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

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This report is being furnished to the U.S. Environmental Protection Agency (EPA) by Abt Associates Inc. in partial fulfillment of Contract No. 68-D-03-002, Work Assignment Nos. 1-15 and 2-22. Some of the preliminary work for this report was completed under Contract No. 68-D-98-001, Work Assignments 1-36, 2-46, 3-51, and 4-65 and Contract No. 68-D-03-002, Work Assignment No. 0-04. The opinions, findings, and conclusions expressed are those of the authors and are not necessarily those of the EPA. This draft report is being circulated for review by the Clean Air Scientific Advisory Committee and the general public. All comments and inquiries should be addressed to Mr. Harvey Richmond, U.S. EPA, Office of Air Quality Planning and Standards, C539-01, Research Triangle Park, North Carolina 27711.

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, Abt Associates expressly understands that the Michigan Health and Hospital Association has not performed an analysis of the hospitalization data obtained or warranted the accuracy of this information and, therefore, it cannot 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. The U.S. Court of Appeals for the District of Columbia Circuit found "ample support" for EPA's decision to regulate coarse particle pollution, but vacated the Agency's 1997 revisions to the PM_{10} standards, concluding in part that PM_{10} is a "poorly matched indicator for coarse particle pollution" because it includes fine particles. (*American Trucking Association v. EPA*, 175 F. 3d 1027, 1053-55 (D.C. Cir. 1999). The 1987 PM_{10} standards remain in effect. 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 $\mu g/m^3$, based on the 3-year average of the 98^{th} 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 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 (U.S. 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's final assessment of the available PM health effects literature is contained in the October 2004 final report, *Air Quality Criteria for Particulate Matter* (U.S. EPA, 2004) (hereafter 2004 PM CD). This final report underwent extensive review and comment by the Clean Air Scientific Advisory Committee's (CASAC) PM Review Panel and the general public over the last three years. The 2004 PM CD includes an evaluation of the scientific evidence on the health effects of PM, including information on exposure, physiological 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 2004 PM CD continue to show associations between serious health effects, including premature mortality, and ambient $PM_{2.5}$ concentrations. In both the last and current 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. In the past review, 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, for the current review there are significantly more studies available 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 2004 PM CD also now includes a relatively smaller set 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, $PM_{10-2.5}$.[1] 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

[1]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).

software often employed to fit these models. Using appropriate modifications of the default convergence 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). Among the panel's general conclusions was that:

> The impact of using more appropriate convergence criteria on the estimates of PM effect in the revised analyses varied greatly across the studies. In some studies, stricter convergence criteria had little impact, and in a few the impact was substantial. In no study were conclusions based on the original analyses changed in a meaningful way by the use of stricter criteria. Explanations for this variability considered by the Panel include the degree of temporal smoothing used in the original analyses, the number of smoothed terms in the models, and the degree of nonlinear collinearity (concurvity) among the smoothed terms. The relative importance of these and other explanations remains unclear. (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. In August 2003 a precursor to the current draft report presented preliminary results from risk assessments for three PM indicators – $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ – and provided a 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. The August 2003 draft report (Abt Associates Inc., 2003) presented assessments of the health risks associated with "as is" concentrations of each of the three PM indicators in excess of their policy relevant background (PRB) levels, as well as an assessment of the risk reductions associated with just meeting the current $PM_{2.5}$ standards.

The risk assessment described in this report focuses on the two PM indicators for which EPA now anticipates making decisions – $PM_{2.5}$ and $PM_{10-2.5}$. The report provides a description of the methodology used, taking into account comments received from the CASAC (Hopke, 2004) and the public on the August 2003 draft report, and describing air quality data and baseline incidence rates for mortality that have been updated since the previous (August 2003) draft report. The report also presents the assessments of the health risks associated with "as is" concentrations of $PM_{2.5,}$ $PM_{10-2.5}$, and PM_{10} in excess of their PRB levels, as well as an assessment of the risk reductions associated with just meeting the current $PM_{2.5}$ standards as well

as alternative $PM_{2.5}$ standards. In addition, the report includes assessments of the health risks associated with just meeting alternative $PM_{10-2.5}$ standards. The risk assessment is based on the health effects evidence assessed in the 2004 PM CD, which includes the re-analyzed studies presented in the HEI special report (HEI, 2003).

The goals of the PM risk assessment are: (1) to provide estimates of the potential magnitude of mortality and morbidity associated with current $PM_{2.5}$ and $PM_{10-2.5}$ levels and with attaining the current suite of $PM_{2.5}$ NAAQS (as well as the additional estimated reductions in effects associated with attaining alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards identified as part of this review) in specific urban areas,[2] (2) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates (e.g., choice of PRB levels, and consideration of potential hypothetical thresholds), and (3) to gain insights into the nature of the risks associated with exposures to ambient PM (e.g., patterns of risk reduction associated with meeting alternative annual and daily standards). As discussed in the January 2005 draft Staff Paper, *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information - OAQPS Staff Paper, Second Draft* (U.S. EPA, 2005) (hereafter 2005 PM SP), the risk assessment in this standards review must take into consideration significant uncertainties associated with the assessment, as discussed in Section 6 below.

As discussed in Chapter 9 of the 2004 PM CD (p. 9-79), "the new evidence from mechanistic studies suggesting plausible biological response pathways, and the extensive body of epidemiology evidence on associations between short- and long-term exposures to ambient thoracic particles (typically indexed by PM_{10}) and a range of health effects, supports the general conclusion that ambient thoracic particles, acting alone and/or in combination with gaseous co-pollutants, are likely causally related to cardiovascular and respiratory mortality and morbidity." The 2004 PM CD (p.9-79) also concludes that "a growing body of evidence both from epidemiological and toxicological studies also supports the general conclusion that $PM_{2.5}$ (or one or more $PM_{2.5}$ components), acting alone and/or in combination with gaseous co-pollutants are likely causally related to cardiovascular and respiratory mortality and morbidity." With respect to $PM_{10-2.5}$, the 2004 PM CD (pp.9-79 to 9-80) finds that there is "a much more limited body of evidence ... suggestive of associations between short-term (but not long-term) exposures to ambient coarse-fraction thoracic particles... and various mortality and morbidity effects observed at times in some locations." The 2004 PM CD (p.9-80) concludes that "the strength of the evidence varies across endpoints, with somewhat stronger evidence for coarse-fraction particle associations with morbidity (especially respiratory) endpoints than for mortality." The $PM_{2.5}$ risk assessment described in this draft report is premised on the assumptions that $PM_{2.5}$ is causally related to the mortality, morbidity, and symptomatic effects (alone and/or in

[2]Risk estimates associated with current PM_{10} levels also have been included in an appendix to this report for those urban areas where $PM_{2.5}$ risks have been estimated to provide additional context.

combination with other pollutants) observed in the epidemiological studies and that $PM_{10-2.5}$ is causally related to the morbidity and symptomatic effects observed in the epidemiology studies. We recognize, as discussed in the PM CD (p.8-327), that "the apparent differences in $PM_{2.5}$ and/or $PM_{10-2.5}$ 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 $PM_{10-2.5}$ 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 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, Seattle, 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 $PM_{10-2.5}$ 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 and health effects for which there is sufficient evidence. While a few studies have reported positive statistically significant associations in some locations between $PM_{10-2.5}$ and non-accidental total mortality and cause-specific mortality (due to short-term exposure), the majority of studies investigating these relationships have not reported statistically significant results. Therefore, EPA does not believe the weight of the evidence supports including short-term exposure mortality in the quantitative $PM_{10-2.5}$ health risk assessment (see 2005 PM SP (U.S. EPA, 2005)) for further discussion of the evidence relating $PM_{10-2.5}$ and short-term exposure mortality). The areas included in the $PM_{10-2.5}$ risk assessment are Detroit and Seattle (where an association has been shown between $PM_{10-2.5}$ and hospital admissions) and St. Louis (where an association has been shown between $PM_{10-2.5}$ and respiratory symptoms).

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"[3] PM concentrations in excess of background concentrations in these locations?[4]

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 $PM_{10\text{-}2.5}$ standards were just met in the selected locations. 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 discussed briefly in Section 2.6 and in greater detail in Section 6. The results of the base case and sensitivity analyses from the first – recent air quality/"as is" – part of the risk assessment are discussed in Section 7, and the results of the second – just meeting the current and alternative standards are discussed in Sections 8 and 9. Section 8 focuses on $PM_{2.5}$ and Section 9 focuses on $PM_{10\text{-}2.5}$. Appendix A discusses the air quality data used in the analyses. Appendix B describes an analysis of historical air quality data relevant to the choice of air quality adjustment

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

[4] 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 the U.S., Canada, and Mexico. Therefore, "background" for the purposes of the PM risk assessment includes PM from natural sources and transport of PM from sources outside of the U.S., Canada, and Mexico.

procedures for simulating attainment of current and alternative $PM_{2.5}$ and $PM_{10-2.5}$ 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 $PM_{2.5}$ and $PM_{10-2.5}$ and a substantial number of urban locations, there are many exhibits of results. The results for both PM indicators are summarized in figures in Sections 7, 8, and 9. Most of the 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, F, G, and H for base case and sensitivity analyses from the second – just meeting the current $PM_{2.5}$ standards and alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards – part of the risk assessment. Finally, Appendix I presents the results of a PM_{10} "as is" air quality risk assessment for locations and health endpoints for which results are presented in the $PM_{2.5}$ 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 $PM_{2.5}$ and $PM_{10\text{-}2.5}$ PRB 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.

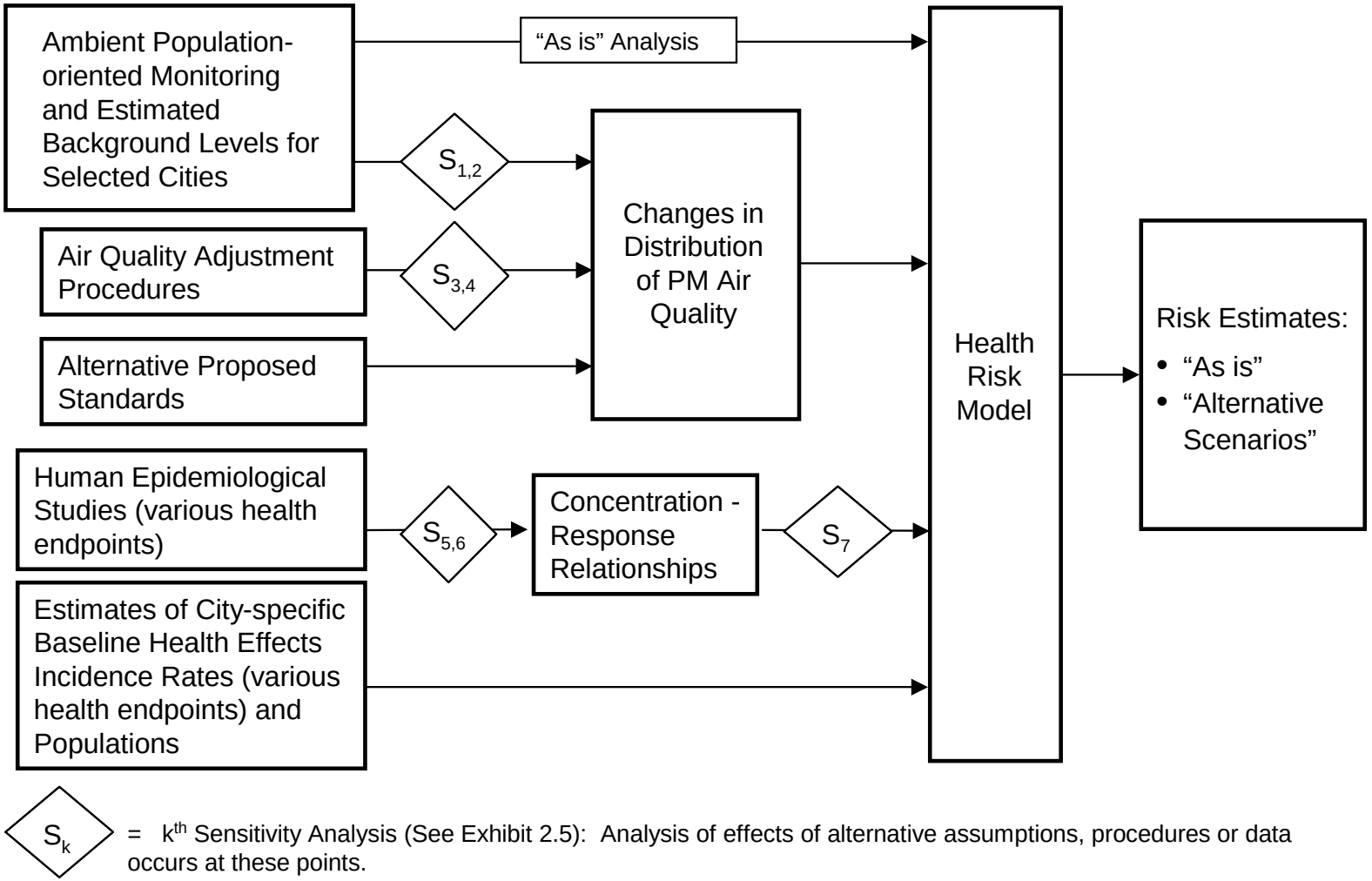
2.1 Basic structure of the risk assessment

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 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 2.6 below.

In the first part of the risk assessment, we estimate risks associated with "as is" PM levels in excess of PRB level, or the lowest measured level (LML) observed in the study, if it is higher than the estimated PRB 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 LML 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 PRB (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 $PM_{10-2.5}$ 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 PRB 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. (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 $PM_{10-2.5}$, 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 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.

2.2 Air quality inputs

2.2.1 Estimating policy relevant PM background levels

Since health risks are calculated only for concentrations exceeding estimated PRB levels, estimates of PRB PM concentrations in the assessment locations are needed to calculate risk at "as is" concentrations in excess of PRB 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 2004 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, p. 3-105). We use the midpoints of these ranges for the base case analysis. Thus PRB $PM_{2.5}$ 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 sensitivity analyses, we examine the impact of assuming (1) a constant background set at the lower and upper end of the range of estimated background levels provided in the 2004 PM CD for the Eastern and Western United States, depending on the assessment location (see s_2 in Exhibit 2.1), and (2) varying daily $PM_{2.5}$ background, using distributions whose means are equal to the values used in the base case analysis and whose distributions are based on an analysis of $PM_{2.5}$ data from relatively remote sites with the sulfate component removed. (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.

The 2004 PM CD (p. 3-83) estimates background annual average $PM_{10-2.5}$ to be approximately $\leq$1 to 9 $\mu g/m^3$ in the East and $\leq$1 to 7 $\mu g/m^3$ in the West. We use 4.5 $\mu g/m^3$ as the estimated PRB for $PM_{10-2.5}$ in the base case analysis for the Eastern coarse risk assessment locations (i.e., Detroit, and St. Louis) and 3.5 $\mu g/m^3$ for Seattle. In a sensitivity analysis, we examine the impact of assuming a constant background set at the lower and upper end of the range of estimated background levels based on the 2004 PM CD (see s_2 in Exhibit 2.1).

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

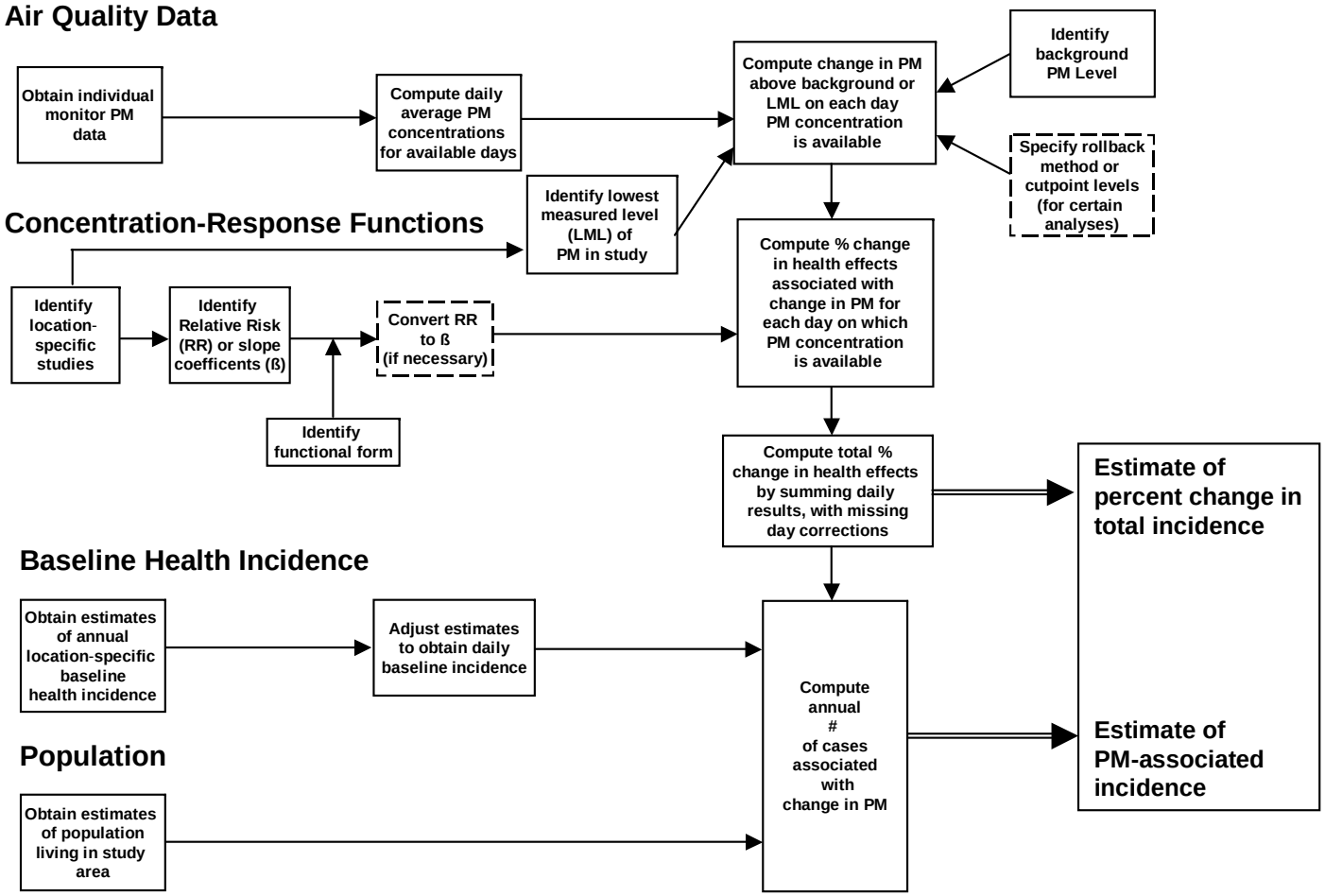
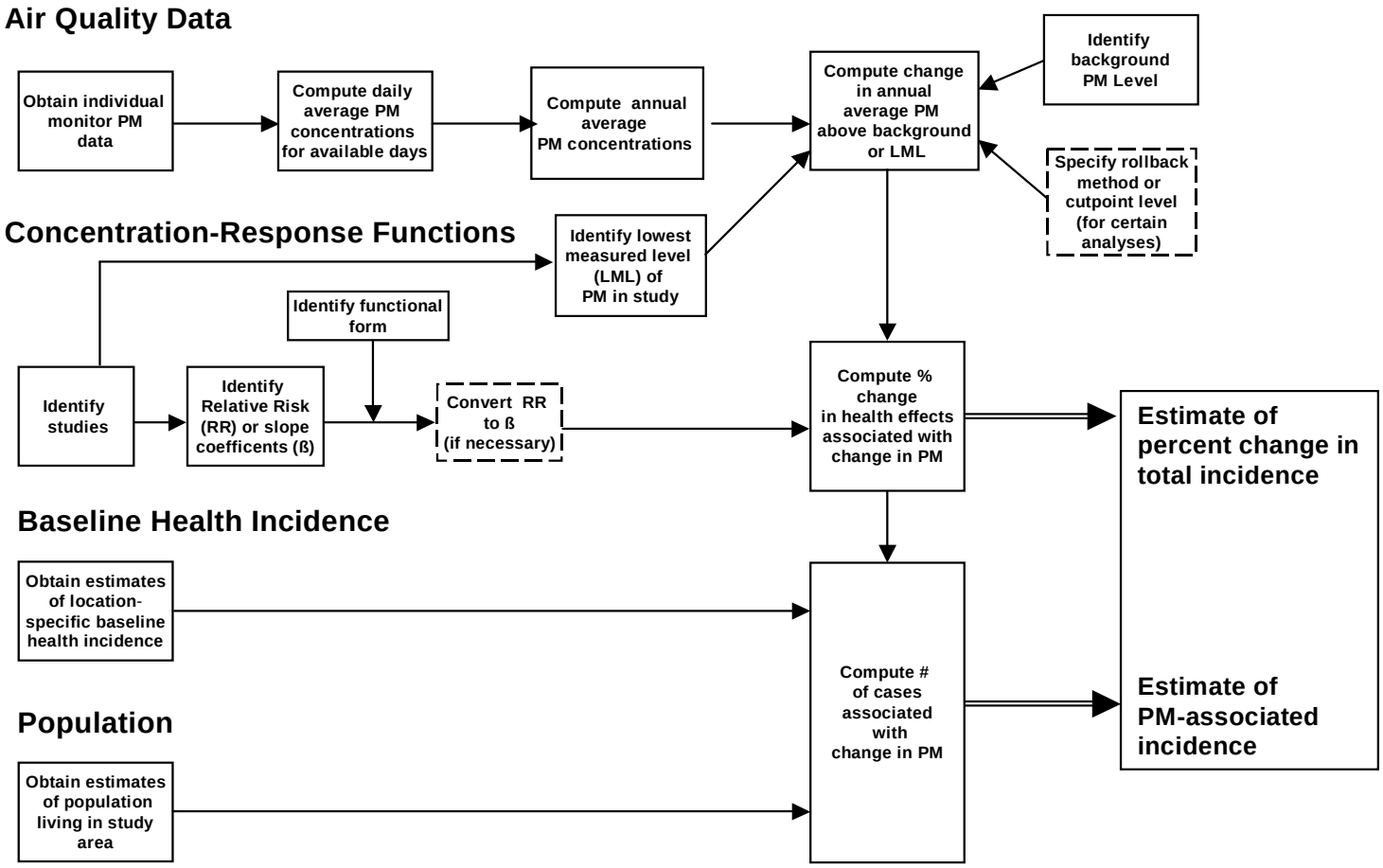


Exhibit 2.3 Flow Diagram of Risk Analyses for Long-Term Exposure Studies



2.2.2 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 generally 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 deemed most appropriate for the risk assessment. Consistent with the approach used in the prior PM risk assessment, a composite monitor data set was created for each assessment location based on a composite of all monitors eligible for comparison with the annual standard with at least 11 observations per quarter.[5] At the time of the prior 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 Air Quality System (AQS) beginning with the year 1999. There were sufficient $PM_{2.5}$ data in AQS for the year 2003 for all of the assessment locations except Phoenix, for which we used year 2001 data.

