Particulate Matter NAAQS

Risk Analysis Scoping Plan

DRAFT

Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina

June 2001

DISCLAIMER

This draft scoping plan has been prepared by staff from the Health and Ecosystems Effects Group, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, in conjunction with Abt Associates Inc. (through Contract No. 68-D-98-001, WA 3-51). Any opinions, findings, conclusions, or recommendations are those of the authors and do not necessarily reflect the views of the U.S. EPA or Abt Associates. This document is being circulated to obtain review and comment from the Clean Air Scientific Advisory Committee (CASAC) and the general public. Comments on this document should be addressed to Harvey Richmond, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, MD-15, Research Triangle Park, North Carolina 27711.

Particulate Matter NAAQS Risk Analysis Scoping Plan

I. Introduction

As part of the Particulate Matter (PM) National Ambient Air Quality Standard (NAAQS) review completed in 1997, the U.S. EPA's Office of Air Quality Planning and Standards (OAQPS) sponsored a risk analysis for two sample urban areas, Philadelphia and Los Angeles Counties, to assess the effects of alternative PM standards on reducing estimated health risks attributable to PM (U.S. EPA, 1996; Deck et al., 2001; Post et al., 2001; and Abt Associates Inc., 1996, Abt Associates Inc., 1997a,b).

As part of the next periodic review of the PM NAAQS, EPA is in the process of updating its assessment of the health effects literature which is contained in the March 2001 external review draft of the *Air Quality Criteria for Particulate Matter, hereafter 2001* PM CD (EPA, 2001). The proposed risk analyses will be based on the health effects evidence assessed in the 2001 PM CD and those studies included in the previous 1996 risk analyses.

This PM NAAQS Risk Analysis Scoping Plan is designed to outline proposed approaches and highlight key issues in the estimation of the health risks posed by PM under existing air quality levels ("as is" health risks) and if various alternative standards were met in selected sample urban areas. This plan is intended to facilitate review by the Clean Air Scientific Advisory Committee (CASAC) and the general public and to obtain advice on the proposed approaches and key issues in advance of the completion of such analyses and presentation of results in the next draft of the OAQPS Staff Paper.

II. Framework for Health Risk Analysis

A. Overview

The primary purpose of the PM health risk analysis project is to provide quantitative estimates of the risk to public health associated with existing air quality levels and with air quality levels that would occur if current and alternative PM standards were met. As part of such an analysis, explicit and, where possible, quantitative characterizations of the uncertainties in the resulting risk estimates will be developed, as well as information on background incidence rates for the health effects endpoints considered in the analyses. Such information is intended to assist the Administrator in selecting primary PM standards that will protect the public health with an adequate margin of safety, recognizing that such standards will not be risk-free.

For fine particulate matter ($PM_{2.5}$), the proposed risk analyses will focus on the most important health effect endpoints from the standpoint of public health significance and for which the weight of the evidence supports the judgment that the effect category is likely caused by

exposure to $PM_{2.5}$ either alone and/or in combination with other pollutants. The staff has judged the following health effect categories as meeting these criteria for inclusion in the planned risk analyses: short- and long-term exposure non-accidental mortality, respiratory and cardiovascular daily mortality, hospital admissions for respiratory and cardiovascular causes, and short- and long-term respiratory illnesses or symptoms in children. Although the risk analyses will not address all of the various health effects for which there is some evidence of association with exposure to $PM_{2.5}$, all such effects are identified and considered in the OAQPS Staff Paper.

In 1997 EPA established PM_{10} and $PM_{2.5}$ standards to address the health effects associated with both fine- and coarse-fraction particles. Since completion of the last review of the PM NAAQS, a number of health effects studies have examined the association of various health endpoints with the coarse fraction of PM using $PM_{10-2.5}$ as the indicator. For coarse-fraction particles the strongest evidence is found relating $PM_{10-2.5}$ ambient concentrations and increased respiratory hospital admissions and respiratory symptoms. EPA is considering the appropriateness of conducting risk analyses for these two health effect categories for recent air quality levels and upon just meeting potential $PM_{10-2.5}$ standards. EPA is soliciting CASAC and public input on this issue. The discussion below includes information on studies and concentration-response functions for both $PM_{2.5}$ and $PM_{10-2.5}$ to help inform a decision on whether or not to proceed with a coarse-fraction risk analysis. Similarly, air quality information for both $PM_{2.5}$ and $PM_{10-2.5}$ and possible urban counties that would be selected for such analyses also are included in this plan.

The proposed PM health risk analyses are similar in many respects to the health risk analyses conducted as part of the prior PM NAAQS review, which were reviewed by CASAC (Wolfe, 1996). Both the prior and the current proposed PM risk analyses:

- estimate risks for sample urban areas, rather than attempt a nationwide analysis.
- analyze risks under a recent year of air quality (labeled “as is”) and under a situation where air quality just attains various alternative standards under consideration.
- estimate risks only for concentrations exceeding an estimated background level.

The PM health risk model combines information about PM air quality for a specific urban area with concentration-response (C-R) functions derived from epidemiological studies and baseline health incidence data for specific health endpoints to derive estimates of the annual incidence of specified health effects attributable to “as is” PM concentrations and the reduction in incidence that would result upon just meeting specified PM standards.

Both the prior and the planned PM risk analyses focus on health endpoints for which C-R functions have been estimated in epidemiological studies. Since these studies estimate C-R functions using air quality data from fixed-site, population-oriented monitors, the appropriate application of these functions in a PM risk analysis similarly requires the use of air quality data at

fixed-site, population-oriented monitors. This approach was taken in the prior PM risk analyses and is proposed for the current PM risk analyses.

The planned PM risk analyses are intended to provide additional insight into the extent to which at-risk populations experience specific health effects when various alternative standards are just met. The staff believes that such information, when available, is useful to inform judgments about alternative standards designed to protect public health with an adequate margin of safety. The staff recognizes that due to the many sources of uncertainty inherent in such analyses, any risk or risk reduction estimates should not be interpreted as precise measures of risk or risk reduction. Some of the major uncertainties are highlighted in the discussion below of the proposed structure of the risk analyses, and are also discussed in the section on "Characterization of Uncertainty."

B. Structure of Risk Analysis

In order to estimate the incidence of a given health effect associated with "as is" conditions in a given county and the change in incidence of the health effect in that county corresponding to a given change in PM levels resulting from just meeting alternative standard scenarios, the following three elements are required:

- **Air Quality Information** including: (1) "as is" air quality data for $PM_{2.5}$ from population-oriented monitors for the selected cities, (2) estimates of background $PM_{2.5}$ concentrations appropriate to those locations, and (3) a method for adjusting the "as is" data to reflect patterns of air quality change estimated to occur when the county meets various alternative standards. To carry out a $PM_{10-2.5}$ risk analysis, "as is" data for $PM_{10-2.5}$ (i.e., both PM_{10} and $PM_{2.5}$ data from co-located population-oriented monitors) and estimates of background $PM_{10-2.5}$ are required.

