Power Plant Emissions: Particulate Matter-Related Health Damages and the Benefits of Alternative Emission Reduction Scenarios

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1 Introduction

Power plants are significant emitters of sulfur dioxide (SO_2) and nitrogen oxides (NO_x). In many parts of the country, especially the Midwest, power plants are the largest contributors. These gases are harmful themselves, and they contribute to the formation of acid rain and particulate matter. Particulate matter (PM) reduces visibility, often producing a milky haze that blankets wide regions, and it is a serious public health problem. Over the past decade and more, hundreds of studies worldwide have linked particulate matter to a wide range of adverse health effects in people of all ages, including premature death, chronic bronchitis, hospital admissions and asthma. While this large body of research cannot establish a cause-and-effect relationship between PM and adverse health effects, the research does provide strong evidence that reducing ambient PM concentrations will lead to improvements in human health. The US EPA developed analytical methods that draw on this health research, combined with estimates of future air pollution emissions and air quality models, to prepare quantified estimates of the avoidable health effects from improving ambient PM levels. The EPA used these analytical methods to estimate the health benefits of a wide variety of actual or proposed individual federal air programs, including programs that reduce emissions from power plants, cars, and both on-road and off-road diesel engines.

This report estimates the avoidable health effects of each of a series of alternative regulatory scenarios for power plants, focusing on the adverse human health effects due to exposure to fine particulate matter ($PM_{2.5}$, which are particles less than 2.5 microns in diameter). This report uses the same analytical methods that the U.S. Environmental Protection Agency used in 2003 to prepare an analysis of the potential health effects of the proposed Clear Skies Act (EPA 2003). This report conducts an analysis of the impacts in 2010 and 2020 of three policy alternatives to the proposed Clear Skies Act:

- Carper/Gregg/Chaffee "The Clean Air Planning Act", S. 834 (henceforth "Carper")
- The Jeffords/Lieberman/Collins "The Clean Power Act", S. 366 (henceforth "Jeffords")
- The EPA August 2001 Straw Proposal (one of several alternatives EPA analyzed prior to the announcement of the Clear Skies Initiative in 2002). Henceforth "Straw"

For comparison purposes, this report includes the results of the EPA 2003 analysis of the Clear Skies Act (henceforth "CSA").

In addition, this report also examines the health impacts associated with the total amount of emissions from coal fired electricity generating units (power plants) in 2010. This "No EGU" analysis is clearly not a policy option, but rather helps gain a better understanding of the total magnitude of the health effects associated with the total emissions from this major sources of pollutants that lead to the formation of PM. It also helps put into better context the health improvements associated with each of the policy option scenarios examined in this report.

Following the methods used in the 2003 EPA analysis of the proposed Clear Skies Act, this study estimates the health impacts from various policy options for reducing power plant air pollution emissions. Using the same emissions estimates and air quality forecasting methods as EPA used in the

Clear Skies Act analysis, we prepare detailed future ambient air quality estimates for each of the nine scenarios described above. We then used the same health assessment methods as EPA to estimate the avoidable health effects associated with the changes in ambient air quality. Because we used the same methods and data as the 2003 EPA analysis, the results here are directly comparable with EPA's estimates of the future baselines for 2010 and 2020, as well as EPA's estimates of the potential improvements if the proposed Clear Skies Act is implemented. EPA has made extensive details of the technical details of their analysis available via the internet at www.epa.gov/clearskies/technical.html. The technical background materials on the methods and data sources for the EPA analysis are applicable to this analysis. In particular, the background paper on the models used in the EPA analysis ("Section H: 2003 Summary of the Models Used for this Analysis", at www.epa.gov/clearskies/03technical_package_sectionh.pdf) contains many details concerning the models used to estimate the electricity generation (IPM™), air quality (REMSAD) and the health analysis model (BenMAP) in both the EPA analysis and this report.

Chapter 2 describes the emissions inventory estimates, and the changes in the emissions associated with each scenario analyzed. Chapter 3 describes the methods used to estimate changes in particulate matter concentrations. Chapter 4 describes general issues arising in estimating and valuing changes in adverse health effects associated with changes in particulate matter. Chapter 5 describes in some detail the methods used for estimating and valuing adverse health effects, and in Chapter 6 we present the results of these analyses. Chapter 7 presents estimates of the impact of these alternative policy options on the PM non-attainment status.

This study has several appendices. Appendix A presents a derivation of the particulate matter concentration-response functions used in all the analyses. Appendix B presents additional detail on the results in Chapter 6, including statistical uncertainty analysis. Appendix C presents additional details about the non-attainment analysis in Chapter 7.

2 Emissions Inventory

The detailed estimates of the future emissions inventory used in this analysis is the same inventory EPA used in their analysis of the Clear Skies Act. In order to conduct an analysis of changes in the levels of ambient PM2.5 in the atmosphere from changes in emissions from power plants, it is necessary to have an estimate of the complete inventory from all sources of precursor emissions, not just the emissions from the source categories. EPA prepared the complete estimated emissions inventory for both 2010 and 2020 necessary to conduct a PM air quality analysis. This inventory includes emissions from not only power plants, but also other large industrial sources, all mobile sources, smaller "Area" emission sources ranging from gasoline stations to household emissions, agricultural emissions, and naturally occurring emissions from forests, grasslands, etc. The location and timing of emissions have an important impact on PM formation, so the emissions inventory includes extensive detail on the location and timing of the estimated emissions. Canadian and Gulf of Mexico sources are included in the inventory as well, as these pollutants effect PM levels in the continental US.

The emissions inventory estimates the quantity of emissions of six pollutants that will occur in specific future years (2010 and 2020 in this analysis) as future base case. For many emission source categories these future base cases have lower emissions in the future than occur now, as the impact of already enacted federal and state programs will increase over time. In particular, as older cars and trucks are replaced with newer, cleaner, vehicles the emissions from mobile sources decreases. Similarly older industrial equipment will be replaced by cleaner new equipment. In aggregate, total emissions are lower in the future base cases than in the 2001 emission inventory. Eventually, however, the improvements from existing programs will diminish as the programs are fully implemented. In addition to the forces that will decrease emissions , there are also forces that will increase emissions. Both a growing population and expanding economy tend to increase emissions. These forces generally grow stronger over time. Eventually the decreasing emissions from existing federal programs are overwhelmed by the increasing emissions from growth, and the total amount of emissions begins to increase.

Modeling the emission from power plants ICF Consulting used the IPM™ to forecast emissions from power plants for the policy options examined in this report. ICF Consulting used the same version of IPM™, with the same data and modeling assumptions, for the analysis in this report as they used for EPA's analysis of the Clear Skies Act.

IPM™ is an industry-leading energy modeling system that simulates the deregulated wholesale market for electricity. The EPA has used IPM™ to evaluate the economic, operational and emission impacts of a wide variety of policies and rulemakings affecting the power sector.

IPM™ is a multi-region linear programming model that determines the least-cost capacity expansion and dispatch strategy for operating the power system over specified future periods, under specified operational, market, and regulatory constraints. Constraints include emissions caps, transmission constraints, regional reserve margins, and meeting regional electric demand. Given a specified set of parameters and constraints, IPM™ develops an optimal capacity expansion plan, dispatch order, and air emissions compliance plan for the power generation system based on factors such as fuel prices, capital costs and operation and maintenance (O&M) costs of power generation, etc. Additional details about the EPA IPM™ model are available at EPA's Clear Skies Website, www.epa.gov/clearskies/technical.html.

The model is dynamic: it makes decisions based on expectations of future conditions, such as fuel prices, and technology costs. Decisions are made on the basis of minimizing the net present value of

capital plus operating costs over the full planning horizon. The model draws on a database containing information on the characteristics of each power plant (such as unit ID, unit type, unit location, fuel used, heat rate, emission rate, existing emission control technology, etc.) in the U.S.

Summary of the National Emissions Inventory

There are six air pollutant emissions that are used to model PM concentrations. The are:

Oxides of Nitrogen (NOx)
Volatile Organic Compounds (VOC)
Ammonia (NH3)
Sulfur Dioxide (SO2)
Direct fine particle emissions (PM25)
Direct coarse particle emissions (PM10)
Primary Elemental Carbon (PMC)

Table 2.1 summarizes the estimated total emissions in the continental United States in 2010 for the six precursor air pollutants. Table 2.2 summarizes the total emissions in 2020.

Table 2.1 2010 Baseline Emissions Inventory (Tons/Year)

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

Table 2.2 2020 Baseline Emissions Inventory (Tons/Year)

Source	NOx	VOC	NH3	SO2	PM10	PM2.5	PMC
EGU	4,056,026	35,389	1,478	8,956,475	227,727	116,895	110,832
Other Industrial	3,393,215	1,894,870	314,898	4,044,693	1,180,614	704,229	476,385
On Road	1,989,951	2,061,066	378,887	35,421	143,600	72,595	71,005
Non Road	2,842,794	2,192,851	59,548	228,308	227,336	186,359	40,977
Area	2,295,578	7,714,354	4,475,040	1,413,461	7,788,908	2,297,748	5,491,160
Total US	14,577,565	13,898,530	5,229,851	14,678,358	9,568,185	3,377,825	6,190,360
Canada & Gulf of Mexico	1,972,010	2,550,200	555,496	1,901,396	1,887,887	419,719	1,468,168
Total Modeled	16,549,575	16,448,730	5,785,347	16,579,754	11,456,072	3,797,545	7,658,528

Each of the policy options examined in this report keep hold the emissions constant from all emissions categories except for the EGU category. The EGU emissions in each policy scenario (including the Baseline scenarios) were modeled using IPM™, combined with additional methods developed by EPA to estimate the unit-specific emissions from each power plant unit. The IPM™ analysis incorporated the targeted emission caps for sulfur (SO2) and nitrogen (NOx) (as well as carbon and mercury if included in the scenario) from EGUs in modeling the emissions from each power plant. The targeted emission caps (referred to as the "nominal caps") are not necessarily met however, because of emissions trading provisions incorporated in each scenario. "Banking" of emission credits allows the modeled emissions to exceed the nominal caps in most policy option scenarios. Because the policy options provide power plant operators some discretion to "bank" emission reduction credits in one year by reducing emissions below that facility's mandatory levels, and in a later year use the banked credits as part of meeting their mandatory levels that year, the total emissions from power plants in a given year can exceed the nominal caps. Banked emission credits can also be sold, and used by another power plant operator. Banking is considered by the IPM™ model, so the air quality analysis (and subsequent health analysis) in both 2010 or 2020 can include emissions in excess of the nominal caps. The health effects estimated in this report therefore reflect the impact of the modeled emission changes, not the changes that would occur if the nominal emission caps are met.

In order to quantify the total contribution from all power plants in the No EGU analysis, we conducted the air quality analysis by eliminating the emissions from all fossil fueled electricity generation units, and calculate the resulting air quality. This identifies the total air quality "footprint" of power plants on fine particulate matter concentrations.

The nominal emission targets and the modeled emissions from electricity generating units are presented in Table 2.3

Table 2.3 Nominal and Modeled Emissions from Electricity Generating Stations

Scenario		Nitrogen	Sulfur
2010 Analysis			
Baseline	Modeled Emissions in 2010	3.9 million tons	9.9 million tons
Clear Skies Act	Nominal Cap	2.1 million ton cap by 2008	4.5 million ton cap by 2008
	Modeled Emissions in 2010	2.2 million tons	6.3 million tons
Straw Proposal	Nominal Cap	1.87 million tons by 2008	2 million ton cap by 2010
	Modeled Emissions in 2010	1.67 million tons	4.53 million tons
Carper Bill	Nominal Cap	1.87 million tons by 2008	4.5 million ton cap by 2009
	Modeled Emissions in 2010	1.83 million tons	4.77 million tons
Jeffords Bill	Nominal Cap	1.51 million tons by 2009	2.25 million ton cap by 2009
	Modeled Emissions in 2010	1.18 million tons	2.3 million tons
2020 Analysis			
Baseline Modeled	Modeled Emissions in 2020	4.06 million tons	8.96 million tons
Clear Skies Act	Nominal Cap	1.7 million ton cap by 2018	3 million ton cap by 2018
	Modeled Emissions in 2020	1.8 million tons	4.35 million tons
Straw Proposal	Nominal Cap	1.25 million ton cap by 2012	2 million ton cap by 2010
	Modeled Emissions in 2020	1.31 million tons	2.87 million tons
Carper Bill	Nominal Cap	1.7 million ton cap by 2013	2.25 million ton cap by 2016
	Modeled Emissions in 2020	1.76 million tons	3.39 million tons
Jeffords Bill	Nominal Cap	1.51 million tons by 2009	2.25 million ton cap by 2009
	Modeled Emissions in 2020	0.91 million tons	2.1 million tons

3 Air Quality Modeling

The analysis used results from the Regulatory Modeling System for Aerosols and Acid Deposition (REMSAD, ver 7.06) to forecast changes in the ambient concentration of both PM_{10} and $PM_{2.5}$ at the REMSAD grid cell level. Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is ideal for evaluating the air-quality effects of emission control scenarios.

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

Particulate Matter Formation

Ambient concentrations of PM are composed of directly emitted particles and of secondary aerosols of sulfate, nitrate, and organics. Particulate matter is the generic term for the mixture of microscopic solid particles and liquid droplets found in the air. The particles are either emitted directly from these combustion sources or are formed in the atmosphere through reactions involving gases, such as SO_2 and NO_x.

REMSAD Air Quality Model

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

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

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

with *modified* or *projected* emission inventories to simulate possible alternative/future emission scenarios.

The PM levels estimated by REMSAD were not directly used in EPA's health analysis of the Clear Skies Act, nor are the directly used here. Instead of using the REMSAD results directly, we use the REMSAD results to estimate the relative change in PM levels. We combine the REMSAD results with actual $PM_{2.5}$ monitor readings from 2001 to estimate the $PM_{2.5}$ levels actually used in the health analysis. This same procedure was used in the EPA Clear Skies Act health analysis. EPA believes this provides a better estimate of future $PM_{2.5}$ levels than the REMSAD modeling data itself.

At the location of each $PM_{2.5}$ monitor, we quantified the relationship between REMSAD estimated levels of $PM_{2.5}$ at the monitor for a base year (2001) and the future year (2010 or 2020). These REMSAD-based adjustment ratios are applied to the actual monitoring data to generate estimates of $PM_{2.5}$ levels at each monitor for each of the future scenarios.

In order to provide estimates of ambient $PM_{2.5}$ levels everywhere in the country, and not just at the monitors, an additional analytical step is required. To calculate population exposure to PM, each REMSAD grid cell was assigned a distance-weighted average of adjusted PM levels from a set of monitors that best surrounds the cell. This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (eVNA) spatial interpolation (See Abt Associates, 2000 for a more detailed description).

The estimated future baseline $PM_{2.5}$ levels estimated using the REMSAD and eVNA method, and the change in $PM_{2.5}$ levels associated with each policy option, are shown in the Exhibits 3.1 to 3.11. The maps depict annual mean $PM_{2.5}$ levels (in $\mu g/m^3$) Exhibits 3.1 and 3.2 show the future baseline $PM_{2.5}$ conditions in 2010 and 2020. Exhibits 3.3 through 3.7 show the estimated 2010 changes in the annual mean $PM_{2.5}$ level for the policy options and the No EGU scenario. Exhibits 3.8 through 3.11 show the estimated changes in 2020 for the policy options.

