Health Risk and Exposure Assessment for Ozone

First External Review Draft

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This preliminary draft document has been prepared by staff from the Risk and Benefits Group, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency. Any opinions, findings, conclusions, or recommendations are those of the authors and do not necessarily reflect the views of the EPA. This document is being circulated for informational purposes and to facilitate discussion with the Clean Air Scientific Advisory Committee (CASAC) on the overall structure, areas of focus, and level of detail to be included in an external review draft Policy Assessment, which EPA plans to release for CASAC review and public comment later this year. Questions related to this preliminary draft document should be addressed to Karen Wesson, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, C504-02, Research Triangle Park, North Carolina 27711 (email: wesson.karen@epa.gov).

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First External Review Draft

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LIST OF ACRONYMS/ABBREVIATIONS

AER	air exchange rate
AHRQ	Agency for Healthcare Research and Quality
APEX	Air Pollution Exposure Model
AQI	Air Quality Index
AQS	Air Quality System
ATUS	American Time Use Survey
BenMAP	Benefits Mapping and Analysis Program
BRFSS	Behavioral Risk Factor Surveillance System
BSA	body surface area
CAA	Clean Air Act
CASAC	Clean Air Science Advisory Committee
CDC	Center for Disease Control and Prevention
CDF	cumulative distribution functions
CH_4	methane
CHAD	Consolidated Human Activity Database
CI	confidence interval
CMAQ	Community Multi-scale Air Quality
CO_2	carbon dioxide
C-R	Concentration Response (function)
ED	emergency department
EGU	electric generating unit
EPA	U.S. Environmental Protection Agency
ER	emergency room
eVNA	enhanced Voronoi Neighbor Averaging
EVR	equivalent ventilation rate
FEM	Federal Equivalent Method
FEV1	one-second forced expiratory volume
FRM	Federal Reference Method
FVC	forced vital capacity

HA	hospital admissions
HDDM	Higher-order Decoupled Direct Method
HNO_3	nitric acid
HO_2	hydro-peroxy radical
HUCP	Healthcare Cost and Utilization Program
IPCC	Intergovernmental Panel on Climate Change
IRP	Integrated Review Plan
ISA	Integrated Science Assessment
LML	lowest measured level
MATS	Modeled Attainment Test Software
METs	metabolic equivalents of work
MSA	Metropolitan Statistical Area
MT	metric ton
NAAQS	National Ambient Air Quality Standards
NCDC	National Climatic Data Center
NEI	National Emissions Inventory
NO	nitric oxide
NO_2	nitrite
NO_x	nitrogen oxides
O_3	Ozone
OAQPS	Office of Air Quality Planning and Standards
OH	hydroxyl radical
PA	Policy Assessment
PDI	pain on deep inspiration
PI	posterior interval
PM	particulate matter
ppb	parts per billion
ppm	parts per million
PRB	Policy Relevant Background
REA	Risk and Exposure Assessment
RR	relative risk

SAB	Science Advisory Board
SEDD	State Emergency Department Databases
SES	socioeconomic status
SID	State Inpatient Databases
SO2	sulfur dioxide
STE	stratosphere-troposphere exchange
TRIM Expo	Total Risk Integrated Methodology Inhalation Exposure
VE	ventilation rate
VNA	Voronoi Neighbor Averaging
VOC	volatile organic carbon
WHO	World Health Organization

1 INTRODUCTION

The U.S. Environmental Protection Agency (EPA) is presently conducting a review of the national ambient air quality standards (NAAQS) for ozone (O_3) and related photochemical oxidants. An overview of the approach to reviewing the O_3 NAAQS is presented in the *Integrated Review Plan for the Ozone National Ambient Air Quality Standards* (IRP, US EPA, 2011a). The IRP discusses the schedule for the review; the approaches to be taken in developing key scientific, technical, and policy documents; and the key policy-relevant issues that will frame our consideration of whether the current NAAQS for O_3 should be retained or revised.

Sections 108 and 109 of the Clean Air Act (CAA) govern the establishment and periodic review of the NAAQS. These standards are established for pollutants that may reasonably be anticipated to endanger public health and welfare, and whose presence in the ambient air results from numerous or diverse mobile or stationary sources. The NAAQS are to be based on air quality criteria, which are to accurately reflect the latest scientific knowledge useful in indicating the kind and extent of identifiable effects on public health or welfare that may be expected from the presence of the pollutant in ambient air. The EPA Administrator is to promulgate and periodically review, at five-year intervals, "primary" (health-based) and "secondary" (welfare-based) NAAQS for such pollutants. Based on periodic reviews of the air quality criteria and standards, the Administrator is to make revisions in the criteria and standards, and promulgate any new standards, as may be appropriate. The Act also requires that an independent scientific review committee advise the Administrator as part of this NAAQS review process, a function performed by the Clean Air Scientific Advisory Committee (CASAC).[1]

The current primary NAAQS for O_3 is set at a level of 0.075 ppm, based on the annual fourth-highest daily maximum 8-hr average concentration, averaged over three years, and the secondary standard is identical to the primary standard (73 FR 16436). The EPA initiated the

[1] The Clean Air Scientific Advisory Committee (CASAC) was established under section 109(d)(2) of the Clean Air Act (CAA) (42 U.S.C. 7409) as an independent scientific advisory committee. CASAC provides advice, information and recommendations on the scientific and technical aspects of air quality criteria and NAAQS under sections 108 and 109 of the CAA. The CASAC is a Federal advisory committee chartered under the Federal Advisory Committee Act (FACA). See http://yosemite.epa.gov/sab/sabpeople.nsf/WebCommitteesSubcommittees/CASAC%20Particulate%20Matter%20Review%20Panel for a list of the CASAC PM Panel members and current advisory activities.

current review of the O_3 NAAQS on September 29, 2008 with an announcement of the development of an O_3 Integrated Science Assessment and a public workshop to discuss policy-relevant science to inform EPA's integrated plan for the review of the O_3 NAAQS (73 FR 56581). The NAAQS review process includes four key phases: planning, science assessment, risk/exposure assessment, and policy assessment/rulemaking.[2] A workshop was held on October 29-30, 2008 to discuss policy-relevant scientific and technical information to inform EPA's planning for the O_3 NAAQS review. Following the workshop, EPA developed a planning document, the *Integrated Review Plan for the Ozone National Ambient Air Quality Standards* (IRP; US EPA, 2011), which outlined the key policy-relevant issues that frame this review, the process and schedule for the review, and descriptions of the purpose, contents, and approach for developing the other key documents for this review.[3] In June 2012, EPA completed the third draft of the O_3 ISA, assessing the latest available policy-relevant scientific information to inform the review of the O_3 standards. The *Integrated Science Assessment for Ozone and Related Photochemical Oxidants - Third External Review Draft* (ISA; US EPA, 2012), includes an evaluation of the scientific evidence on the health effects of O_3, including information on exposure, physiological mechanisms by which O_3 might adversely impact human health, an evaluation of the toxicological and controlled human exposure study evidence, and an evaluation of the epidemiological evidence including information on reported concentration-response (C-R) relationships for O_3-related morbidity and mortality associations, including consideration of effects on susceptible populations.[4]

The EPA's Office of Air Quality Planning and Standards (OAQPS) has developed this first draft quantitative health risk and exposure assessment (REA) describing preliminary quantitative assessments of exposure to O_3 and O_3-related risks to public health to support the review of the primary O_3 standards. This draft document presents the conceptual model, scope, methods, key results, observations, and related uncertainties associated with the quantitative analyses performed. The REA builds upon the health effects evidence presented and assessed in

[2] For more information on the NAAQS review process see http://www.epa.gov/ttn/naaqs/review.html.

[3] On March 30, 2009, EPA held a public consultation with the CASAC Ozone Panel on the draft IRP. The final IRP took into consideration comments received from CASAC and the public on the draft plan as well as input from senior Agency managers.

[4] The ISA also evaluates scientific evidence for the effects of O_3 on public welfare which EPA will consider in its review of the secondary O_3 NAAQS. Building upon the effects evidence presented in the ISA, OAQPS has also developed a second REA titled *Ozone Welfare Effects Risk and Exposure Assessment (US EPA, 2012).*

the ISA, as well as CASAC advice (Samet, 20011) and public comments on a scope and methods planning document for the REA (here after, "Scope and Methods Plan", US EPA, 2011). Revisions to this draft REA will draw upon the final ISA and will reflect consideration of CASAC and public comments on this draft.

The ISA and REA will inform the development of a Policy Assessment (PA) and rulemaking steps that will lead to final decisions on the primary O_3 NAAQS, as described in the IRP. The PA will include staff analysis of the scientific basis for alternative policy options for consideration by senior EPA management prior to rulemaking. The PA integrates and interprets information from the ISA and the REA to frame policy options for consideration by the Administrator. The PA is intended to link the Agency's scientific and technical assessments, presented in the ISA and REA, to judgments required of the Administrator in determining whether it is appropriate to retain or revise the current O_3 standards. Development of the PA is also intended to facilitate elicitation of CASAC's advice to the Agency and recommendations on any new standards or revisions to existing standards as may be appropriate, as provided for in the Clean Air Act (CAA). The first draft PA is planned for release around the middle of August 2012 for review by the CASAC O_3 Panel and the public concurrently with their review of this first draft REA September 11-13, 2012.

1.1 HISTORY

As part of the last O_3 NAAQS review completed in March 2008, EPA's OAQPS conducted quantitative risk and exposure assessments to estimate exposures above health benchmarks and risks of various health effects associated with exposure to ambient O_3 in a number of urban study areas selected to illustrate the public health impacts of this pollutant (U.S. EPA 2007a, U.S. EPA 2007b). The assessment scope and methodology were developed with considerable input from CASAC and the public, with CASAC generally concluding that the exposure assessment reflected generally accepted modeling approaches, and that the risk assessments were well done, balanced and reasonably communicated (Henderson, 2006a). The final quantitative risk and exposure assessments took into consideration CASAC advice (Henderson, 2006a; Henderson, 2006b) and public comments on two drafts of the risk and exposure assessments.

The exposure and health risk assessment conducted in the last review developed exposure and health risk estimates for 12 urban areas across the U.S. based on 2002 to 2004 air quality data. That assessment provided annual or O_3 season-specific exposure and risk estimates for these years of air quality and for air quality scenarios simulating just meeting the then-existing 8-hour O_3 standard set in 1997 at a level of 0.08 ppm and several alternative 8-hour standards. The strengths and limitations in the assessment were characterized, and analyses of key uncertainties were presented.

Exposure estimates from the last assessment were used as an input to the risk assessment for lung function responses (a health endpoint for which exposure-response functions were available from controlled human exposure studies). Exposure estimates were developed for the general population and population groups including school age children with asthma as well as all school age children. The exposure estimates also provided information on exposures to ambient O_3 concentrations at and above specified benchmark levels (referred to as "exposures of concern") to provide some perspective on the public health impacts of health effects associated with O_3 exposures in controlled human exposure studies that could not be evaluated in the quantitative risk assessment (e.g., lung inflammation, increased airway responsiveness, and decreased resistance to infection).

The last human risk assessment included risk estimates based on both controlled human exposure studies and epidemiological and field studies. Ozone-related risk estimates for lung function decrements were generated using probabilistic exposure-response relationships based on data from controlled human exposure studies, together with probabilistic exposure estimates from the exposure analysis. For several other health endpoints, O_3-related risk estimates were generated using concentration-response relationships reported in epidemiological or field studies, together with ambient air quality concentrations, baseline health incidence rates, and population data for the various locations included in the assessment. Health endpoints included in the assessment based on epidemiological or field studies included: hospital admissions for respiratory illness in four urban areas, premature mortality in 12 urban areas, and respiratory symptoms in asthmatic children in 1 urban area.

The last exposure and risk assessment helped to inform the last review and the final decision to revise the primary O_3 NAAQS to a level of 0.075 ppm, as discussed in the Final Rule notice (73 FR 16436; March 27, 2008). As an initial matter, in considering the adequacy of the

then-current standard, while the Administrator placed primary consideration on the body of scientific evidence of O_3-related health effects, he also considered the exposure and risk assessment results and related uncertainties. In so doing, the Administrator considered the estimated percentages of asthmatic and all school age children likely to experience exposures (while at moderate or greater exertion) at and above the benchmark levels of 0.080, 0.070 and 0.060 ppm upon simulation of just meeting the then-current standard, as well as the year-to-year and city-to-city variability and the uncertainties is those estimates. He also considered the estimated health risks for lung function decrements, respiratory symptoms, respiratory-related hospital admissions and mortality upon simulation of just meeting the then-current standard, as well as the variability and uncertainties in those estimates. He recognized that these risk estimates were indicative of a much broader array of O_3-related health endpoints that could not be included in the quantitative assessment (e.g., school absences, increased medication use, emergency department visits) which primarily affect at-risk populations. In considering this information, the Administrator concluded that the estimated exposures and risks were important from a public health perspective and that they provide additional support to the evidence-based conclusion that the then-current standard needed to be revised.

In considering the level at which a revised primary O_3 standard should be set, within the proposed range of 0.070 to 0.075 ppm, the Administrator again placed primary consideration on the body of scientific evidence of O_3-related health effects, while viewing the results of the exposure and risk assessment as providing information in support of his decision. In considering the exposure estimates simulated for meeting alternative standard levels, the Administrator placed greatest weight on estimated exposures at and above the 0.080 ppm benchmark level, less weight on the 0.070 ppm benchmark, and very little weight on the 0.060 ppm benchmark. Given the degree of uncertainty in these estimates, he judged that there was not an appreciable difference, from a public health perspective, in the estimates of exposures associated with just meeting a standard at the upper end (0.075 ppm) versus the lower end (0.070 ppm) of the proposed range of levels. The Administrator placed less weight on the risk estimates for meeting alternative standard levels, and noted that the results suggest a gradual reduction in risks with no clear breakpoint as increasingly lower standard levels are considered. Taken together, the Administrator judged that the exposure and risk information did not provide a clear basis for choosing a specific level within the range of levels being considered. In reaching a final

evidence-based decision to set the standard at a level of 0.075 ppm, the Administrator noted that this level was above the range of levels recommended by CASAC (0.060 to 0.070 ppm). In explaining the basis for this difference with CASAC, the Administrator noted that there is no bright line clearly directing the choice of level, and the choice of an appropriate level is clearly a public health policy judgment. In reaching his final judgment, the Administrator explained in part that CASAC appeared to place greater weight on the results of the risk assessment as a basis for its recommended range, while he more heavily weighed the implications of the uncertainties associated with the exposure and risk assessments.

Following promulgation of the revised O_3 standard in March 2008, state, public health, environmental, and industry petitioners filed suit against EPA regarding that final decision. At EPA's request the consolidated cases were held in abeyance pending EPA's voluntary reconsideration of the 2008 decision. A notice of proposed rulemaking to reconsider the 2008 final decision was issued by the Administrator on January 6, 2010. On September 2, 2011, the Office of Management and Budget returned the draft final rule on reconsideration to EPA for further consideration. EPA decided to coordinate further proceedings on its voluntary rulemaking on reconsideration with this ongoing periodic review, by deferring the completion of its voluntary rulemaking on reconsideration until it completes its statutorily-required periodic review. In light of that, the litigation on the 2008 final decision is no longer being held in abeyance and is proceeding. The 2008 O_3 standards remain in effect.

1.2 CURRENT RISK AND EXPOSURE ASSESSMENT: GOALS AND PLANNED APPROACH

The goals of the current quantitative exposure and health risk assessments are (1) to provide estimates of the number of people in the general population and in sensitive populations with O_3 exposures above benchmark levels while at moderate or greater exertion levels; (2) to provide estimates of the number of people in the general population and in at-risk populations with impaired lung function resulting from exposures to O_3; (3) to provide estimates of the potential magnitude of premature mortality and selected morbidity health effects in the population, including at-risk populations, where data are available to assess these groups, associated with recent ambient levels of O_3 and with just meeting the current primary O_3 standard and any alternative standards that might appropriately be considered in selected urban

study areas; (4) to develop a better understanding of the influence of various inputs and assumptions on the exposure and risk estimates to more clearly differentiate alternative standards that might be considered including potential impacts on various at-risk populations; and (5) to gain insights into the distribution of risks and patterns of risk reduction and uncertainties in those risk estimates. In addition, we have conducted an assessment to provide nationwide estimates of the potential magnitude of premature mortality associated with ambient O_3 exposures to more broadly characterize this risk on a national scale. This assessment includes an evaluation of the distribution of risk across the U.S., to assess the extent to which we have captured the upper end of the risk distribution with our urban study area analyses.

This current quantitative risk and exposure assessment builds on the approach used and lessons learned in the last O_3 risk and exposure assessment and focuses on improving the characterization of the overall confidence in the exposure and risk estimates, including related uncertainties, by incorporating a number of enhancements, in terms of both the methods and data used in the analyses. This risk assessment considers a variety of health endpoints for which, in staff's judgment, there is adequate information to develop quantitative risk estimates that can meaningfully inform the review of the primary O_3 NAAQS.

The results from this risk and exposure assessment will be considered from a policy perspective in the PA. The PA will also evaluate the entire body of scientific evidence of relationships between O_3 and a wide array of health endpoints, including those considered in the risk assessment, from a policy perspective. These evidence-based and exposure/risk-based considerations will inform staff's assessment of various policy options as discussed in the PA.

This first draft REA provides an assessment of exposure and risk associated with recent ambient levels of O_3 and O_3 air quality simulated to just attain the current primary O_3 standards. Subsequent drafts of the REA will evaluate potential alternative O_3 standards based on considerations discussed in the first draft of the Policy Assessment.

1.3 ORGANIZATION OF DOCUMENT

The remainder of this document, when final, will be organized as follows. Chapter 2 provides a conceptual framework for the risk and exposure assessment, including discussions of O_3 chemistry, sources of O_3 precursors, exposure pathways and microenvironments where O_3 exposure can be high, at-risk populations, and health endpoints associated with O_3. This

conceptual framework sets the stage for the scope of the risk and exposure assessments. Chapter 3 provides an overview of the scope of the quantitative risk and exposure assessments, including a summary of the previous risk and exposure assessments, and an overview of the current risk and exposure assessments. Chapter 4 discusses air quality considerations relevant to the exposure and risk assessments, including available O_3 monitoring data, and important inputs to the risk and exposure assessments. Chapter 5 describes the inputs, models, and results for the human exposure assessment, and discusses the literature on exposure to O_3, exposure modeling approaches using the Air Pollution Exposure Model (APEX), the scope of the exposure assessment, inputs to the exposure modeling, sensitivity and uncertainty evaluations, and estimation of results. Chapter 6 describes the estimation of health risks based on application of the results of human clinical studies, including discussions of health endpoint selection, approaches to calculating risk, and results. (We note that work is continuing on Chapter 6 and we expect to release a first draft of that chapter in August.) Chapter 7 describes the estimation of health risks in selected urban areas based on application of the results of observational epidemiology studies, including discussions of air quality characterizations, model inputs, variability and uncertainty, and results. Chapter 8 describes the national scale risk characterization and urban area representativeness analysis. Chapter 9 provides an integrative discussion of the exposure and risk estimates generated in the analyses drawing on the results of the analyses based on both clinical and epidemiology studies, and incorporating considerations from the national scale risk characterization.

2 CONCEPTUAL FRAMEWORK

In this chapter, we summarize the conceptual framework for assessing exposures to O_3 and the associated risks to human populations. This conceptual framework includes elements related to characterization of ambient O_3 and its relation to population exposures (Section 2.1), important sources of O_3 precursors including oxides of nitrogen (NO_x) and volatile organic compounds (VOC) (Section 2.2), exposure pathways and important microenvironments where O_3 exposures may be high (Section 2.3), populations that may be at greater risk due to increased exposure or other factors that increase vulnerability and susceptibility (Section 2.4), and health outcomes identified in the literature as associated with ambient O_3 (Section 2.5).

2.1 OZONE CHEMISTRY

O_3 occurs naturally in the stratosphere where it provides protection against harmful solar ultraviolet radiation, and it is formed closer to the surface in the troposphere by both natural and anthropogenic sources. O_3 is not emitted directly into the air, but is created when its two primary precursors, volatile organic compounds (VOC) and oxides of nitrogen (NO_x), combine in the presence of sunlight. VOC and NO_x are, for the most part, emitted directly into the atmosphere. Carbon monoxide (CO) and methane (CH_4) are also important for O_3 formation (US EPA, 2012, section 3.2.2).

Rather than varying directly with emissions of its precursors, O_3 changes in a nonlinear fashion with the concentrations of its precursors. NO_x emissions lead to both the formation and destruction of O_3, depending on the local quantities of NO_x, VOC, and radicals such as the hydroxyl (OH) and hydro-peroxy (HO2) radicals. In areas dominated by fresh emissions of NO_x, these radicals are removed via the production of nitric acid (HNO3), which lowers the O_3 formation rate. In addition, the scavenging of O_3 by reaction with NO is called “titration,” and is often found in downtown metropolitan areas, especially near busy streets and roads, and in power plant plumes. This titration results in local valleys in which ozone concentrations are low compared to surrounding areas. Titration is usually short-lived confined to areas close to strong NO_x sources, and the NO_2 formed this way leads to O_3 formation later and further downwind. . Consequently, ozone response to reductions in NO_x emissions is complex and may include ozone decreases at some times and locations and increases of ozone to fill in the local valleys of low

ozone. In areas with low NO_x concentrations, such as those found in remote continental areas to rural and suburban areas downwind of urban centers, the net production of O_3 typically varies directly with NO_x concentrations, and increases with increasing NO_x emissions.

In general, the rate of O_3 production is limited by either the concentration of VOCs or NO_x, and O_3 formation using these two precursors relies on the relative sources of OH and NO_x. When OH radicals are abundant and are not depleted by reaction with NO_x and/or other species, O_3 production is referred to as being "NO_x-limited" (US EPA, 2012, section 3.2.4). In this situation, O_3 concentrations are most effectively reduced by lowering NO_x emissions, rather than lowering emissions of VOCs. When the abundance of OH and other radicals is limited either through low production or reactions with NO_x and other species, O_3 production is sometimes called "VOC-limited" or "radical limted" or "NO_x-saturated" (Jaegle et al., 2001), and O_3 is most effectively reduced by lowering VOCs. However, even in NO_x-saturated conditions, very large decreases in NO_x emissions can cause the ozone formation regime to become NO_x limited. Consequently, reductions in NO_x emissions (when large) can make further emissions reductions more effective at reducing ozone. Between the NO_x-limited and NO_x-saturated extremes there is a transitional region where O_3 is relatively insensitive to marginal changes in both NO_x and VOCs. In rural areas and downwind of urban areas, O_3 production is generally NO_x-limited. However, across urban areas with high populations, conditions may vary. For contrast, while data from monitors in Nashville, TN suggest NO_x-limited conditions exist there, data from monitors in Los Angeles suggest NO_x-saturated conditions (US EPA, 2012, Figure 3-3).

2.2 SOURCES OF O_3 AND O_3 PRECURSORS

O_3 precursor emissions can be divided into anthropogenic and natural source categories, with natural sources further divided into biogenic emissions (from vegetation, microbes, and animals) and abiotic emissions (from biomass burning, lightning, and geogenic sources). The anthropogenic precursors of O_3 originate from a wide variety of stationary and mobile sources.

In urban areas, both biogenic and anthropogenic VOCs are important for O_3 formation. Hundreds of VOCs are emitted by evaporation and combustion processes from a large number of anthropogenic sources. Based on the 2005 national emissions inventory (NEI), solvent use and highway vehicles are the two main sources of VOCs, with roughly equal contributions to total emissions (US EPA, 2012, Figure 3-3). The emissions inventory categories of "miscellaneous"

(which includes agriculture and forestry, wildfires, prescribed burns, and structural fires) and off-highway mobile sources are the next two largest contributing emissions categories with a combined total of over 5.5 million metric tons a year (MT/year).

On the U.S. and global scales, emissions of VOCs from vegetation are much larger than those from anthropogenic sources. Emissions of VOCs from anthropogenic sources in the 2005 NEI were ~17 MT/year (wildfires constitute ~1/6 of that total), compared to emissions from biogenic sources of 29 MT/year. Vegetation emits substantial quantities of VOCs, such as isoprene and other terpenoid and sesqui-terpenoid compounds. Most biogenic emissions occur during the summer because of their dependence on temperature and incident sunlight. Biogenic emissions are also higher in southern and eastern states than in northern and western states for these reasons and because of species variations.

Anthropogenic NO_x emissions are associated with combustion processes. Based on the 2005 NEI, the three largest sources of NO_x are on-road and off-road mobile sources (e.g., construction and agricultural equipment) and electric power generation plants (EGUs) (US EPA, 2012, Figure 3-3). Emissions of NO_x therefore are highest in areas having a high density of power plants and in urban regions having high traffic density. However, it is not possible to make an overall statement about their relative impacts on O_3 in all local areas because EGUs are sparser than mobile sources, particularly in the west and south and because of the nonlinear chemistry discussed in Section 2.1.

Major natural sources of NO_x in the U.S. include lightning, soils, and wildfires. Biogenic NO_x emissions are generally highest during the summer and occur across the entire country, including areas where anthropogenic emissions are low. It should be noted that uncertainties in estimating natural NO_x emissions are much larger than for anthropogenic NO_x emissions.

Ozone concentrations in a region are affected both by local formation and by transport from surrounding areas. Ozone transport occurs on many spatial scales including local transport between cities, regional transport over large regions of the U.S. and international/long-range transport. In addition, O_3 is also transfered into the troposphere from the stratosphere, which is rich in O_3, through stratosphere-troposphere exchange (STE). These inversions or "foldings" usually occur behind cold fronts, bringing stratospheric air with them (U.S. EPA, 2012, section 3.4.1.1). Contribution to O_3 concentrations in an area from STE are defined as being part of background O_3 (U.S. EPA, 2012, section 3.4).

2.3 EXPOSURE PATHWAYS AND IMPORTANT MICROENVIRONMENTS

Human exposure to O_3 involves the contact (via inhalation) between a person and the pollutant in the various locations (or microenvironments) in which people spend their time. Ozone concentrations in some indoor microenvironments, such as within homes or offices, are considerably lower than O_3 concentrations in similarly located outdoor microenvironments, primarily due to deposition processes and the transformation of O_3 into other chemical compounds within those indoor microenvironments. Concentrations of O_3 may also be quite different in roadway environments, such as might occur while an individual is in a vehicle.

Thus, three important classes of microenvironments that should be considered when evaluating population exposures to ambient O_3 are indoors, outdoors, and in-vehicle. Within each of these broad classes of microenvironments, there are many subcategories, reflecting types of buildings, types of vehicles, etc. The O_3 ISA evaluated the literature on indoor-outdoor O_3 concentration relationships and found that studies consistently show that indoor concentrations of O_3 are often substantially lower than outdoor concentrations unless indoor sources are present. This relationship is greatly affected by the air exchange rate, which can be affected by open windows, use of air conditioning, and other factors. Ratios of indoor to outdoor O_3 concentrations generally range from about 0.1 to 0.4 (US EPA, 2012, section 4.3.2). In some indoor locations, such as schools, there can be large temporal variability in the indoor-outdoor ratios because of differences in air exchange rates over the day. For example, during the school day, there is an increase in open doors and windows, so the indoor-outdoor ratio is higher during the school day compared with an overall average across all hours and days. In-vehicle concentrations are also likely to be lower than ambient concentrations, although the literature providing quantitative estimates is smaller. Studies of personal exposure to O_3 have identified that O_3 exposures are highest when individuals are in outdoor microenvironments, such as walking outdoors midday, moderate when in vehicle microenvironments, and lowest in residential indoor microenvironments (US EPA, 2012, section 4.3.3). Thus the time spent indoors, outdoors, and in vehicles is likely to be a critical component in estimating O_3 exposures.

Another important issue in characterizing exposure involves consideration of the extent to which people in relevant population groups modify their behavior for the purpose of decreasing their personal exposure to O_3 based on information about air quality levels made public through the Air Quality Index (AQI). The AQI is the primary tool EPA has used to

provide information on expected occurrences of high levels of O_3 and other pollutants. The AQI provides both the expected level of air quality in an area along with a set of actions that individuals and communities can take to reduce exposure to air pollution and thus reduce the risk of health effects associated with breathing ambient air pollution. There are several studies, discussed in the O_3 ISA, that have evaluated the degree to which populations are aware of the AQI and what actions individuals and communities take in response to AQI values in the unhealthy range. These studies suggest that susceptible populations, such as children, older adults, and asthmatics, modify their behavior in response to days with bad air quality, most commonly by reducing their time spent outdoors or limiting their outdoor activity exertion level. The challenge remains in how to consider averting behaviors as they currently exist within the assessment tools we use and how best to quantitatively estimate the impact on estimated exposures and health risks in response to improved knowledge of participation rates, the varying types of actions performed particularly by potentially susceptible individuals, and the duration of these averting behaviors.

2.4 AT-RISK POPULATIONS

The O3 ISA refers to "at risk" populations as an all-encompassing term used for groups with specific factors that increase the risk of an air pollutant- (e.g., O3) related health effect in a population that group (US EPA, 2012, chapter 8). Populations or lifestages can experience elevated risks from O3 exposure for a number of reasons. These include high levels of exposure due to activity patterns which include a high duration of time in high O3 environments, e.g. outdoor recreation or work, high levels of activity which increase the dose of O3, e.g. high levels of exercise, genetic or other biological factors, e.g. life stage, which predispose an individual to sensitivity to a given dose of O3, pre-existing diseases, e.g. asthma or COPD, and socioeconomic factors which may result in more severe health outcomes, e.g. low access to primary care can lead to increased emergency department visits or hospital admissions. Modeling of exposures to O_3 should incorporate information on time spent by potentially at-risk populations in key high O_3 environments. This requires identification of populations with key exposure-related risk factors, e.g. children or adults engaging in activities involving moderate to high levels of outdoor exertion, especially on a repeated basis typical of student athletes or outdoor workers, as well as

identifying populations with high sensitivity to O_3, e.g. asthmatic children. It also requires that information on O_3 concentrations be carefully mapped to environments where at-risk populations are likely to be exposed, e.g. near roadways where running may occur, or at schools or parks where children are likely to be engaged in outdoor activities.

In addition to consideration of factors that lead to increased exposure to O_3, modeling of risk from O_3 exposures should incorporate additional information on factors that can lead to increased dose of O_3 for a given exposure, e.g. increased breathing rates during periods of exertion. These factors are especially important for risk estimates based on application of the results of controlled human exposure studies which attempt to control for dose-related factors. For risk modeling based on application of observational epidemiology results, it is also important to understand characteristics of study populations that can impact observed relationships between ambient O_3 and population health responses.

The O_3 ISA identifies a number of factors which have been associated with modifications of the effect of ambient O_3 on health outcomes. Building on the causal framework used throughout the O_3 ISA, conclusions are made regarding the strength of evidence for each factor that may contribute to increased risk of an O_3-related health effect based on the evaluation and synthesis of evidence across scientific disciplines. The O_3 ISA categorizes potential risk modifying factors by the degree of available evidence. These categories include "adequate evidence," "suggestive evidence," "inadequate evidence," and "evidence of no effect." See Table 8-1 of the O_3 ISA for a discussion of these categories (US EPA, 2012, chapter 8).

Factors categorized as having adequate evidence include asthma, lifestage (children <18 and older adults ≥65 are more susceptible than young and middle aged adults), diets with nutritional deficiencies, and working outdoors. For example, children are the group considered to be at greatest risk because they breathe more air per pound of body weight, are more likely to be active outdoors when O_3 levels are high, are more likely than adults to have asthma, and their lungs continue to develop until they are fully grown. Factors categorized as having suggestive evidence include genetic markers, sex (some studies have shown that females are at greater risk of mortality from O_3 compared to males), low socioeconomic status, and obesity. Factors characterized as having inadequate evidence include influenza and other respiratory infections, COPD, cardiovascular disease, diabetes, hyperthyroidism, race, and smoking (US EPA, 2012, section 8.5, Table 8-4).

Populations with greater proportions of individuals with characteristics associated with higher risk from O_3 exposure are likely to have a greater risk from any given level of O_3. As a result, risk assessments focused on identifying populations with high levels of O_3 risk should focus on locations with high proportions of at-risk populations, including children and older adults and people with asthma and low socioeconomic status.

2.5 HEALTH ENDPOINTS

The O_3 ISA identifies a wide range of health outcomes associated with *short-term* exposure to ambient O_3, including an array of morbidity effects as well as premature mortality. The ISA also identifies several morbidity effects and some evidence for premature mortality associated with *longer-term* exposures to O_3. In considering health endpoints that are appropriate for a risk assessment, it is useful to focus on endpoints that cover susceptible populations, provide additional information about patterns or magnitude of risk, have public health significance, and have sufficient information available in the literature to provide an appropriate concentration-response function, in the case of epidemiological studies, or an appropriate exposure-response function, in the case of controlled human exposure studies.

Generally speaking, epidemiology studies are well suited to risk assessment because they are based on population responses to ambient air pollution exposure, and include responses of populations with a wide range of susceptibility to O_3. Further, such studies can evaluate serious health endpoints, including hospital admissions and premature mortality. However, epidemiology studies have not traditionally been based on observations of personal exposure to ambient O_3, and instead have used population exposure surrogates, often based on simple averages of O_3 monitor observations. Controlled human exposure studies are also useful for risk assessment, in combination with population-level assessments of exposure to ambient O_3, in that they are based on direct measurement of controlled O_3 exposures to individuals. However, controlled human exposure studies are generally focused on small numbers of relatively healthy individuals, and therefore cannot represent the range of susceptibility in the population, and in fact are clearly biased away from highly susceptible individuals. Controlled human exposure studies also can only evaluate less serious indicators of health effects such as one-second forced expiratory volume (FEV1) as an indicator of lung function or respiratory symptoms such as

cough or pain on deep inspiration. Given the strengths and limitations in both types of studies, analyses of risk using the results of both types of studies are appropriate.

Estimates of risk based on results of human controlled human exposure studies are valuable because there is clear evidence from these studies that there is a causal relationship between exposures to O_3 over multiple hours and reductions in lung function at moderate levels of exertion. In addition, results of these studies can be applied to modeled estimates of population exposure to provide additional insights into the types of population exposure characteristics, including activity patterns and microenvironments that are associated with high levels of risk. Estimates of risk based on results of observational epidemiology studies are valuable because they often focus on more serious health endpoints which could not be assessed in controlled human exposure studies. Epidemiological studies generally evaluate health outcomes in an entire population or subpopulation, which includes both more sensitive and less sensitive individuals, and thus may be able to identify more serious health effects in at-risk subpopulations which cannot be evaluated in controlled human exposure studies which generally exclude individuals likely to experience significant adverse health effects from O_3 exposure. Epidemiological studies of O_3 documented in the ISA have evaluated the relationship between O_3 and various endpoints including respiratory symptoms, respiratory-related hospitalizations and emergency department (ED) visits, and premature mortality.

The O_3 ISA makes overall causal determinations based on the full range of evidence including epidemiological, controlled human exposure and toxicological studies. Figure 2-1 shows the O_3 health effects which have been categorized by strength of evidence for causality in the O_3 ISA (US EPA, 2012, chapter 2). These determinations support causal relationships between *short-term* exposure to O_3 and respiratory effects, including respiratory-related morbidity and mortality, a likely causal relationship with all-cause total mortality, and are suggestive of a causal relationship for cardiovascular and central nervous system effects. The determinations also support a likely causal relationship between *long-term* O_3 exposures and respiratory effects (including respiratory symptoms, new-onset asthma, and respiratory mortality), and are suggestive of causal relationships between long-term O_3 exposures and mortality as well as cardiovascular, reproductive and developmental, and central nervous system effects.

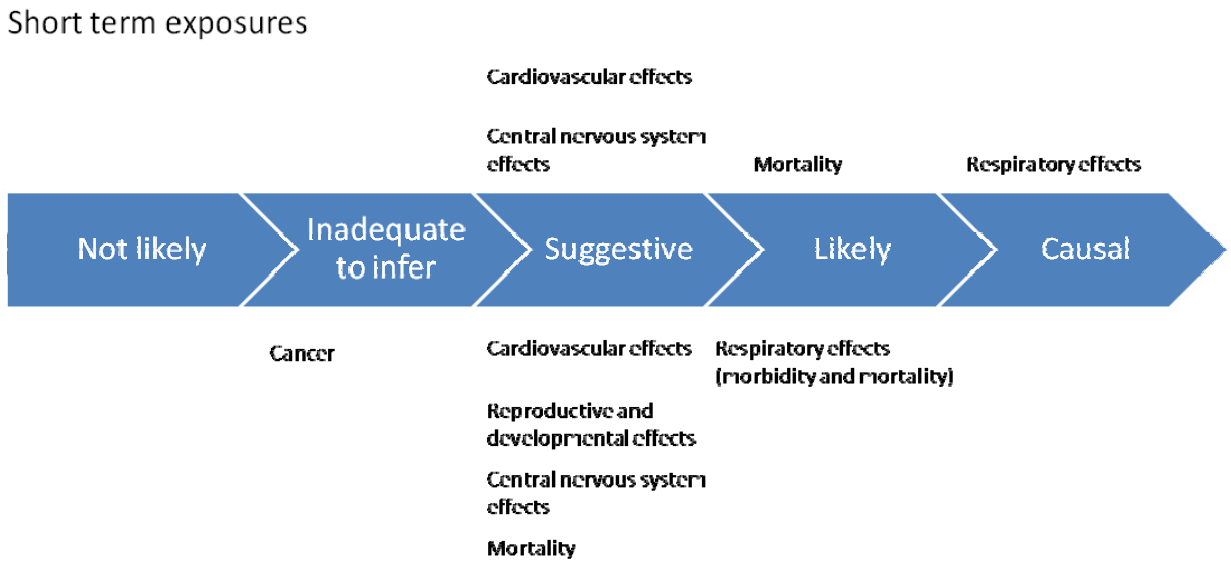

Figure 2-1. Causal Determinations for O_3 Health Effects

The ISA identifies several responses to short-term O_3 exposure that have been evaluated in controlled human exposure studies (US EPA, 2012, section 6.2.1). These include decreased inspiratory capacity, decreased forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1); mild bronchoconstriction; rapid, shallow breathing patterns during exercise; symptoms of cough and pain on deep inspiration (PDI); and pulmonary inflammation. While such studies provide direct evidence of relationships between short-term O_3 exposure and an array of respiratory-related effects, there are only sufficient exposure-response data at different concentrations to develop quantitative risk estimates for O_3-related decrements in FEV1.

Within the broad category of respiratory morbidity effects, the epidemiology literature has provided effect estimates for a wide range of health endpoints associated with short-term O_3 exposures which can be used in risk assessment. These health endpoints include lung function, respiratory symptoms and medication use, respiratory-related hospital admissions and emergency department visits. In the case of respiratory symptoms, the evidence is most consistently supportive of the relationship between short-term ambient O_3 metrics and respiratory symptoms

and asthma medication use in children with asthma, but not for O_3 and these health outcomes in children without asthma. In the case of hospital admissions, there is evidence of associations between shot-term ambient O_3 metrics and general respiratory-related hospital admissions as well as more specific asthma-related hospital admissions.

With regard to mortality, studies have evaluated associations between short-term ambient O_3 metrics and all-cause, non-accidental, and cause-specific (usually respiratory or cardiovascular) mortality. The evidence from respiratory-related morbidity studies provides strong support for respiratory-related mortality for which a causal determination has been made. There are also a number of large studies that have found associations between O_3 and all-cause and all non-accidental mortality for which a likely causal determination has been made. Thus, it is appropriate to assess risks for respiratory-related mortality as well as for all-cause total mortality associated with O_3 exposure.

With regard to effects associated with long-term O_3 exposures, ISA reports a likely causal relationship between O_3 and respiratory-related effects, including respiratory symptoms, new-onset asthma, and respiratory mortality.. This suggests that for long-term exposures, when comparing the evidence for respiratory-related mortality and total mortality, the evidence is most supportive of risks for respiratory-related mortality, supported by the strong evidence for respiratory morbidity. As a result, it is appropriate to consider including respiratory mortality rather than total mortality in the risk assessment, and to give consideration to additional such respiratory-related health endpoints.

2.6 REFERENCES

US EPA. 2012. Integrated Science Assessment of Ozone and Related Photochemical Oxidants (Third External Review Draft). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-10/076C, 2012.S EPA.

3 SCOPE

This chapter provides an overview of the scope and key design elements of this quantitative exposure and health risk assessment. The design of this assessment began with a review of the exposure and risk assessments completed during the last O_3 NAAQS review (US EPA, 2007a,b), with an emphasis on considering key limitations and sources of uncertainty recognized in that analysis.

As an initial step in the current O_3 NAAQS review, in October 2009, EPA invited outside experts, representing a broad range of expertise (e.g., epidemiology, human and animal toxicology, statistics, risk/exposure analysis, atmospheric science) to participate in a workshop with EPA staff to help inform EPA's plan for the review. The participants discussed key policy-relevant issues that would frame the review and the most relevant new science that would be available to inform our understanding of these issues. One workshop session focused on planning for quantitative risk and exposure assessments, taking into consideration what new research and/or improved methodologies would be available to inform the design of quantitative exposure and health risk assessment. Based in part on the workshop discussions, EPA developed a draft IRP (US EPA, 2009) outlining the schedule, process, and key policy-relevant questions that would frame this review. On November 13, 2009, EPA held a consultation with CASAC on the draft IRP (74 FR 54562, October 22, 2009), which included opportunity for public comment. The final IRP incorporated comments from CASAC (Samet, 2009) and the public on the draft plan as well as input from senior Agency managers. The final IRP included initial plans for quantitative risk and exposure assessments for both human health and welfare (US EPA, 2011a, chapters 5 and 6).

As a next step in the design of these quantitative assessments, OAQPS staff developed more detailed planning documents, O_3 *National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment* (Health Scope and Methods Plan; US EPA, 2011b) and O_3 *National Ambient Air Quality Standards: Scope and Methods Plan for Welfare Risk and Exposure Assessment* (Welfare Scope and Methods Plan, US EPA, 2011c). These Scope and Methods Plans were the subject of a consultation with CASAC on May 19-20, 2011 (76 FR 23809, April 28, 2011). Based on consideration of CASAC (Samet, 2011) and public comments on the Scope and Methods Plan and information in the second draft ISA, we

modified the scope and design of the quantitative risk assessment and provided a memo with updates to information presented in the Scope and Methods Plans (Wegman, 2012). The Scope and Methods Plans together with the update memo provide the basis for the discussion of the scope of this exposure and risk assessment provided in this chapter.

In presenting the scope and key design elements of the current risk assessment, this chapter first provides a brief overview of the quantitative exposure and risk assessment completed for the previous O_3 NAAQS review in section 3.1, including key limitations and uncertainties associated with that analysis. Section 3.2 provides a summary of the design of the exposure assessment. Section 3.3 provides a summary of the design of the risk assessment based on application of results of human clinical studies. Section 3.4 provides a summary of the design of the risk assessment based on application of results of epidemiology studies.

3.1 OVERVIEW OF EXPOSURE AND RISK ASSESSMENTS FROM LAST REVIEW

3.1.1 OVERVIEW OF EXPOSURE ASSESSMENT FROM LAST REVIEW

The exposure and health risk assessment conducted in the review completed in March 2008 developed exposure and health risk estimates for 12 urban areas across the U.S., which were chosen, based on the location of O_3 epidemiological studies and to represent a range of geographic areas, population demographics, and O_3 climatology. That analysis was in part based upon the exposure and health risk assessments done as part of the review completed in 1997.[1] The exposure and risk assessment incorporated air quality data (i.e., 2002 through 2004) and provided annual or O_3 season-specific exposure and risk estimates for these recent years of air quality and for air quality scenarios simulating just meeting the existing 8-hour O_3 standard and several alternative 8-hour O_3 standards. Exposure estimates were used as an input to the risk assessment for lung function responses (a health endpoint for which exposure-response functions were available from controlled human exposure studies). Exposure estimates were developed for

[1] In the 1994-1997 Ozone NAAQS review, EPA conducted exposure analyses for the general population, children who spent more time outdoors, and outdoor workers. Exposure estimates were generated for 9 urban areas for as is air quality and for just meeting the existing 1-hour standard and several alternative 8-hour standards. Several reports that describe these analyses can be found at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr.html.

the general population and population groups including school age children with asthma as well as all school-age children. The exposure estimates also provided information on population exposures exceeding potential health effect benchmark levels that were identified based on the observed occurrence of health endpoints not explicitly modeled in the health risk assessment (e.g., lung inflammation, increased airway responsiveness, and decreased resistance to infection) associated with 6-8 hour exposures to O_3 in controlled human exposure studies.

The exposure analysis took into account several important factors including the magnitude and duration of exposures, frequency of repeated high exposures, and breathing rate of individuals at the time of exposure. Estimates were developed for several indicators of exposure to various levels of O_3 air quality, including counts of people exposed one or more times to a given O_3 concentration while at a specified breathing rate, and counts of person-occurrences which accumulate occurrences of specific exposure conditions over all people in the population groups of interest over an O_3 season.

As discussed in the 2007 Staff Paper (US EPA, 2007c) and in Section IIa of the O_3 Final Rule (73 FR 16440 to 16442, March 27, 2008), the most important uncertainties affecting the exposure estimates were related to modeling human activity patterns over an O_3 season, modeling of variations in ambient concentrations near roadways, and modeling of air exchange rates that affect the amount of O_3 that penetrates indoors. Another important uncertainty, discussed in more detail in the Staff Paper (US EPA, 2007c, section 4.3.4.7), was the uncertainty in energy expenditure values which directly affected the modeled breathing rates. These were important since they were used to classify exposures occurring when children were engaged in moderate or greater exertion and health effects observed in the controlled human exposure studies generally occurred under these exertion levels for 6 to 8-hour exposures to O_3 concentrations at or near 0.08 ppm. Reports that describe these analyses (U.S. EPA, 2007a,c; Langstaff, 2007) can be found at: http://www.epa.gov/ttn/naaqs/standards/O_3/s_O_3_index.html.

3.1.2 OVERVIEW OF RISK ASSESSMENT FROM LAST REVIEW

The human health risk assessment presented in the review completed in March 2008 was designed to estimate population risks in a number of urban areas across the U.S., consistent with the scope of the exposure analysis described above (U.S. EPA, 2007b,c). The risk assessment

included risk estimates based on both controlled human exposure studies and epidemiological and field studies. O_3-related risk estimates for lung function decrements were generated using probabilistic exposure-response relationships based on data from controlled human exposure studies, together with probabilistic exposure estimates from the exposure analysis. For several other health endpoints, O_3-related risk estimates were generated using concentration-response relationships reported in epidemiological or field studies, together with ambient air quality concentrations, baseline health incidence rates, and population data for the various locations included in the assessment. Health endpoints included in the assessment based on epidemiological or field studies included: hospital admissions for respiratory illness in four urban areas, premature mortality in 12 urban areas, and respiratory symptoms in asthmatic children in 1 urban area.

In the health risk assessment conducted in the previous review, EPA recognized that there were many sources of uncertainty and variability in the inputs to the assessment and that there was a high degree of uncertainty in the resulting risk estimates. The statistical uncertainty surrounding the estimated O_3 coefficients in epidemiology-based concentration-response functions as well as the shape of the exposure-response relationship chosen for the lung function risk assessment were addressed quantitatively. Additional uncertainties were addressed through sensitivity analyses and/or qualitatively. The risk assessment conducted for the previous O_3 NAAQS review incorporated some of the variability in key inputs to the assessment by using location-specific inputs (e.g., location-specific concentration-response functions, baseline incidence rates and population data, and air quality data for epidemiological-based endpoints, location specific air quality data and exposure estimates for the lung function risk assessment). In that review, several urban areas were included in the health risk assessment to provide some sense of the variability in the risk estimates across the U.S.

Key observations and insights from the O_3 risk assessment, in addition to important caveats and limitations, were addressed in Section II.B of the Final Rule notice (73 FR 16440 to 14 16443, March 27, 2008). In general, estimated risk reductions associated with going from current O_3 levels to just meeting the current and alternative 8-hour standards showed patterns of decreasing estimated risk associated with just meeting the lower alternative 8-hour standards considered. Furthermore, the estimated percentage reductions in risk were strongly influenced by the baseline air quality year used in the analysis, which was due to significant year-to-year

variability in O_3 concentrations. There was also noticeable city-to-city variability in the estimated O_3-related incidence of morbidity and mortality across the 12 urban areas. Uncertainties associated with estimated policy-relevant background (PRB) concentrations[2] were also addressed and revealed differential impacts on the risk estimates depending on the health effect considered as well as the location. EPA also acknowledged that at the time of the previous review there were considerable uncertainties surrounding estimates of O_3 C-R coefficients and the shape for concentration-response relationships and whether or not a population threshold or non-linear relationship exists within the range of concentrations examined in the epidemiological studies.

3.2 PLAN FOR THE CURRENT EXPOSURE AND RISK ASSESSMENTS

The Scope and Methods Plan, including updates (U.S. EPA, 2011b; Wegman, 2012), outlined a planned approach for conducting the current quantitative O_3 exposure and risk assessments, including broad design issues as well as more detailed aspects of the analyses. 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. More specifically, we have identified the following goals for the exposure and risk assessment: (1) to provide estimates of the number of people in the general population and in sensitive populations with O_3 exposures above benchmark levels; (2) to provide estimates of the number of people in the general population and in sensitive populations with impaired lung function resulting from exposures to O_3; (3) to provide estimates of the potential magnitude of premature mortality and/or selected morbidity health effects in the population, including sensitive populations, associated with recent ambient levels of O_3 and with just meeting the current O_3 standard and any alternative standards that might be considered in selected urban study areas; (4) 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 populations; (5) to gain insights into the distribution of risks and patterns of risk reduction and

[2]Policy-relevant background (PRB) ozone has been defined in previous reviews as the distribution of ozone concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made) emissions of ozone precursor emissions (e.g., VOC, CO, NOx) in the U.S., Canada, and Mexico.

uncertainties in those risk estimates; and (6) to understand the national mortality burden associated with recent ambient O_3, and how well the risk estimates for the set of urban areas modeled reflect the national distribution of mortality risk. In addition, we are evaluating the degree to which current evidence supports estimation of morbidity and mortality associated with longer-term exposures to O_3.

The planned approaches for conducting the exposure and risk analyses are briefly summarized below. We begin with a discussion of the air quality data that will be used in both the exposure and risk assessments, and then discuss each component of the exposure and risk assessments.

3.2.1 AIR QUALITY DATA

Air quality inputs to the exposure and risk assessments include: (1) recent air quality data for O_3 from suitable monitors and meteorological data for each selected urban study area; (2) simulated air quality that reflects changes in the distribution of O_3 air quality estimated to occur when an area just meets the current or alternative O_3 standards under consideration[3], and (3) O_3 air quality surfaces for recent years covering the entire continental U.S. for use in the national-scale assessment.

The urban area exposure and risk analyses are based on the five most recent years of air quality data available at this time, 2006-2010. We are including 5 years to reflect the considerable variability in meteorological conditions and the variation in O_3 precursor emissions that have occurred in recent years. The analyses mostly focus on the O_3 season of May to September but also include analysis of additional O_3 measurements during the rest of the year. The required O_3 monitoring season varies for the urban areas as described in more detail in Chapter 4.

Only O_3 data collected by Federal reference or equivalent methods (FRMs or FEMs) are used in the urban area risk and exposure assessments, consistent with the use of such data in most of the health effects studies. In developing the O_3 air quality surfaces for the national-scale analysis, a combination of monitoring data and modeled O_3 concentrations is used to provide

[3] Estimates of U.S. background concentrations (concentrations of ozone estimated to occur if all U.S. anthropogenic emissions of NOx and VOC are eliminated) were used to set a lower bounds for simulating air quality for just meeting the current ozone standard.

greater coverage across the U.S. The procedure for fusing O_3 monitor data with modeling results is described further in Chapter 4.

Several O_3 metrics are generated for use in the urban area exposure and risk analyses. The exposure analyses use hourly O_3 concentrations, while the risk analyses use several different averaging times. The specific metrics used in each analysis are discussed further in following chapters. In addition to temporal averages of O_3 concentrations, spatial averages are also generated for use in the risk analyses based on the specific averaging method applied in the epidemiology studies. Based on the specific approaches used in the source epidemiology studies, we develop a data set for each urban area based on a composite of all monitors according to the method in the epidemiologic study. 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.

Simulation of just meeting the current O_3 standard is accomplished in this first draft REA using a quadratic rollback method similar to what was implemented in the previous risk and exposure analysis for the 2008 O_3 NAAQS review (U.S. EPA, 2007a,b,c). This choice was based on analyses of historical O_3 data which found, from comparing the reductions over time in daily ambient O_3 levels in two locations with sufficient ambient air quality data, that reductions tended to be roughly quadratic. Based on the current understanding of how O_3 forms and reacts to changes in emissions, reductions in emissions that would be needed to meet the current standards are likely to lead to reductions in hourly concentrations for most hours of the day, but may have little impact on concentrations for some hours, and in some cases can lead to increases in O_3 concentrations particularly during nighttime hours. The quadratic rollback method has difficulty representing these complexities in O_3 chemistry and reduces O_3 concentrations over all hours. To address this issue in the rollback methodology for the first draft REA, we are planning to impose a lower bound on O_3 concentration values based on modeled O_3 levels after eliminating all U.S. anthropogenic emissions of O_3 precursors (NOx and VOC). These estimates will be developed using the GEOS-Chem global chemical transport model. This approach is applied so that O_3 concentrations for any particular hour cannot go below the estimated lower bound values.

For the second draft REA, we are evaluating approaches for simulating attainment of current and alternative standards that are based on modeling the response of O_3 concentrations to reductions in anthropogenic NOx and VOC emissions, using the Higher-order Decoupled Direct

Method (HDDM) capabilities in the Community Multi-scale Air Quality (CMAQ) model. This modeling incorporates all known emissions, including emissions from nonanthropogenic sources and anthropogenic emissions from sources in and outside of the U.S. As a result, the need to specify values for U.S. background is not necessary, as it is incorporated in the modeling directly. In simulations of just meeting the standards used to inform the exposure and risk assessment, HDDM sensitivities can be applied relative to ambient measurements of O_3 to estimate how ozone concentrations would respond to changes in anthropogenic emissions within the U.S. The evaluation of this new approach is presented in Chapter 4 of this REA and in more detail in Simon et al. (2012).

In the previous review, background O_3 (referred to in that review as policy relevant background, or PRB) was incorporated into the REA by calculating only risk in excess of PRB. CASAC members recommended that EPA move away from using PRB in calculating risks (Henderson, 2007). EPA is following this advice in the current REA, and as a result, the air quality assessment will not include estimates of background O_3, with the exception of providing a floor for O_3 concentrations when implementing the quadratic rollback method to simulate attainment of the current standards. The evidence and information on background O_3 that is assessed in the Integrated Science Assessment (ISA) will now be considered in the Policy Assessment (PA). With regard to background O_3 concentrations, the PA will consider available information on ambient O_3 concentrations resulting from natural sources, anthropogenic sources outside the U.S., and anthropogenic sources outside of North America.

In providing a broader national characterization of O_3 air quality in the U.S., this REA draws upon air quality data analyzed in the O_3 ISA as well as national and regional trends in air quality as evaluated in EPA's Air Quality Status and Trends document (U.S. EPA, 2008a), and EPA's Report on the Environment (U.S. EPA, 2008b). This information along with additional analyses is used to develop a broad characterization of current air quality across the nation. This characterization includes tables of areas and population in the U.S. exceeding current O_3 standards (and potential alternative standards in the second draft REA). Also included are data on the expected number of days on which the O_3 standards are exceeded, adjusting for the number of days monitored. Further, O_3 levels in locations and time periods relevant to areas assessed in key short-term epidemiological studies used in the risk analysis are characterized. Information on the spatial and temporal characterization of O_3 across the national monitoring

network is also provided. This information is used in the comparison of the attributes of the selected urban study areas to national distributions of attributes to help place the results of that assessment into a broader national context.

3.2.2 EXPOSURE ASSESSMENT

The scope of the exposure assessment will ultimately include the full set of 16 urban areas[4]. For this first draft REA, we have modeled 4 of the 16 urban areas, including Atlanta, Denver, Los Angeles, and Philadelphia. All 16 areas will be modeled in the second draft REA. These areas were selected to be generally representative of a variety of populations, geographic areas, climates, and different O_3 and co-pollutant levels, and are areas where epidemiologic studies have been conducted that support the quantitative risk assessment. In addition to providing population exposures for estimation of lung function effects, the exposure modeling will provide a characterization of urban air pollution exposure environments and activities resulting in the highest exposures, differences in which may partially explain the heterogeneity across urban areas seen in the risks associated with O_3 air pollution.

Population exposure to ambient O_3 levels will be evaluated using version 4.4 of the APEX model. The model and updated documentation are available at http://www.epa.gov/ttn/fera/apex_download.html. APEX is based on the current state of knowledge of inhalation exposure modeling. Exposure estimates are generated for recent O_3 levels, based on 2006-2010 air quality data, and for O_3 levels resulting from simulations of just meeting the current 8-hour O_3 NAAQS and alternative O_3 standards, based on adjusting 2006-2010 air quality data. Exposure estimates are generated for 1) the general population, 2) school-age children (ages 5 to 18), 3) asthmatic school-age children, 4) outdoor workers, and 5) the elderly population (aged 65 and older). This choice of population groups includes a strong emphasis on children, which reflects the results of the last review in which children, especially those who are active outdoors, were identified as the most important at-risk group.

The exposure estimates will be used as an input to the portion of the health risk assessment that is based on exposure-response relationships derived from controlled human

[4] These cities are Atlanta, GA; Baltimore, MD; Boston, MA; Chicago, IL; Cleveland, OH; Dallas, TX; Denver, CO; Detroit, MI; Houston, TX; Los Angeles, CA; New York, NY; Philadelphia, PA; Seattle, WA; Sacramento, CA; St. Louis, MO; and Washington, D.C.

exposure studies. The exposure analysis will also provide information on population exposure exceeding levels of concern that are identified based on evaluation of health effects in the ISA. It will also provide a characterization of populations with high exposures in terms of exposure environments and activities. In addition, the exposure analysis will offer key observations based on the results of the APEX modeling, viewed in the context of factors such as averting behavior and key uncertainties and limitations of the model.

3.2.3 LUNG FUNCTION RISK ASSESSMENT

Prior EPA risk assessments for O_3 have included risk estimates for lung function decrements and respiratory symptoms based on analysis of individual data from controlled human exposure studies. The current assessment applies probabilistic exposure-response relationships which are based on analyses of individual data that describe the relationship between a measure of personal exposure to O_3 and the measure(s) of lung function recorded in the study. The current quantitative risk assessment presents only a partial picture of the risks to public health associated with short-term O_3 exposures, as controlled human exposure studies have only examined markers of short-term reversible lung responses.

The major components in the lung function risk assessment are shown in Figure 3-1. The measure of personal exposure to ambient O_3 is typically some function of hourly exposures – e.g., 1-hour maximum or 8-hour maximum. Therefore, the lung function risk assessment based on exposure-response relationships derived from controlled human exposure study data requires estimates of personal exposure to O_3, typically on a 1-hour or multi-hour basis. Because data on personal hourly O_3 exposures are not available, estimates of personal exposures to varying ambient concentrations are derived through the exposure modeling described above. Controlled human exposure studies, carried out in laboratory settings, are generally not specific to any particular real world location. A controlled human exposure studies-based risk assessment can therefore appropriately be carried out for any locations for which there are adequate air quality data on which to base the modeling of personal exposures.

Modeling of risks of lung function decrements are based on application of results from controlled human exposure studies. These studies involve volunteer subjects who are exposed while engaged in different exercise regimens to specified levels of O_3 under controlled conditions for specified amounts of time. The responses measured in such studies have included

measures of lung function, such as forced expiratory volume in one second (FEV1), respiratory symptoms, airway hyper-responsiveness, and inflammation.

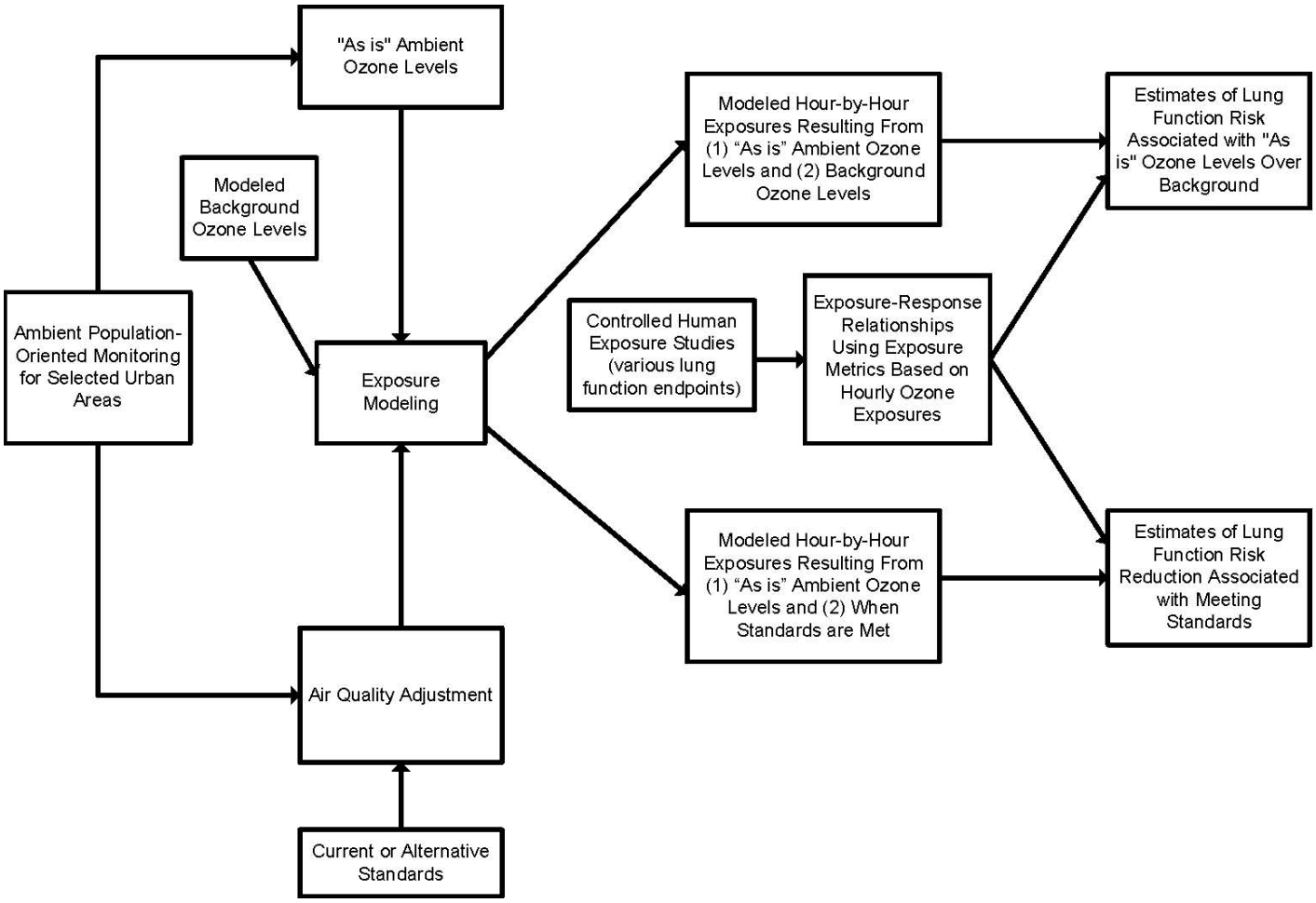

Figure 3- 1 Major Components of Ozone Lung Function Health Risk Assessment Based on Controlled Human Exposure Studies

1

The lung function risk assessment includes lung function decrement risk estimates, using forced expiratory volume in one second (FEV1), for the general population, school age children, asthmatic school age children, outdoor workers, and the elderly population (aged 65 and older) living in 16 urban areas (4 of which are included in this first draft REA) in the U.S. These areas, defined earlier, represent a range of geographic areas, population demographics, and O_3 climatology. These 16 areas also include the 12 urban areas evaluated in the risk analyses based on concentration-response relationships developed from epidemiological or field studies.

This lung function risk assessment estimates lung function decrements (≥ 10, ≥ 24, and $\geq$20% changes in FEV1) in children 5-18 years old associated with 8-hour exposures at moderate exertion. These lung function estimates are based on applying data from adult subjects (18-35 years old) to children 5-18. This is based on findings from other chamber studies and summer camp field studies documented in the 1996 O_3 Staff Paper (US EPA, 1996a) and 1996 O_3 Criteria Document (US EPA, 1996b), that lung function changes in healthy children are similar to those observed in healthy adults exposed to O_3 under controlled chamber conditions.

Risk estimates in this first draft REA are based in part on exposure-response relationships estimated from the combined data sets from multiple O_3 controlled human exposure studies. Data from the studies by Folinsbee et al. (1988), Horstman et al. (1990), and McDonnell et al. (1991) in addition to more recent data from Adams (2002, 2003, 2006) are used to estimate exposure-response relationships for $\geq$ 10, 15, and 20% decrements in FEV1. Based on additional studies identified in the ISA, we will update for the second draft REA the exposure response function using results from two additional recent clinical studies, Kim et al, 2011 and Schelegle, et al, 2009.

Risk measures estimated for lung function risk assessment the numbers of school age children and other groups experiencing one or more occurrences of a lung function decrement >10, > 15, and > 20% in an O_3 season, and total number of occurrences of these lung function decrements in school age children and active school age children.

We are also investigating the possibility of using for the second draft REA an alternative model that estimates FEV1 responses for individuals associated with short-term exposures to O_3 (McDonnell, Stewart, and Smith, 2010). This model is based on the controlled human exposure data included in the prior lung function risk assessment as well as additional data sets for different averaging times and breathing rates. These data were from 15 controlled human O_3

exposure studies that included exposure of 541 volunteers (ages 12 18–35 years) on a total of 864 occasions (see McDonnell et al., 2007, for a description of these data).

3.2.4 URBAN AREA EPIDEMIOLOGY BASED RISK ASSESSMENT

As discussed in the O_3 ISA (US EPA, 2012), a significant number of epidemiological and field studies examining a variety of health effects associated with ambient O_3 concentrations in various locations throughout the U.S., Canada, Europe, and other regions of the world have been published since the last O_3 NAAQS review. As a result of the availability of these epidemiological and field studies and air quality information, this first draft REA includes an assessment of selected health risks attributable to recent ambient O_3 concentrations and health risk reductions associated with attainment of the current O_3 standard in selected urban locations in the U.S. The second draft REA will also include assessments of the health risk reductions associated with attainment of alternative O_3 standards.

The major components of the portion of the health risk assessment based on data from epidemiological and field studies are illustrated in Figure 3-2. The approaches used by staff to select health endpoint categories, urban areas, and epidemiology and field studies to consider for inclusion in the risk assessment are discussed below. Epidemiological and field studies provide estimated concentration-response relationships based on data collected in real world settings. Ambient O_3 concentration is typically measured as the average of monitor-specific measurements, using population-oriented monitors. Population health responses for O_3 have included population counts of school absences, emergency room visits, hospital admissions for respiratory and cardiac illness, respiratory symptoms, and premature mortality. Risk assessment based on epidemiological studies typically requires baseline incidence rates and population data for the risk assessment locations. To minimize uncertainties introduced by extrapolation, a risk assessment based on epidemiological studies can be performed for the locations in which the studies were carried out.

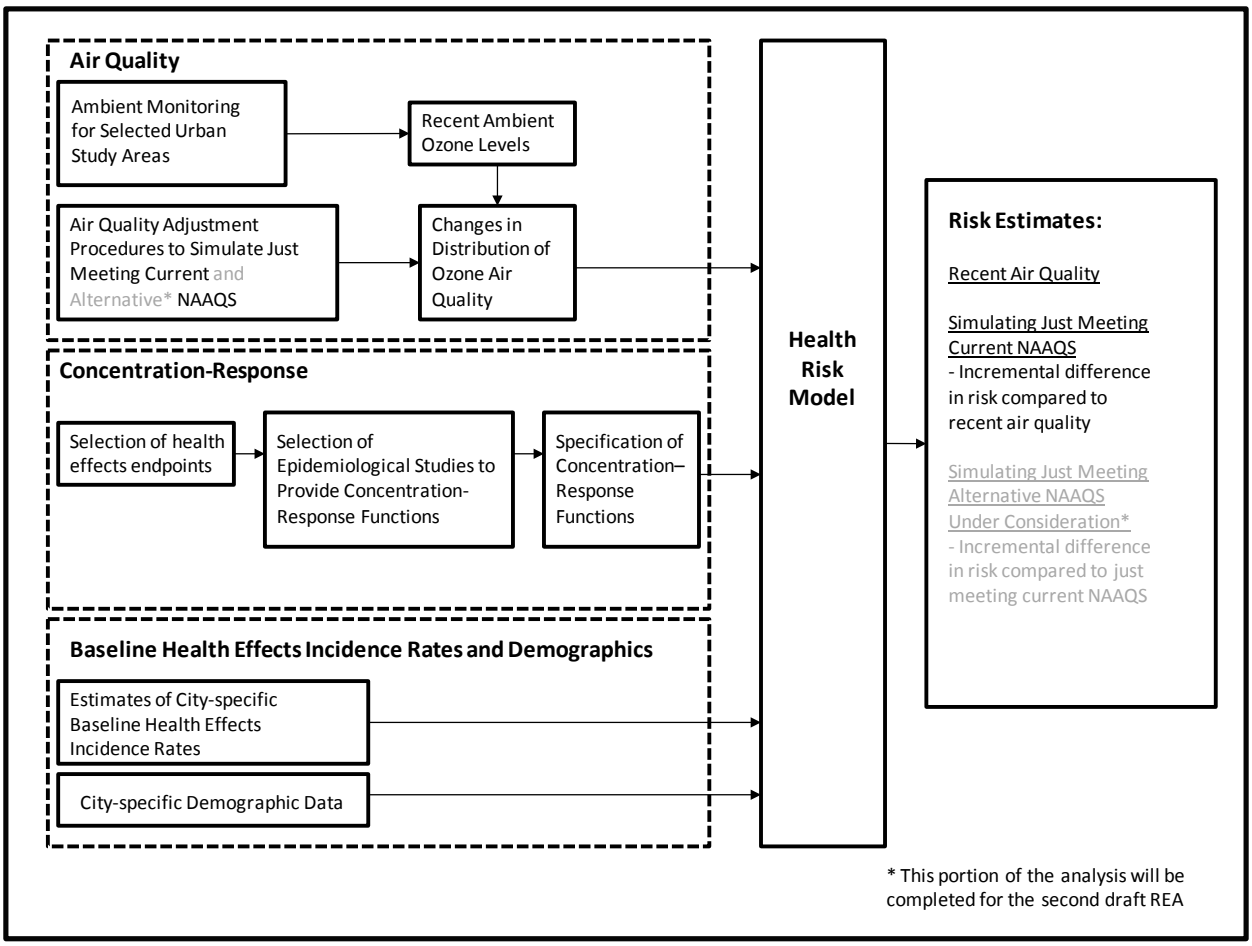

Figure 3- 2 Overview of Risk Assessment Model Based on Results of Epidemiologic Studies

The design of this human health risk assessment reflects goals laid out in the Integrated Review Plan (U.S. EPA, 2011a, section 5.5) including: (1) to provide estimates of the potential magnitude of premature mortality and selected morbidity health effects in the populations in selected urban study areas associated with recent ambient O_3 levels and with just meeting the current suite of O_3 standards and any alternative standards that might be considered; (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.

As in the risk assessment for the previous O_3 NAAQS review, the current risk assessment is focused on modeling risk for a set of selected urban study areas, chosen in order to provide population coverage and to capture the observed heterogeneity in O_3-related risk across selected urban study areas. This assessment also evaluates the risk results for the selected urban areas

within a broader national context to better characterize the nature, magnitude, extent, variability, and uncertainty of the public health impacts associated with O_3 exposures. This national-scale assessment is discussed in the next section.

This risk assessment is focused on health effect endpoints for which the weight of the evidence as assessed in the O_3 ISA supports the judgment that the overall health effect category is at least likely caused by exposure to O_3 either alone and/or in combination with other pollutants. The analysis includes estimates of mortality risk associated with short-term 8-hour O_3 concentrations in all 12 urban case study areas, as well as risk of hospitalization for chronic obstructive pulmonary disease and pneumonia. In addition, the analysis includes additional analysis of hospitalizations for additional respiratory diseases in Los Angeles, New York City, and Detroit, due to limited availability of epidemiology studies covering these endpoints across the 12 urban areas. The analysis also evaluates risks of respiratory related emergency department visits in Atlanta and New York City, and risks of respiratory symptoms in Boston, again based on availability of epidemiology studies in these locations.

This analysis will also consider the respiratory mortality and morbidity risks associated with longer-term exposures to O_3. The third draft ISA classifies respiratory effects, including respiratory mortality and morbidity, as likely causally related to long-term exposures to O_3. However, the availability of epidemiology studies that can provide suitable C-R functions for these endpoints for use in this risk assessment is limited. As a result, for this first draft REA, we are providing a discussion of the potential sources of C-R functions for these endpoints, but are not providing quantitative results, as we are still evaluating the appropriateness of applying the results of the available epidemiology studies for this risk assessment.

We have identified multiple options for specifying the concentration-response functions for particular health endpoints. This risk assessment provides an array of reasonable estimates for each endpoint based on the available epidemiological evidence. This array of results provides a limited degree of information on the variability and uncertainty in risk due to differences in study designs, model specification, and analysis years, amongst other differences. However, the second draft REA will provide a more comprehensive set of sensitivity analyses, especially for the short-term exposure mortality estimates, for which we only provide two sets of estimates based on the primary model specifications in the published studies.

As part of the risk assessment, we address both uncertainty and variability. In the case of uncertainty, we use a four-tiered approach developed by the World Health Organization (WHO) and used in the risk assessment completed for the last PM NAAQS review. The WHO's four-tiered approach matches the sophistication of the assessment of uncertainty to the overall complexity of the risk assessment, while also considering the potential magnitude of the impact that the risk assessment can have from a regulatory/policy perspective (e.g., risk assessments that are complex and are associated with significant regulatory initiatives would likely be subjected to more sophisticated uncertainty analysis). The WHO framework includes the use of sensitivity analysis both to characterize the potential impact of sources of uncertainty on risk estimates and to generate an array of reasonable risk estimates. We will implement the WHO framework more completely in the second draft REA. In the case of variability, we identify key sources of variability associated with O_3 risk (for both short-term and long-term exposure-related endpoints included in the risk assessment) and discuss the degree to which these sources of variability are reflected in the design of the risk assessment.

As part of the analysis, we also provide a representativeness analysis designed to support the interpretation of risk estimates generated for the set of urban study areas included in the risk assessment. The representativeness analysis focuses on comparing the urban study areas to national-scale distributions for key O_3-risk related attributes (e.g., demographics including socioeconomic status, air-conditioning use, baseline incidence rates and ambient O_3 levels). The goal of these comparisons is to assess the degree to which the urban study areas provide coverage for different regions of the country as well as for areas likely to experience elevated O_3-related risk due to their specific mix of attributes related to O_3 risk.

The risk assessment is implemented using the environmental Benefits Mapping and Analysis Program (BenMAP) (Abt Associates, 2008), EPA's GIS-based computer program for the estimation of health impacts associated with air pollution. BenMAP draws upon a database of population, baseline incidence and effect coefficients to automate the calculation of health impacts. EPA has traditionally relied upon the BenMAP program to estimate the health impacts avoided and economic benefits associated with adopting new air quality rules. The following diagram (Figure 3-3) summarizes the data inputs (in black text) and outputs (in blue text) for a typical BenMAP analysis.

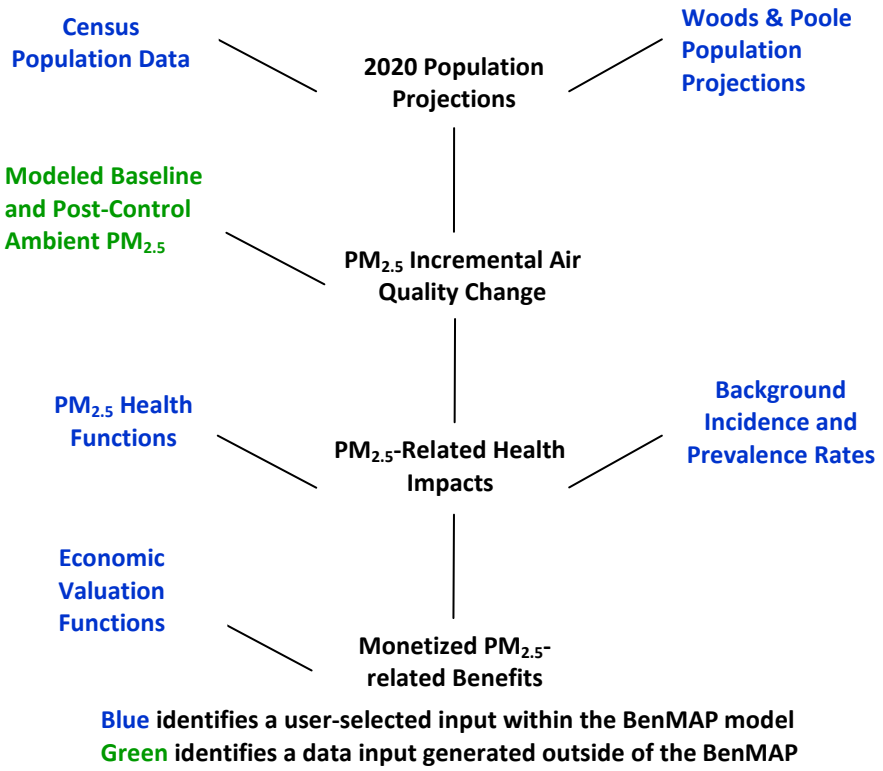

Figure 3- 3 Data Inputs and Outputs for the BenMAP Model

3.2.5 NATIONAL-SCALE MORTALITY RISK ASSESSMENT

The national-scale mortality risk assessment serves two primary purposes. First, it serves as part of the representativeness analysis discussed above, providing an assessment of the degree to which the urban study areas included in the risk assessment provide coverage for areas of the country expected to experience elevated mortality rates due to O_3-exposure. Second, it provides a broader perspective on the distribution of risks associated with recent O_3 concentrations throughout the U.S., and provides a more complete understanding of the overall public health

burden associated with O_3[5]. We note that a national-scale assessment such as this was completed for the risk assessment supporting the latest PM NAAQS review (US EPA, 2010) with the results of the analysis being used to support an assessment of the representativeness of the urban study areas assessed in the PM NAAQS risk assessment, as described here for O_3.

For short-term exposure-related mortality, the assessment provides several estimates of national mortality risk, including a full national-scale estimate including all counties in the continential U.S., and an analysis of just the set of urban areas included in the time series studies that provide the effect estimates used to generate the risk estimates for short-term in the urban case study areas. We have higher confidence in the analysis based on the large urban areas included in the epidemiology studies, but the information from the full analysis of all counties is useful to gain understanding of the potential magnitude of risk in less urbanized areas.

3.2.6 CHARACTERIZATION OF UNCERTAINTY AND VARIABILITY IN THE CONTEXT OF THE O_3 RISK ASSESSMENT

An important component of this population health risk assessment is the characterization of both uncertainty and variability. Variability refers to the heterogeneity of a variable of interest within a population or across different populations. For example, populations in different regions of the country may have different behavior and activity patterns (e.g., air conditioning use, time spent indoors) that affect their exposure to ambient O_3 and thus the population health response. The composition of populations in different regions of the country may vary in ways that can affect the population response to exposure to O_3 – e.g., two populations exposed to the same levels of O_3 might respond differently if one population is older than the other. Variability is inherent and cannot be reduced through further research. Refinements in the design of a population risk assessment are often focused on more completely characterizing variability in key factors affecting population risk – e.g., factors affecting population exposure or response –in

[5] In the previous O3 NAAQS review, CASAC commented that "There is an underestimation of the affected population when one considers only twelve urban "Metropolitan Statistical Areas" (MSAs). The CASAC acknowledges that EPA may have intended to illustrate a range of impacts rather than be comprehensive in their analyses. However, it must be recognized that ozone is a regional pollutant that will affect people living outside these 12 MSAs, as well as inside and outside other urban areas." Inclusion of the national-scale mortality risk assessment partially addresses this concern by providing a broader characterization of risk for an important ozone health endpoint.

order to produce risk estimates whose distribution adequately characterizes the distribution in the underlying population(s).

Uncertainty refers to the lack of knowledge regarding the actual values of inputs to an analysis. Models are typically used in analyses, and there is uncertainty about the true values of the parameters of the model (parameter uncertainty) – e.g., the value of the coefficient for O_3 in a C-R function. There is also uncertainty about the extent to which the model is an accurate representation of the underlying physical systems or relationships being modeled (model uncertainty) – e.g., the shapes of C-R functions. In addition, there may be some uncertainty surrounding other inputs to an analysis due to possible measurement error—e.g., the values of daily O_3 concentrations in a risk assessment location, or the value of the baseline incidence rate for a health effect in a population[6].

In any risk assessment, uncertainty is, ideally, reduced to the maximum extent possible through improved measurement of key variables 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 be both qualitative and, if a sufficient knowledgebase is available, quantitative.

The characterization of uncertainty associated with risk assessment is often addressed in the regulatory context using a tiered approach in which progressively more sophisticated methods are used to evaluate and characterize sources of uncertainty depending on the overall complexity of the risk assessment (WHO, 2008). Guidance documents developed by EPA for assessing air toxics-related risk and Superfund Site risks as well as recent guidance from the World Health Organization specify multitier approaches for addressing uncertainty.

For the O_3 risk assessment, we are using a tiered framework developed by WHO to guide the characterization of uncertainty. The WHO guidance presents a four-tiered approach, where the decision to proceed to the next tier is based on the outcome of the previous tier's assessment. The four tiers described in the WHO guidance include:

[6] It is also important to point out that failure to characterize variability in an input used in modeling can also introduce uncertainty into the analysis. This reflects the important link between uncertainty and variability with the effort to accurately characterize variability in key model inputs actually reflecting an effort to reduce uncertainty.

Tier 0: recommended for routine screening assessments, uses default uncertainty factors (rather than developing site-specific uncertainty characterizations);

Tier 1: the lowest level of site-specific uncertainty characterization, involves qualitative characterization of sources of uncertainty (e.g., a qualitative assessment of the general magnitude and direction of the effect on risk results);

Tier 2: site-specific deterministic quantitative analysis involving sensitivity analysis, interval-based assessment, and possibly probability bounded (high-and low-end) assessment; and

Tier 3: uses probabilistic methods to characterize the effects on risk estimates of sources of uncertainty, individually and combined.

With this four-tiered approach, the WHO framework provides a means for systematically linking the characterization of uncertainty to the sophistication of the underlying risk assessment. Ultimately, the decision as to which tier of uncertainty characterization to include in a risk assessment will depend both on the overall sophistication of the risk assessment and the availability of information for characterizing the various sources of uncertainty.

This risk assessment for the O_3 NAAQS review is relatively complex, thereby warranting consideration of a full probabilistic (WHO Tier 3) uncertainty analysis. However, limitations in available information prevent this level of analysis from being completed for all important elements of uncertainty. In particular, the incorporation of uncertainty related to key elements of C-R functions (e.g., competing lag structures, alternative functional forms, etc.) into a full probabilistic WHO Tier 3 analysis would require that probabilities be assigned to each competing specification of a given model element (with each probability reflecting a subjective assessment of the probability that the given specification is the correct description of reality). However, for most model elements there is insufficient information on which to base these probabilities. One approach that has been taken in such cases is expert elicitation; however, this approach is resource-and time-intensive and consequently, it is not feasible to use this technique in support of this O_3 risk assessment.[7]

[7] While a full probabilistic uncertainty analysis is not undertaken for this risk assessment, we provide a limited assessment using the confidence intervals associated with effects estimates (obtained from epidemiological studies) to incorporate statistical uncertainty associated with sample size considerations in the presentation of risk estimates. Technically, this type of probabilistic simulation represents a Tier 3 uncertainty analysis, although as noted here, it will be limited and only address uncertainty related to the fit of the C-R functions.

For most elements of this risk assessment, rather than conducting a full probabilistic uncertainty analysis, we include a qualitative discussion of the potential impact of uncertainty on risk results (WHO Tier1). The second draft REA will include additional sensitivity analyses assessing the potential impact of sources of uncertainty on risk results (WHO Tier 2). For sensitivity analyses, we will include only those alternative specifications for input parameters or modeling approaches that are deemed to have scientific support in the literature (and so represent alternative reasonable input parameter values or modeling options). This means that the array of risk estimates presented in this assessment are expected to represent reasonable risk estimates that can be used to provide some information regarding the potential impacts of uncertainty in the model elements.

3.2.7 PRESENTATION OF RISK ESTIMATES TO INFORM THE O_3 NAAQS POLICY ASSESSMENT

We plan to conduct the risk assessment in two phases. Phase 1, presented in this first draft REA, includes analysis of risk associated with recent air quality and simulating air quality to just meet the current O_3 NAAQS. Phase 2, which will be included in the second draft REA, will focus on evaluating risk associated with simulating O_3 air quality that just meets alternative O_3 NAAQS under consideration.

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

We present an array of risk estimates in order to provide additional context for understanding the potential impact of uncertainty on the risk estimates. We include risk modeled across the full distribution of O_3 concentrations, as well as core risk estimates ozone concentrations down to zero and down to a surrogate for the lowest measured levels (LML) in the epidemiology studies. According to the O_3 ISA, the controlled human exposure and epidemiologic studies that examined the shape of the C-R function and the potential presence of a threshold have indicated a generally linear C-R function with no indication of a threshold in

analyses that have examined the 8-hour concentrations used in this risk analysis (US EPA, 2012, section 2.5.4.4). The approach most consistent with the statistical models reported in the epidemiological studies is to apply the concentration-response functions to all ozone concentrations down to zero. However, consistent with the conclusions of the ISA, we also recognize that confidence in the nature of the concentration-response function and the magnitude of the risks associated with very low concentrations of ozone is reduced because there are few ozone measurements at the lowest levels in many of the urban areas included in the studies. As a result, the LML provides a cutoff value above which we have higher confidence in the estimated risks. In our judgment, the two sets of estimates based on estimating risk down to zero and estimating risk down to the LML provide a reasonable bound on estimated total risks, reflecting uncertainties about the C-R function below the lowest ozone levels evaluated in the studies.

The results of the representativeness analysis are presented using cumulative probability plots (for the national-level distribution of O_3 risk-related parameters) with the locations where the individual urban study areas fall within those distributions noted in the plots using vertical lines. Similar types of plots are used to present the distribution of national-scale mortality estimates based on the national-scale risk assessment, showing the location of the urban case study areas within the overall national distribution.

3.3 REFERENCES

Abt Associates Inc. (2008). Environmental Benefits Mapping and Analysis Program (Version 3.0). Bethesda, MD. Prepared for Environmental Protection Agency, Office of Air Quality Planning and Standards, Air Benefits and Cost Group. Research Triangle Park, NC.

Folinsbee, L.J.; McDonnell, W.F.; Horstman, D.H. (1988). Pulmonary function and symptom responses after 6.6-hour exposure to 0.12 ppm O_3 with moderate exercise. *JAPCA.* 38: 28-35.

Henderson, R. 2007. Clean Air Scientific Advisory Committee's (CASAC) Review of the Agency's Final O_3 Staff Paper. EPA-CASAC-07-002. March 26.

Horstman, D.H.; Folinsbee, L.J.; Ives, P.J.; Abdul-Salaam, S.; McDonnell, W.F. (1990). O_3 concentration and pulmonary response relationships for 6.6-hr exposures with five hrs of moderate exercise to 0.08, 0.10, and 0.12 ppm. *Am Rev Respir Dis*. 142:1158-1163.

Kim, Chong S., Neil E. Alexis, Ana G. Rappold, Howard Kehrl, Milan J. Hazucha, John C. Lay, Mike T. Schmitt, Martin Case, Robert B. Devlin, David B. Peden, and David Diaz-Sanchez. "Lung Function and Inflammatory Responses in Healthy Young Adults Exposed to 0.06 ppm O_3 for 6.6 Hours." American Journal of Respiratory and Critical Care Medicine 183, no. 9 (2011): 1215-1221.

Langstaff, J.E. (2007). OAQPS Staff Memorandum to O_3 NAAQS Review Docket (EPA HQ-OAR-2005-0172). Subject: Analysis of Uncertainty in O_3 Population Exposure Modeling. [January 31, 2007]. Available at: http://www.epa.gov/ttn/naaqs/standards/O_3/s_O_3_cr_td.html

McDonnell, W.F. et al. (1991). Respiratory response of humans exposed to low levels of O_3 for 6.6 hours. *American Review of Respirtory Disease* 147:804-810.

McDonnell W.F., Stewart P.W., Smith M.V. (2007). The temporal dynamics of O_3 -induced FEV1 changes in humans: an exposure-response model. Inhal Toxicol 19:483–494.

McDonnell W.F., Stewart P.W., Smith M.V. (2010). Prediction of O_3 -induced lung function responses in humans. Inhal Toxicol. 22(2):160-8.

Samet, J. 2009. Consultation on EPA's Draft Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter. EPA-CASAC-10-004. December 3.

Samet, J. 2011. Consultation on EPA's O_3 National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment (April 2011) and O_3 National Ambient Air Quality Standards: Scope and Methods Plan for Welfare Risk and Exposure Assessment (April 2011). EPA-CASAC-11-008. June 21.

Schelegle, Edward S., Christopher A. Morales, William F. Walby, Susan Marion, and Roblee P. Allen. "6.6-Hour Inhalation of O_3 Concentrations from 60 to 87 Parts per Billion in Healthy Humans." American Journal of Respiratory and Critical Care Medicine 180 (2009): 265-272.

Simon, H., Baker, K., Possiel, N., Akhtar, F., Napelenok, S., Timin, B., Wells, B. (2012) Model-based rollback using the higher order direct decoupled method (HDDM). Available on the Internet at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_rea.html.

US EPA. 1996a. Review of National Ambient Air Quality Standards for O_3 : Assessment of Scientific and Technical Information - OAQPS Staff Paper. EPA/452/R-96-007. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available from: NTIS, Springfield, VA; PB96–203435. Available at:

http://www.epa.gov/ttn/naaqs/standards/O_3 /s_o3_pr.html

US EPA. 1996b. Air Quality Criteria for O_3 and Related Photochemical Oxidants. EPA/600/P-93/004aF-cF. Office of Research and Development, National Center for Environmental Assessment, Research Triangle Park, NC. Available at:

http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=2831.

US EPA. 2007a. O_3 Population Exposure Analysis for Selected Urban Areas. Office of Air Quality Planning and Standards, RTP, NC. EPA-452/R-07-010. July.

US EPA. 2007b. O_3 Health Risk Assessment for Selected Urban Areas. Office of Air Quality Planning and Standards, RTP, NC. EPA 452/R-07-009. July.

US EPA, 2007c. Review of the National Ambient Air Quality Standards for O_3: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper. Research Triangle Park, NC: Office of Air Quality Planning and Standards; report no. EPA-452/R-07-007a. Available at: http://www.epa.gov/ttn/naaqs/standards/O_3/s_O_3_cr_sp.html

US EPA. 2008a. *National Air Quality: Status and Trends Through 2007*. Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA-454/R-08-006. November.

Available at: http://www.epa.gov/airtrends/2008/index.html.

US EPA 2008b. *EPA's Report on the Environment.* Washington, DC EPA/600/R-07/045F. May 2008.

Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=190806

US EPA. 2009. Integrated Review Plan for the O_3 National Ambient Air Quality Standards Review: External Review Draft. Environmental Media Assessment Group, National Center for Environmental Assessment and Health and Environmental Impacts Division, Office of Air Quality Planning and Standards, RTP, NC. EPA 452/D-09-001. September.

US EPA. 2010. *Quantitative Health Risk Assessment for Particulate Matter*. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-10-005. Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf

US EPA, 2011a. Integrated Review Plan for the O_3 National Ambient Air Quality Standards. National Center for Environmental Assessment, Office of Research and Development and Office of Air Quality Planning and Standards, Office of Air and Radiation. RTP, NC. EPA 452/R-11-006. April.

US EPA, 2011b. O_3 National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment. Office of Air Quality Planning and Standards. RTP, NC. EPA-452/P-11-001. April.

US EPA, 2011c. O_3 National Ambient Air Quality Standards: Scope and Methods Plan for Welfare Risk and Exposure Assessment. Office of Air Quality Planning and Standards. RTP, NC. EPA-452/P-11-002. April.

Wegman, L. 2012. Updates to information presented in the Scope and Methods Plans for the O_3 NAAQS Health and Welfare Risk and Exposure Assessments. Memorandum from Lydia Wegman, Division Director, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards, Office of Air and Radiation, US EPA to Holly Stallworth, Designated Federal Officer, Clean Air Scientific Advisory Committee, US EPA Science Advisory Board Staff Office. May 2, 2012.

World Health Organization. 2008. Harmonization Project Document No. 6. Part 1: Guidance Document on Characterizing and Communicating Uncertainty in Exposure Assessment.

Available at: http://www.who.int/ipcs/methods/harmonization/areas/exposure/en/.

4 AIR QUALITY CONSIDERATIONS

4.1 INTRODUCTION

Air quality information is used in the risk and exposure analyses (Chapters 5-7) to assess risk and exposure resulting from recent O_3 concentrations, as well as to estimate the relative change in risk and exposure resulting from adjusted O_3 concentrations after simulating just meeting the current O_3 standard of 0.075 ppm. For the population exposure analyses discussed in Chapter 5, 16 urban areas will ultimately be modeled[1]. Four of these urban areas are modeled for this first draft REA, and as a result, air quality information from those 4 urban areas was analyzed for this first draft. The four urban areas evaluated for this first draft include Atlanta, GA; Denver, CO; Los Angeles, CA and Philadelphia, PA. The lung function risk assessment discussed in Chapter 6 uses the same air quality data as the population exposure assessment and models the same four urban areas for the first draft. For the epidemiology-based risk assessment discussed in Chapter 7, 12 of the 16 areas evaluated for population exposure are included, and air quality data for all 12 of these urban areas were analyzed. These 12 urban areas include the 4 cities evaluated in the first draft exposure assessment as well as: Baltimore, MD; Boston, MA; Cleveland, OH; Detroit, MI; Houston, TX; New York, NY; Sacramento, CA; and St. Louis, MO. In addition, Chapter 8 includes an assessment of the national-scale O_3 mortality risk burden based on national-scale air quality information. This chapter describes the air quality information used in these analyses, providing an overview of monitoring data and air quality (section 4.2) as well as an overview of air quality inputs to the risk and exposure assessments (section 4.3).

4.2 OVERVIEW OF OZONE MONITORING AND AIR QUALITY

To monitor compliance with the NAAQS, state and local environmental agencies operate O_3 monitoring sites at various locations, depending on the population of the area and typical peak O_3 concentrations (US EPA, 2012a, sections 3.5.6.1, 3.7.4). In 2010, there were 1,250 state and local O_3 monitors reporting concentrations to EPA (US EPA, 2012a, Figures 3-21 and 3-22). The minimum number of O_3 monitors required in a Metropolitan Statistical Area (MSA) ranges from zero, for areas with a population under 350,000 and with no recent history of an O_3 design value greater than 85% of the NAAQS, to four, for areas with a population greater than 10 million and an O_3 design value greater than 85% of the NAAQS.[2] In areas for which O_3

[1] These cities are Atlanta, GA; Baltimore, MD; Boston, MA; Chicago, IL; Cleveland, OH; Dallas, TX; Denver, CO; Detroit, MI; Houston, TX; Los Angeles, CA; New York, NY; Philadelphia, PA; Seattle, WA; Sacramento, CA; St. Louis, MO; and Washington, D.C.

[2]The current monitor and probe siting requirements have an urban focus and do not address siting in non-urban, rural areas. States may operate ozone monitors in non-urban or rural areas to meet other objectives (e.g., support for research studies of atmospheric chemistry or ecosystem impacts).

monitors are required, at least one site must be designed to record the maximum concentration for that particular metropolitan area. Since O_3 concentrations often decrease significantly in the colder parts of the year in many areas, O_3 is required to be monitored only during the "ozone season," which varies by state (US EPA, 2012a, section 3.5.6 and Figure 3-20).[3]

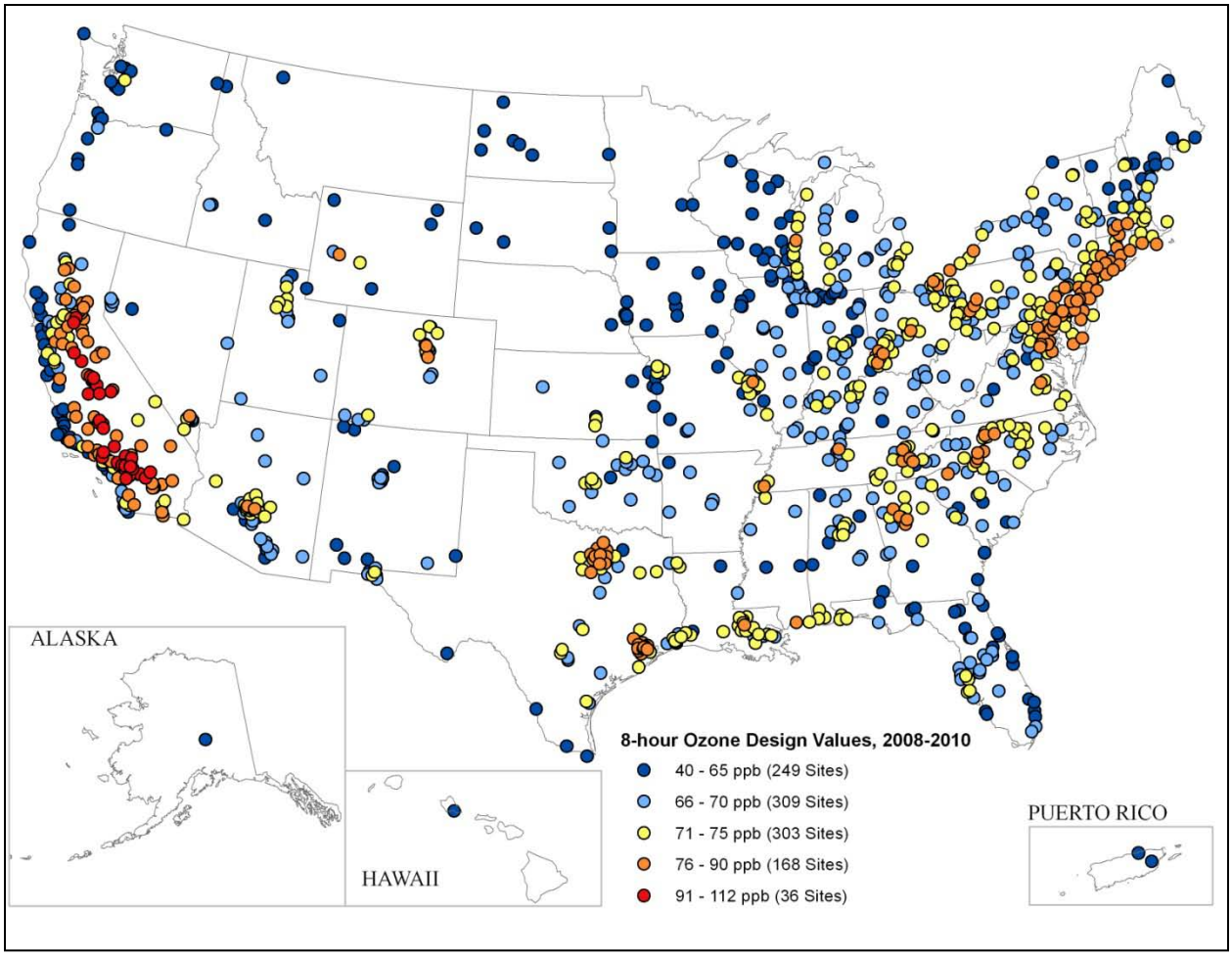

Figure 4-1 Individual monitor 8-h daily max O_3 design values displayed for the 2008-2010 period (U.S. EPA, 2012, Figure 3-52A)

Figure 4-1 shows the location and 8-h O_3 design values (3-year average of the annual 4th highest daily maximum 8-hour O_3 concentration) for all available monitors in the US for the 2008-2010 period. All 12 of the selected urban areas have 2008-2010 8-h O_3 design values at or above the current standard. Figure 4-2 shows how the 4th highest 8-h daily max O_3 concentrations vary for each of the 12 urban areas from 2006-2010. In general, all twelve cities

[3]Some States and Territories operate ozone monitors year-round, including Arizona, California, Hawaii, Louisiana, Nevada, New Mexico, Puerto Rico, Texas, American Samoa, Guam and the Virgin Islands.

show a decrease in O_3 concentrations between 2006 and 2010, with an average decrease in the 4th highest 8-h daily max O_3 concentration of 9 ppb. However, there is significant year-to-year variability, with some locations, such as Sacramento and Houston, showing increases in some years relative to 2006 even though the 2010 values are somewhat lower.

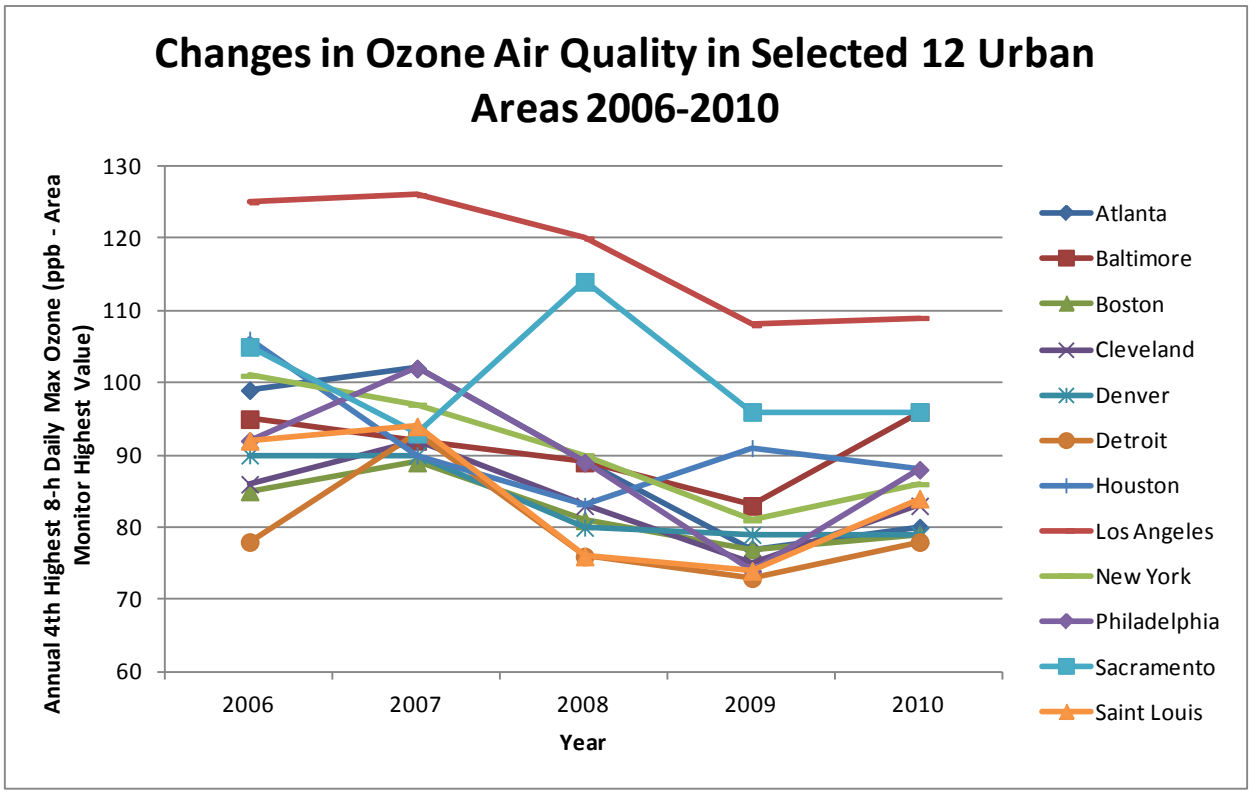

Figure 4-2 Trends in 8-h daily max O_3 for the selected 12 urban areas analyzed in the risk and exposure assessment for 2006-2010 (annual 4th highest 8-h daily max O_3 concentrations in ppm)

Table 4-1 gives the number of monitors and the required O_3 monitoring season for each of the 12 selected urban areas. The counties listed as part of each of the 12 urban areas are based on the counties included in the Zanobetti and Schwartz (2008) study of O_3 and mortality in 48 U.S. cities between 1989 and 2000, which is used in the epidemiology-based health risk assessment[4]. Also listed in Table 4-1 are the 8-h O_3 design values for 2006-2008 and 2008-2010. All of the cities, except for Sacramento (which showed no change), had a decrease in the O_3 design value concentrations between the two 3-year periods with an average change of 7 ppb.

[4] It should be noted that the counties included in Table 4-1 are those analyzed in the epidemiology-based risk assessment (Chapter 7) but differ from the counties included in the population exposure (Chapter 5) and the lung function risk assessment (Chapter 6). These differences are explained in Chapters 5-7.

Table 4-1: Information on the 12 Urban Case Study Areas in the Risk Assessment

Study Area	Counties[5]	Population (2010)	# of O_3 Monitors	Required O_3 Monitoring Season	2006-2008 (ppb)[6]	2008-2010 (ppb)[6]
Atlanta	Cobb County, GA DeKalb County, GA Fulton County, GA Gwinnett County, GA	3,105,873	5	March - October	95	80
Baltimore	Baltimore City, MD Baltimore County, MD	1,425,990	3	April - October	91	89
Boston	Middlesex County, MA Norfolk County, MA Suffolk County, MA	2,895,958	5	April - September	82	76
Cleveland	Cuyahoga County, OH	1,280,122	4	April - October	84	77
Denver	Denver County, CO	600,158	3	March - September	86	78
Detroit	Wayne County, MI	1,820,584	4	April - September	82	75
Houston	Harris County, TX	4,092,459	17	January - December	91	84
Los Angeles	Los Angeles County, CA	9,818,605	17	January - December	119	112
New York	Bronx County, NY Kings County, NY New York County, NY Queens County, NY Richmond County, NY	8,175,133	8	April - October	89	84
Philadelphia	Philadelphia County, PA	1,526,006	4	April - October	92	83
Sacramento	Sacramento County, CA	1,418,788	8	January - December	102	102
St. Louis	St. Louis City, MO St. Louis County, MO	1,318,248	8	April - October	85	77

[5] Counties listed here reflect those included in the Zanobetti and Schwartz (2008) study of ozone and mortality in 48 U.S. cities between 1989 and 2000.

[6] These are values of the highest 4th high 8-h max average (ppb) for the counties listed for each urban area. It should be noted that sometimes monitors with higher values occurred within the urban area but outside of the counties included in the Zanobetti and Schwartz (2008) study and those values are not included in this table.

4.3 OVERVIEW OF AIR QUALITY INPUTS TO RISK AND EXPOSURE ASSESSMENTS

The air quality information input into the risk and exposure assessments includes both recent air quality data from the years 2006-2010, as well as air quality data adjusted to reflect just meeting the current O_3 standard of 0.075 ppm. In this section, we summarize these air quality inputs and discuss the methodology used to simulate air quality to meet the current standard. Additional information is provided in Wells et al. (2012) and Simon et al. (2012).

4.3.1 Urban-scale Air Quality Inputs

4.3.1.1 Recent Air Quality

The air quality monitoring data used to inform the first draft Ozone Risk and Exposure Assessments were hourly O_3 concentrations collected between 1/1/2006 and 12/31/2010 from all US monitors meeting EPA's siting, method, and quality assurance criteria in 40 CFR Part 58. These data were extracted from EPA's Air Quality System (AQS) database[7] on June 27, 2011. Regionally concurred exceptional event data (i.e. data certified by the monitoring agency to have been affected by natural phenomena such as wildfires or stratospheric intrusions, and concurred upon by the EPA regional office) were not included in the assessments. However, concurred exceptional events were rare, accounting for less than 0.01% of the total observations. All concurred exceptional events in 2006-2010 were related to wildfires in California in 2008. There were no concurrences of exceptional event data for stratospheric intrusions in 2006-2010 in the data extracted on June 27, 2011.

In order to compare the monitoring data to the NAAQS, the data were split into two overlapping 3-year periods, 2006-2008 and 2008-2010. The O_3 monitors were checked for data completeness within each period, and all monitors lacking sufficient data to calculate a valid 3-year design value were excluded (see 40 CFR Part 50, Appendix P). All subsequent air quality data analyses described in this chapter were performed separately on the monitoring data within each of the two design value periods.

The sections below summarize the recent air quality data input into the epidemiological study-based risk assessment, and the exposure and clinical study-based risk assessment. More details on these inputs are also provided in Wells et al. (2012).

[7] EPA's Air Quality System (AQS) database is a state-of-the-art repository for many types of air quality and related monitoring data. AQS contains monitoring data for the six criteria pollutants dating back to the 1970's, as well as more recent additions such as air toxics, meteorology, and quality assurance data. At present, AQS receives ozone monitoring data collected hourly from over 1,300 monitors, and is quality assured by one of over 100 state, local, or tribal air quality monitoring agencies.

Epidemiology Based Risk Assessment

Air quality concentration data for the epidemiology-based risk analyses are input into the environmental Benefits Mapping and Analysis Program (BenMAP; Abt Associates, 2010a) for assessment. Gaps of 1 or 2 hours in the hourly concentration data were interpolated. These short gaps tend to occur at regular intervals in the monitoring data due to a requirement for monitoring agencies to turn off their monitors for brief periods in order to perform quality control checks. Generally, quality control checks are performed during nighttime hours (between 12:00 AM and 6:00 AM) when O_3 concentrations tend to be lowest. Missing intervals of 3 hours or more were infrequent and were not replaced.

The air quality monitoring data for the 12 urban areas were area-wide spatial averages of the hourly O_3 concentrations within each area. The area boundaries were chosen to match the study areas in Zanobetti & Schwartz (2008) which generally covered the urban population centers within the larger metropolitan areas. The ambient data from the monitors within each area were averaged hour-by-hour within EPA's required O_3 monitoring season. Although some monitoring data were collected outside of the required season, often fewer monitors in an area remained in operation outside of the required season.

For input into BenMAP, four daily metrics were calculated from the spatially averaged hourly O_3 concentrations. These metrics were:

1. Daily maximum 1-hour concentration
2. Daily maximum 8-hour concentration
3. Daytime 8-hour average concentration (10:00AM to 6:00PM)
4. Daily 24-hour average concentration

Exposure Modeling and Clinical Study Based Risk Assessment

For the exposure modeling and clinical study based risk assessment, the air quality data are input in the Air Pollutants Exposure (APEX) model, also referred to as the Total Risk Integrated Methodology Inhalation Exposure (TRIM.Expo) model (U.S. EPA, 2012b,c). For estimating ambient O_3 concentrations to use in the exposure model, we use hourly O_3 concentrations from the AQS. The specific monitors used in the urban areas modeled and the method for estimating and replacing missing data are described in Appendix 4-B.

4.3.1.2 Air Quality after Simulating "Just Meeting" Current O_3 Standard

In addition to recent air quality concentrations, the risk and exposure assessments also consider the relative change in risk and exposure when considering the distribution of O_3 concentrations after simulating "just meeting" the current O_3 standard of 0.075 ppm. The

sections below summarize the methodology applied for this first draft REA to simulate just meeting the current NAAQS by "rolling back" the baseline distribution of recent O_3 concentrations and an alternative simulation approach being considered for the 2nd draft of the REA. More details on these inputs are also provided in Wells et al. (2012), and a more complete description of the alternative simulation approach is provided in Simon et al. (2012).

Methods

The "quadratic rollback" method was used in the previous O_3 NAAQS review to adjust ambient O_3 concentrations to simulate minimally meeting current and alternative standards (U.S. EPA, 2007). As the name implies, quadratic rollback uses a quadratic equation to reduce high concentrations at a greater rate than low concentrations. The intent is to simulate reductions in O_3 resulting from unspecified reductions in precursor emissions, without greatly affecting concentrations near ambient background levels (Duff et al., 1998).

Two independent analyses (Johnson, 2002; Rizzo, 2005; 2006) were conducted to compare quadratic rollback with other methods such as linear (proportional) rollback and distributional (Weibull) rollback. Both analyses used different rollback methods to reduce concentrations from a high O_3 year to simulate levels achieved during a low O_3 year, then compared the results to the ambient concentrations observed during the low O_3 year. Both analyses concluded that the quadratic rollback method resulted in an 8-hour O_3 distribution most similar to that of the ambient concentrations.

In this review, quadratic rollback was used to simulate reductions in O_3 concentrations in areas which failed to meet EPA's current O_3 NAAQS of 0.075 ppm (75 ppb). Hourly O_3 concentrations were reduced so that the highest design value in each area was exactly 75 ppb, the highest value meeting the NAAQS. Concentrations at the remaining monitors in each area were similarly reduced using the quadratic rollback coefficients calculated at the highest monitor. Quadratic rollback was performed independently within each area for two design value periods, 2006-2008 and 2008-2010. In some of the 12 urban areas, the monitor with the highest design value was not within the area boundaries chosen to match the study areas in Zanobetti & Schwartz (2008). In these cases, the high monitor was included in the quadratic rollback, and the ozone concentrations at the monitors within the Zanobetti & Schwartz (2008) study area were similarly reduced. In this way, while the high monitor outside of the study area would have been simulated to have a design value of 75 ppb to just meet the standard, the design value at the monitors within the study area would have been simulated to have design values below 75 ppb.

To avoid reducing O_3 concentrations below background levels, background "floor" values were set defining minimum values beyond which quadratic rollback would not be applied.

Background concentrations were estimated from two GEOS-Chem modeling simulations for the model year of 2006: one with zero U.S. anthropogenic emissions (i.e. U.S. background) but with all other anthropogenic and natural emissions globally, and the other with all anthropogenic and biogenic emissions included (i.e. base case) (Zhang et al., 2011). The monitors in each study area were paired with their appropriate GEOS-Chem grid cells, potentially matching multiple monitors to the same cell. The paired hourly U.S. background and base case concentrations were then spatially averaged in the same way as the O_3 monitoring data (as described in 4.3.1.1). Medians by area, month, and hour of the day were calculated from the spatially-averaged U.S. background and base case modeled concentrations, and ratios of the U.S. background to base case concentrations were calculated to provide monthly diurnal profiles of the ratio of U.S. background to total ozone for every month for every area[8]. Next, the U.S. background ratios were multiplied by the respective monitored values in each of the 5 years, 2006-2010, to obtain the U.S. background floor values.

The U.S. background floor values were compared to the hourly "rolled back" air quality values for each area. If there was an hour for which the O_3 concentration had been "rolled back" to below the U.S. background floor value, then that hourly concentration value was set equal to whichever was lower: the U.S. background floor value or to the original monitored O_3 concentration value for that hour.

Figure 4-3 shows diurnal profiles of seasonally averaged U.S. background floor values for the 12 urban case study areas in the risk assessment. The U.S. background floor values show a diurnal pattern similar to that of the observed O_3 concentrations, with the highest values occurring in the early afternoon hours and the lowest values occurring around sunrise. Generally, the highest U.S. background values occurred in the spring, while the other three seasons were more difficult to distinguish. Denver was a notable exception to this pattern, having nearly identical U.S. background floor values in the spring and summer months.

Figure 4-4 shows box-and-whisker plots of the U.S. background floor values in the 12 urban case study areas. The distribution of the U.S. background floor values varied from area to area, but generally ranged from near 0 to between 30 and 40 ppb, with median between 10 and 20 ppb.

[8] Values were set equal to one, if greater than one.

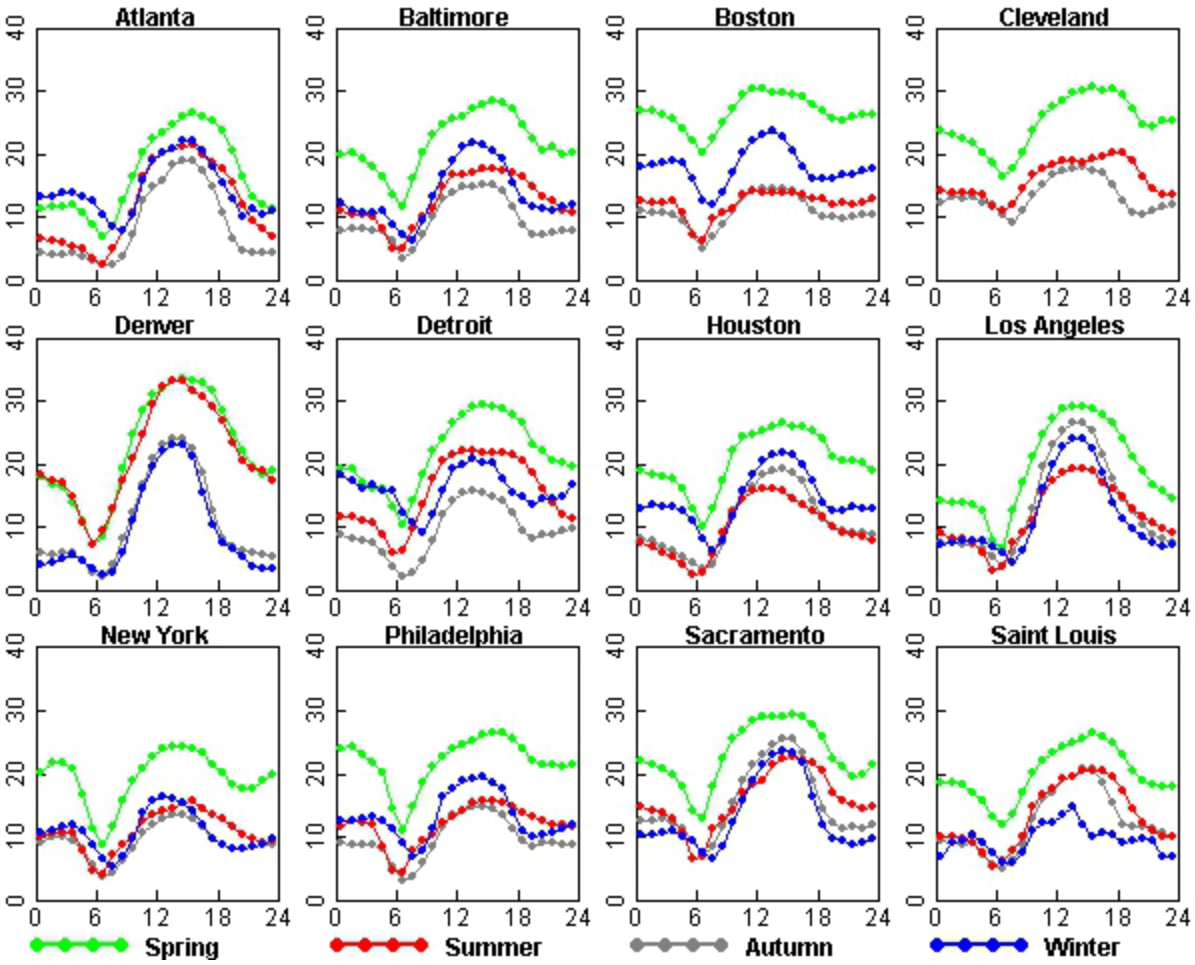

Figure 4-3 Diurnal Profiles of Seasonally Averaged U.S. Background Floor Values in the Urban Case Study Areas

Notes: Values shown are 2006-2010 averages, in parts per billion. Seasons were defined as Spring = March – May, Summer = June – August, Autumn = September – November, Winter = December – February. Winter values are missing for Cleveland because no monitoring data were available for that period.)

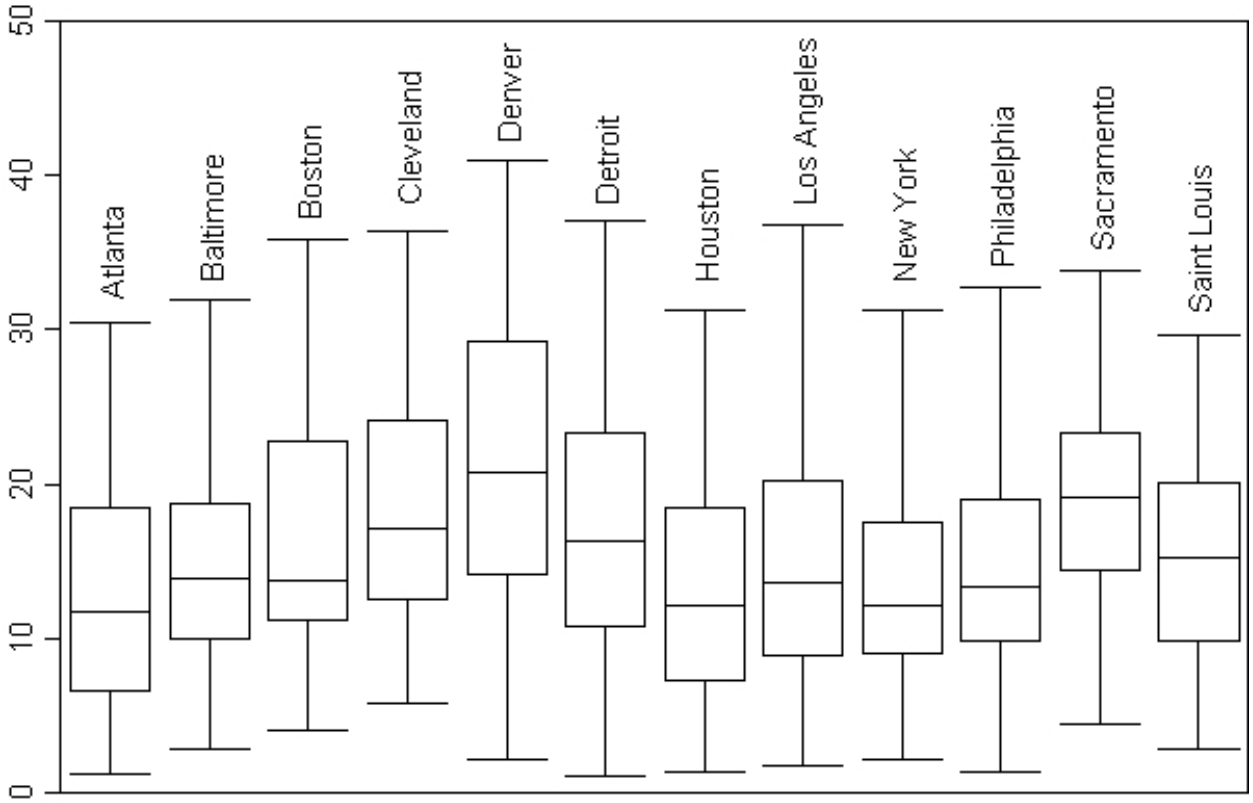

Figure 4-4 Distribution of U.S. Background Floor Values in the Urban Case Study Areas

Table 4-2 contains selected summary statistics generated to evaluate the frequency and magnitude of the U.S. background adjustments in the quadratic rollback procedure. Overall, over 20% of the rollback concentrations were adjusted, however, the average magnitude of the adjustments was very small (< 0.2 ppb), and even the largest adjustment was less than 5 ppb. Over 95% of the adjustments simply returned the rollback concentrations to their original monitored values instead of the modeled U.S background value, and again the average magnitude of the adjustment was very small (< 0.2 ppb). In conclusion, the U.S. background adjustment procedure had little effect on the rollback concentrations.

Table 4-2 Frequency and Magnitude of the U.S. Background Adjustments, 2006 – 2008

Urban Area	% Rollback Values Adjusted	% Replaced with Monitor Values	% Replaced with Floor Values	Average Adjustment (ppb)	Maximum Adjustment (ppb)
Atlanta	16.7	97.2	2.8	0.10	2.3
Baltimore	19.7	96.8	3.2	0.15	2.2
Boston	16.4	96.3	3.7	0.17	1.2
Cleveland	20.0	96.2	3.8	0.18	1.6

Denver	14.4	96.2	3.8	0.20	2.4
Detroit	14.9	96.8	3.2	0.13	1.3
Houston	28.4	96.4	3.6	0.15	1.6
Los Angeles	24.6	93.9	6.1	0.29	4.5
New York	16.4	96.7	3.3	0.09	1.4
Philadelphia	18.7	96.2	3.8	0.16	2.0
Sacramento	24.3	92.1	7.9	0.34	3.0
Saint Louis	12.8	97.1	2.9	0.11	1.1
OVERALL	20.5	95.5	4.5	0.17	4.5

Figure 4-5 shows seasonal average diurnal profiles of the observed and rollback composite monitor values in the 12 urban case study areas for 2006-2008. The gray and blue lines are averages over the required O_3 monitoring season (see Table 4-1), while the red and green lines are averages over the "peak" O_3 months, June – August. The June – August averages are higher than the O_3 season averages, except in Houston where the highest O_3 concentrations are often observed in April-May and September-October. The diurnal patterns are generally quite similar from area to area, with most of the variation occurring in the peak concentration heights during the daytime hours.

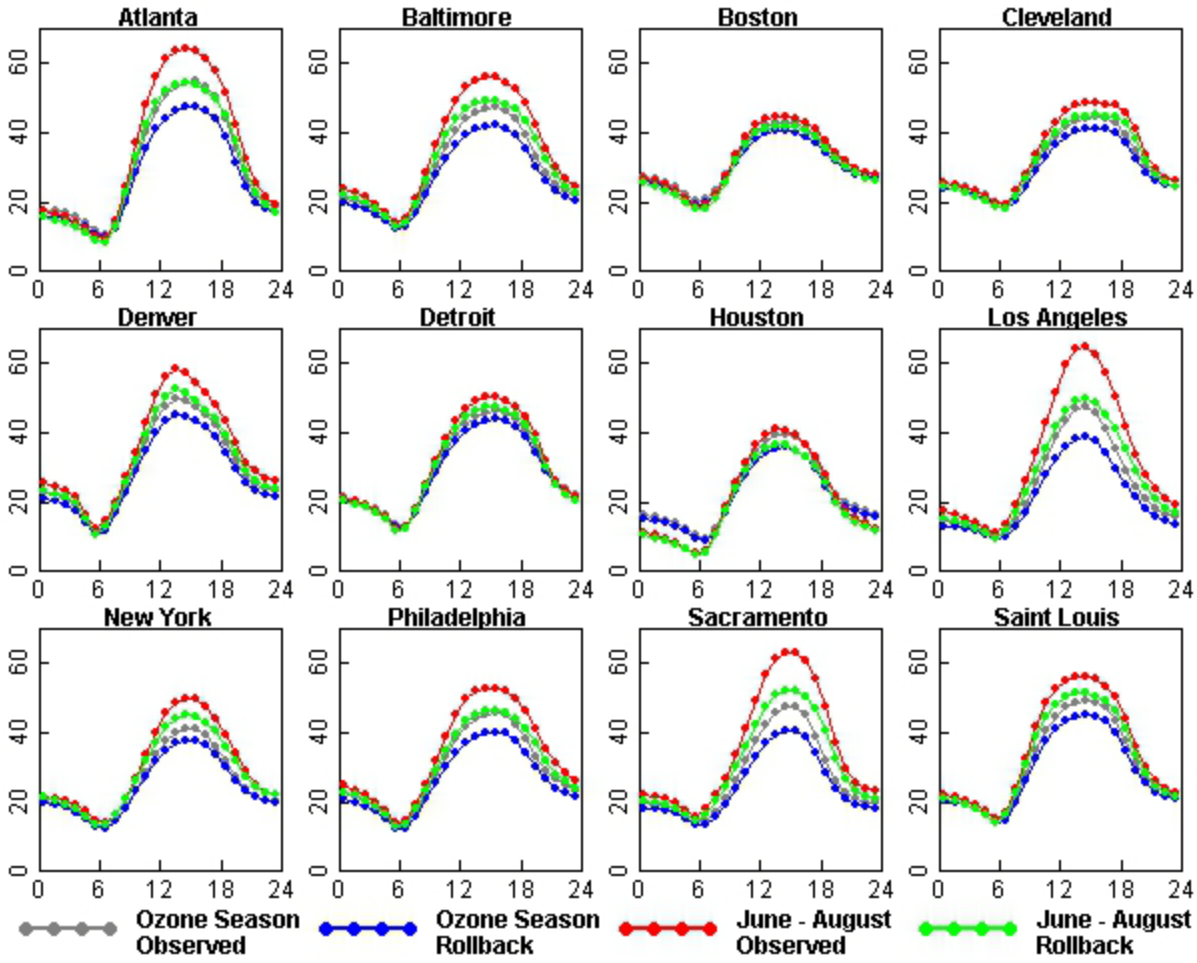

Figure 4-5 Diurnal Profiles of Seasonally Averaged Composite Monitor Values in the Urban Case Study Areas, 2006-2008

Future Directions for Rollback

As described above, for this first draft REA we are using the same quadratic rollback method applied in the previous review. Based on the current understanding of how O_3 forms and reacts to changes in emissions, reductions in emissions that would be needed to meet the current standards are likely to lead to reductions in hourly concentrations for most hours of the day, but these reductions may have little impact on concentrations for some hours, and in some cases can lead to increases in O_3 concentrations, particularly during nighttime hours. The quadratic rollback method has difficulty representing these complexities in O_3 chemistry and reduces O_3 concentrations over all hours; it assumes that all monitors in an area exhibit the same response to emissions changes. (Wells et al., 2012). To address this issue in the rollback methodology for this first draft REA, we imposed a lower bound on O_3 concentration values based on modeled U.S. background O_3 levels.