For the $PM_{10-2.5}$ risk assessment there were sufficient data from co-located monitors in the year 2003 for Detroit, St. Louis, and Seattle. As noted above, $PM_{10-2.5}$ 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 $PM_{10-2.5}$ concentrations that were calculated were negative. In Detroit, 10.4 percent of the days (12 days) for which $PM_{10-2.5}$ concentrations were calculated were negative[6]; in St. Louis, 1.7 percent (1 day) of the days were negative. There were no negative $PM_{10-2.5}$ concentrations calculated in Seattle.

The negative $PM_{10-2.5}$ values in a location will result in a slightly lower calculated annual average $PM_{10-2.5}$ concentration in that location. However, annual averages were not used in the calculation of risks and risk reductions associated with $PM_{10-2.5}$ concentrations, because all C-R functions included in the $PM_{10-2.5}$ risk assessment are short-term (daily) C-R functions. In addition, because values below background concentration don't contribute to risks from anthropogenic (above background) PM, such values aren't considered in the PM risk assessment. Because negative values are below background concentration, they too are not considered.

Appendix A summarizes the $PM_{2.5}$ 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 and 99th percentiles 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

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[5] Based on a review of the monitoring sites included by State air pollution agencies in the classification/designation process for $PM_{2.5}$, which follow the guidance set forth in Part 58 of the CFR, 1 monitoring site in St. Louis and 1 monitoring site in Boston were excluded from consideration for the $PM_{2.5}$ risk assessment.

[6] Based on a review of the monitoring sites used in the Ito (2003) study in Detroit, we selected the two $PM_{10-2.5}$ sites that were closest to those used in the original health effects study.

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 PM standards. 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 nearest 1 $\mu g/m^3$) 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, subject to meeting certain criteria detailed in Part 58 of the CFR, 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 $PM_{2.5}$ 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 current and alternative standards is determined by the $PM_{2.5}$ annual and daily design values. The annual design value (in $\mu g/m^3$) was calculated as follows:

- At each monitor, the annual average $PM_{2.5}$ concentration was calculated for each of the years 2001, 2002, and 2003, and these three annual average concentrations were then averaged.
- The maximum of these monitor-specific 3-year averages of annual averages is the annual design value, denoted dv_{annual};

The 98^{th} (99^{th}) percentile design value (in $\mu g/m^3$) was similarly calculated as follows:

1. At each monitor, the 98^{th} (99^{th}) percentile $PM_{2.5}$ concentration was calculated for each of the years 2001, 2002, and 2003, and these three 98^{th} (99^{th}) percentile concentrations were then averaged.

2. The maximum of these monitor-specific 3-year averages of 98th (99th) percentile concentrations is the daily 98th (99th) percentile design value, denoted $dv_{daily\ 98}$ ($dv_{daily\ 99}$).

Although the design values are based on monitor-specific values, the changes in $PM_{2.5}$ to simulate just meeting the specified standards are made at the composite monitor rather than at the individual monitors.

The method used to simulate just meeting alternative $PM_{2.5}$ or $PM_{10-2.5}$ standards was analogous to the method used to simulate just meeting the current $PM_{2.5}$ standards. Daily $PM_{2.5}$ or $PM_{10-2.5}$ concentrations were changed at a "composite monitor." The percent reduction of the $PM_{2.5}$ concentration at the composite monitor each day resulting from just meeting an alternative $PM_{2.5}$ standard is determined by the $PM_{2.5}$ annual and daily design values. Because only daily standards are being considered for $PM_{10-2.5}$, the percent reduction of the $PM_{10-2.5}$ concentration at the composite monitor each day resulting from just meeting an alternative $PM_{10-2.5}$ standard is determined by the daily design values for $PM_{10-2.5}$, which were calculated in the same way as the $PM_{2.5}$ daily design values. The annual, daily 98th percentile, and daily 99th percentile design values used in assessing the current and alternative standards for $PM_{2.5}$ are given in Exhibit 2.4. The daily 98th and 99th percentile design values used in assessing alternative standards for $PM_{10-2.5}$ are given in Exhibit 2.5.

Exhibit 2.4 EPA Design Values for Annual and 98th and 99th Percentile Daily $PM_{2.5}$ Standards*

Location	Annual	98th Percentile Daily	99th Percentile Daily
Boston	14.4	44	60
Detroit	19.5	44	48
Los Angeles	23.6	62	96
Philadelphia	16.4	51	89
Phoenix	11.5	35	41
Pittsburgh	21.2	63	70
St. Louis	17.5	42	46
San Jose	14.6	47	53
Seattle	11.1	41	48

*The calculation of design values is explained in Section 2.3 above. All design values are in $\mu g/m^3$. While the current daily standard is specified as a 98th percentile form, the 99th percentile form also is included to allow consideration of alternative standards with this alternative form.

Exhibit 2.5 EPA Design Values for 98th and 99th Percentile Daily $PM_{10-2.5}$ Standards*

Location	98th Percentile Daily	99th Percentile Daily
Detroit	70	77
St. Louis	33	47
Seattle	31	39

*The calculation of design values is explained in Section 2.3 above. All design values are in $\mu g/m^3$.

There are many possible ways to create an alternative distribution of daily concentrations that just meets specified $PM_{2.5}$ (or $PM_{10-2.5}$) 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 an 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 and alternative $PM_{2.5}$ 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 $PM_{10-2.5}$ historical air quality data are substantially more sparse and are insufficient to support an analysis analogous to that carried out for $PM_{2.5}$. In the absence of a clearly preferable alternative, we used the same proportional rollback method to simulate just meeting alternative $PM_{10-2.5}$ standards. The uncertainty introduced by this approach in the absence of empirical evidence supporting it is discussed more fully in Section 6.

The percent reduction required to meet a standard (annual, ninety-eighth percentile daily or ninety-ninth percentile daily) was determined by comparing the design value for that standard with the level of the standard. Because pollution abatement methods are applied largely to anthropogenic sources of $PM_{2.5}$ and $PM_{10\text{-}2.5}$, rollbacks were applied only to $PM_{2.5}$ and $PM_{10\text{-}2.5}$ above estimated background levels. The percent reduction was determined by the controlling standard. For example, suppose both an annual and a ninety-eighth percentile 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 annual design value down to the annual standard). Suppose p_d is the percent reduction required to just meet the ninety-eighth percentile daily standard (i.e., the percent reduction of daily $PM_{2.5}$ above background necessary to get the ninety-eighth percentile daily $PM_{2.5}$ design value down to the ninety-eighth percentile 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. 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

$$p_a = 1 - \frac{(std_a - b)}{(dv_{annual} - b)}$$

where b denotes background.

2. 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 (e.g., 98th percentile) standard (denoted $std_{d\,98}$) is

$$p_{d\,98} = 1 - \frac{(std_{d\,98} - b)}{(dv_{daily\,98} - b)}$$

Let p_{max} = maximum of (maximum of p_a and $p_{d\,98}$) and zero.[7]

[7] 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.

3. 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

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

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 results of a sensitivity analysis for 3 urban areas based on the percent rollbacks that would have resulted from using this alternative approach (see Section 8).

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:[8]

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

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.

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:

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

[8] 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).

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:

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

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). Currently, there is insufficient information to adequately adjust for the impact of longer-term exposure on mortality associated with $PM_{2.5}$ 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 PRB 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 PRB PM concentration in that location. The second part considered the differences in health effects incidence associated with differences between "as is" $PM_{2.5}$ and $PM_{10-2.5}$ concentrations and $PM_{2.5}$ and $PM_{10-2.5}$ concentrations that just meet the current standards (for $PM_{2.5}$) and alternative standards.

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 PRB 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 or alternative $PM_{2.5}$ standards (or alternative $PM_{10-2.5}$ 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 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.[9] 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

[9] 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.

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 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 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. Most 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 2003 air quality data for most of the urban locations, and the most recent available mortality baseline incidence rates (from 2001). 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.[10] 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),
  - 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.[11]

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,

[10] 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.

[11] 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.

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 not, for example, reflect the uncertainty concerning whether the PM coefficients in the study location and the assessment location are the same.[12]

- Sensitivity analyses were conducted to illustrate the effects of changing key default assumptions on the results of the assessment.

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 above background or the LML in the studies), 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;

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

- 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 preferable for illustrating the impact on results of using different input component values. Exhibit 2.6 lists the sensitivity analyses that were conducted.

Exhibit 2.6 Sensitivity Analyses

Analysis Number (Exhibit 2.1)	PM Indicator	Component of the Risk assessment	Sensitivity Analysis
1	$PM_{2.5}$	Air Quality	A sensitivity analysis of the effect of assuming a constant background $PM_{2.5}$ level versus different daily background levels
2	$PM_{2.5}$, $PM_{10-2.5}$	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 sensitivity analysis of the effect of just meeting the current annual $PM_{2.5}$ standard of 15 $\mu g/m^3$ using the maximum versus the spatial average of monitor-specific averages
5	$PM_{2.5}$	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	$PM_{2.5}$, $PM_{10-2.5}$	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 2004 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 prior 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 prior 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 2004 PM CD (see section 3 of the 2005 PM SP (U.S. EPA, 2005)). Tables 8A and 8-B in Appendices 8A and 8B of the 2004 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. Section 8.2.3 of the 2004 PM CD (U.S. EPA, 2004) summarizes the available U.S. and Canadian studies that provide effect estimates for $PM_{2.5}$ and other PM indicators for long-term exposure. As discussed in the 2005 PM SP (U.S. EPA, 2005, section 4.2.2.1), 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 2004 PM CD (U.S. EPA, 2004, section 9.2.2), for the relationship between PM and various health outcomes

"...considering results from studies conducted both within and outside the U.S. and Canada, the epidemiological evidence is strong for associations between PM_{10} and $PM_{2.5}$ and mortality, especially for total and cardiovascular mortality. The magnitudes of the associations are relatively small, especially for the multi-city studies. However, there is a pattern of positive and often statistically significant associations across studies for cardiovascular and respiratory health outcomes, including mortality and hospitalization and medical visits for cardiovascular and respiratory diseases, with PM_{10} and $PM_{2.5}$. The few available $PM_{10-2.5}$ studies also provide some evidence for associations between hospitalization for cardiovascular and respiratory

diseases with $PM_{10-2.5}$. ... For $PM_{10-2.5}$, the evidence for association with mortality is more limited." (U.S. EPA, 2004, p.9-32)

With respect to the relationship between long-term exposure to $PM_{2.5}$ and increased mortality, the 2004 PM CD placed the greatest weight on the results of the ACS and Six Cities cohort studies and concluded that "the results of these studies, including the reanalyses results for the Six Cities and ACS studies and the results of the ACS study extension, provide substantial evidence for positive associations between long-term ambient (especially fine) PM exposure and mortality." (U.S. EPA, 2004, p.9-33) The 2004 PM CD (p. 9-34) finds no evidence for associations between long-term exposure to $PM_{10-2.5}$ and either morbidity or mortality health outcomes.

The 2004 PM CD(pp. 9-50 - 9-79) contains an extensive discussion considering both the extent to which the available epidemiological evidence shows associations in the same location with a range of logically linked health endpoints and the extent to which the available toxicological evidence and mechanistic information provides support for the plausibility of the observed epidemiological associations. Based on that review, the 2004 PM CD concludes that,

> "A growing body of evidence both from epidemiologic and toxicologic studies also supports the general conclusion that $PM_{2.5}$ (or one or more $PM_{2.5}$ components), acting alone and/or in combination with gaseous co-pollutants, are likely causally related to cardiovascular and respiratory mortality and morbidity. The strength of the evidence varies across such endpoints, with relatively stronger evidence of associations with cardiovascular than respiratory endpoints, potentially due to reduced statistical power where respiratory outcomes are seen less frequently than cardiovascular outcomes. In addition, mortality associations with long-term exposures to $PM_{2.5}$, in conjunction with evidence of associations with short-term exposures, provide strong evidence in support of a causal inference." (U.S. EPA, 2004, p. 9-79)

Based on its review of the evidence evaluated in the 2004 PM CD, OAQPS included in both the $PM_{2.5}$ and $PM_{10-2.5}$ risk assessments the following broad categories of health endpoints associated with short-term exposures:

- hospital admissions for cardiovascular and respiratory causes;[13] and
- respiratory symptoms not requiring hospitalization.

[13] 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 in the risk assessment.

In addition, non-accidental, cardiovascular, and respiratory mortality due to short-term exposure, as well as total, cardiopulmonary, and lung cancer mortality due to long-term exposure are also 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 2005 PM SP (U.S. EPA, 2005).

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., 2000) suggests there may be 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 $PM_{2.5}$ air quality data if it had at least one $PM_{2.5}$ monitor at which there were at least 11 observations per quarter for a one year period and there were at least 122 observations per year (1 in 3 day monitoring). 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.[14]

- 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).[15]

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[14] We excluded from consideration a few monitors sited in industrial areas that are intended to characterize local conditions near major point sources and which met the EPA criteria contained in Part 58 of the CFR for exclusion from consideration when evaluating whether an area meets the current annual average $PM_{2.5}$ standard .

[15] Urban locations for which C-R functions were estimated sometimes 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.

- For the hospital admission effects category, relatively recent area-specific baseline incidence data, specific to International Classification of Disease (ICD) codes, are available.[16]

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 8-A of the 2004 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.[17] 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 2004 PM CD describes the use of 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 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.[18] 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 (Knoxville, TN and Portage, WI) were excluded based on the air quality

[16] 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.

[17] Tolbert et al. (2000) was excluded from consideration because it presented only preliminary results, and the 2004 PM CD urged caution in interpreting these preliminary results.

[18] 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.

data available in 2003, 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.

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 $PM_{10-2.5}$ 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 the $PM_{2.5}$ risk assessment.

Based on these considerations, we included Detroit, Seattle, and St. Louis in the $PM_{10-2.5}$ risk assessment. 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 $PM_{2.5}$ or $PM_{10-2.5}$ risk assessment had to satisfy the following criteria:

- It is an acceptable, published, peer-reviewed study that has been evaluated in the 2004 PM CD and judged adequate by EPA staff for purposes of inclusion in this risk assessment based on that evaluation.

- It directly measured PM using $PM_{2.5}$ or $PM_{10-2.5}$ as the indicator or for $PM_{2.5}$ was estimated using nephelometry data.[19]

- 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 $PM_{2.5}$, and Exhibit 3.4 for $PM_{10-2.5}$. More detailed information on the studies used is given in Appendix C.

[19] Consistent with advice received from members of the CASAC PM Panel, we have included 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]		
Philadelphia, PA		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	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	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	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	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	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	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	Pope et al. (2002) – ACS extended
Seattle, WA*	Pope et al. (2002) – ACS extended	Pope et al. (2002) – ACS extended	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	Pope et al. (2002) – ACS extended

*Seattle's annual average $PM_{2.5}$ level was lower than the LML of Krewski et al. (2000) – ACS.

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 $PM_{10-2.5}$ 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 8A and 8B in Appendices 8A and 8B of the 2004 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).

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 2005 PM SP (U.S. EPA, 2005, see Figure 3-3), 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. As stated in the 2005 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 differing levels of the gaseous pollutants"(U.S. EPA, 2005, section 3.6.4). 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 weak influence of gaseous co-pollutants on the PM_{10} effect size estimates" (U.S. EPA 2004, p.8-35). The authors concluded that their finding "suggests that the effect of PM_{10} is robust to the inclusion of other pollutants" (Samet et al., 2000, 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 2004 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, inclusion of multiple pollutants in models often produces unstable and statistically insignificant effect estimates for both PM and the co-pollutants (U.S. EPA, 2004, p.8-339). The CD also notes, on the other hand, that omission of potentially-contributing pollutants may incorrectly attribute some of their independent effects to PM (U.S. EPA, 2004, p. 8-339) Given that single and multi-pollutant models each have both potential advantages and disadvantages, with neither type clearly preferable over the other in all cases, we report risk estimates based on both single and multi-pollutant models where both are available.

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 2004 PM CD (section 8.4.4) and 2005 PM SP (U.S. EPA, 2005, sections 3.5.5.2 and 4.2.6.3). The 2004 PM CD points out that

> In considering the results of models for a series of lag days, it is important to consider the pattern of results that is seen across the series of lag periods. If there is an apparent pattern of results across the different lags, ... then selecting the single-day lag with the largest effect from a series of positive associations is reasonable, although it is, in fact, likely to underestimate the overall effect size (since the largest single-lag day results do not fully capture the risk also distributed over adjacent or other days).(U.S. EPA 2004, p.8-270)

As discussed in the 2004 PM CD, a number of the $PM_{2.5}$ short-term exposure mortality studies reported stronger associations with shorter lags, with a pattern of results showing larger associations at the 0- and 1-day lag period that taper off with successive lag days for varying PM indicators. These included the following studies which are used in the $PM_{2.5}$ risk assessment presented in this report: Moolgavkar (2003) and Mar et al. (2000), reanalyzed in Mar et al. (2003). Several studies included in the $PM_{2.5}$ risk assessment used only 0- and 1-day lags in the analyses for $PM_{2.5}$ (for example, Schwartz et al., 1996; Lipfert et al., 2000; Klemm et al., 2000).

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). The 2004 PM CD makes the point that "if one chooses the most significant single-lag day only, and if more than one lag day shows positive (significant or otherwise) associations with mortality, then reporting a RR for only one lag would also underestimate the pollution effects" (U.S. EPA 2004, p.8-279). Because of this, a distributed lag model is considered preferable to a single lag model where there is a consistent pattern of effects shown across several days. Unfortunately, distributed lag models have been estimated in only a few cases (e.g., Schwartz, 2000b for PM_{10}). Distributed lag models available for PM_{10} were included in the August 2003 PM_{10} risk assessment. However, there are currently no distributed lag models available for the studies and health endpoints included in the $PM_{2.5}$ or $PM_{10-2.5}$ risk assessment presented in this report.

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) based on the assessment in the 2004 PM CD and section 3.6.5.2 of the 2005 PM SP. Based on a review of the U.S. and Canadian studies reporting mortality effects associated with PM exposure, the 2004 PM CD states, "These studies reported stronger associations with shorter lags, with a pattern of results showing larger associations at the 0- and 1-day lag period that taper off with successive lag days for varying PM indicators ..." (2004 PM CD, p.8-273). 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 2004 PM 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 for this health endpoint (2004 PM CD, p. 8-279). 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.

We also conducted a sensitivity analysis examining the potential impact of using a distributed lag approach for short-term exposure mortality associated with $PM_{2.5}$, 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, 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.[20] Although this approach does not address the issue of understated standard errors of coefficient estimates, this is probably not a significant drawback. The 2004 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" (2004 PM CD, p. 9-35).

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 2004 PM CD.

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[20] 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 in the base case results.

In Los Angeles, Moolgavkar (2003) used several alternative estimation approaches and lag structures. We included a much wider array of models for this urban location in a sensitivity analysis for "as is" $PM_{2.5}$ concentrations in Section 7 (see Exhibit 7.11) 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 the same lag models noted above. 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); and 0- and 1-day lag models for both cardiovascular and respiratory hospital admissions. Given the inconsistent pattern observed for different lags for COPD mortality in this study, we did not include risk estimates for this endpoint in Los Angeles in either the base case or sensitivity analyses.

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 2004 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 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 included in the $PM_{2.5}$ risk assessment.

More recently, 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. As discussed more fully in Section 8.2.3.2.2 of the 2004 PM CD, the 2002 study has a number of advantages over previous analyses, including: doubling the follow-up time and tripling the number of deaths, expanding the ambient air pollution data to include two recent years of $PM_{2.5}$ data, improving the statistical adjustment for occupational exposure, incorporating data on dietary factors believed to be related to mortality, and using more recent developments in nonparametric spatial smoothing and random effects modeling. Recently, the Health Effects Subcommittee (HES) of the Science Advisory Board's (SAB) Clean Air Act Compliance Council indicated its preference that EPA use the results from this study rather than the results from the Krewski et al. (2000) ACS and/or Six Cities analyses to represent base case estimates for long-term exposure mortality associated with $PM_{2.5}$ concentrations for the purposes of benefits analyses (SAB, 2004). Two periods of $PM_{2.5}$ measurements were considered in the ACS-extended 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. The HES recommended that EPA use the results based on the average of the two periods from this study as representing the best estimates. The HES stated that this choice "may serve to reduce measurement error" (SAB, 2004). We therefore selected the corresponding C-R functions based on $PM_{2.5}$ measurements averaging the air quality data from the two periods to be included in the current $PM_{2.5}$ risk assessment. We note that the relative risks reported by Pope et al. (2002) corresponding to the earlier period (1979-1983) were somewhat smaller (RR = 1.04) than the relative risk reported for either the 1999-2000 or average of the two periods (RR = 1.06).

Two other PM cohort studies that are discussed in the 2004 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 26,000 middle-aged male veterans who were, at the time of recruitment, mildly to moderately hypertensive and having a very high percentage of prior smoking. The 2004 PM CD presents a comparison of the study designs and results (see section 8.2.3.2.5) and concludes:

> In considering the results of these studies together, statistically significant associations are reported between fine particles and mortality in the ACS and Six Cities analyses, inconsistent but generally positive associations with PM were reported in the AHSMOG analyses, and distinctly inconsistent results were reported in the VA study. Based on several factors, the larger study population in the ACS study, the larger air quality data set in the Six Cities study, the more generally representative study populations used in the Six Cities and ACS studies, and the fact that these studies have undergone extensive reanalyses – the greatest weight should be placed on the results of the ACS and Six Cities cohort studies in assessing relationships between long-term PM exposure and mortality.(U.S. EPA 2004, pp.8-120 to 8-121)

Based on this assessment, for purposes of the quantitative risk assessment, only the results of the ACS and Six Cities cohort studies have been included. The Veteran's study, which did not find any positive associations between indicators of long-term exposures to PM, and the Seventh Day Adventist study, which reported some positive but not statistically significant associations for males with long-term exposure to $PM_{2.5}$ are discussed in greater detail in the 2004 PM CD (U.S. EPA, 2004) and the 2005 PM SP (U.S. EPA, 2005).

