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Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment

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Office of Air Quality Planning and Standards
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Research Triangle Park, NC 27711

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Table of Contents

List of Tables

List of Figures

LIST OF ACRONYMS/ABBREVIATIONS

A/C	Air conditioning
Act	Clean Air Act
AERMOD	American Meteorological Society/EPA Regulatory Model Improvement Committee (AERMIC) air quality dispersion modeling system
AHRQ	Agency for Healthcare Research and Quality
AHS	American Housing Survey
APEX	EPA's Air Pollutants Exposure model, version 4
AQS	EPA's Air Quality System
ARIES	Aerosol Research and Inhalation Epidemiological Study
BASE	Building Assessment Survey and Evaluation
BenMAP	Benefits Mapping Analysis Program
CARB	California Air Resources Board
CAMx	Comprehensive Air quality Model
CASAC	Clean Air Scientific Advisory Committee
CEM	Continuous Emission Monitoring
CHAD	EPA's Consolidated Human Activity Database
CMAQ	Community Multiscale Air Quality
CONUS	Continental United States
COV	Coefficient of Variation
C-R	Concentration-response relationship
CSA	Consolidated Statistical Area
CTM	Chemical transport models
EPA	United States Environmental Protection Agency
eVNA	enhanced Voronoi Neighbor Averaging
FEM	Federal Equivalent Method
FIP	Federal Implementation Plan
FRM	Federal Reference Method
GAMs	Generalized additive models
GLMs	Generalized linear models
HCUP	Healthcare Cost and Utilization Project
HEI	Health Effects Institute
IEC	Industrial Economics, Incorporated
IMPROVE	Interagency Monitoring of Protected Visual Environment
ISA	Integrated Science Assessment
ISCST	EPA's Industrial Source Complex Short-Term model
km	Kilometer
ME	Microenvironment
MENTOR	Modeling ENvironment for TOtal Risk
NAAQS	National Ambient Air Quality Standards
NAPS	National Air Pollution Surveillance
NCEA	National Center for Environmental Assessment

NEI	National Emissions Inventory
NEM	NAAQS Exposure Model
NERL	National Exposure Research Laboratory
NCDC	National Climatic Data Center
NMMAPS	National Morbidity, Mortality, and Air Pollution Study
NCore	National Core Monitoring Network
NOx	Nitrogen oxides
O_3	Ozone
OAQPS	Office of Air Quality Planning and Standards
OAR	Office of Air and Radiation
OMB	Office of Management and Budget
ORD	Office of Research and Development
PM	Particulate Matter
PM_X	The legal definition for PM_X, as defined in the Code of Federal Regulations, includes both a 50% cut-point and a penetration curve. A 50% cut-point of X μm diameter means that 50% of particles with aerodynamic diameter of X are removed by the inlet and 50% pass through the inlet and are collected on the filter. Depending on the specific penetration curve specified, particles larger than X μm aerodynamic diameter are collected with an efficiently than decreases rapidly for particles larger than X while the collection efficiency for particles smaller than X increases rapidly with decreasing size until 100 % efficiency is reached.
PM_{10}	Particles with a 50% upper cut-point of 10± 0.5 μm aerodynamic diameter and a penetration curve as specified in the Code of Federal Regulations.
$PM_{2.5}$	Particles with a 50% upper cut-point of 2.5 μm aerodynamic diameter and a penetration curve as specified in the Code of Federal Regulations.
$PM_{10-2.5}$	Particles with a 50% upper cut-point of 10 μm aerodynamic diameter and a lower 50% cut-point of 2.5 μm aerodynamic diameter.
PRB	Policy-Relevant Background
QA	Quality assurance
RR	Relative risk
SAB	Science Advisory Board
REA	Risk and Exposure Assessment
SD	Standard deviation
SEDD	State Emergency Department Databases
SHEDS-PM	Stochastic Human Exposure and Dose Simulation model for PM
SID	State Inpatient Database
SO_2	Sulfur Dioxide
SO_x	Sulfur Oxides
TEACH	Toxicity and Exposure Assessment for Children's Health
TSP	Total suspended particulate
VOC	Volatile organic compounds

1 INTRODUCTION

The U.S. Environmental Protection Agency (EPA) is presently conducting a review of the particulate matter (PM) national ambient air quality standards (NAAQS). EPA's overall plan and schedule for this PM NAAQS review are presented in the *Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter* (US EPA, 2008a). That plan outlines the Clean Air Act (CAA) requirements related to the establishment and reviews of the NAAQS, the process and schedule for conducting the current PM NAAQS review, and two key components in the NAAQS review process: an Integrated Science Assessment (ISA) and a Risk and Exposure Assessment (REA). It also lays out the key policy-relevant issues to be addressed in this review as a series of policy-relevant questions that will frame our approach to determining whether the current primary and secondary NAAQS for PM should be retained or revised.

The ISA prepared by EPA's Office of Research and Development (ORD), National Center for Environmental Assessment (NCEA) provides a critical assessment of the latest available policy-relevant scientific information upon which the NAAQS are to be based. The ISA will critically evaluate and integrate scientific information on the health and welfare effects associated with exposure to PM in the ambient air. The REA, prepared by EPA's Office of Air and Radiation (OAR), Office of Air Quality Planning and Standards (OAQPS), will draw from the information assessed in the ISA. The REA will include, as appropriate, quantitative estimates of human and ecological exposures and/or risks associated with recent ambient levels of PM, with levels simulated to just meet the current standards, and with levels simulated to just meet possible alternative standards.

The REA will be developed in two parts addressing: (1) human health risk and exposure assessment and (2) visibility impairment and other welfare-related effects assessment. This document describes the scope and methods planned to conduct the human health risk and exposure assessments to support the review of the primary (health-based) PM NAAQS. A separate document describes the scope and methods planned to conduct quantitative assessments to support the review of the secondary (welfare-based) PM NAAQS (U.S. EPA, 2009). Preparation of these two planning documents coincides with the development of the first draft

PM ISA (U.S. EPA, 2008b) to facilitate the integration of policy-relevant science into all three documents.

This planning document is intended to provide enough specificity to facilitate consultation with CASAC, as well as for public review, in order to obtain advice on the overall scope, approaches, and key issues in advance of the conduct of the risk and exposure analyses and presentation of results in the first draft REA. NCEA has compiled and assessed the latest available policy-relevant science available to produce a first draft of the ISA and related Annexes (US EPA, 2008b). The first draft ISA has been reviewed by staff and used in the development of the approaches described below. This includes information on atmospheric chemistry, source emissions, air quality, human exposure, and related health effects. CASAC consultation on this planning document coincides with its review of the first draft ISA. CASAC and public comments on this document will be taken into consideration in the development of the first draft REA, the preparation of which will coincide and draw from the second draft ISA. The second draft REA will draw on the final ISA and will reflect consideration of CASAC and public comments on the first draft REA. The final REA will reflect consideration of CASAC and public comments on the second draft REA. The final ISA and final REA will inform the policy assessment and rulemaking steps that will lead to a final decision on the PM NAAQS.

This introductory chapter includes background on the current PM standards and the quantitative risk assessment conducted for the last review; the key issues related to designing the quantitative assessments in this review, building upon the lessons learned in the last review; and an overview introducing the planned assessments that are described in more detail in later chapters. The planned assessments are designed to estimate health risks and/or human exposures that are associated with recent ambient levels, with ambient levels simulated to just meet the current standards, and with ambient levels simulated to just meet alternative standards that may be considered. The major components of the assessments (e.g., air quality analyses, quantitative health risk assessment, risk characterization, and quantitative exposure assessment) briefly outlined in the Integrated Review Plan (U.S. EPA, 2008a, section 5), are conceptually presented in Figures 1-1 and 1-2, and are described in more detail below in Chapters 2, 3, and 4, respectively. The schedule for completing these assessments is presented in Chapter 5.

1.1 BACKGROUND ON LAST PM NAAQS REVIEW

As a first step in developing this planning document, we considered the work completed in previous reviews of the primary NAAQS for PM (U.S., EPA 2008a, see section 1.3) and in particular the quantitative assessments supporting those reviews. The most recent review of the PM standards, completed in 2006 (71 FR 61144, October 17, 2006)[1], evaluated the existing NAAQS and concluded that the standards needed to be revised to provide increased public health protection. The rationale for the final decision on the appropriate revisions to the primary PM NAAQS included consideration of: (1) evidence of health effects related to short- and long-term exposures to particles; (2) insights gained from the quantitative risk assessment; and (3) specific conclusions regarding the need for revisions to the current standards and the elements of the PM standards (i.e., indicator, averaging time, form, and level), that taken together, are requisite to protect public health with an adequate margin of safety.

In summary, with regard to the primary standards for fine particles, EPA revised the level of the 24-hour $PM_{2.5}$ standard to 35 $\mu g/m^3$, retained the level of the annual $PM_{2.5}$ annual standard at 15 $\mu g/m^3$, and revised the form of the annual $PM_{2.5}$ standard by narrowing the constraints on the optional use of spatial averaging. With regard to the primary standards for thoracic coarse particles, EPA retained PM_{10} as the indicator for purposes of regulating the coarse fraction of PM_{10} (referred to as thoracic coarse particles or coarse-fraction particles; generally including particles with a nominal mean aerodynamic diameter greater than 2.5 μm and less than or equal to 10 μm, or $PM_{10-2.5}$). Specifically, EPA retained the 24-hour PM_{10} standard at 150 $\mu g/m^3$ and revoked the annual PM_{10} standard because available evidence generally did not suggest a link between long-term exposure to current ambient levels of thoracic coarse particles and health or welfare effects.

In the last PM NAAQS review, EPA focused on particle mass and primarily distinguished between two categories of particle pollution based on size (i.e., fine- and thoracic coarse-fraction particles), and conducted parallel evaluations of the available scientific evidence relating to each category. The importance of specific PM components and sources were

[1] See also http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_index.html.

evaluated within the context of this basic size differentiation. In that review, it was determined that size-fractionated particle mass, rather than particle composition, remained the most appropriate approach for addressing ambient PM. The EPA conducted a quantitative health risk assessment[2] for selected health endpoints to provide additional information and insights that could help inform decisions on the standards. The limitations of such an assessment were clearly articulated.[3] These assessments are briefly described below. EPA did not conduct an exposure assessment for the PM NAAQS review completed in 2006.

1.1.1 Overview of Health Risk Assessment for Fine Particles from Last Review

The approach used in the last PM NAAQS review to develop quantitative risk estimates associated with exposures to fine particles, using $PM_{2.5}$ as the indicator, was built upon the more limited risk assessment conducted during the review completed in 1997 (Abt Associates, 1996, 2002). The expanded and updated assessment conducted for the review completed in 2006 included estimates of risks of mortality (total non-accidental, cardiovascular, and respiratory), morbidity (hospital admissions for cardiovascular and respiratory causes), and respiratory symptoms (not requiring hospitalization) associated with recent short-term (daily) ambient $PM_{2.5}$ levels and risks of total, cardiopulmonary, and lung cancer mortality associated with long-term exposure to $PM_{2.5}$ in nine urban areas. Nine areas were included in this assessment to provide some sense of the variability in the $PM_{2.5}$-related risk estimates across the U.S. including: Boston, MA; Detroit, MI; Los Angeles, CA; Philadelphia, PA; Phoenix, AZ; Pittsburgh, PA; San Jose, CA; and St. Louis, MO.

EPA recognized that there were many sources of uncertainty and variability inherent in the inputs to this assessment and that there was a high degree of uncertainty in the resulting

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[2] The risk assessment was discussed in the Staff Paper (U.S. EPA, 2005, Section 4) and presented more fully in a technical support document, *Particulate Matter Health Risk Assessment for Selected Urban Areas* (Abt Associates, 2005). The assessment scope and methodology were developed with considerable input from the CASAC Panel and the public, with CASAC concluding that the general assessment methodology and framework were appropriate (Hopke, 2002).

[3] The EPA continues to support the development and application of risk assessment methods with the goal of improving the characterization of risks and the communication of uncertainties in such risk estimates.

$PM_{2.5}$ risk estimates. Such uncertainties generally related to a lack of clear understanding of a number of important factors, including, for example:

- the shape of concentration-response functions, particularly when effect thresholds could neither be discerned nor determined not to exist;
- issues related to selection of appropriate statistical models for the analysis of the epidemiologic data;
- the role of potentially confounding and modifying factors in the concentration-response relationships;
- the method for simulating how daily $PM_{2.5}$ ambient concentrations would likely change in any given area upon meeting a particular standard, since strategies to reduce emissions had not yet been defined; and
- the issue of whether there would be differential reductions in the many components within $PM_{2.5}$ and, if so, whether this would result in differential reductions in risk.

While some of these uncertainties were addressed quantitatively in the form of estimated confidence ranges around central risk estimates, other uncertainties and the variability in key inputs were not reflected in these confidence ranges, but rather were addressed through separate sensitivity analyses or characterized qualitatively (U.S. EPA, 2005, Chapter 4; Abt Associates, 2005). The concentration-response relationships used in the quantitative risk assessment were based on findings from human epidemiologic studies that relied on fixed-site, population-oriented, ambient monitors as a surrogate for actual ambient $PM_{2.5}$ exposures. The assessment included a series of base case estimates that, for example, included various cutpoints intended as surrogates for alternative assumed population thresholds. Other uncertainties were addressed in various sensitivity analyses (e.g., the use of single- versus multi-pollutant models, use of single- versus multi-city models, use of a distributed lag model) and had a more moderate and often variable impact on the risk estimates in some or all of the cities.

Key observations and insights from the $PM_{2.5}$ risk assessment, together with important caveats and limitations, were discussed in Section II.B of the 2006 proposal notice (71 FR 2637 to 2641, January 17, 2006). In general, estimated risk reductions associated with going from just meeting the current suite of $PM_{2.5}$ standards to just meeting alternative suites of annual and 24-hour standards for all the various assumed cutpoints showed patterns of increasing estimated risk reductions as either the annual or 24-hour $PM_{2.5}$ standard, or both, were reduced over the range considered in the assessment, and the estimated percentage reductions in risk were strongly influenced by the assumed cutpoint level (EPA, 2005, see Figures 5-1, 5-2, 5A-1, and 5A-2). In comparing the risk estimates for the only two locations (Philadelphia, PA and Los Angeles, CA) that were included in both this assessment and the prior assessment, the magnitude of the risk estimates associated with just meeting the current annual $PM_{2.5}$ standard, in terms of percentage of total incidence, were very similar for premature mortality associated with long-term exposures.

In making final decisions for the $PM_{2.5}$ NAAQS in 2006, the Administrator relied primarily on evidence-based considerations to inform his conclusions on the levels for the 24-hour and annual standards. The Administrator believed, at that time, that the estimates of risks likely to remain upon attainment of the 1997 suite of $PM_{2.5}$ standards were indicative of risks that could be reasonably judged important from a public health perspective, and, thus, supported revision of the standards. However, the Administrator judged that the quantitative risk assessment had important limitations and did not provide an appropriate basis for selecting either the level of the 24-hour or annual $PM_{2.5}$ standard. The Administrator more heavily weighed the implications of the uncertainties associated with the quantitative risk assessment than CASAC in their comments on the proposed rulemaking and disagreed with CASAC and many public commenters that the risk assessment results could appropriately serve as a primary basis for a decision for the level of either the 24-hour or the annual $PM_{2.5}$ standards.[4]

[4] See discussion in Section II.F of the preamble to the final rule, 71 FR 61167-61177, October 17, 2006.

1.1.2 Overview of Health Risk Assessment for Thoracic Coarse Particles from Last Review

The general overview and discussion of key components of the quantitative risk assessment used to develop risk estimates for $PM_{2.5}$ presented above is also applicable to the risk assessment conducted for $PM_{10-2.5}$ as part of the last review. However, the scope of the risk assessment for $PM_{10-2.5}$ was much more limited than that for $PM_{2.5}$, reflecting the much more limited body of epidemiologic evidence and air quality information available for $PM_{10-2.5}$. As discussed in section 4.5 of the Staff Paper (U.S. EPA, 2005), the $PM_{10-2.5}$ risk assessment included risk estimates for just three urban areas for two categories of health endpoints related to short-term exposure to $PM_{10-2.5}$: hospital admissions for cardiovascular and respiratory causes and respiratory symptoms.

Estimates of hospital admissions attributable to short-term exposure to $PM_{10-2.5}$ were developed for Detroit, MI (cardiovascular and respiratory admissions) and Seattle, WA (respiratory admissions), and estimates of respiratory symptoms were developed for St. Louis, MO. While one of the goals of the $PM_{10-2.5}$ risk assessment was to provide estimates of the risk reductions associated with just meeting alternative $PM_{10-2.5}$ standards, EPA concluded that the nature and magnitude of the uncertainties and concerns associated with this portion of the risk assessment weighed against use of these risk estimates as a basis for recommending specific standard levels (U.S. EPA, 2005, see p. 5-69). These uncertainties and concerns were summarized in the proposal notice (see FR 71 2662, January 17, 2006) and discussed more fully in the Staff Paper (U.S. EPA, 2005, Chapter 4) and associated technical support document (Abt Associates, 2005).

1.2 GOALS FOR FRAMING THE ASSESSMENTS IN THE CURRENT REVIEW

A critical step in designing the quantitative risk and exposure assessments is to clearly identify the policy-relevant questions to be addressed by these assessments. As identified above, the Integrated Review Plan presents a series of key policy questions (U.S. EPA, 2008a, section 3.1). To answer these questions, EPA will integrate information from the ISA and from air quality, risk, and exposure assessments as we evaluate both evidence-based and risk-based considerations.

More specifically, to focus the REA, we have identified the following goals for the risk assessment: (1) to provide estimates of the potential magnitude of premature mortality and/or selected morbidity health effects in the population, including sensitive subpopulations, where data are available to assess these subgroups, associated with recent ambient levels of fine and thoracic coarse particles and with just meeting the current suite of PM standards and any alternative standards that might be considered in selected urban study areas; (2) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates to more clearly differentiate alternative standards that might be considered including potential impacts on various sensitive subpopulations; and (3) to gain insights into the distribution of risks and patterns of risk reduction and uncertainties in those risk estimates. In addition, we are considering conducting an assessment to provide nationwide estimates of the potential magnitude of premature mortality associated with long-term ambient fine particle exposures to more broadly characterize this risk on a national scale and to support the interpretation of the more detailed risk results generated for the selected urban study areas.

We have also identified the primary goal for the exposure assessment as providing insight on population exposures to inform the interpretation of available epidemiologic studies. More specifically, the design of the exposure assessment is planned to inform our understanding of how exposure-related factors contribute to the heterogeneity in responses to ambient PM concentrations observed in epidemiologic studies and/or to provide insights on other issues related to uncertainties in the existing epidemiology evidence.

1.3 OVERVIEW OF CURRENT ASSESSMENT PLAN

This plan outlines the scope and approaches and highlights key issues in our plans to assess human health risks and/or population exposures posed by ambient fine and thoracic coarse particles. The characterizations of the variability and uncertainties associated with the qualitative and quantitative analyses is an important component of our assessments.

1.3.1 Air Quality Assessment

Chapter 2 describes assessments planned for the current review of the primary NAAQS for PM including air quality analyses to be conducted to support quantitative risk and exposure

assessments in selected urban study areas as well as to support evidence-based considerations and to place the results of the quantitative assessments into a broader public health perspective. Air quality inputs will include: (1) recent air quality data for $PM_{2.5}$ and $PM_{10-2.5}$ from suitable monitors for each selected urban study area; (2) estimates of policy-relevant background (PRB)[5] concentrations for each selected urban study area, and (3) simulated air quality that reflects changes in the distribution of PM air quality estimated to occur when an area just meets the current or alternative PM standards under consideration. While incremental risk reductions do not require estimates of PRB, estimates of the risks in excess of PRB remaining upon meeting the current or potential alternative standards, do require us to estimate PRB. Both kinds of risk estimates are considered relevant to inform the Administrator's decision on the adequacy of a given standard. The approach to estimating PRB for $PM_{2.5}$ and $PM_{10-2.5}$ for use in conducting the health risk assessment will be informed by the discussion and evaluation contained in the first draft ISA and will build on the approach used in the last review (Langstaff, 2004, 2005). For the exposure assessment, EPA plans to focus on historical air quality data for $PM_{2.5}$ only considering time periods evaluated in selected epidemiologic studies. The exposure assessment will not evaluate air quality simulated to just meet current or alternative $PM_{2.5}$ standards.

1.3.2 Risk Assessment

Chapter 3 discusses the planned health risk assessment, outlined in Figure 1-1. This assessment will build upon the methodology, analyses, and lessons learned from the assessments conducted for the last review as briefly summarized in section 1.1 above. In the Integrated Review Plan, we recognized a potentially broad scope for the quantitative risk assessment and proposed to focus our efforts on fine particles ($PM_{2.5}$), and to consider, to the extent relevant information is available, risks associated with thoracic coarse particles ($PM_{10-2.5}$), as well as risks associated with specific PM components, sources, and/or environments (U.S. EPA, 2008a, section 5.5).

[5] For the purposes of the risk and exposure assessments, background PM is defined as the distribution of PM concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made) emissions of primary PM and precursor emissions (e.g., VOC, NO_x, SO_x, and NH_3) in the U.S., Canada, and Mexico. We refer to background levels so defined as policy-relevant background (PRB). See section 2.4 for additional information.

