Particulate Matter Health Risk Assessment for Selected Urban Areas: Draft Report

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DISCLAIMER

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Any analyses, interpretations, or conclusions presented in this report based on hospitalization and mortality data obtained from outside sources, are credited to the authors and not the institutions providing the raw data. Furthermore, the authors expressly understand that the Michigan Health and Hospital Association has not performed an analysis of the hospitalization data obtained nor do they warrant the accuracy of this information and, therefore, can not be held responsible in any manner for the outcome.

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PARTICULATE MATTER RISK ASSESSMENT FOR SELECTED URBAN AREAS

1. Introduction

As required by the Clean Air Act, the U.S. Environmental Protection Agency (EPA) periodically reviews the national ambient air quality standards (NAAQS) for particulate matter (PM). As a result of the last review of the PM NAAQS completed in 1997 (62 FR 38652, July 18, 1997), EPA added new standards for $PM_{2.5}$, referring to particles with a mean aerodynamic diameter less than or equal to 2.5 μm, in order to address concerns about the fine fraction of inhalable particles. The existing PM_{10} standards, referring to particles with a mean aerodynamic diameter less than or equal to 10 μm, were originally adopted in 1987; they were retained with minor revisions in 1997 for the purpose of regulating the coarse fraction of inhalable particles.[1] The new primary (health-based) $PM_{2.5}$ standards included: an annual standard of 15 $\mu g/m^3$, based on the 3-year average of annual arithmetic mean $PM_{2.5}$ concentrations from single or multiple community-oriented monitors; and a 24-hour standard of 65 ug/m^3, based on the 3-year average of the 98th percentile of 24-hour $PM_{2.5}$ concentrations at each monitor in an area. These standards were based primarily on a large body of epidemiological evidence relating ambient PM concentrations to various adverse health effects.

As part of its last review, EPA's Office of Air Quality Planning and Standards (OAQPS) sponsored a risk assessment for two sample urban areas, Philadelphia County and Los Angeles County, to assess the risks associated with then-current PM levels and the effects of alternative PM standards on reducing estimated health risks attributable to PM (Abt Associates Inc., 1996; and Abt Associates Inc., 1997a,b. See also Deck et al., 2001 and Post et al., 2001 for published articles describing the risk assessment methodology used in the 1996-1997 analyses). Results were presented and discussed as part of the OAQPS Staff Paper (EPA, 1996b), that presented factors relevant to the evaluation of the then-current primary (health-based) NAAQS, as well as staff conclusions and recommendations of alternative standards for the EPA Administrator to consider.

The next periodic review of the PM NAAQS is now underway. EPA is currently completing the process of assessing the latest available PM health effects literature. The latest draft of this assessment is contained in the June 2003 fourth external review draft of the *Air Quality Criteria for Particulate Matter* (U.S. EPA, 2003a) (hereafter 2003 draft PM CD). This draft was released in June 2003 for review by the Clean Air Scientific Advisory Committee (CASAC) and general public. The 2003 draft PM CD includes an evaluation of the scientific evidence on the health effects of PM, including information on exposure, physiological

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[1]In May 1999, in response to challenges filed by industry groups, the U.S. Court of Appeals for the District of Columbia Circuit vacated the revised PM_{10} standards on the basis that PM_{10} is an "arbitrary indicator for coarse particulate pollution" American Trucking Associations v. EPA, 175 F. 3d 1027, 1053-55 (D.C. Cir. 1999). The 1987 PM_{10} standards remain in effect.

mechanisms by which PM might damage human health, and an evaluation of the epidemiological evidence including reported concentration-response (C-R) relationships.

At the time of the last PM CD (U.S. EPA, 1996a), a number of health studies indicated differences in health effects between fine and coarse fraction particles, and suggested that serious health effects, such as premature mortality, were more closely associated with fine fraction particles. The new studies, summarized in Chapter 8 of the 2003 draft PM CD continue to show associations between serious health effects, including premature mortality, and ambient $PM_{2.5}$ concentrations. In the last PM NAAQS review, there were a greater number of studies assessing the relationship between PM_{10} and various health effects than any other PM indicator. There were only a limited number of studies that assessed the relationship between ambient $PM_{2.5}$ and various health effects, and even fewer that assessed the relationship between ambient $PM_{10-2.5}$ and health effects. As shown in Exhibits C.1 through C.10 in Appendix C, there are significantly more studies available today that address the relationship between ambient $PM_{2.5}$ levels and significant health effects, including increased mortality associated with short- and long-term exposures, increased hospital admissions, and increased respiratory symptoms. As discussed more fully in Sections 3 and 4, these new studies include single-city studies in a variety of locations across the United States and Canada, as well as some multi-city studies. The health effects evidence summarized in Chapter 8 of the 2003 draft PM CD also now includes a number of studies that assess the relationship between ambient $PM_{10-2.5}$ and various health effects.

An initial draft report, "Proposed Methodology for Particulate Matter Risk Analyses for Selected Urban Areas,"was submitted to the CASAC for review and was made available to the public in January 2002. In that draft report, we proposed to focus on assessing risk associated with $PM_{2.5}$ and, to the extent appropriate, $PM_{10-2.5}$.[2] We received both written public comments and comments made by members of the CASAC during and subsequent to an advisory teleconference review of this initial draft report. Among its comments, the CASAC suggested carrying out an additional health risk assessment employing PM_{10} as an indicator to complement the $PM_{2.5}$ risk assessment, since many health studies used PM_{10} as the indicator and PM_{10} air quality data are available (Hopke, 2002). Risks associated with PM_{10} ambient levels are likely to reflect the contribution of $PM_{2.5}$, $PM_{10-2.5}$, or some combination of both depending on the relative composition of PM in various urban areas within the United States and Canada.

Many time-series studies, especially those carried out in recent years, involved use of generalized additive models (GAMs). In late May 2002, EPA was informed by the Health Effects Institute (HEI) of a generally unappreciated aspect in the use of S-Plus statistical software often employed to fit these models. Using appropriate modifications of the default convergence

[2]Coarse particle concentrations have been measured directly using a dichotomous sampler or by subtraction of particles measured by a $PM_{2.5}$ sampler from those measured by a co-located PM_{10} sampler. This measurement is an indicator for the fraction of coarse-mode thoracic particles (i.e., those capable of penetrating to the tracheo-bronchial and the gas-exchange regions of the lung).

criteria code in the S-Plus software and a correct approach to estimating the variance of estimators will change the estimated C-R functions and could change the results of tests of significance of estimates, although it is not possible to predict *a priori* how estimates and significance tests will change. Many but not all of the C-R functions that were originally estimated using the S-Plus software for fitting GAMs have since been re-estimated using revised methods. In May 2003, HEI published a special peer-reviewed panel report describing the issues involved and presenting the results of the re-analyzed studies (HEI, 2003). The panel observed that "the impact of using more appropriate convergence criteria on the estimates of PM effect in the revised analyses varied greatly across the studies," but noted that "in no study were conclusions based on the original analyses changed in a meaningful way by the use of stricter criteria" (p. iii).

A draft memorandum (Post, April 8, 2003) was made available to the CASAC and the public describing changes in the recommended methodology and scope for the $PM_{10-2.5}$ and PM_{10} risk assessments in light of the re-analyzed study results and the CASAC and public comments.

The risk assessment described in this report includes three PM indicators – $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ – and provides an updated description of the methodology initially discussed in the January 2002 draft report, taking into account comments received from the CASAC and the public, as well as changes made in light of studies re-analyzed as a result of the S-Plus/GAM issue.[3] It is based on the health effects evidence assessed in the current draft of the PM CD, which included the re-analyzed studies presented in the HEI special report (HEI, 2003). Because the health effects and studies included are based on the current draft PM CD, they are subject to revision based on any changes made to the draft PM CD as a result of CASAC and public review.

The goals of the PM risk assessment are: (1) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates and (2) to gain insights into the nature of the risks associated with exposures to PM. In addition, the risk assessment also provides a rough sense of the potential magnitude of PM-related mortality and morbidity associated with current PM levels and with attaining the current and alternative PM NAAQS recommended for consideration. The next draft of this report will address any potential alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards identified as part of this review. EPA recognizes that the role of the risk assessment in this standards review will necessarily be limited by significant uncertainties, as discussed in Section 6 below.

As discussed in Chapter 9 of the PM CD (p. 9-67), the newer experimental evidence "... tends to support the likelihood of a causal relationship between low ambient concentrations of

[3] We hereafter refer to the "PM risk assessment" unless reference to a specific PM indicator (e.g., $PM_{2.5}$) is required. The PM risk assessment considers each of the three PM indicators – $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ – in turn.

PM and increased mortality or morbidity risks observed in human populations." The PM risk assessment described in this draft report is premised on the assumption that PM is causally related to the mortality, morbidity, and symptomatic effects (alone and/or in combination with other pollutants) observed in the epidemiological studies. We recognize, as discussed in the draft PM CD (p.8-276), that "the apparent differences in PM_{10} and/or $PM_{2.5}$ effect sizes across different regions should not be attributed merely to possible variations in measurement error or other statistical artifact(s). Some of these differences may reflect: real regional differences in particle composition or co-pollutant mix; differences in relative human exposures to ambient particles or other gaseous pollutants; sociodemographic differences (e.g., percent of infants or elderly in regional population); or other important, as of yet unidentified PM effect modifiers."

Given the availability of additional urban locations with recent and sufficient $PM_{2.5}$ and PM_{10} air quality data, and additional health effect studies in various locations in different regions of the United States, and consistent with the advice of the CASAC, EPA has expanded the scope of its PM risk assessment from the last review to several additional urban areas, consistent with the goals of the assessment. Philadelphia and Los Angeles Counties, which were the only areas included in the prior risk assessment, are included. As discussed in greater detail in Section 3, additional areas included for short- and/or long-term exposure mortality in the $PM_{2.5}$ risk assessment are as follows: Boston, Detroit, Phoenix, Pittsburgh, San Jose, and St. Louis. In addition, increased hospital admissions associated with $PM_{2.5}$ were estimated for Detroit, Los Angeles, and Seattle, and increased respiratory symptoms were estimated for Boston and St. Louis. Inclusion of these additional areas allows EPA to explore potential geographic differences in risk estimates.

The $PM_{10\text{-}2.5}$ risk assessment is more limited because of the more limited air quality data (requiring co-located $PM_{2.5}$ and PM_{10} monitors) as well as the smaller number of studies. Consistent with advice from the CASAC, short-term exposure mortality was included among the health effects in the $PM_{10\text{-}2.5}$ risk assessment. The areas included for short-term exposure mortality in the $PM_{10\text{-}2.5}$ risk assessment are Detroit, Philadelphia, Phoenix, and St. Louis. In addition, increased hospital admissions associated with $PM_{10\text{-}2.5}$ were estimated for Detroit and Seattle, and increased respiratory symptoms were estimated for St. Louis.

The areas included for short-term exposure mortality in the PM_{10} risk assessment are as follows: Boston, Chicago, Detroit, Los Angeles, Minneapolis, Philadelphia, Phoenix, Pittsburgh, Provo, San Jose, Seattle, and St. Louis. In addition, increased hospital admissions associated with PM_{10} were estimated for Chicago, Detroit, Los Angeles, Minneapolis, Pittsburgh, Provo and Seattle, and increased respiratory symptoms were estimated for Boston, Provo, Seattle, and St. Louis.

The PM risk assessment has two parts. The basic question addressed in the first part is of the following form:

For a given human health endpoint (mortality, hospital admissions, etc.), what is the estimated annual incidence of the health endpoint that may be associated with "as is"[4] PM concentrations in excess of background concentrations in these locations?[5]

In the second part, the risk assessment estimates the risk reductions that would result if the current $PM_{2.5}$ standards (15 $\mu g/m^3$ for the annual standard and 65 $\mu g/m^3$ for the daily standard) or alternative $PM_{2.5}$ or $PM_{10-2.5}$ standards were just met in the selected locations.[6] The basic question addressed in this part of the risk assessment is of the following form:

For a given human health endpoint (mortality, hospital admissions, etc.), what is the estimated reduction in annual incidence in terms of percentage and absolute numbers associated with the reduction in PM concentrations that would be expected to result if the current or alternative sets of PM standards were just met?

The methods used to estimate risks and risk reductions in the selected urban areas in the risk assessment are similar to the methods used in the previous PM risk assessment. An overview of these methods is presented in Section 2, including discussion of any significant differences in approach from the risk assessment conducted for the last review. Section 3 discusses the selection of health endpoints and urban areas from a broader list of candidate health endpoints and locations to include in the risk assessment, as well as the selection of studies estimating C-R functions. Section 4 describes the approach to selecting and using C-R functions from the broader candidate pool of C-R functions available. Section 5 presents baseline health effects incidence rates (i.e., the health effects incidence rates associated with "as is" PM levels) for each of the assessment locations. Because the risk assessment was of necessity carried out with incomplete information, several assumptions were made at several points in the analysis process. These assumptions and the various sources of uncertainty in the analyses are briefly discussed in Section 2.6 and in greater detail in Section 6. Appendix A discusses the air quality data used in the analyses. Appendix B describes an analysis of historical air quality data relevant

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[4]"As is" PM concentrations are defined here as a recent year of air quality.

[5]Consistent with the approach taken in the prior PM risk assessment, estimates of risks above background PM concentrations are judged to be more relevant to policy decisions about the level of ambient air quality standards than estimates that include risks potentially attributable to uncontrollable background PM concentrations. Thus, risks are estimated only for concentrations exceeding "background" levels, where "background" is defined as the PM concentrations that would be observed in the U.S. in the absence of anthropogenic, or man-made, emissions of primary PM and precursor emissions of volatile organic compounds, nitrogen oxides, sulfur dioxide, and ammonia in North America. Therefore, "background" for the purposes of the PM risk assessment includes PM from natural sources and transport of PM from sources outside of North America.

[6] Risk reductions associated with just meeting alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards will be included in the next draft of the risk assessment report.

to the choice of air quality adjustment procedure for simulating attainment of current $PM_{2.5}$ standards. Appendix C summarizes relevant study-specific information used to carry out the base case risk assessment and sensitivity analyses. Because the PM risk assessment covers $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} and a substantial number of urban locations, there are many exhibits of results. The results for all three PM indicators are summarized in figures in Sections 7 and 8. Exhibits containing quantitative results are presented in the main body of the report for only one location (Detroit) for illustrative purposes. Results exhibits for the other locations are presented in Appendix D for base case and sensitivity analyses from the first (recent air quality/"as is") part of the risk assessment and Appendices E and F (part of E and all of F related to alternative $PM_{2.5}$ and alternative $PM_{10-2.5}$ standards will be presented in the next draft of this report) for base case and sensitivity analyses from the second (just meeting the current $PM_{2.5}$ standards and alternative $PM_{2.5}$ and alternative $PM_{10-2.5}$ standards) part of the risk assessment.

2. Overview of Methods

This section gives an overview of the methods used in the risk assessment. Section 2.1 presents the basic structure of the risk assessment, distinguishing between its two parts – i.e., risk associated with "as is" PM levels (defined as a recent year of air quality) and risk reductions associated with just meeting the current and potential alternative PM standards – and identifying the basic information elements needed for the analyses. Section 2.2 discusses air quality inputs. Section 2.2.1 discusses the estimation of $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} background levels; Section 2.2.2 discusses the characterization of "as is" PM levels. Section 2.3 discusses the simulation of PM concentrations that just meet specified PM standards. A brief discussion of issues surrounding baseline incidence rates is given in Section 2.4. The calculation of health effects incidence and incidence reductions is described in Section 2.5. Section 2.6 gives an overview of the characterization of uncertainty and variability in the PM risk assessment. Finally, sensitivity analyses are discussed in Section 2.7.

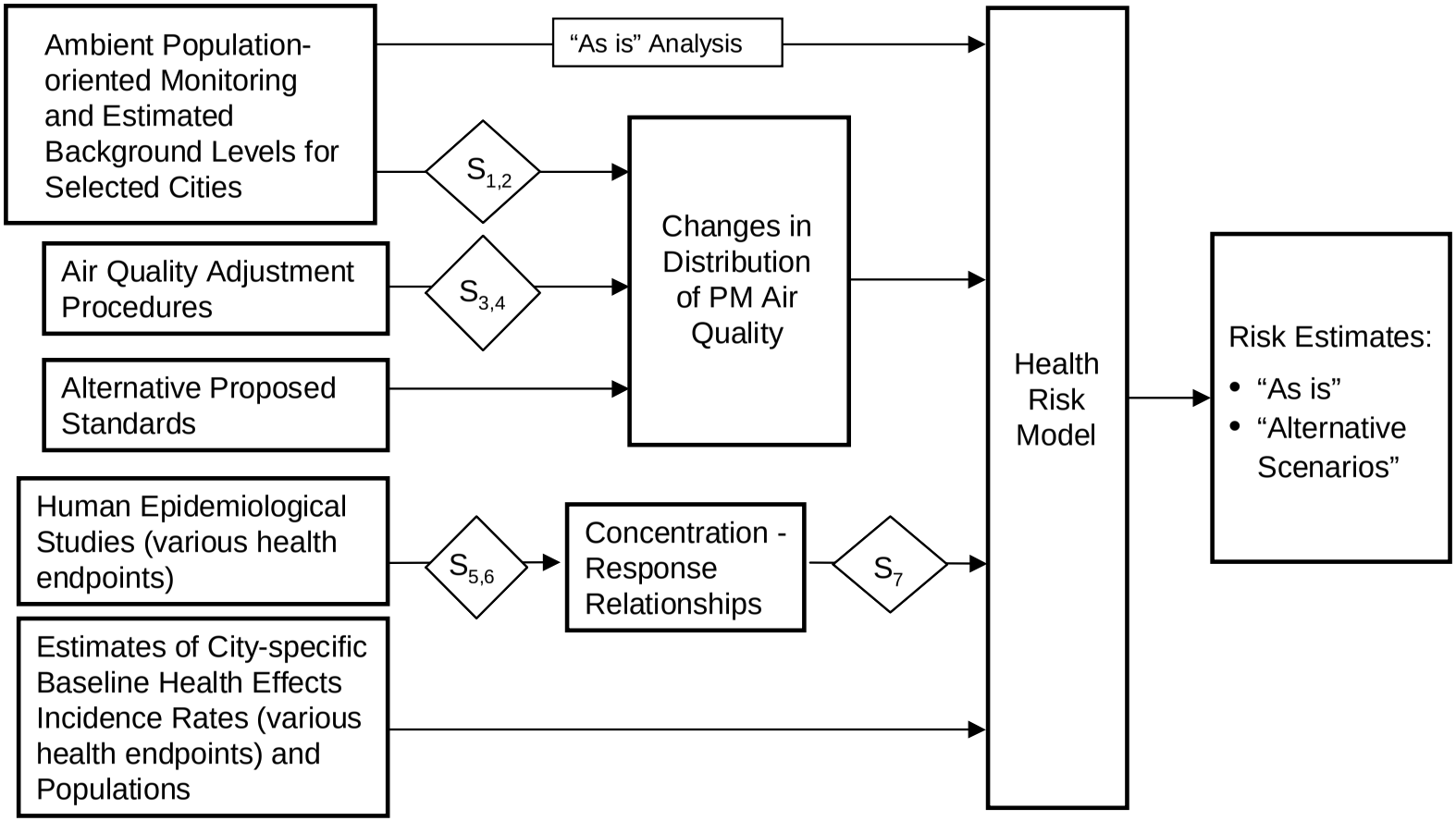
2.1 Basic structure of the risk assessment

The general approach used in both the prior and the current PM risk assessment relies upon C-R functions which have been estimated in epidemiological studies. Since these studies estimate C-R functions using ambient air quality data from fixed-site, population-oriented monitors, the appropriate application of these functions in a PM risk assessment similarly requires the use of ambient air quality data at fixed-site, population-oriented monitors. The general PM health risk model combines information about PM air quality for specific urban areas with C-R functions derived from epidemiological studies and baseline health incidence data for specific health endpoints and population estimates to derive estimates of the annual incidence of specified health effects attributable to ambient PM concentrations. The analyses are conducted for both "as is" air quality and for air quality simulated to reflect attainment of current (and in the next draft of the risk assessment report, alternative) PM ambient standards.

An overview of the major components of the risk assessment discussed in this report is presented in Exhibit 2.1. The points in the risk assessment at which sensitivity analyses were carried out are represented by diamonds. The sensitivity analyses (labeled in Exhibit 2.1 as s_k's) are described in Exhibit 2.5 below.

In the first part of the risk assessment, we estimate risks associated with "as is" PM levels in excess of background level, or the lowest measured level (LML) observed in the study, if it is higher than the estimated background level in the assessment location. A C-R relationship estimated by an epidemiological study may not be valid at concentrations outside the range of concentrations observed during the study. To partially address this problem, risk was not calculated for PM levels below the lowest observed level in a study, if it was available.

Exhibit 2.1 Major Components of Particulate Matter Health Risk Analyses



S_k = k^{th} Sensitivity Analysis (See Exhibit 2.5): Analysis of effects of alternative assumptions, procedures or data occurs at these points.

In the second part of the risk assessment, we estimate the reduction in risk associated with the change from "as is" $PM_{2.5}$ concentrations to those concentrations that would result if the current or alternative PM standards were just met in the assessment locations. The method used in both parts of the risk assessment is basically the same. The important operational difference between the two parts is in the specified lower PM levels. In the first part, the lower PM level is background (or the LML in the study). In contrast, the lower PM levels in the second part of the risk assessment are the estimated PM levels that would occur when the current $PM_{2.5}$ or alternative $PM_{2.5}$ or $PM_{10\text{-}2.5}$ standards are just met in the assessment locations. The second part requires that a method be developed to simulate just meeting the specified standards.

To estimate the change in the incidence of a given health effect resulting from a given change in ambient PM concentrations in an assessment location, the following analysis inputs are necessary:

- **Air quality information** including: (1) "as is" air quality data for PM from population-oriented monitors in the assessment location, (2) estimates of background PM concentrations appropriate to this location, and (3) a method for adjusting the "as is" data to reflect patterns of air quality change estimated to occur when the area just meets the specified standards.[7] (These air quality inputs are discussed in more detail in Section 2.2).

- **Concentration-response function(s)** which provide an estimate of the relationship between the health endpoint of interest and PM concentrations (preferably derived in the assessment location, although functions estimated in other locations can be used at the cost of increased uncertainty -- see Section 6.1.2). For $PM_{2.5}$, C-R functions are available from epidemiological studies for both short- and long-term exposures. For PM_{10} and $PM_{10\text{-}2.5}$, only short-term exposure studies are included in the risk assessment. (Section 2.5 describes the role of C-R functions in estimating health risks associated with PM).

- **Baseline health effects incidence rate and population**. The baseline incidence rate provides an estimate of the incidence rate (number of cases of the health effect per year, usually per 10,000 or 100,000 general population) in the assessment location corresponding to "as is" PM levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number (e.g., if the baseline incidence rate is number of cases per year per 100,000 population, it must be multiplied by the number of 100,000s in the population). (Section 2.4 summarizes

[7] Although in the current draft of the PM risk assessment, we consider only the current $PM_{2.5}$ standards, in a subsequent draft we will consider alternative $PM_{2.5}$ and $PM_{10\text{-}2.5}$ standards. The general method for adjusting "as is" air quality to simulate just meeting a set of standards doesn't depend, however, on the standards themselves.

considerations related to the baseline incidence rate and population data inputs to the risk assessment).

The risk assessment procedure described in more detail below is diagramed in Exhibit 2.2 for analyses based on short-term exposure studies and in Exhibit 2.3 for analyses based on long-term exposure studies.

Exhibit 2.2 Flow Diagram of Risk Analyses for Short-Term Exposure Studies

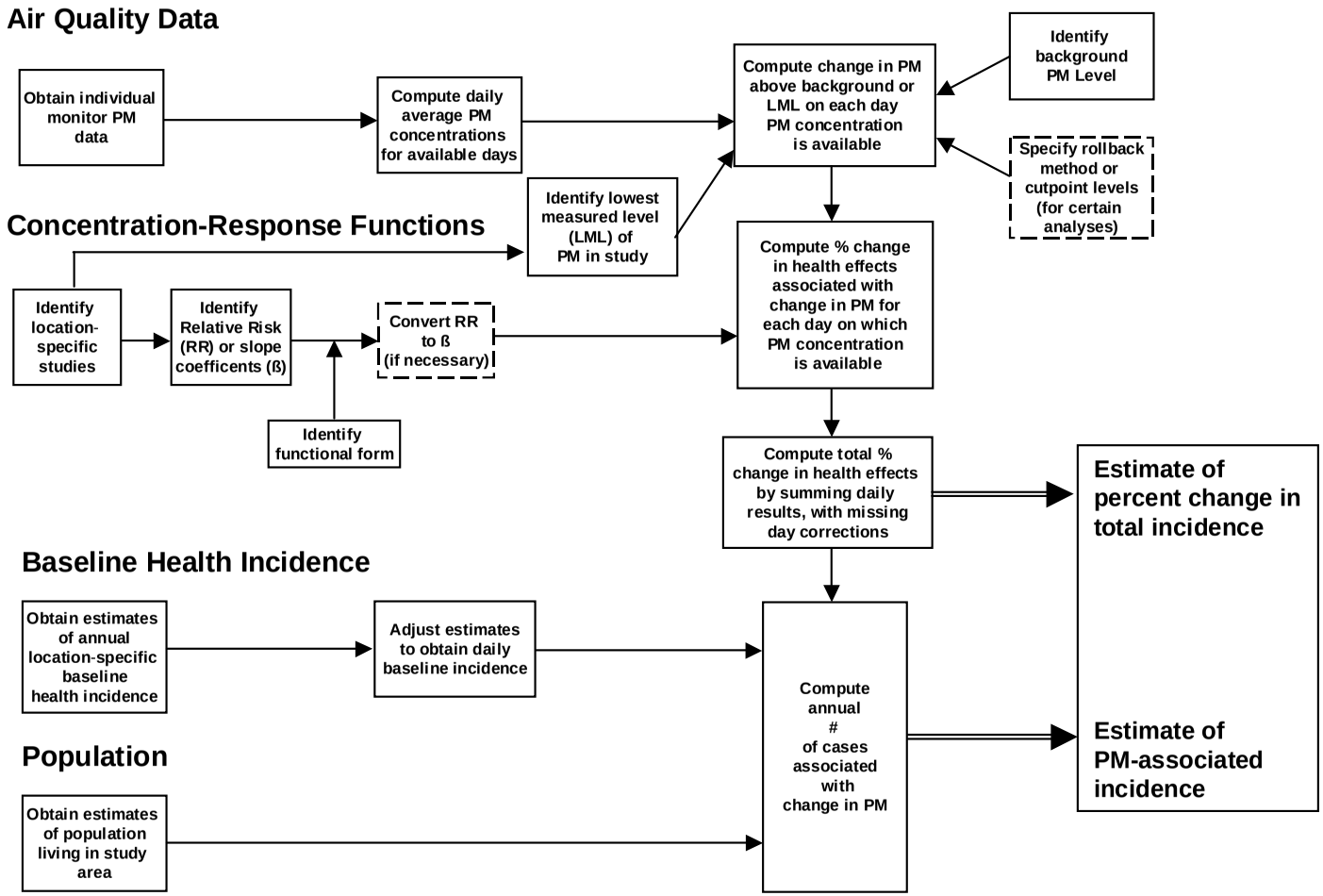
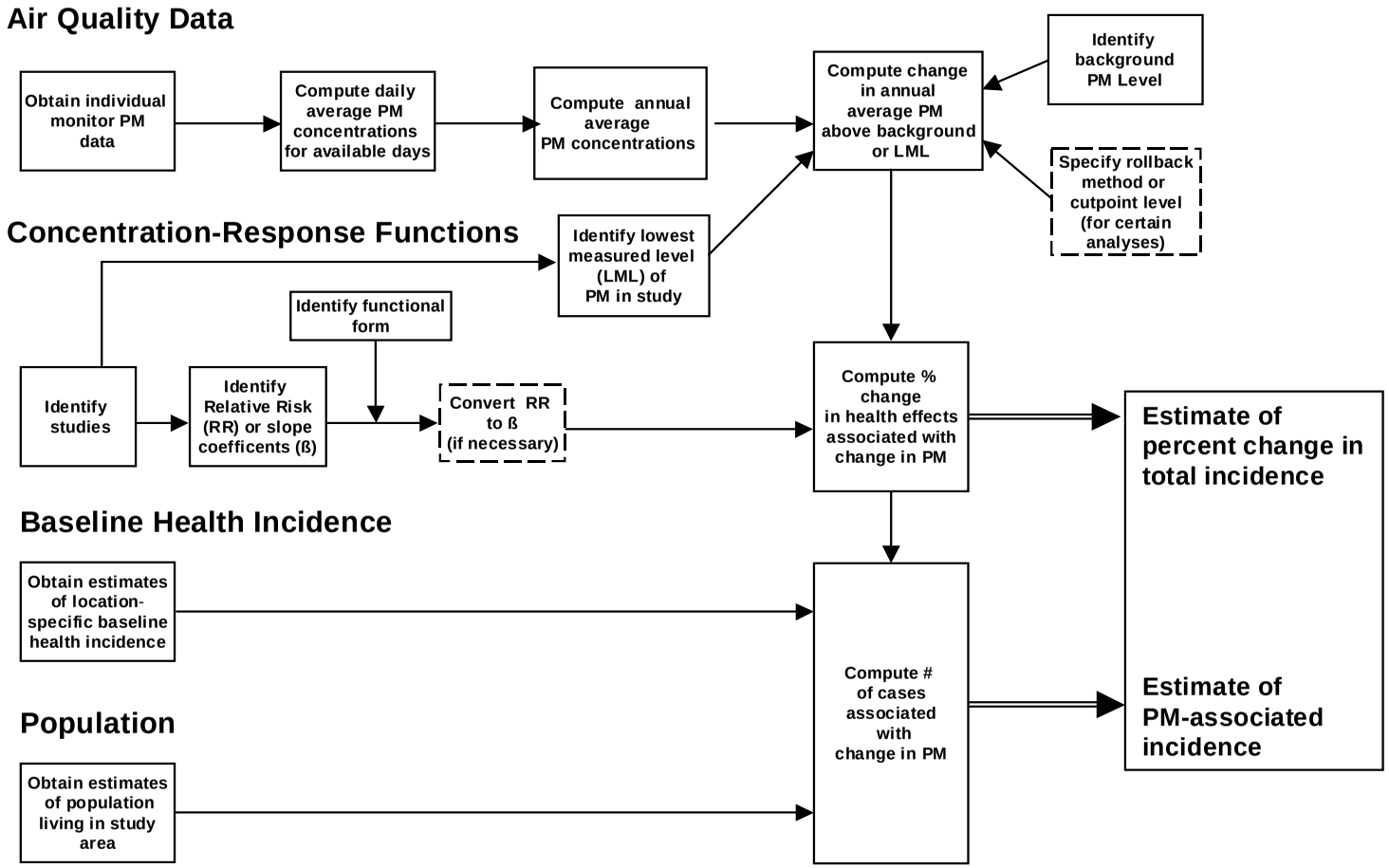


Exhibit 2.3 Flow Diagram of Risk Analyses for Long-Term Exposure Studies



2.2 Air quality inputs

2.2.1 Estimating PM background levels

Since health risks are calculated only for concentrations exceeding estimated background levels, estimates of background PM concentrations in the assessment locations are needed to calculate risk at "as is" concentrations in excess of background and for risk reductions associated with just meeting the current $PM_{2.5}$ ambient standards and just meeting alternative $PM_{2.5}$ and alternative $PM_{10-2.5}$ ambient standards.

Consistent with the prior PM CD, the 2003 draft PM CD estimates background annual average $PM_{2.5}$ concentrations to be in the range of 1 to 4 $\mu g/m^3$ in the Western United States and 2 to 5 $\mu g/m^3$ in the Eastern United States (p.3-82). We use the midpoints of these ranges for the base case analysis. Thus background $PM_{2.5}$ concentrations in the base case analysis are estimated to be 3.5 $\mu g/m^3$ in Boston, Detroit, Philadelphia, Pittsburgh, and St. Louis; and 2.5 $\mu g/m^3$ in Los Angeles, Phoenix, San Jose, and Seattle. In a sensitivity analysis, we allow daily $PM_{2.5}$ background levels to vary according to lognormal distributions whose means are equal to the values used in the base case analysis (see s_1 in Exhibit 2.1). Section 7.2 provides a more detailed discussion of the sensitivity analyses performed, including the different daily background sensitivity analysis.

Also consistent with the prior PM CD, the 2003 draft PM CD estimates background annual average PM_{10} concentrations to be in the range of 4 to 8 $\mu g/m^3$ in the Western United States and 5 to 11 $\mu g/m^3$ in the Eastern United States (p.3-82). We use the midpoints of these ranges for the base case PM_{10} analysis. Thus background PM_{10} concentrations in the base case analysis are estimated to be 8 $\mu g/m^3$ in those urban areas in the East (i.e., Boston, Chicago, Detroit, Minneapolis-St. Paul, Philadelphia, Pittsburgh, and St. Louis); and 6.0 $\mu g/m^3$ in those urban areas in the West (i.e., Los Angeles, Phoenix, Provo, San Jose, and Seattle).

The draft PM CD (p. 3-82) estimates background annual average $PM_{10-2.5}$ to be approximately 3 $\mu g/m^3$ in both the East and the West, with a range of 0 to 9 $\mu g/m^3$ in the East and 0 to 7 $\mu g/m^3$ in the West. We use 3 $\mu g/m^3$ as the estimated background for $PM_{10-2.5}$ in the base case analysis for the coarse risk assessment locations (i.e., Detroit, Philadelphia, Phoenix, Seattle, and St. Louis).

2.2.2 Characterizing "as is" PM air quality

As discussed earlier, a major input to the PM risk assessment is ambient PM air quality data for each assessment location. In order to be consistent with the approach used in the epidemiological studies that estimated PM C-R functions, the *average* ambient PM concentration on each day for which measured data are available is needed for the risk assessment. Consistent

with the approach used in the last PM risk assessment, a composite monitor data set was created for each assessment location based on a composite of all monitors (except those that are identified as being source-oriented monitors) with at least 11 observations per quarter. At the time of the last PM risk assessment, there was no established $PM_{2.5}$ monitoring network and data sets from special studies conducted in Philadelphia and Los Angeles had to be used. There are now substantial $PM_{2.5}$ air quality data in EPA's Aerometric Information Retrieval System (AIRS) beginning with the year 1999. There were sufficient $PM_{2.5}$ data in AIRS for the year 2002 for all of the assessment locations except Phoenix and San Jose, for which we used year 2001 data.

There were sufficient PM_{10} data in AIRS for the year 2002 for all of the assessment locations except Boston and San Jose, for which we used year 1999 data (the most recent year for which there were sufficient data). For the $PM_{10\text{-}2.5}$ risk assessment there were sufficient data from co-located monitors in the year 2002 for Detroit, Philadelphia, and St. Louis. There were sufficient $PM_{10\text{-}2.5}$ air quality data for Seattle in the year 2000. For Phoenix, there were not sufficient $PM_{10\text{-}2.5}$ AIRS data in any of the years from 1999 through 2002. Instead, we used data from 1997, based on co-located TEOM monitors. Because of this, the $PM_{10\text{-}2.5}$ risk assessment in Phoenix is treated as a separate special case.

As noted above, $PM_{10\text{-}2.5}$ air quality was calculated from $PM_{2.5}$ and PM_{10} concentrations at co-located monitors by subtracting the former from the latter. Because of measurement error, some of the $PM_{10\text{-}2.5}$ concentrations that were calculated were negative. In Detroit, 15.7 percent of the days for which $PM_{10\text{-}2.5}$ concentrations were calculated were negative; in St. Louis, 16.7 percent of the days were negative. There were no negative $PM_{10\text{-}2.5}$ concentrations calculated in Philadelphia or Seattle.

If left as they are, negative $PM_{10\text{-}2.5}$ values will result in a positive incidence contribution – that is, going from the negative "as is" value to background will result in an *increase* in cases. While negative $PM_{10\text{-}2.5}$ values are obviously errors, and the consequent increases in cases are likewise wrong, this would presumably somewhat balance the $PM_{10\text{-}2.5}$ measurement errors in the other direction – i.e., errors which resulted in "as is" $PM_{10\text{-}2.5}$ values that are too large. However, because most C-R functions are exponential, even if the errors are randomly and evenly distributed around zero, the corresponding errors in cases contributed will *not* be evenly distributed around zero – in particular, "as is" values that are too small, including negative values, will have a greater impact than "as is" values that are too big, resulting in a net *downward* bias in cases avoided. Alternatively, for those days for which a negative $PM_{10\text{-}2.5}$ concentration was calculated, we can set the estimated risk to zero. This approach leads to *upward*- biased estimate of cases avoided. Either way, however, the biases are likely to be small. The $PM_{10\text{-}2.5}$ coefficient is typically small, so the C-R function, while not linear, is almost linear, in which case the impact of $PM_{10\text{-}2.5}$ values that are too small will be about the same as the impact of those values that are too large. So whatever downward bias we get from the negative values would be minor, because they will be mostly balanced by the upward bias of the values that are too large.

Similarly, if we take the second approach, and allow days with negative "as is" values to contribute zero cases avoided, the upward bias should be relatively small, because there aren't that many days with negative values. We chose to take the second approach, which avoids estimating a positive improvement in health associated with negative ambient PM concentrations. The cases avoided on days with negative estimated $PM_{10-2.5}$ concentrations were set to zero.

Appendix A summarizes the $PM_{2.5,}$ PM_{10}, and $PM_{10-2.5}$ air quality data that were used in each of the assessment locations, including quarterly and annual counts, annual averages, and the 98th percentile of the daily (24-hour) averages. Because the air quality data are not uniformly complete, annual averages were calculated as the average of quarterly averages to minimize the possible bias resulting from differential amounts of missing data in different quarters of the year.

2.3 Simulating PM levels that just meet specified PM standards

This section describes the methodology used to simulate ambient PM levels in an area upon just meeting specified annual and daily PM standards, using the current $PM_{2.5}$ standards to illustrate the approach. The form of the $PM_{2.5}$ standards promulgated in 1997 requires that the 3-year average (rounded to the nearest 0.1 $\mu g/m^3$) of the annual means *from single monitors or the average of multiple monitors* must be at or below the level of the annual standard and the 3-year average (rounded to the nearest1 $\mu g/m^3$) of the ninety-eighth percentile values *at each monitor* cannot exceed the level of the daily standard. In determining attainment of the annual average standard an area may choose to use either the spatially averaged concentrations across all population-oriented monitors or it may use the highest 3-year average based on individual monitors. The most realistic simulation of just meeting both the annual and the daily standards in a location would require changing the distribution of daily $PM_{2.5}$ concentrations at each monitor separately. This would require extensive analysis and assumptions about the nature of future control strategies that was considered beyond the scope of the previous risk assessment and is similarly considered beyond the scope of the current risk assessment.

Consistent with the approach used in the prior PM risk assessment, just meeting the current $PM_{2.5}$ standards was simulated by changing daily $PM_{2.5}$ concentrations at a "composite monitor," which represents the average of the monitors in a location. The $PM_{2.5}$ concentration at the composite monitor on a given day is defined as the average of the $PM_{2.5}$ concentrations of those monitors reporting on that day. The percent reduction of the $PM_{2.5}$ concentration at the composite monitor each day resulting from just meeting the current standard is determined by the $PM_{2.5}$ concentration at the "controlling monitor" – that is, at the monitor with the highest relevant value (e.g., the highest 98th percentile value, for the daily standard). However, the changes in $PM_{2.5}$ to simulate just meeting the standards are made at the composite monitor rather than at the individual monitors. In addition, although the standard refers to the 3-year average, because of

the limited number of years for which $PM_{2.5}$ data are available, we used only a single year of data for the risk assessment.[8]

There are many possible ways to create an alternative distribution of daily concentrations that just meets a specified set of $PM_{2.5}$ standards. The prior PM risk assessment used a proportional rollback of all PM concentrations exceeding the estimated background concentration for its base case estimates. This choice was based on analyses of historical $PM_{2.5}$ data which found that year-to-year reductions in $PM_{2.5}$ levels in a given location tended to be roughly proportional. That is, both high and low daily $PM_{2.5}$ levels decreased proportionally from one year to the next (see Abt Associates Inc., 1996, Section 8.2). This suggests that, in the absence of detailed air quality modeling, a reasonable method to simulate the $PM_{2.5}$ reductions that would result from just meeting a set of standards would be proportional rollbacks -- i.e., to decrease $PM_{2.5}$ levels on all days by the same percentage. Appendix B describes a new analysis of historical air quality data for Philadelphia and Los Angeles which continues to support the hypothesis that changes in $PM_{2.5}$ levels that would result if a $PM_{2.5}$ standard were just met are reasonably modeled by using a proportional rollback approach. We recognize that the historic changes in $PM_{2.5}$ have not been the result of a $PM_{2.5}$ control strategy, but likely result from control programs for PM_{10} and control programs for other pollutants (especially sulfur and nitrogen oxides). The pattern of changes that have occurred in the past, therefore, may not necessarily reflect the changes that will result from future efforts to attain $PM_{2.5}$ standards. However, it is interesting to note that reductions in ambient $PM_{2.5}$ concentrations are reasonably modeled by proportional rollbacks in both Philadelphia and Los Angeles, which likely had very different reductions in terms of types of emissions over this period.

Based on the above considerations, we simulated just meeting the current $PM_{2.5}$ standards by use of a proportional rollback procedure for the base case estimates. That is, average daily $PM_{2.5}$ concentrations at the composite monitor were reduced by the same percentage on all days. The percentage reduction was determined by comparing the maximum of the monitor-specific annual averages (or the maximum of the monitor-specific ninety-eighth percentile daily values) adjusted for background with the level of the annual standard (or daily standard) adjusted for background. Because pollution abatement methods are applied largely to anthropogenic sources of $PM_{2.5}$, rollbacks were applied only to $PM_{2.5}$ above estimated background levels. The percent reduction was determined by the controlling standard. For example, suppose both an annual and a daily $PM_{2.5}$ standard are being simulated. Suppose p_a is the percent reduction required to just meet the annual standard (i.e., the percent reduction of daily $PM_{2.5}$ above background necessary to get the highest of the monitor-specific annual averages down to the annual standard). Suppose p_d is the percent reduction required to just meet the daily standard (i.e., the percent reduction of daily $PM_{2.5}$ above background necessary to get the maximum monitor-specific ninety-eighth

[8]The use of a single year of data may be viewed as equivalent to assuming the distribution of $PM_{2.5}$ concentrations is the same for each year during a three-year period.

percentile daily $PM_{2.5}$ value down to the daily standard). If p_d is greater than p_a, then all daily average $PM_{2.5}$ concentrations above background are reduced by p_d percent. If p_a is greater than p_d, then all daily average $PM_{2.5}$ concentrations are reduced by p_a percent.

The method of rollbacks to meet a set of annual and daily PM standards is summarized as follows:

1. Calculate the annual average of PM concentrations at each monitor[9];
2. Calculate the maximum of these monitor-specific annual averages, denoted aa_{max};
3. The percent by which the above-background portion of all daily PM concentrations (at the composite monitor) would have to be reduced to just meet the annual standard (denoted std_a) is

$$p_a = 1 - \frac{(std_a - b)}{(aa_{\max} - b)}$$

where b denotes background.

4. Calculate the ninety-eighth percentile value of the distribution of daily PM concentrations at each monitor[10];
5. Calculate the maximum of these monitor-specific ninety-eighth percentile values, denoted $98\%ile_{max}$;
6. The percent by which the above-background portion of all daily PM concentrations (at the composite monitor) would have to be reduced to just meet the daily standard (denoted std_d) is

$$p_d = 1 - \frac{(std_d - b)}{(98\%ile_{\max} - b)}$$

.

[9] Because there are missing air quality data, annual averages were calculated as the average of quarterly averages to minimize possible bias resulting from differential amounts of missing data in different quarters of the year.

[10] The method of calculating the ninety-eighth percentile value of daily PM concentrations at a monitor is consistent with the method used by EPA, as described in Appendix N of the July 18, 1997 Federal Register Notice (available on the web at www.epa.gov/ttn/oarpg/t1pfpr.html).

7. Let p_{max} = maximum of (maximum of p_a and p_d) and zero.[11]
8. Then, if PM_o denotes the original PM value on a given day (at the composite monitor), the rolled back PM value on that day, denoted PM_{rb}, is

$$PM_{rb} = b + (PM_o - b) * (1 - p_{\max})$$

The inputs to calculate the percent rollbacks necessary to simulate just meeting annual and daily $PM_{2.5}$ standards of 15 and 65 μg/m^3, respectively, are given in Exhibit 2.4 for St. Louis to illustrate the approach that was taken in all locations. The controlling standard (i.e., daily or annual) and corresponding percent rollback necessary to just meet the current $PM_{2.5}$ standards in St. Louis are also shown in Exhibit 2.4. Since an area could potentially use the spatial average of the population-oriented monitors to determine whether or not it met the annual average standard, the risk assessment report also presents the percent rollbacks that would have resulted from using this alternative approach in each urban study area (see Section 8).

[11] If the percent rollback necessary to just meet the annual standard and the percent rollback necessary to just meet the daily standard were both negative -- i.e., if both standards were already met -- then the percent rollback applied in the risk assessment was zero. That is, PM values were never increased.

Exhibit 2.4 Inputs to Calculation of Rollbacks to Simulate Just Meeting Annual and Daily $PM_{2.5}$ Standards of 15 μg/m^3 and 65 μg/m^3, Respectively, in St. Louis, MO

Monitor Site	Annual Average $PM_{2.5}$ Concentration (μg/m^3)	98th Percentile $PM_{2.5}$ Concentration (μg/m^3)
AIRS 171192009881011	14.7	34.5
AIRS 171634001881011	15.1	37.2
AIRS 290990012881011**	15.1	39.8
AIRS 291831002881011	14.2	35.0
AIRS 291890004881011	13.0	34.0
AIRS 291892003881011	14.6	35.6
AIRS 291895001881011	13.4	38.4
AIRS 295100007881011	15.3	36.5
AIRS 295100085881011*	15.4	36.5
AIRS 295100086881011	14.3	35.5
Maximum of Annual Averages:	**15.4**	**---**
Percent rollback to just meet an annual standard of 15 μg/m^3	**3.36%**	**---**
Maximum of 98th Percentile Values:	**---**	**39.8**
Percent rollback to just meet a daily standard of 65 μg/m^3	**---**	**0**
Controlling standard in St. Louis: Annual Percent rollback required to meet both standards is 3.36%		

*Controlling monitor for the annual standard.

**Controlling monitor for the daily standard.

As noted earlier, proportional rollback is only one of many possible ways to create an alternative distribution of daily concentrations to meet new $PM_{2.5}$ 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 proportional rollbacks, it is possible to meet a daily standard by "peak shaving." The peak shaving method would reduce all daily $PM_{2.5}$ 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. Because the reduction method to attain a daily standard could have a significant impact on the risk assessment results, a sensitivity analysis was conducted using an alternative rollback method (see S_2 in Exhibit 2.1). As with the sensitivity analysis performed for the prior risk assessment, this sensitivity analysis used a rollback method in which the upper 10% of the $PM_{2.5}$ air quality distribution was rolled back to a greater extent than the remaining 90% of the distribution. In particular, the percentage by which the upper 10% of the $PM_{2.5}$ air quality distribution was rolled back was 1.6 times the percentage by which the rest of the distribution was rolled back. See Section 8 for a more detailed discussion of the alternative rollback sensitivity analysis.

2.4 Baseline health effects incidence data

As noted in Section 2.5 below, the form of C-R function most commonly used in epidemiological studies on PM, shown in equation (1), is log-linear. To estimate the change in incidence of a health endpoint associated with a given change in PM concentrations using this form of C-R function requires the baseline incidence rate of the health endpoint, that is, the number of cases per unit time (e.g., per year) in the location *before* a change in PM air quality (denoted y in equations 3 and 4).

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., the number of cases per 10,000 residents in Philadelphia County), 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).

Incidence rates are available for mortality and for specific communicable diseases which state and local health departments are required to report 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. None of the morbidity endpoints in the risk assessment are required to be reported to the federal government.

Although federal agencies collect incidence data on many of the endpoints covered in the PM risk assessment, their data are often available only at the national level, or at the regional or state level. One important exception is mortality rates, which are available at the county level. Because baseline incidence rates can vary from one location to another, location-specific baseline incidence information was obtained. Because hospital admission rates are available for some locations and not others, this was a consideration in the selection of locations for which to

conduct the PM risk assessment. For respiratory symptom health endpoints, the only estimates of baseline incidence rates available are typically from the studies that estimated the C-R functions for those endpoints. However, because risk assessment locations for these endpoints were selected partly on the basis of where studies were carried out, baseline incidence rates reported in the studies should be appropriate to the risk assessment locations to which they are applied. A more detailed discussion of baseline health effects incidence data is presented in Section 5.

2.5 Calculating health effects incidence

2.5.1 General approach

The C-R functions used in the risk assessment are empirically estimated relations between average ambient concentrations of PM and the health endpoints of interest (e.g., short- and long-term exposure mortality or hospital admissions) reported by epidemiological studies for specific locales. This section describes the basic method used to estimate changes in the incidence of a health endpoint associated with changes in PM, using a "generic" C-R function of the most common functional form.

Although one epidemiological study estimated linear C-R functions and one estimated logistic functions, most of the studies used a method referred to as "Poisson regression" to estimate exponential (or log-linear) C-R functions in which the natural logarithm of the health endpoint is a linear function of PM:[12]

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

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 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 log-linear form of C-R function (equation (1)) is by far the most common form, the discussion that follows assumes this form.

[12]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 assessment, however, is simply the form of the estimated relation, as shown in equation (1).

2.5.2 Short- and long-term exposure endpoints

C-R functions that use as input daily average PM levels relate these to the daily incidence of the health endpoint. There are several variants of the short-term (daily) C-R function. Some C-R functions were estimated by using moving averages of ambient 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). Some C-R functions consider the relationship between daily incidence and daily average PM lagged a certain number of days. For example, a study might estimate the C-R relationship between mortality on day t and average PM on day (t-1). The discussion below does not depend on any particular averaging time or lag time and assumes only that the measure of health effect incidence, y, is consistent with the measure of ambient PM concentration, x.

The difference in health effects incidence, $\Delta y = y_0 - y$, from y_0 to the baseline incidence rate, y, corresponding to a given difference in ambient PM levels, $\Delta x = x_0 - x$, can be derived by dividing equation (2) by equation (1), which yields:

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

Alternatively, the difference in health effects incidence can be calculated indirectly using relative risk. Relative risk (RR) is a measure commonly used by epidemiologists to characterize 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 RR for mortality associated with the difference between the two ambient PM levels, x_0 and x. Given a C-R function of the form shown in equation (1) and a particular difference in ambient PM levels, Δx, the RR associated with that difference in ambient PM, denoted as $RR_{\Delta x}$, is equal to $e^{\beta \Delta x}$. The difference in health effects incidence, Δy, corresponding to a given difference in ambient PM levels, Δx, can then be calculated based on this RR:

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

Equations (3) and (4) are simply alternative ways of expressing the relationship between a given difference in ambient PM levels, Δx, and the corresponding difference in health effects incidence, Δy. These equations are the key equations that combine air quality information, C-R information, and baseline health effects incidence information to estimate ambient PM health risk.

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

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

Most daily time-series epidemiological studies estimated C-R functions in which the PM-related incidence on a given day depends only on same-day PM concentration or previous-day PM concentration (or some variant of those, such as a two-day average concentration). Such models necessarily assume that the longer pattern of PM levels preceding the PM concentration on a given day does not affect mortality on that day. To the extent that PM-related mortality on a given day is affected by PM concentrations over a longer period of time, then these models would be mis-specified, and this mis-specification would affect the predictions of daily incidence based on the model.

A few studies estimated distributed lag models, in which health effect incidence is a function of PM concentrations on several days – that is, the incidence of the health endpoint on day t is a function of the PM concentration on day t, day (t-1), day (t-2), and so forth. Such models can be reconfigured so that the sum of the coefficients of the different PM lags in the model can be used to predict the changes in incidence on several days. For example, corresponding to a change in PM on day t in a distributed lag model with 0-day, 1-day, and 2-day lags considered, the sum of the coefficients of the 0-day, 1-day, and 2-day lagged PM concentrations can be used to predict the sum of incidence changes on days t, (t+1) and (t+2).

The extent to which time-series studies using single-day PM concentrations may underestimate the relationship between short-term PM exposure and mortality is unknown; however, there is some evidence, based on analyses of PM_{10} data, that mortality on a given day is influenced by prior PM exposures up to more than a month before the date of death (Schwartz, 2000b). Where results from distributed lag models are available (e.g., PM_{10} mortality), we have included these results. Currently, there is insufficient information to adequately adjust for the impact of longer-term exposure on mortality associated with $PM_{2.5}$ and $PM_{10-2.5}$ exposures and this is an important uncertainty that should be kept in mind as one considers the results from the short-term exposure PM risk assessment.

The first and second parts of the risk assessment are distinguished primarily by the choice of lower PM level(s). When possible, the choice of lower PM level(s) in the first part of the risk

assessment was the lowest PM concentration observed in the study that estimated the C-R function used in the risk assessment. However, some of the short-term exposure PM studies do not report the lowest observed PM concentration. (For example, some studies instead report the lowest decile or quartile values.) When the lowest observed PM concentration is not available (or if it is lower than background level), analyses in the first part of the risk assessment considered the range of "as is" PM concentrations in the assessment location in excess of background PM concentration in that location. The second part considered the differences in health effects incidence associated with differences between "as is" $PM_{2.5}$ concentrations and $PM_{2.5}$ concentrations that just meet the current standards.[13]

In contrast to most short-term exposure studies, long-term exposure studies routinely report the lowest observed annual average PM concentration. The portion of the risk assessment that uses long-term exposure C-R functions therefore considered the difference between "as is" annual average PM in the assessment location and the lowest annual average PM level observed in the study (or background level, if that is higher), for the "as is" part of the analysis, or the annual average that would just meet the current $PM_{2.5}$ standards for the "risk reduction" part of the analysis.

In both parts of the risk assessment, 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 difference 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.

2.5.3 Calculating incidence on an annual basis

The risk assessment estimated health effects incidence, and changes in incidence, on an annual basis. For mortality, both short-term and long-term exposure studies have reported estimated C-R functions. As noted above, most short-term exposure C-R functions estimated by daily time-series epidemiological studies relate daily mortality to same-day PM concentration or previous-day PM concentration (or some variant of those).

To estimate the *daily* health impacts of daily average ambient PM levels above background or above the levels necessary to just meet the current $PM_{2.5}$ standards, C-R 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. (Alternative assumptions about the range of PM levels associated with health effects were explored in

[13]The next draft of this report will include PM risk estimates for just meeting alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards.

sensitivity analyses. Where a minimum concentration for effects (i.e., a hypothetical threshold) was considered, reductions below this concentration did not contribute attributable cases to the calculation. Only reductions down to this concentration contributed attributable cases to the calculation.)

After daily changes in health effects were calculated, an annual change was calculated by summing the daily changes. However, there are some days for which no ambient PM concentration information was available. The predicted annual risks, based on those days for which air quality data are available, were adjusted to take into account the full year. If days with missing air quality data occur randomly or relatively uniformly throughout the year, a simple adjustment can be made to the health effect incidence estimate – the incidence estimate based on the set of days with air quality data can be multiplied by the ratio of the total number of days in the year to the number of days in the year for which direct observations were available, to generate an estimate of the total annual incidence of the health effect.[14] However, if the missing data are not uniformly distributed throughout the year, such a simple adjustment could lead to a biased estimate of the total annual incidence change. To reduce such possible bias, adjustments were made on a quarterly basis. If Q_i is the total number of days in the ith quarter, and n_i is the number of days in the ith quarter for which there are air quality data, then the predicted incidence change in the ith quarter, based on those days for which there are air quality data, was multiplied by Q_i/n_i. The adjusted quarterly incidence changes were summed to derive an estimate of the annual incidence change.

Some short-term exposure C-R functions are based on average PM levels during several days. When such C-R functions were used, the air quality data were averaged for the same number of days. For example, a function based on two-day averages of PM was used in conjunction with two-day averages of PM in the assessment location to predict the incidence of the health effect in that location. In some cases, intervals of two or three consecutive days in a given location may be missing data, and so no multi-day average is available for use with multi-day C-R functions. These cases were treated by multi-day functions just as individual missing days were treated by single-day functions: they contributed no incidence change to the risk assessment, and incidence changes were adjusted for the days on which multi-day averages were missing.

C-R functions from long-term exposure studies (see Exhibit C.10) were used to assess the annual health impacts of changes in annual average ambient PM concentrations. Once again, to minimize the chance of bias due to differential amounts of missing data in different quarters of the year, quarterly averages were calculated based on the days in each quarter for which air

[14] This assumes that the distribution of PM concentrations on those days for which data are missing is essentially the same as the distribution on those days for which we have PM data.

quality data were available, and the "as is" annual average concentration was then calculated as an average of the four quarterly averages.

The mortality associated with long-term exposure is likely to include mortality related to short-term exposures as well as mortality related to longer-term exposures. As discussed previously, estimates of daily mortality based on the time-series studies also are likely influenced by prior PM exposures. Therefore, the estimated annual incidences of mortality calculated based on the short- and long-term exposure studies are not likely to be completely independent and should not simply be added together.

While we can characterize the statistical uncertainty surrounding the estimated PM coefficient in a reported C-R function, there are other sources of uncertainty about the C-R functions used in the risk assessment that are addressed via sensitivity analyses. The sources of uncertainty and how they are addressed in the risk assessment are discussed briefly below in Section 2.6 and in more detail in Section 6. Sensitivity analyses, which consider the impact of one assumption or source of uncertainty at a time, are listed in Section 2.7. All of the sensitivity analyses, described more fully in Section 7, focus on mortality.

2.6 Characterizing uncertainty and variability

Any estimation of "as is" risk and risk reductions associated with just meeting specified PM standards should address both the variability and uncertainty that generally underlie such an analysis. Uncertainty refers to the lack of knowledge regarding the actual values of model input variables (parameter uncertainty) and of physical systems or relationships (model uncertainty – e.g., the shapes of concentration-response functions). The goal of the analyst is to reduce uncertainty to the maximum extent possible. Uncertainty can be reduced by improved measurement and improved model formulation.

Variability refers to the heterogeneity in a population or parameter. Even if there is no uncertainty surrounding inputs to the analysis, there may still be variability. For example, there may be variability among C-R functions describing the relationship between PM and mortality across urban areas. This variability does not imply uncertainty about the C-R function in any of the urban areas, but only that these C-R functions are different in the different locations, reflecting differences in the populations and/or the PM. In general, it is possible to have uncertainty but no variability (if, for instance, there is a single parameter whose value is uncertain) or variability but little or no uncertainty (for example, people's heights vary considerably but can be accurately measured with little uncertainty).

The current risk assessment incorporates some of the variability in key inputs to the analysis by using location-specific inputs (e.g., location-specific C-R functions, baseline incidence rates, and air quality data). Although spatial variability in these key inputs across all

U.S. locations has not been fully characterized, variability across the selected locations is imbedded in the analysis by using, to the extent possible, inputs specific to each urban area. Temporal variability is more difficult to address, because the risk assessment focuses on some unspecified time in the future. To minimize the degree to which values of inputs to the analysis may be different from the values of those inputs at that unspecified time, we have used the most current inputs available – for example, year 2002 air quality data for most of the urban locations, and the most recent available mortality baseline incidence rates (from 1998). However, we have not tried to predict future changes in inputs (e.g., future population levels or possible changes in baseline incidence rates).

There are a number of important sources of uncertainty that were addressed where possible. The following are among the major sources of uncertainty in the risk assessment:

- Uncertainties related to estimating the C-R functions, including the following:
  - There is uncertainty about the extent to which the association between PM and the health endpoint actually reflects a causal relationship.
  - There is uncertainty surrounding estimates of PM coefficients in C-R functions used in the analyses.
  - There is uncertainty about the specification of the model (including the shape of the C-R relationship), particularly whether or not there are thresholds below which no response occurs.
  - There is uncertainty related to the transferability of PM C-R functions from study locations and time periods to the locations and time periods selected for the risk assessment.[15] A C-R function in a study location may not provide an accurate representation of the C-R relationship in the analysis location(s) and time periods because of
    - variations in PM composition across cities or over time,
    - the possible role of associated co-pollutants, which vary from location to location and over time, in influencing PM risk,
    - variations in the relationship of total ambient exposure (both outdoor and ambient contributions to indoor exposure) to ambient monitoring in different locations (e.g, due to differences in air conditioning use in different regions of the U.S. or changes in usage over time),

[15] The risk assessment locations were selected partly on the basis of where C-R functions were estimated, specifically to reduce this important source of uncertainty. Therefore, possible differences due to location is a source of uncertainty in the risk assessment only when C-R functions from multi-city studies or from another location are applied to a risk assessment location.

- differences in population characteristics (e.g., the proportions of members of sensitive subpopulations) and population behavior patterns across locations or over time in the same location.

- Uncertainties related to the air quality adjustment procedure that was used to simulate just meeting the current PM standards, and uncertainties about estimated background concentrations for each location.

- Uncertainties associated with use of baseline health effects incidence information that is not specific to the analysis locations.[16]

The uncertainties from some of these sources -- in particular, the statistical uncertainty surrounding estimates of the PM coefficients in C-R functions -- were characterized quantitatively. It was possible, for example, to calculate confidence intervals around risk estimates based on the uncertainty associated with the estimates of PM coefficients used in the risk assessment. These confidence intervals express the range within which the risks are likely to fall if the uncertainty surrounding PM coefficient estimates were the only uncertainty in the analysis. There are, of course, several other uncertainties in the risk assessment, as noted above. If there were sufficient information to quantitatively characterize these sources of uncertainty, they could be included in a Monte Carlo analysis to produce confidence intervals that more accurately reflect all sources of uncertainty.

We handled uncertainties in the risk assessment in several ways:

- Limitations and assumptions in estimating risks and risk reductions are clearly stated and explained.

- The uncertainty resulting from the statistical uncertainty associated with the estimate of the PM coefficient in a C-R function was characterized by confidence intervals around the corresponding point estimate of risk. As noted above, such a confidence interval expresses the range within which the true risk is likely to fall if the uncertainty surrounding the PM coefficient estimate were the only uncertainty in the analysis. It does

[16] Location-specific baseline incidence rates were obtainable for most health endpoints. The only health endpoints for which this was not the case are respiratory symptoms, for which baseline incidence rates were reported in the studies. For those studies carried out in a single location, this provides location-specific baseline incidence rates. For Schwartz and Neas (2000), the rates were based on six cities combined. Boston and St. Louis, the two assessment locations where these endpoints are evaluated, were two of the six cities.

not, for example, reflect the uncertainty concerning whether the PM coefficients in the study location and the assessment location are the same.[17]

- Sensitivity analyses were conducted to illustrate the effects of changing key default assumptions on the results of the assessment, and quantitative comparisons were presented to inform other analytic choices.[18]

2.7 Summary of key assumptions and sensitivity analyses

In summary, the key assumptions on which the PM risk assessment is based include the following:

- The relationship between PM components examined and health endpoints is causal;
- C-R models are appropriately specified – e.g., the functional forms are correctly specified (including the lack of a threshold), and the lag structure is correctly specified;
- Baseline incidence rates have not changed appreciably from those used in the risk assessment;
- Population sizes and age distributions have not changed appreciably from those used in the risk assessment;
- The distribution of PM concentrations on missing days is essentially the same as the distribution of PM concentrations on days for which we have PM data;
- The estimated background concentration for each component is appropriate for each urban area in the analysis;
- The background concentration for each component is essentially constant across the days in a year;
- A single year of air quality data is appropriate to characterize risks associated with as is and just meeting specified standards,
- Proportional rollback of concentrations over estimated background appropriately represents how standards would be just met;

Sensitivity analyses are 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

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[17] This is not an uncertainty, of course, if the C-R function has been estimated in the assessment location.

[18] "Sensitivity analyses" refers to assessing the effects of uncertainty on some of the final risk estimates; "quantitative comparisons" refer to numerical comparisons (e.g. comparisons of monitor values) that are not carried that far.

preferable for illustrating the impact on results of using different input component values. Exhibit 2.5 lists the sensitivity analyses that were conducted.

Exhibit 2.5 Sensitivity Analyses and Quantitative Comparisons

Analysis Number (Exhibit 2.1)	PM Indicator	Component of the Risk assessment	Sensitivity Analysis or Comparison
1	$PM_{2.5}$	Air Quality	A sensitivity analysis of the effect of assuming a constant background PM level versus different daily background levels
2	$PM_{2.5}$, PM_{10}, $PM_{10-2.5}$	Air Quality	A sensitivity analysis of the effect of assuming different (constant) background PM levels
3	$PM_{2.5}$	Air Quality	A sensitivity analysis of the effect of an alternative air quality adjustment procedure on the estimated risk reductions resulting from just meeting the current 24-hr and annual $PM_{2.5}$ standards
4	$PM_{2.5}$	Air Quality	A comparison of the effect of just meeting the current annual $PM_{2.5}$ Standard of 15 $\mu g/m^3$ using the maximum versus the average of monitor-specific averages
5	$PM_{2.5}$, PM_{10}, $PM_{10-2.5}$	Concentration-Response	A sensitivity analysis using an approach to estimate the possible impact of using a distributed lag C-R function
6	$PM_{2.5}$	Concentration-Response	A sensitivity analysis of the impact on mortality associated with long-term exposure of different assumptions about the role of historical air quality concentrations in contributing to the reported effects
7	$PM_{2.5}$, PM_{10}, $PM_{10-2.5}$	Concentration-Response	A sensitivity analysis assuming alternative hypothetical threshold concentration levels for the occurrence of PM-related response at concentrations above those for background

3. Health Endpoints, Urban Areas, and Studies Selected

As discussed in the 2003 draft PM CD, a significant number of epidemiological studies examining a variety of health effects associated with ambient PM concentrations in various locations throughout the United States and Canada have been published since the last NAAQS review. As a result of the availability of additional health effects studies and air quality information, EPA expanded the geographic scope of the PM risk assessment to include several additional urban areas beyond the two (Philadelphia and Los Angeles Counties) analyzed for the last review, consistent with the goals of the assessment. The approaches that were used to select health endpoint categories, urban areas, and studies to include in the PM risk assessment are discussed below.

3.1 Health endpoints

OAQPS staff carefully reviewed the evidence evaluated in the 2003 draft PM CD. Tables 9-8 and 9-10 in the 2003 draft PM CD summarize the available U.S. and Canadian short-term exposure studies that provide effect estimates for PM (i.e., $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$) for mortality and morbidity, respectively. Table 9-11 summarizes the available U.S. and Canadian studies that provide effect estimates for $PM_{2.5}$ and other PM indicators for long-term exposure. Given the large number of endpoints and studies addressing PM effects, EPA included in the quantitative PM risk assessment only the more severe and better understood (in terms of health consequences) health endpoint categories for which the weight of the evidence supports the assumption of a causal relationship between PM and the effect category. In addition, EPA included only those categories for which there were studies that satisfy the study selection criteria (see Section 3.3 below).

For those health effect categories included, the risk assessment is predicated on the assumption that a causal relationship exists. As discussed in more detail in the draft PM CD (EPA, 2003a, p.9-69 - 9-70),

> "...although associations of PM with harmful effects continue to be observed consistently across most new studies, the newer findings do not fully resolve issues concerning relative contributions to the observed epidemiologic associations of (a) PM acting alone, (b) PM acting in combination with gaseous co-pollutants, (c) the gaseous pollutants per se, and (d) the overall ambient pollutant mix.
>
> "It is possible that, for pollutants whose ambient concentrations are not highly correlated, effects estimates in multipollutant models could be more biologically and epidemiologically sound than those in single-pollutant models, although single-pollutant models could also be credible if independent biological plausibility evidence supported

designation of PM or some other single pollutant as likely being the key toxicant in the ambient pollutant mix evaluated. Because neither of these possibilities have been definitively demonstrated and there is not yet full scientific consensus as to optimal interpretation of modeling outcomes for time series-air pollution studies, the choice of appropriate effects estimates to employ in risk assessments for ambient PM effects remains a difficult issue. Issues related to confounding by co-pollutants, along with issues related to time scales of exposure and response and concentration-response function, still apply to new epidemiologic studies relating concentrations of PM or correlated ambient air pollutants to hospital admissions, exacerbation of respiratory symptoms, asthma in children, reduced pulmonary function in children and adults, and to changes in heart rate and heart rate variability in adults. However, with considerable new experimental evidence now in hand, it is possible to hypothesize various ways in which ambient exposure to PM acting alone or in combination with other co-pollutants can plausibly be involved in the complex chain of biological events leading to harmful health effects in the human population. This newer experimental evidence, coupled with new exposure analyses results, adds much support for interpreting the epidemiologic findings discussed here as likely being indicative of causal relationships between exposures to ambient PM (or specific size or chemical components) and consequent associated increased mortality and morbidity effects."

The 2003 draft PM CD also notes that "considerable coherence exists across newly available epidemiological study findings" (p. 9-127). New evidence related to several hypotheses about possible mechanisms by which ambient PM may exert human health effects (see Section 9.7, draft PM CD), tend "to support the likelihood of a causal relationship between low ambient concentrations of PM and increased mortality or morbidity risks observed in human population studies" (p.9-67). As stated in the 2003 draft PM CD, "much still remains to be done, however, to identify more confidently specific causal agents among typical ambient PM constituents." With regard to short-term exposure, there have been more than 80 new time-series PM mortality studies since the last PM review. "With only a few exceptions, the estimated mortality RR's in these studies are generally positive, many are statistically significant, and they generally comport well with previously reported PM-mortality effects estimates" cited in the 1996 PM CD (EPA, 2003a, p. 9-75). In addition, there are now "numerous additional studies demonstrating associations between short-term (24-h) PM exposures and various morbidity endpoints" (EPA, 2003a, p.9-75). Similarly, the 2003 draft PM CD concludes that the evidence linking long-term exposure to ambient $PM_{2.5}$ and premature mortality has been substantiated further since the last PM review. The 2003 draft PM CD states that "the Harvard Six Cities analyses (as confirmed by the HEI analyses) and the recent extensions of the ACS study by Pope et al. (2002) probably provide the most credible and precise estimates of excess mortality risk associated with long-term PM exposures in the United States" (p. 9-125).

Based on its review of the evidence evaluated in the 2003 draft PM CD, OAQPS included in the risk assessment for all three PM indicators the following broad categories of health endpoints associated with short-term exposures:

- non-accidental total, cardiovascular and respiratory mortality;[19]
- hospital admissions for cardiovascular and respiratory causes;[20] and
- respiratory symptoms not requiring hospitalization.

In addition, total, cardiopulmonary, and lung cancer mortality due to long-term exposure are included in the $PM_{2.5}$ risk assessment. Other effects reported to be associated with PM, such as decreased lung function and changes in heart rate variability, are discussed in the draft OAQPS PM Staff Paper (EPA, 2003b).

3.2 Urban areas

In the prior risk assessment the selection of urban areas to include was determined largely by the very limited availability of recent and sufficiently complete $PM_{2.5}$ ambient air quality data. For the current PM risk assessment, there was a significantly greater number of candidate locations in which epidemiological studies have reported C-R relationships and for which there are sufficient PM ambient air quality data. Recent evidence from the National Mortality and Morbidity Air Pollution Study (NMMAPS) (Samet et al., 2001) suggests there is geographic variability in C-R relationships across many U.S. urban areas. In light of the evidence from NMMAPS, which examined C-R relationships across the 90 largest U.S. cities, we identified candidate areas for the PM risk assessment emphasizing geographically varied urban areas in the United States in which C-R relationships have been estimated.

An urban area in the United States was included in the PM risk assessment only if it satisfied the following criteria:

- It has sufficient air quality data for a recent year (1999 or later). A city was considered to have sufficient PM_{10} air quality data if it had at least one PM_{10} monitor at which there were at least 11 observations per quarter for a one year period. Because there are substantially more monitoring data for $PM_{2.5}$ than for PM_{10}, we added the additional criterion for $PM_{2.5}$ that there be at least 122 observations per year (1 in 3 day monitoring).

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[19] Although mortality effects for $PM_{10-2.5}$ were not included in OAQPS's proposed risk methodology plan, such effects have been added consistent with the advice from the CASAC PM Panel.

[20] The category of emergency room visits was also considered, but there is evidence that baseline incidence rates vary considerably across locations, and location-specific rates were not available. Therefore this health effect was not included.

Sufficient air quality data for $PM_{10-2.5}$ was defined as a one year period with at least 11 daily values per quarter based on data from co-located PM_{10} and $PM_{2.5}$ monitors.[21]

- It is the same as or close to the location where at least one C-R function for one of the recommended health endpoints (see above) has been estimated by a study that satisfies the study selection criteria (see below).[22]
- For the hospital admission effects category, relatively recent area-specific baseline incidence data, specific to International Classification of Disease (ICD) codes, are available.[23]

3.2.1 Additional considerations: the $PM_{2.5}$ risk assessment

The largest data base for health effects associated with short-term (i.e., 24-hour) ambient $PM_{2.5}$ concentrations, in terms of number of studies in different locations, is for non-accidental total and cause-specific mortality. Therefore, OAQPS focused on selecting urban areas for the $PM_{2.5}$ risk assessment primarily on this health effect category supplemented by consideration of morbidity endpoints. We first reviewed the studies listed in Table 9-8 of the 2003 draft PM CD that estimated C-R functions for short-term exposure mortality in U.S. locations and used measured $PM_{2.5}$ or $PM_{2.5}$ estimated by nephelometry as the air quality indicator. A candidate pool of sixteen urban areas in the U.S. was represented among those studies.

We next considered the precision of the effect estimates from those short-term exposure mortality studies identified in the first step.[24] In general, the relative precision of a study increases as the number of its observations increases. The number of observations depends not only on the number of days on which mortality counts were obtained, but also on the size of the mortality counts. The 2003 draft PM CD uses the natural logarithm of the mortality-days (i.e., the natural log of the product of the number of study days and the average number of deaths per day) as a surrogate or indicator reflecting the relative weight of short-term exposure mortality

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[21]To be consistent with the epidemiological studies which generally focus on using only population-oriented monitors, we excluded from consideration any monitors where the monitoring objective was listed as "highest concentration monitor." The few monitors that were excluded are sited in industrial or commercial areas and are intended to characterize local conditions near major point sources.

[22] Urban locations for which C-R functions were estimated often include several counties. (For example, in Klemm et al., 2000, the urban area labeled "Boston" consists of three counties: Middlesex, Norfolk, and Suffolk counties.) To the extent possible, in the PM risk assessment we tried to include the specific counties used in the urban location in the original epidemiological studies.

[23] The absence of hospital admissions baseline incidence data does not necessarily mean that we cannot use an urban area in the risk assessment, only that we cannot use it for the hospital admissions endpoint.

[24] Tolbert et al. (2000) was excluded from consideration because it presented only preliminary results, and the 2003 draft PM CD urged caution in interpreting these preliminary results.

epidemiological studies as an indicator of likely increasing precision for study effect estimates. We considered only those urban areas in which studies with relatively greater precision were conducted – specifically, studies that have a natural log of mortality-days greater than or equal to 9.0 for total non-accidental mortality.[25] This criterion excluded 6 urban areas (Camden, NJ; Coachella Valley, CA; Elizabeth, NJ; Newark, NJ; Steubenville, OH; and Topeka, KS).

We next considered which of those study locations also have sufficient $PM_{2.5}$ monitoring data to support a $PM_{2.5}$ risk assessment. Using the completeness criterion defined above for $PM_{2.5}$, 2 additional areas were excluded (Knoxville, TN and Portage, WI), leaving eight cities in which epidemiological studies reported C-R relationships for $PM_{2.5}$ and mortality and which had sufficient air quality data in a recent year.

The following urban areas satisfied the criteria of availability of C-R functions for short-term exposure mortality, study precision, and availability of sufficiently recent and complete air quality data to be included in the $PM_{2.5}$ risk assessment for short-term exposure mortality:

- Boston, MA
- Detroit, MI
- Los Angeles, CA
- Philadelphia, PA
- Phoenix, AZ
- Pittsburgh, PA
- San Jose, CA
- St. Louis, MO

Because baseline mortality incidence data are available at the county level, this was not a limiting factor in the selection of urban areas for any portion of the PM risk assessment.

The long-term exposure C-R functions used in the $PM_{2.5}$ risk assessment are based on studies involving multiple cities across the United States, and the estimated C-R functions are based on differences in long-term averages observed across the various cities. The issue of matching a risk assessment location with the specific location in which a C-R function was estimated therefore does not arise for long-term exposure mortality in quite the way it does for short-term exposure mortality. We carried out the $PM_{2.5}$ risk assessment for long-term exposure mortality in all the urban locations listed above that are included in the $PM_{2.5}$ risk assessment. Although Seattle is included in the $PM_{2.5}$ risk assessment because of morbidity endpoints (see below), it was not included among the locations for which we carried out the $PM_{2.5}$ risk

[25]Most of the epidemiological studies reporting total non-accidental mortality also report on one or more cause specific mortality categories; in such studies the natural log of mortality days is often less than 9.0 because there are fewer deaths from a specific cause. We included the cause-specific mortality C-R relationships reported in such studies as long as the natural log of total mortality days was greater than or equal to 9.0.

assessment for long-term exposure mortality because Seattle's annual average $PM_{2.5}$ was lower than the LMLs in all three long-term exposure studies (so that the estimated risks would be zero, because we are not estimating risks for concentrations below the LML).

Most of the urban locations in which C-R functions were estimated for health endpoints other than mortality are included in the set of locations available for mortality. A primary consideration in selecting urban locations for these other health endpoints, as with the $PM_{2.5}$ risk assessment for mortality, was that the assessment locations be the same as or close to the study locations where C-R functions were estimated. Second, studies with relatively greater precision were considered preferable. In addition, for the hospital admission effect category, we limited our selection of urban areas to those for which the necessary baseline incidence data were available.

3.2.2 Additional considerations: the PM_{10} risk assessment

In addition to the general selection criteria listed above, the selection of urban areas to include in the PM_{10} risk assessment was further guided by the following considerations:

- Among its comments on EPA's plans to conduct the $PM_{2.5}$ risk assessment, the CASAC recommended that EPA expand its PM risk assessment for the current review to include PM_{10} and to select cities across various parts of the United States.

- In addition, we wanted to include urban areas that would further inform comparisons both across the PM indicators (i.e., $PM_{2.5}$, $PM_{10\text{-}2.5}$, PM_{10}) and across health effects (e.g., mortality, hospital admissions).

- In light of these recommendations and goals, we wanted to include, at a minimum, those urban areas already selected for the $PM_{2.5}$ risk assessment (i.e., Boston, Detroit, Los Angeles, Philadelphia, Phoenix, Pittsburgh, San Jose, Seattle, and St. Louis), for which city-specific C-R functions for short-term exposure mortality are available from the NMMAPS study and/or other studies.

- Finally, in selecting additional urban areas, as with the $PM_{2.5}$ risk assessment, areas for which there are C-R functions available from studies with relatively greater precision were preferred.

Based on the above criteria and considerations, we included the following urban areas in the PM_{10} risk assessment:

- Boston, MA
- Chicago, IL

- Detroit, MI
- Los Angeles, CA
- Minneapolis-St. Paul, MN
- Philadelphia, PA
- Phoenix, AZ
- Pittsburgh, PA
- Provo, UT
- San Jose, CA
- Seattle, WA
- St. Louis, MO

Most of these urban areas allow comparison both across PM indicators and across different health endpoints. While Chicago, Minneapolis-St. Paul, and Provo do not provide comparisons across PM indicators, they do provide comparisons across health endpoints.

3.2.3 Additional considerations: the $PM_{10-2.5}$ risk assessment

We wanted to include urban areas in the $PM_{10-2.5}$ risk assessment for which we were also conducting a $PM_{2.5}$ risk assessment, if there are epidemiological studies reporting associations for $PM_{10-2.5}$ in these locations. Because the $PM_{10-2.5}$ risk assessment requires air quality data for PM_{10} and $PM_{2.5}$ at co-located monitors, the criterion of sufficient air quality data is significantly more limiting in the selection of urban areas for the $PM_{10-2.5}$ risk assessment than for either the PM_{10} or the $PM_{2.5}$ risk assessment.

Based on these considerations, we included Detroit, Philadelphia, Seattle, and St. Louis in the $PM_{10-2.5}$ risk assessment. Although there were no co-located PM_{10} and $PM_{2.5}$ AIRS monitors for a recent year in Phoenix, we obtained data from 1997 from co-located PM_{10} and $PM_{2.5}$ TEOM monitors; because these data were from TEOM monitors rather than AIRS monitors, we included Phoenix as a special case. While sufficient air quality data are also available for Los Angeles, the relevant epidemiological study used the S-Plus/GAM procedure but has not yet been re-analyzed.

3.3 Studies

A study that has estimated one or more C-R functions for a health endpoint in an urban location to be used for the $PM_{2.5}$, $PM_{10-2.5}$, and/or PM_{10} risk assessment had to satisfy the following criteria:

- It is an acceptable, published, peer-reviewed study that has been evaluated in the 2003 draft PM CD.

- It directly measured PM using $PM_{2.5}$, $PM_{10-2.5}$, or PM_{10} as the indicator or for $PM_{2.5}$ was estimated using nephelometry data.[26]

- It either did not rely on GAMs using the S-Plus software to estimate C-R functions or has appropriately re-estimated them using revised methods.

3.4 A summary of health endpoints, urban areas, and studies selected

Based on applying the criteria and considerations discussed above, the health endpoints and the urban locations that were selected, as well as the studies that estimated the C-R functions used in the PM risk assessment are given in Exhibits 3.1 - 3.3 for $PM_{2.5}$, Exhibits 3.4 and 3.5 for PM_{10}, and Exhibits 3.6 and 3.7 for $PM_{10-2.5}$. More detailed information on the studies used is given in Appendix C.