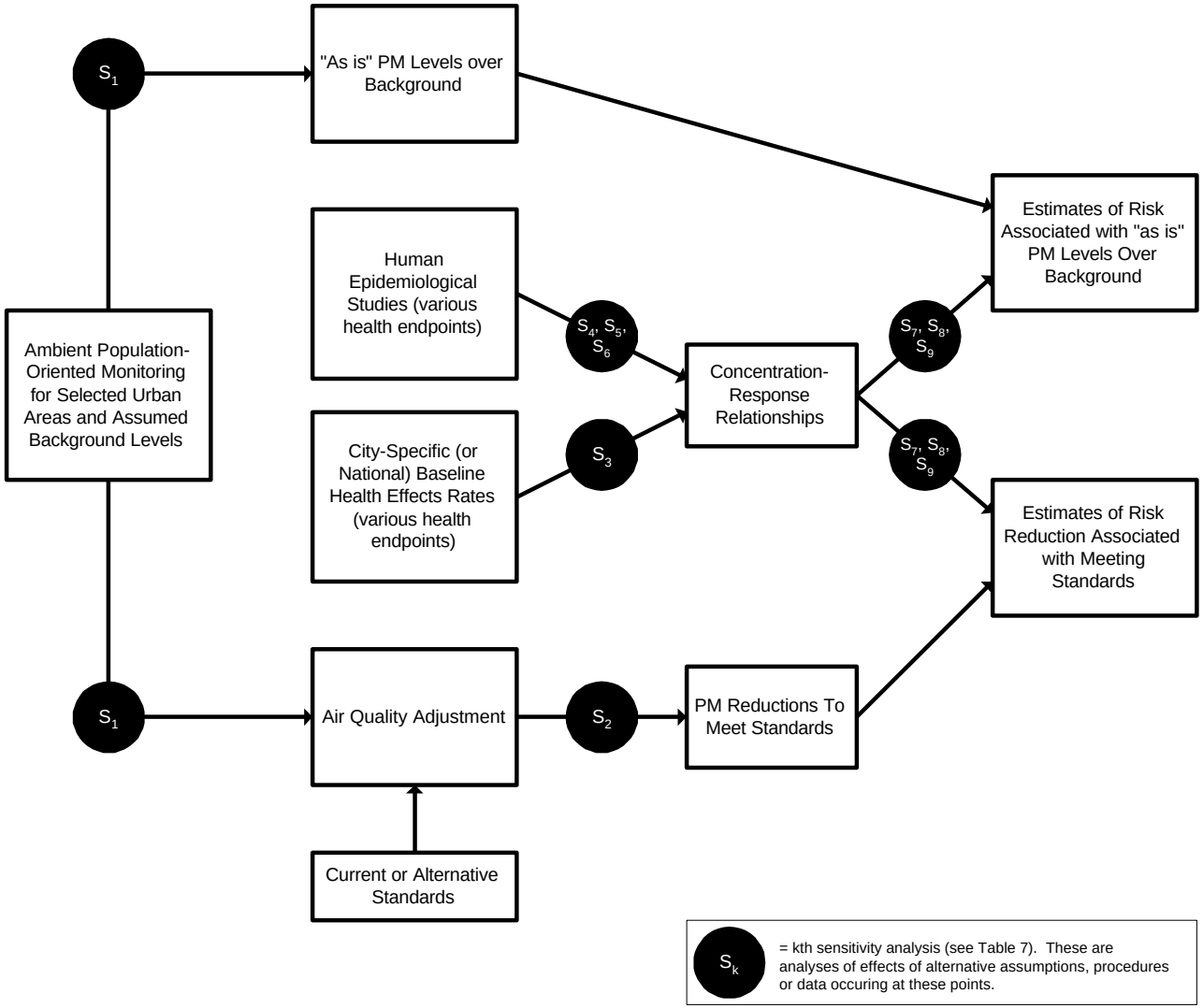
- **Concentration-Response Function(s)** which provide an estimate of the relationship between the health endpoint of interest and PM concentrations.

- **Baseline Health Effects Incidence or Incidence Rate** which provides an estimate of the incidence or incidence rate of the health effect corresponding to "as is" PM levels.[1]

Figure 1 provides a broad schematic of the role of these components in the risk analysis.

[1] If incidence rates are used, they must be used in conjunction with estimates of population in the assessment location(s). Estimates of the changes in incidence of a health effect associated with changes in PM concentrations require as an input an estimate of the baseline incidence of the health effect.

Figure 1. Major Components of Particulate Matter Health Risk Analysis



The most common form of health risk model is given in equation 1, which shows the relationship between changes in PM air quality concentrations (Δx) and changes in the incidence of the health effect (Δy), based on the concentration-response relationship (reflected by β, the PM coefficient derived from epidemiology studies), and the baseline health effects incidence (y).[2]

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

Estimates of risk (i.e. incidences or incidence rates of health effects attributable to PM) will be quantified for PM concentrations above background except for those studies in which the background concentration was not within the range of observable PM concentrations used for the study (e.g., the prospective cohort mortality studies). For these studies effects will be quantified only down to the lowest concentrations observed in the study. A more detailed discussion of the proposed methodology for the PM risk analysis will be presented in "Proposed Methodology for PM Risk Analyses in Philadelphia and Los Angeles" (Abt Associates, 2001), which is currently under preparation. Generally, the methods will be very similar to those relied upon in the 1996 PM risk analyses (Deck et al., 2001; Post et al., 2001; Abt Associates, 1996, 1997a,b). The sections below discuss the key elements of the risk analysis, highlighting those points at which judgments must be made that will determine the nature and scope of the analysis.

C. Selection of Cities and Years to Include in the Analysis

Several objectives were considered in selecting or proposing the urban areas for which to conduct the analyses of $PM_{2.5}$ and $PM_{10-2.5}$ health risks. These general objectives were:

(1) completeness of PM air quality data (referring to both the frequency of monitoring and the number of monitoring sites),

(2) that the analysis include cities where health effect epidemiological studies were conducted,

(3) that the cities include representatives of various PM aerosol mixes (e.g. Eastern cities, Western cities, areas with windblown dust);[3] in addition, that the cities

[2] The health risk model given in equation 1 is based on a concentration-response function in which the natural logarithm of the incidence of the health effect is a linear function of PM concentration.

[3] If different PM aerosol mixes present different health risks, these different health risks will be captured in the PM risk analysis if C-R functions estimated in the different risk analysis locations can be used. If functions have not been estimated in the different assessment locations, health risk differences resulting from different PM aerosol mixes may not be captured in the risk analysis.

provide a representative range of PM concentrations observed in the U.S. (i.e., at least one site with high pollution levels),

(4) recentness of air quality data, and

(5) availability of location-specific information on baseline incidence rates.

Because baseline mortality incidence data are available at the county level, this is not a binding constraint in the selection of urban counties for the PM risk analyses. Information on the incidence of respiratory symptoms and illnesses not requiring hospitalization, in contrast, is generally not available, except in those locations in which studies happen to have been conducted. Data on hospital admissions for recent years, however, specific to International Classification of Disease (ICD) codes, are available in some cities but not others. This category of incidence data was therefore a consideration in the selection of cities to include in the analysis. The most important consideration, however, was sufficient and recent air quality data.

Selection of Urban Counties and Years to Include in the Analysis of $PM_{2.5}$ Health Risks

Based on the above five objectives, Philadelphia and Los Angeles, the two urban counties analyzed in the prior risk analyses, are proposed as locations at which to assess the potential health risks of $PM_{2.5}$ in the current analyses.

In the 1996 risk analyses, air quality data collected from September 1992 through August 1993 at three Harvard School of Public Health monitors were used in the analysis of Philadelphia. Data for Los Angeles came from two monitors maintained by California's South Coast Air Quality Management District (SCAQMD), which had undertaken a special intensive monitoring effort in 1995. The numbers of days on which there was an observation at one or more of the monitors (and therefore an observation at the "composite monitor" -- the average of all monitors reporting on a given day) for PM_{10} and $PM_{2.5}$ in Philadelphia and Los Angeles are shown in Table 1.

Table 1. Numbers of Days with PM Air Quality Observations in the 1996 Risk Analysis

Urban Area	Year of Data	PM_{10}	$PM_{2.5}$
Philadelphia	1992/93	358	352
Los Angeles	1995	215	214

Although 1992/93 and 1995 were quite recent for the 1996 risk analyses, they are less recent for the planned (2001) risk analyses. The Harvard School of Public Health has not collected PM data in Philadelphia since the 1992/1993 data were collected. EPA's Aerometric Information Retrieval System (AIRS), however, contains $PM_{2.5}$ data in Philadelphia for 276 days in 1999.