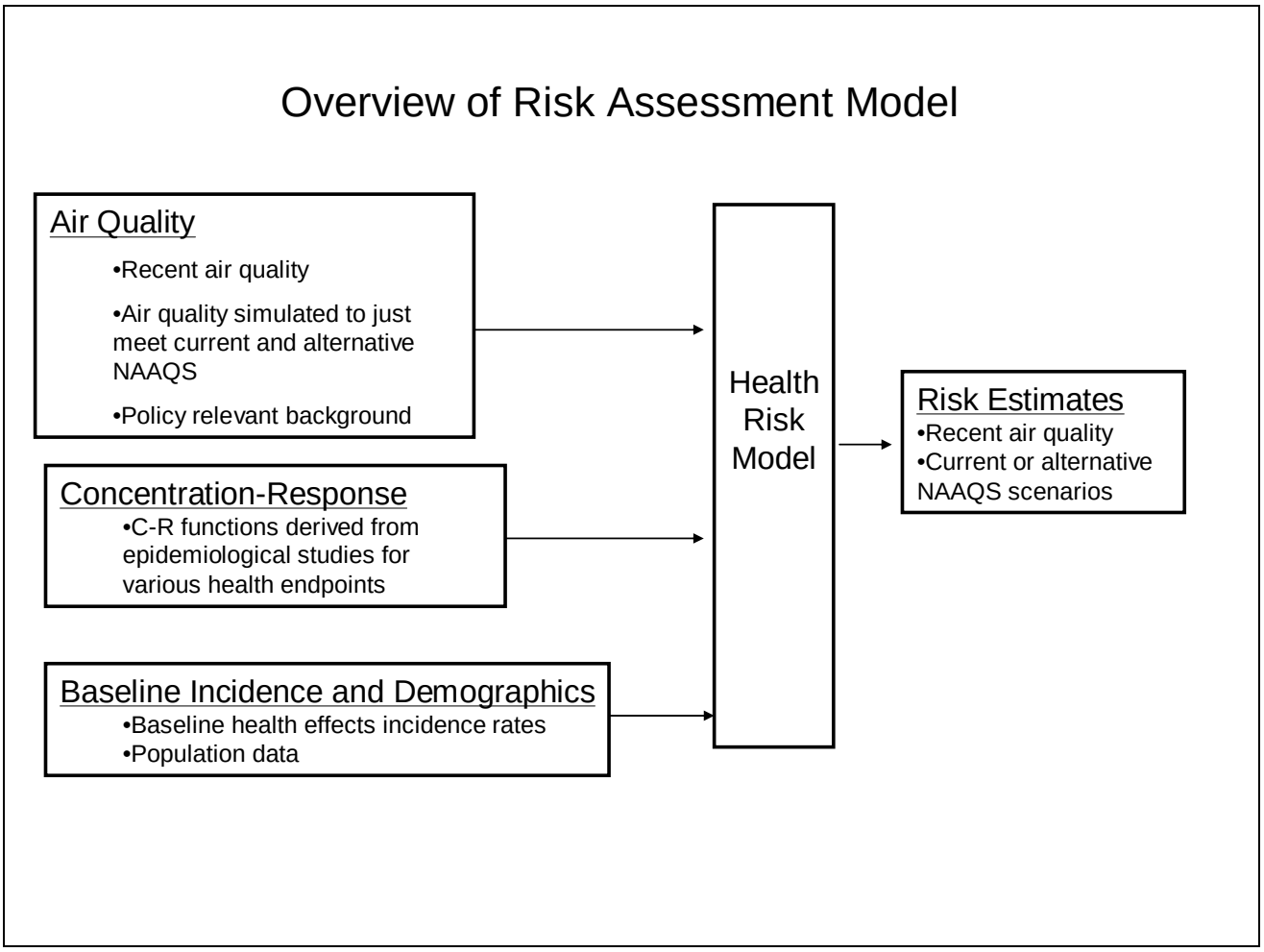


Figure 1-1 Overview of Risk Assessment Model

We plan to focus the risk assessment on selected health effect endpoints (e.g., emergency department visits and hospitalizations for ischemic heart disease) for which the weight of the evidence supports the judgment that the overall health effect category (e.g., cardiovascular morbidity) is at least likely caused by exposure to fine particles ($PM_{2.5}$) either alone and/or in combination with other pollutants. The planned quantitative risk assessment is designed to estimate risks associated with short- (24-hour average) and long-term (annual average) ambient $PM_{2.5}$ concentrations in selected urban study areas. We are considering expanding the focus of the risk assessment for fine particles to include additional health effect categories (e.g., birth outcomes) that are within broader health effect categories (e.g., reproductive, developmental,

prenatal and neonatal outcomes) that have been initially classified in the first draft ISA as having suggestive evidence of a causal association with ambient $PM_{2.5}$ measurements.

With respect to evaluating the public health impacts of thoracic coarse particles, we plan to build on the limited risk assessment conducted in the last review for $PM_{10-2.5}$ as outlined in section 1.2 above. Based upon the information in the first draft ISA, we plan to focus the risk assessment for thoracic coarse particles on health effect categories that we judge to be sufficiently suggestive of a causal association with short-term (24-hour) $PM_{10-2.5}$ ambient measurements.

We have considered the extent to which evidence supports a quantitative risk assessment for specific PM components, sources, and/or environments. Based upon information assessed in the first draft PM ISA, we have provisionally concluded that the available data do not support a quantitative risk assessment for any specific components of $PM_{2.5}$ or $PM10_{-2.5}$, nor for specific sources, environments, or other size fractions (e.g., ultrafine particles).

1.3.3 Exposure Assessment

As part of the last review, EPA did not conduct an exposure assessment. Chapter 4 discusses our plan to conduct a quantitative exposure assessment in this review primarily to provide insights on population exposures with respect to informing the interpretation of the available epidemiologic evidence. The design of the exposure assessment is summarized in Figure 1-2. The Integrated Review Plan (U.S., EPA, 2008a, section 5.4) outlined a second purpose for conducting a population exposure assessment that is, assessing population exposures above benchmark levels of concern, and providing input to quantitative risk assessments based on evidence from clinical studies. At this time, based upon information presented in the first draft ISA, we are unaware of any results from human clinical studies that would provide the basis for exposure-response functions that could inform the selection of benchmark levels of concern or a quantitative risk assessment; therefore, we do not plan to address the second purpose in this review.

The exposure assessment will build upon the methodology, analyses, and lessons learned from assessments conducted for other recent NAAQS reviews (U.S. EPA, 2007, U.S. EPA, 2008c). We plan to focus the exposure assessment on $PM_{2.5}$. We believe, at

this time, that the available monitoring data for $PM_{10-2.5}$ do not provide enough spatial coverage for exposure modeling to be useful.

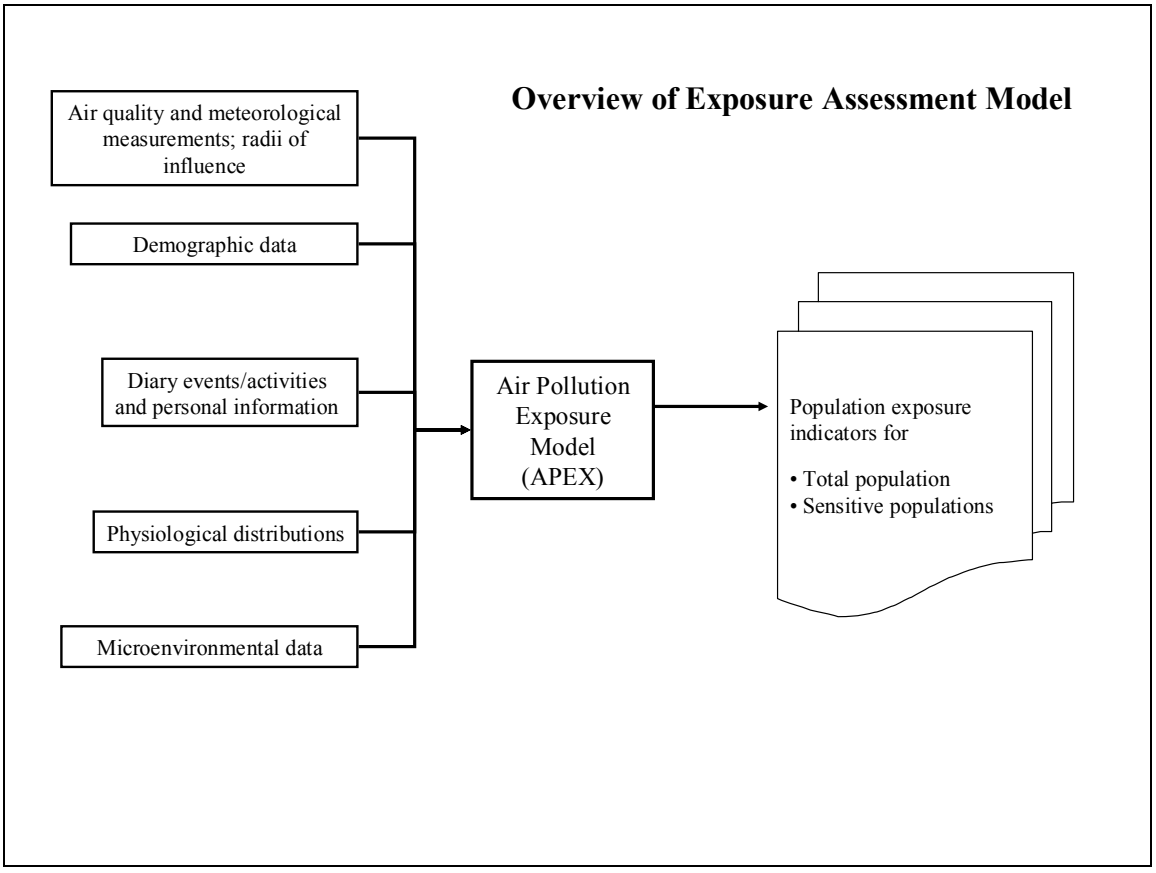


Figure 1-2 Overview of Exposure Assessment Model

EPA plans to model population exposures to ambient $PM_{2.5}$ in a small number of generally representative urban areas across the U.S. Criteria to select the specific urban study areas will include identifying locations where epidemiologic studies have been conducted that are planned to be used to support the quantitative risk assessment. We plan to select urban study areas that will be representative of a variety of populations, geographic areas, climates, and different $PM_{2.5}$ composition and co-pollutant levels. The exposure modeling locations and periods chosen are planned to be selected from the epidemiologic studies evaluated. We do not plan to develop estimates for population exposures associated with simulation of air quality to just meet the current or any alternative standards under consideration.

2 AIR QUALITY CONSIDERATIONS

2.1 INTRODUCTION

A number of air quality analyses are planned to provide inputs for the risk and exposure assessments that will be conducted for selected urban study areas as well as to provide a broader understanding of PM air quality, in order to inform: (1) evidence-based considerations; (2) our understanding of the risk and exposure assessment results to better characterize potential nationwide public health impacts associated with exposures to fine and thoracic coarse particles; and (3) policy considerations related to evaluating possible alternative NAAQS. EPA plans to focus air quality assessments on both fine particles ($PM_{2.5}$) and thoracic coarse particles ($PM_{10-2.5}$), although the availability of ambient air monitoring data is much more robust for $PM_{2.5}$ than for $PM_{10-2.5}$. Specific goals for the planned air quality assessments include:

- Characterizing air quality in various locations across the U.S. in terms of $PM_{2.5}$ and $PM_{10-2.5}$ considering differences in PM ambient concentrations, composition, and spatial and temporal patterns to help inform the selection of specific cities that we plan to include in the risk and exposure assessments. Analyses for this purpose have been ongoing.

- Characterizing policy-relevant background (PRB) based on chemical transport modeling conducted for and described in the first draft ISA (U.S. EPA, 2008b, section 3.6).

- Providing air quality distributions for $PM_{2.5}$ and $PM_{10-2.5}$ for a number of alternative scenarios in the selected urban study areas including:

    - Recent air quality;

    - Simulation of air quality to just meet the current suite of primary PM standards; and

    - Simulation of air quality to just meet potential alternative primary standards for fine and thoracic coarse particles under consideration.

- Providing a broader characterization of current $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10}, concentrations nationally (beyond the locations evaluated in the risk and exposure assessments.

2.2 AIR QUALITY INPUTS TO RISK ASSESSMENTS

Major inputs to the PM risk assessment are ambient PM air quality data. For the assessments described in this chapter, EPA plans to use 2005-2007 air quality data obtained from EPA's Air Quality System (AQS). These ambient measurement data have been collected by State and local air monitoring agencies that are guided by EPA rules, guidance documents, and grant terms. The period of 2005-2007 has been selected because these data are the most recent data that have been: (1) certified by the State/local monitoring organizations as being complete and accurate to the best of their knowledge and (2) EPA has reached final decisions on which of these data may be excluded under the Exceptional Events Rule (71 FR 13560, March 22, 2007) for 24-hour $PM_{2.5}$ NAAQS designations.

2.2.1 Recent Air Quality: $PM_{2.5}$

For $PM_{2.5}$, in general, only data collected by Federal reference or equivalent methods (FRMs or FEMs) will be used in the risk and exposure assessments, consistent with the use of such data in most of the health effects studies. However, if an epidemiologic study used non-FRM/FEM data from a continuous $PM_{2.5}$ monitor(s) as an independent variable for a concentration-response function, consideration will be given to using the same type of data in the quantitative risk assessment for the same location. In order to be consistent with the approach generally used in the epidemiological studies that estimated $PM_{2.5}$ concentration-response (C-R) functions for short-term effects, we plan to average ambient $PM_{2.5}$ concentration on each day for which measured data are available for estimating health effects associated with 24-hour ambient concentrations. Consistent with the approach used in the prior two $PM_{2.5}$ risk assessments, a composite monitor data set will be created for each assessment location based on a composite of all monitors with at least 11 observations per quarter. As in the last review, some monitoring sites may be omitted, if needed, to best match the set of monitors that were used in the epidemiological studies. Most assessment locations will not have a composite estimate for all days because of the variability in local monitoring schedules, in which case adjustments will be made to standardize the risk estimates to 365 days per year.

2.2.2 Recent Air Quality: $PM_{10-2.5}$

For $PM_{10-2.5}$, there are air quality analysis challenges not present in the case of $PM_{2.5}$. $PM_{10-2.5}$ air quality can be estimated from $PM_{2.5}$ and PM_{10} concentrations at co-located monitors by subtracting the former from the latter. When inconsistent samplers and filter weighing procedures are used, some of the $PM_{10-2.5}$ concentrations that are calculated may be negative, and all the calculated values have more uncertainty than when matched, low-volume samplers (which are standard for $PM_{2.5}$ monitoring) are used. Relatively few sites had collocated matched low-volume samplers operating in 2005-2007, as this became the reference method for $PM_{10-2.5}$ only in a rulemaking completed late in 2006 (71 FR, 61144, October 17, 2006). Moreover, the historical monitoring network design strategies have been different for $PM_{2.5}$ and PM_{10}, resulting in many of each type of monitor not having a collocated monitor of the other type. We plan to use the same approach to estimating $PM_{10-2.5}$ in an assessment location as is used in the epidemiological studies that provide C-R functions. We plan to focus the air quality assessment for thoracic coarse particles on 24-hour ambient PM_{10} and $PM_{2.5}$ concentrations since that is the focus of any planned $PM_{10-2.5}$ risk assessment.

2.2.3 Air Quality Data Related to Exceptional Events

State and local agencies and EPA have systematically reviewed $PM_{2.5}$ data for purposes of making requests and decisions regarding the exclusion of data under the Exceptional Events Rule. We would include these decisions regarding specific data that should be excluded from consideration when testing to see if a monitoring site has air quality meeting the current or alternative $PM_{2.5}$ standards. For thoracic coarse particles, we plan to assume that on any day when the $PM_{2.5}$ concentration value has been approved for exclusion from use in determining compliance with the 24-hour $PM_{2.5}$ standard, it is also appropriate to treat the PM_{10} and $PM_{10-2.5}$ concentrations as excludable. In addition, any approved exceptions for PM_{10} data would result in exclusion of $PM_{10-2.5}$ estimates from those days and from consideration in the rollback process (see section 2.3). PM_{10} data have not been as systematically reviewed by State and local agencies and EPA for purposes of making requests and decisions regarding the exclusion of data, so there is more uncertainty about whether specific data should be excluded from consideration when testing to see if a monitoring site has air quality meeting the current PM_{10} standard or

alternative PM_{10} or $PM_{10-2.5}$ standards. There may be days for which States have requested exclusion of PM_{10} data and EPA has not made a decision on the request. In these situations, a sensitivity analysis may be conducted, in which such requests are presumed to be approved.

2.3 DEVELOPMENT OF ESTIMATES OF PM AIR QUALITY ASSUMING "JUST MEETING" CURRENT NAAQS AND POTENTIAL ALTERNATIVE NAAQS

2.3.1 Background and Conceptual Overview

In order to simulate air quality concentrations that "just meet" the current or potential alternative $PM_{2.5}$ standards in a study area, we consider what mathematical approach (commonly referred to as rollback) should be used to transform recent air quality into profiles of adjusted air quality that simulate just meeting the current or alternative standards under consideration. The form of the current $PM_{2.5}$ standards 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 24-hour standard. In determining attainment of the annual average standard, a State may choose to use either the spatially averaged[6] concentrations across all population-oriented monitors, subject to meeting certain criteria detailed in Appendix N of Part 50 of the CFR, or it may use the highest 3-year average concentration based on individual monitors. The form of the current 24-hour PM_{10} standard requires that the expected number of exceedances of the level of the standard per year, averaged over 3 years, is not greater than 1.0. The "expected" refers to an adjustment made to the count of actually monitored exceedances to adjust for monitoring schedules which are not every day.

The challenge in developing estimates of PM air quality for a scenario in which an assessment location is "just meeting" the current standards or alternative standards under consideration is to estimate as realistically as possible how concentrations on all days at all monitors will be affected, not just how the key air quality statistic from the controlling monitor

[6] In the last review of the $PM_{2.5}$ standards, the criteria for spatial averaging were made more restrictive, and presently no area follows the spatial averaging approach nor do we think it likely that any areas will do so in the future.

(or set of monitors being averaged) will be affected. The definition of "just meeting" alternative PM standards uses the same approach as "just meeting" the current standards, although some sets of potential alternative standards may include $PM_{10-2.5}$ standards instead of the current PM_{10} standard.

There are many possible ways to create characterizations of air quality to represent scenarios "just meeting" specified $PM_{2.5}$ (or $PM_{10-2.5}$) standards. The previous two reviews have used a method called proportional rollback, which is described below in section 2.3.2. This choice was based on analyses of historical $PM_{2.5}$ data which found, from comparing the reductions over time in daily ambient $PM_{2.5}$ levels in two locations with sufficient ambient air quality data, that reductions tended to be roughly proportional (Abt Associates, 2005, Appendix B). We recognize that the pattern of changes that have occurred in the past may not necessarily reflect the temporal and spatial patterns of changes that would likely result from future efforts to attain the $PM_{2.5}$ standards; therefore, we are considering examining an alternative prospective approach for rollback, as described in section 2.3.3.

2.3.2 Historical Approach

Prior $PM_{2.5}$ risk assessments that simulated $PM_{2.5}$ reductions that would result from just meeting a set of standards used a proportional adjustment ("proportional rollback") which decreased non-background PM levels on all days by the same percentage for all concentrations exceeding the PRB (U.S. EPA, 2005). The portion of the distribution below the estimated background concentration was not rolled back, since air quality strategies adopted to meet the standards would not be expected to reduce the background contribution to PM concentrations. The percentage amount of rollback was just enough so that neither the 24-hour nor the annual levels of the suite of standards under consideration were exceeded. Generally, the amount of rollback required to just meet the 24-hour and the annual levels were not the same, so, in practice, this brought the design value[7] for one of the 24-hour or the annual standards at

[7] In the risk assessment conducted for the last review, the annual average $PM_{2.5}$ concentration at each monitor 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. At each monitor, the 98th percentile $PM_{2.5}$ concentration was calculated for each of the years 2001, 2002, and 2003, and these three 98th (99th) percentile concentrations were then averaged to calculate the 24-hour $PM_{2.5}$ design value.

the controlling monitor to be equal to the level of the corresponding standard, while the design value of the other standard was reduced to a level below the standard.

In the risk assessment for this review, we will again evaluate the proportional rollback approach by comparing it with historical changes in distributions of $PM_{2.5}$ concentrations in selected locations (and $PM_{10-2.5}$ air quality, to the extent possible). Specifically, EPA plans to evaluate historical $PM_{2.5}$ air quality changes between 1999 and 2007 to assess the implications of using a proportional (PRB-adjusted) rollback approach. This type of analysis is similar to analyses conducted for Los Angeles and Philadelphia in the last risk assessment (Abt Associates, 2005, Appendix B). One difference is that in the last review, the composite (multi-monitor) 24-hour $PM_{2.5}$ concentrations were the subject of the analysis, while for this review, we plan to analyze monitors individually for the purposes of evaluating air quality changes over time.[8] We also plan to consider the premises and outcomes of the proportional rollback approach against our insights regarding known and likely future emission reductions, e.g., whether it is reasonable to expect that future patterns of changes in PM air quality would generally be similar to historical patterns of changes in air quality.

2.3.3 Alternative Approach Under Consideration

In this review, we are also considering an alternative approach to simulating just meeting current or alternative standards under consideration. This alternative approach would take into consideration information about possible future patterns of emissions reductions reflecting both federal regulations that are in place that will affect $PM_{2.5}$ and precursor emissions as well as possible actions by states to meet current NAAQS. With respect to the historical rollback approach described above, we recognized in the last review that the historical changes in ambient $PM_{2.5}$ concentrations found to be generally proportional may not have been the result of control strategies designed to meet a $PM_{2.5}$ NAAQS, but likely resulted from stationary source control programs for PM_{10} and other pollutants (especially sulfur and nitrogen oxides) and from multi-pollutant reductions achieved by the national motor vehicle emission control program.

[8] This type of analysis has been conducted for the ongoing review of the primary NO_2 NAAQS (see Rizzo, 2008 for a more complete explanation).

Therefore, we recognize that the pattern of changes that had occurred in the past may not necessarily reflect changes that may result from future efforts to attain the $PM_{2.5}$ standards.

More specifically, EPA is investigating the possibility of developing and using a new, alternative approach for simulating $PM_{2.5}$ air quality "just meeting" the current or alternative $PM_{2.5}$ NAAQS. This "model-based rollback" approach relies upon results from EPA's Community Multiscale Air Quality (CMAQ) model (Byun et al., 2006 and Byun et al., 1999) that reflect both federal regulations that are in place (or "on the books") as well as possible actions by States to meet the current NAAQS. By using CMAQ modeled response, we can provide information about the non-linear nature of the air quality response to reductions in precursor emissions and direct $PM_{2.5}$ emissions on $PM_{2.5}$ concentrations at monitor locations. Since the available modeled response does not reflect meeting the current or potential alternative standards in all study areas, we also need to consider how to make further adjustments to modeled responses that may be needed in some study areas to simulate just meeting some of the standards under consideration. This modeling approach may serve as the basis for a methodology for monitor rollback that we believe has the potential to better reflect expected reductions in $PM_{2.5}$ concentrations at monitor locations. Therefore, we plan on evaluating the utility of this alternative approach compared to the historical approach based on "proportional rollback" to determine the most appropriate method for adjusting monitors to "just meet" the current and potential alternative $PM_{2.5}$ NAAQS.

2.4 POLICY RELEVANT BACKGROUND

For the purposes of the risk assessment, background PM is defined as the distribution of PM concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made) emissions of primary PM and emissions of precursors to secondary PM (e.g., VOC, NOx, SOx, and NH_3) in the U.S., Canada, and Mexico. We refer to background levels so defined as policy-relevant background (PRB), since this definition of background is intended to facilitate separating pollution levels that can be addressed by U.S. regulations (or through international agreements with neighboring countries) from levels that are generally uncontrollable by U.S. regulations.

For this assessment, we are planning to estimate levels of PRB using a CTM-based approach which involves coupling the global-scale circulation model GEOS-Chem (Fiore et al., 2003) with the regional scale air quality model CMAQ (Byun et al., 2006 and Byun et al, 1999). The GEOS-Chem model is run on a global scale and is used to provide estimates of transported pollutants from emissions of natural and anthropogenic sources outside the U.S., Canada, and Mexico. These transported pollutant concentrations are used to provide the boundary condition concentrations for two CMAQ simulations covering the continental U.S. and adjacent portions of Canada and Mexico (CONUS), one simulation of current conditions to evaluate model performance and one to estimate PRB. In the CMAQ simulation to estimate PRB, only natural emissions in the U.S., Canada, and Mexico are considered. The details of this modeling approach (EPA 2008b, section 3.6) are briefly summarized below.