For this first draft of the REA, we have evaluated approaches for simulating attainment of current and alternative standards that are based on modeling the response of O_3 concentrations to reductions in anthropogenic NO_x and VOC emissions, using the Higher-Order Decoupled Direct Method (HDDM) capabilities in the Community Multi-scale Air Quality (CMAQ) model. This modeling incorporates all known emissions, including emissions from non-anthropogenic sources and anthropogenic emissions from sources in and outside of the U.S. As a result, the need to specify values for U.S. background concentrations is not necessary, as it is incorporated in the modeling directly. In simulations of just meeting the standards used to inform the exposure and risk assessment, HDDM sensitivities can be applied relative to ambient measurements of O_3 to estimate how ozone concentrations would respond to changes in anthropogenic emissions within the U.S. Application of this approach also addresses the recommendation by the National Research Council of the National Academies (NRC, 2008) to explore how emissions reductions might effect temporal and spatial variations in O_3 concentrations, and to include information on how NO_x versus VOC control strategies might affect risk and exposure to O_3. The new approach using HDDM, discussed in detail in Simon et al., 2012, seems promising, and EPA staff propose to use it in simulating just meeting the current and alternative O_3 standards for the second draft of the REA.

4.3.2 National-scale Air Quality Inputs

In contrast to the urban study areas analysis, the national-scale analysis employs a data fusion approach that takes advantage of the accuracy of monitor observations and the comprehensive spatial information of the CMAQ modeling system to create a national-scale "fused" spatial surface of seasonal average O_3. The spatial surface is created by fusing 2006-2008 measured O_3 concentrations with the 2007 CMAQ model simulation, which was run for a 12 km gridded domain, using the EPA's Model Attainment Test Software (MATS; Abt Associates, 2010b), which employs the Voronoi Neighbor Averaging (VNA) technique (Timin et al., 2010) enhanced with information on the spatial gradient of O_3 provided by CMAQ results. More details on the ambient measurements and the 2007 CMAQ model simulation, as well as the spatial fusion technique, can be found in Wells et al. (2012) and Hall et al. (2012). It should also be noted that this same spatial fusion technique was employed for a national-scale risk assessment by Fann et al. (2012) to produce "fused" spatial fields for O_3 and $PM_{2.5}$ and in the PM NAAQS REA to produce a national-scale spatial field for $PM_{2.5}$ (U.S. EPA, 2010).

Two "fused" spatial surfaces were created for: (1) the May-September mean of the 8-hr daily maximum (consistent with the metric used by Bell et al. 2004); and (2) the June-August mean of the 8-hr daily mean from 10am to 6pm (consistent with the metric used by Zanobetti and Schwartz 2008) O_3 concentrations across the continental U.S. Figure 4-6 and Figure 4-7

show the geographic distribution of these spatial surfaces. Figure 4-8 shows the frequency and cumulative percent of the seasonal average O_3 concentrations by grid cell, using both metrics. May-September average 8-hr daily maximum concentrations are most frequently in the 40-50 ppb range, while June-August average 8-hr daily mean concentrations are more evenly distributed across a range of 20-70 ppb. Maximum concentrations for the June-August mean of the 8-hr daily mean concentrations from 10am to 6pm are generally higher than for the May-September mean of the 8-hr daily maximum concentrations since the seasonal definition is limited to the summer months when O_3 tends to be highest. The maximum, minimum, mean, median, and 95th percentile concentrations for both 8-hr daily maximum and 8-hr daily mean are shown in Table 4-3. These seasonal average metrics are not equivalent to the averaging time for the current NAAQS, which is based on the 4th highest value rather than seasonal mean, so the values should not be directly compared against the NAAQS.

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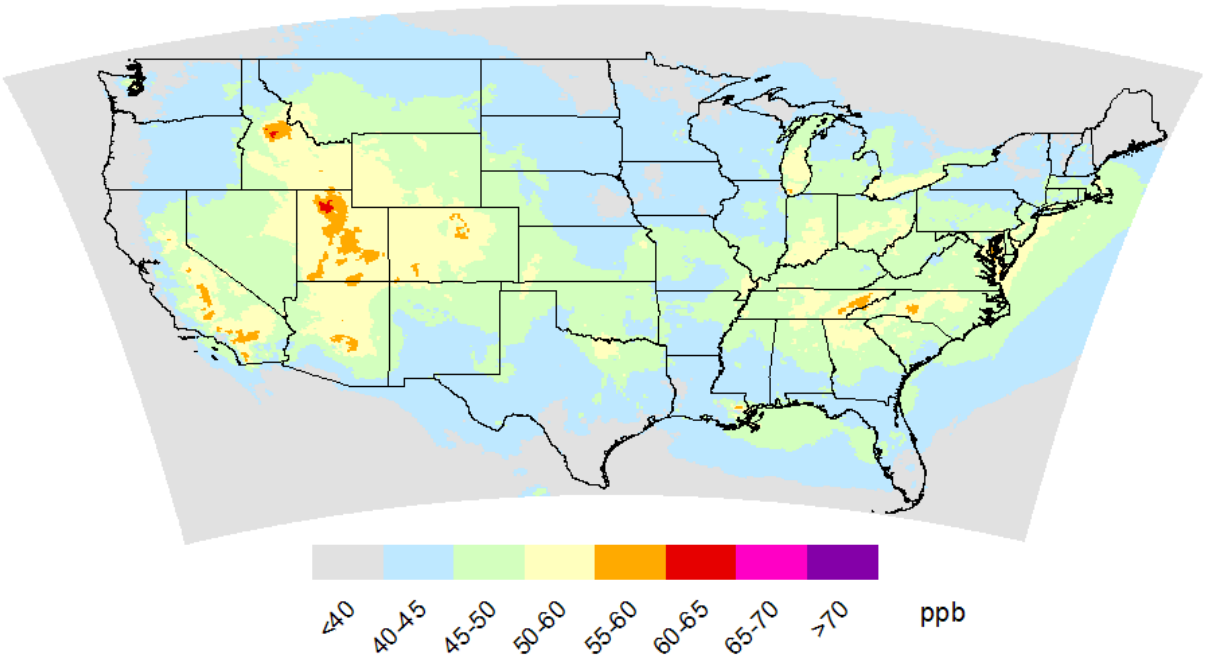


Figure 4-6 **Seasonal (May-September) average 8-hr. daily maximum baseline O_3 concentrations (ppb) at the surface, based on a 2007 CMAQ model simulation fused with average 2006-2008 observations from the O_3 monitor network.**

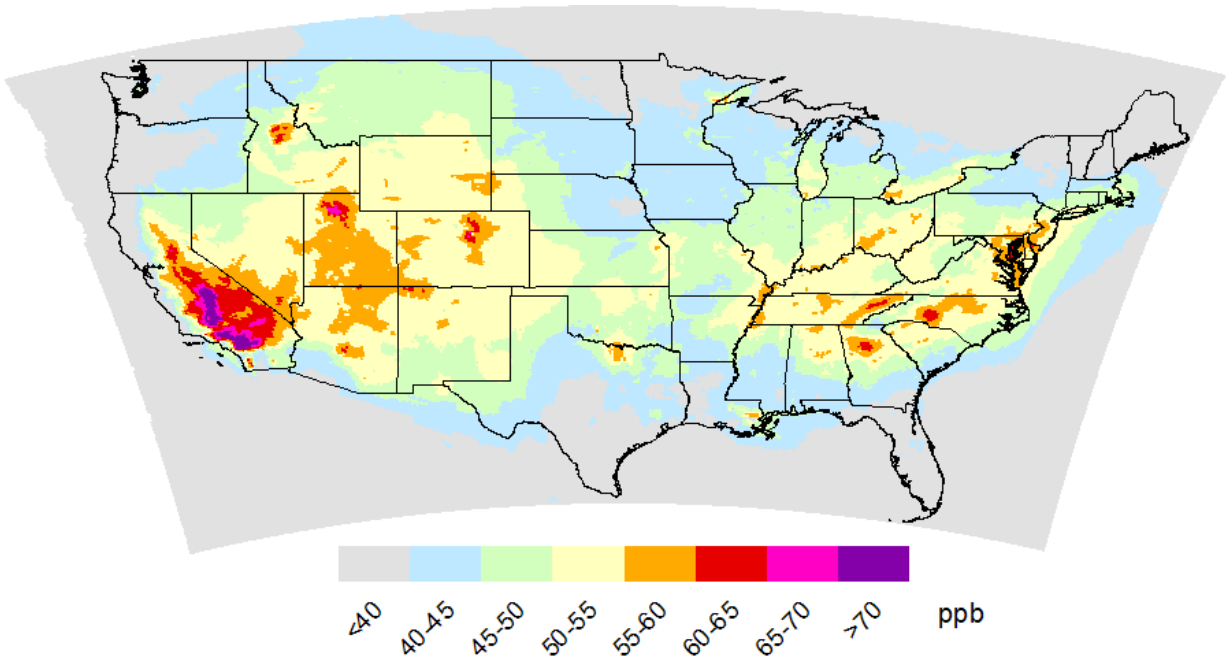


Figure 4-7 **Seasonal (June-August) average 8-hr. daily mean (10am-6pm) baseline O_3 concentrations (ppb) at the surface, based on a 2007 CMAQ model simulation fused with average 2006-2008 observations from the O_3 monitor network.**

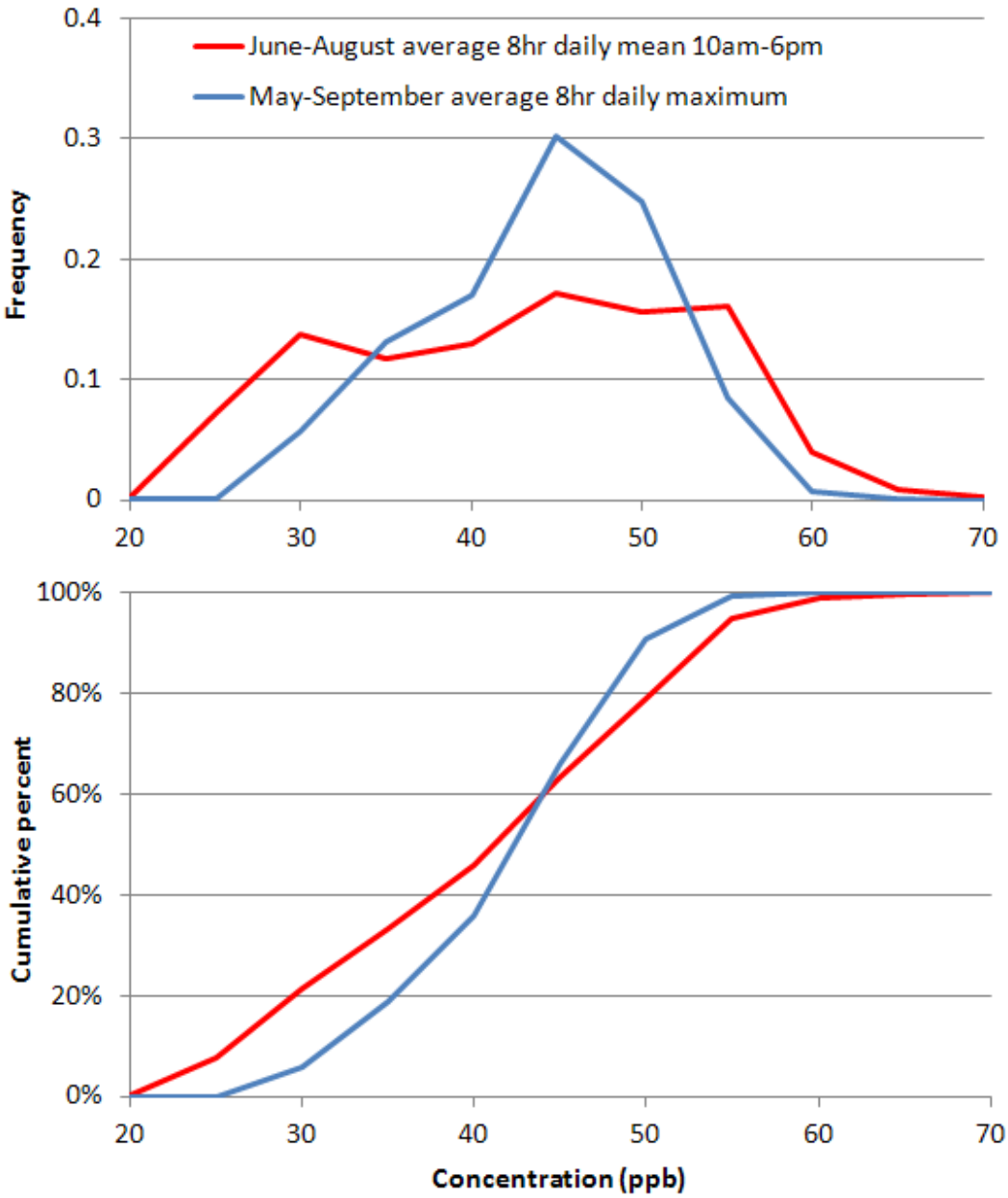


Figure 4-8 **Frequency and cumulative percent of May-September average 8-hr daily maximum and the June-August 8-hr daily mean (10am-6pm) O_3 concentration (ppb) by gridcell, based on 2006-2008 monitor observations fused with 2007 CMAQ-modeled O_3 levels.**

Table 4-3 Statistical characterization of the May-September average 8-hr daily maximum and the June-August 8-hr daily mean (10am-6pm) O_3 concentration (ppb), based on 2006-2008 monitor observations fused with 2007 CMAQ-modeled O_3 levels.

	May-September average 8-hr daily maximum concentration (ppb)	June-August average daily 10am – 6pm daily mean concentration (ppb)
Maximum	65.0	85.5
Minimum	19.7	18.0
Mean	41.8	40.4
Median	42.6	41.3
95^{th} Percentile	51.6	55.1

4.4 REFERENCES

Abt Associates, Inc. (2010a). Environmental Benefits and Mapping Program (Version 4.0). Bethesda, MD. Prepared for U.S. Environmental Protection Agency Office of Air Quality Planning and Standards. Research Triangle Park, NC. Available on the Internet at <http://www.epa.gov/air/benmap>.

Abt Associates, Inc. (2010b). Model Attainment Test Software (Version 2). Bethesda, MD. Prepared for the U.S. Environmental Protection Agency Office of Air Quality Planning and Standards. Research Triangle Park, NC. Available on the Internet at: http://www.epa.gov/scram001/modelingapps.mats.htm.

Bell, M.L., A. McDermott, S.L. Zeger, J.M. Samet, F. Dominici. (2004). Ozone and short-term mortality in 95 US urban communities, 1987-2000. JAMA, 292:2372-2378.

Duff, M., Horst, R. L., Johnson, T. R. (1998). Quadratic Rollback: A Technique to Model Ambient Concentrations Due to Undefined Emission Controls. Presented at the Air and Waste Management Annual Meeting, San Diego, CA. June 14-18, 1998.

Fann N, Lamson AD, Anenberg SC, Wesson K, Risley D, Hubbell BJ. (2012). Estimating the national public health burden associated with exposure to ambient $PM_{2.5}$ and ozone. Risk Analysis, 32:81-95.

Hall, E., Eyth, A., Phillips, S. (2012) Hierarchical Bayesian Model (HBM)-Derived Estimates of Air Quality for 2007: Annual Report. EPA/600/R-12/538. Available on the Internet at: http://www.epa.gov/heasd/sources/projects/CDC/AnnualReports/2007_HBM.pdf

Johnson, T. (2002). A Guide to Selected Algorithms, Distributions, and Databases Used in Exposure Models Developed by the Office of Air Quality Planning and Standards. Prepared by TRJ Environmental, Inc. for U.S. Environmental Protection Agency, Office of Research and Development, Research Triangle Park, NC.

National Research Council of the National Academies (2008). Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. The National Academies Press, Washington, D.C.

Rizzo, M. (2005). A Comparison of Different Rollback Methodologies Applied to Ozone Concentrations. November 7, 2005. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html

Rizzo, M. (2006). A Distributional Comparison between Different Rollback Methodologies Applied to Ambient Ozone Concentrations. May 31, 2006. Available on the Internet: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html

Simon, H., Baker, K., Possiel, N., Akhtar, F., Napelenok, S., Timin, B., Wells, B. (2012) Model-based rollback using the higher order direct decoupled method (HDDM). Available on the Internet at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_rea.html.

Timin B, Wesson K, Thurman J. Application of Model and Ambient Data Fusion Techniques to Predict Current and Future Year $PM_{2.5}$ Concentrations in Unmonitored Areas. (2010). Pp. 175-179 in Steyn DG, Rao St (eds). Air Pollution Modeling and Its Application XX. Netherlands: Springer.

U.S. EPA, 2007. Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper. U.S. Environmental Protection Agency

Office of Air Quality Planning and Standards. Research Triangle Park, North Carolina. EPA-452/R-07-007.U.S. Environmental Protection Agency. (2012a). Integrated Science Assessment for Ozone and Related Photochemical Oxidants: Third External Review Draft, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA/600/R-10/076C

U.S. Environmental Protection Agency (2012b). Total Risk Integrated Methodology (TRIM) - Air Pollutants Exposure Model Documentation (TRIM.Expo / APEX, Version 4.4) Volume I: User's Guide. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA-452/B-12-001a. Available at: http://www.epa.gov/ttn/fera/human_apex.html

U.S. Environmental Protection Agency (2012c). Total Risk Integrated Methodology (TRIM) - Air Pollutants Exposure Model Documentation (TRIM.Expo / APEX, Version 4.4) Volume II: Technical Support Document. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA-452/B-12-001b. Available at: http://www.epa.gov/ttn/fera/human_apex.html

Wells, B., Wesson, K., Jenkins, S. (2012). Analysis of Recent U.S. Ozone Air Quality Data to Support the O3 NAAQS Review and Quadratic Rollback Simulations to Support the First Draft of the Risk and Exposure Assessment. Available on the Internet at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_rea.html

Zhang, L., D.J. Jacob, N.V. Smith-Downey, D.A. Wood, D. Blewitt, C.C. Carouge, A. van Donkelaar, D.B. A. Jones, L.T. Murray, Y. Wang. (2011). Improved estimate of the policy-relevant background ozone in the United States using the GEOS-Chem global model with 1/2°x2/3° horizontal resolution over North America. Atmos Environ, 45:6769-6776.

Zanobetti, A., and J. Schwartz. (2008). Mortality displacement in the association of ozone with mortality: An analysis of 48 cities in the United States. Am J Resp Crit Care Med, 177:184-189.

5 CHARACTERIZATION OF HUMAN EXPOSURE TO OZONE

5.1 INTRODUCTION

As part of the last O_3 NAAQS review, EPA conducted exposure analyses for the general population, all school-age children (ages 5-18), active school-age children, and asthmatic school-age children (EPA, 2007a,b). Exposure estimates were generated for 12 urban areas for recent years of air quality and for just meeting the existing 8-hr standard and several alternative 8-hr standards. EPA also conducted a health risk assessment that produced risk estimates for the number of children and percent of children experiencing impaired lung function and other respiratory symptoms associated with the exposures estimated for these same 12 urban areas.

The exposure analysis conducted for the current review builds upon the methodology and lessons learned from the exposure analyses conducted in previous reviews (U.S. EPA, 1996a, 2007a,b), as well as information provided in the third draft ISA (EPA, 2012a). EPA will be conducting exposure modeling for 16 urban areas located across the U.S., listed in Table 5-3). In this first draft REA, results are presented for four of these areas, Atlanta, Denver, Los Angeles, and Philadelphia.

Population exposures to ambient O_3 levels are modeled using the Air Pollutants Exposure (APEX) model, also referred to as the Total Risk Integrated Methodology Inhalation Exposure (TRIM.Expo) model (U.S. EPA, 2012b,c). Exposure estimates are developed for O_3 levels in recent years, based on 2006 to 2010 ambient air quality measurements. Exposures are also estimated for O_3 levels associated with just meeting the current 8-hr O_3 NAAQS, based on adjusting data derived from the ambient monitoring network as described in Chapter 4 with additional details in Wells et al. (2012). Exposures are modeled for 1) the general population, 2) school-age children (ages 5-18), and 3) asthmatic school-age children. The strong emphasis on children reflects the finding of the last O_3 NAAQS review (EPA, 2007a) and the ISA (EPA, 2012a, Chapter 8) that children are an important at-risk group.

This chapter provides a brief overview of the types of studies that provide data on which this analysis is based, followed by a description of the exposure model used for this analysis, the model input data, and the results of the analysis. The final sections of this chapter summarize the sensitivity analyses and model evaluation that have been conducted for the APEX exposure model, and plans for additional analyses to be included in the second draft REA.

5.2 OZONE EXPOSURE STUDIES

Many studies have produced information and data supporting the development of methods for estimating human exposure to ambient O_3 over the past several decades. These studies have been reviewed in the ISA and previous EPA Ozone Air Quality Criteria Documents (U.S. EPA, 1986, 1996b, 2006, 2012a). The types of studies which provide the basis for modeling human exposure to O_3 include studies of people's activities, work and exercise patterns, physiology, physics and O_3-related chemistry in microenvironments, atmospheric modeling of O_3, chamber studies of atmospheric chemistry, and modeling of meteorology. Measurements that have proven to be useful for understanding and estimating exposure obtained from personal exposure assessment studies include fixed-site ambient concentrations, concentrations in specific indoor and outdoor microenvironments, personal exposure levels, personal activity patterns, air exchange rates, infiltration rates, deposition and decay rates, and meteorology.

Exposure Concepts and Definitions

Human exposure to a contaminant is defined as "contact at a boundary between a human and the environment at a specific contaminant concentration for a specific interval of time," and has units of concentration times duration (National Research Council, 1991). For airborne pollutants the contact boundary is nasal and oral openings in the body, and *personal exposure* of an individual to a chemical in the air for a discrete time period is quantified as (Lioy, 1990; National Research Council, 1991):

$$E_{[t_1, t_2]} = \int_{t_1}^{t_2} C(t)dt \qquad (4\text{-}1)$$

where $E_{[t_1,t_2]}$ is the personal exposure during the time period from t_1 to t_2, and $C(t)$ is the concentration at time t in the breathing zone. We refer to the *exposure concentration* to mean the concentration to which one is exposed. The breathing rate (ventilation rate) at the time of exposure is an important determinant of the dose received by the individual. Although we do not estimate dose, we refer to *intake* as the total amount of O_3 inhaled (product of exposure concentration, duration, and minute ventilation rate).

Personal exposure to O_3 can be estimated directly by monitoring the concentration of O_3 in the person's breathing zone (close to the nose/mouth) using a personal exposure monitor.

Exposure can also be estimated indirectly, by estimating or monitoring the concentrations over time in locations in which the individual spends time and estimating the time and duration the individual spends in each location, as well as the level of activity and resulting ventilation rate. In both of these methods, Equation 4-1 is used to calculate an estimate of personal exposure. A key concept in modeling exposure is the *microenvironment*, a term that refers to the immediate surroundings of an individual. A microenvironment is a location in which pollutant concentrations are relatively homogeneous for short periods of time. Microenvironments can be outdoors or indoors; some examples are outdoors near the home, outdoors near the place of work, bedrooms, kitchens, vehicles, stores, restaurants, street-corner bus stops, schools, and places of work. A bedroom may be treated as a different microenvironment than a kitchen if the concentrations are significantly different in the two rooms. The concentrations in a microenvironment typically change over time; for example, O_3 concentrations in a kitchen while cooking with a gas stove may be lower than when these activities are not being performed, due to scavenging of O_3 by nitric oxide (NO) emissions from the gas burned.

An important factor affecting the concentrations of O_3 indoors is the degree to which the ambient outdoor air is transported indoors. This can be modeled using physical factors such as air exchange rates (AERs), deposition and decay rates, and penetration factors. The *volumetric exchange rate* (m^3/hour) is the rate of air exchange between the indoor and outdoor air. The *AER* between indoors and outdoors is the number of complete air exchanges per hour and is equal to the volumetric exchange rate divided by the volume of the well-mixed indoor air. Indoor concentrations of O_3 can be decreased by uptake of O_3 by surfaces and by chemical reactions. The *deposition* and *chemical decay rates* are the rates (per hour) at which O_3 is removed from the air by surface uptake and chemical reactions. Some exposure models employ an infiltration factor, which is conceptually useful if distinguishing between the air exchange processes of air blowing through open doors and windows and the infiltration of air through smaller openings. Since measurements of AERs account for both of these processes (including infiltration), this distinction is not useful in applied modeling of O_3 exposures and will not be discussed further here. Simpler exposure models use a "factor model" approach to estimate indoor O_3 concentrations by multiplying the ambient outdoor concentrations by an indoor/outdoor concentration ratio, referred to as a *penetration factor*.

5.3 EXPOSURE MODELING

Models of human exposure to airborne pollutants are typically driven by estimates of ambient outdoor concentrations of the pollutants, which vary by time of day as well as by location. These outdoor concentration estimates may be provided by measurements, by air quality models, or by a combination of these. Simulations of scenarios where current or alternative ozone standards are just met require some form of modeling. The main purpose of this exposure analysis is to allow comparisons of population exposures to O_3 within each urban area, associated with recent air quality levels and with several potential alternative air quality standards or scenarios. Human exposure, regardless of the pollutant, depends on where an individual is located and what they are doing. Inhalation exposure models are useful in realistically estimating personal exposures and intake based on activity-specific ventilation rates, particularly when recognizing that these measurements cannot be performed for a given population. This section provides a brief overview of the model used by EPA to estimate O_3 population exposure. A more detailed technical description of APEX is provided in Appendix 5A.

5.3.1 The APEX Model

The EPA has developed the APEX model for estimating human population exposure to criteria and air toxic pollutants. APEX also serves as the human inhalation exposure model within the Total Risk Integrated Methodology (TRIM) framework (Richmond et al., 2002; EPA 2012b,c). APEX is conceptually based on the probabilistic NAAQS Exposure Model (pNEM) that was used in the 1996 O_3 NAAQS review (Johnson et al., 1996a; 1996b; 1996c). Since that time the model has been restructured, improved, and expanded to reflect conceptual advances in the science of exposure modeling and newer input data available for the model. Key improvements to algorithms include replacement of the cohort approach with a probabilistic sampling approach focused on individuals, accounting for fatigue and oxygen debt after exercise in the calculation of ventilation rates, and new approaches for construction of longitudinal activity patterns for simulated persons. Major improvements to data input to the model include updated AERs, census and commuting data, and the daily time-activities database. These improvements are described later in this chapter.

APEX is a probabilistic model designed to account for the numerous sources of variability that affect people's exposures. APEX simulates the movement of individuals through

time and space and estimates their exposure to a given pollutant in indoor, outdoor, and in-vehicle microenvironments. The model stochastically generates simulated individuals using census-derived probability distributions for demographic characteristics. The population demographics are drawn from the year 2000 Census at the tract level, and a national commuting database based on 2000 census data provides home-to-work commuting flows between tracts.[1] Any number of simulated individuals can be modeled, and collectively they approximate a random sampling of people residing in a particular study area.

Daily activity patterns for individuals in a study area, an input to APEX, are obtained from detailed diaries that are compiled in the Consolidated Human Activity Database (CHAD) (McCurdy et al., 2000; EPA, 2002). The diaries are used to construct a sequence of activity events for simulated individuals consistent with their demographic characteristics, day type, and season of the year, as defined by ambient temperature regimes (Graham & McCurdy, 2004). The time-location-activity diaries input to APEX contain information regarding an individuals' age, sex, race, employment status, occupation, day-of-week, daily maximum hourly average temperature, the location, start time, duration, and type of each activity performed. Much of this information is used to best match the activity diary with the generated personal profile, using age, sex, employment status, day of week, and temperature as first-order characteristics. The approach is designed to capture the important attributes contributing to an individuals' behavior, and of particular relevance here, time spent outdoors (Graham and McCurdy, 2004). Furthermore, these diary selection criteria give credence to the use of the variable data that comprise CHAD (e.g., data collected were from different seasons, different states of origin, etc.). Contributing to the uncertainty of the simulated diary sequences is that the approach for creating year-long activity sequences is based on a cross-sectional activity data base of 24-hour records. The typical subject in the time/activity studies in CHAD provided less than 2 days of diary data. 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 series of hourly exposures spanning the time period of interest.

APEX has a flexible approach for modeling microenvironmental concentrations, where the user can define the microenvironments to be modeled and their characteristics. Typical indoor microenvironments include residences, schools, and offices. Outdoor microenvironments

[1] There are approximately 65,400 census tracts in the ~3,200 counties in the U.S.

include near roadways, at bus stops, and playgrounds. Inside cars, trucks, and mass transit vehicles are microenvironments which are classified separately from indoors and outdoors.

Activity-specific simulated breathing rates of individuals are used in APEX to characterize intake received from an exposure. These breathing, or ventilation, rates are derived from energy expenditure estimates for each activity included in CHAD and are adjusted for age- and sex-specific physiological parameters associated with each simulated individual. Energy expenditure estimates themselves are derived from METS (metabolic equivalents of work) distributions associated with every activity in CHAD (McCurdy et al., 2000), largely based upon the Ainsworth et al. (1993) "Compendium of Physical Activities." METS are a dimensionless ratio of the activity-specific energy expenditure rate to the basal or resting energy expenditure rate, and the metric is used by exercise physiologists and clinical nutritionists to estimate work undertaken by individuals as they go through their daily life (Montoye et al., 1996). This approach is discussed more thoroughly in McCurdy (2000).

5.3.2 Key Algorithms

Ozone concentrations in each microenvironment are estimated using either a mass-balance or transfer factors approach, selected by the user. The user specifies probability distributions for the parameters that are used in the microenvironment model that reflect the observed variabilities in the parameters. These distributions can depend on the values of other variables calculated in the model or input to APEX. For example, the distribution of AERs in a home, office, or car can depend on the type of heating and air conditioning present, which are also stochastic inputs to the model, as well as the ambient temperature. The user can choose to keep the value of a stochastic parameter constant for the entire simulation (which would be appropriate for the volume of a house), 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, or seasonal patterns for various microenvironmental parameters. The distributions of parameters input to APEX characterize the variability of parameter values, and are not intended to reflect uncertainties in the parameter estimates.

The mass balance method used within APEX assumes that the air in an enclosed microenvironment is well-mixed and that the air concentration is fairly spatially uniform at a given time within the microenvironment. The following four processes are modeled to predict the concentration of an air pollutant in such a microenvironment:

- 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.

The transfer factors model is simpler than the mass balance model, however, still most parameters are derived from distributions rather than single values, to account for observed variability. It does not calculate concentration in a microenvironment from the concentration in the previous hour and it has only two parameters, a proximity factor, used to account for proximity of the microenvironment to sources or sinks of pollution, or other systematic differences between concentrations just outside the microenvironment and the ambient concentrations (at the measurements site), and a penetration factor, which quantifies the degree to which the outdoor air penetrates into the microenvironment. When there are no indoor sources, the penetration factor is essentially the ratio of the concentration in the microenvironment to the outdoor concentration.

Regardless of the method used to estimate the microenvironmental concentrations, APEX calculates a time series of exposure concentrations that a simulated individual experiences during the modeled time period. APEX estimates the exposure using the concentrations calculated for each microenvironment and the time spent in each of a sequence of microenvironments visited according to the "activity diary" of each individual. The hourly average exposures of each simulated individual are time-weighted averages of the within-hour exposures. From hourly exposures, APEX calculates the time series of 8-hr and daily average exposures that simulated individuals experience during the simulation period. APEX then statistically summarizes and tabulates the hourly, 8-hr, and daily exposures.

Estimation of Ambient Air Quality

For estimating ambient O_3 concentrations to use in the exposure model, the urban areas modeled here have several monitors measuring hourly O_3 concentrations (ranging from 12 in the Atlanta area to 51 in the Los Angeles area, for 2008). Having multiple monitors in the simulated areas collecting time-resolved data allows for the utilization of APEX spatial and temporal capabilities in estimating exposure. Since APEX uses actual records of where individuals are

located at specific times of the day, more realistic exposure estimates are obtained in simulating the contact of individuals with these spatially and temporally diverse concentrations. Primary uncertainties in the air quality data input to the model result from estimating concentrations at locations which may not be in close proximity to monitoring sites (as estimated by spatial interpolation of actual data points) and from the method used to estimate missing data for some hours or days. In addition, concentrations of O_3 near roadways are particularly difficult to estimate due to the rapid reaction of O_3 with nitric oxide emitted from motor vehicles.

We have modeled the O_3 seasons for 2006 to 2010 to account for year-to-year variability of air quality and meteorology in recent years. Having this wide range of air quality data across multiple years available for use in the exposure simulation has a direct impact on more realistically estimating the range of exposures, rather than using a single year of air quality data.

Estimation of Concentrations in Indoor Microenvironments

The importance of estimation of concentrations in indoor microenvironments (e.g., homes, offices, schools, restaurants, vehicles) is underscored by the finding that personal exposure measurements of O_3 may not be well-correlated with ambient measurements and indoor concentrations are usually much lower than ambient concentrations (EPA, 2012a, Section 4.3.3).

APEX has been designed to better estimate human exposure through use of algorithms that attempt to capture the full range of O_3 concentrations expected within several important microenvironments. Parameters used to estimate the concentrations in microenvironments can be highly variable, both between microenvironments (e.g., different houses have varying characteristics) and within microenvironments (e.g., the characteristics of a given house can vary over time). Since APEX is a probabilistic model, if data accurately characterizing this variability are provided to the model, then such variabilities would not result in uncertainties in the estimation of the microenvironmental concentrations. Thus, it is the input data used in development of the parameters that are the limiting factor, and to date, APEX uses the most current available data to develop required distributions of parameters for estimation of microenvironmental concentrations.

Air Exchange Processes

The air exchange rate is the single most important factor in determining the relationship between outdoor and indoor concentrations of O_3. AERs are highly variable, both within a

microenvironment over time and between microenvironments of the same type. AERs depend on the physical characteristics of a microenvironment and also on the behavior of the occupants of the microenvironment. There is a strong dependence on temperature, and some dependence on other atmospheric conditions, such as wind. APEX uses probabilistic distributions of AERs which were derived from several measurement studies in a number of locations, and are stratified by both temperature and the presence or absence of air conditioning. These two variables are the most influential variables influencing AER distributions (see Appendix 5B).

Removal Processes

Concentrations within indoor microenvironments can be reduced due to removal processes such as deposition to surfaces and by reaction with other chemicals in the air. Deposition is modeled probabilistically in APEX by a using a distribution of decay rates. The lack of a better treatment of indoor air chemistry is not considered to be a significant limitation of APEX for modeling O_3.

Characterization of Population Demographics and Activity Patterns

By using actual time-location-activity diaries that capture the duration and frequency of occurrence of visitations/activities performed, APEX can simulate expected variability in human behavior, both within and between individuals. Fundamentals of energy expenditure are then used to estimate relative intensity of activities performed. This, combined with microenvironmental concentrations, allows for the reasonable estimation of the magnitude, frequency, pattern, and duration of exposures an individual experiences.

CHAD is the most complete, high quality source of human activity data for use in exposure modeling. The database contains over 38,000 individual daily diaries including time-location-activity patterns for individuals of both sexes across a wide range of ages (<1 to 94). The database is geographically diverse, containing diaries from individuals residing in major cities, suburban and rural areas across the U.S. Time spent performing activities within particular locations can be on a minute-by minute basis, thus avoiding the smoothing of potential peak exposures longer time periods would give. Table 5-1 summarizes the studies in CHAD used in this modeling analysis.

There are some limitations to the database, however, many of which are founded in the individual studies from which activity patterns were derived (Graham and McCurdy, 2004). A

few questions remain regarding the representativeness of CHAD diaries to the simulated population, such as the age of diary data (i.e., some data were generated in the 1980s) and diary structure differences (i.e., real-time versus recall method of data collection). Many of the assumptions about use of these activity patterns in exposure modeling are strengthened by the manner in which they are used by APEX, through focusing on the most important individual attributes that contribute to variability in human behavior (e.g., age, sex, time spent outdoors, day of week, ambient temperature, occupation).

The extent to which the human activity database provides a balanced representation of the population being modeled is likely to vary across areas. Although the algorithm that constructs activity sequences accounts to some extent for the effects of population demographics and local climate on activity, this adjustment procedure may not account for all inter-city differences in people's activities. A new methodology has been developed to more appropriately assign individual diaries to reflect time-location-activity patterns in simulated individuals (discussed further in section 4.5.3). Input distributions used in the new procedure for constructing multi-day activity patterns are based on longitudinal activity data from children of a specific age range (appropriate for this application where similar aged children are the primary focus), however the data used were limited to one study and may not be appropriate for other simulated individuals. Thus, there are limitations in approximating within-person variance and between-person variance for certain variables (e.g., time spent outdoors). Personal activity patterns are also likely to be affected by many local factors, including topography, land use, traffic patterns, mass transit systems, and recreational opportunities, which are not incorporated in the current exposure analysis approach due to the complexity of scale and lack of data to support the development of a reasonable approach.

Table 5-1. Studies in CHAD used in this analysis

Study name	Geographic coverage	Study time period	Subject ages	Diary-days	Diary-days (ages 5-18)	Diary type and study design	Reference
Baltimore Retirement Home Study (EPA)	One building in Baltimore	01/1997-02/1997, 07/1998-08/1998	72 - 93	391	0	Diary	Williams et al. (2000)
California Youth Activity Patterns Study (CARB)	California	10/1987-09/1988	12 - 17	181	181	Recall; Random	Robinson et al. (1989), Wiley et al. (1991a)
California Adults Activity Patterns Study (CARB)	California	10/1987-09/1988	18 - 94	1,548	36	Recall; Random	Robinson et al. (1989), Wiley et al. (1991a)
California Children Activity Patterns Study (CARB)	California	04/1989- 02/1990	<1 - 11	1,200	683	Recall; Random	Wiley et al. (1991b)
Cincinnati Activity Patterns Study (EPRI)	Cincinnati metro. area	03/1985-04/1985, 08/1985	<1 - 86	2,597	738	Diary; Random	Johnson (1989)
Denver CO Personal Exposure Study (EPA)	Denver metro. area	11/1982- 02/1983	18 - 70	796	7	Diary; Random	Johnson (1984), Akland et al. (1985)
Los Angeles Ozone Exposure Study: Elementary School	Los Angeles	10/1989	10 - 12	49	49	Diary	Spier et al. (1992)
Los Angeles Ozone Exposure Study: High School	Los Angeles	09/1990-10/1990	13 - 17	42	42	Diary	Spier et al. (1992)

National Human Activity Pattern Study (NHAPS): Air	National	09/1992-10/1994	<1 - 93	4,338	634	Recall; Random	Klepeis et al. (1996), Tsang and Klepeis (1996)
National Human Activity Pattern Study (NHAPS): Water	National	09/1992-10/1994	<1 - 93	4,347	691	Recall; Random	Klepeis et al. (1996), Tsang and Klepeis (1996)
National Study of Avoidance of S (NSAS)	7 U.S. metropolitan areas	06/2009-09/2009	35 - 92	6,824	0	Recall; Random	Knowledge Networks (2009)
Population Study of Income Dynamics PSID CDS I (Univ. Michigan I)	National	02/1997-12/1997	<1 - 13	4,988	3,093	Recall; Random	University of Michigan (2012)
Population Study of Income Dynamics PSID CDS II (Univ. Michigan II)	National	01/2002-12/2003	5 - 19	4,773	4,763	Recall; Random	University of Michigan (2012)
RTI Ozone Averting Behavior	35 U.S. metropolitan areas	07/2002-08/2003	2 - 12	2,876	1,944	Recall; Random	Mansfield et al. (2006, 2009)
RTP Panel (EPA)	RTP, NC	06/2000-05/2001	55 - 85	1,000	0	Diary; Panel	Williams et al. (2003a,b)
Seattle	Seattle, WA	10/1999-03/2002	6 - 91	1,688	318	Diary; Panel	Liu et al. (2003)
Washington, D.C. (EPA)	Wash., D.C. metro. area	11/1982-02/1983	18 - 71	695	11	Diary; Random	Hartwell et al. (1984), Akland et al. (1985)
Totals		**1982 - 2009**	**<1 - 94**	**38,333**	**13,190**		

Averting Behavior and Exposure

A growing area of air pollution research involves evaluating the actions persons might perform in response to high O_3 concentration days (ISA, section 4.1.1). Most commonly termed *averting behaviors*, they can be broadly characterized as personal activities that either reduce pollutant emissions or limit personal exposure levels. The latter topic is of particular interest in this REA due to the potential negative impact it could have on O_3 concentration-response (C-R) functions used to estimate health risk and on time expenditure and activity exertion levels recorded in the CHAD diaries used by APEX to estimate O_3 exposures. To this end, we have performed an additional review of the available literature here beyond that summarized in the ISA to include several recent technical reports that collected and/or evaluated averting behavior data. Our purpose is to generate a few reasonable quantitative approximations that allow us to better understand how averting behavior might affect our current population exposure and risk estimates. We expect that the continued development and communication of air quality information via all levels of environmental, health, and meteorological organizations will only further increase awareness of air pollution, its associated health effects, and the recommended actions to take to avoid exposure, thus making averting behaviors and participation rates an even more important consideration in future O_3 exposure and risk assessments. The following is a summary of our current findings, with details provided in Graham (2012).

The first element considered in our evaluation is peoples' general perception of air pollution and whether they were aware of alert notification systems. The prevalence of awareness was variable; about 50% to 90% of survey study participants acknowledge or were familiar with air quality systems (e.g., Blanken et al., 1991; KS DOH, 2006; Mansfield et al., 2006; Semenza et al., 2008) and was dependent on several factors. In studies that considered a persons' health status, e.g., asthmatics or parents of asthmatic children, there was a consistently greater degree of awareness (approximately a few to 15 percentage points) when compared to that of non-asthmatics. Residing in an urban area was also an important influential factor raising awareness, as both the number of high air pollution events and their associated alerts are greater when compared to rural areas. Of lesser importance, though remaining a statistically significant influential variable, were several commonly correlated demographic attributes such as age, education-level, and income-level, with each factor positively associated with awareness.

The second element considered in our evaluation was the type of averting behaviors performed. For our purposes in this O_3 REA, the most relevant studies were those evaluating

outdoor time expenditure, more specifically, the duration of outdoor events and the associated exertion level of activities performed while outdoors. This is because both of these variables are necessary to understanding O_3 exposure and adverse effects and in accurately estimating human health risk.

As stated above regarding air quality awareness, asthmatics consistently indicated a greater likelihood of performing averting behaviors compared to non-asthmatics – estimated to differ by about a factor of two. This difference could be the combined effect of those persons having been advised by health professional to avoid high air pollution events and them being aware of alert notification systems. Based on the survey studies reviewed, we estimate that 30% of asthmatics may reduce their outdoor activity level on alert days (e.g., KS DOH, 2006; McDermott et al., 2006; Wen et al., 2009).[2] An estimate of 15%, derived from reductions in public attendance at outdoor events (Zivin and Neidell, 2009) is consistent with the above estimate when considering that it is likely represented by a non-asthmatic population. That said, both attenuation and the re-establishment of averting behavior was apparent when considering a few to several days above high pollution alert levels (either occurring over consecutive days or across an entire year) (McDermott et al., 2006; Zivin and Neidell, 2009), suggesting that participation in averting behavior over a multiday period for an individual is complex and likely best represented by a time and activity-dependent function rather than a simple point estimate.

There were only a few studies offering quantitative estimates of durations of averting behavior, either considering outdoor exertion level or outdoor time (Bresnahan et al., 1997; Mansfield et.al, 2006, Neidell, 2010; Sexton, 2011). Each of these studies considered outdoor time expenditure during the afternoon hours. Based on the studies reviewed, we estimate that outdoor time/exertion during afternoon hours may be reduced by about 20-40 minutes in response to an air quality alert notification. Generally requisite factors include: a high alert level for the day (e.g., red or greater on the AQI), high O_3 concentrations (above the NAAQS), and persons having a compromised health condition (e.g., asthmatic or elderly).

The third element considered in our evaluation is how to further define the impact of averting behavior on modeled exposure estimates.[3] As described in section 5.3.2, APEX uses time location activity data (diaries) from CHAD to estimate population exposures. These diaries

[2] Many of these studies do not account for one important factor when using a recall questionnaire design: whether the participant's stated response to air pollution is the same as the action they performed.

[3] The discussion of another important effect of averting behavior is on concentration-response functions (more relevant to the risk assessment in chapter 7). This is presented in the ISA (section 4.1.2).

come from a number of differing studies; some were generated as part of an air pollution research study, some may have been collected during a summer/ozone season, while some diary days may have corresponded with high O_3 concentration and air quality alert days. At this time, none of the diary days used by APEX have been identified as representing days where a person did or did not perform an averting behavior to reduce their exposure. In considering the above discussion regarding the potential rate of participation and averting actions performed, it is possible that some of the CHAD diary days express times where that selected individual may have reduced their time spent outdoors or outdoor exertion level. Currently, without having an identifier for averting behavior, the diaries are assigned randomly[4] to a simulated persons' day and do not consider ambient O_3 concentrations. Therefore, there may be instances where, on a given day, a simulated person does appear to engage in averting behavior (a diary having less time than usual spent outdoors in the afternoon), while for most other persons on the same day (or the same person on a different high concentration day) there is no averting behavior. Therefore, averting behavior may be incorporated into our exposure modeling, albeit to an unknown degree,[5] though definitely generating low-biased estimates of exposures that would occur in the complete absence of averting behavior.

Modeling Physiological Processes

The modeling of physiological processes that are relevant to the exposure and intake of O_3 is a complicated endeavor, particularly when attempting to capture inter- and intra-personal variability in these rates. APEX has a physiological module capable of estimating ventilation rates ($\dot{V}_E$) for every activity performed by an individual, which primarily drives O_3 intake dose[6] rate estimates. See Isaacs, et al. (2008) and Chapter 7 of the APEX TSD (EPA, 2012c) for a discussion of this module. Briefly, the module is based on the relationship between energy expenditure and oxygen consumption rate, thus both within- and between-person variability in ventilation can be addressed through utilization of the unique sequence of events individuals go through each simulated day. These activity-specific $\dot{V}_E$ estimates, when normalized by BSA, are then used to characterize an individual's exertion level in compiling the summary exposure tables (Table 5-2). One of the key determinants of estimated $\dot{V}_E$ is the exertion level of an

[4] APEX uses maximum temperature in assigning diaries for a select day in an area, capturing some variability in O_3 concentrations.
[5] Neither the participation rate nor the duration of averting for simulated persons is being strictly controlled for by the model.
[6] Intake dose is a measure related to dose; it is the amount of ozone that enters the lungs.

individual's activity, where exertion levels have units of metabolic equivalents of work (MET), which is the ratio of energy expenditure for an activity to the person's basal, or resting, metabolic rate.

There are some limitations in using MET values for this purpose, due mostly to the manner in which the time-location-activity diaries were generated and subsequent estimates of exertion level. An individual (or their caregiver if younger than eight years old) would record the activity performed with a start and end time, with no information on the associated exertion level of the activity. Exertion level (MET) was then inferred by developers of the CHAD database (McCurdy et al., 2000) using standard values and distributions of those values reported by an expert panel of exercise physiologists (Ainsworth et al., 1993). Although this approach allows for an appropriate range of exertion levels to be assigned to the individuals' activities (and to the simulated population), children's activity levels fluctuate widely within a single activity category; their pattern is often characterized as having bursts of high energy expenditure within a longer time frame of less energy expenditure (Freedson, 1989). These fluctuations in energy expenditure that occur within an activity (and thus a simulated event) are not well captured by the MET assignment procedure.

5.3.3 Model Output

There are several useful indicators of exposure of people to O_3 air pollution and resulting intake of O_3. In this analysis, exposure indicators include daily maximum 1-hr and 8-hr average O_3 exposures, stratified by a measure of the level of exertion at the time of exposure. Factors that are important in calculating these indicators include the magnitude and duration of exposure, frequency of repeated high exposures, and the breathing rate of individuals at the time of exposure. The level of exertion of individuals engaged in particular activities is measured by an equivalent ventilation rate (EVR), ventilation normalized by body surface area (BSA, in m^2), which is calculated as $\dot{V}_E$ /BSA, where $\dot{V}_E$ is the ventilation rate (liters/minute). Table 5-2 lists the ranges of EVR corresponding to moderate and heavy levels of exertion.

Table 5-2. Exertion levels in terms of equivalent ventilation rates (liters/min-m^2 BSA)

Averaging time	Moderate exertion	Heavy exertion
1 hour	16-30 EVR	≥ 30 EVR
8 hour	13-27 EVR	≥ 27 EVR

from Whitfield et al., 1996, page 15.

APEX calculates two general types of exposure estimates: counts of the estimated number of people exposed to a specified O_3 concentration level and the number of times per O_3 season that they are so exposed; the latter metric is in terms of person-occurrences or person-days. The former highlights the number of individuals exposed *one or more* times per O_3 season to the exposure indicator of interest. In the case where the exposure indicator is a benchmark concentration level, the model estimates the number of people who are expected to experience exposures to that level of air pollution, or higher, at least once during the modeled period. APEX also reports counts of individuals with multiple exposures. The person-occurrences measure estimates the number of times per season that individuals are exposed to the exposure indicator of interest and then accumulates these estimates for the entire population residing in an area. This metric conflates people and occurrences: one occurrence for each of 10 people is counted the same as 10 occurrences for one person.

APEX tabulates and displays the two measures for exposures above levels ranging from 0.0 to 0.16 ppm by 0.01 ppm increments, where the exposures are:

- Daily maximum 1-hour average exposures
- Daily maximum 8-hour average exposures
- Daily average exposures.

These results are tabulated for the following population groups:

- All ages and activity levels
- Children at all activity levels
- Asthmatic children.

Separate output tables are produced for different levels of exertion concomitant with the exposures:

- All exertion levels
- Moderate and greater exertion levels

APEX also produces tables of the time spent in different microenvironments, stratified by exposure levels.

5.4 SCOPE OF EXPOSURE ASSESSMENT

5.4.1 Selection of Urban Areas to be Modeled

The selection of urban areas to include in the exposure analysis takes into consideration the location of O_3 epidemiological studies, the availability of ambient O_3 data, and the desire to represent a range of geographic areas, population demographics, and O_3 climatology. The criteria and considerations that went into selection of urban areas for the O_3 risk assessment included the following:

- The overall set of urban locations should represent a range of geographic areas, urban population demographics, and climatology.
- The locations should be focused on areas that do not meet or are close to not meeting the current 8-hr O_3 NAAQS and should include the largest areas with major O_3 nonattainment problems.
- There must be sufficient O_3 air quality data for the recent 2006-2010 period.
- The areas should include the 12 cities modeled in the epidemiologic-based risk assessment.

Based on these criteria, we chose the 16 urban areas listed in Table 5-3 to develop population exposure estimates.[7] As mentioned above, in this first draft REA, results are presented for four of these areas, Atlanta, Denver, Los Angeles, and Philadelphia. The geographic extents of these four modeled areas are illustrated in Appendix 5B.

5.4.1 Time Periods Modeled

We have modeled the O_3 seasons for 2006 to 2010. The exposure periods modeled are the O_3 seasons for which routine hourly O_3 monitoring data are available. These periods include most of the high-ozone events in each area. The time periods modeled for each area are listed in Table 5-3. The number of ozone monitors in each area varies slightly from year-to-year. The number of monitors in 2008 used in the exposure modeling are 12 for the Atlanta area, 17 for Denver, 51 for Los Angeles, and 19 for Philadelphia.

[7] In the remainder of this chapter the city name in bold in Table 4-2 is used to represent the entire urban area.

Table 5-3. Urban Areas and Time Periods Modeled[a]

Urban Area (CBSAs or Counties)	Period modeled
Atlanta area, GA (Barrow, Bartow, Bibb, Butts, Carroll Floyd, Cherokee, Clarke, Clayton, Cobb, Coweta, Dawson, De Kalb, Douglas, Fayette, Forsyth, Fulton, Gwinnett, Hall, Haralson, Heard, Henry, Jasper, Lamar, Meriwether, Gilmer, Newton, Paulding, Pickens, Pike, Polk, Rockdale, Spalding, Troup, Upson, Walton, Chambers (AL))	March 1 to Oct. 31
Baltimore-Towson**,** MD	April 1 to Oct. 31
Boston area, MA (Barnstable, Bristol, Dukes, Essex, Middlesex, Nantucket, Norfolk, Plymouth, Suffolk, Worcester)	April 1 to Sept. 30
Chicago-Naperville-Joliet, IL-IN-WI	April 1 to Sept. 30
Cleveland-Akron-Elyria, OH	April 1 to Oct. 31
Dallas-Fort Worth-Arlington, TX	Jan. 1 to Dec. 30
Denver area, CO (Adams, Arapahoe, Boulder, Broomfield, Clear Creek, Denver, Douglas, Elbert, Gilpin, Jefferson, Park, Larimer, Weld)	April 1 to Sept. 30
Detroit-Warren-Livonia, MI	April 1 to Sept. 30
Houston-Sugar Land-Baytown, TX	Jan. 1 to Dec. 30
Los Angeles-Long Beach-Riverside, CA (Los Angeles, Orange, Riverside (part), San Bernardino (part), Ventura (part))	Jan. 1 to Dec. 30
New York-Northern New Jersey-Long Island, NY-NJ-PA	April 1 to Sept. 30
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	April 1 to Oct. 31
Sacramento--Arden-Arcade--Roseville, CA	Jan. 1 to Dec. 30
Seattle-Tacoma-Bellevue, WA	May 1 to Sept. 30
St. Louis, MO-IL	April 1 to Oct. 31
Washington-Arlington-Alexandria, DC-VA-MD-WV	April 1 to Oct. 31

[a] In this first draft REA, Atlanta, Denver, Los Angeles, and Philadelphia are modeled.

5.4.2 Populations Modeled

Exposure modeling was conducted for the general population residing in each area modeled, as well as for school-age children (ages 5 to 18) and asthmatic school-age children. Due to the increased amount of time spent outdoors engaged in relatively high levels of physical activity (which increases intake), school-age children as a group are particularly at risk for experiencing O_3-related health effects (EPA, 2012a, Chapter 8). We report results for school-age children down to age five, however, there is a trend for younger children to attend school. Some states allow 4-year-olds to attend kindergarten, and most states have preschool programs for children younger than five. In 2000, six percent of U.S. children ages 3 to 19 who attend school

were younger than five years old (2000 Census Summary File 3, Table QT-P19: School Enrollment). We are not taking these younger children into account in our analysis due to a lack of information which would let us characterize this group of children.

The population of asthmatic children is estimated for each city using asthma prevalence data from the National Health Interview Surveys (NHIS) (Dey and Bloom, 2005). Asthma prevalence rates for children aged 0 to 17 years were calculated for each age, sex, and geographic region. For this analysis, asthma prevalence was defined as the probability of a "Yes" response to the question: "do you still have asthma?" among those that responded "Yes" or "No" to this question. A detailed description of this analysis is presented in Appendix 5B.

5.4.3 Microenvironments Modeled

In APEX, microenvironments provide the exposure locations for modeled individuals. For exposures to be accurately estimated, it is important to have realistic microenvironments that are matched closely to where people are physically located on a daily and hourly basis. As discussed in section 4.3.2 above, the two methods available in APEX for calculating pollutant concentrations within microenvironments are a mass balance model and a transfer factor approach. Table 5-4 lists the 28 microenvironments selected for this analysis and the exposure calculation method for each. The parameters used in this analysis for modeling these microenvironments are described in Appendix 5B.

Table 5-4. Microenvironments modeled

	Microenvironment	Calculation Method	Parameters[1]
1	Indoor – Residence	Mass balance	AER and DE
2	Indoor – Community Center or Auditorium	Mass balance	AER and DE
3	Indoor – Restaurant	Mass balance	AER and DE
4	Indoor – Hotel, Motel	Mass balance	AER and DE
5	Indoor – Office building, Bank, Post office	Mass balance	AER and DE
6	Indoor – Bar, Night club, Café	Mass balance	AER and DE
7	Indoor – School	Mass balance	AER and DE
8	Indoor – Shopping mall, Non-grocery store	Mass balance	AER and DE
9	Indoor – Grocery store, Convenience store	Mass balance	AER and DE
10	Indoor – Metro-Subway-Train station	Mass balance	AER and DE

11	Indoor – Hospital, Medical care facility	Mass balance	AER and DE
12	Indoor – Industrial, factory, warehouse	Mass balance	AER and DE
13	Indoor – Other indoor	Mass balance	AER and DE
14	Outdoor – Residential	Factors	None
15	Outdoor – Park or Golf course	Factors	None
16	Outdoor – Restaurant or Café	Factors	None
17	Outdoor – School grounds	Factors	None
18	Outdoor – Boat	Factors	None
19	Outdoor – Other outdoor non-residential	Factors	None
20	Near-road – Metro-Subway-Train stop	Factors	PR
21	Near-road – Within 10 yards of street	Factors	PR
22	Near-road – Parking garage (covered or below ground)	Factors	PR
23	Near-road – Parking lot (open), Street parking	Factors	PR
24	Near-road – Service station	Factors	PR
25	Vehicle – Cars and Light Duty Trucks	Factors	PE and PR
26	Vehicle – Heavy Duty Trucks	Factors	PE and PR
27	Vehicle – Bus	Factors	PE and PR
28	Vehicle – Train, Subway	Factors	PE and PR

[1] AER=air exchange rate, DE=decay-deposition rate, PR=proximity factor, PE=penetration factor

5.4.1 Benchmark Levels Modeled

Benchmark levels used in this assessment include concentrations of 0.060, 0.070 and 0.080 ppm, which are the same benchmark levels used in the exposure assessment conducted in the last review. Estimating exposures to ambient O3 concentrations at and above these benchmark levels is intended to provide some perspective on the public health impacts of O3-related health effects that have been demonstrated in human clinical and toxicological studies, but cannot currently be evaluated in quantitative risk assessments, such as lung inflammation, increased airway responsiveness, and decreased resistance to infection. The 0.080 ppm benchmark represents an exposure level at which there is a substantial amount of clinical evidence demonstrating a range of O3-related effects including lung inflammation and airway responsiveness in healthy individuals. The 0.070 ppm benchmark reflects evidence that asthmatics have larger and more serious effects than healthy people as well as a substantial body of epidemiological evidence of associations with O3 levels that extend will below 0.080 ppm.

The 0.060 ppm benchmark additionally represents the lowest exposure level at which O3-related effects have been observed in clinical studies of healthy individuals.

5.5 VARIABILITY AND UNCERTAINTY

An important issue associated with any population exposure or risk assessment is the characterization of variability and uncertainty. *Variability* refers to the inherent heterogeneity in a population or variable of interest (e.g., residential air exchange rates). The degree of variability cannot be reduced through further research, only better characterized with additional measurement. *Uncertainty* refers to the lack of knowledge regarding the values of model input variables (i.e., *parameter uncertainty*), the physical systems or relationships used (i.e., use of input variables to estimate exposure or risk or *model uncertainty*), and in specifying the scenario that is consistent with purpose of the assessment (i.e., *scenario uncertainty*). Uncertainty is, ideally, reduced to the maximum extent possible through improved measurement of key parameters and iterative model refinement. The approaches used to assess variability and to characterize uncertainty in this REA are discussed in the following two sections. Each section also contains a concise summary of the identified components contributing to uncertainty and how each source may affect the estimated exposures.

5.5.1 Treatment of Variability

The purpose for addressing variability in this REA is to ensure that the estimates of exposure and risk reflect the variability of ambient O_3 concentrations, population characteristics, associated O_3 exposure and dose, and potential health risk across the study area and for the simulated at-risk populations. In this REA, there are several algorithms that account for variability of input data when generating the number of estimated benchmark exceedances or health risk outputs. For example, variability may arise from differences in the population residing within census tracts (e.g., age distribution) and the activities that may affect population exposure to O_3 (e.g., time spent inside vehicles, performing moderate or greater exertion level activities outdoors). A complete range of potential exposure levels and associated risk estimates can be generated when appropriately addressing variability in exposure and risk assessments; note however that the range of values obtained would be within the constraints of the input parameters, algorithms, or modeling system used, not necessarily the complete range of the true exposure or risk values.

Where possible, staff identified and incorporated the observed variability in input data sets to estimate model parameters within the exposure assessment rather than employing standard default assumptions and/or using point estimates to describe model inputs. The details regarding variability distributions used in data inputs are described in Appendix 5B. To the extent possible given the data available for the assessment, staff accounted for variability within the exposure modeling. APEX has been designed to account for variability in some of the input data, including the physiological variables that are important inputs to determining ventilation rates. As a result, APEX addresses much of the variability in factors that affect human exposure. Important sources of the variability accounted for in this analysis are summarized in Appendix 5D.

5.5.2 Characterization of Uncertainty

While it may be possible to capture a range of exposure or risk values by accounting for variability inherent to influential factors, the true exposure or risk for any given individual within a study area is largely unknown. To characterize health risks, exposure and risk assessors commonly use an iterative process of gathering data, developing models, and estimating exposures and risks, given the goals of the assessment, scale of the assessment performed, and limitations of the input data available. However, significant uncertainty often remains and emphasis is then placed on characterizing the nature of that uncertainty and its impact on exposure and risk estimates.

The REA's for the previous O_3, NO_2, SO_2, and CO NAAQS reviews each presented a characterization of uncertainty of exposure modeling (Langstaff, 2007; EPA 2008, 2009, 2010). Details regarding those approaches and a summary of the key findings of those reports that are most relevant to the current ozone exposure assessment are provided in Appendix 5D. The most influential elements of uncertainty are the following:

- Activity patterns
- Air exchange rates (AERs)
- Spatial variability in O_3 concentrations
- METs distributions
- Resting metabolic rate and ventilation rate equations

In the second draft REA, we plan to present the results of sensitivity analyses for each of these five elements. Activity pattern sensitivity analyses will include restricting diaries to more recent years, restricting diaries to be city-specific, and simulating activity patterns for specific cohorts, including school children and outdoor workers. These will include the treatment of activity patterns that can lead to repeated exposures to high ozone. Air exchange rates sensitivity analyses will include restricting AERs to be city-specific. The sensitivity analyses for spatial variability in O_3 concentrations will include varying the radius of influence of the air quality monitors and using photochemical grid modeling results with the monitored concentrations to improve the spatial interpolation of O_3 concentrations. The influence of METs distributions, resting metabolic rate equations, and ventilation rate equations will be ascertained by using updated METs distributions and alternative resting metabolic rate and ventilation rate equations.

5.6 EXPOSURE ASSESSMENT RESULTS

5.6.1 Overview

The results of the exposure analysis are presented as a series of graphs focusing on a range of benchmark levels, described in Chapter 2 and in Section 5.4.1 above, as being of particular health concern. A range of concentrations in the air quality data measured over the five year period (2006-2010) were used in the exposure model, providing a range of estimated exposures output by the model. Exposure results are presented for recent air quality (base years) and for air quality adjusted to just meet the current standards, based on 2006-2008 and 2008-2010 design values, as described in Chapter 3. Estimates of exposures for the year 2008 were developed for both of these sets of design values. This section first addresses the exposures estimated for school children using figures and follows those with tables of estimates of exposures for school-age children (ages 5-18), asthmatic school-age children, and the general population, under moderate or greater exertion.

5.6.2 Exposure Modeling Results

A series of figures are presented for each of the benchmark levels (0.060, 0.070, and 0.080 ppm-8hr), for each of the five years, 2006 – 2010. Exposure estimates are presented for those individuals experiencing moderate or greater levels of exertion averaged over the same 8-hr period that the exposure occurred. The exertion level is characterized by breathing rates, as described in Section 5.3.3. Results for school-age children exposed to O_3 while engaged in moderate exertion are presented in each of the subsequent figures. Results for asthmatic school-

age children have similar exposure outcomes and patterns across the urban areas modeled (see the sets of tables following the figures).

The next set of figures (Figure 5-1 though Figure 5-15) shows the percent of school-age children who experience at least one 8-hour average exposure above the benchmark levels of 0.06, 0.07, and 0.08 ppm-8hr, while at the same time engaged in activities resulting in moderate or greater exertion. On each figure the base case air quality exposure scenario can be compared to exposures with air quality just meeting the current standard. "75 6-8" denotes the current standard of 75 ppb based on 2006-2008 design values, and "75 8-10" denotes the current standard of 75 ppb based on 2008-2010 design values. Note that the year 2008 has results for both of these current standard scenarios, since it occurs in both of the design value periods 2006-2008 and 2008-2010. For example, in Figure 5-7, 18 percent of school-age children in Atlanta are estimated to have experienced one or more 8-hours average exposure of at least 0.06 ppm, while engaged in moderate or greater exertion. When the air quality is adjusted to just meet the current standard based on the 2006-2008 design value for Atlanta, this estimate is reduced to 12 percent. When the air quality is adjusted to just meet the current standard based on the 2008-2010 design value for Atlanta, this estimate is 3 percent.

Figure 5-1. Percent of Children in 2006 With 8-hour Exposures > 0.06 ppm Concomitant With Moderate or Greater Exertion

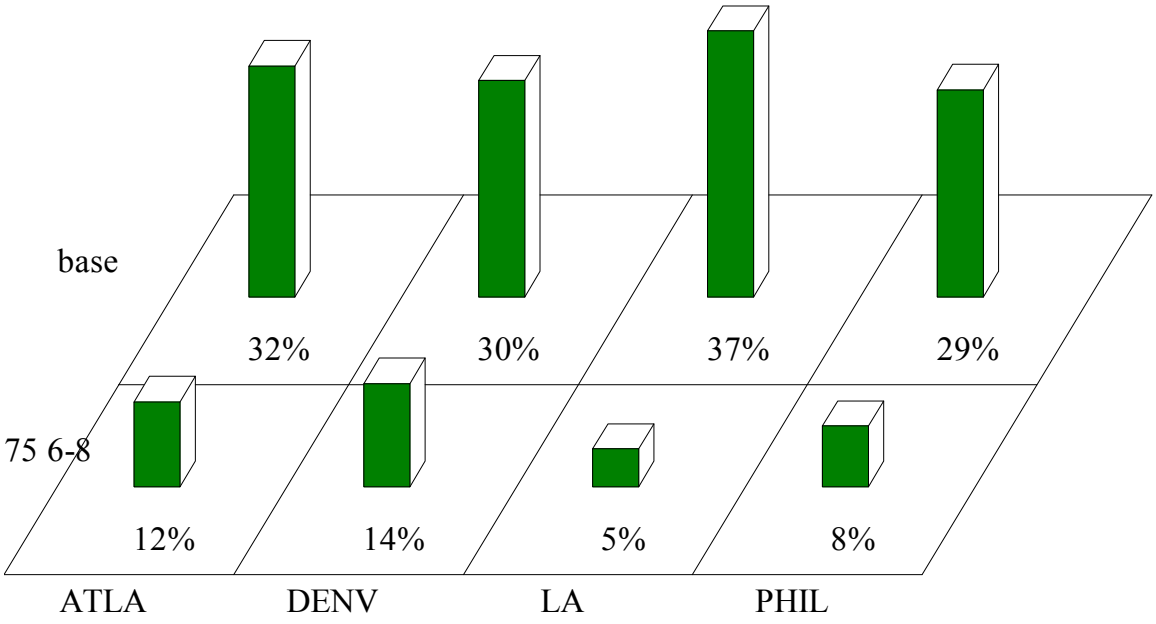


Figure 5-2. Percent of Children in 2006 With 8-hour Exposures > 0.07 ppm Concomitant With Moderate or Greater Exertion

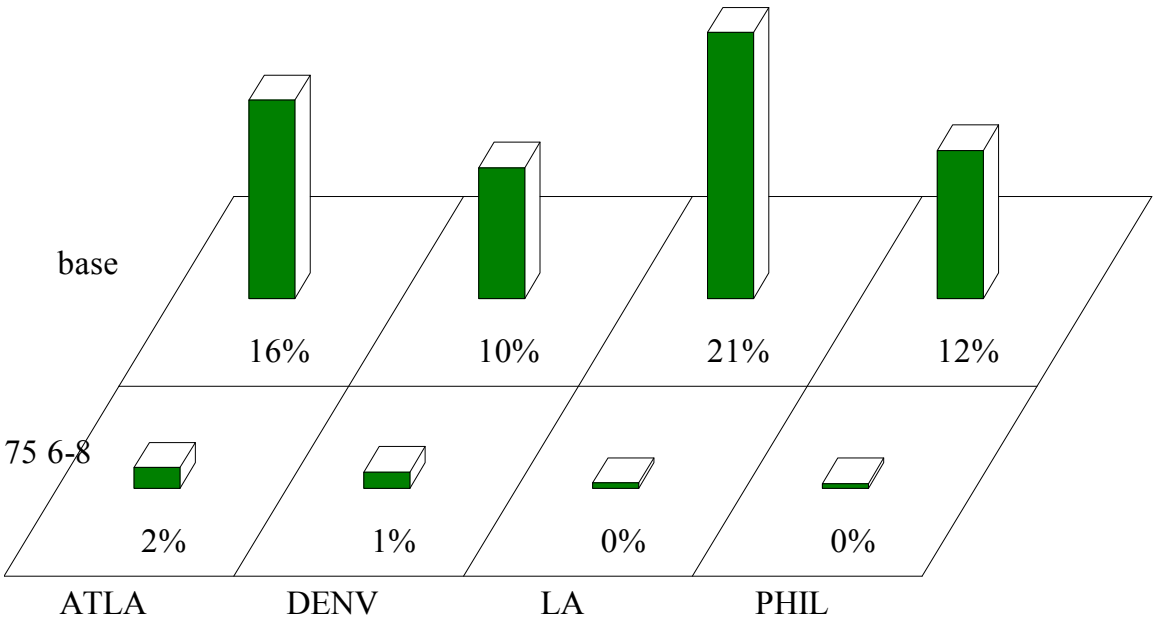


Figure 5-3. Percent of Children in 2006 With 8-hour Exposures > 0.08 ppm Concomitant With Moderate or Greater Exertion

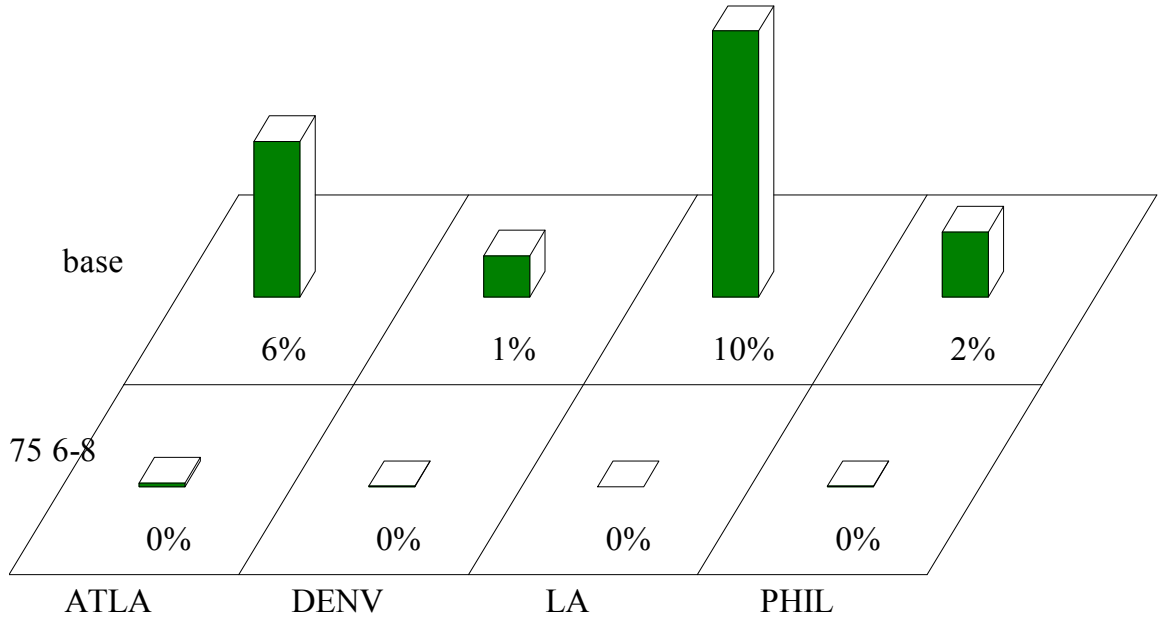


Figure 5-4. Percent of Children in 2007 With 8-hour Exposures > 0.06 ppm Concomitant With Moderate or Greater Exertion

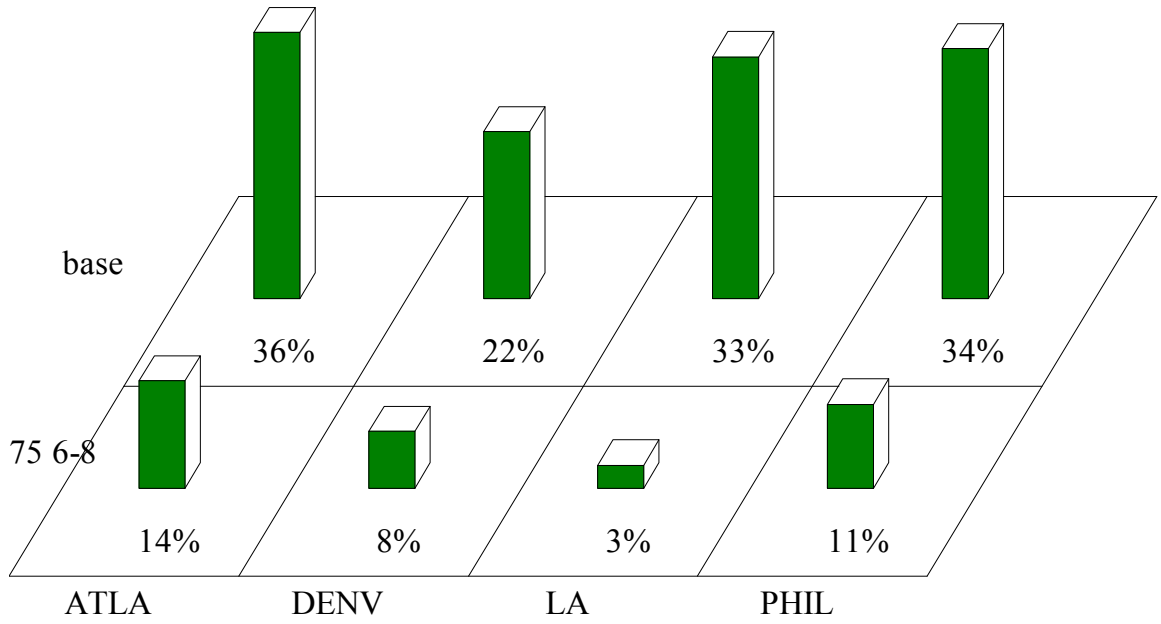


Figure 5-5. Percent of Children in 2007 With 8-hour Exposures > 0.07 ppm Concomitant With Moderate or Greater Exertion

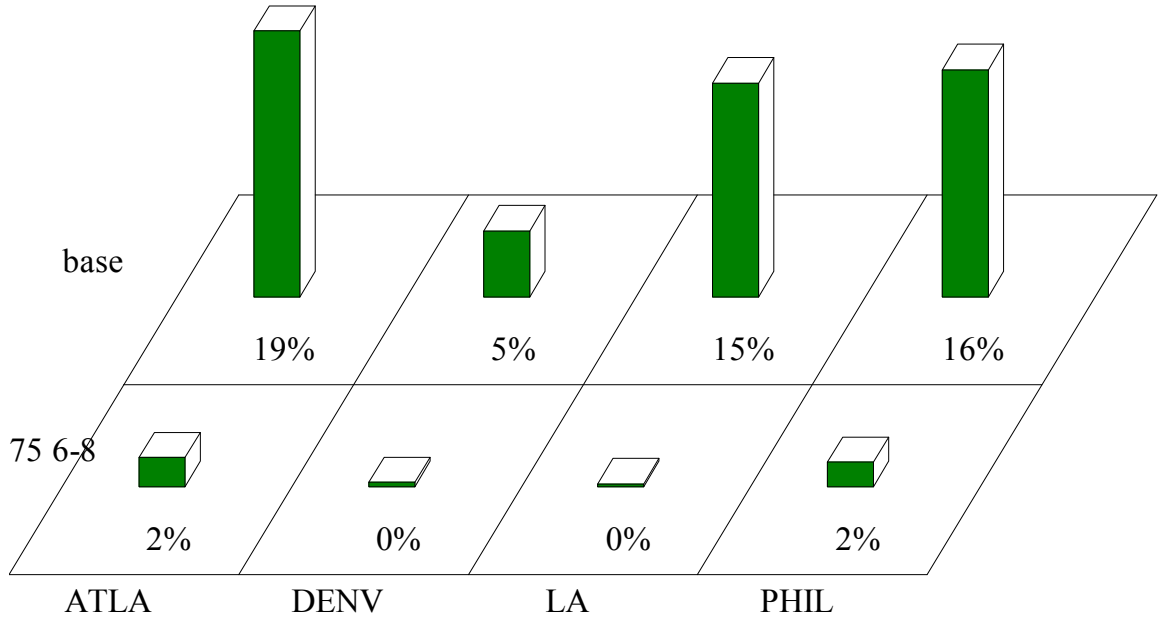


Figure 5-6. Percent of Children in 2007 With 8-hour Exposures > 0.08 ppm Concomitant With Moderate or Greater Exertion

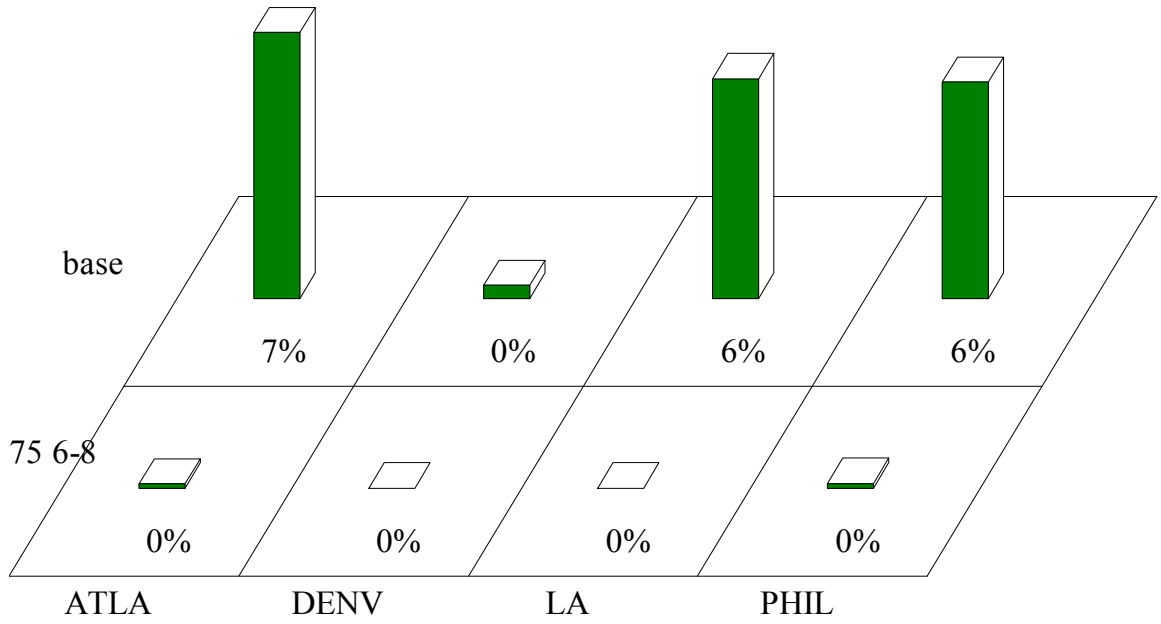


Figure 5-7. Percent of Children in 2008 With 8-hour Exposures > 0.06 ppm Concomitant With Moderate or Greater Exertion

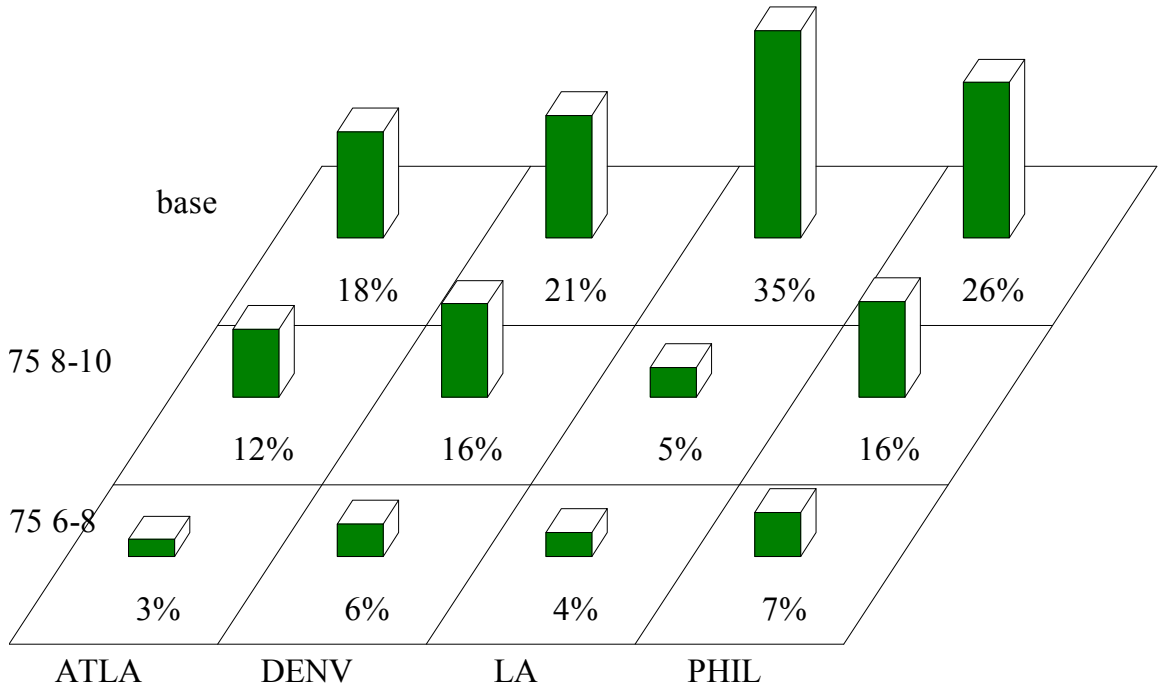


Figure 5-8. Percent of Children in 2008 With 8-hour Exposures > 0.07 ppm Concomitant With Moderate or Greater Exertion

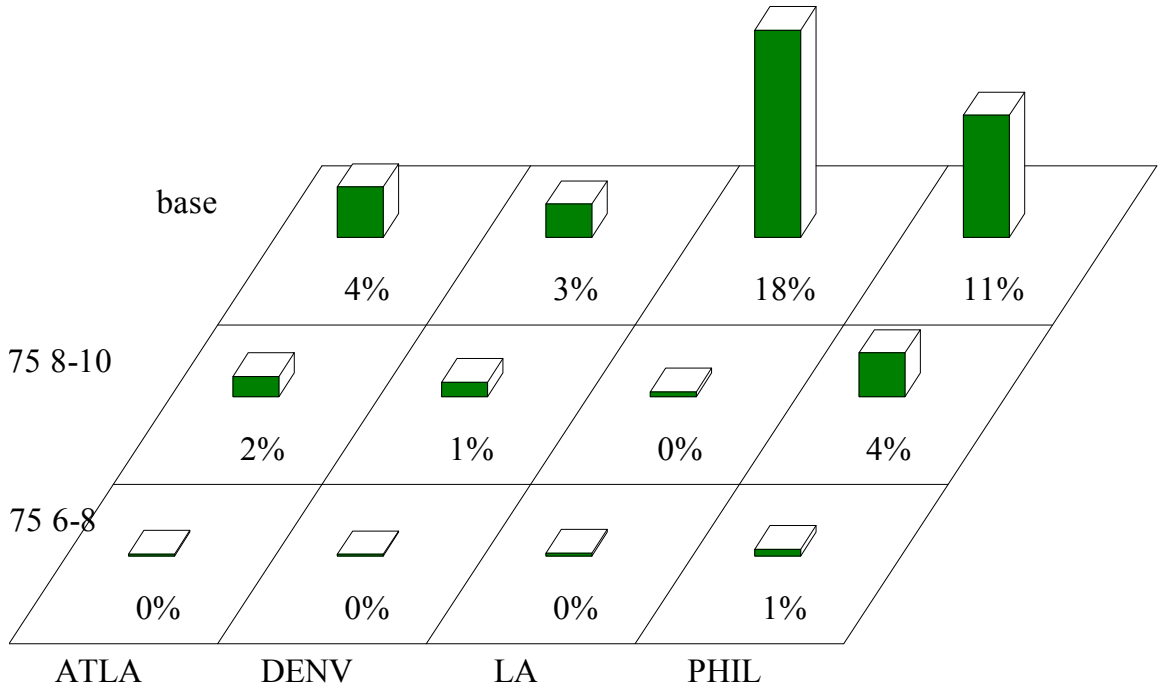


Figure 5-9. Percent of Children in 2008 With 8-hour Exposures > 0.08 ppm Concomitant With Moderate or Greater Exertion

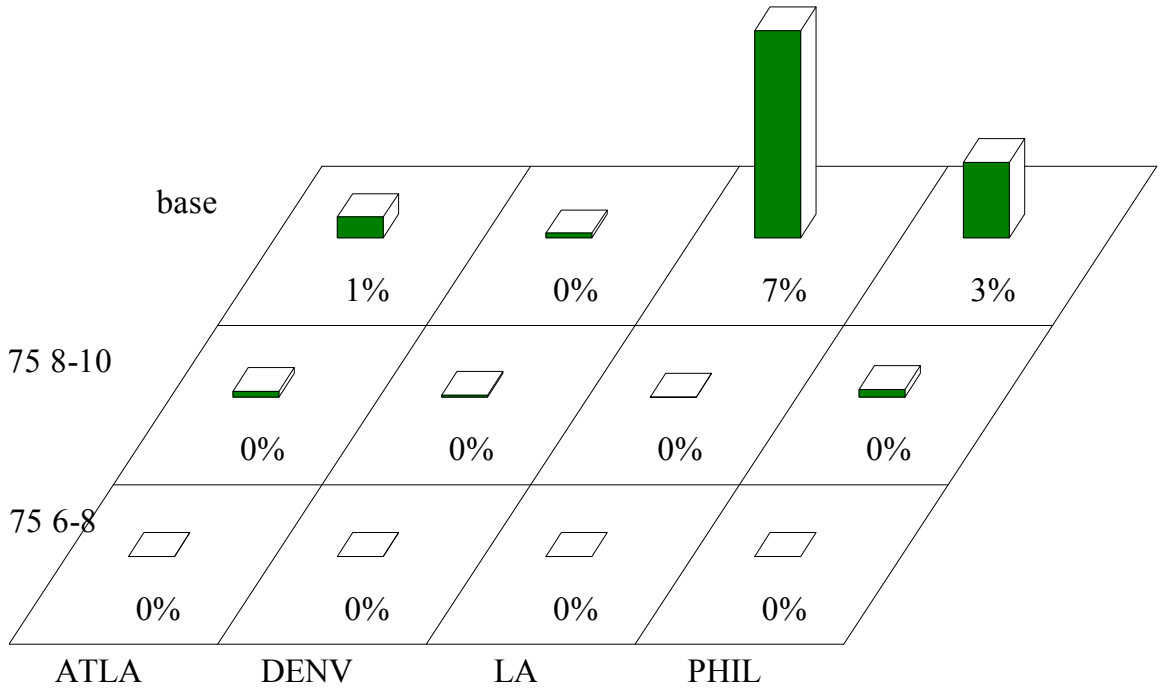


Figure 5-10. Percent of Children in 2009 With 8-hour Exposures > 0.06 ppm Concomitant With Moderate or Greater Exertion

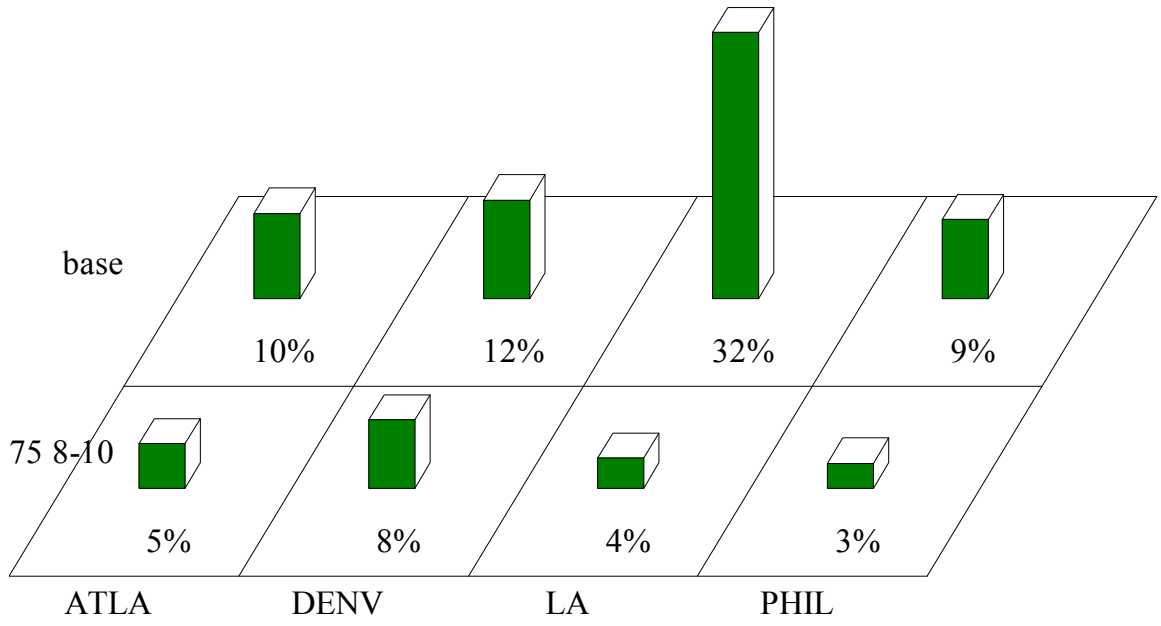


Figure 5-11. Percent of Children in 2009 With 8-hour Exposures > 0.07 ppm Concomitant With Moderate or Greater Exertion

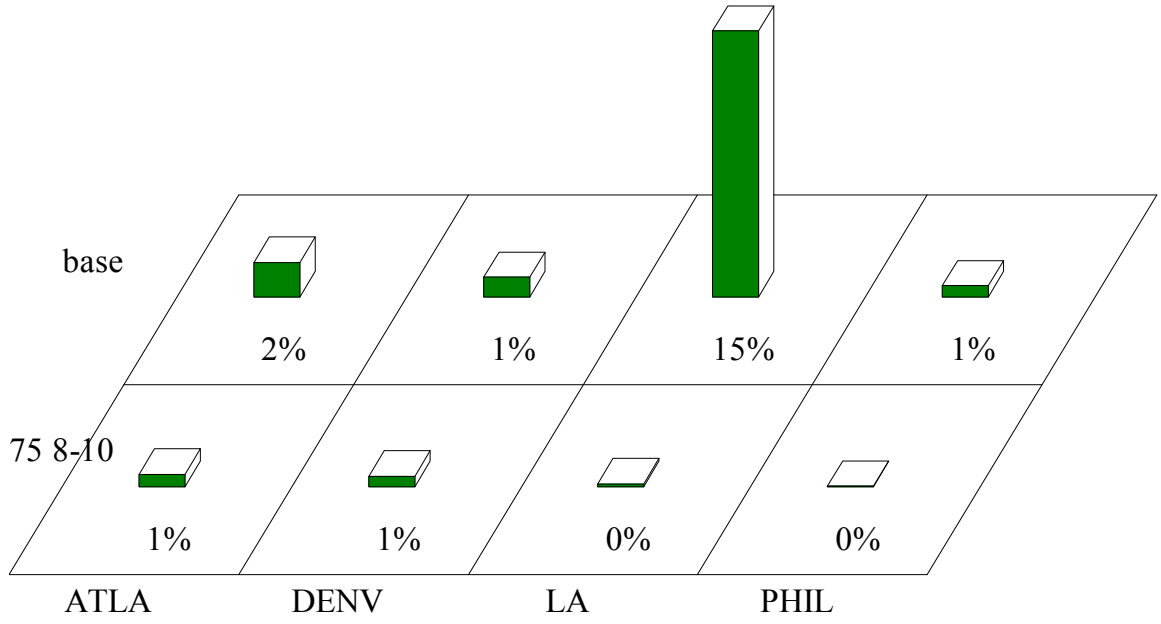


Figure 5-12. Percent of Children in 2009 With 8-hour Exposures > 0.08 ppm Concomitant With Moderate or Greater Exertion

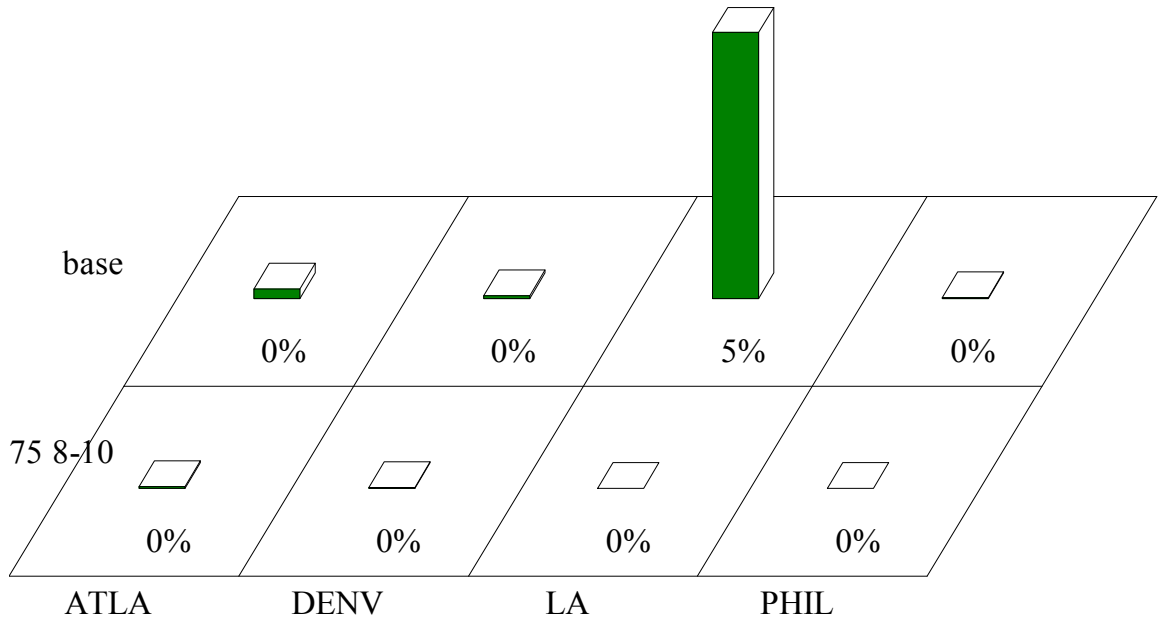


Figure 5-13. Percent of Children in 2010 With 8-hour Exposures > 0.06 ppm Concomitant With Moderate or Greater Exertion

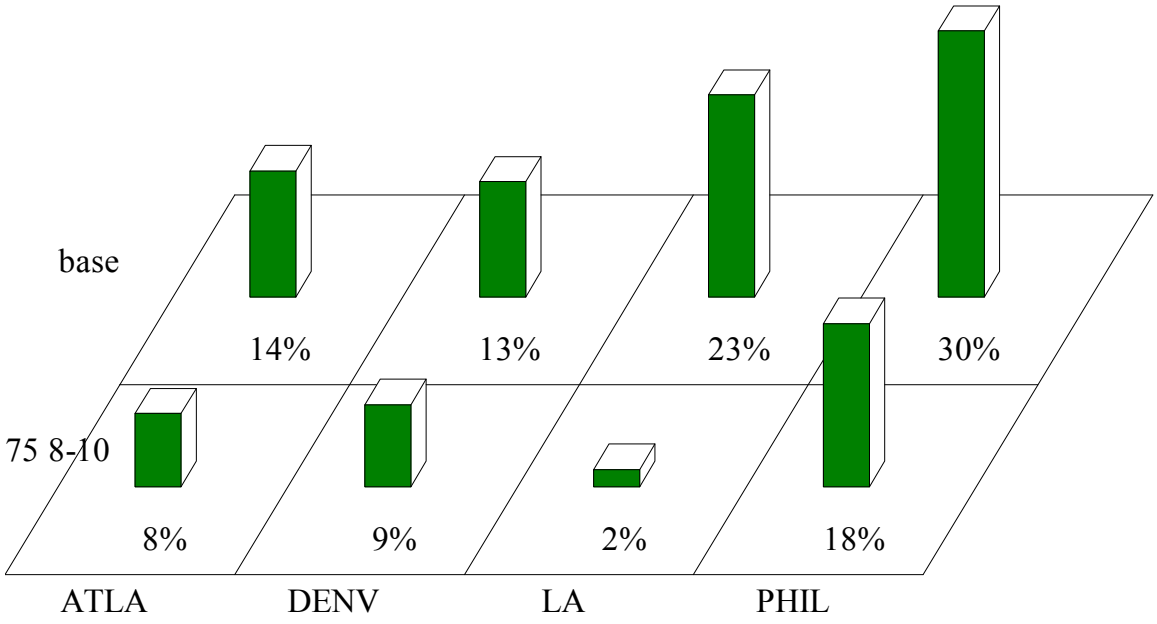


Figure 5-14. Percent of Children in 2010 With 8-hour Exposures > 0.07 ppm Concomitant With Moderate or Greater Exertion

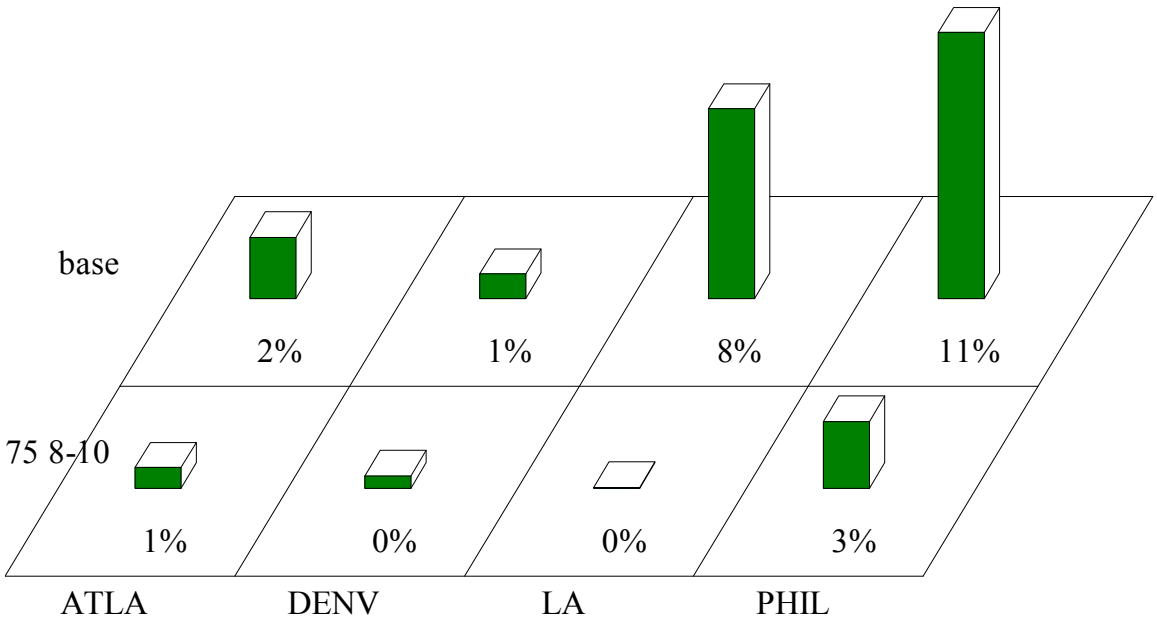
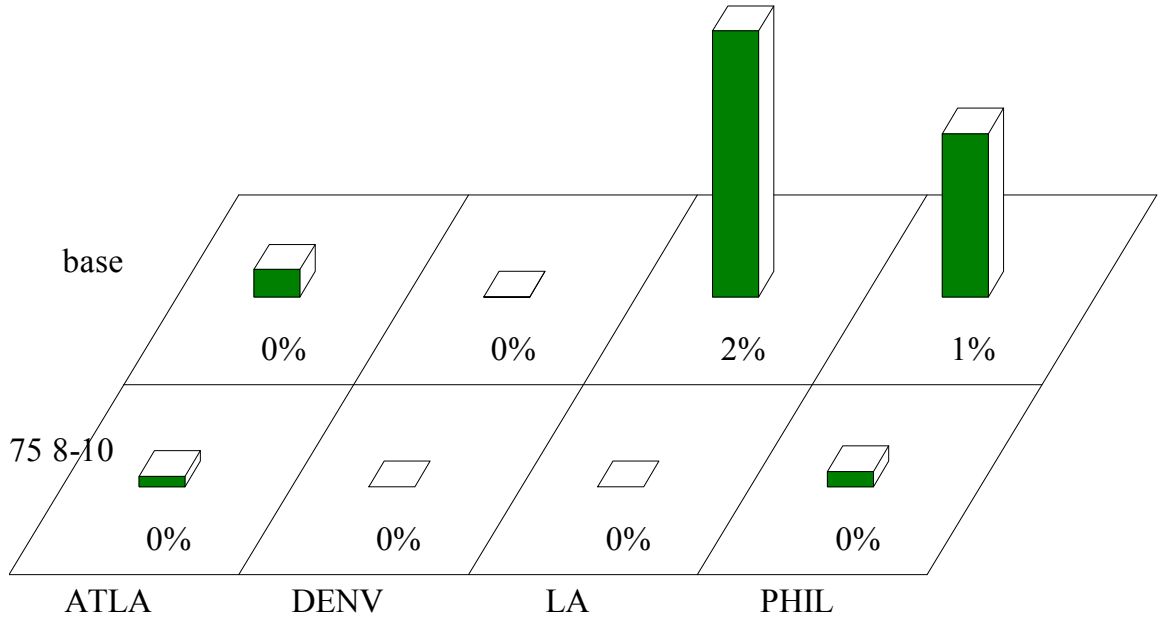


Figure 5-15. Percent of Children in 2010 With 8-hour Exposures > 0.08 ppm Concomitant With Moderate or Greater Exertion



The following tables present results for school-age children, asthmatic school-age children, and the general population.

Table 4-5. Percent of people with 1 or more 8-hour exposures above different levels (ppb-8hr), Children (moderate exertion)

City	myear	Above 0 base	Above 60 75/4 2006-8	Above 60 75/4 2008-10	Above 60 base	Above 70 75/4 2006-8	Above 70 75/4 2008-10	Above 70 base	Above 80 75/4 2006-8	Above 80 75/4 2008-10	Above 80 base
Atlanta	2006	96.8%	11.7%	.	31.9%	1.7%	.	16.1%	0.1%	.	5.7%
Denver	2006	96.3%	14.2%	.	29.8%	1.4%	.	10.4%	0.0%	.	1.5%
Los Angeles	2006	97.2%	5.2%	.	36.7%	0.5%	.	21.1%	0.0%	.	9.7%
Philadelphia	2006	96.4%	8.3%	.	28.6%	0.3%	.	11.8%	0.0%	.	2.4%
Atlanta	2007	96.9%	14.4%	.	35.8%	2.1%	.	19.2%	0.1%	.	7.0%
Denver	2007	96.4%	7.6%	.	22.4%	0.3%	.	4.8%	0.0%	.	0.3%
Los Angeles	2007	97.2%	3.1%	.	32.5%	0.2%	.	15.5%	0.0%	.	5.8%
Philadelphia	2007	96.4%	11.3%	.	33.6%	1.8%	.	16.4%	0.1%	.	5.7%
Atlanta	2008	96.8%	2.8%	11.5%	18.1%	0.2%	1.8%	4.4%	0.0%	0.2%	0.7%
Denver	2008	96.4%	5.6%	15.8%	20.8%	0.2%	1.3%	2.9%	0.0%	0.1%	0.2%
Los Angeles	2008	97.3%	4.1%	5.0%	35.2%	0.3%	0.4%	18.0%	0.0%	0.0%	7.0%
Philadelphia	2008	96.4%	7.5%	16.2%	26.4%	0.6%	3.8%	10.6%	0.0%	0.3%	2.6%
Atlanta	2009	96.9%	.	5.3%	10.1%	.	0.7%	1.9%	.	0.1%	0.2%
Denver	2009	96.4%	.	8.1%	11.7%	.	0.6%	1.1%	.	0.0%	0.0%
Los Angeles	2009	97.4%	.	3.6%	31.5%	.	0.2%	14.8%	.	0.0%	5.4%
Philadelphia	2009	96.4%	.	2.9%	9.3%	.	0.0%	0.6%	.	0.0%	0.0%
Atlanta	2010	97.0%	.	8.3%	14.5%	.	0.9%	2.5%	.	0.1%	0.3%
Denver	2010	96.4%	.	9.0%	13.3%	.	0.4%	1.0%	.	0.0%	0.0%
Los Angeles	2010	97.4%	.	1.8%	23.1%	.	0.0%	8.1%	.	0.0%	2.3%
Philadelphia	2010	96.5%	.	18.4%	30.1%	.	2.8%	10.9%	.	0.1%	1.5%
Atlanta	Mean	96.9%	9.7%	8.4%	22.1%	1.3%	1.1%	8.8%	0.1%	0.1%	2.8%
Denver	Mean	96.4%	9.1%	11.0%	19.6%	0.6%	0.7%	4.0%	0.0%	0.0%	0.4%
Los Angeles	Mean	97.3%	4.1%	3.5%	31.8%	0.3%	0.2%	15.5%	0.0%	0.0%	6.0%
Philadelphia	Mean	96.4%	9.0%	12.5%	25.6%	0.9%	2.2%	10.1%	0.0%	0.1%	2.4%

Table 4-6. Number of people with 1 or more 8-hour exposures above different levels (ppb-8hr), Children (moderate exertion)

City	myear	Above 0 base	Above 60 75/4 2006-8	Above 60 75/4 2008-10	Above 60 base	Above 70 75/4 2006-8	Above 70 75/4 2008-10	Above 70 base	Above 80 75/4 2006-8	Above 80 75/4 2008-10	Above 80 base
Atlanta	2006	829,000	100,000	.	273,000	14,300	.	138,000	1,110	.	48,400
Denver	2006	532,000	78,500	.	165,000	7,500	.	57,600	51	.	8,200
Los Angeles	2006	3,510,000	186,000	.	1,330,000	16,800	.	762,000	0	.	349,000
Philadelphia	2006	1,120,000	96,600	.	332,000	3,480	.	137,000	235	.	27,300
Atlanta	2007	829,000	123,000	.	307,000	18,000	.	165,000	936	.	60,000
Denver	2007	540,000	42,500	.	126,000	1,680	.	26,700	0	.	1,920
Los Angeles	2007	3,500,000	111,000	.	1,170,000	7,730	.	558,000	0	.	209,000
Philadelphia	2007	1,120,000	130,000	.	389,000	20,400	.	190,000	1,460	.	65,900
Atlanta	2008	828,000	24,300	98,700	154,000	1,390	15,100	37,500	76	1,760	5,920
Denver	2008	540,000	31,100	88,500	116,000	871	7,070	16,100	39	390	871
Los Angeles	2008	3,510,000	147,000	182,000	1,270,000	9,390	15,400	651,000	0	224	252,000
Philadelphia	2008	1,120,000	86,600	188,000	306,000	6,860	43,800	123,000	0	3,120	29,500
Atlanta	2009	828,000	.	44,900	86,000	.	5,900	16,300	.	439	2,040
Denver	2009	537,000	.	45,100	65,000	.	3,140	6,030	.	52	195
Los Angeles	2009	3,520,000	.	129,000	1,140,000	.	5,960	534,000	.	0	195,000
Philadelphia	2009	1,120,000	.	33,800	108,000	.	338	7,220	.	0	104
Atlanta	2010	829,000	.	71,100	124,000	.	7,730	21,100	.	592	2,310
Denver	2010	537,000	.	50,200	74,100	.	2,310	5,640	.	0	13
Los Angeles	2010	3,520,000	.	66,000	836,000	.	1,190	292,000	.	0	83,100
Philadelphia	2010	1,120,000	.	213,000	348,000	.	32,800	127,000	.	1,610	17,300
Atlanta	Mean	829,000	82,700	71,600	189,000	11,200	9,580	75,400	707	929	23,700
Denver	Mean	537,000	50,700	61,300	109,000	3,350	4,170	22,400	30	147	2,240
Los Angeles	Mean	3,510,000	148,000	126,000	1,150,000	11,300	7,530	559,000	0	75	218,000
Philadelphia	Mean	1,120,000	105,000	145,000	297,000	10,200	25,600	117,000	564	1,580	28,000

Table 4-11. Percent of people with 1 or more 8-hour exposures above different levels (ppb-8hr), Asthmatic children (moderate exertion)

City	myear	Above 0 base	Above 60 75/4 2006-8	Above 60 75/4 2008-10	Above 60 base	Above 70 75/4 2006-8	Above 70 75/4 2008-10	Above 70 base	Above 80 75/4 2006-8	Above 80 75/4 2008-10	Above 80 base
Atlanta	2006	96.9%	11.7%	.	32.8%	1.7%	.	16.0%	0.1%	.	5.6%
Denver	2006	96.3%	14.9%	.	30.8%	1.3%	.	11.0%	0.1%	.	1.5%
Los Angeles	2006	97.7%	5.1%	.	38.0%	0.6%	.	21.8%	0.0%	.	10.4%
Philadelphia	2006	96.7%	8.6%	.	29.4%	0.3%	.	12.5%	0.0%	.	2.6%
Atlanta	2007	97.0%	15.0%	.	36.6%	1.7%	.	19.8%	0.1%	.	7.3%
Denver	2007	96.5%	7.6%	.	23.7%	0.3%	.	5.1%	0.0%	.	0.3%
Los Angeles	2007	98.0%	3.5%	.	32.5%	0.4%	.	16.7%	0.0%	.	6.6%
Philadelphia	2007	96.9%	12.4%	.	35.2%	1.8%	.	17.9%	0.1%	.	6.0%
Atlanta	2008	97.0%	3.0%	11.8%	18.4%	0.2%	2.0%	4.6%	0.0%	0.2%	0.6%
Denver	2008	96.8%	6.0%	16.5%	22.2%	0.1%	1.4%	3.3%	0.0%	0.1%	0.2%
Los Angeles	2008	97.2%	4.0%	5.0%	36.9%	0.4%	0.5%	18.2%	0.0%	0.0%	6.9%
Philadelphia	2008	97.1%	7.6%	17.0%	27.9%	0.6%	4.1%	10.8%	0.0%	0.3%	2.8%
Atlanta	2009	97.3%	.	5.4%	10.1%	.	0.6%	1.7%	.	0.0%	0.1%
Denver	2009	96.2%	.	8.3%	11.6%	.	0.6%	1.1%	.	0.0%	0.1%
Los Angeles	2009	97.3%	.	3.6%	32.3%	.	0.1%	15.2%	.	0.0%	5.4%
Philadelphia	2009	96.7%	.	3.0%	9.5%	.	0.0%	0.7%	.	0.0%	0.0%
Atlanta	2010	97.3%	.	8.9%	15.1%	.	0.8%	2.4%	.	0.1%	0.2%
Denver	2010	96.5%	.	8.6%	13.0%	.	0.4%	1.1%	.	0.0%	0.0%
Philadelphia	2010	97.0%	.	18.6%	30.5%	.	2.6%	10.8%	.	0.2%	1.4%
Atlanta	Mean	97.1%	9.9%	8.7%	22.7%	1.2%	1.1%	9.0%	0.1%	0.1%	2.8%
Denver	Mean	96.5%	9.4%	11.2%	20.3%	0.6%	0.8%	4.3%	0.0%	0.0%	0.4%
Los Angeles	Mean	97.5%	4.2%	3.4%	.	0.4%	0.2%	.	0.0%	0.0%	.
Philadelphia	Mean	96.9%	9.5%	12.9%	26.4%	0.9%	2.2%	10.5%	0.0%	0.1%	2.5%

Table 4-12. Number of people with 1 or more 8-hour exposures above different levels (ppb-8hr), Asthmatic children (moderate exertion)

City	myear	Above 0 base	Above 60 75/4 2006-8	Above 60 75/4 2008-10	Above 60 base	Above 70 75/4 2006-8	Above 70 75/4 2008-10	Above 70 base	Above 80 75/4 2006-8	Above 80 75/4 2008-10	Above 80 base
Atlanta	2006	83,900	10,200	.	28,400	1,510	.	13,800	76	.	4,830
Denver	2006	47,800	7,380	.	15,300	643	.	5,440	26	.	720
Los Angeles	2006	311,000	16,300	.	121,000	1,780	.	69,300	0	.	33,100
Philadelphia	2006	129,000	11,500	.	39,300	419	.	16,700	26	.	3,430
Atlanta	2007	83,900	13,000	.	31,700	1,490	.	17,100	57	.	6,300
Denver	2007	48,800	3,840	.	12,000	169	.	2,570	0	.	169
Los Angeles	2007	312,000	11,000	.	103,000	1,120	.	53,000	0	.	21,100
Philadelphia	2007	128,000	16,300	.	46,500	2,340	.	23,700	104	.	7,910
Atlanta	2008	83,900	2,580	10,200	15,900	172	1,760	3,950	19	210	554
Denver	2008	48,900	3,020	8,320	11,200	65	728	1,680	0	26	91
Los Angeles	2008	318,000	13,000	16,300	121,000	1,270	1,790	59,800	0	149	22,700
Philadelphia	2008	131,000	10,200	22,900	37,600	831	5,460	14,500	0	338	3,720
Atlanta	2009	81,200	.	4,540	8,450	.	496	1,450	.	0	114
Denver	2009	47,700	.	4,100	5,760	.	298	532	.	0	26
Los Angeles	2009	319,000	.	11,800	106,000	.	373	49,700	.	0	17,700
Philadelphia	2009	130,000	.	4,050	12,800	.	52	909	.	0	26
Atlanta	2010	81,300	.	7,460	12,600	.	649	2,040	.	57	153
Denver	2010	47,800	.	4,270	6,420	.	182	558	.	0	0
Philadelphia	2010	131,000	.	25,000	41,000	.	3,530	14,600	.	234	1,870
Atlanta	Mean	82,900	8,560	7,390	19,400	1,060	967	7,680	51	89	2,390
Denver	Mean	48,200	4,740	5,560	10,100	292	403	2,160	9	9	201
Los Angeles	Mean	315,000	13,400	11,100	.	1,390	770	.	0	50	.
Philadelphia	Mean	130,000	12,700	17,300	35,400	1,200	3,010	14,100	43	191	3,390

Table 4-17. Percent of people with 1 or more 8-hour exposures above different levels (ppb-8hr), All people (moderate exertion)

City	myear	Above 0 base	Above 60 75/4 2006-8	Above 60 75/4 2008-10	Above 60 base	Above 70 75/4 2006-8	Above 70 75/4 2008-10	Above 70 base	Above 80 75/4 2006-8	Above 80 75/4 2008-10	Above 80 base
Atlanta	2006	80.5%	7.7%	.	21.6%	1.2%	.	10.4%	0.1%	.	3.6%
Denver	2006	79.4%	8.6%	.	18.1%	0.9%	.	6.3%	0.0%	.	0.9%
Los Angeles	2006	81.0%	3.2%	.	21.1%	0.4%	.	11.5%	0.0%	.	5.4%
Philadelphia	2006	76.5%	4.6%	.	17.0%	0.2%	.	6.5%	0.0%	.	1.3%
Atlanta	2007	80.7%	8.1%	.	22.9%	1.1%	.	10.9%	0.1%	.	3.6%
Denver	2007	79.4%	4.6%	.	13.4%	0.2%	.	2.7%	0.0%	.	0.2%
Los Angeles	2007	80.9%	2.1%	.	18.7%	0.1%	.	8.8%	0.0%	.	3.4%
Philadelphia	2007	76.6%	6.4%	.	20.2%	0.9%	.	9.2%	0.1%	.	3.1%
Atlanta	2008	80.5%	2.1%	8.0%	12.2%	0.1%	1.4%	3.1%	0.0%	0.2%	0.6%
Denver	2008	79.5%	3.7%	10.2%	13.4%	0.1%	0.9%	2.0%	0.0%	0.0%	0.1%
Los Angeles	2008	80.9%	2.7%	3.3%	21.0%	0.2%	0.3%	10.4%	0.0%	0.0%	4.3%
Philadelphia	2008	76.5%	4.3%	9.5%	15.6%	0.4%	2.2%	6.1%	0.0%	0.2%	1.5%
Atlanta	2009	80.7%	.	3.5%	6.5%	.	0.6%	1.3%	.	0.0%	0.2%
Denver	2009	79.7%	.	4.9%	7.1%	.	0.4%	0.8%	.	0.0%	0.0%
Los Angeles	2009	81.0%	.	2.4%	18.0%	.	0.1%	8.3%	.	0.0%	3.2%
Philadelphia	2009	76.3%	.	1.7%	5.3%	.	0.0%	0.5%	.	0.0%	0.0%
Atlanta	2010	80.8%	.	5.2%	9.3%	.	0.5%	1.5%	.	0.0%	0.1%
Denver	2010	79.7%	.	6.1%	8.8%	.	0.3%	0.8%	.	0.0%	0.0%
Los Angeles	2010	81.0%	.	1.2%	13.3%	.	0.0%	4.8%	.	0.0%	1.4%
Philadelphia	2010	76.6%	.	10.4%	17.7%	.	1.6%	6.1%	.	0.1%	0.9%
Atlanta	Mean	80.7%	6.0%	5.6%	14.5%	0.8%	0.8%	5.5%	0.0%	0.1%	1.6%
Denver	Mean	79.6%	5.6%	7.1%	12.1%	0.4%	0.6%	2.5%	0.0%	0.0%	0.3%
Los Angeles	Mean	81.0%	2.7%	2.3%	18.4%	0.2%	0.2%	8.8%	0.0%	0.0%	3.5%
Philadelphia	Mean	76.5%	5.1%	7.2%	15.2%	0.5%	1.3%	5.7%	0.0%	0.1%	1.4%

Table 4-19. Number of people with 1 or more 8-hour exposures above different levels (ppb-8hr), All people (moderate exertion)

City	myear	Above 0 base	Above 60 75/4 2006-8	Above 60 75/4 2008-10	Above 60 base	Above 70 75/4 2006-8	Above 70 75/4 2008-10	Above 70 base	Above 80 75/4 2006-8	Above 80 75/4 2008-10	Above 80 base
Atlanta	2006	3,080,000	294,000	.	826,000	45,200	.	396,000	3,040	.	138,000
Denver	2006	2,040,000	221,000	.	465,000	23,500	.	161,000	527	.	24,200
Los Angeles	2006	12,100,000	474,000	.	3,140,000	62,500	.	1,720,000	223	.	805,000
Philadelphia	2006	4,000,000	240,000	.	888,000	11,900	.	339,000	785	.	69,900
Atlanta	2007	3,080,000	309,000	.	874,000	42,800	.	418,000	1,990	.	139,000
Denver	2007	2,060,000	119,000	.	347,000	5,730	.	71,200	52	.	5,920
Los Angeles	2007	12,000,000	310,000	.	2,780,000	20,300	.	1,300,000	0	.	501,000
Philadelphia	2007	3,990,000	333,000	.	1,050,000	44,700	.	480,000	3,330	.	160,000
Atlanta	2008	3,080,000	82,000	304,000	466,000	4,280	53,500	119,000	76	5,750	21,700
Denver	2008	2,070,000	95,300	264,000	349,000	2,640	24,200	51,000	39	1,210	2,630
Los Angeles	2008	12,100,000	409,000	496,000	3,130,000	28,000	47,000	1,550,000	0	447	639,000
Philadelphia	2008	3,970,000	224,000	492,000	808,000	18,500	114,000	316,000	78	10,900	76,900
Atlanta	2009	3,080,000	.	135,000	249,000	.	21,100	51,500	.	1,200	6,560
Denver	2009	2,070,000	.	128,000	184,000	.	11,400	20,000	.	584	947
Los Angeles	2009	12,100,000	.	360,000	2,680,000	.	22,100	1,240,000	.	0	477,000
Philadelphia	2009	3,970,000	.	89,200	277,000	.	2,000	24,900	.	0	987
Atlanta	2010	3,080,000	.	200,000	356,000	.	19,600	58,400	.	1,200	5,270
Denver	2010	2,070,000	.	159,000	227,000	.	8,380	20,300	.	0	91
Los Angeles	2010	12,100,000	.	183,000	1,990,000	.	3,500	722,000	.	0	211,000
Philadelphia	2010	3,980,000	.	541,000	922,000	.	85,500	315,000	.	3,820	44,500
Atlanta	Mean	3,080,000	228,000	213,000	554,000	30,800	31,400	209,000	1,700	2,720	62,200
Denver	Mean	2,060,000	145,000	184,000	314,000	10,600	14,700	64,700	206	598	6,750
Los Angeles	Mean	12,100,000	398,000	347,000	2,750,000	37,000	24,200	1,310,000	74	149	527,000
Philadelphia	Mean	3,980,000	266,000	374,000	789,000	25,000	67,300	295,000	1,400	4,910	70,400

5.6.3 Characterization Of Factors Influencing High Exposures

In this analysis, we investigated the particular factors that influence estimated exposures with a focus on persons experiencing the highest daily maximum 8-hour exposures within each study area. This analysis required the generation of detailed APEX output files having varying time intervals, that is, the daily, hourly, and minute-by-minute (or *events*) files. Given that the size of these time-series files is dependent on the number of persons simulated, we simulated 5,000 persons and restricted the analysis to a single year (2006) to make this evaluation tractable.[8] Both the base case (unadjusted or 'as is' recent air quality conditions) and ambient O_3 adjusted to just meet the current standard (0.075 ppm) air quality scenarios were evaluated in each of the four study areas. All APEX conditions (e.g., ME descriptions, AERs, MET data) were consistent with the 200,000 person APEX simulations that generated all of summary output discussed in the main body of this chapter.

We were interested in identifying the specific microenvironments and activities most important to O_3 exposure and evaluating their duration and particular times of the day persons were engaged in them. Because ambient O_3 concentrations peak mainly in the afternoon hours, we focused our microenvironmental time expenditure analysis on the hours between 12PM and 8PM. For every day of the exposure simulation, we aggregated the time spent outdoors, indoors, near-roadways, and inside vehicles during these afternoon hours (i.e., the time of interest summed to 480 minutes per person day). Data from several APEX output files were then combined to generate a single daily file for each person containing a variety of personal attributes (e.g., age, sex), their daily maximum 8-hour ambient and exposure concentrations, and the aforementioned time expenditure metrics.

We performed an analysis of variance (ANOVA) using SAS PROC GLM (SAS, 2012) to determine the factors contributing most to variability in the dependent variable, i.e., each person's daily maximum 8-hour O_3 exposure concentrations. This analysis was distinct for five

[8] We recognize that there is year-to-year variability in ambient O_3 concentrations and it is possible that fewer persons simulated could result in differences in exposures compared to large-scale multi-year model simulations. Based on a similar detailed evaluation performed for the Carbon Monoxide REA (US EPA, 2010), it is expected any differences that exist between exposures estimated in a large simulation versus that using a smaller subset of persons would be small and of limited importance to this particular evaluation.

age-groups of interest (<5, 5-17, 18-35, 36-65, >65 years of age). The final models[9] included a total of seven explanatory variables: the main effects of (1) daily maximum 8-hour ambient O_3, (2-4) afternoon time spent outdoors, near-roads, and inside vehicles,[10] and (5) PAI, while also including interaction effects from (6) afternoon time outdoors by daily maximum 8-hour ambient concentration and (7) PAI by afternoon time outdoors. Two conditions were considered: all person days of the simulation, and only those days where a person's 8-hour maximum exposure concentration was ≥0.05 ppm.[11] Selected output from this ANOVA included parameter estimates for each variable, model R-square statistic (R^2), and Type III model sums of squares (SS3).[12]

Model fits, as indicated by an R^2 value, were reasonable across each of the study areas (Table 5-5). The selected factors explain about 40-80% of the total variability in 8-hour daily maximum exposures. Model fits were best when using all person days of the simulation and results were similar for both air quality scenarios. When considering only those days where persons had 8-hour daily maximum O_3 exposures ≥0.05 ppm, consistently less variability was explained by the factors included in each model, though overall model fits were acceptable. Furthermore, the most robust models were those developed using either children aged 5-17 or adults 18-35 years old (e.g., see Table 5-6 for Los Angeles model R^2 by age groups).

Table 5-5. Range of ANOVA model R^2 fit statistics by study area, air quality scenario, and exposure level.

	Base Case Model R^2		Current Standard Model R^2	
Study Area	**All Person Days**	**Person Days with 8-hour Exposure ≥ 0.05 ppm**	**All Person Days**	**Person Days with 8-hour Exposure ≥ 0.05 ppm**
Atlanta	0.64 – 0.75	0.55 – 0.63	0.62 – 0.74	0.52 – 0.64
Denver	0.62 – 0.69	0.41 – 0.62	0.61 – 0.68	0.45 – 0.62
Los Angeles	0.72 – 0.79	0.47 – 0.68	0.69 – 0.76	0.54 – 0.66
Philadelphia	0.65 – 0.71	0.43 – 0.64	0.63 – 0.69	0.41 – 0.64

[9] In this investigation, we also evaluated the influence of sex, work and home districts, meteorological zones, each with varying statistical significance, though overall adding little to explaining variability beyond the final explanatory variables included.

[10] Including indoor afternoon time creates a strict linear dependence among these four variables and generates biased estimates, thus it was neither included nor needed in this analysis.

[11] This breakpoint was selected due to the limited sample size (5,000 total simulated persons), an issue of increasing importance when selecting for persons with the highest exposures.

[12] In each of the ANOVA models constructed, type II = type III = type IV sums of squares.

Table 5-6. ANOVA model R^2 fit statistics in Los Angeles by age group, air quality scenario, and exposure level.

Study Area	Age Group (years)	Base Case Model R^2		Current Standard Model R^2	
		All Person Days	Person Days with 8-hour Exposure ≥ 0.05 ppm	All Person Days	Person Days with 8-hour Exposure ≥ 0.05 ppm
Los Angeles	<5	0.74	0.47	0.71	0.59
	5-17	0.79	0.61	0.76	0.54
	18-36	0.73	0.65	0.70	0.65
	36-64	0.73	0.68	0.70	0.66
	>65	0.72	0.58	0.69	0.62

We evaluated the relative contribution each variable had on the total explained variability using the SS3 in each respective model.[13] As with the R^2 statistics generated above, there were four separate model results generated per study area, with relative contribution results for Los Angeles illustrated in Figure 5-16. When considering all person days of the simulation (left side of figure), the daily maximum 8-hour ambient O_3 concentration variable contributes the greatest to the explained model variance, consistently estimated to be about 80% across all age groups and for either air quality scenario. The interaction of this variable with afternoon outdoor time contributes an additional 10% to the explained variance, indicating that both ambient concentration and time spent outdoors collectively contribute to 90% or more of the explained model variance when evaluating all (both high, mid and low) daily maximum 8-hour O_3 exposure concentrations. The main effect of outdoor time contributed very little to the explained variance under these conditions as did contributions from the other included variables, except for time spent near-roads (about a 5% contribution). These results suggest that when considering the Los Angeles study population broadly, the daily maximum 8-hour ambient O_3 concentration is the most important driver in estimating population exposures O_3, nearly regardless of specific microenvironmental locations where exposure might occur.

When considering only person days having daily maximum 8-hour O_3 exposures ≥ 0.05 ppm and for either air quality scenario in Los Angeles, collectively the main effects of ambient concentration and outdoor time combined with their interaction similarly contribute to approximately 80% of the total explained variance (right side of Figure 5-16). However, the

[13] Type III sums of squares (SS3) for a given effect are adjusted for all other effects evaluated in the model, regardless of whether they contain the given effect or not. Thus the SS3 for each variable represents the individual effect sums of squares that sum to the total effect sums of squares (or the total model explained variance).

main effect of the 8-hour daily maximum ambient O_3 concentration variable has a sharply lower contribution (generally about 5-15%) along with greater contribution from the main effects variable outdoor time (15-20% contribution) and its interaction with the ambient concentration variable (50-60%). These results suggest that for highly exposed persons, the most important drivers are time spent outdoors corresponding with high daily maximum 8-hour ambient O_3 concentrations.

Results for Atlanta were generally similar to Los Angeles (Figure 5-17), with notable differences discussed here.[14] The contribution of the maximum 8-hour ambient O_3 concentration variable to the total explained variance (about 40-50%) was less than that observed in Los Angeles when considering all person days (left side of figures 5-16 and 5-17), while the contribution from the outdoor time/ambient O_3 interaction variable was greater in Atlanta (about 20-40% versus 10% in Los Angeles). This dissimilarity is likely driven by the differences in A/C prevalence rates and AER distributions used for each study area. Los Angeles has lower A/C prevalence and higher AERs, thus a greater contribution to exposure is expected from ambient concentrations by infiltrating to indoor microenvironments and hence, reflected in the strong main effects for the 8-hour daily maximum ambient O_3 concentration variable in Los Angeles. Afternoon time spent near Atlanta roads was estimated to contribute to about 20-30% of the total explained variance when considering all person days and exposures, a value greater than that estimated for Los Angeles (generally about 5%) again possibly reflecting an increased importance of outdoor microenvironments in Atlanta relative to that in Los Angeles and the other study locations (not shown).