[26] Although not included in the draft risk methodology plan (Abt, 2002), consistent with advice received from members of the CASAC PM Panel, we have added studies that used nephelometry to estimate $PM_{2.5}$ concentrations where gravimetric measurements were not available.

Exhibit 3.1 The $PM_{2.5}$ Risk Assessment: Mortality Associated with Short-Term Exposure				
Urban Location	**Total (non-accidental)**	**Cardiovascular**	**Circulatory**	**Respiratory**
Boston, MA	Schwartz et al. (1996)[A] *	Klemm et al. (2000)[B] – ischemic heart disease *		Klemm et al. (2000)[B] – COPD *, pneumonia *
Detroit, MI	Lippmann et al. (2000)[C]		Lippmann et al. (2000)[C]	Lippmann et al. (2000)[C]
Los Angeles, CA	Moolgavkar (2000a)[D]	Moolgavkar (2000a)[D]		Moolgavkar (2000a)[D] – COPD
Philadelphia, PA	Lipfert et al. (2000)	Lipfert et al. (2000) *		
Phoenix, AZ		Mar et al. (2000)[E]		
Pittsburgh, PA	Chock et al. (2000)			
San Jose, CA	Fairley (1999)[F]	Fairley (1999)[F]		Fairley (1999)[F]
St. Louis, MO	Schwartz et al. (1996)[A]	Klemm et al. (2000)[B] – ischemic heart disease *		Klemm et al. (2000)[B] – COPD *, pneumonia *

*Includes a multi-city or multi-county C-R function

[A] Reanalyzed in Schwartz (2003b)

[B] Reanalyzed in Klemm and Mason (2003)

[C] Reanalyzed in Ito (2003)

[D] Reanalyzed in Moolgavkar (2003)

[E] Reanalyzed in Mar et al. (2003)

[F] Reanalyzed in Fairley (2003)

Exhibit 3.2 The $PM_{2.5}$ Risk Assessment: Mortality Associated with Long-Term Exposure			
Urban Location*	**Total**	**Cardiopulmonary**	**Lung Cancer**
Boston, MA	Krewski et al. (2000) – 6 cities Krewski et al. (2000) – ACS Pope et al. (2002) – ACS extended	Krewski et al. (2000) – 6 cities Krewski et al. (2000) – ACS	Pope et al. (2002) – ACS extended
Detroit, MI	Krewski et al. (2000) – ACS Pope et al. (2002) – ACS extended	Krewski et al. (2000) – ACS	Pope et al. (2002) – ACS extended
Los Angeles, CA	Krewski et al. (2000) – ACS Pope et al. (2002) – ACS extended	Krewski et al. (2000) – ACS	Pope et al. (2002) – ACS extended
Philadelphia, PA	Krewski et al. (2000) – ACS Pope et al. (2002) – ACS extended	Krewski et al. (2000) – ACS	Pope et al. (2002) – ACS extended
Phoenix, AZ	Krewski et al. (2000) – ACS Pope et al. (2002) – ACS extended	Krewski et al. (2000) – ACS	Pope et al. (2002) – ACS extended
Pittsburgh, PA	Krewski et al. (2000) – ACS Pope et al. (2002) – ACS extended	Krewski et al. (2000) – ACS	Pope et al. (2002) – ACS extended
San Jose, CA	Krewski et al. (2000) – ACS Pope et al. (2002) – ACS extended	Krewski et al. (2000) – ACS	Pope et al. (2002) – ACS extended
St. Louis, MO	Krewski et al. (2000) – 6 cities Krewski et al. (2000) – ACS Pope et al. (2002) – ACS extended	Krewski et al. (2000) – 6 cities Krewski et al. (2000) – ACS	Pope et al. (2002) – ACS extended

*Seattle's annual average $PM_{2.5}$ level was lower than the LMLs of all three long-term exposure mortality studies. Seattle was not included in this part of the risk assessment, since its risks would be estimated to be zero, because we are not estimating risks for concentrations below the LML.

Exhibit 3.3 The $PM_{2.5}$ Risk Assessment: Morbidity Associated with Short-Term Exposure			
Urban Location	**Cardiovascular Hospital Admissions**	**Respiratory Hospital Admissions**	**Respiratory Symptoms**
Boston, MA			Schwartz and Neas (2000) – cough, lower respiratory symptoms (LRS)
Detroit, MI	Lippmann et al. (2000)[A] – ischemic heart disease, congestive heart failure, dysrhythmias	Lippmann et al. (2000)[A] – pneumonia, COPD	
Los Angeles, CA	Moolgavkar (2000b)[B]	Moolgavkar (2000c)[B] – COPD	
Seattle, WA		Sheppard et al. (1999)[C] – asthma	
St. Louis, MO			Schwartz and Neas (2000) – cough, LRS

[A] Reanalyzed in Ito (2003)

[B] Reanalyzed in Moolgavkar (2003)

[C] Reanalyzed in Sheppard (2003)

Exhibit 3.4 The PM_{10} Risk Assessment: Mortality Associated with Short-Term Exposure					
Urban Location	**Total (non-accidental or all cause)**	**Cardiovascular**	**Cardiorespiratory**	**Circulatory**	**Respiratory**
Boston, MA	Klemm et al. (2000)[A] * Samet et al. (2000)[B] *		Samet et al. (2000)[B] *		
Chicago, IL	Schwartz (2000b)[C]* Samet et al. (2000)[B,C] * Styer et al. (1995) Ito and Thurston (1996) Moolgavkar (2000a)[D] Schwartz (2000a)[C]*	Moolgavkar (2000a)[D] Braga et al. (2001)[C] *	Samet et al. (2000)[B] *	Ito and Thurston (1996)	Ito and Thurston (1996) Moolgavkar (2000a)[D] – COPD Braga et al. (2001)[C] – COPD *, pneum. *
Detroit, MI	Schwartz (2000b)[C]* Schwartz (2000a)[C]* Samet et al. (2000)[B,C] * Lippmann et al. (2000)[E]	Braga et al. (2001)[C] *	Samet et al. (2000)[B] *	Lippmann et al. (2000)[E]	Lippmann et al. (2000)[E] Braga et al. (2001)[C] – COPD * pneum. *
Los Angeles, CA	Samet et al. (2000)[B] * Kinney et al. (1995) Moolgavkar (2000a)[D]	Moolgavkar (2000a)[D]	Samet et al. (2000)[B] *		Moolgavkar (2000a)[D] – COPD
Minneapolis, MN	Schwartz (2000b)[C] * Schwartz (2000a)[C] * Samet et al. (2000)[B,C] *	Braga et al. (2001)[C] *	Samet et al. (2000)[B] *		Braga et al. (2001)[C] – COPD * pneum. *
Philadelphia, PA	Samet et al. (2000)[B] * Lipfert et al. (2000)	Lipfert et al. (2000) *	Samet et al. (2000)[B] *		
Phoenix, AZ	Samet et al. (2000)[B] *	Mar et al. (2000)[F]	Samet et al. (2000)[B] *		

Exhibit 3.4 The PM_{10} Risk Assessment: Mortality Associated with Short-Term Exposure					
Urban Location	**Total (non-accidental or all cause)**	**Cardiovascular**	**Cardiorespiratory**	**Circulatory**	**Respiratory**
Pittsburgh, PA	Schwartz (2000b)[C] * Schwartz (2000a)[C] * Samet et al. (2000)[B,C] * Chock et al. (2000)	Braga et al. (2001)[C] *	Samet et al. (2000)[B] *		Braga et al. (2001)[C] – COPD * pneum. *
Provo, UT	Samet et al. (2000)[B] **				
San Jose, CA	Samet et al. (2000)[B] * Fairley (1999)[G]	Fairley (1999)[G]	Samet et al. (2000)[B] *		Fairley (1999)[G]
Seattle, WA	Schwartz (2000b)[C] * Schwartz (2000a)[C] * Samet et al. (2000)[B,C] *	Braga et al. (2001)[C] *	Samet et al. (2000)[B] *		Braga et al. (2001)[C] – COPD * pneum. *
St. Louis, MO	Klemm et al. (2000)[A] * Samet et al. (2000)[B] *		Samet et al. (2000)[B] *		

*Includes multi-county, multi-city, regional, or national C-R function

**C-R function was estimated for Salt Lake City, UT, which is close to Provo.

[A] Reanalyzed in Klemm and Mason (2003)

[B] Reanalyzed in Dominici et al. (2003)

[C] Reanalyzed in Schwartz (2003a)

[D] Reanalyzed in Moolgavkar (2003)

[E] Reanalyzed in Ito (2003)

[F] Reanalyzed in Mar et al. (2003)

[G] Reanalyzed in Fairley (2003)

Exhibit 3.5 The PM_{10} Risk Assessment: Morbidity Associated with Short-Term Exposure			
Urban Location	**Cardiovascular Hospital Admissions**	**Respiratory Hospital Admissions**	**Respiratory Symptoms**
Boston, MA			Schwartz et al. (1994) – Lower respiratory symptoms (LRS), upper respiratory symptoms (URS)
Chicago, IL	Samet et al. (2000)[A] * Moolgavkar (2000b)[B] Morris and Naumova (1998) – congestive heart failure	Samet et al. (2000)[A] – pneumonia, COPD * Moolgavkar (2000c)[B] – COPD	
Detroit, MI	Samet et al. (2000)[A] * Schwartz and Morris (1995) – Congestive heart failure, Ischemic heart disease, Dysrhythmias Lippmann et al. (2000)[C] – Congestive heart disease, Ischemic heart disease Dysrhythmias	Samet et al. (2000)[A] – pneumonia, COPD * Lippmann et al. (2000)[C] – Pneumonia, COPD Schwartz (1994a) – pneumonia, COPD	
Los Angeles, CA	Moolgavkar (2000b)[B] Linn et al. (2000)	Moolgavkar (2000c)[B] – COPD Linn et al. (2000) – respiratory, COPD, asthma Nauenberg and Basu (1999) – asthma	
Minneapolis, MN	Samet et al. (2000)[A] *	Samet et al. (2000)[A] – pneumonia, COPD * Schwartz (1994b) – pneumonia, COPD	
Pittsburgh, PA	Samet et al. (2000)[A] *	Samet et al. (2000)[A] – pneumonia, COPD *	

Exhibit 3.5 The PM_{10} Risk Assessment: Morbidity Associated with Short-Term Exposure			
Urban Location	**Cardiovascular Hospital Admissions**	**Respiratory Hospital Admissions**	**Respiratory Symptoms**
Provo, UT	Samet et al. (2000)[A] *	Samet et al. (2000)[A] – pneumonia, COPD *	Pope et al. (1991) – LRS, upper respiratory symptoms (URS)
Seattle, WA	Samet et al. (2000)[A] *	Samet et al. (2000)[A] – pneumonia, COPD * Sheppard et al. (1999)[D] – asthma	Yu et al. (2000) – asthma
St. Louis, MO			Schwartz et al. (1994) – LRS, URS

*Includes multi-city, regional, or national C-R function

[A] Reanalyzed in Zanobetti and Schwartz (2003)

[B] Reanalyzed in Moolgavkar (2003)

[C] Reanalyzed in Ito (2003)

[D] Reanalyzed in Sheppard (2003)

Exhibit 3.6 The $PM_{10-2.5}$ Risk Assessment: Mortality Associated with Short-Term Exposure				
Urban Location	**Total (non-accidental or all cause)**	**Cardiovascular**	**Circulatory**	**Respiratory**
Detroit, MI	Lippmann et al. (2000)[A]		Lippmann et al. (2000)[A]	Lippmann et al. (2000)[A]
Philadelphia, PA	Lipfert et al. (2000)	Lipfert et al. (2000) *		
Phoenix, AZ		Mar et al. (2000)[B]		
St. Louis, MO	Schwartz et al. (1996)[C]			

*Includes multi-county, multi-city, regional, or national C-R function

[A] Reanalyzed in Ito (2003)

[B] Reanalyzed in Mar et al. (2003)

[C] Reanalyzed in Schwartz (2003b)

Exhibit 3.7 The $PM_{10-2.5}$ Risk Assessment: Morbidity Associated with Short-Term Exposure			
Urban Location	**Cardiovascular Hospital Admissions**	**Respiratory Hospital Admissions**	**Respiratory Symptoms**
Detroit, MI	Lippmann et al. (2000)[A] – Congestive heart disease, Ischemic heart disease Dysrhythmias	Lippmann et al. (2000)[A] – Pneumonia, COPD	
Seattle, WA		Sheppard et al. (1999)[B] – asthma	
St. Louis, MO			Schwartz and Neas (2000) – LRS, cough

*Includes multi-city, regional, or national C-R function

[A] Reanalyzed in Ito (2003)

[B] Reanalyzed in Sheppard (2003)

4. Selecting Concentration-Response Functions

For the most part, the selection of studies from which to draw C-R relationships for the PM risk assessment was determined by the choice of health endpoints to include in the analyses and by the process used to select the urban areas, discussed in the previous section. C-R functions that were not statistically significant were included if the overall weight of the evidence from the collective body of studies supported the conclusion that there was a likely causal relationship between PM and the health endpoint under consideration. The C-R functions of interest for the PM risk assessment are from epidemiological studies investigating the relations between PM and a variety of health endpoints. Both single-pollutant, and where available, multi-pollutant C-R functions used in the PM risk assessment were obtained for the studies listed in Tables 9-8, 9-10, and 9-11 of the 2003 draft PM CD that met the criteria discussed previously in Section 3. Some of these studies were used in the prior (1996) PM risk assessment (Abt Associates Inc, 1996). As noted earlier, the selection of studies is preliminary and will be reviewed after comments are received from the CASAC and the general public on the 2003 draft PM CD.

Studies often report more than one estimated C-R function for the same location and health endpoint. Sometimes models including different sets of co-pollutants are estimated in a study; sometimes different lags are estimated. In some cases, two or more different studies estimated a C-R function for PM and the same health endpoint (this is the case, for example, with $PM_{2.5}$ and long-term exposure mortality).

4.1 Single and multi-city functions

All else being equal, a C-R function estimated in the assessment location is preferable to a function estimated elsewhere since it avoids uncertainties related to potential differences due to geographic location. That is why the urban areas selected for the PM risk assessment were those locations in which C-R functions have been estimated. There are several advantages, however, to using estimates from multi-city studies versus studies carried out in single cities. Multi-city studies are applicable to a variety of settings, since they estimate a central tendency across multiple locations. When they are estimating a single C-R function based on several cities, multi-city studies also tend to have more statistical power and provide effect estimates with relatively greater precision than single city studies due to larger sample sizes, reducing the uncertainty around the estimated coefficient. Because single-city and multi-city studies have different advantages, if a single-city C-R function has been estimated in a risk assessment location and a multi-city study which includes that location is also available for the same health endpoint, the results from both were used for that location and reported in the base case risk assessment.

4.2 Single and multi-pollutant models

For several of the epidemiological studies from which C-R relationships for the PM risk assessment were obtained, C-R functions are reported both for the case where only PM levels were entered into the health effects model (i.e., single pollutant models) and where PM and one or more other measured gaseous co-pollutants (i.e., ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide) were entered into the health effects model (i.e., multi-pollutant models). To the extent that any of the co-pollutants present in the ambient air may have contributed to the health effects attributed to PM in single pollutant models, risks attributed to PM might be overestimated where C-R functions are based on single pollutant models. However, as shown in the preliminary draft PM SP (see Figure 3-11, p.3-62 - 3-63), the magnitude and statistical significance of the associations reported between PM and mortality due to short-term exposure show no trends with the levels of any of the four gaseous co-pollutants examined.[27] As stated in the preliminary draft PM SP, "While not definitive, these consistent patterns indicate that it is more likely that there is an independent effect of $PM_{2.5}$, ... that is not confounded or appreciably modified by the gaseous co-pollutants"(draft PM SP, p.3-64). The findings from NMMAPS, which characterized the effects of PM_{10} and each of the gaseous co-pollutants, alone and in combination, also are relevant to the potential role of gaseous pollutants in modifying the effects associated with $PM_{2.5}$. An important finding of the NMMAPS analyses was the "lack of sensitivity of PM_{10} risk estimates to gaseous pollutants"(draft PM CD, p.8-40). Less consistent associations were found for the gaseous pollutants than for PM_{10}, though in the overall analyses, weak associations were found with SO_2, NO_2, and CO, and there was a significant association with ozone in the summer months (CD, p. 8-38). The authors concluded that their finding "suggests that the effect of PM_{10} is robust to the inclusion of other pollutants" (Samet et al., 2000b, p. 19).

For some of the gaseous co-pollutants, such as carbon monoxide, nitrogen dioxide, and sulfur dioxide, which tend to be highly correlated with ambient $PM_{2.5}$ concentrations in some cities, it is difficult to sort out whether these pollutants are exerting any independent effect from that attributed to $PM_{2.5}$. As discussed in the 2003 draft PM CD, inclusion of pollutants that are highly correlated with one another can lead to misleading conclusions in identifying a specific causal pollutant. When collinearity exists, multi-pollutant models would be expected to produce unstable and statistically insignificant effects estimates for both PM and the co-pollutants (2003 draft PM CD, p.9-130). Given that single and multi-pollutant models each have both potential advantages and disadvantages, with neither type clearly preferable over the other, we report risk estimates based on both single and multi-pollutant models where both are available.

[27]This is also consistent with updated analyses which are included in the current draft PM Staff Paper (EPA, 2003b).

4.3 Single, multiple, and distributed lag functions

The question of lags and the problems of correctly specifying the lag structure in a model is discussed extensively in the 2003 draft PM CD (Section 8.4.5). The 2003 draft PM CD points out that, in most PM times-series studies, after the basic model is fit (before considering PM), several different PM lags are typically fit in separate single-lag models and the most significant lag is chosen. The 2003 draft PM CD notes that "while this practice may bias the chance of finding a significant association, without a firm biological reason to establish a fixed pre-determined lag, it appears reasonable" (p. 8-234). There is recent evidence (Schwartz, 2000b) that the relationship between PM and health effects may best be described by a distributed lag (i.e., the incidence of the health effect on day n is influenced by PM concentrations on day n, day n-1, day n-2 and so on). If this is the case, a model that includes only a single lag (e.g., a 0-day lag or a 1-day lag) is likely to understate the total impact of PM. The 2003 draft PM CD makes this point, noting that "it is possible that current PM risk estimates, most frequently computed for a single-day or for two-day averages, may be underestimating these multi-day effects" (p. 8-239).

Because of this, a distributed lag model is considered preferable to a single lag model. Unfortunately, distributed lag models have been estimated in only a few cases (e.g., Schwartz, 2000b). Where distributed lag models are available, they were included in the PM risk assessment.[28] When a study reports several single lag models, unless the study authors identify a "best lag," we selected both 0- and 1-day lag models for mortality (both total and cause-specific) because, as indicated in the 2003 draft PM CD, these are the most often reported optimal lags (PM CD, p. 8-234). For hospital admission endpoints, unless the author specified an optimal lag, we selected both 0- and 1-day lag models for cardiovascular admissions since the draft CD indicates that recent evidence from time series studies strongly suggests maximal effects at 0-day lag with some carryover to 1-day lag and little evidence for effects beyond 1-day (CD, p. 8-147). Since many of the time-series studies addressing COPD hospital admissions report effects at somewhat longer lags, we selected 0-, 1- and 2-day lag models (if all three were available) for this health endpoint category.

For two urban locations, Chicago and Los Angeles, we included all single lag models estimated in a study, to show the effect of lag structure on the results of the risk assessment. We also conducted a sensitivity analysis examining the potential impact of using a distributed lag

[28] As explained in Schwartz (2000b), a "distributed lag coefficient" equal to the sum of the coefficients of the lagged PM concentrations can be used to predict the changes in the health endpoint resulting from a change in PM on a given day. For example, if there are three lags (0-day, 1-day, and 2-day lags) considered in the distributed lag model, then the distributed lag coefficient can be used to predict the sum of changes in the health endpoint on the same day, the next day, and the day after that. Confidence bounds around that sum can also be derived, given the standard error of the distributed lag coefficient.

approach for short-term exposure mortality associated with PM, based on the distributed lag analysis of PM_{10} and short-term exposure mortality by Schwartz (2000b).

4.4 Alternative approaches to estimating short-term exposure C-R functions

As noted in Sections 1 and 3, many studies that originally relied on GAMs using the S-Plus software to estimate short-term exposure C-R functions were subsequently reanalyzed. Many researchers used not just one but several alternative estimation approaches. In addition to GAMs with a more stringent convergence criterion, generalized linear model (GLM) approaches (with differing numbers of degrees of freedom, and different types of splines) were also used to reanalyze C-R functions. Thus, corresponding to a single log-linear C-R function with a single lag structure, there were often several different PM coefficients, each resulting from a different estimation approach.

Including all the alternative C-R functions in all the urban locations in the PM risk assessment would result in a prohibitively large set of results. Instead, for all urban locations except Chicago and Los Angeles, we included only GAM with a more stringent convergence criterion (denoted "GAM (stringent)"), because this approach most directly addresses the original issue while deviating the least from the estimation approach originally chosen by the study authors.[29] Although this approach does not address the issue of understated standard errors of coefficient estimates, this is probably not a significant drawback. The 2003 draft PM CD concludes that "the extent of downward bias in standard error reported in these data (a few percent to ~15%) also appears not to be very substantial, especially when compared to the range of standard errors across studies due to differences in population size and numbers of days available" (2003 draft PM CD, p. 8-197).

In those cases in which more than one lag model was estimated with each estimation approach, we followed the same procedure described in Section 4.3 above: where the best lag was identified by the study authors, we used this lag in the risk assessment. Where several lags were presented and the authors did not identify a best lag, we selected both 0- and 1-day lag models for mortality (both total and cause-specific), 0- and 1-day lag models for both cardiovascular and respiratory hospital admissions, and 0-, 1-, and 2-day lag models (if all three were available) for COPD hospital admissions, based on the discussion of lags in the 2003 draft PM CD.

In Chicago and Los Angeles, Moolgavkar (2000a) used several alternative estimation approaches and lag structures. We included a much wider array of models for these two urban

[29] In some cases (e.g., Moolgavkar, 2000a) two different versions of the "GAM (stringent)" approach were used – one with 30 degrees of freedom (df) and the other with 100 df. In those cases, we included only the version with 30 df.

locations to show the effects of different estimation approaches, for a given lag structure, and the effects of different lag structures, for a given estimation approach. First, for total non-accidental mortality, using the "GAM stringent" approach (with 30 degrees of freedom), we included all of the lag models used in the study (from 0-day lag to 5-day lag). Next, we included all of the estimation approaches for each of the lag models listed above for the different endpoints: both 0- and 1-day lag models for mortality (both total and cause-specific); 0- and 1-day lag models for both cardiovascular and respiratory hospital admissions; and 0-, 1-, and 2-day lag models (if all three were available) for COPD hospital admissions.

4.5 Long-term exposure mortality C-R functions

There are far fewer long-term exposure studies than short-term exposure studies cited in the draft PM CD. The available long-term exposure mortality C-R functions are all based on cohort studies, in which a cohort of individuals is followed over time. Two cohorts that have been studied are particularly relevant for the purposes of this risk assessment. One cohort, in six U.S. cities, was originally followed in a study referred to as the Six Cities study. The other cohort, of participants enrolled by American Cancer Society (ACS) volunteers, was composed of a much larger sample of individuals from many more cities. It was originally followed in a study referred to as the ACS study. There have recently been reanalyses of both the Six Cities study and the ACS study by Krewski et al. (2000), referred to here as Krewski et al. (2000) – Six Cities and Krewski et al. (2000) – ACS. Both of these reanalyses are used in the $PM_{2.5}$ risk assessment.

In addition, Pope et al. (2002) extended the follow-up period for the ACS cohort to sixteen years and published findings on the relationship of long-term exposure to $PM_{2.5}$ and all-cause mortality as well as cardiopulmonary and lung cancer mortality. Two periods of $PM_{2.5}$ measurements were considered in the study. The first, from 1979 through 1983, was the period considered in the original ACS study as well as in the Krewski reanalysis. The second was 1999-2000. The authors also report results based on an average of the two periods. It isn't clear which of the two periods is preferable to base risk estimates on, or whether the average of the two periods should be used. However, in their abstract Pope et al. (2002) report relative risks only based on $PM_{2.5}$ measurements from 1979 through 1983. We therefore selected the corresponding C-R functions based on $PM_{2.5}$ measurements from 1979-1983 to be included in the current $PM_{2.5}$ risk assessment. The relative risks reported by Pope et al. (2002) corresponding to the later period and to the average of the two periods were greater than the relative risk reported for the 1979-1983 period, so risk estimates based on either of the corresponding C-R functions would similarly be greater.

Two other PM cohort studies that are discussed in the 2003 draft PM CD are not included in the PM risk assessment. The Adventist Health Study of Smog (AHSMOG) followed 6,338 non-smoking non-Hispanic white Seventh day Adventist residents of California. The other study, the EPRI-Washington University Veteran's study, followed a cohort of middle-aged male

veterans who were, at the time of recruitment, mildly to moderately hypertensive. Because the cohorts in both studies are non-representative of the general U.S. population in several important ways, however, neither study was included in the risk assessment. These studies are discussed in greater detail in the draft PM CD (EPA, 2003a) and draft PM Staff Paper (EPA, 2003b)

4.6 Summary

To summarize, the basic approach to selecting C-R functions was as follows:

- if a single-city C-R function has been estimated in a risk assessment location and a multi-city study on the same health endpoint which includes that location is also available, risk and risk reduction estimates based on both are reported in the base case analysis; and

- if both single-pollutant and multi-pollutant C-R functions are available, risk and risk reduction estimates based on both are reported;

- if a distributed lag model was available from a study, that model was preferred; if only single lag models were available, unless the study authors identified a "best lag," we selected both 0- and 1-day lag models for mortality (both total and cause-specific), 0- and 1-day lag models for both cardiovascular and respiratory hospital admissions, and 0-, 1-, and 2-day lag models (if all three were available) for COPD hospital admissions, based on the discussion of lags in the 2003 draft PM CD. If the study authors did identify a best lag, however, we focused on the lag they identified as best.

- For short-term exposure studies that were reanalyzed in light of the GAM/S-Plus issue, if more than one alternative estimation approach was used, we selected the GAM approach with a more stringent convergence criterion; if more than one lag model was estimated, we followed the procedure we used for all studies, described above.

- For two cities (Chicago and Los Angeles), we included alternative approaches to estimating the C-R function (e.g., use of GLM) and evaluation of various lags to illustrate the effects of these alternative model specifications on the risk estimates.

- For long-term exposure mortality, the most recent C-R functions are used in the $PM_{2.5}$ risk assessment. Two of these are based on reanalyses of original cohort data; one (Pope et al., 2002) is an extension of the original study.

5. Baseline Health Effects Incidence Rates

Most of the epidemiology studies used in the PM risk assessment directly estimate the percentage change in incidence (i.e., the RR), rather than the absolute number of cases for an endpoint. To estimate the annual number of PM-associated cases using these studies, it is necessary to know the annual baseline incidence, that is, the annual 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, hospital admission, premature death) 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).[30] The sizes of the populations in the assessment locations that are relevant to the risk assessment (i.e., the populations for which the PM C-R functions are estimated and to which the baseline incidences refer) are given in Exhibits 5.1, 5.2, and 5.3 for the $PM_{2.5}$, PM_{10}, and $PM_{10\text{-}2.5}$ risk assessments, respectively.

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 included in the risk assessment 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 included in the risk assessment, their data are often available only at the national level (national averages), or at the regional or state level. We contacted state and local health departments and hospital planning commissions to obtain location-specific rates of cause-specific hospital admissions.

[30] 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.

Exhibit 5.1 Relevant Population Sizes for $PM_{2.5}$ Risk Assessment Locations

City	Population[a]							
	Total	Ages 7-14	Ages ≥25	Ages ≥30	Ages <65	Ages ≥ 65	Ages <75	Ages ≥75
Boston[1]	2,806,000	283,000 (10%)	1,903,000 (68%)	1,673,000 (60%)	---		---	---
Detroit[2]	2,061,000		---	1,153,000 (56%)	---	249,000 (12%)	---	---
Los Angeles[3]	9,519,000		---	5,092,000 (53%)	---	927,000 (10%)	---	---
Philadelphia[4]	1,518,000		---	852,000 (56%)	---	---	---	---
Phoenix[5]	---		---	1,684,000 (55%)	---	359,000 (12%)	---	---
Pittsburg[6]	---		---	814,000 (64%)	---	---	1,166,000 (91%)	116,000 (9%)
San Jose[7]	1,683,000		---	965,000 (57%)	---	---	---	---
Seattle[8]	---		---	1,044,000 (60%)	1,555,000 (90%)	---	---	---
St. Louis[9]	2,518,000	308,000 (12%)	1,637,000 (65%)	1,475,000 (59%)	---	---	---	---

[a] Total population and age-specific population estimates are based on 2000 U.S. Census data. See http://factfinder.census.gov/. Populations are rounded to the nearest thousand. The urban areas given in this exhibit are those considered in the studies used in the $PM_{2.5}$ risk assessment. The percentages in parentheses indicate the percentage of the total population in the specific age category.

[1] Middlesex, Norfolk, and Suffolk Counties. [2] Wayne County. [3] Los Angeles County. [4] Philadelphia County.

[5] Maricopa County. [6] Allegheny County. [7] Santa Clara County. [8] King County.

[9] St. Louis, Franklin, Jefferson, St. Charles, Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City.

Exhibit 5.2 Relevant Population Sizes for PM_{10} Risk Assessment Locations

City	Population[a]									
	Total	Ages 5-12 (asthmatics)*	Ages 7-14	Ages 9-11	Ages < 30	Ages ≥30	Ages <65	Ages ≥ 65	Ages < 75	Ages ≥ 75
Boston[1]	2,806,000	---	283,000 (10%)	---	---	---	---	---	---	---
Chicago[2]	5,377,000	---	---	---	---	---	---	630,000 (12%)	---	---
Detroit[3]	2,061,000	---	---	---	---	---	---	249,000 (12%)	---	---
Los Angeles[4]	9,519,000	---	---	---	4,428,000 (47%)	5,092,000 (53%)	---	927,000 (10%)	---	---
Minneapolis[5]	1,627,000	---	---	---	---	---	---	182,000 (11%)	---	---
Philadelphia[6]	1,518,000	---	---	---	---	---	---	---	---	---
Phoenix[7]	3,072,000	---	---	---	---	---	---	359,000 (12%)	---	---
Pittsburg[8]	1,282,000	---	---	---	---	---	---	228,000 (18%)	1,166,000 (91%)	116,000 (9%)
Provo[9]	369,000	---	---	20,000 (5%)	---	---	---	24,000 (6%)	---	---

City	Population[a]									
	Total	**Ages 5-12 (asthmatics)***	**Ages 7-14**	**Ages 9-11**	**Ages < 30**	**Ages ≥30**	**Ages <65**	**Ages ≥ 65**	**Ages < 75**	**Ages ≥ 75**
San Jose[10]	1,683,000	---	---	---	---	---	---	---	---	---
Seattle[11]	1,737,000	12,000 (1%)	---	---	---	---	1,555,000 (90%)	182,000 (10%)	---	---
St. Louis[12]	2,518,000	---	307,000 (12%)	---	---	---	---	---	---	---

[a] Total population and age-specific population estimates are based on 2000 U.S. Census data. See http://factfinder.census.gov/. Populations are rounded to the nearest thousand. The urban areas given in this exhibit are those considered in the studies used in the PM_{10} risk assessment. The percentages in parentheses indicate the percentage of the total population in the specific age category.

[1] Middlesex, Norfolk, and Suffolk Counties. [2] Cook County. [3] Wayne County.

[4] Los Angeles County. [5] Hennepin and Ramsey Counties. [6] Philadelphia County.

[7] Maricopa County. [8] Allegheny County. [9] Utah County.

[10] Santa Clara County. [11] King County.

[12] St. Louis, Franklin, Jefferson, St. Charles, Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City.

* The number of asthmatic children aged 5 to 12 was calculated by taking the King County population aged 5 to 12 (178,000 children) and multiplying this value by the asthma prevalence rate for children of this age category (6.9%). The prevalence rate was provided by the Seattle and King County Department of Health, and was calculated from the King County data gathered for the 1999-2000 Behavioral Risk Factor Surveillance System (BRFSS).

Exhibit 5.3 Relevant Population Sizes for $PM_{10-2.5}$ Risk Assessment Locations

City	Population[a]			
	Total	Ages 7-14	Ages ≥ 65	Ages <65
Detroit[1]	2,061,000	---	249,000 (12%)	---
Philadelphia[2]	1,518,000	---	---	---
Phoenix[3]	---	---	359,000 (12%)	---
Seattle[4]	---	---	---	1,555,000 (90%)
St. Louis[5]	2,518,000	307,000 (12%)	---	---

[a] Total population and age-specific population estimates are based on 2000 U.S. Census data. See http://factfinder.census.gov/. Populations are rounded to the nearest thousand. The urban areas given in this exhibit are those considered in the studies used in the $PM_{10-2.5}$ risk assessment. The percentages in parentheses indicate the percentage of the total population in the specific age category.
[1] Wayne County.
[2] Philadelphia County
[3] Maricopa County
[4] King County.
[5] St. Louis, Franklin, Jefferson, St. Charles, Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City.

We obtained estimates of location-specific baseline mortality rates for each of the assessment locations for 1998 from CDC Wonder, an interface for public health data dissemination from the Centers for Disease Control (CDC).[31] The mortality rates are derived from U.S. death records and U.S. Census Bureau post-censal population estimates, and are reported in Exhibit 5.4 through Exhibit 5.6 per 100,000 general population. 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). National rates are provided for comparison for 1998 from CDC Wonder.

Baseline incidence rates for both cardiovascular and respiratory hospital admissions were obtained for those locations in which hospital admissions C-R functions were estimated: Detroit, Chicago, Los Angeles, Minneapolis-St. Paul, Pittsburgh, Provo, and Seattle. Year 2000 hospitalization data were obtained for Wayne County (Detroit) from the Michigan Health and Hospital Association; year 2000 hospitalization data were obtained for Cook County (Chicago) from the Illinois Hospital Association. Year 1999 hospitalization data were obtained for Los Angeles County from California's Office of Statewide Health Planning and Development – Health Care Information Resource Center. Year 2000 hospitalization data were obtained for Hennepin and Ramsey Counties (Minneapolis/St. Paul) from the Minnesota Hospital Association (MHA), Minnesota Hospital and Healthcare Partnership, and from the Veterans Affairs Hospital (VAH). Data from the MHA did not include discharges from the VAH and from a few small hospitals. Data for the VAH were obtained directly from the hospital for the year 2000. Year 2000 hospitalization data were obtained for Allegheny County (Pittsburgh) from the Pennsylvania Health Care Cost Containment Council. Year 2000 hospitalization data were obtained for Utah County (Provo) from the Utah Department of Health, Utah Health Data Committee. Finally, year 2000 hospitalization data were obtained for King County (Seattle) from the State of Washington Department of Health, Center for Health Statistics, Office of Hospital and Patient Data Systems. These data are presented in Exhibit 5.7 through Exhibit 5.9. The data from these counties are actually annual hospital discharge data, which are used as a proxy for hospital admissions. Hospital discharges are issued to all people who are admitted to the hospital, including those who die in the hospital. By using the annual discharge rate, we assume that the admissions at the end of the year (e.g. 2000) that carry over to the beginning of the next year (e.g. 2001), and are therefore not included in the discharge data are offset by the admissions in the previous year (e.g. 1999) that carry over to the beginning of the current year (e.g. 2000).

[31] See http://wonder.cdc.gov/.

Exhibit 5.4 Baseline Mortality Rates for 1998 for $PM_{2.5}$ Risk Assessment Locations

Health Effect	Boston[1]	Detroit[2]	Los Angeles[3]	Philadelphia[4]	Phoenix[5]	Pittsburgh[6]	San Jose[7]	St. Louis[8]	National Average
Mortality[a]:									
A. Mortality Rates Used in Risk Analysis for Short-Term Exposure Studies[b] (deaths per 100,000 general population/year)									
Non-accidental (all ages): ICD codes <800	797	891	600	1137	---	---	518	909	808
Non-accidental (75+): ICD codes <800	---	---	---	---	---	707	---	---	468
Non-accidental (<75): ICD codes <800	---	---	---	---	---	420	---	---	340
Cardiovascular (all ages): ICD codes: 390-459	---	406	---	---	---	---	221	---	349
Cardiovascular (all ages): ICD codes: 390-448	---	---	---	454	---	---	---	---	348
Cardiovascular (65+): ICD codes: 390-448	---	---	---	---	233	---	---	---	294
Cardiovascular (all ages): ICD codes: 390-429	---	---	219	---	---	---	---	---	273

Health Effect	Boston[1]	Detroit[2]	Los Angeles[3]	Philadelphia[4]	Phoenix[5]	Pittsburgh[6]	San Jose[7]	St. Louis[8]	National Average
Ischemic Heart Disease (all ages): ICD codes: 410-414	140	---	---	---	---	---	---	128	170
Respiratory (all ages): ICD codes: 11, 35, 472-519, 710.0, 710.2, 710.4	---	---	---	---	---	---	68	---	89
Respiratory (all ages): ICD codes: 460-519	---	80	---	---	---	---	---	---	88
COPD without Asthma (all ages): ICD codes: 490-492, 494-496	33	---	---	---	---	---	---	43	39
COPD with Asthma (all ages): ICD codes: 490-496	---	---	31	---	---	---	---	---	42
Pneumonia (all ages): ICD codes: 480-487	45	---	---	---	---	---	---	42	34
B. Mortality Rates Used in Risk Analysis for Long-term Exposure Studies[b] (deaths per 100,000 general population/year)									
Total mortality (25+): ICD codes: all	819	---	---	---	---	---	---	938	837

Health Effect	Boston[1]	Detroit[2]	Los Angeles[3]	Philadelphia[4]	Phoenix[5]	Pittsburgh[6]	San Jose[7]	St. Louis[8]	National Average
Total mortality (30+): ICD codes: all	813	907	610	1165	716	1157	527	929	830
Cardiopulmonary Mortality (25+): ICD codes: 400-440, 485-495	335	---	---	---	---	---	---	438	370
Cardiopulmonary Mortality (30+): ICD codes: 401-440, 460-519	385	466	340	543	348	593	276	486	415
Lung Cancer Mortality (30+): ICD code: 162	58	62	34	88	47	81	32	70	57

[a] Mortality figures were obtained from CDC Wonder for 1998. See http://wonder.cdc.gov/.

[b] Mortality rates are presented only for the locations in which the C-R functions were estimated. All incidence rates are rounded to the nearest unit. Mortality rates for St. Louis may be slightly underestimated because some of the mortality counts in the smaller counties were reported as missing in CDC Wonder.

[1] Middlesex, Norfolk, and Suffolk Counties. [2] Wayne County. [3] Los Angeles County. [4] Philadelphia County.

[5] Maricopa County. [6] Allegheny County. [7] Santa Clara County.

[8] St. Louis, Franklin, Jefferson, St. Charles, Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City.

Exhibit 5.5 Baseline Mortality Rates for 1998 for PM_{10} Risk Assessment Locations

Health Effect	Boston[1]	Chicago[2]	Detroit[3]	Los Angeles[4]	Minnea-polis[5]	Phila-delphia[6]	Phoenix[7]	Pittsburgh[8]	Provo[9]	San Jose[10]	Seattle[11]	St. Louis[12]	Nat Avg.
Mortality Rates[a] Used in Risk Analysis for Short-Term Exposure Studies[b] (deaths per 100,000 general population/year)													
All cause (all ages) ICD codes <999.9	---	881	958	---	789	---	---	1182	---	---	696	---	864
Non-accidental (all ages): ICD codes <800	797	824	891	600	737	1137	690	1126	407	518	649	909	808
Non-accidental (75+): ICD codes <800	---	---	---	---	---	---	---	707	---	---	---	---	468
Non-accidental (<75): ICD codes <800	---	---	---	---	---	---	---	420	---	---	---	---	340
Non-accidental (65+): ICD codes <800	---	610	---	---	---	---	---	---	---	---	---	---	633

Health Effect	Boston[1]	Chicago[2]	Detroit[3]	Los Angeles[4]	Minnea-polis[5]	Phila-delphia[6]	Phoenix[7]	Pittsburgh[8]	Provo[9]	San Jose[10]	Seattle[11]	St. Louis[12]	Nat Avg.
Cardio-respiratory (all ages): ICD codes: 390-448, 480-487, 490-496, 507	390	422	475	345	328	552	355	603	---	283	330	494	428
Cardiovascular (all ages): ICD codes: 390-459	---	353	406	---	---	---	---	---	---	221	---	---	349
Cardiovascular (all ages): ICD codes: 390-448	---	---	---	---	---	454	---	---	157	---	---	---	348
Cardiovascular (65+): ICD codes: 390-448	---	---	---	---	---	---	233	---	---	---	---	---	294
Cardiovascular (all ages): ICD codes: 390-429	---	284	331	219	179	---	---	409	---	---	188	---	273
Respiratory (all ages): ICD codes: 480-486, 490-496	---	---	---	---	---	---	---	---	33	---	---	---	75

Health Effect	Boston[1]	Chicago[2]	Detroit[3]	Los Angeles[4]	Minnea-polis[5]	Phila-delphia[6]	Phoenix[7]	Pittsburgh[8]	Provo[9]	San Jose[10]	Seattle[11]	St. Louis[12]	Nat Avg.
Respiratory (all ages): ICD codes: 11, 35, 472-519, 710.0, 710.2, 710.4	---	---	---	---	---	---	---	---	---	68	---	---	89
Respiratory (all ages): ICD codes: 460-519	---	78	80	---	---	---	---	---	---	---	---	---	88
COPD with Asthma (all ages): ICD codes: 490-496	---	31	35	31	38	---	---	48	---	---	34	---	42
Pneumonia (all ages): ICD codes: 480-487	---	34	32	---	32	---	---	40	---	---	32	---	34

[a] Mortality figures were obtained from CDC Wonder for 1998. See http://wonder.cdc.gov/. Nat Avg: national average.

[b] Mortality rates are presented only for the locations in which the C-R functions were estimated. All incidence rates are rounded to the nearest unit. Mortality rates for St. Louis may be slightly underestimated because some of the mortality counts in the smaller counties were reported as missing in CDC Wonder.

[1] Middlesex, Norfolk, and Suffolk Counties. [2] Cook County. [3] Wayne County.
[4] Los Angeles County. [5] Hennepin and Ramsey Counties. [6] Philadelphia County.
[7] Maricopa County. [8] Allegheny County. [9] Utah County.
[10] Santa Clara County. [11] King County.
[12] St. Louis, Franklin, Jefferson, St. Charles, Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City.

Exhibit 5.6 Baseline Mortality Rates for 1998 for $PM_{10-2.5}$ Risk Assessment Locations

Health Effect	Detroit[1]	Philadelphia[2]	Phoenix[3]	St. Louis[4]	National Average
Mortality Rates[a] Used in Risk Analysis for Short-Term Exposure Studies[b] (deaths per 100,000 general population/year)					
Non-accidental (all ages): ICD codes <800	891	1137	---	909	808
Cardiovascular (all ages): ICD codes: 390-459	406	---	---	---	349
Cardiovascular (all ages): ICD codes: 390-448	---	454	---	---	348
Cardiovascular (65+): ICD codes: 390-448	---	---	233	---	294
Respiratory (all ages): ICD codes: 460-519	80	---	---	---	88

[a] Mortality figures were obtained from CDC Wonder for 1998. See http://wonder.cdc.gov/.

[b] Mortality rates are presented only for the locations in which the C-R functions were estimated. All incidence rates are rounded to the nearest unit. Mortality rates for St. Louis may be slightly underestimated because some of the mortality counts in the smaller counties were reported as missing in CDC Wonder.

[1] Wayne County. [2] Philadelphia County. [3] Maricopa County.

[4] St. Louis, Franklin, Jefferson, St. Charles, Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City.

Exhibit 5.7 Baseline Hospitalization Rates for $PM_{2.5}$ Risk Assessment Locations [a]

Health Effect	Detroit[1]	Los Angeles[2]	Seattle[3]
Hospital Admissions (per 100,000 general population/year)			
Pneumonia admissions (65 and over): ICD codes 480-486	250	---	---
COPD and asthma admissions (all ages): ICD codes 490-496	---	318	---
COPD and asthma admissions (65 and over): ICD codes 490-496	192	---	---
Asthma (<65): ICD code 493	---	---	92
Cardiovascular admissions (65 and over): ICD codes: 390-429	---	728	---
Ischemic heart disease (65 and over): ICD codes 410-414	487	---	---
Dysrhythmias (65 and over): ICD code 427	161	---	---
Congestive heart failure (65 and over): ICD code 428	341	---	---

[a] Hospitalization rates are presented only for the locations in which the C-R functions were estimated. For each location, the number of discharges was divided by the location's population from the 2000 U.S. Census estimates to obtain rates. All incidence rates are rounded to the nearest unit.

1. Wayne County. Year 2000 hospitalization data were obtained from the Michigan Health and Hospital Association.

2. Los Angeles County. Year 1999 hospitalization data were obtained from California's Office of Statewide Health Planning and Development – Health Care Information Resource Center.

3. King County. Year 2000 hospitalization data were obtained from the State of Washington Department of Health, Center for Health Statistics, Office of Hospital and Patient Data Systems.

Exhibit 5.8 Baseline Hospitalization Rates for PM_{10} Risk Assessment Locations [a]

Health Effect	Chicago[1]	Detroit[2]	Los Angeles[3]	Minneapolis[4]	Pittsburgh[5]	Provo[6]	Seattle[7]
Hospital Admissions (per 100,000 general population/year)							
Pneumonia admissions (65 and over): ICD codes 480-487	237	252	---	192	353	110	152
Pneumonia admissions (65 and over): ICD codes 480-486	---	250	---	186	---	---	---
COPD and asthma admissions (all ages): ICD codes 490-496	447	---	---	---	---	---	---
COPD and asthma admissions (65 and over): ICD codes 490-496	---	192	152	102	---	---	---
COPD without asthma admissions (65 and over): ICD codes 490-492, 494-496	121	163	---	86	272	19	62
COPD without asthma admissions (65 and over): ICD codes 491, 492, 494-496	---	162	---	---	---	---	---
Emergency and urgent asthma-related admissions (all ages)[b]	---	---	11	---	---	---	---
Asthma admissions (<30): ICD code 493	---	---	57	---	---	---	---
Asthma admissions (30 and over): ICD code 493	---	---	70	---	---	---	---
Asthma admissions (<65): ICD code 493	---	---	---	---	---	---	92

Health Effect	Chicago[1]	Detroit[2]	Los Angeles[3]	Minneapolis[4]	Pittsburgh[5]	Provo[6]	Seattle[7]
Cardiovascular admissions (65 and over): ICD codes: 390-429	1014	1160	728	721	1643	388	545
Ischemic heart disease (65 and over): ICD codes 410-414	---	487	---	---	---	---	---
Dysrhythmias (65 and over): ICD code 427	---	161	---	---	---	---	---
Congestive heart failure (65 and over): ICD code 428	320	341	---	---	---	---	---

[a] Hospitalization rates are presented only for the locations in which the C-R functions were estimated. For each location, the number of discharges was divided by the location's population from the 2000 U.S. Census estimates to obtain rates. All incidence rates are rounded to the nearest unit.

[b] The Nauenberg and Basu (1999) study in Los Angeles examines the effect of PM10 on hospital admissions – excluding scheduled admissions and transfers from other facilities – with a principal diagnosis of 493, or a principal diagnosis within 466.0, 480-483, 485-487, 518.81, 518.82, 786.09 and a secondary diagnosis of 493.

[1] Cook County. Year 2000 hospitalization data were obtained from the Illinois Hospital Association.

[2] Wayne County. Year 2000 hospitalization data were obtained from the Michigan Health and Hospital Association.

[3] Los Angeles County. Year 1999 hospitalization data were obtained from California's Office of Statewide Health Planning and Development – Health Care Information Resource Center.

[4] Hennepin and Ramsey Counties. Year 2000 hospitalization data were obtained from the Minnesota Hospital Association (MHA) – Minnesota Hospital and Healthcare Partnership, and from the Veterans Affairs Hospital (VAH). Data from the MHA did not include discharges from the VAH and from a few small hospitals. Data for the VAH were obtained directly from the hospital for the year 2000.

[5] Allegheny County. Year 2000 hospitalization data were obtained from the Pennsylvania Health Care Cost Containment Council.

[6] Utah County. Year 2000 hospitalization data were obtained from the Utah Department of Health, Utah Health Data Committee.

[7] King County. Year 2000 hospitalization data were obtained from the State of Washington Department of Health, Center for Health Statistics, Office of Hospital and Patient Data Systems.

Exhibit 5.9 Baseline Hospitalization Rates for $PM_{10-2.5}$ Risk Assessment Locations [a]

Health Effect	Detroit[1]	Seattle[2]
Hospital Admissions (per 100,000 general population/year)		
Pneumonia admissions (65 and over): ICD codes 480-486	250	---
COPD with asthma (65 and over): ICD codes 490-496	192	---
Asthma (<65): ICD code 493	---	92
Ischemic heart disease (65 and over): ICD codes 410-414	487	---
Dysrhythmias (65 and over): ICD code 427	161	---
Congestive heart failure (65 and over): ICD code 428	341	---

[a] Hospitalization rates are presented only for the locations in which the C-R functions were estimated. For each location, the number of discharges was divided by the location's population from the 2000 U.S. Census estimates to obtain rates. All incidence rates are rounded to the nearest unit.

1. Wayne County. Year 2000 hospitalization data were obtained from the Michigan Health and Hospital Association.

2. King County. Year 2000 hospitalization data were obtained from the State of Washington Department of Health, Center for Health Statistics, Office of Hospital and Patient Data Systems.

For respiratory symptoms the only available estimates of baseline incidence rates are from the studies that estimated the C-R functions for those endpoints. Four respiratory symptom studies (Schwartz and Neas, 2000; Schwartz et al., 1994; Pope et al., 1991; and Yu et al., 2000) are included in the PM risk assessment. Schwartz and Neas (2000) and Schwartz et al. (1994) estimated multi-city C-R functions using six cities, including Boston and St. Louis. The baseline incidence rates in these studies are likewise based on all six cities combined. The C-R functions and the baseline incidence rates (for lower and upper respiratory symptoms, in Schwartz et al., 1994; and for lower respiratory symptoms and cough, in Schwartz and Neas, 2000) were used in Boston and St. Louis. The other two studies (Pope et al., 1991; and Yu et al., 2000) each estimated C-R functions and reported baseline incidence rates for a single city. The C-R functions and baseline incidence rates for upper and lower respiratory symptoms from Pope et al. (1991) were reported for and applied in the risk assessment to Provo, Utah. The C-R function and baseline incidence rate for asthma symptoms from Yu et al. (2000) were reported for and applied in the risk assessment to Seattle, WA.

6. Sources of Uncertainty and Variability

The PM health risk models that were used in the risk assessment combined information about PM for specific urban areas to derive estimates of the annual incidence of specified health effects associated with "as is" PM concentrations and the reduction in incidence that would result upon just meeting the current $PM_{2.5}$ standards in those areas. The three main inputs to such analyses -- air quality information, C-R information, and baseline incidence and population information -- all vary from one time and location to another time and location. In addition, there are uncertainties associated with each of these three main inputs to the health risk assessment.

We were able to obtain air quality information for many, but not all days in the year for each assessment location. Some uncertainty surrounding the results of the analyses therefore arises from the incompleteness of the air quality data. Even if the air quality data were complete, there is always some degree of measurement error with any monitoring data, including that of PM. We also recognize that for any given assessment location there is year to year variability in the distribution of daily PM ambient concentrations and annual average concentrations. The current health risk assessment focuses on a single year and does not incorporate year-to-year variability.

We were able to obtain baseline incidence rates specific to each assessment location (specifically, for all counties included in each assessment location). However, the available information was not specific to the exact analysis period, although it was possible to obtain baseline incidence rates from quite recent years (e.g., mortality rates were obtained for 1998). The risk assessment also does not reflect any year-to-year variability that may exist in baseline incidence rates. These factors result in some additional uncertainty surrounding the results of the risk assessment, although this uncertainty component is likely to be small.

Finally, even if the input values were from the same times and locations as the analysis periods and locations, they are only *estimates*, and therefore have statistical uncertainty, including sampling error, surrounding them. The specific sources of uncertainty in the PM risk assessment are described in detail below and are summarized in Exhibit 6.1.

Although the PM risk assessment considered mortality as well as several morbidity health effects, not all health effects which may result from PM exposure were included. Only those for which there was sufficient epidemiological evidence from studies which met the study selection criteria (see Section 3) were included in the risk assessment. Other possible health effects reported to be associated with short- and/or long-term exposures to PM are considered qualitatively in the draft OAQPS Staff Paper. Thus, the PM risk assessment does not represent all of the health risks associated with PM exposures.

Exhibit 6.1 Key Uncertainties in the Risk Assessment

Uncertainty	Comments
Causality	Statistical association does not prove causation. However, the risk assessment considers only health endpoints for which the overall weight of the evidence supports the assumption that PM is likely causally related.
Empirically estimated C-R relations	Because C-R functions are empirically estimated, there is uncertainty surrounding these estimates. Omitted confounding variables could cause bias in the estimated PM coefficients. However, including potential confounding variables that are highly correlated with one another can lead to unstable estimators. Both single- and multi-pollutant models were used where available.
Functional form of C-R relation	Statistical significance of coefficients in an estimated C-R function does not necessarily mean that the mathematical form of the function is the best model of the true C-R relation. The impact of possible alternative threshold models was explored in a sensitivity analysis.
Lag structure of C-R relation	There is some evidence of a distributed lag. Most models, however, included only one lag. Omitted lags could cause downward bias in the predicted incidence associated with a given reduction in PM concentrations. A comparison or sensitivity analysis using an approach to estimate the possible impact of using a distributed lag C-R function was carried out.
Transferability of C-R relations	C-R functions may not provide an adequate representation of the C-R relationship in times and places other than those in which they were estimated. For example, populations in the analysis locations may have more or fewer members of sensitive subgroups than locations in which functions were derived, which would introduce additional uncertainty related to the use of a given C-R function in the analysis location. However, in the majority of cases, the risk assessment relies on C-R functions estimated from studies conducted in the same location.

Uncertainty	Comments
Extrapolation of C-R relations beyond the range of observed PM data	A C-R relationship estimated by an epidemiological study may not be valid at concentrations outside the range of concentrations observed during the study. To partially address this problem, risk was not calculated for PM levels below the LML in a study, if it was available. If the LML was not available, risk was estimated down to background level, which may be lower than the lowest PM level measured in the study.
Truncation of risk estimates at the lowest PM concentration observed in a study	To avoid relying on a C-R function below the lowest PM concentration from which it was estimated, risk was not calculated for PM levels below the LML in a study, if it was available. If there is any positive relationship between PM and the health response below this level, this procedure will understate the PM impact.
Adequacy of PM characterization	Only size differentiated particle mass per unit volume has been explicitly considered, and not, for example, chemical composition. However, in the majority of cases, the risk assessment relies on C-R functions estimated from studies conducted in the same location as the assessment location. Therefore differences in PM between the study location in which a C-R function was estimated and the assessment location to which it is applied are, in general, minimal (arising only from possible temporal changes).
Accuracy of PM mass measurement	Possible differences in measurement error, losses of particular components, and measurement method between the assessment locations and the study locations would be expected to add uncertainty to quantitative estimates of risk.
Adjustment of air quality distributions to simulate just meeting current $PM_{2.5}$ standards	The pattern and extent of daily reductions in $PM_{2.5}$ concentrations that would result if current $PM_{2.5}$ standards were just met is not known. Although the assumption that $PM_{2.5}$ concentrations would be reduced by the same percentage on all days appears reasonable given the patterns observed based on historical data, there remains uncertainty about the shape of the air quality distribution of daily levels upon just meeting alternative $PM_{2.5}$ standards which will depend on future air quality control strategies.

Uncertainty	Comments
Background PM concentrations	The calculation of PM risk associated with "as is" air quality and of risk reductions that would result if current standards were just met requires as inputs the background PM concentrations in each of the assessment locations. Background concentrations were estimated for the eastern and western regions of the country, but not specifically for the assessment locations. In addition, a constant value is used for the estimated background, which does not take into account seasonal or daily variability in background concentrations. Sensitivity analyses were conducted, however, exploring the impact of assuming both a constant background level at the lower and upper end of the range estimated in the draft PM CD and of allowing daily background levels to vary day by day.
Baseline health effects data	Data on baseline incidence is uncertain 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.

In addition, we limited application of a C-R function to only that portion of the population on which estimation of the function was based. For example, lower respiratory symptoms were examined in Schwartz and Neas (2000) for children ages 7-14. It is likely that the effect of PM on lower respiratory symptoms does not begin at age 7 and end at age 14; however, data are not available to estimate the number of cases avoided for other age groups. Therefore, a substantial number of potentially avoided health effects were likely not captured in this analysis.

6.1 Concentration-response functions

The C-R function is a key element of the PM risk assessment. The quality of the risk assessment depends, in part, on (1) whether the C-R functions used in the risk assessment are good estimates of the relationship between the population health response and ambient PM concentration in the study locations, (2) how applicable these functions are to the analysis periods and locations, 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.

6.1.1 Uncertainty associated with the estimated concentration-response functions in the study locations

The uncertainty associated with an estimate of a C-R function reported by a study depends on the sample size and the study design. The 2003 draft PM CD 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 ambient PM, the adequacy of the health effects incidence data, and the consideration of potentially important health determinants and potential confounders and effect modifiers such as:

- other pollutants;
- exposure to other health risks, such as smoking and occupational exposure; and
- demographic characteristics, including age, sex, socioeconomic status, and access to medical care.

The selection of studies included in the PM risk assessment was guided by the evaluations in the 2003 draft PM CD. One of the criteria for selecting studies addresses the adequacy of the measurement of ambient PM. This criterion was that PM was directly measured using $PM_{2.5}$, PM_{10}, or $PM_{10\text{-}2.5}$ as the indicator or, for $PM_{2.5}$, was estimated using nephelometry data where direct $PM_{2.5}$ measurement data were not available. This criterion was designed to minimize error in the estimated PM coefficients in the C-R functions used in the risk assessment.

To the extent that a study did not address all relevant factors (i.e., all factors that affect the health endpoint), there is uncertainty associated with the C-R function estimated in that study, beyond that reflected in the confidence interval. It may result in either over- or underestimates of risk associated with ambient PM concentrations in the location in which the study was carried out. Techniques for addressing the problem of confounding factors and other study design issues have improved over the years, however, and the epidemiological studies currently available for use in the PM risk assessment provide a higher level of confidence in study quality than ever before.

When a study is conducted in a single location, the problem of possible confounding co-pollutants may be particularly difficult, if co-pollutants are highly correlated in the study location. Single-pollutant models, which omit co-pollutants, may produce overestimates of the PM effect, if some of the effects of other pollutants (omitted from the model) are falsely attributed to PM. With regard to gaseous co-pollutants as potential confounders, a new multi-city study (NMMAPS; Samet et al., 2000; Dominici et al., 2003) has evaluated the effects of PM_{10} alone and in combination with each of the monitored gaseous co-pollutants across the 90 largest U.S. cities and reported that associations found between PM_{10} and mortality were not confounded by the presence of the gaseous co-pollutants (2003 draft PM CD, p. 8-35). It is likely that this is true for $PM_{2.5}$ as well, although there is no equivalent $PM_{2.5}$ study like the NMMAPS. Statistical estimates of a PM effect based on a multi-pollutant model can be more uncertain, and even statistically insignificant, if the co-pollutants included in the model are highly correlated with PM. This means that, although the expected value of the estimated PM coefficient is correct, the estimate based on any particular sample may be too low or too high. As a result of these considerations, we report risk estimates based on both single-pollutant and multi-pollutant models, when both are reported by a study.

6.1.2 Applicability of concentration-response functions in different locations

As described in Section 3, risk assessment locations were selected on the basis of where C-R functions have been estimated, to avoid the uncertainties associated with applying a C-R function estimated in one location to another location. However, multi-county, multi-city, and/or regional C-R functions were also applied to any risk assessment location contained in the set of locations used to estimate the C-R function.[32] The accuracy of the results based on a multi-location C-R function rests in part on how well this multi-location C-R function represents the relationship between ambient PM and the given population health response in the individual cities involved in the study.

[32] In addition, national multi-pollutant C-R functions for mortality associated with short-term exposure to PM_{10} were applied to all of the urban areas in the PM_{10} risk assessment. The results based on national multi-pollutant C-R functions are shown in the exhibits for the PM_{10} risk assessment locations, but not in the figures.

The relationship between ambient PM concentration and the incidence of a given health endpoint in the population (the population health response) depends on (1) the relationship between ambient PM concentration and personal exposure to ambient-generated PM and (2) the relationship between personal exposure to ambient-generated PM and the population health response. Both of these are likely to vary to some degree from one location to another.

The relationship between ambient PM concentration and personal exposure to ambient-generated PM will depend on patterns of behavior, such as the amount of time spent outdoors, as well as on factors affecting the extent to which ambient-generated PM infiltrates into indoor environments. The relationship between personal exposure to ambient-generated PM and the population health response will depend on both the composition of the PM and on the composition of the population exposed to it.

The composition of PM (e.g., the chemical constituents of the PM) is known to differ from one location to another. As discussed in the 2003 draft PM CD, recent studies provide some evidence for health effect associations with different $PM_{2.5}$ components, including sulfates, acids, and metals. However, the 2003 draft PM CD (p. 9-61) also states that "[a] primary causative attribute may not exist but rather many attributes may contribute to a complex mechanism driven by the nature of a given PM and its contributing sources. The multiple interactions that may occur in eliciting a response in a host may make the identification of any single causal component difficult and may account for the fact that mass as the most basic metric shows the relationships to health outcomes that it does."

Exposed populations also differ from one location to another in characteristics that are likely to affect their susceptibility to PM air pollution. For instance, people with pre-existing conditions such as chronic bronchitis are probably more susceptible to the adverse effects of exposure to PM, and populations vary from one location to another in the prevalence of specific diseases. Also, some age groups may be more susceptible than others, and population age distributions also vary from one location to another. Closely matching populations observed in studies to the populations of the assessment locations is not possible for many characteristics (for example, smoking status, workplace exposure, socioeconomic status, and the prevalence of highly susceptible subgroups).

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

In summary, the C-R relationship is most likely not the same everywhere. Even if the relationship between personal exposure to ambient-generated PM and population health response were the same everywhere, the relationship between ambient concentrations and personal

exposure to ambient-generated PM may differ among locations. Similarly, even if the relationship between ambient concentrations and personal exposure to ambient-generated PM were the same everywhere, the relationship between personal exposure to ambient-generated PM and population health response may differ among locations. In either case, the C-R relationship would differ.

6.1.3 Extrapolation beyond observed air quality levels

Although a C-R function describes the relationship between ambient PM and a given health endpoint for all possible PM levels (potentially down to zero), the estimation of a C-R 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. Thus, uncertainty in the shape of the estimated C-R function increases considerably outside the range of PM concentrations observed in the study.

The PM risk assessment assumes that the estimated C-R functions adequately represent the true C-R relationship down to background PM levels in the assessment locations, in cases in which this background level is above the lowest concentrations used to derive the C-R functions. Estimates of risk were not generated for concentrations below the minimum concentrations observed in the studies. Applying proportional rollbacks to the concentration distributions in the assessment locations may result in some modeled PM concentrations below the lowest levels observed in the studies. In such cases, the difference in PM was taken to be the difference between the "as is" levels and the lowest observed level in the study. This procedure avoids relying on a C-R function below the level of PM concentrations from which it was estimated. However, it will tend to understate the impact of just meeting a set of standards if there is actually a C-R relationship below these lowest observed PM levels.

It is possible that there is a minimum concentration (i.e., threshold) below which PM is not associated with health effects. If there is such a concentration, including incidence reductions associated with reducing PM levels below this minimum threshold level in the total incidence reduction would overstate the risk attributable to PM or the incidence reductions that would result from just attaining a standard. We conducted sensitivity analyses to examine the sensitivity of risk estimates to different assumptions about hypothetical thresholds, as described in Section 7.

The C-R relationship may also be less certain towards the upper end of the concentration range being considered in a risk assessment, particularly if the PM concentrations in the assessment location exceed the PM concentrations observed in the study location. Even though it may be reasonable to model the C-R relationship as log-linear over the ranges of PM

concentrations typically observed in epidemiological studies, it may not be log-linear over the entire range of PM levels at the locations considered in the PM risk assessment.[33]

6.2 The air quality data

6.2.1 Use of PM as the indicator

PM is often measured in units of mass per unit volume, and typically reported in micrograms per cubic meter ($\mu g/m^3$). The PM risk assessment used PM size classes – $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ – and the chemical composition of PM was not considered explicitly (as it was not in most of the epidemiological studies used in these analyses). As summarized in Chapter 9 of the 2003 draft PM CD, recent studies provide new evidence for health effects associations with many different PM components. Recognizing that ambient PM exposure has been associated with increases in numerous health indices, the evidence is still too limited to allow identification of which PM components or sources might be more toxic than others, and growing evidence indicates that there are numerous potentially toxic PM components and some components may act in combination (see 2003 draft PM CD, Section 8.2.2.4). It is possible that PM risks may differ from one area to another with differing PM composition, but this potential source of uncertainty cannot be tested in this risk assessment. However, because the risk assessment primarily uses C-R functions estimated from studies conducted in the same location as the analysis location, the C-R functions already capture to some extent the potential impact of differential composition. To the extent that composition differentially affects toxicity and if future control strategies alter the composition in an area, then this introduces an additional uncertainty into the risk estimates associated with just meeting the current or any alternative PM standards.

6.2.2 Adequacy of PM air quality data

The method of averaging data from monitors across a metropolitan area in the risk assessment is similar to the methods used to characterize ambient air quality in most of the epidemiology studies. Ideally, the measurement of average daily ambient PM concentrations in the study location is unbiased. In this case, unbiased risk predictions in the assessment location depend, in part, on an unbiased measurement of average daily ambient PM concentrations in the assessment location as well. If, however, the measurement of average daily ambient PM concentrations in the study location is biased, unbiased risk predictions in the assessment location are still possible if the measurement of average daily ambient PM concentrations in the assessment location incorporates the same bias as exists in the study location measurements.

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[33]Although most of the C-R functions are log-linear, they are practically linear. It is still unlikely, however, that a linear function is appropriate over a very wide range of PM concentrations.

Because this is not known, however, the errors in the PM measurements in the assessment locations are a source of uncertainty in the risk assessment.

PM air quality data were not available for all days of the year chosen for the risk assessment in many of the assessment locations.[34] 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 2.6 for an explanation of the method of extrapolating changes in health effects incidence to an entire year). If there are seasonal differences in average PM levels and in monitoring frequencies, a simple annual adjustment for missing data could result in a biased estimate of total annual incidence change. To minimize the presence of bias due to an uneven distribution of missing data throughout the year, incidence changes in different quarters of the year were scaled separately, and the scaled quarterly results were added.

Because the PM data in each assessment location were limited to a specific year (usually 2002), the results of the risk assessment are generalizable to the present only to the extent that ambient PM levels in the available data are similar to current ambient PM levels in those locations. A substantial difference between PM levels in the year used in the risk assessment and current PM levels could imply a substantial difference in predicted incidences of health effects. This is not expected to be a large problem for the PM risk assessment, however, because adequate PM monitoring data were available for most of the assessment locations in the year 2002, which is quite recent, or if not in 2002 then in a relatively recent year (1999 or later, except for the $PM_{10-2.5}$ risk assessment in Phoenix, which used data from 1997).

6.2.3 Simulation of reductions in $PM_{2.5}$ concentrations to just meet the current standards

The pattern of daily $PM_{2.5}$ concentrations that would result if the current $PM_{2.5}$ standards were just met in any of the assessment locations is, of course, not known. Although the assumption that $PM_{2.5}$ concentrations will be reduced by the same percentage on all days may be a reasonable approximation, it is only an approximation. There is therefore uncertainty surrounding the predicted daily changes in $PM_{2.5}$ concentrations that would result if the current $PM_{2.5}$ standards were just met, and consequently uncertainty surrounding the associated daily changes in population health response.

[34] $PM_{2.5}$ monitor data were available for all days in the year for four of the locations in the $PM_{2.5}$ risk assessment, and almost complete data were available for most of the other locations; monitor data were substantially more sparse, however, for PM_{10} and $PM_{10-2.5}$.

6.3 Baseline health effects incidence rates

Most of the C-R functions used in the PM risk assessment are log-linear (see equations 1 through 3 in Section 2.5).[35] Given this functional form, the percent change in incidence of a health effect corresponding to a change in PM 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, as shown in equation 3 in Section 2.5:

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

in which $e^{\beta\Delta x}$ is the RR, and [$e^{\beta\Delta x}$ - 1] is the percent change associated with a change in PM of Δx. If there has been an increase in PM (i.e., if Δx positive), then the RR will be greater than 1.0. If, for example, the RR associated with a change in PM of Δx is 1.05, then the percent change in incidence of the health effect is 0.05 (5%). The change in incidence of the health effect associated with a change in PM of Δx is, then, 5 percent of the baseline incidence, y. Predicted changes in incidence therefore depend on the baseline incidence of the health effect.

6.3.1 Quality of incidence data

County-specific incidence data were available for mortality for all counties. We have also obtained hospital admissions baseline incidence data for all the urban areas for which we have hospital admissions C-R functions for one or more of the PM indicators (Chicago, Detroit, Los Angeles, Minneapolis, Pittsburgh, Provo, and Seattle). This is clearly preferable to using non-local 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.

Data on hospital admissions are actually hospital discharge data rather than admissions data. Because of this, the date associated with a given hospital stay is the date of discharge rather than the date of admissions. Therefore, there may be some hospital admissions in an assessment location in the year of interest (e.g., 2000) that are not included in the baseline incidence rate, if the date of discharge was after the year ended, even though the date of admissions was within the

[35] The exceptions to this are Lipfert et al. (2000), which reports linear C-R functions for mortality, and the studies which report logistic functions for respiratory symptoms (Schwartz and Neas, 2000; Schwartz et al., 1994; Pope et al., 1991; and Yu et al., 2000).

year. Similarly, there may be some hospital admissions that preceded the year of interest that are included in the baseline incidence rate because the date of discharge was within the year of interest. This is a very minor problem, however, partly because the percentage of such cases is likely to be very small, and partly because the error at the beginning of the year (i.e., admissions that should not have been included but were) will largely cancel the error at the end of the year (i.e., admissions that should have been included but were not).

Another minor uncertainty surrounding the hospital admissions baseline incidence rates arises from the fact that these rates are based on the reporting of hospitals within each of the assessment counties. Hospitals report the numbers of ICD code-specific discharges in a given year. If people from outside the county use these hospitals, and/or if residents of the county use hospitals outside the county, these rates will not accurately reflect the numbers of county residents who were admitted to the hospital for specific illnesses during the year, the rates that are required for the risk assessment. Once again, however, this is likely to be a very minor problem because the health conditions studied tend to be acute events that require immediate hospitalization, rather than planned hospital stays.

Incidence rates for respiratory symptoms were obtained from the studies reporting the C-R functions for those endpoints (Schwartz and Neas, 2000; Schwartz et al., 1994; Pope et al., 1991; and Yu et al., 2000). All of these studies except Schwartz and Neas (2000) considered only a single location and reported a baseline incidence rate for that location. Schwartz and Neas (2000) considered six cities, and the baseline incidence rates reported in that study were based on all six locations combined. Therefore there is some uncertainty associated with applying it to the individual locations (Boston and St. Louis) that are in both the study and the PM risk assessment. In addition, because this study is a reanalysis of data collected earlier, changes in baseline incidence rates over time could have introduced additional uncertainty into the analysis.

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.

Both morbidity and mortality rates change over time for various reasons. One of the most important of these is that population age distributions change over time. 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 assessment. However, the average age of the population in many locations will increase as 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. 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.

6.3.2 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 analyses based on C-R 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. However, only annual baseline incidence rates are available. Average daily baseline incidence rates, necessary for short-term daily C-R functions, were calculated by dividing the annual rate by the number of days in the year for which the baseline incidence rates were obtained.[36] To the extent that PM affects health, however, actual incidence rates would be expected to be somewhat higher than average on days with high PM concentrations; using an average daily incidence rate would therefore result in underestimating the changes in incidence on such days. Similarly, actual incidence rates would be expected to be somewhat lower than average on days with low PM concentrations; using an average daily incidence rate would therefore result in overestimating the changes in incidence on low PM days. Both effects would be expected to be small, however, and should largely cancel one another out.

[36] This is 365, unless the baseline incidence data were obtained from the year 2000, which is a leap year, and therefore has 366 days.

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

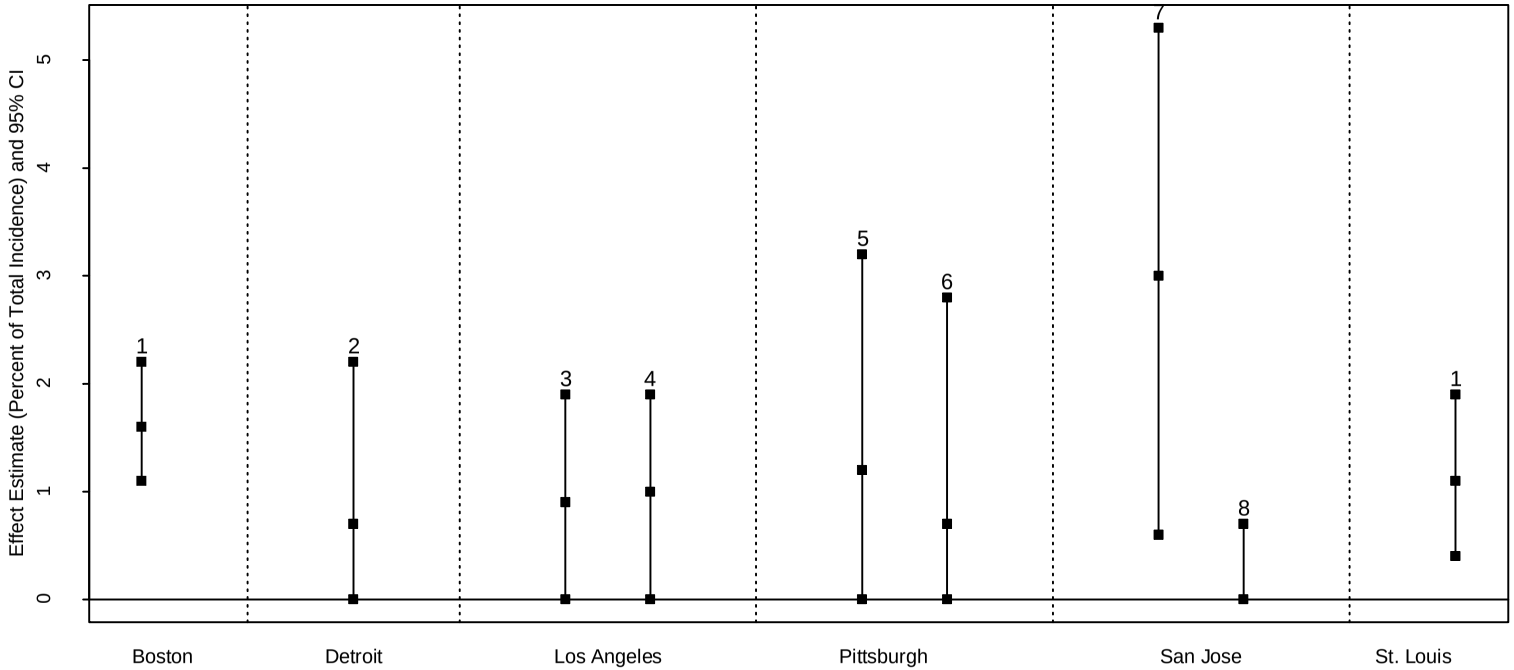
7.1 Base case analysis

The results of the first part of the risk assessment, assessing the health risks associated with "as is" PM concentrations in excess of background concentration, are summarized across urban areas in figures. The percent of total incidence that is PM-related is shown in Figures 7.1 through 7.6 for $PM_{2.5}$, Figures 7.7 through 7.11 for PM_{10}, and Figure 7.12 for $PM_{10-2.5}$. Corresponding figures showing PM-related incidence are shown, in Appendix D, in Figures D.1 through D.6 for $PM_{2.5}$, Figures D.7 through D.11 for PM_{10}, and Figure D.12 for $PM_{10-2.5}$. The legend of symbols used for the different health endpoints in the figures is given in Figure 7.13. In addition, results are given for Detroit in Exhibits 7.1 through 7.3 for $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$, respectively. The corresponding exhibits for the other urban locations are given in Appendix D. A separate exhibit is presented for each combination of PM indicator and assessment location. All estimated incidences were rounded to the nearest 10, except respiratory symptoms, which were rounded to the nearest 1,000. All percentages were rounded to one decimal place.

As discussed in Chapter 3, assessment locations were chosen in part on the basis of whether an acceptable C-R function had been reported for that location. As a result, risks were estimated in a given assessment location only for those health endpoints for which there is at least one acceptable C-R function reported for that location. The set of health effects shown in Exhibits 7.1 through 7.3 and Exhibits D.1 through D.23 therefore varies from one location to another. For example, mortality associated with short-term and long-term exposure to $PM_{2.5}$ and respiratory symptoms are included in Exhibit D.1 for Boston, but hospital admissions are not included because there was no study that met the selection criteria that reports a C-R function for hospital admissions reported in the $PM_{2.5}$ epidemiological literature for Boston. Exhibit D.7 for Seattle, in contrast, includes only hospital admissions because there was no study in the epidemiological literature that met the selection criteria that reports C-R functions for mortality or respiratory symptoms in Seattle associated with short-term exposure to $PM_{2.5}$, and the annual average $PM_{2.5}$ concentration in Seattle was lower than the LML in all three long-term exposure mortality studies.

There is substantial uncertainty surrounding all estimates of incidence associated with "as is" PM concentrations in any location. We tried to minimize the extent of this uncertainty by avoiding the application of a C-R function estimated in one location to another location as much as possible. As discussed in Section 6, however, there are other sources of uncertainty. The uncertainty surrounding risk estimates resulting from the statistical uncertainty of the PM coefficients in the C-R functions used is characterized by ninety-five percent confidence intervals around incidence estimates and estimates of the percent of total incidence that PM-related incidence comprises.