The two models have been applied to simulate one year of air quality data for 2004. The base case CMAQ run for 2004 includes meteorology and all the anthropogenic and natural sources both within and outside of the U.S., Canada and Mexico. This run was performed to provide a comparison of model predictions with measurements. The first draft ISA characterizes the CMAQ performance for the annual average concentrations and for most of the seasonal averages of $PM_{2.5}$ at remote sites as very good in the East and Midwest. In the West, predictions at remote sites are generally too low in all seasons. The first draft ISA further notes that degraded performance in the West is not unexpected because the grid resolution in the CMAQ model simulation (36 km for this application) will smooth out significant variations in terrain that influence measured concentrations, particularly concentrations attributable to anthropogenic emissions which in the West are often concentrated in basin settings where local meteorological conditions coupled with local emissions of primary particles may dominate $PM_{2.5}$ concentrations. However, looking across the U.S., the model does correctly reproduce broad geospatial differences in that predicted $PM_{2.5}$ concentrations are lower at western locations than they are in the East, consistent with measured ambient data. Also, natural emissions in the West are less concentrated in basin settings and terrain and, therefore, western terrain may have less effect on model performance when estimating PRB.

In addition to the "base case" run which includes all anthropogenic and biogenic emissions, CMAQ was also run for a second scenario to estimate PRB, with the same boundary conditions but with only natural emissions from within the U.S., Canada, and Mexico. The hourly outputs from this second CMAQ run were used to calculate seasonal and annual average estimates of PRB within seven regions of the U.S. These data are provided in Table 3-26 of the first draft ISA (U.S. EPA, 2008b, page 3-128). We plan to use 24-hour average concentrations of PRB from this CMAQ run (not reported in the first draft ISA) as input to portions of the risk assessment.

2.5 BROADER AIR QUALITY CHARACTERIZATION

Information presented in the REA will draw upon air quality data analyzed in the ISA as well as national and regional trends in air quality as evaluated in EPA's Air Quality Status and Trends document (U.S., 2008d), and EPA's Report on the Environment (U.S. EPA, 2008e). We plan to use this information, and additional analyses, as needed, to develop a broad characterization of current air quality across the nation. For example, tables of areas and population in the U.S. exceeding current $PM_{2.5}$ and PM_{10} standards and potential alternative standards would be prepared. Additional information would be generated on the expected number of days on which the 24-hour $PM_{2.5}$ and PM_{10} standards are exceeded, adjusting for the number of days monitored. Further, daily $PM_{2.5}$ levels in locations and time periods relevant to areas assessed in key short-term epidemiological studies would be characterized. Information on the spatial, temporal, and compositional characterization of $PM_{2.5}$ across the national monitoring network would be compiled. To the extent possible, we plan to compare these data to the same parameters in the selected urban study areas considered in the quantitative risk assessment to help place the results of that assessment into a broader context.

3 SCOPE AND APPROACH FOR THE HEALTH RISK ASSESSMENT

3.1 INTRODUCTION

This chapter presents an overview of the design of the human health risk assessment to be conducted in the current review of the PM NAAQS. This design reflects goals laid out in the Integrated Review Plan (U.S. EPA, 2008a, section 5.5) including: (1) to provide estimates of the potential magnitude of premature mortality and/or selected morbidity health effects in the population associated with recent ambient PM levels and with just meeting the current suite of PM standards and any alternative standards that might be considered in selected urban study areas; (2) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates; and (3) to gain insights into the distribution of risks and patterns of risk reduction and uncertainties in those risk estimates. In addition, we are considering conducting an assessment to provide nationwide estimates of the potential magnitude of premature mortality associated with long-term exposure to ambient $PM_{2.5}$ to more broadly characterize this risk on a national scale and to support the interpretation of the more detailed risk estimates generated for selected urban study areas. As part of the risk assessment, where feasible, quantitative characterizations of the uncertainties associated with the risk estimates will be developed.

As outlined in section 1.3.2, the planned health risk assessment will build upon the methodology, analyses, and lessons learned from the assessments conducted for the last review. In the Integrated Review Plan, we recognized a potentially broad scope for the quantitative risk assessment and proposed to focus our efforts on fine particles ($PM_{2.5}$), and to consider, to the extent relevant information is available, risks associated with thoracic coarse particles ($PM_{10-2.5}$), as well as risks associated with specific PM components, sources, and/or environments (U.S. EPA, 2008a, section 5.5).

With respect to fine particles, based upon the information assessed in the first draft ISA, we plan to focus the risk assessment on health effect endpoints for which the weight of the evidence as assessed in the ISA supports the judgment that the overall health effect category is at least likely caused by exposure to fine particles ($PM_{2.5}$) either alone and/or in combination with other pollutants. The planned quantitative risk assessment, is designed to estimate risks associated with short- (24-hour

average) and long-term (annual average) ambient $PM_{2.5}$ concentrations in selected urban study areas. We are considering expanding the focus of this risk assessment to include additional health effect categories beyond those classified as casual or likely causal, when available evidence presented in the ISA is sufficiently suggestive of a causal association to support conducting quantitative risk assessment and when inclusion of that endpoint category will allow us to address potentially important policy issues related to reviewing the $PM_{2.5}$ NAAQS. For example, we are considering including information on birth outcome effects associated with ambient $PM_{2.5}$ which would allow us to evaluate additional potentially sensitivity subpopulations (i.e., pregnant women and infants) not previously evaluated in the quantitative risk assessment conducted in the last review. EPA recognizes that a decision to include these additional endpoint categories needs to consider the increased uncertainty that their inclusion could introduce into the risk assessment; specifically, the potential for these endpoints not to be associated with $PM_{2.5}$ exposure, despite the generation of risk estimates.

Building upon the assessment completed in the last review, we plan to focus the $PM_{2.5}$ assessment on modeling risk for a set of selected urban study areas, expanding the number of study areas modeled from nine areas to a somewhat larger set of urban areas (e.g., 15-20 study areas) in order to provide greater population coverage and to better portray the observed heterogeneity in $PM_{2.5}$-related risk across selected urban study areas. EPA is considering ways to put the quantitative risk assessment results conducted for a limited number of locations and selected health endpoints into a broader context to better characterize the nature, magnitude, extent, variability, and uncertainty of the public health impacts associated with $PM_{2.5}$ exposures. This includes plans for (1) an exposure assessment (Chapter 4.0); (2) evaluation of the urban study areas with respect to key $PM_{2.5}$ risk-related parameters to help inform judgments about the representativeness of the urban areas included in the assessment (section 3.4); and (3) consideration of a national-scale health impact assessment (section 3.5).

With respect to evaluating the public health impacts of thoracic coarse particles ($PM_{10-2.5}$), we recognize that the first draft ISA presents more limited data for this size fraction. As outlined in section 1.3.2, the planned health risk assessment will build upon the methodology, analyses, and lessons learned from the assessments conducted for the last review. Based on the information assessed in the first draft ISA, no health effect categories have been classified as having a likely causal or causal

association with ambient $PM_{10-2.5}$. Taking this into account, we plan to focus this assessment on selected health effect endpoints within broader health effect categories for which the evidence, as assessed in the first draft ISA is sufficiently suggestive of a casual association to support conducting a quantitative risk assessment related to short-term (24-hour) ambient $PM_{10-2.5}$ exposures. The planned approach to model risk for $PM_{10-2.5}$ will, in many respects, follow the approach planned for evaluating $PM_{2.5}$-related risks though with a much more limited set of health endpoints and a smaller number of selected urban study areas. We recognize that the current standard uses a PM_{10} indicator to provide protection against exposures to thoracic coarse particles and that a large number of epidemiological studies use PM_{10} as an air quality metric. However, based upon the information presented in the first draft ISA, it is difficult to evaluate the health effects associated with fine versus thoracic coarse particles in studies that use PM_{10} as a metric.

Regarding PM composition, based on information analyzed in the first draft ISA, we do not plan to develop separate risk estimates for $PM_{2.5}$ or $PM_{10-2.5}$ components and we plan to continue to model risk using the basic mass size-fraction approach used in the last review.[9] In addition, based on information presented in the first draft ISA, we believe the data are too limited to support conducting a quantitative risk assessment for ultrafine particles.

The following discussion begins by presenting the framework for the risk assessment developed to evaluate $PM_{2.5}$ with more detailed discussions of key components of the risk assessment model including air quality considerations, selection of health effects endpoints to include in the assessment, and specification of concentration-response (C-R) functions (section 3.2). As part of the assessment, where feasible, quantitative characterizations addressing uncertainty and variability associated with the $PM_{2.5}$ risk estimates will be developed (section 3.3). This section not only presents how we plan to assess and characterize uncertainty potentially impacting the risk assessment but also discusses the degree to which variability related to $PM_{2.5}$ risk is captured in the analysis design. Section 3.4 discusses the types of risk metrics that may be generated for $PM_{2.5}$, including how these results could be used to inform consideration of existing and alternative standards. In section 3.5, we

[9] See section 2.3.3 of the first draft ISA (U.S. EPA, 2008b) for a discussion of evidence related to $PM_{2.5}$ components/sources and associations with specific health effects. We do not plan to incorporate component-specific risk modeling into our analysis, however, as discussed in Section 3.3.2, the use of location-specific effects estimates potentially reflects underlying differences in $PM_{2.5}$ composition as well as additional factors related to $PM_{2.5}$-related risk including underlying health status of the study population and exposure-related factors such as prevalence of air conditioning use.

discuss the approach being considered for conducting a national-scale health impact assessment. Lastly, in section 3.6 we present plans to estimate risks associated with thoracic coarse particles ($PM_{10-2.5}$). Given the similarity of the risk assessment approaches for $PM_{2.5}$ and $PM_{10-2.5}$, much of section 3.6 references earlier sections covering elements of the $PM_{2.5}$ risk assessment approach.

3.2 FRAMEWORK FOR THE $PM_{2.5}$ HEALTH RISK ASSESSMENT

Overview of Modeling Approach

Building on the risk assessment conducted for the last review, the modeling approach we plan to use is based on a risk model whose components are illustrated in Figure 3-1. The calculation of risks is based on Equation 3-1 which combines information about changes in ambient $PM_{2.5}$ air quality concentrations (Δx) with C-R relationships (reflected by ß, the $PM_{2.5}$ coefficient derived from epidemiological studies) and baseline health incidence data for specific health endpoints (y) to derive estimates of the change in incidence (Δy) of specific health effects attributable to ambient PM concentrations during the period examined.[10]

Equation 3-1 $$\Delta y = y\left[e^{\beta \Delta x} - 1\right]$$

This type of risk model is based on epidemiological studies characterizing the relationship between ambient $PM_{2.5}$ levels measured at fixed-site population-oriented monitors and the incidence of specific health endpoints in the population, therefore, it does not require more detailed individual-level exposure modeling and relies instead, on the use of ambient monitoring data. Specifically, a change in the level of ambient $PM_{2.5}$ is translated through the effect estimate (coefficient ß) to a change in the baseline rate of a particular health effect(s) in the study population. This adjustment to the baseline incidence rate can then be combined with population estimates to generate an overall change in the incidence of a specific health endpoint(s) which is attributable to the change in ambient $PM_{2.5}$ concentrations.

As illustrated in Figure 3-1, this risk assessment approach requires specifying a number of modeling components related to (a) characterizing air quality, (b) establishing the C-R functions, and

(c) specifying the baseline incidence rates and population demographics. The remainder of this section discusses each of these modeling components in detail.

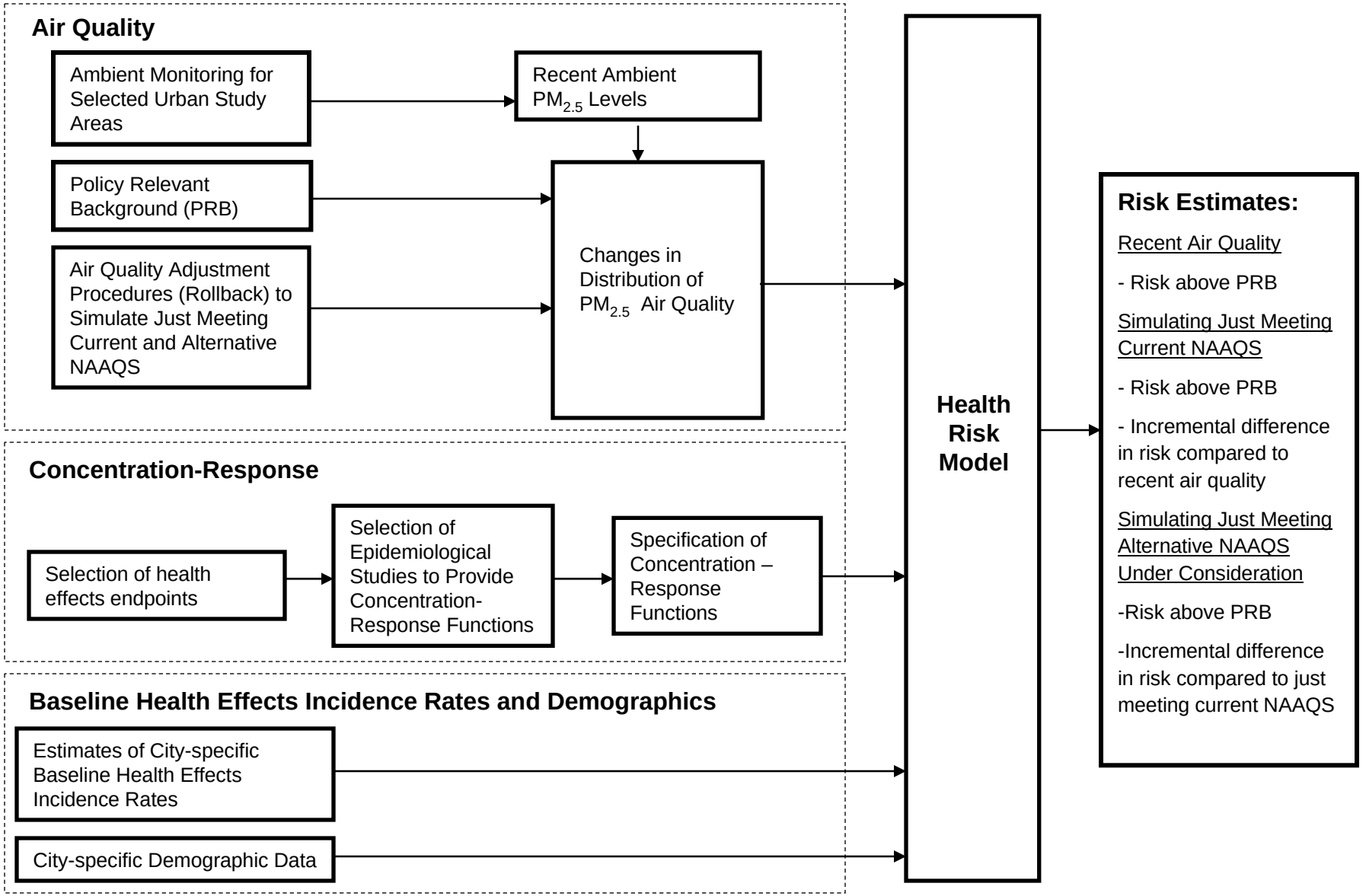
3.2.1 Air Quality Considerations

There are several air quality inputs to the risk assessment as illustrated in Figure 3-1. These have been described in Chapter 2 and include: (a) ambient $PM_{2.5}$ levels for each selected urban study area (including identification of population-oriented monitors and a method for aggregating those monitors and linking them to the study population, in a manner consistent with the approach used in the epidemiological studies underlying the concentration-response functions) for each year of the assessment, (b) quarterly average PRB concentrations for each selected urban study area, and (c) ambient air quality data sets adjusted to simulate air quality conditions that just meet current and alternative PM NAAQS under consideration. Additional detail on these inputs is presented below:

- **Characterizing recent ambient $PM_{2.5}$ levels for selected urban study areas**: EPA plans to use 3 years (2005-2007) of ambient $PM_{2.5}$ measurement data to characterize recent air quality conditions (see section 2.2). In aggregating monitoring data (to form composite monitor(s) for each study area) and linking those monitors to study populations within a particular study area, we plan to match, to the extent possible, the approach for analyzing air quality data used in the epidemiological studies from which the C-R functions are obtained. For example, in order to be consistent with the approach generally used in the epidemiological studies from which C-R functions have been estimated for effects associated with long-term $PM_{2.5}$ exposures, we plan to develop and use ambient data for a single composite monitor based on monitored data from all eligible monitors in that study area. Note, that some epidemiological studies have used more sophisticated (and spatially-refined) methods for associating ambient $PM_{2.5}$ data with a study population (e.g., Jerrett et al., 2005). In cases where we include C-R functions from studies using alternative methods to link ambient $PM_{2.5}$ concentrations with health effects information in our risk assessment, we may consider a more refined approach for linking $PM_{2.5}$ monitoring data with study populations, to match the approach used in the study.

[10] The health risk model given in Equation 3-1 is based on a concentration-response function in which the natural logarithm of the incidence of the health effect is a linear function of $PM_{2.5}$ concentration. We plan to consider other mathematical forms where epidemiological studies have reported effects using other model forms.

Figure 3-1 Overview of Risk Assessment Model for $PM_{2.5}$ (including key model components)



- **Characterizing PRB:** As noted in section 2.4, we will rely on regionally-differentiated characterization of PRB provided by the assessment summarized in the first draft ISA.

- **Method for adjusting ambient air quality levels to simulate air quality just meeting current and potential alternative $PM_{2.5}$ NAAQS**: As discussed in section 2.3, EPA is considering two methods for simulating $PM_{2.5}$ levels to just meet current or alternative NAAQS – a proportional rollback approach and a model-based approach.

3.2.2 Selection of Health Effects Endpoint Categories

As noted in section 3.1, based on review of the first draft ISA, we plan to focus the risk assessment primarily on fine particles, estimating potential health impacts associated with both short-term and long-term exposures to $PM_{2.5}$. In selecting health effects endpoints to include as an initial matter in the risk assessment, we have considered the following factors based upon review of the first draft ISA (U.S. EPA, 2008b; Chapters 2, 6, and 7): (a) the extent to which the health effect endpoints are considered significant from a public health standpoint, (b) the overall weight of the evidence from the collective body of epidemiological, clinical, and toxicological studies and the inferences made in the first draft ISA as to whether there is a causal or likely causal relationship between $PM_{2.5}$ and the health effect category, (c) whether there is sufficient evidence to support a causal or likely causal relationship for the specific health endpoint within the health effect category to warrant inclusion in the risk assessment, and (d) whether there are well-conducted studies reporting estimated C-R functions for specific health endpoints within the broader health effect endpoint category associated with ambient $PM_{2.5}$ levels.

Based upon review of the first draft ISA, we plan to consider the following health effect endpoint categories in this assessment:

Health Effect Categories Associated with Short-term $PM_{2.5}$ Exposure

- cardiovascular morbidity (causal association)
- respiratory morbidity (likely causal association)
- mortality (likely causal association)

Health Effect Categories Associated with Long-term $PM_{2.5}$ Exposure

- cardiovascular morbidity (likely casual association)

- respiratory morbidity (likely casual association)
- mortality (likely casual association)

We are considering expanding the focus of the $PM_{2.5}$ risk assessment to include additional health effect endpoints from health effect categories that have been initially judged in the first draft PM ISA to have a suggestive causal association with ambient $PM_{2.5}$ measurements. We plan to consider including these additional endpoints when they allow us to address potentially important policy issues related to reviewing the current $PM_{2.5}$ standards. For example, we are considering including information on birth outcome effects associated with ambient $PM_{2.5}$ which would allow us to evaluate additional potentially sensitive subpopulations (i.e., pregnant women and infants) not previously evaluated in the quantitative risk assessment conducted in the last review. [11] EPA recognizes that a decision to include this additional endpoint category would need to appropriately characterize the increased uncertainty associated with these additional outcomes.

3.2.3 Specification of Concentration-Response Functions

As noted above, the risk assessment conducted in this review will build on the approach developed and applied in the last review. EPA will rely on a weight-of evidence approach, based on the ISA's evaluation of new and previously reviewed epidemiologic studies including identification of relevant C-R functions that characterize the relationships between short- and long-term $PM_{2.5}$ exposures and health outcomes, particularly those conducted at or near current ambient concentrations. Quantitative relationships provided in the specific studies (or to be derived by EPA from the data presented in the epidemiologic studies) describe the change in concentration (generally based on ambient fixed-site monitors) associated with a change in health response. These C-R relationships will be combined with air quality data, baseline incidence data, and population data to develop population health risk estimates.

[11] As noted in the first draft ISA , there are limitations in the evidence assessing the relation between $PM_{2.5}$ exposure and reproductive/developmental effects (U.S. EPA, 2008b, page 7-84). Specifically, there are fewer studies with often inconsistent results in comparison to the evidence available for other endpoint categories (e.g., cardiovascular/ respiratory morbidity and mortality). In addition, characterizing $PM_{2.5}$ exposure at the etiologically relevant time period for developmental effects, and understanding the biological mechanisms underpinning these relations remain important challenges. Despite these limitations, studies have reported associations between ambient $PM_{2.5}$ exposure and low birth weight, preterm birth, and respiratory-specific infant mortality, respectively. These results support the consideration of risk associated with developmental effects in relation to ambient $PM_{2.5}$ exposure.

We plan to use specific criteria to select the epidemiological studies that will be used to provide C-R functions for the quantitative risk assessment including:

- The study addresses one of the health effects endpoint categories identified for inclusion in the risk assessment.
- The study was peer-reviewed study, evaluated in the first draft ISA, and judged adequate by EPA staff for purposes of inclusion in the risk assessment. Criteria considered by staff include: whether the study provides C-R relationships for locations in the U.S., whether the study has sufficient sample size to provide effect estimates with a sufficient degree of precision and power, whether the study is a multi-city study, and whether adequate information is provided to characterize statistical uncertainty.
- The study directly measured $PM_{2.5}$ (i.e., it did not use a surrogate measure such as airport visibility).
- The study is not superseded by another study (e.g., if a later study is an extension or replication of a former study, the later study would effectively replace the former study), unless the earlier study has characteristics that are clearly preferable.