Exhibit 3.1 2010 Baseline Annual Mean $PM_{2.5}$ Levels

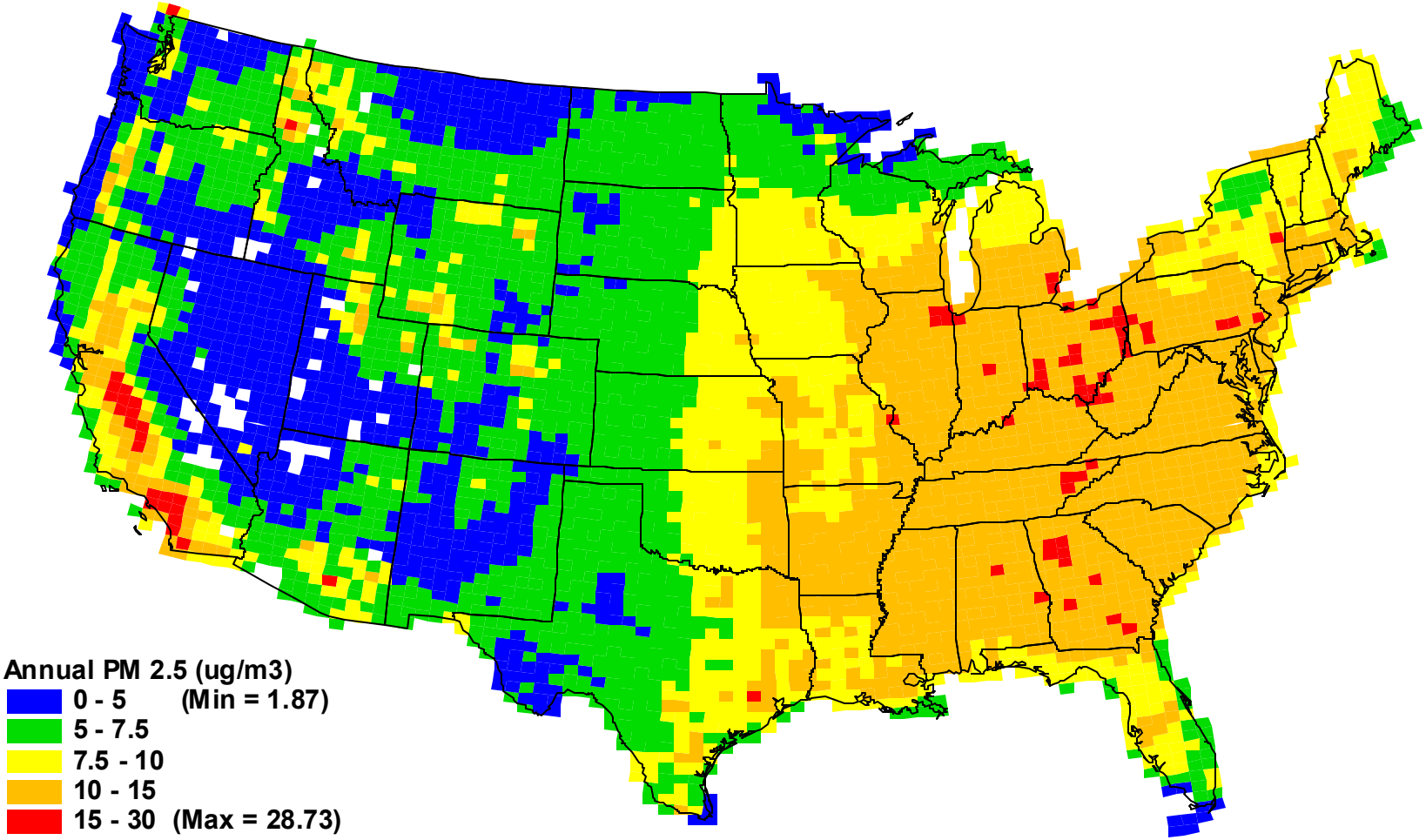


Exhibit 3.2 2020 Baseline Annual Mean $PM_{2.5}$ Levels

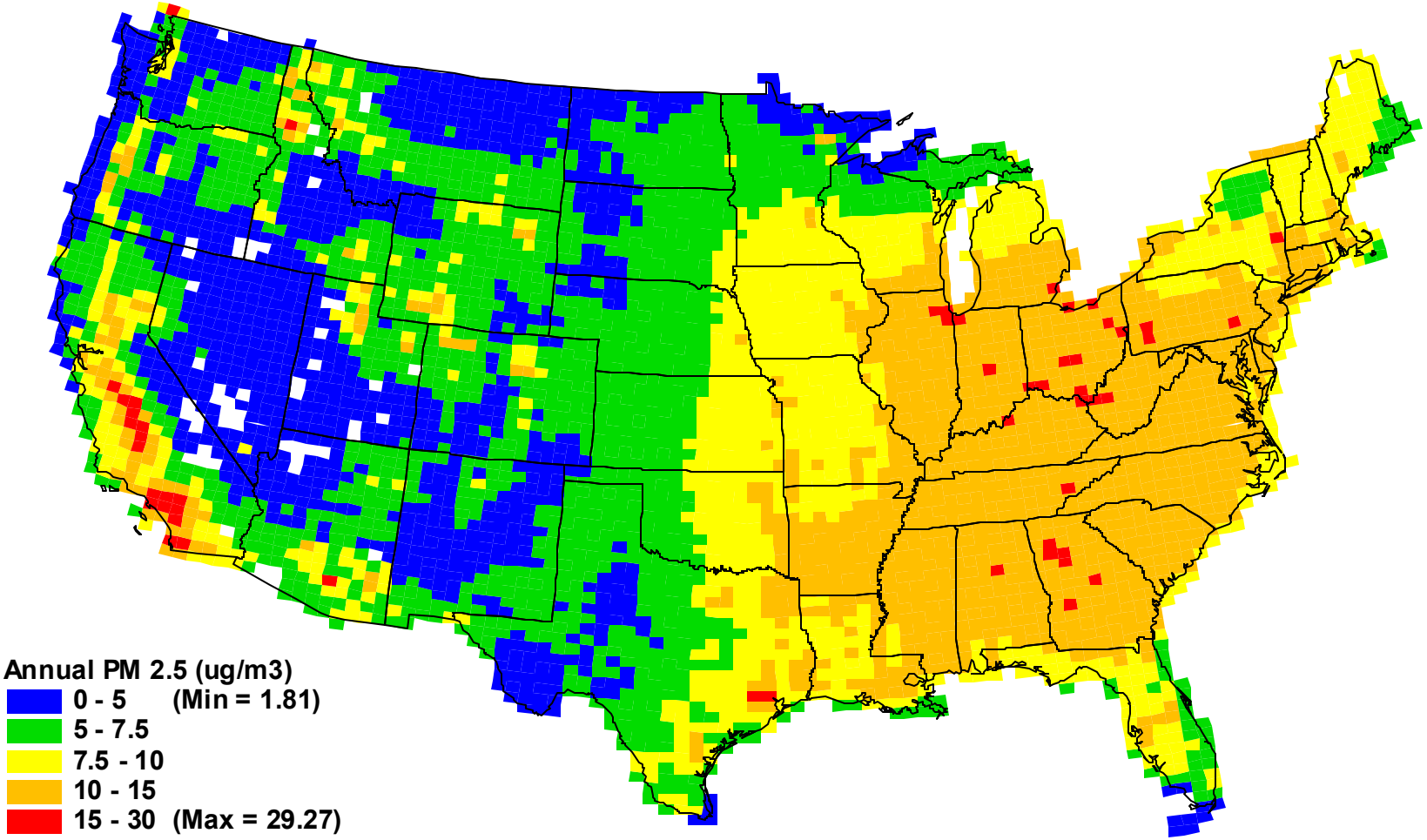


Exhibit 3.3 Change in 2010 Annual Mean $PM_{2.5}$ Levels with Clear Skies Act

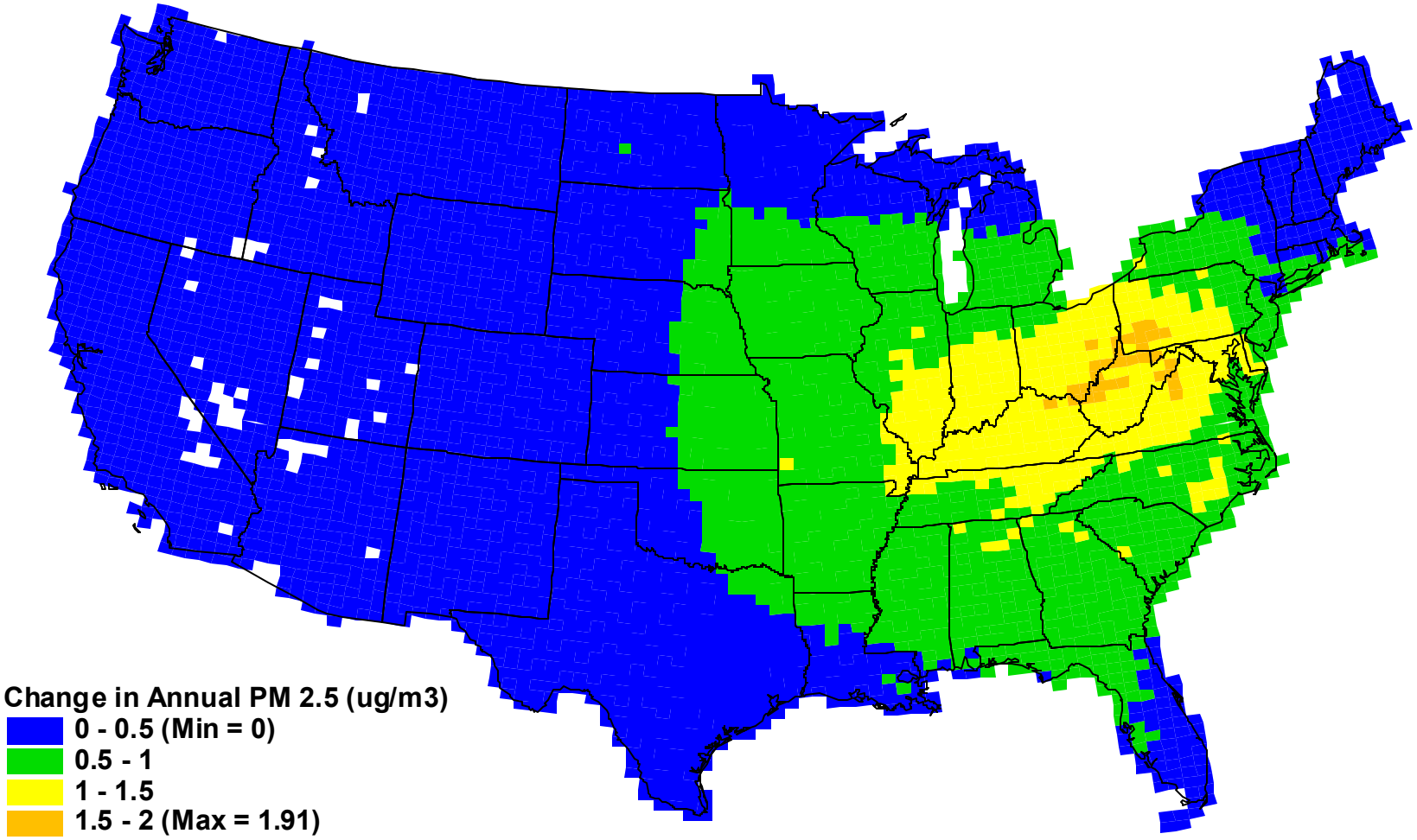


Exhibit 3.4 Change in 2010 Annual Mean $PM_{2.5}$ Levels with Carper Bill

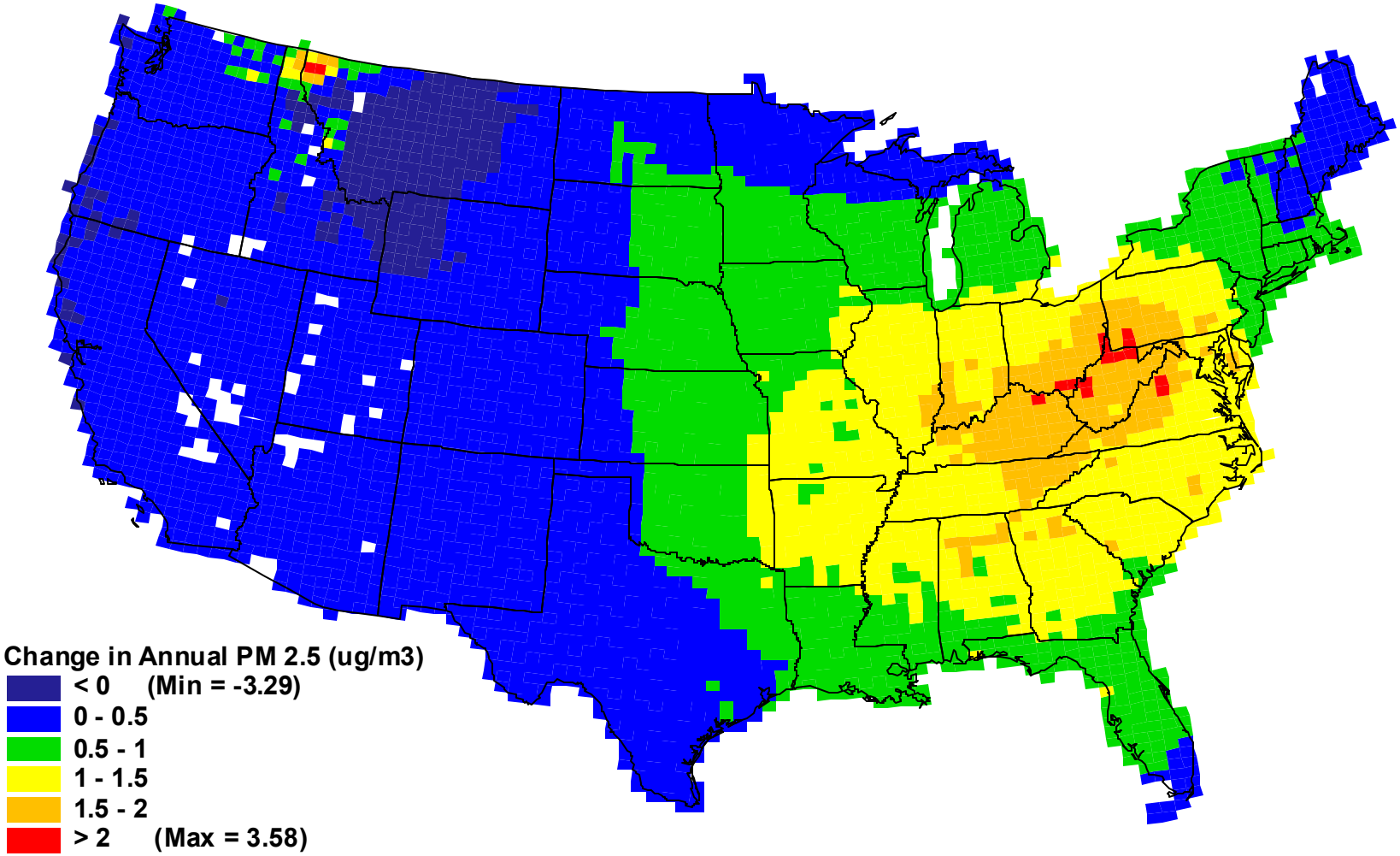


Exhibit 3.5 Change in 2010 Annual Mean $PM_{2.5}$ Levels with EPA Straw Proposal

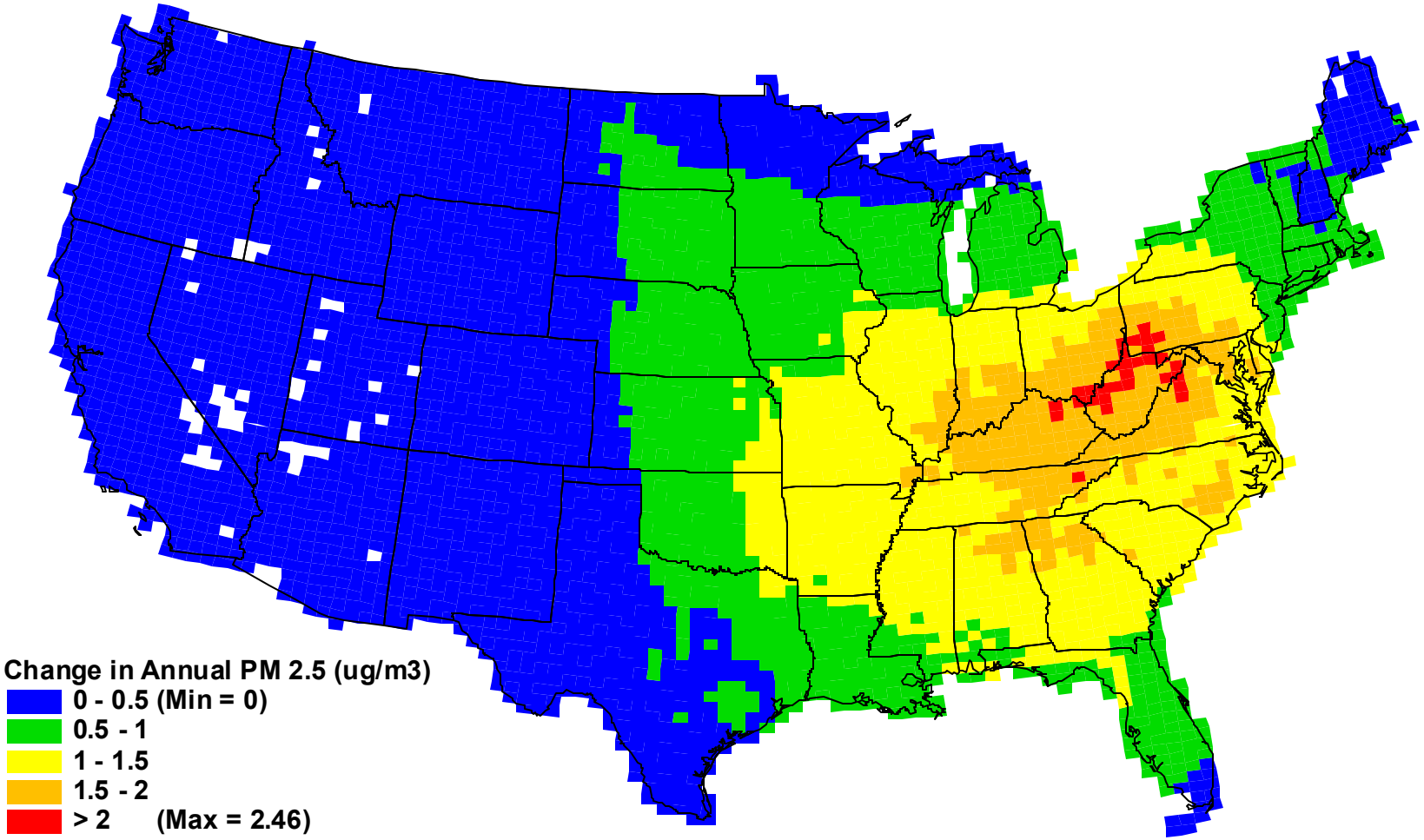


Exhibit 3.6 Change in 2010 Annual Mean $PM_{2.5}$ Levels with Jeffords Bill

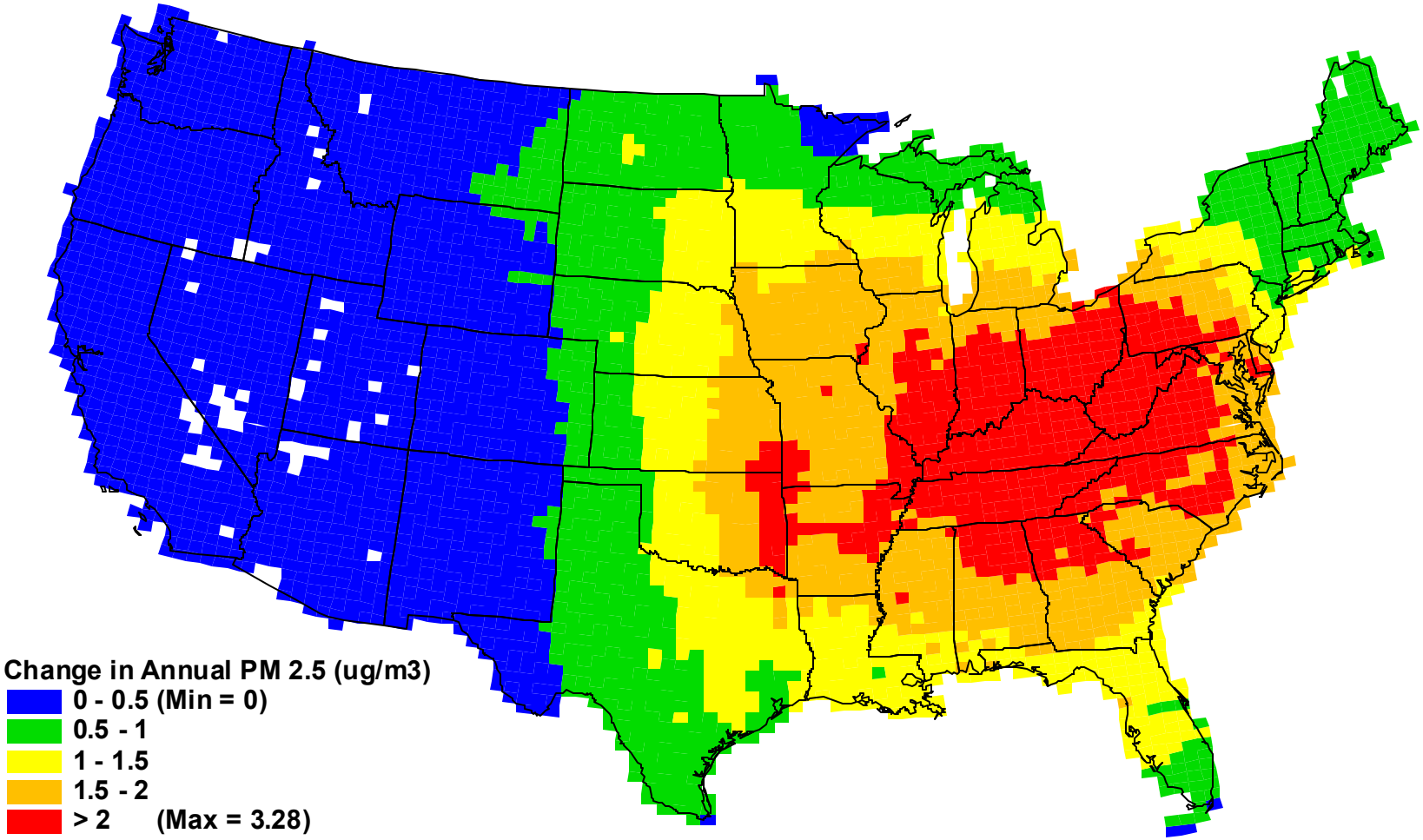


Exhibit 3.7 Change in 2010 Annual Mean $PM_{2.5}$ Levels for "No EGU" Scenario

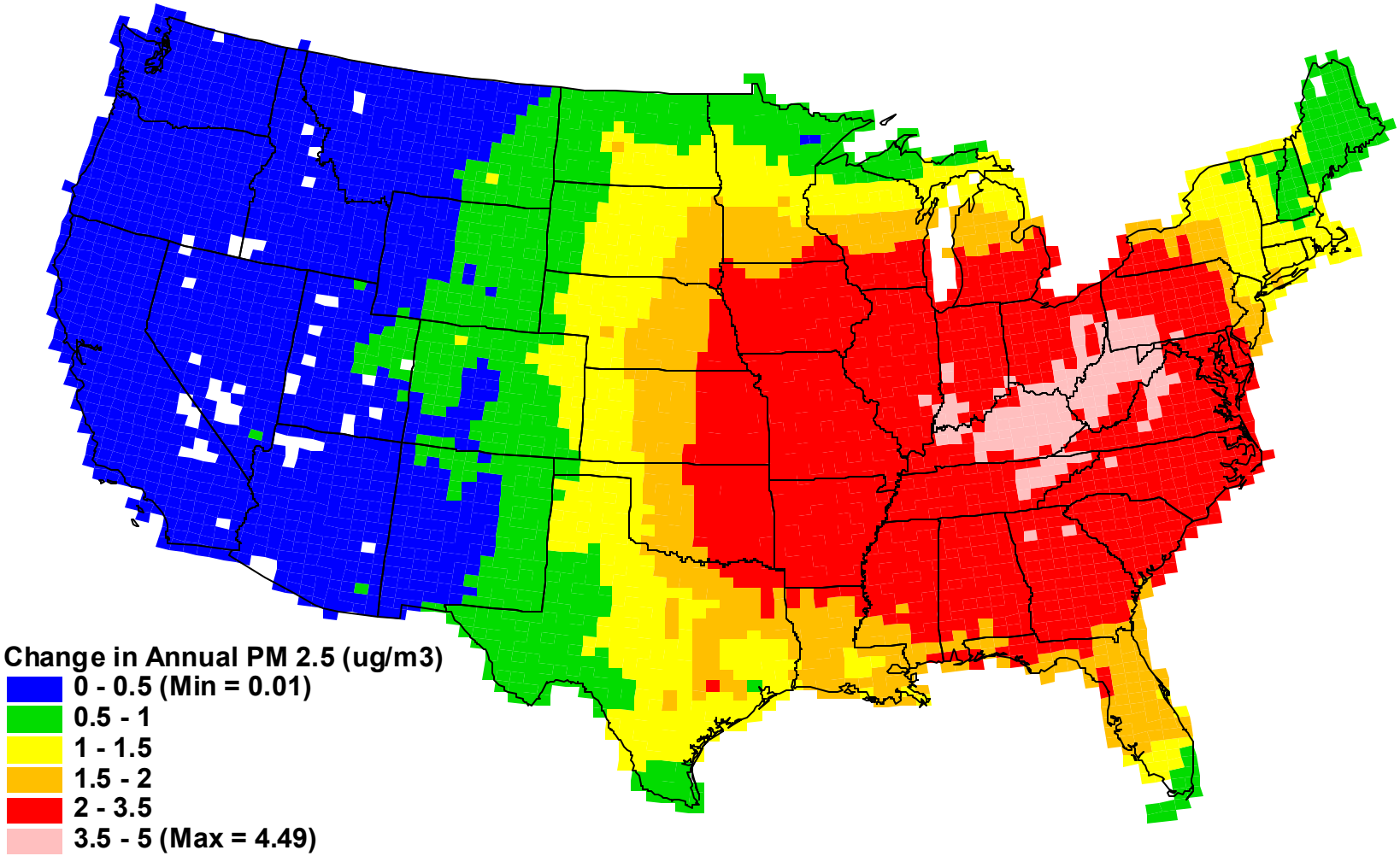


Exhibit 3.8 Change in 2020 Annual Mean $PM_{2.5}$ Levels with Clear Skies Act

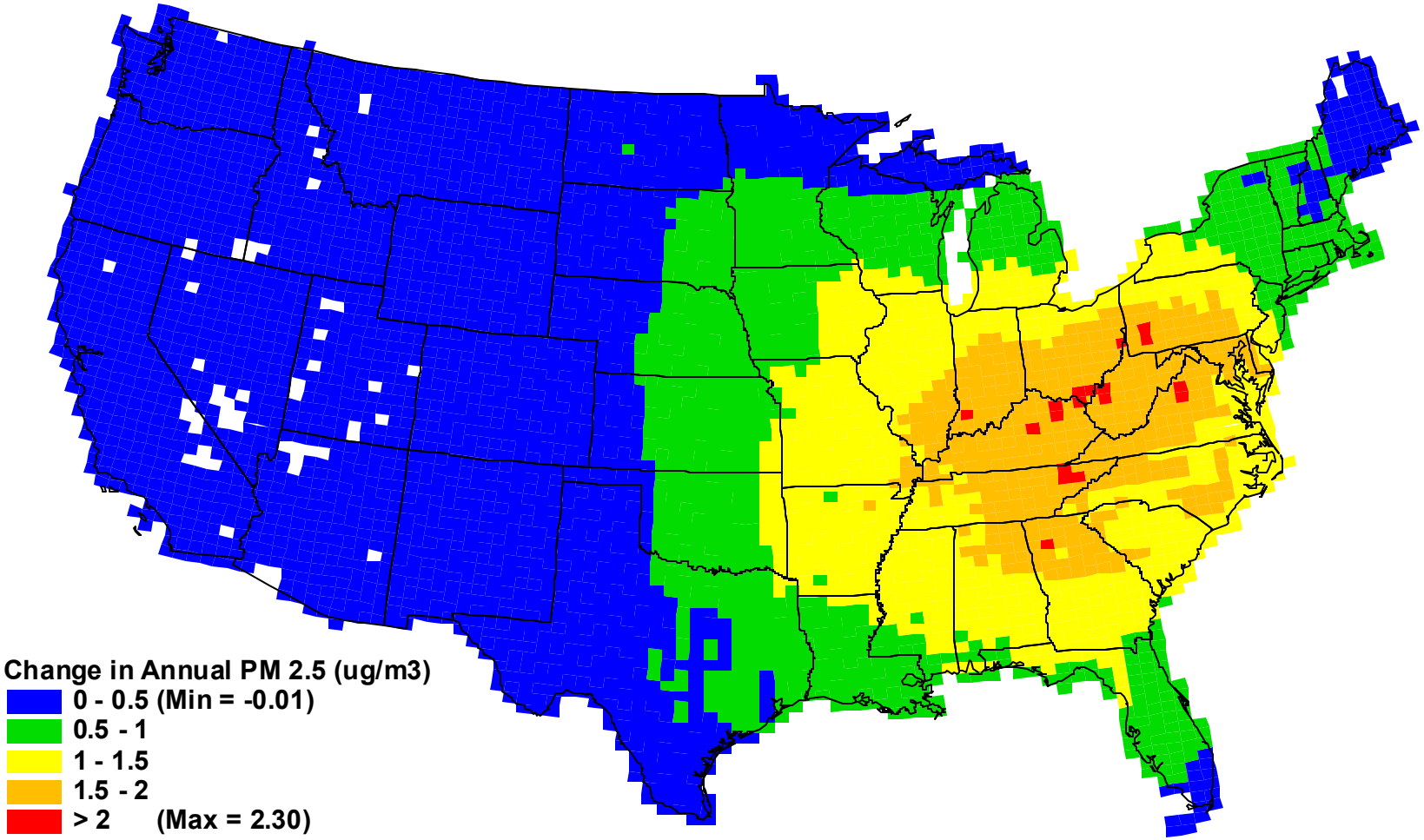


Exhibit 3.9 Change in 2020 Annual Mean $PM_{2.5}$ Levels with EPA Straw Proposal

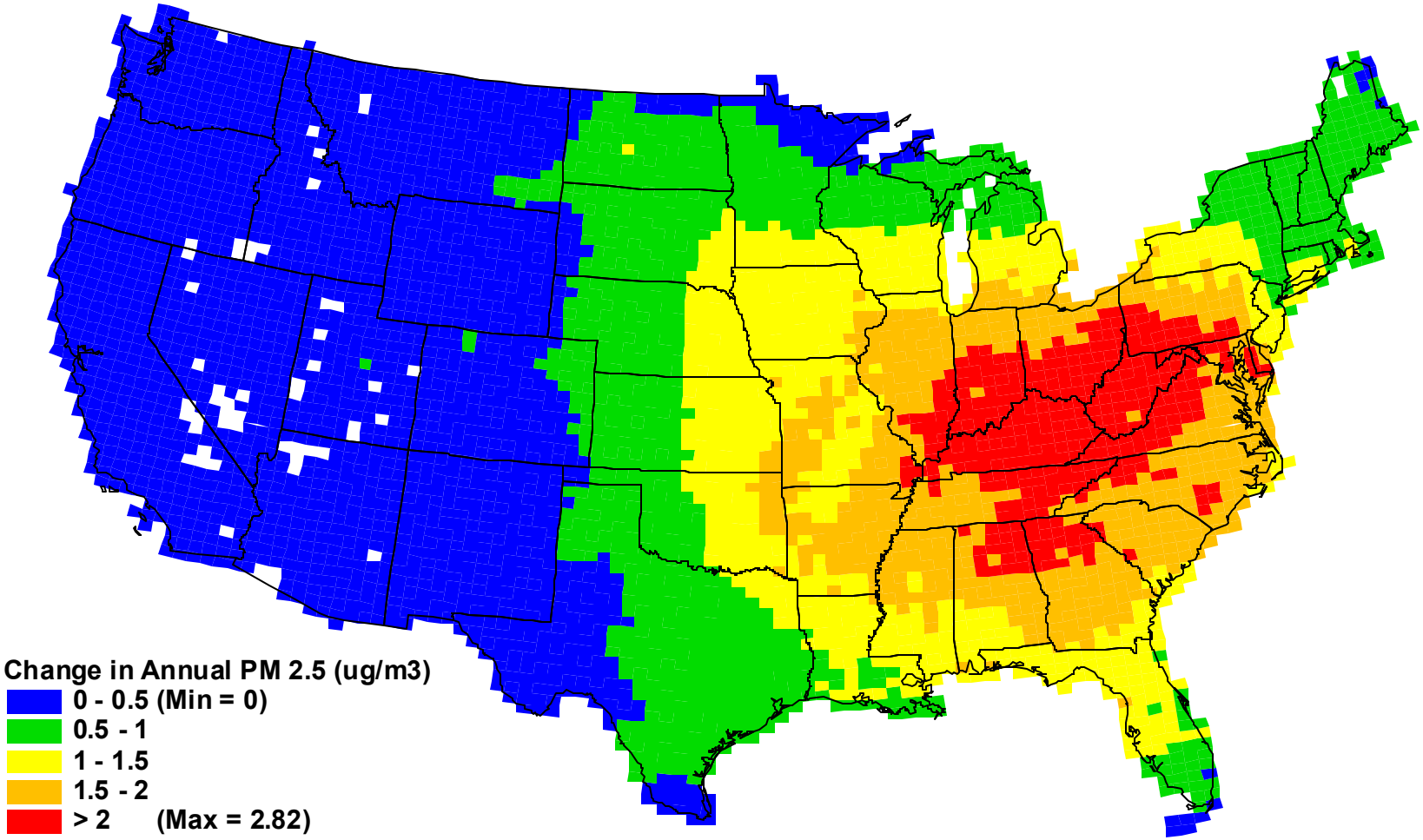


Exhibit 3.10 Change in 2020 Annual Mean $PM_{2.5}$ Levels with Carper Bill

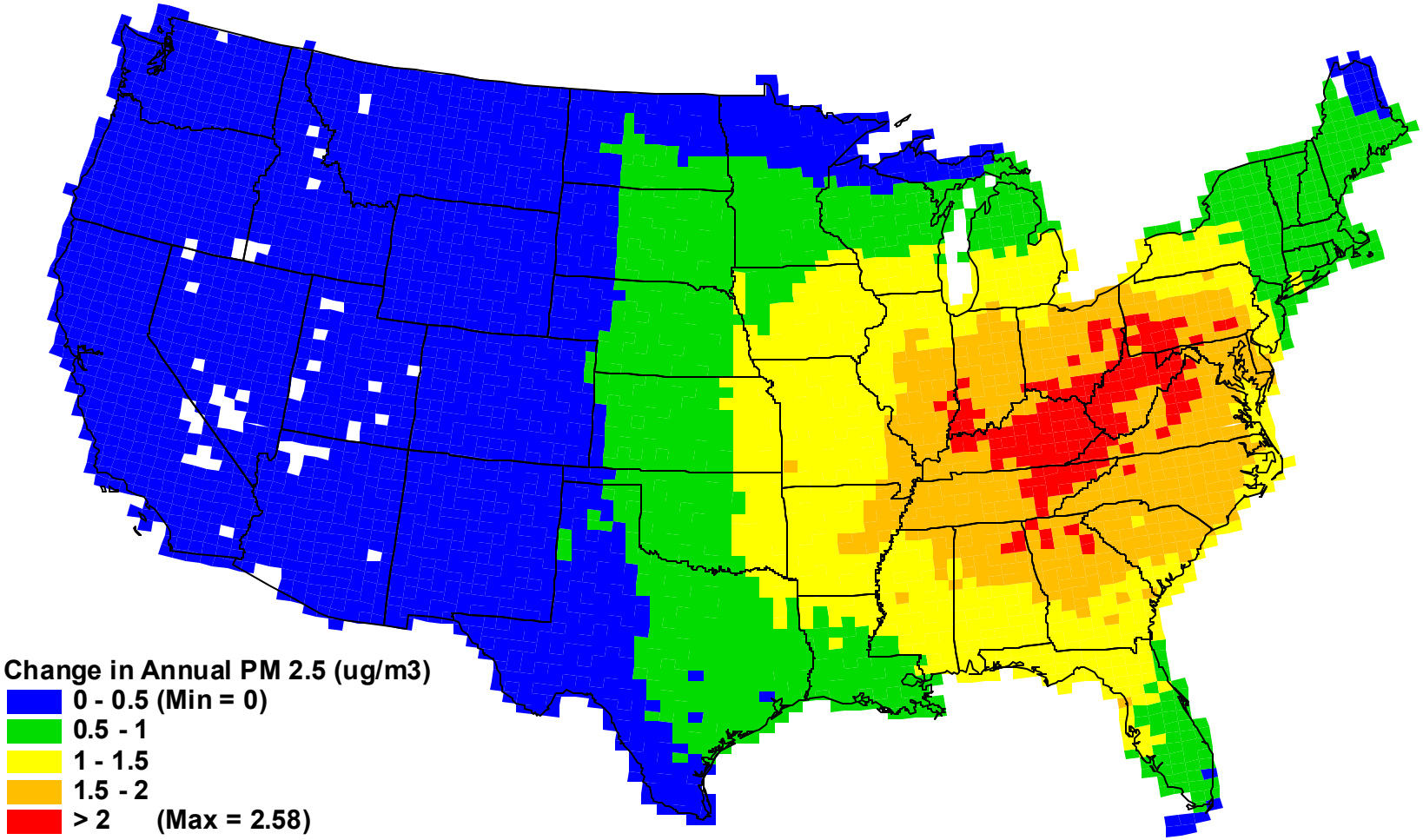
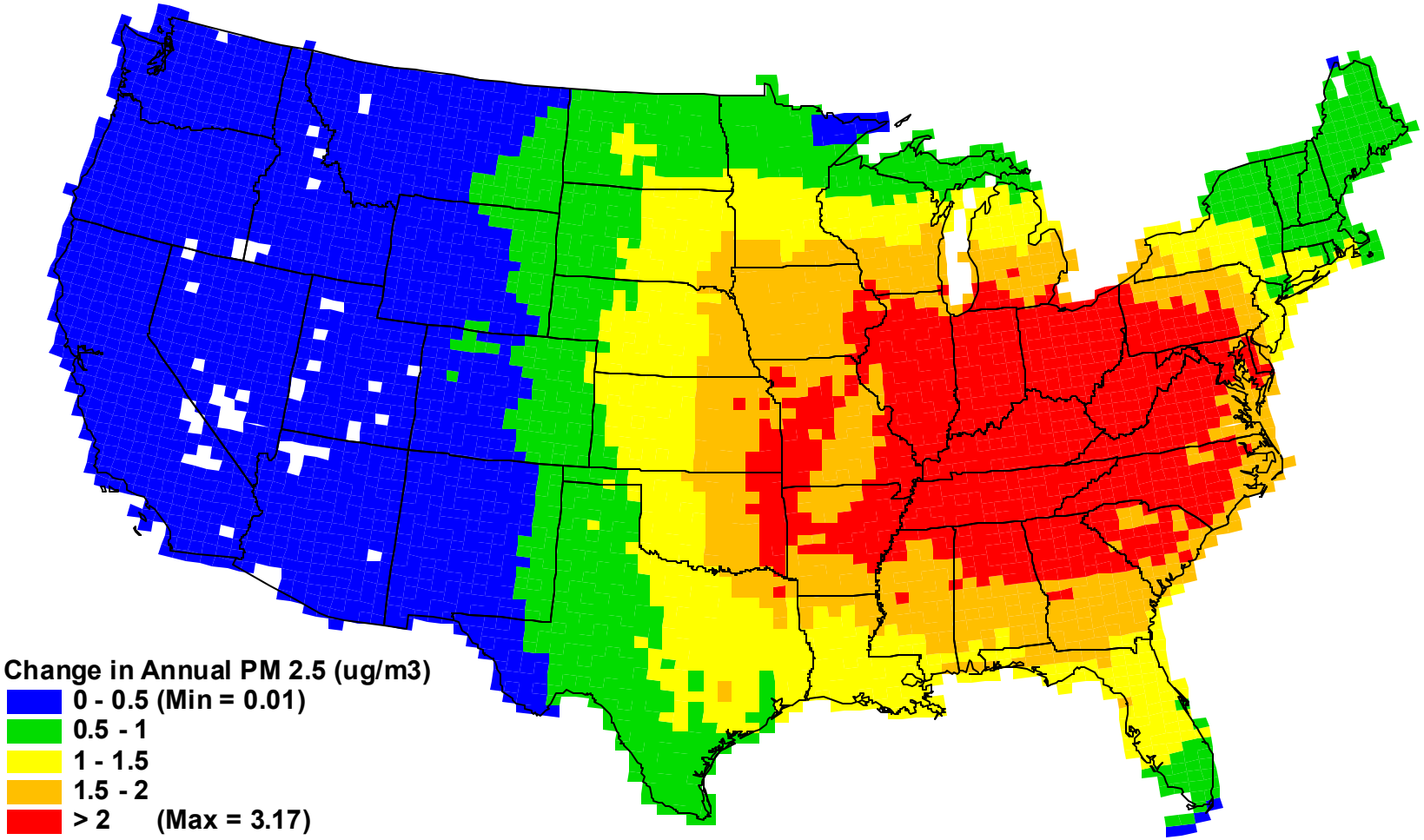


Exhibit 3.11 Change in 2020 Annual Mean $PM_{2.5}$ Levels with Jeffords Bill



4 Issues in Estimating Health Benefits

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

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 BenMAP 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.

