A Particulate Matter Risk Assessment for Philadelphia and Los Angeles

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REVISED REPORT

This is a revised version of the report submitted to EPA on July 3, 1996. The revisions correct minor errata discovered in the tables presented in the July 3rd report. No new analyses are presented in this report. Only minimal revisions have been made to the text where necessary to reflect the changes in the tables. The pagination has been changed somewhat to incorporate these changes. Revised version submitted to EPA November, 1996 by Abt Associates Inc.

DISCLAIMER

This report is being furnished to the U.S. Environmental Protection Agency by Abt Associates Inc. in partial fulfillment of Contract No. 68-W4-0029, Work Assignment No. 15-1. Some of the preliminary work for this report was completed under Work Assignments 9 and 9-1 of the same contract. The opinions, findings, and conclusions expressed are those of the authors and are not necessarily those of the Environmental Protection Agency. Inquiries should be addressed to Mr. Harvey Richmond, U.S. EPA, Office of Air Quality Planning and Standards, MD-15, Research Triangle Park, North Carolina 27711.

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A PARTICULATE MATTER RISK ANALYSIS FOR PHILADELPHIA AND LOS ANGELES

1. Introduction

Assessing the impacts of ambient particulate matter (PM) on human health has been a concern of epidemiological research and government policy for many years. Because PM is identified as a "criteria pollutant" by the Environmental Protection Agency (EPA) under the Clean Air Act, PM standards must be reevaluated periodically. An assessment of the current health risks due to PM and the reduction in health risks associated with achieving alternative PM standards is part of this process. This document reports the method and results of analyses to assess the risks associated with current levels of ambient PM in two selected locations and to estimate the risk reductions that might be achieved in those locations by attainment of alternative PM standards.

The Criteria Document (EPA, 1996a) and Staff Paper (EPA, 1996b) evaluate the scientific evidence on the health effects of PM, including information on exposure routes, the physiological mechanisms by which PM might damage human health, and concentration-response components of risk assessment. The risk analysis described in this report builds on that work. It draws on the hazard identification and concentration-response information provided in the Criteria Document in order to estimate the incidence of health effects associated with "as is" ambient PM concentrations and the incidence of health effects that might be avoided by the attainment of alternative PM standards or sets of standards.

The relationship between a health response and ambient PM concentration is referred to in this report as the (ambient) concentration-response relationship. It is the relationship between the average ambient concentration of PM (in $\mu g/m^3$) and the population response (number of individuals exhibiting the health response). In contrast, the relationship between a health response and individual exposure to PM is referred to as an individual exposure-response relationship. This is the relationship between the actual exposure to PM (in $\mu g/m^3$) experienced by the individual and the probability that that individual will exhibit the health response.

Both the individual exposure-response relationship and the ambient concentration-response relationship are of interest. The individual exposure-response relationship is of clear scientific interest. This is the relationship that epidemiological studies would presumably estimate if data on individual exposure were available for each member of a population.[1] However, for the National Ambient Air Quality Standards (NAAQS), which influence ambient concentrations of PM (through environmental policies and regulations that lead to meeting the

[1]Because of the lack of individual exposure data, epidemiological studies typically use ambient concentration as a surrogate for individual exposure, effectively estimating the ambient concentration-response function rather than the individual exposure-response function. While doing this may result in a biased estimate of the individual exposure-response function, it does not result in a biased estimate of the concentration-response function, which is what is actually estimated. This is explained more fully in Appendix 1.

standards), the ambient concentration-response relationship is of primary regulatory interest. That is, it is important to predict the risk reduction associated with changing ambient concentrations, rather than the risk reduction associated with changing individual exposure (which is not directly controlled by the NAAQS). It is therefore the concentration-response functions which are appropriate to use in the PM risk analysis. The relationship between the individual exposure-response function and the (ambient) concentration-response function is examined formally in Appendix 1.

The risk analysis considers two different PM indicators. The indicator for the current air quality standard is defined as those particles of diameter less than or equal to 10 microns and is denoted as PM-10. The Staff Paper (EPA, 1996b) recommends consideration of an indicator measuring fine particles, defined as those of aerodynamic diameter less than or equal to 2.5 microns and denoted as PM-2.5. Both PM-10 and PM-2.5 are examined in the risk analyses.[2]

There are two major phases of the risk analysis. The first phase assesses the risks associated with "as is" PM concentrations in a specified location.[3] If the location is not in attainment of current standards, risk analyses are carried out in two ways: (1) daily PM concentrations are left unadjusted, and (2) daily PM concentrations are first adjusted to simulate attainment of the current standards prior to the analysis. The method of adjustment is described in Section 2.2 below. The basic question addressed in the first phase of the risk analysis is of the following form:

> *For a given human health endpoint (mortality, hospital admissions, etc.), what is the estimated incidence of the health endpoint that may be associated with "as is" PM concentrations?*

The second phase of the risk analysis estimates the risk reductions that would be associated with the attainment of alternative PM standards as opposed to attainment of current standards. Annual average PM-2.5 standards of 15 and 20 $\mu g/m^3$ are each considered alone as well as in combination with daily PM-2.5 standards of 65, 50, and 25 $\mu g/m^3$, respectively. Attainment of a standard or set of standards is simulated by adjusting "as is" daily PM concentrations to daily PM concentrations that would just meet the standard(s). The impact on human health is assessed by comparing the health risks associated with PM concentrations that attain the alternative PM-2.5 standards with the health risks associated with the "as is" PM

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[2]While "risk analysis" is used to refer to each separate analysis (e.g., for a particular location under particular assumptions), the entire collection of risk analyses is referred to interchangeably as (the set of) "risk analyses" or as a "risk analysis."

[3]Risk is assessed for PM levels down to the lowest level observed in the study reporting the concentration-response function, but not lower than background level in the sample location. If the lowest observed PM level was not reported, risk is assessed down to background level in the sample location. Background PM level is the PM concentration in the absence of controllable anthropogenic sources in North America. Background concentrations are treated in a manner consistent with the Criteria Document.

concentrations that attain the current (PM-10) standards. The basic question addressed is of the following form:

> *For a given reduction in PM concentrations and a given human health endpoint (mortality, hospital admissions, etc.), what is the estimated reduction in incidence of the health endpoint associated with the reduction in PM concentrations?*

As in the first phase of the risk analysis, if the location is not in attainment of current standards, daily PM concentrations are adjusted to simulate attainment of current standards prior to the analysis.

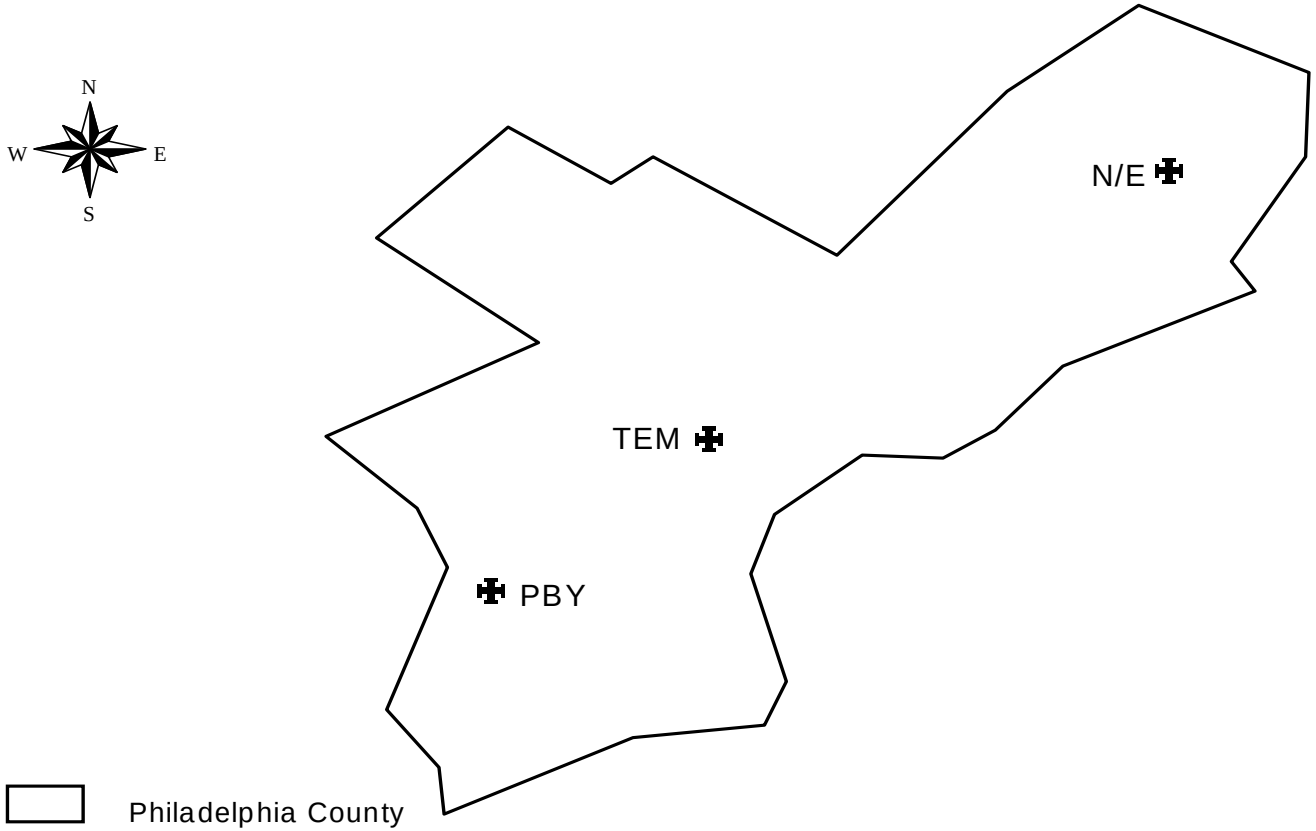
The PM risk analysis described in this report is not a national risk assessment, nor does it model micro-environment exposure (as was done as part of the risk assessment prepared for the recent review of the ozone NAAQS[4]). Extensive risk analyses are instead carried out for two sample locations by applying concentration-response functions from epidemiological studies to data on daily ambient PM-10 and PM-2.5 levels in each location (consistent with the general approach taken in the ozone risk analyses involving risk estimates based on epidemiology studies).

The two locations chosen for risk analysis are Philadelphia County, Pennsylvania and Southeast Los Angeles County, California. The geographic region comprised, the population encompassed within the region, and the placement of air quality monitors used in the risk analysis are illustrated in Exhibit 1.1 for Philadelphia County and Exhibit 1.2 for Southeast Los Angeles County. A portion of southeastern Los Angeles County selected for use in the analysis includes the portion of the county with the highest PM-10 levels. The region included in this analysis approximates the portion of the county reported to have an annual average PM-10 level above 40 $\mu g/m^3$ in 1994 (from "Air Quality Standards Compliance Report," South Coast Air Quality Management District, 1995). The size and age distribution of the population living within the selected region was estimated by totaling the population of U.S. Census block groups falling within the region. A block group is considered to be within the region if the population-weighted centroid of the block group is within the boundary of the region.

[4]"Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information" (EPA, 1996c), and "A Probabilistic Assessment of Health Risks Associated With Short-Term Exposure to Tropospheric Ozone" (Whitfield et al., 1996).

Exhibit 1.1

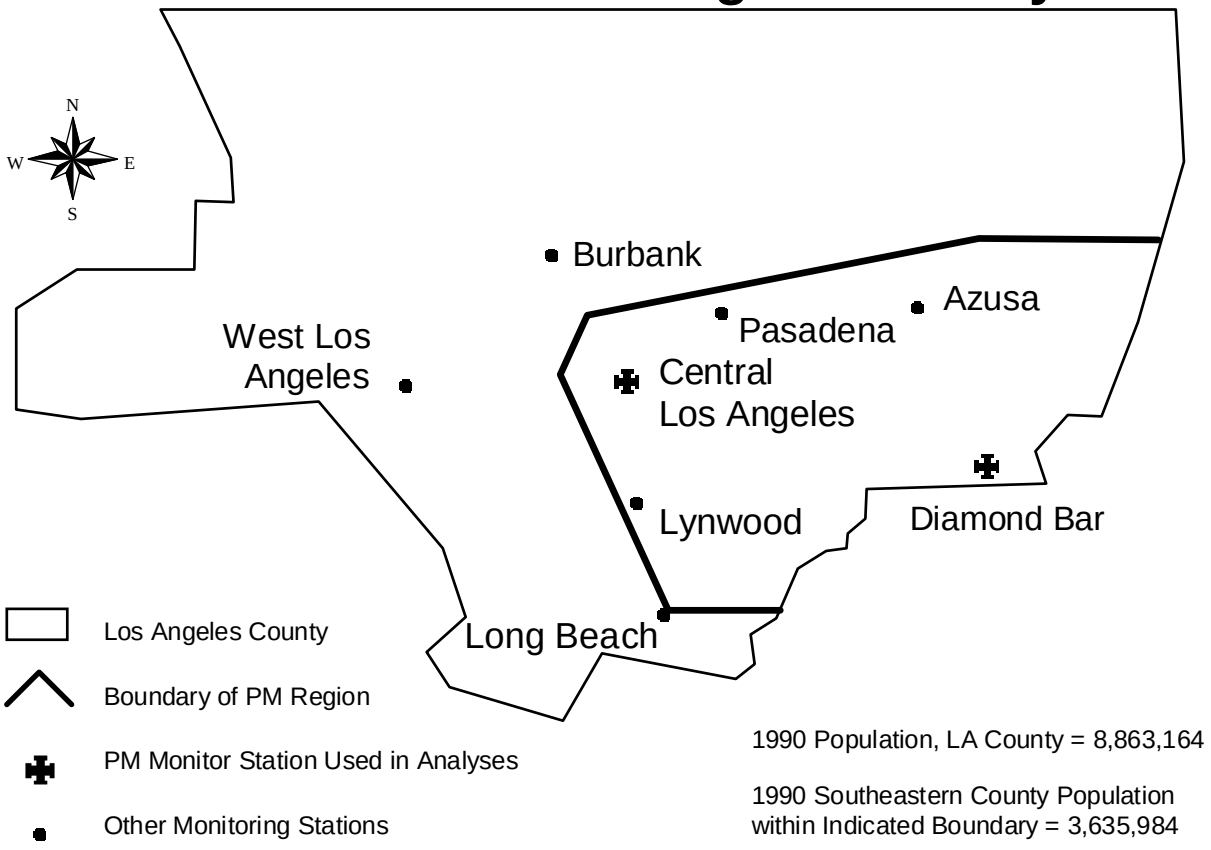
Monitor Locations Used for PM Analyses in Philadelphia County



1990 Population, Philadelphia County = 1,585,577

Exhibit 1.2

Geographic Region Used for PM Analyses in Southeast Los Angeles County



Philadelphia County has virtually complete daily air quality data for both PM-10 and PM-2.5. For a one-year period from September 1992 through August 1993, monitor data for PM-10 are available for 98.6 percent of the days in the year, and monitor data for PM-2.5 are available for 96.4 percent of the days in the year. In addition, Philadelphia County has been the site of extensive investigation of the health effects of air pollution.

Southeast Los Angeles County, a western location, provides a contrast to Philadelphia County in type of particulate matter. In addition, as in Philadelphia County, substantial air quality data for both PM-10 and PM-2.5 are available for Los Angeles. Finally, several health studies have been carried out in this city.

Numerous epidemiological studies are used in the risk analyses. Most studies focus on adverse effects associated with elevations in PM levels during short time periods. These studies, referred to as "short-term exposure" studies, draw current incidence levels primarily from hospital and vital statistics records. The "long-term exposure" studies, on the other hand, evaluate mortality or morbidity in relation to long-term air quality, characterized by annual mean levels of PM. These studies used large cohorts of adults with specifically defined characteristics who were followed over years of observation. The health endpoints for which the largest number of studies are available are mortality, hospital admissions for pneumonia, hospital admissions for Chronic Obstructive Pulmonary Disease (COPD), and "total respiratory" hospital admissions. (The exact set of ICD codes included in "total respiratory" admissions varies from study to study.)

In some cases, most notably in the case of mortality and PM-10, concentration-response relationships have been estimated by several studies in the literature. For those health effects for which associations with PM have been estimated in several studies, ideally, the data sets from these studies could be combined and re-analyzed to produce a more robust estimate of PM health effects. When it is impossible to combine the data, however, there are various ways to pool the results of the studies to derive a concentration-response function. One method, which is used in this risk analysis, is described in Appendix 2.

In the first phase of the risk analysis, assessing the risk associated with "as is" ambient PM concentrations, the number of separate analyses is determined by the number of health endpoint-PM indicator combinations for which concentration-response functions have been estimated. In the second phase of the risk analysis, assessing the risk reduction associated with attaining possible alternative (sets of) standards, the number of separate analyses is determined by the number of health endpoints and the number of (sets of) alternative standards considered. If there are N health endpoints and M sets of PM standards of interest, there is a maximum of N*M analyses possible.

An overview of the major components of the PM risk analysis discussed in this report is presented in Exhibit 1.3. Each separate analysis in the first phase of the risk analysis depends on the following four basic components:

- air quality information,
- information on the concentration-response relationship between the health endpoint of interest and the PM indicator of interest in the location of interest;
- baseline health incidence information for the location of interest; and
- the size of the population living in the location of interest.

If the location is not in attainment of current PM standards (as in Los Angeles), the first phase of the risk analysis requires, prior to risk analysis,

- the simulation of attainment of current standards.

Each separate analysis in the second phase of the risk analysis depends on an additional component:

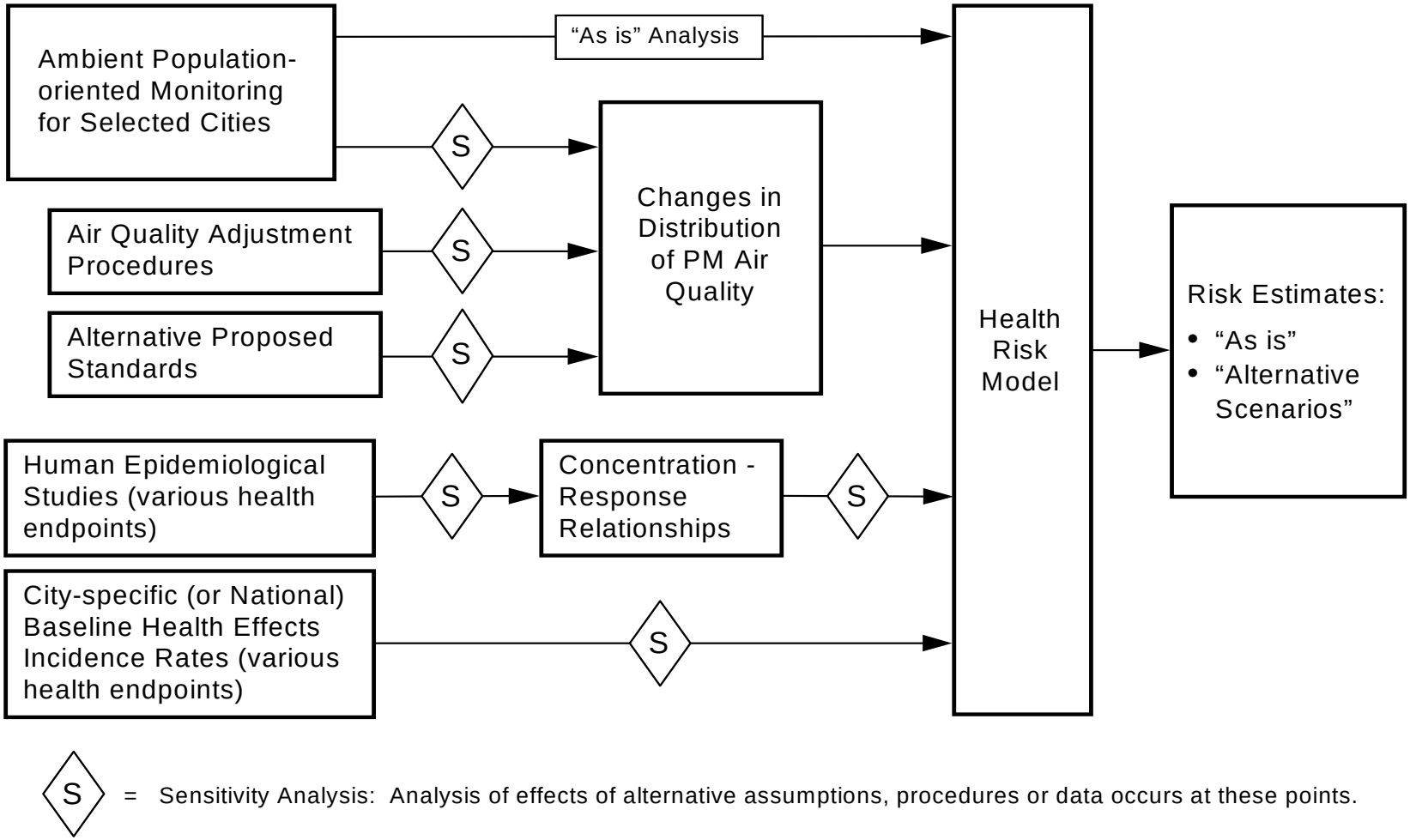
- the simulation of attainment of a set of alternative PM-2.5 standards.

There are substantive issues surrounding each of these components. These issues and approaches in the absence of complete information on any one or more of these risk analysis components are discussed at length in the sections that follow.

The basic methods used in all the analyses, and methodological issues specific to particular parts of the risk analysis (e.g., to one phase or the other), are discussed in Section 2. Because the risk analyses were carried out in the face of incomplete information, it was necessary to make assumptions at several points in the analysis process. These assumptions and the various sources of uncertainty surrounding risk estimates are discussed in Section 3. Section 4 discusses the Philadelphia County and Southeast Los Angeles County air quality data used in the analyses, that is, the ambient PM-10 and PM-2.5 data from these locations. The PM-10 and PM-2.5 concentration-response functions used in the analyses are discussed in Section 5. Concentration-response functions in the epidemiological literature that were considered in the risk analysis are given in Section 5.1. The estimation of a distribution of concentration-response functions (across locations), the estimation of a concentration-response function in any given location, and the characterization of the uncertainty surrounding concentration-response functions is discussed in Section 5.2. Section 6 presents baseline health effects incidence rates for each of the locations from vital statistics sources. These are the health effects incidence rates associated with “as is” PM levels.

In both phases of the risk analysis, there is substantial uncertainty. The results of the analyses depend on a number of analytic choices and will change if different choices are made.

Exhibit 1.3 Major Components of Particulate Matter Health Risk Analysis



S = Sensitivity Analysis: Analysis of effects of alternative assumptions, procedures or data occurs at these points.

One way to assess the impact of particular analytic choices on the results of the risk analyses is through sensitivity analyses, in which the impact of different analytic choices on the results of the risk analysis is assessed.

The assessment of the risk associated with "as is" PM concentrations in Philadelphia County and Southeast Los Angeles (the first phase of the risk analysis) is presented in Section 7. The results and associated sensitivity analyses are presented in Section 7.1. Monte Carlo propagation of uncertainty analyses, considering the uncertainty from several sources, are presented in Section 7.2.

The assessment of the risk reduction associated with attaining possible alternative (sets of) standards in these two locations (the second phase of the risk analysis) is presented in Section 8. The results and associated sensitivity analyses are presented in Section 8.1. Other sensitivity analyses concerning the rollback method are presented in Sections 8.3. Alternative forms of PM standards are considered in Section 8.3. Monte Carlo propagation of uncertainty analyses, considering the uncertainty from several sources, are presented in Section 8.4. Finally, issues of interpretation of the results of the risk analysis are discussed in Section 9.

2. Methods

This section describes the basic methods of the risk analysis. Conducting a risk analysis requires substantial information. For each of the elements of the risk analyses described below complete and certain information is not available, resulting in a significant degree of uncertainty. The sources of uncertainty and the assumptions made to perform risk analyses in the face of incomplete information are discussed in Section 3.

2.1. Overview

Each separate analysis in either the first phase or the second phase of the risk analysis can be characterized as estimating the change in the incidence of a given health effect resulting from a given change in PM concentrations. In the first phase, risk analyses consider the health effects incidence associated with "as is" PM above either the lowest PM level observed in the study or background level. (In Los Angeles County, where "as is" concentrations do not meet current PM standards, the health effects incidence associated with unadjusted "as is" PM concentrations and the health effects incidence associated with "as is" PM concentrations adjusted to simulate attainment of current standards are both considered.) This is equivalent to assessing the potential change in health effects incidence associated with a reduction in PM concentrations from "as is" levels (in or out of attainment with current standards) to the specified lower PM level (either the lowest observed in the study or background level).

In the second phase, risk analyses consider the change from "as is" PM concentrations to those concentrations that would just attain a specified set of alternative PM standards. The method used in both phases of the risk analysis is therefore basically the same. The important difference between the two phases is in the specified alternative (lower) PM levels: in the first phase the alternative air quality is either the lowest PM level observed in the study or background PM level, whereas in the second phase the alternative air quality is based on attainment of a set of alternative PM-2.5 standards. The first phase therefore requires either a reported lowest observed PM level or an estimate of background PM (PM-10 and PM-2.5) level; the second phase requires that a method be developed to simulate attainment of the specified standard(s). This method is applied to the first phase as well to simulate attainment of current PM-10 standards where appropriate prior to risk analyses.

To estimate the change in the incidence of a given health effect resulting from a given change in ambient PM concentrations in a sample location, the following elements are necessary:

(1) air quality data from the sample location to estimate both "as is" PM concentrations and, for the second phase of the risk analysis, the concentrations associated with attainment of proposed PM standards;

(2) a concentration-response function estimating the relationship between ambient PM concentrations and the health endpoint in the sample location (preferably

derived in the same location, although functions estimated in other locations can be used at the cost of increased uncertainty -- see Section 3.1.2); and

(3) an estimate of the baseline health effect incidence (rate) corresponding to "as is" PM levels (since most of the available concentration-response functions give a percent change in incidence rather than an absolute number of cases).

The change in the health endpoint may be measured as a daily change, corresponding to changes in daily average ambient PM concentrations from "as is" levels to some alternative levels (e.g., either background or those levels corresponding to attainment of a set of standards). Alternatively, the change in the health endpoint may be measured as an annual change, corresponding to a change in the annual average PM concentration. When concentration-response functions from short-term exposure studies are used, it is appropriate to assess daily effects. When concentration-response functions from long-term exposure studies are used, it is appropriate to assess annual effects. When daily effects are calculated, these daily changes are aggregated, and, in the absence of PM data for all 365 days of the year, adjusted to reflect the total for the entire year, as described below. All changes in health effects, whether calculated on a daily or annual basis, are therefore aggregates for an entire year. The risk analysis procedure described in more detail below is diagramed in Exhibit 2.1 for analyses based on short-term exposure studies and Exhibit 2.2 for analyses based on long-term exposure studies.

Because there are substantive methodological issues involved in simulating the attainment of a set of standards (either current PM-10 standards or alternative PM-2.5 standards), this is discussed separately in Section 2.2 below. The functional form of the concentration-response relationships used in the risk analyses, and the prediction of changes in health effects incidence associated with changes in ambient PM concentrations using these concentration-response functions is described in Section 2.3. Issues involved in the calculation of annual health effects incidence are discussed in Section 2.4. A brief discussion of issues involved in attaining baseline incidence rates is given in Section 2.5. Finally, the sensitivity analyses carried out in both phases of the risk analysis, and any methodological issues pertaining to them, are described in Section 2.6.

2.2. Modeling attainment of alternative (or current) standards

Predicting the change in risk due to a change in air quality from an "as is" annual mean to meet a lower annual standard when using a concentration-response function from a long-term exposure study is straightforward: the "as is" mean is simply reduced to the standard level. In this case, simulating attainment of a standard does not involve generating an alternative set of daily PM concentrations, because the concentration-response function

Exhibit 2.1

Flow Diagram of Typical Risk Analysis for Short-Term Exposure Studies

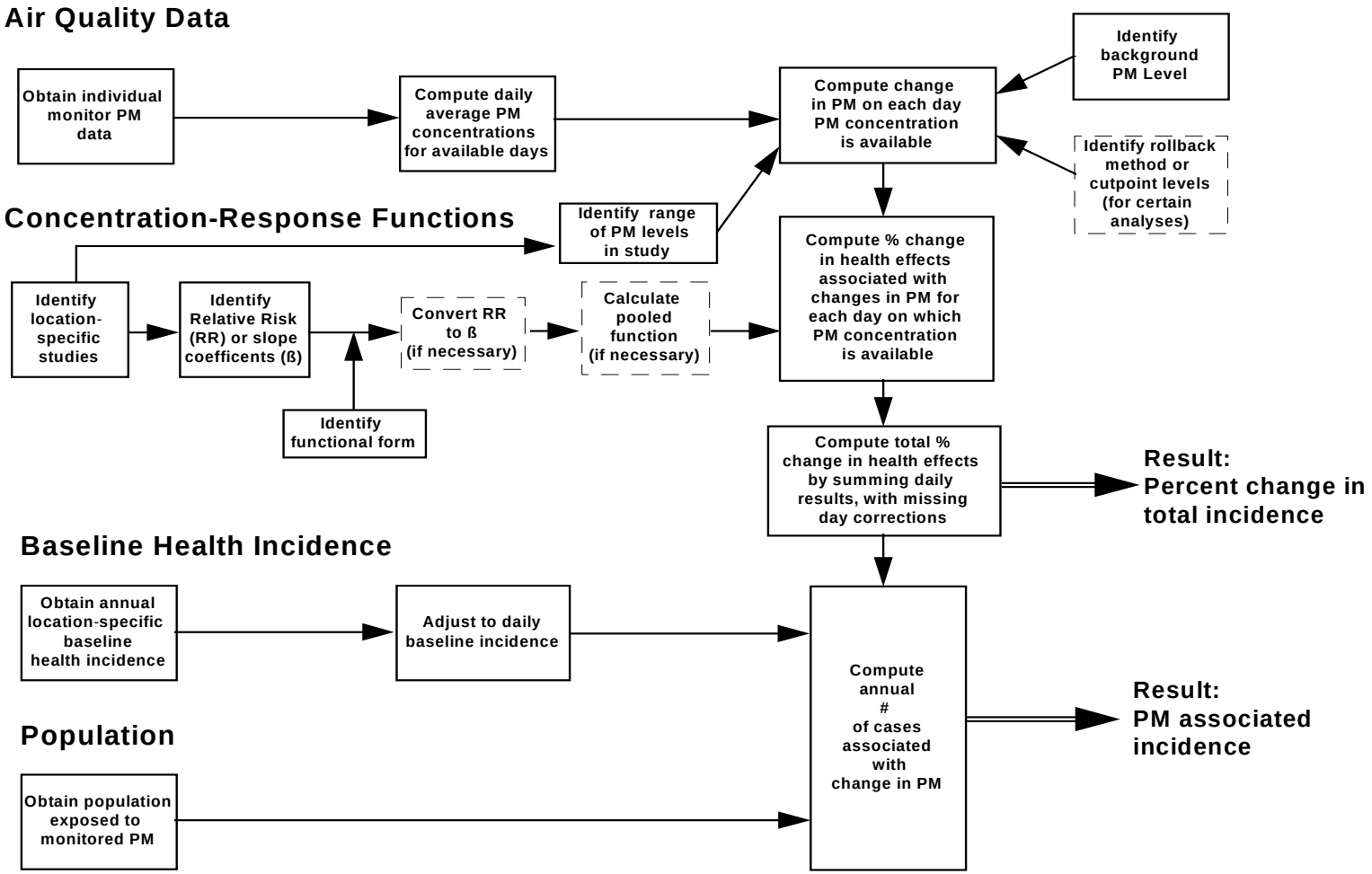
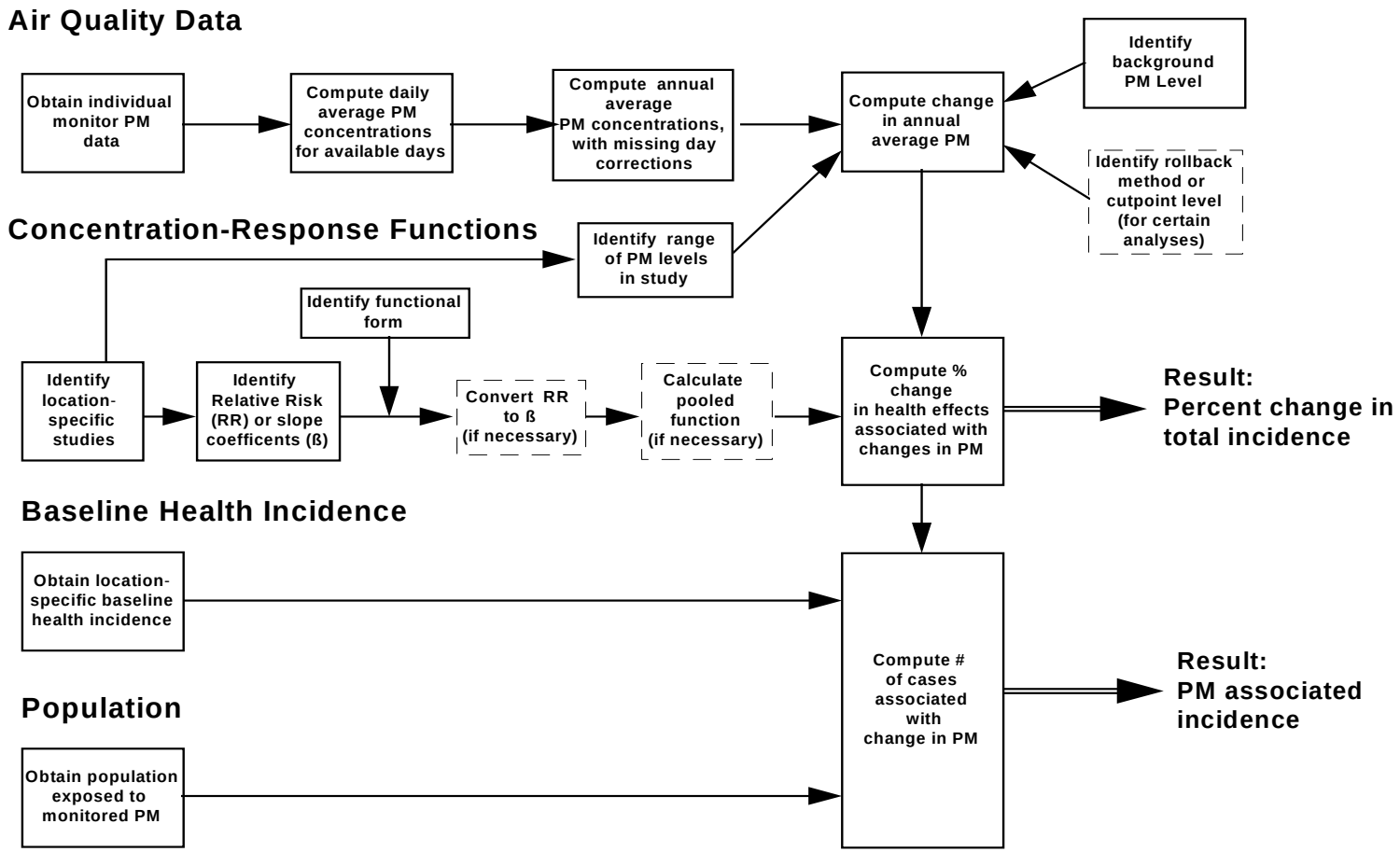


Exhibit 2.2

Flow Diagram of Typical Risk Analysis for Long-Term Exposure Studies



estimated in a long-term exposure study is based on annual, rather than daily PM concentrations.

When a concentration-response function from a short-term exposure study is used, however, attainment of an alternative standard or set of standards is best simulated by changing the distribution of daily PM concentrations. This section discusses the methods used to change daily PM concentrations in a sample location to simulate the attainment of a new standard or set of standards. The methods described below are also applicable to the simulation of attainment of current standards when a location is not already in attainment, as discussed below.

An area is considered in attainment of a standard if all PM monitors in the area are in attainment. An area is in attainment of an annual standard if the annual average PM concentration at each monitor in the area is at or below the standard. An area is in attainment of a daily standard (which currently allows one exceedence) if no more than one monitor-day exceeds the daily standard. Although it is possible to change the daily PM concentrations at each monitor separately (to separate degrees) to simulate attainment, this would require extensive analysis that is beyond the scope of this risk analysis. Therefore, although the amount or percent of reduction on a given day might be determined by the PM concentration at a single monitor on a single day, attainment is simulated by changing daily concentrations averaged over all monitors.

There are many different methods of reducing daily PM levels that would result in attainment of a given PM standard or set of standards. Preliminary analyses of historical PM data found that year-to-year reductions in PM levels in a given location tended to be roughly linear. That is, both high and low daily PM levels decreased proportionally. (This is discussed more fully in the discussion of the sensitivity of results to the rollback method, in Section 8.) This suggests that, in the absence of detailed air quality modeling, it is reasonable to simulate PM reduction to bring a sample location into attainment of new proposed standards by proportional rollbacks (i.e., by decreasing PM levels on all days by the same percentage).

Proportional (linear) rollback is only one of many possible ways, however, to create an alternative distribution of daily concentrations to meet new PM standards. One could, for example, reduce the high days by one percentage and the low days by another percentage, choosing the percentages so that the new distribution achieves the new standard. At the opposite end of the spectrum from linear rollbacks, it is possible to meet a daily standard by "peak shaving." The peak shaving method would reduce all daily PM concentrations above a certain concentration to that concentration (e.g., the standard) while leaving daily concentrations at or below this value unchanged. While a strict peak shaving method of attaining a standard is unrealistic, it is illustrative of the principal that patterns different from a proportional rollback might be observed in areas attempting to come into compliance with revised standards.

If the short-term exposure concentration-response functions were exactly linear, then the overall estimated change in health effects associated with short-term exposure would depend only on the total change in PM concentration (above the lowest level at which PM pollution causes health effects). Because the concentration-response functions being considered are almost linear, the method by which daily PM concentrations are reduced to meet annual standards makes almost no difference.

However, the method by which daily concentrations are reduced to meet daily standards may make a sizeable difference, since it is the distribution of all the daily changes in air quality concentrations (above the lowest observed level at which PM pollution is associated with health effects) that results in the aggregate annual risk estimates. If one rollback method results in an air quality distribution with considerably more days with large changes in air quality than another method that also attains a given daily standard, the two methods will estimate significantly different health risks.

The estimated change in health effects based on short-term (daily) exposure concentration-response functions, then, is sensitive to the reduction method only to the extent that different reduction methods result in different total amounts of PM being removed from the atmosphere. Therefore, when the annual mean standard is the controlling standard (so a given total amount of PM must be removed), results should be relatively insensitive to different reduction methods (and would be totally insensitive to the reduction method if the concentration-response functions were exactly linear). When a daily standard is the controlling standard, however, results will be sensitive to different reduction methods.

Attainment of a set of standards was simulated by proportional rollback. That is, average daily PM concentrations were reduced by the same percentage on all days. Because pollution abatement methods are applied largely to anthropogenic sources of PM, rollbacks were applied only to PM above estimated background levels. (Rollbacks were estimated only for PM-2.5. Background PM-2.5 concentrations were estimated as 3.5 $\mu g/m^3$ in Philadelphia County, and 2.5 $\mu g/m^3$ in Southeast Los Angeles County. This is consistent with the approach of the Criteria Document.) The percent reduction was determined by the controlling standard. For example, suppose both an annual and a daily PM-2.5 standard are proposed. Suppose p_a is the percent reduction required to attain the annual standard, i.e., the percent reduction of daily PM above background necessary to get the annual average at the monitor with the highest annual average down to the standard. Suppose p_d is the percent reduction required to attain the daily standard with one exceedence, i.e., the percent reduction of daily PM above background necessary to get the second highest monitor-day down to the daily standard. If p_d is greater than p_a, then all daily average PM concentrations above background are reduced by p_d percent. If p_a is greater than p_d, then all daily average PM concentrations are reduced by p_a percent. Information on controlling monitors and percent rollbacks necessary to simulate attainment of alternative PM-2.5 standards in Philadelphia County and Southeast Los Angeles County is given in Exhibits 2.3 and 2.4.

Because the reduction method to attain a daily standard could have a significant impact on the risk analysis results, sensitivity analyses were conducted on different rollback methods for meeting proposed standards. The results of these analyses are presented in Section 8.

Exhibit 2.3. Controlling Monitors for Rollbacks to Attain Alternative PM-2.5 Standards

Monitor Site	Weighted Annual Average PM-2.5 Concentration*	Second Daily Maximum 24-Hour PM-2.5 Concentration*	Controlling Monitor
Philadelphia County			
N/E	15.5	65.1	
PBY	16.7	72.2	For daily standard
TEM	17.1	70.0	For annual standard
Southeast Los Angeles County			
Central LA	24.1	91.1	For annual standard
Diamond Bar	21.9	101.7	For daily standard

All concentrations are given in $\mu g/m^3$.*Both weighted annual averages and second daily maximum concentrations at the two monitors in Southeast Los Angeles County were adjusted to reflect attainment of the current PM-10annual standard of 50 $\mu g/m^3$ and the current PM-10 daily standard of 150 $\mu g/m^3$. These standards are currently attained in Philadelphia County.

Exhibit 2.4. Controlling Standards and Percent Rollbacks Necessary to Attain Alternative PM-2.5 Standards

Alternative PM-2.5 Standards		**Philadelphia County**	**Southeast Los Angeles County**
Annual Avg. Standard	24-Hour Standard	Controlling Standard and Percent Rollback*	Controlling Standard and Percent Rollback**
20 alone		----	Annual -- 18.8%
20	65	Daily -- 10.4%	Daily -- 37.0%
20	50	Daily -- 32.3%	Daily -- 52.1%
20	25	Daily -- 68.7%	Daily -- 77.3%
15 alone		Annual -- 15.5%	Annual -- 42.0%
15	65	Annual -- 15.5%	Annual -- 42.0%
15	50	Daily -- 32.3%	Daily -- 52.1%
15	25	Daily -- 68.7%	Daily -- 77.3%

All concentrations are given in $\mu g/m^3$.
*Based on controlling values for Philadelphia County of 17.1 $\mu g/m^3$ for the annual standard and 72.2 $\mu g/m^3$ for the daily standard.
** Based on controlling values for Southeast Los Angeles County of 24.1 $\mu g/m^3$ for the annual standard and 101.7 $\mu g/m^3$ for the daily standard.

