Health Benefits of Alternative $PM_{2.5}$ Standards

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Executive Summary

The United States Environmental Protection Agency (EPA) is presently conducting a review of the national ambient air quality standards (NAAQS) for particulate matter (PM). These standards are established for pollutants that may reasonably be anticipated to endanger public health and welfare, and whose presence in the ambient air results from numerous or diverse mobile or stationary sources. As part of its periodic review of the PM NAAQS, EPA released in June 2010 two reports: *Quantitative Health Risk Assessment for Particulate Matter* and the *Second External Review Draft of the Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards*. These reports are reviewed by the Clean Air Scientific Advisory Committee and help to inform the EPA Administrator's decision to revise the air quality standards as appropriate.

The present report seeks to augment the information developed by EPA and to highlight the potential health benefits that might be realized nationwide from the standards under consideration in the current NAAQS review. EPA's risk assessment focused on 15 major urban areas while looking at alternative annual standards levels of 14, 13, and 12 $\mu g/m^3$ combined with a daily standard of 35, and two combinations of an annual and daily standard were analyzed – 13 $\mu g/m^3$ annual coupled with a 30 $\mu g/m^3$ daily standard (denoted 13/30) as well as a 12/25 standard. This report evaluates more stringent standard combinations, including an 11/35 standard put forth in the April 2011 Policy Assessment for the Review of the PM NAAQS (U.E. EPA 2011). In addition, the risk assessment includes an abbreviated national-scale analysis using the Environmental Benefits Mapping and Analysis Program (BenMAP), to examine the representativeness of its 15-city risk assessment.

The goal of the present report is to conduct a national analysis of the mortality and morbidity benefits of a greater range of annual and daily standards, and to conduct the analysis with the same types of tools that EPA uses in its own analyses.

To estimate $PM_{2.5}$-related human health impacts, this analysis uses the Benefits Mapping and Analysis Program (BenMAP), the model which the EPA uses in regulatory benefit analyses (e.g., U.S. EPA2010b). Abt Associates developed this tool for EPA to analyze the health impacts of national-scale air quality regulations. BenMAP calculates the difference in air quality between two scenarios, designated as "baseline" and "control," in each grid cell for which BenMAP has estimated population exposure to $PM_{2.5}$. Given the difference in exposure between the two scenarios, BenMAP then calculates the associated change in adverse health effects, such as premature mortality, using health impact functions. Table 1 presents key mortality results from the primary analysis by scenario.

Lastly, BenMAP assigns an economic value to these effects. To estimate the economic benefit of the estimated change in health incidence, BenMAP multiplies the number of adverse cases of a specific type of effect (e.g., mortality) by its associated unit value and then adjusts for the estimated change in income over time:

$$\$Benefit = Cases\ Health\ Effects * Unit\ Value * Income\ Adjustment$$

A reasonable range of estimated results is presented as a 90 percent confidence interval (5^{th} to 95^{th} percentiles along with the mean estimate ; however, not all sources of certainty can be quantified. The estimated population exposure uncertainty is unquantified, and similarly, the uncertainty associated with the proportional rollback approach used in this analysis is unquantified.

To help avoid over-estimating the likely impacts of alternative standards, conservative assumptions are used throughout the analysis. When calculating mortality, no effects are assumed to occur below the lowest measured level found in the study. In addition, the rollback approach used in the present analysis tends to result in relatively small changes in $PM_{2.5}$ levels, in comparison to the rollback approaches used by EPA (2010a).

Table 1. Updated Air Quality Standards for Fine Particles Will Save Lives : Estimated Premature Deaths Avoided in U.S. by Alternative PM2.5 Air Quality Standards and Epidemiological Study (relative to current air quality), Mean and 90% Confidence Interval

Scenario	Krewski *et al.* (2009)	Laden *et al.* (2006)
A15D35	2,540	5,240
	(1,850 – 3,220)	(2,850 – 7,570)
A13D35	**3,700**	**8,190**
	(2,700 – 4,700)	**(4,450 – 11,900)**
A13D30	**6,410**	**13,500**
	(4,680 – 8,130)	**(7,330 – 19,600)**
A13D25	16,700	32,700
	(12,200 – 21,200)	(17,700 – 47,300)
A12D35	**6,380**	**15,000**
	(4,650 – 8,100)	**(8,140 – 21,800)**
A12D30	7,980	17,500
	(5820 – 10,100)	(9,490 – 25,400)
A12D25	16,800	33,000
	(12,300 – 21,300)	(17,900 – 47,800)
A11D35	**11,200**	**27,300**
	(8,200 – 14,300)	**(14,800 – 39,600)**
A11D30	12,100	27,900
	(8,830 – 15,400)	(15,100 – 40,400)
A11D25	17,900	35,700
	(13,100 – 22,700)	(19,400 – 51,800)

Bold type highlights options put forth by EPA in its Policy Assessment

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List of Acronyms

Acronym	Description
ALA	American Lung Association
BenMAP	Environmental Benefits Mapping and Analysis Program
CASAC	Clean Air Scientific Advisory Committee
CATF	Clean Air Task Force
CMAQ	Community Multi-Scale Air Quality Modeling System
COI	cost of illness
DV	design value
EPA	U.S. Environmental Protection Agency
HCUP	Healthcare Cost and Utilization Project
ICD	International Classification of Disease
LML	lowest measured level (in an epidemiological study)
NAAQS	national ambient air quality standard
$PM_{2.5}$	fine particulate matter less than 2.5 microns in aerodynamic diameter
PRB	policy relevant background
$\mu g/m^3$	micrograms per meter cubed
VNA	voronoi neighbor averaging
VSL	value of a statistical life
WTP	willingness to pay

1. Introduction

The United States Environmental Protection Agency (EPA) is presently conducting a review of the national ambient air quality standards (NAAQS) for particulate matter (PM). These standards are established for pollutants that may reasonably be anticipated to endanger public health and welfare, and whose presence in the ambient air results from numerous or diverse mobile or stationary sources. As part of its periodic review of the PM NAAQS, EPA released in June 2010 two reports: Quantitative Health Risk Assessment for Particulate Matter (EPA-452/R-10-005) and the Second External Review Draft of the Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards (EPA-452/P-10-007). These reports are reviewed by the Clean Air Scientific Advisory Committee (CASAC) and help to inform the EPA Administrator's decision to revise the air quality standards as appropriate.

The present report seeks to augment the information developed by EPA and to highlight the potential health benefits that might be realized nationwide from the standards under consideration in the current NAAQS review. EPA's risk assessment focused on 15 major urban areas while looking at alternative annual standards levels of 14, 13, and 12 $\mu g/m^3$ combined with a daily standard of 35, and two combinations of an annual and daily standard were analyzed – 13 $\mu g/m^3$ annual coupled with a 30 µg/m3 daily standard (denoted 13/30) as well as a 12/25 standard. In addition, the risk assessment conducted an abbreviated national-scale analysis using the Environmental Benefits Mapping and Analysis Program (BenMAP), using the limited national analysis, which focused on mortality impacts, to examine the representativeness of its 15-city risk assessment.

In April 2011, EPA published a final Policy Assessment reflecting additional analysis and the comments of the CASAC and the public on two prior draft documents. The final policy assessment lays out EPA staff scientists' recommendations for revised $PM_{2.5}$ standards. They proposed somewhat different levels than used in the risk assessment. Specifically, the policy assessment concluded that the current annual $PM_{2.5}$ standard of 15 $\mu g/m^3$ should be revised to a level within the range of 13 to 11 $\mu g/m^3$, with evidence most strongly supporting a standard in the range of 12 to 11 $\mu g/m^3$. In addition, staff recommended retention of the current 24-hour standard of 35 $\mu g/m^3$ with possible consideration of a revised standard of 30 $\mu g/m^3$ in conjunction with an annual standard level of 13 $\mu g/m^3$.

The goal of the present report is to conduct a national analysis of the mortality and morbidity benefits of a greater range of annual and daily standards, including those recommended in the final Policy Assessment, and to conduct the analysis, using the same types of tools that EPA uses in its own analyses. The report is outlined as follows: Section 2 describes the $PM_{2.5}$ monitoring data used in this analysis, the calculation of annual and daily design values, and the approach used to reduce, or rollback, daily $PM_{2.5}$ values so as to meet alternative standards. Section 3 describes the use of BenMAP in estimating the avoided health impacts due to meeting more stringent $PM_{2.5}$ standards, as well as the economic value of these avoided health effects. Section 4 presents the results of the analysis. Finally, Appendix A details the health impact functions used in this report; Appendix B provides a brief summary of some of the key health incidence databases used; and Appendix C describes the population data.

2. Estimating $PM_{2.5}$ Exposure with Alternative Annual & Daily Standards

To estimate $PM_{2.5}$ levels in a uniform grid across the United States, this analysis uses the domain from the Community Multi-Scale Air Quality Modeling System (CMAQ), which has grid cells that are approximately 12 kilometers by 12 kilometers. CMAQ is one of EPA's preferred air quality modeling tools.[1] The first step in estimating $PM_{2.5}$ exposure is to work with the monitoring data to develop as complete a set of data as possible. The second step is to adjust, or roll back each monitor so that it just meets the prescribed annual and daily standards. The final step is to calculate for each grid cell an inverse-distance-weighted concentration of the monitors within 60 kilometers of the center of each grid cell, dropping from consideration any grid cell further than 60 kilometers from a monitor.

The present analysis uses three years of monitoring data so as to follow as closely as possible EPA's approach for calculating whether a region is in attainment or not. Note, however, that this analysis differs in a number of ways from EPA's (2010a) *Quantitative Health Risk Assessment for Particulate Matter*, which evaluates the health impacts of alternative $PM_{2.5}$ standards. The present analysis is national in scope, whereas the EPA analysis focuses on 15 urban areas representative of the continental US.[2] The present analysis uses monitoring data from 2007–2009 (the most recent complete set of data available), while the EPA analysis uses data from 2005–2007. The present analysis combines the three years of data to calculate design values and then calculates the associated health impacts for each standard combination. The EPA analysis used the maximum design value in an urban area to calculate the required percent reduction in ambient $PM_{2.5}$ to meet the standard combination, determined the 'controlling' standard, and then applied the reduction to a composite monitor in each urban area in each year individually.[3] Finally, the present analysis uses a proportional rollback approach to estimate the percent reduction needed to meet an alternative standard at each monitor, whereas the EPA used a different proportional rollback approach as well as two other rollback approaches.

Below, there is a description of the monitoring data and how they are rolled back to meet different combinations of annual and daily standards, and then how the BenMAP model averaged the monitoring data to estimate exposure in each grid cell.

2.1 Monitoring Data

This analysis relies on the three most recent available years of available quality-controlled ambient air quality $PM_{2.5}$ data, 2007 to 2009. For most monitoring sites, three years of data were available for the

[1] The CMAQ model is described at the EPA website: http://www.epa.gov/ttn/scram/photochemicalindex.htm
[2] EPA (2010a, Appendix G) also conducted a national benefits analysis, but this was not designed to evaluate the benefits of alternative scenarios.
[3] Details on the rollback approaches used by EPA can be found in EPA (2010a) starting on page 3-18.

calculation of annual and daily design values.[4] For sites with fewer than three years of data, annual and 98th percentile calculations were constructed based on available complete years. In some cases, two nearby sites with incomplete three-year data sets were combined to cover the entire three-year period. In a couple instances, the annual averages were constructed from multiple years such that all four seasons were sampled (e.g., winter 2007 through fall 2008, reported as 2008).[5]

Included in the analysis are all monitors with at least one year of data, in which each quarter in that year has at least nine daily values. For these monitors, an annual design value was calculated, which is an average of up to three years of data, using the following series of formulas:

$$Quarterly\ Average_j = \sum_{i=1}^{n} \frac{PM_{2.5,i}}{n}$$

where:
n = number of daily observations in a given quarter "j".

$$Annual\ Average_k = \sum_{j=1}^{4} \frac{Quarterly\ Average_j}{4}$$

$$Annual\ Design\ Value_m = \sum_{k=1}^{l} \frac{Annual\ Average_k}{l}$$

where:
l = number of annual averages available for a given monitor "m."

For each monitor for which an annual design value was calculated, a daily design value was also calculated. The first step was to count the number of daily $PM_{2.5}$ measurements in a given year, and then, based on this number, to calculate the 98th percentile value:

*98^{th} $Percentile_k$ = $NumberDays_k$ – TruncationFunction(0.98 * $NumberDays_k$)*

where:
$NumberDays_k$ = number of days with daily $PM_{2.5}$ values in year "k."
TruncationFunction = function in Excel that keeps the integers to the left of the decimal point.

An annual peak is then calculated for each year:

$Annual\ Peak_k$ = Large($ArrayPM_{2.5,k}$, 98^{th} $Percentile_k$)

where:
Array $PM_{2.5,k}$ = array of $PM_{2.5}$ values in a given year "k."
Large = Excel function that identifies a peak value specified, in this case, by the 98th $Percentile_k$ value.

[4] Details on the calculation of design values can be found in 40 CFR, Part 50, Appendix N: http://edocket.access.gpo.gov/cfr_2009/julqtr/pdf/40cfr50AppN.pdf.

[5] The $PM_{2.5}$ monitor data were downloaded from the U.S. Environmental Protection Agency's website (http://www.epa.gov/airexplorer/) between November 15 and November 23, 2010 by John Graham of the Clean Air Task Force, who then added the data to an Excel spreadsheet. As discussed in the text, he combined some monitor data, so as to provide as complete a monitor record as possible, and then provided the Excel files to Don McCubbin for use in this analysis.

The daily design value is then calculated as an average of the available annual peak values:

$$Daily\ Design\ Value_m = \sum_{k=1}^{l} \frac{Annual\ Peak_k}{l}$$

where:

l = number of annual peak values for a given monitor "m."

2.2 Rolling Back Monitoring Data to Meet Alternative Air Quality Standards

For each monitor, the anthropogenic fraction of each daily $PM_{2.5}$ value was rolled back by the percentage reductions needed to attain various combinations of annual and daily standards (see Table 2). Following EPA methods in their 2010 Risk Assessment (EPA 2010a), the anthropogenic fraction is simply the daily value minus the "policy relevant background" or PRB; PRB has regional values as determined by air quality modeling[6]. The daily values were reduced until the annual standard was met. After this initial reduction, if a daily standard was not yet met, then a second round of reductions was performed to meet the daily standard.

The reduction needed to meet the annual standard was calculated as follows:

annualRollbackFraction = (baselineAnnualDV - annualStandard) / (baselineAnthropogenicAnnualDV)

where:

baselineAnnualDV = annual design value calculated from the unadjusted, or baseline, $PM_{2.5}$ monitor values.

annualStandard = annual ambient air quality standard.

baselineAnthropogenicAnnualDV = annual design value for anthropogenic $PM_{2.5}$ values, calculated by first subtracting the PRB from each day, and then calculating an annual design value the same way as described in the previous section.