Basic Concentration-Response Model

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

Different epidemiological studies may have estimated the relationship between PM and a particular health endpoint in different locations. The C-R functions estimated by these different studies may differ from each other in several ways. They may have different functional forms; they may have measured PM concentrations in different ways; they may have characterized the health endpoint, y, in slightly different ways; or they may have considered different types of populations. For example, some studies of the relationship between ambient PM concentrations and mortality have excluded accidental deaths from their mortality counts; others have included all deaths. One study may have measured daily (24-hour) average PM concentrations while another study may have used two-day averages. Some studies have assumed that the relationship between y and PM is best described by a linear form (i.e., the relationship between y and PM is estimated by a linear regression in which y is the dependent variable and PM is one of several independent variables). Other studies have assumed that the relationship is best described by a log-linear form (i.e., the relationship between the natural logarithm of y and PM is estimated by a linear regression).[1] Finally, one study may have considered changes in the health endpoint only among members of a particular subgroup of the population (e.g., individuals 65 and older), while other studies may have considered the entire population in the study location.

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

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

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

or, equivalently,

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

where the parameter B is the incidence of y when the concentration of PM is zero, the parameter β is the coefficient of PM, ln(y) is the natural logarithm of y, and $\alpha = \ln(B)$.[3] If the functional form of the C-R relationship is log-linear, the relationship between ΔPM and Δy is:

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

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

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

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

[3] 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, $B = B_o \exp\{\beta_1 x_1 + \ldots + \beta_n x_n\}$, where B_o is the incidence of y when all covariates in the model are zero, and $x_1, \ldots, x_n$ are the other covariates evaluated at their mean values. The parameter B drops out of the model, however, when changes in incidences are calculated, and is therefore not important.

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

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

The linear relationship is of the form:

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

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

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

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

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

Calculation of Adverse Health Effects with BenMAP

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

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

equal to the standard error of the estimate. Using a Latin Hypercube method,[4] 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 BenMAP grid cell. This distribution is stored, and BenMAP proceeds to the next grid cell, where the process is repeated. We calculate the distribution of the national change (or change in a designated geographical area) by summing the n^{th} percentile grid cell-specific changes, for n = 0.5, 1.5, ..., 99.5.

Overlapping Health Effects

Several endpoints reported in the health effects literature overlap with each other. For example, hospital admissions for single respiratory ailments (e.g. pneumonia) overlap with estimates of hospital admissions for "all respiratory" ailments.[5] 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 considers the issue of double-counting benefits that might arise from overlapping health effects.

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 REMSAD/BenMAP 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 BenMAP 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 Appendix A.

Thresholds

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[4]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).

[5]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.

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.

None of the epidemiological functions relating PM to various health endpoints incorporate thresholds. Instead, all of these functions are continuous and differentiable down to zero pollutant levels. A threshold may be imposed on these models, however, in several ways, and there are various points at which the threshold could be set. (A threshold can be set at any point. There are some points, however, that may be considered more obvious candidates than others.) One possible threshold might be the background level of the pollutant. Another might be 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 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:

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

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

$$\ln(y) = \alpha' \ , \ \ for \ PM \le T \ , and$$
$$\ln(y) = \alpha' + \beta' \cdot PM \ \ , \ for \ PM > T \ .$$

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 (equation (6)), 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.

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.

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.

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

Many studies include multiple pollutants, like ozone and particulate matter, in their final models. For this analysis, however, we are estimating benefits for only particulate matter. This presents a challenge because 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 PM-specific benefit 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.

Though this analysis estimates the benefits associated with reductions in PM alone, it is worth mentioning that there is the possibility of mis-characterizing benefits if some of the studies used are single pollutant models. Suppose, for example, that only ozone is actually associated with a given endpoint, but PM appears to be associated only because it is correlated with ozone. The benefits predicted by a single pollutant PM model would, in that case, actually reflect the benefits of reducing ozone, to the extent that PM and ozone are correlated. If only one pollutant is being associated with the endpoint in this analysis (e.g., chronic bronchitis is associated only with PM in this analysis), this is not a problem.

Pooling Study Results

When only a single study 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.[6]

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[6] 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

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.

Valuing Changes in Health Effects

This section discusses a number of issues that arise in valuing changes in health 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 inflation issues are addressed. The WTP estimates were originally calculated in a variety of different years, and hence reflect values in values expressed in the a variety of different inflation amounts. The fourth section describes how we adjust the original WTP estimates dollars to correct for inflation to get estimates in 1999 dollars. In the last section, we briefly review how we aggregate benefits estimates.

Willingness To Pay Estimation

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

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violate the assumption of the "fixed effects" model. However, applying a random effects model assumes that the observed set of coefficients is representative of coefficients in the policy region.

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, 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:

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

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:

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

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.[7] Derivation of the dollar value estimates discussed below was often limited by available information.

Change Over Time in WTP in Real Dollars

The WTP for health-related environmental improvements (in real dollars) could change between now and the years 2010 and 2020. If real income increases between now and the year 2010, for example, it is reasonable to expect that WTP, in real dollars, would also increase. Below we summarize the evidence regarding this effect, however we do not adjust our results in this analysis, because of the uncertainty regarding the size of the effect.

Based on historical trends, the U.S. Bureau of Economic Analysis projects that, for the United States as a whole as well as for regions and states within the U.S., mean per capita real income will increase. For the U.S. as a whole, for example, mean per capita personal income is projected to increase by about 16 percent from 1993 to 2005 (U.S. Bureau of Economic Analysis, 1995).

The mean WTP in the population is the correct measure of the value of a health problem avoided, and that WTP is a function of income. If the mean per capita real income rises by the year 2010, the mean WTP would probably rise as well. While this is most likely true, the degree to which mean WTP rises with a rise in mean per capita income is unclear unless the elasticity of WTP with respect to changes over time in real income is 1.0.

There is some evidence (Loehman and De, 1982; Mitchell and Carson, 1986; Alberini et al., 1997) that the elasticity of WTP for health-related environmental improvements with respect to real income is less than 1.0, possibly substantially so. If this is the case, then changes in mean income cannot be readily translated into corresponding changes in mean WTP. Although an increase in mean income is likely to imply an increase in mean WTP, the degree of the increase cannot be ascertained from information only about the means.

Several factors, in addition to real income, that could affect the estimated benefit associated with reductions in air pollution concentrations could also change in the future Demographic characteristics of exposed populations could change. Technological advances could change both the nature of precursor emissions to the ambient air and the susceptibility of individuals to air pollution. Any such changes would be reflected in C-R functions that differ from those that describe current relationships between ambient concentrations and the various health endpoints. While adjustments of WTP to reflect changes in real income are of interest, such adjustments would by no means necessarily reflect all possible changes that could affect the future benefits of reduced air pollution.

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[7] 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).

Adjusting Benefits Estimates to Year 1999 Dollars

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

Table 4-1 Bases of Benefits Estimation

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

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

Benefits estimates based directly on estimates of WTP have been adjusted using the CPI-U for "all items." The CPI-Us, published by the U.S. Dept. of Labor, Bureau of Labor Statistics, can be found in Council of Economic Advisers (2004, 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 Table 4-2.

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

	1990 (1)	1999 (2)	Adjustment Factor [a] (2)/(1)	Relevant Endpoints
CPI-U for "All Items" [b]	130.7	166.6	1.275	WTP-based valuation: 1. Statistical lives saved [c] 2. Chronic bronchitis 3. Morbidity endpoints using WTP [d]
CPI-U for Medical Care [b]	162.8	250.6	1.539	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, 1997).

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 1997 would be $105.1, or rounded to the nearest dollar, $105.

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.

As stated earlier, 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 j^{th} 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.[8] 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. 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:

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[8]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.

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

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:

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

The total monetized PM-related benefit to be estimated for a location is thus a function of 2N parameters: the coefficient of PM, β_{jk}, in the C-R function for the j^{th} health 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.

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:

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

There are many locations in the United States, however, and the individual location-specific values of β_j (the β_{jk}'s) are not known.[9] 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:

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

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

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

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

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

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

The total regional monetized (PM-related) benefit is thus a function of 2N population means: the β* for the j^{th} health 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).

Both the endpoint-specific coefficients (the $\ddot{y}_j$'s) and the endpoint-specific mean WTPs (the $MWTP_j$'s) are uncertain. 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 effect. Second, we randomly select a value from the distribution

[9]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.

of unit dollar values for that health effect. And third, we multiply the two values. The result is a distribution of (5000) monetized benefits associated with the given health effect. From this distribution, we present the mean as well as the 5th and 95th percentiles. We discuss the background of the Monte Carlo in the following sub-section.

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.

Table 4-3 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.

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. Of the primary endpoints we do quantify, a number of alternative measures of mortality incidence can be calculated. We present the full suite of alternative mortality calculations as a way to address the range of plausible mortality incidence estimates. This is discussed in greater detail in Chapter 5.

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

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

1. Uncertainties Associated With Concentration-Response Functions
-The value of the PM-coefficient in each C-R function.
-Application of a single C-R function to pollutant changes and populations in all locations.
-Similarity of future year C-R relationships to current C-R relationships.
-Correct functional form of each C-R relationship.
-Extrapolation of C-R relationships beyond the range of PM concentrations observed in the study.
2. Uncertainties Associated With PM Concentrations
-Estimating future-year baseline daily PM concentrations.
-Estimating the change in PM resulting from the control policy.
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 responsible for reported health effects 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 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
-What portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year, and what portion 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 well approximate what baseline incidence rates will be in the year 2030.
-Projected population and demographics -- used to derive incidences – may not well approximate future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
-Unit dollar values associated with health are only estimates of mean WTP and therefore have uncertainty surrounding them.
-Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors.
7. Uncertainties Associated With Aggregation of Monetized Benefits
-Health benefits estimates are limited to the available C-R functions. Thus, unquantified benefit categories will cause total benefits to be underestimated.

Statistical Uncertainty Bounds

Although there are several sources of uncertainty affecting estimates of endpoint-specific benefits, the sources of uncertainty that are most readily quantifiable in this analysis are the incidence changes (deriving from uncertainty about the C-R relationships) and uncertainty about unit dollar values. The total dollar benefit associated with a given endpoint depends on how much the endpoint will change due to the final standard (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth).[10] Based on these distributions, we provide estimates of the 5th and 95th percentile values of the distribution of estimated benefits. However, we hasten to add that this omits important sources of uncertainty, such as the contribution of air quality changes, baseline population incidences, projected populations exposed, transferability of the C-R function to diverse locations, and uncertainty about premature mortality. Thus, a confidence interval based on the standard error would provide a misleading picture about the overall uncertainty in the estimates. The empirical evidence about uncertainty is presented where it is available.

Both the uncertainty about the incidence changes and uncertainty about unit dollar values can be characterized by *distributions*. Each "uncertainty distribution" characterizes our beliefs about what the true value of an unknown (e.g., the true change in incidence of a given health effect) is likely to be, based on the available information from relevant studies.[11] 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.[12] If this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated.

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[10] 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^*.

[11] 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 Kennedy1990, pp. 168-172).

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

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.

Unquantified Benefits

In considering the monetized benefits estimates, the reader should remain aware of the limitations. One significant limitation of benefits analyses is the inability to quantify many of the PM adverse effects. For many effects, reliable C-R functions and/or valuation functions are not currently available such as infant mortality. In general, if it were possible to monetize these benefits categories, the benefits estimates presented here would increase.

5 Health Benefits

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

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

Table 5-1 PM-Related Health Endpoints

Endpoint	Population	PM Measure	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, 63 city Dichotomous samplers.
Chronic Illness			
Chronic Bronchitis	Ages 27+	$PM_{2.5}$	Abbey et al. (1995c)
Heart Attacks			
Acute Myocardial Infarction(Non-fatal)	Ages 18+	$PM_{2.5}$	Peters et al. (2001)
Hospital Admissions			
Chronic Lung Disease Less Asthma(ICD codes 490-492, 494-496)	Ages 18-64	$PM_{2.5}$	Moolgavkar (2000c)
Asthma (ICD code 493)	< 65	$PM_{2.5}$	Sheppard et al. (1999)
Pneumonia (ICD-9 codes 480-487)	Ages 65+	$PM_{2.5}$	Lippmann et al. (2000, Detroit)
Chronic Lung Disease (ICD codes 490-496)	Ages 65+	$PM_{2.5}$	Pooled Estimate: Lippmann et al. (2000), Moolgavkar (2000b)
Cardiovascular (ICD codes 390-409, 411-429)	Ages 20-64	$PM_{2.5}$	Moolgavkar (2000a, Los Angeles)
Cardiovascular ((ICD codes 390-409, 411-429)	age 65+	$PM_{2.5}$	Pooled Estimate: Moolgavkar (2000a), Lippmann et al. (2000)
Asthma-related ER visits (ICD code 493)	< 18	$PM_{2.5}$	Norris et al. (1999)
Respiratory Symptoms/Illnesses Not Requiring Hospitalization			
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1996)
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)

[a] 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.

[b] 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.

Table 5-2 Unit Values for Economic Valuation of Health Endpoints (1999 $)

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

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

[b] An adjustment for lagged mortality, discussed in the text, is used in this analysis. The lag-adjusted value of a statistical life is approximately 92% of the full value presented here.

[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.

Health Endpoint	Mean Estimate [a]	Uncertainty Distribution [a]

[d] 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.

Premature Mortality

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

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.

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

Table 5-3 Alternative Mortality Concentration-Response Functions

Endpoint	Population	PM Indicator	Study
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, 63 city Dichotomous sampler
Associated with long-term exposure	Ages 25+	$PM_{2.5}$	Krewski et al., 2000 - Reanalysis of Dockery et al. (1993)
Associated with long-term exposure(Lung Cancer)	Ages 30+	$PM_{2.5}$	Pope et al., 2002 - Based on ACS Cohort: Mean $PM_{2.5}$

Short-Term Versus Long-Term Studies

Long-term studies (e.g., Krewski et al., 2000, and 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(1995) or the Krewski et al (2000)) 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. To avoid double counting, as well as issues involving short-term harvesting(discussed below in detail), we have used only long-term studies for this analysis.

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. However, there is little evidence to bear on this question. 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. A recent study by Zeger et al. (1999), that focused exclusively on this issue, reported that short-term harvesting may be a quite small fraction of mortality.[13]

It is not likely, however, that the excess mortality reported in a long-term prospective cohort studies like Pope et al. (1995) or Krewski et al. (2000), contain 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.

In developing and improving the methods for estimating and valuing the potential reductions in mortality risk over the years, EPA has consulted with a panel of the Science Advisory Board(SAB). That panel recommended use of long-term prospective cohort studies in estimating mortality risk reduction (U.S. EPA, 1999a). This recommendation has been confirmed by a recent report from the National Research Council, which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure (National Research Council, 2002, p. 108). The Krewski et al. analysis also includes a broader geographic scope than the original study (63 cities versus 50). The SAB has recently agreed with EPA's selection of this specification for use in analyzing mortality benefits of PM reductions (U.S. EPA, 2001).

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

It is not possible to estimate with any degree of confidence how premature is the PM-related mortality. Making such an estimate requires considerable more understanding of the relationships between PM and human health than is currently available. As the amount of prematurity is potentially a very important issue for public policy, however, EPA did develop an estimate. Using an approach developed by the World Health Organization, the EPA estimated that "The average number of life-years lost by individuals dying prematurely from exposure to PM is 14 years." (*Final Report to Congress on Benefits and Costs of the Clean Air Act, 1970 to 1990*", EPA 410-R-97-002 p. I-23.

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. (1995) 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 Report (U.S. EPA, 1999a, p. 12). An advantage of Krewski et al. (2000) over Pope et al. (1995) is that Krewski et al.'s (2000) reanalysis of the Pope data 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. However, estimates of annual mean levels are inherently less stable than annual median estimates, and are more sensitive to the estimates on the highly polluted days. Specifically, we use the Krewski results (Table 31, Krewski et al. (2000)) based on dichotomous samplers in 63 cities (rather than the 50 cities used in the Pope et al. $PM_{2.5}$ analysis).

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, and more locations (63 cities) in the United States, than other studies. We also consider the Krewski study preferable to the original Pope et al. (1995) study because it uses the annual mean $PM_{2.5}$ rather than the median.

It is unlikely that the Krewski et al. 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 and Pope 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 Krewski et al., 2000 - Reanalysis of Dockery et al. (1993), the Krewski et al. study 2000-Reanalysis of Pope et al.(1995) study 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 (EPA-SAB-COUNCIL-ADV-00-001, 1999). It should be noted that the selection of a five-year lag structure is not directly supported by any PM-specific literature. Rather, it is intended to be a best guess at the appropriate distribution of avoided incidences of PM-related mortality.

(1) Alternative Calculation: PM-Related Mortality Based on Krewski et al., 2000 - Reanalysis of Dockery et al. (1993)

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

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

(2) Alternative Calculation: Mortality, Lung Cancer (Pope et al., 2002) - Based on ACS Cohort: Mean $PM_{2.5}$

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

Valuing Premature Mortality

The "statistical lives lost" approach to valuing premature mortality estimates the value of a statistical death to be $6.12 million (in 1999 $). We assume for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the five years following exposure (the

five-year mortality lag). To take this into account in the valuation of reductions in premature mortalities, we apply an annual five percent discount rate to the value of premature mortalities occurring in future years.[14]

• Statistical Lives Lost

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

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 Table 5-4. The references for all but Gegax et al. (1985) and V.K. Smith (1983) may be found in Viscusi (1992). Although each of the studies estimated the mean WTP (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 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.

[14]The choice of a five percent discount rate is based on the technical recommendation of the SAB for computing the value of a statistical life-year (U.S. EPA, 1999c, p. 14).

Table 5-4 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), although 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 Table 5-5. Although most of the individual factors tend to have a downward bias, the overall effect of these biases is unclear.

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

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

Chronic Illness

Researchers have linked air pollution with a variety of adverse health effects that have long-term, or chronic implications. The onset of bronchitis has been associated with exposure to air pollutants. Studies have linked the onset of chronic bronchitis in adults to particulate matter (Schwartz, 1993; Abbey et al., 1995c). 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).

Chronic Bronchitis

Chronic bronchitis is characterized by mucus in the lungs and a persistent wet cough for at least three months a year for several years in a row, and affects roughly five percent of the U.S. population (American Lung Association, 2002b, Table 4). There are a limited number of studies that have estimated the impact of air pollution on new incidences of chronic bronchitis. Schwartz (1993) and Abbey et al.(1995c) provide evidence that long-term PM exposure gives rise to the development of chronic bronchitis in the U.S.

We estimate the changes in the number of new cases of PM-related chronic bronchitis using a study by Abbey et al. (1995c) which is based on a sample of California residents. The study by Abbey et al. (1995c) examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh-Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for airway obstructive disease (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.

Table 5-6 Chronic Bronchitis Study

Location	Study	Pollutants Used in Final Model	Age of Study Population
California	Abbey et al. (1995c)	$PM_{2.5}$	>26

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

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

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

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

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

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

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

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

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

The derivation of the WTP to avoid a case of pollution-related chronic bronchitis is based on three components, each of which is uncertain: (1) the WTP to avoid a case of severe CB, as described in the Viscusi et al. (1991) study, (2) the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi et al.), and (3) the elasticity of WTP with respect to severity of the illness. Because of these three sources of uncertainty, the WTP is uncertain. Based on assumptions about the distributions of each of the three uncertain components, a distribution of WTP to avoid a pollution-related case of CB was derived by Monte Carlo methods. The mean of this distribution, which was about $319,000 ($331,000 in 1999$), is taken as the central tendency estimate of WTP to avoid a pollution-related case of CB. Each of the three underlying distributions is described briefly below.

1. The distribution of WTP to avoid a severe case of CB was based on the distribution of WTP responses in the Viscusi et al. (1991) study. Viscusi et al. derived respondents' implicit WTP to avoid a statistical case of chronic bronchitis from their WTP for a specified reduction in risk. The mean response implied a WTP of about $1,275,000 (1999 $)[15]; the median response implied a WTP of about $676,000 (1999

[15]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.

$). However, the extreme tails of distributions of WTP responses are usually considered unreliable. Because the mean is much more sensitive to extreme values, the median of WTP responses is often used rather than the mean. Viscusi et al. report not only the mean and median of their distribution of WTP responses, however, but the decile points as well. The distribution of reliable WTP responses from the Viscusi et al. study could therefore be approximated by a discrete uniform distribution giving a probability of 1/9 to each of the first nine decile points. This omits the first five and the last five percent of the responses (the extreme tails, considered unreliable). This trimmed distribution of WTP responses from the Viscusi et al. study was assumed to be the distribution of WTPs to avoid a severe case of CB. The mean of this distribution is about $918,000 (1999 $).

2. The distribution of the severity level of an average case of pollution-related CB was modeled as a triangular distribution centered at 6.5, with endpoints at 1.0 and 12.0. These severity levels are based on the severity levels used in Krupnick and Cropper (1992), which estimated the relationship between ln(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 $331,000 (1999$).

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

Heart Attacks

Non-Fatal Myocardial Infarction (Heart Attacks)

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

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

Valuing Non-Fatal Myocardial Infarction (Heart Attack)

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

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

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

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

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

Table 5-7. Summary of Studies Valuing Reduced Incidences of Myocardial Infarction

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

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

Table 5-8. Estimated Costs Over a 5-Year Period of a Non-Fatal Myocardial Infarction

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

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

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

Hospital Admissions

We estimate the impact of PM on both respiratory and cardiovascular hospital admissions. In addition, we estimate the impact of these pollutants on emergency room visits for asthma. The respiratory and cardiovascular hospital admissions studies used in the primary analysis are listed in Tables 5-7 and 5-8, respectively. Appendix A provides details on each study. Due to the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, we pooled some of the hospital admission endpoints, using the results of a number of studies. Although the benefits associated with respiratory and cardiovascular hospital admissions are estimated separately in the analysis, the methods used to estimate changes in incidence and to value those changes are the same for both broad categories of hospital admissions. The two categories of hospital admissions are therefore discussed together in this section.

Table 5-9 Respiratory Hospital Admission Studies

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

Table 5-10 Cardiovascular Hospital Admission Study

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
PM-Related Hospital Admissions				
Los Angeles, CA	Moolgavkar (2000a)	Cardiovascular (ICD codes 390-409, 411-429)[16]	$PM_{2.5}$	Ages 20-64
Los Angeles (Moolgavkar), Detroit (Lippman)	Moolgavkar (2000a), Lippmann et al. (2000)	Cardiovascular ((ICD codes 390-409, 411-429)[17]	$PM_{2.5}$	age 65+

PM-Related Respiratory and Cardiovascular Hospital Admissions

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

To estimate total avoided incidences of respiratory hospital admissions, we use C-R functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, there are additional published studies showing a statistically significant relationship between PM_{10} and respiratory hospital admissions. We use only those focusing on $PM_{2.5}$. Both Moolgavkar (2000a) and Lippmann et al (2000) estimated C-R functions for COPD in populations over 65, allowing us to pool the C-R functions for this group. Only Moolgavkar estimated a separate C-R function for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Lippmann et al estimated pneumonia,

[16] Moolgavkar (2000a) reports results that include ICD code 410 (heart attack). In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

[17] Moolgavkar (2000a) reports results for ICD codes 390-429. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

and only for the population 65 and older. In addition, Sheppard et al (1999) estimated a C-R function for asthma hospital admissions for populations under age 65. Total avoided incidences of PM-related respiratory-related hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

Valuing Respiratory and Cardiovascular Hospital Admissions

Society's WTP to avoid a hospital admission includes medical expenses, lost work productivity, the non-market costs of treating illness (i.e., air, water and solid waste pollution from hospitals and the pharmaceutical industry), and the pain and suffering of the affected individual as well as of that of relatives, friends, and associated caregivers.[18]

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

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

Following the method used in the §812 analysis (U.S. EPA, 1999b), ICD-code-specific COI estimates used in our analysis consist of two components: estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). The opportunity cost of a day spent in the hospital is estimated as the value of the lost daily wage, regardless of whether or not the individual is in the workforce. This is estimated at $106 (U.S. Bureau of the Census, 1992).

For all hospital admissions included in this analysis, estimates of hospital charges and lengths of hospital stays were based on discharge statistics provided by Elixhauser et al. (1993). 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

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[18] 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.

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, 1999 #1872]. The study-specific values for valuing respiratory and cardiovascular hospital admissions are shown in Tables 5-9 and 5-10, respectively.

The mean hospital charges and mean lengths of stay provided by Elixhauser et al. (1993) 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.