In Los Angeles, the SCAQMD undertook a second period of intensive data collection for a year-long period that crossed calendar years 1998 and 1999. In addition, there are $PM_{2.5}$ data from AIRS monitors in Los Angeles. If data from both the SCAQMD monitors and the AIRS monitors are used, there are 199 days of observations for $PM_{2.5}$. Table 2 summarizes the numbers of days with available $PM_{2.5}$ data for recent years, as well as the annual average and 98th percentile daily average $PM_{2.5}$ concentrations at the "composite monitor", in both cities. The composite monitor in Philadelphia is a composite of those AIRS monitors in Philadelphia County that had at least 8 observations in each quarter of the year (AIRS monitors 41, 471, and 1361). The composite monitor in Los Angeles is a composite of those monitors in Southeast Los Angeles County that had at least 15 observations in each quarter (any monitor that had any observations in each quarter had at least 15 observations per quarter in Los Angeles). This includes the two SCAQMD monitors used in the prior risk analyses as well as two AIRS monitors (21 and 40021).

Table 2. Annual and Quarterly Numbers of Days with $PM_{2.5}$ Data, Annual Averages, and Ninety-Eighth Percentile Values

Urban County - Year of Data	Counts					Annual Average* (g/m^3)	98th Percentile 24-Hr Average** (g/m^3)
	Annual	Q1	Q2	Q3	Q4		
Philadelphia - 1999	272	47	65	80	80	14.9	34.4
Los Angeles - 1998/1999	199	62	64	42	31	24.2	59.5

*To avoid bias due to differential amounts of missing data in different quarters of the year, each annual average is calculated as the average of the four quarterly averages at the composite monitor.

**For the purposes of this scoping plan, this table includes the 98th percentile values at the composite monitors in Philadelphia and Los Angeles. The current form of the 24-hour standard, however, requires that the 98th percentile value *at each monitor* not exceed the standard, and the actual PM risk analyses would be based on adjusting the air quality distribution at the highest monitor.

In addition to substantial recent air quality data, recent ICD code-specific hospital admission data are available for both Philadelphia and Los Angeles. This is discussed more fully in Section F below.

Selection of Urban Counties and Years to Include in the Potential Analysis of $PM_{10-2.5}$ Health Risks

The analysis of the health risks attributable to $PM_{10-2.5}$ requires PM_{10} and $PM_{2.5}$ data at co-located monitors. Because $PM_{10-2.5}$ tends to be a larger proportion of PM_{10} in the Western United States than in the East, we prefer to include at least one Western urban county in this part of the risk analysis. Urban counties that have sufficient co-located PM_{10} and $PM_{2.5}$ AIRS data for 1999 that are currently being considered for a $PM_{10-2.5}$ risk analysis, in addition to Los Angeles, are Las Vegas and Salt Lake City. The annual and quarterly counts of days on which there are $PM_{10-2.5}$ data, as well as the annual average levels of $PM_{10-2.5}$, are shown in Table 3.

Table 3. Annual and Quarterly Numbers of Days with $PM_{10-2.5}$ Data, Annual Averages, and Ninety-Eighth Percentile Values

Urban County	Counts[a]					Annual Average $PM_{10-2.5}$ (g/m^3)	98th Percentile $PM_{10-2.5}$ (g/m^3)
	Annual	Q1	Q2	Q3	Q4		
Los Angeles[b]	130	61	32	16	21	26.2	54
Las Vegas[c]	112 275	27 39	29 68	28 82	28 86	12.3 31.3	39 66
Cleveland[c]	107 286	20 72	28 82	30 74	29 58	23.8 21.6	76 56
Salt Lake[c]	285	76	70	68	71	15.8	44

[a] The year for which we have data for Los Angeles August 18, 1998 - August 17, 1999. The quarters are therefore the first 91 days, the second 91 days, etc. Data for Las Vegas, Cleveland, and Salt Lake are for the calendar year 1999. The quarters for these cities refer to the usual quarters of a year (e.g., the first quarter is January - March).

[b] The counts are the numbers of days on which at least one monitor has co-located PM_{10} and $PM_{2.5}$ data.

[c] Information is shown only for those sites with co-located data for at least 100 days in the year and at least 11 days in each quarter. There may be other sites with data that would be included in a "composite monitor" for the urban county.

A consideration in selecting one or more of these urban areas to be included in a possible analysis of the health risks attributable to $PM_{10-2.5}$ is the availability of location-specific ICD code-specific hospital admissions data. While there were sufficient $PM_{10-2.5}$ data for the urban county that includes Cleveland, OH there was no available database to estimate baseline hospital admission rates. OAQPS is considering Los Angeles County and Salt Lake County as the urban

study areas for the potential $PM_{10-2.5}$ risk analysis, given the availability of relatively complete $PM_{10-2.5}$ data, the availability of baseline hospital admission rates, and the population sizes of these two counties.

D. Air Quality Considerations

Estimating Air Quality Concentrations Under "As is" Air Quality

Most C-R functions reported in epidemiological studies are estimated relationships between daily average PM, averaged across monitors in the study area, and daily incidence of the health effect being studied. Analogously, the average PM concentration across all population-oriented PM monitors for a county in the PM risk analysis will be calculated for each day, barring situations of obvious bias. If not all monitors have PM data for a given day, the average PM concentration will be based on those monitors that do have data for that day. Information will be provided as to how air quality varies among monitors in the risk analyses methodology report.

As can be seen in Tables 2 and 3, however, there are some days for which no monitors in a county have $PM_{2.5}$ (or $PM_{10-2.5}$) data. Since the proposed presentation of results will report changes in the total annual incidence of health effects associated with short-term and long-term exposures for a particular year in each county, simply summing up daily changes in incidence on those days for which air quality data are available would result in downward biased risk estimates. Adjustments will therefore be required to estimate the annual effects of $PM_{2.5}$ (or $PM_{10-2.5}$) on health. 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 (365) 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. If, however, monitoring frequency changed significantly within the year during which air quality data were collected, adjustments will be made to the different periods separately to minimize the chance of bias in the final estimate of health effects incidence.

Estimating Background Concentrations

Since health risks will be calculated only for concentrations exceeding estimated background levels, estimates of background $PM_{2.5}$ and $PM_{10-2.5}$ concentrations in the assessment locations are needed to calculate risk at "as is" concentrations over background and for alternative standard scenarios. The background concentrations used for the Eastern and Western United States will be consistent with the assessment of background concentrations contained in the 2001 PM CD.

Appropriate Adjustment Procedures to Model Attainment of Alternative Standards

To estimate the reductions in health risks that would occur as a result of meeting the current and/or alternative PM standards, it is necessary to project what PM air quality would be in an area just meeting these standards. Any such projection introduces a significant additional degree of uncertainty into the risk analyses. However, it is impossible to analyze potential effects of current or alternative standards on reducing health risk from PM without making some assumptions about the pattern of air quality change. This issue is important because many studies have associated 24-hr concentrations of PM with health effects; thus assumptions about the resultant pattern of 24-hr concentrations that would be observed after meeting current or alternative standard scenarios may have a noticeable impact on the amount of risk reduction estimated.

As a starting assumption, it is proposed that "as is" PM levels in an urban county be adjusted using a proportional change in air quality to just meet alternative standard scenarios in that urban county. This is the method that was used in the prior risk analysis. This "proportional rollback" of air quality values would calculate the amount of reduction required in an air quality statistic (e.g., annual mean, 98th percentile of daily 24-hr concentrations) in excess of background levels to meet the specified regulatory scenario, and reduce all daily air quality values in excess of the background concentration in the original set of "as is" concentrations by the same proportion as that required by the concentration or average that makes up the statistic itself. If different standards in a set of standards (e.g., a daily standard and an annual standard) require different percent rollbacks, the largest percent rollback would be the controlling one (i.e., it would be necessary to "roll back" all daily air quality values by the largest percentage necessary to meet all the standards in a specified standard scenario).