The linear rollback methods described above to simulate attainment of alternative sets of PM-2.5 standards are also used to simulate attainment of current PM-10 standards prior to analyses in both the first and second phases of the risk analysis for Southeast Los Angeles County, which is out of attainment for current standards. Analyses for PM-10 in the first phase of the risk analysis use exactly the rollback method described above. Analyses for PM-2.5 in the first and second phases of the risk analysis assume that PM-2.5 is rolled back proportionately to PM-10 rollbacks. If, for example, daily PM-10 concentrations are decreased by 10 percent to simulate attainment of current PM-10 standards, it is assumed that daily PM-2.5 concentrations decrease by 10 percent as well. It is these adjusted PM-2.5 concentrations, the "as is" PM-2.5 concentrations in attainment of current standards, that are then reduced again by proportional rollback methods to simulate the attainment of alternative PM-2.5 standards in the second phase of the risk analysis. The adjustment of PM-10 and PM-2.5 concentrations in Southeast Los Angeles County to simulate attainment of current PM-10 standards is summarized in Exhibit 2.5.

Exhibit 2.5. Adjustment of PM Concentrations in Southeast Los Angeles County to Simulate Attainment of Current PM Standards

	Unadjusted Concentrations		Concentrations adjusted for compliance with current PM-10 standards (21.8 % rollback above background required to reduce second daily max. to the standard)	
	PM-10			
Monitor	Annual Mean	24-hr 2nd high	Annual Mean	24-hr 2nd high
CELA	51.7	195.2	41.8	154.0
DBAR	46.1	170.7	37.4	134.8
Controlling	51.7	195.2	41.8	154.0
	PM-2.5			
Monitor	Annual Mean	24-hr 2nd high	Annual Mean	24-hr 2nd high
CELA	30.1	115.7	24.1	91.1
DBAR	27.3	129.3	21.9	101.7
Controlling	30.1	129.3	24.1	101.7

All concentrations are given in $\mu g/m^3$.
The adjustment was performed assuming background concentrations of 6 $\mu g/m^3$ for PM-10, and 2.5 $\mu g/m^3$ for PM-2.5. Both the PM-10 annual mean and 2nd daily maximum PM-10 concentration exceed current standards: 50 $\mu g/m^3$ and 150 $\mu g/m^3$ respectively. Since levels of 54 $\mu g/m^3$ and 154 $\mu g/m^3$ are de facto considered to be in compliance, meeting the annual standard requires no rollback. However, meeting the daily standard requires a proportional rollback of (195.2-154.0)/(195.2-6.0) = 21.8% of air quality concentrations above background. (The 6.0 in the denominator takes into account that only concentrations above background are reduced.) This rollback was applied to the PM concentrations above 6 $\mu g/m^3$, and to the PM-2.5 concentrations above 2.5 $\mu g/m^3$.

2.3. The concentration-response function and estimation of health effect incidence changes

The concentration-response functions used in this risk analysis are empirically estimated relationships between average ambient concentrations of the pollutant of interest (PM-10 or PM-2.5) and the health endpoints of interest (e.g., mortality or hospital admissions) reported by epidemiological studies.[5] The choice of studies is discussed in Section 5. In some cases, separate risk analyses were performed using the concentration-response functions from separate studies; in other cases, only a "pooled function" derived from several studies of the same health endpoint was used.

[5]Although, as noted above, epidemiological studies might prefer to estimate individual exposure-response relationships, because of the lack of individual exposure data, these studies typically estimate the ambient concentration-response relationship instead. Although this is not necessarily the relationship of ultimate interest to health scientists, it is the relationship that is appropriate to use in the PM risk analysis with ambient PM data.

Although some epidemiological studies estimated linear concentration-response functions, most of the studies used a method referred to as "Poisson regression" to estimate exponential concentration-response functions in which the natural logarithm of the health endpoint is a linear function of PM[6]:

$$y = B\, e^{\beta x} \quad , \qquad \textbf{(1)}$$

where x is the ambient PM level, y is the incidence of the health endpoint of interest at PM level x, β is the coefficient of ambient PM concentration, and B is the incidence at x=0, i.e., when there is no ambient PM. The relationship between a specified ambient PM level, x_0, for example, and the incidence (rate) of a given health endpoint associated with that level (denoted as y_0) is then

$$y_0 = B\, e^{\beta x_0} \quad . \qquad \textbf{(2)}$$

Because the exponential form of concentration-response function (equation (1)) is by far the most common form, the discussion that follows assumes this form. However, because the coefficients estimated by the epidemiology studies are extremely small, these exponential functions are nearly linear. The consequences of this near-linearity are discussed below.

Ambient PM levels may be based on any averaging time, e.g., they may be daily averages or annual averages, as long as the health effect incidence corresponds to the PM averaging time. For example, the concentration-response function may describe the relationship between daily average ambient PM concentrations and daily mortality, or it may describe the relationship between annual average ambient PM concentrations and annual mortality. Some concentration-response functions were estimated by using moving averages of PM to predict daily health effects incidence. Such a function might, for example, relate the incidence of the health effect on day t to the average of PM concentrations on days t and (t-1). (This may be considered a variant on the short-term, or daily concentration-response function.) The discussion below does not indicate averaging times and simply assumes that the measure of health effect incidence, y, is consistent with the measure of ambient PM concentration, x.

The change in health effects incidence, $\Delta y = y_0 - y$, from y_0 to the baseline incidence rate, y, corresponding to a given change in ambient PM levels, $\Delta x = x_0 - x$, can be derived from equations (1) and (2) (as shown in Appendix 3) as

$$\Delta y = y[e^{\beta \Delta x} - 1] \; . \qquad \textbf{(3)}$$

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[6]Poisson regression is essentially a linear regression of the natural logarithm of the dependent variable on the independent variable, but with an error structure that accounts for the particular type of heteroskedasticity that is believed to occur in health response data. What matters for the risk analysis, however, is simply the form of the estimated relationship, as shown in equation (1).

Alternatively, the change in health effects incidence can be calculated indirectly using relative risk. Relative risk (RR) is a well known measure of the comparative health effects associated with a particular air quality comparison. The risk of mortality at ambient PM level x_0 relative to the risk of mortality at ambient PM level x, for example, may be characterized by the ratio of the two mortality rates: the mortality rate among individuals when the ambient PM level is x_0 and the mortality rate among (otherwise identical) individuals when the ambient PM level is x. This is the relative risk for mortality associated with the difference between the two ambient PM levels, x_0 and x. Given a concentration-response function and a particular change in ambient PM levels, Δx, the relative risk associated with that change in ambient PM, denoted as $RR_{\Delta x}$, is equal to $e^{\beta \Delta x}$. The change in health effects incidence, Δy, corresponding to a given change in ambient PM levels, Δx, can then be calculated based on this relative risk:

$$\Delta y = y(RR_{\Delta x} - 1) . \quad \textbf{(4)}$$

Equations (3) and (4) are simply alternative ways of writing the relationship between a given change in ambient PM levels, Δx, and the corresponding change in health effects incidence, Δy. The derivation of equation (4) is shown in Appendix 3. These equations are the key equations that combine air quality information, concentration-response information, and baseline health effects incidence information to estimate ambient PM health risk.

Given a concentration-response function and air quality data (ambient PM values) from a sample location, then, the change in the incidence of the health endpoint ($\Delta y = y_0 - y$) corresponding to a change in ambient PM level of $\Delta x = x_0 - x$ is determined. This can either be done with equation (3), using the coefficient, β, from a concentration-response function, or with equation (4), by first calculating the appropriate relative risk from the concentration-response function.

Because the estimated change in health effect incidence, Δy, depends on the particular change in PM concentrations, Δx, being considered, the choice of PM concentration change considered is important. These changes in PM concentrations are generally reductions from the current levels of PM ("as is" levels) to some alternative, lower level(s).

If a location is not in attainment of current PM standards, as is the case in Southeast Los Angeles County, current levels may be characterized in two ways. It may be of interest to compare health effects at "as is" PM concentrations (that do not attain the current standards) with health effects at some alternative PM level(s). Alternatively, it is also of interest to compare health effects at those PM concentrations that just attain the current PM-10 standards with health effects at those PM concentrations that just attain some alternative (PM.2.5) standards. This is an appropriate context for examining the potential risk reductions associated with revising the current standard.

The first and second phases of the risk analysis are distinguished primarily by the choice of lower PM level(s). The second phase considers the changes in health effects

incidence associated with changes from PM concentrations that meet the current standards to PM concentrations that just meet alternative PM-2.5 standards.

When possible, the choice of lower PM level(s) in an analysis in the first phase of the risk analysis is the lowest PM concentration observed in the study that estimated the concentration-response function used in the risk analysis. This is the lowest PM concentration at which the concentration-response function is supported. However, many of the short-term exposure PM studies do not report the lowest observed PM concentration. (For example, many studies report the lowest decile or quartile values.) When the lowest observed PM concentration is not reported (or if it is lower than background level), analyses in the first phase of the risk analysis consider the range of "as is" PM concentrations in the sample location down to background PM concentration in that location.

In contrast to most short-term exposure studies, long-term exposure studies routinely report the lowest observed annual average PM concentration. Risk analyses that use long-term exposure concentration-response functions therefore consider the range of "as is" annual average PM in the sample location to the lowest annual average PM level observed in the study.

In both phases of the risk analysis, the ambient PM concentrations to which "as is" ambient PM concentrations are compared are generally lower than or equal to "as is" concentrations. Therefore $\Delta x = x_0 - x$ is negative (or zero), and so the corresponding change in incidence of health effects, Δy, is also negative (or zero). That is, there are fewer cases of any given health effect at lower ambient PM levels. Alternatively, $-\Delta y$ may be interpreted as the health effects attributable to PM concentrations between x_0 and x.

Because different epidemiological studies report different estimated concentration-response functions for a given health endpoint, predicted changes in health effects incidences depend on the concentration-response function used. The uncertainty introduced into the risk analyses by this is assessed both through sensitivity analyses and through Monte Carlo methods (see Section 9).

2.4. Calculating the aggregate health effects incidence on an annual basis from the changes in daily health effects incidence

To assess the *daily* health impacts of daily average ambient PM levels above background or above the levels necessary to achieve a given standard, concentration-response functions from short-term exposure studies were used together with estimated changes in daily ambient PM concentrations to calculate the daily changes in the incidence of the health endpoint. Adding these changes over all the days in a year yields the annual change. (Alternative assumptions about the range of PM levels associated with health effects were explored in sensitivity analyses. When a minimum concentration for effects is considered, reductions below this concentration do not contribute attributable cases to the calculation. Only reductions down to this concentration contribute attributable cases to the calculation.)

After daily changes in health effects are calculated, an annual change is calculated by summing the daily changes. However, there are some days for which no ambient PM concentration information is available. The predicted estimated risks, based on those days for which air quality data are available, must be adjusted to take into account the full year.

In Philadelphia County, there are very few missing days, and these are distributed evenly throughout the year. In this case, the adjusted health effects incidence is the original incidence multiplied by the number of days in a year and divided by the number of days for which data are available; that is, the figure is simply scaled for the fraction of days on which there are data. In Philadelphia County, for example, PM-10 data are available for 358 days evenly distributed throughout the year. Suppose the sum of the daily premature deaths associated with PM on those 358 days is 600, then the adjusted figure is 612 (i.e., 600 x 365/358). This reflects the assumption that the distribution of PM concentrations on those days for which data are available accurately reflects the distribution of ambient PM concentrations for the entire year, and that the concentration-response functions were estimated using data from the entire year.

In Southeast Los Angeles County, however, the distribution of missing days varies significantly in different periods of the year. During the first quarter of 1995, air quality monitoring was done on roughly one in six days; during the second quarter, it was done on roughly one in three days; and during the third and fourth quarters, it was done almost every day. Because of this, adjustments were made separately in each quarter, and the results added. Adjustment of health effects incidence within a quarter in Southeast Los Angeles County was done with the same method used to adjust health effects incidence throughout the year in Philadelphia County.

Some concentration-response functions are based on average PM levels during several days. When these concentration-response functions are used, the air quality data are averaged for the same number of days. For example, a function based on two-day averages of PM would be used in conjunction with two-day averages of PM in the sample location to predict the incidence of the health effect in that location. In some cases, intervals of three or more consecutive days in a given location are missing data, and so no multi-day average is available for use with multi-day concentration-response functions. These cases were treated by multi-day functions just as individual missing days were treated by single-day functions: they contributed no cases to the risk analysis, and figures were adjusted for the days on which multi-day averages were missing.

Concentration-response functions from long-term exposure studies were used to assess the annual health impacts of changes in annual average ambient PM concentrations. In this case, the “as is” annual concentration is simply the average concentration for those days on which data are available, if missing days are evenly distributed throughout the year (as in Philadelphia County), or a composite of quarterly averages if missing days are not evenly distributed throughout the year (as in Southeast Los Angeles County).

Note that while the long-term exposure studies use annual average PM concentration as the PM indicator, the studies were conducted in such a way that they may have detected effects due to PM exposure over some longer period. For example, average PM concentrations over the course of five years might be the appropriate measure. It is therefore possible that the full benefits of reducing PM predicted by these studies would not appear in the first year after reductions to attain a standard, but would be "phased in" gradually as concentrations during successive years were also reduced. If average PM concentrations over five years is the appropriate measure, for example, the benefits of a standard would gradually increase to their full level over the course of the five years after the new standard had been attained. The risk analysis makes no attempt to determine the appropriate exposure period for long-term exposure studies. The estimated annual benefits of reduced long-term exposure are assumed to be completely achieved by the future year for which attainment of the new standard is being modeled. The issue is partially addressed, however, in a sensitivity analysis which examines the effect of altering the "slope" parameter in the long-term exposure concentration-response function.

2.5. Baseline health effects incidence data

Baseline health effects incidence rates (e.g., death rates) and population sizes (to calculate baseline incidence levels) for the selected locations were obtained from vital statistics sources. Location-specific information was used whenever possible. However, location-specific baseline incidence data for hospital admissions and other morbidity endpoints are not as readily available as for mortality from national data sources. Where possible, local sources of data (e.g., from city, county or state health agencies) were obtained. However, such data are not uniformly available, and alternative procedures were used in some instances. For respiratory symptom or illness health endpoints, routine surveillance and reporting is not generally conducted in metropolitan areas, in contrast to the data gathered on mortality and hospital admissions. For these endpoints, estimates of baseline incidence were derived from the studies themselves to provide what should be viewed as only a rough estimate of magnitude of potential effects, given the much greater degree of uncertainty concerning baseline incidence information for these endpoints. The baseline health effects incidence data are presented and discussed more fully in Section 6.

2.6. Sensitivity analyses

The predictions of the risk analyses depend on the input components discussed above. Changes in the values of these input components change the predictions of the analyses. This is an important issue in risk analysis because the true values of parameters necessary for such analyses, e.g., the location-specific concentration-response relationships and the location-specific baseline health effects incidence rates, are often not known exactly and must be estimated. The sensitivity of the results of an analysis to changes in the values of the input

components (or in assumptions or procedures that affect these values) is therefore an important consideration.

The uncertainty associated with having to estimate parameter values can be assessed by uncertainty analyses, in which the probability distribution of values for an input component is estimated, and the resulting distribution of possible outcomes is assessed. Uncertainty analyses to assess the uncertainty associated with key parameters of the risk assessment model, focusing primarily on the concentration-response function, are discussed in Section 9.

Alternatively, sensitivity analyses can be used to illustrate the sensitivity of analysis results to different possible input values or to different assumptions or procedures that may affect these input values. Although a sensitivity analysis is not as comprehensive as an uncertainty analysis, selecting only a few possible alternative values of an input component rather than characterizing the entire distribution of these values, it is precisely the simplicity of a sensitivity analysis that makes it preferable for illustrating the impact on results of using different input component values. Exhibit 2.6 lists the sensitivity analyses carried out for each of the two phases of the risk analysis. The results of those sensitivity analyses pertaining to the first phase of the risk analysis (the "as is" analyses) are presented in Section 7; the results of those sensitivity analyses pertaining to the second phase of the risk analysis (the alternative standards analyses) are presented in Section 8.

Exhibit 2.6. Sensitivity Analyses

Sensitivity analyses associated with the "as is" risk analyses:
1. Sensitivity analysis of the effect of alternative assumed background levels on predicted health effects associated with "as is" PM (PM-10 and PM-2.5) above background.
2. Sensitivity analysis of the effect of using alternative "hockey stick" models on predicted short-term exposure health effects associated with "as is" PM concentrations above specified model cutpoints.
3. Sensitivity analysis of the effect of alternative cutpoints (PM levels below which health effects incidence is not considered) on predicted long-term exposure health effects associated with "as is" PM above cutpoint.*
4. Sensitivity analysis of the effect of combining different averaging times in pooled short-term exposure mortality concentration-response functions on predicted health effects associated with "as is" PM-10 concentrations above background.*
5. Sensitivity analysis of the effect of using concentration-response functions for short-term mortality from different individual studies on predicted health effects associated with "as is" PM-10 and PM-2.5**
6. Sensitivity analysis of the effect of copollutants in the concentration-response model on the predicted relative risk for a change in PM-10 concentration of 50 $\mu g/m^3$ and a change in PM-2.5 concentration of 25 $\mu g/m^3$.
7. Sensitivity analysis of the effect of copollutants in the concentration-response model on the predicted health effects associated with "as is" PM above background.
8. Sensitivity analysis of the effect of historical previous air quality on estimated mortality associated with long-term exposure to PM-2.5.*
Sensitivity analyses associated with the alternative standards analyses:
9. Sensitivity analysis of the effect of different background levels on rollbacks required to simulate attainment of alternative PM-2.5 standards.
10. Sensitivity analysis of the effect of different rollback methods to simulate attainment of alternative PM-2.5 standards.*

*Sensitivity analysis done for Philadelphia County only. With the exception of sensitivity analysis number 6, which is not specific to any location, other analyses were done for both Philadelphia County and Southeast Los Angeles County (see Exhibit 7.5).

**A preliminary (unpublished) study of short-term exposure mortality has been conducted in Philadelphia for PM-2.5 (Dockery et al., Abstr., 1996). The results of this study are compared with the results obtained by using the pooled analysis function separately in Section 7. This study is not included, however, among the studies in this sensitivity analysis and is not included in the main results because it is not yet published.

3. Assumptions and Caveats

To carry any risk analysis to completion in the face of incomplete information, it is necessary to make a variety of assumptions. The necessity of making simplifying assumptions characterizes most scientific analyses, because analysis is usually performed with only limited information. Some of the assumptions necessary in the risk analyses are assumptions generally made in scientific analyses (for example, that the model used to describe the relationship between variables does accurately describe this relationship). Other assumptions are specific to these risk analyses. (Assumptions may be characterized instead as caveats: the validity of the results of the analysis depend in part on the extent to which the underlying assumptions are valid.)

The risk analyses discussed in this report are only as good as the inputs to the analyses -- that is, the concentration-response functions, the air quality data, the health effects incidence rates, and the population sizes. The quality of each component is stated as an assumption or, alternatively, discussed as a caveat. Other assumptions/caveats concerning how each of the three analysis components are used in the risk analyses are discussed below in turn. For many of the uncertainties, it is not known whether the factors discussed might lead to over- or under-estimates of risk. Exhibit 3.1 summarizes some of the key uncertainties in the risk analysis, which are discussed in more detail below.

3.1. Concentration-response functions

The concentration-response function is a key element of risk assessment. The quality of the risk analysis depends, in part, on (1) how well the concentration-response functions used in the risk analyses have been estimated (e.g., whether they are unbiased estimates of the relationship between the health response and ambient PM concentration in the study locations), (2) how applicable these functions are to locations and times other than those in which they were estimated, and (3) the extent to which these relationships apply beyond the range of the PM concentrations from which they were estimated. These issues are discussed in the subsections below.

3.1.1. Accuracy of the estimates of concentration-response functions

The adequacy of the estimation of the relationships between PM and various health endpoints in epidemiological studies has received considerable attention. A significant portion of this attention has focused on the issue of using average ambient PM concentration as a measure of actual exposure to PM. Although they might prefer to estimate the individual exposure-response relationship, such studies are actually estimating the concentration-response relationship, as discussed in Section 1. Concern that this practice may produce biased estimates of individual exposure-response relationships may be valid. However, because the risk analysis examines the association between changes in health effects incidence and changes in ambient PM concentrations, (ambient) concentration-response functions, rather than

Exhibit 3.1. Key Uncertainties in the Risk Analysis

Uncertainty	Direction of Potential Error	Comments
Empirically estimated concentration-response relationships	?	Statistical association does not prove causation. Because concentration-response functions are empirically estimated, there is uncertainty surrounding these estimates. Omitted confounding variables could cause upward bias.
Functional form of concentration-response relationship	?	Statistical significance of coefficients in an estimated concentration-response function does not necessarily mean that the mathematical form of the function is the best model of the true concentration-response relationship.
Transferability of concentration-response relationships	?	Concentration-response functions may not be valid in times and places other than those in which they were estimated.
Extrapolation of concentration-response relationships beyond observed data range	+	A concentration-response relationship estimated by an epidemiological study may not be valid at concentrations outside the range of concentrations observed during the study.
Adequacy of PM characterization	?	Only particle mass per unit volume has been considered, and not, for example, chemical composition or any other particle characteristics.
Accuracy of PM mass measurement	?	Possible differences in measurement error, losses of particular components, and measurement method between the two risk analysis locations and between these locations and the original studies would be expected to add uncertainty to quantitative estimates of risk.
Adjustment of air quality distributions to reflect attainment of proposed alternative standards	?	There is uncertainty in the pattern and extent of reductions in daily PM concentrations that would take place to attain proposed standards.
Baseline health effects data	?	Data may not be exactly appropriate for a variety of reasons. For example, location- and age-group-specific baseline rates may not be available in all cases. Baseline incidence may change over time for reasons unrelated to PM.
Sensitive subgroups	?	Populations in the sample locations may have more or fewer members of sensitive subgroups than locations in which functions were derived. Thus functions might not be appropriate. (This is a subset of the uncertainty associated with transferring concentration-response functions from one location to another (see above).
Omitted effects	-	Some health effects caused by PM may have been omitted.

individual exposure-response functions, are relevant to the analysis discussed in this report. The important question here, then, is whether epidemiological studies have produced accurate, unbiased estimates of ambient concentration-response functions.

The accuracy of an estimate of a concentration-response function reported by a study depends on the study design. The Criteria Document has evaluated the substantial body of PM epidemiological studies. In general, critical considerations in evaluating the design of an epidemiological study include the adequacy of the measurement of average ambient PM, the adequacy of the health effects incidence data, and the consideration of potentially important health determinants and causal factors such as:

- copollutant air quality;
- exposure to other health risks, such as smoking and occupational exposure;
- demographic characteristics, including age, sex, socioeconomic status, and access to medical care; and
- population health status independent of PM air quality.

Other specific characteristics of concern depend on the health endpoints in the studies. Study selection for the risk analysis was guided by the evaluations in the PM Criteria Document (EPA, 1996a).

Concentration-response functions may not be identical for all members or all subgroups of a population; however, the concentration-response functions used in the risk analysis reflect overall population responses at air quality levels similar to those found in the sample locations (see Section 3.1.3).

To the extent that the studies did not address all critical factors, the concentration-response functions may be limited. They may result in either over- or underestimates of risk associated with ambient PM concentrations in the locations in which the studies were done. It is possible, then, that their application to the sample locations in the risk analyses might also have resulted in biased estimates of risk in those locations.

One possible source of bias in the estimation of concentration-response functions warrants special note. A concentration-response function could be biased if the measurement of average ambient PM concentration is inaccurate in a systematic way. Most epidemiological studies use the average PM levels reported at some number of PM monitors as the measure of the average ambient PM concentration (which, for the purposes of the studies, is itself a surrogate for individual exposure to PM). This may or may not yield accurate measurements of the actual daily average ambient PM concentrations in the study city. Depending on how the monitors are placed, it could yield systematically inaccurate, or biased measurements. What is important for the purpose of the risk analysis, in this case, is that the measurement of daily average ambient PM concentrations in the sample location be biased in the same way as in the study city. That is, a systematic bias in the measurement of daily average ambient PM concentrations in the study city is not a problem for the risk analysis if there is the same

systematic bias in the measurement of daily average ambient PM concentrations in the sample location. Whether this is the case, however, is unlikely to be known.

3.1.2. Applicability of concentration-response functions in different locations

The method described in Section 2 combines PM data from a single year in a specific location (the sample location) with a concentration-response relationship estimated by an epidemiological study to predict the change in incidence of a given health endpoint associated with a given change in ambient PM concentrations in that location. Preferably, this concentration-response function is obtained from a study conducted in the sample location itself. However, if no such study is available, a concentration-response function derived from a study that was performed in a different location (or a pooled analysis concentration-response function derived from several such studies) is used. The precision of these predictions therefore rests in part on the "transferability" of the concentration-response relationship from one location to another. That is, it rests on the assumption that the relationship between ambient PM (either PM-10 or PM-2.5) and a given health endpoint is the same in the two locations.

The relationship between average ambient PM concentration and the incidence of a given health endpoint, the concentration-response relationship, depends on (1) the relationship between individual exposure and average ambient PM concentration and (2) the relationship between the health response and individual exposure (as described formally in Appendix 1). One or both of these relationships may depend on the exposed population (for example, the extent of susceptible subgroups) and/or the composition of the PM and other air quality indicators to which the population is exposed. Both the population and the composition of PM and other air quality indicators could vary significantly from one location to another. In this case, the concentration-response relationship could vary significantly from one location to another as well. There are various reasons why one or both of the relationships upon which the concentration-response relationship depends might vary from one location to another.

The relationship between individual exposure and average ambient PM concentration might differ among locations if people's behavior patterns differ significantly from one location to another. Suppose, for example, people in the study city spend a lot more time outdoors than people in the sample location. Suppose also that ambient (outdoor) concentrations of PM are greater in both locations than indoor concentrations. Then a given ambient PM concentration will be associated with higher individual exposures among people in the study location than among people in the sample location.

Suppose, alternatively, that coarse particles are less likely to infiltrate indoor air. In this case, PM-10 with a high proportion of coarse particles would result in lower indoor exposure than PM-10 with a high proportion of fine particles. If the percent of time spent indoors is the same in different locations, then the location with the coarser PM-10 would have lower individual exposure to PM-10 than the location with the finer PM-10.

The relationship between the health response and individual exposure might differ among locations if, for example, the population in one location has a higher proportion of a susceptible subgroup than another location. Closely matching populations used in studies to the populations of the sample locations is not possible for many characteristics (for example, smoking status, workplace exposure, socioeconomic status, and the prevalence of highly susceptible subgroups).

Alternatively, the PM-10 in one location may be largely fine particles (PM-2.5), whereas in another location it may be made up predominantly of coarse particles. If PM-2.5 is more potent than coarse particles in causing the health effect, then there will be a greater incidence of the health effect corresponding to a given level of individual exposure to PM-10 in the first location, all else equal, than in the second location (see Appendix 4).

Other pollutants, such as carbon monoxide and ozone, may also play a role in causing health effects, either independently or in combination with PM. Interlocational differences in these pollutants could also induce differences in the concentration-response relationship between one location and another.

In summary, the concentration-response relationship in one location may not be the same as the concentration-response relationship in another location. Even if the relationship between the health response and individual exposure is the same in both locations, the relationship between individual exposure and ambient concentrations may differ between the two locations. Similarly, even if the relationship between individual exposure and ambient concentrations is the same in both locations, the relationship between the health response and individual exposure may differ between the two locations. In either case, the concentration-response relationship would differ.

Instead of a single concentration-response function that characterizes the relationship between ambient PM (either PM-10 or PM-2.5) and a given health endpoint everywhere in the United States, then, a more realistic model may be a *distribution* of concentration-response functions, or, equivalently, a distribution of values of the parameter β in the concentration-response function. If concentration-response functions were available for all health endpoints for each of the sample locations, the precision of the risk analyses would be improved. The necessity of applying concentration-response functions estimated in locations other than the sample locations for which risk is being analyzed introduces uncertainty into the results of the risk analyses. This is particularly apparent in the case of mortality, for which there are many estimated concentration-response functions. The degree of this uncertainty is assessed by Monte Carlo methods, as described in Section 9.

The uncertainty associated with the application of concentration-response functions from epidemiological studies to the sample locations is nonetheless smaller than the uncertainty associated with the use of concentration-response functions from animal studies. Such studies are often used in risk assessments, especially when epidemiological results are not available, but are not used in the risk analysis described in this report.

3.1.3. Extrapolation beyond observed air quality levels

Although a concentration-response function describes the theoretical relationship between ambient PM and a given health endpoint for all possible PM levels (down to zero), the estimation of a concentration-response function is based on real ambient PM values that are limited to the range of PM concentrations in the location in which the study was conducted. The actual shape of the concentration-response function is not known outside the observed air quality range. Several of the mortality studies discussed in the Criteria Document (EPA, 1996a), including Pope et al.(1992), reported measured PM-10 levels as low as 4 $\mu g/m^3$. Nonetheless, the concentration-response relationship may be less certain towards the lower end of the concentration range if few days had such low concentrations.

The risk analyses assume that the estimated concentration-response functions adequately represent the true concentration-response relation down to background levels in the sample locations, in cases in which this background level is above the lowest concentrations used to derive the concentration-response functions. For studies in which the lowest concentrations studied are likely to be above background (e.g., the long-term exposure study of Pope et al. 1995) estimates of risk are not generated for concentrations below the minimum concentrations observed in the studies. The estimates of risk for the lowest concentrations considered are more uncertain than the estimates for concentrations in the middle of the range of study data.

The concentration-response relationship may also be less certain towards the upper end of the concentration range being considered in a risk analysis, particularly if the PM concentrations in the sample location exceed the PM concentrations observed in the study location. Even though it may be reasonable to model the concentration-response relationship as exponential over the ranges of PM concentrations typically observed in epidemiological studies, it is unlikely to be exponential over a very wide range of PM levels.[7] Rather, at very high PM concentrations, the concentration-response function is likely to begin to flatten out. In a location such as Los Angeles, where pollution levels are generally higher than average in the United States, and possibly substantially higher, it is possible that some PM concentrations fall in the range in which the exponential function is no longer appropriate. To the extent that this is the case, it would contribute to overestimation of PM-related health effects incidence in such a location.

The standards that EPA has chosen to evaluate in the second phase of the risk analysis lie in the middle range of pollution levels observed in epidemiological studies. Applying uniform linear rollbacks to the concentration distributions in the sample locations, however, will result in some modeled PM concentrations well below these levels. It is possible that there is a minimum concentration below which PM is not associated with health effects. To the extent that reducing concentrations below such a concentration is counted as reducing PM-

[7]Although the concentration-response functions are exponential, they are practically linear. It is still unlikely, however, that a linear function is appropriate over a very wide range of PM concentrations.

related health effects, the health benefits attributed in the risk analyses to reducing PM are overestimated. The degree of overestimation depends on the frequency of modeled PM concentrations that are lower than the lowest level at which effects actually occur, and how much lower the modeled PM concentrations are. Sensitivity analyses address the sensitivity of the results of the risk analysis to different assumptions about the minimum concentration at which health effects occur.

3.2. The air quality data

3.2.1. Appropriateness of the PM indicator

The ozone air quality risk analysis modeled people's activity patterns and their resulting exposure to different ozone micro-environments. This was of interest because controlled experiments have measured people's reactions to varying ozone concentrations. No such controlled experiments for PM were used in the PM risk analyses. Instead, the PM risk analysis used estimates of average ambient air quality in a given location as measured at monitors. This matches the measure of exposure used in PM epidemiology studies.

PM is measured in units of mass per unit volume, typically in micrograms per cubic meter, rather than in density of particles (i.e., in parts per million). The only distinction made between different kinds of particles in the risk analysis is one of size: PM-2.5 vs. PM-10. This may not be the only distinction of interest. The chemical composition of PM, for example, was not considered in any of the risk analyses (as it was not in most of the epidemiological studies used in these analyses).

3.2.2. Adequacy of PM air quality data

The method of averaging data from monitors across a metropolitan area in the risk analysis is similar to the methods used to characterize ambient air quality in most of the epidemiology studies. The important issue, however, is whether any biases in the measurement of average daily ambient PM concentrations in the study location are matched in the sample location, as discussed in Section 3.1.1. Ideally, the measurement of average daily ambient PM concentrations in the study location are unbiased. In this case, unbiased risk predictions in the sample location depend, in part, on an unbiased measurement of average daily ambient PM concentrations in the sample location as well. If, however, the measurement of average daily ambient PM concentrations in the study location are biased, unbiased risk predictions in the sample location are still possible if the measurement of average daily ambient PM concentrations in the sample location incorporate the same bias as exists in the study location measurements. Because this is not known, however, the adequacy of the PM measurements in the sample locations is a source of uncertainty in the risk analysis.

PM air quality data are not available for all days of the year chosen for risk analysis in either of the sample locations. The change in the incidence of a health effect over the course of the year corresponding to a given change in daily PM levels is calculated based on the

assumption that PM levels on those days with PM data are representative of levels on those days without PM data (see Section 2 for an explanation of the method of extrapolating changes in health effects incidence to an entire year). Where available concentration data are evenly distributed throughout the year, the extrapolation can be performed in a single step. Where available concentration data are unevenly distributed through the year, results from different parts of the year are scaled separately, and the results added. This avoids bias due to seasonal differences in average PM levels and monitoring frequencies.

Because the PM data in each sample location are limited to a specific year, the results of the risk analyses are generalizable to the present only to the extent that ambient PM levels in the available data are similar to current ambient PM levels. A substantial difference between PM levels in the years used in the risk analyses and current PM levels could imply a substantial difference in predicted incidences of health effects. This is not expected to be a large problem, however, because adequate PM-10 and PM-2.5 monitoring data are available for Philadelphia County for 1992-1993 and for Southeast Los Angeles County for 1995.

3.3. Baseline health effects incidence rates

3.3.1. Quality of incidence data

Local incidence data were obtained for mortality and for hospital admissions for both Philadelphia and Los Angeles (see Section 6). This is clearly preferable to using nonlocal data, such as national incidence rates. As with any health statistics, however, misclassification of disease, errors in coding, and difficulties in correctly assigning residence location are potential problems. These same potential sources of error are present in most epidemiological studies. In most cases, the reporting institutions and agencies utilize standard forms and codes for reporting, and quality control is monitored.

When national rates are used, the estimated rates are generally considered reliable, due to the large sample size available. As the source population becomes smaller and the event rarer, the reliability may decrease, due the infrequency of occurrence. Most endpoints considered in this report are common occurrences and the locations are sufficiently large, however, that the statistics reported are likely to be adequately representative of the occurrences, even though the data are limited to one year.

Incidence rates for some health endpoints (in particular, for lower respiratory symptoms) were obtained from the studies reporting the concentration-response functions. There is greater uncertainty in the application of incidence data obtained from specific studies to locations across the country, because the rates are specific to a certain location, time, and cohort. Where possible, baseline incidence rates were obtained for the age cohorts matching the populations studied. In addition, some rates taken from studies (for example, lower respiratory symptoms from Pope et al. 1991) are age-specific. Since actual rates for some endpoints (including lower respiratory symptoms) are known to vary with age, rates for some

age cohorts may not be accurately represented. For example, it is likely that lower respiratory symptom rates are underestimated for young children.

Regardless of the data source, if actual incidence rates are higher than the incidence rates used risks will be underestimated. If incidence rates are lower than the incidence rates used, then risks will be overestimated. For most of the concentration-response functions, the incidence rates affect the estimation of the changes in the number of cases associated with changes in PM, but not the estimation of the percentage changes in PM-related cases. The uncertainties in identifying the correct baseline incidence rates therefore affect only one portion of the results.

Both morbidity and mortality rates change over time for various reasons. One of the most important of these is the age distribution of the population. The old and the extremely young are more susceptible to many health problems than is the population as a whole. The most recent available data were used in the risk analysis. However, the average age of the population in many locations will increase as the post-WWII children age. Consequently, the baseline incidence rates for some endpoints may rise, resulting in an increase in the number of cases attributable to any given level of PM pollution. Alternatively, areas which experience rapid in-migration, as is currently occurring in the south and west, may tend to have a decreasing mean population age and corresponding changes in incidence rates and risk. Although temporal changes in incidence are relevant to both morbidity and mortality endpoints, however, the most recent available data were used in all cases, so temporal changes are not expected to be a large source of uncertainty.

3.3.2. Lack of daily health effects incidence rates

Both ambient PM levels and the daily health effects incidence rates corresponding to ambient PM levels vary somewhat from day to day. Those risk analyses based on concentration-response functions estimated by short-term exposure studies calculate daily changes in incidence and sum them over the days of the year to predict an annual change in health effect incidence.

Most of the concentration-response functions calculate relative risk, that is, *percent* change in health effects, which depends only on the *change* in PM levels (and not the actual value of either the initial or final PM concentration). This percent change is multiplied by a baseline incidence in order to determine the change in health effects incidence. That is,

$$\Delta\ incidence = baseline\ incidence\ (PM) * RR(\Delta PM),$$

where the relative risk (RR) depends only on the change in PM levels (ΔPM), not the actual values, and the baseline incidence may depend on the actual PM concentrations.

If PM does indeed affect health, then actual incidence rates can be expected to be somewhat higher than average on days with high PM concentrations, and somewhat lower than

average on days with low PM concentration. However, only annual average incidence rates are available from vital statistics sources. Daily incidence rates corresponding to "as is" daily PM levels were therefore approximated by the average daily incidence rates, calculated from the annual figures:

$$\Delta\ incidence\ =\ average\ baseline\ *\ RR(\Delta PM).$$

The annual average baseline incidence rate is expected to be lower than the actual baseline incidence rate on days with high PM, and so the predicted change in incidence will also be lower than it should be. Similarly, the annual average baseline incidence is expected to be higher than the actual baseline incidence rate on days with low PM, and so the predicted change in incidence will also be higher than it should be. The change in health effects incidence may therefore be slightly underestimated on days with high PM levels and slightly overestimated on days with low PM levels. Both effects are small, however, and should largely cancel one another.

3.4. Further caveats

3.4.1. Highly susceptible subgroups

Highly susceptible subgroups, such as asthmatics and people with cardiovascular or pulmonary disease, are of particular concern with regard to PM pollution. These groups comprise, presumably, a substantial portion of hospital admissions, deaths, and morbidity counts enumerated in this risk analysis. To the extent that a location has a larger (smaller) proportion of people in these groups than the location in which a concentration-response function was estimated, however, risk may be underestimated (overestimated). This is one of the reasons that concentration-response functions may not be transferable, as discussed in Section 3.1.2. In addition, the health effects experienced by a susceptible subgroup may be much greater than those experienced by the population at large. Given the lack of data on the representation of various potentially susceptible subgroups in specific locations, this question cannot be addressed with certainty given available data.

3.4.2. Possible omitted health effects

Although this risk analysis method considers both mortality and a variety of morbidity health effects, it does not include all health effects which may result from PM exposure. Only a subset of those endpoints that have been the subject of quantitative epidemiological studies are enumerated. Other possible health effects reported to be associated with short-term exposures to PM-10 include emergency room visits for asthma (Schwartz et al. 1993), respiratory hospitalization in children (Pope et al. 1991), school absences (Ransom and Pope 1992), symptoms of cough (Schwartz et al. 1994; Ostro et al. 1991), and asthma medication usage (Pope et al. 1991). Other possible health effects reported to be associated with short-term exposures to PM-2.5 include respiratory-related restricted activity days and work loss days in adults (Ostro et al. 1987). Health effects that have been associated with long-term

exposures to PM-10, not included in the risk analysis, are chronic bronchitis in adults (Abbey et al. 1995a) and decreased lung function in children (Raizenne et al. 1996). Other possible effects of concern include cardiovascular and respiratory episodes and diseases not measured in the hospitalization studies, and effects in those under 65 for health effects for which only studies on those over 65 are available. The omission of some endpoints may lead to an underestimate of total risk to the population.

4. Air Quality Assessment: The PM Data

This section describes the PM-10 and PM-2.5 data for Philadelphia County and Southeast Los Angeles County used in the risk analysis. Average ambient PM-10 concentration in a sample location on a given day is represented by the *average* of reported PM-10 levels at the different monitors in that location on that day. The same approach is used for PM-2.5. This approach is consistent with what has been done in epidemiological studies estimating PM concentration-response functions. Also, because people are often quite mobile (e.g., living in one part of a city and working in another), a city-wide average PM level may be a more meaningful measure of ambient PM concentration than PM levels at individual monitors. Ito et al. 1995 found that averaging PM-10 concentrations reported at monitors in different places generally improved the significance of the association between PM-10 and mortality in Chicago, compared with using individual monitors.

Frequency graphs of average daily PM levels in each of the sample locations are shown for both PM-10 and PM-2.5 for Philadelphia County in Exhibit 4.1, and for PM-10 and PM-2.5 in Southeast Los Angeles County in Exhibits 4.2 and 4.3, respectively.

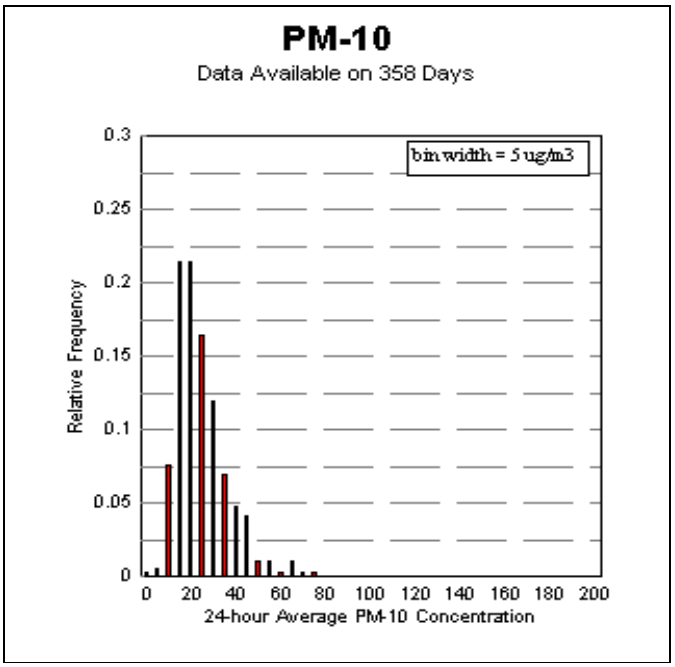
4.1. The Philadelphia County PM data

Air pollution data were collected in Philadelphia County by the Harvard School of Public Health (HSPH) Exposure Assessment and Engineering Program for the EPA's Atmospheric Research and Exposure Assessment Laboratory. Each monitor recorded pollution levels for PM-10 and PM-2.5. No monitor gave information on every day. Exhibit 4.4 lists the monitors used. Exhibits 4.5 and 4.6 show the number of days on which PM-10 and PM-2.5 concentration data were available at each monitor. Concentration data were available almost every day in Philadelphia County (as compare with the situation in Southeast Los Angeles County, described below). Exhibits 4.7 through 4.9 summarize the reported PM-10 and PM-2.5 concentrations at the three monitors used, and for a composite monitor assumed to report on each day the average of any concentrations reported by the three monitors.