In addition to the above criteria, other factors, which may be specific to a particular health effect endpoint, or even to a set of studies, may be considered. For example, several of the studies have improved upon the method of estimating the exposure metric used in most studies which have generally relied upon population-oriented monitoring data. Instead of assigning the same ambient $PM_{2.5}$ concentration to all individuals in a city (based on a central monitor or the average of several monitors in a city), these studies have assigned "exposures" according to monitors that better approximate conditions near subjects' residences (for example, see Jerrett et al., 2005). In addition, at least one long-term exposure mortality study (Villeneuve et al., 2002) takes into account that exposure changes over time. These and similar studies may provide additional insights into whether reductions in mortality are attributable to recent, or more historical changes in patterns of long-term $PM_{2.5}$ exposure.

We also plan to consider the overall study design, including the method used to adjust for covariates (including confounders and effects modifiers) in identifying candidate studies. For example, if a given study uses ecological-defined variables (e.g., smoking rates) as the basis for controlling for confounding, concerns may be raised as to the effectiveness of that control.

These factors related to confounding control and consideration of effects modification also will be considered in identifying studies for use as the basis of C-R functions.

Based on application of these criteria to the set of studies reporting $PM_{2.5}$ effect estimates evaluated in the first draft ISA, we have identified a provisional set of studies as candidate studies for use in specifying C-R functions relating short-term $PM_{2.5}$ ambient concentrations with health effects. Table 3-1 includes information on this initial set of studies, as well as the health effect category (and specific endpoints) evaluated in the studies. We plan to continue to refine this list in order to identify a final set of studies for use in deriving C-R functions for inclusion in the risk assessment.

With regard to long-term $PM_{2.5}$ exposure studies, application of the criteria listed above has resulted in a preliminary set of studies listed in Table 3-2 . We plan to continue to refine this list prior to selecting specific C-R functions for use in the quantitative risk assessment evaluating $PM_{2.5}$-related health effects associated with long-term ambient exposures (annual average concentrations).

Once the final set of epidemiological studies is chosen, the next step will be the selection of C-R functions from those studies. A number of factors need to be considered in specifying C-R functions related to short- and long-term exposure studies. The factors being considered in selecting C-R functions include:

- **Single- and multi-pollutant models (*pertains to both short-term and long-term exposure studies*)**: Epidemiological studies often consider health effects associated with ambient $PM_{2.5}$ independently as well as together with co-pollutants (e.g., ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide). To the extent that any of the co-pollutants present in the ambient air may have contributed to health effects attributed to $PM_{2.5}$ in single pollutant models, risks attributed to $PM_{2.5}$ may be overestimated if C-R functions are based on single pollutant models. This would argue for inclusion of models reflecting consideration of co-pollutants. Conversely, in those instances where co-pollutants are highly correlated with $PM_{2.5}$, inclusion of those pollutants in the health impact model can produce unstable and statistically insignificant effect estimates for both $PM_{2.5}$ and the co-pollutants. This situation would argue for inclusion of a model based exclusively on $PM_{2.5}$. Given that single and multi-pollutant models each have potential advantages and disadvantages, we plan to include both types of C-R functions in the risk assessment.

Table 3-1. Provisional Summary of Short-Term Epidemiological Studies Being Considered as Basis for Selecting C-R Functions for the PM2.5 Risk Assessment

Location	All cause mortality	Cardiovascular mortality	Respiratory mortality	Respiratory hospital admissions	Cardiovascular hospital admissions	ER visits	Respiratory Symptoms (Not requiring hospitalization)
Region: West/Southwest (Minus CA)							
Anchorage, AK				Chimonas et al. (2007) Dominici et al. (2006)***			
El Paso, TX	Franklin et al.(2008)	Franklin et al.(2008)	Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Las Vegas, NV	Franklin et al.(2007)**	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)****	Dominici et al. (2006) Bell et al. (2008)		
Phoenix, AZ	Franklin et al.(2007)	Franklin et al.(2007) Wilson et al. (2007) Mar (2003) [reanalysis of Mar (2000)]	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Seattle, WA	Franklin et al.(2008)** Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008) Sheppard (2003) [reanalysis of Sheppard et al. (1999)] - asthma	Dominici et al. (2006) Bell et al. (2008)		Slaughter et al. 2003
Spokane, WA	Slaughter et al.(2005)			Dominici et al. (2006) Bell et al. (2008) Slaughter et al. (2005)	Dominici et al. (2006) Bell et al. (2008) Slaughter et al. (2005)	Slaughter et al. (2005) (respiratory)	Mar et al. 2004
Region: California							
Bakersfield/ Kern Co., CA	Franklin et al.(2008) Ostro et al. (2007)	Franklin et al.(2008) Ostro et al. (2006) Ostro et al. (2007)	Franklin et al.(2008) Ostro et al. (2006)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Contra Costa, CA				Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Fresno, CA	Franklin et al.(2008) Franklin et al.(2007) Ostro et al. (2007)	Franklin et al.(2008) Franklin et al.(2007) Ostro et al. (2006) Ostro et al. (2007)	Franklin et al.(2008) Franklin et al.(2007) Ostro et al. (2006)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Los Angeles, CA	Franklin et al.(2008) Franklin et al.(2007) Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)] Jerrett et al. (2005)	Franklin et al.(2008) Franklin et al.(2007) Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)] Jerrett et al. (2005) Ostro et al. (2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008) Moolgavkar 2003 (Reanalysis of Moolgavkar, 2000)		
Orange, CA		Ostro et al. (2007)		Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Riverside, CA	Franklin et al.(2008) Ostro et al. (2007) Franklin et al.(2007)	Franklin et al.(2008) Ostro et al. (2006) Ostro et al. (2007) Franklin et al.(2007)	Franklin et al.(2008) Ostro et al. (2006) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		

Table 3-1. Provisional Summary of Short-Term Epidemiological Studies Being Considered as Basis for Selecting C-R Functions for the PM2.5 Risk Assessment

Location	All cause mortality	Cardiovascular mortality	Respiratory mortality	Respiratory hospital admissions	Cardiovascular hospital admissions	ER visits	Respiratory Symptoms (Not requiring hospitalization)
Sacramento, CA	Franklin et al.(2008) Ostro et al. (2007) Franklin et al.(2007)	Franklin et al.(2008) Ostro et al. (2006) Ostro et al. (2007) Franklin et al.(2007)	Franklin et al.(2008) Ostro et al. (2006) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
San Diego, CA	Franklin et al.(2008) Ostro et al. (2007) Franklin et al.(2007)	Franklin et al.(2008) Ostro et al. (2006) Ostro et al. (2007) Franklin et al.(2007)	Franklin et al.(2008) Ostro et al. (2006) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
San Jose, CA	Fairley (2003) [reanalysis of Fairley (1999)]	Fairley (2003) [reanalysis of Fairley (1999)]	Fairley (2003) [reanalysis of Fairley (1999)]	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Santa Clara Co., CA	Ostro et al. (2006) Ostro et al. (2007)	Ostro et al. (2007)	Ostro et al. (2006)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Region: South							
Atlanta, GA	Klemm et al. (2004)			Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)	Metzger et al. (2004) (cardiovascular); Peel et al. (2005) (respiratory) Tolbert et al. (2007) (cardiovascular)	
Beaumont, TX	Franklin et al.(2008)	Franklin et al.(2008)	Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Birmingham, AL	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Dallas, TX	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Durham, Guilford, and Wake Counties, NC		Holloman et al. (2004)		Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Houston, TX	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Kingston/Harriman, TN	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]		Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		Schwartz and Neas, 2000
Memphis, TN	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Palm Beach, FL	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Tampa, FL	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		

Table 3-1. Provisional Summary of Short-Term Epidemiological Studies Being Considered as Basis for Selecting C-R Functions for the PM2.5 Risk Assessment

Location	All cause mortality	Cardiovascular mortality	Respiratory mortality	Respiratory hospital admissions	Cardiovascular hospital admissions	ER visits	Respiratory Symptoms (Not requiring hospitalization)
Region: Midwest/Central							
Akron, OH	Franklin et al.(2008)	Franklin et al.(2008)	Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Chicago, IL	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Cincinnati, OH	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Cleveland, OH	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Columbus, OH	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Dayton, OH	Franklin et al.(2008)	Franklin et al.(2008)	Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Detroit, MI	Ito (2003) [reanalysis of Lippmann et al. (2000)] Franklin et al.(2008) Franklin et al.(2007)	Ito (2003) [reanalysis of Lippmann et al. (2000)] Franklin et al.(2008) Franklin et al.(2007)	Ito (2003) [reanalysis of Lippmann et al. (2000)] Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Ito (2003) [Reanalysis of Lippmann et al. 2000] Dominici et al. (2006) Bell et al. (2008)		
Erie, PA	Franklin et al.(2008)	Franklin et al.(2008)	Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Harrisburg, PA	Franklin et al.(2008)	Franklin et al.(2008)	Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Indianapolis, IN	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Kansas City, MO	Franklin et al.(2008)	Franklin et al.(2008)	Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Milwaukee, WI	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Minneapolis, MN	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Pittsburgh, PA	Franklin et al.(2008) Franklin et al.(2007) Chock et al. (2000)	Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Portage, WI	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]		Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		Schwartz and Neas, 2000
St. Louis, MO	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] Franklin et al.(2008)	Franklin et al.(2008)	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		Schwartz and Neas, 2000
Steubenville, OH	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]		Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		Schwartz and Neas, 2000

Table 3-1. Provisional Summary of Short-Term Epidemiological Studies Being Considered as Basis for Selecting C-R Functions for the PM2.5 Risk Assessment

Location	All cause mortality	Cardiovascular mortality	Respiratory mortality	Respiratory hospital admissions	Cardiovascular hospital admissions	ER visits	Respiratory Symptoms (Not requiring hospitalization)
Toledo, OH	Franklin et al.(2008)	Franklin et al.(2008)	Franklin et al.(2008)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Topeka, KA	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]		Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		Schwartz and Neas, 2000
Region: Northeast							
Baltimore, MD				Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008) Symons et al. (2006)		
Boston, MA	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] Franklin et al.(2008) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		Schwartz and Neas, 2000
New York, NY	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Bell et al. 2008 Dominici et al. (2006)	Dominici et al. (2006) Bell et al. (2008)	Ito et al. (2007) (respiratory)	
Philadelphia, PA	Franklin et al.(2008) Lipfert et al. (2000) Franklin et al.(2007)	Franklin et al.(2008) Lipfert et al. (2000) Franklin et al.(2007)	Franklin et al.(2008) Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008)	Dominici et al. (2006) Bell et al. (2008)		
Washington, DC	Franklin et al.(2007)	Franklin et al.(2007)	Franklin et al.(2007)	Dominici et al. (2006) Bell et al. (2008) Babin et al (2007)	Dominici et al. (2006) Bell et al. (2008)	Babin et al (2007) (respiratory)	

*Studies were included in this table regardless of whether their results were statistically significant, as long as they reported results for one of the health endpoint categories in the table.

**Location-specific results are shown in these papers only in a figure (but may be available from the study authors).

***Dominici et al. (2006) included the 204 U.S. counties with populations > 200,000, but reported results by region of the country. The individual counties included in this study are listed at: http://www.biostat.jhsph.edu/MCAPS/county-info.html.

****Bell et al. (2008) used the same set of counties as Dominici et al. (2006) (minus 4 counties); results are reported by region of the country; if there are county-specific results for the county of the location,

Table 3-2. Provisional Summary of Long-Term Epidemiological Studies Being Considered for Selecting C-R Functions for the $PM_{2.5}$ Risk Assessment

Study	Health effect endpoint categories (mortality categories, unless otherwise noted)	Study locations
Dockery et al., 1993 (Six Cities Study)	• all-cause, • lung cancer, • cardiopulmonary	6 U.S. cities – Watertown, MA; Kingston/Harriman, TN; St. Louis, MO; Steubenville, OH; Portage, WI; Topeka, KS
Krewski et al. (2000) - Reanalysis of Six Cities Study	• all-cause, • lung cancer, • cardiopulmonary	
Villeneuve et al. (2002)	• all-cause	
Laden et al. (2006)	• all-cause, • lung cancer, • cardiovascular	
Eftim et al. (2008)	• all-cause	6 Harvard Six Cities locations and 110 counties (within the 50 ACS metropolitan areas)
Pope et al. (1995) - American Cancer Society (ACS) Study	• all-cause, • lung cancer, • cardiopulmonary	ACS locations (50 metropolitan areas)
Krewski et al. (2000) - Reanalysis of ACS Study	• all-cause, • lung cancer, • cardiopulmonary	ACS locations (50 metropolitan areas)
Pope et al. (2002) - ACS extended	• all-cause, • lung cancer, • cardiopulmonary	ACS metropolitan areas: 1979-1983: 61; 1999-2000: 116; average: 51.
Pope et al. (2004) -- ACS study	• cardiovascular	ACS metropolitan areas: 1979-1983: 61; 1999-2000: 116; average: 51.
Jerrett et al. (2005)	• all-cause, • lung cancer, • cardiopulmonary	Los Angeles
Enstrom (2005)	• all-cause, • cardiopulmonary	11 CA counties
McDonnell et al. (2000) (AHSMOG study)	• all-cause, • lung cancer • nonmalignant respiratory diseases*	11 airsheds in CA

Study	Health effect endpoint categories (mortality categories, unless otherwise noted)	Study locations
Chen et al. (2005) (AHSMOG study)	• ischemic heart disease/coronary heart disease	San Francisco, South Coast, and San Diego air basins in CA, plus a random sample from the rest of CA
Goss et al. (2004)	***Mortality:*** • all-cause ***Morbidity:*** Cystic Fibrosis pulmonary exacerbations	Locations around the U.S.**
Lipfert et al. (2006a and b)	• all-cause	Cohort in the following states: CT, DC, IL, IN, MD, MI, NJ, NY, OH, PA, AL, FL, TN, VA, AR, IA, KS, LA, MN, MS, WI, AZ, CA, OK, TX, UT, WA
Chen et al. (2005) (AHSMOG study)	• ischemic heart disease/coronary heart disease	San Francisco, South Coast, and San Diego air basins in CA, plus a random sample from the rest of CA
Miller at el. (2007)	***Mortality:*** • cardiovascular **Morbidity:** First event of any of the following: • Any cardiovascular event, • Coronary heart disease • Cerebrovascular disease • MI • Coronary revascularization Stroke	36 US metropolitan areas

*As either the underlying or a contributing cause of death.
** Subjects were taken from patients enrolled in the Cystic Fibrosis Foundation National Patient Registry in 1999 and 2000.

- **Single- versus multi-city studies (*typically a factor in short-term exposure studies)***: All else being equal, we judge C-R functions estimated in the assessment location as preferable to a function estimated in some other location, to avoid uncertainties that may exist due to differences associated with geographic location. 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. 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 health coefficient. However, multi-city studies may also mask potential differences in $PM_{2.5}$ composition and the impact that compositional differences may have on the magnitude of effects estimates. By contrast, single-city studies, while often having lower statistical power and varying study designs which can make comparison across cities challenging, do reflect location-specific factors such as $PM_{2.5}$ compositional differences, differences in underlying health status, and differences in exposure-related factors such as air conditioner use and urban density with larger populations exposed near high-traffic roads. Because single- and multi-city studies have different advantages, we plan to include both types of functions in this analysis, where they are available. We plan to place greater weight on the use of C-R relationships reflecting adjusted single-city estimates from multi-city studies. This would include Empirical Bayes adjusted city-specific estimates. These types of effect estimates benefit both from increased statistical power, as well as the potential for specification of city-specific effect estimates. Conversely, if a multi-city study only provides aggregated effect estimates, but does differentiate those estimates regionally, we plan to use those regional-specific estimates rather than a single national-level estimate by matching selected urban study areas to these regions.

- **Multiple lag models (*pertinent to short-term exposure time-series studies*)**: If information is available for a distributed lag model, we plan to use that model. Where there are multiple lags presented, but a distributed lag model is not included, we plan to consider information presented in the first draft ISA to determine if there is biological support for selecting a specific lag period for a given health effect endpoint.

- **Seasonally-differentiated effects estimates (*pertinent to short-term studies*)**: In those instances where studies presented effect estimates associated with short-term ambient $PM_{2.5}$ concentrations differentiated by season, we plan to use these seasonal estimates. We plan to link seasonal effect estimates with seasonal $PM_{2.5}$ air quality data in conducting the risk assessment for selected urban study areas.

- **Shape of the functional form of the risk model:** In the risk assessment conducted in the last review, EPA included C-R relationships that reflected linear or log-linear C-R functions that extended down to estimated PRB levels for effects related to short-term exposure and down to lowest measured ambient levels for effects related to long-term exposure, as well as adjusting these models to reflect various alternative "cutpoint" models. The alternative cutpoint models imposed an assumed threshold on the original C-R function, below which there is little or no population response. The first draft ISA

concludes that there is little support in the literature for a population threshold for mortality effects associated with long-term or short-term $PM_{2.5}$ ambient concentrations, although it also states that alternate model forms (including presence of a threshold – inference added) can not be ruled out on a city-specific basis (see U.S. EPA, 2008b, pp. 2-21 to 2-22). For this reason, while we plan to emphasize non-threshold C-R functions in the risk assessment model, we may also consider various cutpoints, or hypothetical population thresholds, as part of the uncertainty analysis.

In addition to the factors listed above, there are additional factors related to the design of individual epidemiological studies which we plan to consider in selecting the C-R functions to be included in the assessment. For example, studies often include adjustment for covariates with varying degrees of freedom, reflecting the tradeoff between bias and over-adjustment (loss of efficiency). In these cases, we plan to consider any information provided for specific studies within the first draft ISA and also plan to consider which model form has the strongest statistical fit, while still considering overall biological plausibility.

3.2.4 Selection of Urban Study Areas

We plan to build on the risk assessment conducted for the last review and continue to focus the risk assessment on a set of selected urban study areas. In the last review, nine urban areas were evaluated. In this review, we plan to focus on 15 to 20 urban areas. The decision to continue to focus on modeling a set of selected urban study areas reflects the goal of providing risk estimates that have higher overall confidence due to the use of location-specific data when available for these urban locations. In addition, given the greater availability of location-specific data a more rigorous evaluation of the impact of uncertainty and variability can be conducted for a set of selected urban study areas, than would be possible for a broader regional or national-scale analysis. We plan to consider the following factors in the selection of urban study areas:

- **Air quality data**: The urban area has sufficient recent (2005-2007) air quality data to conduct the risk assessment (See section 2.2.1).

- **Location-specific C-R functions**: There are C-R functions available from epidemiological studies identified in section 3.2.3, for one or more of the selected health endpoints. This primarily applies to short-term epidemiological studies, which more often include city-specific effect estimates (see Table 3-1). C-R functions available from long-term epidemiological studies generally combine data from multiple cities. Specific cities evaluated in the key long-term studies would be considered for inclusion in the risk assessment (see **Table 3-2**). We plan to include urban study areas that have been

assessed in epidemiological studies that have evaluated health effects associated with both short- and long-term $PM_{2.5}$ exposures and, to the extent possible, locations where both morbidity and mortality health endpoints have been evaluated.

- **Baseline incidence rates and demographic data**: The required urban area-specific baseline incidence rates and population data are available for a recent year for at least one of the health endpoints.

- **Geographic heterogeneity**: Because $PM_{2.5}$ composition and populations vary geographically across the U.S., we plan to select a set of urban study areas in which each region of the country is represented. We plan to define these regions in such a way as to reflect differences in factors related to $PM_{2.5}$ composition, sources, co-pollutants, exposure, and/or effect estimates.

- **Representing areas with relatively larger vulnerable populations**: Baseline incidence rates (e.g., mortality rates) and PM exposures are higher in some parts of the country than others. We plan to select a set of urban study areas that will include representation of sensitive subpopulations (e.g., those with higher baseline incidence rates of the health effect endpoints being evaluated, lower air conditioning usage which has been related to higher ambient PM exposures).

- **Consideration of epidemiology studies with more refined exposure metrics**: We plan to include urban study areas for which there is a C-R function estimated using a more refined metric of exposure (e.g., smaller geographic units linked to nearest PM monitors, rather than constructing a single composite monitor for an entire metropolitan area), where available.

3.2.5 Baseline Health Effects Incidence Data and Demographic Data

As noted earlier (section 3.2.1), the most common epidemiological-based health risk model expresses the reduction in health risk (Δy) associated with a given reduction in $PM_{2.5}$ concentrations (Δx) as a percentage of the baseline incidence (y). To accurately assess the impact of $PM_{2.5}$ air quality on health risk in the selected urban areas, information on the baseline incidence of health effects (i.e., the incidence under recent air quality conditions) in each location is needed. Where at all possible, we plan to use county-specific incidences or incidence rates (in combination with county-specific populations). A summary of available baseline incidence data for specific categories of effects is presented below:

- **Availability of baseline incidence data on mortality**: County-specific (and, if desired, age- and race-specific) baseline incidence data are available for all-cause and cause-

specific mortality from CDC Wonder.[12] The most recent year for which data are available online is 2005.[13]

- **Availability of baseline incidence data for hospital admissions and emergency room (ER) visits**:
    - Cause-specific hospital admissions baseline incidence data are available for each of 40 states from the State Inpatient Databases (SID).
    - Cause-specific ER visit baseline incidence data are available for 26 states from the State Emergency Department Databases (SEDD).
    - SID and SEDD are both developed through the Healthcare Cost and Utilization Project (HCUP), sponsored by the Agency for Healthcare Research and Quality (AHRQ).
    - The data generated from HCUPnet (HCUP's online interactive tool) are state-level summary statistics, whereas the data from the HCUP distributor are at the individual discharge level.
    - In addition to being able to estimate State-level rates, SID and SEDD can also be used to obtain county-level hospital admission and ER visit counts by aggregating the discharge records by county.

EPA is in the process of obtaining the county-specific hospital admission and ER visit baseline incidence data for the most recent single year available for most of the States included in the HCUP data. While we recognize that there is year-to-year variability in baseline incidence data, a single year of data is being obtained due to resource constraints. We plan to examine the potential variability in baseline incidence data and the impact this might have on the risk estimates in sensitivity analyses based on endpoints and locations where we can obtain multi-year baseline incidence data at little or no cost and by examining the variability in baseline incidence rates at the State level.

[12] http://wonder.cdc.gov/mortsql.html

[13] Note: For years 1999 – 2005, CDC Wonder uses ICD-10 codes; for years prior to 1999, it uses ICD-9 codes. Since most of the studies use ICD-9 codes, this means that EPA will have to create or find a mapping from ICD-9 codes to ICD-10 codes if the most recent data available are to be used.

3.3 CHARACTERIZATION OF UNCERTAINTY AND VARIABILITY IN THE CONTEXT OF THE $PM_{2.5}$ RISK ASSESSMENT

3.3.1 Differentiating Between Uncertainty and Variability

An important issue associated with any population health risk assessment is the characterization of uncertainty and variability. *Variability* refers to the heterogeneity in a population or variable of interest that is inherent and cannot be reduced through further research. For example, there may be variability among C-R functions describing the relation between $PM_{2.5}$ and mortality across selected urban study areas. This variability may be due to differences in population (e.g., age distribution), population activities that affect exposure to $PM_{2.5}$ (e.g., air conditioning use), levels and composition of $PM_{2.5}$ and/or co-pollutants, and/or other factors that vary either within or across urban areas.