There are other ways the attainment of alternative standards might be modeled. For example, one alternative could be a rollback in which extreme values were reduced more than annual mean concentrations (i.e., peak concentrations are sizably reduced with little change to the rest of the distribution). Sensitivity analyses will be provided to help bound the potential differences in risk reduction estimated under different assumed patterns of air quality change (i.e., proportional rollbacks versus other possibilities).[4]

It is expected that variations in rollback procedure will have little or no impact on the rolled back annual mean PM concentration, and therefore little or no impact on the estimates of health risk reductions associated with changes in annual mean PM. However, for those alternative standard scenarios in which the 24-hr standard is the controlling form of the standard, differences in patterns of air quality reduction may lead to differences in the estimated health risk reduction.

---

[4] Analyses of historical $PM_{2.5}$ data from monitors with at least two consecutive years of data during the 1980s and early 1990s supports the hypothesis that changes in the distribution of daily $PM_{2.5}$ concentrations from one year to the next are well modeled as linear. Proportional rollbacks may therefore reasonably model the pattern of $PM_{2.5}$ air quality reductions observed. See Abt Associates Inc. July 3, 1996 (Revised November 1996), Section 8.2.

E. Concentration-Response Considerations

The OAQPS staff has selected for inclusion in the PM risk analyses the most significant health effect endpoints for which the weight of the evidence is supportive of an effect occurring. In cases where all of the available studies failed to find a statistically significant relationship, the effect endpoint was excluded. In situations where there is a mixture of statistically significant and non-significant findings for a given health effect endpoint and PM indicator (e.g., hospital admisisons for COPD patients and $PM_{2.5}$), staff also considered evidence from available PM_{10} studies in making a judgment on whether effects are likely related to PM.

The health endpoints that are proposed to be included in the $PM_{2.5}$ analyses include mortality (due to short- and long-term exposure), hospital admissions, emergency room visits, and respiratory illnesses and/or symptoms not requiring hospitalization. (Lung function studies will not be included.) Inclusion of a health endpoint in the analysis will be based on the weight of the evidence overall. Once it has been determined that a health endpoint will be included in the analysis, inclusion of a study on that health endpoint will not be based on statistical significance. That is, consistent with the approach taken in the prior PM risk analyses, no credible study on an included health endpoint will be excluded from the analysis on the basis of lack of statistical significance.

For the potential $PM_{10-2.5}$ risk analyses, EPA is considering increased respiratory-related hospital admissions and increased respiratory symptoms. These are the two health effect categories with the strongest evidence for effects being associated with $PM_{10-2.5}$ exposure. While there is evidence for other effects being associated with $PM_{10-2.5}$, the staff believes that the evidence is insufficient to justify conducting a quantitative risk analysis for these other health endpoints. These other effects are addressed qualitatively in the OAQPS PM Staff Paper.

Study Selection Criteria

In selecting studies to be considered for use in the PM risk analyses, the staff set forth several criteria, all of which had to be met to be included for consideration in the planned risk analyses for this review. These include:

- studies conducted within the United States or Canada that are listed in Tables 9-3, 9-4, and/or 9-6 of the March 2001 PM CD;
- the measure of particulate matter was $PM_{2.5}$ or $PM_{2.1}$ (or, in analyses of the coarse fraction, $PM_{10-2.5}$); and
- $PM_{2.5}$ was measured rather than estimated on a reasonable proportion of the days in the study

The staff recognizes that the draft CD is currently under review by the CASAC and general public, and, thus, the final group of studies to be included in the planned risk analyses may change based on the review of the draft PM CD.

Selection of a Single Concentration-Response Function from a Study

Studies often report more than one estimated C-R function for the same location. Sometimes models including different sets of co-pollutants are estimated in a study; sometimes different lags are estimated. To select a single model from a study that reports more than one estimated C-R function, we will be guided by the following preferences:

- single-pollutant models are preferred (for the base analysis) to multi-pollutant models; and
- if several lag models have been estimated, the model that results in the greatest predicted relative risk is preferred.

We propose to use single pollutant C-R functions in the base analysis and then conduct a sensitivity analysis with multi-pollutant C-R functions. There are potential problems with either category of model. Omitting from the model other pollutants that are (positively) correlated with PM and with the health endpoint will contribute to an upward bias in the estimated PM coefficient. (This does not mean that the estimated PM coefficient will necessarily be biased upward, however, because there may be downward biases from other sources.) Including these correlated pollutants in the model, however, will tend to inflate the variance of the estimator of the PM coefficient, and may inflate it substantially. An estimator with a large variance means that the *actual value of any estimate* of the coefficient could be greatly off, either much too high or much too low -- with the consequent possibility that we could greatly *understate* the risk associated with PM. To avoid this possibility, we propose to rely on single-pollutant models in the base analysis.

There is recent evidence (Samet et al., 2000), that the relationship between PM and health effects may best be described by a distributed lag (i.e., the incidence of the health effect on day n is influenced by PM concentrations on day n, day n-1, day n-2 and so on). If this is the case, a model that includes only a single lag (e.g., a 0 day lag or a 1 day lag) is likely to understate the total impact of PM. Because of this, when a study reports several estimated lag models, the one that produces the greatest relative risk is likely to minimize the degree of understatement of models that include only one lag at a time.

Concentration-Response Functions Proposed for Use in the PM Risk Analysis

Based on the study selection criteria and the model selection criteria discussed above, we propose to select C-R functions to be used in the PM risk analysis from among the $PM_{2.5}$ and $PM_{10-2.5}$ C-R functions shown in Tables 4 through 6. Tables 4 and 5 give short-term ("daily") and long-term ("chronic") exposure C-R functions, respectively, for $PM_{2.5}$. Table 6 gives C-R

functions for $PM_{10-2.5}$. As discussed more fully below, not all C-R functions in a given health effect category will necessarily be pooled together. In some cases, the study-specific estimates may be reported separately. A few studies were not included because they report results based on a subset of a more comprehensive dataset for which results are reported in another paper (e.g., Laden et al., 2000 and Schwartz 2000 both rely on a subset of the data used in Schwartz et al., 1996). Although these studies may consider new issues or new analytical techniques, these new considerations were not considered relevant to the proposed PM risk analyses.

Table 4. $PM_{2.5}$ Concentration-Response Functions With Short-Term Exposure for Potential Use in the PM Risk Analysis

Short-term exposure non-accidental mortality

Study*	Population (age)	Location/Years	Model	Lag/Exposure Metric**	Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)
Burnett et al. (2000)	All ages	1986-96, 8 Canadian cities	Log-linear	1	3.0 (1.1, 5.0)
Fairley (1999)	All ages	1989-96, Santa Clara, CA	Log-linear	0	8.5 (3.2, 14.0)
Goldberg et al. (2000)	All ages	1984-93, Montreal, Canada	Log-linear	1	2.9 (-0.1, 6.0)
Lipfert et al. (2000)	All ages	1991-95, Philadelphia, PA	Linear	2 day avg	4.2[1]
Lippman et al. (2000)	All ages	1992-94, Detroit, MI	Log-linear	3	3.1 (-0.6, 7.0)
Mar et al. (2000)	All ages	1995-97, Phoenix, AZ	Log-linear	n/a	6.0 (0.0, 15.4)
Moolgavkar (2000a)	All ages	1987-95, Los Angeles, CA	Log-linear	0	1.4 (-0.1, 2.9)
Schwartz et al. (1996)	All ages	1979-88, Boston, St Louis, Kingston/Knoxville, Portage, Steubenville, Topeka	Log-linear	2 day avg	3.8 (2.8, 4.8)
Tsai et al. (2000)	All ages	1981-83, Newark, Camden, and Elizabeth, NJ	Log-linear	0	1.8 - 5.7[4]
Goldberg et al. (2000)	65+	1984-93, Montreal, Canada	Log-linear	1	3.3 (-0.2, 6.9)
Schwartz et al. (1996)	65+	1979-88, Boston, St Louis, Kingston/Knoxville, Portage, Steubenville, Topeka	Log-linear	2 day avg	4.3 (3.0, 5.6)
Smith et al. (2000)	65+	1995-97, Phoenix, AZ	Sqrt transformed linear regression	3 day avg	Depends on starting $PM_{2.5}$