Because afternoon outdoor time expenditure and 8-hour daily maximum ambient O_3 concentrations are an important determinant for maximum O_3 exposures regardless of air quality scenario, we compared the distributions of the two variables considering person day exposures below and at or above 0.05 ppm. Figure 5-18 presents an example of this comparison for Los

[14] This discussion regarding the relative contribution of the variables to the total explained model variance also applies to the other two study areas, whereas results for Denver and Philadelphia were generally similar to Los Angeles. While A/C prevalence is greatest in Philadelphia compared to LA and Denver, the AER distributions are identical to those used for Denver and similar to LA.

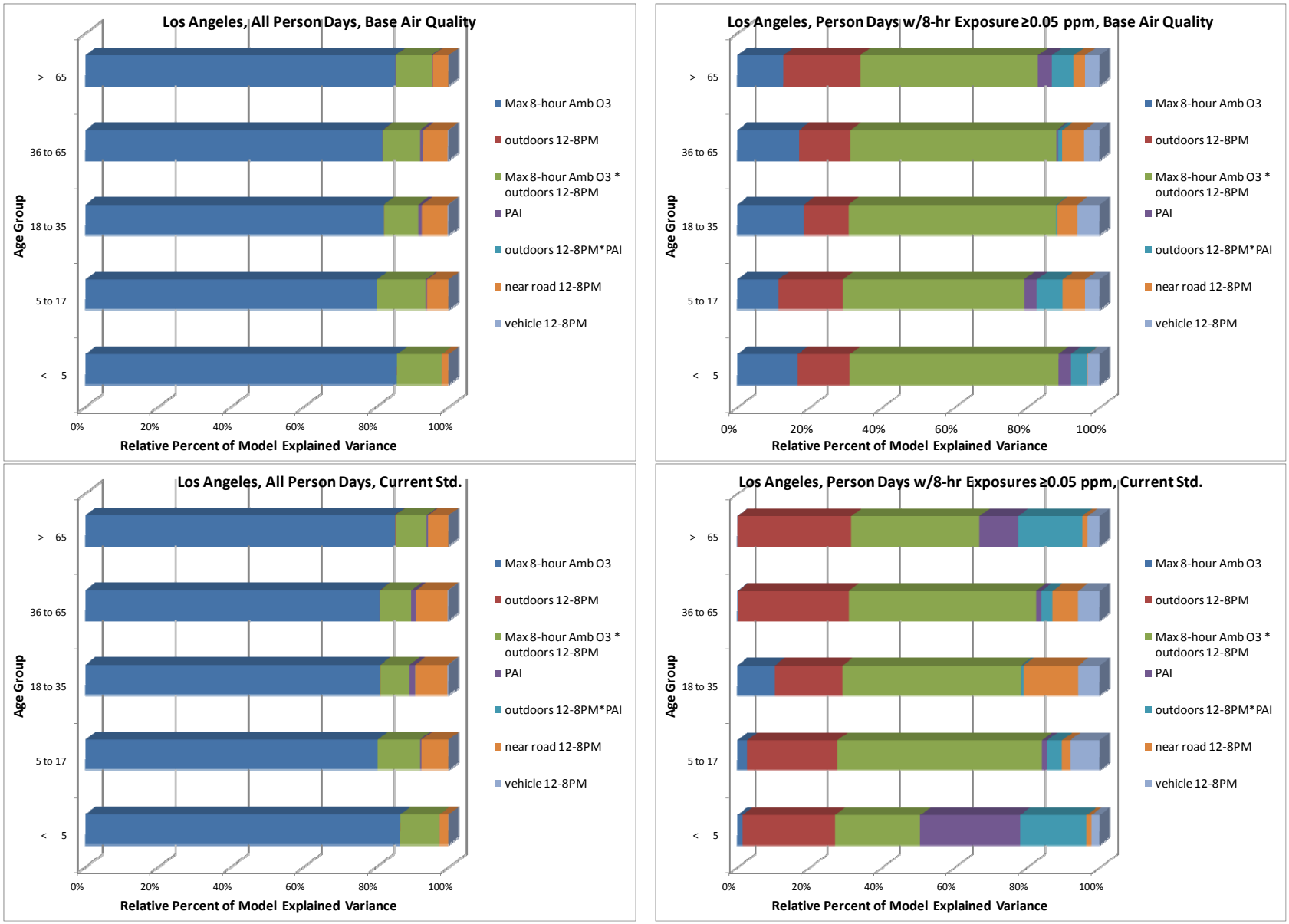


Figure 5-16. Contribution of individual variables to total model explained variance by age group, air quality scenario, exposure level in Los Angeles.

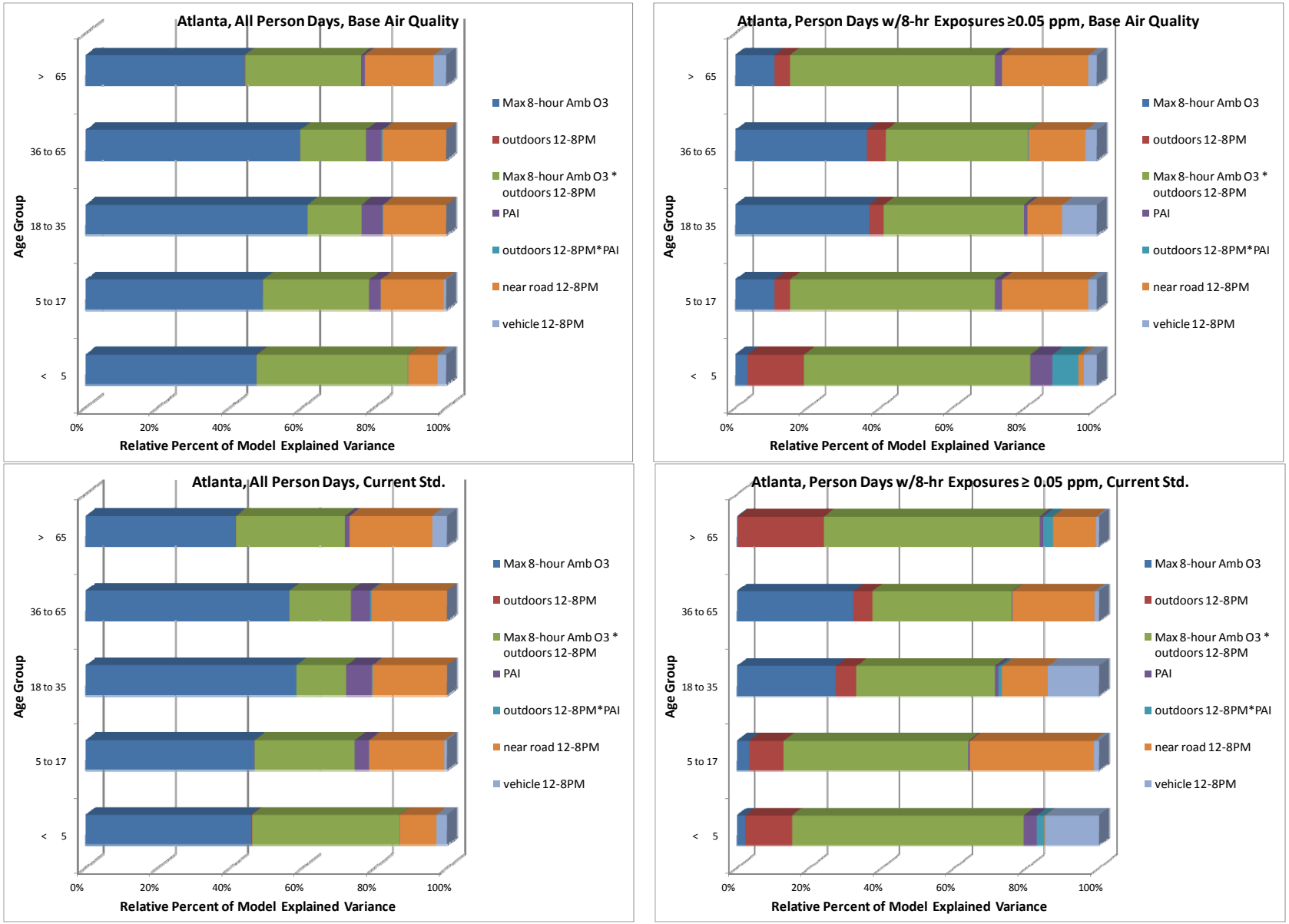
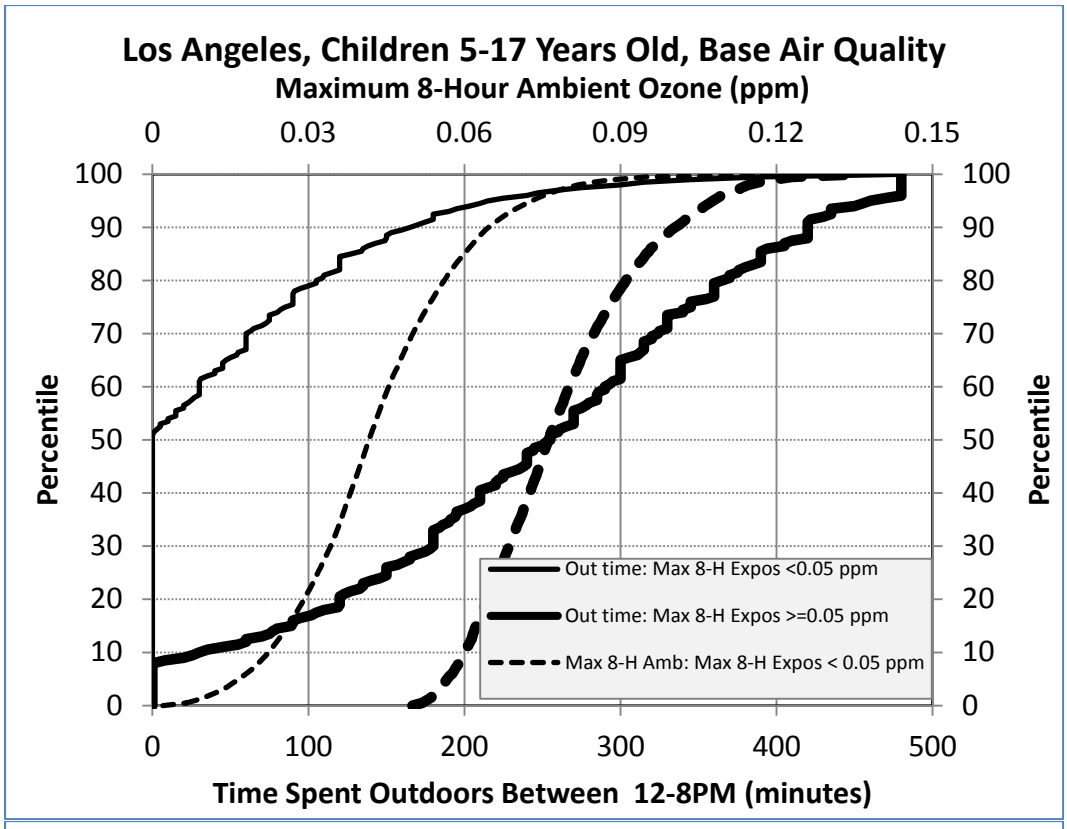


Figure 5-17. Contribution of individual variables to total model explained variance by age group, air quality scenario, exposure level in Atlanta.

Angeles children[15] and considering the base air quality for year 2006 (top). Not surprising, the distributions for both the outdoor time and ambient concentration variables are shifted to the right of the figure for person days where 8-hour daily maximum exposures ≥ 0.05 ppm, as more than half of the days, simulated persons spend about 250 minutes outdoors during the afternoon hours along with experiencing daily maximum 8-hour ambient O_3 concentrations ≥ 0.075 ppm. For days where daily maximum 8-hour O_3 exposure ≤ 0.05 ppm, greater than half of the person days had no time spent outdoors and 8-hour daily maximum ambient O_3 concentrations ≤ 0.045 ppm. By design, when air quality is simulated to just meet the current standard (Figure 5-18, bottom) upper percentile ambient concentrations are dramatically reduced compared to those comprising the base air quality such that the majority of concentrations fall well below the current standard level of 0.075 ppm. Given so few occurrences of very high 8-hour ambient O_3 concentrations for this air quality scenario, only those persons having a majority of their time spent outdoors experienced the highest 8-hour O_3 exposure concentrations.

By definition, an 8-hour exposure is time-averaged across all microenvironmental concentrations therefore several different microenvironments may contribute to each person's daily maximum level. Understandably based on the above analysis, the outdoor microenvironment is the most important for those having the highest O_3 exposures, but we are also interested in the percentage of time expenditure spent among detailed indoor, outdoor, and vehicular locations people may inhabit during the afternoon. As an example, Figure 5-19 presents this information for Los Angeles children (ages 5-17) having daily maximum 8-hour average O_3 exposures ≥ 0.05 ppm and considering base air quality conditions. On average, approximately 50% of total afternoon time is spent outdoors, of which half of this portion is spent outdoors at home, with parks and other non-residential outdoor locations comprising the remaining portion. Approximately 40% of the children's time on high exposure days is spent indoors, while only 10% of time is spent near-roads or inside motor vehicles. Afternoon microenvironmental time expenditure for highly exposed adults in Los Angeles was generally similar with these estimates (data not shown).

[15] The overall features of these two outdoor time and ambient concentration distributions are similar in the other study areas (data not shown).



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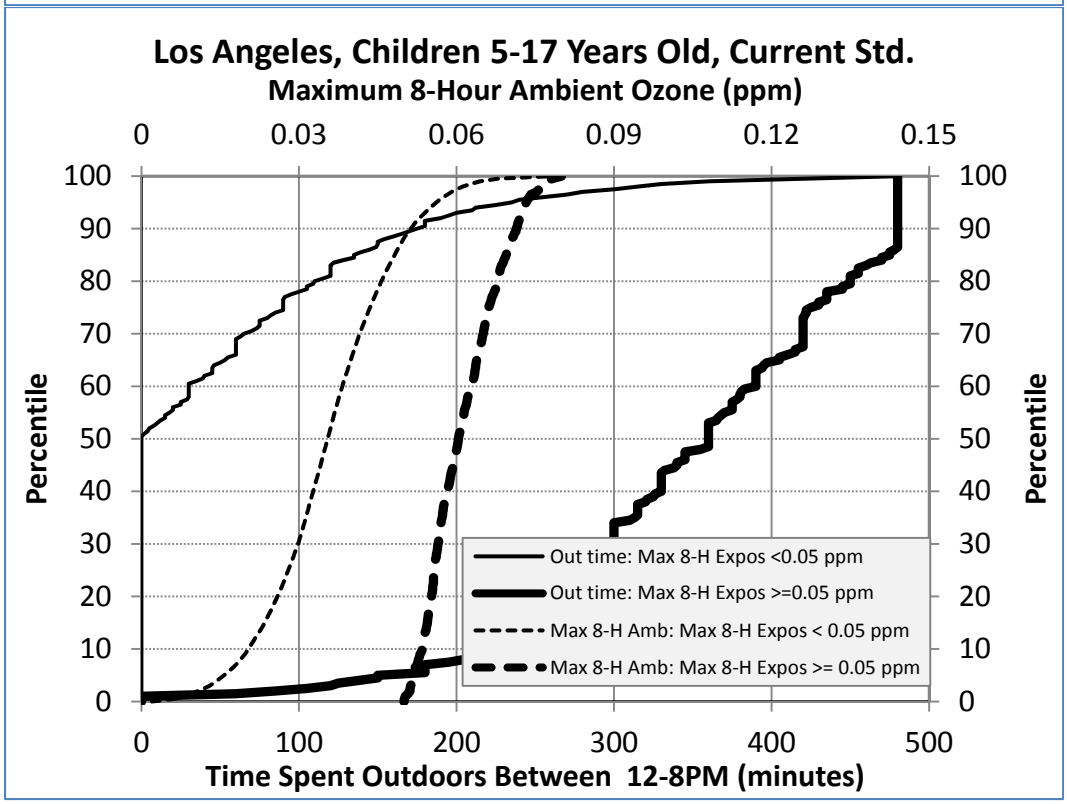


2

3 Figure 5-18. Distributions of afternoon outdoor time expenditure and 8-hour daily maximum
4 ambient O_3 concentrations for Los Angeles children (0-17) person days with 8-hour daily
5 maximum exposures ≥ 0.05 ppm.

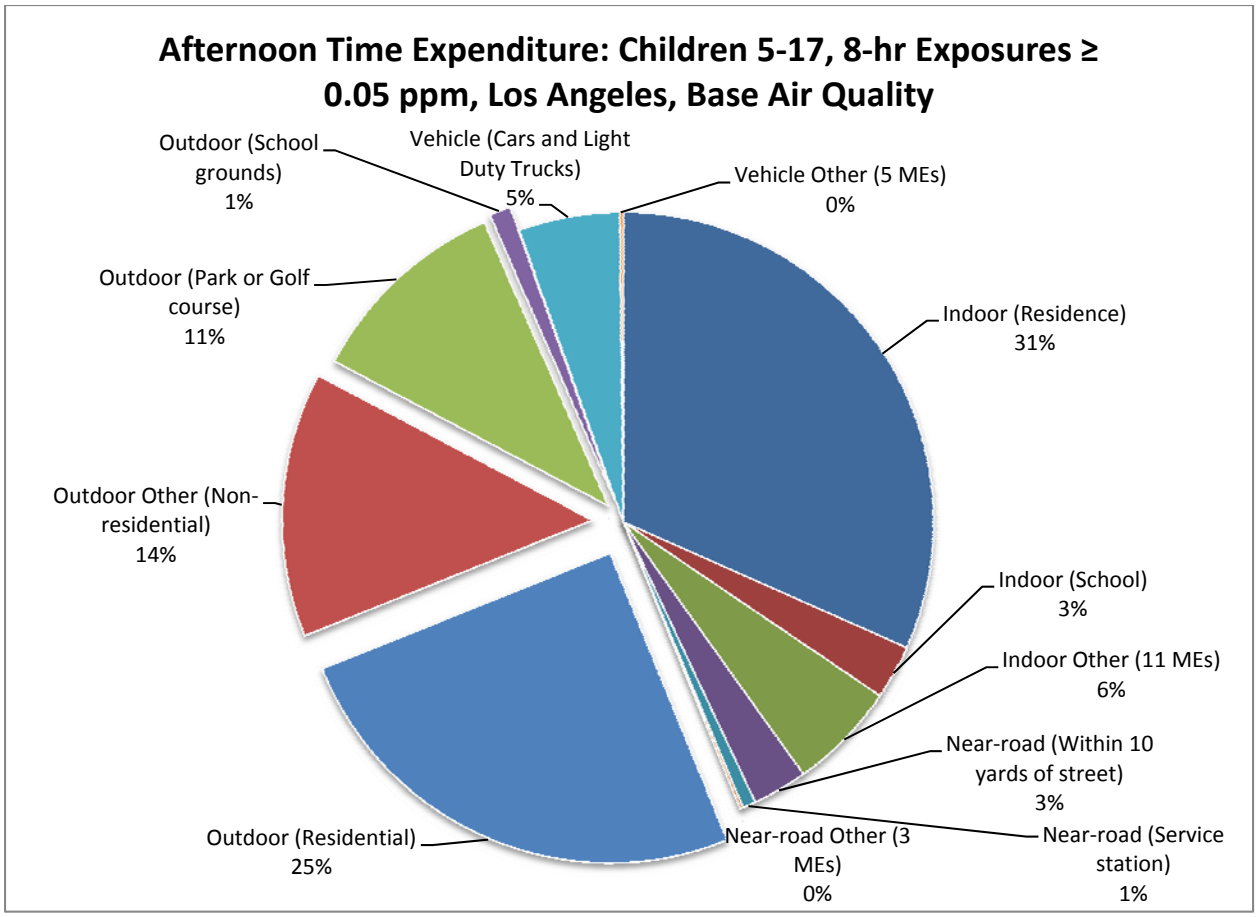


Figure 5-19. Afternoon microenvironmental time expenditure for Los Angeles children (ages 5-17) experiencing 8-hour daily maximum O_3 exposures ≥ 0.05 ppm, base air quality.

A person's activity level plays an important role in estimating the risk of adverse health responses. As such, we evaluated the activities performed by highly exposed individuals while they spent time outdoors during the afternoon hours. Note there are over 100 specific activity codes used in CHAD/APEX, though not all of these will be used in an exposure modeling simulation depending on the diaries that are selected to represent the simulated population. We summed the time spent in each specific activity across all highly exposed persons that spent time outdoors, ranked them, and identified the top ten activities performed. An aggregate of any remaining less often performed activities was generated to complete this analysis of activity time expenditure.

Figure 5-20 shows results for Los Angeles children, indicating that greater than half of the time highly exposed children spent outdoors specifically involves performing a moderate or greater exertion level activity, such as a sporting activity. The same type of analysis was done

for highly exposed adults in Los Angeles (Figure 5-21), whereas about 25% of the outdoor time expenditure was spent engaged in a paid work related activity (though not necessarily a high exertion level activity), 20% of the time was spent playing sports or other moderate or greater exertion level activity, with much of the remaining specific activities associated with low exertion level (e.g., eating, sitting, visiting) or other less frequently performed activities of variable exertion level.

These results support our earlier assessment results in identifying children as an important exposure population group, largely a result of the combined outdoor time expenditure along with concomitantly performing moderate or high exertion level activities. However, one issue not explicitly addressed in the exposure modeling and remaining as a limitation to the results is that outdoor workers are not addressed by our modeling.

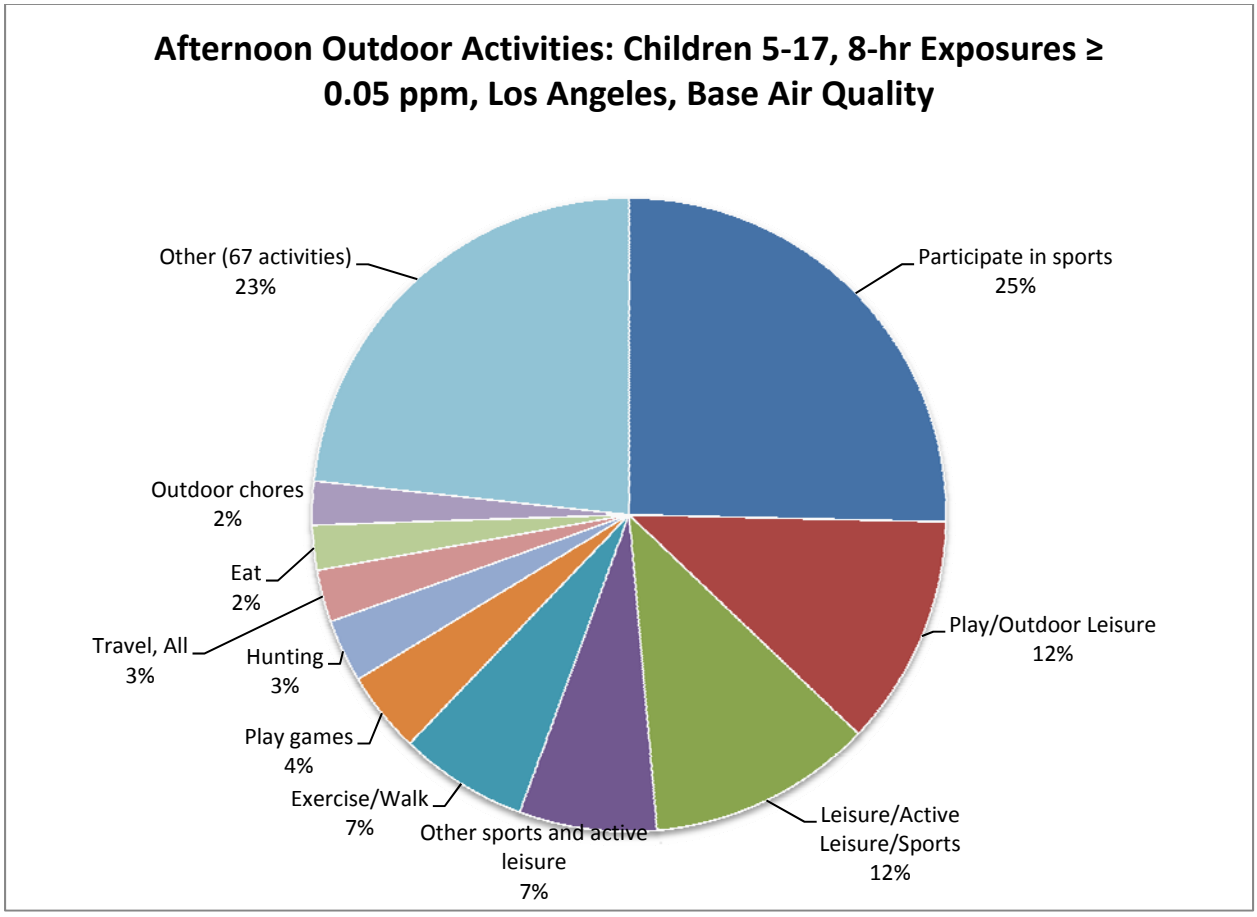


Figure 5-20. Activities performed during afternoon time outdoors for Los Angeles children (ages 5-17) experiencing 8-hour daily maximum O_3 exposures ≥ 0.05 ppm, base air quality.

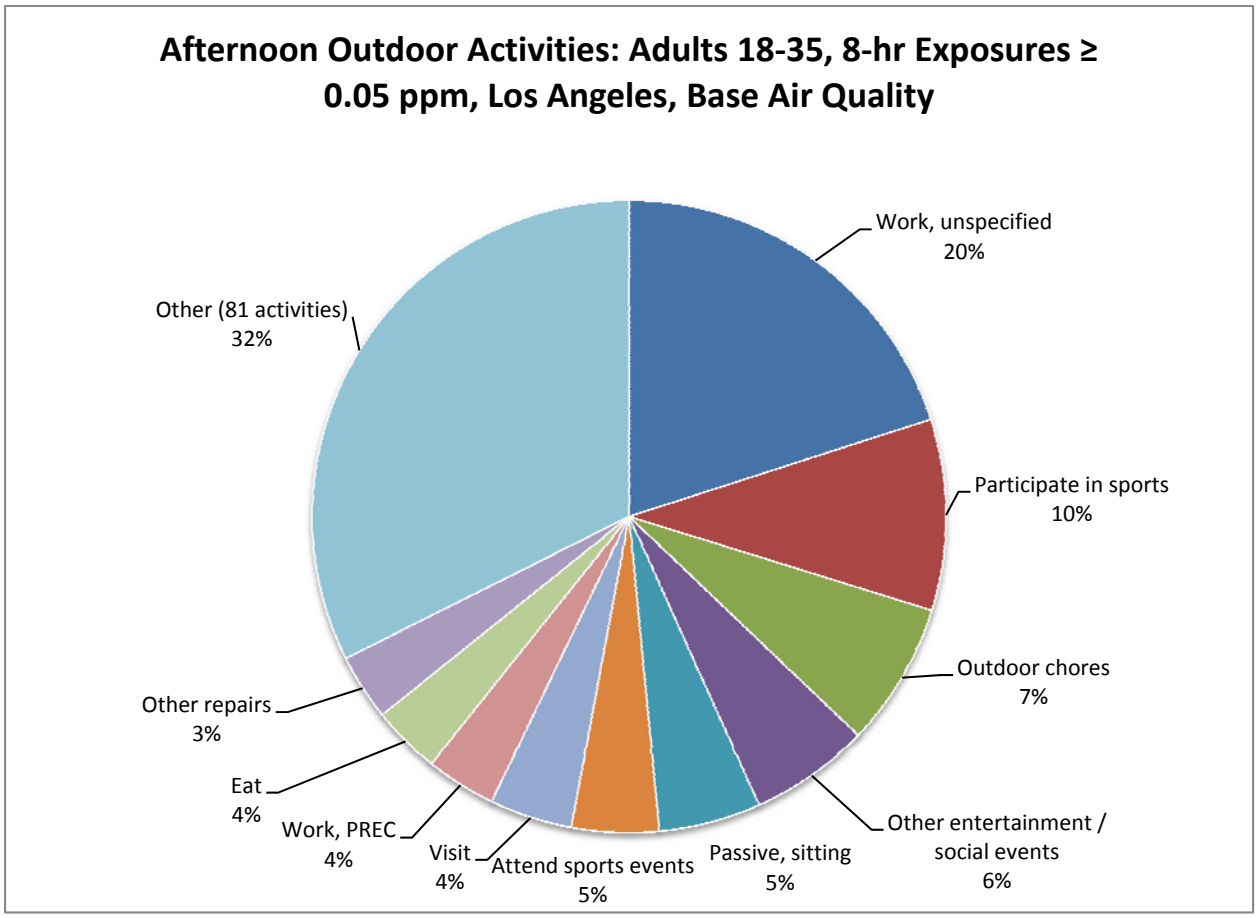


Figure 5-21. Activities performed during afternoon time outdoors for Los Angeles adults (ages 18-35) experiencing 8-hour daily maximum O_3 exposures ≥ 0.05 ppm, base air quality.

5.6.4 Discussion of Exposure Modeling Results

The patterns of estimated exposures are variable from city to city, primarily due to differences in air quality (local emissions and meteorology affect these), the rollback procedure as applied to each separate area, and people's time-location-activity patterns. Inspection of Figures 4-1 to 4-15 shows marked differences between urban areas in the levels of exposures, both for the base case and current standard scenarios. For example, under the current standard, it is estimated that 14 percent of the Denver children but very few of the Los Angeles children experience 8-hr O_3 exposures above 0.06 ppm-8hr while engaged in moderate exertion based on 2006. In 2007, the percents of exposures above 0.06 ppm-8hr ranged from 14 percent in Atlanta to 3 percent in Los Angeles; in 2010 the percents ranged from 18 percent in Philadelphia to 2 percent in Los Angeles. Los Angeles in most cases has a smaller percent of children with exposures above 0.06 and 0.07 ppm-8hr than the other cities. In Los Angeles, because of the highly skewed nature of the distribution of ozone concentrations, much more of the upper range of the air quality distribution needed to be rolled back to allow for the meeting of the current standards, thus significantly reducing the frequency of occurrence of high ambient concentrations (and therefore exposures).

After simulating just meeting the current standard, estimates of exposures above 0.07 ppm-8hr while engaged in moderate exertion are 2 percent or below, except for Philadelphia, which has estimates of 4 percent in 2008 and 3 percent in 2010 for children. Estimates of exposures above 0.08 ppm-8hr while engaged in moderate exertion are less than 0.5 percent for all cities and years after simulating just meeting the current standard.

As discussed in Chapter 3, multiple exposures pose a greater health concern than single exposures. However, multiple repeated exposures are greatly underestimated by APEX (Langstaff, 2007, p. 49-50). This underestimation results primarily from the way that people's activities are modeled using CHAD, which does not properly account for repeated behavior of individuals. Repeated routine behavior from one weekday to the next is not simulated. For example, there are no simulated individuals representing children in summer camps who spend a large portion of their time outdoors, or adults with well-correlated weekday schedules. These limitations apply to both children and adults, and therefore multiple exposures to children are also expected to be underestimated by APEX. The second draft REA will provide quantitative

estimates of the extent of repeated exposures for selected populations for which sequences of daily activities can be reliably constructed.

The year-to-year variability in exposures in recent years, due in varying degrees to changes in weather and emissions of precursors to O_3, can be seen in Figures 5-22 to 5-25, which show results for the 2006 to 2010 base case scenarios for each urban area and illustrate the range of exposures generated by the use of multiple years of ambient air quality data. These figures show the percent of school-age children who experience at least one 8-hour average exposure above levels ranging from 0.04 to 0.08 ppm-8hr, with all five years presented in each graph. Figure 5-22 illustrates the estimates of the percent of children in Atlanta who experience 8-hr O_3 exposures above levels ranging from 0.04 to 0.08 ppm-8hr while engaged in moderate exertion. Each line represents the estimates for one year, from 2006 to 2010. In Atlanta, 2007 had the most exposures, while 2009 saw the least. Figures 5-23, 24, and 25 illustrate these results for Denver, Los Angeles, and Philadelphia. These figures demonstrate that, while different years have the highest and lowest numbers of exposed children for different cities, the trends across exposure levels are similar, both across cities and across years.

The exposure modeling results are discussed further in Chapter 9.

Figure 5-22. Percent of Children (moderate exertion) in Atlanta with at least one 8-hour exposure above different levels, across years

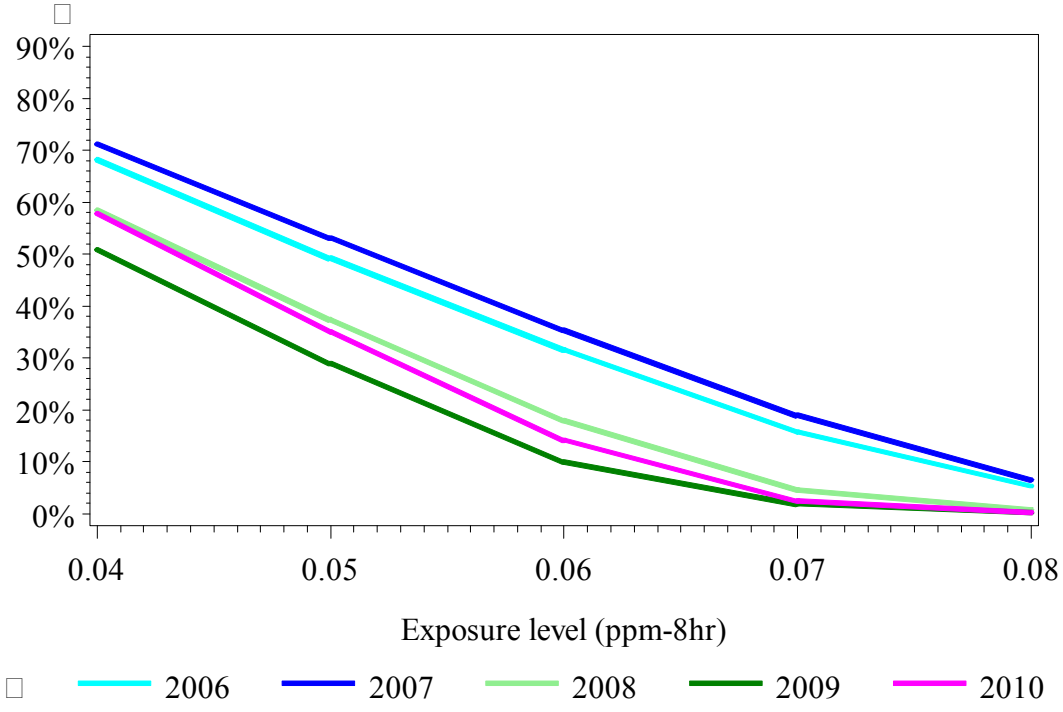


Figure 5-23. Percent of Children (moderate exertion) in Denver with at least one 8-hour exposure above different levels, across years

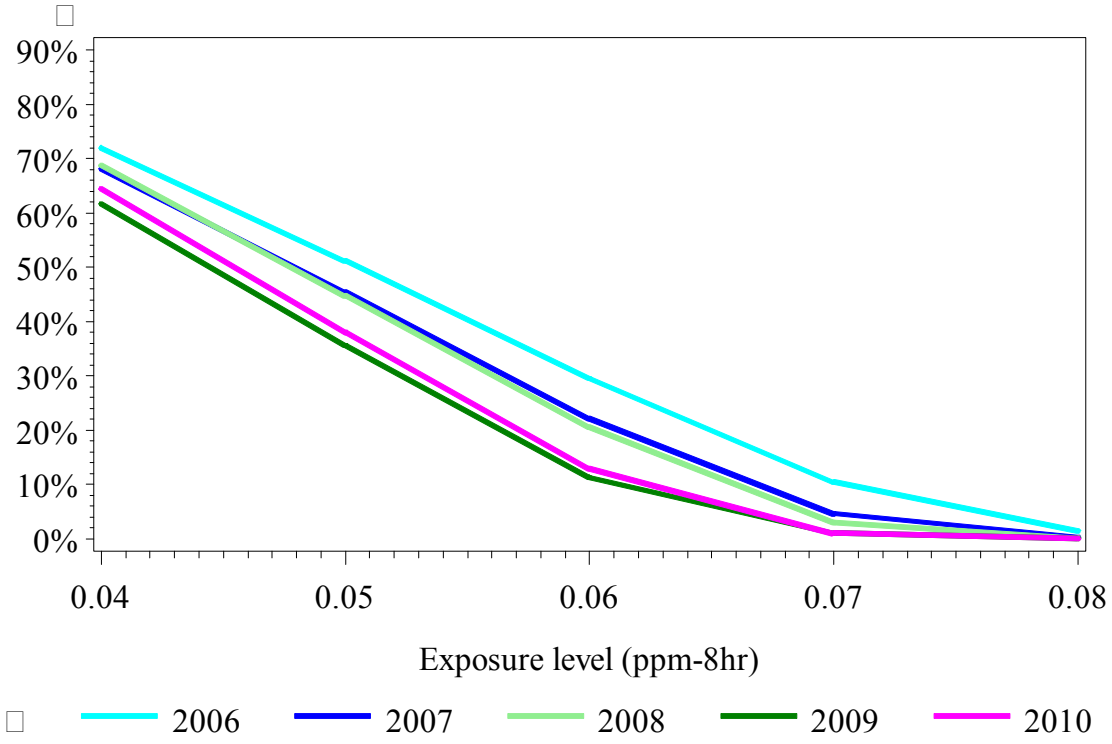


Figure 5-24. Percent of Children (moderate exertion) in Los Angeles with at least one 8-hour exposure above different levels, across years

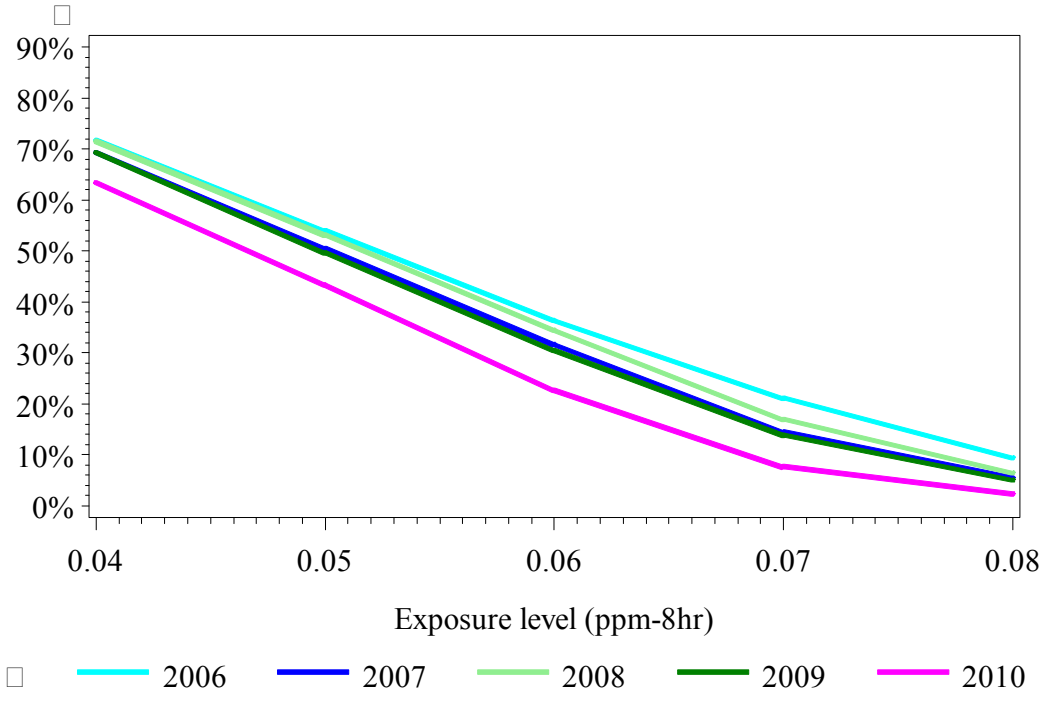
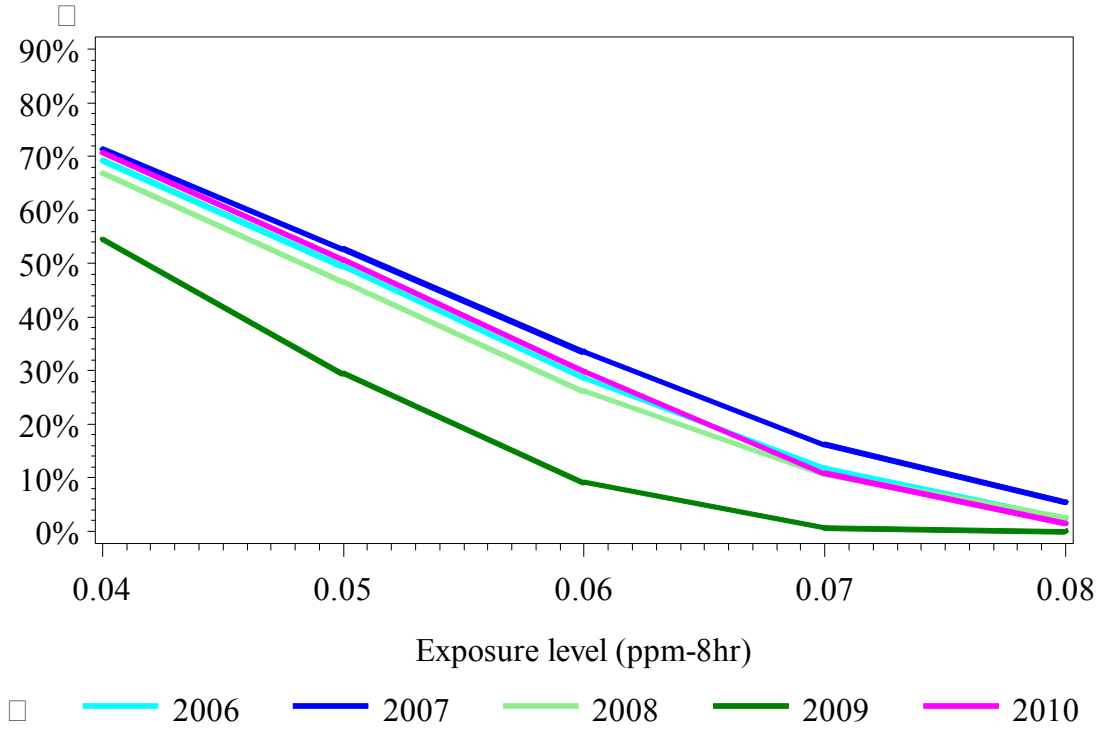


Figure 5-25. Percent of Children (moderate exertion) in Philadelphia with at least one 8-hour exposure above different levels, across years



REFERENCES

Ainsworth, B. E., Haskell, W. L., Leon, A. S., Jacobs, D. R., Jr., Montoye, H. J., Sallis, J. F., Paffenbarger, R. S., Jr. (1993). Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exer*. 25: 71-80.

Akland, G. G., Hartwell, T. D., Johnson, T. R., Whitmore, R. W. (1985). Measuring human exposure to carbon monoxide in Washington, D. C. and Denver, Colorado during the winter of 1982-83. *Environ Sci Technol*. 19: 911-918.

Blanken PD, Dillon J, Wismann G. (2001). The impact of an air quality advisory program on voluntary mobile source air pollution reduction. *Atmos Environ*. 35:2417-2421.

Bresnahan BW, Dickie M, Gerking S. (1997). Averting behavior and air pollution. *Land Econ*. 73:340-357.

Dey, A. N., and Bloom B. (2005). Summary Health Statistics for U.S. Children: National Health Interview Survey, 2003. National Center for Health Statistics. Vital Health Stat 10(223). Available at: http://www.cdc.gov/nchs/data/series/sr_10/sr10_223.pdf

Freedson, P. S. (1989). Field monitoring of physical activity in children. *Pediatr Exerc Sci*. 1:8-18

Graham SE (2012). Comprehensive review of published averting behavior studies and available technical documents. Memo to Bryan Hubbell, Group Leader, Risk and Benefits Group, Office of Air Quality Planning and Standards. EPA docket #

Graham, S. E. and McCurdy, T. (2004). Developing meaningful cohorts for human exposure models. *J Expo Anal Environ Epidemiol*. 14: 23-43.

Hartwell, T. D., Clayton, C. A., Ritchie, R. M., Whitmore, R. W., Zelon, H. S., Jones, S. M., Whitehurst, D. A. (1984). Study of Carbon Monoxide Exposure of Residents of Washington, DC and Denver, Colorado. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development, Environmental Monitoring Systems Laboratory. EPA-600/4-84-031.

Houtven GV, Johnson FR, Mansfiled C, Yang J-C, Pyles A. (2003). Parental Averting Behavior With Respect to Ozone Alerts. For presentation at AEA/ASSA Meeting, San Diego CA, January 2004.

Isaacs , Kristin, Graham Glen, Thomas Mccurdy and Luther Smith. Modeling energy expenditure and oxygen consumption in human exposure models: accounting for fatigue and EPOC. *Journal of Exposure Science and Environmental Epidemiology* (2008) 18, 289–298.

Johnson, T. (1984). A Study of Personal Exposure to Carbon Monoxide in Denver, Colorado. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Monitoring Systems Laboratory. EPA-600/4-84-014.

Johnson, T. (1989). Human Activity Patterns in Cincinnati, Ohio. Palo Alto, CA: Electric Power Research Institute. EPRI EN-6204.

Johnson, T., Capel, J., McCoy, M. (1996a). Estimation of Ozone Exposures Experienced by Urban Residents Using a Probabilistic Version of NEM and 1990 Population Data. Prepared by International Technology Air Quality Services for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. Contract no. 63-D-30094. April 1996. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr_td.html.

Johnson, T., Capel, J., McCoy, M., Warnasch, J. (1996b). Estimation of Ozone Exposures Experienced by Outdoor Children in Nine Urban Areas Using a Probabilistic Version of NEM. Prepared by International Technology Air Quality Services for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. Contract no. 63-D-30094. April 1996. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr_td.html

Johnson, T., Capel, J., McCoy, M., Warnasch, J. (1996c). Estimation of Ozone Exposures Experienced by Outdoor Workers in Nine Urban Areas Using a Probabilistic Version of NEM. 1996. Prepared by International Technology Air Quality Services for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. Contract no. 63-D-30094. April, 1996. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr_td.html

Klepeis, N. E., Tsang, A. M., Behar, J. V. (1996). Analysis of the National Human Activity Pattern Survey (NHAPS) Respondents from a Standpoint of Exposure Assessment. Washington, DC: U.S. Environmental Protection Agency, Office of Research and Development. EPA/600/R-96/074.

Knowledge Networks. (2009). Field Report: National Scale Activity Survey (NSAS). Conducted for Research Triangle Institute. Submitted to Carol Mansfield November 13, 2009.

KS DOH. (2006). Environmental Factors, Outdoor Air Quality, and Activity Level. Results from 2005 Kansas Behavioral Risk Factor Surveillance System. Office of Health Promotion, Kansas Department of Health and Environment. Available at: http://www.kdheks.gov/brfss/PDF/cste_report_final.pdf

Langstaff, J. E. (2007). OAQPS Staff Memorandum to Ozone NAAQS Review Docket (OAR-2005-0172). Subject: Analysis of Uncertainty in Ozone Population Exposure Modeling. [January 31, 2007]. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html

Lioy, P.J. (1990). The analysis of total human exposure for exposure assessment: A multi-discipline science for examining human contact with contaminants. *Environ Sci Technol*. 24: 938-945.

Liu L-JS, Box M, Kalman D, Kaufman J, Koenig J, Larson T, Lumley T, Sheppard L, Wallace L. (2003). Exposure assessment of particulate matter for susceptible populations in Seattle. *Environ Health Persp*. 111: 909-918.

Mansfield C, Houtven GV, Johnson FR, Yang J-C. (2009). Environmental Risks and Behavior: Do children spend less time outdoors when ozone pollution is high? ASSA annual meeting, January 5, 2009. Update of Houtven et al. (2003), using the OAB CHAD data set, and related to Mansfield et al. (2006).

Mansfield C, Johnson FR, Van Houtven G. (2006). The missing piece: averting behavior for children's ozone exposures. *Resource Energy Econ*. 28:215-228.

McCurdy, T. (2000). Conceptual basis for multi-route intake dose modeling using an energy expenditure approach. *J Expo Anal Environ Epidemiol*. 10: 1-12.

McCurdy, T., Glen, G., Smith, L., Lakkadi, Y. (2000). The National Exposure Research Laboratory's Consolidated Human Activity Database. *J Expo Anal Environ Epidemiol*. 10: 566-578.

McDermott M, Srivastava R, Croskell S. (2006). Awareness of and compliance with air pollution advisories: A comparison of parents of asthmatics with other parents. *J Asthma*. 43:235-239.

Montoye, H. J., Kemper, H. C. G., Saris, W.H.N., Washburn, R.A. (1996). Measuring Physical Activity and Energy Expenditure. Champaign IL: Human Kinetics.

National Research Council (1991). Human Exposure Assessment for Airborne Pollutants: Advances and Opportunities. Washington, DC: National Academy of Sciences.

Neidell M. (2010). Air quality warnings and outdoor activities: evidence from Southern California using a regression discontinuity approach design. *J Epidemiol Community Health*. 64:921-926.

Richmond H., Palma, T, Langstaff, J., McCurdy, T., Glenn, G., Smith, L. (2002). Further Refinements and Testing of APEX (3.0): EPA's population exposure model for criteria and air toxic inhalation exposures. Joint meeting of the International Society of Exposure Analysis and International Society of Environmental Epidemiology, Vancouver, CAN. August 11-15, 2002.

Robinson, J. P., Wiley, J. A., Piazza, T., Garrett, K., and Cirksena, K. (1989). Activity Patterns of California Residents and their Implications for Potential Exposure to Pollution. California Air Resources Board, Sacramento, CA. CARB-A6-177-33.

SAS (2012). SAS/STAT 9.2 User's Guide, Second Edition. Available at: http://support.sas.com/documentation/cdl/en/statug/63033/PDF/default/statug.pdf

Semenza JC, Wilson DJ, Parra J, Bontempo BD, Hart M, Sailor DJ, George LA. (2008). Public perception and behavior change in relationship to hot weather and air pollution. *Environ Res*. 107:401-411.

Sexton AL. (2011). Responses to Air Quality Alerts: Do Americans Spend Less Time Outdoors? Available at: http://www.apec.umn.edu/prod/groups/cfans/@pub/@cfans/@apec/documents/asset/cfans_asset_365645.pdf

Spier, C. E., Little, D. E., Trim, S. C., Johnson, T. R., Linn, W. S., Hackney, J. D. (1992). Activity patterns in elementary and high school students exposed to oxidant pollution. *J Expo Anal Environ Epidemiol*. 2: 277-293.

Tsang A. M., and Klepeis, N. E. (1996). Descriptive Statistics Tables from a Detailed Analysis of the National Human Activity Pattern Survey (NHAPS) Data. U.S. Environmental Protection Agency. EPA/600/R-96/148.

U.S. Environmental Protection Agency (1986). Air Quality Criteria for Ozone and Other Photochemical Oxidants. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA-600/8-84-020aF-eF. Available from: NTIS, Springfield, VA., PB87-142949.

U.S. Environmental Protection Agency (1996a). Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information - OAQPS Staff Paper. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA/452/R-96-007. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr_sp.html

U.S. Environmental Protection Agency (1996b). Air Quality Criteria for Ozone and Related Photochemical Oxidants. Office of Research and Development, National Center for Environmental Assessment, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA/600/P-93/004aF-cF. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=2831

U.S. Environmental Protection Agency (2002). Consolidated Human Activities Database (CHAD) Users Guide. Database and documentation available at: http://www.epa.gov/chadnet1/

U.S. Environmental Protection Agency (2006). Air Quality Criteria for Ozone and Related Photochemical Oxidants (Final). National Center for Environmental Assessment, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA/600/R-05/004aF-cF. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=149923

U.S. Environmental Protection Agency (2007a). Review of National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information - OAQPS Staff Paper. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA-452/R-07-007. Available at:

U.S. Environmental Protection Agency (2007b). Ozone Population Exposure Analysis for Selected Urban Areas. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html

U.S. Environmental Protection Agency (2008). Risk and Exposure Assessment to Support the Review of the NO_2 Primary National Ambient Air Quality Standard. Report no. EPA-452/R-08-008a. November 2008. Available at: http://www.epa.gov/ttn/naaqs/standards/nox/data/20081121_NO2_REA_final.pdf.

U.S. Environmental Protection Agency (2009). Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standard. Report no. EPA-452/R-09-007. August 2009. Available athttp://www.epa.gov/ttn/naaqs/standards/so2/data/200908SO2REAFinalReport.pdf.

U.S. Environmental Protection Agency (2010). Quantitative Risk and Exposure Assessment for Carbon Monoxide – Amended. EPA Office of Air Quality Planning and Standards. EPA-452/R-10-009. July 2010. Available at: http://www.epa.gov/ttn/naaqs/standards/co/data/CO-REA-Amended-July2010.pdf

U.S. Environmental Protection Agency (2012a). Integrated Science Assessment of Ozone and Related Photochemical Oxidants (Third External Review Draft). U.S. Environmental Protection Agency, National Center for Environmental Assessment, U.S. Environmental Protection Agency, Research Triangle Park, N. EPA/600/R-10/076C, 2012. Available at: http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=242490

U.S. Environmental Protection Agency (2012b). Total Risk Integrated Methodology (TRIM) - Air Pollutants Exposure Model Documentation (TRIM.Expo / APEX, Version 4.4) Volume I: User's Guide. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA-452/B-12-001a. Available at: http://www.epa.gov/ttn/fera/human_apex.html

U.S. Environmental Protection Agency (2012c). Total Risk Integrated Methodology (TRIM) - Air Pollutants Exposure Model Documentation (TRIM.Expo / APEX, Version 4.4) Volume II: Technical Support Document. Office of Air Quality Planning and Standards, U.S.

Environmental Protection Agency, Research Triangle Park, NC. EPA-452/B-12-001b. Available at: http://www.epa.gov/ttn/fera/human_apex.html

University of Michigan. (2012). The Panel Study of Income Dynamics (PSID). Data and documentation available at http://psidonline.isr.umich.edu .

Wells, B., Wesson, K., Jenkins, S. (2012). Analysis of Recent U.S. Ozone Air Quality Data to Support the O3 NAAQS Review and Quadratic Rollback Simulations to Support the First Draft of the Risk and Exposure Assessment. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_rea.html

Wen X-J, Balluz L, Mokdad A. (2009). Association between media alerts of Air Quality Index and change of outdoor activity among adult asthma in six states, BRFSS, 2005. *J Comm Health*. 34:40-46.

Whitfield, R., Biller, W., Jusko, M., Keisler, J. (1996). A Probabilistic Assessment of Health Risks Associated with Short- and Long-Term Exposure to Tropospheric Ozone. Argonne National Laboratory, Argonne, IL.

Wiley, J. A., Robinson, J. P., Piazza, T., Garrett, K., Cirksena, K., Cheng, Y.-T., Martin, G. (1991a). Activity Patterns of California Residents: Final Report. California Air Resources Board, Sacramento, CA. ARB/R93/487. Available from: NTIS, Springfield, VA., PB94-108719.

Wiley, J. A., Robinson, J. P., Cheng, Y.-T., Piazza, T., Stork, L., Pladsen, K. (1991b). Study of Children's Activity Patterns: Final Report. California Air Resources Board, Sacramento, CA. ARB-R-93/489.

Williams R, Suggs J, Rea A, Leovic K, et al. (2003a). The Research Triangle particulate panel study: PM mass concentrations relationships. *Atmos Environ*. 37:5349-5363.

Williams R, Suggs J, Rea A, Sheldon L, et al. (2003b). The Research Triangle particulate panel study: modeling ambient source contributions to personal and residential PM mass concentrations. *Atmos Environ*. 37:5365-5378.

Williams, R., Suggs, J., Creason, J., Rodes, C., Lawless, P., Kwok, R., Zweidinger, R., Sheldon, L. (2000). The 1998 Baltimore particulate matter epidemiology-exposure study: Part 2. Personal exposure associated with an elderly population. *J Expo Anal Environ Epidemiol*. 10(6): 533-543.

Zivin JG, Neidell M. (2009). Days of haze: Environmental information disclosure and intertemporal avoidance behavior. *J Environment Econ Manag*. 58:119

6 CHARACTERIZATION OF HEALTH RISK BASED ON CONTROLLED HUMAN EXPOSURE STUDIES

[This chapter is still under development and will be submitted separately in August]

7 CHARACTERIZATION OF HEALTH RISK BASED ON EPIDEMIOLOGICAL STUDIES

This section provides an overview of the methods used in the urban study area risk assessment. Section 7.1 discusses the basic structure of the risk assessment, identifying the modeling elements and related sources of input data needed for the analysis and presenting an overview of the approach used in calculating health effect incidence using concentration-response functions based on epidemiological studies. Section 7.2 discusses air quality considerations. Section 7.3 discusses the selection of model inputs including: (a) selection and delineation of urban study areas, (b) selection of epidemiological studies and specification of concentration-response functions (C-R functions), (c) defining O_3 concentration ranges for which there is increased confidence in estimating risk (d) specification of baseline health effect incidence and prevalence rates, and (e) estimation of population (demographic) counts. Section 7.4 describes how uncertainty and variability are addressed in the risk assessment. Section 7.5 summarizes the risk estimates that are generated. Section 7.6 provides and integrative discussion of risk estimates with consideration for key sources of variability and uncertainty associated with the overall analysis. Finally, Section 7.7 describes potential refinements to the first draft analysis described here which will be considered for the second draft risk and exposure analysis (REA).

7.1 GENERAL APPROACH

7.1.1 Basic Structure of the Risk Assessment

This risk assessment involves the estimation of the incidence of specific health effect endpoints associated with exposure to ambient O_3 for defined populations located within a set of urban study areas. Because the risk assessment focuses on health effect incidence experienced by defined populations, it represents a form of population-level risk assessment. This analysis does not estimate risks to individuals within the population.

The general approach used in both the prior and current O_3 risk assessments rely on C-R functions based on effect estimates and model specifications obtained from epidemiological studies. Since these studies derive effect estimates and model specifications using ambient air quality data from fixed-site, population-oriented monitors, uncertainty in the application of these functions in an O_3 risk assessment is minimized if, in modeling risk, we also use ambient air quality data at fixed-site, population-oriented monitors to characterize exposure. Therefore, we developed a composite monitor for each urban study area to represent population by averaging across the monitors in that study area to produce a single composite hourly time series of averaged values. The O_3 metrics used in evaluating risk are derived form the composite monitor

hourly time series distribution (see sections 7.2 and Chapter 4 for additional detail on the characterization of ambient O_3 levels).

The general O_3 health risk model, illustrated in Figure 7-1, combines O_3 air quality data, C-R functions, baseline health incidence and prevalence data, and population data (all specific to a given urban study area) to derive estimates of the annual incidence of specified health effects for that urban study area. This first draft exposure and risk assessment (first draft REA) models risk for 12 urban study areas selected to provide coverage for the types of urban O_3 scenarios likely to exist across the U.S. (see section 7.4.1).

The analyses conducted for this review focus on estimating risks associated with recent O_3 air quality and estimating changes in risk associated with air quality simulated to just meet the current O_3 ambient air quality standard (simulation of risk associated with meeting alternative O_3 standard levels will be completed for second Draft of the risk assessment). In simulating just meeting the current O_3 standard level, we assume that reductions in O_3 precursor emissions would only apply to U.S. anthropogenic emissions sources. This was implemented by using modeled estimates of U.S. background O_3, (i.e. O_3 concentrations in the absence of continental emissions of U.S. anthropogenic NOx and VOC), as a lower bound in conducting the rollback of hourly O_3 levels to simulate just meeting the current standard. In other words, we did not allow any single hourly monitored value to be rolled down below U.S. background. We were able to simulate just meeting the current standard in all twelve urban study areas through the reduction of U.S.-anthropogenic O_3 alone. The procedures for modeling U.S. background O_3 and simulating attainment with the current O_3 standards are discussed in Chapter 4 and in the Air Quality Appendices accompanying this REA.

As discussed in Chapters 2 and 3, in modeling risk we employ continuous non-threshold C-R functions relating ozone exposure to health effect incidence. The use of non-threshold functions reflects the conclusion reached in the ISA based on a thorough review of available evidence (see O_3 ISA, section 2.5.4.4, U.S. EPA 2012). However, also consistent with the conclusions of the ISA, we recognize that there is less confidence in specifying the shape of the C-R function at O_3 levels towards the lower end of the distribution of data used in fitting the curve. In particular, we would expect our overall confidence in specifying the magnitude of risk associated with each unit of O_3 exposure to be significantly reduced at levels below the lowest measured level (LML) used in the epidemiological study. Similarly, we would expect our confidence in specifying the magnitude of risk to be increasing with the level of ozone above the LML, and become appreciably greater at ozone concentrations closer to the central mass of measurements used in the underlying epidemiological study. In order to reflect considerations of the differences in relative confidence above and below the LML, we generate two types of risk

estimates for a particular scenario which when considered together inform consideration of uncertainty related to application of the C-R function at low O_3 levels:

- *Risk modeled down to the LML*: This is a higher confidence estimate of risk since it only considers exposure levels within the range of the O_3 data used in the derivation of the C-R function (i.e., exposures down to the LML). However, given that there is no evidence of a threshold for these health effects, and that the statistical models used in the epidemiology studies did not specific a cutoff at the LML, exclusion of exposures below the LML is likely to result in a low-biased risk estimate.

- *Risk modeled down to zero O3:* With this estimate, consistent with the underlying statistical models used in the epidemiology studies, we apply the C-R function across the full range of ambient O3 levels in the study area. While this estimate will reflect the full range of potential exposure and risk (all the way down to zero O3), there is a higher degree of uncertainty about the estimates because they include risks based on extrapolating the C-R function beyond the range of observed O3.

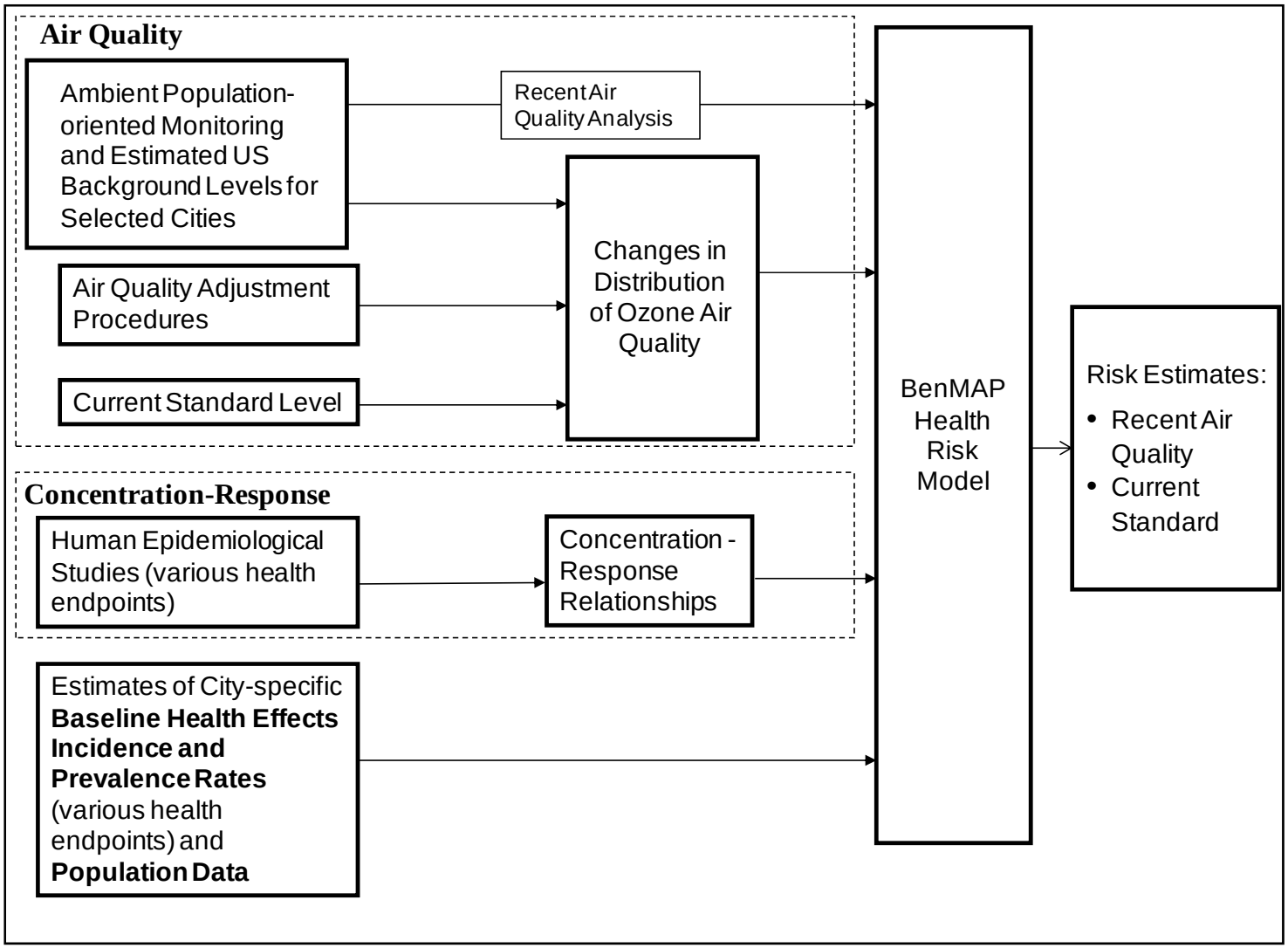
Due to data limitations, we were not able to specify LMLs for the full set of epidemiological studies supporting C-R functions used in the risk assessment. Therefore, we used a surrogate metric as a stand-in for the actual study-based LMLs. Specifically, we used the lowest O_3 values from the composite monitor O_3 distribution used in modeling risk for a particular combination of *urban study area, health endpoint* and *simulation year* to represent the LML for that combination. We recognize that these estimates are not the best surrogates for the true study-specific LMLs, and are evaluating alternative approaches for the second draft REA. While the surrogate LMLs in most cases match the O_3 metric and ozone season used in the underlying epidemiological study, the surrogate LMLs are based on composite monitor distributions specified for the two years ¡ncluded in the risk assessment (2007 and 2009), while O_3 levels used in the epidemiological studies typically reflect several years from an earlier time period (varies across studies). This mismatch in timeframes between the surrogate LMLs and actual study-specific LMLs introduce uncertainty into the analysis. For the second draft REA, we are working to obtain actual LML values used in the source epidemiological studies underlying C-R functions used in the risk assessment (see section 7.7). The specific technical approach used to integrate the LMLs into the generation of risk estimates is discussed in section 7.1.2.1.

In modeling risk for all health endpoints included in the analysis, for recent O_3 conditions and just meeting the current standard, we estimated total risk, both above zero and above the LML. For meeting the current standard, we estimated both total risk as well as the difference in risk, or the risk delta, representing the degree of risk reduction (benefit) associated with just meeting the current standard.

In previous NAAQS-related risk assessments, we have generated two categories of risk estimates, including a set of core (or primary) estimates and an additional set of sensitivity

analyses. The core risk estimates utilize C-R functions based on epidemiological studies for which we have relatively greater overall confidence. While it is generally not possible to assign quantitative levels of confidence to these core risk estimates, they are generally based on inputs having higher overall levels of confidence relative to risk estimates that are generated using other C-R functions. Therefore, emphasis is placed on the core risk estimates in making observations regarding total risk and risk reductions associated with recent conditions and the simulated just meeting the current and alternative standard levels. By contrast, the sensitivity analysis results typically reflect application of C-R functions covering a wider array of design elements which can impact risk (e.g., copollutants models, lag structures, statistical modeling methods etc). The sensitivity analysis results provide insights into the potential impact of these design elements on the core risk estimates, thereby informing our characterization of overall confidence in the core risk estimates.

Figure 7-1. Major components of O_3 health risk assessment.



For first draft of this analysis, we have focused primarily on generating a robust set of core risk estimates and have not developed a comprehensive set of sensitivity analyses due to limitations in the available data from published epidemiology studies. Specifically, for mortality, we obtained Bayes-adjusted city-specific effect estimates which reflected single pollutant models based on 8-hour O_3 metrics for a common lag structure directly from the authors and incorporated those into city-specific risk simulations to generate risk estimates for each of the 12 urban study areas. However, we were not able to obtain similar estimates for other model specifications (e.g. co-pollutant models, alternative lags, etc) typically considered in sensitivity analyses. For the second draft REA, we are investigating methods for obtaining alternative model specifications for use in sensitivity analyses. However, we would note that the set of core risk estimates for short-term exposure morbidity generated for this first draft include coverage for a variety of design elements (including multi-/single-pollutant models and lag structures) and therefore, the array of core risk estimates informs consideration of the impact that these design elements have on risk estimates (see section 7.5).

The risk assessment reflects consideration for five years of recent air quality data from 2006 through 2010, with these five years reflecting two three-year attainment simulation periods that share a common overlapping year (i.e., 2006-2008 and 2008-2010 - see section 7.2). These two attainment periods were selected to provide coverage for a more recent time period with relatively elevated O_3 levels (2006-2008) and recent time period with relatively lower O_3 levels (2008-2010). For the first draft analysis, we modeled risk for the middle year of each three-year attainment simulation period in order to provide estimates of risk for a year with generally higher O_3 levels (2007) and a year with generally lower O_3 levels (2009). In modeling risk, we matched the population data used in the risk assessment to the year of the air quality data. For example, when we used 2007 air quality data, we used 2007 population estimates. For baseline incidence and prevalence, rather than interpolating rates for the two specific years modeled in the risk assessment, we selected the closest year for which we had existing incidence/prevalence data (i.e., for simulation year 2007, we used available data for 2005 and for simulation year 2009, we used data from 2010). The calculation of baseline incidence and prevalence rates is described in detail in section 7.3.4.

The risk assessment procedures described in more detail below are diagramed in Figure 7-2. To estimate the change in incidence of a given health effect resulting from a given change in ambient O_3 concentrations in an assessment location, the following analysis inputs are necessary:

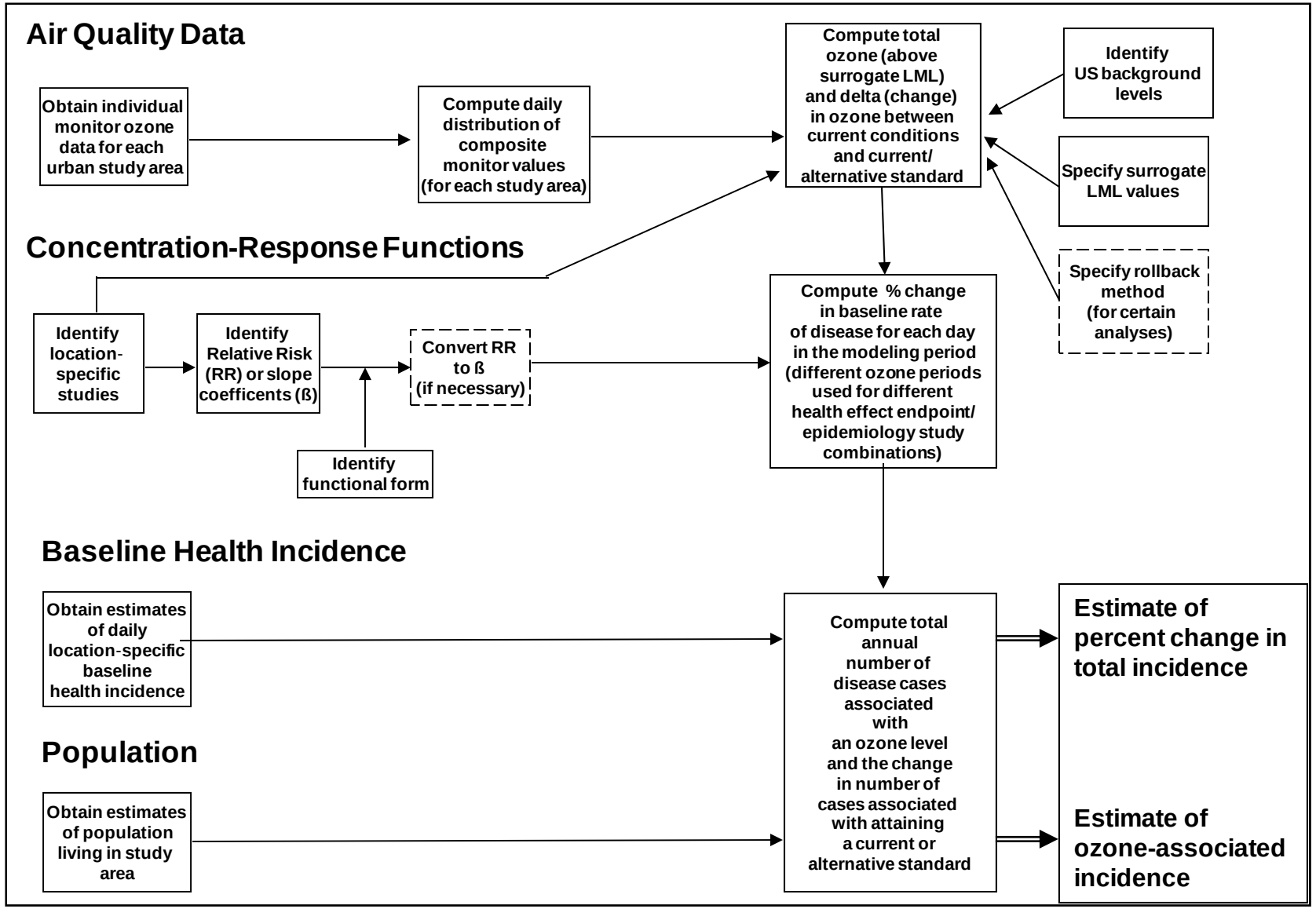
- **Air quality information including:** (1) O_3 air quality data from each of the simulation years included in the analysis (2007 and 2009) from population-oriented monitors in the assessment location, (2) estimates of U.S.-background O_3

concentrations appropriate to this location, and (3) a method for adjusting the air quality data to simulate just meeting the current or alternative suite of O_3 standards. (These air quality inputs are discussed in more detail in section 7.2).

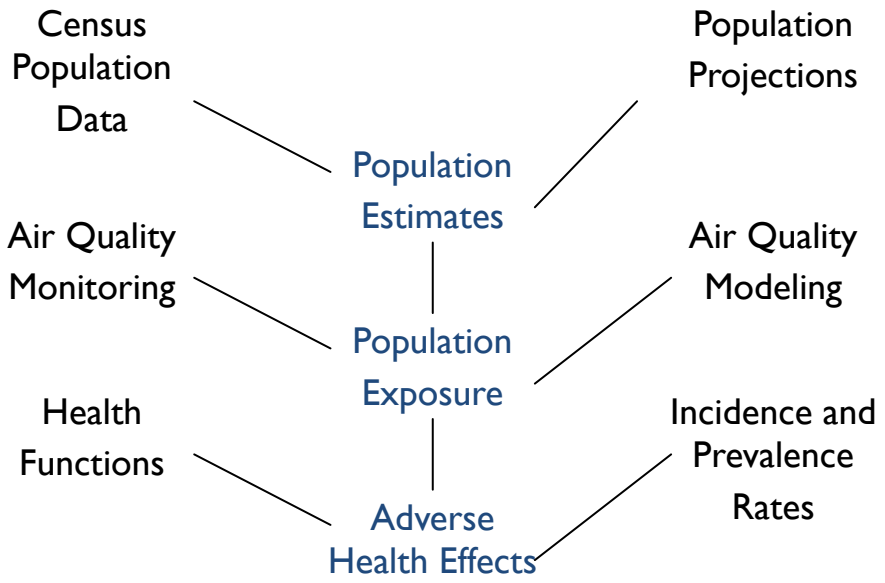
- **C-R function(s)** which provide an estimate of the relationship between the health endpoint of interest and O_3 concentrations (for this analysis, the majority of C-R functions used were applied to urban study areas matching the assessment locations from the epidemiological studies used in deriving the functions, in order to increase overall confidence in the risk estimates generated - see section 7.3.2). For O_3, epidemiological studies providing information necessary to specify C-R functions are readily available for O_3-related health effects associated with short-term exposures (Section 7.1.2 describes the role of C-R functions in estimating health risks associated with O_3). For the first draft analysis, we have not modeled any endpoints associated with long-term O_3 exposure (the potential for modeling these health endpoints is discussed in sections 7.7).

- **Baseline health affects incidence and prevalence rates and population.** The baseline incidence provides an estimate of the incidence rate (number of cases of the health effect per year or day, depending on endpoint, usually per 10,000 or 100,000 general population) in the assessment location corresponding to recent ambient O_3 levels in that location. The baseline prevalence rate describes the prevalence of a given disease state or conditions (e.g., asthma) within the population (number of individuals with the disease state/condition, usually per 10,000 or 100,000 general population). To derive the total baseline incidence or prevalence per year, this rate must be multiplied by the corresponding population number (e.g., if the baseline incidence rate is number of cases per year per 100,000 population, it must be multiplied by the number of 100,000s in the population). (Section 7.3.4 summarizes considerations related to the baseline incidence and prevalence rates and population data inputs to the risk assessment).

Figure 7-2. Flow diagram of risk assessment for short-term exposure studies.



This risk assessment was implemented using the EPA's Benefits Mapping and Analysis Program (BenMAP) (Abt, 2010). This GIS-based computer program draws upon a database of population, baseline incidence/prevalence rates and effect coefficients to automate the calculation of health impacts. For this analysis, the standard set of effect coefficients and health effect incidence data available in BenMAP has been augmented to reflect the latest studies and data available for modeling O_3 risk. EPA has traditionally relied upon the BenMAP program to estimate the health impacts avoided and economic benefits associated with adopting new air quality rules. For this analysis, EPA used the model to estimate O_3-related risk for the suite of health effects endpoints described in section 7.3.2. The following figure summarizes the data inputs (in black text) and outputs (in blue text) for a typical BenMAP analysis.



There are three primary advantages to using BenMAP for this analysis, as compared to the procedure for estimating population risk followed in the last review. First, once we have configured the BenMAP software for this particular O_3 analysis, the program can produce risk estimates for an array of modeling scenarios across a large number of urban areas. Second, the program can more easily accommodate a variety of sensitivity analyses (which we are evaluating for inclusion in second Draft). Third, BenMAP allowed us to complete the national assessment of O_3 mortality described in Chapter 8, which plays in important role in assessing the representativeness of the urban study area analysis.

7.1.2 Calculating O_3-Related Health Effects Incidence

The C-R functions used in the risk assessment are empirically estimated associations between average ambient concentrations of O_3 and the health endpoints of interest (e.g., mortality, hospital admissions, emergency department visits). This section describes the basic method used to estimate changes in the incidence of a health endpoint associated with changes in O_3, using a "generic" C-R function of the most common functional form.

Although some epidemiological studies have estimated linear C-R functions and some have estimated logistic functions, most of the studies used a method referred to as "Poisson regression" to estimate exponential (or log-linear) C-R functions in which the natural logarithm of the health endpoint is a linear function of O_3:

$$y = Be^{\beta x} \quad (1)$$

where x is the ambient O_3 level, y is the incidence of the health endpoint of interest at O_3 level x, β is the coefficient relating ambient O_3 concentration to the health endpoint, and B is the incidence at x=0, i.e., when there is no ambient O_3. The relationship between a specified ambient O_3 level, x_0, for example, and the incidence of a given health endpoint associated with that level (denoted as y_0) is then

$$y_0 = Be^{\beta x_0} \quad (2)$$

Because the log-linear form of a C-R function (equation (1)) is by far the most common form, we use this form to illustrate the "health impact function" used in the O_3 risk assessment.

If we let x_0 denote the baseline (upper) O_3 level, and x_1 denote the lower O_3 level, and y_0 and y_1 denote the corresponding incidences of the health effect, we can derive the following relationship between the change in x, $\Delta x = (x_0 - x_1)$, and the corresponding change in y, Δy, from equation (1).[1]

$$\Delta y = (y_0 - y_1) = y_0[1 - e^{-\beta \Delta x}]. \quad (3)$$

Alternatively, the difference in health effects incidence can be calculated indirectly using relative risk. Relative risk (RR) is a measure commonly used by epidemiologists to characterize the comparative health effects associated with a particular air quality comparison. The risk of

[1] If $\Delta x < 0$ – i.e., if $\Delta x = (x_1 - x_0)$ – then the relationship between Δx and Δy can be shown to be $\Delta y = (y_1 - y_0) = y_0[e^{\beta \Delta x} - 1]$. If $\Delta x < 0$, Δy will similarly be negative. However, the *magnitude* of Δy will be the same whether $\Delta x > 0$ or $\Delta x < 0$ – i.e., the absolute value of Δy does not depend on which equation is used.

mortality at ambient O_3 level x_0 relative to the risk of mortality at ambient O_3 level x_1, for example, may be characterized by the ratio of the two mortality rates: the mortality rate among individuals when the ambient O_3 level is x_0 and the mortality rate among (otherwise identical) individuals when the ambient O_3 level is x_1. This is the RR for mortality associated with the difference between the two ambient O_3 levels, x_0 and x_1. Given a C-R function of the form shown in equation (1) and a particular difference in ambient O_3 levels, Δx, the RR associated with that difference in ambient O_3, denoted as $RR\Delta x$, is equal to $e^{\beta\Delta x}$. The difference in health effects incidence, Δy, corresponding to a given difference in ambient O_3 levels, Δx, can then be calculated based on this $RR\Delta x$ as:

$$\Delta y = (y_0 - y_1) = y_0[1 - (1/RR_{\Delta x})]. \quad (4)$$

Equations (3) and (4) are simply alternative ways of expressing the relationship between a given difference in ambient O_3 levels, $\Delta x > 0$, and the corresponding difference in health effects incidence, Δy. These health impact equations are the key equations that combine air quality information, C-R function information, and baseline health effects incidence information to estimate ambient O_3 health risk.

7.1.2.1 Incorporating LMLs into the estimation of risk

This risk analysis provides two types of risk estimates for each scenario evaluated including: (a) risk modeled down to zero O_3 concentration and (b) risk modeled down to the LML from the epidemiological study providing the C-R function. When considered together these two types of risk estimates inform consideration of uncertainty related to application of the C-R functions at low O_3 levels. As noted in section 7.1.1, due to data limitations, we are using surrogate LML values for the first draft REA in place of actual LMLs from the studies underlying the C-R functions. Specifically, we used the composite monitor dataset used in modeling risk for a particular health endpoint (e.g., the 8hr max set of hourly values used in modeling short-term exposure-related mortality for L.A.) as a surrogate for the set of measured O_3 levels used in deriving the C-R function for that endpoint/city combination. The LML of the composite monitor dataset was used to define an O_3 exposure range of increased confidence in estimating risk for a particular endpoint/location combination.

The LMLs were incorporated in calculation risk as follows. In modeling absolute risk for the recent conditions scenario, we modeled risk for the O_3 increment from the recent conditions down to the LML. Similarly, when estimating the delta (risk reduction) in going from recent conditions to just meeting the current standard, we model risk only for that increment of the change in O_3 that occurred above the LML. As would be expected, application of the LML did

affect estimates of total O_3-attributable risk for both the *recent conditions* and *meeting the current standard* scenarios, with the LML-based estimates being lower. However, estimates of the change in risk between these two air quality scenarios (i.e., in going from recent conditions to meeting the current standard) was not significantly affected by application of the LML since on a daily basis, the recent conditions and current standard values typically occurred above the LML, which meant that the differences between the two levels (on a particular day) nearly always occurred at levels of absolute O_3 well above the LML. The surrogate LMLs used in the first draft REA are presented in section 7.3.3.

7.2 AIR QUALITY CONSIDERATIONS

Air quality data are discussed in detail in Chapter 4 of this report. Here we describe those air quality considerations that are directly relevant to the estimation of health risks in the epidemiology based portion of the risk assessment. As described in section 7.1.1, the risk assessment uses composite monitor values derived for each urban study area as the basis for characterizing population exposure in modeling risk. The use of composite monitors reflects consideration for the way ambient O_3 data are used in the epidemiological studies providing the C-R functions (see section 7.1.1). Because the O_3 risk assessment focuses on short-term exposure related health endpoints, the composite monitor values derived for this analysis include hourly time series for each study area (where the O_3 value for each hour is the average of measurements across the monitors in that study area reporting values for that hour).

For this analysis, reflecting consideration for available evidence in the published literature (see section 7.3.2), we have focused the analysis on short-term peak O_3 metrics including 1hr max, 8hr mean and 8hr max. The more generalized 24 hour average has been deemphasized for this analysis, although it is still used in risk modeling when use of C-R functions based on this metric allow us to cover a specific health effect endpoint/location of particular interest – see section 7.3.2).

For the first draft REA, we estimate risk associated with recent conditions as well as risk associated with simulating just meeting the current standard. While the derivation of composite monitor hourly O_3 distributions (and associated peak exposure metrics) for recent conditions is relatively straightforward, the generation of these estimates for the scenario of just meeting the current standard is more complex. Simulating meeting the current O_3 standard involves application of modeled U.S. background O_3 levels as a floor for hourly O_3 concentrations in the quadratic rollback procedure. The procedure for generating composite monitor values for the recent conditions scenario, along with a summary of the resulting composite monitor values is presented in section 7.2.1. We then describe the procedure used to estimate U.S. background levels for each urban study area, in section 7.2.2. Finally, in section 7.2.3, we briefly describe the

quadratic rollback approach used to simulate just meeting the current standard level and we provide a summary of the resulting composite monitor O_3 metrics. A more complete discussion of these procedures is provided in the air quality chapter (see Chapter 4).

7.2.1 Characterizing Recent Conditions

Recent conditions were characterized using composite monitor-based peak O_3 metrics generated for each of the five years considered in the simulation (additional detail on the generation of composite monitor values is presented in Chapter 4). As noted in section 7.1.1, risk estimates where only generated for 2007 and 2009, which represent the middle years for each of the 3-year attainment periods considered in the analysis. The composite monitors were specified as hourly time series with each hour reflecting the average of available measurements across monitors in a particular study area. The 12 urban study areas included in the analysis are based on the set of counties used in one of the two epidemiology studies providing C-R functions for modeling short-term exposure-related mortality (Zanobetti and Schwartz., 2008b). This county-level specification of the urban study areas resulted in each study area having between one and five counties, with a composite monitor being developed for each study area. The composite monitors for each area were derived using the ambient O_3 monitors falling within each urban area, with the number ranging from three to seventeen monitors per study area. Table 7-1 identifies (a) the counties used in specifying each urban study area, (b) the number of O_3 monitors associated with each and (c) the O_3 season for each study area.

Table 7-1 Information on the 12 Urban Case Study Areas in the Risk Assessment

Study Area	Counties	# of O_3 Monitors	Required O_3 Monitoring Season
Atlanta	Cobb County, GA DeKalb County, GA Fulton County, GA Gwinnett County, GA	5	March - October
Baltimore	Baltimore City, MD Baltimore County, MD	3	April - October
Boston	Middlesex County, MA Norfolk County, MA Suffolk County, MA	5	April - September
Cleveland	Cuyahoga County, OH	4	April - October
Denver	Denver County, CO	3	March - September
Detroit	Wayne County, MI	4	April - September
Houston	Harris County, TX	17	January - December
Los Angeles	Los Angeles County, CA	17	January - December
New York	Bronx County, NY Kings County, NY New York County, NY Queens County, NY Richmond County, NY	8	April - October
Philadelphia	Philadelphia County, PA	4	April - October
Sacramento	Sacramento County, CA	8	January - December
St. Louis	St. Louis City, MO St. Louis County, MO	8	April - October

The O_3 season is an important factor in the risk assessment. In modeling risk for a particular health endpoint, we attempted to match the O_3 season used in deriving the composite monitor value to the O_3 period utilized in the epidemiology study supplying the underlying C-R function. Consequently, there were several versions of the daily peak O_3 metrics generated for the risk assessment (to match the various O_3 periods used in the underlying epidemiology studies). To keep the task of deriving the daily peak O_3 metrics tractable, rather than explicitly matching the O_3 periods used in <u>each</u> of the mortality and morbidity studies providing C-R functions used in the analysis, we elected to match the sets of O_3 periods used in the two epidemiology studies providing C-R functions used in the core analysis for modeling short-term exposure-related mortality (i.e., the Zanobetti and Schwartz 2008b and Bell et al., 2004 studies). The Zanobetti and Schwartz 2008b study used a fixed O_3 period of June-August (combined with an 8hr mean daily O_3 measurement), while the Bell et al., 2004 study reflected the O_3 monitoring period (essentially the O_3 season) specific to each study area - this is the period reflected in Table 7-1 (combined with an 8hr max daily O_3 measurement).[2] For all other health effects endpoints

[2] The ozone monitoring periods used in these two studies are reflected in modeling risk based on C-R functions derived from these studies. Therefore, because the Zanobetti and Schwartz (2008b) study uses a notably shorter monitoring period relative to the Bell et al., (2005) study, risk estimates generated based on C-R functions

modeled for the first draft REA, we then matched up each study to whichever of these two O_3 periods provided the closest match, although we also included a 1hr max daily O_3 metric and a 24hr average metric to comply with the metrics used in several of the studies (see section 7.3.2 for a description of the studies used including their air metrics).

In deriving the composite monitor values, we did not interpolate any missing data and instead took the average of available measurements for each hour. We are evaluating this approach and for the second draft, and may consider application of interpolation methods as a sensitivity analysis to evaluate the potential bias introduced into the analysis by not interpolating missing measurements – see section 7.7. Peak O_3 daily metrics including 1hr max, 8hr mean and 8hr max values were derived from the composite monitor values and used in generating risk estimates. In addition, 24hr average values were also derived as note earlier.

Table 7-2 presents a summary of the composite monitor-based daily metrics for the two short-term exposure-related mortality studies used in the analysis: Zanobetti and Schwartz 2008b (8hr mean metric for June-August) and Bell et al., 2004 (8hr max metric for the city-specific O_3 seasons). These two metrics were selected for illustrating composite monitor values used in the analysis since they provide O_3 air metrics for the majority of health endpoints used in the analysis. These composite monitor summary statistics, which represent recent O_3 conditions for the 12 urban study areas, are presented for 2007 and 2009, reflecting the two simulation years included in the first draft.

obtained from the former study will be notably smaller (other factors equal) than risk estimates generated using C-R functions based on the latter study. This is an important factor which is considered when we review the mortality risk estimates that are generated (see section 7.1.5).

Table 7-2 Composite monitor values (recent conditions) for 2007 and 2009 for air metrics used in modeling short-term exposure-related mortality

Urban study area	8hr (mean) (June-August) (ppb)					8hr max (city-specific O_3 season) (ppb)				
	Min	10th	Mean	90th	Max	Min	10th	Mean	90th	Max
2007 Simulation year										
Atlanta	24	36	**60**	81	104	17	32	**53**	73	106
Baltimore	13	31	**48**	64	81	13	25	**43**	62	81
Boston	19	25	**43**	64	89	12	26	**43**	65	89
Cleveland	6	25	**43**	65	79	12	27	**44**	65	88
Denver	21	36	**50**	60	72	4	27	**44**	57	72
Detroit	19	29	**48**	69	86	13	30	**47**	70	89
Houston	10	17	**33**	56	72	6	18	**35**	56	79
Los Angeles	31	42	**54**	67	80	9	21	**40**	60	87
New York	10	22	**43**	66	82	10	19	**38**	62	85
Philadelphia	12	27	**49**	68	96	13	26	**45**	66	96
Sacramento	30	37	**51**	65	99	13	23	**41**	59	99
St. Louis	22	38	**56**	77	93	8	32	**50**	71	93
2009 Simulation year										
Atlanta	21	29	**49**	65	81	5	24	**42**	60	83
Baltimore	24	32	**48**	62	70	9	25	**42**	58	72
Boston	17	24	**37**	50	70	12	26	**39**	53	76
Cleveland	16	25	**40**	58	66	15	24	**40**	56	73
Denver	22	36	**48**	58	68	16	31	**45**	56	68
Detroit	11	20	**40**	56	84	14	26	**42**	57	86
Houston	15	22	**37**	57	76	7	18	**35**	55	90
Los Angeles	22	33	**52**	68	91	8	22	**42**	63	91
New York	12	23	**40**	57	73	8	19	**36**	55	73
Philadelphia	14	23	**41**	57	77	9	21	**38**	55	78
Sacramento	30	35	**52**	71	82	5	20	**41**	66	90
St. Louis	22	32	**44**	56	68	7	24	**41**	57	68

7.2.2 Estimating U.S. Background

Model based estimates of U.S. Background O_3 levels specific to each urban study area are used as a lower bound for hourly O_3 concentrations in the quadratic rollback procedure used to simulate just meeting the current standard level. This approach reflects the assumption that reductions in O_3 precursor emissions would only apply to U.S. anthropogenic emissions sources. The derivation of the model-based U.S. Background estimates is described in detail in Chapter 4

and consequently, we only provide a brief discussion here, focusing on aspects particularly relevant to the risk assessment.

U.S. background O_3 was modeled at the 70km grid cell level of spatial resolution using a combination of GEOS-Chem (for international transport) with a nested CMAQ model (for more refined transport and atmospheric chemistry within the U.S.). The simulation provides hourly-level estimates of U.S. background O_3 for 2006 (no other years were simulated). Each of the O_3 monitors within a given urban study area is then assigned the U.S. Background hourly profile associated with the 70km grid within which that monitor falls. Because the characterization of U.S. background is model-based and only simulated for 2006, we could not directly match up absolute U.S. background values to absolute measured O_3 levels at a particular monitor on an hour-by-hour basis. Therefore, we developed a more generalized representation of U.S. background levels in the form of U.S. background ratios for each hour/month combination at each monitor. For example we would have a ratio of U.S. background to total O_3 for the 2pm hour in October at a particular monitor. These more generalized U.S. Background ratios can then be multiplied by the actual measured O_3 level at a given monitor for a particular hour (at any time during the 5 year simulation period) to generate the U.S. background estimate for that specific hour/monitor combination. This procedure is repeated for all O_3 measurements associated with a particular monitor within a study area. This distribution of estimated U.S. background levels then serves as the lower bound floor when applying quadratic rollback to that monitor. Additional detail on the derivation of U.S. background values to support quadratic rollback is provided in Chapter 4.

7.2.3 Simulating Air Quality to Just Meet Current and Alternative Standards

Simulating just meeting the current standard uses the same quadratic rollback method as was used in the risk assessment completed for the last O_3 NAAQS review (U.S.EPA, 2007). However for this analysis, we use model-derived estimates of U.S. Background as a lower bound for application of the quadratic rollback.

Quadratic rollback uses a quadratic equation to reduce high concentrations at a greater rate than low concentrations. The intent is to simulate reductions in O_3 resulting from unspecified reductions in precursor emissions, without greatly affecting concentrations near ambient background levels (Duff et al., 1998) (see Chapter 4 for additional detail on application of the quadratic rollback). We are considering the use of a more sophisticated and representative method for the second Draft analysis (the DDM method). Specifically, we are evaluating the Decoupled Direct Method (DDM) approach implemented using the Community Multi-scale Air Quality (CMAQ) model. This approach simulates just meeting the current (as well as alternative) standard levels based on modeling the response of ozone concentrations to reduction in

anthropogenic NOx and VOC emissions (see Chapter 4 for additional detail). In the risk assessment, quadratic rollback is applied to adjust the distribution of O_3 levels at each monitor within a study area such that the O_3 standard is attained at the design monitor within that study area. The rollback procedure is applied to each of the three years of monitoring data associated with each attainment period considered in the analysis (i.e., 2006-2008 and 2008-2010). Once the rollback has been fully implemented and the current O_3 standard is just met for that study area, we then recompute the composite monitor with its daily peak O_3 metrics. This procedure is described in section 7.2.1.

Table 7-3 presents summary statistics for the composite monitor values at each of the urban study areas (for 2006 and 2009) following simulation of just meeting the current standard level.

Table 7-3 Composite monitor values (simulation of meeting current standard) for 2007 and 2009 for air metrics used in modeling short-term exposure-related mortality

Urban study area	8hr (mean) (June-August) (ppb)					8hr max (city-specific O_3 season) (ppb)				
	Min	10th	Mean	90th	Max	Min	10th	Mean	90th	Max
2007 Simulation year										
Atlanta	23	33	**51**	67	79	16	29	**46**	61	81
Baltimore	13	29	**43**	55	68	13	23	**39**	54	68
Boston	18	23	**40**	60	81	12	25	**41**	60	81
Cleveland	6	24	**40**	59	71	11	25	**41**	59	78
Denver	21	34	**45**	54	64	4	26	**41**	52	64
Detroit	19	28	**45**	64	78	12	29	**44**	64	81
Houston	10	16	**30**	50	62	6	17	**32**	50	67
Los Angeles	27	35	**43**	52	57	8	19	**33**	47	61
New York	11	20	**39**	58	70	11	20	**35**	55	71
Philadelphia	13	24	**43**	58	82	14	25	**40**	57	82
Sacramento	27	33	**43**	53	74	13	21	**36**	49	74
St. Louis	22	35	**51**	69	81	8	30	**46**	64	81
2009 Simulation year										
Atlanta	20	28	**46**	62	76	5	23	**40**	57	78
Baltimore	22	30	**43**	55	61	9	23	**38**	52	63
Boston	16	23	**36**	49	69	12	25	**38**	52	75
Cleveland	15	24	**39**	56	64	15	24	**38**	55	70
Denver	22	35	**47**	56	65	16	30	**44**	55	65
Detroit	11	20	**40**	56	84	14	26	**42**	57	86
Houston	14	21	**35**	52	68	6	17	**33**	50	79
Los Angeles	20	29	**43**	53	64	8	20	**36**	50	64
New York	11	22	**37**	52	66	7	18	**34**	51	66
Philadelphia	13	22	**38**	53	70	8	20	**35**	52	71
Sacramento	27	32	**44**	57	65	5	19	**36**	55	69
St. Louis	21	31	**43**	55	66	6	23	**40**	55	67

7.3 SELECTION OF MODEL INPUTS

7.3.1 Selection and Delineation of Urban Study Areas

This analysis focuses on modeling risk for a set of urban study areas, reflecting 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. The following factors were considered in selecting the 12 urban study areas included in this analysis:

- **Air quality data**: An urban area has reasonably comprehensive monitoring data for the period of interest (2006-2010) to support the risk assessment. This criterion was evaluated qualitatively by considering the number of monitors within the attainment area associated with prospective urban areas. Locations with one or two monitors would be excluded since they had relatively limited spatial coverage in characterizing O_3 levels. Ideally, at least three monitors and upwards of five would be present to provide reasonable spatial coverage, but the determination of "reasonable coverage" is complicated since it reflects consideration for population density together with potential gradients in O_3 (and commuting patterns). A rigorous analysis of the degree of effective coverage of monitoring networks for urban populations (and prospective exposure and risk) would not only support a more rigorous selection of urban study areas, but also a better understanding of potential measurement error associated with the epidemiological studies used in risk modeling.

- **Elevated ambient O_3 levels**: Because we are interested in evaluating the potential magnitude of risk reductions associated with just meeting the current and alternative O_3 standard levels, we need to include study areas with elevated ambient O_3 levels such that they are not currently meeting the current O_3 standard, or at least have ambient levels close to the current standard, such that alternative O_3 standard levels to be simulated in the second Draft risk assessment would result in some degree of risk reduction. Consequently, in selecting urban study areas, we considered their status regarding just meeting the current standard, favoring locations that are either not in attainment, or are just barely attaining the standard

- **Location-specific C-R functions**: Given the health endpoints selected for inclusion in the analysis (see section 7.3.2), there are epidemiological studies of sufficient quality available for these urban study areas to provide the C-R functions necessary for modeling risk. This criterion primarily applies to short-term epidemiological studies since the associated health effect endpoints are the primary focus of the first draft REA. Note, that short-term exposure-related epidemiological studies often include city-specific effect estimates, and in some cases are multi-city studies that provide estimates for multiple cities. This is case for mortality where, for this analysis, we have obtained city-specific Bayesian adjusted effect estimates for all selected cities from multi-city studies. (see section 7.3.2).

- **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 O_3 distributions and population characteristics vary geographically across the U.S., we selected urban study areas to provide coverage for regional variability in factors related to O_3 risk including inter-urban gradients in O_3, co-pollutant concentrations, population exposure (differences in residential housing density, air conditioning use and commuting patterns), population vulnerability (baseline incidence rates, SES demographics) and variability in effect estimates. The degree to which the set of urban study areas provided coverage for regional differences across the U.S. in many of these O_3 risk-related factors was evaluated as part of the representativeness analysis presented in Chapter 8.

Application of the above criteria resulted in the selection of 12 urban study areas for inclusion in the risk assessment including:

- Atlanta, GA
- Baltimore, MD
- Boston. MA
- Cleveland, OH
- Denver, CO
- Detroit, MI
- Houston, TX
- Los Angeles, CA
- New York, NY
- Philadelphia, PA
- Sacramento, CA
- St. Louis, MO

The footprint of each urban study area was based on the set of counties included in one of the two epidemiological studies providing city-specific C-R functions for modeling short-term exposure related mortality (Zanobetti and Schwartz., 2008b). This decision reflects the fact that this health endpoint is considered the most important endpoint modeled in this first draft REA and consequently, matching the shape of the study areas to the specific set of counties modeled in one of the two studies supporting modeling of this critical health endpoint, would increase overall confidence in modeling that endpoint. Note, we had considered developing a second set of study area delineations to match the other epidemiology study used in modeling short-term exposure related mortality (Bell et al., 2004), however, this was not feasible given resources and time, and would add an additional difference between the risk estimates for the two studies and reduce the ability to compare risk estimates across the studies. We would point out however, that the two studies have relatively similar county-level delineations of these urban study areas and therefore, the degree of uncertainty introduced into modeling mortality using the Bell et al., 2004 C-R functions (matched to study areas delineations reflecting the Zanobetti and Schwartz, 2008b

study) is expected to be low. The specific set of counties used in defining each of the 12 urban study areas is presented in Table 7-1.

7.3.2 Selection of Epidemiological Studies and Specification of Concentration-Response Functions

Once the set of health effect endpoints to be included in the risk assessment has been specified, the next step was to select the set of epidemiological studies that will provide the effect estimates and model specifications used in the C-R functions. This section describes the approach used in completing these tasks and presents a summary of the epidemiological studies and associated C-R functions specified for use in the risk assessment.

In Chapter 2, section 2.5 we identified the set of health effect categories and associated endpoints to be included in the first draft REA, based on review of the evidence provided in the O_3 ISA (U.S. EPA, 2012). The selection of specific health effect endpoints to model within a given health effect endpoint category is an iterative process involving review of both the strength of evidence (for a given endpoint) as summarized in the O_3 ISA together with consideration for the available epidemiological studies supporting a given endpoint and the ability to specific key inputs needed for risk modeling, including effect estimates and model forms. Ultimately, endpoints are only selected if (a) they are associated with an overarching effect endpoint category selected for inclusion in the risk assessment and (b) they have sufficient epidemiological study support to allow their modeling in the risk assessment. Health effect endpoints selected for inclusion in the first draft REA include, specifically for short-term related O_3 exposure:

- Mortality (likely casual relationship)
  - Non-accidental
  - All-cause
  - Cardiovascular
  - Respiratory
- Respiratory effects (causal relationship)
  - ED (asthma, wheeze, all respiratory symptoms)
  - HA (unscheduled pulmonary illness, asthma)
  - Respiratory symptoms

In addition, as noted in section 2.5, long-term O_3 exposure, represented primarily by studies of peak exposures averaged over longer time periods, was associated with respiratory effects (likely causal relationship), including both respiratory mortality and morbidity. While we have not modeled any long-term exposure related health endpoints for the first draft risk assessment, we are considering the estimation of long-term exposure related respiratory mortality for the second Draft risk assessment (see section 7.7). The remainder of this section deals exclusively with the selection of epidemiological studies and specification of C-R functions for

health effect endpoints associated with short-term O_3 exposure. We provide an evaluation of potential endpoints associated with long term exposures in Section 7.7.

The selection of epidemiological studies to support modeling of the health effect endpoints listed above reflected application of a number of criteria including[3]:

- The study was peer-reviewed, evaluated in the O_3 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, and whether adequate information is provided to characterize statistical uncertainty.

- The study is multicity and ideally, includes Bayes-adjusted city-specific effect estimates (or provides data that supports their derivation) since these effect estimates combine local signals with broader regional or national signals. However, in the case of respiratory morbidity endpoints, in most cases we did not have multicity studies and instead, relied upon city-specific studies to provide coverage for these important endpoints.

- The study design is considered robust and scientifically defensible, particularly in relation to methods for covariate adjustment (including confounders and effects modifiers). For example, if a given study used 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.

- 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.

While the first draft REA applies results from epidemiological studies using composite monitors, we are also evaluating studies which utilized more sophisticated and potentially representative exposure surrogates in characterizing population exposure (e.g., linking exposures in individual counties or U.S. Census tracts to the nearest monitor, rather than using a composite monitor value to represent the entire study area). Depending on the results of our evaluation, we may include these types of epidemiology studies as sensitivity analyses in the second Draft risk assessment (see section 7.7). If we are to use effect estimates from these studies that reflect more sophisticated exposure surrogates, it is important that we also utilize those same exposure surrogates in our risk assessment and not link effect estimates (based on more refined exposure surrogates) with the more generalized composite monitors used in modeling most endpoints in

[3] In addition to the criteria listed here, we also attempted to include studies that provide coverage for populations considered particularly at-risk for a particular health (e.g., children, individuals with preexisting disease). However, a study would have to meet the criteria listed here (in addition to providing coverage for an at-risk population) in order for that study to be used to derive C-R functions.

the risk assessment. As part of the evaluation of these types of studies, we are determining the feasibility of generating these more customized exposure surrogates to match specific epidemiological studies.

Application of the above criteria resulted in the set of epidemiological studies presented in Table 7-4 being identified for use in specifying C-R functions for the first draft analysis (Note, that Table 7-4 also describes elements of the C-R functions specified using each epidemiological study, as discussed below).

Once the set of epidemiology studies was selected, the next step was to specify C-R functions for use in the risk assessment using those studies. Several factors were considered in identifying the effect estimates and model forms used in specifying C-R functions for each endpoint. These factors are described below:

- **O_3 exposure metric:** In the risk assessment supporting the previous O_3 NAAQS review, for short-term exposure, we had included C-R functions based on both 24hr averages as well as a number of peak O_3 measurements. However, based on review of information provided in the O_3 ISA (U.S. EPA, 2012), we now believe there is increased confidence associated with modeling short-term exposure-related health endpoints using peak O_3 metrics (i.e., 1hr max, 8hr max and 8hr means) relative to modeling risk using 24hr averages. Consequently, for the first draft REA, we have focused on the peak O_3 metrics and excluded C-R functions based on 24hr averages (with one exception).[4] The rational for focusing on peak metrics reflects consideration for a number of factors. A study of respiratory ED visits in Atlanta (Darrow et al., 2011) found stronger associations with peak metrics (including 1hr and 8hr max measurements) compared with 24hr averages (see O_3 ISA section 6.2.7.3 and Figure 6-16, U.S. EPA, 2012). Controlled human exposure studies have also demonstrated effects on FEV1, respiratory symptoms, and inflammatory responses associated with exposures up to 8hr (see ISA section 2.5.3). With regard to mortality, the picture is not as clear, primarily due to limitations in the number of epidemiological studies comparing the association of peak O_3 metrics and the 24hr average metric with mortality. However, when we consider the other information described here, we conclude that it is generally appropriate to place greater emphasis on C-R functions (for both mortality and morbidity) that utilize peak exposure metrics.[5]

[4] As noted earlier, in order to provide estimates of respiratory-related HA for LA, we did include a C-R function based on Linn et al., 2000, which utilizes a 24hr average exposure metric.

[5] In addition, peak ozone metrics, by focusing on daily ozone levels, avoid the issue where simulation of meeting the current standard results in nighttime ozone levels actually increasing in some situations (this is a concern for the 24hr ozone metrics, where these increases in nighttime ozone can dampen predicted reductions in daytime ozone).

Table 7-4 Overview of Epidemiological Studies Used in Specifying C-R Functions

Epidemiological study (*stratified by short-term exposure-related health endpoints*)	Health endpoints	Location (urban study area(s) covered)	Exposure metric (and modeling period)	Additional study design details	Notes regarding application in first Draft analysis
Mortality					
Bell et al., 2004	Non-accidental, respiratory, cardiovascular	95 large urban communities (provides coverage for all 12 urban study areas)	24hr avg, 8hr max, 1hr max. April through October and all year	Adjusting for time-varying confounders (PM, weather, seasonality). Lag structure included 0, 1, 2 and day 3 lag as well as 0-6 day distributed lag. Age range: all ages.	Obtained Bayes-adjusted city-specific effect estimates for non-accidental mortality from Dr. Bell (personal communication, Dr. Michelle Bell, December 22, 2011). Effect estimates based on constrained distributed lag (0-6 days) for the 8hr max peak metric evaluated for the fullest of monitored data associated with each urban area (for most urban areas, this represents measurements taken during city-specific ozone season). For this reason, we constrained risk modeling using these effect estimates to the ozone season specific to each urban study area (see Table 7-1).
Zanobetti and Schwartz (2008b)	Non-accidental, respiratory, cardiovascular	48 U.S. cities (provides coverage for the 12 urban study areas)	8hr max. June-August	Effect controlled for season, day of week, and temperature. Lag structure included 0-3d, 0-20 and 4-20 day). Age range: all ages	Obtained Bayes-adjusted city-specific effect estimates for non-accidental, respiratory and cardiovascular from Dr. Zanobetti (personal communication, Dr. Antonella Zanobetti, January 5, 2012). These effect estimates reflect a 0-3 day distributed lag and are based on 8hr mean ozone levels measured between June and August. Consequently, we constrained modeling of risk with these effect estimates to June-August for each urban study area.
Morbidity - HA for respiratory effect)					
Medina-Ramon et al., 2006.	HA: COPD, pneumonia	36 cities (provides coverage for all 12 urban study areas)	8hr mean. warm (May-August), cool (October-April), all year	Distributed lag (0-1 day). Age range: ≥ 65yrs.	Generated risk estimates based on warm season (used existing June-August composite monitor 8hr mean values).
Linn et al., 2000	HA: unscheduled for pulmonary illness	LA only	24hr mean, LA ozone season (all year)	Lag 0. Age range: all ages	Included effect estimate based on 24hr avg metric since this provided additional coverage for HA in L.A.
Lin et al., 2008	HA: respiratory disease	NY State (used to cover NYC)	1hr max (for 10am-6pm interval), warm season (April-October)	Lag 0, 1, 2, 3. Age range: <18yrs	Used 1hr max metric applied to the city-specific ozone season for NYC (April-October).
Katsouyanni et al 2009	HA: cardiovascular disease, chronic	14 cities (provides coverage for Detroit only)	1hr max. Summer only and all year	Lag 0-1day. Age range: ≥ 65yrs.	C-R function applied only for all respiratory endpoint. Used June-August-based composite monitor.

Epidemiological study (*stratified by short-term exposure-related health endpoints*)	Health endpoints	Location (urban study area(s) covered)	Exposure metric (and modeling period)	Additional study design details	Notes regarding application in first Draft analysis
	obstructive pulmonary disease, pneumonia, all respiratory				
Silverman et al., 2010	HA: asthma (ICU and non-ICU)	NYC	8hr max. Warm season (April-August)	Includes control for $PM_{2.5}$. Lag 0-1 day. Age range: children 6-18yrs	Applied C-R function (for ozone and ozone with control for $PM_{2.5}$) to the city-specific ozone season for NYC (slightly longer than the modeling period used in the study).
Morbidity – ED and ER visits (respiratory)					
Ito et al., 2007	ED: asthma	NYC	8hr max. Warm season (April-September)	Includes models controlling for SO_2, NO_2, CO and $PM_{2.5}$. Lag: 0, 1, and distributed lag (0-1 day). Age range: all ages	Applied C-R functions (for ozone alone and ozone with control for listed pollutants) to the city-specific ozone season for NYC (slightly longer than the modeling period used in the study).
Tolbert et al., 2007	ED: all respiratory	Atlanta	8hr max. Summer (March-October)	Includes models controlling for NO_2, CO, PM_{10} and NO_2/ NO_2. Age range: all ages	Applied C-R functions (for ozone alone and ozone with control for listed pollutants) to the city-specific ozone season for Atlanta.
Strickland et al., 2010	ER: respiratory	Atlanta	8hr max (based on population weighted average across monitors). Warm season (May to October) and cool (November to April)	Lag: average of 0-2 day, distributed lag 0-7 day. Age range: 5-17yrs	Included effect estimates based on both lag structures and used composite monitor values for city-specific ozone season.
Darrow etl al., 2011	ED: all respiratory	Atlanta	8hr max, 1hr max, 24hr avg for summer (March-October).	Lag: 1day. Age range: all ages	Used city-specific ozone season-based composite monitor values.
Morbidity – respiratory symptoms					
Gent et al., 2003	Respiratory symptoms: wheeze, persistent	Springfield MA (study used to cover Boston)	1hr max, 8hr max	Lag: 0 and 1 day. Age range: asthmatic children <12 yrs.	Included effect estimates for different symptoms based on both 8hr max and 1hr max metrics (for city-specific ozone season composite monitor values for Boston). The study area (which focuses on Springfield and the northern portion of Connecticut) does not

Epidemiological study (*stratified by short-term exposure-related health endpoints*)	Health endpoints	Location (urban study area(s) covered)	Exposure metric (and modeling period)	Additional study design details	Notes regarding application in first Draft analysis
	cough, chest tightness, shortness of breath				encompass Boston. However, we are willing to accept uncertainty associated with using effect estimates from this study to provide coverage for Boston given the goal of providing coverage for this morbidity endpoint. However, there is increased uncertainty associated with modeling for this endpoint..

- **Single- and multi-pollutant models (*pertains to both short-term and long-term exposure studies*)**: Epidemiological studies often consider health effects associated with ambient O_3 independently as well as together with co-pollutants (e.g., O_3, 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 O_3 in single pollutant models, risks attributed to O_3 may be overestimated or underestimated 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 O_3, inclusion of those pollutants in the health impact model can produce unstable and statistically insignificant effect estimates for both O_3 and the co-pollutants. This situation would argue for inclusion of a model based exclusively on O_3. Given that single and multi-pollutant models each have potential advantages and disadvantages, to the extent possible, given available information we have included both types of C-R functions in the risk assessment.

- **Single-city versus multi-city 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. By contrast, single-city studies, while often having lower statistical power and varying study designs which can make comparison across cities challenging, reflect location-specific factors such as 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. There is a third type of study design that generates Bayes-adjusted city-specific effect estimates, thereby combining the advantages of both city-specific and multi-city studies. Bayes-adjusted city-specific estimates begin with a city-specific effect estimate and shrink that towards a multi-city mean effect estimate based on consideration for the degree of variance in both estimates. For the first draft REA, we have elected to place greater confidence on these types of Bayesian-adjusted effect estimates when they are available. Otherwise, given the advantages for both city-specific and multi-city effect estimates, we have used both types when available. In those instances where a multi-city study only provides aggregated effect estimates, but does differentiate those estimates regionally, we would use those regional-specific estimates rather than a single national-level estimate by matching selected urban study areas to these regions. For the epidemiological studies we identified for this first draft analysis, none included these types of regional effect estimates – see Table 7-4.

- **Multiple lag models**: Based on our review of evidenced provided in the ISA, we believe there is increased confidence in modeling both mortality and respiratory morbidity risk based on exposures occurring up to a few days prior to the health effect, with less support

for associations over longer exposure periods or effects lagged more than a few days from the exposure (see O_3 ISA section 2.5.4.3, U.S. EPA, 2012). Consequently, we have favored C-R functions reflecting shorter lag periods (e.g., 0, 1 or 1-2 days). With regard to the specific lag structure (e.g, single day versus distributed lags), the O_3 ISA notes that epidemiological studies involving respiratory morbidity have suggested that both single day and multi-day average exposures are associated with adverse health effects (see O_3 ISA section 2.5.4.3). Therefore, when available both types of lag structures where considered in specifying C-R functions.

- **Seasonally-differentiated effects estimates**: The previous O_3 AQCD (published in 2006) concluded that aggregate population time-series studies demonstrates a positive and robust association between ambient O_3 concentrations and respiratory-related hospitalizations and asthma ED visits during the warm season (see O_3 ISA section 2.5.3m U.S. EPA, 2012). The current O_3 ISA notes that recent studies of short-term exposure-related respiratory mortality in the U.S. suggest that the effect is strengthened in the summer season (O_3 ISA section 6.6.2.5, U.S. EPA, 2012). In addition, we note that many of the key epidemiological studies exploring both short-term exposure related mortality and morbidity discussed in the current O_3 ISA have larger (and more statistically significant) effect estimates when evaluated for the summer (O_3) season, relative to the full year (see O_3 ISA Figures 6-18 and 6-26, U.S. EPA, 2012). Given that we anticipate O_3 levels to be elevated during the O_3 season resulting in increased exposure and risk, we favored C-R functions based on O_3 measurements taken during the O_3 (or warm/summer) season and placed less emphasis on C-R functions reflecting O_3 measured over the entire year (unless, as with L.A. the O_3 period is the entire year).

- **Shape of the functional form of the risk model (including threshold):** The current O_3 ISA concludes that there is little support in the literature for a population threshold for short-term exposure-related effects, although in the case of mortality, the O_3 ISA notes that the nature of the mortality effect as well as study design may mean that these studies are not well suited to identify a threshold should it exist (see O_3 ISA, section 2.5.4.4, U.S. EPA, 2012). Given the above observation from the ISA regarding the potential for thresholds, we did not include C-R functions for any of the short-term exposure-related health endpoints modeled that incorporated a threshold.

Application of the above criteria resulted in an array of C-R functions specified for the risk assessment (see Table 7-4). In presenting the C-R functions in Table 7-4, we have focused on describing key attributes of each C-R function (and associated source epidemiological study) relevant to a review of their use in the risk assessment. More detailed technical information including effect estimates and model specification is provided in Appendix 7-A (Table 7A-1). Specific summary information provided in Table 7-4 includes:

- *Health endpoints:* identifies the specific endpoints evaluated in the study. Generally we included all of these in our risk modeling, however, when a subset was modeled, we reference that in the "Notes" column (last column in the table).

- *Location*: identifies the specific urban areas included in the study and maps those to the set of 12 urban study areas included in the risk assessment.

- *Exposure metric*: describes the exposure metric used in the study, including the specific modeling period (e.g., O_3 season, warm season, full year). As noted earlier, for the first draft REA, we developed two categories of composite monitor values to match the modeling periods used in the two short-term exposure-related mortality studies providing C-R functions for the analysis. For the remaining morbidity endpoints, we mapped specific C-R functions to whichever of these two composite monitor categories most closely matched the modeling period used in the underlying epidemiological study. This mapping (for morbidity endpoint C-R functions) is described in the "Notes" column (the seasons reflecting in modeling for each C-R function are also presented in Appendix 7-A, Table 7A-1).

- *Additional study design details*: this column provides additional information primarily covering the lag structure and age ranges used in the study.

- *Notes regarding application in first draft analysis*: as the name implies, this column provides notes particular to the application of a particular epidemiological study and associated C-R functions in the risk assessment.

7.3.3 Defining O_3 concentration ranges (down to the LML) for which there is increased confidence in estimating risk

As discussed in section 7.1.1 and 7.3.2, for this first draft REA, we did not incorporate thresholds in modeling risk, reflecting consideration of the evidence as summarized in the O_3 ISA (see section 2.5.4.4, U.S. EPA, 2012). However, we did identify O_3 concentration ranges for which there is increased confidence in estimating risk. Specifically, we note that modeling risk within the range of O_3 levels used in the derivation the C-R function has increased confidence relative to modeling risk for O_3 levels below that range. Therefore, we can use the LML associated with the derivation of a particular C-R function to help define an O_3 concentration range with increased confidence in estimating risk. Overall confidence is further increased as we model risk closer to the central mass of O_3 levels used in the derivation of the C-R function. Ideally we would have access to the O_3 monitor-based datasets used in each of the epidemiological studies providing C-R functions used in this analysis so that we could define these ranges of increased confidence accordingly. In the case of city-specific effect estimates ideally we would obtain the underlying O_3 measurement data stratified by urban study area. Note, also that, when we reference "measurement data" we are actually referring to the specific exposure surrogate used in deriving the C-R function and not simply the array of hourly values for each monitor. However, data limitations prevented us from identifying the LML each study and therefore, as noted in section 7.1.1, we used the distributions of composite monitor values

calculated for each of the two simulation years included in the analysis (2007 and 2009) to estimate surrogates for the LML.

Given the different dimensions associated with risk estimates generated for this analysis (e.g., 12 urban study areas, two simulation years, several different daily peak O_3 level metrics associated with different C-R functions) an array of LMLs had to be extracted from the composite monitor values used in the risk assessment. The set of LML values used to define O_3 concentration ranges for which there is increased confidence in estimating risk is presented below in Table 7-5. The set of LMLs is also provided as part of the full set of model inputs presented in Appendix 7A, Table 7A-1.

LML values presented in Table 7-5 were linked to a given C-R function based on the air quality metric used by the C-R function. For example, with short-term exposure related mortality estimated for Baltimore in 2007 using the Bell et al., (2004) study and associated C-R function, we used the LML value for the 8hr max metric (city-specific O_3 season), reflecting the metric used for that C-R function (see Table 7-4 and Appendix 7A, Table 7A-1). Consequently, we would identify 13 ppb from Table 7-5 (and Table 7A-1) as the LML for modeling that endpoint. As noted earlier in section 7.1.2, we then use the LML as a lower bound on the C-R function (i.e., risk would not be modeled below 13ppb), in generating higher confidence risk estimates.[6]

[6] The values presented in Table 7-5 allowed us to define exposure ranges with increased confidence for most of the endpoints included in this analysis (see Table 7-4 and Appendix 7A, Table 7A-1 for details on which air metrics were used in modeling specific health endpoints and consequently, which of the values from Table 7-5 would be used in specifying regions of increased confidence). However, several short-term exposure-related morbidity studies used ozone metrics different form the 8hr mean (June-August) and 8hr max (city-specific ozone season) reflected in the statistics presented in Table 7-5 and therefore, we had to identify LML values from different composite monitors in order to specify regions of increased confidence for these endpoints (the full set of LMLs for all C-R functions is presented in Appendix 7A, Table 7A-1).

Table 7-5 Composite Monitor O_3 LML Used in Defining Ranges of Increased Confidence in Modeling Risk

Urban Study Area	8r max (city-specific O_3 season) ppb	8hr mean (reflects June-August levels) ppb
Metrics Based on 2007 Composite Monitors		
Atlanta	17	24
Baltimore	13	13
Boston	12	19
Cleveland	12	6
Denver	4	21
Detroit	13	19
Houston	6	10
Los Angeles	9	31
New York	10	10
Philadelphia	13	12
Sacramento	13	30
St. Louis	8	22
Metrics Based on 2009 Composite Monitors		
Atlanta	5	21
Baltimore	9	24
Boston	12	17
Cleveland	15	16
Denver	16	22
Detroit	14	11
Houston	7	15
Los Angeles	8	22
New York	8	12
Philadelphia	9	14
Sacramento	5	30
St. Louis	7	22

7.3.4 Baseline health effect incidence and prevalence data

As noted earlier (section 7.1.2), the most common epidemiological-based health risk model expresses the reduction in health risk (Δy) associated with a given reduction in O_3 concentrations (Δx) as a percentage of the baseline incidence (y). To accurately assess the impact of O_3 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. In some instances, health endpoints are modeled for a population with an existing health condition, necessitating the use of a prevalence rate. Where at all possible, we use county-specific incidences or incidence rates (in combination with county-specific populations). In some instances, when county-level incidence rates were not available, BenMAP can calculate and employ more generalized regional rates (see BenMAP Guidance Manual for additional detail, Abt Associates, Inc. 2010). For prevalence rates (which were only necessary for modeling respiratory symptoms among asthmatic children using Gent et al., (2008) - see Table 7-4), we utilized a national-level prevalence rate appropriate for the age group being modeled. A summary of available baseline incidence data for specific categories of effects (and prevalence rates for asthma) is presented below:

- *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.[7] The most recent year for which data are available online is 2005 and this was the source of incidence data for the risk assessment.[8]

- *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). 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.

- *Asthma prevalence rates:* state-level prevalence rates that are age group stratified are available from the Centers for Disease Control and Prevention (CDC) Behavioral Risk Factor Surveillance System (BRFSS) (U.S. CDC, 2010).

Incidence and prevalence rates used in the first draft REA are presented as part of the full set of model inputs documented in Appendix 7A, Table 7A-1. The incidence rates and prevalence rates provided in Table 7A-1 are weighted average values for the age group associated with each of the C-R functions. These weighted averages are calculated within BenMAP using more refined age-differentiated incidence and prevalence rates originally obtained from the data sources listed in the bullets above.

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

[8] 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.

7.3.5 Population (demographic) data

To calculate baseline incidence rate, in addition to the health baseline incidence data we also need the corresponding population. We obtained population data from the U.S. Census bureau (http://www.census.gov/popest/counties/asrh/). These data, released on May 14, 2009, are the population estimates of the resident populations by selected age groups and sex for counties in each U.S. state from 2000 to 2008. Total population counts used in modeling each of the health endpoints evaluated in the analysis (differentiated by urban study area and simulation year) are provided as part model inputs presented in Appendix 7A, Table 7A-1.

7.4 ADDRESSING VARIABILITY AND UNCERTAINTY

An important component of a population risk assessment is the characterization of both uncertainty and variability. *Variability* refers to the heterogeneity of a variable of interest within a population or across different populations. For example, populations in different regions of the country may have different behavior and activity patterns (e.g., air conditioning use, time spent indoors) that affect their exposure to ambient O_3 and thus the population health response. The composition of populations in different regions of the country may vary in ways that can affect the population response to exposure to O_3 – e.g., two populations exposed to the same levels of O_3 might respond differently if one population is older than the other. Variability is inherent and cannot be reduced through further research. Refinements in the design of a population risk assessment are often focused on more completely characterizing variability in key factors affecting population risk – e.g., factors affecting population exposure or response – in order to produce risk estimates whose distribution adequately characterizes the distribution in the underlying population(s).

Uncertainty refers to the lack of knowledge regarding the actual values of inputs to an analysis. Models are typically used in analyses, and there is uncertainty about the true values of the parameters of the model (parameter uncertainty) – e.g., the value of the coefficient for O_3 in a C-R function. There is also uncertainty about the extent to which the model is an accurate representation of the underlying physical systems or relationships being modeled (model uncertainty) – e.g., the shapes of C-R functions. In addition, there may be some uncertainty surrounding other inputs to an analysis due to possible measurement error—e.g., the values of daily O_3 concentrations in a risk assessment location, or the value of the baseline incidence rate for a health effect in a population.[9] In any risk assessment, uncertainty is, ideally, reduced to the maximum extent possible through improved measurement of key variables and ongoing model

[9] It is also important to point out that failure to characterize variability in an input used in modeling can also introduce uncertainty into the analysis. This reflects the important link between uncertainty and variability with the effort to accurately characterize variability in key model inputs actually reflecting an effort to reduce uncertainty.

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 be both qualitative and, if a sufficient knowledgebase is available, quantitative.

The selection of urban study areas for the O_3 risk assessment was designed to cover the range of O_3-related risk experienced by the U.S. population and, in general, to adequately reflect the inherent variability in those factors affecting the public health impact of O_3 exposure. Sources of variability reflected in the risk assessment design are discussed in section 7.4.1, along with a discussion of those sources of variability which are not fully reflected in the risk assessment and consequently introduce uncertainty into the analysis.

The characterization of uncertainty associated with risk assessment is often addressed in the regulatory context using a tiered approach in which progressively more sophisticated methods are used to evaluate and characterize sources of uncertainty depending on the overall complexity of the risk assessment (WHO, 2008). 3Guidance documents developed by EPA for assessing air toxics-related risk and Superfund Site risks (U.S.EPA, 2004 and 2001, respectively) as well as recent guidance from the World Health Organization (WHO, 2008) specify multi-tiered approaches for addressing uncertainty.

The WHO guidance, in particular, presents a four-tiered approach for characterizing uncertainty (see Chapter 3, section 3.2.6 for additional detail on the four tiers included in the WHO's guidance document). With this four-tiered approach, the WHO framework provides a means for systematically linking the characterization of uncertainty to the sophistication of the underlying risk assessment. Ultimately, the decision as to which tier of uncertainty characterization to include in a risk assessment will depend both on the overall sophistication of the risk assessment and the availability of information for characterizing the various sources of uncertainty. EPA staff has used the WHO guidance as a framework for developing the approach used for characterizing uncertainty in this risk assessment.