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;

- based on the evaluation of the issue of selecting appropriate lags in the 2004 PM CD, if only single lag models were available, 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, where there was a consistent pattern across these lags. 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 one city (Los Angeles), we included alternative approaches to estimating the C-R function (e.g., use of GLM) in combination with the preferred lags discussed above to illustrate the effects of these alternative model specifications on the risk estimates.

- For long-term exposure mortality, the most recently published 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).[21] 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 and 5.2 for the $PM_{2.5}$ 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.

[21] 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]	3,072,000		---	1,684,000 (55%)	---	359,000 (12%)	---	---
Pittsburg[6]	1,281,666		---	814,000 (64%)	---	---	1,166,000 (91%)	116,000 (9%)
San Jose[7]	1,683,000		---	965,000 (57%)	---	---	---	---
Seattle[8]	1,737,000		---	1,044,000 (60%)	1,555,000 (90%)	---	---	---
St. Louis[9]	2,518,000	307,000 (12%)	1,637,000 (65%)	1,475,000 (59%)	---	---	---	---

[a] Total population and age-specific population estimates taken from the CDC Wonder website 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 $PM_{10-2.5}$ Risk Assessment Locations

City	Population[a]			
	Total	Ages 7-14	Ages ≥ 65	Ages <65
Detroit[1]	2,061,000	---	249,000 (12%)	---
Seattle[2]	1,737,000	---	---	1,555,000 (90%)
St. Louis[3]	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] King County.
[3] 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 $PM_{2.5}$ risk assessment locations for 2001 from CDC Wonder, an interface for public health data dissemination from the Centers for Disease Control (CDC).[22] The mortality rates are derived from U.S. death records and U.S. Census Bureau post-censal population estimates, and are reported in Exhibit 5.3 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 2001 from CDC Wonder for comparison. The epidemiological studies used in the risk assessment reported causes of mortality using the ninth revision of the International Classification of Diseases (ICD-9) codes. However, the tenth revision has since come out, and baseline mortality incidence rates for 2001 shown in Exhibit 5.3 use ICD-10 codes. The groupings of ICD-9 codes used in the epidemiological studies and the corresponding ICD-10 codes used to calculate year 2001 baseline incidence rates is given in Exhibit 5.4.

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, Los Angeles, and Seattle. Year 2000 hospitalization data were obtained for Wayne County (Detroit) from the Michigan Health and 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. Finally, year 2000 hospitalization data were obtained for King County (Seattle) from the State of Washington

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

Department of Health, Center for Health Statistics, Office of Hospital and Patient Data Systems. These data are presented in Exhibits 5.5 and 5.6. 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).

Exhibit 5.3 Baseline Mortality Rates for 2001 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]	Seattle[9]	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-9 codes < 800	776	916	581	1070	---	---	494	869	---	791
Non-accidental (75+): ICD-9 codes < 800	---	---	---	---	---	761	---	---	---	469
Non-accidental (<75): ICD-9 codes < 800	---	---	---	---	---	399	---	---	---	322
Cardiovascular (all ages): ICD-9 codes: 390-459	---	416	---	---	---	---	206	---	---	328
Cardiovascular (all ages): ICD-9 codes: 390-448	---	---	---	418	---	---	---	---	---	324
Cardiovascular (65+): ICD-9 codes: 390-448	---	---	---	---	211	---	---	---	---	273
Cardiovascular (all ages): ICD-9 codes: 390-429	---	---	207	---	---	---	---	---	---	252

Health Effect	Boston[1]	Detroit[2]	Los Angeles[3]	Philadelphia[4]	Phoenix[5]	Pittsburgh[6]	San Jose[7]	St. Louis[8]	Seattle[9]	National Average
Ischemic Heart Disease (all ages): ICD-9 codes: 410-414	122	---	---	---	---	---	---	206	---	152
Respiratory (all ages): ICD-9 codes: 11, 35, 472-519, 710.0, 710.2, 710.4	---	---	---	---	---	---	51	---	---	80
Respiratory (all ages): ICD-9 codes: 460-519	---	72	---	---	---	---	---	---	---	79
COPD without Asthma (all ages): ICD-9 codes: 490-492, 494-496	36	---	---	---	---	---	---	39	---	42
COPD with Asthma (all ages): ICD-9 codes: 490-496	---	---	30	---	---	---	---	---	---	43
Pneumonia (all ages): ICD-9 codes: 480-487	26	---	---	---	---	---	---	27	---	22
B. Mortality Rates Used in Risk Analysis for Long-term Exposure Studies[b] (deaths per 100,000 general population/year)										
Total mortality (25+): ICD-9 codes: all	803	---	---	---	---	---	---	905	---	822

Health Effect	Boston[1]	Detroit[2]	Los Angeles[3]	Philadelphia[4]	Phoenix[5]	Pittsburgh[6]	San Jose[7]	St. Louis[8]	Seattle[9]	National Average
Total mortality (30+): ICD-9 codes: all	797	937	591	1100	676	1189	499	897	637	814
Cardiopulmonary Mortality (25+): ICD-9 codes: 400-440, 485-495	297	---	---	---	---	---	---	391	---	341
Cardiopulmonary Mortality (30+): ICD-9 codes: 401-440, 460-519	347	468	313	489	313	573	247	439	287	391
Lung Cancer Mortality (30+): ICD-9 code: 162	55	64	33	72	42	78	30	61	44	55

*The epidemiological studies used in the risk assessment reported causes of mortality using the ninth revision of the International Classification of Diseases (ICD-9) codes. However, the tenth revision has since come out, and baseline mortality incidence rates for 2001 shown in this exhibit use ICD-10 codes. The groupings of ICD-9 codes used in the epidemiological studies and the corresponding ICD-10 codes used to calculate year 2001 baseline incidence rates is given in Exhibit 5.4.

[a] Mortality figures were obtained from CDC Wonder for 2001. 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.

[9] King County.

Exhibit 5.4 ICD-9 Codes used in Epidemiological Studies and Corresponding ICD-10 Codes

Causes of Death	ICD-9 Codes	ICD-10 Codes
A. Causes of Death used in Short-Term Exposure Studies		
Non-accidental	<800	A00-R99
Cardiovascular	390-459	G45.0-G45.2, G45.4,G45.9, G54.0, G90.3, G93.6, G93.8, G95.1, I00-I13.9, I20.0-I22.9, I24.1-I64, I67.0-I87.9, I89.0-I95.9, I99, K66.1, K92.2, M21.9, M30.0-M31.9, R00.1, R00.8, R01.2, R58
Cardiovascular	390-448	G45.0-G45.2, G45.4-G45.9, G54.0, G93.6, G93.8, G95.1, I00-I13.9, I20.0-I22.9, I24.1-I64, I67.0-I78.9, M21.9, M30.0-M31.9, R00.1, R00.8, R01.2
Cardiovascular	390-429	I00-I13.9, I20.0-I22.9, I24.1-I51.9, I71.9, M21.9, R00.1, R00.8, R01.2
Ischemic Heart Disease	410-414	I20.0-I22.9, I23.6, I24.0-I24.9, I25.1-I25.9, M21.9
Respiratory	11, 35, 472-519, 710.0, 710.2, 710.4	A16.2, A16.4, A16.9, A46, A48.1, B05.2, B90.9, J65, J02.9, J03.9, J05.0, J10.0-J16.8, J18.0-J18.9, J20.9, J30.0-J32.9, J33.9-J34.1, J34.3-J39.8, J40-J64, J66.0-J94.9, J98.0-J98.9, M32.0-M32.9, M35.0, M33.2, P28.8, R09.1
Respiratory	460-519	J00-J01.9, J02.8-J02.9, J03.8-J64, J66.0-J94.9, J98.0-J98.9, P28.8, R06.5, R09.1
COPD without Asthma	490-492, 494-496	J20.9, J40-J44.9, J47, J67.0-J67.9, J98.0
COPD with Asthma	490-496	J20.9, J40-J47, J67.0-J67.9 J98.0
Pneumonia	480-487	A48.1, B05.2, J10.0-J18.9, J99.8
B. Health Effects used in Long-term Exposure Studies		
Total Mortality	all	all

Causes of Death	ICD-9 Codes	ICD-10 Codes
Cardiopulmonary Mortality	400-440, 485-495	G45.0-G45.2, G45.4-G45.9, G93.6, G93.8, G95.1, I10-170.9, I72.9, M21.9, R00.1, R00.8, R01.2, J10.0-J11.8, J18.0, J18.2-J18.9, J20.9, J40-J43.9, J44.1-J44.8, J45.0-J47, J67.0-J67.9, J98.0
Cardiopulmonary Mortality	401-440, 460-519	G45.0-G45.2, G45.4-G45.9, G93.6, G93.8, G95.1, I10-170.9, I72.9, M21.9, R00.1, R00.8, R01.2, A48.1, B05.2, J00-J01.9, J02.8-J02.9, J03.8-J64, J66.0-J94.9, J98.0-J98.9, P28.8, R06.5, R09.1
Lung Cancer Mortality	162	C33-C34.9, C39.8, C45.7

Exhibit 5.5 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 5.6 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. Schwartz and Neas (2000) is the only respiratory symptom study included in the PM risk assessment. This study estimated multi-city C-R functions using six cities, including Boston and St. Louis. The baseline incidence rates in this study are likewise based on all six cities combined. The C-R functions and the baseline incidence rates (for lower respiratory symptoms and cough) were used in Boston and St. Louis.

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, except in its use of design values which were based on the most recent three-year period available.

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 2001). 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 2005 PM SP (U.S. EPA, 2005). 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 $PM_{2.5}$ is likely causally related or, for $PM_{10-2.5}$ that the evidence is suggestive of a causal relationship.
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 hypothetical 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 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.
Adequacy of ambient PM monitors as surrogate for population exposure	Possible differences in how the spatial variation in ambient $PM_{2.5}$ levels across each urban area are characterized in the original epidemiological studies compared to the more recent ambient $PM_{2.5}$ data used to characterize current air quality would contribute to uncertainty in the health risk estimates. This would be expected to add even more uncertainty in the case of the $PM_{10-2.5}$ risk assessment where greater spatial variability in ambient monitoring data within an urban area has been observed.

Uncertainty	Comments
Adjustment of air quality distributions to simulate just meeting current $PM_{2.5}$ standards or alternative $PM_{2.5}$ or $PM_{10-2.5}$ standards	The pattern and extent of daily reductions in $PM_{2.5}$ concentrations that would result if current or alternative $PM_{2.5}$ 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. There is much greater uncertainty about the use of a proportional air quality adjustment procedure to simulate the daily distribution of ambient $PM_{10-2.5}$ concentrations upon just meeting alternative $PM_{10-2.5}$ standards due to the lack of sufficient $PM_{10-2.5}$ air quality data over time to evaluate the reasonableness of this assumption.
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 ranges estimated in the 2004 PM CD for $PM_{2.5}$ and $PM_{10-2.5}$ and of allowing daily $PM_{2.5}$ 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 2004 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 2004 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 $PM_{2.5}$ or, for $PM_{10-2.5}$, $PM_{2.5}$ and PM_{10} at co-located monitors, 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 in short-term exposure studies, 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 (2004 PM CD, p. 9-36). 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.

With respect to the $PM_{10-2.5}$ health risk assessment, the locations used in the risk assessment are not representative of urban areas in the U.S. that experience the most significant 24-hour peak $PM_{10-2.5}$ concentrations, and thus, observations about relative risk reductions associated with alternative standards may not be as relevant to the areas expected to have the greatest health risks associated with elevated ambient $PM_{10-2.5}$ levels. In considering the $PM_{10-2.5}$ risk estimates it also is important to recognize that there is a much smaller health effects database from which to obtain the C-R relationships used in this portion of the risk assessment, compared to that available for $PM_{2.5}$ and, thus, there is significantly greater uncertainty associated with the $PM_{10-2.5}$ risk estimates.

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. 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.

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 2004 PM CD (See section 8.2.2.4), growing evidence indicates that there are numerous potentially toxic PM components and some components may act in combination.

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 may also play a role in either causing or modifying health effects, either independently or in combination with PM (see section 8.1.3.2 in the 2004 PM CD). 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 differs 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 (referred to as the "lowest measured levels" (LML) in this report). Applying proportional rollbacks to the concentration distributions in the assessment locations may result in some modeled PM concentrations below the LML observed in the studies. In such cases, the difference in PM was taken to be the difference between the "as is" levels and the LML in the study. This procedure avoids extrapolating 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 the LML.

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.[23]

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 – $PM_{2.5}$ 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 2004 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 2004 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. 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.

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[23] 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.

As discussed in the draft PM Staff Paper (EPA, 2005; see section 5), the uncertainty related to exposure measurement error in epidemiologic studies linking health effects to $PM_{10-2.5}$ is potentially quite large, and this contributes to much larger uncertainty surrounding the $PM_{10-2.5}$ risk estimates included in this report. For example, in the Detroit study (Ito 2003) the $PM_{10-2.5}$ air quality values were based on air quality monitors located in Windsor, Canada. The study authors determined that the air quality values from these monitors were generally well correlated with air quality values monitored in Detroit, where the hospital admissions data were gathered, and thus concluded that these monitors were appropriate for use in exploring the association between $PM_{10-2.5}$ air quality and hospital admissions in Detroit. However, the $PM_{10-2.5}$ levels reported in this study are significantly lower than the $PM_{10-2.5}$ levels measured at some of the Detroit monitors in 2003 – an annual mean level of 13.3 $\mu g/m^3$ is reported in the study, based on 1992 to 1994 data, as compared to an average annual mean level of 21.7 $\mu g/m^3$ measured at two urban-center monitors in 2003 (which is used as the basis for the risk assessment). EPA staff evaluation comparing Windsor and Detroit monitoring data has shown that in recent years the Windsor monitors used in this study typically have recorded $PM_{10-2.5}$ levels that are generally less than half the levels recorded at urban-center Detroit monitors (EPA, 2005). EPA staff have concluded that the association observed in the Detroit study likely reflects exposure levels potentially much higher in the central city area than those reported in that study (EPA, 2005). Thus, there are concerns that the current $PM_{10-2.5}$ levels measured at ambient monitoring sites in recent years may be quite different from the levels used to characterize exposure in the original $PM_{10-2.5}$ epidemiologic studies based on monitoring sites in different locations, thus possibly over- or underestimating population risk levels.

PM air quality data were not available for all days of the year chosen for the risk assessment in many of the assessment locations.[24] 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 2003), 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

[24] $PM_{2.5}$ monitor data were available for all days in the year for two 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 $PM_{10-2.5}$.

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 $PM_{2.5}$ risk assessment, however, because adequate $PM_{2.5}$ monitoring data were available for all but one of the assessment locations in the year 2003, which is quite recent; $PM_{2.5}$ monitoring data were available in 2001 for Phoenix, AZ.

6.2.3 Simulation of reductions in $PM_{2.5}$ and $PM_{10-2.5}$ 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. The assumption that $PM_{2.5}$ concentrations will be reduced by the same percentage on all days is believed to be a reasonable approximation based on an evaluation of how $PM_{2.5}$ concentration distributions have changed historically in some areas (see Appendix B). There is, however, some uncertainty surrounding the predicted daily changes in $PM_{2.5}$ concentrations that would result if the current or alternative standards were just met, and consequently there is some uncertainty surrounding the associated daily changes in population health response. With respect to the $PM_{10-2.5}$ health risk estimates, there is much greater uncertainty about the reasonableness of the use of proportional rollback to simulate attainment of alternative $PM_{10-2.5}$ daily standards in any urban area due to the limited availability of $PM_{10-2.5}$ air quality data over time. This is one of several factors that contributes to the greater uncertainty associated with the $PM_{10-2.5}$ risk estimates.

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).[25] 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 (3-1) in Section 2.5:

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

[25] The exceptions to this are Lipfert et al. (2000), which reports linear C-R functions for cardiovascular mortality, and Schwartz and Neas (2000), which reports logistic functions for respiratory symptoms.

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 (Detroit, Los Angeles, 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 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 study reporting the C-R functions for those endpoints (Schwartz and Neas, 2000). 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-World War II 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.[26] 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

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

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.

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 (representing levels measured in 2003 for most of the assessment locations) in excess of an estimated PRB 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 $PM_{2.5}$ and Figure 7.7 for $PM_{10-2.5}$. Corresponding figures showing PM-related incidence are shown in Appendix D, in Figures D.1 through D.6 for $PM_{2.5}$ and Figure D.7 for $PM_{10-2.5}$. In addition, results are given for Detroit in Exhibits 7.1 and 7.2 for $PM_{2.5}$ 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. The central tendency estimates in all of the figures and exhibits are based on the PM coefficients estimated in the studies, and the ranges are based on the 95 percent confidence intervals (CIs) around those estimates. All estimated incidences were rounded to the nearest whole number, except respiratory symptoms, which were rounded to the nearest 100. 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 and 7.2 and Exhibits D.1 through D.8 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. For total non-accidental mortality associated with short-term exposure to $PM_{2.5}$, Figure 7.1 displays estimates for only six of the nine risk assessment locations because acceptable C-R functions for this health outcome were not available for the other three locations.

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. In some cases, the lower bound of a confidence interval falls below zero. This does not imply that additional exposure to PM has a beneficial effect, but only that

the estimated PM coefficient in the C-R function was not statistically significantly different from zero. Lack of statistical significance could mean that there is no relationship between PM and the health endpoint or it could mean that there wasn't sufficient statistical power to detect a relationship that exists.

Figure 7.2 shows estimated mortality and morbidity effects associated with short-term exposure to $PM_{2.5}$ based on C-R functions in which $PM_{2.5}$ was the only pollutant in the model versus C-R functions in which there was at least one additional pollutant included. There was no consistent pattern. For example, in Los Angeles the addition of CO to the model substantially decreased the $PM_{2.5}$ effect estimates for non-accidental mortality, cardiovascular hospital admissions, and COPD hospital admissions but increased the $PM_{2.5}$ effect estimate for cardiovascular mortality. In Pittsburgh and San Jose, the addition of single co-pollutants or a combination of co-pollutants only had a modest impact on the $PM_{2.5}$ effect estimates.

Figure 7.3 compares single vs. multi-city models. Only two assessment locations (Boston and St. Louis) had available PM coefficients based on both single and multi-city models, from the Six Cities study. In all cases, the confidence intervals for the estimates from the multi-city model were tighter than those for the single-city model estimates. In most, but not all cases, the central estimates for the single vs. multi-city models did not vary greatly.

Figure 7.4 shows the effect of different lag structures in the C-R function. Based on the discussion of selection of lags earlier in this report (see Section 4.3), estimates are shown for alternative lags dependent on the type of health endpoint. For non-accidental mortality in Los Angeles there is little difference in effect estimates considering lags ranging from 0 to 2 days and also little difference in effect estimates for cardiovascular mortality in this same location between 0-day and 1-day lags. For cardiovascular mortality in Phoenix the effect estimate from a 1-day lag model is larger than that from a 0-day lag model. However, this is insufficient evidence upon which to base any general conclusion about the lag structure between $PM_{2.5}$ and this health endpoint.