For each day above the PRB, the anthropogenic fraction of the daily value was reduced as follows:

*adjustedAnthroDay = baselineAnthroDay * (1-annualRollbackFraction)*

where:

baselineAnthroDay = daily $PM_{2.5}$ value minus the PRB.

The PRB was then added back to get an estimate of the daily value after meeting the annual standard.

rollbackDay = adjustedAnthroDay + PRB.

After this initial reduction, the daily design value ("estimatedDailyDV") was calculated and compared to the daily standard. If the estimatedDailyDV exceeded the daily standard, then it was reduced as follows:

dailyRollbackFraction = (estimatedDailyDV -dailyStandard) / (estimatedDailyDV - PRB)

[6] More details are available in Section 3.7 of the Integrated Science Assessment for Particulate Matter (U.S. EPA 2009).

where:
estimatedDailyDV = daily design value calculated after meeting the annual standard.

For each day above the PRB, the anthropogenic fraction of the previously reduced daily values was reduced again, and then the PRB was added, as follows:
*finalDailyValue = adjustedAnthroDay * (1-dailyRollbackFraction) + PRB.*

After adjusting the monitor values, a new annual average design value was calculated, which was then used as input to BenMAP. A variation of the above steps was performed for each of the scenarios listed in Table 2. These scenarios include most of the scenarios analyzed by EPA (2010a), as well as a number of additional scenarios so as to augment the available information for consideration in the current NAAQS review.

Table 2. Rollback Scenarios

Name	Annual Design Value ($\mu g/m^3$)	Daily Design Value ($\mu g/m^3$)	Included in EPA (2010a) Risk Assessment*
_A15	15	--	No
_A15D35	15	35	Yes
_A13	13	--	No
_A13D35	**13**	**35**	**Yes**
_A13D30	**13**	**30**	**Yes**
_A13D25	13	25	No
_A12	12	--	No
_A12D35	**12**	**35**	**Yes**
_A12D30	12	30	No
_A12D25	12	25	Yes
_A11	11	--	No
_A11D35	**11**	**35**	**No**
_A11D30	11	30	No
_A11D25	11	25	No

Note that as part of a sensitivity analysis some of the scenarios involve just meeting an annual standard (e.g., A15 meets an annual standard of 15 $\mu g/m^3$). * The EPA (U.S. EPA, 2010a) Risk Assessment includes an analysis of a 14/35 scenario, which is not included here as the Policy Assessment (U.S. EPA, 2011) does not recommend this.

2. 3 Interpolating Monitoring Data to CMAQ 12 km Grid Cells

After loading into BenMAP the monitoring data for the baseline case and for each of the scenarios described in Table 2, the fixed radius option in BenMAP was used to estimate the air quality in each

CMAQ grid cell that is within 60 kilometers of a monitor.[7] The fixed radius option works by calculating an inverse distance-weighted average of the monitoring data within a prescribed distance of the center of each grid cell. This is described in Appendix C of the BenMAP User Manual (Abt Associates Inc., 2010).[8]

There is a tradeoff between choosing a relatively short maximum distance, which may better represent the exposure estimate, and choosing a relatively large maximum distance, which will increase the number of people included in the analysis. As noted in Table 3, limiting the interpolation to 60 kilometers captures 91% of the population (ages 30 and up). No estimate of adverse impacts is made for the population that resides beyond the area with estimated air pollution levels.

Table 3. Population Coverage at Varying Fixed Radius Distances

Fixed Radius Distance	2008 Population Ages 30+ (million)	Percent of No Maximum Distance Population
30 km	135.2	76%
40 km	147.8	83%
50 km	156.5	88%
60 km*	162.5	91%
100 km	173.0	97%
No Maximum	178.0	100%

* Distance used in this report.

[7] The California Air Resources Board (2009) used a similar approach when it interpolated monitor values within 50 kilometers of census tracts in their analysis of alternative $PM_{2.5}$ standards. The EPA (1999) also used a 50 kilometer maximum distance when evaluating the benefits of the Clean Air Act.

[8] An alternative to the fixed radius approach is Voronoi Neighbor Averaging (VNA), which has the option of using a maximum interpolation distance. The VNA approach uses the Voronoi algorithm to identify nearby or "neighbor" monitors, and then BenMAP takes a distance-weighted average of these "neighbor" monitors. A downside is that this approach excludes monitors that are within the specified distance, if there is another monitor between it and the center of the CMAQ grid cell. Nevertheless, both the fixed radius and VNA approaches give broadly similar results, generally within 5-10% based on test results using 2007 monitor data.

3. Estimating Human Health Benefits of Meeting Alternative $PM_{2.5}$ Standards

To estimate $PM_{2.5}$-related human health impacts, this analysis used BenMAP, which the EPA used in its analysis of alternative ambient air quality standards (e.g., U.S. EPA2010a) and air pollution regulations (e.g., U.S. EPA2010b). BenMAP calculates the difference in air quality between two scenarios, designated as "baseline" and "control," in each grid cell for which BenMAP has estimated population exposure to $PM_{2.5}$. Given the difference in exposure between the two scenarios, BenMAP then calculates the associated change in adverse health effects, such as premature mortality, using health impact functions. Lastly, BenMAP assigns an economic value to these effects.

Table 4 presents the baseline and control scenario pairs used in BenMAP to calculate health impacts. The next section details the approach used to estimate adverse health impacts. The section after describes the basic steps involved in calculating and placing an economic value on human health impacts associated with $PM_{2.5}$.

Table 4. Baseline and Control Scenarios Used to Calculate Health Impacts

Results Name	Baseline Scenario	Control Scenario
A15	2007-2009 $PM_{2.5}$	_A15
A15D35	2007-2009 $PM_{2.5}$	_A15D35
A13	2007-2009 $PM_{2.5}$	_A13
A13D35	2007-2009 $PM_{2.5}$	_A13D35
A13D30	2007-2009 $PM_{2.5}$	_A13D30
A13D25	2007-2009 $PM_{2.5}$	_A13D25
A12	2007-2009 $PM_{2.5}$	_A12
A12D35	2007-2009 $PM_{2.5}$	_A12D35
A12D30	2007-2009 $PM_{2.5}$	_A12D30
A12D25	2007-2009 $PM_{2.5}$	_A12D25
A11	2007-2009 $PM_{2.5}$	_A11
A11D35	2007-2009 $PM_{2.5}$	_A11D35
A11D30	2007-2009 $PM_{2.5}$	_A11D30
A11D25	2007-2009 $PM_{2.5}$	_A11D25

Table 2 describes the baseline and control scenarios.

3.1 Estimating Cases of Adverse Health Impacts

Derived from concentration-response functions reported in the epidemiological literature, health impact functions quantify the relationship between changes in air pollution and adverse health impacts. A typical health impact function has four components:

- **Effect estimate**. An effect estimate ("beta") quantifies the change in health effects per unit of change in a pollutant and is derived from an epidemiological study.
- **$PM_{2.5}$ change**. The estimated change in the concentration of ambient $PM_{2.5}$.
- **Incidence rate**. The baseline incidence rate for the health effect due to all causes.
- **Population**. The affected population; the age range included depends on the ages included in the epidemiological study. This analysis used 2008 population estimates, the calculation of which is described in Appendix C.

The typical log-linear health impact function looks as follows:

$$\Delta Health = \left(1 - \frac{1}{\exp(Beta * \Delta PM2.5)}\right) * Incidence * Population$$

Another common form for health impact functions is the logistic, which appears as follows:

$$\Delta Health = \left(1 - \left(\frac{1}{(1 - Incidence) * \exp(Beta * \Delta PM2.5) + Incidence}\right)\right) * Incidence * Population$$

All of the health impact functions used are in one of these two main forms. Both types have the same four elements. Appendix A derives these two forms and presents details on the derivation of each effect estimate, and Appendix B describes the health incidence databases used for the calculation of premature mortality, hospital admissions, emergency room visits, and heart attacks.[9] Table 5 presents the $PM_{2.5}$-related health endpoints included in the analysis, which follows the approach currently under development by EPA for its benefits analyses.

[9] The incidence and prevalence rates for the other health endpoints, such as acute bronchitis, asthma exacerbation, and other effects not requiring hospitalization are provided in the derivation of the health impact functions in Appendix A.

Table 5. Epidemiological Studies Used to Estimate Adverse Health Impacts of $PM_{2.5}$

Endpoint	Author	Age
Mortality, All Cause	Laden *et al.* (2006)	25 - 99
Mortality, All Cause	Pope *et al.* (2002)	30 - 99
Mortality, Various*	Krewski *et al.* (2009)	30 - 99
Mortality, All Cause	Woodruff *et al.* (1997)	Infant
Heart Attack, Nonfatal	Peters *et al.* (2001)	18 - 99
Heart Attack, Nonfatal	Pope *et al.* (2006)	18 - 99
Heart Attack, Nonfatal	Sullivan *et al.* (2005)	18 - 99
Heart Attack, Nonfatal	Zanobetti and Schwartz (2006)	18 - 99
Heart Attack, Nonfatal	Zanobetti *et al.* (2009)	18 - 99
HA, All Cardiovascular (less Myocardial Infarctions)	Bell *et al.* (2008)	65 - 99
HA, All Cardiovascular (less Myocardial Infarctions)	Peng *et al.* (2008)	65 - 99
HA, All Cardiovascular (less Myocardial Infarctions)	Peng *et al.* (2009)	65 - 99
HA, All Cardiovascular (less Myocardial Infarctions)	Zanobetti *et al.* (2009)	65 - 99
HA, All Cardiovascular (less Myocardial Infarctions)	Moolgavkar (2000b)	18 - 64
HA, All Respiratory	Zanobetti *et al.* (2009)	65 - 99
HA, Chronic Lung Disease	Moolgavkar (2000a)	18 - 64
HA, Asthma	Babin *et al.* (2007)	0 - 17
HA, Asthma	Sheppard (2003)	0 - 17
Emergency Room Visits, Asthma	Mar *et al.* (1999; 2010)	0 - 99
Emergency Room Visits, Asthma	Slaughter *et al.* (2005)	0 - 99
Acute Bronchitis	Dockery *et al.* (1996)	8 - 12
Lower Respiratory Symptoms	Schwartz and Neas (2000)	7 - 14
Upper Respiratory Symptoms	Pope *et al.* (1991)	9 - 11
Asthma Exacerbation, Cough	Ostro *et al.* (2001)	6 - 18
Asthma Exacerbation, Cough	Mar *et al.* (2004)	6 - 18
Asthma Exacerbation, Shortness of Breath	Ostro *et al.* (2001)	6 - 18
Asthma Exacerbation, Shortness of Breath	Mar *et al.* (2004)	6 - 18
Asthma Exacerbation, Wheeze	Ostro *et al.* (2001)	6 - 18
Work Loss Days (WLD)	Ostro (1987)	18 - 64
Minor Restricted Activity Days (MRAD)	Ostro and Rothschild (1989)	18 - 64

Note: HA = hospital admissions. * The study by Krewski *et al.* (2009) was used to develop mortality functions for four mortality endpoints: all-cause, ischemic heart disease, cardiopulmonary, and lung cancer.

Sensitivity Analyses

As a sensitivity analysis, a variety of mortality functions were included in the present study. The primary estimate is based on studies by Laden et al. (2006) and Krewski et al. (2009) and assumes that no impacts occurred when the baseline air quality level falls below the lowest measured level (LML) in the underlying epidemiological studies. This assumption may underestimate benefits as no known threshold for health effects of $PM_{2.5}$ has been identified. Additional results show the impact of relaxing this restriction. Table 6 presents the LMLs for the mortality studies used in this analysis.

In addition, two estimates are presented for nonfatal heart attacks. One is based on the study by Peters *et al.* (2001), which EPA (2004; 2010b) has long used to estimate in regulatory impact assessments. A second estimate is based on four studies (Sullivan et al., 2005; Pope et al., 2006; Zanobetti and Schwartz, 2006; Zanobetti et al., 2009), which give an estimate significantly lower than that based on

Peters *et al*. This second estimate, based on a number of more recent studies over a broader geographical area is included in the primary set of results, with the Peters *et al*. result presented as a sensitivity analysis.

Table 6. Lowest Measured Levels for Mortality Epidemiological Studies

Epidemiological Study	LML (µg/m^3)
Pope *et al.*	7.5
Laden *et al.*	10
Krewski *et al.*	5.8

Pooling Health Effect Estimates

When there are several effect estimates for a given health endpoint, these estimates are quantitatively combined or "pooled" to derive a more robust estimate. This analysis generally used fixed or random effects models to pool estimates from different studies of the same health endpoint. Fixed effects pooling simply weights each study's estimate by the inverse variance, giving more weight to studies with lower variance. Random effects pooling accounts for both within-study variance and between-study variability, due, for example, to differences in population susceptibility. The fixed effects model is used as the null hypothesis, with a statistical test of the data determining whether the null should be rejected, in which case the random effects model is used.[10] Additional details on pooling are provided in Appendix A.

3.2 Valuing Cases of Health Effects

To estimate the economic benefit of the estimated change in health incidence, BenMAP multiplies the number of adverse cases of a specific type of effect (e.g., mortality) by its associated unit value and then adjusts for the estimated change in income over time:

$$\$Benefit = Cases\ Health\ Effects * Unit\ Value * Income\ Adjustment$$

A *unit value* gives the estimated economic value of the avoidance of a single case of a particular endpoint — a single death, for example, or a single hospital admission. Unit values are derived from the economics literature, and come in several varieties.

- For some endpoints, such as hospital admissions, *cost of illness* (COI) unit values are used, which estimate the cost of treating or mitigating the effect. These estimates generally underestimate the true value of reductions in risk of a health effect, since they include hospital costs and lost wages, but do not include any estimate of the value of avoided pain and suffering.
- Other endpoints, such as asthma exacerbation, involve *willingness to pay* (WTP) unit values, which estimate willingness to pay for reductions in the risk of a health effect.

[10] This statistical test is described in the BenMAP User Manual (Abt Associates Inc., 2010, p. 404).

- Typically *value of statistical life* (VSL) unit values are used for reductions in risk of premature mortality.

Several issues arise when determining unit values, such as taking into account inflation and adjusting for growth in income. The following section discusses these issues.

Issues in Valuation

An air pollution benefit analysis tries to measure the full value to society of better health. There are different components to this value, including: (1) medical costs, (2) lost productivity when someone cannot go to work, and (3) pain and suffering. Medical costs and lost productivity are relatively easy to value, since markets exist where these goods are bought and sold. However, pain and suffering is more difficult to measure, since there is no market.