Figure 7.1. Estimated Annual Percent of Total (Non-Accidental) Mortality Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant, Single-City Models‡

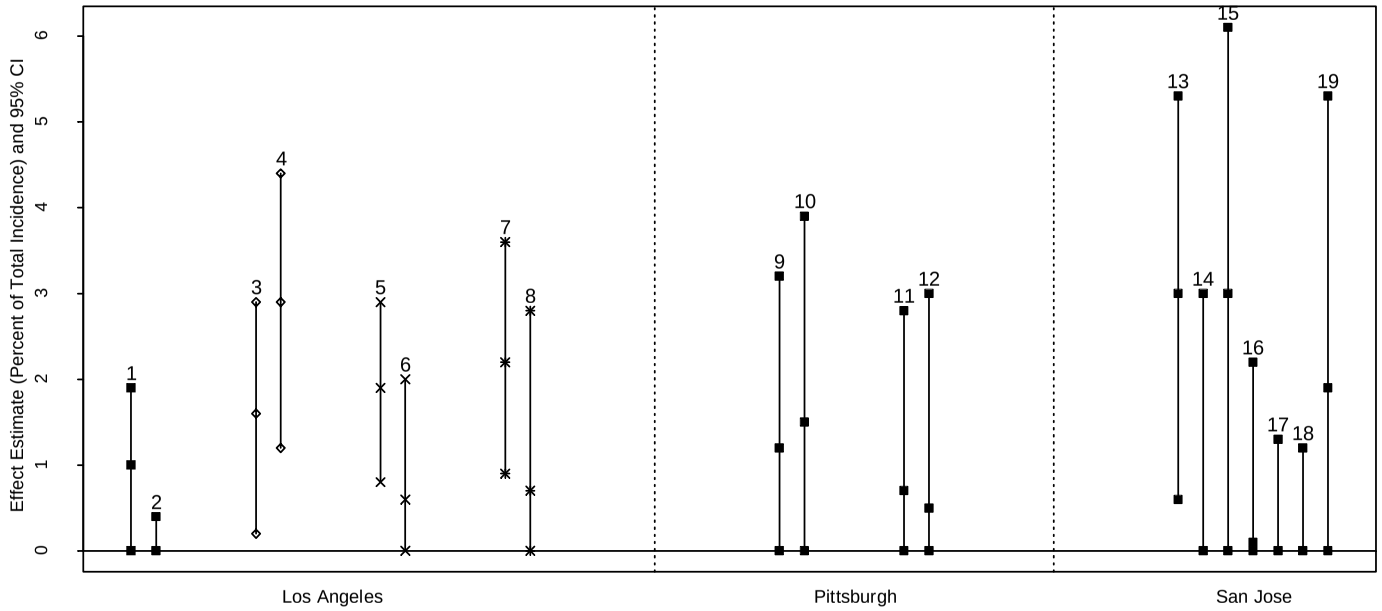


1. Schwartz et al. (1996)*
2. Lippmann et al. (2000)**
3. Moolgavkar (2000a)*** – 0-day lag
4. Moolgavkar (2000a)*** – 1-day lag
5. Chock et al. (2000) (age<75)
6. Chock et al. (2000) (age>75)
7. Fairley (1999)**** – 0-day lag
8. Fairley (1999)**** – 1-day lag

*Reanalyzed in Schwartz (2003b)
**Reanalyzed in Ito (2002)
***Reanalyzed in Moolgavkar (2003)
****Reanalyzed in Fairley (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.2. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-Pollutant versus Multi-Pollutant Models‡
(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)

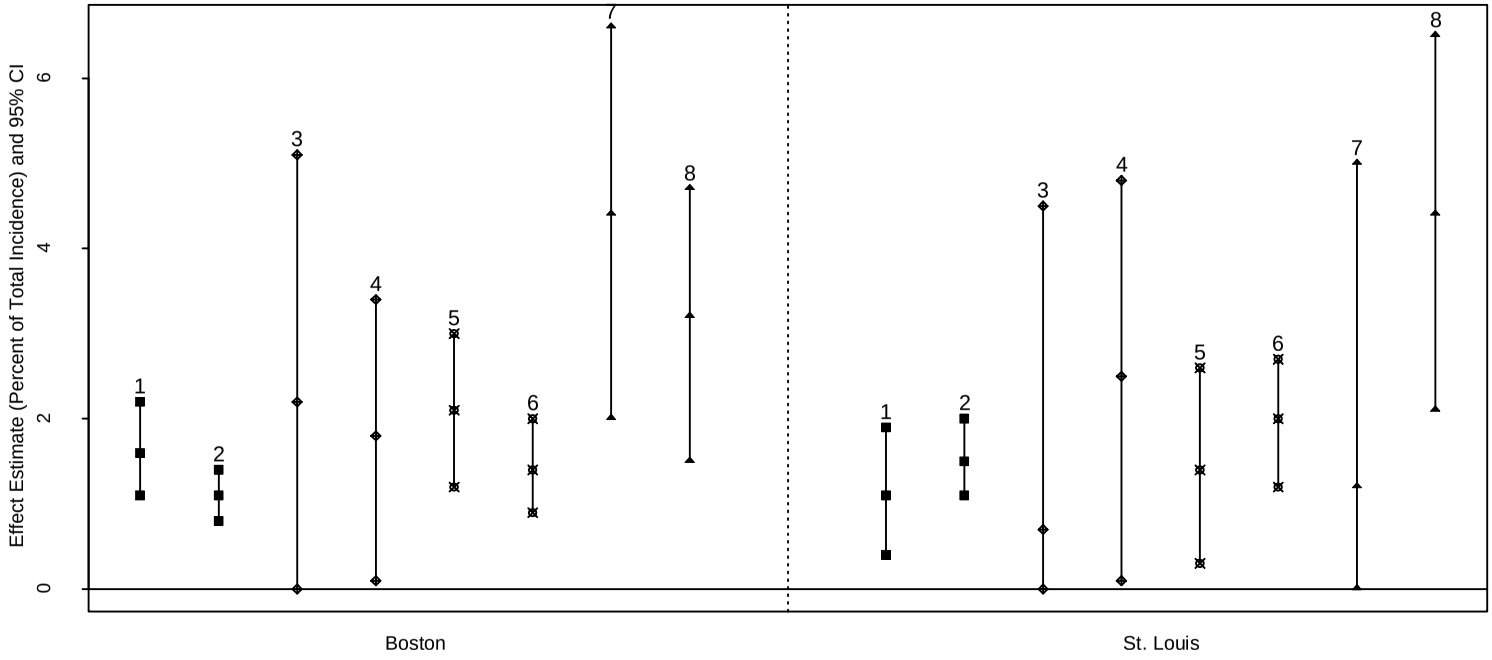


1. Moolgavkar (2000a)* – non-accid. mort.
2. Moolgavkar (2000a)* – non-accid. mort.; + CO
3. Moolgavkar (2000a)* – cardiovasc. mort.
4. Moolgavkar (2000a)* – cardiovasc. mort.; + CO
5. Moolgavkar (2000b)* – cardiovasc. hosp. adm.
6. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; + CO
7. Moolgavkar (2000c)* – COPD hosp. adm.
8. Moolgavkar (2000c)* – COPD hosp. adm.; + NO2
9. Chock et al. (2000) (age<75) – non-accid. mort.
10. Chock et al. (2000) (age<75) – non-accid. mort.; + CO, O3, SO2, PM10-2.5
11. Chock et al. (2000) (age>75) – non-accid. mort.
12. Chock et al. (2000) (age>75) – non-accid. mort.; + CO, O3, SO2, PM10-2.5
13. Fairley (1999)** – non-accid. mort.;
14. Fairley (1999)** – non-accid. mort.; + COH
15. Fairley (1999)** – non-accid. mort.; + NO3
16. Fairley (1999)** – non-accid. mort.; with SO4
17. Fairley (1999)** – non-accid. mort.; with NO2
18. Fairley (1999)** – non-accid. mort.; + CO
19. Fairley (1999)** – non-accid. mort.; + 8 hr O3

*Reanalyzed in Moolgavkar (2003)
**Reanalyzed in Fairley (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.3. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models‡
(Single-city models are always on the left, followed by the corresponding multi-city models)

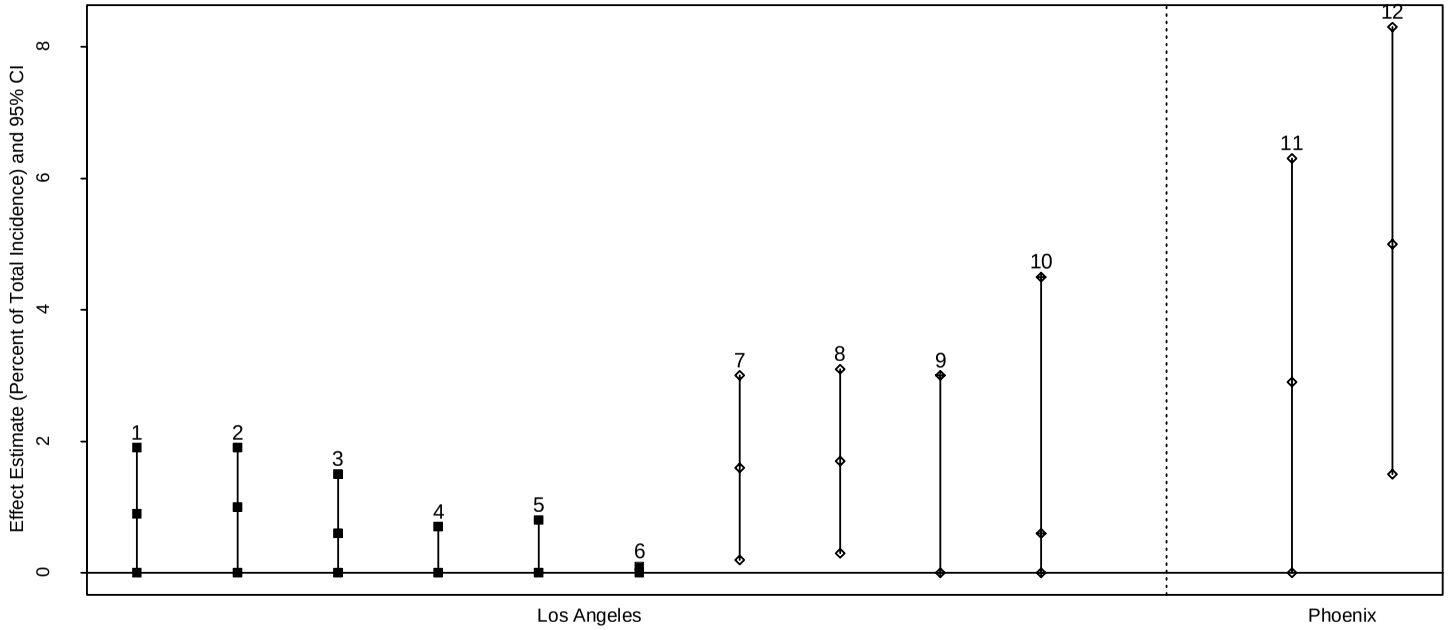


1. Schwartz et al. (1996)* – single-city; non-accid. mort.
2. Schwartz et al. (1996)* – 6 cities; non-accid. mort.
3. Klemm et al. (2000)** – single-city; COPD mort.
4. Klemm et al. (2000)** – 6 cities; COPD mort.
5. Klemm et al. (2000)** – single-city; ischemic heart disease mort.
6. Klemm et al. (2000)** – 6 cities; ischemic heart disease mort.
7. Klemm et al. (2000)** – single-city; pneumonia mort.
8. Klemm et al. (2000)** – 6 cities; pneumonia mort.

*Reanalyzed in Schwartz (2003b)
** Reanalyzed in Klemm and Mason (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.4. Estimated Annual Percent of Mortality Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Effect of Different Lag Models‡

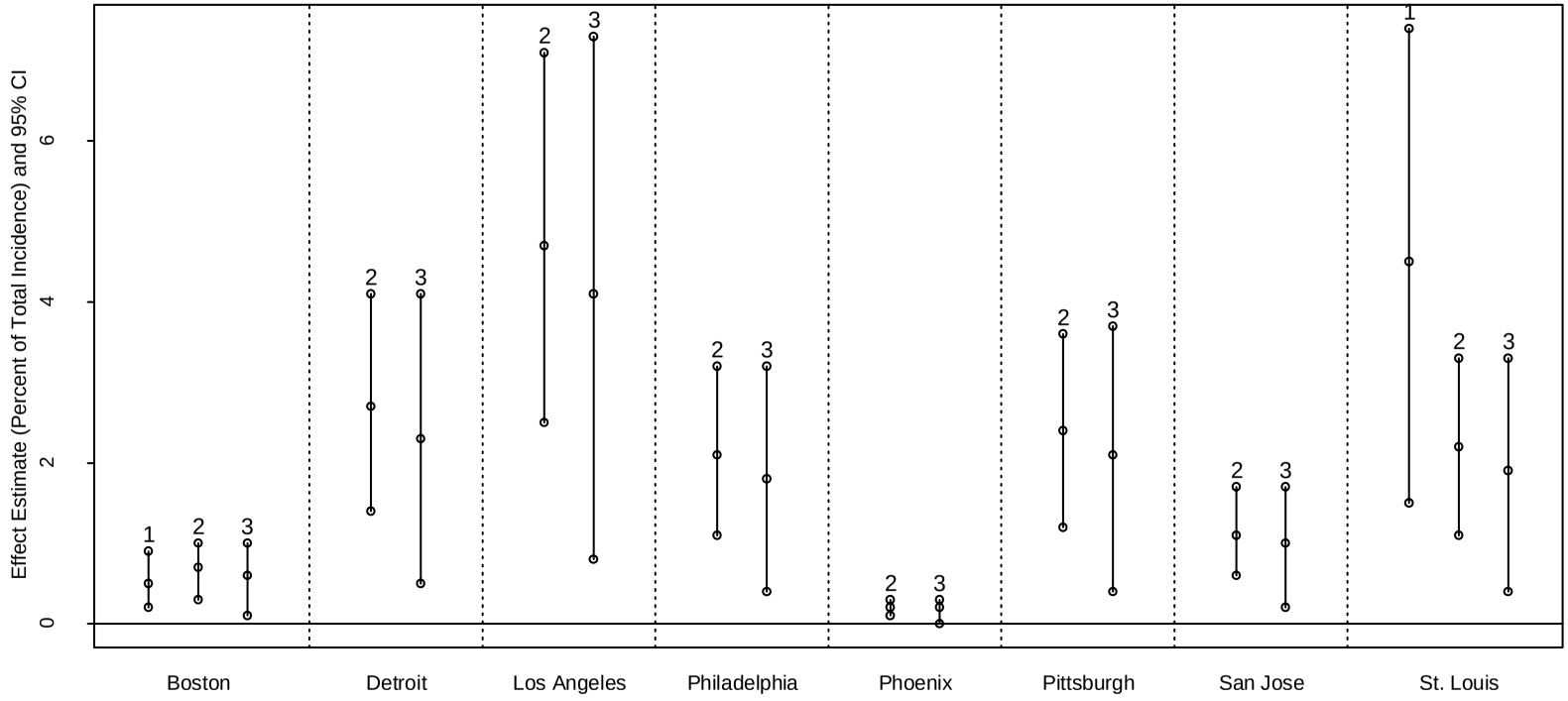


1. Moolgavkar (2000a)* – non-accid. mort. – 0-day lag
2. Moolgavkar (2000a)* – non-accid. mort. – 1-day lag
3. Moolgavkar (2000a)* – non-accid. mort. – 2-day lag
4. Moolgavkar (2000a)* – non-accid. mort. – 3-day lag
5. Moolgavkar (2000a)* – non-accid. mort. – 4-day lag
6. Moolgavkar (2000a)* – non-accid. mort. – 5-day lag
7. Moolgavkar (2000a)* – cardiovascular mort. – 0-day lag
8. Moolgavkar (2000a)* – cardiovascular mort. – 1-day lag
9. Moolgavkar (2000a)* – COPD mort. – 0-day lag
10. Moolgavkar (2000a)* – COPD mort. – 1-day lag
11. Mar et al. (2000)** – cardiovascular mort. – 0-day lag
12. Mar et al. (2000)** – cardiovascular mort. – 1-day lag

*Reanalyzed in Moolgavkar (2003)
**Reanalyzed in Mar et al. (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.5. Estimated Annual Percent of Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant Models[‡] [†]

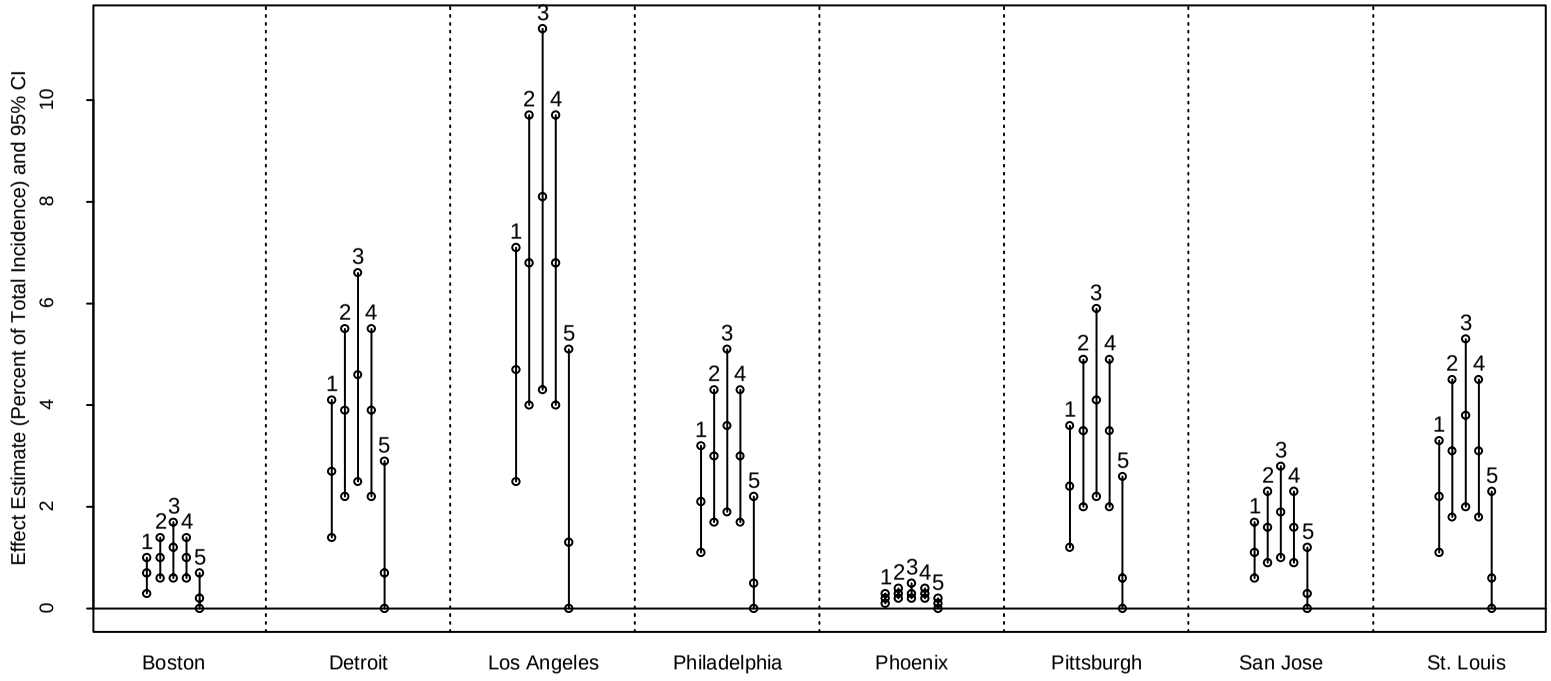


1. Krewski et al. (2000) – Six Cities
2. Krewski et al. (2000) – ACS
3. Pope et al. (2002) – ACS extended

[‡] In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

[†] Seattle is not shown because annual average $PM_{2.5}$ in Seattle is less than the LML in all three studies, so the risk would be estimated to be zero.

Figure 7.6. Estimated Annual Percent of Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant and Multi-Pollutant Models‡ †
(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)

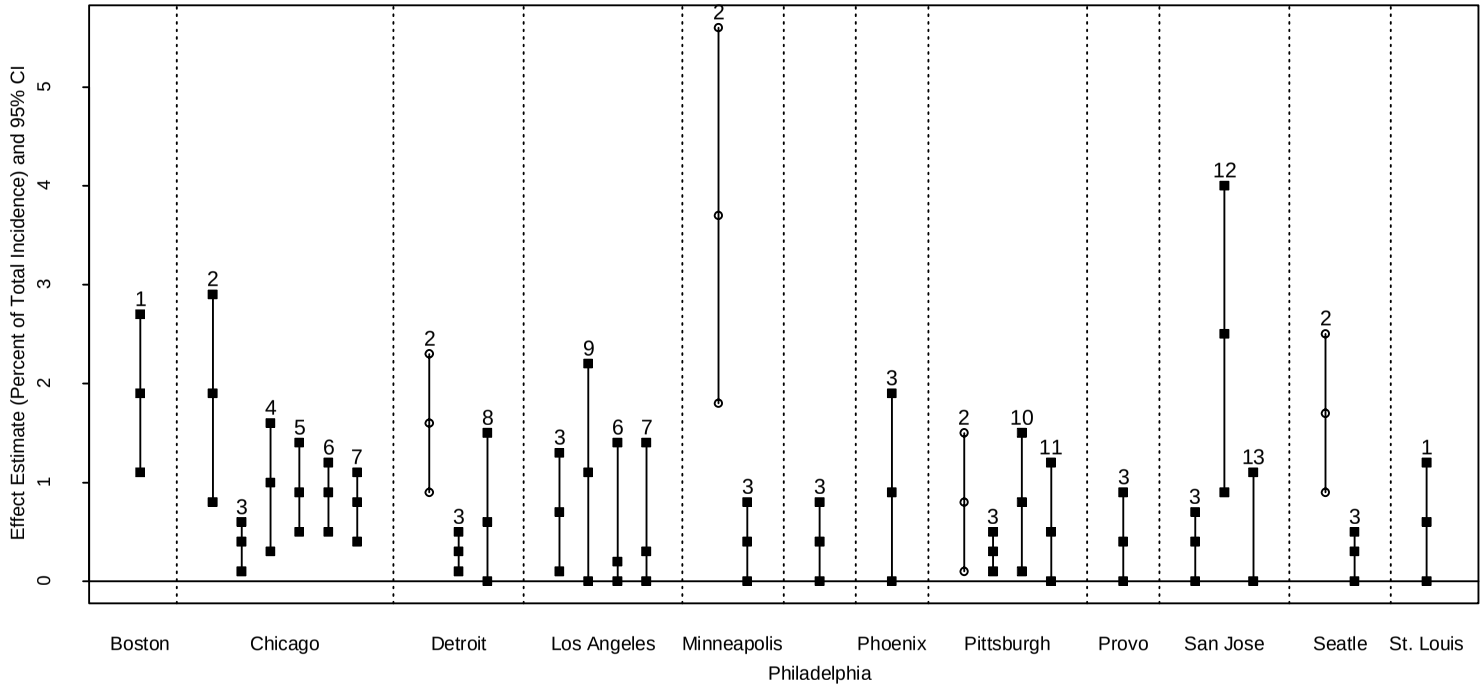


1. Krewski et al. (2000) – ACS
2. Krewski et al. (2000) – ACS – with CO
3. Krewski et al. (2000) – ACS – with NO2
4. Krewski et al. (2000) – ACS – with O3
5. Krewski et al. (2000) – ACS – with SO2

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

† Seattle is not shown because annual average $PM_{2.5}$ in Seattle is less than the LML in all three studies, so the risk would be estimated to be zero.

Figure 7.7. Estimated Annual Percent of Total (Non-Accidental) Mortality Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Single-Pollutant, Single-City Models‡

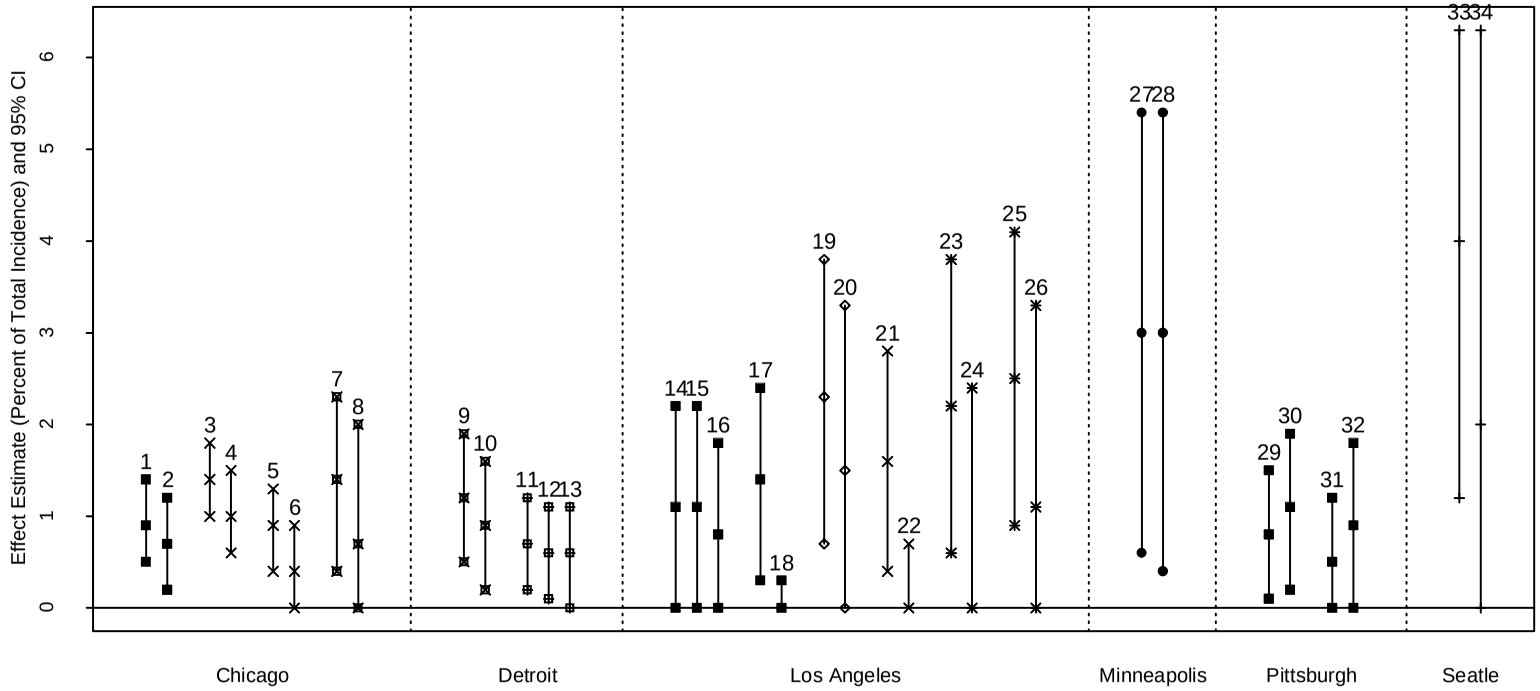


1. Klemm et al. (2000)*
2. Schwartz (2000b)** — distr. lag
3. Samet et al. (2000)***
4. Styer et al. (1995
5. Ito and Thurston (1996)
6. Moolgavkar (2000a)**** – 0-day lag
7. Moolgavkar (2000a)**** – 1-day lag
8. Lippmann et al. (2000)*****
9. Kinney et al. (1995)
10. Chock et al. (2000) – age<75
11. Chock et al. (2000) – age>75
12. Fairley (1999)****** – 0-day lag
13. Fairley (1999)****** – 1-day lag

*Reanalyzed in Klemm and Mason (2003)
**Reanalyzed in Schwartz (2003a)
***Reanalyzed in Dominici et al. (2003)
****Reanalyzed in Moolgavkar (2002)
*****Reanalyzed in Ito (2003)
******Reanalyzed in Fairley (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.8. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Results Based on Single-Pollutant versus Multi-Pollutant Models‡
(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)



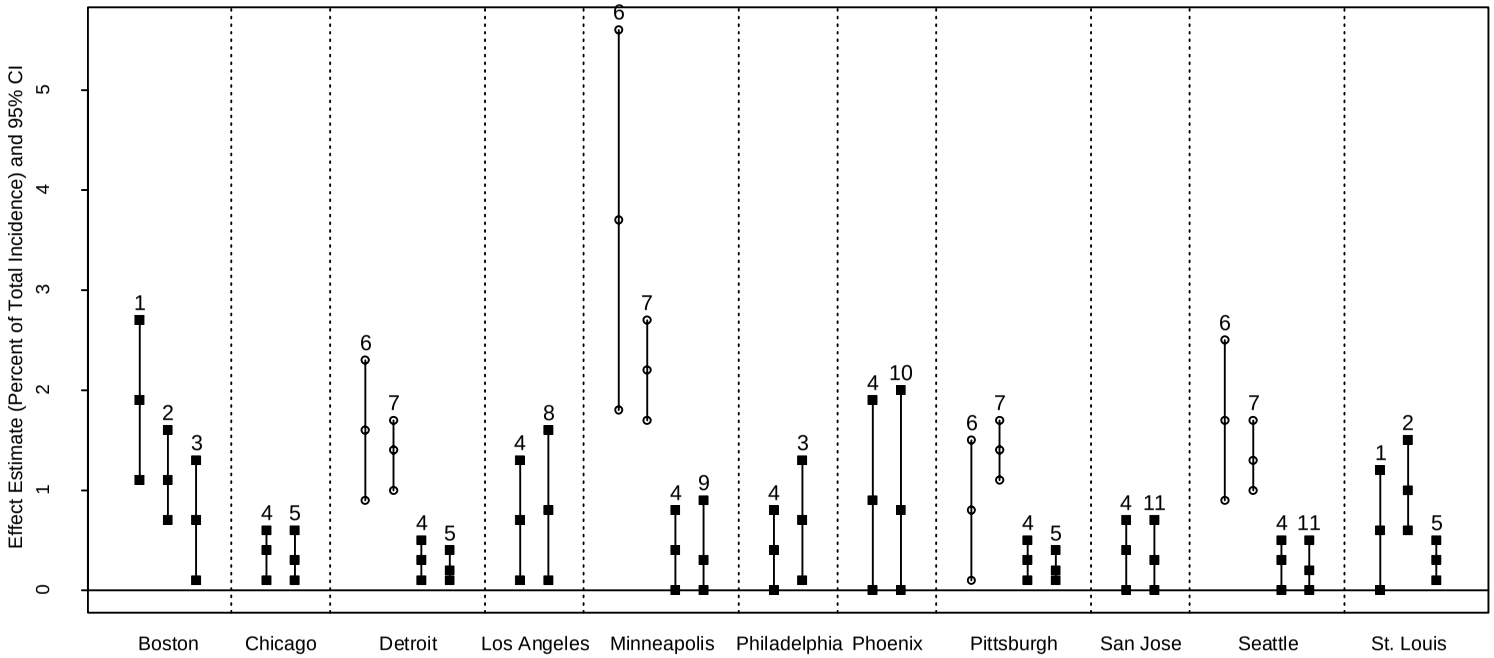
1. Ito and Thurston (1996) – non-accid. mort.
2. Ito and Thurston (1996) – non-accid. mort.; +O3
3. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 0-day lag
4. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 0-day lag +CO
5. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 1-day lag
6. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 1-day lag +CO
7. Morris and Naumova (1998) – cong. heart failure hosp. adm.
8. Morris and Naumova (1998) – cong. heart failure hosp. adm.; +CO, SO2, NO2, O3
9. Schwartz and Morris (1995) – cong. heart failure hosp. adm.
10. Schwartz and Morris (1995) – cong. heart failure hosp. adm.; +CO
11. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.
12. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.; +CO
13. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.; +SO2
14. Kinney et al. (1995) – non-accid. mort.
15. Kinney et al. (1995) – non-accid. mort.; +O3
16. Kinney et al. (1995) – non-accid. mort.; +CO

17. Moolgavkar (2000a)* – non-accid. mort.; 2-day lag*
18. Moolgavkar (2000a)* – non-accid. mort.; 2-day lag; + CO*
19. Moolgavkar (2000a)* – cardiovasc. mort.; 2-day lag*
20. Moolgavkar (2000a)* – cardiovasc. mort.; 2-day lag; + CO*
21. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 2-day lag*
22. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 2-day lag; + CO*
23. Moolgavkar (2000c)* – COPD. hosp. adm.; 0-day lag
24. Moolgavkar (2000c)* – COPD. hosp. adm.; 0-day lag; + NO2
25. Moolgavkar (2000c)* – COPD. hosp. adm.; 1-day lag
26. Moolgavkar (2000c)* – COPD. hosp. adm.; 1-day lag; + NO2
27. Schwartz (1994b) – pneumonia hosp. adm.
28. Schwartz (1994b) – pneumonia hosp. adm.; + O3
29. Chock et al. (2000), age<75 – non-accid. mort.
30. Chock et al. (2000), age<75 – non-accid. mort.; + NO2
31. Chock et al. (2000), age>75 – non-accid. mort.
32. Chock et al. (2000), age>75 – non-accid. mort.; + CO
33. Yu et al. (2000) – asthma symptoms
34. Yu et al. (2000) – asthma symptoms; + CO, SO2

*Reanalyzed in Moolgavkar (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.9. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models: Mortality‡
(Single-city models are always on the left, followed by the corresponding multi-city models)

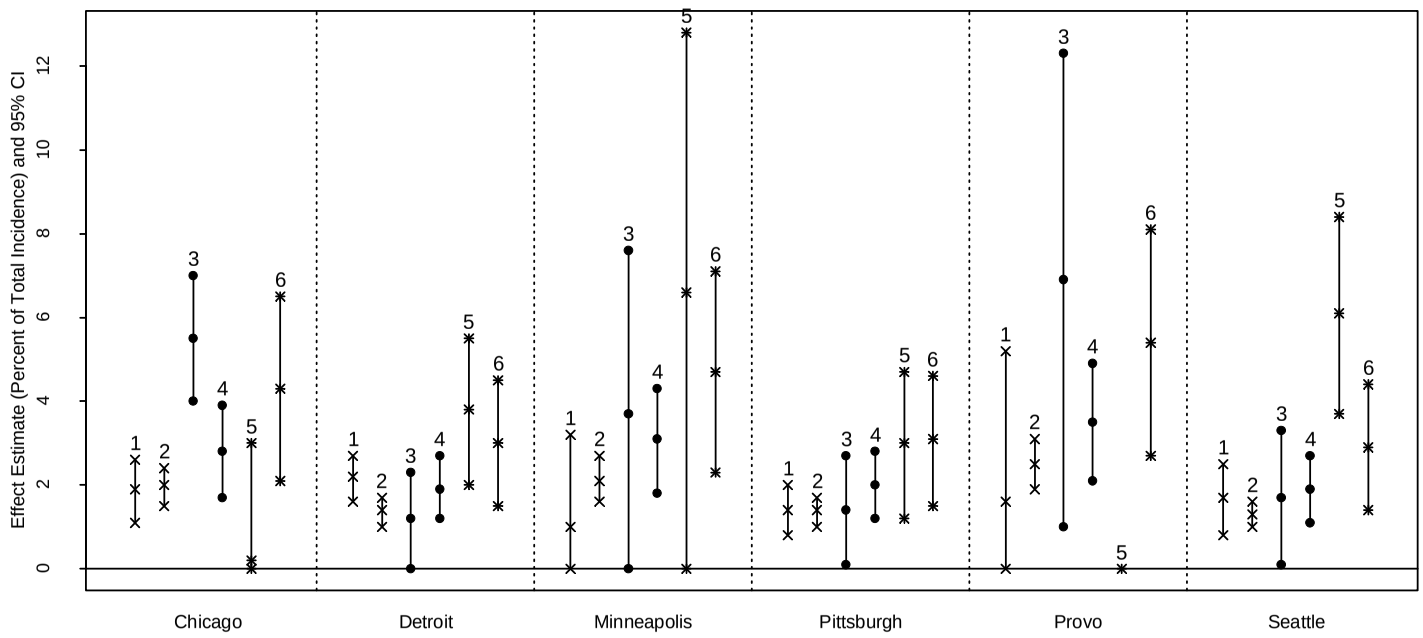


1. Klemm et al. (2000)* – single-city model
2. Klemm et al. (2000)* – six-city model
3. Samet et al. (2000)** – Northeast regional model
4. Samet et al. (2000)** – single-city model
5. Samet et al. (2000)** – Industrial Midwest regional model
6. Schwartz (2000b)*** – single-city model
7. Schwartz (2000b)*** – 10-city model
8. Samet et al. (2000)** – South California regional model
9. Samet et al. (2000)** – Upper Midwest regional model
10. Samet et al. (2000)** – Southwest regional model
11. Samet et al. (2000)** – Northwest regional model

*Reanalyzed in Klemm and Mason (2003)
**Reanalyzed in Dominici et al. (2003)
***Reanalyzed in Schwartz (2003a)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.10. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models: Morbidity‡
(Single-city models are always on the left, followed by the corresponding multi-city models)

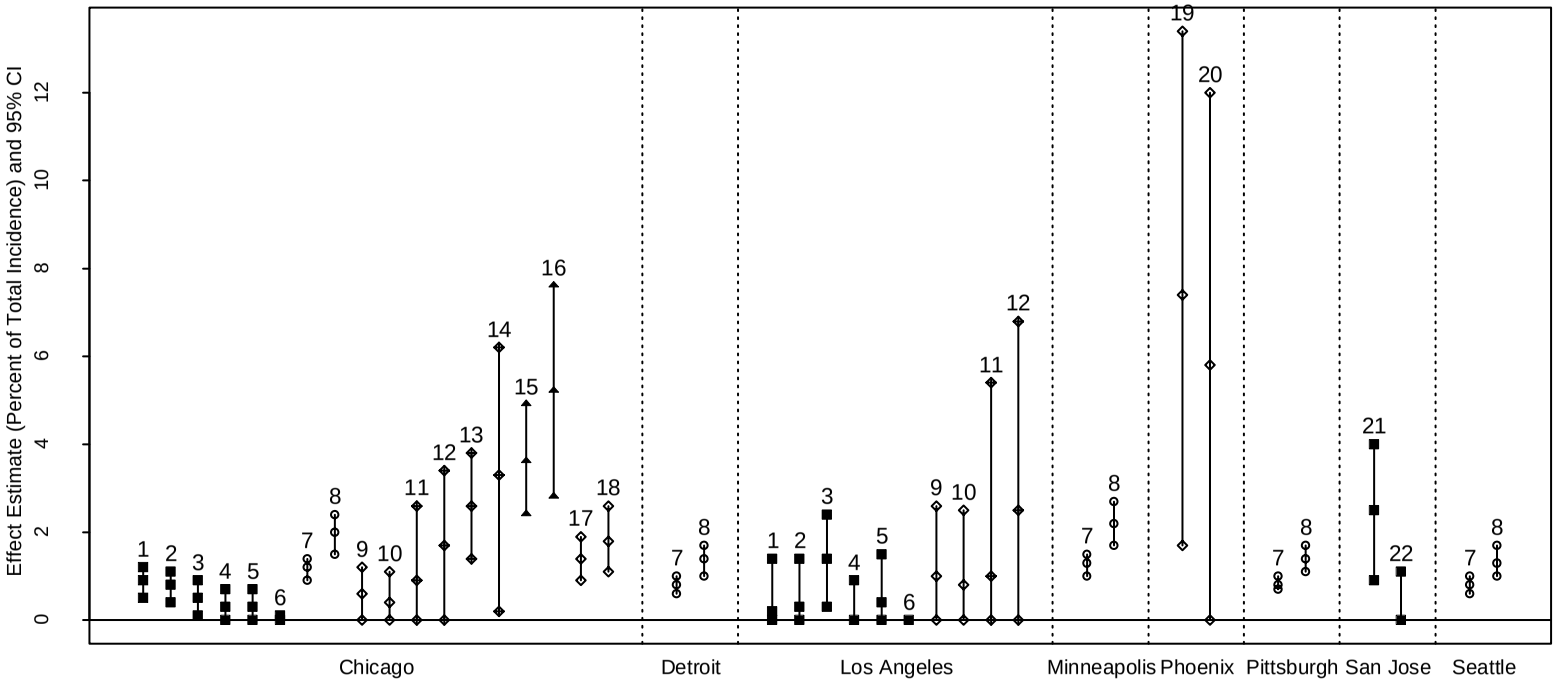


1. Samet et al. (2000)* – single-city model; cardiovasc. hosp. adms.
2. Samet et al. (2000)* – 14-city model; cardiovasc. hosp. adms.
3. Samet et al. (2000)* – single-city model; pneumonia hosp. adms.
4. Samet et al. (2000)* – 14-city model; pneumonia hosp. adms.
5. Samet et al. (2000)* – single-city model; COPD hosp. adms.
6. Samet et al. (2000)* – 14-city model; COPD hosp. adms.

*Reanalyzed in Zanobetti and Schwartz (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.11. Estimated Annual Percent of Mortality Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Effect of Different Lag Models‡



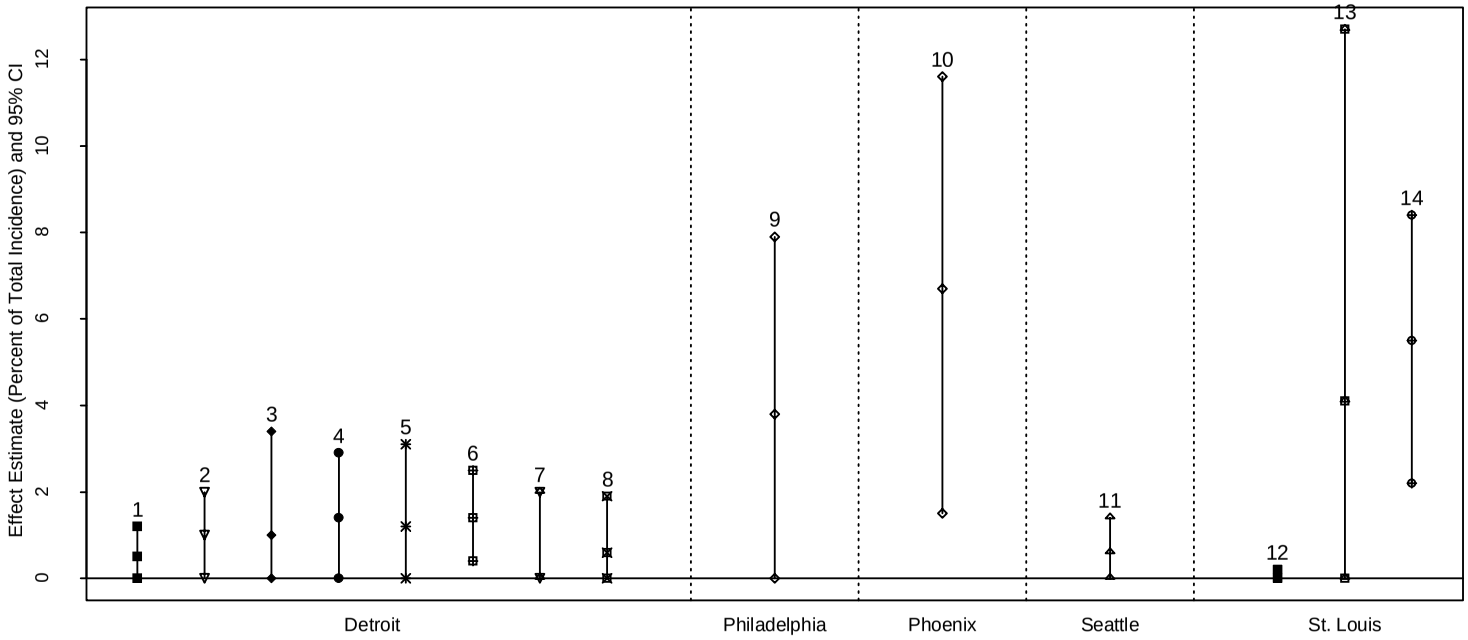
1. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 0-day lag
2. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 1-day lag
3. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 2-day lag
4. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 3-day lag
5. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 4-day lag
6. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 5-day lag
7. Schwartz (2000a)** – 10-city model; all cause mort.; mean of 0- and 1-day lags
8. Schwartz (2000b)** – 10-city model; all cause mort.; distr. lag
9. Moolgavkar (2000a)* – single-city model; cardiovasc. mort.; 0-day lag
10. Moolgavkar (2000a)* – single-city model; cardiovasc. mort.; 1-day lag
11. Moolgavkar (2000a)* – single-city model; COPD mort.; 0-day lag
12. Moolgavkar (2000a)* – single-city model; COPD mort.; 1-day lag
13. Braga et al. (2001)** – 10-city model; COPD mort.; mean of 0- and 1-day lags
14. Braga et al. (2001)** – 10-city model; COPD mort.; distr. lag
15. Braga et al. (2001)** – 10-city model; pneumonia mort.; mean of 0- and 1-day lags
16. Braga et al. (2001)** – 10-city model; pneumonia mort.; distr. lag
17. Braga et al. (2001)** – 10-city model; cardiovasc. mort.; mean of 0- and 1-day lags
18. Braga et al. (2001)** – 10-city model; cardiovasc. mort.; distr. lag

19. Mar et al. (2000)*** – cardiovasc. mort.; 0-day lag
20. Mar et al. (2000)*** – cardiovasc. mort.; 1-day lag
21. Fairley (1999)**** – non-accid. mort.; 0-day lag
22. Fairley (1999)**** – non-accid. mort.; 1-day lag

*Reanalyzed in Moolgavkar (2002); **Reanalyzed in Schwartz (2003a);
***Reanalyzed in Mar et al. (2003); ****Reanalyzed in Fairley (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.12. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{10-2.5}$ (and 95 Percent Confidence Interval)‡



1. Lippmann et al. (2000)* – non-accid. mort.
2. Lippmann et al. (2000)* – circulatory mort.
3. Lippmann et al. (2000)* – respiratory mort.
4. Lippmann et al. (2000)* – pneumonia hosp. adms.
5. Lippmann et al. (2000)* – COPD hosp. adms.
6. Lippmann et al. (2000)* – ischemic heart disease hosp. adms.
7. Lippmann et al. (2000)* – dysrhythmia hosp. adms.
8. Lippmann et al. (2000)* – congestive heart failure hosp. adms.
9. Lipfert et al. (2000) – 7-county model; cardiovasc. mort.
10. Mar et al. (2000)** – cardiovasc. mort.
11. Sheppard et al. (1999)*** – asthma hosp. adms.
12. Schwartz et al. (1996)**** – non-accid. mort.
13. Schwartz and Neas (2000)***** – 6-city model; lower resp. symptoms
14. Schwartz and Neas (2000)***** – 6-city model; cough

*Reanalyzed in Ito (2003)
**Reanalyzed in Mar et al. (2003)
****Reanalyzed in Schwartz (2003b)
*****Numbers of symptoms were in the thousands; numbers of thousands are shown in graph.

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 7.13 Legend for Health Endpoints in Figures

○	All-cause Mortality
△	Asthma Hospital Admissions
+	Asthma Symptoms
×	Cardiovascular Hospital Admissions
◇	Cardiovascular Mortality
▽	Circulatory Mortality
⊠	Congestive Heart Failure Hospital Admissions
✳	COPD Hospital Admissions
⊕	COPD Mortality
⊕	Cough
⊠	Dysrhythmias Hospital Admissions
⊞	Ischemic Heart Disease Hospital Admissions
⊠	Ischemic Heart Disease Mortality
⊠	Lower Respiratory Symptoms
■	Non-accidental Mortality
⬟	Pneumonia Hospital Admissions
▲	Pneumonia Mortality
◆	Respiratory Mortality

Exhibit 7.1. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day		130 (0 - 400)	0.7% (0.0% - 2.2%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	all	1 day		70 (0 - 250)	0.9% (0.0% - 2.9%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	all	0 day		10 (0 - 100)	0.9% (0.0% - 5.8%)
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+			500 (260 - 760)	2.7% (1.4% - 4.1%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			510 (330 - 710)	5.3% (3.5% - 7.4%)
	Pope et al. (2002) - ACS extended	All cause	30+			430 (90 - 770)	2.3% (0.5% - 4.1%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			320 (80 - 540)	3.3% (0.9% - 5.7%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			60 (10 - 100)	4.5% (0.6% - 8.2%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+		CO	720 (420 - 1030)	3.9% (2.2% - 5.5%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	860 (460 - 1220)	4.6% (2.5% - 6.6%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	720 (420 - 1030)	3.9% (2.2% - 5.5%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	130 (0 - 540)	0.7% (0.0% - 2.9%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**						
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	65+	1 day		200 (40 - 350)	3.8% (0.7% - 6.8%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD	65+	3 day		50 (0 - 200)	1.2% (0.0% - 5.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	65+	2 day		140 (0 - 360)	1.4% (0.0% - 3.6%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	65+	1 day		210 (40 - 370)	3.0% (0.5% - 5.3%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	65+	1 day		40 (0 - 160)	1.2% (0.0% - 5.0%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit 7.2. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	unconst. distr. lag		320 (180 - 450)	1.6% (0.9% - 2.3%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		160 (130 - 190)	0.8% (0.6% - 1.0%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	unconst. distr. lag		270 (200 - 330)	1.4% (1.0% - 1.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		50 (10 - 100)	0.3% (0.1% - 0.5%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	1 day		100 (0 - 270)	0.6% (0.0% - 1.5%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		40 (10 - 80)	0.2% (0.1% - 0.4%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		30 (10 - 60)	0.4% (0.1% - 0.6%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	unconst. distr. lag		80 (50 - 120)	1.2% (0.7% - 1.8%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	all	1 day		80 (0 - 180)	0.9% (0.0% - 2.2%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	unconst. distr. lag		20 (0 - 30)	2.3% (0.1% - 4.3%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	unconst. distr. lag		20 (10 - 40)	3.6% (1.9% - 5.3%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	all	0 day		20 (0 - 70)	1.2% (0.0% - 4.2%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)						
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	150 (80 - 230)	0.8% (0.4% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	210 (130 - 300)	1.1% (0.7% - 1.5%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	170 (100 - 220)	0.8% (0.5% - 1.1%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	60 (30 - 100)	0.3% (0.2% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	50 (0 - 100)	0.3% (0.0% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	50 (0 - 90)	0.3% (0.0% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	50 (10 - 100)	0.3% (0.1% - 0.5%)
Hospital Admissions	**Single Pollutant Models**						
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	65+	unconst. distr. lag		510 (380 - 640)	2.2% (1.6% - 2.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		320 (240 - 400)	1.4% (1.0% - 1.7%)
	Schwartz and Morris (1995)	Congestive heart failure	65+	mean of lag 0 & 1		80 (30 - 130)	1.2% (0.5% - 1.9%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	65+	0 day		100 (0 - 210)	1.5% (0.0% - 3.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		130 (70 - 180)	3.8% (2.0% - 5.5%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		100 (50 - 150)	3.0% (1.5% - 4.5%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD+	65+	3 day		40 (0 - 140)	1.1% (0.0% - 3.4%)
	Schwartz and Morris (1995)	Dysrhythmias	65+	1 day		20 (0 - 50)	0.7% (0.0% - 1.6%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	65+	1 day		20 (0 - 90)	0.5% (0.0% - 2.9%)
	Schwartz and Morris (1995)	Ischemic heart disease	65+	0 day		70 (20 - 120)	0.7% (0.2% - 1.2%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	65+	2 day		130 (0 - 260)	1.3% (0.0% - 2.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		60 (0 - 120)	1.2% (0.0% - 2.3%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	65+	1 day		140 (50 - 240)	2.8% (0.9% - 4.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		100 (60 - 140)	1.9% (1.2% - 2.7%)
	Multi-Pollutant Models						
	Schwartz and Morris (1995)	Congestive heart failure	65+	mean of lag 0 & 1	CO	60 (10 - 110)	0.9% (0.2% - 1.6%)
	Schwartz (1994a)	COPD	65+	0 day	O3	80 (30 - 130)	2.4% (1.1% - 3.8%)
	Schwartz and Morris (1995)	Ischemic heart disease	65+	0 day	CO	60 (10 - 110)	0.6% (0.1% - 1.1%)
	Schwartz and Morris (1995)	Ischemic heart disease	65+	0 day	SO2	60 (0 - 110)	0.6% (0.0% - 1.1%)
	Schwartz (1994a)	Pneumonia	65+	0 day	O3	70 (20 - 120)	1.4% (0.5% - 2.3%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit 7.3. Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	1 day		90 (0 - 220)	0.5% (0.0% - 1.2%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	all	1 day		80 (0 - 170)	1.0% (0.0% - 2.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	all	2 day		20 (0 - 60)	1.0% (0.0% - 3.4%)
Hospital Admissions	**Single Pollutant Models**						
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	65+	1 day		70 (0 - 150)	1.4% (0.0% - 2.9%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD+	65+	3 day		50 (0 - 120)	1.2% (0.0% - 3.1%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	65+	2 day		140 (40 - 250)	1.4% (0.4% - 2.5%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	65+	0 day		0 (0 - 60)	0.0% (0.0% - 2.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	65+	0 day		40 (0 - 130)	0.6% (0.0% - 1.9%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

As would be expected, there were substantial differences across cities, at least in part reflecting differences in air quality and populations exposed. For example, using Krewski et al. (2000) – ACS, 4.7 percent of premature mortality was associated with long-term exposure to $PM_{2.5}$ in excess of background levels in Los Angeles, which has the highest $PM_{2.5}$ levels among the assessment locations; in contrast, using the same study, only 0.7 percent of premature mortality was associated with long-term exposure to $PM_{2.5}$ in excess of background levels in Boston, which has much lower levels of $PM_{2.5}$. The corresponding incidences of premature mortality using that same study (2,730 cases in Los Angeles versus 150 cases in Boston) reflect not only differences in $PM_{2.5}$ levels in the two locations but also differences in population size (Los Angeles has a population of over 9.5 million whereas Boston's population is only about 2.8 million) (see Appendix D). Some of the differences seen in the incidences of other health effects may be due in part to differences among studies reporting the C-R functions used in the risk assessment. For example, based on the C-R function estimated in Schwartz et al. (1996) [reanalyzed in Schwartz (2003b)], 1.6 percent of short-term exposure non-accidental mortality among people of all ages was predicted to be associated with ambient $PM_{2.5}$ levels in Boston, whereas only 0.7 percent (Krewski et al., 2000 – ACS) or 0.6 percent (Pope et al., 2002 – ACS extended) of long-term exposure mortality among people age 30 and over was predicted to be associated with ambient $PM_{2.5}$ levels.

The incidence and the percent of total incidence of long-term exposure mortality was generally greater, and sometimes substantially greater than that of short-term exposure mortality in most assessment locations. This varied significantly, however, from one location to another (and may have depended on the particular studies used in the different locations). For example, in Los Angeles, 0.9 percent of short-term exposure non-accidental mortality was associated with "as is" $PM_{2.5}$ concentrations in excess of background (Moolgavkar (2000a) [reanalyzed in Moolgavkar (2002), using the GAM (stringent) model with 30 df and 0-day lag]) compared with anywhere from 4.1 percent (Pope et al., 2002 – ACS extended) to 4.7 percent (Krewski et al., 2000 – ACS) of long-term exposure mortality. In Philadelphia, however, 1.9 percent of short-term exposure non-accidental mortality was associated with "as is" $PM_{2.5}$ concentrations in excess of background, compared with anywhere from 1.8 percent (Pope et al., 2002 – ACS extended) to 2.1 percent (Krewski et al., 2000 – ACS) of long-term exposure total mortality cases.

Seattle had the lowest $PM_{2.5}$ concentrations among the assessment locations. It's annual mean of 9.3 $\mu g/m^3$ was lower than the LML (10 $\mu g/m^3$) in both Pope et al. (2002) – ACS extended and Krewski et al. (2000) – ACS. Because risks were calculated only down to the LML in the study, the risks in Seattle associated with long-term exposure mortality would have been predicted to be zero. It was therefore not included in this portion of the risk assessment.

7.2 Sensitivity analyses

Several sensitivity analyses were carried out to assess the sensitivity of the results of the first ("as is") part of the risk assessment to various assumptions underlying the analyses. In general, we carried out each sensitivity analysis listed for a given PM indicator in each of the assessment locations included for that indicator (see Exhibit 2.5). However, to reduce the number of exhibits in this Section of the report, we selected one location (Detroit) to include here for illustrative purposes. Exhibits of the results of location-specific sensitivity analyses that are not presented here are given in Appendix D. To reduce the quantity of numbers reported, with the exception of the sensitivity analysis of background concentrations we focused the sensitivity analyses on total (or non-accidental) mortality. The sensitivity analyses in this section and the exhibits presenting their results are summarized in Exhibit 7.4. They are explained briefly below.

Exhibit 7.4 Summary of Sensitivity Analyses Associated with the "As Is" Part of the Risk Assessment

Sensitivity Analysis*	Applied to	Exhibit
$PM_{2.5}$ risk assessment:		
Estimated annual health risks associated with "as is" $PM_{2.5}$ concentrations above background, using three different background levels	all health endpoints	Exhibit 7.5
Estimated annual health risks associated with "as is" PM concentrations using alternative hypothetical thresholds	mortality associated with short-term and long-term exposure	Exhibit 7.6
Estimated annual health risks associated with "as is" PM concentrations with adjustments for the estimated increases in incidence if distributed lag models had been estimated	mortality associated with short-term exposure	Exhibit 7.7
The effect of assumptions about historical air quality on estimates associated with "as is" $PM_{2.5}$ concentrations	mortality associated with long-term exposure	Exhibit 7.8
Estimated annual health risks associated with "as is" $PM_{2.5}$ concentrations using a constant background level versus different daily background levels	mortality associated with short-term and long-term exposure	(No exhibit – see text)
PM_{10} risk assessment:		
Estimated annual health risks associated with "as is" $PM_{2.5}$ concentrations above background, using three different background levels	all health endpoints	Exhibit 7.9
Estimated annual health risks associated with "as is" PM concentrations using alternative hypothetical thresholds	mortality associated with short-term exposure	Exhibit 7.10
Estimated annual health risks associated with "as is" PM concentrations with adjustments for the estimated increases in incidence if distributed lag models had been estimated	mortality associated with short-term exposure	Exhibit 7.11
$PM_{10-2.5}$ risk assessment:		

Sensitivity Analysis*	Applied to	Exhibit
Estimated annual health risks associated with "as is" $PM_{10-2.5}$ concentrations above background, using three different background levels	all health endpoints	Exhibit 7.12
Estimated annual health risks associated with "as is" $PM_{10-2.5}$ concentrations using alternative hypothetical thresholds	mortality associated with short-term exposure	Exhibit 7.13
Estimated annual health risks associated with "as is" $PM_{10-2.5}$ concentrations with adjustments for the estimated increases in incidence if distributed lag models had been estimated	mortality associated with short-term exposure	Exhibit 7.14

*Sensitivity analyses presented in this section are for Detroit, MI, unless otherwise stated.

Exhibit 7.5. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day		130 (0 - 400)	0.7% (0.0% - 2.2%)	130 (0 - 400)	0.7% (0.0% - 2.2%)	130 (0 - 400)	0.7% (0.0% - 2.2%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	all	1 day		70 (0 - 250)	0.9% (0.0% - 2.9%)	70 (0 - 250)	0.9% (0.0% - 2.9%)	70 (0 - 250)	0.9% (0.0% - 2.9%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	all	0 day		10 (0 - 100)	0.9% (0.0% - 5.8%)	10 (0 - 100)	0.9% (0.0% - 5.8%)	10 (0 - 100)	0.9% (0.0% - 5.8%)
Long-Term Exposure Mortality	**Single Pollutant Models**										
	Krewski et al. (2000) - ACS	All cause	30+			500 (260 - 760)	2.7% (1.4% - 4.1%)	500 (260 - 760)	2.7% (1.4% - 4.1%)	500 (260 - 760)	2.7% (1.4% - 4.1%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			510 (330 - 710)	5.3% (3.5% - 7.4%)	510 (330 - 710)	5.3% (3.5% - 7.4%)	510 (330 - 710)	5.3% (3.5% - 7.4%)
	Pope et al. (2002) - ACS extended	All cause	30+			430 (90 - 770)	2.3% (0.5% - 4.1%)	430 (90 - 770)	2.3% (0.5% - 4.1%)	430 (90 - 770)	2.3% (0.5% - 4.1%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			320 (80 - 540)	3.3% (0.9% - 5.7%)	320 (80 - 540)	3.3% (0.9% - 5.7%)	320 (80 - 540)	3.3% (0.9% - 5.7%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			60 (10 - 100)	4.5% (0.6% - 8.2%)	60 (10 - 100)	4.5% (0.6% - 8.2%)	60 (10 - 100)	4.5% (0.6% - 8.2%)
	Multi-Pollutant Models										
	Krewski et al. (2000) - ACS	All cause	30+		CO	720 (420 - 1030)	3.9% (2.2% - 5.5%)	720 (420 - 1030)	3.9% (2.2% - 5.5%)	720 (420 - 1030)	3.9% (2.2% - 5.5%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	860 (460 - 1220)	4.6% (2.5% - 6.6%)	860 (460 - 1220)	4.6% (2.5% - 6.6%)	860 (460 - 1220)	4.6% (2.5% - 6.6%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	720 (420 - 1030)	3.9% (2.2% - 5.5%)	720 (420 - 1030)	3.9% (2.2% - 5.5%)	720 (420 - 1030)	3.9% (2.2% - 5.5%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	130 (0 - 540)	0.7% (0.0% - 2.9%)	130 (0 - 540)	0.7% (0.0% - 2.9%)	130 (0 - 540)	0.7% (0.0% - 2.9%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**										
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	65+	1 day		200 (40 - 350)	3.8% (0.7% - 6.8%)	200 (40 - 350)	3.8% (0.7% - 6.8%)	200 (40 - 350)	3.8% (0.7% - 6.8%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD	65+	3 day		50 (0 - 200)	1.2% (0.0% - 5.0%)	50 (0 - 200)	1.2% (0.0% - 5.0%)	50 (0 - 200)	1.2% (0.0% - 5.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	65+	2 day		140 (0 - 360)	1.4% (0.0% - 3.6%)	140 (0 - 360)	1.4% (0.0% - 3.6%)	140 (0 - 360)	1.4% (0.0% - 3.6%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	65+	1 day		210 (40 - 370)	3.0% (0.5% - 5.3%)	210 (40 - 370)	3.0% (0.5% - 5.3%)	210 (40 - 370)	3.0% (0.5% - 5.3%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	65+	1 day		40 (0 - 160)	1.2% (0.0% - 5.0%)	40 (0 - 160)	1.2% (0.0% - 5.0%)	40 (0 - 160)	1.2% (0.0% - 5.0%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit 7.6. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using Atlernative Hypothetical Threshold Models*
Detroit, MI, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day		0.7% (0.0% - 2.2%)	0.5% (0.0% - 1.5%)	0.3% (0.0% - 0.9%)	0.2% (0.0% - 0.5%)
Long-Term Exposure Mortality						**BASE CASE: Background =3.5 µg/m3**	**Hypothetical Threshold =10 µg/m3**	**Hypothetical Threshold =12.5 µg/m3**	**Hypothetical Threshold =15 µg/m3**
	Single Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+			2.7% (1.4% - 4.1%)	2.7% (1.4% - 4.1%)	1.7% (0.9% - 2.6%)	0.5% (0.2% - 0.7%)
	Pope et al. (2002) - ACS extended	All cause	30+			2.3% (0.5% - 4.1%)	2.3% (0.5% - 4.1%)	1.5% (0.3% - 2.7%)	0.4% (0.1% - 0.8%)
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	3.9% (2.2% - 5.5%)	3.9% (2.2% - 5.5%)	2.4% (1.4% - 3.5%)	0.7% (0.4% - 1.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	4.6% (2.5% - 6.6%)	4.6% (2.5% - 6.6%)	2.9% (1.5% - 4.2%)	0.8% (0.4% - 1.2%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	3.9% (2.2% - 5.5%)	3.9% (2.2% - 5.5%)	2.4% (1.4% - 3.5%)	0.7% (0.4% - 1.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	0.7% (0.0% - 2.9%)	0.7% (0.0% - 2.9%)	0.4% (0.0% - 1.8%)	0.1% (0.0% - 0.5%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit 7.7. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day		130 (0 - 400)	0.7% (0.0% - 2.2%)	260 (0 - 770)	1.4% (0.0% - 4.2%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit 7.8. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations
Detroit, MI, 2002

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence*		
					Base Case: Assuming AQ as Reported	Assuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+		2.7% (1.4% - 4.1%)	1.8% (0.9% - 2.7%)	1.3% (0.7% - 2.1%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		5.3% (3.5% - 7.4%)	3.6% (2.3% - 5.0%)	2.7% (1.8% - 3.8%)
	Pope et al. (2002) - ACS extended	All cause	30+		2.3% (0.5% - 4.1%)	1.6% (0.3% - 2.8%)	1.2% (0.2% - 2.1%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+		3.3% (0.9% - 5.7%)	2.2% (0.6% - 3.8%)	1.7% (0.4% - 2.9%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+		4.5% (0.6% - 8.2%)	3.0% (0.4% - 5.5%)	2.3% (0.3% - 4.2%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+	CO	3.9% (2.2% - 5.5%)	2.6% (1.5% - 3.7%)	2.0% (1.1% - 2.8%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	4.6% (2.5% - 6.6%)	3.1% (1.6% - 4.4%)	2.3% (1.2% - 3.3%)
	Krewski et al. (2000) - ACS	All cause	30+	O3	3.9% (2.2% - 5.5%)	2.6% (1.5% - 3.7%)	2.0% (1.1% - 2.8%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	0.7% (0.0% - 2.9%)	0.5% (0.0% - 1.9%)	0.4% (0.0% - 1.4%)

* Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit 7.9. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	unconst. distr. lag		390 (220 - 560)	2.0% (1.1% - 2.8%)	320 (180 - 450)	1.6% (0.9% - 2.3%)	240 (140 - 350)	1.2% (0.7% - 1.8%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		200 (150 - 240)	1.0% (0.8% - 1.2%)	160 (130 - 190)	0.8% (0.6% - 1.0%)	120 (100 - 150)	0.6% (0.5% - 0.8%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	unconst. distr. lag		330 (250 - 410)	1.7% (1.3% - 2.1%)	270 (200 - 330)	1.4% (1.0% - 1.7%)	210 (160 - 260)	1.1% (0.8% - 1.3%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		70 (10 - 120)	0.4% (0.1% - 0.7%)	50 (10 - 100)	0.3% (0.1% - 0.5%)	40 (10 - 70)	0.2% (0.0% - 0.4%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	1 day		100 (0 - 270)	0.6% (0.0% - 1.5%)	100 (0 - 270)	0.6% (0.0% - 1.5%)	100 (0 - 270)	0.6% (0.0% - 1.5%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		50 (10 - 100)	0.3% (0.1% - 0.5%)	40 (10 - 80)	0.2% (0.1% - 0.4%)	30 (10 - 60)	0.2% (0.0% - 0.3%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		40 (10 - 70)	0.4% (0.1% - 0.7%)	30 (10 - 60)	0.4% (0.1% - 0.6%)	30 (10 - 50)	0.3% (0.1% - 0.5%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	unconst. distr. lag		100 (60 - 150)	1.5% (0.9% - 2.2%)	80 (50 - 120)	1.2% (0.7% - 1.8%)	60 (40 - 90)	0.9% (0.6% - 1.4%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	all	1 day		80 (0 - 180)	0.9% (0.0% - 2.2%)	80 (0 - 180)	0.9% (0.0% - 2.2%)	80 (0 - 180)	0.9% (0.0% - 2.2%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	unconst. distr. lag		20 (0 - 40)	2.8% (0.2% - 5.3%)	20 (0 - 30)	2.3% (0.1% - 4.3%)	10 (0 - 20)	1.8% (0.1% - 3.3%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	unconst. distr. lag		30 (20 - 40)	4.5% (2.4% - 6.5%)	20 (10 - 40)	3.6% (1.9% - 5.3%)	20 (10 - 30)	2.8% (1.5% - 4.1%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	all	0 day		20 (0 - 70)	1.2% (0.0% - 4.2%)	20 (0 - 70)	1.2% (0.0% - 4.2%)	20 (0 - 70)	1.2% (0.0% - 4.2%)
	Multi-Pollutant Models (Total Mortality)										
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	190 (100 - 280)	1.0% (0.5% - 1.4%)	150 (80 - 230)	0.8% (0.4% - 1.2%)	120 (60 - 180)	0.6% (0.3% - 0.9%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	260 (160 - 370)	1.3% (0.8% - 1.9%)	210 (130 - 300)	1.1% (0.7% - 1.5%)	160 (100 - 230)	0.8% (0.5% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	200 (130 - 280)	1.0% (0.7% - 1.4%)	170 (100 - 220)	0.8% (0.5% - 1.1%)	130 (80 - 170)	0.7% (0.4% - 0.9%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	80 (30 - 120)	0.4% (0.2% - 0.7%)	60 (30 - 100)	0.3% (0.2% - 0.5%)	50 (20 - 80)	0.3% (0.1% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	60 (0 - 120)	0.3% (0.0% - 0.7%)	50 (0 - 100)	0.3% (0.0% - 0.5%)	40 (0 - 80)	0.2% (0.0% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	60 (10 - 120)	0.3% (0.0% - 0.6%)	50 (0 - 90)	0.3% (0.0% - 0.5%)	40 (0 - 70)	0.2% (0.0% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	70 (10 - 120)	0.4% (0.1% - 0.7%)	50 (10 - 100)	0.3% (0.1% - 0.5%)	40 (10 - 70)	0.2% (0.1% - 0.4%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**										
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	65+	unconst. distr. lag		640 (480 - 790)	2.7% (2.0% - 3.3%)	510 (380 - 640)	2.2% (1.6% - 2.7%)	400 (300 - 500)	1.7% (1.2% - 2.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		400 (300 - 500)	1.7% (1.3% - 2.1%)	320 (240 - 400)	1.4% (1.0% - 1.7%)	250 (190 - 310)	1.0% (0.8% - 1.3%)
	Schwartz and Morris (1995)	Congestive heart failure	65+	mean of lag 0 & 1		100 (40 - 170)	1.5% (0.6% - 2.4%)	80 (30 - 130)	1.2% (0.5% - 1.9%)	60 (20 - 100)	0.9% (0.4% - 1.5%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	65+	0 day		100 (0 - 210)	1.5% (0.0% - 3.0%)	100 (0 - 210)	1.5% (0.0% - 3.0%)	100 (0 - 210)	1.5% (0.0% - 3.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		160 (80 - 230)	4.7% (2.5% - 6.8%)	130 (70 - 180)	3.8% (2.0% - 5.5%)	100 (50 - 140)	2.9% (1.6% - 4.2%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		120 (60 - 190)	3.7% (1.8% - 5.5%)	100 (50 - 150)	3.0% (1.5% - 4.5%)	80 (40 - 120)	2.3% (1.1% - 3.5%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD+	65+	3 day		40 (0 - 140)	1.1% (0.0% - 3.4%)	40 (0 - 140)	1.1% (0.0% - 3.4%)	40 (0 - 140)	1.1% (0.0% - 3.4%)
	Schwartz and Morris (1995)	Dysrhythmias	65+	1 day		30 (0 - 70)	0.9% (0.0% - 2.0%)	20 (0 - 50)	0.7% (0.0% - 1.6%)	20 (0 - 40)	0.6% (0.0% - 1.3%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	65+	1 day		20 (0 - 90)	0.5% (0.0% - 2.9%)	20 (0 - 90)	0.5% (0.0% - 2.9%)	20 (0 - 90)	0.5% (0.0% - 2.9%)
	Schwartz and Morris (1995)	Ischemic heart disease	65+	0 day		80 (20 - 150)	0.8% (0.2% - 1.5%)	70 (20 - 120)	0.7% (0.2% - 1.2%)	50 (10 - 90)	0.5% (0.2% - 0.9%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	65+	2 day		130 (0 - 260)	1.3% (0.0% - 2.6%)	130 (0 - 260)	1.3% (0.0% - 2.6%)	130 (0 - 260)	1.3% (0.0% - 2.6%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		70 (0 - 150)	1.4% (0.0% - 2.9%)	60 (0 - 120)	1.2% (0.0% - 2.3%)	50 (0 - 90)	0.9% (0.0% - 1.8%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	65+	1 day		140 (50 - 240)	2.8% (0.9% - 4.6%)	140 (50 - 240)	2.8% (0.9% - 4.6%)	140 (50 - 240)	2.8% (0.9% - 4.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		120 (70 - 170)	2.4% (1.4% - 3.4%)	100 (60 - 140)	1.9% (1.2% - 2.7%)	80 (50 - 110)	1.5% (0.9% - 2.1%)
	Multi-Pollutant Models										
	Schwartz and Morris (1995)	Congestive heart failure	65+	mean of lag 0 & 1	CO	80 (10 - 140)	1.1% (0.2% - 2.0%)	60 (10 - 110)	0.9% (0.2% - 1.6%)	50 (10 - 90)	0.7% (0.1% - 1.3%)
	Schwartz (1994a)	COPD	65+	0 day	O3	100 (40 - 160)	3.0% (1.3% - 4.7%)	80 (30 - 130)	2.4% (1.1% - 3.8%)	60 (30 - 100)	1.9% (0.8% - 2.9%)
	Schwartz and Morris (1995)	Ischemic heart disease	65+	0 day	CO	80 (10 - 140)	0.8% (0.1% - 1.4%)	60 (10 - 110)	0.6% (0.1% - 1.1%)	50 (10 - 90)	0.5% (0.1% - 0.9%)
	Schwartz and Morris (1995)	Ischemic heart disease	65+	0 day	SO2	70 (0 - 140)	0.7% (0.1% - 1.4%)	60 (0 - 110)	0.6% (0.0% - 1.1%)	40 (0 - 90)	0.4% (0.0% - 0.9%)
	Schwartz (1994a)	Pneumonia	65+	0 day	O3	90 (30 - 150)	1.7% (0.6% - 2.9%)	70 (20 - 120)	1.4% (0.5% - 2.3%)	60 (20 - 90)	1.1% (0.4% - 1.8%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit 7.10. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations Using Alternative Hypothetical Threshold Models*
Detroit, MI, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-10 Above Hypothetical Threshold***			
						BASE CASE: Background =8 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	Hypothetical Threshold =30 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		0.3% (0.1% - 0.5%)	0.2% (0.0% - 0.3%)	0.1% (0.0% - 0.2%)	0.1% (0.0% - 0.1%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	1 day		0.6% (0.0% - 1.5%)	0.5% (0.0% - 1.2%)	0.3% (0.0% - 0.8%)	0.1% (0.0% - 0.4%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit 7.11. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		160 (130 - 190)	0.8% (0.6% - 1.0%)	310 (250 - 380)	1.6% (1.3% - 1.9%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		50 (10 - 100)	0.3% (0.1% - 0.5%)	100 (20 - 190)	0.6% (0.1% - 1.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	1 day		100 (0 - 270)	0.6% (0.0% - 1.5%)	200 (0 - 520)	1.1% (0.0% - 2.8%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		40 (10 - 80)	0.2% (0.1% - 0.4%)	90 (20 - 150)	0.5% (0.1% - 0.8%)
	Multi-Pollutant Models (Total Mortality)								
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	150 (80 - 230)	0.8% (0.4% - 1.2%)	300 (160 - 450)	1.5% (0.8% - 2.3%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	210 (130 - 300)	1.1% (0.7% - 1.5%)	420 (260 - 580)	2.1% (1.3% - 2.9%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	170 (100 - 220)	0.8% (0.5% - 1.1%)	330 (200 - 440)	1.7% (1.0% - 2.2%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	60 (30 - 100)	0.3% (0.2% - 0.5%)	120 (50 - 190)	0.7% (0.3% - 1.1%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	50 (0 - 100)	0.3% (0.0% - 0.5%)	90 (0 - 190)	0.5% (0.0% - 1.1%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	50 (0 - 90)	0.3% (0.0% - 0.5%)	90 (10 - 190)	0.5% (0.1% - 1.0%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	50 (10 - 100)	0.3% (0.1% - 0.5%)	110 (20 - 190)	0.6% (0.1% - 1.0%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit 7.12. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations, Using Different Background Levels
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background: Background = 0		Health Effects Associated with PM10-2.5 Above Background: Background = 3		Health Effects Associated with PM10-2.5 Above Background: Background = 9	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	1 day		90 (0 - 220)	0.5% (0.0% - 1.2%)	90 (0 - 220)	0.5% (0.0% - 1.2%)	30 (0 - 70)	0.2% (0.0% - 0.4%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	all	1 day		80 (0 - 170)	1.0% (0.0% - 2.0%)	80 (0 - 170)	1.0% (0.0% - 2.0%)	30 (0 - 60)	0.3% (0.0% - 0.7%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	all	2 day		20 (0 - 60)	1.0% (0.0% - 3.4%)	20 (0 - 60)	1.0% (0.0% - 3.4%)	10 (0 - 20)	0.3% (0.0% - 1.1%)
Hospital Admissions	**Single Pollutant Models**										
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	65+	1 day		70 (0 - 150)	1.4% (0.0% - 2.9%)	70 (0 - 150)	1.4% (0.0% - 2.9%)	20 (0 - 50)	0.5% (0.0% - 1.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD+	65+	3 day		50 (0 - 120)	1.2% (0.0% - 3.1%)	50 (0 - 120)	1.2% (0.0% - 3.1%)	20 (0 - 40)	0.4% (0.0% - 1.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	65+	2 day		140 (40 - 250)	1.4% (0.4% - 2.5%)	140 (40 - 250)	1.4% (0.4% - 2.5%)	50 (10 - 80)	0.5% (0.1% - 0.8%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	65+	0 day		0 (0 - 60)	0.0% (0.0% - 2.0%)	0 (0 - 60)	0.0% (0.0% - 2.0%)	0 (0 - 20)	0.0% (0.0% - 0.7%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	65+	0 day		40 (0 - 130)	0.6% (0.0% - 1.9%)	40 (0 - 130)	0.6% (0.0% - 1.9%)	10 (0 - 40)	0.2% (0.0% - 0.6%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East (with a range from 0.0 to 7.0 ug/m3) and 3.0 ug/m3 in the West (with a range from 0.0 to 9.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

Exhibit 7.13. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{10-2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
Detroit, MI, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM10-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =3 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	1 day		0.5% (0.0% - 1.2%)	0.1% (0.0% - 0.4%)	0.1% (0.0% - 0.2%)	0.0% (0.0% - 0.1%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

Exhibit 7.14. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{10-2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background: Single Lag		Health Effects Associated with PM10-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	1 day		90 (0 - 220)	0.5% (0.0% - 1.2%)	170 (0 - 430)	0.9% (0.0% - 2.4%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

The first set of sensitivity analyses, shown in Exhibits 7.5, 7.9, and 7.12 for $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$, respectively, examine the effect of background concentration on the estimated effect of PM concentrations above background in Detroit. The results for the other assessment locations are shown in Appendix D. In many cases, changing the assumed background concentration had a significant effect. For example, changing from the midpoint estimate of 8 $\mu g/m^3$ for PM_{10} background in the Eastern U.S. to the lower end of the range for PM_{10} background (5 $\mu g/m^3$) increased the estimated percent of total incidence that is PM_{10}-related using Schwartz (2003a) [reanalysis of Schwartz (2000b)] in Detroit by about 24 percent (from 1.6 percent to 2.0 percent). Changing from the midpoint estimate to the upper end of the range for PM_{10} (11 $\mu g/m^3$) decreased the percent of total incidence that is PM_{10}-related using that same study to about 78 percent of what it was (from 1.6 percent to 1.2 percent). In some cases, there was no effect, because all three background levels used were lower than the lowest measured level (LML) in the study. For example, the LML for $PM_{2.5}$ in Ito (2003) [reanalysis of Lippmann et al. (2000)] was 6 $\mu g/m^3$, which is larger than all three $PM_{2.5}$ background levels considered in the sensitivity analysis for $PM_{2.5}$ in the Eastern U.S. (2, 3.5, and 5 $\mu g/m^3$). Therefore changing background levels had no effect using that study. In general, however, unless the LML exceeded the upper end of the range of background concentrations, changing the assumed background concentration had a significant impact on the results.

The second set of sensitivity analyses address the issue of possible thresholds below which there may be no PM effects. Hypothetical thresholds of 10, 15, and 20 $\mu g/m^3$ were used for mortality associated with short-term exposure to both $PM_{2.5}$ and $PM_{10-2.5}$; hypothetical thresholds of 10, 12.5, and 15 $\mu g/m^3$ were used for mortality associated with long-term exposure to $PM_{2.5}$; and hypothetical thresholds of 15, 20, and 30 μg/m3 were used for mortality associated with short-term exposure to PM_{10}.

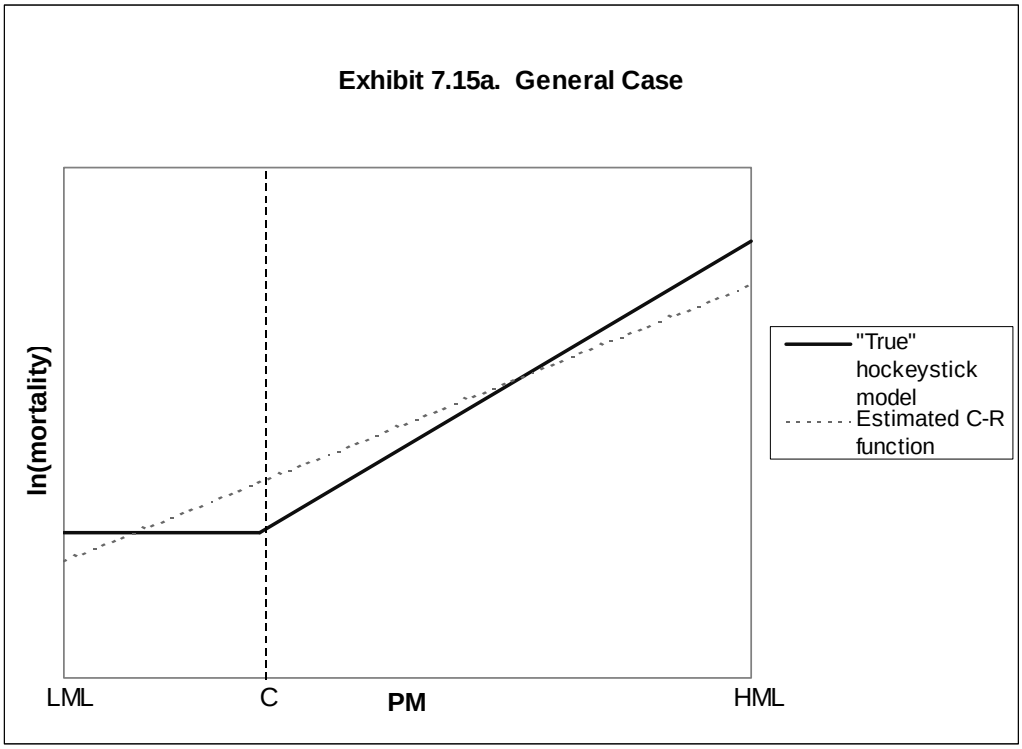
The rationale for considering hypothetical thresholds is the hypothesis that there is some PM concentration below which there are no PM-related effects. If the original data in the study reporting a C-R function supported a threshold hypothesis, then the model that best fit the data would look like a hockeystick – that is, there would be no relationship (i.e., a horizontal line) between PM and the health endpoint below (to the left of) the hypothetical threshold, and there would be an upward-sloping relationship (e.g., linear or log-linear) above (to the right of) the hypothetical threshold. If this is the case, then the log-linear model that was fit to the original data in the study would not have been the appropriate functional form.