The overall analysis in the O_3 NAAQS risk assessment is relatively complex, thereby warranting consideration of a full probabilistic (WHO Tier 3) uncertainty analysis. However, limitations in available information prevent this level of analysis from being completed at this time. In particular, the incorporation of uncertainty related to key elements of C-R functions (e.g., competing lag structures, alternative functional forms, etc.) into a full probabilistic WHO Tier 3 analysis would require that probabilities be assigned to each competing specification of a given model element (with each probability reflecting a subjective assessment of the probability that the given specification is the "correct" description of reality). However, for many model elements there is insufficient information on which to base these probabilities. One approach that has been taken in such cases is expert elicitation; however, this approach is resource- and time-

intensive and consequently, it was not feasible to use this technique in the current O_3 NAAQS review to support a WHO Tier 3 analysis.[10]

For most elements of this risk assessment, rather than conducting a full probabilistic uncertainty analysis, we have included qualitative discussions of the potential impact of uncertainty on risk results (WHO Tier1). As discussed in section 7.1.1, we had originally planned to complete a comprehensive sensitivity analysis exploring the potential impact of various design elements on the core risk estimates being generated (WHO Tier 2). However, the effort required to complete a comprehensive set of core risk estimates for the mortality and morbidity endpoints included in the analysis prevented us from completing a comprehensive sensitivity analysis for the first draft REA. We do note however, that the set of core risk estimates generated for the analysis does provide, for some of the health endpoints (i.e., respiratory morbidity) an array of estimates that covers a number of modeling elements (e.g., copollutants models, lag structure, air quality metric). Insights into the potential impact of these design elements on the core risk estimates are discussed as those risk estimates are summarized in sections 7.1.4.2. Sensitivity analyses being considered for the second draft REA are described in section 7.7.1.

In addition to the qualitative and quantitative treatment of uncertainty and variability which are described here, we have also completed an analysis to evaluate the representativeness of the selected urban study areas against national distributions for key O_3 risk-related attributes to determine whether they are nationally representative or more focused on a particular portion of the distribution for a given attribute (see Chapter 8, section 8.2.1). In addition, we have completed a second analysis addressing the representativeness issue, which identified where the 12 urban study areas included in this risk assessment fall along a distribution of national-level long-term exposure-related mortality risk (see Chapter 8, section 8.2.2). This analysis allowed us to assess the degree of which the 12 urban study areas capture locations within the U.S. likely to experience elevated levels of risk related to O_3 exposure.

The remainder of this section is organized as follows. Key sources of variability which are reflected in the design of the risk assessment, along with sources excluded from the design, are discussed in section 7.1.4.1. A qualitative discussion of key sources of uncertainty associated with the risk assessment (including the potential direction, magnitude and degree of confidence associated with our understanding of the source of uncertainty – the knowledge base) is presented in section 7.1.4.2.

[10] Note, that while a full probabilistic uncertainty analysis was not completed for this risk assessment, we were able to use confidence intervals associated with effects estimates (obtained from epidemiological studies) to incorporate statistical uncertainty associated with sample size considerations in the presentation of risk estimates.

7.4.1 Treatment of Key Sources of Variability

The risk assessment was designed to cover the key sources of variability related to population exposure and exposure response, to the extent supported by available data. Here, the term *key sources of variability* refers to those sources that the EPA staff believes have the potential to play an important role in impacting population incidence estimates generated for this risk assessment. Specifically, EPA staff has concluded that these sources of variability, if fully addressed and integrated into the analysis, could result in adjustments to the core risk estimates which might be relevant from the standpoint of interpreting the risk estimates in the context of the O_3 NAAQS review. The identification of sources of variability as "key" reflects consideration for sensitivity analyses conducted for previous O_3 NAAQS risk assessments, which have provided insights into which sources of variability (reflected in different elements of those earlier sensitivity analyses) can influence risk estimates, as well as information presented in the O_3 ISA.

As with all risk assessments, there are sources of variability which have not been fully reflected in the design of the risk assessment and consequently introduce a degree of uncertainty into the risk estimates. While different sources of variability were captured in the risk assessment, it was generally not possible to separate out the impact of each factor on population risk estimates, since many of the sources of variability are reflected collectively in a specific aspect of the risk model. For example, inclusion of urban study areas from different regions of the country likely provides some degree of coverage for a variety of factors associated with O_3 risk (e.g., air conditioner use, differences in population commuting and exercise patterns, weather). However, the model is not sufficiently precise or disaggregated to allow the individual impacts of any one of these sources of variability on the risk estimates to be characterized.

Key sources of potential variability that are likely to affect population risks are discussed below, including the degree to which they are captured in the design of the risk assessment:

- **Heterogeneity in the effect of O_3 on health across different urban areas:** A number of studies cited in the ISA have found evidence for regional heterogeneity in the short-term exposure-related mortality effect (Smith et al., 2009 and Bell and Dominici, 2008, Bell et al., 2004, Zanobetti an Schwartz 2008b – see O_3 ISA section 6.6.2.2, U.S. EPA, 2012). These studies have demonstrated that the cross-city differences in effect estimates can be quite substantial (see ISA Figures 6-31 and 6-32). For the short-term exposure-related mortality endpoint, we have used Bayes-adjusted city-specific effect estimates which are intended to capture cross-city differences in effect estimates for the mortality endpoint (while still utilizing information provided by a more stable national-level estimate). However, Smith et al., 2009 had recommended that Bayes-adjusted city-specific effect estimates such as those cited in Bell et al., 2004, utilize regionally-differentiate effect estimates for updating the city specific effect estimates, rather than a national-level effect estimate, in order to more fully capture spatial heterogeneity in the O_3 effect. This

recommended refinement by Smith et al., 2009 to the derivation of effect estimates using the Bayes-adjustment technique has not been implemented, but may be considered for the second draft analysis (see section 7.7.1). For short-term morbidity endpoints, typically we have used city-specific effect estimates, however, for most endpoints, we only have estimates for a subset of the urban study areas (typically NYC, Atlanta and/or LA). Therefore, while our risk estimates do reflect the application of city-specific effect estimates, because we do not have estimates for all 12 urban study areas, we do not provide comprehensive coverage for heterogeneity in modeling the respiratory morbidity endpoint category.

- **Intra-urban variability in ambient O_3 levels**: The picture with regard to within city variability in ambient O_3 levels and the potential impact on epidemiologic-based effect estimates is somewhat more complicated. The ISA notes that spatial variability in O_3 levels is dependent on spatial scale with O_3 levels being more homogeneous over a few kilometers due to the secondary formation nature of O_3, while levels can vary substantially over tens of kilometers. Community exposure may not be well represented when monitors cover large areas with several subcommunities having different sources and topographies as exemplified by Los Angeles which displays significantly greater variation in inter-monitor correlations than does for example, Atlanta or Boston (see O_3 ISA section 4.6.2.1 U.S. EPA 2012). Despite the potential for substantial variability across monitors (particularly in larger urban areas with greater variation in sources and topography), the ISA notes that studies have tended to demonstrate that monitor selection has only a limited effect on the association of short-term O_3 exposure with health effects. The likely explanation for this is that, while absolute values for a fixed point in time can vary across monitors in an urban area, the temporal patterns of O_3 variability across those same monitors tends to be well correlated. Given that most of the O_3 epidemiological studies are time series in nature, the O_3 ISA notes that the stability of temporal profiles across monitors within most urban areas means that monitor selection will have little effect on the outcomes of an epidemiological study examining short-term exposure-related mortality or morbidity. For this reason, we conclude that generally intra-city heterogeneity in O_3 levels is not a significant factor likely to impact the risk assessment. One exception is LA which, due to its size and variation in O_3 sources and other factors impacting O_3 patterns such as topography, may display significant variation in ambient O_3 levels with a subsequent impact on risk. However, in the case of LA (as with the other urban study areas), we model risk using composite monitors which do not provide spatially-differentiated representations of exposure and consequently, we do not address this source of variability in the first draft analysis.

- **Variability in the patterns of ambient O_3 reduction across urban areas:** In simulating just meeting the current or alternative suites of standards, there can be considerable variability in the patterns of ambient O_3 reductions that result from different simulation approaches (i.e., they can be more localized, more regional, or some combination thereof). Given the secondary formation of O_3, variation in the spatial pattern of O_3 reductions is likely to be dampened somewhat. For the first draft REA, we have only included one strategy for simulating the just meeting the current O_3 standard (quadratic rollback). As noted in section 7.2.3, we may employ a more

sophisticated method for predicting ambient O_3 under current and alternate standard levels for the second Draft analysis. Therefore, while we have not rigorously evaluated potential variability in the reduction of O_3 levels in response to simulating the current standard level for the first draft analysis, we may have a more comprehensive treatment of the issue for the second Draft analysis.

- **Copollutant concentrations**: Recent studies examining the potential for confounding by PM (and it constituents) of the short-term exposure-related mortality effect yielded mixed results with some studies showing little attenuation, while other studies suggest modest attenuation (O_3 ISA section 6.6.3, U.S. EPA, 2012). However, the ISA concludes that "…across studies, the potential impact of PM indices on O_3-mortality risk estimates tended to be much smaller than the variation in O_3-mortality risk estimates across cities suggesting that O_3 effects are independent of the relationship between PM and mortality. Although some studies suggest that O_3-mortality risk estimates may be confounded by PM or its chemical components the interpretation of these results requires caution due to the limited PM datasets used as a result of the every-3rd- and 6th-day PM sampling schedule." (O_3ISA, section 6.6.3). While these observations suggest that copollutants confounding may not be a significant issue, stated concerns regarding the every 3^{rd} and 6^{th} day sampling schedule leave the possibility that the sampling strategy is masking a copollutants effect. Due to limits in available data from the multi-city O_3 mortality studies, we did not include multipollutant model specifications for mortality. Multipollutant effect estimates were available for a number of the respiratory morbidity endpoints, and we include risk results based on those estimates in the array of core results. Therefore, we are in a position to evaluate to some extent the potential impact of copollutants confounding on the respiratory effects category.

- **Demographics and socioeconomic-status (SES)-related factors:** Variability in population density, particularly in relation to elevated levels of O_3 has the potential to influence population risk, although the significance of this factor also depends on the degree of intra-urban variation in O_3 levels (as discussed above). In addition, community characteristics such as pre-existing health status, ethnic composition, SES and the age of housing stock (which can influence rates of air conditioner use thereby impacting rates of infiltration of O_3 indoors) can contribute to observed differences in O_3-related risk (discussed in O_3 ISA – section 2.5.4.5, U.S. EPA, 2012). Some of the heterogeneity observed in effect estimates between cities in the multicity studies may be due to these demographic and SES factors, and while we cannot determine how much of that heterogeneity is attributable to these factors, the degree of variability in effect estimates between cities in our analysis should help to capture some of the latent variability in SES and demographics.

- **Baseline incidence of disease**: We collected baseline health effects incidence data (for mortality and morbidity endpoints) from a number of different sources (see section 7.3.4). Often the data were available at the county-level, providing a relatively high degree of spatial refinement in characterizing baseline incidence given the overall level of spatial refinement reflected in the risk assessment as a whole. Otherwise, for urban study areas without county-level data, either (a) a surrogate urban study area (with its baseline incidence rates) was used, or (b) less refined state-level incidence rate data were used.

7.4.2 Qualitative Assessment of Uncertainty

As noted in section 7.4, we have based the design of the uncertainty analysis carried out for this risk assessment on the framework outlined in the WHO guidance document (WHO, 2008). That guidance calls for the completion of a Tier 1 qualitative uncertainty analysis, provided the initial Tier 0 screening analysis suggests there is concern that uncertainty associated with the analysis is sufficient to significantly impact risk results (i.e., to potentially affect decision making based on those risk results). Given previous sensitivity analyses completed for prior O_3 NAAQS reviews, which have shown various sources of uncertainty to have a potentially significant impact on risk results, we believe that there is justification for conducting a Tier 1 analysis.

For the qualitative uncertainty analysis, we have described each key source of uncertainty and qualitatively assessed its potential impact (including both the magnitude and direction of the impact) on risk results, as specified in the WHO guidance. Similar to our discussion of variability in the last section, the term *key sources of uncertainty* refers to those sources that the EPA staff believes have the potential to play an important role in impacting population incidence estimates generated for this risk assessment (i.e., these sources of uncertainty, if fully addressed could result in adjustments to the core risk estimates which might impact the interpretation of those risk estimates in the context of the O_3 NAAQS review). These key sources of uncertainty have been identified through consideration for sensitivity analyses conducted for previous O_3 NAAQS risk assessments, together with information provided in the final O_3 ISA and comments provided by CASAC on the analytical plan for the risk assessment.

As shown in Table 7-6, for each source of uncertainty, we have (a) provided a description, (b) estimated the direction of influence (over, under, both, or unknown) and magnitude (low, medium, high) of the potential impact of each source of uncertainty on the risk estimates, (c) assessed the degree of uncertainty (low, medium, or high) associated with the knowledge-base (i.e., assessed how well we understand each source of uncertainty), and (d) provided comments further clarifying the qualitative assessment presented. Table 7-6 includes all key sources of uncertainty identified for the O_3 REA.

The categories used in describing the potential magnitude of impact for specific sources of uncertainty on risk estimates (i.e., low, medium, or high) reflect EPA staff consensus on the degree to which a particular source could produce a sufficient impact on risk estimates to influence the interpretation of those estimates in the context of the O_3 NAAQS review.[11] Sources

[11] For example, if a particular source of uncertainty were more fully characterized (or if that source was resolved, potentially reducing bias in a core risk estimate), could the estimate of incremental risk reduction in going from the current to an alternative standard level change sufficiently to produce a different conclusion regarding the magnitude of that risk reduction in the context of the O_3 NAAQS review?

classified as having a "low" impact would not be expected to impact the interpretation of risk estimates in the context of the O_3 NAAQS review; sources classified as having a "medium" impact have the potential to change the interpretation; and sources classified as "high" are likely to influence the interpretation of risk in the context of the O_3 NAAQS review. Because this classification of the potential magnitude of impact of sources of uncertainty is qualitative and not informed directly by any type of analytical results, it is not possible to place a quantitative level of impact on each of the categories. Therefore, the results of the qualitative analysis of uncertainty have limited utility in informing consideration of overall confidence in the core risk estimates and, instead, serve primarily as a means for guiding future research to reduce uncertainty related to O_3 risk assessment.

As with the qualitative discussion of sources of variability included in the last section, the characterization and relative ranking of sources of uncertainty addressed here is based on consideration by EPA staff of information provided in previous O_3 NAAQS risk assessments (particularly past sensitivity analyses), the results of risk modeling completed for the current O_3 NAAQS risk assessment and information provided in the third draft O_3 ISA as well as earlier O_3 Criteria Documents. Where appropriate, in Table 7-6, we have included references to specific

1

2 **Table 7-6 Summary of Qualitative Uncertainty Analysis of Key Modeling Elements in the O3 NAAQS Risk Assessment.**

Source	Description	Potential influence of uncertainty on risk estimates		Knowledge-Base uncertainty*	Comments (KB: knowledge base, INF: influence of uncertainty on risk estimates)
		Direction	Magnitude		
A. Characterizing ambient O_3 levels for study populations using the existing ambient monitoring network	If the set of monitors used in a particular urban study area to characterize population exposure as part of an ongoing risk assessment do not match the ambient monitoring data used in the original epidemiological study, then uncertainty can be introduced into the risk estimates.	Both	Low-medium	Low-medium	KB and INF: In modeling risk, we used a study area definition for each urban area based on the set of counties used in the Zanobetti and Schwartz (2008b) study of short-term exposure-related mortality. In those instances where other epidemiological studies used different county definitions in specifying the set of O_3 monitors used in characterizing uncertainty, then uncertainty may be introduced into the risk assessment and it is challenging to evaluate the nature and magnitude of the impact that that uncertainty would have on risk estimates, given the complex interplay of factors associated with mismatched monitoring networks (i.e., differences in the set of monitors used in modeling risk and those used in the underlying epidemiological study).
B. Characterizing U.S. Background O_3 levels	For this analysis, we have used modeling to estimate U.S. background levels for each urban study area. Depending on the nature of errors reflected in that modeling, uncertainty (in both directions) may be introduced into the analysis.	Both	Low	Low	INF: Given that the risk assessment focuses primarily on the reduction in risk associated with moving from recent conditions to simulated just meeting the current standard, the impact of uncertainty in U.S. background levels on the risk estimates is expected to be low, since generally, both recent conditions and current standard O_3 levels occur well above U.S. Background (for a particular day) and consequently, consideration of U.S. background does not factor into estimating the magnitude of deltas (risk reductions).
C. Characterizing intra-urban population exposure in the context of epidemiology studies linking O_3 to specific health effects	Exposure misclassification within communities that is associated with the use of generalized population monitors (which may miss important patterns of exposure within urban study areas) introduces uncertainty into the effect estimates obtained from epidemiology studies.	Under (generally)	Low-medium	Medium	KB and INF: Despite the potential for substantial variability in O_3 levels across monitors (particularly in larger urban areas with greater variation in sources and topography such as L.A.), the ISA notes that studies have tended to demonstrate that monitor selection has only a limited effect on the association of short-term O_3 exposure with health effects (see ISA section???). However, s noted here, this issue could be more of a concern in larger urban areas which may exhibit greater variation in O_3 levels due to diverse sources, topography and patterns of commuting.
D. Statistical fit	Exposure measurement error	Both	Medium	Medium	INF: For short-term mortality and morbidity health endpoints, there is

Source	Description	Potential influence of uncertainty on risk estimates		Knowledge-Base uncertainty*	Comments (KB: knowledge base, INF: influence of uncertainty on risk estimates)
		Direction	Magnitude		
of the C-R functions	combined with other factors (e.g., size of the effect itself, sample size, control for confounders) can effect the overall level of confidence associated with the fitting of statistical effect-response models in epidemiological studies.		(short-term health endpoints)		greater uncertainty associated with the fit of models given the smaller sample sizes often involved, difficulty in identifying the etiologically relevant time period for short-term O_3 exposure, and the fact that models tend to be fitted to individual counties or urban areas (which introduces the potential for varying degrees of confounding and effects modification across the locations). These studies can also have effects estimates that are not statistically significant. Note, however that for this risk assessment, in modeling short-term mortality, we are not relying on location-specific models. Instead, we are using city-specific effects estimates derived using Bayesian techniques (these combine national-scale models with local-scale models).
E. Shape of the C-R functions	Uncertainty in predicting the shape of the C-R function, particularly in the lower exposure regions which are often the focus in O_3 NAAQS regulatory reviews.	Both	Medium	Low-medium	KB and INF: Studies reviewed in the O_3 ISA that attempt to characterize the shape of the O3 C-R curve along with possible "thresholds" (i.e., O3 concentrations which must be exceeded in order to elicit an observable health response) have indicated a generally linear C-R function with no indication of a threshold (for analyses that have examined 8-h max and 24-h avg O_3 concentrations). However, the ISA notes that there is less certainty in the shape of the C-R curve at the lower end of the distribution of O_3 concentrations due to the low density of data in this range. Therefore, while there is increased uncertainty in specifying the nature of the C-R function at lower exposure levels, we do not believe that the risk drops to zero outside of the range of O_3 data used in the underlying epidemiological study providing the C-R function. As discussed in section 7.1.1, we are including risk estimates where we model exposure down to a surrogate for the LML of the underlying epidemiological study in order to evaluate the impact of modeling risk over a range of exposures where we have greater confidence (relative to modeling all the way down to zero O_3).
F. Surrogate LMLs used in defining ranges of increased confidence in estimating risk	Ideally, we would use LMLs from epidemiological studies supporting the C-R functions used in modeling risk to identify a range of O_3 concentrations with greater	Both	Medium	Low-medium	INF: Because the surrogate LMLs are based on individual years not matched to the analysis periods used in the epidemiological studies underlying the C-F functions, there is uncertainty associated with use of the surrogate LMLs. In addition, there is the potential that that way the composite monitor distributions were designed (surrogate LMLs are obtained from these distributions) may differ from the way air

Source	Description	Potential influence of uncertainty on risk estimates		Knowledge-Base uncertainty*	Comments (KB: knowledge base, INF: influence of uncertainty on risk estimates)
		Direction	Magnitude		
	confidence in modeling risk (i.e., only modeling risk matching the range of data used in fitting the C-R function). However, data limitations meant that we used surrogate LMLs in place of the study-specific LMLs (the surrogate LMLs were obtained from the composite monitor distributions used in risk modeling – see section 7.1.1).				quality data were used in the epidemiological studies - this would add additional uncertainty into the use of the surrogate LMLs. KB: we do not have comprehensive LML data form any of the epidemiological studies at this time and therefore, are not able to rigorously evaluate the degree to which the surrogate LMLs match actual study-based LMLs.[12]
G. Addressing co-pollutants	The inclusion or exclusion of co-pollutants which may confound, or in other ways, affect the O_3 effect, introduces uncertainty into the analysis.	Both	Low-medium	Medium	KB and INF: The O_3 ISA notes that across studies, the potential impact of PM indices on O3-mortality risk estimates tended to be much smaller than the variation in O3-mortality risk estimates across cities. This suggests that O3 effects are independent of the relationship between O_3 and mortality. However, interpretation of the potential confounding effects of PM on O3-mortality risk estimates requires caution. This is because the PM-O3 correlation varies across regions, due to the difference in PM components, complicating the interpretation of the combined effect of PM on the relationship between O3 and mortality. Additionally, the limited PM or PM component datasets used as a result of the every-3rd- and 6th-day PM sampling schedule instituted in most cities limits the overall sample size employed to examine whether PM or one of its components confounds the O3-mortality relationship (ISA section 2.5.4.5).
H. Specifying lag structure (short-term exposure studies)	There is uncertainty associated with specifying the exact lag structure to use in modeling short-term exposure-related mortality and respiratory-	Both	Low-Medium	Low	KB and INF: The majority of studies examining different lag models suggest that O_3 effects on mortality occur within a few days of exposure. Similar, studies examining the impact of O_3 exposure on respiratory-related morbidity endpoints suggests a rather immediate response, within the first few days of O_3 exposure (see ISA section

[12] We are in the process of evaluating descriptive statistics (including LMLs) reflecting data used in Zanobetti and Schwartz (2008b). However at the time of the first draft REA, we were not yet in a position to use these data to complete a rigorous performance evaluation of the surrogate LMLs developed for this (or other) health endpoints modeled in the analysis.

Source	Description	Potential influence of uncertainty on risk estimates		Knowledge-Base uncertainty*	Comments (KB: knowledge base, INF: influence of uncertainty on risk estimates)
		Direction	Magnitude		
	related morbidity.				2.5.4.3). Consequently, while the exact nature of the ideal lag models remains uncertain, generally, we are fairly confident that they would be on the order of a day to a few days following exposure.
I. Using studies from one geographic area to cover urban areas outside of the study area	In the case of Gent et al., 2003 (used in modeling asthma exacerbations in Boston), we are using C-R functions based on an epidemiological study of a region (northern Connecticut and Springfield) that does not encompass the actual urban study area assessed for risk (Boston).	Both	Medium	Low	INF: Factors related to O_3 exposure including commuting patterns, exercise levels etc may differ between the region reflected in the epidemiological study and Boston. If these differences are great, then applying the effect estimate from the epidemiological study to Boston could be subject to considerable uncertainty and potential bias. We have not conducted a more rigorous comparison of the two locations with regard to attributes impacting O_3 (including monitor levels) but that may be undertaken as part of the second draft ERA in order to increase our understanding of potential uncertainty associated with this category of risk estimate.
J. Characterizing baseline incidence rates	Uncertainty can be introduced into the characterization of baseline incidence in a number of different ways (e.g., error in reporting incidence for specific endpoints, mismatch between the spatial scale in which the baseline data were captured and the level of the risk assessment).	Both	Low-medium	Low	INF: The degree of influence of this source of uncertainty on the risk estimates likely varies with the health endpoint category under consideration. There is no reason to believe that there are any systematic biases in estimates of the baseline incidence data. The influence on risk estimates that are expressed as incremental risk reductions between alternative standards should be relatively unaffected by this source of uncertainty. KB: The county level baseline incidence and population estimates at the county level were obtained from data bases where the relative degree of uncertainty is low.

1 * Refers to the degree of uncertainty associated with our understanding of the phenomenon, in the context of assessing and characterizing its uncertainty
2 (specifically in the context of modeling PM risk)
3
4
5

sources of information considered in arriving at a ranking and classification for a particular source of uncertainty.

7.5 URBAN STUDY AREA RESULTS

This section presents and discusses risk estimates generated for the set of 12 urban study areas, including estimates generated to characterize recent O_3 conditions as well as estimates generated after simulated just meeting the current O_3 standard level in each urban study area. Risk estimates for alternative standard levels will be generated as part of the second draft analysis.

A number of details regarding these risk estimates should be kept in mind when reviewing the estimates presented in this section:

- **All risk estimates presented represent core (higher confidence) estimates – sensitivity analyses will be completed for the second draft analysis:** As discussed in section 7.1.1, the risk estimates generated for the first draft analysis focus on an array of core (higher confidence) analyses. A supporting set of comprehensive sensitivity analyses to help interpret overall confidence in the core estimates will be included in the second draft analysis. However, specifically in the case of short-term exposure-related morbidity, the array of core analyses includes coverage for a variety of design elements (including multi-/single-pollutant models and lag structures) and therefore, the array of core risk estimates does inform our consideration of the impact that these design elements has on risk estimates for this category of morbidity endpoints.

- **Estimates are presented for two simulation years (2007 and 2009):** Each simulation year represents the middle year of a 3 year attainment period (2006-2008 and 2008-2010, respectively). The two attainment periods were selected to provide coverage for generally lower and higher O_3 periods (i.e., 2006-2008 being relatively higher in general terms compared with the 2008-2010 period although this does not hold across all 12 urban study areas).

- **All estimates reflect short-term exposure-related endpoints**: Analysis of evidence presented in the O_3 ISA combined with consideration for the availability of data required to model specific health endpoints resulted in our designing the first draft REA to cover the health endpoints listed at the beginning of this section which are all related to short-term O_3 exposure. We also completed a review of evidence supporting modeling of long-term exposure-related mortality and morbidity. Treatment of those endpoints categories as planned for the second Draft analysis is discussed below in section 7.7.3.

- **Short-term exposure-related <u>mortality</u> estimates are generated for all 12 urban study areas, while most <u>morbidity</u> estimates (depending on the specific health endpoint) are generated for only a subset of urban study areas:** All mortality estimates are generated using Bayes-adjusted city-specific effect estimates obtained form Bell et al., (2004) (for all-cause mortality only) and Zanobetti and Schwartz

(2008b) (for all-cause, respiratory and cardiovascular-related mortality). For morbidity endpoints, coverage for the urban study areas differed depending on the specific endpoint with (a) ER visits evaluated for Atlanta and New York City, (b) HA evaluated for all 12 urban study areas with additional coverage for New York, Detroit and LA and (c) asthma exacerbations evaluated for Boston.

- **For short-term exposure-related mortality, we include two types of risk estimates for each scenario which, when considered together, inform consideration of uncertainty related to application of the C-R functions at low O_3 levels**: For short-term exposure-related mortality, we include (a) estimates of risk reflecting modeling of exposure down to zero O_3 and (b) higher confidence estimates of risk reflecting exposures modeled down to a surrogate for the LML used in fitting the C-R function (see 7.1.1). While risk modeled down to the LML has greater overall confidence since we are modeling exposure reflected in the fitting of the C-R function, estimates bounded by the LML are also likely biased low since they do not include exposures between the LML and zero O_3. By contrast, estimates of risk all the way to zero O_3 benefit from considering the full range of exposure, but also incorporate a range of exposure associated with reduced confidence in modeling risk (i.e., O_3 levels below those used in fitting the C-R function used in modeling risk). When considered together these two types of risk estimates inform consideration of uncertainty related to application of the C-R function at low O_3 levels. It is important to point out that only the LML-based risk estimates were generated for the short-term exposure-related morbidity endpoints (these did not include estimates based on modeling exposure down to zero O_3).

There are several categories of risk metrics generated for the mortality and morbidity endpoints modeled in this analysis. These metrics are described below (these descriptions are separated into *mortality-related tables* and *morbidity-related tables*):

I. Tables presenting mortality estimates

- **Heat map tables for mortality illustrating distribution of mortality across daily O_3 levels (Tables 7-7 through 7-10)**: The heat map tables illustrate the distribution of estimated O_3-related deaths across daily O_3 levels for each city. The color gradient reflects the distribution of mortality across the range of daily 8-hour ozone levels with colors ranging from green (low) to red (high). The color gradients are a visual tool to explore trends in mortality counts across daily O_3 levels and between cities. As an example, with Table 7-7 (which presents recent conditions mortality risk estimates for 2007 based on Zanobetti and Schwartz, 2008b C-R functions), the value of 72 in the "New York" row and "60-65" column represents the fact that 72 of the total of 708 deaths estimated for New York city occurred on days with O_3 levels between 60 and 65 ppb. Similarly, in that same table, we see that only 13 of the estimated deaths in New York City occurred on days with 8hr mean O_3 levels between 20 and 25 ppb. The heat map tables allow us to evaluate which days (in terms of O_3 levels) are associated with the majority of estimated O_3-related deaths. When we compare heat

map tables between recent conditions and simulating just meeting the current standard, we can look at how that distribution of estimated O_3-related deaths across daily O_3 levels shifts (i.e., the entire distribution shifts to the left, reflecting the fact that the distribution of daily O_3 levels is reduced when we simulate just meeting the current standard). Separate sets of heat map tables were generated using C-R functions based on Bell et al., (2004) and Zanobetti and Schwartz (2008b). The heat-map tables were only generated for the 2007 simulation year, given that the general pattern displayed in these tables would also hold for 2009. In addition, heat-map tables were only generated for all-cause mortality – the patterns displayed in the table would hold for other mortality categories modeled in the analysis. Estimates presented in the heat-map tables reflect application of the LMLs (i.e., risks were modeled down to LML, and not down to zero).

- **Tables presenting estimates of O_3-related mortality with consideration for ranges of increased confidence defined based on the composite monitor LMLs (Tables 7-11 Through 7-14):** As discussed in sections 7.1.1 and 7.1.2, rather than incorporating a biological threshold into modeling risk, we have defined ranges of increased confidence corresponding to levels of O_3 similar to those used in the epidemiological studies providing the C-R functions used in the analysis. However, as noted in those earlier sections, due to data limitations we used statistics obtained from the set of composite monitor values used in modeling risk as surrogates for statistics that would have come from the actual epidemiological studies. Specifically, we estimated risks down to LMLs from the composite monitor data sets. Estimates of risk presented in these tables include estimates modeled all the way down to zero to establish a baseline of the highest potential estimated risk. Estimates presented in Tables 7-11 through 7-14, reflect all-cause mortality and include 95th percentile confidence intervals representing uncertainty associated with the statistical fit of the effect estimates used. Estimates are presented based both on Bell et al., (2004) and Zanobetti and Schwartz (2008b) C-R functions. Note, that 95th% confidence intervals are not presented for the delta (risk reduction) estimates since these were calculated off of point estimates (for the recent conditions and current standard level) and were not based on separate model runs for the delta O_3 levels. Estimates presented in these tables allow for consideration for the pattern of risk reduction (in incidence) in going from recent conditions to just meeting the current standard level and how that pattern varies across urban study areas. Estimates in these tables also illustrate how risk changes when consideration is given to different levels of confidence about risks attributable to O_3 concentrations at the lower end of the observed O_3 data used in the underlying epidemiology studies.

- **Tables comparing cause-specific mortality for the recent conditions (2007) scenario:** Table 7-15 presents estimates of cause-specific mortality (all-cause, respiratory and cardiovascular) for the 2007 simulation year based on C-R functions obtained from Zanobetti and Schwartz (2008b). These tables include consideration for the range of increased confidence defined using the LMLs as cutoffs for modeling risk. The estimates presented in these tables allow consideration for differences in the magnitude of mortality risk associated with different mortality categories.

- **Tables presenting estimates of the percent of total mortality attributable to O_3:** Tables 7-16 through 7-19 present estimates of the percent of total (all-cause) mortality attributable to O_3 for the recent conditions and simulation of the current standard scenarios and for the delta (risk reduction) between these two scenarios. Estimates presented in these tables include those generated with consideration for ranges of increased confidence based on the composite monitor LMLs, as well as estimates of risk based on modeling all the way to zero O_3. Results are presented based on estimates of mortality derived using C-R functions obtained both from Bell et al., (2004) and Zanobetti and Schwartz (2008b). Estimates presented in these tables allow for consideration for the pattern of risk reduction (in terms of the percent of total mortality) in going from recent conditions to just meeting the current standard level and how that pattern varies across urban study areas. Estimates in these tables also illustrate how risk changes when consideration is given to different levels of confidence about risks attributable to O_3 concentrations at the lower end of the observed O_3 data used in the underlying epidemiology studies.

- **Tables presenting estimates of the percent reduction in ozone-related mortality incidence:** Table 7-20 presents estimates of the reduction in ozone-related mortality incidence in going from recent conditions to the simulation of the current ozone standard level. This table includes consideration for the range of increased confidence defined based on composite monitor LMLs, as well as estimates of risk based on modeling all the way to zero O_3. Results are presented based on estimates of mortality derived using C-R functions obtained both from Bell et al., (2004) and Zanobetti and Schwartz (2008b). Estimates presented in these tables allow consideration for how the pattern of reductions in ozone-related mortality (in going from recent conditions to meeting the current standard) varies across urban study areas. Estimates in these tables also illustrate how risk changes when consideration is given to different levels of confidence about risks attributable to O_3 concentrations at the lower end of the observed O_3 data used in the underlying epidemiology studies.

II. Tables presenting morbidity estimates

- **Table summarizing risk estimates for short-term exposure-related ER visits (for respiratory symptoms including asthma):** Table 7-21 presents estimates of the incidence of ER visits for respiratory symptoms and asthma) specifically for New York City and Atlanta based on C-R functions obtained from several epidemiological studies. The C-R functions available for modeling this category of health effect endpoints included consideration for a number of design elements (copollutants and lag structure). Therefore, while the set of risk estimates presented in these tables does collectively represent the core simulation for this endpoint, consideration for different design elements also allows us to evaluate their potential impact on core risk estimates. Risk estimates presented in these tables include: (a) point estimates and 95^{th} percentile estimates for O_3-attributable incidence, (b) percent of baseline incidence (the increment of total ER attributable to O_3 exposure), (c) risk reductions (deltas) in both O_3-related incidence and the fraction of total incidence attributable to O_3 and (d) reduction in O_3-related mortality.

- **Tables summarizing risk estimates for short-term exposure-related HA visits (for respiratory symptoms including asthma):** Tables 7-22 and 7-23 present estimates of the incidence of HA (for respiratory symptoms, chronic lung disease and asthma). Risk estimates are generated for a subset of the urban study areas for some of the health endpoints (e.g., New York City for HA [chronic lung disease and asthma]), while HA (respiratory-related) estimates cover all 12 urban study areas. These estimates include the same mix of risk metrics and other parameters described for the ER-visit estimates (see above).

- **Table summarizing risk estimates for short-term exposure-related asthma exacerbation:** Table 7-24 presents estimates of the incidence of asthma exacerbations (including estimates for a range of symptoms) for Boston (the only urban study area with C-R functions supporting modeling for this endpoint). Risk estimates presented in Table 7-24 include consideration for a number of modeling elements (O_3 metrics, lag structure and copollutants). The array of risk estimates presented in these tables collectively represents the core simulation for this endpoint. Consideration for different design elements allows us to evaluate their potential impact on core risk estimates. As with the other short-term exposure-related morbidity risk estimates, estimates presented in this tables include: (a) point estimates and 95th percentile estimates for O_3-attributable incidence, (b) percent of baseline incidence (the increment of total ER attributable to O_3 exposure), (c) risk reductions (deltas) in both O_3-related incidence and the fraction of total incidence attributable to O_3 and (d) reduction in O_3-related mortality.

In reviewing the risk estimates generated for the first draft analysis we have focused on developing a set of key observations reflecting consideration for goals originally set out for the risk assessments in the Scope and Methods Plan (U.S. EPA, 2011). These goals included:

- Provide estimates of the potential magnitude of premature mortality and/or selected morbidity health effects associated with recent conditions and with the simulated just meeting just meeting the current suite of O_3 standards and any alternative standards that might be considered in selected urban study areas (note, alternative standards will be evaluated in the second Draft analysis).

- 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 populations.

- Gain insights into the distribution of risks and patterns of risk reduction and uncertainties in those risk estimates.

Typically, the last two bullets are addressed primarily through sensitivity analysis runs that provide additional perspective on the impact of varying modeling elements (including aspects of C-R function specification) on risk estimates. These sensitivity analyses will be included in the second draft REA– see section 7.7.1. Therefore, the discussion presented below focuses primarily on characterizing the magnitude of risk and risk reduction associated with the

O_3 scenarios modeled and also provides some ally insights on the distribution of risks and patterns of risk reduction.

Table 7-7 Heat Map Table: Short-Term O_3 Exposure-Related All-Cause Mortality – Recent Conditions (2007) (Zanobetti and Schwartz, 2008b C-R functions) (illustrates distribution of O_3-related all-cause mortality across distribution of daily 8hr mean O_3 levels for each urban study area – colors in cells reflect size of mortality estimate)

	Daily 8hr Mean Ozone Level (ppb)																
Study area	0-5	5-10	10-15	15-20	20-25	25-30	30-35	35-40	40-45	45-50	50-55	55-60	60-65	65-70	70-75	>75	Total
Atlanta, GA	0	0	0	0	0	0	1	1	1	2	5	7	7	8	5	19	56
Baltimore, MD	0	0	0	0	0	1	1	6	11	12	12	11	15	4	4	3	84
Boston, MA	0	0	0	0	1	7	5	13	9	17	15	15	12	11	3	14	123
Cleveland, OH	0	0	0	1	1	2	7	8	11	13	9	7	5	10	2	3	78
Denver, CO	0	0	0	0	0	0	0	0	1	1	3	2	1	0	0	0	10
Detroit, MI	0	0	0	0	0	4	5	10	17	20	17	12	9	15	8	19	135
Houston, TX	0	0	0	1	2	1	2	3	2	1	2	2	0	1	1	0	20
Los Angeles, CA	0	0	0	0	0	0	0	1	5	10	16	27	12	11	7	6	96
New York, NY	0	0	2	1	13	41	26	95	102	61	68	33	72	117	29	47	708
Philadelphia, PA	0	0	0	0	1	2	4	4	8	13	11	8	14	7	5	9	87
Sacramento, CA	0	0	0	0	0	0	0	1	3	5	5	3	6	3	1	3	30
St. Louis, MO	0	0	0	0	0	0	1	3	6	8	7	10	10	10	7	24	86

Table 7-8 Heat Map Table: Short-Term O_3 Exposure-Related All-Cause Mortality – Simulation of Meeting the Current Standard (2007) (Zanobetti and Schwartz, 2008b C-R functions) (illustrates distribution of O_3-related all-cause mortality across distribution of daily 8hr mean O_3 levels for each urban study area – colors in cells reflect size of mortality estimate)

	Daily 8hr Mean Ozone Level (ppb)																	
Study area	0-5	5-10	10-15	15-20	20-25	25-30	30-35	35-40	40-45	45-50	50-55	55-60	60-65	65-70	70-75	>75	Total	Delta
Atlanta, GA	0	0	0	0	0	0	1	1	3	5	7	8	9	4	2	1	42	14
Baltimore, MD	0	0	0	0	1	1	3	12	12	12	16	7	4	3	0	0	71	13
Boston, MA	0	0	0	0	3	5	7	15	11	17	15	11	12	5	3	7	110	13
Cleveland, OH	0	0	0	1	2	2	9	10	12	10	8	7	6	3	1	0	72	7
Denver, CO	0	0	0	0	0	0	0	1	1	3	2	1	0	0	0	0	9	2
Detroit, MI	0	0	0	0	0	5	5	11	24	11	17	12	13	7	11	6	122	14
Houston, TX	0	0	0	1	2	1	2	3	1	2	2	1	1	0	0	0	17	3
Los Angeles, CA	0	0	0	0	0	0	0	6	14	16	9	5	0	0	0	0	50	46
New York, NY	0	0	3	4	17	35	64	113	44	102	39	130	48	13	14	0	626	81
Philadelphia, PA	0	0	0	0	2	2	4	7	16	10	7	13	4	3	2	3	72	14
Sacramento, CA	0	0	0	0	0	0	0	2	4	5	5	2	1	0	1	0	20	11
St. Louis, MO	0	0	0	0	0	0	2	6	6	6	12	9	10	10	10	3	73	13

Table 7-9 Heat Map Table: Short-Term O_3 Exposure-Related All-Cause Mortality – Recent Conditions (2007) (Bell et al, 2004 C-R functions) (illustrates distribution of O_3-related all-cause mortality across distribution of daily 8hr max O_3 levels for each urban study area – colors in cells reflect size of mortality estimate)

	Daily 8hr Max Ozone Level (ppb)																
Study area	0-5	5-10	10-15	15-20	20-25	25-30	30-35	35-40	40-45	45-50	50-55	55-60	60-65	65-70	70-75	>75	Total
Atlanta, GA	0	0	0	0	1	3	10	13	17	22	42	51	39	40	34	52	323
Baltimore, MD	0	0	0	0	2	4	6	14	14	14	14	10	16	4	5	3	106
Boston, MA	0	0	0	0	6	20	26	32	43	39	23	33	13	26	19	26	307
Cleveland, OH	0	0	0	0	1	5	7	13	13	15	14	9	6	10	7	7	109
Denver, CO	0	0	0	0	1	1	2	3	5	6	6	6	2	1	0	0	32
Detroit, MI	0	0	0	0	1	2	6	9	13	17	5	10	6	6	5	13	94
Houston, TX	0	0	2	7	18	23	34	30	26	28	21	15	9	19	12	2	244
Los Angeles, CA	0	0	1	10	26	41	69	66	99	87	103	88	46	40	24	27	729
New York, NY	0	0	0	15	22	60	69	70	99	60	49	40	50	73	27	23	658
Philadelphia, PA	0	0	0	0	1	4	6	10	11	11	12	11	11	8	7	7	98
Sacramento, CA	0	0	0	1	3	5	9	14	14	19	17	8	8	3	4	4	110
St. Louis, MO	0	0	0	0	1	3	8	14	18	16	24	21	24	10	9	25	174

Table 7-10 Heat Map Table: Short-Term O_3 Exposure-Related All-Cause Mortality – Simulation of Meeting the Current Standard (2007) (Bell et al., 2004 C-R functions) (illustrates distribution of O_3-related all-cause mortality across distribution of daily 8hr max O_3 levels for each urban study area – colors in cells reflect size of mortality estimate)

	Daily 8hr Max Ozone Level (ppb)																	
Study area	0-5	5-10	10-15	15-20	20-25	25-30	30-35	35-40	40-45	45-50	50-55	55-60	60-65	65-70	70-75	>75	Total	Delta
Atlanta, GA	0	0	0	0	2	5	12	18	21	57	43	45	29	16	6	4	260	63
Baltimore, MD	0	0	0	0	3	4	10	15	15	15	14	8	5	2	0	0	90	16
Boston, MA	0	0	0	1	8	23	24	48	26	39	32	14	26	24	7	11	282	26
Cleveland, OH	0	0	0	1	2	5	11	13	16	14	10	9	9	5	2	2	98	11
Denver, CO	0	0	0	0	1	1	3	3	7	7	5	2	0	0	0	0	30	3
Detroit, MI	0	0	0	0	1	3	7	10	17	9	9	7	7	6	6	4	86	8
Houston, TX	0	0	2	8	22	24	37	31	28	21	12	18	11	1	0	0	217	27
Los Angeles, CA	0	0	2	17	35	64	70	119	113	81	44	15	7	0	0	0	567	162
New York, NY	0	0	1	13	31	69	76	103	55	63	50	66	44	0	13	0	585	73
Philadelphia, PA	0	0	0	0	2	5	8	12	12	12	8	11	5	2	1	2	82	16
Sacramento, CA	0	0	0	1	3	7	15	14	21	13	8	5	3	0	1	0	90	20
St. Louis, MO	0	0	0	0	2	4	10	20	16	23	24	21	12	12	10	4	157	17

Table 7-11 Short-Term O_3 Exposure-Related All Cause Mortality Incidence (2007) (Zanobetti and Schwartz, 2008b C-R Functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone-Exposure Related All-Cause Mortality (2007) (Zanobetti and Schwartz, 2008b)					
	Recent conditions		Current standard		Delta (risk reduction)	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	94	56	80	42	14	14
	(-88 - 269)	(-51 - 160)	(-74 - 230)	(-39 - 121)	NA	
Baltimore, MD	117	84	104	71	13	13
	(-23 - 252)	(-17 - 182)	(-21 - 225)	(-14 - 155)	NA	
Boston, MA	223	123	209	110	14	13
	(13 - 426)	(7 - 236)	(12 - 401)	(6 - 211)	NA	
Cleveland, OH	92	78	85	72	7	6
	(-15 - 196)	(-13 - 167)	(-14 - 182)	(-12 - 153)	NA	
Denver, CO	18	10	16	9	2	1
	(-23 - 58)	(-13 - 33)	(-21 - 53)	(-11 - 28)	NA	
Detroit, MI	226	135	212	122	14	13
	(82 - 365)	(49 - 220)	(77 - 344)	(44 - 198)	NA	
Houston, TX	29	20	26	17	3	3
	(-63 - 118)	(-43 - 81)	(-57 - 107)	(-37 - 70)	NA	
Los Angeles, CA	227	96	180	50	47	46
	(-121 - 566)	(-51 - 241)	(-96 - 451)	(-27 - 126)	NA	
New York, NY	931	708	849	626	82	82
	(544 - 1310)	(412 - 997)	(495 - 1197)	(365 - 884)	NA	
Philadelphia, PA	116	87	102	72	14	15
	(2 - 227)	(1 - 170)	(1 - 200)	(1 - 142)	NA	
Sacramento, CA	74	30	63	20	11	10
	(-26 - 170)	(-10 - 71)	(-22 - 146)	(-7 - 45)	NA	
St. Louis, MO	143	86	130	73	13	13
	(-29 - 308)	(-17 - 186)	(-27 - 281)	(-15 - 159)	NA	

Table 7-12 Short-Term O_3 Exposure-Related All Cause Mortality Incidence (2009) (Zanobetti and Schwartz, 2008b C-R Functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone-Exposure Related All-Cause Mortality (2007) (Zanobetti and Schwartz,					
	Recent conditions		Current standard		Delta (risk reduction)	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	77	43	73	40	4	3
	(-71 - 221)	(-40 - 125)	(-68 - 211)	(-37 - 115)	NA	
Baltimore, MD	113	56	103	45	10	11
	(-22 - 245)	(-11 - 121)	(-20 - 222)	(-9 - 98)	NA	
Boston, MA	185	98	180	93	5	5
	(10 - 354)	(5 - 189)	(10 - 345)	(5 - 179)	NA	
Cleveland, OH	81	49	79	46	2	3
	(-14 - 174)	(-8 - 104)	(-13 - 168)	(-8 - 99)	NA	
Denver, CO	17	9	16	8	1	1
	(-22 - 53)	(-12 - 29)	(-21 - 52)	(-11 - 27)	NA	
Detroit, MI	178	128	178	127	0	1
	(64 - 288)	(46 - 207)	(64 - 288)	(46 - 207)	NA	
Houston, TX	32	19	30	17	2	2
	(-70 - 132)	(-41 - 78)	(-65 - 122)	(-36 - 69)	NA	
Los Angeles, CA	215	123	175	83	40	40
	(-115 - 537)	(-66 - 309)	(-93 - 438)	(-44 - 210)	NA	
New York, NY	835	579	777	521	58	58
	(487 - 1176)	(337 - 817)	(453 - 1095)	(303 - 736)	NA	
Philadelphia, PA	92	60	86	54	6	6
	(1 - 180)	(1 - 117)	(1 - 169)	(1 - 106)	NA	
Sacramento, CA	75	32	64	21	11	11
	(-26 - 173)	(-11 - 73)	(-22 - 147)	(-7 - 48)	NA	
St. Louis, MO	108	53	105	50	3	3
	(-22 - 234)	(-11 - 116)	(-21 - 228)	(-10 - 110)	NA	

Table 7-13 Short-Term O_3 Exposure-Related All Cause Mortality Incidence (2007) (Bell et al., 2004 C-R Functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone-Exposure Related All-Cause Mortality (2007) (Zanobetti and Schwartz,					
	Recent conditions		Current standard		Delta (risk reduction)	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	479	323	415	260	64	63
	(181 - 769)	(122 - 520)	(157 - 668)	(98 - 419)	NA	
Baltimore, MD	153	106	137	90	16	16
	(-70 - 370)	(-48 - 257)	(-63 - 332)	(-41 - 219)	NA	
Boston, MA	430	307	404	282	26	25
	(105 - 748)	(75 - 535)	(98 - 704)	(68 - 491)	NA	
Cleveland, OH	151	109	140	98	11	11
	(-59 - 355)	(-43 - 256)	(-55 - 330)	(-38 - 231)	NA	
Denver, CO	36	32	33	30	3	2
	(-21 - 92)	(-19 - 83)	(-19 - 85)	(-17 - 76)	NA	
Detroit, MI	132	94	124	86	8	8
	(-71 - 330)	(-51 - 237)	(-67 - 309)	(-46 - 216)	NA	
Houston, TX	297	244	270	217	27	27
	(-102 - 687)	(-83 - 564)	(-92 - 625)	(-74 - 503)	NA	
Los Angeles, CA	950	729	786	567	164	162
	(-379 - 2243)	(-290 - 1725)	(-313 - 1862)	(-225 - 1346)	NA	
New York, NY	901	658	827	585	74	73
	(-168 - 1940)	(-122 - 1421)	(-154 - 1784)	(-109 - 1265)	NA	
Philadelphia, PA	139	98	123	82	16	16
	(-97 - 368)	(-68 - 260)	(-86 - 325)	(-57 - 217)	NA	
Sacramento, CA	163	110	142	90	21	20
	(-63 - 382)	(-42 - 259)	(-55 - 335)	(-34 - 212)	NA	
St. Louis, MO	210	174	193	157	17	17
	(-106 - 516)	(-88 - 429)	(-97 - 474)	(-79 - 387)	NA	

Table 7-14 Short-Term O_3 Exposure-Related All Cause Mortality Incidence (2009) (Bell et al., 2004 C-R Functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone-Exposure Related All-Cause Mortality (2007) (Zanobetti and Schwartz,					
	Recent conditions		Current standard		Delta (risk reduction)	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	381	332	364	315	17	17
	(144 - 614)	(125 - 534)	(138 - 586)	(119 - 507)	NA	
Baltimore, MD	145	112	132	99	13	13
	(-66 - 351)	(-51 - 272)	(-60 - 320)	(-45 - 242)	NA	
Boston, MA	378	259	369	250	9	9
	(92 - 658)	(63 - 453)	(90 - 642)	(61 - 437)	NA	
Cleveland, OH	129	79	125	75	4	4
	(-50 - 303)	(-31 - 187)	(-49 - 295)	(-29 - 179)	NA	
Denver, CO	35	22	34	21	1	1
	(-20 - 88)	(-13 - 57)	(-20 - 86)	(-12 - 54)	NA	
Detroit, MI	110	72	110	72	0	0
	(-60 - 276)	(-39 - 182)	(-60 - 276)	(-39 - 182)	NA	
Houston, TX	292	231	272	211	20	20
	(-100 - 674)	(-79 - 534)	(-93 - 628)	(-72 - 489)	NA	
Los Angeles, CA	976	781	821	628	155	153
	(-389 - 2303)	(-311 - 1847)	(-326 - 1942)	(-249 - 1488)	NA	
New York, NY	820	630	764	576	56	54
	(-153 - 1767)	(-117 - 1362)	(-142 - 1649)	(-107 - 1245)	NA	
Philadelphia, PA	108	81	102	75	6	6
	(-76 - 287)	(-57 - 216)	(-71 - 270)	(-52 - 199)	NA	
Sacramento, CA	162	140	141	120	21	20
	(-62 - 380)	(-54 - 330)	(-54 - 332)	(-46 - 282)	NA	
St. Louis, MO	168	138	164	134	4	4
	(-85 - 414)	(-69 - 340)	(-83 - 404)	(-67 - 330)	NA	

Table 7-15 Pathway-Specific Mortality Incidence (2007 recent conditions) (Zanobetti and Schwartz, 2008b, C-R functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone Exposure-Related Mortality (recent conditions - 2007) (Zanobetti and Schwartz , 2008b)					
	Total		Respiratory		Cardiovascular	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	94	56	27	16	52	31
Baltimore, MD	117	84	19	13	81	58
Boston, MA	223	123	36	20	77	42
Cleveland, OH	92	78	11	10	45	38
Denver, CO	18	10	5	3	11	6
Detroit, MI	226	135	17	10	119	72
Houston, TX	29	20	6	4	33	22
Los Angeles, CA	227	96	33	14	94	40
New York, NY	931	708	64	49	451	343
Philadelphia, PA	116	87	11	9	57	42
Sacramento, CA	74	30	11	5	32	13
St. Louis, MO	143	86	20	12	80	48

Table 7-16 Percent of Total All-Cause Mortality Attributable to O_3 (2007) (Zanobetti and Schwartz, 2008b C-R functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone Exposure-Related All Cause Mortality - PERCENT of total baseline (2007) (Zanobetti and Schwartz, 2008b C-R functions)					
	Recent conditions		Current standard		Delta (reduction)	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	1.7	1.0	1.5	0.8	0.3	0.3
Baltimore, MD	2.4	1.8	2.2	1.5	0.3	0.3
Boston, MA	2.9	1.6	2.7	1.4	0.2	0.2
Cleveland, OH	2.5	2.2	2.4	2.0	0.2	0.2
Denver, CO	1.7	1.0	1.6	0.8	0.1	0.1
Detroit, MI	4.9	3.0	4.6	2.7	0.3	0.3
Houston, TX	0.5	0.4	0.5	0.3	0.05	0.05
Los Angeles, CA	1.5	0.6	1.2	0.3	0.3	0.3
New York, NY	4.6	3.5	4.2	3.1	0.4	0.4
Philadelphia, PA	3.0	2.2	2.6	1.9	0.4	0.4
Sacramento, CA	2.8	1.2	2.4	0.8	0.4	0.4
St. Louis, MO	3.0	1.8	2.7	1.6	0.3	0.3

Table 7-17 Percent of Total All-Cause Mortality Attributable to O_3 (2009) (Zanobetti and Schwartz, 2008b C-R functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone Exposure-Related All Cause Mortality - PERCENT of total baseline (2007) (Zanobetti and Schwartz, 2008b C-R functions)					
	Recent conditions		Current standard		Delta (reduction)	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	1.4	0.8	1.4	0.7	0.1	0.1
Baltimore, MD	2.4	1.2	2.2	1.0	0.2	0.2
Boston, MA	2.5	1.3	2.4	1.3	0.1	0.1
Cleveland, OH	2.4	1.4	2.3	1.4	0.1	0.1
Denver, CO	1.7	0.9	1.6	0.9	0.05	0.05
Detroit, MI	4.1	3.0	4.1	3.0	0.003	0.003
Houston, TX	0.6	0.4	0.6	0.3	0.04	0.04
Los Angeles, CA	1.4	0.8	1.2	0.6	0.3	0.3
New York, NY	4.3	3.0	4.0	2.7	0.3	0.3
Philadelphia, PA	2.5	1.6	2.3	1.5	0.2	0.2
Sacramento, CA	2.9	1.2	2.5	0.8	0.4	0.4
St. Louis, MO	2.3	1.2	2.3	1.1	0.1	0.1

Table 7-18 Percent of Total All-Cause Mortality Attributable to O_3 (2007) (Bell et al., 2004 C-R functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone Exposure-Related All Cause Mortality - PERCENT of total baseline (2007) (Bell et al., 2004 C-R functions)					
	Recent conditions		Current standard		Delta (reduction)	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	3.7	2.5	3.2	2.0	0.5	0.5
Baltimore, MD	1.5	1.0	1.3	0.9	0.2	0.2
Boston, MA	2.9	2.1	2.7	1.9	0.2	0.2
Cleveland, OH	1.9	1.4	1.8	1.2	0.1	0.1
Denver, CO	1.7	1.5	1.5	1.4	0.1	0.1
Detroit, MI	1.5	1.1	1.4	1.0	0.1	0.1
Houston, TX	1.5	1.3	1.4	1.1	0.1	0.1
Los Angeles, CA	1.7	1.3	1.4	1.0	0.3	0.3
New York, NY	2.0	1.5	1.8	1.3	0.2	0.2
Philadelphia, PA	1.7	1.2	1.5	1.0	0.2	0.2
Sacramento, CA	1.7	1.2	1.5	1.0	0.2	0.2
St. Louis, MO	2.0	1.7	1.9	1.5	0.2	0.2

Table 7-19 Percent of Total All-Cause Mortality Attributable to O_3 (2009) (Bell et al., 2004 C-R functions) (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Ozone Exposure-Related All Cause Mortality - PERCENT of total baseline (2007) (Zanobetti and Schwartz, 2008b C-R functions)					
	Recent conditions		Current standard		Delta (reduction)	
	no cutoff	LML cutoff	no cutoff	LML cutoff	no cutoff	LML cutoff
Atlanta, GA	2.9	2.6	2.8	2.4	0.1	0.1
Baltimore, MD	1.4	1.1	1.3	1.0	0.1	0.1
Boston, MA	2.7	1.8	2.6	1.8	0.1	0.1
Cleveland, OH	1.7	1.1	1.7	1.0	0.05	0.05
Denver, CO	1.7	1.1	1.7	1.1	0.05	0.05
Detroit, MI	1.4	0.9	1.4	0.9	0.0006	0.0005
Houston, TX	1.5	1.2	1.4	1.1	0.1	0.1
Los Angeles, CA	1.7	1.4	1.5	1.1	0.3	0.3
New York, NY	1.9	1.5	1.8	1.3	0.1	0.1
Philadelphia, PA	1.4	1.1	1.3	1.0	0.1	0.1
Sacramento, CA	1.7	1.5	1.5	1.3	0.2	0.2
St. Louis, MO	1.7	1.4	1.6	1.4	0.04	0.04

Table 7-20 Percent Reduction in Ozone-Attributable Short-Term Exposure-Related Mortality (*no cutoff* and *LML cutoff* columns present O_3-attributable risks modeled down to zero O_3 and the surrogate LML, respectively)

Urban study area	Percent Reduction in Ozone Exposure-Related All-Cause Mortality			
	Zanobetti and Scnwartz 2008b-based C-R functions		Bell et al., 2004-based C-R functions	
	no cutoff	LML cutoff	no cutoff	LML cutoff
2007 Simulation Year				
Atlanta, GA	15%	25%	13%	20%
Baltimore, MD	11%	15%	10%	15%
Boston, MA	6%	11%	6%	8%
Cleveland, OH	7%	8%	7%	10%
Denver, CO	8%	15%	8%	9%
Detroit, MI	6%	10%	6%	9%
Houston, TX	9%	13%	9%	11%
Los Angeles, CA	20%	48%	17%	22%
New York, NY	9%	11%	8%	11%
Philadelphia, PA	12%	16%	12%	17%
Sacramento, CA	15%	36%	12%	18%
St. Louis, MO	9%	15%	8%	10%
2009 Simulation Year				
Atlanta, GA	5%	8%	4%	5%
Baltimore, MD	9%	19%	9%	11%
Boston, MA	3%	5%	2%	4%
Cleveland, OH	3%	5%	3%	4%
Denver, CO	3%	5%	3%	4%
Detroit, MI	0.08%	0.11%	0.04%	0.06%
Houston, TX	7%	12%	7%	8%
Los Angeles, CA	19%	32%	16%	20%
New York, NY	7%	10%	7%	9%
Philadelphia, PA	6%	9%	6%	8%
Sacramento, CA	15%	35%	13%	15%
St. Louis, MO	3%	5%	3%	3%

Table 7-21 Short-Term Ozone Exposure-Related Morbidity (ER visits)

Urban study area (endpoint)	Study author	Effect estimator differentiators	Recent conditions					Simulation of meeting current standard					Delta (risk reduction)		
			point estimate	95th Confidence Interval			% of baseline	point estimate	95th Confidence Interval			% of baseline	point estimate	% of baseline	% reduction in ozone-related morbidity
				2.5		97.5			2.5		97.5				
2007 Simulation															
Atlanta GA															
ER visits (Resp)	Tolbert		5,054	3,496	-	6,586	4.5	4,076	2,814	-	5,320	3.7	979	0.9	19
ER visits (Resp)	Tolbert	CO	4,498	2,760	-	6,202	4.0	3,625	2,220	-	5,008	3.3	873	0.8	19
ER visits (Resp)	Tolbert	NO2	4,066	2,147	-	5,944	3.6	3,275	1,726	-	4,798	2.9	791	0.7	19
ER visits (Resp)	Tolbert	PM10	3,190	1,125	-	5,209	2.9	2,567	903	-	4,201	2.3	623	0.6	20
ER visits (Resp)	Tolbert	PM10, NO2	3,080	1,010	-	5,103	2.8	2,478	810	-	4,115	2.2	602	0.5	20
ER visits (Resp)	Darrow	Darrow	2,728	1,657	-	3,787	2.4	2,194	1,331	-	3,049	2.0	534	0.5	20
ER visits (Resp)	Strickland	dist lag 0-7	5,978	4,248	-	7,603	15.6	4,894	3,455	-	6,262	12.8	1,084	2.8	18
ER visits (Resp)	Strickland	avg day lag 0-2	3,522	1,922	-	5,037	9.2	2,858	1,551	-	4,109	7.5	664	1.7	19
New York															
ER visits (asthma)	Ito		10,232	6,951	-	13,312	13.5	9,199	6,223	-	12,015	12.1	1,034	1.4	10
ER visits (asthma)	Ito	PM2.5	7,974	4,270	-	11,433	10.5	7,149	3,810	-	10,294	9.4	826	1.1	10
ER visits (asthma)	Ito	NO2	6,572	2,939	-	9,972	8.6	5,881	2,619	-	8,962	7.7	691	0.9	11
ER visits (asthma)	Ito	CO	10,818	7,630	-	13,814	14.2	9,733	6,837	-	12,476	12.8	1,085	1.4	10
ER visits (asthma)	Ito	SO2	8,233	4,766	-	11,483	10.8	7,383	4,256	-	10,340	9.7	850	1.1	10
2009 Simulation															
Atlanta GA															
ER visits (Resp)	Tolbert		6,063	4,197	-	7,895	5.3	5,795	4,009	-	7,548	5.0	269	0.2	4
ER visits (Resp)	Tolbert	CO	5,397	3,314	-	7,436	4.7	5,157	3,165	-	7,109	4.5	240	0.2	4
ER visits (Resp)	Tolbert	NO2	4,879	2,579	-	7,127	4.2	4,662	2,463	-	6,813	4.0	218	0.2	4
ER visits (Resp)	Tolbert	PM10	3,830	1,352	-	6,248	3.3	3,658	1,290	-	5,972	3.2	172	0.1	4
ER visits (Resp)	Tolbert	PM10, NO2	3,697	1,213	-	6,121	3.2	3,532	1,158	-	5,850	3.1	166	0.1	4
ER visits (Resp)	Darrow	Darrow	3,276	1,991	-	4,545	2.8	3,129	1,901	-	4,342	2.7	147	0.1	4
ER visits (Resp)	Strickland	dist lag 0-7	7,056	5,026	-	8,951	18.0	6,768	4,813	-	8,599	17.3	287	0.7	4
ER visits (Resp)	Strickland	avg day lag 0-2	4,171	2,281	-	5,953	10.7	3,992	2,180	-	5,706	10.2	179	0.5	4
New York															
ER visits (asthma)	Ito		12,945	8,828	-	16,777	16.9	12,152	8,266	-	15,787	15.9	793	1.0	6
ER visits (asthma)	Ito	PM2.5	10,115	5,439	-	14,443	13.2	9,479	5,082	-	13,571	12.4	636	0.8	6
ER visits (asthma)	Ito	NO2	8,350	3,750	-	12,620	10.9	7,816	3,500	-	11,844	10.2	534	0.7	6
ER visits (asthma)	Ito	CO	13,677	9,682	-	17,399	17.9	12,844	9,071	-	16,379	16.8	832	1.1	6
ER visits (asthma)	Ito	SO2	10,441	6,068	-	14,505	13.7	9,786	5,672	-	13,630	12.8	655	0.9	6

Table 7-22 Short-Term Ozone Exposure-Related Morbidity (Hospital Admissions – 2007 simulation year)

Urban study area (endpoint)	Study author	Effect estimator differentiators	Current conditions simualtion (2007)					Current standard simulation (2007)					Delta (risk reduction)		
			point estimate	95th Confidence 2.5		97.5	% of baseline	point estimate	95th Confidence 2.5		97.5	% of baseline	point estimate	% of baseline	% reduction in ozone-related morbidity
New York															
HA (chronic lung dis	Lin		133	78	-	188	2.2	115	67	-	162	1.9	18	0.3	14
HA (asthma)	Silverman		694	49	-	1,192	19.0	628	43	-	1,094	17.3	66	1.8	10
HA (asthma)	Silverman	PM2.5	508	-175	-	1,036	13.8	457	-155	-	946	12.4	51	1.3	10
Detroit															
HA (respiratory)	Katsouyanni	1hr max, penalized spines	55	-13	-	121	1.8	49	-11	-	108	1.6	6	0.2	11
HA (respiratory)	Katsouyanni	1hr max, natural spines	53	-16	-	120	1.8	47	-14	-	107	1.6	6	0.2	11
HA (respiratory)	Medina-Ramon	8hr mean													
Atlanta, GA			35	10	-	60	3.2	26	7	-	45	2.4	9	0.8	25%
Baltimore, MD			21	6	-	36	2.5	16	5	-	28	2.0	5	0.6	23%
Boston, MA			30	8	-	51	2.3	24	7	-	41	1.8	6	0.5	20%
Cleveland, OH			15	4	-	25	2.3	11	3	-	20	1.8	3	0.5	21%
Denver, CO			3	1	-	5	2.6	2	1	-	4	2.1	1	0.5	20%
Detroit, MI			21	6	-	36	2.5	17	5	-	29	2.1	4	0.5	19%
Houston, TX			13	4	-	23	1.8	10	3	-	17	1.3	4	0.5	27%
Los Angeles, CA			55	15	-	93	2.9	37	10	-	64	2.0	17	0.9	31%
New York, NY			61	17	-	105	2.3	47	13	-	81	1.8	14	0.5	23%
Philadelphia, PA			13	4	-	21	2.6	9	3	-	16	1.9	3	0.6	25%
Sacramento, CA			7	2	-	11	2.7	5	1	-	8	2.0	2	0.7	26%
St. Louis, MO			23	6	-	39	3.0	18	5	-	31	2.4	4	0.6	19%
LA															
HA (respiratory)	Linn	1hr max, penalized spines	106	-137	-	344	0.7	62	-80	-	202	0.4	44	0.3	42

Table 7-23 Short-Term Ozone Exposure-Related Morbidity (Hospital Admissions – 2009 simulation year)

Urban study area (endpoint)	Study author	Effect estimator differentiators	Current conditions simualtion (2007)					Current standard simulation (2007)					Delta (risk reduction)		
			point estimate	95th Confidence 2.5		97.5	% of baseline	point estimate	95th Confidence 2.5		97.5	% of baseline	point estimate	% of baseline	% reduction in ozone-related morbidity
New York															
HA (chronic lung disease)	Lin		192	112	-	271	3.2	179	104	-	252	3.0	13	0	7
HA (asthma)	Silverman		876	62	-	1,482	23.7	825	58	-	1,409	22.4	51	1	6
HA (asthma)	Silverman	PM2.5	644	-226	-	1,294	17.2	605	-210	-	1,227	16.2	39	1	6
Detroit															
HA (respiratory)	Katsouyanni	1hr max, penalized spines	75	-18	-	165	2.6	75	-18	-	165	2.6	0	0	0
HA (respiratory)	Katsouyanni	1hr max, natural spines	72	-22	-	163	2.5	72	-22	-	163	2.5	0	0	0
HA (respiratory)	Medina-Ramon	8hrmax													
Atlanta, GA			31	9	-	52	2.6	29	8	-	50	2.5	1	0.1	5%
Baltimore, MD			22	6	-	37	2.5	20	5	-	33	2.3	2	0.2	9%
Boston, MA			26	7	-	44	1.9	25	7	-	43	1.9	1	0.1	3%
Cleveland, OH			13	4	-	23	2.2	13	4	-	22	2.1	0.4	0.1	3%
Denver, CO			3	1	-	5	2.6	3	1	-	5	2.5	0.1	0.1	3%
Detroit, MI			17	5	-	29	2.1	17	5	-	29	2.1	0.0	0.0	0%
Houston, TX			16	4	-	27	2.0	15	4	-	25	1.8	1	0.1	7%
Los Angeles, CA			55	15	-	93	2.8	44	12	-	76	2.3	10	0.5	19%
New York, NY			57	16	-	98	2.1	53	15	-	91	2.0	4	0.2	7%
Philadelphia, PA			10	3	-	17	2.2	10	3	-	16	2.0	1	0.1	6%
Sacramento, CA			7	2	-	12	2.8	6	2	-	10	2.4	1	0.4	15%
St. Louis, MO			18	5	-	31	2.3	18	5	-	30	2.3	0	0.1	3%
LA															
HA (respiratory)	Linn	1hr max, penalized spines	272	-358	-	876	1.7	255	-334	-	822	1.6	17	0	6

Table 7-24 Short-Term Ozone Exposure-Related Morbidity (Asthma Exacerbations)

Urban study area (endpoint)	Study author	Effect estimator differentiators	Current conditions simualtion (2007)					Current standard simulation (2007)					Delta (risk reduction)		
			point estimate	95th Confidence			% of baseline	point estimate	95th Confidence			% of baseline	point estimate	% of baseline	% reduction in ozone-related morbidity
				2.5		97.5			2.5		97.5				
2007 Simulation															
Boston MA															
asthma exacer (Chest tightness)	Gent	1hr max, lag 1	28,639	14,989	-	40,322	22.0	26,401	13,720	-	37,412	20.2	2,238	1.7	8
asthma exacer (shortness of breath)	Gent	1hr max, lag 1	20,035	2,485	-	35,259	12.2	18,348	2,260	-	32,495	11.2	1,687	1.0	8
asthma exacer (Chest tightness)	Gent	8hr max, lag 1	20,493	6,722	-	32,412	15.7	18,932	6,172	-	30,114	14.5	1,562	1.2	8
asthma exacer (shortness of breath)	Gent	8hr max, lag 1	23,700	4,749	-	39,922	14.4	21,878	4,354	-	37,082	13.4	1,822	1.1	8
Asthma exacer (chest tightness)	Gent	1hr max PM2.5 lag 0	28,949	13,374	-	42,008	22.1	26,691	12,231	-	39,014	20.4	2,258	1.7	8
asthma exacer (Chest tightness)	Gent	1hr max PM2.5 lag 1	26,701	10,632	-	40,156	20.4	24,589	9,711	-	37,255	18.8	2,112	1.6	8
asthma exacer (wheeze)	Gent	1hr max, PM2.5, lag 0	53,682	19,682	-	82,795	17.6	49,333	17,956	-	76,598	16.2	4,350	1.4	8
2009 Simulation															
Boston MA															
asthma exacer (Chest tightness)	Gent	1hr max	24,387	12,553	-	34,861	18.5	23,588	12,124	-	33,767	17.9	799	0.6	3
asthma exacer (shortness of breath)	Gent	1hr max	16,799	2,050	-	30,007	10.2	16,226	1,978	-	29,021	9.8	573	0.3	3
asthma exacer (Chest tightness)	Gent	8hr max	18,340	5,943	-	29,329	13.9	17,726	5,736	-	28,389	13.4	614	0.5	3
asthma exacer (shortness of breath)	Gent	8hr max	21,180	4,188	-	36,107	12.8	20,467	4,040	-	34,947	12.4	713	0.4	3
Asthma exacer (chest tightness)	Gent	1hr max PM2.5 lag 0	24,661	11,179	-	36,402	18.7	23,853	10,795	-	35,267	18.1	807	0.6	3
asthma exacer (Chest tightness)	Gent	1hr max PM2.5 lag 1	22,682	8,859	-	34,710	17.2	21,934	8,552	-	33,620	16.6	748	0.6	3
asthma exacer (wheeze)	Gent	1hr max, PM2.5	45,379	16,356	-	71,096	14.7	43,862	15,786	-	68,819	14.2	1,517	0.5	3

The presentation of key observations drawn from review of the risk estimates is divided into two sections including: the assessment of health risks associated with recent conditions (section 7.5.1) and with just meeting the current and alternative standards (sections 7.5.2). As noted earlier, for the first draft REA we are only presenting results for the simulation of just meeting the current standard. Risks under simulated just meeting alternative standards will be presented in the second draft analysis. The presentation of key observations (for both recent conditions and the simulated just meeting the suite of current O_3 standards) is further separated into those associated with mortality estimates and morbidity estimates.

7.5.1 Assessment of Health Risk Associated with Recent conditions

The assessment of risk for the recent conditions scenario for the 12 urban study areas (for short-term exposure-related mortality) focuses on characterizing absolute risk using two types of risk estimates (a) risk modeled down to zero O_3, which reflects consideration for the full range of exposure and (b) risk modeled down to the LML, which represents a higher confidence estimate with the caveat that it excludes exposures below the LML (and is therefore likely biased low). For short-term exposure-related morbidity endpoints, we only included estimates of risk down to the LML. Estimates of the reduction in risk (deltas) are not relevant in evaluating the recent conditions scenario, but are an important part of the analysis completed for the simulation of just meeting the current standard level (presented in the next section).

Short-term O_3 exposure-related mortality

- Higher confidence estimates of O_3-related all-cause mortality (modeled down to LML) range 0.4 to 3.5% of total mortality across the 12 urban study areas (for 2007) using Zanobetti and Schwartz (2008b) C-R functions. Estimates of O_3-related all-cause mortality (modeled down to zero O_3) range from 0.5 to 4.9% of total mortality (for 2007) using Zanobetti and Schwartz (2008b) C-R functions (see Table 7-16). This translates into from 10 to 710 O_3-related deaths across the 12 urban study areas when exposure is modeled down to the LML and from 20 to 930 deaths when exposure is modeled down to zero O_3. Of particular note regarding the mortality estimates based on the Zanobetti and Schwartz (2008b) C-R functions are the higher risk estimates generated for Detroit and New York (see Table 7-16 and 7-17). In both cases, these higher estimates reflect the use of effect estimates which are substantially larger than estimates used for other urban study areas. As part of the second draft REA, we will explore this observation (regarding higher risk related to notably higher effect estimates) in greater detail (see section 7.7).

- Higher confidence estimates of O_3-related all-cause mortality (modeled down to LML) range from 1.0 to 2.5% of total mortality across the 12 urban study areas (for 2007) using Bell et al., (2004) C-R functions. Estimates of O_3-related all-cause mortality (modeled down to zero O_3) range from 1.5 to 3.7% of total mortality (for

2007) using Bell et al., (2004) C-R functions (see Table 7-16). This translates into from 30 to 730 O_3-related deaths across the 12 urban study areas when exposure is modeled down to the LML and from 40 to 950 deaths when exposure is modeled down to zero O_3.

- While we have a high degree of overall confidence in estimates generated using C-R functions based on Zanobetti and Schwartz (2008b) and Bell et al (2004), resulting in both sets of risk estimates being considered core estimates, we would note that Zanobetti and Schwartz (2008b)-based estimates, only included exposures associated with June-August and therefore may bias estimates of O_3-related deaths low by not considering O_3 exposure occurring during the rest of the O_3 season defined for each urban study area. By contrast, Bell et al (2004)-based C-R functions provide coverage for O_3 exposure occurring across the full O_3 season defined for each urban study area. This potential low-bias in the Zanobetti and Schwartz (2008b)-based risk estimates effects *incidence count* metrics.

- For a number of the urban study areas, confidence intervals (but not point estimates) for short-term all-cause mortality (using C-R functions derived both from Zanobetti and Schwartz 2008b and Bell et al., 2004) include values that fall below zero (see Tables 7-11 through 7-14). Population incidence estimates with negative lower-confidence bounds do not imply that additional exposure to O_3 has a beneficial effect, but only that the estimated O_3 effect estimate in the C-R function was not statistically significantly different from zero.

- Cause-specific mortality could only be evaluated using C-R functions based on Zanobetti and Schwartz (2008b) (Bayes-shrunken city-specific estimates for cause specific mortality were not available for Bell et al, 2004). For 2007, estimates of cardiovascular-related mortality incidence (associated with O_3 exposure) were substantially larger than estimates of respiratory-related mortality incidence (see Table 7-15). The sum of cardiovascular and respiratory does not equal total mortality for most of the urban study areas and in some cases can be substantially lower than total mortality (see Table 7-15). We may explore potential explanations for this as part of the second draft REA.

- All-cause mortality estimates derived using C-R functions from both Zanobetti and Schwartz (2008b) and Bell et al (2004) are driven largely by days with total O_3 levels falling in the range of 35 to 70 ppb, with a substantial portion of the mortality estimate associated with days having O_3 levels above 60 ppb (for 2007 - see Tables 7-7 and 7-9, respectively).[13]

- Generally, all-cause mortality risks decrease somewhat for simulation year 2009 compared with estimates generated for 2007, reflecting the lower measured O_3 levels

[13] Characterization of ozone level ranges in Tables 7-7 through 7-10 is based on the air metric used by each C-R function. The Zanobetti and Schwartz (2008b) based C-R functions uses daily 8hr mean daily values for the composite monitor in a given urban area for the simulation period June through August. The Bell et al (2004) based C-R functions uses 8hr max daily values for the composite monitor in a given urban area for the ozone season specific to that urban area.

in the later simulation year (with the exception of Atlanta, Baltimore, Los Angeles, Sacramento and Houston, depending on the C-R function used, which did not have lower O_3 levels in 2009) - compare LML-based estimates presented in Table 7-16 with estimates in 7-17 and/or compare estimates in Table 7-18 with those in Table 7-19.

Short-term O_3 exposure-related morbidity

- Estimates of O_3- attributable ER visits (respiratory symptoms) for 2007 in Atlanta (based on modeling exposure down to the LML) range from roughly 2.4 to 15.6% of total baseline incidence which translates into from 3,100 to 6,000 visits depending on the model formulation (i.e., epidemiological study providing the C-R function and the treatment of lag and copollutants) (see Table 7-21). Estimates of O_3- attributable ER visits (for asthma) for 2007 in New York range form roughly 8.6 to 14.2% of total baseline which translates into 6,600 to 10,800 visits again depending on the treatment of copollutants in the model (see Table 7-21).

- Estimates of ER visits in both urban study areas are modestly larger for 2009, reflecting higher O_3 levels (for the O_3 metrics involved in modeling these endpoints) (see Table 7-21).

- Estimates of O_3- attributable HA (for asthma) in New York in 2007 (based on modeling risk down to LML) range form roughly 13.8 to 19% of baseline incidence which translates into roughly 500 to 700 admissions depending whether $PM_{2.5}$ is included in the model (see Table 7-22). Estimates of HA (for chronic lung disease) in New York in 2007 are approximately 2.2% of baseline which translates into 130 admissions (see Table 7-22). Estimates of O_3- attributable HA (respiratory symptoms) across the 12 urban study areas range from 0.7 to 3.2% of baseline, which translates into 3 to 110 admissions (see Table 7-22).

- Estimates of HA visits for simulation year 2009 are generally marginally lower across most cities, reflecting lower measured O_3 levels (with the notable exception of New York, which had notably higher estimates of HA for asthma in 2009, reflecting higher O_3 levels in 2009 for the metric used in modeling risk) (compare estimates in Table 7-23 to those in Table 7-22).

- Estimates of O_3- attributable asthma exacerbations for Boston in 2007 (based on modeling risk down to LML) range from roughly 12.2 to 22.1% of baseline incidence which translates into 20,000 to 29,000 events (for chest tightness or shortness of breath). This range reflects differences in model specification (e.g., lag structure and peak O_3 metric used). Estimates of O_3- attributable asthma exacerbation (wheeze) was 17.6% of baseline which translates into 55,000 events (see Table 7-24). These estimates were somewhat lower in 2009 (see Table 7-24).

- While estimates for both ER visits and asthma exacerbations included 95th percentile confidence intervals that did not include negative values, several of the analyses involving HA did include negative lower estimates for the 2.5th percentile values (i.e.,

the lower bound of the 95th percentile intervals for the incidence estimates) (see Table 7-22 and 7-23). The negative lower bound values for the subset of HA estimates likely reflects, at least in part, the considerably smaller sample size associated with modeling for this endpoint compared with other HA-related endpoints and both ER and asthma exacerbation endpoints included in this analysis. And, as was discussed above in relation to short-term exposure-related mortality, negative values for lower bound statistics does not imply that O_3 is beneficial, but rather speaks to the lower sample size, as discussed here.

7.5.2 Assessment of Health Risk Associated with Simulating Meeting the Current Suite of O_3 Standards

The analysis of risk after simulating just meeting the current standard includes both (a) assessment of absolute risk remaining and (b) the risk reduction (delta) associated with a comparison of O_3 levels for recent conditions with O_3 levels after simulating just meeting the current primary O_3 standard. In both cases, we generated two types of risk estimates including an assessment of risk based on modeling exposure down to zero O_3 and a higher confidence estimate based on modeling risk down to the surrogate LML. As noted earlier in section 7.1.2.1, constraining the analysis to only consider exposures above the LML did not have a substantial impact on delta (risk reduction) estimates, since most of the daily reductions in O_3 occurred at levels well above the applicable LML. Our discussion of risk estimates presented below focuses primarily on the level of O_3- attributable risk remaining after simulation of meeting the current standard level.

<u>Short-term O_3 exposure-related mortality</u>

- Higher confidence estimates of O_3-related all-cause mortality (modeled down to LML) range 0.3 to 3.1% of total mortality across the 12 urban study areas (for 2007) using Zanobetti and Schwartz (2008b) C-R functions. Estimates of total O_3-related all-cause mortality (modeled down to zero O_3) range from 0.5 to 4.6% of total mortality (for 2007) using Zanobetti and Schwartz (2008b) C-R functions (see Table 7-16). This translates into from 10 to 630 O_3-related deaths across the 12 urban study areas when exposure is modeled down to the LML and from 20 to 850 deaths when exposure is modeled down to zero O_3. As with risk estimated for recent conditions, the mortality estimates generated for Detroit and New York are notably higher than those for the remaining 10 study areas (see Table 7-16 and 7-17). As stated earlier, these higher estimates reflect the use of effect estimates which are substantially larger than estimates used for other urban study areas. As part of the second draft REA, we will explore this issue in greater detail (see section 7.7).

- Higher confidence estimates of O_3-related all-cause mortality (modeled down to LML) range 0.9 to 2.0% of total mortality across the 12 urban study areas (for 2007) using Bell et al., (2004) C-R functions. Estimates of total O_3-related all-cause mortality (modeled down to zero O_3) range from 1.3 to 3.2% of total mortality (for

2007) using Bell et al., (2004) C-R functions (see Table 7-16). This translates into from 30 to 590 O_3-related deaths across the 12 urban study areas when exposure is modeled down to the LML and from 3 to 830 deaths when exposure is modeled down to zero O_3.

- Delta risk reductions for all-cause mortality associated with the simulation of the current standard level (for 2007 using Zanobetti and Schwartz (2008b)-based C-R functions) range roughly from 1 to 80 deaths averted across the 12 urban study areas whether we model risk down to the LML, or down to zero. As noted above, this risk metric is fairly invariant to consideration of the LML, since most reductions in O_3 occur at levels well above the LML. If we use C-R functions based on Bell et al., (2004), then delta risk ranges from 2 to 160 deaths averted across the 12 urban study areas.

- As noted earlier, estimates generated using C-R functions based on Zanobetti and Schwartz (2008b) may be biased low since they only considered exposures between June and August. By contrast, Bell et al (2004)-based C-R functions model risk for the entire ozone season specific to each urban study area. This potential low-bias in the Zanobetti and Schwartz (2008b)-based risk estimates effects *incidence count* metrics.

- As noted earlier, population incidence estimates with negative lower-confidence bounds do not imply that additional exposure to O_3 has a beneficial effect, but only that the estimated O_3 effect estimate in the C-R function was not statistically significantly different from zero.

- As with risk estimates generated for the recent conditions scenario, estimates of O_3-attributable cardiovascular-related mortality incidence were substantially larger than estimates of respiratory-related mortality incidence (see Table 7-15).

- Even after simulation of urban study areas meeting the current ozone standard, all-cause mortality estimates derived using C-R functions from both Zanobetti and Schwartz (2008b) and Bell et al (2004) continue to be driven largely by days with total O_3 levels falling in the range of 35 to 70 ppb, with a substantial portion of the mortality estimate associated with days having O_3 levels above 60 ppb (for 2007 - see Tables 7-7 and 7-9, respectively).

- Generally, O_3-attributable all-cause mortality risks continue to be lower for the 2009 simulation year as compared with the 2007 simulation year (with the exception of Atlanta, Baltimore, Los Angeles, Sacramento and Houston, depending on the C-R function used, which did not have lower O_3 levels in 2009) - compare LML-based estimates presented in Table 7-16 with estimates in 7-17 and/or compare estimates in Table 7-18 with those in Table 7-19.

Short-term O_3 exposure-related morbidity

- Estimates of O_3- attributable ER visits (respiratory symptoms) for 2007 in Atlanta (based on modeling exposure down to the LML) range from roughly 2.0 to 12.8% of total baseline incidence which translates into from 2,200 to 4,900 visits depending on the model formulation (i.e., epidemiological study providing the C-R function and the treatment of lag and copollutants) (see Table 7-21). Estimates of O_3- attributable ER visits (for asthma) for 2007 in New York range form roughly 7.7 to 12.8% of total baseline which translates into 5,900 to 9,700 visits again depending on the treatment of copollutants in the model (see Table 7-21).

- Estimates of ER visits in both urban study areas are larger for 2009, reflecting higher O_3 levels (for the O_3 metrics involved in modeling these endpoints) (see Table 7-21).

- Estimates of O_3- attributable HA (for asthma) in New York in 2007 (when modeling risk down to LML) range form roughly 12.4 to 17.3% of baseline incidence which translates into roughly 500 to 600 admissions depending whether $PM_{2.5}$ is included in the model (see Table 7-22). Estimates of HA (for chronic lung disease) in New York in 2007 are approximately 1.9% of baseline which translates into 120 admissions (see Table 7-22). Estimates of O_3- attributable HA (respiratory symptoms) across the 12 urban study areas range from 0.4 to 2.4% of baseline, which translates into 2 to 60 admissions (see Table 7-22).

- Estimates of O_3- attributable asthma exacerbations for Boston in 2007 (based on modeling risk down to LML) range from roughly 11.2 to 20.4% of baseline incidence which translates into 18,000 to 27,000 events (for chest tightness or shortness of breath). This range reflects differences in model specification (e.g., lag structure and peak O_3 metric used). Estimates of O_3- attributable asthma exacerbation (wheeze) was 16.2% of baseline which translates into 49,000 events (see Table 7-24). These estimates were somewhat lower in 2009 (see Table 7-24).

- Risk reductions (comparing recent conditions to meeting the current standard) for ER visits (respiratory) in Atlanta (2007) range from 500 to 1,100 visits averted (see Table 7-21). Delta risk for ER visits (asthma) in New York (2007) range from 700 to 1,100 visits averted (see Table 7-21). Risk reductions for HA (asthma) in New York (2007) range from 50 to 70 admissions averted (see Table 7-22). Risk reduction for HA (chronic lung disease) in New York is estimated at 18 admissions averted. Risk reductions for HA (respiratory) across the 12 urban study areas range from 1 to 40 admissions averted (see Table 7-22). Risk reduction for asthma exacerbations (shortness of breath or chest tightness) in Boston (2007) ranges from 1,600 to 2,300 cases averted (see Table 7-24). We estimate that in 2007 in Boston, we would see 4,400 fewer asthma exacerbations (wheeze) the city was in attainment. All risk reduction estimates summarized in this bullet reflect modeling of risk down to the LML.

- Estimates of HA visits for simulation year 2009 are generally marginally lower across most cities, reflecting lower measured O_3 levels (with the notable exception of New

York, which had notably higher estimates of HA for asthma in 2009, reflecting higher O_3 levels in 2009 for the metric used in modeling risk) (compare estimates in Table 7-23 to those in Table 7-22).

- As noted earlier, negative lower bound values for the subset of HA estimates likely reflects, at least in part, the considerably smaller sample size associated with modeling for this endpoint compared with other HA-related endpoints as well as both ER and asthma exacerbation endpoints included in this analysis. And, as was discussed above in relation to short-term exposure-related mortality, negative values for lower bound statistics does not imply that O_3 is beneficial, but rather reflect the lower sample size.

7.6 KEY OBSERVATIONS DRAWN FROM THE URBAN CASE STUDY ANALYSIS OF O_3-RELATED RISK

This chapter provides key observations regarding: (a) overall confidence in the analysis reflecting both the design of the risk assessment and the degree to which variability and uncertainty have been addressed (section 7.6.1) and (b) risk estimates generated for both the recent conditions and just meeting the current standard level (including the distribution of risks and pattern of risk reduction across the 12 urban study areas and two simulation years evaluated) (section 7.6.2).

7.6.1 Overall Confidence in the Risk Assessment and Risk Estimates

Based on consideration for observations listed as bullets below, EPA staff preliminarily concludes that there is a reasonable degree of confidence in the core risk estimates generated for mortality associated with short-term O_3 exposure. However, we differentiate between the estimates of risk based on modeling exposure down to zero O_3 and those based on modeling risk down to the LML. Generally, we have higher confidence in the estimates of risk based on modeling risk down to the LML, since these reflect the O_3 levels used in fitting the C-R functions underlying the risk estimates. However, the LML estimates are likely low-biased given that they exclude exposures below the LML. In this context, the estimates of risk down to zero O_3 may be particularly useful in gaining perspective on the potential magnitude of this excluded risk (i.e., the risk associated with exposures below the LML).

Overall confidence in estimating mortality risk will likely be increased further with the inclusion of a sensitivity analysis in the second draft REA, exploring the potential impact of design elements on these risk estimates. Confidence in risk estimates generated for all of the health endpoints will be further increased if we can obtain the actual LMLs associated with the studies underlying the C-R functions, since that will allow us to estimate risk with consideration for the actual range of data used in fitting the C-R functions (and not a surrogate).

Confidence in our characterization of short-term exposure-related morbidity risk is somewhat lower (but still reasonable) given that morbidity effects are only evaluated (for most endpoints) for a subset of urban study areas and because we do not have multiple C-R functions from multiple studies for the same endpoint. In addition, most of the epidemiological studies covering respiratory morbidity endpoints are city-specific and it would be preferable to also have Bayes-shrunken estimates which combine both a local and broader-scale regional or national signal in modeling risk for each urban area.

Key observations addressing overall confidence in the analysis include:

- A deliberative process was used in specifying each of the analytical elements comprising the risk model. This process included first identifying specific goals for the analysis, and then designing the analysis to meeting those goals, given available information and methods. Specific analytical elements reflected in the design include: selection of urban study areas, characterization of ambient air O_3 levels, selection of health endpoints to model and selection of epidemiological studies (and specification of C-R functions) (see sections 7.1.1 and 7.3).

- Modeling of short-term exposure-related mortality (the key endpoint in the analysis) utilized Bayes-adjusted city-specific effect estimates (see section 7.1.1 and section 7.3.2). These effect estimates are considered to have increased overall confidence since they combine elements of the local city-specific signal with a broader scale (national) signal.

- Review of available literature (as specified in the O_3 ISA, U.S. EPA. 2012), resulted in a decision not to incorporate a true (no effect) threshold into our risk modeling. Conversely, the literature supports a log-linear, no-threshold relationship down to concentrations at the lower end of the range of ambient O_3 concentrations. To explore the impact of focusing risk modeling on ranges of increased confidence, we generated risk estimates reflecting the range of exposures used in deriving the C-R functions underlying the risk estimates (see section 7.1.1). However, we also included estimates of risk reflecting the full range of exposures down to zero O_3. Together, these two types of risk estimates inform consideration of uncertainty related to application of the C-R function at low ozone levels.

- Evaluation of the degree to which key sources of variability impacting O_3 risk were incorporated into the design of the analysis (see section 7.4.1). Some of the key sources considered in the design include: heterogeneity in effect O_3 across cities, intra-urban variability in O_3 levels, variability in the pattern of O_3 reductions within urban areas when simulating just meeting the current standard, inter-urban and intra-urban variability in copollutants levels and their role as potential confounders, variability in demographic and SES-related factors, and variability in baseline incidence rates.

- Application of a strategy based on the WHO's 4-tiered approach for characterizing uncertainty to evaluate the potential impact of uncertainty on risk estimates (see

section 7.4.2). This approach involves both a quantitative sensitivity analysis to evaluate the potential impact of specific design elements on risk estimates and completion of a qualitative analysis to provide additional coverage for potential sources of uncertainty. For the first draft analysis, we completed the qualitative analysis, however, we did not complete the sensitivity analysis (that is planned for the second draft analysis). The qualitative analysis of uncertainty suggested that the statistical fit and shape of C-R functions together with the use of surrogate LMLs to define ranges of increased confidence in estimating risk could have a medium impact on risk estimates. Other factors (e.g., characterization of ambient air O_3 levels, addressing copollutants in the context of deriving C-R functions) could have a low-medium impact (see section 7.4.2).

7.6.2 Risk Estimates Generated for Both the Recent Conditions and Simulation of Meeting the Current Standard

Key observations regarding risk estimates generated for both the recent conditions and simulating just meeting the current standard level are presented below:

- Estimates of short-term exposure-related all-cause mortality attributable to O_3 under recent conditions vary widely across urban study areas, reflecting differences both in ambient O_3 levels and population counts, as well as differences in effect estimates. Risk based on modeling exposure down to the LML (for simulation year 2007) is estimated to range from 0.4 to 3.5% of total baseline mortality across the 12 urban study areas which translate into from roughly 10 to 710 deaths across the 12 urban study areas. When risk is modeled for ozone exposures down to zero O_3 (i.e., considering the full range of potential exposures), then O_3-related risk (again for 2007) ranges from 0.5 to 4.9% of total mortality, which translates into from roughly 20 and 930 deaths.

- Estimates of O_3-attributable all-cause mortality under recent conditions in 2007 are driven largely by days with O_3 levels falling in the range of 35 to 70ppb (for the metrics involved in risk modeling – 8hr max and 8hr averages). A substantial portion of the mortality risk is associated with days having O_3 levels even higher, above 60 ppb. This observation accounts for the notable magnitude of risk reduction seen with simulation of just meeting the current standard (see below).

- For most of the study areas, estimates of short-term exposure-related all-cause mortality attributable to O_3 are somewhat (but not substantially) smaller for simulation year 2009 as compared with simulation year 2007. This reflects primarily the lower O_3 levels seen in 2009.

- Estimates of short-term exposure-related morbidity attributable to O_3 under recent conditions for 2007 include: (a) ER visits (for respiratory symptoms in Atlanta) range from roughly 2.4 to 15.6% of total baseline incidence which translates into from 3,100 to 6,000, (b) ER visits (for asthma in New York City) range form roughly 8.6 to 14.2% of total baseline which translates into 6,600 to 10,800, (c) HA (for asthma in New York City) range form roughly 13.8 to 19% of baseline incidence which translates into roughly 500 to 700 admissions, (d) HA (for chronic lung disease

New York City) are roughly 2.2% of baseline which translates into 130 admissions, (e) HA (respiratory symptoms across the 12 urban study areas) range from 0.7 to 3.2% of baseline, which translates into 3 to 110 admissions, (f) asthma exacerbations (chest tightness or shortness of breath for Boston) range from roughly 12.2 to 22.1% of baseline incidence which translates into 20,000 to 29,000 events (for chest tightness or shortness of breath) and (g) asthma exacerbation (wheeze in Boston) was 17.6% of baseline which translates into 55,000 events. All these estimates reflect modeling exposure down to the applicable LML value (and not down to zero O_3).

- Estimates of short-term exposure-related all-cause mortality attributable to O_3 after simulating meeting the current standard vary widely across urban study areas, reflecting differences both in ambient O_3 levels and population counts, as well as differences in effect estimates. Risk based on modeling exposure down to the LML (for simulation year 2007) is estimated to range from 0.3 to 3.1% of total baseline mortality across the 12 urban study areas which translate into from roughly 10 to 630 deaths across the 12 urban study areas. If we model risk all the way down to zero O_3 (i.e., considering the full range of potential exposures), then O_3-related risk (again for 2007) ranges from 0.5 to 4.6% of total mortality, which translates into from roughly 20 and 850 deaths.

- Estimates of O_3-attributable all-cause mortality after simulating meeting the current standard in 2007 are driven largely by days with O_3 levels falling in the range of 35 to 70ppb (for the metrics involved in risk modeling – 8hr max and 8hr averages). A substantial portion of the mortality risk continues to be associated with days having O_3 levels even higher, above 60 ppb. This observation accounts for the notable magnitude of risk reduction seen with simulation of just meeting the current standard (see below).

- For most of the study areas, estimates of short-term exposure-related all-cause mortality attributable to O_3 are somewhat (but not substantially) smaller for simulation year 2009 as compared with simulation year 2007. This reflects primarily the lower O_3 levels seen in 2009.

- Estimates of short-term exposure-related morbidity attributable to O_3 after simulating meeting the current standard for 2007 include: (a) ER visits (for respiratory symptoms in Atlanta) range from roughly 2.0 to 12.8% of total baseline incidence which translates into from 2,200 to 4,900, (b) ER visits (for asthma in New York City) range form roughly 7.7 to 12.8% of total baseline which translates into 5,900 to 9,700, (c) HA (for asthma in New York City) range form roughly 12.4 to 17.3% of baseline incidence which translates into roughly 500 to 600 admissions, (d) HA (for chronic lung disease New York City) are roughly 1.9% of baseline which translates into 120 admissions, (e) HA (respiratory symptoms across the 12 urban study areas) range from 0.4 to 2.4% of baseline, which translates into 2 to 60 admissions, (f) asthma exacerbations (chest tightness or shortness of breath for Boston) range from roughly 11.2 to 20.4% of baseline incidence which translates into 18,000 to 27,000 events (for chest tightness or shortness of breath) and (g) asthma exacerbation (wheeze in Boston) was 16.2% of baseline which translates into 49,000 events. All these

estimates reflect modeling exposure down to the applicable LML value (and not down to zero O_3).

- Under simulation of just meeting the current standard, we see a shift in the daily metric profile for O_3, as would be expected given application of the quadratic rollback method in predicting reductions in O_3. However, we still see that all-cause mortality attributable to O_3 is driven by days in the higher O_3 ranges (i.e., 30 to 70ppb, with a significant portion associated with days above 60 ppb).

- Generally, for most of the urban study areas, reductions in all-cause mortality risk associated with simulated just meeting the current standard is significantly lower for simulation year 2009 compared with estimates generated for 2007, reflecting the lower measured O_3 levels in the later simulation year.

- Risk reductions for all-cause mortality associated with the simulation of the current standard level (for 2007) range roughly from 1 to 160 deaths averted across the 12 urban study areas whether we model risk down to the LML, or down to zero. Risk reductions for morbidity endpoints are: (a) ER visits (respiratory) in Atlanta (2007) range from 500 to 1,100 visits averted, (b) ER visits (asthma) in New York (2007) range from 700 to 1,100 visits averted, (c) HA (asthma) in New York (2007) ranges from 50 to 70 admissions averted, (d) HA (chronic lung disease) in New York is estimated at 18 admissions averted, (e) HA (respiratory) across the 12 urban study areas ranges from 1 to 40 admissions averted, (f) asthma exacerbations (shortness of breath or chest tightness) in Boston (2007) ranges from 1,600 to 2,300 cases averted, and (g) in Boston, we estimate 4,400 fewer asthma exacerbations (wheeze). All risk reduction estimates summarized in this bullet reflect modeling of risk down to the LML.

7.7 POTENTIAL REFINEMENTTS FOR SECOND DRAFT RISK ASSESSMENT

This section describes potential refinements for the second draft REA which include: (a) sensitivity analyses intended to enhance our understanding of the impact of design elements on core risk assessments, (c) additional refinements to the core sets of risk estimates presented in the first draft REA, and (c) treatment of both long-term exposure-related mortality and morbidity endpoints. Each of theses topics is discussed separately.

7.7.1 Potential sensitivity analyses

As noted earlier in section 7.1.1, we did not complete a comprehensive set of sensitivity analyses for the first draft REA due to emphasis being placed on generating a set of core risk estimates. The following set of sensitivity analyses will be considered for the second Draft risk assessment, in order to gain further insights into the potential impact of modeling design choices on risk estimates.

- *Interpolation of missing air quality data*: For the first draft risk assessment, we did not fill in any missing monitoring data in generating the composite monitor

distributions (see section 7.2.1). For the second draft REA, we may explore this issue of interpolating missing measurement data as part of the sensitivity analysis. The goal would be to determine whether incorporating interpolation of missing data has a significant impact on risk estimates. The sensitivity analysis could consider (a) interpolation methods used in key epidemiological studies supporting the C-R functions used in the risk assessment (to the extent that those studies used interpolation) and/or (b) interpolation methods used in the first draft exposure analysis (see section 5.5.6)..

- *Short-term exposure-related mortality*: Because we believe that greater confidence is associated with the use of Bayes-adjusted city-specific effect estimates, we also believe that ideally, sensitivity analyses (examining different model design options) should also be based on Bayes-adjusted city-specific effect estimates. This would necessitate that, if we are to conduct sensitivity analyses for this endpoint group, we obtain Bayes-adjusted city-specific effect estimates reflecting different design options (e.g., lag structures, copollutants models). While, we would consider using regional- and national-level effect estimates differentiated for different design element options, insights gained through these use of these non-city specific effect estimates would be more limited. Possible design element choices considered for sensitivity analyses included: (a) lag structure, (b) copollutants models, (c) regional versus national adjustment (in the context of generating Bayes-adjusted city-specific effect estimates) and (d) modeling period and air quality metric combinations (summer versus ozone season for 8hr mean and 8hr max metrics).

- *Short-term exposure-related morbidity (hospital admissions, emergency visits and asthma exacerbations)*: Additional coverage for lag structure, copollutants models and combinations of modeling periods and air quality metrics would be considered, depending on coverage in the available literature.

While sensitivity analyses described above would both provide additional insights into overall confidence in both short-term exposure-related morbidity and mortality, given the emphasis placed on mortality in this risk assessment (as the most significant health endpoint), we would focus on completing sensitivity analyses for the mortality endpoint group.

7.7.2 Additional refinements to the core risk estimates completed for the first draft REA

A number of refinements to the set of core risk estimates would be considered for the second draft risk assessment, including:

- *Generate confidence intervals for the delta (risk reduction) estimates*: The method used for generating delta (risk reduction) estimates in the first draft REA, while providing sound point estimates, did not allow for the generating of confidence intervals reflecting the impact of statistical uncertainty associated with the fit of the effect estimates used (CIs were only generated for absolute risk for both the recent conditions and simulated attainment of the current standard scenarios). For the second draft, we will consider also generating CIs for the delta risk estimates.

- *Rigorous comparison of O_3 air quality data used in source epidemiological studies and the design of the composite monitors used in risk assessment*: For the second draft analysis, we will complete a more rigorous comparison of the composite monitor design used in the first draft REA with the methods used in the epidemiological studies underlying the C-R functions used in the risk assessment. It is likely that there will be varying degrees of agreement across the C-R functions (in relation to the way air quality data are integrated), leading to different degrees of uncertainty being introduced into the analysis. As part of the second draft analysis, we will characterize this uncertainty and will consider using alternate composite monitor designs if (a) they would more closely match the approach used in a given epidemiological study and (b) EPA staff believes this refinement is likely to make a substantial difference in risk characterization. Aspects of this task may fall into the category of sensitivity analysis, depending on how they are implemented, in which case they will be presented as part of the sensitivity analysis.

- *Further exploration of patterns of potential interest in the risk estimates*: We will complete a more thorough review of the risk estimates generated with emphasis on explaining any patterns of particular interest. An excellent example of this involves short-term exposure-related mortality modeled using C-R functions based on Zanobetti and Schwartz (2008b). As noted in section 7.5.1, these risk estimates in the form of *percent of baseline mortality* (which is normalized on population count) are up to 50% for New York City and Detroit compared with the other urban study areas. In this case, these larger risk estimates directly reflect larger effect estimates specified for these two cities in the underlying epidemiological study. As part of the second draft REA, we would provide a more thorough assessment of regionality in effect estimates reflected in this example and its impact on risk.

- *Characterizing "ranges of O_3 concentrations with increased confidence" using data from the underlying epidemiological studies rather than the use of composite monitor-based LMLs*: depending on available data, we may use LMLs values from the actual epidemiological studies underlying C-R functions to define *ranges of increased confidence* used in the risk assessment (in place of the surrogate values obtained form the composite monitor distributions used in the first draft REA). In the event that we are not able to obtain LMLs for all of the epidemiological studies used in the risk assessment, we may also consider generating surrogate LMLs based on obtaining O_3 monitoring data that matches the measurement period (range of years) used in a particular epidemiological study, rather than using the composite monitor-based LML values from the modeled (simulation) years as was done here for the first draft REA. Based on consideration for CASAC and public comments we will also consider using additional metrics, besides the LML, in specifying *ranges of increased confidence* in estimating risk. For example, we could include estimates of risk down to O_3 levels higher than the LML, to explore modeling of risk closer to the central mass of measurement data used in the epidemiological studies supporting the C-R functions.

7.7.3 Treatment of both long-term exposure-related mortality and morbidity endpoints

For the second draft REA, based on review of the evidence as summarized in the O_3 ISA (U.S. EPA, 2012), we are planning to model risk for long-term exposure-related mortality. Our rationale for this decision is laid out in greater detail in section 8.1.1.5 (Chapter 8 discusses the national-scale risk assessment, but the rationale for including long-term exposure-related mortality as presented there, also applies for the urban study area risk assessment). In summary, the decision to model long-term exposure-related mortality reflects consideration for evidence supporting the endpoint category which is suggestive of a casual association (for long-term mortality), but likely to be causal for the broader category of long-term exposure-related respiratory health effects (which includes mortality). Given that our analysis would focus on respiratory mortality (see below), we conclude that modeling long-term exposure-related (respiratory) mortality would be reasonably well-supported by the evidence. In modeling the endpoint for the urban study area risk assessment, as with the national-scale analysis, we would use the national-level respiratory effect estimate reflecting control for $PM_{2.5}$ (from Jarrett et al., 2009), with that single effect estimate being applied to each of the urban study areas. In addition, as a sensitivity analysis, we would consider modeling risk using the regional-level respiratory effect estimates presented in Table 4 of the study, although it is important to note that (a) these regional effect estimates do not include control for $PM_{2.5}$ and (b) regional differences in the ozone effect may reflect to a great extent, differing degrees of exposure measurement error (e.g., related to temperature, differing residential/commuting patterns).

With regard to long-term exposure-related morbidity, after careful review of the available evidence as summarized in the O_3 ISA (U.S. EPA, 2012), we have concluded that, while the overall body of evidence supports a likely causal association between long-term exposure and respiratory health effects, limitations in the study-level data required to support risk assessment prevents us at this point from completing a quantitative risk assessment for this category of health endpoints with a reasonable degree of confidence. It is important to emphasize that these limitations do not prevent the use of this evidence from informing consideration of the levels of exposure at which specific types of health effects may occur (i.e., the evidence analysis, which is an important aspect of the ozone NAAQS review). Rather, these limitations only prevent the quantitative estimation of risk with a reasonable degree of confidence.

In considering the potential for modeling risk for long-term exposure-related morbidity, we first identified a subset of epidemiological studies as candidates for supporting the specification of C-R functions including: (a) Meng et al., 2010 (HA and ED visits by asthmatics in San Joaquin Valley, CA), (b) Akinbami et al., 2010 (current asthma and asthma attack prevalence in children in U.S metropolitan areas), (c) Lin et al., 2008 (first asthma HA in

children in NYC and NY state), and (d) Moore et al., 2008 (hospital discharges for asthma in Southern CA). The discussion of limitations in the evidence focuses on these studies:

- When considering these studies and their potential use in quantitative analyses it is important to recognize that Meng et al. (2010) and Akinbami et al. (2010) are both cross-sectional studies. CASAC has advised us on numerous occasions to place less emphasis on the results from this type of study design due to implicit limitations and difficulty in interpreting the results.

- It is also important to consider the age range included in some of these studies that are relying on an asthma diagnosis. Diagnosing asthma in very young children (<4) is difficult. Both Lin et al. (2008) and Akinbami et al. (2010) recognize this, and to account for it exclude children under the age of 1 and 3, respectively. Still, the majority of the children included in the analysis by Lin et al (2008) are between 1 and 2 years of age, which introduces uncertainty into the diagnosis.

- Moore et al. (2008) includes a series of cross-sectional studies, where the exposure is limited to a quarterly average and linked to hospital admissions during that quarter; the analysis includes two quarters each year (spring and summer) over an 18 year period. This type of longitudinal cross-sectional study design is unusual. Although the research group behind this study has published multiple papers in high quality journals it remains unclear if using DSA in the model building step is appropriate - mostly because it is unclear how this approach selects the appropriate model.

- Lin et al. (2008a) represents the strongest of the long-term O_3 exposure and respiratory morbidity studies and its strengths are discussed in the O_3 ISA (U.S. EPA, 2012). Lin et al. is a retrospective cohort study that focuses on first time asthma hospital admission in NY state. Never the less, there are concerns related to this study when considering as the basis for C-R functions used in risk assessment:

    - Enrollment and follow-up of the cohort was done using administrative records; follow-up questionnaires were not sent out to each child that entered the cohort so that children that may have moved out of state are considered to be part of the cohort, even though they may have had a hospital admission in another state. It is unclear how this influences the overall results of the study.

    - The majority of admissions are for children between the age of 1-2, as stated previously it is sometimes difficult to diagnose asthma in children of this age. Therefore, the study may more accurately represent hospital admissions for a respiratory condition and not necessarily asthma alone. It is not known what level of uncertainty this might introduce and if the discharge diagnosis might impact this.

    - Finally, this study could be compared with Lin et al. (2008b) (Environmental Research, 108 (2008): 42-47), which examined short-term O3 exposure and respiratory hospital admissions in NY state to compare the risk estimates obtained in both studies. Further CASAC comments note the issue of

controlling for effects due to short-term exposures in such long term studies. The revised ISA notes this but does not further inform the level of uncertainty related to this issue (i.e., a long-term exposure-related capturing a short-term exposure-related signal).

Taken together, the limitations presented above resulted in EPA staff concluding that, at this time, we could not generate risk estimates for the long-term exposure-related respiratory morbidity effect category (specifically the set of health effect reflected in the four studies identified above) with a reasonable degree of confidence.

REFERENCES

Abt Associates Inc. (1996). A Particulate Matter Risk Assessment for Philadelphia and Los Angeles. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. July 1996. Available electronically on the internet at: http://www.epa.gov/ttnnaaqs/standards/pm/data/jly3amd.pdf

Abt Associates, Inc. 2010. Environmental Benefits and Mapping Program (Version 4.0). Bethesda, MD. Prepared for U.S. Environmental Protection Agency Office of Air Quality Planning and Standards. Research Triangle Park, NC. Available on the Internet at <http://www.epa.gov/air/benmap>.

Akinbami, LJ; Lynch, CD; Parker, JD; Woodruff, TJ. (2010). The association between childhood asthma prevalence and monitored air pollutants in metropolitan areas, United States, 2001-2004. Environ Res 110: 294-301.

Bell, ML; Dominici, F. (2008). Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 U.S. communities. Am J Epidemiol 167: 986-997.

Bell, ML; McDermott, A; Zeger, SL; Samet, JM; Dominici, F. (2004). Ozone and short-term mortality in 95 U.S. urban communities, 1987-2000. JAMA 292: 2372-2378.

Darrow, L. A., Klein, M., Sarnat, J. A., Mulholland, J. A., Strickland, M. J., Sarnat, S. E., et al. (2011). The use of alternative pollutant metrics in time-series studies of ambient air pollution and respiratory emergency department visits. Journal of Exposure Science and Environmental Epidemiology, 21, 10-19.

Gent, J. F., Triche, E. W., Holford, T. R., Belanger, K., Bracken, M. B., Beckett, W. S., et al. (2003). Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. Jama, 290(14), 1859-1867.

Ito, K., Thurston, G. D., & Silverman, R. A. (2007). Characterization of PM2.5, gaseous pollutants, and meteorological interactions in the context of time-series health effects models. J Expo Sci Environ Epidemiol, 17 Suppl 2, S45-60.

Katsouyanni, K., Samet, J. M., Anderson, H. R., Atkinson, R., Tertre, A. L., Medina, S., et al. (2009). Air Pollution and Health: A European and North American Approach (APHENA): Health Effects Institute

Lin, S., Bell, E. M., Liu, W., Walker, R. J., Kim, N. K., & Hwang, S. A. (2008a). Ambient ozone concentration and hospital admissions due to childhood respiratory diseases in New York State, 1991– 2001. Environmental Research, 108, 42-47.

Lin, S; Liu, X; Le, LH; Hwang, SA. (2008b). Chronic exposure to ambient ozone and asthma hospital admissions among children. Environ Health Perspect 116: 1725-1730.

Linn, W. S., Szlachcic, Y., Gong, H., Jr., Kinney, P. L., & Berhane, K. T. (2000). Air pollution and daily hospital admissions in metropolitan Los Angeles. Environ Health Perspect, 108(5), 427-434.

Medina-Ramon, M., Zanobetti, A., & Schwartz, J. (2006). The effect of ozone and PM10 on hospital admissions for pneumonia and chronic obstructive pulmonary disease: a national multicity study. Am J Epidemiol, 163(6), 579-588.

Meng, YY; Rull, RP; Wilhelm, M; Lombardi, C; Balmes, J; Ritz, B. (2010). Outdoor air pollution and uncontrolled asthma in the San Joaquin Valley, California. J Epidemiol Community Health 64: 142-147.

Moore, K; Neugebauer, R; Lurmann, F; Hall, J; Brajer, V; Alcorn, S; Tager, I. (2008). Ambient ozone concentrations cause increased hospitalizations for asthma in children: An 18-year study in Southern California. Environ Health Perspect 116: 1063-1070.

Silverman, R. A., & Ito, K. (2010). Age-related association of fine particles and ozone with severe acute asthma in New York City. J Allergy Clin Immunol, 125(2), 367-373 e365.

Smith, RL; Xu, B; Switzer, P. (2009b). Reassessing the relationship between ozone and short- term mortality in U.S. urban communities. Inhal Toxicol 21: 37-61.

Strickland, M. J., Darrow, L. A., Klein, M., Flanders, W. D., Sarnat, J. A., Waller, L. A., et al. (2010). Short-term Associations between Ambient Air Pollutants and Pediatric Asthma Emergency Department Visits. Am J Respir Crit Care Med, 182, 307-316.

Tolbert, P. E., Klein, M., Peel, J. L., Sarnat, S. E., & Sarnat, J. A. (2007). Multipollutant modeling issues in a study of ambient air quality and emergency department visits in Atlanta. J Expo Sci Environ Epidemiol, 17 Suppl 2, S29-35.

U.S. CDC, 2010. Centers for Disease Control and Prevention, Behavioral Risk Factor Surveillance System (BRFSS), 2010, Table "Table C1 Adult Self-Reported Current Asthma Prevalence Rate (Percent) and Prevalence (Number) by State or Territory", available at: http://www.cdc.gov/asthma/brfss/2010/current/tableC1.htm

U.S. Environmental Protection Agency (2001) Risk assessment guidance for Superfund. Vol. III, Part A. Process for conducting probabilistic risk assessment (RAGS 3A). Washington, DC, United States Environmental Protection Agency (EPA 540-R-02-002; OSWER 9285.7-45; PB2002 963302; http://www.epa.gov/oswer/riskassessment/rags3adt/index.htm).

U.S. Environmental Protection Agency (2004) EPA's risk assessment process for air toxics: History and overview. In: Air toxics risk assessment reference library. Vol. 1. Technical resource manual. Washington, DC, United States Environmental Protection Agency, pp. 3-1 – 3-30 (EPA-453-K-04-001A; http://www.epa.gov/ttn/fera/data/risk/vol_1/chapter_03.pdf).

U.S. Environmental Protection Agency (2007) Ozone Health Risk Assessment for Selected Urban Areas. Office of Air Quality Planning and Standards, Research Triangle Park, EPA 452/R-07-009. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr.html

U.S. Environmental Protection Agency. (2011). Ozone National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA-452/P-11-001.

U.S. Environmental Protection Agency. (2012). Integrated Science Assessment for Ozone and Related Photochemical Oxidants: Third External Review Draft, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA/600/R-10/076C.

World Health Organization. (2008). Part 1: Guidance Document on Characterizing and Communicating Uncertainty in Exposure Assessment, Harmonization Project Document No. 6. Published under joint sponsorship of the World Health Organization, the International Labour Organization and the United Nations Environment Programme. WHO Press, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland (tel.: +41 22 791 2476).

Zanobetti, A; Schwartz, J. (2008b). Mortality displacement in the association of ozone with mortality: An analysis of 48 cities in the United States. Am J Respir Crit Care Med 177: 184-189.

8 NATIONAL-SCALE RISK ASSESSMENT AND REPRESENTATIVENESS ANALYSIS

8.1 INTRODUCTION

In this section we estimate nationwide premature mortality resulting from recent exposures to ambient O_3. There are two main goals for this assessment: (1) estimate the incidence of premature mortality within the U.S. attributable to recent O_3 concentrations (Section 7.3); (2) identify where the subset of counties assessed in the urban case study areas analysis fall along the distribution of national county-level risk (Section 7.4). Compared with the urban scale analysis in Section 7.2, this analysis includes full spatial coverage across the U.S. but has less specificity in the risk-related attributes that are inputs to the health impact calculation. The national scale analysis is therefore intended as a complement to the urban scale analysis, providing both a broader assessment of O_3-related health risks across the U.S. as well as an evaluation of how well the urban study areas examined in Section 7.2 represent the full distribution of O_3-related health risks in the U.S. To perform this assessment we use a national-scale "fused" spatial surface of seasonal average O_3 concentrations from a 2007 simulation from the Community Multiscale Air Quality (CMAQ) model (Byun and Schere, 2006) and 2006-2008 O_3 air quality data. These gridded seasonal average O_3 concentrations are input into the environmental Benefits Mapping and Analysis Program (BenMAP; Abt Associates, 2010) to estimate short-term O_3-related premature mortality nationwide using city-specific mortality risk estimates from the Bell et al. (2004) study of 95 urban communities and from the Zanobetti and Schwartz (2008) study of 48 U.S. cities.

Using these methods, we estimate the total all-cause deaths associated with average 2006-2008 O_3 levels across the continental U.S. We provide three analyses to give perspective on the confidence in the estimates of O_3-related mortality: (1) risk bounded by applying the concentration-response functions down to zero (no O_3 concentration cutoff) and down to the lowest measured levels in Zanobetti and Schwartz (2008), (2) risk estimated only within the urban areas included by Bell et al. (2004) and Zanobetti and Schwartz (2008); and (3) the distribution of O_3-related deaths across the range of 2006-2008 average O_3 concentrations.

For the application of Bell et al. (2004) effect estimates for May-September, we estimate 18,000 (95% CI, 5,700-30,000) premature O_3-related deaths with no concentration cutoff and 15,000 (95% CI, 4,800-25,000) with the LML cutoff of 7.5 ppb. The estimated percentage of total county-level mortality attributable to O_3 ranges from 0.4% to 4.2% (median 1.9%) with no concentration cutoff and 0.3% to 3.5% (median 1.6%) with the LML cutoff of 7.5 ppb. For the application of Zanobetti and Schwartz (2008) effect estimates for June-August, we estimate 15,000 (95% CI, 5,800-24,000) premature O_3-related deaths with no concentration cutoff and

13,000 (95% CI, 4,900-21,000) with the LML cutoff of 7.5 ppb. The estimated percentage of total county-level mortality attributable to O_3 ranges from 0.5% to 5.2% (median 2.5%) with no concentration cutoff and 0.4% to 4.4% (median 2.1%) with the LML cutoff of 7.5 ppb. For both epidemiology studies, we find that 85-90% of O_3-related deaths occur in locations where the May to September average 8-hr daily maximum or the June-August average 8-hr daily mean (10am-6pm) O_3 concentration is greater than 40 ppb, corresponding to 4th high 8-hr daily maximum O_3 concentrations ranging from approximately 50 ppb to 100 ppb.

8.1.1 Methods

This assessment combines information regarding estimated O_3 concentrations, population projections, baseline mortality rates, and mortality risk coefficients to estimate O_3-related premature mortality. Figure 1.1 below provides a conceptual diagram detailing each of the key steps involved in performing this health impact assessment.

8.1.1.1 Estimates of Population Exposures to Ambient O_3 Concentrations

BenMAP uses projections of the size and geographic distribution of the potentially exposed population along with estimates of the ambient O_3 concentrations to estimate population exposure[1]. In contrast to the urban study areas analysis, the national scale analysis employed a data fusion approach to take advantage of the accuracy of monitor observations and the comprehensive spatial information of the CMAQ modeling system to create a national-scale "fused" spatial surface of seasonal average O_3. The spatial surface is created by fusing 2006-2008 measured O_3 concentrations with the 2007 CMAQ model simulation, which was run for a 12 km gridded domain, using the EPA's Model Attainment Test Software (MATS; Abt Associates, 2010), which employs the enhanced Voronoi Neighbor Averaging (eVNA) technique (Timin et al., 2010). More details on the ambient measurements and the 2007 CMAQ model simulation, as well as the spatial fusion technique, can be found in Wells et al. (2012). It should also be noted that this same spatial fusion technique was employed for a national-scale risk assessment by Fann et al. (2012) to produce "fused" spatial fields for O_3 and $PM_{2.5}$ and in the PM NAAQS REA to produce a national-scale spatial field for $PM_{2.5}$ (U.S. EPA, 2010). Two "fused" spatial surfaces were created for: (1) the May-September mean of the 8-hr daily maximum (consistent with the metric used by Bell et al. 2004); and (2) the June-August mean of the 8-hr daily mean from 10am to 6pm (consistent with the metric used by Zanobetti and Schwartz 2008) O_3 concentrations across the continental U.S. Figure 1.2 and Figure 1.3 show the geographic

[1] Population exposure refers to the ambient concentrations estimated for populations living in specific locations, rather than individual personal exposure to ozone (see Chapter 5 for a discussion of personal exposure modeling).

distribution of these spatial surfaces. Figure 1.4 shows the frequency and cumulative percent of the seasonal average O_3 concentrations by gridcell, using both metrics. May-September average 8-hr daily maximum concentrations are most frequently in the 40-50 ppb range, while June-August average 8-hr daily mean concentrations are more evenly distributed across a range of 20-70 ppb. Maximum concentrations for the June-August mean of the 8-hr daily mean concentrations from 10am to 6pm are generally higher than for the May-September mean of the 8-hr daily maximum concentrations since the seasonal definition is limited to the summer months when O_3 tends to be highest. The maximum, minimum, mean, median, and 95th percentile concentrations for both 8-hr daily maximum and 8-hr daily mean are shown in Table 1.1. These seasonal average metrics are not equivalent to the averaging time for the current NAAQS, which is based on the 4th highest value rather than seasonal mean, so the values should not be directly compared against the NAAQS.

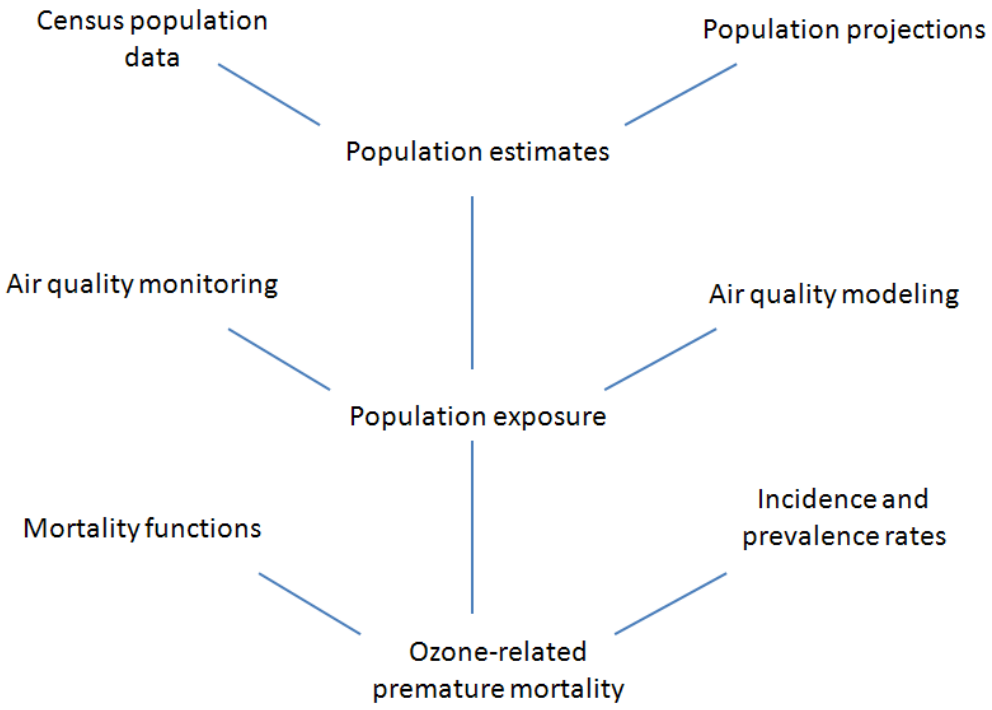


Figure 1.1 Conceptual diagram of data inputs and outputs for national short-term mortality risk assessment

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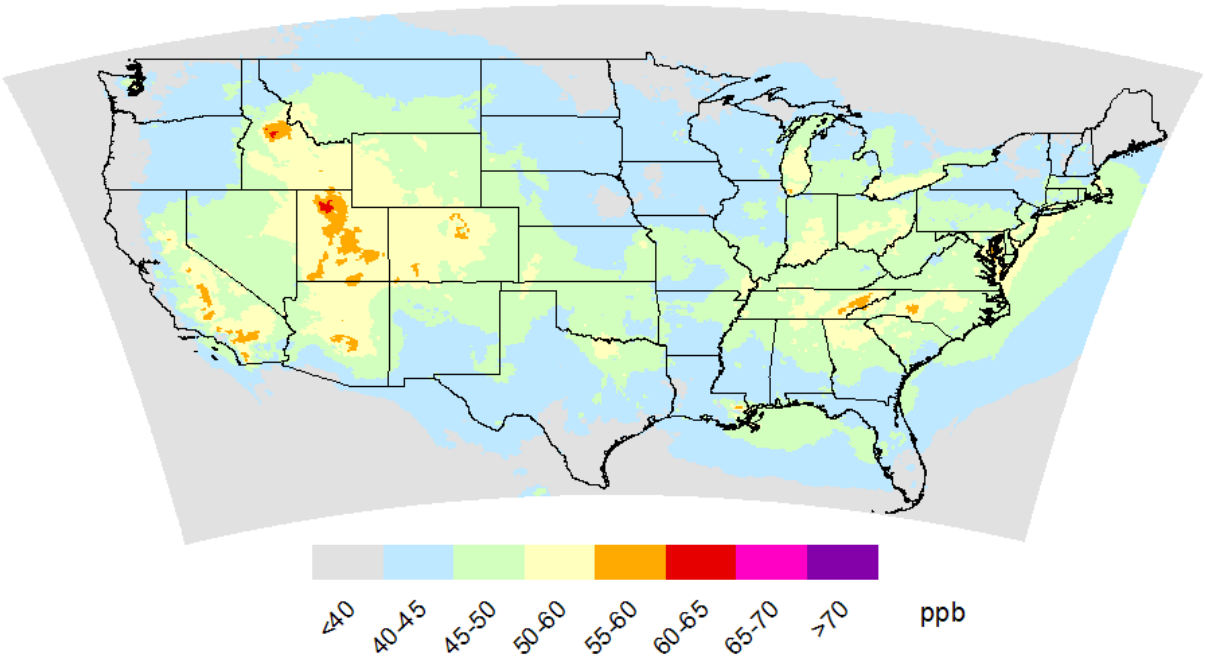

Figure 1.2 **Seasonal (May-September) average 8-hr. daily maximum baseline O_3 concentrations (ppb) at the surface, based on a 2007 CMAQ model simulation fused with average 2006-2008 observations from the O_3 monitor network.**

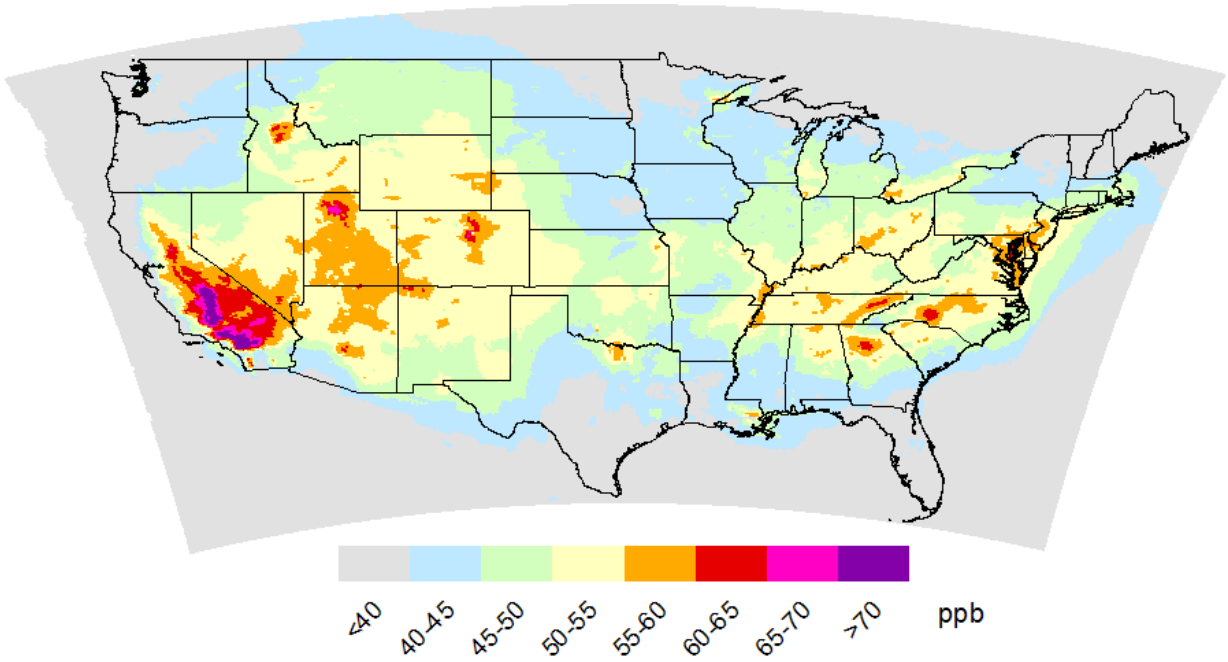

Figure 1.3 **Seasonal (June-August) average 8-hr. daily mean (10am-6pm) baseline O_3 concentrations (ppb) at the surface, based on a 2007 CMAQ model simulation fused with average 2006-2008 observations from the O_3 monitor network.**

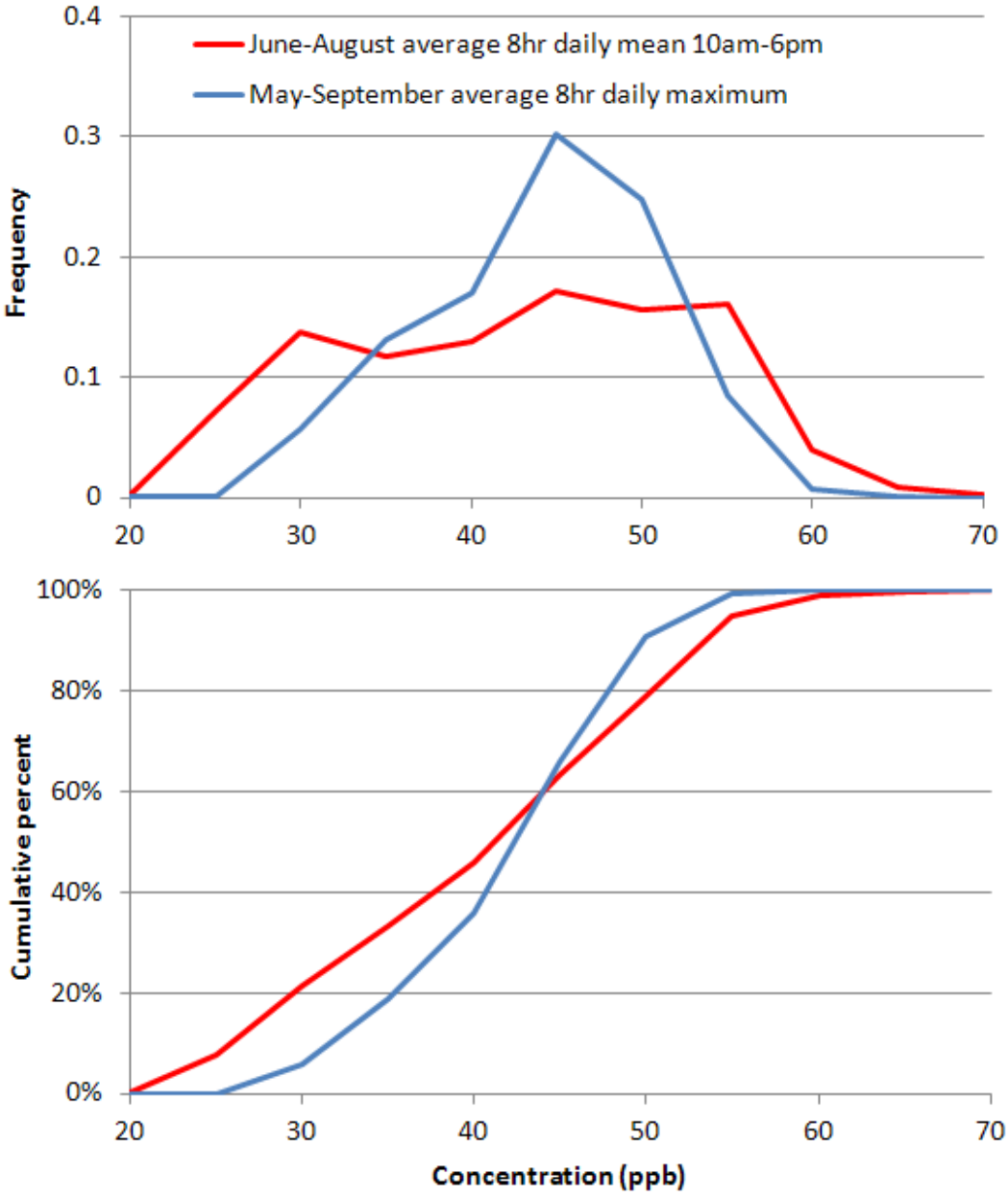

Figure 1.4 **Frequency and cumulative percent of May-September average 8-hr daily maximum and the June-August 8-hr daily mean (10am-6pm) O_3 concentration (ppb) by gridcell, based on 2006-2008 monitor observations fused with 2007 CMAQ-modeled O_3 levels.**

Table 1.1 Statistical characterization of the May-September average 8-hr daily maximum and the June-August 8-hr daily mean (10am-6pm) O_3 concentration (ppb), based on 2006-2008 monitor observations fused with 2007 CMAQ-modeled O_3 levels.

