-R functions are estimated are shown in Exhibits 4-1 and 4-2, respectively. The unit monetary values for each of these endpoints, and associated uncertainty distributions, are presented in Exhibit 4-3. As we discuss in the appropriate sections, we needed to assume the shape of the distribution for some of the endpoints, such as the value of MRADs.

Note also, that in some cases there are alternative and/or supplemental endpoints, studies, or unit dollar values that could be used in calculating the benefits of a change in pollution. These alternatives are presented where appropriate in Exhibits 4-1, 4-2, and 4-3 in italics to indicate that they are not used in the primary analysis but may be used in alternative analyses or used to supplement the existing analyses. Appendices B and C present the functional forms for each C-R function and how they were derived.

Issues relating to the calculation of changes in incidence and the monetization of these changes are discussed below for each endpoint. For some of the endpoint-pollutant combinations, there are several epidemiological studies that have estimated C-R functions. In these cases, the information in the multiple studies is pooled, so that the estimation of the change in incidence and the corresponding monetized value of that change is based on a synthesis of the information in all the available studies. A general discussion of pooling issues is provided above. A detailed description of the method used to pool multiple studies in this analysis is given below for those endpoints for which pooling was used.

Exhibit 4-1 PM-Related Health Endpoints

Endpoint	Population	PM	Study
Mortality			
Associated with long-term exposure	Ages 30+	$PM_{2.5}$	Krewski et al. (2000), reanalysis of Pope et al. (1995) using the annual mean and all-cause mortality
Associated with long-term exposure [a]	*Ages 30+*	*$PM_{2.5}$*	*Krewski et al. (2000), reanalysis of Pope et al. (1995) using the annual median*
Associated with long-term exposure	*Ages 30+*	*$PM_{2.5}$*	*Krewski et al. (2000), reanalysis of Pope et al. (1995) using the annual median, random effects, independent cities*
Associated with long-term exposure	*Ages 30+*	*$PM_{2.5}$*	*Krewski et al. (2000), reanalysis of Pope et al. (1995) using the annual median, random effects, regional adjustment*
Associated with long-term exposure	*Ages 30+*	*$PM_{2.5}$*	*Krewski et al. (2000), reanalysis of Dockery et al. (1993)*
Associated with long-term exposure	*Ages 30+*	*$PM_{2.5}$*	*Pope et al. (1995)*
Associated with long-term exposure	*Ages 27+*	*$PM_{2.5}$*	*Dockery et al. (1993)*
Associated with long-term exposure	*1-12 Months*	*PM_{10}*	*Woodruff et al. (1997)*
Associated with short-term exposure	*All ages*	*$PM_{2.5}$*	*Schwartz et al. (1996)*
Chronic Illness			
Chronic Bronchitis	varies by study	varies by study	Two studies [b]
Hospital Admissions			
COPD (ICD-9 codes 4490-492, 494-496)	age 65+	PM_{10}	Samet et al. (2000a)[c]
Pneumonia (ICD-9 codes 480-487)	age 65+	PM_{10}	Samet et al. (2000a)[c]
Cardiovascular (ICD-9 codes 390-429)	age 65+	PM_{10}	Samet et al. (2000a)[c]
Asthma (ICD code 493)	< 65	$PM_{2.5}$	Sheppard et al. (1999)
Asthma-related ER visits	< 65	PM_{10}	Schwartz et al. (1993)
COPD (ICD-9 codes 490-496)	*>64*	*PM_{10}*	*Moolgavkar et al. (1997)*
Pneumonia (ICD-9 codes 480-487)	*>64*	*PM_{10}*	*Moolgavkar et al. (1997)*
Respiratory Symptoms/Illnesses Not Requiring Hospitalization			
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1989)
Lower respiratory symptoms (LRS)	Ages 7-14	$PM_{2.5}$	Schwartz et al. (1994)
Upper respiratory symptoms (URS)	Asthmatics, ages 9-11	PM_{10}	Pope et al. (1991)
Minor restricted activity day (MRAD) (adjusted for asthma attacks)	Ages 18-65	$PM_{2.5}$ (estimated)	Ostro and Rothschild (1989),
Work loss days (WLDs)	Ages 18-65	$PM_{2.5}$	Ostro (1987)
Asthma Attacks	Asthmatics, all ages	PM_{10}	Whittemore and Korn (1980)
Any of 19 respiratory symptoms	*Ages 18-65*	*PM_{10}*	*Krupnick et al. (1990)*

Exhibit 4-1 PM-Related Health Endpoints (cont.)

Endpoint	Population	PM	Study
Moderate or worse asthma status	*Asthmatics, all ages*	*$PM_{2.5}$*	*Ostro et al. (1991),*
Shortness of breath (days with)	*African-American asthmatics, ages 7-12*	*PM_{10}*	*Ostro et al. (1995)*

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

[b] The incidence changes, and the associated monetized benefits, predicted by two studies are pooled. The separate studies and the method of pooling are described below.

[c] The pooled estimate, based on distributed lag models in each of 14 cities, is used because the estimated coefficients based on pooling are substantially more stable than the individual city-specific estimates.

Exhibit 4-2 Ozone-Related Health Endpoints

Endpoint	Population to Which Applied	Study
Chronic Illness		
Chronic asthma[a]	*non-asthmatic males, age 27+*	*McDonnell et al. (1999)*
Hospital Admissions		
Respiratory	varies by study	Multiple studies [b]
Cardiovascular: Dysrhythmias		Burnett et al. (1999)
Asthma-related ER visits	varies by study	Multiple studies [b]
Symptoms/Illnesses Not Requiring Hospitalization		
Minor restricted activity day (MRAD) (adjusted for asthma attacks)	Ages 18-65	Ostro and Rothschild (1989)
Worker productivity	Working population	Crocker and Horst (1981) and EPA (1994)
Asthma attacks [c]	Asthmatics, all ages	Whittemore and Korn (1980)
Any of 19 respiratory symptoms	*Ages 18-65*	*Krupnick et al. (1990)*

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

[b] The incidence changes, and the associated monetized benefits, predicted by several studies are pooled. The separate studies and the method of pooling are described below.

[c] We include the number of avoided asthma attacks in the primary analysis. However, we present the value of these avoided attacks as an alternative calculation.

Exhibit 4-3 Unit Values for Economic Valuation of Health Endpoints (1999 $)

Health Endpoint	Mean Estimate [a]	Assumed Uncertainty Distribution [a]
Mortality		
Value of a statistical life	$6.12 million per statistical life	Weibull distribution, mean = $6.12 million; std. dev. = $4.13 million.
Value of a statistical life year [b]	*$2.8 million per statistical life (mean of 14 years of life saved)*	*Based on the Weibull distribution for the value of a statistical life, from which the value of a statistical life year is derived.*
Chronic Bronchitis		
WTP approach	$331,000 per case	A Monte Carlo-generated distribution, based on three underlying distributions.
Chronic Asthma		
WTP approach	*$31,875 per case*	*Triangular distribution centered at $31,875 over the interval [$24,225, $38,250].*
Hospital Admissions		
Pneumonia (ICD codes 480-487)	— [d]	None available [c]
COPD (ICD codes 490-492, 494-496)	— [d]	None available [c]
Respiratory	— [d]	None available [c]
Cardiovascular	— [d]	None available [c]
Asthma-related ER visits	$298.62 per visit	Triangular distribution centered at $299 over the interval [$221.65, $414.07].
Respiratory Ailments Not Requiring Hospitalization		
Acute bronchitis	$57.34 per case	Continuous uniform distribution over [$16.57, $98.15].
Lower resp. Symptoms	$15.30 per symptom-day	Continuous uniform distribution over [$6.37, $24.22].
Upper resp. Symptoms	$24.22 per symptom-day	Continuous uniform distribution over [$8.93,$42.06].
Minor restricted activity day (MRAD)	$48.43 per day	Triangular distribution centered at $48.43 over [$20.34, $77.76].
Shortness of breath	$6.76 per symptom-day	Continuous uniform distribution over [$0, $13.51]
Work loss days	$106 per day	None available
Worker productivity	Change in daily wages adjusted by regional variations in income	None available
Asthma attacks	*$40.79 per symptom-day*	*Continuous uniform distribution over [$15.30, $68.83]*
Asthma – moderate or worse	*$40.79 per day of that asthma status*	*Continuous uniform distribution over [$15.30, $68.83]*
Any of 19 acute respiratory symptoms	*$22.95 per symptom-day*	*Continuous uniform distribution over the interval [$0,$47.17].*
Restricted activity day (RAD)	*Based on MRAD valuation*	*Values based on MRAD valuation*

[a] The derivation of each of the estimates is discussed in the text. All WTP-based dollar values were obtained by multiplying rounded 1990 $ values used in the §812 Prospective Analysis by 1.275 to adjust to 1999 $. Entries in italics are not used in the primary benefits analysis.

[b] Based on a 3 percent discount rate, a value of $284,325 per life year (in 1999 $), a five-year lag structure, 1997 life expectancies, and 8,105 implied deaths (derived from the number of estimated life years lost). This is explained in greater detail in the text below.

Exhibit 4-3 Unit Values for Economic Valuation of Health Endpoints (1999 $) (cont.)

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

[d] Definitions of endpoints vary by study. For example, "all respiratory illnesses" includes ICD-9 codes 460-519 in some studies, but only subsets of that group in other studies. Cost of illness unit dollar values were derived for each separate set of ICD codes for which a C-R model was estimated. These are given below.

4.1 Premature Mortality

The effects of changes in PM concentrations on mortality may be estimated by a count of the expected number of deaths avoided due to a given reduction in PM concentrations. An alternative measure is to infer the number of years of life that are saved by a given reduction in PM concentrations: years of life that each individual was expected to live and that would have been lost had the reduction in PM concentrations not occurred. Both measures of mortality are estimated in this analysis to provide a range of the possible cost of premature mortality.

Both ozone and particulate matter have been associated with increased risk of premature mortality, which is a very important health endpoint in this economic analysis due to the high monetary value associated with risks to life. There are two types of exposure to elevated levels of air pollution that may result in premature mortality. Acute (short-term) exposure (e.g., exposure on a given day) to peak pollutant concentrations may result in excess mortality on the same day or within a few days of the elevated exposure. Chronic (long-term) exposure (e.g., exposure over a period of a year or more) to levels of pollution that are generally higher may result in mortality in excess of what it would be if pollution levels were generally lower. The excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels.

4.1.1 Short-Term Versus Long-Term Studies

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

4.1.2 Degree of Prematurity of Mortality

It is possible that the short-term studies are detecting an association between PM and mortality that is primarily occurring among terminally ill people. Critics of the use of short-term studies for policy analysis purposes correctly point out that an added risk factor that results in terminally ill people dying a few days or weeks earlier than they otherwise would have (referred to as "short-term harvesting") is potentially included in the measured PM mortality "signal" detected in such a study. While some of the detected excess deaths may have resulted in a substantial reduction in lifespan, others may have resulted in a relatively small decrease in lifespan. Studies by Spix et al (1993) and Pope et al. (1992) yield conflicting evidence, suggesting that harvesting may represent anywhere from zero to 50 percent of the deaths estimated in short-term studies. However, recent work by Zeger et al. (1999) and Schwartz (2000a) that focused exclusively on this issue, reported that short-term harvesting does not play a major role in the PM-mortality relationship.[24]

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

4.1.3 Estimating PM-Related Premature Mortality

The benefits analysis estimates $PM_{2.5}$ -related mortality using the C-R function estimated by Krewski et al. (2000). This study is a reanalysis of Pope et al. (1995), which estimated the association between long-term (chronic) exposure to $PM_{2.5}$ and the survival of members of a large study population. Our decision to use Pope et al. in previous benefits analyses reflected the Science Advisory Board's explicit recommendation for modeling the mortality effects of PM in both the§812 Retrospective Report to Congress and the §812 Prospective study (U.S. EPA, 1999a, p. 12). An advantage of Krewski et al. over Pope et al. is that Krewski et al.'s reanalysis uses the annual mean $PM_{2.5}$ concentration rather than the annual median. Because the mean is more readily affected by high PM values than is the median, if high PM days are actually important in causing premature mortality, the annual mean may be a preferable measure of long-term exposure than the median.

The Krewski et al. (2000) long-term study is selected for use in the benefits analysis instead of short-term (daily pollution) studies for a number of reasons. It is used alone– rather than considering the total effect to be the sum of estimated short-term and long-term effects– because summing creates the possibility of double-counting a portion of PM-related mortality. The Krewski et al. study and the Pope study it reanalyzes are considered preferable to other available long-term studies because they use better statistical methods, have a much larger sample size, the longest exposure interval, and more locations (51 cities) in the United States, than other studies. The Krewski study is considered preferable to the original Pope et al. (1995) study it reanalyzes because it uses the annual mean PM rather than the median.

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

It is unlikely that the Krewski et al. (2000) study contains any significant amount of short-term harvesting. First, the health status of each individual tracked in the study is known at the beginning of the study period. Persons with known pre-existing serious illnesses were excluded from the study population. Second, the statistical model used in the Krewski et al. and Pope et al. (1995) studies examines the question of survivability throughout the study period (ten years). Deaths that are premature by only a few days or weeks within the ten-year study period (for example, the deaths of terminally ill patients, triggered by a short duration PM episode) are likely to have little impact on the calculation of the average probability of surviving the entire ten year interval. In relation to the "Six-cities" study by Dockery et al. (1993), both the Krewski et al. study and the Pope et al. studies found smaller increases in excess mortality for a given PM air quality change.

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

Alternative Calculations: PM-Related Premature Mortality

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

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

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

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

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

Sensitivity Calculation: Mortality Lag Structure

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

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

Sensitivity Calculation: Threshold Analysis

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

Exhibit 4-4 Mortality Lag Structure

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

Supplemental Calculation: Ozone-Related Mortality

Epidemiological studies suggest that there may be a link between ozone exposures and premature mortality, however possible confounding with PM-related mortality precludes its inclusion in the primary analysis. As an alternative, an ozone-related mortality meta-analysis was conducted to provide an alternative calculation of mortality incidence. Using a random-effects pooling procedure, we take the incidence estimates of four U.S. ozone-related mortality studies -- Ito and Thurston (1996), Kinney et al. (1995), Moolgavkar et al. (1995), and Samet et al. (1997) -- and estimate the mortality incidence changes for a given rule. For a complete discussion of ozone mortality and the pooling procedure, see the Technical Support Document for the proposed Tier II rule (Abt Associates Inc., 1999b).

Supplemental Calculation: Short-Term Exposure Mortality

Schwartz et al. (1996) estimated a relationship between daily $PM_{2.5}$ and daily mortality in six U.S. cities. As noted above, however, because a long-term exposure study may detect some of the same short-term exposure effects detected by short-term studies, including both types of study in a benefit analysis would likely result in some degree of double counting of benefits. We therefore pooled the six city-specific results from Schwartz et al., which are presented as a supplemental calculation.

Supplemental Calculation: Neonatal Mortality

Woodruff et al. (1997) associated changes in annual PM_{10} levels with changes in post-neonatal mortality of infants aged 28 to 364 days. Conceptually, any additional mortality from this function could be added to the premature mortality predicted by Krewski et al. (2000), because the Krewski et al.

reanalysis of the Pope et al. (1995) function covers only the population over 29 years old. The EPA Clean Air Council, in advice issued during the §812 Prospective Analysis, recommended that this endpoint not be included because it was a new endpoint that had not been replicated in other studies in the U.S. (U.S. EPA, 1999a, p. 12). The Council deemed that the coherence and consistency arguments which support the use of other studies are not present with this study. Instead, results for this endpoint are presented as a supplemental calculation to the primary analysis.

4.1.4 Valuing Premature Mortality

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

Statistical Lives Lost

The estimated value of a "statistical life lost" is an intermediate value from a variety of estimates in the economics literature, and is a value that EPA has frequently used in RIAs for other rules. This estimate is the mean of a distribution fitted to the estimates from 26 value-of-life studies identified in the §812 study as "applicable to policy analysis." The approach and set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria used by Viscusi in his review of value-of-life studies. The estimate is consistent with Viscusi's conclusion (updated to 1999 $) that "most of the reasonable estimates of the value of life are clustered in the $3.8 to $8.9 million range." Uncertainty associated with the valuation of premature mortality avoided is expressed through a Weibull distribution (see Exhibit 4-3) (IEc 1992, p. 2).

Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. The 26 studies are listed in Exhibit 4-5. The references for all but Gegax et al. (1985) and V.K. Smith (1983) may be found in Viscusi (1992). Although each of the studies estimated the mean WTP (MWTP) for a given reduction in mortality risk, the amounts of reduction in risk being valued were not necessarily the same across studies, nor were they necessarily the same as the amounts of reduction in mortality risk that would actually be conferred by a given reduction in ambient pollutant concentrations.

The transferability of estimates of the value of a statistical life from the 26 studies to this analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction, or equivalently, that the marginal willingness to pay curve is horizontal within a reasonable range. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is $30. Suppose, however, that the actual mortality risk reduction resulting from a given air quality improvement is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a

WTP of $30 for a reduction of 1/100,000 implies a WTP of $300 for a risk reduction of 1/10,000 (which is ten times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the value of a statistical life does not depend on the particular amount of risk reduction being valued.

Exhibit 4-5 Summary of Mortality Valuation Estimates

Study	Type of Estimate	Valuation (millions 1999 $)
Kneisner and Leeth (1991) (US)	Labor Market	0.7
Smith and Gilbert (1984)	Labor Market	0.9
Dillingham (1985)	Labor Market	1.1
Butler (1983)	Labor Market	1.5
Miller and Guria (1991)	Contingent Valuation	1.6
Moore and Viscusi (1988)	Labor Market	3.2
Viscusi et al. (1991)	Contingent Valuation	3.4
Gegax et al. (1985; 1991)	Contingent Valuation	4.3
Marin and Psacharopoulos (1982)	Labor Market	3.5
Kneisner and Leeth (1991) (Australia)	Labor Market	4.3
Gerking et al. (1988)	Contingent Valuation	4.4
Cousineau et al. (1988; 1992)	Labor Market	4.6
Jones-Lee (1989)	Contingent Valuation	4.9
Dillingham (1985)	Labor Market	5.1
Viscusi (1978; 1979)	Labor Market	5.2
R.S. Smith (1976)	Labor Market	5.8
V.K. Smith (1983)	Labor Market	6.0
Olson (1981)	Labor Market	6.6
Viscusi (1981)	Labor Market	8.3
R.S. Smith (1974)	Labor Market	9.1
Moore and Viscusi (1988)	Labor Market	9.3
Kneisner and Leeth (1991) (Japan)	Labor Market	9.7
Herzog and Schlottman (1987; 1990)	Labor Market	11.6
Leigh and Folson (1984)	Labor Market	12.4
Leigh (1987)	Labor Market	13.3
Garen (1988)	Labor Market	17.2

Source: Viscusi (1992, Table 4.1).

Although the particular amount of mortality risk reduction being valued in a study may not affect the transferability of the WTP estimate from the study to this analysis, the characteristics of the study subjects and the nature of the mortality risk being valued in the study could be important. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the MWTP to reduce the risk. The appropriateness of the MWTP estimates from the 26 studies for

valuing the mortality-related benefits of reductions in ambient air concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on (1) the extent to which the subjects in the studies are similar to the population affected by changes in ambient air concentrations and (2) the extent to which the risks being valued are similar.

Focusing on the wage-risk studies, which make up the substantial majority of the 26 studies relied upon, the likely differences between (1) the subjects in these studies and the population affected by changes in air concentrations and (2) the nature of the mortality risks being valued in these studies and the nature of air pollution-related mortality risk are considered. The direction of bias in which each difference is likely to result is also considered.

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

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

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

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

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

There is substantial evidence that the income elasticity of WTP for health risk reductions is positive (Loehman and De, 1982; Jones-Lee et al., 1985; Mitchell and Carson, 1986; Gerking et al., 1988; Alberini et al., 1997). However, there is uncertainty about the exact value of this elasticity. Individuals

with higher incomes (or greater wealth) should, then, be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. Whether the average income or level of wealth of the population affected by ambient air pollution reductions is likely to be significantly different from that of subjects in wage-risk studies, however, is unclear.

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

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

The potential sources of bias in an estimate of MWTP to reduce the risk of air pollution related mortality based on wage-risk studies are summarized in Exhibit 4-6. Although most of the individual factors tend to have a downward bias, the overall effect of these biases is unclear.

Exhibit 4-6 Potential Sources of Bias in Estimates of Mean WTP to Reduce the Risk of PM Related Mortality Based on Wage-Risk Studies

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

Alternative Calculation: Statistical Life-Years Lost

In an alternative calculation, we value statistical life-years, rather than valuing statistical lives. Moore and Viscusi (1988) value a statistical life-year lost, by assuming that the WTP to save a statistical life is the value of a single year of life times the expected number of years of life remaining for an individual. They suggest that a typical respondent in a mortal risk study has a life expectancy of an additional 35 years. Using a mean estimate of $4.8 million (1990 $) to save a statistical life, their approach yields an estimate of $137,000 per life-year lost or saved, assuming no discounting. If an individual discounts future additional years using a standard discounting procedure, the value of each life-year lost must be greater than the value assuming no discounting. Using a 35 year life expectancy, a $6.12 million value of a statistical life, and a three percent discount rate, the implied value of each life-year lost is $284,325 in 1999 dollars.

In addition, the "statistical life-years lost" analysis must accommodate the five-year lag structure. For each person dying at a given age, using the expected number of years remaining for that age, based on

1997 life expectancy tables (National Center for Health Statistics, 1999, Table 5), and a VSLY of $284,325, we calculate the present discounted value (discounted back to the beginning of the year of death) for that person. All values are then discounted back to the beginning of 2030, whether the individual dies in 2030 or in a subsequent year. The present discounted value (discounted back to the beginning of 2030) of an avoided premature mortality will vary from one individual to another, depending on the age of the individual at death and on the extent of lag between exposure and death. The age at death determines the expected number of life-years lost, while the extent of lag between exposure and death determines the amount of discounting needed.

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

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

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

4.2 Chronic Illness

Onset of bronchitis and asthma, two chronic illnesses, have both been associated with exposure to air pollutants. Three studies have linked the onset of chronic bronchitis in adults to particulate matter; one study has linked the onset of chronic asthma in adults to ozone. These results are consistent with research

that has found chronic exposure to pollutants leads to declining pulmonary functioning (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998).

4.2.1 Chronic Bronchitis

We estimate the changes in the number of new cases of PM-related chronic bronchitis using studies by Schwartz (1993) and Abbey et al. (1995b). The Schwartz study is somewhat older and uses a cross-sectional design; however, it is based on a national sample, unlike the Abbey et al. study which is based on a sample of California residents. Our analysis pools the estimates from these studies to develop a C-R function linking PM to chronic bronchitis. The Schwartz study examined the relationship between exposure to PM_{10} and prevalence of chronic bronchitis. The Abbey et al. study examined the relationship between $PM_{2.5}$ and new incidences of chronic bronchitis. Both studies have strengths and weaknesses which suggest that pooling the effect estimates from each study may provide a better estimate of the expected change in incidences of chronic bronchitis than using either study alone.