Because not all monitors report PM concentrations on all days, the effect of estimating missing concentrations was explored in a previous analysis[8] of a data set including monitors with many more days missing than those used in this analysis. Adding estimated values where there had been missing values proved to have virtually no effect on the distribution of PM concentrations. Therefore, no such estimation was attempted in this analysis, and Philadelphia air quality is represented on each day by the average of any concentrations reported by the three monitors. The method used to adjust health effect incidence estimates from those corresponding to the number of non-missing days in the year to estimates corresponding to a full year is described in Section 2.

[8] Proposed Methodology for PM Risk Analyses in Selected Cities. Abt Associates, February 1996.

Exhibit 4.1
Daily Average PM Concentration Frequencies
Philadelphia County, September 1992 - August 1993



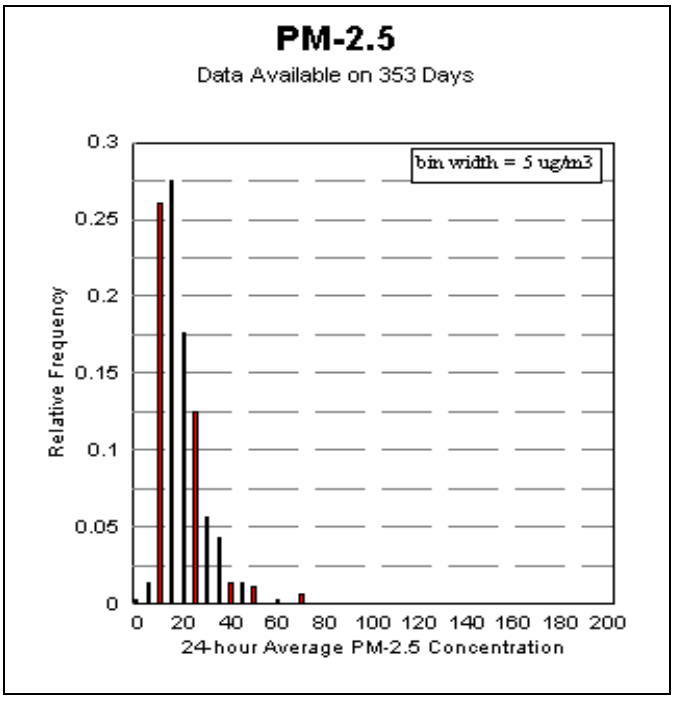
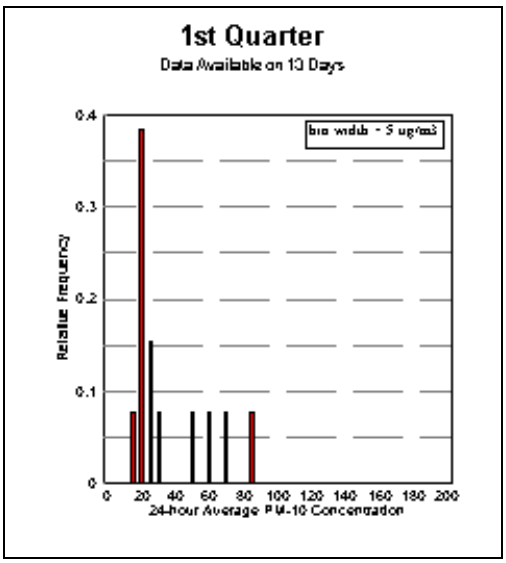
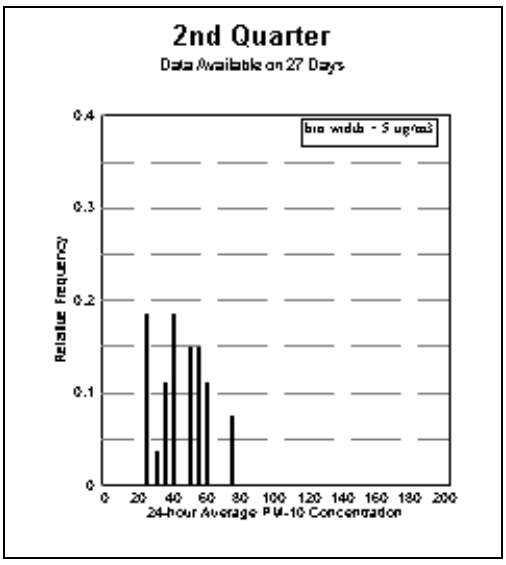
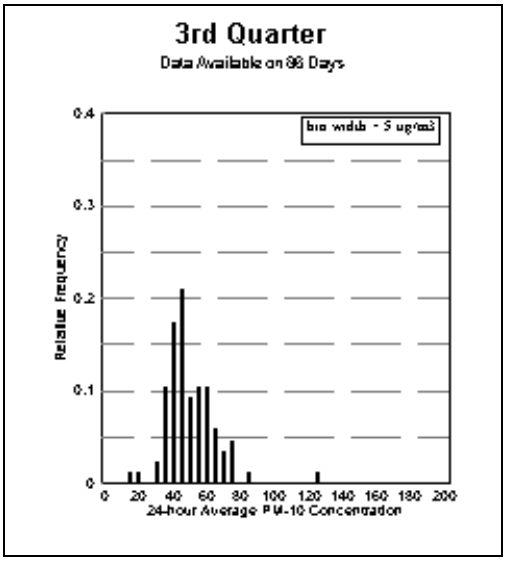


Exhibit 4.2
Daily Average PM-10 Concentration Frequencies
Southeast Los Angeles County, 1995







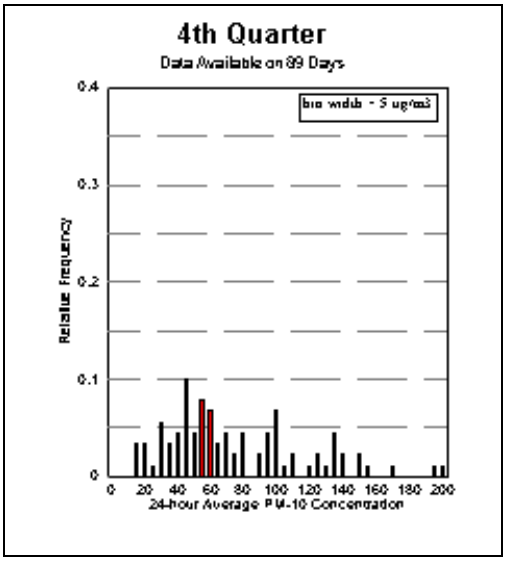
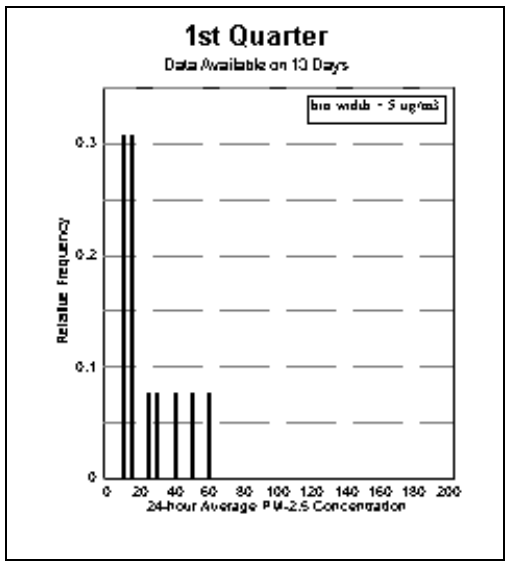
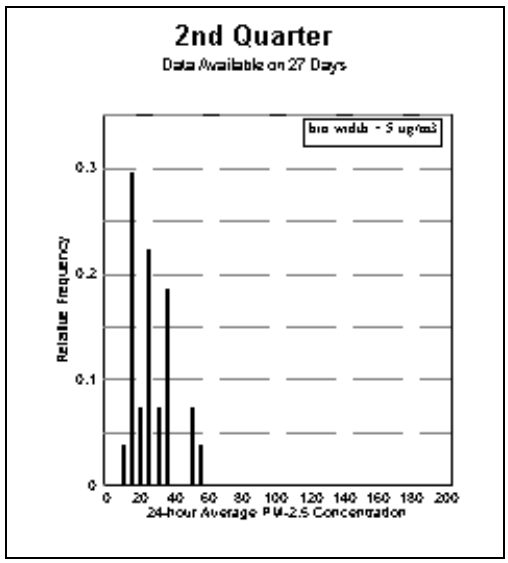
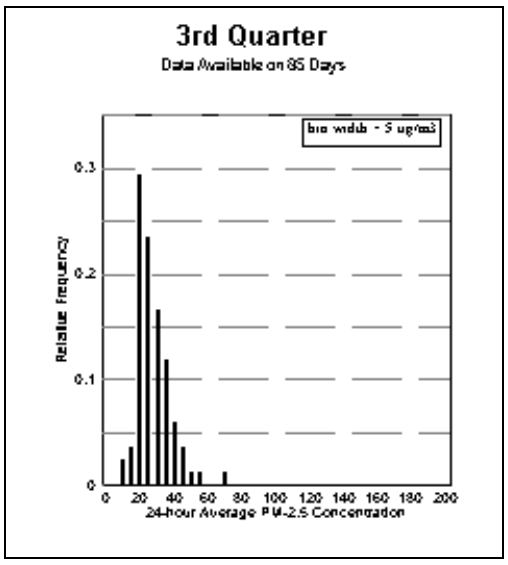


Exhibit 4.3
Daily Average PM-2.5 Concentration Frequencies
Southeast Los Angeles County, 1995







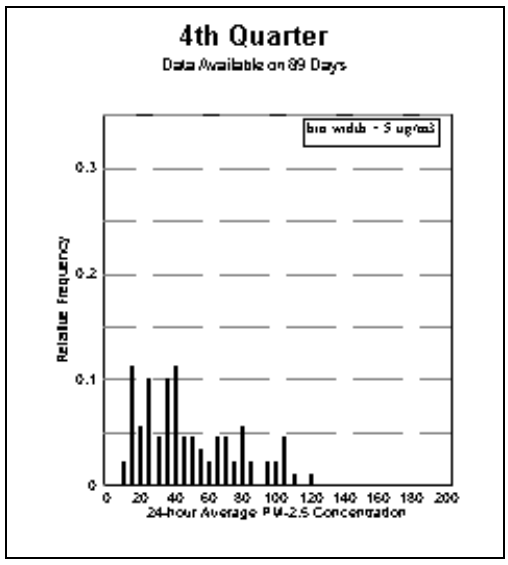


Exhibit 4.4. Philadelphia County Monitor IDs and Locations

Monitor Code	AIRS ID (if AIRS monitor)	Location
N/E		Philadelphia NE Airport: Grant/Ashton Roads
PBY		59th St. and Greenway Ave.
TEM	421010037	13th St. and Montgomery Ave.

Exhibit 4.5. Number of Days on which PM-10 Concentration Data are Available, by Quarter. Philadelphia, September 1992 - August 1993.

	Q1	Q2	Q3	Q4	Year Total
N/E	52	87	77	35	251
PBY	88	88	90	84	350
TEM	47	83	70	31	231
Composite	87	91	92	88	358

Exhibit 4.6. Number of Days on which PM-2.5 Concentration Data are Available, by Quarter. Philadelphia, September 1992 - August 1993.

	Q1	Q2	Q3	Q4	Year Total
N/E	52	81	76	34	243
PBY	81	84	87	86	338
TEM	43	89	71	35	238
Composite	84	91	90	88	353

Exhibit 4.7. PM-10 Concentrations by Quarter
Philadelphia, September 1992 - August 1993

	Q1	Q2	Q3	Q4	Weighted Year Avg.	2nd Daily Max
N/E	15.7	21.3	26.2	18.7	20.5	70.7
PBY	18.6	24.3	29.0	19.6	22.9	72.3
TEM	18.6	25.0	30.3	23.9	24.5	76.8
Composite	18.1	23.7	27.1	20.1	22.2	67.2

All concentrations are in $\mu g/m^3$.

Exhibit 4.8. PM-2.5 Concentrations, by Quarter
Philadelphia, September 1992 - August 1993

	Q1	Q2	Q3	Q4	WeightedYear Avg.	2nd Daily Max
N/E	12.9	16.3	20.0	13.2	15.6	65.1
PBY	14.3	17.6	21.8	14.2	17.0	72.6
TEM	13.6	17.3	22.0	15.6	17.1	70.0
Composite	13.8	17.0	20.9	14.1	16.5	69.3

All concentrations are in $\mu g/m^3$.

Exhibit 4.9. Percentile Points of Composite Distribution
Philadelphia, September 1992 - August 1993

	10th	25th	50th	75th	90th	Max
PM-10	10.4	14.2	19.7	28.2	44.2	72.6
PM-2.5	7.2	9.7	14.0	21.0	29.5	69.8

All concentrations are in $\mu g/m^3$.

4.2. The Southeast Los Angeles County PM data

Two California Air Resources Board monitors in Los Angeles County, designated Central Los Angeles (CELA) and Diamond Bar (DBAR) were selected to represent air quality. A portion of southeastern Los Angeles County selected for use in the analysis includes the portion of the county with the highest PM-10 levels. The region included in this analysis approximates the portion of the county reported to have an annual average PM-10 level above $40\mu g/m^3$ in 1994 (from "Air Quality Standards Compliance Report," South Coast Air Quality Management District, 1995). The two monitors reported concentrations infrequently during the first quarter of 1995, somewhat more frequently in the second quarter of 1995, and almost every day during the second and third quarters. Exhibits 4.10 and 4.11 show the number of days for which data were available in each quarter. Exhibits 4.12 and 4.13 show the average concentration reported in each quarter, as well as a weighted year average (weighted by quarter, as per the 1987 Federal Register notice, Vol. 52, No. 126, p. 24667 (July 1, 1987), and as used for annual average data reported in AIRS) and the second-highest reported value. Percentile points of the distributions are not provided, because the unequal distribution of monitor-days among quarters, even for the composite monitor, provides a skewed picture of air quality. All of the tables include statistics for the CELA and DBAR monitors, as well as for a composite monitor, assumed to report on each day the average of whatever concentrations were reported by the CELA and DBAR monitors.

Exhibit 4.10. Number of Days on which PM-10 Concentration Data are Available, by Quarter. Southeast Los Angeles County, 1995.

	Q1	Q2	Q3	Q4	Year Total
CELA	12	26	83	85*	206
DBAR	10	26	82	81	199
Composite	13	27	86	89	215

*One concentration, on October 30, was omitted as an obvious error.

Exhibit 4.11. Number of Days on which PM-2.5 Concentration Data are Available, by Quarter. Southeast Los Angeles County, 1995.

	Q1	Q2	Q3	Q4	Year Total
CELA	12	26	80	83	201
DBAR	9	26	82	84	201
Composite	13	27	85	89	214

Exhibit 4.12. PM-10 Concentrations by Quarter
Southeast Los Angeles County, 1995

	Q1	Q2	Q3	Q4	WeightedYear Average	2nd Daily Max
CELA	36.4	45.8	52.0	72.8	51.7	195.2
DBAR	30.4	40.0	43.2	72.2	46.4	170.7
Composite	32.6	42.4	47.5	72.4	48.7	193.4

All concentrations are in $\mu g/m^3$.

Exhibit 4.13. PM-2.5 Concentrations by Quarter
Southeast Los Angeles County, 1995

	Q1	Q2	Q3	Q4	Weighted Year Average	2nd Daily Max
CELA	21.6	26.0	27.0	45.6	30.1	115.7
DBAR	18.5	20.2	23.1	47.6	27.3	129.3
Composite	20.6	23.3	25.1	45.8	28.7	106.2

All concentrations are in $\mu g/m^3$.

5. Concentration-Response Functions

5.1. Concentration-response functions taken from the literature

Study selection decisions are among the most important subjective decisions that must

be made in a risk analysis. These judgements include decisions on which endpoints to consider quantitatively, as well as decisions about which of the available scientific studies should be used for quantitative risk analysis. The concentration-response functions reported by epidemiological studies are estimates of the relationships between PM-10 and PM-2.5 and a variety of health endpoints. The choice of functions for use in the risk analysis was guided by the PM Criteria Document (EPA 1996a, Tables 13-3 to 13-5).

The studies highlighted by the CD, and those used in this risk analysis to derive quantitative estimates of risk, used PM-10 or PM-2.5 mass (or other fine particle indicators) as their indicator of PM. This eliminated, for example, the extensive studies of air pollution and mortality conducted in Philadelphia (e.g., Health Effects Institute 1995, Moolgavkar et al. 1995a, Schwartz and Dockery 1992). In addition, two studies were omitted because other studies in the same locations were considered more appropriate for use in this risk analysis. Styer et al., 1995 estimated a concentration-response function for mortality in Chicago only for autumn; Ito and Thurston 1996 estimated a concentration-response function for mortality in Chicago for the whole year, and was therefore preferable. Dockery et al. 1992 (St. Louis and East Tennessee) was superseded by Schwartz et al. 1996, which considered much larger data sets in the same locations. Finally, for some studies of respiratory symptoms, the definitions of cough and lower respiratory symptoms can overlap; thus for risk analyses using these studies only lower respiratory symptoms were evaluated.

The health endpoints and the epidemiology studies used to estimate concentration-response functions for these endpoints that have been considered in the risk analysis are summarized in Exhibit 5.1.

As can be seen in Exhibit 5.1, most of the concentration-response functions were not estimated in either Philadelphia or Los Angeles. If the concentration-response relationship for a given combination of health endpoint and PM indicator were the same everywhere, then a concentration-response function estimated in one location could be applied to another location, and the only uncertainty would be from the usual sampling error associated with any estimate. There is no reason to believe, however, that the concentration-response relationship is the same everywhere. If a concentration-response function has not been estimated for the location of interest (e.g., Philadelphia County or Southeast Los Angeles County), this presents the problem of how to best estimate the concentration-response relationship in that location, based on information from other locations. Even if a concentration-response function has been estimated in the location of interest, the estimate of that location-specific function may be improved by incorporating information from other locations into the estimate. The more sampling error there is around the location-specific

Exhibit 5.1. Studies of the Health Effects Associated with Particulate Matter Pollution Used in the Risk Analysis

Endpoint	Study	City/Location	PM Indicator
Short-Term Exposure Mortality:	Ito and Thurston, 1996	Chicago, IL	PM-10
	Schwartz et al., 1996a	Six Cities	PM-10
	Pope et al. 1992	Utah Valley, UT	PM-10
	Schwartz, 1993	Birmingham, AL	PM-10
	Kinney et al . 1995	Los Angeles, CA	PM-10
	Schwartz et al., 1996a	Six Cities	PM-2.5
Long-Term Exposure Mortality:	Pope et al. 1995	51 U.S. Cities	PM-2.5
Hospital Admissions:			
Respiratory Disease:	Schwartz et al. 1995	Tacoma, WA	PM-10
	Schwartz et al. 1995	New Haven, CT	PM-10
	Schwartz, 1996	Spokane, WA	PM-10
	Thurston et al. 1994	Ontario, CA	PM-2.5*
COPD:	Schwartz, 1994a	Birmingham, AL	PM-10
	Schwartz, 1994b	Detroit, MI	PM-10
	Schwartz, 1994c	Minneapolis, MN	PM-10
	Schwartz, 1996	Spokane, WA	PM-10
Pneumonia:	Schwartz, 1994a	Birmingham, AL	PM-10
	Schwartz, 1994b	Detroit, MI	PM-10
	Schwartz, 1994c	Minneapolis, MN	PM-10
	Schwartz, 1996	Spokane, WA	PM-10
Ischemic Heart Disease:	Schwartz & Morris, 1995	Detroit, MI	PM-10
Congestive Heart Failure:	Schwartz & Morris, 1995	Detroit, MI	PM-10
Respiratory Symptoms:	Dockery et al., 1989	Six Cities	PM-10
	Pope et al. 1991	Utah Valley, UT	PM-10
	Ostro et al., 1995	Los Angeles, CA	PM-10

*Thurston et al.(1994) reports both a PM-10 and a PM-2.5 coefficient. In a later paper (Thurston and Kinney, 1995), however, the authors interpret their findings as "clear that the FP [fine particle] portion of the mass (including particle strong acidity, H+) is driving the apparent relationships seen for the PM-10 and TSP metrics." The risk analysis therefore uses only the PM-2.5 results from Thurston et al. (1994).

estimate, the greater will be the improvement achieved by incorporating information from other locations. This is discussed below.

5.2. Estimation of a distribution of β's, estimation of β in any given location, and characterization of the uncertainty surrounding that estimate

The concentration-response function is an important component of the risk analyses and a source of substantial uncertainty in those analyses. The exponential concentration-response function (equation (1), Section 2) commonly assumed in the epidemiological literature on particulate matter pollution health effects and used as the basis for the risk analyses implies that the relationship between a given *change* in PM concentration, Δx, and the corresponding *change* in the health endpoint, Δy,

$$\Delta y = y[e^{\beta \Delta x} - 1]$$

(see Section 2 and Appendix 3), depends critically on the value of β. The larger the value of β, the greater the change in the health effect associated with a given change in PM concentration. For ease of discussion, the health endpoint is taken to be mortality. However, the discussion below applies to any health endpoint.

It is possible that there is only a single value of β, that is, that the concentration-response relationship between PM and mortality is the same everywhere. If this is the case, different estimates of β reported by different epidemiological studies are all estimates of the same underlying parameter and differ from each other only because of sampling error.

A more general and a more plausible model, however, is that there is not just a single concentration-response relationship between PM and mortality, but that this relationship varies from one location to another, depending on such factors as the composition of the PM and the composition of the exposed population. Even if the form of the concentration-response function is the same everywhere, the value of β may change from one location to another, reflecting differences in these factors. For example, it may be the case that in Philadelphia County,

$$\Delta y = y[e^{\beta_1 \Delta x} - 1] \quad ,$$

whereas in Los Angeles,

$$\Delta y = y[e^{\beta_2 \Delta x} - 1] \quad .$$

with $\beta_1 \neq \beta_2$.

The concentration-response relationship between PM and mortality (for example, throughout the United States) may be characterized, then, by a *distribution* of β's. For any given interval of possible values of β, this distribution describes the probability that β (and

therefore the concentration-response relationship) at any particular location is within that interval.[9,10]

If there is an underlying distribution of β's, then differences in reported estimates of β among studies carried out on a *single* population in a single location (using identical averaging times, methodology, etc.) would reflect only sampling error, because all such studies are estimating the same β. Differences in reported estimates among studies carried out on different populations in different locations, however, may also reflect differences in the β's being estimated. There are, then, two potential sources of variability among concentration-response estimates:

(1) within-study variability, or sampling error (so that even two studies estimating the same β are likely to report different estimates), and

(2) between-study variability derived from the fact that studies may be estimating different underlying parameter values,

and associated with these two sources of variability is uncertainty about the correct concentration-response function for a given location.

If the underlying distribution of β's were known, this distribution could be used to characterize the uncertainty surrounding an estimate of β applied to a given location (in the absence of an estimated concentration-response function for that location). Suppose, for example, that $\beta_{0.05}$ is the 5th percentile of the distribution of β's and $\beta_{0.95}$ is the 95th percentile. Then, for a randomly selected location (e.g., for a sample location), there is a 90 percent probability that β in that location falls within the interval ($\beta_{0.05}$, $\beta_{0.95}$). The distribution of β's thus allows uncertainty bounds to be associated with any estimate of β applied to a particular location.

[9]This is technically referred to as a probability density function, but will be called a distribution here for simplicity.

[10]Since the concentration-response relationship (β) in a given location is uncertain, the mortality predicted using any given concentration-response relationship is also uncertain. Corresponding to the underlying distribution of β's, there is a distribution of PM-related mortality, describing the probability that the incidence of mortality associated with a given PM level falls within a given interval.

5.2.1. Estimation of the distribution of β's

The distribution of β's is not known, however, and must be estimated. Once the distribution is estimated, the interval from the 5th percentile to the 95th percentile of the estimated distribution, referred to as the "90 percent credible interval," is used to characterize the uncertainty associated with β in any location for which a concentration-response function has not been estimated (and for which there is therefore no information more specific than the general distribution of β's).

If only a single study has estimated a concentration-response function for a given endpoint, then the only available information about the distribution of β's comes from that study. Within the general case of n studies reporting β's, this is just the special case in which n=1. The discussion below refers to the general case of n studies (where n may be greater than or equal to 1).

If each study were reporting the *true* concentration-response function for the location studied, then the set of reported β's would be a sample from the underlying distribution of β's and could therefore be used to help estimate this distribution. What each study reports, however, is an *estimate* of the concentration-response function (or, equivalently, an estimate of β) for the location studied. The reported β for each study location therefore has some sampling error associated with it.

Under the assumption that the true β's in the various study locations are all drawn from the same distribution of β's, an estimate of β for a given study location that uses information from *all* the study locations is generally better than an estimate that uses information from only the given study location (see, for example, Efron and Morris, 1973; Laird and Ware, 1982; and Laird and Ware, 1984).[11] That implies that the estimates of β reported for each study location can be improved upon. Suppose, for example, that the concentration-response function for PM-10 and mortality has been estimated in locations A, B, C, and D. Let MLE_A denote the maximum likelihood estimate of β in location A, MLE_B the maximum likelihood estimate of β in location B, and so on. Let pooled(A,B,C,D) denote a pooled estimate of the concentration-response function, pooling the concentration-response functions in locations A through D. Then a weighted average of MLE_A and pooled(A,B,C,D) is a better estimator of β in location A than MLE_A, and similarly for locations B, C, and D.

A good estimate of the distribution of β's, then, relies on first adjusting the β's estimated in individual study locations by incorporating information from the other study locations (if there is more than one study location). The estimation of the distribution of β's for a given combination of health endpoint and PM indicator is a three-step procedure which efficiently uses the available information as follows:

[11]Estimator A is better than estimator B if the mean squared error associated with estimator A is less than the mean squared error associated with estimator B.

In step 1, a random effects pooled estimate of β is calculated, using the β's reported by those studies that estimated a concentration-response function for the given combination of health endpoint and PM indicator. The random effects pooled estimate is based on the assumption that there is an underlying distribution of β's, with variance η^2. The pooled estimate is a weighted average of the reported β's (which are assumed to be estimates of β's drawn from the distribution). The weights are a function of both the sampling error (the within-study variability) and η^2 (the between-study variability). The calculation of the weights is described in Appendix 2. The pooled estimates, calculated for all health endpoint PM indicator combinations for which there is more than one study, are given in Section 5.2.2.

In step 2, β in each study location is re-estimated, using a weighted average of the β reported for that location and the random effects pooled estimate calculated in step 1. (This shifts the individual β's towards the pooled estimate.) The standard error of the estimate of β is similarly recalculated. (This reduces the standard errors associated with the individual β's.) The uncertainty associated with β in the ith location is, as before the adjustment, described by a normal distribution. The adjustment in step 1 simply shifts the mean of that distribution from the β reported by the study to the re-estimated β. (The adjustment also reduces the standard deviation of this normal distribution by incorporating information from all study locations into the estimate of β in the ith study location.) The formulas for the adjusted mean and standard deviations are given in Appendix 5.

In step 3, the underlying distribution of β's is estimated as an (unweighted) average of the normal distributions derived in step 2. Suppose, for example, that three epidemiology studies reported estimates of β for PM and mortality, each in a different location. The available information about what β might be in a randomly selected location (not necessarily one of the three for which β has been estimated) is contained, then, in three normal distributions, adjusted in step 2 above. These adjusted distributions, denoted f_1, f_2, and f_3, are centered at three different values, β_1, β_2, and β_3 (the adjusted β's). The underlying distribution of β's is estimated as follows: For any possible value of β, x, the value of the distribution of β's at x is estimated as

$$f(x) = \frac{1}{3}\left[f_1(x) + f_2(x) + f_3(x)\right] \quad ,$$

normalized so that the area under the distribution function integrates to 1. In general, the estimate of the distribution of β's based on n studies is

$$f(x) = \frac{1}{n}\sum_{i=1}^{n} f_i(x)$$

normalized so that the distribution function, f(x), integrates to 1.

If n=1, that is, if there is only a single study for a given combination of health endpoint and PM indicator, the estimated distribution of β's is just the distribution for that study -- that is, a normal distribution, with mean equal to the reported β in the study and standard deviation equal to the reported standard error of the mean. (There is no pooled estimate when n=1, and therefore no adjustment of the β reported by the single study.)

The 5th and 95th percentiles of this estimated distribution, f(x), are then the 90 percent credible interval -- that is, the estimate of the interval within which the true value of β at a randomly selected location (e.g., the location of interest) lies with 90 percent probability.

The adjustment of the normal distributions in step 2 above may also be seen in a Bayesian framework, in which the distributions reported by the studies are considered prior distributions, and the adjusted distributions are considered posterior distributions, incorporating the evidence of the random effects analysis. The procedure is referred to as an "empirical Bayes" estimation procedure because the prior distributions are based on empirical evidence (see, for example, Efron and Morris, 1971; Efron and Morris, 1972a; Efron and Morris, 1972b; Efron and Morris, 1973; and Cox and Hinkley, 1974) . Exhibit 5.3 shows the prior and posterior distributions for the set of 10 functions relating short-term PM-10 exposure and mortality. The adjustment pulls all mean relative risk estimates towards the random-effects distribution average relative risk of 1.040, with those starting furthest from the average being changed the most. In addition, the standard deviations are reduced, since the combination of several coherent analyses reduces overall uncertainty.

The classic Monte Carlo technique, which consists of generating a large number of observations from a known distribution, is another technique often used to assess uncertainty. When there are several sources of uncertainty, or equivalently, several parameters in the model whose values are uncertain, Monte Carlo methods are often used to generate observations from several distributions. On each of a large number of iterations, an observation is randomly drawn from each of the distributions, and the model output based on the parameter values drawn is calculated. (This is referred to as "propagating uncertainty through the model.") N iterations thus generate N model output values. As N approaches infinity, the distribution of output values approaches the distribution of output values consistent with the distributions of input parameter values. A ninety percent confidence interval around the model output can then be derived from the 5th and 95th percentiles of this distribution.

The distribution, f(x),whose derivation is discussed above, can be shown to be the limit distribution of a Monte Carlo "propagation of uncertainty" procedure in which there is only a single source of uncertainty (the concentration-response function). That is, this distribution would have been approached by a Monte Carlo procedure analogous to the procedure used in the typical "propagation of uncertain" exercise. Because there is only a single source of uncertainty, the Law of Large Numbers allows the analytic calculation of the distribution reached in the limit, that is, as the number of trials in the Monte Carlo procedure approaches infinity. The analytic limit distribution was used in these risk analyses.

Exhibit 5.3. Empirical Bayes Estimation of Distributions of Relative Risk for Mortality for a 50 μg/m^3 Increase in PM-10: Prior and Posterior Distributions

Study	Location	Prior (unadjusted)		Posterior (adjusted)	
		mean	std. dev.	mean	std. dev.
Pope et al '92	Utah	1.076	0.017	1.056	0.011
Schwartz '93	Birmingham	1.054	0.022	1.044	0.012
Kinney et al '95	LA	1.025	0.014	1.032	0.010
Schwartz et al 1996a	Boston	1.061	0.013	1.052	0.010
	Knoxville	1.046	0.023	1.042	0.012
	St. Louis	1.030	0.012	1.034	0.009
	Steubenville	1.046	0.020	1.042	0.012
	Portage	1.035	0.028	1.039	0.013
	Topeka	0.975	0.036	1.032	0.013
Ito et al '95	Chicago	1.025	0.006	1.027	0.006

As an alternative to the three-step method described above, a standard functional form for the underlying distribution of β's may be *assumed* (for example, it may be assumed to be a normal distribution or a beta distribution). In this case, the reported estimates of β could be treated as a sample from this distribution and used to estimate the values of the parameters of the (assumed) distribution. Because the method described above does not impose any particular standard functional form on the underlying distribution of β's, however, but instead allows the reported estimates of β and the user's confidence in these estimates to suggest the form, this method is preferred as a way of providing an estimate of the underlying distribution that is most consistent with the evidence from the epidemiological studies.

5.2.2. Estimating β in a given location

In the absence of any location-specific information, the most reasonable estimate of β in a given location (other than the study locations) is the mean of the estimated distribution of β's. When there is only a single study that has estimated a concentration-response function, the estimated distribution of β's is just a normal distribution with mean equal to the β reported by the study. In this case, the single reported β is therefore the best estimate of β in the given location. When there is more than one study, it can be shown that the mean of the estimated distribution of β's is the random effects pooled estimate derived in step 1 and used in step 2. This pooled estimate, then, is the best estimate of β in the given location. The uncertainty bounds around the estimate are just the 90 percent credible interval, described above. (In the case of a single study, this is the same as the 90 percent confidence interval around the mean.)

5.2.3. Pooled estimates of β

Many studies have attempted to determine the influence of particulate matter pollution on human health. Usually this involves estimation of relative risk for a given change in pollutant concentration. Each study provides an estimate of the relative risk, along with a measure of the uncertainty of the estimate. Because uncertainty decreases as sample size increases, combining data sets is expected to yield more reliable estimates of relative risk. Combining data from several comparable studies in order to analyze them together is often referred to as meta-analysis.

For a number of reasons, including data confidentiality, it is often impractical or impossible to combine the original data sets. Combining the *results* of studies in order to produce better estimates of relative risk provides a second-best but still valuable way to synthesize information (DerSimonian and Laird, 1986). This is referred to as "pooled analysis" in this report. This kind of pooled analysis requires that all estimates of relative risk be made using the same change in pollutant concentration. The method of pooled analysis used is described briefly below and in more detail in Appendix 2. Appendix 3 discusses how relative risks for different pollutant concentration changes can be made comparable.

One method of pooled analysis is simply averaging all reported relative risks. This has the advantage of simplicity, but the disadvantage of not taking into account the measured uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty.

It seems reasonable that a "pooled estimate" which combines the estimates from different studies should give more weight to estimates from studies with little reported uncertainty than to estimates with a great deal of uncertainty. The exact way in which weights are assigned to estimates of relative risk from different studies in a pooled analysis depends on the underlying assumption about how the different estimates are related to each other.

Under the assumption that there is a distribution of β's (referred to as the random effects model), the different relative risks (or β's) reported by different studies may be estimates of *different* underlying relative risks (corresponding to a given change in concentration), rather than just different estimates of the same relative risk. The random-effects model is preferred here to the fixed effects model (which assumes that there is only one β everywhere), because it does not assume that all studies are estimating the same parameter.[12] (Some researchers (e.g. Moolgavkar & Luebeck, 1996; in press) suggest, however, that the

[12] In studies of the effects of PM-10 on mortality, for example, if the composition of PM-10 varies among study locations the underlying relationship between mortality and PM-10 may be different from one study location to another. For example, fine particles make up a greater fraction of PM-10 in Philadelphia County than in Southeast Los Angeles County. If fine particles are disproportionately responsible for mortality relative to coarse particles, then one would expect the true value of β for PM-10 in Philadelphia County to be greater than the true value of β for PM-10 in Southeast Los Angeles County. This would violate the assumption of the fixed effects model.

heterogeneity in estimates of PM effects makes single estimates of risk difficult to estimate.)

Several pooled analyses, performed on a number of combinations of PM-10 studies identified in the Criteria Document for PM-10 (EPA, 1996a), are described below. Studies were aggregated for analysis based upon the health endpoint of concern (only mortality and hospital admissions are considered) and the period of exposure evaluated (e.g., one day, three day). In addition, a pooled function for mortality and PM-2.5, derived by Schwartz et al.1996a from their studies in six cities, is also presented.

5.2.3.1. Pooled analyses of mortality PM-10 concentration-response functions

Pooled analyses were performed on various subsets of the following short-term exposure mortality studies cited in the Criteria Document for PM-10 (EPA, 1996a):[13]

- Ito and Thurston 1996 (Chicago, IL);
- Kinney et al. 1995 (Los Angeles, CA);
- Pope et al. 1992 (Utah Valley, UT);
- Schwartz 1993 (Birmingham, AL);
- Schwartz et al. 1996a (Boston; Knoxville, TN; St. Louis; Steubenville, OH; Portage, WI; Topeka, KS)

Exhibit 5.4 shows the relative risks reported in the original studies for a change in PM-10 concentration of 50 $\mu g/m^3$. It is explained in Appendix 3 how the relative risk corresponding to one PM concentration change can be adjusted to reflect other concentration changes. Exhibit 5.5 shows the studies included in each pooled analysis.

The "all averaging times" pooled analysis includes studies that used the average PM concentration on a single day as the pollution indicator as well as studies that used the average PM concentration over a 2-, 3- or 5-day period. Those studies which use multi-day averages are in effect using a smoothed data set, comparing each day's mortality to recent average exposure rather than simply exposure on the same day. The averaging times applied

[13]Although there are five studies, Schwartz et al. 1996a effectively conducted six separate studies in the six cities listed. Following Schwartz et al., the pooled analyses treat these six cities as separate studies. There are therefore ten PM-10 mortality studies on which pooled analyses were based.

Exhibit 5.4. Relative Risks of Mortality Associated With a Change in PM-10 Concentration of 50 μg/m^3.

Study	Location	Relative Risk	Standard Error
Ito and Thurston, 1996	Chicago	1.025	0.006
Kinney et al., 1995	Los Angeles	1.025	0.014
Pope et al., 1992	Utah	1.076	0.017
Schwartz, 1993	Birmingham	1.054	0.022
Schwartz et al.,1996a	Boston	1.061	0.013
Schwartz et al., 1996a	Knoxville, TN	1.046	0.023
Schwartz et al., 1996a	St. Louis	1.030	0.012
Schwartz et al., 1996a	Steubenville, OH	1.046	0.020
Schwartz et al., 1996a	Portage, WI	1.035	0.027
Schwartz et al., 1996a	Topeka, KA	0.975	0.036

Exhibit 5.5. Studies Included in Each Pooled Analysis for PM-10

Study	all averaging times	single-day averaging only	2-day averaging only	multi-day averaging only
Ito et al. '95	✓		✓	
Kinney et al. '95	✓	✓		
Pope et al. '92	✓			✓
Schwartz '93	✓			✓
Schwartz et al. 1996a	✓		✓	

to the PM data used in the risk analyses were chosen to correspond to the majority of functions used in a pooled analysis. Because seven of the ten functions included in the “all averaging times” pooled analysis are based on two-day PM averages, two-day PM averages were used with this pooled function. Single-day PM concentrations were used with the one single-day function; two-day PM averages were used with the two two-day functions; and five-day PM averages were used with the two multi-day functions (one of which is a three-day function and the other of which is a five-day function). The more nearly linear the concentration-response function, however, the less difference it makes whether multi-day averaging functions are used with single-day PM data. (If the functions were perfectly linear, it would make no difference at all.) The concentration-response functions considered here are nearly linear, as discussed above. Because all of the studies in the "all averaging times" pooled analysis focus on the results of short-term exposure to pollution (as opposed to long-term exposure, measured in years), it is appropriate to consider these studies together.

Exhibit 5.6 shows the average relative risk of the mortality studies considered in each pooled analysis, as well as the relative risk estimates (and standard errors) from pooled analyses based on the fixed effects model and, where possible, the random effects model. The random effects model is preferred because it takes into account possible geographic variability, and pooled functions were based on the random effects model except when this was not possible due to insufficient difference among the reported studies (see Appendix 2). Relative risks are expressed for a 50 $\mu g/m^3$ increase in PM-10 concentration. For the single-day averaging time study, it was not possible to calculate a random effects model estimate, and so only the result based on the fixed effects model is shown. Exhibit 5.7 shows the relative risk results of the original studies and the inverse variance weighting pooled analyses graphically, including the 95 percent confidence bounds.

Exhibit 5.6. Pooled Analyses of Mortality for PM-10 Relative Risk Estimates for a 50 $\mu g/m^3$ Increase in PM-10

Group	N	Arithmetic Average Relative Risk	Fixed Effects Inverse Variance Weighting		Random Effects Inverse Variance Weighting	
			est. RR	s.e.	est. RR	s.e.
All averaging times	10	1.037	1.035	0.004	1.040	0.007
Single-day averaging only	1	1.025	1.025*	0.014*	n/a	n/a
2-day averaging only	7	1.031	1.032	0.005	1.035	0.007
>2 -day averaging only	2	1.065	1.068	0.013	n/a	n/a

*Although there is only one single-day averaging study (Kinney et al., 1995), and therefore no weighting is possible, the figures in this column are presented to include the standard error.

Exhibits 5.6 and 5.7 illustrate that the selection of studies to include in the pooled analysis is a critical choice. For example, pooling the two studies that use average PM concentrations over three to five days increases the relative risk estimated for a 50 $\mu g/m^3$ increase in PM-10, compared to that estimated by the pooled function based on studies that use average PM concentrations over only two days.

5.2.3.2. Pooled analyses of mortality PM-2.5 concentration-response functions

Exhibit 5.8 summarizes the available short-term exposure mortality studies that use PM-2.5 as the particulate matter indicator. Schwartz et al. 1996a reported relative risk estimates for six cities, along with an estimate for all six cities combined, based on a method similar to the pooled analysis method described above. Exhibit 5.8 presents Schwartz et al.'s relative risk estimates for a 25 $\mu g/m^3$ increase in PM-2.5 for each of the six cities separately as well as the relative risk estimate based on the pooled function provided in the study.