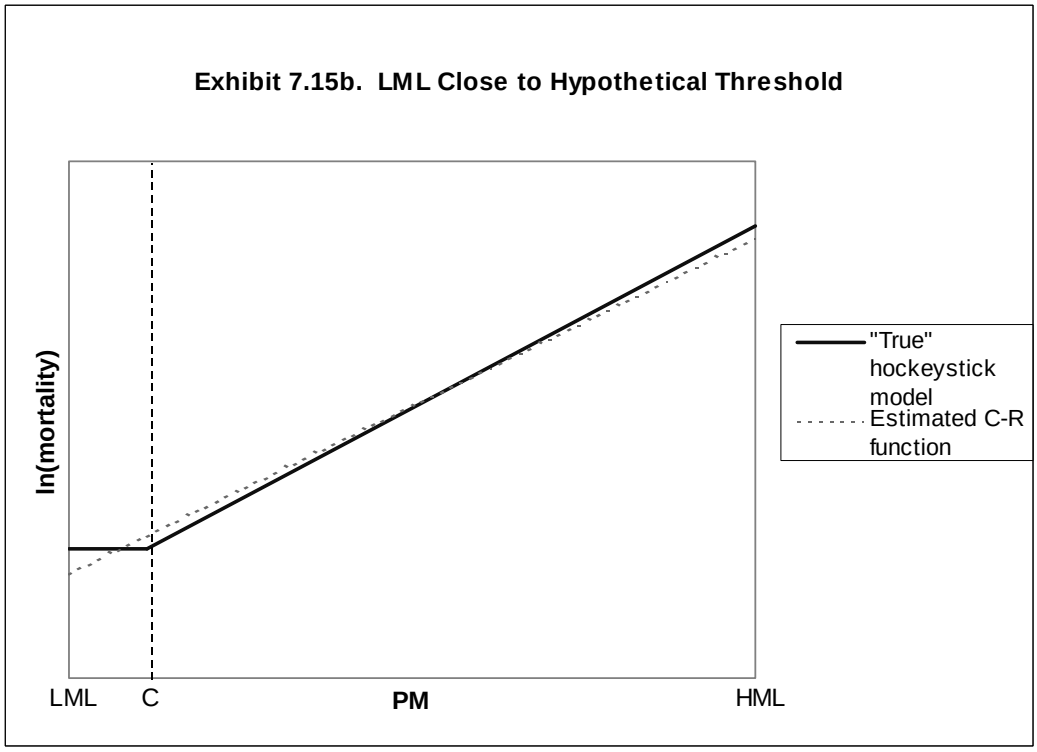
If the researchers in the original study fit a log-linear or a linear model through data that actually better support a hockeystick functional form, the slope of the fitted curve would be smaller than the slope of the upward-sloping portion of the "true" hockeystick relationship, as shown in Exhibit 7.15a. The horizontal portion of the data below the hypothetical threshold would essentially cause the estimated slope to be biased downward relative to the "true" slope of the upward-sloping portion of the hockeystick. The slope of the upward-sloping portion of the

hockeystick model should therefore be adjusted upward (from the slope of the reported C-R function) – i.e., if the data in the original study actually supported a hockeystick model better than a log-linear model, then the log-linear fitted curve reported by the study would have understated the degree to which PM is associated with mortality above the hypothetical threshold, as shown in Exhibit 7.15a. This rationale applies equally in the case of long- and short-term exposure mortality. In each case, under the threshold hypothesis a log-linear curve has been fit to data that are better characterized by a hockeystick model. In the case of a short-term exposure mortality study, the curve represents the relationship between daily PM and daily mortality; in the case of a long-term exposure mortality study, the curve represents the relationship between annual average PM and annual mortality. In both cases, however, if the "true" relationship looks like a hockeystick, then the log-linear curve fitted to the data would understate the impact of increases in PM (either daily, in the case of a short-term study, or annual average, in the case of a long-term study) on mortality at PM levels above the hypothetical threshold.

If the data used in a study do not extend down below the hypothetical threshold or extend only slightly below it, then the extent of the downward bias of the reported PM coefficient will be minimal. This is the case, for example, when the hypothetical threshold is 10 $\mu g/m^3$ or 12.5 $\mu g/m^3$ for long-term exposure mortality, given that the LMLs in the long-term exposure mortality studies were 10 $\mu g/m^3$ and 11 $\mu g/m^3$. In this case, the data in the study provided hardly any information about the relationship between $PM_{2.5}$ and mortality at levels below the hypothetical thresholds and would have biased an estimate of the slope of the upward-sloping portion of a hockeystick only minimally if at all, as illustrated in Exhibit 7.15b.

Exhibit 7.15 Relationship Between Estimated Log-Linear Concentration-Response Function and Hockeystick Model





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

- LML denote the lowest measured PM level in the study,
- c denote the hypothetical threshold,
- HML denote the highest measured PM level in the study,
- β^{est} denote the slope (the PM coefficient) estimated in the study (using a log-linear model), and
- β^{T} denote the "true" slope of the upward-sloping portion of the hockeystick,

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

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

and, solving for β^{T},

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

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

Exhibits 7.6, 7.10, and 7. 13 show the percentages of total incidence predicted to be associated with $PM_{2.5}$, PM_{10}, and $PM_{10\text{-}2.5}$, respectively, in excess of background in Detroit, using the original C-R function as well as each of the hypothetical thresholds, using the slope adjustment method described above. The results for the other assessment locations are shown in Appendix D. Because the method of adjusting the slope of the positive-sloped portion of the C-R function requires the HML of PM in the study, this sensitivity analysis was carried out only on those mortality C-R functions from studies for which this maximum concentration was available. For some combinations of urban area and PM indicator, the HML was unavailable for all of the

total (or non-accidental) mortality studies, eliminating the exhibit entirely. This was the case, for example, for PM_{10} in Boston and PM_{10} in Phoenix.

The slope adjustment method used in these sensitivity analyses is intended only to be illustrative rather than definitive. Different choices of slope adjustment methods can yield substantially different results. A better evaluation of the effect of hypothetical thresholds would require re-analysis of original health and air quality data.

The third sensitivity analysis attempts to estimate how different the results would be if the C-R functions used had been distributed lag models rather than single lag models, using the results of a study by Schwartz (2000b). Schwartz (2000b) estimated constrained and unconstrained distributed lag C-R functions for PM_{10} and daily deaths of persons 65 years and older in 10 U.S. cities. Using an unconstrained distributed lag model, he estimated a 1.29% increase in mortality associated with an increase of 10 $\mu g/m^3$ PM_{10}. Using a constrained model (which assumed that the effect all occurs in one day) he estimated a 0.65% increase associated with a 10 $\mu g/m^3$ increase in PM_{10} (see Schwartz, 2000b, Table 3). The PM_{10} coefficient corresponding to the constrained model result is 0.00065. The PM_{10} coefficient corresponding to the unconstrained model (i.e., the value that a single coefficient would have to be to result in a relative risk of 1.013) is 0.00128. The ratio of those coefficients is 1.98. That is, a distributed lag model predicted the same relative risk that a single lag model would have predicted if the coefficient were 1.98 times what it was estimated to be. To simulate what the results might have been had a distributed lag model been estimated instead of a single lag model, we multiplied the PM coefficients for total mortality by 1.98. The results are shown for Detroit in Exhibits 7.7, 7.11, and 7.14 for $PM_{2.5}$, PM_{10}, and $PM_{10\text{-}2.5}$, respectively (and for the other urban areas in exhibits in Appendix D). As would be expected, the results are almost double using the distributed lag approximation.

An important source of uncertainty in applying the long-term exposure studies in a risk assessment is what the relevant period of exposure is and the extent, if any, of a lag period between exposure and effects. If air quality was historically 50 percent higher than the levels measured in the long-term exposure mortality studies, and if the historical air quality levels were the relevant levels, then the $PM_{2.5}$ coefficients that would have been estimated using the historical $PM_{2.5}$ levels would have been two-thirds (=1/1.5) the coefficients that were actually estimated in the studies. Similarly, if air quality was historically twice the levels measured in the long-term exposure mortality studies, and if the historical air quality levels were the relevant levels, then the $PM_{2.5}$ coefficients that would have been estimated using the historical $PM_{2.5}$ levels would have been half (=1/2.0) the coefficients that were actually estimated in the studies. The impact of varying assumptions about historical air quality on estimates of long-term exposure mortality associated with "as is" $PM_{2.5}$ concentrations is shown for Detroit in Exhibit 7.8. The results for the other assessment locations are shown in Appendix D.

To assess the impact of using different daily background $PM_{2.5}$ concentrations on the estimates of risk associated with "as is" $PM_{2.5}$ concentrations in excess of background, two distributions of background levels, one for the East (including Boston, Philadelphia, St. Louis, and Detroit), and one for the West (including San Jose, Los Angeles, Phoenix, and Seattle), were developed. The Eastern distribution was lognormal with a mean of 3.5 $\mu g/m^3$ and a standard deviation of 3.8. The Western distribution was lognormal with a mean of 2.5 $\mu g/m^3$ and a standard deviation of 1.5. The standard deviations were based on the standard deviations of daily $PM_{2.5}$ measurements from the Interagency Monitoring of Protected Visual Environments (IMPROVE) program. IMPROVE is a cooperative visibility monitoring effort between the EPA, federal land management agencies, and state air agencies. One of the functions of this program is to monitor visibility and aerosol conditions in Class I areas, and for the most part the IMPROVE monitors are located in rural areas. IMPROVE data from 1988 to 1999 were used in this analysis. All IMPROVE sites measure some $PM_{2.5}$ from anthropogenic sources, and this typically will inflate the standard deviation over what it would be if we were able to measure background concentrations as defined for purposes of these analyses.[37] Because of this, OAQPS selected the smallest standard deviation among all IMPROVE sites in the East (3.8) and in the West (1.5) (Langstaff, 2003). These were not substantially smaller than the next two smallest standard deviations (3.8 and 4.0 in the East, and 1.6 and 1.6 in the West).

It is likely that background levels are positively correlated with "as is" levels in a given location. To reflect this likelihood, we assigned a background level to each day that was the same percentile in the distribution of daily background levels as the corresponding "as is" level was in its distribution. For example, the "as is" level at the composite monitor in Boston on May 29, 2002 was 20.2 $\mu g/m^3$. This was the 88.2nd percentile point of the distribution of "as is" $PM_{2.5}$ concentrations at the composite monitor in Boston in 2002. The 88.2nd percentile point of the background distribution for the East is 5.2 $\mu g/m^3$, which was designated the background level on May 29, 2002 in Boston. We examined both incidences and percentages of total incidence, using a constant background and different daily backgrounds. In almost all cases the differences in the results are so small that they do not show up after rounding. We therefore do not present the results in exhibits.

Finally, we considered a sensitivity analysis to examine the effect of estimating the risks of short-term exposure mortality associated with "as is" $PM_{2.5}$ and PM_{10} concentrations separately by season in San Jose, CA, for which Fairley et al. (2003) report season-specific C-R functions. However, in both cases the annual estimates were substantially greater than the sum of the seasonal estimates, and it is not clear why this is the case.

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[37] Recall that "background" is defined as the PM concentrations that would be observed in the U.S. in the absence of anthropogenic, or man-made, emissions of primary PM and precursor emissions of volatile organic compounds, nitrogen oxides, sulfur dioxide, and ammonia in North America. Thus, "background" for the purposes of the PM risk assessment includes PM from natural sources and transport of PM from sources outside of North America.

8. Assessment of the Health Risk Reductions Associated with Just Meeting the Current and Alternative $PM_{2.5}$ Standards

The second part of the risk assessment estimates the risk reductions that would result if the current annual $PM_{2.5}$ standard of 15 μg/m^3 and the current daily $PM_{2.5}$ standard of 65 μg/m^3 were just met in the assessment locations. Several of the assessment locations (Boston, Phoenix, San Jose, and Seattle) already meet the current $PM_{2.5}$ standards. This part of the risk assessment therefore considers only those locations that do not meet the current standards (Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis). The results of the base case analysis are shown in Exhibit 8.1 for Detroit, and in Appendix E for the other urban areas that do not meet the current $PM_{2.5}$ standards (Los Angeles, Philadelphia, Pittsburgh, and St. Louis). Reductions in incidence are shown in these exhibits both as a percent of PM-related incidence and as a percent of total incidence.[38] Differences between lower bound, central, and upper bound estimates of incidence reduction as a percent of PM-related incidence were generally quite small, and in some cases were lost in the rounding.[39] The percent reductions in health risks across urban areas for mortality associated with short-term and long-term exposures to $PM_{2.5}$ are summarized in Figures 8.1 and 8.2, respectively. The corresponding estimated reductions in incidence across urban areas are shown in Appendix E, in Figures E.1 and E.2 for short-term and long-term mortality, respectively.

The plausibility of proportional rollbacks to simulate the pattern by which daily $PM_{2.5}$ concentrations would change if an urban area just met the current $PM_{2.5}$ standards is discussed briefly in Section 2.4 and in more detail in Appendix B. Although an examination of the evidence suggests that proportional rollbacks are a reasonable way to simulate the change in daily $PM_{2.5}$ concentrations, there are other patterns of changes that are also plausible. We examined one such pattern, in which the highest $PM_{2.5}$ concentrations are reduced more than the rest of the $PM_{2.5}$ concentrations. In particular, in this sensitivity analysis, we hypothesized that the top 10 percent of the distribution of $PM_{2.5}$ concentrations is reduced by 1.6 times as much as the lower 90 percent of concentrations. We examined the effects of this hypothesis on incidence reductions that would result from meeting the annual standard because it was the controlling standard in all five study areas that do not meet the current $PM_{2.5}$ standards based on year 2002 air quality data.

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[38] Incidence reduction as a percent of PM-related incidence was calculated by dividing the incidence reduction achieved by rolling back $PM_{2.5}$ above background to just meet the current standards by the incidence associated with "as is" $PM_{2.5}$ above background. In those cases in which the incidence associated with "as is" $PM_{2.5}$ above background was estimated to be zero, this percent could therefore not be calculated.

[39] Both the numerator and the denominator in the percent of PM-related incidence depend on the PM coefficient. Both become smaller when the lower bound of the 95 percent confidence interval around the PM coefficient is used, and both become larger when the upper bound of the 95 percent confidence interval around the PM coefficient is used. As a result, the percent changes very little when either the lower bound or the upper bound of the 95 percent confidence interval of the PM coefficient is used instead of the PM coefficient itself.

Exhibit 8.1. Estimated Annual Health Risk Reductions Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**							
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day		50 (0 - 140)	36.2% (* - 36.5%)	0.3% (0.0% - 0.8%)
	Single Pollutant Models (Cause-Specific Mortality)							
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	all	1 day		30 (0 - 90)	36.3% (* - 36.7%)	0.3% (0.0% - 1.1%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	all	0 day		10 (0 - 40)	36.3% (* - 37.4%)	0.3% (0.0% - 2.2%)
Long-Term Exposure Mortality	**Single Pollutant Models**							
	Krewski et al. (2000) - ACS	All cause	30+			310 (160 - 480)	62.6% (62.5% - 62.8%)	1.7% (0.9% - 2.5%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			320 (210 - 450)	63.0% (62.7% - 63.2%)	3.4% (2.2% - 4.7%)
	Pope et al. (2002) - ACS extended	All cause	30+			270 (50 - 480)	62.6% (62.4% - 62.8%)	1.5% (0.3% - 2.6%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			200 (50 - 340)	62.7% (62.4% - 63.0%)	2.1% (0.5% - 3.6%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			40 (10 - 70)	62.9% (62.4% - 63.3%)	2.8% (0.4% - 5.2%)
	Multi-Pollutant Models							
	Krewski et al. (2000) - ACS	All cause	30+		CO	450 (260 - 650)	62.8% (62.6% - 63.0%)	2.4% (1.4% - 3.5%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	540 (290 - 770)	62.9% (62.6% - 63.1%)	2.9% (1.5% - 4.1%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	450 (260 - 650)	62.8% (62.6% - 63.0%)	2.4% (1.4% - 3.5%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	80 (0 - 340)	62.4% (* - 62.7%)	0.4% (0.0% - 1.8%)

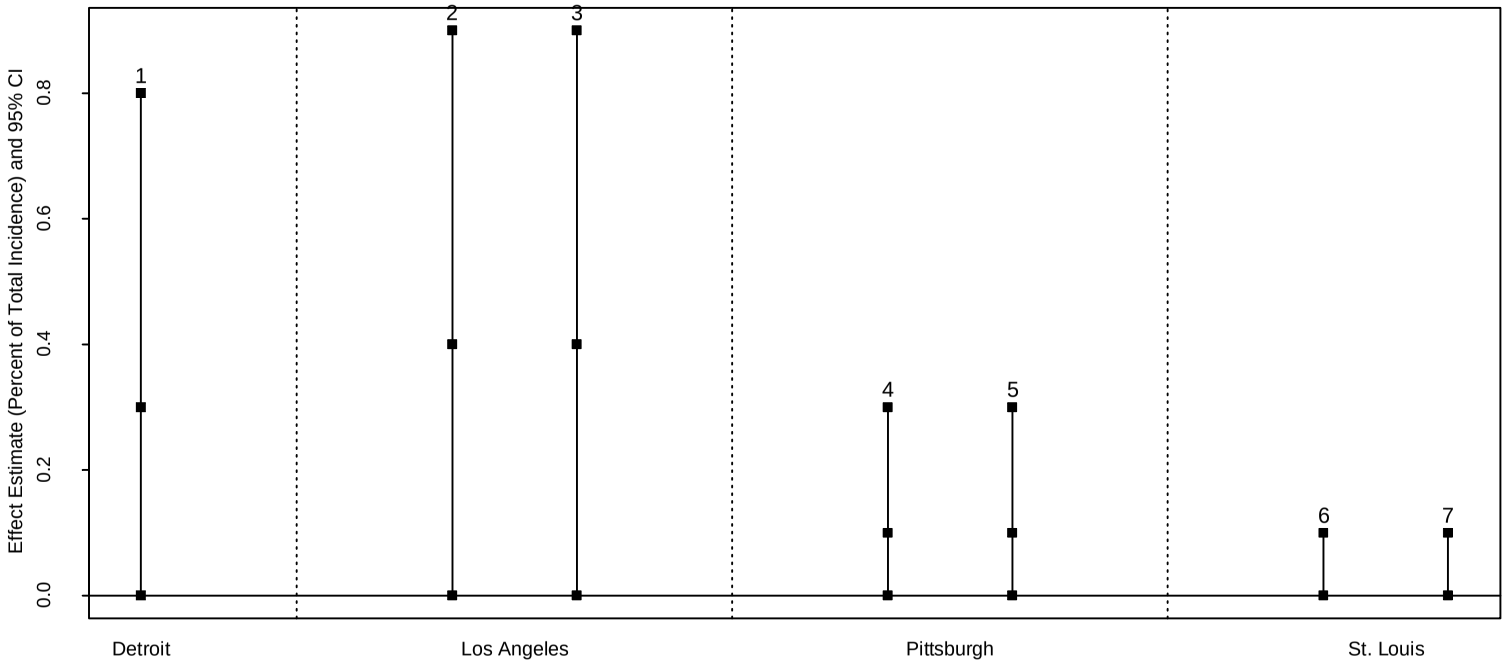
Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**							
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	65+	1 day		70 (10 - 130)	36.9% (36.2% - 37.6%)	1.4% (0.3% - 2.5%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD	65+	3 day		20 (0 - 70)	36.3% (* - 37.2%)	0.4% (0.0% - 1.8%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	65+	2 day		50 (0 - 130)	36.4% (* - 36.9%)	0.5% (0.0% - 1.3%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	65+	1 day		80 (10 - 140)	36.7% (36.2% - 37.3%)	1.1% (0.2% - 2.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	65+	1 day		10 (0 - 60)	36.3% (* - 37.2%)	0.5% (0.0% - 1.8%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. An asterisk indicates that the lower bound could not be calculated.

Figure 8.1. Estimated Annual Percent Reduction of Health Risks Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 (and 95 Percent Confidence Interval): Non-accidental Mortality Associated with Short-Term Exposure to $PM_{2.5}$ ‡

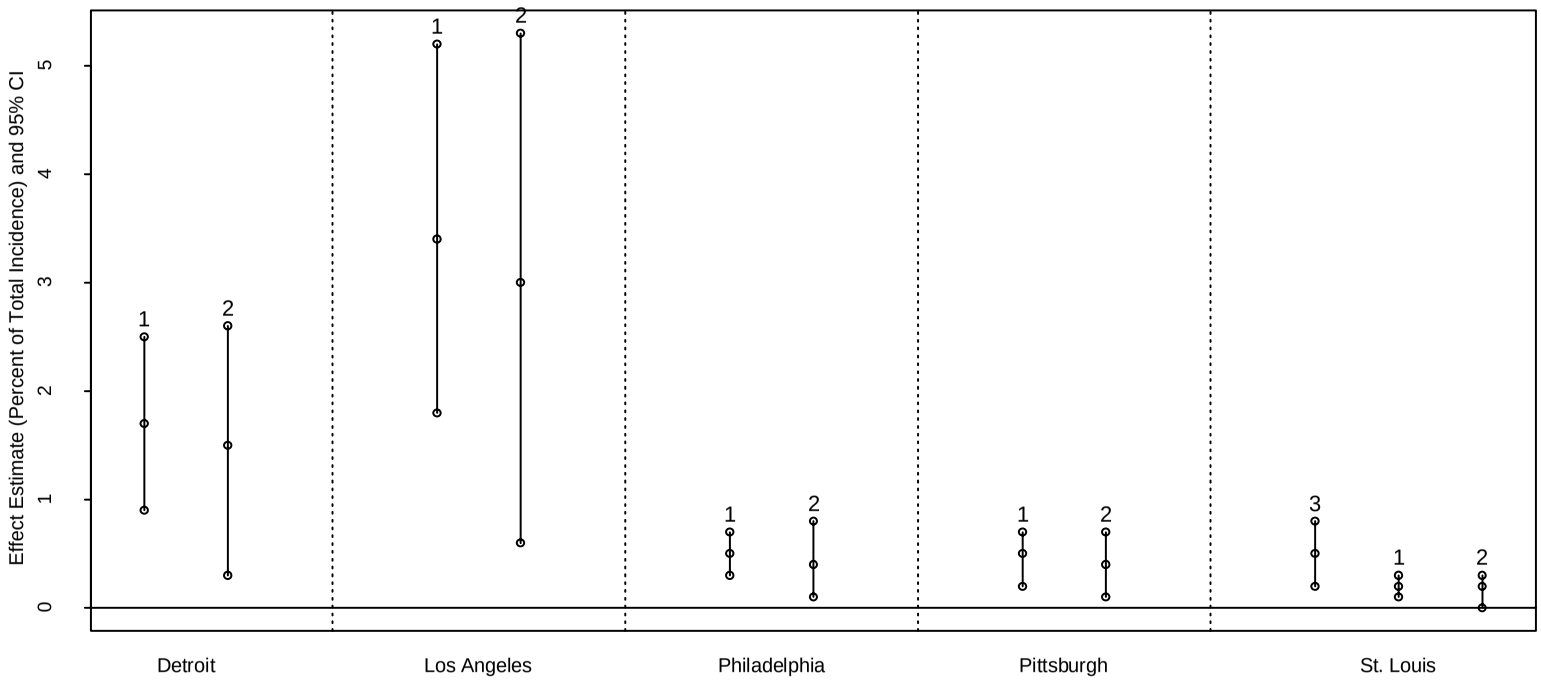


1. Lippmann et al. (2000)*
2. Moolgavkar (2000a)** – 0-day lag
3. Moolgavkar (2000a)** – 1-day lag
4. Chock et al. (2000) (age<75)
5. Chock et al. (2000) (age>75)
6. Schwartz et al. (1996)***
7. Schwartz et al. (1996)*** – 6-city model

*Reanalyzed in Ito (2003)
**Reanalyzed in Moolgavkar (2002)
***Reanalyzed in Schwartz (2003b)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 8.2. Estimated Annual Percent Reduction of Health Risks Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 (and 95 Percent Confidence Interval): Mortality Associated with Long-Term Exposure to $PM_{2.5}$‡



1. Krewski et al. (2000) – ACS
2. Pope et al. (2002) – ACS extended
3. Krewski et al. (2000) – Six Cities

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

To meet the annual standard, the annual average must not exceed the annual standard of 15 μg/m^3. If

- p_a denotes the percent rollback necessary to just meet the annual standard if all days are rolled back the same proportion,
- aa denotes the annual average
- b denotes background level,
- $a_{0.9}$ denotes the average of the lower 90 percent of concentrations,
- $a_{0.1}$ denotes the average of the upper 10 percent of concentrations,
- and x denotes the percent rollback that would be applied to the lower 90 percent of the distribution of concentrations, if 1.6x is the percent rollback applied to the upper 10 percent of the concentrations, so that the resulting rolled back annual average just attains the annual standard, then

$$x = p_a * (aa - b) / (0.9 * (a_{0.9} - b) + 0.1 * 1.6 * (a_{0.1} - b)) \ .$$

The results of this sensitivity analysis are shown for Detroit in Exhibit 8.2. The results for the other assessment locations that do not meet the current standards are shown in Appendix E. The results are based on the controlling standard, which, in all cases, is the annual standard of 15 μg/m^3.

Exhibit 8.2. Sensitivity Analysis: Estimated Annual Reductions of Short-Term and Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 Using an Alternative Rollback Method
Detroit, MI, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Annual and Daily Standards	Percent Change in PM-Associated Incidence**		Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback Method
							All PM concentrations rolled back equally	Percent rollback of upper 10% of AQ distribution = 1.6 x percent rollback of lower 90% of AQ distribution	
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day		15 ug/m3 annual 65 ug/m3 daily	36.2% 0.0%	36.4% 0.0%	100.6% --
Long-Term Exposure Mortality	**Single Pollutant Models**								
	Krewski et al. (2000) - ACS	All cause	30+			15 ug/m3 annual 65 ug/m3 daily	62.6% 0.0%	62.9% 0.0%	100.5% --
	Pope et al. (2002) - ACS extended	All cause	30+			15 ug/m3 annual 65 ug/m3 daily	62.6% 0.0%	62.8% 0.0%	100.3% --
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	15 ug/m3 annual 65 ug/m3 daily	62.8% 0.0%	63.0% 0.0%	100.3% --
	Krewski et al. (2000) - ACS	All cause	30+		NO2	15 ug/m3 annual 65 ug/m3 daily	62.9% 0.0%	63.1% 0.0%	100.3% --
	Krewski et al. (2000) - ACS	All cause	30+		O3	15 ug/m3 annual 65 ug/m3 daily	62.8% 0.0%	63.0% 0.0%	100.3% --
	Krewski et al. (2000) - ACS	All cause	30+		SO2	15 ug/m3 annual 65 ug/m3 daily	62.4% 0.0%	62.6% 0.0%	100.3% --

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Only those C-R functions for which rollbacks are predicted to result in a positive number of cases avoided are included.

Finally, the percent rollback necessary to just meet the annual standards depends on whether the maximum or the spatial average of the monitor-specific annual averages is used. Exhibit 8.3 shows the percent rollbacks that would be required to just meet the current annual standard using each of the two measures in each of the assessment locations which do not currently meet the annual standard.

Exhibit 8.3 Air Quality Adjustments Required to Just Meet the Current Annual $PM_{2.5}$ Standard of 15 μg/m^3 Using the Maximum vs. the Average of Monitor-Specific Averages

Assessment Location	Percent Rollback Necessary to Just Meet the Annual $PM_{2.5}$ Standard	
	Using Maximum of Monitor-Specific Annual Averages	Using Average of Monitor-Specific Annual Averages
Detroit	29.5%	10.9%
Los Angeles	41.9%	30.2%
Philadelphia	9.5%	0.0%
Pittsburgh	8.7%	0.0%
St. Louis	3.4%	0.0%

9. Assessment of the Health Risk Reductions Associated with Just Meeting Alternative $PM_{10-2.5}$ Standards (to be added in the next draft of this report)

References

Abt Associates Inc. July 3, 1996 (Revised November 1996). "A Particulate Matter Risk Assessment for Philadelphia and Los Angeles." Prepared for the Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Contract No. 68-W4-0029. Available electronically on the web at: http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_pr_td.html .

Abt Associates Inc. June 5, 1997a. Revision of Mortality Incidence Estimates Based on Pope et al. (1995) in the Abt Particulate Matter Risk Assessment Report. Memorandum from Ellen Post and John Voyzey, Abt Associates Inc. to John Bachmann, Allyson Siwik, Michele McKeever, and Harvey Richmond, U.S. EPA/OAQPS.

Abt Associates Inc. June 6, 1997b. Revision of Mortality Incidence Estimates Based on Pope et al. (1995) in the December 1996 Supplement to the Abt Particulate Matter Risk Assessment Report. Memorandum from Ellen Post, Abt Associates Inc. to John Bachmann, Allyson Siwik, Michele McKeever, and Harvey Richmond, U.S. EPA/OAQPS.

Braga, A. L. F., A. Zanobetti, J. Schwartz. 2001. The Lag Structure Between Particulate Air Pollution and Respiratory and Cardiovascular Deaths in 10 US Cities. Journal of Occupational and Environmental Medicine, vol 43, num 11.

Chock, D., S. Winkler, C. Chen. 2000. A Study of the Association between Daily Mortality and Ambient Air Pollutant Concentrations in Pittsburgh, Pennsylvania. Journal of the Air and Waste Management Association, 50:1481-1500.

Deck, L. B., E. S. Post, E. Smith, M. Wiener, K. Cunningham, and H. Richmond. 2001 Estimates of the Health Risk Reductions Associated with Attainment of Alternative Particulate Matter Standards in Two U.S. Cities. Risk Analysis Vol. 21(5): 821-835.

Dominici, F., et. al. 2003. National Morbidity and Mortality Air Pollution Study, Updated City-Specific Estimates of PM10 Relative Risks. Available electronically on the internet at: http://www.biostat.jhsph.edu/biostat/research/web.est.xls.

Fairley, D. 1999. Daily mortality and air pollution in Santa Clara County, California: 1989-1996. Environmental Health Perspectives. Vol. 107(8): 637-41.

Fairley, D. 2003. Mortality and Air Pollution for Santa Clara County, California, 1989-1996. Health Effects Institute, Cambridge, MA.

HEI, May 2003. Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute, Cambridge, MA.

Hopke, P., 2002. Letter from Dr. Phil Hopke, Chair, Clean Air Scientific Advisory Committee (CASAC) Particulate Matter Review Panel, to Honorable Christine Todd Whitman, Administrator, U.S. EPA. Final advisory review report by the CASAC Particulate Matter Review Panel on the proposed particulate matter risk assessment. EPA-SAB-CASAC-ADV-02-002. May 23. Available electronically on the internet at: http://www.epa.gov/sab/pdf/casacadv02002.pdf

Ito, K. 2003. Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan. Health Effects Institute, Cambridge, MA.

Ito, K., P. L. Kinney, and G.D. Thurston 1995. Variations in PM-10 Concentrations Within Two Metropolitan Areas and Their Implications For Health Effects Analyses. Inhalation Toxicology 7(5): 735-745.

Ito, K.; Thurston, G. D. 1996. Daily PM10/mortality associations: an investigation of at-risk subpopulations. J. Exposure Anal. Environ. Epidemiol. 6:79-95.

Kinney, P. L.; Ito, K.; Thurston, G. D. 1995. A sensitivity analysis of mortality/PM-10 associations in Los Angeles. Inhalation Toxicology. 7:59-69.

Klemm, R. J., R. M. Mason, C. M. Heilig, L. M. Neas, D. W. Dockery, 2000. Is Daily Mortality Associated Specifically with Fine Particles? Journal of the Air Waste Management Association, 50:1215-1222.

Klemm, R. J., R. Mason, 2003. Replication of Reanalysis of Harvard Six-City Study. Health Effects Institute, Cambridge, MA.

Krewski, D. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report. Health Effects Institute, Cambridge, MA.

Langstaff, J. 2003. "PM risk assessment: Variability of $PM_{2.5}$ background concentrations." EPA memorandum to file. August 15, 2003.

Linn, W. S.; Szlachcic, Y.; Gong, H., Jr.; Kinney, P. L.; Berhane, K. T. 2000. Air pollution and daily hospital admissions in metropolitan Los Angeles. Environmental Health Perspectives. 108: 427-434.

Lipfert, F.W., S.C. Morris and R.E. Wyzga. 2000. Daily mortality in the Philadelphia metropolitan area and size-classified particulate matter. Journal of the Air and Waste Management Association. Vol. 50(8): 1501-13.

Lippmann, M., K. Ito, A. Náádas, and R.T. Burnett. 2000. Association of Particulate Matter Components with Daily Mortality and Morbidity in Urban Populations. Research Report 95. Health Effects Institute, Cambridge, MA.

Mar, T.F., G.A. Norris, J.Q. Koenig and T.V. Larson. 2000. Associations between air pollution and mortality in Phoenix, 1995-1997. Environmental Health Perspectives. Vol. 108(4): 347-53.

Mar, T. F., G. A. Norris, T. V. Larson, W. E. Wilson, J. Q. Koenig. 2003. Air Pollution and Cardiovascular Mortality in Phoenix, 1995-1997. Health Effects Institute, Cambridge, MA.

Moolgavkar, S.H. 2000a. Air Pollution and Daily Mortality in Three U.S. Counties. Environmental Health Perspectives. Vol. 108(8): 777-784.

Moolgavkar, S.H. 2000b. Air pollution and hospital admissions for diseases of the circulatory system in three U.S. metropolitan areas. Journal of the Air and Waste Management Association. Vol. 50(7): 1199-206.

Moolgavkar, S.H. 2000c. Air Pollution and Hospital Admissions for Chronic Obstructive Pulmonary Disease in Three Metropolitan Areas in the United States. Inhalation Toxicology. Vol. 12(Supplement 4): 75-90.

Moolgavkar, S. H. 2003. Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties. Health Effects Institute, Cambridge, MA.

Moolgavkar, S.H., W. Hazelton and G. Luebeck. 2000. Air Pollution, Pollens, and Admissions for Chronic Respiratory Disease in King County, Washington. Inhalation Toxicology. Vol. 12(Supplement 1): 157-171.

Morris, R.D., and E.N. Naumova. 1998. Carbon Monoxide and Hospital Admissions for Congestive Heart Failure: Evidence of an Increased Effect at Low Temperatures. Environmental Health Perspectives. 106: 649-653.

Nauenberg, E.; Basu, K. 1999. Effect of insurance coverage on the relationship between asthma hospitalizations and exposure to air pollution. Public Health Rep. 114: 135-148.

Pope, C. A., III; D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. Respiratory Health and PM10 Pollution. Am Rev. Respir Dis 144:668-674.

Pope, C. A., III; Hill, R. W.; Villegas, G. M. 1999. Particulate air pollution and daily mortality on Utah's Wasatch Front. Environmental Health Perspectives. 107:567-573.

Pope, C. A., R. T. Burnett, M. J. Thun, E. E. Calle, D. Krewski, K. Ito, G. D. Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. Journal of the American Medical Association, vol 287, no 9: 287:1132-1141.

Post, E., L. Deck, K. Larntz, D. Hoaglin. 2001. An Application of an Empirical Bayes Estimation Technique to the Estimation of Mortality Related to Short-Term Exposure to Particulate Matter. Risk Analysis Vol. 21(5): 837-842.

Post, E. April 8, 2003. "Preliminary Recommended Methodology for PM_{10} and $PM_{10-2.5}$ Risk Analyses in Light of Reanalyzed Study Results." Draft memorandum to Harvey Richmond, U.S. EPA/OAQPS. Available electronically on the internet at: http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_td.html

Romieu, I., F. Meneses, S. Ruiz, J.J. Sienra, J. Huerta, M.C. White and R.A. Etzel. 1996. Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. Am J Respir Crit Care Med. Vol. 154(2 Pt 1): 300-7.

Samet, J.M., S.L. Zeger, F. Dominici, F. Curriero, I. Coursac, D.W. Dockery, J. Schwartz, and A. Zanobetti. 2000. The National Morbidity, Mortality, and Air Pollution Study, Part II: Morbidity, Mortality, and Air Pollution in the United States. Research Report 94, Part II. Health Effects Institute, Cambridge, MA

Schwartz, J. 1994a. Air pollution and hospital admissions for the elderly in Detroit, Michigan. Am J Resp Crit Care Med 159:648-655.

Schwartz, J. 1994b. PM10, ozone, and hospital admissions for the elderly in Minneapolis, MN. Arch. Environ. Health 49:366-374.

Schwartz, J. 2000a. Assessing Confounding, Effect Modification, and Thresholds in the Association between Ambient particles and Daily Deaths. Environmental Health Perspectives. 108 (6): 563 - 568.

Schwartz, J. 2000b. The distributed lag between air pollution and daily deaths. Epidemiology 11(3): 320-326.

Schwartz, J. 2003a. Airborne Particles and Daily Deaths in 10 US Cities. Health Effects Institute, Cambridge, MA.

Schwartz, J. 2003b. Daily Deaths Associated with Air Pollution in Six US Cities and Short-Term Mortality Displacement in Boston. Health Effects Institute, Cambridge, MA.

Schwartz, J., D.W. Dockery, L.M. Neas, D. Wypij, J.H. Ware, J.D. Spengler, P. Koutrakis, F.E. Speizer and B.G. Ferris. 1994. Acute Effects of Summer Air Pollution On Respiratory Symptom Reporting in Children. Am J Respir Crit Care Med. Vol. 150(5): 1234-1242.

Schwartz, J. and R. Morris. 1995. Air Pollution and Hospital Admissions for Cardiovascular Disease in Detroit, Michigan. American Journal of Epidemiology, vol 142, no 1, 142:23-35.

Schwartz, J., D.W. Dockery and L.M. Neas. 1996. Is Daily Mortality Associated Specifically With Fine Particles. Journal of the Air & Waste Management Association. Vol. 46(10): 927-939.

Schwartz, J. and L.M. Neas. 2000. Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren [see comments]. Epidemiology. Vol. 11(1): 6-10.

Sheppard, L. 2003. Ambient Air Pollution and Non-Elderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994. Health Effects Institute, Cambridge, MA.

Sheppard, L.; Levy, D.; Norris, G.; Larson, T. V.; Koenig, J. Q. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. Epidemiology 10: 23-30.

Styer, P.; McMillan, N.; Gao, F.; Davis, J.; Sacks, J. 1995. Effect of outdoor airborne particulate matter on daily death counts. Environmental Health Perspectives. 103:490-497.

U.S. Environmental Protection Agency (U.S. EPA). 1996a. Air Quality Criteria for Particulate Matter, (EPA 600/P-95/001aF), 3v, National Center for Environmental Assessment, Office of Research and Development, Research Triangle Park, NC. Available electronically on the internet at: http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_pr_cd.html

U.S. Environmental Protection Agency (U.S. EPA) July 1996b. Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information - OAQPS Staff Paper, (EPA/452/R-96-013), Office of Air Quality Planning and Standards, Research Triangle Park, NC 27711. Available from: NTIS, Springfield, VA; PB97-115406REB or electronically on the internet at:
http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_pr_sp.html

U.S. Environmental Protection Agency (U.S. EPA) June 2001a. Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information - OAQPS Staff Paper, Preliminary Draft, Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available electronically on the internet at:

http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html

U.S. Environmental Protection Agency (U.S. EPA) June 2003a. Air Quality Criteria for Particulate Matter, Fourth External Review Draft (EPA 600/P-99/002aD and bD..01), 2v, National Center for Environmental Assessment, Office of Research and Development, Research Triangle Park, NC. Available electronically on the internet at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=58003

U.S. Environmental Protection Agency (U.S. EPA) August 2003b. Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information - OAQPS Staff Paper, Draft, Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available electronically on the internet at: http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html

Yu, O., L. Sheppard, T. Lumley, J. Q. Koenig, G. Shapiro. 2000. Effects of Ambient Air Pollution on Symptoms of Asthma in Seattle-Area Children Enrolled in the CAMP Study. Environmental Health Perspectives, vol 108 no 12, 108:1209-1214.

Zanobetti, A. and J. Schwartz. 2003. Airborne Particles and Hospital Admissions for Heart and Lung Disease. Health Effects Institute, Cambridge, MA.

Appendix A. Air Quality Assessment: The PM Data

Appendix A. Air Quality Assessment: The PM Data

This Appendix describes the PM data for the urban counties used in the risk assessment (see Section 3 for selection of locations). The average ambient PM concentration in an assessment location on a given day is represented by the *average* of 24-hour average PM levels at the different monitors in that location that reported on that day. This approach is consistent with what has been done in epidemiological studies estimating PM C-R functions. Also, because people are often quite mobile (e.g., living in one part of a county and working in another), an area-wide average PM level may be a more meaningful measure of ambient PM concentration than PM levels at individual monitors. Ito et al. (1995), for example, 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.

In order for an urban area to be included in a PM risk assessment (e.g., the PM_{10} risk assessment), the location must contain at least one monitor (for the $PM_{10\text{-}2.5}$ risk assessment, at least one pair of co-located PM_{10} and $PM_{2.5}$ monitors) with 11 or more observations per quarter. Because there are substantially more monitoring data for $PM_{2.5}$ than for PM_{10}, we added the additional criterion for the $PM_{2.5}$ risk assessment that there be at least 122 observations per year (1 in 3 day monitoring). Once the criteria for inclusion were met, all monitors with at least 11 observations per quarter were used for each location. The cutoff of 11 observations per quarter is based on EPA guidance on measuring attainment of the daily and annual particulate matter standards outlined in Appendix N of the July 18, 1997 Federal Register Notice (available on the web at www.epa.gov/ttn/oarpg/t1pfpr.html). The guidance requires that at least 75 percent of the scheduled sampling days for each quarter have valid data. Based on a one in six day sampling protocol, the minimum required number of observations would be 11 per quarter.

The numbers of days of observations by monitor and at the composite monitor, by quarter and for the year, along with annual averages and 98th percentile concentrations, are given in the exhibits below. In these exhibits the first five digits, which denote the FIPS code designation, are omitted in the legends. The annual average at each monitor, and at the composite monitor, is the average of the four quarterly averages at the monitor. The 98th percentile at each monitor, and at the composite monitor, is calculated using the method used by EPA, as described in Appendix N of the July 18, 1997 Federal Register Notice (available on the web at www.epa.gov/ttn/oarpg/t1pfpr.html). The only difference between the method used in the risk assessment and the standard EPA convention in calculating annual averages and 98th percentile values is that the EPA convention uses three years of data whereas the risk assessment is based on only a single year of data (which is equivalent to assuming three identical years).

A.1. The $PM_{2.5}$ data

$PM_{2.5}$ data for each of the urban areas identified in Section 3 for the $PM_{2.5}$ risk assessment (Boston, Detroit, Los Angeles, Philadelphia, Phoenix, Pittsburgh, San Jose, Seattle, and St. Louis) were obtained for the years 1999 through 2002 from EPA's Aerometric Information Retrieval System (AIRS). For all urban areas except Phoenix and San Jose, year 2002 data were used. For Phoenix and San Jose, there were no monitors in 2002 that met the inclusion criterion, so year 2001 data were used. The numbers of days of observations by monitor and at the composite monitor, by quarter and for the year, along with annual averages and 98th percentile concentrations, are given in Exhibits A.1 through A.9.

The maximum of monitor-specific annual averages is used to determine the percent rollback necessary to meet an annual standard; the highest monitor-specific 98th percentile value is used to determine the percent rollback necessary to meet a daily standard. Although the composite monitor is not used in determining the percent rollback in the PM risk assessment, the percent rollback to simulate just meeting alternative standards is applied to the composite monitor.

Exhibit A.1. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. Boston, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 250170008881011	27	29	19	28	103	11.3	29.2
AIRS 250210007881011	29	30	31	28	118	12.2	48.1
AIRS 250250042881011	52	50	79	83	264	11.3	33.0
AIRS 250250043881011	21	24	29	12	86	13.5	29.8
Composite[1]	66	62	85	86	299	11.4	33.0

*All concentrations are in $\mu g/m^3$; includes Middlesex, Norfolk and Suffolk Counties.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.2. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. Detroit, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 261630001881011	79	72	66	87	304	15.9	39.6
AIRS 261630015881011	27	27	25	29	108	17.4	38.2
AIRS 261630016881011	74	82	75	88	319	15.6	42.7
AIRS 261630019881011	26	28	28	30	112	15.6	34.4
AIRS 261630025881011	18	28	29	28	103	14.4	32.7
AIRS 261630033881011	29	28	29	30	116	19.8	45.7
AIRS 261630036881011	24	28	28	25	105	16.3	34.1
Composite[1]	87	90	87	92	356	15.8	36.7

*All concentrations are in $\mu g/m^3$; includes Wayne County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.3. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. Los Angeles, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 060370002881011	72	88	88	80	328	21.0	51.0
AIRS 060371002881011	30	30	31	31	122	24.0	55.2
AIRS 060371103881011	87	87	88	62	324	22.1	55.3
AIRS 060371201881011	28	30	31	31	120	18.9	45.4
AIRS 060371301881011	30	30	31	31	122	23.3	52.5
AIRS 060371601881011	30	30	31	27	118	24.0	57.9
AIRS 060372005881011	30	29	31	31	121	20.3	49.1
AIRS 060374002881011	85	90	91	91	356	19.5	47.1
AIRS 060379033881011	23	27	28	29	107	10.4	20.0
Composite[1]	90	91	92	92	365	20.4	47.2

*All concentrations are in $\mu g/m^3$; includes Los Angeles County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.4. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. Philadelphia, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 421010004881011	80	84	80	89	333	14.8	40.9
AIRS 421010020881011	27	30	29	31	117	14.4	39.7
AIRS 421010024881011	29	30	29	26	114	14.3	38.2
AIRS 421010047881011	26	22	29	30	107	16.2	38.7
AIRS 421010136881011	75	84	88	84	331	14.4	42.9
Composite[1]	90	91	92	92	365	14.5	40.9

*All concentrations are in $\mu g/m^3$; includes Philadelphia County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.5. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. Phoenix, 2001*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 040130019881011	50	85	91	86	312	10.9	30.4
AIRS 040139990881011	27	31	28	30	116	9.4	22.7
AIRS 040139992881011	77	87	85	87	336	10.9	35.3
AIRS 040139997881011	75	75	91	73	314	9.2	25.0
Composite[1]	88	90	92	92	362	10.4	28.9

*All concentrations are in $\mu g/m^3$; includes Maricopa County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.6. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. Pittsburgh, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 420030008881011	85	82	87	91	345	15.4	41.4
AIRS 420030067881011	28	22	26	23	99	12.2	47.6
AIRS 420030093881011	14	12	13	12	51	13.5	52.5
AIRS 420030095881011	14	11	14	12	51	13.5	46.2
AIRS 420030116881011	30	22	23	23	98	13.9	38.4
AIRS 420031008881011	30	28	26	26	110	16.1	46.2
Composite[1]	86	86	89	91	352	15.2	41.4

*All concentrations are in $\mu g/m^3$; includes Allegheny County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.7. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. San Jose, 2001*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 060850004881012	80	16	16	82	194	12.4	57.5
Composite[1]	80	16	16	82	194	12.4	57.5

*All concentrations are in $\mu g/m^3$; includes Santa Clara County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.8. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. Seattle, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 530330017881011	30	27	30	30	117	5.7	16.6
AIRS 530330021881011	87	87	84	85	343	10.3	29.9
AIRS 530330024881011	29	29	28	30	116	10.8	34.8
AIRS 530330027881011	28	30	28	31	117	7.8	22.5
AIRS 530330037881011	30	30	31	26	117	8.3	22.1
AIRS 530330080881011	82	90	90	92	354	8.6	24.9
AIRS 530332004881011	32	52	28	31	143	10.3	34.4
Composite[1]	90	91	92	92	365	9.3	24.8

*All concentrations are in $\mu g/m^3$; includes King County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.9. Number of Days on which $PM_{2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{2.5}$ Concentrations. St. Louis, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 171192009881011	26	30	31	31	118	14.7	34.5
AIRS 171634001881011	26	29	27	30	112	15.1	37.2
AIRS 290990012881011	29	30	81	83	223	15.1	39.8
AIRS 291831002881011	29	28	30	31	118	14.2	35.0
AIRS 291890004881011	28	28	31	31	118	13.0	34.0
AIRS 291892003881011	30	30	68	90	218	14.6	35.6
AIRS 291895001881011	30	30	31	31	122	13.4	38.4
AIRS 295100007881011	84	89	85	91	349	15.3	36.5
AIRS 295100085881011	90	91	92	92	365	15.4	36.5
AIRS 295100086881011	89	87	92	88	356	14.3	35.5
Composite[1]	90	91	92	92	365	14.7	34.2

*All concentrations are in $\mu g/m^3$; includes St. Louis (MO), Franklin (MO), Jefferson (MO), St. Charles (MO), Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City (MO).

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

A.2. The PM_{10} data

PM_{10} data for each of the urban areas identified in Section 3 for the PM_{10} risk assessment (Boston, Chicago, Detroit, Los Angeles, Minneapolis, Philadelphia, Phoenix, Pittsburgh, Provo, San Jose, Seattle, and St. Louis) were obtained for the years 1999 through 2002 from EPA's Aerometric Information Retrieval System (AIRS). For all urban areas except Boston and San Jose, year 2002 data were used. For Boston and San Jose, there were no monitors in any year after 1999 that met the inclusion criterion, so year 1999 data were used.

PM_{10} data for all cities were obtained from monitors measuring concentrations at standard temperature and pressure, because significantly more AIRS PM_{10} data are reported under standard conditions than local conditions. The numbers of days of observations by monitor and at the composite monitor, by quarter and for the year, along with annual averages and maximum concentrations, are given in Exhibits A.10 through A.21 for each of the locations in the PM_{10} risk assessment.

Exhibit A.10. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Boston, 1999*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 250250024811021	11	14	15	12	52	24.3	50
Composite[1]	11	14	15	12	52	24.3	50

*All concentrations are in $\mu g/m^3$; includes Middlesex, Norfolk and Suffolk Counties.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.11. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Chicago, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 170310001811022	15	15	15	15	60	23.0	46
AIRS 170310060811021	14	15	15	15	59	31.0	63
AIRS 170311901811021	12	15	15	15	57	23.1	44
AIRS 170312001811021	14	15	15	14	58	27.0	52
AIRS 170314101811021	13	15	16	14	58	23.6	67
Composite[1]	15	15	16	15	61	25.7	55

*All concentrations are in $\mu g/m^3$; includes Cook County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.12. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Detroit, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 261630001811021	15	15	15	14	59	20.2	49
Composite[1]	15	15	15	14	59	20.2	49

*All concentrations are in $\mu g/m^3$; includes Wayne County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.13. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Los Angeles, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 060370002811022	14	15	16	12	57	45.8	79
AIRS 060371002811022	14	14	16	14	58	37.7	71
AIRS 060374002811022	14	15	15	14	58	36.0	62
AIRS 060375001811021	15	15	16	15	61	37.2	97
AIRS 060376012811021	15	15	16	14	60	33.3	56
AIRS 060379033811021	14	15	14	15	58	29.7	48
Composite[1]	18	19	20	18	75	36.6	55

*All concentrations are in $\mu g/m^3$; includes Los Angeles County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.14. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Minneapolis, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 270531007811021	15	14	15	15	59	23.6	48
AIRS 270532006811021	15	15	15	15	60	21.2	48
AIRS 271230021811021	15	14	16	14	59	25.8	51
AIRS 271230866811021	15	15	16	14	60	32.2	58
AIRS 271230868811021	59	57	38	15	169	28.6	62
AIRS 271231003811021	12	15	16	14	57	29.1	59
Composite[1]	65	62	41	15	183	27.5	61

*All concentrations are in $\mu g/m^3$; includes Hennepin and Ramsey Counties.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.15. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Philadelphia, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 421010004811021	12	15	14	15	56	22.3	51
AIRS 421010037811021	15	13	15	11	54	26.5	83
AIRS 421010149811021	11	15	16	14	56	24.6	71
AIRS 421010449811021	11	11	15	14	51	25.1	64
Composite[1]	15	15	16	15	61	25.4	72

*All concentrations are in $\mu g/m^3$; includes Philadelphia County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.16. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Phoenix, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 040130019811021	15	15	16	15	61	52.5	98
AIRS 040131003811021	15	15	16	15	61	37.9	86
AIRS 040131004811021	15	15	16	14	60	36.9	72
AIRS 040132001811021	15	15	16	14	60	40.3	85
AIRS 040133002811022	15	15	16	15	61	43.1	76
AIRS 040133003811021	15	15	16	15	61	36.7	62
AIRS 040133006811021	15	15	13	13	56	44.6	90
AIRS 040133007811022	14	15	16	15	60	80.5	174
AIRS 040133010811021	15	15	16	15	61	54.6	102
AIRS 040134003811021	15	15	16	15	61	59.5	123
AIRS 040134004811021	15	15	16	15	61	38.5	77
AIRS 040134006811021	13	15	16	14	58	62.5	134
AIRS 040134007811021	14	14	16	15	59	31.9	67
AIRS 040139812811021	15	15	16	15	61	69.9	158
AIRS 040139993811021	15	15	15	14	59	28.8	78
Composite[1]	15	15	16	15	61	47.9	83.9

*All concentrations are in $\mu g/m^3$; includes Maricopa County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.17. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Pittsburgh, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 420030067811021	13	15	16	13	57	19.3	54
AIRS 420030092811021	13	14	16	15	58	23.2	61
AIRS 420030095811021	13	13	16	13	55	18.5	59
Composite[1]	13	15	16	15	59	20.5	58

*All concentrations are in $\mu g/m^3$; includes Allegheny County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.18. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Provo, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 490490002811022	29	30	30	31	120	28.6	72
Composite[1]	29	30	30	31	120	28.6	72

*All concentrations are in $\mu g/m^3$; includes Utah County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.19. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. San Jose, 1999*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 060850004811024	13	15	12	15	55	24.6	77
Composite[1]	13	15	12	15	55	24.6	77

*All concentrations are in $\mu g/m^3$; includes Santa Clara County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.20. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. Seattle, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 530332004811022	15	15	16	15	61	18.0	44
Composite[1]	15	15	16	15	61	18.0	44

*All concentrations are in $\mu g/m^3$; includes King County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.21. Number of Days on which PM_{10} Concentration Data are Available, by Monitor and by Quarter, and PM_{10} Concentrations. St. Louis, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 171630010811021	15	13	15	15	58	29.8	93
AIRS 291895001811022	15	15	16	15	61	16.7	36
Composite[1]	15	15	18	15	63	22.8	69

*All concentrations are in $\mu g/m^3$; includes St. Louis (MO), Franklin (MO), Jefferson (MO), St. Charles (MO), Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City (MO).

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

A.3. The $PM_{10-2.5}$ data

$PM_{10-2.5}$ data for each of the urban areas except Phoenix identified in Section 3 for the $PM_{10-2.5}$ risk assessment (Detroit, Philadelphia, Seattle, and St. Louis) were calculated based on data obtained from EPA's Aerometric Information Retrieval System (AIRS) for 1999-2001. PM_{10} and $PM_{2.5}$ monitoring data collected on the same day at the same site were used to calculate $PM_{10-2.5}$ levels. Year 2002 data were used for Detroit, Philadelphia, and St. Louis. In Seattle, the most recent year for which there was at least one set of co-located monitors satisfying the completeness criterion was 2000.

In the AIRS database, $PM_{2.5}$ data is collected and reported at local temperature and pressure. In order to calculate $PM_{10-2.5}$ levels using comparable data, PM_{10} local condition data were obtained, when available, and PM_{10} standard condition data were converted to PM_{10} local condition data using site-specific algorithms.

For Phoenix, TEOM data for the years 1995 through 1998 were obtained from William Wilson, U.S. EPA, NCEA (personal communication, June 4, 2003). $PM_{10-2.5}$ concentrations were calculated from co-located PM_{10} and $PM_{2.5}$ monitor data. $PM_{10-2.5}$ data from the year 1997 were used for Phoenix in the $PM_{10-2.5}$ risk assessment.

The numbers of days of observations by monitor and at the composite monitor, by quarter and for the year, along with annual averages and maximum concentrations, are given in Exhibits A.22 through A.26 for each of the urban locations in the $PM_{10-2.5}$ risk assessment. Since $PM_{10-2.5}$ data are based on co-located monitors at a single site, the data are presented by site, rather than by monitor. As with the PM_{10} and $PM_{2.5}$ data, the annual average at each site, and at the composite site, is the average of the four quarterly averages at the site.

Exhibit A.22. Number of Days on which $PM_{10-2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{10-2.5}$ Concentrations. Detroit, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 261630001	14	12	11	14	51	6.5	23.5
Composite[1]	14	12	11	14	51	6.5	23.5

*All concentrations are in $\mu g/m^3$; includes Wayne County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.23. Number of Days on which $PM_{10-2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{10-2.5}$ Concentrations. Philadelphia, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 421010004	11	14	14	15	54	9.2	22.1
Composite[1]	11	14	14	15	54	9.2	22.1

*All concentrations are in $\mu g/m^3$; includes Philadelphia County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.24. Number of Days on which $PM_{10-2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{10-2.5}$ Concentrations. Phoenix, 1997*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
TEOM PM coarse data	89	90	66	75	320	33.3	70.6
Composite[1]	89	90	66	75	320	33.3	70.6

*All concentrations are in $\mu g/m^3$; includes Maricopa County. Terminal Operations and Environmental Monitoring Committee (TEOM) data.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.25. Number of Days on which $PM_{10-2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{10-2.5}$ Concentrations. Seattle, 2000*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 530330024	15	14	14	12	55	5.7	11.4
Composite[1]	15	14	14	12	55	5.7	11.4

*All concentrations are in $\mu g/m^3$; includes King County.

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Exhibit A.26. Number of Days on which $PM_{10-2.5}$ Concentration Data are Available, by Monitor and by Quarter, and $PM_{10-2.5}$ Concentrations. St. Louis, 2002*

Monitor	Q1	Q2	Q3	Q4	Year Total	Annual Avg.	98th Percentile
AIRS 291895001	15	15	15	15	60	3.5	11.4
Composite[1]	15	15	15	15	60	3.5	11.4

*All concentrations are in $\mu g/m^3$; includes St. Louis (MO), Franklin (MO), Jefferson (MO), St. Charles (MO), Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City (MO).

1. The number of days at the composite monitor is the number of days on which at least one of the monitors reported.

Appendix B. Linear Trends in Historical $PM_{2.5}$ Data in Philadelphia and Los Angeles

Abt

memorandum

Environmental Research Area

4800 Montgomery Lane, Suite 600 ■ Bethesda, MD 20814-5341 ■ (301) 913-0500

Abt Associates Inc.

Date May 8, 2002

To Harvey Richmond, U.S. EPA/OAQPS

From Ellen Post, Abt Associates Inc.

Subject **Linear Trends in Historical $PM_{2.5}$ Data in Philadelphia and Los Angeles: Revision of November 26, 2001 Memo**

The method used to simulate just meeting a standard in the 1995/96 PM risk analysis and proposed for the current risk analysisis to "roll back" the anthropogenic portion of PM levels (i.e., the portion above background level) by the same percentage on each day. This method assumes that, all else held constant:

$$(y_i - B) = \beta * (x_i - B) \quad ,$$

where[1]

- x_i is the ith $PM_{2.5}$ concentration in a location before the standard is met,
- y_i is the ith $PM_{2.5}$ concentration in that location when the standard is just met,
- B is the background concentration in that location, and
- $\beta < 1$.

We don't have data on $PM_{2.5}$ concentrations in any location before and after the $PM_{2.5}$ standards have just been met, so we cannot directly test whether this "rollback" assumption accurately models how $PM_{2.5}$

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[1] We first examined the plausibility of this assumption in preparation for the PM risk analysis carried out in 1995/1996. At that time, we examined pairs of years of $PM_{2.5}$ data in several locations, but none of the data reflected efforts to meet $PM_{2.5}$ standards, because this exercise (and the data it used) preceded the setting of $PM_{2.5}$ standards. That investigation, however, found that the change in the distribution of $PM_{2.5}$ concentrations from one year to another year in the same location tended to be linear. This is described in Section 8.2 of Abt Associates Inc., 1996. "A Particulate Matter Risk Assessment for Philadelphia and Los Angeles."

concentrations would change if a standard were just met. We can, however, examine historical changes in $PM_{2.5}$ concentrations for any location for which we have sufficient data to determine if the proportional rollback model is consistent with these historical changes. We currently have sufficient data in each of two locations, Philadelphia and Los Angeles, to compare the distribution of daily $PM_{2.5}$ concentrations in the year 2000 with the distribution in an earlier year. In each location, we compared the two distributions to see if the change was well described as proportional. The method and results are described below.

In Philadelphia we have 353 days of observations in a year which crosses calendar years 1992 and 1993, and 296 days of observations in the year 2000. In Los Angeles we have 214 days of observations in 1995 and 357 days of observations in 2000. We first grouped the $PM_{2.5}$ concentrations in each distribution into deciles and averaged the concentrations within each decile.[2] These average concentrations within deciles are shown in Exhibit B.1 and in graph form in Exhibits B.2 and B.3, for Philadelphia and Los Angeles, respectively.

[2] We considered using the decile points themselves rather than the averages within deciles. However, the decile points would be expected to be less stable from one year to another than the averages of the concentrations within deciles. A comparison of the averages within deciles from one year to another is therefore likely to give a more accurate picture of how the distribution has changed from one year to another. This is the method that was used in the earlier comparison for the 1995/96 PM risk assessment.

Exhibit B.1. Average $PM_{2.5}$ Concentrations ($\mu g/m^3$) in Each Decile of Earlier Year and Year 2000 Distributions at Composite Monitors in Philadelphia and Los Angeles*

Decile*	Philadelphia		Los Angeles	
	1992/93	2000	1995	2000
1	5.91	4.62	10.02	6.67
2	7.94	6.58	14.62	10.19
3	9.71	8.82	18.50	12.39
4	11.19	10.25	21.06	14.59
5	13.07	12.07	24.19	16.59
6	14.87	13.72	28.40	18.55
7	17.23	16.01	32.96	21.27
8	20.67	19.4	39.72	24.22
9	25.34	23.77	54.77	28.27
10	37.90	32.58	87.12	50.50

*The first decile is the tenth percentile, the second decile is the twentieth percentile, and so on. The average concentration in the nth decile is the average of those values that are greater than the (n-1)st decile point and less than or equal to the nth decile point.

Exhibit B.2

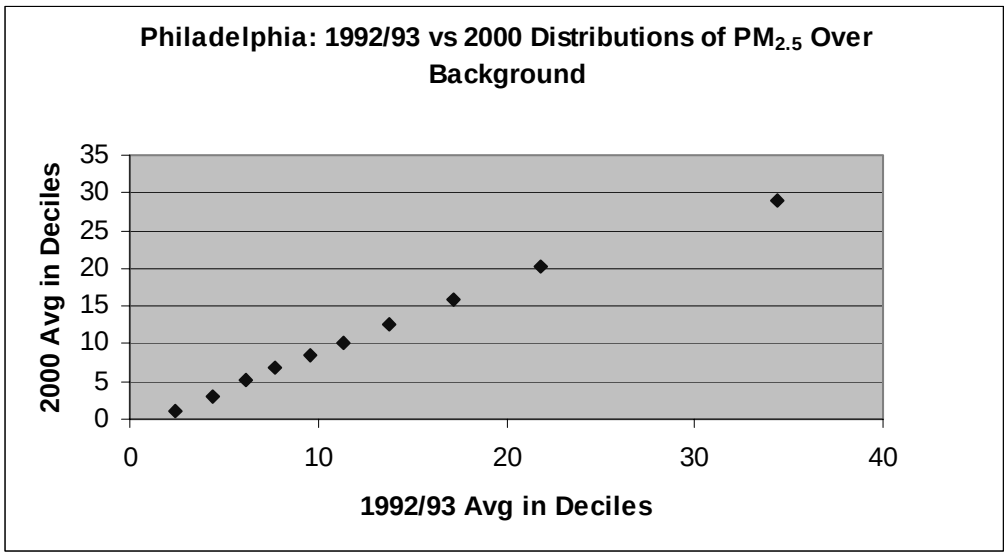
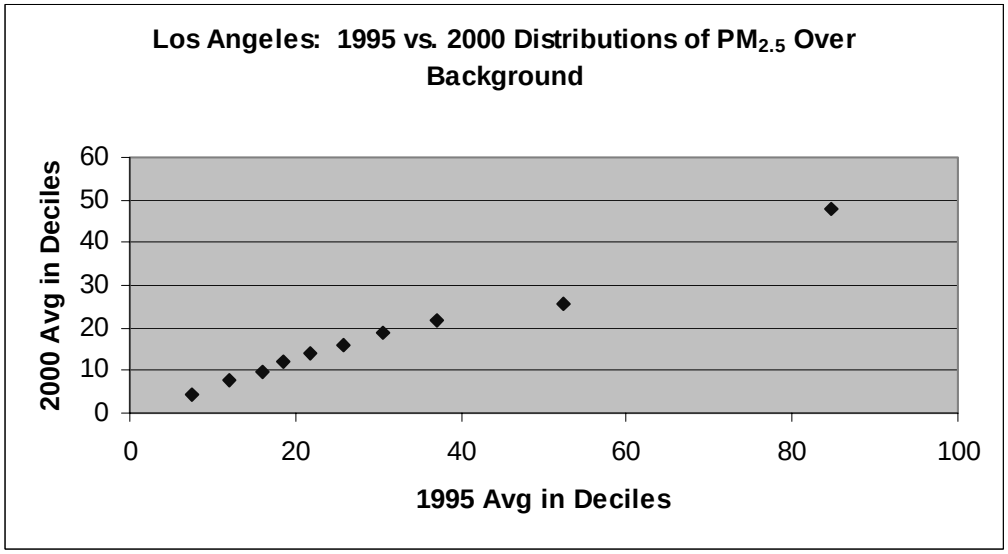


Exhibit B.3



To examine how reasonable the proportional rollback hypothesis is we estimated the following regression equation separately for Philadelphia and for Los Angeles:

$$(y_i - B) = \alpha + \beta * (x_i - B) + \varepsilon_i$$

where now,

- y_i is the average $PM_{2.5}$ concentration in the ith decile of the distribution of $PM_{2.5}$ concentrations in the location in the year 2000,
- x_i is the average $PM_{2.5}$ concentration in the ith decile of the distribution of $PM_{2.5}$ concentrations in that location in an earlier year (1995 for Los Angeles and 1992/93 for Philadelphia),
- B is the background concentration in that location (2.5 $\mu g/m^3$ in Los Angeles and 3.5 $\mu g/m^3$ in Philadelphia), and
- ε_i is an error term.

If the change in $PM_{2.5}$ concentrations from the earlier year to the year 2000 is consistent with a proportional rollback model, we would expect

- the linear fit to be good,
- the slope (β) to be less than one, and
- the intercept (α) to be close to zero

The results of the regressions in Philadelphia and Los Angeles do support the hypothesis underlying the proportional rollback method, as shown in Exhibit B.4. In both cases, the linear fit is very good (R^2 = 0.992 in Philadelphia and 0.986 in Los Angeles), the slopes are less than 1.0, and the intercepts are close to zero.[3] This supports the hypothesis that, at least in these two locations, the change in daily $PM_{2.5}$ concentrations that would result if a $PM_{2.5}$ standard were just met is reasonably modeled as a proportional rollback.

[3] Because decile points are not independent observations, the usual test of statistical significance are not valid. What is most important, however, is that the linear relationship is very good and the intercept is near zero.

Exhibit B.4. Results of Regressions of Year 2000 Average $PM_{2.5}$ Concentrations over Background on Earlier Year Average $PM_{2.5}$ Concentrations over Background.

	Philadelphia	Los Angeles
Intercept	-0.136	1.387
Slope	0.886	0.537
R^2	0.992	0.986

Appendix C. Study-Specific Information for the $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ Risk Assessments

C.1. The $PM_{2.5}$ data

Exhibit C.1. Study-Specific Information for $PM_{2.5}$ Studies in Boston, MA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	< 800	all	log-linear, GAM (stringent)	none	0	70.8	mean of lag 0 & 1	2-day avg	0.0020600	0.0013936	0.0027264
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	< 800	all	log-linear, GAM (stringent)	none	0	174	mean of lag 0 & 1	2-day avg	0.0013700	0.0009780	0.0017620
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	490-492, 494-496	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0027615	-0.0013085	0.0065788
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	410-414	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0026642	0.0014889	0.0038259
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0057325	0.0025668	0.0087095
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	COPD	490-492, 494-496	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0022739	0.0001000	0.0044017
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Ischemic heart disease	410-414	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0017840	0.0010940	0.0024693
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0040182	0.0018822	0.0060154
Respiratory Symptoms and Illnesses -- Single Pollutant Models**												
Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	n/a	7-14	logistic	none	N/A	N/A	1 day	1-day avg	0.0190119	0.0069573	0.0304950
Schwartz and Neas (2000) -- 6 cities	Cough	n/a	7-14	logistic	none	N/A	N/A	0 day	3-day avg	0.0098947	-0.0006700	0.0204990

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Respiratory Symptoms and Illnesses -- Multi-Pollutant Models**												
Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	n/a	7-14	logistic	PM10-2.5	N/A	N/A	1 day	1-day avg	0.0169761	0.0038846	0.0300717
Schwartz and Neas (2000) -- 6 cities	Cough	n/a	7-14	logistic	PM10-2.5	N/A	N/A	0 day	3-day avg	0.0045106	-0.0070240	0.0154074
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - Six Cities	All cause	all	25+	log-linear	none	11	29.6	n/a	annual mean	0.0124254	0.0041377	0.0207130
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - Six Cities	Cardiopulmonary	400-440, 485-495	25+	log-linear	none	11	29.6	n/a	annual mean	0.0169253	0.0056108	0.0278921
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Exhibit C.2. Study-Specific Information for $PM_{2.5}$ Studies in Detroit, MI

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	6	86	3 day	1-day avg	0.0007401	-0.0007318	0.0022149
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	390-459	all	log-linear, GAM (stringent)	none	6	86	1 day	1-day avg	0.0008750	-0.0013081	0.0030486
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	460-519	all	log-linear, GAM (stringent)	none	6	86	0 day	1-day avg	0.0009019	-0.0043840	0.0061762
Hospital Admissions -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	480-486	65+	log-linear, GAM (stringent)	none	6	86	1 day	1-day avg	0.0039787	0.0007401	0.0072451
Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD	490-496	65+	log-linear, GAM (stringent)	none	6	86	3 day	1-day avg	0.0011695	-0.0028650	0.0052261
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	410-414	65+	log-linear, GAM (stringent)	none	6	86	2 day	1-day avg	0.0014345	-0.0008175	0.0037127
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	428	65+	log-linear, GAM (stringent)	none	6	86	1 day	1-day avg	0.0030735	0.0005501	0.0056146
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	427	65+	log-linear, GAM (stringent)	none	6	86	1 day	1-day avg	0.0012493	-0.0027421	0.0052261
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.3. Study-Specific Information for $PM_{2.5}$ Studies in Los Angeles, CA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.0003200	-0.0002251	0.0008622
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.0001000	-0.0004610	0.0006579
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	2 day	1-day avg	-0.0000900	-0.0006265	0.0004436
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	3 day	1-day avg	-0.0004800	-0.0009841	0.0000216
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	4 day	1-day avg	-0.0002800	-0.0009296	0.0003654
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	5 day	1-day avg	-0.0007200	-0.0014024	-0.0000422
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	0.0005400	-0.0000668	0.0011432
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 30 df	none	4	86	0 day	1-day avg	0.0004000	-0.0003394	0.0011340
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.0003200	-0.0002251	0.0008622
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	0.0003000	-0.0004264	0.0010211
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.0005900	0.0000017	0.0011748
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 30 df	none	4	86	1 day	1-day avg	0.0005500	-0.0001673	0.0012622
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.0001000	-0.0004610	0.0006579
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	-0.0000100	-0.0009949	0.0009653

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	0.0009900	0.0001007	0.0018715
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.0009700	0.0001444	0.0017888
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	0.0009700	-0.0000155	0.0019459
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.0010300	0.0001575	0.0018950
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.0008000	-0.0000308	0.0016240
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	0.0006900	-0.0003174	0.0016873
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	-0.0005600	-0.0030434	0.0018632
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	-0.0014200	-0.0038624	0.0009642
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	-0.0012100	-0.0041448	0.0016411
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.0003800	-0.0021243	0.0028232
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.0008600	-0.0015918	0.0032531
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	0.0002000	-0.0028556	0.0031650
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	CO	4	86	1 day	1-day avg	-0.0005300	-0.0013183	0.0002521
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	CO	4	86	1 day	1-day avg	-0.0003300	-0.0010537	0.0003885
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	CO	4	86	1 day	1-day avg	-0.0003300	-0.0011763	0.0005092

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Cause-Specific Mortality -- Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	CO	4	86	0 day	1-day avg	0.0017800	0.0007639	0.0027859
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	CO	4	86	0 day	1-day avg	0.0018800	0.0006833	0.0030625
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	CO	4	86	1 day	1-day avg	0.0009100	-0.0001229	0.0019323
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	CO	4	86	1 day	1-day avg	0.0009100	-0.0003425	0.0021470
Hospital Admissions -- Single Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	0.0015800	0.0009137	0.0022419
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.0011600	0.0005083	0.0018074
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	0.0012600	0.0004546	0.0020590
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.0013900	0.0006951	0.0020801
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.0011300	0.0004682	0.0017874
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	0.0012000	0.0003898	0.0020037
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	0.0016700	0.0006927	0.0026378
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.0013800	0.0005240	0.0022287
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	0.0014900	0.0004186	0.0025500

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.0011900	0.0002290	0.0021418
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.0007500	-0.0001069	0.0015996
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	0.0007700	-0.0002682	0.0017975
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	4	86	2 day	1-day avg	0.0018500	0.0008365	0.0028533
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	4	86	2 day	1-day avg	0.0011400	0.0002191	0.0020525
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	4	86	2 day	1-day avg	0.0010300	-0.0001118	0.0021589
Hospital Admissions -- Single City, Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	CO	4	86	0 day	1-day avg	0.0003900	-0.0004411	0.0012142
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	CO	4	86	0 day	1-day avg	0.0005800	-0.0004077	0.0015580
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	CO	4	86	1 day	1-day avg	0.0002400	-0.0006494	0.0011215
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	CO	4	86	1 day	1-day avg	0.0002700	-0.0007501	0.0012798
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	4	86	0 day	1-day avg	0.0004200	-0.0009110	0.0017335
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	4	86	1 day	1-day avg	-0.0000400	-0.0016211	0.0015165
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	4	86	2 day	1-day avg	0.0003500	-0.0010266	0.0017079
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	4	86	3 day	1-day avg	-0.0010900	-0.0024076	0.0002105

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.4. Study-Specific Information for Studies $PM_{2.5}$ in Philadelphia, PA

Study*	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.**	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Lipfert et al. (2000)	Non-accidental	< 800	all	linear	none	-0.6	72.6	0 day	2-day avg	0.0835783		
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Lipfert et al. (2000) -- 7 counties	Cardiovascular	390-448	all	linear	none	-0.6	72.6	1 day	1-day avg	0.1044000	0.0498336	0.1589664
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Lipfert et al. (2000)	Non-accidental	< 800	all	linear	O3	-0.6	72.6	0 day	2-day avg	0.0598219		
Short-Term Exposure Cause-Specific Mortality -- Multi-Pollutant Models												
Lipfert et al. (2000) -- 7 counties	Cardiovascluar	390-448	all	linear	O3	-0.6	72.6	0 day	2-day avg	0.0315438		
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM2.5 non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient.

**Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.5. Study-Specific Information for $PM_{2.5}$ Studies in Phoenix, AZ

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	390-448.9	65+	log-linear, GAM (stringent)	none	0	42	0 day	1-day avg	0.0037100	-0.0010136	0.0084336
Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	390-448.9	65+	log-linear, GAM (stringent)	none	0	42	1 day	1-day avg	0.0066100	0.0019256	0.0112944
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.6. Study-Specific Information for $PM_{2.5}$ Studies in Pittsburgh, PA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Chock et al. (2000)	Non-accidental	<800	<75	log-linear	none	3	86	0 day	1-day avg	0.0010100	-0.0007896	0.0028096
Chock et al. (2000)	Non-accidental	<800	75+	log-linear	none	3	86	0 day	1-day avg	0.0005900	-0.0012456	0.0024256
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Chock et al. (2000)	Non-accidental	<800	<75	log-linear	CO, O3, SO2, NO2, PM10-2.5	3	86	0 day	1-day avg	0.0013000	-0.0008593	0.0034593
Chock et al. (2000)	Non-accidental	<800	75+	log-linear	CO, O3, SO2, NO2, PM10-2.5	3	86	0 day	1-day avg	0.0004000	-0.0017778	0.0025778
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.7. Study-Specific Information for $PM_{2.5}$ Studies in San Jose, CA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	2	105	0 day	1-day avg	0.0031432	0.0006371	0.0056683
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	2	105	1 day	1-day avg	-0.0015324	-0.0038026	0.0007072
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Fairley (2003) [reanalysis of Fairley (1999)]	Respiratory	11, 35, 472-519, 710.0, 710.2, 710.4	all	log-linear, GAM (stringent)	none	2	105	0 day	1-day avg	0.0044596	-0.0041619	0.0130725
Fairley (2003) [reanalysis of Fairley (1999)]	Cardiovascular	390-459	all	log-linear, GAM (stringent)	none	2	105	0 day	1-day avg	0.0024831	-0.0016818	0.0066600
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	lag COH	2	105	0 day	1-day avg	-0.0003589	-0.0038821	0.0031105
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	NO3	2	105	0 day	1-day avg	0.0030778	-0.0003229	0.0065115
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	SO4	2	105	0 day	1-day avg	0.0000714	-0.0021719	0.0022826
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	NO2	2	105	0 day	1-day avg	-0.0014952	-0.0009042	0.0012976
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	lag CO	2	105	0 day	1-day avg	-0.0009042	-0.0030556	0.0012631
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	oz8hr	2	105	0 day	1-day avg	0.0019798	-0.0016818	0.0056378
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.8. Study-Specific Information for $PM_{2.5}$ Studies in Seattle, WA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Hospital Admissions -- Single Pollutant Models												
Sheppard (2003) [reanalysis of Sheppard et al. (1999)]**	Asthma	493	<65	log-linear, GAM (stringent)	none	N/A	N/A	1 day	1-day avg	0.0033238	0.0008432	0.0049380
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM2.5 values obtained from nephelometry measurements rather than from air quality monitors.