	May-September average 8-hr daily maximum concentration (ppb)	June-August average daily 10am – 6pm daily mean concentration (ppb)
Maximum	65.0	85.5
Minimum	19.7	18.0
Mean	41.8	40.4
Median	42.6	41.3
95^{th} Percentile	51.6	55.1

8.1.1.2 Baseline incidence estimates

Epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship between air quality changes and the relative risk of a health effect, rather than estimating the absolute number of avoided cases. For example, a typical result might be that a 10 ppb decrease in daily O_3 levels might, in turn, decrease hospital admissions by 3%. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. A baseline incidence rate is the estimated number of cases of the health effect per year in the assessment location, as it corresponds to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number. For example, if the baseline incidence rate is the number of cases per year per million people, that number must be multiplied by the millions of people in the total population. We derive baseline incidence rates for mortality from the CDC Wonder database (CDC, 2004-2006). The CDC Wonder database provides baseline mortality estimates that are age-, cause-, and county-specific. As this database only provides baseline incidence rates in 5-year increments, we use data for the year 2005, the closest year to the analysis year 2007 used for the population and air quality modeling.

8.1.1.3 Population estimates

The starting point for estimating the size and demographics of the potentially exposed population is the 2000 census-block level population, which BenMAP aggregates up to the same grid resolution as the air quality model. BenMAP projects this 2000 population to the analysis year of 2007 using county-level growth factors based on economic projections (Woods and

Poole Inc., 2008). We use 2007 population because it matches both the year of the emissions inventory and meteorology used for the air quality modeling.

8.1.1.4 Premature mortality estimates

To quantify the impact of O_3 concentrations on mortality, we applied risk estimates drawn from two major short-term epidemiological studies. These studies are consistent with those used in the analysis of O_3-related risk in selected urban areas (Section 7.2). We use city-specific and national average risk estimates drawn from the Bell et al. (2004) study of O_3 and mortality in 95 U.S. urban communities between 1987 and 2000, and the Zanobetti and Schwartz (2008) study of O_3 and mortality in 48 U.S. cities between 1989 and 2000. City-specific effect estimates for both studies are provided in Appendix 4-A.

Bell et al. (2004) found that the average non-accidental mortality increase across all 95 urban areas was 0.64% (95% posterior interval [PI], 0.41%-0.86%) for a 15 ppb increase in the previous week's 8-hr daily maximum O_3 concentration (equivalent to 0.43% for a 10 ppb increase), based on yearly O_3 observations (often just the O_3 season, April to October). As the national-scale analysis requires a single modeling period definition, the corresponding city-specific effect estimates are applied to each day from May to September in BenMAP using the 2006-2008 average May to September mean 8-hr daily maximum O_3 concentration. The length of the O_3 season can affect the magnitude of mortality effect estimates. Bell et al. (2004) reported that a 10 ppb increase in 24-hr average O_3 concentration was associated with a 0.52% (95% PI, 0.27%-0.77%) increase in mortality using all O_3 data and a 0.39% (95% PI, 0.13%-0.65%) increase in mortality using only days from April to October. Since O_3 values are typically higher during the summer season, the higher effect estimate derived from year-round O_3 data may yield an equivalent O_3 mortality impact as the lower effect estimate derived from the warm season O_3 data only. For the second draft Risk and Exposure Assessment, EPA staff proposes to use city-specific 8-hr daily maximum effect estimates for the warm season only, if available, to model risk for the corresponding months.

Zanobetti and Schwartz (2008) found that the average total mortality increase across all 48 cities was 0.53% (95% confidence interval, 0.28%-0.77%) for a 10 ppb increase in June-August 8-hr daily mean O_3 concentration from 10 am to 6 pm, using a 0-3 day lag. We apply the city-specific effect estimates that correspond to this national average effect estimate each day from June to August in BenMAP using the 2006-2008 June to August mean 8-hr daily mean O_3 concentration.

As this national assessment applies to the entire geographical scale of the continental U.S. in a gridded format, it includes locations not covered by the Bell et al. (2004) and Zanobetti

and Schwartz (2008) studies. For gridcells outside of the urban areas included by the epidemiological studies, we assign the average effect estimate derived from all the urban areas included in each of the studies ("national average"). Applying the national average estimate takes advantage of a broader population and the variability among population response to O_3 introduced by effect modifying characteristics, compared with an alternative approach of assigning these gridcells the effect estimate from the nearest urban area. Since both national average estimates from these studies are based on urban areas only, we have higher confidence in their application to other U.S. urban areas than to rural areas. To demonstrate the magnitude of the results for which we have the highest confidence, we present the percentage of estimated deaths occurring within the urban areas included in the epidemiological studies. It should be noted, however, that we also have high confidence in the magnitude of results in U.S. urban areas that were excluded from the epidemiological studies, since results from the 48 city study by Zanobetti and Schwartz (2008) were generally comparable to results from the larger 90 city study by Bell et al. (2004). In addition, lower confidence in the results for rural areas does not indicate that the mortality risk among populations living in such areas is unaffected by O_3 pollution. Rather, the level of understanding for the O_3-mortality relationship in these areas is simply lower due to a lack of available epidemiological data at these levels.

The current literature does not support the existence of concentration thresholds below which O_3 is not associated with health effects (U.S. EPA 2012a). However, the concentration-response relationship is less certain at lower O_3 concentrations since fewer observations at those levels exist to inform the epidemiology studies. Consistent with the approach used in the urban case studies (see Chapter 7), in addition to estimating risk for the full distribution of concentrations (i.e. down to zero), we estimate risk occurring above the lowest measured level (LML) in the underlying epidemiological studies. In order to apply the LML in all locations in the U.S., we use the average LML across all cities in the Zanobetti and Schwartz (2008) study, 7.5 ppb, as a surrogate for the location specific LML. In the second draft REA we will explore the implications of variability in the LML on the national mortality risk estimates. We apply the LML of 7.5 ppb in estimating mortality risks using the C-R functions from both Zanobetti and Schwartz (2008) and Bell et al. (2004) because the data on LMLs were not available for the Bell et al. (2004) study. We also show the distribution of O_3-related deaths by baseline O_3 concentration to provide context for interpreting confidence in the magnitude of the mortality estimates.

8.1.1.5 Consideration of long-term O_3-related mortality

The Integrated Science Assessment for O_3 and Related Photochemical Oxidants (O_3 ISA) concluded that the evidence supports a likely to be causal relationship between long-term O_3 exposure and respiratory effects, including respiratory morbidity and respiratory-related mortality (U.S. EPA, 2012a). One major national-scale cohort study has found a significant positive relationship between long-term O_3 exposure and mortality (Jerrett et al. 2009). Another study with a cohort limited to individuals with chronic conditions that might predispose to O_3 effects (chronic obstructive pulmonary disease, diabetes, congestive heart failure, and myocardial infarction) also found that long-term O_3 exposure is associated with increased risk of death in these groups (Zanobetti and Schwartz 2011). The O_3 ISA concluded that these findings are consistent and coherent with the evidence from the epidemiologic, controlled human exposure, and animal toxicological studies for the effects of long-term exposure to O_3 on respiratory effects (U.S. EPA 2012a, Section 7.7.1).

After considering its strengths and weaknesses, EPA staff considers the Jerrett et al. (2009) study to be an appropriate basis for estimating long-term O_3-related respiratory mortality risk in the 2nd draft REA. Key strengths of this study are that it included 1.2 million participants in the American Cancer Society cohort from all 50 states, DC, and Puerto Rico; included O_3 data from 1977 (5 years before enrollment in the cohort began) to 2000; considered co-pollutant models that controlled for $PM_{2.5}$; and evaluated for threshold concentrations. Key limitations are possible exposure misclassification and uncontrolled confounding by $PM_{2.5}$ and temperature, which are endemic to most long-term epidemiological studies. We note that while Jerrett et al. (2009) found negative associations between O_3 exposure and cardiovascular mortality when controlling for $PM_{2.5}$, null or negative associations are consistent with the evidence that $PM_{2.5}$ is strongly associated with cardiovascular disease (EPA 2009 PM ISA). Based largely on the findings of this study and considering its strengths and weaknesses, the O_3 ISA concluded that the evidence was strong enough to be suggestive of a causal relationship for long-term O_3 exposure and mortality.

Recent studies have used long-term O_3-mortality relationships found by Jerrett et al. (2009) to quantify the burden of mortality due to anthropogenic O_3 globally (Anenberg et al. 2010, 2011) and for the U.S. specifically (Fann et al. 2012). These studies have found that using Jerrett et al. (2009) long-term effect estimates yields O_3-related mortality burden estimates that are approximately two to four times larger than estimates based on Bell et al. (2004) short-term effect estimates. Since long-term mortality relationships include both acute and chronic exposure effects, the significantly larger mortality estimates calculated using long-term concentration-mortality relationships suggest that considering only short-term mortality may exclude a substantial portion of O_3-related risk.

EPA staff plans to quantify long-term O_3-attributable respiratory-mortality in the 2nd draft Risk and Exposure Assessment to be completed in November 2012 for two main reasons: (1) the O_3 ISA has concluded that evidence indicates a likely to be causal relationship for long-term ozone exposure and respiratory effects, including respiratory morbidity and respiratory-related mortality, and (2) long-term respiratory-related mortality estimates may provide a more comprehensive estimate of O_3-related health risks, as they include both acute and chronic exposure effects. To quantify long-term O_3-attributable respiratory-related mortality risks, EPA staff plans to use the respiratory mortality effect estimates from the Jerrett et al. (2009) two-pollutant model that controlled for $PM_{2.5}$ concentrations, applied to each gridcell across the entire United States. This model found that a 10 ppb increase in the May-September average of the 1-hr daily maximum O_3 concentration was associated with a 4% (95% confidence interval, 1.0%-6.7%) increase in respiratory mortality.

8.1.2 Results

Table 1.2 summarizes the estimated O_3-related premature mortality associated with 2006-2008 average O_3 concentrations under various assumptions for the health impact function. For the application of Bell et al. (2004) effect estimates for May-September, we estimate 18,000 (95% CI, 5,700-30,000) premature O_3-related deaths with no concentration cutoff and 15,000 (95% CI, 4,800-25,000) with the LML cutoff of 7.5 ppb. For the application of Zanobetti and Schwartz (2008) effect estimates for June-August, we estimate 15,000 (95% CI, 5,800-24,000) premature O_3-related deaths with no concentration cutoff, and 13,000 (95% CI, 4,900-21,000) with the LML cutoff of 7.5 ppb. These results are calculated by applying the city-specific risk estimates from each epidemiological study to the gridcells corresponding to each urban area, and applying the national average risk estimate (based on all urban areas included in the study) from the same study to all other gridcells. Figure 1.5 and Figure 1.6 show that estimated O_3-related mortality is most concentrated in highly populated counties or those counties with urban areas found to have high effect estimates by Bell et al. (2004) or Zanobetti and Schwartz (2008).

Because the epidemiological studies included only selected urban areas, we are more confident in the magnitude of the estimated O_3-related deaths occurring within those urban areas. Approximately 35% and 30% of the estimated O_3-related deaths occur in the urban locations included by Bell et al. (2004; 95 urban areas) and Zanobetti and Schwartz (2008; 48 urban areas), respectively. We also have high confidence in extrapolating the national average effect estimates to other urban areas, as the national average estimates are based on all urban areas included by the study. While our confidence is lower when the national average effect estimates

are extrapolated to rural areas, it is important to note that less certainty in the magnitude of O_3-related deaths in rural areas does not imply a null effect of O_3 on health in these areas.

Table 1.2 also shows O_3-related deaths estimated by applying the national average risk estimate from the epidemiological studies to all gridcells in the United States. Compared with applying city-specific effect estimates to the gridcells corresponding to each urban area, using the national average effect estimate for all gridcells yields equivalent central estimates. However, applying the national average also results in tighter confidence intervals since the national average effect estimates had higher statistical power and thus tighter confidence bounds compared with the effect estimates for individual cities.

Table 1.3 shows the mean, median, minimum, and maximum of the estimated percentage of mortality attributable to ambient O_3 across all counties in the U.S. Using Bell et al. (2004) effect estimates, the estimated percentage of total county-level mortality attributable to O_3 ranges from 0.4% to 4.2% (median 1.9%) with no concentration cutoff and from 0.3% to 3.5% (median 1.6%) with the LML cutoff of 7.5 ppb. For results using Zanobetti and Schwartz (2008) effect estimates, the estimated percentage of total county-level mortality attributable to O_3 ranges from 0.5% to 5.2% (median 2.5%) with no concentration cutoff and from 0.4% to 4.4% (median 2.1%) with the LML cutoff of 7.5 ppb. Figure 1.7 and Figure 1.8 show that the counties with the highest percentage of mortality attributable to O_3 are typically those with the highest O_3 levels (see Figure 1.2 and Figure 1.3).

Figure 1.9 displays the cumulative distribution of the percent of county-level total mortality attributable to ambient O_3 using effect estimates from both epidemiological studies with no concentration cutoff and using the LML cutoff. For the results based on Bell et al. (2004) effect estimates with no concentration cutoff, 1.5% to 2.2% of total mortality is attributable to O_3 for approximately 95% of U.S. counties. For the results based on Zanobetti and Schwartz (2008) effect estimates with no concentration cutoff, between 2% and 3% of total mortality is attributable to O_3 for approximately 90% of U.S. counties.

Table 1.2 Estimated O_3-related premature mortality associated with 2006-2008 average O_3 concentrations (95th percentile confidence interval)

Risk estimate and concentration cutoff	City-specific effect estimates[1]	National average effect estimate[2]	% reduced from no concentration cutoff
Bell et al. (2004), May-September			
None	18,000 (5,700-30,000)	18,000 (12,000-24,000)	-
7.5 ppb (LML)	15,000 (4,800-25,000)		17%
Zanobetti and Schwartz (2008), June-August			
None	15,000 (5,800-24,000)	15,000 (8,200-22,000)	-
7.5 ppb (LML)	13,000 (4,900-21,000)		28%

[1]City-specific effect estimates are applied to the gridcells lying within the cities defined in the epidemiological studies. Average effect estimates across all cities included in the epidemiological studies (national average) are applied to all other gridcells.

[2]National average effect estimates are based on the average of all cities included in the epidemiological studies.

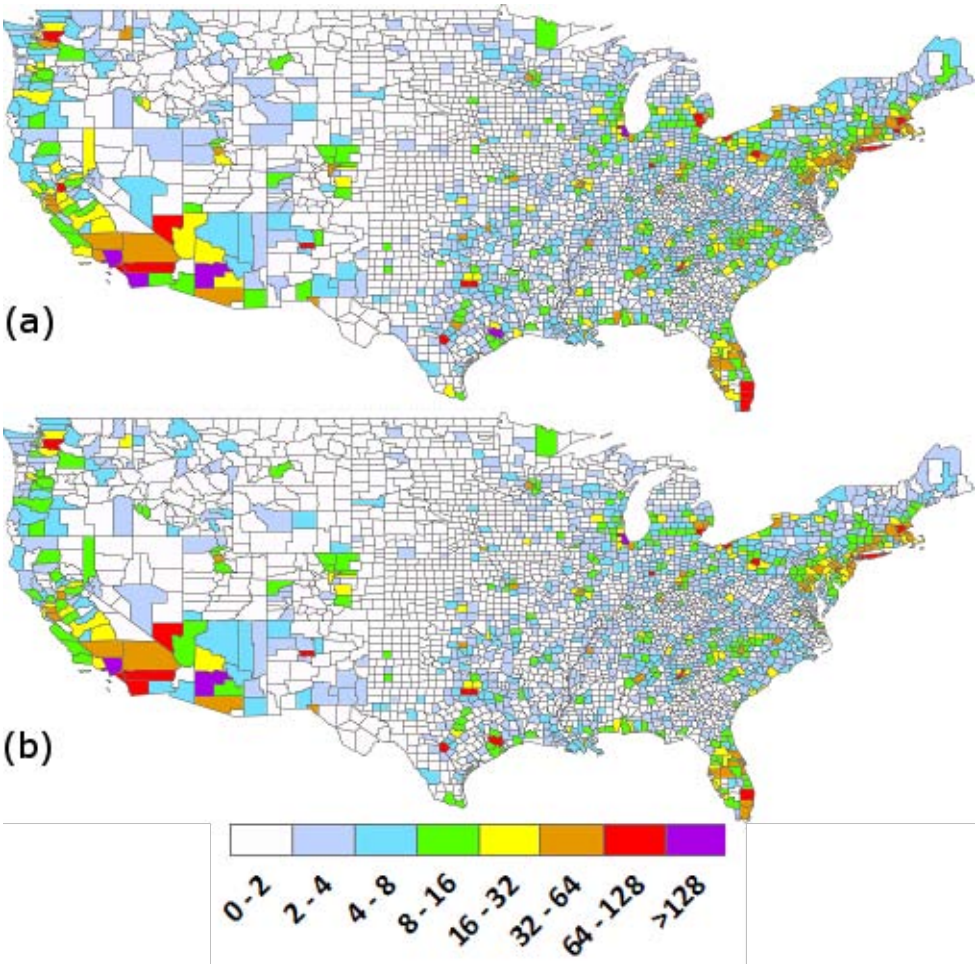

Figure 1.5 **Estimated non-accidental deaths associated with average 2006-2008 May-September average 8-hr daily maximum O_3 levels by county using Bell et al. (2004) effect estimates and (a) no concentration cutoff, (b) LML cutoff of 7.5 ppb.**

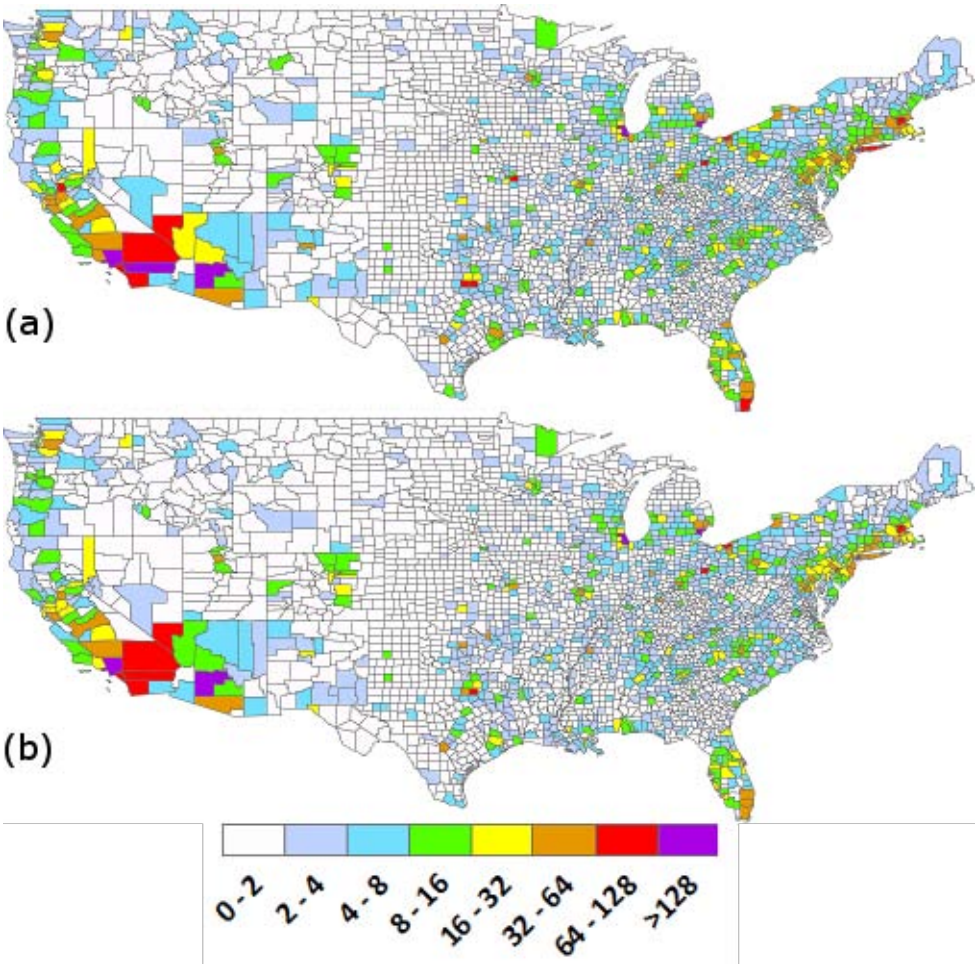

Figure 1.6 **Estimated all-cause deaths associated with average 2006-2008 June-August average 8-hr daily mean (10am-6pm) O_3 levels by county using Zanobetti and Schwartz (2008) effect estimates and (a) no concentration cutoff, (b) LML cutoff of 7.5 ppb.**

Table 1.3 Mean, median, minimum, and maximum of the estimated percentage of mortality attributable to ambient O_3 for all U.S. counties.

Risk estimate and concentration cutoff	Mean (%)	Median (%)	Minimum (%)	Maximum (%)
Bell et al. (2004), May-September				
None	1.9	1.9	0.4	4.2
7.5 ppb (LML)	1.6	1.6	0.3	3.5
Zanobetti and Schwartz (2008), June-August				
None	2.5	2.5	0.5	5.2
7.5 ppb (LML)	2.1	2.1	0.4	4.4

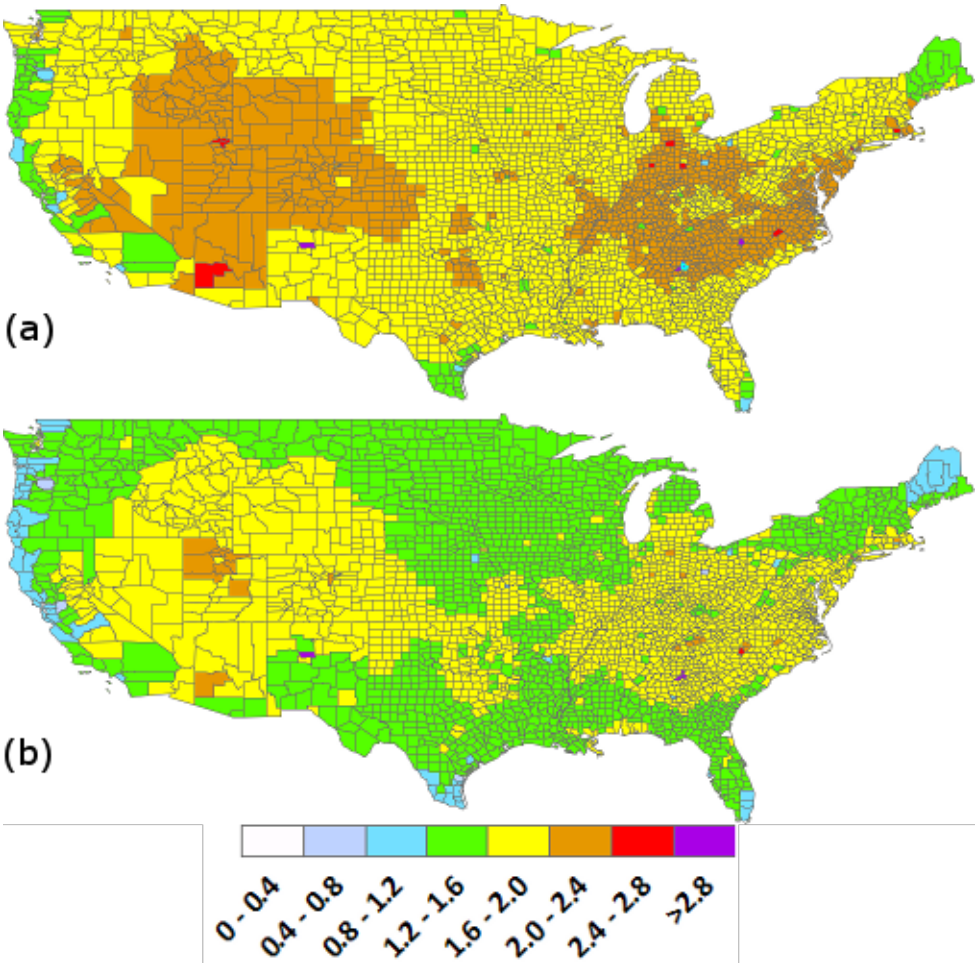

Figure 1.7 Estimated percentage of May-September total mortality attributable to 2006-2008 average O_3 levels by county using Bell et al. (2004) effect estimates and (a) no concentration cutoff, (b) LML cutoff of 7.5 ppb.

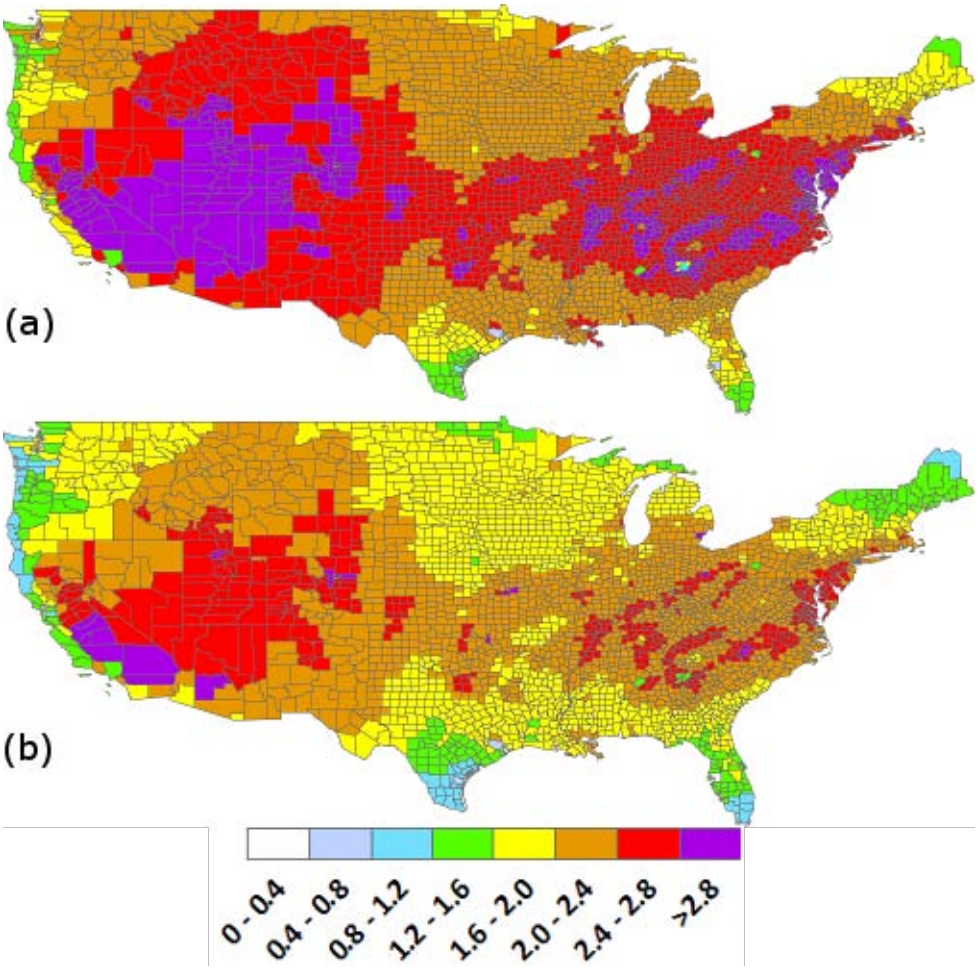

Figure 1.8 **Estimated percentage of June-August total mortality attributable to 2006-2008 average O_3 levels by county using Zanobetti and Schwartz (2008) effect estimates and (a) no concentration cutoff, (b) LML cutoff of 7.5 ppb.**

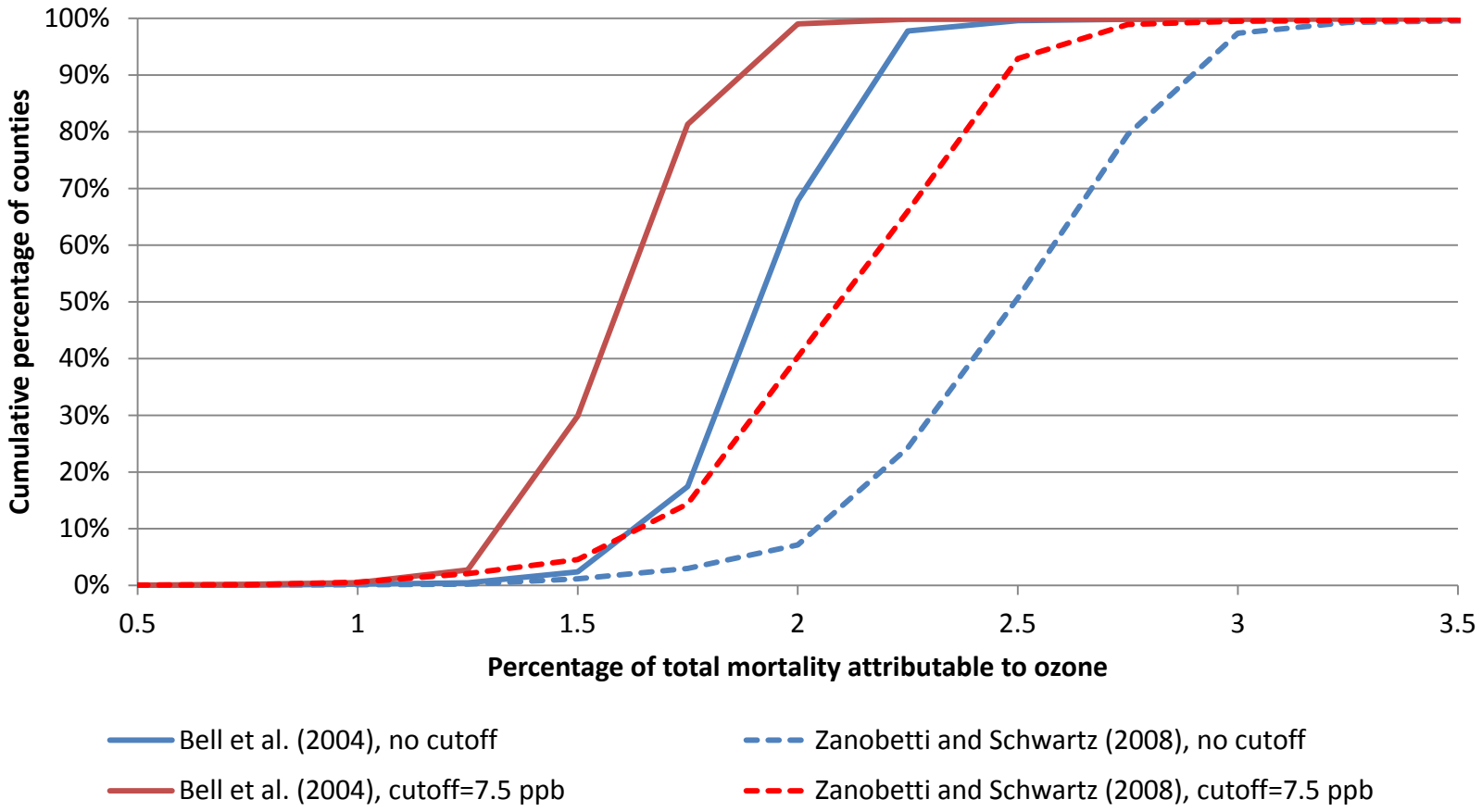

Figure 1.9 **Cumulative distribution of county-level percentage of total mortality attributable to 2006-2008 average O_3 for the U.S., using city-specific effect estimates. Results based on Bell et al. (2004) effect estimates are for non-accidental mortality, while those based on Zanobetti and Schwartz (2008) effect estimates are for all-cause mortality.**

Figure 1.10 shows the cumulative distribution of the county-level percent of total O_3-related deaths by O_3 concentration. The mortality results based on Bell et al. (2004) concentration-response functions are compared with the May-September average of the 8-hr daily maximum O_3 concentration, while those based on Zanobetti and Schwartz (2008) concentration-response functions are compared with the June-August average of the 8-hr mean O_3 concentration from 10am to 6pm, consistent with the O_3 concentration metrics used in each study. The mortality results based on Zanobetti and Schwartz (2008) effect estimates are shifted to the right of the mortality results based on the Bell et al. (2004) concentration response functions because the seasonal averaging time for the results based on Zanobetti and Schwartz (2008) is limited to the summer months when O_3 tends to be highest. The 4^{th} highest 8-hr daily maximum O_3 concentrations are typically 50% higher than the corresponding May-September average of the 8-hr daily maximum concentration, with a range across all gridcells of 14% to 270% (Figure 1.11). For the June-August average of the 8-hr daily mean from 10am-6pm, the corresponding 4^{th} high 8-hr daily maximum concentrations are typically 60% higher, with a range from 13% to 360% (Figure 1.12). For both epidemiology studies, we find that 85-90% of O_3-related deaths occur in locations where the May to September average 8-hr daily maximum or June to August 8-hr daily mean (10am-6pm) O_3 concentrations are greater than 40 ppb. When the May to September average of the 8-hr daily maximum is 40 ppb, the 4^{th} high 8-hr daily maximum ranges from approximately 50 ppb to 90 ppb (Figure 1.11). When the June to August average of the 8-hr daily mean from 10am-6pm is 40 ppb, the 4^{th} high 8-h daily maximum ranges from approximately 50 ppb to 100 ppb (Figure 1.12).

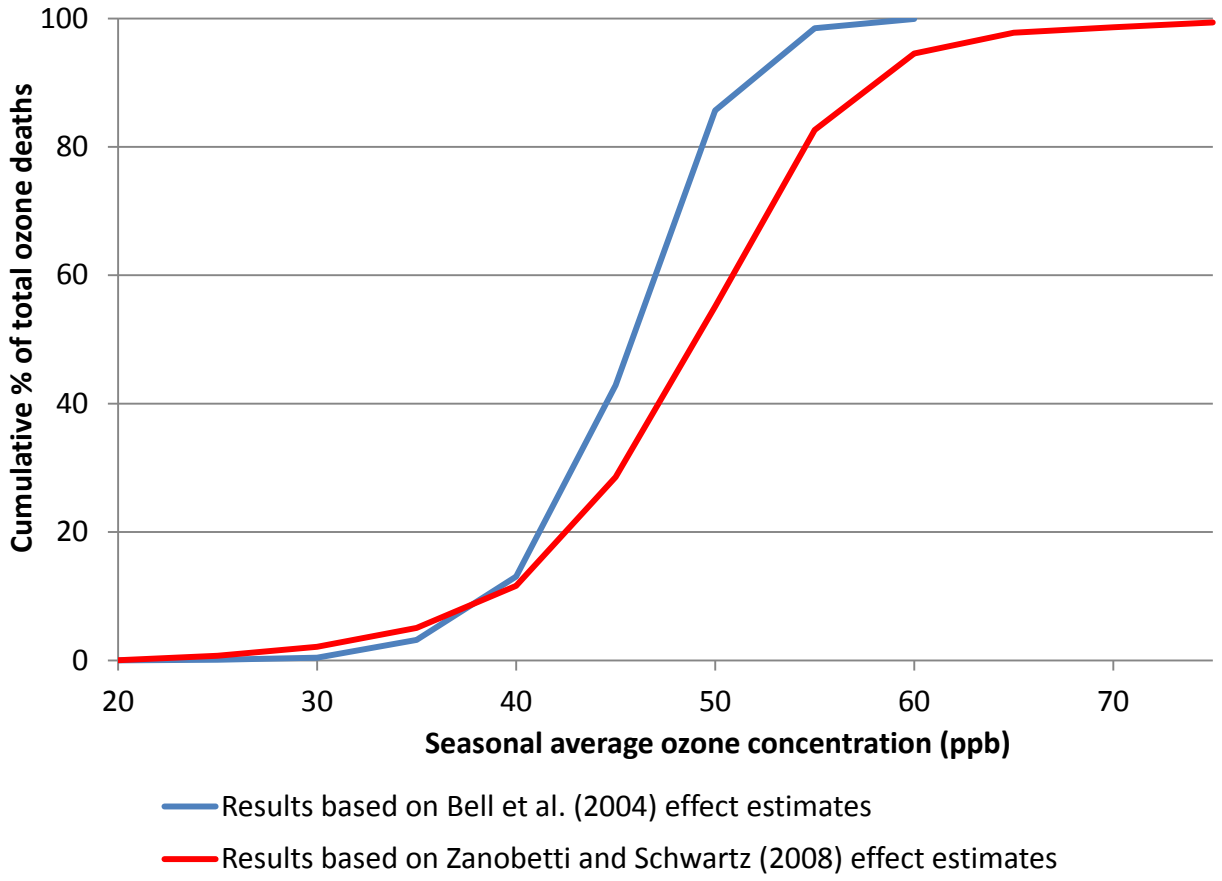

Figure 1.10 Cumulative percentage of total O_3 deaths by baseline O_3 concentration, using city-specific effect estimates. O_3 concentrations are reported as May-September average 8-hr daily maximum for results based on Bell et al. (2004) effect estimates and June-August average 8-hr mean (10am to 6pm) for results based on Zanobetti and Schwartz (2008) effect estimates.

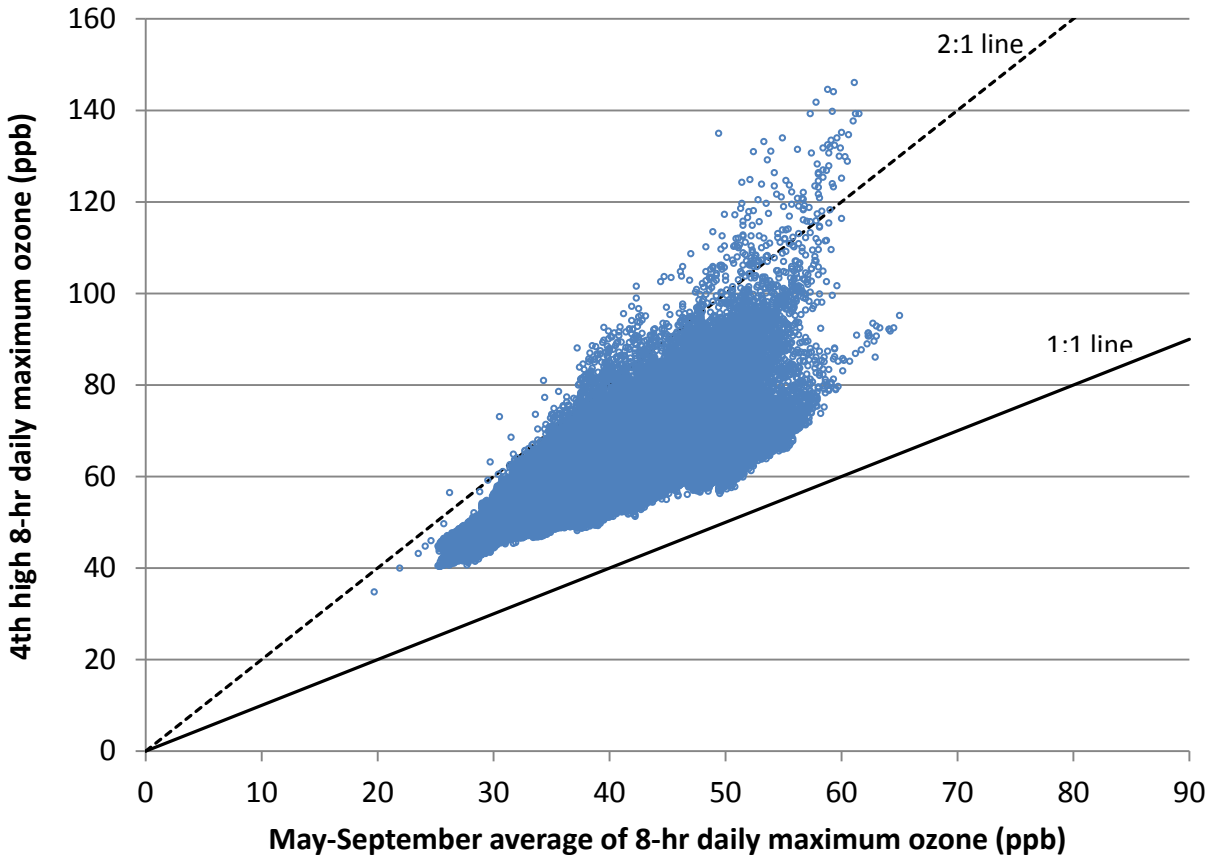

Figure 1.11 Gridcell values of 4th high 8-hr daily maximum O_3 concentrations versus May-September average of 8-hr daily maximum O_3 concentrations for the average of 2006-2008.

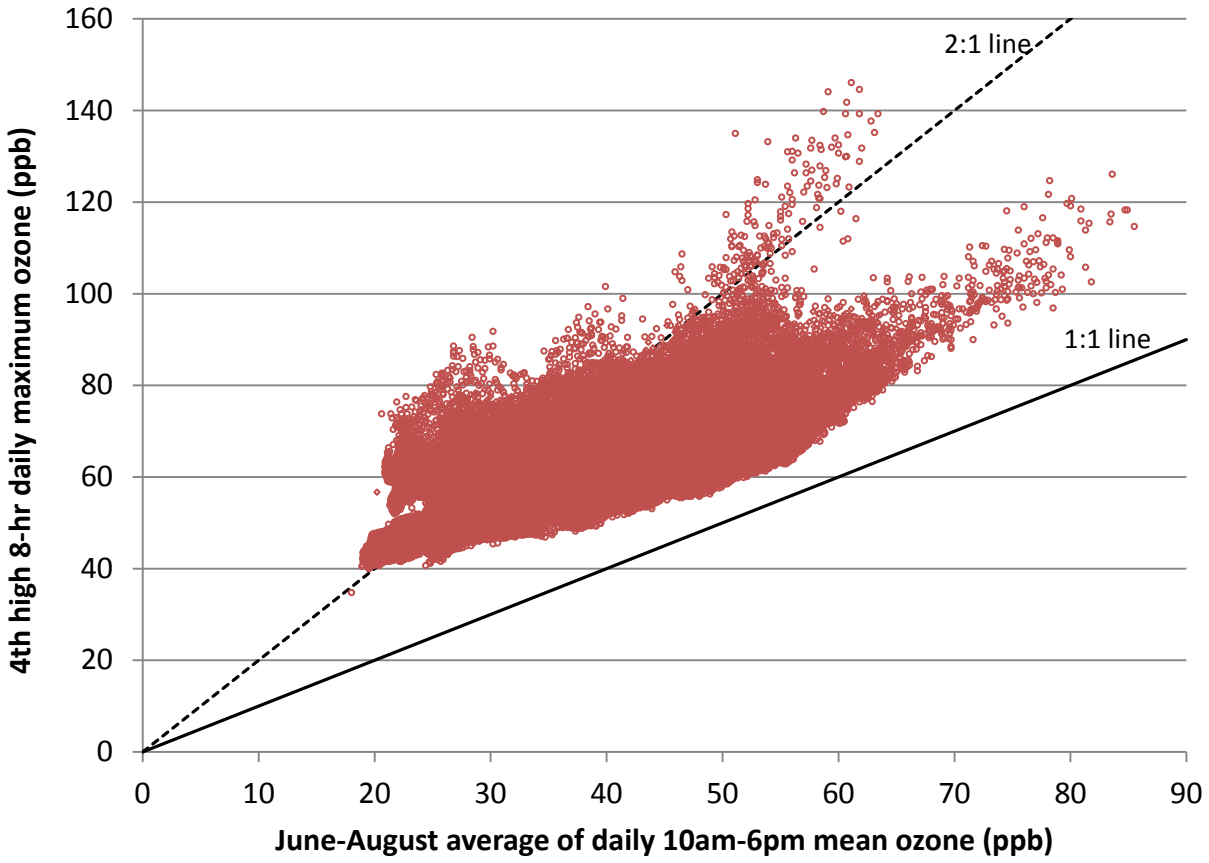

Figure 1.12 Gridcell values of 4th high 8-hr daily maximum O_3 concentrations versus June-August average of 8-hr daily 10am-6pm mean O_3 concentrations for the average of 2006-2008.

8.1.3 Discussion

We estimated the total all-cause deaths associated with short-term exposure to recent O_3 levels across the continental U.S., using average 2006-2008 observations from the O_3 monitoring network fused with a 2007 CMAQ simulation and city-specific O_3-mortality effect estimates from two short-term epidemiology studies. For the application of Bell et al. (2004) effect estimates for May-September, we estimate 18,000 (95% CI, 5,700-30,000) premature O_3-related deaths with no concentration cutoff and 15,000 (95% CI, 4,800-25,000) with the LML cutoff of 7.5 ppb. The estimated percentage of total county-level mortality attributable to O_3 ranges from 0.4% to 4.2% (median 1.9%) with no concentration cutoff and from 0.3% to 3.5% (median 1.6%) with the LML cutoff of 7.5 ppb. For the application of Zanobetti and Schwartz (2008) effect estimates for June-August, we estimate 15,000 (95% CI, 5,800-24,000) premature O_3-related deaths with no concentration cutoff and 13,000 (95% CI, 4,900-21,000) with the LML

cutoff of 7.5 ppb. The estimated percentage of total county-level mortality attributable to O_3 ranges from 0.5% to 5.2% (median 2.5%) with no concentration cutoff, and from 0.4% to 4.4% (median 2.1%) with the LML cutoff of 7.5 ppb. For both epidemiology studies, we find that 85-90% of O_3-related deaths occur in locations where the seasonal average 8-hr daily maximum or 8-hr daily mean (10am-6pm) O_3 concentration is greater than 40 ppb, corresponding to 4th high 8-hr daily maximum O_3 concentrations ranging from approximately 50 ppb to 100 ppb.

A previous analysis estimated that short-term O_3 exposure was associated with 4,700 (95% CI, 1,800-7,500) premature deaths nationwide, based on 2005 O_3 concentrations and Bell et al. (2004) national average effect estimates (Fann et al. 2012). The results estimated here are generally higher, depending on the concentration cutoff. These methods differ from those of Fann et al. (2012) in two important ways. First, Fann et al. (2012) estimated risk only above North American background, simulated O_3 concentrations in the absence of North American anthropogenic emissions, which was set to 22 ppb in the east and 30 ppb in the west. The mortality results shown in Table 1.2 that are based on the most comparable concentration cutoff of 29 ppb (10th percentile of O_3 concentrations observed by Zanobetti and Schwartz 2008) are approximately 40% larger than the estimate by Fann et al. (2012). Another important difference is that Fann et al. (2012) used a national average mortality effect estimate for 8-hr daily maximum O_3 during the warm season only, calculated using ratios of 24-hr mean concentrations to 8-hr daily maximum concentrations (see Abt Associates 2010). The Bell et al. (2004) national average beta used here, 0.000425, is based on yearly O_3 data and is approximately 60% larger than that used by Fann et al. (2012), 0.000261. Since the risk modeling period (and the seasonal definition for the seasonal average 8-hr daily maximum concentration) was May to September for both studies, the higher beta used here yields a larger O_3 mortality estimate. These two differences in methods explain the larger O_3 mortality estimates of this analysis compared with the previous estimate by Fann et al. (2012). As previously mentioned, for the second draft Risk and Exposure Assessment, EPA staff proposes to use city-specific 8-hr daily maximum effect estimates for the warm season only, if available, to model risk for the corresponding months.

8.2 EVALUATING THE REPRESENTATIVENESS OF THE URBAN STUDY AREAS IN THE NATIONAL CONTEXT

The goal in selecting the 12 urban study areas included in this risk assessment was twofold: (1) to choose urban locations with relatively elevated ambient O_3 levels (in order to evaluate risk for locations likely to experience some degree of risk reduction under alternative standards) and (2) to include a range of urban areas reflecting heterogeneity in other O_3 risk related attributes across the country. When selecting the cities, we took into account the

following criteria:(1) availability of data; (2) O_3 concentrations measured between 2006-2010; (3) inclusion of sensitive populations; and (4) geographical heterogeneity. The "data availability" criteria reflected the need for the urban area to have short-term mortality and morbidity study data that could be used in the risk and exposure assessment, detailed air conditioning prevalence data (that could be used in the exposure assessment analyses described in Chapter 5), and baseline health information. The other selection criteria reflect the desire to include urban areas that had relatively elevated ambient O_3 levels and that geographically represented the different regions of the U.Ss, as well as the desire to include sensitive population in the risk and exposure assessment.

To further support interpretation of risk estimates generated in Section 7.2, we included two analyses that assess the representativeness of the 12 urban study areas in the national context. First, we assessed the degree to which the urban study areas represent the range of key O_3 risk-related attributes that spatially vary across the nation. We have partially addressed this issue by selecting urban study areas that provide coverage for different O_3 regions of the country (see Section 7.2). In addition, we have evaluated how well the selected urban areas represent the overall U.S. for a set of spatially-distributed O_3 risk related variables (e.g. weather, demographics including socioeconomic status, baseline health incidence rates). This analysis, which is discussed in Section 7.4.1, helps inform how well the urban study areas reflect national-level variability in these key O_3 risk-related variables. The second representativeness analysis, which is discussed in Section 7.4.2, identified where the 23 counties comprising our 12 urban study areas fall along the distribution of national county-level O_3-attributable mortality risk. This analysis allowed us to assess the degree of which the 12 urban study areas capture locations within the U.S. likely to experience elevated levels of risk related to ambient O_3.

We observe that the 23 counties for the 12 urban study areas considered in Section 7.2 capture urban areas that are among the most populated in the U.S., have relatively high O_3 levels, and represent the range of city-specific effect estimates found by Bell et al. (2004) and Zanobetti and Schwartz (2008). These three factors suggest that the urban study areas capture overall risk for the nation well, with a potential for better characterization of the high end of the risk distribution. We find that the urban study areas are not capturing areas with the highest baseline mortality rates, those with the oldest populations, and those with the lowest air conditioning prevalence. These areas tend to have relatively low O_3 concentrations and low total population, suggesting that the urban study areas are not missing high risk populations that have high O_3 concentrations in addition to greater susceptibility per unit O_3. The second representativeness analysis demonstrated that the 12 urban study areas represent the full range of county-level O_3-related risk across the entire U.S.

8.2.1 Analysis Based on Consideration of National Distributions of Risk-Related Attributes

As noted above, the first representativeness analysis evaluated how well the urban study areas reflect national-level variability in a series of O_3 risk-related variables. For this analysis, we first generated distributions for risk-related variables across U.S. counties and for the specific counties considered in Section 7.2 from generally available data (e.g. from the 2000 Census, Centers for Disease Control (CDC), or other sources). We then plotted the specific values of these variables for the selected urban study areas on these distributions, and evaluated how representative the selected study areas are of the national distributions for these individual variables.

Estimates of risk (either relative or absolute, e.g. number of cases) within our risk assessment framework are based on four elements: population, baseline incidence rates, air quality, and the coefficient relating air quality and the health outcome (i.e. the O_3 effect estimates). Each of these elements can contribute to heterogeneity in risk across urban locations, and each is variable across locations. In addition, there may be additional identifiable factors that contribute to the variability of the four elements across locations. In this assessment, we examine the representativeness of the selected urban area locations for the four main elements, as well as factors that have been identified as influential in determining the magnitude of the C-R function across locations.

While personal exposure is not incorporated directly into O_3 epidemiology studies, differences in the O_3 effect estimates between cities is impacted by differing levels of exposure which in turn are related to a number of exposure determinants. The correlation between monitored O_3 and personal O_3 exposure also varies between cities. The O_3 ISA has comprehensively reviewed epidemiological and toxicological studies to identify variables which may affect the O_3 effect estimates used in the city-specific risk analysis in Section 7.2 and the national-scale risk analysis in Section 7.3 (U.S. EPA 2012a Section 6.6). Broadly speaking, determinants of the O_3 effect estimates used in risk assessment can be grouped into three areas:

- Demographics: education, income, age, unemployment rates, race, body mass index and physical conditioning, public transportation use, and time spent outdoors.
- Baseline health conditions: asthma, chronic obstructive pulmonary disease, cardiovascular disease (atherosclerosis, congestive heart disease, atrial fibrillation, stroke), diabetes, inflammatory diseases, and smoking prevalence.
- Climate and air quality: O_3 levels, co-pollutant levels (annual mean $PM_{2.5}$), temperatures (days above 90 degrees, mean summer temp, 98^{th} percentile temp), and air conditioning prevalence.

Based on these identified potential risk determinants, we identified datasets that could be used to generate nationally representative distributions for each parameter. We were not able to identify readily available national datasets for all variables. In these cases, if we were able to identify a broad enough dataset covering a large enough portion of the U.S., we used that dataset to generate the parameter distribution. In addition, we were not able to find exact matches for all of the variables identified through our review of the literature. In cases where an exact match was not available, we identified proxy variables to serve as surrogates. For each parameter, we report the source of the dataset, its degree of coverage, and whether it is a direct measure of the parameter or a proxy measure. The target variables and sources for the data are provided in Table 1.4. Summary statistics for the most relevant variables are provided in Table 1.5.

Figure 1.13 through Figure 1.19 show the cumulative distribution functions (CDF) plotted for the nation for the four critical risk function elements (population, air quality, baseline incidence, and the O_3 effect estimate), as well as where the urban study areas fall on the distribution. These figures focus on critical variables representing each type of risk determinant, e.g. we focus on all-cause and non-accidental mortality rates, but we also have conducted analyses for cardiovascular and respiratory mortality separately. The vertical black lines in each graph show the values of the variables for the individual urban study areas. The city-specific values that comprise the national CDF for mortality risks found by Zanobetti and Schwartz (2008) are also displayed on the graphs of those attributes, as the number of cities included in that study is smaller (48 cities). The complete set of analyses is provided in Appendix 4-A.

These figures show that the selected urban study areas represent the upper percentiles of the distributions of population and do not represent the locations with lower populations (urban study areas are all above the 90^{th} percentile of U.S. county populations). This is consistent with the objectives of our case study selection process, e.g. we are characterizing risk in areas that are likely to be experiencing excess risk due to O_3 levels above alternative standards. The urban study areas span the full range of seasonal average 8-hr daily maximum O_3 concentrations in monitored U.S. counties and the full distribution of O_3 risk coefficients across the cities included by Bell et al. (2004) and Zanobetti and Schwartz (2008). We have included the two cities with the highest risk coefficients found by Zanobetti and Schwartz (2008), New York City and Detroit. We have not included the two highest found by Bell et al. (2004), Albuquerque and Honolulu, but have included the 3^{rd} and 4^{th} highest, Atlanta and Boston. The urban study areas do not capture the upper end of the distribution of baseline all-cause and non-accidental mortality. The interpretation of this is that the case study risk estimates may not capture the additional risk that may exist in locations that have the highest baseline mortality rates.

Table 1.4 Data sources for O_3 risk-related attributes

Potential risk determinant	Metric	Year	Source	Degree of national coverage
Demographics				
Age	Percent age 85 years and older	2005	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties
Age	Percent age 65 years and older	2005	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties
Age	Percent age 14 years and younger	2005	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties
Education	Population with less than high school diploma	2000	USDA/ERS, http://www.ers.usda.gov/Data/Education/	All counties
Unemployment	Percent unemployed	2005	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties
Income	Per capita personal income	2005	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties
Race	Percent nonwhite	2006	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties

Population	Total population	2008	Cumulative Estimates of Resident Population Change for the United States, States, Counties, Puerto Rico, and Puerto Rico Municipios: April 1, 2000 to July 1, 2008, Source: Population Division, U.S. Census Bureau	All counties
Population density	Population/square mile	2008	Cumulative Estimates of Resident Population Change for the United States, States, Counties, Puerto Rico, and Puerto Rico Municipios: April 1, 2000 to July 1, 2008, Source: Population Division, U.S. Census Bureau	All counties
Urbanicity	ERS Classification Code	2003	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties
Climate and Air Quality				
O_3 levels	Monitored 4th high 8-hr daily maximum	2007	EPA Air Quality System (AQS)	725 Monitored counties
O_3 levels	Seasonal mean 8-hr daily maximum	Avg. 2006-2008	AQS	671 Monitored counties
O_3 levels	Seasonal mean 1-hr daily maximum	Avg. 2006-2008	AQS	671 Monitored counties
O_3 levels	Seasonal mean	Avg. 2006-2008	AQS	671 Monitored counties
$PM_{2.5}$ levels	Monitored annual mean	2007	AQS	617 Monitored counties

Temperature	Mean July temp	1941-1970	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties
Relative Humidity	Mean July RH	1941-1970	County Characteristics, 2000-2007 Inter-university Consortium for Political and Social Research	All counties
Ventilation	Percent residences with no air conditioning	2004	American Housing Survey	76 cities
Baseline Health Conditions				
Baseline mortality	All Cause		CDC Wonder 1999-2005	All counties
Baseline mortality	Non Accidental		CDC Wonder 1999-2006	All counties
Baseline mortality	Cardiovascular		CDC Wonder 1999-2007	All counties
Baseline mortality	Respiratory		CDC Wonder 1999-2008	All counties
Baseline morbidity	Acute myocardial infarction prevalence	2007	Behavioral Risk Factor Surveillance System (BRFSS)	184 metropolitan statistical areas (MSA)
Baseline morbidity	Diabetes prevalence	2007	BRFSS	184 MSA
Baseline morbidity	Stroke prevalence	2007	BRFSS	184 MSA
Baseline morbidity	Congestive heart disease prevalence	2007	BRFSS	184 MSA
Obesity	Body Mass Index	2007	BRFSS	184 MSA
Level of exercise	Vigorous activity 20 minutes	2007	BRFSS	184 MSA
Level of exercise	Moderate activity 30 minutes or vigorous activity 20 minutes	2007	BRFSS	184 MSA

Respiratory risk factors	Current asthma	2007	BRFSS	184 MSA
Smoking	Ever smoked	2007	BRFSS	184 MSA
C-R Estimates				
Mortality risk	Non Accidental	2004	Bell et al. (2004)	95 cities
Mortality risk	All Cause	2008	Zanobetti and Schwartz (2008)	48 cities
Mortality risk	Cardiovascular	2008	Zanobetti and Schwartz (2008)	48 cities
Mortality risk	Respiratory	2008	Zanobetti and Schwartz (2008)	48 cities

Table 1.5 Summary statistics for selected O_3 risk-related attributes

Risk Attribute	Average		Standard Deviation		Maximum		Minimum		Sample Size (# of counties or cities)	
	Urban Study Areas	U.S. Dataset	Urban Study Areas	U.S. Dataset	Urban Study Areas	U.S. Dataset	Urban Study Areas	U.S. Dataset	Urban Study Areas	U.S. Dataset
Demographics										
Population	1,642,198	97,020	1,972,403	312,348	9,862,049	9,862,049	354,361	42	23	3143
Population density (Pop/sq mile)	10,378	258	16,550	1,757	71,758	71,758	1,313	0	23	3143
Median age (Years)	35.7	38.6	2.3	4.4	40.0	55.3	32.1	20.1	23	3141
% Age 0 to 14 years	20.7	19.0	2.4	2.9	24.6	36.8	14.7	0.0	23	3141
% Age 65+ years	11.3	14.9	2.5	4.1	15.2	34.7	5.8	2.3	23	3141
% Age 85+ years	1.7	2.1	0.6	0.9	2.5	7.7	0.5	0.1	23	3141
Unemployment rate (%)	5.7	5.4	1.2	1.8	8.6	20.9	4.1	1.9	23	3133
% with less than high school diploma	20.9	22.6	7.9	8.8	37.7	65.3	8.7	3.0	23	3141
Income ($)	40305	27367	14238	6604	93377	93377	23513	5148	23	3086
% Non-white	36.4	13.0	15.3	16.2	86.7	95.3	31.7	0.0	23	3141
% Commute by public transportation*	7.1	1.6	8.1	2.5	30.7	30.7	1.5	0.0	12	366
Health Conditions										
Prevalence of CHD (%) *	3.6	4.3	0.8	1.3	4.6	8.7	2.6	1.8	11	184
Prevalence of asthma (%) *	8.5	8.1	1.3	1.9	11.2	13.2	6.0	3.6	11	184
Prevalence of diabetes (%) *	8.1	8.5	1.2	2.1	10.6	16.5	5.4	2.2	11	184
Prevalence of AMI (%) *	3.6	4.1	0.6	1.3	4.8	10.2	2.8	1.7	11	184
Prevalence of obesity (%) *	24.7	26.0	4.0	4.1	32.7	35.7	18.7	14.0	11	182
Prevalence of stroke (%) *	2.6	2.7	0.7	1.0	3.7	6.5	1.5	0.7	11	184
Prevalence of ever smoked (%)*	18.3	19.6	3.1	4.0	23.1	34.4	14.2	6.5	11	184
Prevalence of exercise (20 minutes, %)*	29.5	28.0	2.7	4.8	33.8	44.1	23.7	15.4	11	183
Prevalence of exercise (30 minutes,%)*	50.2	49.7	2.3	5.4	55.3	67.1	47.4	37.3	11	182
Non-accidental mortality (deaths per 100,000 people)	756.2	950.6	204.1	249.6	1139.5	1958.4	361.6	117.7	23	3142

All cause mortality (deaths per 100,000 people)	810.1	1022.3	217.4	258.6	1257.8	2064.2	402.5	176.8	23	3142
Cardiovascular mortality (deaths per 100,000 people)	310.5	392.1	93.9	121.0	459.6	970.4	122.4	37.5	23	3142
Respiratory mortality (deaths per 100,000 people)	66.2	97.3	17.0	32.3	90.1	351.0	34.8	13.3	23	3136
Air Quality and Climate										
O_3 4th high maximum 8-hr average (ppb)	0.087	0.077	0.009	0.010	0.105	0.126	0.072	0.033	23	725
O_3 seasonal mean (ppb)	33.9	34.5	5.4	6.6	51.0	64.8	25.8	8.6	22	671
O_3 seasonal mean of maximum 8-hr average (ppb)	50.7	48.6	7.5	7.2	70.2	79.7	40.8	13.3	22	671
O_3 seasonal mean of 1-hr daily maximum (ppb)	58.8	54.7	7.5	8.0	85.1	92.4	46.5	17.6	22	671
$PM_{2.5}$ annual mean (µg/m3)	14.1	11.7	2.6	3.1	16.9	22.5	8.4	3.4	23	617
$PM_{2.5}$ 98th %ile daily average (µg/m3)	35.8	30.7	8.1	9.3	59.0	81.1	21.2	9.1	23	617
Average temperature (°F)	57.2	57.2	5.0	7.9	70.3	76.2	50.1	39.0	23	202
July temperature long term average (°F)	76.0	75.9	3.4	5.4	83.3	93.7	68.5	55.5	23	3104
July Relative Humidity long term average (%)	61.5	56.2	10.2	14.6	70.0	80.0	28.0	14.0	23	3104
% No air conditioning*	15.5	16.6	85.7	79.1	42.9	86.7	0.4	0.0	12	76
C-R Estimates										
Non-accidental mortality O_3 risk*	0.000515	0.000423	0.000138	0.000133	0.000705	0.000940	0.000331	0.000088	12	95
All Cause mortality O_3 risk*	0.000627	0.000527	0.000314	0.000205	0.001092	0.001092	0.000163	0.000096	12	48
Respiratory mortality O_3 risk*	0.000877	0.000800	0.000282	0.000186	0.001424	0.001424	0.000307	0.000307	12	48
Cardiovascular mortality O_3 risk*	0.000898	0.000825	0.000173	0.000124	0.001064	0.001064	0.000418	0.000418	12	48

*Attribute for which only city-specific data were available

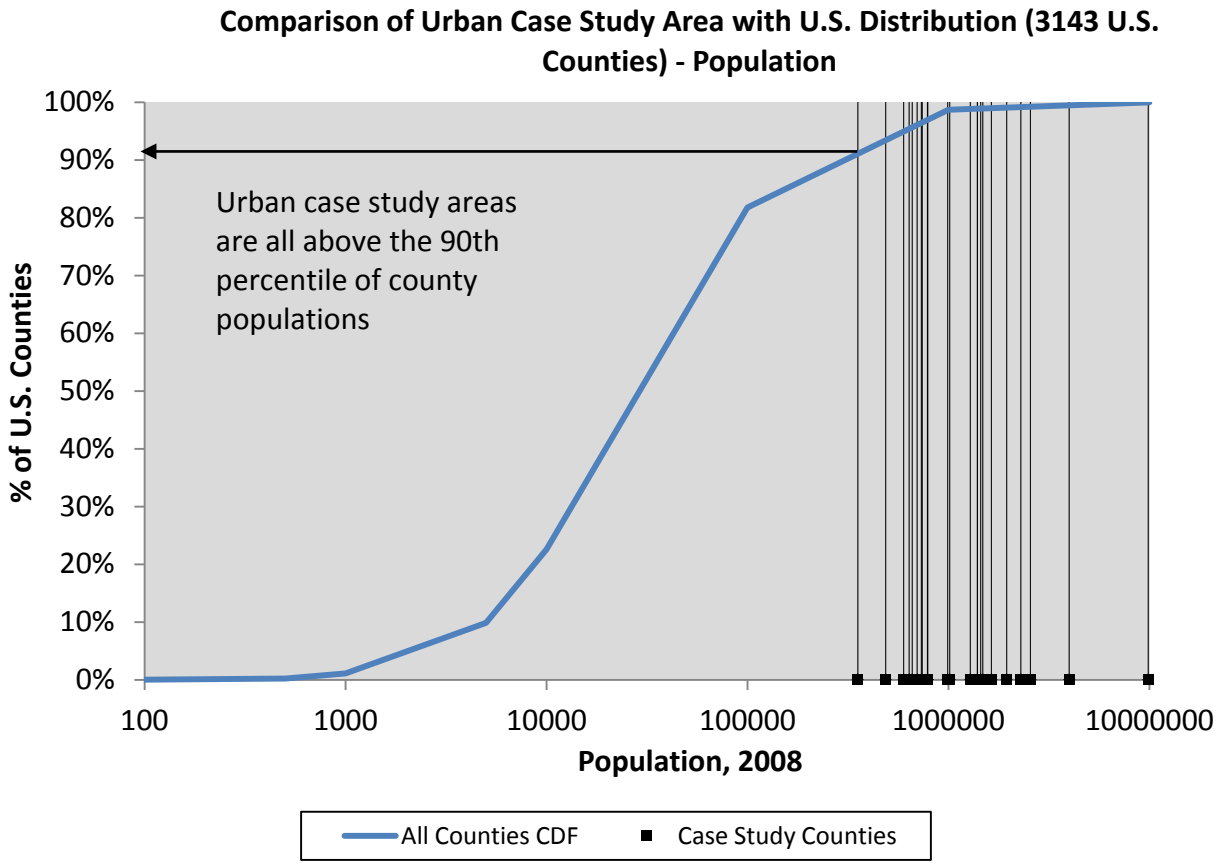

Figure 1.13 Comparison of distributions for key elements of the risk equation: Total population.

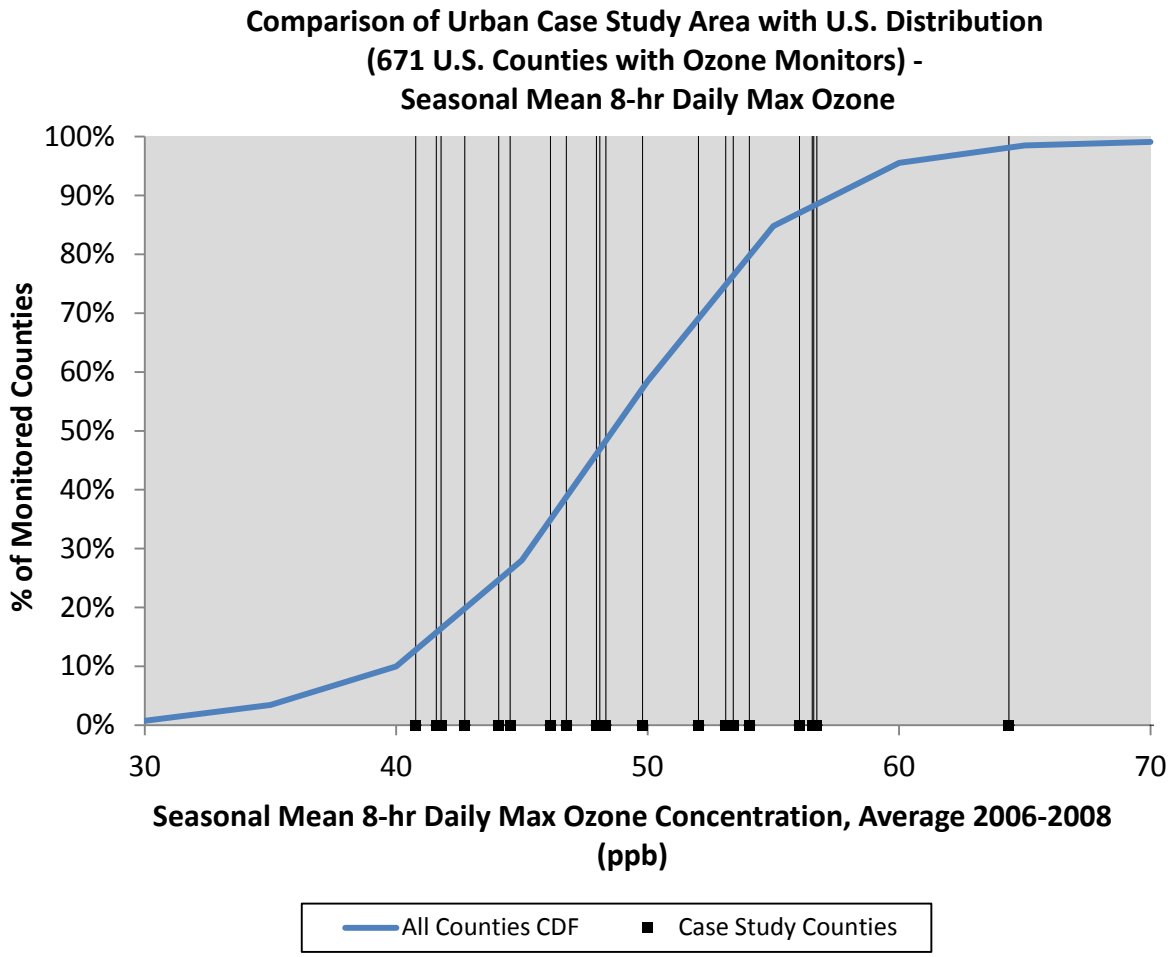

Figure 1.14 Comparison of distributions for key elements of the risk equation: Seasonal mean 8-hr daily maximum O_3 concentration.

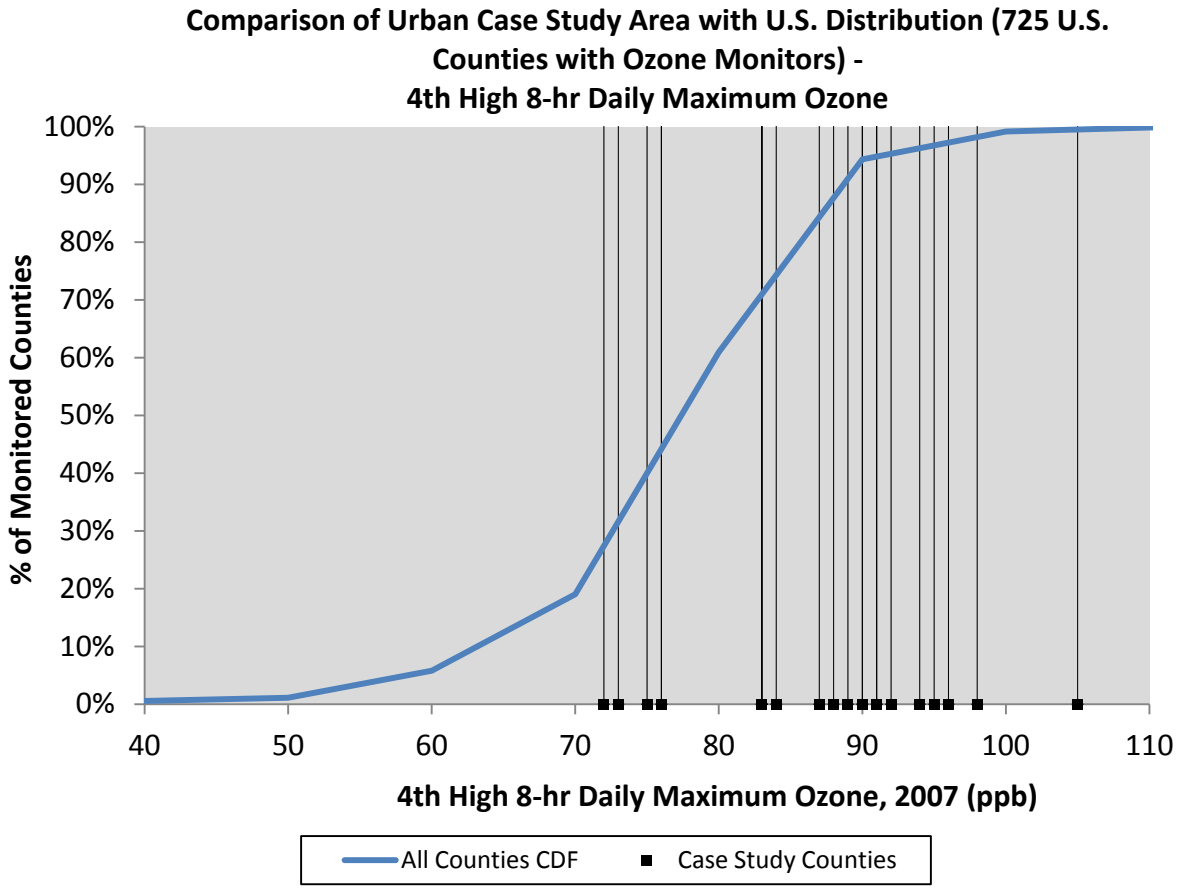

Figure 1.15 Comparison of distributions for key elements of the risk equation: 4th highest 8-hr daily maximum O_3 concentration.

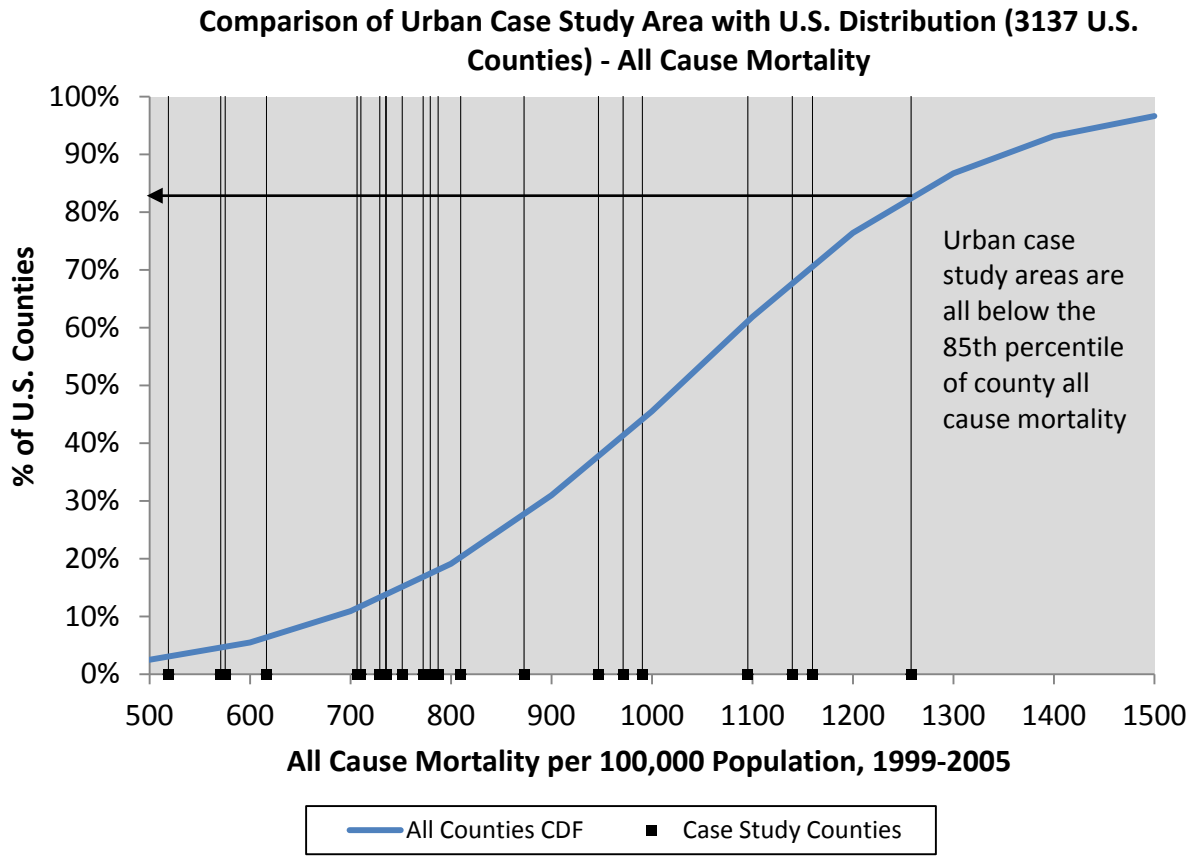

Figure 1.16 Comparison of distributions for key elements of the risk equation: Baseline all-cause mortality rate.

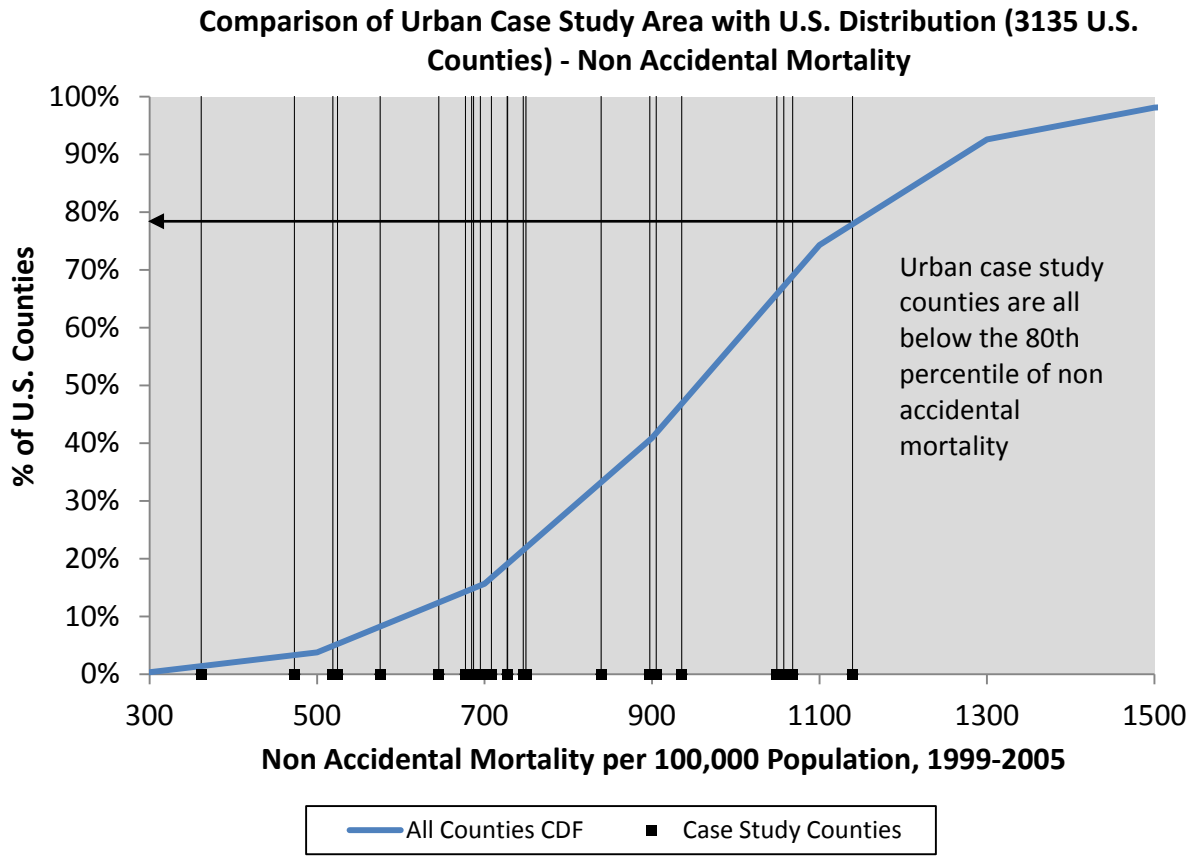

Figure 1.17 Comparison of distributions for key elements of the risk equation: Baseline non-accidental mortality rate.

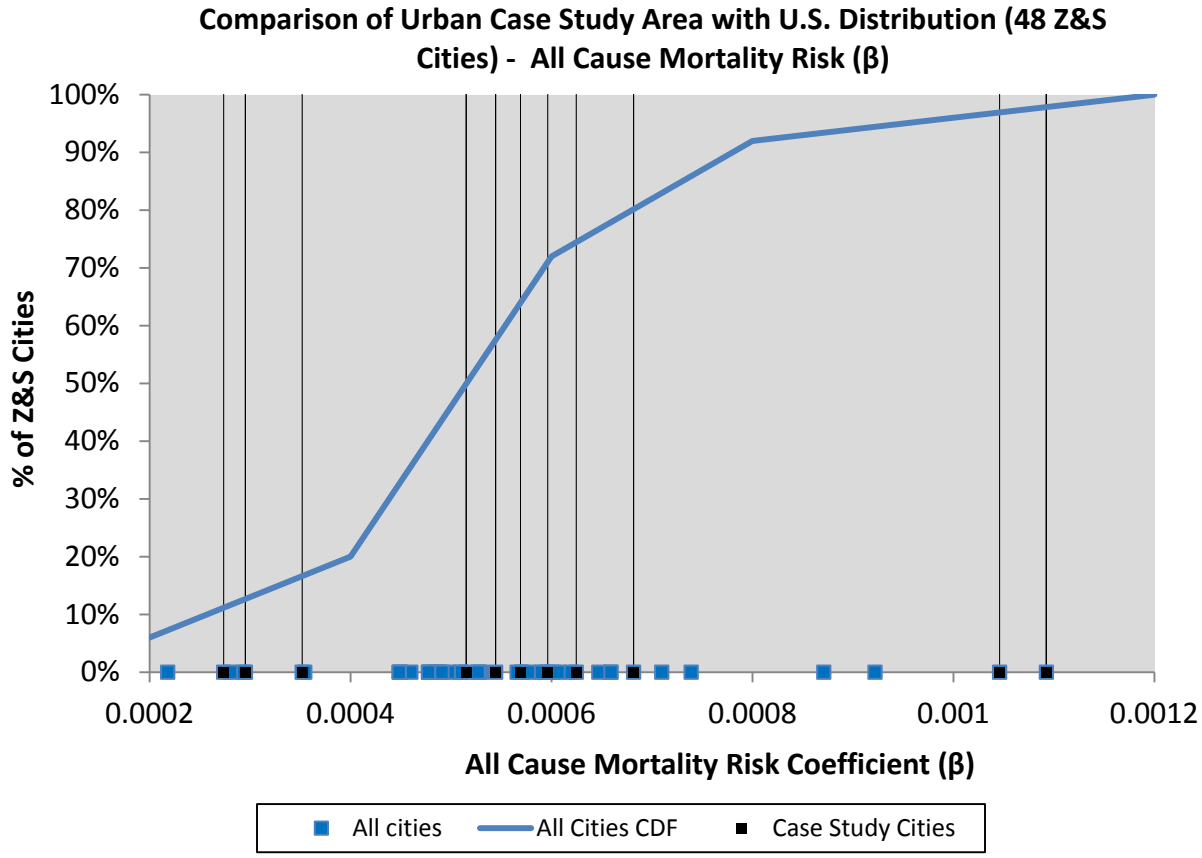

Figure 1.18 Comparison of distributions for key elements of the risk equation: All-cause mortality risk coefficient from Zanobetti and Schwartz (2008).

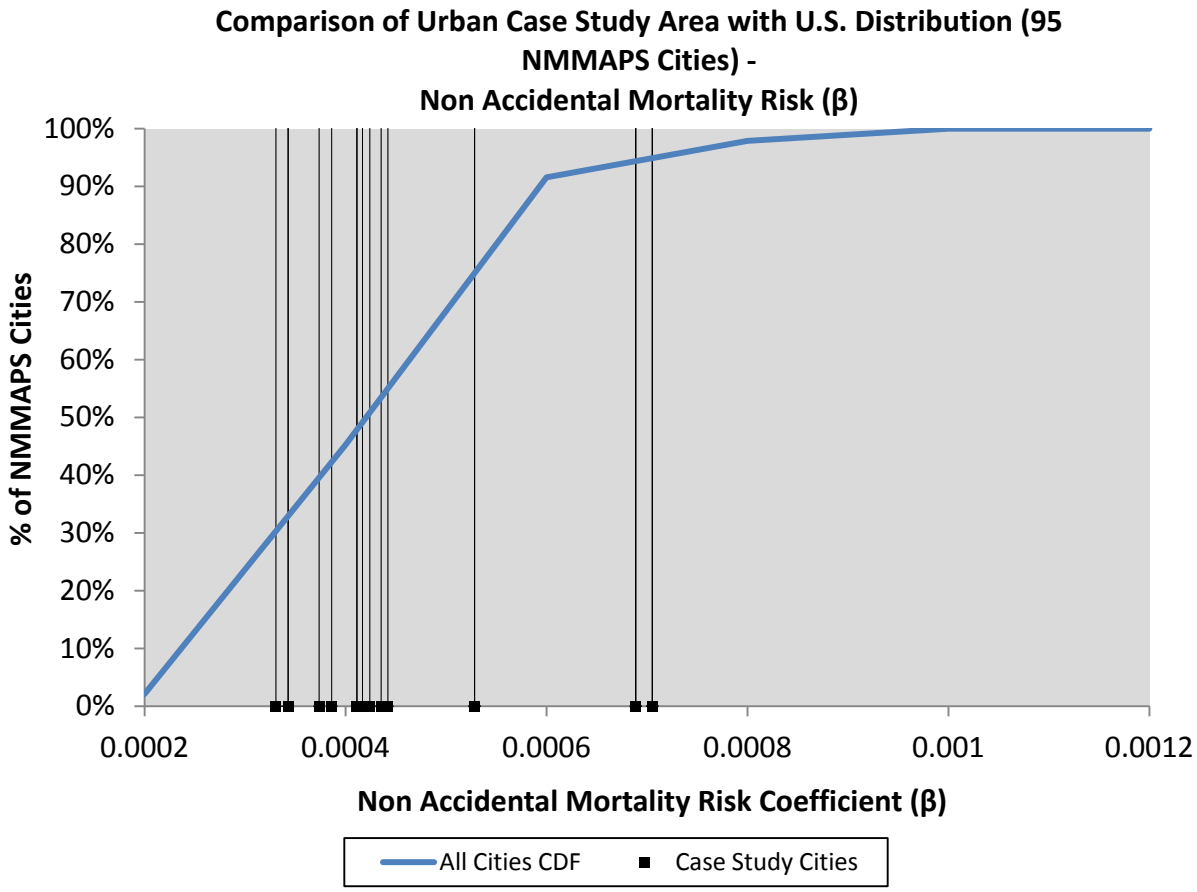

Figure 1.19 Comparison of distributions for key elements of the risk equation: Non-accidental mortality risk coefficient from Bell et al. (2004).

Figure 1.20 through Figure 1.25 show national CDFs and the urban study area values for several selected potential risk attributes. These potential risk attributes do not directly enter the risk equations, but have been identified in the literature as potentially affecting the magnitude of the O_3 C-R functions reported in the epidemiological literature. Comparison graphs for other risk attributes are provided in Appendix 4-A. The selected urban study areas do not capture the higher end percentiles of several risk characteristics, including populations 65 years and older, baseline cardiovascular disease prevalence, baseline respiratory disease prevalence, and smoking prevalence. Summarizing the analyses of the other risk attributes, we conclude that the urban study areas provide adequate coverage across population, population density, O_3 levels (seasonal mean, seasonal mean 8-hr daily maximum, and seasonal mean 1-hr daily maximum), $PM_{2.5}$ co-pollutant levels, temperature and relative humidity, unemployment rates, percent non-white population, asthma prevalence obesity prevalence, income, and less than high school education. We also conclude that while the urban study areas cover a wide portion of the distributions, they do not provide coverage for the upper end of the distributions of percent of population 65 and

older (below 60th percentile), percent of population 85 years and older (below 75th percentile), prevalence of angina/coronary heart disease (below 70th percentile), prevalence of diabetes (below 85th percentile), stroke prevalence (below 90th percentile), prevalence of heart attack (below 80th percentile), prevalence of smoking (below 85th percentile), all-cause mortality rates (below 85th percentile), non-accidental mortality rates (below 80th percentile), cardiovascular mortality rates (below 75th percentile) and respiratory mortality rates (below 50th percentile), and percent of residences without air conditioning (below 90th percentile). In addition, the urban study areas do not capture the highest or lowest ends of the distribution of exercise prevalence and do not capture the low end of the distribution of public transportation use (above the 65th percentile).

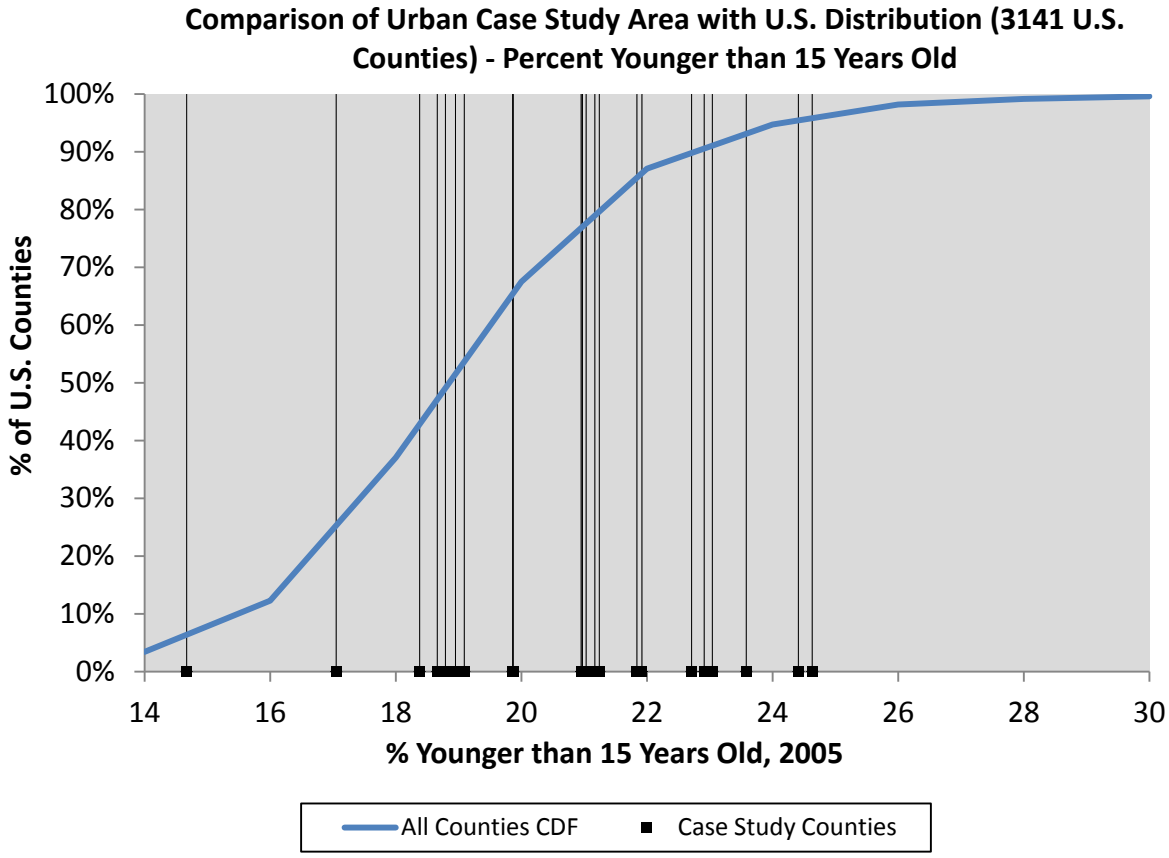

Figure 1.20 Comparison of distributions for selected variables expected to influence the relative risk from O_3: Percent of population younger than 15 years old.

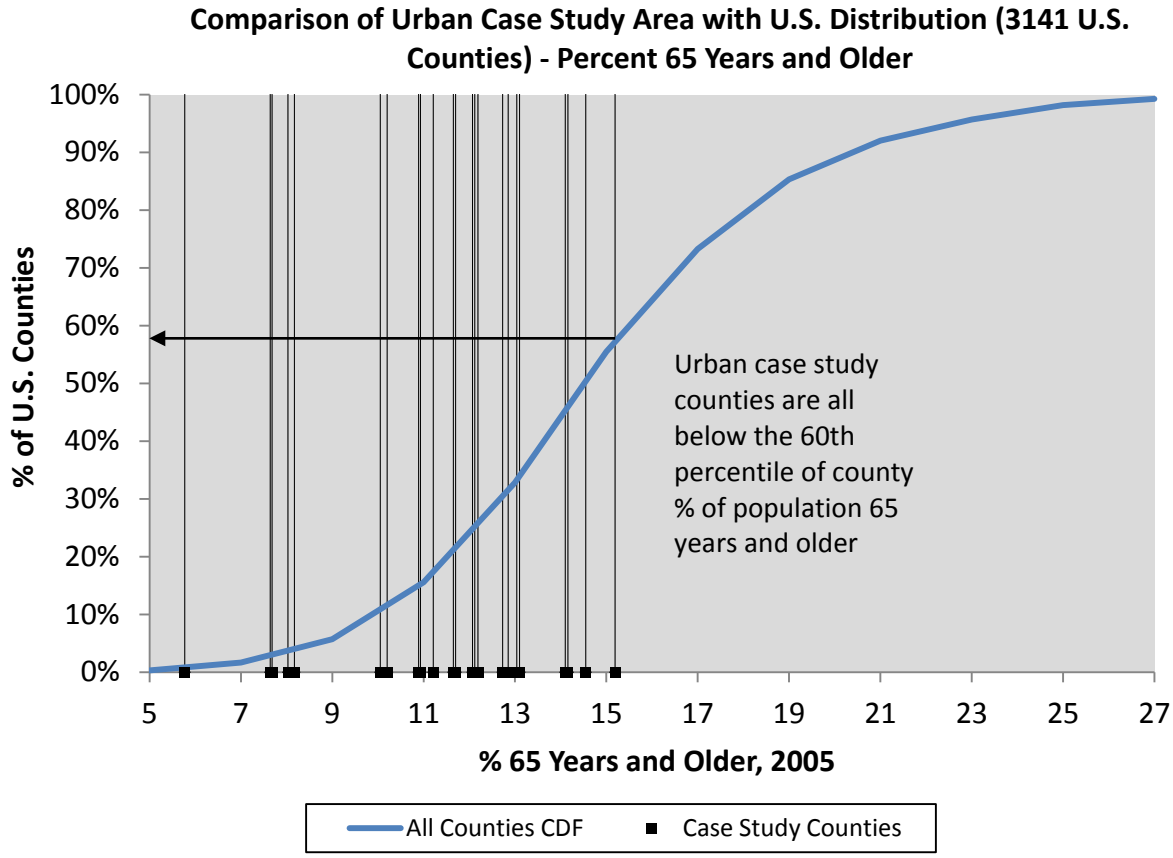

Figure 1.21 Comparison of distributions for selected variables expected to influence the relative risk from O_3: Percent of population age 65 years and older.

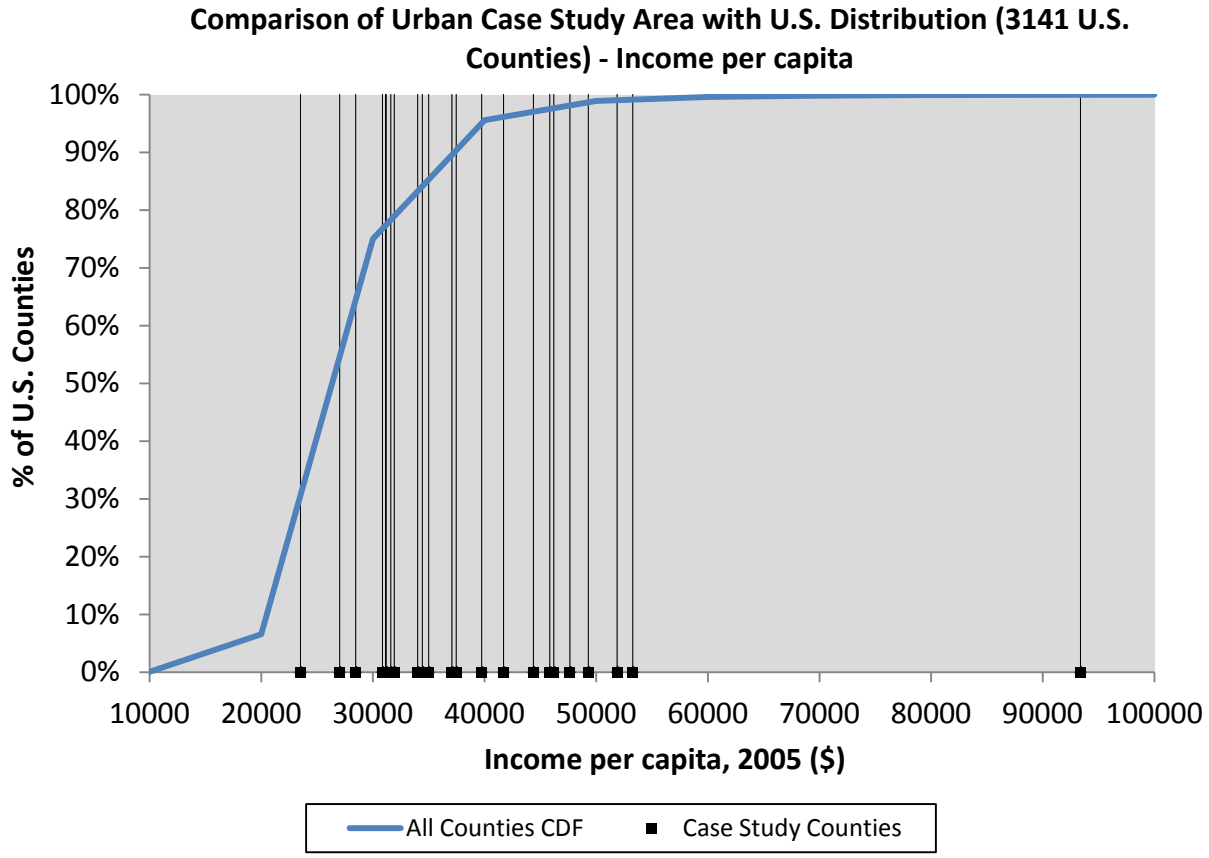

Figure 1.22 Comparison of distributions for selected variables expected to influence the relative risk from O_3: Income per capita.

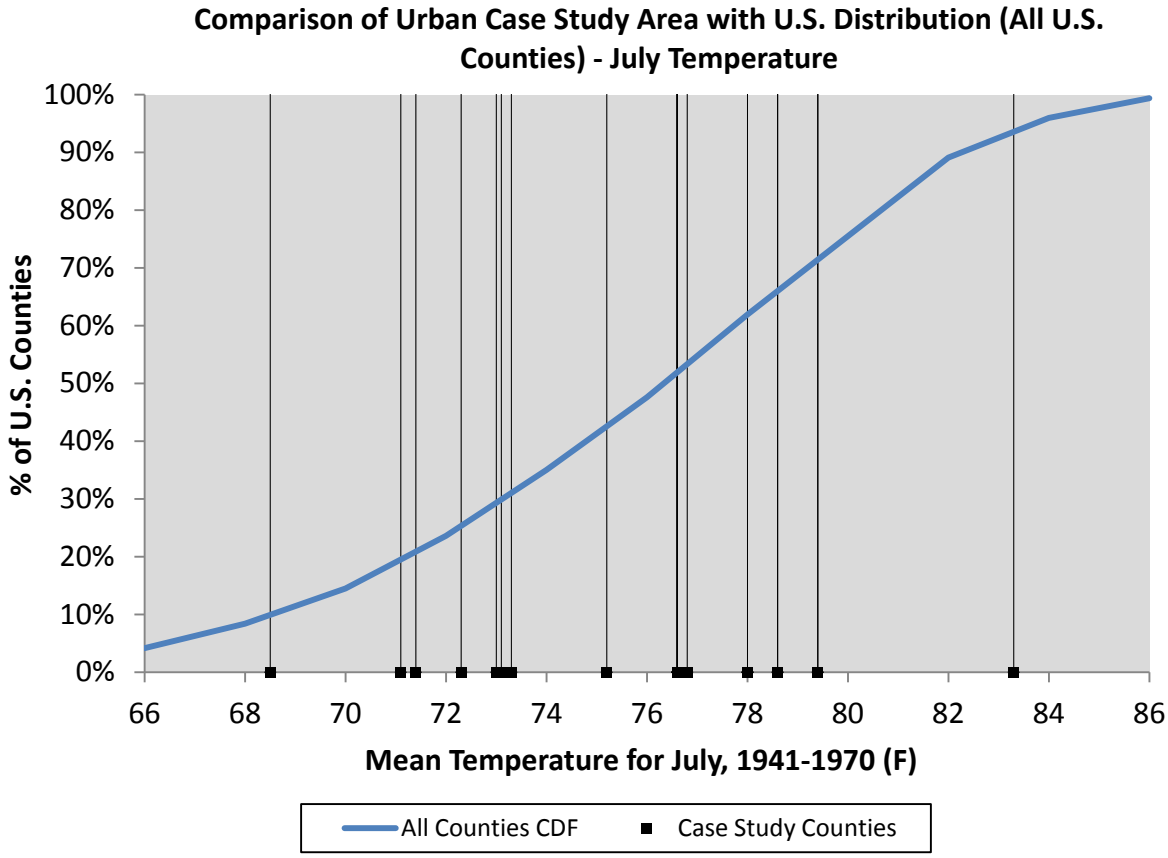

Figure 1.23 Comparison of distributions for selected variables expected to influence the relative risk from O_3: July temperature.

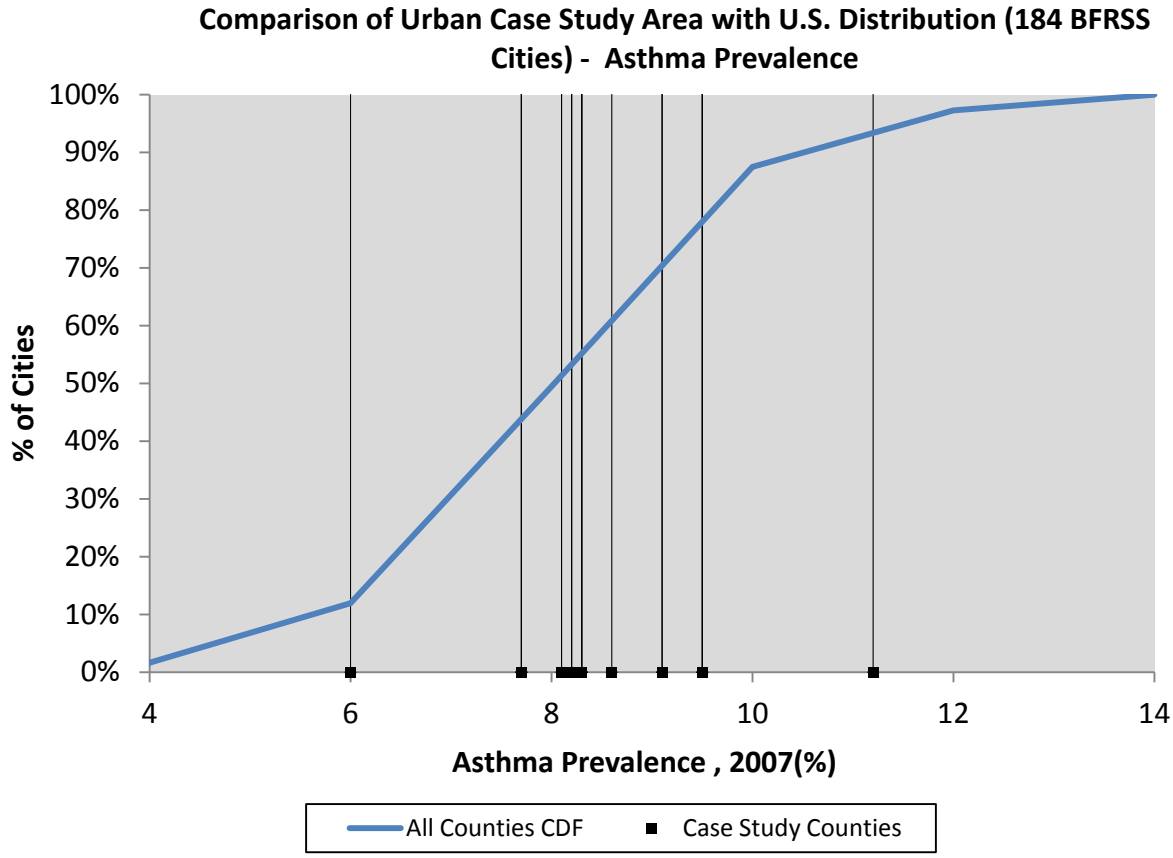

Figure 1.24 Comparison of distributions for selected variables expected to influence the relative risk from O_3: Asthma prevalence.

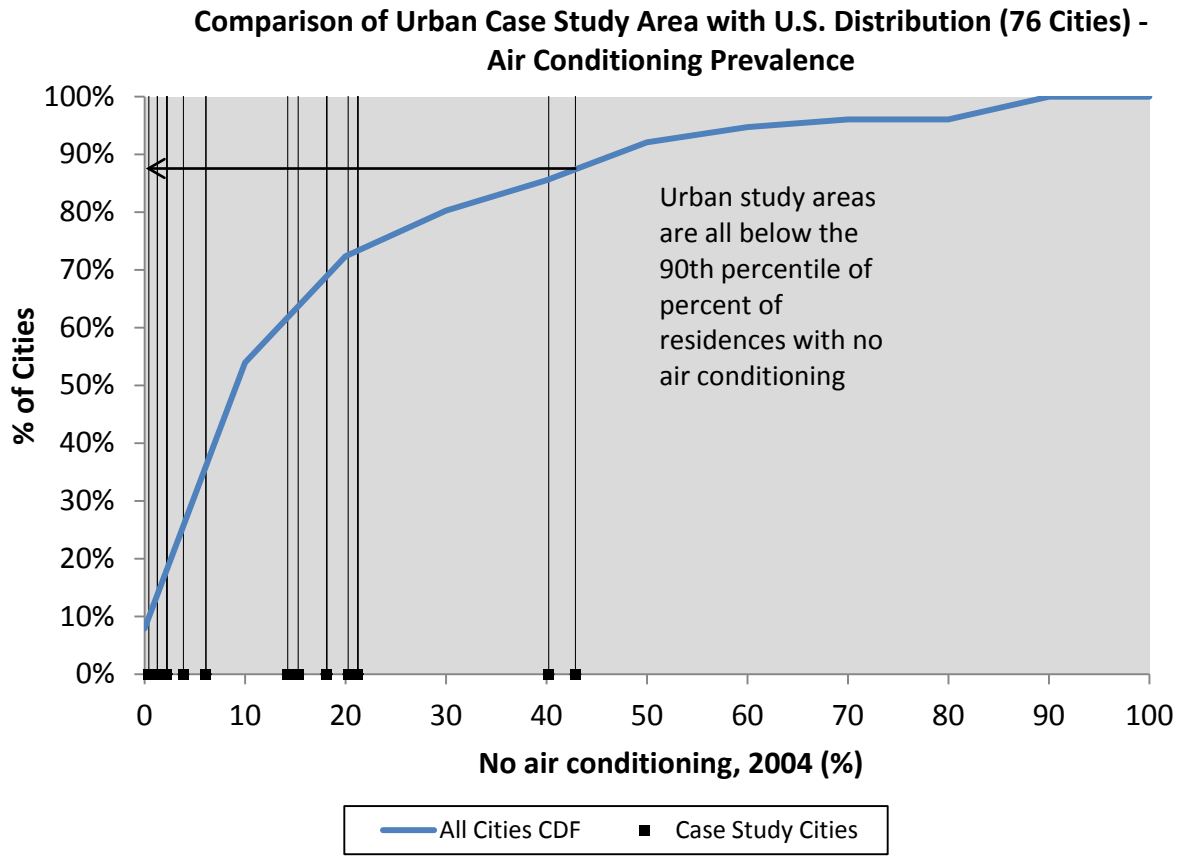

Figure 1.25 Comparison of distributions for selected variables expected to influence the relative risk from O_3: Air conditioning prevalence.

Based on the above analyses, we can draw several inferences regarding the representativeness of the urban case studies. First, the case studies represent urban areas that are among the most populated in the U.S. Second, they represent areas with relatively high levels of O_3 (4^{th} high 8-hr daily maximum, seasonal mean 8-hr daily maximum, seasonal mean 1-hr daily maximum, and seasonal mean). Third, they capture well the range of city-specific effect estimates found by Bell et al. (2004) and Zanobetti and Schwartz (2008) studies. These three factors would suggest that the urban study areas should capture well overall risk for the nation, with a potential for better characterization of the high end of the risk distribution. However, there are several other factors that suggest that the urban study areas may not be representing areas that may have a high risk per ppb of O_3. The analysis suggests that the urban study areas are not capturing areas with the highest baseline mortality rates nor those with the oldest populations. These areas may have higher risks per ppb of O_3, and thus the high end of the risk distribution may not be captured. However, the impact on characterization of overall O_3 risk may not be as large, since overall O_3 risk depends on a combination of factors, including O_3 levels and total population, in addition to age distribution and baseline mortality rates.

It should be noted that several of the factors with underrepresented tails, including age and baseline mortality are spatially correlated (R=0.81), so that certain counties which have high proportions of older adults also have high baseline mortality and high prevalence of underlying chronic health conditions. Because of this, omission of certain urban areas with higher percentages of older populations, for example, cities in Florida, may lead to underrepresentation of high risk populations. However, with the exception of areas in Florida, most locations with high percentages of older populations have low overall populations, less than 50,000 people in a county. And even in Florida, the counties with the highest O_3 levels do not have a high percent of older populations. This suggests that while the risk per exposed person per ppb of O_3 may be higher in these locations, the overall risk to the population is likely to be within the range of risks represented by the urban case study locations.

The urban study areas also do not capture the highest end of percent of residences without air conditioning. If the cities with the lowest air conditioning prevalence also have high O_3 levels, we could be missing a high risk portion of the population that is exposed to O_3 indoors as air infiltrates indoors from outdoors. However, 4th highest 8-hr daily maximum O_3 levels in the cities in the top 10th percentile of percentage of residences without air conditioning (mainly in northern California and Washington) are approximately average (0.08 ppm) or lower than average. The relatively low O_3 concentrations in these areas with low air conditioning prevalence suggests that we are not excluding a high risk population that has both low air conditioning prevalence and high O_3 concentrations, and the overall risk to the population is likely to be within the range of risks represented by the urban case study locations.

There is no nationally representative data base that will allow us to compare the time spent outdoors among persons residing in each of the urban case study areas. As time spent outdoors is an important personal attribute that influences exposure to O_3 (US EPA, 2007), EPA staff is considering evaluating data from the American Time Use Survey (ATUS) for the 2nd draft REA. ATUS is a recent (2003-2011) nationally representative survey that contains information on people's time expenditure, many of whom reside in the urban case study areas modeled in this assessment. ATUS does however have a few noteworthy limitations: (1) there are no survey participants under 15 years of age, (2) time spent at home locations is neither distinguished as indoors or outdoors, (3) missing or unknown location data can comprise a significant portion of a persons' day (on average, about 40% (George and McCurdy, 2009)), (4) only a single day is available for each participant, and (5) influential meteorological conditions affecting time expenditure were not recorded (e.g., daily temperature and precipitation (Graham and McCurdy, 2004)). To overcome a few of the ATUS limitations, EPA staff is planning to (1) use particular activity codes (e.g., participation in a sport) to better approximate outdoor time expenditure, (2) link National Climatic Data Center (NCDC) meteorological data to each ATUS

diary, and (3) control for diaries having significant missing or unknown location information to allow for a relative comparison of outdoor time across the urban case study areas.

8.2.2 Analysis Based on Consideration of National Distribution of O_3-Related Mortality Risk

In this section we discuss the second representativeness analysis which identified where the counties comprising the 12 urban study areas fall along a distribution of estimated national-scale mortality risk. This assessment reveals whether the baseline O_3 mortality risks in the 12 urban case study areas represent more typical or higher end risk relative to the national risk distribution (see Section 7.3). For ease of comparison, we use only the estimates of mortality associated with total O_3 (i.e. no concentration cutoff). Applying a concentration cutoff is unlikely to change the conclusions of this assessment.

The results of this representativeness analysis are presented graphically in Figure 1.26 and Figure 1.27, which display the cumulative distribution of total mortality attributable to ambient O_3 at the county level developed as part of the national-scale analysis (see Figure 1.9). Values for the 23 counties included in the urban case study analysis are then superimposed on top of the cumulative distribution to assess the representativeness of the urban case study areas. For the results based on Bell et al. (2004) effect estimates, Atlanta and Boston have the highest percentage of total mortality attributable to ambient O_3 of the 12 urban study areas and are located at the highest end of the distribution of U.S. O_3-related mortality risk. Of the 12 urban study areas, these two cities had the highest effect estimates found by Bell et al. (2004; See Appendix 4-A). Overall, O_3 mortality risk in the 12 urban study areas are representative of the full distribution of U.S. O_3-related mortality risk, with the percentage of total mortality attributable to O_3 ranging from 1.4% to 3.6%, assuming no concentration cutoff.

For the results based on Zanobetti and Schwartz (2008) effect estimates, Detroit and New York City are at the very highest end of the U.S. distribution of county-level risk of mortality due to ambient O_3. These two cities had the highest effect estimates of the 48 cities included in the study (see Appendix 4-A). For this study, Houston and Los Angeles had the lowest risk and were located at the very lowest end of the U.S. distribution of county-level risk of mortality due to ambient O_3. These two cities had the lowest effect estimates found by Zanobetti and Schwartz (2008). The low effect estimates in Houston and Los Angeles could be due to several factors. Both cities cover a large spatial extent and have high rates of time spent driving, possibly leading to exposure misclassification in the underlying epidemiologic study. Houston also has a very high rate of air conditioning use (nearly 100% of residences) and Los Angeles has been shown to have high rates of adaptive behavior on high ambient O_3 days (i.e. more time spent indoors as a

result of high ambient O_3 concentrations; Neidell 2009, 2010), both of which would lead to lower personal O_3 exposure relative to other cities. Overall, O_3 mortality risk in the 12 urban study areas are representative of the full distribution of U.S. O_3-related mortality risk, with the percentage of total mortality attributable to O_3 ranging from 0.6% to 4.8%, assuming no concentration cutoff.

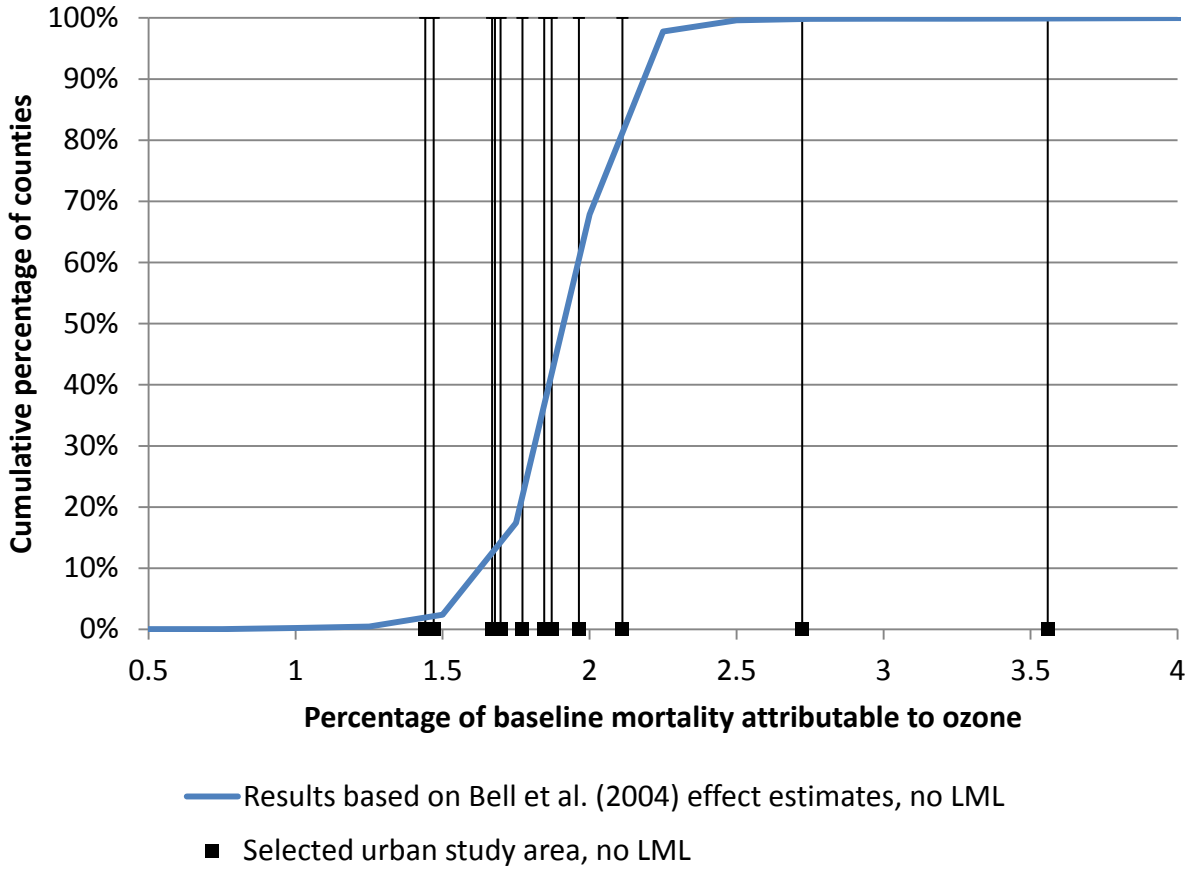

Figure 1.26. Cumulative distribution of county-level percentage of total non-accidental mortality attributable to 2006-2008 average O_3 for the U.S. and the locations of the selected urban study areas along the distribution, using Bell et al. (2004) effect estimates.

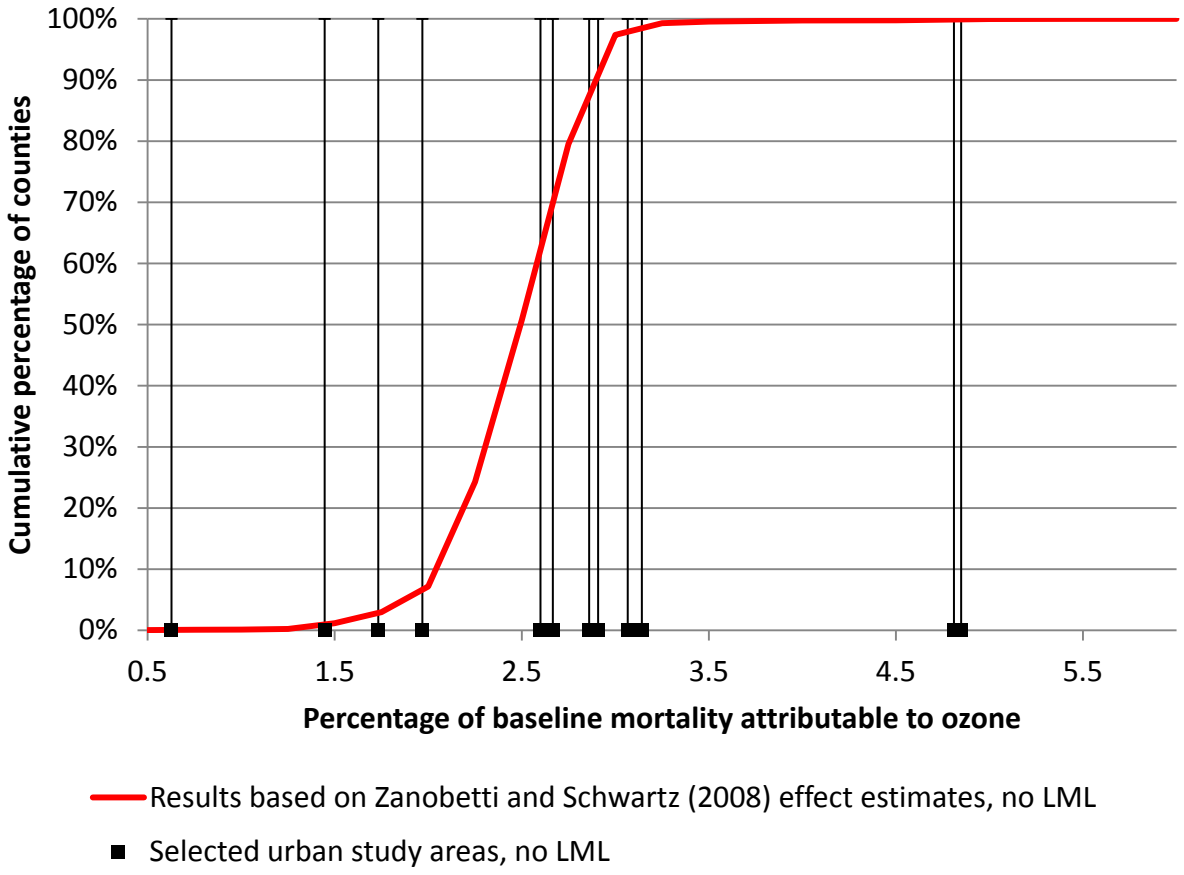

Figure 1.27. Cumulative distribution of county-level percentage of total all-cause mortality attributable to 2006-2008 average O_3 for the U.S. and the locations of the selected urban study areas along the distribution, using Zanobetti and Schwartz (2008) effect estimates.

8.2.3 Discussion

We conducted two analyses to assess the representativeness of the 12 urban study areas examined in Section 7.2 in the national context. First, we assessed the degree to which the urban study areas represent the range of key O_3 risk-related attributes that spatially vary across the nation. We examined both the specific elements of our risk assessment framework (population, baseline incidence rates, air quality, and the coefficient relating air quality and the health outcome) in addition to factors that have been identified as influential in determining the magnitude of the C-R function across locations (demographics, baseline heath conditions, and climate and air quality attributes). The second representativeness analysis, which is discussed in Section 7.4.2, identified where the 12 urban study areas fall along the distribution of national county-level O_3-attributable mortality risk. This analysis allowed us to assess the degree of which the 12 urban study areas capture locations within the U.S. likely to experience elevated levels of risk related to O_3 exposure.

We observe that the 23 counties for the 12 urban study areas considered in Section 7.2 capture urban areas that are among the most populated in the U.S., have relatively high O_3 levels, and represent the range of city-specific effect estimates found by Bell et al. (2004) and Zanobetti and Schwartz (2008). These three factors suggest that the urban study areas capture overall risk for the nation well, with a potential for better characterization of the high end of the risk distribution. We find that the urban study areas are not capturing areas with the highest baseline mortality rates, those with the oldest populations, and those with the lowest air conditioning prevalence. These areas tend to have relatively low O_3 concentrations and low total population, suggesting that the urban study areas are not missing high risk populations that have high O_3 concentrations in addition to greater susceptibility per unit O_3. The second representativeness analysis demonstrated that the 12 urban study areas represent the full range of county-level O_3-related risk across the entire U.S. We conclude from these analyses that the 12 urban study areas adequately represent O_3-related risk across the U.S.

8.3 REFERENCES

Abt Associates, Inc. (2010). Model Attainment Test Software (Version 2). Bethesda, MD. Prepared for the U.S. Environmental Protection Agency Office of Air Quality Planning and Standards. Research Triangle Park, NC. Available on the Internet at: http://www.epa.gov/scram001/modelingapps.mats.htm.