Table 5-11 Unit Values for Respiratory Hospital Admissions*

Location	Study	Endpoints Estimated (ICD code)	Age of Study Population	COI [a] (1999 $)
PM-Related Hospital Admissions				
Los Angeles, CA	Moolgavkar (2000c)	Chronic Lung Disease Less Asthma(ICD codes 490-492, 494-496)	Ages 18-64	$11,333
Seattle, WA	Sheppard et al. (1999)	Asthma (493)	<65	$7,467
Detroit, MI	Lippmann et al. (2000)	Pneumonia (ICD-9 codes 480-487)	Ages 65+	$17,106
Detroit (Lippman) Chicago, Los Angeles, and Phoenix (Moolgavkar)	Lippmann et al. (2000), Moolgavkar (2000b)	Chronic Lung Disease (ICD codes 490-496)	Ages 65+	$13,083

Location	Study	Endpoints Estimated (ICD code)	Age of Study Population	COI [a] (1999 $)
Seattle, WA	Norris et al. (1999)	Asthma-related ER visits (ICD code 493)	< 18	$275

[*] The unit value for a group of ICD-9 codes is the weighted average of ICD-9 code-specific values, from Elixhauser et al. (1993). The weights are the relative frequencies of hospital discharges in Elixhauser et al. for each ICD-9 code in the group. 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. (2000) of people age >64.

Table 5-12 Unit Values for Cardiovascular Hospital Admissions*

Location	Study	Endpoints Estimated (ICD code)	Age of Study Population	COI [a] (1999 $)
PM-Related Hospital Admissions				
Los Angeles, CA	Moolgavkar (2000a)	Cardiovascular (ICD codes 390-409, 411-429)[19]	Ages 20-64	$21,864(ICD codes 390-429)
Los Angeles (Moolgavkar), Detroit (Lippman)	Moolgavkar (2000a), Lippmann et al. (2000)	Cardiovascular ((ICD codes 390-409, 411-429)[20]	age 65+	$20,334(ICD codes 390-429)

* The unit value for a group of ICD-9 codes is the weighted average of ICD-9 code-specific values, from Elixhauser et al. (1993). The weights are the relative frequencies of hospital discharges in Elixhauser et al. for each ICD-9 code in the group.

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.

Asthma-Related Emergency Room (ER) Visits

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

[19] Moolgavkar (2000a) reports results that include ICD code 410 (heart attack). In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

[20] Moolgavkar (2000a) reports results for ICD codes 390-429. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

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

Valuing Asthma-Related Emergency Room (ER) Visits

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 $298.62, on the interval [$221.65, $414.07].

A second unit value is $249.86($1999) from Stanford et al. (1999). This study considered asthmatics in 1996-1997, in comparison to the Smith et al. (1997) study, which used 1987 National Medical Expenditure Survey (NMES) data). In comparing their study, the authors note that the 1987 NMES, used by Smith et al., "may not reflect changes in treatment patterns during the 1990s." In addition, its costs are the costs to the hospital (or ER) for treating asthma rather than charges or payments by the patient and/or third party payer. Costs to the ER are probably a better measure of the value of the medical resources used up on an asthma ER visit (see above for a discussion of costs versus charges). An average of these two values gives an estimate of $275($1999) for an Asthma-Related ER visits.

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").

Table 5-13 Studies of Symptoms/Illnesses not Requiring Hospitalization

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

Acute Bronchitis

Around five percent of U.S. children between ages five and seventeen experience episodes of acute bronchitis annually (Adams and Marano, 1995). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia[21], with the exception of cough, most acute bronchitis symptoms abate within 7 to 10 days. We estimated the incidence of episodes of acute bronchitis in children between the ages 8-12 using a C-R function developed from Dockery et al. (1996).

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

Three unit values are available in BenMAP for acute bronchitis in children. In previous benefits analyses, EPA used a unit value of $57.38. This is the midpoint between a low estimate and a high estimate. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day.

The above unit value assumes that an episode of acute bronchitis lasts only one day. However, this is generally not the case. More typically, it can last for 6 or 7 days. A simple adjustment, then, would be to multiply the original unit value of $57.38 by 6 or 7. A second unit value of $344 (=$57.38 x 6) was therefore derived.

Finally, as noted above, the epidemiological study relating air pollution to the incidence of acute bronchitis referred to children specifically. The value of an avoided case should therefore be WTP to avoid a case in a child, which may be different from WTP to avoid a case in an adult. Recent work by Dickie and Ulery (2002) suggests, in fact, that parents are generally willing to pay about twice as much to avoid sickness in their children as in themselves.[22] In one of several models they estimated, the natural logarithm of parents' WTP was related both to the number of symptom-days avoided and to whether it was their child or themselves at issue. Dickie and Ulery noted that "experiencing all of the symptoms [considered in their study

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

[22] This is, to our knowledge, the only estimate, based on empirical data, of parental WTP for their children versus themselves.

– cough and phlegm, shortness of breath/wheezing, chest pain, and fever] for 7 days, or 28 symptom-days altogether, is roughly equivalent to a case of acute bronchitis ..." Using this model, and assuming that a case of acute bronchitis can be reasonably modeled as consisting of 28 symptom-days, we estimated parents' WTP to avoid a case of acute bronchitis in a child to be $358($1999).[23]

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 Table 5-12. The three individual symptoms listed in Table 5-12 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. Using the symptom symbols in Table 5-12, these seven possible symptom complexes are presented in Table 5-13. It is assumed that each of these seven URS complexes is equally likely.[24] The point estimate of MWTP to

[23] The mean household income among participants in the Dickie and Ulery CV survey was slightly higher than the national average. We therefore adjusted all WTP estimates that resulted from their models downward slightly, using an income elasticity of WTP of 0.147, the average of the income elasticities estimated in the four models in the study. The adjustment factor thus derived was 0.9738.

[24] 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

avoid an occurrence of URS is just an average of the seven estimates of MWTP for the different URS complexes – $18.70, or about $19 in 1990 $. This is $24.23 (=$19*1.275) in 1999 $. In the absence of information surrounding 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 Table 5-13, with a range of [$7, $33], or [$8.93, $42.08] in 1999 $.

Table 5-14 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.

most reasonable "default" assumption.

Table 5-15 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 Table 5-12.

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.

Lower Respiratory Symptoms (LRS)

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

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

Valuing 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 Table 5-14.[25]

Table 5-14 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: $15.07

Based on values reported in Table 5-12.

We assumed that each of the eleven types of LRS is equally likely.[26] The mean WTP to avoid a day of LRS as defined by Schwartz et al. (1994) is therefore the average of the mean WTPs to avoid each type of LRS, – $11.82. This is $15.07 (=1.275*$11.82) in 1999 $. This is the point estimate used in the benefit analysis for the dollar value for LRS as defined by Schwartz et al. The WTP estimates are based on studies which considered the value of a *day* of avoided symptoms, whereas the Schwartz et al. study used as its

[25] 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.

[26] 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.

measure a *case* of LRS. Because a case of LRS usually lasts at least one day, and often more, WTP to avoid a day of LRS should be a conservative estimate of WTP to avoid a case of LRS.

In the absence of information about the frequency of each of the seven types of LRS among all occurrences of LRS, the uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution of MWTPs in Table 5-12, with a range of [$5, $19], or [$6.37, $24.22] in 1999 $. This is the same procedure as that used in the URS uncertainty analysis.

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.

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 $42.06 (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 was mentioned in the two previous sections, the interval for URS is [$8.93, $42.06], and for LRS, the interval is [$6.37, $24.22].

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 Table 5-15.

Table 5-16 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

Minor Restricted Activity Days (MRADs)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRAD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. We developed separate coefficients for each year in the analysis (1976-1981), which were then combined for use in this analysis. The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (Ostro, 1987, Table IV) using the inverse of the variance as the weight.

Valuing Minor Restricted Activity Days (MRADs)

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

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

moreover, one level of severity of a symptom could induce restriction of activity for one individual while not doing so for another. The same is true for any particular combination of symptoms.

Given that there is inherently a substantial degree of arbitrariness in any point estimate of WTP to avoid a MRRAD (or other kinds of restricted activity days), the reasonable bounds on such an estimate must be considered. By definition, a MRRAD does not result in loss of work. WTP to avoid a MRRAD should therefore be less than WTP to avoid a WLD. At the other extreme, WTP to avoid a MRRAD should exceed WTP to avoid a single mild symptom. The highest IEc midrange estimate of WTP to avoid a single symptom is $15.72 (1990 $), or about $16, for eye irritation. The point estimate of WTP to avoid a WLD in the benefit analysis is $83 (1990 $). If all the single symptoms evaluated by the studies are not severe, then the estimate of WTP to avoid a MRRAD should be somewhere between $16 and $83. Because the IEc estimate of $38 falls within this range (and acknowledging the degree of arbitrariness associated with any estimate within this range), the IEc estimate is used as the mean of a triangular distribution centered at $38, ranging from $16 to $61. Adjusting to 1999 $, this is a triangular distribution centered at $48.43, ranging from $20.34 to $77.76.

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.

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 $105.8 in 1999$. This is the value used to represent work loss days (WLD). An uncertainty distribution for this endpoint was unavailable, therefore the same central estimate ($105.8) was used to value incidence changes at the fifth, mean, and ninety-fifth percentiles.

6 Results

This chapter provides estimates of the magnitude and value of changes in adverse health effects associated with each of the different policy scenarios we considered.

Tables 6-1 through 6-2 present the estimated number of avoidable health effects for each endpoint in each policy option. Tables 6.1 presents the results for 2010 (including the No-EGU analysis, which shows the number of attributable cases of health effects rather than avoidable health effects), and Table 6.2 presents the similar table for 2020. Tables 6-3 and 6-4 present the monetary value of the avoidable health effects for 2010 and 2020, respectively.

Additional details of the results shown in Tables 6-1 through 6.4 are included in Appendix B. The Tables in Appendix B provide uncertainty ranges (5^{th} and 95^{th} percentile values) of the health and valuation estimates.

The estimates of premature mortality included in this report are all based on estimates of the risk of dying attributable to the estimated PM levels in each policy option. As described in Chapter 5, these attributable risks from the estimated annual PM levels for each scenario are estimated in each location. The estimated mortal risk involve not only the changes in PM concentrations, but also data on the age-specific mortality rates in each location. Exhibits 6-1 through 6-10 are maps depicting the estimated mortality rates per 100,000 population from $PM_{2.5}$ from electricity generating units associated with each scenario. In addition to the risks from $PM_{2.5}$ from electricity generating units, there is additional risk from $PM_{2.5}$ coming from other sources. This additional, non-EGU risk is not shown on Exhibits 6-1 to 6-10.

As discussed in Chapter 5, additional epidemiology-based health research has been published since the time the health effects were selected for inclusion in EPA's Clear Skies Analysis. One such important new research paper is the Pope et al., 2002, paper. This research extends previously published results based on the American Cancer Society cohort tracking data.. The primary premature mortality estimates included in the EPA Clear Skies Analysis and in this paper are based earlier results from the ACS cohort data (Krewski et al., 2000). Along with using additional years of follow-on data than was previously available, Pope et al., 2002 also found a statistically significant relationship between $PM_{2.5}$ levels and a specific cause of death: lung cancer.

The EPA Clear Skies Analysis did not include estimates of deaths from lung cancer, so they are not included in the primary result set in this paper. It is possible, however, to use the lung cancer/PM relationship from the Pope et al., 2002 paper to estimate the numbers of avoidable lung cancer premature mortalities under each policy option considered in this paper. Table 6-5 presents estimates of the number of PM-related premature deaths from lung cancer, as well as the total mortality estimates previously presented.

The lung cancer mortality estimates are not additional deaths beyond the estimates from the Krewski et al., 2000 results. The mortality estimates from lung cancer are included in the total premature mortality estimates; the remaining cases of premature mortality (approximately 88 percent of the total) are from other causes, including both respiratory and cardio-vascular diseases.

In addition to the primary mortality estimate (which is based on Krewski et al., 2000 reanalysis of the American Cancer Society data), it is also possible to use other health studies as the basis of additional sensitivity estimates of mortality. Different health studies would produce different estimates of the avoidable cases of premature mortality. For example, a different estimate of the amount of premature mortality could

be based on the Krewski et al., 2000 reanalysis of the 6 Cities (Dockery et al., 1993) cohort data. The reanalysis of the 6 Cities data produced a relative risk factor nearly three times as high as the reanalysis of the American Cancer Society data. Therefore, using the 6 Cities reanalysis result produces almost three times as large an estimate of the numbers of cases of attributable premature mortality. For No EGU scenario, the 6 Cities reanalysis-based mortality relationship estimates there would be 67, 719 attributable cases of premature mortality in 2010, compared with 23,604 using the American Cancer Society cohort results..

Another health effect associated with exposure to PM are asthma attacks. Because of possible double counting with endpoints that are included (such as emergency room visits for asthma and upper respiratory symptom days), EPA does not quantify the number of asthma attacks. Using the methods previously used by EPA, there are 554,448 PM-related asthma attacks in the 2010 No EGU analysis.

Table 6-1 2010 Health Benefits Estimates: Numbers of Cases Reduced

	CSA	No EGU	Carper	Straw	Jeffords
Mortality	7,861	23,604	10,430	11,100	16,575
Chronic Bronchitis	5,400	16,221	7,160	7,615	11,397
Heart Attacks	13,115	38,198	17,218	18,244	27,039
Hospital Admissions-Respiratory					
Chronic Lung, less Asthma(20-64)	374	1,127	496	527	791
Asthma(0-64)	651	1,946	860	912	1,362
Pneumonia(65+)	2,653	8,040	3,515	3,733	5,628
Chronic Lung(65+)	332	1,000	441	468	702
Total Hospital Admissions-Respiratory	4,010	12,113	5,313	5,640	8,484
Hospital Admissions Cardiovascular					
All Cardiovascular,(20-64)	1,332	4,028	1,778	1,893	2,829
All Cardiovascular,(65+)	1,903	5,707	2,521	2,677	4,006
Total Hospital Admissions-Cardiovascular	3,235	9,735	4,299	4,570	6,835
Emergency Room Visits for Asthma	8,316	25,999	11,108	11,811	18,205
Acute Bronchitis	12,522	37,705	16,614	17,669	26,554
Lower Respiratory Symptoms	142,621	429,980	189,214	201,197	302,678
Upper Respiratory Symptoms	113,707	348,823	151,390	161,069	243,760
Work Loss Days	1,050,415	3,186,036	1,395,098	1,483,765	2,231,223

Minor Restricted Activity Days	6,258,491	18,916,818	8,306,310	8,832,956	13,265,510

Table 6-2 2020 Health Benefits Estimates: Numbers of Cases Reduced

	CSA	Carper	Straw	Jeffords
Mortality	14,104	16,166	18,355	21,749
Chronic Bronchitis	8,770	10,048	11,422	13,586
Heart Attacks	23,009	26,280	29,798	35,230
Hospital Admissions - Respiratory				
Chronic Lung, less Asthma (20-64)	610	699	794	945
Asthma (0-64)	1,151	1,145	1,302	1,545
Pneumonia (65+)	4,972	5,705	6,496	7,749
Chronic Lung (65+)	650	746	848	1,008
Total Hospital Admissions - Respiratory	7,383	8,295	7,513	11,247
Hospital Admissions Cardiovascular				
All Cardiovascular, (20-64)	2,139	2,452	2,782	3,296
All Cardiovascular, (65+)	3,632	4,165	4,731	5,615
Total Hospital Admissions - Cardiovascular	5,771	6,617	7,513	8,911
Emergency Room Visits for Asthma	13,223	15,191	17,373	21,050
Acute Bronchitis	19,919	22,823	25,971	31,013
Lower Respiratory Symptoms	226,616	259,649	295,492	353,091
Upper Respiratory Symptoms	181,286	208,106	237,294	284,295
Work Loss Days	1,602,343	1,837,341	2,091,325	2,495,685
Minor Restricted Activity Days	9,519,433	10,910,946	12,413,325	14,800,704

Table 6-3 2010 Value of Health Benefits (in millions of $1999)

	CSA	NoEGU	Carper	Straw	Jeffords
Mortality	$51,974	$149,274	$65,959	$70,198	$104,823
Chronic Bronchitis	$2,046	$5,523	$2,438	$2,592	$3,881
Heart Attacks	$1,127	$3,284	$1,480	$1,568	$2,324
Hospital Admissions - Respiratory					
Chronic Lung, less Asthma (20-64)	$4	$13	$6	$6	$9
Asthma (0-64)	$5	$15	$7	$7	$11
Pneumonia (65+)	$47	$143	$63	$67	$100
Chronic Lung (65+)	$4	$13	$6	$6	$9
Total Hospital Admissions - Respiratory	$60	$187	$82	$87	$132
Hospital Admissions Cardiovascular					
All Cardiovascular, (20-64)	$30	$92	$41	$43	$64
All Cardiovascular, (65+)	$39	$116	$51	$54	$81
Total Hospital Admissions - Cardiovascular	$69	$206	$92	$97	$146
Emergency Room Visits for Asthma	$2	$7	$3	$3	$5
Acute Bronchitis	$5	$13	$6	$7	$10
Lower Respiratory Symptoms	$2	$7	$3	$3	$5
Upper Respiratory Symptoms	$3	$9	$4	$4	$6
Work Loss Days	$136	$367	$161	$171	$257
Minor Restricted Activity Days	$327	$956	$420	$447	$670

Table 6-4 2020 Value of Health Benefits (in millions of $1999)

	CSA 2020	Carper	Straw	Jeffords
Mortality	$106,996	$117,302	$133,186	$157,813
Chronic Bronchitis	$3,880	$3,995	$4,540	$5,401
Heart Attacks	$1,961	$2,240	$2,540	$3,003
Hospital Admissions - Respiratory				
Chronic Lung, less Asthma (20-64)	$7	$8	$9	$11
Asthma (0-64)	$9	$9	$10	$12
Pneumonia (65+)	$89	$102	$116	$138
Chronic Lung (65+)	$9	$10	$11	$14
Total Hospital Admissions - Respiratory	$114	$131	$149	$177
Hospital Admissions Cardiovascular				
All Cardiovascular, (20-64)	$49	$56	$63	$75
All Cardiovascular, (65+)	$74	$84	$96	$114
Total Hospital Admissions - Cardiovascular	$123	$140	$159	$188
Emergency Room Visits for Asthma	$4	$4	$5	$6
Acute Bronchitis	$8	$9	$10	$12
Lower Respiratory Symptoms	$4	$4	$5	$6
Upper Respiratory Symptoms	$5	$6	$6	$8
Work Loss Days	$208	$212	$241	$288
Minor Restricted Activity Days	$522	$578	$658	$784

Exhibit 6-1 2010 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, 2010 Baseline

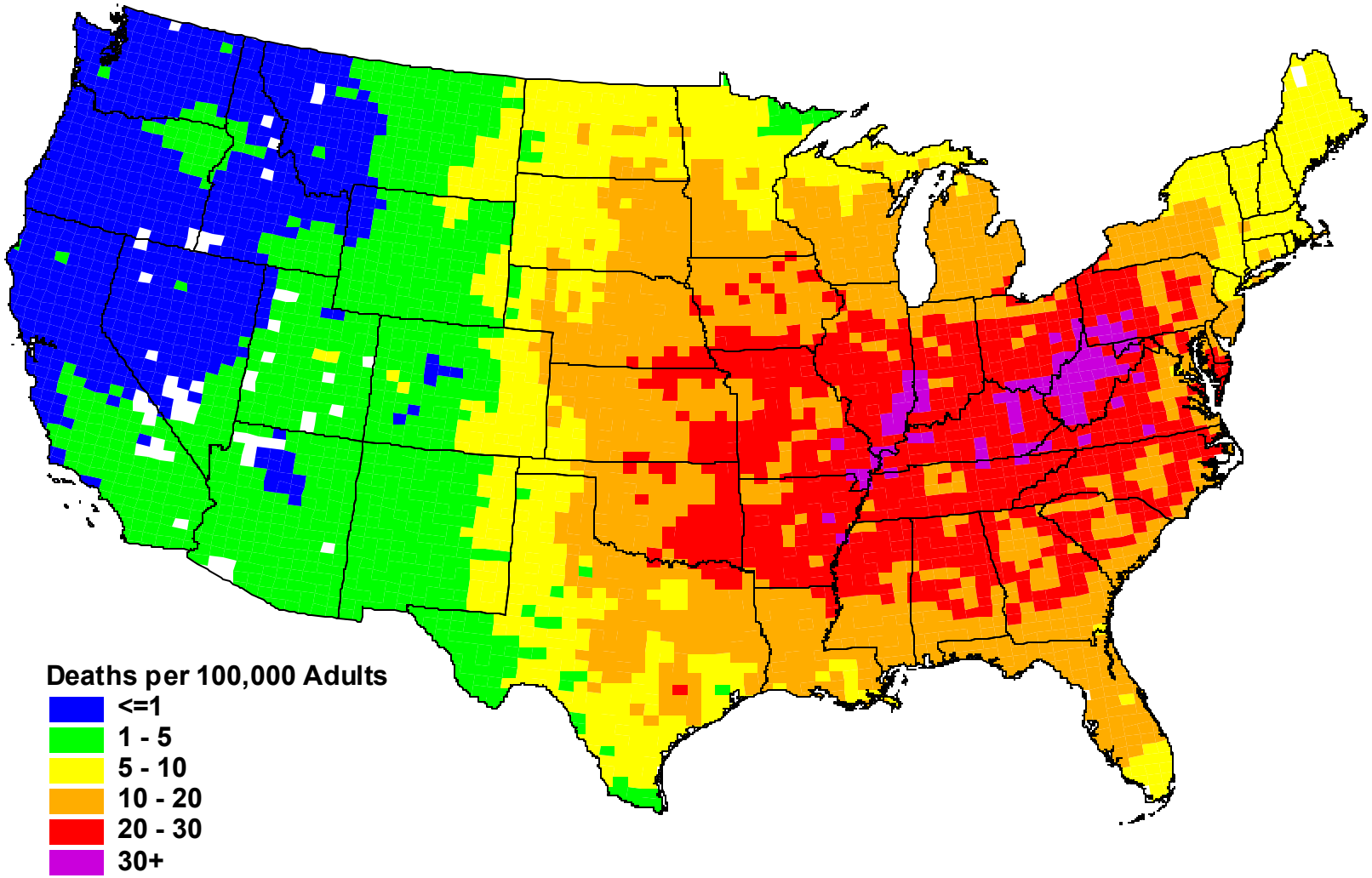

Exhibit 6-2 2010 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, With Clear Skies Act

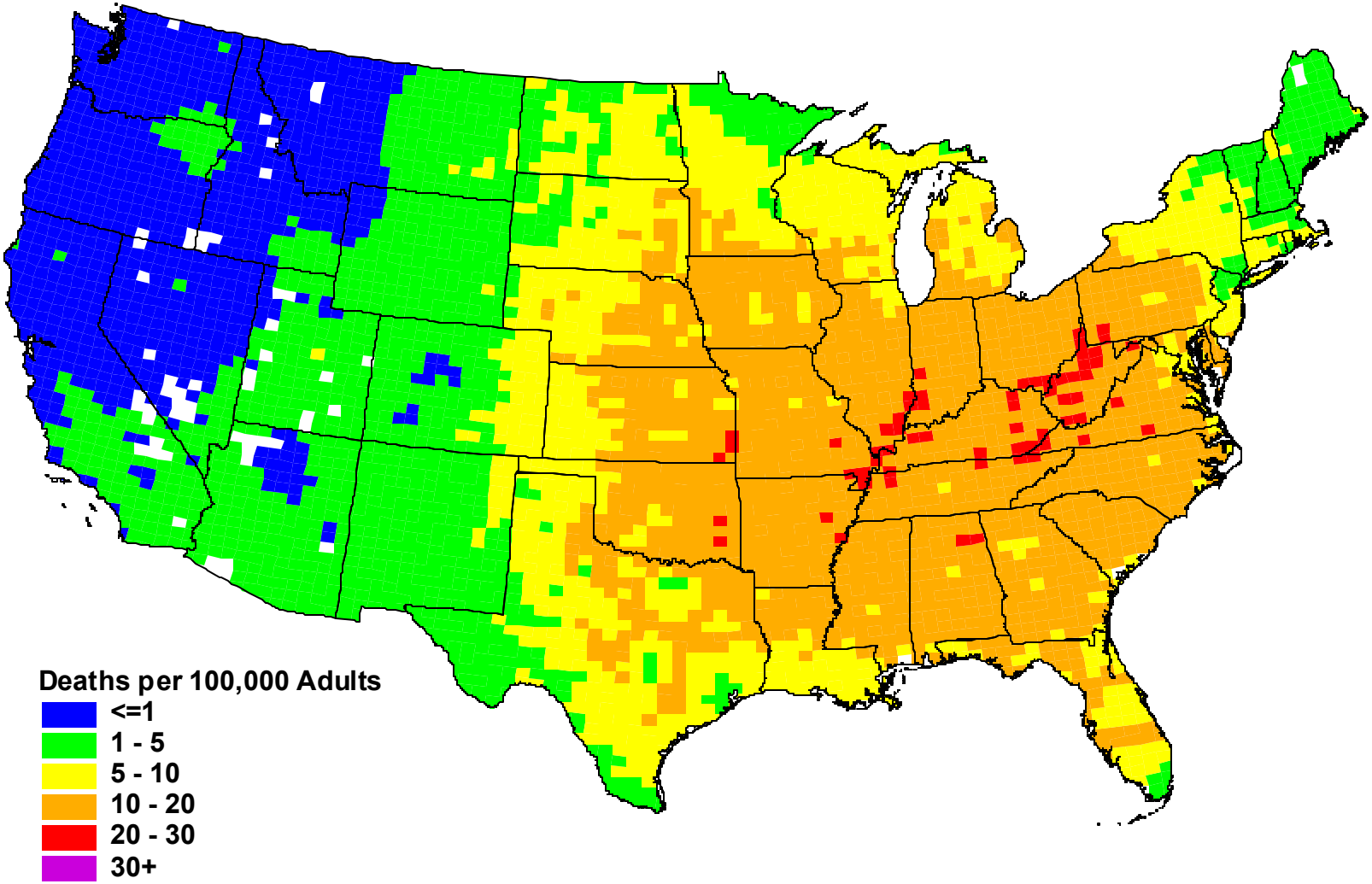

Exhibit 6-3 2010 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, With Carper