Exhibit C.9. Study-Specific Information for $PM_{2.5}$ Studies in St. Louis, MO

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	< 800	all	log-linear, GAM (stringent)	none	0.9	88.9	mean of lag 0 & 1	2-day avg	0.0010200	0.0003732	0.0016668
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	< 800	all	log-linear, GAM (stringent)	none	0	174	mean of lag 0 & 1	2-day avg	0.0013700	0.0009780	0.0017620
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	490-492, 494-496	all	Log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0005982	-0.0029429	0.0041142
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	410-414	all	Log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0012916	0.0002996	0.0023717
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	480-487	all	Log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0010940	-0.0025318	0.0045929
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	COPD	490-492, 494-496	all	Log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0022739	0.0001000	0.0044017
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Ischemic heart disease	410-414	all	Log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0017840	0.0010940	0.0024693
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Pneumonia	480-487	all	Log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.0040182	0.0018822	0.0060154
Respiratory Symptoms and Illnesses -- Single Pollutant Models**												
Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	n/a	7-14	logistic	none	N/A	N/A	1 day	1-day avg	0.0190119	0.0069573	0.0304950
Schwartz and Neas (2000) -- 6 cities	Cough	n/a	7-14	logistic	none	N/A	N/A	0 day	3-day avg	0.0098947	-0.0006700	0.0204990

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Respiratory Symptoms and Illnesses -- Multi-Pollutant Models**												
Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	n/a	7-14	logistic	PM10-2.5	N/A	N/A	1 day	1-day avg	0.0169761	0.0038846	0.0300717
Schwartz and Neas (2000) -- 6 cities	Cough	n/a	7-14	logistic	PM10-2.5	N/A	N/A	0 day	3-day avg	0.0045106	-0.0070240	0.0154074
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - Six Cities	All cause	all	25+	log-linear	none	11	29.6	n/a	annual mean	0.0124254	0.0041377	0.0207130
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.0046257	0.0023783	0.0071001
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.0040182	0.0007968	0.0072321
Krewski et al. (2000) - Six Cities	Cardiopulmonary	400-440, 485-495	25+	log-linear	none	11	29.6	n/a	annual mean	0.0169253	0.0056108	0.0278921
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.0094331	0.0060580	0.0131463
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.0057325	0.0014889	0.0099845
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.0078811	0.0010940	0.0146694
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.0081164	0.0042596	0.0116400
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.0067557	0.0038902	0.0097558
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.0012065	-0.0020936	0.0049885

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Exhibit C.10. Study-Specific Information for Studies on Mortality Associated with Long-Term Exposure to $PM_{2.5}$

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Long-Term Exposure Mortality -- Single Pollutant Models												
Krewski et al. (2000) - Six Cities	All cause	all	25+	log-linear	none	11	29.6	n/a	annual mean	0.012425	0.004138	0.020713
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.004626	0.002378	0.007100
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	10	30	n/a	annual mean	0.004018	0.000797	0.007232
Krewski et al. (2000) - Six Cities	Cardiopulmonary	400-440, 485-495	25+	log-linear	none	11	29.6	n/a	annual mean	0.016925	0.005611	0.027892
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.009433	0.006058	0.013146
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	30	n/a	annual mean	0.005733	0.001489	0.009985
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	10	30	n/a	annual mean	0.007881	0.001094	0.014669
Long-Term Exposure Mortality -- Multi-Pollutant Models												
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.006756	0.003890	0.009756
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.008116	0.004260	0.011640
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.006756	0.003890	0.009756
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.001206	-0.002094	0.004988

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

C.2. The PM_{10} data

Exhibit C.11. Study-Specific Information for PM_{10} Studies in Boston, MA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.001193	0.000698	0.001686
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Non-accidental	<800	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.000698	0.000399	0.000995
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000408	0.000036	0.000780
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000499	0.000050	0.000936
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Respiratory Symptoms and Illnesses -- Single Pollutant Models**												
Schwartz et al. (1994) -- 6 cities	Lower respiratory symptoms	n/a	7 - 14	logistic	none	N/A	117	1 day	1-day avg	0.014176	0.006077	0.022261
Schwartz et al. (1994) -- 6 cities	Upper respiratory symptoms	n/a	7 - 14	logistic	none	N/A	117	2 day	1-day avg	0.006628	-0.000673	0.013957

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Exhibit C.12. Study-Specific Information for PM_{10} Studies in Chicago, IL

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001054	0.000471	0.001635
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	all	log-linear, penalized splines	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000975	0.000469	0.001479
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM	none	N/A	114.7	1 day	1-day avg	0.000151	-0.000071	0.000373
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	114.7	1 day	1-day avg	0.000204	0.000046	0.000362
Styer et al. (1995)	Non-accidental	<800	65+	log-linear	none	4	365	0 day	3-day avg	0.000540	0.000148	0.000932
Ito and Thurston (1996)	Non-accidental	<800	all	log-linear	none	N/A	128	0 day	2-day avg	0.000488	0.000296	0.000770
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	3	365	0 day	1-day avg	0.000480	0.000274	0.000686
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	3	365	0 day	1-day avg	0.000510	0.000314	0.000706
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	none	3	365	0 day	1-day avg	0.000510	0.000298	0.000722
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	3	365	1 day	1-day avg	0.000430	0.000212	0.000648
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	3	365	1 day	1-day avg	0.000380	0.000176	0.000584
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	none	3	365	1 day	1-day avg	0.000370	0.000149	0.000591

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	3	365	2 day	1-day avg	0.000260	0.000036	0.000484
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	3	365	3 day	1-day avg	0.000170	-0.000051	0.000391
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	3	365	4 day	1-day avg	0.000150	-0.000065	0.000365
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	3	365	5 day	1-day avg	-0.000130	-0.000337	0.000077
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000658	0.000519	0.000797
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, natural splines	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000548	0.000389	0.000698
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001124	0.000852	0.001395
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	all	log-linear, penalized splines	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001025	0.000753	0.001296
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000194	0.000040	0.000347
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Ito and Thurston (1996)	Respiratory	460-519	all	log-linear	none	N/A	N/A	0 day	2-day avg	0.001310	0.000392	0.002231
Ito and Thurston (1996)	Circulatory	390-459	all	log-linear	none	N/A	N/A	0 day	2-day avg	0.000296	-0.000202	0.000677
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 30 df	none	3	365	0 day	1-day avg	0.000320	-0.000038	0.000678
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	3	365	0 day	1-day avg	0.000390	0.000061	0.000719
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	none	3	365	0 day	1-day avg	0.000390	0.000046	0.000734

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 30 df	none	3	365	1 day	1-day avg	0.000240	-0.000127	0.000607
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	3	365	1 day	1-day avg	0.000210	-0.000127	0.000547
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	none	3	365	1 day	1-day avg	0.000210	-0.000151	0.000571
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	3	365	0 day	1-day avg	0.000480	-0.000500	0.001460
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	3	365	0 day	1-day avg	0.000480	-0.000461	0.001421
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	3	365	0 day	1-day avg	0.000550	-0.000477	0.001577
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	3	365	1 day	1-day avg	0.000950	-0.000035	0.001935
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	3	365	1 day	1-day avg	0.000880	-0.000100	0.001860
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	3	365	1 day	1-day avg	0.000940	-0.000113	0.001993
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000290	0.000080	0.000489
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	490-496	all	log-linear, GAM (stringent)	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.001489	0.000797	0.002176
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	490-496	all	log-linear, penalized splines	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.001094	0.000300	0.001882
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	490-496	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001882	0.000100	0.003633
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	490-496	all	log-linear, penalized splines	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001390	-0.000501	0.003343

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.002078	0.001390	0.002859
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	480-487	all	log-linear, penalized splines	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.001390	0.000499	0.002274
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.003053	0.001587	0.004497
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	480-487	all	log-linear, penalized splines	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002176	0.000598	0.003730
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	390-429	all	log-linear, GAM (stringent)	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000797	0.000499	0.001094
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	390-429	all	log-linear, penalized splines	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000797	0.000499	0.000995
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	390-429	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000995	0.000598	0.001489
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	390-429	all	log-linear, penalized splines	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000896	0.000399	0.001390
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Ito and Thurston (1996)	Non-accidental	<800	all	log-linear	O3	N/A	N/A	0 day	2-day avg	0.000392	0.000100	0.000677
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	SO2	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000638	0.000329	0.000946
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, natural splines	SO2	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000548	0.000200	0.000896
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	CO	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000886	0.000539	0.001232
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, natural splines	CO	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000717	0.000319	0.001124

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	O3	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000688	0.000429	0.000936
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, natural splines	O3	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000588	0.000319	0.000876
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Hospital Admissions -- Single Pollutant Models												
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.001037	0.000732	0.001342
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	390-429	65+	log-linear, natural spline, Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.000843	0.000500	0.001185
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	390-429	65+	log-linear, penalized spline, Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.001048	0.000749	0.001346
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	390-429	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	0.001050	0.000607	0.001493
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	390-429	65+	log-linear, natural spline, Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	0.001135	0.000653	0.001617
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	390-429	65+	log-linear, penalized spline, Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	0.001078	0.000611	0.001545

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 30 df	none	3	365	0 day	1-day avg	0.000760	0.000530	0.000990
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	none	3	365	0 day	1-day avg	0.000810	0.000580	0.001040
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	none	3	365	0 day	1-day avg	0.000850	0.000604	0.001096
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 30 df	none	3	365	1 day	1-day avg	0.000510	0.000264	0.000756
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	none	3	365	1 day	1-day avg	0.000490	0.000247	0.000733
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	none	3	365	1 day	1-day avg	0.000510	0.000245	0.000775
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	480-487	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.002166	0.001569	0.002763
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	480-487	65+	log-linear, natural spline, Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.000928	0.000274	0.001582
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	480-487	65+	log-linear, penalized spline, Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.003885	0.003267	0.004503
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	480-487	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	0.003223	0.002331	0.004116
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	480-487	65+	log-linear, natural spline, Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	0.001111	0.000085	0.002137
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	480-487	65+	log-linear, penalized spline, Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	0.001476	0.000507	0.002444

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	490-492,494-496	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.001350	0.000349	0.002351
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	490-492,494-496	65+	log-linear, natural spline, Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.000494	-0.000634	0.001622
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	490-492,494-496	65+	log-linear, penalized spline, Bayes adjusted	none	N/A	114.7	mean of lag 0 & 1	2-day avg	0.001081	-0.000045	0.002207
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	490-492,494-496	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	0.000102	-0.001521	0.001725
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	490-492,494-496	65+	log-linear, natural spline, Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	-0.000850	-0.002610	0.000910
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	490-492,494-496	65+	log-linear, penalized spline, Bayes adjusted	none	N/A	114.7	unconst. distr. lag	1-day avg	0.000461	-0.001262	0.002184
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	3	365	0 day	1-day avg	0.000640	0.000062	0.001218
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	3	365	1 day	1-day avg	0.000480	-0.000083	0.001043
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	3	365	2 day	1-day avg	-0.000060	-0.000620	0.000500
Morris and Naumova (1998)	Congestive Heart Failure	428	65+	log-linear	none	6	117	0 day	1-day avg	0.000769	0.000195	0.001327
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	390-429	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001114	0.000836	0.001390
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	390-429	65+	log-linear, natural spline	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001104	0.000787	0.001430
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	390-429	65+	log-linear, penalized spline	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001114	0.000827	0.001400

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	480-487	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001607	0.000946	0.002264
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	480-487	65+	log-linear, natural spline	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000010	-0.000642	0.000668
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	480-487	65+	log-linear, penalized spline	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000797	0.000140	0.001449
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	490-492,494-496	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002499	0.001203	0.003797
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	490-492,494-496	65+	log-linear, natural spline	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002088	0.000628	0.003546
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	490-492,494-496	65+	log-linear, penalized spline	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002508	0.001193	0.003816
Hospital Admissions -- Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	CO	3	365	0 day	1-day avg	0.000590	0.000343	0.000837
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	CO	3	365	0 day	1-day avg	0.000620	0.000356	0.000884
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	CO	3	365	1 day	1-day avg	0.000250	-0.000011	0.000511
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	CO	3	365	1 day	1-day avg	0.000260	-0.000026	0.000546
Morris and Naumova (1998)	Congestive heart failure	428	65+	log-linear	CO, SO2, NO2, O3	6	117	0 day	1-day avg	0.000388	-0.000197	0.001143

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.13. Study-Specific Information for PM_{10} Studies in Detroit, MI

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001321	0.000739	0.001900
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	None	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000658	0.000519	0.000797
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001124	0.000852	0.001395
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	133.4	1 day	1-day avg	0.000234	0.000038	0.000429
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	12	105	1 day	1-day avg	0.000656	-0.000396	0.001708
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000194	0.000040	0.000347
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000290	0.000080	0.000489
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	390-429	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000995	0.000598	0.001489
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	390-459	all	log-linear, GAM (stringent)	none	12	105	1 day	1-day avg	0.001050	-0.000537	0.002604
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	490-496	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001882	0.000100	0.003633

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.003053	0.001587	0.004497
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	460-519	all	log-linear, GAM (stringent)	none	12	105	0 day	1-day avg	0.001454	-0.002219	0.005114
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	SO2	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000638	0.000329	0.000946
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	CO	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000886	0.000539	0.001232
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	O3	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000688	0.000429	0.000936
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Hospital Admissions -- Single Pollutant Models												
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	133.4	unconst. distr. lag	1-day avg	0.001786	0.001328	0.002244
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	390-429	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001114	0.000836	0.001390
Schwartz and Morris (1995)	Congestive heart failure	428	65+	log-linear	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000984	0.000373	0.001584
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	428	65+	log-linear, GAM (stringent)	none	12	105	0 day	1-day avg	0.001762	-0.000059	0.003575

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	490-492,494-496	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	133.4	unconst. distr. lag	1-day avg	0.003180	0.001666	0.004695
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	490-492,494-496	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002499	0.001203	0.003797
Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD+	490-496	65+	log-linear, GAM (stringent)	none	12	105	3 day	1-day avg	0.001253	-0.001635	0.004139
Schwartz and Morris (1995)	Dysrhythmias	427	65+	log-linear	none	N/A	N/A	1 day	1-day avg	0.000588	-0.000125	0.001346
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	427	65+	log-linear, GAM (stringent)	none	12	105	1 day	1-day avg	0.000561	-0.002307	0.003427
Schwartz and Morris (1995)	Ischemic heart disease	410-414	65+	log-linear	none	N/A	N/A	0 day	1-day avg	0.000557	0.000156	0.000984
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	410-414	65+	log-linear, GAM(stringent)	none	12	105	2 day	1-day avg	0.001545	-0.000059	0.003162
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	480-487	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	133.4	unconst. distr. lag	1-day avg	0.000942	-0.000034	0.001917
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	480-486	65+	log-linear, GAM (stringent)	none	12	105	1 day	1-day avg	0.003328	0.001031	0.005621
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	480-487	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001607	0.000946	0.002264
Hospital Admissions -- Multi-Pollutant Models												
Schwartz and Morris (1995)	Congestive heart failure	428	65+	log-linear	CO	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000741	0.000125	0.001346
Schwartz (1994a)	COPD	491,492,494-496	65+	log-linear	O3	N/A	N/A	0 day	1-day avg	0.002020	0.000864	0.003176
Schwartz and Morris (1995)	Ischemic heart disease	410-414	65+	log-linear	CO	N/A	N/A	0 day	1-day avg	0.000496	0.000062	0.000924
Schwartz and Morris (1995)	Ischemic heart disease	410-414	65+	log-linear	SO2	N/A	N/A	0 day	1-day avg	0.000465	0.000031	0.000924
Schwartz (1994a)	Pneumonia	480-486	65+	log-linear	O3	N/A	N/A	0 day	1-day avg	0.001150	0.000386	0.001914

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.14. Study-Specific Information for PM_{10} Studies in Los Angeles, CA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM	none	N/A	N/A	1 day	1-day avg	0.000143	-0.000296	0.000579
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000216	0.000019	0.000413
Kinney et al. (1995)	Non-accidental	<800	all	log-linear	none	15	177	0 day	1-day avg	0.000488	0.000000	0.001044
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	7	166	0 day	1-day avg	0.000050	-0.000358	0.000458
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 30 df	none	7	166	0 day	1-day avg	0.000000	0.000000	0.000000
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	7	166	0 day	1-day avg	0.000070	-0.000301	0.000441
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	none	7	166	0 day	1-day avg	0.000040	-0.000396	0.000476
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	7	166	1 day	1-day avg	0.000100	-0.000263	0.000463
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 30 df	none	7	166	1 day	1-day avg	0.000070	-0.000373	0.000513
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	7	166	1 day	1-day avg	-0.000030	-0.000397	0.000337
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	none	7	166	1 day	1-day avg	-0.000030	-0.000482	0.000422
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	7	166	2 day	1-day avg	0.000470	0.000103	0.000837
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	7	166	3 day	1-day avg	-0.000070	-0.000441	0.000301

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	7	166	4 day	1-day avg	0.000140	-0.000226	0.000506
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	7	166	5 day	1-day avg	-0.000410	-0.000779	-0.000041
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000272	0.000031	0.000513
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 30 df	none	7	166	0 day	1-day avg	0.000330	-0.000228	0.000888
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	7	166	0 day	1-day avg	0.000400	-0.000119	0.000919
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	none	7	166	0 day	1-day avg	0.000440	-0.000194	0.001074
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 30 df	none	7	166	1 day	1-day avg	0.000280	-0.000292	0.000852
[reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	7	166	1 day	1-day avg	0.000190	-0.000342	0.000722
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	none	7	166	1 day	1-day avg	0.000230	-0.000433	0.000893
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]**	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	7	166	2 day	1-day avg	0.000770	0.000244	0.001296
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	7	166	0 day	1-day avg	0.000330	-0.001210	0.001870
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	7	166	0 day	1-day avg	0.000260	-0.001239	0.001759
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	7	166	0 day	1-day avg	0.000370	-0.001489	0.002229

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	7	166	1 day	1-day avg	0.000860	-0.000659	0.002379
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	7	166	1 day	1-day avg	0.001210	-0.000301	0.002721
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	7	166	1 day	1-day avg	0.001210	-0.000687	0.003107
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000310	0.000020	0.000588
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Kinney et al. (1995)	Non-accidental	<800	all	log-linear	O3	15	177	0 day	1-day avg	0.000488	0.000000	0.001044
Kinney et al. (1995)	Non-accidental	<800	all	log-linear	CO	15	177	0 day	1-day avg	0.000392	-0.000202	0.000862
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	CO	7	166	2 day	1-day avg	-0.000320	-0.000756	0.000116
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Short-Term Exposure Cause-Specific Mortality -- Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	CO	7	166	2 day	1-day avg	0.000500	-0.000128	0.001128

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Hospital Admissions -- Single Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 30 df	none	7	166	0 day	1-day avg	0.000670	0.000241	0.001099
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	none	7	166	0 day	1-day avg	0.000540	0.000122	0.000958
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	none	7	166	0 day	1-day avg	0.000550	0.000027	0.001073
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 30 df	none	7	166	1 day	1-day avg	0.000510	0.000075	0.000945
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	none	7	166	1 day	1-day avg	0.000480	0.000058	0.000902
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	none	7	166	1 day	1-day avg	0.000490	-0.000026	0.001006
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	7	166	0 day	1-day avg	0.001090	0.000450	0.001730
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	7	166	0 day	1-day avg	0.000750	0.000193	0.001307
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	7	166	0 day	1-day avg	0.000710	0.000007	0.001413
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	7	166	1 day	1-day avg	0.001110	0.000474	0.001746
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	7	166	1 day	1-day avg	0.000870	0.000313	0.001427
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	7	166	1 day	1-day avg	0.000780	0.000079	0.001481

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	7	166	2 day	1-day avg	0.001510	0.000848	0.002172
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	7	166	2 day	1-day avg	0.001080	0.000502	0.001658
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	7	166	2 day	1-day avg	0.000980	0.000244	0.001716
Linn et al. (2000) -- Calif. South Coast Air Basin***	Asthma	493	30+	log-linear	none	5	132	0 day	1-day avg	0.000300	-0.000484	0.001084
Linn et al. (2000) -- Calif. South Coast Air Basin***	Asthma	493	<30	log-linear	none	5	132	0 day	1-day avg	0.001100	-0.000076	0.002276
Hospital Admissions -- Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	CO	7	166	0 day	1-day avg	-0.000260	-0.000760	0.000240
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	CO	7	166	0 day	1-day avg	-0.000220	-0.000836	0.000396
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	7	166	0 day	1-day avg	-0.000030	-0.000870	0.000810
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	7	166	1 day	1-day avg	0.000380	-0.000372	0.001132
Nauenberg and Basu (1999)****	Emergency and urgent asthma-related admissions	493 prim. diag.; or pneumonia or acute bronchitis prim. diag. & 493 sec. diag.	all	linear	O3	10	132	0 day	1-day avg	0.028300	0.003408	0.053192

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

***Health effects estimated from this 2-day lag cardiovascular mortality PM10 coefficient are calculated in this PM risk assessment to compare with estimates from the multi-pollutant coefficient for the same lag, model and endpoints. The Moolgavkar (2002) reanalysis did not estimate cardiovascular mortality using a 0 or 1 day lag multi-pollutant PM10 coefficient.

***The California South Coast Air Basin represents the area included in Los Angeles, Riverside, San Bernadino and Orange Counties, excluding the mountain and desert regions of the first three counties.

****The Nauenberg and Basu (1999) study examined the effect of PM2.5 levels on emergency and urgent asthma-related hospital admissions, excluding scheduled admissions and transfers from other facilities. The study looked at patients with 493 as primary diagnosis, or 466.0, 480-483, 485-487, 518.81, 581.82, or 786.09 as primary diagnosis and 493 as secondary diagnosis.

Exhibit C.15. Study-Specific Information for PM_{10} Studies in Minneapolis, MN

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001951	0.000927	0.002964
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000658	0.000519	0.000797
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001124	0.000852	0.001395
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	141.5	1 day	1-day avg	0.000198	-0.000029	0.000425
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000134	-0.000193	0.000460
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000260	-0.000110	0.000628
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	390-429	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000995	0.000598	0.001489
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	490-496	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001882	0.000100	0.003633
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.003053	0.001587	0.004497

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	SO2	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000638	0.000329	0.000946
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	CO	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000886	0.000539	0.001232
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	O3	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000688	0.000429	0.000936
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Hospital Admissions -- Single Pollutant Models												
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	390-429	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	141.5	unconst. distr. lag	1-day avg	0.000523	-0.000644	0.001691
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	390-429	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001114	0.000836	0.001390
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	490-492,494-496	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	141.5	unconst. distr. lag	1-day avg	0.003557	-0.000062	0.007176
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	490-492,494-496	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002499	0.001203	0.003797
Schwartz (1994b)	COPD+	490-496	65+	log-linear	none	N/A	N/A	0 day	2-day avg	0.004511	0.001823	0.007227

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	480-487	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	141.5	unconst. distr. lag	1-day avg	0.001967	-0.000167	0.004101
Schwartz (1994b)	Pneumonia	480-486	65+	log-linear	none	N/A	N/A	0 day	1-day avg	0.001570	0.000296	0.002852
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	480-487	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001607	0.000946	0.002264
Hospital Admissions -- Multi-Pollutant Models												
Schwartz (1994b)	Pneumonia	480-486	65+	log-linear	O3	N/A	N/A	0 day	1-day avg	0.001570	0.000198	0.002852

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.16. Study-Specific Information for PM_{10} Studies in Philadelphia, PA

Study*	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.**	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Lipfert et al. (2000)	Non-accidental	<800	all	linear	none	-1.6	82.4	0 day	2-day avg	0.059416		
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000247	0.000007	0.000487
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000408	0.000036	0.000780
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Lipfert et al. (2000) -- 7 counties	Cardiovascular	390-448	all	linear	none	-1.6	82.4	1 day	1-day avg	0.084500	0.039000	0.130000
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000499	0.000050	0.000936
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Lipfert et al. (2000)	Non-accidental	<800	all	linear	O3	-1.6	82.4	0 day	2-day avg	0.040765		
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Short-Term Exposure Cause-Specific Mortality -- Multi-Pollutant Models												
Lipfert et al. (2000) -- 7 counties	Cardiovascluar	390-448	all	linear	O3	-0.6	72.6	0 day	2-day avg	0.023718		

*The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM2.5 non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient.

**Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.17. Study-Specific Information for PM_{10} Studies in Phoenix, AZ

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000224	-0.000005	0.000452
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000189	-0.000103	0.000480
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000389	0.000020	0.000747
Mar et al. (2003) [reanalysis of Mar et al (2000)]	Cardiovascular	390-448.9	65-100	log-linear, GAM (stringent)	none	5	213	0 day	1-day avg	0.001850	0.000400	0.003464
Mar et al. (2003) [reanalysis of Mar et al (2000)]	Cardiovascular	390-448.9	65-100	log-linear, GAM (stringent)	none	5	213	1 day	1-day avg	0.001430	0.000000	0.003093
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.18. Study-Specific Information for PM_{10} Studies in Pittsburgh, PA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000618	0.000052	0.001181
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000658	0.000519	0.000797
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001124	0.000852	0.001395
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	139.3	1 day	1-day avg	0.000220	0.000041	0.000398
Chock et al. (2000)	Non-accidental	<800	<75	log-linear	0	6.5	240	0 day	1-day avg	0.000610	0.000041	0.001179
Chock et al. (2000)	Non-accidental	<800	75+	log-linear	0	6.5	240	0 day	1-day avg	0.000390	-0.000189	0.000969
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000194	0.000040	0.000347

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000290	0.000080	0.000489
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	390-429	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000995	0.000598	0.001489
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	490-496	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001882	0.000100	0.003633
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.003053	0.001587	0.004497
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	SO2	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000638	0.000329	0.000946
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	CO	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000886	0.000539	0.001232
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	O3	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000688	0.000429	0.000936
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Chock et al. (2000)	Non-accidental	<800	<75	log-linear	NO2	6.5	240	0 day	1-day avg	0.000850	0.000141	0.001559
Chock et al. (2000)	Non-accidental	<800	75+	log-linear	CO	6.5	240	0 day	1-day avg	0.000720	0.000028	0.001412

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Hospital Admissions -- Single Pollutant Models												
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	390-429	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	139.3	unconst. distr. lag	1-day avg	0.001148	0.000659	0.001636
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	390-429	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001114	0.000836	0.001390
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	490-492,494-496	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	139.3	unconst. distr. lag	1-day avg	0.002422	0.000981	0.003863
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	490-492,494-496	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002499	0.001203	0.003797
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	480-487	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	139.3	unconst. distr. lag	1-day avg	0.001131	0.000071	0.002191
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	480-487	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001607	0.000946	0.002264

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.19. Study-Specific Information for PM_{10} Studies in Provo, UT

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]**	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	241	1 day	1-day avg	0.000195	-0.000025	0.000415
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Respiratory Symptoms and Illnesses* -- Single Pollutant Models**												
Pope et al. (1991)	Upper respiratory disease	N/A	9 -11	logistic	none	11	195	0 day	1-day avg	0.003600	0.000660	0.006540
Pope et al. (1991)	Lower respiratory disease	N/A	9 -11	logistic	none	11	195	0 day	1-day avg	0.005000	0.001080	0.008920

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Hospital Admissions -- Single Pollutant Models												
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	390-429	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	150	unconst. distr. lag	1-day avg	0.000698	-0.001006	0.002401
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	390-429	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001114	0.000836	0.001390
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	490-492,494-496	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	150	unconst. distr. lag	1-day avg	-0.008503	-0.016279	-0.000728
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	490-492,494-496	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002499	0.001203	0.003797
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	480-487	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	150	unconst. distr. lag	1-day avg	0.003201	0.000434	0.005969
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	480-487	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001607	0.000946	0.002264

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**The Dominici et al. (2003) [reanalysis of Samet et al. (2000)] C-R function used in Provo was estimated in Salt Lake City, UT, which is close to Provo.

***The C-R functions for upper and lower respiratory symptoms were calculated for a 3.5 month period starting on December 1rst.

Exhibit C.20. Study-Specific Information for PM_{10} Studies in San Jose, CA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000215	0.000004	0.000427
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	6	165	0 day	1-day avg	0.001502	0.000552	0.002462
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	6	165	1 day	1-day avg	-0.000221	-0.001131	0.000688
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000180	-0.000043	0.000402
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000290	0.000010	0.000558
Fairley (2003) [reanalysis of Fairley (1999)]	Respiratory	11, 35, 472-519, 710.0, 710.2, 710.4	all	log-linear, GAM (stringent)	none	6	165	0 day	1-day avg	0.002033	-0.000754	0.004812
Fairley (2003) [reanalysis of Fairley (1999)]	Cardiovascular	390-459	all	log-linear, GAM (stringent)	none	6	165	0 day	1-day avg	0.001632	0.000120	0.003140
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.21. Study-Specific Information for PM_{10} Studies in Seattle, WA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001440	0.000781	0.002094
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000658	0.000519	0.000797
Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001124	0.000852	0.001395
Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	145.9	1 day	1-day avg	0.000229	0.000020	0.000438
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000180	-0.000043	0.000402
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000290	0.000010	0.000558
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	390-429	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.000995	0.000598	0.001489
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	490-496	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001882	0.000100	0.003633
Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.003053	0.001587	0.004497

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	SO2	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000638	0.000329	0.000946
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	CO	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000886	0.000539	0.001232
Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	all	log-linear, GAM (stringent)	O3	N/A	N/A	mean of lag 0 & 1	2-day avg	0.000688	0.000429	0.000936
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Respiratory Symptoms and Illnesses -- Single Pollutant Models**												
Yu et al. (2000)	Asthma	N/A	5-12	logistic, transition GEE	none	7.67	86.3	1 day	1-day avg	0.009531	0.002956	0.014842
Respiratory Symptoms and Illnesses -- Multi-Pollutant Models**												
Yu et al. (2000)	Asthma	N/A	5-12	logistic, transition GEE	CO, SO2	7.67	86.3	1 day	1-day avg	0.004879	-0.005129	0.014842

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Hospital Admissions -- Single Pollutant Models												
Sheppard (2003) [reanalysis of Sheppard et al. (1999)]***	Asthma	493	<65	log-linear, GAM (stringent)	none	N/A	N/A	1 day	1-day avg	0.002064	0.000524	0.003561
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	390-429	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	145.9	unconst. distr. lag	1-day avg	0.001414	0.000702	0.002125
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	390-429	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001114	0.000836	0.001390
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	490-492,494-496	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	145.9	unconst. distr. lag	1-day avg	0.005362	0.003144	0.007581
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	490-492,494-496	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.002499	0.001203	0.003797
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	480-487	65+	log-linear, GAM (stringent), Bayes adjusted	none	N/A	145.9	unconst. distr. lag	1-day avg	0.001461	0.000096	0.002827
Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	480-487	65+	log-linear, GAM (stringent)	none	N/A	N/A	unconst. distr. lag	1-day avg	0.001607	0.000946	0.002264

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**The C-R functions for asthma symptoms were calculated for children 7 to 12 with a history of chronic mild to moderate asthma.

***Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM10 values obtained from nephelometry measurements rather than from air quality monitors.

Exhibit C.22. Study-Specific Information for PM_{10} Studies in St. Louis, MO

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM10 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.001193	0.000698	0.001686
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Non-accidental	<800	all	log-linear, GAM (stringent)	none	N/A	N/A	0 day	2-day avg	0.000698	0.000399	0.000995
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000408	0.000036	0.000780
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	390-448,480-487,490-496,507	all	log-linear, GLM, Bayes adjusted	none	N/A	N/A	1 day	1-day avg	0.000499	0.000050	0.000936
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3	N/A	N/A	1 day	1-day avg	0.000270	0.000120	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, NO2	N/A	N/A	1 day	1-day avg	0.000210	-0.000010	0.000439
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, SO2	N/A	N/A	1 day	1-day avg	0.000210	0.000020	0.000419
Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	<800	all	log-linear, GLM, Bayes adjusted	O3, CO	N/A	N/A	1 day	1-day avg	0.000240	0.000050	0.000429
Respiratory Symptoms and Illnesses -- Single Pollutant Models**												
Schwartz et al. (1994) -- 6 cities	Lower respiratory symptoms	n/a	7 - 14	logistic	none	N/A	117	1 day	1-day avg	0.014176	0.006077	0.022261
Schwartz et al. (1994) -- 6 cities	Upper respiratory symptoms	n/a	7 - 14	logistic	none	N/A	117	2 day	1-day avg	0.006628	-0.000673	0.013957

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

C.3. The $PM_{10-2.5}$ data

Exhibit C.23. Study-Specific Information for $PM_{10-2.5}$ Studies in Detroit, MI

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM Coarse Coefficient	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	4	50	1 day	1-day avg	0.0012721	-0.0007568	0.0032838
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	390-459	all	log-linear, GAM (stringent)	none	4	50	1 day	1-day avg	0.0025848	-0.0004188	0.0055690
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	460-519	all	log-linear, GAM (stringent)	none	4	50	2 day	1-day avg	0.0027021	-0.0039754	0.0093975
Hospital Admissions -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	480-486	65+	log-linear, GAM (stringent)	none	4	50	1 day	1-day avg	0.0037814	-0.0004188	0.0079769
Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD+	490-496	65+	log-linear, GAM (stringent)	none	4	50	3 day	1-day avg	0.0033223	-0.0019622	0.0085917
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	410-414	65+	log-linear, GAM (stringent)	none	4	50	2 day	1-day avg	0.0038954	0.0009475	0.0068258
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	427	65+	log-linear, GAM (stringent)	none	4	50	0 day	1-day avg	0.0000416	-0.0052791	0.0053863
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	428	65+	log-linear, GAM (stringent)	none	4	50	0 day	1-day avg	0.0017142	-0.0016142	0.0050924

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.24. Study-Specific Information for $PM_{10-2.5}$ Studies in Philadelphia, PA

Study*	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.**	Lag	Exposure Metric	PM Coarse Coefficient	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Lipfert et al. (2000)	Non-accidental	<800	all	linear	none	-20	28.3	0 day	2-day avg	0.1007197		
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Lipfert et al. (2000) -- 7 counties	Cardiovascular	390-448	all	linear	none	-20	28.3	1 day	1-day avg	0.1141000	-0.0087769	0.2369769
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Lipfert et al. (2000)	Non-accidental	<800	all	linear	O3	-20	28.3	0 day	2-day avg	0.0833075		
Short-Term Exposure Cause-Specific Mortality -- Multi-Pollutant Models												
Lipfert et al. (2000) -- 7 counties	Cardiovascluar	390-448	all	linear	O3	-20	28.3	0 day	2-day avg	0.0423723		

*The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM coarse non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient.

**Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.25. Study-Specific Information for $PM_{10-2.5}$ Studies in Phoenix, AZ

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM Coarse Coefficient	Lower Bound	Upper Bound
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	390-448.9	65+	log-linear, GAM (stringent)	none	5	187	0 day	1-day avg	0.0024700	0.0005159	0.0044241

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

Exhibit C.26. Study-Specific Information for $PM_{10-2.5}$ Studies in Seattle, WA

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM Coarse Coefficient	Lower Bound	Upper Bound
Hospital Admissions -- Single Pollutant Models												
Sheppard (2003) [reanalysis of Sheppard et al. (1999)]**	Asthma	493	<65	log-linear, GAM (stringent)	none	N/A	N/A	1 day	1-day avg	0.0021293	0.0000000	0.0052463

*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM2.5 values obtained from nephelometry measurements rather than from air quality monitors.

Exhibit C.27. Study-Specific Information for Studies in St. Louis, MO

Study	Health Effect	ICD Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.*	Lag	Exposure Metric	PM Coarse Coefficient	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	< 800	all	log-linear, penalized spline model	none	-2.3	102.6	0 day	2-day avg	0.0001090	-0.0008632	0.0010812
Respiratory Symptoms and Illnesses -- Single Pollutant Models**												
Schwartz and Neas, 2000 -- 6 cities	Lower respiratory symptoms	N/A	7-14	logistic	none	N/A	N/A	0 day	3-day avg	0.0163785	-0.0025253	0.0633522
Schwartz and Neas, 2000 -- 6 cities	Cough	N/A	7-14	logistic	none	N/A	N/A	0 day	3-day avg	0.0227902	0.0084573	0.0375131
Respiratory Symptoms and Illnesses -- Multi-Pollutant Models**												
Schwartz and Neas, 2000 -- 6 cities	Lower respiratory symptoms	N/A	7-14	logistic	PM2.5	N/A	N/A	0 day	3-day avg	0.0060988	-0.0131701	0.0258768
Schwartz and Neas, 2000 -- 6 cities	Cough	N/A	7-14	logistic	PM2.5	N/A	N/A	0 day	3-day avg	0.0206893	0.0049026	0.0365837

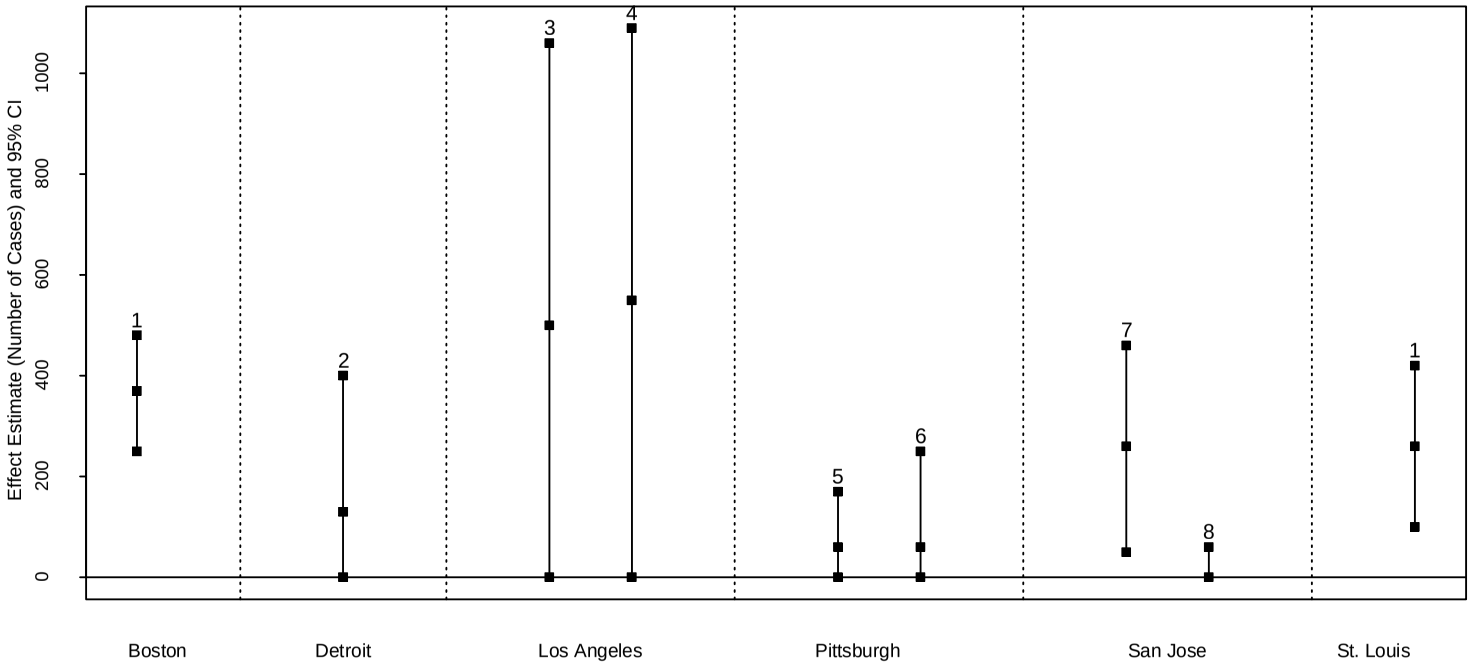
*Used only for mortality in the sensitivity analyses of hypothetical threshold models.

**The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Appendix D. Estimated Annual Health Risks Associated with "As Is" PM Concentrations

D.1. Incidence Figures: $PM_{2.5}$

Figure D.1 Estimated Annual Total (Non-Accidental) Mortality Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant, Single-City Models‡

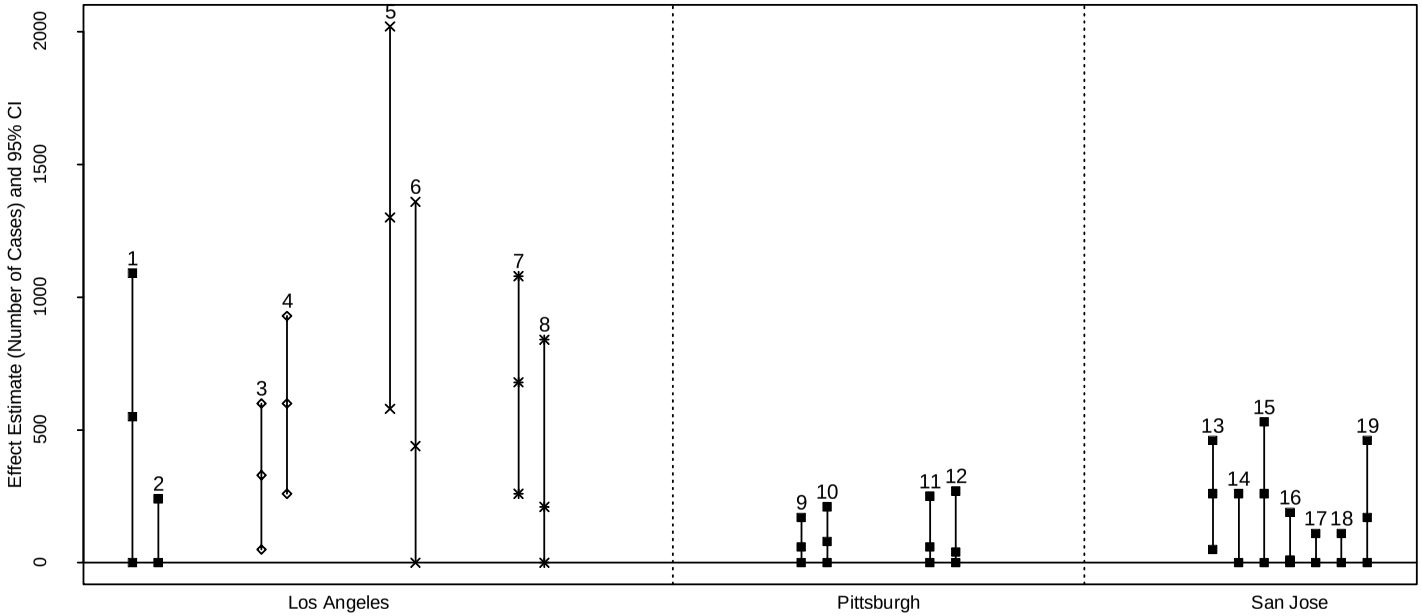


1. Schwartz et al. (1996)*
2. Lippmann et al. (2000)**
3. Moolgavkar (2000a)*** – 0-day lag
4. Moolgavkar (2000a)*** – 1-day lag
5. Chock et al. (2000) (age<75)
6. Chock et al. (2000) (age>75)
7. Fairley (1999)**** – 0-day lag
8. Fairley (1999)**** – 1-day lag

*Reanalyzed in Schwartz (2003b)
**Reanalyzed in Ito (2002)
***Reanalyzed in Moolgavkar (2003)
****Reanalyzed in Fairley (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure D.2 Estimated Annual Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-Pollutant versus Multi-Pollutant Models‡
(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)

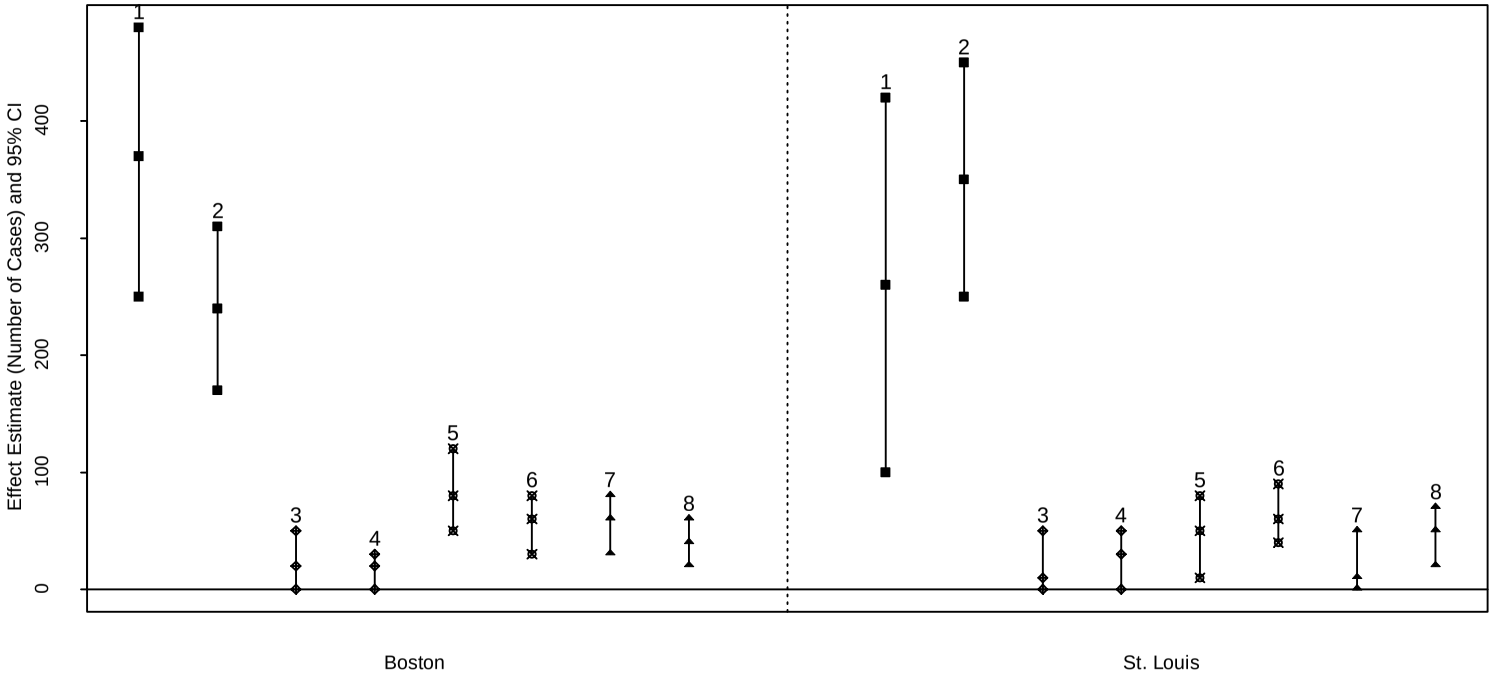


1. Moolgavkar (2000a)* – non-accid. mort.
2. Moolgavkar (2000a)* – non-accid. mort.; + CO
3. Moolgavkar (2000a)* – cardiovasc. mort.
4. Moolgavkar (2000a)* – cardiovasc. mort.; + CO
5. Moolgavkar (2000b)* – cardiovasc. hosp. adm.
6. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; + CO
7. Moolgavkar (2000c)* – COPD hosp. adm.
8. Moolgavkar (2000c)* – COPD hosp. adm.; + NO2
9. Chock et al. (2000) (age<75) – non-accid. mort.
10. Chock et al. (2000) (age<75) – non-accid. mort.; + CO, O3, SO2, PM10-2.5
11. Chock et al. (2000) (age>75) – non-accid. mort.
12. Chock et al. (2000) (age>75) – non-accid. mort.; + CO, O3, SO2, PM10-2.5
13. Fairley (1999)** – non-accid. mort.;
14. Fairley (1999)** – non-accid. mort.; + COH
15. Fairley (1999)** – non-accid. mort.; + NO3
16. Fairley (1999)** – non-accid. mort.; with SO4
17. Fairley (1999)** – non-accid. mort.; with NO2
18. Fairley (1999)** – non-accid. mort.; + CO
19. Fairley (1999)** – non-accid. mort.; + 8 hr O3

*Reanalyzed in Moolgavkar (2003)
**Reanalyzed in Fairley (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure D.3 Estimated Annual Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models‡
(Single-city models are always on the left, followed by the corresponding multi-city models)

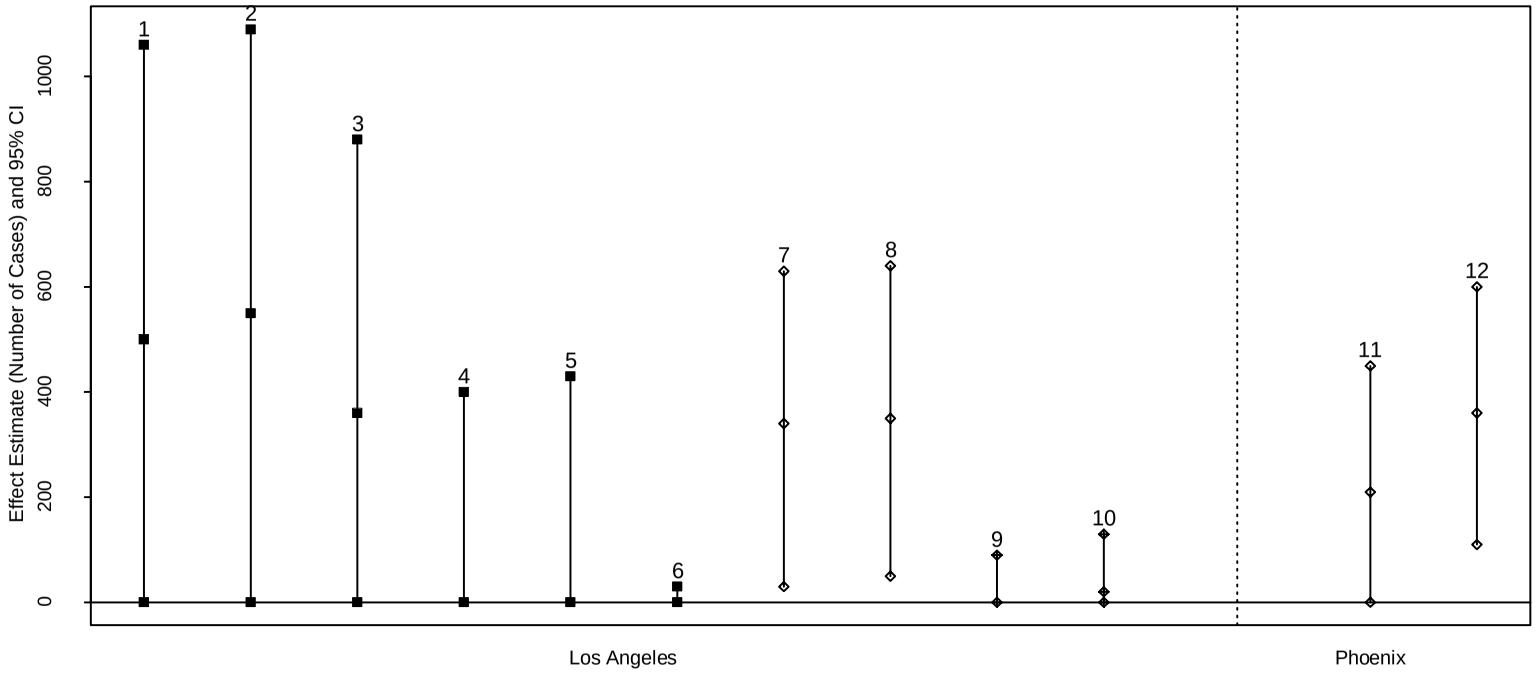


1. Schwartz et al. (1996)* – single-city; non-accid. mort.
2. Schwartz et al. (1996)* – 6 cities; non-accid. mort.
3. Klemm et al. (2000)** – single-city; COPD mort.
4. Klemm et al. (2000)** – 6 cities; COPD mort.
5. Klemm et al. (2000)** – single-city; ischemic heart disease mort.
6. Klemm et al. (2000)** – 6 cities; ischemic heart disease mort.
7. Klemm et al. (2000)** – single-city; pneumonia mort.
8. Klemm et al. (2000)** – 6 cities; pneumonia mort.

*Reanalyzed in Schwartz (2003b)
** Reanalyzed in Klemm and Mason (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure D.4 Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Effect of Different Lag Models‡

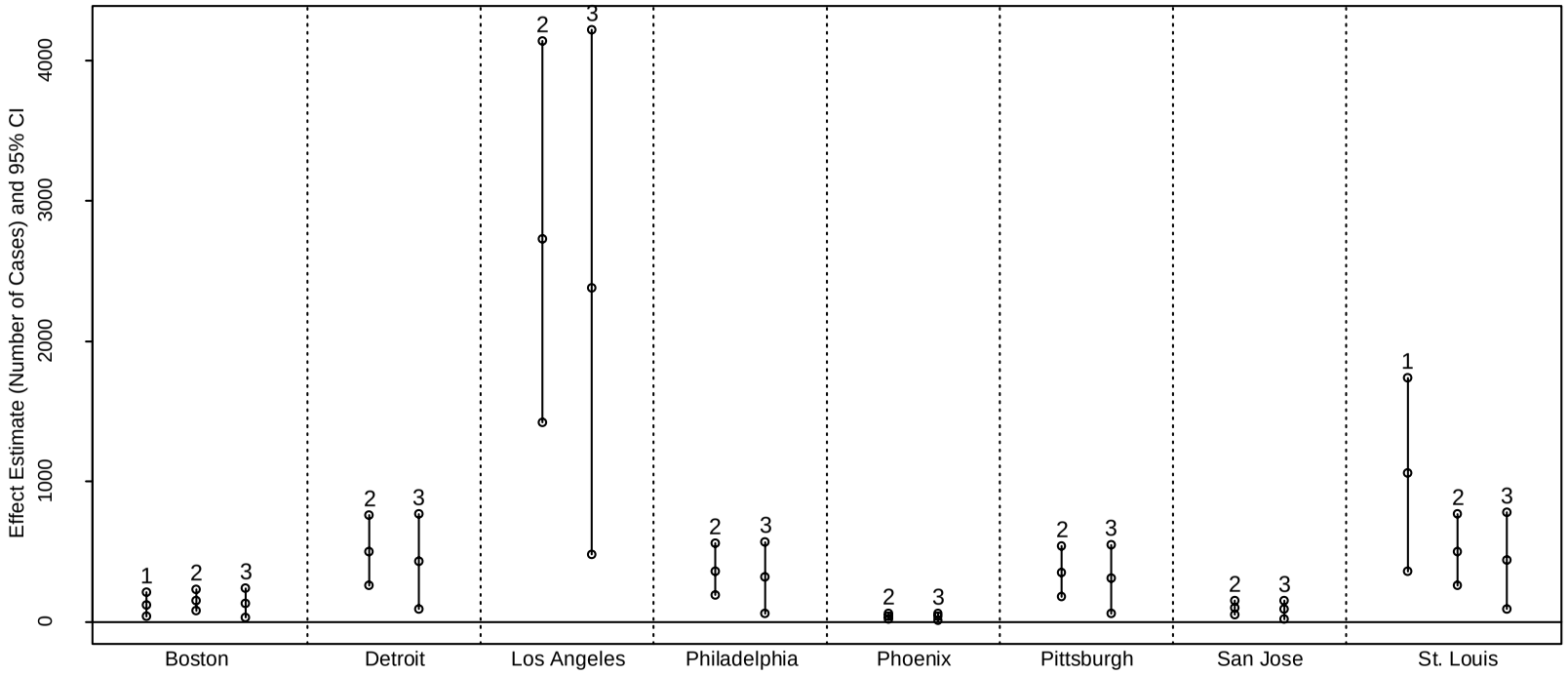


1. Moolgavkar (2000a)* – non-accid. mort. – 0-day lag
2. Moolgavkar (2000a)* – non-accid. mort. – 1-day lag
3. Moolgavkar (2000a)* – non-accid. mort. – 2-day lag
4. Moolgavkar (2000a)* – non-accid. mort. – 3-day lag
5. Moolgavkar (2000a)* – non-accid. mort. – 4-day lag
6. Moolgavkar (2000a)* – non-accid. mort. – 5-day lag
7. Moolgavkar (2000a)* – cardiovascular mort. – 0-day lag
8. Moolgavkar (2000a)* – cardiovascular mort. – 1-day lag
9. Moolgavkar (2000a)* – COPD mort. – 0-day lag
10. Moolgavkar (2000a)* – COPD mort. – 1-day lag
11. Mar et al. (2000)** – cardiovascular mort. – 0-day lag
12. Mar et al. (2000)** – cardiovascular mort. – 1-day lag

*Reanalyzed in Moolgavkar (2003)
**Reanalyzed in Mar et al. (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure D.5 Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant Models‡ †



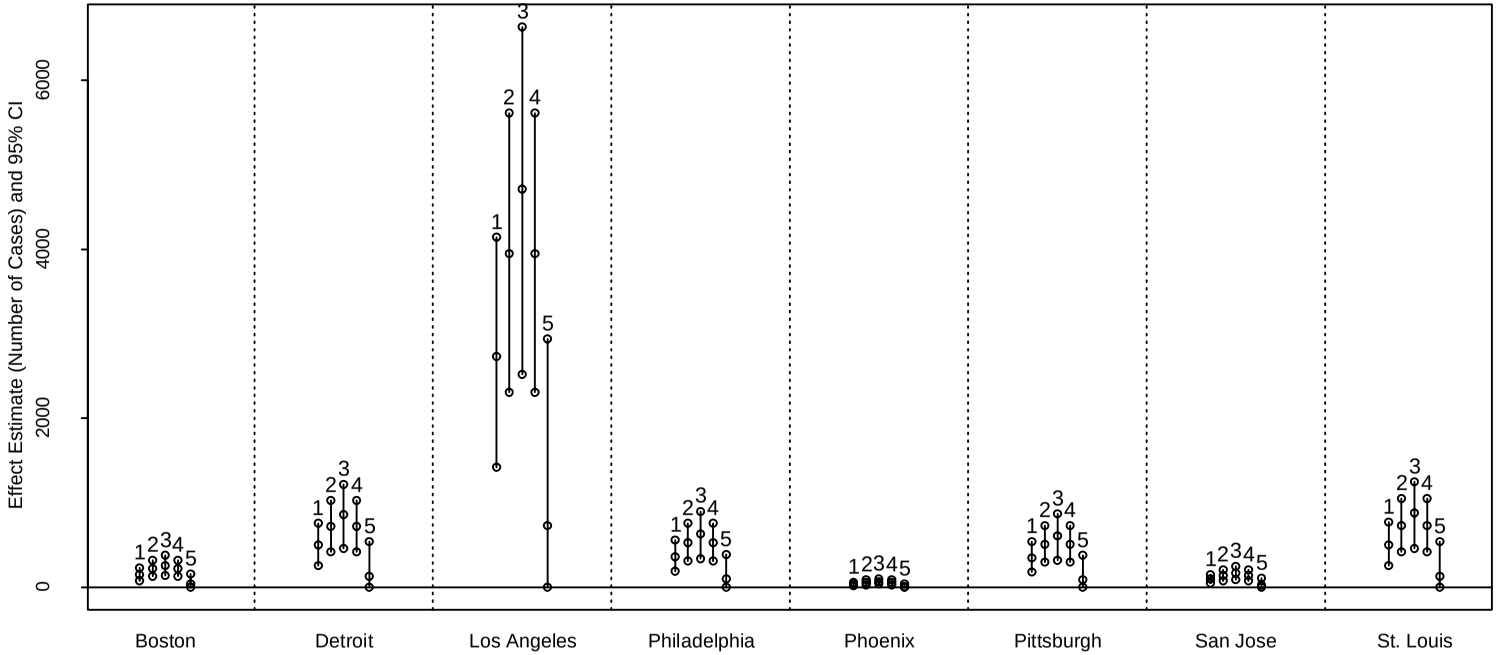
1. Krewski et al. (2000) – Six Cities
2. Krewski et al. (2000) – ACS
3. Pope et al. (2002) – ACS extended

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

† Seattle is not shown because annual average $PM_{2.5}$ in Seattle is less than the LML in all three studies, so the risk would be estimated to be zero.

Figure D.6 Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant and Multi-Pollutant Models‡ †

(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)



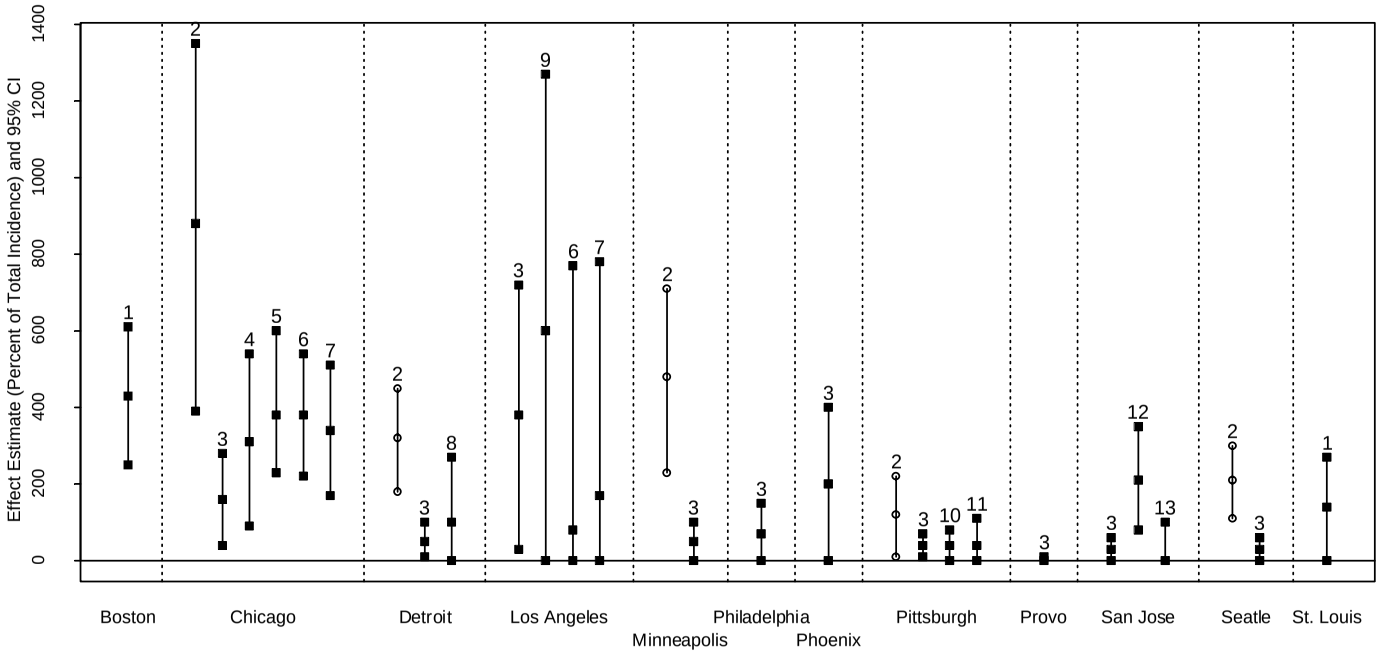
1. Krewski et al. (2000) – ACS
2. Krewski et al. (2000) – ACS – with CO
3. Krewski et al. (2000) – ACS – with NO2
4. Krewski et al. (2000) – ACS – with O3
5. Krewski et al. (2000) – ACS – with SO2

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

† Seattle is not shown because annual average $PM_{2.5}$ in Seattle is less than the LML in all three studies, so the risk would be estimated to be zero.

D.2. Incidence Figures: PM_{10}

Figure D.7 Estimated Annual Total (Non-Accidental) Mortality Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Single-Pollutant, Single-City Models‡



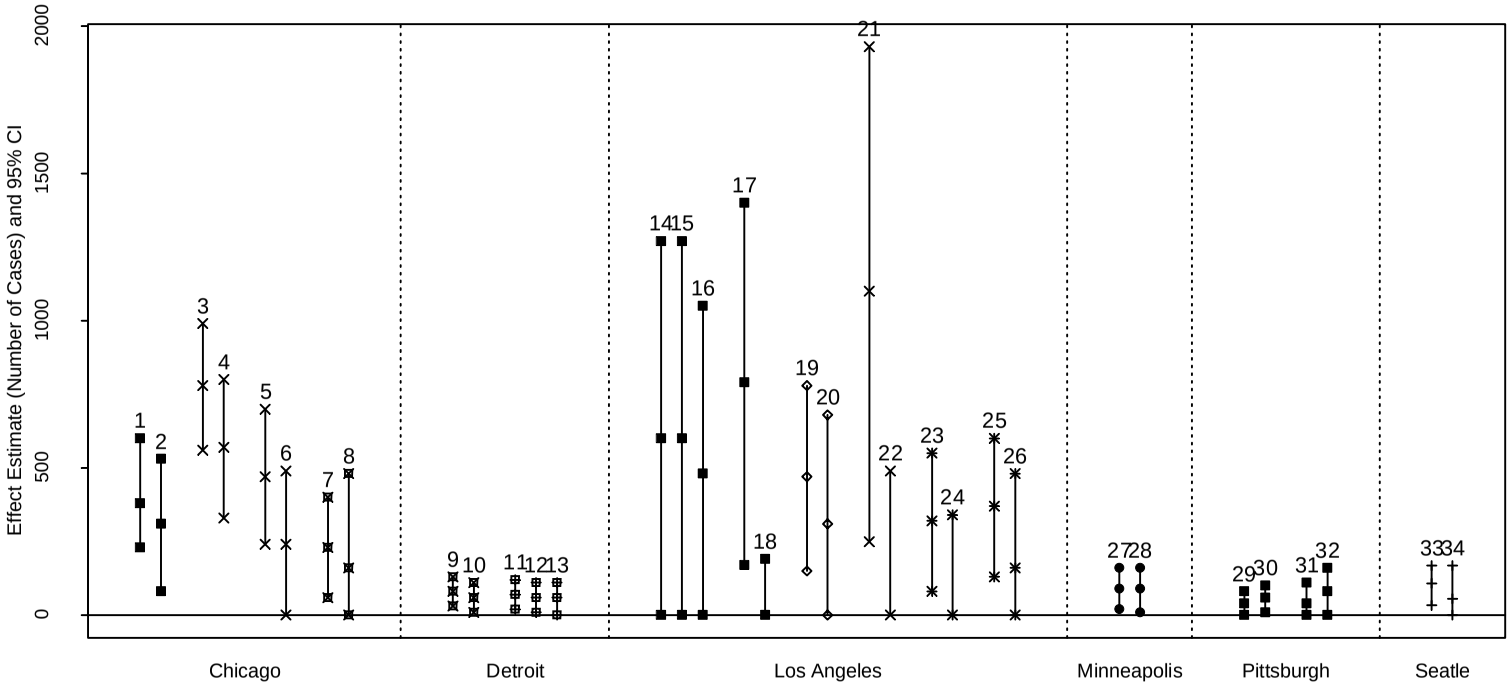
1. Klemm et al. (2000)*
2. Schwartz (2000b) — distr. lag**
3. Samet et al. (2000)***
4. Styer et al. (1995
5. Ito and Thurston (1996)
6. Moolgavkar (2000a) – 0-day lag****
7. Moolgavkar (2000a) – 1-day lag****
8. Lippmann et al. (2000)*****
9. Kinney et al. (1995)
10. Chock et al. (2000) – age<75
11. Chock et al. (2000) – age>75
12. Fairley (1999) – 0-day lag******
13. Fairley (1999) – 1-day lag******

*Reanalyzed in Klemm and Mason (2003)
**Reanalyzed in Schwartz (2003a)
***Reanalyzed in Dominici et al. (2003)
****Reanalyzed in Moolgavkar (2002)
*****Reanalyzed in Ito (2003)
******Reanalyzed in Fairley (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure D.8 Estimated Annual Health Effects Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Results Based on Single-Pollutant versus Multi-Pollutant Models‡

(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)



1. Ito and Thurston (1996) – non-accid. mort.
2. Ito and Thurston (1996) – non-accid. mort.; +O3
3. Moolgavkar (2000b) – cardiovasc. hosp. adm.; 0-day lag*
4. Moolgavkar (2000b) – cardiovasc. hosp. adm.*; 0-day lag +CO
5. Moolgavkar (2000b) – cardiovasc. hosp. adm.; 1-day lag*
6. Moolgavkar (2000b) – cardiovasc. hosp. adm.*; 1-day lag +CO
7. Morris and Naumova (1998) – cong. heart failure hosp. adm.
8. Morris and Naumova (1998) – cong. heart failure hosp. adm.; +CO, SO2, NO2, O3
9. Schwartz and Morris (1995) – cong. heart failure hosp. adm.
10. Schwartz and Morris (1995) – cong. heart failure hosp. adm.; +CO
11. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.
12. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.; +CO
13. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.; +SO2
14. Kinney et al. (1995) – non-accid. mort.
15. Kinney et al. (1995) – non-accid. mort.; +O3
16. Kinney et al. (1995) – non-accid. mort.; +CO

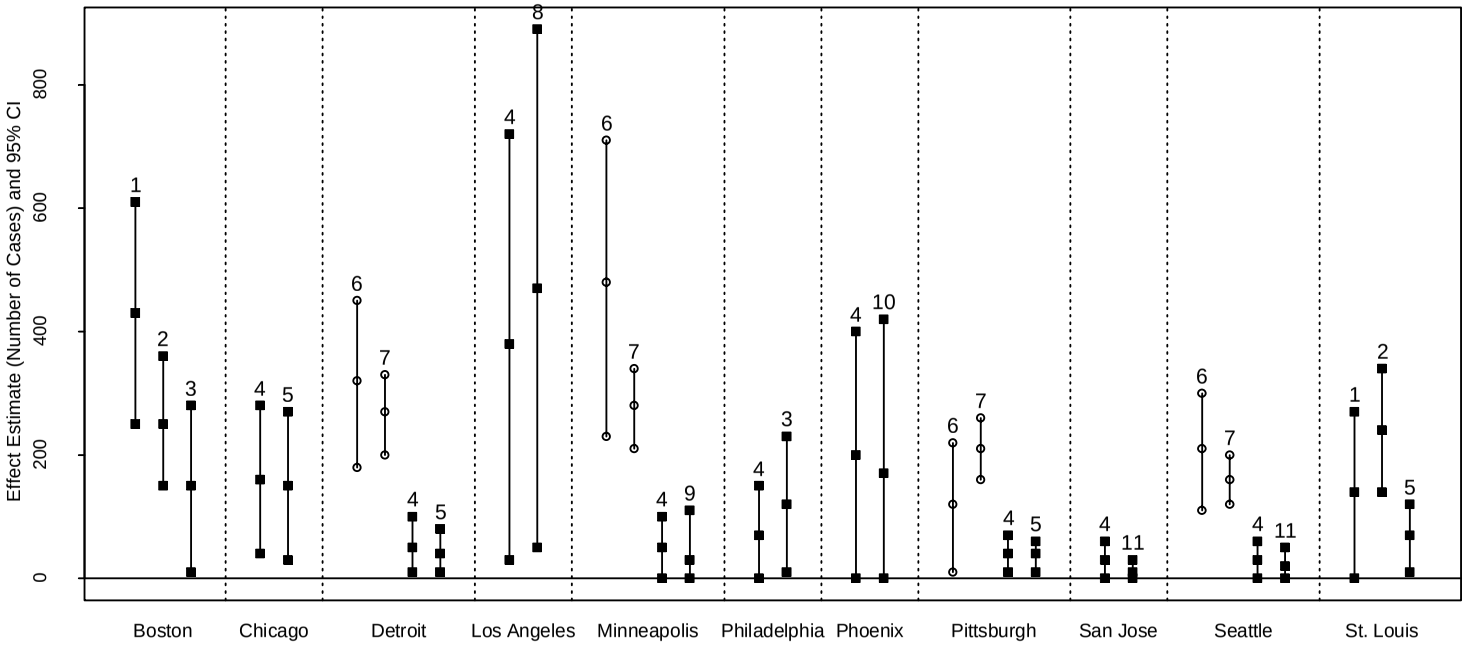
17. Moolgavkar (2000a) – non-accid. mort.; 2-day lag*
18. Moolgavkar (2000a) – non-accid. mort.; 2-day lag; + CO*
19. Moolgavkar (2000a) – cardiovasc. mort.; 2-day lag*
20. Moolgavkar (2000a) – cardiovasc. mort.; 2-day lag; + CO*
21. Moolgavkar (2000b) – cardiovasc. hosp. adm.; 2-day lag*
22. Moolgavkar (2000b) – cardiovasc. hosp. adm.; 2-day lag; + CO*
23. Moolgavkar (2000c) – COPD. hosp. adm.; 0-day lag
24. Moolgavkar (2000c) – COPD. hosp. adm.; 0-day lag; + NO2
25. Moolgavkar (2000c) – COPD. hosp. adm.; 1-day lag
26. Moolgavkar (2000c) – COPD. hosp. adm.; 1-day lag; + NO2
27. Schwartz (1994b) – pneumonia hosp. adm.
28. Schwartz (1994b) – pneumonia hosp. adm.; + O3
29. Chock et al. (2000), age<75 – non-accid. mort.
30. Chock et al. (2000), age<75 – non-accid. mort.; + NO2
31. Chock et al. (2000), age>75 – non-accid. mort.
32. Chock et al. (2000), age>75 – non-accid. mort.; + CO
33. Yu et al. (2000) – asthma symptoms**
34. Yu et al. (2000) – asthma symptoms; + CO, SO2**

*Reanalyzed in Moolgavkar (2003)

**Numbers of symptoms were in the thousands; numbers of thousands are shown in graph.

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure D.9 Estimated Annual Health Effects Associated with Short-Term Exposure to PM_{10}: Results Based on Single-City versus Multi-City Models (and 95 Percent Confidence Interval): Mortality‡
(Single-city models are always on the left, followed by the corresponding multi-city models)

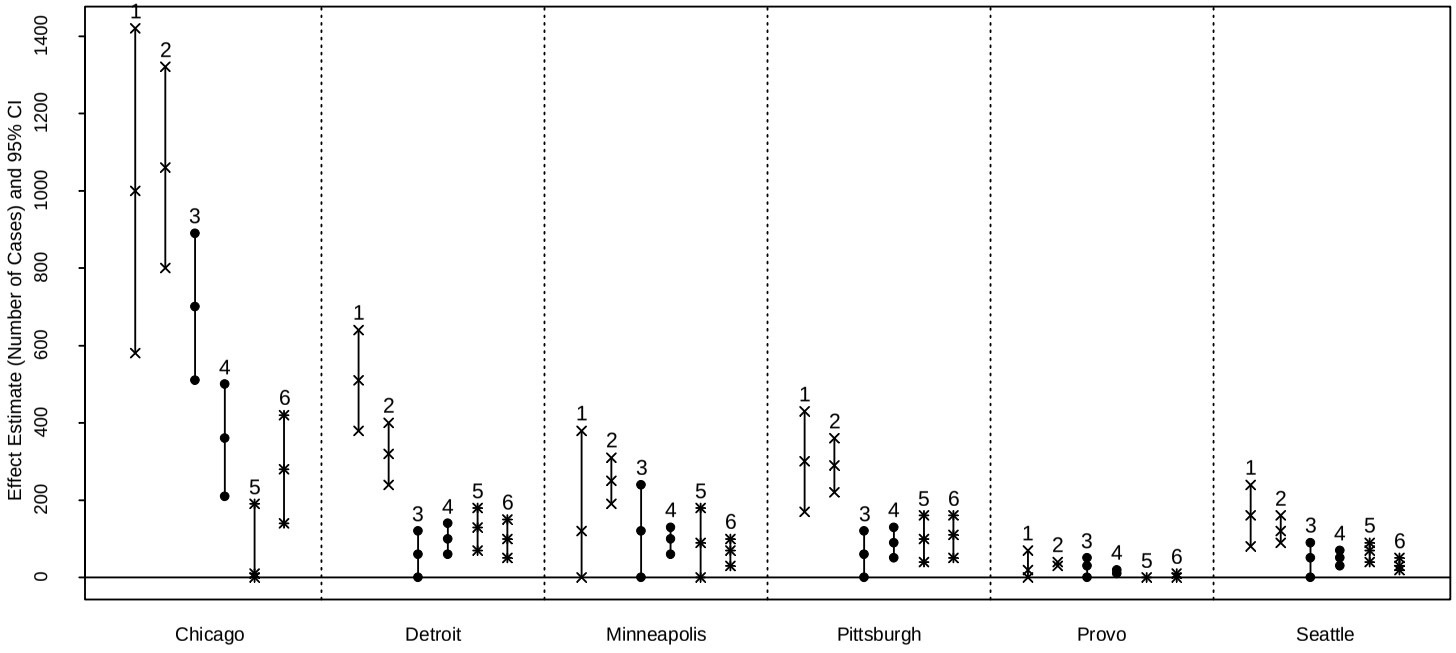


1. Klemm et al. (2000) – single-city model*
2. Klemm et al. (2000) – six-city model*
3. Samet et al. (2000) – Northeast regional model**
4. Samet et al. (2000) – single-city model**
5. Samet et al. (2000) – Industrial Midwest regional model**
6. Schwartz (2000b) – single-city model***
7. Schwartz (2000b) – 10-city model***
8. Samet et al. (2000) – South California regional model**
9. Samet et al. (2000) – Upper Midwest regional model**
10. Samet et al. (2000) – Southwest regional model**
11. Samet et al. (2000) – Northwest regional model**

*Reanalyzed in Klemm and Mason (2003)
**Reanalyzed in Dominici et al. (2003)
***Reanalyzed in Schwartz (2003a)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure D.10 Estimated Annual Health Effects Associated with Short-Term Exposure to PM_{10}: Results Based on Single-City versus Multi-City Models (and 95 Percent Confidence Interval): Morbidity‡
(Single-city models are always on the left, followed by the corresponding multi-city models)

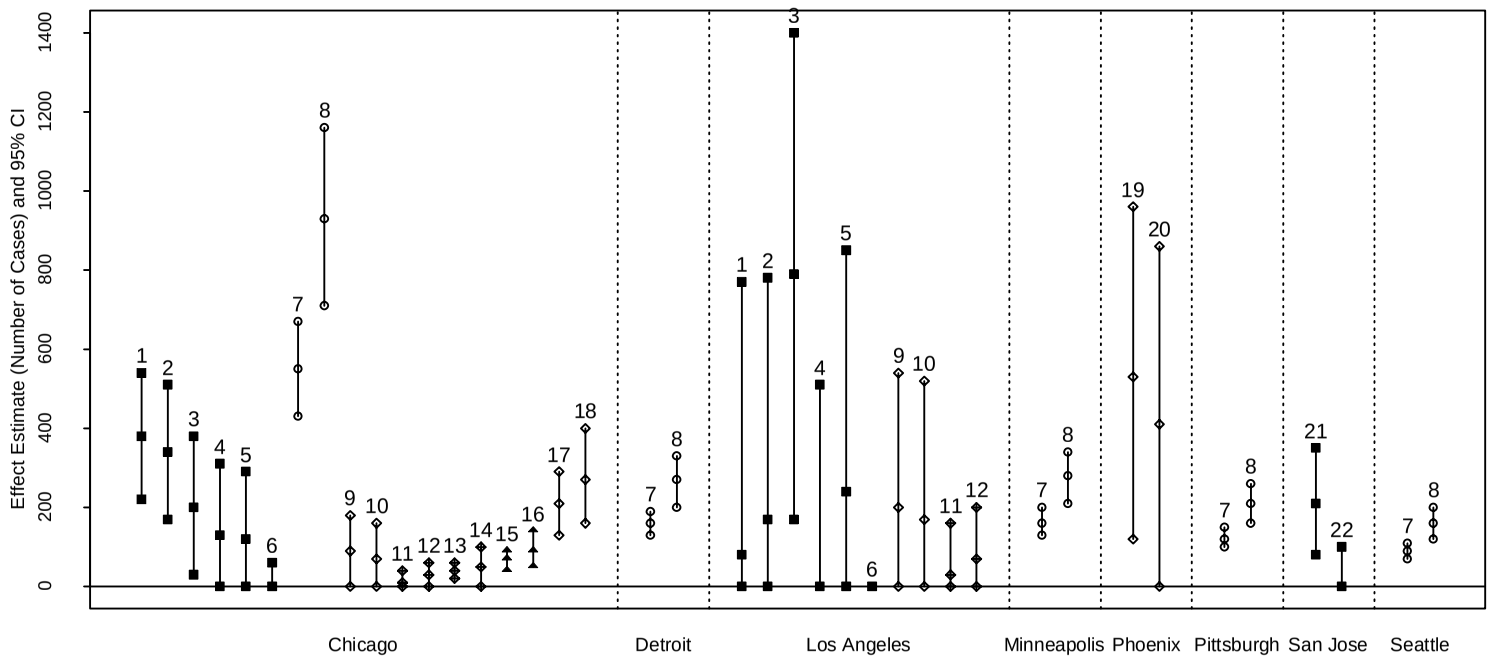


1. Samet et al. (2000) – single-city model; cardiovasc. hosp. adms.*
2. Samet et al. (2000) – 14-city model; cardiovasc. hosp. adms.*
3. Samet et al. (2000) – single-city model; pneumonia hosp. adms.*
4. Samet et al. (2000) – 14-city model; pneumonia hosp. adms.*
5. Samet et al. (2000) – single-city model; COPD hosp. adms.*
6. Samet et al. (2000) – 14-city model; COPD hosp. adms.*

*Reanalyzed in Zanobetti and Schwartz (2003)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure D.11 Estimated Annual Mortality Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Effect of Different Lag Models‡



1. Moolgavkar (2000a) – single-city model; non-accid. mort.; 0-day lag*
2. Moolgavkar (2000a) – single-city model; non-accid. mort.; 1-day lag*
3. Moolgavkar (2000a) – single-city model; non-accid. mort.; 2-day lag*
4. Moolgavkar (2000a) – single-city model; non-accid. mort.; 3-day lag*
5. Moolgavkar (2000a) – single-city model; non-accid. mort.; 4-day lag*
6. Moolgavkar (2000a) – single-city model; non-accid. mort.; 5-day lag*
7. Schwartz (2000a) – 10-city model; all cause mort.; mean of 0- and 1-day lags**
8. Schwartz (2000b) – 10-city model; all cause mort.; distr. lag**
9. Moolgavkar (2000a) – single-city model; cardiovasc. mort.; 0-day lag*
10. Moolgavkar (2000a) – single-city model; cardiovasc. mort.; 1-day lag*
11. Moolgavkar (2000a) – single-city model; COPD mort.; 0-day lag*
12. Moolgavkar (2000a) – single-city model; COPD mort.; 1-day lag*
13. Braga et al. (2001) – 10-city model; COPD mort.; mean of 0- and 1-day lags**
14. Braga et al. (2001) – 10-city model; COPD mort.; distr. lag**
15. Braga et al. (2001) – 10-city model; pneumonia mort.; mean of 0- and 1-day lags**
16. Braga et al. (2001) – 10-city model; pneumonia mort.; distr. lag**
17. Braga et al. (2001) – 10-city model; cardiovasc. mort.; mean of 0- and 1-day lags**
18. Braga et al. (2001) – 10-city model; cardiovasc. mort.; distr. lag**
19. Mar et al. (2000) – cardiovasc. mort.; 0-day lag***
20. Mar et al. (2000) – cardiovasc. mort.; 1-day lag***
21. Fairley (1999) – non-accid. mort.; 0-day lag****
22. Fairley (1999) – non-accid. mort.; 0-day lag****

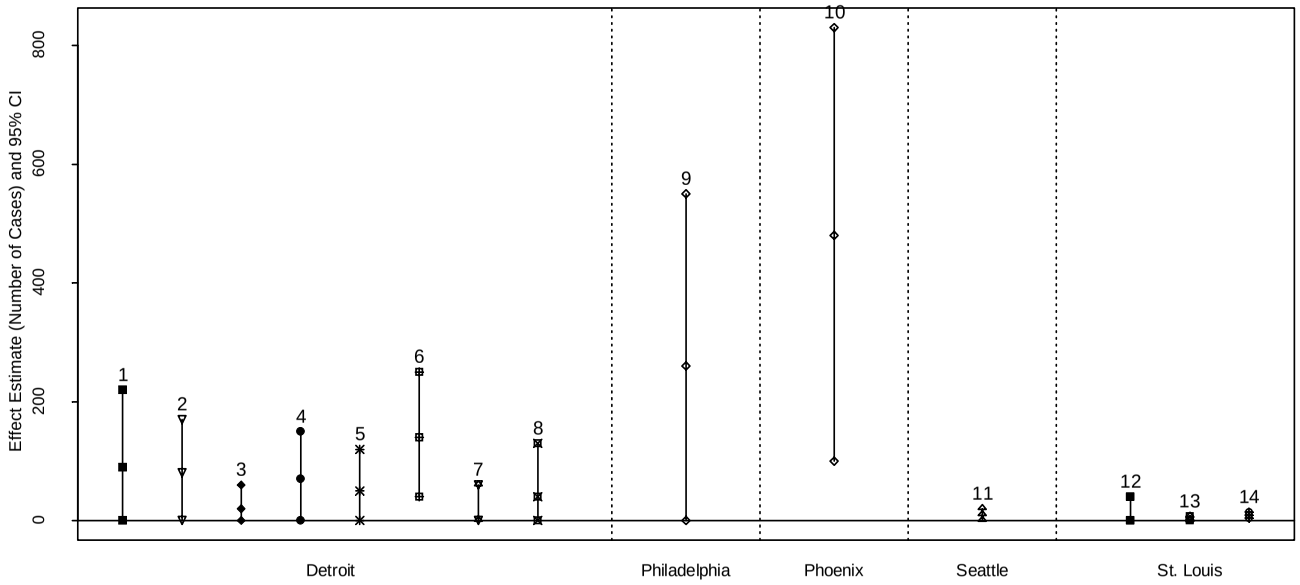
*Reanalyzed in Moolgavkar (2002)

**Reanalyzed in Schwartz (2003a)
***Reanalyzed in Mar et al. (2003)
****Reanalyzed in Fairley (20030)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

D.3. Incidence Figures: $PM_{10-2.5}$

Figure D.12 Estimated Annual Health Effects Associated with Short-Term Exposure to $PM_{10-2.5}$ (and 95 Percent Confidence Interval)‡



1. Lippmann et al. (2000) – non-accid. mort.*
2. Lippmann et al. (2000) – circulatory mort.*
3. Lippmann et al. (2000) – respiratory mort.*
4. Lippmann et al. (2000) – pneumonia hosp. adms.*
5. Lippmann et al. (2000) – COPD hosp. adms.*
6. Lippmann et al. (2000) – ischemic heart disease hosp. adms.*
7. Lippmann et al. (2000) – dysrhythmia hosp. adms.*
8. Lippmann et al. (2000) – congestive heart failure hosp. adms.*
9. Lipfert et al. (2000) – 7-county model; cardiovasc. mort.
10. Mar et al. (2000) – cardiovasc. mort.**
11. Sheppard et al. (1999) – asthma hosp. adms.***
12. Schwartz et al. (1996) – non-accid. mort.****
13. Schwartz and Neas (2000) – 6-city model; lower resp. symptoms*****
14. Schwartz and Neas (2000) – 6-city model; cough*****

*Reanalyzed in Ito (2003)
**Reanalyzed in Mar et al. (2003)
****Reanalyzed in Schwartz (2003b)
*****Numbers of symptoms were in the thousands; numbers of thousands are shown in graph.