However, the HD Engine/Diesel Fuel rule is expected to result in reductions in both the fine and coarse fractions of PM_{10}. As such, reliance on the Abbey et al. (1995b) estimate will result in an underestimate of the change in chronic bronchitis incidences if both the fine and coarse fractions of PM_{10} are associated with chronic bronchitis. To address this problem, we apply the C-R functions from both Schwartz (1993) and Abbey, et al. to generate the changes in chronic bronchitis incidences associated with the change in $PM_{2.5}$ and then pool the incidence estimates to obtain a primary estimate of avoided $PM_{2.5}$ related chronic bronchitis incidences. We then apply the Schwartz C-R function to the change in coarse PM ($PM_{2.5-10}$) to obtain a primary estimate of avoided incidences of chronic bronchitis due to the change in coarse fraction PM. The primary estimate of total avoided incidences is then the sum of the avoided incidences from changes in $PM_{2.5}$ and $PM_{2.5-10}$.[25] The two studies used in our pooled estimate are listed in Exhibit 4-7.

Exhibit 4-7 Chronic Bronchitis Studies

Location	Study	Pollutants Used in Final Model	Age of Study Population
California	Abbey et al. (1995b)	$PM_{2.5}$	>26
United States	Schwartz (1993)	PM_{10}	>29

Alternative Calculation: Chronic Bronchitis Reversals

In developing the C-R functions for chronic bronchitis, it is necessary to estimate its annual incidence rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by

[25] This assumption implies that the observed relationship between chronic bronchitis and PM_{10} in the Schwartz (1993) study is equally attributable to the fine and coarse fractions of PM_{10}. If the relationship is due primarily to the fine fraction, then the estimate of avoided incidences associated with coarse fraction PM changes will be overstated. However, if this is the case then the estimate of avoided incidences associated with fine fraction will be somewhat understated. The net effect on avoided incidences of chronic bronchitis is ambiguous.

the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate.[26] Reversals refer to those cases of chronic bronchitis that were reported at the start of the Abbey et al. survey, but were subsequently not reported at the end of the survey. Since we assume that chronic bronchitis is a permanent condition, we subtract these reversals. Nevertheless, reversals may likely represent a real effect that should be included in the analysis. To allow for this possibility, we present an estimate of reversals in an alternative calculation in which reversals are considered to be chronic bronchitis cases of the lowest severity level, as described below.

Valuing Chronic Bronchitis

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

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

The study by Viscusi et al. (1991) uses a sample that is larger and more representative of the general population than the study by Krupnick and Cropper (1992), which selects people who have a relative with the disease. Thus, the valuation for the high-end estimate is based on the distribution of WTP responses from Viscusi et al. The WTP to avoid a case of pollution-related chronic bronchitis (CB) is derived by starting with the WTP to avoid a severe case of chronic bronchitis, as described by Viscusi et al. (1991), and adjusting it downward to reflect (1) the decrease in severity of a case of pollution-related CB relative to the severe case described in the Viscusi et al. study, and (2) the elasticity of WTP with respect to severity reported in the Krupnick and Cropper study. Because elasticity is a marginal concept and because it is a function of severity (as estimated from Krupnick and Cropper), WTP adjustments were made incrementally, in one percent steps. A severe case of CB was assigned a severity level of 13 (following Krupnick and Cropper). The WTP for a one percent decrease in severity is given by:

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

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

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

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

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

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

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

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

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

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

1. The distribution of WTP to avoid a severe case of CB was based on the distribution of WTP responses in the Viscusi et al. (1991) study. Viscusi et al. derived respondents' implicit WTP to avoid a statistical case of chronic bronchitis from their WTP for a specified reduction in risk. The mean response implied a WTP of about $1,275,000 (1999 $)[27]; the median response implied a WTP of about $676,000 (1999 $). However, the extreme tails of distributions of WTP responses are usually considered unreliable. Because the mean is much more sensitive to extreme values, the median of WTP responses is often used rather than the mean. Viscusi et al. report not only the mean and median of their distribution of WTP responses, however, but the decile points as well. The distribution of reliable WTP responses from the Viscusi et al. study could therefore be approximated by a discrete uniform distribution giving a probability of 1/9 to each of the first nine decile points. This omits the first five and the last five percent of the responses (the extreme tails, considered unreliable). This trimmed distribution of WTP responses from the Viscusi et al. study was assumed to be the distribution of WTPs to avoid a severe case of CB.

[27]There is an indication in the Viscusi et al. (1991) paper that the dollar values in the paper are in 1987 dollars. Under this assumption, the dollar values were converted to 1999 dollars.

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

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

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

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

Alternative Calculation: Valuing Chronic Bronchitis Reversals

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

4.2.2 Sensitivity Calculation: Chronic Asthma

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

study by McDonnell et al. (1999) carefully measured ozone exposure over 15 years, and found ozone exposure was linked to the onset of asthma in adult males.

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

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

Valuing Chronic Asthma

Two studies have estimated WTP to avoid chronic asthma in adults. Blumenschein and Johannesson (1998) used two different contingent valuation (CV) methods, the dichotomous choice method and a bidding game, to estimate mean willingness to pay for a cure for asthma. The mean WTP elicited from the bidding game was $189 per month, or $2,268 per year (in 1996$). The mean WTP elicited from the dichotomous choice approach was $343 per month, or $4,116 per year (in 1996$). Using $2,268 per year, a five percent discount rate, and 1997 life expectancies for males in the United States (National Center for Health Statistics, 1999, Table 5), the present discounted value of the stream of annual WTPs is about $38,250 (in 1999 $).

O'Conor and Blomquist (1997) estimated WTP to avoid chronic asthma from estimates of risk-risk tradeoffs. Combining the risk-risk tradeoffs with a statistical value of life, the annual value of avoiding asthma can be derived. Assuming a value of a statistical life of $6 million, they derived an annual WTP to avoid asthma of $1500 (O'Connor and Blomquist, 1997, p. 677). For a value of a statistical life of $5,894,400 (in 1997 $), the corresponding implied annual value of avoiding chronic asthma, based on O'Conor and Blomquist would be $1,474. Assuming a five percent discount rate and 1997 life

expectancies for males in the United States, the present discounted value of the stream of annual WTPs would be about $24,225 (in 1999 $).[28]

Following the method used for the §812 Prospective analysis, the uncertainty surrounding the WTP to avoid a case of chronic asthma among adult males was characterized by a triangular distribution on the range determined by the two WTP estimates. The range used in the §812 Prospective analysis was [$24,225, $38,250], centered at $31,875 (in 1999 $). In the current analysis these dollar values are converted to 1999 $ using the CPI-U for "all items."

4.3 Hospital Admissions

We estimate the impact of ozone and PM on both respiratory and cardiovascular hospital admissions. In addition, we estimate the impact of these pollutants on emergency room visits for asthma. The respiratory and cardiovascular hospital admissions studies used in the primary analysis are listed in Exhibits 4-8 and 4-9, respectively. (Appendices B and C provide details on each study.) Although the benefits associated with respiratory and cardiovascular hospital admissions are estimated separately in the analysis, the methods used to estimate changes in incidence and to value those changes are the same for both broad categories of hospital admissions. The two categories of hospital admissions are therefore discussed together in this section.

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

Exhibit 4-8 Respiratory Hospital Admission Studies

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
PM-Related Hospital Admissions				
Fourteen U.S. Cities*	Samet et al. (2000a)	pneumonia (480-487); COPD (490-492, 494-6)	PM_{10}	>64
Seattle, WA	Sheppard et al. (1999)	asthma (493)	$PM_{2.5}$	<65
Minneapolis-St. Paul, MN	*Moolgavkar et al., (1997)*	*pneumonia (480-487); COPD (490-496)*	*O_3, PM_{10} (pneumonia); O_3, PM_{10} (COPD)*	*>64*
Ozone-Related Hospital Admissions				
Toronto, Canada	Burnett et al. (1997)	all respiratory (464-466, 480-486, 490-494, 496)	$PM_{10-2.5}$, O_3	all ages
Toronto, Canada	Burnett et al. (1999)	asthma (493); respiratory infection (464, 466, 480-487, 494); COPD (490-492, 496)	O_3, $PM_{10-2.5}$ (asthma); O_3, $PM_{2.5}$ (respiratory infection); O_3, $PM_{10-2.5}$ (COPD)	all ages
Toronto, Canada	Thurston et al. (1994)	all respiratory (466, 480-482, 485, 490-493)	O_3, $PM_{2.5}$	all ages
Minneapolis-St. Paul, MN	*Moolgavkar et al. (1997)*	*pneumonia (480-487); COPD (490-496)*	*O_3, PM_{10} (pneumonia); O_3, PM_{10} (COPD)*	*>64*
Minneapolis-St. Paul, MN	Schwartz (1994a)	pneumonia (480-486); COPD (490-496)	O_3, PM_{10} (pneumonia); PM_{10} (COPD)	>64
Detroit, MI	Schwartz (1994b)	pneumonia (480-486); non-asthma COPD (491-492, 494-496)	O_3, PM_{10}	>64
New Haven, CT	Schwartz (1995)	all respiratory (460-519)	O_3, PM_{10}	>64
Tacoma, WA	Schwartz (1995)	all respiratory (460-519)	O_3, PM_{10}	>64

*Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis/St. Paul, Minnesota; Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo/Orem, Utah; Seattle, Washington; Spokane, Washington; and Youngstown, Ohio.

Exhibit 4-9 Cardiovascular Hospital Admission Studies

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
PM-Related Hospital Admissions				
Fourteen U.S. Cities*	Samet et al. (2000a)	cardiovascular illness (390 - 429)	PM_{10}	>64
Ozone-Related Hospital Admissions				
Toronto, Canada	Burnett et al. (1999)	dysrhythmias (427);	$PM_{2.5}$, O_3	all ages

4.3.1 PM-Related Respiratory and Cardiovascular Hospital Admissions

Respiratory and cardiovascular hospital admissions are the two broad categories of hospital admissions that have been related to exposure to both PM and ozone. Several epidemiological studies have estimated C-R functions that included both PM and ozone. However, a recent study by the Health Effects Institute (HEI) (Samet et al., 2000a) estimated separate models for PM_{10} and pneumonia, COPD and cardiovascular diseases in each of fourteen cities in the United States, as well as pooled estimates across these cities. The fourteen cities included in the HEI hospital admissions study are Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis/St. Paul, Minnesota; Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo/Orem, Utah; Seattle, Washington; Spokane, Washington; and Youngstown, Ohio.

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

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

The HEI study estimates separate C-R functions for pneumonia and COPD hospital admissions for people 65 years and older. In addition, another study by Sheppard et al. (1999) estimates a C-R function for asthma hospital admissions for people under 65. The results of these three non-overlapping PM-related respiratory C-R functions are aggregated using the relevant steps of a pooling procedure described below.

Alternative Calculation: Moolgavkar et al. (1997) Pneumonia and COPD

Because the Samet et al. (2000a) study is the only one used to predict PM-related pneumonia- and COPD-related hospital admissions in the primary analysis, we present cases of pneumonia and COPD estimated by Moolgavkar et al. (1997) here as alternative calculations. In previous analyses, the study by Moolgavkar et al. was one of a number of studies pooled together to estimate PM-related respiratory hospital admission incidence. We still use the Moolgavkar et al. study in the primary ozone analysis.

4.3.2 Ozone-Related Respiratory and Cardiovascular Hospital Admissions

We estimate the impact of ozone on cardiovascular and respiratory hospital admissions. The evidence seems strongest for the link between ozone and respiratory admissions. However, as we discuss below, some evidence suggests a link between ozone and dysrhythmias admissions.

Cardiovascular Hospital Admissions

Several studies have investigated the relationship between ozone and hospital admissions for cardiovascular illnesses (Schwartz and Morris, 1995; Burnett et al., 1997; Schwartz, 1997; Burnett et al., 1999; Schwartz, 1999). However, the range of ICD codes included in these studies has varied substantially. Burnett (1997) included ICD codes 410-414 and 427-428, and reported an ozone coefficient that was one or more orders of magnitude greater than any of the other reported ozone coefficients. At the other extreme, three of the studies failed to find any ozone effect and did not include ozone in their final models (Schwartz and Morris, 1995; Schwartz, 1997; Schwartz, 1999). Two of these studies, Schwartz (1997; 1999), included a broad range of ICD codes, 390 - 429. The third included 410-414 (ischemic heart disease) and 428 (congestive heart failure). A fifth study, Burnett et al. (1999), included only ICD code 427 (dysrhythmias) and found a modest (statistically insignificant) ozone effect.

Because the result reported by Burnett et al. (1997) is so large, we believe it may be spurious and therefore did not use it. On the other hand, the lack of an ozone effect in the Schwartz studies may be the result of including too large a group of illnesses. If only one illness (e.g., dysrhythmias) is actually related to ozone, the inclusion of so many additional illnesses in the study may have simply added too much noise to detect anything. It is possible that there is a real relationship between ozone and ICD code 427, the only ICD code that was included in both studies reporting positive results. We therefore chose only the study by Burnett et al. (1999), which reported a relationship between ozone and ICD code 427 (dysrhythmias).

Respiratory Hospital Admissions

To estimate the incidence and monetary value of avoided ozone-related respiratory hospital admissions, we pool the incidences and the monetary values corresponding to the incidence estimates from a variety of U.S. and Canadian studies, using a random effects weighting procedure. These studies differ from each other in two important ways: (1) Some studies considered people of all ages while others considered only people ages 65 and older; and (2) The ICD codes included in the studies vary substantially.

The broadest classification includes ICD codes 460-519 (e.g., Schwartz 1995). Other studies, however, considered only subsets of the broader classification. For example, Schwartz (1994b) considered ICD-9 codes 480-486, 491-492, and 494-496. It is unclear what the correct set of ICD codes is. If the broadest category (460-519) is too broad, including respiratory illnesses that are not linked to air pollution, we would expect the estimated pollutant coefficients to be biased downward; however, they would be used in combination with a larger baseline incidence in estimating changes in respiratory hospital admissions associated with changes in pollutant concentrations. If the broadest category is correct (i.e., if all the respiratory illnesses included are actually associated with air pollution), then studies using only subsets of ICD codes within that category would presumably understate the change in respiratory hospital admissions. It is likely, however, that all the studies have included the most important respiratory illnesses, and that the impact of differences in the definition of “all respiratory illnesses” may be less than that of other study

design characteristics. We therefore treat each study equally, at least initially, in the pooling process, assuming that each study gives a reasonable estimate of the impact of air pollution on respiratory hospital admissions. The pooling process involves several steps, as described below.

4.3.3 Pooling the Results of More Than One Study

For ozone-related respiratory hospital admissions there is more than one relevant C-R function, some of which overlap with each other while others do not. The results of the overlapping functions should be pooled; the results of the non-overlapping functions should be aggregated. The procedure we used to pool and aggregate the information in these functions is described below.

1. For each study, develop a study-specific distribution of pollutant[29] coefficients. If separate non-overlapping sets of illnesses were considered in the study, develop a distribution for each set.

The value of the pollutant coefficient in a C-R function is estimated. Because of the statistical uncertainty surrounding the estimated coefficient, the C-R function is uncertain. We assume a normal distribution of the pollutant coefficient in the C-R function, with mean equal to the estimated coefficient reported in the study and standard deviation equal to the reported standard error of that estimate. If separate models were estimated for separate non-overlapping sets of illnesses (e.g., Moolgavkar et al. 1997) estimated separate models for pneumonia (ICD codes 480-487) and one for COPD (ICD codes 490-496), we develop a distribution of coefficients for each non-overlapping hospital admissions category.

2. For each study, develop a distribution of unit monetary values. If separate non-overlapping sets of respiratory illnesses were considered in the study, develop a distribution of unit monetary values for each set.

The monetary value of an avoided hospital admission depends on the particular type of illness (i.e., the ICD code) and the length of stay in the hospital, which itself varies with the type of admission. The monetary value of a set of hospital admissions (i.e., a set of ICD codes) is estimated as a weighted average of the individual ICD-code-specific values in the set. The valuation of hospital admissions is described more fully below.

3. For each study, develop a distribution of incidence changes and a distribution of monetary benefits resulting from a given change in pollutant concentrations.

On each iteration of a Monte Carlo procedure, for each non-overlapping hospital admissions category considered in the study,

- we randomly select a pollutant coefficient from the distribution of coefficients.
- Given the coefficient and the pollutant change, we calculate the incidence change.
- We randomly select a unit dollar value from the corresponding dollar distribution;
- The benefit is the product of the incidence change and the unit dollar value.

If the study has considered several non-overlapping hospital admissions categories, we sum the incidences and the dollar benefits across categories. For example, we estimated and summed the incidences

[29] "Pollutant" can mean either PM or ozone.

for the two separate models estimated by Moolgavkar et al. (1997). A series of many (e.g., 5000) iterations therefore produces (1) a series (distribution) of incidence changes for each non-overlapping hospital admissions category considered by the study as well as for all categories combined, and (2) a distribution of the dollar benefits associated with hospital admissions that would be predicted by the study.

4. For ozone-related respiratory hospital admissions: Pool estimates of respiratory hospital admissions changes.

The study-specific incidence estimates are then pooled using a random effects pooling procedure, as described above. The study-specific dollar benefits estimates are similarly pooled.

4.3.4 Valuing Respiratory and Cardiovascular Hospital Admissions

Society's WTP to avoid a hospital admission includes medical expenses, lost work productivity, the non-market costs of treating illness (i.e., air, water and solid waste pollution from hospitals and the pharmaceutical industry), and the pain and suffering of the affected individual as well as of that of relatives, friends, and associated caregivers.[30]

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 previous RIAs for PM and ozone, as well as the revised RIA for ozone and PM NAAQS, did adjust the COI estimate upward. Based on SAB advice, the COI values used in this benefits analysis will not be adjusted to better reflect the total WTP.

Following the method used in the §812 analysis (U.S. EPA, 1999b), ICD-code-specific COI estimates used in our analysis consist of two components: estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness).

[30] 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.

The opportunity cost of a day spent in the hospital is estimated as the value of the lost daily wage, regardless of whether or not the individual is in the workforce.

For all hospital admissions included in this analysis, estimates of hospital charges and lengths of hospital stays were based on discharge statistics provided by the Agency for Healthcare Research and Quality's Healthcare Utilization Project (2000). The total COI for an ICD-code-specific hospital stay lasting n days, then, would be estimated as the mean hospital charge plus $106*n. Most respiratory hospital admissions categories considered in epidemiological studies consisted of sets of ICD codes. The unit dollar value for the set of ICD codes was estimated as the weighted average of the ICD-code-specific mean hospital charges 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). The study-specific values for valuing respiratory and cardiovascular hospital admissions are shown in Exhibits 4-10 and 4-11, respectively.

The mean hospital charges and mean lengths of stay provided by (AHRQ 2000) are based on a very large nationally representative sample of about seven million hospital discharges, and are therefore the best estimates of mean hospital charges and mean lengths of stay available, with negligible standard errors. However, because of distortions in the market for medical services, the hospital charge may exceed "the cost of a hospital stay." We use the example of a hospital visit to illustrate the problem. Suppose a patient is admitted to the hospital to be treated for an asthma episode. The patient's stay in the hospital (including the treatments received) costs the hospital a certain amount. This is the hospital cost – i.e., the short-term expenditures of the hospital to provide the medical services that were provided to the patient during his hospital stay. The hospital then charges the payer a certain amount – the hospital charge. If the hospital wants to make a profit, is trying to cover costs that are not associated with any one particular patient admission (e.g., uninsured patient services), and/or has capital expenses (building expansion or renovation) or other long term costs, it may charge an amount that exceeds the patient-specific short term costs of providing services. The payer (e.g., the health maintenance organization or other health insurer) pays the hospital a certain amount – the payment – for the services provided to the patient. The less incentive the payer has to keep costs down, the closer the payment will be to the charge. If, however, the payer has an incentive to keep costs down, the payment may be substantially less than the charge; it may still, however, exceed the short-term cost for services to the individual patient.

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

Exhibit 4-10 Unit Values for Respiratory Hospital Admissions*

Location	Study	Endpoints Estimated (ICD code)	Age of Study Population	COI [a] (1999 $)
PM-Related Hospital Admissions				
Fourteen U.S. Cities	Samet et al. (2000a)	pneumonia (480-487)	>64	$14,693
		COPD (490-492, 494-6)		$12,378
Seattle, WA	Sheppard et al. (1999)	asthma (493)	<65	$6,633
Minneapolis-St. Paul, MN	*Moolgavkar et al. (1997)*	*pneumonia (480-487)*	*>64*	*$14,693*
		COPD (490-496)		*$12,149*
Ozone-Related Hospital Admissions				
Toronto, Canada	Burnett et al. (1997)	all respiratory (464-466, 480-486, 490-494, 496)	all ages	$11,175
Toronto, Canada	Burnett et al. (1999)	asthma (493)	all ages	$7,218
		respiratory infection (464, 466, 480-487, 494)		$12,087
		COPD (490-492, 496)		$12,159
Toronto, Canada	Thurston et al. (1994)	all respiratory (466, 480-482, 485, 490-493)	all ages	$11,046
Minneapolis-St. Paul, MN	Moolgavkar et al. (1997)	pneumonia (480-487)	>64	$14,693
		COPD (490-496)		$12,149
Minneapolis-St. Paul, MN	Schwartz (1994a)	pneumonia (480-486)	>64	$14,768
Detroit, MI	Schwartz (1994b)	pneumonia (480-486)	>64	$14,768
		non-asthma COPD (491-492, 494-496)		$12,464
New Haven, CT	Schwartz (1995)	all respiratory (460-519)	>64	$15,631
Tacoma, WA	Schwartz (1995)	all respiratory (460-519)	>64	$15,631

* 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). The monetized benefits of non-overlapping endpoints within each study were aggregated. Monetized benefits for asthma among people age <65 (Sheppard et al., 1999) were aggregated with the monetized benefits in Samet et al. (2000a) of people age >64.

Exhibit 4-11 Unit Values for Cardiovascular Hospital Admissions*

Location	Study	Endpoints Estimated (ICD code)	Age of Study Population	COI [a] (1999 $)
PM-Related Hospital Admissions				
Fourteen U.S. Cities	Samet et al. (2000a)	cardiovascular illness (390 - 429)	>64	$18,387
Ozone-Related Hospital Admissions				
Toronto, Canada	Burnett et al. (1999)	dysrhythmias (427);	all ages	$12,441

* 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).

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

Alternative Calculation: Valuing Moolgavkar et al. (1997) Pneumonia and COPD

The value of PM-related pneumonia and COPD cases estimated by Moolgavkar et al. (1997)are the same as those used to value ozone-related cases of pneumonia and COPD.

4.3.5 Asthma-Related Emergency Room (ER) Visits

We use the four C-R functions in Exhibit 4-12 to estimate the effects of exposure to PM and ozone on asthma-related ER visits. For ozone-related asthma ER visits, we use three epidemiological studies: Cody et al. (1992), Weisel et al. (1995), and Stieb et al. (1996). Working in central New Jersey, Cody et al. examined asthma-related ER visits over a 16 month period between May, 1988 and August, 1989, and found that ozone was linked to asthma-related ER visits. No significant effect was seen for PM_{10} or SO_2. Using a one-pollutant model, Weisel et al. also found ozone linked to asthma-related ER visits in an all-age 1990 population for eight New Jersey counties. Stieb et al. (1996) examined the relationship between ER visits and air pollution for persons of all ages in St. John, New Brunswick, Canada, from May through September in 1984-1992. Ozone was significantly linked to ER visits, especially when ozone levels exceeded 75 ppb. Schwartz et al. (1993) failed to find a significant relationship between asthma-related ER visits and ozone. In this study of older Seattle residents, Schwartz et al. instead found PM_{10} to be significantly related to asthma-related ER visits.