Economists have devised alternative approaches to valuing non-market goods. One is an indirect (or "revealed preference") approach, whereby one infers the values people place on non-market "goods" by examining their preferences revealed by their behaviors in associated markets. For a long time seatbelts were not standard equipment in automobiles, and car buyers needed to specifically order and pay for them. A market existed and seatbelts had a specific cost. Seatbelts also reduced the risk of premature mortality, and so economists inferred the value of avoiding premature mortality risk by studying the associated seatbelt market. There are many other similar examples. Perhaps the most common is calculating the value of avoiding risk by comparing jobs with different risk levels and determining how much more people have to be paid to work the riskier job.

Valuing "Statistical Cases" Avoided

Reductions in ambient concentrations of air pollution generally lower the risk of adverse health effects by a small amount for a large population. 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. This reduction in risk for individuals results in a decrease in the expected number of cases of the adverse health effect in the population.

Suppose, for example, that a given reduction in particulate matter (PM) concentrations results in a decrease in mortality risk of 1/10,000. Then for every 10,000 individuals, we expect one fewer death than would have been expected if PM concentrations had not been reduced. Whose lives will be saved cannot be known *ex ante*. We therefore refer to "statistical lives" and, in benefits analysis, the "value of a statistical life." Similarly with other adverse health effects, such as hospitalization for a respiratory illness, it is reductions in risk that are conferred on individuals and "statistical cases" that are avoided by a reduction in pollution concentrations.

In theory, it is the risk reductions resulting from a reduction in pollutant concentrations that should be valued, and some studies have been designed to estimate individuals' willingness to pay (WTP)[11] for small reductions in risk. Wage-risk studies, for example, use data on wages in occupations with varying degrees of mortal risk (*e.g.,* from accidents) to tease out estimates of the average willingness to pay for mortal risk reductions. From this, the "value of a statistical life" can be inferred.

Taking the example above, suppose that a given reduction in PM concentrations results in a decrease in mortality risk of 1/10,000. If the average WTP for this 1/10,000 decrease in mortality risk is $500, then the value of a "statistical life saved" is 10,000 x $500, or $5 million. Note that no one individual is willing to pay $5 million dollars to avoid certain death; instead, 10,000 individuals are each willing to pay $500 to reduce the *risk* of dying by 1/10,000.

Change over Time in WTP in Real Dollars

The WTP for health-related environmental improvements (in real dollars) could change over time if, for example, real income changes. Generally, the more income a person has, the more he is willing to pay for a good or service (although the percent increase in WTP does not necessarily equal the percent increase in income). If average income (in real dollars) has increased since the time that a wage-risk study was conducted, for example, it is reasonable to expect that WTP, in real dollars, would have increased as well. If a WTP estimate is not adjusted for increases (over time) in real income, it will be biased downward as a result. As a result, this analysis includes an adjustment for income change over time, following an approach used in EPA analyses.[12]

Economists estimate "elasticities" to describe by what percent WTP goes up for a given percentage increase in income. As it turns out, these estimated elasticities are much less than one, however, there is considerable uncertainty over the precise value. This report follows the approach used by EPA in recent regulatory analyses (U.S. EPA, 2008), which used elasticity estimates that vary by type of health effect, with relatively minor effects having a smaller elasticity than more severe effects. Multiplying these elasticities by historical and forecasted income data, EPA developed income adjustment factors which are used in this report. Table 7 presents the elasticities and associated income adjustment factors used in this report.

[11] WTP is a measure of the value an individual places on gaining an outcome viewed as desirable, whether it 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 (e.g., avoiding premature death) and having the money.
[12] See for example the regulatory impact analysis of the Proposed Federal Transport Rule (U.S. EPA, 2010b).

Table 7. Elasticity of WTP and Income Adjustment by Type of Health Effect

Health Effect	Central Elasticity Estimate	Income Adjustment for 2008
Minor Health Effect	0.14	1.030
Severe & Chronic Health Effects	0.45	1.098
Premature Mortality	0.4	1.087

Source: EPA (2005, p. 4-18).

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

Adjusting for Inflation

An adjustment was made for inflation, which increases the nominal value – *i.e.*, the price of goods – from one year to the next without a corresponding increase in the actual value of those goods. So, for example, a good that cost $200 in 1996 might cost $250 in 2008. This increase in the nominal price of the good does not imply an increase in the value of the good, if both prices and incomes have increased by the same percentage over that time period.

To get all dollar values in year 2008 dollars, this analysis used standard inflation adjusters. In particular, to adjust WTP estimates, the consumer price index for "all items" (CPI-U "all items") was used. To adjust estimated hospital costs, the consumer price index for medical care and services was used (*see* Bureau of Labor Statistics website, at: http://data.bls.gov/cgi-bin/surveymost). Similarly, a wage index was used to adjust for wage inflation.[13]

Mortality Adjustments

The delay, or lag, between changes in PM exposures and changes in mortality rates is not precisely known. The current scientific literature on adverse health effects, such as those associated with $PM_{2.5}$ (e.g., smoking-related disease) and the difference in the estimated effect of chronic exposure studies versus daily mortality studies, suggests that it is likely that not all cases of avoided premature mortality associated with a given incremental reduction in PM exposure would occur in the same year as the exposure reduction.

EPA analyses (U.S. EPA, 2006, p. 5-21) have used a 20-year lag structure, with 30 percent of premature deaths occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in

[13] Additional details on the inflation adjustment can be found in the BenMAP User Manual (Abt Associates Inc., 2010)

$PM_{2.5}$, and 20 percent occurring evenly over years 6 to 20 after the reduction in $PM_{2.5}$. It should be noted, however, that the selection of a 20-year lag structure is not directly supported by any PM-specific literature. Rather, it is intended to be a reasonable estimate of the appropriate time distribution of avoided cases of PM-related mortality. The distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. This is a conservative approach, as recent work by Schwartz *et al.* (2008) suggests that most deaths occur within the first two years of exposure.

Accounting for the lag is important because people are generally willing to pay more for something now than for the same thing later. They would, for example, be willing to pay more for a reduction in the risk of premature death in the same year as exposure is reduced than for that same risk reduction to be received the following year. This time preference for receiving benefits now, rather than later, is expressed by discounting benefits received later. The exact discount rate that is appropriate (i.e., that represents people's time preference) is a topic of much debate. EPA has typically presented results based on a discount rate of three percent and seven percent; a similar procedure is followed here.

Quantifying the Value of Avoiding Adverse Health Impacts

Table 8 presents the mean estimate of the unit values used in the analysis. The approach used is the same as that used in recent EPA analyses, and details on the values can be found in the BenMAP User Manual (Abt Associates Inc., 2010) and in recent EPA regulatory impact assessments (e.g., EPA2010b).

Table 8. Mean Value for Economic Valuation of Health Endpoints (based on 2008 income and 2008$)

Health Endpoint	Age Range	No Discount Used ($)	3% Discount Rate ($)	7% Discount Rate ($)
Mortality*	0 – 99	--	$7,790,000	$7,010,000
Acute Myocardial Infarction, Nonfatal**	0 – 24	--	$92,000	$91,100
Acute Myocardial Infarction, Nonfatal**	25 – 44	--	$103,000	$101,000
Acute Myocardial Infarction, Nonfatal**	45 – 54	--	$109,000	$106,000
Acute Myocardial Infarction, Nonfatal**	55 – 64	--	$190,000	$179,000
Acute Myocardial Infarction, Nonfatal**	65 – 99	--	$92,000	$91,100
HA, All Cardiovascular (less AMI)	65 – 99	$29,500	--	--
HA, All Cardiovascular (less AMI)	18 – 64	$31,700	--	--
HA, All Respiratory	65 – 99	$25,600	--	--
HA, Chronic Lung Disease	18 – 64	$14,600	--	--
HA, Asthma***	0 – 64	$10,800	--	--
Asthma ER Visits****	0 – 99	$399	--	--
Acute Bronchitis	8 – 12	$458	--	--
Lower Resp. Symptoms	7 – 14	$20	--	--
Upper Resp. Symptoms	9 – 11	$32	--	--
Asthma Exacerbation	6 – 18	$55	--	--
Work Loss Days*****	18 – 64	$146	--	--
Minor Restricted Activity Days	18 – 64	$69	--	--

Note: Numbers rounded to three significant digits. HA = hospital admissions. * Mortality value after adjustment for 20-year lag. **The age-specific acute myocardial infarction unit values are based on an average of two estimates: one based on Russell (1998) and one based on Wittels (1990). *** Asthma hospital admissions valuation based on data for ages 0-64 and applied to the age group 0-17. **** Asthma ER visits based on the average of two studies by Smith *et al.* (1997) and Stanford *et al.* (1999). ***** BenMAP uses county-specific median daily wage; a national average of the county-level results is presented here.

4. Results

BenMAP calculates the health benefits of alternative standards for grid cell in the CMAQ 12 kilometer modeling domain. These results were then aggregated to the county and national level. National-level results are presented here.

Table 9 presents the estimated adverse health impacts avoided nationwide each year by moving from typical $PM_{2.5}$ air quality levels in the period 2007-2009 and attaining a variety of standards. The results for an annual standard of 15 $\mu g/m^3$ and a daily standard of 35 $\mu g/m^3$ are in the column denoted A15D35, and the results for other combinations of annual and daily standards are presented in a similar way in Table 9. Table 10 presents the dollar value of the avoided health impacts for each of the scenarios presented in Table 9.

The mortality estimates in Table 9 and Table 10 assume that no deaths occur below the lowest measured level (LML). This is a relatively conservative assumption. For the Laden *et al.* (2006) estimate, with an LML of 10 $\mu g/m^3$, assuming no effect below the LML reduces the estimated mortality by almost 30 percent for some standards (see Table 11). For Krewski *et al.* (2009), with an LML of 5.8 $\mu g/m^3$, the LML has no effect, while for Pope *et al.* (2002), with a LML of 7.5 $\mu g/m^3$, the effect is very small.

To quantify some of the uncertainty in the estimated results, a 90 percent confidence interval (5^{th} to 95^{th} percentiles) is presented along with the mean estimate. The quantified uncertainty is based on the estimated uncertainty in the underlying epidemiological studies used in the effect estimates and in the economic literature used to value the estimated health impacts. It should be noted that not all sources of uncertainty are quantified. The estimated population exposure uncertainty is unquantified; similarly, the uncertainty associated with the proportional rollback approach used in this analysis is unquantified. In its quantitative assessment of alternative standards, EPA (2010a, p. 4-37) noted that the rollback choice can have a "notable impact" on the results. To provide greater coverage for the variability associated with rollbacks, EPA used three rollback approaches, "proportional," "locally focused," and "hybrid," with the "proportional" approach tending to produce the biggest reduction in city-wide PM2.5 levels, and the "locally focused" approach the smallest.[14] The rollback approach in the present analysis, though also termed a "proportional" rollback, is generally much more conservative than the "proportional" approach used by EPA, as well as the "hybrid" and "locally focused" approaches, and tends to generate relatively small reductions in $PM_{2.5}$, as seen in Table 13. The approach here closely resembles EPA's locally focused approach, except for the spatial averaging. EPA takes a straight average of the PM changes and applies that result to the entire urban area, while the approach in this report uses inverse distance weighting of the PM reductions. As a result, the estimated health benefits in the present analysis will tend to be more conservatively estimated.

[14] This is noted by EPA (U.S. EPA, 2010a, p. 3-17): "In considering the three rollback methods collectively, the proportional and locally focused methods represent approaches more likely to capture "bounding" behavior related to the spatial pattern of future reductions in ambient $PM_{2.5}$ levels. By contrast, the hybrid approach can be interpreted as reflecting a more plausible or representative rollback strategy in principle…"

To help characterize the uncertainty of some of the assumptions made in this analysis, alternative sets of results are presented. Table 10 presents mortality and nonfatal heart attack valuations with three percent and seven percent discount rates, the effect of which is relatively minor. Table 11 presents a range of mortality health impact functions based on Krewski *et al.* (2009), Laden *et al.* (2006) and Pope *et al.* (2002), and it presents the effect of the LML on the estimated mortality estimates. As noted above, the LML has a strong effect on the Laden *et al.* result. Finally, Table 12 presents the pooled estimate of nonfatal heart attacks used in the primary set of health impact results (Table 9) as well as an estimate based on the study by Peters *et al.* (2001), which has been used by EPA in recent regulatory impact analyses (e.g., EPA2010b) to estimate nonfatal heart attacks. The Peters *et al.* estimate is much larger than the pooled estimate; however, the pooled estimate is based on more recent studies from a greater number of areas, so it is used as the primary estimate.