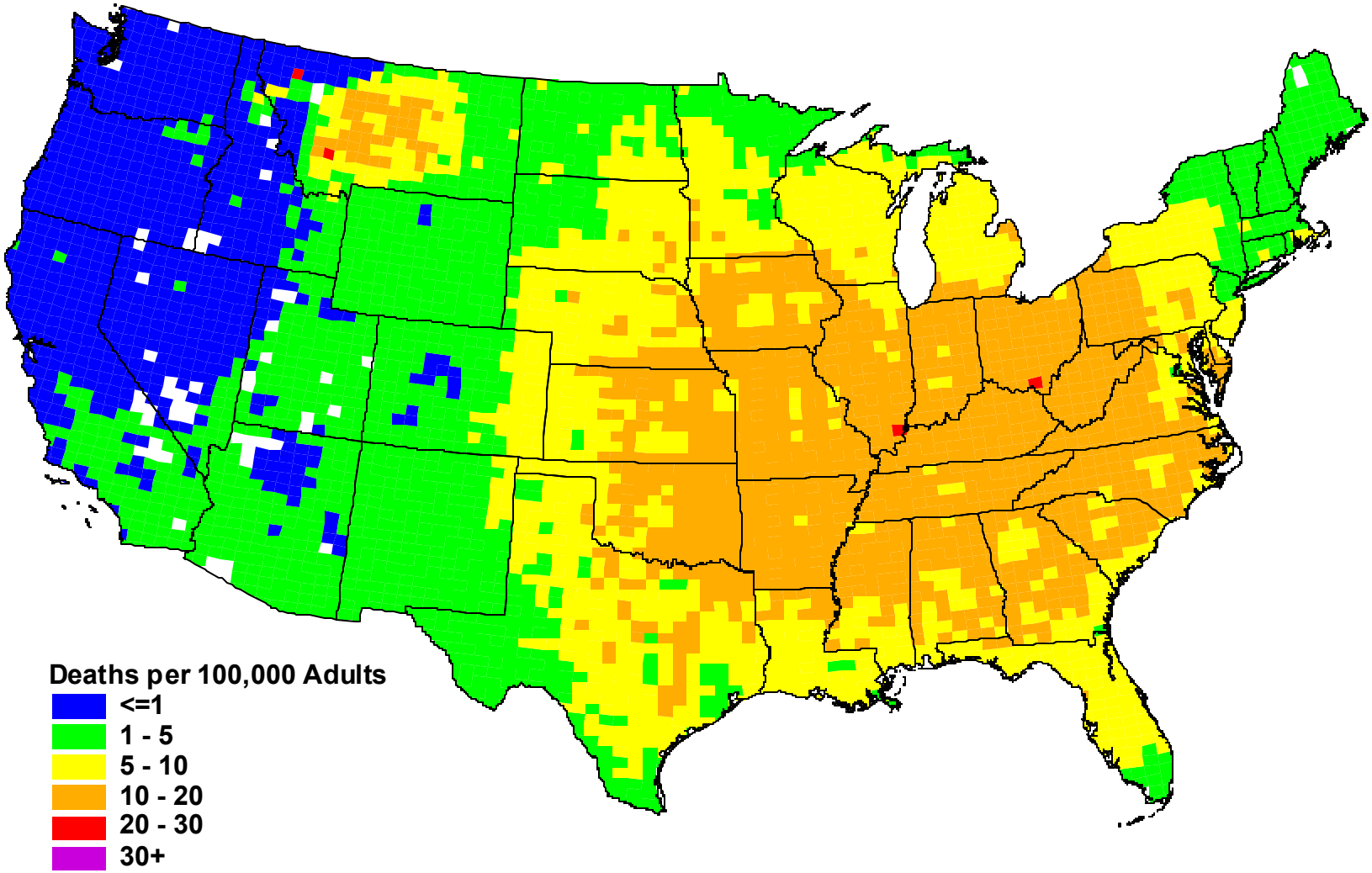

Exhibit 6-4 2010 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, With Straw Proposal

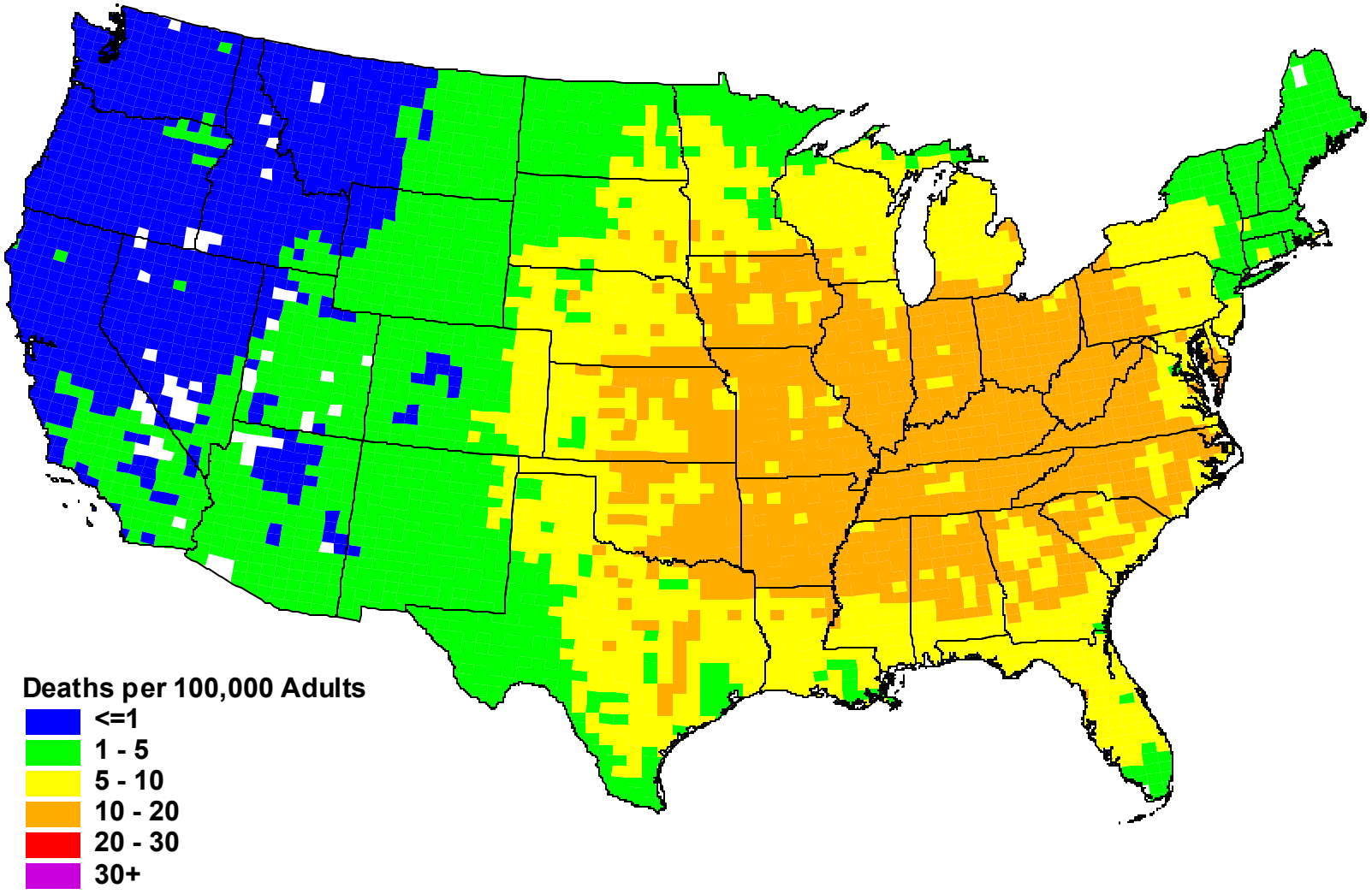

Exhibit 6-5 2010 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, With Jeffords

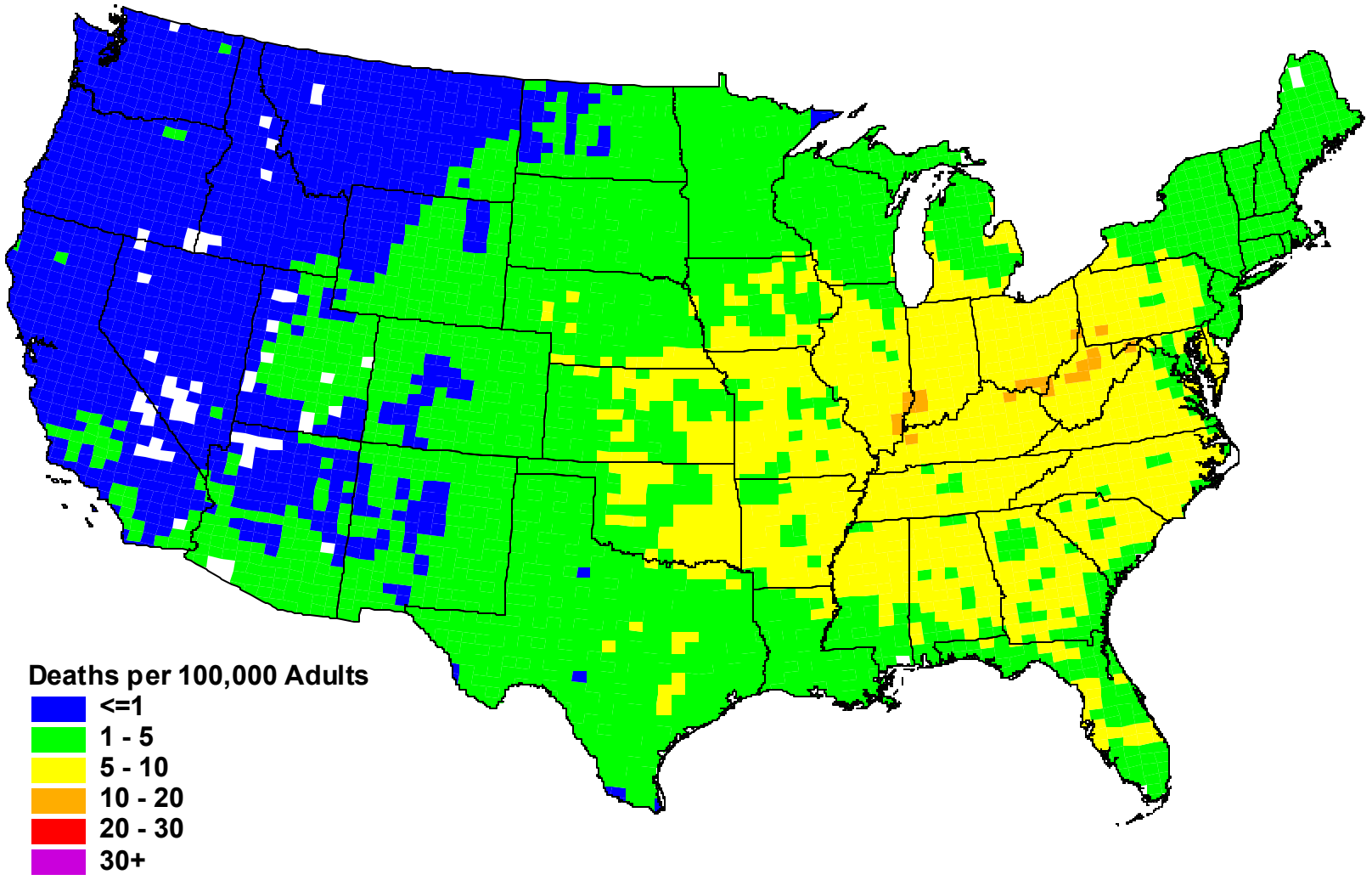

Exhibit 6-6 2020 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, Baseline

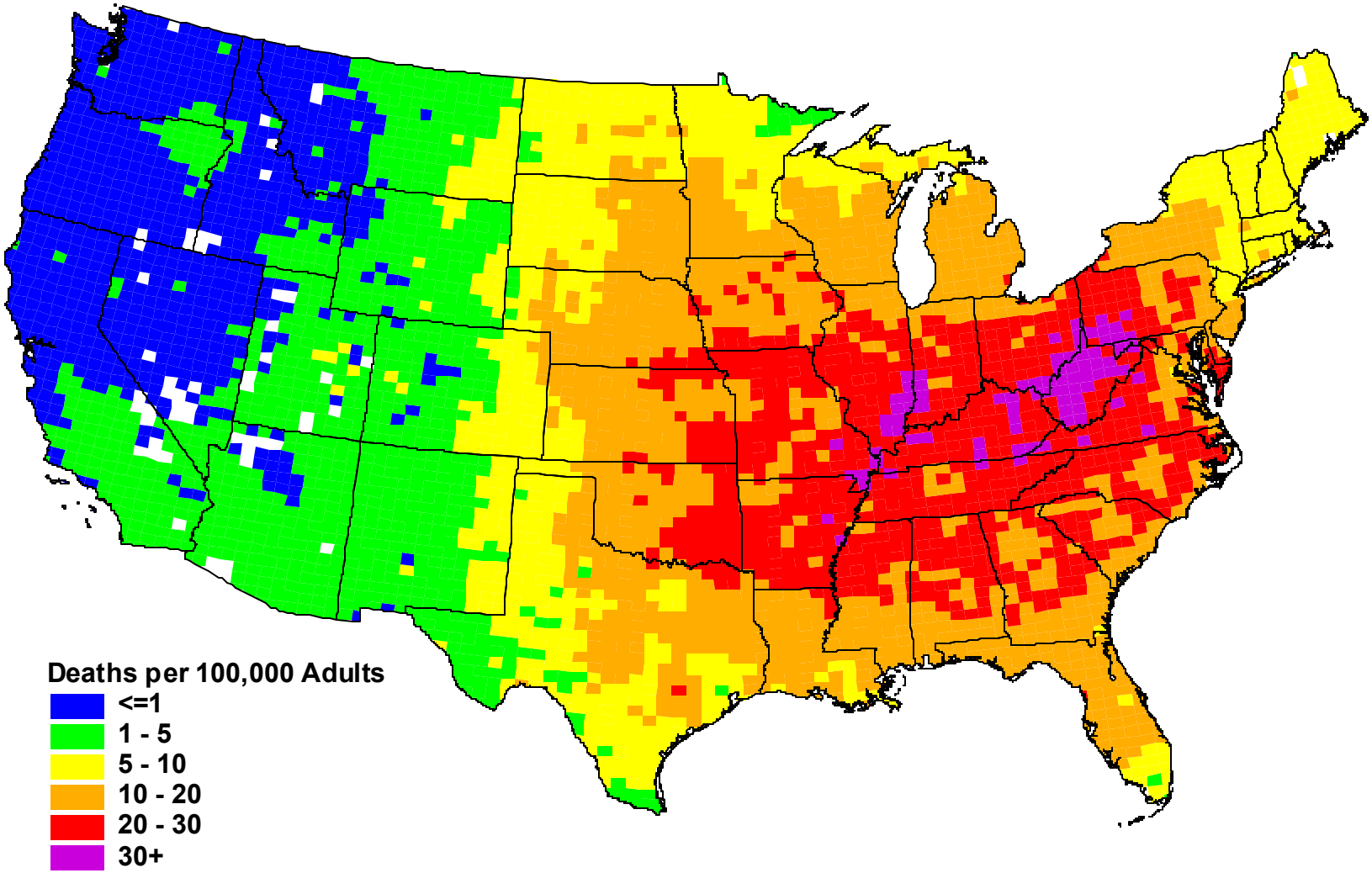

Exhibit 6-7 2020 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, With Clear Skies Act

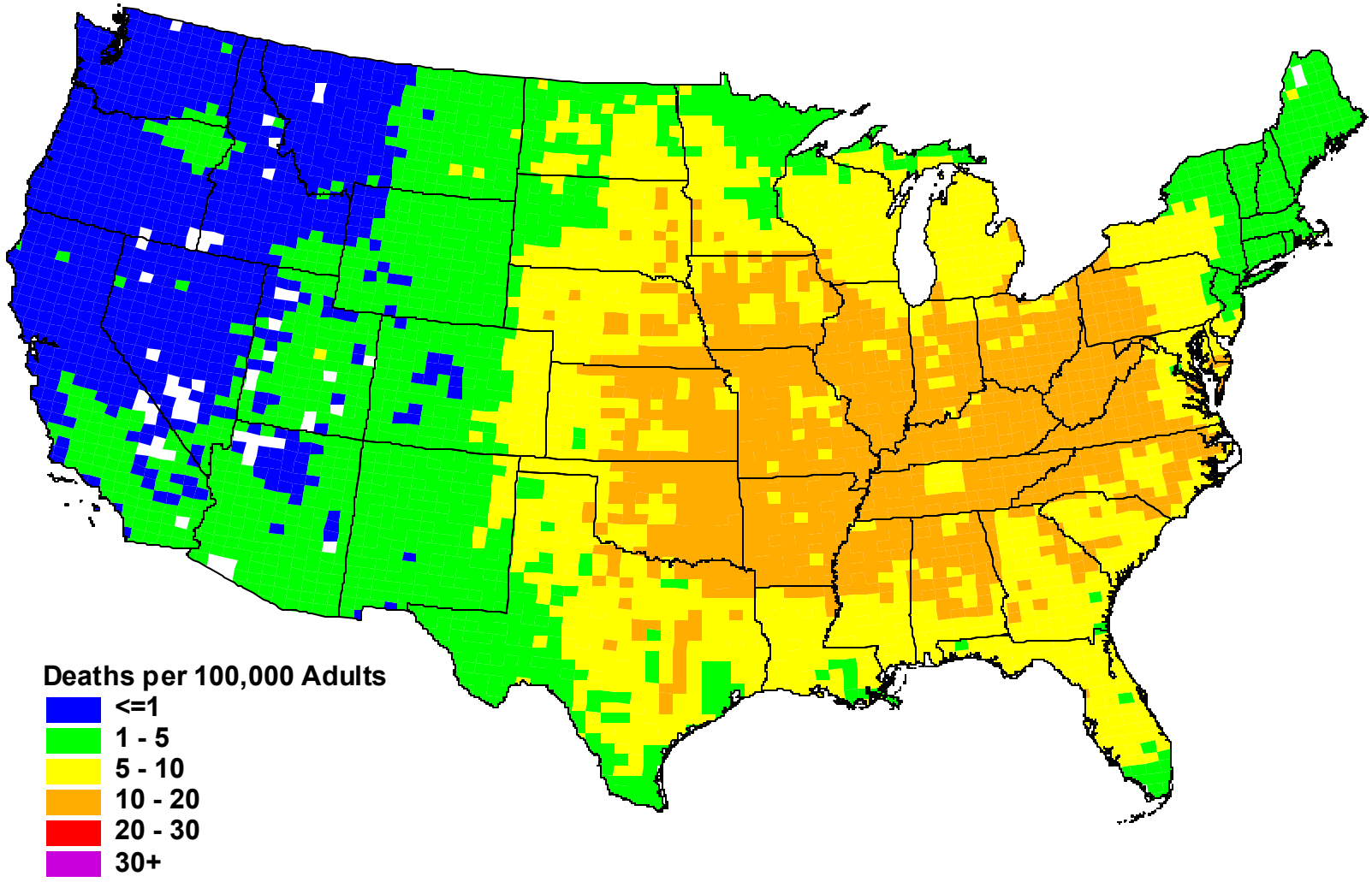

Exhibit 6-8 2020 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, With Carper

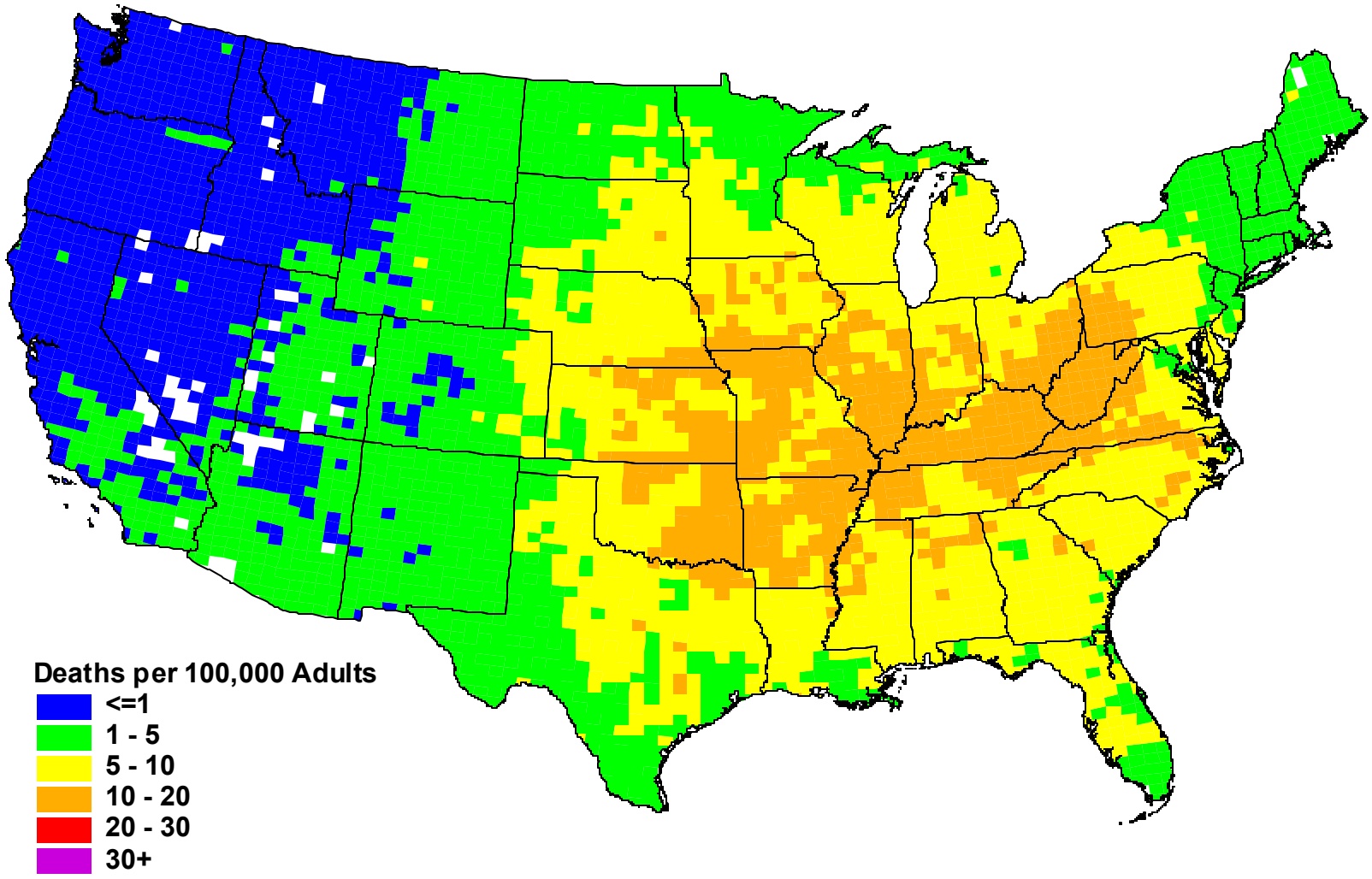

Exhibit 6-9 2020 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, With Straw Proposal

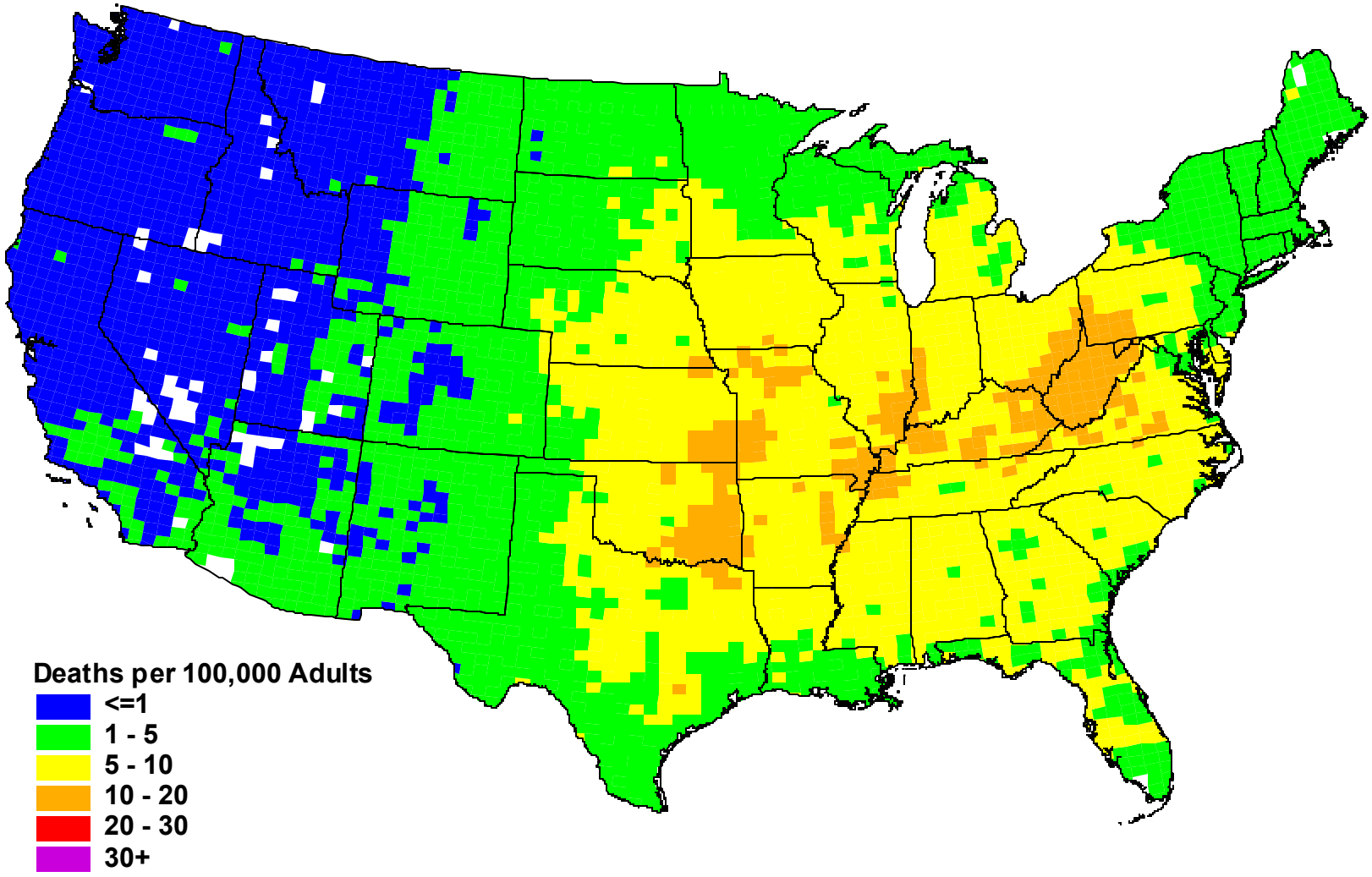

Exhibit 6-10 2020 Premature Mortality Risk Attributable to $PM_{2.5}$ from Power Plants, With Jeffords

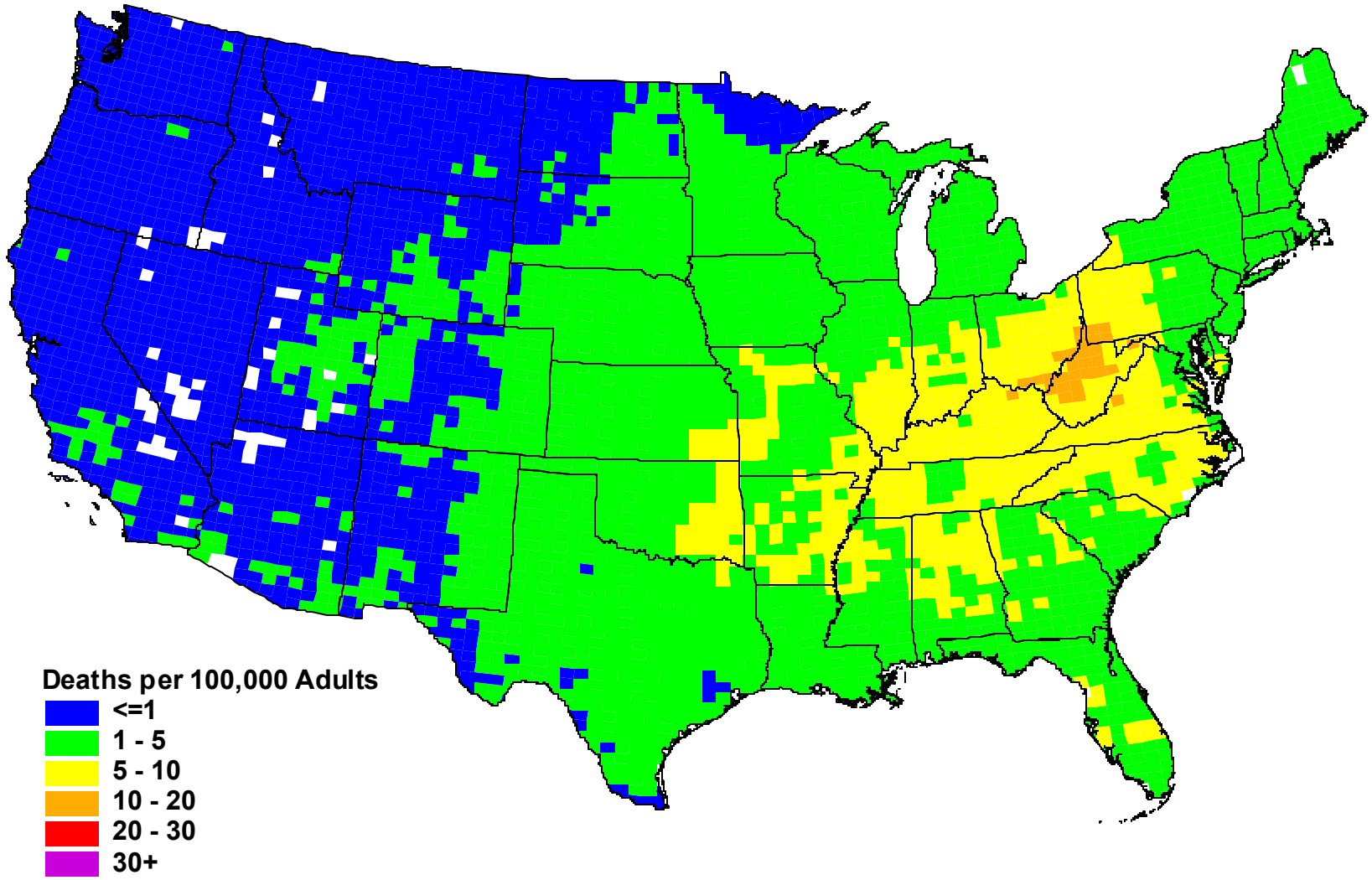

Table 6.5 Lung Cancer Mortality Estimates

	Lung Cancer Mortality (Pope et al., 2002)	Adult Mortality (Krewski et al., 2000)
2010		
CSA	944	7,861
No EGU	2,826	23,604
Carper	1,253	10,430
Straw	1,334	11,100
Jeffords	1,990	16,575
2020		
CSA	1,758	14,104
Carper	2,015	16,166
Straw	2,288	18,355
Jeffords	2,711	21,749

7 Non-Attainment Analysis

The reductions in ambient levels of $PM_{2.5}$ will not only reduce the numbers of adverse health effects attributable to PM, but will also have an influence on what portions of the country are predicted to exceed the National Ambient Air Quality Standards (NAAQS) for PM. In 2001 EPA issued draft guidance that describes a procedure for combining monitored data with REMSAD results to estimate future concentrations of PM2.5. The procedure, known as the Speciated Modeled Attainment Test (SMAT) uses estimates of current and future levels of six components of $PM_{2.5}$. The six components of $PM_{2.5}$ used in a SMAT analysis are: sulfates, nitrates, organic carbon, elemental carbon, crustal material, and un-attributed mass.