Abt Associates, Inc. (2010). Environmental Benefits and Mapping Program (Version 4.0). Bethesda, MD. Prepared for U.S. Environmental Protection Agency Office of Air Quality Planning and Standards. Research Triangle Park, NC. Available on the Internet at <http://www.epa.gov/air/benmap>.

Anenberg, S.C., J.J. West, L.W. Horowitz, D.Q. Tong. (2010). An estimate of the global burden of anthropogenic O_3 and fine particulate matter on premature human mortality using atmospheric modeling. Environ Health Perspect, 118:1189-1195.

Anenberg, S.C., J.J. West, L.W. Horowitz, D.Q. Tong. (2011). The global burden of air pollution mortality: Anenberg et al. respond. Environ Health Perspect, 119:A158-A425.

Bell, M.L., A. McDermott, S.L. Zeger, J.M. Samet, F. Dominici. (2004). O_3 and short-term mortality in 95 US urban communities, 1987-2000. JAMA, 292:2372-2378.

Byun, D., and K.L. Schere. (2006). Review of Governing Equations, Computational Algorithms, and Other Components of the Models-3 Community Multiscale Air Quality (CMAQ) Modeling System. Applied Mechanics Reviews, 59:51-77.

Centers for Disease Control: Wide-ranging OnLine Data for Epidemiological Research (CDC-Wonder) (data from years 2004-2006), Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Available on the Internet at http://wonder.cdc.gov.

Fann N, Lamson AD, Anenberg SC, Wesson K, Risley D, Hubbell BJ. (2012). Estimating the national public health burden associated with exposure to ambient $PM_{2.5}$ and O_3. Risk Analysis, 32:81-95.

George BJ and McCurdy T. (2009). Investigating the American Time Use Survey from an exposure modeling perspective. JESEE. 21:92-105.

Graham S and McCurdy T. (2004). Developing meaningful cohorts for human exposure models. JEAEE. 14:23-43.

Hollman, F.W., T.J. Mulder, and J.E. Kallan. (2000). Methodology and Assumptions for the Population Projections of the United States: 1999 to 2100. Population Division Working Paper No. 38, Population Projections Branch, Population Division, U.S. Census Bureau, Department of Commerce.

Jerrett, M., R.T. Burnett, C.A. Pope III, K. Ito, G. Thurston, D. Krewski, Y. Shi, E. Calle, M. Thun. (2009). Long-term O_3 exposure and mortality. N. Eng. J. Med., 360:1085-1095.

Neidell M. (2009). Information, avoidance behavior and health. J Human Res. 44:450-478.

Neidell M. (2010). Air quality warnings and outdoor activities: evidence from Southern California using a regression discontinuity approach design. J Epidemiol Community Health. 64:921-926.

Timin B, Wesson K, Thurman J. Application of Model and Ambient Data Fusion Techniques to Predict Current and Future Year $PM_{2.5}$ Concentrations in Unmonitored Areas. (2010). Pp. 175-179 in Steyn DG, Rao St (eds). Air Pollution Modeling and Its Application XX. Netherlands: Springer.

U.S. Environmental Protection Agency. (2012a). Integrated Science Assessment for O_3 and Related Photochemical Oxidants: Third External Review Draft, U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. Environmental Protection Agency. (2010). Quantitative Health Risk Assessment for Particulate Matter, U.S. Environmental Protection Agency, Research Triangle Park, NC, EPA-452/R-10-005.

U.S. Environmental Protection Agency. (2009). Integrated Science Assessment for Particulate Matter, U.S. Environmental Protection Agency, Research Triangle Park, NC, EPA/600/R-08/139F.

US EPA. (2007). O_3 Population Exposure Analysis for Selected Urban Areas. U.S. Environmental Protection Agency, Research Triangle Park, NC, USA, (EPA-452/R-07-010). Available at: http://www.epa.gov/ttn/naaqs/standards/O_3/data/2007_07_O_3_exposure_tsd.pdf

Wells, B., Wesson, K., Jenkins, S. (2012). Analysis of Recent U.S. Ozone Air Quality Data to Support the O3 NAAQS Review and Quadratic Rollback Simulations to Support the First Draft of the Risk and Exposure Assessment. Available on the Internet at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_rea.html

Woods and Poole Inc. (2008). Population by Single Year of Age CD. CD-ROM. Woods and Poole Economics, Inc.

Zanobetti, A., and J. Schwartz. (2008). Mortality displacement in the association of O_3 with mortality: An analysis of 48 cities in the United States. Am J Resp Crit Care Med, 177:184-189.

Zanobetti, A., and J. Schwartz. (2011). O_3 and survival in four cohorts with potentially predisposing diseases. Am J Resp Crit Care Med, 194:836-841.

Zhang, L., D.J. Jacob, N.V. Smith-Downey, D.A. Wood, D. Blewitt, C.C. Carouge, A. van Donkelaar, D.B. A. Jones, L.T. Murray, Y. Wang. (2011). Improved estimate of the policy-relevant background O_3 in the United States using the GEOS-Chem global model with 1/2°x2/3° horizontal resolution over North America. Atmos Environ, 45:6769-6776.

9 SYNTHESIS

This assessment has estimated exposures to O_3 and resulting health risks for both current O_3 levels and O_3 levels after simulating just meeting the current primary O_3 standard of 0.075 ppm for the 4th highest 8-hour daily maximum, averaged over 3 years. The results from these assessments will help inform consideration of the adequacy of the current O_3 standards in the first draft Policy Assessment.

The remaining sections of this chapter provide key observations regarding the exposure assessment (Section 9.1), lung function risk assessment (Section 9.2), epidemiology based risk assessment (Section 9.3), and a set of integrated findings providing insights drawn from evaluation of the full assessment (Section 9.4).

9.1 SUMMARY OF KEY RESULTS OF POPULATION EXPOSURE ASSESSMENT

The first draft population exposure assessment evaluated exposures to O_3 using the APEX exposure model for the general population, all school-aged children (ages 5-18), and asthmatic children, with a focus on populations engaged in moderate or greater exertion, for example, children engaged in outdoor recreational activities. The strong emphasis on children reflected the finding of the last O_3 NAAQS review (EPA, 2007) and the ISA (EPA, 2012, Chapter 8) that children are an important at-risk group. Children breathe more air per pound of body weight, are more likely than adults to have asthma, and their lungs continue to develop until they are fully grown.

In this first draft, exposure is assessed for 4 cities, Atlanta, Denver, Los Angeles, and Philadelphia, for recent air quality (2006-2010) and for air quality simulated to just meet the current standard. The analysis provided estimates of the percent of children exposed to concentrations above three health-relevant 8-hour average O_3 exposure benchmarks: 0.060, 0.070, and 0.080 ppm. The ISA includes studies showing significant effects at each of these benchmark levels (U.S. EPA, 2012). These benchmarks were selected so as to provide some perspective on the public health impacts of O_3-related health effects that have been demonstrated in human clinical and toxicological studies, but cannot currently be evaluated in quantitative risk assessments, such as lung inflammation and increased airway responsiveness. In addition, the first draft exposure assessment also identified the specific microenvironments and activities most important for exposure and evaluated their duration and time of the day persons were engaged in them, with a focus on persons experiencing the highest daily maximum 8-hour exposure within each study area.

It should also be noted that with regard to the exposure estimates, the APEX model is not proficient at modeling activity patterns that lead to repeated exposures to elevated ozone

concentrations. As a result, while we are able to report the percent of children with at least one exposure greater than the alternative exposure benchmarks, we are not able to report with confidence the percent of children with more than one exposure. Children with repeated exposures may be at greater risk of significant health effects. In addition, we were only able to model exposure in four cities for this first draft assessment. It is likely that variation in exposure will be larger when we have modeled the full set of 16 cities in the second draft REA.

The key results of the first draft exposure assessment include:

- Exposure Assessment for Recent Conditions
  - The average (i.e., average across years 2006 to 2010) percentages of school age children estimated to experience one or more exposures per year to 8-hour O_3 concentrations at and above 0.060 ppm, while at moderate or greater exertion, were approximately 20% for Denver (corresponding to 109,000 children), 22% for Atlanta (corresponding to 189,000 children), 26% for Philadelphia (corresponding to 297,000 children), and 32% for Los Angeles (corresponding to 1,150,000 children). There was considerable variability in these percentages across the years evaluated, ranging from approximately 12 to 30% in Denver, 10 to 36% in Atlanta, 9 to 34% in Philadelphia, and 23 to 37% in Los Angeles. When considering exposures at and above 0.060 ppm in asthmatic children at moderate or greater exertion, the results were similar in term of percentages, corresponding to average numbers of exposed asthmatic children of approximately 10,000 per year in Denver, 19,000 per year in Atlanta, 35,000 per year in Philadelphia, and 110,000 per year in Los Angeles.
  - The average (i.e., average across years 2006 to 2010) percentages of school age children estimated to experience one or more exposures per year to 8-hour O_3 concentrations at and above 0.070 ppm, while at moderate or greater exertion, were approximately 4% for Denver (corresponding to 22,000 children), 9% for Atlanta (corresponding to 75,000 children), 10% for Philadelphia (corresponding to 117,000 children), and 15% for Los Angeles (corresponding to 559,000 children). There was considerable variability in these percentages across the years evaluated, ranging from approximately 1 to 10% in Denver, 2 to 19% in Atlanta, 1 to 16% in Philadelphia, and 8 to 21% in Los Angeles. When considering exposures at and above 0.070 ppm in asthmatic children at moderate or greater exertion, the results were similar in term of

percentages, corresponding to average numbers of exposed asthmatic children of approximately 2,000 per year in Denver, 8,000 per year in Atlanta, 14,000 per year in Philadelphia, and 54,000 per year in Los Angeles.

- The average (i.e., average across years 2006 to 2010) percentages of school age children estimated to experience one or more exposures per year to 8-hour O_3 concentrations at and above 0.080 ppm, while at moderate or greater exertion, were approximately 0.4% for Denver (corresponding to 2,000 children), 2% for Philadelphia (corresponding to 28,000 children), 3% for Atlanta (corresponding to 24,000 children), and 6% for Los Angeles (corresponding to 218,000 children). There was considerable variability in these percentages across the years evaluated, ranging from approximately 0 to 1% in Denver, 0 to 7% in Atlanta, 0 to 6% in Philadelphia, and 2 to 10% in Los Angeles. When considering exposures at and above 0.080 ppm in asthmatic children at moderate or greater exertion, the results were similar in term of percentages, corresponding to average numbers of exposed asthmatic children of approximately 200 per year in Denver, 2,000 per year in Atlanta, 3,000 per year in Philadelphia, and 22,000 per year in Los Angeles.
- Between years, the pattern of exposures across cities differed. Generally, from 2006 to 2009, O_3 exposures fell, but in 2010, exposures increased somewhat with the exception of Los Angeles. In the worst O_3 year (2006), the percent of children exposed, while at moderate or greater exertion, to concentrations at and above the lowest health benchmark, 0.060 ppm, ranged from 30 to 37% across the 4 study areas. The percent at and above 0.070 ppm ranged from 10 to 21%, and the percent at and above 0.080 ppm ranged from 1 to 10%. In the best O_3 year (2009), the percent of children ranged from 9 to 32% for exposures at and above 0.060 ppm, from 1 to 15% for exposures at and above 0.070 ppm, and from 0 to 5% for exposures at and above 0.080 ppm, while at moderate or greater exertion.

- Exposure Assessment for Simulating Meeting the Current O_3 Standard
  - Simulating just meeting the current O_3 standard reduces exposures such that across the 5 years the estimated percent of children exposed to concentrations at and above the lowest health benchmark, 0.060 ppm, while at moderate or greater exertion, ranged from 3 to 14% for Atlanta

(corresponding to approximately 24,000 to 123,000 children), 6 to 16% for Denver (corresponding to approximately 31,000 to 89,000 children), 2 to 5% for Los Angeles (corresponding to approximately 66,000 to 186,000 children), and 3 to 18% for Philadelphia (corresponding to approximately 34,000 to 213,000 children).

- Just meeting the current standard in Los Angeles has the largest impact across the four cities on the percent of children exposed above 0.060 ppm. After simulating just meeting the current standard, the estimated percent of children exposed above 0.060 ppm for the five years falls to a maximum of 5% (with a range between 2 to 5%), compared with a minimum of 23% (with a range between 23 to 37%) under recent conditions.
- After just meeting the current O_3 standard, the estimated percent of children exposed to concentrations above 0.070 ppm, while at moderate or greater exertion, ranged across the 5 years from 0.2 to 2% for Atlanta (corresponding to approximately 1,000 to 18,000 children), 0.2 to 1.4% for Denver (corresponding to approximately 1,000 to 7,000 children), 0 to 0.5% for Los Angeles (corresponding to approximately 1,000 to 17,000 children), and 0 to 4.0% for Philadelphia (corresponding to approximately 300 to 44,000 children).
- After just meeting the current O_3 standard, the estimated percent of children exposed to concentrations above 0.080 ppm, while at moderate or greater exertion, ranged across the 5 years from 0 to 0.2% for Atlanta (corresponding to approximately 0 to 2,000 children), 0 to 0.1% for Denver (corresponding to approximately 0 to 400 children), 0% for Los Angeles (corresponding to approximately 0 to 200 children), and 0 to 0.3% for Philadelphia (corresponding to approximately 0 to 3,000 children).

- Characterization of Factors Influencing High Exposures
  - Children are an important exposure population subgroup, largely a result of the combined outdoor time expenditure along with concomitantly performing moderate or high exertion level activities.
  - Persons having a majority of their time spent outdoors experienced the highest 8-hour O_3 exposure concentrations given that O_3 concentrations in other microenvironments were simulated to be lower than ambient concentrations.

- Simulations of highly exposed children in Los Angeles estimate that they spend half of their outdoor time engaged in moderate or greater exertion levels, such as in sporting activities. Highly exposed adults are estimated to have lower activity levels during time spent outdoors.
- For populations experiencing one or more exposures per year to 8-hour O_3 concentrations above 0.050 ppm, the highest modeled exposures are determined primarily by amount of time spent outdoors in locations with high ambient O_3 concentrations. There are differences in the influence of outdoor time relative to ambient concentrations between locations, likely due to air conditioning prevalence.

9.2 SUMMARY OF KEY RESULTS FOR HEALTH RISKS BASED ON CONTROLLED HUMAN EXPOSURE STUDIES

The first draft lung function risk assessment evaluated risks of lung function decrements due to O_3 exposure for all children and children with asthma. The analysis applies probabilistic exposure-response relationships for lung function decrements (measured as percent reductions in FEV1) associated with 8-hour moderate exertion exposures. The analysis provides estimates of the percent of children experiencing a reduction in lung function for three different levels of impact, 10, 15, and 20 percent decrements in FEV1. These levels of impact were selected based on the literature discussing the adverse associated with these types of lung function decrements (US EPA, 2012, Section 6.2.1.1; Henderson, 2006). For the first draft assessment, lung function risks were estimated for 4 cities, Atlanta, Denver, Los Angeles, and Philadelphia. Key results include: *[To be provided in an updated draft anticipated to be available in August, 2012]*

- ……..
- ……..

9.3 SUMMARY OF KEY RESULTS FOR HEALTH RISKS BASED ON EPIDEMIOLOGICAL STUDIES

The first draft risk assessment also evaluated risks of mortality and morbidity from short-term exposures to O_3 based on application of concentration-response functions derived from epidemiology studies. The analysis included both a set of urban area case studies and a national scale assessment. The urban case study analyses evaluated mortality and morbidity risks, including emergency department (ED) visits, hospitalizations, and respiratory symptoms associated with recent O_3 concentrations (2006-2010) and with O_3 concentrations simulating just

meeting the current O_3 standard. Mortality and hospital admissions (HA) were evaluated in 12 urban areas, while ED visits and respiratory symptoms were evaluated in a subset of areas. These 12 urban areas were: Atlanta, GA; Baltimore, MD; Boston, MA; Cleveland, OH; Denver, CO; Detroit, MI; Houston, TX; Los Angeles, CA; New York, NY; Philadelphia, PA; Sacramento, CA; and St. Louis, MO. The urban case study analyses focus on risk estimates for the middle year of each three-year attainment simulation period (2006-2008 and 2008-2010) in order to provide estimates of risk for a year with generally higher O_3 levels (2007) and a year with generally lower O_3 levels (2009).

The national scale assessment evaluated only mortality associated with recent O_3 concentrations across the entire U.S for 2006-2008. The national scale assessment is a complement to the urban scale analysis, providing both a broader assessment of O_3-related health risks across the U.S., as well as an evaluation of how well the 12 urban study areas represented the full distribution of ozone-related health risks in the U.S.

Both the urban area and national scale assessments provide the absolute incidence and percent of incidence attributable to O_3. Risk estimates are presented for ozone concentrations down to zero, as well as down to the lowest measured levels (LML) of O3 in the year of the analysis, as a weak surrogate for the LML in the epidemiology studies. The approach most consistent with the statistical models reported in the epidemiological studies is to apply the concentration-response functions to all ozone concentrations down to zero. However, consistent with the conclusions of the ISA, we also recognize that confidence in the nature of the concentration-response function and the magnitude of the risks associated with very low concentrations of ozone is reduced because there are few ozone measurements at the lowest levels in many of the urban areas included in the studies. As a result, the LML provides a cutoff value above which we have higher confidence in the estimated risks. In our judgment, the two sets of estimates based on estimating risk down to zero and estimating risk down to the LML provide a reasonable bound on estimated total risks, reflecting uncertainties about the C-R function below the lowest ozone levels evaluated in the studies.

Key results of the urban area case studies include:

- Short-term Mortality Risks Associated with Recent Air Quality
    - There are significant differences in the spatial pattern of mortality risks based on application of results from the two large multi-city epidemiology studies. The estimates based on Zanobetti and Schwartz (2008) show the largest impacts in Boston, Detroit, Los Angeles, and New York, while the estimates based on Bell et al (2004) show the largest impacts in Atlanta, Boston, Houston, Los Angeles, and New York.

- Estimates of mortality attributable to short term O_3 exposure under recent conditions vary widely across urban study areas, reflecting differences in ambient O_3 levels and populations, as well as differences in city-specific effect estimates. The patterns of variability across cities differs between the Zanobetti and Schwartz (2008) and Bell et al (2004) based results because of differences in the effect estimates and differences in the O3 metrics (daily 8-hour maximum vs fixed 8-hour mean).
- The O_3 attributable mortality risk estimates for 2007 based on the two epidemiology studies range across the 12 urban areas from 20 to approximately 930 deaths and approximately 0.5 to 4.9% of total baseline all-cause mortality, with no concentration cutoff, and 10 to approximately 730deaths and approximately 0.4 to 3.5% of total baseline all-cause mortality, with a concentration cutoff of the estimated LML. For 2009, the O_3 attributable mortality risk estimates range across the 12 urban study areas from 20 to approximately 980 deaths and approximately 0.6 to 4.3% of total baseline all-cause mortality, with no concentration cutoff, and 10 to approximately 780 deaths and approximately 0.4 to 3.0% of total baseline all-cause mortality, with a concentration cutoff of the estimated LML. For most (but not all, e.g. Los Angeles) of the urban areas, O_3-attributable mortality risks are somewhat smaller in 2009 as compared with 2007. This reflects primarily the lower O_3 levels seen in 2009.
- Twenty-five to 80% of the mortality risk is associated with days having O_3 levels above 55 to 60 ppb.

- Short-term Mortality Risks Associated with Simulating Meeting the Current O_3 Standard
  - After simulating just meeting the current standard in 2007 across the 12 urban study areas, we estimate O_3 attributable mortality to vary from 20 to 850 deaths and approximately 0.5 to 4.6% of total baseline all-cause mortality, with no concentration cutoff, and 10 to approximately 630 deaths and approximately 0.3 to 3.1% of total baseline all-cause mortality, with a concentration cutoff of LML. After simulating just meeting the current standard in 2009, we estimate O_3 attributable mortality across the 12 urban study areas to vary from 20 to 820 deaths and approximately 0.6-4.1% of total baseline all-cause mortality, with no concentration cutoff, and 10 to approximately 630 deaths and approximately 0.3 to 3.0% of total baseline all-cause mortality, with a concentration cutoff of LML.

- Five to 60% of mortality reductions occur due to reductions in O_3 on days when 8-hour O_3 is greater than 55 to 60 ppb. As is expected, after simulating just meeting the current standard, the percent of risk occurring on days with 8-hour O3 greater than 55 to 60 ppb falls to 4 to 59%.

- Short-term Morbidity Risks Associated with Recent Conditions
  - Estimates of morbidity attributable to short-term O_3 exposure in 2007 include: (a) 3,000 to 6,000 respiratory ED visits for Atlanta and 7,000 to 11,000 for asthma ED visits in New York, (b) 20,000 to 30,000 asthma exacerbations in Boston, (c) 500 to 700 asthma HA in New York and (d) up to 60 COPD and pneumonia HA in each of the 12 urban study areas.
- Short-term Morbidity Risks Associated with Simulating Meeting the Current O_3 Standard
  - Morbidity risks decrease after simulating just meeting the current standards in 2007, although greater than 80% of ED visits remain in Atlanta, 90% of ED visits remain in New York, and greater than 70% of HA remain in most of the other urban areas.
  - Ozone-related hospital admissions for respiratory causes remaining upon just meeting the current standard, ranging across the 12 case study locations, are estimated to be between 1.3 to 2.4% of all respiratory-related hospital admissions. Further, in New York City, additional information is available on ozone-related hospital admissions for asthma, which upon just meeting the current standard are estimated to be approximately 12 to 17% of total asthma-related hospital admissions.

Key results of the national scale assessment of mortality risk for recent (2006-2008) O_3 concentrations:

- National-scale Short-term Mortality Risk
  - The central estimates of the national burden of total O_3 attributable mortality based on Zanobetti and Schwartz (2008) and Bell et al (2004) and recent O_3 levels are estimated to be 13,000 and 18,000, respectively, in 2006-2008.
  - There is considerable variation between estimates based on the Zanobetti and Schwartz (2008) results and those based on the Bell et al (2004) results. The estimated percentage of total county-level mortality attributable to O_3 across all counties for the Zanobetti and Schwartz based estimates ranges from 0.5 to 5.2%, with a median of 2.5%, with no

concentration cutoff and from 0.4 to 4.4%, with a median of 2.1%, with a concentration cutoff at 7.5 ppb, which is the average LML across cities as reported by Zanobetti and Schwartz. The estimated percentage of total county-level mortality attributable to O_3 for the Bell et al (2004) based estimates ranges from 0.4 to 4.2%, with a median of 1.9%, with no concentration cutoff and from 0.3 to 3.5%, with a median of 1.6%, with a concentration cutoff at 7.5 ppb.

  - For estimates based on both epidemiology studies, we find that 85-90% of O_3-related deaths occur in locations where the seasonal average 8-hr daily maximum or 8-hr daily mean (10am-6pm) O_3 concentration is greater than 40 ppb, corresponding to 4th high 8-hr daily maximum O_3 concentrations ranging from approximately 50 ppb to 100 ppb.

- Representativeness of the Urban Study Areas in the National Context
  - We observe that the 23 counties for the 12 urban study areas considered capture urban areas that are among the most populated in the U.S., have relatively high ozone levels, and represent the range of city-specific effect estimates found by Bell et al. (2004) and Zanobetti and Schwartz (2008). These three factors suggest that the urban study areas represent the overall distribution of risk across the nation well, with a potential for better characterization of the high end of the risk distribution.
  - We find that the urban study areas are not capturing areas with the highest baseline mortality rates, those with the oldest populations, and those with the lowest air conditioning prevalence. These areas tend to have relatively low ozone concentrations and low total population, suggesting that the urban study areas are not missing high risk populations that have high ozone concentrations in addition to greater susceptibility per unit ozone.
  - The second representativeness analysis demonstrated that the 12 urban study areas represent the the overall distribution of ozone-related risk across the entire U.S.

9.4 OBSERVATIONS

[These observations have been prepared based on the exposure and epidemiological risk estimates available for the July public release of the first draft REA. We anticipate providing Chapter 9, with additional observations based on the lung function risk analysis, when we provide supplemental REA materials along with the submissions of the first draft Policy Assessment for public review in August]

Recent O_3 concentrations have in general been declining over the period of analysis, 2006 to 2010. As a result, the risks and exposures associated with O_3 have also been declining. However, while the overall trend in O_3 has been downward, for some locations, O_3 has displayed a more variable pattern, for example, while most study locations saw a decrease in O_3 between 2007 and 2008, Sacramento saw an increase to its highest level in 2008. In addition, the downward trend generally did not hold in 2010, which saw slightly higher O_3 concentrations in almost all of the study areas. Thus, while 2007 and 2009 generally represent worst case and best case years within this five-year period, it should be recognized that additional variability in results exists. In general, year to year variability in results is as significant as variability between urban areas for both exposure and risk.

The results of the risk and exposure assessment suggest that while O_3 concentrations have generally been declining over the analytical period from 2006 to 2010, there are still remaining exposures to elevated levels of O_3, and health risks associated with those exposures. These exposures and health risks vary across the urban case study areas, but are generally consistent in showing exposures above health benchmarks and risks associated with recent O_3 concentrations. On a national scale, recent O_3 concentrations (2006-2008) are associated with a significant public health burden, and risks are widespread across the U.S., with 50% of counties experiencing at least 0.7 to 1.0% mortality attributable to recent O_3 concentrations.

There are several important factors to consider when evaluating exposures and risks associated with recent exposures to O_3. First, with regard to the epidemiology based risk estimates, while we have included a number of different model specifications to begin understanding how variability in the underlying epidemiological studies can affect results, there are still a number of variables that might affect risk results that we have not been able to include in this first draft assessment, particularly in the case of modeling short-term exposure-related mortality risk. Some of these include alternative lag structures and treatment of co-pollutants.

Second, with regard to the exposure estimates, the APEX model is not proficient at modeling repeated exposures. As a result, while we are able to report the percent of children with at least one exposure greater than the alternative exposure benchmarks, we are not able to report with confidence the percent of children with more than one exposure. Children with repeated exposures may be at greater risk of significant health effects. In addition, we were only able to model exposure in four cities for this first draft assessment. It is likely that variation in exposure will be larger when we have modeled the full set of 16 cities in the second draft REA.

Third, for this first draft of the REA, while we used a relatively simple roll-back approach tor simulating just meeting the current standard, we also discussed the use of other approaches that are based on modeling the response of O_3 concentrations to reductions in anthropogenic NOx and VOC emissions, using the Higher-Order Decoupled Direct Method

(HDDM) capabilities in the Community Multi-scale Air Quality (CMAQ) model. This modeling incorporates all known emissions, including emissions from non-anthropogenic sources and anthropogenic emissions from sources in and outside of the U.S. As a result, the need to specify values for U.S. background concentrations is not necessary, as it is incorporated in the modeling directly. We plan to further explore the use of this methodology in the second draft of the REA. Application of this approach also addresses the recommendation by the National Research Council of the National Academies (NRC, 2008) to explore how emissions reductions might effect temporal and spatial variations in O_3 concentrations, and to include information on how NO_x versus VOC control strategies might affect risk and exposure to O_3.

This first draft REA provides preliminary estimates of exposures and risks which provide information that can be used to begin discussions in the Policy Assessment regarding the adequacy of the current standard. The second draft REA will further refine the estimates of exposure and risk by incorporating additional urban areas into the exposure and lung function risk analyses, and by expanding the sensitivity analyses supporting the epidemiology based risk estimates. In addition, based on advice and comments received on this first draft REA, the second draft REA may include additional health endpoints associated with longer-term exposures to O_3. The second draft REA will also evaluate any alternative O_3 standards identified in the first draft Policy Assessment following evaluation of any advice and comments on those potential alternative standards provided during the review by the CASAC O_3 Panel. Finally, we anticipate that the second draft REA will incorporate an improved approach to adjusting O_3 concentrations based on simulations of just meeting the current and alternative O_3 standards.

Appendix 5-A

Description of the Air Pollutants Exposure Model (APEX)

1. Overview

APEX estimates human exposure to criteria and toxic air pollutants at local, urban, or regional scales using a stochastic, microenvironmental approach. That is, the model randomly selects data on a sample of hypothetical individuals in an actual population database and simulates each individual's movements through time and space (e.g., at home, in vehicles) to estimate their exposure to the pollutant. APEX can assume people live and work in the same general area (i.e., that the ambient air quality is the same at home and at work) or optionally can model commuting and thus exposure at the work location for individuals who work.

The APEX model is a microenvironmental, longitudinal human exposure model for airborne pollutants. It is applied to a specified study area, which is typically a metropolitan area. The time period of the simulation is typically one year, but can easily be made either longer or shorter. APEX uses census data, such as gender and age, to generate the demographic characteristics of simulated individuals. It then assembles a composite activity diary to represent the sequence of activities and microenvironments that the individual experiences. Each microenvironment has a user-specified method for determining air quality. The inhalation exposure in each microenvironment is simply equal to the air concentration in that microenvironment. When coupled with breathing rate information and a physiological model, various measures of dose can also be calculated.

The term *microenvironment* is intended to represent the immediate surroundings of an individual, in which the pollutant of interest is assumed to be well-mixed. Time is modeled as a sequence of discrete time steps called *events*. In APEX, the concentration in a microenvironment may change between events. For each microenvironment, the user specifies the method of concentration calculation (either mass balance or regression factors, described later in this paper), the relationship of the microenvironment to the ambient air, and the strength of any pollutant sources specific to that microenvironment. Because the microenvironments that are relevant to exposure depend on the nature of the target chemical and APEX is designed to be

applied to a wide range of chemicals, both the total number of microenvironments and the properties of each are free to be specified by the user.

The ambient air data are provided as input to the model in the form of time series at a list of specified locations. Typically, hourly air concentrations are used, although temporal resolutions as small as one minute may be used. The spatial range of applicability of a given ambient location is called an air district. Any number of air districts can be accommodated in a model run, subject only to computer hardware limitations. In principle, any microenvironment could be found within a given air district. Therefore, to estimate exposures as an individual engages in activities throughout the period it is necessary to determine both the microenvironment and the air district that apply for each event.

An *exposure event* is determined by the time reported in the activity diary; during any event the district, microenvironment, ambient air quality, and breathing rate are assumed to remain fixed. Since the ambient air data change every hour, the maximum duration of an event is limited to one hour. The event duration may be less than this (as short as one minute) if the activity diary indicates that the individual changes microenvironments or activities performed within the hour.

The APEX simulation includes the following steps:

1. Characterize the study area - APEX selects sectors (e.g., census tracts) within a study area based on user-defined criteria and thus identifies the potentially exposed population and defines the air quality and weather input data required for the area.
2. Generate simulated individuals - APEX stochastically generates a sample of simulated individuals based on the census data for the study area and human profile distribution data (such as age-specific employment probabilities). The user must specify the size of the sample. The larger the sample, the more representative it is of the population in the study area and the more stable the model results are (but also the longer the computing time).
3. Construct a long-term sequence of activity events and determine breathing rates - APEX constructs an event sequence (activity pattern) spanning the period of simulation for each simulated person. The model then stochastically assigns breathing rates to each event, based on the type of activity and the physical characteristics of the simulated person.
4. Calculate pollutant concentrations in microenvironments - APEX enables the user to define any microenvironment that individuals in a study area would visit. The model then calculates concentrations of each pollutant in each of the microenvironments.

5. Calculate pollutant exposures for each simulated individual - Microenvironmental concentrations are time weighted based on individuals' events (i.e., time spent in the microenvironment) to produce a sequence of time-averaged exposures (or minute by minute time series) spanning the simulation period.

6. Estimate dose - APEX can also calculate the dose time series for each of the simulated individuals based on the exposures and breathing rates for each event. For CO there is a physiologically-based dosimetry module that estimates blood carboxyhemoglobin (COHb) levels resulting from CO exposure. When modeling particulate matter, the rate of mass deposition in the respiratory system is calculated using an empirical model (ICRP 1994). For all other pollutants, an intake dose can be estimated using the exposure concentration multiplied by breathing rate.

The model simulation continues until exposures are determined for the user-specified number of simulated individuals. APEX then calculates population exposure statistics (such as the number of exposures exceeding user-specified levels) for the entire simulation and writes out tables of distributions of these statistics.

2. Model Inputs

APEX requires certain inputs from the user. The user specifies the geographic area and the range of ages and age groups to be used for the simulation. Hourly (or shorter) ambient air quality and hourly temperature data must be furnished for the entire simulation period. Other hourly meteorological data (humidity, wind speed, wind direction, precipitation) can be used by the model to estimate microenvironmental concentrations, but are optional.

In addition, most variables used in the model algorithms are represented by user-specified probability distributions which capture population variability. APEX provides great flexibility in defining model inputs and parameters, including options for the frequency of selecting new values from the probability distributions. The model also allows different distributions to be used at different times of day or on different days, and the distribution can depend conditionally on values of other parameters. The probability distributions available in APEX include beta, binary, Cauchy, discrete, exponential, extreme value, gamma, logistic, lognormal, loguniform, normal, off/on, Pareto, point (constant), triangle, uniform, Weibull, and nonparametric distributions. Minimum and maximum bounds can be specified for each distribution if a truncated distribution is appropriate. There are two options for handling truncation. The generated samples outside the truncation points can be set to the truncation limit; in this case,

samples “stack up” at the truncation points. Alternatively, new random values can be selected, in which case the probability outside the limits is spread over the specified range, and thus the probabilities inside the truncation limits will be higher than the theoretical untruncated distribution.

3. Demographic Characteristics

The starting point for constructing a simulated individual is the population census database; this contains population counts for each combination of age, gender, race, and *sector*. The user may decide what spatial area is represented by a sector, but the default input file defines a sector as a *census tract*. Census tracts are variable in both geographic size and population number, though usually have between 1,500 and 8,000 persons. Currently, the default file contains population counts from the 2000 census for every census tract in the United States, thus the default file should be sufficient for most exposure modeling purposes. The combination of age, gender, race, and sector are selected first. The sector becomes the *home sector* for the individual, and the corresponding air district becomes the *home district*. The probabilistic selection of individuals is based on the sector population and demographic composition, and taken collectively, the set of simulated individuals constitutes a random sample from the study area.

The second step in constructing a simulated individual is to determine their employment status. This is determined by a probability which is a function of age, gender, and home sector. An input file is provided which contains employment probabilities from the 2000 census for every combination of age (16 and over), gender, and census tract. APEX assumes that persons under age 16 do not commute. For persons who are determined to be workers, APEX then randomly selects a *work sector*, based on probabilities determined from the commuting matrix. The work sector is used to assign a *work district* for the individual that may differ from the home district, and thus different ambient air quality may be used when the individual is at work.

The commuting matrix contains data on flows (number of individuals) traveling from a given home sector to a given work sector. Based on commuting data from the 2000 census, a commuting data base for the entire United States has been prepared. This permits the entire list of non-zero flows to be specified on one input file. Given a home sector, the number of destinations to which people commute varies anywhere from one to several hundred other tracts.

4. Attributes of Individuals

In addition to the above demographic information, each individual is assigned status and physiological attributes. The status variables are factors deemed important in estimating microenvironmental concentrations, and are specified by the user. Status variables can include, but are not limited to, people's housing type, whether their home has air conditioning, whether they use a gas stove at home, whether the stove has a gas pilot light, and whether their car has air conditioning. Physiological variables are important when estimating pollutant specific dose. These variables could include height, weight, blood volume, pulmonary diffusion rate, resting metabolic rate, energy conversion factor (liters of oxygen per kilocalorie energy expended), hemoglobin density in blood, maximum limit on MET ratios (see below), and endogenous CO production rate. All of these variables are treated probabilistically taking into account interdependencies, reflecting variability in the population.

5. Construction of Activity Diaries

The activity diary determines the sequence of microenvironments visited by the simulated person. A longitudinal sequence of daily diaries must be constructed for each simulated individual to cover the entire simulation period. The default activity diaries in APEX are derived from those in the EPA's Consolidated Human Activity Database (CHAD), although the user could provide area specific diaries if available. There are over 33,000 CHAD diaries, each covering a 24 hour period, that have been compiled from several studies. CHAD is essentially a cross-sectional database that, for the most part, only has one diary per person. Therefore, APEX must assemble each longitudinal diary sequence for a simulated individual from many single-day diaries selected from a pool of similar people.

APEX selects diaries from CHAD by matching gender and employment status, and by requiring that age falls within a user-specified range on either side of the age of the simulated individual. For example, if the user specifies plus or minus 20%, then for a 40 year old simulated individual, the available CHAD diaries are those from persons aged 32 to 48. Each simulated individual therefore has an age window of acceptable diaries; these windows can partially overlap those for other simulated individuals. This differs from a cohort-based approach, where the age windows are fixed and non-overlapping. The user may optionally request that APEX allow a decreased

probability for selecting diaries from ages outside the primary age window, and also for selecting diaries from persons of missing gender, age, or employment status. These options allow the model to continue the simulation when diaries are not available within the primary window.

The available CHAD diaries are classified into *diary pools*, based on the temperature and day of the week. The model will select diaries from the appropriate pool for days in the simulation having matching temperature and day type characteristics. The rules for defining these pools are specified by the user. For example, the user could request that all diaries from Monday to Friday be classified together, and Saturday and Sunday diaries in another class. Alternatively, the user could instead create more than two classes of weekdays, combine all seven days into one class, or split all seven days into separate classes.

The temperature classification can be based either on daily maximum temperature, daily average temperature, or both. The user specifies both the ranges and numbers of temperatures classes. For example, the user might wish to create four temperature classes and set their ranges to below 50, 50-69, 70-84, and above a daily maximum of 84°F. Then day type and temperature classes are combined to create the diary pools. For example, if there are four temperature classes and two day type classes, then there will be eight diary pools.

APEX then determines the day-type and the applicable temperature for each person's simulated day. APEX allows multiple temperature stations to be used; the sectors are automatically mapped to the nearest temperature station. This may be important for study areas such as the greater Los Angeles area, where the inland desert sectors may have very different temperatures from the coastal sectors. For selected diaries, the temperature in the home sector of the simulated person is used. For each day of the simulation, the appropriate diary pool is identified and a CHAD dairy is randomly drawn. When a diary for every day in the simulation period has been selected, they are concatenated into a single longitudinal diary covering the entire simulation for that individual. APEX contains three algorithms for stochastically selecting diaries from the pools to create the longitudinal diary. The first method selects diaries at random after stratification by age, gender, and diary pool; the second method selects diaries based on metrics related to exposure (e.g., time spent outdoors) with the goal of creating longitudinal

diaries with variance properties designated by the user; and the third method uses a clustering algorithm to obtain more realistic recurring behavioral patterns.

The final step in processing the activity diary is to map the CHAD location codes into the set of APEX microenvironments, supplied by the user as an input file. The user may define the number of microenvironments, from one up to the number of different CHAD location codes (which is currently 115).

6. Microenvironmental Concentrations

The user provides rules for determining the pollutant concentration in each microenvironment. There are two available models for calculating microenvironmental concentrations: mass balance and regression factors. Any indoor microenvironment may use either model; for each microenvironment, the user specifies whether the mass balance or factors model will be used.

6.1 Mass Balance Model

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. The concentration of an air pollutant in such a microenvironment is estimated using the following four processes (as illustrated in Figure 1):

- 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.

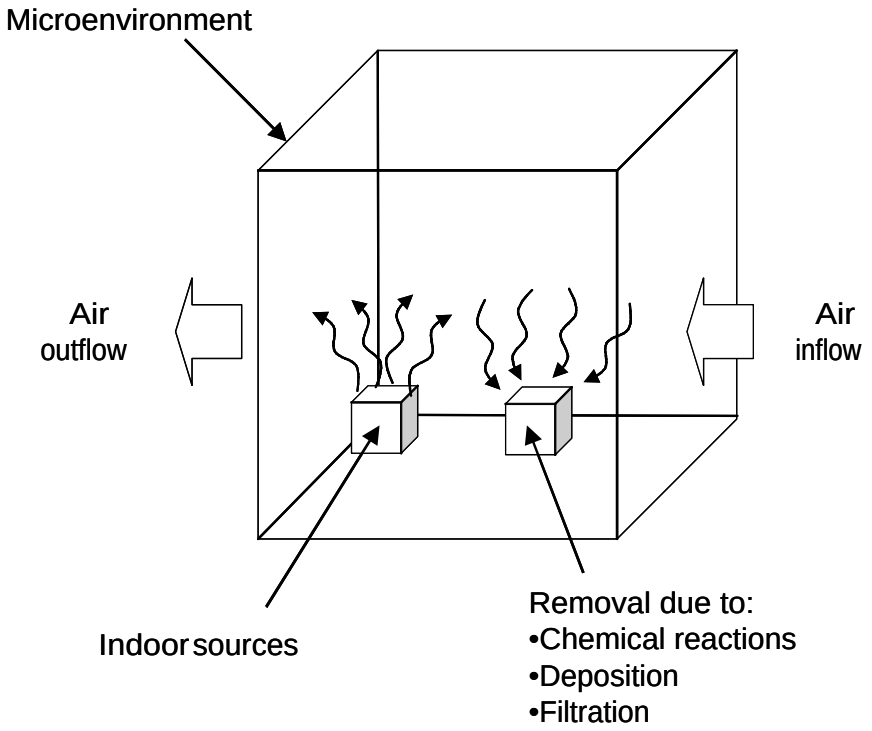

Figure 1. Components of the Mass Balance Model Used by APEX.

Considering the microenvironment as a well-mixed fixed volume of air, the mass balance equation for a pollutant in the microenvironment can be written in terms of concentration:

$$\frac{dC(t)}{dt} = \dot{C}_{in} - \dot{C}_{out} - \dot{C}_{removal} + \dot{C}_{source} \qquad (1)$$

where:

$C(t)$	=	Concentration in the microenvironment at time t
$\dot{C}_{in}$	=	Rate of change in $C(t)$ due to air entering the microenvironment
$\dot{C}_{out}$	=	Rate of change in $C(t)$ due to air leaving the microenvironment
$\dot{C}_{removal}$	=	Rate of change in $C(t)$ due to all internal removal processes
$\dot{C}_{source}$	=	Rate of change in $C(t)$ due to all internal source terms

Concentrations are calculated in the same units as the ambient air quality data, e.g., ppm, ppb, ppt, or $\mu g/m^3$. In the following equations concentration is shown only in $\mu g/m^3$ for brevity. The change in microenvironmental concentration due to influx of air, $\dot{C}_{in}$, is given by:

$$\dot{C}_{in} = C_{outdoor} \times f_{penetration} \times R_{air\ exchange} \quad (2)$$

where:

$C_{outdoor}$	=	Ambient concentration at an outdoor microenvironment or outside an indoor microenvironment ($\mu g/m^3$)
$f_{penetration}$	=	Penetration factor (unitless)
$R_{air\ exchange}$	=	Air exchange rate (hr^{-1})

Since the air pressure is approximately constant in microenvironments that are modeled in practice, the flow of outside air into the microenvironment is equal to that flowing out of the microenvironment, and this flow rate is given by the air exchange rate. The air exchange rate (hr^{-1}) can be loosely interpreted as the number of times per hour the entire volume of air in the microenvironment is replaced. For some pollutants (especially particulate matter), the process of infiltration may remove a fraction of the pollutant from the outside air. The fraction that is retained in the air is given by the penetration factor $f_{penetration}$.

A proximity factor ($f_{proximity}$) and a local outdoor source term are used to account for differences in ambient concentrations between the geographic location represented by the ambient air quality data (e.g., a regional fixed-site monitor) and the geographic location of the microenvironment. That is, the outdoor air at a particular location may differ systematically from the concentration input to the model representing the air quality district. For example, a playground or house might be located next to a busy road in which case the air at the playground or outside the house would have elevated levels for mobile source pollutants such as carbon monoxide and benzene. The concentration in the air at an outdoor location or directly outside an indoor microenvironment ($C_{outdoor}$) is calculated as:

$$C_{outdoor} = f_{proximity} C_{ambient} + C_{LocalOutdoorSources} \quad (3)$$

where:

$C_{ambient}$	=	Ambient air district concentration ($\mu g/m^3$)
$f_{proximity}$	=	Proximity factor (unitless)
$C_{LocalOutdoorSources}$	=	The contribution to the concentration at this location from local sources not represented by the ambient air district concentration ($\mu g/m^3$)

During exploratory analyses, the user may examine how a microenvironment affects overall exposure by setting the microenvironment's proximity or penetration factor to zero, thus effectively eliminating the specified microenvironment.

Change in microenvironmental concentration due to outflux of air is calculated as the concentration in the microenvironment *C(t)* multiplied by the air exchange rate:

$$\dot{C}_{out} = R_{air\ exchange} \times C(t) \tag{4}$$

The third term ($\dot{C}_{removal}$) in the mass balance calculation (1) represents removal processes within the microenvironment. There are three such processes in general: chemical reaction, deposition, and filtration. Chemical reactions are significant for O_3, for example, but not for carbon monoxide. The amount lost to chemical reactions will generally be proportional to the amount present, which in the absence of any other factors would result in an exponential decay in the concentration with time. Similarly, deposition rates are usually given by the product of a (constant) deposition velocity and a (time-varying) concentration, also resulting in an exponential decay. The third removal process is filtration, usually as part of a forced air circulation or HVAC system. Filtration will normally be more effective at removing particles than gases. In any case, filtration rates are also approximately proportional to concentration.

Change in concentration due to deposition, filtration, and chemical degradation in a microenvironment is simulated based on the first-order equation:

$$\begin{aligned}\dot{C}_{removal} &= \left(R_{deposition} + R_{filtration} + R_{chemical}\right) \times C(t) \\ &= R_{removal} \times C(t)\end{aligned} \tag{5}$$

where:

$\dot{C}_{removal}$	=	Change in microenvironmental concentration due to removal processes ($\mu g/m^3/hr$)
$R_{deposition}$	=	Removal rate of a pollutant from a microenvironment due to deposition (hr^{-1})
$R_{filtration}$	=	Removal rate of a pollutant from a microenvironment due to filtration (hr^{-1})
$R_{chemical}$	=	Removal rate of a pollutant from a microenvironment due to chemical degradation (hr^{-1})
$R_{removal}$	=	Removal rate of a pollutant from a microenvironment due to the combined effects of deposition, filtration, and chemical degradation (hr^{-1})

The fourth term in the mass balance calculation represents pollutant sources within the microenvironment. This is the most complicated term, in part because several sources may be present. APEX allows two methods of specifying source strengths: emission sources and concentration sources. Either may be used for mass balance microenvironments, and both can be used within the same microenvironment. The source strength values are used to calculate the term $\dot{C}_{source}$ (µg/m^3/hr).

Emission sources are expressed as emission rates in units of µg/hr, irrespective of the units of concentration. To determine the rate of change of concentration associated with an emission source S_E, it is divided by the volume of the microenvironment:

$$\dot{C}_{source,SE} = \frac{S_E}{V} \quad (6)$$

where:

$\dot{C}_{source,SE}$ = Rate of change in $C(t)$ due to the emission source S_E (µg/m^3/hr)

S_E = The emission rate (µg/hr)

V = The volume of the microenvironment (m^3)

Concentration sources (S_C) however, are expressed in units of concentration. These must be the same units as used for the ambient concentration (e.g., µg/m^3). Concentration sources are normally used as additive terms for microenvironments using the factors model. Strictly speaking, they are somewhat inconsistent with the mass balance method, since concentrations should not be inputs but should be consequences of the dynamics of the system. Nevertheless, a suitable meaning can be found by determining the rate of change of concentration ($\dot{C}_{source}$) that would result in a mean increase of S_C in the concentration, given constant parameters and equilibrium conditions, in this way:

Assume that a microenvironment is always in contact with clean air (ambient = zero), and it contains one constant concentration source. Then the mean concentration over time in this microenvironment from this source should be equal to S_C. The mean source strength expressed in ppm/hr or µg/m^3/hr is the rate of change in concentration ($\dot{C}_{source,SC}$). In equilibrium,

$$C_S = \frac{\dot{C}_{source,SC}}{R_{air\ exchange} + R_{removal}} \quad (7)$$

where *Cs* is the mean increase in concentration over time in the microenvironment due to the source $\dot{C}_{source,SC}$. $\dot{C}_{source,SC}$ can thus be written as

$$\dot{C}_{source,SC} = C_S \times R_{mean} \quad (8)$$

where R_{mean} is the chemical removal rate. From Eq. 7, R_{mean} is equal to the sum of the air exchange rate and the removal rate ($R_{air\ exchange} + R_{removal}$) under equilibrium conditions. In general, however, the microenvironment will not be in equilibrium, but in such conditions there is no clear meaning to attach to $\dot{C}_{source,SC}$ since there is no fixed emission rate that will lead to a fixed increase in concentration. The simplest solution is to use $R_{mean} = R_{air\ exchange} + R_{removal}$. However, the user is given the option of specifically specifying R_{mean} (see discussion of parameters below). This may be used to generate a truly constant source strength $\dot{C}_{source,SC}$ by making S_C and R_{mean} both constant in time. If this is not done, then R_{mean} is simply set to the sum of ($R_{air\ exchange} + R_{removal}$). If these parameters change over time, then $\dot{C}_{source,SC}$ also changes. Physically, the reason for this is that in order to maintain a fixed elevation of concentration over the base conditions, then the source emission rate would have to rise if the air exchange rate were to rise.

Multiple emission and concentration sources within a single microenvironment are combined into the final total source term by combining equations 6 and 8:

$$\dot{C}_{source} = \dot{C}_{source,SE} + \dot{C}_{source,SC} = \frac{1}{V}\sum_{i=1}^{n_e} E_{Si} + R_{mean}\sum_{i=1}^{n_c} C_{Si} \quad (9)$$

where:

S_{Ei}	=	Emission source strength for emission source *i* (µg/hr, irrespective of the concentration units)
S_{Ci}	=	Emission source strength for concentration source *i* (µg/m^3)
n_e	=	Number of emission sources in the microenvironment
n_c	=	Number of concentration sources in the microenvironment

In equations 6 and 9, if the units of air quality are ppm rather than µg/m^3, *1/V* is replaced by *f/V*, where *f* = ppm / µg/m^3 = gram molecular weight / 24.45. (24.45 is the volume (liters) of a mole of the gas at 25°C and 1 atmosphere pressure.)

Equations 2, 4, 5, and 9 can now be combined with Eq. 1 to form the differential equation for the microenvironmental concentration C(t). Within the time period of a time step (at most 1 hour), $\dot{C}_{source}$ and $\dot{C}_{in}$ are assumed to be constant. Using $\dot{C}_{combined} = \dot{C}_{source} + \dot{C}_{in}$ leads to:

$$\begin{aligned}\frac{dC(t)}{dt} &= \dot{C}_{combined} - R_{air\ exchange}C(t) - R_{removal}C(t) \\ &= \dot{C}_{combined} - R_{mean}C(t)\end{aligned} \tag{10}$$

Solving this differential equation leads to:

$$C(t) = \frac{\dot{C}_{combined}}{R_{mean}} + \left(C(t_0) - \frac{\dot{C}_{combined}}{R_{mean}}\right)e^{-R_{mean}(t-t_0)} \tag{11}$$

where:

$C(t_0)$ = Concentration of a pollutant in a microenvironment at the beginning of a time step ($\mu g/m^3$)

$C(t)$ = Concentration of a pollutant in a microenvironment at time *t* within the time step ($\mu g/m^3$).

Based on Eq. 11, the following three concentrations in a microenvironment are calculated:

$$C_{equil} = C(t \to \infty) = \frac{\dot{C}_{combined}}{R_{mean}} = \frac{\dot{C}_{source} + \dot{C}_{in}}{R_{air\ exchange} + R_{removal}} \tag{12}$$

$$C(t_0 + T) = C_{equil} + \left(C(t_0) - C_{equil}\right)e^{-R_{mean}T} \tag{13}$$

$$C_{mean} = \frac{1}{T}\int_{t_0}^{t_0+T} C(t)\,dt = C_{equil} + \left(C(t_0) - C_{equil}\right)\frac{1 - e^{-R_{mean}T}}{R_{mean}T} \tag{14}$$

where:

C_{equil} = Concentration in a microenvironment ($\mu g/m^3$) if $t \to \infty$ (equilibrium state).

$C(t_0)$ = Concentration in a microenvironment at the beginning of the time step ($\mu g/m^3$)

$C(t_0+T)$ = Concentration in a microenvironment at the end of the time step ($\mu g/m^3$)

C_{mean} = Mean concentration over the time step in a microenvironment ($\mu g/m^3$)

R_{mean} = $R_{air\ exchange} + R_{removal}$ (hr^{-1})

At each time step of the simulation period, APEX uses Eqs. 12, 13, and 14 to calculate the equilibrium, ending, and mean concentrations, respectively. The calculation continues to the next time step by using $C(t_0+T)$ for the previous hour as $C(t_0)$.

6.2 Factors Model

The factors model is simpler than the mass balance model. In this method, the value of the concentration in a microenvironment is not dependent on the concentration during the previous time step. Rather, this model uses the following equation to calculate the concentration in a microenvironment from the user-provided hourly air quality data:

$$C_{mean} = C_{ambient}\ f_{proximity}\ f_{penetration} + \sum_{i=1}^{n_c} S_{Ci} \quad (15)$$

where:

C_{mean} = Mean concentration over the time step in a microenvironment ($\mu g/m^3$)

$C_{ambient}$ = The concentration in the ambient (outdoor) environment ($\mu g/m^3$)

$f_{proximity}$ = Proximity factor (unitless)

$f_{penetration}$ = Penetration factor (unitless)

S_{Ci} = Mean air concentration resulting from source i ($\mu g/m^3$)

n_c = Number of concentration sources in the microenvironment

The user may specify distributions for proximity, penetration, and any concentration source terms. All of the parameters in the above equation are evaluated for each time step, although these values might remain constant for several time steps or even for the entire simulation.

The ambient air quality data are supplied as time series over the simulation period at several locations across the modeled region. The other variables in the factors and mass balance equations are randomly drawn from user-specified distributions. The user also controls the frequency and pattern of these random draws. Within a single day, the user selects the number of random draws to be made and the hours to which they apply. Over the simulation, the same set of 24 hourly values may either be reused on a regular basis (for example, each winter weekday), or a new set of values may be drawn. The usage patterns may depend on day of the week, on month, or both. It is also possible to define different distributions that apply if specific conditions are met. The air exchange rate is typically modeled with one set of distributions for

buildings with air conditioning and another set of distributions for those which do not. The choice of a distribution within a set typically depends on the outdoor temperature and possibly other variables. In total there are eleven such *conditional variables* which can be used to select the appropriate distributions for the variables in the mass balance or factors equations.

For example, the hourly emissions of CO from a gas stove may be given by the product of three random variables: a binary on/off variable that indicates if the stove is used at all during that hour, a usage duration sampled from a continuous distribution, and an emission rate per minute of usage. The binary on/off variable may have a probability for *on* that varies by time of day and season of the year. The usage duration could be taken from a truncated normal or lognormal distribution that is resampled for each cooking event, while the emission rate could be sampled just once per stove.

7. Exposure time series and dose calculation

The activity diaries provide the time sequence of microenvironments visited by the simulated individual and the activities performed by each individual. The pollutant concentration in the air in each microenvironment is assumed to be spatially uniform throughout the microenvironment and unchanging within each diary event and is calculated by either the factors or the mass balance method, as specified by the user. The exposure of the individual is given by the time sequence of airborne pollutant concentrations that are encountered in the microenvironments visited. Figure 2 illustrates the exposures for one simulated 12-year old child over a 2-day period. On both days the child travels to and from school in an automobile, goes outside to a playground in the afternoon while at school, and spends time outside at home in the evening (H: home, A: automobile, S: school, P: playground, O: outdoors at home).

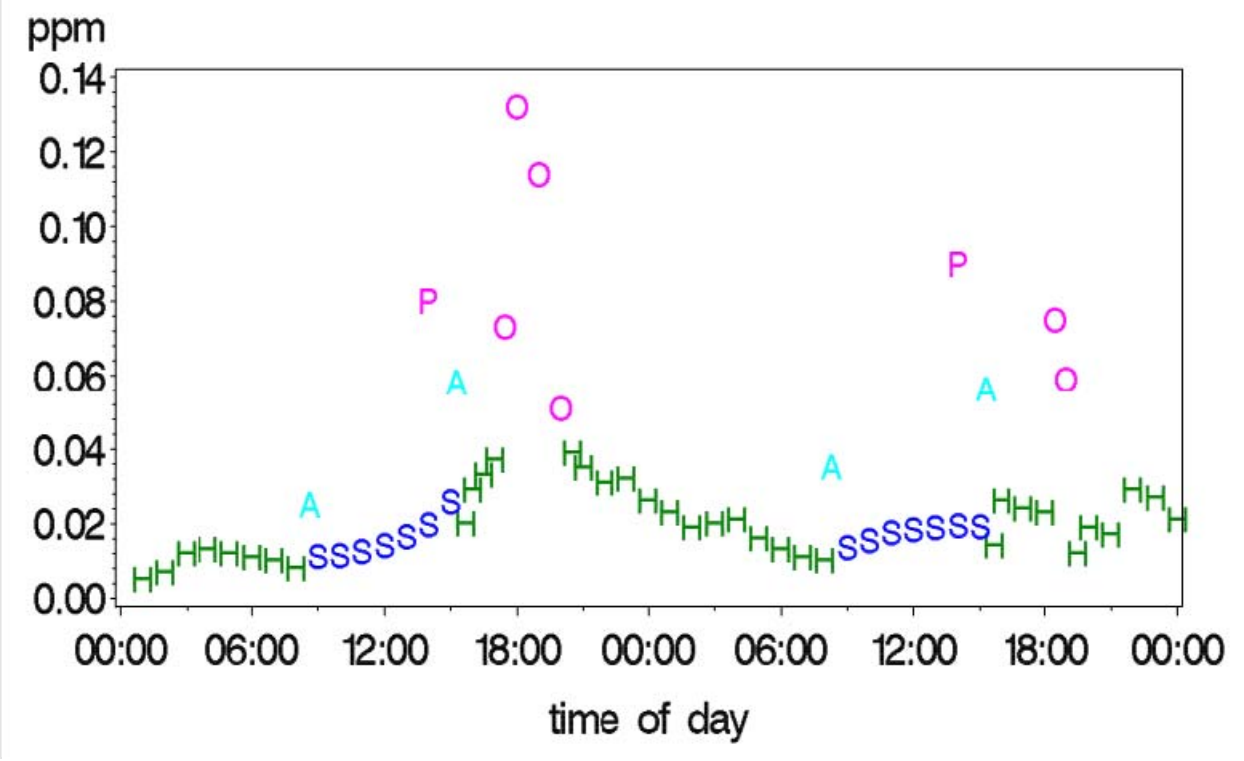

Figure 2. Microenvironmental and Exposure Concentrations for a Simulated Individual over 48 Hours.

In addition to exposure, APEX models breathing rates based on the physiology of each individual and the exertion levels associated with the activities performed. For each activity type in CHAD, a distribution is provided for a corresponding normalized Metabolic Energy for a Task (MET ratio). The MET ratio is a ratio of the metabolic energy requirements for the specific activity as compared to the resting, or basal, metabolic rate. The MET ratios have less interpersonal variation than do the absolute energy expenditures. Based on age and gender, the resting metabolic rate, along with other physiological variables is determined for each individual as part of their anthropometric characteristics. Because the MET ratios are sampled independently from distributions for each diary event, it would be possible to produce time-series of MET ratios that are physiologically unrealistic. APEX employs a MET adjustment algorithm based on a modeled oxygen deficit to prevent such overestimation of MET and breathing rates. The relationship between the oxygen deficit and the applied limits on MET ratios are nonlinear and are derived from published data on work capacity and oxygen consumption. The resulting combination of microenvironmental concentration and breathing ventilation rates provides a time series of inhalation intake dose for most pollutants.

APEX uses additional dose algorithms for the pollutants CO and $PM_{2.5}$. For CO exposures, APEX can calculate the time series of blood carboxyhemoglobin (COHb) levels. These are determined by solving the non-linear Coburn, Forster, Kane equation using a fourth-order Taylor series method. This algorithm is explicit (non-iterative), fast, and accurate, for any practical COHb level (up to more than 50% COHb). $PM_{2.5}$ dose is modeled as the mass of PM depositing in the entire respiratory system, including the extrathoracic regions (mouth, nose, and oropharynx) and the lungs. The PM dose algorithm was developed from the empirical lung deposition equations of the International Commission on Radiological Protection's Human Respiratory Tract Model for Radiological Protection. The empirical equations estimate deposition by both aerodynamic and thermodynamic processes as a function of breathing rate, lung physiology, and particle characteristics.

8. Model output

APEX calculates the exposure and dose time series based on the events as listed on the activity diary with a minimum of one event per hour but usually more during waking hours. APEX can aggregate the event level exposure and dose time series to output hourly, daily, monthly, and annual averages . The types of output files are selected by the user, and can be as detailed as event-level data for each simulated individual (note, Figure 2 was produced from the event output file). A set of summary tables are produced for a variety of exposure and dose measures. These include tables of person-minutes at various exposure levels, by microenvironment, a table of person-days at or above each average daily exposure level, and tables describing the distributions of exposures for different groups. An example of how APEX results can be depicted is given in Figure 3, which shows the percent of children with at least one 8-hour average exposure at or above different exposure levels, concomitant with moderate or greater exertion. These are results from a simulation of O_3 exposures for the greater Washington, D.C. metropolitan area for the year 2002. From this graph ones sees, for example, that APEX estimates 30 percent of the children in this area experience exposures above 0.08 ppm-8hr while exercising, at least once during the year.

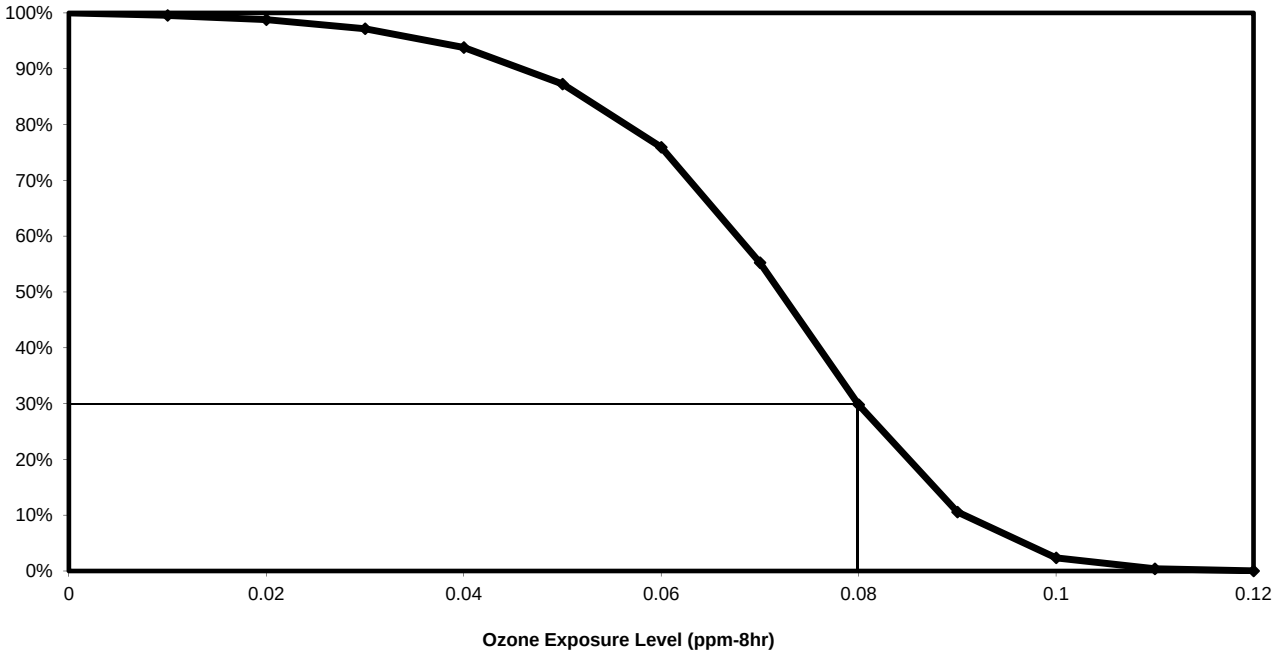

Figure 3. The Percent of Simulated Children (ages 5-18) at or above 8-hour Average O_3 Exposure Levels While Exercising.

Appendix 5B

Inputs to the APEX Exposure Model

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The APEX model inputs require extensive analysis and preparation in order to ensure the model run gives valid and relevant results. This Appendix describes preparation and the sources of data for the APEX input files.

5B-1. POPULATION DEMOGRAPHICS

APEX takes population characteristics into account to develop accurate representations of study area demographics. Population counts and employment probabilities by age and gender are used to develop representative profiles of hypothetical individuals for the simulation. Tract-level population counts by age in one-year increments, from birth to 99 years, come from the 2000 Census of Population and Housing Summary File 1. The Summary File 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. Employment data from the 2000 census can be found on the U.S. census web site at the address http://www.census.gov/population/www/cen2000/phc-t28.html (Employment Status: 2000-Supplemental Tables). The file input to APEX is 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 >75. Children under 16 years of age are assumed to be not employed.

5B-2. POPULATION COMMUTING PATTERNS

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.

Commuting data were originally derived from the 2000 Census and were collected as part of the Census Transportation Planning Package (CTPP). These data are available from the U.S. DOT Bureau of Transportation Statistics (BTS) at the web site http://transtats.bts.gov/. 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 were 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.

Commuting within the Home Tract

The APEX data set does not differentiate people that work at home from those that commute within their home tract.

Commuting Distance Cutoff

A preliminary data analysis of the home-work counts showed that a graph of log(flows) versus log(distance) had a near-constant slope out to a distance of around 120 kilometers. Beyond that distance, the relationship also had a fairly constant slope but it was flatter, meaning that flows were not as sensitive to distance. A simple interpretation of this result is that up to 120 km, the majority of the flow was due to persons traveling back and forth daily, and the numbers of such persons decrease fairly rapidly with increasing distance. Beyond 120 km, the majority of the flow is made up of persons who stay at the workplace for extended times, in which case the separation distance is not as crucial in determining the flow.

To apply the home-work data to commuting patterns in APEX, a simple rule was chosen. It was assumed that all persons in home-work flows up to 120 km are daily commuters, and no persons in more widely separated flows commute daily. This meant that the list of destinations for each home tract was restricted to only those work tracts that are within 120 km of the home tract. When the same cutoff was performed on the 1990 census data, it resulted in 4.75% of the home-work pairs in the nationwide database being eliminated, representing 1.3% of the workers. The assumption is that this 1.3% of workers do not commute from home to work on a daily basis. It is expected that the cutoff reduced the 2000 data by similar amounts.

Eliminated Records

A number of tract-to-tract pairs were eliminated from the database for various reasons. A fair number of tract-to-tract pairs represented workers who either worked outside of the U.S. (9,631 tract pairs with 107,595 workers) or worked in an unknown location (120,830 tract pairs with 8,940,163 workers). An additional 515 workers in the commuting database whose data were missing from the original files, possibly due to privacy concerns or errors, were also deleted.

APEX allows the user to specify how to handle individuals who commute to destinations outside the study area. For this application, we do not simulate those individuals, since we have not estimated ambient concentrations of O_3 in counties outside of the modeled areas.

5B-3. ASTHMA PREVALENCE RATES

One of the important population subgroups for the exposure assessment is asthmatic children. Evaluation of the exposure of this group with APEX requires the estimation of children's asthma prevalence rates. The estimates are based on children's asthma prevalence data from the National Health Interview Survey (NHIS). A detailed description of how the NHIS data were processed for input to APEX is provided in Appendix 5C.

5B-4. HUMAN ACTIVITY DATA

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 (EPA, 2002). 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 1 summarizes the studies in CHAD used in this modeling analysis, providing over 38,000 diary-days of activity data (over 13,000 diary-days for ages 5-18) collected between 1982 and 2009. In general, the surveys have a data foundation based on daily diaries of human activity. This is the foundation from which CHAD was created. Individuals filled out diaries of their daily activities and this information was input 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.

A key issue in this assessment is the development of an approach for creating O_3-season or year-long activity sequences for individuals based on a cross-sectional activity data base of 24-hour records. The typical subject in the time/activity studies in CHAD provided less than two days of diary data. For this reason, the construction of a season-long activity sequence for each individual requires some combination of repeating the same data from one subject and using data from multiple subjects. 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. The method in APEX for creating longitudinal diaries was designed to capture the tendency of individuals to repeat activities, based on reproducing realistic variation in a key diary variable, which is a user-selected function of diary variables. For this analysis the key variable is set to the amount of time an individual spends outdoors each day, which is one of the most important determinants of exposure to high levels of O_3.

The actual diary construction method targets two statistics, a population diversity statistic (***D***) and a within-person autocorrelation statistic (***A***). The ***D*** statistic reflects the relative importance of within-person variance and between-person variance in the key variable. The ***A*** statistic quantifies the lag-one (day-to-day) key variable autocorrelation. Desired ***D*** and ***A*** values for the key variable are selected by the user and set in the APEX parameters file, and the method algorithm constructs longitudinal diaries that preserve these parameters. Longitudinal diary data from a field study of children ages 7-12 (Geyh et al., 2000; Xue et al., 2004) estimated values of approximately 0.2 for ***D*** and 0.2 for ***A***. In the absence of data for estimating these statistics for younger children and others outside the study age range, and since APEX tends to underestimate repeated activities, values of 0.5 for ***D*** and 0.2 for ***A*** are used for all ages.

CHAD Updates Since The Previous Ozone Review

Since the time of the prior O_3 NAAQS review conducted in 2007, there have been a number new data sets incorporated into CHAD and used in our current exposure assessment, most of which were from recently conducted studies. The data from these six additional studies incorporated in CHAD have more than doubled the total activity pattern data used in the 2007 O_3 exposure modeling. The studies from which these new data were derived are briefly described below.

- **UMC and ISR**. These diaries are from phase I (1997) and phase II (2002-03) of the University of Michigan's Panel Study of Income Dynamics (PSID), respectively

(University of Michigan, 2012). Activity pattern data were collected from nearly 10,000 children ages 0-13 (phase I) and 5-19 (phase II) across the U.S. For each child, diary data were collected on two nonconsecutive days in a single week, in no particular season, though mostly occurring during the spring and fall (phase I), and winter (phase II) months.

- **NSA**. The diaries were collected as part of the National Scale Activity Survey (NSAS), an EPA-funded study of averting behavior related to air quality alerts (Knowledge Networks, 2009). Data were collected from about 1,200 adults aged 35-92 in seven metropolitan areas (Atlanta, St. Louis, Sacramento, Washington DC, Dallas, Houston, and Philadelphia). Data were collected over 1-15 (partially consecutive) days across the 2009 ozone season, totaling approximately 7,000 person days of data.

- **OAB.** These diaries were collected in a study of children's activities on high and low ozone days during the 2002 ozone season (Mansfield et al., 2009). Children from 35 U.S. metropolitan areas having the worst O_3 pollution households were studied, of whom about half of the children were asthmatics. Activity data were collected on 6 nonconsecutive days from each subject, with some subjects providing fewer days, totaling nearly 3,000 persons days of data.

- **SEA**. These diaries are from a PM exposure study of susceptible populations living in Seattle, WA between 1999 to 2002 (Liu et al., 2003). Two cohorts were studied: an older adult group with either chronic obstructive pulmonary disease (COPD) or coronary heart disease and a child group with asthma. Activity data were collected on 10 consecutive days from each subject, with some subjects providing fewer days. Over 1,300 daily diaries were collected from the adult group and more than 300 from the children cohort.

- **RTP**. These diaries were collected in a panel study of PM exposure in the Research Triangle Park, NC area (Williams et al., 2003a, b). Two older adult cohorts (ages 55-85) were studied: a cohort having implanted cardiac defibrillators living in Chapel Hill, NC and a second group of 30 people having controlled hypertension and residing in a low-to-moderate SES neighborhood in Raleigh, NC. Data were collected on approximately 8 consecutive days in 4 consecutive seasons in 2000-2001. A total of 1000 diary-days are included.

Table 1. Studies in the Consoloidated Human Activity Database (CHAD)

Study name	Geographic coverage	Study time period	Subject ages	Diary-days	Diary-days (ages 5-18)	Diary type and study design	Reference
Baltimore Retirement Home Study (EPA)	One building in Baltimore	01/1997-02/1997, 07/1998-08/1998	72 - 93	391	0	Diary	Williams et al. (2000)
California Youth Activity Patterns Study (CARB)	California	10/1987-09/1988	12 - 17	181	181	Recall; Random	Robinson et al. (1989), Wiley et al. (1991a)
California Adults Activity Patterns Study (CARB)	California	10/1987-09/1988	18 - 94	1,548	36	Recall; Random	Robinson et al. (1989), Wiley et al. (1991a)
California Children Activity Patterns Study (CARB)	California	04/1989- 02/1990	<1 - 11	1,200	683	Recall; Random	Wiley et al. (1991b)
Cincinnati Activity Patterns Study (EPRI)	Cincinnati metro. area	03/1985-04/1985, 08/1985	<1 - 86	2,597	738	Diary; Random	Johnson (1989)
Denver CO Personal Exposure Study (EPA)	Denver metro. area	11/1982- 02/1983	18 - 70	796	7	Diary; Random	Johnson (1984), Akland et al. (1985)
Los Angeles Ozone Exposure Study: Elementary School	Los Angeles	10/1989	10 - 12	49	49	Diary	Spier et al. (1992)
Los Angeles Ozone Exposure Study: High School	Los Angeles	09/1990-10/1990	13 - 17	42	42	Diary	Spier et al. (1992)

National Human Activity Pattern Study (NHAPS): Air	National	09/1992-10/1994	<1 - 93	4,338	634	Recall; Random	Klepeis et al. (1996), Tsang and Klepeis (1996)
National Human Activity Pattern Study (NHAPS): Water	National	09/1992-10/1994	<1 - 93	4,347	691	Recall; Random	Klepeis et al. (1996), Tsang and Klepeis (1996)
National Study of Avoidance of S (NSAS)	7 U.S. metropolitan areas	06/2009-09/2009	35 - 92	6,824	0	Recall; Random	Knowledge Networks (2009)
Population Study of Income Dynamics PSID CDS I (Univ. Michigan I)	National	02/1997-12/1997	<1 - 13	4,988	3,093	Recall; Random	University of Michigan (2012)
Population Study of Income Dynamics PSID CDS II (Univ. Michigan II)	National	01/2002-12/2003	5 - 19	4,773	4,763	Recall; Random	University of Michigan (2012)
RTI Ozone Averting Behavior	35 U.S. metropolitan areas	07/2002-08/2003	2 - 12	2,876	1,944	Recall; Random	Mansfield et al. (2006, 2009)
RTP Panel (EPA)	RTP, NC	06/2000-05/2001	55 - 85	1,000	0	Diary; Panel	Williams et al. (2003a,b)
Seattle	Seattle, WA	10/1999-03/2002	6 - 91	1,688	318	Diary; Panel	Liu et al. (2003)
Washington, D.C. (EPA)	Wash., D.C. metro. area	11/1982-02/1983	18 - 71	695	11	Diary; Random	Hartwell et al. (1984), Akland et al. (1985)
Totals		**1982 - 2009**	**<1 - 94**	**38,333**	**13,190**		

1

5B-5. PHYSIOLOGICAL DATA

APEX requires values for several physiological parameters for subjects in order to accurately model their metabolic processes that affect pollutant intake. This is because physiological differences may cause people with the same exposure and activity scenarios to have different pollutant intake levels. The physiological parameters file distributed with APEX contains physiological data or distributions by age and gender for maximum ventilatory capacity (in terms of age- and gender-specific maximum oxygen consumption potential), body mass, resting metabolic rate, and oxygen consumption-to-ventilation rate relationships.

Also input to APEX are metabolic information for different activities listed in the diary file. These metabolic activity levels are in the form of distributions. Some activities are specified as a single point value (for instance, sleep), while others, such as athletic endeavors or manual labor, are normally, lognormally, or otherwise statistically distributed. APEX samples from these distributions and calculates values to simulate the variable nature of activity levels among different people.

5B-6. MICROENVIRONMENTS MODELED

In APEX, microenvironments provide the exposure locations for modeled individuals. For exposures to be accurately estimated, it is important to have realistic microenvironments that are matched closely to where people are physically located on a daily and hourly basis. As discussed in Appendix 5A, the two methods available in APEX for calculating pollutant concentrations within microenvironments are a mass balance model and a transfer factor approach. Table 2 lists the 28 microenvironments selected for this analysis and the exposure calculation method for each. The parameters used in this analysis for modeling these microenvironments are described in this section.

Table 2. Microenvironments modeled

	Microenvironment	Calculation Method	Parameters[1]
1	Indoor – Residence	Mass balance	AER and DE
2	Indoor – Community Center or Auditorium	Mass balance	AER and DE
3	Indoor – Restaurant	Mass balance	AER and DE
4	Indoor – Hotel, Motel	Mass balance	AER and DE
5	Indoor – Office building, Bank, Post office	Mass balance	AER and DE
6	Indoor – Bar, Night club, Café	Mass balance	AER and DE
7	Indoor – School	Mass balance	AER and DE
8	Indoor – Shopping mall, Non-grocery store	Mass balance	AER and DE
9	Indoor – Grocery store, Convenience store	Mass balance	AER and DE
10	Indoor – Metro-Subway-Train station	Mass balance	AER and DE
11	Indoor – Hospital, Medical care facility	Mass balance	AER and DE
12	Indoor – Industrial, factory, warehouse	Mass balance	AER and DE
13	Indoor – Other indoor	Mass balance	AER and DE
14	Outdoor – Residential	Factors	None
15	Outdoor – Park or Golf course	Factors	None
16	Outdoor – Restaurant or Café	Factors	None
17	Outdoor – School grounds	Factors	None
18	Outdoor – Boat	Factors	None
19	Outdoor – Other outdoor non-residential	Factors	None
20	Near-road – Metro-Subway-Train stop	Factors	PR
21	Near-road – Within 10 yards of street	Factors	PR
22	Near-road – Parking garage (covered or below ground)	Factors	PR
23	Near-road – Parking lot (open), Street parking	Factors	PR
24	Near-road – Service station	Factors	PR
25	Vehicle – Cars and Light Duty Trucks	Factors	PE and PR
26	Vehicle – Heavy Duty Trucks	Factors	PE and PR
27	Vehicle – Bus	Factors	PE and PR
28	Vehicle – Train, Subway	Factors	PE and PR

[1] AER=air exchange rate, DE=decay-deposition rate, PR=proximity factor, PE=penetration factor

5B-7. AIR EXCHANGE RATES FOR INDOOR RESIDENTIAL ENVIRONMENTS

Distributions of AERs for the indoor microenvironments were developed using data from several studies. The analysis of these data and the development of the distributions used in the modeling are described in detail in EPA (2007) Appendix A. This analysis showed that the AER distributions for the residential microenvironments depend on the type of air conditioning (A/C) and on the outdoor temperature, as well as other variables for which we do not have sufficient data to estimate. This analysis clearly demonstrates that the AER distributions vary greatly across cities and A/C types and temperatures, so that the selected AER distributions for the modeled cities should also depend upon the city, A/C type, and temperature. For example, the mean AER for residences with A/C ranges from 0.39 for Los Angeles between 30 and 40 ºC to 1.73 for New York between 20 and 25 ºC. The mean AER for residences without A/C ranges from 0.46 for San Francisco on days with temperature between 10 and 20 ºC to 2.29 for New York on days with temperature between 20 and 25 ºC. The need to account for the city as well as the A/C type and temperature is illustrated by the result that for residences with A/C on days with temperature between 20 and 25 ºC, the mean AER ranges from 0.52 for Research Triangle Park to 1.73 for New York. For each combination of A/C type, city, and temperature with a minimum of 11 AER values, exponential, lognormal, normal, and Weibull distributions were fit to the AER values and compared. Generally, the lognormal distribution was the best-fitting of the four distributions, and so, for consistency, the fitted lognormal distributions are used for all the cases.

One limitation of this analysis was that distributions were available only for selected cities, and yet the summary statistics and comparisons demonstrate that the AER distributions depend upon the city as well as the temperature range and A/C type. Another important limitation of the analysis was that distributions were not able to be fitted to all of the temperature ranges due to limited data in these ranges. A description of how these limitations were addressed can be found in EPA (2007) Appendix A.

City-specific AER distributions were used where possible; otherwise data for a similar city were used. The AER distributions used for the exposure modeling are given in Table 3 (Atlanta), Table 4 (Denver and Philadelphia), and Table 5 (Los Angeles).

Table 3. AERs for Atlanta (Indoors – residences)

Microenvironment	Conditions [a]		Distribution
	°F	A/C	(GM, GSD, min, max)
Indoors - residences	< 50	yes	Lognormal(0.962, 1.809, 0.1, 10)
	50 - 67	yes	Lognormal(0.562, 1.906, 0.1, 10)
	68 - 76	yes	Lognormal(0.397, 1.889, 0.1, 10)
	> 76	yes	Lognormal(0.380, 1.709, 0.1, 10)
	< 50	no	Lognormal(0.926, 2.804, 0.1, 10)
	50 - 67	no	Lognormal(0.733, 2.330, 0.1, 10)
	> 67	no	Lognormal(1.378, 2.276, 0.1, 10)

[a] Average daily temperature range (°F) and presence or absence of air conditioning

Table 4. AERs for Denver and Philadelphia (Indoors – residences)

Microenvironment	Conditions [a]		Distribution
	°F	A/C	(GM, GSD, min, max)
Indoors - residences	< 50	yes	Lognormal(0.711, 2.018, 0.1, 10)
	50 - 76	yes	Lognormal(1.139, 2.677, 0.1, 10)
	> 76	yes	Lognormal(1.244, 2.177, 0.1, 10)
	< 50	no	Lognormal(1.016, 2.138, 0.1, 10)
	50 - 67	no	Lognormal(0.791, 2.042, 0.1, 10)
	> 67	no	Lognormal(1.606, 2.119, 0.1, 10)

[a] Average daily temperature range (°F) and presence or absence of air conditioning

Table 5. AERs for Los Angeles (Indoors – residences)

Microenvironment	Conditions [a]		Distribution
	°F	A/C	(GM, GSD, min, max)
Indoors - residences	< 68	Central	Lognormal(0.577, 1.897, 0.1, 10)
	68 – 76	Central	Lognormal(1.084, 2.336, 0.1, 10)
	> 76	Central	Lognormal(0.861, 2.344, 0.1, 10)
	< 68	Room	Lognormal(0.672, 1.863, 0.1, 10)
	68 – 76	Room	Lognormal(1.674, 2.223, 0.1, 10)
	> 76	Room	Lognormal(0.949, 1.644, 0.1, 10)
	< 68	None	Lognormal(0.744, 2.057, 0.1, 10)
	68 – 76	None	Lognormal(1.448, 2.315, 0.1, 10)
	> 76	None	Lognormal(0.856, 2.018, 0.1, 10)

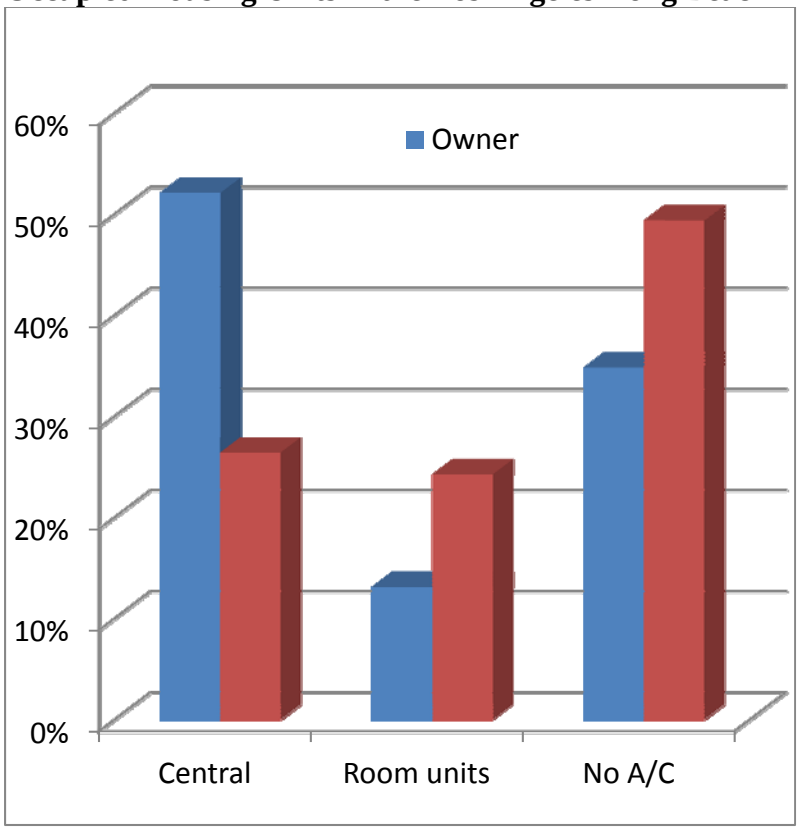
[a] Average daily temperature range (°F) and type of air conditioning

5B-8. AIR CONDITIONING PREVALENCE

In previous applications of APEX, we obtained A/C prevalence from the American Housing Survey (AHS), at the level of the metropolitan area. For this application, we take

advantage of A/C differentials between owner-occupied and rental housing to estimate A/C prevalence at the Census tract level. In this first draft REA, we have done this additional breakdown for Los Angeles only; in the next draft, this will be done for all cities. For example, the AHS data for A/C prevalence in Los Angeles[1] finds that owner-occupied housing units have 52% central A/C, while rental units have 26% central A/C. For housing units with no central and one window A/C, the owner-occupied prevalence is 9% and the rentals 21% (Figure 1). The net results of this is that owner-occupied housing tends to be much more airtight than rentals in Los Angeles.

Figure 1. Air Conditioning Prevalence for Owner- and Renter-Occupied Housing Units in the Los Angeles-Long Beach Area in 2003



Data from the American Housing Survey for the Los Angeles Metropolitan Area in 2003, Current Housing Reports, Table 1-4

Since APEX is able to read in tract-level data, such as A/C prevalence, distance to roadways, etc., and use these as conditional variables for microenvironmental distributions, we use tract-level information on owner-occupied and rental housing units, together with the

[1] Table 1-4. Selected Equipment and Plumbing – All Housing Units. American Housing Survey for the Los Angeles Metropolitan Area in 2003, U.S. Department of Housing and Urban Development and U.S. Census Bureau.

corresponding AHS breakdown for each urban area (Table 6), and obtain tract-level variation in A/C prevalence.

5B-9. AER DISTRIBUTIONS FOR OTHER INDOOR ENVIRONMENTS

To estimate AER distributions for non-residential, indoor environments (e.g., offices and schools), we obtained and analyzed two AER data sets: "Turk" (Turk et al., 1989); and "Persily" (Persily and Gorfain, 2004; Persily et al., 2005). The Turk data set includes 40 AER measurements from offices (25 values), schools (7 values), libraries (3 values), and multi-purpose buildings (5 values), each measured using an SF_6 tracer over two or four hours in different seasons of the year. The Persily data were derived from the U.S. EPA Building Assessment Survey and Evaluation (BASE) study, which was conducted to assess indoor air quality, including ventilation, in a large number of randomly selected office buildings throughout the U.S. This data base consists of a total of 390 AER measurements in 96 large, mechanically ventilated offices. AERs were measured both by a volumetric method and by a CO_2 ratio method, and included their uncertainty estimates. For these analyses, we used the recommended "Best Estimates" defined by the values with the lower estimated uncertainty; in the vast majority of cases the best estimate was from the volumetric method.

Due to the small sample size of the Turk data, the data were analyzed without stratification by building type and/or season. For the Persily data, the AER values for each office space were averaged, rather using the individual measurements, to account for the strong dependence of the AER measurements for the same office space over a relatively short period. The mean values are similar for the two studies, but the standard deviations are about twice as high for the Persily data. We fitted exponential, lognormal, normal, and Weibull distributions to the 96 office space average AER values from the more recent Persily data, and the best fitting of these was the lognormal. The fitted parameters for this distribution are a geometric mean of 1.109 and a geometric standard deviation of 3.015. These are used for AER distributions for the indoor non-residential microenvironments, except for restaurants, bars, night clubs, and cafés.

Table 6. American Housing Survey A/C prevalence from Current Housing Reports Table 1-4 For Selected Urban Areas (Total: seasonal, occupied, vacant) (housing units in thousands)

Metropolitan area	Area	Years	Total housing units	Central A/C	additional central	1 room unit	2 room units	3+ room units	Percent central A/C	Percent window units	Sum of %central & %window
Atlanta	MA	2004	1802.8	1649.5	265.9	47.8	34.5	18.9	91	6	97
Boston	CMSA	2007	1151.0	307.6	20.3	275.5	202.0	157.8	27	55	82
Chicago	PMSA	2003	3198.9	1919.6	87.6	500.8	340.5	102.8	60	30	90
		2009	3010.7	2050.6	116.2	412.0	265.1	124.4	68	27	95
Cleveland	PMSA	2004	856.1	439.5	14.8	143.8	48.2	17.6	51	24	76
Dallas	PMSA	2002	1365.4	1256.9	185.3	31.8	32.1	29.6	92	7	99
Ft. Worth - Arlington	PMSA	2002	639.4	556.0	70.5	19.9	26.6	24.4	87	11	98
Denver	MA	2004	949.1	469.7	18.6	138.0	22.6	4.1	49	17	67
Detroit	PMSA	2003	1900.6	1157.4	39.4	261.3	106.0	39.8	61	21	82
		2009	1672.5	1194.3	46.5	192.3	82.8	29.2	71	18	90
Houston	PMSA	2007	2160.1	1924.4	167.8	59.1	67.8	62.9	89	9	98
Los Angeles-Long Beach	PMSA	2003	3318.5	1284.8	84.6	495.5	80.0	43.7	39	19	57
Riverside-San Bernardino-Ontario	PMSA	2002	1229.5	866.5	68.2	123.8	31.2	5.0	70	13	83
Anaheim - Santa Ana	PMSA	2002	995.6	472.1	25.8	134.7	13.7	4.7	47	15	63
New York-Nassau-Suffolk-Orange	PMSA	2003	4849.8	794.6	50.2	1401.5	1155.7	690.3	16	67	83
		2009	4493.3	872.4	38.2	1036.9	1184.1	812.6	19	68	87
Northern NJ	PMSA	2003	2589.1	1184.3	70.2	460.0	429.3	324.5	46	47	93
		2009	2681.7	1334.4	106.7	318.0	412.2	375.1	50	41	91
Philadelphia	PMSA	2003	2068.8	1001.8	54.6	328.1	317.0	241.1	48	43	91
		2009	2122.2	1169.4	56.1	225.8	269.9	275.2	55	36	91
Sacramento	PMSA	2004	727.5	581.4	32.4	62.7	12.6	2.4	80	11	91
St. Louis	MA	2004	1139.6	974.4	53.7	65.8	43.5	16.6	86	11	97
Seattle-Everett	PMSA	2004	1075.6	77.9	1.6	56.9	14.8	6.4	7	7	15
		2009	1331.7	172.7	6.7	121.8	27.5	8.6	13	12	25
Washington, DC	MA	2007	2133.5	1881.3	150.8	76.9	69.0	66.8	88	10	98
Baltimore	MSA	2007	1109.6	828.8	46.2	63.7	76.5	66.3	75	19	93

MA – metropolitan area; CMSA – consolidated metropolitan statistical area; PMSA – primary metropolitan statistical area.

The AER distribution used for schools is a discrete distribution with values (0.8 1.3 1.8 2.19 2.2 2.21 3.0 0.6 0.1 0.6 0.2 1.8 1.3 1.2 2.9 0.9 0.9 0.9 0.9 0.4 0.4 0.4 0.4 0.9 0.9 0.9 0.9 0.3 0.3 0.3 0.3), taken from from Turk et al., 1989 and Shendell et al., 2004.

The AER distribution used for restaurants, bars, night clubs, and cafés is a discrete distribution with values (1.46 2.64 5.09 9.07 4.25 3.46), from Bennett et al., 2012, who measured these six values in restaurants. This distribution is also used for the Bar, Night club, and Café microenvironments.

5B-10. PROXIMITY AND PENETRATION FACTORS FOR OUTDOORS AND IN-VEHICLE MICROENVIRONMENTS

For the outdoors near-road, public garage/parking lot, and in-vehicle proximity factors, and for the in-vehicle penetration factors, we use distributions developed from the Cincinnati Ozone Study (American Petroleum Institute, 1997, Appendix B; Johnson et al., 1995). This field study was conducted in the greater Cincinnati metropolitan area in August and September, 1994. Vehicle tests were conducted according to an experimental design specifying the vehicle type, road type, vehicle speed, and ventilation mode. Vehicle types were defined by the three study vehicles: a minivan, a full-size car, and a compact car. Road types were interstate highways (interstate), principal urban arterial roads (urban), and local roads (local). Nominal vehicle speeds (typically met over one minute intervals within 5 mph) were at 35 mph, 45 mph, or 55 mph. Ozone concentrations were measured inside the vehicle, outside the vehicle, and at six fixed-site monitors in the Cincinnati area. Table 7 lists the distributions developed for penetration and proximity factors for in-vehicle microenvironments, which are used in this modeling analysis.

Table 7. Distributions of penetration and proximity factors for in-vehicle microenvironments

Gaussian distributions	Mean	Standard deviation
Penetration factors	0.300	0.232
Proximity factors		
local roads	0.755	0.203
urban roads	0.754	0.243
interstate roads	0.364	0.165

The Vehicle Miles Of Travel (VMT) fractions (Table 8, summarized from the U.S. Department of Transportation, Federal Highway Administration annual *Highway Statistics* reports, Tables HM-71) are used as conditional variables, which determine selection of the proximity factor distributions for in-vehicle microenvironments. For local and interstate road types, the VMT for the same Department of Transportation (DOT) categories are used. For urban roads, the VMT for all other DOT road types are summed (Other freeways/expressways, Other principal arterial, Minor arterial, Collector). At the time of this writing, data were only available for three of our modeled years, 2006-2008. We are assuming that 2009 and 2010 would be best represented by 2008. We plan to use the 2009 and 2010 statistics in the second draft REA if they are available.

Table 8. VMT fractions of interstate, urban and local roads in the study areas

City	2006			2007			2008		
	inter-state	urban	local	inter-state	urban	local	inter-state	urban	local
Atlanta	0.34	0.46	0.20	0.34	0.47	0.19	0.32	0.45	0.23
Baltimore	0.34	0.59	0.07	0.34	0.59	0.07	0.34	0.59	0.07
Boston	0.32	0.55	0.13	0.32	0.55	0.13	0.32	0.54	0.14
Chicago	0.30	0.58	0.12	0.30	0.58	0.12	0.31	0.57	0.12
Cleveland	0.40	0.44	0.16	0.40	0.44	0.16	0.39	0.45	0.16
Dallas	0.30	0.66	0.04	0.30	0.66	0.04	0.30	0.65	0.05
Denver-Aurora	0.23	0.67	0.10	0.24	0.66	0.10	0.25	0.65	0.10
Detroit	0.25	0.65	0.10	0.25	0.65	0.10	0.24	0.66	0.10
Houston	0.24	0.72	0.04	0.24	0.72	0.04	0.24	0.73	0.03
Los Angeles-Long Beach-Santa Ana	0.29	0.66	0.05	0.29	0.67	0.04	0.28	0.67	0.05
New York-Newark	0.19	0.66	0.15	0.19	0.65	0.16	0.19	0.66	0.15
Philadelphia	0.23	0.65	0.12	0.24	0.65	0.11	0.24	0.65	0.11
Sacramento	0.25	0.72	0.03	0.24	0.70	0.06	0.24	0.69	0.08
Seattle	0.29	0.60	0.11	0.29	0.60	0.11	0.29	0.60	0.11
St. Louis	0.36	0.45	0.19	0.37	0.45	0.18	0.37	0.45	0.18
Washington, DC	0.30	0.62	0.08	0.31	0.61	0.08	0.30	0.62	0.08

U.S. Department of Transportation, Federal Highway Administration. Annual *Highway Statistics*, Table HM-71: Urbanized Areas - Miles And Daily Vehicle Miles Of Travel. Some fractions have been adjusted so the three fractions sum to 1.00.

5B-11. OZONE DECAY AND DEPOSITION RATES

A distribution for combined O_3 decay and deposition rates was obtained from the analysis of measurements from a study by Lee et al. (1999). This study measured decay rates in the living rooms of 43 residences in Southern California. Measurements of decay rates in a second room were made in 24 of these residences. The 67 decay rates range from 0.95 to 8.05 $hour^{-1}$. A lognormal distribution was fit to the measurements from this study, yielding a geometric mean of 2.5 and a geometric standard deviation of 1.5. These values are constrained to lie between 0.95 and 8.05 $hour^{-1}$. This distribution is used for all indoor microenvironments.

5B-12. AMBIENT OZONE CONCENTRATIONS

APEX requires hourly ambient O_3 concentrations at a set of locations in the study area. Data from EPA's AIRS Air Quality System (AQS) were used to prepare the ambient air quality input files for 2006 to 2010 (see REA Section 4.3). The hourly O_3 concentrations at the AIRS sites in and around each urban area were used as input to APEX to represent the ambient concentrations within each urban area. A 30 km radius of influence was used for each monitoring site. This means that the ambient concentrations assigned to a Census tract are those at the closest monitor, if that monitor is with 30 km of the center of the tract and the county is in the list of modeled counties (Table 9); otherwise, the population in that county is not modeled. Figures X to X show the monitoring sites with their 30 km radii of influence. The modeled area is the interestion of the 30 km disks with the counties specified in Table 9.

Table 9. Counties Modeled in Each Area

Urban Area (List of Counties)
Atlanta area, GA (Barrow, Bartow, Bibb, Butts, Carroll Floyd, Cherokee, Clarke, Clayton, Cobb, Coweta, Dawson, De Kalb, Douglas, Fayette, Forsyth, Fulton, Gwinnett, Hall, Haralson, Heard, Henry, Jasper, Lamar, Meriwether, Gilmer, Newton, Paulding, Pickens, Pike, Polk, Rockdale, Spalding, Troup, Upson, Walton, Chambers (AL))
Denver area, CO (Adams, Arapahoe, Boulder, Broomfield, Clear Creek, Denver, Douglas, Elbert, Gilpin, Jefferson, Park, Larimer, Weld)
Los Angeles area, CA (Los Angeles, Orange, Riverside, San Bernardino, Ventura)
Philadelphia area (Kent, DE; New Castle, DE; Sussex, DE; Cecil, MD; Atlantic, NJ; Camden, NJ; Cumberland, NJ; Gloucester, NJ; Mercer, NJ; Ocean, NJ; Berks, PA; Bucks, PA; Chester,

PA; Delaware, PA; Montgomery, PA; Philadelphia, PA)

Figure 2. Atlanta Ozone Monitors With 30 km Radii of Influence

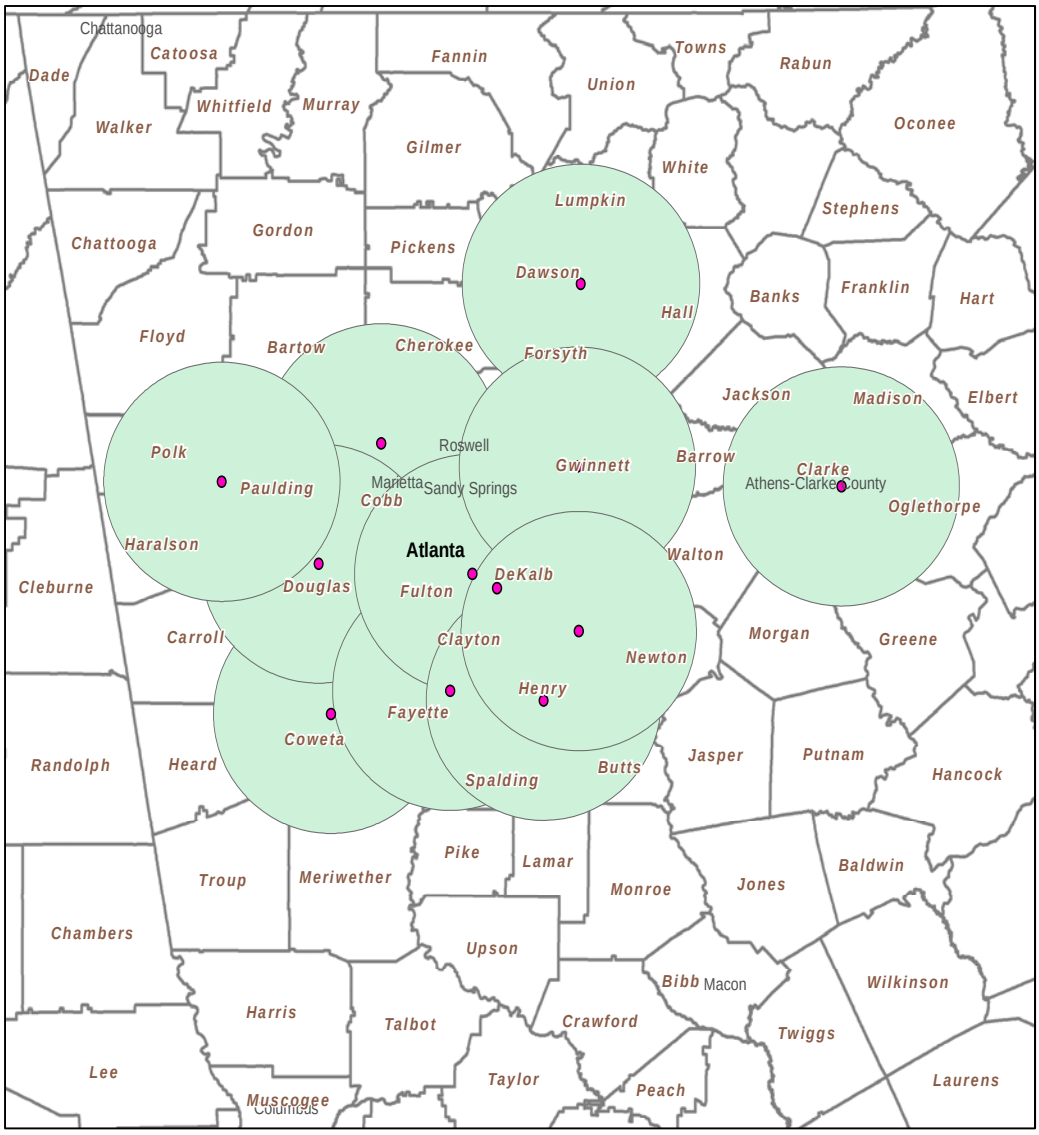


Figure 3. Denver Ozone Monitors With 30 km Radii of Influence

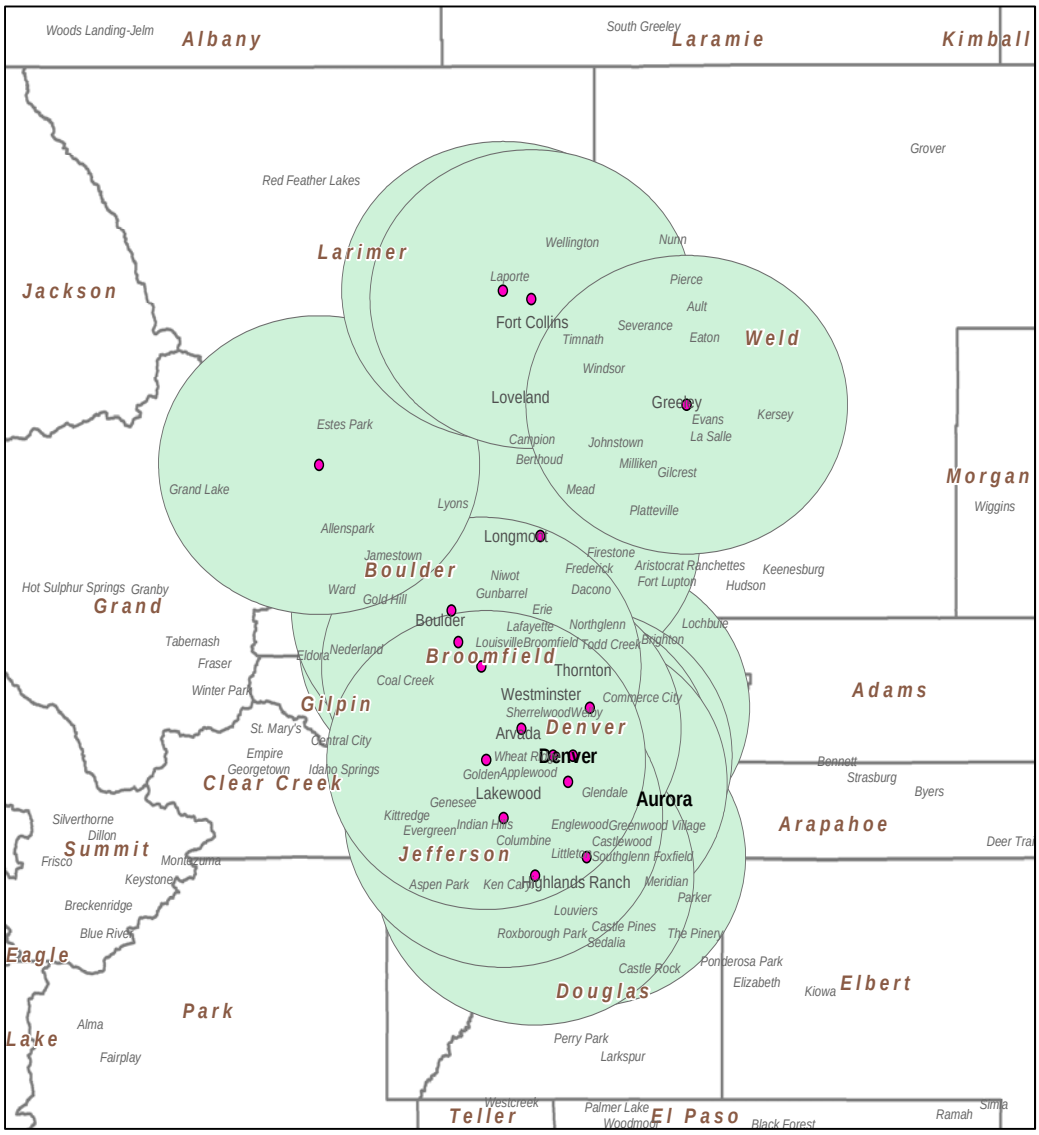


Figure 4. Los Angeles Ozone Monitors With 30 km Radii of Influence

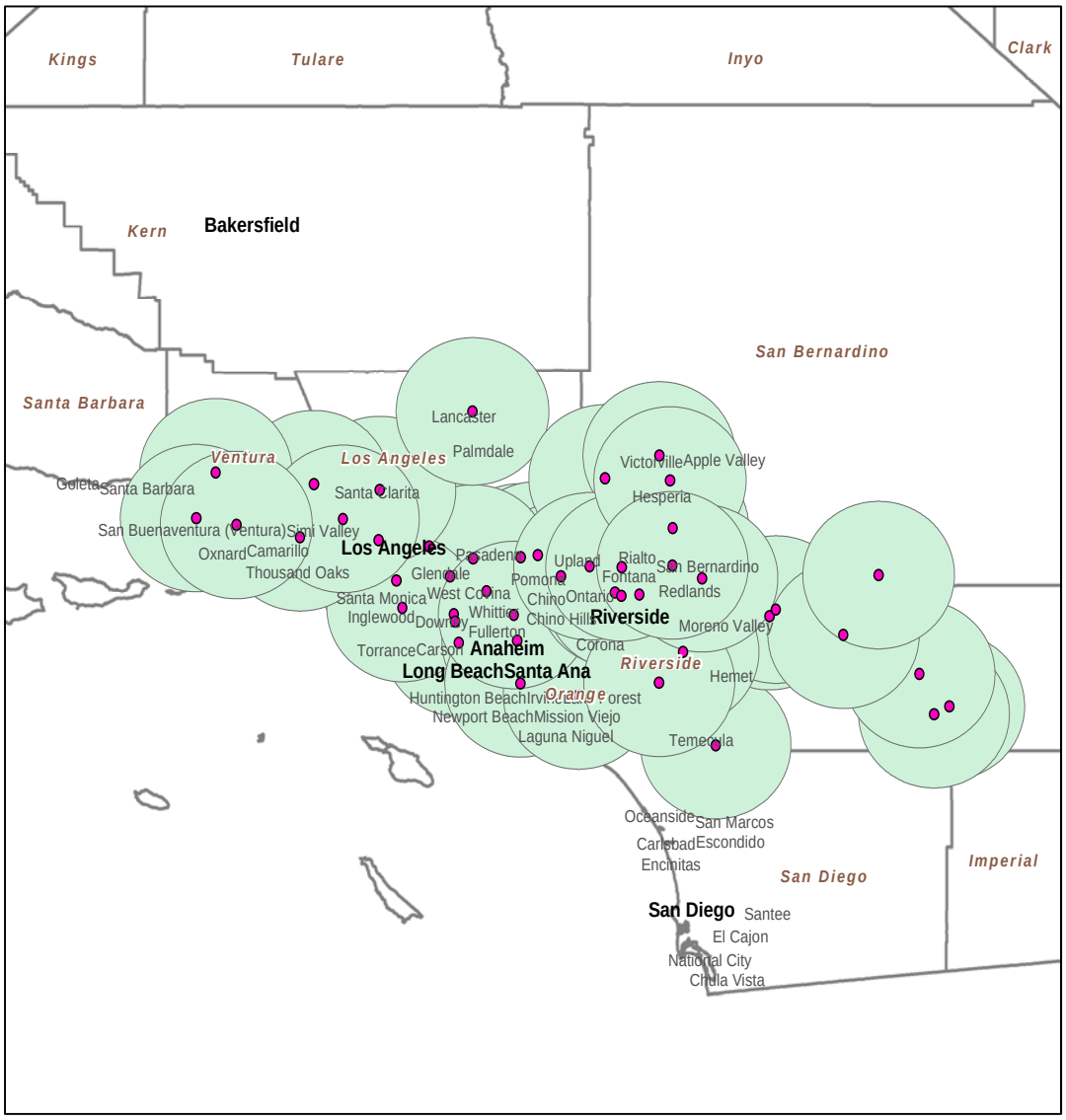
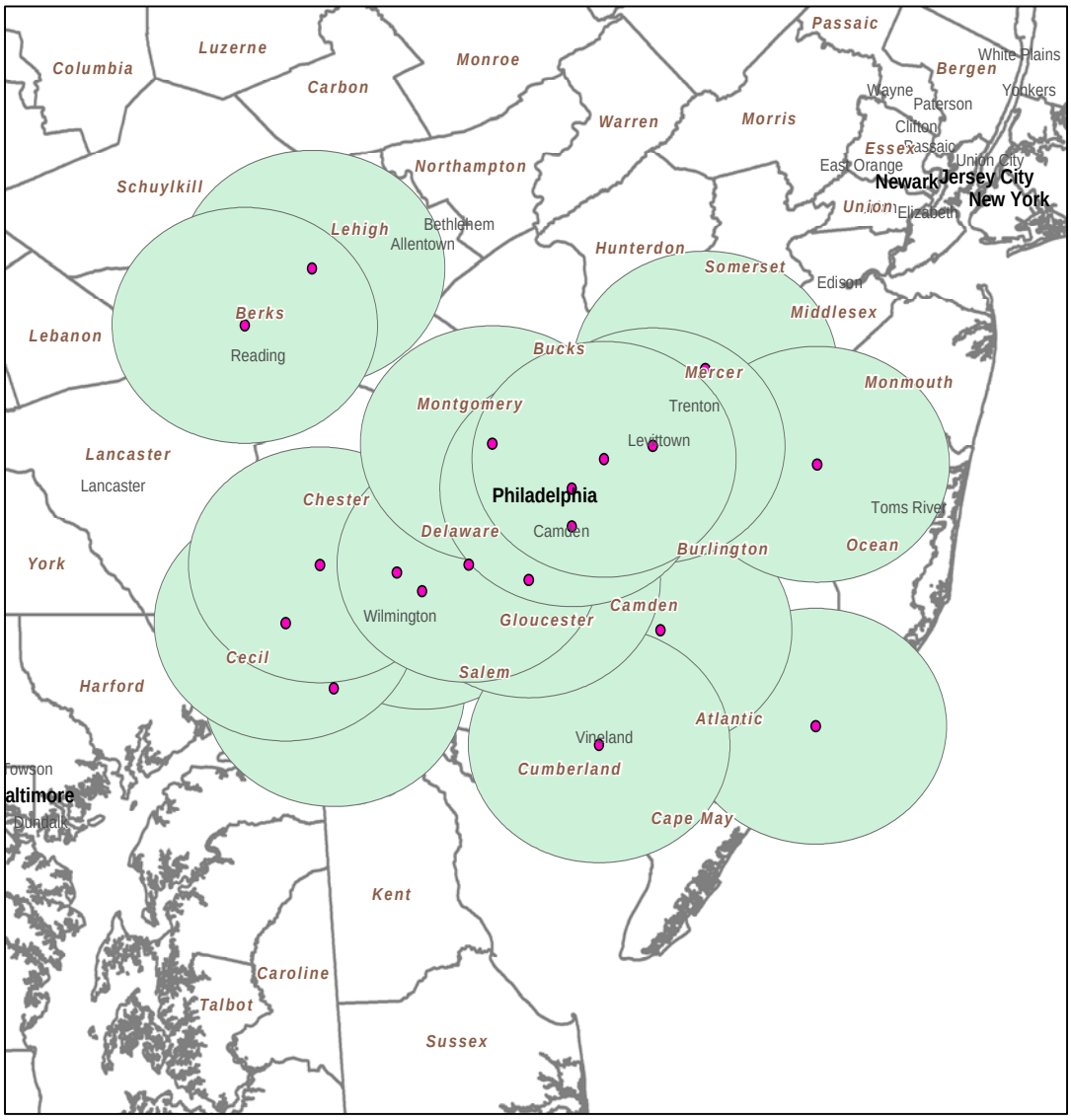


Figure 5. Philadelphia Ozone Monitors With 30 km Radii of Influence



Ozone Monitoring Sites

Tables 9 to 12 list the ozone monitoring sites that were used in this analysis.

Table 10. Atlanta ozone monitoring sites

Monitor id	County
13021-0012-1	Bibb, GA
13021-0013-1	Bibb, GA
13055-0001-1	Floyd, GA
13059-0002-1	Clarke, GA
13067-0003-1	Cobb, GA
13077-0002-1	Coweta, GA

13085-0001-2	Dawson, GA
13089-0002-1	DeKalb, GA
13089-3001-1	DeKalb, GA
13097-0004-1	Douglas, GA
13113-0001-1	Fayette, GA
13121-0055-1	Fulton, GA
13135-0002-1	Gwinnett, GA
13151-0002-1	Henry, GA
13213-0003-1	Gilmer, GA
13223-0003-1	Paulding, GA
13247-0001-1	Rockdale, GA

Table 11. Denver ozone monitoring sites

Monitor id	County
08001-3001-2	Adams, CO
08005-0002-1	Arapahoe, CO
08005-0006-1	Arapahoe, CO
08013-0011-1	Boulder, CO
08013-7001-1	Boulder, CO
08013-7002-1	Boulder, CO
08031-0002-5	Denver, CO
08031-0014-2	Denver, CO
08031-0025-1	Denver, CO
08035-0004-1	Douglas, CO
08059-0002-1	Jefferson, CO
08059-0005-1	Jefferson, CO
08059-0006-1	Jefferson, CO
08059-0011-1	Jefferson, CO
08059-0013-1	Jefferson, CO
08069-0007-1	Larimer, CO
08069-0011-1	Larimer, CO
08069-0012-1	Larimer, CO
08069-1004-1	Larimer, CO
08123-0009-1	Weld, CO

Table 12. Los Angeles ozone monitoring sites

Monitor id	County
06037-0002-1	Los Angeles, CA
06037-0016-1	Los Angeles, CA
06037-0113-1	Los Angeles, CA
06037-1002-1	Los Angeles, CA
06037-1103-1	Los Angeles, CA
06037-1201-1	Los Angeles, CA
06037-1301-1	Los Angeles, CA
06037-1302-1	Los Angeles, CA
06037-1602-1	Los Angeles, CA
06037-1701-1	Los Angeles, CA
06037-2005-1	Los Angeles, CA

06037-4002-1	Los Angeles, CA
06037-4006-1	Los Angeles, CA
06037-5005-1	Los Angeles, CA
06037-6012-1	Los Angeles, CA
06037-9033-1	Los Angeles, CA
06037-9034-1	Los Angeles, CA
06059-0007-1	Orange, CA
06059-1003-1	Orange, CA
06059-2022-1	Orange, CA
06059-5001-1	Orange, CA
06065-0004-1	Riverside, CA
06065-0008-1	Riverside, CA
06065-0009-1	Riverside, CA
06065-0012-1	Riverside, CA
06065-1004-1	Riverside, CA
06065-1010-1	Riverside, CA
06065-1016-1	Riverside, CA
06065-1999-1	Riverside, CA
06065-2002-1	Riverside, CA
06065-5001-1	Riverside, CA
06065-6001-1	Riverside, CA
06065-8001-1	Riverside, CA
06065-8005-1	Riverside, CA
06065-9001-1	Riverside, CA
06065-9003-1	Riverside, CA
06071-0001-1	San Bernardino, CA
06071-0005-1	San Bernardino, CA
06071-0012-1	San Bernardino, CA
06071-0306-1	San Bernardino, CA
06071-1001-1	San Bernardino, CA
06071-1004-2	San Bernardino, CA
06071-1234-1	San Bernardino, CA
06071-2002-1	San Bernardino, CA
06071-4001-1	San Bernardino, CA
06071-4003-1	San Bernardino, CA
06071-9002-1	San Bernardino, CA
06071-9004-1	San Bernardino, CA
06073-0001-1	San Diego, CA
06073-0003-1	San Diego, CA
06073-0006-1	San Diego, CA
06073-1001-1	San Diego, CA
06073-1002-1	San Diego, CA
06073-1006-1	San Diego, CA
06073-1008-1	San Diego, CA
06073-1010-1	San Diego, CA
06073-1011-3	San Diego, CA
06073-1016-1	San Diego, CA
06073-1201-1	San Diego, CA
06073-2007-1	San Diego, CA
06111-0007-1	Ventura, CA
06111-0009-1	Ventura, CA
06111-1004-1	Ventura, CA

06111-2002-1	Ventura, CA
06111-2003-1	Ventura, CA
06111-3001-1	Ventura, CA

Table 13. Philadelphia ozone monitoring sites

Monitor id	County
10001-0002-1	Kent, DE
10003-1007-1	New Castle, DE
10003-1010-1	New Castle, DE
10003-1013-1	New Castle, DE
10005-1002-1	Sussex, DE
10005-1003-1	Sussex, DE
24015-0003-1	Cecil, MD
34001-0005-1	Atlantic, NJ
34001-0006-1	Atlantic, NJ
34007-0003-1	Camden, NJ
34007-1001-1	Camden, NJ
34011-0007-1	Cumberland, NJ
34015-0002-1	Gloucester, NJ
34021-0005-1	Mercer, NJ
34029-0006-1	Ocean, NJ
42011-0006-1	Berks, PA
42011-0009-1	Berks, PA
42011-0010-1	Berks, PA
42011-0011-1	Berks, PA
42017-0012-1	Bucks, PA
42029-0100-1	Chester, PA
42045-0002-1	Delaware, PA
42091-0013-1	Montgomery, PA
42101-0004-1	Philadelphia, PA
42101-0014-1	Philadelphia, PA
42101-0024-1	Philadelphia, PA
42101-0136-1	Philadelphia, PA

Estimation of Missing Data

Missing air quality data were estimated by the following procedure. Where there were consecutive strings of missing values (data gaps) of 4 or fewer hours, missing values were estimated by linear interpolation between the valid values at the ends of the gap. Remaining missing values at a monitor were estimated by fitting linear regression models for each hour of the day, with each of the other monitors, and choosing the model which maximizes R^2, for each hour of the day, subject to the constraints that R^2 be greater than 0.50 and the number of regression data values (days) is at least 60. If there were any remaining missing values at this point, for gaps of 6 or fewer hours, missing values were estimated by linear interpolation

between the valid values at the ends of the gap. Any remaining missing values were replaced with the value at the closest monitoring site for that hour.

Spatial Interpolation

The O_3 concentration for each hour at each Census tract is set to the concentration at the O_3 monitor closest to the center of the Census tract. If no monitors are within 30 km of the tract center, then the persons living in that tract are not modeled. This method was used in the previous O_3 NAAQS review. In the second draft REA, we plan to perform a sensitivity analysis and compare this approach with using the prediction of a photochemical grid model to augment the monitored concentrations to create a smooth spatial surface of O_3 concentrations.

5B-1. METEOROLOGICAL DATA

Hourly surface temperature measurements were obtained from the National Weather Service ISH data files.[2] The weather stations used for each city are given in Tables 9 to 12. Missing data are estimated using the same algorithm as for missing air quality data (Section 5B.12). APEX uses the data from the closest weather station to each Census tract. Temperatures are used in APEX both in selecting human activity data and in estimating AERs for indoor microenvironments.

Table 14. Atlanta Meteorological Stations, Locations, and Hours of Missing Data

Station[a]	Latitude	Longitude	2006	2007	2008	2009	2010
722190-13874	33.633	-84.433	0	0	101	41	18
722195-03888	33.767	-84.517	14	15	113	103	29
722270-13864	33.917	-84.517	2506	1647	267	93	74
723200-93801	34.350	-85.167	14	30	187	59	68

[a] USAF ID–WBAN ID

Table 15. Denver Meteorological Stations, Locations, and Hours of Missing Data

Station	Latitude	Longitude	2006	2007	2008	2009	2010
724660-93037	38.817	-104.717	2	2	108	110	71
724666-93067	39.567	-104.850	2	1	104	53	45

[2] http://www.ncdc.noaa.gov/oa/climate/surfaceinventories.html

Station	Latitude	Longitude	2006	2007	2008	2009	2010
724695-23036	39.717	-104.750	33	42	104	53	33
725650-03017	39.833	-104.650	0	2	91	44	40

Table 16. Los Angeles Meteorological Stations, Locations, and Hours of Missing Data

Station	Latitude	Longitude	2006	2007	2008	2009	2010
722860-23119	33.900	-117.250	13	25	103	48	29
722880-23152	34.200	-118.350	2	12	152	86	37
722950-23174	33.933	-118.400	0	0	113	44	19
722970-23129	33.833	-118.167	2	4	99	173	269
723816-03159	34.733	-118.217	126	11	438	176	411
723926-23136	34.217	-119.083	21	47	311	218	139

Table 17. Philadelphia Meteorological Stations, Locations, and Hours of Missing Data

Station	Latitude	Longitude	2006	2007	2008	2009	2010
724070-93730	39.450	-74.567	4	3	142	112	161
724075-13735	39.367	-75.083	20	84	268	73	74
724080-13739	39.867	-75.233	1	0	122	57	21
724085-94732	40.083	-75.017	0	10	143	60	38
724089-13781	39.667	-75.600	22	1	156	244	89
724096-14706	40.017	-74.600	66	63	132	83	122
725170-14737	40.650	-75.450	5	4	148	74	51

1

REFERENCES

AHS (2003). U.S. Bureau of the Census and U.S. Department of Housing and Urban Development. 2003 American Housing Survey (AHS): National Survey Data. Available at: http://www.census.gov/hhes/www/housing/ahs/ahs.html, and http://www.huduser.org/datasets/ahs.html

Akland, G. G., Hartwell, T. D., Johnson, T. R., Whitmore, R. W. (1985). Measuring human exposure to carbon monoxide in Washington, D. C. and Denver, Colorado during the winter of 1982-83. *Environ Sci Technol*. 19: 911-918.

American Petroleum Institute. (1997). Sensitivity Testing of pNEM/O_3 Exposure to Changes in the Model Algorithms. Health and Environmental Sciences Department.

Bennett, D. H, W. Fisk, M. G. Apte, X. Wu, A. Trout, D. Faulkner, D. Sulivan (2012). Ventilation, Temperature, and HVAC Characteristics in Small and Medium Commercial Buildings (SMCBs) in California. *Indoor Air*. 22(4):309-320.

Geyh, A. S., Xue, J., Ozkaynak, H., Spengler, J. D. (2000). The Harvard Southern California chronic ozone exposure study: assessing ozone exposure of grade-school-age children in two southern California communities. *Environ Health Perspect*. 108: 265-270.

Hartwell, T. D., Clayton, C. A., Ritchie, R. M., Whitmore, R. W., Zelon, H. S., Jones, S. M., Whitehurst, D. A. (1984). Study of Carbon Monoxide Exposure of Residents of Washington, DC and Denver, Colorado. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development, Environmental Monitoring Systems Laboratory. EPA-600/4-84-031.

Johnson, T. (1984). A Study of Personal Exposure to Carbon Monoxide in Denver, Colorado. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Monitoring Systems Laboratory. EPA-600/4-84-014.

Johnson, T. (1989). Human Activity Patterns in Cincinnati, Ohio. Palo Alto, CA: Electric Power Research Institute. EPRI EN-6204.

Johnson, T., Pakrasi, A., Wisbeth, A., Meiners, G., Ollison, W. (1995). Ozone exposures Within Motor Vehicles – Results of a Field Study in Cincinnati, Ohio. Proceedings 88th annual meeting and exposition of the Air & Waste Management Association, San Antonio, TX. June 18-23, 1995. Preprint paper 95-WA84A.02.

Klepeis, N. E., Tsang, A. M., Behar, J. V. (1996). Analysis of the National Human Activity Pattern Survey (NHAPS) Respondents from a Standpoint of Exposure Assessment. Washington, DC: U.S. Environmental Protection Agency, Office of Research and Development. EPA/600/R-96/074.

Knowledge Networks. (2009). Field Report: National Scale Activity Survey (NSAS). Conducted for Research Triangle Institute. Submitted to Carol Mansfield November 13, 2009.

Lee, K., Vallarino, J., Dumyahn, T., Ozkaynak, H., and Spengler, J. D. (1999). Ozone decay rates in residences. *J Air Waste Manag Assoc*. 49: 1238-1244.

Liu L-JS, Box M, Kalman D, Kaufman J, Koenig J, Larson T, Lumley T, Sheppard L, Wallace L. (2003). Exposure assessment of particulate matter for susceptible populations in Seattle. *Environ Health Persp*. 111: 909-918.

Mansfield C, Houtven GV, Johnson FR, Yang J-C. (2009). Environmental Risks and Behavior: Do children spend less time outdoors when ozone pollution is high? ASSA annual meeting, January 5, 2009. Update of Houtven et al. (2003), using the OAB CHAD data set, and related to Mansfield et al. (2006).

Mansfield C, Johnson FR, Van Houtven G. (2006). The missing piece: averting behavior for children's ozone exposures. *Resource Energy Econ.* 28:215-228.

McCurdy, T., Glen, G., Smith, L., Lakkadi, Y. (2000). The National Exposure Research Laboratory's Consolidated Human Activity Database. *J Expo Anal Environ Epidemiol*. 10: 566-578.

Persily, A. and Gorfain, J. (2004). Analysis of Ventilation Data from the U.S. Environmental Protection Agency Building Assessment Survey and Evaluation (BASE) Study. National Institute of Standards and Technology, NISTIR 7145, December 2004.

Persily, A., J. Gorfain, G. Brunner (2005). Ventilation Design and Performance in U.S. Office Buildings. *ASHRAE Journal.* April 2005, 30-35.

Robinson, J. P., Wiley, J. A., Piazza, T., Garrett, K., and Cirksena, K. (1989). Activity Patterns of California Residents and their Implications for Potential Exposure to Pollution. California Air Resources Board, Sacramento, CA. CARB-A6-177-33.

Shendell, D. G., A. M. Winer, R. Weker, S. D. Colome. (2004). Evidence of inadequate ventilation in portable classrooms: results of a pilot study in Los Angeles County. *Indoor Air* 2004; 14: 154–158.

Spier, C. E., Little, D. E., Trim, S. C., Johnson, T. R., Linn, W. S., Hackney, J. D. (1992). Activity patterns in elementary and high school students exposed to oxidant pollution. *J Expo Anal Environ Epidemiol*. 2: 277-293.

Tsang A. M., and Klepeis, N. E. (1996). Descriptive Statistics Tables from a Detailed Analysis of the National Human Activity Pattern Survey (NHAPS) Data. U.S. Environmental Protection Agency. EPA/600/R-96/148.

Turk, B. H., Grimsrud, D. T., Brown, J. T., Geisling-Sobotka, K. L., Harrison, J., Prill, R. J. (1989). Commercial Building Ventilation Rates and Particle Concentrations. ASHRAE No. 3248.

U.S. EPA (2002). Consolidated Human Activities Database (CHAD) Users Guide. Database and documentation available at: http://www.epa.gov/chadnet1/

U.S. EPA (2007). Ozone Population Exposure Analysis for Selected Urban Areas. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html

University of Michigan. (2012). The Panel Study of Income Dynamics (PSID). Data and documentation available at http://psidonline.isr.umich.edu/.

Wiley, J. A., Robinson, J. P., Piazza, T., Garrett, K., Cirksena, K., Cheng, Y.-T., Martin, G. (1991a). Activity Patterns of California Residents: Final Report. California Air Resources Board, Sacramento, CA. ARB/R93/487. Available from: NTIS, Springfield, VA., PB94-108719.