Table 9. Adverse Health Impacts Avoided by Meeting Alternative $PM_{2.5}$ Standards – Mean Number of Cases and 90% Confidence Interval

Effect	A15D35	A13D35	A13D30	A13D25	A12D35	A12D30	A12D25	A11D35	A11D30	A11D25
Mortality, Krewski, LML	2,540	3,700	6,410	16,700	6,380	7,980	16,800	11,200	12,100	17,900
	1,850 – 3,220	2,700 – 4,700	4,680 – 8,130	12200 - 21200	4,650 – 8,100	5,820 – 10,100	12,300 – 21,300	8,200 – 14,300	8,830 – 15,400	13,100 – 22,700
Mortality, Laden, LML	5,240	8,190	13,500	32,700	15,000	17,500	33,000	27,300	27,900	35,700
	2,850 – 7,570	4,450 – 11,900	7,330 – 19,600	17700 - 47300	8,140 – 21,800	9,490 – 25,400	17,900 – 47,800	14,800 – 39,600	15,100 – 40,400	19,400 – 51,800
Mortality, Infants	7	10	16	36	18	21	37	32	33	40
	3 - 10	5 - 15	8 - 24	18 - 54	9 - 27	10 - 31	18 - 55	16 - 48	16 - 49	20 - 61
Heart Attack, Nonfatal	300	460	803	2,180	828	1,020	2,200	1,490	1,590	2,350
	60 - 663	92 – 1,020	163 – 1,780	448 - 4820	166 – 1,830	206 – 2,270	451 – 4,850	299 – 3,300	320 – 3,530	478 – 5,200
Hospital Admis., Cardiovascular	708	1,120	1,960	5,420	2,050	2,510	5,460	3,730	3,950	5,830
	369 – 1,200	582 – 1,900	1,020 – 3,330	2820 - 9210	1,070 – 3,470	1,310 – 4,260	2,840 – 9,280	1,940 – 6,320	2,060 – 6,700	3,040 – 9,910
Hospital Admis., Respiratory	761	1,220	2,120	5,890	2,250	2,750	5,930	4,110	4,360	6,360
	455 – 1,070	726 – 1,720	1,260 – 2,990	3500 - 8280	1,340 – 3,170	1,640 – 3,870	3,530 – 8,350	2,450 – 5,800	2,590 – 6,140	3,780 – 8,950
Emergency Room Visits, Asthma	1,530	2,310	3,960	10,400	3,980	4,930	10,500	7,040	7,510	11,100
	48 - 2980	72 – 4,500	124 – 7,730	324 - 20200	124 – 7,780	154 – 9,630	327 – 20,400	219 – 13,700	234 – 14,700	347 – 21,700
Upper Respiratory Symptoms	51,800	70,000	118,000	279,000	112,000	141,000	281,000	187,000	203,000	298,000
	16,300 – 87,200	22,000 – 118,000	37,100 – 198,000	87900 - 470000	35,100 – 188,000	44,400 – 237,000	88,600 – 473,000	58,900 – 315,000	63,900 – 342,000	93,900 – 502,000
Lower Respiratory Symptoms	66,500	90,300	152,000	358,000	145,000	182,000	361,000	242,000	262,000	383,000
	32,500 – 99,500	44,000 – 135,000	74,000 – 227,000	175000 - 536000	70,300 – 217,000	88,600 – 272,000	176,000 – 540,000	118,000 – 363,000	128,000 – 393,000	187,000 – 573,000
Asthma Exacerbation	244,000	329,000	554,000	1,310,000	525,000	663,000	1,320,000	880,000	955,000	1,400,000
	13,200 – 1,280,000	17,800 – 1,740,000	30,000 – 2,920,000	71100 - 6900000	28,400 – 2,780,000	35,900 – 3,490,000	71,600 – 6,950,000	47,600 – 4,640,000	51,700 – 5,040,000	75,900 – 7370,000
Work Loss Days	431,000	596,000	1,020,000	2,490,**000**	975,000	1,230,000	2,510,000	1,660,000	1,800,000	2,670,000
	376,000 – 486,000	519,000 – 672,000	888,000 – 1,150,000	2170000 - 2810000	850,000 – 1,100,000	1,070,000 – 1,390,000	2,190,000 – 2,830,000	1,450,000 – 1,870,000	1,570,000 – 2,030,000	2,330,000 – 3,010,000
Minor Restricted Activity Days	2,520,000	3,490,000	5,970,000	14,700,000	5,730,000	7,240,000	14,800,000	9,770,000	10,600,000	15,700,000
	2,130,000 – 2,900,000	2,950,000 – 4,030000	5,050,000 – 6,890,000	12400000 - 16900000	4,840,000 – 6,610,000	6,120,000 – 8,350,000	12,500,000 – 17,000,000	8,260,000 – 11,300,000	8,960,000 – 12,200,000	13,300,000 – 18,100,000

Table 10. Value of Adverse Health Impacts Avoided by Meeting Alternative $PM_{2.5}$ Standards – Mean Value and 90% Confidence Interval (million 2008 $)

Effect	A15D35	A13D35	A13D30	A13D25	A12D35	A12D30	A12D25	A11D35	A11D30	A11D25
Mortality, Krewski, LML, 3% DR	$19,800	$28,800	$49,900	$130,000	$49,700	$62,100	$131,000	$87,500	$94,200	$140,000
	($2,970 - $46,400)	($4,330 - $67,700)	($7,490 - $117,000)	($19,500 - $306,000)	($7,460 - $117,000)	($9,330 - $146,000)	($19,700 - $308,000)	($13,100 - $206,000)	($14,100 - $221,000)	($20,900 - $328,000)
Mortality, Krewski, LML, 7% DR	$17,800	$26,000	$44,900	$117,000	$44,800	$56,000	$118,000	$78,800	$84,800	$126,000
	($2,670 - $41,800)	($3,900 - $61,000)	($6,750 - $106,000)	($17,600 - $275,000)	($6,720 - $105,000)	($8,400 - $131,000)	($17,700 - $277,000)	($11,800 - $185,000)	($12,700 - $199,000)	($18,900 - $295,000)
Mortality, Laden, LML, 3% DR	$40,800	$63,800	$105,000	$254,000	$117,000	$136,000	$257,000	$212,000	$217,000	$278,000
	($5,740 - $100,000)	($8,960 - $157,000)	($14,800 - $258,000)	($35,700 - $625,000)	($16,400 - $287,000)	($19,100 - $335,000)	($36,100 - $630,000)	($29,800 - $521,000)	($30,500 - $533,000)	($39,100 - $683,000)
Mortality, Laden, LML, 7% DR	$36,700	$57,500	$94,700	$229,000	$105,000	$123,000	$231,000	$191,000	$196,000	$251,000
	($5,170 - $90,100)	($8,070 - $141,000)	($13,300 - $233,000)	($32,200 - $563,000)	($14,800 - $258,000)	($17,200 - $301,000)	($32,500 - $568,000)	($26,800 - $469,000)	($27,500 - $480,000)	($35,200 - $615,000)
Mortality, Infants	$58	$87	$135	$309	$153	$176	$314	$272	$279	$347
	($8 - $144)	($12 - $217)	($19 - $336)	($42 - $769)	($21 - $380)	($24 - $438)	($43 - $780)	($37 - $677)	($38 - $694)	($47 - $862)
Heart Attack, Nonfatal, 3% DR	$34	$53	$93	$257	$97	$120	$259	$176	$188	$277
	($1 - $98)	($1 - $152)	($2 - $265)	($5 - $725)	($2 - $275)	($2 - $340)	($5 - $730)	($3 - $498)	($3 - $532)	($5 - $783)
Heart Attack, Nonfatal, 7% DR	$33	$52	$91	$249	$94	$116	$251	$171	$182	$269
	($1 - $97)	($1 - $149)	($2 - $261)	($4 - $713)	($2 - $271)	($2 - $334)	($4 - $718)	($3 - $490)	($3 - $523)	($4 - $770)
Hospital Admis., Cardiovascular	$22	$34	$60	$166	$63	$77	$167	$114	$121	$178
	($11 - $37)	($18 - $58)	($31 - $101)	($87 - $280)	($33 - $105)	($40 - $130)	($87 - $282)	($60 - $192)	($63 - $204)	($93 - $301)
Hospital Admis., Respiratory	$17	$27	$47	$131	$50	$61	$132	$92	$97	$141
	($11 - $24)	($17 - $38)	($29 - $66)	($81 - $182)	($31 - $70)	($38 - $85)	($81 - $183)	($56 - $127)	($60 - $134)	($87 - $196)
Emergency Room Visits, Respiratory	$0.6	$0.9	$1.6	$4.1	$1.6	$2.0	$4.2	$2.8	$3.0	$4.4
	($0 - $1.2)	($0 - $1.8)	($0 - $3)	($0.1 - $7.9)	($0 - $3.1)	($0.1 - $3.8)	($0.1 - $8)	($0.1 - $5.4)	($0.1 - $5.8)	($0.1 - $8.5)
Upper Respiratory Symptoms	$1.6	$2.2	$3.7	$8.8	$3.5	$4.5	$8.9	$5.9	$6.4	$9.5
	($0.5 - $3.6)	($0.6 - $4.9)	($1.1 - $8.2)	($2.5 - $19.5)	($1 - $7.8)	($1.3 - $9.8)	($2.5 - $19.6)	($1.7 - $13)	($1.8 - $14.2)	($2.7 - $20.8)

Effect	A15D35	A13D35	A13D30	A13D25	A12D35	A12D30	A12D25	A11D35	A11D30	A11D25
Lower Respiratory Symptoms	$1.3	$1.8	$3.0	$7.2	$2.9	$3.6	$7.2	$4.8	$5.3	$7.7
	($0.5 - $2.5)	($0.7 - $3.3)	($1.2 - $5.6)	($2.8 - $13.2)	($1.1 - $5.3)	($1.4 - $6.7)	($2.9 - $13.3)	($1.9 - $8.9)	($2.1 - $9.7)	($3 - $14.1)
Asthma Exacerbation	$13	$18	$31	$72	$29	$37	$73	$49	$53	$77
	($1 - $71)	($1 - $97)	($2 - $162)	($4 - $384)	($2 - $154)	($2 - $194)	($4 - $387)	($3 - $258)	($3 - $280)	($4 - $410)
Work Loss Days	$69	$96	$163	$400	$156	$198	$403	$266	$289	$428
	($60 - $78)	($83 - $108)	($143 - $184)	($349 - $451)	($136 - $176)	($172 - $223)	($351 - $454)	($232 - $301)	($252 - $326)	($373 - $483)
Minor Restricted Activity Days	$164	$227	$389	$954	$373	$471	$961	$636	$689	$1,020
	($96 - $236)	($133 - $327)	($227 - $560)	($558 - $1,370)	($218 - $537)	($275 - $678)	($562 - $1,380)	($372 - $916)	($403 - $992)	($597 - $1,470)
Total, 3% DR, Krewski	$20,200	$29,400	$50,800	$132,000	$50,600	$63,300	$133,000	$89,100	$95,900	$142,000
	($3,160 - $47,100)	($4,590 - $68,700)	($7,950 - $119,000)	($20,700 - $310,000)	($7,910 - $118,000)	($9880 - $148,000)	($20,800 - $312,000)	($13,900 - $209,000)	($15,000 - $225,000)	($22,200 - $332,000)
Total, 7% DR, Krewski	$18,200	$26,500	$45,900	$119,000	$45,700	$57,100	$120,000	$80,400	$86,600	$128,000
	($2,860 - $42,500)	($4,160 - $62,000)	($7,200 - $107,000)	($18,700 - $279,000)	($7,160 - $107,000)	($8,960 - $134,000)	($18,900 - $281,000)	($12,600 - $188,000)	($13,600 - $203,000)	($20,100 - $300,000)
Total, 3% DR, Laden	$41,200	$64,400	$106,000	$257,000	$118,000	$137,000	$259,000	$214,000	$219,000	$281,000
	($5,920 - $101,000)	($9,220 - $158,000)	($15,200 - $260,000)	($36,900 - $629,000)	($16,800 - $289,000)	($19,700 - $337,000)	($37,200 - $635,000)	($30,600 - $524,000)	($31,300 - $536,000)	($40,300 - $688,000)
Total, 7% DR, Laden	$37,100	$58,000	$95,700	$232,000	$106,000	$124,000	$234,000	$193,000	$197,000	$253,000
	($5,350 - $90,800)	($8,330 - $142,000)	($13,800 - $234,000)	($33,300 - $567,000)	($15,200 - $260,000)	($17,800 - $303,000)	($33,600 - $572,000)	($27,600 - $472,000)	($28,300 - $483,000)	($36,400 - $620,000)

Results rounded to three digits. Results reflect the use of both a 3 and 7 percent discount rate, as recommended by EPA's Guidelines for Preparing Economic Analyses and OMB Circular A-4.

Table 11. Alternative Mortality Estimates, Mean Number of Cases and 90% Confidence Interval

Description	A15	A15D35	A13	A13D35	A13D30	A13D25
Pope, All-Cause, LML 7.5	582	2,530	2,600	3,690	6,330	16,200
	(228 - 932)	(995 – 4,050)	(1,020 – 4,180)	(1,450 – 5,910)	(2,490 – 10,100)	(6,380 – 26,000)
Pope, All-Cause	582	2,540	2,610	3,700	6,410	16,700
	(228 - 932)	(999 – 4,060)	(1,020 – 4,180)	(1,450 – 5,930)	(2,520 – 10,300)	(6,570 – 26,700)
Laden, All-Cause, LML 10	1,450	5,240	6,590	8,190	13,500	32,700
	(787 – 2,110)	(2,850 – 7,570)	(3,570 – 9,580)	(4,450 – 11,900)	(7,330 – 19,600)	(17,700 – 47,300)
Laden, All-Cause	1,490	6,450	6,650	9,430	16,300	42,400
	(806 – 2,160)	(3,510 – 9,330)	(3,600 – 9,670)	(5,110 – 13,700)	(8,840 – 23,600)	(23,000 – 61,400)
Krewski, All-Cause, LML 5.8	582	2,540	2,610	3,700	6,410	16,700
	(424 - 739)	(1,850 – 3,220)	(1,900 – 3,310)	(2,700 – 4,700)	(4,680 – 8,130)	(12,200 – 21,200)
Krewski, All-Cause	582	2,540	2,610	3,700	6,410	16,700
	(424 - 739)	(1,850 – 3,220)	(1,900 – 3,310)	(2,700 – 4,700)	(4,680 – 8,130)	(12,200 – 21,200)
Krewski, Ischemic, LML 5.8	480	1,940	2,030	2,780	4,800	12,200
	(406 - 553)	(1,640 – 2,230)	(1,710 – 2,340)	(2,350 – 3,200)	(4,060 – 5,530)	(10,300 – 14,000)
Krewski, Ischemic	480	1,940	2,030	2,780	4,800	12,200
	(406 - 553)	(1,640 – 2,230)	(1,710 – 2,340)	(2,350 – 3,200)	(4,060 – 5,530)	(10,300 – 14,000)
Krewski, Cardio., LML 5.8	589	2,510	2,560	3,610	6,230	16,000
	(483 - 694)	(2,060 – 2,960)	(2,100 – 3,030)	(2,960 – 4,260)	(5,110 – 7,340)	(13,100 – 18,800)
Krewski, Cardio.	589	2,510	2,560	3,610	6,230	16,000
	(483 - 694)	(2,060 – 2,960)	(2,100 – 3,030)	(2,960 – 4,260)	(5,110 – 7,340)	(13,100 – 18,900)
Krewski, Lung Cancer, LML 5.8	74	330	361	507	878	2,390
	(40 - 109)	(175 - 483)	(190 - 530)	(267 - 743)	(464 – 1,290)	(1,260 – 3,490)
Krewski, Lung Cancer	74	330	361	507	878	2,390
	(40 - 109)	(175 - 483)	(190 - 530)	(267 - 743)	(464 – 1,290)	(1,260 – 3,490)