Uncertainty refers to the lack of knowledge regarding both the actual values of model input variables (parameter uncertainty) and the physical systems or relationships (model uncertainty – e.g., the shapes of concentration-response functions). In any risk assessment, uncertainty is, ideally, reduced to the maximum extent possible, through improved measurement of key parameters and ongoing model refinement. However, significant uncertainty often remains and emphasis is then placed on characterizing the nature of that uncertainty and its impact on risk estimates. The characterization of uncertainty can include both qualitative and quantitative analyses, the latter requiring more detailed information and often, the application of sophisticated analytical techniques such as 2-stage Monte Carlo simulation.

While the goal in designing a quantitative risk assessment is to reduce uncertainty to the extent possible; with variability, the goal is to incorporate the sources of variability into the analysis approach to insure that the risk estimates are representative of the actual response of a study population (including the distribution of that adverse response across the study population). An additional aspect of variability which is pertinent to this risk assessment is the degree to which the set of selected urban study areas provide coverage for the range of $PM_{2.5}$-related risk experienced by the U.S. population.

We plan to more fully differentiate variability and uncertainty in the design of the risk assessment to more clearly address (a) the extent to which the risk estimates represent the distribution of health impacts across a population, including impacts on more susceptible and/or vulnerable subpopulations[14] and (b) the extent to which risk estimates are impacted by key sources of uncertainty which could prevent a clear differentiation between regulatory alternatives based on risk estimates.

The remainder of this section discusses how we are planning to address variability and uncertainty within the PM NAAQS risk assessment. The treatment of variability is discussed first (section 3.3.2) by identifying sources of variability associated with the modeling of $PM_{2.5}$-related risk and noting which of those sources are reflected in the risk modeling approach presented here. Next, the treatment of uncertainty is addressed, which will include both a qualitative and quantitative component. The qualitative component is described first (section 3.3.3), including plans for identifying and describing key sources of uncertainty, and noting whether those sources of uncertainty are addressed quantitatively in the risk assessment model. A preliminary list of key sources of uncertainty for the risk assessment is provided as part of this discussion. The quantitative component of the uncertainty characterization approach, which is structured around single-factor and multi-factor sensitivity analysis methods, is then described (section 3.3.4).

3.3.2 Addressing Variability

Key sources of variability associated with the modeling of population-level risk associated with $PM_{2.5}$ exposure are presented below, including whether, and to what extent, we plan to address each source of variability:

- **$PM_{2.5}$ composition**: We plan to address differences in $PM_{2.5}$ composition by using city-specific effects estimates, which can reflect, among other location-specific factors related to $PM_{2.5}$ exposure and risk, differences in $PM_{2.5}$ composition. We do not plan to explicitly consider $PM_{2.5}$ composition within the risk assessment because C-R functions for specific $PM_{2.5}$ components/sources have not been identified.

[14] *Susceptibility* refers to innate (e.g., genetic or developmental) or acquired (e.g., age, disease, or smoking) factors that make individuals more likely to experience effects with exposure to PM. *Vulnerability* refers to PM-related effects due to factors including socioeconomic status (e.g., reduced access to health care) or particularly elevated exposure levels.

- **Spatial gradients in $PM_{2.5}$ (and related population exposure)**: This source of variability is likely to be less-well captured in the risk assessment primarily because the majority of epidemiological studies providing effect estimates are themselves limited in reflecting more detailed patterns of $PM_{2.5}$ exposure among populations. More specifically, the epidemiological studies typically use an average ambient concentration developed across population-oriented monitors as a surrogate for exposure. Note, however that the exposure assessment described in Chapter 4 may allow this issue to be investigated to some degree, particularly as it impacts on exposure error misclassification in the epidemiological studies underpinning the C-R functions used in this risk assessment. In addition, a few epidemiological studies being considered for inclusion in this analysis include more refined characterization of population-level exposure (e.g., based on more spatially differentiated linkages between population-level monitors and segments of the study population). We plan to consider the use of those studies with more refined population exposure characterization to examine the issue of spatial gradients in $PM_{2.5}$ and demographics and the degree to which this source of variability impacts risk estimates.

- **Demographics (i.e., greater concentrations of susceptible subpopulations in certain locations)**: We plan to include multiple urban study areas reflecting differences in demographics in different regions of the country to address this issue. In addition, as noted in the previous bullet, we plan to consider studies with more refined characterization of population-level exposure, to provide insights into the degree to which this source of variability impacts risk estimates.

- **Behavior related to $PM_{2.5}$ exposure (e.g., outdoor time, air conditioning use)**: We plan to include multiple urban study areas reflecting differences in a variety of factors related to $PM_{2.5}$ exposure (e.g., time spent outdoors, air conditioner use, housing stock which can impact $PM_{2.5}$ infiltration, and commuting patterns).

- **Susceptibility to specific populations to $PM_{2.5}$ exposure** (note – this could include a number of factors e.g., magnitude of the effect estimate, underlying health status): We plan to consider this source of variability by using effect estimates and lag structures specific to each urban study location.

- **Differences in baseline incidence of disease**: This source of variability would potentially be captured through the use of localized baseline incidence data (e.g., county-level).

- **Longer-term temporal variability in ambient $PM_{2.5}$ levels** (reflecting meteorological trends, as well as future changes in the mix of $PM_{2.5}$ sources and regulations impacting $PM_{2.5}$): This is more difficult to incorporate into the analysis and reflects a combination of variability as well as uncertainty.

3.3.3 Uncertainty Characterization – Qualitative Assessment

We plan to include a qualitative discussion of uncertainty in the risk assessment which will include: (1) identification and description of key sources of uncertainty, noting whether they are addressed quantitatively in the risk assessment model and (2) a qualitative assessment of those sources of uncertainty in terms of their potential impact on risk using a "high," "medium," and "low" designation. A preliminary list of potentially important sources of uncertainty has been developed for this plan and is presented below (note, some of these sources may be addressed in the quantitative uncertainty analysis, when feasible):

- **Statistical uncertainty associated with the fit of the C-R function**.
- **Shape of the C-R function:** (e.g., shape of the function, including the potential for a population threshold). Of particular concern is uncertainty related to the shape of the C-R function at lower exposure levels where there is less exposure and response data.
- **Potential role of co-pollutants and different lag structures**: these are related to the C-R function (and nature of the associated effects estimate).
- **Transferability of C-R functions from study locations to urban study area locations**: this reflects variation in (a) $PM_{2.5}$ composition, (b) the possible role of copollutants in influencing risk, (c) relationship between ambient $PM_{2.5}$ and actual exposure, and (d) differences in population characteristics. However, it is anticipated that the transferability issue will play less of a role in the upcoming analysis, since studies used to derive C-R functions will often be matched to our urban study area locations. However, there may still be transferability issues arising from changes in these factors between the time period when the C-R functions were estimated and the time period of this risk analysis.
- **Procedures for adjusting air quality to simulate alternate standard levels:** There is uncertainty in developing the method for adjusting current ambient $PM_{2.5}$ levels (at individual monitors used in the risk assessment) to simulate just attaining alternative standard (methods available are likely to include both retrospective empirical monitor-based trend analysis and forward-looking model-based predictions – see section 3.2.1 and section 2.3 for additional detail).
- **The impact of historical air quality on estimates of health risk from long-term $PM_{2.5}$ exposures** (i.e., the amount of time that a population experiences new lower ambient $PM_{2.5}$ levels before there is a noticeable reduction in health effect incidence): Some studies of long-term mortality provide effect estimates differentiated by consecutive, multi-year time periods (e.g., Pope et al., 2002). These studies may provide insights into this issue and the degree to which it could impact risk estimates (by providing different effect estimates).

- **Estimates of policy-relevant background $PM_{2.5}$ levels in a particular location**. There is uncertainty associated with characterizing PRB for individual locations (see Section 3.2.1 for additional detail).

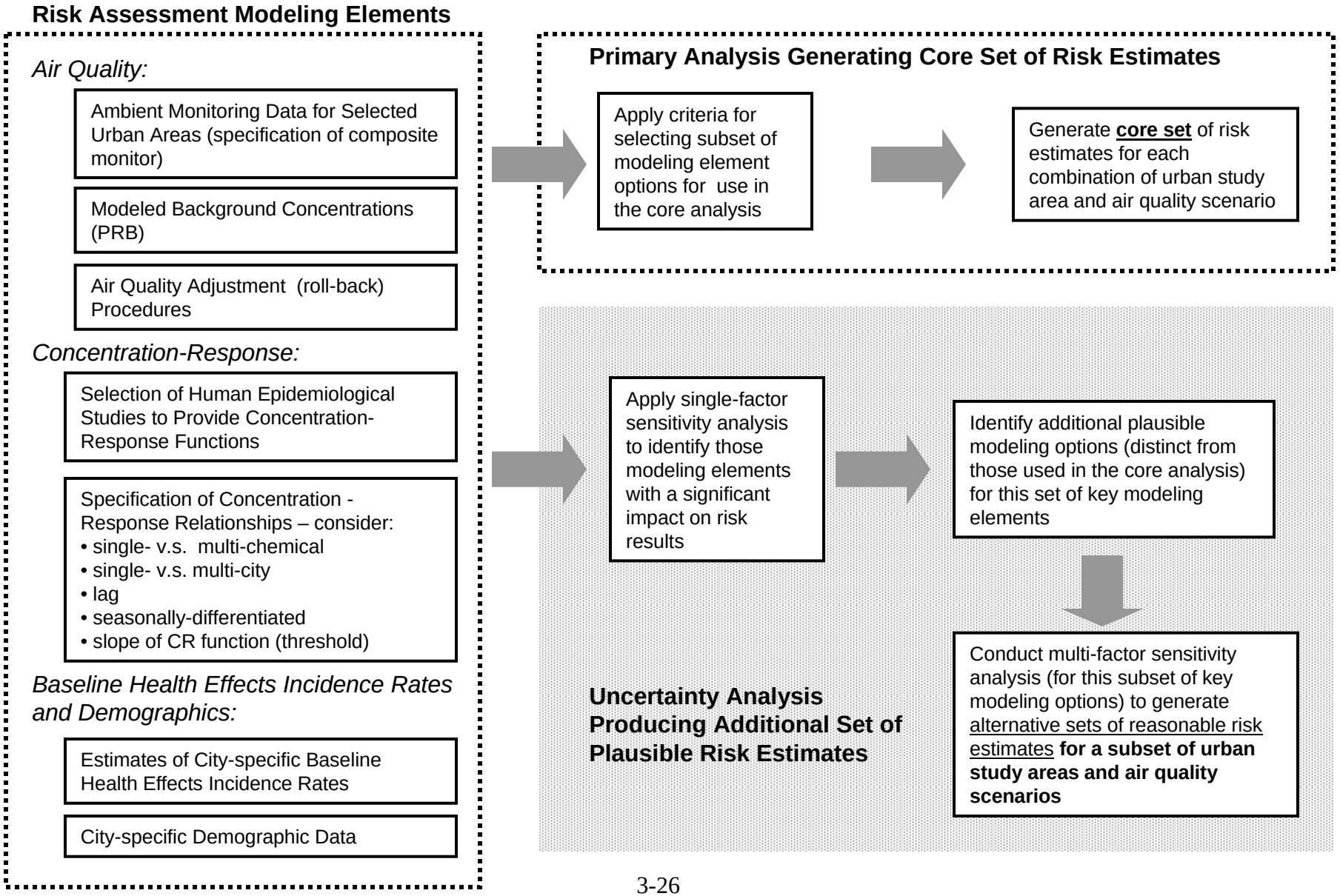
3.3.4 Uncertainty Characterization – Quantitative Analysis

The quantitative uncertainty analysis planed for the $PM_{2.5}$ risk assessment will utilize a deterministic sensitivity analysis-based approach, designed to provide the decision maker with a reasonable alternative set of risk estimates to supplement the set of core risk estimates that are generated. This set of additional risk estimates will provide insights into the impact of uncertainty on the initial set of core risk estimates (the deterministic uncertainty approach is illustrated in Figure 3-2).

Ideally, a 2-dimensional probabilistic simulation would be used as the basis for a quantitative analysis of uncertainty, where one dimension of the simulation reflected variability in risk and the other the impact of multiple sources of uncertainty. However, this type of probabilistic simulation requires that we have: (a) clearly-defined uncertainty distributions for inputs to the risk model, (b) any existing correlations between those input parameters clearly defined, and (c) confidence levels assigned to each model for those analysis steps where we have multiple competing modeling options. At this point, we do not have sufficient information to meet these requirements for many of the key modeling elements associated with the risk assessment.

In the absence of these types of data required to support a 2-dimensional probabilistic uncertainty analysis, deterministic sensitivity analysis-based methods can be used to support quantitative characterization of uncertainty. Specifically, alternate modeling options for key modeling elements can be considered (either one at a time, or in combination) to determine the potential impact on risk estimates. The option of considering these factors in combination (i.e., a multi-factor sensitivity analysis) can be used to derive a set of alternative plausible risk estimates which provides insights into the impact of uncertainty on risk estimates. This application of multi-factor sensitivity analysis forms the basis for the deterministic uncertainty analysis approach developed for this risk assessment.

Figure 3-2. Overview of Uncertainty Analysis Approach Developed for the PM NAAQS Risk Assessment



The step-wise procedure for conducting the deterministic uncertainty analysis is illustrated in Figure 3-2. It is important to point out that we plan to generate a core set of risk estimates prior to conducting the uncertainty analysis. This core set of risk estimates would be derived by first applying the criteria discussed in preceding sections (sections 3.2.1 through 3.2.5) to identify those options for key modeling elements which have the strongest scientific support (with these determinations being based primarily on the evaluation provided in the ISA).[15] The core set of risk estimates will be generated for each combination of urban study area and air quality scenario.

Once the core set of risk estimates has been generated, the uncertainty analysis will begin with a single-factor sensitivity analysis intended to identify those modeling elements (comprising the $PM_{2.5}$ risk assessment framework) that have the potential to significantly impact risk estimates. This set of key modeling elements would form the basis for the uncertainty analysis. Next, plausible modeling options (distinct from those used in the core analysis) would be specified for each of these key modeling elements. In identifying these plausible modeling options, we plan to place emphasis on identifying input factors or modeling approaches which, while representing alternatives to those used in the core simulation, still have some degree of scientific support in the literature. Consequently, while we may have less confidence in risk estimates generated using these alternate modeling options relative to the core risk estimates, they could still considered reasonable and consequently may be interpreted as providing additional perspective on overall uncertainty associated with the core set of risk estimates.

Once the set of plausible modeling options is specified for the key modeling elements, we plan to use a multi-factor sensitivity analysis to generate a set of reasonable alternative risk estimates. Specifically, various combinations of these alternative modeling options would be

[15] For example, as noted in section 3.2.3, if a study provides both single-day and distributed lag models, the distributed lag model would be used in the core analysis, while the individual day lags, if retained in the risk assessment, would be included in the uncertainty analysis. With regard to non-linearity in functions, including the potential for thresholds, generally non-threshold models will be used in the core analysis (based on information provided in the ISA) and thresholds models, if they are considered at all, would be reserved for the uncertainty analysis. It is also important to point out that for some of the modeling elements, multiple options may be included as part of the core simulation (e.g., both multi- and single-component models may be used in core simulations for specific health endpoints).

used to generate risk estimates, each representing an uncertainty simulation.[16] We plan to generate this set of alternative risk estimates for a subset of the urban study areas and air quality scenarios.

The combined sets of core results and alternative risk estimates (for a combination of urban study area and air quality scenario) could be interpreted as representing an initial characterization of risk for that combination of urban study area and air quality scenario, reflecting recognized sources of uncertainty in risk modeling. However, this interpretation needs to be tempered by consideration of several factors: (a) this does not represent a characterization of a distribution of uncertainty around the core set of risk estimates, it merely represents several point estimates likely falling within that uncertainty distribution and (b) the set of modeled risk estimates may not contain actual upper-bound and lower-bound risk estimates given scientifically defensible modeling options. Despite these caveats, the risk estimates defined by the sets of core and alternative risk estimates should be useful to characterize confidence associated with the results of the application of the $PM_{2.5}$ risk assessment model.

3.4 PRESENTATION OF RISK ESTIMATES TO INFORM CONSIDERATION OF STANDARDS

This section discusses the nature of the risk estimates that we plan to generate as part of the review of the PM NAAQS. We plan to conduct the risk assessment in two phases. Phase 1 would include analysis of risk associated with recent air quality and simulating air quality to just meet the current NAAQS. Phase 2 would focus on evaluating risk associated with simulating air quality that just meets alternative NAAQS under consideration.

We plan to present risk estimates in two ways: (1) <u>total (absolute) health effects incidence (above PRB)</u> for recent air quality and simulations of air quality just meeting the current and alternative NAAQS under consideration, and (2) <u>risk reduction estimates,</u> reflecting the difference between (a) risks associated with recent air quality compared to risks associated with just meeting the current NAAQS and (b) reflecting the difference between risks associated

[16] Note, that care would be taken in linking these modeling options together to insure that they are compatible and do not represent combinations that are scientifically not defensible.

with just meeting the current compared to risks associated with just meeting alternative NAAQS under consideration.

In presenting risk estimates, we plan to emphasize the core (base-case) estimates given that these would include risk estimates with greater overall confidence. We plan to also present additional risk estimates generated as part of the uncertainty analyses in order to provide additional context for understanding the potential impact of uncertainty on the risk estimates and particularly on the core estimates of risk.

To further support interpretation of risk estimates generated in this analysis, we may also consider the urban study areas with respect to the degree to which they represent the range of key $PM_{2.5}$ risk-related attributes that are spatially differentiated across the nation. In general, we plan to consider the degree to which the urban study areas provide coverage for different regions of the country, defined by similarities in $PM_{2.5}$-related parameters. Alternatively, we are consideration a more detailed analysis that would evaluate a set of spatially-distributed $PM_{2.5}$-related parameters (e.g., $PM_{2.5}$ composition, air-conditioning use, demographics including socio-economic status (SES), baseline health incidence rates). This analysis would allow us to determine whether the selected urban study areas reflect national-level variability in these key $PM_{2.5}$-related parameters, or whether they are more concentrated in terms of their coverage. Based on generally available data, e.g. from the 2000 Census, CDC, or other sources, distributions for risk-related parameters across U.S. counties would be generated. The specific values of these parameters for the selected urban study areas would then be plotted on these distributions, and an evaluation of how representative the selected study areas are of the individual parameters, relative to the national distributions, could be done.

The specific choices of parameters for which we would examine the representativeness of the selected urban study areas would be informed through an assessment of the epidemiology literature. We plan to particularly focus on meta-analyses and multi-city studies which have identified parameters that influence heterogeneity in $PM_{2.5}$ effect estimates, and exposure studies which have explored determinants of differences in personal exposures to ambient $PM_{2.5}$. While personal exposure is not generally incorporated directly into epidemiology studies evaluating ambient $PM_{2.5}$-related effects, differences in the $PM_{2.5}$ effect estimates between cities clearly is

impacted by differing levels of those exposure determinants. Once we have identified these parameters, we plan to develop national distributions for those parameters (or reasonable surrogates) based on readily available data sources. Formal comparisons of parameter distributions for the set of urban study areas and the national parameter distributions would be conducted using standard statistical tests, e.g. the Kolmogorov-Smirnov non-parametric test for equality of distributions. In addition, we plan to consider visual comparisons using probability density functions, cumulative distribution functions, and boxplots.

3.5 NATIONAL SCALE HEALTH IMPACT ASSESSMENT FOR LONG-TERM EXPOSURE MORTALITY RELATED TO $PM_{2.5}$ EXPOSURES

While the risk analysis focused on selected urban study areas is designed to provide information on risks in specific areas with a range of attributes reflecting different characteristics of susceptibility and vulnerability, this assessment would not provide a sense of the overall magnitude of the public health burden imposed by recent levels of ambient $PM_{2.5}$. As noted earlier, to address this broader question, we are considering conducting a national assessment of the mortality impacts in the U.S. population associated with long-term exposures to ambient $PM_{2.5}$. This estimate could provide context for the magnitude of the long-term $PM_{2.5}$ exposure-related mortality estimated in the risk analysis for the selected urban study areas, as well as some indication of the significance of the public health impacts associated with recent ambient $PM_{2.5}$ levels. We are not considering an evaluation of all health impacts in this national assessment, but rather, we would focus our analyses on mortality associated with long-term $PM_{2.5}$ exposures because of the significance of this endpoint and because of the strength of the available C-R information.[17] In addition, we would not consider it appropriate to compare the results of this assessment to the results from the selected urban area case studies, due to differences in the methods used to estimate air quality concentrations.

We are considering using EPA's peer-reviewed environmental Benefits Mapping Analysis Program (BenMAP; Abt Associates, 2008) to estimate the total incidence of premature

[17] Modeling of short-term (i.e., 24-hour average) $PM_{2.5}$- related health impacts is subject to greater uncertainty than modeling of long-term exposure-related mortality, reflecting the requirement (in modeling short-term endpoints) to extrapolate city-specific studies to other locations in order to generate national-scale estimates. Due to the greater

mortality associated with recent ambient $PM_{2.5}$ concentrations. Similar to the urban study areas analyses, the health impact functions for long-term exposure mortality require three elements: (1) the effect estimate relating ambient $PM_{2.5}$ concentrations to mortality, obtained from the epidemiology literature, (2) baseline incidence of mortality, and (3) annual average ambient $PM_{2.5}$ concentrations.

In the national-scale mortality impact analysis, we would consider using the same C-R function(s) for mortality associated with long-term $PM_{2.5}$ exposures as we plan to use in modeling the selected urban study areas (e.g., those based on national cohort studies such as the American Cancer Society study (Pope et al., 2004), and the Harvard Six-city Study (Laden et al., 2006)). We would also obtain baseline incidence of mortality from the same sources as the urban study areas risk assessment (i.e., the CDC Wonder database).

The national-scale mortality assessment would differ from the urban study areas analyses in the methodology used to estimate ambient $PM_{2.5}$. We would use a data fusion approach, which combines monitored ambient $PM_{2.5}$ concentrations with modeled $PM_{2.5}$ concentrations based on the Community Model for Air Quality (CMAQ). The data fusion approach is known as enhanced Voronoi Neighbor Averaging (eVNA), which is essentially an inverse distance weighted interpolation of monitored $PM_{2.5}$ values, scaled to reflect the ratios of monitored to modeled $PM_{2.5}$ values. We would calculate ambient $PM_{2.5}$ concentrations at a 12 km grid resolution throughout the continental U.S. A modeled ambient $PM_{2.5}$ field would be generated based on recent U.S. conditions for a single year (e.g., 2005). This analysis would estimate the health impact associated with ambient $PM_{2.5}$ above the lowest observed annual average levels measured in the cohort studies, recognizing that there is more empirical support for the C-R function in the range above the lowest measured level for the time period examined in the epidemiological studies.