Short-term exposure cardiovascular mortality					
Study*	**Population (age)**	**Location/Years**	**Model**	**Lag/Exposure Metric****	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Fairley (1999)	All ages	1989-96, Santa Clara, CA	Log-linear	0	6.6 (-1.1, 14.9)
Goldberg et al. (2000)	All ages	1984-93, Montreal, Canada	Log-linear	1	3.4 (-1.2, 8.1)
Lipfert et al. (2000)	All ages	1991-95, Philadelphia, PA	Linear	2 day avg	4.3[1]
Lippman et al. (2000)	All ages	1992-94, Detroit, MI	Log-linear	1	3.2 (-2.3, 8.9)
Mar et al. (2000)	All ages	1995-97, Phoenix, AZ	Log-linear	1	18.7 (5.7, 33.2)
Moolgavkar (2000a)	All ages	1987-95, Los Angeles, CA	Log-linear	1	2.6 (0.4, 4.9)
Schwartz et al. (1996)	All ages	1979-88, Boston, St Louis, Kingston/Knoxville, Portage, Steubenville, Topeka	Log-linear	2 day avg	5.3 (3.5, 7.1)
Short-term exposure respiratory mortality					
Study*	**Population (age)**	**Location/Years**	**Model**	**Lag/Exposure Metric****	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Fairley (1999)	All ages	1989-96, Santa Clara, CA	Log-linear	0	12.1 (-2.2, 28.4)
Goldberg et al. (2000)	All ages	1984-93, Montreal, Canada	Log-linear	1	11.9 (1.5, 23.4)
Lipfert et al. (2000)	All ages	1991-95, Philadelphia, PA	Linear	2 day avg	2.2[1]
Lippman et al. (2000)	All ages	1992-94, Detroit, MI	Log-linear	0	2.3 (-10.3, 16.7)
Moolgavkar (2000a)	All ages	1987-95, Los Angeles, CA	Log-linear	1	2.7 (-3.4, 9.1)
Schwartz et al. (1996)	All ages	1979-88, Boston, St Louis, Kingston/Knoxville, Portage, Steubenville, Topeka	Log-linear	2 day avg	8.5 - 10.3[4]
Tsai et al. (2000)	All ages	1981-83, Newark, Camden, Elizabeth, NJ	Log-linear	0	2.3 - 6.2[4]
Goldberg et al. (2000)	65+	1984-93, Montreal, Canada	Log-linear	1	13.1 (1.9, 25.5)

Short-term exposure cardiovascular hospital admissions					
Study*	**Population (age)**	**Location/Years**	**Model**	**Lag/Exposure Metric****	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Burnett et al. (1997)	All ages	Summers of 1992-94, Toronto, Canada	Log-linear	4 day avg, 2 day lag	7.2 (-0.6, 15.6)
Lippman et al. (2000)	All ages	1992-94, Detroit, MI	Log-linear	1-2	3.2 - 9.1[2]
Moolgavkar (2000b)	20-64	1987-95, Los Angeles, CA	Log-linear	0	3.5 (1.8, 5.3)
Moolgavkar (2000b)	65+	1987-95, Los Angeles, CA	Log-linear	0	4.3 (2.5, 6.1)
Short-term exposure respiratory hospital admissions					
Study*	**Population (age)**	**Location/Years**	**Model**	**Lag/Exposure Metric****	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Burnett et al. (1997)	All ages	Summers of 1992-94, Toronto, Canada	Log-linear	4 day avg, 1 day lag	8.6 (3.4, 14.1)
Lippman et al. (2000)	All ages	1992-94, Detroit, MI	Log-linear	1, 3	5.5 - 12.5[2]
Moolgavkar et al. (2000)	All ages	1987-95, King County, WA	Log-linear	3	6.5 (1.3, 11.8)
Thurston et al. (1994)	All ages	Summers of 1986-88, Toronto, Canada	Linear	0	6.1 - 15.0[2]
Moolgavkar (2000c)	0-19	1987-95, Los Angeles, CA	Log-linear	0	4.3 (-0.1, 8.9)
Moolgavkar (2000c)	20-64	1987-95, Los Angeles, CA	Log-linear	2	5.6 (1.9, 9.4)
Moolgavkar (2000c)	65+	1987-95, Los Angeles, CA	Log-linear	2	5.1 (0.9, 9.4)

Short-term exposure cardiovascular emergency department visits					
Study*	**Population (age)**	**Location/Years**	**Model**	**Lag/Exposure Metric****	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Stieb et al. (2000)	All ages	Jul 92-Mar 96, Saint John, Canada	Log-linear	3	15.1 (-0.2, 32.8)
Short-term exposure respiratory emergency department visits					
Study*	**Population (age)**	**Location/Years**	**Model**	**Lag/Exposure Metric****	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Stieb et al. (2000)	All ages	Jul 92-Mar 96, Saint John, Canada	Log-linear	5 day avg, 3 day lag	5.7 (0.6, 11.0)
Delfino et al. (1997)	65+	Summer of 1993, Montreal, Canada	Linear	1	23.9 (4.9, 42.8)
Short-term exposure respiratory illnesses and/or symptoms (that do not require hospitalization)					
Study	**Population (age)**	**Location/Years**	**Model**	**Lag/Exposure Metric****	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Neas et al. (1995) Evening cough	*Children*	*Uniontown, PA*	*Logistic*		See footnote 3.
Neas et al. (1996) Cough, cold	Children	State College, PA	Logistic		See footnote 3
Schwartz and Neas (2000) - Lower respiratory symptoms, Cough	Children (grades 2-5)	Apr-Aug 1984-88, Six Cities	Logistic	1	See footnote 3

* Studies in italics were used in the previous PM risk analyses (U.S. EPA, 1996; Deck et al., 2001; Post et al., 2000; and Abt Associates Inc., 1996, Abt Associates Inc., 1997a,b)

** The lag with the largest percent increase is presented here. Many studies looked at multiple models with different lag structures.

[1] The confidence interval is not readily available.

[2] The range in percent increase represents estimates for different combinations of ICD codes.

[3] Percent increase for the logistic regression depends on the baseline incidence, which is location-specific.

[4] The range in percent increase represents estimates for different locations.