Exhibit 4-12 Asthma-Related Emergency Room Visit Studies

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

Because we are estimating ER visits as well as hospital admissions for asthma, we must avoid counting twice the ER visits for asthma that are subsequently admitted to the hospital. To avoid double-counting, the baseline incidence rate for emergency room visits is adjusted by subtracting the percentage of patients who are admitted into the hospital. Three studies provide some information on which to base this adjustment: Richards et al. (1981, p. 350) reported that 13% of children's ER visits ended up as hospital admissions; Lipfert (1993, p. 230) reported that ER visits (for all causes) are two to five times more frequent than hospital admissions; Smith et al. (1997, p. 789) reported 445,000 asthma-related hospital admissions in 1987 and 1.2 million asthma ER visits. The study by Smith et al. seems the most relevant since it is a national study and looks at all age groups.

Assuming that air-pollution related hospital admissions first pass through the ER, the reported incidence rates suggest that 37% (=445,000/1,200,000) of ER visits are subsequently admitted to the hospital, or that ER visits for asthma occur 2.7 times as frequently as hospital admissions for asthma. The baseline incidence of asthma ER visits is therefore taken to be 2.7 times the baseline incidence of hospital admissions for asthma. To avoid double-counting, however, only 63% of the resulting change in asthma ER visits associated with a given change in pollutant concentrations is counted in the ER visit incidence change.

Valuing Asthma-Related Emergency Room (ER) Visits

The value of an avoided asthma-related ER visit was based on national data reported in Smith et al. (1997). Smith et al. 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 $298.62 in 1999 $ (using the CPI-U for medical care to adjust to 1999 $). The uncertainty surrounding this estimate, based on the uncertainty surrounding the number of ER visits and the total cost of all visits reported by Smith et al. was characterized by a triangular distribution centered at $299, on the interval [$221.65, $414.07].

4.4 Acute Illnesses and Symptoms Not Requiring Hospitalization

We consider in this section a number of acute symptoms that do not require hospitalization, such as acute bronchitis, and upper and lower respiratory symptoms. Several of these illnesses and symptoms were considered in the §812 Prospective analysis as well. The unit values and the uncertainty distributions for those acute illnesses and symptoms that were also considered in the §812 Prospective analysis were obtained by adjusting the unit values used in that analysis from 1990 $ to 1999 $ by multiplying by 1.275 (based on the CPI-U for “all items”).

Exhibit 4-13 Studies of Symptoms/Illnesses Not Requiring Hospitalization

Endpoint [a]	Study	Pollutants	Study Population
Acute bronchitis	Dockery et al. (1996)	$PM_{2.5}$	Ages 8-12
Upper respiratory symptoms (URS)	Pope et al. (1991)	PM_{10}	Asthmatics, ages 9-11
Lower respiratory symptoms (LRS)	Schwartz et al. (1994)	$PM_{2.5}$	Ages 7-14
Minor restricted activity day (MRAD)	Ostro and Rothschild (1989),	$PM_{2.5}$ (estimated), O_3	Ages 18-65
Asthma Attacks [b]	Whittemore and Korn (1980)	PM_{10}, O_3	asthmatics, all ages
Work loss days (WLDs)	Ostro (1987)	$PM_{2.5}$	Ages 18-65
Worker productivity	Crocker and Horst (1981) and EPA (1994)	O_3	Working population
Any of 19 respiratory symptoms	*Krupnick et al. (1990)*	*PM_{10}, O_3*	*Ages 18-65*
Moderate or worse asthma status	*Ostro et al. (1991),*	*$PM_{2.5}$*	*asthmatics, all ages*
Shortness of breath (days with)	Ostro et al. (1995)	PM_{10}	African-American asthmatics, ages 7-12

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

[b] Note that we present the number of avoided asthma attacks in the primary analysis. However, we present the *value* of these avoided asthma attacks as an alternative calculation.

4.4.1 Acute Bronchitis

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

Valuing Acute Bronchitis

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

With these caveats in mind, the values used for acute bronchitis in this analysis were obtained by adjusting the values used in the §812 Prospective analysis from 1990 $ to 1999 $ by multiplying by 1.275. WTP to avoid a case of acute bronchitis was estimated as the midpoint between a low estimate and a high estimate. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day. The unit value is the midpoint between the low and high estimates. The low, high, and midpoint estimates used in the §812 Prospective analysis were $13, $77, and $45, respectively, in 1990 $. The corresponding values in 1999 $ are $16.58, $98.18, and $57.38, respectively.

4.4.2 Upper Respiratory Symptoms (URS)

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

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

Valuing URS

Willingness to pay to avoid a day of URS is based on symptom-specific WTPs to avoid those symptoms identified by Pope et al. as part of the URS complex of symptoms. Three contingent valuation (CV) studies have estimated WTP to avoid various morbidity symptoms that are either within the URS symptom complex defined by Pope et al. (1991) or are similar to those symptoms identified by Pope et al. In each CV study, participants were asked their WTP to avoid a day of each of several symptoms. The WTP estimates corresponding to the morbidity symptoms valued in each study are presented in Exhibit 4-14. The three individual symptoms listed in Exhibit 4-14 that were identified as most closely matching those listed by Pope, et al. for URS are cough, head/sinus congestion, and eye irritation, corresponding to "wet cough," "runny or stuffy nose," and "burning, aching or red eyes," respectively. A day of URS could consist of any one of the seven possible "symptom complexes" consisting of at least one of these three symptoms. These seven possible symptom complexes are presented in Exhibit 4-15. It is assumed that each of these seven URS complexes is equally likely.[31] The point estimate of MWTP to avoid an

[31] 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.

occurrence of URS is just an average of the seven estimates of MWTP for the different URS complexes. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, an uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution of MWTPs in Exhibit 4-15.

Exhibit 4-14 Median WTP Estimates and Derived Midrange Estimates (in 1999 $)

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

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

[b] 10% trimmed mean.

Exhibit 4-15 Estimates of MWTP to Avoid Upper Respiratory Symptoms (1999 $)

Symptom Combinations Identified as URS by Pope et al. (1991)	MWTP to Avoid Symptom(s)
Coughing	$8.93
Head/Sinus Congestion	$12.75
Eye Irritation	$20.03
Coughing, Head/Sinus Congestion	$21.67
Coughing, Eye Irritation	$28.96
Head/Sinus Congestion, Eye Irritation	$32.78
Coughing, Head/Sinus Congestion, Eye Irritation	$41.71
	Average: $23.83

Based on values reported in Exhibit 4-14.

It is worth emphasizing that what is being valued here is URS *as defined by Pope et al. (1991).* While other definitions of URS are certainly possible, this definition of URS is used in this benefit analysis because it is the incidence of this specific definition of URS that has been related to PM exposure by Pope et al.

4.4.3 Lower Respiratory Symptoms (LRS)

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

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+.

Valuing LRS

The method for deriving a point estimate of mean WTP to avoid a day of LRS is the same as for URS. Schwartz et al. (1994, p. 1235) define LRS as at least two of the following symptoms: cough, chest pain, phlegm, and wheeze. The symptoms for which WTP estimates are available that reasonably match those listed by Schwartz et al. for LRS are cough (C), chest tightness (CT), coughing up phlegm (CP), and wheeze (W). A day of LRS, as defined by Schwartz et al., could consist of any one of the 11 combinations of at least two of these four symptoms, as displayed in Exhibit 4-16.[32]

[32] Because cough is a symptom in some of the URS clusters as well as some of the LRS clusters, there is the possibility of a very small amount of double counting – if the same individual were to have an occurrence of URS which included cough and an occurrence of LRS 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 URS is applied only to asthmatics ages 9-11 (a very small population), the amount of potential double counting should be truly negligible.

Exhibit 4-16 Estimates of MWTP to Avoid Lower Respiratory Symptoms (1999 $)

Symptom Combinations Identified as LRS by Schwartz et al. (1994)	MWTP to Avoid Symptom(s)
Coughing, Chest Tightness	$16.95
Coughing, Coughing Up Phlegm	$12.42
Coughing, Wheeze	$12.13
Chest Tightness, Coughing Up Phlegm	$11.53
Chest Tightness, Wheeze	$11.24
Coughing Up Phlegm, Wheeze	$6.72
Coughing, Chest Tightness, Coughing Up Phlegm	$20.46
Coughing, Chest Tightness, Wheeze	$20.17
Coughing, Coughing Up Phlegm, Wheeze	$15.64
Chest Tightness, Coughing Up Phlegm, Wheeze	$14.75
Coughing, Chest Tightness, Coughing Up Phlegm, Wheeze	$23.67
	Average: $15.06

Based on values reported in Exhibit 4-14.

We assumed that each of the eleven types of LRS is equally likely.[33] The mean WTP to avoid a day of LRS as defined by Schwartz et al. (1994) is therefore the average of the mean WTPs to avoid each type of LRS. This is the point estimate used in the benefit analysis for the dollar value for LRS as defined by Schwartz et al. The WTP estimates are based on studies which considered the value of a *day* of avoided symptoms, whereas the Schwartz et al. study used as its measure a *case* of LRS. Because a case of LRS usually lasts at least one day, and often more, WTP to avoid a day of LRS should be a conservative estimate of WTP to avoid a case of LRS.

In the absence of information about the frequency of each of the eleven types of LRS among all occurrences of LRS, the uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution of MWTPs in Exhibit 4-16. This is the same procedure as that used in the URS uncertainty analysis.

As with URS, it is worth emphasizing that what is being valued here is LRS *as defined by Schwartz et al. (1994)*. While other definitions of LRS are certainly possible, this definition of LRS is used in this benefit analysis because it is the incidence of this specific definition of LRS that has been related to PM exposure by Schwartz et al.

[33] 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.

Issues in the Valuation of URS and LRS

The point estimates derived for mean WTP to avoid a day of URS and a case of LRS are based on the assumption that WTPs are additive. For example, if WTP to avoid a day of cough is $8.93, and WTP to avoid a day of shortness of breath is $6.37, then WTP to avoid a day of both cough and shortness of breath is $15.30. If there are no synergistic effects among symptoms, then it is likely that the marginal utility of avoiding symptoms decreases with the number of symptoms being avoided. If this is the case, adding WTPs would tend to overestimate WTP for avoidance of multiple symptoms. However, there may be synergistic effects– that is, the discomfort from two or more simultaneous symptoms may exceed the sum of the discomforts associated with each of the individual symptoms. If this is the case, adding WTPs would tend to underestimate WTP for avoidance of multiple symptoms. It is also possible that people may experience additional symptoms for which WTPs are not available, again leading to an underestimate of the correct WTP. However, for small numbers of symptoms, the assumption of additivity of WTPs is unlikely to result in substantive bias.

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

Information about the degree of uncertainty from either the second or the third source is not available. The first source of uncertainty, however, is addressed because an occurrence of URS or LRS may vary in symptoms. For example, seven different symptom complexes that qualify as URS, as defined by Pope et al. (1991), were identified above. The estimates of MWTP to avoid these seven different kinds of URS range from $8.93 (to avoid an occurrence of URS that consists of only coughing) to $41.71 (to avoid an occurrence of URS that consists of coughing plus head/sinus congestion plus eye irritation). There is no information, however, about the frequency of each of the seven types of URS among all occurrences of URS.

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

Alternatively, a discrete distribution of the seven unit dollar values associated with each of the seven types of URS identified could be used. This would provide a distribution whose mean is the same as the point estimate of MWTP. A continuous uniform distribution, however, is probably more reasonable than a discrete uniform distribution. The differences between the means of the discrete uniform distributions (the point estimates) and the means of the continuous uniform distributions are relatively small, as shown in Exhibit 4-17.

Exhibit 4-17 Comparison of the Means of Discrete and Continuous Uniform Distributions of MWTP Associated with URS and LRS (1990 $)

Health Endpoint	Mean of Discrete Uniform Distribution (Point Est.)	Mean of Continuous Uniform Distribution
URS (Pope et al., 1991)	18.70	19.86
LRS (Schwartz et al., 1994)	11.82	11.92

4.4.4 Minor Restricted Activity Days (MRADs) Adjusted for Asthma Attacks

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRAD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. We developed separate coefficients for each year in the analysis (1976-1981), which were then combined for use in this analysis. The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild (1989), Table 4, using the inverse of the variance as the weight. To avoid double counting, the number of asthma attacks estimated by the Whittemore and Korn-based (1980) C-R functions were subtracted from the number of MRADs estimated by Ostro and Rothschild.

Valuing Minor Restricted Activity Days (MRADs)

The unit value and uncertainty distribution for MRADs for this analysis were obtained by adjusting the (rounded) values in 1990 $ used in the §812 Prospective analysis to 1999 $ by multiplying by 1.275. No studies are reported to have estimated WTP to avoid a minor restricted activity day (MRAD). However, IEc (1993) has derived an estimate of WTP to avoid a minor respiratory restricted activity day (MRRAD), using WTP estimates from Tolley et al. (1986) for avoiding a three-symptom combination of coughing, throat congestion, and sinusitis. This estimate of WTP to avoid a MRRAD, so defined, is $38.37 (1990 $), or about $38. Although Ostro and Rothschild (1989) estimated the relationship between $PM_{2.5}$ and MRADs, rather than MRRADs (a component of MRADs), it is likely that most of the MRADs associated with exposure to $PM_{2.5}$ are in fact MRRADs. For the purpose of valuing this health endpoint, then, we assumed that MRADs associated with PM exposure may be more specifically defined as MRRADs, and therefore used the estimate of mean WTP to avoid a MRRAD. We subtract asthma attacks from MRADs before they are valued.

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

Given that there is inherently a substantial degree of arbitrariness in any point estimate of WTP to avoid a MRRAD (or other kinds of restricted activity days), the reasonable bounds on such an estimate must be considered. By definition, a MRRAD does not result in loss of work. WTP to avoid a MRRAD should therefore be less than WTP to avoid a WLD. At the other extreme, WTP to avoid a MRRAD should exceed WTP to avoid a single mild symptom. The highest IEc midrange estimate of WTP to avoid a single symptom is $15.72 (1990 $), or about $16, for eye irritation. The point estimate of WTP to avoid a WLD in the benefit analysis is $83 (1990 $). If all the single symptoms evaluated by the studies are not severe, then the estimate of WTP to avoid a MRRAD should be somewhere between $16 and $83. Because the IEc estimate of $38 falls within this range (and acknowledging the degree of arbitrariness associated with any estimate within this range), the IEc estimate is used as the mean of a triangular distribution centered at $38, ranging from $16 to $61. Adjusting to 1999 $, this is a triangular distribution centered at $48.43, ranging from $20.40 to $77.76.

4.4.5 Asthma Attacks

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants. Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks.

Alternative Calculation: Valuing Asthma Attacks

Although we include the number of avoided asthma attacks in the primary analysis, we present the value of these avoided attacks as an alternative calculation. The value of avoiding an asthma attack is estimated as the mean of four WTP estimates obtained in a study by Rowe and Chestnut (1986). The four WTP estimates correspond to four severity definitions of a "bad asthma day." The mean of the four average WTPs is $32 (1990 $), or $40.80 in 1999 $. The uncertainty surrounding this estimate was characterized by a continuous uniform distribution on the range defined by the lowest and highest of the four average WTP estimates from Rowe and Chestnut, [$12, $54], or [$15.30, $68.85] in 1999 $.

4.4.6 Work Loss Days (WLD)

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

Valuing WLD

Willingness to pay to avoid the loss of one day of work was estimated by dividing the median weekly wage for 1990 (U.S. Bureau of the Census, 1992) by five (to get the median daily wage). This values the loss of a day of work at the national median wage for the day lost. To account for regional variations in median wages, the national daily median wage was adjusted on a county-by-county basis using a factor based on the ratio of national median household income divided by each county's median income. Each county's income-adjusted willingness to pay to avoid the loss of one day of work was then used to value the number of work loss days attributed to that county. 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.[34]

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. The median daily wage in 1990 was $83, or $105.83 in 1999$. This is the value that was used to represent work loss days (WLD). An uncertainty distribution for this endpoint was unavailable, therefore the same central estimate ($101.92) was used to value incidence changes at the fifth, mean, and ninety-fifth percentiles.

4.4.7 Worker Productivity

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst (1981) and summarized in EPA (1994). Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration (-0.1427).[35] The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income. Given the median daily income for outdoor workers engaged in strenuous activity reported by the 1990 U.S. Census, $93.05 per day (1999 $), a ten percent reduction in ozone yields about $1.31 in increased daily wages. The median daily income for outdoor workers is a national estimate, however. We adjust this estimate to reflect

[34] The estimate of the value of work loss days avoided could be improved if, instead of a single national wage rate, state-specific or county-specific wage rates were used.

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

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

4.4.8 Supplemental Endpoints: Acute Illnesses And Symptoms Not Requiring Hospitalization

The benefits associated with several endpoints are estimated separately but are not included in the total benefits estimates because of the possibility of double counting of benefits.

"Any of 19 Respiratory Symptoms"

Krupnick et al. (1990) estimated the impact of coefficient of haze (COH, a measure of particulate matter concentrations), ozone and SO_2 on the incidence of any of 19 symptoms or conditions in the adult population, ages 18 to 65.[36] They used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model is that the model they used includes a lagged value of the dependent variable. This makes the derivation of a C-R function somewhat more complicated than the usual logistic regression.[37]

The presence of "any of 19 acute respiratory symptoms" is a somewhat subjective health effect used by Krupnick et al. (1990). Moreover, not all 19 symptoms are listed in the Krupnick et al. study. It is therefore not clear exactly what symptoms were included in the study. Even if all 19 symptoms were known, it is unlikely that WTP estimates could be obtained for all of the symptoms. Finally, even if all 19 symptoms were known and WTP estimates could be obtained for all 19 symptoms, the assumption of additivity of WTPs becomes tenuous with such a large number of symptoms. The likelihood that all 19 symptoms would occur simultaneously, moreover, is very small.

Valuing "Any of 19 Respiratory Symptoms"

The unit value and uncertainty distribution for "any of 19 respiratory symptoms" for this analysis were obtained by adjusting the (rounded) values in 1990 $ used in the §812 Prospective analysis to 1999 $ by multiplying by 1.275. Acute respiratory symptoms must be either upper respiratory symptoms or lower respiratory symptoms. In the absence of further knowledge about which of the two types of symptoms is more likely to occur among the "any of 19 acute respiratory symptoms," we assumed that they occur with equal probability. Because this health endpoint may also consist of combinations of symptoms, it was also assumed that there is some (smaller) probability that upper and lower respiratory symptoms occur together. To value avoidance of a day of "the presence of any of 19 acute respiratory symptoms" we therefore assumed that this health endpoint consists either of URS, or LRS, or both. We also assumed that it is as likely to be URS as LRS and that it is half as likely to be both together. That is, it was assumed that "the

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

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

presence of any of 19 acute respiratory symptoms" is a day of URS with 40 percent probability, a day of LRS with 40 percent probability, and a day of both URS and LRS with 20 percent probability. Using the point estimates of WTP to avoid a day of URS and LRS derived above, the point estimate of WTP to avoid a day of "the presence of any of 19 acute respiratory symptoms" is:

(0.40)($18.70) + (0.40)($11.82) + (0.20)($18.70 + $11.82) = $18.31, or about $18 (1990 $) .

This is $22.95 (=$18*1.275) in 1999 $. Because this health endpoint is only vaguely defined, and because of the lack of information on the relative frequencies of the different combinations of acute respiratory symptoms that might qualify as "any of 19 acute respiratory symptoms," the unit dollar value derived for this health endpoint must be considered only a rough approximation.

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

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

Moderate or Worse Asthma

Ostro et al. (1991) examined the effect of air pollution on asthmatics, ages 18 to 70, living in Denver, Colorado from December 1987 to February 1988. The respondents in this study were asked to record daily a subjective rating of their overall asthma status each day (0=none, 1=mild, 2=moderate, 3=severe, 4=incapacitating). Ostro et al. then examined the relationship between moderate (or worse) asthma and H^+, sulfate, SO_2, $PM_{2.5}$, estimated $PM_{2.5}$, PM_{10}, nitrate, and nitric acid. Daily levels of H^+ were linked to cough, asthma, and shortness of breath. $PM_{2.5}$ was linked to asthma. SO_2 was linked to shortness of breath. No effects were seen for other pollutants.

Valuing Moderate or Worse Asthma

The unit value and uncertainty distribution for moderate or worse asthma were assumed to be the same as for an asthma attack (see above), based on four WTP estimates from Rowe and Chestnut (1986). The mean of the four average WTPs is $32 (1990 $), or $40.80 in 1999$. The uncertainty surrounding this estimate was characterized by a continuous uniform distribution on the range defined by the lowest and highest of the four average WTP estimates from Rowe and Chestnut, [$12, $54], or [$15.30, $68.85] in 1999 $.

Although subjects' assessment of what constitutes a "bad asthma day" varied considerably in the Rowe and Chestnut (1986) study, the subjective assessment of an asthma day being bad is very similar to

the subjective assessment of an asthma day being "of moderate or worse status" in the Ostro et al. (1991) study, in which subjects were also asked their subjective assessments.

Shortness of Breath

Using logistic regression, Ostro et al. (1995) estimated the impact of PM_{10}, ozone, NO_2, and SO_2 on the incidence of coughing, shortness of breath, and wheezing in 83 African-American asthmatic children aged 7-12 living in Los Angeles from August through September 1992. Regression results show both PM_{10} and ozone significantly linked to shortness of breath; the beginning of an asthma episode was also significantly linked to ozone. Results for single-pollutant models only were presented in the published paper.

Valuing Shortness of Breath

A point estimate of mean WTP to avoid a day of shortness of breath was derived as the mean of the median estimates from two studies that evaluated this symptom. The median estimate from Dickie et al. (1987), was $0.00; the median estimate from Loehman et al. (1979) was $10.57, or about $10.60 (1990 $). The mean of these two medians is $5.30, or $6.76 in 1999$. In the absence of sufficient information to characterize the distribution of MWTP to avoid a day of shortness of breath, this distribution is roughly approximated by a continuous distribution on the interval from the low estimate to the high estimate – [$0.00, $10.60] in 1990 $, or [$0.00, $13.52] in 1999 $.

5 Welfare Benefits

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

5.1 Visibility Benefits

Visibility degradation estimates used in this analysis are generated by the REMSAD model. Because these air quality-related changes in visibility are directly used in the benefits analysis, the methodology for predicting visibility changes is not discussed here. The visibility estimation is described in detail in EPA (2000b), and is based on the methods in Sisler (1996).

Economic benefits may result from two broad categories of visibility changes: (1) changes in "residential" visibility – i.e., the visibility in and around the locations where people live; and (2) changes in "recreational" visibility at Class I areas – i.e., visibility at Class I national parks and wilderness areas.[38] In this analysis, only those recreational benefits in Class I areas that have been directly studied (in California, the Southeast, and the Southwest) are included in the primary presentation of benefits; residential benefits and recreational benefits in all U.S. Class I areas are presented as alternative calculations of visibility benefits.

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

To value estimated visibility changes, we are using an approach consistent with economic theory. Below we discuss an application of the Constant Elasticity of Substitution (CES) utility function approach[39] to value both residential visibility improvements and visibility improvements at Class I areas in the United States. This approach is based on the preference calibration method developed by Smith et al. (1999). The presentation of this methodology is organized as follows. The basic utility model is presented in Section 5.1.1. In Section 5.1.2 we discuss the measurement of visibility, and the mapping from environmental "bads" to environmental "goods." In Sections 5.1.3 and 5.1.4 we summarize the information that is available to estimate the parameters of the model corresponding to visibility at in-region

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

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

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

5.1.1 Basic Utility Model

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

(1) "all consumption goods," X,
(2) visibility in the residential area in which the household is located ("residential visibility"),[40]
(3) visibility at Class I areas in the same region as the household ("in-region recreational visibility"), and
(4) visibility at Class I areas outside the household's region ("out-of-region recreational visibility").