Description	A12	A12D35	A12D30	A12D25	A11	A11D35	A11D30	A11D25
Pope, All-Cause, LML 7.5	5,590	6,370	7,900	16,400	10,600	11,200	12,000	17,400
	(2,190 – 8,970)	(2,500 – 10,200)	(3,100 – 12,700)	(6,430 – 26,200)	(4,180 – 17,100)	(4,410 – 18,000)	(4,720 – 19,200)	(6,860 – 27,900)
Pope, All-Cause	5,590	6,380	7,980	16,800	10,600	11,200	12,100	17,900
	(2,190 – 8,970)	(2,510 – 10,200)	(3,130 – 12,800)	(6,620 – 26,900)	(4,180 – 17,100)	(4,410 – 18,000)	(4,750 – 19,400)	(7,050 – 28,700)
Laden, All-Cause, LML 10	14,200	15,000	17,500	33,000	26,900	27,300	27,900	35,700
	(7,680 – 20,600)	(8,140 – 21,800)	(9,490 – 25,400)	(17,900 – 47,800)	(14,600 – 39,000)	(14,800 – 39,600)	(15,100 – 40,400)	(19,400 – 51,800)
Laden, All-Cause	14,300	16,300	20,300	42,700	27,100	28,600	30,800	45,500
	(7,720 – 20,700)	(8,810 – 23,600)	(11,000 – 29,400)	(23,200 – 61,800)	(14,700 – 39,300)	(15,500 – 41,500)	(16,700 – 44,600)	(24,700 – 65,800)
Krewski, All-Cause, LML 5.8	5,590	6,380	7,980	16,800	10,600	11,200	12,100	17,900
	(4,080 – 7,100)	(4,650 – 8,100)	(5,820 – 10,100)	(12,300 – 21,300)	(7,770 – 13,500)	(8,200 – 14,300)	(8,830 – 15,400)	(13,100 – 22,700)
Krewski, All-Cause	5,590	6,380	7,980	16,800	10,600	11,200	12,100	17,900
	(4,080 – 7,100)	(4,650 – 8,100)	(5,820 – 10,100)	(12,300 – 21,400)	(7,770 – 13,500)	(8,200 – 14,300)	(8,830 – 15,400)	(13,100 – 22,700)
Krewski, Ischemic, LML 5.8	4,170	4,690	5,840	12,200	7,750	8,120	8,680	12,900
	(3,530 – 4,810)	(3,960 – 5,400)	(4,940 – 6,730)	(10,400 – 14,100)	(6,550 – 8,930)	(6,870 – 9,360)	(7,340 – 10,000)	(10,900 – 14,900)
Krewski, Ischemic	4,170	4,690	5,840	12,300	7,750	8,120	8,680	12,900
	(3,530 – 4,810)	(3,960 – 5,400)	(4,940 – 6,730)	(10,400 – 14,100)	(6,550 - 8,930)	(6,870 – 9,360)	(7,340 – 10,000)	(10,900 – 14,900)
Krewski, Cardio., LML 5.8	5,410	6,160	7,690	16,100	10,200	10,800	11,600	17,100
	(4,430 – 6,380)	(5,050 – 7,260)	(6,300 – 9,060)	(13,200 – 19,000)	(8,360 – 12,000)	(8,820 – 12,700)	(9,480 – 13,600)	(14,000 – 20,100)
Krewski, Cardio.	5,410	6,160	7,690	16,100	10,200	10,800	11,600	17,100
	(4,430 – 6,380)	(5,050 – 7,260)	(6,300 – 9,060)	(13,200 – 19,000)	(8,360 – 12,000)	(8,820 – 12,700)	(9,480 – 13,600)	(14,000 – 20,200)
Krewski, Lung Cancer, LML 5.8	804	911	1,130	2,410	1,560	1,640	1,760	2,580
	(424 – 1,180)	(480 – 1,340)	(595 – 1,650)	(1,270 – 3,520)	(825 – 2,290)	(867 – 2,410)	(929 – 2,580)	(1,370 – 3,780)
Krewski, Lung Cancer	804	911	1,130	2,410	1,560	1,640	1,760	2,590
	(424 – 1,180)	(480 – 1,340)	(595 – 1,650)	(1,270 – 3,520)	(825 – 2,290)	(867 – 2,410)	(929 – 2,580)	(1,370 – 3,790)

Table 12. Alternative Nonfatal Heart Attack Estimate, Mean Number of Cases and 90% Confidence Interval

Author	A15	A15D35	A13	A13D35	A13D30	A13D25	A12	A12D35	A12D30	A12D25	A11	A11D35	A11D30	A11D25
Pooled Estimate	69	300	333	460	803	2,180	738	828	1,020	2,200	1,430	1,490	1,590	2,350
	(14 - 153)	(60 - 663)	(67 - 738)	(92 - 1,020)	(163 - 1,780)	(448 – 4,820)	(148 – 1,630)	(166 – 1,830)	(206 – 2,270)	(451 – 4,850)	(286 – 3,160)	(299 – 3,300)	(320 – 3,530)	(478 – 5,200)
Peters *et al.* (2001)	543	2,330	2,630	3,600	6,280	17,000	5,820	6,510	8,020	17,200	11,200	11,700	12,500	18,300
	(202 - 874)	(876 - 3,720)	(977 - 4,240)	(1,350 - 5,790)	(2,350 – 10,100)	(6,390 – 27,200)	(2,160 – 9,370)	(2,420 – 10,500)	(3,000 – 12,900)	(64,30 – 27,400)	(4,170 – 18,000)	(4,370 – 18,800)	(4,670 – 20,000)	(6,880 – 29,300)

Results rounded to three digits.

Table 13. PM2.5 Levels by City, Comparison of Present Analysis & EPA (2010a)

City	Source of Rollback Results	Baseline (ug/m^3)	Baseline Minus $PM_{2.5}$ Level in Scenario (ug/m^3)				
			A15D35	A13D35	A12D35	A13D30	A12D25
Atlanta	Present Analysis	13.3	0.0	0.4	1.3	0.4	1.7
	EPA - Proportional	15.3	1.1	3.0	3.9	3.0	4.1
Baltimore	Present Analysis	12.3	0.0	0.1	0.5	0.4	2.0
	EPA - Proportional	13.9	0.8	2.3	3.2	2.6	4.4
	EPA - Hybrid		0.9	2.1	3.0	2.7	4.5
	EPA - Locally Focused		0.3				3.9
Birmingham	Present Analysis	13.1	0.0	0.5	1.1	0.6	2.1
	EPA - Proportional	15.7	3.0	4.7	5.5	4.7	6.3
	EPA - Hybrid		1.5	3.4	4.3	3.4	5.0
	EPA - Locally Focused						4.3
Dallas	Present Analysis	10.7	0.0	0.0	0.0	0.0	0.2
	EPA - Proportional	11.4	0.0	0.0	0.7	0.0	0.7
Detroit	Present Analysis	12.1	0.0	0.1	0.3	0.6	2.3
	EPA - Proportional	13.9	2.5	3.3	4.1	4.0	5.6
	EPA - Hybrid		2.2	2.4	3.3	3.8	5.4
	EPA - Locally Focused		1.4			2.9	4.7
Fresno	Present Analysis	16.4	5.8	5.8	5.8	7.3	8.7
	EPA - Proportional	17.4	7.5	7.5	7.5	8.8	10.1
	EPA - Locally Focused		7.1	7.1	7.1	8.5	9.9
Houston	Present Analysis	12.6	0.0	0.4	0.9	0.4	1.0
	EPA - Proportional	13.2	0.7	2.3	3.1	2.3	3.1
Los Angeles	Present Analysis	14.0	1.3	1.5	2.2	2.8	4.4
	EPA - Proportional	14.6	5.1	5.1	5.6	6.4	7.6
	EPA - Hybrid		4.1	4.3	5.1	5.5	6.9
	EPA - Locally Focused		2.5	2.5		4.0	5.5
New York	Present Analysis	12.1	0.1	0.3	0.6	0.6	2.3
	EPA - Proportional	13.8	2.2	2.5	3.4	3.8	5.4
	EPA - Hybrid		2.0	2.5	3.4	3.6	5.3
	EPA - Locally Focused		0.5			2.2	4.0
Philadelphia	Present Analysis	12.4	0.0	0.1	0.6	0.4	2.1
	EPA - Proportional	13.4	1.1	1.8	2.7	2.7	4.4
	EPA - Locally Focused		0.4			2.1	3.9
Phoenix	Present Analysis	9.5	0.1	0.2	0.2	0.2	0.6
	EPA - Proportional	9.9	0.0	0.0	0.5	0.6	2.1
	EPA - Locally Focused					0.2	0.9
Pittsburgh	Present Analysis	13.4	0.6	0.9	1.6	1.5	3.3
	EPA - Proportional	14.9	3.3	3.7	4.4	4.9	6.5
	EPA - Locally Focused		1.7	3.1	3.7	3.5	5.3

City	Source of Rollback Results	Baseline (ug/m^3)	Baseline Minus $PM_{2.5}$ Level in Scenario (ug/m^3)				
			A15D35	A13D35	A12D35	A13D30	A12D25
Salt Lake City	Present Analysis	10.4	1.9	1.9	1.9	2.8	3.8
	EPA - Proportional	11.4	3.9	3.9	3.9	4.8	5.8
	EPA - Locally Focused		1.7	1.7	1.7	2.6	3.7
St. Louis	Present Analysis	12.8	0.0	0.1	0.8	0.2	1.5
	EPA - Proportional	14.3	1.4	3.0	3.9	3.2	5.0
	EPA - Hybrid		0.8	2.6	3.5	2.6	4.4
	EPA - Locally Focused		0.2			1.9	3.9
Tacoma	Present Analysis	9.2	1.3	1.3	1.3	2.0	2.7
	EPA - Proportional	9.7	1.7	1.7	1.7	2.7	3.7
	EPA - Locally Focused		1.7	1.7	1.7	2.7	3.7

Note: The EPA estimates are from Table F-50 of EPA (2010a), which presents results based on 2007 monitoring data. The results for the present analysis are a population-weighted (ages 30+) estimate of $PM_{2.5}$ based on data for 2007-2009.

Appendix A. Human Health Impact Function Details

This appendix presents the derivation of the two main health impact functions used in this analysis (log-linear and logistic), as well as details on each function used.

A.1 Deriving Health Impact Functions

Presented below is a derivation of the mean coefficient estimates for log-linear and logistic health impact functions.

Log-Linear Derivation

$y_0 = Incidence\ under\ baseline\ conditions$

$y_c = Incidence\ under\ control\ conditions$

$\Delta y = y_0 - y_c$

$PM_0 = PM\ levels\ under\ baseline\ conditions$

$PM_c = PM\ levels\ under\ control\ conditions$

$\Delta PM = PM_0 - PM_c$

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

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

$$y_0 = Be^{\beta PM_0}$$

$$y_c = Be^{\beta PM_c}$$

$$\Delta y = Be^{\beta PM_0} - Be^{\beta PM_c}$$

$$\Delta y = Be^{\beta PM_0} \cdot \left(1 - \frac{Be^{\beta PM_c}}{Be^{\beta PM_0}}\right)$$

$$\Delta y = Be^{\beta PM_0} \cdot \left(1 - e^{\beta \cdot \left(PM_c - PM_0\right)}\right)$$

$$\Delta y = Be^{\beta PM_0} \cdot \left(1 - e^{-\beta \Delta PM}\right)$$

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

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

Logistic Derivation

$y_0 = Incidence\ under\ baseline\ conditions$

$y_c = Incidence\ under\ control\ conditions$

$\Delta y = y_0 - y_c$

$PM_0 = PM\ levels\ under\ baseline\ conditions$

$PM_c = PM\ levels\ under\ control\ conditions$

$\Delta PM = PM_0 - PM_c$

$X = vector\ of\ explanatory\ variables$

$\mathrm{B} = vector\ of\ coefficients$

$\beta = coefficient\ of\ the\ PM\ variable$

$$y = \left(\frac{e^{X\mathrm{B}}}{1 + e^{X\mathrm{B}}}\right) = \frac{1}{1 + e^{-X\mathrm{B}}}$$

$$odds = \frac{y}{1 - y} = \frac{\left(\frac{1}{1 + e^{-X\mathrm{B}}}\right)}{1 - \left(\frac{1}{1 + e^{-X\mathrm{B}}}\right)}$$

$$odds = \frac{\left(\frac{1}{1 + e^{-X\mathrm{B}}}\right)}{\left(\frac{e^{-X\mathrm{B}}}{1 + e^{-X\mathrm{B}}}\right)} = \frac{1}{e^{-X\mathrm{B}}} = e^{X\mathrm{B}}$$

$$odds\ ratio = \frac{e^{X_0\mathrm{B}}}{e^{X_c\mathrm{B}}} = \frac{e^{\gamma} \cdot e^{PM_0\beta}}{e^{\gamma} \cdot e^{PM_c\beta}} = e^{\Delta PM\beta}$$

$$\frac{\left(\frac{y_0}{1-y_0}\right)}{\left(\frac{y_c}{1-y_c}\right)} = e^{\Delta PM\beta}$$

$$\left(\frac{y_0}{1-y_0}\right)\cdot e^{-\Delta PM\beta} = \frac{y_c}{1-y_c}$$

$$y_c = (1-y_c)\cdot\left(\frac{y_0}{1-y_0}\right)\cdot e^{-\Delta PM\beta}$$

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

$$y_c\cdot\left[1+\left(\frac{y_0}{1-y_0}\right)\cdot e^{-\Delta PM\beta}\right] = \left(\frac{y_0}{1-y_0}\right)\cdot e^{-\Delta PM\beta}$$

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

$$y_c = \frac{y_0\cdot e^{-\Delta PM\beta}}{1-y_0+y_0\cdot e^{-\Delta PM\beta}}$$

$$y_c = \frac{y_0}{(1-y_0)\cdot e^{\Delta PM\beta}+y_0}$$

$$y_0 - y_c = y_0 - \frac{y_0}{(1-y_0)\cdot e^{\Delta PM\beta}+y_0}$$

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

A.2 $PM_{2.5}$ Human Health Impact Functions

This analysis uses a range of health impact functions, including those used to estimate premature mortality, chronic bronchitis, and hospital admissions. These health impact functions are the same ones used in recent EPA analyses (e.g., U.S. EPA2010b). Presented below is a table with the health impact functions used to estimate $PM_{2.5}$-related adverse health effects. Following the table is a brief summary of each of the studies along with details not in the summary table.

Some additional details should be noted regarding the calculation of heart attacks and cardiovascular hospital admissions. 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). As a result, this analysis assumes that 93 percent of heart attacks are not fatal and that nonfatal heart attacks result in a hospitalization. In addition, the studies used to estimate hospital admissions for all cardiovascular causes reported results that include ICD code 410 (heart attack). Since this analysis estimates avoided nonfatal heart attacks separately, ICD code 410 was excluded from the baseline incidence rate used in the calculation of hospital admissions for all cardiovascular causes, so as to avoid double counting heart attack hospitalizations.