Table 5.	**$PM_{2.5}$ Concentration-Response Functions With Long-Term Exposure for Potential Use in the PM Risk Analysis**			
Long-term exposure total mortality				
Study	**Population (age)**	**Location/Years**	**Model**	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Krewski et al. (2000, Six City Reanalysis)	25+	1974-91, Portage, WI; Topeka, KS; Harriman, TN; Watertown, MA; St. Louis, MO; Steubenville, OH	Log-linear	39.3 (12.3, 70.9)
Krewski et al. (2000, ACS Study Reanalysis)	30+	1982-89, All 50 states	Log-linear	18.4 (9.2, 26.6)
Long-term exposure cardiopulmonary mortality				
Study	**Population (age)**	**Location/Years**	**Model**	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{2.5}$ (95% CI)**
Krewski et al. (2000, Six City Reanalysis)	25+	1974-91, Portage, WI; Topeka, KS; Harriman, TN; Watertown, MA; St. Louis, MO; Steubenville, OH	Log-linear	45.2 (9.5, 92.8)
Krewski et al. (2000, ACS Study Reanalysis)	30+	1982-89, All 50 states	Log-linear	30.7 (17.4, 45.1)

Table 6. $PM_{10-2.5}$ Respiratory-Related Concentration-Response Functions for Potential Use in the PM Risk Analysis					
Study*	**Health Endpoint**	**Population (age)**	**Location/Years**	**Model**	**Percent Increase Associated with a 25 g/m^3 Increase in $PM_{10-2.5}$**
Burnett et al. (1997)	Total respiratory hospital admissions	All ages	Summers of 1992-94, Toronto, Canada	Log-linear	12.7 (5.2, 20.7)
Lippman et al. (2000)	Hospital admissions for pneumonia	All ages	1992-94, Detroit, MI	Log-linear	11.9 (0.6, 24.4)
Lippman et al. (2000)	Hospital admissions for COPD and asthma	All ages	1992-94, Detroit, MI	Log-linear	9.3 (-4.2, 24.7)
Naeher et al. (1999)	Runny or stuffy nose		Southwest Virginia	Logistic	—**
Schwartz and Neas (2000)	Cough, lower respiratory symptoms	School children, grades 2 - 5	Six Cities in U.S.	Logistic	—**
Thurston et al. (1994)	Total respiratory hospital admissions	All ages	Summers of 1986-88, Toronto, Canada	Linear	22.3 (-9.6, 54.2)
Thurston et al. (1994)	Hospital admissions for asthma	All ages	Summers of 1986-88, Toronto, Canada	Linear	12.2 (-8.2, 32.5)

* Studies in italics were used in the previous PM risk analyses (U.S. EPA, 1996; Deck et al., 2001; Post et al., 2001; and Abt Associates Inc., 1996, Abt Associates Inc., 1997a, b).
**Percent increase for the logistic regression depends on the baseline incidence, which is location-specific.

Pooling Estimates from Multiple Studies or Locations

Since the 1996 PM risk analyses were carried out, several new studies have investigated the relationship between PM and a health endpoint (e.g., short-term exposure mortality) in multiple cities using consistent methodological approaches in all locations examined. As noted in the March 2001 PM CD (see, in particular, Section 9.6.2.1.2), such multi-location studies are preferable, all else equal, to meta-analyses of the results of multiple independent single-location studies carried out in different locations. The primary advantage of such multi-location studies is the consistency in methodology used in all locations, eliminating the possibility that inter-locational differences might be due to differences in study design. In addition, multi-location studies are not subject to the omission of negative results due to publication bias that could affect a meta-analysis of the results of published single-location studies. Finally, any geographical variability in air pollution effects can be systematically evaluated in a multi-location study. For

these reasons, such multi-location studies, if available, are preferred to meta-analyses of independent single-location studies.

Consistent with the approach taken in the prior PM risk analyses, if there is no multi-location study for a health endpoint, and if several single-location studies have been identified as appropriate for inclusion in the PM risk analyses, we propose to combine the C-R functions from these studies to form a "pooled" estimate of the risk of that health effect attributable to $PM_{2.5}$ (or $PM_{10-2.5}$) and the risk reductions that would result from meeting current or alternative standards. The relationship between a pollutant and a health effect in a population may vary from one location to another due, for instance, to inter-locational differences in the composition of PM and/or the populations exposed. Pooling the estimates from several studies provides a central tendency estimate of the effect in any randomly selected location, as well as a characterization of the uncertainty about the effect in that location.

To pool estimates from different C-R functions requires that the C-R functions be based on certain underlying similarities. In particular, they should be based on (1) similar population groups, (2) similar definitions of the health endpoint, (3) similar pollutant definitions, and, if possible, (4) similar C-R models. For example, it would be reasonable to pool several studies, each of which estimated a log-linear model of non-accidental daily mortality in an entire population (all ages) as a function of daily same-day (0 lag) $PM_{2.5}$. In practice, however, there are generally differences across studies, ranging from very minor to substantial. Some C-R functions are based on a 0-day lag, for example, while others are based on 1-, 2-, or 3-day lags. One study might use single day (e.g., same day) PM, while another might use a two-day average of PM (i.e., an average of same day and previous day PM) or a three-day average (i.e., an average of same day PM and PM for the previous two days). Some hospital admissions studies define "respiratory illnesses" as a broad group of ICD codes, while others omit specific ICD codes from the group they consider. Because of this, judgment is required in deciding which studies to pool. We will follow some basic guidelines, however. For instance, "all ages" studies will not be pooled with "age 65 and over" studies. However, we may pool studies that use different lag structures or studies that use different averaging times.

If the functional forms of the C-R functions to be pooled are all the same, it is possible to pool the PM coefficients. In this case, the pooled coefficient is then used in conjunction with air quality data to estimate attributable risk (incidence of the health effect associated with PM) and risk reductions (changes in incidence of the health effect associated with changes in PM). If the functional forms differ (e.g., if some C-R functions are log-linear and some are linear), however, we propose to estimate study-specific attributable risk (or risk reduction) and then pool these estimates. Whether the pooling is done in "coefficient space" or in "incidence space," pooling would be done using random effects models.

Prior to pooling, study-location-specific estimates can be improved by using an empirical Bayes estimation technique. This technique was used in the prior PM risk analyses; its application in that analysis is described elsewhere (Post et al., 2000; Abt Associates, 1996). Sensitivity

analyses will be performed to assess the impact of study selection on the pooled function. In addition, C-R functions included in the previous risk analyses or in Tables 9-3 or 9-4 of the March 2001 PM CD that have been estimated in any of the urban counties selected for the PM risk analysis will be used in a sensitivity analysis to estimate PM health risk in those areas.

The uncertainty of risk estimates based on C-R functions from a single study will be characterized by confidence intervals derived from the statistical uncertainty surrounding the pollutant coefficient estimate from the study[5]. The uncertainty of risk estimates based on pooled C-R functions derived from multiple studies will be characterized by credible intervals developed from a Monte Carlo analysis. Each iteration of the Monte Carlo procedure will be a two-step process: first, one of the studies will be selected from the set of studies used to derive the pooled function; second, a pollutant coefficient will be selected from the distribution of coefficients possible from that study (based on the point estimate and the standard error of the estimate reported in the study). It is anticipated that this procedure will result in improved characterization of the degree of uncertainty contained in the C-R functions resulting from both within-study uncertainty and between-study variability.

Concerns that are more difficult to treat quantitatively will be discussed in the text of the PM risk analysis report – for example, the potential differences in risk estimates that may result from: (1) differences in PM composition between the cities in which concentration-response functions were estimated and the cities to which those concentration-response functions are applied in the risk analysis, and (2) varying levels of associated co-pollutants in different cities. In addition, as discussed earlier a quantitative sensitivity analysis will compare the risk estimates resulting from use of single-pollutant PM concentration-response functions versus functions in which PM effects were assessed simultaneously with the effects of other pollutants (which is one possible approach to addressing the uncertainty concerning the role of co-pollutants).

F. Baseline Health Effects Incidence Considerations

The most common health risk model expresses the reduction in health risk (Δy) associated with a given reduction in PM concentrations (Δx) as a percentage of the baseline incidence (y). To accurately assess the impact of PM air quality on health risk in the selected urban areas, information on the baseline incidence of health effects (i.e., the incidence under "as is" air quality conditions) in each location is therefore needed. Where at all possible, county-specific incidences or incidence rates (in combination with county-specific populations) will be used. County-specific mortality incidences are available from the National Center for Health Statistics.

[5] It will be noted in the text of the PM risk analysis report that estimates of uncertainty based on a single study will tend to understate the true uncertainty surrounding the risk in an assessment location, because such estimates reflect only the uncertainty surrounding a single coefficient estimate, and do not reflect any inter-locational variability among coefficients.