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

$$U_{ni} = \left(X^r + qZ_n^r + \sum_{k=1}^{N_i} g_{ik} Q_{ik}^r + \sum_{j \neq i} \sum_{k=1}^{N_j} d_{jk} Q_{jk}^r \right)^{1/r} ,$$

$$q > 0, g_{ik} > 0, \forall i,k, d_{jk} > 0, \forall j,k, \; r \leq 1.$$

where

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

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

The household's budget constraint is:

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

then the environmental "bad" can be related to utility via the corresponding environmental "good":[41]

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

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

There is a potential problem with this approach, however. If the function relating B and Q is not the same everywhere (i.e., if for a given value of B, the value of Q depends on other factors as well), then there can be more than one value of the environmental good corresponding to any given value of the environmental bad, and it is not clear which value to use. This has been identified as a problem with translating deciviews (an environmental "bad") into visual range (an environmental "good"). It has been noted that, for a given deciview value, there can be many different visual ranges, depending on the other factors that affect visual range – such as light angle and altitude. We note here, however, that this problem is not unique to visibility, but is a general problem when trying to translate environmental "bads" into "goods."[42]

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

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

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

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

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

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

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

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

This conversion is based on specific assumptions characterizing the "average" conditions of those factors, such as light angle, that affect visual range. To the extent that specific locations depart from the average conditions, the relationship will be an imperfect approximation.[43]

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

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

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

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

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

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

and

$$d_j = \frac{(m - WTP_j^{out})^r - m^r}{(Q_{0j}^r - Q_{1j}^r)}.$$

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

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

areas for which in-region and out-of-region WTP estimates are available from Chestnut and Rowe (1990), and the sources of benefits transfer-based estimates that we employ in the absence of estimates, are summarized in Exhibit 5-1. In all cases, WTP refers to WTP per household.

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

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

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

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

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

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

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

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

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

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

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

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

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

(1) We estimate region-specific parameters for the region in the modeled domain covered by Chestnut and Rowe (1990) (California, the Colorado Plateau, and the Southeast) – γ_1, γ_2, and γ_3 and δ_1, δ_2, and δ_3. **(2)** We infer region-specific parameters for those regions not covered by the Chestnut and Rowe study (the Northwest United States, the Northern Rockies, and the rest of the U.S.) – γ_4, γ_5, and γ_6 and δ_4, δ_5, and δ_6. **(3)** We derive park- and wilderness area-specific parameters within each region (γ_{1k} and δ_{1k}, for k=1, ..., N_1; γ_{2k} and δ_{2k}, for k=1, ..., N_2; and so forth).

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

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

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

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

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

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

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

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

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

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

$$d_4 = \frac{(m - WTP_4^{out})^r - m^r}{Q_{04}^r - Q_{14}^r}$$

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

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

One statistic which is likely to reflect both the quality and quantity of national parks in a region is the average annual visitation rate to the parks in that region. A reasonable way to gauge the extent to which out-of-region people would be willing to pay for visibility changes in parks in the Northwest United States versus in California might be to compare visitation rates in the two regions.[44] Suppose, for example, that twice as many visitor-days are spent in California parks per year as in parks in the Northwest United States per year. This could be an indication that the parks in California are in some way more desirable than those in the Northwest United States and/or that there are more of them -- i.e., that the environmental goods being valued in the two regions ("visibility at national parks") are not the same.

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

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

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

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

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

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

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

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

Estimating Park- and Wilderness Area-Specific Parameters

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

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

If study subjects were lumping all Class I areas together in their minds when giving their WTP responses, then it would be reasonable to allocate that WTP among the specific parks and wilderness areas in the region to derive park- and wilderness area-specific γ's and δ's for the region. If, on the other hand, study subjects were thinking only of the (mostly) parks shown on the map when they gave their WTP

response, then there are two possible approaches that could be taken. One approach assumes that households would be willing to pay some additional amount for the same visibility improvement in additional Class I areas that were not shown, and that this additional amount can be estimated using the same benefits transfer approach used to estimate region-specific WTPs in regions not covered by Chestnut and Rowe (1990).

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

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

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

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

and

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

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

While this is not a perfect allocation scheme, it is a reasonable scheme, given the limitations of data. Visitors to national parks in the United States are not all from the United States, and certainly not all from the region in which the park is located. A very large proportion of the visitors to Yosemite National Park in California, for example, may come from outside the U.S. The above allocation scheme implicitly assumes that the relative frequencies of visits to the parks in a region *from everyone in the world* is a

reasonable index of the relative WTP of an average household in that region (WTP_j^{in}) or out of that region (but in the U.S.) (WTP_j^{out}) for visibility improvements at these parks.[45]

A possible problem with this allocation scheme is that the relative frequency of visits is an indicator of use value but not necessarily of nonuse value, which may be a substantial component of the household's total WTP for a visibility improvement at Class I areas. If park A is twice as popular (i.e., has twice as many visitors per year) as park B, this does not necessarily imply that a household's WTP for an improvement in visibility at park A is twice its WTP for the same improvement at park B. Although an allocation scheme based on relative visitation frequencies has some obvious problems, however, it is still probably the best way to allocate a collective WTP.

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

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

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

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

then[46]:

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

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

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

and

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

which implies that:

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

where:

$$\alpha_{jk} = \frac{(m - WTP_{jk}^{in})^r - m^r}{(m - WTP_j^{in})^r - m^r} .$$

We use the adjustment factor, a_{jk}, to derive γ_{jk} from γ_j*, for the k^{th} Class I area in the j^{th} region. We use an analogous procedure to derive δ_{jk} from δ_j* for the k^{th} Class I area in the j^{th} region (where, in this case, we use WTP_j^{out} and WTP_{jk}^{out} instead of WTP_j^{in} and WTP_{jk}^{in}).[47]

5.1.4 Estimating the Parameter for Visibility in Residential Areas: θ

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

$$q = \frac{(m - WTP)^r - m^r}{(Z_0^r - Z_1^r)} .$$

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

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

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

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

The national benefits associated with any suite of visibility changes is properly calculated as the sum of these household WTPs for those changes. The benefit of any subset of visibility changes (e.g., changes in visibility only at Class I areas in California) can be calculated by setting all the other components of the WTP function to zero (that is, by assuming that all other visibility changes that are not of interest are zero). This is effectively the same as assuming that the subset of visibility changes of interest is the first or the only set of changes being valued by households. Estimating benefit components in this way will yield slightly upward biased estimates of benefits, because disposable income, m, is not being reduced by the WTPs for any prior visibility improvements. That is, each visibility improvement (e.g., visibility at Class I areas in the California) is assumed to be the first, and they cannot all be the first. The upward bias should be extremely small, however, because all of the WTPs for visibility changes are likely to be very small relative to income.

5.2 Agricultural Benefits

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

5.2.1 Exposure-Response Functions

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

range of responsiveness. The most tolerant and responsive functions are used to represent minimum and maximum impacts, *within the limits of available data*.

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

Use of cumulative exposure-response functions is relatively recent, and few experiments have been designed or reported in terms of the SUM06 index. Because the NCLAN program used a consistent protocol and developed a database of experimental conditions and results for all of its studies, U.S. EPA's Environmental Research Laboratory (ERL) was able to use original data from NCLAN studies to develop SUM06 exposure response functions for most NCLAN crops[48] (Lee and Hogsett, 1996). In addition, the agricultural model used in this analysis does not reflect non-commodity crops such as lettuce and kidney beans (described below). Exhibit 5-3 presents the exposure-response functions used in this analysis.

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

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

Source: Lee and Hogsett (1996)

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

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

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

where:

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

Application of Exposure Response Functions to a Non-Zero Baseline

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

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

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

Ozone Index Computation

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

Finally, because growing seasons vary throughout the U.S., the exposure needs to reflect the months in which a crop would be grown in a given location. To calculate the SUM06 index for the

appropriate growing season, state-level data on planting and harvesting dates was used in this analysis[49] (U.S. Department of Agriculture, 1984; U.S. EPA, 1993). To calculate the cumulative SUM06 index, the experimental duration for each crop was anchored on that crop's harvest date in each state in order to most closely approximate the relevant period of exposure for yield analysis. The harvest date was assumed to be the first day in the month of harvest, so that the SUM06 index includes the months up to but not including the harvest month.

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

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

5.2.2 Estimation of Yield Changes

In this analysis, use of a single exposure response function to estimate changes in yields implies that all producers are using a single cultivar of a given crop. This, combined with the limited number of cultivars evaluated in the NCLAN program, introduces an unquantifiable uncertainty into the estimation of yield changes. The most sensitive cultivar was used to represent the upper bound of the range that could be estimated, and the least sensitive cultivar was used to represent the lower bound of that range.

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

5.2.3 AGSIM© Model

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

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

The model is capable of analyzing the effects of changes in policies that affect crop yields or production costs. This is achieved by estimating how farmers will adjust crop acreage between commodities when relative profitability changes as a result of policy-induced crop yield and/or production cost changes.[50] Acreage and yield changes from various scenarios will affect total production of crops, which simultaneously affects both commodity prices and consumption. Commodity price changes, in turn, affect profitability and cropping patterns in subsequent years. Federal farm program and conservation reserve effects are also incorporated into the model. The model has been adapted to reflect the projections to 2010 from the last future year for which baseline forecasts are available: 2007. Although ozone impacts will be experienced far in the future, it was not possible to forecast the AGSIM© model far beyond USDA baseline forecasts that extend to 2007. Therefore, the 2030 ozone conditions were modeled using the 2010 version of the model.

Model Specification

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

Supply Components

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

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

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

farm programs, expected prices determine acreage. For these reasons, AGSIM© should be valid for simulating agricultural markets under the market conditions established under the 1996 FAIR Act.

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

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

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

where:

acresp(ic,it,irun)	=	acreage planted to the ic^{th} crop in the it^{th} year and in simulation "irun",
acrp(ic,it,irun)	=	acreage of crop "ic" that was placed in the conservation reserve program,
acrediv	=	acreage diverted under annual set-aside programs,
rerentm(ic,it,irun)	=	real expected per acre returns over variable costs for the ic^{th} crop,
rerentnp(it,irun)	=	real expected per acre returns over variables costs computed as a weighted average[51] of rerentm(ic,it,irun) over all endogenous crops,
time(it)	=	a time-trend variable, and
dumb83(it)	=	a binary dummy variable to account for the PIK program in crop year 1983.

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

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

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

where:

[51]Weights used in computing a composite expected return variable were the acreage harvested of each crop the previous year divided by total acreage harvested the previous year.

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

Expected yield is based on trend-line regressions:

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

where:
cint(ic) and by(ic) are estimated coefficients.

In the policy run, expected yield is adjusted for exogenously specified percentage yield changes ("dyld"):

(1c) ey(ic,it,irun) = [cint(ic) + by(ic)*time(it)]*(1.0 + dyld(ic,it)/100.)

Changes in real variable costs of production can also be exogenously specified for the policy simulation run. Thus, yield and cost changes directly impact acreage planted through equation (1), and indirectly impact acreage planted because of the resulting impact on prices in equation (1a) and thus in equation (1).

Given signs and magnitudes of estimated coefficients in equation (1), an increase in expected returns of the ic^{th} crop will increase acreage planted of that crop, while an increase in expected returns of other endogenous crops will decrease acreage of the ic^{th} crop. The estimated coefficient on lagged acreage planted in equation (1) is positive and less than one in value for all crops, which means that acreage planted is dynamically stable. The estimated coefficient on the set-aside acreage is negative and less than one in absolute value for all crops except oats, which reflects acreage slippage in the ARP program. Oats were typically planted to set-aside acreage, thus the estimated coefficient on set-aside acreage is positive in the oats equation, as expected. Further comments will be made on the acreage diverted effects on planted acreage after participation rate and acreage diverted equations, which are endogenous, are presented below.

Acreage Harvested Equations. Acreage harvested depends primarily on acreage planted:

(2) acresh(ic,it,irun) = bch(ic) + baph(ic)*acresp(ic,it,irun) + byrh(ic)*time(it) + bdvh(ic)*acrediv

where:
acresh(ic,it,irun) = the acreage harvested of the ic^{th} crop in the it^{th} year and in simulation "irun",

and other variables are as defined previously.

The estimated coefficient baph(ic) is positive and less than one, indicating that not all planted acreage is harvested, as expected. The coefficient bdvh(ic) on the acreage diverted variable is non-zero for oats only, in which case it is negative. This adjusts oat acreage harvested for the complexity of oats being planted (but not harvested) on ARP acreage. A time-trend variable for corn and grain sorghum, but not other crops shows how harvested acreage as a percentage of planted acreage has been increasing slightly over time.

Participation Rate in Farm Programs. Participation rates in the annual set-aside programs under the 1985 FSA and the 1990 FACTA were endogenized in the model with the set of equations:

(3) part(ic,it,irun) = bcp(ic) + brmp(ic)*rerntm(ic,it,irun) + brpp(ic)*rerntp(ic,it,irun) + byr(ic)*time(ic) + bpart(ic)*part(ic,it-1,irun) + bedpp(ic)*redp(ic,it,irun) + bd83p(ic)*dumb83(it)

where:

part(ic,it,irun) = the participation rate in the farm program for the ic^{th} crop in the it^{th} year and in simulation "irun",

rerntp(ic,it,irun)= real expected returns over variable costs based on the support (target) price for that crop,

redp(ic,it,irun) = real effective acreage diversion payment rate,

and other variables are as defined previously.

Estimated coefficients brpp(ic) are non-negative, indicating that an increase in expected returns based on support price will increase participation, while estimated coefficients brmp(ic) are non-positive, indicating that an increase in expected returns based on expected market price will decrease participation. Lagged participation rate in equation (3) shows strong dynamics with respect to farm program participation.

Acreage Diverted under Farm Programs. Acreage diverted under annual set-aside (or ARP) programs is modeled as:

(4) adiv(ic,it,irun) = bcd(ic) + bd83d(ic)*dumb83(it) + bedpd(ic)*redp(ic,it,irun) + byrd(ic)*time(it) + bpsa(ic)*sa(ic,it,irun)*part(ic,it,irun)

where:

adiv(ic,it,irun) = acreage diverted under annual diversion programs for the ic^{th} crop in the it^{th} year and in simulation "irun",

sa(ic,it,irun) = the set-aside rate specified by the Secretary of Agriculture under 1985 FSA and 1990 FACTA,

and other variables are as defined previously.

Acreage slippage (with respect to historical set-aside) in farm programs is implicit in the model specification, and results from the complex simultaneity of farm program variables in sets of equations (1), (3), and (4).

Acreage in Cultivated Summer Fallow. Acreage in cultivated summer fallow is modeled by the equation:

(5) afl(it,irun) = bcfl + bafl*afl(it-1,irun) + berfl*rerentnp(it,irun) + byrfl*time(it) + bd83fl*dumb83(it)

where:

afl(it,irun) = acreage fallowed in year it in simulation run "irun".

Although the acreage in cultivated summer fallow is highly inelastic, this equation shows that an increase in expected returns based on expected market price results in a small decrease in acreage fallowed.

Demand Components

The crop demand component of AGSIM© is based on a set of demand equations for each crop for utilization categories of (a) imports, (b) exports,(c) livestock feed, (d) food, fiber, ethanol production and other domestic uses, (e) ending stocks, and (f) residual use. Each demand component depends on current market price for that commodity and, where relevant, prices of other commodities. The model specification of each utilization category follows.

Imports. Imports of agricultural commodities are modeled by the set of equations:

(6) qd(ic,it,irun,1) = bim(1,ic) + bim(2,ic)*rp(ic,it,irun)*xrate(ic,it-1,irun) + bim(3,ic)*qd(ic,it-1,irun,1) + bim(4,ic)*time(it) + bim(5,ic)*uspop(it,irun)

where:

qd(ic,it,irun,1)	=	the quantity of crop ic imported in year it in simulation run "irun",
rp(ic,it,irun)	=	real market price,
xrate(ic,it-1,irun)	=	the real trade-weighted exchange rate,
uspop(it,irun)	=	the United States population,

and bim(j,ic) are estimated coefficients. Lagged imports in equation (6) reflects dynamic adjustments.

Exports. Exports of agricultural commodities are modeled by the set of equations:

(7) qd(ic,it,irun,2) = bex(1,ic) + bex(2,ic)*rp(ic,it,irun)*xrate(ic,it-1,irun) + bex(3,ic)* qd(ic,it-1,irun,2) + bex(4,ic)*time(it) + bex(5,ic)*wpop(it,irun)

where:

qd(ic,it,irun,2)	=	the quantity of crop ic exported in year it in simulation run "irun", and
wpop(it,irun)	=	world population.

Feed, Fiber and Crushing Use. Domestic utilization of crops for feed, fiber or crushing (depending on the crop) is modeled by the set of equations:

(8) qd(ic,it,irun,3) = bfd(1,ic) + $\sum_{jc}$bfdcross(ic,jc)*rp(jc,it,irun) + bfd(2,ic)*qd(ic,it-1,irun,3) + bfd(3,ic)*time(it)

where:

qd(ic,it,irun,3)	=	utilization for feed, fiber or crushing.

Note that cross-price effects are incorporated into this set of equations through the set of estimated coefficients bfdcross(ic,jc). Symmetry of cross-price effects, consistent with microeconomic theory, was imposed on estimation so that bfdcross(ic,jc) = bfdcross(jc,ic) for ic $\neq$ jc. Own-price effects are all negative, as expected.

Domestic Food Use. The set of equations to represent domestic food use is:

(9) qd(ic,it,irun,4) = bfo(1,ic) + bfo(2,ic)*rp(ic,it,irun) + bfo(3,ic)*qd(ic,it-1,irun,4) + bfo(4,ic)*time(it) + bfo(5,ic)*uspop(it,irun) + bfo(6,ic)*rdincome(it,irun)

where:

rdincome(it,irun) = real per-capita disposable income in the United States,

and other variables are as defined previously. In the case of peanuts, the real market price is replaced by the fixed quota price that applies to all domestically consumed peanuts. This quota price for peanuts applies to the 1985 FSA, the 1990 FACTA, and continues with the 1996 FAIR Act.

Ending Stocks. Ending stocks are viewed as another component of demand. Although commodities are often held to maintain pipeline inventories, commodities are also held for speculative purposes. Thus, stock levels respond strongly to prices, so the stock relationships were specified and estimated as

(10) qd(ic,it,irun,5) = bst(1,ic) + bst(2,ic)*rp(ic,it,irun) + bst(3,ic)*qd(ic,it-1,irun,5) + bst(4,ic)*time(it)

where qd(ic,it,irun,5) is ending stocks in year t.

Residual Use. For some crops (rice, peanuts, and cottonseed), supply and utilization data show a residual category, which is modeled as,

(11) qd(ic,it,irun,6) = brs(1,ic) + brs(2,ic)*rp(ic,it,irun) + brs(3,ic)*time(it)

where:

qd(ic,it,irun,6) = residual use.

Although quantities in this residual use category are never used, the level of the residual does respond negatively to the real price, and is thus viewed as another utilization (demand) category.

Market Clearing Identities

In supply and demand specification outlined above, supply generally depends on past prices, while demand depends on current prices. In simulating these econometrically estimated equations into the future, simulated prices are solved by simultaneously solving the market clearing identities

(12) qs(ic,it,irun) +qd(ic,it-1,irun,5) = qd(ic,it,irun,1) + qd(ic,it,irun,2) + qd(ic,it,irun,3) + qd(ic,it,irun,4) + qd(ic,it,irun,5) + qd(ic,it,irun,6)

where:

qs(ic,it,irun) = the quantity produced of crop ic in year it in simulation “irun”.

Production is defined to be qs(ic,it,irun) = acresh(ic,it,irun)*ey(ic,it,irun). The left hand side of the equal sign in (12) gives total supply (production plus beginning stocks), while the right-hand side of (12) gives total utilization, including ending stocks.

In the simulation model this set of simultaneous equations are numerically solved to get the market clearing prices in a given year. This process is continued, considering the dynamics of the model, indefinitely into the future.

Historical Observation Period

Many econometric relationships in the model were estimated with data for the 1975-1995 time period. However, where structural change was apparent, such as with stock holding behavior and international trade, some of the early years were dropped from statistical analysis so that the simulation model would better reflect the future.

Alternative Specifications Considered

Many different specifications of how farm programs influence crop acreage have been considered in the evolution of AGSIM©, including: (a) acreage depends on support price, (b) acreage depends on the maximum of expected market price and support price, (c) acreage depends on a weighted average of support and expected market prices, with weights based on program and non-program acreage of the crop, and (d) acreage depends on expected market price. Models for expected market price have considered complex distributed lags that go back several years in time, to a simple model that expected market price is actual price the previous year. Acreage equations have also been specified to depend on expected returns of: (1) all competing individual crops with no parameter restrictions, (2) all competing individual crops with full symmetry of cross-effects imposed on estimation, (3) major competing individual crops, and (4) a weighted average of all expected returns for all other crops. Many different ways of incorporating participation rates and acreage diverted into the model have also been considered. Several alternative functional forms (linear, log-linear, nonlinear share equations, asymptotic) have also been considered. Theoretical specifications considered have ranged from *ad hoc* models to very tightly specified and detailed theoretical economic models based on complex assumptions. The present model draws from economic theory (e.g. symmetry of cross-price effects in demand and homogeneity of degree zero of all supply and demand equations with respect to prices), but does not specify the model so tightly with untested assumptions and functional forms that empirical data has almost no role in the resulting estimates. Alternative estimation techniques, ranging from simultaneous equations techniques, to Zellner's seemingly unrelated regressions, to ordinary least squares regression have been used. The current version of AGSIM© reflects a degree of subjective judgement of what best reflects supply and demand of agricultural commodities based on microeconomic theory, traditional statistical criteria, and substantive direct contact with farmers and ranchers in most regions of the United States.

Baseline

The current version of AGSIM© is designed to estimate *changes* in the agricultural sector resulting from pesticide or other policy. Changes in economic variables are computed by comparing a policy simulation of the model with a baseline simulation of the model. For *ex post* (retrospective) evaluations, the baseline reflects actual farm programs, prices, acreages, etc. However, for *ex ante* evaluations,

AGSIM© is calibrated to an external baseline. The calibration is done by comparing an internally generated baseline to the external baseline and computing adjusted intercepts for all of the relevant demand and supply relationships in AGSIM©**.**

For the 1999 version of AGSIM© the externally specified year 2010 baseline is forecasted from the 2007 baseline reported by USDA (1988b). A few endogenous variables in AGSIM© were not included in the USDA baseline. In those cases, the 1997 FAPRI baseline was used (FAPRI, 1997).

It should be noted that the baseline is not especially critical to estimates of *changes* in the agricultural sector, except for the case of price support policy, which is not relevant here. That is, sensitivity analyses with previous versions of AGSIM© have shown that estimates of *changes* in variables are not very sensitive to baseline absolute values of variables. Use of the USDA baseline to the extent possible assures consistency with other governmental mandated agricultural policy analyses.