Wiley, J. A., Robinson, J. P., Cheng, Y.-T., Piazza, T., Stork, L., Pladsen, K. (1991b). Study of Children's Activity Patterns: Final Report. California Air Resources Board, Sacramento, CA. ARB-R-93/489.

Williams R, Suggs J, Rea A, Leovic K, et al. (2003a). The Research Triangle particulate panel study: PM mass concentrations relationships. *Atmos Environ*. 37:5349-5363.

Williams R, Suggs J, Rea A, Sheldon L, et al. (2003b). The Research Triangle particulate panel study: modeling ambient source contributions to personal and residential PM mass concentrations. *Atmos Environ*. 37:5365-5378.

Williams, R., Suggs, J., Creason, J., Rodes, C., Lawless, P., Kwok, R., Zweidinger, R., Sheldon, L. (2000). The 1998 Baltimore particulate matter epidemiology-exposure study: Part 2. Personal exposure associated with an elderly population. *J Expo Anal Environ Epidemiol*. 10(6): 533-543.

Xue, J., McCurdy, T., Spengler, J., Özkaynak, H. (2004). Understanding variability in time spent in selected locations for 7-12-year old children. *J Expo Anal Environ Epidemiol*. 14(3): 222-33.

APPENDIX 5C: GENERATION OF ADULT AND CHILD CENSUS-TRACT LEVEL ASTHMA PREVALENCE USING NHIS (2006-2010) AND US CENSUS (2000) DATA

5C-1. OVERVIEW

This describes the generation of our census tract level children and adult asthma prevalence data developed from the 2006-2010 National Health Interview Survey (NHIS) and census tract level poverty information from the 2000 US Census. The approach is, for the most part, a reapplication of work performed by Cohen and Rosenbaum (2005), though here we incorporated a few modifications as described below. Details regarding the earlier asthma prevalence work are documented in Appendix G of US EPA (2007).

Briefly in the earlier development work, Cohen and Rosenbaum (2005) calculated asthma prevalence for children aged 0 to 17 years for each age, gender, and four US regions using 2003 NHIS survey data. The four regions defined by NHIS were 'Midwest', 'Northeast', 'South', and 'West'. The asthma prevalence was defined as the probability of a 'Yes' response to the question "EVER been told that [the child] had asthma?"[1] among those persons that responded either 'Yes' or 'No' to this question.[2] The responses were weighted to take into account the complex survey design of the NHIS.[3] Standard errors and confidence intervals for the prevalence were calculated using a logistic model (PROC SURVEY LOGISTIC; SAS, 2012). A scatter-plot technique (LOESS SMOOTHER; SAS, 2012) was applied to smooth the prevalence curves and compute the standard errors and confidence intervals for the smoothed prevalence estimates. Logistic analysis of the raw and smoothed prevalence curves showed statistically significant differences in prevalence by gender and region, supporting their use as stratification variables in the final data set. These smoothed prevalence estimates were used as an input to EPA's Air Pollution Exposure Model (APEX) to estimate air pollutant exposure in asthmatic children (US EPA, 2007; 2008; 2009).

For the current asthma prevalence data set development, several years of recent NHIS survey data (2006-2010) were combined and used to calculate asthma prevalence. The current approach estimates asthma prevalence for children (by age in years) as was done previously by Cohen and Rosenbaum (2005) but now includes an estimate of adult asthma prevalence (by age groups). In addition, two sets of asthma prevalence for each adults and children were estimated

[1] The response was recorded as variable "CASHMEV" in the downloaded dataset. Data and documentation are available at http://www.cdc.gov/nchs/nhis/quest_data_related_1997_forward.htm.

[2] If there were another response to this variable other than "yes" or "no" (i.e., refused, not ascertained, don't know, and missing), the surveyed individual was excluded from the analysis data set.

[3] In the SURVEY LOGISTIC procedure, the variable "WTF_SC" was used for weighting, "PSU" was used for clustering, and "STRATUM" was used to define the stratum.

here. The first data set, as was done previously, was based on responses to the question "EVER been told that [the child] had asthma". The second data set was developed using the probability of a 'Yes' response to a question that followed those that answered 'Yes' to the first question regarding ever having asthma, specifically, do those persons "STILL have asthma?"[4] And finally, in addition to the nominal variables region and gender (and age and age groups), the asthma prevalence in this new analysis were further stratified by a family income/poverty ratio (i.e., whether the family income was considered below or at/above the US Census estimate of poverty level for the given year).

These new asthma prevalence data sets were linked to the US census tract level poverty ratios probabilities (US Census, 2007), also stratified by age and age groups. Given 1) the significant differences in asthma prevalence by age, gender, region, and poverty status, 2) the variability in the spatial distribution of poverty status across census tracts, stratified by age, and 3) the spatial variability in local scale ambient concentrations of many air pollutants, it is hoped that the variability in population exposures is now better represented when accounting for and modeling these newly refined attributes of this susceptible population.

5C-2. RAW ASTHMA PREVALENCE DATA SET DESCRIPTION

In this section we describe the asthma prevalence data sets used and identify the variables retained for our final data set. First, raw data and associated documentation were downloaded from the Center for Disease Control (CDC) and Prevention's National Health Interview Survey (NHIS) website.[5] The 'Sample Child' and 'Sample Adult' files were selected because of the availability of person-level attributes of interest within these files, i.e., age in years ('age_p'), gender ('sex'), US geographic region ('region'), coupled with the response to questions of whether or not the surveyed individual ever had and still has asthma. In total, five years of recent survey data were obtained, comprising over 50,000 children and 120,000 children for years 2006-2010 (Table 5C-1).

Information regarding personal and family income and poverty ranking are also provided by the NHIS in separate files. Five files ('INCIMPx.dat') are available for each survey year, each containing either the actual responses (where recorded or provided by survey participant) or imputed values for the desired financial variable.[6] For this current analysis, the ratio of income to poverty was used to develop a nominal variable: either the survey participant was below or

[4] While we estimated two separate sets of prevalence using the "STILL" and "EVER" variables, only the "STILL" data were used as input to our exposure model.

[5] See http://www.cdc.gov/nchs/nhis.htm (accessed October 4, 2011).

[6] Financial information was not collected from all persons; therefore the NHIS provides imputed data. Details into the available variables and imputation method are provided with each year's data set. For example see "Multiple Imputation of Family Income and Personal Earnings in the National Health Interview Survey: Methods and Examples" at http://www.cdc.gov/nchs/data/nhis/tecdoc_2010.pdf.

at/above a selected poverty threshold. This was done in this manner to be consistent with data generated as part of a companion data set, i.e., census tract level poverty ratio probabilities stratified by age (see section 5C-5 below).

Given the changes in how income data were collected over the five year period of interest and the presence of imputed data, a data processing methodology was needed to conform each of the year's data sets to a compatible nominal variable. Briefly, for survey years 2006-2008, poverty ratios ('RAT_CATI') are provided for each person as a categorical variable, ranging from <0.5 to 5.0 by increments of either 0.25 (for poverty ratios categories between <0.5 – 2.0) and 0.50 (for poverty ratios >5.0). For 2009 and 2010 data, the poverty ratio was provided as a continuous variable ('POVRATI3') rather than a categorical variable.[7]

When considering the number of stratification variables, the level of asthma prevalence, and poverty distribution among the survey population, sample size was an important issue. For the adult data, there were insufficient numbers of persons available to stratify the data by single ages (for some years of age there were no survey persons). Therefore, the adult survey data were grouped as follows: ages 18-24, 25-34, 35-44, 45-54, 55-64, 65-74, and, ≥75.[8] To increase the number of persons within the age, gender, and four region groupings of our characterization of 'below poverty' asthmatics persons, the poverty ratio threshold was selected as <1.5, therefore including persons that were within 50% above the poverty threshold. As there were five data sets containing variable imputed poverty ratios (as well as a non varying values for where income information was reported) for each year, the method for determining whether a person was below or above the poverty threshold was as follows. If three or more of the five imputed/recorded values were <1.5, the person's family income was categorized 'below' the poverty threshold, if three or more of the 5 values were ≥1.5, the person's family income was categorized 'above' the poverty threshold. The person-level income files were then merged with the sample adult and child files using the 'HHX' (a household identifier), 'FMX' (a family identifier), and 'FPX' (an individual identifier) variables. Note, all persons within the sample adult and child files had corresponding financial survey data.

Two asthma survey response variables were of interest in this analysis and were used to develop the two separate prevalence data sets for each children and adults. The response to the first question "Have you EVER been told by a doctor or other health professional that you [or

[7] Actually, the 2009 data had continuous values for the poverty ratios ('POVRATI2') but the quality was determined by us to be questionable: the value varied among family members by orders of magnitude – however, it should be a constant. The income data ('FAMINCI2') provided were constant among family members, therefore we combined these data with poverty thresholds obtained from the US Census (available at: http://www.census.gov/hhes/www/poverty/data/threshld/thresh08.html) for year 2008 by family size (note, income is the annual salary from the prior year) and calculated an appropriate poverty ratio for each family member.

[8] These same age groupings were used to create the companion file containing the census tract level poverty ratio probabilities (section 5C-5).

your child] had asthma?" was recorded as variable name 'CASHMEV' for children and 'AASMEV' for adults. Only persons having responses of either 'Yes' or 'No' to this question were retained to estimate the asthma prevalence. This assumes that the exclusion of those responding otherwise, i.e., those that 'refused' to answer, instances where it was "not ascertained', or the person 'does not know', does not affect the estimated prevalence rate if either 'Yes' or 'No' answers could actually be given by these persons. There were very few persons (<0.3%) that did provide an unusable response (Table 5C-1), thus the above assumption is reasonable. A second question was asked as a follow to persons responding "Yes" to the first question, specifically, "Do you STILL have asthma?" and noted as variables 'CASSTILL' and 'AASSTILL' for children and adults, respectively. Again, while only persons responding 'Yes' and 'No' were retained for further analysis, the representativeness of the screened data set is assumed unchanged from the raw survey data given the few persons having unusable data (<0.5%).

Table 5C-1. Number of total surveyed persons from NHIS (2006-2010) sample adult and child files and the number of those responding to asthma survey questions.

CHILDREN	**2010**	**2009**	**2008**	**2007**	**2006**	**TOTAL**
All Persons	11,277	11,156	8,815	9,417	9,837	50,502
Yes/No Asthma	11,256	11,142	8,800	9,404	9,815	50,417
Yes/No to Still Have + No Asthma	11,253	11,129	8,793	9,394	9,797	50,366
ADULTS	**2010**	**2009**	**2008**	**2007**	**2006**	**TOTAL**
All Persons	27,157	27,731	21,781	23,393	24,275	124,337
Yes/No Asthma	27,157	27,715	21,766	23,372	24,242	124,252
Yes/No to Still Have + No Asthma	27,113	27,686	21,726	23,349	24,208	124,082

5C-3. ASTHMA PREVALENCE: LOGISTIC MODELING

As described in the previous section, four person-level analytical data sets were created from the raw NHIS data files, generally containing similar variables: a 'Yes' or 'No' asthma response variable (either 'EVER' or 'STILL'), an age (or age group for adults), their gender ('male' or 'female'), US geographic region ('Midwest', 'Northeast', 'South', and 'West'), and poverty status ('below' or above'). One approach to calculate prevalence rates and their uncertainties for a given gender, region, poverty status, and age is to calculate the proportion of 'Yes' responses among the 'Yes' and 'No' responses for that demographic group, appropriately weighting each response by the survey weight. This simplified approach was initially used to develop 'raw' asthma prevalence rates however this approach may not be completely appropriate. The two main issues with such a simplified approach are that the distributions of the estimated prevalence rates would not be well approximated by normal distributions and that

the estimated confidence intervals based on a normal approximation would often extend outside the [0, 1] interval. A better approach for such survey data is to use a logistic transformation and fit the model:

Prob(asthma) = exp(beta) / (1 + exp(beta)),

where *beta* may depend on the explanatory variables for age, gender, poverty status, or region. This is equivalent to the model:

Beta = logit {prob(asthma) } = log { prob(asthma) / [1 – prob(asthma)] }.

The distribution of the estimated values of *beta* is more closely approximated by a normal distribution than the distribution of the corresponding estimates of prob(asthma). By applying a logit transformation to the confidence intervals for *beta*, the corresponding confidence intervals for prob(asthma) will always be inside [0, 1]. Another advantage of the logistic modeling is that it can be used to compare alternative statistical models, such as models where the prevalence probability depends upon age, region, poverty status, and gender, or on age, region, poverty status but not gender.

A variety of logistic models were fit and compared to use in estimating asthma prevalence, where the transformed probability variable beta is a given function of age, gender, poverty status, and region. I used the SAS procedure SURVEYLOGISTIC to fit the various logistic models, taking into account the NHIS survey weights and survey design (using both stratification and clustering options), as well as considering various combinations of the selected explanatory variables.

As an example, Table 5C-2 lists the models fit and their log-likelihood goodness-of-fit measures using the sample child data and for the "EVER" asthma response variable. A total of 32 models were fit, depending on the inclusion of selected explanatory variables and how age was considered in the model. The 'Strata' column lists the eight possible stratifications: no stratification, stratified by gender, by region, by poverty status, by region and gender, by region and poverty status, by gender and poverty status, and by region, gender and poverty status. For example, "5. region, gender" indicates that separate prevalence estimates were made for each combination of region and gender. As another example, "2. gender" means that separate prevalence estimates were made for each gender, so that for each gender, the prevalence is assumed to be the same for each region. Note the prevalence estimates are independently calculated for each stratum.

The 'Description' column of Table 5C-2 indicates how beta depends upon the age:

Linear in age	Beta = $\alpha + \beta \times$ age, where α and β vary with strata.
Quadratic in age	Beta = $\alpha + \beta \times \text{age} + \gamma \times \text{age}^2$, where α β and γ vary with strata.
Cubic in age	Beta = $\alpha + \beta \times \text{age} + \gamma \times \text{age}^2 + \delta \times \text{age}^3$, where α, β, γ, and δ vary with the strata.
f(age)	Beta = arbitrary function of age, with different functions for different strata

The category *f(age)* is equivalent to making age one of the stratification variables, and is also equivalent to making beta a polynomial of degree 16 in age (since the maximum age for children is 17), with coefficients that may vary with the strata.

The fitted models are listed in order of complexity, where the simplest model (1) is an unstratified linear model in age and the most complex model (model 32) has a prevalence that is an arbitrary function of age, gender, poverty status, and region. Model 32 is equivalent to calculating independent prevalence estimates for each of the 288 combinations of age, gender, poverty status, and region.

Table 5C-2. Example of alternative logistic models evaluated to estimate child asthma prevalence using the "EVER" asthma response variable and goodness of fit test results.

Model	Description	Strata	- 2 Log Likelihood	DF
1	1. logit(prob) = linear in age	1. none	288740115.1	2
2	1. logit(prob) = linear in age	2. gender	287062346.4	4
3	1. logit(prob) = linear in age	3. region	288120804.1	8
4	1. logit(prob) = linear in age	4. poverty	287385013.1	4
5	1. logit(prob) = linear in age	5. region, gender	286367652.6	16
6	1. logit(prob) = linear in age	6. region, poverty	286283543.6	16
7	1. logit(prob) = linear in age	7. gender, poverty	285696164.7	8
8	1. logit(prob) = linear in age	8. region, gender, poverty	284477928.1	32
9	2. logit(prob) = quadratic in age	1. none	286862135.1	3
10	2. logit(prob) = quadratic in age	2. gender	285098650.6	6
11	2. logit(prob) = quadratic in age	3. region	286207721.5	12
12	2. logit(prob) = quadratic in age	4. poverty	285352164	6
13	2. logit(prob) = quadratic in age	5. region, gender	284330346.1	24
14	2. logit(prob) = quadratic in age	6. region, poverty	284182547.5	24
15	2. logit(prob) = quadratic in age	7. gender, poverty	283587631.7	12
16	2. logit(prob) = quadratic in age	8. region, gender, poverty	282241318.6	48
17	3. logit(prob) = cubic in age	1. none	286227019.6	4
18	3. logit(prob) = cubic in age	2. gender	284470413	8
19	3. logit(prob) = cubic in age	3. region	285546716.1	16
20	3. logit(prob) = cubic in age	4. poverty	284688169.9	8
21	3. logit(prob) = cubic in age	5. region, gender	283662673.5	32
22	3. logit(prob) = cubic in age	6. region, poverty	283404487.5	32
23	3. logit(prob) = cubic in age	7. gender, poverty	282890785.3	16
24	3. logit(prob) = cubic in age	8. region, gender, poverty	281407414.3	64
25	4. logit(prob) = f(age)	1. none	285821686.2	18
26	4. logit(prob) = f(age)	2. gender	283843266.2	36
27	4. logit(prob) = f(age)	3. region	284761522.8	72
28	4. logit(prob) = f(age)	4. poverty	284045849.2	36
29	4. logit(prob) = f(age)	5. region, gender	282099156.1	144
30	4. logit(prob) = f(age)	6. region, poverty	281929968.5	144
31	4. logit(prob) = f(age)	7. gender, poverty	281963915.7	72
32	4. logit(prob) = f(age)	8. region, gender, poverty	278655423.1	288

Table 5C-2 also includes the -2 Log Likelihood statistic, a goodness-of-fit measure, and the associated degrees of freedom (DF), which is the total number of estimated parameters. Any two models can be compared using their -2 Log Likelihood values: models having lower values are preferred. If the first model is a special case of the second model, then the approximate statistical significance of the first model is estimated by comparing the difference in the -2 Log Likelihood values with a chi-squared random variable having *r* degrees of freedom, where *r* is the difference in the DF (hence a likelihood ratio test). For all pairs of models from Table 5C-2, all the differences in the -2 Log Likelihood statistic are at least 600,000 and thus significant at p-values well below 1 percent. Based on its having the lowest -2 Log Likelihood value, the last model fit (model 32: retaining all explanatory variables and using *f(age)*) was preferred and used to estimate the asthma prevalence.[9]

The SURVEYLOGISTIC procedure produces estimates of the beta values and their 95% confidence intervals for each combination of age, region, poverty status, and gender. By applying the inverse logit transformation,

$$\text{Prob(asthma)} = \exp(\text{beta}) / (1 + \exp(\text{beta})),$$

one can convert the beta values and associated 95% confidence intervals into predictions and 95% confidence intervals for the prevalence. The standard error for the prevalence was estimated as

$$\text{Std Error}\{\text{Prob(asthma)}\} = \text{Std Error (beta)} \times \exp(-\text{beta}) / (1 + \exp(\text{beta}))^2,$$

which follows from the delta method (i.e., a first order Taylor series approximation). Estimated asthma prevalence using this approach and termed here as 'unsmoothed' are provided in Attachment A. Results for children are given in Attachment A Tables 1 ('EVER' had Asthma) and 2 ('STILL' have asthma) while adults are provided in Attachment A Tables 3 ('EVER' had Asthma) and 4 ('STILL' have asthma). Graphical representation is also provided in a series of plots within Attachment A Figures 1 – 4. The variables provided in the tabular presentation are:

- Region
- Gender

[9] Similar results were obtained when estimating prevalence using the 'STILL' have asthma variable as well as when investigating model fit using the adult data sets. Note that because age was a categorical variable in the adult data sets it could only be evaluated using f(age_group). See Attachment B Tables 1 - 4 for all model fit results.

- Age (in years) or Age_group (age categories)
- Poverty Status
- Prevalence = predicted prevalence
- SE = standard error of predicted prevalence
- LowerCI = lower bound of 95 % confidence interval for predicted prevalence
- UpperCI = upper bound of 95 % confidence interval for predicted prevalence

5C-4. ASTHMA PREVALENCE: APPLICATION OF LOESS SMOOTHER

The estimated prevalence curves shows that the prevalence is not necessarily a smooth function of age. The linear, quadratic, and cubic functions of age modeled by SURVEYLOGISTIC were identified as a potential method for smoothing the curves, but they did not provide the best fit to the data. One reason for this might be due to the attempt to fit a global regression curve to all the age groups, which means that the predictions for age *A* are affected by data for very different ages. A local regression approach that separately fits a regression curve to each age A and its neighboring ages was used, giving a regression weight of 1 to the age *A*, and lower weights to the neighboring ages using a tri-weight function:

$$\text{Weight} = \{1 - [\ |\text{age} - A| / q\ ]^3\}, \text{ where } |\text{age} - A| <= q.$$

The parameter *q* defines the number of points in the neighborhood of the age *A*. Instead of calling *q* the smoothing parameter, SAS defines the smoothing parameter as the proportion of points in each neighborhood. A quadratic function of age to each age neighborhood was fit separately for each gender and region combination. These local regression curves were fit to the beta values, the logits of the asthma prevalence estimates, and then converted them back to estimated prevalence rates by applying the inverse logit function exp(beta) / (1 + exp(beta)). In addition to the tri-weight variable, each beta value was assigned a weight of $1 / [\text{std error (beta)}]^2$, to account for their uncertainties.

In this application of LOESS, weights of $1 / [\text{std error (beta)}]^2$ were used such that $\sigma^2 = 1$. The LOESS procedure estimates σ^2 from the weighted sum of squares. Because it is assumed $\sigma^2 = 1$, the estimated standard errors are multiplied by 1 / estimated σ and adjusted the widths of the confidence intervals by the same factor.

One data issue was an overly influential point that needed to be adjusted to avoid imposing wild variation in the "smoothed" curves: for the West region, males, age 0, above poverty threshold, there were 249 children surveyed that all gave 'No' answers to the asthma question, leading to an estimated value of -14.203 for beta with a standard error of 0.09. In this case the raw probability of asthma equals zero, so the corresponding estimated beta would be

negative infinity, but SAS's software gives -14.203 instead. To reduce the excessive impact of this single data point, we replaced the estimated standard error by 4, which is approximately four times the maximum standard error for all other region, gender, poverty status, and age combinations.

There are several potential values that can be selected for the smoothing parameter; the optimum value was determined by evaluating three regression diagnostics: the residual standard error, normal probability plots, and studentized residuals. To generate these statistics, the LOESS procedure was applied to estimated smoothed curves for beta, the logit of the prevalence, as a function of age, separately for each region, gender, and poverty classification. For the children data sets, curves were fit using the choices of 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, and 1.0 for the smoothing parameter. This selected range of values was bounded using the following observations. With only 18 points (i.e., the number of ages), a smoothing parameter of 0.2 cannot be used because the weight function assigns zero weights to all ages except age *A*, and a quadratic model cannot be uniquely fit to a single value. A smoothing parameter of 0.3 also cannot be used because that choice assigns a neighborhood of 5 points only ($0.3 \times 18 = 5$, rounded down), of which the two outside ages have assigned weight zero, making the local quadratic model fit exactly at every point except for the end points (ages 0, 1, 16 and 17). Usually one uses a smoothing parameter below 1 so that not all the data are used for the local regression at a given *x* value. Note also that a smoothing parameter of 0 can be used to generate the unsmoothed prevalence. The selection of the smoothing parameter used for the adult curves would follow a similar logic, although the lower bound could effectively be extended only to 0.9 given the number of age groups. This limits the selection of smoothing parameter applied to the two adult data sets to a value of 0.9, though values of 0.8 – 1.0 were nevertheless compared for good measure.

The first regression diagnostic used was the residual standard error, which is the LOESS estimate of σ. As discussed above, the true value of σ equals 1, so the best choice of smoothing parameter should have residual standard errors as close to 1 as possible. Attachment B, Tables 5 – 8 contain the residual standard errors output from the LOESS procedure, considering region, gender, poverty status and each data set examined. For children 'EVER' having asthma and when considering the best 20 models (of the 112 possible) using this criterion (note also within 0.06 RSE units of 1), the best choice varies with gender, region, and poverty status between smoothing parameters of 0.6, 0.7, and 0.8 (Table 5C-3). Similar results were observed for the 'STILL' data set, though a value of 0.6 would be slightly preferred. Either adult data set could be smoothed using a value of 0.8 or 0.9 given the limited selection of smoothing values, though 0.9 appears a better value for the 'STILL' data set.

Table 5C-3. Top 20 model smoothing fits where residual standard error at or a value of 1.0.

Data Set	Asthma	Smoothing Parameter						
		0.4	**0.5**	**0.6**	**0.7**	**0.8**	**0.9**	**1.0**
Children	EVER	2	2	5	5	4	1	1
	STILL	2	3	4	2	3	3	3
Adults	EVER	n/a	n/a	n/a	n/a	6	6	8
	STILL	n/a	n/a	n/a	n/a	5	7	8

The second regression diagnostic was developed from an approximate studentized residual. The residual errors from the LOESS model were divided by standard error (beta) to make their variances approximately constant. These approximately studentized residuals should be approximately normally distributed with a mean of zero and a variance of $\sigma^2 = 1$. To test this assumption, normal probability plots of the residuals were created for each smoothing parameter, combining all the studentized residuals across genders, regions, poverty status, and ages. These normal probability plots are provided in Attachment B, Figures 1 – 4. The results for the children data indicate little distinction or affect by the selection of a particular smoothing parameter (e.g., see Figure 5C-1 below), although linearity in the plotted curve is best expressed with smoothing parameters at or above values of 0.6. When considering the adult data sets, again the appropriate value would be 0.9, as Attachment B Figures 3 and 4 support this conclusion.

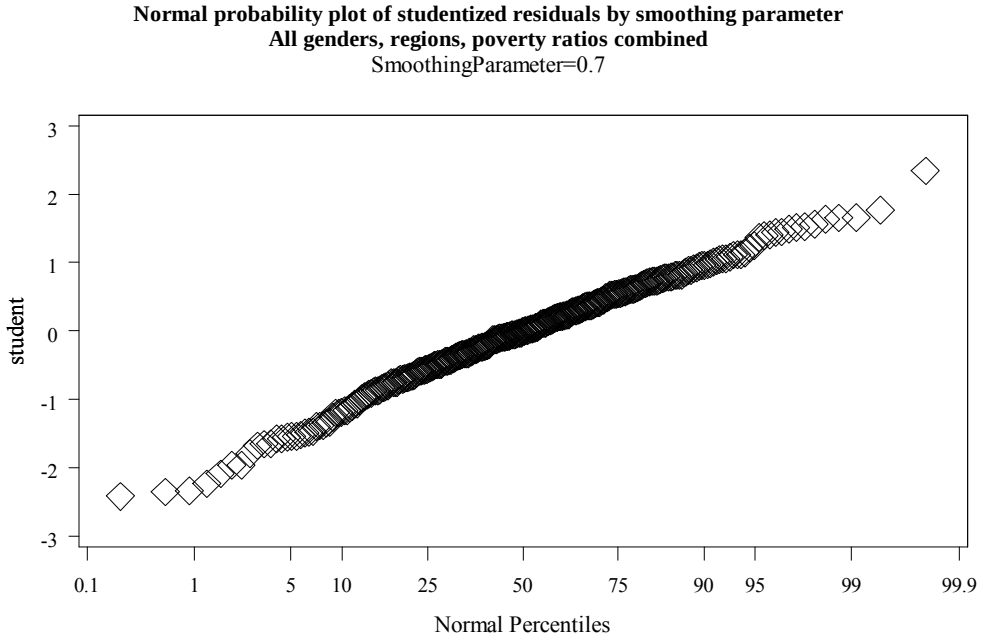


Figure 5C-1. Normal probability plot of studentized residuals generated using logistic model, smoothing set to 0.7, and the children 'EVER' asthmatic data set.

The third regression diagnostic, presented in Attachment B Figures 5 – 8 are plots of the studentized residuals against the smoothed beta values. All the studentized residuals for a given smoothing parameter are plotted together within the same graph. Also plotted is a LOESS smoothed curve fit to the same set of points, with SAS's optimal smoothing parameter choice, to indicate the typical pattern. Ideally there should be no obvious pattern and an average studentized residual close to zero with no regression slope (e.g., see Figure 5C-2). For the children data sets, these plots generally indicate no unusual patterns, and the results for smoothing parameters 0.4 through 0.6 indicate a fit LOESS curve closest to the studentized residual equals zero line. When considering the adult data sets, again the appropriate value would be 0.9, as Attachment B Figures7 and 8 support this conclusion.

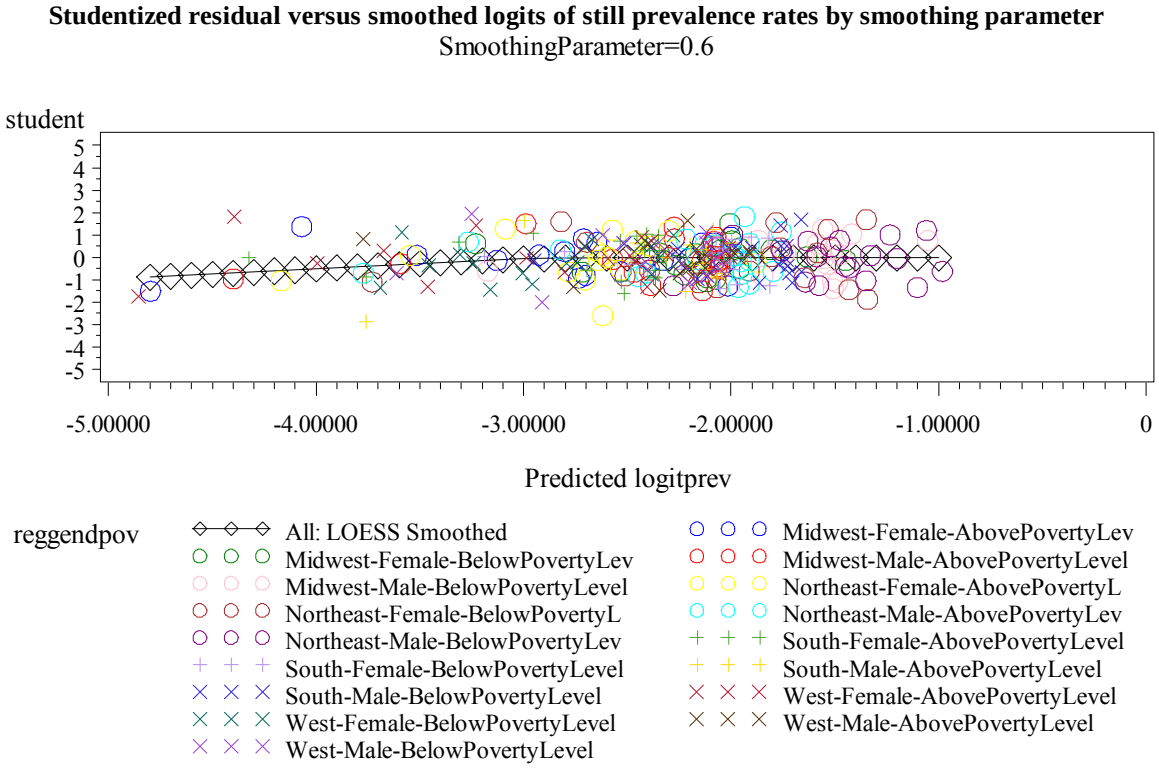


Figure 5C-2. Studentized residuals versus model predicted betas generated using a logistic model and using the children 'EVER' asthmatic data set, with smoothing set to 0.6.

When considering both children asthma prevalence responses evaluated, the residual standard error (estimated values for sigma) suggests the choice of smoothing parameter as 0.6 to 0.8. The normal probability plots of the studentized residuals suggest preference for smoothing at or above 0.6. The plots of residuals against smoothed predictions suggest the choices of 0.4

through 0.6. We therefore chose the final value of 0.6 to use for smoothing the children's asthma prevalence. For the adults, 0.9 was selected for smoothing.

Smoothed asthma prevalence and associated graphical presentation are provided in Attachment C, following a similar format as the unsmoothed data provided in Attachment A.

5C-5. CENSUS TRACT LEVEL POVERTY RATIO DATA SET DESCRIPTION AND PROCESSING

This section describes the approach used to generate census tract level poverty ratios for all US census tracts, stratified by age and age groups where available. The data set generation involved primarily two types of data downloaded from the 2000 US Census, each are described below.

First, individual state level SF3 geographic data ("geo") .uf3 files and associated documentation were downloaded[10] and, following import by SAS (SAS, 2012), were screened for tract level information using the "sumlev" variable equal to '140'. For quality control purposes and ease of matching with the poverty level data, our geo data set retained the following variables: stusab, sumlev, logrecno, state, county, tract, name, latitude, and longitude.

Second, the individual state level SF3 files ("30";) were downloaded, retaining the number of persons across the variable "PCT50" for all state "logrecno". [11] The data provided by the PCT50 variable is stratified by age or age groups (ages <5, 5, 6-11, 12-14, 15, 16-17, 18-24, 25-34, 35-44, 45-54, 55-64, 65-74, and ≥75) and income/poverty ratios, given in increments of 0.25. We calculated two new variables for each state logrecno using the number of persons from the PCT50 stratifications; the fraction of those persons having poverty ratios < 1.5 and ≥ 1.5 by summing the appropriate PCT50 variable and dividing by the total number of persons in that age/age group. Finally the poverty ratio data were combined with the above described census tract level geographic data using the "stusab" and "logrecno" variables. The final output was a single file containing relevant tract level poverty probabilities by age groups for all US census tracts (where available).

[10] Geographic data were obtained from http://www2.census.gov/census_2000/datasets/Summary_File_3/. Information regarding variable names is given in Figure 2-5 of US Census (2007).

[11] Poverty ratio data were obtained from http://www2.census.gov/census_2000/datasets/Summary_File_3/. Information regarding poverty ratio names variable names is given in chapter 6 of US Census Bureau (2007). We used the variable "PCT50", an income to poverty ratio variable stratified by various ages and age groups and described in chapter 7 of US Census Bureau (2007).

5C-6. COMBINED CENSUS TRACT LEVEL POVERTY RATIO AND ASTHMA PREVALENCE DATA

Because the prevalence data are stratified by standard US Census defined regions,[12] we first mapped the tract level poverty level data to an appropriate region based on the State. Further, as APEX requires the input data files to be complete, additional processing of the poverty probability file was needed. For where there was missing tract level poverty information[13], we substituted an age-specific value using the average for the particular county the tract was located within. The frequency of missing data substitution comprised 1.7% of the total poverty probability data set. The two data sets were merged and the final asthma prevalence was calculated using the following weighting scheme:

```
prevalence=round((pov_prob*prev_poor)+((1-pov_prob)*prev_notpoor),0.0001);
```

whereas each US census tract value now expresses a tract specific poverty-weighted prevalence, stratified by ages (children 0-17), age groups (adults), and two genders. These final prevalence data are found within the APEX *asthmaprevalence.txt* file.

5C-7. REFERENCES

Cohen J and Rosenbaum A. (2005). Analysis of NHIS Asthma Prevalence Data. Memorandum to John Langstaff by ICF Incorporated. For US EPA Work Assignment 3-08 under EPA contract 68D01052.

SAS. (2012). SAS/STAT 9.2 User's Guide, Second Edition. Available at: http://support.sas.com/documentation/cdl/en/statug/63033/PDF/default/statug.pdf.

US Census Bureau. (2007). 2000 Census of Population and Housing. Summary File 3 (SF3) Technical Documentation, available at: http://www.census.gov/prod/cen2000/doc/sf3.pdf. Individual SF3 files '30' (for income/poverty variables pct50) for each state were downloaded from: http://www2.census.gov/census_2000/datasets/Summary_File_3/.

US EPA. (2007). Ozone Population Exposure Analysis for Selected Urban Areas (July 2007). Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-010. Available at: http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.

US EPA. (2008). Risk and Exposure Assessment to Support the Review of the NO_2 Primary National Ambient Air Quality Standard. Report no. EPA-452/R-08-008a. November 2008. Available at: http://www.epa.gov/ttn/naaqs/standards/nox/data/20081121_NO2_REA_final.pdf.

US EPA. (2009). Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standard. Report no. EPA-452/R-09-007. August 2009. Available at: http://www.epa.gov/ttn/naaqs/standards/so2/data/200908SO2REAFinalReport.pdf.

[12] For example, see http://www.cdc.gov/std/stats10/census.htm.

[13] Whether there were no data collected by the Census or whether there were simply no persons in that age group is relatively inconsequential to estimating the asthmatic persons exposed, particularly considering latter case as no persons in that age group would be modeled.

APPENDIX 5C, ATTACHMENT A: UNSMOOTHED ASTHMA PREVALENCE TABLES AND FIGURES.

Appendix 5C, Attachment A, Table-1. Unsmoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	Midwest	Female	Above Poverty	0	0.0018	0.0018	0.0002	0.0129
No	Midwest	Female	Above Poverty	1	0.0387	0.0233	0.0117	0.1208
No	Midwest	Female	Above Poverty	2	0.0367	0.0148	0.0165	0.0797
No	Midwest	Female	Above Poverty	3	0.0395	0.0186	0.0155	0.0972
No	Midwest	Female	Above Poverty	4	0.0815	0.0298	0.0390	0.1624
No	Midwest	Female	Above Poverty	5	0.0885	0.0207	0.0556	0.1382
No	Midwest	Female	Above Poverty	6	0.0438	0.0200	0.0176	0.1046
No	Midwest	Female	Above Poverty	7	0.1374	0.0277	0.0916	0.2010
No	Midwest	Female	Above Poverty	8	0.0820	0.0246	0.0450	0.1450
No	Midwest	Female	Above Poverty	9	0.1027	0.0220	0.0669	0.1545
No	Midwest	Female	Above Poverty	10	0.0995	0.0193	0.0675	0.1442
No	Midwest	Female	Above Poverty	11	0.1129	0.0277	0.0688	0.1797
No	Midwest	Female	Above Poverty	12	0.1752	0.0391	0.1112	0.2652
No	Midwest	Female	Above Poverty	13	0.1331	0.0256	0.0905	0.1916
No	Midwest	Female	Above Poverty	14	0.1944	0.0477	0.1173	0.3049
No	Midwest	Female	Above Poverty	15	0.1383	0.0302	0.0890	0.2086
No	Midwest	Female	Above Poverty	16	0.1731	0.0341	0.1160	0.2502
No	Midwest	Female	Above Poverty	17	0.1311	0.0256	0.0885	0.1898
No	Midwest	Female	Below Poverty	0	0.0564	0.0353	0.0160	0.1799
No	Midwest	Female	Below Poverty	1	0.0585	0.0197	0.0299	0.1112
No	Midwest	Female	Below Poverty	2	0.1256	0.0487	0.0567	0.2552
No	Midwest	Female	Below Poverty	3	0.1127	0.0419	0.0529	0.2240
No	Midwest	Female	Below Poverty	4	0.1746	0.0395	0.1100	0.2658
No	Midwest	Female	Below Poverty	5	0.1584	0.0447	0.0888	0.2664
No	Midwest	Female	Below Poverty	6	0.1229	0.0417	0.0616	0.2301
No	Midwest	Female	Below Poverty	7	0.0867	0.0353	0.0381	0.1851
No	Midwest	Female	Below Poverty	8	0.1523	0.0392	0.0902	0.2456
No	Midwest	Female	Below Poverty	9	0.2070	0.0486	0.1275	0.3182
No	Midwest	Female	Below Poverty	10	0.2293	0.1109	0.0800	0.5043
No	Midwest	Female	Below Poverty	11	0.1359	0.0470	0.0670	0.2562
No	Midwest	Female	Below Poverty	12	0.1501	0.0484	0.0774	0.2710
No	Midwest	Female	Below Poverty	13	0.1527	0.0380	0.0921	0.2427
No	Midwest	Female	Below Poverty	14	0.1197	0.0462	0.0544	0.2431
No	Midwest	Female	Below Poverty	15	0.2103	0.0760	0.0980	0.3949
No	Midwest	Female	Below Poverty	16	0.2054	0.0597	0.1121	0.3462
No	Midwest	Female	Below Poverty	17	0.1844	0.1134	0.0491	0.4976
No	Midwest	Male	Above Poverty	0	0.0061	0.0044	0.0015	0.0247
No	Midwest	Male	Above Poverty	1	0.0258	0.0178	0.0066	0.0957
No	Midwest	Male	Above Poverty	2	0.0848	0.0231	0.0491	0.1426
No	Midwest	Male	Above Poverty	3	0.0996	0.0261	0.0588	0.1636
No	Midwest	Male	Above Poverty	4	0.0876	0.0223	0.0527	0.1423
No	Midwest	Male	Above Poverty	5	0.1593	0.0313	0.1069	0.2306
No	Midwest	Male	Above Poverty	6	0.0977	0.0229	0.0611	0.1527
No	Midwest	Male	Above Poverty	7	0.1793	0.0313	0.1259	0.2489
No	Midwest	Male	Above Poverty	8	0.1503	0.0356	0.0930	0.2340
No	Midwest	Male	Above Poverty	9	0.1418	0.0265	0.0973	0.2021
No	Midwest	Male	Above Poverty	10	0.1569	0.0322	0.1035	0.2306
No	Midwest	Male	Above Poverty	11	0.1717	0.0371	0.1106	0.2568
No	Midwest	Male	Above Poverty	12	0.2054	0.0338	0.1470	0.2795
No	Midwest	Male	Above Poverty	13	0.1846	0.0358	0.1244	0.2650
No	Midwest	Male	Above Poverty	14	0.1671	0.0291	0.1175	0.2322
No	Midwest	Male	Above Poverty	15	0.1454	0.0356	0.0885	0.2297
No	Midwest	Male	Above Poverty	16	0.1557	0.0278	0.1087	0.2182
No	Midwest	Male	Above Poverty	17	0.1320	0.0233	0.0926	0.1848
No	Midwest	Male	Below Poverty	0	0.0293	0.0176	0.0089	0.0922
No	Midwest	Male	Below Poverty	1	0.1051	0.0376	0.0509	0.2047
No	Midwest	Male	Below Poverty	2	0.1786	0.0652	0.0835	0.3418
No	Midwest	Male	Below Poverty	3	0.2066	0.0513	0.1236	0.3247

Appendix 5C, Attachment A, Table-1. Unsmoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	Midwest	Male	Below Poverty	4	0.2770	0.0638	0.1703	0.4170
No	Midwest	Male	Below Poverty	5	0.2504	0.0499	0.1656	0.3600
No	Midwest	Male	Below Poverty	6	0.2186	0.0447	0.1436	0.3184
No	Midwest	Male	Below Poverty	7	0.2192	0.0456	0.1428	0.3211
No	Midwest	Male	Below Poverty	8	0.2902	0.0649	0.1806	0.4312
No	Midwest	Male	Below Poverty	9	0.1242	0.0437	0.0607	0.2374
No	Midwest	Male	Below Poverty	10	0.2897	0.0639	0.1815	0.4285
No	Midwest	Male	Below Poverty	11	0.2669	0.0613	0.1646	0.4021
No	Midwest	Male	Below Poverty	12	0.2589	0.1050	0.1068	0.5051
No	Midwest	Male	Below Poverty	13	0.2429	0.0693	0.1329	0.4017
No	Midwest	Male	Below Poverty	14	0.1470	0.0490	0.0742	0.2703
No	Midwest	Male	Below Poverty	15	0.1965	0.0509	0.1150	0.3151
No	Midwest	Male	Below Poverty	16	0.1855	0.0611	0.0935	0.3345
No	Midwest	Male	Below Poverty	17	0.3740	0.1042	0.1998	0.5884
No	Northeast	Female	Above Poverty	0	0.0055	0.0054	0.0008	0.0368
No	Northeast	Female	Above Poverty	1	0.0296	0.0164	0.0099	0.0854
No	Northeast	Female	Above Poverty	2	0.0697	0.0252	0.0337	0.1384
No	Northeast	Female	Above Poverty	3	0.0723	0.0250	0.0362	0.1394
No	Northeast	Female	Above Poverty	4	0.1142	0.0254	0.0731	0.1741
No	Northeast	Female	Above Poverty	5	0.1058	0.0296	0.0602	0.1793
No	Northeast	Female	Above Poverty	6	0.0933	0.0254	0.0541	0.1563
No	Northeast	Female	Above Poverty	7	0.1084	0.0251	0.0681	0.1682
No	Northeast	Female	Above Poverty	8	0.0780	0.0221	0.0442	0.1339
No	Northeast	Female	Above Poverty	9	0.1362	0.0374	0.0780	0.2272
No	Northeast	Female	Above Poverty	10	0.0979	0.0298	0.0530	0.1738
No	Northeast	Female	Above Poverty	11	0.1697	0.0382	0.1073	0.2578
No	Northeast	Female	Above Poverty	12	0.0535	0.0229	0.0228	0.1204
No	Northeast	Female	Above Poverty	13	0.0910	0.0273	0.0499	0.1604
No	Northeast	Female	Above Poverty	14	0.1500	0.0207	0.1138	0.1953
No	Northeast	Female	Above Poverty	15	0.1733	0.0355	0.1142	0.2541
No	Northeast	Female	Above Poverty	16	0.1884	0.0510	0.1077	0.3085
No	Northeast	Female	Above Poverty	17	0.1694	0.0395	0.1052	0.2613
No	Northeast	Female	Below Poverty	0	0.0315	0.0251	0.0064	0.1404
No	Northeast	Female	Below Poverty	1	0.1230	0.0576	0.0469	0.2852
No	Northeast	Female	Below Poverty	2	0.0703	0.0277	0.0319	0.1479
No	Northeast	Female	Below Poverty	3	0.1860	0.0555	0.1002	0.3193
No	Northeast	Female	Below Poverty	4	0.1666	0.0598	0.0791	0.3175
No	Northeast	Female	Below Poverty	5	0.2347	0.0636	0.1329	0.3802
No	Northeast	Female	Below Poverty	6	0.0682	0.0250	0.0327	0.1366
No	Northeast	Female	Below Poverty	7	0.0972	0.0362	0.0458	0.1944
No	Northeast	Female	Below Poverty	8	0.2049	0.0604	0.1107	0.3478
No	Northeast	Female	Below Poverty	9	0.1695	0.0698	0.0717	0.3505
No	Northeast	Female	Below Poverty	10	0.0988	0.0440	0.0400	0.2240
No	Northeast	Female	Below Poverty	11	0.2622	0.0734	0.1445	0.4277
No	Northeast	Female	Below Poverty	12	0.1377	0.0525	0.0629	0.2752
No	Northeast	Female	Below Poverty	13	0.3506	0.0762	0.2188	0.5100
No	Northeast	Female	Below Poverty	14	0.1869	0.0537	0.1031	0.3148
No	Northeast	Female	Below Poverty	15	0.1965	0.0534	0.1120	0.3217
No	Northeast	Female	Below Poverty	16	0.1986	0.0470	0.1221	0.3065
No	Northeast	Female	Below Poverty	17	0.1625	0.0602	0.0754	0.3158
No	Northeast	Male	Above Poverty	0	0.0256	0.0130	0.0094	0.0679
No	Northeast	Male	Above Poverty	1	0.0542	0.0231	0.0231	0.1218
No	Northeast	Male	Above Poverty	2	0.0635	0.0220	0.0318	0.1228
No	Northeast	Male	Above Poverty	3	0.0835	0.0232	0.0478	0.1418
No	Northeast	Male	Above Poverty	4	0.1378	0.0329	0.0849	0.2158
No	Northeast	Male	Above Poverty	5	0.1444	0.0357	0.0875	0.2291
No	Northeast	Male	Above Poverty	6	0.2175	0.0482	0.1376	0.3263
No	Northeast	Male	Above Poverty	7	0.2019	0.0343	0.1429	0.2774
No	Northeast	Male	Above Poverty	8	0.1878	0.0373	0.1252	0.2719
No	Northeast	Male	Above Poverty	9	0.1286	0.0342	0.0751	0.2115
No	Northeast	Male	Above Poverty	10	0.1879	0.0278	0.1394	0.2485
No	Northeast	Male	Above Poverty	11	0.2532	0.0420	0.1799	0.3439
No	Northeast	Male	Above Poverty	12	0.1801	0.0233	0.1388	0.2303
No	Northeast	Male	Above Poverty	13	0.1581	0.0340	0.1022	0.2366

Appendix 5C, Attachment A, Table-1. Unsmoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	Northeast	Male	Above Poverty	14	0.2043	0.0447	0.1303	0.3056
No	Northeast	Male	Above Poverty	15	0.1752	0.0287	0.1257	0.2387
No	Northeast	Male	Above Poverty	16	0.1798	0.0360	0.1195	0.2614
No	Northeast	Male	Above Poverty	17	0.1836	0.0282	0.1346	0.2454
No	Northeast	Male	Below Poverty	0	0.0375	0.0275	0.0087	0.1477
No	Northeast	Male	Below Poverty	1	0.1649	0.0506	0.0877	0.2887
No	Northeast	Male	Below Poverty	2	0.2200	0.0503	0.1371	0.3337
No	Northeast	Male	Below Poverty	3	0.1124	0.0445	0.0501	0.2330
No	Northeast	Male	Below Poverty	4	0.2651	0.0909	0.1262	0.4738
No	Northeast	Male	Below Poverty	5	0.2398	0.0651	0.1355	0.3885
No	Northeast	Male	Below Poverty	6	0.3209	0.0432	0.2427	0.4107
No	Northeast	Male	Below Poverty	7	0.2651	0.0572	0.1686	0.3908
No	Northeast	Male	Below Poverty	8	0.2905	0.0969	0.1401	0.5070
No	Northeast	Male	Below Poverty	9	0.3810	0.0773	0.2446	0.5392
No	Northeast	Male	Below Poverty	10	0.3382	0.1019	0.1732	0.5551
No	Northeast	Male	Below Poverty	11	0.2485	0.0708	0.1359	0.4102
No	Northeast	Male	Below Poverty	12	0.2819	0.0705	0.1656	0.4371
No	Northeast	Male	Below Poverty	13	0.2961	0.0685	0.1808	0.4448
No	Northeast	Male	Below Poverty	14	0.2876	0.0713	0.1695	0.4440
No	Northeast	Male	Below Poverty	15	0.2632	0.0661	0.1548	0.4107
No	Northeast	Male	Below Poverty	16	0.2407	0.0559	0.1483	0.3660
No	Northeast	Male	Below Poverty	17	0.3123	0.0734	0.1885	0.4701
No	South	Female	Above Poverty	0	0.0129	0.0080	0.0038	0.0427
No	South	Female	Above Poverty	1	0.0191	0.0084	0.0080	0.0447
No	South	Female	Above Poverty	2	0.0558	0.0147	0.0330	0.0928
No	South	Female	Above Poverty	3	0.0793	0.0200	0.0479	0.1286
No	South	Female	Above Poverty	4	0.0834	0.0184	0.0537	0.1273
No	South	Female	Above Poverty	5	0.0932	0.0222	0.0579	0.1467
No	South	Female	Above Poverty	6	0.1446	0.0226	0.1057	0.1948
No	South	Female	Above Poverty	7	0.1439	0.0248	0.1017	0.1996
No	South	Female	Above Poverty	8	0.1111	0.0194	0.0784	0.1550
No	South	Female	Above Poverty	9	0.1258	0.0222	0.0883	0.1762
No	South	Female	Above Poverty	10	0.0626	0.0154	0.0383	0.1005
No	South	Female	Above Poverty	11	0.1288	0.0210	0.0928	0.1759
No	South	Female	Above Poverty	12	0.1064	0.0182	0.0756	0.1478
No	South	Female	Above Poverty	13	0.1387	0.0222	0.1006	0.1881
No	South	Female	Above Poverty	14	0.1621	0.0243	0.1198	0.2156
No	South	Female	Above Poverty	15	0.1399	0.0169	0.1100	0.1763
No	South	Female	Above Poverty	16	0.1362	0.0253	0.0938	0.1938
No	South	Female	Above Poverty	17	0.1299	0.0197	0.0959	0.1737
No	South	Female	Below Poverty	0	0.0495	0.0216	0.0207	0.1137
No	South	Female	Below Poverty	1	0.0734	0.0210	0.0415	0.1268
No	South	Female	Below Poverty	2	0.0828	0.0207	0.0503	0.1336
No	South	Female	Below Poverty	3	0.0973	0.0271	0.0556	0.1649
No	South	Female	Below Poverty	4	0.1578	0.0372	0.0976	0.2450
No	South	Female	Below Poverty	5	0.1409	0.0300	0.0917	0.2103
No	South	Female	Below Poverty	6	0.1536	0.0381	0.0927	0.2439
No	South	Female	Below Poverty	7	0.1658	0.0332	0.1104	0.2414
No	South	Female	Below Poverty	8	0.1428	0.0302	0.0931	0.2126
No	South	Female	Below Poverty	9	0.2123	0.0413	0.1425	0.3042
No	South	Female	Below Poverty	10	0.1408	0.0347	0.0855	0.2233
No	South	Female	Below Poverty	11	0.2249	0.0466	0.1467	0.3288
No	South	Female	Below Poverty	12	0.1741	0.0519	0.0941	0.2997
No	South	Female	Below Poverty	13	0.1463	0.0296	0.0972	0.2142
No	South	Female	Below Poverty	14	0.2428	0.0437	0.1675	0.3382
No	South	Female	Below Poverty	15	0.1947	0.0399	0.1280	0.2847
No	South	Female	Below Poverty	16	0.1285	0.0344	0.0747	0.2122
No	South	Female	Below Poverty	17	0.1322	0.0323	0.0807	0.2092
No	South	Male	Above Poverty	0	0.0135	0.0065	0.0052	0.0342
No	South	Male	Above Poverty	1	0.0782	0.0162	0.0517	0.1165
No	South	Male	Above Poverty	2	0.1134	0.0190	0.0811	0.1563
No	South	Male	Above Poverty	3	0.1063	0.0211	0.0714	0.1554
No	South	Male	Above Poverty	4	0.1679	0.0303	0.1165	0.2360
No	South	Male	Above Poverty	5	0.1644	0.0226	0.1247	0.2136

Appendix 5C, Attachment A, Table-1. Unsmoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	South	Male	Above Poverty	6	0.1328	0.0212	0.0964	0.1802
No	South	Male	Above Poverty	7	0.1542	0.0270	0.1083	0.2148
No	South	Male	Above Poverty	8	0.1502	0.0224	0.1114	0.1994
No	South	Male	Above Poverty	9	0.1522	0.0232	0.1121	0.2033
No	South	Male	Above Poverty	10	0.1485	0.0240	0.1073	0.2018
No	South	Male	Above Poverty	11	0.1767	0.0255	0.1322	0.2323
No	South	Male	Above Poverty	12	0.1915	0.0236	0.1495	0.2419
No	South	Male	Above Poverty	13	0.1939	0.0255	0.1487	0.2487
No	South	Male	Above Poverty	14	0.1381	0.0196	0.1039	0.1813
No	South	Male	Above Poverty	15	0.1579	0.0246	0.1154	0.2122
No	South	Male	Above Poverty	16	0.1698	0.0193	0.1352	0.2110
No	South	Male	Above Poverty	17	0.1530	0.0240	0.1117	0.2061
No	South	Male	Below Poverty	0	0.0610	0.0181	0.0338	0.1076
No	South	Male	Below Poverty	1	0.1005	0.0206	0.0667	0.1488
No	South	Male	Below Poverty	2	0.1102	0.0225	0.0732	0.1626
No	South	Male	Below Poverty	3	0.1699	0.0324	0.1154	0.2431
No	South	Male	Below Poverty	4	0.1642	0.0288	0.1152	0.2285
No	South	Male	Below Poverty	5	0.2510	0.0485	0.1682	0.3572
No	South	Male	Below Poverty	6	0.2064	0.0339	0.1477	0.2808
No	South	Male	Below Poverty	7	0.1588	0.0309	0.1072	0.2290
No	South	Male	Below Poverty	8	0.2518	0.0503	0.1663	0.3622
No	South	Male	Below Poverty	9	0.2246	0.0381	0.1588	0.3078
No	South	Male	Below Poverty	10	0.2022	0.0368	0.1394	0.2839
No	South	Male	Below Poverty	11	0.1890	0.0344	0.1305	0.2658
No	South	Male	Below Poverty	12	0.2322	0.0383	0.1656	0.3153
No	South	Male	Below Poverty	13	0.2345	0.0454	0.1573	0.3345
No	South	Male	Below Poverty	14	0.2265	0.0489	0.1448	0.3361
No	South	Male	Below Poverty	15	0.1801	0.0371	0.1183	0.2645
No	South	Male	Below Poverty	16	0.1286	0.0303	0.0799	0.2005
No	South	Male	Below Poverty	17	0.1916	0.0297	0.1399	0.2566
No	West	Female	Above Poverty	0	0.0049	0.0037	0.0011	0.0216
No	West	Female	Above Poverty	1	0.0390	0.0202	0.0139	0.1048
No	West	Female	Above Poverty	2	0.0269	0.0097	0.0132	0.0541
No	West	Female	Above Poverty	3	0.0439	0.0153	0.0219	0.0858
No	West	Female	Above Poverty	4	0.0232	0.0079	0.0118	0.0450
No	West	Female	Above Poverty	5	0.0988	0.0294	0.0544	0.1730
No	West	Female	Above Poverty	6	0.0829	0.0223	0.0484	0.1384
No	West	Female	Above Poverty	7	0.1065	0.0281	0.0627	0.1752
No	West	Female	Above Poverty	8	0.0960	0.0280	0.0534	0.1666
No	West	Female	Above Poverty	9	0.1124	0.0296	0.0662	0.1846
No	West	Female	Above Poverty	10	0.0978	0.0285	0.0545	0.1695
No	West	Female	Above Poverty	11	0.1186	0.0188	0.0864	0.1606
No	West	Female	Above Poverty	12	0.1655	0.0352	0.1074	0.2463
No	West	Female	Above Poverty	13	0.0855	0.0196	0.0542	0.1324
No	West	Female	Above Poverty	14	0.1258	0.0278	0.0806	0.1911
No	West	Female	Above Poverty	15	0.1482	0.0213	0.1111	0.1949
No	West	Female	Above Poverty	16	0.1394	0.0254	0.0967	0.1969
No	West	Female	Above Poverty	17	0.2285	0.0375	0.1632	0.3101
No	West	Female	Below Poverty	0	0.0064	0.0064	0.0009	0.0441
No	West	Female	Below Poverty	1	0.0443	0.0195	0.0185	0.1025
No	West	Female	Below Poverty	2	0.0523	0.0220	0.0226	0.1166
No	West	Female	Below Poverty	3	0.0403	0.0140	0.0202	0.0788
No	West	Female	Below Poverty	4	0.0346	0.0177	0.0126	0.0919
No	West	Female	Below Poverty	5	0.0887	0.0372	0.0380	0.1934
No	West	Female	Below Poverty	6	0.1351	0.0432	0.0703	0.2439
No	West	Female	Below Poverty	7	0.1364	0.0360	0.0798	0.2234
No	West	Female	Below Poverty	8	0.1106	0.0244	0.0711	0.1682
No	West	Female	Below Poverty	9	0.1254	0.0405	0.0650	0.2283
No	West	Female	Below Poverty	10	0.0585	0.0204	0.0292	0.1137
No	West	Female	Below Poverty	11	0.0747	0.0264	0.0368	0.1460
No	West	Female	Below Poverty	12	0.0720	0.0279	0.0331	0.1496
No	West	Female	Below Poverty	13	0.1898	0.0591	0.0993	0.3323
No	West	Female	Below Poverty	14	0.1431	0.0431	0.0773	0.2495
No	West	Female	Below Poverty	15	0.1168	0.0304	0.0692	0.1906

Appendix 5C, Attachment A, Table-1. Unsmoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	West	Female	Below Poverty	16	0.0814	0.0290	0.0398	0.1593
No	West	Female	Below Poverty	17	0.0637	0.0235	0.0305	0.1285
No	West	Male	Above Poverty	0	0.0000	0.0000	0.0000	0.0000
No	West	Male	Above Poverty	1	0.0244	0.0121	0.0092	0.0635
No	West	Male	Above Poverty	2	0.0517	0.0155	0.0285	0.0920
No	West	Male	Above Poverty	3	0.0601	0.0172	0.0339	0.1041
No	West	Male	Above Poverty	4	0.1698	0.0275	0.1224	0.2307
No	West	Male	Above Poverty	5	0.1236	0.0288	0.0772	0.1918
No	West	Male	Above Poverty	6	0.1376	0.0264	0.0934	0.1980
No	West	Male	Above Poverty	7	0.1288	0.0354	0.0738	0.2152
No	West	Male	Above Poverty	8	0.1018	0.0223	0.0657	0.1547
No	West	Male	Above Poverty	9	0.1884	0.0315	0.1342	0.2579
No	West	Male	Above Poverty	10	0.1604	0.0273	0.1138	0.2215
No	West	Male	Above Poverty	11	0.2121	0.0298	0.1596	0.2762
No	West	Male	Above Poverty	12	0.1833	0.0349	0.1244	0.2618
No	West	Male	Above Poverty	13	0.2105	0.0397	0.1431	0.2987
No	West	Male	Above Poverty	14	0.1475	0.0309	0.0966	0.2187
No	West	Male	Above Poverty	15	0.1641	0.0263	0.1188	0.2224
No	West	Male	Above Poverty	16	0.1958	0.0282	0.1463	0.2569
No	West	Male	Above Poverty	17	0.2113	0.0289	0.1602	0.2733
No	West	Male	Below Poverty	0	0.0135	0.0128	0.0020	0.0832
No	West	Male	Below Poverty	1	0.0812	0.0317	0.0370	0.1691
No	West	Male	Below Poverty	2	0.0417	0.0131	0.0224	0.0765
No	West	Male	Below Poverty	3	0.1182	0.0351	0.0647	0.2061
No	West	Male	Below Poverty	4	0.1349	0.0329	0.0823	0.2131
No	West	Male	Below Poverty	5	0.1562	0.0401	0.0926	0.2514
No	West	Male	Below Poverty	6	0.1853	0.0444	0.1133	0.2883
No	West	Male	Below Poverty	7	0.1484	0.0343	0.0928	0.2288
No	West	Male	Below Poverty	8	0.1549	0.0343	0.0988	0.2346
No	West	Male	Below Poverty	9	0.1275	0.0418	0.0654	0.2338
No	West	Male	Below Poverty	10	0.1742	0.0431	0.1049	0.2751
No	West	Male	Below Poverty	11	0.1909	0.0554	0.1046	0.3227
No	West	Male	Below Poverty	12	0.1678	0.0599	0.0800	0.3185
No	West	Male	Below Poverty	13	0.1793	0.0491	0.1021	0.2959
No	West	Male	Below Poverty	14	0.1919	0.0454	0.1180	0.2966
No	West	Male	Below Poverty	15	0.1410	0.0577	0.0606	0.2946
No	West	Male	Below Poverty	16	0.1863	0.0384	0.1223	0.2734
No	West	Male	Below Poverty	17	0.2030	0.0493	0.1229	0.3165

Appendix 5C, Attachment A, Table 2. Unsmoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	Midwest	Female	Above Poverty	0	0.0018	0.0018	0.0002	0.0129
No	Midwest	Female	Above Poverty	1	0.0387	0.0233	0.0117	0.1208
No	Midwest	Female	Above Poverty	2	0.0302	0.0135	0.0125	0.0715
No	Midwest	Female	Above Poverty	3	0.0395	0.0186	0.0155	0.0972
No	Midwest	Female	Above Poverty	4	0.0531	0.0214	0.0238	0.1142
No	Midwest	Female	Above Poverty	5	0.0617	0.0173	0.0354	0.1055
No	Midwest	Female	Above Poverty	6	0.0386	0.0192	0.0143	0.0999
No	Midwest	Female	Above Poverty	7	0.0801	0.0239	0.0442	0.1411
No	Midwest	Female	Above Poverty	8	0.0492	0.0151	0.0267	0.0888
No	Midwest	Female	Above Poverty	9	0.0789	0.0200	0.0476	0.1280
No	Midwest	Female	Above Poverty	10	0.0625	0.0162	0.0373	0.1029
No	Midwest	Female	Above Poverty	11	0.0856	0.0232	0.0498	0.1433
No	Midwest	Female	Above Poverty	12	0.1269	0.0357	0.0717	0.2145
No	Midwest	Female	Above Poverty	13	0.1089	0.0264	0.0669	0.1724
No	Midwest	Female	Above Poverty	14	0.1580	0.0478	0.0849	0.2751
No	Midwest	Female	Above Poverty	15	0.0863	0.0213	0.0526	0.1382
No	Midwest	Female	Above Poverty	16	0.1300	0.0319	0.0792	0.2062
No	Midwest	Female	Above Poverty	17	0.0989	0.0236	0.0613	0.1556
No	Midwest	Female	Below Poverty	0	0.0564	0.0353	0.0160	0.1799
No	Midwest	Female	Below Poverty	1	0.0486	0.0183	0.0229	0.1000
No	Midwest	Female	Below Poverty	2	0.0959	0.0434	0.0383	0.2206
No	Midwest	Female	Below Poverty	3	0.0697	0.0338	0.0263	0.1723
No	Midwest	Female	Below Poverty	4	0.1697	0.0387	0.1065	0.2594
No	Midwest	Female	Below Poverty	5	0.0819	0.0265	0.0428	0.1512
No	Midwest	Female	Below Poverty	6	0.0809	0.0357	0.0332	0.1840
No	Midwest	Female	Below Poverty	7	0.0680	0.0325	0.0261	0.1661
No	Midwest	Female	Below Poverty	8	0.1257	0.0346	0.0719	0.2105
No	Midwest	Female	Below Poverty	9	0.1394	0.0398	0.0779	0.2369
No	Midwest	Female	Below Poverty	10	0.1871	0.1071	0.0548	0.4777
No	Midwest	Female	Below Poverty	11	0.0726	0.0266	0.0349	0.1451
No	Midwest	Female	Below Poverty	12	0.1101	0.0452	0.0477	0.2340
No	Midwest	Female	Below Poverty	13	0.1258	0.0354	0.0711	0.2130
No	Midwest	Female	Below Poverty	14	0.0999	0.0435	0.0413	0.2226
No	Midwest	Female	Below Poverty	15	0.1648	0.0745	0.0640	0.3629
No	Midwest	Female	Below Poverty	16	0.1647	0.0576	0.0799	0.3094
No	Midwest	Female	Below Poverty	17	0.1747	0.1141	0.0429	0.4997
No	Midwest	Male	Above Poverty	0	0.0061	0.0044	0.0015	0.0247
No	Midwest	Male	Above Poverty	1	0.0214	0.0175	0.0042	0.1008
No	Midwest	Male	Above Poverty	2	0.0752	0.0222	0.0417	0.1319
No	Midwest	Male	Above Poverty	3	0.0692	0.0203	0.0385	0.1213
No	Midwest	Male	Above Poverty	4	0.0527	0.0201	0.0247	0.1090
No	Midwest	Male	Above Poverty	5	0.1293	0.0303	0.0805	0.2011
No	Midwest	Male	Above Poverty	6	0.0710	0.0193	0.0413	0.1193
No	Midwest	Male	Above Poverty	7	0.1369	0.0301	0.0878	0.2072
No	Midwest	Male	Above Poverty	8	0.1047	0.0299	0.0589	0.1793
No	Midwest	Male	Above Poverty	9	0.1096	0.0269	0.0669	0.1745
No	Midwest	Male	Above Poverty	10	0.1004	0.0281	0.0571	0.1704
No	Midwest	Male	Above Poverty	11	0.1340	0.0348	0.0791	0.2179
No	Midwest	Male	Above Poverty	12	0.1093	0.0242	0.0700	0.1665
No	Midwest	Male	Above Poverty	13	0.1029	0.0210	0.0684	0.1520
No	Midwest	Male	Above Poverty	14	0.1230	0.0236	0.0837	0.1771
No	Midwest	Male	Above Poverty	15	0.1007	0.0305	0.0548	0.1780
No	Midwest	Male	Above Poverty	16	0.1141	0.0268	0.0711	0.1780
No	Midwest	Male	Above Poverty	17	0.0644	0.0193	0.0354	0.1143
No	Midwest	Male	Below Poverty	0	0.0274	0.0175	0.0077	0.0925
No	Midwest	Male	Below Poverty	1	0.0892	0.0369	0.0386	0.1927
No	Midwest	Male	Below Poverty	2	0.1786	0.0652	0.0835	0.3418
No	Midwest	Male	Below Poverty	3	0.1620	0.0475	0.0888	0.2772
No	Midwest	Male	Below Poverty	4	0.2557	0.0634	0.1517	0.3974
No	Midwest	Male	Below Poverty	5	0.1914	0.0400	0.1248	0.2821
No	Midwest	Male	Below Poverty	6	0.1432	0.0333	0.0894	0.2215
No	Midwest	Male	Below Poverty	7	0.1788	0.0378	0.1162	0.2649
No	Midwest	Male	Below Poverty	8	0.2414	0.0604	0.1429	0.3780
No	Midwest	Male	Below Poverty	9	0.1114	0.0404	0.0533	0.2180

Appendix 5C, Attachment A, Table 2. Unsmoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	Midwest	Male	Below Poverty	10	0.2022	0.0624	0.1061	0.3511
No	Midwest	Male	Below Poverty	11	0.1731	0.0406	0.1072	0.2675
No	Midwest	Male	Below Poverty	12	0.2271	0.1064	0.0822	0.4908
No	Midwest	Male	Below Poverty	13	0.1627	0.0591	0.0767	0.3125
No	Midwest	Male	Below Poverty	14	0.0967	0.0413	0.0406	0.2129
No	Midwest	Male	Below Poverty	15	0.1509	0.0506	0.0757	0.2781
No	Midwest	Male	Below Poverty	16	0.1167	0.0490	0.0495	0.2512
No	Midwest	Male	Below Poverty	17	0.3301	0.1005	0.1683	0.5456
No	Northeast	Female	Above Poverty	0	0.0055	0.0054	0.0008	0.0368
No	Northeast	Female	Above Poverty	1	0.0296	0.0164	0.0099	0.0854
No	Northeast	Female	Above Poverty	2	0.0697	0.0252	0.0337	0.1384
No	Northeast	Female	Above Poverty	3	0.0470	0.0158	0.0240	0.0897
No	Northeast	Female	Above Poverty	4	0.0717	0.0199	0.0413	0.1218
No	Northeast	Female	Above Poverty	5	0.0642	0.0196	0.0349	0.1151
No	Northeast	Female	Above Poverty	6	0.0709	0.0254	0.0346	0.1398
No	Northeast	Female	Above Poverty	7	0.0697	0.0180	0.0416	0.1143
No	Northeast	Female	Above Poverty	8	0.0609	0.0209	0.0307	0.1171
No	Northeast	Female	Above Poverty	9	0.0996	0.0334	0.0507	0.1865
No	Northeast	Female	Above Poverty	10	0.0740	0.0260	0.0366	0.1439
No	Northeast	Female	Above Poverty	11	0.1028	0.0305	0.0565	0.1797
No	Northeast	Female	Above Poverty	12	0.0386	0.0187	0.0147	0.0975
No	Northeast	Female	Above Poverty	13	0.0187	0.0095	0.0069	0.0500
No	Northeast	Female	Above Poverty	14	0.0907	0.0181	0.0609	0.1330
No	Northeast	Female	Above Poverty	15	0.1270	0.0344	0.0733	0.2108
No	Northeast	Female	Above Poverty	16	0.0974	0.0267	0.0562	0.1636
No	Northeast	Female	Above Poverty	17	0.1239	0.0375	0.0671	0.2177
No	Northeast	Female	Below Poverty	0	0.0078	0.0078	0.0011	0.0541
No	Northeast	Female	Below Poverty	1	0.1230	0.0576	0.0469	0.2852
No	Northeast	Female	Below Poverty	2	0.0658	0.0272	0.0287	0.1436
No	Northeast	Female	Below Poverty	3	0.1700	0.0576	0.0842	0.3133
No	Northeast	Female	Below Poverty	4	0.1139	0.0456	0.0503	0.2376
No	Northeast	Female	Below Poverty	5	0.2219	0.0583	0.1282	0.3561
No	Northeast	Female	Below Poverty	6	0.0583	0.0290	0.0215	0.1484
No	Northeast	Female	Below Poverty	7	0.0495	0.0252	0.0179	0.1294
No	Northeast	Female	Below Poverty	8	0.0850	0.0368	0.0354	0.1903
No	Northeast	Female	Below Poverty	9	0.0652	0.0294	0.0264	0.1521
No	Northeast	Female	Below Poverty	10	0.0988	0.0440	0.0400	0.2240
No	Northeast	Female	Below Poverty	11	0.2587	0.0734	0.1416	0.4249
No	Northeast	Female	Below Poverty	12	0.0882	0.0426	0.0332	0.2146
No	Northeast	Female	Below Poverty	13	0.3162	0.0739	0.1913	0.4746
No	Northeast	Female	Below Poverty	14	0.1293	0.0372	0.0722	0.2209
No	Northeast	Female	Below Poverty	15	0.1798	0.0479	0.1039	0.2930
No	Northeast	Female	Below Poverty	16	0.1429	0.0381	0.0831	0.2348
No	Northeast	Female	Below Poverty	17	0.1133	0.0426	0.0527	0.2269
No	Northeast	Male	Above Poverty	0	0.0131	0.0101	0.0029	0.0574
No	Northeast	Male	Above Poverty	1	0.0505	0.0227	0.0206	0.1185
No	Northeast	Male	Above Poverty	2	0.0635	0.0220	0.0318	0.1228
No	Northeast	Male	Above Poverty	3	0.0582	0.0216	0.0277	0.1181
No	Northeast	Male	Above Poverty	4	0.1007	0.0281	0.0574	0.1705
No	Northeast	Male	Above Poverty	5	0.1245	0.0318	0.0742	0.2013
No	Northeast	Male	Above Poverty	6	0.1990	0.0511	0.1171	0.3177
No	Northeast	Male	Above Poverty	7	0.1240	0.0274	0.0795	0.1885
No	Northeast	Male	Above Poverty	8	0.1482	0.0321	0.0956	0.2227
No	Northeast	Male	Above Poverty	9	0.0980	0.0321	0.0506	0.1813
No	Northeast	Male	Above Poverty	10	0.0999	0.0216	0.0648	0.1509
No	Northeast	Male	Above Poverty	11	0.1805	0.0342	0.1229	0.2573
No	Northeast	Male	Above Poverty	12	0.1204	0.0211	0.0848	0.1682
No	Northeast	Male	Above Poverty	13	0.0855	0.0237	0.0491	0.1449
No	Northeast	Male	Above Poverty	14	0.1243	0.0351	0.0702	0.2108
No	Northeast	Male	Above Poverty	15	0.1249	0.0247	0.0839	0.1819
No	Northeast	Male	Above Poverty	16	0.1198	0.0283	0.0744	0.1872
No	Northeast	Male	Above Poverty	17	0.0690	0.0173	0.0418	0.1117
No	Northeast	Male	Below Poverty	0	0.0375	0.0275	0.0087	0.1477
No	Northeast	Male	Below Poverty	1	0.1649	0.0506	0.0877	0.2887

Appendix 5C, Attachment A, Table 2. Unsmoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	Northeast	Male	Below Poverty	2	0.1621	0.0496	0.0864	0.2835
No	Northeast	Male	Below Poverty	3	0.1015	0.0440	0.0420	0.2255
No	Northeast	Male	Below Poverty	4	0.2486	0.0909	0.1131	0.4621
No	Northeast	Male	Below Poverty	5	0.1479	0.0487	0.0753	0.2701
No	Northeast	Male	Below Poverty	6	0.2630	0.0391	0.1939	0.3463
No	Northeast	Male	Below Poverty	7	0.1707	0.0507	0.0926	0.2935
No	Northeast	Male	Below Poverty	8	0.2056	0.0966	0.0751	0.4521
No	Northeast	Male	Below Poverty	9	0.3343	0.0680	0.2162	0.4776
No	Northeast	Male	Below Poverty	10	0.2276	0.0786	0.1093	0.4145
No	Northeast	Male	Below Poverty	11	0.1643	0.0600	0.0770	0.3164
No	Northeast	Male	Below Poverty	12	0.1117	0.0389	0.0552	0.2132
No	Northeast	Male	Below Poverty	13	0.1931	0.0430	0.1223	0.2914
No	Northeast	Male	Below Poverty	14	0.1714	0.0664	0.0764	0.3410
No	Northeast	Male	Below Poverty	15	0.2043	0.0555	0.1162	0.3338
No	Northeast	Male	Below Poverty	16	0.1684	0.0501	0.0912	0.2901
No	Northeast	Male	Below Poverty	17	0.2140	0.0526	0.1286	0.3345
No	South	Female	Above Poverty	0	0.0129	0.0080	0.0038	0.0427
No	South	Female	Above Poverty	1	0.0144	0.0076	0.0051	0.0402
No	South	Female	Above Poverty	2	0.0452	0.0169	0.0215	0.0926
No	South	Female	Above Poverty	3	0.0675	0.0196	0.0379	0.1175
No	South	Female	Above Poverty	4	0.0540	0.0150	0.0311	0.0920
No	South	Female	Above Poverty	5	0.0572	0.0138	0.0354	0.0911
No	South	Female	Above Poverty	6	0.1002	0.0186	0.0692	0.1431
No	South	Female	Above Poverty	7	0.0894	0.0191	0.0584	0.1346
No	South	Female	Above Poverty	8	0.0762	0.0160	0.0502	0.1141
No	South	Female	Above Poverty	9	0.0969	0.0210	0.0627	0.1466
No	South	Female	Above Poverty	10	0.0473	0.0135	0.0269	0.0819
No	South	Female	Above Poverty	11	0.0847	0.0165	0.0576	0.1231
No	South	Female	Above Poverty	12	0.0768	0.0152	0.0518	0.1124
No	South	Female	Above Poverty	13	0.0700	0.0158	0.0447	0.1080
No	South	Female	Above Poverty	14	0.1059	0.0211	0.0711	0.1550
No	South	Female	Above Poverty	15	0.0930	0.0186	0.0624	0.1364
No	South	Female	Above Poverty	16	0.0702	0.0156	0.0451	0.1077
No	South	Female	Above Poverty	17	0.0867	0.0162	0.0597	0.1242
No	South	Female	Below Poverty	0	0.0404	0.0203	0.0149	0.1050
No	South	Female	Below Poverty	1	0.0613	0.0183	0.0338	0.1085
No	South	Female	Below Poverty	2	0.0704	0.0193	0.0408	0.1189
No	South	Female	Below Poverty	3	0.0812	0.0254	0.0434	0.1471
No	South	Female	Below Poverty	4	0.1404	0.0367	0.0826	0.2286
No	South	Female	Below Poverty	5	0.1276	0.0304	0.0789	0.1997
No	South	Female	Below Poverty	6	0.0792	0.0288	0.0381	0.1573
No	South	Female	Below Poverty	7	0.1262	0.0305	0.0775	0.1989
No	South	Female	Below Poverty	8	0.1185	0.0290	0.0724	0.1881
No	South	Female	Below Poverty	9	0.1147	0.0286	0.0694	0.1836
No	South	Female	Below Poverty	10	0.1038	0.0301	0.0579	0.1792
No	South	Female	Below Poverty	11	0.1461	0.0366	0.0879	0.2331
No	South	Female	Below Poverty	12	0.1299	0.0490	0.0600	0.2589
No	South	Female	Below Poverty	13	0.1013	0.0262	0.0602	0.1655
No	South	Female	Below Poverty	14	0.1699	0.0385	0.1071	0.2590
No	South	Female	Below Poverty	15	0.1591	0.0365	0.0998	0.2441
No	South	Female	Below Poverty	16	0.0633	0.0273	0.0267	0.1427
No	South	Female	Below Poverty	17	0.0975	0.0299	0.0526	0.1737
No	South	Male	Above Poverty	0	0.0044	0.0025	0.0014	0.0135
No	South	Male	Above Poverty	1	0.0700	0.0162	0.0442	0.1092
No	South	Male	Above Poverty	2	0.0911	0.0195	0.0595	0.1373
No	South	Male	Above Poverty	3	0.0962	0.0206	0.0627	0.1449
No	South	Male	Above Poverty	4	0.1230	0.0259	0.0805	0.1833
No	South	Male	Above Poverty	5	0.1321	0.0204	0.0970	0.1774
No	South	Male	Above Poverty	6	0.0999	0.0192	0.0681	0.1443
No	South	Male	Above Poverty	7	0.1114	0.0214	0.0758	0.1608
No	South	Male	Above Poverty	8	0.0946	0.0168	0.0664	0.1330
No	South	Male	Above Poverty	9	0.1108	0.0202	0.0770	0.1569
No	South	Male	Above Poverty	10	0.1010	0.0186	0.0699	0.1438
No	South	Male	Above Poverty	11	0.0946	0.0175	0.0655	0.1348

Appendix 5C, Attachment A, Table 2. Unsmoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	South	Male	Above Poverty	12	0.1340	0.0207	0.0983	0.1801
No	South	Male	Above Poverty	13	0.1122	0.0226	0.0750	0.1646
No	South	Male	Above Poverty	14	0.0713	0.0153	0.0466	0.1077
No	South	Male	Above Poverty	15	0.0899	0.0158	0.0635	0.1260
No	South	Male	Above Poverty	16	0.0871	0.0147	0.0623	0.1206
No	South	Male	Above Poverty	17	0.0700	0.0178	0.0421	0.1141
No	South	Male	Below Poverty	0	0.0477	0.0162	0.0242	0.0916
No	South	Male	Below Poverty	1	0.0859	0.0197	0.0544	0.1330
No	South	Male	Below Poverty	2	0.0820	0.0201	0.0503	0.1309
No	South	Male	Below Poverty	3	0.1434	0.0319	0.0914	0.2178
No	South	Male	Below Poverty	4	0.1320	0.0265	0.0881	0.1931
No	South	Male	Below Poverty	5	0.2314	0.0486	0.1498	0.3397
No	South	Male	Below Poverty	6	0.1395	0.0302	0.0902	0.2097
No	South	Male	Below Poverty	7	0.1207	0.0269	0.0771	0.1840
No	South	Male	Below Poverty	8	0.2064	0.0474	0.1285	0.3145
No	South	Male	Below Poverty	9	0.1364	0.0279	0.0903	0.2009
No	South	Male	Below Poverty	10	0.1473	0.0315	0.0956	0.2203
No	South	Male	Below Poverty	11	0.1390	0.0286	0.0917	0.2051
No	South	Male	Below Poverty	12	0.1673	0.0339	0.1109	0.2445
No	South	Male	Below Poverty	13	0.1684	0.0449	0.0975	0.2752
No	South	Male	Below Poverty	14	0.0936	0.0305	0.0485	0.1729
No	South	Male	Below Poverty	15	0.1379	0.0353	0.0820	0.2226
No	South	Male	Below Poverty	16	0.0816	0.0275	0.0415	0.1544
No	South	Male	Below Poverty	17	0.1057	0.0289	0.0609	0.1772
No	West	Female	Above Poverty	0	0.0013	0.0013	0.0002	0.0095
No	West	Female	Above Poverty	1	0.0353	0.0202	0.0113	0.1045
No	West	Female	Above Poverty	2	0.0159	0.0076	0.0062	0.0401
No	West	Female	Above Poverty	3	0.0284	0.0132	0.0113	0.0695
No	West	Female	Above Poverty	4	0.0183	0.0071	0.0085	0.0389
No	West	Female	Above Poverty	5	0.0689	0.0276	0.0308	0.1468
No	West	Female	Above Poverty	6	0.0477	0.0166	0.0239	0.0928
No	West	Female	Above Poverty	7	0.0469	0.0144	0.0255	0.0846
No	West	Female	Above Poverty	8	0.0756	0.0263	0.0376	0.1459
No	West	Female	Above Poverty	9	0.0686	0.0196	0.0388	0.1185
No	West	Female	Above Poverty	10	0.0791	0.0250	0.0420	0.1440
No	West	Female	Above Poverty	11	0.0763	0.0124	0.0553	0.1043
No	West	Female	Above Poverty	12	0.1023	0.0260	0.0614	0.1655
No	West	Female	Above Poverty	13	0.0571	0.0163	0.0323	0.0989
No	West	Female	Above Poverty	14	0.1012	0.0251	0.0615	0.1622
No	West	Female	Above Poverty	15	0.0923	0.0207	0.0590	0.1416
No	West	Female	Above Poverty	16	0.0787	0.0214	0.0458	0.1322
No	West	Female	Above Poverty	17	0.1303	0.0294	0.0827	0.1993
No	West	Female	Below Poverty	0	0.0064	0.0064	0.0009	0.0441
No	West	Female	Below Poverty	1	0.0443	0.0195	0.0185	0.1025
No	West	Female	Below Poverty	2	0.0249	0.0153	0.0074	0.0805
No	West	Female	Below Poverty	3	0.0372	0.0137	0.0179	0.0756
No	West	Female	Below Poverty	4	0.0114	0.0102	0.0020	0.0638
No	West	Female	Below Poverty	5	0.0491	0.0294	0.0148	0.1506
No	West	Female	Below Poverty	6	0.1016	0.0419	0.0440	0.2174
No	West	Female	Below Poverty	7	0.0908	0.0302	0.0464	0.1698
No	West	Female	Below Poverty	8	0.0874	0.0258	0.0484	0.1529
No	West	Female	Below Poverty	9	0.0839	0.0267	0.0443	0.1532
No	West	Female	Below Poverty	10	0.0275	0.0137	0.0103	0.0715
No	West	Female	Below Poverty	11	0.0339	0.0160	0.0133	0.0839
No	West	Female	Below Poverty	12	0.0551	0.0254	0.0219	0.1315
No	West	Female	Below Poverty	13	0.1028	0.0393	0.0474	0.2089
No	West	Female	Below Poverty	14	0.1312	0.0440	0.0662	0.2435
No	West	Female	Below Poverty	15	0.0630	0.0247	0.0288	0.1324
No	West	Female	Below Poverty	16	0.0758	0.0287	0.0354	0.1546
No	West	Female	Below Poverty	17	0.0328	0.0163	0.0122	0.0850
No	West	Male	Above Poverty	0	0.0000	0.0000	0.0000	0.0000
No	West	Male	Above Poverty	1	0.0039	0.0040	0.0005	0.0289
No	West	Male	Above Poverty	2	0.0305	0.0113	0.0147	0.0623
No	West	Male	Above Poverty	3	0.0384	0.0129	0.0197	0.0735

Appendix 5C, Attachment A, Table 2. Unsmoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	West	Male	Above Poverty	4	0.1363	0.0261	0.0927	0.1960
No	West	Male	Above Poverty	5	0.0933	0.0268	0.0523	0.1608
No	West	Male	Above Poverty	6	0.0803	0.0208	0.0478	0.1317
No	West	Male	Above Poverty	7	0.1014	0.0320	0.0537	0.1834
No	West	Male	Above Poverty	8	0.0537	0.0182	0.0273	0.1029
No	West	Male	Above Poverty	9	0.1120	0.0242	0.0726	0.1689
No	West	Male	Above Poverty	10	0.1202	0.0253	0.0788	0.1791
No	West	Male	Above Poverty	11	0.1333	0.0271	0.0885	0.1959
No	West	Male	Above Poverty	12	0.1258	0.0286	0.0796	0.1934
No	West	Male	Above Poverty	13	0.1039	0.0328	0.0549	0.1879
No	West	Male	Above Poverty	14	0.0873	0.0217	0.0531	0.1404
No	West	Male	Above Poverty	15	0.0881	0.0222	0.0532	0.1425
No	West	Male	Above Poverty	16	0.1066	0.0230	0.0692	0.1607
No	West	Male	Above Poverty	17	0.1364	0.0284	0.0897	0.2021
No	West	Male	Below Poverty	0	0.0135	0.0128	0.0020	0.0832
No	West	Male	Below Poverty	1	0.0812	0.0317	0.0370	0.1691
No	West	Male	Below Poverty	2	0.0308	0.0080	0.0185	0.0510
No	West	Male	Below Poverty	3	0.0944	0.0311	0.0486	0.1755
No	West	Male	Below Poverty	4	0.1056	0.0306	0.0588	0.1822
No	West	Male	Below Poverty	5	0.0856	0.0256	0.0471	0.1508
No	West	Male	Below Poverty	6	0.1277	0.0356	0.0726	0.2149
No	West	Male	Below Poverty	7	0.0943	0.0353	0.0443	0.1897
No	West	Male	Below Poverty	8	0.1282	0.0343	0.0746	0.2115
No	West	Male	Below Poverty	9	0.0883	0.0287	0.0459	0.1632
No	West	Male	Below Poverty	10	0.0697	0.0228	0.0363	0.1298
No	West	Male	Below Poverty	11	0.0954	0.0365	0.0440	0.1947
No	West	Male	Below Poverty	12	0.0759	0.0316	0.0329	0.1655
No	West	Male	Below Poverty	13	0.0600	0.0276	0.0239	0.1427
No	West	Male	Below Poverty	14	0.1457	0.0391	0.0844	0.2398
No	West	Male	Below Poverty	15	0.1099	0.0551	0.0394	0.2713
No	West	Male	Below Poverty	16	0.0957	0.0350	0.0458	0.1894
No	West	Male	Below Poverty	17	0.1136	0.0421	0.0534	0.2254

Appendix 5C, Attachment A, Table 3. Unsmoothed prevalence for adults "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age_grp**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	Midwest	Female	Above Poverty Level	18-24	0.1633	0.0154	0.1353	0.1958
No	Midwest	Female	Above Poverty Level	25-34	0.1347	0.0096	0.1169	0.1547
No	Midwest	Female	Above Poverty Level	35-44	0.1214	0.0084	0.1059	0.1389
No	Midwest	Female	Above Poverty Level	45-54	0.1157	0.0072	0.1022	0.1306
No	Midwest	Female	Above Poverty Level	55-64	0.1360	0.0103	0.1171	0.1575
No	Midwest	Female	Above Poverty Level	65-74	0.1104	0.0107	0.0910	0.1332
No	Midwest	Female	Above Poverty Level	75+	0.0990	0.0095	0.0819	0.1193
No	Midwest	Female	Below Poverty Level	18-24	0.1990	0.0156	0.1701	0.2314
No	Midwest	Female	Below Poverty Level	25-34	0.1896	0.0177	0.1573	0.2268
No	Midwest	Female	Below Poverty Level	35-44	0.1789	0.0209	0.1415	0.2237
No	Midwest	Female	Below Poverty Level	45-54	0.1903	0.0180	0.1576	0.2281
No	Midwest	Female	Below Poverty Level	55-64	0.2760	0.0255	0.2289	0.3285
No	Midwest	Female	Below Poverty Level	65-74	0.1459	0.0205	0.1101	0.1908
No	Midwest	Female	Below Poverty Level	75+	0.1295	0.0202	0.0948	0.1744
No	Midwest	Male	Above Poverty Level	18-24	0.1658	0.0158	0.1371	0.1990
No	Midwest	Male	Above Poverty Level	25-34	0.1254	0.0092	0.1085	0.1446
No	Midwest	Male	Above Poverty Level	35-44	0.0934	0.0083	0.0784	0.1109
No	Midwest	Male	Above Poverty Level	45-54	0.0659	0.0057	0.0555	0.0779
No	Midwest	Male	Above Poverty Level	55-64	0.0856	0.0086	0.0701	0.1040
No	Midwest	Male	Above Poverty Level	65-74	0.0884	0.0106	0.0697	0.1114
No	Midwest	Male	Above Poverty Level	75+	0.0808	0.0110	0.0617	0.1050
No	Midwest	Male	Below Poverty Level	18-24	0.1672	0.0182	0.1345	0.2060
No	Midwest	Male	Below Poverty Level	25-34	0.1103	0.0156	0.0832	0.1447
No	Midwest	Male	Below Poverty Level	35-44	0.0945	0.0191	0.0632	0.1391
No	Midwest	Male	Below Poverty Level	45-54	0.1445	0.0204	0.1089	0.1893
No	Midwest	Male	Below Poverty Level	55-64	0.1623	0.0203	0.1263	0.2061
No	Midwest	Male	Below Poverty Level	65-74	0.1474	0.0307	0.0968	0.2182
No	Midwest	Male	Below Poverty Level	75+	0.0830	0.0217	0.0492	0.1367
No	Northeast	Female	Above Poverty Level	18-24	0.1834	0.0199	0.1476	0.2256
No	Northeast	Female	Above Poverty Level	25-34	0.1375	0.0107	0.1178	0.1598
No	Northeast	Female	Above Poverty Level	35-44	0.1297	0.0109	0.1097	0.1527
No	Northeast	Female	Above Poverty Level	45-54	0.1209	0.0095	0.1034	0.1409
No	Northeast	Female	Above Poverty Level	55-64	0.1306	0.0106	0.1113	0.1528
No	Northeast	Female	Above Poverty Level	65-74	0.1244	0.0130	0.1010	0.1523
No	Northeast	Female	Above Poverty Level	75+	0.0844	0.0101	0.0666	0.1064
No	Northeast	Female	Below Poverty Level	18-24	0.1642	0.0194	0.1296	0.2059
No	Northeast	Female	Below Poverty Level	25-34	0.1726	0.0170	0.1418	0.2084
No	Northeast	Female	Below Poverty Level	35-44	0.1771	0.0172	0.1459	0.2132
No	Northeast	Female	Below Poverty Level	45-54	0.2140	0.0204	0.1767	0.2567
No	Northeast	Female	Below Poverty Level	55-64	0.2174	0.0232	0.1753	0.2664
No	Northeast	Female	Below Poverty Level	65-74	0.1752	0.0186	0.1417	0.2147
No	Northeast	Female	Below Poverty Level	75+	0.0941	0.0132	0.0712	0.1234
No	Northeast	Male	Above Poverty Level	18-24	0.1658	0.0223	0.1265	0.2142
No	Northeast	Male	Above Poverty Level	25-34	0.1262	0.0126	0.1034	0.1531
No	Northeast	Male	Above Poverty Level	35-44	0.0773	0.0094	0.0607	0.0980
No	Northeast	Male	Above Poverty Level	45-54	0.0976	0.0086	0.0820	0.1158
No	Northeast	Male	Above Poverty Level	55-64	0.0911	0.0096	0.0740	0.1117
No	Northeast	Male	Above Poverty Level	65-74	0.0926	0.0128	0.0704	0.1209
No	Northeast	Male	Above Poverty Level	75+	0.0689	0.0127	0.0478	0.0982
No	Northeast	Male	Below Poverty Level	18-24	0.1753	0.0200	0.1395	0.2179
No	Northeast	Male	Below Poverty Level	25-34	0.1255	0.0178	0.0945	0.1648
No	Northeast	Male	Below Poverty Level	35-44	0.1317	0.0244	0.0909	0.1872
No	Northeast	Male	Below Poverty Level	45-54	0.1189	0.0162	0.0906	0.1545
No	Northeast	Male	Below Poverty Level	55-64	0.1681	0.0490	0.0923	0.2865
No	Northeast	Male	Below Poverty Level	65-74	0.1383	0.0313	0.0875	0.2118
No	Northeast	Male	Below Poverty Level	75+	0.0943	0.0265	0.0536	0.1606
No	South	Female	Above Poverty Level	18-24	0.1501	0.0121	0.1279	0.1754
No	South	Female	Above Poverty Level	25-34	0.1290	0.0084	0.1134	0.1464
No	South	Female	Above Poverty Level	35-44	0.1050	0.0074	0.0914	0.1205
No	South	Female	Above Poverty Level	45-54	0.1163	0.0060	0.1051	0.1285
No	South	Female	Above Poverty Level	55-64	0.1279	0.0087	0.1119	0.1459
No	South	Female	Above Poverty Level	65-74	0.1231	0.0102	0.1044	0.1446
No	South	Female	Above Poverty Level	75+	0.0939	0.0092	0.0773	0.1136
No	South	Female	Below Poverty Level	18-24	0.1511	0.0133	0.1269	0.1790

Appendix 5C, Attachment A, Table 3. Unsmoothed prevalence for adults "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age_grp**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	South	Female	Below Poverty Level	25-34	0.1336	0.0087	0.1175	0.1515
No	South	Female	Below Poverty Level	35-44	0.1452	0.0125	0.1224	0.1714
No	South	Female	Below Poverty Level	45-54	0.1622	0.0128	0.1386	0.1889
No	South	Female	Below Poverty Level	55-64	0.2039	0.0179	0.1711	0.2413
No	South	Female	Below Poverty Level	65-74	0.1616	0.0163	0.1321	0.1962
No	South	Female	Below Poverty Level	75+	0.1127	0.0133	0.0891	0.1415
No	South	Male	Above Poverty Level	18-24	0.1438	0.0100	0.1253	0.1645
No	South	Male	Above Poverty Level	25-34	0.1095	0.0078	0.0952	0.1258
No	South	Male	Above Poverty Level	35-44	0.0890	0.0066	0.0769	0.1027
No	South	Male	Above Poverty Level	45-54	0.0704	0.0051	0.0610	0.0811
No	South	Male	Above Poverty Level	55-64	0.0782	0.0071	0.0654	0.0932
No	South	Male	Above Poverty Level	65-74	0.0789	0.0078	0.0649	0.0956
No	South	Male	Above Poverty Level	75+	0.0893	0.0111	0.0698	0.1135
No	South	Male	Below Poverty Level	18-24	0.1473	0.0152	0.1199	0.1797
No	South	Male	Below Poverty Level	25-34	0.0914	0.0122	0.0701	0.1184
No	South	Male	Below Poverty Level	35-44	0.0972	0.0139	0.0732	0.1280
No	South	Male	Below Poverty Level	45-54	0.1062	0.0138	0.0821	0.1363
No	South	Male	Below Poverty Level	55-64	0.1068	0.0156	0.0799	0.1414
No	South	Male	Below Poverty Level	65-74	0.0966	0.0149	0.0710	0.1301
No	South	Male	Below Poverty Level	75+	0.0702	0.0130	0.0486	0.1004
No	West	Female	Above Poverty Level	18-24	0.1595	0.0150	0.1323	0.1911
No	West	Female	Above Poverty Level	25-34	0.1387	0.0096	0.1209	0.1586
No	West	Female	Above Poverty Level	35-44	0.1368	0.0109	0.1168	0.1595
No	West	Female	Above Poverty Level	45-54	0.1431	0.0092	0.1261	0.1621
No	West	Female	Above Poverty Level	55-64	0.1478	0.0094	0.1303	0.1671
No	West	Female	Above Poverty Level	65-74	0.1541	0.0130	0.1302	0.1813
No	West	Female	Above Poverty Level	75+	0.1231	0.0117	0.1020	0.1479
No	West	Female	Below Poverty Level	18-24	0.1522	0.0184	0.1195	0.1920
No	West	Female	Below Poverty Level	25-34	0.1191	0.0118	0.0978	0.1441
No	West	Female	Below Poverty Level	35-44	0.1466	0.0182	0.1145	0.1859
No	West	Female	Below Poverty Level	45-54	0.1874	0.0219	0.1483	0.2341
No	West	Female	Below Poverty Level	55-64	0.1747	0.0181	0.1419	0.2131
No	West	Female	Below Poverty Level	65-74	0.1318	0.0179	0.1005	0.1709
No	West	Female	Below Poverty Level	75+	0.1370	0.0198	0.1027	0.1806
No	West	Male	Above Poverty Level	18-24	0.1499	0.0188	0.1167	0.1905
No	West	Male	Above Poverty Level	25-34	0.1304	0.0107	0.1108	0.1527
No	West	Male	Above Poverty Level	35-44	0.0984	0.0080	0.0837	0.1153
No	West	Male	Above Poverty Level	45-54	0.0944	0.0081	0.0796	0.1116
No	West	Male	Above Poverty Level	55-64	0.0917	0.0075	0.0780	0.1076
No	West	Male	Above Poverty Level	65-74	0.1168	0.0126	0.0943	0.1438
No	West	Male	Above Poverty Level	75+	0.1208	0.0160	0.0928	0.1558
No	West	Male	Below Poverty Level	18-24	0.1589	0.0222	0.1201	0.2073
No	West	Male	Below Poverty Level	25-34	0.0846	0.0128	0.0626	0.1133
No	West	Male	Below Poverty Level	35-44	0.0760	0.0135	0.0535	0.1069
No	West	Male	Below Poverty Level	45-54	0.1422	0.0214	0.1052	0.1894
No	West	Male	Below Poverty Level	55-64	0.0979	0.0176	0.0684	0.1381
No	West	Male	Below Poverty Level	65-74	0.1349	0.0323	0.0831	0.2116
No	West	Male	Below Poverty Level	75+	0.0937	0.0194	0.0620	0.1393

Appendix 5C, Attachment A, Table 4. Unsmoothed prevalence for adults "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age_grp**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	Midwest	Female	Above Poverty Level	18-24	0.1062	0.0133	0.0828	0.1354
No	Midwest	Female	Above Poverty Level	25-34	0.0859	0.0090	0.0699	0.1052
No	Midwest	Female	Above Poverty Level	35-44	0.0859	0.0081	0.0713	0.1031
No	Midwest	Female	Above Poverty Level	45-44	0.0858	0.0061	0.0746	0.0986
No	Midwest	Female	Above Poverty Level	55-64	0.0996	0.0090	0.0832	0.1188
No	Midwest	Female	Above Poverty Level	65-74	0.0755	0.0083	0.0608	0.0934
No	Midwest	Female	Above Poverty Level	75+	0.0643	0.0073	0.0514	0.0802
No	Midwest	Female	Below Poverty Level	18-24	0.1306	0.0144	0.1049	0.1614
No	Midwest	Female	Below Poverty Level	25-34	0.1329	0.0143	0.1073	0.1634
No	Midwest	Female	Below Poverty Level	35-44	0.1354	0.0187	0.1027	0.1764
No	Midwest	Female	Below Poverty Level	45-44	0.1398	0.0166	0.1102	0.1757
No	Midwest	Female	Below Poverty Level	55-64	0.2110	0.0221	0.1709	0.2575
No	Midwest	Female	Below Poverty Level	65-74	0.1190	0.0180	0.0879	0.1590
No	Midwest	Female	Below Poverty Level	75+	0.1029	0.0183	0.0722	0.1448
No	Midwest	Male	Above Poverty Level	18-24	0.0790	0.0125	0.0577	0.1071
No	Midwest	Male	Above Poverty Level	25-34	0.0599	0.0066	0.0482	0.0743
No	Midwest	Male	Above Poverty Level	35-44	0.0486	0.0063	0.0377	0.0625
No	Midwest	Male	Above Poverty Level	45-44	0.0447	0.0049	0.0360	0.0554
No	Midwest	Male	Above Poverty Level	55-64	0.0555	0.0059	0.0450	0.0683
No	Midwest	Male	Above Poverty Level	65-74	0.0524	0.0076	0.0394	0.0694
No	Midwest	Male	Above Poverty Level	75+	0.0477	0.0088	0.0331	0.0682
No	Midwest	Male	Below Poverty Level	18-24	0.0938	0.0143	0.0693	0.1258
No	Midwest	Male	Below Poverty Level	25-34	0.0572	0.0137	0.0355	0.0908
No	Midwest	Male	Below Poverty Level	35-44	0.0731	0.0162	0.0470	0.1119
No	Midwest	Male	Below Poverty Level	45-44	0.0969	0.0208	0.0630	0.1461
No	Midwest	Male	Below Poverty Level	55-64	0.1350	0.0205	0.0997	0.1804
No	Midwest	Male	Below Poverty Level	65-74	0.1349	0.0294	0.0869	0.2035
No	Midwest	Male	Below Poverty Level	75+	0.0643	0.0213	0.0332	0.1208
No	Northeast	Female	Above Poverty Level	18-24	0.1123	0.0148	0.0864	0.1447
No	Northeast	Female	Above Poverty Level	25-34	0.0917	0.0102	0.0735	0.1138
No	Northeast	Female	Above Poverty Level	35-44	0.0944	0.0092	0.0778	0.1141
No	Northeast	Female	Above Poverty Level	45-44	0.0858	0.0080	0.0714	0.1029
No	Northeast	Female	Above Poverty Level	55-64	0.0945	0.0086	0.0790	0.1127
No	Northeast	Female	Above Poverty Level	65-74	0.0898	0.0106	0.0711	0.1128
No	Northeast	Female	Above Poverty Level	75+	0.0706	0.0098	0.0537	0.0924
No	Northeast	Female	Below Poverty Level	18-24	0.1232	0.0182	0.0918	0.1634
No	Northeast	Female	Below Poverty Level	25-34	0.1180	0.0147	0.0921	0.1499
No	Northeast	Female	Below Poverty Level	35-44	0.1265	0.0138	0.1018	0.1560
No	Northeast	Female	Below Poverty Level	45-44	0.1745	0.0185	0.1412	0.2137
No	Northeast	Female	Below Poverty Level	55-64	0.1744	0.0211	0.1369	0.2196
No	Northeast	Female	Below Poverty Level	65-74	0.1388	0.0148	0.1123	0.1704
No	Northeast	Female	Below Poverty Level	75+	0.0488	0.0088	0.0341	0.0693
No	Northeast	Male	Above Poverty Level	18-24	0.0888	0.0161	0.0620	0.1257
No	Northeast	Male	Above Poverty Level	25-34	0.0655	0.0093	0.0495	0.0862
No	Northeast	Male	Above Poverty Level	35-44	0.0409	0.0061	0.0304	0.0547
No	Northeast	Male	Above Poverty Level	45-44	0.0564	0.0078	0.0429	0.0738
No	Northeast	Male	Above Poverty Level	55-64	0.0469	0.0085	0.0328	0.0667
No	Northeast	Male	Above Poverty Level	65-74	0.0641	0.0105	0.0463	0.0880
No	Northeast	Male	Above Poverty Level	75+	0.0527	0.0110	0.0348	0.0789
No	Northeast	Male	Below Poverty Level	18-24	0.0780	0.0129	0.0562	0.1075
No	Northeast	Male	Below Poverty Level	25-34	0.0847	0.0171	0.0566	0.1248
No	Northeast	Male	Below Poverty Level	35-44	0.0795	0.0212	0.0467	0.1322
No	Northeast	Male	Below Poverty Level	45-44	0.0798	0.0196	0.0489	0.1275
No	Northeast	Male	Below Poverty Level	55-64	0.1322	0.0492	0.0617	0.2608
No	Northeast	Male	Below Poverty Level	65-74	0.1055	0.0296	0.0600	0.1789
No	Northeast	Male	Below Poverty Level	75+	0.0758	0.0247	0.0395	0.1406
No	South	Female	Above Poverty Level	18-24	0.0893	0.0090	0.0732	0.1086
No	South	Female	Above Poverty Level	25-34	0.0731	0.0064	0.0615	0.0866
No	South	Female	Above Poverty Level	35-44	0.0689	0.0051	0.0595	0.0797
No	South	Female	Above Poverty Level	45-44	0.0716	0.0049	0.0626	0.0818
No	South	Female	Above Poverty Level	55-64	0.0865	0.0064	0.0747	0.1000
No	South	Female	Above Poverty Level	65-74	0.0914	0.0090	0.0753	0.1105
No	South	Female	Above Poverty Level	75+	0.0599	0.0072	0.0473	0.0756
No	South	Female	Below Poverty Level	18-24	0.0996	0.0119	0.0786	0.1254

Appendix 5C, Attachment A, Table 4. Unsmoothed prevalence for adults "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age_grp**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
No	South	Female	Below Poverty Level	25-34	0.0867	0.0079	0.0725	0.1035
No	South	Female	Below Poverty Level	35-44	0.1152	0.0113	0.0948	0.1393
No	South	Female	Below Poverty Level	45-44	0.1369	0.0123	0.1144	0.1629
No	South	Female	Below Poverty Level	55-64	0.1780	0.0173	0.1467	0.2144
No	South	Female	Below Poverty Level	65-74	0.1303	0.0152	0.1033	0.1631
No	South	Female	Below Poverty Level	75+	0.0895	0.0118	0.0689	0.1154
No	South	Male	Above Poverty Level	18-24	0.0608	0.0079	0.0471	0.0782
No	South	Male	Above Poverty Level	25-34	0.0471	0.0053	0.0377	0.0587
No	South	Male	Above Poverty Level	35-44	0.0451	0.0048	0.0365	0.0556
No	South	Male	Above Poverty Level	45-44	0.0359	0.0040	0.0288	0.0446
No	South	Male	Above Poverty Level	55-64	0.0413	0.0055	0.0317	0.0535
No	South	Male	Above Poverty Level	65-74	0.0441	0.0057	0.0342	0.0567
No	South	Male	Above Poverty Level	75+	0.0636	0.0097	0.0470	0.0855
No	South	Male	Below Poverty Level	18-24	0.0617	0.0086	0.0468	0.0810
No	South	Male	Below Poverty Level	25-34	0.0344	0.0064	0.0239	0.0494
No	South	Male	Below Poverty Level	35-44	0.0488	0.0109	0.0314	0.0751
No	South	Male	Below Poverty Level	45-44	0.0800	0.0131	0.0579	0.1097
No	South	Male	Below Poverty Level	55-64	0.0676	0.0122	0.0473	0.0957
No	South	Male	Below Poverty Level	65-74	0.0687	0.0129	0.0473	0.0987
No	South	Male	Below Poverty Level	75+	0.0331	0.0083	0.0202	0.0539
No	West	Female	Above Poverty Level	18-24	0.0908	0.0143	0.0663	0.1231
No	West	Female	Above Poverty Level	25-34	0.0819	0.0070	0.0691	0.0968
No	West	Female	Above Poverty Level	35-44	0.0994	0.0090	0.0830	0.1186
No	West	Female	Above Poverty Level	45-44	0.0937	0.0095	0.0766	0.1141
No	West	Female	Above Poverty Level	55-64	0.1013	0.0087	0.0854	0.1197
No	West	Female	Above Poverty Level	65-74	0.1103	0.0114	0.0898	0.1347
No	West	Female	Above Poverty Level	75+	0.0783	0.0092	0.0621	0.0982
No	West	Female	Below Poverty Level	18-24	0.0901	0.0135	0.0669	0.1202
No	West	Female	Below Poverty Level	25-34	0.0861	0.0111	0.0667	0.1105
No	West	Female	Below Poverty Level	35-44	0.1081	0.0143	0.0831	0.1394
No	West	Female	Below Poverty Level	45-44	0.1391	0.0179	0.1075	0.1781
No	West	Female	Below Poverty Level	55-64	0.1293	0.0164	0.1005	0.1648
No	West	Female	Below Poverty Level	65-74	0.1053	0.0166	0.0770	0.1425
No	West	Female	Below Poverty Level	75+	0.1061	0.0162	0.0782	0.1424
No	West	Male	Above Poverty Level	18-24	0.0620	0.0104	0.0445	0.0858
No	West	Male	Above Poverty Level	25-34	0.0528	0.0068	0.0410	0.0679
No	West	Male	Above Poverty Level	35-44	0.0582	0.0061	0.0473	0.0715
No	West	Male	Above Poverty Level	45-44	0.0499	0.0065	0.0386	0.0642
No	West	Male	Above Poverty Level	55-64	0.0542	0.0072	0.0416	0.0702
No	West	Male	Above Poverty Level	65-74	0.0756	0.0102	0.0579	0.0982
No	West	Male	Above Poverty Level	75+	0.0711	0.0133	0.0491	0.1019
No	West	Male	Below Poverty Level	18-24	0.0741	0.0132	0.0520	0.1046
No	West	Male	Below Poverty Level	25-34	0.0457	0.0097	0.0301	0.0689
No	West	Male	Below Poverty Level	35-44	0.0344	0.0089	0.0207	0.0568
No	West	Male	Below Poverty Level	45-44	0.1119	0.0198	0.0786	0.1570
No	West	Male	Below Poverty Level	55-64	0.0528	0.0137	0.0316	0.0870
No	West	Male	Below Poverty Level	65-74	0.1159	0.0336	0.0644	0.1996
No	West	Male	Below Poverty Level	75+	0.0442	0.0131	0.0246	0.0781

Figure 1. Raw asthma 'EVER' prevalence rates and confidence intervals
region=Midwest pov_rat=Above Poverty Level

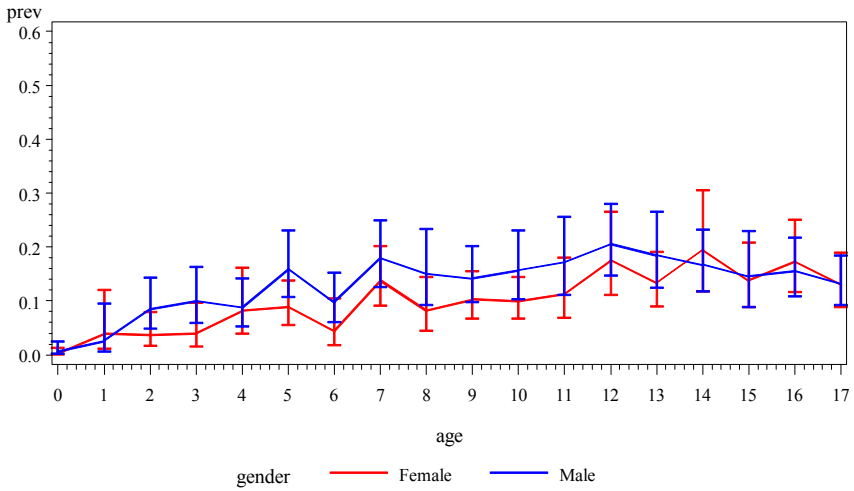


Figure 1. Raw asthma 'EVER' prevalence rates and confidence intervals
region=Northeast pov_rat=Above Poverty Level

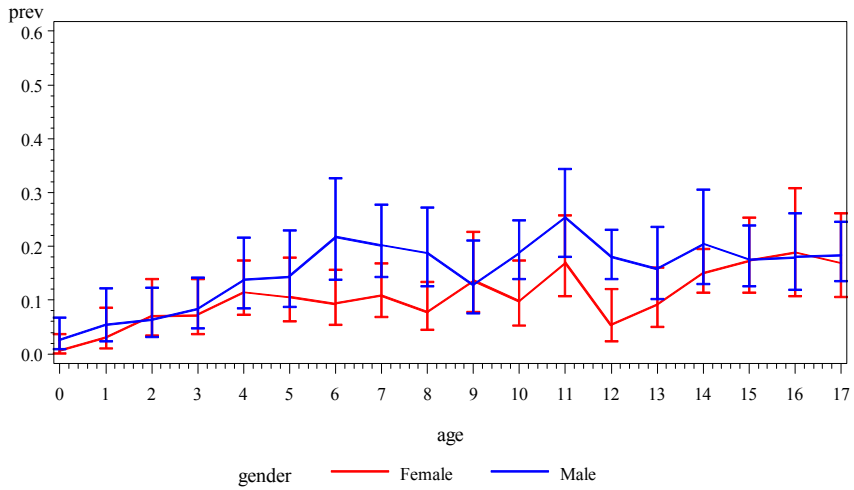


Figure 1. Raw asthma 'EVER' prevalence rates and confidence intervals
region=Midwest pov_rat=Below Poverty Level

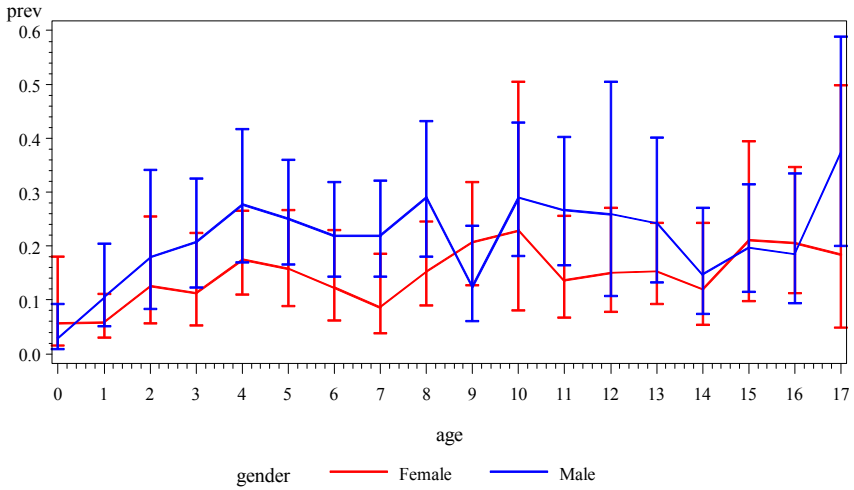
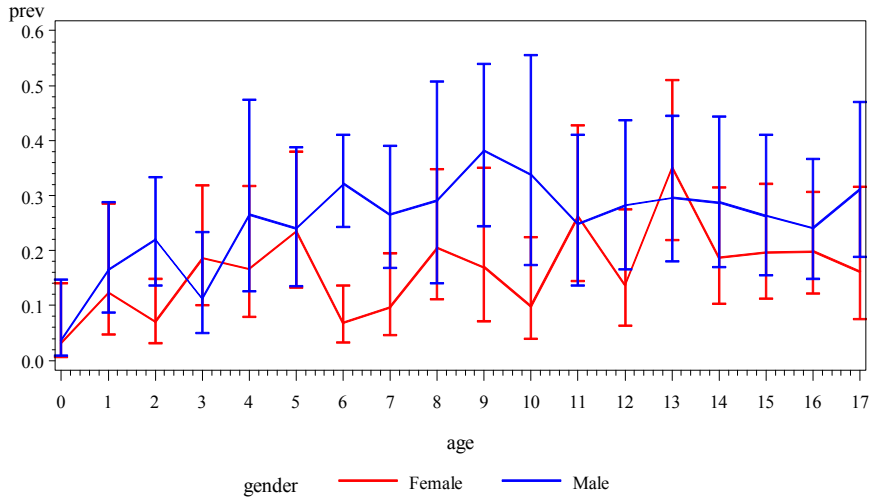


Figure 1. Raw asthma 'EVER' prevalence rates and confidence intervals
region=Northeast pov_rat=Below Poverty Level



Appendix 5C, Attachment A, Figure 1. Unsmoothed prevalence and confidence intervals for children 'EVER' having asthma.

Figure 1. Raw asthma 'EVER' prevalence rates and confidence intervals
region=South pov_rat=Above Poverty Level

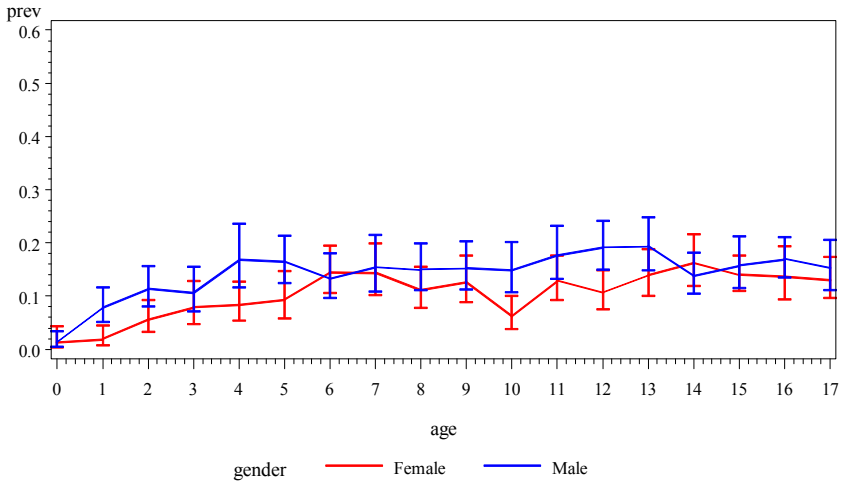


Figure 1. Raw asthma 'EVER' prevalence rates and confidence intervals
region=West pov_rat=Above Poverty Level

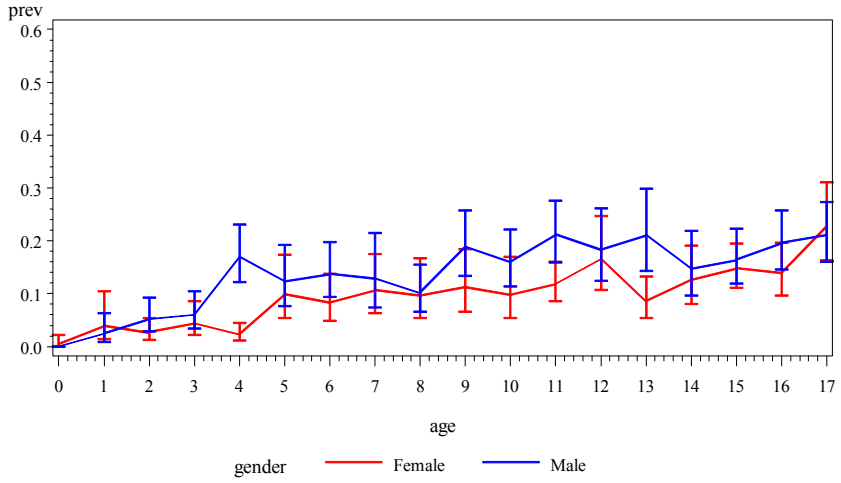


Figure 1. Raw asthma 'EVER' prevalence rates and confidence intervals
region=South pov_rat=Below Poverty Level

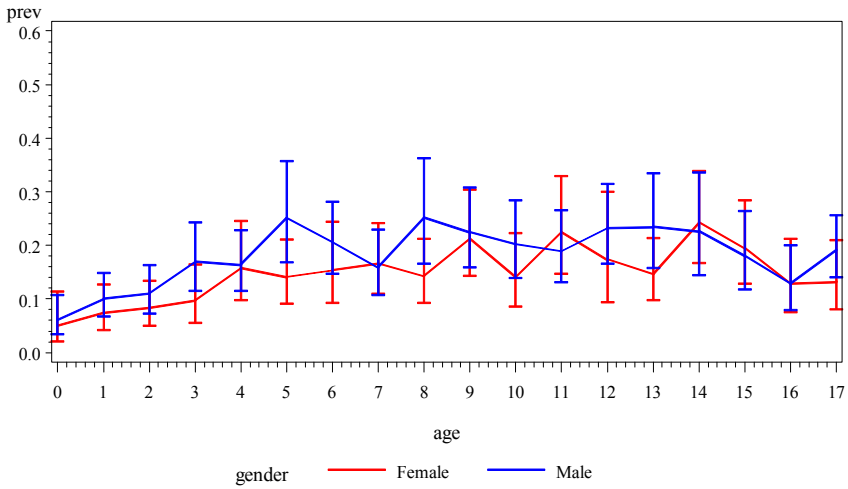
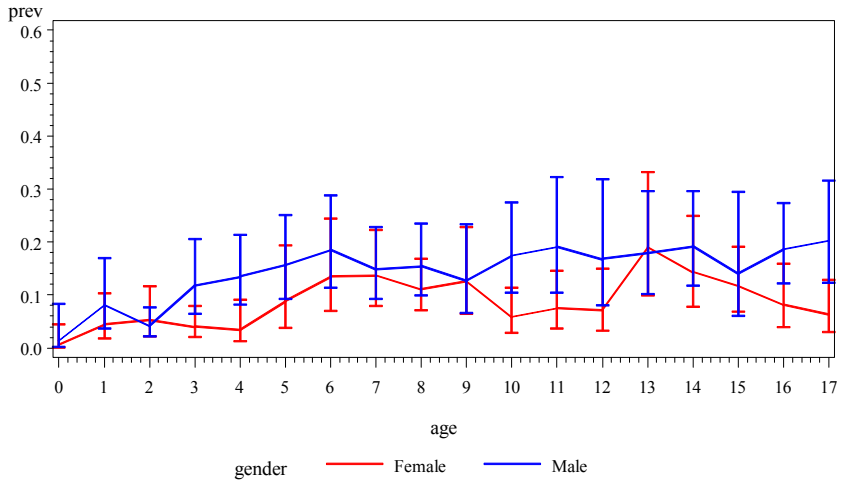


Figure 1. Raw asthma 'EVER' prevalence rates and confidence intervals
region=West pov_rat=Below Poverty Level



Appendix 5C, Attachment A, Figure 1, cont. Unsmoothed prevalence and confidence intervals for children 'EVER' having asthma.

Figure 2. Raw asthma 'STILL' prevalence rates and confidence intervals
region=Midwest pov_rat=Below Poverty Level

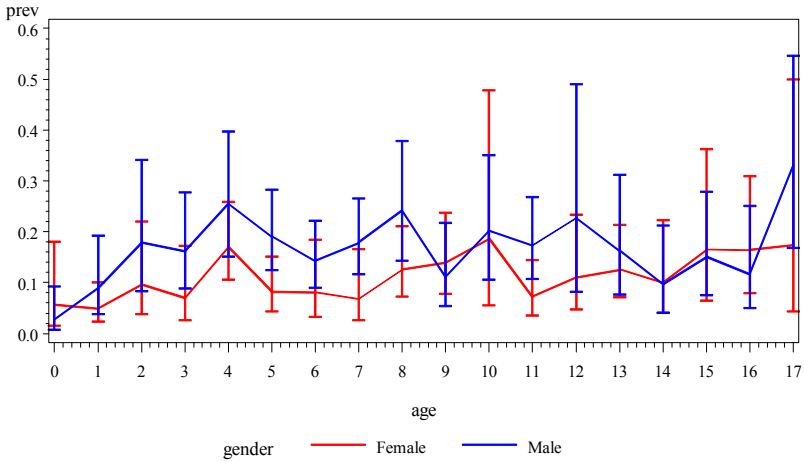


Figure 2. Raw asthma 'STILL' prevalence rates and confidence intervals
region=Northeast pov_rat=Above Poverty Level

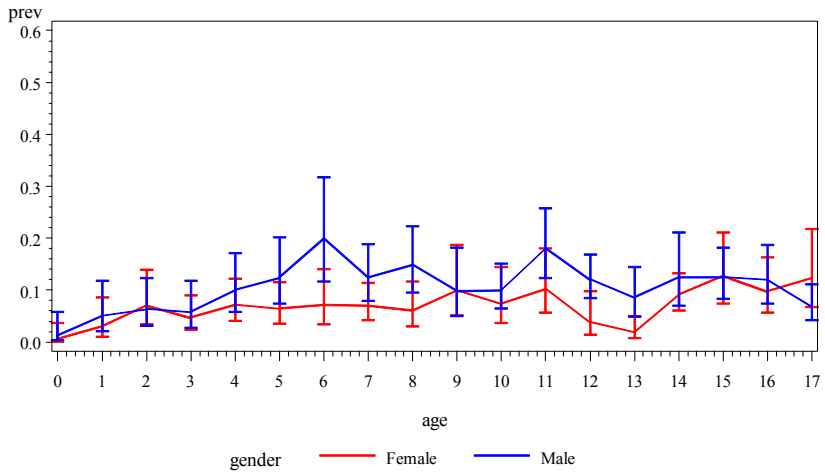


Figure 2. Raw asthma 'STILL' prevalence rates and confidence intervals
region=Midwest pov_rat=Above Poverty Level

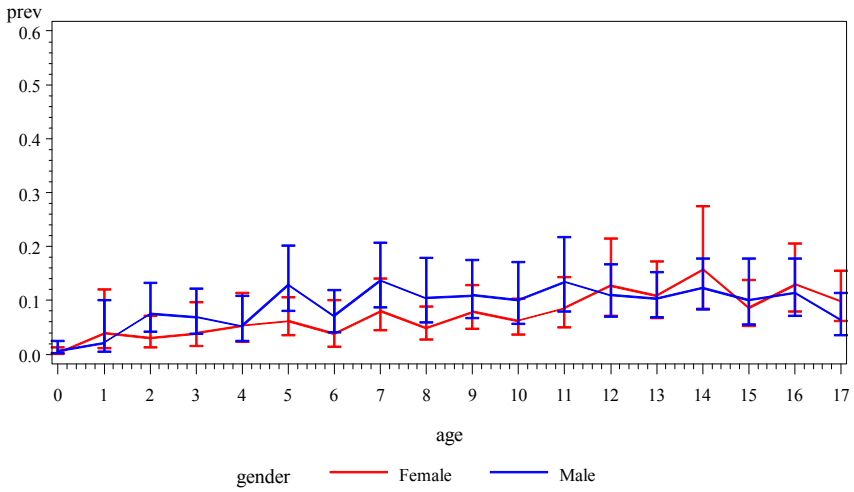
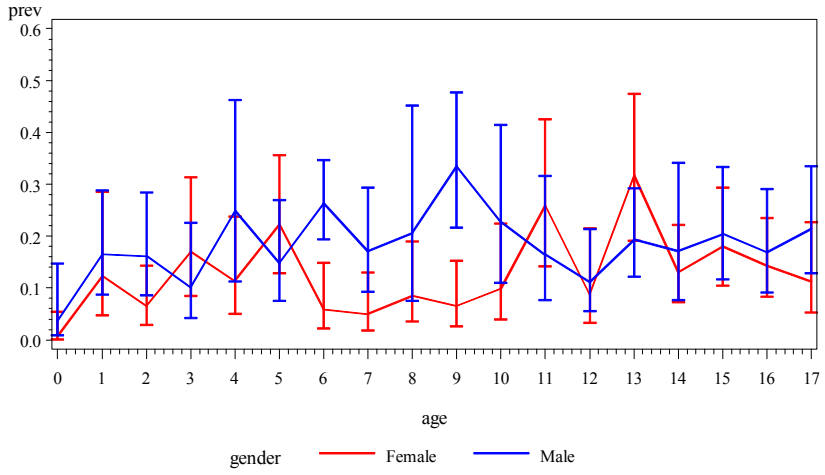


Figure 2. Raw asthma 'STILL' prevalence rates and confidence intervals
region=Northeast pov_rat=Below Poverty Level



Appendix 5C, Attachment A, Figure 2. Unsmoothed prevalence and confidence intervals for children 'STILL' having asthma.