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

D.4. The $PM_{2.5}$ data: Primary analysis

Exhibit D.1. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations Boston, MA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		370 (250 - 480)	1.6% (1.1% - 2.2%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		240 (170 - 310)	1.1% (0.8% - 1.4%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	all	0 day		20 (0 - 50)	2.2% (0.0% - 5.1%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	all	0 day		80 (50 - 120)	2.1% (1.2% - 3.0%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	all	0 day		60 (30 - 80)	4.4% (2.0% - 6.6%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	COPD	all	0 day		20 (0 - 30)	1.8% (0.1% - 3.4%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Ischemic heart disease	all	0 day		60 (30 - 80)	1.4% (0.9% - 2.0%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Pneumonia	all	0 day		40 (20 - 60)	3.2% (1.5% - 4.7%)
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - Six Cities	All cause	25+			120 (40 - 210)	0.5% (0.2% - 0.9%)
	Krewski et al. (2000) - ACS	All cause	30+			150 (80 - 230)	0.7% (0.3% - 1.0%)
	Krewski et al. (2000) - Six Cities	Cardiopulmonary	25+			70 (20 - 110)	0.7% (0.2% - 1.2%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			150 (90 - 200)	1.3% (0.9% - 1.9%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
	Pope et al. (2002) - ACS extended	All cause	30+			130 (30 - 240)	0.6% (0.1% - 1.0%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			90 (20 - 150)	0.8% (0.2% - 1.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			20 (0 - 30)	1.1% (0.2% - 2.1%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+		CO	220 (130 - 320)	1.0% (0.6% - 1.4%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	260 (140 - 380)	1.2% (0.6% - 1.7%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	220 (130 - 320)	1.0% (0.6% - 1.4%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	40 (0 - 160)	0.2% (0.0% - 0.7%)
Respiratory Symptoms* **	**Single Pollutant Models**						
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day		8000 (4000 - 15000)	15.0% (7.6% - 28.4%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day		13000 (0 - 24000)	8.3% (0.0% - 15.9%)
	Multi-Pollutant Models						
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day	PM10-2.5	7000 (2000 - 15000)	13.6% (4.3% - 28.1%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day	PM10-2.5	6000 (0 - 19000)	3.9% (0.0% - 12.4%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.2. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
Los Angeles, CA, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
							Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		500 (0 - 1060)	0.9% (0.0% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		550 (0 - 1090)	1.0% (0.0% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		360 (0 - 880)	0.6% (0.0% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		0 (0 - 400)	0.0% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		0 (0 - 430)	0.0% (0.0% - 0.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0 (0 - 30)	0.0% (0.0% - 0.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		500 (0 - 1060)	0.9% (0.0% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	0 day		370 (0 - 1050)	0.7% (0.0% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		300 (0 - 800)	0.5% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		280 (0 - 950)	0.5% (0.0% - 1.7%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
							Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		550 (0 - 1090)	1.0% (0.0% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	1 day		510 (0 - 1170)	0.9% (0.0% - 2.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		90 (0 - 610)	0.2% (0.0% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		0 (0 - 900)	0.0% (0.0% - 1.6%)
	Single Pollutant Models (Cause-Specific Mortality)							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	0 day		340 (30 - 630)	1.6% (0.2% - 3.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	0 day		330 (50 - 600)	1.6% (0.2% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	0 day		330 (0 - 650)	1.6% (0.0% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	1 day		350 (50 - 640)	1.7% (0.3% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	1 day		270 (0 - 550)	1.3% (0.0% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	1 day		240 (0 - 570)	1.1% (0.0% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		0 (0 - 90)	0.0% (0.0% - 3.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		0 (0 - 50)	0.0% (0.0% - 1.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	0 day		0 (0 - 80)	0.0% (0.0% - 2.7%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
							Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		20 (0 - 130)	0.6% (0.0% - 4.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		40 (0 - 150)	1.4% (0.0% - 5.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	1 day		10 (0 - 150)	0.3% (0.0% - 5.0%)
	Multi-Pollutant Models (Total Mortality)							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day	CO	0 (0 - 240)	0.0% (0.0% - 0.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day	CO	0 (0 - 360)	0.0% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day	CO	0 (0 - 480)	0.0% (0.0% - 0.8%)
	Multi-Pollutant Models (Cause-Specific Mortality)							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	0 day	CO	600 (260 - 930)	2.9% (1.2% - 4.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	0 day	CO	630 (230 - 1020)	3.0% (1.1% - 4.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	1 day	CO	310 (0 - 650)	1.5% (0.0% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	1 day	CO	310 (0 - 720)	1.5% (0.0% - 3.4%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
							Incidence**	Percent of Total Incidence**
Long-Term Exposure Mortality	**Single Pollutant Models**							
	Krewski et al. (2000) - ACS	All cause	30+	log-linear			2730 (1420 - 4140)	4.7% (2.5% - 7.1%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+	log-linear			3030 (1980 - 4150)	9.4% (6.1% - 12.8%)
	Pope et al. (2002) - ACS extended	All cause	30+	log-linear			2380 (480 - 4220)	4.1% (0.8% - 7.3%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+	log-linear			1880 (500 - 3200)	5.8% (1.5% - 9.9%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+	log-linear			260 (40 - 460)	7.9% (1.1% - 14.2%)
	Multi-Pollutant Models							
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		CO	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		NO2	4710 (2520 - 6630)	8.1% (4.3% - 11.4%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		O3	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		SO2	730 (0 - 2940)	1.3% (0.0% - 5.1%)
Hospital Admissions	**Single Pollutant Models**							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	0 day		1770 (1030 - 2490)	2.6% (1.5% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day		1300 (580 - 2020)	1.9% (0.8% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day		1410 (510 - 2290)	2.0% (0.7% - 3.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	1 day		1560 (780 - 2310)	2.3% (1.1% - 3.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day		1270 (530 - 1990)	1.8% (0.8% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day		1350 (440 - 2230)	1.9% (0.6% - 3.2%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
							Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		810 (340 - 1270)	2.7% (1.1% - 4.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		680 (260 - 1080)	2.2% (0.9% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	0 day		730 (210 - 1230)	2.4% (0.7% - 4.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		580 (110 - 1040)	1.9% (0.4% - 3.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		370 (0 - 780)	1.2% (0.0% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	1 day		380 (0 - 880)	1.3% (0.0% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	2 day		900 (410 - 1370)	3.0% (1.4% - 4.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day		560 (110 - 1000)	1.9% (0.4% - 3.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	2 day		510 (0 - 1050)	1.7% (0.0% - 3.5%)
	Multi-Pollutant Models							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day	CO	440 (0 - 1360)	0.6% (0.0% - 2.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day	CO	660 (0 - 1740)	1.0% (0.0% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day	CO	270 (0 - 1260)	0.4% (0.0% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day	CO	310 (0 - 1440)	0.4% (0.0% - 2.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
							Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day	NO2	210 (0 - 840)	0.7% (0.0% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day	NO2	0 (0 - 740)	0.0% (0.0% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day	NO2	170 (0 - 830)	0.6% (0.0% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	3 day	NO2	0 (0 - 100)	0.0% (0.0% - 0.4%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.3. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Lipfert et al. (2000)	Non-accidental	all	0 day		340	1.9%
	Single Pollutant Models (Cause-Specific Mortality)						
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day		420 (200 - 640)	6.1% (2.9% - 9.3%)
	Multi-Pollutant Models (Total Mortality)						
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	240	1.4%
	Multi-Pollutant Models (Cause-Specific Mortality)						
	Lipfert et al. (2000) -- 7 counties	Cardiovascluar	all	0 day	O3	130	1.8%
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+			360 (190 - 560)	2.1% (1.1% - 3.2%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			340 (220 - 470)	4.2% (2.7% - 5.8%)
	Pope et al. (2002) - ACS extended	All cause	30+			320 (60 - 570)	1.8% (0.4% - 3.2%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			210 (60 - 360)	2.6% (0.7% - 4.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			50 (10 - 80)	3.5% (0.5% - 6.4%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+		CO	530 (310 - 760)	3.0% (1.7% - 4.3%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	630 (340 - 900)	3.6% (1.9% - 5.1%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	530 (310 - 760)	3.0% (1.7% - 4.3%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	100 (0 - 390)	0.5% (0.0% - 2.2%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM2.5 non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Note 2: Multi-county short-term exposure C-R functions were applied only to counties included among those used to estimate the function.

Exhibit D.4. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
Phoenix, AZ, 2001

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Cause-Specific Mortality)**						
	Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	65+	0 day		210 (0 - 450)	2.9% (0.0% - 6.3%)
	Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	65+	1 day		360 (110 - 600)	5.0% (1.5% - 8.3%)
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+			40 (20 - 60)	0.2% (0.1% - 0.3%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			40 (30 - 60)	0.4% (0.2% - 0.5%)
	Pope et al. (2002) - ACS extended	All cause	30+			40 (10 - 60)	0.2% (0.0% - 0.3%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			20 (10 - 40)	0.2% (0.1% - 0.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			0 (0 - 10)	0.3% (0.0% - 0.6%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+		CO	60 (30 - 90)	0.3% (0.2% - 0.4%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	70 (40 - 100)	0.3% (0.2% - 0.5%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	60 (30 - 90)	0.3% (0.2% - 0.4%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	10 (0 - 40)	0.1% (0.0% - 0.2%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.5. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
Pittsburgh, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Chock et al. (2000)	Non-accidental	<75	0 day		60 (0 - 170)	1.2% (0.0% - 3.2%)
	Chock et al. (2000)	Non-accidental	75+	0 day		60 (0 - 250)	0.7% (0.0% - 2.8%)
	Multi-Pollutant Models (Total Mortality)						
	Chock et al. (2000)	Non-accidental	<75	0 day	CO, O3, SO2, NO2, PM10-2.5	80 (0 - 210)	1.5% (0.0% - 3.9%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO, O3, SO2, NO2, PM10-2.5	40 (0 - 270)	0.5% (0.0% - 3.0%)
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+			350 (180 - 540)	2.4% (1.2% - 3.6%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			360 (240 - 500)	4.8% (3.1% - 6.6%)
	Pope et al. (2002) - ACS extended	All cause	30+			310 (60 - 550)	2.1% (0.4% - 3.7%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			220 (60 - 380)	2.9% (0.8% - 5.1%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			40 (10 - 80)	4.0% (0.6% - 7.3%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+		CO	510 (300 - 730)	3.5% (2.0% - 4.9%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	610 (320 - 870)	4.1% (2.2% - 5.9%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	510 (300 - 730)	3.5% (2.0% - 4.9%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	90 (0 - 380)	0.6% (0.0% - 2.6%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.6. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
San Jose, CA, 2001

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day		260 (50 - 460)	3.0% (0.6% - 5.3%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	1 day		0 (0 - 60)	0.0% (0.0% - 0.7%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Fairley (2003) [reanalysis of Fairley (1999)]	Respiratory	all	0 day		50 (0 - 130)	4.2% (0.0% - 11.5%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Cardiovascular	all	0 day		90 (0 - 230)	2.4% (0.0% - 6.2%)
	Multi-Pollutant Models (Total Mortality)						
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	lag COH	0 (0 - 260)	0.0% (0.0% - 3.0%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	NO3	260 (0 - 530)	3.0% (0.0% - 6.1%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	SO4	10 (0 - 190)	0.1% (0.0% - 2.2%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	NO2	0 (0 - 110)	0.0% (0.0% - 1.3%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	lag CO	0 (0 - 110)	0.0% (0.0% - 1.2%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	oz8hr	170 (0 - 460)	1.9% (0.0% - 5.3%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+			100 (50 - 150)	1.1% (0.6% - 1.7%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			100 (70 - 150)	2.3% (1.5% - 3.1%)
	Pope et al. (2002) - ACS extended	All cause	30+			90 (20 - 150)	1.0% (0.2% - 1.7%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			60 (20 - 110)	1.4% (0.4% - 2.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			10 (0 - 20)	1.9% (0.3% - 3.5%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+		CO	140 (80 - 210)	1.6% (0.9% - 2.3%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	170 (90 - 250)	1.9% (1.0% - 2.8%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	140 (80 - 210)	1.6% (0.9% - 2.3%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	30 (0 - 110)	0.3% (0.0% - 1.2%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.7. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
Seattle, WA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**						
	Sheppard (2003) [reanalysis of Sheppard et al. (1999)]***	Asthma	<65	1 day		40 (10 - 50)	2.2% (0.6% - 3.3%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

***Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM2.5 values obtained from nephelometer measurements rather than from air quality monitors.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.8. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		260 (100 - 420)	1.1% (0.4% - 1.9%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		350 (250 - 450)	1.5% (1.1% - 2.0%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	all	0 day		10 (0 - 50)	0.7% (0.0% - 4.5%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	all	0 day		50 (10 - 80)	1.4% (0.3% - 2.6%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	all	0 day		10 (0 - 50)	1.2% (0.0% - 5.0%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	COPD	all	0 day		30 (0 - 50)	2.5% (0.1% - 4.8%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Ischemic heart disease	all	0 day		60 (40 - 90)	2.0% (1.2% - 2.7%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Pneumonia	all	0 day		50 (20 - 70)	4.4% (2.1% - 6.5%)
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - Six Cities	All cause	25+			1060 (360 - 1740)	4.5% (1.5% - 7.4%)
	Krewski et al. (2000) - ACS	All cause	30+			500 (260 - 770)	2.2% (1.1% - 3.3%)
	Krewski et al. (2000) - Six Cities	Cardiopulmonary	25+			670 (230 - 1080)	6.1% (2.1% - 9.8%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			530 (340 - 730)	4.3% (2.8% - 6.0%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
	Pope et al. (2002) - ACS extended	All cause	30+			440 (90 - 780)	1.9% (0.4% - 3.3%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			330 (90 - 560)	2.7% (0.7% - 4.6%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			60 (10 - 120)	3.6% (0.5% - 6.7%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+		CO	730 (420 - 1050)	3.1% (1.8% - 4.5%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	880 (460 - 1250)	3.8% (2.0% - 5.3%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	730 (420 - 1050)	3.1% (1.8% - 4.5%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	130 (0 - 540)	0.6% (0.0% - 2.3%)
Respiratory Symptoms* **	**Single Pollutant Models**						
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day		11000 (5000 - 17000)	20.2% (8.1% - 29.8%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day		19000 (0 - 35000)	11.3% (0.0% - 21.5%)
	Multi-Pollutant Models						
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day	PM10-2.5	10000 (3000 - 17000)	18.3% (4.7% - 29.5%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day	PM10-2.5	9000 (0 - 28000)	5.4% (0.0% - 16.8%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.9. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations Boston, MA, 1999

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Non-accidental	all	0 day		430 (250 - 610)	1.9% (1.1% - 2.7%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Non-accidental	all	0 day		250 (150 - 360)	1.1% (0.7% - 1.6%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		150 (10 - 280)	0.7% (0.1% - 1.3%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		90 (10 - 170)	0.8% (0.1% - 1.5%)
	Multi-Pollutant Models (Total Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3	100 (40 - 160)	0.4% (0.2% - 0.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, NO2	80 (0 - 160)	0.3% (0.0% - 0.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, SO2	80 (10 - 150)	0.3% (0.0% - 0.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, CO	90 (20 - 160)	0.4% (0.1% - 0.7%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Respiratory Symptoms* **	**Single Pollutant Models**						
	Schwartz et al. (1994) -- 6 cities	Lower respiratory symptoms	7 - 14	1 day		12000 (16000 - 50000)	22.2% (31.6% - 95.4%)
	Schwartz et al. (1994) -- 6 cities	Upper respiratory symptoms	7 - 14	2 day		35000 (0 - 66000)	22.8% (0.0% - 43.8%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.10. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Chicago, IL, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**							
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	log-linear, GAM (stringent)	unconst. distr. lag		880 (390 - 1350)	1.9% (0.8% - 2.9%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	log-linear, penalized splines	unconst. distr. lag		810 (390 - 1220)	1.7% (0.8% - 2.6%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM	1 day		120 (0 - 290)	0.3% (0.0% - 0.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		160 (40 - 280)	0.4% (0.1% - 0.6%)
	Styer et al. (1995)	Non-accidental	65+	log-linear	0 day		310 (90 - 540)	1.0% (0.3% - 1.6%)
	Ito and Thurston (1996)	Non-accidental	all	log-linear	0 day		380 (230 - 600)	0.9% (0.5% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		380 (220 - 540)	0.9% (0.5% - 1.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		400 (250 - 550)	0.9% (0.6% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		400 (230 - 560)	0.9% (0.5% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		340 (170 - 510)	0.8% (0.4% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		300 (140 - 460)	0.7% (0.3% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		290 (120 - 460)	0.7% (0.3% - 1.0%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		380 (220 - 540)	0.9% (0.5% - 1.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		340 (170 - 510)	0.8% (0.4% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		200 (30 - 380)	0.5% (0.1% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		130 (0 - 310)	0.3% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		120 (0 - 290)	0.3% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0 (0 - 60)	0.0% (0.0% - 0.1%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1		550 (430 - 670)	1.2% (0.9% - 1.4%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, natural splines	mean of lag 0 & 1		460 (330 - 580)	1.0% (0.7% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	unconst. distr. lag		930 (710 - 1160)	2.0% (1.5% - 2.4%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	log-linear, penalized splines	unconst. distr. lag		850 (630 - 1070)	1.8% (1.3% - 2.3%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		150 (30 - 270)	0.3% (0.1% - 0.6%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Single Pollutant Models (Cause-Specific Mortality)							
	Ito and Thurston (1996)	Respiratory	all	log-linear	0 day		100 (30 - 160)	2.3% (0.7% - 3.9%)
	Ito and Thurston (1996)	Circulatory	all	log-linear	0 day		100 (0 - 230)	0.5% (0.0% - 1.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	0 day		90 (0 - 180)	0.6% (0.0% - 1.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	0 day		110 (20 - 190)	0.7% (0.1% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	0 day		110 (10 - 200)	0.7% (0.1% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	1 day		70 (0 - 160)	0.4% (0.0% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	1 day		60 (0 - 150)	0.4% (0.0% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	1 day		60 (0 - 150)	0.4% (0.0% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		10 (0 - 40)	0.9% (0.0% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		10 (0 - 40)	0.9% (0.0% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	0 day		20 (0 - 50)	1.0% (0.0% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		30 (0 - 60)	1.7% (0.0% - 3.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		30 (0 - 50)	1.6% (0.0% - 3.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	1 day		30 (0 - 60)	1.7% (0.0% - 3.5%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	log-linear, GLM, Bayes adjusted	1 day		120 (30 - 200)	0.5% (0.1% - 0.9%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	log-linear, GAM (stringent)	mean of lag 0 & 1		40 (20 - 60)	2.6% (1.4% - 3.8%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	log-linear, penalized splines	mean of lag 0 & 1		30 (10 - 50)	1.9% (0.5% - 3.3%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	log-linear, GAM (stringent)	unconst. distr. lag		50 (0 - 100)	3.3% (0.2% - 6.2%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	log-linear, penalized splines	unconst. distr. lag		40 (0 - 100)	2.4% (0.0% - 5.7%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	log-linear, GAM (stringent)	mean of lag 0 & 1		70 (40 - 90)	3.6% (2.4% - 4.9%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	log-linear, penalized splines	mean of lag 0 & 1		40 (20 - 70)	2.4% (0.9% - 3.9%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	log-linear, GAM (stringent)	unconst. distr. lag		90 (50 - 140)	5.2% (2.8% - 7.6%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	log-linear, penalized splines	unconst. distr. lag		70 (20 - 110)	3.8% (1.1% - 6.3%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	log-linear, GAM (stringent)	mean of lag 0 & 1		210 (130 - 290)	1.4% (0.9% - 1.9%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	log-linear, penalized splines	mean of lag 0 & 1		210 (130 - 270)	1.4% (0.9% - 1.8%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	log-linear, GAM (stringent)	unconst. distr. lag		270 (160 - 400)	1.8% (1.1% - 2.6%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	log-linear, penalized splines	unconst. distr. lag		240 (110 - 370)	1.6% (0.7% - 2.4%)
	Multi-Pollutant Models (Total Mortality)							
	Ito and Thurston (1996)	Non-accidental	all	log-linear	0 day	O3	310 (80 - 530)	0.7% (0.2% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	SO2	530 (280 - 790)	1.1% (0.6% - 1.7%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, natural splines	mean of lag 0 & 1	SO2	460 (170 - 750)	1.0% (0.4% - 1.6%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	CO	740 (450 - 1020)	1.6% (1.0% - 2.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, natural splines	mean of lag 0 & 1	CO	600 (270 - 930)	1.3% (0.6% - 2.0%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	O3	570 (360 - 780)	1.2% (0.8% - 1.7%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, natural splines	mean of lag 0 & 1	O3	490 (270 - 730)	1.0% (0.6% - 1.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3	210 (90 - 340)	0.5% (0.2% - 0.8%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, NO2	160 (0 - 340)	0.4% (0.0% - 0.8%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, SO2	160 (20 - 330)	0.4% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, CO	190 (40 - 340)	0.4% (0.1% - 0.8%)
Hospital Admissions	**Single Pollutant Models**							
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	65+	log-linear, GAM (stringent), Bayes adjusted	mean of lag 0 & 1		990 (700 - 1280)	1.8% (1.3% - 2.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	65+	log-linear, natural spline, Bayes adjusted	mean of lag 0 & 1		810 (480 - 1130)	1.5% (0.9% - 2.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardiovascular	65+	log-linear, penalized spline, Bayes adjusted	mean of lag 0 & 1		1000 (720 - 1280)	1.8% (1.3% - 2.4%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	log-linear, GAM (stringent), Bayes adjusted	unconst. distr. lag		1000 (580 - 1420)	1.9% (1.1% - 2.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	log-linear, natural spline, Bayes adjusted	unconst. distr. lag		1080 (630 - 1530)	2.0% (1.2% - 2.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	log-linear, penalized spline, Bayes adjusted	unconst. distr. lag		1030 (590 - 1470)	1.9% (1.1% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	0 day		730 (510 - 950)	1.3% (0.9% - 1.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day		780 (560 - 990)	1.4% (1.0% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day		810 (580 - 1050)	1.5% (1.1% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	1 day		490 (260 - 720)	0.9% (0.5% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day		470 (240 - 700)	0.9% (0.4% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day		490 (240 - 740)	0.9% (0.4% - 1.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	65+	log-linear, GAM (stringent), Bayes adjusted	mean of lag 0 & 1		480 (350 - 600)	3.8% (2.7% - 4.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	65+	log-linear, natural spline, Bayes adjusted	mean of lag 0 & 1		210 (60 - 350)	1.6% (0.5% - 2.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	65+	log-linear, penalized spline, Bayes adjusted	mean of lag 0 & 1		840 (710 - 960)	6.6% (5.6% - 7.6%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	log-linear, GAM (stringent), Bayes adjusted	unconst. distr. lag		700 (510 - 890)	5.5% (4.0% - 7.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	log-linear, natural spline, Bayes adjusted	unconst. distr. lag		250 (20 - 470)	2.0% (0.2% - 3.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	log-linear, penalized spline, Bayes adjusted	unconst. distr. lag		330 (110 - 540)	2.6% (0.9% - 4.2%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	65+	log-linear, GAM (stringent), Bayes adjusted	mean of lag 0 & 1		150 (40 - 260)	2.4% (0.6% - 4.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	65+	log-linear, natural spline, Bayes adjusted	mean of lag 0 & 1		60 (0 - 180)	0.9% (0.0% - 2.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	65+	log-linear, penalized spline, Bayes adjusted	mean of lag 0 & 1		120 (0 - 250)	1.9% (0.0% - 3.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	log-linear, GAM (stringent), Bayes adjusted	unconst. distr. lag		10 (0 - 190)	0.2% (0.0% - 3.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	log-linear, natural spline, Bayes adjusted	unconst. distr. lag		0 (0 - 100)	0.0% (0.0% - 1.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	log-linear, penalized spline, Bayes adjusted	unconst. distr. lag		50 (0 - 240)	0.8% (0.0% - 3.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		270 (30 - 510)	1.1% (0.1% - 2.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		200 (0 - 440)	0.9% (0.0% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day		0 (0 - 210)	0.0% (0.0% - 0.9%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Morris and Naumova (1998)	Congestive heart failure	65+	log-linear	0 day		230 (60 - 400)	1.4% (0.4% - 2.3%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	log-linear, GAM (stringent)	unconst. distr. lag		1060 (800 - 1320)	2.0% (1.5% - 2.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	log-linear, natural spline	unconst. distr. lag		1050 (750 - 1360)	1.9% (1.4% - 2.5%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	log-linear, penalized spline	unconst. distr. lag		1060 (790 - 1330)	2.0% (1.5% - 2.5%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	log-linear, GAM (stringent)	unconst. distr. lag		360 (210 - 500)	2.8% (1.7% - 3.9%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	log-linear, natural spline	unconst. distr. lag		0 (0 - 150)	0.0% (0.0% - 1.2%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	log-linear, penalized spline	unconst. distr. lag		180 (30 - 320)	1.4% (0.3% - 2.5%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	log-linear, GAM (stringent)	unconst. distr. lag		280 (140 - 420)	4.3% (2.1% - 6.5%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	log-linear, natural spline	unconst. distr. lag		230 (70 - 390)	3.6% (1.1% - 6.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	log-linear, penalized spline	unconst. distr. lag		280 (140 - 420)	4.3% (2.1% - 6.5%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day	CO	570 (330 - 800)	1.0% (0.6% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day	CO	600 (340 - 850)	1.1% (0.6% - 1.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day	CO	240 (0 - 490)	0.4% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day	CO	250 (0 - 530)	0.5% (0.0% - 1.0%)
	Morris and Naumova (1998)	Congestive heart failure	65+	log-linear	0 day	CO, SO2, NO2, O3	160 (0 - 480)	0.7% (0.0% - 2.0%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.11. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Los Angeles, CA, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**							
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM	1 day		250 (0 - 1000)	0.4% (0.0% - 1.8%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		380 (30 - 720)	0.7% (0.1% - 1.3%)
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day		600 (0 - 1270)	1.1% (0.0% - 2.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		80 (0 - 770)	0.2% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	0 day		0 (0 - 0)	0.0% (0.0% - 0.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		120 (0 - 740)	0.2% (0.0% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		70 (0 - 800)	0.1% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		170 (0 - 780)	0.3% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	1 day		120 (0 - 860)	0.2% (0.0% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		0 (0 - 570)	0.0% (0.0% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		0 (0 - 710)	0.0% (0.0% - 1.2%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		80 (0 - 770)	0.2% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		170 (0 - 780)	0.3% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		790 (170 - 1400)	1.4% (0.3% - 2.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		0 (0 - 510)	0.0% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		240 (0 - 850)	0.4% (0.0% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0 (0 - 0)	0.0% (0.0% - 0.0%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		470 (50 - 890)	0.8% (0.1% - 1.6%)
	Single Pollutant Models (Cause-Specific Mortality)							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	0 day		200 (0 - 540)	1.0% (0.0% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	0 day		250 (0 - 560)	1.2% (0.0% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	0 day		270 (0 - 650)	1.3% (0.0% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	1 day		170 (0 - 520)	0.8% (0.0% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	1 day		120 (0 - 440)	0.6% (0.0% - 2.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	1 day		140 (0 - 540)	0.7% (0.0% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]***	Cardiovascular	all	log-linear, GAM (stringent), 100 df	2 day		470 (150 - 780)	2.3% (0.7% - 3.8%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		30 (0 - 160)	1.0% (0.0% - 5.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		20 (0 - 150)	0.8% (0.0% - 5.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	0 day		30 (0 - 190)	1.1% (0.0% - 6.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		70 (0 - 200)	2.5% (0.0% - 6.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		100 (0 - 230)	3.5% (0.0% - 7.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	1 day		100 (0 - 260)	3.5% (0.0% - 8.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	log-linear, GLM, Bayes adjusted	1 day		310 (20 - 590)	0.9% (0.1% - 1.8%)
	Multi-Pollutant Models (Total Mortality)							
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day	O3	600 (0 - 1270)	1.1% (0.0% - 2.2%)
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day	CO	480 (0 - 1050)	0.8% (0.0% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day	CO	0 (0 - 190)	0.0% (0.0% - 0.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3	470 (210 - 760)	0.8% (0.4% - 1.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, NO2	370 (0 - 760)	0.6% (0.0% - 1.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, SO2	370 (30 - 730)	0.6% (0.1% - 1.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, CO	420 (90 - 740)	0.7% (0.2% - 1.3%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Cause-Specific Mortality)							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	2 day	CO	310 (0 - 680)	1.5% (0.0% - 3.3%)
Hospital Admissions	**Single Pollutant Models**							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	0 day		1360 (490 - 2210)	2.0% (0.7% - 3.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day		1100 (250 - 1930)	1.6% (0.4% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day		1120 (50 - 2160)	1.6% (0.1% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	1 day		1040 (150 - 1910)	1.5% (0.2% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day		980 (120 - 1820)	1.4% (0.2% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day		1000 (0 - 2030)	1.4% (0.0% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		460 (190 - 720)	3.2% (1.3% - 5.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		320 (80 - 550)	2.2% (0.6% - 3.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	0 day		300 (0 - 590)	2.1% (0.0% - 4.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		470 (200 - 730)	3.2% (1.4% - 5.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		370 (130 - 600)	2.5% (0.9% - 4.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	1 day		330 (30 - 620)	2.3% (0.2% - 4.3%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
							Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	2 day		630 (360 - 900)	4.4% (2.5% - 6.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day		450 (210 - 690)	3.1% (1.5% - 4.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	2 day		410 (100 - 710)	2.9% (0.7% - 4.9%)
	Linn et al. (2000) -- Calif. South Coast Air Basin****	Asthma	30+	log-linear	0 day		60 (0 - 220)	0.9% (0.0% - 3.3%)
	Linn et al. (2000) -- Calif. South Coast Air Basin****	Asthma	<30	log-linear	0 day		180 (0 - 370)	3.3% (0.0% - 6.7%)
	Multi-Pollutant Models							
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day	CO	0 (0 - 490)	0.0% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day	CO	0 (0 - 810)	0.0% (0.0% - 1.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day	NO2	0 (0 - 340)	0.0% (0.0% - 2.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day	NO2	160 (0 - 480)	1.1% (0.0% - 3.3%)
	Nauenberg and Basu (1999)*****	Emergency and urgent asthma-related admissions	all	linear	0 day	O3	290 (30 - 540)	6.8% (0.8% - 12.7%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

***Health effects estimated using a 2-day lag cardiovascular mortality PM10 coefficient are provided here to compare with estimates from the multi-pollutant coefficient for the same lag, model and endpoints. The Moolgavkar (2002) reanalysis did not estimate cardiovascular mortality using a 0 or 1 day lag multi-pollutant PM10 coefficient.

****The California South Coast Air Basin represents the area included in Los Angeles, Riverside, San Bernadino and Orange Counties, excluding the mountain and desert regions of the first three counties.

*****The Nauenberg and Basu (1999) study examined the effect of PM2.5 levels on emergency and urgent asthma-related hospital admissions, excluding scheduled admissions and transfers from other facilities.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city or multi-county short-term exposure C-R functions were applied only to counties included among those used to estimate the function.

Exhibit D.12. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Minneapolis/St. Paul, MN, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	unconst. distr. lag		480 (230 - 710)	3.7% (1.8% - 5.6%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		160 (130 - 200)	1.3% (1.0% - 1.5%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	unconst. distr. lag		280 (210 - 340)	2.2% (1.7% - 2.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		50 (0 - 100)	0.4% (0.0% - 0.8%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		30 (0 - 110)	0.3% (0.0% - 0.9%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		30 (0 - 60)	0.5% (0.0% - 1.2%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	unconst. distr. lag		60 (30 - 80)	1.9% (1.2% - 2.9%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	unconst. distr. lag		20 (0 - 40)	3.6% (0.2% - 6.8%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	unconst. distr. lag		30 (20 - 40)	5.7% (3.0% - 8.3%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)						
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	160 (80 - 230)	1.2% (0.6% - 1.8%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	220 (130 - 300)	1.7% (1.1% - 2.4%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	170 (110 - 230)	1.3% (0.8% - 1.8%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	60 (30 - 100)	0.5% (0.2% - 0.9%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	50 (0 - 100)	0.4% (0.0% - 0.9%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	50 (0 - 100)	0.4% (0.0% - 0.8%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	60 (10 - 100)	0.5% (0.1% - 0.8%)
Hospital Admissions	**Single Pollutant Models**						
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	unconst. distr. lag		120 (0 - 380)	1.0% (0.0% - 3.2%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		250 (190 - 310)	2.1% (1.6% - 2.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		90 (0 - 180)	6.6% (0.0% - 12.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		70 (30 - 100)	4.7% (2.3% - 7.1%)
	Schwartz (1994b)	COPD+	65+	0 day		140 (60 - 210)	8.3% (3.5% - 12.9%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		120 (0 - 240)	3.7% (0.0% - 7.6%)
	Schwartz (1994b)	Pneumonia	65+	0 day		90 (20 - 160)	3.0% (0.6% - 5.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		100 (60 - 130)	3.1% (1.8% - 4.3%)
	Multi-Pollutant Models Schwartz (1994b)	Pneumonia	65+	0 day	O3	90 (10 - 160)	3.0% (0.4% - 5.4%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.13. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Lipfert et al. (2000)	Non-accidental	all	0 day		380	2.2%
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		70 (0 - 150)	0.4% (0.0% - 0.8%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		120 (10 - 230)	0.7% (0.1% - 1.3%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day		540 (250 - 830)	7.8% (3.6% - 12.0%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		70 (10 - 130)	0.9% (0.1% - 1.6%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)						
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	260	1.5%
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	80 (40 - 130)	0.5% (0.2% - 0.8%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	60 (0 - 130)	0.4% (0.0% - 0.8%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	60 (10 - 130)	0.4% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	70 (20 - 130)	0.4% (0.1% - 0.7%)
	Multi-Pollutant Models (Cause-Specific Mortality)						
	Lipfert et al. (2000) -- 7 counties	Cardiovascluar	all	0 day	O3	150	2.2%

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM10 non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Note 2: Multi-city or multi-county short-term exposure C-R functions were applied only to counties included among those used to estimate the function.

Exhibit D.14. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Phoenix, AZ, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		200 (0 - 400)	0.9% (0.0% - 1.9%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		170 (0 - 420)	0.8% (0.0% - 2.0%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		180 (10 - 340)	1.6% (0.1% - 3.1%)
	Mar et al. (2003) [reanalysis of Mar et al (2000)]	Cardiovascular	65-100	0 day		530 (120 - 960)	7.4% (1.7% - 13.4%)
	Mar et al. (2003) [reanalysis of Mar et al (2000)]	Cardiovascular	65-100	1 day		410 (0 - 860)	5.8% (0.0% - 12.0%)
	Multi-Pollutant Models (Total Mortality)						
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	240 (110 - 390)	1.1% (0.5% - 1.8%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	190 (0 - 390)	0.9% (0.0% - 1.8%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	190 (20 - 370)	0.9% (0.1% - 1.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	210 (40 - 380)	1.0% (0.2% - 1.8%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.15. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Pittsburgh, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	unconst. distr. lag		120 (10 - 220)	0.8% (0.1% - 1.5%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		120 (100 - 150)	0.8% (0.7% - 1.0%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	unconst. distr. lag		210 (160 - 260)	1.4% (1.1% - 1.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		40 (10 - 70)	0.3% (0.1% - 0.5%)
	Chock et al. (2000)	Non-accidental	<75	0 day		40 (0 - 80)	0.8% (0.1% - 1.5%)
	Chock et al. (2000)	Non-accidental	75+	0 day		40 (0 - 110)	0.5% (0.0% - 1.2%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		40 (10 - 60)	0.2% (0.1% - 0.4%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
	Single Pollutant Models (Cause-Specific Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		30 (10 - 50)	0.4% (0.1% - 0.6%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	unconst. distr. lag		60 (40 - 100)	1.2% (0.8% - 1.9%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	unconst. distr. lag		10 (0 - 30)	2.3% (0.1% - 4.4%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	unconst. distr. lag		20 (10 - 30)	3.7% (2.0% - 5.4%)
	Multi-Pollutant Models (Total Mortality)						
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	120 (60 - 180)	0.8% (0.4% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	170 (100 - 230)	1.1% (0.7% - 1.5%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	130 (80 - 180)	0.9% (0.5% - 1.2%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	50 (20 - 80)	0.3% (0.2% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	40 (0 - 80)	0.3% (0.0% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	40 (0 - 80)	0.3% (0.0% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	40 (10 - 80)	0.3% (0.1% - 0.5%)
	Chock et al. (2000)	Non-accidental	<75	0 day	NO2	60 (10 - 100)	1.1% (0.2% - 1.9%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO	80 (0 - 160)	0.9% (0.0% - 1.8%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**						
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	unconst. distr. lag		300 (170 - 430)	1.4% (0.8% - 2.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		290 (220 - 360)	1.4% (1.0% - 1.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		100 (40 - 160)	3.0% (1.2% - 4.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		110 (50 - 160)	3.1% (1.5% - 4.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		60 (0 - 120)	1.4% (0.1% - 2.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		90 (50 - 130)	2.0% (1.2% - 2.8%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.16. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Provo, UT, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]***	Non-accidental	all	1 day		10 (0 - 10)	0.4% (0.0% - 0.9%)
	Multi-Pollutant Models (Total Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	10 (0 - 10)	0.6% (0.3% - 1.0%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	10 (0 - 10)	0.5% (0.0% - 1.0%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	10 (0 - 10)	0.5% (0.1% - 0.9%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	10 (0 - 10)	0.5% (0.1% - 1.0%)
Hospital Admissions	**Single Pollutant Models**						
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	unconst. distr. lag		20 (0 - 70)	1.6% (0.0% - 5.2%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		40 (30 - 40)	2.5% (1.9% - 3.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		0 (0 - 0)	0.0% (0.0% - 0.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		0 (0 - 10)	5.4% (2.7% - 8.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		30 (0 - 50)	6.9% (1.0% - 12.3%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		10 (10 - 20)	3.5% (2.1% - 4.9%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Respiratory Symptoms****	**Single Pollutant Models**						
	Pope et al. (1991)	Upper respiratory disease	9 -11	0 day		39000 (7000 - 69000)	5.5% (1.0% - 9.8%)
	Pope et al. (1991)	Lower respiratory disease	9 -11	0 day		31000 (7000 - 52000)	9.4% (2.1% - 15.9%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The Dominici et al. (2003) [reanalysis of Samet et al. (2000)] C-R function used in Provo was estimated in Salt Lake City, UT, which is close to Provo.

****The C-R functions for upper and lower respiratory symptoms were calculated for a 3.5 month period starting on December 1rst.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.17. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
San Jose, CA, 1999

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		30 (0 - 60)	0.4% (0.0% - 0.7%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day		210 (80 - 350)	2.5% (0.9% - 4.0%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	1 day		0 (0 - 100)	0.0% (0.0% - 1.1%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		10 (0 - 30)	0.3% (0.0% - 0.7%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		20 (0 - 40)	0.5% (0.0% - 0.9%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Respiratory	all	0 day		40 (0 - 90)	3.3% (0.0% - 7.5%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Cardiovascular	all	0 day		100 (10 - 190)	2.7% (0.2% - 5.0%)
	Multi-Pollutant Models (Total Mortality)						
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	40 (20 - 60)	0.5% (0.2% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	30 (0 - 60)	0.4% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	30 (0 - 60)	0.4% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	30 (10 - 60)	0.4% (0.1% - 0.7%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.18. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
Seattle, WA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	unconst. distr. lag		210 (110 - 300)	1.7% (0.9% - 2.5%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		90 (70 - 110)	0.8% (0.6% - 1.0%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	unconst. distr. lag		160 (120 - 200)	1.3% (1.0% - 1.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		30 (0 - 60)	0.3% (0.0% - 0.5%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		20 (0 - 50)	0.2% (0.0% - 0.5%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		20 (0 - 40)	0.4% (0.0% - 0.7%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	unconst. distr. lag		40 (20 - 60)	1.2% (0.7% - 1.8%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	unconst. distr. lag		10 (0 - 20)	2.2% (0.1% - 4.2%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	unconst. distr. lag		20 (10 - 30)	3.6% (1.9% - 5.2%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)						
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	90 (50 - 140)	0.8% (0.4% - 1.1%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	130 (80 - 180)	1.1% (0.6% - 1.5%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	100 (60 - 130)	0.8% (0.5% - 1.1%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	40 (20 - 60)	0.3% (0.1% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	30 (0 - 60)	0.3% (0.0% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	30 (0 - 60)	0.3% (0.0% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	30 (10 - 60)	0.3% (0.1% - 0.5%)
Hospital Admissions	**Single Pollutant Models**						
	Sheppard (2003) [reanalysis of Sheppard et al. (1999)]***	Asthma	<65	1 day		40 (10 - 70)	2.4% (0.6% - 4.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	unconst. distr. lag		160 (80 - 240)	1.7% (0.8% - 2.5%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		120 (90 - 160)	1.3% (1.0% - 1.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		70 (40 - 90)	6.1% (3.7% - 8.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		30 (20 - 50)	2.9% (1.4% - 4.4%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		50 (0 - 90)	1.7% (0.1% - 3.3%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		50 (30 - 70)	1.9% (1.1% - 2.7%)
Respiratory Symptoms****	**Single Pollutant Models** Yu et al. (2000)	Asthma	5-12	1 day		108000 (33000 - 168000)	4.0% (1.2% - 6.3%)
	Multi-Pollutant Models Yu et al. (2000)	Asthma	5-12	1 day	CO, SO2	55000 (0 - 168000)	2.0% (0.0% - 6.3%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM2.5 values obtained from nephelometer measurements rather than from air quality monitors.

****The C-R functions for asthma symptoms were calculated for children 7 to 12 with a history of chronic mild to moderate asthma.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.19. Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Non-accidental	all	0 day		140 (0 - 270)	0.6% (0.0% - 1.2%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Non-accidental	all	0 day		240 (140 - 340)	1.0% (0.6% - 1.5%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		70 (10 - 120)	0.3% (0.1% - 0.5%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		50 (10 - 90)	0.4% (0.1% - 0.7%)
	Multi-Pollutant Models (Total Mortality)						
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	90 (40 - 150)	0.4% (0.2% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	70 (0 - 150)	0.3% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	70 (10 - 140)	0.3% (0.0% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	80 (20 - 150)	0.4% (0.1% - 0.6%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background	
						Incidence**	Percent of Total Incidence**
Respiratory Symptoms* **	**Single Pollutant Models**						
	Schwartz et al. (1994) -- 6 cities	Lower respiratory symptoms	7 - 14	1 day		4000 (2000 - 6000)	7.1% (3.3% - 10.2%)
	Schwartz et al. (1994) -- 6 cities	Upper respiratory symptoms	7 - 14	2 day		6000 (0 - 11000)	3.6% (0.0% - 7.0%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

D.6. The $PM_{10-2.5}$ data: Primary analysis

Exhibit D.20. Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Lipfert et al. (2000)	Non-accidental	all	0 day		230	1.4%
	Single Pollutant Models (Cause-Specific Mortality)						
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day		260 (0 - 550)	3.8% (0.0% - 7.9%)
	Multi-Pollutant Models (Total Mortality)						
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	190	1.1%
	Multi-Pollutant Models (Cause-Specific Mortality)						
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	0 day	O3	100	1.4%

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM coarse non-accidental mortality coefficients. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Exhibit D.21. Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations
Phoenix, AZ, 1997

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Cause-Specific Mortality)**						
	Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	65+	0 day		480 (100 - 830)	6.7% (1.5% - 11.6%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

Exhibit D.22. Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations
Seattle, WA, 2000

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models** Sheppard (2003) [reanalysis of Sheppard et al. (1999)]***	Asthma	<65	1 day		10 (0 - 20)	0.6% (0.0% - 1.4%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

***Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM coarse values obtained from nephelometer measurements rather than from air quality monitors.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

Exhibit D.23. Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background	
						Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**						
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	0 day		0 (0 - 40)	0.0% (0.0% - 0.2%)
Respiratory Symptoms* **	**Single Pollutant Models**						
	Schwartz and Neas, 2000 -- 6 cities	Lower respiratory symptoms	7-14	0 day		2000 (0 - 7000)	4.1% (0.0% - 12.7%)
	Schwartz and Neas, 2000 -- 6 cities	Cough	7-14	0 day		9000 (4000 - 14000)	5.5% (2.2% - 8.4%)
	Multi-Pollutant Models						
	Schwartz and Neas, 2000 -- 6 cities	Lower respiratory symptoms	7-14	0 day	PM2.5	1000 (0 - 3000)	1.6% (0.0% - 6.1%)
	Schwartz and Neas, 2000 -- 6 cities	Cough	7-14	0 day	PM2.5	8000 (2000 - 13000)	5.0% (1.3% - 8.2%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.24. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
Boston, MA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		430 (290 - 570)	1.9% (1.3% - 2.6%)	370 (250 - 480)	1.6% (1.1% - 2.2%)	300 (200 - 390)	1.3% (0.9% - 1.8%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		290 (210 - 370)	1.3% (0.9% - 1.7%)	240 (170 - 310)	1.1% (0.8% - 1.4%)	200 (140 - 260)	0.9% (0.6% - 1.1%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	all	0 day		20 (0 - 60)	2.6% (0.0% - 6.0%)	20 (0 - 50)	2.2% (0.0% - 5.1%)	20 (0 - 40)	1.8% (0.0% - 4.1%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	all	0 day		100 (60 - 140)	2.5% (1.4% - 3.6%)	80 (50 - 120)	2.1% (1.2% - 3.0%)	70 (40 - 100)	1.7% (1.0% - 2.5%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	all	0 day		70 (30 - 100)	5.3% (2.4% - 7.8%)	60 (30 - 80)	4.4% (2.0% - 6.6%)	50 (20 - 70)	3.6% (1.7% - 5.4%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	COPD	all	0 day		20 (0 - 40)	2.1% (0.1% - 4.1%)	20 (0 - 30)	1.8% (0.1% - 3.4%)	10 (0 - 30)	1.5% (0.1% - 2.8%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Ischemic heart disease	all	0 day		70 (40 - 90)	1.7% (1.0% - 2.3%)	60 (30 - 80)	1.4% (0.9% - 2.0%)	50 (30 - 60)	1.2% (0.7% - 1.6%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Pneumonia	all	0 day		50 (20 - 70)	3.7% (1.8% - 5.5%)	40 (20 - 60)	3.2% (1.5% - 4.7%)	30 (20 - 50)	2.6% (1.2% - 3.8%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Long-Term Exposure Mortality	**Single Pollutant Models**										
	Krewski et al. (2000) - Six Cities	All cause	25+			120 (40 - 210)	0.5% (0.2% - 0.9%)	120 (40 - 210)	0.5% (0.2% - 0.9%)	120 (40 - 210)	0.5% (0.2% - 0.9%)
	Krewski et al. (2000) - ACS	All cause	30+			150 (80 - 230)	0.7% (0.3% - 1.0%)	150 (80 - 230)	0.7% (0.3% - 1.0%)	150 (80 - 230)	0.7% (0.3% - 1.0%)
	Krewski et al. (2000) - Six Cities	Cardiopulmonary	25+			70 (20 - 110)	0.7% (0.2% - 1.2%)	70 (20 - 110)	0.7% (0.2% - 1.2%)	70 (20 - 110)	0.7% (0.2% - 1.2%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			150 (90 - 200)	1.3% (0.9% - 1.9%)	150 (90 - 200)	1.3% (0.9% - 1.9%)	150 (90 - 200)	1.3% (0.9% - 1.9%)
	Pope et al. (2002) - ACS extended	All cause	30+			130 (30 - 240)	0.6% (0.1% - 1.0%)	130 (30 - 240)	0.6% (0.1% - 1.0%)	130 (30 - 240)	0.6% (0.1% - 1.0%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			90 (20 - 150)	0.8% (0.2% - 1.4%)	90 (20 - 150)	0.8% (0.2% - 1.4%)	90 (20 - 150)	0.8% (0.2% - 1.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			20 (0 - 30)	1.1% (0.2% - 2.1%)	20 (0 - 30)	1.1% (0.2% - 2.1%)	20 (0 - 30)	1.1% (0.2% - 2.1%)
	Multi-Pollutant Models										
	Krewski et al. (2000) - ACS	All cause	30+		CO	220 (130 - 320)	1.0% (0.6% - 1.4%)	220 (130 - 320)	1.0% (0.6% - 1.4%)	220 (130 - 320)	1.0% (0.6% - 1.4%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	260 (140 - 380)	1.2% (0.6% - 1.7%)	260 (140 - 380)	1.2% (0.6% - 1.7%)	260 (140 - 380)	1.2% (0.6% - 1.7%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	220 (130 - 320)	1.0% (0.6% - 1.4%)	220 (130 - 320)	1.0% (0.6% - 1.4%)	220 (130 - 320)	1.0% (0.6% - 1.4%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	40 (0 - 160)	0.2% (0.0% - 0.7%)	40 (0 - 160)	0.2% (0.0% - 0.7%)	40 (0 - 160)	0.2% (0.0% - 0.7%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Respiratory Symptoms* **	**Single Pollutant Models**										
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day		9000 (5000 - 17000)	17.4% (8.9% - 32.8%)	8000 (4000 - 15000)	15.0% (7.6% - 28.4%)	7000 (3000 - 12000)	12.7% (6.4% - 23.8%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day		15000 (0 - 28000)	9.6% (0.0% - 18.5%)	13000 (0 - 24000)	8.3% (0.0% - 15.9%)	10000 (0 - 20000)	6.9% (0.0% - 13.3%)
	Multi-Pollutant Models										
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day	PM10-2.5	8000 (3000 - 17000)	15.8% (5.1% - 32.4%)	7000 (2000 - 15000)	13.6% (4.3% - 28.1%)	6000 (2000 - 12000)	11.5% (3.6% - 23.5%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day	PM10-2.5	7000 (0 - 22000)	4.6% (0.0% - 14.4%)	6000 (0 - 19000)	3.9% (0.0% - 12.4%)	5000 (0 - 16000)	3.3% (0.0% - 10.4%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.25. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
Boston, MA, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		1.6% (1.1% - 2.2%)	0.7% (0.5% - 0.9%)	0.3% (0.2% - 0.5%)	0.2% (0.1% - 0.2%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		1.1% (0.8% - 1.4%)	0.4% (0.3% - 0.5%)	0.2% (0.2% - 0.3%)	0.1% (0.1% - 0.1%)
Long-Term Exposure Mortality						**BASE CASE: Background =3.5 µg/m3**	**Hypothetical Threshold =10 µg/m3**	**Hypothetical Threshold =12.5 µg/m3**	**Hypothetical Threshold =15 µg/m3**
	Single Pollutant Models								
	Krewski et al. (2000) - Six Cities	All cause	25+			0.5% (0.2% - 0.9%)	0.5% (0.2% - 0.9%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+			0.7% (0.3% - 1.0%)	0.7% (0.3% - 1.0%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Pope et al. (2002) - ACS extended	All cause	30+			0.6% (0.1% - 1.0%)	0.6% (0.1% - 1.0%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)

	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	1.0% (0.6% - 1.4%)	1.0% (0.6% - 1.4%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	1.2% (0.6% - 1.7%)	1.2% (0.6% - 1.7%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	1.0% (0.6% - 1.4%)	1.0% (0.6% - 1.4%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	0.2% (0.0% - 0.7%)	0.2% (0.0% - 0.7%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.26. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Boston, MA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		370 (250 - 480)	1.6% (1.1% - 2.2%)	710 (490 - 940)	3.2% (2.2% - 4.2%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		240 (170 - 310)	1.1% (0.8% - 1.4%)	480 (340 - 610)	2.1% (1.5% - 2.7%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.27. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations
Boston, MA, 2002

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence*		
					Base Case: Assuming AQ as Reported	Assuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - Six Cities	All cause	25+		0.5% (0.2% - 0.9%)	0.4% (0.1% - 0.6%)	0.3% (0.1% - 0.5%)
	Krewski et al. (2000) - ACS	All cause	30+		0.7% (0.3% - 1.0%)	0.4% (0.2% - 0.7%)	0.3% (0.2% - 0.5%)
	Krewski et al. (2000) - Six Cities	Cardiopulmonary	25+		0.7% (0.2% - 1.2%)	0.5% (0.2% - 0.8%)	0.4% (0.1% - 0.6%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		1.3% (0.9% - 1.9%)	0.9% (0.6% - 1.3%)	0.7% (0.4% - 0.9%)
	Pope et al. (2002) - ACS extended	All cause	30+		0.6% (0.1% - 1.0%)	0.4% (0.1% - 0.7%)	0.3% (0.1% - 0.5%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+		0.8% (0.2% - 1.4%)	0.6% (0.1% - 1.0%)	0.4% (0.1% - 0.7%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+		1.1% (0.2% - 2.1%)	0.8% (0.1% - 1.4%)	0.6% (0.1% - 1.1%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+	CO	1.0% (0.6% - 1.4%)	0.6% (0.4% - 0.9%)	0.5% (0.3% - 0.7%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	1.2% (0.6% - 1.7%)	0.8% (0.4% - 1.1%)	0.6% (0.3% - 0.8%)
	Krewski et al. (2000) - ACS	All cause	30+	O3	1.0% (0.6% - 1.4%)	0.6% (0.4% - 0.9%)	0.5% (0.3% - 0.7%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	0.2% (0.0% - 0.7%)	0.1% (0.0% - 0.5%)	0.1% (0.0% - 0.4%)

* Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.28. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
Los Angeles, CA, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		500 (0 - 1060)	0.9% (0.0% - 1.9%)	500 (0 - 1060)	0.9% (0.0% - 1.9%)	500 (0 - 1060)	0.9% (0.0% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		550 (0 - 1090)	1.0% (0.0% - 1.9%)	550 (0 - 1090)	1.0% (0.0% - 1.9%)	550 (0 - 1090)	1.0% (0.0% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		360 (0 - 880)	0.6% (0.0% - 1.5%)	360 (0 - 880)	0.6% (0.0% - 1.5%)	360 (0 - 880)	0.6% (0.0% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		0 (0 - 400)	0.0% (0.0% - 0.7%)	0 (0 - 400)	0.0% (0.0% - 0.7%)	0 (0 - 400)	0.0% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		0 (0 - 430)	0.0% (0.0% - 0.8%)	0 (0 - 430)	0.0% (0.0% - 0.8%)	0 (0 - 430)	0.0% (0.0% - 0.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0 (0 - 30)	0.0% (0.0% - 0.1%)	0 (0 - 30)	0.0% (0.0% - 0.1%)	0 (0 - 30)	0.0% (0.0% - 0.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		500 (0 - 1060)	0.9% (0.0% - 1.9%)	500 (0 - 1060)	0.9% (0.0% - 1.9%)	500 (0 - 1060)	0.9% (0.0% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	0 day		370 (0 - 1050)	0.7% (0.0% - 1.8%)	370 (0 - 1050)	0.7% (0.0% - 1.8%)	370 (0 - 1050)	0.7% (0.0% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		300 (0 - 800)	0.5% (0.0% - 1.4%)	300 (0 - 800)	0.5% (0.0% - 1.4%)	300 (0 - 800)	0.5% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		280 (0 - 950)	0.5% (0.0% - 1.7%)	280 (0 - 950)	0.5% (0.0% - 1.7%)	280 (0 - 950)	0.5% (0.0% - 1.7%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		550 (0 - 1090)	1.0% (0.0% - 1.9%)	550 (0 - 1090)	1.0% (0.0% - 1.9%)	550 (0 - 1090)	1.0% (0.0% - 1.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	1 day		510 (0 - 1170)	0.9% (0.0% - 2.0%)	510 (0 - 1170)	0.9% (0.0% - 2.0%)	510 (0 - 1170)	0.9% (0.0% - 2.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		90 (0 - 610)	0.2% (0.0% - 1.1%)	90 (0 - 610)	0.2% (0.0% - 1.1%)	90 (0 - 610)	0.2% (0.0% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		0 (0 - 900)	0.0% (0.0% - 1.6%)	0 (0 - 900)	0.0% (0.0% - 1.6%)	0 (0 - 900)	0.0% (0.0% - 1.6%)
	Single Pollutant Models (Cause-Specific Mortality)											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	0 day		340 (30 - 630)	1.6% (0.2% - 3.0%)	340 (30 - 630)	1.6% (0.2% - 3.0%)	340 (30 - 630)	1.6% (0.2% - 3.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	0 day		330 (50 - 600)	1.6% (0.2% - 2.9%)	330 (50 - 600)	1.6% (0.2% - 2.9%)	330 (50 - 600)	1.6% (0.2% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	0 day		330 (0 - 650)	1.6% (0.0% - 3.1%)	330 (0 - 650)	1.6% (0.0% - 3.1%)	330 (0 - 650)	1.6% (0.0% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	1 day		350 (50 - 640)	1.7% (0.3% - 3.1%)	350 (50 - 640)	1.7% (0.3% - 3.1%)	350 (50 - 640)	1.7% (0.3% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	1 day		270 (0 - 550)	1.3% (0.0% - 2.6%)	270 (0 - 550)	1.3% (0.0% - 2.6%)	270 (0 - 550)	1.3% (0.0% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	1 day		240 (0 - 570)	1.1% (0.0% - 2.7%)	240 (0 - 570)	1.1% (0.0% - 2.7%)	240 (0 - 570)	1.1% (0.0% - 2.7%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		0 (0 - 90)	0.0% (0.0% - 3.0%)	0 (0 - 90)	0.0% (0.0% - 3.0%)	0 (0 - 90)	0.0% (0.0% - 3.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		0 (0 - 50)	0.0% (0.0% - 1.6%)	0 (0 - 50)	0.0% (0.0% - 1.6%)	0 (0 - 50)	0.0% (0.0% - 1.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	0 day		0 (0 - 80)	0.0% (0.0% - 2.7%)	0 (0 - 80)	0.0% (0.0% - 2.7%)	0 (0 - 80)	0.0% (0.0% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		20 (0 - 130)	0.6% (0.0% - 4.5%)	20 (0 - 130)	0.6% (0.0% - 4.5%)	20 (0 - 130)	0.6% (0.0% - 4.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		40 (0 - 150)	1.4% (0.0% - 5.2%)	40 (0 - 150)	1.4% (0.0% - 5.2%)	40 (0 - 150)	1.4% (0.0% - 5.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	1 day		10 (0 - 150)	0.3% (0.0% - 5.0%)	10 (0 - 150)	0.3% (0.0% - 5.0%)	10 (0 - 150)	0.3% (0.0% - 5.0%)
	Multi-Pollutant Models (Total Mortality)											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day	CO	0 (0 - 240)	0.0% (0.0% - 0.4%)	0 (0 - 240)	0.0% (0.0% - 0.4%)	0 (0 - 240)	0.0% (0.0% - 0.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day	CO	0 (0 - 360)	0.0% (0.0% - 0.6%)	0 (0 - 360)	0.0% (0.0% - 0.6%)	0 (0 - 360)	0.0% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day	CO	0 (0 - 480)	0.0% (0.0% - 0.8%)	0 (0 - 480)	0.0% (0.0% - 0.8%)	0 (0 - 480)	0.0% (0.0% - 0.8%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Cause-Specific Mortality)											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	0 day	CO	600 (260 - 930)	2.9% (1.2% - 4.4%)	600 (260 - 930)	2.9% (1.2% - 4.4%)	600 (260 - 930)	2.9% (1.2% - 4.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	0 day	CO	630 (230 - 1020)	3.0% (1.1% - 4.9%)	630 (230 - 1020)	3.0% (1.1% - 4.9%)	630 (230 - 1020)	3.0% (1.1% - 4.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	1 day	CO	310 (0 - 650)	1.5% (0.0% - 3.1%)	310 (0 - 650)	1.5% (0.0% - 3.1%)	310 (0 - 650)	1.5% (0.0% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	1 day	CO	310 (0 - 720)	1.5% (0.0% - 3.4%)	310 (0 - 720)	1.5% (0.0% - 3.4%)	310 (0 - 720)	1.5% (0.0% - 3.4%)
Long-Term Exposure Mortality	**Single Pollutant Models**											
	Krewski et al. (2000) - ACS	All cause	30+	log-linear			2730 (1420 - 4140)	4.7% (2.5% - 7.1%)	2730 (1420 - 4140)	4.7% (2.5% - 7.1%)	2730 (1420 - 4140)	4.7% (2.5% - 7.1%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+	log-linear			3030 (1980 - 4150)	9.4% (6.1% - 12.8%)	3030 (1980 - 4150)	9.4% (6.1% - 12.8%)	3030 (1980 - 4150)	9.4% (6.1% - 12.8%)
	Pope et al. (2002) - ACS extended	All cause	30+	log-linear			2380 (480 - 4220)	4.1% (0.8% - 7.3%)	2380 (480 - 4220)	4.1% (0.8% - 7.3%)	2380 (480 - 4220)	4.1% (0.8% - 7.3%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+	log-linear			1880 (500 - 3200)	5.8% (1.5% - 9.9%)	1880 (500 - 3200)	5.8% (1.5% - 9.9%)	1880 (500 - 3200)	5.8% (1.5% - 9.9%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+	log-linear			260 (40 - 460)	7.9% (1.1% - 14.2%)	260 (40 - 460)	7.9% (1.1% - 14.2%)	260 (40 - 460)	7.9% (1.1% - 14.2%)
	Multi-Pollutant Models											
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		CO	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		NO2	4710 (2520 - 6630)	8.1% (4.3% - 11.4%)	4710 (2520 - 6630)	8.1% (4.3% - 11.4%)	4710 (2520 - 6630)	8.1% (4.3% - 11.4%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		O3	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		SO2	730 (0 - 2940)	1.3% (0.0% - 5.1%)	730 (0 - 2940)	1.3% (0.0% - 5.1%)	730 (0 - 2940)	1.3% (0.0% - 5.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	0 day		1770 (1030 - 2490)	2.6% (1.5% - 3.6%)	1770 (1030 - 2490)	2.6% (1.5% - 3.6%)	1770 (1030 - 2490)	2.6% (1.5% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day		1300 (580 - 2020)	1.9% (0.8% - 2.9%)	1300 (580 - 2020)	1.9% (0.8% - 2.9%)	1300 (580 - 2020)	1.9% (0.8% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day		1410 (510 - 2290)	2.0% (0.7% - 3.3%)	1410 (510 - 2290)	2.0% (0.7% - 3.3%)	1410 (510 - 2290)	2.0% (0.7% - 3.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	1 day		1560 (780 - 2310)	2.3% (1.1% - 3.3%)	1560 (780 - 2310)	2.3% (1.1% - 3.3%)	1560 (780 - 2310)	2.3% (1.1% - 3.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day		1270 (530 - 1990)	1.8% (0.8% - 2.9%)	1270 (530 - 1990)	1.8% (0.8% - 2.9%)	1270 (530 - 1990)	1.8% (0.8% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day		1350 (440 - 2230)	1.9% (0.6% - 3.2%)	1350 (440 - 2230)	1.9% (0.6% - 3.2%)	1350 (440 - 2230)	1.9% (0.6% - 3.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		810 (340 - 1270)	2.7% (1.1% - 4.2%)	810 (340 - 1270)	2.7% (1.1% - 4.2%)	810 (340 - 1270)	2.7% (1.1% - 4.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		680 (260 - 1080)	2.2% (0.9% - 3.6%)	680 (260 - 1080)	2.2% (0.9% - 3.6%)	680 (260 - 1080)	2.2% (0.9% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	0 day		730 (210 - 1230)	2.4% (0.7% - 4.1%)	730 (210 - 1230)	2.4% (0.7% - 4.1%)	730 (210 - 1230)	2.4% (0.7% - 4.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		580 (110 - 1040)	1.9% (0.4% - 3.4%)	580 (110 - 1040)	1.9% (0.4% - 3.4%)	580 (110 - 1040)	1.9% (0.4% - 3.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		370 (0 - 780)	1.2% (0.0% - 2.6%)	370 (0 - 780)	1.2% (0.0% - 2.6%)	370 (0 - 780)	1.2% (0.0% - 2.6%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	1 day		380 (0 - 880)	1.3% (0.0% - 2.9%)	380 (0 - 880)	1.3% (0.0% - 2.9%)	380 (0 - 880)	1.3% (0.0% - 2.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	2 day		900 (410 - 1370)	3.0% (1.4% - 4.5%)	900 (410 - 1370)	3.0% (1.4% - 4.5%)	900 (410 - 1370)	3.0% (1.4% - 4.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day		560 (110 - 1000)	1.9% (0.4% - 3.3%)	560 (110 - 1000)	1.9% (0.4% - 3.3%)	560 (110 - 1000)	1.9% (0.4% - 3.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	2 day		510 (0 - 1050)	1.7% (0.0% - 3.5%)	510 (0 - 1050)	1.7% (0.0% - 3.5%)	510 (0 - 1050)	1.7% (0.0% - 3.5%)
	Multi-Pollutant Models											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day	CO	440 (0 - 1360)	0.6% (0.0% - 2.0%)	440 (0 - 1360)	0.6% (0.0% - 2.0%)	440 (0 - 1360)	0.6% (0.0% - 2.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day	CO	660 (0 - 1740)	1.0% (0.0% - 2.5%)	660 (0 - 1740)	1.0% (0.0% - 2.5%)	660 (0 - 1740)	1.0% (0.0% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day	CO	270 (0 - 1260)	0.4% (0.0% - 1.8%)	270 (0 - 1260)	0.4% (0.0% - 1.8%)	270 (0 - 1260)	0.4% (0.0% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day	CO	310 (0 - 1440)	0.4% (0.0% - 2.1%)	310 (0 - 1440)	0.4% (0.0% - 2.1%)	310 (0 - 1440)	0.4% (0.0% - 2.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day	NO2	210 (0 - 840)	0.7% (0.0% - 2.8%)	210 (0 - 840)	0.7% (0.0% - 2.8%)	210 (0 - 840)	0.7% (0.0% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day	NO2	0 (0 - 740)	0.0% (0.0% - 2.5%)	0 (0 - 740)	0.0% (0.0% - 2.5%)	0 (0 - 740)	0.0% (0.0% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day	NO2	170 (0 - 830)	0.6% (0.0% - 2.8%)	170 (0 - 830)	0.6% (0.0% - 2.8%)	170 (0 - 830)	0.6% (0.0% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	3 day	NO2	0 (0 - 100)	0.0% (0.0% - 0.4%)	0 (0 - 100)	0.0% (0.0% - 0.4%)	0 (0 - 100)	0.0% (0.0% - 0.4%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.29. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
Los Angeles, CA, 2002

Health Effects**	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-2.5 Above Hypothetical Threshold***			
							BASE CASE: Background =2.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**									
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		0.9% (0.0% - 1.9%)	0.6% (0.0% - 1.3%)	0.4% (0.0% - 0.9%)	0.3% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		1.0% (0.0% - 1.9%)	0.7% (0.0% - 1.4%)	0.5% (0.0% - 0.9%)	0.3% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		0.6% (0.0% - 1.5%)	0.4% (0.0% - 1.1%)	0.3% (0.0% - 0.8%)	0.2% (0.0% - 0.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		0.0% (0.0% - 0.7%)	0.0% (0.0% - 0.5%)	0.0% (0.0% - 0.3%)	0.0% (0.0% - 0.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		0.0% (0.0% - 0.8%)	0.0% (0.0% - 0.5%)	0.0% (0.0% - 0.4%)	0.0% (0.0% - 0.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0.0% (0.0% - 0.1%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)

	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		0.9% (0.0% - 1.9%)	0.6% (0.0% - 1.3%)	0.4% (0.0% - 0.9%)	0.3% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	0 day		0.7% (0.0% - 1.8%)	0.5% (0.0% - 1.3%)	0.3% (0.0% - 0.9%)	0.2% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		0.5% (0.0% - 1.4%)	0.4% (0.0% - 1.0%)	0.3% (0.0% - 0.7%)	0.2% (0.0% - 0.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		0.5% (0.0% - 1.7%)	0.4% (0.0% - 1.2%)	0.2% (0.0% - 0.8%)	0.2% (0.0% - 0.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		1.0% (0.0% - 1.9%)	0.7% (0.0% - 1.4%)	0.5% (0.0% - 0.9%)	0.3% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	1 day		0.9% (0.0% - 2.0%)	0.6% (0.0% - 1.5%)	0.4% (0.0% - 1.0%)	0.3% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		0.2% (0.0% - 1.1%)	0.1% (0.0% - 0.8%)	0.1% (0.0% - 0.5%)	0.1% (0.0% - 0.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		0.0% (0.0% - 1.6%)	0.0% (0.0% - 1.1%)	0.0% (0.0% - 0.8%)	0.0% (0.0% - 0.5%)

	Multi-Pollutant Models (Total Mortality)									
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day	CO	0.0% (0.0% - 0.4%)	0.0% (0.0% - 0.3%)	0.0% (0.0% - 0.2%)	0.0% (0.0% - 0.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day	CO	0.0% (0.0% - 0.6%)	0.0% (0.0% - 0.5%)	0.0% (0.0% - 0.3%)	0.0% (0.0% - 0.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day	CO	0.0% (0.0% - 0.8%)	0.0% (0.0% - 0.6%)	0.0% (0.0% - 0.4%)	0.0% (0.0% - 0.3%)
Long-Term Exposure Mortality							**BASE CASE: Background =2.5 µg/m3**	**Hypothetical Threshold =10 µg/m3**	**Hypothetical Threshold =12.5 µg/m3**	**Hypothetical Threshold =15 µg/m3**
	Single Pollutant Models									
	Krewski et al. (2000) - ACS	All cause	30+	log-linear			4.7% (2.5% - 7.1%)	4.7% (2.5% - 7.1%)	3.9% (2.1% - 6.0%)	3.0% (1.6% - 4.6%)
	Pope et al. (2002) - ACS extended	All cause	30+	log-linear			4.1% (0.8% - 7.3%)	4.1% (0.8% - 7.3%)	3.6% (0.7% - 6.3%)	2.9% (0.6% - 5.1%)
	Multi-Pollutant Models									
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		CO	6.8% (4.0% - 9.7%)	6.8% (4.0% - 9.7%)	5.7% (3.3% - 8.1%)	4.4% (2.5% - 6.2%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		NO2	8.1% (4.3% - 11.4%)	8.1% (4.3% - 11.4%)	6.8% (3.6% - 9.6%)	5.2% (2.8% - 7.4%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		O3	6.8% (4.0% - 9.7%)	6.8% (4.0% - 9.7%)	5.7% (3.3% - 8.1%)	4.4% (2.5% - 6.2%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		SO2	1.3% (0.0% - 5.1%)	1.3% (0.0% - 5.1%)	1.0% (0.0% - 4.3%)	0.8% (0.0% - 3.2%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.30. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated Los Angeles, CA, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**									
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		500 (0 - 1060)	0.9% (0.0% - 1.9%)	990 (0 - 2070)	1.7% (0.0% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		550 (0 - 1090)	1.0% (0.0% - 1.9%)	1080 (0 - 2120)	1.9% (0.0% - 3.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		360 (0 - 880)	0.6% (0.0% - 1.5%)	700 (0 - 1720)	1.2% (0.0% - 3.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		0 (0 - 400)	0.0% (0.0% - 0.7%)	0 (0 - 780)	0.0% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		0 (0 - 430)	0.0% (0.0% - 0.8%)	0 (0 - 850)	0.0% (0.0% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0 (0 - 30)	0.0% (0.0% - 0.1%)	0 (0 - 60)	0.0% (0.0% - 0.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		500 (0 - 1060)	0.9% (0.0% - 1.9%)	990 (0 - 2070)	1.7% (0.0% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	0 day		370 (0 - 1050)	0.7% (0.0% - 1.8%)	740 (0 - 2050)	1.3% (0.0% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		300 (0 - 800)	0.5% (0.0% - 1.4%)	590 (0 - 1570)	1.0% (0.0% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		280 (0 - 950)	0.5% (0.0% - 1.7%)	550 (0 - 1850)	1.0% (0.0% - 3.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		550 (0 - 1090)	1.0% (0.0% - 1.9%)	1080 (0 - 2120)	1.9% (0.0% - 3.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	1 day		510 (0 - 1170)	0.9% (0.0% - 2.0%)	1010 (0 - 2280)	1.8% (0.0% - 4.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		90 (0 - 610)	0.2% (0.0% - 1.1%)	190 (0 - 1200)	0.3% (0.0% - 2.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		0 (0 - 900)	0.0% (0.0% - 1.6%)	0 (0 - 1750)	0.0% (0.0% - 3.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)									
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day	CO	0 (0 - 240)	0.0% (0.0% - 0.4%)	0 (0 - 470)	0.0% (0.0% - 0.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day	CO	0 (0 - 360)	0.0% (0.0% - 0.6%)	0 (0 - 710)	0.0% (0.0% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day	CO	0 (0 - 480)	0.0% (0.0% - 0.8%)	0 (0 - 930)	0.0% (0.0% - 1.6%)
Long-Term Exposure Mortality	**Single Pollutant Models**									
	Krewski et al. (2000) - ACS	All cause	30+	log-linear			2730 (1420 - 4140)	4.7% (2.5% - 7.1%)	5280 (2780 - 7910)	9.1% (4.8% - 13.6%)
	Pope et al. (2002) - ACS extended	All cause	30+	log-linear			2380 (480 - 4220)	4.1% (0.8% - 7.3%)	4620 (950 - 8040)	8.0% (1.6% - 13.9%)
	Multi-Pollutant Models									
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		CO	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)	7550 (4480 - 10580)	13.0% (7.7% - 18.2%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		NO2	4710 (2520 - 6630)	8.1% (4.3% - 11.4%)	8950 (4880 - 12390)	15.4% (8.4% - 21.3%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		O3	3950 (2310 - 5610)	6.8% (4.0% - 9.7%)	7550 (4480 - 10580)	13.0% (7.7% - 18.2%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		SO2	730 (0 - 2940)	1.3% (0.0% - 5.1%)	1430 (0 - 5680)	2.5% (0.0% - 9.8%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.31. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations
Los Angeles, CA, 2002

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence*		
					Base Case: Assuming AQ as Reported	Assuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+		4.7% (2.5% - 7.1%)	3.2% (1.6% - 4.8%)	2.4% (1.2% - 3.6%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		9.4% (6.1% - 12.8%)	6.3% (4.1% - 8.7%)	4.8% (3.1% - 6.6%)
	Pope et al. (2002) - ACS extended	All cause	30+		4.1% (0.8% - 7.3%)	2.8% (0.6% - 4.9%)	2.1% (0.4% - 3.7%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+		5.8% (1.5% - 9.9%)	3.9% (1.0% - 6.7%)	2.9% (0.8% - 5.1%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+		7.9% (1.1% - 14.2%)	5.3% (0.8% - 9.7%)	4.0% (0.6% - 7.4%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+	CO	6.8% (4.0% - 9.7%)	4.6% (2.7% - 6.6%)	3.5% (2.0% - 5.0%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	8.1% (4.3% - 11.4%)	5.5% (2.9% - 7.8%)	4.1% (2.2% - 5.9%)
	Krewski et al. (2000) - ACS	All cause	30+	O3	6.8% (4.0% - 9.7%)	4.6% (2.7% - 6.6%)	3.5% (2.0% - 5.0%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	1.3% (0.0% - 5.1%)	0.8% (0.0% - 3.4%)	0.6% (0.0% - 2.6%)

* Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.32. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Lipfert et al. (2000)	Non-accidental	all	0 day		380	2.2%	340	1.9%	290	1.7%
	Single Pollutant Models (Cause-Specific Mortality)										
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day		480 (230 - 730)	6.9% (3.3% - 10.5%)	420 (200 - 640)	6.1% (2.9% - 9.3%)	360 (170 - 560)	5.3% (2.5% - 8.1%)
	Multi-Pollutant Models (Total Mortality)										
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	270	1.6%	240	1.4%	210	1.2%
	Multi-Pollutant Models (Cause-Specific Mortality)										
	Lipfert et al. (2000) -- 7 counties	Cardiovascluar	all	0 day	O3	140	2.1%	130	1.8%	110	1.6%
Long-Term Exposure Mortality	**Single Pollutant Models**										
	Krewski et al. (2000) - ACS	All cause	30+			360 (190 - 560)	2.1% (1.1% - 3.2%)	360 (190 - 560)	2.1% (1.1% - 3.2%)	360 (190 - 560)	2.1% (1.1% - 3.2%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			340 (220 - 470)	4.2% (2.7% - 5.8%)	340 (220 - 470)	4.2% (2.7% - 5.8%)	340 (220 - 470)	4.2% (2.7% - 5.8%)
	Pope et al. (2002) - ACS extended	All cause	30+			320 (60 - 570)	1.8% (0.4% - 3.2%)	320 (60 - 570)	1.8% (0.4% - 3.2%)	320 (60 - 570)	1.8% (0.4% - 3.2%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			210 (60 - 360)	2.6% (0.7% - 4.4%)	210 (60 - 360)	2.6% (0.7% - 4.4%)	210 (60 - 360)	2.6% (0.7% - 4.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			50 (10 - 80)	3.5% (0.5% - 6.4%)	50 (10 - 80)	3.5% (0.5% - 6.4%)	50 (10 - 80)	3.5% (0.5% - 6.4%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models										
	Krewski et al. (2000) - ACS	All cause	30+		CO	530 (310 - 760)	3.0% (1.7% - 4.3%)	530 (310 - 760)	3.0% (1.7% - 4.3%)	530 (310 - 760)	3.0% (1.7% - 4.3%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	630 (340 - 900)	3.6% (1.9% - 5.1%)	630 (340 - 900)	3.6% (1.9% - 5.1%)	630 (340 - 900)	3.6% (1.9% - 5.1%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	530 (310 - 760)	3.0% (1.7% - 4.3%)	530 (310 - 760)	3.0% (1.7% - 4.3%)	530 (310 - 760)	3.0% (1.7% - 4.3%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	100 (0 - 390)	0.5% (0.0% - 2.2%)	100 (0 - 390)	0.5% (0.0% - 2.2%)	100 (0 - 390)	0.5% (0.0% - 2.2%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM2.5 non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Note 2: Multi-county short-term exposure C-R functions were applied only to counties included among those used to estimate the function.