Regional Effects Sub-Model

AGSIM© subroutines are also available to combine AGSIM© output with production cost information to estimate net farm income impacts for the policy scenario at the regional level (or farm, representative farm, area or state level). Required information for this type of evaluation includes for each farm or area: (a) yield and cost changes (which often differ from the national yield and cost changes for the policy scenario), (b) baseline production costs, and(c) acreages of each crop. This information is combined with price impacts estimated with AGSIM©, and regional supply elasticities from a prior version of AGSIM© (or from other sources) to estimate net farm income changes for the farms or areas considered.

The conceptual foundation for regional evaluation in this version of AGSIM© begins with a net farm income formula,

$$\Pi_{ir} = \sum_{ic} A_{ic,ir} R_{ic,ir} \tag{13}$$

where:

Π_{ir} = net farm income in region ir,
Aic,ir = acreage harvested of the icth crop in that region, and
$R_{ic,ir}$ = per-acre net return in that region.

Based on equation (13), it can be shown that the theoretically appropriate formula for computing net farm income *changes* for different regional situations is:

$$\frac{\Delta \Pi_{ir}}{\Delta Z} \cong \sum_{ic} R_{ic,ir} \sum_{jc} \frac{\Delta A_{ic,ir}}{\Delta R_{jc,ir}} \frac{\Delta R_{jc,ir}}{\Delta Z} + \sum_{ic} A_{ic,ir} \sum_{jc} \frac{\Delta R_{ic,ir}}{\Delta Z} \tag{14}$$

where:

Δ represents a discrete change,
ΔZ represents the discrete policy change,
ic and jc are crop indices,

and other variables are as previously defined.

Equation (14) can be expressed in acreage elasticity (with respect to per-acre income) form,

$$(15) \qquad \frac{\Delta \Pi_{ir}}{\Delta Z} \cong \sum_{ic} R_{ic,ir} \sum_{jc} e_{ic,ij,ir} \frac{R_{ic,ir}}{A_{jc,ir}} \frac{\Delta R_{jc,ir}}{\Delta Z} + \sum_{ic} A_{ic,ir} \sum_{jc} \frac{\Delta R_{ic,ir}}{\Delta Z}$$

where:

$\epsilon_{ic,ij,ir}$ = elasticity of acreage of the icth crop in the irth region with respect to per-acre income of the jcth crop in that region.

The term $\Delta R_{ic,ij}/\Delta Z$ in equations (14) and (15) can be further expanded to give

$$(16) \qquad \frac{\Delta R_{ic,ir}}{\Delta Z} \cong P_{ic} \Delta Y_{ic,ir} + Y_{ic,ir} \Delta P_{ic} - \Delta C_{ic,ir}$$

Formula (15) along with (16) can be empirically implemented to estimate the change in regional (or farm, representative farm, area or state level) farm income with the following information for each region: (a) crop budgets, (b) the change in yield and cost associated with the policy in question, price impacts estimated with AGSIM©, and externally specified (from an older version of AGSIM©, from subjective estimates, or from the literature) elasticities.

The first term on the right-hand side of (14) and (15) represents the change in net income resulting from increased or decreased acreage, while the last term on the right-hand side of (14) and (15) represents the change in net farm income on existing acreage of crops in the region. Since acreage response is generally inelastic, the last term on the right-hand side of (14) and (15) dominates the change in net farm income in a region; thus, elasticities generally will not have a major impact on regional net farm income changes estimated with the above approach.

AGSIM© Output

The major outputs from AGSIM© are changes in crop acreage, production, price, income, foreign consumer benefits, domestic consumer benefits, and farm program costs. The traditional method of economic welfare analysis (which is based on the concept of economic surplus) of policy changes is used to compute the sum of changes in producer surplus (net farm income) plus changes to all consumers (changes in consumers surplus) plus any changes in farm program payments (zero under 1996 FAIR). To avoid the possibility of inappropriately comparing a baseline with a policy scenario that was actually based on another baseline, a single run of AGSIM© produces both the baseline tables and the policy scenario tables, then computes economic surplus and price changes based on these two runs of the model.

Output from each run of the model includes two sets of tables for each crop; one set of tables for supply variables and another set of tables for supply and utilization variables. Each table includes historical statistics as well as simulations into the future. These tables are constructed for the baseline and the policy scenario.

5.3 Consumer Cleaning Cost Savings

Particulate matter air pollution has been shown to result in dirtier clothes, which in turn results in higher annual cleaning costs for consumers. One benefit of reduced particulate matter, then, is the consequent reduction in cleaning costs for consumers. Several studies have provided estimates of the cost to households of PM soiling. The study that is cited by ESEERCO (1994) as one of the most sophisticated and is relied upon by EPA in its 1988 Regulatory Impact Analysis for SO_2 is Manuel et al. (1982). Using a household production function approach and household expenditure data from the 1972-73 Bureau of Labor Statistics Consumer Expenditure Survey for over twenty cities in the United States, Manuel et al. estimated the annual cost of cleaning per $\mu g/m^3$ PM per household as $1.55 ($0.59 per person times 2.63 persons per household). This estimate is low compared with others (e.g., estimates provided by Cummings et al. (1985) and Watson and Jaksch (1982) are about eight times and five times greater, respectively). The ESEERCO report notes, however, that the Manuel estimate is probably downward biased because it does not include the time cost of do-it-yourselfers. Estimating that these costs may comprise at least half the cost of PM-related cleaning costs, they double the Manuel estimate to obtain a point estimate of $3.10 (reported by ESEERCO in 1992 dollars as $2.70).

The Manuel et al. (1982) study measured particulate matter as TSP rather than PM_{10} or $PM_{2.5}$. If a one $\mu g/m^3$ increase in TSP causes $1.55 worth of cleaning expenses per household, the same unit dollar value can be used for PM_{10} (or $PM_{2.5}$) only if particle size doesn't matter -- i.e., only if particles of all sizes are equally soiling. Suppose, for example, that PM_{10} is 75% of TSP and that all particles are equally soiling. Then 75% of the damage caused by a one $\mu g/m^3$ increase in TSP is due to PM_{10}. This is (0.75)($1.55) = $1.16. However, this corresponds to a 0.75 $\mu g/m^3$ increase in PM_{10}. A one $\mu g/m^3$ increase in PM_{10} would therefore yield a dollar soiling damage of $1.16/0.75 = $1.55.

Suppose, however, that only PM_{10} matters. Then the $1.55 underestimates the impact of a one $\mu g/m^3$ increase in PM_{10}, because it corresponds to a less than one $\mu g/m^3$ increase in PM_{10} (e.g., a 0.75 $\mu g/m^3$ increase in PM_{10}). In this case, the correct unit value per unit of PM_{10} would be ($1.55)/0.75 = $2.07. If only PM_{10} matters, then either (1) the dollar value can be adjusted by dividing it by the percentage of TSP that is PM_{10} and PM_{10} can be used in the soiling damage function, or (2) the dollar value can be left unadjusted and TSP, rather than PM_{10}, can be used in the soiling damage function.

Finally, it is possible that, while both PM_{10} and $PM_{2.5}$ are components of TSP that cause consumer cleaning costs, the remaining portion of TSP has a greater soiling capability than either the PM_{10} or $PM_{2.5}$ component. In this case, using either PM_{10} or $PM_{2.5}$ air quality data with a household soiling function based on TSP would yield overestimates of the PM_{10}- or $PM_{2.5}$-related consumer cleaning costs avoided by reductions in concentration of these pollutants.

There is, however, insufficient information on the relative soiling capabilities of the different components of TSP. This analysis assumes that all components of TSP have an equivalent soiling capacity.

6 Results

This chapter provides estimates of the magnitude and value of changes in selected health and welfare endpoints associated with HD Engine/Diesel Fuel -related changes in ambient ozone and PM concentrations. The total dollar benefits associated with a given endpoint depend on how much the endpoint will change (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth).

To place estimated incidence changes into context with predicted baseline incidence, Exhibit 6-1 displays the baseline incidence figures for those endpoints for which one can be calculated. Due to the nature of the endpoints, baseline incidence can be calculated only for ozone- and PM-related health effects. In addition to baseline incidence, for each health effect, both the mean estimated incidence change and corresponding percent change between post-control incidence reductions and the predicted incidence baseline is presented. Note that these baseline incidences include all incidences, not just those associated with air pollution.

Exhibits 6-2 and 6-3 present the primary incidence and benefit estimates associated with the primary scenario. A 5^{th} percentile, mean, and 95^{th} percentile estimate for both incidence and benefits is presented for each endpoint, as well as the simple mean benefit (calculated by multiplying the mean estimate of incidence by the corresponding mean valuation). Total benefits are also displayed, calculated by simply summing the simple mean of each endpoint.

Exhibit 6-4 displays alternative incidence and benefit calculations to those included in the primary analysis. Where possible, a 5^{th} percentile, mean, and 95^{th} percentile estimate for incidence and/or benefits is presented for each alternative endpoint. Exhibit 6-5 presents the aggregate uncertainty results (5^{th}, mean, and 95^{th} percentiles) for PM- and ozone-related benefits, as well as for total benefits (PM + ozone).

Exhibit 6-1 Baseline Percentages

Endpoint	Reference	2030 Control Scenario	
		Mean	% of Baseline
PM-related Baseline Percentages			
Ages 30+	Krewski et al. (2000)	8,292	0.31%
Chronic Bronchitis	Pooled Analysis	5,461	0.80%
COPD (ICD-9 codes 4490-492, 494-496)	Samet et al. (2000a)	900	0.18%
Pneumonia (ICD-9 codes 480-487)	Samet et al. (2000a)	1,106	0.13%
Asthma (ICD code 493)	Sheppard et al. (1999)	881	0.18%
Cardiovascular (ICD-9 codes 390-429)	Samet et al. (2000a)	2,667	0.08%
Asthma-related ER visits	Schwartz et al. (1993)	2,064	0.24%
Acute Bronchitis	Dockery et al. (1996)	17,590	1.62%
Upper Respiratory Symptoms	Pope et al. (1991)	193,402	0.15%
Lower Respiratory Symptoms	Schwartz et al. (1994)	192,899	1.13%
Asthma Attacks	Whittemore and Korn (1980)	175,931	0.09%
Work Loss Days	Ostro (1987)	1,539,396	0.30%
MRAD - Adjusted	Ostro and Rothschild (1989)	7,990,406	0.47%
Ozone-related Baseline Percentages			
Respiratory-Related	Pooled Analysis	1,173	0.03%
Dyrhythmias	Burnett et al. (1999)	312	0.04%
Asthma-Related ER Visits	Pooled Analysis	283	0.03%
Asthma Attacks	Whittemore and Korn (1980)	185,517	0.10%
MRAD (Adjusted for Asthma Attacks)	Ostro and Rothschild (1989)	1,848,092	0.11%

Exhibit 6-2 Estimated PM-Related Health and Welfare Benefits Associated with Air Quality Changes Resulting from the HD Engine/Diesel Fuel Rule 2030 Control Scenario

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 1999$)			Simple Mean
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile	
Mortality								
Ages 30+	Krewski et al. (2000)	4,829	8,292	11,698	$6,516	$48,129	$119,286	$48,245
		4,829	8,292	11,698	$6,120	$45,207	$112,042	$45,316
Chronic Illness								
Chronic Bronchitis	Pooled Analysis	1,884	5,478	9,464	$173	$1,803	$5,937	$1,805
Hospitalization								
COPD (ICD-9 codes 4490-492, 494-496)	Samet et al. (2000a)	164	900	1,607	$2	$11	$20	$11
Pneumonia (ICD-9 codes 480-487)	Samet et al. (2000a)	610	1,106	1,601	$9	$16	$24	$16
Asthma (ICD code 493)	Sheppard et al. (1999)	385	881	1,402	$3	$6	$10	$6
Cardiovascular (ICD-9 codes 390-429)	Samet et al. (2000a)	2,252	2,667	3,067	$41	$49	$56	$49
Asthma-related ER visits	Schwartz et al. (1993)	864	2064	3213	$0.3	$0.6	$1.1	$0.6
Minor Illness								
Acute Bronchitis	Dockery et al. (1996)	-88	17,590	35,900	$0.0	$1.0	$2.5	$1.0
Upper Respiratory Symptoms	Pope et al. (1991)	65,290	193,402	325,371	$1.2	$4.9	$10.3	$4.7
Lower Respiratory Symptoms	Schwartz et al. (1994)	88,308	192,899	295,784	$1	$3	$6	$3
Asthma Attacks	Whittemore and Korn (1980)	60,984	175,931	291,914	– [a]	– [a]	– [a]	– [a]
Work Loss Days	Ostro (1987)	1,337,267	1,539,396	1,733,280	$155	$178	$200	$163
MRAD - Adjusted	Ostro and Rothschild (1989)	6,806,718	7,990,406	9,104,836	$233	$391	$560	$387
Welfare Effects								
Recreational Visibility	Study Regions Only (CA, SW, and SE)	Direct Economic Valuation			--	$1,789	--	$1,789
Total Primary PM-related Benefits (3% discount rate)		--	--	--	--	--	--	**$52,488**
Total Primary PM-related Benefits (7% discount rate)		--	--	--	--	--	--	**$49,559**

Exhibit 6-2 Estimated PM-Related Health and Welfare Benefits Associated with Air Quality Changes Resulting from the HD Engine/Diesel Fuel Rule 2030 Control Scenario (cont.)

[a] Because of uncertainty surrounding the magnitude of the effect of ozone on asthma attacks, we do not value asthma attacks in the primary analysis. Instead, we assume that the valuation of asthma attacks is an alternative calculation.

Exhibit 6-3 Estimated Ozone-Related Health and Welfare Benefits Associated with Air Quality Changes Resulting from the HD Engine/Diesel Fuel Rule 2030 Control Scenario

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 1999$)			Simple Mean
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile	
Hospitalization								
Respiratory-Related	Pooled Analysis	205	1,173	2,085	$3	$17	$29	$17
Dyrhythmias	Burnett et al. (1999)	6	312	619	$0	$4	$8	$4
Asthma-Related ER Visits	Pooled Analysis	88	283	453	$0.03	$0.09	$0.15	$0.08
Minor Illness								
Asthma Attacks	Whittemore and Korn (1980)	70,352	185,517	305,807	– [a]	– [a]	– [a]	– [a]
MRAD (Adjusted for Asthma Attacks)	Ostro and Rothschild (1989)	988,645	1,848,092	2,706,607	$41	$90	$151	$90
Welfare Effects								
Decreased Worker Productivity	Crocker and Horst (1981) and EPA (1994)	Direct Economic Valuation			--	$142	–	$142
Agriculture		Direct Economic Valuation			--	$1,078	–	$1,078
Total Primary Ozone-related Benefits								$1,330

[a] Because of uncertainty surrounding the magnitude of the effect of ozone on asthma attacks, we do not value asthma attacks in the primary analysis. Instead, we assume that the valuation of asthma attacks is an alternative calculation.

Exhibit 6-4 Alternative Benefit Calculations for the HD Engine/Diesel Fuel Rule 2030 Control Scenario

Endpoint	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 1999$)			Simple Mean
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile	
PM-related Alternative Calculations								
Life Years Lost, by age:	Krewski et al. (2000)							
30-34		3,490	6,256	8,867	-	-	-	-
35-44		8,350	14,969	21,217	-	-	-	-
45-54		8,175	14,655	20,771	-	-	-	-
55-64		11,680	20,938	29,674	-	-	-	-
65-74		14,842	26,605	37,706	-	-	-	-
75-84		11,611	20,813	29,497	-	-	-	-
85+		6,455	11,571	16,399	-	-	-	-
Life years lost	3% discount rate	-	-	-	$3,480	$22,758	$52,486	$22,758
Life years lost	7% discount rate	-	-	-	$4,068	$26,554	$60,902	$26,554
Age-Adjusted Value of Statistical Lives Lost	Jones-Lee et al. (1989) 3% discount rate	-	-	-	$14,673	$26,303	$37,278	$43,049
	Jones-Lee et al. (1989) 7% discount rate	-	-	-	$13,782	$24,706	$35,014	$24,706
	Jones-Lee et al. (1993) 3% discount rate	-	-	-	$24,015	$43,049	$61,010	$43,049
	Jones-Lee et al. (1993) 7% discount rate	-	-	-	$22,556	$40,435	$57,306	$40,435
Chronic Bronchitis	Reversals	1,652	4,770	8,258	$47	$696	$2,394	$695
Hospital Admissions	Moolgavkar et al. (1997)							
COPD-related		-138	335	829	-$2	$4	$10	$4
Pneumonia-related		-165	265	696	-$2	$4	$10	$4
Recreational Visibility	All U.S. Class I Areas	Direct Economic Valuation			-	$2,486	-	$2,486
Residential Visibility	Eastern U.S.	Direct Economic Valuation			-	$688	-	$688
Residential Visibility	Western U.S.	Direct Economic Valuation			-	$500	-	$500
Household Soiling Damage	ESEERCO (1994)	Direct Economic Valuation			$145	$261	$472	$261
Ozone-related Alternative Calculations								
Chronic Asthma	McDonnell et al. (1999)	185	816	1,452	$5	$26	$47	$26

Exhibit 6-5 Measures of Aggregate Uncertainty in the Benefits Analysis

Benefits Aggregation	Monetary Benefits (millions 1999$) [a]		
	5th %ile [b]	Mean	95th %ile [b]
Total Ozone-Related Benefits	$477	$1,444	$2,403
Total PM-Related Benefits	$14,863	$69,247	$163,699
Total HD Engine/Diesel Fuel Rule Primary Analysis Benefits (Ozone + PM)	$16,176	$70,691	$165,184

[a] Measures of aggregate uncertainty also include an adjustment to account for growth in income from 1999 to 2030. See Table VII-12 in the Regurlatory Impact Analysis for the final HD Engine/Diesel Fuel rule for the adjustment factors used.

[b] Our calculations of the 5th and 95th percentile estimates include the value of asthma attacks; the effect is small. We excluded this from the mean estimates.

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Appendix A: Results for Supplementary Calculations and Sensitivity Analyses

Exhibit A-1 Supplemental Benefit Estimates for the 2030 Control Scenario

			Avoided Incidence (cases/year)			Monetary Benefits (millions 1999$)		
Endpoint	**Reference**	**Pollutant**	**5th %ile**	**Mean**	**95th %ile**	**5th %ile**	**Mean**	**95th %ile**
Premature Mortality (short-term)	Schwartz et al. (1996)	PM	2,217	2,588	2,961	$2,238	$14,826	$34,278
Premature Mortality (short-term)	Pooled analysis	Ozone	13	472	987	$14	$2,646	$7,808
Premature Mortality (infant population)	Woodruff et al. (1997)	PM	17	34	51	$26	$199	$505
Shortness of breath		PM	12,009	38,961	66,504	$0.0	$0.3	$0.6
Any of 19 Acute Respiratory Symptoms	Krupnick et al. (1990)	PM	4,177,871	24,556,892	45,842,008	$65	$606	$1,538
Any of 19 Acute Respiratory Symptoms	Krupnick et al. (1990)	Ozone	809,687	5,883,786	10,696,309	$14	$115	$277
Moderate or Worse Asthma	Ostro et al. (1991)	PM	27,731	182,500	328,015	$1	$8	$17

Exhibit A-2 Sensitivity Analysis Results for the 2030 Control Scenario

Mortality Lag	**Reference/Alternative Valuation**	Avoided Incidence (cases/year)			Monetary Benefits (millions 1997$)		
		5th %ile	**Mean**	**95th %ile**	**5th %ile**	**Mean**	**95th %ile**
No Lag		-	8,292	-	$50,747	$50,747	$50,747
5 Year	25%, 25%, 17%, 17%, 16%	-	8,292	-	$48,245	$46,727	$45,316
8 Year	Incidence Occurs 8th Year	-	8,292	-	$41,262	$36,065	$31,603
15 Year	Incidence Occurs 15th Year	-	8,292	-	$33,550	$25,631	$19,681
15 Year	Incidence Skewed Early	-	8,292	-	$47,237	$45,288	$43,586
15 Year	Incidence Skewed Late	-	8,292	-	$36,394	$29,492	$24,117

Exhibit A-3 Sensitivity Analysis: Effect of Thresholds on Estimated PM-Related Mortality Based on Krewski et al. (2000) - Mean, All-Cause

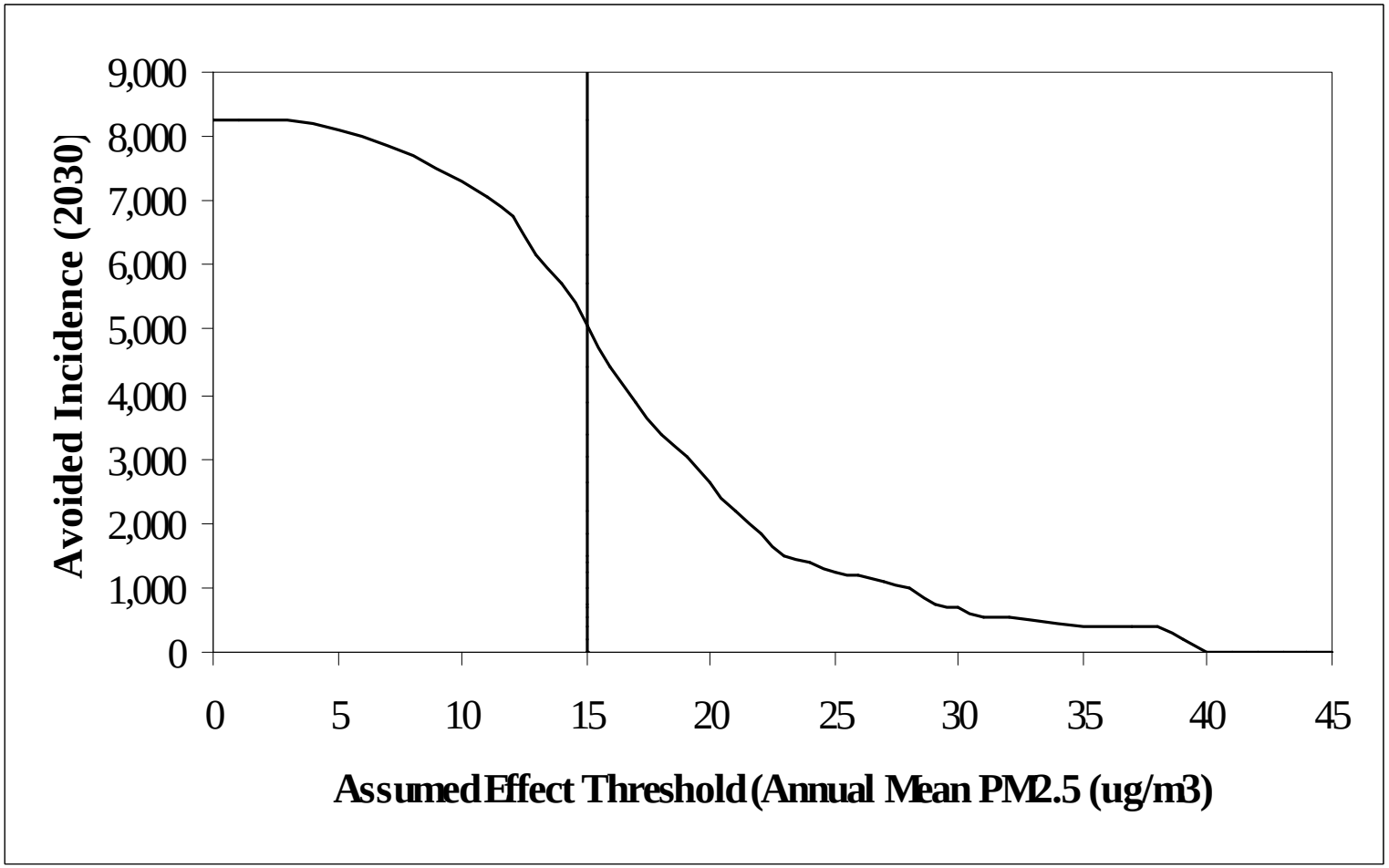

Exhibit A-4 Alternative Mortality Calculations

Age Group	Statistic	Mortality	Reference	Mortality Incidence (cases/year)			Monetary Benefits (million $ 1999)		
				5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Age 30+	Median	Non-Accidental	Pope et al. (1995)	5,905	9,418	13,010	$7,863	$55,274	$136,695
Age 30+	Median	All-Cause	Pope et al. (1995)	5,962	9,906	13,635	$8,639	$58,102	$139,341
Age 30+	Median	Non-Accidental	Krewski et al. (2000)	4,447	7,873	11,345	$6,272	$45,970	$114,045
Age 30+	Mean	Non-Accidental	Krewski et al. (2000)	4,606	7,910	11,158	$6,380	$45,934	$113,041
Age 30+	Median	All-Cause	Krewski et al. (2000) - Random Effects, Independent Cities	8,906	16,014	23,616	$12,560	$93,944	$235,287
Age 30+	Median	All-Cause	Krewski et al. (2000) - Random Effects, Regional Adjustment	499	9,360	18,212	$1,410	$53,928	$153,906
Age 25+	Mean	Non-Accidental	Dockery et al. (1993)	9,124	21,503	33,252	$15,118	$124,064	$314,567
	Mean	All-Cause	Dockery et al. (1993)	9,621	22,602	35,064	$15,712	$131,549	$330,979
Age 25+	Mean	Non-Accidental	Krewski et al. (2000)	11,803	23,079	34,993	$19,547	$135,373	$333,275
Age 25+	Mean	All-Cause	Krewski et al. (2000)	12,446	24,243	36,900	$18,895	$139,614	$348,525