Exhibit 5.7. Relative Risk of Mortality Associated with a Change in PM-10 Concentration of 50 µg/m3 (Individual Studies and Pooled Analyses)

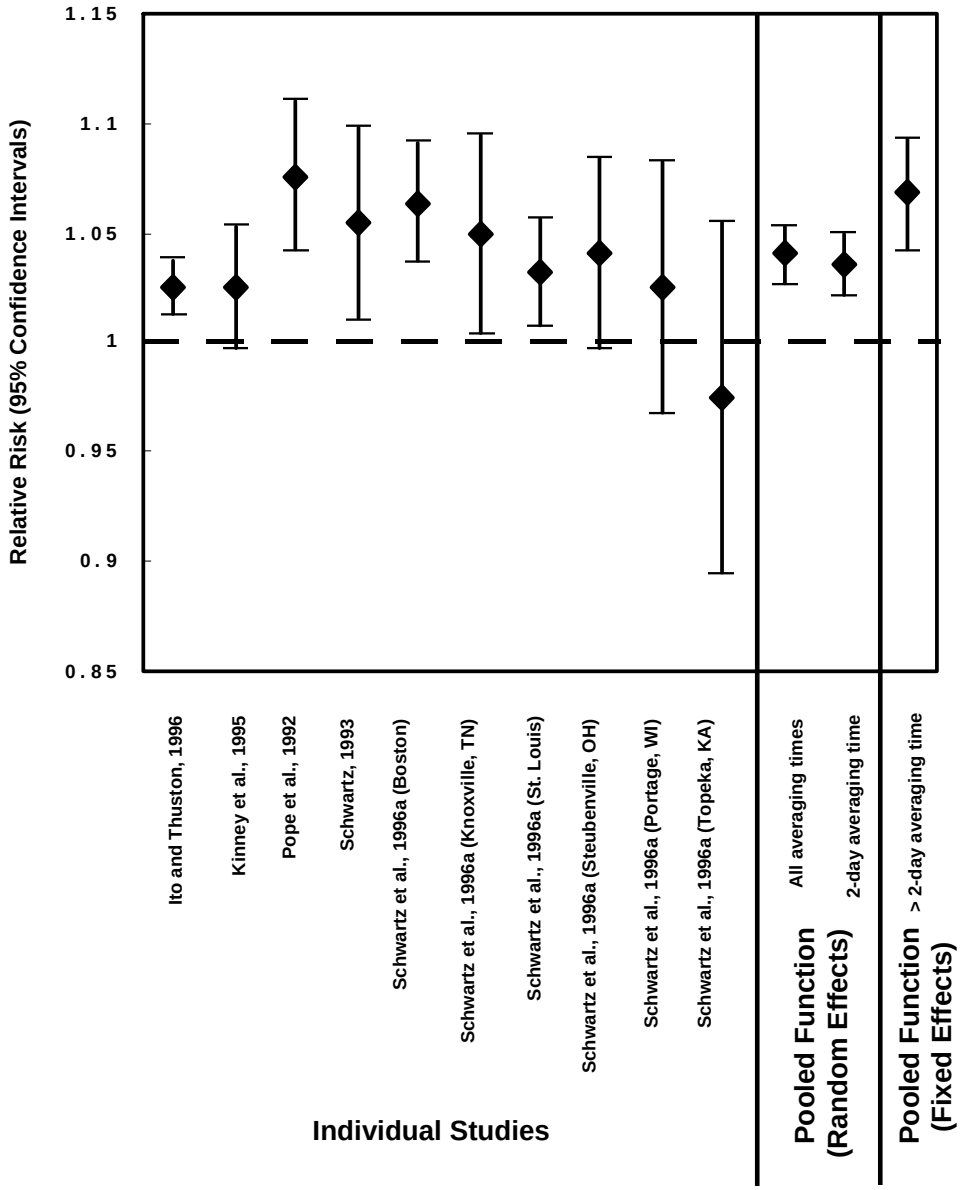


Exhibit 5.8. Short-term Exposure Mortality Studies Using PM-2.5 as the Indicator of Particulate Matter, with Pooled Analyses. Relative Risk Estimates for a 25 μg/m^3 Increase in PM-2.5

Study City	Relative Risk (95% Confidence Interval)	Source
Watertown, MA	1.056 (1.038, 1.074)	Schwartz et al. 1996a
Knoxville, TN	1.035 (1.005, 1.066)	Schwartz et al. 1996a
St. Louis, MO	1.028 (1.010, 1.043)	Schwartz et al. 1996a
Steubenville, OH	1.025 (0.998, 1.053)	Schwartz et al. 1996a
Portage, WI	1.030 (0.993, 1.071)	Schwartz et al. 1996a
Topeka, KS	1.020 (0.951, 1.092)	Schwartz et al. 1996a
Pooled function, using fixed effects inverse variance weighting	1.038 (1.028, 1.048)	Schwartz et al. 1996a

5.2.3.3. Pooled analyses of morbidity concentration-response functions

Exhibit 5.9 shows the results of pooled analyses performed on studies of various other health effects of PM-10, as measured by hospital admissions. The four studies for hospital admissions for pneumonia and COPD come from Schwartz 1994a (Birmingham), Schwartz 1994b (Detroit), Schwartz 1994c (Minneapolis/St. Paul), and Schwartz 1996 (Spokane). The three figures used for "total respiratory" hospital admissions are from Schwartz 1995 (New Haven, CT and Tacoma, WA) and Schwartz 1996 (Spokane). A fourth study of respiratory hospital admissions, using data from Cleveland, was excluded, because it considers 2-day average PM-10 rather than same-day average PM-10 as the other studies do. (Including the Cleveland study makes virtually no difference in the results.) In only one case was the variation in results among the studies great enough to allow the use of a random-effects model. In the other cases, there is insufficient evidence that the different studies are estimating different underlying concentration-response functions.

Exhibit 5.9. Pooled Analyses of Effects of PM-10 on Hospital Admissions Relative Risk Estimates for a 50 μg/m^3 Increase in PM-10

Health Effect*	N	Arithmetic Average Relative Risk	Fixed Effects Inverse Variance Weighting		Random Effects Inverse Variance Weighting	
			est. RR	s.e.	est. RR	s.e.
hospital admissions- "total respiratory"	3	1.098	1.088	0.022	n/a	n/a
hospital admissions- pneumonia	4	1.071	1.069	0.014	n/a	n/a
hospital admissions- COPD	4	1.163	1.137	0.023	1.140	0.024

*All hospital admissions in this exhibit refer to individuals age 65 or older.

5.2.4. Quantitative assessment of uncertainty surrounding β's applied to Philadelphia and Los Angeles: results

An uncertainty analysis (i.e., estimation of the distribution of β's and calculation of a 90 percent credible interval) was carried out in each case in which a pooled analysis was performed (see Section 5.2.3) -- that is, in each case in which there is more than one reported estimate of the concentration-response function.[14] (The one exception is the concentration-response function for PM-10 and mortality, based on 1-day averaging, for which there is only one study. This was included within the set of pooled analyses for mortality for completeness.)

The estimated distribution of β's can be translated into a distribution of avoided health effects incidences corresponding to a given change in PM concentrations, as described above. Alternatively, it can be translated into a distribution of relative risks associated with a given change in PM concentrations, because each value of β corresponds to a particular relative risk for a given change in PM (see Section 2 and Appendix 3). The results of the uncertainty analyses, presented below, are in terms of relative risks associated with a 50 μg/m^3 change in PM-10 or a 25 μg/m^3 change in PM-2.5. The mean, the 95 percent credible interval (i.e., the 2.5 and 97.5 percentile points), and the 90 percent credible interval (i.e., the 5 and 95 percentile points) of the estimated distribution of relative risks for each uncertainty analysis are presented in Exhibit 5.10. The 90 percent credible intervals are reported along with the quantitative estimates of risk. This approach is consistent with the 90 percent confidence intervals used to characterize uncertainty in other Agency risk analyses, such as those conducted for ozone and lead. In addition, the 95 percent confidence intervals around the pooled analysis estimate of the mean of each underlying distribution of relative risks is presented for comparison both with the uncertainties as reported in the original studies and

[14]In those cases for which there is only a single study, the best estimates of the 5th and 95th percentiles of the distribution of β's are the 5th and 95th percentiles of the normal distribution with mean equal to the β reported by the study and standard deviation equal to the standard error of the mean reported by the study. There is therefore no "analysis" necessary.

with the 95 percent credible interval from the estimated distribution of relative risks.

The distinction between a credible interval and the corresponding confidence interval around the pooled estimate is worth a further note for the sake of clarity. The 90 percent confidence interval around the pooled estimate is derived from the standard error of the estimate of the mean, which is a measure of how good an estimate of the mean of the underlying distribution the pooled analysis estimate is. The 90 percent confidence interval around the pooled estimate is the interval within which the true mean of the underlying distribution lies with 90 percent confidence. (For a precise definition of a confidence interval, see, for example, Mood et al., 1974, p. 375.) The confidence interval around the pooled estimate, then, comprises uncertainty bounds around the true mean of the distribution.

The 90 percent credible interval, consisting of the 5th and 95th percentiles of the estimated underlying distribution, comprises uncertainty bounds around the true value of β (or the true relative risk) in a particular location. In the absence of any further information about that location, the 90 percent credible interval is an estimate of the interval within which β in that location will fall with 90 percent probability. This is the appropriate measure of uncertainty surrounding the estimate of β applied to a specific location.

The 90 percent credible interval will always be at least as wide as, and usually wider than the 90 percent confidence interval around the pooled estimate of the mean. The greater the variance of the underlying distribution (i.e., the larger η^2) the more of a discrepancy there will be between the two types of uncertainty bounds. As η^2 approaches zero, the 90 percent credible interval approaches the 90 percent confidence interval around the pooled estimate of the mean. This is the case, for example, when there is only a single study. In this case, there is no evidence of differing β's (because β has been estimated in only a single location) and therefore no evidence that η^2 is positive. In this case, the 90 percent credible interval equals the 90 percent confidence interval around the estimate of the mean, which is based only on the within-study sampling error.

To illustrate the comparison between the confidence interval around the pooled estimate of the mean of the distribution of β's and the corresponding credible interval for a location-specific β, Exhibits 5.11 and 5.12 show graphically the normal distributions representing the within-study variability around the β's reported by the ten "all averaging times" mortality PM-

Exhibit 5.10. Results of Uncertainty Analyses: Means and Ninety-Five Percent Credible Intervals of Estimated Distributions of Relative Risk

Health Endpoint	Number of Studies	Mean of Estimated Distribution of Relative Risk	95% Confidence Interval around Random Effects Pooled Analysis Estimate*	95% Credible Interval (2.5th and 97.5th percentile points)	90% Credible Interval (5th and 95th percentile points)
For a 50 μg/m^3 Increase in PM-10:					
A. Mortality					
all averaging times	10	1.040	(1.026, 1.053)	(1.014, 1.069)	(1.018, 1.064)
2-day averaging time	7	1.035	(1.021, 1.049)	(1.013, 1.059)	(1.017, 1.055)
> 2-day averaging time	2	1.068	(1.043, 1.093)**	(1.043, 1.093)**	(1.047, 1.090)**
1-day averaging time	1	1.025	(0.998, 1.052)**	(0.998, 1.052)**	(1.002, 1.048)**
B. Morbidity: Hospital admissions					
"Total respiratory"	3	1.089	(1.045, 1.131)**	(1.045, 1.131)**	(1.053, 1.125)**
COPD	4	1.140	(1.093, 1.187)	(1.087, 1.195)	(1.094, 1.185)
pneumonia	4	1.069	(1.042, 1.097)**	(1.042, 1.097)**	(1.046, 1.093)**
For a 25 μg/m^3 Increase in PM-2.5:					
Mortality	6 cities (in one study)	1.036	(1.026, 1.047)	(1.019, 1.053)	(1.022, 1.051)

* The random effects pooled analysis estimate of central tendency is the same as the mean of the uncertainty analysis distribution based on the same weights.

** A random effects pooled analysis could not be performed. Results are from a fixed effects pooled analysis.

10 studies as well as both the estimated distribution of β's and the corresponding pooled analysis estimate of the mean of the distribution. Exhibit 5.11 shows the ten unadjusted normal distributions representing the within-study variability around each of the ten reported β's. Exhibit 5.12 superimposes on the ten adjusted normal distributions (1) the pooled analysis estimate of the mean of the distribution of β's and the normal distribution around this estimate, based on the standard error of the estimate, and (2) the estimated distribution of β's generated from the underlying ten adjusted normal distributions, using the three-step estimation procedure described in Section 5.2.1.

Note that, even though the distributions around the reported estimates of β's are normal, the estimated distribution of β's derived from them is not normal. As noted above, it does not have a standard functional form but instead reflects the evidence from the particular studies on which it is based. Note also that it is possible, when sampling from the estimated distribution of β's, to select a negative number. The probability of doing so, however, is extremely small.

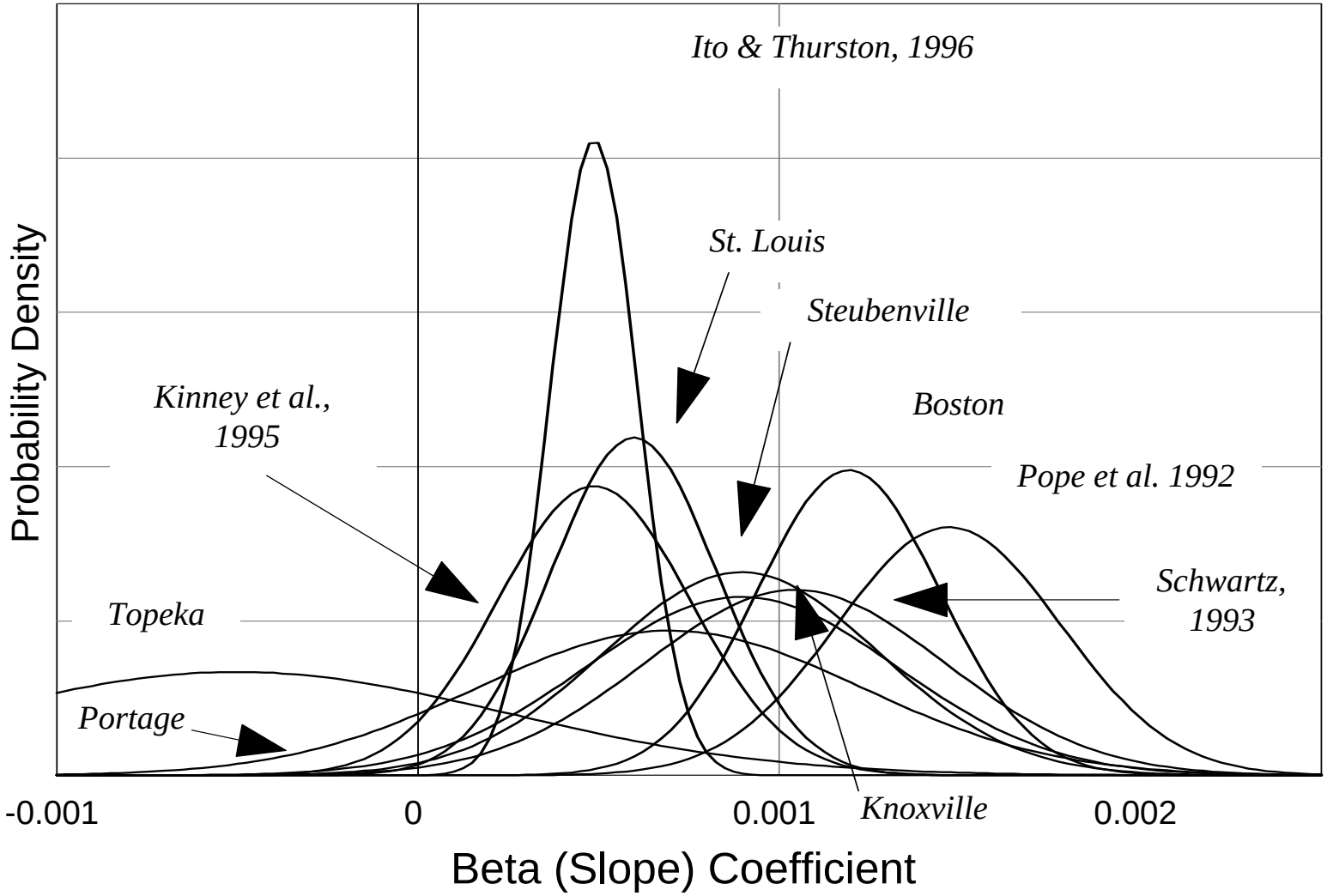
5.2.5. Translating a 90 percent credible interval for β into a 90 percent credible interval for avoided health effect incidence

As in Section 5.2 above, the health effect will be taken to be mortality for ease of discussion in this section. The discussion is, however, generalizable to any health effect. For a given set of PM reductions in a given location, to any value of β there corresponds a predicted avoided mortality. There is therefore a distribution of avoided mortality corresponding to the distribution of β's. Ideally, the 5th and 95th percentiles of the estimated distribution of avoided mortality would compose the 90 percent credible interval around a point estimate of avoided mortality in the location of interest. If the concentration-response function is linear, the mortality predicted by the concentration-response function evaluated at the x percentile β would be the same as the x percentile of the distribution of mortality. That is, if β_x denotes the x percentile value of β from the distribution of β's, and Δy_x denotes the x percentile value of the distribution of avoided mortality from the corresponding distribution, then

$$\Delta y_x = y[e^{\beta_x \Delta PM} - 1] \quad .$$

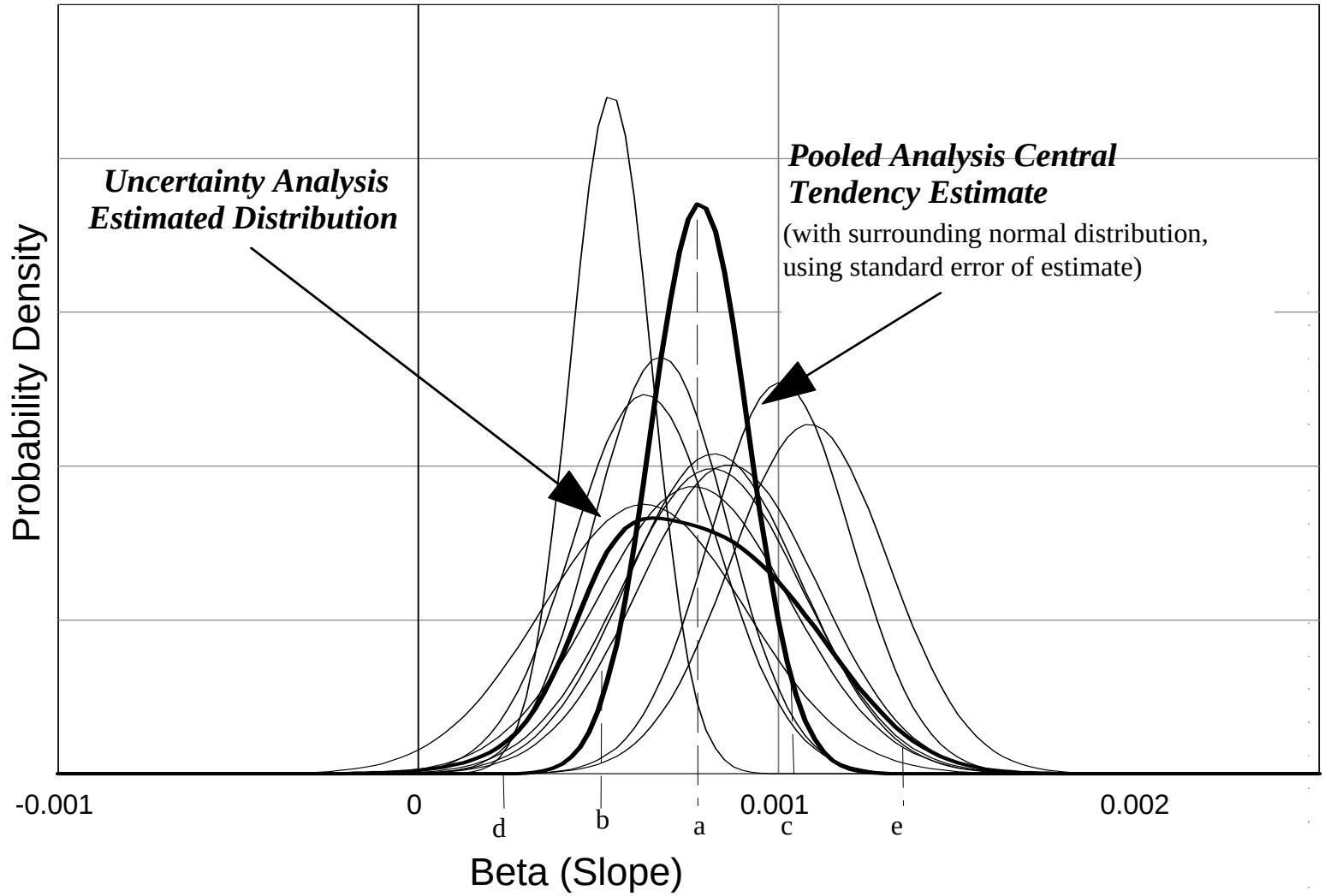
If, however, the concentration-response function is convex or concave, the equality becomes an inequality. Because the concentration-response functions are almost linear, however, the discrepancy will be very small, and the 5th and the 95th percentile β's from the estimated distribution of β's were applied to the air quality data in a given location (Philadelphia County or Southeast Los Angeles County) to obtain a close approximation to a 90 percent "credible interval" in avoided incidences of the given health effect in that location.

Exhibit 5.11: Probability Density of Beta (Slope) Coefficients
For PM-10 Mortality Studies in Ten Locations (Before Empirical Bayes Adjustments)



Curves labelled with only a city name come from Schwartz et al., 1996a.

Exhibit 5.12: Mortality Studies from ten Locations (Adjusted Using Empirical Bayes Method), Pooled Analysis Central Tendency Estimate, and Uncertainty Analysis Estimated Distribution
Results Based on Random Effects Weighting



a = Pooled analysis central tendency estimate = mean of uncertainty analysis estimated distribution
(b,c) = 95% confidence interval around the mean
(d,e) = 95% "credible interval" from uncertainty analysis distribution (i.e., 2.5 and 97.5 percentiles of the distribution)

6. Baseline Health Effects Incidence Rates

Incidence rates are required inputs for many, but not all, concentration-response functions. Many of the epidemiology studies used in this analysis directly estimate the percentage change in incidence (i.e., the relative risk), rather than the absolute number of cases for an endpoint. To estimate the number of PM-associated cases using these studies, it is necessary to know the baseline incidence, that is, the number of cases in a location *before* a change in PM air quality.

Incidence rates express the occurrence of a disease or event (e.g., asthma episode, death, hospital admission) in a specific period of time, usually per year. Rates are expressed either as a value per population group (e.g., the number of cases in Philadelphia County) or a value per number of people (e.g., number of cases per 10,000 residents), and may be age and sex specific. Incidence rates vary among geographic areas due to differences in population characteristics (e.g, age distribution) and factors promoting illness (e.g., smoking, air pollution levels).[15] The sizes of the populations in Philadelphia County and Southeast Los Angeles County that are relevant to the risk analyses reported here (i.e., the populations to which the baseline incidences refer) are given in Exhibit 6.1.

Exhibit 6.1. Relevant Population Sizes for Philadelphia County and Southeast Los Angeles County

Population	Philadelphia County	Southeast Los Angeles County
Total	1,586,000	3,636,000
Ages ≥ 65	240,800 (15.2%)	322,100 (8.9%)
Children, ages 8-12	102,900 (6.5%)	282,100 (7.8%)
Children, ages 10-12	61,700 (3.9%)	165,800 (4.6%)
Asthmatic Children, ages 9-11	3,900* (0.25%)	10,700* (0.29%)
Asthmatic African-American Children, ages 7-12	--	1,800* (0.05%)

*Incidences for asthmatic children were obtained using the national asthma prevalence among children (6.3%). The incidence of asthmatic African-American children ages 7-12 in Southeast L.A. County, for example, is 3,636,000 multiplied by {0.0937 (the proportion of the population that is ages 7-12) x 0.085 (the proportion of the population that is African-American) x 0.063 (the proportion of the national population of children that are asthmatic)}.

[15] Incidence rates also vary within a geographic area due to the same factors; however, statistics regarding within-city variations are rarely available and are not necessary for this analysis.

6.1. Sources of incidence data

Incidence rates are available for mortality (death rates) and for specific communicable diseases which state and local health departments are required to report to the federal government. None of the morbidity endpoints in the risk analysis are required to be reported to the federal government. In addition to the required federal reporting, many state and local health departments collect information on some additional endpoints. These most often are restricted to hospital admission or discharge diagnoses, which are collected to assist in planning medical services. Data may also be collected for particular studies of health issues of concern.

Although federal agencies collect incidence data on many of the endpoints covered in this report, their data are often available only at the national level (national averages), or at the regional or state level. When possible, state and local health departments and hospital planning commissions were contacted to obtain location-specific rates.

Estimates of location-specific baseline mortality rates for Philadelphia County and Southeast Los Angeles County were obtained from the National Center for Health Statistics (NCHS), a national repository for morbidity, mortality, and health services data. Baseline incidence rates for hospital admissions (for pneumonia, COPD, "total respiratory," congestive heart disease, and ischemic heart failure) were obtained for Philadelphia County from the Delaware Valley Hospital Council for 1993-1994 (fiscal year 1994), and for Los Angeles from California's Office of Statewide Health Planning and Development Data Users Support Group. Finally, in the absence of other sources of baseline incidence data for respiratory symptoms and acute bronchitis, baseline rates for these health endpoints were taken from the studies which estimated the concentration-response functions used for these endpoints in the risk analysis.

Baseline health effects incidence rates used in the risk analysis are given in Exhibit 6.2. In all cases, the incidence rates listed correspond to the ages of the populations studied in the relevant epidemiology studies, e.g., individuals over 65 years of age. The national incidence rates given in Exhibit 6.2 may differ from those given in the Criteria Document for several reasons, including differences in the years used and differences in the ICD codes included within a health effects category.

Exhibit 6.2. Baseline Health Effects Incidence Rates

Health Effect	Philadelphia County	Southeast Los Angeles County	National Average[a]
Short-Term Exposure Mortality[b] (per 100,000 general population/year)	1280	676	830
Long-Term Exposure Mortality (age 30 and older) (per 100,000 general population/year)	1154*	657*	--
Morbidity:			
A. Hospital Admissions (per 100,000 general population/year)			
Total respiratory hospital admissions[c] (all ages): ICD codes 466, 480-482, 485, 490-493	816	427	--
Total respiratory hospital admissions (65 and older): ICD codes 460-519	650	428	504
COPD admissions (65 and older): ICD codes 490-496	202	116	103
Pneumonia admissions (65 and older): ICD codes 480-487	257	205	229
Ischemic heart failure (65 and older): ICD codes 410-414	614	307	450
Congestive Heart Disease (65 and older): ICD code 428	487	197	231
B. Respiratory Symptoms (percent of relevant population)			
Lower Respiratory Symptoms (LRS) in children, ages 8-12 (number of cases of symptoms per day)	0.15%**	0.15%**	--
Lower Respiratory Symptoms (LRS) in asthmatic children, ages 9-11 (number of days of symptoms)	16%**	16%**	--
Shortness of breath (number of days) in asthmatic African-American children, ages 7-12	--	5.6%**	--
(Doctor diagnosed) acute bronchitis in children ages 10-12 per yr.	6.5%**	6.5%**	--

All incidence rates are rounded to the nearest unit.

a. National rates for hospital admissions for patients over 64 years of age were obtained from Vital and Health Statistics, Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1990. June, 1992. CDC. Hyattsville, Md. Each rate is based on the number of discharges divided by the 1990 population of 248,709,873 .

b. Mortality figures exclude suicide, homicide, and accidental death, which corresponds to the measures used in the epidemiological studies employed in this analysis.

c. Although a baseline incidence rate is not needed for calculating the incidence of total respiratory hospital admissions associated with PM (because the concentration-response function predicts cases rather than percent change), it is needed for calculating the PM-related percent change in total incidence.

*Although county-specific total mortality incidences (over all ages) were available for both Philadelphia and Los Angeles, age-specific mortality incidences were not available. Baseline mortality incidences among individuals aged 30 and over in Philadelphia and Southeast Los Angeles Counties were therefore estimated by applying national age-specific death rates to county-specific age distributions, and adjusting the resulting estimated age-specific incidences so that the estimated total incidences (including all ages) equaled the actual county-specific total incidences. For example, if the total of the estimated age-specific incidences obtained in this way was 5%

higher than the actual total incidence for a county, then each of the estimated age-specific incidences was multiplied by (1/1.05). Using this method, the baseline mortality incidences among individuals aged 30 and over were estimated to be 90% and 97% of the baseline incidences including all ages in Philadelphia County and Southeast Los Angeles County, respectively.
**Baseline incidence rates for respiratory symptoms were taken from the original studies.

7. Assessment of the Health Risks Associated with "As Is" PM Concentrations Above Background

7.1. Results and sensitivity analyses

The results of the first phase of the risk analysis, assessing the health risks associated with "as is" PM concentrations are given in Exhibit 7.1 for Philadelphia County in 1992-1993 and Exhibits 7.2 and 7.3 for Southeast Los Angeles County in 1995. Because Southeast Los Angeles County was not in attainment of current PM-10 standards in 1995, the health risks associated with "as is" PM concentrations in that location was assessed in two ways. First, the assessment was carried out using "as is" PM concentrations (Exhibit 7.2). Second, health risks were assessed using daily PM concentrations adjusted to simulate attainment of current standards (Exhibit 7.3). The method of adjusting daily PM concentrations to simulate attainment of current standards is described in Section 2.2.

All estimated incidences were rounded to the nearest 10, except lower respiratory symptoms, which are reported to the nearest 1000, and shortness of breath among African-American asthmatics, which is rounded to the nearest 100. All percentages were rounded to one decimal place. Rounding was done for convenience of presentation and is not intended to imply a particular level of precision.

There is substantial uncertainty surrounding all estimates of incidence associated with "as is" PM concentrations, in both Philadelphia County and Southeast Los Angeles County. The incidence of a health effect predicted to be associated with "as is" PM concentrations in a given location depends on the concentration-response function in that location. Because the true concentration-response functions (for the relevant health effects) are not known, they must be estimated. If concentration-response functions had been estimated for Philadelphia County and Southeast Los Angeles County specifically, then the only uncertainty associated with using these estimated concentration-response functions in the risk analyses for Philadelphia County and Southeast Los Angeles County would be the uncertainty as to how well the estimated concentration-response functions approximate the true concentration-response functions in these locations. This uncertainty is typically expressed as a 90 or 95 percent confidence interval around the estimate.

However, because concentration-response functions have, for most health endpoints, not been estimated for Philadelphia County or Southeast Los Angeles County specifically, concentration-response functions estimated in other locations, or a central tendency estimate derived by pooling these, have been used instead. This adds a second source of uncertainty to the risk analyses. If there is true geographic variability in the concentration-response function for a given health effect, then it is uncertain how well the concentration-response function in one location (or the mean of concentration-response functions in several locations) approximates that for a different location. (This is discussed in more detail in Section 3 and Section 5.2.)

To assess the total uncertainty surrounding the concentration-response function applied to a given location (e.g., Philadelphia County) in the risk analysis, in this case, the full range of possibilities of what the function in that location might be is characterized by estimating the *distribution* of possible values of the "slope" parameter, β, in the concentration-response function. This distribution is estimated based on the limited information from studies conducted in various locations throughout the U.S. Lacking further information, this distribution characterizes the range of possibilities of what the concentration-response function might be in a randomly selected location anywhere in the United States. If nothing more about a location is known, then, it is estimated that with 90 percent probability, β in that location lies between the 5th and the 95th percentiles of this estimated distribution of β's. This interval is referred to as the "90 percent credible interval" for any location in the U.S., including Philadelphia County or Southeast Los Angeles County. Each predicted PM-related incidence and each predicted PM-related percent of total incidence is accompanied by its associated 90 percent credible interval (in parentheses below it). In those cases in which the distribution of β's is estimated from only a single study, this 90 percent credible interval coincides with the 90 percent confidence interval around the estimate of the concentration-response function in the study location. The estimation of the distribution of β's from which to calculate the 90 percent credible interval is described in Section 5.2.1.

PM-related health effects incidence and percent of total incidence predicted in Southeast Los Angeles County are uniformly greater than those predicted in Philadelphia County. The generally higher pollution levels and greater population size in Southeast Los Angeles County are undoubtedly the primary reasons for this. In some instances (e.g., for lower respiratory symptoms among 8-12 year old children), however, the predictions of PM-related health effects incidence in Southeast Los Angeles County seem questionably high. It is important to bear in mind that most predictions are based on only a single study, and there are reasons to be cautious in accepting the results of any single study without corroboration from other studies. In addition, pollution levels in Southeast Los Angeles County are notably high. PM concentrations in that location exceeded the range of PM observations on which the estimation of the concentration-response function for lower respiratory symptoms among 8-12 year old children (Schwartz et al. 1994) was based. For example, the highest PM-10 concentration observed in the Schwartz study was 117 $\mu g/m^3$, as compared with a second highest PM-10 concentration of 193.4 $\mu g/m^3$ in Southeast Los Angeles County (Exhibit 4.12). The highest PM-2.5 concentration observed in the Schwartz study was 86 $\mu g/m^3$, as compared with a second highest PM-2.5 concentration of 106.2 $\mu g/m^3$ in Southeast Los Angeles County (Exhibit 4.13). It is possible that the Los Angeles PM concentrations exceeded the range on which the estimated concentration-response relationship is a plausible model (see Section 3.1.3). To the extent that this was the case, the incidence of PM-related health effects would have been overestimated for Southeast Los Angeles County. The numbers of days on which PM concentrations reported in Southeast Los Angeles County in 1995 exceeded maximum PM concentrations observed in studies estimating concentration-response functions is given, for each health endpoint considered in the risk analyses, in Exhibit 7.4.

Exhibit 7.1

Estimated Annual Health Risks Associated with "As Is" PM Concentrations in Philadelphia County, September 1992- August 1993 (for base case assumptions)

Health Effects*		Health Effects Associated with PM-10 Above Background**		Health Effects Associated with PM-2.5 Above Background**	
		Incidence	Percent of Total Incidence	Incidence	Percent of Total Incidence
Mortality	(A) Associated with short-term exposure (all ages)	220 (160 - 290)	1.1% (0.8 - 1.4)	370 (230 - 510)	1.8% (1.1 - 2.5)
	(B) Assoc. with long-term exposure (age 30 and over) (51 locations)	-- -- -- -- -- --	-- -- -- -- -- --	860 (540 - 1170)	4.7% (2.9 - 6.4)
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	-- -- -- -- -- --	-- -- -- -- -- --	260 (70 - 450)	2.0% (0.5 - 3.5)
	(D) Total respiratory (>64 years old)	250 (150 - 340)	2.4% (1.5 - 3.3)	-- -- -- -- -- --	-- -- -- -- -- --
	(E) COPD (>64 years old)	120 (80 - 150)	3.7% (2.5 - 4.7)	-- -- -- -- -- --	-- -- -- -- -- --
	(F) Pneumonia (>64 years old)	80 (50 - 100)	1.9% (1.3 - 2.6)	-- -- -- -- -- --	-- -- -- -- -- --
Hospital Admissions Cardiac	(G) Ischemic Heart Disease *** (>64 years old)	80 (30 - 120)	0.8% (0.3 - 1.3)	70 (30 - 120)	0.7% (0.3 - 1.2)
	(H) Congestive Heart Failure *** (>64 years old)	110 (50 - 160)	1.4% (0.7 - 2.1)	100 (50 - 150)	1.3% (0.6 - 2.0)
Lower Respiratory Symptoms in Children****	(I) Lower Respiratory Symptoms (# of cases) (8-12 year olds)	< 10000 > (8000 - 11000)	17.5% (15.3 - 19.6)	< 11000 > (6000 - 15000)	20.0% (10.3 - 28.2)
	(J) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)	< 16000 > (6000 - 25000)	6.8% (2.4 - 10.9)	-- -- -- -- -- --	-- -- -- -- -- --
	(K) Doctor-diagnosed Acute Bronchitis associated with long-term exposure (10-12 year olds)	< 190 > (20 - 370)	0.3% (0.0 - 0.6)	-- -- -- -- -- --	-- -- -- -- -- --

* Health effects are associated with short-term exposure to PM, unless otherwise specified.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Background PM-10 is assumed to be 8 ug/m3; background PM-2.5 is assumed to be 3.5 ug/m3.

*** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

****Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates.

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) Functions:
(A) PM-10 C-R function based on pooled results from studies in 10 locations; PM-2.5 C-R function based on pool results from studies in six locations.
(B) Pope et al., 1995
(C) Thurston, et al., 1994
(D) PM-10 C-R based on pooled results from 4 functions
(E) PM-10 C-R based on pooled results from 4 functions
(F) PM-10 C-R based on pooled results from 4 functions
(G) Schwartz & Morris, 1995
(H) Schwartz & Morris, 1995
(I) Schwartz, et al., 1994
(J) Pope et al., 1991
(K) Dockery et al., 1989

Exhibit 7.2

Estimated Annual Health Risks Associated with "As Is" PM Concentrations in Southeast Los Angeles County, 1995* (for base case assumptions)

Health Effects**			Health Effects Associated with PM-10 Above Background***		Health Effects Associated with PM-2.5 Above Background***	
			Incidence	Percent of Total Incidence	Incidence	Percent of Total Incidence
Mortality	(A) Associated with short-term exposure (all ages)		800 (570 - 1020)	3.3% (2.3 - 4.1)	900 (540 - 1230)	3.7% (2.2 - 5.0)
	(B) Associated with short-term exposure (all ages; study done in Los Angeles)		400 (40 - 750)	1.6% (0.2 - 3.1)	-- -- -- -- -- --	-- -- -- -- -- --
	(C) Associated with long-term exposure (age 30 and over; 51 locations)		-- -- -- -- -- --	-- -- -- -- -- --	2,800 (1800-3800)	11.9% (7.5 - 16.0)
Hospital Admissions Respiratory	(D) Total Respiratory (all ages)		-- -- -- -- -- --	-- -- -- -- -- --	1,200 (330 - 2080)	7.7% (2.1 - 13.4)
	(E) Total Respiratory (>64 years old)		1,070 (660 - 1460)	6.9% (4.2 - 9.4)	-- -- -- -- -- --	-- -- -- -- -- --
		(F) COPD (>64 years old)	440 (310 - 560)	10.3% (7.3 - 13.1)	-- -- -- -- -- --	-- -- -- -- -- --
		(G) Pneumonia (>64 years old)	420 (290 - 550)	5.6% (3.9 - 7.3)	-- -- -- -- -- --	-- -- -- -- -- --
Hospital Admissions Cardiac	(H) Ischemic Heart Disease**** (>64 years old)		260 (100 - 420)	2.3% (0.9 - 3.7)	160 (60 - 260)	1.4% (0.6 - 2.3)
	(I) Congestive Heart Failure**** (>64 years old)		290 (140 - 430)	4.1% (2.0 - 6.1)	180 (90 - 270)	2.5% (1.2 - 3.8)
Lower Respiratory Symptoms in Children *****	(J) Lower Respiratory Symptoms (# of cases) (8-12 year olds)		< 62000 > (56000 - 68000)	41.4% (37.2 - 45.2)	< 51000 > (28000 - 68000)	34.4% (19.1 - 45.7)
	(K) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)		< 115000 > (43000 - 175000)	18.4% (6.9 - 28.0)	-- -- -- -- -- --	-- -- -- -- -- --
	(L) Days of shortness of breath (7-12 year old African American asthmatics in Los Angeles)		< 7200 > (2400 - 10900)	19.3% (6.4 - 29.2)	-- -- -- -- -- --	-- -- -- -- -- --
	(L) Doctor-diagnosed Acute Bronchitis associated with long-term exposure (10-12 year olds)		< 5090 > (680 - 7750)	3.1% (0.4 - 4.7)	-- -- -- -- -- --	-- -- -- -- -- --

* Southeast Los Angeles County was not in attainment of current PM-10 standards (50 ug/m3 annual average standard and 150 ug/m3 daily standard) in 1995. Figures shown use the actual reported concentrations.

** Health effects are associated with short-term exposure to PM, unless otherwise specified.

*** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Background PM-10 is assumed to be 6.0 ug/m3 and background PM-2.5 is assumed to be 2.5 ug/m3.

**** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

*****Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates.

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) Functions:
(A) PM-10 C-R function based on pooled results from studies in 10 locations; PM-2.5 C-R function based on pooled results from studies in six locations.
(B) Kinney et al.,1995
(C) Pope et al., 1995
(D) Thurston, et al., 1994
(E) PM-10 C-R based on pooled results from 4 functions
(F) PM-10 C-R based on pooled results from 4 functions
(G) PM-10 C-R based on pooled results from 4 functions
(H) Schwartz & Morris, 1995
(I) Schwartz & Morris, 1995
(J) Schwartz, et al., 1994
(K) Pope et al., 1991
(L) Dockery et al., 1989

Exhibit 7.3

Estimated Annual Health Risks Associated with Attainment of Current Standards in Southeast Los Angeles County, 1995* (for base case assumptions)

Health Effects**			Health Effects Associated with PM-10 Above Background***		Health Effects Associated with PM-2.5 Above Background***	
			Incidence	Percent of Total Incidence	Incidence	Percent of Total Incidence
Mortality	(A) Associated with short-term exposure (all ages)		630 (450 - 800)	2.6% (1.8 - 3.3)	710 (430 - 970)	2.9% (1.7 - 3.9)
	(B) Associated with short-term exposure (all ages; study done in Los Angeles)		290 (30 - 550)	1.2% (0.1 - 2.2)	-- -- -- -- -- --	-- -- -- -- -- --
	(C) Associated with long-term exposure (age 30 and over; 51 locations)		-- -- -- -- -- --	-- -- -- -- -- --	2,050 (1250-2690)	8.6% (5.4 - 11.7)
Hospital Admissions Respiratory	(D) Total Respiratory (all ages)		-- -- -- -- -- --	-- -- -- -- -- --	940 (250 - 1630)	6.1% (1.6 - 10.5)
	(E) Total Respiratory (>64 years old)		840 (520 - 1160)	5.4% (3.3 - 7.4)	-- -- -- -- -- --	-- -- -- -- -- --
		(F) COPD (>64 years old)	350 (240 - 440)	8.2% (5.8 - 10.5)	-- -- -- -- -- --	-- -- -- -- -- --
		(G) Pneumonia (>64 years old)	330 (230 - 430)	4.4% (3.1 - 5.8)	-- -- -- -- -- --	-- -- -- -- -- --
Hospital Admissions Cardiac	(H) Ischemic Heart Disease**** (>64 years old)		200 (80 - 330)	1.8% (0.7 - 2.9)	130 (50 - 200)	1.1% (0.4 - 1.8)
	(I) Congestive Heart Failure**** (>64 years old)		230 (110 - 340)	3.2% (1.5 - 4.8)	140 (70 - 210)	2.0% (1.0 - 3.0)
Lower Respiratory Symptoms in Children *****	(J) Lower Respiratory Symptoms (# of cases) (8-12 year olds)		< 52000 > (46000 - 57000)	34.8% (31.0 - 38.4)	< 43000 > (23000 - 58000)	28.7% (15.4 - 39.0)
	(K) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)		< 93000 > (34000 - 143000)	14.9% (5.5 - 23.0)	-- -- -- -- -- --	-- -- -- -- -- --
	(L) Days of shortness of breath (7-12 year old African American asthmatics in Los Angeles)		< 5200 > (1700 - 8100)	14.1% (4.6 - 21.8)	-- -- -- -- -- --	-- -- -- -- -- --
	(L) Doctor-diagnosed Acute Bronchitis associated with long-term exposure (10-12 year olds)		< 3760 > (470 - 6190)	2.3% (0.3 - 3.7)	-- -- -- -- -- --	-- -- -- -- -- --

* Southeast Los Angeles County was not in attainment of current PM-10 standards (50 ug/m3 annual average standard and 150 ug/m3 daily standard) in 1995. "As is" daily PM-10 concentrations were first rolled back to simulate attainment of these standards. "As is" daily PM-2.5 concentrations were rolled back by the same percent as daily PM-10 concentrations. See text in Chapter VI for details.

** Health effects are associated with short-term exposure to PM, unless otherwise specified.

*** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Background PM-10 is assumed to be 6.0 ug/m3 and background PM-2.5 is assumed to be 2.5 ug/m3.

**** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

*****Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) Functions:

(A) PM-10 C-R function based on pooled results from studies in 10 locations; PM-2.5 C-R function based on pooled results from studies in six locations.
(B) Kinney et al.,1995
(C) Pope et al., 1995
(D) Thurston, et al., 1994
(E) PM-10 C-R based on pooled results from 4 functions
(F) PM-10 C-R based on pooled results from 4 functions
(G) PM-10 C-R based on pooled results from 4 functions
(H) Schwartz & Morris, 1995
(I) Schwartz & Morris, 1995
(J) Schwartz, et al., 1994
(K) Pope et al., 1991
(L) Dockery et al., 1989

Exhibit 7.4

Number of Days on Which PM Concentration Reported in Southeast Los Angeles County, 1995 Exceeds Maximum Observed PM in Studies for Each Endpoint

Health Effects			Maximum Concentration Observed in Study (in ug/m3)	Number of days with AQ data on which Reported Concentration Exceeds the Maximum Observed in Studies	
				PM-10	PM-2.5
Mortality (all ages)	(A) Associated with short-term exposure		251 (PM-10) 170 (PM-2.5)**	0	0
	(B) Associated with short-term exposure (study done in Los Angeles)		177 (PM-10)	2	n/a
	(C) Associated with long-term exposure (51 locations)		34 (PM-2.5)	n/a	falls within range
Hospital Admissions Respiratory	(D) Total Respiratory (all ages)		66 (PM-2.5)	n/a	22
	(E) Total Respiratory* (>64 years old)		83 (PM-10)*	33	12
		(F) COPD* (>64 years old)	83 (PM-10)*	33	12
		(G) Pneumonia* (>64 years old)	83 (PM-10)*	33	12
Hospital Admissions Cardiac	(H) Ischemic Heart Disease* (>64 years old)		83 (PM-10)*	33	12
	(I) Congestive Heart Failure* (>64 years old)		83 (PM-10)*	33	12
Lower Respiratory Symptoms in Children	(J) Lower Respiratory Symptoms (# of cases) (8-12 year olds)		117 (PM-10) 86 (PM-2.5)	16	10
	(K) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)		195 (PM-10)	1	n/a
	(L) Days of shortness of breath (7-12 year old African American asthmatics in Los Angeles)		101 (PM-10)	19	n/a
	(L) Doctor-diagnosed Acute Bronchitis associated with long-term exposure (10-12 year olds)		59 (PM-10)***	falls within range	n/a

Southeast Los Angeles AQ Data, 1995 Composite monitor
Annual average PM-10: 49 ug/m3
Annual average PM-2.5: 29 ug/m3
Daily Max PM-10: 197 ug/m3
Daily Max PM-2.5: 120 ug/m3

* Based on reported 90th percentile of distribution reported in study.
** Based on reported 95th percentile of distribution reported in study.
*** Based on reported PM-15 distribution.

Sources of Concentration-Response (C-R) Functions:
(A) PM-10 C-R function based on pooled results from studies in 10 locations; PM-2.5 C-R function based on pooled results from studies in six locations.
(B) Kinney et al.,1995
(C) Pope et al., 1995
(D) Thurston, et al., 1994
(E) PM-10 C-R based on pooled results from 4 functions
(F) PM-10 C-R based on pooled results from 4 functions
(G) PM-10 C-R based on pooled results from 4 functions
(H) Schwartz & Morris, 1995
(I) Schwartz & Morris, 1995
(J) Schwartz, et al., 1994
(K) Pope et al., 1991
(L) Dockery et al., 1989

Another reason that the estimated percentage of lower respiratory symptoms may be so large is that the original study of lower respiratory symptoms among a general population of 8-12 year old children (Schwartz et al. 1994) restricted the analysis to a period from April to August. During these months, the respiratory symptoms incidences from other causes are at a minimum. Thus, applying the changes observed in the odds ratio associated with PM over these months across an entire year could result in an overestimate of the percentage incidence of new cases of respiratory symptoms overall.

The effect of other pollutants, such as ozone, as confounders or effects modifiers, would also be higher in the summer months and could possibly lead to some overestimation of effects. Schwartz et al.1994 indicates that a general consistency of findings in both summer studies in the Six Cities and winter studies in Utah Valley suggests that the association with respiratory symptoms is not limited to photochemically produced aerosols. However, the consistency between studies is greater for cough than for lower respiratory symptoms, the endpoint examined in this analysis (Schwartz et at. 1994, Table 6). Thus questions still exist as to whether factors such as the differing prevalence of respiratory symptoms between winter and summer or effects modification by ozone may play some role in the large odds ratio for lower respiratory symptoms predicted by the Six Cities study (Schwartz et al. 1994).

As seen in Exhibits 7.2 and 7.3, Pope et al. (1991) also examined lower respiratory symptoms among asthmatic children (a possible sensitive subgroup) in the Utah Valley, and found significantly lower PM-associated incidence. Ostro et al. (1995) examined shortness of breath among African-American asthmatic children and estimated a PM effect roughly 50 percent stronger than that from the Utah Valley. Shortness of breath might be considered a less severe effect than "lower respiratory symptoms" (and therefore be exacerbated by lower levels of PM), and African-Americans may be a sensitive subgroup for PM effects on asthmatic symptoms. Both of these factors might lead one to expect a somewhat strong PM effect on shortness of breath in African-American asthmatics, as observed. The results from Ostro et al. (1995) might therefore be considered consistent with the symptom results presented in Exhibits 7.2 and 7.3.

Although in most cases concentration-response functions estimated in the sample locations were not available, for short-term exposure mortality there is a single preliminary (unpublished) study carried out in Philadelphia for PM-2.5 and a single (published) study (Kinney et al. 1995) carried out in Los Angeles for PM-10. The results from these functions are compared with the results using the corresponding pooled analysis functions for short-term exposure mortality in Exhibit 7.5.

Exhibit 7.5. Comparison of Predicted Short-Term Exposure Mortality Incidence Using Pooled Analysis Functions and Location-Specific Functions

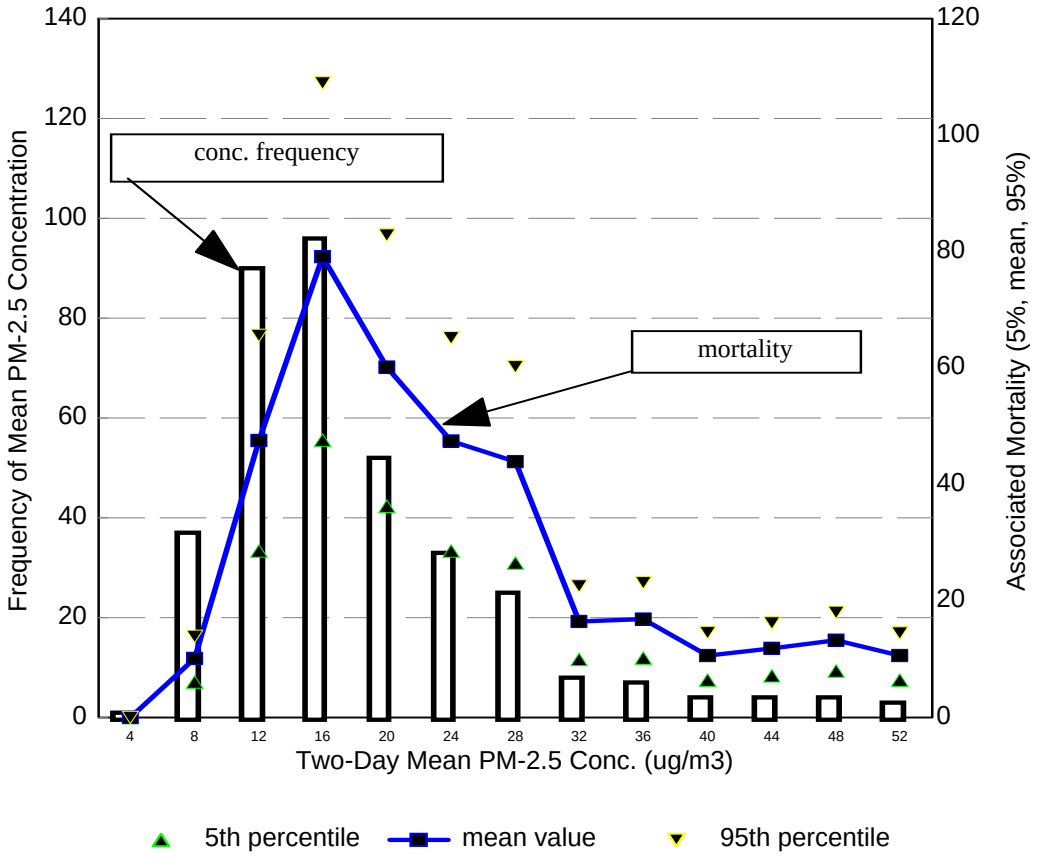
Concentration-Response Function	Health Effects Associated with PM-10 Above Background in Southeast Los Angeles County		Health Effects Associated with PM-2.5 Above Background in Philadelphia County	
	Incidence	Percent of Total Incidence	Incidence	Percent of Total Incidence
PM-10 pooled analysis function based on 10 studies	800 (570 - 1020)	3.3% (2.3 - 4.1)	---	---
Study done in Los Angeles (Kinney et al., 1995)	400 (40 - 750)	1.6% (0.2 - 3.1)	---	---
PM-2.5 pooled analysis function based on 10 studies	---	---	370 (220 - 510)	1.8% (1.1 - 2.5)
Study done in Philadelphia (Dockery et al., Abstract, 1996)*	---	---	510 (190 - 840)	2.5% (0.9 - 4.1)

*This study is as yet only in the form of an unpublished abstract and is therefore not included among the studies in the Exhibits of results.

Exhibit 7.6 provides a different way of looking at the "as is" results for Philadelphia County. The histogram shows the number of days on which PM-2.5 is within a given range in the Philadelphia County data. The line shows the number of deaths associated with PM-2.5 on those days. The number of deaths associated with PM-2.5 depends both on the number of days at a given concentration and on the concentration itself. Therefore the bulk of PM-related mortality is associated with PM-2.5 concentrations of between 12 and 24 $\mu g/m^3$ simply because most days have concentrations in that range. The small number of days with large concentrations of PM-2.5, although they contribute more deaths per day, do not contribute as large a number of deaths overall.

Several sensitivity analyses were performed to assess the sensitivity of the results of analyses in the first phase of the risk analysis to various assumptions underlying the analyses. The sensitivity analyses and the exhibits presenting their results are summarized in Exhibit 7.7.

Exhibit 7.6
Distributions of Two-Day Mean PM-2.5 Concentration and of Mortality Associated With the Excess of Those Concentrations Above Background
Philadelphia, September 1992 - August 1993



The 5th percentile, mean, an 95th percentile mortality concentration-response functions (Betas) are from the distribution of Betas based on the empirical-Bayes-adjusted Betas (see Section 5.2.1) from Schwartz et al., 1996a.

Frequencies shown for the 364 days on which two-day mean PM-2.5concentrations were actually available. Bar width is 4 ug/m3, including allconcentrations less than the value indicated under the bar and including,as the lower bound, the concentration value under the next lowest bar. Associatedmortality figures are for a full year, calculated assuming that the distributionof concentrations on days with available data is representative of thedistribution of concentrations for the entire year.

Exhibit 7.7. Summary of Sensitivity Analyses Associated with "As Is" Phase of RiskAnalysis

Sensitivity Analysis of:	Exhibit(s)
the effect of alternative background levels on predicted health effects associated with "as is" PM-10 and PM-2.5 in Philadelphia County	Exhibits 7.9 and 7.10
the effect of alternative background levels on predicted health effects associated with "as is" PM-10 and PM-2.5 in Los Angeles County	Exhibits 7.11 and 7.12
the effect of alternative cutpoint models on predicted health effects associated with "as is" PM-10, using two different methods of slope adjustment in Philadelphia County	Exhibits 7.13 and 7.14
the effect of alternative cutpoint models on predicted health effects associated with "as is" PM-2.5, using two different methods of slope adjustment in Philadelphia County	Exhibits 7.15 and 7.16
the effect of alternative cutpoint models on predicted health effects associated with "as is" PM-10, using two different methods of slope adjustment in Los Angeles County	Exhibits 7.17 and 7.18
the effect of alternative cutpoint models on predicted health effects associated with "as is" PM-2.5, using two different methods of slope adjustment in Los Angeles County	Exhibits 7.19 and 7.20
the effect of combining different averaging times in pooled short-term exposure mortality functions on predicted health effects associated with "as is" PM-10 in Philadelphia County	Exhibit 7.21
the effect of using concentration-response functions for short-term mortality from different individual studies on predicted health effects associated with "as is" PM-10 and PM-2.5 in Philadelphia County	Exhibit 7.22
the effect of copollutants on relative risks for a change of 50 $\mu g/m^3$ PM-10 or 25 $\mu g/m^3$ PM-2.5*	Exhibit 7.23
the effect of copollutants on predicted health effects associated with "as is" PM in Philadelphia County	Exhibit 7.24
the effect of copollutants on predicted health effects associated with PM after meeting current PM-10 standards in Los Angeles County	Exhibit 7.25
the effect of differing cutpoints on estimated mortality associated with long-term exposure to PM-2.5 (no slope adjustment) in Philadelphia County	Exhibit 7.26
the effect of historical previous air quality on estimated mortality associated with long-term exposure to PM-2.5 (in Philadelphia County)	Exhibit 7.27

*This sensitivity analysis is not location-specific. It examines the effect of having copollutants in the models estimated by epidemiological studies on relative risks associated with specified changes in PM concentration.

The sensitivity analyses of alternative cutpoint models considered the effect of using alternative concentration-response models. The exponential model estimated by most epidemiological studies (and therefore used in most of the risk analyses (see Section 2,

equation 1)) assumes that there is no PM level at which the relationship between PM and the health effect fundamentally changes. Using the relative risk version of the model (see Appendix 3, equation 9), this means that there is a linear relationship between the natural logarithm of the relative risk, ln(RR), and PM, as shown in Exhibit 7.18. As an alternative to this simple linear relationship, a "hockey stick" model was considered. The hockey stick model is determined by (1) a cutpoint, below which the model is a horizontal line (i.e., the slope is zero), and (2) the positive slope of the line corresponding to PM concentrations greater than the cutpoint. Changing the cutpoint and/or the positive slope results in different hockey stick models.

Exhibit 7.7a compares the results of using a cutpoint with no slope adjustment and of using a cutpoint while doubling the slope. In each case, there is no additional risk at or below the cutpoint; that is, the relative risk at or below the cutpoint is equal to one (so that the natural logarithm of the relative risk is zero). Appendix E of the Staff Paper (EPA, 1996b) discusses the choice of the particular cutpoints presented. Philadelphia "as is" results, and Los Angeles results assuming attainment of current standards are examined. The base case results (which require no slope adjustment, since no cutpoint is imposed) are provided for comparison. Doubling the slope roughly doubles the estimate of mortality associated with short-term exposure to PM-2.5.

Exhibit 7.7a. Comparison of the Effect of Cutpoints with and without Slope Adjustment. Mortality Associated with Short-Term Exposure to PM-2.5.

Cutpoint	Philadelphia County		Southeast Los Angeles County	
	no adjustment	slope doubled	no adjustment	slope doubled
Background ("Base Case")	1.8% (1.1, 4.4)	---	2.9% (1.7, 3.9)	---
10 $\mu g/m^3$	1.0% (0.6, 1.3)	1.9% (1.2, 2.6)	1.9% (1.1, 2.6)	3.7% (2.2, 5.0)
18 $\mu g/m^3$	0.4% (0.2, 0.5)	0.7% (0.4, 1.0)	1.1% (0.7, 1.5)	2.2% (1.3, 3.0)
30 $\mu g/m^3$	0.09% (0.06, 0.13)	0.2% (0.1, 0.3)	0.5% (0.3, 0.7)	1.0% (0.6, 1.4)

Exhibits 7.13 through 7.20 provide another perspective on slope adjustment. Two different methods of adjusting the positive slope of the "hockey stick" concentration-response function were used. The methods result in different slope adjustments being applied when different cutpoints are selected.

The first slope adjustment method preserves the area of the triangle formed by (1) the x-axis, (2) a vertical line at the maximum observed PM concentration in the study that estimated the original exponential concentration-response function, and (3) the original concentration-response function itself (i.e., the linear relationship between ln(RR) and PM). That is, the slope of the hockey stick is adjusted so that the area under the new line (down to the x-axis and out to the vertical line at the maximum observed PM level in the study) is the same as the corresponding area under the original function. The original function spans a wider range on the x-axis, and a smaller range on the y-axis, than the adjusted function. That is, to compensate for fewer PM-associated health effects at low concentrations (and no effects at all below the cutpoint), the adjusted function rises more quickly than does the original function.

If the actual PM concentrations where the original function was estimated were evenly distributed along the x-axis, then the area under the original function (the triangle described above) is an approximation of the total health effects predicted by the original function. In general, however, distributions of PM concentrations tend to be skewed to lower concentrations, with relatively long tails. A better approximation would take account of this. However, such an approximation would predict an even steeper slope than the method used here, since the days with lower PM concentrations would need to account for the health effects previously accounted for by days with high PM concentrations.

The second slope adjustment method assumes that the relative risk associated with the maximum observed concentration remains the same in the hockey stick model as in the original model that did not assume a cutpoint. Thus the positive-sloped portion of the hockey stick extends from the cutpoint to the relative risk achieved by the original function at the maximum observed concentration. This method adjusts the slope by less than the first method.

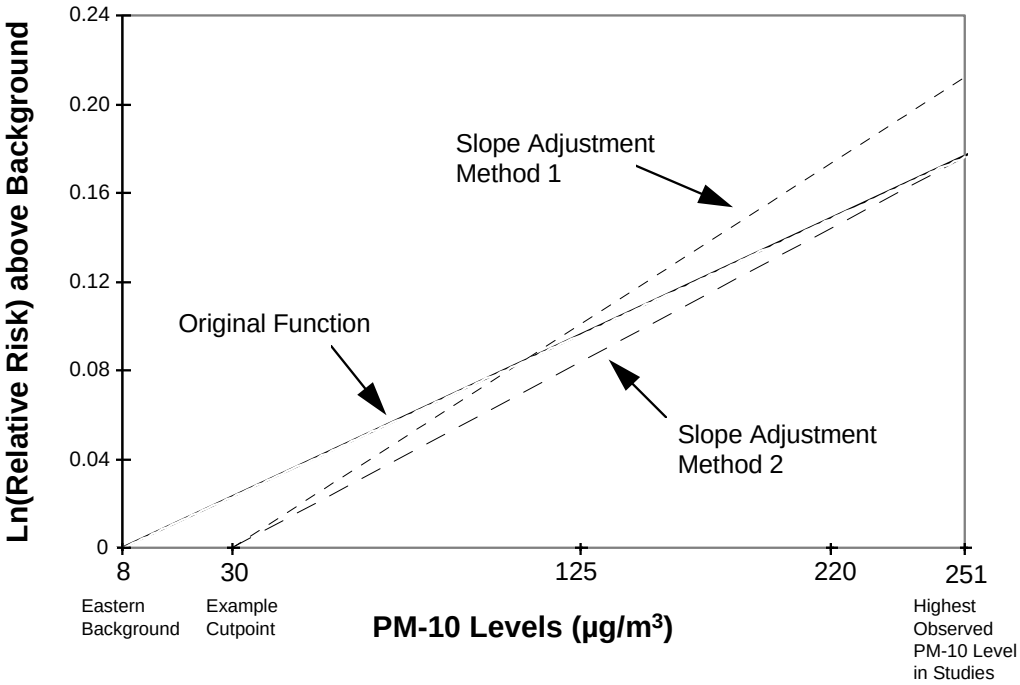
Exhibit 7.8 illustrates the two slope adjustment methods. It is important to keep in mind that these adjustment methods are illustrative, rather than definitive. Different choices of slope adjustments can yield substantially different results. Proper evaluation of the effect of cutpoints would require re-analysis of original health and air quality data, as noted above.

Exhibits 7.22, 7.23, and 7.24 show the effect of including copollutants in the analysis. The figures presented in these exhibits derive from models that include PM and one other pollutant at a time. No models considering three or more pollutants at a time are included.

Exhibit 7.8

Sensitivity Analysis: Slope Adjustment

PM-10 Pooled Mortality Function



Relative Risks shown are the risks associated with elevated PM-10 levels relative to the risks associated with the background PM level (8 µg/m^3).

Exhibit 7.9

Sensitivity Analysis: The Effect of Alternative Background Levels on Predicted Health Effects Associated With "As-Is" PM-10 Philadelphia County, September 1992 - August 1993

Health Effects*			Percent of Total Incidence Associated with PM-10 Above Background**		
			BASE CASE Background = 8 µg/m3	Background = 5 µg/m3	Background = 11 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure		**1.1% (0.8 - 1.4)**	1.3% (1.0 - 1.7)	0.9% (0.6 - 1.1)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)		**2.4% (1.5 - 3.3)**	2.87% (1.8 - 4.0)	1.9% (1.2 - 2.7)
		(C) COPD (>64 years old)	**3.7% (2.5 - 4.7)**	4.4% (3.1 - 5.7)	3.0% (2.1 - 3.8)
		(D) Pneumonia (>64 years old)	**1.9% (1.3 - 2.6)**	2.3% (1.6 - 3.1)	1.6% (1.1 - 2.1)
Hospital Admissions Cardiac	(E) Ischemic Heart Disease (>64 years old)		**0.8% (0.3 - 1.3)**	1.0% (0.4 - 1.5)	0.6% (0.2 - 1.0)
	(F) Congestive Heart Failure (>64 years old)		**1.4% (0.7 - 2.1)**	1.7% (0.8 - 2.5)	1.1% (0.5 - 1.7)
Lower Respiratory Symptoms in Children	(G) Lower Respiratory Symptoms (# of cases) (8 - 12 year olds)		**17.5% (15.3 - 19.6)**	20.8% (18.2 - 23.3)	14.2% (12.4 - 15.9)
	(H) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)		**6.8% (2.4 - 10.9)**	8.2% (2.9 - 13.0)	5.5% (2.0 - 8.8)

* Health effects associated with short-term exposure to PM.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Background PM-10 is assumed to be 8 ug/m3 .

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) Functions:

(A) PM-10 C-R function based on pooled results from studies in 10 locations.

(B) PM-10 C-R based on pooled results from 4 functions

(C) PM-10 C-R based on pooled results from 4 functions

(D) PM-10 C-R based on pooled results from 4 functions

(E) Schwartz & Morris, 1995

(F) Schwartz & Morris, 1995

(G) Schwartz, et al., 1994

(H) Pope et al., 1991

Exhibit 7.10

Sensitivity Analysis: The Effect of Alternative Background Levels on Predicted Health Effects Associated With "As-Is" PM-2.5 Philadelphia County, September 1992 - August 1993

Health Effects*		Percent of Total Incidence Associated with PM-2.5 Above Background**		
		BASE CASE Background = 3.5 µg/m3	Background = 2.0 µg/m3	Background = 5.0 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.8%** **(1.1 - 2.5)**	2.1% (1.2 - 2.8)	1.6% (1.0 - 2.2)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**2.0%** **(0.5 - 3.5)**	2.2% (0.6 - 3.9)	1.8% (0.5 - 3.1)
Hospital Admissions Cardiac (>64 years old)	(C) Ischemic Heart Disease***	**0.7%** **(0.3 - 1.2)**	0.8% (0.3 - 1.3)	0.7% (0.3 - 1.1)
	(D) Congestive Heart Failure***	**1.3%** **(0.6 - 2.0)**	1.5% (0.7 - 2.2)	1.2% (0.6 - 1.8)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases) (8-12 years old)	**20.0%** **(10.3 - 28.2)**	22.2% (11.5 - 31.1)	17.8% (9.2 - 25.2)

* Health effects associated with short-term exposure to PM.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Background PM-2.5 is assumed to be 3.5 ug/m3.

*** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) Functions:

(A) PM-2.5 C-R function based on pooled results from 6 locations.

(B) Thurston, et al., 1994

(C) Schwartz & Morris, 1995

(D) Schwartz & Morris, 1995

(E) Schwartz, et al., 1994

Exhibit 7.11

Sensitivity Analysis: The Effect of Alternative Background Levels on Predicted Health Effects Associated With PM-10 After Attainment of Current Standards* Southeast Los Angeles County, 1995

Health Effects**			Percent of Total Incidence Associated with PM-10 Above Background***		
			BASE CASE Background = 6 µg/m3	Background = 4 µg/m3	Background = 8 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure		**2.6%** **(1.8 - 3.3)**	2.7% (1.9 - 3.4)	2.4% (1.7 - 3.1)
	(A') Associated with short-term exposure (Study done in Los Angeles)		**1.2%** **(0.1 - 2.2)**	1.2% (0.1 - 2.2)	1.2% (0.1 - 2.2)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)		**5.4%** **(3.3 - 7.4)**	5.7% (3.5 - 7.9)	5.1% (3.1 - 7.0)
		(C) COPD (>64 years old)	**8.2%** **(5.8 - 10.5)**	8.7% (6.1 - 11.1)	7.7% (5.4 - 9.9)
		(D) Pneumonia (>64 years old)	**4.4%** **(3.1 - 5.8)**	4.7% (3.3 - 6.1)	4.2% (2.9 - 5.5)
Hospital Admissions Cardiac	(E) Ischemic Heart Disease (>64 years old)		**1.8%** **(0.7 - 2.9)**	1.9% (0.7 - 3.1)	1.7% (0.7 - 2.8)
	(F) Congestive Heart Failure (>64 years old)		**3.2%** **(1.5 - 4.8)**	3.4% (1.6 - 5.1)	3.0% (1.5 - 4.5)
Lower Respiratory Symptoms in Children	(G) Lower Respiratory Symptoms (# of cases) (8 - 12 year olds)		**34.8%** **(31.0 - 38.4)**	36.7% (32.7 - 40.3)	33.0% (29.4 - 36.3)
	(H) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)		**14.9%** **(5.5 - 23.0)**	15.7% (5.8 - 24.2)	14.0% (5.2 - 21.7)

* Current standards are 50 ug/m3 annual average PM-10, 150 ug/m3 second daily maximum PM-10.

** Health effects associated with short-term exposure to PM.

*** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) Functions:

(A) PM-10 C-R function based on pooled results from studies in 10 locations.

(A') Kinney et al., 1995

(B) PM-10 C-R based on pooled results from 4 functions

(C) PM-10 C-R based on pooled results from 4 functions

(D) PM-10 C-R based on pooled results from 4 functions

(E) Schwartz & Morris, 1995

(F) Schwartz & Morris, 1995

(G) Schwartz, et al., 1994

(H) Pope et al., 1991

Exhibit 7.12

Sensitivity Analysis: The Effect of Alternative Background Levels on Predicted Health Effects Associated With PM-2.5 After Attainment of Current Standards* Southeast Los Angeles County, 1995

Health Effects**		Percent of Total Incidence Associated with PM-2.5 Above Background***		
		BASE CASE Background = 3.5 µg/m3	Background = 1.0 µg/m3	Background = 4.0 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**2.9% (1.7 - 3.9)**	3.1% (1.9 - 4.2)	2.7% (1.6 - 3.7)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**6.1% (1.6 - 10.5)**	6.5% (1.8 - 11.2)	5.6% (1.5 - 9.7)
Hospital Admissions Cardiac (>64 years old)	(C) Ischemic Heart Disease****	**1.1% (0.4 - 1.8)**	1.2% (0.5 - 2.0)	1.1% (0.4 - 1.7)
	(D) Congestive Heart Failure****	**2.0% (1.0 - 3.0)**	2.1% (1.0 - 3.2)	1.8% (0.9 - 2.8)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases) (8-12 years old)	**28.7% (15.4 - 39.0)**	30.6% (16.5 - 41.4)	26.7% (14.4 - 36.3)

* Current standards are 50 ug/m3 annual average PM-10, 150 ug/m3 second daily maximum PM

** Health effects associated with short-term exposure to PM.

*** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level

**** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) functions:
(A) PM-2.5 C-R function based on pooled results from 6 locations.
(B) Thurston, et al., 1994
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz, et al., 1994

Exhibit 7.13

Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With "As-Is" PM-10 Slope Adjustment Method 1* Philadelphia County, September 1992 - August 1993

Health Effects**		Percent of Total Incidence Associated with PM-10 Above Cutpoint			
		BASE CASE Background = 8 µg/m3	Cutpoint = 20 µg/m3	Cutpoint = 30 µg/m3	Cutpoint = 40 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.1%** **(0.8 - 1.4)**	0.4% (0.3 - 0.6)	0.2% (0.1 - 0.2)	0.1% (0.0 - 0.1)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)	**2.4%** **(1.5 - 3.3)**	1.3% (0.8 - 1.7)	0.7% (0.4 - 0.9)	0.4% (0.2 - 0.5)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**0.8%** **(0.3 - 1.3)**	0.3% (0.1 - 0.4)	0.1% (0.1 - 0.2	0.1% (0.0 - 0.1)
	(D) Congestive Heart Failure (>64 years old)	**1.4%** **(0.7 - 2.1)**	0.5% (0.2 - 0.2)	0.2% (0.1 - 0.1)	0.1% (0.1 - 0.2)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases) (8-12 year olds)	**17.5%** **(15.3 - 19.6)**	9.3% (5.4 - 12.7)	6.3% (3.9 - 8.1)	4.7% (3.4 - 5.5)

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study. In Adjustment Method 1, the cutpoint C-R relationship is modeled to intersect with the original relationship, exceeding the RRs predicted for the original study at higher concentrations. The relationship was modeled to match the reduction in the range of PM concentrations upon application of the cutpoint with an identical percentage increase in the risk observed at the highest concentration. Method 2 estimates a smaller increase in the slope. See text in Section 7 for details.

**Health effects associated with short-term exposure to PM.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from 10 locations.
(B) C-R function based on pooled results from 4 locations.
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz, et al., 1994

Exhibit 7.14

Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With "As-Is" PM-10 Slope Adjustment Method 2* Philadelphia County, September 1992 - August 1993

Health Effects**		Percent of Total Incidence Associated with PM-10 Above Cutpoint			
		BASE CASE Background = 8 µg/m3	Cutpoint = 20 µg/m3	Cutpoint = 30 µg/m3	Cutpoint = 40 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.1%** **(0.8 - 1.4)**	0.4% (0.3 - 0.5)	0.1% (0.1 - 0.2)	0.1% (0.0 - 0.1)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)	**2.4%** **(1.5 - 3.3)**	1.0% (0.6 - 1.3)	0.4% (0.3 - 0.6)	0.2% (0.1 - 0.3)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**0.8%** **(0.3 - 1.3)**	0.3% (0.1 - 0.4)	0.1% (0.0 - 0.2)	0.0% (0.0 - 0.1)
	(D) Congestive Heart Failure (>64 years old)	**1.4%** **(0.7 - 2.1)**	0.5% (0.2 - 0.7)	0.2% (0.1 - 0.3)	0.1% (0.0 - 0.1)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases) (8-12 year olds)	**17.5%** **(15.3 - 19.6)**	7.9% (4.5 - 11.0)	4.1% (2.4 - 5.6)	2.5% (1.5 - 3.2)

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study. In Adjustment Method 2, the slope is increased so that the new C-R function estimates the same health risk at the highest observed PM value as the original function. Method 1 estimates a larger increase in the slope. See text in Section 7 for details.

**Health effects associated with short-term exposure to PM.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from 10 locations.
(B) C-R function based on pooled results from 4 locations.
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz, et al., 1994

Exhibit 7.15

Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With "As Is" PM-2.5 Slope Adjustment Method 1* Philadelphia County, September 1992 - August 1993

Health Effects**		Percent of Total Incidence Associated with PM-2.5 Above Cutpoint		
		BASE CASE: Background = 3.5 µg/m3	Cutpoint = 10 µg/m3	Cutpoint = 18 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.8% (1.1 - 2.5)**	1.1% (0.7 - 1.5)	0.5% (0.3 - 0.6)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**2.0% (0.5 - 3.5)**	1.4% (0.4 - 2.4)	0.8% (0.2 - 1.4)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**0.7% (0.3 - 1.2)**	0.4% (0.1 - 0.6)	0.2% (0.1 - 0.3)
	(D) Congestive Heart Failure (>64 years old)	**1.3% (0.6 - 2.0)**	0.7% (0.3 - 1.0)	0.4% (0.2 - 0.5)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (8 - 12 years old)	**20.0% (10.3 - 28.2)**	13.2% (7.2 - 18.6)	9.9% (5.7 - 13.1)

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) a specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be gre than the slope in the original study. In Adjustment Method 1, the cutpoint C-R relationship is modeled to intersect with the orig relationship, exceeding the RRs predicted for the original study at higher concentrations. The relationship was modeled to mat the range of PM concentrations upon application of the cutpoint with an identical percentage increase in the risk observed at the highest concentration. Method 2 estimates a smaller increase in the slope. See text in Section 7 for details.

** Health effects associated with short-term exposure to PM.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from six locations.
(B) Thurston, et al., 1994
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz et al., 1994

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Section 7 for details.

Exhibit 7.16

Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With "As Is" PM-2.5 Slope Adjustment Method 2* Philadelphia County, September 1992 - August 1993

Health Effects**		Percent of Total Incidence Associated with PM-2.5 Above Cutpoint			
		BASE CASE: Background = 3.5 µg/m3	Cutpoint = 10 µg/m3	Cutpoint = 18 µg/m3	Cutpoint = 30 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.8%** **(1.1 - 2.5)**	1.0% (0.6 - 1.4)	0.4% (0.3 - 0.6)	0.1% (0.1 - 0.2)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**2.0%** **(0.5 - 3.5)**	1.2% (0.3 - 2.2)	0.6% (0.2 - 1.1)	0.2% (0.1 - 0.4)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**0.7%** **(0.3 - 1.2)**	0.4% (0.2 - 0.6)	0.2% (0.1 - 0.3)	0.1% (0.0 - 0.1)
	(D) Congestive Heart Failure (>64 years old)	**1.3%** **(0.6 - 2.0)**	0.7% (0.3 - 1.0)	0.3% (0.2 - 0.5)	0.1% (0.0 - 0.1)
Lower Respiratory Symptoms	(E) Lower Respiratory Symptoms (8 - 12 years old)	**20.0%** **(10.3 - 28.2)**	12.2% (6.5 - 17.3)	7.1% (3.9 - 9.8)	3.8% (2.4 - 4.7)

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study. In Adjustment Method 2, the slope is increased so that the new C-R function estimates the same health risk at the highest observed PM value as the original function. Method 1 estimates a larger increase in the slope. See text in Section 7 for details.

**Health effects associated with short-term exposure to PM.

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All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from six locations.
(B) Thurston, et al., 1994
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz et al., 1994

Exhibit 7.17

Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With PM-10 After Attainment of Current Standards* Slope Adjustment Method 1** Southeast Los Angeles County, 1995

Health Effects***		Percent of Total Incidence Associated with PM-10 Above Cutpoint			
		BASE CASE Background = 8 µg/m3	Cutpoint = 20 µg/m3	Cutpoint = 30 µg/m3	Cutpoint = 40 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**2.6%** **(1.8 - 3.3)**	1.8% (1.3 - 2.3)	1.2% (0.9 - 1.6)	0.8% (0.6 - 1.0)
	(A') Associated with short-term exposure (Study done in Los Angeles)	**1.2%** **(0.1 - 2.2)**	1.1% (0.1 - 2.1)	0.8% (0.1 - 1.5)	0.6% (0.1 - 1.1)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)	**5.4%** **(3.3 - 7.4)**	4.6% (2.8 - 6.3)	4.1% (2.6 - 5.6)	3.7% (2.3 - 4.9)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**1.8%** **(0.7 - 2.9)**	1.0% (0.4 - 1.7)	0.9% (0.4 - 1.5)	0.9% (0.3 - 1.4)
	(D) Congestive Heart Failure (>64 years old)	**3.2%** **(1.5 - 4.8)**	1.8% (0.9 - 0.9)	1.7% (0.8 - 0.8)	1.5% (0.7 - 2.2)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases (8-12 year olds)	**34.8%** **(31.0 - 38.4)**	26.4% (16.1 - 34.6)	27.7% (18.4 - 34.1)	27.2% (21.4 - 30.3)

* Current standards are 50 ug/m3 annual average PM-10, 150 ug/m3 second daily maximum PM-1

** Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study. In Adjustment Method 1, the cutpoint C-R relationship is modeled to intersect with the original relationship, exceeding the RRs predicted for the original study at higher concentrations. The relationship was modeled to match the reduction in the range of PM concentrations upon application of the cutpoint with an identical percentage increase in the risk observed at the highest concentration. Method 2 estimates a smaller increase in the slope. See text for further information.

*** Health effects associated with short-term exposure to PM.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from 10 locations.
(A') Kinney et al., 1995
(B) C-R function based on pooled results from 4 locations.
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz, et al., 1994

Exhibit 7.18

Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With PM-10 After Attainment of Current Standards* Slope Adjustment Method 2** Southeast Los Angeles County, 1995

Health Effects***		Percent of Total Incidence Associated with PM-10 Above Cutpoint			
		BASE CASE Background = 8 µg/m3	Cutpoint = 20 µg/m3	Cutpoint = 30 µg/m3	Cutpoint = 40 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**2.6%** **(1.8 - 3.3)**	1.6% (1.2 - 2.1)	1.1% (0.8 - 1.4)	0.7% (0.5 - 0.9)
	(A') Associated with short-term exposure (Study done in Los Angeles)	**1.2%** **(0.1 - 2.2)**	1.0% (0.1 - 2.0)	0.7% (0.1 - 1.3)	0.5% (0.0 - 0.9)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)	**5.4%** **(3.3 - 7.4)**	3.5% (2.2 - 4.8)	2.7% (1.7 - 3.7)	2.0% (1.3 - 2.7)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**1.8%** **(0.7 - 2.9)**	1.1% (0.4 - 1.8)	0.8% (0.3 - 1.3)	0.6% (0.2 - 1.0)
	(D) Congestive Heart Failure (>64 years old)	**3.2%** **(1.5 - 4.8)**	1.9% (0.9 - 2.8)	1.4% (0.7 - 2.1)	1.1% (0.5 - 1.6)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases (8-12 year olds)	**34.8%** **(31.0 - 38.4)**	22.9% (13.7 - 30.5)	19.4% (12.0 - 25.2)	16.7% (11.3 - 20.5)

* Current standards are 50 ug/m3 annual average PM-10, 150 ug/m3 second daily maximum PM-1

** Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study. In Adjustment Method 2, the slope is increased so that the new C-R function estimates the same health risk at the highest observed PM value as the original function. Method 1 estimates a larger increase in the slope.

***Health effects associated with short-term exposure to PM.

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Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from 10 locations.
(A') Kinney et al., 1995
(B) C-R function based on pooled results from 4 locations.
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz, et al., 1994

Exhibit 7.19

Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With PM-2.5 After Attainment of Current Standards* Slope Adjustment Method 1** Southeast Los Angeles County, 1995

Health Effects***		Percent of Total Incidence Associated with PM-2.5 Above Cutpoint			
		BASE CASE: **Backgrount = 3.5 µg/m3**	Cutpoint = 10 µg/m3	Cutpoint = 18 µg/m3	Cutpoint = 30 µg/m3
Mortality (all ages)	(A) Associated with with short-term exposure	**2.9%** **(1.7 - 3.9)**	2.1% (1.3 - 2.8)	1.3% (0.8 - 1.8)	0.7% (0.4 - 1.0)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**6.1%** **(1.6 - 10.5)**	4.9% (1.3 - 8.5)	4.0% (1.1 - 7.0)	3.4% (0.9 - 5.9)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**1.1%** **(0.4 - 1.8)**	0.7% (0.3 - 1.1)	0.6% (0.2 - 0.9)	0.4% (0.2 - 0.7)
	(D) Congestive Heart Failure (>64 years old)	**2.0%** **(1.0 - 3.0)**	1.2% (0.6 - 1.8)	1.0% (0.5 - 1.5)	0.7% (0.4 - 1.1)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (8 - 12 years old)	**28.7%** **(15.4 - 39.0)**	22.0% (12.5 - 29.7)	21.0% (13.2 - 26.5)	19.7% (17.0 - 20.9)

* Current standards are 50 ug/m3 annual average PM-10, 150 ug/m3 second daily maximum PM-10

** Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be gr than the slope in the original study. In Adjustment Method 1, the cutpoint C-R relationship is modeled to intersect with the ori relationship, exceeding the RRs predicted for the original study at higher concentrations. The relationship was modeled to match the reduction in the range of PM concentrations upon application of the cutpoint with an identical percentage increase in the risk observed at the highest concentration. Method 2 estimates a smaller increase in the slope. See text for further information.

*** Health effects associated with short-term exposure to PM.

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All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.

See text in Section 7 for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from six locations
(B) Thurston, et al., 1994
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz et al., 1994

Exhibit 7.20

Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With PM-2.5 After Attainment of Current Standards* Slope Adjustment Method 2** Southeast Los Angeles County, 1995

Health Effects***		Percent of Total Incidence Associated with PM-2.5 Above Cutpoint			
		BASE CASE:	Cutpoint	Cutpoint	Cutpoint
		Background = 3.5 µg/m3	= 10 µg/m3	= 18 µg/m3	= 30 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**2.9%** **(1.7 - 3.9)**	1.9% (1.2 - 2.7)	1.2% (0.7 - 1.7)	0.6% (0.4 - 0.8)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**6.1%** **(1.6 - 10.5)**	4.4% (1.2 - 7.6)	3.1% (0.8 - 5.4)	2.0% (0.5 - 3.4)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**1.1%** **(0.4 - 1.8)**	0.7% (0.3 - 1.2)	0.5% (0.2 - 0.8)	0.3% (0.1 - 0.5)
	(D) Congestive Heart Failure (>64 years old)	**2.0%** **(1.0 - 3.0)**	1.3% (0.6 - 1.9)	0.9% (0.4 - 1.3)	0.5% (0.2 - 0.8)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (8 - 12 years old)	**28.7%** **(15.4 - 39.0)**	20.4% (11.5 - 27.9)	15.8% (9.3 - 20.9)	13.1% (9.1 - 15.5)

* Current standards are 50 ug/m3 annual average PM-10, 150 ug/m3 second daily maximum PM-

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficier above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is s be greater than the slope in the original study. In Adjustment Method 2, the slope is increased so that the new C-R function estimates the same health risk at the highest observed PM value as the original function. Method 1 estimates a larger increase in the slope.