Table 14. Details of $PM_{2.5}$ Human Health Impact Functions

Endpoint Name	Study	Location	Age	Beta	Std Error	Functional Form
Adult mortality, All-Cause	Laden *et al.* (2006)	6 cities	25-99	0.01484	0.00417	Log-linear
Adult mortality, All-Cause	Pope *et al.* (2002)	51 cities	30-99	0.00583	0.00216	Log-linear
Adult mortality, All-Cause	Krewski *et al.* (2009)	116 cities	30-99	0.00583	0.00096	Log-linear
Adult mortality, Cardiopulmonary	Krewski *et al.* (2009)	116 cities	30-99	0.01222	0.00135	Log-linear
Adult mortality, Ischemic Heart Disease	Krewski *et al.* (2009)	116 cities	30-99	0.02151	0.00206	Log-linear
Adult mortality, Lung Cancer	Krewski *et al.* (2009)	116 cities	30-99	0.01310	0.00379	Log-linear
Infant mortality, All-Cause	Woodruff *et al.* (1997)	86 cities	0-0	0.00392	0.00122	Logistic
Heart Attack, Nonfatal	Peters *et al.* (2001)	Boston, MA	18-99	0.02412	0.00929	Logistic
Heart Attack, Nonfatal	Pope *et al.* (2006)	Wasatch Front, UT	18-99	0.00481	0.00199	Logistic
Heart Attack, Nonfatal	Sullivan *et al.* (2005)	King County, WA	18-99	0.00198	0.00224	Logistic
Heart Attack, Nonfatal	Zanobetti *et al.* (2009)	26 cities	18-99	0.00225	0.00059	Log-linear
Heart Attack, Nonfatal	Zanobetti and Schwartz (2006)	Boston, MA	18-99	0.00530	0.00221	Logistic
HA, All Cardiovascular (less AMI)	Bell *et al.* (2008)	202 counties	65-99	0.00080	0.00011	Log-linear
HA, All Cardiovascular (less AMI)	Peng *et al.* (2008)	108 counties	65-99	0.00071	0.00013	Log-linear
HA, All Cardiovascular (less AMI)	Peng *et al.* (2009)	119 counties	65-99	0.00068	0.00021	Log-linear
HA, All Cardiovascular (less AMI)	Zanobetti *et al.* (2009)	26 cities	65-99	0.00189	0.00028	Log-linear
HA, All Cardiovascular (less AMI)	Moolgavkar (2000b)	Los Angeles, CA	18-64	0.00140	0.00034	Log-linear
HA, All Respiratory	Zanobetti *et al.* (2009)	26 cities	65-99	0.00207	0.00045	Log-linear
HA, Chronic Lung Disease	Moolgavkar (2000a)	Los Angeles, CA	18-64	0.002200	0.00073	Log-linear
HA, Asthma	Babin *et al.* (2007)	Washington, DC	0-17	0.00200	0.00430	Log-linear
HA, Asthma	Sheppard (2003)	Seattle, WA	0-17	0.003324	0.00105	Log-linear
Emergency Room Visits, Asthma	Mar *et al.* (1999; 2010)	Tacoma, WA	0-99	0.005603	0.00210	Log-linear
Emergency Room Visits, Asthma	Slaughter *et al.* (2005)	Spokane, WA	0-99	0.002956	0.00271	Log-linear
Acute Bronchitis	Dockery *et al.* (1996)	24 communities	8-12	0.027212	0.017096	Logistic
Lower Respiratory Symptoms	Schwartz and Neas (2000)	6 U.S. cities	7-14	0.019012	0.006005	Logistic
Upper Respiratory Symptoms	Pope *et al.* (1991)	Utah Valley	9-11	0.0036	0.0015	Logistic
Asthma Exacerbation, Cough	Ostro *et al.* (2001)	Los Angeles, CA	6-18	0.000985	0.000747	Logistic
Asthma Exacerbation, Cough	Mar *et al.* (2004)	Spokane, WA	6-18	0.01906	0.00983	Logistic

Endpoint Name	Study	Location	Age	Beta	Std Error	Functional Form
Asthma Exacerbation, Shortness of Breath	Ostro *et al.*	Los Angeles, CA	6-18	0.002565	0.001335	Logistic
Asthma Exacerbation, Shortness of Breath	Mar *et al.* (2004)	Spokane, WA	6-18	0.01222	0.01385	Logistic
Asthma Exacerbation, Wheeze	Ostro *et al.*	Los Angeles, CA	6-18	0.00194	0.00080	Logistic
Work Loss Days (WLD)	Ostro (1987)	Nationwide	18-64	0.00460	0.00036	Log-linear
Minor Restricted Activity Days (MRAD)	Ostro & Rothschild (1989)	Nationwide	18-64	0.00741	0.00070	Log-linear

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

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

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

Functional Form: Log-linear
Coefficient: 0.01484
Standard Error: 0.00417
Incidence Rate: county-specific annual all-cause mortality rate per person ages 30 and older
Population: population of ages 25 and older.

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

The Pope *et al.* (2002) analysis is a longitudinal cohort tracking study that uses the same American Cancer Society cohort as the original Pope *et al.* (1995) study, and the Krewski *et al.* (2000) reanalysis. Pope *et al.* (2002) analyzed survival data for the cohort from 1982 through 1998, 9 years longer than the original Pope study. Pope *et al.* (2002) 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.[15] 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.

The coefficient and standard error for $PM_{2.5}$ using the average of '79-'83 and '99-'00 PM data are estimated from the relative risk (1.06) and 95% confidence interval (1.02-1.11) associated with a change in annual mean exposure of 10 $\mu g/m^3$ Pope *et al.* (2002, Table 2).

Functional Form: Log-linear
Coefficient: 0.005827
Standard Error: 0.002157
Incidence Rate: county-specific annual all-cause mortality rate per person ages 30 and older
Population: population of ages 30 and older.

[15] 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.

Mortality -- All Cause, Cardiopulmonary, Ischemic Heart Disease, and Lung Cancer (Krewski et al., 2009)

Krewski *et al.* (2009) analysis is a longitudinal cohort tracking study that uses an updated version of the same American Cancer Society cohort as the original Pope *et al.* (1995) study, and the Krewski *et al.* (2000) reanalysis. Krewski *et al.* (2009) analyzed survival data for the cohort from 1982 through 2000, 11 years longer than the original Pope study. Krewski *et al.* (2002) reported significant impacts for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and ischemic heart disease (ICD-9 codes: 410-414).

Krewski *et al.* (2009) present results for two exposure estimates. One based on PM2.5 levels in 1979-1983 and the other for PM2.5 levels in 1999-2000. The effect estimates below for are based on the 1999-2000 exposure estimate, as the later exposure period has better monitor coverage with participants from 116 cities versus 58 cities for the earlier exposure period. In addition, to the extent that the number of deaths is increasing as the cohort ages, then the later exposure period will be more representative of the majority of the deaths that are occurring.

All-Cause Mortality

The coefficient and standard error for $PM_{2.5}$ (using the'99-'00 PM data and the Random Effects Cox Model) are estimated from the relative risk (1.06) and 95% confidence interval (1.04-1.08) associated with a change in annual mean exposure of 10 μg/m^3 Krewski *et al.* (2009, Commentary Table 4).

Functional Form: Log-linear
Coefficient: 0.00583
Standard Error: 0.00096
Incidence Rate: county-specific annual all-cause mortality rate per person ages 30 and older
Population: population of ages 30 and older.

Cardiopulmonary Mortality

The coefficient and standard error for $PM_{2.5}$ (using the'99-'00 PM data and the Random Effects Cox Model) are estimated from the relative risk (1.13) and 95% confidence interval (1.10-1.16) associated with a change in annual mean exposure of 10 μg/m^3 Krewski *et al.* (2009, Commentary Table 4).

Functional Form: Log-linear
Coefficient: 0.01222
Standard Error: 0.00135
Incidence Rate: county-specific annual cardiopulmonary mortality rate per person ages 30 and older
Population: population of ages 30 and older.

Ischemic Heart Disease Mortality

The coefficient and standard error for $PM_{2.5}$ (using the'99-'00 PM data and the Random Effects Cox Model) are estimated from the relative risk (1.24) and 95% confidence interval (1.19-1.29) associated with a change in annual mean exposure of 10 μg/m^3 Krewski *et al.* (2009, Commentary Table 4).

Functional Form: Log-linear

Coefficient: 0.02151
Standard Error: 0.00206
Incidence Rate: county-specific annual ischemic heart disease mortality rate per person ages 30 and older
Population: population of ages 30 and older.

Lung Cancer Mortality

The coefficient and standard error for $PM_{2.5}$ (using the'99-'00 PM data and the Random Effects Cox Model) are estimated from the relative risk (1.14) and 95% confidence interval (1.06-1.23) associated with a change in annual mean exposure of 10 µg/m^3 Krewski *et al.* (2009, Commentary Table 4).

Functional Form: Log-linear
Coefficient: 0.01310
Standard Error: 0.00379
Incidence Rate: county-specific annual lung cancer mortality rate per person ages 30 and older
Population: population of ages 30 and older.

Infant Mortality (Woodruff et al., 1997)

Woodruff *et al.* (1997) examined the relationship between post-neonatal all-cause mortality and exposure to $PM_{2.5}$ in infants born In a study of four million infants in 86 U.S. metropolitan areas conducted from1989 to 1991. Woodruff *et al.* (1997) found a significant link between PM_{10} exposure in the first two months of an infant's life with the probability of dying between the ages of 28 days and 364 days. PM10 exposure was significant for all-cause mortality. PM10 was also significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants.

The coefficient and standard error are based on the odds ratio (1.04) and the 95% confidence interval (1.02-1.07) associated with a 10 µg/m3 change in PM_{10} (Woodruff et al., 1997, Table 3).

Functional Form: Logistic
Coefficient: 0.003922
Standard Error: 0.001221
Incidence Rate: county-specific annual post-neonatal[16] infant deaths per infant under the age of one
Population: population of infants under one year old.

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

Peters *et al.* (2001) studied the relationship between increased particulate air pollution and onset of heart attacks in the Boston area from 1995 to 1996. The authors used air quality data for PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$, "black carbon", O_3, CO, NO_2, and SO_2 in a case-crossover analysis. For each subject, the case period was matched to three control periods, each 24 hours apart. In univariate analyses, the authors observed a positive association between heart attack occurrence and $PM_{2.5}$ levels hours before and days before onset. The authors estimated multivariate conditional logistic models including two-hour and

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

twenty-four hour pollutant concentrations for each pollutant. They found significant and independent associations between heart attack occurrence and both two-hour and twenty-four hour $PM_{2.5}$ concentrations before onset. Significant associations were observed for PM_{10} as well. None of the other particle measures or gaseous pollutants was significantly associated with acute myocardial infarction for the two hour or twenty-four hour period before onset.

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, this analysis assumes an age range of 18 and over in the C-R function. According to the National Hospital Discharge Survey, there were no hospitalizations for heart attacks among children <15 years of age in 1999 and only 5.5% of all hospitalizations occurred in 15-44 year olds (Popovic, 2001, Table 10).

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

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

Heart Attacks (Acute Myocardial Infarction), Nonfatal (Pope et al., 2006)

Pope *et al.* (2006) evaluated the association between short-term exposure to $PM_{2.5}$ and acute ischemic heart disease events, including acute nonfatal myocardial infarction, all acute coronary events, and subsequent myocardial infarctions in individuals living in greater Salt Lake City, Utah. In a case-crossover study, these ischemic events were assessed in relation to a 10 ug/m^3 increase in $PM_{2.5}$.

The coefficient and standard error are calculated from a 4.81% increase in index myocardial infarction and unstable angina (95% CI 0.98%-8.79%) for a 10 $\mu g/m^3$ increase in twenty-four hour average $PM_{2.5}$ (Pope et al., 2006, Table 3).

Functional Form: Logistic
Coefficient: 0.00481
Standard Error: 0.00199
Incidence Rate: region-specific daily nonfatal heart attack rate per person 18+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410)
Population: population of ages 18 and older.[17]

[17] The study included people of all ages. To be comparable with the age groups used with Peters et al (2001) and because the rate of heart attack in the age group 0-17 is very low, effects were calculated for ages 18 and older.

Heart Attacks (Acute Myocardial Infarction), Nonfatal (Sullivan et al., 2005)

Sullivan *et al.* (2005) studied the relationship between onset time of acute myocardial infarction and the preceding hourly $PM_{2.5}$ concentrations in 5,793 confirmed cased of myocardial infarction through King County, Washington. In this case-crossover study from 1988-1994, air pollution exposure levels averaged 1 hour, 2 hours, 4 hours, and 24 hours before onset of myocardial infarction were compared to a set of time-stratified referent exposures from the same day of the week in the month of the case event. The authors reported a relatively weak association between myocardial infarction onset and exposure to $PM_{2.5}$.

The coefficient and standard error are calculated from an odds ratio of 1.02 (95% CI 0.98-1.07) for a 10 $\mu g/m^3$ increase in twenty-four hour average $PM_{2.5}$ (Sullivan et al., 2005, Table 3).

Functional Form: Logistic
Coefficient: 0.00198
Standard Error: 0.00224
Incidence Rate: region-specific daily nonfatal heart attack rate per person 18+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410)
Population: population of ages 18 and older. [18]

Heart Attacks (Acute Myocardial Infarction), Nonfatal (Zanobetti et al., 2009)

Zanobetti *et al.* (2009) examined the relationship between daily $PM_{2.5}$ levels and emergency hospital admissions for cardiovascular causes, myocardial infarction, congestive heart failure, respiratory disease and diabetes among people ages 65 and older in 26 U.S. communities from 2000-2003. The authors used meta-regression to examine how this association was modified by season- and community-specific $PM_{2.5}$ composition while controlling for seasonal temperature as a substitute for ventilation. The authors found that $PM_{2.5}$ mass higher in a variety of elements, including arsenic and organic carbon, significantly increased its effects on hospital admissions.

The coefficient and standard error are calculated from a 2.25% increase in hospital admissions for myocardial infarction (95% CI 1.10%-3.42%) for a 10 $\mu g/m^3$ increase in two-day averaged $PM_{2.5}$ (Zanobetti et al., 2009, Table 3).

Functional Form: Log-linear
Coefficient: 0.00225
Standard Error: 0.00059
Incidence Rate: region-specific daily nonfatal heart attack rate per person 65+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410)
Population: population of ages 65 and older.

[18] The study included people of all ages. To be comparable with the age groups used with Peters et al (2001) and because the rate of heart attack in the age group 0-17 is very low, effects were calculated for ages 18 and older.

Heart Attacks (Acute Myocardial Infarction), Nonfatal (Zanobetti and Schwartz, 2006)

Zanobetti and Schwartz (2006) analyzed hospital admissions through emergency department for myocardial infarction (ICD-9 code 410) and pneumonia (ICD-9 codes 480-487) for associations with fine particulate air pollution, ozone, black carbon, nitrogen dioxide (NO_2), PM not from traffic, and CO in the greater Boston area from 1995-1999. The authors used a case-crossover analysis with control days matched on temperature. Significant associations were detected for NO_2, $PM_{2.5}$, and black carbon in emergency myocardial infarction hospitalizations. Significant associations were also identified for $PM_{2.5}$ in pneumonia hospitalizations.