Exhibit A-5 Underlying Estimates and Weights for Pooled Estimate of PM-Related Chronic Bronchitis Studies

Study	Ages Affected	Study Weights	mean	Std. Dev.
Abbey et al. (1995b): $PM_{2.5}$	>26	0.23	6,331	3,230
Schwartz (1993): $PM_{2.5}$	>29	0.77	5,105	1,782
Schwartz (1993): coarse PM_{10}	>29	1.00	100	35
Pooled estimate of chronic bronchitis			5,478	2,314

Exhibit A-6 Underlying Estimates and Weights for Pooled Estimate of Ozone-Related Respiratory Hospital Admissions

Study	Ages affected	Study weights	mean	Std. Dev.
Burnett et al. (1997), Toronto	all ages	0.01	6,996	1,484
Burnett et al. (1999), Toronto	all ages	0.01	1,737	3,09
Thurston et al. (1994), Toronto	all ages	0.01	1,151	6,64
Moolgavkar et al. (1997), Twin Cities	>64	0.32	1,102	305
Schwartz (1994a), Twin Cities	>64	0.28	552	328
Schwartz (1994b), Detroit	>64	0.26	1,601	341
Schwartz (1996), New Haven	>64	0.08	1,149	604
Schwartz (1996), Tacoma	>64	0.02	3,126	1,106
Pooled estimate of respiratory hospital admissions			1,173	838

Exhibit A-7 Underlying Estimates and Weights for Pooled Estimate of Ozone-Related Asthma ER Visits

Study	Ages Affected	Study Weights	mean	Std. Dev.
Cody et al. (1992)	>26	0.49	393	138
Weisel et al. (1995)	>26	0.49	858	139
Stieb et al. (1996)	>29	0.02	2,879	1,471
Pooled estimate of asthma ER			283	200

Appendix B: Ozone Concentration-response Functions

Note that ΔO_3 is defined as ($O_{3,\ baseline}$ - $O_{3,\ control}$), and that the change is defined as: ($incidence_{control}$ - $incidence_{baseline}$).

B.1 Short-term Ozone-related Mortality (Four U.s. Studies)

Four studies were used to estimate the possible relationship between ozone and increased mortality.

B.1.1 Short-Term Mortality (U.S.) (Ito and Thurston, 1996)

Ito and Thurston (1996) examined the relationship between daily non-accidental mortality and air pollution levels in Cook County, Illinois from 1985 to 1990. They examined daily levels of ozone, PM_{10}, SO_2, and CO, and found a significant relationship for ozone and PM_{10} with both pollutants in the model; no significant effects were found for SO_2 and CO. The ozone coefficient is estimated from a model with PM_{10}.

The C-R function to estimate the change in short-term mortality associated with a change in ozone is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot \left(e^{-b \cdot \Delta O_3} - 1\right)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = ozone coefficient = 0.000634 (Ito, 1998)[52]
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of all ages
$_{\beta}$ = standard error of β = 0.000251 (Ito, 1998).

Incidence Rate. To estimate county-specific baseline mortality incidence, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

B.1.2 Short-Term Mortality (U.S.) (Kinney et al., 1995)

Kinney et al. (1995) examined the relationship between daily non-accidental mortality and air pollution levels in Los Angeles, California from 1985 to 1990. They examined ozone, PM_{10}, and CO, and found a significant relationship for each pollutant in single pollutant models. The effect for ozone dropped to zero with the inclusion of PM_{10} in the model, while the effect for CO and PM_{10} appeared independent of each other and were of a similar magnitude.

[52]The published paper has an incorrect coefficient and standard error; updated estimates were obtained from the author.

The C-R function to estimate the change in short-term mortality associated with a change in ozone is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = ozone coefficient = 0
ΔO_3 = change in daily 1-hour maximum ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000214.

Incidence Rate. To estimate county-specific baseline mortality incidence, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). In a model with PM_{10}, the ozone coefficient (β) for non-accidental mortality is estimated from the relative risk (1.00) associated with a change in daily one-hour maximum ozone of 143 ppb (Kinney et al., 1995, Table 2 and Figure 3):

$$b = \frac{\ln(1.00)}{(143)} = 0.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Kinney et al., 1995, Table 2 and Figure 3):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.06)}{143} - \frac{\ln(1.00)}{143}\right)}{1.96} = 0.000208$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.00)}{143} - \frac{\ln(0.94)}{143}\right)}{1.96} = 0.000221$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000214.$$

B.1.3 Short-Term Mortality (U.S.) (Moolgavkar et al., 1995)

Moolgavkar et al. (1995) examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1973 to 1988. They examined ozone, TSP, and SO_2 in a three-pollutant model, and found a significant relationship for ozone and SO_2; TSP was not significant.

The C-R function to estimate the change in short-term mortality associated with a change in ozone is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = ozone coefficient = 0.000611
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000216

Incidence Rate. To estimate county-specific baseline mortality incidence, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). Based on a model with TSP and SO_2, the coefficient (β) for non-accidental mortality is estimated from the relative risk (1.063) associated with a change in daily average ozone of 100 ppb (Moolgavkar et al., 1995, Table 5):

$$b = \frac{\ln(1.063)}{(100)} = 0.000611.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1995, Table 5):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.108)}{100} - \frac{\ln(1.063)}{100}\right)}{1.96} = 0.000212$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.063)}{100} - \frac{\ln(1.018)}{100}\right)}{1.96} = 0.000221$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000216.$$

B.1.4 Short-Term Mortality (U.S.) (Samet et al., 1997)

Samet et al. (1997) examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1974 to 1988. They examined ozone, TSP, SO_2, NO_2, and CO in a five-pollutant model, and found a significant relationship for each pollutant.

The C-R function to estimate the change in short-term mortality associated with a change in ozone is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = ozone coefficient = 0.000936
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000312

Incidence Rate. To estimate county-specific baseline mortality incidence, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). In a model with TSP, SO_2, NO_2, and CO, the ozone coefficient (β) for non-accidental mortality is estimated from the relative risk (1.0191) associated with a change in the two-day average ozone level of 20.219 ppb (Samet et al., 1997, Table 9):

$$b = \frac{\ln(1.0191)}{(20.219)} = 0.000936.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the reported t-value (t=3) (Samet et al., 1997, Table 9):

$$s_b = \frac{.000936}{3} = 0.000312.$$

B.2 Chronic Illness

In recent years, a number of studies have investigated the possible link between ozone and the development of chronic illness. Abbey et al. (1991; 1993) reported a significant link between ozone and the development of asthma, and Portney and Mullahy (1990) found ozone linked to sinusitis and hay fever. A review of research data by EPA (1996b, p. 9-35) concluded that prolonged ozone exposure causes structural changes in several regions of the respiratory tract, and the available epidemiological studies are suggestive of a link between chronic health effects in humans and long-term ozone exposure. Most recently, a study by McDonnell et al. (1999) carefully measured ozone exposure for Seventh Day Adventists living in California.

B.2.1 Asthma Adult Onset (McDonnell et al., 1999)

The McDonnell et al. (1999) study used the same cohort of Seventh-Day Adventists as Abbey et al. (1991; 1993), and examined the association between air pollution and the onset of asthma in adults

between 1977 and 1992. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures, controlling for other covariates; no significant effect was found between ozone exposure and asthma in females. No significant effect was reported for females or males due to exposure to PM, NO_2, SO_2, or SO_4.

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

$$\Delta Chronic\ Asthma = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta O_3 \cdot b} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = annual asthma incidence rate per person (McDonnell et al., 1999, Table 4) = 0.00219
β = estimated O_3 coefficient (McDonnell et al., 1999, Table 5) = 0.0277
ΔO_3 = change in annual average 8-hour O_3 concentration[53]
pop = population of non-asthmatic males ages 27 and older[54] = 96.66% of males 27+
$_{\beta}$ = standard error of β (McDonnell et al., 1999, Table 5) = 0.0135

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (32), dividing by the number of individuals in the sample (972), as reported by (McDonnell et al., 1999, Table 4), and then dividing by the 15 years in the sample.

B.3 Hospital Admissions

We estimate the impact of ozone on hospital admissions using a number of epidemiological studies. Most of the studies focus on the link between ozone and respiratory-related hospital admissions.

B.3.1 Hospital Admissions for Asthma (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for best fitting model were chosen using stepwise regression based on AIC criterion. Asthma admissions were linked to O_3, CO, and $PM_{2.5\text{-}10}$. This C-R function is based on the results of this three-pollutant model.

The C-R function to estimate the change in hospital admissions for asthma associated with daily changes in ozone is:

$$\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-b\cdot\Delta O_3} - 1)\right]\cdot pop,$$

[53]The eight-hour ozone concentration is defined as 9:00 A.M. to 4:59 P.M. The study used the 1973-1992 mean 8-hour average ambient ozone concentration (McDonnell et al., 1999, p. 113).

[54]The population weighted average incidence of asthma in males 27 and older is 3.34 percent. Population data from U.S. Census Bureau (1997, Table 14); asthma prevalence for males from Collins (1997, Table 9).

where:

y_0 = daily hospital admission rate for asthma per person = 4.75 E-6
β = ozone coefficient = 0.00250
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000718

Incidence Rate. Hospital admissions for obstructive lung disease (ICD-9 codes: 490-492, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.547 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 4.99 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0499. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0499)}{19.5} = 0.00250.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=3.48) (Burnett, 1999):

$$s_b = \frac{0.00250}{3.48} = 0.000718.$$

B.3.2 Hospital Admissions for Obstructive Lung Disease (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for the best fitting model were chosen using stepwise regression based on AIC criterion. Admissions for chronic obstructive pulmonary disease (COPD) were linked to O_3 and $PM_{2.5\text{-}10}$. This C-R function is based on the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for obstructive lung disease associated with daily changes in ozone is:

$$\Delta \textit{Obstructive Lung Disease Admissions} = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for obstructive lung disease per person = 5.76 E-6
β = ozone coefficient = 0.00303
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages

$_{\beta}$ = standard error of β = 0.00110

Incidence Rate. Hospital admissions for respiratory infection (ICD-9 codes: 464, 466, 480-487, 494) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (1.485 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 6.08 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0608. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0608)}{19.5} = 0.00303.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated using the t-value (t=2.74) (Burnett, 1999):

$$s_b = \frac{0.00303}{2.74} = 0.00110.$$

B.3.3 Hospital Admissions for Respiratory Infection (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from 1980 to 1994. They estimated multiple pollutant models, where pollutants for the best fitting model were chosen using stepwise regression based on AIC criterion. Respiratory infection admissions were linked to O_3, NO_2, and $PM_{2.5}$. This C-R function is based on the results from this three-pollutant model.

The C-R function to estimate the change in hospital admissions for respiratory infection associated with daily changes in ozone is:

$$\Delta\ Respiratory\ Infection\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for respiratory infection per person = 1.56 E-5
β = ozone coefficient = 0.00198
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_{\beta}$ = standard error of β = 0.000520

Incidence Rate. Hospital admissions for respiratory infections (ICD-9 codes: 464-466, 480-486, 490-494, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.452 million) divided by the 1994 population

(260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 3.93 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0393. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0393)}{19.5} = 0.00198 .$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated using the t-value (t=3.80) (Burnett, 1999):

$$s_b = \frac{0.00198}{3.80} = 0.000520 .$$

B.3.4 Hospital Admissions for All Respiratory (Burnett et al., 1997, Toronto)

Burnett et al. (1997) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada during the summers of 1992-1994. All respiratory admissions were linked to coefficient of haze (COH) and O_3; other PM measures were less strongly linked. In two pollutant models, they found that CO, NO_2, and SO_2 were not significant, controlling for COH. They found that O_3 was still significant, controlling for COH. This C-R function is based on the results from the four-pollutant model ($PM_{2.5-10}$, O_3, NO_2, and SO_2) to estimate all respiratory incidence.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in ozone is:

$$\Delta All\ Respiratory\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop ,$$

where:

y_0 = daily hospital admission rate for all respiratory admissions per person = 2.58 E-5
β = O_3 coefficient = 0.00498
ΔO_3 = change in daily 12-hour average O_3 concentration (ppb)[55]
pop = population of all ages
$_{\beta}$ = standard error of β = 0.00106

Incidence Rate. Hospital admissions for all respiratory causes (ICD-9 codes: 464-466, 480-486, 490-494, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.452 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

[55] Burnett et al. (1997, Table 2 and p. 614) reported using the daytime average ozone level from 8 A.M. to 8 P.M.

Coefficient Estimate (β). The estimated coefficient (β) is based on a relative risk of 1.059 due to a change of 11.50 ppb in the daily average for O_3 (Burnett et al., 1997, Tables 2 and 6). The coefficient is calculated as follows:

$$b = \frac{\ln(1.059)}{11.50} = 0.00498.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=4.71) (Burnett et al., 1997, Table 6)

$$s_b = \frac{.00498}{4.71} = 0.00106.$$

B.3.5 Hospital Admissions for All Respiratory (Thurston et al., 1994, Toronto)

Thurston et al. (1994) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada, for six weeks in July and August 1986-1988. In single-pollutant models, ozone and various measures of PM were linked to all respiratory admissions. In two-pollutant models, ozone was still significant, but measures of PM were often not significant; only H^+ was significant. This C-R function is based on the results of a two-pollutant model ($PM_{2.5}$ and ozone).

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in ozone is:

$$\Delta \, All\,Respiratory\,Admissions = b \cdot \Delta O_3 \cdot pop,$$

where:

β = ozone coefficient = 1.68 E-8
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 9.71 E-9 .

Coefficient Estimate (β). Based on a linear model with $PM_{2.5}$, the one-hour maximum ozone coefficient comes from an estimated coefficient of 0.0404, which estimates admissions per ppb of ozone (Thurston et al., 1994, Table 3).[56] The population of Toronto was estimated to be 2.4 million (U.S. EPA, 1997b, Table D-7). We estimated a coefficient estimating admissions per person per ppb of ozone as follows:

$$b = \frac{0.0404}{2{,}400{,}000} = 1.68E-8.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated in a similar fashion (Thurston et al., 1994, Table 3):

[56]The 812 Retrospective analysis (U.S. EPA, 1997b, Table D-7) used an ozone coefficient based on a model with PM_{10}.

$$s_b = \frac{0.0233}{2{,}400{,}000} = 9.71E-9.$$

B.3.6 Hospital Admissions for Pneumonia (Moolgavkar et al., 1997, Minneapolis)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a four pollutant model examining pneumonia admissions in Minneapolis, ozone was significant, while NO_2, SO_2, and PM_{10} were not significant. This C-R function is based on the results from the four-pollutant model to estimate pneumonia incidence.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in ozone is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.30 E-5
β = O_3 coefficient = 0.00370
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population of ages 65 and older
$_\beta$ = standard error of β = 0.00103

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 5.7 percent increase in admissions due to a O_3 change of 15 ppb (Moolgavkar et al., 1997, Table 4 and p. 366); the model with a 130 df smoother was reported to be optimal (p. 368). This translates to a relative risk of 1.057. The coefficient is calculated as follows:

$$b = \frac{\ln(1.057)}{15} = 0.00370.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.089)}{15} - \frac{\ln(1.057)}{15}\right)}{1.96} = 0.00101$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.057)}{15} - \frac{\ln(1.025)}{15}\right)}{1.96} = 0.00105$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00103.$$

B.3.7 Hospital Admissions for COPD (Moolgavkar et al., 1997, Minneapolis)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. No significant effect found for any pollutant; the effect for ozone was marginally significant. This C-R function is based on the results from a three-pollutant model (O_3, CO, PM_{10}) to estimate COPD incidence.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in ozone is:

$$\Delta\, COPD\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person = 3.75 E-5
β = O_3 coefficient = 0.00274
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population of ages 65 and older
$_\beta$ = standard error of β = 0.00170

Incidence Rate. Hospital admissions for COPD (ICD-9 codes: 490-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.454 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 4.2 percent increase in admissions due to a O_3 change of 15 ppb (Moolgavkar et al., 1997, Table 4 and p. 366); the model with a 100 df smoother was reported to be optimal (p. 368). This translates to a relative risk of 1.042. The coefficient is calculated as follows:

$$b = \frac{\ln(1.042)}{15} = 0.00274.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.094)}{15} - \frac{\ln(1.042)}{15}\right)}{1.96} = 0.00166$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.042)}{15} - \frac{\ln(0.99)}{15}\right)}{1.96} = 0.00174$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00170.$$

B.3.8 Hospital Admissions for Pneumonia (Schwartz, 1994a, Minneapolis)

Schwartz (1994a) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1989. In a two-pollutant model, Schwartz found PM_{10} significantly related to pneumonia; ozone was weakly linked to pneumonia. This C-R function is based on the results of the two-pollutant model (PM_{10}, O_3) to estimate pneumonia incidence.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in ozone is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.30 E-5
β = O_3 coefficient = 0.00280
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population of ages 65 and older
$_\beta$ = standard error of β = 0.00172

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with ozone, the coefficient (β) is estimated from the relative risk (1.15) associated with a 50 ppb change in the daily average ozone level (Schwartz, 1994a, Table 4 and p. 369):

$$b = \frac{\ln(1.15)}{50} = 0.00280.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994a, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.36)}{50} - \frac{\ln(1.15)}{50}\right)}{1.96} = 0.00171$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.15)}{50} - \frac{\ln(0.97)}{50}\right)}{1.96} = 0.00174$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00172 .$$

B.3.9 Hospital Admissions for Pneumonia (Schwartz, 1994b, Detroit)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD; no significant link to asthma admissions was found for either pollutant. We use the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in ozone is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.18 E-5
β = O_3 coefficient (Schwartz, 1994b, Table 4) = 0.00521
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population of ages 65 and older
$_\beta$ = standard error of β (Schwartz, 1994b, Table 4) = 0.0013

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-486) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.627 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

B.3.10 Hospital Admissions for COPD (Schwartz, 1994b, Detroit)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant

model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD; no significant link to asthma admissions was found for either pollutant. We use the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in O_3 is:

$$\Delta \, COPD \, Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person = 3.05 E-5
β = O_3 coefficient (Schwartz, 1994b, Table 4) = 0.00549
ΔO_3 = change in daily average O_3 concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β (Schwartz, 1994b, Table 4) = 0.00205

Incidence Rate. Hospital admissions for COPD (ICD-9 codes: 491-492, 494-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.369 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

B.3.11 Hospital Admissions for All Respiratory (Schwartz, 1995, New Haven)

Schwartz (1996) examined the relationship between air pollution and hospital admissions for individuals 65 and older in New Haven, Connecticut, from January 1988 to December 1990. In single-pollutant models, PM_{10} and SO_2 were significant, while ozone was marginally significant. In two-pollutant models, ozone was significant in one of two models, and had stable coefficient estimates; PM_{10} was significant in two of two models, but had less stable estimates. SO_2 was significant in one of four models. The C-R function in this analysis is based on a two-pollutant model with ozone and PM_{10}.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in ozone is:

$$\Delta \, All \, Respiratory \, Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory conditions per person 65 and older = 1.187 E-4
β = ozone coefficient = 0.00265
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.00140

Incidence Rate. All respiratory hospital admissions (ICD-9 codes: 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years

and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with PM_{10}, the coefficient (β) is estimated from the relative risk (1.07) associated with a change in ozone exposure of 50 μg/m^3 (Schwartz, 1995, Table 3 and p. 535):[57]

$$b = \frac{\ln(1.07)}{\left(\frac{50}{1.96}\right)} = 0.00265.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1995, Table 3).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.15)}{50/1.96} - \frac{\ln(1.07)}{50/1.96}\right)}{1.96} = 0.00144$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.07)}{50/1.96} - \frac{\ln(1.00)}{50/1.96}\right)}{1.96} = 0.00135$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00140.$$

B.3.12 Hospital Admissions for All Respiratory (Schwartz, 1995, Tacoma)

Schwartz (1996) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Tacoma, Washington, from January 1988 to December 1990. In single-pollutant models, PM_{10}, ozone, and SO_2 were all significant. In two-pollutant models, ozone was significant in two of two models, and had stable coefficient estimates; PM_{10} was significant in one of two models, but had less stable estimates; SO_2 was not significant in either of the two-pollutant models. The C-R function in this analysis is based on a two-pollutant model with ozone and PM_{10}.

The C-R function to estimate the change in hospital admissions for all-respiratory causes associated with daily changes in ozone is:

[57] A conversion of 1.96 μg/m^3 per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million μg in a gram, this density means that there are 1.96 billion μg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 μg of ozone (i.e., one ppb = 1.96 μg/m^3).

$$\Delta \, All\ Respiratory\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory conditions per person 65 and older = 1.187 E-4
β = ozone coefficient = 0.00715
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.00257

Incidence Rate. All respiratory hospital admissions (ICD-9 codes: 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with PM_{10}, the coefficient (β) is estimated from the relative risk (1.20) associated with a change in ozone exposure of 50 µg/m^3 (Schwartz, 1995, Table 6 and p. 535):[58]

$$b = \frac{\ln(1.20)}{\left(\frac{50}{1.96}\right)} = 0.00715.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1995, Table 6):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.37)}{50/1.96} - \frac{\ln(1.20)}{50/1.96}\right)}{1.96} = 0.00265$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.20)}{50/1.96} - \frac{\ln(1.06)}{50/1.96}\right)}{1.96} = 0.00248$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00257.$$

[58] A conversion of 1.96 µg/m^3 per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million µg in a gram, this density means that there are 1.96 billion µg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 µg of ozone (i.e., one ppb = 1.96 µg/m^3).