**Health effects associated with short-term exposure to PM.

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter 7 for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from six locations.
(B) Thurston, et al., 1994
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz et al., 1994

Exhibit 7.21

Sensitivity Analysis: Effect of Combining Different Averaging Times In Pooled Short-Term Exposure Mortality Functions on Predicted Health Effects Associated With "As-Is" PM-10 Philadelphia County, September 1992 - August 1993

	Percent of Total Incidence Associated with PM-10 Above Background*			
	BASE CASE Studies Using All Averaging Times (10 studies)**	Studies using 1-day average PM (1 study)	Studies using 2-day average PM (7 studies)	Studies using 3-5 day average PM (2 studies)
Matching study and data averaging times	**2-day average PM**	1-day average PM	2-day average PM	5-day average PM
	1.1% **(0.8 - 1.4)**	0.4% (0.0 - 0.8)	1.0% (0.5 - 1.5)	1.8% (1.3 - 2.4)
Using 2-day average PM data	2-day average PM	2-day average PM	2-day average PM	2-day average PM
	same	0.4% (0.0 - 0.8)	same	1.9% (1.3 - 2.4)
Using 1-day average PM data	1-day average PM			
	1.1% (0.8 - 1.4)			
Using 5-day average PM data	5-day average PM			
	1.1% (0.8 - 1.4)			

*Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Background PM-10 is assumed to be 8 ug/m3 .

** The base case is a random-effects pooled function used with 2-day average PM data. All other pooled functions are also random effects, except the pooled function derived from studies using 3-5 day average PM data, for which a fixed effects model was used, since it is not possible to calculate a random effects model for those two functions.

The numbers in parentheses for pooled functions are NOT standard confidence interva
All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

The studies that contribute to the pooled function are:

1-day: Kinney et al., 1995 (Los Angeles)
2-day: Ito and Thurston, 1996 (Chicago)
Schwartz et al. 1996 (Boston, MA; Knoxville, TN; St. Louis, MO; Steubenville, OH; Portage, WI; Topeka, KS)
3-day: Schwartz 1993 (Birmingham, AL)
5-day: Pope et al., 1992 (Utah Valley)

Exhibit 7.22

Sensitivity Analysis: The Effect of Considering Different Epidemiology Studies Relating Mortality and Short-Term Exposure to PM on Estimated Annual Mortality Risks Associated with "As Is" PM Concentrations in Philadelphia County, September 1992- August 1993 (for base case assumptions)

Study	Location	Associated with PM-10		Associated with PM-2.5	
		Incidence	Percent of Total Incidence	Incidence	Percent of Total Incidence
Pooled function		**220**	**1.1%**	**NA**	**NA**
		(160 - 290)	**(0.8 - 1.4)**	**NA**	**NA**
Ito & Thurston 1996	Chicago	170	0.8%	--	--
		(70 - 270)	(0.3 - 1.3)		
Kinney et al. 1995	Los Angeles	80	0.4%	--	--
		(10 - 160)	(0.0 - 0.8)		
Pope et al. 1992	Utah Valley	420	2.1%	--	--
		(270 - 560)	(1.4 - 2.8)		
Schwartz 1993	Birmingham, AL	300	1.5%	--	--
		(100 - 490)	(0.5 - 2.4)		
Schwartz et al., 1996a	Boston	360	1.8%	NA	NA
		(230 - 480)	(1.1 - 2.4)	NA	NA
	Knoxville, TN	280	1.4%	NA	NA
		(490 - 490)	(2.4 - 2.4)	NA	NA
	St. Louis	160	0.8%	NA	NA
		(70 - 290)	(0.3 - 1.4)	NA	NA
	Steubenville, OH	230	1.1%	NA	NA
		(30 - 430)	(0.2 - 2.1)	NA	NA
	Madison, WI	140	0.7%	NA	NA
		(-130 - 410)	(-0.6 - 2.0)	NA	NA
	Topeka, KS	-150	-0.7%	NA	NA
		(-560 - 250)	(-2.7 - 1.2)	NA	NA

Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Background PM-10 is assumed to be 8 ug/m3; background PM-2.5 is assumed to be 3.5 ug/m3.

The presence of negative numbers (for Madison, WI and Topeka, KS) is due to statistical uncertainty in the estimation of relative risks, and does not reflect a belief that increased particulate matter pollution may actually be beneficial to health.

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Section 7 for details.

Table 7.23

Sensitivity Analysis: Effect of Copollutants
Relative Risks for Change of 50 ug/m3 PM-10 or 25 ug/m3 PM-2.5

Endpoint		Study, Pollutant, & Location	Relative risk no copollutant	Relative Risk with daily average SO2	Relative Risk with daily 1-hour maximum CO	Relative Risk with daily average O3	Relative Risk with daily 1-hour maximum O3
Mortality		Ito & Thurston 1995, PM-10 Chicago	**1.02** **(1.02 - 1.04)**			1.02 (1.01 - 1.03)	
		Kinney et al., 1995, PM-10 Los Angeles	**1.02** **(1.00 - 1.05)**		1.02 (0.99 - 1.04)		1.02 (1.00 - 1.05)
		Pope 1994, PM-10 Utah Valley, summer only	1.11 (0.95 - 1.31)			1.14 (0.96 - 1.37)	1.19 (1.00 - 1.43)
Hospital Admissions	All respiratory (all ages)	Thurston et al., 1994, PM-2.5 Ontario, Canada	**0.086*** **(0.024 - 0.15)**				0.045* (-0.028 - 0.12)
	All respiratory (ages >64)	Schwartz 1995, PM-10 New Haven	**1.06** **(1.01 - 1.12)**	1.07 (1.02 - 1.13)		1.09 (1.01 - 1.18)	
		Schwartz 1995, PM-10 Tacoma	**1.10** **(1.04 - 1.16)**	1.11 (1.03 - 1.19)		1.12 (0.99 - 1.26)	
	Pneumonia (ages >64)	Schwartz 1994, PM-10 Minneapolis/St. Paul				**1.08** **(1.02 - 1.14)**	
		Schwartz 1994, PM-10 Detroit				**1.06** **(1.03 - 1.09)**	
	COPD (ages >64)	Schwartz 1994, PM-10 Detroit				**1.10** **(1.06 - 1.16)**	
	Ischemic Heart Disease	Schwartz & Morris 1995, PM-10 Detroit	**1.028** **(1.011 - 1.047)**	1.024** (1.005 - 1.043)	1.025 (1.007 - 1.044)		
	Congestive Heart Failure	Schwartz & Morris 1995, PM-10 Detroit	**1.050** **(1.024 - 1.077)**		1.038 (1.011 - 1.064)		

Results presented in bold come from functions used in the base case analysis.
The number of significant digits given for each relative risk is the same as the number reported in the original study.

* Thurston et al. 1994 provides a function relating changes in PM to changes in the number of cases.
The relative risk calculated from this coefficient may vary widely from location to location, depending on baseline incidences.
Therefore, the coefficient, adjusted to a rate per 100,000 people, is reported, instead of a relative risk.

** Based on 1-hour maximum SO2.

Exhibit 7.24

Sensitivity Analysis: Effect of Copollutants c
Predicted Health Effects Associated With "As-Is" PM*
Philadelphia County, September 1992 - August 1993

			Percent of total incidence associated with PM above backgr				
Health Effects		Study & Location	with no copollutant	with daily average SO2	with daily 1-hour maximum CO	with daily average O3	with daily 1-hour maximum O3
Mortality		Ito & Thurston 1996, PM-10 Chicago	**0.8% (0.3 - 1.3)**			0.6% (0.2 - 0.9)	
		Kinney et al., 1995, PM-10 Los Angeles	**0.4% (0.0 - 0.8)**		0.3% (-0.0 - 0.7)		0.4% (0.0 - 0.8)
		Pope 1994, PM-10 Utah Valley, summer only	3.0% (-1.5 - 7.2)			3.7% (-1.3 - 8.3)	4.8% (-0.2 - 9.4)
Hospital Admissions	All respiratory (ages >64)	Thurston et al., 1994, PM-2.5 Ontario, Canada	**2.0% (0.5 - 3.5)**				1.1% (-0.7 - 2.8)
	All respiratory (ages >64)	Schwartz 1995, PM-10 New Haven	**2.4% (0.3 - 4.5)**	1.9% (0.6 - 3.4)		2.4% (0.4 - 4.6)	
		Schwartz 1995, PM-10 Tacoma	**3.2% (-0.2 - 6.4)**	2.9% (1.0 - 4.7)		3.2% (-0.2 - 6.4)	
	Pneumonia (ages >64)	Schwartz 1994, PM-10 Minneapolis/St. Paul				**2.2% (0.6 - 3.8)**	
		Schwartz 1994, PM-10 Detroit				**1.6% (0.7 - 2.5)**	
	COPD (ages >64)	Schwartz 1994, PM-10 Detroit				**2.8% (1.5 - 4.2)**	
	Ischemic Heart Disease	Schwartz & Morris 1995, PM- Detroit	**0.8% (0.3 - 1.3)**	0.7%** (0.1 - 1.2)	0.7% (0.2 - 1.2)		
	Congestive Heart Failur	Schwartz & Morris 1995, PM- Detroit	**1.4% (0.7 - 2.1)**		1.1% (0.3 - 1.8)		

Results presented in bold come from functions used in the base case analysis.

* Health effects associated with short-term exposure to PM. Incidence was quantified across the range of PM concentrations observed in each study, but not below background PM levels, assumed to be 8 ug/m3 for PM-10 and 3.5 ug/m3 for PM-

** Based on 1-hour maximum SO2.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possibl variability. See text for details.

Exhibit 7.25

Sensitivity Analysis: Effect of Copollutants on Predicted Health Effects Associated With PM* After Attainment of Current Standards Southeast Los Angeles County, 1995**

			Percent of total incidence associated with PM above background				
Health Effects		Study & Location	with no copollutant	with daily average SO2	with daily 1-hour maximum CO	with daily average O3	with daily 1-hour maximum O3
Mortality		Ito & Thurston 1996, PM-10 Chicago	**1.9% (0.7 - 3.1)**			1.3% (0.5 - 2.1)	
		Kinney et al., 1995, PM-10 Los Angeles	**1.2% (0.1 - 2.2)**		1.0% (-0.1 - 2.0)		1.2% (0.1 - 2.2)
		Pope 1994, PM-10 Utah Valley, summer only	6.7% (-3.6 - 15.7)			8.3% (-3.0 - 17.9)	10.7% (-0.4 - 20.1)
Hospital Admissions	All respiratory (all ages)	Thurston et al., 1994, PM-2.5 Ontario, Canada	**NA NA**				NA NA
	All respiratory (ages >64)	Schwartz 1995, PM-10 New Haven	**5.5% (0.6 - 10.1)**	4.4% (1.3 - 7.6)		5.5% (1.0 - 10.3)	
		Schwartz 1995, PM-10 Tacoma	**7.2% (-0.4 - 14.0)**	6.6% (2.2 - 10.5)		7.2% (-0.4 - 14.0)	
	Pneumonia (ages >64)	Schwartz 1994, PM-10 Minneapolis/St. Paul				**5.1% (1.5 - 8.4)**	
		Schwartz 1994, PM-10 Detroit				**3.7% (1.7 - 5.7)**	
	COPD (ages >64)	Schwartz 1994, PM-10 Detroit				**6.4% (3.4 - 9.3)**	
	Ischemic Heart Disease	Schwartz & Morris 1995, PM-10 Detroit	**1.8% (0.7 - 2.9)**	1.5%*** (0.3 - 2.8)	1.6% (0.4 - 2.8)		
	Congestive Heart Failure	Schwartz & Morris 1995, PM-10 Detroit	**3.2% (1.5 - 4.8)**		2.4% (0.7 - 4.0)		

Results presented in bold come from functions used in the base case analysis

* Health effects associated with short-term exposure to PM. Incidence was quantified across the range of PM concentrations observed in each study, but not below background PMlevels, assumed to be 6 ug/m3 for PM-10 and 2.5 ug/m3 for PM-2.5.

** Current standards are 50 ug/m3 annual average PM-10, 150 ug/m3 second daily maximum PM-10.

*** Based on 1-hour maximum SO2.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text for details.

Exhibit 7.26

Sensitivity Analysis: The Effect of Differing Cutpoints on Estimated Mortality Associated with Long-term Exposure to PM-2.5 Philadelphia County, September 1992 - August 1993

	BASE CASE Lowest Observed = 9 ug/m3	Cutpoint = 12.5 ug/m3	Cutpoint = 15 ug/m3	Cutpoint = 18 ug/m3
(A) Mortality associated with long-term exposure	**4.7% (2.9 - 6.4)**	2.5% (1.6 - 3.5)	1.0% (0.6 - 1.3)	0.0% (0.0 - 0.0)

(A) Pope et al., 1995

Health effects incidence was calculated down to the lowest level observed in the study (9 ug/m3).
No adjustments to the slope were performed.

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter 7 for details.

Exhibit 7.27

Sensitivity Analysis: The Effect of Concentration-Response Function Slope on Estimated Mortality Associated with Long-term Exposure to PM-2.5 Philadelphia County, September 1992 - August 1993

	BASE CASE **Assuming AQ as reported**	Assuming relevant AQ 50% higher*	Assuming relevant AQ twice as high*
(A) Mortality associated with long-term exposure	**4.7%** **(2.9 - 6.4)**	3.5% (2.2 - 4.9)	2.4% (1.5 - 3.3)

Health effects incidence was calculated down to the lowest level observed in the study (A) Pope et al., 1995

* Adjusted function from Pope et al., 1995. Had historical air quality been 50% higher, the relative risk calculated by the study would have been two thirds of that reported. Had historical air quality been twice as high, the relative risk calculated would have been half that reported. See text for details.

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter 7 for details.

7.2. Uncertainty Analyses

A set of risk analyses considering different sample locations, different PM indicators, different PM standards, different health endpoints, and different concentration-response functions for each health endpoint can be expected to produce a set of results with a large degree of variability. Some of this variability reflects actual variability in the underlying population parameters. For example, "as is" PM concentrations in one location may be much greater than in another location. Some of the variability in the outcomes of the risk analyses, however, reflects uncertainty about the true values of the parameters of the risk analyses. The substantial variability generated by applying different concentration-response functions to a given sample location, for example, does not reflect real variability that exists in that location, but instead reflects uncertainty about the actual concentration-response relationship between PM and the population response in that location.

There are several sources of possible uncertainty associated with the estimation of PM-related incidence in the risk analysis (as discussed in Section 3). These include, for example, uncertainty about the appropriate concentration-response function for a given location, uncertainty about the baseline health effect incidence rates in the location, uncertainty about average daily PM concentrations in the location, and uncertainty about the background PM concentration in the location. In addition, there are sources of uncertainty inherent in any empirical investigation, such as the uncertainty about the correct functional form of the (concentration-response) model.

Some of these analysis input components are likely to involve much greater degrees of uncertainty than others. The concentration-response function, for example, is considered to be a source of substantial uncertainty. In contrast, because it was possible to obtain local baseline incidence rates for many of the health endpoints, the degree of uncertainty associated with these incidence rates is considered to be quite small. Similarly, although there may be some uncertainty associated with average daily PM concentrations (especially related to those days for which no PM monitoring was done), the overall level of uncertainty associated with PM concentrations is judged to be small relative to that associated with the concentration-response function. First, the percent of days without monitoring was very small in Philadelphia County and fairly small in Southeast Los Angeles County. Second, if the uncertainty is associated largely with random measurement errors, then small errors in one direction on one day are likely to be counterbalanced by small errors in the other direction on another day. Because daily incidences are totaled over the year, minor daily discrepancies related to PM measurement errors are likely to largely cancel each other out in the total.

In addition to differences in the degrees of uncertainty associated with different components of the analysis, there are differences in the degree to which these uncertainties can be quantified. A sensitivity analysis (like those presented in Section 7.1) considers how the end result of an analysis varies as a particular input parameter value is varied. Such an analysis requires only the possible alternative parameter values but does not require that the probabilities of each possible input parameter value be known. In contrast, a quantitative

assessment of the uncertainty associated with an input parameter requires either the distribution of possible parameter values, or, at a minimum, some information on which to base an estimate of this distribution.

The source of the largest amount of uncertainty in the risk analysis is likely to be the concentration-response function. In addition, although the amount of information about the distribution of possible values of the parameter in the concentration-response function varies from one function to another (e.g., for short-term exposure mortality there is substantial information, whereas for hospital admissions for respiratory illness there is much less), there is some information for each category of concentration-response function. For these reasons, the concentration-response function is the primary focus of the uncertainty analysis. Uncertainty bounds characterizing the uncertainty associated with the concentration-response function alone were derived for each combination of health endpoint and PM indicator. The methods used to derive these uncertainty bounds are discussed in Section 5.2. The emphasis in this discussion is on the general case when there is more than one study reporting a concentration-response function for a particular combination of health endpoint and PM indicator (e.g., short-term exposure mortality and PM-10). The results of these uncertainty analyses are shown in Section 5.2.4.

Other sources of uncertainty were sequentially added to the uncertainty associated with the concentration-response function, using Monte Carlo propagation of uncertainty methods, which allow multiple sources of uncertainty to be considered simultaneously. The method and results of this propagation of uncertainty are presented below.

7.2.1. A Monte Carlo analysis: propagation of uncertainties from several sources

The sensitivity analyses presented in Section 7.1 (and for meeting alternative standards, in Section 8 below) illustrate the dependence of the results of the risk analyses on certain key parameters and assumptions. The uncertainty analysis presented in Sections 5.3.1 and 5.3.2 attempts to quantify the uncertainty associated with a single parameter of the risk analysis, namely, the concentration-response function. While sensitivity analyses may help to identify those parameters that most influence the results of an analysis, such analyses do not indicate the likelihood that the true value of the parameter is any of those values considered. In addition, neither the sensitivity analyses nor the uncertainty analysis presented above consider more than one source of uncertainty at a time. This section presents a set of analyses intended to more fully characterize the uncertainty surrounding the risk estimates presented in Sections 7.1 and 8.1. The analyses use a Monte Carlo procedure to include uncertainty from several sources simultaneously. These integrated uncertainty analyses are limited to consideration of mortality associated with short-term and long-term exposure to PM-2.5.

As noted above, a Monte Carlo procedure refers to the drawing of observations from a known distribution. Uncertainty from several sources may be propagated through the risk model by simultaneously drawing observations from a set of distributions, one for each source

of uncertainty considered. Suppose, for example, there are three unknown parameters in the analysis model, each of which has been estimated. Each parameter estimate is the mean of a distribution of possible values. On each iteration of the Monte Carlo procedure, an observation is randomly selected from each of the three distributions. Each iteration therefore produces a triple -- three values, one for each unknown parameter. Given these three particular parameter values, there is a particular value of the endpoint of the analysis (e.g., a particular value of avoided mortality). On each of many iterations, the Monte Carlo procedure produces a randomly selected set of parameter values (selected from the distributions for these parameters) which in turn produces a particular value of the endpoint of the analysis. The procedure therefore produces a *distribution* of values of the analysis endpoint (e.g., a distribution of avoided mortality) corresponding to the set of distributions of parameter values. This distribution characterizes the uncertainty surrounding the analysis endpoint resulting from the uncertainty surrounding the input parameters considered.

Monte Carlo propagation of uncertainty analyses were carried out for both the short-term exposure mortality and the long-term exposure mortality risk analyses. The sources of uncertainty included in the Monte Carlo analysis are listed in Exhibit 7.28, along with theircorresponding distributions. Uncertainties were incorporated into the analysis one at a time in order to demonstrate the effect of each one.

The distributions of parameter values are not known but instead were estimated from the available information The estimation of the distribution of β in the concentration-response function, for example, is described in Section 5.2. The distributions for background PM-2.5 were based on estimates of background PM-2.5 concentrations ranging from 2 $\mu g/m^3$ to 5 $\mu g/m^3$ in the Eastern U.S. and from 1 $\mu g/m^3$ to 4 $\mu g/m^3$ in the Western U.S. With no further information about the distribution of background concentrations in the Eastern or Western U.S., uniform distributions on these intervals seemed reasonable.

Not all uncertainties associated with the model are incorporated into the propagated uncertainty analysis. Some uncertainties, including, for example, the possible influence of copollutants, were excluded from this analysis due to a lack of sufficient quantitative information from which to estimate a distribution. Despite a lack of quantitative information, cutpoints were included because of their key role in the analysis. Because there is no information on which to base an estimate of the distribution of values of a possible cutpoint, three alternative distributions, each consisting of weights for four discrete cutpoint values, were used to illustrate the impact on the risk estimates of alternative viewpoints about the likelihood of a threshold existing above background PM-2.5 levels. The weighting schemes, which are included for illustrative purposes, are shown in Exhibit 7.29. Case I represents a judgement that concentration-response functions are likely to be valid down to either background or 10 $\mu g/m^3$; case III represents a judgement that concentration-response functions are likely to have cutpoints at or above 18 $\mu g/m^3$; and case II is intermediate between the other two.

Exhibit 7.28. Summary of Uncertainties Incorporated into Integrated Uncertainty Analysis

Uncertainty	Distribution
Coefficient (β) in concentration-response function	Short-term exposure function (for which there were several epidemiological studies): 200 points representing the estimated distribution of β for short-term exposure mortality, derived in Section 5 Long-term exposure function: normal distribution based on results of the single study (Pope et al., 1995) used in the risk analysis
Cutpoints in concentration-response function	4 cutpoints, 3 discrete weighting schemes, 2 slope adjustment methods
Background PM-2.5 concentration	uniform distributions on the intervals [2,5] and [1,4] ($\mu g/m^3$) for Philadelphia County and Los Angeles County, respectively (these are the ranges identified in the Criteria Document)
Form of PM reductions to achieve alternative standards	130 points representing distribution of a certain kind of non-proportionality (described in Section 8)

Exhibit 7.29. Three weighting schemes for cutpoints in integrated uncertainty analyses: Mortality associated with short-term exposure to PM-2.5

	Case I	Case II	Case III
Background	0.50	0.20	0.05
10 $\mu g/m^3$	0.30	0.30	0.15
18 $\mu g/m^3$	0.15	0.30	0.50
30 $\mu g/m^3$	0.05	0.20	0.30

Ideally, to derive a concentration-response function with a cutpoint, the data on which the original concentration-response function (without a cutpoint) was based would be re-analyzed, excluding those points falling below the selected PM level. If a threshold existed in the original data, this would presumably result in a different, steeper estimated function. Since the data on which the concentration-response functions were based are not readily available, two methods of adjusting the slope of concentration-response functions (described in Section 7.1) were considered. In the integrated uncertainty analyses, they are given equal weight.

Exhibits 7.30 and 7.31 show the integrated uncertainty analysis for mortality due to short-term exposure for "as is" conditions in Philadelphia County, and results assuming attainment of current PM-10 standards in Southeast Los Angeles County, respectively. Each

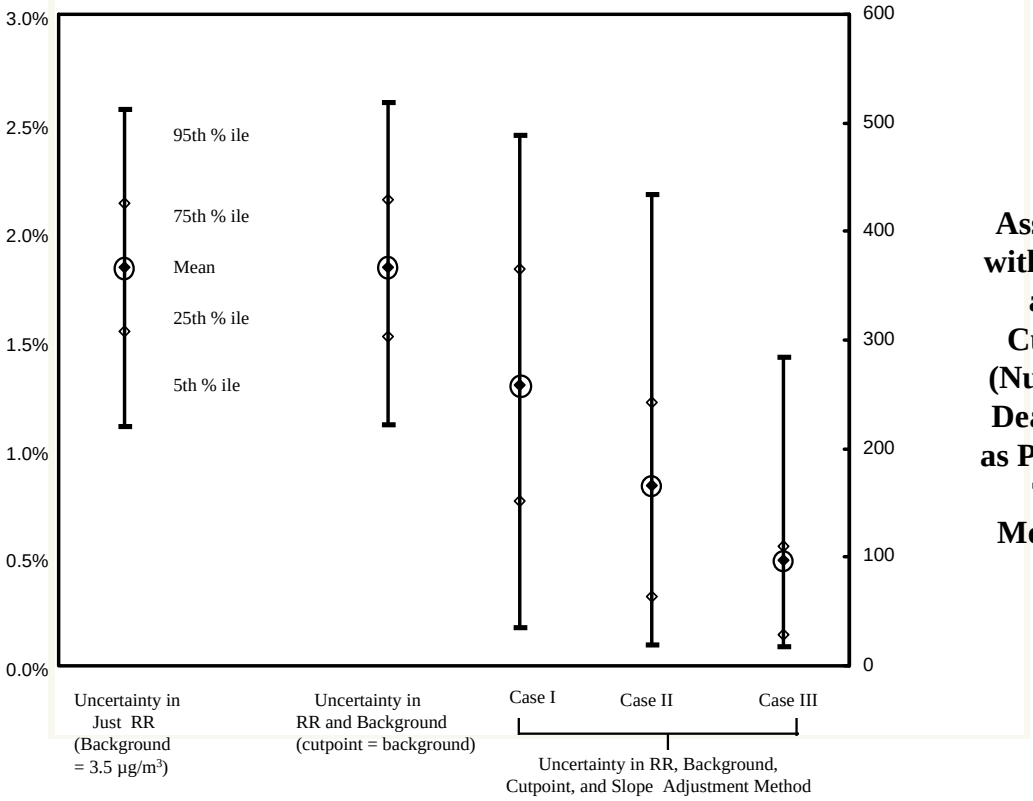
vertical bar represents an estimate of the health effects that includes a certain set of uncertainties (identified below the bars). The mean estimate is shown, as well as the 5th, 25th, 75th, and 95th percentiles. Note that in weighting case III, the 25th percentile becomes very close to the 5th percentile, and the 75th percentile becomes very close to the mean. This is a result of the discrete nature of the distribution of cutpoints (as well as of the specific weights assigned to each cutpoint). The lowest thirty percent of values in the distribution of estimates for Case III come from iterations when the cutpoint is assumed equal to 30 $\mu g/m^3$. Iterations when the cutpoint is assumed to be lower give substantially higher values; therefore the 25th percentile is very close to the 5th percentile (compared to the range of values). A jump would be expected to occur around the 30th percentile. Similarly, since the next fifty percent of values come from iterations when the cutpoint is assumed equal to 18 $\mu g/m^3$, and since these values are substantially lower than those predicted when the cutpoint is assumed to be lower, the 75th percentile value is close to the mean (again, compared to the range of values). If all the individual values obtained were plotted, the graph would show clusters of points.

Because the lower cutpoints yield larger ranges of values (as well as larger values in general; see Exhibits 7.15 and 7.16), Case I , which gives greater weight to the lower cutpoints, does not show this kind of bunching of the distribution. Case II is intermediate between Cases I and III. Similarly, because of Los Angeles County's generally higher PM concentrations, this phenomenon is not as pronounced in Exhibit 7.31.

Exhibit 7.30

Uncertainty Analysis: Effect of Uncertainty of Relative Risks, Background Concentrations, Cutpoint and Slope Adjustment Method

Mortality Associated With Short-Term Exposure to PM-2.5
Philadelphia, September 1992 - August 1993
(Population: 1.6 Million)



Uncertainty in background concentration enters into these calculations only when the cutpoint is set equal to background. The other cutpoints are greater than the highest background concentration considered. When a cutpoint other than background is chosen, each of the two slope adjustment methods has a fifty percent chance of selection.

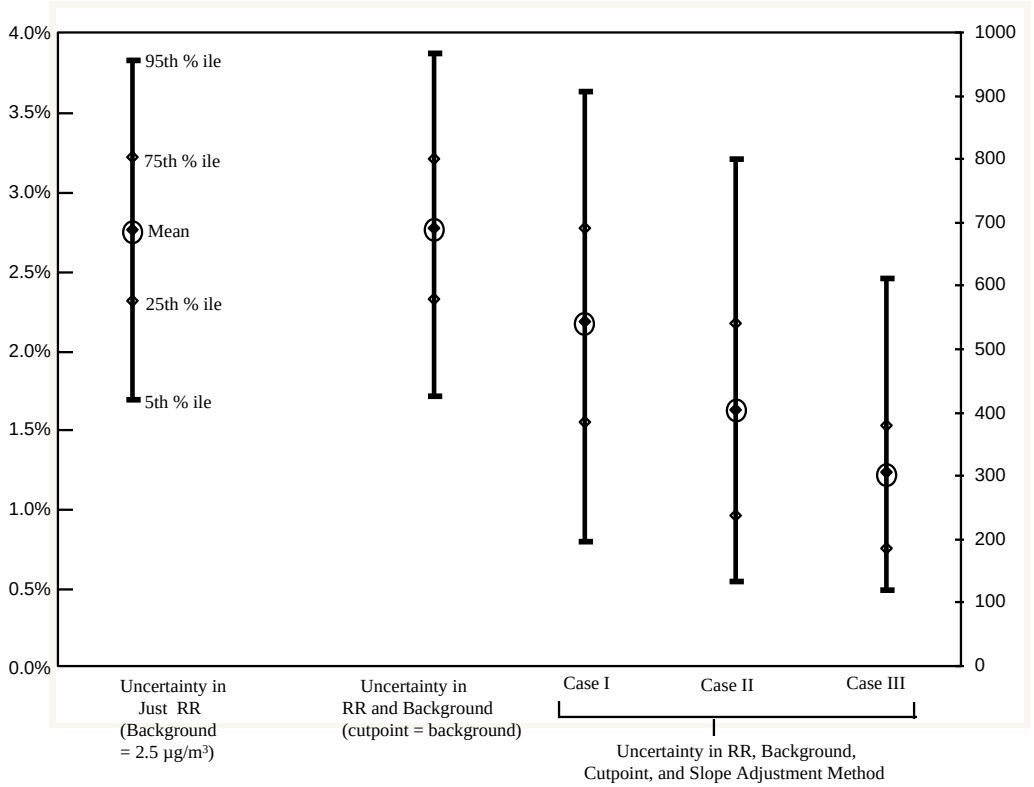
Cutpoint Weighting Schemes

	Case I	Case II	Case III
Background	0.5	0.2	0.05
10 $\mu g/m^3$	0.3	0.3	0.15
18 $\mu g/m^3$	0.15	0.3	0.5
30 $\mu g/m^3$	0.05	0.2	0.3

Exhibit 7.31

Uncertainty Analysis: Effect of Uncertainty of Relative Risks, Background Concentrations, Cutpoint and Slope Adjustment Method

Mortality Associated With Short-Term Exposure to PM-2.5
After Meeting Current PM-10 Standards
Southeast Los Angeles County, 1995
(Population: 3.6 Million)



Uncertainty in background concentration enters into these calculations only when the cutpoint is set equal to background. The other cutpoints are greater than the highest background concentration considered. When a cutpoint other than background is chosen, each of the slope adjustment methods has a fifty percent chance of selection.

Cutpoint Weighting Schemes

	Case I	Case II	Case III
Background	0.5	0.2	0.05
10 $\mu g/m^3$	0.3	0.3	0.15
18 $\mu g/m^3$	0.15	0.3	0.5
30 $\mu g/m^3$	0.05	0.2	0.3

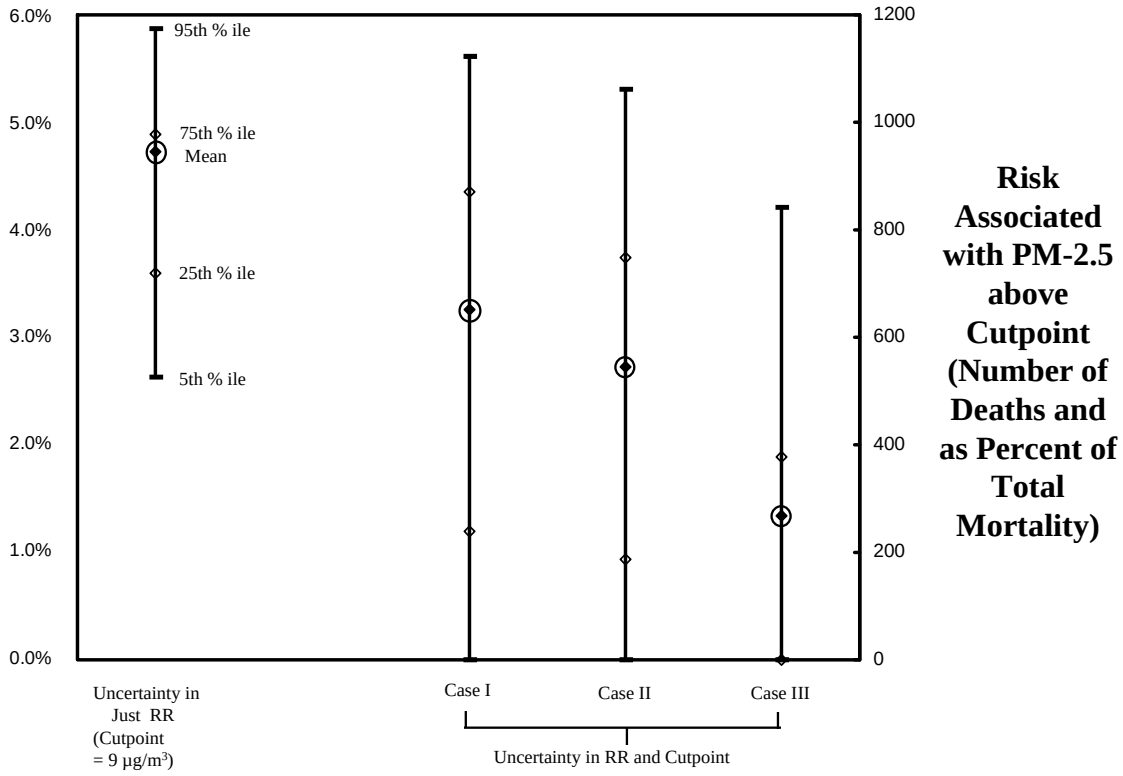
A similar uncertainty analysis was carried out for mortality associated with long-term exposure. Exhibit 7.32 shows the cutpoint weighting schemes for this analysis. Like the weighting schemes for short-term exposure cutpoints (see Exhibit 7.29), these are used for illustrative purposes. Exhibit 7.33 shows the results of the uncertainty analysis for mortality associated with long-term exposure in Philadelphia County.

Exhibit 7.32. Three weighting schemes for cutpoints in integrated uncertainty analyses: Mortality associated with long-term exposure to PM-2.5

	Case I	Case II	Case III
9 $\mu g/m^3$	0.55	0.35	0.10
12.5 $\mu g/m^3$	0.20	0.35	0.20
15 $\mu g/m^3$	0.15	0.20	0.40
18 $\mu g/m^3$	0.10	0.10	0.30

Exhibit 7.33
Uncertainty Analysis: Effect of Uncertainty of Relative Risk and Cutpoint

Mortality Associated With Long-Term Exposure to PM-2.5
Philadelphia, September 1992 - August 1993
(Population: 1.6 Million)



The lowest observed level in the long-term exposure mortality study (Pope et al., 1995) is 9 µg/m³. Because this is above background PM-2.5 in Philadelphia, background does not enter into these calculations.

Cutpoint Weighting Schemes

	Case I	Case II	Case III
9 µg/m³	0.55	0.35	0.10
12.5 µg/m³	0.20	0.35	0.20
15 µg/m³	0.15	0.20	0.40
18 µg/m³	0.10	0.10	0.30

8. Assessment of the Health Risk Reductions Associated with Attainment of Alternative PM Standards

8.1. Results and sensitivity analyses

The results of the second phase of the risk analysis, assessing the health risk reductions associated with attaining alternative PM-2.5 standards (as opposed to current standards), are given in Exhibit 8.1 for Philadelphia County in 1992-1993 and Exhibit 8.2 for Southeast Los Angeles County in 1995. Because Southeast Los Angeles County is not in attainment of current PM-10 standards (and was not in attainment in 1995), PM-2.5 concentrations were adjusted prior to the risk analyses, to simulate attainment of current standards. The method for adjusting daily PM-2.5 concentrations to simulate attainment of alternative PM-2.5 standards and to simulate attainment of current PM-10 standards (in Southeast Los Angeles County) is described in Section 2.2.

The results of the analyses in this second phase of the risk analysis follow a pattern similar to that of the first phase. Predicted reductions in health effects incidence and predicted reductions in PM-related percent of total incidence associated with attaining alternative PM-2.5 standards in Southeast Los Angeles County are uniformly greater than those predicted in Philadelphia County. The generally higher pollution levels and greater population size in Southeast Los Angeles County are, as in the first phase of the risk analysis, the primary reasons for this.

Because reduction of PM by the linear rollback method removes a given percent of daily PM over background, the amount of PM removed each day depends on what background PM concentration is. Analyses were conducted to illustrate the sensitivity of predicted avoided mortality to changes in assumed background PM concentration. The results are shown in Exhibit 8.3 for Philadelphia County and Exhibit 8.4 for Southeast Los Angeles County.

8.2 An assessment of the plausibility of linear rollbacks and associated sensitivity analysis

As described in Section 2.2, the method of adjusting daily PM concentrations to simulate attainment of alternative standards could significantly influence the results of these analyses, especially when the alternative standard is a daily standard.

To assess the plausibility of the linear rollback method, analyses were carried out to evaluate the extent to which historical changes in PM-2.5 air quality can be modeled using linear rollbacks. The historic changes in PM-2.5 have not been the result of a PM-2.5 control strategy, however. The PM-2.5 changes likely result from control programs for other pollutants (especially PM-10, ozone, and sulfates) and from weather variability. The pattern of changes that have occurred in the past, therefore, may not necessarily accurately estimate the changes that may occur from future control programs.

Exhibit 8.1

Estimated Changes in Health Risks Associated with Meeting Alternative PM-2.5 Standards in Philadelphia County, September 1992 - August 1993 (for base case assumptions)

The Daily Standards Allow One Exceedance at Each Monitor; the Annual Standards Apply to the Annual Average at Each Monitor#

Health Effects*		PM-2.5-Associated Incidence associated with current standards**	Reduction in Incidence Associated with Meeting Alternative Standards			
			20 ug/m3 annual	20 ug/m3 annual and 65 ug/m3 daily	20 ug/m3 annual and 50 ug/m3 daily	20 ug/m3 annual and 25 ug/m3 daily
Mortality	(A) Associated with short-term exposure (all ages)	370 (230 - 510)	0 (0 - 0)	40 (20 - 60)	120 (70 - 170)	260 (160 - 360)
	Percent Reduction in PM-Associated Incidence:* **		0.0%	10.8%	32.4%	70.3%
	Percent Reduction in Total Incidence:** **		0.0%	0.2%	0.6%	1.3%
	(B) Associated with long-term exposure (age 30 and over)	860 (540-1170)	0 (0 - 0)	170 (130-290)	500 (320-720)	860 (540-1170)
	Percent Reduction in PM-Associated Incidence:		0.0%	20.0%	57.9%	100.0%
	Percent Reduction in Total Incidence:		0	0.9%	2.7%	4.7%
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	260 (70 - 450)	0 (0 - 0)	30 (10 - 50)	90 (20 - 150)	180 (50 - 310)
	Percent Reduction in PM-Associated Incidence:		0.0%	11.5%	34.6%	69.2%
	Percent Reduction in Total Incidence:		0.0%	0.2%	0.7%	1.4%
Hospital Admissions Cardiac	(D) Ischemic Heart Disease***** (>64 years old)	70 (30 - 120)	0 (0 - 0)	10 (0 - 10)	20 (10 - 40)	50 (20 - 80)
	(E) Congestive Heart Failure***** (>64 years old)	100 (50 - 150)	0 (0 - 0)	10 (10 - 20)	30 (20 - 50)	70 (30 - 110)
	Range of Percent Reductions in PM-Associated Incidence:		0.0% - 0.0%	10.0% - 14.3%	28.6% - 30.0%	70.0% - 71.4%
	Range of Percent Reductions in Total Incidence:		0.0% - 0.0%	0.1% - 0.1%	0.2% - 0.4%	0.5% - 0.9%
(F) Lower Respiratory Symptoms (8-12 yr. olds) ******		< 11000 > (6000 - 15000)	< 0 > (0 - 0)	< 1000 > (1000 - 2000)	< 4000 > (2000 - 6000)	< 8000 > (4000 - 11000)
	Percent Reduction in PM-Associated Incidence:		0.0%	9.1%	36.4%	72.7%
	Percent Reduction in Total Incidence:		0.0%	1.8%	7.3%	14.6%

* Health effects are associated with short-term exposure to PM, unless otherwise specified.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 3.5 ug/m3 in Philadelphia County.

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by meeting both a 20 ug/m3 annual and a 65 ug/m3 daily standard is 40/370=10.8%.

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence.

***** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

******Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates.

Sources of Concentration-Response (C-R) Functions:
(A) C-R function based on pooled results from studies in six locations.
(B) Pope et al., 1995
(C) Thurston, et al., 1994
(D) Schwartz & Morris, 1995
(E) Schwartz & Morris, 1995
(F) Schwartz, et al., 1994

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

#The one exceedance form of the daily std. requires that the second highest concentration (the second daily max.) at each monitor (rounded to the nearest ug/m3) meets the std. The highest second daily maximum concentration at a monitor in Philadelphia is 72.6 ug/m3. The annual standard requires that the annual average at each monitor (rounded to the nearest 0.1 ug/m3) meets the std. The highest annual avg. at a monitor in Philadelphia is 17.1 ug/m3. Therefore the 20 ug/m3 annual standard is already met in Philadelphia.

Exhibit 8.1 (cont.)

Estimated Changes in Health Risks Associated with Meeting Alternative PM-2.5 Standards in Philadelphia County, September 1992 - August 1993 (for base case assumptions)

The Daily Standards Allow One Exceedance at Each Monitor; the Annual Standards Apply to the Annual Average at Each Monitor#

Health Effects*		PM-2.5-Associated Incidence associated with current standards**	Reduction in Incidence Associated with Meeting Alternative Standards			
			15 ug/m3 annual	15 ug/m3 annual and 65 ug/m3 daily	15 ug/m3 annual and 50 ug/m3 daily	15 ug/m3 annual and 25 ug/m3 daily
Mortality	(A) Associated with short-term exposure (all ages)	370 (230 - 510)	60 (40 - 80)	60 (40 - 80)	120 (70 - 170)	260 (160 - 360)
	Percent Reduction in PM-Associated Incidence:* **		16.2%	16.2%	32.4%	70.3%
	Percent Reduction in Total Incidence:** **		0.3%	0.3%	0.6%	1.3%
	(B) Associated with long-term exposure (age 30 and over)	860 (540-1170)	230 (170-380)	230 (170-380)	500 (5320-720)	860 (540-1170)
	Percent Reduction in PM-Associated Incidence:		27.4%	27.4%	57.9%	100.0%
	Percent Reduction in Total Incidence:		1.3%	1.3%	2.7%	4.7%
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	260 (70 - 450)	40 (10 - 70)	40 (10 - 70)	90 (20 - 150)	180 (50 - 310)
	Percent Reduction in PM-Associated Incidence:		15.4%	15.4%	34.6%	69.2%
	Percent Reduction in Total Incidence:		0.3%	0.3%	0.7%	1.4%
Hospital Admissions Cardiac	(D) Ischemic Heart Disease***** (>64 years old)	70 (30 - 120)	10 (0 - 20)	10 (0 - 20)	20 (10 - 40)	50 (20 - 80)
	(E) Congestive Heart Failure***** (>64 years old)	100 (50 - 150)	20 (10 - 20)	20 (10 - 20)	30 (20 - 50)	70 (30 - 110)
	Range of Percent Reductions in PM-Associated Incidence:		14.3% - 20.0%	14.3% - 20.0%	28.6% - 30.0%	70.0% - 71.4%
	Range of Percent Reductions in Total Incidence:		0.1% - 0.3%	0.1% - 0.3%	0.2% - 0.4%	0.5% - 0.9%
(F) Lower Respiratory Symptoms (8-12 yr. olds) ******		< 11000 > (6000 - 15000)	< 2000 > (1000 - 3000)	< 2000 > (1000 - 3000)	< 4000 > (2000 - 6000)	< 8000 > (4000 - 11000)
	Percent Reduction in PM-Associated Incidence:		18.2%	18.2%	36.4%	72.7%
	Percent Reduction in Total Incidence:		3.6%	3.6%	7.3%	14.6%

* Health effects are associated with short-term exposure to PM, unless otherwise specified.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 3.5 ug/m3 in Philadelphia County.