ICD code specific baseline hospital admission rates have been obtained from the Pennsylvania Health Care Cost Containment Council for Philadelphia County, and from California's Office of Statewide Health Planning and Development for Los Angeles County. The availability of hospital admission data for the cities proposed for the $PM_{10-2.5}$ analysis (see Table 3) has been investigated. A preliminary investigation has uncovered data for Salt Lake County from the Utah Department of Health, Office of Health Care Statistics. However, as noted above, there are no available data to estimate baseline hospital admissions rates in Cleveland, Ohio. We will continue to investigate the availability of hospital admission data for the Las Vegas, Nevada metropolitan area.

For other morbidity endpoints, such as respiratory symptoms in children, incidence information aggregated at higher than the city- or county-level may be all that is available. The level of aggregation closest to county-specific will be used; however, for some morbidity endpoints, it may be necessary to estimate county-specific incidence using national-level incidence rates. For some health endpoints, there may be no information on incidence other than the information provided for the city or county in which the concentration-response function was estimated. A discussion will be presented of the rationale for the choice of incidence data used for each health endpoint in each location.

Lack of county-specific incidence data will increase the uncertainty surrounding estimates of risk for the specific cities selected for the risk analysis. To the extent possible, a quantitative comparison will be provided to help assess the accuracy of using incidence rates at a higher level of aggregation (e.g., national incidence rates) by comparing these rates to county-specific incidence rates where these are available.

III. Characterization of Uncertainty

Any estimation of attributable risk and risk reductions under current or alternative standard scenarios will involve substantial uncertainties, and there are additional uncertainties for a pollutant such as PM (as opposed to, for example, ozone), given the diversity of composition in this generally defined pollutant. Among the major sources of uncertainty in this risk analysis are:

- The statistical uncertainty surrounding estimates of $PM_{2.5}$ (and $PM_{10-2.5}$) coefficients in concentration-response functions used in the analysis.

- The transferability of PM concentration-response functions from study locations to the locations selected for the risk analysis. A C-R function in a study location may not provide an accurate representation of the C-R relationship in the assessment location(s) because of
  - variations in PM composition across cities,
  - the possible role of associated co-pollutants in influencing PM risk,

- variations in the relation 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.),
- differences in population characteristics (e.g., the proportions of members of sensitive subpopulations) and population behavior patterns across locations.

- The air quality adjustment procedure that will be used to simulate just meeting current or alternative PM standards.

- Use of baseline health effects incidence information that is not specific to the urban area in question.

- Applying pooled concentration-response functions to represent the overall effect of particles on a particular health endpoint from studies in several locations.

- The impact of historical air quality on estimates of health risk from long-term PM exposures -- the duration of time that a reduction in particle concentrations must be maintained in a given location in order to experience the predicted reduction in health risk and/or the possibility of lags between exposure and health effect.

- The effect of normalizing to different degrees the amounts of health risk experienced or reduced in different locations because of differences in the completeness of the air quality data sets.

- Estimated background concentrations for each location.

The uncertainties from some of these sources -- in particular, the statistical uncertainty surrounding estimates of the PM coefficients in C-R functions -- can be characterized quantitatively. It will be possible, for example, to calculate confidence intervals around risk estimates based on the uncertainty associated with the estimates of pollutant coefficients used in the risk analysis. These confidence intervals will express the range within which the true 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 analysis, as noted above. If there were sufficient information to quantitatively characterize these sources of uncertainty, they could be included in a Monte Carlo analysis to produce confidence intervals that more accurately reflect all sources of uncertainty.

We propose to handle uncertainties in the risk analysis in the following ways:

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

- For any endpoint for which only a single concentration-response function has been estimated, the uncertainty resulting from the statistical uncertainty associated with the estimate of the pollutant coefficient will be characterized by confidence intervals around the point estimate of risk. As noted above, such a confidence interval will express the range within which the true risk is likely to fall *if the uncertainty surrounding the pollutant coefficient estimate were the only uncertainty in the analysis*. It will not, for example, reflect the uncertainty concerning whether the pollutant coefficients in the study location and the assessment location are the same.[6]

- For any endpoint for which a pooled function has been derived from two or more studies, a credible interval will be presented along with the point estimate of risk. Credible intervals will reflect not only the within-study statistical uncertainty, but the between-study variability in pollutant coefficients as well. These credible intervals will therefore, to some extent, also reflect the uncertainty associated with applying functions estimated in locations other than the assessment location.

- Sensitivity analyses will be conducted to illustrate the effects of changing key default assumptions on the results of the assessment, and quantitative comparisons will be presented to inform other analytic choices.[7]

Possible additional or alternative approaches to characterizing uncertainty that are being considered include the following:

- To include in an overall assessment of uncertainty those sources of uncertainty that cannot readily be quantified, "integrated sensitivity analyses" will be presented. These analyses rely on judgment to assign probabilities to possible alternatives. For example, judgment could be made concerning the likelihood that each of several possible alternative assumptions is the correct one. This procedure allows sources of uncertainty that are otherwise not quantifiable to be included in a Monte Carlo sensitivity analysis.

- Different sets of plausible assumptions that would result in "low end," "middle," and "high end" estimates of incidence could be identified, and the estimates resulting under each set of assumptions could be presented as alternatives.

---

[6] This is not an uncertainty, of course, if the concentration-response function has been estimated in the assessment location.

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

IV. Presentation of Risk Results

A. As-Is Risk Analysis for $PM_{2.5}$ and $PM_{10-2.5}$

Estimates of annual health risks associated with "as is" PM air quality will be presented in both tabular and graphic form, including indications of statistical uncertainty (confidence intervals or credible intervals) for health effects from both short-term and long-term exposure for the urban areas analyzed. For concentration-response relationships involving short-term (daily) exposures, this would represent the cumulative total impact of effects from the short-term ambient PM levels across the year.

Information will be presented in several ways, including cumulative incidence curves,[8] graphs of the distribution of risk across different parts of the air quality distribution, and tables that allow the summary of many results in a compact format.

B. Current and Alternative Standards Analysis

Most of the results from analyses of current and alternative standards will be provided in tabular form. Tables will show the estimated amounts of risk reduction (cases avoided) as well as the estimated percentage risk reductions associated with just meeting the current and any alternative standards in each urban area for a variety of endpoints. Percentage risk reductions will be presented as cases avoided as a percentage of the total number of cases under the "as is" scenario or as a percentage of the number of PM-related cases under the "as is" scenario. Point estimates will be accompanied by confidence or credible intervals based on the statistical uncertainty surrounding estimates of PM coefficients in concentration-response functions. The initial scenario to be analyzed will be the current suite of annual and 24-hour $PM_{2.5}$ standards. The risk analyses for $PM_{2.5}$ and $PM_{10-2.5}$ also will examine any alternative standards that may be identified as appropriate for consideration during the course of the current review of the PM NAAQS. An additional table comparing results across urban areas, at least for some endpoints, is also planned.

C. Sensitivity Analyses

Table 7 describes the sensitivity analyses and quantitative comparisons proposed for this analysis.

[8] A cumulative incidence curve shows the cumulative annual incidence of a health effect associated with PM concentrations above background up to level n, for increasing values of n.