EPA used the SMAT technique to estimate the numbers of counties that will not attain the annual mean $PM_{2.5}$ NAAQS levels with and without the Clear Skies Act. They have also conducted SMAT analysis for other proposed rules currently under consideration. The most complete description of the SMAT method is available as part of the documentation of the January 30, 2004 proposed Clean Air Interstate Rule (CAIR). In particular, the SMAT procedures are described in "Appendix E: Speciated Modeled Attainment Test (SMAT) Documentation", a part of the *Technical Support Document for the Interstate Air Quality Rule Air Quality Modeling Analysis* available online at http://www.epa.gov/interstateairquality/tsd0162.pdf.

While the method used in the SMAT have not changed since EPA conducted the analysis of the Clear Skies Act, for the CAIR and other subsequent rules EPA has updated and refined some of the monitor data used in a SMAT.

This chapter provides the results of a SMAT analysis on each of the policy options considered in this report. While the method used in the SMAT have not changed since EPA conducted the analysis of the Clear Skies Act, for the CAIR and other subsequent rules EPA has updated and refined some of the historic monitor data and analysis used in a SMAT. The analysis in this chapter uses the same historic monitor data and analysis as the was used in EPA's analysis of the Clear Skies Act.

EPA's SMAT method is only applicable to counties with adequate $PM_{2.5}$. monitor data. The SMAT analysis of the Clear Skies Act used actual monitor data from 1999 through 2001, and analyzed a total of 307 counties. While these counties include many of the most heavily populated counties in the United States, a sizable portion of the population lives in the 2,802 counties that did not have sufficient $PM_{2.5}$ monitors in 1999-2001 to be included in those analyses.

The results of the SMAT analysis for the policy options examined in this report are included in Table 7-1. The analysis of the Clear Skies Act is from the EPA analysis. County results, including the Design Value (estimated $PM_{2.5}$ level at the highest monitor in the county) are presented in Appendix C.

Table 7-1 SMAT Results: Estimated Number of Non-Attainment Counties

All Years	# Counties Analyzed	307
Year	Policy Option	# of Counties Exceeding Annual Mean Standard
'99-'01	Observed Monitors	129
	Base Case	80
	Clear Skies	38
	Jeffords	16
	Straw	24
	Carper	27
	No EGU	13
2020	Base Case	53
	Clear Skies	18
	Jeffords	13
	Straw	13
	Carper	15

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Appendix A: Particulate Matter C-R Functions

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

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.

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

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

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

where:

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

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

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.12) associated with a change in mean exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) (Krewski et al., 2000, Part II - Table 31, 63 city Dichotomous sampler).

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

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

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

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

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

Chronic Illness

Schwartz (1993) and Abbey et al. (1993; 1995c) provide evidence that PM exposure over a number of years gives rise to the development of chronic bronchitis in the U.S., and a recent study by McDonnell et al. (1999) provides evidence that ozone exposure is linked to the development of asthma in adults. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998).[27]

Chronic Bronchitis (Abbey et al., 1995c, California)

Abbey et al. (1995c) examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for AOD or asthma; PM_{10} was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined. The C-R function is based on the results of the single pollutant model presented in Table 2.

Single Pollutant Model

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

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[27] 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.

Functional Form: Logistic
Coefficient: 0.0137
Standard Error: 0.00680
Incidence Rate: annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378
Population: population of ages 27 and older[28] without chronic bronchitis = 95.57%[29] of population 27+

Heart Attacks

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

Peters et al. (2001) studied the relationship between increased particulate air pollution and onset of heart attacks in the Boston area from 1995 to 1996. The authors used air quality data for PM10, PM10-2.5, PM2.5,"black carbon", O3, CO, NO2, and SO2 in a case-crossover analysis. For each subject, the case period was matched to three control periods, each 24 hours apart. In univariate analyses, the authors observed a positive association between heart attack occurrence and PM2.5 levels hours before and days before onset. The authors estimated multivariate conditional logistic models including two-hour and twenty-four hour pollutant concentrations for each pollutant. They found significant and independent associations between heart attack occurrence and both two-hour and twenty-four hour PM2.5 concentrations before onset. Significant associations were observed for PM10 as well. None of the other particle measures or gaseous pollutants were significantly associated with acute myocardial infarction for the two hour or twenty-four hour period before onset.

The patient population for this study was selected from health centers across the United States. The mean age of participants was 62 years old, with 21% of the study population under the age of 50. In order to capture the full magnitude of heart attack occurrence potentially associated with air pollution and because age was not listed as an inclusion criteria for sample selection, we apply an age range of 18 and over in the C-R function. According to the National Hospital Discharge Survey, there were no hospitalizations for heart attacks among children <15 years of age in 1999 and only 5.5% of all hospitalizations occurred in 15-44 year olds (Popovic, 2001, Table 10).

Single Pollutant Model

The coefficient and standard error are calculated from an odds ratio of 1.62 (95% CI 1.13-2.34) for a 20 $\mu g/m^3$ increase in twenty-four hour average $PM_{2.5}$ (Peters et al., 2001, Table 4, p. 2813).

Functional Form: Logistic
Coefficient: 0.024121

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

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

Standard Error: 0.009285

Incidence Rate: region-specific daily nonfatal heart attack rate per person 18+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410) [30]

Population: population of ages 18 and older

Hospital Admissions

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

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

Hospital Admissions for Chronic Lung Disease Less Asthma *(*Moolgavkar, 2000c)

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 2.0[31] and t-statistic of 2.2 for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ in the two-day lag model (Moolgavkar, 2000c, Table 4, p. 81).

Functional Form: Log-linear

Coefficient: 0.0020

Standard Error: 0.000909

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[30]This estimate assumes that all heart attacks that are not instantly fatal will result in a hospitalization. In addition, Rosamond et al. (1999) report that approximately six percent of male and eight percent of female hospitalized heart attack patients die within 28 days (either in or outside of the hospital). We applied a factor of 0.93 to the number of hospitalizations to estimate the number of nonfatal heart attacks per year.

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

Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 18-64 (ICD codes 490-492, 494-496)[32]

Population: population of ages 18 to 64

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

Sheppard et al. (1999) studied the relation between air pollution in Seattle and nonelderly (<65) hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM10_{10-2.5}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects.[33] They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, CO, and ozone. They did not observe an association for SO_2. They found PM and CO to be jointly associated with asthma admissions. The best fitting co-pollutant models were found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. For the remaining pollutants, the best fitting models included $PM_{2.5}$ and CO. Results for other co-pollutant models were not reported. The $PM_{2.5}$ C-R function is based on the multipollutant model.

Multipollutant Model ($PM_{2.5}$ and CO)

The coefficient and standard error for the co-pollutant model with CO are calculated from a relative risk of 1.03 (95% CI 1.01-1.06) for an 11.8 $\mu g/m^3$ increase[34] in $PM_{2.5}$ (Sheppard et al., 1999, p. 28).

Functional Form: Log-linear

Coefficient: 0.002505

Standard Error: 0.001045

Incidence Rate: region-specific daily hospital admission rate for asthma admissions per person <65 (ICD code 493)

Population: population of ages 65 and under

Hospital Admissions for Pneumonia (Lippmann et al., 2000, Detroit)

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[32] Moolgavkar (2000c) reports results for ICD codes 490-496. In order to avoid double counting non-elderly asthma hospitalizations (ICD code 493) with Sheppard et al. (1999) in a total benefits estimation, we have excluded ICD code 493 from the baseline incidence rate used in this function.

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

[34] The reported Inter Quartile Range(11.8 $\mu g/m^3$) change in the abstract and text is smaller than reported in Table 3. We assume the change reported in the abstract and text to be correct because greater number of significant figures are reported.

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

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.175 (95% CI 1.026-1.345) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 26).

Functional Form: Log-linear

Coefficient: 0.004480

Standard Error: 0.001918

Incidence Rate: region-specific daily hospital admission rate for pneumonia admissions per person 65+ (ICD codes 480-487)

Population: population of ages 65 and older

Hospital Admissions for Chronic Lung Disease

The following two studies, Lippmann (2000) and Moolgavkar (2000b), were combined together using a random/fixed effects pooling method. The random/fixed effects weighting for each study was as follows: Lippmann(2000) study was 15% and Moolgavkar(2000b) study was 85%. The pertinent information for the individual studies has been included below.

1) Lippmann et al., 2000, Detroit

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

co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on results of the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.040 (95% CI 0.877-1.234) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 26).

Functional Form: Log-linear

Coefficient: 0.001089

Standard Error: 0.002420

Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 65+ (ICD codes 490-496)

Population: population of ages 65 and older

2) Moolgavkar, 2000b

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

The $PM_{2.5}$ C-R functions are based on the co-pollutant models ($PM_{2.5}$ and CO) reported for the 20-64 and 65+ age groups. Since the true PM effect is most likely best represented by a distributed lag model, then any single lag model should underestimate the total PM effect. As a result, we selected the lag models with the greatest effect estimates for use in the C-R functions.

Ages 65 and older

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.8[35] and t-statistic of 0.8 for a 10 μg/m^3 increase in $PM_{2.5}$ in the two-day lag model (Moolgavkar, 2000b, Table 3, p. 80).

Functional Form: Log-linear

Coefficient: 0.0008

Standard Error: 0.001000

Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 65+ (ICD codes 490-496)

Population: population of ages 65 and older

Hospital Admissions, All Cardiovascular(20-64) (Moolgavkar, 2000a, Los Angeles)

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

Ages 18 to 64[36]

Multipollutant Model ($PM_{2.5}$ and CO)

[35] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.8 would result in a relative risk of 1.008 and coefficient of 0.000797. The "estimated" percent change, as reported by Moolgavkar, of 0.8 results in a relative risk of 1.008032 and coefficient of 0.0008.

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

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.9[37] and t-statistic of 1.8 for a 10 μg/m^3 increase in $PM_{2.5}$ in the zero lag model (Moolgavkar, 2000a, Table 4, p. 1203).

Functional Form: Log-linear

Coefficient: 0.0009

Standard Error: 0.000500

Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person ages 18 to 64 (ICD codes 390-409, 411-459)[38]

Population: population of ages 18 to 64

Hospital Admissions for All Cardiovascular(65+)

The following four studies, Moolgavkar (2000a), and Lippmann (2000) Dysrhythmia, Lippmann (2000) Heart Failure, and Lippmann (2000) Ischemic Heart Disease were combined together using a random/fixed effects pooling method. The random/fixed effects weighting for each study was as follows: Moolgavkar(2000a) study was 76% and the sum of the three Lippmann studies was weighted 24%. The pertinent information for the individual studies has been included below.

1) Moolgavkar, 2000a, Los Angeles

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

Ages 65 and older

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[37] In a log-linear model, the percent change is equal to (RR - 1) * 100. In a similar hospitalization study by Moolgavkar (2000b), he defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.9 would result in a relative risk of 1.009 and coefficient of 0.000896. Assuming that the 0.9 is the "estimated" percent change described previously would result in a relative risk of 1.009041 and coefficient of 0.0009. We assume that the "estimated" percent changes reported in this study reflect the definition from (Moolgavkar, 2000b).

[38] Moolgavkar (2000a) reports results that include ICD code 410 (heart attack). In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.5[39] and t-statistic of 0.9 for a 10 μg/m^3 increase in $PM_{2.5}$ in the one day lag model (Moolgavkar, 2000a, Table 3, p. 1202).

Functional Form: Log-linear

Coefficient: 0.0005

Standard Error: 0.000556

Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person 65+ (ICD codes 390-409, 411-459)[40]

Population: population of ages 65 and older

2) Lippmann et al., 2000, Detroit

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

a) Hospital Admissions for Dysrhythmia

Multipollutant Model ($PM_{2.5}$ and ozone)

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[39] In a log-linear model, the percent change is equal to (RR - 1) * 100. In a similar hospitalization study by Moolgavkar (2000b), he defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.5 would result in a relative risk of 1.005 and coefficient of 0.000499. Assuming that the 0.5 is the "estimated" percent change described previously would result in a relative risk of 1.005013 and coefficient of 0.0005. We assume that the "estimated" percent changes reported in this study reflect the definition from (Moolgavkar, 2000b).

[40] Moolgavkar (2000a) reports results for ICD codes 390-429. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.080 (95% CI 0.904-1.291) for a 36 μg/m^3 increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 27).

Functional Form: Log-linear

Coefficient: 0.002138

Standard Error: 0.002525

Incidence Rate: region-specific daily hospital admission rate for dysrhythmia admissions per person 65+ (ICD code 427)

Population: population of ages 65 and older

b) Hospital Admissions for Heart Failure

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.183 (95% CI 1.053-1.329) for a 36 μg/m^3 increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 27).

Functional Form: Log-linear

Coefficient: 0.004668

Standard Error: 0.001650

Incidence Rate: region-specific daily hospital admission rate for heart failure admissions per person 65+ (ICD code 428)

Population: population of ages 65 and older

c) Hospital Admissions for Ischemic Heart Disease

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.041 (95% CI 0.947-1.144) for a 36 μg/m^3 increase in $PM_{2.5}$ (Lippmann et al., 2000, Table 14, p. 27).

Functional Form: Log-linear

Coefficient: 0.001116

Standard Error: 0.001339

Incidence Rate: region-specific daily hospital admission rate for ischemic heart disease admissions per person 65+ (ICD codes 411-414)[41]

Population: population of ages 65 and older

Emergency Room Visits

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

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

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

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

Multipollutant Model ($PM_{2.5}$, NO_2, and SO_2)

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

Functional Form: Log-linear

Coefficient: 0.016527

Standard Error: 0.004139

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[41] Lippmann et al. (2000) reports results for ICD codes 410-414. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

Incidence Rate: region-specific daily emergency room rate for asthma admissions per person <18 (ICD code 493)

Population: population of ages under 18

Acute Morbidity

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

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

Acute Bronchitis (Dockery et al., 1996)

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.[42] 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.

[42] 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:

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

where:

y_0 = annual bronchitis incidence rate per person = 0.044

β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0272

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

pop = population of ages 8-12

σ_β = standard error of β = 0.0171

Incidence Rate. Bronchitis was counted in the study only if there were "reports of symptoms in the past 12 months" (Dockery et al., 1996, p. 501). It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. Dockery et al. found no relationship between PM 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).[43] 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.[44]

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

[44]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.

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.

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

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

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

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

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

Lower Respiratory Symptoms (Schwartz et al., 1994)

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

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

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

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

σ_β = standard error of β = 0.00586

Incidence Rate. The proposed incidence rate, 0.12 percent, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). They did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated values are 10th = 0 percent, 25th = 0 percent, 50th = 0 percent, 75th = 0.29 percent, and 90th = 0.34 percent. The most conservative estimate consistent with the data are to assume the incidence is zero up to the 75th percentile, a constant 0.29 percent between the 75th and 90th percentiles, and a constant 0.34 percent between the 90th and 100th percentiles. Alternatively, assuming a linear slope between the 50th and 75th, 75th and 90th, and 90th to 100th percentiles, the estimated mean incidence rate is 0.12 percent,[45] 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):

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

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

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

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

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

Population. Schwartz et al. (1994, Table 5 and p. 1235) enrolled 1,844 children into a year-long study conducted in different years in different cities; the students were in grades two through five and lived in six U.S. cities. All study participants were enrolled in September 1984; the actual study was conducted in

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

Watertown, MA in 1984/85; Kingston-Harriman, TN, and St. Louis, MO in 1985/86; Steubenville, OH, and Portage, WI in 1986/87; and Topeka, KS in 1987/88. The study does not publish the age range of the children when they participated. As a result, the study is somewhat unclear about the appropriate age range for the resulting C-R function. If all the children were in second grade in 1984 (ages 7-8) then the Topeka cohort would be in fifth grade (ages 10-11) when they participated in the study. It appears from the published description, however, that the students were in grades two through five in 1984.[46] By the completion of the study, some students in the Topeka cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

Upper Respiratory Symptoms (Pope et al., 1991)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

Single Pollutant Model

The coefficient and standard error for a one $\mu g/m^3$ change in PM_{10} is reported in Table 5.

Functional Form: Logistic

Coefficient: 0.0036

Standard Error: 0.0015

Incidence Rate: daily upper respiratory symptom incidence rate per person = 0.3419 (Pope et al., 1991, Table 2)

Population: asthmatic population[47] ages 9 to 11 = 5.67% of population ages 9 to 11

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

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

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, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b). On the other hand, the number of workers over the age of 65 is relatively small; it was under 3% of the total workforce in 1996 (U.S. Bureau of the Census, 1997, Table 633).

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

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

where:

y_0 = daily work-loss-day incidence rate per person = 0.00648

β = inverse-variance weighted $PM_{2.5}$ coefficient = 0.0046

$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[48]

pop = population of ages 18 to 65

σ_β = standard error of β = 0.00036

Incidence Rate. The estimated 1994 annual incidence rate is the annual number (376,844,000) of WLD per person in the age 18-64 population divided by the number of people in 18-64 population (159,361,000). The

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

1994 daily incidence rate is calculated as the annual rate divided by 365.[49] Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight:

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

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

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

This eventually reduces down to:

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

Minor Restricted Activity Days (Ostro and Rothschild, 1989)

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

[49]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).

groups (e.g., Schwartz, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b).

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient used in this analysis is a weighted average of the coefficients (Ostro, 1987, Table IV) using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) is:

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

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[50]

pop = adult population ages 18 to 65

σ_β = standard error of β = 0.0007

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

Coefficient Estimate (β). The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight:

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

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

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

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

This reduces down to:

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

Supplemental Concentration Response Functions

Mortality (Krewski et al., 2000) - Reanalysis of Dockery et al. (1993)

Krewski et al. (2000) performed a validation and replication analysis of Dockery et al. (1993). The original investigators examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved exposure estimates, a slightly broader study population (adults aged 25 and older; a higher proportion without a high school education), and a follow-up period nearly twice as long as that of Pope et al. (1995). Krewski et al. (2000, Part II - Table 52) found that educational status was a strong effect modifier of the PM - mortality relationship in both studies, with the strongest effect seen among the less educated. Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

After an audit of the air pollution data, demographic variables, and cohort selection process, Krewski et al. (2000) noted that a small portion of study participants were mistakenly censored early. The following C-R function is based on the risk estimate from the audited data, with the inclusion of those person-years mistakenly censored early.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.28) and 95% confidence interval (1.10-1.48) associated with a change in *annual mean* $PM_{2.5}$ exposure of 18.6 $\mu g/m^3$ to 29.6 $\mu g/m^3$ (Krewski et al., 2000, Part I - Table 19c).

Functional Form: Log-linear

Coefficient: 0.013272

Standard Error: 0.004070

Incidence Rate: county-specific annual all cause mortality rate per person ages 25 and older

Population: population of ages 25 and older

Mortality, Lung Cancer (Pope et al., 2002) - Based on ACS Cohort: Mean $PM_{2.5}$

Pope et al. (2002) followed Krewski et al. (2000) and Pope et al. (1995, Table 2) and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths.[51] Like the earlier studies, Pope et al. (2002) found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. (2002) found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with "all other" deaths.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.082) and 95% confidence interval (1.011-1.158) associated with a change in *annual mean* exposure of 10.0 $\mu g/m^3$. Pope et al. (2002, Table 2).[52]

Functional Form: Log-linear

Coefficient: 0.007881

Standard Error: 0.003463

Incidence Rate: county-specific annual lung cancer mortality rate (ICD code 162) per person ages 30 and older

Population: population of ages 30 and older

[51]All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

[52]Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

Appendix B: Benefits Estimate: Uncertainty Results

Uncertainty estimates (5^{th} and 95^{th} percentile estimates) for the health and valuation results are shown in Tables B-1 through B-4.