B.3.13 Hospital Admissions for Dysrhythmias (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for best fitting model were chosen using stepwise regression based on AIC criterion. Dysrhythmias admissions were linked to O_3, CO, and $PM_{2.5}$. This C-R function is based on the results of this three-pollutant model.

The C-R function to estimate the change in hospital admissions for dysrhythmias associated with daily changes in ozone is:

$$\Delta Dysrhythmias\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for dysrhythmias per person = 6.46 E-6
β = ozone coefficient = 0.00168
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.00103

Incidence Rate. Hospital admissions for dysrhthmias (ICD-9 code: 427) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.614 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 3.34 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0334. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0334)}{19.5} = 0.00168.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=1.63) (Burnett, 1999):

$$s_b = \frac{0.00168}{1.63} = 0.00103.$$

B.4 Emergency Room Visits

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

hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

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

B.4.1 Emergency Room Visits for Asthma (Cody et al., 1992, Northern NJ)

Cody et al. (1992) examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1988-1989. In a two pollutant model, ozone was linked to asthma visits, and no effect was seen for SO_2. PM_{10} considered in separate analysis, because of limited (every sixth day) sampling; no significant effect was seen for PM_{10}.

The C-R function to estimate the change in asthma ER visits associated with daily changes in ozone is:

$$\Delta\, Asthma\, ERVisits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop \cdot (1 - 0.37),$$

where:
β = ozone coefficient (Cody et al., 1992, Table 6) = 0.0203
BasePop = baseline population in northern New Jersey[59] = 4,436,976
ΔO_3 = change in daily five-hour average ozone concentration (ppb)[60]
pop = population of all ages
$_{\beta}$ = standard error of β (Cody et al., 1992, Table 6) = 0.00717

Correction for Double Counting. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. This percentage is then subtracted from the estimated change in asthma-related ER visits.

B.4.2 Emergency Room Visits for Asthma (Weisel et al., 1995, Northern NJ)

Weisel et al. (1995) examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1986-1990. A significant relationship was reported for ozone.

[59]The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. (1992, Figure 1) presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

[60]The coefficients in the study were based on the five-hour (10:00 am to 2:59 pm) ozone average in ppm; they have been converted to ppb.

The C-R function to estimate the change in asthma ER visits associated with daily changes in ozone is:

$$\Delta\, Asthma\, ERVisits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop \cdot (1 - 0.37),$$

where:

β = ozone coefficient = 0.0443
BasePop = baseline population in northern New Jersey[61] = 4,436,976
ΔO_3 = change in daily five-hour average ozone concentration (ppb)[62]
pop = population of all ages
s_β = standard error of β = 0.00723

Correction for Double Counting. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. This percentage is then subtracted from the estimated change in asthma-related ER visits.

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Weisel et al. (1995, Table 2) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{s_{b_i}^2}} \right) = 0.0443.$$

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

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{s_{b_i}^2}} \right) = \left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{g} \right) = \sum_{i=1986}^{1990} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g} \right).$$

This eventually reduces down to:

[61]The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. (1992, Figure 1) presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

[62]The coefficients in the study were based on the five-hour (10:00 am to 2:59 pm) ozone average in ppm; they have been converted to ppb.

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00723.$$

B.4.3 Emergency Room Visits for Asthma (Stieb et al., 1996, New Brunswick)

Stieb et al. (1996) examined the relationship between ER visits and air pollution for persons of all ages in St. John, New Brunswick, Canada, from May through September in 1984-1992. Ozone was significantly linked to ER visits, especially when ozone levels exceeded 75 ppb.

The C-R function to estimate the change in asthma ER visits associated with daily changes in ozone is:

$$\Delta\, Asthma\, ERVisits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop \cdot (1 - 0.37),$$

where:

β	= ozone coefficient (Stieb et al., 1996, Table 2 linear model) = 0.0035
BasePop	= baseline population in Saint John, New Brunswick (Stieb et al., 1996, p. 1354) = 125,000
ΔO_3	= change in the daily one-hour maximum ozone concentration (ppb)
pop	= population of all ages
$_{\beta}$	= standard error of β (Stieb et al., 1996, Table 2 linear model) = 0.0018

Correction for Double Counting. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. This percentage is then subtracted from the estimated change in asthma-related ER visits.

B.5 Acute Morbidity

B.5.1 Asthma Attacks: Whittemore and Korn (1980)

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants (O_x). Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks. The results from this model were used, and the oxidant result was adjusted below so it may be used with ozone data.

The C-R function to estimate the change in asthma attacks associated with a change in daily ozone is:

$$\Delta Asthma\ Attacks = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta O_3 \cdot b} + y_0} - y_0\right] \cdot pop,$$

where:

y_0 = daily incidence of asthma attacks = 0.027 (Krupnick, 1988, p. 4-6)
β = ozone coefficient = 0.00184
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of asthmatics of all ages = 5.61% of the population of all ages (Adams and Marano, 1995 Table 57).
$_{\beta}$ = standard error of β = 0.000714

Incidence Rate. The annual rate of 9.9 asthma attacks per astmatic is divided by 365 to get a daily rate. A figure of 9.9 is roughly consistent with the recent statement that "People with asthma have more than 100 million days of restricted activity" each year (National Heart, 1997). This 100 million incidence figure coupled with the 1996 population of 265,557,000 (U.S. Bureau of the Census, 1997, Table 2) and the latest asthmatic prevalence rate of 5.61% (Adams and Marano, 1995, Table 57), suggest an annual asthma attach rate per asthmatic of 6.7.

Coefficient Estimate (β). Based on a model with TSP, the daily one-hour ozone coefficient is based on an oxidant coefficient (1.66) estimated from data expressed in ppm (Whittemore and Korn, 1980, Table 5):[63]

$$b = \frac{1.66 \cdot 1.11}{1000} = 0.00184.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

$$s_b = \frac{b}{t} = \frac{0.184}{2.576} = 0.000714.$$

[63]The study used oxidant measurements in ppm (Whittemore and Korn, 1980, p. 688); these have been converted to ozone measurements in ppb, assuming ozone comprises 90% of oxidants (i.e., 1.11*ozone=oxidant). It is assumed that the harm of oxidants is caused by ozone. The view expressed in the Ozone Staff Paper (U.S. EPA, 1996a, p.164) is consistent with assuming that ozone is the oxidant of concern at normal ambient concentrations: "Further, among the photochemical oxidants, the acute-exposure chamber, field, and epidemiological human health data base raises concern only for O_3 at levels of photochemical oxidants commonly reported in ambient air. Thus, the staff recommends that O_3 remain as the pollutant indicator for protection of public health from exposure to all photochemical oxidants found in the ambient air."

B.5.2 Minor Restricted Activity Days: Ostro and Rothschild (1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average O_3 has highly variable association with RRADs and MRADs. Controlling for O_3, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.

The study is based on a "convenience" sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994d, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994b; Schwartz, 1994c).

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient used in this analysis is a weighted average of the coefficients in Ostro and Rothschild (1989), Table 4, using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) associated with a change in daily O_3 is:

$$\Delta MRAD = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily MRAD daily incidence rate per person = 0.02137
β = inverse-variance weighted O_3 coefficient = 0.00220
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)[64]
pop = adult population aged 18 to 65
$_{\beta}$ = standard error of β = 0.000658

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

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight:[65]

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.00220.$$

[64]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation. The study used ozone measurements in $\mu g/m^3$; a conversion of 1.96 $\mu g/m^3$ = 1 ppb is assumed here.

[65]The calculation of the MRAD coefficient and its standard error is exactly analogous to the calculation done for the work-loss days coefficient based on Ostro (1987).

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

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g}\right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g}\right).$$

This reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.000658.$$

B.5.3 Worker Productivity: Crocker and Horst (1981)

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst (1981) and summarized in EPA (1994). Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration (-0.1427).[66] The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income. Given the median daily income for outdoor workers engaged in strenuous activity reported by the 1990 U.S. Census, $89.64 per day (1997$), a ten percent reduction in ozone yields about $1.26 in increased daily wages. The median daily income for outdoor workers is a national estimate, however. We adjust this estimate to reflect regional variations in income using a factor based on the ratio of national median household income divided by a county's median household income. No information was available for quantifying the uncertainty associated with the central valuation estimate. Therefore, no uncertainty analysis was conducted for this endpoint.

B.5.4 Any of 19 Respiratory Symptoms: Krupnick (1990)

Krupnick et al. (1990) estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. (1990) listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

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

In their analysis, they included coefficient of haze (COH, a measure of particulate matter concentrations), ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily O_3, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

The C-R function used to estimate the change in ARD2 associated with a change in daily one-hour maximum ozone is based on Krupnick et al. (1990, p. 12):[67]

$$\Delta ARD2 \cong b^{*} \cdot \Delta O_3 \cdot pop\ ,$$

where:

β^*	=	first derivative of the stationary probability = 0.000137
ΔO_3	=	change in daily one-hour maximum ozone concentration (ppb)[68]
pop	=	population aged 18-65 years old[69]
$_{\beta}$	=	standard error of β^* = 0.0000697

Coefficient Estimate (β^*). The logistic regression model used by Krupnick et al. (1990) takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$p_i = probability(ARD2 | sickness\ or\ not_{t-1}) = \frac{1}{1 - e^{b_0 + b_1 \cdot ARD2_{t-1} + X \cdot b}}\ , for\ i = 0,1.$$

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

[67]Krupnick and Kopp (1988, p. 2-24) and ESEERCO (1994, p. V-32) used the same C-R functional form as that used here.

[68]Krupnick et al. (1990) used parts per hundred million (pphm) to measure ozone; the coefficient used here is based on ppb.

[69]The coefficient estimates are based on the sample of "adults," and assumes that individuals 18 and older were considered adult. According to Krupnick et al. (1990, Table 1), about 0.6 percent of the study sample was over the age of 60. This is a relatively small fraction, so it is further assumed that the results do not apply to individuals over the age of 65.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of ozone (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in ozone and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before} .$$

However the full suite of coefficient estimates are not available.[70] Rather than use the full suite of coefficient values, the impact of ozone on the probability of ARD2 may be approximated by the derivative of ARD2 with respect to ozone:[71]

$$\frac{\partial probability(ARD2)}{\partial O_3} = \frac{p_0 \cdot (1 - p_1) \cdot b \cdot [p_1 + (1 - p_0)]}{(1 - p_1 + p_0)^2} = b^* ,$$

where β is the reported logistic regression coefficient for ozone. The change in the incidence of ARD2 associated with a given change in ozone is then estimated by:

$$\frac{\partial ARD2}{\partial O_3} \cong \frac{\Delta ARD2}{\Delta O_3}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta O_3} \cong b^*$$

$$\Rightarrow \Delta ARD2 \cong b^* \cdot \Delta O_3 .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO (1994, p. V-32) for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$b^* = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00055 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000137 .$$

[70]The model without NO_2 (Krupnick et al., 1990, Table V equation 3) was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., Table V). Both the ozone and COH coefficients dropped by about a factor of two or more.

[71]The derivative result is reported by Krupnick et al. (1990, p. 12).

Standard Error ($_{\beta}$). The standard error for the coefficient ($_{\beta}$) is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. (1990, Table V):

$$b_{high} = 0.00055 + (1.96 \cdot 0.00027) = 0.00108$$

$$\Rightarrow b^{*}_{high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00108 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000268$$

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{(0.000268 - 0.000137)}{1.96} = 0.0000668$$

$$b_{low} = 0.00055 - (1.96 \cdot 0.00027) = 0.0000208$$

$$\Rightarrow b^{*}_{low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.0000208 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 5.17 \cdot 10^{-6}$$

$$\Rightarrow s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(0.000137 + 5.17 \cdot 10^{-6}\right)}{1.96} = 0.0000725$$

$$s_{b} = \frac{s_{b,high} + s_{b,low}}{2} = 0.0000697 .$$

Appendix C: Particulate Matter C-R Functions

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

C.1 Mortality

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

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

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

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

where:

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

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

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

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

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

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.19)}{24.5} - \frac{\ln(1.12)}{24.5}\right)}{1.96} = 0.0012625$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.12)}{24.5} - \frac{\ln(1.06)}{24.5}\right)}{1.96} = 0.0011466$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0012046$$

C.1.2 Mortality (Krewski et al., 2000), Based on ACS Cohort: Median $PM_{2.5}$

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

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

where:
y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.0053481
$\Delta PM_{2.5}$ = change in annual median $PM_{2.5}$ concentration
pop = population of ages 30 and older
$_\beta$ = standard error of β = 0.0014638

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

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.14) associated with a change in median exposure of 24.5 $\mu g/m^3$ (based on original ACS study) (Krewski et al., 2000, Part II - Table 31):

$$b = \frac{\ln(1.14)}{(24.5)} = 0.0053481.$$

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

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.22)}{24.5} - \frac{\ln(1.14)}{24.5}\right)}{1.96} = 0.0014124$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.14)}{24.5} - \frac{\ln(1.06)}{24.5}\right)}{1.96} = 0.0015152$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0014638$$

C.1.3 Mortality (Krewski et al., 2000), Based on ACS Cohort, Random Effects with Regional Adjustment: Median $PM_{2.5}$

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

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

where:
y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.00605796
$\Delta PM_{2.5}$ = change in annual median $PM_{2.5}$ concentration
pop = population of ages 30 and older
$_\beta$ = standard error of β = 0.0033826

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

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.16) associated with a change in median exposure of 24.5 $\mu g/m^3$ (based on original ACS study) (Krewski et al., 2000, Part II - Table 46):

$$b = \frac{ln(1.16)}{(24.5)} = 0.00605796.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II - Table 46):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{ln(1.37)}{24.5} - \frac{ln(1.16)}{24.5}\right)}{1.96} = 0.0034650$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{ln(1.16)}{24.5} - \frac{ln(0.99)}{24.5}\right)}{1.96} = 0.0033001$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0033826$$

C.1.4 Mortality (Krewski et al., 2000), Based on ACS Cohort, Random Effects with Independent Cities: Median $PM_{2.5}$

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

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

where:

y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.0103936
$\Delta PM_{2.5}$ = change in annual median $PM_{2.5}$ concentration
pop = population of ages 30 and older
s_β = standard error of β = 0.0029021

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

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.29) associated with a change in median exposure of 24.5 $\mu g/m^3$ (based on original ACS study) (Krewski et al., 2000, Part II - Table 46):

$$b = \frac{ln(1.29)}{(24.5)} = 0.0103936.$$

Standard Error (s_β). The standard error (s_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II - Table 46):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{ln(1.48)}{24.5} - \frac{ln(1.29)}{24.5}\right)}{1.96} = 0.0028613$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{ln(1.29)}{24.5} - \frac{ln(1.12)}{24.5}\right)}{1.96} = 0.0029428$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0029021$$

C.1.5 Mortality (Pope et al., 1995), Based on ACS Cohort: Median $PM_{2.5}$

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

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

where:

y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.006408
$\Delta PM_{2.5}$ = change in annual <u>median</u> $PM_{2.5}$ concentration
pop = population of ages 30 and older
s_β = standard error of β = 0.001509

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

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.17) associated with a change in median exposure going from 9 $\mu g/m^3$ to 33.5 $\mu g/m^3$ (Pope et al., 1995, Table 2).

$$b = \frac{\ln(1.17)}{(33.5 - 9)} = 0.006408.$$

Standard Error (s_β). The standard error (s_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Pope et al., 1995, Table 2).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.26)}{24.5} - \frac{\ln(1.17)}{24.5}\right)}{1.96} = 0.001543$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.17)}{24.5} - \frac{\ln(1.09)}{24.5}\right)}{1.96} = 0.001475$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.001509 .$$

C.1.6 Mortality (Krewski et al., 2000), Based on Six-City Cohort: Mean $PM_{2.5}$

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

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

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

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

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

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

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

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.48)}{18.6} - \frac{\ln(1.28)}{18.6}\right)}{1.96} = 0.003982$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.28)}{18.6} - \frac{\ln(1.10)}{18.6}\right)}{1.96} = 0.004157$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.004070$$

C.1.7 Mortality (Dockery et al., 1993), Based on Six-City Cohort: Mean $PM_{2.5}$

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

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

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

where:

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

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

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

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

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

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.47)}{18.6} - \frac{\ln(1.26)}{18.6}\right)}{1.96} = 0.00423$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.26)}{18.6} - \frac{\ln(1.08)}{18.6}\right)}{1.96} = 0.00423$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00423.$$

C.1.8 Short-Term Mortality (Schwartz et al., 1996)

Schwartz et al. (1996) pooled the results from six cities in the U.S. and found a significant relationship between daily $PM_{2.5}$ concentration and non-accidental mortality.[72] Abt Associates Inc. (1996b, p. 52) used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to reestimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error (Abt Associates Inc., 1996a, p. 65).

The C-R function to estimate the change in mortality associated with daily changes in $PM_{2.5}$ is:

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

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = $PM_{2.5}$ coefficient (Abt Associates Inc., 1996a, Exhibit 7.2) = 0.001433
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of all ages
$_\beta$ = standard error of β (Abt Associates Inc., 1996a, Exhibit 7.2) = 0.000129

C.1.9 Neonatal Mortality (Woodruff et al., 1997)

[72]Schwartz et al. (1996, p. 929) defined non-accidental mortality as all-cause mortality less deaths due to accidents and other external causes (ICD-9 codes: 800-999). Other external causes includes suicide, homicide, and legal intervention (National Center for Health Statistics, 1994).

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

In addition to the work by Woodruff et al., work in Mexico City (Loomis et al., 1999), the Czech Republic (Bobak and Leon, 1992), Sao Paulo (Saldiva et al., 1994; Pereira et al., 1998), and Beijing (Wang et al., 1997) provides additional evidence that particulate levels are significantly related to infant or child mortality, low birth weight or intrauterine mortality.

Conceptually, neonatal or child mortality could be added to the premature mortality predicted by Pope et al. (1995), because the Pope function covers only the population over 30 years old.[73] However, the EPA Science Advisory Board recently advised the Agency not to include post-neonatal mortality in this analysis because the study is of a new endpoint and the results have not been replicated in other studies (U.S. EPA, 1999a, p. 12). The estimated avoided incidences of neonatal mortality are estimated and presented as a sensitivity analysis, and are not included in the primary analysis.

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

$$\Delta Infant\ Mortality = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = county annual postneonatal[74] infant deaths per infant under the age of one
β = PM_{10} coefficient = 0.00392
ΔPM_{10} = change in annual average PM_{10} concentration[75]
pop = population of infants under one year old
$_{\beta}$ = standard error of β = 0.00122

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.04) associated with a 10 $\mu g/m^3$ change in PM_{10} (Woodruff et al., 1997, Table 3). The coefficient is calculated as follows:

$$b_{PM_{10}} = \frac{\ln(1.04)}{10} = 0.00392\ .$$

Standard Error ($_{\beta}$). The standard error for the coefficient is calculated as the average of the standard errors implied by the reported lower and upper bounds of the odds ratio (1.02 to 1.07) (Woodruff et al., 1997, Table 3). This reproduces both the lower and upper bounds of the odds ratio:

[73] Predicted neonatal mortality could not be added to the premature mortality predicted by the daily (short-term exposure) mortality studies, however, because these studies cover all ages.

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

[75] Woodruff et al. (1997) used PM_{10} exposure in the first two months of an infant's life.

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.07)}{10} - \frac{\ln(1.04)}{10}\right)}{1.96} = 0.001451$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.04)}{10} - \frac{\ln(1.02)}{10}\right)}{1.96} = 0.000991$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00122 .$$

C.2 Chronic Morbidity

Schwartz (1993) and Abbey et al. (1993; 1995b) provide evidence that PM exposure over a number of years gives rise to the development of chronic bronchitis in the U.S., and a recent study by McDonnell et al. (1999) provides evidence that ozone exposure is linked to the development of asthma in adults. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998).[76]

We estimate the changes in the new cases of chronic bronchitis by pooling the estimates from the studies by Schwartz (1993) and Abbey et al. (1995b). The Schwartz study is somewhat older and uses a cross-sectional design, however, it is based on a national sample, unlike the Abbey et al. study which is based on a sample of California residents.

C.2.1 Chronic Bronchitis (Schwartz, 1993)

Schwartz (1993) examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1974 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz (1993, Table 3) reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the prevalence of reported chronic bronchitis, asthma, shortness of breath (dyspnea) and respiratory illness[77], and the annual levels of TSP, collected in the year prior to the survey (TSP was the only pollutant examined in this study). TSP was significantly related to the prevalence of chronic bronchitis, and marginally significant for respiratory illness. No effect was found for asthma or dyspnea.

[76] There are a limited number of studies that have estimated the impact of air pollution on chronic bronchitis. An important hindrance is the lack of health data and the associated air pollution levels over a number of years.

[77] Respiratory illness defined as a significant condition, coded by an examining physician as ICD-8 code 460-519.

Schwartz (1993) examined the *prevalence* of chronic bronchitis, not its *incidence*. To use Schwartz's study and still estimate the change in incidence, there are at least two possible approaches. The first is to simply assume that it is appropriate to use the baseline *incidence* of chronic bronchitis in a C-R function with the estimated coefficient from Schwartz's study, to directly estimate the change in incidence. The second is to estimate the percentage change in the prevalence rate for chronic bronchitis using the estimated coefficient from Schwartz's study in a C-R function, and then to assume that this percentage change applies to a baseline incidence rate obtained from another source. (That is, if the prevalence declines by 25 percent with a drop in PM, then baseline incidence drops by 25 percent with the same drop in PM.) This analysis is using the latter approach, and estimates a percentage change in prevalence which is then applied to a baseline incidence rate.

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

$$\Delta Chronic\ Bronchitis = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot\left[\frac{z_0}{y_0}\right]\cdot pop\ ,$$

where:

y_0 = national chronic bronchitis prevalence rate for individuals 18 and older (Adams and Marano, 1995, Table 62 and 78) = 0.0535

z_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378

β = estimated PM_{10} logistic regression coefficient = 0.0123

ΔPM_{10} = change in annual average PM_{10} concentration

pop = population of ages 30 and older without chronic bronchitis = 0.9465*population 30+

$_{\beta}$ = standard error of β = 0.00434 .

Prevalence Rate. The national chronic bronchitis prevalence rate was not available for individuals 30 and older. Instead, we used the prevalence rate for individuals 18 and older (Adams and Marano, 1995, Table 62 and 78). The 1994 national figures are the latest available, and are suggested here.

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

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.07) associated with 10 $\mu g/m^3$ change in TSP (Schwartz, 1993, p. 9). Assuming that PM_{10} is 55 percent of TSP[78] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b_{PM_{10}} = \frac{ln(1.07)}{0.55\cdot 10} = 0.0123.$$

[78]The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).

Standard Error (β) The standard error for the coefficient (β) is calculated from the reported lower and upper bounds of the odds ratio (1.02 to 1.12) (Schwartz, 1993, p. 9):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.12)}{0.55 \cdot 10} - \frac{\ln(1.07)}{0.55 \cdot 10}\right)}{1.96} = 0.00424$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.07)}{0.55 \cdot 10} - \frac{\ln(1.02)}{0.55 \cdot 10}\right)}{1.96} = 0.00444$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00434 .$$

Population. The study population in Schwartz (1993) includes 3,874 individuals over the age of 30, living in 57 urban areas in the United States. To what extent the study should be applied to individuals under the age of 30 is unclear, and no effect is assumed for these individuals.