Table 7. Planned Sensitivity Analyses and Quantitative Comparisons

Analysis Number (Figure 1)	Component of the Risk Analysis	Sensitivity Analysis or Comparison
1	Air Quality	A sensitivity analysis of the effect of different assumptions about background PM levels
2	Air Quality	A sensitivity analysis of the effect of different air quality adjustment procedures on the estimated risk reductions resulting from just meeting alternative 24-hr and annual standards
3	Baseline Incidence	A comparison of using more aggregate incidence data (national, state, etc) versus county-specific information in the county with the best local incidence data
4	Concentration-Response	A comparison or sensitivity analysis of methods of combining averaging times of from 1 to 5 days in the short-term mortality and hospital admissions studies
5	Concentration-Response	A sensitivity analysis or comparison of the effects of including or excluding individual studies from pooled functions to show the sensitivity of the function to inclusion of specific studies
6	Concentration-Response	A comparison or 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	Concentration-Response	A sensitivity analysis comparing the risks estimated by using concentration-response functions derived for the specific county in question versus pooled functions for endpoints
8	Concentration-Response	A sensitivity analysis using concentration-response functions for PM from multi-pollutant regressions with co-pollutants versus single pollutant regressions
9	Concentration-Response	A sensitivity analysis assuming alternative minimum concentration levels for the occurrence of PM response at concentrations above those for background

References

Abt Associates Inc. July 3, 1996 (Revised November 1996). "A Particulate Matter Risk Assessment for Philadelphia and Los Angeles." Prepared for the Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Contract No. 68-W4-0029. Available electronically on the web at: www.epa.gov/ttn/oarpg/t1sp.html.

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

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

Burnett, R.T., S. Cakmak, J.R. Brook and D. Krewski. 1997. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. *Environ Health Perspect.* Vol. 105(6): 614-20.

Burnett, R.T., J. Brook, T. Dann, C. Delocla, O. Philips, S. Cakmak, R. Vincent, M.S. Goldberg and D. Krewski. 2000. Association between Particulate and Gas-Phase Components of Urban Air Pollution and Daily Mortality in Eight Canadian Cities. *Inhalation Toxicology*. Vol. 12(Supplement 4): 15-39.

Chock, D.P., S.L. Winkler and C. Chen. 2000. A study of the association between daily mortality and ambient air pollutant concentrations in Pittsburgh, Pennsylvania. *J Air Waste Manag Assoc*. Vol. 50(8): 1481-500.

Deck, L. B., E. S. Post, E. Smith, M. Wiener, K. Cunningham, and H. Richmond. Estimates of the Health Risk Reductions Associated with Attainment of Alternative Particulate Matter Standards in Two U.S. Cities. Accepted by *Risk Analysis*, March 2001.

Delfino, R.J., A.M. MurphyMoulton, R.T. Burnett, J.R. Brook and M.R. Becklake. 1997. Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec. *Am J Respir Crit Care Med.* Vol. 155(2): 568-576.

Dockery, D.W., F.E. Speizer, D.O. Stram, J.H. Ware, J.D. Spengler and B.G. Ferris, Jr. 1989. Effects of Inhalable Particles on Respiratory Health of Children. *Am Rev Respir Dis.* Vol. 139: 587-594.

Dockery, D.W., J. Schwartz and J.D. Spengler. 1992. Air Pollution and Daily Mortality - Associations With Particulates and Acid Aerosols. *Environmental Research*. Vol. 59(2): 362-373.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer. 1996. Health Effects of Acid Aerosols On North American Children - Respiratory Symptoms. *Environ Health Perspect*. Vol. 104(5): 500-505.

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

Goldberg, M.S., J.C. Bailar III, R.T. Burnett, J.R. Brook, R. Tamblyn, Y. Bonvalot, P. Ernst, K.M. Flegel, R.K. Singh, M. and Valois. Identifying Subgroups of the General Population That May Be Susceptible to Short-Term Increases in Particulate Air Pollution: A Time-Series Study in Montreal, Quebec. Health Effects Institute Research Report, Number 97, October 2000.

Klemm, R.J. and R.M. Mason, Jr. 2000. Aerosol Research and Inhalation Epidemiological Study (ARIES): air quality and daily mortality statistical modeling--interim results. *J Air Waste Manag Assoc*. Vol. 50(8): 1433-9.

Krewski, D., R. Burnett, M. Goldberg, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz and M. White. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Health Effects Institute. Cambridge. July.

Laden, F., L.M. Neas, D.W. Dockery and J. Schwartz. 2000. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environ Health Perspect*. Vol. 108(10): 941-7.

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

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

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

McConnell, R., K. Berhane, F. Gilliland, S.J. London, H. Vora, E. Avol, W.J. Gauderman, H.G. Margolis, F. Lurmann, D.C. Thomas and J.M. Peters. 1999. Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ Health Perspect*. Vol. 107(9): 757-60.

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

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

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

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

Peters, J.M., E. Avol, W. Navidi, S.J. London, W.J. Gauderman, F. Lurmann, W.S. Linn, H. Margolis, E. Rappaport, H. Gong and D.C. Thomas. 1999. A Study of Twelve Southern California Communities with Differing Levels and Types of Air Pollution. I. prevalence of respiratory morbidity. *Am J Respir Crit Care Med*. Vol. 159(3): 760-767.

Post, E., L. Deck, K. Larntz, D. Hoaglin. An Application of an Empirical Bayes Estimation Technique to the Estimation of Mortality Related to Short-Term Exposure to Particulate Matter. Accepted by *Risk Analysis*, December, 2000.

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

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

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

Schwartz, J. 2000. Harvesting and long term exposure effects in the relation between air pollution and mortality [see comments]. *Am J Epidemiol*. Vol. 151(5): 440-8.

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

Smith, R.L., D. Spitzner, Y. Kim and M. Fuentes. 2000. Threshold dependence of mortality effects for fine and coarse particles in Phoenix, Arizona. *J Air Waste Manag Assoc*. Vol. 50(8): 1367-79.

Stieb, D.M., R.C. Beveridge, J.R. Brook, M. Smith-Doiron, R.T. Burnett, R.E. Dales, S. Beaulieu, S. Judek and A. Mamedov. 2000. Air pollution, aeroallergens and cardiorespiratory emergency department visits in Saint John, Canada. *J Expo Anal Environ Epidemiol*. Vol. 10(5): 461-77.

Thurston, G.D., K. Ito, C.G. Hayes, D.V. Bates and M. Lippmann. 1994. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. *Environ Res*. Vol. 65(2): 271-290.

Tolbert, P.E., M. Klein, K.B. Metzger, J. Peel, W.D. Flanders, K. Todd, J.A. Mulholland, P.B. Ryan and H. Frumkin. 2000. Interim results of the study of particulates and health in Atlanta (SOPHIA). *J Expo Anal Environ Epidemiol*. Vol. 10(5): 446-60.

Tsai, F.C., M.G. Apte and J.M. Daisey. 2000. An Exploratory Analysis of the Relationship between Mortality and the Chemical Composition of Airborne Particulate Matter. *Inhalation Toxicology*. Vol. 12(Supplement 2): 121-135.

U.S. Environmental Protection Agency (U.S. EPA) 1996a. *Air Quality Criteria for Particulate Matter*, (EPA/600/P-95/001aF-cF), 3v, National Center for Environmental Assessment-RTP Office, Research Triangle Park, NC.

U.S. Environmental Protection Agency (U.S. EPA) July 1996. *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information - OAQPS Staff Paper,* (EPA/452/R-96-013), Office of Air Quality Planning and Standards, Research Triangle Park, NC 27711. Available from: NTIS, Springfield, VA; PB97-115406REB or electronically on the internet at: http://www.epa.gov/ttn/oarpg/t1sp.html.

U.S. Environmental Protection Agency (U.S. EPA) March 2001. *Air Quality Criteria for Particulate Matter,* (EPA 600/P-99/002aB), 2v, National Center for Environmental Assessment, Office of Research and Development, Research Triangle Park, NC Available electronically on the internet at: http://www.epa.gov/ncea/partmatt.htm

Wolff, G.T.. 1996. Letter from George T. Wolff, Chair, Clean Air Scientific Advisory Committee. Closure by the Clean Air Scientific Advisory Committee (CASAC) on the Staff Paper for Particulate Matter. June 13, 1996.