Exhibit D.33. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using Alternative Hypothetical Threshold Models* Philadelphia, PA, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Lipfert et al. (2000)	Non-accidental	all	0 day		1.9%	1.0%	0.6%	0.4%
	Multi-Pollutant Models (Total Mortality)								
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	1.4%	0.7%	0.4%	0.3%
Long-Term Exposure Mortality						**BASE CASE: Background =3.5 µg/m3**	**Hypothetical Threshold =10 µg/m3**	**Hypothetical Threshold =12.5 µg/m3**	**Hypothetical Threshold =15 µg/m3**
	Single Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+			2.1% (1.1% - 3.2%)	2.1% (1.1% - 3.2%)	1.0% (0.5% - 1.6%)	0.0% (0.0% - 0.0%)
	Pope et al. (2002) - ACS extended	All cause	30+			1.8% (0.4% - 3.2%)	1.8% (0.4% - 3.2%)	0.9% (0.2% - 1.6%)	0.0% (0.0% - 0.0%)
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	3.0% (1.7% - 4.3%)	3.0% (1.7% - 4.3%)	1.5% (0.9% - 2.1%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	3.6% (1.9% - 5.1%)	3.6% (1.9% - 5.1%)	1.8% (0.9% - 2.5%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	3.0% (1.7% - 4.3%)	3.0% (1.7% - 4.3%)	1.5% (0.9% - 2.1%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	0.5% (0.0% - 2.2%)	0.5% (0.0% - 2.2%)	0.3% (0.0% - 1.1%)	0.0% (0.0% - 0.0%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM2.5 non-accidental mortality coefficients. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Exhibit D.34. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Lipfert et al. (2000)	Non-accidental	all	0 day		340	1.9%	660	3.9%
	Multi-Pollutant Models (Total Mortality)								
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	240	1.4%	470	2.8%

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM2.5 non-accidental mortality coefficients. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Exhibit D.35. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations
Philadelphia, PA, 2002

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence*		
					Base Case: Assuming AQ as Reported	Assuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+		2.1% (1.1% - 3.2%)	1.4% (0.7% - 2.1%)	1.0% (0.5% - 1.6%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		4.2% (2.7% - 5.8%)	2.8% (1.8% - 3.9%)	2.1% (1.4% - 2.9%)
	Pope et al. (2002) - ACS extended	All cause	30+		1.8% (0.4% - 3.2%)	1.2% (0.2% - 2.2%)	0.9% (0.2% - 1.6%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+		2.6% (0.7% - 4.4%)	1.7% (0.5% - 3.0%)	1.3% (0.3% - 2.2%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+		3.5% (0.5% - 6.4%)	2.3% (0.3% - 4.3%)	1.8% (0.3% - 3.3%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+	CO	3.0% (1.7% - 4.3%)	2.0% (1.2% - 2.9%)	1.5% (0.9% - 2.2%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	3.6% (1.9% - 5.1%)	2.4% (1.3% - 3.4%)	1.8% (1.0% - 2.6%)
	Krewski et al. (2000) - ACS	All cause	30+	O3	3.0% (1.7% - 4.3%)	2.0% (1.2% - 2.9%)	1.5% (0.9% - 2.2%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	0.5% (0.0% - 2.2%)	0.4% (0.0% - 1.5%)	0.3% (0.0% - 1.1%)

* Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.36. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
Phoenix, AZ, 2001

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Cause-Specific Mortality)**										
	Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	65+	0 day		240 (0 - 540)	3.4% (0.0% - 7.5%)	210 (0 - 450)	2.9% (0.0% - 6.3%)	170 (0 - 370)	2.3% (0.0% - 5.1%)
	Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	65+	1 day		430 (130 - 710)	5.9% (1.8% - 9.9%)	360 (110 - 600)	5.0% (1.5% - 8.3%)	290 (90 - 480)	4.1% (1.2% - 6.7%)
Long-Term Exposure Mortality	**Single Pollutant Models**										
	Krewski et al. (2000) - ACS	All cause	30+			40 (20 - 60)	0.2% (0.1% - 0.3%)	40 (20 - 60)	0.2% (0.1% - 0.3%)	40 (20 - 60)	0.2% (0.1% - 0.3%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			40 (30 - 60)	0.4% (0.2% - 0.5%)	40 (30 - 60)	0.4% (0.2% - 0.5%)	40 (30 - 60)	0.4% (0.2% - 0.5%)
	Pope et al. (2002) - ACS extended	All cause	30+			40 (10 - 60)	0.2% (0.0% - 0.3%)	40 (10 - 60)	0.2% (0.0% - 0.3%)	40 (10 - 60)	0.2% (0.0% - 0.3%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			20 (10 - 40)	0.2% (0.1% - 0.4%)	20 (10 - 40)	0.2% (0.1% - 0.4%)	20 (10 - 40)	0.2% (0.1% - 0.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			0 (0 - 10)	0.3% (0.0% - 0.6%)	0 (0 - 10)	0.3% (0.0% - 0.6%)	0 (0 - 10)	0.3% (0.0% - 0.6%)
	Multi-Pollutant Models										
	Krewski et al. (2000) - ACS	All cause	30+		CO	60 (30 - 90)	0.3% (0.2% - 0.4%)	60 (30 - 90)	0.3% (0.2% - 0.4%)	60 (30 - 90)	0.3% (0.2% - 0.4%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	70 (40 - 100)	0.3% (0.2% - 0.5%)	70 (40 - 100)	0.3% (0.2% - 0.5%)	70 (40 - 100)	0.3% (0.2% - 0.5%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	60 (30 - 90)	0.3% (0.2% - 0.4%)	60 (30 - 90)	0.3% (0.2% - 0.4%)	60 (30 - 90)	0.3% (0.2% - 0.4%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	10 (0 - 40)	0.1% (0.0% - 0.2%)	10 (0 - 40)	0.1% (0.0% - 0.2%)	10 (0 - 40)	0.1% (0.0% - 0.2%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.37. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
Phoenix, AZ, 2001

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =2.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12.5 µg/m3	Hypothetical Threshold =15 µg/m3
Long-Term Exposure Mortality	**Single Pollutant Models**								
	Krewski et al. (2000) - ACS	All cause	30+			0.2% (0.1% - 0.3%)	0.2% (0.1% - 0.3%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Pope et al. (2002) - ACS extended	All cause	30+			0.2% (0.0% - 0.3%)	0.2% (0.0% - 0.3%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	0.3% (0.2% - 0.4%)	0.3% (0.2% - 0.4%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	0.3% (0.2% - 0.5%)	0.3% (0.2% - 0.5%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	0.3% (0.2% - 0.4%)	0.3% (0.2% - 0.4%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	0.1% (0.0% - 0.2%)	0.1% (0.0% - 0.2%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM2.5 unless otherwise specified.

taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.38. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations
Phoenix, AZ, 2001

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence*		
					Base Case: Assuming AQ as Reported	Assuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+		0.2% (0.1% - 0.3%)	0.1% (0.1% - 0.2%)	0.1% (0.1% - 0.1%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		0.4% (0.2% - 0.5%)	0.3% (0.2% - 0.4%)	0.2% (0.1% - 0.3%)
	Pope et al. (2002) - ACS extended	All cause	30+		0.2% (0.0% - 0.3%)	0.1% (0.0% - 0.2%)	0.1% (0.0% - 0.2%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+		0.2% (0.1% - 0.4%)	0.2% (0.0% - 0.3%)	0.1% (0.0% - 0.2%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+		0.3% (0.0% - 0.6%)	0.2% (0.0% - 0.4%)	0.2% (0.0% - 0.3%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+	CO	0.3% (0.2% - 0.4%)	0.2% (0.1% - 0.3%)	0.1% (0.1% - 0.2%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	0.3% (0.2% - 0.5%)	0.2% (0.1% - 0.3%)	0.2% (0.1% - 0.2%)
	Krewski et al. (2000) - ACS	All cause	30+	O3	0.3% (0.2% - 0.4%)	0.2% (0.1% - 0.3%)	0.1% (0.1% - 0.2%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	0.1% (0.0% - 0.2%)	0.0% (0.0% - 0.1%)	0.0% (0.0% - 0.1%)

* Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.39. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
Pittsburgh, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Chock et al. (2000)	Non-accidental	<75	0 day		70 (0 - 180)	1.2% (0.0% - 3.3%)	60 (0 - 170)	1.2% (0.0% - 3.2%)	60 (0 - 150)	1.0% (0.0% - 2.8%)
	Chock et al. (2000)	Non-accidental	75+	0 day		60 (0 - 260)	0.7% (0.0% - 2.9%)	60 (0 - 250)	0.7% (0.0% - 2.8%)	50 (0 - 220)	0.6% (0.0% - 2.4%)
	Multi-Pollutant Models (Total Mortality)										
	Chock et al. (2000)	Non-accidental	<75	0 day	CO, O3, SO2, NO2, PM10-2.5	80 (0 - 220)	1.6% (0.0% - 4.1%)	80 (0 - 210)	1.5% (0.0% - 3.9%)	70 (0 - 180)	1.3% (0.0% - 3.4%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO, O3, SO2, NO2, PM10-2.5	40 (0 - 280)	0.5% (0.0% - 3.1%)	40 (0 - 270)	0.5% (0.0% - 3.0%)	40 (0 - 230)	0.4% (0.0% - 2.6%)
Long-Term Exposure Mortality	**Single Pollutant Models**										
	Krewski et al. (2000) - ACS	All cause	30+			350 (180 - 540)	2.4% (1.2% - 3.6%)	350 (180 - 540)	2.4% (1.2% - 3.6%)	350 (180 - 540)	2.4% (1.2% - 3.6%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			360 (240 - 500)	4.8% (3.1% - 6.6%)	360 (240 - 500)	4.8% (3.1% - 6.6%)	360 (240 - 500)	4.8% (3.1% - 6.6%)
	Pope et al. (2002) - ACS extended	All cause	30+			310 (60 - 550)	2.1% (0.4% - 3.7%)	310 (60 - 550)	2.1% (0.4% - 3.7%)	310 (60 - 550)	2.1% (0.4% - 3.7%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			220 (60 - 380)	2.9% (0.8% - 5.1%)	220 (60 - 380)	2.9% (0.8% - 5.1%)	220 (60 - 380)	2.9% (0.8% - 5.1%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			40 (10 - 80)	4.0% (0.6% - 7.3%)	40 (10 - 80)	4.0% (0.6% - 7.3%)	40 (10 - 80)	4.0% (0.6% - 7.3%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models										
	Krewski et al. (2000) - ACS	All cause	30+		CO	510 (300 - 730)	3.5% (2.0% - 4.9%)	510 (300 - 730)	3.5% (2.0% - 4.9%)	510 (300 - 730)	3.5% (2.0% - 4.9%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	610 (320 - 870)	4.1% (2.2% - 5.9%)	610 (320 - 870)	4.1% (2.2% - 5.9%)	610 (320 - 870)	4.1% (2.2% - 5.9%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	510 (300 - 730)	3.5% (2.0% - 4.9%)	510 (300 - 730)	3.5% (2.0% - 4.9%)	510 (300 - 730)	3.5% (2.0% - 4.9%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	90 (0 - 380)	0.6% (0.0% - 2.6%)	90 (0 - 380)	0.6% (0.0% - 2.6%)	90 (0 - 380)	0.6% (0.0% - 2.6%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.40. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
Pittsburgh, PA, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Chock et al. (2000)	Non-accidental	<75	0 day		1.2% (0.0% - 3.2%)	0.7% (0.0% - 1.8%)	0.4% (0.0% - 1.1%)	0.2% (0.0% - 0.6%)
	Chock et al. (2000)	Non-accidental	75+	0 day		0.7% (0.0% - 2.8%)	0.4% (0.0% - 1.6%)	0.2% (0.0% - 0.9%)	0.1% (0.0% - 0.6%)
	Multi-Pollutant Models (Total Mortality)								
	Chock et al. (2000)	Non-accidental	<75	0 day	CO, O3, SO2, NO2, PM10-2.5	1.5% (0.0% - 3.9%)	0.8% (0.0% - 2.2%)	0.5% (0.0% - 1.3%)	0.3% (0.0% - 0.8%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO, O3, SO2, NO2, PM10-2.5	0.5% (0.0% - 3.0%)	0.3% (0.0% - 1.7%)	0.2% (0.0% - 1.0%)	0.1% (0.0% - 0.6%)
Long-Term Exposure Mortality						**BASE CASE: Background =3.5 µg/m3**	**Hypothetical Threshold =10 µg/m3**	**Hypothetical Threshold =12.5 µg/m3**	**Hypothetical Threshold =15 µg/m3**
	Single Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+			2.4% (1.2% - 3.6%)	2.4% (1.2% - 3.6%)	1.4% (0.7% - 2.1%)	0.1% (0.1% - 0.2%)
	Pope et al. (2002) - ACS extended	All cause	30+			2.1% (0.4% - 3.7%)	2.1% (0.4% - 3.7%)	1.2% (0.3% - 2.2%)	0.1% (0.0% - 0.2%)

	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	3.5% (2.0% - 4.9%)	3.5% (2.0% - 4.9%)	2.0% (1.2% - 2.9%)	0.2% (0.1% - 0.2%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	4.1% (2.2% - 5.9%)	4.1% (2.2% - 5.9%)	2.4% (1.3% - 3.4%)	0.2% (0.1% - 0.3%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	3.5% (2.0% - 4.9%)	3.5% (2.0% - 4.9%)	2.0% (1.2% - 2.9%)	0.2% (0.1% - 0.2%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	0.6% (0.0% - 2.6%)	0.6% (0.0% - 2.6%)	0.4% (0.0% - 1.5%)	0.0% (0.0% - 0.1%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM2.5 unless otherwise specified.

taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.41. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Pittsburgh, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Chock et al. (2000)	Non-accidental	<75	0 day		60 (0 - 170)	1.2% (0.0% - 3.2%)	120 (0 - 330)	2.3% (0.0% - 6.2%)
	Chock et al. (2000)	Non-accidental	75+	0 day		60 (0 - 250)	0.7% (0.0% - 2.8%)	120 (0 - 490)	1.4% (0.0% - 5.4%)
	Multi-Pollutant Models (Total Mortality)								
	Chock et al. (2000)	Non-accidental	<75	0 day	CO, O3, SO2, NO2, PM10-2.5	80 (0 - 210)	1.5% (0.0% - 3.9%)	160 (0 - 410)	2.9% (0.0% - 7.5%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO, O3, SO2, NO2, PM10-2.5	40 (0 - 270)	0.5% (0.0% - 3.0%)	80 (0 - 520)	0.9% (0.0% - 5.7%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.42. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations
Pittsburgh, PA, 2002

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence*		
					Base Case: Assuming AQ as Reported	Assuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+		2.4% (1.2% - 3.6%)	1.6% (0.8% - 2.4%)	1.2% (0.6% - 1.8%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		4.8% (3.1% - 6.6%)	3.2% (2.1% - 4.5%)	2.4% (1.6% - 3.4%)
	Pope et al. (2002) - ACS extended	All cause	30+		2.1% (0.4% - 3.7%)	1.4% (0.3% - 2.5%)	1.0% (0.2% - 1.9%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+		2.9% (0.8% - 5.1%)	2.0% (0.5% - 3.4%)	1.5% (0.4% - 2.6%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+		4.0% (0.6% - 7.3%)	2.7% (0.4% - 5.0%)	2.0% (0.3% - 3.7%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+	CO	3.5% (2.0% - 4.9%)	2.3% (1.3% - 3.3%)	1.7% (1.0% - 2.5%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	4.1% (2.2% - 5.9%)	2.8% (1.5% - 4.0%)	2.1% (1.1% - 3.0%)
	Krewski et al. (2000) - ACS	All cause	30+	O3	3.5% (2.0% - 4.9%)	2.3% (1.3% - 3.3%)	1.7% (1.0% - 2.5%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	0.6% (0.0% - 2.6%)	0.4% (0.0% - 1.7%)	0.3% (0.0% - 1.3%)

* Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.43. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
San Jose, CA, 2001

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day		280 (60 - 490)	3.2% (0.7% - 5.6%)	260 (50 - 460)	3.0% (0.6% - 5.3%)	220 (50 - 400)	2.6% (0.5% - 4.5%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	1 day		0 (0 - 60)	0.0% (0.0% - 0.7%)	0 (0 - 60)	0.0% (0.0% - 0.7%)	0 (0 - 50)	0.0% (0.0% - 0.6%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Fairley (2003) [reanalysis of Fairley (1999)]	Respiratory	all	0 day		50 (0 - 140)	4.5% (0.0% - 12.1%)	50 (0 - 130)	4.2% (0.0% - 11.5%)	40 (0 - 110)	3.6% (0.0% - 9.8%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Cardiovascular	all	0 day		90 (0 - 240)	2.5% (0.0% - 6.5%)	90 (0 - 230)	2.4% (0.0% - 6.2%)	80 (0 - 200)	2.1% (0.0% - 5.3%)
	Multi-Pollutant Models (Total Mortality)										
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	lag COH	0 (0 - 270)	0.0% (0.0% - 3.1%)	0 (0 - 260)	0.0% (0.0% - 3.0%)	0 (0 - 220)	0.0% (0.0% - 2.6%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	NO3	270 (0 - 560)	3.1% (0.0% - 6.4%)	260 (0 - 530)	3.0% (0.0% - 6.1%)	220 (0 - 450)	2.5% (0.0% - 5.2%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	SO4	10 (0 - 200)	0.1% (0.0% - 2.3%)	10 (0 - 190)	0.1% (0.0% - 2.2%)	10 (0 - 160)	0.1% (0.0% - 1.9%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	NO2	0 (0 - 120)	0.0% (0.0% - 1.3%)	0 (0 - 110)	0.0% (0.0% - 1.3%)	0 (0 - 90)	0.0% (0.0% - 1.1%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	lag CO	0 (0 - 110)	0.0% (0.0% - 1.3%)	0 (0 - 110)	0.0% (0.0% - 1.2%)	0 (0 - 90)	0.0% (0.0% - 1.1%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	oz8hr	180 (0 - 480)	2.0% (0.0% - 5.6%)	170 (0 - 460)	1.9% (0.0% - 5.3%)	140 (0 - 390)	1.6% (0.0% - 4.5%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Long-Term Exposure Mortality	**Single Pollutant Models**										
	Krewski et al. (2000) - ACS	All cause	30+			100 (50 - 150)	1.1% (0.6% - 1.7%)	100 (50 - 150)	1.1% (0.6% - 1.7%)	100 (50 - 150)	1.1% (0.6% - 1.7%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			100 (70 - 150)	2.3% (1.5% - 3.1%)	100 (70 - 150)	2.3% (1.5% - 3.1%)	100 (70 - 150)	2.3% (1.5% - 3.1%)
	Pope et al. (2002) - ACS extended	All cause	30+			90 (20 - 150)	1.0% (0.2% - 1.7%)	90 (20 - 150)	1.0% (0.2% - 1.7%)	90 (20 - 150)	1.0% (0.2% - 1.7%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			60 (20 - 110)	1.4% (0.4% - 2.4%)	60 (20 - 110)	1.4% (0.4% - 2.4%)	60 (20 - 110)	1.4% (0.4% - 2.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			10 (0 - 20)	1.9% (0.3% - 3.5%)	10 (0 - 20)	1.9% (0.3% - 3.5%)	10 (0 - 20)	1.9% (0.3% - 3.5%)
	Multi-Pollutant Models										
	Krewski et al. (2000) - ACS	All cause	30+		CO	140 (80 - 210)	1.6% (0.9% - 2.3%)	140 (80 - 210)	1.6% (0.9% - 2.3%)	140 (80 - 210)	1.6% (0.9% - 2.3%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	170 (90 - 250)	1.9% (1.0% - 2.8%)	170 (90 - 250)	1.9% (1.0% - 2.8%)	170 (90 - 250)	1.9% (1.0% - 2.8%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	140 (80 - 210)	1.6% (0.9% - 2.3%)	140 (80 - 210)	1.6% (0.9% - 2.3%)	140 (80 - 210)	1.6% (0.9% - 2.3%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	30 (0 - 110)	0.3% (0.0% - 1.2%)	30 (0 - 110)	0.3% (0.0% - 1.2%)	30 (0 - 110)	0.3% (0.0% - 1.2%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.44. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
San Jose, CA, 2001

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =2.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day		3.0% (0.6% - 5.3%)	1.3% (0.3% - 2.3%)	0.8% (0.2% - 1.4%)	0.6% (0.1% - 1.0%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	1 day		0.0% (0.0% - 0.7%)	0.0% (0.0% - 0.3%)	0.0% (0.0% - 0.2%)	0.0% (0.0% - 0.1%)
	Multi-Pollutant Models (Total Mortality)								
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	lag COH	0.0% (0.0% - 3.0%)	0.0% (0.0% - 1.3%)	0.0% (0.0% - 0.8%)	0.0% (0.0% - 0.5%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	NO3	3.0% (0.0% - 6.1%)	1.3% (0.0% - 2.6%)	0.8% (0.0% - 1.5%)	0.5% (0.0% - 1.1%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	SO4	0.1% (0.0% - 2.2%)	0.0% (0.0% - 1.0%)	0.0% (0.0% - 0.6%)	0.0% (0.0% - 0.4%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	NO2	0.0% (0.0% - 1.3%)	0.0% (0.0% - 0.6%)	0.0% (0.0% - 0.3%)	0.0% (0.0% - 0.2%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	lag CO	0.0% (0.0% - 1.2%)	0.0% (0.0% - 0.5%)	0.0% (0.0% - 0.3%)	0.0% (0.0% - 0.2%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	oz8hr	1.9% (0.0% - 5.3%)	0.8% (0.0% - 2.3%)	0.5% (0.0% - 1.4%)	0.4% (0.0% - 1.0%)

						BASE CASE: Background =2.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12.5 µg/m3	Hypothetical Threshold =15 µg/m3
Long-Term Exposure Mortality									
	Single Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+			1.1% (0.6% - 1.7%)	1.1% (0.6% - 1.7%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Pope et al. (2002) - ACS extended	All cause	30+			1.0% (0.2% - 1.7%)	1.0% (0.2% - 1.7%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	1.6% (0.9% - 2.3%)	1.6% (0.9% - 2.3%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	1.9% (1.0% - 2.8%)	1.9% (1.0% - 2.8%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	1.6% (0.9% - 2.3%)	1.6% (0.9% - 2.3%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	0.3% (0.0% - 1.2%)	0.3% (0.0% - 1.2%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.45. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated San Jose, CA, 2001

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day		260 (50 - 460)	3.0% (0.6% - 5.3%)	510 (110 - 870)	5.8% (1.2% - 10.0%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	1 day		0 (0 - 60)	0.0% (0.0% - 0.7%)	0 (0 - 120)	0.0% (0.0% - 1.4%)
	Multi-Pollutant Models (Total Mortality)								
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	lag COH	0 (0 - 260)	0.0% (0.0% - 3.0%)	0 (0 - 500)	0.0% (0.0% - 5.8%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	NO3	260 (0 - 530)	3.0% (0.0% - 6.1%)	500 (0 - 990)	5.7% (0.0% - 11.4%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	SO4	10 (0 - 190)	0.1% (0.0% - 2.2%)	10 (0 - 370)	0.1% (0.0% - 4.3%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	NO2	0 (0 - 110)	0.0% (0.0% - 1.3%)	0 (0 - 220)	0.0% (0.0% - 2.5%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	lag CO	0 (0 - 110)	0.0% (0.0% - 1.2%)	0 (0 - 210)	0.0% (0.0% - 2.4%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	oz8hr	170 (0 - 460)	1.9% (0.0% - 5.3%)	330 (0 - 870)	3.7% (0.0% - 10.0%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.46. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations
San Jose, CA, 2001

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence*		
					Base Case: Assuming AQ as Reported	Assuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+		1.1% (0.6% - 1.7%)	0.7% (0.4% - 1.1%)	0.6% (0.3% - 0.9%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		2.3% (1.5% - 3.1%)	1.5% (1.0% - 2.1%)	1.1% (0.7% - 1.6%)
	Pope et al. (2002) - ACS extended	All cause	30+		1.0% (0.2% - 1.7%)	0.6% (0.1% - 1.2%)	0.5% (0.1% - 0.9%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+		1.4% (0.4% - 2.4%)	0.9% (0.2% - 1.6%)	0.7% (0.2% - 1.2%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+		1.9% (0.3% - 3.5%)	1.3% (0.2% - 2.3%)	1.0% (0.1% - 1.8%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+	CO	1.6% (0.9% - 2.3%)	1.1% (0.6% - 1.6%)	0.8% (0.5% - 1.2%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	1.9% (1.0% - 2.8%)	1.3% (0.7% - 1.9%)	1.0% (0.5% - 1.4%)
	Krewski et al. (2000) - ACS	All cause	30+	O3	1.6% (0.9% - 2.3%)	1.1% (0.6% - 1.6%)	0.8% (0.5% - 1.2%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	0.3% (0.0% - 1.2%)	0.2% (0.0% - 0.8%)	0.2% (0.0% - 0.6%)

* Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.47. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
Seattle, WA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 1		Health Effects Associated with PM2.5 Above Background: Background = 2.5		Health Effects Associated with PM2.5 Above Background: Background = 4	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models** Sheppard (2003) [reanalysis of Sheppard et al. (1999)]***	Asthma	<65	1 day		40 (10 - 60)	2.7% (0.7% - 4.0%)	40 (10 - 50)	2.2% (0.6% - 3.3%)	30 (10 - 40)	1.8% (0.5% - 2.6%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

***Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM2.5 values obtained from nephelometer measurements rather than from air quality monitors.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.48. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations, Using Different Background Levels
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		290 (110 - 480)	1.3% (0.5% - 2.1%)	260 (100 - 420)	1.1% (0.4% - 1.9%)	230 (80 - 370)	1.0% (0.4% - 1.6%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		390 (280 - 510)	1.7% (1.2% - 2.2%)	350 (250 - 450)	1.5% (1.1% - 2.0%)	300 (220 - 390)	1.3% (1.0% - 1.7%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	all	0 day		10 (0 - 50)	0.8% (0.0% - 5.1%)	10 (0 - 50)	0.7% (0.0% - 4.5%)	10 (0 - 40)	0.6% (0.0% - 3.9%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	all	0 day		50 (10 - 100)	1.6% (0.4% - 3.0%)	50 (10 - 80)	1.4% (0.3% - 2.6%)	40 (10 - 70)	1.3% (0.3% - 2.3%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	all	0 day		10 (0 - 60)	1.4% (0.0% - 5.6%)	10 (0 - 50)	1.2% (0.0% - 5.0%)	10 (0 - 50)	1.1% (0.0% - 4.3%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	COPD	all	0 day		30 (0 - 60)	2.8% (0.1% - 5.4%)	30 (0 - 50)	2.5% (0.1% - 4.8%)	20 (0 - 40)	2.2% (0.1% - 4.2%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Ischemic heart disease	all	0 day		70 (40 - 100)	2.2% (1.4% - 3.1%)	60 (40 - 90)	2.0% (1.2% - 2.7%)	60 (30 - 80)	1.7% (1.1% - 2.4%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Pneumonia	all	0 day		50 (30 - 80)	5.0% (2.4% - 7.3%)	50 (20 - 70)	4.4% (2.1% - 6.5%)	40 (20 - 60)	3.8% (1.8% - 5.6%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Long-Term Exposure Mortality	**Single Pollutant Models**										
	Krewski et al. (2000) - Six Cities	All cause	25+			1060 (360 - 1740)	4.5% (1.5% - 7.4%)	1060 (360 - 1740)	4.5% (1.5% - 7.4%)	1060 (360 - 1740)	4.5% (1.5% - 7.4%)
	Krewski et al. (2000) - ACS	All cause	30+			500 (260 - 770)	2.2% (1.1% - 3.3%)	500 (260 - 770)	2.2% (1.1% - 3.3%)	500 (260 - 770)	2.2% (1.1% - 3.3%)
	Krewski et al. (2000) - Six Cities	Cardiopulmonary	25+			670 (230 - 1080)	6.1% (2.1% - 9.8%)	670 (230 - 1080)	6.1% (2.1% - 9.8%)	670 (230 - 1080)	6.1% (2.1% - 9.8%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			530 (340 - 730)	4.3% (2.8% - 6.0%)	530 (340 - 730)	4.3% (2.8% - 6.0%)	530 (340 - 730)	4.3% (2.8% - 6.0%)
	Pope et al. (2002) - ACS extended	All cause	30+			440 (90 - 780)	1.9% (0.4% - 3.3%)	440 (90 - 780)	1.9% (0.4% - 3.3%)	440 (90 - 780)	1.9% (0.4% - 3.3%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			330 (90 - 560)	2.7% (0.7% - 4.6%)	330 (90 - 560)	2.7% (0.7% - 4.6%)	330 (90 - 560)	2.7% (0.7% - 4.6%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			60 (10 - 120)	3.6% (0.5% - 6.7%)	60 (10 - 120)	3.6% (0.5% - 6.7%)	60 (10 - 120)	3.6% (0.5% - 6.7%)
	Multi-Pollutant Models										
	Krewski et al. (2000) - ACS	All cause	30+		CO	730 (420 - 1050)	3.1% (1.8% - 4.5%)	730 (420 - 1050)	3.1% (1.8% - 4.5%)	730 (420 - 1050)	3.1% (1.8% - 4.5%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	880 (460 - 1250)	3.8% (2.0% - 5.3%)	880 (460 - 1250)	3.8% (2.0% - 5.3%)	880 (460 - 1250)	3.8% (2.0% - 5.3%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	730 (420 - 1050)	3.1% (1.8% - 4.5%)	730 (420 - 1050)	3.1% (1.8% - 4.5%)	730 (420 - 1050)	3.1% (1.8% - 4.5%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	130 (0 - 540)	0.6% (0.0% - 2.3%)	130 (0 - 540)	0.6% (0.0% - 2.3%)	130 (0 - 540)	0.6% (0.0% - 2.3%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Background: Background = 2		Health Effects Associated with PM2.5 Above Background: Background = 3.5		Health Effects Associated with PM2.5 Above Background: Background = 5	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Respiratory Symptoms* **	**Single Pollutant Models**										
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day		13000 (5000 - 19000)	22.4% (9.1% - 32.9%)	11000 (5000 - 17000)	20.2% (8.1% - 29.8%)	10000 (4000 - 15000)	17.9% (7.2% - 26.5%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day		21000 (0 - 39000)	12.6% (0.0% - 23.9%)	19000 (0 - 35000)	11.3% (0.0% - 21.5%)	16000 (0 - 31000)	10.0% (0.0% - 19.1%)
	Multi-Pollutant Models										
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day	PM10-2.5	11000 (3000 - 18000)	20.4% (5.2% - 32.6%)	10000 (3000 - 17000)	18.3% (4.7% - 29.5%)	9000 (2000 - 15000)	16.2% (4.1% - 26.2%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day	PM10-2.5	10000 (0 - 31000)	6.0% (0.0% - 18.7%)	9000 (0 - 28000)	5.4% (0.0% - 16.8%)	8000 (0 - 24000)	4.7% (0.0% - 14.9%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East (with a range from 2.0 to 5.0 ug/m3) and 2.5 ug/m3 in the West (with a range from 1.0 to 4.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.49. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
St. Louis, MO, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		1.1% (0.4% - 1.9%)	0.6% (0.2% - 1.0%)	0.3% (0.1% - 0.5%)	0.2% (0.1% - 0.2%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		1.5% (1.1% - 2.0%)	0.8% (0.5% - 1.0%)	0.4% (0.3% - 0.5%)	0.2% (0.1% - 0.2%)
Long-Term Exposure Mortality						**BASE CASE: Background =3.5 µg/m3**	**Hypothetical Threshold =10 µg/m3**	**Hypothetical Threshold =12.5 µg/m3**	**Hypothetical Threshold =15 µg/m3**
	Single Pollutant Models								
	Krewski et al. (2000) - Six Cities	All cause	25+			4.5% (1.5% - 7.4%)	4.3% (1.4% - 7.0%)	2.9% (1.0% - 4.8%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+			2.2% (1.1% - 3.3%)	2.2% (1.1% - 3.3%)	1.1% (0.6% - 1.7%)	0.0% (0.0% - 0.0%)
	Pope et al. (2002) - ACS extended	All cause	30+			1.9% (0.4% - 3.3%)	1.9% (0.4% - 3.3%)	1.0% (0.2% - 1.8%)	0.0% (0.0% - 0.0%)

	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	3.1% (1.8% - 4.5%)	3.1% (1.8% - 4.5%)	1.6% (0.9% - 2.3%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	3.8% (2.0% - 5.3%)	3.8% (2.0% - 5.3%)	1.9% (1.0% - 2.8%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	3.1% (1.8% - 4.5%)	3.1% (1.8% - 4.5%)	1.6% (0.9% - 2.3%)	0.0% (0.0% - 0.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	0.6% (0.0% - 2.3%)	0.6% (0.0% - 2.3%)	0.3% (0.0% - 1.2%)	0.0% (0.0% - 0.0%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.50. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-2.5 Above Background: Single Lag		Health Effects Associated with PM-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		260 (100 - 420)	1.1% (0.4% - 1.9%)	510 (190 - 830)	2.2% (0.8% - 3.6%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		350 (250 - 450)	1.5% (1.1% - 2.0%)	680 (490 - 870)	3.0% (2.1% - 3.8%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.51. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations
St. Louis, MO, 2002

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence*		
					Base Case: Assuming AQ as Reported	Assuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - Six Cities	All cause	25+		4.5% (1.5% - 7.4%)	3.0% (1.0% - 5.0%)	2.3% (0.8% - 3.8%)
	Krewski et al. (2000) - ACS	All cause	30+		2.2% (1.1% - 3.3%)	1.4% (0.7% - 2.2%)	1.1% (0.6% - 1.7%)
	Krewski et al. (2000) - Six Cities	Cardiopulmonary	25+		6.1% (2.1% - 9.8%)	4.1% (1.4% - 6.7%)	3.1% (1.0% - 5.0%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		4.3% (2.8% - 6.0%)	2.9% (1.9% - 4.0%)	2.2% (1.4% - 3.0%)
	Pope et al. (2002) - ACS extended	All cause	30+		1.9% (0.4% - 3.3%)	1.3% (0.3% - 2.2%)	0.9% (0.2% - 1.7%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+		2.7% (0.7% - 4.6%)	1.8% (0.5% - 3.1%)	1.3% (0.4% - 2.3%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+		3.6% (0.5% - 6.7%)	2.4% (0.3% - 4.5%)	1.8% (0.3% - 3.4%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+	CO	3.1% (1.8% - 4.5%)	2.1% (1.2% - 3.0%)	1.6% (0.9% - 2.3%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	3.8% (2.0% - 5.3%)	2.5% (1.3% - 3.6%)	1.9% (1.0% - 2.7%)
	Krewski et al. (2000) - ACS	All cause	30+	O3	3.1% (1.8% - 4.5%)	2.1% (1.2% - 3.0%)	1.6% (0.9% - 2.3%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	0.6% (0.0% - 2.3%)	0.4% (0.0% - 1.6%)	0.3% (0.0% - 1.2%)

* Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Exhibit D.52. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Boston, MA, 1999

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Non-accidental	all	0 day		510 (300 - 710)	2.3% (1.3% - 3.2%)	430 (250 - 610)	1.9% (1.1% - 2.7%)	360 (210 - 500)	1.6% (0.9% - 2.2%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Non-accidental	all	0 day		300 (170 - 420)	1.3% (0.8% - 1.9%)	250 (150 - 360)	1.1% (0.7% - 1.6%)	210 (120 - 300)	0.9% (0.5% - 1.3%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		180 (20 - 330)	0.8% (0.1% - 1.5%)	150 (10 - 280)	0.7% (0.1% - 1.3%)	120 (10 - 230)	0.6% (0.1% - 1.0%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		100 (10 - 200)	1.0% (0.1% - 1.8%)	90 (10 - 170)	0.8% (0.1% - 1.5%)	70 (10 - 140)	0.7% (0.1% - 1.3%)
	Multi-Pollutant Models (Total Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3	120 (50 - 190)	0.5% (0.2% - 0.8%)	100 (40 - 160)	0.4% (0.2% - 0.7%)	80 (40 - 130)	0.4% (0.2% - 0.6%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, NO2	90 (0 - 190)	0.4% (0.0% - 0.8%)	80 (0 - 160)	0.3% (0.0% - 0.7%)	60 (0 - 130)	0.3% (0.0% - 0.6%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, SO2	90 (10 - 180)	0.4% (0.0% - 0.8%)	80 (10 - 150)	0.3% (0.0% - 0.7%)	60 (10 - 130)	0.3% (0.0% - 0.6%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, CO	100 (20 - 180)	0.5% (0.1% - 0.8%)	90 (20 - 160)	0.4% (0.1% - 0.7%)	70 (20 - 130)	0.3% (0.1% - 0.6%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Respiratory Symptoms* **	**Single Pollutant Models**										
	Schwartz et al. (1994) -- 6 cities	Lower respiratory symptoms	7 - 14	1 day		13000 (19000 - 56000)	25.3% (36.2% - 107.8%)	12000 (16000 - 50000)	22.2% (31.6% - 95.4%)	10000 (14000 - 43000)	19.0% (26.9% - 82.2%)
	Schwartz et al. (1994) -- 6 cities	Upper respiratory symptoms	7 - 14	2 day		40000 (0 - 76000)	26.1% (0.0% - 49.9%)	35000 (0 - 66000)	22.8% (0.0% - 43.8%)	29000 (0 - 57000)	19.4% (0.0% - 37.5%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.53. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated Boston, MA, 1999

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Non-accidental	all	0 day		430 (250 - 610)	1.9% (1.1% - 2.7%)	840 (500 - 1180)	3.8% (2.2% - 5.3%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Non-accidental	all	0 day		250 (150 - 360)	1.1% (0.7% - 1.6%)	500 (290 - 710)	2.2% (1.3% - 3.2%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		150 (10 - 280)	0.7% (0.1% - 1.3%)	290 (30 - 560)	1.3% (0.1% - 2.5%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)								
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3	100 (40 - 160)	0.4% (0.2% - 0.7%)	190 (90 - 320)	0.9% (0.4% - 1.4%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, NO2	80 (0 - 160)	0.3% (0.0% - 0.7%)	150 (0 - 320)	0.7% (0.0% - 1.4%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, SO2	80 (10 - 150)	0.3% (0.0% - 0.7%)	150 (10 - 300)	0.7% (0.1% - 1.4%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- national	Non-accidental	all	1 day	O3, CO	90 (20 - 160)	0.4% (0.1% - 0.7%)	170 (40 - 310)	0.8% (0.2% - 1.4%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.54. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Chicago, IL, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**											
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	log-linear, GAM (stringent)	unconst. distr. lag		1020 (460 - 1570)	2.2% (1.0% - 3.3%)	880 (390 - 1350)	1.9% (0.8% - 2.9%)	740 (330 - 1140)	1.6% (0.7% - 2.4%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	log-linear, penalized splines	unconst. distr. lag		950 (460 - 1430)	2.0% (1.0% - 3.0%)	810 (390 - 1220)	1.7% (0.8% - 2.6%)	680 (330 - 1030)	1.4% (0.7% - 2.2%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM	1 day		140 (0 - 340)	0.3% (0.0% - 0.8%)	120 (0 - 290)	0.3% (0.0% - 0.7%)	100 (0 - 250)	0.2% (0.0% - 0.6%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		190 (40 - 330)	0.4% (0.1% - 0.8%)	160 (40 - 280)	0.4% (0.1% - 0.6%)	130 (30 - 240)	0.3% (0.1% - 0.5%)
	Styer et al. (1995)	accidental	65+	log-linear	0 day		370 (100 - 630)	1.1% (0.3% - 1.9%)	310 (90 - 540)	1.0% (0.3% - 1.6%)	260 (70 - 450)	0.8% (0.2% - 1.4%)
	Ito and Thurston (1996)	Non-accidental	all	log-linear	0 day		450 (270 - 700)	1.0% (0.6% - 1.6%)	380 (230 - 600)	0.9% (0.5% - 1.4%)	320 (200 - 510)	0.7% (0.4% - 1.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		440 (250 - 630)	1.0% (0.6% - 1.4%)	380 (220 - 540)	0.9% (0.5% - 1.2%)	320 (180 - 450)	0.7% (0.4% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		470 (290 - 640)	1.1% (0.7% - 1.5%)	400 (250 - 550)	0.9% (0.6% - 1.3%)	340 (210 - 460)	0.8% (0.5% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		470 (270 - 660)	1.1% (0.6% - 1.5%)	400 (230 - 560)	0.9% (0.5% - 1.3%)	340 (200 - 470)	0.8% (0.4% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		390 (190 - 590)	0.9% (0.4% - 1.3%)	340 (170 - 510)	0.8% (0.4% - 1.1%)	280 (140 - 430)	0.6% (0.3% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		350 (160 - 530)	0.8% (0.4% - 1.2%)	300 (140 - 460)	0.7% (0.3% - 1.0%)	250 (120 - 380)	0.6% (0.3% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		340 (140 - 540)	0.8% (0.3% - 1.2%)	290 (120 - 460)	0.7% (0.3% - 1.0%)	240 (100 - 390)	0.6% (0.2% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		440 (250 - 630)	1.0% (0.6% - 1.4%)	380 (220 - 540)	0.9% (0.5% - 1.2%)	320 (180 - 450)	0.7% (0.4% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		390 (190 - 590)	0.9% (0.4% - 1.3%)	340 (170 - 510)	0.8% (0.4% - 1.1%)	280 (140 - 430)	0.6% (0.3% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		240 (30 - 440)	0.5% (0.1% - 1.0%)	200 (30 - 380)	0.5% (0.1% - 0.9%)	170 (20 - 320)	0.4% (0.1% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		160 (0 - 360)	0.4% (0.0% - 0.8%)	130 (0 - 310)	0.3% (0.0% - 0.7%)	110 (0 - 260)	0.3% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		140 (0 - 330)	0.3% (0.0% - 0.8%)	120 (0 - 290)	0.3% (0.0% - 0.7%)	100 (0 - 240)	0.2% (0.0% - 0.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0 (0 - 70)	0.0% (0.0% - 0.2%)	0 (0 - 60)	0.0% (0.0% - 0.1%)	0 (0 - 50)	0.0% (0.0% - 0.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1		640 (510 - 780)	1.4% (1.1% - 1.6%)	550 (430 - 670)	1.2% (0.9% - 1.4%)	460 (370 - 560)	1.0% (0.8% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, natural splines	mean of lag 0 & 1		540 (380 - 680)	1.1% (0.8% - 1.4%)	460 (330 - 580)	1.0% (0.7% - 1.2%)	390 (270 - 490)	0.8% (0.6% - 1.0%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	unconst. distr. lag		1090 (830 - 1350)	2.3% (1.8% - 2.8%)	930 (710 - 1160)	2.0% (1.5% - 2.4%)	790 (600 - 970)	1.7% (1.3% - 2.1%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	log-linear, penalized splines	unconst. distr. lag		990 (730 - 1250)	2.1% (1.6% - 2.7%)	850 (630 - 1070)	1.8% (1.3% - 2.3%)	720 (530 - 900)	1.5% (1.1% - 1.9%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		180 (40 - 320)	0.4% (0.1% - 0.7%)	150 (30 - 270)	0.3% (0.1% - 0.6%)	130 (30 - 230)	0.3% (0.1% - 0.5%)
	Single Pollutant Models (Cause-Specific Mortality)											
	Ito and Thurston (1996)	Respiratory	all	log-linear	0 day		110 (30 - 190)	2.7% (0.8% - 4.5%)	100 (30 - 160)	2.3% (0.7% - 3.9%)	80 (20 - 140)	1.9% (0.6% - 3.3%)
	Ito and Thurston (1996)	Circulatory	all	log-linear	0 day		120 (0 - 260)	0.6% (0.0% - 1.4%)	100 (0 - 230)	0.5% (0.0% - 1.2%)	80 (0 - 190)	0.4% (0.0% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 30 df	0 day		100 (0 - 210)	0.7% (0.0% - 1.4%)	90 (0 - 180)	0.6% (0.0% - 1.2%)	70 (0 - 150)	0.5% (0.0% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 100 df	0 day		120 (20 - 230)	0.8% (0.1% - 1.5%)	110 (20 - 190)	0.7% (0.1% - 1.3%)	90 (10 - 160)	0.6% (0.1% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GLM, 100 df	0 day		120 (10 - 230)	0.8% (0.1% - 1.5%)	110 (10 - 200)	0.7% (0.1% - 1.3%)	90 (10 - 170)	0.6% (0.1% - 1.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 30 df	1 day		80 (0 - 190)	0.5% (0.0% - 1.3%)	70 (0 - 160)	0.4% (0.0% - 1.1%)	50 (0 - 140)	0.4% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 100 df	1 day		70 (0 - 170)	0.4% (0.0% - 1.1%)	60 (0 - 150)	0.4% (0.0% - 1.0%)	50 (0 - 120)	0.3% (0.0% - 0.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GLM, 100 df	1 day		70 (0 - 180)	0.4% (0.0% - 1.2%)	60 (0 - 150)	0.4% (0.0% - 1.0%)	50 (0 - 130)	0.3% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		20 (0 - 50)	1.0% (0.0% - 3.0%)	10 (0 - 40)	0.9% (0.0% - 2.6%)	10 (0 - 40)	0.7% (0.0% - 2.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		20 (0 - 50)	1.0% (0.0% - 2.9%)	10 (0 - 40)	0.9% (0.0% - 2.5%)	10 (0 - 30)	0.7% (0.0% - 2.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	0 day		20 (0 - 50)	1.1% (0.0% - 3.2%)	20 (0 - 50)	1.0% (0.0% - 2.8%)	10 (0 - 40)	0.8% (0.0% - 2.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		30 (0 - 70)	2.0% (0.0% - 3.9%)	30 (0 - 60)	1.7% (0.0% - 3.4%)	20 (0 - 50)	1.4% (0.0% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		30 (0 - 60)	1.8% (0.0% - 3.8%)	30 (0 - 50)	1.6% (0.0% - 3.2%)	20 (0 - 50)	1.3% (0.0% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	1 day		30 (0 - 70)	1.9% (0.0% - 4.0%)	30 (0 - 60)	1.7% (0.0% - 3.5%)	20 (0 - 50)	1.4% (0.0% - 2.9%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardio-respiratory	all	log-linear, GLM, Bayes adjusted	1 day		140 (40 - 230)	0.6% (0.2% - 1.0%)	120 (30 - 200)	0.5% (0.1% - 0.9%)	100 (30 - 160)	0.4% (0.1% - 0.7%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	log-linear, GAM (stringent)	mean of lag 0 & 1		50 (30 - 70)	3.0% (1.6% - 4.4%)	40 (20 - 60)	2.6% (1.4% - 3.8%)	40 (20 - 50)	2.2% (1.2% - 3.2%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	log-linear, penalized splines	mean of lag 0 & 1		40 (10 - 60)	2.2% (0.6% - 3.8%)	30 (10 - 50)	1.9% (0.5% - 3.3%)	30 (10 - 50)	1.6% (0.5% - 2.8%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	log-linear, GAM (stringent)	unconst. distr. lag		60 (0 - 120)	3.8% (0.2% - 7.2%)	50 (0 - 100)	3.3% (0.2% - 6.2%)	50 (0 - 90)	2.8% (0.2% - 5.2%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	log-linear, penalized splines	unconst. distr. lag		50 (0 - 110)	2.8% (0.0% - 6.6%)	40 (0 - 100)	2.4% (0.0% - 5.7%)	30 (0 - 80)	2.1% (0.0% - 4.8%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	log-linear, GAM (stringent)	mean of lag 0 & 1		80 (50 - 100)	4.2% (2.8% - 5.7%)	70 (40 - 90)	3.6% (2.4% - 4.9%)	50 (40 - 70)	3.0% (2.1% - 4.1%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	log-linear, penalized splines	mean of lag 0 & 1		50 (20 - 80)	2.8% (1.0% - 4.6%)	40 (20 - 70)	2.4% (0.9% - 3.9%)	40 (10 - 60)	2.1% (0.7% - 3.3%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	log-linear, GAM (stringent)	unconst. distr. lag		110 (60 - 160)	6.1% (3.2% - 8.8%)	90 (50 - 140)	5.2% (2.8% - 7.6%)	80 (40 - 120)	4.4% (2.3% - 6.4%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	log-linear, penalized splines	unconst. distr. lag		80 (20 - 130)	4.4% (1.2% - 7.4%)	70 (20 - 110)	3.8% (1.1% - 6.3%)	60 (20 - 100)	3.2% (0.9% - 5.3%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardio-vascular	all	log-linear, GAM (stringent)	mean of lag 0 & 1		250 (160 - 340)	1.6% (1.0% - 2.2%)	210 (130 - 290)	1.4% (0.9% - 1.9%)	180 (110 - 250)	1.2% (0.7% - 1.6%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardio-vascular	all	log-linear, penalized splines	mean of lag 0 & 1		250 (160 - 310)	1.6% (1.0% - 2.0%)	210 (130 - 270)	1.4% (0.9% - 1.8%)	180 (110 - 220)	1.2% (0.7% - 1.5%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardio-vascular	all	log-linear, GAM (stringent)	unconst. distr. lag		310 (190 - 460)	2.0% (1.2% - 3.0%)	270 (160 - 400)	1.8% (1.1% - 2.6%)	220 (140 - 330)	1.5% (0.9% - 2.2%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardio-vascular	all	log-linear, penalized splines	unconst. distr. lag		280 (130 - 430)	1.8% (0.8% - 2.8%)	240 (110 - 370)	1.6% (0.7% - 2.4%)	200 (90 - 310)	1.3% (0.6% - 2.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)											
	Ito and Thurston (1996)	Non-accidental	all	log-linear	0 day	O3	360 (90 - 620)	0.8% (0.2% - 1.4%)	310 (80 - 530)	0.7% (0.2% - 1.2%)	260 (70 - 440)	0.6% (0.2% - 1.0%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	SO2	620 (320 - 920)	1.3% (0.7% - 1.9%)	530 (280 - 790)	1.1% (0.6% - 1.7%)	450 (230 - 660)	1.0% (0.5% - 1.4%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, natural splines	mean of lag 0 & 1	SO2	540 (200 - 870)	1.1% (0.4% - 1.8%)	460 (170 - 750)	1.0% (0.4% - 1.6%)	390 (140 - 630)	0.8% (0.3% - 1.3%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	CO	860 (530 - 1190)	1.8% (1.1% - 2.5%)	740 (450 - 1020)	1.6% (1.0% - 2.2%)	620 (380 - 860)	1.3% (0.8% - 1.8%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, natural splines	mean of lag 0 & 1	CO	700 (310 - 1090)	1.5% (0.7% - 2.3%)	600 (270 - 930)	1.3% (0.6% - 2.0%)	500 (230 - 790)	1.1% (0.5% - 1.7%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	O3	670 (420 - 910)	1.4% (0.9% - 1.9%)	570 (360 - 780)	1.2% (0.8% - 1.7%)	480 (300 - 660)	1.0% (0.6% - 1.4%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, natural splines	mean of lag 0 & 1	O3	570 (310 - 850)	1.2% (0.7% - 1.8%)	490 (270 - 730)	1.0% (0.6% - 1.5%)	410 (230 - 610)	0.9% (0.5% - 1.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3	250 (110 - 400)	0.6% (0.3% - 0.9%)	210 (90 - 340)	0.5% (0.2% - 0.8%)	180 (80 - 290)	0.4% (0.2% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, NO2	190 (0 - 400)	0.4% (0.0% - 0.9%)	160 (0 - 340)	0.4% (0.0% - 0.8%)	140 (0 - 290)	0.3% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, SO2	190 (20 - 380)	0.4% (0.0% - 0.9%)	160 (20 - 330)	0.4% (0.0% - 0.7%)	140 (10 - 280)	0.3% (0.0% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, CO	220 (50 - 390)	0.5% (0.1% - 0.9%)	190 (40 - 340)	0.4% (0.1% - 0.8%)	160 (30 - 280)	0.4% (0.1% - 0.6%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**											
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardio-vascular	65+	log-linear, GAM (stringent), Bayes adjusted	mean of lag 0 & 1		1160 (820 - 1490)	2.1% (1.5% - 2.7%)	990 (700 - 1280)	1.8% (1.3% - 2.4%)	830 (590 - 1070)	1.5% (1.1% - 2.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardio-vascular	65+	log-linear, natural spline, Bayes adjusted	mean of lag 0 & 1		940 (560 - 1320)	1.7% (1.0% - 2.4%)	810 (480 - 1130)	1.5% (0.9% - 2.1%)	680 (400 - 950)	1.3% (0.7% - 1.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Cardio-vascular	65+	log-linear, penalized spline, Bayes adjusted	mean of lag 0 & 1		1170 (840 - 1490)	2.2% (1.5% - 2.8%)	1000 (720 - 1280)	1.8% (1.3% - 2.4%)	840 (600 - 1080)	1.6% (1.1% - 2.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardio-vascular	65+	log-linear, GAM (stringent), Bayes adjusted	unconst. distr. lag		1170 (680 - 1650)	2.2% (1.3% - 3.0%)	1000 (580 - 1420)	1.9% (1.1% - 2.6%)	840 (490 - 1190)	1.6% (0.9% - 2.2%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardio-vascular	65+	log-linear, natural spline, Bayes adjusted	unconst. distr. lag		1260 (730 - 1790)	2.3% (1.3% - 3.3%)	1080 (630 - 1530)	2.0% (1.2% - 2.8%)	910 (530 - 1290)	1.7% (1.0% - 2.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardio-vascular	65+	log-linear, penalized spline, Bayes adjusted	unconst. distr. lag		1200 (680 - 1710)	2.2% (1.3% - 3.1%)	1030 (590 - 1470)	1.9% (1.1% - 2.7%)	870 (490 - 1230)	1.6% (0.9% - 2.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 30 df	0 day		850 (590 - 1100)	1.6% (1.1% - 2.0%)	730 (510 - 950)	1.3% (0.9% - 1.7%)	610 (430 - 800)	1.1% (0.8% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 100 df	0 day		910 (650 - 1160)	1.7% (1.2% - 2.1%)	780 (560 - 990)	1.4% (1.0% - 1.8%)	650 (470 - 840)	1.2% (0.9% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GLM, 100 df	0 day		950 (680 - 1220)	1.8% (1.2% - 2.2%)	810 (580 - 1050)	1.5% (1.1% - 1.9%)	680 (490 - 880)	1.3% (0.9% - 1.6%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 30 df	1 day		570 (300 - 840)	1.1% (0.6% - 1.6%)	490 (260 - 720)	0.9% (0.5% - 1.3%)	410 (210 - 610)	0.8% (0.4% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 100 df	1 day		550 (280 - 820)	1.0% (0.5% - 1.5%)	470 (240 - 700)	0.9% (0.4% - 1.3%)	400 (200 - 590)	0.7% (0.4% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GLM, 100 df	1 day		570 (280 - 870)	1.1% (0.5% - 1.6%)	490 (240 - 740)	0.9% (0.4% - 1.4%)	410 (200 - 620)	0.8% (0.4% - 1.2%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	65+	log-linear, GAM (stringent), Bayes adjusted	mean of lag 0 & 1		560 (410 - 700)	4.4% (3.2% - 5.5%)	480 (350 - 600)	3.8% (2.7% - 4.8%)	400 (290 - 510)	3.2% (2.3% - 4.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	65+	log-linear, natural spline, Bayes adjusted	mean of lag 0 & 1		240 (70 - 410)	1.9% (0.6% - 3.2%)	210 (60 - 350)	1.6% (0.5% - 2.8%)	170 (50 - 300)	1.4% (0.4% - 2.3%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	Pneumonia	65+	log-linear, penalized spline, Bayes adjusted	mean of lag 0 & 1		970 (830 - 1120)	7.7% (6.5% - 8.8%)	840 (710 - 960)	6.6% (5.6% - 7.6%)	710 (600 - 810)	5.6% (4.7% - 6.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	log-linear, GAM (stringent), Bayes adjusted	unconst. distr. lag		820 (600 - 1030)	6.4% (4.7% - 8.1%)	700 (510 - 890)	5.5% (4.0% - 7.0%)	590 (430 - 750)	4.6% (3.4% - 5.9%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	log-linear, natural spline, Bayes adjusted	unconst. distr. lag		290 (20 - 550)	2.3% (0.2% - 4.3%)	250 (20 - 470)	2.0% (0.2% - 3.7%)	210 (20 - 400)	1.6% (0.1% - 3.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	log-linear, penalized spline, Bayes adjusted	unconst. distr. lag		380 (130 - 630)	3.0% (1.1% - 4.9%)	330 (110 - 540)	2.6% (0.9% - 4.2%)	280 (100 - 450)	2.2% (0.8% - 3.6%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	65+	log-linear, GAM (stringent), Bayes adjusted	mean of lag 0 & 1		180 (50 - 310)	2.8% (0.7% - 4.7%)	150 (40 - 260)	2.4% (0.6% - 4.1%)	130 (30 - 220)	2.0% (0.5% - 3.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	65+	log-linear, natural spline, Bayes adjusted	mean of lag 0 & 1		70 (0 - 210)	1.0% (0.0% - 3.3%)	60 (0 - 180)	0.9% (0.0% - 2.8%)	50 (0 - 150)	0.7% (0.0% - 2.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)]	COPD	65+	log-linear, penalized spline, Bayes adjusted	mean of lag 0 & 1		140 (0 - 290)	2.2% (0.0% - 4.5%)	120 (0 - 250)	1.9% (0.0% - 3.8%)	100 (0 - 210)	1.6% (0.0% - 3.2%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	log-linear, GAM (stringent), Bayes adjusted	unconst. distr. lag		10 (0 - 230)	0.2% (0.0% - 3.5%)	10 (0 - 190)	0.2% (0.0% - 3.0%)	10 (0 - 160)	0.2% (0.0% - 2.5%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	log-linear, natural spline, Bayes adjusted	unconst. distr. lag		0 (0 - 120)	0.0% (0.0% - 1.9%)	0 (0 - 100)	0.0% (0.0% - 1.6%)	0 (0 - 90)	0.0% (0.0% - 1.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	log-linear, penalized spline, Bayes adjusted	unconst. distr. lag		60 (0 - 280)	1.0% (0.0% - 4.4%)	50 (0 - 240)	0.8% (0.0% - 3.8%)	40 (0 - 210)	0.7% (0.0% - 3.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		320 (30 - 600)	1.3% (0.1% - 2.5%)	270 (30 - 510)	1.1% (0.1% - 2.1%)	230 (20 - 430)	1.0% (0.1% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		240 (0 - 510)	1.0% (0.0% - 2.1%)	200 (0 - 440)	0.9% (0.0% - 1.8%)	170 (0 - 370)	0.7% (0.0% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day		0 (0 - 250)	0.0% (0.0% - 1.0%)	0 (0 - 210)	0.0% (0.0% - 0.9%)	0 (0 - 180)	0.0% (0.0% - 0.7%)
	Morris and Naumova (1998)	Congestive heart failure	65+	log-linear	0 day		260 (70 - 440)	1.5% (0.4% - 2.6%)	230 (60 - 400)	1.4% (0.4% - 2.3%)	200 (50 - 330)	1.1% (0.3% - 2.0%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardio-vascular	65+	log-linear, GAM (stringent)	unconst. distr. lag		1240 (930 - 1540)	2.3% (1.7% - 2.8%)	1060 (800 - 1320)	2.0% (1.5% - 2.4%)	890 (670 - 1110)	1.6% (1.2% - 2.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardio-vascular	65+	log-linear, natural spline	unconst. distr. lag		1230 (880 - 1580)	2.3% (1.6% - 2.9%)	1050 (750 - 1360)	1.9% (1.4% - 2.5%)	890 (630 - 1140)	1.6% (1.2% - 2.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardio-vascular	65+	log-linear, penalized spline	unconst. distr. lag		1240 (920 - 1550)	2.3% (1.7% - 2.9%)	1060 (790 - 1330)	2.0% (1.5% - 2.5%)	890 (670 - 1120)	1.6% (1.2% - 2.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	log-linear, GAM (stringent)	unconst. distr. lag		420 (250 - 580)	3.3% (1.9% - 4.6%)	360 (210 - 500)	2.8% (1.7% - 3.9%)	300 (180 - 420)	2.4% (1.4% - 3.3%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	log-linear, natural spline	unconst. distr. lag		0 (0 - 170)	0.0% (0.0% - 1.4%)	0 (0 - 150)	0.0% (0.0% - 1.2%)	0 (0 - 130)	0.0% (0.0% - 1.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	log-linear, penalized spline	unconst. distr. lag		210 (40 - 380)	1.6% (0.3% - 3.0%)	180 (30 - 320)	1.4% (0.3% - 2.5%)	150 (30 - 270)	1.2% (0.2% - 2.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	log-linear, GAM (stringent)	unconst. distr. lag		320 (160 - 480)	5.0% (2.5% - 7.5%)	280 (140 - 420)	4.3% (2.1% - 6.5%)	230 (110 - 350)	3.6% (1.8% - 5.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	log-linear, natural spline	unconst. distr. lag		270 (80 - 450)	4.2% (1.3% - 7.0%)	230 (70 - 390)	3.6% (1.1% - 6.0%)	200 (60 - 330)	3.1% (0.9% - 5.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	log-linear, penalized spline	unconst. distr. lag		330 (160 - 490)	5.0% (2.4% - 7.5%)	280 (140 - 420)	4.3% (2.1% - 6.5%)	240 (110 - 350)	3.6% (1.8% - 5.5%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 100 df	0 day	CO	660 (390 - 930)	1.2% (0.7% - 1.7%)	570 (330 - 800)	1.0% (0.6% - 1.5%)	480 (280 - 670)	0.9% (0.5% - 1.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GLM, 100 df	0 day	CO	690 (400 - 990)	1.3% (0.7% - 1.8%)	600 (340 - 850)	1.1% (0.6% - 1.6%)	500 (290 - 710)	0.9% (0.5% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 100 df	1 day	CO	280 (0 - 570)	0.5% (0.0% - 1.1%)	240 (0 - 490)	0.4% (0.0% - 0.9%)	200 (0 - 410)	0.4% (0.0% - 0.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GLM, 100 df	1 day	CO	290 (0 - 610)	0.5% (0.0% - 1.1%)	250 (0 - 530)	0.5% (0.0% - 1.0%)	210 (0 - 440)	0.4% (0.0% - 0.8%)
	Morris and Naumova (1998)	Congestive heart failure	65+	log-linear	0 day	CO, SO2, NO2, O3	180 (0 - 530)	0.8% (0.0% - 2.2%)	160 (0 - 480)	0.7% (0.0% - 2.0%)	140 (0 - 400)	0.6% (0.0% - 1.7%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.55. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations Using Alternative Hypothetical Threshold Models* Chicago, IL, 2002

Health Effects**	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-10 Above Hypothetical Threshold***			
							BASE CASE: Background =8 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	Hypothetical Threshold =30 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**									
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		0.4% (0.1% - 0.6%)	0.3% (0.1% - 0.4%)	0.2% (0.0% - 0.3%)	0.1% (0.0% - 0.2%)
	Styer et al. (1995)	Non-accidental	65+	log-linear	0 day		1.0% (0.3% - 1.6%)	0.6% (0.2% - 1.1%)	0.4% (0.1% - 0.7%)	0.2% (0.1% - 0.3%)
	Ito and Thurston (1996)	Non-accidental	all	log-linear	0 day		0.9% (0.5% - 1.4%)	0.6% (0.4% - 0.9%)	0.4% (0.3% - 0.7%)	0.2% (0.1% - 0.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		0.9% (0.5% - 1.2%)	0.6% (0.3% - 0.8%)	0.4% (0.2% - 0.5%)	0.2% (0.1% - 0.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		0.8% (0.4% - 1.1%)	0.5% (0.2% - 0.7%)	0.3% (0.2% - 0.5%)	0.2% (0.1% - 0.2%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.56. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Chicago, IL, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**									
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		160 (40 - 280)	0.4% (0.1% - 0.6%)	320 (70 - 560)	0.7% (0.2% - 1.3%)
	Styer et al. (1995)	Non-accidental	65+	log-linear	0 day		310 (90 - 540)	1.0% (0.3% - 1.6%)	620 (170 - 1050)	1.9% (0.5% - 3.2%)
	Ito and Thurston (1996)	Non-accidental	all	log-linear	0 day		380 (230 - 600)	0.9% (0.5% - 1.4%)	750 (460 - 1180)	1.7% (1.0% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		380 (220 - 540)	0.9% (0.5% - 1.2%)	740 (420 - 1050)	1.7% (1.0% - 2.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		340 (170 - 510)	0.8% (0.4% - 1.1%)	660 (330 - 990)	1.5% (0.7% - 2.3%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		150 (30 - 270)	0.3% (0.1% - 0.6%)	300 (60 - 540)	0.7% (0.1% - 1.2%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)									
	Ito and Thurston (1996)	Non-accidental	all	log-linear	0 day	O3	310 (80 - 530)	0.7% (0.2% - 1.2%)	610 (150 - 1040)	1.4% (0.4% - 2.3%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	SO2	530 (280 - 790)	1.1% (0.6% - 1.7%)	1050 (550 - 1540)	2.2% (1.2% - 3.3%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	CO	740 (450 - 1020)	1.6% (1.0% - 2.2%)	1450 (890 - 1990)	3.1% (1.9% - 4.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	log-linear, GAM (stringent)	mean of lag 0 & 1	O3	570 (360 - 780)	1.2% (0.8% - 1.7%)	1130 (710 - 1520)	2.4% (1.5% - 3.2%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3	210 (90 - 340)	0.5% (0.2% - 0.8%)	420 (190 - 680)	0.9% (0.4% - 1.5%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, NO2	160 (0 - 340)	0.4% (0.0% - 0.8%)	330 (0 - 680)	0.7% (0.0% - 1.5%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, SO2	160 (20 - 330)	0.4% (0.0% - 0.7%)	330 (30 - 650)	0.7% (0.1% - 1.5%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, CO	190 (40 - 340)	0.4% (0.1% - 0.8%)	370 (80 - 660)	0.8% (0.2% - 1.5%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.57. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Los Angeles, CA, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**											
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM	1 day		260 (0 - 1070)	0.5% (0.0% - 1.9%)	250 (0 - 1000)	0.4% (0.0% - 1.8%)	230 (0 - 940)	0.4% (0.0% - 1.6%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		400 (40 - 760)	0.7% (0.1% - 1.3%)	380 (30 - 720)	0.7% (0.1% - 1.3%)	350 (30 - 670)	0.6% (0.1% - 1.2%)
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day		600 (0 - 1270)	1.1% (0.0% - 2.2%)	600 (0 - 1270)	1.1% (0.0% - 2.2%)	600 (0 - 1270)	1.1% (0.0% - 2.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		80 (0 - 770)	0.2% (0.0% - 1.4%)	80 (0 - 770)	0.2% (0.0% - 1.4%)	80 (0 - 740)	0.1% (0.0% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	0 day		0 (0 - 0)	0.0% (0.0% - 0.0%)	0 (0 - 0)	0.0% (0.0% - 0.0%)	0 (0 - 0)	0.0% (0.0% - 0.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		120 (0 - 740)	0.2% (0.0% - 1.3%)	120 (0 - 740)	0.2% (0.0% - 1.3%)	110 (0 - 710)	0.2% (0.0% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		70 (0 - 800)	0.1% (0.0% - 1.4%)	70 (0 - 800)	0.1% (0.0% - 1.4%)	70 (0 - 770)	0.1% (0.0% - 1.4%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		170 (0 - 780)	0.3% (0.0% - 1.4%)	170 (0 - 780)	0.3% (0.0% - 1.4%)	160 (0 - 750)	0.3% (0.0% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	1 day		120 (0 - 860)	0.2% (0.0% - 1.5%)	120 (0 - 860)	0.2% (0.0% - 1.5%)	110 (0 - 830)	0.2% (0.0% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		0 (0 - 570)	0.0% (0.0% - 1.0%)	0 (0 - 570)	0.0% (0.0% - 1.0%)	0 (0 - 550)	0.0% (0.0% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		0 (0 - 710)	0.0% (0.0% - 1.2%)	0 (0 - 710)	0.0% (0.0% - 1.2%)	0 (0 - 690)	0.0% (0.0% - 1.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		80 (0 - 770)	0.2% (0.0% - 1.4%)	80 (0 - 770)	0.2% (0.0% - 1.4%)	80 (0 - 740)	0.1% (0.0% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		170 (0 - 780)	0.3% (0.0% - 1.4%)	170 (0 - 780)	0.3% (0.0% - 1.4%)	160 (0 - 750)	0.3% (0.0% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		790 (170 - 1400)	1.4% (0.3% - 2.4%)	790 (170 - 1400)	1.4% (0.3% - 2.4%)	760 (170 - 1350)	1.3% (0.3% - 2.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		0 (0 - 510)	0.0% (0.0% - 0.9%)	0 (0 - 510)	0.0% (0.0% - 0.9%)	0 (0 - 490)	0.0% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		240 (0 - 850)	0.4% (0.0% - 1.5%)	240 (0 - 850)	0.4% (0.0% - 1.5%)	230 (0 - 820)	0.4% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0 (0 - 0)	0.0% (0.0% - 0.0%)	0 (0 - 0)	0.0% (0.0% - 0.0%)	0 (0 - 0)	0.0% (0.0% - 0.0%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		500 (60 - 950)	0.9% (0.1% - 1.7%)	470 (50 - 890)	0.8% (0.1% - 1.6%)	440 (50 - 830)	0.8% (0.1% - 1.5%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Single Pollutant Models (Cause-Specific Mortality)											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 30 df	0 day		200 (0 - 540)	1.0% (0.0% - 2.6%)	200 (0 - 540)	1.0% (0.0% - 2.6%)	200 (0 - 520)	0.9% (0.0% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 100 df	0 day		250 (0 - 560)	1.2% (0.0% - 2.7%)	250 (0 - 560)	1.2% (0.0% - 2.7%)	240 (0 - 540)	1.1% (0.0% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GLM, 100 df	0 day		270 (0 - 650)	1.3% (0.0% - 3.1%)	270 (0 - 650)	1.3% (0.0% - 3.1%)	260 (0 - 630)	1.3% (0.0% - 3.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 30 df	1 day		170 (0 - 520)	0.8% (0.0% - 2.5%)	170 (0 - 520)	0.8% (0.0% - 2.5%)	170 (0 - 500)	0.8% (0.0% - 2.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 100 df	1 day		120 (0 - 440)	0.6% (0.0% - 2.1%)	120 (0 - 440)	0.6% (0.0% - 2.1%)	110 (0 - 430)	0.5% (0.0% - 2.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GLM, 100 df	1 day		140 (0 - 540)	0.7% (0.0% - 2.6%)	140 (0 - 540)	0.7% (0.0% - 2.6%)	140 (0 - 530)	0.7% (0.0% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]***	Cardio-vascular	all	log-linear, GAM (stringent), 100 df	2 day		470 (150 - 780)	2.3% (0.7% - 3.8%)	470 (150 - 780)	2.3% (0.7% - 3.8%)	450 (150 - 760)	2.2% (0.7% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		30 (0 - 160)	1.0% (0.0% - 5.4%)	30 (0 - 160)	1.0% (0.0% - 5.4%)	30 (0 - 150)	0.9% (0.0% - 5.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		20 (0 - 150)	0.8% (0.0% - 5.1%)	20 (0 - 150)	0.8% (0.0% - 5.1%)	20 (0 - 140)	0.7% (0.0% - 4.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	0 day		30 (0 - 190)	1.1% (0.0% - 6.4%)	30 (0 - 190)	1.1% (0.0% - 6.4%)	30 (0 - 180)	1.1% (0.0% - 6.2%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		70 (0 - 200)	2.5% (0.0% - 6.8%)	70 (0 - 200)	2.5% (0.0% - 6.8%)	70 (0 - 190)	2.4% (0.0% - 6.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		100 (0 - 230)	3.5% (0.0% - 7.7%)	100 (0 - 230)	3.5% (0.0% - 7.7%)	100 (0 - 220)	3.4% (0.0% - 7.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	1 day		100 (0 - 260)	3.5% (0.0% - 8.7%)	100 (0 - 260)	3.5% (0.0% - 8.7%)	100 (0 - 250)	3.4% (0.0% - 8.5%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardio-respiratory	all	log-linear, GLM, Bayes adjusted	1 day		330 (20 - 620)	1.0% (0.1% - 1.9%)	310 (20 - 590)	0.9% (0.1% - 1.8%)	290 (20 - 550)	0.9% (0.1% - 1.7%)
	Multi-Pollutant Models (Total Mortality)											
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day	O3	600 (0 - 1270)	1.1% (0.0% - 2.2%)	600 (0 - 1270)	1.1% (0.0% - 2.2%)	600 (0 - 1270)	1.1% (0.0% - 2.2%)
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day	CO	480 (0 - 1050)	0.8% (0.0% - 1.8%)	480 (0 - 1050)	0.8% (0.0% - 1.8%)	480 (0 - 1050)	0.8% (0.0% - 1.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day	CO	0 (0 - 190)	0.0% (0.0% - 0.3%)	0 (0 - 190)	0.0% (0.0% - 0.3%)	0 (0 - 190)	0.0% (0.0% - 0.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3	500 (220 - 810)	0.9% (0.4% - 1.4%)	470 (210 - 760)	0.8% (0.4% - 1.3%)	440 (200 - 710)	0.8% (0.3% - 1.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, NO2	390 (0 - 810)	0.7% (0.0% - 1.4%)	370 (0 - 760)	0.6% (0.0% - 1.3%)	340 (0 - 710)	0.6% (0.0% - 1.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, SO2	390 (40 - 770)	0.7% (0.1% - 1.4%)	370 (30 - 730)	0.6% (0.1% - 1.3%)	340 (30 - 680)	0.6% (0.1% - 1.2%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, CO	440 (90 - 790)	0.8% (0.2% - 1.4%)	420 (90 - 740)	0.7% (0.2% - 1.3%)	390 (80 - 700)	0.7% (0.1% - 1.2%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Cause-Specific Mortality)											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardio-vascular	all	log-linear, GAM (stringent), 100 df	2 day	CO	310 (0 - 680)	1.5% (0.0% - 3.3%)	310 (0 - 680)	1.5% (0.0% - 3.3%)	300 (0 - 660)	1.4% (0.0% - 3.2%)
Hospital Admissions	**Single Pollutant Models**											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 30 df	0 day		1360 (490 - 2210)	2.0% (0.7% - 3.2%)	1360 (490 - 2210)	2.0% (0.7% - 3.2%)	1310 (480 - 2140)	1.9% (0.7% - 3.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 100 df	0 day		1100 (250 - 1930)	1.6% (0.4% - 2.8%)	1100 (250 - 1930)	1.6% (0.4% - 2.8%)	1060 (240 - 1870)	1.5% (0.4% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GLM, 100 df	0 day		1120 (50 - 2160)	1.6% (0.1% - 3.1%)	1120 (50 - 2160)	1.6% (0.1% - 3.1%)	1080 (50 - 2090)	1.6% (0.1% - 3.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 30 df	1 day		1040 (150 - 1910)	1.5% (0.2% - 2.8%)	1040 (150 - 1910)	1.5% (0.2% - 2.8%)	1000 (150 - 1840)	1.5% (0.2% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 100 df	1 day		980 (120 - 1820)	1.4% (0.2% - 2.6%)	980 (120 - 1820)	1.4% (0.2% - 2.6%)	940 (120 - 1760)	1.4% (0.2% - 2.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GLM, 100 df	1 day		1000 (0 - 2030)	1.4% (0.0% - 2.9%)	1000 (0 - 2030)	1.4% (0.0% - 2.9%)	960 (0 - 1960)	1.4% (0.0% - 2.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		460 (190 - 720)	3.2% (1.3% - 5.0%)	460 (190 - 720)	3.2% (1.3% - 5.0%)	440 (180 - 700)	3.1% (1.3% - 4.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		320 (80 - 550)	2.2% (0.6% - 3.8%)	320 (80 - 550)	2.2% (0.6% - 3.8%)	310 (80 - 530)	2.1% (0.6% - 3.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	0 day		300 (0 - 590)	2.1% (0.0% - 4.1%)	300 (0 - 590)	2.1% (0.0% - 4.1%)	290 (0 - 570)	2.0% (0.0% - 4.0%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		470 (200 - 730)	3.2% (1.4% - 5.0%)	470 (200 - 730)	3.2% (1.4% - 5.0%)	450 (190 - 700)	3.1% (1.3% - 4.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		370 (130 - 600)	2.5% (0.9% - 4.1%)	370 (130 - 600)	2.5% (0.9% - 4.1%)	350 (130 - 580)	2.5% (0.9% - 4.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	1 day		330 (30 - 620)	2.3% (0.2% - 4.3%)	330 (30 - 620)	2.3% (0.2% - 4.3%)	320 (30 - 600)	2.2% (0.2% - 4.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	2 day		630 (360 - 900)	4.4% (2.5% - 6.2%)	630 (360 - 900)	4.4% (2.5% - 6.2%)	610 (350 - 870)	4.2% (2.4% - 6.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day		450 (210 - 690)	3.1% (1.5% - 4.8%)	450 (210 - 690)	3.1% (1.5% - 4.8%)	440 (210 - 670)	3.0% (1.4% - 4.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	2 day		410 (100 - 710)	2.9% (0.7% - 4.9%)	410 (100 - 710)	2.9% (0.7% - 4.9%)	400 (100 - 690)	2.8% (0.7% - 4.8%)
	Linn et al. (2000) -- Calif. South Coast Air Basin****	Asthma	30+	log-linear	0 day		60 (0 - 220)	0.9% (0.0% - 3.4%)	60 (0 - 220)	0.9% (0.0% - 3.3%)	60 (0 - 200)	0.9% (0.0% - 3.1%)
	Linn et al. (2000) -- Calif. South Coast Air Basin****	Asthma	<30	log-linear	0 day		190 (0 - 380)	3.4% (0.0% - 6.9%)	180 (0 - 370)	3.3% (0.0% - 6.7%)	170 (0 - 340)	3.1% (0.0% - 6.3%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models											
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GAM (stringent), 100 df	0 day	CO	0 (0 - 490)	0.0% (0.0% - 0.7%)	0 (0 - 490)	0.0% (0.0% - 0.7%)	0 (0 - 470)	0.0% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardio-vascular	65+	log-linear, GLM, 100 df	0 day	CO	0 (0 - 810)	0.0% (0.0% - 1.2%)	0 (0 - 810)	0.0% (0.0% - 1.2%)	0 (0 - 780)	0.0% (0.0% - 1.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day	NO2	0 (0 - 340)	0.0% (0.0% - 2.4%)	0 (0 - 340)	0.0% (0.0% - 2.4%)	0 (0 - 330)	0.0% (0.0% - 2.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day	NO2	160 (0 - 480)	1.1% (0.0% - 3.3%)	160 (0 - 480)	1.1% (0.0% - 3.3%)	160 (0 - 460)	1.1% (0.0% - 3.2%)
	Nauenberg and Basu (1999)*****	Emergency and urgent asthma-related admissions	all	linear	0 day	O3	290 (30 - 540)	6.8% (0.8% - 12.7%)	290 (30 - 540)	6.8% (0.8% - 12.7%)	290 (30 - 540)	6.8% (0.8% - 12.7%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

***Health effects estimated using a 2-day lag cardiovascular mortality PM10 coefficient are provided here to compare with estimates from the multi-pollutant coefficient for the same lag, model and endpoints. The Moolgavkar (2002) reanalysis did not estimate cardiovascular mortality using a 0 or 1 day lag multi-pollutant PM10 coefficient.

****The California South Coast Air Basin represents the area included in Los Angeles, Riverside, San Bernadino and Orange Counties, excluding the mountain and desert regions of the first three counties.

*****The Nauenberg and Basu (1999) study examined the effect of PM2.5 levels on emergency and urgent asthma-related hospital admissions, excluding scheduled admissions and transfers from other facilities.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city or multi-county short-term exposure C-R functions were applied only to counties included among those used to estimate the function.