C.2.2 Chronic Bronchitis (Abbey et al., 1995b, California)

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

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

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

where:

y_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0132
$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration
pop = population of ages 27 and older without chronic bronchitis[79] = 0.9465*population 27+
$_{\beta}$ = standard error of β = 0.00680

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

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

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

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

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(3.25)}{45} - \frac{\ln(1.81)}{45}\right)}{1.96} = 0.00664$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.81)}{45} - \frac{\ln(0.98)}{45}\right)}{1.96} = 0.00696$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00680.$$

C.3 Hospital Admissions

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

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

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

C.3.1 Hospital Admissions for COPD (Samet et al., 2000a, 14 Cities)

Samet et al. (2000a) examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[80] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[81] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically

[80]The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

[81] Joel Schwartz (co-author), personal communication.

impact the results.[82] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

The C-R function to estimate the change in hospital admissions for COPD[83] associated with daily changes in PM_{10} is:

$$\Delta COPD\,Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for COPD per person 65 and older = 3.12 E-5
β	= PM_{10} coefficient = 0.00288
ΔPM_{10}	= change in daily average PM_{10} concentration
pop	= population age 65 and older
$_{\beta}$	= standard error of β = 0.00139

Incidence Rate. COPD hospital admissions (ICD-9 codes: 490-492, 494-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.378 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 2.88 percent increase in admissions due to a PM_{10} change of 10.0 $\mu g/m^3$ (Samet et al., 2000a, Part II - Table 14)[84]. This translates to a relative risk of 1.029. The coefficient is calculated as follows:

$$b = \frac{\ln(1.029)}{10.0} = 0.00288.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000a, Part II - Table 14):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{0.0564}{10} - \frac{0.0288}{10}\right)}{1.96} = 0.00141$$

[82] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

[83] ICD-9 codes 490-492 and 494-496.

[84] The random effects estimate of the unconstrained distributed lag model was chosen for COPD admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{0.0288}{10} - \frac{0.0019}{10}\right)}{1.96} = 0.00137$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00139.$$

C.3.2 Hospital Admissions for Pneumonia (Samet et al., 2000a, 14 Cities)

The C-R function to estimate the change in hospital admissions for pneumonia[85] associated with daily changes in PM_{10} is:

$$\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for pneumonia per person 65 and older = 5.30 E-5
β	= PM_{10} coefficient = 0.00207
ΔPM_{10}	= change in daily average PM_{10} concentration
pop	= population age 65 and older
$_{\beta}$	= standard error of β = 0.00058

Incidence Rate. Congestive heart failure hospital admissions (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

[85] ICD-9 codes 480-487.

Coefficient Estimate (β). The estimated coefficient (β) is based on a 2.07 percent increase in admissions due to a PM_{10} change of 10.0 μg/m^3 (Samet et al., 2000a, Part II - Table 14)[86]. This translates to a relative risk of 1.021. The coefficient is calculated as follows:

$$b = \frac{\ln(1.021)}{10.0} = 0.00207 .$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000a, Part II - Table 14):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{0.0322}{10} - \frac{0.0207}{10}\right)}{1.96} = 0.00059$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{0.0207}{10} - \frac{0.0094}{10}\right)}{1.96} = 0.00058$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00058 .$$

C.3.3 Hospital Admissions for Asthma (Sheppard et al., 1999, Seattle)

Sheppard et al. (1999) studied the relation between air pollution in Seattle and nonelderly hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM_{2.5\text{-}10}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects. They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, coarse $PM_{2.5\text{-}10}$, 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 model was found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. The C-R function in this analysis is based on a two-pollutant model with CO and $PM_{2.5}$.

[86] The random effects estimate of the unconstrained distributed lag model was chosen for pneumonia admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

The C-R function to estimate the change in hospital admissions for asthma associated with daily changes in $PM_{2.5}$ is:

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

where:

y_0 = daily hospital admission rate for asthma per person = 4.52 E-6
β = $PM_{2.5}$ coefficient = 0.00227
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of ages less than 65
$_\beta$ = standard error of β = 0.000948

Incidence Rate. Hospital admissions for asthma (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.375 million) divided by the 1994 population (227.210 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with CO, the daily average coefficient (β) is estimated from the relative risk (1.03) associated with a change in $PM_{2.5}$ exposure over the interquartile range of 8 to 21 $\mu g/m^3$ (Sheppard et al., 1999, Table 3 and p. 28):

$$b = \frac{\ln(1.03)}{13} = 0.00227.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Sheppard et al., 1999, p. 28):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.06)}{13} - \frac{\ln(1.03)}{13}\right)}{1.96} = 0.00113$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.03)}{13} - \frac{\ln(1.01)}{13}\right)}{1.96} = 0.000770$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000948.$$

C.3.4 Hospital Admissions for Cardiovascular Disease (Samet et al., 2000a, 14 Cities)

The C-R function to estimate the change in hospital admissions for cardiovascular disease[87] associated with daily changes in PM_{10} is:

$$\Delta CVD\,Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for cardiovascular disease per person 65 and older = 2.23 E-4
β	= PM_{10} coefficient = 0.00119
ΔPM_{10}	= change in daily average PM_{10} concentration
pop	= population age 65 and older
s_β	= standard error of β = 0.00011

Incidence Rate. Congestive heart failure hospital admissions (ICD-9 codes: 390-429) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.695 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 1.19 percent increase in admissions due to a PM_{10} change of 10.0 $\mu g/m^3$ (Samet et al., 2000a, Part II - Table 14)[88]. This translates to a relative risk of 1.012. The coefficient is calculated as follows:

$$b = \frac{\ln(1.012)}{10.0} = 0.00119.$$

Standard Error (s_β). The standard error (s_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000a, Part II - Table 14):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{0.0141}{10} - \frac{0.0119}{10}\right)}{1.96} = 0.00011$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{0.0119}{10} - \frac{0.0097}{10}\right)}{1.96} = 0.00011$$

[87] ICD-9 codes 390-429.

[88] The fixed effects estimate of the unconstrained distributed lag model was chosen for CVD admissions since the chi-square test of heterogeneity was non-significant (see Samet et al., 2000, Part II - Table 15).

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00011.$$

C.4 Emergency Room Visits

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

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

C.4.1 Emergency Room Visits for Asthma (Schwartz et al., 1993, Seattle)

Schwartz et al. (1993) examined the relationship between air quality and emergency room visits for asthma in persons under 65 and 65 and over, living in Seattle from September 1989 to September 1990. Using single-pollutant models they found daily levels of PM_{10} linked to ER visits in individuals ages under 65, and they found no effect in individuals ages 65 and over. They did not find a significant effect for SO_2 and ozone in either age group. The results of the single pollutant model for PM_{10} are used in this analysis.

The C-R function to estimate the change in daily emergency room visits for asthma associated with daily changes in PM_{10} is:

$$\Delta\ Asthma\ ER visits = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily ER visits for asthma per person under 65 years old = 7.69 E-6
β = PM_{10} coefficient (Schwartz et al., 1993, p. 829) = 0.00367
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 0-64
$_{\beta}$ = standard error of β (Schwartz et al., 1993, p. 829) = 0.00126

Incidence Rate. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. As described below, the 1994 asthma admission rate for people less than 65 is 4.522 E-6. So one might assume, ER visits = (1/0.37)*asthma admission rate = 2.7*asthma admission rate = 1.22 E-5. Now, ER visits (subtracting out those visits that end up as admissions)= 1.7*asthma admission rate = 7.69 E-6.

Asthma hospital admissions (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.375 million) divided by the 1994 population of individuals under 65 years old (227.21 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

C.5 Acute Morbidity

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

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

C.5.1 Acute Bronchitis C-R Function (Dockery et al., 1996)

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.1}$ and PM_{10} were marginally significantly related to bronchitis.[89] 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.

[89] 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.

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

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

where:

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

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

In 1994, 2,115,000 children ages 5-17 experienced acute conditions (Adams and Marano, 1995, Table 6) out of population of 48.110 million children ages 5-17 (U.S. Bureau of the Census, 1998, Table 14), or 4.4 percent of this population. This figure is somewhat lower than the 5.34 percent of children under the age of 18 reported to have chronic bronchitis in 1990-1992 (Collins, 1997, Table 8). Dockery et al. (1996, p. 503) reported that in the 24 study cities the bronchitis rate varied from three to ten percent. Finally a weighted average of the incidence rates in the six cities in the Dockery et al. (1989) study is 6.34 percent , where the sample size from each city is used to weight the respective incidence rate (Dockery et al., 1989, Tables 1 and 4).[90] This analysis assumes a 4.4 percent prevalence rate is the most representative of the national population. Note that this measure reflects the fraction of children that have a chest ailment diagnosed as bronchitis in the past year, not the number of days that children are adversely affected by acute bronchitis.[91]

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.50) associated with being in the most polluted city ($PM_{2.1}$ = 20.7 $\mu g/m^3$) 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.

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

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

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

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

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(2.47)}{14.9} - \frac{\ln(1.50)}{14.9}\right)}{1.96} = 0.0171$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.50)}{14.9} - \frac{\ln(0.91)}{14.9}\right)}{1.96} = 0.0171$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.0171.$$

C.5.2 Upper Respiratory Symptoms (Pope et al., 1991)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

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

$$\Delta Upper\ \mathrm{Re}spiratory\ Symptoms = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop\,,$$

where:

y_0 = daily upper respiratory symptom incidence rate per person = 0.3419
β = estimated PM_{10} logistic regression coefficient (Pope et al., 1991, Table 5) = 0.0036
ΔPM_{10} = change in daily average PM_{10} concentration
pop = asthmatic population[92] ages 9 to 11 = 6.91% of population ages 9 to 11
$_\beta$ = standard error of β (Pope et al., 1991, Table 5) = 0.0015

Incidence Rate. The incidence rate is published in Pope et al. (Pope et al., 1991, Table 2). Taking a sample-size-weighted average, one gets an incidence rate of 0.3419.

C.5.3 Lower Respiratory Symptoms (Schwartz et al., 1994)

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

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+.

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

$$\Delta Lower\ Re spiratory\ Symptoms = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{2.5}\cdot b} + y_0} - y_0\right]\cdot pop\,.$$

where:

y_0 = daily lower respiratory symptom incidence rate per person = 0.0012
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.01823
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of ages 7-14
$_\beta$ = standard error of β = 0.00586

[92] Adams (1995, Table 57) reported that in 1994, 6.91% of individuals under the age of 18 have asthma.

Incidence Rate. The proposed incidence rate, 0.12 percent, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). They did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated values are 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 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 is 0.12 percent,[93] which is used in this analysis.

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

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

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

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.82)}{20} - \frac{\ln(1.44)}{20}\right)}{1.96} = 0.00597$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.44)}{20} - \frac{\ln(1.15)}{20}\right)}{1.96} = 0.00574$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00586.$$

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

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

1984.[94] By the completion of the study, some students in the Topeka cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

C.5.4 Asthma Attacks: Whittemore and Korn (1980)

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and ozone. Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and O_x were significantly related to reported asthma attacks.

The C-R function to estimate the change in the number of asthma attacks is:

$$\Delta asthma\ attacks = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = daily incidence of asthma attacks = 0.027 (Krupnick, 1988, p. 4-6)
β = PM_{10} coefficient = 0.00144
ΔPM_{10} = change in daily PM_{10} concentration
pop = population of asthmatics of all ages = 5.61% of the population of all ages (Adams and Marano, 1995 Table 57).
$_{\beta}$ = standard error of β = 0.000556

Incidence Rate. The annual rate of 9.9 asthma attacks per astmatic is divided by 365 to get a daily rate. A figure of 9.9 is roughly consistent with the recent statement that "People with asthma have more than 100 million days of restricted activity" each year (National Heart, 1997, p. 1). This 100 million incidence figure coupled with the 1996 population of 265,557,000 (U.S. Bureau of the Census, 1997, Table 2) and the latest asthmatic prevalence rate of 5.61% (Adams and Marano, 1995, Table 57), suggest an annual asthma attach rate per asthmatic of 6.7.

Coefficient Estimate (β). Based on a model with ozone, the coefficient is based on a TSP coefficient (0.00079) (Whittemore and Korn, 1980, Table 5). Assuming that PM_{10} is 55 percent of TSP[95] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b = \frac{0.00079}{0.55} = 0.00144.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

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

[95]The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).

$$s_b = \frac{b}{t} = \frac{0.144}{2.576} = 0.000556.$$

C.5.5 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. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

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

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

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

where:

y_0 = daily work-loss-day incidence rate per person = 0.00648
β = inverse-variance weighted $PM_{2.5}$ coefficient = 0.0046
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[96]
pop = population of ages 18 to 65
$_{\beta}$ = standard error of β = 0.00036

Incidence Rate. The estimated 1994 annual incidence rate is the annual number (376,844,000) of WLD per person in the age 18-64 population divided by the number of people in 18-64 population (159,361,000). The 1994 daily incidence rate is calculated as the annual rate divided by 365.[97] Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

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

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

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.0046 .$$

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

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g} \right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g} \right).$$

This eventually reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00036 .$$

C.5.6 Minor Restricted Activity Days (Ostro and Rothschild, 1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average O_3 has highly variable association with RRADs and MRADs. Controlling for O_3, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.

The study is based on a "convenience" sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994d, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994b; Schwartz, 1994c).

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient used in this analysis is a weighted average of the coefficients in Ostro and Rothschild (1989), Table 4, using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) is:

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

where:

y_0 = daily MRAD daily incidence rate per person = 0.02137
β = inverse-variance weighted $PM_{2.5}$ coeffcient = 0.00741
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[98]
pop = adult population ages 18 to 65
$_{\beta}$ = standard error of β = 0.0007

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

Coefficient Estimate (β). The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.00741.$$

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

C.5.7 Any of 19 Respiratory Symptoms (Krupnick et al., 1990)

Krupnick et al. (1990) estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. (1990) listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included COH, ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily O_3, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

The C-R function used to estimate ARD2 is based on Krupnick et al. (1990, p. 12):[99]

$$\Delta ARD2 \cong b^{*}_{PM_{10}} \cdot \Delta PM_{10} \cdot pop\,,$$

where:

β^* = first derivative of the stationary probability = 0.000461
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 18-65 (Krupnick et al., 1990, Table 1)[100]
$_{\beta}$ = standard error of β^* = 0.000239

Coefficient Estimate (β^*). The logistic regression model used by Krupnick et al. (1990) takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$probability(ARD2 \mid sickness\ or\ not_{t-1}) = p_i = \frac{1}{1 - e^{b_0 + b_1 \cdot ARD2_{t-1} + X \cdot b}}\,,\ for\ i = 0,1.$$

[99]Krupnick and Kopp (1988, p. 2-24) and ESEERCO (1994, p. V-32) used the same C-R functional form as that used here.

[100]Krupnick et al. (1990, Table 1) reported the age distribution in their complete data, but they did not report the ages of individuals that were considered "adult." This analysis assumes that individuals 18 and older were considered adult. Only a small percentage (0.6%) of the study population is above the age of 60, so the C-R function was limited to the adult population up through the age of 65.

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of COH (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in COH and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before} .$$

However the full suite of coefficient estimates are not available.[101] Rather than use the full suite of coefficient values, the impact of COH on the probability of probability of ARD2 may be approximated by the derivative of ARD2 with respect to COH:

$$\frac{\partial probability(ARD2)}{\partial COH} = \frac{p_0 \cdot (1 - p_1) \cdot b_{COH} \cdot \left[p_1 + (1 - p_0) \right]}{(1 - p_1 + p_0)^2} = b^*_{COH} ,$$

where β_{COH} is the reported logistic regression coefficient for COH. Since COH data are not available for the benefits analysis, an estimated PM_{10} logistic regression coefficient is used based on the following assumed relationship between PM_{10}, COH, and TSP:

$$COH = 0.116 \cdot TSP$$

$$PM_{10} = 0.55 \cdot TSP$$

$$\Rightarrow COH = 0.2109 \cdot PM_{10}$$

[101]The model without NO_2 (Krupnick et al., 1990, Table V equation 3) was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (1990, Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., 1990, Table IV). Both the ozone and COH coefficients dropped by about a factor of two or more.

This analysis uses $\beta_{COH} = 0.0088$ (Krupnick et al., 1990, Table V equation 3). The conversion

$$\Rightarrow b_{PM_{10}} = 0.2109 \cdot b_{COH} = 0.2109 \cdot 0.0088 = 0.001856 .$$

from COH to TSP is based on study-specific information provided to ESEERCO (1994, p. V-32). The conversion of TSP to PM_{10} is from also from ESEERCO (1994, p. V-5), which cited studies by EPA (1986) and the California Air Resources Board (1982).

The change in the incidence of ARD2 associated with a given change in COH is then estimated by:

$$\frac{\P ARD2}{\P PM_{10}} \cong \frac{\Delta ARD2}{\Delta PM_{10}}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta PM_{10}} \cong b^{*}_{PM_{10}}$$

$$\Rightarrow \Delta ARD2 \cong b^{*}_{PM_{10}} \cdot \Delta PM_{10} .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO (1994, p. V-32), for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$b^{*}_{PM_{10}} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.001856 \cdot [0.7775_1 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000461 .$$

Standard Error (β). The standard error for the coefficient (β) is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. (1990, Table V):

$$\Rightarrow b_{PM_{10}, high} = 0.2109 \cdot b_{COH, high} = 0.2109 \cdot (0.0088 + (1.96 \cdot 0.0046)) = 0.003757$$

$$\Rightarrow b^{*}_{PM_{10}, high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.003757 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000934$$

$$s_{b, high} = \frac{b_{PM_{10}, high} - b_{PM_{10}}}{1.96} = \frac{(0.000934 - 0.000461)}{1.96} = 0.000236$$

$$b_{PM_{10}, low} = 0.2109 \cdot b_{COH, low} = 0.2109 \cdot (0.0088 - (1.96 \cdot 0.0046)) = -4.555 \cdot 10^{-5}$$

$$\Rightarrow b^{*}_{PM_{10},\,low} = \frac{0.0468\cdot(1-0.7775)\cdot(-4.555\cdot10^{-5})\cdot\left[0.7775+(1-0.0468)\right]}{(1-0.7775+0.0468)^{2}} = -1.132\cdot10^{-5}$$

$$\Rightarrow s_{b,\,low} = \frac{b-b_{low}}{1.96} = \frac{\left(0.000461+1.132\cdot10^{-5}\right)}{1.96} = 0.000241$$

$$s_{b} = \frac{s_{b,\,high}+s_{b,\,low}}{2} = 0.000239\,.$$

C.5.8 Shortness of Breath (Ostro et al., 1995)

Using a logistic regression estimation, Ostro et al. (1995) estimated the impact of PM_{10}, ozone, NO_2, and SO_2 on the incidence of coughing, shortness of breath, and wheezing in 83 African-American asthmatic children ages 7-12 living in Los Angeles from August through September 1992. Regression results show both PM_{10} and ozone significantly linked to shortness of breath; the beginning of an asthma episode was also significantly linked to ozone. No effect was seen for NO_2 and SO_2. Results for single-pollutant models only were presented in the published paper.

The C-R function to estimate the change in shortness of breath days is:

$$\Delta Shortness\,of\;\;Breath = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b}+y_0}-y_0\right]\cdot pop\,,$$

where:

y_0 = daily shortness of breath incidence rate per person (Ostro et al., 1995, p. 715) = 0.056
β = estimated PM_{10} logistic regression coefficient = 0.00841
ΔPM_{10} = change in daily average PM_{10} concentration
pop = asthmatic African-American population ages 7 to 12 = 6.91% of African-American population ages 7 to 12
$_{\beta}$ = standard error of β = 0.00363

Prevalence. Adams (1995, Table 57) reported that in 1994, 6.91% of individuals under the age of 18 have asthma. It has been reported that African-Americans have a higher prevalence of asthma (e.g., see U.S. EPA, 1996a). Ostro et al. (1995, p. 711) noted that "Although prevalence is only somewhat greater among African-Americans than among whites, rates of morbidity are markedly higher." Indeed, the asthma rates for whites and African-Americans were almost identical in 1994 (1995, Table 59), so no correction is made to the estimated prevalence rate for asthma in African-Americans.

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio of 1.60 (Ostro et al., 1995, Table 3) associated with a change in mean PM_{10} of 55.87 $\mu g/m^3$ (Ostro et al., 1995, Table 2). The coefficient is calculated as follows:

$$b = \frac{\ln(1.60)}{(55.87)} = 0.00841.$$

Standard Error ($_{\beta}$). The standard error for the coefficient ($_{\beta}$) is calculated from the reported lower and upper bounds of the odds ratio (Ostro et al., 1995, Table 2):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(2.37)}{55.87} - \frac{\ln(1.60)}{55.87}\right)}{1.96} = 0.003588$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{ln(1.60)}{55.87} - \frac{ln(1.07)}{55.87}\right)}{1.96} = 0.003674$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.003631.$$

C.5.9 Moderate (or Worse) Asthma (Ostro et al., 1991)

Ostro et al. (1991) examined the effect of air pollution on asthmatics, ages 18 to 70, living in Denver, Colorado from December 1987 to February 1988. The respondents in this study were asked to record daily a subjective rating of their overall asthma status each day (0=none, 1=mild, 2=moderate, 3=severe, 4=incapacitating). Ostro et al. then examined the relationship between moderate (or worse) asthma and H^+, sulfate, SO_2, $PM_{2.5}$, estimated $PM_{2.5}$, PM_{10}, nitrate, and nitric acid. Daily levels of H^+ were linked to cough, asthma, and shortness of breath. $PM_{2.5}$ was linked to asthma. Sulfate was linked to shortness of breath. No effects seen for other pollutants. The C-R function is based on a single-pollutant linear regression model where the log of the pollutant is used.

The C-R function to estimate the change in the number of days with moderate (or worse) asthma is:

$$\Delta Days\ Moderate/Worse\ Asthma = -b \cdot \ln\left(\frac{PM_{2.5,\,after}}{PM_{2.5,\,before}}\right) \cdot pop,$$

where:

β = estimated $PM_{2.5}$ coefficient (Ostro et al., 1991, Table 5) = 0.0006
$PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = asthmatic population of all ages = 5.61% of the population of all ages (Adams and Marano, 1995 Table 57)
$_{\beta}$ = standard error of β (Ostro et al., 1991, Table 5) = 0.0003

Coefficient Estimate (β). Two $PM_{2.5}$ coefficients are presented, both equal 0.0006, however only one is significant. The coefficient based on data that does not include estimates of missing $PM_{2.5}$ values is not

significant ($_{\beta}$ = 0.0053); the coefficient that includes estimates of missing $PM_{2.5}$ values (estimated using a function of sulfate and nitrate) is significant at $p < 0.5$ ($_{\beta}$ = 0.0003). The latter coefficient is used here.

Population. The C-R function is applied to asthmatics of all ages, where it is assumed that 5.61 percent of the population of all ages is asthmatic. This raises two issues: the age group for which the function should be used, and the fraction of the population that is asthmatic. The study population consists of asthmatics between the ages of 18 and 70. It seems reasonable to assume that individuals over the age of 70 are at least as susceptible as individuals in the study population. It also seems reasonable to assume that individuals under the age of 18 are also susceptible. For example, controlling for oxidant levels, Whittemore and Korn (1980) found TSP significantly related to asthma attacks in a study population comprised primarily (59 percent) of individuals less than 16 years of age.

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

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g}\right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g}\right).$$

This reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00070.$$