There are a number of important uncertainties that enter into the national scale assessment beyond those identified for the urban study areas assessment. These include uncertainties regarding the estimation of $PM_{2.5}$ air quality in areas away from monitors, which

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degree of uncertainty associated with modeling short-term endpoints, we have decided to focus this national-scale analysis on long-term exposure-related mortality.

include both interpolation uncertainties and uncertainties in the CMAQ modeling (which include uncertainties in both the CMAQ modeling structure, and in the inputs to the CMAQ modeling, e.g., meteorology and emissions). Also, adding to the uncertainty is the application of the C-R functions based on a limited number of urban areas to populations throughout the U.S. We would need to include a qualitative discussion of key uncertainties in the assessment. This qualitative assessment would, in part, be informed by the characterization of uncertainties provided by the expert elicitation addressing $PM_{2.5}$-related mortality (Roman et al., 2008).

Results from the national scale mortality impact assessment described above would include national incidence estimates, as well as percent of total incidence attributed to exposure to $PM_{2.5}$. Analyses would include results based on multiple C-R functions. Results would also include a limited quantitative uncertainty assessment based on the statistical error reported in the epidemiological studies.

In addition to providing an overall estimate of the mortality impact associated with recent long-term ambient $PM_{2.5}$ concentrations, the national scale mortality impact assessment would estimate the cumulative distribution of mortality risk from long-term $PM_{2.5}$ concentrations across the U.S. population. This would allow us to assess the degree to which our selected urban study areas analysis characterizes long-term mortality risk for the more vulnerable and susceptible populations in the U.S. In other words, do the urban study area risk estimates include the upper end of the national risk distribution, or are they more representative of a different part of the distribution? This analysis would not involve a direct comparison of risk estimates for the urban study areas compared with risk levels generated for those same urban areas within the national-scale analysis, but rather would focus on comparisons of the urban area risk estimates with percentiles of the national risk distribution.

3.6 OVERVIEW OF HEALTH RISK ASSESSMENT APPROACH DEVELOPED FOR $PM_{10-2.5}$

As noted in Section 3.1, we plan to build on the risk assessment completed in the last PM NAAQS review to conduct a risk assessment for $PM_{10-2.5}$. While the first draft ISA does not identify any of the health effect endpoint categories evaluated for $PM_{10-2.5}$ to have sufficient support to be classified as causal or likely causal, we believe that the available evidence is

sufficiently suggestive of a causal association to support a limited risk assessment for some health endpoints related to short-term (daily) exposures to $PM_{10-2.5}$. The prior $PM_{10-2.5}$ risk assessment included hospital admissions for cardiovascular and/or respiratory causes in two locations (Detroit and Seattle) and respiratory symptoms in St. Louis. For the current review, we plan to review the scientific evidence for the health endpoints included in the prior risk assessment and to consider whether there are additional C-R relationships and locations that should be included in a $PM_{10-2.5}$ risk assessment for these health outcomes. Further, we also plan to consider whether there is sufficient information to warrant inclusion of an expanded set of health endpoints such as additional respiratory morbidity outcomes (e.g., asthma-related outpatient and emergency department visits and hospitalization) as well as mortality. Our judgments about which endpoints and locations to include in the risk assessment will be informed by the continuing scientific assessment as well as comments provided by the CASAC panel and general public. A provisional set of studies that we would consider in identifying health effects endpoints to potentially model for $PM_{10-2.5}$ (based on information provided in the first draft ISA) is provided in Table 3-3.

We recognize that there will be significantly greater uncertainties associated with $PM_{10-2.5}$ risk estimates relative to the planned $PM_{2.5}$ risk assessment due to a number of factors including: (1) much greater uncertainty in whether an observed association reflects a causal relationship for $PM_{10-2.5}$ and various health outcomes; (2) more limited $PM_{10-2.5}$ ambient air quality data which often is obtained by calculation from measurements for co-located PM_{10} and $PM_{2.5}$ monitors; (3) far fewer health studies and locations with more mixed effect estimates for $PM_{10-2.5}$; and (4) greater concern about exposure measurement error for $PM_{10-2.5}$ compared to $PM_{2.5}$ in large part due to the greater spatial variability in ambient $PM_{10-2.5}$ concentrations and exposures.

The basic approach we plan to use to estimate risks for $PM_{10-2.5}$ is identical to the approach used for $PM_{2.5}$ as illustrated in Figure 3-1. In order to estimate the incidence of a particular health effect associated with recent conditions in a specific county or set of counties attributable to $PM_{10-2.5}$ daily exposures in excess of PRB, as well as the change in incidence of the health effect in that county or set of counties corresponding to a given change in

Table 3-3 Provisional List of Studies to be Considered for $PM_{10-2.5}$ Risk Assessment: Cardiovascular and Respiratory Morbidity Associated with Short-term $PM_{10-2.5}$ Exposure

Urban Location	Cardiovascular Hospital Admissions	Respiratory Hospital Admissions (and Emergency Room Visits*)	Respiratory Symptoms
Detroit, MI	Lippmann et al. (2000)[1] Congestive heart disease, Ischemic heart disease Dysrhythmias	Lippmann et al. (2000)[1] Pneumonia, COPD+	
Seattle, WA		Sheppard et al. (1999)[2] Asthma	
St. Louis, MO			Schwartz and Neas (2000) Lower respiratory symptoms
Spokane, WA		Slaughter et al. (2005)* All respiratory Acute asthma COPD	
Atlanta, GA		Peel et al. (2005)* All respiratory Upper respiratory infection Asthma Pneumonia COPD	
108 U.S. Counties		Peng et al. (2008) Cardiovascular disease Peripheral vascular disease	

[1] Reanalyzed in Ito (2003); COPD+ is indicated here because the authors included asthma in their definition of COPD. [2] Reanalyzed in Sheppard (2003).

ambient $PM_{10-2.5}$ levels resulting from just meeting a specified set of alternative $PM_{10-2.5}$ standards under consideration, the following three elements are required:

- Air quality information including: (1) recent air quality data for $PM_{10-2.5}$ from ambient monitors for the selected locations, (2) estimates of PRB appropriate for these locations, and (3) a method for adjusting the recent data to reflect the patterns of air quality estimated to occur when an area just meets a alternative $PM_{10-2.5}$ standards under consideration;

- Relative risk-based C-R functions (preferably derived from epidemiological studies conducted in the assessment location) which provide an estimate of the relationship between the health endpoints of interest and ambient $PM_{10-2.5}$ concentrations;

Annual or seasonal baseline health effects incidence rates and population data are needed to provide an estimate of the annual or seasonal baseline incidence of health effects in an area before any changes related to $PM_{10-2.5}$ air quality could be considered.

4 SCOPE AND APPROACH FOR POPULATION EXPOSURE ANALYSIS

4.1 INTRODUCTION

As part of the last PM NAAQS review, EPA did not conduct an exposure assessment. There is much new scientific information available since the last review on human health effects, exposure, and PM air quality, which provides the basis for conducting a quantitative exposure assessment in this review. This assessment is planned to focus on evaluating fine particle exposures ($PM_{2.5}$) and will build upon the information presented in the ISA. This assessment will include discussions of factors that affect exposure to ambient $PM_{2.5}$ and the use of fixed site measurements of ambient $PM_{2.5}$ concentrations as a surrogate for population (or community) average exposure to ambient PM in epidemiologic studies. There are two specific purposes that such an assessment could serve: (1) providing insight on population exposures with respect to informing the interpretation of available epidemiologic studies; and (2) assessing population exposures above benchmark levels of concern, and providing input to quantitative risk assessments based on evidence from clinical studies. At this time, based upon the first draft ISA (U.S., EPA, 2008b), we are unaware of any results from human clinical studies that would provide the basis for exposure-response functions that could inform a quantitative risk assessment or benchmark levels of concern; therefore, the planned assessment focuses on the first purpose. The available monitoring data for $PM_{10-2.5}$ are much more limited than ambient monitoring data available for $PM_{2.5}$ and do not provide enough spatial coverage for exposure modeling to be credible. Therefore, we do not plan on conducting an exposure assessment for thoracic coarse particles ($PM_{10-2.5}$).

Performing an exposure analysis will be helpful for identifying the various personal and building-related factors which may be responsible for some of the differences observed in epidemiologic studies of ambient $PM_{2.5}$. Exposure-related factors may contribute to city-to-city differences (mostly seen in time-series studies) in the reported $PM_{2.5}$ concentration-response (C-R) functions or in the results from intra-urban studies (e.g., cohort studies of long-term

exposures to $PM_{2.5}$). Thus, an exposure assessment for $PM_{2.5}$ has the potential to shed some light on these and other issues related to uncertainties in the existing $PM_{2.5}$ epidemiology literature.

We plan to model population exposures to ambient $PM_{2.5}$ in approximately 10 urban areas across the U.S. where epidemiological studies have been conducted that are the basis of C-R functions used in the planned quantitative risk assessment described in Chapter 3. The basis for the selection of areas to model is described in section 4.4 below. The exposure periods we plan to model will be the periods studied in the corresponding epidemiologic studies.

The primary measures of interest are estimated population distributions of 24-hour average exposures. Additional quantitative measures of exposure may take into account factors including the magnitude and duration of $PM_{2.5}$ exposures and the frequency of repeated peak exposures. We plan to develop estimates for population exposures associated with historical $PM_{2.5}$ levels only. We do not plan to estimate population exposures for scenarios that simulate just meeting the current $PM_{2.5}$ standards or potential alternative primary $PM_{2.5}$ standards under consideration, as these are not directly relevant to interpreting currently available epidemiologic studies based on past environmental conditions.

4.2 THE APEX POPULATION EXPOSURE MODEL

As stated in the Integrated Review Plan (U.S. EPA, 2008a, section 5.4), EPA considered using the Air Pollutants Exposure (APEX) model (Richmond et al., 2002; U.S. EPA, 2008 e,f) and/or the Stochastic Human Exposure and Dose Simulation model for PM (SHEDS-PM) (Burke and Vedantham, 2005) in conducting an exposure assessment for this review. [18] APEX, also referred to as the Total Risk Integrated Methodology/Exposure (TRIM.Expo) model, has its origins in the NAAQS Exposure Model (NEM), which was developed in the early 1980's (Biller

[18] A third model considered for this application is the MENTOR-1A (Modeling ENvironment for TOtal Risk studies in a "One Atmosphere" setting) model. MENTOR-1A includes both gas and aerosol phase microenvironmental processes (Georgopoulos & Lioy, 2006; Georgopoulos et al., 2005). MENTOR-1A also includes optional selection of Models-3/CMAQ compatible modules for microenvironmental transformations, both physical (e.g. condensation to form secondary aerosol) and chemical (with CB4 and RADM2 based alternative mechanisms). MENTOR-1A performs exposure calculations for multiple pollutants and allows for direct linkage to compute the uptakes, and links with physiologically based toxicokinetic models (PBTK) for assessing distributions of target tissue doses for populations, which can be used for evaluation with population-based biomarker studies. Since the underlying exposure model of MENTOR-1A is similar to SHEDS-PM and the additional complexity involving chemistry and dose are not required for this application, this model was not selected for use.

et al., 1981; McCurdy, 1994, 1995). SHEDS-PM was developed by EPA/ORD's National Exposure Research Laboratory (NERL) (Burke et al., 2001; Özkaynak et al 2009). APEX and SHEDS-PM are Monte Carlo simulation models that simulate a large number of randomly sampled individuals within a metropolitan area to represent area-wide population exposures. Although these models were developed independently, they are both based on the current state of knowledge of inhalation exposure modeling and are fundamentally similar. Both models simulate the movements of individuals through time and space and their exposure to a given pollutant in indoor, outdoor, and in-vehicle microenvironments. The models stochastically generate simulated individuals using census-derived probability distributions for demographic characteristics. A large number of simulated individuals are modeled and collectively they represent a random sample of the study area population. Research conducted by NERL has been used in the development of the APEX model in a number of areas including: incorporation of the excess post-exercise oxygen consumption model (McCurdy, 2000; Isaacs et al., 2008); inclusion of an improved method for constructing longitudinal diaries (Glen et al., 2008); continuation of updates to the Consolidated Human Activity Database (CHAD) (McCurdy et al., 2000); and incorporation of a model for calculating deposition of PM to lungs.

EPA staff has considered the features of these two models for the specific purpose of conducting an exposure assessment to inform this review of the PM NAAQS and has concluded that APEX is the most appropriate model for this particular regulatory application. The rationale for selecting the APEX model includes consideration of the following factors:

- APEX is available to the public with non-proprietary code and documentation, whereas, at this time, SHEDS-PM is not. This allows interested members of the public access to the model and, if desired, the ability to recreate the technical analyses supporting the NAAQS review. Transparency and availability of the methods and data used in the technical analyses is an integral part of the NAAQS review process and encouraged by the provisions of the Information Quality Act.

- APEX was designed to allow users the ability to model any airborne pollutant. This model has been primarily used for regulatory purposes, with significant applications to inform the recent reviews of the ozone, sulfur dioxide, and nitrogen dioxide NAAQS. Periodic reviews by CASAC and the public and application of APEX in

other NAAQS reviews have resulted in an ongoing and chronologically documented process of refinement of the model.

- APEX can be run using a batch mode, which is particularly important for this application, to allow for the large number of simulations that are performed for NAAQS assessments.

- Quantitative assessment of uncertainty and variability, a critical component of this analysis, is integrated into SHEDS-PM so that a single model run yields both variability and uncertainty results (Burke et al., 2001). In order to analyze uncertainty with APEX, we plan to use external software that modifies APEX inputs according to distributions reflecting uncertainty and runs APEX numerous times for the outer uncertainty loop (see section 4.8). This would result in a 2-dimensional Monte Carlo treatment that tracks variability and uncertainty separately. While this approach requires more effort than an uncertainty analysis with SHEDS-PM, the flexibility of this method is deemed important for performing the planned assessment of uncertainty.

APEX simulates the movement of individuals through time and space and their exposure to a given pollutant in indoor, outdoor, and in-vehicle microenvironments. Figure 4-1 provides a schematic overview of the APEX model. The model stochastically generates simulated individuals using census-derived probability distributions for demographic characteristics (Figure 4-1, steps 1-3). The population demographics are from the 2000 Census data at the tract or block level, and a national commuting database based on 2000 Census data provides home-to-work commuting flows between tracts. A large number of simulated individuals are modeled, and collectively, they represent a random sample of the study area population.

Diary-derived time activity data are used to construct a sequence of activity events for each simulated individual consistent with the individual's demographic characteristics and accounting for effects of day type (e.g., weekday, weekend) and outdoor temperature on daily activities (Figure 4-1, step 4). APEX calculates the concentration in the microenvironment associated with each event in an individual's activity pattern and sums the event-specific exposures within each hour to obtain a continuous time series of hourly exposures spanning the time period of interest (Figure 4-1, steps 5 and 6). From these exposure estimates, APEX calculates exposures for averaging times greater than one hour.

Figure 4-1. Overview of the APEX Model

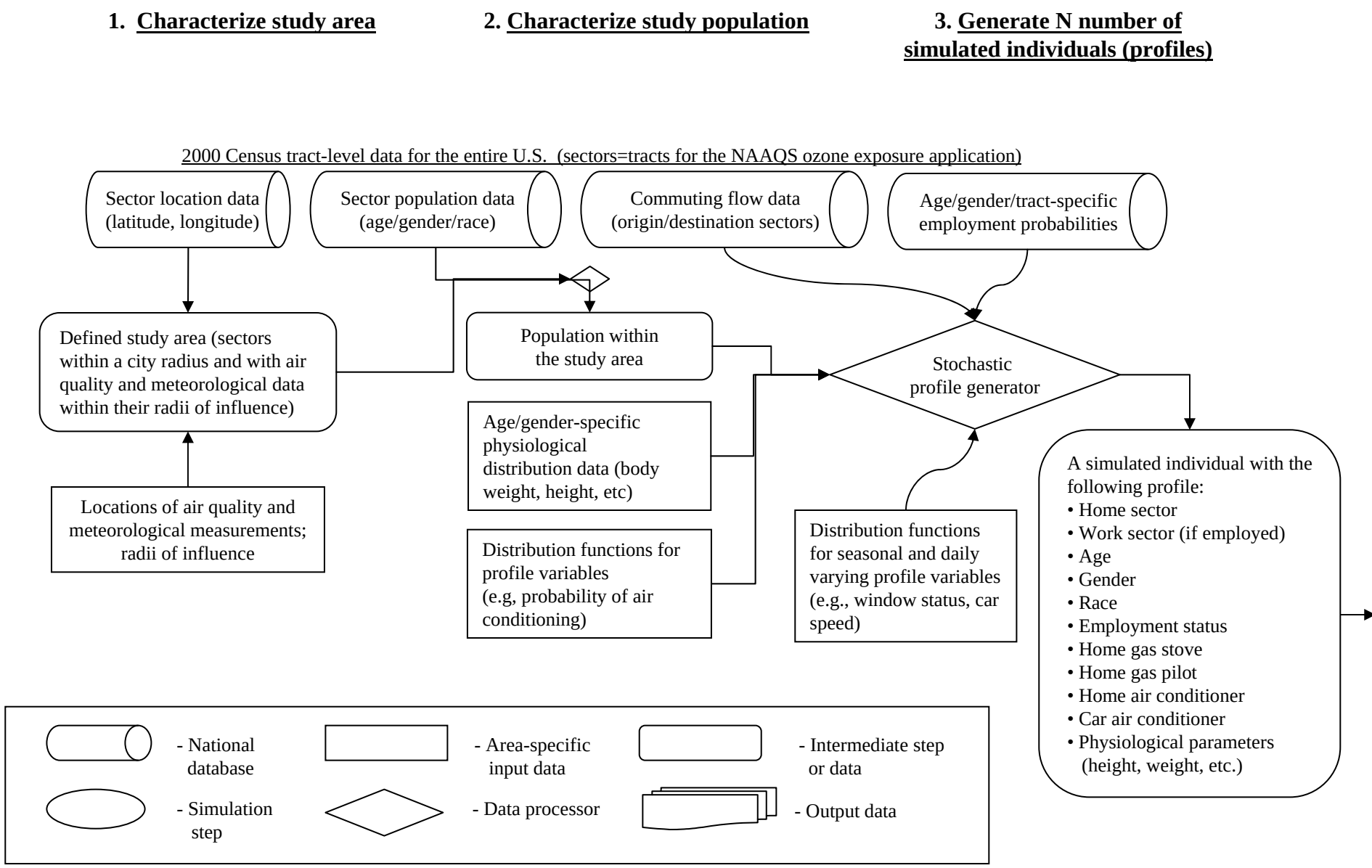


Figure 4-1. Overview of the APEX Model, continued

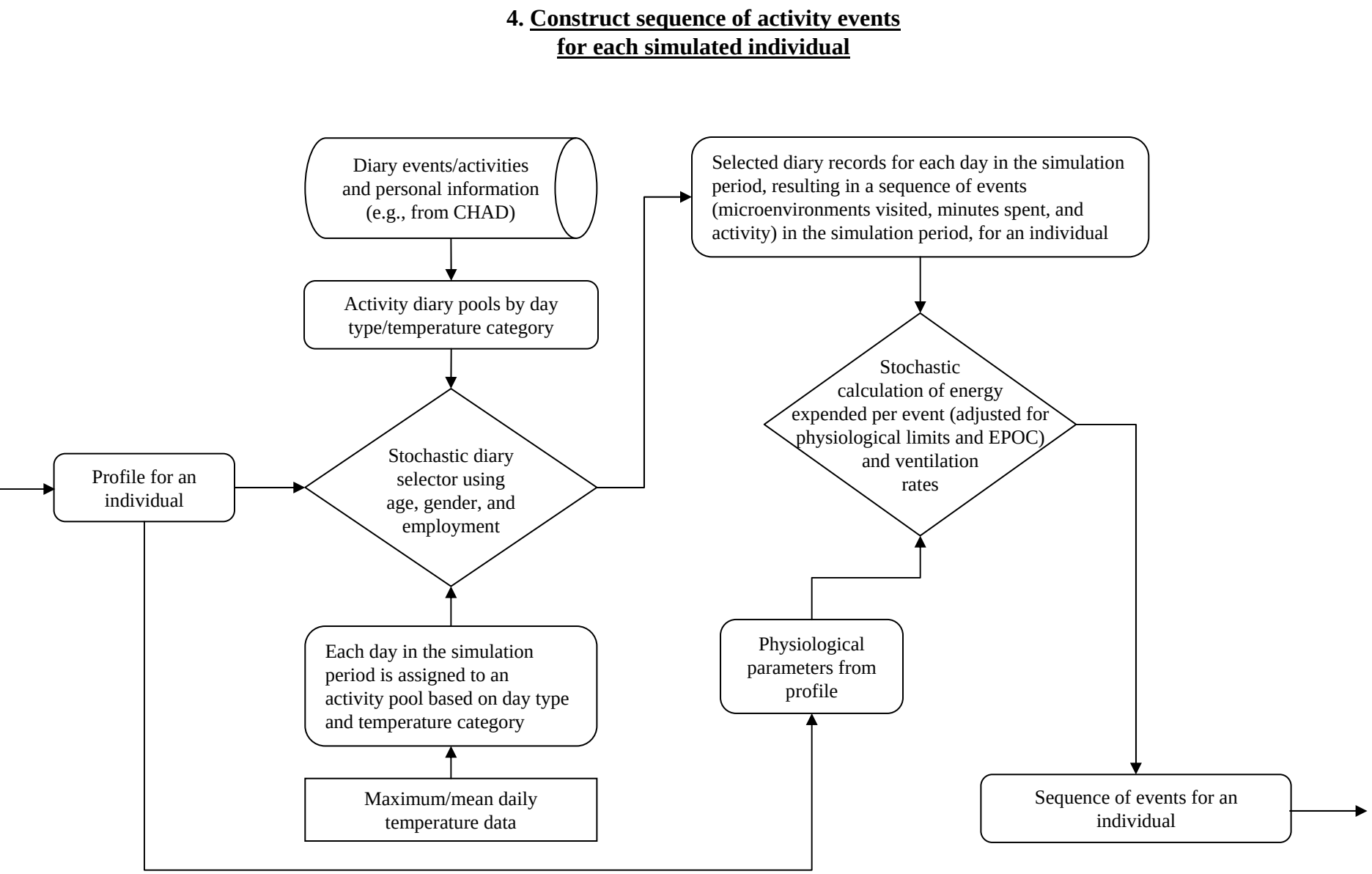
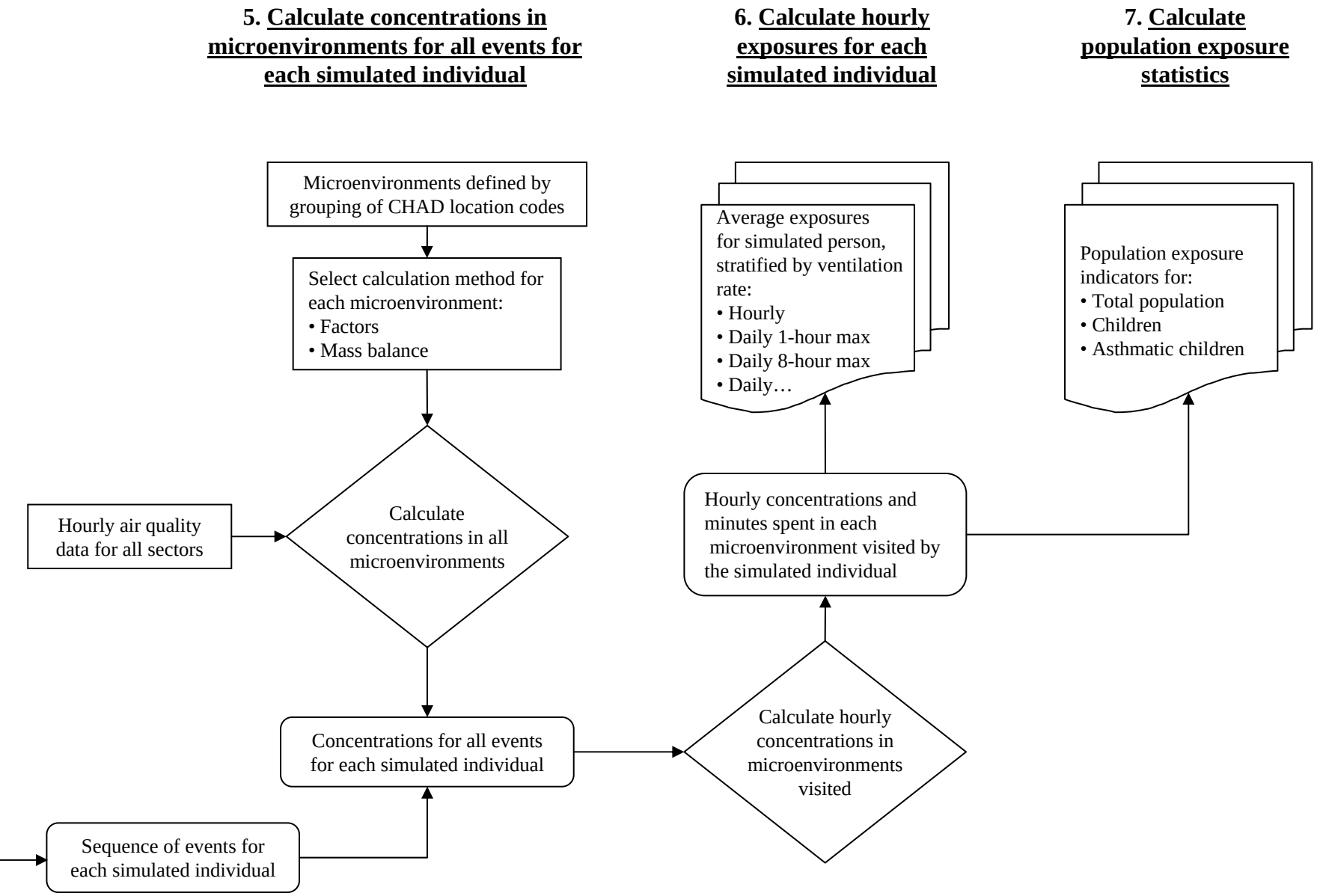