Exhibit D.58. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations Using Alternative Hypothetical Threshold Models* Los Angeles, CA, 2002

Health Effects**	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-10 Above Hypothetical Threshold***			
							BASE CASE: Background =6 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	Hypothetical Threshold =30 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**									
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day		1.1% (0.0% - 2.2%)	1.1% (0.0% - 2.2%)	0.9% (0.0% - 1.8%)	0.5% (0.0% - 1.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		0.2% (0.0% - 1.4%)	0.1% (0.0% - 1.0%)	0.1% (0.0% - 0.8%)	0.1% (0.0% - 0.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		0.3% (0.0% - 1.4%)	0.2% (0.0% - 1.1%)	0.2% (0.0% - 0.9%)	0.1% (0.0% - 0.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]****	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		1.4% (0.3% - 2.4%)	1.1% (0.2% - 1.9%)	0.9% (0.2% - 1.5%)	0.5% (0.1% - 0.8%)
	Multi-Pollutant Models (Total Mortality)									
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day	O3	1.1% (0.0% - 2.2%)	1.1% (0.0% - 2.2%)	0.9% (0.0% - 1.8%)	0.5% (0.0% - 1.0%)
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day	CO	0.8% (0.0% - 1.8%)	0.8% (0.0% - 1.8%)	0.7% (0.0% - 1.5%)	0.4% (0.0% - 0.8%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day	CO	0.0% (0.0% - 0.3%)	0.0% (0.0% - 0.3%)	0.0% (0.0% - 0.2%)	0.0% (0.0% - 0.1%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

****Health effects estimated using a 2-day lag non-accidental mortality PM10 coefficient are provided in the sensitivity analyses to compare with the estimates from the multi-pollutant coefficient for the same lag, model and endpoints. The Moolgavkar (2002) reanalysis did not estimate non-accidental mortality using a 0 or 1 day lag multi-pollutant PM10 coefficient.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.59. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Los Angeles, CA, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**									
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		380 (30 - 720)	0.7% (0.1% - 1.3%)	740 (70 - 1410)	1.3% (0.1% - 2.5%)
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day		600 (0 - 1270)	1.1% (0.0% - 2.2%)	1180 (0 - 2480)	2.1% (0.0% - 4.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		80 (0 - 770)	0.2% (0.0% - 1.4%)	170 (0 - 1510)	0.3% (0.0% - 2.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		170 (0 - 780)	0.3% (0.0% - 1.4%)	330 (0 - 1520)	0.6% (0.0% - 2.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]***	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		790 (170 - 1400)	1.4% (0.3% - 2.4%)	1550 (340 - 2720)	2.7% (0.6% - 4.8%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day		470 (50 - 890)	0.8% (0.1% - 1.6%)	930 (110 - 1740)	1.6% (0.2% - 3.1%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
							Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)									
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day	O3	600 (0 - 1270)	1.1% (0.0% - 2.2%)	1180 (0 - 2480)	2.1% (0.0% - 4.4%)
	Kinney et al. (1995)	Non-accidental	all	log-linear	0 day	CO	480 (0 - 1050)	0.8% (0.0% - 1.8%)	950 (0 - 2060)	1.7% (0.0% - 3.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day	CO	0 (0 - 190)	0.0% (0.0% - 0.3%)	0 (0 - 390)	0.0% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3	470 (210 - 760)	0.8% (0.4% - 1.3%)	920 (410 - 1500)	1.6% (0.7% - 2.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, NO2	370 (0 - 760)	0.6% (0.0% - 1.3%)	720 (0 - 1500)	1.3% (0.0% - 2.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, SO2	370 (30 - 730)	0.6% (0.1% - 1.3%)	720 (70 - 1430)	1.3% (0.1% - 2.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	log-linear, GLM, Bayes adjusted	1 day	O3, CO	420 (90 - 740)	0.7% (0.2% - 1.3%)	820 (170 - 1460)	1.4% (0.3% - 2.6%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

***Health effects estimated using a 2-day lag non-accidental mortality PM10 coefficient are provided in the sensitivity analyses to compare with the estimates from the multi-pollutant coefficient for the same lag, model and endpoints. The Moolgavkar (2002) reanalysis did not estimate non-accidental mortality using a 0 or 1 day lag multi-pollutant PM10 coefficient.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.60. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Minneapolis/St. Paul, MN, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	unconst. distr. lag		550 (260 - 820)	4.3% (2.1% - 6.4%)	480 (230 - 710)	3.7% (1.8% - 5.6%)	400 (190 - 610)	3.2% (1.5% - 4.7%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		190 (150 - 230)	1.5% (1.2% - 1.8%)	160 (130 - 200)	1.3% (1.0% - 1.5%)	140 (110 - 170)	1.1% (0.9% - 1.3%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	unconst. distr. lag		320 (240 - 400)	2.5% (1.9% - 3.1%)	280 (210 - 340)	2.2% (1.7% - 2.7%)	240 (180 - 290)	1.8% (1.4% - 2.3%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		50 (0 - 110)	0.4% (0.0% - 1.0%)	50 (0 - 100)	0.4% (0.0% - 0.8%)	40 (0 - 80)	0.3% (0.0% - 0.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		40 (0 - 120)	0.3% (0.0% - 1.0%)	30 (0 - 110)	0.3% (0.0% - 0.9%)	30 (0 - 90)	0.2% (0.0% - 0.8%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		30 (0 - 70)	0.6% (0.0% - 1.4%)	30 (0 - 60)	0.5% (0.0% - 1.2%)	20 (0 - 60)	0.4% (0.0% - 1.0%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	unconst. distr. lag		60 (40 - 100)	2.2% (1.3% - 3.3%)	60 (30 - 80)	1.9% (1.2% - 2.9%)	50 (30 - 70)	1.6% (1.0% - 2.4%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	unconst. distr. lag		30 (0 - 50)	4.1% (0.2% - 7.8%)	20 (0 - 40)	3.6% (0.2% - 6.8%)	20 (0 - 40)	3.0% (0.2% - 5.8%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	unconst. distr. lag		30 (20 - 50)	6.6% (3.5% - 9.5%)	30 (20 - 40)	5.7% (3.0% - 8.3%)	30 (10 - 40)	4.9% (2.6% - 7.0%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)										
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	180 (90 - 270)	1.4% (0.7% - 2.1%)	160 (80 - 230)	1.2% (0.6% - 1.8%)	130 (70 - 200)	1.1% (0.5% - 1.6%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	250 (150 - 350)	2.0% (1.2% - 2.7%)	220 (130 - 300)	1.7% (1.1% - 2.4%)	190 (110 - 260)	1.5% (0.9% - 2.0%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	200 (120 - 270)	1.5% (1.0% - 2.1%)	170 (110 - 230)	1.3% (0.8% - 1.8%)	140 (90 - 200)	1.1% (0.7% - 1.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	70 (30 - 120)	0.6% (0.3% - 1.0%)	60 (30 - 100)	0.5% (0.2% - 0.9%)	50 (20 - 90)	0.4% (0.2% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	60 (0 - 120)	0.5% (0.0% - 1.0%)	50 (0 - 100)	0.4% (0.0% - 0.9%)	40 (0 - 90)	0.4% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	60 (10 - 110)	0.5% (0.1% - 0.9%)	50 (0 - 100)	0.4% (0.0% - 0.8%)	40 (0 - 80)	0.4% (0.0% - 0.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	60 (10 - 120)	0.5% (0.1% - 1.0%)	60 (10 - 100)	0.5% (0.1% - 0.8%)	50 (10 - 80)	0.4% (0.1% - 0.7%)
Hospital Admissions	**Single Pollutant Models**										
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	unconst. distr. lag		140 (0 - 430)	1.2% (0.0% - 3.7%)	120 (0 - 380)	1.0% (0.0% - 3.2%)	100 (0 - 320)	0.9% (0.0% - 2.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		290 (220 - 360)	2.5% (1.9% - 3.1%)	250 (190 - 310)	2.1% (1.6% - 2.7%)	210 (160 - 260)	1.8% (1.4% - 2.3%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		110 (0 - 200)	7.6% (0.0% - 14.6%)	90 (0 - 180)	6.6% (0.0% - 12.8%)	80 (0 - 150)	5.6% (0.0% - 10.9%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		80 (40 - 110)	5.4% (2.7% - 8.1%)	70 (30 - 100)	4.7% (2.3% - 7.1%)	60 (30 - 80)	4.0% (2.0% - 6.0%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Schwartz (1994b)	COPD+	65+	0 day		160 (70 - 240)	9.6% (4.0% - 14.8%)	140 (60 - 210)	8.3% (3.5% - 12.9%)	120 (50 - 180)	7.1% (3.0% - 11.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		130 (0 - 270)	4.3% (0.0% - 8.7%)	120 (0 - 240)	3.7% (0.0% - 7.6%)	100 (0 - 200)	3.2% (0.0% - 6.5%)
	Schwartz (1994b)	Pneumonia	65+	0 day		100 (20 - 190)	3.5% (0.7% - 6.2%)	90 (20 - 160)	3.0% (0.6% - 5.4%)	80 (10 - 140)	2.6% (0.5% - 4.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		110 (70 - 150)	3.5% (2.1% - 4.9%)	100 (60 - 130)	3.1% (1.8% - 4.3%)	80 (50 - 110)	2.6% (1.6% - 3.6%)
	Multi-Pollutant Models										
	Schwartz (1994b)	Pneumonia	65+	0 day	O3	100 (10 - 190)	3.5% (0.4% - 6.2%)	90 (10 - 160)	3.0% (0.4% - 5.4%)	80 (10 - 140)	2.6% (0.3% - 4.6%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.61. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations Using Alternative Hypothetical Threshold Models* Minneapolis/St. Paul, MN, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-10 Above Hypothetical Threshold***			
						BASE CASE: Background =8 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	Hypothetical Threshold =30 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		0.4% (0.0% - 0.8%)	0.3% (0.0% - 0.6%)	0.2% (0.0% - 0.4%)	0.1% (0.0% - 0.2%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.62. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Minneapolis/St. Paul, MN, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		160 (130 - 200)	1.3% (1.0% - 1.5%)	320 (250 - 390)	2.5% (2.0% - 3.0%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		50 (0 - 100)	0.4% (0.0% - 0.8%)	90 (0 - 190)	0.8% (0.0% - 1.6%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		30 (0 - 110)	0.3% (0.0% - 0.9%)	60 (0 - 210)	0.5% (0.0% - 1.8%)
	Multi-Pollutant Models (Total Mortality)								
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	160 (80 - 230)	1.2% (0.6% - 1.8%)	310 (160 - 460)	2.4% (1.3% - 3.6%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	220 (130 - 300)	1.7% (1.1% - 2.4%)	430 (260 - 590)	3.4% (2.1% - 4.6%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	170 (110 - 230)	1.3% (0.8% - 1.8%)	340 (210 - 450)	2.6% (1.6% - 3.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	60 (30 - 100)	0.5% (0.2% - 0.9%)	120 (60 - 200)	1.0% (0.5% - 1.7%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	50 (0 - 100)	0.4% (0.0% - 0.9%)	100 (0 - 200)	0.8% (0.0% - 1.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	50 (0 - 100)	0.4% (0.0% - 0.8%)	100 (10 - 190)	0.8% (0.1% - 1.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	60 (10 - 100)	0.5% (0.1% - 0.8%)	110 (20 - 200)	0.9% (0.2% - 1.6%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.63. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Lipfert et al. (2000)	Non-accidental	all	0 day		440	2.6%	380	2.2%	320	1.8%
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		90 (0 - 170)	0.5% (0.0% - 1.0%)	70 (0 - 150)	0.4% (0.0% - 0.8%)	60 (0 - 120)	0.4% (0.0% - 0.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		140 (10 - 270)	0.8% (0.1% - 1.6%)	120 (10 - 230)	0.7% (0.1% - 1.3%)	100 (10 - 190)	0.6% (0.1% - 1.1%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day		630 (290 - 970)	9.2% (4.2% - 14.1%)	540 (250 - 830)	7.8% (3.6% - 12.0%)	450 (210 - 690)	6.5% (3.0% - 10.1%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		80 (10 - 160)	1.0% (0.1% - 1.9%)	70 (10 - 130)	0.9% (0.1% - 1.6%)	60 (10 - 110)	0.7% (0.1% - 1.3%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)										
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	300	1.8%	260	1.5%	220	1.3%
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	90 (40 - 150)	0.6% (0.2% - 0.9%)	80 (40 - 130)	0.5% (0.2% - 0.8%)	70 (30 - 110)	0.4% (0.2% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	70 (0 - 150)	0.4% (0.0% - 0.9%)	60 (0 - 130)	0.4% (0.0% - 0.8%)	50 (0 - 110)	0.3% (0.0% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	70 (10 - 150)	0.4% (0.0% - 0.9%)	60 (10 - 130)	0.4% (0.0% - 0.7%)	50 (10 - 100)	0.3% (0.0% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	80 (20 - 150)	0.5% (0.1% - 0.9%)	70 (20 - 130)	0.4% (0.1% - 0.7%)	60 (10 - 110)	0.4% (0.1% - 0.6%)
	Multi-Pollutant Models (Cause-Specific Mortality)										
	Lipfert et al. (2000) -- 7 counties	Cardiovascluar	all	0 day	O3	180	2.6%	150	2.2%	130	1.8%

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM10 non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Note 2: Multi-city or multi-county short-term exposure C-R functions were applied only to counties included among those used to estimate the function.

Exhibit D.64. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations Using Alternative Hypothetical Threshold Models* Philadelphia, PA, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-10 Above Hypothetical Threshold***			
						BASE CASE: Background =8 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	Hypothetical Threshold =30 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Lipfert et al. (2000)	Non-accidental	all	0 day		2.2%	1.5%	1.2%	0.7%
	Multi-Pollutant Models (Total Mortality)								
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	1.5%	1.1%	0.8%	0.5%

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM10 non-accidental mortality coefficients. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.65. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Lipfert et al. (2000)	Non-accidental	all	0 day		380	2.2%	750	4.3%
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		70 (0 - 150)	0.4% (0.0% - 0.8%)	150 (0 - 290)	0.9% (0.0% - 1.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		120 (10 - 230)	0.7% (0.1% - 1.3%)	240 (20 - 450)	1.4% (0.1% - 2.6%)
	Multi-Pollutant Models (Total Mortality)								
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	260	1.5%	510	3.0%
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	80 (40 - 130)	0.5% (0.2% - 0.8%)	160 (70 - 260)	0.9% (0.4% - 1.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	60 (0 - 130)	0.4% (0.0% - 0.8%)	120 (0 - 260)	0.7% (0.0% - 1.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	60 (10 - 130)	0.4% (0.0% - 0.7%)	120 (10 - 250)	0.7% (0.1% - 1.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	70 (20 - 130)	0.4% (0.1% - 0.7%)	140 (30 - 250)	0.8% (0.2% - 1.5%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM10 non-accidental mortality coefficients. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.66. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Phoenix, AZ, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 8	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		210 (0 - 420)	1.0% (0.0% - 2.0%)	200 (0 - 400)	0.9% (0.0% - 1.9%)	190 (0 - 380)	0.9% (0.0% - 1.8%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		180 (0 - 440)	0.8% (0.0% - 2.1%)	170 (0 - 420)	0.8% (0.0% - 2.0%)	160 (0 - 400)	0.8% (0.0% - 1.9%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		180 (10 - 350)	1.7% (0.1% - 3.2%)	180 (10 - 340)	1.6% (0.1% - 3.1%)	170 (10 - 320)	1.5% (0.1% - 2.9%)
	Mar et al. (2003) [reanalysis of Mar et al (2000)]	Cardiovascular	65-100	0 day		540 (120 - 980)	7.6% (1.7% - 13.7%)	530 (120 - 960)	7.4% (1.7% - 13.4%)	510 (110 - 910)	7.1% (1.6% - 12.8%)
	Mar et al. (2003) [reanalysis of Mar et al (2000)]	Cardiovascular	65-100	1 day		420 (0 - 880)	5.9% (0.0% - 12.3%)	410 (0 - 860)	5.8% (0.0% - 12.0%)	400 (0 - 820)	5.5% (0.0% - 11.5%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 8	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)										
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	250 (110 - 400)	1.2% (0.5% - 1.9%)	240 (110 - 390)	1.1% (0.5% - 1.8%)	230 (100 - 370)	1.1% (0.5% - 1.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	190 (0 - 400)	0.9% (0.0% - 1.9%)	190 (0 - 390)	0.9% (0.0% - 1.8%)	180 (0 - 370)	0.8% (0.0% - 1.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	190 (20 - 390)	0.9% (0.1% - 1.8%)	190 (20 - 370)	0.9% (0.1% - 1.7%)	180 (20 - 350)	0.8% (0.1% - 1.7%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	220 (50 - 390)	1.1% (0.2% - 1.9%)	210 (40 - 380)	1.0% (0.2% - 1.8%)	200 (40 - 360)	1.0% (0.2% - 1.7%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D. 67. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated Phoenix, AZ, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		200 (0 - 400)	0.9% (0.0% - 1.9%)	390 (0 - 780)	1.8% (0.0% - 3.7%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		170 (0 - 420)	0.8% (0.0% - 2.0%)	330 (0 - 820)	1.6% (0.0% - 3.9%)
	Multi-Pollutant Models (Total Mortality)								
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	240 (110 - 390)	1.1% (0.5% - 1.8%)	470 (210 - 750)	2.2% (1.0% - 3.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	190 (0 - 390)	0.9% (0.0% - 1.8%)	360 (0 - 750)	1.7% (0.0% - 3.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	190 (20 - 370)	0.9% (0.1% - 1.7%)	360 (40 - 720)	1.7% (0.2% - 3.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	210 (40 - 380)	1.0% (0.2% - 1.8%)	420 (90 - 740)	2.0% (0.4% - 3.5%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.68. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Pittsburgh, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	unconst. distr. lag		140 (10 - 270)	1.0% (0.1% - 1.8%)	120 (10 - 220)	0.8% (0.1% - 1.5%)	90 (10 - 170)	0.6% (0.1% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		150 (120 - 190)	1.0% (0.8% - 1.2%)	120 (100 - 150)	0.8% (0.7% - 1.0%)	100 (80 - 120)	0.6% (0.5% - 0.8%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	unconst. distr. lag		260 (200 - 320)	1.7% (1.3% - 2.1%)	210 (160 - 260)	1.4% (1.1% - 1.7%)	170 (130 - 210)	1.1% (0.8% - 1.4%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		50 (10 - 90)	0.3% (0.1% - 0.6%)	40 (10 - 70)	0.3% (0.1% - 0.5%)	30 (10 - 60)	0.2% (0.0% - 0.4%)
	Chock et al. (2000)	Non-accidental	<75	0 day		50 (0 - 90)	0.9% (0.1% - 1.6%)	40 (0 - 80)	0.8% (0.1% - 1.5%)	30 (0 - 60)	0.6% (0.0% - 1.2%)
	Chock et al. (2000)	Non-accidental	75+	0 day		50 (0 - 120)	0.6% (0.0% - 1.4%)	40 (0 - 110)	0.5% (0.0% - 1.2%)	30 (0 - 90)	0.4% (0.0% - 1.0%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		40 (10 - 80)	0.3% (0.1% - 0.5%)	40 (10 - 60)	0.2% (0.1% - 0.4%)	30 (10 - 50)	0.2% (0.0% - 0.3%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		30 (10 - 60)	0.5% (0.1% - 0.8%)	30 (10 - 50)	0.4% (0.1% - 0.6%)	20 (10 - 40)	0.3% (0.1% - 0.5%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardiovascular	all	unconst. distr. lag		80 (50 - 120)	1.5% (0.9% - 2.3%)	60 (40 - 100)	1.2% (0.8% - 1.9%)	50 (30 - 80)	1.0% (0.6% - 1.5%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	unconst. distr. lag		20 (0 - 30)	2.9% (0.2% - 5.4%)	10 (0 - 30)	2.3% (0.1% - 4.4%)	10 (0 - 20)	1.8% (0.1% - 3.5%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	unconst. distr. lag		20 (10 - 30)	4.6% (2.4% - 6.6%)	20 (10 - 30)	3.7% (2.0% - 5.4%)	10 (10 - 20)	2.9% (1.5% - 4.2%)
	Multi-Pollutant Models (Total Mortality)										
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	150 (80 - 220)	1.0% (0.5% - 1.5%)	120 (60 - 180)	0.8% (0.4% - 1.2%)	90 (50 - 140)	0.6% (0.3% - 0.9%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	210 (130 - 290)	1.4% (0.8% - 1.9%)	170 (100 - 230)	1.1% (0.7% - 1.5%)	130 (80 - 180)	0.9% (0.5% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	160 (100 - 220)	1.1% (0.7% - 1.4%)	130 (80 - 180)	0.9% (0.5% - 1.2%)	100 (60 - 140)	0.7% (0.4% - 0.9%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	60 (30 - 100)	0.4% (0.2% - 0.7%)	50 (20 - 80)	0.3% (0.2% - 0.6%)	40 (20 - 60)	0.3% (0.1% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	50 (0 - 100)	0.3% (0.0% - 0.7%)	40 (0 - 80)	0.3% (0.0% - 0.6%)	30 (0 - 60)	0.2% (0.0% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	50 (0 - 90)	0.3% (0.0% - 0.7%)	40 (0 - 80)	0.3% (0.0% - 0.5%)	30 (0 - 60)	0.2% (0.0% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	50 (10 - 100)	0.4% (0.1% - 0.7%)	40 (10 - 80)	0.3% (0.1% - 0.5%)	30 (10 - 60)	0.2% (0.1% - 0.4%)
	Chock et al. (2000)	Non-accidental	<75	0 day	NO2	60 (10 - 120)	1.2% (0.2% - 2.2%)	60 (10 - 100)	1.1% (0.2% - 1.9%)	40 (10 - 80)	0.8% (0.1% - 1.5%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO	90 (0 - 180)	1.0% (0.0% - 2.0%)	80 (0 - 160)	0.9% (0.0% - 1.8%)	60 (0 - 120)	0.7% (0.0% - 1.4%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models**										
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	unconst. distr. lag		370 (210 - 520)	1.8% (1.0% - 2.5%)	300 (170 - 430)	1.4% (0.8% - 2.0%)	230 (140 - 330)	1.1% (0.7% - 1.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		360 (270 - 450)	1.7% (1.3% - 2.1%)	290 (220 - 360)	1.4% (1.0% - 1.7%)	230 (170 - 280)	1.1% (0.8% - 1.4%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		130 (50 - 200)	3.7% (1.5% - 5.7%)	100 (40 - 160)	3.0% (1.2% - 4.7%)	80 (30 - 130)	2.3% (1.0% - 3.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		130 (60 - 200)	3.8% (1.8% - 5.7%)	110 (50 - 160)	3.1% (1.5% - 4.6%)	80 (40 - 130)	2.4% (1.2% - 3.6%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		80 (0 - 150)	1.7% (0.1% - 3.3%)	60 (0 - 120)	1.4% (0.1% - 2.7%)	50 (0 - 100)	1.1% (0.1% - 2.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		110 (70 - 150)	2.5% (1.5% - 3.4%)	90 (50 - 130)	2.0% (1.2% - 2.8%)	70 (40 - 100)	1.6% (0.9% - 2.2%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.69. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations Using Alternative Hypothetical Threshold Models*
Pittsburgh, PA, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-10 Above Hypothetical Threshold***			
						BASE CASE: Background =8 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	Hypothetical Threshold =30 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		0.3% (0.1% - 0.5%)	0.2% (0.0% - 0.3%)	0.1% (0.0% - 0.2%)	0.0% (0.0% - 0.1%)
	Chock et al. (2000)	Non-accidental	<75	0 day		0.8% (0.1% - 1.5%)	0.4% (0.0% - 0.8%)	0.3% (0.0% - 0.5%)	0.1% (0.0% - 0.2%)
	Chock et al. (2000)	Non-accidental	75+	0 day		0.5% (0.0% - 1.2%)	0.3% (0.0% - 0.7%)	0.2% (0.0% - 0.4%)	0.1% (0.0% - 0.2%)
	Chock et al. (2000)	Non-accidental	<75	0 day	NO2	1.1% (0.2% - 1.9%)	0.6% (0.1% - 1.1%)	0.4% (0.1% - 0.7%)	0.2% (0.0% - 0.3%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO	0.9% (0.0% - 1.8%)	0.3% (0.0% - 0.6%)	0.1% (0.0% - 0.3%)	0.0% (0.0% - 0.0%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.70. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated Pittsburgh, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		120 (100 - 150)	0.8% (0.7% - 1.0%)	240 (190 - 300)	1.6% (1.3% - 2.0%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		40 (10 - 70)	0.3% (0.1% - 0.5%)	80 (10 - 140)	0.5% (0.1% - 1.0%)
	Chock et al. (2000)	Non-accidental	<75	0 day		40 (0 - 80)	0.8% (0.1% - 1.5%)	80 (10 - 150)	1.5% (0.1% - 2.9%)
	Chock et al. (2000)	Non-accidental	75+	0 day		40 (0 - 110)	0.5% (0.0% - 1.2%)	90 (0 - 210)	1.0% (0.0% - 2.4%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		40 (10 - 60)	0.2% (0.1% - 0.4%)	70 (10 - 120)	0.5% (0.1% - 0.9%)
	Multi-Pollutant Models (Total Mortality)								
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	120 (60 - 180)	0.8% (0.4% - 1.2%)	240 (120 - 350)	1.6% (0.8% - 2.3%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	170 (100 - 230)	1.1% (0.7% - 1.5%)	330 (200 - 450)	2.2% (1.3% - 3.0%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	130 (80 - 180)	0.9% (0.5% - 1.2%)	260 (160 - 350)	1.7% (1.1% - 2.3%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	50 (20 - 80)	0.3% (0.2% - 0.6%)	100 (40 - 160)	0.7% (0.3% - 1.1%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	40 (0 - 80)	0.3% (0.0% - 0.6%)	80 (0 - 160)	0.5% (0.0% - 1.1%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	40 (0 - 80)	0.3% (0.0% - 0.5%)	80 (10 - 150)	0.5% (0.1% - 1.0%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	40 (10 - 80)	0.3% (0.1% - 0.5%)	90 (20 - 150)	0.6% (0.1% - 1.1%)
	Chock et al. (2000)	Non-accidental	<75	0 day	NO2	60 (10 - 100)	1.1% (0.2% - 1.9%)	110 (20 - 200)	2.1% (0.4% - 3.8%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO	80 (0 - 160)	0.9% (0.0% - 1.8%)	160 (10 - 310)	1.8% (0.1% - 3.4%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.71. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Provo, UT, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Multi-Pollutant Models (Total Mortality)**										
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	10 (0 - 20)	0.7% (0.3% - 1.1%)	10 (0 - 10)	0.6% (0.3% - 1.0%)	10 (0 - 10)	0.6% (0.3% - 0.9%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	10 (0 - 20)	0.5% (0.0% - 1.1%)	10 (0 - 10)	0.5% (0.0% - 1.0%)	10 (0 - 10)	0.4% (0.0% - 0.9%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	10 (0 - 20)	0.5% (0.1% - 1.0%)	10 (0 - 10)	0.5% (0.1% - 0.9%)	10 (0 - 10)	0.4% (0.0% - 0.9%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	10 (0 - 20)	0.6% (0.1% - 1.1%)	10 (0 - 10)	0.5% (0.1% - 1.0%)	10 (0 - 10)	0.5% (0.1% - 0.9%)
Hospital Admissions	**Single Pollutant Models**										
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardiovascular	65+	unconst. distr. lag		20 (0 - 80)	1.7% (0.0% - 5.7%)	20 (0 - 70)	1.6% (0.0% - 5.2%)	20 (0 - 70)	1.4% (0.0% - 4.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardiovascular	65+	unconst. distr. lag		40 (30 - 50)	2.7% (2.0% - 3.3%)	40 (30 - 40)	2.5% (1.9% - 3.1%)	30 (20 - 40)	2.3% (1.7% - 2.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		0 (0 - 0)	0.0% (0.0% - 0.0%)	0 (0 - 0)	0.0% (0.0% - 0.0%)	0 (0 - 0)	0.0% (0.0% - 0.0%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		0 (0 - 10)	5.9% (2.9% - 8.8%)	0 (0 - 10)	5.4% (2.7% - 8.1%)	0 (0 - 10)	5.0% (2.4% - 7.4%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		30 (0 - 50)	7.5% (1.1% - 13.3%)	30 (0 - 50)	6.9% (1.0% - 12.3%)	30 (0 - 50)	6.3% (0.9% - 11.3%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		20 (10 - 20)	3.9% (2.3% - 5.4%)	10 (10 - 20)	3.5% (2.1% - 4.9%)	10 (10 - 20)	3.2% (1.9% - 4.5%)
Respiratory Symptoms* **	**Single Pollutant Models**										
	Pope et al. (1991)	Upper respiratory disease	9 -11	0 day		39000 (7000 - 69000)	5.5% (1.0% - 9.8%)	39000 (7000 - 69000)	5.5% (1.0% - 9.8%)	39000 (7000 - 69000)	5.5% (1.0% - 9.8%)
	Pope et al. (1991)	Lower respiratory disease	9 -11	0 day		31000 (7000 - 52000)	9.4% (2.1% - 15.9%)	31000 (7000 - 52000)	9.4% (2.1% - 15.9%)	31000 (7000 - 52000)	9.4% (2.1% - 15.9%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for upper and lower respiratory symptoms were calculated for a 3.5 month period starting on December 1rst.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.72. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated Provo, UT, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Multi-Pollutant Models (Total Mortality)**								
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	10 (0 - 10)	0.6% (0.3% - 1.0%)	20 (10 - 30)	1.2% (0.5% - 1.9%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	10 (0 - 10)	0.5% (0.0% - 1.0%)	10 (0 - 30)	0.9% (0.0% - 1.9%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	10 (0 - 10)	0.5% (0.1% - 0.9%)	10 (0 - 30)	0.9% (0.1% - 1.9%)
	Dominici et al. (2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	10 (0 - 10)	0.5% (0.1% - 1.0%)	20 (0 - 30)	1.1% (0.2% - 1.9%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.73. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
San Jose, CA, 1999

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		40 (0 - 70)	0.4% (0.0% - 0.8%)	30 (0 - 60)	0.4% (0.0% - 0.7%)	30 (0 - 50)	0.3% (0.0% - 0.6%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day		240 (90 - 390)	2.7% (1.0% - 4.4%)	210 (80 - 350)	2.5% (0.9% - 4.0%)	180 (70 - 290)	2.0% (0.8% - 3.3%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	1 day		0 (0 - 110)	0.0% (0.0% - 1.3%)	0 (0 - 100)	0.0% (0.0% - 1.1%)	0 (0 - 80)	0.0% (0.0% - 0.9%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		20 (0 - 40)	0.4% (0.0% - 0.8%)	10 (0 - 30)	0.3% (0.0% - 0.7%)	10 (0 - 30)	0.3% (0.0% - 0.6%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		30 (0 - 50)	0.6% (0.0% - 1.1%)	20 (0 - 40)	0.5% (0.0% - 0.9%)	20 (0 - 40)	0.4% (0.0% - 0.8%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Respiratory	all	0 day		40 (0 - 100)	3.7% (0.0% - 8.4%)	40 (0 - 90)	3.3% (0.0% - 7.5%)	30 (0 - 70)	2.7% (0.0% - 6.2%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Cardiovascular	all	0 day		110 (10 - 210)	3.0% (0.2% - 5.6%)	100 (10 - 190)	2.7% (0.2% - 5.0%)	80 (10 - 150)	2.2% (0.2% - 4.2%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)										
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	50 (20 - 70)	0.5% (0.2% - 0.9%)	40 (20 - 60)	0.5% (0.2% - 0.7%)	30 (10 - 50)	0.4% (0.2% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	40 (0 - 70)	0.4% (0.0% - 0.9%)	30 (0 - 60)	0.4% (0.0% - 0.7%)	30 (0 - 50)	0.3% (0.0% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	40 (0 - 70)	0.4% (0.0% - 0.8%)	30 (0 - 60)	0.4% (0.0% - 0.7%)	30 (0 - 50)	0.3% (0.0% - 0.6%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	40 (10 - 70)	0.5% (0.1% - 0.8%)	30 (10 - 60)	0.4% (0.1% - 0.7%)	30 (10 - 50)	0.3% (0.1% - 0.6%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.74. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations Using Alternative Hypothetical Threshold Models*
San Jose, CA, 1999

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-10 Above Hypothetical Threshold***			
						BASE CASE: Background =8 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	Hypothetical Threshold =30 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day		2.5% (0.9% - 4.0%)	1.6% (0.6% - 2.6%)	1.1% (0.4% - 1.8%)	0.6% (0.2% - 0.9%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	1 day		0.0% (0.0% - 1.1%)	0.0% (0.0% - 0.7%)	0.0% (0.0% - 0.5%)	0.0% (0.0% - 0.3%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.75. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated San Jose, CA, 1999

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		30 (0 - 60)	0.4% (0.0% - 0.7%)	60 (0 - 120)	0.7% (0.0% - 1.4%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day		210 (80 - 350)	2.5% (0.9% - 4.0%)	410 (160 - 660)	4.8% (1.8% - 7.6%)
	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	1 day		0 (0 - 100)	0.0% (0.0% - 1.1%)	0 (0 - 190)	0.0% (0.0% - 2.2%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		10 (0 - 30)	0.3% (0.0% - 0.7%)	30 (0 - 60)	0.6% (0.0% - 1.3%)
	Multi-Pollutant Models (Total Mortality)								
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	40 (20 - 60)	0.5% (0.2% - 0.7%)	80 (30 - 120)	0.9% (0.4% - 1.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	30 (0 - 60)	0.4% (0.0% - 0.7%)	60 (0 - 120)	0.7% (0.0% - 1.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	30 (0 - 60)	0.4% (0.0% - 0.7%)	60 (10 - 120)	0.7% (0.1% - 1.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	30 (10 - 60)	0.4% (0.1% - 0.7%)	70 (10 - 120)	0.8% (0.2% - 1.4%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.76. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
Seattle, WA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- distr. lag model	All cause	all	unconst. distr. lag		240 (130 - 350)	2.0% (1.1% - 2.9%)	210 (110 - 300)	1.7% (0.9% - 2.5%)	170 (90 - 250)	1.4% (0.8% - 2.1%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		110 (90 - 130)	0.9% (0.7% - 1.1%)	90 (70 - 110)	0.8% (0.6% - 1.0%)	80 (60 - 100)	0.7% (0.5% - 0.8%)
	Schwartz (2003a) [reanalysis of Schwartz (2000b)] -- 10 cities	All cause	all	unconst. distr. lag		190 (140 - 230)	1.6% (1.2% - 1.9%)	160 (120 - 200)	1.3% (1.0% - 1.7%)	140 (100 - 170)	1.1% (0.9% - 1.4%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		40 (0 - 70)	0.3% (0.0% - 0.6%)	30 (0 - 60)	0.3% (0.0% - 0.5%)	30 (0 - 50)	0.2% (0.0% - 0.4%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		30 (0 - 60)	0.3% (0.0% - 0.6%)	20 (0 - 50)	0.2% (0.0% - 0.5%)	20 (0 - 50)	0.2% (0.0% - 0.4%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardio-respiratory	all	1 day		20 (0 - 40)	0.4% (0.0% - 0.8%)	20 (0 - 40)	0.4% (0.0% - 0.7%)	20 (0 - 30)	0.3% (0.0% - 0.6%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Cardio-vascular	all	unconst. distr. lag		40 (30 - 70)	1.4% (0.8% - 2.1%)	40 (20 - 60)	1.2% (0.7% - 1.8%)	30 (20 - 50)	1.0% (0.6% - 1.5%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	COPD+	all	unconst. distr. lag		20 (0 - 30)	2.6% (0.1% - 4.9%)	10 (0 - 20)	2.2% (0.1% - 4.2%)	10 (0 - 20)	1.9% (0.1% - 3.5%)
	Schwartz (2003a) [reanalysis of Braga et al. (2001)] -- 10 cities	Pneumonia	all	unconst. distr. lag		20 (10 - 30)	4.1% (2.2% - 6.0%)	20 (10 - 30)	3.6% (1.9% - 5.2%)	20 (10 - 20)	3.0% (1.6% - 4.3%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models (Total Mortality)										
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	110 (60 - 160)	0.9% (0.5% - 1.3%)	90 (50 - 140)	0.8% (0.4% - 1.1%)	80 (40 - 110)	0.6% (0.3% - 0.9%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	150 (90 - 210)	1.2% (0.8% - 1.7%)	130 (80 - 180)	1.1% (0.6% - 1.5%)	110 (70 - 150)	0.9% (0.5% - 1.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	120 (70 - 160)	1.0% (0.6% - 1.3%)	100 (60 - 130)	0.8% (0.5% - 1.1%)	80 (50 - 110)	0.7% (0.4% - 0.9%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	40 (20 - 70)	0.4% (0.2% - 0.6%)	40 (20 - 60)	0.3% (0.1% - 0.5%)	30 (10 - 50)	0.3% (0.1% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	30 (0 - 70)	0.3% (0.0% - 0.6%)	30 (0 - 60)	0.3% (0.0% - 0.5%)	20 (0 - 50)	0.2% (0.0% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	30 (0 - 70)	0.3% (0.0% - 0.6%)	30 (0 - 60)	0.3% (0.0% - 0.5%)	20 (0 - 50)	0.2% (0.0% - 0.4%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	40 (10 - 70)	0.3% (0.1% - 0.6%)	30 (10 - 60)	0.3% (0.1% - 0.5%)	30 (10 - 50)	0.2% (0.1% - 0.4%)
Hospital Admissions	**Single Pollutant Models**										
	Sheppard (2003) [reanalysis of Sheppard et al. (1999)]***	Asthma	<65	1 day		50 (10 - 80)	2.8% (0.7% - 4.8%)	40 (10 - 70)	2.4% (0.6% - 4.1%)	30 (10 - 60)	2.0% (0.5% - 3.5%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Cardio-vascular	65+	unconst. distr. lag		180 (90 - 270)	2.0% (1.0% - 2.9%)	160 (80 - 240)	1.7% (0.8% - 2.5%)	130 (70 - 200)	1.4% (0.7% - 2.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Cardio-vascular	65+	unconst. distr. lag		150 (110 - 180)	1.5% (1.2% - 1.9%)	120 (90 - 160)	1.3% (1.0% - 1.6%)	100 (80 - 130)	1.1% (0.8% - 1.4%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 4		Health Effects Associated with PM10 Above Background: Background = 6		Health Effects Associated with PM10 Above Background: Background = 8	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	COPD	65+	unconst. distr. lag		80 (50 - 110)	7.1% (4.3% - 9.8%)	70 (40 - 90)	6.1% (3.7% - 8.4%)	60 (30 - 80)	5.1% (3.1% - 7.1%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	COPD	65+	unconst. distr. lag		40 (20 - 60)	3.4% (1.7% - 5.1%)	30 (20 - 50)	2.9% (1.4% - 4.4%)	30 (10 - 40)	2.5% (1.2% - 3.7%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- distr. lag model	Pneumonia	65+	unconst. distr. lag		50 (0 - 100)	2.0% (0.1% - 3.8%)	50 (0 - 90)	1.7% (0.1% - 3.3%)	40 (0 - 70)	1.5% (0.1% - 2.8%)
	Zanobetti and Schwartz (2003) [reanalysis of Samet et al. (2000)] -- 14 cities	Pneumonia	65+	unconst. distr. lag		60 (30 - 80)	2.2% (1.3% - 3.1%)	50 (30 - 70)	1.9% (1.1% - 2.7%)	40 (20 - 60)	1.6% (0.9% - 2.2%)
Respiratory Symptoms****	**Single Pollutant Models** Yu et al. (2000)	Asthma	5-12	1 day		108000 (33000 - 168000)	4.0% (1.2% - 6.3%)	108000 (33000 - 168000)	4.0% (1.2% - 6.3%)	104000 (32000 - 163000)	3.9% (1.2% - 6.1%)
	Multi-Pollutant Models Yu et al. (2000)	Asthma	5-12	1 day	CO, SO2	55000 (0 - 168000)	2.0% (0.0% - 6.3%)	55000 (0 - 168000)	2.0% (0.0% - 6.3%)	53000 (0 - 163000)	2.0% (0.0% - 6.1%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM2.5 values obtained from nephelometer measurements rather than from air quality monitors.

****The C-R functions for asthma symptoms were calculated for children 7 to 12 with a history of chronic mild to moderate asthma.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.77. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations Using Alternative Hypothetical Threshold Models*
Seattle, WA, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM-10 Above Hypothetical Threshold***			
						BASE CASE: Background =6 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	Hypothetical Threshold =30 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		0.3% (0.0% - 0.5%)	0.1% (0.0% - 0.2%)	0.1% (0.0% - 0.2%)	0.0% (0.0% - 0.1%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.
**Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.
Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.
Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.78. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated Seattle, WA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1		90 (70 - 110)	0.8% (0.6% - 1.0%)	190 (150 - 220)	1.5% (1.2% - 1.9%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)]	Non-accidental	all	1 day		30 (0 - 60)	0.3% (0.0% - 0.5%)	60 (10 - 120)	0.5% (0.1% - 1.0%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		20 (0 - 50)	0.2% (0.0% - 0.5%)	50 (0 - 110)	0.4% (0.0% - 1.0%)
	Multi-Pollutant Models (Total Mortality)								
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	SO2	90 (50 - 140)	0.8% (0.4% - 1.1%)	180 (90 - 270)	1.5% (0.8% - 2.2%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	CO	130 (80 - 180)	1.1% (0.6% - 1.5%)	250 (150 - 340)	2.1% (1.3% - 2.9%)
	Schwartz (2003a) [reanalysis of Schwartz (2000a)] -- 10 cities	All cause	all	mean of lag 0 & 1	O3	100 (60 - 130)	0.8% (0.5% - 1.1%)	190 (120 - 260)	1.6% (1.0% - 2.2%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	40 (20 - 60)	0.3% (0.1% - 0.5%)	70 (30 - 120)	0.6% (0.3% - 1.0%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	30 (0 - 60)	0.3% (0.0% - 0.5%)	60 (0 - 120)	0.5% (0.0% - 1.0%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	30 (0 - 60)	0.3% (0.0% - 0.5%)	60 (10 - 110)	0.5% (0.1% - 1.0%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	30 (10 - 60)	0.3% (0.1% - 0.5%)	60 (10 - 110)	0.6% (0.1% - 1.0%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.79. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" PM_{10} Concentrations, Using Different Background Levels
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Non-accidental	all	0 day		160 (0 - 320)	0.7% (0.0% - 1.4%)	140 (0 - 270)	0.6% (0.0% - 1.2%)	110 (0 - 220)	0.5% (0.0% - 1.0%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Non-accidental	all	0 day		280 (160 - 400)	1.2% (0.7% - 1.8%)	240 (140 - 340)	1.0% (0.6% - 1.5%)	190 (110 - 270)	0.8% (0.5% - 1.2%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		80 (20 - 140)	0.4% (0.1% - 0.6%)	70 (10 - 120)	0.3% (0.1% - 0.5%)	50 (10 - 90)	0.2% (0.1% - 0.4%)
	Single Pollutant Models (Cause-Specific Mortality)										
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Cardiorespiratory	all	1 day		60 (20 - 110)	0.5% (0.1% - 0.9%)	50 (10 - 90)	0.4% (0.1% - 0.7%)	40 (10 - 70)	0.3% (0.1% - 0.6%)
	Multi-Pollutant Models (Total Mortality)										
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	110 (50 - 180)	0.5% (0.2% - 0.8%)	90 (40 - 150)	0.4% (0.2% - 0.7%)	70 (30 - 120)	0.3% (0.1% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	90 (0 - 180)	0.4% (0.0% - 0.8%)	70 (0 - 150)	0.3% (0.0% - 0.7%)	60 (0 - 120)	0.3% (0.0% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	90 (10 - 170)	0.4% (0.0% - 0.7%)	70 (10 - 140)	0.3% (0.0% - 0.6%)	60 (10 - 110)	0.3% (0.0% - 0.5%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	100 (20 - 170)	0.4% (0.1% - 0.8%)	80 (20 - 150)	0.4% (0.1% - 0.6%)	60 (10 - 120)	0.3% (0.1% - 0.5%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10 Above Background: Background = 5		Health Effects Associated with PM10 Above Background: Background = 8		Health Effects Associated with PM10 Above Background: Background = 11	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Respiratory Symptoms* **	**Single Pollutant Models**										
	Schwartz et al. (1994) -- 6 cities	Lower respiratory symptoms	7 - 14	1 day		5000 (2000 - 7000)	8.2% (3.9% - 11.7%)	4000 (2000 - 6000)	7.1% (3.3% - 10.2%)	3000 (2000 - 5000)	5.9% (2.8% - 8.5%)
	Schwartz et al. (1994) -- 6 cities	Upper respiratory symptoms	7 - 14	2 day		7000 (0 - 13000)	4.2% (0.0% - 8.1%)	6000 (0 - 11000)	3.6% (0.0% - 7.0%)	5000 (0 - 10000)	3.0% (0.0% - 5.8%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East (with a range from 5 to 11 ug/m3) and 6 ug/m3 in the West (with a range from 4 to 8 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.80. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" PM_{10} Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM-10 Above Background: Single Lag		Health Effects Associated with PM-10 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Non-accidental	all	0 day		140 (0 - 270)	0.6% (0.0% - 1.2%)	270 (0 - 530)	1.2% (0.0% - 2.3%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Non-accidental	all	0 day		240 (140 - 340)	1.0% (0.6% - 1.5%)	460 (270 - 660)	2.0% (1.2% - 2.9%)
	Dominici et al. (2003) [reanalysis of Samet et al. (2000)] -- regional	Non-accidental	all	1 day		70 (10 - 120)	0.3% (0.1% - 0.5%)	130 (30 - 230)	0.6% (0.1% - 1.0%)
	Multi-Pollutant Models (Total Mortality)								
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3	90 (40 - 150)	0.4% (0.2% - 0.7%)	180 (80 - 290)	0.8% (0.4% - 1.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, NO2	70 (0 - 150)	0.3% (0.0% - 0.7%)	140 (0 - 290)	0.6% (0.0% - 1.3%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, SO2	70 (10 - 140)	0.3% (0.0% - 0.6%)	140 (10 - 280)	0.6% (0.1% - 1.2%)
	Dominici et al.(2003) [reanalysis of Samet et al.(2000)] -- national	Non-accidental	all	1 day	O3, CO	80 (20 - 150)	0.4% (0.1% - 0.6%)	160 (30 - 290)	0.7% (0.2% - 1.3%)

*Health effects are associated with short-term exposure to PM10 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. Average background PM10 is taken to be 8 ug/m3 in the East and 6 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

D.9. The $PM_{10-2.5}$ data: Sensitivity analyses

Exhibit D.81. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations, Using Different Background Levels
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background: Background = 0		Health Effects Associated with PM10-2.5 Above Background: Background = 3		Health Effects Associated with PM10-2.5 Above Background: Background = 9	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Lipfert et al. (2000)	Non-accidental	all	0 day		340	2.0%	230	1.4%	80	0.4%
	Single Pollutant Models (Cause-Specific Mortality)										
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day		380 (0 - 800)	5.6% (0.0% - 11.6%)	260 (0 - 550)	3.8% (0.0% - 7.9%)	90 (0 - 180)	1.3% (0.0% - 2.6%)
	Multi-Pollutant Models (Total Mortality)										
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	280	1.6%	190	1.1%	60	0.4%
	Multi-Pollutant Models (Cause-Specific Mortality)										
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	0 day	O3	140	2.1%	100	1.4%	30	0.5%

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East (with a range from 0.0 to 7.0 ug/m3) and 3.0 ug/m3 in the West (with a range from 0.0 to 9.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM coarse non-accidental mortality coefficients. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Exhibit D.82. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{10-2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
Philadelphia, PA, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM10-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =3 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Lipfert et al. (2000)	Non-accidental	all	0 day		1.4%	0.5%	0.2%	0.1%
	Multi-Pollutant Models (Total Mortality)								
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	1.1%	0.4%	0.2%	0.1%

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM coarse non-accidental mortality coefficients. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Exhibit D.83. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{10-2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background: Single Lag		Health Effects Associated with PM10-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Lipfert et al. (2000)	Non-accidental	all	0 day		230	1.4%	460	2.7%
	Multi-Pollutant Models (Total Mortality)								
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	190	1.1%	380	2.2%

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM coarse non-accidental mortality coefficients. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated.

Exhibit D.84. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations, Using Different Background Levels
Phoenix, AZ, 1997

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background: Background = 0		Health Effects Associated with PM10-2.5 Above Background: Background = 3		Health Effects Associated with PM10-2.5 Above Background: Background = 7	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Cause-Specific Mortality)**										
	Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	65+	0 day		480 (100 - 830)	6.7% (1.5% - 11.6%)	480 (100 - 830)	6.7% (1.5% - 11.6%)	450 (100 - 770)	6.2% (1.4% - 10.8%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East (with a range from 0.0 to 7.0 ug/m3) and 3.0 ug/m3 in the West (with a range from 0.0 to 9.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

Exhibit D.85. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations, Using Different Background Levels
Seattle, WA, 2000

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background: Background = 0		Health Effects Associated with PM10-2.5 Above Background: Background = 3		Health Effects Associated with PM10-2.5 Above Background: Background = 7	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Hospital Admissions	**Single Pollutant Models** Sheppard (2003) [reanalysis of Sheppard et al. (1999)]***	Asthma	<65	1 day		20 (0 - 50)	1.2% (0.0% - 2.9%)	10 (0 - 20)	0.6% (0.0% - 1.4%)	0 (0 - 0)	0.1% (0.0% - 0.2%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East (with a range from 0.0 to 7.0 ug/m3) and 3.0 ug/m3 in the West (with a range from 0.0 to 9.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

***Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM coarse values obtained from nephelometer measurements rather than from air quality monitors.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

Exhibit D.86. Sensitivity Analysis: Estimated Annual Health Risks Associated with "As Is" $PM_{10-2.5}$ Concentrations, Using Different Background Levels
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background: Background = 0		Health Effects Associated with PM10-2.5 Above Background: Background = 3		Health Effects Associated with PM10-2.5 Above Background: Background = 9	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**										
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	0 day		10 (0 - 100)	0.0% (0.0% - 0.4%)	0 (0 - 40)	0.0% (0.0% - 0.2%)	0 (0 - 10)	0.0% (0.0% - 0.0%)
Respiratory Symptoms* **	**Single Pollutant Models**										
	Schwartz and Neas, 2000 -- 6 cities	Lower respiratory symptoms	7-14	0 day		4000 (0 - 14000)	7.8% (0.0% - 24.5%)	2000 (0 - 7000)	4.1% (0.0% - 12.7%)	1000 (0 - 2000)	1.3% (0.0% - 3.6%)
	Schwartz and Neas, 2000 -- 6 cities	Cough	7-14	0 day		17000 (7000 - 26000)	10.4% (4.2% - 16.1%)	9000 (4000 - 14000)	5.5% (2.2% - 8.4%)	3000 (1000 - 4000)	1.7% (0.7% - 2.5%)
	Multi-Pollutant Models										
	Schwartz and Neas, 2000 -- 6 cities	Lower respiratory symptoms	7-14	0 day	PM2.5	2000 (0 - 7000)	3.0% (0.0% - 11.7%)	1000 (0 - 3000)	1.6% (0.0% - 6.1%)	0 (0 - 1000)	0.5% (0.0% - 1.9%)
	Schwartz and Neas, 2000 -- 6 cities	Cough	7-14	0 day	PM2.5	16000 (4000 - 26000)	9.6% (2.5% - 15.8%)	8000 (2000 - 13000)	5.0% (1.3% - 8.2%)	3000 (1000 - 4000)	1.6% (0.4% - 2.5%)

*Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East (with a range from 0.0 to 7.0 ug/m3) and 3.0 ug/m3 in the West (with a range from 0.0 to 9.0 ug/m3). Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit D.87. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{10-2.5}$ Concentrations Using Alternative Hypothetical Threshold Models*
St. Louis, MO, 2002

Health Effects**	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of Total Incidence Associated with PM10-2.5 Above Hypothetical Threshold***			
						BASE CASE: Background =3 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	0 day		0.0% (0.0% - 0.2%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)

*This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.

**Health effects are associated with short-term exposure to PM coarse 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. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

Exhibit D.88. Sensitivity Analysis: Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with "As Is" $PM_{10-2.5}$ Concentrations With Adjustments for the Estimated Increases in Incidence if Distributed Lag Models Had Been Estimated
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM10-2.5 Above Background: Single Lag		Health Effects Associated with PM10-2.5 Above Background: Adjusted for Distributed Lag	
						Incidence**	Percent of Total Incidence**	Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	0 day		0 (0 - 40)	0.0% (0.0% - 0.2%)	10 (0 - 40)	0.0% (0.0% - 0.2%)

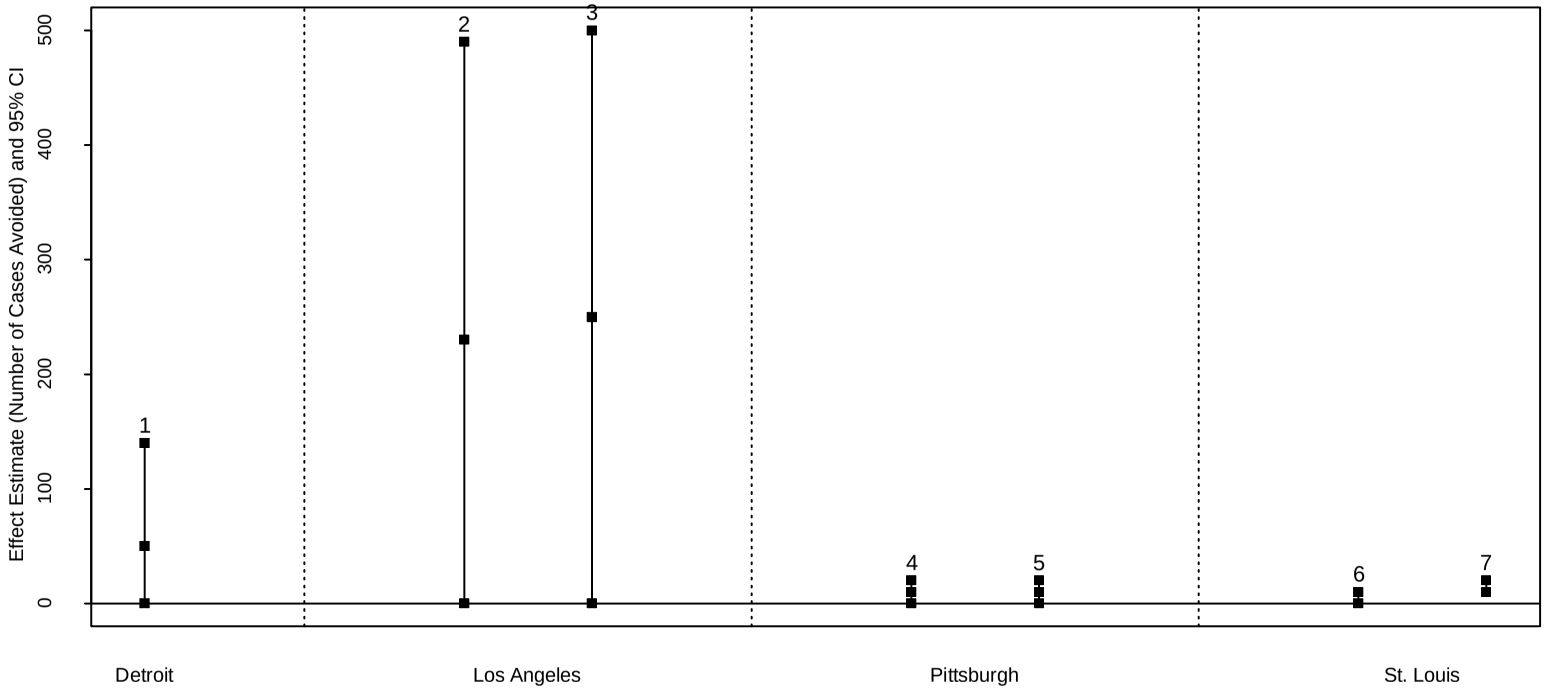
*Health effects are associated with short-term exposure to PM coarse unless otherwise specified.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background PM coarse is taken to be 3.0 ug/m3 in the East and in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM coarse coefficient.

Appendix E. Estimated Annual Health Risk Reductions Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet Current Annual and Daily Standards

Figure E.1. Estimated Annual Health Risk Reductions Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 (and 95 Percent Confidence Interval): Non-accidental Mortality Associated with Short-Term Exposure to $PM_{2.5}$ ‡

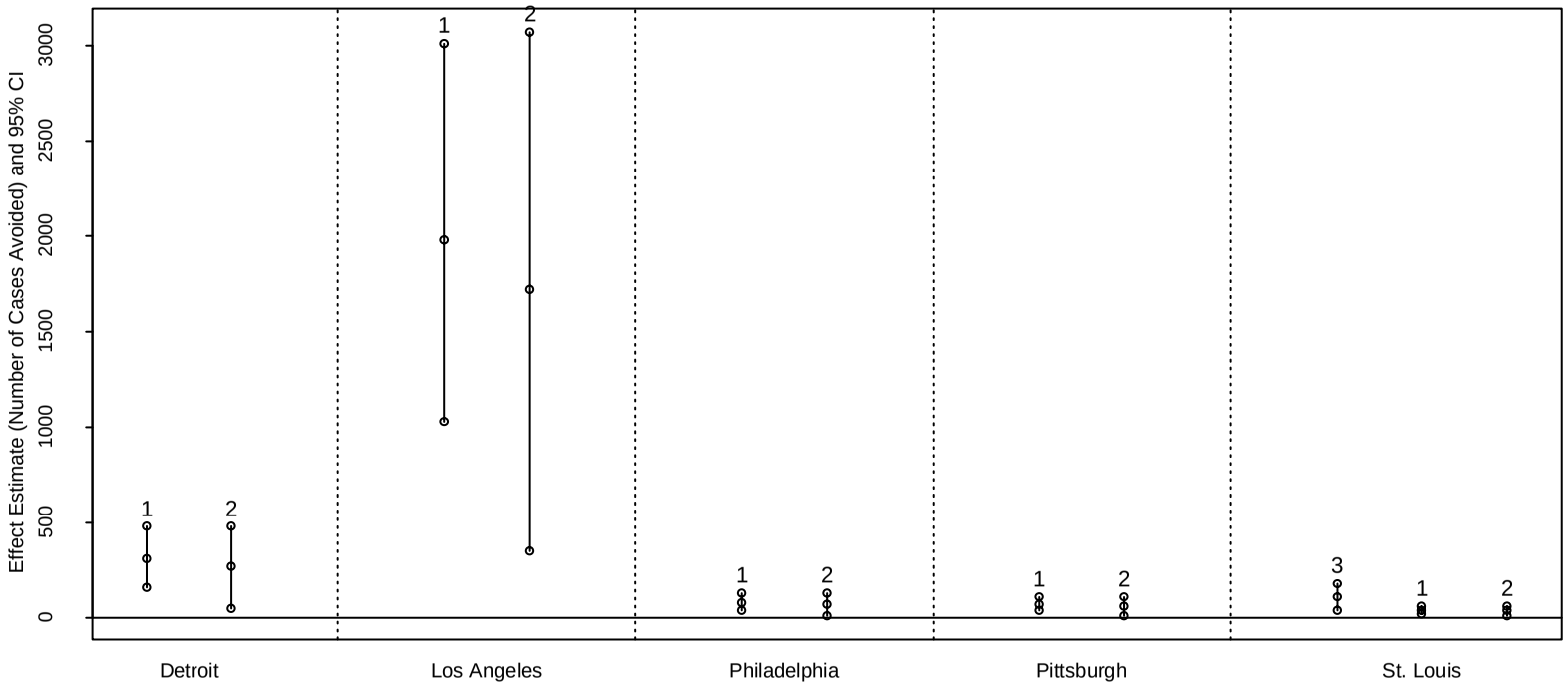


1. Lippmann et al. (2000)*
2. Moolgavkar (2000a)** – 0-day lag
3. Moolgavkar (2000a)** – 1-day lag
4. Chock et al. (2000) (age<75)
5. Chock et al. (2000) (age>75)
6. Schwartz et al. (1996)***
7. Schwartz et al. (1996)*** – 6-city model

*Reanalyzed in Ito (2003)
**Reanalyzed in Moolgavkar (2002)
***Reanalyzed in Schwartz (2003b)

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure E.2. Estimated Annual Health Risk Reductions Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 (and 95 Percent Confidence Interval): Mortality Associated with Long-Term Exposure to $PM_{2.5}$‡



1. Krewski et al. (2000) – ACS
2. Pope et al. (2002) – ACS extended
3. Krewski et al. (2000) – Six Cities

‡ In cases where the lower bound on the confidence interval was less than zero, the interval shown does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Exhibit E.1. Estimated Annual Health Risk Reductions Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3
Los Angeles, CA, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
							Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		230 (0 - 490)	45.8% (* - 46.0%)	0.4% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		250 (0 - 500)	45.9% (45.7% - 46.0%)	0.4% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		160 (0 - 400)	45.8% (* - 46.0%)	0.3% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	3 day		0 (0 - 180)	* (* - 45.8%)	0.0% (0.0% - 0.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	4 day		0 (0 - 200)	* (* - 45.8%)	0.0% (0.0% - 0.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	5 day		0 (0 - 10)	* (* - 45.7%)	0.0% (0.0% - 0.0%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		230 (0 - 490)	45.8% (* - 46.0%)	0.4% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	0 day		170 (0 - 480)	45.8% (* - 46.0%)	0.3% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		140 (0 - 370)	45.8% (* - 45.9%)	0.2% (0.0% - 0.6%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		130 (0 - 440)	45.8% (* - 46.0%)	0.2% (0.0% - 0.8%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
							Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		250 (0 - 500)	45.9% (45.7% - 46.0%)	0.4% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	1 day		240 (0 - 540)	45.8% (* - 46.1%)	0.4% (0.0% - 0.9%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		40 (0 - 280)	45.7% (* - 45.9%)	0.1% (0.0% - 0.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		0 (0 - 410)	* (* - 46.0%)	0.0% (0.0% - 0.7%)
	Single Pollutant Models (Cause-Specific Mortality)								
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	0 day		150 (20 - 290)	46.0% (45.7% - 46.2%)	0.7% (0.1% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	0 day		150 (20 - 280)	46.0% (45.7% - 46.2%)	0.7% (0.1% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	0 day		150 (0 - 300)	46.0% (* - 46.3%)	0.7% (0.0% - 1.5%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 30 df	1 day		160 (20 - 290)	46.0% (45.7% - 46.2%)	0.8% (0.1% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	1 day		120 (0 - 250)	45.9% (* - 46.2%)	0.6% (0.0% - 1.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	1 day		110 (0 - 260)	45.9% (* - 46.2%)	0.5% (0.0% - 1.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		0 (0 - 40)	* (* - 46.2%)	0.0% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		0 (0 - 20)	* (* - 46.0%)	0.0% (0.0% - 0.7%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	0 day		0 (0 - 40)	* (* - 46.2%)	0.0% (0.0% - 1.2%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
							Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		10 (0 - 60)	45.8% (* - 46.5%)	0.3% (0.0% - 2.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		20 (0 - 70)	45.9% (* - 46.6%)	0.6% (0.0% - 2.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	COPD+	all	log-linear, GLM, 100 df	1 day		0 (0 - 70)	45.7% (* - 46.6%)	0.2% (0.0% - 2.3%)
	Multi-Pollutant Models (Total Mortality)								
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day	CO	0 (0 - 110)	* (* - 45.8%)	0.0% (0.0% - 0.2%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day	CO	0 (0 - 170)	* (* - 45.8%)	0.0% (0.0% - 0.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day	CO	0 (0 - 220)	* (* - 45.8%)	0.0% (0.0% - 0.4%)
	Multi-Pollutant Models (Cause-Specific Mortality)								
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	0 day	CO	280 (120 - 430)	46.2% (45.9% - 46.5%)	1.3% (0.6% - 2.1%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	0 day	CO	290 (110 - 470)	46.2% (45.9% - 46.6%)	1.4% (0.5% - 2.3%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GAM (stringent), 100 df	1 day	CO	140 (0 - 300)	45.9% (* - 46.2%)	0.7% (0.0% - 1.4%)
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	all	log-linear, GLM, 100 df	1 day	CO	140 (0 - 330)	45.9% (* - 46.3%)	0.7% (0.0% - 1.6%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
							Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Long-Term Exposure Mortality	**Single Pollutant Models**								
	Krewski et al. (2000) - ACS	All cause	30+	log-linear			1980 (1030 - 3010)	72.5% (72.2% - 72.7%)	3.4% (1.8% - 5.2%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+	log-linear			2210 (1440 - 3040)	73.0% (72.6% - 73.4%)	6.8% (4.4% - 9.4%)
	Pope et al. (2002) - ACS extended	All cause	30+	log-linear			1720 (350 - 3070)	72.4% (72.1% - 72.7%)	3.0% (0.6% - 5.3%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+	log-linear			1360 (360 - 2340)	72.6% (72.1% - 73.0%)	4.2% (1.1% - 7.2%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+	log-linear			190 (30 - 340)	72.8% (72.1% - 73.5%)	5.7% (0.8% - 10.4%)
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		CO	2870 (1670 - 4100)	72.7% (72.4% - 73.0%)	4.9% (2.9% - 7.1%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		NO2	3430 (1830 - 4860)	72.8% (72.4% - 73.2%)	5.9% (3.2% - 8.4%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		O3	2870 (1670 - 4100)	72.7% (72.4% - 73.0%)	4.9% (2.9% - 7.1%)
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		SO2	520 (0 - 2130)	72.1% (* - 72.5%)	0.9% (0.0% - 3.7%)
Hospital Admissions	**Single Pollutant Models**								
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	0 day		810 (470 - 1150)	46.1% (45.9% - 46.3%)	1.2% (0.7% - 1.7%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day		600 (260 - 930)	46.0% (45.8% - 46.2%)	0.9% (0.4% - 1.3%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day		650 (240 - 1060)	46.0% (45.8% - 46.3%)	0.9% (0.3% - 1.5%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 30 df	1 day		720 (360 - 1070)	46.1% (45.9% - 46.3%)	1.0% (0.5% - 1.5%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day		580 (240 - 920)	46.0% (45.8% - 46.2%)	0.8% (0.4% - 1.3%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day		620 (200 - 1030)	46.0% (45.8% - 46.3%)	0.9% (0.3% - 1.5%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
							Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	0 day		380 (160 - 590)	46.2% (45.9% - 46.5%)	1.2% (0.5% - 2.0%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day		310 (120 - 500)	46.1% (45.8% - 46.3%)	1.0% (0.4% - 1.7%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	0 day		340 (90 - 570)	46.1% (45.8% - 46.4%)	1.1% (0.3% - 1.9%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	1 day		270 (50 - 480)	46.0% (45.7% - 46.3%)	0.9% (0.2% - 1.6%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day		170 (0 - 360)	45.9% (* - 46.1%)	0.6% (0.0% - 1.2%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	1 day		170 (0 - 400)	45.9% (* - 46.2%)	0.6% (0.0% - 1.3%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 30 df	2 day		420 (190 - 640)	46.2% (45.9% - 46.5%)	1.4% (0.6% - 2.1%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day		260 (50 - 460)	46.0% (45.7% - 46.3%)	0.9% (0.2% - 1.5%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GLM, 100 df	2 day		230 (0 - 480)	46.0% (* - 46.3%)	0.8% (0.0% - 1.6%)
	Multi-Pollutant Models								
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	0 day	CO	200 (0 - 630)	45.8% (* - 46.0%)	0.3% (0.0% - 0.9%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	0 day	CO	300 (0 - 800)	45.8% (* - 46.1%)	0.4% (0.0% - 1.2%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GAM (stringent), 100 df	1 day	CO	120 (0 - 580)	45.7% (* - 46.0%)	0.2% (0.0% - 0.8%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	65+	log-linear, GLM, 100 df	1 day	CO	140 (0 - 660)	45.8% (* - 46.1%)	0.2% (0.0% - 1.0%)

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
							Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	0 day	NO2	100 (0 - 390)	45.8% (* - 46.2%)	0.3% (0.0% - 1.3%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	1 day	NO2	0 (0 - 340)	* (* - 46.1%)	0.0% (0.0% - 1.1%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	2 day	NO2	80 (0 - 380)	45.8% (* - 46.2%)	0.3% (0.0% - 1.3%)
	Moolgavkar (2002) [reanalysis of Moolgavkar (2000c)]	COPD+	all	log-linear, GAM (stringent), 100 df	3 day	NO2	0 (0 - 50)	* (* - 45.7%)	0.0% (0.0% - 0.2%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. An asterisk indicates that the lower bound (and, in some cases, the central estimate) could not be calculated.

Exhibit E.2. Estimated Annual Health Risk Reductions Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**							
	Lipfert et al. (2000)	Non-accidental	all	0 day		30	9.5%	0.2%
	Single Pollutant Models (Cause-Specific Mortality)							
	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day		40 (20 - 60)	9.5% (9.5% - 9.5%)	0.6% (0.3% - 0.9%)
	Multi-Pollutant Models (Total Mortality)							
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	20	9.5%	0.1%
	Multi-Pollutant Models (Cause-Specific Mortality)							
	Lipfert et al. (2000) -- 7 counties	Cardiovascluar	all	0 day	O3	10	9.5%	0.2%
Long-Term Exposure Mortality	**Single Pollutant Models**							
	Krewski et al. (2000) - ACS	All cause	30+			80 (40 - 130)	23.3% (23.2% - 23.4%)	0.5% (0.3% - 0.7%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			80 (50 - 110)	23.5% (23.3% - 23.6%)	1.0% (0.6% - 1.4%)
	Pope et al. (2002) - ACS extended	All cause	30+			70 (10 - 130)	23.3% (23.1% - 23.4%)	0.4% (0.1% - 0.8%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			50 (10 - 90)	23.3% (23.2% - 23.5%)	0.6% (0.2% - 1.0%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			10 (0 - 20)	23.4% (23.1% - 23.7%)	0.8% (0.1% - 1.5%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models							
	Krewski et al. (2000) - ACS	All cause	30+		CO	120 (70 - 180)	23.4% (23.2% - 23.5%)	0.7% (0.4% - 1.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	150 (80 - 210)	23.4% (23.3% - 23.6%)	0.8% (0.4% - 1.2%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	120 (70 - 180)	23.4% (23.2% - 23.5%)	0.7% (0.4% - 1.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	20 (0 - 90)	23.1% (* - 23.3%)	0.1% (0.0% - 0.5%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM2.5 non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient. Therefore the 95% confidence intervals for the corresponding health effects estimates can not be calculated. An asterisk indicates that the lower bound could not be calculated.

Note 2: Multi-county short-term exposure C-R functions were applied only to counties included among those used to estimate the function.

Exhibit E.3. Estimated Annual Health Risk Reductions Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3
Pittsburgh, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**							
	Chock et al. (2000)	Non-accidental	<75	0 day		10 (0 - 20)	8.8% (* - 8.9%)	0.1% (0.0% - 0.3%)
	Chock et al. (2000)	Non-accidental	75+	0 day		10 (0 - 20)	8.8% (* - 8.9%)	0.1% (0.0% - 0.3%)
	Multi-Pollutant Models (Total Mortality)							
	Chock et al. (2000)	Non-accidental	<75	0 day	CO, O3, SO2, NO2, PM10-2.5	10 (0 - 20)	8.8% (* - 9.0%)	0.1% (0.0% - 0.4%)
	Chock et al. (2000)	Non-accidental	75+	0 day	CO, O3, SO2, NO2, PM10-2.5	0 (0 - 20)	8.8% (* - 8.9%)	0.0% (0.0% - 0.3%)
Long-Term Exposure Mortality	**Single Pollutant Models**							
	Krewski et al. (2000) - ACS	All cause	30+			70 (40 - 110)	19.8% (19.8% - 19.9%)	0.5% (0.2% - 0.7%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			70 (50 - 100)	20.0% (19.9% - 20.2%)	1.0% (0.6% - 1.3%)
	Pope et al. (2002) - ACS extended	All cause	30+			60 (10 - 110)	19.8% (19.7% - 20.0%)	0.4% (0.1% - 0.7%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			40 (10 - 80)	19.9% (19.7% - 20.1%)	0.6% (0.2% - 1.0%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			10 (0 - 20)	20.0% (19.7% - 20.3%)	0.8% (0.1% - 1.5%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
	Multi-Pollutant Models							
	Krewski et al. (2000) - ACS	All cause	30+		CO	100 (60 - 150)	19.9% (19.8% - 20.1%)	0.7% (0.4% - 1.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	120 (60 - 180)	20.0% (19.8% - 20.1%)	0.8% (0.4% - 1.2%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	100 (60 - 150)	19.9% (19.8% - 20.1%)	0.7% (0.4% - 1.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	20 (0 - 80)	19.7% (* - 19.9%)	0.1% (0.0% - 0.5%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. An asterisk indicates that the lower bound could not be calculated.

Exhibit E.4. Estimated Annual Health Risk Reductions Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**							
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		10 (0 - 10)	3.4% (3.4% - 3.4%)	0.0% (0.0% - 0.1%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		10 (10 - 20)	3.4% (3.4% - 3.4%)	0.1% (0.0% - 0.1%)
	Single Pollutant Models (Cause-Specific Mortality)							
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	all	0 day		0 (0 - 0)	3.4% (* - 3.5%)	0.0% (0.0% - 0.2%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	all	0 day		0 (0 - 0)	3.4% (3.4% - 3.4%)	0.1% (0.0% - 0.1%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	all	0 day		0 (0 - 0)	3.4% (* - 3.5%)	0.0% (0.0% - 0.2%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	COPD	all	0 day		0 (0 - 0)	3.4% (3.4% - 3.5%)	0.1% (0.0% - 0.2%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Ischemic heart disease	all	0 day		0 (0 - 0)	3.4% (3.4% - 3.4%)	0.1% (0.0% - 0.1%)
	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Pneumonia	all	0 day		0 (0 - 0)	3.5% (3.4% - 3.5%)	0.2% (0.1% - 0.2%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Long-Term Exposure Mortality	**Single Pollutant Models**							
	Krewski et al. (2000) - Six Cities	All cause	25+			110 (40 - 180)	10.4% (10.2% - 10.5%)	0.5% (0.2% - 0.8%)
	Krewski et al. (2000) - ACS	All cause	30+			40 (20 - 60)	8.1% (8.1% - 8.1%)	0.2% (0.1% - 0.3%)
	Krewski et al. (2000) - Six Cities	Cardiopulmonary	25+			70 (20 - 120)	10.5% (10.3% - 10.7%)	0.6% (0.2% - 1.0%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			40 (30 - 60)	8.2% (8.1% - 8.2%)	0.4% (0.2% - 0.5%)
	Pope et al. (2002) - ACS extended	All cause	30+			40 (10 - 60)	8.1% (8.0% - 8.1%)	0.2% (0.0% - 0.3%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			30 (10 - 50)	8.1% (8.0% - 8.2%)	0.2% (0.1% - 0.4%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			10 (0 - 10)	8.1% (8.0% - 8.3%)	0.3% (0.0% - 0.6%)
	Multi-Pollutant Models							
	Krewski et al. (2000) - ACS	All cause	30+		CO	60 (30 - 90)	8.1% (8.1% - 8.2%)	0.3% (0.2% - 0.4%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	70 (40 - 100)	8.2% (8.1% - 8.2%)	0.3% (0.2% - 0.4%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	60 (30 - 90)	8.1% (8.1% - 8.2%)	0.3% (0.2% - 0.4%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	10 (0 - 40)	8.0% (* - 8.1%)	0.1% (0.0% - 0.2%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Reductions Associated with Just Meeting Current PM-2.5 Standards		
						Reduction in Incidence**	Percent of PM-Related Incidence**	Percent of Total Incidence**
Respiratory Symptoms* **	**Single Pollutant Models**							
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day		0 (0 - 1000)	4.0% (3.6% - 4.2%)	0.8% (0.3% - 1.3%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day		1000 (0 - 1000)	3.6% (* - 3.9%)	0.4% (0.0% - 0.9%)
	Multi-Pollutant Models							
	Schwartz and Neas (2000) -- 6 cities	Lower respiratory symptoms	7-14	1 day	PM10-2.5	0 (0 - 1000)	3.9% (3.5% - 4.2%)	0.7% (0.2% - 1.2%)
	Schwartz and Neas (2000) -- 6 cities	Cough	7-14	0 day	PM10-2.5	0 (0 - 1000)	3.5% (* - 3.8%)	0.2% (0.0% - 0.6%)

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10, except respiratory symptoms incidences which are rounded to the nearest 1000; percents are rounded to the nearest tenth.

***The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Note 1: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient. An asterisk indicates that the lower bound could not be calculated.

Note 2: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Exhibit E.5. Sensitivity Analysis: Estimated Annual Reductions of Short-Term and Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 Using an Alternative Rollback Method
Los Angeles, CA, 2002

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Annual and Daily Standards	Percent Change in PM-Associated Incidence**		Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback Method
								All PM concentrations rolled back equally	Percent rollback of upper 10% of AQ distribution = 1.6 x percent rollback of lower 90% of AQ distribution	
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**									
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		15 ug/m3 annual 65 ug/m3 daily	45.8% 0.0%	45.9% 0.0%	100.2% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		15 ug/m3 annual 65 ug/m3 daily	45.9% 0.0%	45.9% 0.0%	100.0% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	2 day		15 ug/m3 annual 65 ug/m3 daily	45.8% 0.0%	45.9% 0.0%	100.2% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	0 day		15 ug/m3 annual 65 ug/m3 daily	45.8% 0.0%	45.9% 0.0%	100.2% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	0 day		15 ug/m3 annual 65 ug/m3 daily	45.8% 0.0%	45.9% 0.0%	100.2% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	0 day		15 ug/m3 annual 65 ug/m3 daily	45.8% 0.0%	45.9% 0.0%	100.2% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	0 day		15 ug/m3 annual 65 ug/m3 daily	45.8% 0.0%	45.9% 0.0%	100.2% --

Health Effects*	Study	Type	Ages	Model	Lag	Other Pollutants in Model	Annual and Daily Standards	Percent Change in PM-Associated Incidence**		Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback Method
								All PM concentrations rolled back equally	Percent rollback of upper 10% of AQ distribution = 1.6 x percent rollback of lower 90% of AQ distribution	
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 30 df	1 day		15 ug/m3 annual 65 ug/m3 daily	45.9% 0.0%	45.9% 0.0%	100.0% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 30 df	1 day		15 ug/m3 annual 65 ug/m3 daily	45.8% 0.0%	45.9% 0.0%	100.2% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GAM (stringent), 100 df	1 day		15 ug/m3 annual 65 ug/m3 daily	45.7% 0.0%	45.8% 0.0%	100.2% --
	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	log-linear, GLM, 100 df	1 day		15 ug/m3 annual 65 ug/m3 daily	45.7% 0.0%	45.8% 0.0%	100.2% --
Long-Term Exposure Mortality	**Single Pollutant Models**									
	Krewski et al. (2000) - ACS	All cause	30+	log-linear			15 ug/m3 annual 65 ug/m3 daily	72.5% 0.0%	72.7% 0.0%	100.3% --
	Pope et al. (2002) - ACS extended	All cause	30+	log-linear			15 ug/m3 annual 65 ug/m3 daily	72.4% 0.0%	72.6% 0.0%	100.3% --
	Multi-Pollutant Models									
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		CO	15 ug/m3 annual 65 ug/m3 daily	72.7% 0.0%	72.9% 0.0%	100.3% --
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		NO2	15 ug/m3 annual 65 ug/m3 daily	72.8% 0.0%	73.0% 0.0%	100.3% --
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		O3	15 ug/m3 annual 65 ug/m3 daily	72.7% 0.0%	72.9% 0.0%	100.3% --
	Krewski et al. (2000) - ACS	All cause	30+	log-linear		SO2	15 ug/m3 annual 65 ug/m3 daily	72.1% 0.0%	72.3% 0.0%	100.3% --

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Only those C-R functions for which rollbacks are predicted to result in a positive number of cases avoided are included.

Exhibit E.6. Sensitivity Analysis: Estimated Annual Reductions of Short-Term and Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 Using an Alternative Rollback Method
Philadelphia, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Annual and Daily Standards	Percent Change in PM-Associated Incidence**		Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback Method
							All PM concentrations rolled back equally	Percent rollback of upper 10% of AQ distribution = 1.6 x percent rollback of lower 90% of AQ distribution	
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Lipfert et al. (2000)	Non-accidental	all	0 day		15 ug/m3 annual	9.5%	9.1%	95.8%
						65 ug/m3 daily	0.0%	0.0%	--
	Multi-Pollutant Models (Total Mortality)								
	Lipfert et al. (2000)	Non-accidental	all	0 day	O3	15 ug/m3 annual	9.5%	9.1%	95.8%
						65 ug/m3 daily	0.0%	0.0%	--
Long-Term Exposure Mortality	**Single Pollutant Models**								
	Krewski et al. (2000) - ACS	All cause	30+			15 ug/m3 annual	23.3%	23.4%	100.4%
						65 ug/m3 daily	0.0%	0.0%	--
	Pope et al. (2002) - ACS extended	All cause	30+			15 ug/m3 annual	23.3%	23.3%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	15 ug/m3 annual	23.4%	23.4%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Krewski et al. (2000) - ACS	All cause	30+		NO2	15 ug/m3 annual	23.4%	23.5%	100.4%
						65 ug/m3 daily	0.0%	0.0%	--
	Krewski et al. (2000) - ACS	All cause	30+		O3	15 ug/m3 annual	23.4%	23.4%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Krewski et al. (2000) - ACS	All cause	30+		SO2	15 ug/m3 annual	23.1%	23.2%	100.4%
						65 ug/m3 daily	0.0%	0.0%	--

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Only those C-R functions for which rollbacks are predicted to result in a positive number of cases avoided are included.

Exhibit E.7. Sensitivity Analysis: Estimated Annual Reductions of Short-Term and Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 Using an Alternative Rollback Method
Pittsburgh, PA, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Annual and Daily Standards	Percent Change in PM-Associated Incidence**		Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback Method
							All PM concentrations rolled back equally	Percent rollback of upper 10% of AQ distribution = 1.6 x percent rollback of lower 90% of AQ distribution	
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Chock et al. (2000)	Non-accidental	<75	0 day		15 ug/m3 annual	8.8%	8.9%	101.1%
						65 ug/m3 daily	0.0%	0.0%	--
	Chock et al. (2000)	Non-accidental	75+	0 day		15 ug/m3 annual	8.8%	8.9%	101.1%
						65 ug/m3 daily	0.0%	0.0%	--
	Multi-Pollutant Models (Total Mortality)								
	Chock et al. (2000)	Non-accidental	<75	0 day	CO, O3, SO2, NO2, PM10-2.5	15 ug/m3 annual	8.8%	8.9%	101.1%
						65 ug/m3 daily	0.0%	0.0%	--
	Chock et al. (2000)	Non-accidental	75+	0 day	CO, O3, SO2, NO2, PM10-2.5	15 ug/m3 annual	8.8%	8.9%	101.1%
						65 ug/m3 daily	0.0%	0.0%	--
Long-Term Exposure Mortality	**Single Pollutant Models**								
	Krewski et al. (2000) - ACS	All cause	30+			15 ug/m3 annual	19.8%	20.0%	101.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Pope et al. (2002) - ACS extended	All cause	30+			15 ug/m3 annual	19.8%	20.0%	101.0%
						65 ug/m3 daily	0.0%	0.0%	--

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Annual and Daily Standards	Percent Change in PM-Associated Incidence**		Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback Method
							All PM concentrations rolled back equally	Percent rollback of upper 10% of AQ distribution = 1.6 x percent rollback of lower 90% of AQ distribution	
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	15 ug/m3 annual 65 ug/m3 daily	19.9% 0.0%	20.1% 0.0%	101.0% --
	Krewski et al. (2000) - ACS	All cause	30+		NO2	15 ug/m3 annual 65 ug/m3 daily	20.0% 0.0%	20.2% 0.0%	101.0% --
	Krewski et al. (2000) - ACS	All cause	30+		O3	15 ug/m3 annual 65 ug/m3 daily	19.9% 0.0%	20.1% 0.0%	101.0% --
	Krewski et al. (2000) - ACS	All cause	30+		SO2	15 ug/m3 annual 65 ug/m3 daily	19.7% 0.0%	19.9% 0.0%	101.0% --

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Only those C-R functions for which rollbacks are predicted to result in a positive number of cases avoided are included.

Exhibit E.8. Sensitivity Analysis: Estimated Annual Reductions of Short-Term and Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 Using an Alternative Rollback Method
St. Louis, MO, 2002

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Annual and Daily Standards	Percent Change in PM-Associated Incidence**		Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback Method
							All PM concentrations rolled back equally	Percent rollback of upper 10% of AQ distribution = 1.6 x percent rollback of lower 90% of AQ distribution	
Short-Term Exposure Mortality	**Single Pollutant Models (Total Mortality)**								
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1		15 ug/m3 annual	3.4%	3.3%	97.1%
						65 ug/m3 daily	0.0%	0.0%	--
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	all	mean of lag 0 & 1		15 ug/m3 annual	3.4%	3.3%	97.1%
						65 ug/m3 daily	0.0%	0.0%	--
Long-Term Exposure Mortality	**Single Pollutant Models**								
	Krewski et al. (2000) - Six Cities	All cause	25+			15 ug/m3 annual	10.4%	10.4%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Krewski et al. (2000) - ACS	All cause	30+			15 ug/m3 annual	8.1%	8.1%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Pope et al. (2002) - ACS extended	All cause	30+			15 ug/m3 annual	8.1%	8.1%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Multi-Pollutant Models								
	Krewski et al. (2000) - ACS	All cause	30+		CO	15 ug/m3 annual	8.1%	8.1%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Krewski et al. (2000) - ACS	All cause	30+		NO2	15 ug/m3 annual	8.2%	8.2%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Krewski et al. (2000) - ACS	All cause	30+		O3	15 ug/m3 annual	8.1%	8.1%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--
	Krewski et al. (2000) - ACS	All cause	30+		SO2	15 ug/m3 annual	8.0%	8.0%	100.0%
						65 ug/m3 daily	0.0%	0.0%	--

*Health effects are associated with short-term exposure to PM2.5 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. Average background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note 1: Multi-city short-term exposure C-R functions were applied only to urban areas included among the cities used to estimate the function.

Note 2: Only those C-R functions for which rollbacks are predicted to result in a positive number of cases avoided are included.