The coefficient and standard error are calculated from a 8.65% increase in hospital admissions for myocardial infarction (95% CI 1.22%-15.38%) for a 16.32 $\mu g/m^3$ increase in daily $PM_{2.5}$ (Zanobetti and Schwartz, 2006, Table 4).

Functional Form: Log-linear
Coefficient: 0.00530
Standard Error: 0.00221
Incidence Rate: region-specific daily nonfatal heart attack rate per person 65+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410)
Population: population of ages 65 and older.

Pooling Estimates of Heart Attacks (Acute Myocardial Infarction), Nonfatal

As the valuation of a nonfatal heart varies by age, he heart attack results were calculated for five age groups: 18-24, 25-4, 45-54, 55-64, and 65+. For the first four age groups, the studies by Pope *et al.* (2006) and Sullivan *et al.* (2005) were combined with a random/fixed effects approach for each age group. For the 65 and older age group, the Zanobetti *et al.* (2009) and Zanobetti and Schwartz (2006) studies, which are based on just the elderly population, were included in the random/fixed effects pooling. Once each of the age groups were pooled separately, they were added together to get an overall estimate of nonfatal heart attacks.

Hospital Admissions for All Cardiovascular Causes (less AMI) (Bell et al., 2008)

Bell *et al.* (2008) evaluated the association between short-term exposure to $PM_{2.5}$ and the risk of cardiovascular (ICD-9 codes 410-414, 26-427, 428, 429, 430-438, and 440-449) and respiratory (ICD-9 codes 464-466, 480-487, and 490-492) hospital admissions. The target population was Medicare enrollees ≥65 years old in 202 U.S. counties with populations greater than 200,000 from 1999-2005. Three time-series models were used to provide three key variables: consistent PM effects across the year, different PM effects by season, and smoothly varying PM effects throughout the year. A two-stage Bayesian hierarchical model was used to estimate the association between $PM_{2.5}$ and hospitalization rates, with the first stage estimating the association within a single county and the second stage combining county-specific estimates.

The coefficient and standard error are calculated from a 0.80% increase in hospital admissions for cardiovascular-related problems (95% CI 0.59%-1.01%) for a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ (Bell et al., 2008, Table 1).

Functional Form: Log-linear
Coefficient: 0.00080
Standard Error: 0.00011
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions (less acute myocardial infarction) per person ages 65 and older (ICD codes 390-409, 411-429)
Population: population of ages 65 and older.

Hospital Admissions for All Cardiovascular Causes (less AMI) (Peng et al., 2008)

Peng *et al.* (2008) examined the risk of hospital admissions for cardiovascular and respiratory diseases in relation to particulate matter ($PM_{10-2.5}$ and $PM_{2.5}$). To accomplish this, the authors utilized a database of 108 U.S. counties with daily emergency hospital admission rates for cardiovascular and respiratory diseases among Medicare enrollees living 9 miles from air monitors, temperature, and dew-point temperature. $PM_{10-2.5}$ and $PM_{2.5}$ concentrations were calculated by using monitoring data from January 1, 1999 through December 31, 2005. Overall, there were 3.7 million cardiovascular disease and 1.4 million respiratory disease-related hospital admissions for the time period assessed.

The coefficient and standard error are calculated from a 0.71% increase in hospital admissions for cardiovascular-related problems (95% CI 0.45%-0.96%) for a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ (Peng et al., 2008, p. 2175).

Functional Form: Log-linear
Coefficient: 0.00071
Standard Error: 0.00013
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions (less acute myocardial infarction) per person ages 65 and older (ICD codes 390-409, 411-429)
Population: population of ages 65 and older.

Hospital Admissions for All Cardiovascular Causes (less AMI) (Peng et al., 2009)

Peng *et al.* (2009) investigated the relationship between hospital admissions for cardiovascular and respiratory disease and the chemical components of $PM_{2.5}$ across 119 U.S. urban communities for 12 million Medicare enrollees using log-linear Poisson regression models. This was achieved using a national database with daily data from 2000-2006 on emergency hospital admissions of cardiovascular and respiratory outcomes, ambient levels of $PM_{2.5}$ components and weather variables. Bayesian hierarchical statistical models were used to estimate the associations.

The coefficient and standard error are calculated from a 0.68% increase in hospital admissions for cardiovascular-related problems (95% CI 0.26%-1.10%) for a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ (Peng et al., 2009, p. 960).

Functional Form: Log-linear
Coefficient: 0.00068
Standard Error: 0.00021
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions (less acute myocardial infarction) per person ages 65 and older (ICD codes 390-409, 411-429)
Population: population of ages 65 and older.

Hospital Admissions for All Cardiovascular Causes (less AMI) (Zanobetti et al., 2009)

Zanobetti *et al.* (2009) examined the relationship between daily $PM_{2.5}$ levels and emergency hospital admissions for cardiovascular causes, myocardial infarction, congestive heart failure, respiratory disease and diabetes among people ages 65 and older in 26 U.S. communities from 2000-2003. The authors used meta-regression to examine how this association was modified by season- and community-specific $PM_{2.5}$ composition while controlling for seasonal temperature as a substitute for ventilation. The authors found that $PM_{2.5}$ mass higher in a variety of elements, including arsenic and organic carbon, significantly increased its effects on hospital admissions.

The coefficient and standard error are calculated from a 1.89% increase in hospital admissions for myocardial infarction (95% CI 1.34%-2.45%) for a 10 $\mu g/m^3$ increase in two-day averaged $PM_{2.5}$ (Zanobetti et al., 2009, Table 3).

Functional Form: Log-linear
Coefficient: 0.00189
Standard Error: 0.00028
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions (less acute myocardial infarction) per person ages 65 and older (ICD codes 390-409, 411-429)
Population: population of ages 65 and older.

Hospital Admissions for All Cardiovascular Causes (less AMI) (Moolgavkar, 2000b)

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

In response to concerns with the Splus issue, Moolgavkar (2003) reanalyzed his earlier study. In the reanalysis, he reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates. Not all of the original results were replicated, however, so this analysis uses a result from the original study.

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

Functional Form: Log-linear
Coefficient: 0.00140
Standard Error: 0.00034
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person ages 18 to 64 (ICD codes 390-409, 411-429)
Population: population of ages 18 to 64.[19]

Pooling Estimates of Hospital Admissions for All Cardiovascular Causes (less AMI)

The all-cardiovascular hospital admission studies for ages 65 and older are themselves large, multi-city cities. Because the authors themselves report a pooled estimate, it would not be appropriate to then used random/fixed effects to pool their individual estimates. The results from the two studies by Peng *et al.* (2008; 2009), which are based on similar databases, were averaged, and then this average was given an equal weight with the studies by Bell *et al.* (2008) and Zanobetti et al (2009). The pooled result from the 65 and older studies was then added to the result for ages 18-64 based on Moolgavkar (2000b).

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

Hospital Admissions for All Respiratory Causes (Zanobetti et al., 2009)

Zanobetti *et al* (2009) examined the relationship between daily $PM_{2.5}$ levels and emergency hospital admissions for cardiovascular causes, myocardial infarction, congestive heart failure, respiratory disease and diabetes among people ages 65 and older in 26 U.S. communities from 2000-2003. The authors used meta-regression to examine how this association was modified by season- and community-specific $PM_{2.5}$ composition while controlling for seasonal temperature as a substitute for ventilation. The authors found that $PM_{2.5}$ mass higher in a variety of elements, including arsenic and organic carbon, significantly increased its effects on hospital admissions.

The coefficient and standard error are calculated from a 2.07% increase in hospital admissions for myocardial infarction (95% CI 1.20%-2.95%) for a 10 $\mu g/m^3$ increase in two-day averaged $PM_{2.5}$ (Zanobetti et al., 2009, Table 3).

Functional Form: Log-linear
Coefficient: 0.00207
Standard Error: 0.00045
Incidence Rate: region-specific daily hospital admission rate for all respiratory admissions per person 65 and older (ICD codes 460-519)
Population: population of ages 65 and older.

Hospital Admissions for Chronic Lung Disease (Moolgavkar, 2000a)

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

In response to concerns with the Splus issue, Moolgavkar (2003) reanalyzed his earlier study. In the reanalysis, he reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates. Not all of the original results were replicated, however, so this analysis uses a result from the original study. The $PM_{2.5}$ C-R functions for the 20-64 age group are based on the original study's single-pollutant model. Since the true PM effect is most likely best represented by a distributed lag model, then any single lag model should underestimate the total PM effect. As a result, the lag models with the greatest effect estimates were selected for use in the C-R functions.

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

Functional Form: Log-linear
Coefficient: 0.0022
Standard Error: 0.000733
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 18-64 (ICD codes 490-492, 494-496)[20]
Population: population of ages 18 to 64.[21]

Hospital Admissions for Asthma (Sheppard *et al.*, 1999; 2003; Babin *et al.*, 2007)

Babin *et al.* (2007) examined pediatric asthma-related emergency room (ER) visits and hospital admissions (ICD-9 code 493) in Washington, D.C. from 2001-2004 and their short-term associations with ozone, particulate matter, socioeconomic status, and age group. Applying Poisson regression analyses, the authors found significant associations between asthma ER visits and outdoor ozone concentrations for the 5-12 year old age group. A weak association was found between $PM_{2.5}$ and asthma hospitalization.

The coefficient and standard error are calculated from a 0.2% increase in hospital admissions for myocardial infarction (95% CI -0.6%-1.1%) for a 1 μg/m^3 increase in daily $PM_{2.5}$ (Babin *et al.*, 2007, Table 2).

Functional Form: Log-linear
Coefficient: 0.00200
Standard Error: 0.00434
Incidence Rate: region-specific daily hospital admission rate for asthma admissions per person ages 0 to 17 (ICD code 493)
Population: population of ages 0 to 17.

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

Sheppard *et al.* (1999) studied the relation between air pollution in Seattle and nonelderly (ages <65) hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM10_{10\text{-}2.5}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects. They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, $PM_{10\text{-}2.5}$, CO, and ozone. They did not observe an association for SO_2. They found PM and CO to be jointly associated with asthma admissions. The best fitting co-pollutant models were

[20] Moolgavkar (2000a) reports results for ICD codes 490-496. In order to avoid double counting non-elderly asthma hospitalizations (ICD code 493), this analysis excludes ICD code 493 from the baseline incidence rate used in this function.
[21] 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.

found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. For the remaining pollutants, the best fitting models included $PM_{2.5}$ and CO. Results for other co-pollutant models were not reported.

In response to concerns that the work by Sheppard *et al.* (1999) may be biased because of the Splus issue, Sheppard (2003) reanalyzed some of this work, in particular Sheppard reanalyzed the original study's $PM_{2.5}$ single pollutant model.

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

Functional Form: Log-linear
Coefficient: 0.003324
Standard Error: 0.001045
Incidence Rate: region-specific daily hospital admission rate for asthma admissions per person ages 0 to 17 (ICD code 493)
Population: population of ages 0 to 17.

Pooling Estimates of Hospital Admissions for Asthma

The estimates generated from the Babin *et al.* (2007) and Sheppard *et al.* (2003) studies were pooled using a random/fixed effects approach to get a single estimate of hospital admissions due to asthma.

Emergency Room Visits for Asthma (Mar *et al.*, 2010)

Mar *et al.* (2010) assessed the effect of particulate matter air pollution, including emissions from diesel generators, on emergency room visits for asthma in the greater Tacoma, Washington area from January 3, 1998 to May 30, 2002 using Poisson regression models. Health data were collected for individuals of all ages from six Tacoma hospitals. Overall, the researchers found an association between daily $PM_{2.5}$ levels and emergency room visits for asthma at lag days 2 and 3, with a relative risk for lag day 2 of 1.04 (95% CI: 1.01-1.07) and a relative risk for lag day 3 of 1.03 (95% CI: 1.0-1.06). No significant association between emergency room visits for asthma and increased use of the diesel generators was observed.

The coefficient and standard error are calculated from a relative risk of 1.04 (95% CI 1.01-1.07) for a 7 μg/m^3 increase in $PM_{2.5}$ (Mar *et al.*, 2010, Table 5).

Functional Form: Log-linear
Coefficient: 0.00560
Standard Error: 0.00210
Incidence Rate: region-specific daily emergency room rate for asthma admissions per person ages 0 to 17 (ICD code 493)
Population: population of ages 0 to 17.

Emergency Room Visits for Asthma (Slaughter *et al.*, 2005)

Slaughter *et al.* (2005) examined the short-term association of particulate matter (PM_1, $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$) and carbon monoxide with hospital admissions and emergency room visits for respiratory and cardiac outcomes and mortality in Spokane, Washington from January 1995 to June 2001 using a log-linear generalized linear model.

The coefficient and standard error are calculated from a relative risk of 1.03 (95% CI 0.98-1.09) for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ (Slaughter *et al.*, 2005, Table 4).

Functional Form: Log-linear
Coefficient: 0.00296
Standard Error: 0.00271
Incidence Rate: region-specific daily emergency room rate for asthma admissions per person ages 0 to 17 (ICD code 493)
Population: population of ages 0 to 17.

Pooling Estimates of Emergency Room Visits for Asthma

The estimates generated from the Mar *et al.* (2010) and Slaughter *et al.* (2005) studies were pooled using a random/fixed effects approach to get a single estimate of emergency room visits due to asthma.

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.[22] 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.

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. This analysis assumes that the C-R function based on Dockery *et al.* is measuring acute bronchitis.

The estimated logistic coefficient and standard error are based on the odds ratio (1.50) and 95% confidence interval (0.91-2.47) associated with being in the most polluted city ($PM_{2.1}$ = 20.7 $\mu g/m^3$)

[22] 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.

versus the least polluted city ($PM_{2.1}$ = 5.8 µg/m^3) (Dockery *et al*., 1996, Tables 1 and 4). The original study used $PM_{2.1}$, however, this analysis uses the $PM_{2.1}$ coefficient and applies it to $PM_{2.5}$ data.

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

Lower Respiratory Symptoms (Schwartz and Neas, 2000)

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

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

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

Upper Respiratory Symptoms (Pope, 1991)

Using logistic regression, Pope *et al*. (1991)estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope *et al*. study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM_{10} concentrations. Pope *et al*. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of

three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma'(Pope, 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope, 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

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

Functional Form: Logistic
Coefficient: 0.0036
Standard Error: 0.0015
Incidence Rate: daily upper respiratory symptom incidence rate per person = 0.3419 (Pope, 1991, Table 2)
Population: asthmatic population ages 9 to 11 = 5.67%[23] of population ages 9 to 11.

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

Ostro *et al*. (2001)studied the relation between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects.[24] Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found cough prevalence associated with PM_{10} and $PM_{2.5}$ and cough incidence associated with $PM_{2.5}$, PM_{10}, and NO_2. Ozone was not significantly associated with cough among asthmatics.