Table B-1 2010 Health Benefits with Uncertainty: Numbers of Avoided Cases

	CSA	No EGU			Carper		
	Mean	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Mortality	7,861	13,527	23,604	33,632	5,969	10,430	14,880
Chronic Bronchitis	5,400	3,025	16,221	29,069	1,326	7,160	12,921
Heart Attacks	13,115	14,377	38,198	60,841	6,401	17,218	27,753
Hospital Admissions-Respiratory							
Chronic Lung, less Asthma(20-64)	374	284	1,127	1,975	125	496	868
Asthma (0-64)	651	610	1,946	3,291	270	860	1,453
Pneumonia (65+)	2,653	2,364	8,040	13,780	1,036	3,515	6,008
Chronic Lung (65+)	332	(1,464)	1,000	3,641	(647)	441	1,602
Total Hospital Admissions-Respiratory	4,010		12,113			5,313	
Hospital Admissions Cardiovascular							
All Cardiovascular,(20-64)	1,332	347	4,028	7,719	153	1,778	3,406
All Cardiovascular,(65+)	1,903	(2,547)	5,707	18,651	(1,126)	2,521	8,207
Total Hospital Admissions-Cardiovascular	3,235		9,735			4,299	
Emergency Room Visits for Asthma	8,316	15,103	25,999	37,163	6,493	11,108	15,779
Acute Bronchitis	12,522	(1,297)	37,705	74,385	(562)	16,614	33,303
Lower Respiratory Symptoms	142,621	206,086	429,980	646,906	89,990	189,214	286,836
Upper Respiratory Symptoms	113,707	109,901	348,823	587,102	47,666	151,390	254,973
Work Loss Days	1,050,415	2,778,689	3,186,036	3,592,565	1,216,135	1,395,098	1,573,877
Minor Restricted Activity Days	6,258,491	16,008,538	18,916,818	21,813,846	7,022,655	8,306,310	9,587,433

Table B-1 2010 Health Benefits with Uncertainty: Numbers of Avoided Cases (continued)

	CSA	Straw			Jeffords		
	Mean	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Mortality	7,861	6,353	11,100	15,836	9,492	16,575	23,634
Chronic Bronchitis	5,400	1,411	7,615	13,737	2,118	11,397	20,500
Heart Attacks	13,115	6,786	18,244	29,394	10,110	27,039	43,342
Hospital Admissions - Respiratory							
Chronic Lung, less Asthma (20-64)	374	133	527	922	199	791	1,386
Asthma (0-64)	651	286	912	1,540	427	1,362	2,301
Pneumonia (65+)	2,653	1,100	3,733	6,381	1,657	5,628	9,632
Chronic Lung (65+)	332	(687)	468	1,702	(1,029)	702	2,554
Total Hospital Admissions - Respiratory	4,010		5,640			8,484	
Hospital Admissions Cardiovascular							
All Cardiovascular, (20-64)	1,332	163	1,893	3,625	244	2,829	5,420
All Cardiovascular, (65+)	1,903	(1,195)	2,677	8,712	(1,787)	4,006	13,064
Total Hospital Admissions - Cardiovascular	3,235		4,570			6,835	
Emergency Room Visits for Asthma	8,316	6,901	11,811	16,782	10,610	18,205	25,936
Acute Bronchitis	12,522	(598)	17,669	35,393	(905)	26,554	52,826
Lower Respiratory Symptoms	142,621	95,720	201,197	304,907	144,480	302,678	457,198
Upper Respiratory Symptoms	113,707	50,715	161,069	271,268	76,773	243,760	410,415
Work Loss Days	1,050,415	1,293,454	1,483,765	1,673,875	1,945,445	2,231,223	2,516,574
Minor Restricted Activity Days	6,258,491	7,468,187	8,832,956	10,194,911	11,220,404	13,265,510	15,304,720

Table B-2 Value of 2010 Health Benefits with Uncertainty (Millions of $1999)

	CSA	No EGU			Carper		
	Mean	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Mortality	$51,974	$21,838	$149,274	$359,554	$9,645	$65,959	$158,961
Chronic Bronchitis	$2,046	$466	$5,523	$18,709	$204	$2,438	$8,262
Heart Attacks	$1,127	$950	$3,284	$7,573	$425	$1,480	$3,447
Hospital Admissions - Respiratory							
Chronic Lung, less Asthma (20-64)	$4	$3	$13	$24	$2	$6	$11
Asthma (0-64)	$5	$5	$15	$25	$2	$7	$10
Pneumonia (65+)	$47	$41	$143	$237	$18	$63	$104
Chronic Lung (65+)	$4	-$18	$13	$47	-$8	$6	$21
Total Hospital Admissions - Respiratory	$60		$187			$82	
Hospital Admissions Cardiovascular							
All Cardiovascular, (20-64)	$30	$8	$92	$169	$3	$41	$75
All Cardiovascular, (65+)	$39	-$64	$116	$335	-$28	$51	$147
Total Hospital Admissions - Cardiovascular	$69		$206			$92	
Emergency Room Visits for Asthma	$2	$4	$7	$11	$2	$3	$5
Acute Bronchitis	$5	$0	$13	$33	$0	$6	$14
Lower Respiratory Symptoms	$2	$3	$7	$12	$1	$3	$6
Upper Respiratory Symptoms	$3	$3	$9	$19	$1	$4	$7
Work Loss Days	$136	$338	$367	$437	$148	$161	$191
Minor Restricted Activity Days	$327	$549	$956	$1,340	$241	$420	$589

Table B-2 Value of 2010 Health Benefits with Uncertainty (Millions of $1999) (Continued)

	CSA	Straw			Jeffords		
	Mean	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Mortality	$51,974	$10,265	$70,198	$169,171	$15,331	$104,823	$252,558
Chronic Bronchitis	$2,046	$217	$2,592	$8,786	$326	$3,881	$13,148
Heart Attacks	$1,127	$450	$1,568	$3,651	$670	$2,324	$5,389
Hospital Admissions - Respiratory							
Chronic Lung, less Asthma (20-64)	$4	$2	$6	$11	$2	$9	$17
Asthma (0-64)	$5	$2	$7	$11	$3	$11	$17
Pneumonia (65+)	$47	$19	$67	$110	$29	$100	$166
Chronic Lung (65+)	$4	-$8	$6	$22	-$13	$9	$33
Total Hospital Admissions - Respiratory	$60		$87			$132	
Hospital Admissions Cardiovascular							
All Cardiovascular, (20-64)	$30	$4	$43	$80	$6	$64	$119
All Cardiovascular, (65+)	$39	-$30	$54	$156	-$45	$81	$234
Total Hospital Admissions - Cardiovascular	$69		$97			$146	
Emergency Room Visits for Asthma	$2	$2	$3	$5	$3	$5	$8
Acute Bronchitis	$5	$0	$7	$15	$0	$10	$23
Lower Respiratory Symptoms	$2	$1	$3	$6	$2	$5	$8
Upper Respiratory Symptoms	$3	$1	$4	$8	$2	$6	$13
Work Loss Days	$136	$158	$171	$204	$237	$257	$306
Minor Restricted Activity Days	$327	$257	$447	$626	$385	$670	$940

Table B-3 2020 Health Benefits with Uncertainty: Numbers of Avoided Cases

	CSA	Carper			Straw		
	Mean	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Mortality	14,104	9,255	16,166	23,057	10,510	18,355	26,174
Chronic Bronchitis	8,770	1,864	10,048	18,105	2,121	11,422	20,560
Heart Attacks	23,009	9,795	26,280	42,253	11,126	29,798	47,827
Hospital Admissions - Respiratory							
Chronic Lung, less Asthma (20-64)	610	176	699	1,223	200	794	1,390
Asthma (0-64)	1,151	359	1,145	1,934	408	1,302	2,199
Pneumonia (65+)	4,972	1,681	5,705	9,756	1,913	6,496	11,114
Chronic Lung (65+)	650	(1,093)	746	2,711	(1,242)	848	3,083
Total Hospital Admissions - Respiratory	7,383		8,295			7,513	
Hospital Admissions Cardiovascular							
All Cardiovascular, (20-64)	2,139	211	2,452	4,697	240	2,782	5,329
All Cardiovascular, (65+)	3,632	(1,868)	4,165	13,712	(2,141)	4,731	15,381
Total Hospital Admissions - Cardiovascular	5,771		6,617			7,513	
Emergency Room Visits for Asthma	13,223	8,867	15,191	21,608	10,132	17,373	24,734
Acute Bronchitis	19,919	(775)	22,823	45,580	(884)	25,971	51,755
Lower Respiratory Symptoms	226,616	123,702	259,649	392,945	140,932	295,492	446,707
Upper Respiratory Symptoms	181,286	65,533	208,106	350,443	74,731	237,294	399,557
Work Loss Days	1,602,343	1,601,815	1,837,341	2,072,576	1,823,365	2,091,325	2,358,915
Minor Restricted Activity Days	9,519,433	9,226,638	10,910,946	12,591,232	10,498,482	12,413,325	14,323,062

Table B-3 2010 Health Benefits with Uncertainty: Numbers of Avoided Cases (Continued)

	CSA	Jeffords		
	Mean	5th Percentile	Mean	95th Percentile
Mortality	14,104	12,456	21,749	31,006
Chronic Bronchitis	8,770	2,526	13,586	24,421
Heart Attacks	23,009	13,185	35,230	56,418
Hospital Admissions - Respiratory				
Chronic Lung, less Asthma (20-64)	610	238	945	1,655
Asthma (0-64)	1,151	484	1,545	2,610
Pneumonia (65+)	4,972	2,281	7,749	13,264
Chronic Lung (65+)	650	(1,477)	1,008	3,667
Total Hospital Admissions - Respiratory	7,383		11,247	
Hospital Admissions Cardiovascular				
All Cardiovascular, (20-64)	2,139	284	3,296	6,314
All Cardiovascular, (65+)	3,632	(2,537)	5,615	18,273
Total Hospital Admissions - Cardiovascular	5,771		8,911	
Emergency Room Visits for Asthma	13,223	12,263	21,050	30,005
Acute Bronchitis	19,919	(1,059)	31,013	61,598
Lower Respiratory Symptoms	226,616	168,653	353,091	533,005
Upper Respiratory Symptoms	181,286	89,544	284,295	478,639
Work Loss Days	1,602,343	2,176,116	2,495,685	2,814,756
Minor Restricted Activity Days	9,519,433	12,519,823	14,800,704	17,074,718

Table B-4 Value of 2020 Health Benefits with Uncertainty (Millions of $1999)

	CSA	Carper			Straw		
	Mean	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Mortality	$106,996	$17,154	$117,302	$282,662	$19,478	$133,186	$320,915
Chronic Bronchitis	$3,880	$335	$3,995	$13,536	$381	$4,540	$15,386
Heart Attacks	$1,961	$638	$2,240	$5,221	$724	$2,540	$5,912
Hospital Admissions - Respiratory							
Chronic Lung, less Asthma (20-64)	$7	$2	$8	$15	$2	$9	$17
Asthma (0-64)	$9	$3	$9	$14	$3	$10	$16
Pneumonia (65+)	$89	$29	$102	$168	$33	$116	$191
Chronic Lung (65+)	$9	-$14	$10	$36	-$15	$11	$40
Total Hospital Admissions - Respiratory	$114		$131			$149	
Hospital Admissions Cardiovascular							
All Cardiovascular, (20-64)	$49	$5	$56	$103	$5	$63	$117
All Cardiovascular, (65+)	$74	-$47	$84	$244	-$54	$96	$277
Total Hospital Admissions - Cardiovascular	$123		$140			$159	
Emergency Room Visits for Asthma	$4	$2	$4	$7	$3	$5	$7
Acute Bronchitis	$8	$0	$9	$20	$0	$10	$23
Lower Respiratory Symptoms	$4	$2	$4	$7	$2	$5	$8
Upper Respiratory Symptoms	$5	$2	$6	$11	$2	$6	$13
Work Loss Days	$208	$195	$212	$252	$222	$241	$287
Minor Restricted Activity Days	$522	$333	$578	$811	$378	$658	$923

Table B-4 Value of 2020 Health Benefits with Uncertainty (Millions of $1999) (Continued)

	CSA 2020	Jeffords		
	Mean	5th Percentile	Mean	95th Percentile
Mortality	$106,996	$23,083	$157,813	$380,215
Chronic Bronchitis	$3,880	$455	$5,401	$18,299
Heart Attacks	$1,961	$859	$3,003	$6,977
Hospital Admissions - Respiratory				
Chronic Lung, less Asthma (20-64)	$7	$3	$11	$20
Asthma (0-64)	$9	$4	$12	$19
Pneumonia (65+)	$89	$40	$138	$229
Chronic Lung (65+)	$9	-$18	$14	$47
Total Hospital Admissions - Respiratory	$114		$177	
Hospital Admissions Cardiovascular				
All Cardiovascular, (20-64)	$49	$6	$75	$139
All Cardiovascular, (65+)	$74	-$63	$114	$330
Total Hospital Admissions - Cardiovascular	$123		$188	
Emergency Room Visits for Asthma	$4	$3	$6	$9
Acute Bronchitis	$8	$0	$12	$28
Lower Respiratory Symptoms	$4	$2	$6	$10
Upper Respiratory Symptoms	$5	$2	$8	$16
Work Loss Days	$208	$265	$288	$342
Minor Restricted Activity Days	$522	$451	$784	$1,100

Appendix C: Details of SMAT Non-Attainment Analysis

Table C-1 reports the estimated design values for the 307 counties included in the EPA Clear Skies Act SMAT analysis. Table C-1 includes the EPA estimates of the observed monitors (1999-2001) and of the Clear Skies Act, as well as the estimates for all the policy options included in this report.

Table C-1 County Estimates of PM2.5 Design Values

State	County	'99-'01 *EPA CSA Analysis* PM2.5 1999-2001 Ambient Design Value	2010 PM2.5 Base case 2010	2010 PM2.5 Clear Skies 2010	2010 Jeffords	2010 Straw	2010 Carper	2010 No EGU	2020 *EPA CSA Analysis* PM2.5 Base case 2020	2020 PM2.5 Clear Skies 2020	2020 Jeffords	2020 Straw	2020 Carper
Total National Number of Non-Attaining Counties		129	80	38	16	24	27	13	53	18	13	13	15
Alabama	Clay	15.55	14.27	13.31	11.71	12.50	12.70	10.76	13.59	11.87	11.07	11.31	10.47
Alabama	Colbert	15.25	13.46	12.21	10.74	11.47	11.65	9.87	12.74	10.87	10.14	10.36	9.59
Alabama	DeKalb	16.76	15.24	14.09	11.95	13.02	13.22	10.75	14.40	12.34	11.25	11.25	11.62
Alabama	Houston	16.33	15.19	14.18	12.47	13.32	13.53	11.46	14.84	13.09	12.20	12.47	11.54
Alabama	Jefferson	21.58	20.07	18.96	17.15	18.13	18.32	15.96	19.22	17.38	16.35	16.35	16.60
Alabama	Madison	15.50	13.97	12.79	10.95	11.98	12.15	9.85	13.19	11.33	10.30	10.30	10.66
Alabama	Mobile	15.34	14.45	13.62	12.45	13.11	13.26	11.61	14.40	12.93	12.39	12.39	12.63
Alabama	Montgomery	16.79	15.71	14.74	13.06	13.89	14.15	12.06	15.31	13.63	12.74	12.74	12.95
Alabama	Morgan	19.30	17.74	16.24	14.28	15.26	15.50	13.12	16.82	14.46	13.48	13.78	12.75
Alabama	Russell	18.39	17.11	15.93	14.06	14.96	15.23	12.87	16.48	14.36	13.43	13.43	13.67
Alabama	Shelby	16.58	15.32	14.44	13.03	13.82	14.00	12.10	14.70	13.20	12.43	12.43	12.68
Alabama	Talladega	17.76	16.42	15.53	13.54	14.41	14.79	12.42	15.85	14.16	13.14	13.14	13.37
Arizona	Coconino	7.50	7.20	7.16	7.13	7.14	7.15	7.11	7.12	7.07	7.01	7.03	7.05
Arizona	Gila	9.60	9.11	9.05	9.02	9.03	9.04	9.00	9.01	8.93	8.87	8.89	8.91
Arizona	Maricopa	11.20	10.78	10.72	10.69	10.70	10.71	10.67	10.79	10.71	10.65	10.67	10.69
Arizona	Pinal	8.60	8.30	8.23	8.20	8.21	8.22	8.18	8.30	8.22	8.16	8.18	8.20
Arizona	Santa Cruz	12.10	11.70	11.58	11.55	11.56	11.57	11.53	11.71	11.55	11.49	11.51	11.53
California	Alameda	12.20	11.22	11.21	11.18	11.19	11.20	11.16	10.53	10.52	10.46	10.48	10.50
California	Butte	15.40	14.03	14.01	13.98	13.99	14.00	13.96	13.33	13.31	13.25	13.27	13.29
California	Calaveras	9.40	8.39	8.38	8.35	8.36	8.37	8.33	7.80	7.79	7.73	7.75	7.77
California	Colusa	10.30	9.55	9.54	9.51	9.52	9.53	9.49	9.18	9.17	9.11	9.13	9.15
California	El Dorado	8.10	7.34	7.33	7.30	7.31	7.32	7.28	6.93	6.91	6.85	6.87	6.89
California	Fresno	24.00	21.76	21.73	21.70	21.71	21.72	21.68	20.85	20.82	20.76	20.78	20.80
California	Humboldt	9.20	8.58	8.58	8.55	8.56	8.57	8.53	8.56	8.55	8.49	8.51	8.53
California	Imperial	15.70	13.83	13.75	13.72	13.73	13.74	13.70	13.38	13.28	13.22	13.24	13.26
California	Kern	23.70	20.68	20.64	20.61	20.62	20.63	20.59	19.62	19.58	19.52	19.54	19.56
California	Kings	16.60	14.29	14.26	14.23	14.24	14.25	14.21	13.16	13.13	13.07	13.09	13.11
California	Los Angeles	25.90	23.73	23.69	23.66	23.67	23.68	23.64	23.84	23.80	23.74	23.76	23.78
California	Mendocino	8.00	7.21	7.20	7.17	7.18	7.19	7.15	6.85	6.84	6.78	6.80	6.82
California	Merced	18.90	16.51	16.48	16.45	16.46	16.47	16.43	15.20	15.17	15.11	15.13	15.15
California	Modoc	8.00	7.42	7.41	7.38	7.39	7.40	7.36	7.13	7.11	7.05	7.07	7.09

State	County	'99-'01 *EPA CSA Analysis* PM2.5 1999-2001 Ambient Design Value	2010 PM2.5 Base case 2010	2010 PM2.5 Clear Skies 2010	2010 Jeffords	2010 Straw	2010 Carper	2010 No EGU	2020 *EPA CSA Analysis* PM2.5 Base case 2020	2020 PM2.5 Clear Skies 2020	2020 Jeffords	2020 Straw	2020 Carper
California	Orange	22.40	20.76	20.71	20.68	20.69	20.70	20.66	21.16	21.10	21.04	21.06	21.08
California	Placer	12.50	11.29	11.28	11.25	11.26	11.27	11.23	10.72	10.71	10.65	10.67	10.69
California	Riverside	29.80	27.98	27.92	27.89	27.90	27.91	27.87	27.94	27.87	27.81	27.83	27.85
California	San Bernardino	25.80	24.22	24.18	24.15	24.16	24.17	24.13	24.19	24.13	24.07	24.09	24.11
California	San Diego	17.10	16.00	15.97	15.94	15.95	15.96	15.92	16.30	16.26	16.20	16.22	16.24
California	San Joaquin	16.40	14.78	14.76	14.73	14.74	14.75	14.71	13.89	13.87	13.81	13.83	13.85
California	San Luis Obispo	10.00	9.16	9.15	9.12	9.13	9.14	9.10	8.92	8.90	8.84	8.86	8.88
California	Shasta	10.40	9.45	9.44	9.41	9.42	9.43	9.39	9.07	9.06	9.00	9.02	9.04
California	Sonoma	11.10	9.91	9.90	9.87	9.88	9.89	9.85	9.40	9.39	9.33	9.35	9.37
California	Stanislaus	19.70	17.39	17.37	17.34	17.35	17.36	17.32	16.05	16.03	15.97	15.99	16.01
California	Sutter	12.90	11.87	11.86	11.83	11.84	11.85	11.81	11.34	11.32	11.26	11.28	11.30
California	Tulare	24.70	22.18	22.15	22.12	22.13	22.14	22.10	21.23	21.20	21.14	21.16	21.18
California	Ventura	14.50	13.71	13.69	13.66	13.67	13.68	13.64	13.85	13.82	13.76	13.78	13.80
Colorado	Boulder	9.20	8.79	8.65	8.62	8.63	8.64	8.60	8.79	8.61	8.55	8.57	8.59
Colorado	Mesa	7.30	6.80	6.66	6.63	6.64	6.65	6.61	6.68	6.51	6.45	6.47	6.49
Connecticut	Fairfield	13.59	12.53	11.75	11.19	11.57	11.61	10.66	12.07	10.99	10.65	10.65	10.74
Connecticut	New Haven	16.81	15.47	14.57	14.00	14.42	14.46	13.41	14.99	13.73	13.42	13.42	13.54
Delaware	Kent	12.90	11.89	10.70	9.89	10.41	10.49	9.04	11.21	9.56	9.20	9.20	9.36
Delaware	New Castle	16.62	15.49	14.26	13.35	13.86	13.94	12.48	14.80	13.09	12.49	12.49	12.65
Delaware	Sussex	14.48	13.34	11.99	10.76	11.44	11.53	9.75	12.65	10.78	10.06	10.06	10.22
D.C.	District of Columl	16.56	15.48	13.90	12.84	13.39	13.48	11.72	14.65	12.53	11.93	12.08	11.08
Florida	Alachua	10.86	9.87	9.10	8.10	8.54	8.64	7.40	9.53	8.34	7.69	7.69	7.77
Florida	Broward	9.04	8.37	7.91	7.76	7.99	8.02	7.44	8.26	7.55	7.59	7.59	7.64
Florida	Citrus	10.54	9.46	8.54	7.67	8.04	8.23	6.83	9.18	7.80	7.48	7.48	7.44
Florida	Escambia	13.38	12.38	11.52	9.91	10.72	10.88	9.13	12.03	10.47	9.64	9.64	9.97
Florida	Hillsborough	12.65	11.01	10.38	9.73	10.08	10.17	9.09	10.70	9.63	9.31	9.31	9.40
Florida	Lee	9.63	8.53	7.98	7.49	7.80	7.84	6.99	8.21	7.33	7.08	7.08	7.16
Florida	Leon	13.36	12.18	11.27	10.36	10.79	10.88	9.63	11.75	10.19	9.90	10.01	9.15
Florida	Miami-Dade	8.48	7.67	7.23	7.27	7.46	7.49	6.99	7.54	6.83	7.08	7.08	7.13
Florida	Orange	11.36	10.27	9.58	8.89	9.29	9.35	8.12	9.91	8.82	8.37	8.37	8.53
Florida	Pinellas	11.83	10.30	9.70	9.14	9.46	9.54	8.54	10.02	9.01	8.74	8.74	8.83
Florida	St. Lucie	9.56	8.52	7.96	7.66	7.91	7.90	7.10	8.23	7.32	7.22	7.22	7.25
Florida	Sarasota	10.52	9.18	8.60	8.20	8.50	8.56	7.67	8.87	7.91	7.77	7.77	7.85
Florida	Seminole	10.50	9.30	8.63	8.03	8.38	8.45	7.37	8.90	7.84	7.50	7.50	7.61
Florida	Volusia	10.62	9.53	8.82	8.22	8.59	8.66	7.56	9.12	8.00	7.69	7.69	7.80
Georgia	Bibb	17.63	16.42	15.28	13.20	14.16	14.40	12.10	15.93	13.83	12.86	13.15	11.83

State	County	'99-'01 *EPA CSA Analysis* PM2.5 1999-2001 Ambient Design Value	2010 PM2.5 Base case 2010	2010 PM2.5 Clear Skies 2010	2010 Jeffords	2010 Straw	2010 Carper	2010 No EGU	2020 *EPA CSA Analysis* PM2.5 Base case 2020	2020 PM2.5 Clear Skies 2020	2020 Jeffords	2020 Straw	2020 Carper
Georgia	Chatham	16.50	15.63	14.55	12.57	13.48	13.71	11.53	15.65	13.75	12.78	13.07	11.77
Georgia	Clarke	18.62	17.10	15.76	13.43	14.51	14.86	12.21	16.08	13.53	12.53	12.53	12.85
Georgia	Clayton	19.16	17.79	16.64	14.47	15.44	15.68	13.34	16.82	14.58	13.61	13.61	13.89
Georgia	Cobb	18.56	16.84	15.80	13.39	14.39	14.59	12.19	15.88	13.55	12.53	12.53	12.88
Georgia	DeKalb	19.56	18.31	17.11	14.89	15.95	16.18	13.79	17.65	15.40	14.37	14.37	14.66
Georgia	Dougherty	16.61	15.69	14.66	12.67	13.59	13.82	11.61	15.37	13.39	12.45	12.74	11.46
Georgia	Floyd	18.46	17.01	15.79	13.61	14.67	14.90	12.42	16.25	14.00	12.98	12.98	13.33
Georgia	Fulton	21.21	19.85	18.58	16.21	17.34	17.59	15.05	19.13	16.75	15.65	15.65	15.95
Georgia	Hall	17.25	15.68	14.43	12.21	13.19	13.39	11.02	14.66	12.31	11.32	11.32	11.63
Georgia	Muscogee	17.98	16.73	15.57	13.74	14.62	14.89	12.57	16.11	14.03	13.13	13.13	13.36
Georgia	Paulding	16.77	15.43	14.31	12.36	13.33	13.56	11.28	14.67	12.65	11.70	11.70	12.00
Georgia	Richmond	17.12	16.04	14.78	12.70	13.59	13.88	11.62	15.27	13.06	12.01	12.01	12.28
Georgia	Washington	16.47	15.41	14.31	12.37	13.26	13.49	11.34	14.89	12.90	12.00	12.27	11.05
Georgia	Wilkinson	17.76	16.73	15.68	13.98	14.81	15.07	12.97	16.25	14.21	13.50	13.50	13.76
Idaho	Ada	9.50	8.59	8.58	8.55	8.56	8.57	8.53	8.21	8.19	8.13	8.15	8.17
Idaho	Bannock	10.00	9.25	9.21	9.18	9.19	9.20	9.16	9.06	9.01	8.95	8.97	8.99
Idaho	Canyon	10.20	9.07	9.06	9.03	9.04	9.05	9.01	8.91	8.89	8.83	8.85	8.87
Idaho	Twin Falls	3.20	2.99	2.98	2.95	2.96	2.97	2.93	2.90	2.88	2.82	2.84	2.86
Illinois	Champaign	13.79	13.03	11.78	10.47	11.44	11.48	9.45	12.34	10.67	9.64	9.64	10.15
Illinois	Cook	18.79	17.98	16.90	15.67	16.58	16.61	14.74	17.44	15.94	14.73	14.73	15.36
Illinois	DuPage	15.45	14.79	13.81	12.63	13.53	13.55	11.76	14.18	12.83	11.73	11.73	12.34
Illinois	Madison	17.27	16.32	15.19	13.72	14.76	14.80	12.75	15.71	14.14	12.86	12.86	13.46
Illinois	Randolph	13.91	12.75	11.38	10.32	11.12	11.15	9.54	11.95	10.40	9.46	9.94	9.43
Illinois	St. Clair	17.43	16.39	15.10	13.59	14.67	14.72	12.59	15.74	14.01	12.71	12.71	13.30
Illinois	Sangamon	14.16	13.06	11.93	10.64	11.58	11.62	9.70	12.38	10.90	9.81	9.81	10.33
Illinois	Will	15.87	15.26	14.23	12.92	13.94	13.96	12.02	14.73	13.32	12.11	12.11	12.81
Indiana	Clark	17.34	15.95	14.37	12.62	13.70	13.83	11.15	15.29	13.06	11.89	11.89	12.34
Indiana	Floyd	15.60	14.34	12.89	11.30	12.29	12.41	9.96	13.74	11.71	10.65	10.65	11.06
Indiana	Lake	16.26	15.49	14.49	13.31	14.17	14.22	12.43	14.85	13.47	12.40	12.40	12.98
Indiana	Marion	17.01	15.97	14.45	12.94	13.91	14.01	11.73	15.13	13.19	12.01	12.01	12.45
Indiana	Porter	13.93	13.26	12.41	11.39	12.14	12.17	10.62	12.71	11.53	10.61	10.61	11.11
Iowa	Black Hawk	11.74	10.72	9.91	8.83	9.68	9.67	8.16	9.94	8.88	7.88	7.88	8.40
Iowa	Clinton	12.44	11.52	10.63	9.43	10.38	10.37	8.68	10.84	9.61	8.55	8.55	9.14
Iowa	Johnson	11.64	10.73	9.87	8.71	9.60	9.61	7.98	10.00	8.87	7.81	7.81	8.35
Iowa	Linn	11.35	10.51	9.69	8.51	9.39	9.40	7.79	9.83	8.75	7.63	7.63	8.18
Iowa	Polk	10.85	9.96	9.22	8.24	8.98	9.00	7.62	9.26	8.31	7.35	7.35	7.82