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

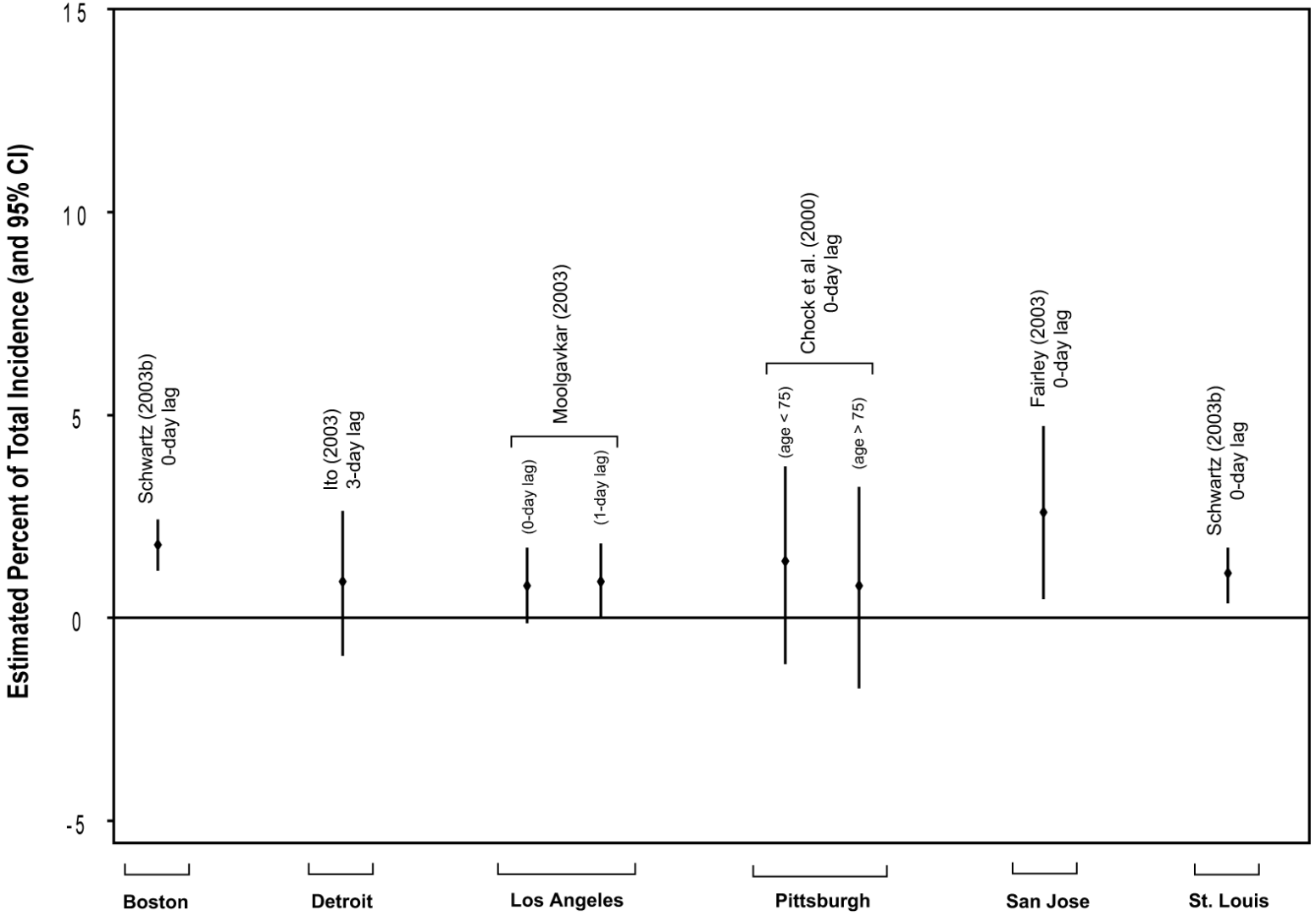


Figure 7.2. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-Pollutant versus Multi-Pollutant Models

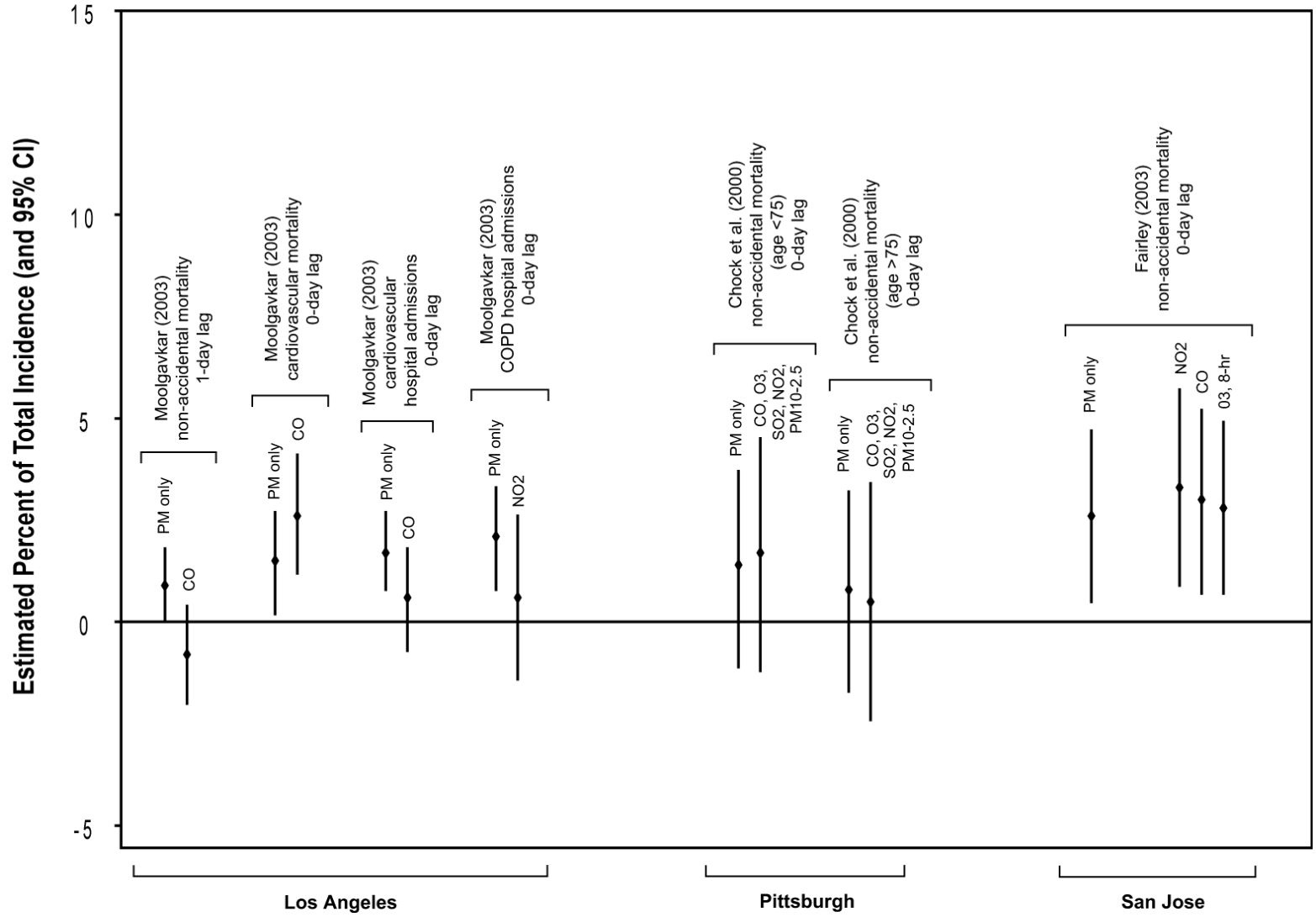


Figure 7.3. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models

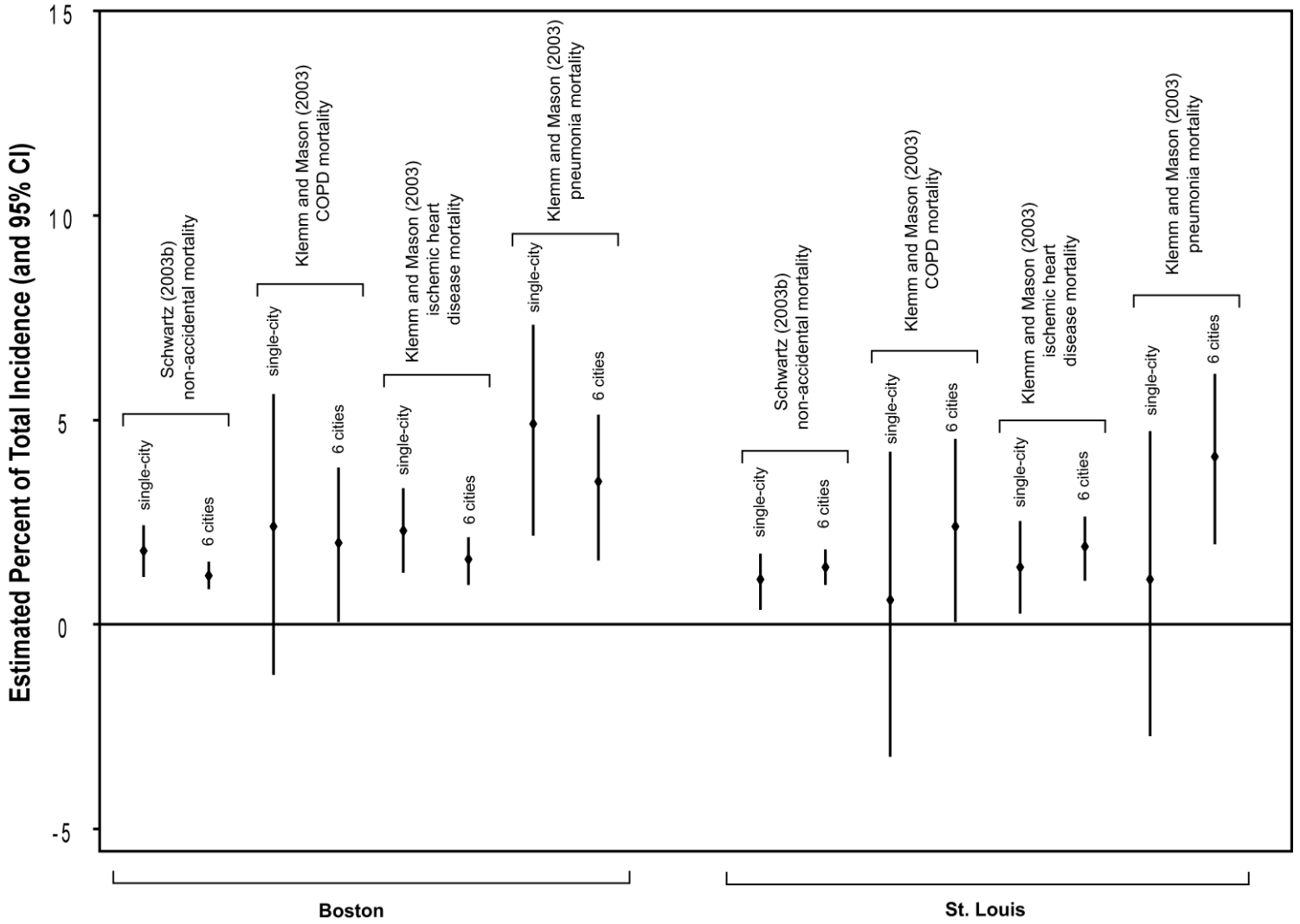


Figure 7.4. Estimated Annual Percent of Mortality Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Effect of Different Lag Models

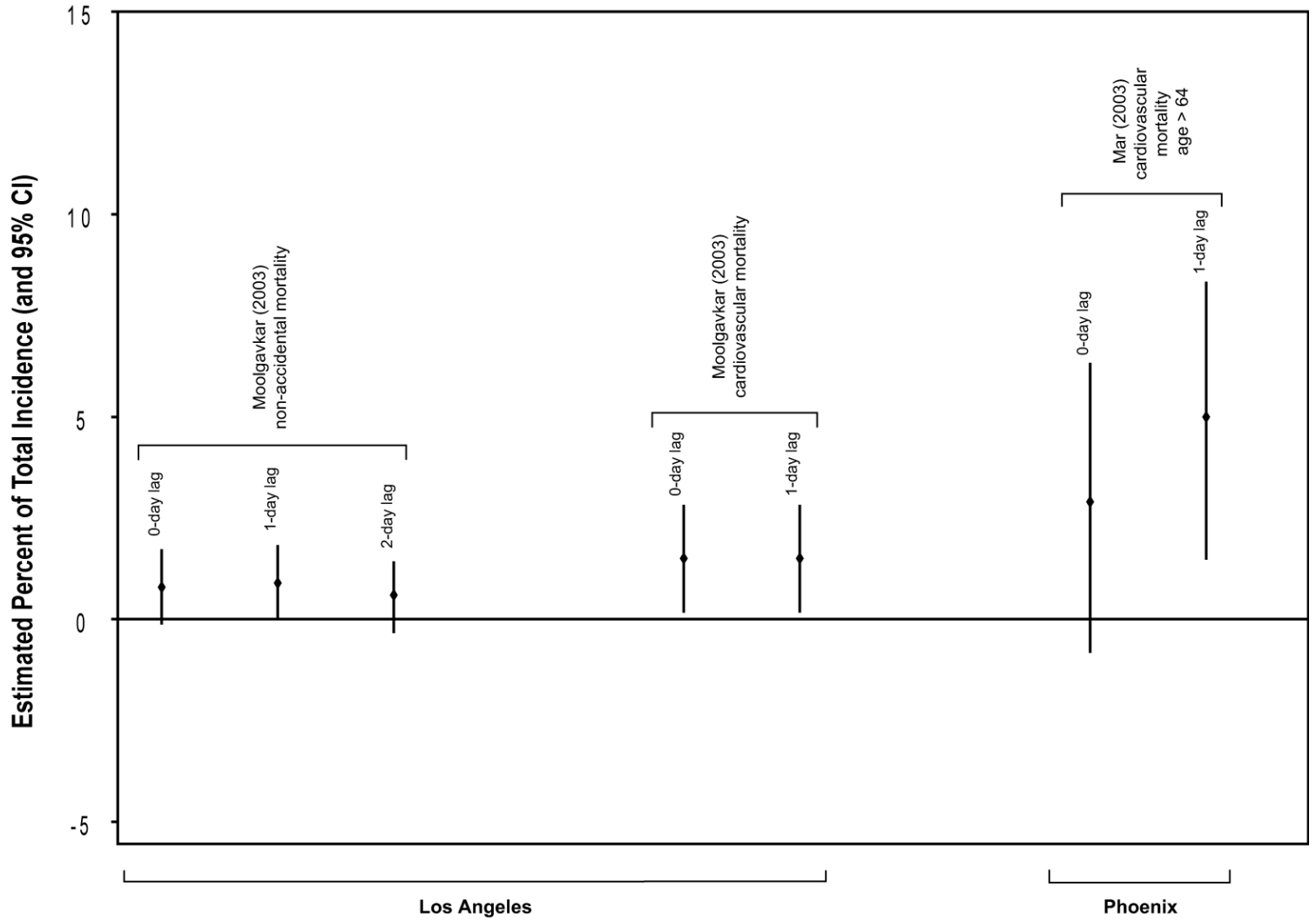


Figure 7.5. Estimated Annual Percent of Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant Models

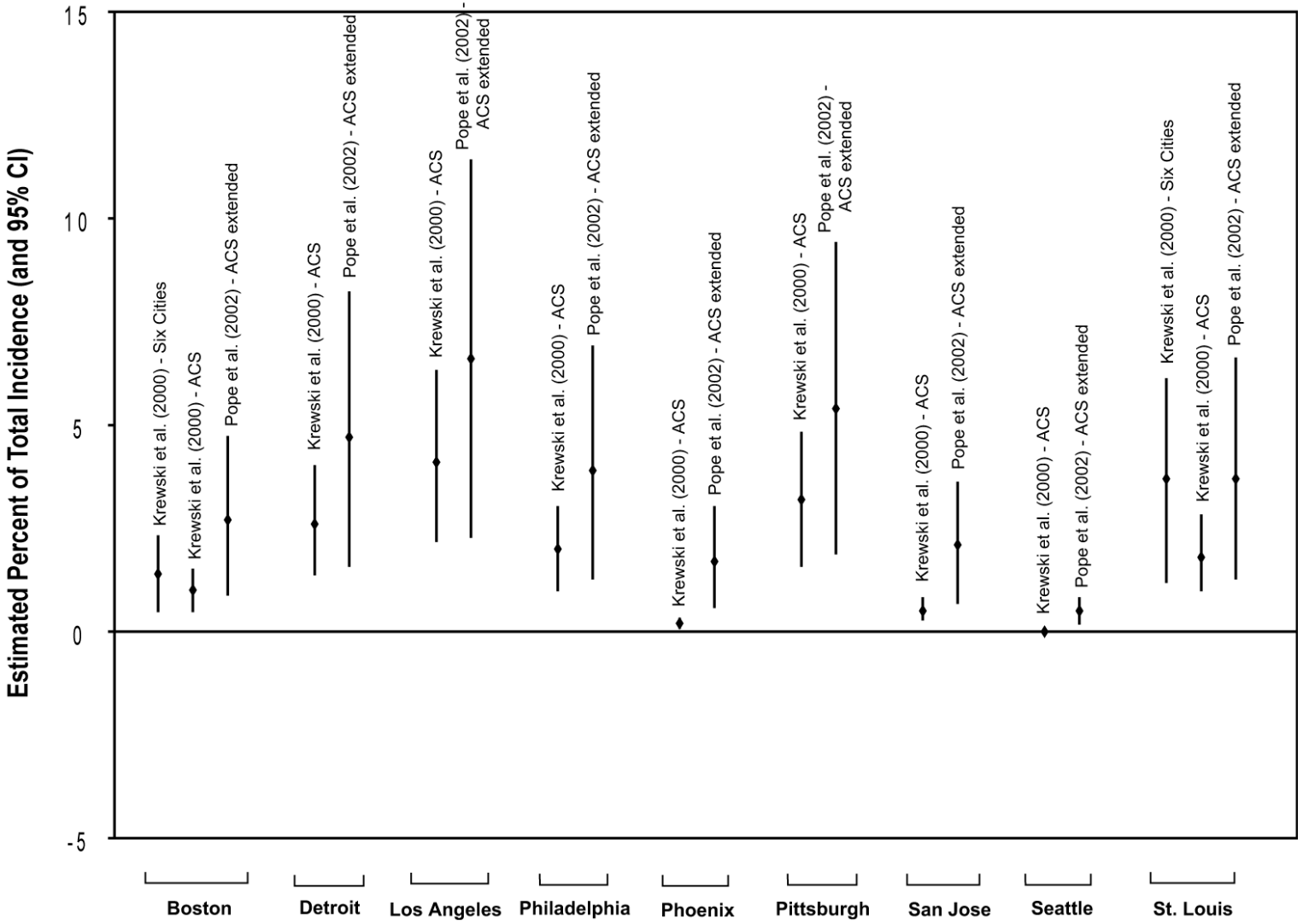
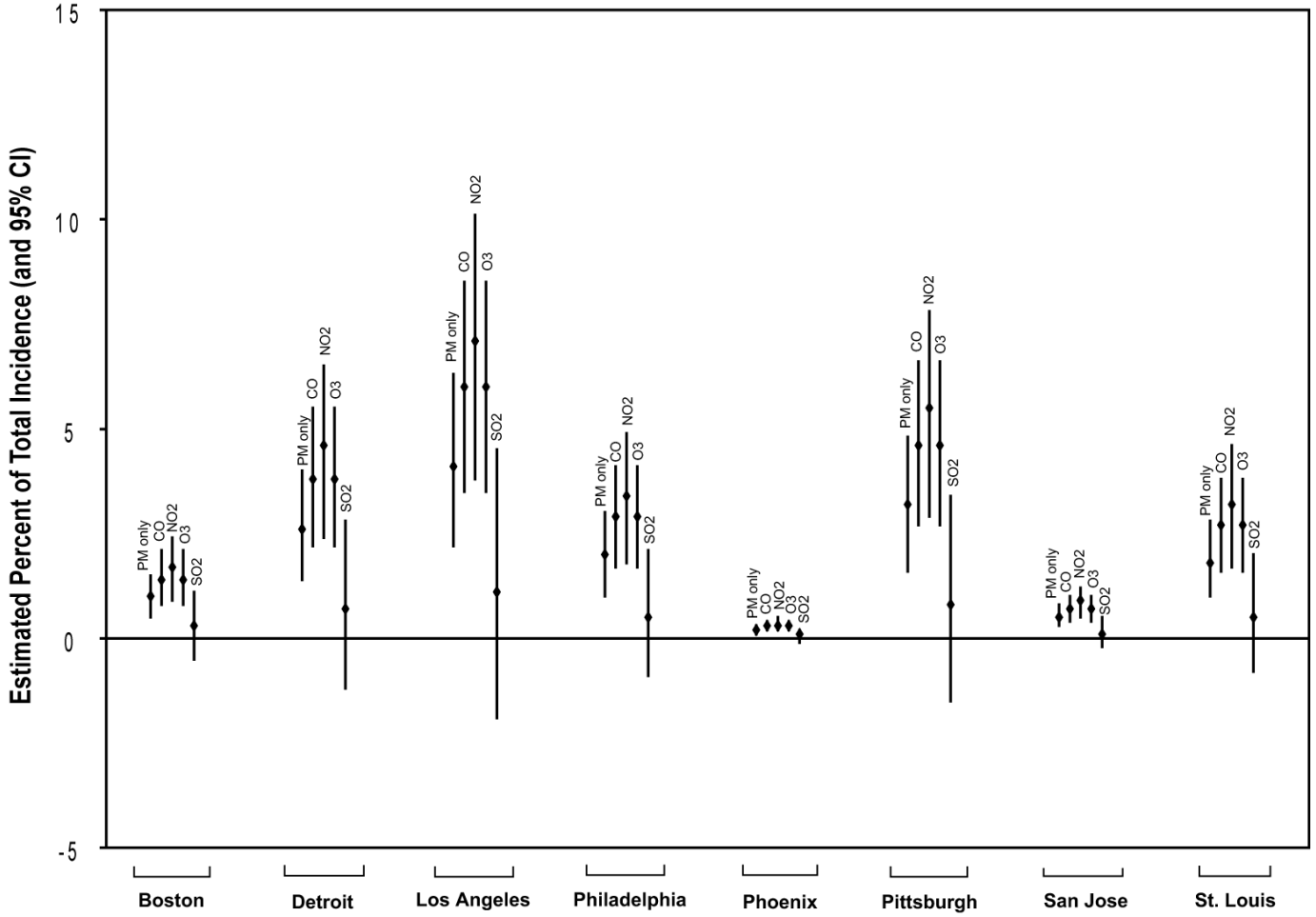


Figure 7.6. Estimated Annual Percent of Mortality Associated with Long-Term Exposure to $PM_{2.5}$ Above Background: Single-Pollutant and Multi-Pollutant Models*



*Based on Krewski et al. (2000) – ACS

Figure 7.7. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{10-2.5}$ Above Background

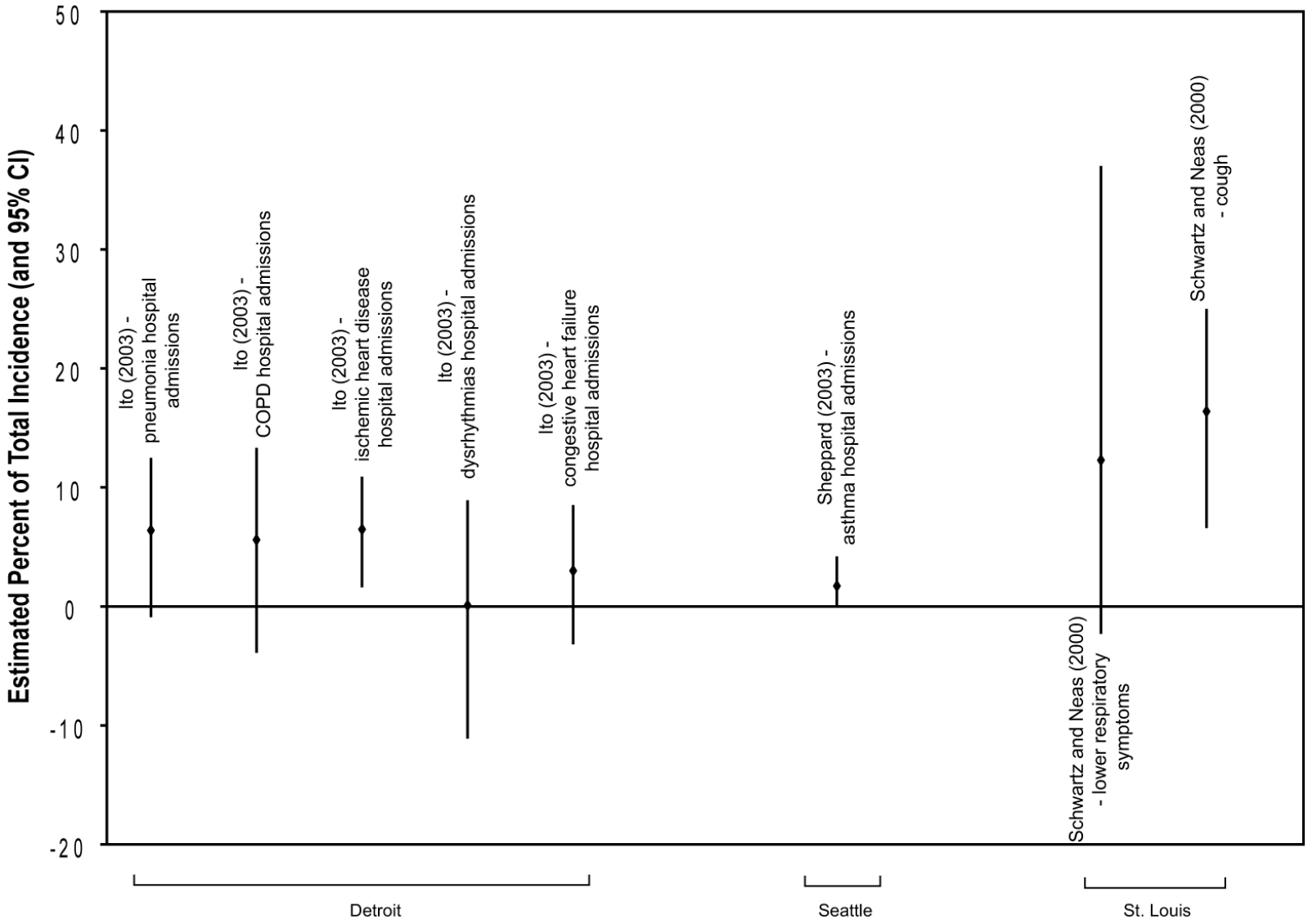


Exhibit 7.1. Estimated Annual Health Risks Associated with "As Is" $PM_{2.5}$ Concentrations
Detroit, MI, 2003