Figure 4-1. Overview of the APEX Model, concluded



APEX employs a flexible approach for simulating microenvironmental concentrations, where the user can define any number of microenvironments to be modeled and their characteristics. For this modeling application, we propose modeling the microenvironments listed in Table 4-1.

Table 4-1 Microenvironments to be Modeled

Microenvironment	Method
Indoors – residence	mass balance
Indoors – restaurants, bars	mass balance
Indoors – schools	mass balance
Indoors – day care centers (commercial)	mass balance
Indoors – offices	mass balance
Indoors – shopping malls	mass balance or factors
Indoors – other (e.g., stores not in malls)	mass balance or factors
Outdoors – bus stop	factors
Outdoors – near road	factors
Outdoors – other (e.g., playgrounds, parks)	factors
In vehicle – cars and light trucks	mass balance or factors
In vehicle – heavy trucks	mass balance or factors
In vehicle – school buses	mass balance or factors
In vehicle – mass transit vehicles – buses and trolleys	factors
In vehicle – mass transit vehicles – underground (subways)	factors

We plan to calculate the concentrations in each microenvironment using either a factors or mass-balance approach[19], depending upon data availability, with probability distributions for the parameters that enter into the calculations (e.g., indoor-outdoor air exchange rates) supplied as inputs to the model. These distributions represent the variability of parameters, and can vary spatially and can be set up to depend on the values of other variables in the model. For example, the distribution of air exchange rates in a home, office, or car depends on the ambient temperature and the type of heating and air conditioning present. The user can choose to keep the value of a stochastic parameter constant for an individual for the entire simulation (e.g.,

[19] The factors and mass-balance approaches are described in section 4.5.5.

house volume), or can specify that a new value shall be drawn hourly, daily, or seasonally from specified distributions. APEX also allows the user to specify diurnal, weekly, and seasonal patterns for microenvironmental parameters.

4.3 MEASURES OF EXPOSURE

EPA plans to estimate distributions of 24-hour average exposures using APEX for population groups represented in epidemiologic studies. We are considering whether shorter averaging times would also be informative for differentiating exposure characteristics in different cities. We may consider additional indicators of exposure that incorporate factors such as relationships between magnitude and duration of exposures, frequency of repeated high exposures, and ventilation rate (i.e., breathing rate) of the individual at the time of exposure. In addition to these measures, we plan to summarize distributions of personal exposure factors, which we define as ratios of personal exposures to ambient concentrations. To support interpretation of multi-city epidemiologic studies, we plan to conduct an analysis of the dependence of these distributions and other exposure measures on city-specific attributes.

4.4 SELECTION OF URBAN AREAS AND TIME PERIODS

EPA plans to conduct exposure analyses for about 10 metropolitan areas that have been considered in multi-city epidemiologic studies. We plan to use multi-city studies instead of individual city studies because some sources of inter-city heterogeneity may be avoided using these data, such as the use of different statistical methods, different treatment of confounders, the use of different types of data, and publication bias. The time periods and geographic areas to be modeled will coincide with those of the corresponding epidemiologic studies conducted in these areas. The selection of urban study areas to include in the exposure analysis will also take into consideration the availability of ambient $PM_{2.5}$ data, and the desire to represent a range of geographic areas, population demographics, $PM_{2.5}$ climatology, $PM_{2.5}$ species and air pollution composition, and other variables that could account for variation in population exposures between cities.

4.5 DEVELOPMENT OF MODEL INPUTS

4.5.1 Population Demographics

We plan to obtain tract-level population counts from the 2000 Census of Population and Housing Summary File 120. Summary File 1 (SF 1) contains the 100-percent data, which is the information compiled from the questions asked of all people and about every housing unit.

In the 2000 U.S. Census, estimates of employment were developed by census tract[21]. The file input to APEX will be broken down by gender and age group, so that each gender/age group combination is given an employment probability fraction (ranging from 0 to 1) within each census tract. The age groupings in this file are: 16-19, 20-21, 22-24, 25-29, 30-34, 35-44, 45-54, 55-59, 60-61, 62-64, 65-69, 70-74, and greater than 75 years of age. Children under 16 years of age will be assumed to be not employed.

4.5.2 Commuting

As part of the population demographics inputs, it is important to integrate working patterns into the assessment. In addition to using estimates of employment by tract, APEX also incorporates home-to-work commuting data. We plan to use the national commuting database provided with APEX in this analysis. Commuting data were derived from the 2000 Census and were collected as part of the Census Transportation Planning Package (CTPP) (U.S. DOT, 2000)[22]. The data used to generate APEX inputs were taken from the "Part 3-The Journey To Work" files. These files contain counts of individuals commuting from home to work locations at a number of geographic scales. These data have been processed to calculate fractions for each tract-to-tract flow to create the national commuting data distributed with APEX. This database contains commuting data for each of the 50 states and Washington, D.C. This data set does not differentiate people that work at home from those that commute within their home tract.

[20] http://www.census.gov/prod/cen2000/doc/sf1.pdf
[21] Employment data from the 2000 Census can be found on the U.S. Census web site: http://www.census.gov/population/www/cen2000/phc-t28.html (Employment Status: 2000- Supplemental Tables).
[22] These data are available from the U.S. DOT Bureau of Transportation Statistics (BTS) at the web site: http://transtats.bts.gov/.

4.5.3 Ambient $PM_{2.5}$ Concentrations

We plan to conduct exposure modeling based on $PM_{2.5}$ concentrations measured at ambient air monitors in and near the areas being modeled. Sources for these data include the daily and hourly concentration measurements from the monitoring data maintained in EPA's Air Quality System (AQS), the Interagency Monitoring of Protected Visual Environments network (IMPROVE, 2008), and the Canadian National Air Pollution Surveillance (NAPS) network (Environment Canada, 2008). We plan to input hourly ambient concentrations into APEX, and allocate the daily data to hourly values according to the diurnal profiles of the hourly monitors. Methods for spatial interpolation and for estimating $PM_{2.5}$ concentrations near roadways are being developed.

4.5.4 Meteorological Data

Surface meteorological observations will be obtained from the National Climatic Data Center[23] to provide hourly temperatures for input to APEX. We plan to use all meteorological stations within and nearby each selected urban study area.

4.5.5 Specification of Microenvironments

Parameters defining each microenvironment will be specified by distributions which reflect the variability of these parameters. The parameters needed depend on whether a microenvironment is modeled using the factors model or the mass balance model.

We plan to use the factors model to model simple environments, like outdoor areas, that do not contain pollutant sources, or microenvironments for which data are not available to use the mass-balance model. Two parameters affect the pollutant concentration calculation in the factors method, the proximity and infiltration factors. The proximity factor ($\boldsymbol{F}_{PR}$) is a unitless parameter that represents the relationship of the ambient concentration outside of the microenvironment ($\boldsymbol{C}_O$) to the concentration at a monitoring station ($\boldsymbol{C}_A$) by the equation $\boldsymbol{C}_O = \boldsymbol{F}_{PR}\,\boldsymbol{C}_A$. The infiltration factor ($\boldsymbol{F}_{inf}$) is a unitless parameter that represents the equilibrium fraction of pollutant entering a microenvironment from outside the microenvironment and

[23] See http://www.ncdc.noaa.gov/oa/ncdc.html

remaining suspended. The concentration inside the microenvironment ($\boldsymbol{C}_I$) is estimated by the equation $\boldsymbol{C}_I = \boldsymbol{F}_{inf}\,\boldsymbol{C}_O$. The infiltration factor in the factors model is often expressed as:

$$\boldsymbol{F}_{\text{inf}} = \frac{\boldsymbol{Pa}}{\boldsymbol{a}+\boldsymbol{k}}$$

where $\boldsymbol{P}$ is a penetration coefficient, $\boldsymbol{a}$ is an air exchange rate, and $\boldsymbol{k}$ is a particle loss rate (ISA, page 3-130). APEX draws values of these parameters from distributions specified by the user, to model the stochastic nature of these factors.

The mass balance model is more appropriate for complex environments. The mass balance method assumes that an enclosed microenvironment (e.g., a room in a residence) is a single well-mixed volume in which the air concentration is approximately spatially uniform. APEX estimates the concentration of an air pollutant in such a microenvironment by using the following four processes (as illustrated in Figure 4-2):

- Inflow of air into the microenvironment;
- Outflow of air from the microenvironment;
- Removal of a pollutant from the microenvironment due to deposition, filtration, and chemical degradation; and
- Emissions from sources of a pollutant inside the microenvironment.

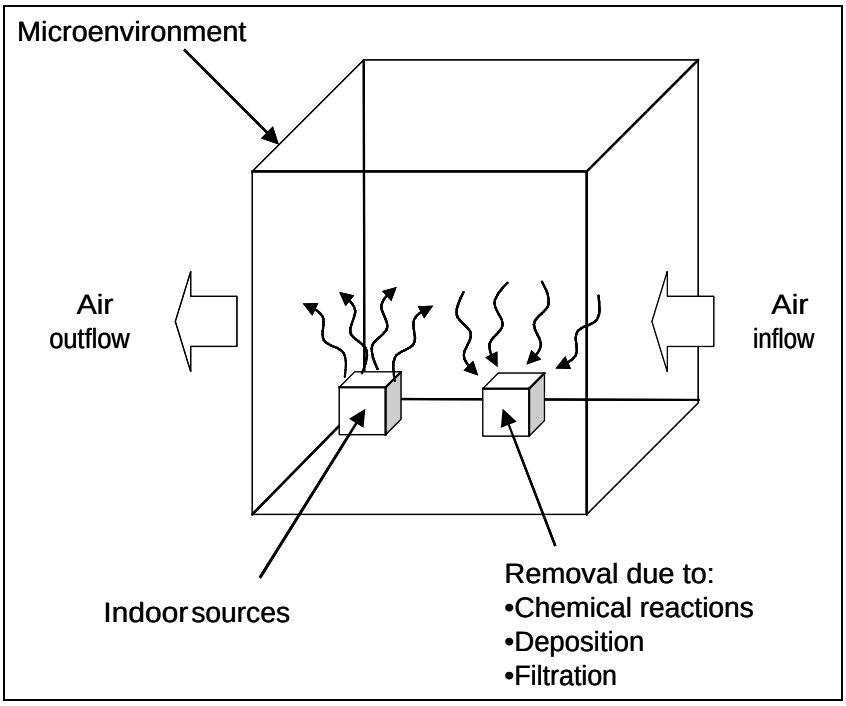


Figure 4-2. The Mass Balance Model

Considering the microenvironment as a distinct, well-mixed volume of air, the mass balance equation for a pollutant can be described by:

$$\frac{dC(t)}{dt} = \frac{dC_{in}(t)}{dt} - \frac{dC_{out}(t)}{dt} - \frac{dC_{loss}(t)}{dt} + \frac{dC_{source}(t)}{dt}$$

where:

$C(t)$ = Concentration in the microenvironment at time t ($\mu g/m^3$)

$\frac{dC_{in}(t)}{dt}$ = Rate of change in C(t) due to air entering the micro

$\frac{dC_{out}(t)}{dt}$ = Rate of change in C(t) due to air leaving the micro

$\frac{dC_{loss}(t)}{dt}$ = Rate of change in C(t) due to all removal processes

$\frac{dC_{source}(t)}{dt}$ = Rate of change in C(t) due to all source terms

In addition to proximity factors, this method supports parameter distributions for time varying emissions sources, decay rate, air exchange rate, volume, and removal rate. We plan to estimate the distributions of these parameters based on available data and a review of the literature.

4.5.6 Indoor Sources

We do not plan to model indoor sources of $PM_{2.5}$ in this analysis, since our focus is on exposure to $PM_{2.5}$ of ambient origin. Differences in the characteristics of indoor sources for different cities may be a factor contributing to inter-city heterogeneity of C-R functions; however, staff feels that there is not sufficient city-specific information in indoor source types and usage to be informative in this analysis, with the possible exception of wood stoves. The formation of ultrafine PM via chemical reactions of air fresheners and ozone (Weschler et al., 2003) is considered an indoor source, and will not be modeled.

The "personal cloud" is the assemblage of particles adjacent to a person that is generated by personal activities such as resuspension of particles from floors, furniture, or clothes, and not by other indoor sources of PM. In order to be able to model this in the framework of a mass-balance model, the personal cloud is defined in terms of processes that generate the PM personal cloud, and not as the difference between predicted and measured personal PM exposures. The extent to which the particles comprising personal clouds are of ambient origin is not known, and will depend heavily on household and behavioral characteristics. Wallace et al. (2006) report estimates from various studies of the indoor personal cloud ranging from 2 – 4 $\mu g/m^3$. It seems reasonable to assume that most of this is not of ambient origin and that the personal cloud is not likely to be a significant contributor to the highest 24-hour or annual average exposures of the population. Therefore, we do not plan to model the personal cloud.

4.5.7 Activity Patterns

Exposure models use human activity pattern data to predict and estimate exposure to pollutants. Different human activities, such as outdoor exercise, indoor reading, or driving, have different pollutant exposure characteristics. In addition, different human activities require

different metabolic rates, and higher rates lead to higher doses. To accurately model individuals and their exposure to pollutants, it is critical to have a firm understanding of their daily activities.

The Consolidated Human Activity Database (CHAD) provides data on human activities through a database system of collected human diaries, or daily activity logs (McCurdy et al., 2000; US EPA, 2002; Graham and McCurdy, 2004). The purpose of CHAD is to provide a basis for conducting multi-route, multi-media exposure assessments (McCurdy et al., 2000). The data contained within CHAD come from multiple surveys with varied structures (Table 4-2). In general, the surveys have a data foundation based on daily diaries of human activity. Individuals filled out diaries of their daily activities and this information was entered and stored in CHAD. Relevant data for these individuals, such as age, are included as well. In addition, CHAD contains activity-specific metabolic distributions developed from literature-derived data, which are used to provide an estimate of metabolic rates of respondents through their various activities.

The locations used in the CHAD diaries must be assigned appropriately to the APEX microenvironments listed in Table 4-1. Each of the microenvironments is designed to simulate an environment in which people spend time during the day. There are many more CHAD locations than microenvironments being modeled (there are over 100 CHAD locations and 15 proposed microenvironments modeled in this assessment) thus, most of the microenvironments have multiple CHAD locations mapped to them.

Table 4-2 Studies in CHAD used in this analysis

Study name	Geographic coverage	Study time period	Subject ages	Diary−days	Number of subjects	Diary type and study design	Reference
Baltimore	One building in Baltimore	1/1997−2/1997, 7/1998−8/1998	72 − 93	292	26	Diary	Williams et al. (2000)
California Adults (CARB)	California	10/1987−9/1988	18 − 94	1,561	1,561	Recall (next day telephone survey); Random	Robinson et al. (1989), Wiley et al. (1991a)
California Children (CARB)	California	04/1989−2/1990	<1 − 11	1,200	1,200	Recall (next day telephone survey); Random	Wiley et al. (1991b)
California Adolescents (CARB)	California	10/1987−9/1988	12 − 17	181	181	Recall (next day telephone survey); Random	Robinson et al. (1989), Wiley et al. (1991a)
Cincinnati (EPRI)	Cincinnati metro. area	3/1985, 8/1985	<1 − 86	2,611	888	Diary; Random	Johnson (1989)
Denver (EPA)	Denver metro. area	11/1982−2/1983	18 − 70	800	444	Diary; Random	Johnson (1984), Akland et al. (1985)
Los Angeles: Elementary School	Los Angeles	10/1989	10 − 12	51	17	Diary	Spier et al. (1992)
Los Angeles: High School	Los Angeles	10/1990	13 − 17	42	19	Diary	Spier et al. (1992)
NHAPS[2]−Air	National	9/1992−10/1994	<1 − 93	4,383	4,383	Recall; Random	Klepeis et al. (1996), Tsang and Klepeis (1996)

NHAPS−Water	National	9/1992−10/1994	<1 − 93	4,392	4,392	Recall; Random	Klepeis et al. (1996), Tsang and Klepeis (1996)
PSID CDS[3] I	National	3/1997−6/1997, 9/1997−12/1997	<1 − 13	4,993	2,703	Diary; Random	Hofferth et al. (1999)
PSID CDS II	National	10/2002−6/2003	5 − 18	4,744	2,503	Diary; Random	Mainieri et al. (2004)
Seattle	Seattle, WA	12/2000−5/2001	6 − 13	1,644	1,639	Diary	Liu et al. (2003)
RTP Panel	Chapel Hill, Raleigh, NC	6/2000−5/2001	55 − 85	871	37	Diary	Williams et al. (2003a,b)
Washington, D.C.	Wash., D.C. metro. area	11/1982−2/1983	18 − 71	689	689	Diary; Random	Hartwell et al. (1984), Akland et al. (1985)
Totals				**28,454**	**20,682**		

NOTE: The counts in this table refer to subsets of the studies for which data can be used in APEX.

[2] National Human Activity Pattern Survey. http://www.exposurescience.org/NHAPS

[3] The Panel Study of Income Dynamics, Child Development Supplement. http://psidonline.isr.umich.edu/

4.6 PILOT EXPOSURE ASSESSMENT

We are conducting $PM_{2.5}$ exposure modeling for one city (Detroit, Michigan) for two months (January and July 2002) to explore ways of approaching some exposure modeling issues before we start on the multi-city, multi-year exposure modeling we plan to conduct for this assessment. This pilot exposure assessment is planned to provide additional information to inform our proposed exposure modeling approach. This urban area and time period were selected for the pilot assessment since air quality modeling results using AERMOD[24], CAMx[25], and CMAQ[26] at a detailed spatial resolution were readily available. At this time, the pilot exposure modeling is not far along enough to give insights into the issues that we are trying to address.[27] In this section, we discuss how we plan to approach this analysis. The preparation of inputs to the exposure model (APEX) for this pilot assessment is described in Appendix A.

Estimation of ambient concentrations between monitoring sites

An evaluation of methods for spatially interpolating concentrations is being conducted using air quality modeling as well as monitoring data, and a method will be selected taking into account the performance of methods and the effort required to apply the methods.

Development of a parametric model for estimation of ambient concentrations at or close to roadways

It is likely that the use of air quality models in conjunction with monitoring data will result in more accurate estimates of ambient concentrations near roadways than interpolation methods relying solely on monitored concentrations, because the air quality models have the potential to be able to resolve near-roadway concentration gradients that the monitors cannot.

Air quality modeling using link-based roadway emissions can be used for estimating ambient concentrations close to roadways (US EPA, 2008c). However, the time and resources required to prepare the link-based emissions inventories and the meteorological model inputs to

[24] AERMOD is an EPA regulatory air quality dispersion model that replaces ISC3. See http://www.epa.gov/scram001/dispersion_prefrec.htm
[25] See http://www.camx.com
[26] See http://www.epa.gov/asmdnerl/CMAQ/cmaq_model.html
[27] To the extent that results are available in advance of the upcoming CASAC consultation meeting on this planning document, we plan to provide a summary of results at that time.

run air quality models for an urban area preclude their use for more than a few cities in the context of this review. We plan to perform air quality modeling for a small number of urban areas and develop a stochastic model for estimating near-road concentrations based on the air quality modeling results. If this modeling effort is successful, this algorithm would be used in exposure modeling for the full set of cities to be modeled.

The dependent variables in this model are the near-road concentrations estimated by the air dispersion modeling. A set of independent variables to be considered is being developed, including block-level Census data and measures of local roadway intensity which incorporate summations of nearby roadways, weighted by a surrogate for traffic volume and inverse distance from roadway. These variables will need to be readily available for large urban areas in the U.S.