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by meeting both a 15 ug/m3 annual and a 65 ug/m3 daily standard is 60/370=16.2%.

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence.

***** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

******Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates.

Sources of Concentration-Response (C-R) Functions:
(A) C-R function based on pooled results from studies in six locations.
(B) Pope et al., 1995
(C) Thurston, et al., 1994
(D) Schwartz & Morris, 1995
(E) Schwartz & Morris, 1995
(F) Schwartz, et al., 1994

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

#The one exceedance form of the daily std. requires that the second highest concentration (the second daily max.) at each monitor (rounded to the nearest ug/m3) meets the std. The highest second daily maximum concentration at a monitor in Philadelphia is 72.6 ug/m3.
The annual standard requires that the annual average at each monitor (rounded to the nearest 0.1 ug/m3) meets the std. The highest annual avg. at a monitor in Philadelphia is 17.1 ug/m3. Therefore the 20 ug/m3 annual standard is already met in Philadelphia.

Exhibit 8.2

Estimated Changes in Health Risks Associated with Meeting Alternative PM-2.5 Standards in Southeast Los Angeles County, 1995* (for base case assumptions)

The Daily Standards Allow One Exceedance at Each Monitor; the Annual Standards Apply to the Annual Average at Each Monitor#

Health Effects		PM-2.5-Related Incidence associated with current standards**	Reduction in Incidence Associated with Meeting Alternative Standards: 20 ug/m3 annual	20 ug/m3 annual and 65 ug/m3 daily	20 ug/m3 annual and 50 ug/m3 daily	20 ug/m3 annual and 25 ug/m3 daily
Mortality	(A) Associated with short-term exposure (all ages)	710 (430 - 970)	140 (80 - 190)	270 (160 - 370)	370 (220 - 510)	550 (330 - 750)
	Percent Reduction in PM-Associated Incidence:* **		19.7%	38.0%	52.1%	77.5%
	Percent Reduction in Total Incidence:** **		0.6%	1.1%	1.5%	2.2%
	(B) Associated with long-term exposure (age 30 and over)	2050 (1290-2770)	550 (340 - 760)	1130 (710-1550)	1580 (990-2150)	2050 (1290 - 2770)
	Percent Reduction in PM-Associated Incidence:		27.0%	55.5%	77.3%	100.0%
	Percent Reduction in Total Incidence:		2.3%	4.8%	6.6%	8.6%
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	940 (250 - 1630)	180 (50 - 310)	350 (90 - 600)	490 (130 - 850)	730 (200 - 1260)
	Percent Reduction in PM-Associated Incidence:		19.1%	37.2%	52.1%	77.7%
	Percent Reduction in Total Incidence:		1.2%	2.3%	3.2%	4.7%
Hospital Admissions Cardiac	(D) Ischemic Heart Disease ***** (>64 years old)	130 (50 - 200)	20 (10 - 40)	50 (20 - 80)	70 (30 - 110)	100 (40 - 160)
	(E) Congestive Heart Failure ***** (>64 years old)	140 (70 - 210)	30 (10 - 40)	50 (30 - 80)	70 (40 - 110)	110 (50 - 170)
	Range of Percent Reductions in PM-Associated Incidence:		15.4% - 21.4%	35.7% - 38.5%	50.0% - 53.8%	76.9% - 78.6%
	Range of Percent Reductions in Total Incidence:		0.2% - 0.4%	0.4% - 0.7%	0.6% - 1.0%	0.9% - 1.6%
(F) Lower Respiratory Symptoms (8-12 yr. olds)******		< 43000 > (23000 - 58000)	< 10000 > (5000 - 15000)	< 19000 > (9000 - 27000)	< 25000 > (13000 - 36000)	< 35000 > (18000 - 49000)
	Percent Reduction in PM-Associated Incidence:		23.3%	44.2%	58.1%	81.4%
	Percent Reduction in Total Incidence:		6.7%	12.7%	16.7%	23.3%

Health effects are associated with short-term exposure to PM, unless otherwise specified.

* Los Angeles County was not in attainment of current PM-10 standards in 1995. Figures shown assume actual PM-10 concentrations are first rolled back to simulate attainment of these standards, and that actual PM-2.5 concentrations are rolled back by the same percent as PM-10. See text in Chapter VI for details.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 2.5 ug/m3 in Southeast Los Angeles County.

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by meeting both a 20 ug/m3 annual and a 65 ug/m3 daily standard is 270/710 = 38.0%.

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence.

***** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

******Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates.

Sources of Concentration-Response (C-R) Functions:
(A) C-R function based on pooled results from studies in 6 locations
(B) Pope et al., 1995
(C) Thurston, et al., 1994
(D) Schwartz & Morris, 1995
(E) Schwartz & Morris, 1995
(F) Schwartz, et al., 1994

The numbers in parentheses for pooled studies are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

#The one exceedance form of the daily std. requires that the second highest concentration (the second daily max.) at each monitor (rounded to the nearest ug/m3) meets the std. The highest second daily maximum concentration at a monitor in L.A. is 101.7 ug/m3 (after adjustment to simulate attainment of current stds). The annual standard requires that the annual average at each monitor (rounded to the nearest 0.1 ug/m3) meets the std. The highest annual avg. at a monitor in L.A. is 24.1 ug/m3 (after adjustment to simulate attainment of current stds).

Exhibit 8.2 (cont.)

Estimated Changes in Health Risks Associated with Meeting Alternative PM-2.5 Standards in Southeast Los Angeles County, 1995* (for base case assumptions)

The Daily Standards Allow One Exceedance at Each Monitor; the Annual Standards Apply to the Annual Average at Each Monitor#

Health Effects		PM-2.5-Related Incidence associated with current standards**	Reduction in Incidence Associated with Meeting Alternative Standards			
			15 ug/m3 annual	15 ug/m3 annual and 65 ug/m3 daily	15 ug/m3 annual and 50 ug/m3 daily	15 ug/m3 annual and 25 ug/m3 daily
Mortality	(A) Associated with short-term exposure (all ages)	710 (430 - 970)	300 (180 - 410)	300 (180 - 410)	370 (220 - 510)	550 (330 - 750)
	Percent Reduction in PM-Associated Incidence:* **		42.3%	42.3%	52.1%	77.5%
	Percent Reduction in Total Incidence:** **		1.2%	1.2%	1.5%	2.2%
	(B) Associated with long-term exposure (age 30 and over)	2050 (1290-2770)	1260 (790-1720)	1220 (790-1720)	1580 (990-2150)	2050 (1290-2770)
	Percent Reduction in PM-Associated Incidence:		61.6%	61.6%	77.3%	100.0%
	Percent Reduction in Total Incidence:		5.3%	5.3%	6.6%	8.6%
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	940 (250 - 1630)	400 (110 - 680)	400 (110 - 680)	490 (130 - 850)	730 (200 - 1260)
	Percent Reduction in PM-Associated Incidence:		42.6%	42.6%	52.1%	77.7%
	Percent Reduction in Total Incidence:		2.6%	2.6%	3.2%	4.7%
Hospital Admissions Cardiac	(D) Ischemic Heart Disease ***** (>64 years old)	130 (50 - 200)	50 (20 - 90)	50 (20 - 90)	70 (30 - 110)	100 (40 - 160)
	(E) Congestive Heart Failure ***** (>64 years old)	140 (70 - 210)	60 (30 - 90)	60 (30 - 90)	70 (40 - 110)	110 (50 - 170)
	Range of Percent Reductions in PM-Associated Incidence:		38.5% - 42.9%	38.5% - 42.9%	50.0% - 53.8%	76.9% - 78.6%
	Range of Percent Reductions in Total Incidence:		0.4% - 0.8%	0.4% - 0.8%	0.6% - 1.0%	0.9% - 1.6%
(F) Lower Respiratory Symptoms (8-12 yr. olds)******		< 43000 > (23000 - 58000)	< 21000 > (10000 - 30000)	< 21000 > (10000 - 30000)	< 25000 > (13000 - 36000)	< 35000 > (18000 - 49000)
	Percent Reduction in PM-Associated Incidence:		48.8%	48.8%	58.1%	81.4%
	Percent Reduction in Total Incidence:		14.0%	14.0%	16.7%	23.3%

Health effects are associated with short-term exposure to PM, unless otherwise specified.

* Los Angeles County was not in attainment of current PM-10 standards in 1995. Figures shown assume actual PM-10 concentrations are first rolled back to simulate attainment of these standards, and that actual PM-2.5 concentrations are rolled back by the same percent as PM-10. See text in Chapter VI for details.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 2.5 ug/m3 in Southeast Los Angeles County.

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by meeting both a 15 ug/m3 annual and a 65 ug/m3 daily standard is 300/710 = 42.3%.

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence.

***** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

******Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates.

Sources of Concentration-Response (C-R) Functions:
(A) C-R function based on pooled results from studies in 6 locations
(B) Pope et al., 1995
(C) Thurston, et al., 1994
(D) Schwartz & Morris, 1995
(E) Schwartz & Morris, 1995
(F) Schwartz, et al., 1994

The numbers in parentheses for pooled studies are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

#The one exceedance form of the daily std. requires that the second highest concentration (the second daily max.) at each monitor (rounded to the nearest ug/m3) meets the std.
The highest second daily maximum concentration at a monitor in L.A. is 101.7 ug/m3 (after adjustment to simulate attainment of current stds).
The annual standard requires that the annual average at each monitor (rounded to the nearest 0.1 ug/m3) meets the std.
The highest annual avg. at a monitor in L.A. is 24.1 ug/m3 (after adjustment to simulate attainment of current stds).

Exhibit 8.3

Sensitivity Analysis: The Effect of Alternative Background Levels on Estimated Changes in Health Risks Associated with Meeting a PM-2.5 Standard of 15 ug/m3 Annual, 50 ug/m3 Daily in Philadelphia County, September 1992 - August 1993 (for base case assumptions)

Health Effects*		PM-2.5-Associated Incidence associated with current standards** Background = 3.5 ug/m3	Reduction in Incidence Associated with Meeting Standard		
			Background = 3.5 ug/m3	Background = 2.0 ug/m3	Background = 5.0 ug/m3
Mortality	(A) Associated with short-term exposure (all ages)	370 (230 - 510)	120 (70 - 170)	130 (80 - 190)	110 (70 - 150)
	Percent Reduction in PM-Associated Incidence:* **		32.4%	35.1%	29.7%
	Percent Reduction in Total Incidence:** **		0.6%	0.6%	0.5%
	(B) Associated with long-term exposure (age 30 and over)	860 (540-1170)	550 (500-720)	540 (360-780)	450 (300-650)
	Percent Reduction in PM-Associated Incidence:		57.9%	63.2%	52.6%
	Percent Reduction in Total Incidence:		2.7%	3.0%	2.5%

* Health effects are associated with short-term exposure to PM, unless otherwise specified.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 3.5 ug/m3 in Philadelphia County.

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by meeting both a 15 ug/m3 annual and a 50 ug/m3 daily standard, assuming that background PM-2.5 concentration is 3.5 ug/m3 is 120/370 = 32.4%.

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence.

The numbers in parentheses for pooled functions (mortality associated with short-term exposure) are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% confidence intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (CR) functions:
(A) C-R function based on pooled results from studies in 6 locations.
(B) Pope et al., 1995

Exhibit 8.4

Sensitivity Analysis: The Effect of Alternative Background Levels on Estimated Changes in Health Risks Associated with Meeting a PM-2.5 Standard of 15 ug/m3 Annual, 50 ug/m3 Daily Southeast Los Angeles County, 1995

Health Effects*		PM-2.5-Associated Incidence associated with current standards** Background = 2.5 ug/m3	Reduction in Incidence Associated with Meeting Standard		
			Background = 2.5 ug/m3	Background = 1.0 ug/m3	Background = 4.0 ug/m3
Mortality	(A) Associated with short-term exposure (all ages)	710 (430 - 970)	370 (220 - 510)	390 (240 - 540)	350 (210 - 480)
	Percent Reduction in PM-Associated Incidence:* **		52.1%	54.9%	49.3%
	Percent Reduction in Total Incidence:** **		1.5%	1.6%	1.4%
	(B) Associated with long-term exposure (age 30 and over)	2050 (1290-2770)	1580 (990-2150)	1670 (1050-2270)	1480 (930-2030)
	Percent Reduction in PM-Associated Incidence:		77.3%	81.5%	72.5%
	Percent Reduction in Total Incidence:		6.6%	7.0%	6.2%

* Health effects are associated with short-term exposure to PM, unless otherwise specified.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 2.5 ug/m3 in Los Angeles County.

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by meeting both a 15 ug/m3 annual and a 50 ug/m3 daily standard, assuming that background PM-2.5 concentration is 2.5 ug/m3 is 370/710 = 52.1%.

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence.

The numbers in parentheses for pooled functions (short-term exposure) are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter 7 for details.

Sources of Concentration-Response (CR) functions:
(A) C-R function based on pooled results from studies in 6 locations.
(B) Pope et al., 1995

The historic PM-2.5 data come from monitors operated by the California Air Resources Board (CARB) and from the EPA's National Aerometric Monitoring System (NAMS) and the Harvard monitoring system. Some monitors reported concentrations on only a few days in a given year; such data were excluded. Only monitor-years represented by fifty or more days were included in the analysis. This corresponds to standard one-in-six monitoring, with allowances for a limited number of missed days. Exhibit 8.5 shows the number of days available at each monitor for each year (blank entries indicate either no monitoring or insufficient monitoring). In all, 230 monitor-years from 57 sites are represented.

Air quality data from different years at a single monitor could not be compared directly, because different years were represented by different numbers of days. Therefore, the reported concentrations from each monitor-year were grouped into deciles[16], and all of the observations in each decile were averaged to produce a representative concentration for that decile. The averaging (as opposed to selecting specific percentile values) was meant to promote stability of results between two monitor-years actually reporting the same air quality (with sampling error).[17] The second-highest reported concentration was also retained for each year. Each monitor-year was therefore represented by ten decile averages plus the second highest reported concentration.

To determine the extent to which the historic air quality changes were linear, the deciles from one monitor-year were regressed against the corresponding deciles from another monitor-year. In the primary analyses consecutive years from single monitors were compared; these results were later compared to those comparing the years from a given site with highest and lowest average reported concentrations.

A regression gives a linear equation of the form

$$y = \alpha x + \beta \quad .$$

A linear rollback over background, however, is represented by an equation of the form

$$y = A(x - B) + B \ ,$$

where x is the earlier year's PM decile, y is the later year's PM decile, and B is background concentration, which is subtracted from all PM concentrations, and therefore from each PM decile, x. If the equations are to agree -- that is, if the relationship between one year's PM

[16] Each decile was one tenth of the observations, rather than spanning one tenth of the concentration range.

[17] Analysis of data from several years in Pittsburgh indicated that most of the distribution was in any case fairly uniformly distributed, with significant variations only at the highest concentrations reported.

Exhibit 8.5
PM-2.5 Monitors with at Least Two Years In Which >50 Days Each Year Have Reported PM Concentrations: Number of Days with Reported Concentrations

				82	83	84	85	86	87	88	89	90	91	92	93	94
CARB Monitors																
		Monitor														
		1000246	Fresno, CA										60	56	60	61
		1300694	El Centro, CA								58	59	55	58	61	57
		1400699	Lone Pine, CA								56	58	53	52	52	
		1500203	Bakersfield, CA								58	58	55	55	55	
		1500250	Taft College, CA										56		57	
		1600715	Corcoran, CA												61	60
		1700720	Anderson Springs, CA								61	57				
		1700728	Glenbrook, CA								62	52				
		2000002	Madera, CA								59			59	60	62
		2600785	Mammoth Lakes, CA								58	59		59		
		2900794	Truckee, CA							52	54					
		3300144	Rubidoux, CA								55	54	51	56	60	60
		3400305	Sacramento, CA											66	73	80
		3600188	Trona, CA								53	52			56	
		3900252	Stockton-Hazelton, CA								56	62	55		60	60
		4300382	San Jose, CA									56	58	59	61	61
		5000567	Modesto, CA										58	61	60	62
		5400568	Visalia, CA								57	54	53	58	61	60
		7000060	Azusa, CA									58	53	56	58	58
		7000072	North Long Beach, CA								60		57	60	61	60
AIRS Monitors																
State	County	Monitor														
6	19	5	Fresno, CA							60	57					
	37	4002	Long Beach, CA							60	60					
	65	8001	Rubidoux, CA							57	54					
	107	2002	Visalia, CA							61	57					
22	71	10	New Orleans, LA					141	111	61	61	59				
23	3	1005	Presque Isle, ME		51	85										
	9	3	Acadia Nat'l Park, ME				74	95	111	92						
	11	1	Augusta, ME	87	66											
25	15	4002	Ware, MA					188	90							
	25	2	Boston, MA					90	68							
29	189	2003	Clayton, MO (SL Co.)								61		57	59	59	
		5001	Ferguson, MO (SL Co.)				104	169	173	63	58	57	59	60	58	
		7002	St. Ann, MO (SL Co.)											61	59	
34	13	11	Newark, NJ						59	60	58	58				
	39	4	Elizabeth, NJ						61	59	53	57				
36	61	56	New York City, NY								53					51
		69	New York City, NY						58		52	52				
		77	New York City, NY							59	55			56	52	55
	67	1016	Syracuse, NY									53	55	57	58	
37	67	9	Winston-Salem, NC					54	60	52						
41	5	4	Canby, OR		59	91	61									
	17	1	Bend, OR			56	54	57								
	29	1001	Central Point, OR			59	58	61								
		3001	Medford, OR			60	58									
	51	15	Portland, OR		65	91	71	57								
		80	Portland, OR		60	82	65	58								
42	3	21	Pittsburgh, PA					56	55							
		27	Pittsburgh, PA				86	170	171	166	167	159	164			
	101	4	Philadelphia, PA						59		61	59				
45	79	7	Columbia, SC										62		84	
48	29	36	San Antonio, TX				110	164	179	115						
	141	37	El Paso, TX				128	164	134	81						
	167	1002	Texas City, TX				86	93								
	201	24	Harris Co., TX				111	125	146	71						
	355	12	Corpus Christi, TX				112	155	160	88						
	439	60	Fort Worth, TX				112	132								
49	35	3	Salt Lake Co., UT					54	56							

deciles and another year's PM deciles is to be linear then it is necessary that A equal α, and

$$B = \frac{\beta}{1 - A} .$$

Since changes in air quality from year to year tend to be small, however (since $A \approx 1$), the denominator in the equation above is often close to zero. Therefore, small changes in the estimate of A lead to large changes in the estimated background, and regressions for different years estimate backgrounds differing by an order of magnitude (and in some cases substantially higher than maximum PM-2.5 concentrations). In order to avoid this, the intercept was constrained to be zero in all regressions. When background concentrations were considered, they were actually subtracted from all concentration measurements, and regressions again performed with the intercept constrained to be zero.

All estimated slope coefficients were significant at the 0.01 confidence level, and the regressions explained the vast majority of the variation in almost all year pairs. Exhibit 8.6 describes the distributions of r^2 values obtained in four sets of regressions. The first set includes all consecutive years at single monitors. The second set considers a single pair of years from each monitor, the year with the lowest average concentration and the year with the highest average concentration. These two sets assume no background concentration. The final two sets consider consecutive year pairs in the eastern and western United States separately, incorporating an estimate of background concentration in each case. Note that including reasonable estimates of background concentrations improves the predictive power of the worst of the regressions (a minimum r^2 with estimated backgrounds of 0.71, as opposed to 0.61).

Exhibit 8.6. Distributions of r^2 Statistics for Regressions on Different Sets of Year-Pairs

	N	Mean (St. Dev.)	Min	5th %-ile	95th %-ile
All, Consecutive Years (no background)	130	0.95 (0.06)	0.61	0.86	0.99
All, High Year vs. Low Year (no background)	57	0.94 (0.07)	0.63	0.79	0.99
East, Consecutive Years (background = 3.5 $\mu g/m^3$)	45	0.96 (0.04)	0.77	0.88	0.99
West, Consecutive Years (background = 2.0 $\mu g/m^3$)	85	0.95 (0.05)	0.71	0.86	0.99

The statistics in Exhibit 8.6 show that a linear rollback above some background can account for the vast majority of the change in PM concentrations between two years. However, predicting the proper slope (percent change) is more difficult. The percent change

in the mean, as might be expected, is generally close to the percent change predicted by the regression. Therefore, rollbacks to meet annual standards present little problem. However, the percent change necessary to bring the second highest value down to meet a standard is not particularly well correlated (correlation coefficient ≈ 0.66) with the percent change indicated by the regression. Exhibit 8.7 gives some statistics on the distribution of the ratio of the regression slope to the ratio of second highest values. When the two percent changes are in agreement, the ratio is one. When the second high changes more than the distribution as a whole, the ratio is less than one, and when the second high changes less than the distribution as a whole, the ratio is greater than one.

Exhibit 8.7. Statistics on the Distribution of (Regression Slope)/(Ratio of Second High Values) All Consecutive Years (129 pairs)

Mean	1.02
Standard Deviation	0.18
1st percentile	0.66
5th percentile	0.72
25th percentile	0.90
50th percentile	1.03
75th percentile	1.12
95th percentile	1.27
99th percentile	1.62

These statistics, along with the correlation coefficient of 0.66, show that the regression slopes are not well predicted by the ratio of second high values. (Linear regression accounts for 43% of the variation; exponential and logarithmic forms do worse.)

A sensitivity analysis was carried out for Philadelphia County to examine the effect of different rollback methods on PM-related health effects. The results are given in Exhibits 8.8 and 8.9. Rollbacks designed to meet annual standards, which remove the same total amount of PM from the air no matter how the reductions are distributed, result in similar changes in incidence. They result in exactly the same reductions when the reductions are calculated using functions relying on only the annual mean. The small differences produced by the different methods when reductions are measured using functions relying on daily PM concentrations are due to the slight nonlinearity of the functions. (Linear functions would produce identical results under the three methods.)

Rollbacks designed to simulate the attainment of daily standards, however, produce notably different results, since they result in the elimination of widely different amounts of PM from the air. The percent rollback necessary to bring the second highest value down to a given value remains the same no matter what the rollback method; however, the amount of PM removed from the lower 90% of the distribution changes. When the higher concentrations are reduced more than the lower, the result is that the lower concentrations are reduced less than under a strictly proportional rollback, so less PM is removed from the air and health effects are reduced less. Conversely, when higher concentrations are reduced less than lower ones, the result is that the lower concentrations are reduced more than under a strictly proportional rollback, so more PM is removed from the air and health effects are reduced more.

The degree to which these deviations from proportional rollbacks might be expected to be observed in areas attempting to come into attainment with new standards is unclear, especially the case in which higher concentrations are reduced less than those in the bulk of the distribution. For any sets of standards in which daily standards were controlling in an area, a set of control strategies that reduced high concentrations less than the overall distribution would actually make achieving attainment more difficult, although this case might approximate an instance in which reducing high peaks was particularly difficult for some reason.

Exhibit 8.8

Sensitivity Analysis: Effect of Alternative Rollback Methods on Mortality Estimates
Short-term Exposure (Pooled Function) and Long-term Exposure PM-2.5 Mortality Functions
Philadelphia County, September 1992 - August 1993

Initial Air Quality: 16.5 ug/m3 annual average, 69.3 ug/m3 2nd daily maximum

		Percent Change in PM-Associated Incidence*			Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback	
	Alternative Standard	**All PM concentrations rolled back equally**	Higher PM concentrations reduced more	Higher PM concentrations reduced less	Higher PM concentrations reduced more	Higher PM concentrations reduced less
(A) Mortality associated with short-term exposure	15 ug/m3 annual	**11.4%**	11.4%	11.4%	100.4%	100.4%
	50 ug/m3 daily	**29.7%**	21.5%	39.0%	72.6%	131.3%
(B) Mortality associated with long-term exposure	15 ug/m3 annual	**20.8%**	20.8%	20.9%	100.2%	100.5%
	50 ug/m3 daily	**53.8%**	39.1%	70.5%	72.6%	131.1%

* Health effects incidence was quantified across the range of PM concentrations observed in each study, but not below background PM-2.5 level, which is assumed to be 3.5 ug/m3.

(A) C-R function based on studies in 6 cities
(B) Pope et al., 1995

In the alternative rollback cases, the upper 10% of the PM distribution was reduced by more or less than the lower 90%. See text in Section 8.2 for details.

Exhibit 8.9

Sensitivity Analysis: Effect of Alternative Rollback Methods on Mortality Estimates
Short-term Exposure (Pooled Function) and Long-term Exposure PM-2.5 Mortality Functions
Philadelphia County, September 1992 - August 1993
Details of Rollbacks for Proportional and One Alternative Rollback

Initial Air Quality: 16.5 ug/m3 annual average, 69.3 ug/m3 2nd daily maximum

		Entire AQ distribution reduced equally			Upper 10% of AQ distribution reduced more			
	Alternative Standard	AQ Rollback Required	Resulting AQ (Annual Mean / 2nd Daily Max)	Percent change in PM-Associated Incidence*	AQ Rollback Required (upper / lower)	Resulting AQ (Annual Mean / 2nd Daily Max)	Percent Change in PM-Associated Incidence*	Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback**
(A) Mortality associated with short-term exposure	15 ug/m3 annual	11.3%	15.0 61.9	11.4%	15.6% 9.8%	15.0 59.2	11.4%	100.4%
	50 ug/m3 daily	29.4%	12.7 50.0	29.7%	29.4% 18.4%	13.7 50.0	21.5%	72.6%
(B) Mortality associated with long-term exposure	15 ug/m3 annual	11.3%	15.0 61.9	20.8%	15.6% 9.8%	15.0 59.2	20.8%	100.2%
	50 ug/m3 daily	29.4%	12.7 50.0	53.8%	29.4% 18.4%	13.7 50.0	39.1%	72.6%

* Health effects incidence was quantified across the range of PM concentrations observed in each study, but not below background PM-2.5 level, which is assumed to be 3.5 ug/m3.

** The percent of PM-associated incidence achieved by the alternative rollback method (i.e., the upper 10% of the air quality distribution being reduced by more than the lower 90% of the distribution). For example, in the second row 62.6% = 18.6%/29.7% x 100.

(A) C-R function based on studies in 6 cities
(B) Pope et al., 1995

8.3. Uncertainty analyses

Section 7.2 describes the integrated uncertainty analysis methods. Similar analyses were conducted to assess the uncertainty surrounding estimates of avoided health effects associated with attaining alternative PM-2.5 standards. Results are presented in terms of the number of cases avoided, rather than the number of cases remaining.

Exhibit 8.10 shows the estimated health benefits associated with attaining an annual standard of 15 $\mu g/m^3$ and a daily standard of 50 $\mu g/m^3$ in Southeast Los Angeles County**,** progressively including more sources of uncertainty from left to right in the diagram. The first line shows the estimate when only uncertainty in relative risk is included. The next line shows the estimate when uncertainty in relative risk and background (but not cutpoint) is included, and the next three lines show the estimates using the three cutpoint weighting cases. The four lines on the right repeat the last four lines on the left, adding uncertainty in the form of the rollback. The diagram shows that adding this uncertainty does not significantly change the uncertainty in the estimates produced by the model.

Exhibit 8.11 compares the benefits associated with meeting an annual standard of 15 $\mu g/m^3$ and either no daily standard or a daily standard of 65, 50, or 25 $\mu g/m^3$ in Southeast Los Angeles County. For each standard, estimates are presented assuming that the cutpoint is equal to background, as well as for cutpoint weighting cases I, II, and III. All the estimates presented in Exhibit 8.11 include uncertainty in the form of the rollback, as described in Section 8.2 and Exhibit 8.7.

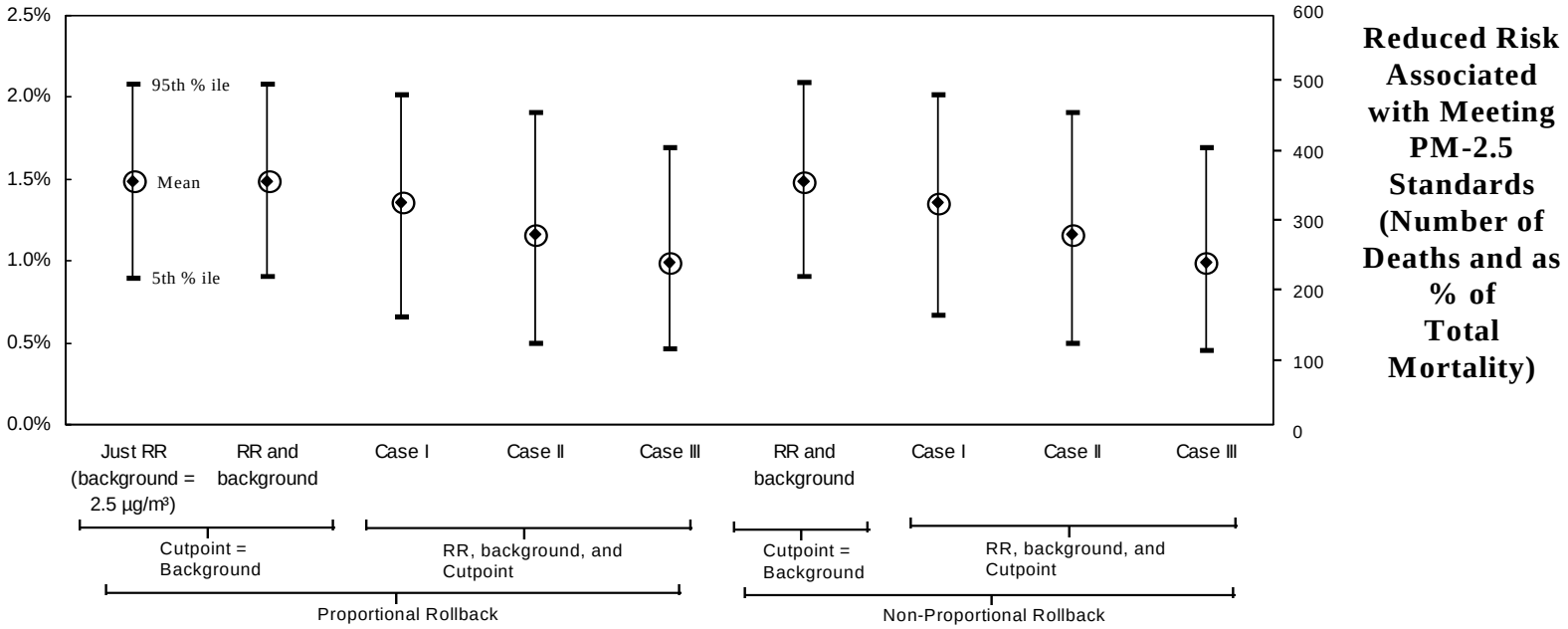
Exhibit 8.10

Uncertainty Analysis: Effect of Uncertainty of Relative Risk, Background Concentration, Cutpoint, Slope Adjustment Method, and Form of Rollback

Reduced Risk Associated with Meeting a PM-2.5 Standard of 15 μg/m^3 Annual and 50 μg/m^3 Daily

Mortality Associated With Short-term Exposure to PM-2.5

Southeast Los Angeles County, 1995 (Population: 3.6 Million)



Cutpoint Weighting Schemes

	Case I	Case II	Case III
Background	0.5	0.2	0.05
10 μg/m^3	0.3	0.3	0.15
18 μg/m^3	0.15	0.3	0.5
30 μg/m^3	0.05	0.2	0.3

Mean Reduced Risk as % of Total PM-Associated Risk

	Just RR	RR and Background	Case I	Case II	Case III
Proportional	52.1%	52.3%	60.5%	69.5%	78.8%
Non-Proportional	--	52.4%	60.7%	70.0%	79.2%

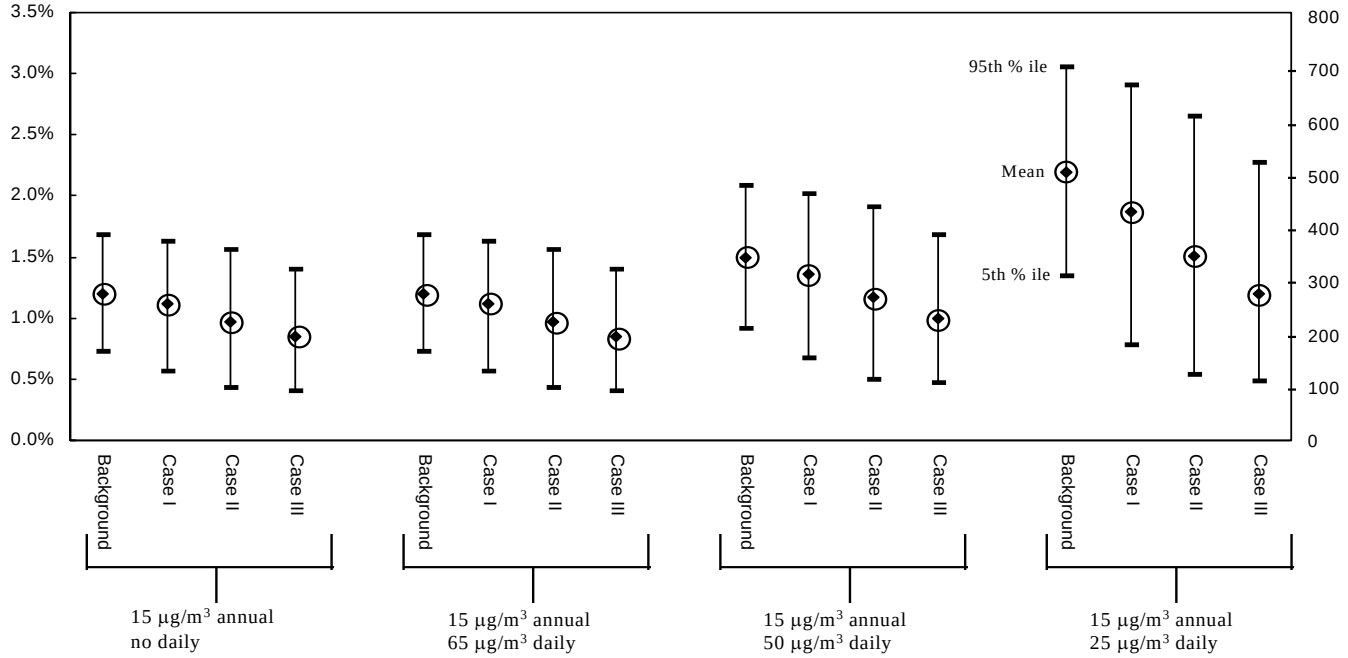
Exhibit 8.11

Uncertainty Analysis: Effect of Uncertainty of Relative Risk, Background Concentration, Cutpoint, Slope Adjustment Method, and Form of Rollback

Reduced Risk Associated with Meeting Alternative PM-2.5 Standards

Mortality Associated With Short-term Exposure to PM-2.5

Southeast Los Angeles County, 1995 (Population: 3.6 Million)



Reduced Risk Associated with Meeting PM-2.5 Standards (Number of Deaths and as % of Total Mortality)

Cutpoint Weighting Schemes

	Case I	Case II	Case III
Background	0.5	0.2	0.05
10 µg/m³	0.3	0.3	0.15
18 µg/m³	0.15	0.3	0.5
30 µg/m³	0.05	0.2	0.3

Mean Reduced Risk as % of Total PM-Associated Risk

(µg/m³)	RR and Background	Case I	Case II	Case III
15 Annual only	42.2%	49.7%	57.9%	67.3%
15 Annual/65 Daily	42.2%	49.7%	57.9%	67.3%
15 Annual/50 Daily	52.4%	60.7%	70.0%	79.2%
15 Annual/25 Daily	76.9%	83.6%	90.0%	95.4%

9. Characterization of Risk Associated with PM Pollution: Interpreting the Results of the Risk Analysis

This section discusses some issues related to the interpretation of the results of the risk analyses presented above. Several risk analyses were carried out for each of the sample locations (1) to assess the risks associated with "as is" PM levels and just attaining current PM-10 standards, and with combinations of alternative daily and annual proposed standards; (2) for several different health endpoints; (3) considering PM-10 and PM-2.5; and (4) using concentration-response functions estimated by different studies as well as pooled analysis concentration-response functions. The following points are discussed below:

(1) For any given concentration-response function, both the predicted health risk associated with "as is" PM concentrations and the predicted risk reductions associated with attaining alternative standards differ substantially between the two sample locations;

(2) At each sample location, both the predicted health risk associated with "as is" PM concentrations and the predicted risk reductions associated with attaining alternative standards are surrounded by substantial uncertainty;

(3) The PM-related health risk in one location may appear greater or smaller than the PM-related health risk in another location, depending upon how PM-related health risk is measured -- as a percent of total incidence or in absolute number of cases. (This distinction is not obvious from the particular two locations examined in this report, but could well occur in comparisons of PM-related health risks in other locations);

(4) The indicator of particulate matter (i.e., PM-10 vs. PM-2.5) may be very important in assessing PM-associated health risks;

(5) The mortality associated with annual average PM-2.5, estimated using the long-term exposure studies, is notably greater than the mortality associated with daily average PM-2.5, estimated by the short-term exposure studies; the estimate of mortality associated with long-term exposure is also more uncertain, given greater uncertainty surrounding past exposures; and

(6) The health effects incidence estimated by a risk analysis is based on the assumption that the concentration-response relationship applies for all PM concentrations considered in the analysis (e.g., down to the lowest PM level observed in the study which estimated the concentration-response function or down to background level). If the relationship between PM and a given health effect does not extend as low as the lowest level considered in an analysis, then the predictions of incidence for that health effect will be overstated. Similarly, if the PM concentrations considered in a risk analysis far exceed those observed in the study estimating the concentration-response function, it may be the case that the estimated concentration-response function is inappropriate for the very high PM concentrations considered in the risk analysis. The reader must infer the projected impact of PM on a location's public health based

on his or her judgement of the lowest concentrations for which a relationship between PM and health can plausibly be drawn and the highest concentrations for which an exponential concentration-response function is an appropriate model.

Each of the above points is discussed in turn below.

9.1. Variability of predicted health risks

There are substantial differences in the PM-related health risks estimated in Philadelphia County and in Southeast Los Angeles County, even when the same concentration-response function is used in both locations. When the measure of risk is the *incidence* of health effects associated with "as is" PM concentrations or the *incidence* of health effects associated with attaining alternative PM standards, these differences reflect, to a large extent, the substantial differences in (1) the sizes of the exposed populations in Philadelphia County (population in 1990 = 1.6 million) and Southeast Los Angeles County (population in 1990 = 3.6 million), (2) the baseline health effects incidence rates in the two locations, and (3) estimated PM levels in the two locations (as is in Philadelphia, assuming attainment of current standards in Los Angeles). When the measure of risk is in *percentage* terms (e.g., the number of cases avoided due to attaining a standard divided by the total number of cases in the absence of the standard), the difference in predicted risk based on a given concentration-response function reflects only the difference in estimated PM levels between the locations (see Section 9.3).

The differences discussed above do not include the differences that would be expected due to the fact that the concentration-response function for any given health endpoint probably varies from one location to another. The *actual* differences in PM-related risks between the sample locations could therefore be greater than the differences apparent in the risk analyses. (It is possible, but unlikely, that the concentration-response functions and PM exposure estimates would vary in such a way that the actual differences are less than the differences apparent in the risk analyses.) The uncertainty surrounding the differences in risk estimates between the two locations stems from the same sources of uncertainty surrounding risk estimations within each location, described below.

9.2. Uncertainty surrounding predicted risks

As noted in Section 2, the risk analysis requires knowledge about relationships between ambient PM concentrations and health effects, information on ambient air quality, baseline incidence rates, and population sizes. Uncertainty in estimating each of these four factors contributes to the uncertainty of predicted risks.

One of the primary quantifiable sources of uncertainty in the risk analyses is the concentration-response function. The predictions of changes in health risks associated with changes in PM concentrations (e.g., to attain an alternative standard) depend crucially on this function. For example, there are two estimates of short-term exposure mortality associated

with PM-10 above background in Southeast Los Angeles County (Exhibit 7.2), one using a pooled analysis concentration-response function and the other using a concentration-response function estimated by a study done in Los Angeles (Kinney et al., 1995). The PM-related incidence of mortality estimated by the former is twice that estimated by the latter.

There are two sources of uncertainty associated with the concentration-response function in the risk analyses. First, a concentration-response function appropriate for one location may not be appropriate for another location. As discussed in Sections 3 and 9, true underlying values of β in this function are likely to vary from one location to another. There is therefore uncertainty associated with applying concentration-response functions estimated in study locations (or functions derived by pooling these functions) to the sample locations. Second, because the concentration-response functions are empirically estimated functions, there is uncertainty surrounding these estimates. The Monte Carlo analyses presented in Section 9, and the credible intervals (presented with all estimated incidences) derived from these Monte Carlo analyses indicate a substantial degree of both kinds of uncertainty.

One reason for the uncertainty surrounding estimates of concentration-response functions is the difficulty of accounting for possible confounding factors, including weather and other pollutants. Both of these are often highly correlated with elevated PM concentrations, the first because weather conditions can keep PM in the air longer than usual (in inversions, for example), and the second because many pollution sources emit more than one pollutant. As in any regression, it is difficult to determine the separate effects of highly correlated variables. In addition, a study recently reported by the Health Effects Institute (HEI 1995) found that terms taking into account the interactions of multiple pollutants were significant predictors of health effects.

A concentration-response function could be biased if the measurement of average ambient PM concentration is inaccurate in a systematic way. Most epidemiological studies use the average PM levels reported at some number of PM monitors as the measure of the average ambient PM concentration. This may or may not yield accurate measurements of the actual daily average ambient PM concentrations in the study city. Depending on how the monitors are placed, it could yield systematically inaccurate, or biased measurements. What is important for the purpose of the risk analysis, in this case, is whether the measurement of daily average ambient PM concentrations in the sample location is biased in the same way as in the study city. That is, a systematic bias in the measurement of daily average ambient PM concentrations in the study city is not a problem for the risk analysis if there is the same systematic bias in the measurement of daily average ambient PM concentrations in the sample location. Unfortunately, whether this is the case is unlikely to be known. Uncertainty about the degree to which this is the case is another uncertainty in the risk analyses.