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Lowest Measured Level in Study or Policy Relevant 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		163 (-163 - 481)	0.9% (-0.9% - 2.6%)
	Single Pollutant Models (Cause-Specific Mortality)						
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	all	1 day		88 (-134 - 299)	1.0% (-1.6% - 3.5%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	all	0 day		16 (-80 - 102)	1.1% (-5.4% - 6.9%)
Long-Term Exposure Mortality	**Single Pollutant Models**						
	Krewski et al. (2000) - ACS	All cause	30+			506 (262 - 772)	2.6% (1.4% - 4.0%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+			508 (330 - 701)	5.3% (3.4% - 7.3%)
	Pope et al. (2002) - ACS extended	All cause	30+			906 (313 - 1592)	4.7% (1.6% - 8.2%)
	Pope et al. (2002) - ACS extended	Cardiopulmonary	30+			661 (232 - 1110)	6.9% (2.4% - 11.5%)
	Pope et al. (2002) - ACS extended	Lung cancer	30+			135 (42 - 207)	10.2% (3.2% - 15.7%)
	Multi-Pollutant Models						
	Krewski et al. (2000) - ACS	All cause	30+		CO	735 (427 - 1052)	3.8% (2.2% - 5.5%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	879 (467 - 1249)	4.6% (2.4% - 6.5%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	735 (427 - 1052)	3.8% (2.2% - 5.5%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	133 (-234 - 545)	0.7% (-1.2% - 2.8%)

Health Effects*	Study	Type	Ages	Lag	Other Pollutants in Model	Health Effects Associated with PM2.5 Above Lowest Measured Level in Study or Policy Relevant Background**	
						Incidence	Percent of Total Incidence
Hospital Admissions	**Single Pollutant Models**						
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	65+	1 day		231 (44 - 409)	4.5% (0.9% - 8.0%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD	65+	3 day		54 (-137 - 231)	1.4% (-3.5% - 5.9%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	65+	2 day		166 (-97 - 422)	1.7% (-1.0% - 4.2%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	65+	1 day		246 (45 - 439)	3.5% (0.6% - 6.3%)
	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	65+	1 day		48 (-109 - 193)	1.5% (-3.3% - 5.9%)

*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 policy relevant background level. Average policy relevant background PM2.5 is taken to be 3.5 ug/m3 in the East and 2.5 ug/m3 in the West. Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 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 Pope et al. (2002) – ACS extended, 6.6 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 2.1 percent of premature mortality was associated with long-term exposure to $PM_{2.5}$ in excess of background levels in San Jose, which has much lower levels of $PM_{2.5}$. The corresponding incidences of premature mortality using that same study (about 3,700 cases in Los Angeles versus about 170 cases in San Jose) 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 San Jose's population is only about 1.7 million) (see Appendix D).

The risk estimates shown in Fig. 7.5 and in the exhibits in Appendices D, E, F, and G for mortality associated with long-term exposure to $PM_{2.5}$ based on the C-R function from the ACS-extended study are greater than the risk estimates based on the Six City function, even though the Six City Study reported a higher RR, because the LML in the ACS-extended study is 7.5 $\mu g/m^3$ whereas the LML in the Six Cities study is 11 $\mu g/m^3$. The fact that the C-R function is applied in the range from 7.5 to 11 $\mu g/m^3$ only for the ACS extended study results in higher risk estimates associated with that study.

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 short-term exposure mortality studies used in the different locations). For example, in Los Angeles, 0.8 percent of short-term exposure non-accidental mortality was associated with "as is" $PM_{2.5}$ concentrations in excess of background (Moolgavkar (2003) [using the GAM (stringent) model with 30 df and 0-day lag]) compared with anywhere from 4.1 percent (Krewski et al., 2000 - ACS) to 6.6 percent (Pope et al., 2002 – ACS extended) of long-term exposure mortality. In San Jose, however, 2.6 percent of short-term exposure non-accidental mortality was associated with "as is" $PM_{2.5}$ concentrations in excess of background (Fairley, 2003) [0-day lag, single pollutant model], compared with 0.5 percent (Krewski et al., 2000 – ACS) to 2.1 percent (Pope et al., 2002 – ACS extended) of long-term exposure total mortality cases.

Seattle had the lowest $PM_{2.5}$ concentrations among the assessment locations. It's annual mean of 8.3 $\mu g/m^3$ was lower than the LML (10 $\mu g/m^3$) in the Krewski et al. (2000) – ACS and close to the LML (7.5 $\mu g/m^3$) in the ACS-extended study. Because risks were calculated only down to the LML in the study, the risks in Seattle associated with long-term exposure mortality are shown only for the ACS-extended study.

Figure 7.6 shows the effect of having only $PM_{2.5}$ in the model (single pollutant model) vs. having other pollutants in the model as well (multi-pollutant model), using the effect estimates

for long-term exposure mortality based on Krewski et al. (2000) - ACS study. The bars labeled "PM only" represent the effect on mortality associated with $PM_{2.5}$ exposures estimated by a model in which $PM_{2.5}$ is the only pollutant in the model. The bars labeled with other pollutants (e.g., CO, NO_2, SO_2) represent the effect on mortality associated with $PM_{2.5}$ exposures estimated when other pollutants are also included in the health effects model. The $PM_{2.5}$ effect estimates are generally increased with the addition of CO, NO_2, or O_3 in two-pollutant models and are substantially decreased with the addition of SO_2 in such models.

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 2.6). 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 alternative constant background concentrations we focused the $PM_{2.5}$ sensitivity analyses on total (or non-accidental) mortality. The sensitivity analyses in this section and the exhibits presenting their results are summarized in Exhibit 7.3. The results of the sensitivity analyses for Detroit are shown in Exhibits 7.4 through 7.8 for $PM_{2.5}$, and Exhibits 7.12 and 7.13 for $PM_{10-2.5}$.

In addition to the sensitivity analyses carried out in all locations included in the $PM_{2.5}$ risk assessment, we carried out two sensitivity analyses in single locations. In 2002, natural fires in Quebec, Canada resulted in several days of exceptionally high levels of PM in the Northeastern United States. Exhibit 7.9 shows the impact of these "exceptional event episodes" on air quality in Boston, MA in 2002, and Exhibit 7.10 shows the impact on the estimated annual health risks associated with "as is" $PM_{2.5}$ concentrations.

Finally, using Moolgavkar (2003), we examined the effect of different model specifications on estimated annual health risks associated with "as is" $PM_{2.5}$ concentrations in Los Angeles. The results are shown in Exhibits 7.11a (for mortality) and 7.11b (for morbidity).

Exhibit 7.3 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 7.4 Exhibits D.11 - D.18
Estimated annual health risks associated with "as is" PM concentrations using alternative hypothetical thresholds	mortality associated with short-term and long-term exposure	Exhibit 7.5 Exhibits D.19 - D.26
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 7.6 Exhibits D.27 - D.31
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 7.7 Exhibits D.32 - D.39
Estimated annual health risks associated with "as is" $PM_{2.5}$ concentrations using a constant background level versus different daily background levels	non-accidental mortality associated with short-term exposure - Boston, Detroit, and St. Louis	Exhibit 7.8 Exhibit D.40
Estimated annual health risks associated with "as is" $PM_{2.5}$ concentrations, with and without "exceptional/natural event episodes"	mortality and respiratory symptoms associated with short-term exposure – Boston only	Exhibits 7.9 and 7.10
Estimated annual health risks associated with short-term exposures, using alternative model specifications – Los Angeles only	health risks associated with short-term exposures - Los Angles only	Exhibit 7.11
$PM_{10-2.5}$ risk assessment:		
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 Exhibits D.41and D.42

Sensitivity Analysis*	Applied to	Exhibit
Estimated annual health risks associated with "as is" $PM_{10-2.5}$ concentrations using alternative hypothetical thresholds	Hospital admissions and respiratory symptoms – Detroit**	Exhibit 7.13

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

**The threshold sensitivity analysis requires a highest measured level from the study. Only Ito (2003) in Detroit provided this information, so Detroit is the only location for which a threshold analysis could be carried out for $PM_{10-2.5}$.

Exhibit 7.9. Comparison of $PM_{2.5}$ Concentrations in Boston, MA in 2002 With and Without Monitor-Days Flagged as "Exceptional/Natural Event Episodes"

Monitor:	250170008881011		250210007881011		250250042881011		250250043881011		composite	
	Including All Days	Excluding Exceptional Event Days	Including All Days	Excluding Exceptional Event Days	Including All Days	Excluding Exceptional Event Days	Including All Days	Excluding Exceptional Event Days	Including All Days	Excluding Exceptional Event Days
number of monitor days	103	102	118	117	264	261	86	85	299	296
mean ($\mu g/m^3$)	10.8	10.3	12.2	11.7	11.4	11.1	13.9	13.3	11.5	11.2
75^{th} percentile ($\mu g/m^3$)	12.5	12.3	15.5	14.8	14.0	13.7	17.5	17.4	14.3	14.2
90^{th} percentile ($\mu g/m^3$)	21.1	20.8	23.0	22.6	21.2	20.4	25.1	24.4	21.2	20.4
95^{th} percentile ($\mu g/m^3$)	27.5	23.8	27.7	26.2	24.9	23.8	27.1	26.4	25.2	24.0
98^{th} percentile ($\mu g/m^3$)	29.2	28.5	48.1	33.8	33.0	26.4	29.8	28.2	33.0	27.4
maximum value ($\mu g/m^3$)	65.1	30.6	66.9	66.9	59	52.4	63.1	29.8	63.1	51.2
number of days above 30 $\mu g/m^3$	2	1	5	4	6	4	1	0	6	4
number of days above 50 $\mu g/m^3$	1	0	2	1	3	2	1	0	2	1

The first set of sensitivity analyses, shown in Exhibits 7.4 and 7.12 for $PM_{2.5}$ and $PM_{10-2.5}$, respectively, examine the effect of alternative assumptions about PRB 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 noticeable effect. For example, changing from the midpoint estimate of 3.5 $\mu g/m^3$ for $PM_{2.5}$ background in the Eastern U.S. to the lower end of the range for $PM_{2.5}$ background (2 $\mu g/m^3$) increased the estimated percent of total incidence that is $PM_{2.5}$-related using Schwartz (2003b) in Boston by about 17 percent (from 1.8 percent to 2.1 percent). Similarly, changing from the midpoint estimate to the upper end of the range for $PM_{2.5}$ (5 $\mu g/m^3$) decreased the percent of total incidence that is $PM_{2.5}$-related using that same study by about 17 percent (from 1.8 percent to 1.5 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 Pope et al. (2002) – ACS extended was 7.5 $\mu g/m^3$, 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 noticeable impact on the estimates.

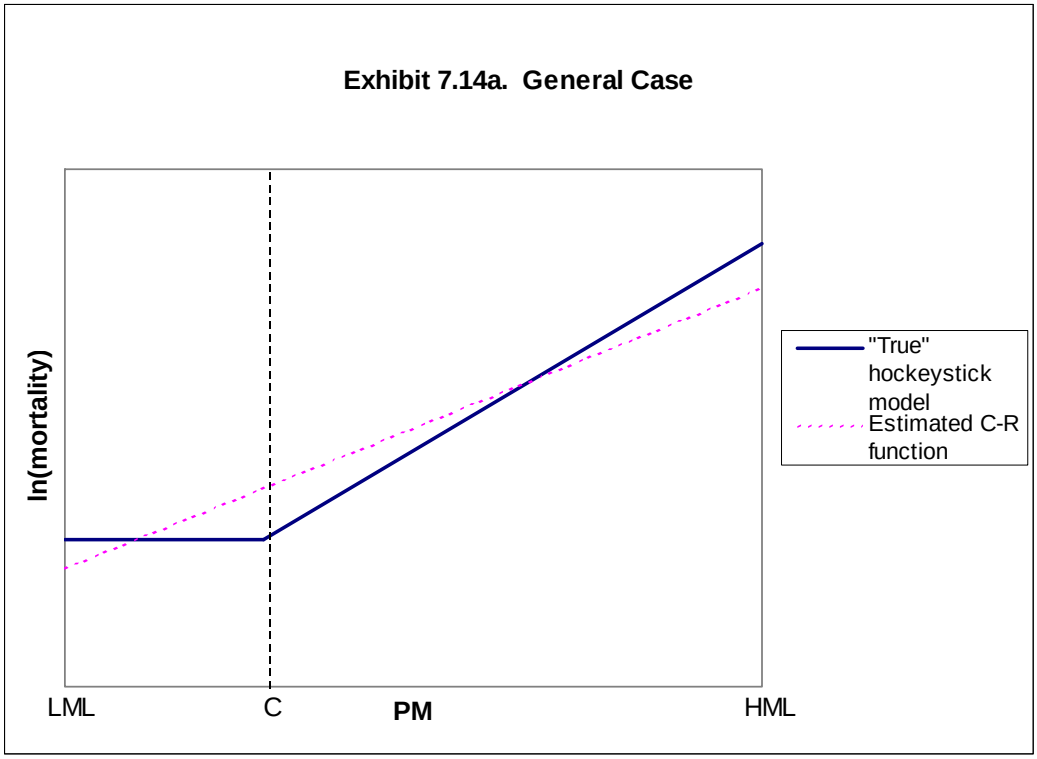
The second set of sensitivity analyses address the issue of possible thresholds below which there may be no PM effects. While the 2004 PM CD concludes that there is no strong evidence of a clear threshold for PM mortality effects, it also notes "nor is there clear evidence against possible thresholds for PM-related effects" (EPA, 2004; p.8-322). The 2004 PM CD also states that "some single-city studies do provide some suggestive hints for possible thresholds, but not in a statistically clear manner" (EPA, 2004; p.8-322). Therefore, sensitivity analyses have been conducted that include hypothetical alternative thresholds, where risks are estimated only due to $PM_{2.5}$ or $PM_{10-2.5}$ concentrations exceeding the assumed threshold concentrations. Based on the EPA staff evaluation (EPA, 2005; see section 3.6.6 and section 4), three hypothetical thresholds (10, 15, and 20 $\mu g/m^3$**)** were used in sensitivity analyses for short-term exposure mortality for $PM_{2.5}$ and short-term exposure morbidity for $PM_{10-2.5}$ and two hypothetical thresholds (10 and 12 $\mu g/m^3$) were used in sensitivity analyses for long-term exposure mortality associated with $PM_{2.5.}$

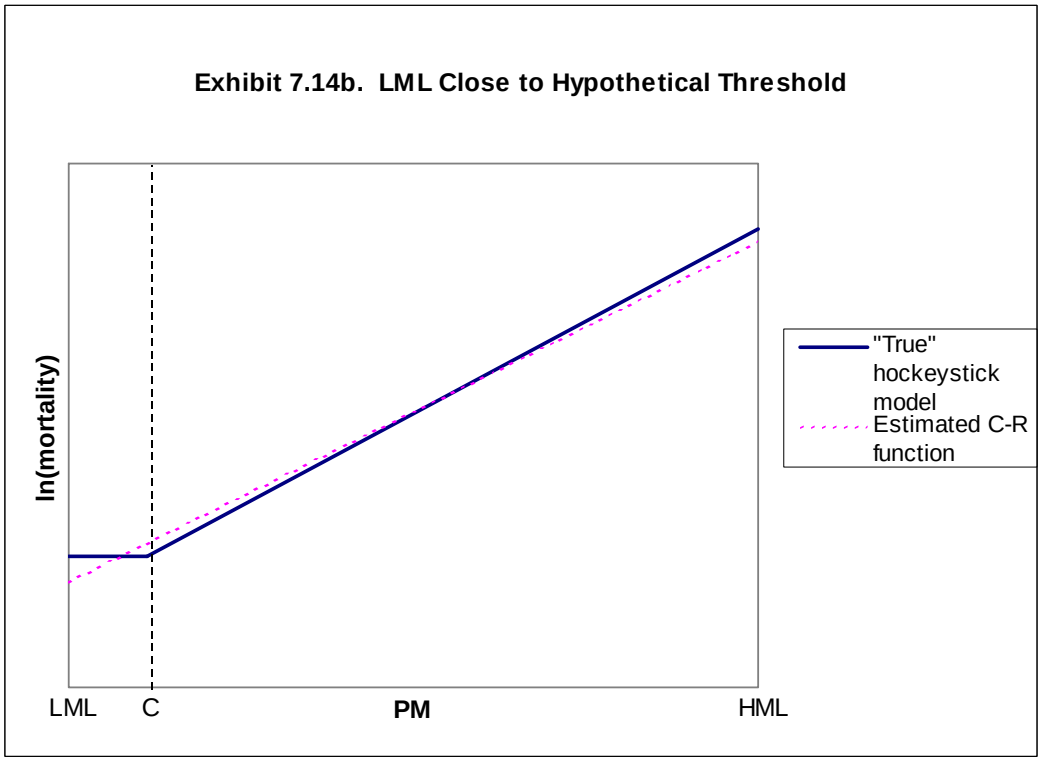
The rationale for considering hypothetical thresholds is the hypothesis that there is some PM concentration below which there are no PM-related effects. If the original data in the study reporting a C-R function supported a threshold hypothesis, then the model that best fit the data would look like a 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 7.14a. 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 or morbidity above the hypothetical threshold, as shown in Exhibit 7.14a. 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 or morbidity study, the curve represents the relationship between daily PM and daily mortality or morbidity; 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 or morbidity 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 $\mu g/m^3$ for long-term exposure mortality, given that the LMLs in the long-term exposure mortality studies were 7.5, 10, or 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 7.14b.

Exhibit 7.14 Relationship Between Estimated Log-Linear Concentration-Response Function and Hockeystick Model With Threshold C





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. We believe that this slope adjustment method is a reasonable approach to illustrate the potential impact of using a non-linear approach. A more definitive evaluation of the effect of hypothetical thresholds and use of alternative non-linear approaches would require re-analysis of the original health and air quality data, which is beyond the scope of this risk assessment.

Exhibits 7.5 and 7.13 show the percentages of total incidence in Detroit predicted to be associated with $PM_{2.5}$ and $PM_{10\text{-}2.5}$, respectively, in excess of an estimated LML of 4 $\mu g/m^3$, in the case of $PM_{2.5}$, and an estimated PRB of 4.5 $\mu g/m^3$, in the case of $PM_{10\text{-}2.5}$, 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.

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 Exhibit 7.6 (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 7.7. The results for the other assessment locations are shown in Appendix D.

The impact of using different daily background $PM_{2.5}$ concentrations (versus a constant background concentration) on the estimates of risk associated with "as is" $PM_{2.5}$ concentrations in excess of background was assessed in Detroit and St. Louis. Daily background values were generated first assuming no correlation between the anthropogenic portion of "as is" concentrations and background concentrations, and then assuming a moderate correlation of 0.4. The method of generating daily background concentrations is described in detail in Langstaff

(2004). Using different daily background $PM_{2.5}$ concentrations had only a minimal effect on the estimates of risk reduction. The estimated percent of total non-accidental mortality associated with short-term exposure to $PM_{2.5}$ in excess of background levels in Detroit decreased from 0.9 percent (163 cases) to 0.8 percent (153 cases) when different daily background concentrations (assuming zero correlation with the anthropogenic portion of "as is" concentrations) were substituted for a constant PRB (Exhibit 7.8). The changes in percent of total non-accidental mortality in St. Louis (assuming a 0.4 correlation of background and anthropogenic concentrations) were not sufficiently large to be detected when results were rounded to one decimal place. (The results for St. Louis are therefore not shown.)

As noted earlier, a sensitivity analysis was conducted examining the impact on PM risk estimates of a large natural fire that occurred in July 2002 in Quebec that resulted in unusually high $PM_{2.5}$ concentrations being reported at ambient monitors in the northeastern portions of the U.S. The exclusion of "exceptional event episodes" in Boston in 2002 resulted in three fewer days at the composite monitor (296 vs. 299 days with composite monitor values), a decrease in the maximum $PM_{2.5}$ value from 63.1 to 51.2 $\mu g/m^3$, and a decrease in the annual average $PM_{2.5}$ concentration from 11.5 to 11.2 $\mu g/m^3$ (see Exhibit 7.9). The corresponding decreases in mortality associated with short-term exposure to $PM_{2.5}$ were quite modest (see Exhibit 7.10). The incidence of PM-related non-accidental mortality estimated using Schwartz (2003b), for example, decreased from 356 to 345; the corresponding change in the estimated percent of total incidence was not sufficiently large to be detected when results were rounded to one decimal place. Decreases in long-term exposure mortality were somewhat larger. The incidence of PM-related mortality estimated using Pope et al. (2002) – ACS extended decreased from 507 to 477; the estimated percent of total incidence decreased from 2.3 percent to 2.1 percent.

The impact of different model specifications and different lag structures on mortality and morbidity risks in Los Angeles is shown in Exhibits 7.11a and 7.11b, respectively. As noted in Section 1 above, many time-series studies which reported log-linear C-R functions based on generalized additive models (GAMs) estimated using the S-Plus software had to re-estimate those C-R functions using appropriate modifications of the default convergence criteria code. In re-estimating C-R functions for $PM_{2.5}$ and mortality and morbidity in Los Angeles, Moolgavkar (2003) presented three different model specifications – GAMs with more stringent convergence criteria with 30 degrees of freedom (df) and with 100 df, as well as a generalized linear model (GLM) with 100 df. Results are shown based on each of these model specifications in combination with both 0-day and 1-day lags for all health endpoints, and with a 2-day lag in addition for hospital admissions for COPD.