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Iowa	Scott	13.03	12.17	11.24	10.04	10.98	10.99	9.26	11.45	10.21	9.11	9.11	9.70
Iowa	Woodbury	10.00	9.19	8.57	7.66	8.40	8.43	7.10	8.55	7.75	6.83	6.83	7.32
Kansas	Johnson	11.80	10.78	10.06	9.07	9.79	9.86	8.50	10.17	9.24	8.32	8.32	8.80
Kansas	Linn	11.20	10.22	9.46	8.26	9.14	9.22	7.62	9.56	8.56	7.50	7.50	8.08
Kansas	Sedgwick	11.77	10.87	10.21	8.98	9.86	9.97	8.35	10.33	9.50	8.38	8.38	9.03
Kansas	Shawnee	11.25	10.32	9.61	8.55	9.34	9.41	7.90	9.76	8.87	7.83	7.83	8.37
Kentucky	Boyd	15.46	14.49	12.76	11.32	12.20	12.31	10.15	13.72	11.55	10.76	10.76	11.02
Kentucky	Bullitt	16.04	14.39	12.81	11.09	12.16	12.29	9.69	13.61	11.35	10.24	10.24	10.68
Kentucky	Campbell	15.46	14.29	12.79	11.30	12.25	12.35	10.09	13.53	11.54	10.56	10.56	10.86
Kentucky	Carter	12.90	11.92	10.55	9.15	9.99	10.09	8.09	11.25	9.32	8.47	8.76	8.03
Kentucky	Fayette	16.82	15.29	13.64	11.73	12.84	12.98	10.30	14.46	12.14	10.96	10.96	11.35
Kentucky	Franklin	14.53	13.19	11.74	10.03	11.00	11.13	8.77	12.46	10.42	9.35	9.35	9.69
Kentucky	Jefferson	17.08	15.70	14.12	12.33	13.42	13.55	10.84	15.05	12.83	11.62	11.62	12.07
Kentucky	Kenton	15.86	14.61	13.05	11.37	12.45	12.54	10.13	13.91	11.79	10.67	10.67	11.04
Kentucky	McCracken	15.10	13.85	12.53	10.87	11.87	11.99	9.61	13.19	11.41	10.37	10.73	9.83
Kentucky	Pike	16.14	14.87	13.24	11.47	12.49	12.66	10.19	14.08	11.72	10.85	10.85	11.15
Kentucky	Warren	15.41	13.81	12.33	10.43	11.53	11.69	9.24	12.96	10.80	9.65	9.65	10.05
Louisiana	Caddo	13.69	12.86	11.96	10.81	11.67	11.74	10.17	12.57	11.28	10.61	10.61	11.09
Louisiana	Calcasieu	12.75	12.36	11.78	10.50	11.38	11.45	9.96	12.34	11.38	10.65	10.65	11.07
Louisiana	East Baton Roug	14.55	14.03	13.41	12.30	12.96	13.04	11.75	14.25	13.19	12.56	12.56	12.85
Louisiana	Iberville	13.88	13.39	12.72	11.45	12.22	12.31	10.81	13.51	12.35	11.61	11.96	11.66
Louisiana	Jefferson	13.59	13.09	12.37	11.14	11.89	11.97	10.52	13.03	11.84	11.13	11.47	11.19
Louisiana	Lafayette	12.44	11.76	11.10	9.85	10.62	10.70	9.25	11.59	10.50	9.74	9.74	10.10
Louisiana	Orleans	14.15	13.63	12.89	11.67	12.35	12.45	11.00	13.57	12.34	11.64	11.64	11.92
Louisiana	Ouachita	13.04	12.31	11.57	10.45	11.19	11.27	9.82	12.17	11.05	10.32	10.32	10.70
Louisiana	Rapides	13.26	12.38	11.70	10.46	11.25	11.32	9.80	12.50	11.37	10.62	10.62	11.00
Louisiana	Tangipahoa	13.47	12.61	11.88	10.68	11.36	11.46	10.00	12.36	11.13	10.44	10.44	10.73
Louisiana	West Baton Rou	14.06	13.56	12.95	11.88	12.51	12.59	11.34	13.77	12.74	12.12	12.12	12.40
Maine	Androscoggin	10.31	9.35	8.77	8.44	8.73	8.76	8.07	8.97	8.21	8.11	8.11	8.18
Maine	Aroostook	10.79	10.37	9.89	9.62	9.85	9.87	9.34	10.21	9.59	9.51	9.51	9.57
Maine	Cumberland	11.65	10.51	9.88	9.59	9.90	9.94	9.21	10.06	9.23	9.18	9.18	9.26
Maine	Hancock	6.03	5.51	5.08	4.87	5.05	5.08	4.67	5.34	4.77	4.75	4.75	4.79
Maine	Kennebec	9.97	9.04	8.47	8.16	8.43	8.47	7.82	8.66	7.91	7.83	7.83	7.90
Maine	Oxford	10.45	9.74	9.10	8.62	8.90	8.93	8.28	9.42	8.61	8.53	8.60	8.04
Maine	Penobscot	9.38	8.55	7.99	7.72	7.97	8.00	7.40	8.21	7.49	7.43	7.43	7.49
Maryland	Baltimore city	17.83	16.62	14.98	13.88	14.47	14.57	12.67	15.83	13.57	12.91	12.91	13.07

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Massachusetts	Hampden	14.10	13.02	12.18	11.67	12.07	12.10	11.09	12.55	11.42	11.17	11.17	11.26
Massachusetts	Hampshire	9.02	8.27	7.69	7.19	7.46	7.50	6.70	7.92	7.16	7.42	6.98	6.47
Massachusetts	Worcester	12.68	11.55	10.81	10.42	10.75	10.78	9.95	10.99	9.99	9.79	9.79	9.88
Michigan	Allegan	12.23	11.44	10.81	9.93	10.63	10.64	8.99	10.85	9.84	8.97	8.97	9.46
Michigan	Berrien	12.51	11.67	10.89	9.96	10.67	10.69	9.13	11.02	9.89	9.03	9.03	9.49
Michigan	Genesee	12.70	11.90	11.13	10.39	10.96	10.97	9.70	11.30	10.25	9.56	9.56	9.87
Michigan	Ingham	13.15	12.23	11.43	10.52	11.24	11.24	9.68	11.55	10.42	9.55	9.55	9.98
Michigan	Kalamazoo	15.01	13.99	13.02	11.94	12.76	12.78	11.01	13.22	11.84	10.88	10.88	11.37
Michigan	Kent	14.06	13.11	12.29	11.29	12.07	12.08	10.37	12.39	11.20	10.27	10.27	10.76
Michigan	Macomb	13.25	12.52	11.72	11.02	11.56	11.56	10.34	11.94	10.84	10.24	10.24	10.51
Michigan	Muskegon	12.19	11.48	10.84	10.04	10.68	10.69	9.29	10.95	9.97	9.15	9.15	9.64
Michigan	Ottawa	13.33	12.43	11.65	10.70	11.44	11.45	9.82	11.75	10.62	9.72	9.72	10.19
Michigan	St. Clair	13.80	13.08	12.22	11.28	12.00	12.01	10.42	12.53	11.33	11.28	10.90	10.22
Michigan	Wayne	18.91	17.98	16.81	15.76	16.56	16.57	14.76	17.25	15.62	14.75	14.75	15.11
Mississippi	DeSoto	13.98	12.70	11.63	10.30	11.01	11.15	9.54	12.02	10.46	10.92	10.10	9.39
Mississippi	Hancock	12.16	11.39	10.70	9.64	10.27	10.38	8.99	11.26	10.09	9.55	9.55	9.80
Mississippi	Hinds	15.09	13.94	13.02	11.73	12.51	12.61	11.02	13.46	11.95	11.24	11.24	11.57
Mississippi	Jackson	13.82	12.93	12.16	10.87	11.49	11.70	10.03	12.79	11.21	10.71	10.71	10.94
Mississippi	Jones	16.62	15.21	14.16	12.62	13.50	13.66	11.70	14.76	12.96	12.16	12.16	12.50
Mississippi	Lee	14.20	12.84	11.80	10.26	11.16	11.30	9.32	12.19	10.54	9.62	9.62	9.99
Missouri	Buchanan	12.43	11.34	10.55	9.42	10.26	10.33	8.69	10.63	9.60	8.53	8.53	9.08
Missouri	Cedar	11.52	10.51	9.63	8.34	9.28	9.36	7.63	9.80	8.68	7.55	7.55	8.16
Missouri	Clay	12.84	11.83	11.09	10.06	10.81	10.88	9.41	11.26	10.31	9.30	9.30	9.81
Missouri	Greene	12.24	11.27	10.30	8.96	9.91	9.98	8.14	10.59	9.34	8.25	8.25	8.80
Missouri	Jackson	13.87	12.76	11.98	10.89	11.68	11.75	10.19	12.14	11.14	10.07	10.07	10.61
Missouri	Jasper	13.69	12.54	11.50	9.95	11.05	11.15	9.14	11.71	10.39	9.08	9.08	9.79
Missouri	Jefferson	14.97	14.11	12.93	11.58	12.55	12.60	10.69	13.57	12.01	10.86	10.86	11.38
Missouri	Monroe	11.01	10.08	9.23	8.15	8.96	8.99	7.44	9.42	8.32	7.38	7.38	7.87
Missouri	St. Charles	14.64	13.85	12.85	11.54	12.47	12.50	10.68	13.33	11.96	10.80	10.80	11.33
Missouri	Ste. Genevieve	14.19	13.08	11.87	10.45	11.48	11.54	9.51	12.37	10.77	9.62	9.62	10.17
Missouri	St. Louis	14.12	13.35	12.40	11.00	11.88	11.93	10.19	12.84	11.53	10.29	10.29	10.77
Missouri	St. Louis city	16.28	15.32	14.13	12.73	13.73	13.78	11.81	14.72	13.12	11.92	11.92	12.47
Montana	Lewis And Clark	8.50	8.30	8.28	8.25	8.26	8.27	8.23	8.32	8.29	8.23	8.25	8.27
Montana	Lincoln	16.40	15.52	15.49	15.46	15.47	15.48	15.44	14.97	14.94	14.88	14.90	14.92
Montana	Missoula	11.80	11.15	11.12	11.09	11.10	11.11	11.07	10.84	10.81	10.75	10.77	10.79
Montana	Yellowstone	8.00	7.63	7.53	7.50	7.51	7.52	7.48	7.55	7.41	7.35	7.37	7.39

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Nebraska	Lancaster	10.52	9.65	8.96	8.93	8.94	8.95	8.91	9.04	8.19	8.13	8.15	8.17
Nevada	Clark	11.00	10.39	10.32	10.29	10.30	10.31	10.27	9.83	9.71	9.65	9.67	9.69
Nevada	Washoe	9.70	8.93	8.92	8.89	8.90	8.91	8.87	8.55	8.53	8.47	8.49	8.51
New Jersey	Hudson	17.22	14.65	13.77	13.17	13.51	13.56	12.58	14.11	12.86	12.46	12.46	12.55
New Jersey	Mercer	14.31	13.46	12.45	11.97	12.23	12.26	11.51	12.95	11.54	12.11	11.31	10.76
New Jersey	Union	16.27	14.20	13.56	13.17	13.40	13.44	12.75	13.91	12.96	12.68	12.68	12.77
New Mexico	Dona Ana	10.90	10.15	10.00	9.97	9.98	9.99	9.95	9.92	9.71	9.65	9.67	9.69
New Mexico	Grant	5.70	5.52	5.47	5.44	5.45	5.46	5.42	5.52	5.45	5.39	5.41	5.43
New Mexico	Lea	6.90	6.47	6.30	6.27	6.28	6.29	6.25	6.30	6.07	6.01	6.03	6.05
New Mexico	Sandoval	5.00	4.91	4.84	4.81	4.82	4.83	4.79	4.96	4.88	4.82	4.84	4.86
New Mexico	Santa Fe	4.80	4.53	4.47	4.44	4.45	4.46	4.42	4.45	4.36	4.30	4.32	4.34
New York	New York	18.05	16.35	15.37	14.60	15.02	15.06	13.96	15.49	14.14	13.60	13.60	13.70
North Carolina	Alamance	15.32	13.91	12.59	11.37	12.17	12.34	10.31	13.05	11.07	10.71	10.71	10.93
North Carolina	Cabarrus	15.67	13.71	12.67	11.07	12.04	12.18	10.04	12.88	11.14	10.28	10.28	10.55
North Carolina	Catawba	17.11	15.37	14.01	12.12	13.20	13.35	10.97	14.43	12.19	11.32	11.32	11.59
North Carolina	Chatham	13.42	12.22	11.08	9.90	10.62	10.75	8.98	11.50	9.72	9.28	9.28	9.47
North Carolina	Cumberland	15.44	14.24	12.92	11.50	12.27	12.41	10.50	13.55	11.55	10.91	10.91	11.10
North Carolina	Davidson	17.28	15.58	14.27	12.56	13.59	13.74	11.40	14.69	12.53	11.79	11.79	12.03
North Carolina	Duplin	12.65	11.57	10.46	9.22	9.89	10.00	8.35	11.00	9.34	8.71	8.71	8.88
North Carolina	Durham	15.35	14.25	12.91	11.58	12.38	12.51	10.58	13.50	11.49	10.97	10.97	11.15
North Carolina	Forsyth	16.23	14.52	13.26	11.69	12.65	12.88	10.53	13.60	11.63	10.88	10.88	11.15
North Carolina	Gaston	15.29	13.87	12.89	11.16	12.22	12.31	10.21	13.12	11.25	10.49	10.49	10.77
North Carolina	Guilford	16.25	14.79	13.41	11.72	12.65	12.80	10.62	13.86	11.79	12.60	11.34	10.14
North Carolina	Haywood	15.38	13.90	12.74	10.88	11.93	12.10	9.75	13.24	11.18	10.30	10.30	10.65
North Carolina	McDowell	16.17	14.61	13.33	11.56	12.60	12.77	10.40	13.91	11.76	10.97	10.97	11.25
North Carolina	Mecklenburg	16.77	15.22	14.08	12.29	13.25	13.41	11.23	14.33	12.37	11.49	11.49	11.74
North Carolina	Mitchell	15.46	13.97	12.71	10.85	11.94	12.12	9.69	13.24	11.06	10.28	10.28	10.60
North Carolina	New Hanover	12.19	11.33	10.43	9.50	10.08	10.18	8.74	10.98	9.51	9.10	9.10	9.28
North Carolina	Onslow	12.14	11.16	10.13	9.04	9.65	9.76	8.21	10.63	9.05	8.53	8.53	8.69
North Carolina	Orange	14.32	13.05	11.83	10.55	11.33	11.47	9.57	12.28	10.38	9.90	9.90	10.11
North Carolina	Swain	14.12	12.83	11.69	9.99	10.95	11.13	8.94	12.15	10.19	9.43	9.43	9.75
North Carolina	Wake	15.30	14.21	12.87	11.49	12.29	12.42	10.49	13.46	11.45	10.85	10.85	11.03
North Carolina	Wayne	15.30	14.11	12.75	11.31	12.09	12.24	10.24	13.43	11.44	10.76	10.76	10.95
North Dakota	Cass	8.58	7.90	7.45	6.78	7.27	7.30	6.43	7.40	6.86	6.15	6.15	6.51
North Dakota	Mercer	6.90	6.37	6.01	5.50	5.90	5.92	5.22	5.68	5.52	5.39	5.24	4.98
North Dakota	Steele	6.93	6.36	6.03	5.52	5.92	5.94	5.23	5.91	5.51	5.41	5.23	4.98

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Ohio	Butler	17.41	16.14	14.57	13.15	14.06	14.15	11.83	15.24	13.14	12.19	12.19	12.46
Ohio	Cuyahoga	20.25	19.20	17.69	16.60	17.37	17.42	15.56	18.37	16.38	15.64	15.64	15.94
Ohio	Franklin	18.13	16.80	15.06	13.73	14.65	14.74	12.52	16.00	13.78	12.97	12.97	13.29
Ohio	Hamilton	19.29	17.86	16.04	14.26	15.40	15.52	12.79	16.92	14.49	13.31	13.31	13.68
Ohio	Jefferson	18.90	18.08	16.21	15.02	15.81	15.92	13.80	17.36	15.06	14.44	14.44	14.70
Ohio	Lake	13.95	13.47	12.25	11.51	12.12	12.15	10.72	12.91	11.34	10.88	10.88	11.10
Ohio	Lorain	15.08	14.28	13.14	11.94	12.69	12.76	10.92	13.58	12.08	12.41	11.65	10.79
Ohio	Mahoning	16.42	15.46	13.79	12.78	13.52	13.59	11.73	14.66	12.63	12.08	12.08	12.30
Ohio	Montgomery	17.65	16.50	14.92	13.45	14.40	14.49	12.22	15.65	13.57	12.49	12.49	12.84
Ohio	Portage	15.29	14.47	13.00	12.01	12.74	12.79	11.05	13.75	11.93	11.34	11.34	11.58
Ohio	Scioto	20.04	18.54	16.41	14.68	15.79	15.94	13.13	17.42	14.71	13.83	13.83	14.13
Ohio	Stark	18.29	17.16	15.26	13.91	14.84	14.92	12.65	16.20	13.85	13.05	13.05	13.35
Ohio	Summit	17.34	16.42	14.77	13.63	14.46	14.52	12.56	15.61	13.56	12.88	12.88	13.15
Ohio	Trumbull	16.16	15.20	13.60	12.61	13.33	13.40	11.60	14.41	12.45	11.90	11.90	12.12
Oregon	Benton	7.40	6.97	6.97	6.94	6.95	6.96	6.92	6.79	6.78	6.72	6.74	6.76
Oregon	Columbia	6.60	6.08	6.06	6.03	6.04	6.05	6.01	5.82	5.80	5.74	5.76	5.78
Oregon	Jackson	11.30	10.34	10.33	10.30	10.31	10.32	10.28	9.77	9.76	9.70	9.72	9.74
Oregon	Klamath	9.70	9.13	9.12	9.09	9.10	9.11	9.07	8.86	8.85	8.79	8.81	8.83
Oregon	Lake	7.60	7.18	7.17	7.14	7.15	7.16	7.12	6.98	6.97	6.91	6.93	6.95
Oregon	Lane	13.20	12.23	12.21	12.18	12.19	12.20	12.16	11.77	11.75	11.69	11.71	11.73
Oregon	Marion	8.20	7.59	7.58	7.55	7.56	7.57	7.53	7.28	7.27	7.21	7.23	7.25
Oregon	Multnomah	9.10	8.58	8.57	8.54	8.55	8.56	8.52	8.36	8.35	8.29	8.31	8.33
Oregon	Umatilla	8.80	8.17	8.15	8.12	8.13	8.14	8.10	7.82	7.80	7.74	7.76	7.78
Oregon	Washington	7.80	7.35	7.34	7.31	7.32	7.33	7.29	7.16	7.15	7.09	7.11	7.13
Pennsylvania	Allegheny	21.02	19.30	16.73	14.97	16.00	16.18	13.56	18.03	15.11	14.15	14.15	14.44
Pennsylvania	Berks	15.62	14.48	13.15	12.23	12.72	12.79	11.36	13.82	11.98	11.38	11.38	11.51
Pennsylvania	Cambria	15.32	14.19	12.25	11.10	11.82	11.94	9.99	13.32	11.02	10.44	10.44	10.64
Pennsylvania	Dauphin	15.52	14.33	12.69	11.57	12.12	12.21	10.53	13.62	11.37	10.70	10.70	10.85
Pennsylvania	Lancaster	16.91	15.43	13.82	12.58	13.18	13.27	11.56	14.53	12.39	11.58	11.58	11.73
Pennsylvania	Philadelphia	16.55	15.66	14.59	13.68	14.13	14.19	12.91	15.18	13.66	13.06	13.06	13.18
Pennsylvania	Washington	15.55	14.29	12.31	10.92	11.73	11.87	9.83	13.33	11.10	10.35	10.35	10.57
Pennsylvania	Westmoreland	15.60	14.30	12.36	11.07	11.83	11.96	10.03	13.34	11.14	10.44	10.44	10.65
Pennsylvania	York	16.25	15.03	13.46	12.41	12.97	13.05	11.35	14.22	12.12	11.45	11.45	11.61
South Carolina	Charleston	12.62	11.85	11.04	9.74	10.34	10.47	8.94	11.62	10.19	9.35	9.35	9.52
South Carolina	Georgetown	13.91	12.90	11.92	10.35	11.15	11.31	9.44	12.57	10.79	11.07	10.29	9.25
South Carolina	Greenville	16.51	15.16	13.89	11.87	12.99	13.17	10.78	14.33	12.01	11.12	11.12	11.45

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South Carolina	Lexington	15.62	14.43	13.28	11.73	12.53	12.74	10.70	13.73	11.69	10.99	10.99	11.32
South Carolina	Oconee	12.29	11.16	10.19	8.85	9.53	9.67	8.07	10.51	8.72	7.50	8.31	7.47
South Carolina	Richland	15.39	14.25	13.10	11.50	12.29	12.47	10.49	13.58	11.54	10.78	10.78	11.07
South Carolina	Spartanburg	15.37	14.09	12.92	11.06	12.10	12.27	10.04	13.30	11.18	10.35	10.35	10.66
South Dakota	Minnehaha	10.42	9.62	8.94	8.91	8.92	8.93	8.89	8.88	8.06	8.00	8.02	8.04
Tennessee	Davidson	17.05	15.37	13.95	12.01	13.19	13.33	10.75	14.62	12.49	11.32	11.32	11.78
Tennessee	Hamilton	18.46	16.83	15.37	13.37	14.56	14.77	12.09	15.88	13.44	12.63	12.63	13.01
Tennessee	Knox	20.42	18.41	16.74	14.21	15.55	15.84	12.72	17.41	14.53	13.35	13.35	13.78
Tennessee	Roane	17.02	15.22	13.78	11.60	12.75	13.07	10.27	14.35	11.88	10.85	10.85	11.24
Tennessee	Shelby	15.56	14.86	13.75	12.23	13.17	13.36	11.30	14.26	12.51	11.55	11.55	12.03
Tennessee	Sullivan	16.98	15.35	13.92	11.93	13.11	13.24	10.74	14.60	12.16	11.28	11.28	11.61
Tennessee	Sumner	15.68	14.12	12.79	10.96	12.06	12.20	9.78	13.42	11.43	10.32	10.32	10.75
Utah	Davis	9.00	8.83	8.79	8.76	8.77	8.78	8.74	8.67	8.61	8.55	8.57	8.59
Utah	Salt Lake	13.60	13.35	13.28	13.25	13.26	13.27	13.23	13.10	13.01	12.95	12.97	12.99
Utah	Tooele	7.20	7.22	7.19	7.16	7.17	7.18	7.14	7.29	7.25	7.19	7.21	7.23
Utah	Utah	10.40	10.11	10.04	10.01	10.02	10.03	9.99	10.02	9.93	9.87	9.89	9.91
Utah	Weber	8.80	8.56	8.51	8.48	8.49	8.50	8.46	8.46	8.39	8.33	8.35	8.37
Vermont	Bennington	9.86	9.12	8.45	8.01	8.34	8.37	7.57	8.81	7.91	7.71	7.71	7.78
Vermont	Chittenden	6.76	6.31	5.83	5.65	5.84	5.86	5.39	6.07	5.46	5.41	5.41	5.46
Vermont	Rutland	11.32	10.46	9.71	9.22	9.59	9.62	8.75	10.06	9.07	8.84	8.84	8.93
Vermont	Washington	10.47	9.72	9.04	8.60	8.93	8.96	8.17	9.35	8.47	8.26	8.26	8.34
Virginia	Bristol city	16.01	14.33	13.00	11.18	12.22	12.38	10.08	13.55	11.35	10.52	10.52	10.81
Virginia	Newport News ci	12.67	11.88	10.91	10.05	10.62	10.70	9.38	11.55	10.17	9.74	9.74	9.94
Virginia	Roanoke city	15.24	14.00	12.42	10.82	11.76	11.90	9.67	13.20	11.07	10.31	10.31	10.53
Virginia	Virginia Beach ci	13.21	12.41	11.39	10.45	11.06	11.15	9.70	12.10	10.60	10.14	10.14	10.35
Washington	King	11.90	11.38	11.34	11.31	11.32	11.33	11.29	11.13	11.07	11.01	11.03	11.05
Washington	Pierce	11.70	11.00	10.96	10.93	10.94	10.95	10.91	10.69	10.63	10.57	10.59	10.61
Washington	Snohomish	11.40	10.66	10.61	10.58	10.59	10.60	10.56	10.25	10.19	10.13	10.15	10.17
Washington	Spokane	10.40	9.61	9.58	9.55	9.56	9.57	9.53	9.14	9.11	9.05	9.07	9.09
Washington	Thurston	9.70	8.72	8.68	8.65	8.66	8.67	8.63	8.25	8.20	8.14	8.16	8.18
Washington	Whatcom	7.90	7.48	7.45	7.42	7.43	7.44	7.40	7.26	7.23	7.17	7.19	7.21
West Virginia	Berkeley	16.01	14.77	12.93	11.65	12.37	12.49	10.43	13.88	11.55	10.92	10.92	11.05
West Virginia	Brooke	17.40	16.65	14.91	13.80	14.54	14.64	12.67	15.97	13.84	13.27	13.27	13.51
West Virginia	Cabell	17.85	16.51	14.50	12.97	13.97	14.10	11.65	15.55	13.05	12.31	12.31	12.60
West Virginia	Hancock	17.36	16.60	14.88	13.77	14.50	14.60	12.64	15.93	13.82	13.23	13.23	13.47
West Virginia	Harrison	14.78	13.63	11.75	10.39	11.27	11.39	9.12	12.83	10.54	9.94	9.94	10.15

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West Virginia	Kanawha	18.39	17.19	14.89	13.43	14.42	14.55	12.03	16.25	13.50	12.85	12.85	13.14
West Virginia	Marshall	16.52	15.58	13.43	11.96	12.88	12.99	10.70	14.57	12.18	11.52	11.52	11.72
West Virginia	Monongalia	14.95	13.83	11.80	10.51	11.35	11.47	9.27	12.91	10.61	10.05	10.05	10.23
West Virginia	Ohio	15.66	14.69	12.59	11.31	12.14	12.31	10.13	13.74	11.37	10.76	10.76	10.99
West Virginia	Raleigh	14.02	12.83	11.31	9.95	10.81	10.93	8.82	12.09	10.00	9.42	9.42	9.64
West Virginia	Summers	10.89	9.96	8.70	7.62	8.31	8.41	6.72	9.36	7.71	7.24	7.24	7.41
West Virginia	Wood	17.62	16.36	14.21	12.71	13.73	13.86	11.33	15.39	12.91	12.18	12.18	12.47
Wisconsin	Brown	11.43	10.63	9.94	9.11	9.78	9.77	8.45	10.06	9.12	8.30	8.30	8.75
Wisconsin	Dane	13.16	12.22	11.37	10.32	11.17	11.17	9.60	11.52	10.36	9.33	9.33	9.93
Wisconsin	Dodge	11.77	10.86	10.08	9.13	9.88	9.88	8.43	10.18	9.10	8.16	8.16	8.66
Wisconsin	Door	8.02	7.58	7.14	6.65	7.06	7.06	6.21	7.31	6.72	6.24	6.24	6.51
Wisconsin	Douglas	8.32	7.88	7.52	7.12	7.47	7.47	6.80	7.83	7.35	6.97	6.97	7.22
Wisconsin	Grant	12.27	11.24	10.37	9.54	10.22	10.22	8.90	10.46	9.31	9.48	8.96	8.48
Wisconsin	Jefferson	12.52	11.60	10.80	9.86	10.61	10.61	9.15	10.94	9.81	8.89	8.89	9.40
Wisconsin	Kenosha	12.14	11.60	10.87	9.96	10.72	10.73	9.29	11.13	10.12	9.21	9.21	9.79
Wisconsin	Manitowoc	10.25	9.55	8.94	8.18	8.78	8.77	7.56	9.08	8.23	7.44	7.44	7.87
Wisconsin	Milwaukee	14.18	13.56	12.77	11.86	12.61	12.62	11.10	13.08	11.93	10.97	10.97	11.53
Wisconsin	Outagamie	11.27	10.47	9.82	9.05	9.66	9.66	8.45	9.97	9.08	8.29	8.29	8.72
Wisconsin	Vilas	6.39	5.97	5.61	5.16	5.53	5.53	4.81	5.70	5.23	4.40	5.03	4.76
Wisconsin	Waukesha	14.10	13.28	12.45	11.47	12.26	12.26	10.73	12.63	11.45	10.47	10.47	11.03
Wisconsin	Winnebago	11.19	10.34	9.66	8.85	9.50	9.50	8.21	9.75	8.82	8.01	8.01	8.45
Wisconsin	Wood	10.61	9.71	9.09	8.36	8.96	8.96	7.80	9.15	8.31	7.13	8.00	7.56
Wyoming	Laramie	5.40	5.25	5.14	5.11	5.12	5.13	5.09	5.32	5.17	5.11	5.13	5.15
Wyoming	Sheridan	10.90	10.21	10.05	10.02	10.03	10.04	10.00	10.02	9.79	9.73	9.75	9.77