Finally, the prediction of health effects incidence (e.g., associated with attainment of a PM standard or with “as is” PM concentrations) depends on the accuracy of the baseline incidence data used. Obviously, multiplying the baseline incidence of some health effect by some factor will multiply the predicted PM health effects by the same factor. Because county-

specific rates for mortality and hospital admissions are available from public health agencies for both Philadelphia County and Los Angeles County, these estimates are considered quite reliable. In the absence of city-specific rates for incidence of some respiratory symptoms, the risk analyses used rates from the epidemiological studies. This introduced another component of uncertainty into the risk analyses.

9.3. Importance of the measure of risk

A PM-related health effect risk may be characterized in terms of the *percent* of the total incidence of the health effect that is associated with PM concentrations above a certain level or the actual *number* of cases (i.e., the incidence) of the health effect associated with PM concentrations above a certain level. Both measures are presented in this report. The measure of risk used may affect the assessment of the degree of risk in one location relative to another. Suppose, for example, that location X has much higher PM levels than location Y. If PM-related mortality risk is measured in terms of the percent of total mortality associated with PM, location X will appear to have greater PM-related mortality risk than location Y. However, if location Y's population is much larger than location X's, it is quite possible that a greater *incidence* of PM-related mortality will be predicted in location Y than location X. (This possibility was not apparent in the results from Philadelphia County and Southeast Los Angeles County because both the PM levels and the population of the latter exceed that of the former.)

Unlike the incidence of PM-related mortality, the percent of total mortality that is PM-related is affected by neither the size of the exposed population nor the baseline health effect incidence rate. Given a concentration-response function (i.e., a β), this measure of risk is affected only by the actual PM concentrations (relative to the alternative being considered). For an *individual* considering the PM-related risks to himself in one location versus another, the percent of the total incidence of the health effect is the appropriate measure of risk. It yields an ordering of locations by their PM-related risk that is the same as the ordering achieved by simply measuring the PM concentrations in the different locations. PM levels were higher in Southeast Los Angeles County than in Philadelphia County, so for any given individual the PM-related health risks are correspondingly higher in Southeast Los Angeles County than in Philadelphia County. (Compare, for example, the "percent of total incidence" columns for Philadelphia County, in Exhibit 7.1, with the corresponding columns for Southeast Los Angeles County, in Exhibit 7.2.)

To measure risks to *society*, however, the number of affected individuals is important. Even if the risk per individual is higher in location A than location B, if many more individuals are exposed in location B, the total risk to society may be greater in location B. The actual number of PM-related cases and the percent of all cases that are PM-related are both appropriate measures of risk that deal with different questions.

9.4. Importance of the indicator of PM: PM-10 vs. PM-2.5

One important reason that the concentration-response relationship between PM-10 and a given health endpoint may vary from one location to another is that the composition of PM-10 varies significantly throughout the United States. In some areas, particulate matter is composed mostly of coarse particles; in other areas there is a much larger fine particle fraction. In Philadelphia County, for example, PM-2.5 comprises about 73 percent of PM-10 (see Exhibits 4.7 and 4.8), whereas in Southeast Los Angeles County, PM-2.5 comprises only about 59 percent of PM-10 (Exhibits 4.12 and 4.13). If all particle sizes are equally harmful in causing a health effect, then this type of variability in PM-10 composition will not matter. If different size particles are differentially harmful, however, a given concentration of PM-10 in one location may have a different impact on health than the same concentration of PM-10 in another location. (The chemical composition of the PM may be important as well. For example, there has been some investigation of the health impacts of sulfates, a common chemical component of PM pollution. However, particle size is one important factor and the one for which the most complete data are available.)

Suppose, for example, that only the fine fraction (PM-2.5) adversely affects human health and the rest of PM-10 has no adverse effect at all. Suppose also that in location A, only fifty percent of PM-10 is PM-2.5 whereas in location B 90 percent of PM-10 is PM-2.5. In this case, 1 $\mu g/m^3$ of PM-10 in location A translates into 0.5 $\mu g/m^3$ of PM-2.5, whereas in location B 1 $\mu g/m^3$ of PM-10 translates into 0.9 $\mu g/m^3$ of PM-2.5. What appear to be equal exposures in the two locations when PM-10 is used as the indicator of particulate matter pollution exposure, are actually substantially different exposures to the only component that actually affects health.

There are, then, two important issues for a risk analysis concerning the choice of indicator of particulate matter pollution:

(1) different components of PM-10 may affect a health endpoint to different degrees (i.e., may be differentially "potent"), and
(2) the ratios of these different components within PM-10 may change over time or from place to place, changing the health effects associated with particulate matter pollution.

If all particle sizes are equally potent in causing a given health effect, then the relationship between PM-10 and that health effect is adequately described by the basic model introduced in equation (1) in Section 2. If, however, PM-2.5 and the coarse fraction of PM-10 are differentially potent, then that model may not adequately describe the relationship between PM-10 and the health effect, because a given concentration of PM-10 may be associated with different levels of health effect depending on the composition of the PM-10.

A generalization of the basic exponential concentration-response relationship between a health effect and PM, either PM-10 or PM-2.5 presented in Section 2 (equation (1) is presented

in Appendix 4. In this generalization, health effect incidence is a function of both PM-2.5 and the coarse fraction of PM-10. The basic model (equation (1)) is then seen to be the special case in which PM-2.5 and the coarse fraction are equally potent. It is also shown that if PM-2.5 and the coarse fraction are not equally potent, then the relationship between PM-10 and a given health endpoint will depend both on the relative potencies of the two fractions and on the ratio of PM-2.5 to PM-10. If this ratio varies from one location to another, then the PM-10 concentration-response function will vary as well, even if the exposed populations are identical.

In the absence of such information, risk analyses considering PM-10 use only PM-10 concentration-response functions (acknowledging the uncertainty introduced by the possibility that the composition of the PM-10 in the study location may differ from the composition of the PM-10 in the sample locations in which the function is applied). Risk analyses considering only PM-2.5 use PM-2.5 concentration-response functions when they are available. In a few cases (e.g., for ischemic heart disease and congestive heart failure), PM-2.5 data are used with PM-10 functions (in the absence of PM-2.5 functions). The health effects incidence predictions from such analyses are unlikely to overestimate the PM-2.5-related health risks.

9.5. Risk predictions based on concentration-response functions from long-term exposure studies versus those from short-term exposure studies

The long-term exposure study (Pope et al., 1995) estimates the apparent effect of PM on mortality to be much greater than do short-term exposure studies. This suggests that the effects of long-term exposure may not merely be the sum of the effects of short-term exposures over the course of a year. It is possible that the long-term exposure study is detecting mortality related to long-term PM exposure, in addition to the mortality precipitated by short-term PM exposure. It is not unreasonable to suspect that prolonged exposure to elevated PM levels, as well as exposure to short-term peak PM levels, might cause health problems. In addition, the effects of the two types of exposure might be related.

While long-term exposure studies use long-term average PM concentration (which can be approximated by annual average PM concentration) as the PM indicator, these studies are generally conducted in such a way that they may be detecting effects due to PM exposure over some longer period. For example, average PM concentrations over the course of five years might be the appropriate measure. Such possible discrepancies between the actual relevant exposure period (e.g., five years) and the exposure period considered by a long-term exposure study (e.g., one year) could have at least two effects.

It is possible that the full benefits of reducing PM predicted by such studies would not appear in the first year after reductions to attain a standard, but would be “phased in” gradually as concentrations during successive years were also reduced. If average PM concentrations over five years is the appropriate measure, for example, the benefits of a standard would gradually increase to their full level over the course of the five years after the new standard had been attained. The risk analysis does not attempt to determine the appropriate exposure period

for the long-term exposure study on mortality. The estimated annual benefits of reduced long-term exposure are assumed to be completely achieved by the future year for which attainment of the new standard is being modeled.

It is also possible that the predicted incidence of mortality that is associated with an annual average PM level could be either an over- or underestimation of risk. This could be the case if the actual relevant exposure period is substantially different from one year, and the average PM levels over the relevant exposure period are substantially different from the annual average PM levels used in a long-term exposure study.

Finally, the prematurity of deaths associated with PM may be of crucial importance, whether the short-term exposure or the long-term exposure study is used. PM-related deaths that are days or even weeks earlier than they would be at lower PM levels may cause less public-health concern than PM-related deaths of individuals who could otherwise expect to live many more years. Even finding that most of the people who die during PM episodes are seriously ill would not resolve the question, since without the additional stress of high PM concentrations, some of those people might fully recover and live significantly longer. This issue so far has not been resolved in the epidemiological literature.

As indicated in the CD (EPA, 1996a) and the Staff Paper (EPA, 1996b), the public health burden of ambient PM-mediated mortality depends on both the number of deaths and the shortening of life that PM exposure causes or promotes (CD, p. 13-44). Risk analysis estimates of percentage incidence (and incidence counts) of mortality associated with PM could vary substantially depending on the general prematurity of death involved. For instance, if prematurity of death associated with short-term exposures to PM generally was only on the order of days or weeks, then ambient PM concentrations in the two risk analysis locations would be expected to have less of an impact on annual mortality incidence than indicated by the base case analysis.[18] In this case, PM would be temporally associated with the proportion of annual mortality events reported in the risk analysis, but PM would be associated with a lower proportion of the overall mortality rate. This would result because many of the events that PM would be associated with, if the assumption of little prematurity of death in general is accurate, would have occurred in the absence of PM involvement days or weeks later. Thus the absence or reduced concentrations of PM would not affect mortality rates to the same extent to which PM was temporally associated with mortality events.

For this reason, the alternate health measure of Life Years Lost is often employed to measure the public health burden of environmental factors, rather than simply estimates of mortality incidence. As the CD indicates, however, confident quantitative determination of years of life lost to ambient PM exposure is not yet possible (CD p. 13-44). A couple of studies of mortality from short-term exposures suggest that some portion of PM-induced mortality may occur among individuals already so ill that they would soon die without PM

[18]Such a pattern of apparent mortality displacement of only a few days is often seen for some other environmental effects, such as high temperature (Staff Paper, V-17).

exposure (Spix et al. 1993; Cifuentes and Lave 1996), while other studies (Dockery et al. 1993; Pope et al. 1995) report associations between PM and changes in mortality rates, findings that cannot be solely explained by death-bed effects (i.e., mortality with little prematurity) (Utell and Frampton 1995). A sustained reduction in particle levels in Utah Valley over 14 months was also accompanied by a drop in mortality rates, consistent with the hypothesis that a substantial portion of PM-associated mortality may involve mortality of sufficient prematurity to affect mortality rates.

Incomplete knowledge of the true excess mortality and prematurity of death associated with PM exposures complicates this risk analysis and adds uncertainty to the interpretation of the mortality risk estimates. This difficulty would be expected to most greatly complicate interpretation of the estimates of mortality associated with short-term exposures compared to the estimates of mortality associated with long-term exposures.

9.6. Dependence of results on the assumption that the concentration-response relationship is applicable at low concentrations

The change in health effects incidence predicted by a risk analysis to be associated with reducing "as is" PM levels either to background or to concentrations that meet alternative standards is based on the assumption that the concentration-response relationship applies down to the lowest concentration considered in the analysis. If the relationship between PM and a given health effect does not extend to the lowest PM levels under consideration, then the predictions of health effects incidence will be biased. If, for example, there is a level above the lowest PM level considered below which there are no health effects, then the change in health effects incidence will be overstated. The degree of overstatement will depend on the discrepancy between the lowest level considered and the lowest PM level at which there are PM-related health effects. In the absence of knowledge concerning the lowest level at which effects occur, the reader must infer the projected impact of particulate matter on a location's public health based on his or her judgement of the lowest concentrations for which the given relationship between PM and health can plausibly be drawn and the plausible shape of the concentration-response function below that level. This issue has been partially explored in a series of sensitivity and uncertainty analyses presented in Section 7.

Appendix 1: The Relationship Between the Ambient Concentration-Response Function and the Individual Exposure-Response Function

1. Individual exposure versus ambient concentration and the individual exposure-response relationship versus the ambient concentration-response relationship

There has been persistent concern that epidemiological studies which use ambient PM concentration data as a surrogate for individual exposure to PM may produce biased estimates of individual exposure-response functions relating health effects to individual exposure. This is a valid concern. If such functions were used with individual exposure data from the study location, the predicted health responses could be biased, as discussed below. If such functions are used with ambient PM concentration data from the study location, however, there is no reason to suspect that the predicted health responses would be biased (unless there are other sources of bias). The relationship estimated in the studies is, after all, between the population health response and average ambient PM concentration.

To help clarify some potential confusion, two relationships are distinguished. The relationship between a health response and individual exposure to PM is referred to as an individual exposure-response relationship. On an individual level, this is the relationship between the actual exposure to PM (in $\mu g/m^3$) experienced by the individual and the probability that that individual will exhibit the health response. On an aggregate level, it is the relationship between the average exposure to PM (in $\mu g/m^3$) by individuals in the population and the population response (number of individuals exhibiting the health response).[19]

The relationship between a health response and ambient PM concentration is referred to as the ambient concentration-response relationship. It is the relationship between the average ambient concentration of PM (in $\mu g/m^3$) and the population response.[20] Both the individual exposure-response relationship and the ambient concentration-response relationship are of interest. The individual exposure-response relationship is of clear scientific interest. This is the relationship that epidemiological studies would presumably estimate if they had data on individual exposure. Because the NAAQS influence ambient concentrations of PM, it is the ambient concentration-response relationship that is of interest for this risk analysis, because the risk analysis examines the risk reduction associated with changing ambient concentrations, rather than the risk reduction associated with changing individual exposure (which is not directly controlled by the NAAQS).

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[19]Each individual has an average exposure to PM over time (e.g., a daily average). The average individual exposure to PM is the average, over all individuals, of these time-averages. Because there are factors other than average ambient PM concentration that affect individual exposure (as described in the model discussed below), average individual exposure to PM does not necessarily equal average ambient PM concentration.

[20]The average ambient PM concentration is an average over both time and space (e.g., the average over a given geographic area of 24-hour averages).

The two relationships (the individual exposure-response relationship and the ambient concentration-response relationship) are related by the connection between individual exposure and ambient concentration, as detailed in the model below. Let

Z = average individual exposure to PM (in $\mu g/m^3$),
X = average ambient concentration of PM (in $\mu g/m^3$),
Y = the health response (e.g., mortality),
Q = a vector of variables, other than ambient concentration, that affect individual exposure, (e.g., average percent of time spent indoors), and
T = a vector of variables, other than individual exposure to PM, that affect the health response.

For ease of illustration, certain functional forms are assumed. The relationship between individual exposure and ambient concentration is assumed to be linear. The relationship between the health response and individual exposure to PM is assumed to be log-linear. In addition, it is also assumed, for ease of discussion, that Q and T are each single variables.

The equations that follow are, as noted above, intended only to provide an example to illustrate the relationship between average individual exposure, average ambient concentration, and population health response. While deviations from any of the assumptions stated in this example may alter the particular functional forms of the equations presented below, they will not affect the basic ideas discussed in this appendix.

The relationship between average individual exposure to PM (Z) and average ambient concentration of PM (X) is given by

$$Z = \alpha_o + \beta_o X + \theta_o Q \qquad \textbf{(16)}$$

and the relationship between the population health response (Y) and average individual exposure (Z), the individual exposure-response relationship, is given by

$$\ln Y = \delta + \lambda Z + \gamma T \quad . \qquad \textbf{(17)}$$

Substituting equation (1) into equation (2) yields the ambient concentration-response relationship:

$$\ln Y = \delta + \lambda(\alpha_o + \beta_o X + \theta_o Q) + \gamma T \qquad \textbf{(18)}$$

$$= \alpha + \beta X + \theta Q + \gamma T \quad , \qquad \textbf{(19)}$$

where

$$\alpha = \delta + \lambda\alpha_o \ , \quad \beta = \lambda\beta_o \ , \quad \textit{and,} \quad \theta = \lambda\theta_o \ . \qquad \textbf{(20)}$$

It is of scientific interest to estimate an individual exposure-response relationship, equation (2). Lacking data on individual exposure, Z, however, epidemiological studies use ambient concentration, X, as a surrogate for individual exposure, Z. Such studies estimate equation (4), then, instead of equation (2). If the estimate of β is an unbiased estimate of β, then it can also be an unbiased estimate of λ, the coefficient of individual exposure in the individual exposure-response function, if and only if $\beta_o = 1$. That is,

$$E(\hat{\beta}) = \beta = \lambda\beta_o = \lambda \textit{ if and only if } \beta_o = 1 \ .$$

Therefore the source of the bias in estimating the coefficient of individual exposure, λ, does not exist for estimating the coefficient of ambient concentration, β. This is precisely because it is the ambient concentration-response relationship, rather than the individual exposure-response relationship that is being estimated in the epidemiological studies. Whereas this may present a problem for the epidemiological studies that seek to estimate an individual exposure-response relationship, it should not present a problem for a risk assessment, for which it is the ambient concentration-response relationship that is of interest.

2. Other possible sources of bias in the estimate of the ambient concentration-response relationship and mitigating influences

Although epidemiological studies usually include in their models those variables that are likely to affect the health response of interest (variables in the vector T, such as temperature and time trends), they do not necessarily include those variables that may affect individual exposure (variables in the vector Q, such as the average percent of time spent indoors). While the actual ambient concentration-response relationship is equation (4), the model estimated is more likely to be

$$\ln Y = \alpha + \beta X + \gamma T \ . \qquad \textbf{(22)}$$

That is, the model estimated may have omitted variables. This raises the possibility that omitted variables could cause bias in the estimates of coefficients in the model. Those variables that are omitted, however, are likely to be highly correlated with the variables in the vector T -- in particular, with temperature. (For example, the percent of time spent indoors should be correlated with temperature.) While omission of these variables may cause bias in the coefficient of temperature, then, it is unlikely to cause bias in the coefficient of X. In fact, the more highly correlated an omitted variable is with a variable in T, e.g., temperature, the less of a bias problem there is in estimating the coefficient of ambient concentration, β.

Including variables such as temperature in the model, then, would tend to mitigate any bias problem.

3. Transferability of a concentration-response relationship estimated in one location to another location

It is argued in sections 1 and 2 above that, while using ambient concentration as a proxy for individual exposure may produce a biased estimate of the individual exposure-response relationship in the study location, it should produce an unbiased estimate of the ambient concentration-response relationship in that location. Applying ambient concentration data from the study location, then, should produce unbiased predictions of health response in that location (barring any other possible sources of bias).

If ambient concentration data from a *different* location are applied to the estimated ambient concentration-response function from the study location, will the predicted health response in that different location be biased? That depends on whether the ambient concentration-response relationship in the study location is the same as that in the location to which it is being applied. This is the issue of transferability.

Recall that the ambient concentration-response relationship estimated is

$$\ln Y = \alpha + \beta X + \gamma T \quad .$$

where $\beta = \lambda\beta_o$. If either λ or β_o differs between one location and another, then β will differ, and the ambient concentration-response relationship will be different as well. β_o might differ among locations if, for example, coarse particles are less likely to infiltrate indoor air. In this case, PM-10 with a high proportion of coarse particles would result in lower indoor exposure than PM-10 with a high proportion of fine particles. If the percent of time spent indoors is the same, then the location with the coarser PM-10 would have lower individual exposure to PM-10 than the location with the finer PM-10. λ might differ among locations if, for example, the population in one location has a higher proportion of a susceptible subgroup than another location. Another reason λ might differ among locations is if the composition of the PM among locations differs and the composition of the PM affects its toxicity (see Appendix 4).

While applying an ambient concentration-response function estimated in one location to another location may give biased health response predictions, the direction or magnitude of the bias is not known, and will depend on the particular pair of locations. This issue is less one of bias and more one of uncertainty and is addressed in Section 9 of the report.

Appendix 2: Pooling the Results of Different Studies

Many studies have attempted to determine the influence of particulate matter pollution on human health. Usually this involves estimation of a parameter β in a concentration-response function, which may be linear or non-linear, as discussed above. Each study provides an estimate of β, along with a measure of the uncertainty of the estimate. Because uncertainty decreases as sample size increases, combining data sets is expected to yield more reliable estimates of β. Combining data from several comparable studies in order to analyze them together is often referred to as meta-analysis.

For a number of reasons, including data confidentiality, it is often impractical or impossible to combine the original data sets. Combining the *results* of studies in order to produce better estimates of β provides a second-best but still valuable way to synthesize information (DerSimonian and Laird, 1986). This is referred to as pooling results in this report. Pooling requires that all of the studies contributing estimates of β use the same functional form for the concentration-response function. That is, the β's must be measuring the same thing.

One method of pooling study results is simply averaging all reported β's. This has the advantage of simplicity, but the disadvantage of not taking into account the uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty. For example, consider the three studies whose results are presented in Exhibit A2.1.

Exhibit A2.1. Three Sample Studies.

Study	Estimate of β	Standard Deviation	Variance
Study 1	0.75	0.35	0.1225
Study 2	1.25	0.05	0.0025
Study 3	1.00	0.10	0.0100

The average of the three estimates is 1.0. However, the study 2 estimate has much less uncertainty associated with it (variance = 0.0025) than either the study 1 or study 3 estimates. It seems reasonable that a pooled estimate which combines the estimates from all three studies should therefore give more weight to the estimate from the second study than to the estimates from the first and third studies. A common method for weighting estimates involves using their variances. Variance takes into account both the consistency of data and the sample size used to obtain the estimate, two key factors that influence the reliability of results.

The exact way in which variances are used to weight the estimates from different studies in a pooled estimate depends on the underlying model assumed. The next Section

discusses the two basic models that might underlie a pooling and the weighting scheme derived from each.

A2.1 The fixed effects model

The fixed effects model assumes that there is a single true concentration-response relationship and therefore a single true value for the parameter β. Differences among β's reported by different studies are therefore simply the result of sampling error. That is, each reported β is an estimate of the *same underlying parameter*. The certainty of an estimate is reflected in its variance (the larger the variance, the less certain the estimate). Pooling that assumes a fixed effects model therefore weights each estimate under consideration in proportion to the *inverse* of its variance.

Suppose there are n studies, with the ith study providing an estimate β_i with variance v_i (I = 1, ..., n). Let

$$S = \Sigma \frac{1}{v_i} ,$$

denote the sum of the inverse variances. Then the weight, w_i , given to the ith estimate, β_i , is

$$w_i = \frac{1/v_i}{S} .$$

This means that estimates with small variances (i.e., estimates with relatively little uncertainty surrounding them) receive large weights, and those with large variances receive small weights.

The estimate produced by pooling based on a fixed effects model, then, is just a weighted average of the estimates from the studies being considered, with the weights as defined above. That is,

$$\beta_{fe} = \Sigma \, w_i * \beta_i .$$

The variance associated with this pooled estimate is the inverse of the sum of the inverse variances:

$$v_{fe} = \frac{1}{\Sigma \, 1/v_i} .$$

Exhibit A2.2 shows the relevant calculations for this pooling for the three sample studies summarized in Exhibit A2.1.

Exhibit A2.2. Fixed Effect Model Calculations.

Study	β_i	v_i	$1/v_i$	w_i	$w_i*\beta_i$
1	0.75	0.1225	8.16	0.016	0.012
2	1.25	0.0025	400	0.787	0.984
3	1.00	0.0100	100	0.197	0.197
Sum			$\sum$ = 508.16	$\sum$ = 1.000	$\sum$ = 1.193

The sum of weighted contributions in the last column is the pooled estimate of β based on the fixed effects model. This estimate (1.193) is considerably closer to the estimate from study 2 (1.25) than is the estimate (1.0) that simply averages the study estimates. This reflects the fact that the estimate from study 2 has a much smaller variance than the estimates from the other two studies and is therefore more heavily weighted in the pooling.

The variance of the pooled estimate, v_{fe}, is the inverse of the sum of the variances, or 0.00197. (The sums of the β_i and v_i are not shown, since they are of no importance. The sum of the $1/v_i$ is S, used to calculate the weights. The sum of the weights, w_i , I=1, ..., n, is 1.0, as expected.)

A2.2 The random effects model

An alternative to the fixed effects model is the random effects model, which allows the possibility that the estimates β_i from the different studies may in fact be estimates of *different* parameters, rather than just different estimates of a single underlying parameter. In studies of the effects of PM-10 on mortality, for example, if the composition of PM-10 varies among study locations the underlying relationship between mortality and PM-10 may be different from one study location to another. For example, fine particles make up a greater fraction of PM-10 in Philadelphia than in El Paso. If fine particles are disproportionately responsible for mortality relative to coarse particles, then one would expect the true value of β in Philadelphia to be greater than the true value of β in El Paso. This would violate the assumption of the fixed effects model.

The following procedure can test whether it is appropriate to base the pooling on the random effects model (vs. the fixed effects model):

A test statistic, Q_w , the weighted sum of squared differences of the separate study estimates from the pooled estimate based on the fixed effects model, is calculated as:

$$Q_w = \Sigma_i \frac{1}{v_i} (\beta_{fe} - \beta_i)^2.$$

Under the null hypothesis that there is a single underlying parameter, β, of which all the β_i 's are estimates, Q_w has a chi-squared distribution with n-1 degrees of freedom. (Recall that n is the number of studies in the meta-analysis.) If Q_w is greater than the critical value corresponding to the desired confidence level, the null hypothesis is rejected. That is, in this case the evidence does not support the fixed effects model, and the random effects model is assumed, allowing the possibility that each study is estimating a different β.

The weights used in a pooling based on the random effects model must take into account not only the within-study variances (used in a meta-analysis based on the fixed effects model) but the between-study variance as well. These weights are calculated as follows:

Using Q_w , the between-study variance, η^2, is:

$$\eta^2 = \frac{Q_w - (n-1)}{\Sigma 1/v_i - \frac{\Sigma 1/v_i^2}{\Sigma 1/v_i}} .$$

It can be shown that the denominator is always positive. Therefore, if the numerator is negative (i.e., if $Q_w < n-1$), then η^2 is a negative number, and it is not possible to calculate a random effects estimate. In this case, however, the small value of Q_w would presumably have led to accepting the null hypothesis described above, and the meta-analysis would be based on the fixed effects model. The remaining discussion therefore assumes that η^2 is positive.

Given a value for η^2 , the random effects estimate is calculated in almost the same way as the fixed effects estimate. However, the weights now incorporate both the within-study variance (v_i) and the between-study variance (η^2). Whereas the weights implied by the fixed effects model used only v_i, the within-study variance, the weights implied by the random effects model use $v_i + \eta^2$.

Let $v_i^* = v_i + \eta^2$. Then

$$S^* = \Sigma \frac{1}{v_i^*} ,$$

and

$$w_i^* = \frac{1/v_i^*}{S^*} \quad .$$

The estimate produced by pooling based on the random effects model, then, is just a weighted average of the estimates from the studies being considered, with the weights as defined above. That is,

$$\beta_{rand} = \Sigma \; w_i^* * \beta_i \; .$$

The variance associated with this random effects pooled estimate is, as it was for the fixed effects pooled estimate, the inverse of the sum of the inverse variances:

$$v_{rand} = \frac{1}{\Sigma \; 1/v_i^*} \; .$$

The weighting scheme used in a pooling based on the random effects model is basically the same as that used if a fixed effects model is assumed, but the variances used in the calculations are different. This is because a fixed effects model assumes that the variability among the estimates from different studies is due only to sampling error (i.e., each study is thought of as representing just another sample from the same underlying population), while the random effects model assumes that there is not only sampling error associated with each study, but that there is also *between-study* variability -- each study is estimating a different underlying β. Therefore, the sum of the within-study variance and the between-study variance yields an overall variance estimate.

A2.3 An example

This Section demonstrates the relevant calculations for pooling using the example in Exhibit A2.1 above.

First calculate Q_w , as shown in Exhibit A2.3.

Exhibit A2.3: Calculation of Q_w

Study	β_i	$1/v_i$	$1/v_i * (\beta_i - \beta_{fe})^2$
1	0.75	8.16	1.601
2	1.25	400	1.300
3	1.00	100	3.725
			$\sum = Q_w = 6.626$

In this example the test statistic $Q_w = 6.626$. The example considers three studies, so Q_w is distributed as a chi-square on two degrees of freedom. The critical value for the 5 percent level (i.e., corresponding to a 95 percent level of confidence) for a chi-square random variable on 2 degrees of freedom is 5.99. Because $Q_w = 6.626 > 5.99$, hence the null hypothesis is rejected. That is, the evidence does not support the fixed effects model. Therefore assume the random effects model is appropriate.

Then calculate the between-study variance:

$$\eta^2 = \frac{6.626 - (3 - 1)}{508.16 - \frac{170066.65}{508.16}} = 0.0267 \quad .$$

From this and the within-study variances, calculate the pooled estimate based on the random effects model, as shown in Exhibit A2.4.

Exhibit A2.4. Random Effects Model Calculations.

Study	β_i	$v_i + \eta^2$	$1/(v_i + \eta^2)$	w_i^*	$w_i^* \times \beta_i$
1	0.75	0.1492	6.70	0.098	0.0735
2	1.25	0.0292	34.25	0.502	0.6275
3	1.00	0.0367	27.25	0.400	0.400
Sum			$\sum = 68.20$	$\sum = 1.000$	$\sum = 1.101$

The random effects pooled estimate, β_{rand}, is 1.101. It's variance, v_{rand}, is 1/(68.2) = 0.015.

Appendix 3: The Concentration-Response Function and Relative Risk

The basic "Poisson regression" concentration-response relationship commonly found in the epidemiological literature is

$$y = B\, e^{\beta x} \quad , \tag{35}$$

where x is the PM level, y is the incidence of the health endpoint of interest at PM level x, β is the coefficient of PM, and B is the incidence at x=0, i.e., when there is no particulate matter. (Either incidence or incidence rate may be used as long as y and B are consistent.)

If x denotes the actual ("as is") PM level and y denotes the baseline incidence (rate) of the health endpoint, i.e., the incidence (rate) corresponding to the "as is" PM level, letting x_0 denote some specified alternative PM level and y_0 denote the incidence (rate) associated with that alternative PM level, then

$$y_0 = B\, e^{\beta x_0} \quad . \tag{36}$$

The *change* in health effects incidence, $\Delta y = y_0 - y$, corresponding to a given change in PM levels, $\Delta x = x_0 - x$, can be derived from equations (1) and (2)[21] as follows:

First, dividing equation (2) by equation (1) yields

$$\frac{y_0}{y} = \frac{e^{\beta x_0}}{e^{\beta x}} = e^{\beta(x_0 - x)} = e^{\beta \Delta x} \quad . \tag{37}$$

Then multiplying through by y yields

$$y_0 = y e^{\beta \Delta x} \quad . \tag{38}$$

Subtracting y from both sides gives

$$\Delta y = y_0 - y = y e^{\beta \Delta x} - y = y[e^{\beta \Delta x} - 1] \quad , \tag{39}$$

or

[21]Because the Poisson regression form of concentration-response function (equation (1)) is by far the most common form, the discussion that follows assumes that form.

$$\Delta y = y[e^{\beta \Delta x} - 1] \ . \qquad \textbf{(40)}$$

Alternatively, the change in health effects incidence can be calculated using relative risk. Relative risk (RR) is a well known measure of the comparative health effects associated with a particular exposure comparison. The risk of mortality at PM level x_0 relative to the risk of mortality at PM level x, for example, may be characterized by the ratio of the two mortality rates: the mortality rate among individuals exposed to PM level x_0, i.e., y_0, and the mortality rate among (otherwise identical) individuals exposed to PM level x, i.e., y. This is the left-hand side of equation (3). That is,

$$RR_{\Delta x} = \frac{y_0}{y} = \frac{e^{\beta x_0}}{e^{\beta x}} = e^{\beta(x_0 - x)} = e^{\beta \Delta x} \ , \qquad \textbf{(41)}$$

or

$$RR_{\Delta x} = e^{\beta \Delta x} \ . \qquad \textbf{(42)}$$

Given a concentration-response function (i.e., a particular value for the coefficient, β), then, and a particular change in PM levels, Δx, the relative risk associated with that change in PM, denoted as $RR_{\Delta x}$, can be calculated from equation (8). This is particularly significant, because it means that the relative risk corresponding to any change in PM levels is easily calculated. In particular, using equation (8), it is straightforward to convert a relative risk corresponding to one Δx into a relative risk corresponding to a different Δx. Suppose, for example, that a relative risk from a study reflects the relative mortality risks associated with the two PM levels, x_0 and x_1. Then from equation (8),

$$RR_{(x_0 - x_1)} = e^{\beta(x_0 - x_1)} \ . \qquad \textbf{(43)}$$

Solving for β yields

$$\beta = \frac{\log RR_{(x_0 - x_1)}}{(x_0 - x_1)} \ .$$

Now the relative risk corresponding to $\Delta x=(x_1 - x_2)$ can be calculated, using β and equation (8) again as

$$RR_{(x_1 - x_2)} = e^{\beta(x_1 - x_2)} \ . \qquad \textbf{(45)}$$

Substituting equation (8) into equation (6), it becomes clear that the change in health effects incidence, Δy, corresponding to a given change in PM levels, Δx, can be calculated based on the relative risk corresponding to Δx as:

$$\Delta y = y(RR_{\Delta x} - 1) \; . \qquad \textbf{(46)}$$

Equations (6) and (12) are simply alternative ways of writing the relationship between a given change in PM levels, Δx, and the corresponding change in health effects incidence, Δy.

Note that, in the above, the baseline health effect incidence (rate), y, refers to the incidence (rate) corresponding to the "as is" PM level. This is because the baseline incidences used in the calculations for the risk analysis are drawn from available health statistics which *include* the effects of exposure to air pollution. Changes in incidence, Δy, correspond to reductions in PM concentrations. Because Δx is negative (a reduction in PM concentration), $RR_{\Delta x}$ will be less than 1, and Δy will also be negative -- that is, the number of cases of the health effect *avoided*.

If the general population is not typically exposed to the risk factor of interest, however, then the baseline incidence (rate) would be the incidence in the *absence* of exposure to the risk factor under consideration. In this case, the relative risk associated with exposure to the risk factor would be positive -- the increase in cases due to exposure, as opposed to the baseline incidence, in the absence of exposure. This is a common situation, an example of which is provided by Samet and Spengler, 1993.

The formula for "population attributable risk" given by Samet and Spengler, 1993

$$\frac{B\ (RR - 1)}{B\ RR} \quad ;$$

where RR is the relative risk associated with an increase in the risk factor (i.e., Δx is positive here), and B is the baseline incidence -- now, the incidence of the health effect *in the absence of exposure to the risk factor under consideration*. That is, the formula computes the increase in incidence divided by total incidence.

Assume now that the risk factor in both cases (i.e., in the risk analysis reported here and in Samet and Spengler) is PM. Then a relationship exists between the baseline incidence, B, defined by Samet and Spengler as the incidence in the *absence* of exposure to the risk factor, and the baseline incidence used in the calculations for this risk analysis, y, defined as the incidence associated with the "as is" level of the risk factor (PM). In particular,y = B * RR. That is, the incidence of the health effect associated with "as is" PM levels is the incidence of the health effect in the absence of PM (or at background PM level) times the relative risk associated with the change in PM levels

Substituting B = y/RR from above in the Samet and Spengler formula yields

$$\frac{y - y\,(1/RR)}{y} \quad ,$$

or, now cancelling y,

$$1 - (1/RR) \quad ,$$

which is the formula used in the calculations in the risk analysis. If the relative risk RR is associated with an increase in pollution, Δx, then a decrease in pollution, $-\Delta x$, is associated with a relative risk 1/RR. Therefore, the formula used in these calculations computes the percent change in incidence associated with a decrease in pollution from some previously existing level (in this analysis, from observed concentrations to background or cutpoint concentrations). The percent is calculated with respect to the incidence at the existing level, that is, from the full incidence in the population.

As an example, assume that RR = 1.2. Then Samet and Spengler's formula gives (1.2 - 1)/(1.2) = .167, that is, 16.7% of incidence attributable to the risk factor. Our formula is 1- (1/1.2) = 1- .833 = .167, the same result given by Samet and Spengler.
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Appendix 4: A Generalization of the Basic Concentration-Response Function

This appendix presents a model which generalizes the basic concentration-response model to explicitly incorporate two important considerations: (1) the fine and coarse fractions of PM-10 may affect a health endpoint in very different ways, and (2) the ratio of fine to coarse particles in PM-10 can change over time or from place to place, changing the health effects associated with particulate matter pollution.

Assume that fine and coarse particles cause health effects independently of one another. Assume further that the Poisson regression model used in most epidemiological studies is an appropriate model. The model postulates that the effects of a pollutant are *multiplicative*, that is, that an increase in pollution implies some percent increase in health effects. Therefore, if B is the hypothetical "base incidence" when particulate concentration is zero, then health effects are estimated using the equation

$$y = B * e^{\beta_f * x_f} * e^{\beta_c * x_c}, \quad \textbf{(50)}$$

where x_f = the amount of fine particles (PM-2.5) (in micrograms per cubic meter),
x_c = the amount of coarse particles (PM-10 minus PM-2.5),
β_f = the beta regression coefficient measuring the effect of fine particles,
β_c = the beta regression coefficient measuring the effect of coarse particles,
B = the (hypothetical) base incidence rate when no particulate matter is present, that is, when $x_f = x_c = 0$, and
y = the health effect (mortality is used for purposes of discussion below).

Similarly, let

$x_{10} = x_f + x_c$ = the total amount of PM-10, and
β_{10} = the beta regression coefficient measuring the effects of PM-10.

Note that if $\beta_f = \beta_c$ (i.e., if there is no difference in potency between the fine and the coarse fractions), then the generalized model (1) reduces to the basic model,

$$y = B * e^{\beta_{10}} \quad . \quad \textbf{(51)}$$

Because multiplication is commutative, and because exp[a]*exp[b] = exp[a+b], it does not matter in what order exposure to additional pollutants is thought of as taking place. (Note that one could assume a base rate B' at some non-zero pollutant combination, x_f^0 and x_c^0; the equation would remain the same under the change of variables $x_f' = x_f - x_f^0$ and $x_c' = x_c - x_c^0$. This would make it unnecessary to model health effects at pollution levels below the range of data. None of the following discussion would be changed; the equation as written is retained for convenience.)

Define

$$q = \frac{x_f}{x_f + x_c} = \frac{x_f}{x_{10}} \quad \textbf{(52)}$$

and

$$r = \frac{\beta_c}{\beta_f} \quad . \quad \textbf{(53)}$$

The parameter q is just the ratio of PM-2.5 to PM-10. The parameter r may be thought of as the relative "potency" of coarse and fine particles in causing mortality (or, equivalently, as a measure of their relative toxicities). If coarse and fine particles are equally potent in causing mortality (i.e., if particle size doesn't matter), then r=1. If only fine particles matter, in which case $\beta_c = 0$, then r=0. Assuming that fine particles cause at least their share of the mortality associated with PM-10, r will lie somewhere between 0 and 1.

Equation (2) implies

$$x_f = q * x_{10} \quad \textbf{(54)}$$

and therefore also

$$x_c = (1-q) * x_{10} \quad . \quad \textbf{(55)}$$

Equation (3) implies also that

$$\beta_c = r * \beta_f \quad . \quad \textbf{(56)}$$

Substituting equations (4), (5), and (6) back into the model (equation (1)) yields

$$y = B * e^{\beta_f * (qx_{10} + r(1-q)x_{10})} \quad \textbf{(57)}$$

or, rearranging terms,

where

$$\begin{aligned} \beta_{10} &= \beta_f * [q + r(1-q)] \\ y &= B * e^{\beta_f * [q + r(1-q)] * x_{10}} \\ &= B * e^{\beta_{10} * x_{10}} \end{aligned}.$$

The factor [q + r(1 - q)] may be thought of as a "scaling" factor that, under the assumptions stated above, converts the PM-2.5 coefficient to the corresponding PM-10 coefficient.

Even if the relationship between PM-2.5 and mortality (i.e., β_f) is the same everywhere, then, the relationship between PM-10 and mortality (i.e., β_{10}) may vary from one place to another if the ratio of PM-2.5 to PM-10 (q) varies and/or the relative potencies of the fine and coarse fractions (r) varies.

Suppose, for example, that $\beta_f = 0.001$ everywhere and that the relative potencies of the fine to coarse fractions (r) is 0.5 everywhere (i.e., the fine fraction is twice as harmful as the coarse fraction everywhere). Suppose, however, that in Provo, Utah, only twenty percent of PM-10 is fine particles whereas in Philadelphia, eighty percent is fine particles. The coefficient of PM-10 in Provo is then

$$\beta_{10,Provo} = 0.001*[0.20 + 0.5(0.80)] = 0.0006$$

whereas in Philadelphia it is

$$\beta_{10,Philadelphia} = 0.001*[0.80 + 0.5(0.20)] = 0.0009.$$

Therefore, if the composition of PM-10 and/or the relative potencies of the fine and coarse fractions of PM-10 varies significantly from one location to another, the PM-10 concentration-response function estimated in one location may not be entirely applicable to a different location.

The generalized model (equation (1)) allows more specific analysis of alternative policy options. It also demonstrates that the PM-10 model, which does not distinguish between fine and coarse particles, may significantly misestimate the health effects associated with a given concentration of PM-10 if there really is a difference in the potency of PM-2.5 and the coarse fraction and if the proportion of PM-10 that is fine particles varies significantly from one location to another.

Appendix 5: Adjustment of Means and Standard Deviations of Distributions for Location-Specific β's

Location-specific estimates of β are adjusted to take into account all the information about β in all locations for which it has been estimated, assuming the random effects model in which location-specific β's are regarded as a sample from an underlying distribution of β's. Let

β_i denote the estimate of β in the ith location;
V_i denote the variance of the estimate, β_i ;
η^2 denote the variance of the underlying distribution of β's;
β_{pooled} denote the estimate of the mean of the distribution, derived by pooling the sample of estimates of the β_i's ; and
V_{pooled} denote the variance of the estimate of the mean, β_{pooled} .

The unadjusted probability distribution describing the probability that the true value of β in the ith location is within any given interval is a normal distribution with mean equal toβ_i and variance equal toV_i.

The *adjusted* probability distribution is a normal distribution with mean equal to

$$(\beta_i/V_i + \beta_{pooled}/\eta^2)/(1/V_i + 1/\eta^2)$$

and variance equal to

$$[1/(1/V_i + 1/\eta^2)] + [V_{pooled}/(\eta^2)^2/[1/V_i + 1/\eta^2]^2] \quad .$$

The adjusted mean is a weighted average of the original estimate, β_i , and the pooled estimate of the mean of the distribution, β_{pooled} . The larger the variance around the location-specific estimate, β_i (i.e., the less certain it is), the less weight it has in the adjusted mean.

The first term in the adjusted variance combines the within study variance (V_i) and the between study variance, η^2 . The second term in the adjusted variance is a correction for the fact that the mean of the distribution is not known but is only estimated (by β_{pooled}). This estimate therefore has some variability associated with it.

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