Estimated mortality and morbidity risks varied substantially with model specification and lag structure, although there was no obvious consistent pattern across all health endpoints. GLM estimates were generally higher than GAM estimates with the same number of df for both cardiovascular and COPD hospital admissions, but lower for non-accidental and cardiovascular mortality. Not surprisingly, increasing the df generally lowered the estimated incidence. The

highest non-accidental mortality risk estimate (491 PM-related deaths associated with "as is"$PM_{2.5}$ concentrations, or 0.9 percent of total incidence) was produced by the GAM with 30 df and a 1-day lag, and the lowest (-8 PM-related deaths, or 0.0 percent of total incidence) was produced by the GLM with 100 df and a 1-day lag. Cardiovascular mortality exhibited the same pattern as non-accidental mortality across model specifications and lag structures. For cardiovascular hospital admissions, PM-related incidence estimates ranged from 1627 (2.4 percent of total incidence) produced by the GAM with 30 df and a 0-day lag to 1170 (1.7 percent of total incidence) produced by the GAM with 100 df and a 1-day lag. For COPD hospital admissions, PM-related incidence estimates ranged from 829 (2.7 percent of total incidence) produced by the GAM with 30 df and a 2-day lag to 341 (1.1 percent of total incidence) produced by the GAM with 100 df and a 1-day lag.

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

8.1 Health risk reductions associated with just meeting the current $PM_{2.5}$ standards

The second part of the risk assessment estimates the risk reductions that would result if the current $PM_{2.5}$ standards or alternative $PM_{2.5}$ standards were just met in the assessment locations. Several of the assessment locations (Boston, Phoenix, San Jose, and Seattle) already meet the current $PM_{2.5}$ annual standard of 15 $\mu g/m^3$ and the current daily $PM_{2.5}$ standard of 65 $\mu g/m^3$. In considering risk reductions associated with just meeting the current standards, 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.[27] 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.[28] 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.

[27] 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.

[28] 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.

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 μg/m^3 and the Current Daily Standard of 65 μg/m^3 (and 95 Percent Confidence Interval): Non-accidental Mortality Associated with Short-Term Exposure to $PM_{2.5}$

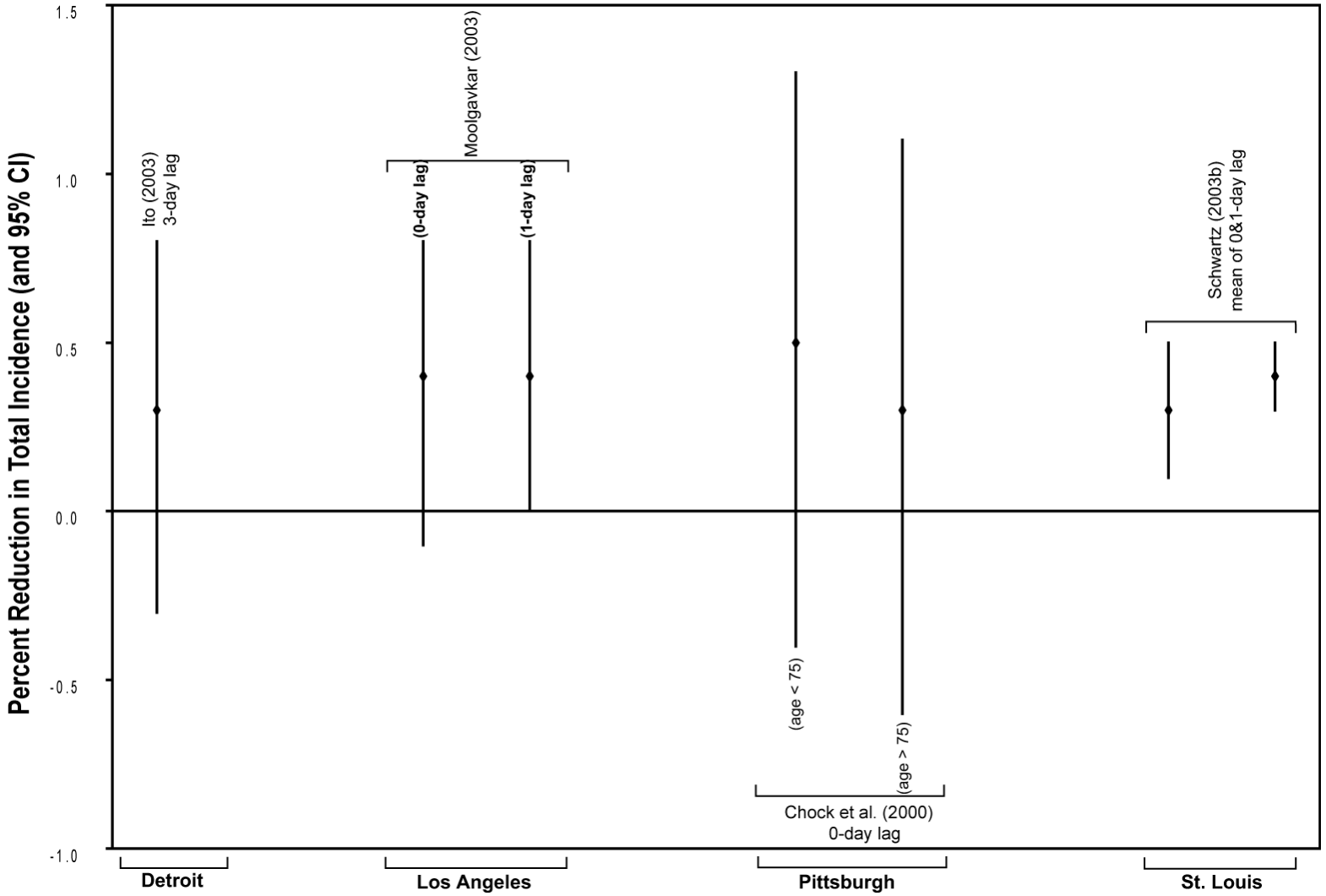
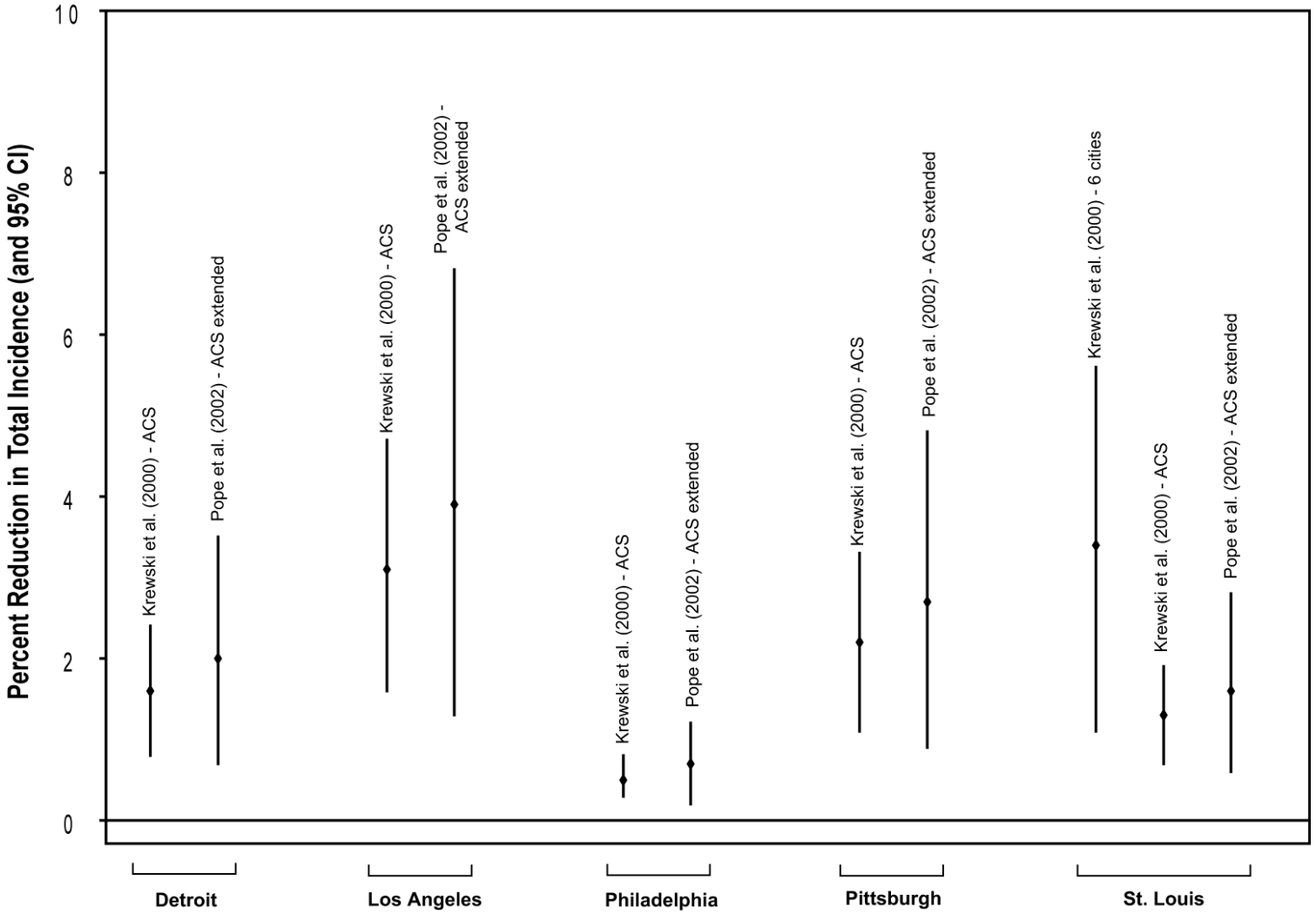


Figure 8.2. Estimated Annual Percent Reduction of Health Risks Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standard of 15 μg/m^3 and the Current Daily Standard of 65 μg/m^3 (and 95 Percent Confidence Interval): Mortality Associated with Long-Term Exposure to $PM_{2.5}$



8.2 Health risk reductions associated with just meeting alternative $PM_{2.5}$ standards

In this part of the risk assessment, we estimated health risk reductions associated with just meeting alternative sets of $PM_{2.5}$ standards. For those locations that have not met the current $PM_{2.5}$ standards (Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis), these reductions in risk were those above and beyond what would be achieved by meeting the current standards. Reductions in incidence associated with just meeting an alternative set of standards in these locations were thus calculated as the reduction in incidence if "as is" $PM_{2.5}$ were rolled back to just meet the alternative standards *minus the reduction in incidence if "as is" $PM_{2.5}$ were rolled back to just meet the current standards*. For example, using Schwartz (2003b) we estimated that just meeting the current $PM_{2.5}$ standards in St. Louis would result in 62 non-accidental deaths avoided (Exhibit E.4). Meeting the more stringent annual and daily 98th percentile standards of 14 $\mu g/m^3$ and 40 $\mu g/m^3$, respectively, was estimated to result in 77 non-accidental deaths avoided, or *an additional* (77 - 62 =)15 non-accidental deaths avoided (Exhibit F.5c).

For those locations that have met the current $PM_{2.5}$ standards (Boston, Phoenix, San Jose, and Seattle), the health risk reductions associated with just meeting alternative sets of $PM_{2.5}$ standards were calculated as the reduction in incidence if "as is" $PM_{2.5}$ were rolled back to just meet the alternative standards

Annual standards of 15, 14, 13, and 12 $\mu g/m^3$ were each combined with ninety-eighth percentile daily standards of 40, 35, 30, and 25 $\mu g/m^3$, and ninety-ninth percentile daily standards at the same levels. In addition, an annual standard of 15 $\mu g/m^3$ was combined with a ninety-ninth percentile daily standard of 65 $\mu g/m^3$. Among those locations that have not met the current $PM_{2.5}$ standards (Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis), there was no difference in the percent rollback required to just meet any of the alternative annual standards (14, 13, and 12 $\mu g/m^3$) in combination with the current ninety-eighth percentile daily standard of 65 $\mu g/m^3$ versus the percent rollback required to just meet any of these annual standards in combination with the more stringent ninety-eighth percentile daily standard of 40 $\mu g/m^3$ – except in Philadelphia, where just meeting the 14 $\mu g/m^3$ annual standard combined with the ninety-eighth percentile daily standard of 40 $\mu g/m^3$ requires a 23.2% rollback compared with an 18.6% rollback necessary to just meet the 14 $\mu g/m^3$ annual standard combined with the ninety-eighth percentile daily standard of 65 $\mu g/m^3$. Because of this, the 14 $\mu g/m^3$ annual standard was combined with the ninety-eighth percentile daily standard of 65 $\mu g/m^3$ only in Philadelphia. The combinations of annual and daily alternative standards used in the $PM_{2.5}$ risk assessment are summarized in Exhibit 8.2.[29] The results are given in Figures 8.3 - 8.6 and Exhibits 8.3a through 8.3h for Detroit, in Appendix F for the other locations that have not met the current standards (Los Angeles, Philadelphia, Pittsburgh, and St. Louis), and in Appendix G for the locations that have met the current standards (Boston, Phoenix, San Jose, and Seattle).

[29] See U.S. EPA(2005) for a discussion of the rationale for selecting these alternative standards.

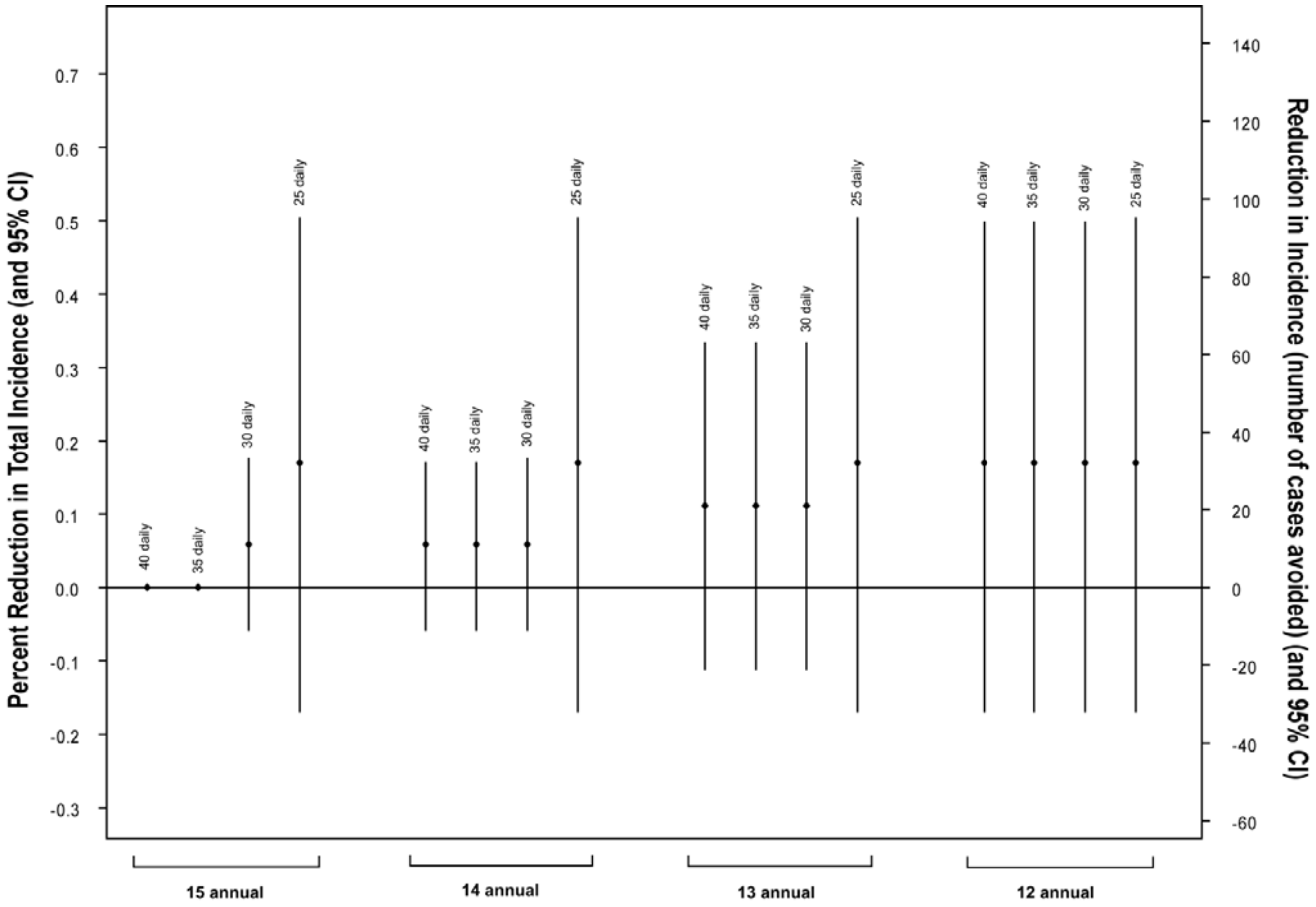
Exhibit 8.2 Alternative Sets of $PM_{2.5}$ Standards Considered in the $PM_{2.5}$ Risk Assessment*

Annual Standard		98th Percentile Daily Standard				99th Percentile Daily Standard				
	65	40	35	30	25	65	40	35	30	25
15		x	x	x	x	x	x	x	x	x
14	x**	x	x	x	x		x	x	x	x
13		x	x	x	x		x	x	x	x
12		x	x	x	x		x	x	x	x

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

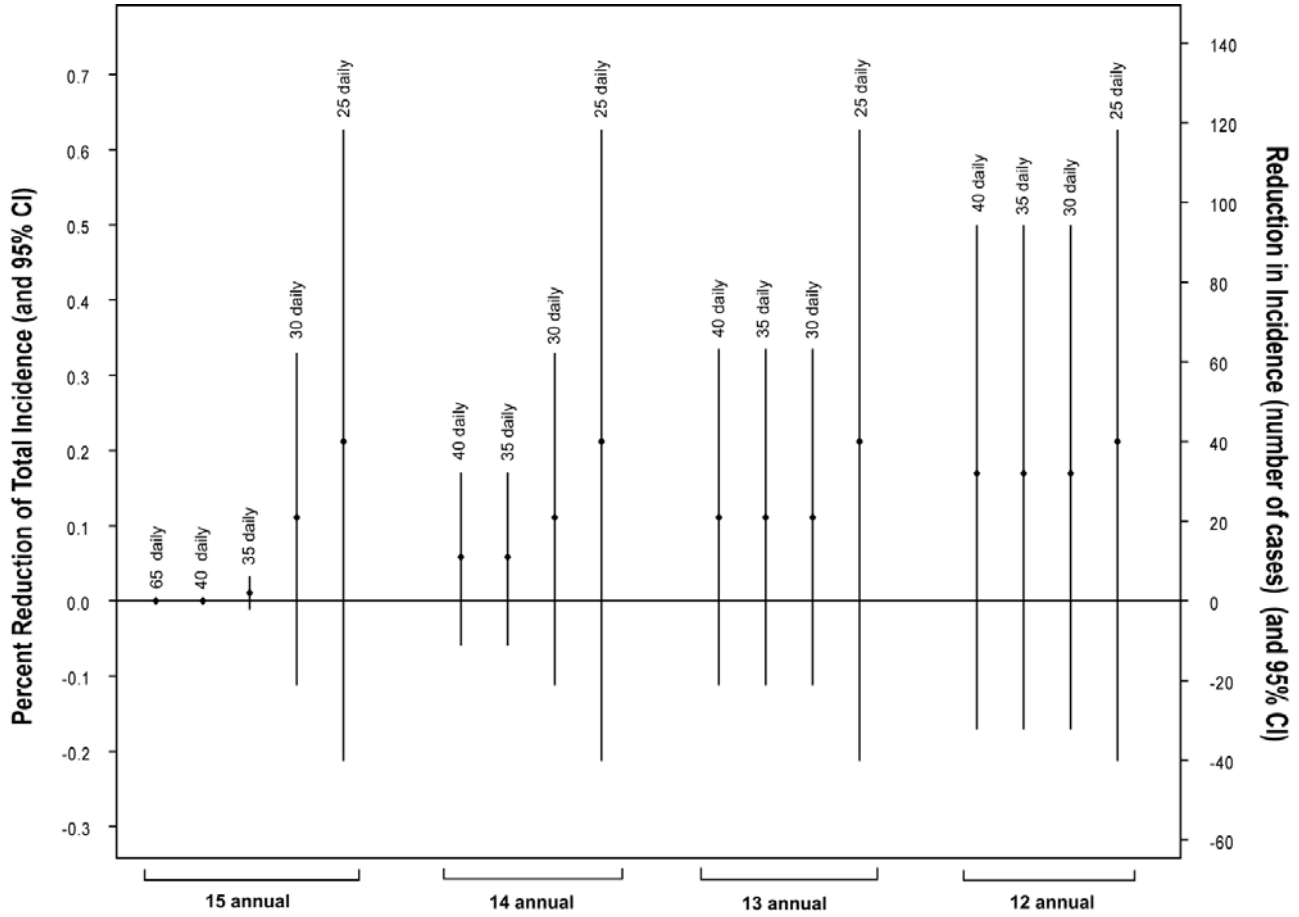
**Only in Philadelphia.