Note that the study focused on African-American children ages 8 to 13 years old. However, it is assumed that the results from this study are applicable to the general population ages 6 to 18 years old.

Asthma Exacerbation, Cough

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

Functional Form: Logistic
Coefficient: 0.000985
Standard Error: 0.000747
Incidence Rate: daily cough rate per person (Ostro *et al*., 2001, p. 202) = 0.145

[23] The American Lung Association (2002a, Table 7) estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).
[24] The authors note that there were 26 days in which $PM_{2.5}$ concentrations were reported higher than PM_{10} concentrations. The majority of results the authors reported were based on the full dataset. These results were used for the basis for the C-R functions.

Population: asthmatic population ages 6 to 18 = 10.7%[25]

Asthma Exacerbation, Shortness of Breath

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

Functional Form: Logistic
Coefficient: 0.002565
Standard Error: 0.001335
Incidence Rate: daily shortness of breath rate per person (Ostro *et al*., 2001, p. 202) = 0.074
Population: asthmatic population ages 6 to 18 = 10.7%

Asthma Exacerbation, Wheeze

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

Functional Form: Logistic
Coefficient: 0.001942
Standard Error: 0.000803
Incidence Rate: daily wheeze rate per person (Ostro *et al*., 2001, p. 202) = 0.173
Population: asthmatic population ages 6 to 18 = 10.7%

Asthma Exacerbation: Cough and Shortness of Breath (Vedal *et al.*, 1998; Mar *et al.*, 2004)

Mar *et al.* (2004) studied the effects of various size fractions of particulate matter on respiratory symptoms of adults and children with asthma, monitored over many months. The study was conducted in Spokane, Washington, a semiarid city with diverse sources of particulate matter. Data on respiratory symptoms and medication use were recorded daily by the study's subjects, while air pollution data was collected by the local air agency and Washington State University. Subjects in the study consisted of 16 adults—the majority of whom participated for over a year—and nine children, all of whom were studied for over eight months. Among the children, the authors found a strong association between cough symptoms and several metrics of particulate matter, including $PM_{2.5}$. However, the authors found no association between respiratory symptoms and PM of any metric in adults.

Asthma Exacerbation, Cough

The coefficient and standard error are based on an odds ratio of 1.21 (95% CI 1.99-1.47) for a 10 μg/m^3 increase in daily average $PM_{2.5}$ concentration (Mar *et al*., 2004, Table 7).

Functional Form: Logistic
Coefficient: 0.01906

[25] The American Lung Association (2010, Table 7) estimates asthma prevalence for children 5-17 at 10.7% in 2008.

Standard Error: 0.00983
Incidence Rate: daily cough rate per person (Ostro *et al*., 2001, p. 202) = 0.145
Population: asthmatic population ages 6 to 18 = 10.7%[26]

Asthma Exacerbation, Shortness of Breath

The coefficient and standard error are based on an odds ratio of 1.13 (95% CI 0.86-1.48) for a 10 μg/m^3 increase in daily average $PM_{2.5}$ concentration (Mar *et al*., 2004, Table 7).

Functional Form: Logistic
Coefficient: 0.01222
Standard Error: 0.01385
Incidence Rate: daily shortness of breath rate per person (Ostro *et al*., 2001, p. 202) = 0.074
Population: asthmatic population ages 6 to 18 = 10.7%

Pooling Estimates of Asthma Exacerbation

The following approach was used to combine the estimates generated using effect estimates from the Ostro *et al.* (2001) and Mar *et al.* (2004) studies to produce a single asthma exacerbation incidence estimate. First, the separate incidence estimates from the Ostro *et al.* (2001) study for cough, shortness of breath, and wheeze and from the Mar *et al.* (2004) study for cough and shortness of breath were generated. The two estimates for cough were pooled using a random/fixed effects approach, and the two estimates for shortness of breath were pooled. The pooled estimates for cough and shortness of were then averaged with wheeze estimate, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions.

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

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, restricted activity days (RADs), and respiratory-related restricted activity days (RRADs),

[26] The American Lung Association (2010, Table 7) estimates asthma prevalence for children 5-17 at 10.7% in 2008.

however there was some year-to-year variability in the results.[27] Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function presented here is a weighted average of the coefficients in Ostro (1987, Table 3) using the inverse of the variance as the weight.

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table 3) using the inverse of the variance as the weight. The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent.

Functional Form: Log-linear
Coefficient: 0.0046
Standard Error: 0.00036
Incidence Rate: daily work-loss-day incidence rate per person ages 18 to 64 = 0.00595 (U.S. Bureau of the Census, 1997, No. 22; Adams *et al.*, 1999, Table 41)
Population: adult population ages 18 to 64.[28]

Minor Restricted Activity Days (Ostro, 1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone has highly variable association with RRADs and MRADs. Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.[29] The C-R function for PM is based on this co-pollutant model.

Using the results of the two-pollutant model, separate coefficients were developed for each year in the analysis, which were then combined for use in this analysis. The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight. The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent.

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

[27] The study used a two-week average pollution concentration; the C-R function uses a daily average, which is assumed to be a reasonable approximation.
[28] The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65.
[29] The study used a two-week average pollution concentration; the C-R function uses a daily average, which is assumed to be a reasonable approximation.

Population: adult population ages 18 to 64.[30]

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

Appendix B. Health Incidence Data

Health impact functions developed from log-linear or logistic models estimate the percent change in an adverse health effect associated with a given pollutant change. In order to estimate the absolute change in incidence using these functions, this analysis needed the baseline incidence rate of the adverse health effect, or the number of cases experienced by a given population per unit of time. And in the case of asthma, a prevalence rate, or percentage of the population affected (in this case asthmatic), was needed as well. This appendix describes the data used to estimate baseline incidence and prevalence rates for the health effects considered in this analysis.

The incidence data used in this analysis are available in version 4.0 of BenMAP and are described in Appendix E of the BenMAP User Manual.[31] The description below for mortality, hospitalization, emergency room visits, and heart attacks is drawn from the BenMAP User Manual. Details on incidence and prevalence rates for other health impacts are described in the Appendix detailing each health impact function.

Mortality Incidence Rate

The mortality data used in this analysis is a forecasted estimate for 2010 that comes with the BenMAP software. It would have been preferred to have a 2008 mortality estimate, as this is the midpoint of our ambient $PM_{2.5}$ data range of 2007-2009, but this was not available. Since mortality rates have gradually declined over time, using a 2010 rate rather than 2008 is a conservative approach, tending to slightly under-estimate the mortality rate.

The 2010 mortality rate estimates are ultimately based on individual-level mortality data from2004-2006 for the whole United States were obtained from the Centers for Disease Control (CDC), National Center for Health Statistics (NCHS). Since the detailed mortality data obtained from the CDC do not include population, these data were combined with U.S. Census Bureau post-censal population estimates exported from BenMAP. Age-, cause-, and county-specific mortality rates were then generated.

To estimate age- and county-specific mortality rates in years 2010, the developers of BenMAP calculated adjustment factors, based on a series of Census Bureau projected national mortality rates (for all-cause mortality), to adjust the age- and county-specific mortality rates calculated using 2004-2006 data. Details of these calculations are described in Appendix E of the BenMAP User Manual (Abt Associates Inc., 2010).

[31] The BenMAP software and its user manual are available from the BenMAP website: http://www.epa.gov/oaqps001/benmap/download.html.

Hospital Admission Incidence Rate

Hospitalization rates were calculated using data from the Healthcare Cost and Utilization Project (HCUP). As noted in Appendix E of the BenMAP User Manual, HCUP is a family of health care databases developed through a Federal-State-Industry partnership and sponsored by the Agency for Healthcare Research and Quality. HCUP products include the State Inpatient Databases, the State Emergency Department Databases, the Nationwide Inpatient Sample, and the Nationwide Emergency Department Sample. HCUP databases can be obtained from the following data services:

HCUP Central Distributor: Many of the HCUP databases are available for purchase through the HCUP Central Distributor. The databases include detailed information for individual discharges, such as primary diagnosis (in ICD-9 codes), patient's age and residence county.

HCUP State Partners: Some HCUP participating states do not release their data to the Central Distributor; however, the data may be obtained through contacting the State Partners. Some State Partners (e.g., CA, TX, and NY) provided discharge-level data; others (e.g., OH) provided summarized data.

HCUPnet: This is a free, on-line query system based on data from HCUP.

The developers of BenMAP combined the data from these different sources and developed a nominally county-level database, however, in some cases more aggregated data were used. For some states neither discharge-level nor state-level data were available. In such cases regional statistics were used from HCUPnet to estimate hospitalization rates for those states. The data year for most states is 2007; the exception is MA, for which the data year is 2006.

Non-Fatal Heart Attack Incidence Rate

As in the case of hospital admissions, the emergency room visit incidence rate is based on HCUP data, and thus comes from a variety of sources. The data year for most states is 2007; the exception is MA, for which the data year is 2006.

Emergency Room Visit Incidence Rate

As in the case of hospital admissions, the emergency room visit incidence rate is based on HCUP data, and thus comes from a variety of sources. The data year varies across the states from 2005 to 2007; we assumed that ER visit rates are reasonably constant across these three years and consider them as 2006 rates.

Appendix C. Population Data

The population estimate used in this analysis is for 2008, which is the midpoint of the 2007-2009 air quality monitoring used in the rollback analyses. To develop the 2008 population estimates, the analysis started with block data from the 2000 Census.[32] To account for the change in population between 2000 and 2008, the analysis adjusted the U.S. Census Bureau data with population projections based on economic forecasting models developed by Woods and Poole,[33] which has the most detailed (county-level) forecasts available covering the entire nation.

The Woods and Poole database contains county level projections of population by age, sex, race, and ethnicity. Projections in each county are determined simultaneously with every other county in the U.S. to take into account patterns of economic growth and migration. The sum of growth in county level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates.[34] According to Woods and Poole, linking county level growth projections together and constraining to a national level total growth avoids potential errors introduced by forecasting each county independently.

Woods and Poole developed county projections in a four stage process. First, national level variables such as income, employment, populations, *etc*. are forecasted. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an "export-base" approach, which relies on linking industrial sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-base approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector. Third, population is projected for each economic area based on net migration rates derived from employment opportunities, and following a cohort-component method based on fertility and mortality in each area. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2025 based on historical rates of mortality, fertility, and migration.

The present analysis estimated 2008 grid-cell population by using 2000 Census block-level population estimates and the percentage change in population estimates based on the county-level Woods & Poole data. This occurred in a three-step process.

Step 1. Using the PopGrid model, the analysis summed the 2000 block data to the level of the air quality modeling data (CAMx grid-cell), keeping track of the (one or more) counties that overlap with the grid-cell. The 2000 block data are the most disaggregated data provided by the Census Bureau, and typically cover a much smaller geographic area than the air quality models. This summing generated CAMx

[32] (GeoLytics Inc., 2002)
[33] (Woods & Poole Economics Inc., 2001)
[34] (Hollman *et al.*, 2000)

population grid-cells with year 2000 population data, as well as the percentage of the total population in each grid-cell falling within a particular county.

Step 2. The analysis used the Woods and Poole data to estimate the percentage change (from 2000 to 2008) in the population by age, sex, and race. The analysis then calculated a growth adjustment factor equal to one plus the percentage change (*e.g.,* if the percentage change equals two percent, then the adjustment factor is 1.02).

Step 3. Finally, the analysis multiplied the appropriate age, sex, and race growth adjustment factor for each county with the appropriate population in each grid-cell.

For example, when forecasting a single population variable, say, children ages 4 to 9 in the year 2008, BenMAP calculated:

$$age_{4-9,\,g,\,2008} = age_{4-9,\,g,\,2000} * \frac{age_{4-9,\,county,\,2008}}{age_{4-9,\,county,\,2000}}$$

where the g[th] population grid-cell is wholly located within a given county.

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

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

BenMAP multiplied this fraction with the number of individuals ages 4 to 9 in the year 2000, to give an estimate of the number of individuals ages 4 to 9 that reside in the fraction of the county within the g[th] grid-cell in the year 2000:

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

To then forecast the population in 2008, BenMAP scaled the 2000 estimate with the ratio of the county projection for 2008 to the county projection for 2000:

$$age_{4-9,\,g\ in\ county_c,\,2008} = age_{4-9,\,g,\,2000} * \frac{age_{4-9,\,county_c,\,2008}}{age_{4-9,\,county_c,\,2000}}$$

Combining the contributions from each county within each given grid-cell gave population of persons ages 4 to 9 in the year 2008.

Appendix D. Resume of Dr. McCubbin

Dr. Donald McCubbin has over 15 years of experience conducting analyses of environmental issues. He managed the development of BenMAP, led training programs for state, federal and international clients, wrote the BenMAP user manual, and generated and maintained extensive databases of population, health statistics, epidemiological results, and health costs. Dr. McCubbin analyzed the health benefit of the Clean Air Act, major regulations, such as the Heavy Duty Diesel and Clean Air Interstate rules, and the impacts of power plants, motor vehicles, and other pollution sources. He has written a number of peer-reviewed articles and book chapters, as well as many reports on air quality issues and health impacts. He has given expert witness testimony for the Department of Justice, and he received EPA's Level 1 Science and Technological Achievement Award for a peer-reviewed research article on the health benefits of alternative ozone standards.

Education

Ph.D. Economics, University of California, San Diego
M.S. Agricultural Economics, University of California, Davis
M.S. Ecology, University of California, Davis
B.A. Biological Basis of Behavior, University of Pennsylvania

Relevant Professional Experience

Institute of Transportation Studies, University of CA, Davis. Research Analyst. Developing a model of the climate change impacts associated with transportation fuels. (2009-Present)

Abt Associates Inc. Principal Associate. Designed and managed the development of innovative software tools to estimate air pollution exposure and the associated health and economic costs. Prepared numerous reports, including examining the co-control benefits of greenhouse gas emission reductions, dioxin fate and transport, and the environmental and health impacts of ammonia from livestock production. Worked on a variety of international projects in Mexico, Dominican Republic, Guatemala, Belize, Nicaragua, Chile, Bolivia, India, Tanzania, and Ghana. (1998-2009)

Awards

EPA Level I Scientific And Technological Achievement Award. 2005. Given by EPA for an exceptionally high-quality research of national significance, recognizable as a major scientific/technological achievement within its discipline or field of study

Daniel Bell Award. 2005. Given by Abt Associates each year for outstanding social science research.

Selected Publications

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Davidson, K, A. Hallberg, B. Hubbell, D.R. McCubbin (2007). Analysis of PM2.5 Using the Environmental Benefits Mapping and Analysis Program (BenMAP). Journal of Toxicology and Environmental Health, Part A, 70: 332-346.

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