Estimation of ambient concentrations for days with no measurements at sites that provide measurements every three or every six days

We will consider a method similar to that used for hourly data (described in Appendix A) as well as methods of spatial interpolation for filling in missing daily concentrations.

Estimation of hourly concentrations from daily monitoring data

One approach to this issue could be to apply the diurnal profiles from nearby hourly monitors to the 24-hour average $PM_{2.5}$ concentrations at a daily monitor (not changing the 24-hour averages). Air quality modeling may be used to estimate hourly concentrations from 24-hour monitoring data by applying the modeled diurnal profile to the 24-hour monitored $PM_{2.5}$ concentrations. Alternatively, we are considering using some combination of air quality modeling and hourly measurements. An analysis of these options is planned

Evaluation of the utility of the refinement of $PM_{2.5}$ into size classes for the microenvironmental mass-balance calculations

We plan to perform exposure model simulations with and without resolution into $PM_{2.5}$ size classes. If we have alternative ways of apportioning $PM_{2.5}$ into size classes, we will consider conducting simulations using this information. We plan to assess these results to see whether or not the refinement of microenvironmental parameters into size classes of $PM_{2.5}$ makes any significant difference in the measures of exposure of interest.

Modeling people's near-road and in-vehicle activity patterns

Near-road and in-vehicle activities are important components of exposure to PM; staff are working to develop an improved methodology for characterizing these activities.

4.7 EXPOSURE MODELING ISSUES

In this section, we highlight some aspects of the proposed exposure modeling that have the potential to significantly contribute to uncertainties in the exposure analysis. These aspects of people's exposures are either not modeled or are based on limited information. The uncertainty analysis will attempt to quantitatively estimate the effect of these aspects on the modeled exposures.

- **Representativeness of Personal Activity Patterns**

The human activity data will be drawn from CHAD. The CHAD includes data from several surveys covering specific time periods at city, state, and national levels, with varying degrees of representativeness. The extent to which the human activity database provides a balanced representation of the population being modeled varies across areas. Although the algorithm that constructs activity sequences attempts to account for the effects of population demographics and local climate on activity, this adjustment procedure does not fully account for all intercity differences in people's activities. Activity patterns are affected by many local factors, including topography, land use, traffic patterns, mass transit systems, and recreational opportunities.

- **Longitudinal Personal Activity Patterns**

The average subject in the CHAD time/activity studies provided less than two days of diary data. For this reason, the construction of a year-long activity sequence for each individual requires some combination of repeating data from one subject and using data from multiple subjects. A key issue in this assessment is the development of an approach for creating year-long activity sequences for individuals based on a cross-sectional activity data base that includes 24-hour records. We believe an appropriate approach should adequately account for the day-to-day and week-to-week repetition of activities common to individuals while maintaining realistic variability between individuals.

- **Averting Behavior**

Behavior changes in response to $PM_{2.5}$ pollution or in response to air quality index (AQI) notification ("averting behavior") is not being taken into account in our planned exposure modeling (Mansfield and Corey, 2003; Wen et al., 2009). We do not feel that this is a relatively influential uncertainty at this time.

- **Modeling Near-Traffic Outdoor Environments and Public Transportation**

Modeling activities such as walking next to roads, waiting at bus stops, bicycling, and riding motorcycles, buses, subways and trains is difficult due to the limited information available about these activities. It is also difficult to estimate the ambient concentrations in these environments. As mentioned in the previous section, staff are working on methods to characterize these exposures. An important part of this effort will be to characterize the uncertainties of these methods that we plan to incorporate into the quantitative uncertainty analysis.

4.8 UNCERTAINTY AND VARIABILITY

The primary difficulty in performing an exposure modeling uncertainty analysis is the quantitative characterization of the uncertainties of the model inputs and model formulation. Information about the variability of model inputs or the variability and uncertainty combined is often available, but it is usually difficult to estimate the uncertainty separately from the variability. In considering the use of APEX for a $PM_{2.5}$ exposure assessment, EPA has considered the availability of information to provide plausible distributions or ranges for the uncertainties of all of the model inputs. EPA plans to build upon the APEX exposure modeling uncertainty analysis conducted in support of the recent review of the ozone NAAQS (Langstaff, 2007). We plan to improve on these distributions of variability and uncertainty, where data are available to do so, and to extend the analysis of model formulation uncertainty.

Once estimates of the uncertainty of the model inputs have been developed, we plan to propagate these uncertainties through the model to quantify the resultant uncertainty of the model predictions. The APEX uncertainty analysis methodology incorporates a 2-dimensional Monte Carlo sampling approach that explicitly characterizes and models the variability and uncertainty in inputs and outputs. Essentially, this approach entails performing thousands of model runs with model inputs randomly sampled from specified distributions reflecting uncertainty of the model inputs, while each single APEX run simulates distributions of variability. This 2-dimensional Monte Carlo method allows for the separate characterization of the variability and uncertainty in the model results (Morgan and Henrion, 1990; Cullen and Frey, 1999). This approach allows for great flexibility in specifying uncertainty distributions for any of the model inputs and parameters that are supplied to APEX by input files. Furthermore, this allows us to specify conditional distributions and joint distributions between parameters for

which we have data, which can be critically important in modeling uncertainty (Haas, 1997; Haas, 1999; Wu and Tsang, 2004).

Uncertainties are inherent in modeled representations of physical reality due to simplifying assumptions and other aspects of model formulation. The methods for assessing input parameter uncertainty and model formulation or structure uncertainty are different. It is difficult to incorporate the uncertainties due to the model formulation into a quantitative assessment of uncertainty in a straightforward manner. The preferred way to assess model formulation uncertainty is by comparing model predictions with measured values, while having fairly complete knowledge of the uncertainty due to input parameters. EPA plans to ascertain whether sufficient data are available to perform such an evaluation. For example, we will consider using the data collected in the Detroit Study (DEARS[28]) for this purpose. In the absence of measurements that can be used to estimate model uncertainty, our planned approach to assessing model formulation uncertainty will be to partition this uncertainty into that of the components, or sub-models, of APEX. For each of the sub-models within APEX, we plan to discuss the simplifying assumptions and those uncertainties associated with the sub-models which are distinct from the input data uncertainties. Where possible, we plan to evaluate these sub-models by comparing their predictions with measured data. Alternatively, we may formulate an informed judgment as to a range of plausible uncertainties for the sub-models. We plan to quantitatively assemble the different types of uncertainties and variability to present an integrated analysis of uncertainty and variability.

[28] See http://www.epa.gov/dears

5 SCHEDULE AND MILESTONES

Table 5-1 lists the key milestones for the Risk and Exposure Assessment (REA) that are planned as part of the current PM NAAQS review. Consultation with the CASAC PM Panel is scheduled for April 1-2, 2009 to obtain review of the first draft Integrated Science Assessment (ISA) and to obtain input on the plans to conduct quantitative assessments. EPA staff will then proceed to develop health risk estimates associated with recent PM ambient concentrations and levels representing just meeting the current PM standards and exposure estimates associated with historical ambient $PM_{2.5}$ concentrations. These estimates and the methodologies used will be presented in the first draft PM REA. CASAC and public comments on this plan will be taken into consideration in the development of the first draft REA, the preparation of which will coincide and draw from the second draft ISA. The first draft report is scheduled to be released for CASAC and public review in August 2009. EPA will receive comments on this draft document from the CASAC and the general public at a meeting planned for September 2009. The second draft REA will draw on the final ISA and will reflect consideration of CASAC and public comments on the first draft REA. The second draft REA will include assessments for just meeting potential alternative standards. We plan to release the second draft REA in March 2010 for review by CASAC and the general public at a meeting that is planned for April 2010. Staff will consider these review comments and prepare a final REA, currently planned to be completed in July 2010. The final REA will reflect consideration of CASAC and public comments on the second draft REA. The final ISA and final REA will inform the policy assessment and rulemaking steps that will lead to a final decision of the PM NAAQS. Our current schedule includes plans for issuing a proposed rule in January 2011 and a final rule in October 2011.

Table 5-1 Key Milestones for the Risk and Exposure Assessment

Milestone	Date
Release first draft PM ISA	December 2008
Release draft PM REA Scope and Methods Plans	February 2009
CASAC/public review and meeting on first draft PM ISA	April 1, 2009
CASAC consultation on draft PM REA Scope and Methods Plans	April 2, 2009
Release second draft PM ISA	July 2009
Release first draft of the PM REA	August 2009
CASAC/public review and meeting on second draft PM ISA and first draft REA	September 2009
Final PM ISA	December 2009
Release second draft of the PM REA	March 2010
CASAC/public review and meeting on second draft of the PM REA	April 2010
Final PM REA	July 2010

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APPENDIX A: PREPARATION OF MODEL INPUTS FOR THE PILOT EXPOSURE ASSESSMENT

This appendix describes the APEX model inputs and the data used to create the APEX input files for the pilot exposure assessment. Note that these model inputs are preliminary and ongoing efforts to compile data to better characterize the distributions of parameters used for APEX inputs will be described in the first draft REA. For example, recent air exchange rate (AER) data will be used to update the distributions of AERs.

The geographic extent of the modeling region consists of the census tracts in and overlapping the Detroit domain for recent photochemical model simulations on a 1 km grid. This domain is bounded by longitudes from -83.8410 to -82.7875 degrees and latitudes from 41.8039 to 42.8552 degrees, and has 72 columns and 108 rows. Figure A- 1 shows this domain as well as the locations of the $PM_{2.5}$ ambient monitors in this area in 2002. The population, commuting, activity, and meteorological data used are described in section 4.6 above.

The microenvironments that we plan to model are those listed in Table 4-1 in Chapter 4. There are some particle size dependent parameters used in the mass-balance model. We plan to model $PM_{2.5}$ in four size ranges to be able to capture size dependencies. The size categories of $PM_{2.5}$ modeled in this simulation are 0-0.03, 0.03-0.1, 0.1-1.0 and 1.0-2.5 μm. It is likely that we will revise these ranges after additional studies are reviewed. Although exposures will be modeled for these size categories, we plan to combine them in order to obtain $PM_{2.5}$ exposures and do not plan to report the size categories separately. If the estimated exposures are not sensitive to this refinement in this pilot application, we plan to reconsider the appropriateness of modeling separate size classes in the additional cities analyzed.

The microenvironment parameters that must be specified for the mass-balance model include air exchange rates, penetration factors, and decay and deposition rates for indoor areas. The parameters for the factors model calculations include proximity factors for outdoor microenvironments and vehicles and penetration values for vehicles.

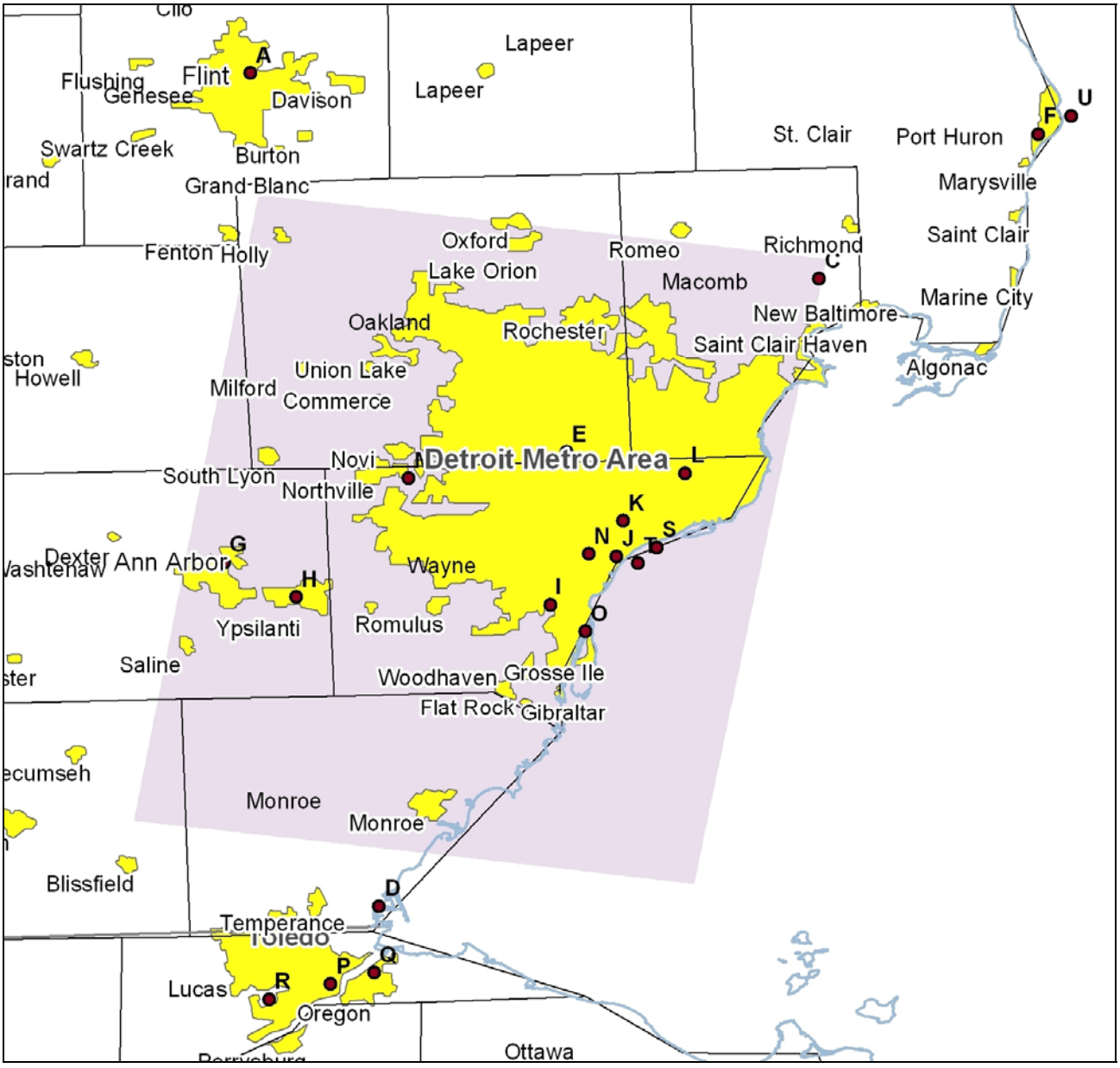

Figure A- 1. The Detroit Modeling Region and $PM_{2.5}$ Monitors

Residential Indoor Microenvironments

We plan to use the air exchange rates for residential microenvironments identified in **Table A-1** in the Detroit pilot analysis. These values are the same as AERs used in the exposure analysis conducted to support the recent ozone NAAQS review (EPA, 2007). The fraction of

residences with central or room air conditioning in Detroit is estimated by the 2003 American Housing Survey to be 0.81 (AHS, 2003).

Table A-1. Lognormal Distribution Parameters for Residential Air Exchange Rates (hr^{-1})

Temp (F)	A/C	Geometric mean	Geometric standard deviation
< 50	Yes	0.71	2.02
50 - 68	Yes	1.14	2.68
68 - 77	Yes	1.14	2.68
77 - 86	Yes	1.24	2.18
> 86	Yes	1.24	2.18
< 50	No	1.02	2.14
50 - 68	No	0.79	2.04
68 - 77	No	1.61	2.12
77 - 86	No	1.61	2.12
> 86	No	1.61	2.12

minimum = 0.1, maximum = 10

The deposition values used in this analysis are presented in Table A- 2 . These were estimated using data from Long et al. (2001), who estimated average decay rates based on their measurements of 31 homes (Long et al. 2000). The data from the study were remapped from smaller size intervals onto the four ranges selected for this simulation. The less than 0.03 bin is represented by the 0.02-0.03 Long et al. bin; the 0.03-0.10 bin is the sum of four Long et al. bins: 0.03-0.04, 0.04-0.06, 0.06-0.08, and 0.08-1.0. The 0.1-1.0 bin is the sum of the six Long et al. bins: 0.1-0.15, 0.15-0.2, 0.2-0.3, 0.3-0.4, 0.4-0.5, and 0.7-1.0. The 1.0-2.5 bin is the sum of the 1-2 and 2-3 Long et al. bins. Uncertainties were converted from standard errors to standard deviations and added in quadrature. The Long et al. (2001) study differentiates summer and winter because the houses included in the study were located in the Northeast U.S. and residents typically opened windows during the summer months for air ventilation. We define the summer months as the six warmest months of the year, May through October.

Table A- 2 Normal Distribution Parameters for Depositional/Decay Rates (hr^{-1})

Size (µm)	Season	Mean	Standard deviation
<0.03	Summer	0.59	0.84
0.03 - 0.1	Summer	0.35	0.30
0.1 – 1.0	Summer	0.20	0.23
1.0 – 2.5	Summer	0.72	1.63
<0.03	Winter	0.13	0.64
0.03 - 0.1	Winter	0.11	0.27
0.1 – 1.0	Winter	0.22	0.26
1.0 – 2.5	Winter	0.43	0.41

minimum = 0, maximum = 5

Penetration rates for residences (Table A- 3) are also estimated using data presented by Long et al. (2001), who measured penetration rates of 41 homes. The data from the study were remapped from smaller size intervals onto the four ranges selected for this simulation. The effect of this remapping is reflected in the larger standard deviations. As described above, the study divided the year into summer and winter months.

Table A- 3 Normal Distribution Parameters for Penetration Rates

Size (µm)	Season	Mean	Standard deviation
<0.03	Summer	0.90	0.89
0.03 - 0.1	Summer	0.98	0.38
0.1 – 1.0	Summer	0.94	0.32
1.0 – 2.5	Summer	0.63	1.28
<0.03	Winter	0.55	0.57
0.03 - 0.1	Winter	0.66	0.29
0.1 – 1.0	Winter	0.75	0.26
1.0 – 2.5	Winter	0.57	0.32

minimum = 0, maximum = 1

Non-residential Indoor Microenvironments

Air exchange rates for non-residential microenvironments are specified as lognormal distributions with geometric mean of 1.109 hr^{-1}, and geometric standard deviation of 3.015. Minimum and maximum bounds of 0.07 to 13.8 are used, with resampling if a sample is outside of these bounds. This distribution is described in (U.S. EPA, 2007) and is based on two studies:

the Building Assessment Survey and Evaluation (BASE) Study (Persily and Gorfain 2004; Persily et al. 2005) and a study presented in Turk et al. (1989).

Outdoor Microenvironments and Vehicles

The parameters for modeling these microenvironments are currently being developed. For this analysis, we are planning to use as preliminary distributions the values used in an exposure modeling study of New Haven, CT conducted by ORD using SHEDS-PM (pers. comm., Janet Burke, 3/21/08). The proximity factors for outdoors near-road, public garages and parking lot, and all in-vehicle microenvironments are assigned lognormal distributions with geometric mean 3.320 and geometric standard deviation 0.902 on weekdays, and geometric mean 2.075 and geometric standard deviation 1.498 on weekends. These factors are intended to account for the typically higher ambient concentrations in these areas than the concentrations at monitoring locations. If we use near-road dispersion modeling in estimating the spatial concentration fields, then we will use proximity factors centered around 1.0. Penetration factors for vehicles are assigned normal distributions with mean 0.3 and standard deviation 0.232. For this APEX application, we plan to assign minimum and maximum values of 0.2 and 20 for the proximity factors, and 0.1 and 1.0 for penetration factors, and resample if these bounds are exceeded. We incorporate bounds on the distributions so that unrealistic values will not be generated.

Ambient $PM_{2.5}$ Concentrations

There were six hourly and 12 daily $PM_{2.5}$ monitors in and near the greater Detroit metropolitan area in 2002. Three of the hourly monitors are in Canada and three are in Michigan; the daily monitors are all in Michigan (for these counts collocated monitors are counted as single monitors). The monitors in Canada are in the NAPS network, and the Michigan monitors are all from AQS. Table A- 4 lists the averaging time, period, number of days with measurements, and the monitoring method for each monitor during the period July 1-31, 2002. Collocated monitors are grouped within rows. Figure A- 1 shows the locations of these monitors using the map location codes in Table A- 4, except for location B, which is to the west in Lansing.

Table A- 4 $PM_{2.5}$ Monitors in Detroit Region, July 1-31, 2002

Monitor id [1]	Averaging time (hours)	Period (days)	# Days with data	Monitor method [2]	Map location
2604900211 2604900213	24 1	3 -	11 31	118 701	A
2606500122	24	6	6	118	B
2609900091	24	3	10	118	C
2611500051 2611500055	24 24	3 6	10 6	118 810	D
2612500011	24	3	8	118	E
2614700051 2614700052	24 24	3 6	10 5	118 118	F
2616100051	24	3	10	118	G
2616100081 2616100082 2616100083	24 24 1	3 6 -	10 3 31	118 118 701	H
2616300011 2616300012 2616300013 2616300015	24 24 1 24	1 6 - 3	19 3 31 10	118 118 701 810	I
2616300151	24	3	8	118	J
2616300161	24	1	28	118	K
2616300191	24	3	11	118	L
2616300251	24	3	11	118	M
2616300331 2616300335	24 24	3 6	11 6	118 810	N
2616300361	24	3	9	118	O
3909500241	24	1	31	120	P
3909500251	24	3	11	120	Q
3909500261 3909500265	24 24	1 6	30 6	120 810	R
NAPS060204	1	-	31	cont	S
NAPS060211	1	-	31	cont	T
NAPS061004	1	-	31	cont	U

[1] Collocated monitors are grouped within rows

[2] Method 118: FRM - R & P MODEL 2025 PM2.5 SEQUENTIAL w/WINS / Gravimetric
Method 120: FRM - ANDERSEN RAAS2.5-300 PM2.5 SEQ w/WINS / Gravimetric
Method 701: TEOM Gravimetric 50º C with $PM_{2.5}$ SCC and no correction factor.
Method 810: SASS / Gravimetric

We plan to estimate missing air quality data from hourly monitors using the following procedure: where there are consecutive strings of missing values (data gaps) of less than six

hours, missing values will be estimated by linear interpolation between the valid values at the ends of the gap and any remaining missing values at a monitor will be estimated by fitting linear regression models for each hour of the day with each of the other monitors, and for each hour of the day choosing the model which maximizes R^2, subject to the constraints that R^2 must be greater than 0.5 and the number of regression data values is at least 50 (the maximum number of values possible is 365 when one year is modeled). If there are any remaining missing values at this point, for gaps of less than nine hours, we plan to estimate missing values by linear interpolation between the valid values at the ends of the gap. Any remaining missing values after that would be replaced with the region-wide mean concentration for that hour. We plan to continue to investigate methods for estimating missing data from daily monitors.

One of the major difficulties to be resolved is how to estimate the hourly spatially-varying $PM_{2.5}$ air quality concentration fields, which are inputs to the APEX exposure model. $PM_{2.5}$ air quality monitors are spatially sparse and often 24-hour average measurements are only reported every three or six days. We plan to evaluate the use of air quality models to augment the monitored ambient $PM_{2.5}$ concentrations to better estimate ambient concentrations at locations between monitors and for days with no measurements, as discussed in section 4.6.

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