Figure 8.3. Estimated Annual Reduction in Short-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations that Just Meet the Current Annual Standard of 15 μg/m^3 and the Current Daily Standard of 65 μg/m^3 to $PM_{2.5}$ Concentrations that Just Meet Alternative Suites of $PM_{2.5}$ Annual and Daily 98th Percentile Standards: Detroit, MI, 2003*



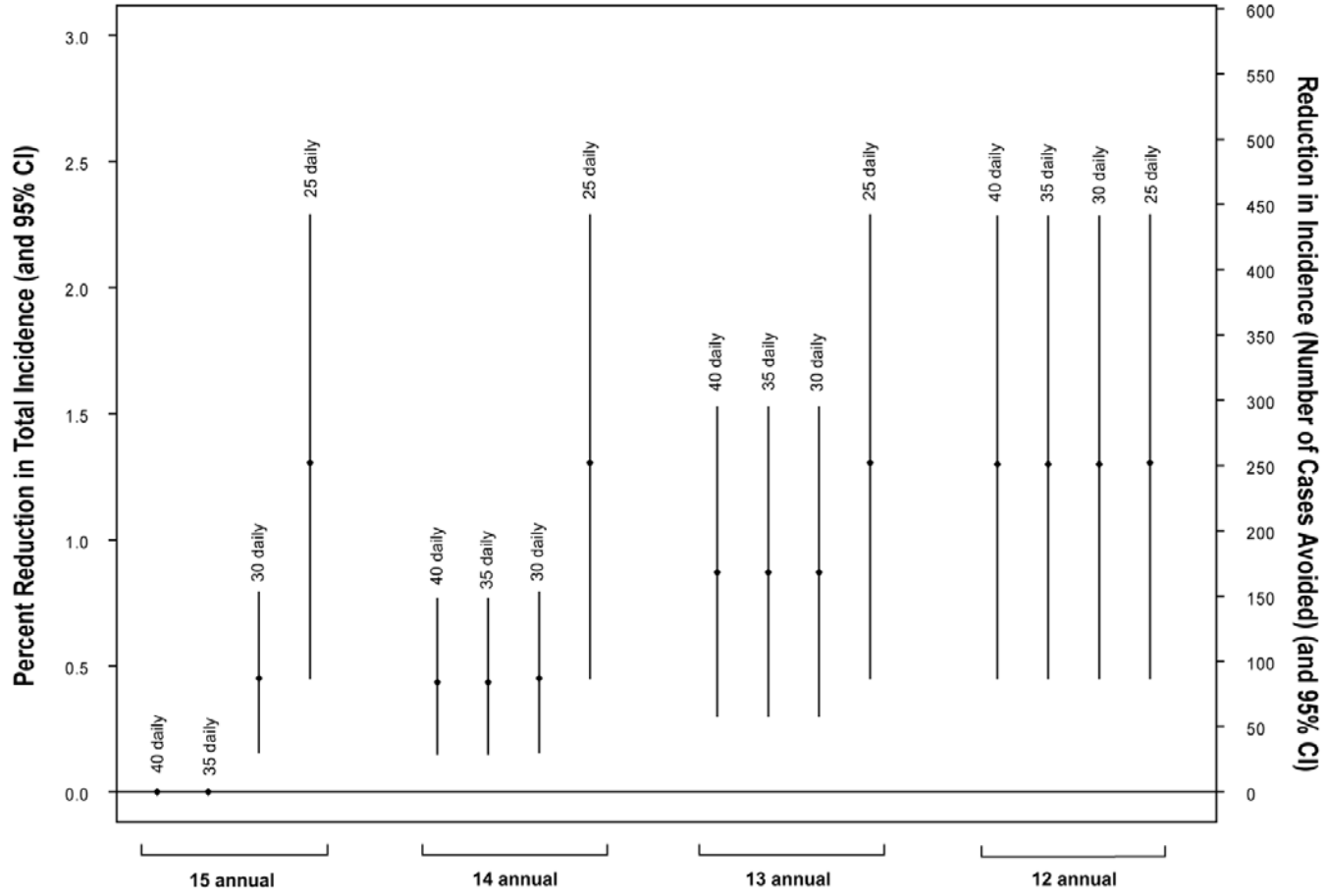
*Based on Ito (2003)

Figure 8.4. Estimated Annual Reduction in Short-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations that Just Meet the Current Annual Standard of 15 μg/m^3 and the Current Daily Standard of 65 μg/m^3 to $PM_{2.5}$ Concentrations that Just Meet Alternative Suites of $PM_{2.5}$ Annual and Daily 99th Percentile Standards: Detroit, MI, 2003*



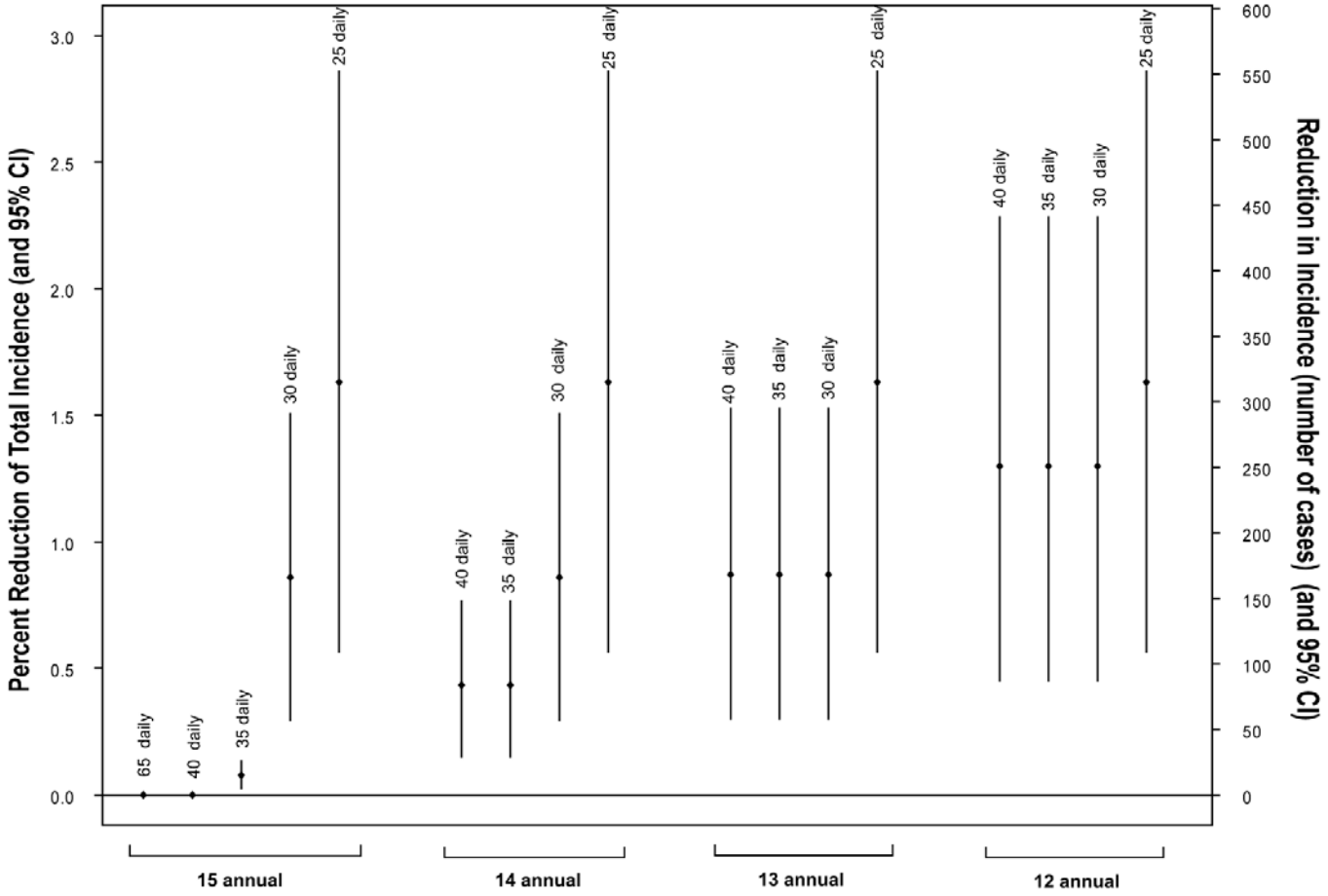
*Based on Ito (2003)

Figure 8.5. Estimated Annual Reduction in Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations that Just Meet the Current Annual Standard of 15 μg/m^3 and the Current Daily Standard of 65 μg/m^3 to $PM_{2.5}$ Concentrations that Just Meet Alternative Suites of $PM_{2.5}$ Annual and Daily 98th Percentile Standards: Detroit, MI, 2003*



*Based on Pope et al. (2002) – ACS extended

Figure 8.6. Estimated Annual Reduction in Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations that Just Meet the Current Annual Standard of 15 μg/m^3 and the Current Daily Standard of 65 μg/m^3 to $PM_{2.5}$ Concentrations that Just Meet Alternative Suites of $PM_{2.5}$ Annual and Daily 99th Percentile Standards: Detroit, MI, 2003*



*Based on Pope et al. (2002) – ACS extended

Exhibit 8.3a Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003

Exhibit 8.3a Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3a Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3b Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003

Exhibit 8.3b Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003
(continued)

Exhibit 8.3b Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3c Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003

Exhibit 8.3c Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3c Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3d Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003

Exhibit 8.3d Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003
(continued)

Exhibit 8.3d Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003
(continued)

Exhibit 8.3e Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003

Exhibit 8.3e Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3e Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3f Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003

Exhibit 8.3f Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3f Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3g Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003

Exhibit 8.3g Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3g Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003 (continued)

Exhibit 8.3h Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003

Exhibit 8.3h Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003
(continued)

Exhibit 8.3h Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{2.5}$ Standards: Detroit, MI, 2003
(continued)

8.3 Sensitivity analyses for health risk reductions associated with just meeting the current and alternative $PM_{2.5}$ standards

8.3.1 The effect of alternative rollback methods

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 2001 - 2003 air quality data.

To meet the annual standard, the annual average must not exceed the annual standard of 15 $\mu g/m^3$. 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 8.4. 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 $\mu g/m^3$.

Exhibit 8.4 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 $\mu g/m^3$ and the Current Daily Standard of 65 $\mu g/m^3$ Using an Alternative Rollback Method: Detroit, MI, 2003

8.3.2 Health risks associated with just meeting the current and alternative $PM_{2.5}$ standards under different hypothetical threshold assumptions

Among the alternative assumptions examined in the sensitivity analyses on estimated health risks associated with "as is" $PM_{2.5}$ concentrations, the impact of hypothetical thresholds was the greatest. We therefore carried out an additional set of sensitivity analyses to examine the impact of different hypothetical threshold assumptions on estimated risks associated with just meeting the current $PM_{2.5}$ standards and each of the alternative $PM_{2.5}$ standards considered in Section 8.2. Some locations already meet the current standards, and some alternative standards as well. Whenever standards are already met, the risks estimated are associated with "as is" $PM_{2.5}$ concentrations. Estimates of risk for all cause mortality, cardiopulmonary mortality, and lung cancer mortality associated with long-term exposure to $PM_{2.5}$ were based on the C-R relationship derived from Pope et al. (2002) – ACS extended. In addition, we considered non-accidental mortality associated with short-term exposure to $PM_{2.5}$. If there was no C-R function available for non-accidental mortality in a location, a cause-specific mortality was used instead. In one location (Seattle), there were no C-R functions for non-accidental or any cause-specific mortality. In this case, we used hospital admissions for asthma (among ages < 65). Hypothetical thresholds of 10, 15, and 20 $\mu g/m^3$ were considered for health endpoints associated with short-term exposures, and hypothetical thresholds of 10 and 12 $\mu g/m^3$ were considered for the mortality endpoints associated with long-term exposure. The results of these sensitivity analyses for Detroit are shown in Exhibits 8.5 - 8.8 for non-accidental mortality associated with short-term exposure to $PM_{2.5}$, and all cause, cardiopulmonary, and lung cancer mortality associated with long-term exposure to $PM_{2.5}$, respectively. The results for the other locations are shown in the first section of Appendix H.

Not surprisingly, estimated PM-related incidences varied substantially with both hypothetical threshold assumptions and alternative standards. In Detroit, for example, the estimated number of cases of mortality associated with short-term exposure to $PM_{2.5}$ when the current standards are just met decreases from 115, under the assumption of no threshold, to 54, 26, and 12 under hypothetical threshold assumptions of 10, 15, and 20 $\mu g/m^3$, respectively. Because those cases that get eliminated under hypothetical threshold assumptions are associated with $PM_{2.5}$ at levels below the assumed threshold and those cases that are eliminated when alternative standards are just met are associated with $PM_{2.5}$ at higher ambient levels, higher hypothetical thresholds tend to increase the percent reduction associated with just meeting lower concentration level alternative standards. For example, going from just meeting the current standards (15 $\mu g/m^3$ annual and 65 $\mu g/m^3$ daily 98th percentile value) to just meeting the most stringent standards considered (12 $\mu g/m^3$ annual and 25 $\mu g/m^3$ daily 99th percentile value) results in a reduction in cases of (115 - 75)/115 = 34.8 percent under the assumption of no threshold, but under the assumption of a threshold of 10 $\mu g/m^3$ it results in a reduction of (54 - 22)/54 = 59.3 percent. Under hypothetical thresholds of 15 and 20 $\mu g/m^3$, the reductions are 73.1 percent and 83.3 percent, respectively. The same general patterns can be seen in all locations and for all health endpoints considered.

Exhibit 8.5. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003

Exhibit 8.5. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003 (con't)

Exhibit 8.5. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003 (con't)

Exhibit 8.6. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003

Exhibit 8.6. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003 (con't)

Exhibit 8.6. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003 (con't)

Exhibit 8.7. Sensitivity Analysis: Estimated Annual Cardiopulmonary Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003

Exhibit 8.7. Sensitivity Analysis: Estimated Annual Cardiopulmonary Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003 (con't)

Exhibit 8.7. Sensitivity Analysis: Estimated Annual Cardiopulmonary Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003 (con't)

Exhibit 8.8. Sensitivity Analysis: Estimated Annual Lung Cancer Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003

Exhibit 8.8. Sensitivity Analysis: Estimated Annual Lung Cancer Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003 (con't)

Exhibit 8.8. Sensitivity Analysis: Estimated Annual Lung Cancer Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003 (con't)

8.3.3 Comparison of risk estimates based on annual standard design value calculated from maximum versus average of monitor-specific averages

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 8.9 shows the percent rollbacks that would be required to just meet the current annual standard using four of the assessment locations which do not currently meet the annual standard and which meet the minimum criteria for use of spatial averaging.[30] The three-year period from 2001-2003 was used to determine the amount of rollback required to meet the current annual standard.

Exhibit 8.9 Air Quality Adjustments Required to Just Meet the Current Annual $PM_{2.5}$ Standard of 15 $\mu g/m^3$ 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	28.1%	11.5%
Philadelphia	10.9%	-0.9%
Pittsburgh	35.0%	22.8%
St. Louis	17.9%	13.5%

*The design values based on the maximum of monitor-specific annual averages are given in Exhibit 2.4. The design values based on the spatial average of the monitor-specific annual averages are 16.5 for Detroit, 14.9 for Philadelphia, 18.4 for Pittsburgh, and 16.8 for St. Louis.

The results shown in Exhibits 8.5 - 8.8 and H.1 - H.34 are based on percent rollbacks calculated from annual and daily standard design values that used the maximum of monitor-specific values. If the design values had been based on the average, rather than the maximum, of monitor-specific values, the estimated mortality would have been, in many cases, greater than the estimates shown in those exhibits.

Exhibits 8.10 and 8.11 show the effect of using an annual standard design value based on the *maximum* versus the *average* of monitor-specific averages (while keeping the design values for the daily standards as they were, based on the maximum of monitor-specific values). Exhibit 8.10 shows estimated mortality associated with short-term exposures to $PM_{2.5}$ in Detroit; Exhibit 8.11 shows estimated mortality associated with long-term exposures. Both exhibits present the

[30] The Los Angeles area does not meet the minimum EPA criteria for considering the use of spatial averaging and, thus is not included in Exhibit 8.9.

results based on annual standard design values calculated from the maximum of monitor-specific values (see Exhibits 8.5 and 8.6) alongside the corresponding results based on design values calculated from the average of monitor-specific values. The corresponding comparisons for Pittsburgh, PA and St. Louis, MO (the other two locations that do not meet the current standards and for which both the maximum-based and the average-based annual standard design values result in positive percent rollbacks) are given in Exhibits H.35 - H.38.

Changing the basis of the annual standard design value from the maximum to the average of monitor-specific averages reduces the percent rollback necessary to just meet an annual standard. If the daily standard had previously been the controlling standard (i.e., requiring a greater percent rollback than the annual standard), then reducing the percent rollback necessary to just meet the annual standard will have no effect. Annual/daily standard combinations for which this was the case are not shown in Exhibits 8.10, 8.11, and H.35 - H.38.

If, however, the annual standard had previously been the controlling standard, the new (smaller) percent rollback necessary to just meet the annual standard using an average-based annual standard design value will result in a larger estimated mortality associated with any set of alternative standards. The new smaller percent rollback may still exceed the percent rollback necessary to meet the daily standard. In this case, the annual standard will still be the controlling standard, but the incidence reduction achieved by just meeting the alternative standards will be smaller than it would be if the maximum-based annual standard design value were used. Alternatively, the new (smaller) percent rollback necessary to just meet the annual standard may be less than the percent rollback necessary to meet the daily standard. In this case, the daily standard would become controlling, and the incidence reduction achieved by just meeting the alternative standards would be smaller than it had previously been. However, the incidence reduction, in this case, would be larger than it would be if the new annual standard based on the average of monitor-specific averages were controlling.

Exhibits 8.10 and 8.11 provide examples of each possibility. To meet an annual standard of 15 $\mu g/m^3$ requires a 28.1% rollback when the annual standard design value is based on the maximum of monitor-specific averages, and an 11.5% rollback when it is based on the average of monitor-specific averages. To meet a daily standard of 25 $\mu g/m^3$ based on the 98^{th} percentile value, however, requires a 46.9% rollback (using a daily design value based on the maximum of monitor-specific values). In this case, the daily standard is controlling, whichever of the two annual standard design values is used (so the 15 $\mu g/m^3$ annual standard and 25 $\mu g/m^3$ daily standard combination does not appear in Exhibits 8.10 and 8.11). The combination of an annual standard of 15 $\mu g/m^3$ and a daily standard of 35 $\mu g/m^3$ based on the 98^{th} percentile value presents a different situation. To just meet this daily standard (using the maximum of monitor-specific values) requires a 22.2% rollback. As noted above, the required rollbacks to just meet the annual standard are 28.1% and 11.5% when the annual standard design value is based on the maximum

Exhibit 8.10. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models – Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages: Detroit, MI, 2003

Exhibit 8.10. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models – Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages: Detroit, MI, 2003 (cont'd)

Exhibit 8.11. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models – Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages: Detroit, MI, 2003

Exhibit 8.11. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models – Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages: Detroit, MI, 2003 (cont'd)

Exhibit 8.11. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models – Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages: Detroit, MI, 2003 (cont'd)

and average, respectively, of monitor-specific averages. In this case, the change from maximum-monitor based to averaged-monitors based annual standard design value changes the controlling standard from the annual to the daily standard. The estimated mortality associated with short-term exposures to $PM_{2.5}$ in Detroit correspondingly increases from 115 to 125 (see Exhibit 8.10). If the averaged-monitors based annual standard had been controlling, the estimated mortality associated with short-term exposure would have been 143 (as it is whenever the averaged-monitors based annual standard of 15 $\mu g/m^3$ is controlling – for example, when combined with daily standards of 65 or 40 $\mu g/m^3$ based on the 98th percentile value).

The change from a maximum-monitor based to an averaged-monitors based annual standard design value induces a change not only in the estimated mortality associated with just meeting alternative more stringent standards, but also in the estimated mortality associated with just meeting the current standards. Because of this, there does not appear to be any clear pattern to the impact on the percent reduction achieved by just meeting alternative more stringent standards. For example, mortality associated with long-term exposure to $PM_{2.5}$ in Detroit when the current standards are just met is estimated to be 522 using a maximum-monitor based annual standard design value, and 747 using an annual standard design value that is averaged-monitors based. The percent reduction in incidence when the more stringent 14 $\mu g/m^3$ annual and 40 $\mu g/m^3$ daily 98th percentile standards are just met is greater when the maximum-monitor based annual standard design value is used – a 16 percent (= (522-438)/522) reduction in mortality using the maximum-monitor based annual standard design value versus a 14 percent (= (747 - 642)/747) reduction using the averaged-monitors based annual standard design value. In contrast, the percent reduction in incidence when the more stringent 14 $\mu g/m^3$ annual and 35 $\mu g/m^3$ daily 99th percentile standards are just met is greater when the averaged-monitors based annual standard design value is used – a 32 percent (= (747-507)/747) reduction in mortality using the averaged-monitors based annual standard design value vs. a 16 percent (= (522-438)/522) reduction using the maximum-monitor based annual standard design value.

The assumption of a threshold tends to accentuate the impact of the change from a maximum-monitor based to an averaged-monitors based annual standard design value in the estimation of mortality when alternative standards are just met. As noted above, using an averaged-monitors based annual standard design value will result in mortality estimates that are at least as large as those calculated using the maximum-monitor based annual standard design value, and in many cases (whenever the annual standard was the controlling standard) mortality estimates will be larger. In almost all cases, the percent increase (from one estimate to the other) is larger under the assumption of a 10 $\mu g/m^3$ threshold than it is in the base case, and it increases with increasing thresholds. For example, when a 15 $\mu g/m^3$ annual standard and a 40 $\mu g/m^3$ 98th percentile daily standard are just met, estimated mortality associated with long-term exposure to $PM_{2.5}$ in Detroit calculated using an averaged-monitors based annual standard design value is 747 in the base case; using a maximum-monitor based annual standard design value it is 522 in

the base case. The percent increase in the base case is therefore 43.2% (=(747-522)/522).[31] The corresponding mortality estimates under the assumption of a 10 $\mu g/m^3$ threshold are 535 and 282, resulting in a percent increase of 89.6% (=(535-282)/282). The corresponding percent increase under a threshold assumption of 12 $\mu g/m^3$ is 684.3% (=(322-41)/41).

The pattern is the same for mortality associated with short-term exposure to $PM_{2.5}$. For example, estimated mortality associated with short-term exposure to $PM_{2.5}$ in Detroit when a 15 $\mu g/m^3$ annual standard and a 65 $\mu g/m^3$ 98^{th} percentile daily standard are just met, calculated using an averaged-monitors based annual standard design value is 143 in the base case; using a maximum-monitor based annual standard design value it is 115 in the base case. The percent increase in the base case is therefore 24.6% (=(143-115)/115). The corresponding mortality estimates under the assumption of a 10 $\mu g/m^3$ threshold are 80 and 54, resulting in a percent increase of 47.5% (=(80-54)/54). The corresponding percent increases under threshold assumptions of 15 $\mu g/m^3$ and 20 $\mu g/m^3$ are 75.9% and 109.8%, respectively.

Measured in terms of percent increase (from the maximum-monitor based to the averaged-monitorsbased mortality estimate), there was substantial variability in the impact of using an averaged-monitors based annual standard design value versus one that is maximum-monitor based. For mortality associated with short-term exposure to $PM_{2.5}$ in Detroit in the base case, for example, positive percent increases ranged from 0.3% to about 25%.[32] Under the assumption of a 20 $\mu g/m^3$ threshold, positive percent increases ranged from 1.3% to about 143%.

[31] Incidence numbers shown in Exhibits 8.10, 8.11, and H.35 - H.38 are rounded to the nearest whole number; the percentages shown here are based on the unrounded incidence numbers.

[32] Whenever the daily standard had been the controlling standard, the change from maximum-monitor based to averaged-monitors based annual standard design value had no impact – i.e., the percent increase was zero.

9. Assessment of the Health Risk Reductions Associated with Just Meeting Alternative $PM_{10-2.5}$ Standards

9.1 Base case: Health risk reductions associated with just meeting alternative $PM_{10-2.5}$ standards

In this part of the risk assessment, we estimated health risk reductions associated with just meeting alternative $PM_{10-2.5}$ standards in Detroit, Seattle, and St. Louis. Only daily standards were considered. One set of standards was based on the ninety-eighth percentile daily value, and another set was based on the ninety-ninth percentile value. The alternative daily $PM_{10-2.5}$ standards considered in this part of the risk assessment are given in Exhibit 9.1.[33] The design values used to calculate percent rollbacks necessary to just meet alternative $PM_{10-2.5}$ standards were given in Exhibit 2.5. The resulting estimated risk reductions are given in Figures 9.1 through 9.4 and Exhibits 9.2a and 9.2b for Detroit and Appendix G for Seattle and St. Louis.

Exhibit 9.1. Alternative $PM_{10-2.5}$ Standards Considered in the $PM_{10-2.5}$ Risk Assessment*

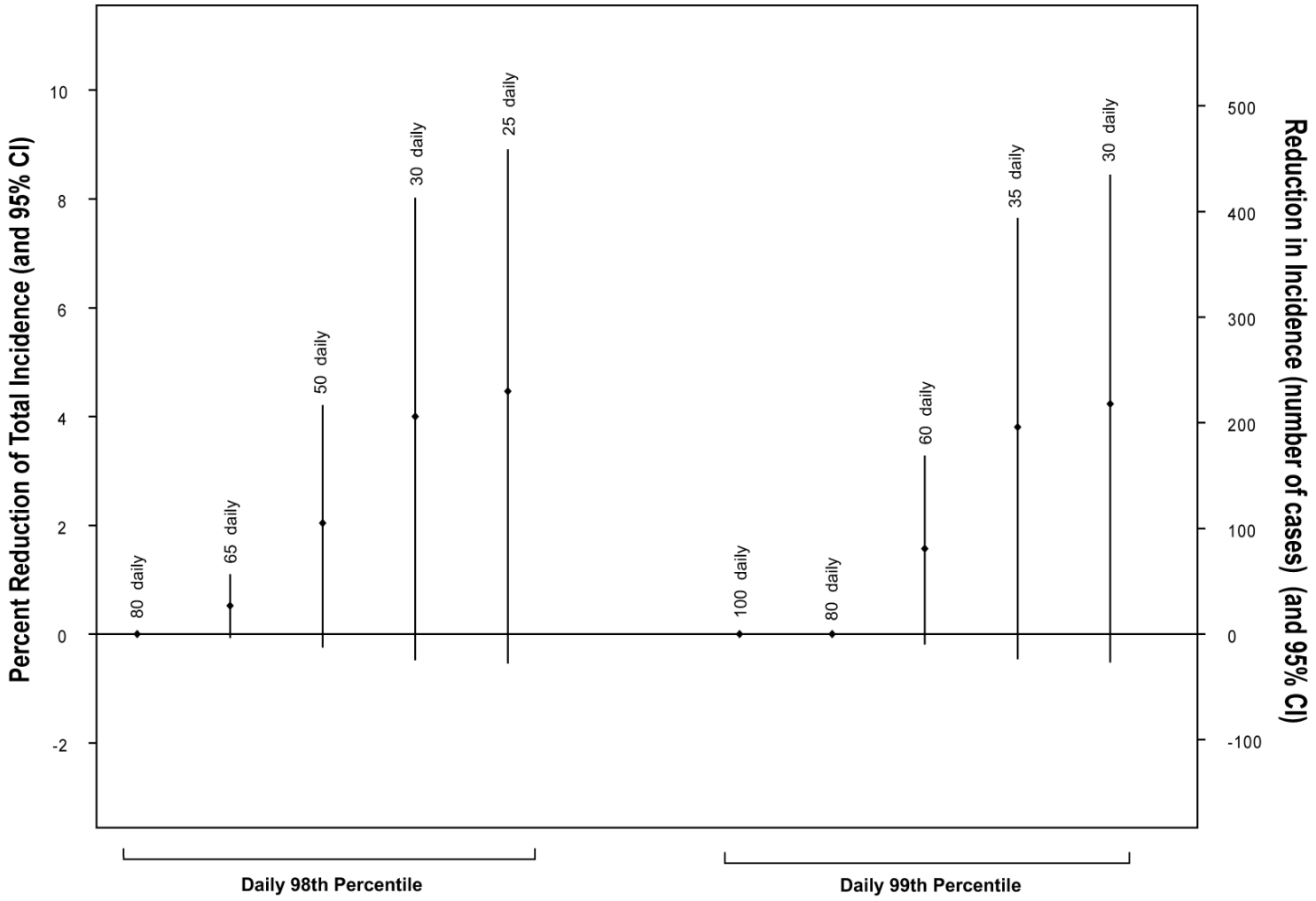
Daily Standards Based on the 98th Percentile Value	Daily Standards Based on the 99th Percentile Value
80	100
65	80
50	60
30	35
25	30

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

Reductions in incidence associated with just meeting an alternative standard in these locations were calculated as the reduction in incidence if "as is" $PM_{10-2.5}$ were rolled back to just meet the alternative standard. For example, using Ito (2003) we estimated that just meeting a $PM_{10-2.5}$ daily standard of 50 $\mu g/m^3$ based on the 98th percentile value in St. Louis would result in 105 hospital admissions for pneumonia avoided (Exhibit G.6a).

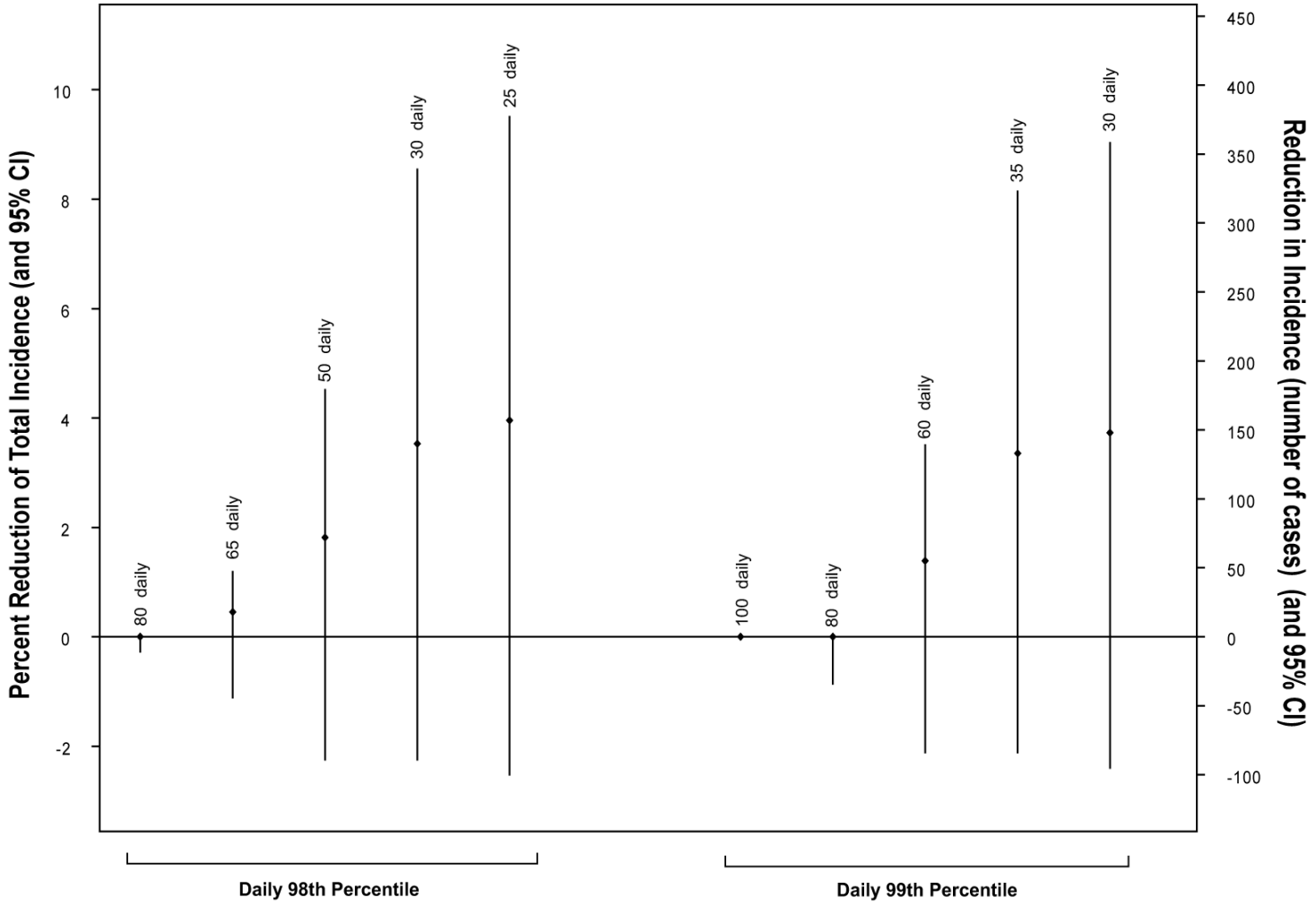
[33] See U.S. EPA (2005) for a discussion of the rationale for selecting these alternative standards.

Figure 9.1. Estimated Annual Reduction of Hospital Admissions for Pneumonia Associated with Rolling Back "As Is" $PM_{10-2.5}$ Concentrations to $PM_{10-2.5}$ Concentrations that Just Meet Alternative $PM_{10-2.5}$ Daily Standards: Detroit, MI, 2003*



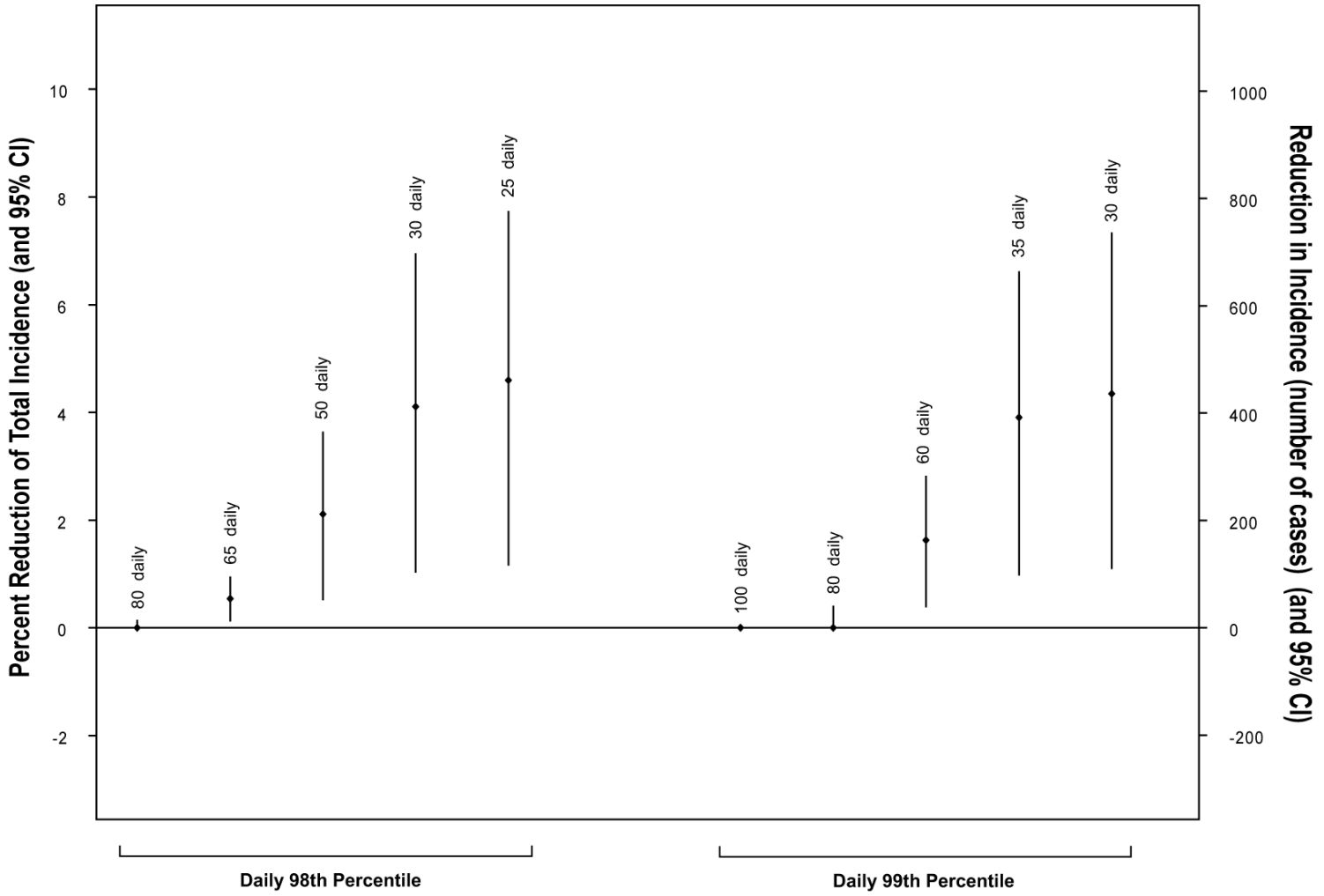
*Based on Ito (2003)

Figure 9.2. Estimated Annual Reduction of Hospital Admissions for COPD Associated with Rolling Back "As Is" $PM_{10-2.5}$ Concentrations to $PM_{10-2.5}$ Concentrations that Just Meet Alternative $PM_{10-2.5}$ Daily Standards: Detroit, MI, 2003*



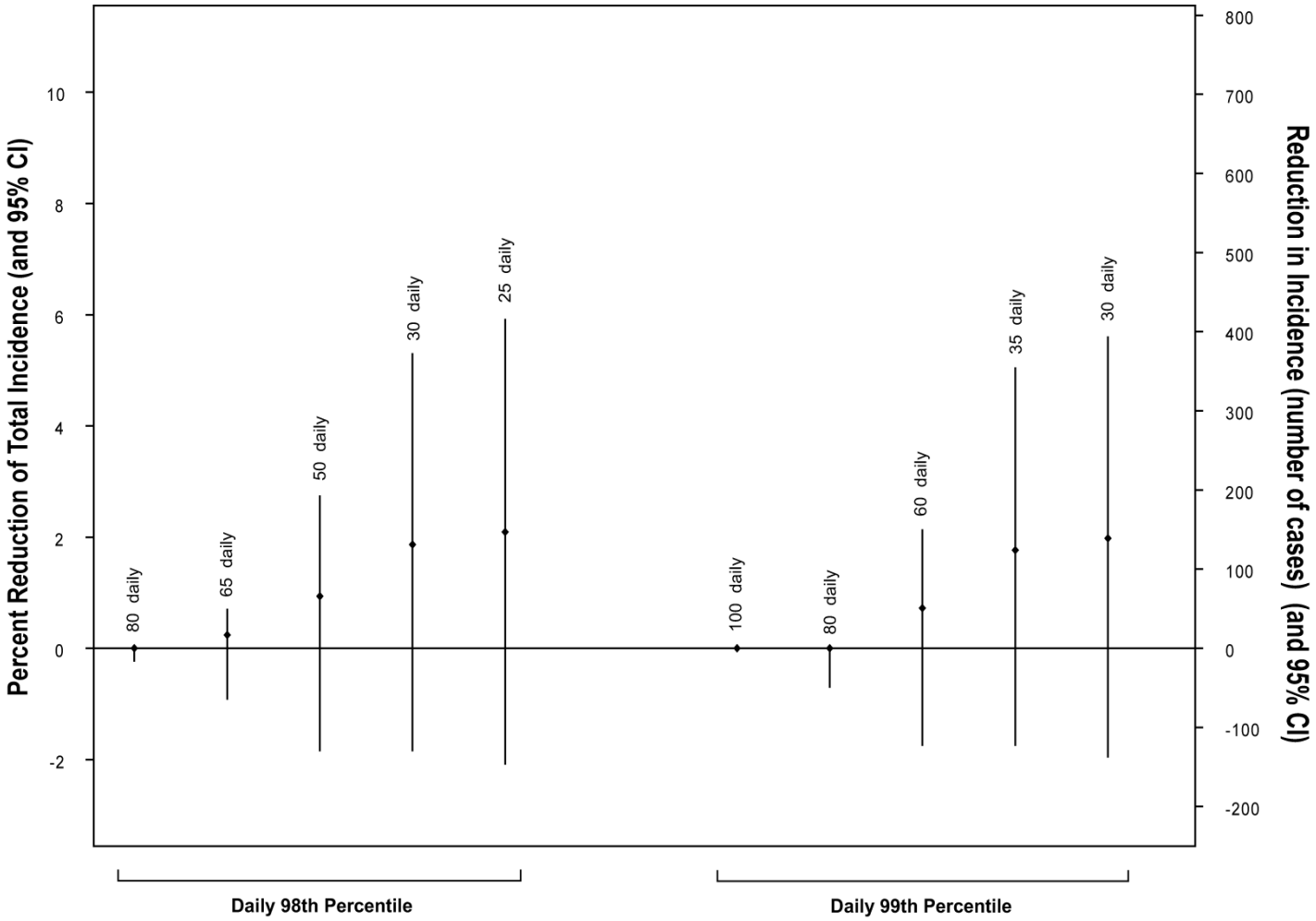
*Based on Ito (2003)

Figure 9.3. Estimated Annual Reduction of Hospital Admissions for Ischemic Heart Disease Associated with Rolling Back "As Is" $PM_{10-2.5}$ Concentrations to $PM_{10-2.5}$ Concentrations that Just Meet Alternative $PM_{10-2.5}$ Daily Standards: Detroit, MI, 2003*



*Based on on Ito (2003)

Figure 9.4. Estimated Annual Reduction of Hospital Admissions for Congestive Heart Failure Associated with Rolling Back "As Is" $PM_{10-2.5}$ Concentrations to $PM_{10-2.5}$ Concentrations that Just Meet Alternative $PM_{10-2.5}$ Daily Standards: Detroit, MI, 2003*



*Based on on Ito (2003)

Exhibit 9.2a. Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{10-2.5}$ Standards: Detroit, MI, 2003*

Exhibit 9.2b. Estimated Changes in Health Risks Associated with Just Meeting Alternative $PM_{10-2.5}$ Standards: Detroit, MI, 2003*

As noted in Section 2, estimated risk reductions are determined by changes only at the composite monitor for a location and only for a single year (2003, in the case of $PM_{10-2.5}$). The percent reduction of $PM_{10-2.5}$ concentrations at the composite monitor to just meet a standard, however, is determined by the design value for that location based on data from 2001 - 2003. (EPA design values for 98th and 99th percentile daily $PM_{10-2.5}$ standards are given in Exhibit 2.5.) In Detroit, the design value for the 98th percentile daily $PM_{10-2.5}$ standards is 70 $\mu g/m^3$ whereas the 98th percentile daily value in 2003 based on the measured $PM_{10-2.5}$ values is 105.9 $\mu g/m^3$. Because the design value is lower than 80 ug/m^3, the highest 98th percentile daily $PM_{10-2.5}$ standard, zero risk reductions were estimated to result from this standard, even though the 98th percentile daily value at the composite monitor in 2003, 105.9 $\mu g/m^3$, is well above the standard. Similarly, the design value for the 99th percentile daily $PM_{10-2.5}$ standards is 77 $\mu g/m^3$ for Detroit, whereas the 99th percentile daily value at the composite monitor in Detroit in 2003 is substantially greater than 100 $\mu g/m^3$, the highest 99th percentile daily $PM_{10-2.5}$ standard. So zero risk reductions were similarly estimated to result from this standard. In general, estimated risk reductions increase and the confidence intervals around the estimates widen as the daily standards become more stringent.

9.2 Sensitivity Analyses: Health risk reductions associated with just meeting alternative $PM_{10-2.5}$ standards under different hypothetical threshold assumptions

As with $PM_{2.5}$, we carried out sensitivity analyses to examine the impact of different hypothetical threshold assumptions on estimated risks associated with just meeting each of the alternative $PM_{10-2.5}$ standards considered in Section 9.1. Because mortality was not considered in the $PM_{10-2.5}$ risk assessment, we selected the following health endpoints associated with short-term exposure to $PM_{10-2.5}$: hospital admissions for ischemic heart disease in Detroit; hospital admissions for asthma (age < 65) in Seattle; and days of cough among children in St. Louis. Hypothetical thresholds of 10, 15, and 20 $\mu g/m^3$ were considered. The results of these sensitivity analyses for Detroit are shown in Exhibit 9.3. The results for the other two locations are shown in Appendix H.

The same patterns can be observed in the results for $PM_{10-2.5}$ as were observed in the results for $PM_{2.5}$. Estimated $PM_{10-2.5}$-related incidences varied substantially with both hypothetical threshold assumptions and alternative standards. For example, the estimated number of hospital admissions for ischemic heart disease associated with short-term exposure to "as is" $PM_{10-2.5}$ concentrations above PRB in Detroit decreases from 654 under the assumption of no threshold, to 569, 489, and 426 under hypothetical threshold assumptions of 10, 15, and 20 $\mu g/m^3$, respectively. Going from "as is" concentrations to just meeting the most stringent standards considered (30 $\mu g/m^3$ daily 99th percentile value) results in a reduction in cases of (654 - 218)/654 = 66.7 percent under the assumption of no threshold, but under the assumption of a threshold of 10 $\mu g/m^3$ it results in a reduction of (569 - 103)/569 = 81.9 percent. Under hypothetical thresholds of 15 and 20 $\mu g/m^3$, the reductions are 89.6 percent and 92.0 percent, respectively. The same general patterns can be seen in the other two locations.

Exhibit 9.3. Sensitivity Analysis: Estimated Annual Hospital Admissions for Ischemic Heart Disease Associated with Short-Term Exposure to $PM_{10-2.5}$ When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models: Detroit, MI, 2003

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