Figure 2. Raw asthma 'STILL' prevalence rates and confidence intervals
region=South pov_rat=Above Poverty Level

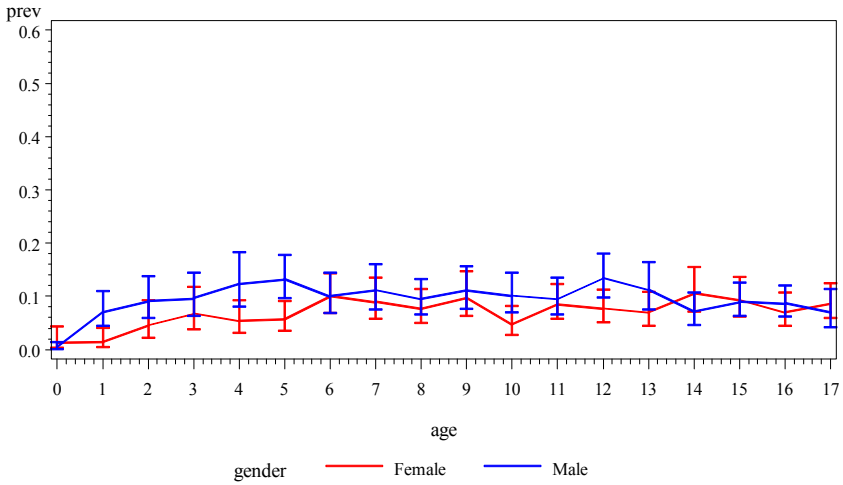


Figure 2. Raw asthma 'STILL' prevalence rates and confidence intervals
region=West pov_rat=Above Poverty Level

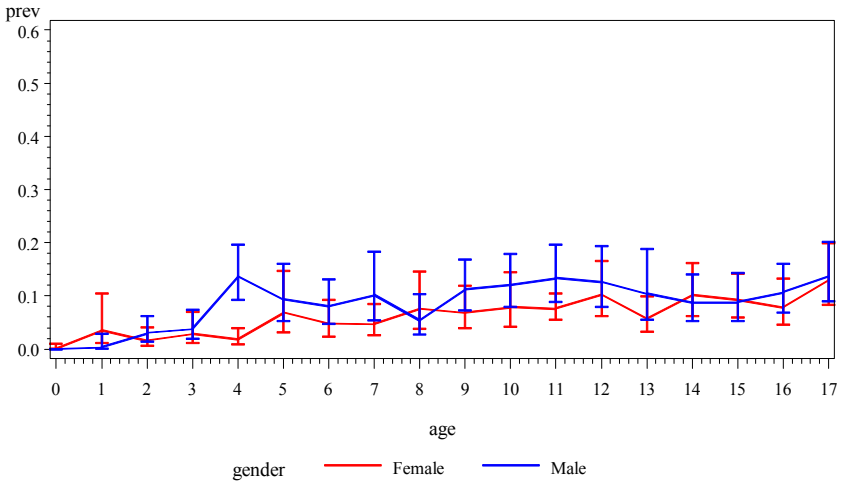


Figure 2. Raw asthma 'STILL' prevalence rates and confidence intervals
region=South pov_rat=Below Poverty Level

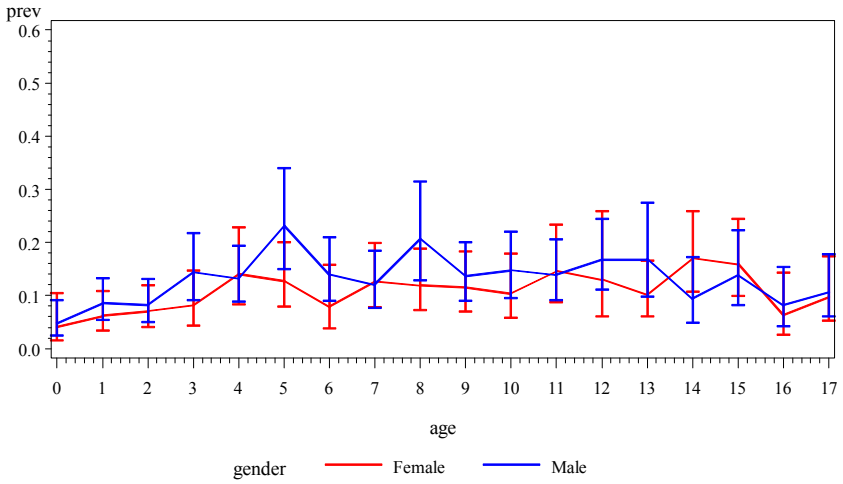
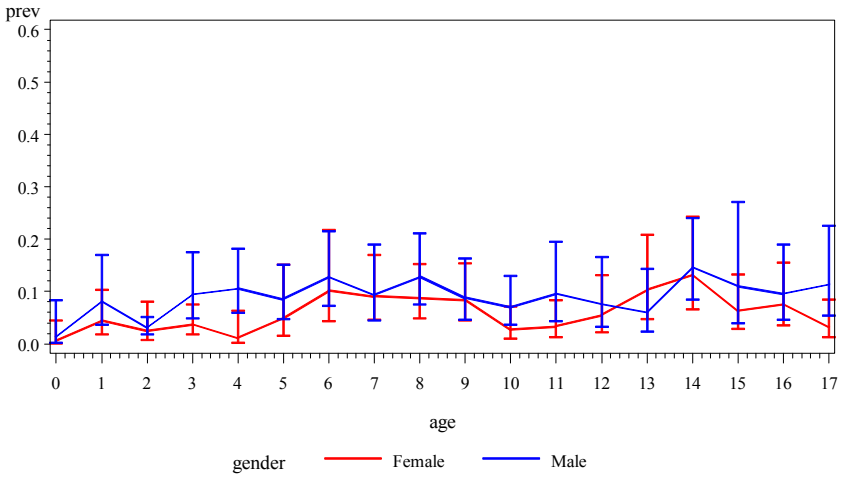


Figure 2. Raw asthma 'STILL' prevalence rates and confidence intervals
region=West pov_rat=Below Poverty Level



Appendix 5C, Attachment A, Figure 2, cont. Unsmoothed prevalence and confidence intervals for children 'STILL' having asthma.

Figure 3. Raw adult asthma 'EVER' prevalence rates and confidence intervals
region=Midwest pov_rat=Above Poverty Level

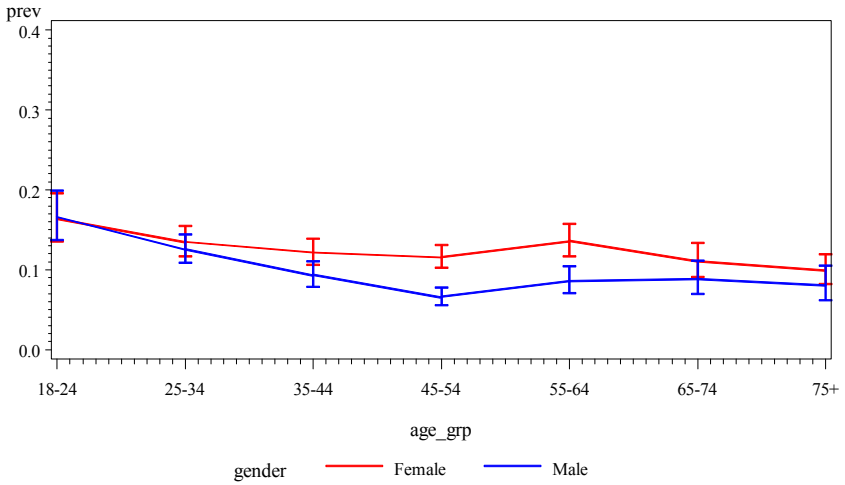


Figure 3. Raw adult asthma 'EVER' prevalence rates and confidence intervals
region=Northeast pov_rat=Above Poverty Level

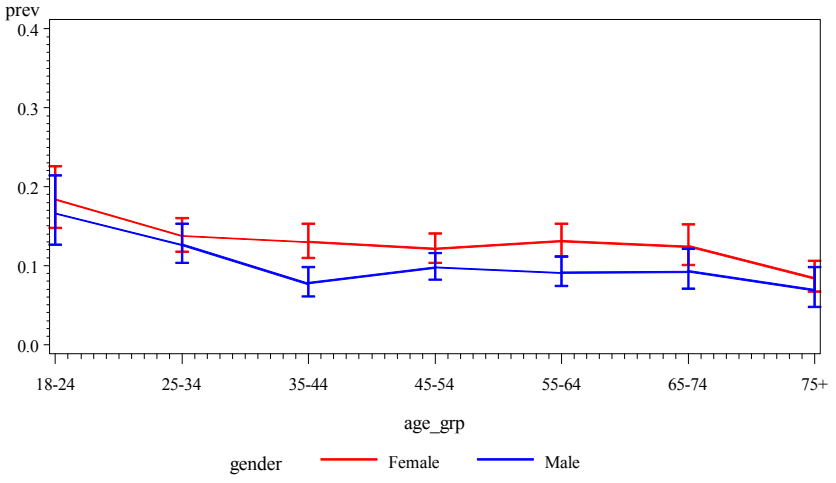


Figure 3. Raw adult asthma 'EVER' prevalence rates and confidence intervals
region=Midwest pov_rat=Below Poverty Level

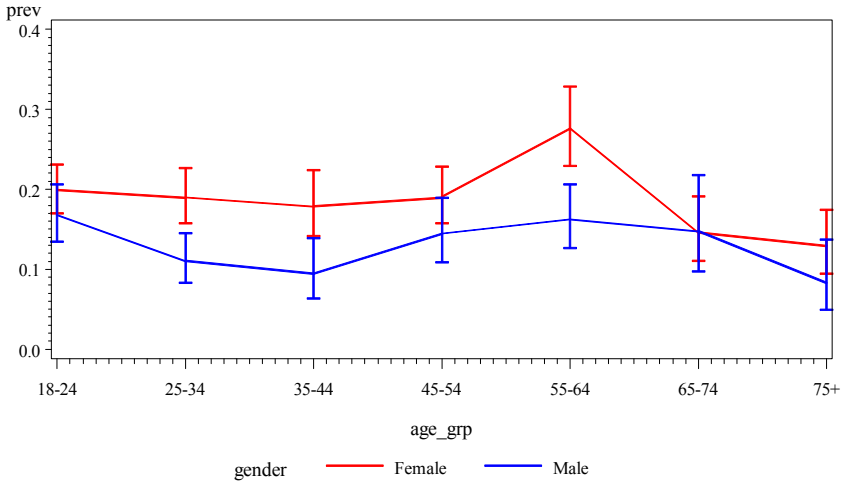
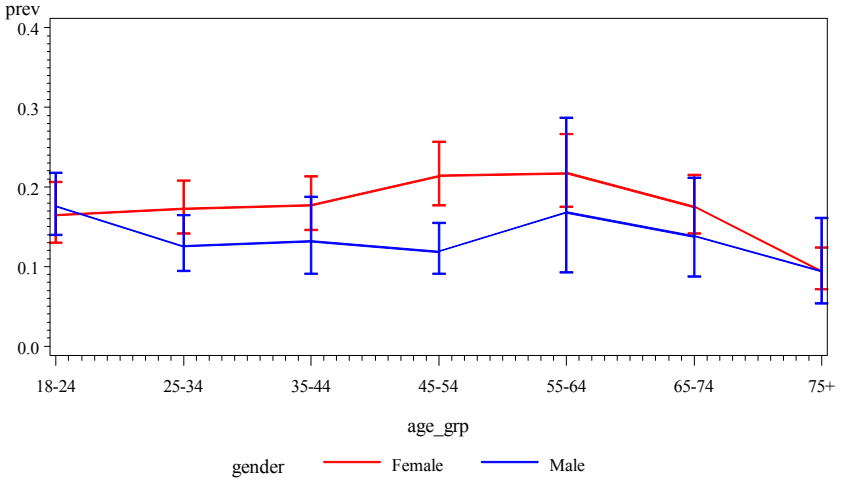


Figure 3. Raw adult asthma 'EVER' prevalence rates and confidence intervals
region=Northeast pov_rat=Below Poverty Level



Appendix 5C, Attachment A, Figure 3. Unsmoothed prevalence and confidence intervals for adults 'EVER' having asthma.

Figure 3. Raw adult asthma 'EVER' prevalence rates and confidence intervals
region=South pov_rat=Above Poverty Level

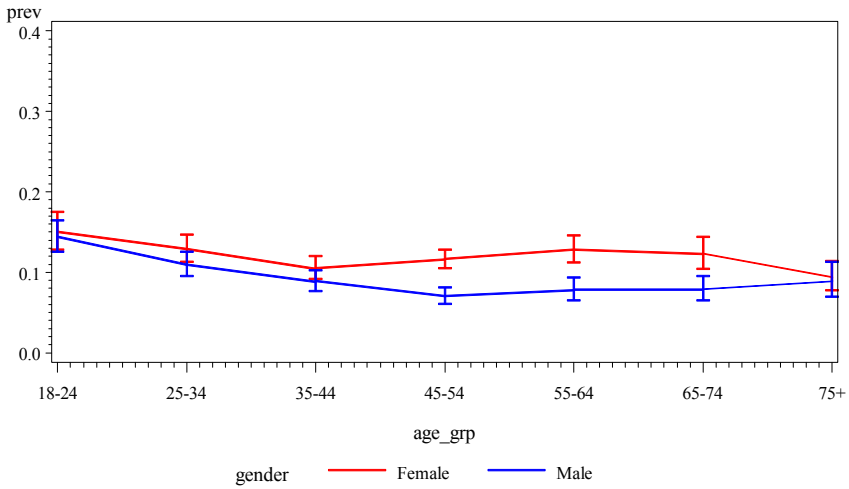


Figure 3. Raw adult asthma 'EVER' prevalence rates and confidence intervals
region=West pov_rat=Above Poverty Level

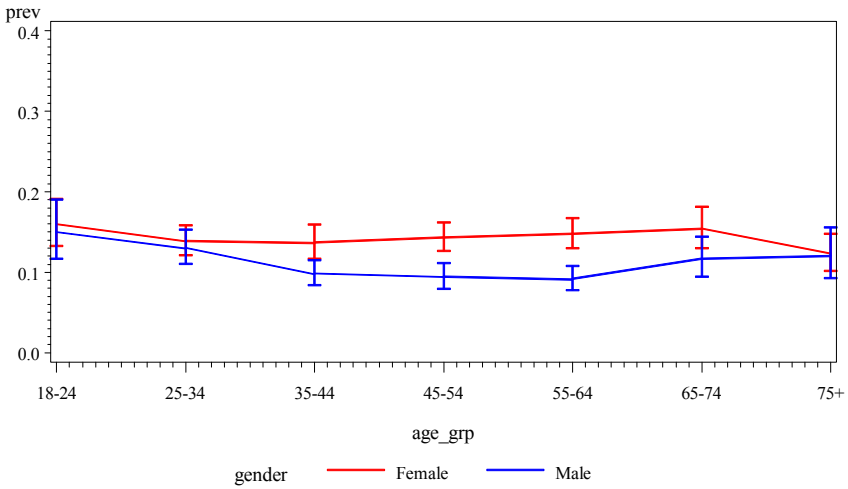


Figure 3. Raw adult asthma 'EVER' prevalence rates and confidence intervals
region=South pov_rat=Below Poverty Level

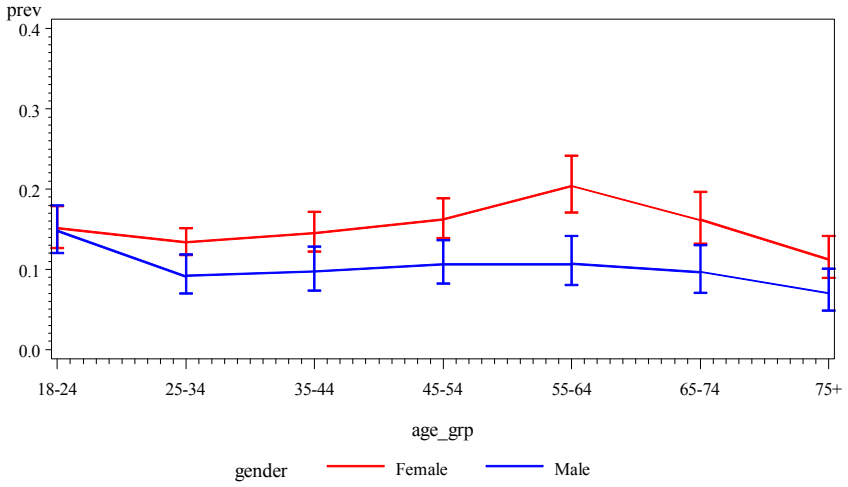
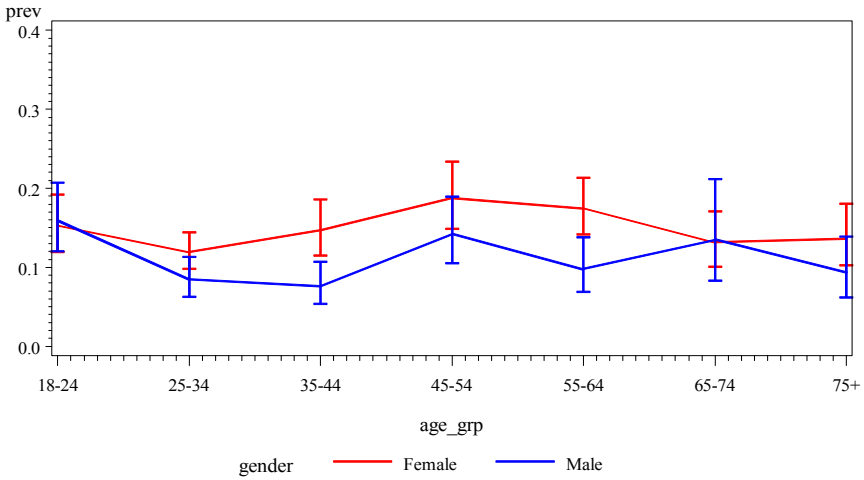


Figure 3. Raw adult asthma 'EVER' prevalence rates and confidence intervals
region=West pov_rat=Below Poverty Level



Appendix 5C, Attachment A, Figure 3, cont. Unsmoothed prevalence and confidence intervals for adults 'EVER' having asthma.

Figure 4. Raw adult asthma 'STILL' prevalence rates and confidence intervals
region=Midwest pov_rat=Above Poverty Level

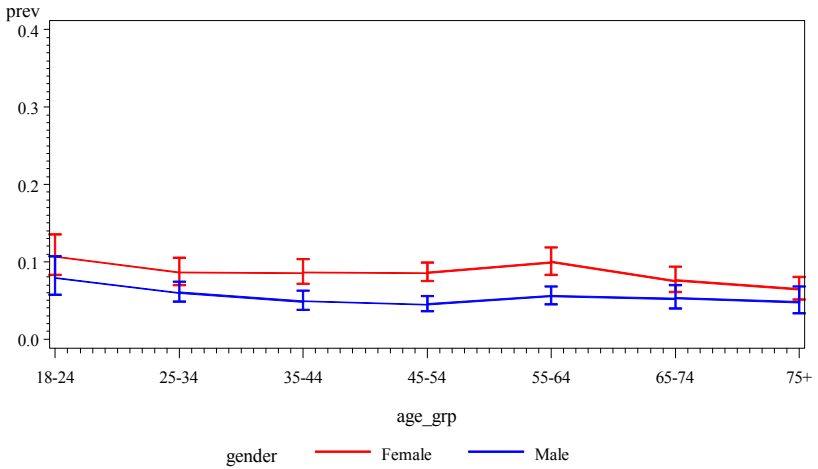


Figure 4. Raw adult asthma 'STILL' prevalence rates and confidence intervals
region=Northeast pov_rat=Above Poverty Level

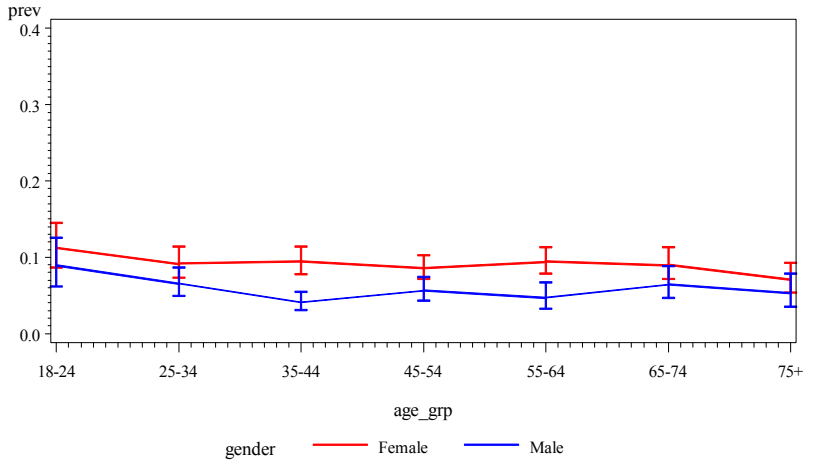


Figure 4. Raw adult asthma 'STILL' prevalence rates and confidence intervals
region=Midwest pov_rat=Below Poverty Level

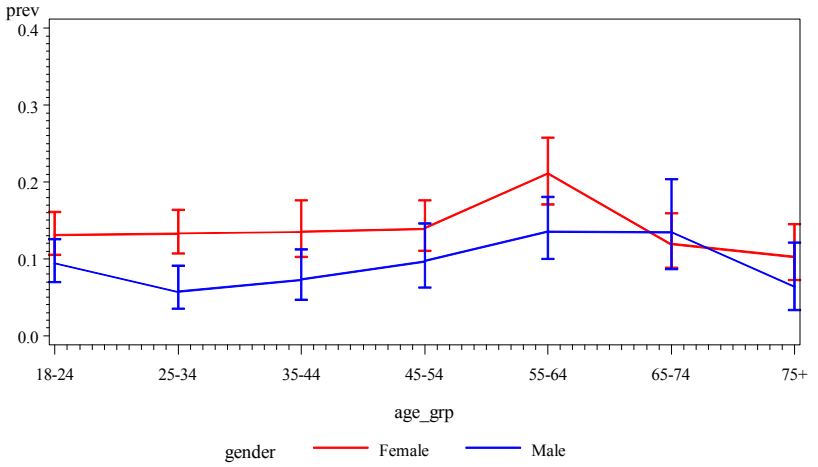
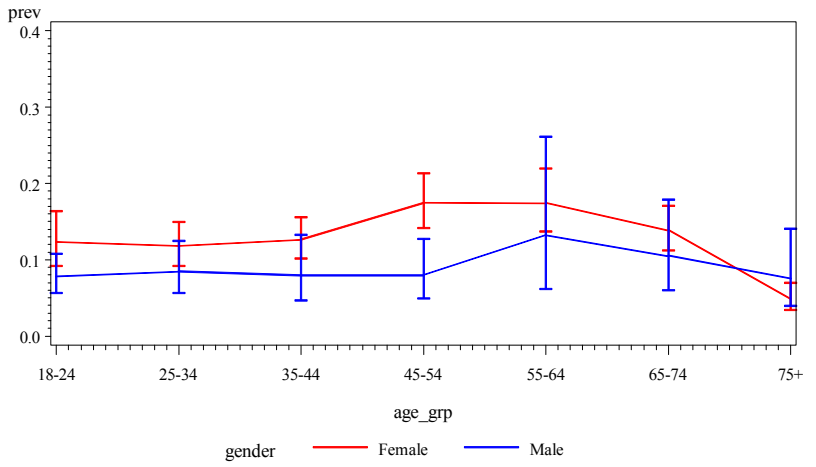


Figure 4. Raw adult asthma 'STILL' prevalence rates and confidence intervals
region=Northeast pov_rat=Below Poverty Level



Appendix 5C, Attachment A, Figure 4. Unsmoothed prevalence and confidence intervals for adults 'STILL' having asthma.

Figure 4. Raw adult asthma 'STILL' prevalence rates and confidence intervals
region=South pov_rat=Above Poverty Level

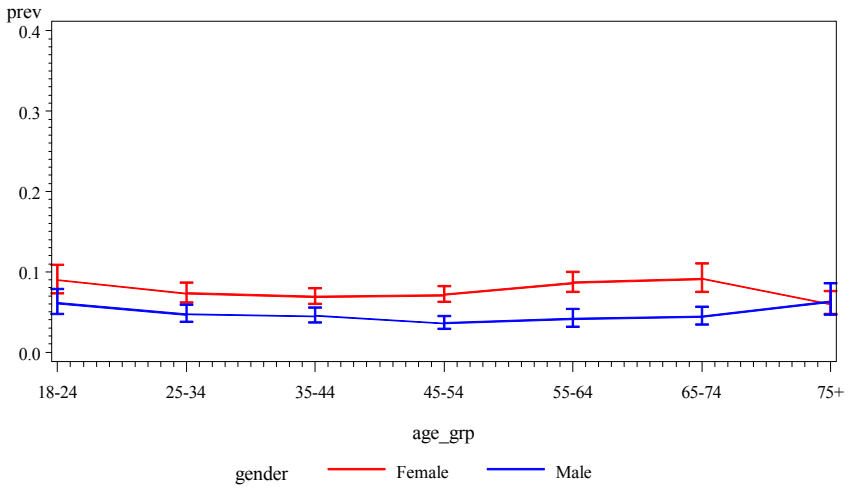


Figure 4. Raw adult asthma 'STILL' prevalence rates and confidence intervals
region=West pov_rat=Above Poverty Level

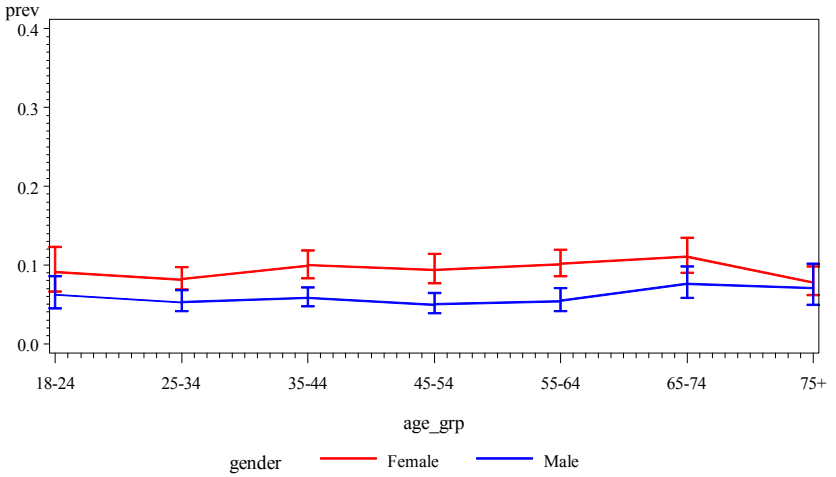


Figure 4. Raw adult asthma 'STILL' prevalence rates and confidence intervals
region=South pov_rat=Below Poverty Level

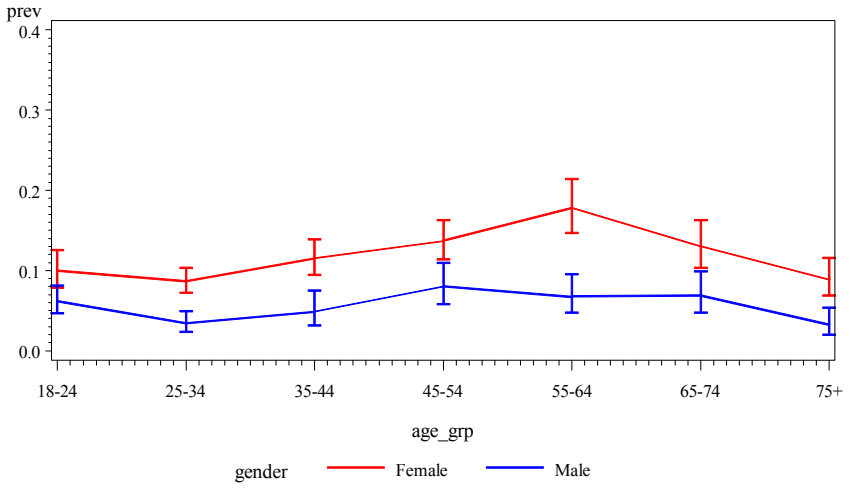
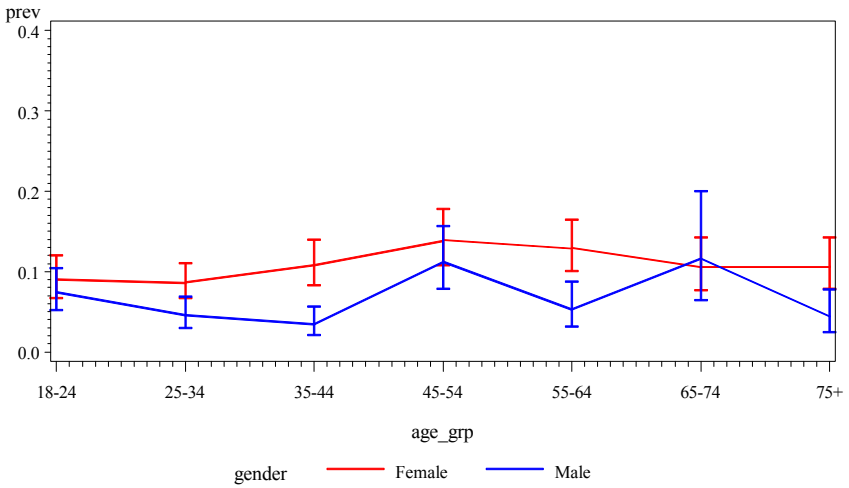


Figure 4. Raw adult asthma 'STILL' prevalence rates and confidence intervals
region=West pov_rat=Below Poverty Level



Appendix 5C, Attachment A, Figure 4, cont. Unsmoothed prevalence and confidence intervals for adults 'STILL' having asthma.

APPENDIX 5C, ATTACHMENT B: LOGISTIC MODEL FIT TABLES AND FIGURES.

Appendix 5C, Attachment B, Table 1. Alternative logistic models for estimating child asthma prevalence using the "EVER" asthma response variable and goodness of fit test results.

Description	Stratification Variable	-2 log likelihood	DF
1. logit(prob) = linear in age	1. none	288740115.1	2
1. logit(prob) = linear in age	2. gender	287062346.4	4
1. logit(prob) = linear in age	3. region	288120804.1	8
1. logit(prob) = linear in age	4. poverty	287385013.1	4
1. logit(prob) = linear in age	5. region, gender	286367652.6	16
1. logit(prob) = linear in age	6. region, poverty	286283543.6	16
1. logit(prob) = linear in age	7. gender, poverty	285696164.7	8
1. logit(prob) = linear in age	8. region, gender, poverty	284477928.1	32
2. logit(prob) = quadratic in age	1. none	286862135.1	3
2. logit(prob) = quadratic in age	2. gender	285098650.6	6
2. logit(prob) = quadratic in age	3. region	286207721.5	12
2. logit(prob) = quadratic in age	4. poverty	285352164	6
2. logit(prob) = quadratic in age	5. region, gender	284330346.1	24
2. logit(prob) = quadratic in age	6. region, poverty	284182547.5	24
2. logit(prob) = quadratic in age	7. gender, poverty	283587631.7	12
2. logit(prob) = quadratic in age	8. region, gender, poverty	282241318.6	48
3. logit(prob) = cubic in age	1. none	286227019.6	4
3. logit(prob) = cubic in age	2. gender	284470413	8
3. logit(prob) = cubic in age	3. region	285546716.1	16
3. logit(prob) = cubic in age	4. poverty	284688169.9	8
3. logit(prob) = cubic in age	5. region, gender	283662673.5	32
3. logit(prob) = cubic in age	6. region, poverty	283404487.5	32
3. logit(prob) = cubic in age	7. gender, poverty	282890785.3	16
3. logit(prob) = cubic in age	8. region, gender, poverty	281407414.3	64
4. logit(prob) = f(age)	1. none	285821686.2	18
4. logit(prob) = f(age)	2. gender	283843266.2	36
4. logit(prob) = f(age)	3. region	284761522.8	72
4. logit(prob) = f(age)	4. poverty	284045849.2	36
4. logit(prob) = f(age)	5. region, gender	282099156.1	144
4. logit(prob) = f(age)	6. region, poverty	281929968.5	144
4. logit(prob) = f(age)	7. gender, poverty	281963915.7	72
4. logit(prob) = f(age)	8. region, gender, poverty	278655423.1	288

Appendix 5C, Attachment B, Table 2. Alternative logistic models for estimating child asthma prevalence using the "STILL" asthma response variable and goodness of fit test results.

Description	Stratification Variable	-2 log likelihood	DF
1. logit(prob) = linear in age	1. none	181557347.7	2
1. logit(prob) = linear in age	2. gender	180677544.6	4
1. logit(prob) = linear in age	3. region	180947344.2	8
1. logit(prob) = linear in age	4. poverty	180502490.5	4
1. logit(prob) = linear in age	5. region, gender	179996184.8	16
1. logit(prob) = linear in age	6. region, poverty	179517528	16
1. logit(prob) = linear in age	7. gender, poverty	179637601.4	8
1. logit(prob) = linear in age	8. region, gender, poverty	178567573.9	32
2. logit(prob) = quadratic in age	1. none	180752073.1	3
2. logit(prob) = quadratic in age	2. gender	179771977.6	6
2. logit(prob) = quadratic in age	3. region	180088080.5	12
2. logit(prob) = quadratic in age	4. poverty	179611530.4	6
2. logit(prob) = quadratic in age	5. region, gender	179004935.6	24
2. logit(prob) = quadratic in age	6. region, poverty	178519078.1	24
2. logit(prob) = quadratic in age	7. gender, poverty	178640744.8	12
2. logit(prob) = quadratic in age	8. region, gender, poverty	177414967.2	48
3. logit(prob) = cubic in age	1. none	180247874.1	4
3. logit(prob) = cubic in age	2. gender	179235170	8
3. logit(prob) = cubic in age	3. region	179583725.1	16
3. logit(prob) = cubic in age	4. poverty	179067549.2	8
3. logit(prob) = cubic in age	5. region, gender	178407915.7	32

Appendix 5C, Attachment B, Table 2. Alternative logistic models for estimating child asthma prevalence using the "STILL" asthma response variable and goodness of fit test results.			
Description	**Stratification Variable**	**-2 log likelihood**	**DF**
3. logit(prob) = cubic in age	6. region, poverty	177897359.3	32
3. logit(prob) = cubic in age	7. gender, poverty	178029240	16
3. logit(prob) = cubic in age	8. region, gender, poverty	176642073.7	64
4. logit(prob) = f(age)	1. none	179972765.3	18
4. logit(prob) = f(age)	2. gender	178918713.8	36
4. logit(prob) = f(age)	3. region	178852704.9	72
4. logit(prob) = f(age)	4. poverty	178599743.4	36
4. logit(prob) = f(age)	5. region, gender	177075815.4	144
4. logit(prob) = f(age)	6. region, poverty	176418872.7	144
4. logit(prob) = f(age)	7. gender, poverty	177422457.4	72
4. logit(prob) = f(age)	8. region, gender, poverty	173888684.9	288

Appendix 5C, Attachment B, Table 3. Alternative logistic models for estimating adult asthma prevalence using the "EVER" asthma response variable and goodness of fit test results.			
Description	**Stratification Variable**	**-2 log likelihood**	**DF**
4. logit(prob) = f(age_grp)	1. none	825494282	7
4. logit(prob) = f(age_grp)	2. gender	821614711.2	14
4. logit(prob) = f(age_grp)	3. region	824598583.4	28
4. logit(prob) = f(age_grp)	4. poverty	823443004.3	14
4. logit(prob) = f(age_grp)	5. region, gender	820520390.7	56
4. logit(prob) = f(age_grp)	6. region, poverty	821958349.1	56
4. logit(prob) = f(age_grp)	7. gender, poverty	819560679.9	28
4. logit(prob) = f(age_grp)	8. region, gender, poverty	817723710	112

Appendix 5C, Attachment B, Table 4. Alternative logistic models for estimating adult asthma prevalence using the "STILL" asthma response variable and goodness of fit test results.			
Description	**Stratification Variable**	**-2 log likelihood**	**DF**
4. logit(prob) = f(age_grp)	1. none	600538044.1	7
4. logit(prob) = f(age_grp)	2. gender	594277797.3	14
4. logit(prob) = f(age_grp)	3. region	599561222.3	28
4. logit(prob) = f(age_grp)	4. poverty	597511872.6	14
4. logit(prob) = f(age_grp)	5. region, gender	593112157.6	56
4. logit(prob) = f(age_grp)	6. region, poverty	596008068.6	56
4. logit(prob) = f(age_grp)	7. gender, poverty	591394271.8	28
4. logit(prob) = f(age_grp)	8. region, gender, poverty	589398969.5	112

Appendix 5C, Attachment B, Table 5. Effect on residual standard error by varying LOESS smoothing parameter while fitting children "EVER" having asthma data set.

Region	Gender	Poverty Ratio	Smoothing Parameter	Residual Standard Error
South	Female	Above Poverty Level	0.5	0.999919
Northeast	Female	Above Poverty Level	0.7	1.00088
South	Male	Above Poverty Level	0.6	1.003839
Midwest	Male	Above Poverty Level	0.9	1.00548
Midwest	Male	Below Poverty Level	0.8	1.010889
South	Female	Above Poverty Level	0.8	1.012178
South	Male	Above Poverty Level	0.5	0.982885
Midwest	Male	Above Poverty Level	1	1.023284
West	Female	Below Poverty Level	0.7	0.973279
South	Female	Above Poverty Level	0.7	0.97298
Midwest	Female	Above Poverty Level	0.7	1.028007
Midwest	Male	Above Poverty Level	0.4	0.970948
Midwest	Male	Above Poverty Level	0.8	0.965591
Midwest	Female	Above Poverty Level	0.6	1.038233
Northeast	Female	Above Poverty Level	0.4	0.961444
South	Male	Above Poverty Level	0.7	1.040867
South	Female	Above Poverty Level	0.6	0.954946
Midwest	Female	Above Poverty Level	0.8	1.045107
West	Male	Above Poverty Level	0.6	1.052418
Northeast	Female	Above Poverty Level	0.6	0.946315
South	Female	Below Poverty Level	0.5	0.945525
Northeast	Female	Above Poverty Level	0.8	1.054556
Midwest	Male	Above Poverty Level	0.7	0.940657
Northeast	Female	Above Poverty Level	0.5	0.940383
Midwest	Male	Below Poverty Level	0.9	1.063971
West	Female	Below Poverty Level	0.8	1.066819
West	Male	Above Poverty Level	0.5	1.067075
South	Female	Above Poverty Level	0.9	1.067923
South	Female	Below Poverty Level	0.4	0.930104
Midwest	Male	Below Poverty Level	0.7	0.929292
Midwest	Female	Above Poverty Level	0.9	1.072631
South	Male	Below Poverty Level	0.6	0.927161
Northeast	Female	Above Poverty Level	0.9	1.074984
Midwest	Male	Above Poverty Level	0.5	0.917969
South	Male	Below Poverty Level	0.7	0.912266
South	Female	Above Poverty Level	0.4	1.089646
Midwest	Male	Above Poverty Level	0.6	0.90827
Midwest	Male	Below Poverty Level	0.4	0.906073
Midwest	Male	Below Poverty Level	1	1.094737
Midwest	Female	Above Poverty Level	0.5	1.096459
South	Male	Above Poverty Level	0.8	1.099725
South	Male	Below Poverty Level	0.5	0.898228
Northeast	Female	Above Poverty Level	1	1.101884
South	Male	Below Poverty Level	1	0.896985
Midwest	Female	Above Poverty Level	1	1.103976
West	Male	Below Poverty Level	0.4	0.894137
South	Male	Below Poverty Level	0.8	0.893364
South	Female	Below Poverty Level	0.6	0.891551
South	Male	Below Poverty Level	0.9	0.890138
West	Female	Below Poverty Level	0.9	1.111538
South	Male	Above Poverty Level	0.4	0.885511
West	Male	Above Poverty Level	0.4	1.115223
South	Female	Below Poverty Level	0.7	0.86999
Northeast	Male	Below Poverty Level	0.6	0.86934
Midwest	Male	Below Poverty Level	0.6	0.86245
Midwest	Male	Below Poverty Level	0.5	0.857982
South	Female	Below Poverty Level	0.8	0.857778
Northeast	Male	Below Poverty Level	0.5	0.857592
West	Female	Below Poverty Level	0.6	0.852664
West	Female	Below Poverty Level	1	1.147894

Appendix 5C, Attachment B, Table 5. Effect on residual standard error by varying LOESS smoothing parameter while fitting children "EVER" having asthma data set.

Region	Gender	Poverty Ratio	Smoothing Parameter	Residual Standard Error
South	Female	Below Poverty Level	1	0.849143
South	Female	Below Poverty Level	0.9	0.847567
Northeast	Male	Below Poverty Level	0.7	0.844668
West	Male	Above Poverty Level	0.7	1.163749
West	Female	Above Poverty Level	0.9	1.163943
West	Female	Above Poverty Level	0.8	1.166005
South	Male	Below Poverty Level	0.4	0.826195
West	Female	Above Poverty Level	0.7	1.174564
West	Female	Above Poverty Level	1	1.178045
South	Male	Above Poverty Level	0.9	1.178803
Northeast	Male	Below Poverty Level	0.8	0.820245
South	Female	Above Poverty Level	1	1.182254
West	Female	Above Poverty Level	0.6	1.187757
Northeast	Male	Below Poverty Level	1	0.811815
West	Female	Below Poverty Level	0.5	0.808706
Northeast	Male	Below Poverty Level	0.9	0.805685
West	Male	Below Poverty Level	1	0.804743
Midwest	Female	Below Poverty Level	1	0.799988
Northeast	Male	Above Poverty Level	1	0.799128
Northeast	Male	Above Poverty Level	0.7	0.798212
Midwest	Female	Above Poverty Level	0.4	1.20612
West	Male	Below Poverty Level	0.5	0.793132
Midwest	Female	Below Poverty Level	0.9	0.788082
Northeast	Male	Above Poverty Level	0.6	0.78547
South	Male	Above Poverty Level	1	1.216423
Northeast	Male	Above Poverty Level	0.8	0.78144
West	Male	Below Poverty Level	0.9	0.780843
Northeast	Male	Above Poverty Level	0.9	0.779772
West	Female	Above Poverty Level	0.5	1.224495
Northeast	Male	Below Poverty Level	0.4	0.769037
West	Male	Below Poverty Level	0.6	0.763027
West	Female	Below Poverty Level	0.4	0.762134
Midwest	Female	Below Poverty Level	0.8	0.758775
West	Male	Below Poverty Level	0.8	0.756848
West	Male	Below Poverty Level	0.7	0.752592
Northeast	Male	Above Poverty Level	0.5	0.729776
West	Male	Above Poverty Level	0.8	1.284153
Northeast	Female	Below Poverty Level	0.8	1.292845
Northeast	Female	Below Poverty Level	0.7	1.296274
Northeast	Female	Below Poverty Level	0.9	1.308752
Northeast	Female	Below Poverty Level	0.6	1.309671
Midwest	Female	Below Poverty Level	0.7	0.688366
Northeast	Female	Below Poverty Level	0.5	1.314991
West	Female	Above Poverty Level	0.4	1.31595
Northeast	Female	Below Poverty Level	1	1.327129
West	Male	Above Poverty Level	0.9	1.35931
Northeast	Female	Below Poverty Level	0.4	1.37577
Northeast	Male	Above Poverty Level	0.4	0.618785
Midwest	Female	Below Poverty Level	0.6	0.607758
West	Male	Above Poverty Level	1	1.395061
Midwest	Female	Below Poverty Level	0.5	0.541466
Midwest	Female	Below Poverty Level	0.4	0.522325

Appendix 5C, Attachment B, Table 6. Effect on residual standard error by varying LOESS smoothing parameter while fitting children "STILL" having asthma data set.

Region	Gender	Poverty Ratio	Smoothing Parameter	Residual Standard Error
South	Female	Above Poverty Level	1	1.000117
Northeast	Male	Above Poverty Level	0.9	1.000909
Northeast	Male	Below Poverty Level	0.7	1.000993
Northeast	Male	Below Poverty Level	0.9	0.997502
Northeast	Male	Below Poverty Level	0.4	0.997275
Midwest	Male	Above Poverty Level	0.7	0.996943
Midwest	Male	Above Poverty Level	0.8	0.996544
Midwest	Female	Above Poverty Level	1	1.003498
Midwest	Male	Above Poverty Level	0.6	0.995815
Northeast	Male	Below Poverty Level	0.8	0.995723
South	Male	Below Poverty Level	0.6	1.007198
Midwest	Female	Above Poverty Level	0.5	0.99235
Northeast	Male	Above Poverty Level	0.5	1.008536
South	Female	Above Poverty Level	0.4	0.99041
Northeast	Male	Below Poverty Level	0.6	1.009859
Northeast	Male	Below Poverty Level	0.5	1.01048
Northeast	Male	Above Poverty Level	0.8	1.011028
Midwest	Male	Above Poverty Level	0.9	1.011038
South	Female	Above Poverty Level	0.6	1.013156
Northeast	Male	Above Poverty Level	1	1.01445
Northeast	Male	Below Poverty Level	1	1.016505
Midwest	Male	Above Poverty Level	0.5	1.01692
Midwest	Female	Above Poverty Level	0.9	0.979917
Midwest	Male	Above Poverty Level	1	1.020707
Northeast	Male	Above Poverty Level	0.7	1.021388
Midwest	Male	Below Poverty Level	0.7	0.977074
South	Female	Above Poverty Level	0.7	0.976479
Northeast	Male	Above Poverty Level	0.6	1.024042
South	Male	Below Poverty Level	1	0.975784
West	Male	Below Poverty Level	0.9	1.025093
South	Male	Below Poverty Level	0.5	1.026184
South	Male	Below Poverty Level	0.7	0.971057
South	Female	Above Poverty Level	0.8	0.965833
South	Female	Above Poverty Level	0.9	0.965238
West	Male	Below Poverty Level	0.8	1.03481
South	Male	Below Poverty Level	0.9	0.964953
West	Female	Below Poverty Level	0.7	1.036384
West	Female	Above Poverty Level	1	1.040924
South	Male	Below Poverty Level	0.8	0.957162
West	Female	Above Poverty Level	0.9	1.044522
Midwest	Male	Below Poverty Level	0.8	1.04601
West	Male	Below Poverty Level	0.7	1.04802
West	Male	Below Poverty Level	1	1.050309
Midwest	Female	Above Poverty Level	0.8	0.946142
Northeast	Female	Above Poverty Level	0.4	0.94543
West	Female	Above Poverty Level	0.8	1.055218
Midwest	Female	Above Poverty Level	0.6	0.938888
West	Male	Above Poverty Level	0.7	1.063545
South	Female	Above Poverty Level	0.5	1.063816
Midwest	Female	Above Poverty Level	0.7	0.931681
West	Male	Below Poverty Level	0.6	1.079146
Midwest	Male	Above Poverty Level	0.4	1.080605
Northeast	Female	Above Poverty Level	0.5	1.083479
West	Female	Above Poverty Level	0.7	1.084472
Midwest	Male	Below Poverty Level	0.9	1.084476
Midwest	Female	Below Poverty Level	0.9	0.914962
Midwest	Female	Below Poverty Level	1	0.913089
South	Male	Below Poverty Level	0.4	1.087093

Appendix 5C, Attachment B, Table 6. Effect on residual standard error by varying LOESS smoothing parameter while fitting children "STILL" having asthma data set.

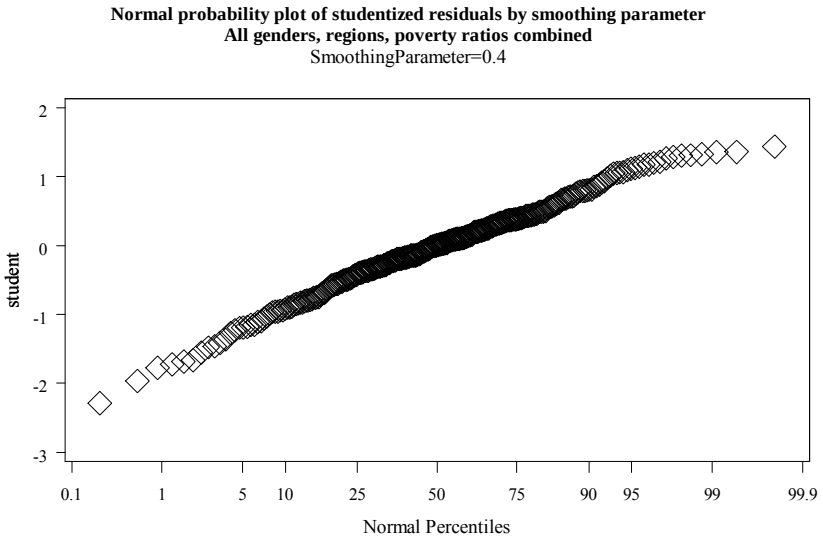
Region	Gender	Poverty Ratio	Smoothing Parameter	Residual Standard Error
Midwest	Female	Below Poverty Level	0.8	0.912722
West	Female	Below Poverty Level	0.6	0.912605
Midwest	Male	Below Poverty Level	0.6	0.907737
Midwest	Male	Below Poverty Level	1	1.103127
Northeast	Female	Above Poverty Level	0.6	1.103286
South	Male	Above Poverty Level	0.4	1.112998
Midwest	Male	Below Poverty Level	0.5	0.878223
West	Female	Above Poverty Level	0.6	1.124127
Midwest	Female	Below Poverty Level	0.7	0.875579
Northeast	Male	Above Poverty Level	0.4	0.874469
West	Female	Below Poverty Level	0.5	0.873529
West	Male	Below Poverty Level	0.5	1.127032
South	Female	Below Poverty Level	0.6	0.87206
Midwest	Male	Below Poverty Level	0.4	0.869726
Midwest	Female	Above Poverty Level	0.4	1.135372
West	Female	Below Poverty Level	0.8	1.136048
South	Female	Below Poverty Level	1	0.863066
Northeast	Female	Above Poverty Level	0.7	1.140006
South	Female	Below Poverty Level	0.5	0.858107
Northeast	Female	Above Poverty Level	0.9	1.147352
Northeast	Female	Above Poverty Level	1	1.148471
West	Male	Below Poverty Level	0.4	1.152015
Northeast	Female	Above Poverty Level	0.8	1.153553
West	Male	Above Poverty Level	0.4	0.845979
South	Female	Below Poverty Level	0.7	0.842335
West	Male	Above Poverty Level	0.6	0.8413
South	Female	Below Poverty Level	0.9	0.841106
West	Female	Above Poverty Level	0.5	1.166931
South	Female	Below Poverty Level	0.8	0.830955
West	Female	Below Poverty Level	0.4	0.826586
West	Female	Below Poverty Level	0.9	1.183444
West	Male	Above Poverty Level	0.5	0.815615
Midwest	Female	Below Poverty Level	0.6	0.802622
West	Female	Below Poverty Level	1	1.20757
Midwest	Female	Below Poverty Level	0.4	0.78769
South	Male	Above Poverty Level	0.5	1.214019
South	Male	Above Poverty Level	0.6	1.216661
South	Female	Below Poverty Level	0.4	0.781555
South	Male	Above Poverty Level	0.7	1.242272
West	Female	Above Poverty Level	0.4	1.252141
West	Male	Above Poverty Level	0.8	1.254244
Midwest	Female	Below Poverty Level	0.5	0.742493
South	Male	Above Poverty Level	0.8	1.294055
Northeast	Female	Below Poverty Level	0.7	1.32003
Northeast	Female	Below Poverty Level	0.6	1.355219
West	Male	Above Poverty Level	0.9	1.356792
South	Male	Above Poverty Level	0.9	1.365737
Northeast	Female	Below Poverty Level	0.8	1.39015
West	Male	Above Poverty Level	1	1.405599
South	Male	Above Poverty Level	1	1.408469
Northeast	Female	Below Poverty Level	0.5	1.431367
Northeast	Female	Below Poverty Level	0.9	1.503674
Northeast	Female	Below Poverty Level	1	1.574778
Northeast	Female	Below Poverty Level	0.4	1.605

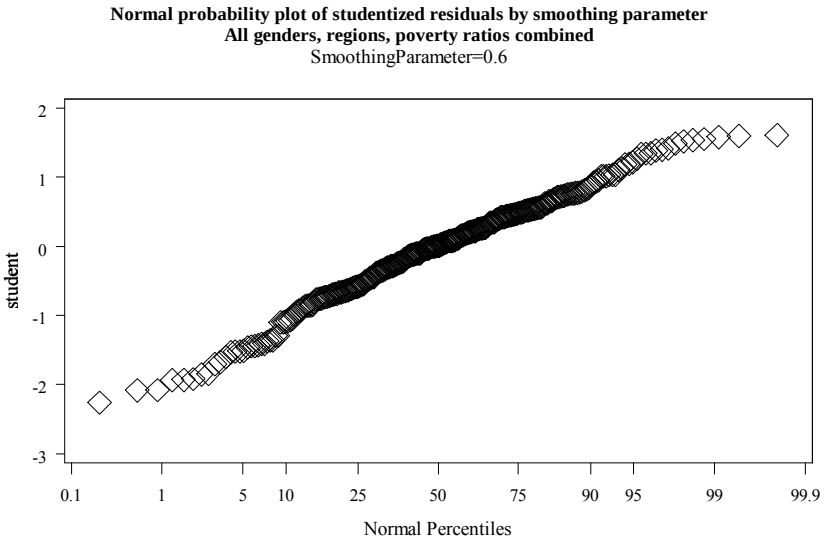
Appendix 5C, Attachment B, Table 7. Effect on residual standard error by varying LOESS smoothing parameter while fitting adults "EVER" having asthma data set.

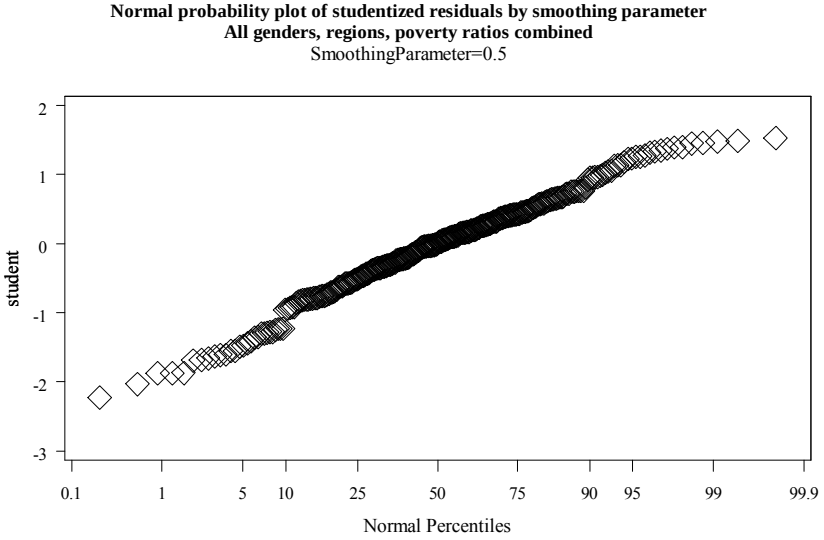
Region	Gender	Poverty Ratio	Smoothing Parameter	Residual Standard Error
Midwest	Female	Above Poverty Level	1	0.983356
South	Female	Below Poverty Level	1	1.040607
West	Female	Below Poverty Level	0.9	1.044712
West	Male	Above Poverty Level	0.8	0.937658
South	Female	Above Poverty Level	1	1.06598
Midwest	Female	Above Poverty Level	0.9	0.911278
West	Male	Below Poverty Level	0.8	1.095844
West	Female	Below Poverty Level	0.8	0.893319
West	Male	Above Poverty Level	0.9	0.886119
Northeast	Female	Above Poverty Level	1	0.875056
West	Male	Above Poverty Level	1	0.858542
Midwest	Female	Above Poverty Level	0.8	0.843191
Northeast	Male	Above Poverty Level	0.8	1.177547
South	Male	Below Poverty Level	1	0.813689
Midwest	Male	Above Poverty Level	0.9	1.190978
Midwest	Male	Below Poverty Level	1	0.785268
South	Female	Above Poverty Level	0.9	0.77381
Northeast	Male	Above Poverty Level	1	1.241548
South	Female	Above Poverty Level	0.8	0.751726
South	Female	Below Poverty Level	0.9	0.747912
South	Female	Below Poverty Level	0.8	0.740577
Northeast	Male	Below Poverty Level	1	0.732859
West	Female	Below Poverty Level	1	1.275049
South	Male	Above Poverty Level	0.9	0.708509
South	Male	Above Poverty Level	1	0.706944
Northeast	Female	Above Poverty Level	0.9	0.699107
Northeast	Male	Above Poverty Level	0.9	1.301543
Northeast	Male	Below Poverty Level	0.9	0.677309
West	Female	Above Poverty Level	1	0.669638
Northeast	Female	Below Poverty Level	1	0.662619
Northeast	Male	Below Poverty Level	0.8	0.646318
South	Male	Below Poverty Level	0.9	0.64328
Midwest	Male	Above Poverty Level	1	1.395026
West	Female	Above Poverty Level	0.8	0.597305
South	Male	Below Poverty Level	0.8	0.58427
West	Female	Above Poverty Level	0.9	0.567466
Northeast	Female	Above Poverty Level	0.8	0.528031
Midwest	Male	Below Poverty Level	0.9	0.49517
West	Male	Below Poverty Level	0.9	1.523816
West	Male	Below Poverty Level	1	1.537805
South	Male	Above Poverty Level	0.8	0.400237
Northeast	Female	Below Poverty Level	0.9	0.394894
Northeast	Female	Below Poverty Level	0.8	0.362058
Midwest	Male	Below Poverty Level	0.8	0.306085
Midwest	Male	Above Poverty Level	0.8	0.169594
Midwest	Female	Below Poverty Level	1	1.910643
Midwest	Female	Below Poverty Level	0.9	1.920542
Midwest	Female	Below Poverty Level	0.8	2.249162

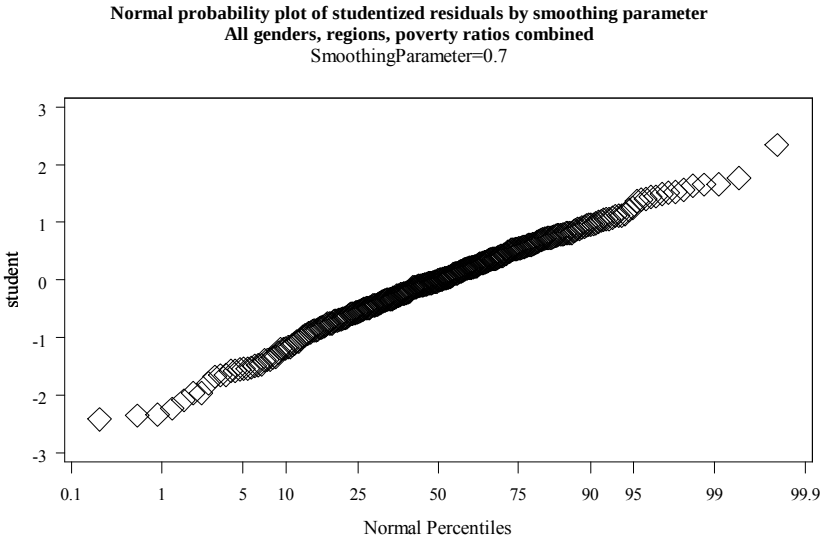
Appendix 5C, Attachment B, Table 8. Effect on residual standard error by varying LOESS smoothing parameter while fitting adults "STILL" having asthma data set.

Region	Gender	Poverty Ratio	Smoothing Parameter	Residual Standard Error
South	Male	Below Poverty Level	0.8	1.015193
West	Female	Above Poverty Level	0.8	1.045714
West	Female	Above Poverty Level	0.9	1.051807
West	Female	Above Poverty Level	1	1.061488
West	Male	Above Poverty Level	1	0.92928
West	Male	Above Poverty Level	0.8	0.925921
West	Male	Above Poverty Level	0.9	0.915895
South	Female	Below Poverty Level	0.9	1.097531
Midwest	Female	Above Poverty Level	1	0.89825
Northeast	Female	Below Poverty Level	1	1.102905
Midwest	Female	Above Poverty Level	0.9	0.876146
South	Female	Below Poverty Level	0.8	1.128781
Midwest	Female	Above Poverty Level	0.8	0.870507
South	Female	Above Poverty Level	1	1.130393
South	Female	Above Poverty Level	0.9	0.835583
West	Female	Below Poverty Level	1	0.825684
South	Male	Below Poverty Level	0.9	1.192655
Midwest	Male	Below Poverty Level	1	0.788217
Northeast	Female	Below Poverty Level	0.9	0.786205
Northeast	Male	Above Poverty Level	1	1.21537
South	Female	Below Poverty Level	1	1.23752
South	Male	Above Poverty Level	0.9	0.748499
South	Male	Above Poverty Level	0.8	0.717121
West	Female	Below Poverty Level	0.9	0.670751
South	Male	Above Poverty Level	1	0.664236
Northeast	Female	Below Poverty Level	0.8	0.65848
Northeast	Female	Above Poverty Level	1	0.653985
Midwest	Male	Above Poverty Level	1	0.650735
Northeast	Female	Above Poverty Level	0.9	0.630298
Northeast	Male	Above Poverty Level	0.9	1.370134
Northeast	Male	Above Poverty Level	0.8	1.375365
Midwest	Male	Below Poverty Level	0.9	0.620174
South	Male	Below Poverty Level	1	1.400273
Northeast	Male	Below Poverty Level	1	0.581032
South	Female	Above Poverty Level	0.8	0.568428
Midwest	Male	Above Poverty Level	0.9	0.508247
Midwest	Male	Below Poverty Level	0.8	0.503315
Northeast	Female	Above Poverty Level	0.8	0.478186
West	Female	Below Poverty Level	0.8	0.464598
Northeast	Male	Below Poverty Level	0.9	0.453855
Northeast	Male	Below Poverty Level	0.8	0.396203
Midwest	Female	Below Poverty Level	1	1.616706
Midwest	Female	Below Poverty Level	0.9	1.636938
Midwest	Male	Above Poverty Level	0.8	0.295923
Midwest	Female	Below Poverty Level	0.8	1.883863
West	Male	Below Poverty Level	0.8	2.16547
West	Male	Below Poverty Level	1	2.200364
West	Male	Below Poverty Level	0.9	2.396381

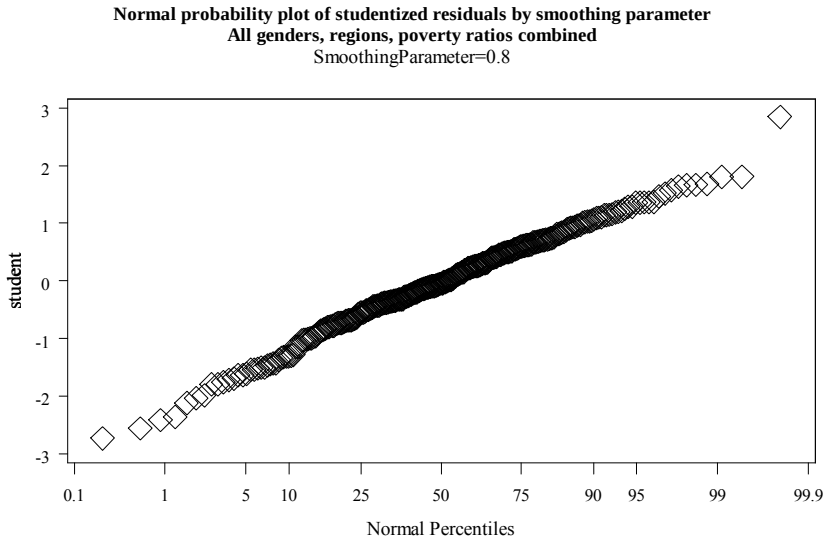


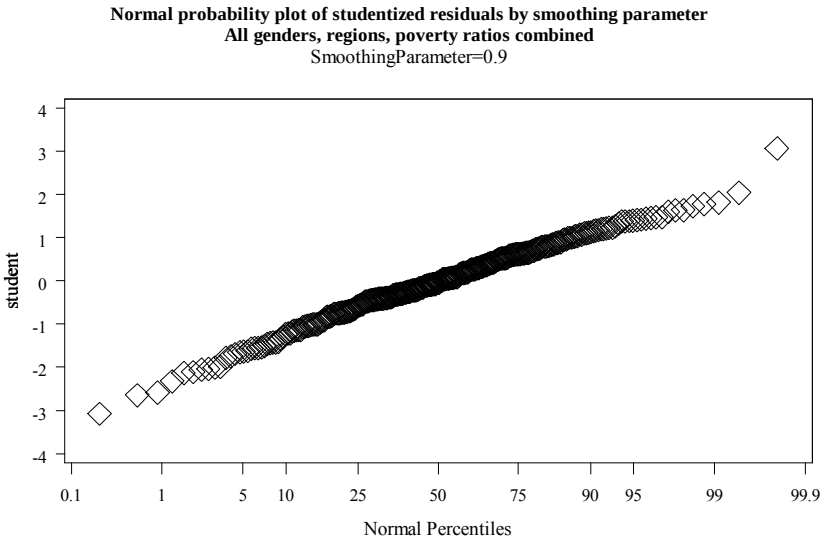


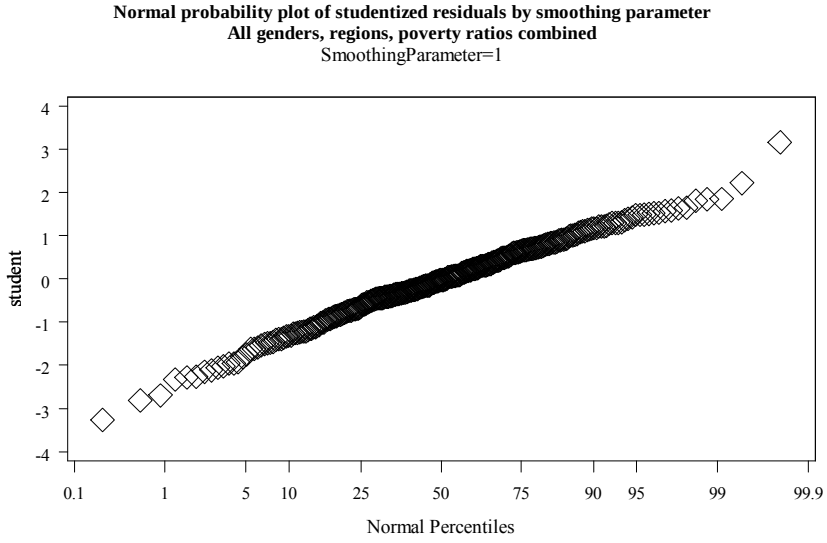




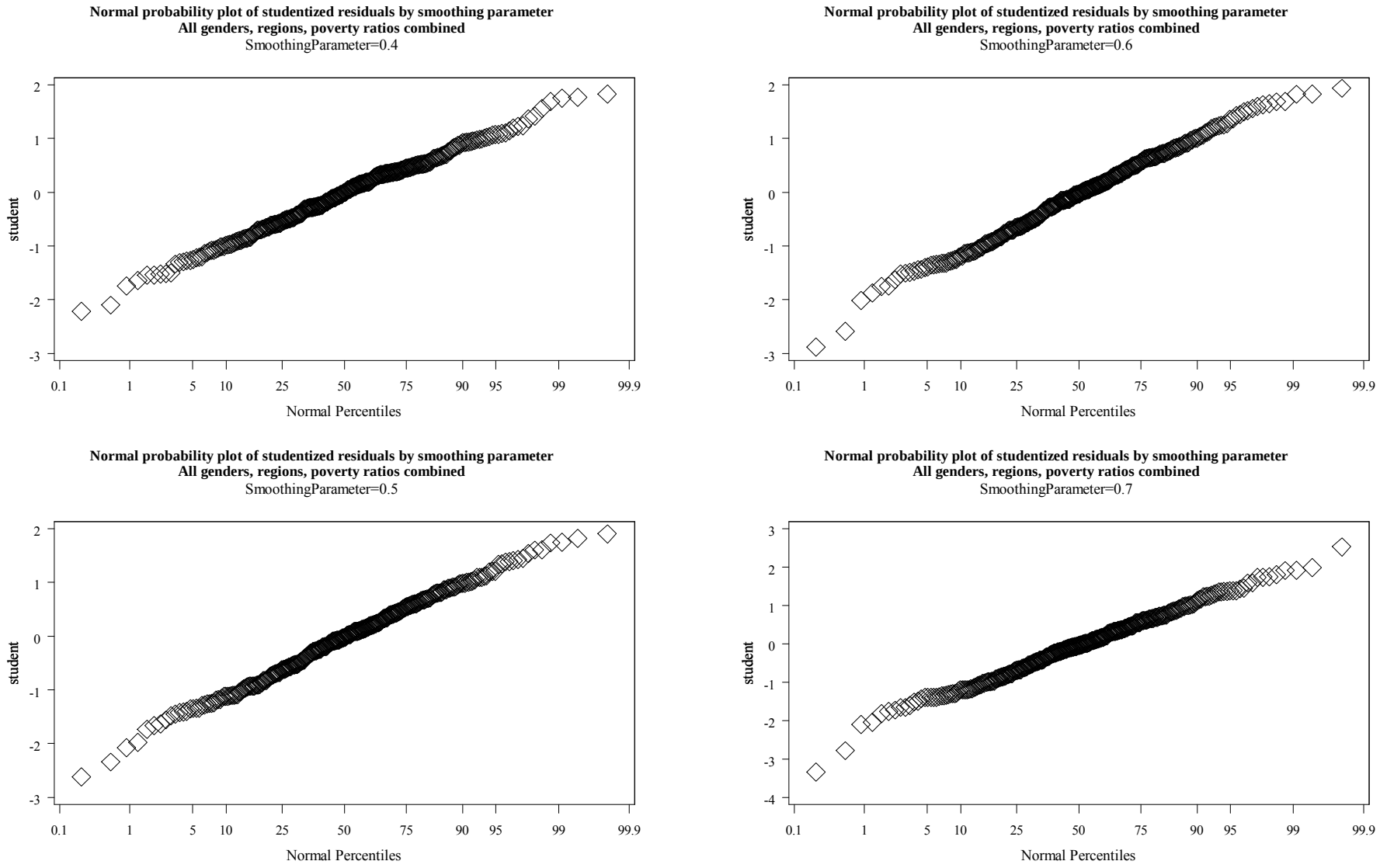
Appendix 5C, Attachment B, Figure 1. Normal probability plots of studentized residuals generated using logistic model and children 'EVER' asthmatic data set.



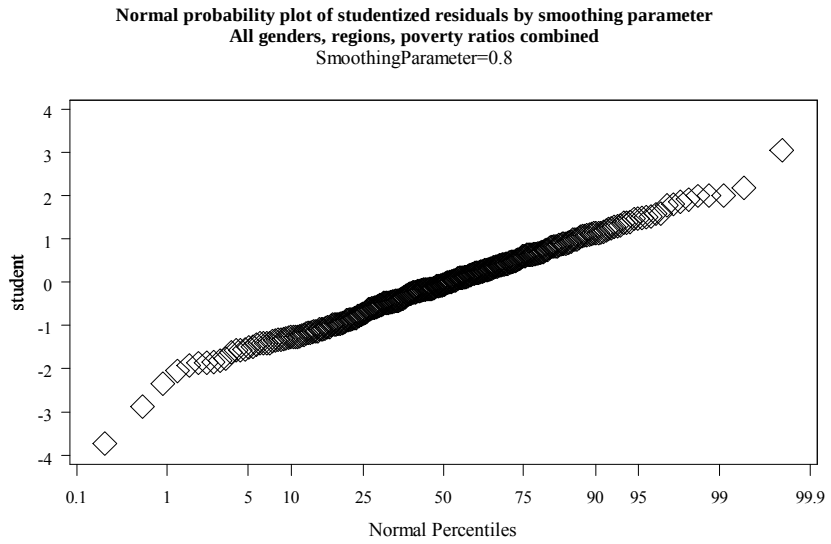


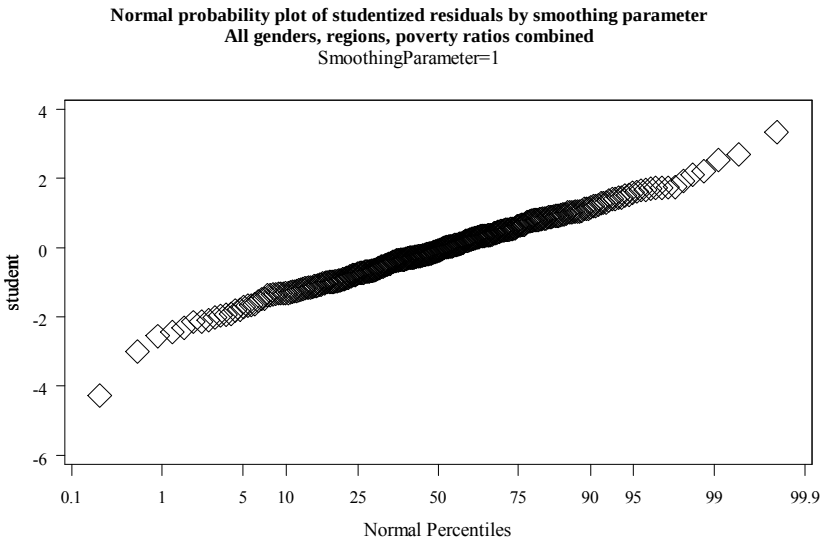


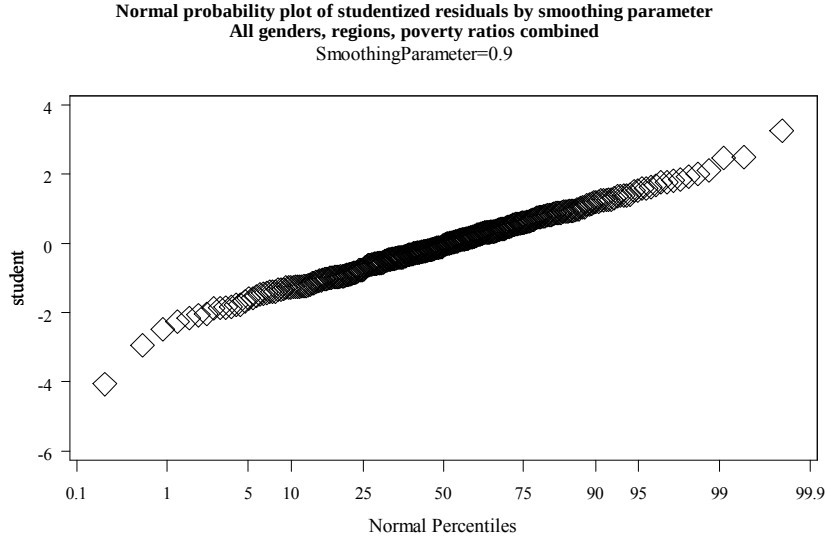
Appendix 5C, Attachment B, Figure 1, cont. Normal probability plots of studentized residuals generated using logistic model and children 'EVER' asthmatic data set.



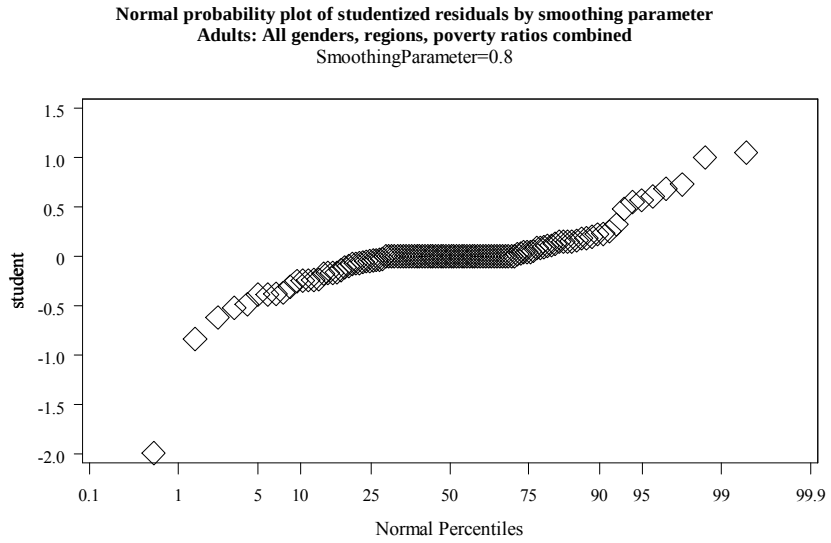
Appendix 5C, Attachment B, Figure 2. Normal probability plots of studentized residuals generated using logistic model and children 'STILL' asthmatic data set.

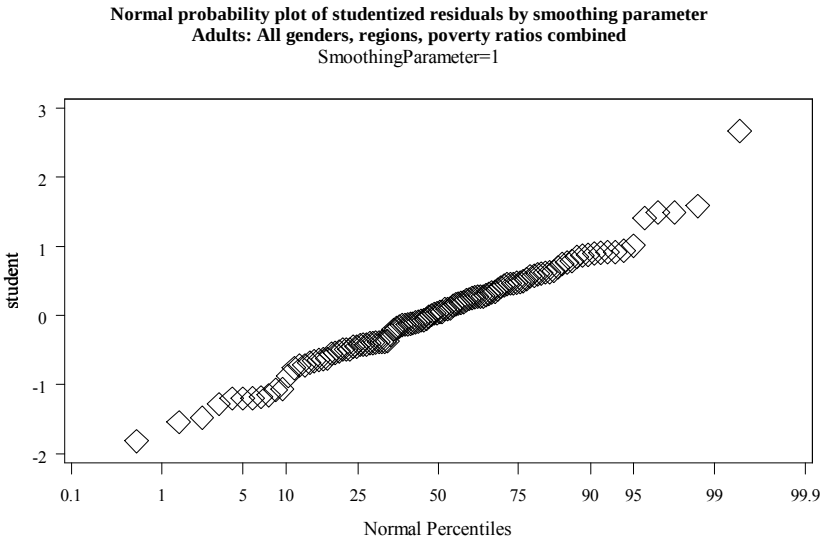


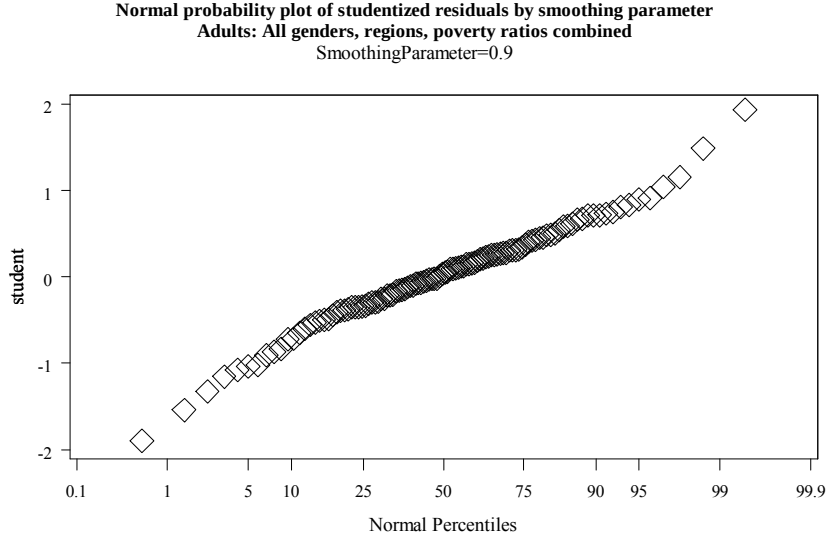




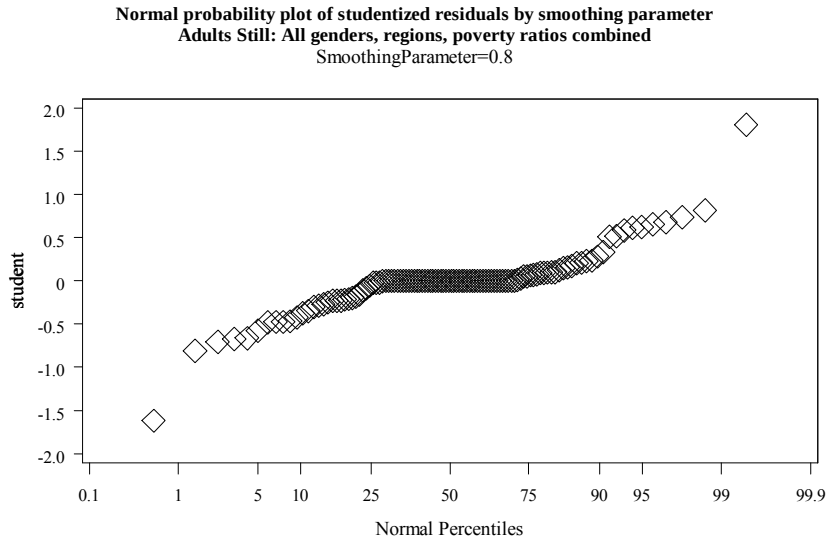
Appendix 5C, Attachment B, Figure 2, cont. Normal probability plots of studentized residuals generated using logistic model and children 'STILL' asthmatic data set.

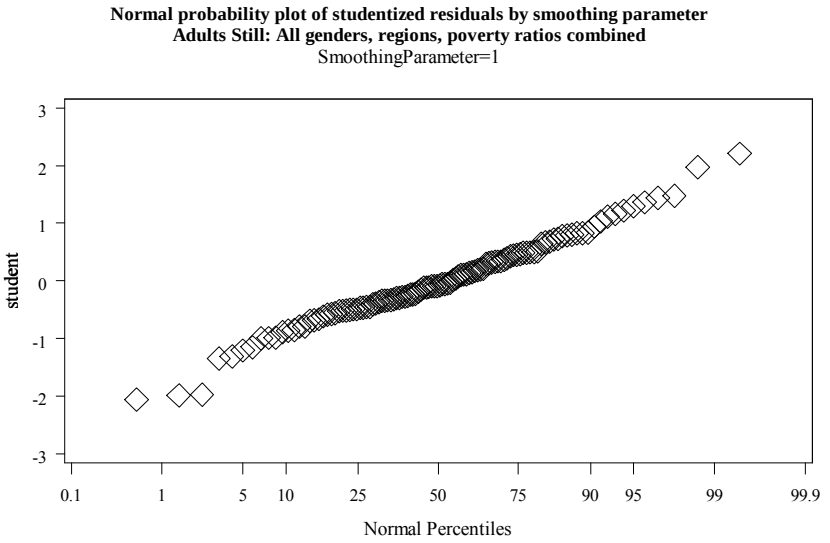


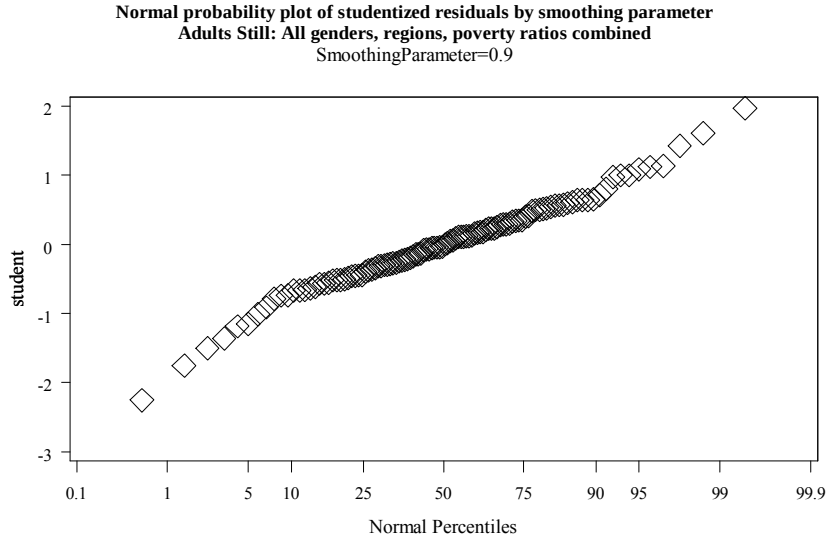




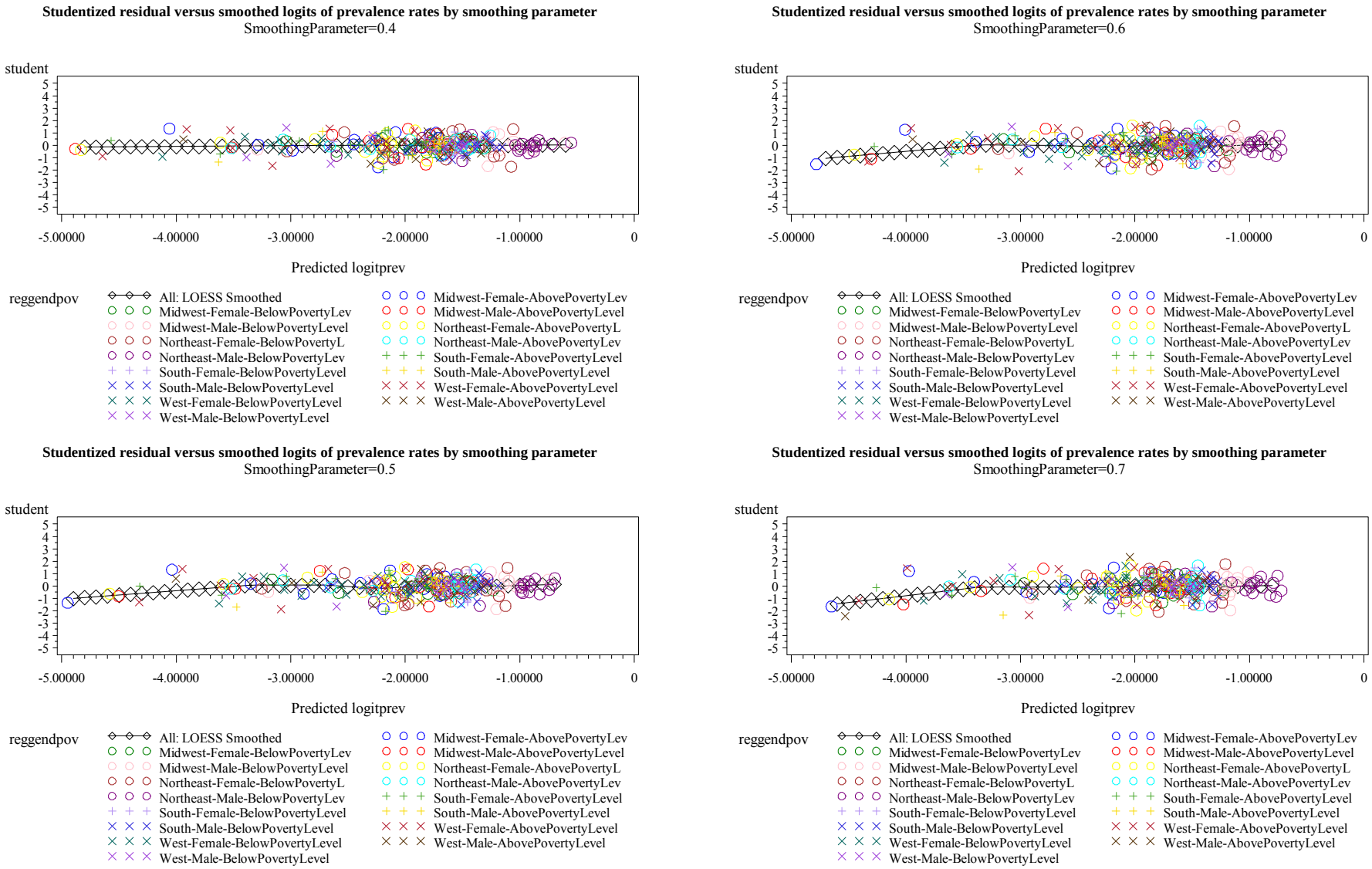
Appendix 5C, Attachment B, Figure 3. Normal probability plots of studentized residuals generated using logistic model and adult 'EVER' asthmatic data set.



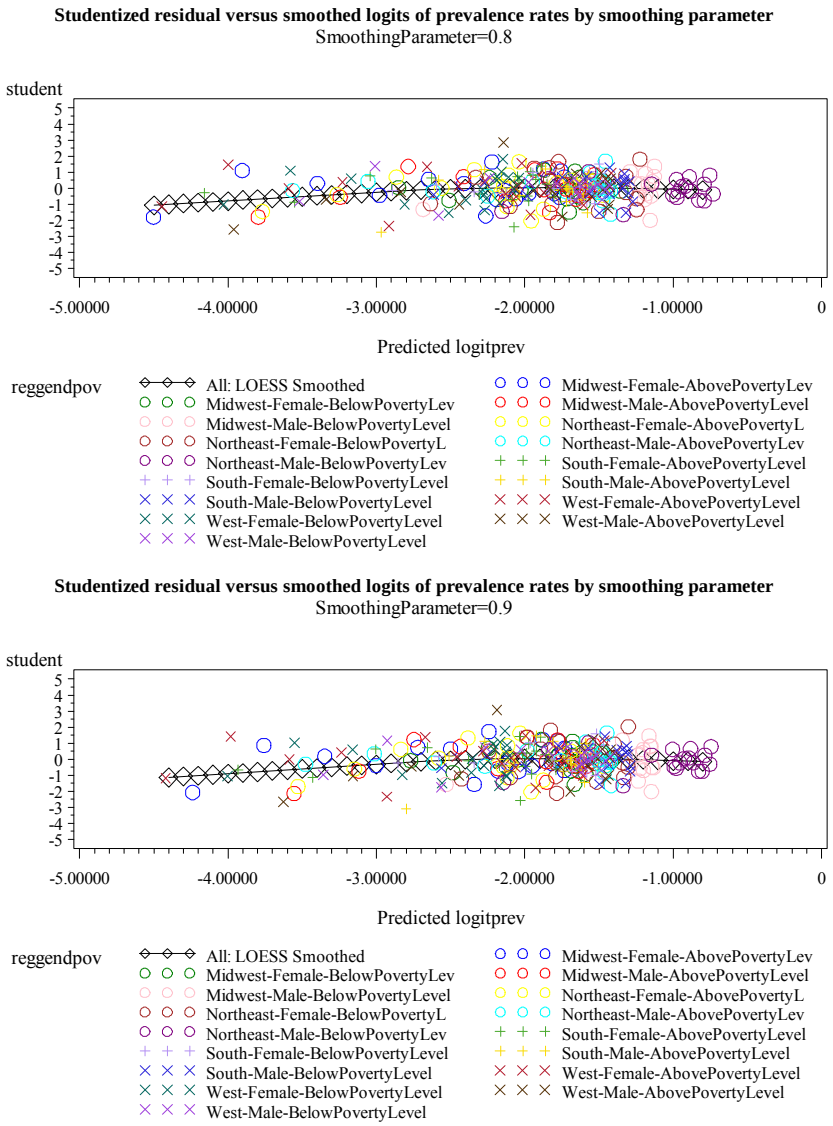


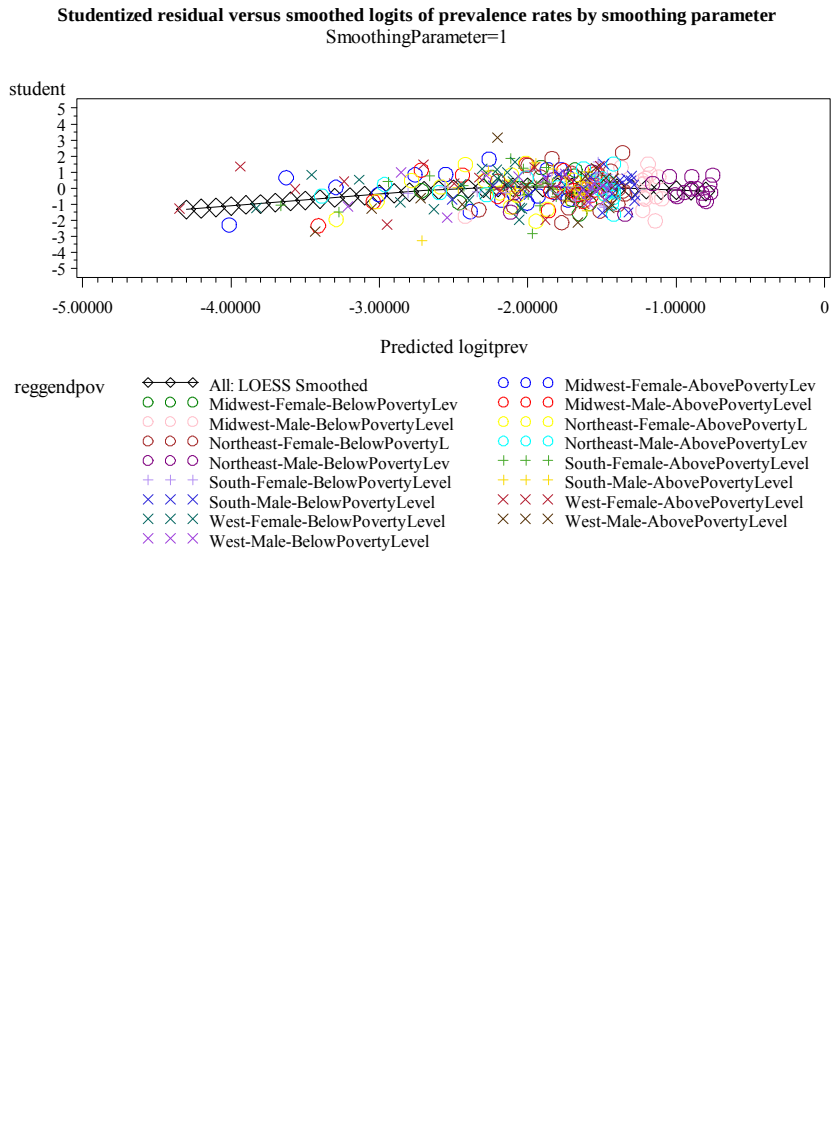


Appendix 5C, Attachment B, Figure 4. Normal probability plots of studentized residuals generated using logistic model and adult 'STILL' asthmatic data set.

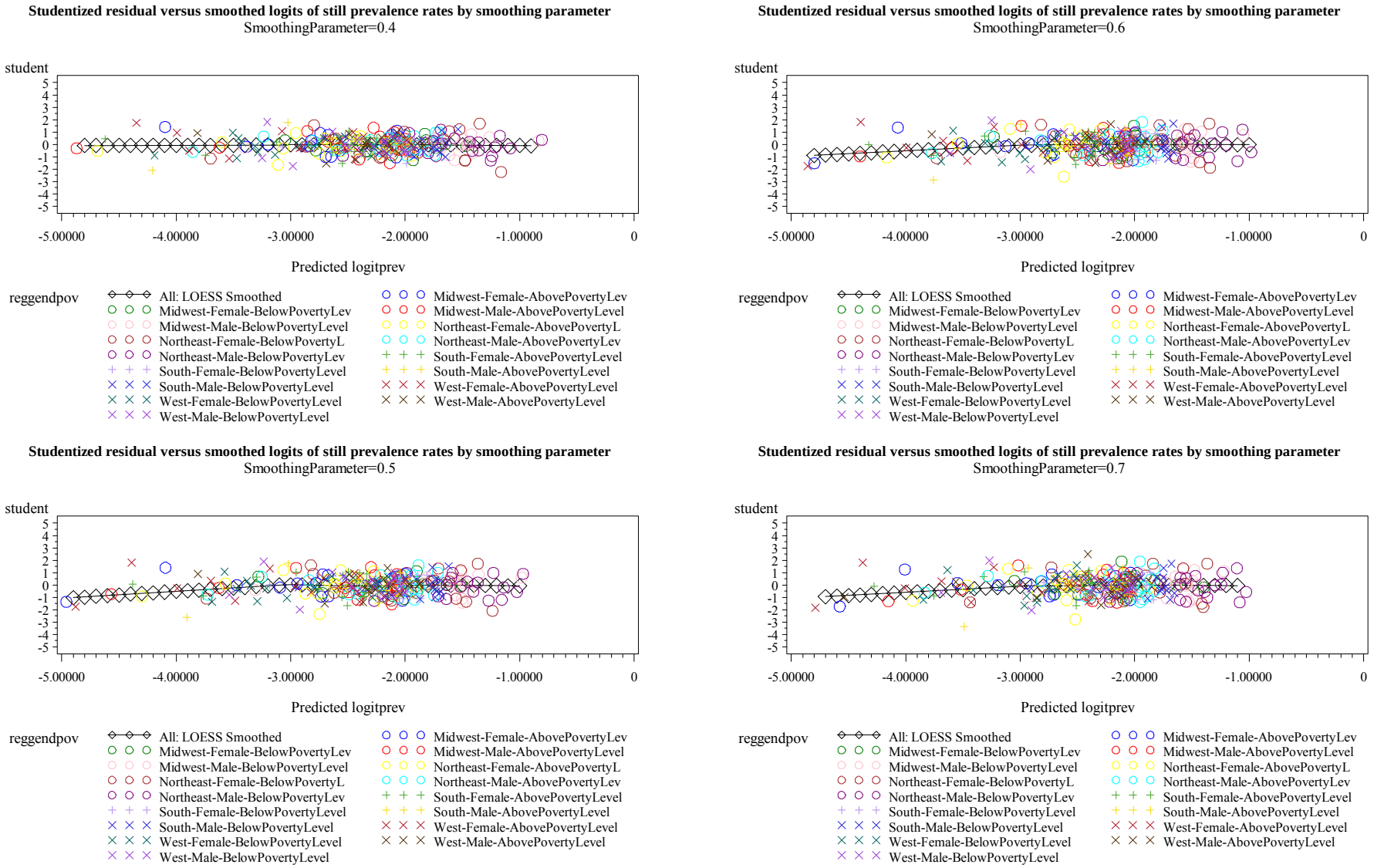


Appendix 5C, Attachment B, Figure 5. Studentized residuals generated using logistic model versus model predicted betas and the child 'EVER' asthmatic data set.

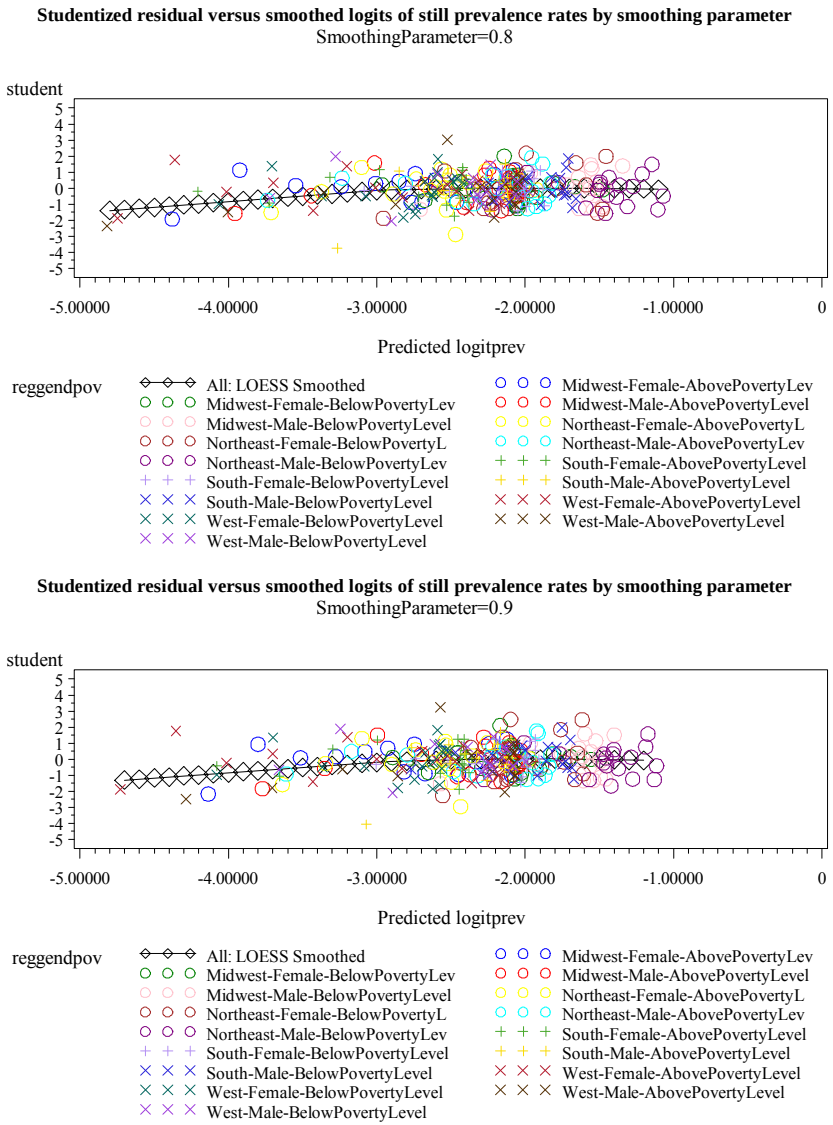


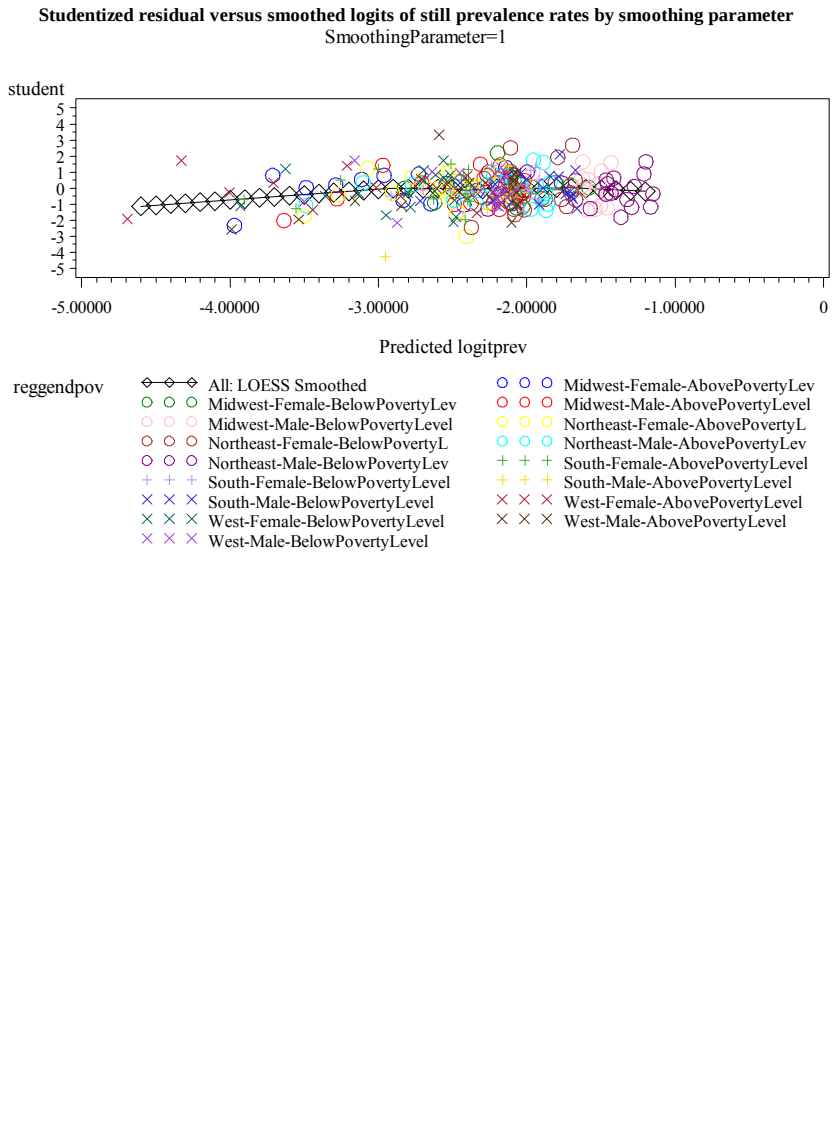


Appendix 5C, Attachment B, Figure 5, cont. Studentized residuals generated using logistic model versus model predicted betas and the child 'EVER' asthmatic data set.

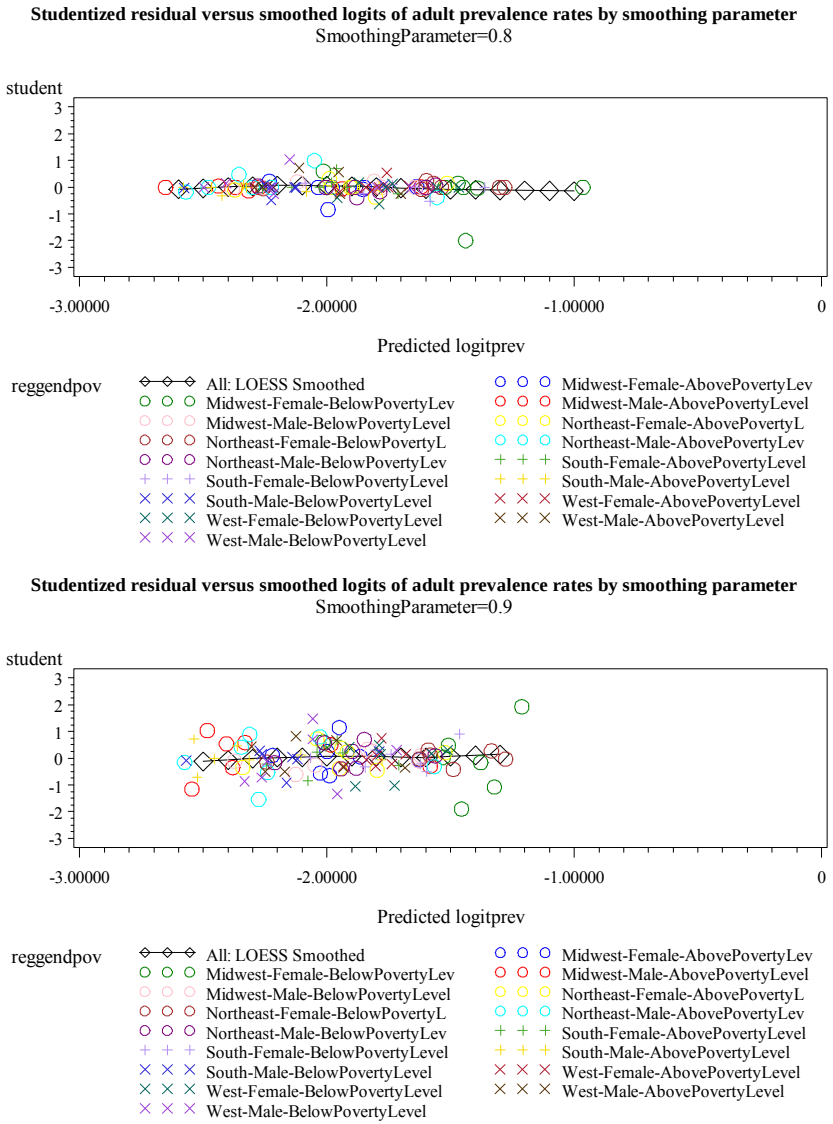


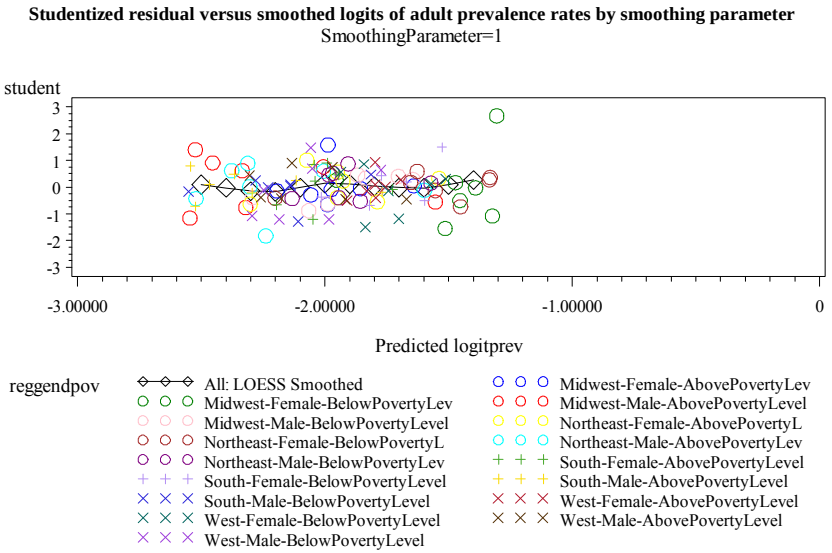
Appendix 5C, Attachment B, Figure 6. Studentized residuals generated using logistic model versus model predicted betas and the child 'STILL' asthmatic data set.



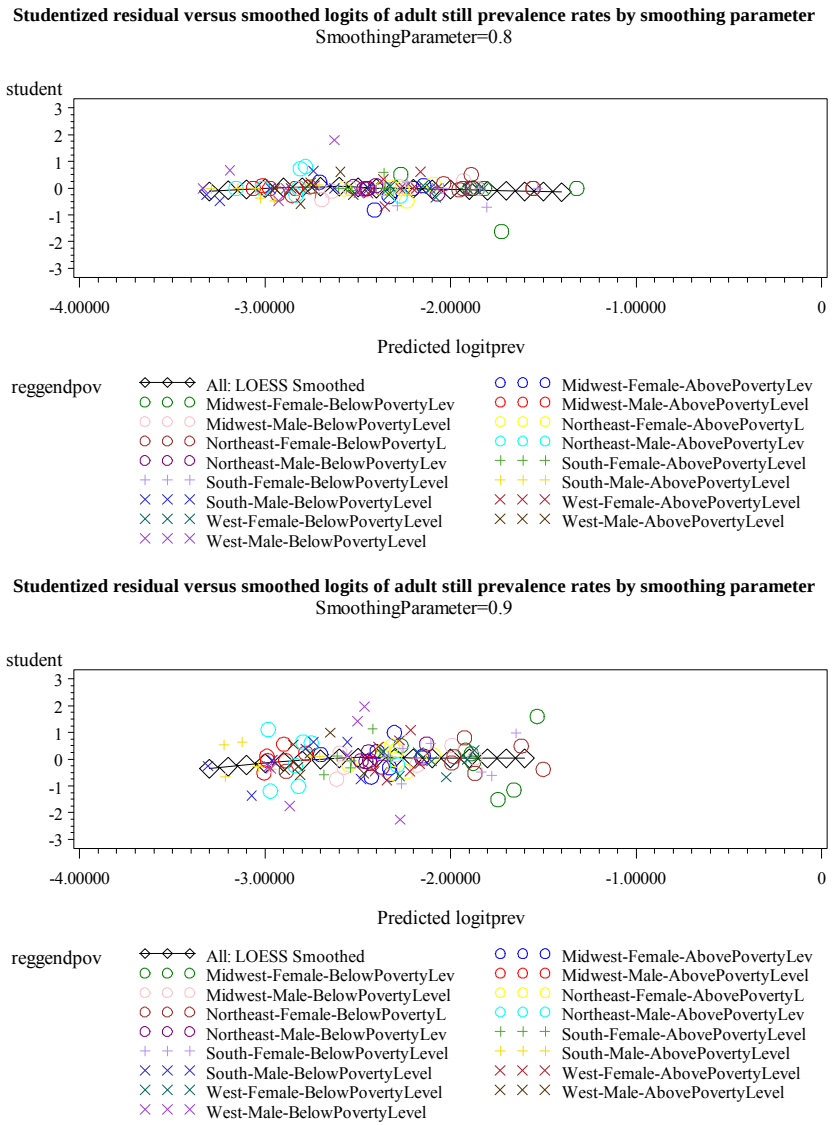


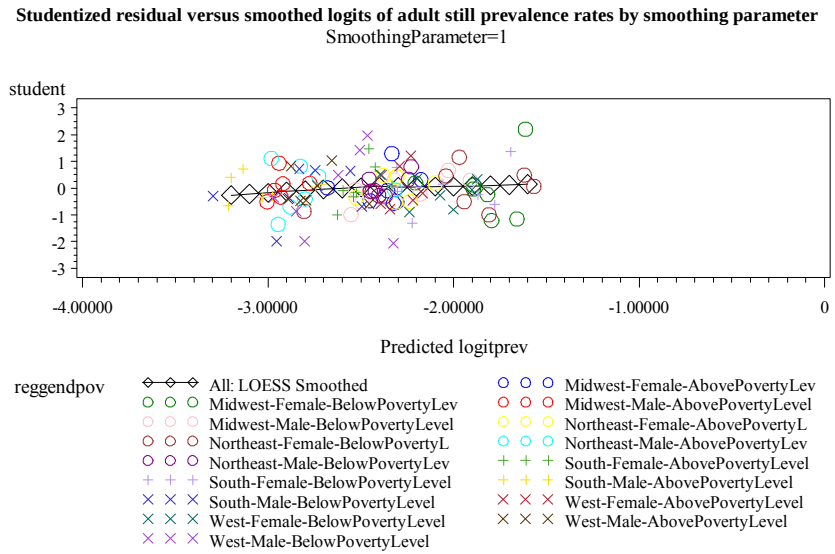
Appendix 5C, Attachment B, Figure 6, cont. Studentized residuals generated using logistic model versus model predicted betas using child 'STILL' asthmatic data set.





Appendix 5C, Attachment B, Figure 7. Studentized residuals generated using logistic model versus model predicted betas using adult 'EVER' asthmatic data set.





Appendix 5C, Attachment B, Figure 8. Studentized residuals generated using logistic model versus model predicted betas using adult 'STILL' asthmatic data set.

APPENDIX 5C, ATTACHMENT C: SMOOTHED ASTHMA PREVALENCE TABLES AND FIGURES.

Appendix 5C, Attachment C, Table 1. Smoothed prevalence for children "EVER" having asthma.

Smoothed	Region	Gender	Poverty Status	Age	Prevalence	SE	LowerCI	UpperCI
Yes	Midwest	Female	Above Poverty Level	0	0.0083	0.0050	0.0022	0.0310
Yes	Midwest	Female	Above Poverty Level	1	0.0179	0.0066	0.0079	0.0397
Yes	Midwest	Female	Above Poverty Level	2	0.0327	0.0076	0.0195	0.0541
Yes	Midwest	Female	Above Poverty Level	3	0.0509	0.0096	0.0336	0.0766
Yes	Midwest	Female	Above Poverty Level	4	0.0671	0.0122	0.0448	0.0993
Yes	Midwest	Female	Above Poverty Level	5	0.0854	0.0134	0.0602	0.1198
Yes	Midwest	Female	Above Poverty Level	6	0.0995	0.0141	0.0725	0.1351
Yes	Midwest	Female	Above Poverty Level	7	0.1041	0.0145	0.0765	0.1403
Yes	Midwest	Female	Above Poverty Level	8	0.1024	0.0132	0.0769	0.1352
Yes	Midwest	Female	Above Poverty Level	9	0.1020	0.0121	0.0784	0.1317
Yes	Midwest	Female	Above Poverty Level	10	0.1055	0.0127	0.0806	0.1369
Yes	Midwest	Female	Above Poverty Level	11	0.1192	0.0137	0.0922	0.1527
Yes	Midwest	Female	Above Poverty Level	12	0.1390	0.0163	0.1070	0.1787
Yes	Midwest	Female	Above Poverty Level	13	0.1529	0.0176	0.1182	0.1956
Yes	Midwest	Female	Above Poverty Level	14	0.1603	0.0176	0.1254	0.2026
Yes	Midwest	Female	Above Poverty Level	15	0.1597	0.0160	0.1277	0.1979
Yes	Midwest	Female	Above Poverty Level	16	0.1517	0.0161	0.1197	0.1903
Yes	Midwest	Female	Above Poverty Level	17	0.1374	0.0229	0.0945	0.1956
Yes	Midwest	Female	Below Poverty Level	0	0.0413	0.0168	0.0167	0.0985
Yes	Midwest	Female	Below Poverty Level	1	0.0706	0.0168	0.0416	0.1174
Yes	Midwest	Female	Below Poverty Level	2	0.1047	0.0173	0.0724	0.1491
Yes	Midwest	Female	Below Poverty Level	3	0.1356	0.0208	0.0962	0.1879
Yes	Midwest	Female	Below Poverty Level	4	0.1553	0.0237	0.1100	0.2146
Yes	Midwest	Female	Below Poverty Level	5	0.1488	0.0229	0.1053	0.2062
Yes	Midwest	Female	Below Poverty Level	6	0.1327	0.0228	0.0902	0.1910
Yes	Midwest	Female	Below Poverty Level	7	0.1341	0.0224	0.0920	0.1912
Yes	Midwest	Female	Below Poverty Level	8	0.1535	0.0239	0.1080	0.2136
Yes	Midwest	Female	Below Poverty Level	9	0.1729	0.0270	0.1215	0.2401
Yes	Midwest	Female	Below Poverty Level	10	0.1861	0.0311	0.1272	0.2640
Yes	Midwest	Female	Below Poverty Level	11	0.1691	0.0300	0.1131	0.2451
Yes	Midwest	Female	Below Poverty Level	12	0.1470	0.0247	0.1006	0.2097
Yes	Midwest	Female	Below Poverty Level	13	0.1439	0.0239	0.0990	0.2045
Yes	Midwest	Female	Below Poverty Level	14	0.1541	0.0244	0.1078	0.2156
Yes	Midwest	Female	Below Poverty Level	15	0.1707	0.0275	0.1186	0.2395
Yes	Midwest	Female	Below Poverty Level	16	0.1962	0.0427	0.1187	0.3065
Yes	Midwest	Female	Below Poverty Level	17	0.2323	0.0813	0.1002	0.4512
Yes	Midwest	Male	Above Poverty Level	0	0.0133	0.0066	0.0045	0.0391
Yes	Midwest	Male	Above Poverty Level	1	0.0313	0.0091	0.0164	0.0588
Yes	Midwest	Male	Above Poverty Level	2	0.0585	0.0102	0.0398	0.0851
Yes	Midwest	Male	Above Poverty Level	3	0.0898	0.0121	0.0666	0.1200
Yes	Midwest	Male	Above Poverty Level	4	0.1111	0.0145	0.0831	0.1471
Yes	Midwest	Male	Above Poverty Level	5	0.1256	0.0149	0.0964	0.1621
Yes	Midwest	Male	Above Poverty Level	6	0.1411	0.0158	0.1100	0.1793
Yes	Midwest	Male	Above Poverty Level	7	0.1496	0.0164	0.1171	0.1892
Yes	Midwest	Male	Above Poverty Level	8	0.1502	0.0161	0.1182	0.1891
Yes	Midwest	Male	Above Poverty Level	9	0.1542	0.0166	0.1211	0.1942
Yes	Midwest	Male	Above Poverty Level	10	0.1627	0.0173	0.1283	0.2041
Yes	Midwest	Male	Above Poverty Level	11	0.1760	0.0181	0.1397	0.2193
Yes	Midwest	Male	Above Poverty Level	12	0.1876	0.0186	0.1501	0.2319
Yes	Midwest	Male	Above Poverty Level	13	0.1847	0.0181	0.1483	0.2277
Yes	Midwest	Male	Above Poverty Level	14	0.1764	0.0170	0.1422	0.2167
Yes	Midwest	Male	Above Poverty Level	15	0.1641	0.0149	0.1341	0.1994
Yes	Midwest	Male	Above Poverty Level	16	0.1487	0.0144	0.1198	0.1833
Yes	Midwest	Male	Above Poverty Level	17	0.1318	0.0201	0.0937	0.1823
Yes	Midwest	Male	Below Poverty Level	0	0.0429	0.0176	0.0173	0.1026
Yes	Midwest	Male	Below Poverty Level	1	0.0908	0.0214	0.0536	0.1498
Yes	Midwest	Male	Below Poverty Level	2	0.1530	0.0235	0.1084	0.2118
Yes	Midwest	Male	Below Poverty Level	3	0.2110	0.0277	0.1566	0.2780

Appendix 5C, Attachment C, Table 1. Smoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	Midwest	Male	Below Poverty Level	4	0.2428	0.0303	0.1828	0.3150
Yes	Midwest	Male	Below Poverty Level	5	0.2458	0.0285	0.1888	0.3133
Yes	Midwest	Male	Below Poverty Level	6	0.2393	0.0270	0.1853	0.3033
Yes	Midwest	Male	Below Poverty Level	7	0.2261	0.0268	0.1729	0.2900
Yes	Midwest	Male	Below Poverty Level	8	0.2225	0.0290	0.1655	0.2924
Yes	Midwest	Male	Below Poverty Level	9	0.2354	0.0311	0.1741	0.3101
Yes	Midwest	Male	Below Poverty Level	10	0.2499	0.0339	0.1831	0.3311
Yes	Midwest	Male	Below Poverty Level	11	0.2553	0.0357	0.1852	0.3409
Yes	Midwest	Male	Below Poverty Level	12	0.2512	0.0377	0.1779	0.3423
Yes	Midwest	Male	Below Poverty Level	13	0.2149	0.0355	0.1473	0.3025
Yes	Midwest	Male	Below Poverty Level	14	0.1941	0.0308	0.1353	0.2703
Yes	Midwest	Male	Below Poverty Level	15	0.2027	0.0292	0.1462	0.2741
Yes	Midwest	Male	Below Poverty Level	16	0.2364	0.0390	0.1617	0.3320
Yes	Midwest	Male	Below Poverty Level	17	0.3045	0.0768	0.1652	0.4921
Yes	Northeast	Female	Above Poverty Level	0	0.0115	0.0066	0.0032	0.0402
Yes	Northeast	Female	Above Poverty Level	1	0.0278	0.0095	0.0131	0.0583
Yes	Northeast	Female	Above Poverty Level	2	0.0533	0.0108	0.0340	0.0827
Yes	Northeast	Female	Above Poverty Level	3	0.0823	0.0127	0.0584	0.1150
Yes	Northeast	Female	Above Poverty Level	4	0.1027	0.0152	0.0737	0.1413
Yes	Northeast	Female	Above Poverty Level	5	0.1066	0.0150	0.0777	0.1445
Yes	Northeast	Female	Above Poverty Level	6	0.1023	0.0143	0.0749	0.1383
Yes	Northeast	Female	Above Poverty Level	7	0.0979	0.0137	0.0715	0.1325
Yes	Northeast	Female	Above Poverty Level	8	0.1010	0.0144	0.0734	0.1375
Yes	Northeast	Female	Above Poverty Level	9	0.1146	0.0166	0.0828	0.1566
Yes	Northeast	Female	Above Poverty Level	10	0.1179	0.0171	0.0852	0.1611
Yes	Northeast	Female	Above Poverty Level	11	0.1170	0.0175	0.0836	0.1615
Yes	Northeast	Female	Above Poverty Level	12	0.1154	0.0164	0.0838	0.1568
Yes	Northeast	Female	Above Poverty Level	13	0.1246	0.0148	0.0955	0.1611
Yes	Northeast	Female	Above Poverty Level	14	0.1405	0.0148	0.1109	0.1765
Yes	Northeast	Female	Above Poverty Level	15	0.1551	0.0152	0.1245	0.1916
Yes	Northeast	Female	Above Poverty Level	16	0.1714	0.0209	0.1302	0.2223
Yes	Northeast	Female	Above Poverty Level	17	0.1883	0.0376	0.1189	0.2851
Yes	Northeast	Female	Below Poverty Level	0	0.0394	0.0211	0.0119	0.1222
Yes	Northeast	Female	Below Poverty Level	1	0.0754	0.0229	0.0383	0.1433
Yes	Northeast	Female	Below Poverty Level	2	0.1188	0.0229	0.0770	0.1789
Yes	Northeast	Female	Below Poverty Level	3	0.1539	0.0265	0.1043	0.2214
Yes	Northeast	Female	Below Poverty Level	4	0.1684	0.0295	0.1131	0.2432
Yes	Northeast	Female	Below Poverty Level	5	0.1503	0.0269	0.1003	0.2193
Yes	Northeast	Female	Below Poverty Level	6	0.1355	0.0245	0.0902	0.1987
Yes	Northeast	Female	Below Poverty Level	7	0.1263	0.0231	0.0836	0.1862
Yes	Northeast	Female	Below Poverty Level	8	0.1322	0.0257	0.0853	0.1993
Yes	Northeast	Female	Below Poverty Level	9	0.1583	0.0301	0.1029	0.2358
Yes	Northeast	Female	Below Poverty Level	10	0.1818	0.0342	0.1183	0.2689
Yes	Northeast	Female	Below Poverty Level	11	0.2030	0.0358	0.1355	0.2926
Yes	Northeast	Female	Below Poverty Level	12	0.2293	0.0359	0.1600	0.3172
Yes	Northeast	Female	Below Poverty Level	13	0.2437	0.0366	0.1726	0.3323
Yes	Northeast	Female	Below Poverty Level	14	0.2368	0.0335	0.1713	0.3179
Yes	Northeast	Female	Below Poverty Level	15	0.2188	0.0286	0.1625	0.2879
Yes	Northeast	Female	Below Poverty Level	16	0.1906	0.0298	0.1335	0.2645
Yes	Northeast	Female	Below Poverty Level	17	0.1572	0.0443	0.0822	0.2796
Yes	Northeast	Male	Above Poverty Level	0	0.0279	0.0107	0.0119	0.0639
Yes	Northeast	Male	Above Poverty Level	1	0.0444	0.0103	0.0265	0.0733
Yes	Northeast	Male	Above Poverty Level	2	0.0668	0.0106	0.0470	0.0940
Yes	Northeast	Male	Above Poverty Level	3	0.0948	0.0134	0.0692	0.1284
Yes	Northeast	Male	Above Poverty Level	4	0.1269	0.0174	0.0933	0.1702
Yes	Northeast	Male	Above Poverty Level	5	0.1665	0.0209	0.1257	0.2173
Yes	Northeast	Male	Above Poverty Level	6	0.1891	0.0207	0.1478	0.2387
Yes	Northeast	Male	Above Poverty Level	7	0.1901	0.0204	0.1494	0.2389
Yes	Northeast	Male	Above Poverty Level	8	0.1858	0.0189	0.1479	0.2307
Yes	Northeast	Male	Above Poverty Level	9	0.1873	0.0189	0.1494	0.2322
Yes	Northeast	Male	Above Poverty Level	10	0.1908	0.0180	0.1545	0.2333
Yes	Northeast	Male	Above Poverty Level	11	0.1926	0.0163	0.1595	0.2307
Yes	Northeast	Male	Above Poverty Level	12	0.1934	0.0168	0.1592	0.2329
Yes	Northeast	Male	Above Poverty Level	13	0.1847	0.0172	0.1499	0.2253

Appendix 5C, Attachment C, Table 1. Smoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	Northeast	Male	Above Poverty Level	14	0.1797	0.0168	0.1458	0.2195
Yes	Northeast	Male	Above Poverty Level	15	0.1781	0.0156	0.1465	0.2149
Yes	Northeast	Male	Above Poverty Level	16	0.1795	0.0162	0.1467	0.2178
Yes	Northeast	Male	Above Poverty Level	17	0.1838	0.0251	0.1350	0.2452
Yes	Northeast	Male	Below Poverty Level	0	0.0946	0.0396	0.0365	0.2240
Yes	Northeast	Male	Below Poverty Level	1	0.1345	0.0296	0.0817	0.2134
Yes	Northeast	Male	Below Poverty Level	2	0.1759	0.0264	0.1251	0.2416
Yes	Northeast	Male	Below Poverty Level	3	0.2132	0.0326	0.1503	0.2932
Yes	Northeast	Male	Below Poverty Level	4	0.2353	0.0361	0.1653	0.3236
Yes	Northeast	Male	Below Poverty Level	5	0.2638	0.0316	0.2004	0.3388
Yes	Northeast	Male	Below Poverty Level	6	0.2909	0.0305	0.2287	0.3621
Yes	Northeast	Male	Below Poverty Level	7	0.3169	0.0339	0.2475	0.3954
Yes	Northeast	Male	Below Poverty Level	8	0.3272	0.0405	0.2451	0.4214
Yes	Northeast	Male	Below Poverty Level	9	0.3238	0.0439	0.2356	0.4265
Yes	Northeast	Male	Below Poverty Level	10	0.3163	0.0429	0.2304	0.4169
Yes	Northeast	Male	Below Poverty Level	11	0.3022	0.0412	0.2199	0.3995
Yes	Northeast	Male	Below Poverty Level	12	0.2846	0.0388	0.2074	0.3769
Yes	Northeast	Male	Below Poverty Level	13	0.2779	0.0367	0.2048	0.3651
Yes	Northeast	Male	Below Poverty Level	14	0.2702	0.0343	0.2016	0.3518
Yes	Northeast	Male	Below Poverty Level	15	0.2698	0.0316	0.2062	0.3445
Yes	Northeast	Male	Below Poverty Level	16	0.2745	0.0349	0.2048	0.3573
Yes	Northeast	Male	Below Poverty Level	17	0.2843	0.0575	0.1760	0.4250
Yes	South	Female	Above Poverty Level	0	0.0137	0.0056	0.0056	0.0334
Yes	South	Female	Above Poverty Level	1	0.0266	0.0064	0.0156	0.0450
Yes	South	Female	Above Poverty Level	2	0.0453	0.0068	0.0325	0.0629
Yes	South	Female	Above Poverty Level	3	0.0687	0.0086	0.0522	0.0901
Yes	South	Female	Above Poverty Level	4	0.0928	0.0112	0.0710	0.1203
Yes	South	Female	Above Poverty Level	5	0.1142	0.0123	0.0900	0.1439
Yes	South	Female	Above Poverty Level	6	0.1298	0.0128	0.1042	0.1605
Yes	South	Female	Above Poverty Level	7	0.1333	0.0123	0.1085	0.1627
Yes	South	Female	Above Poverty Level	8	0.1231	0.0117	0.0996	0.1512
Yes	South	Female	Above Poverty Level	9	0.1095	0.0109	0.0877	0.1359
Yes	South	Female	Above Poverty Level	10	0.1033	0.0102	0.0830	0.1279
Yes	South	Female	Above Poverty Level	11	0.1086	0.0103	0.0881	0.1332
Yes	South	Female	Above Poverty Level	12	0.1212	0.0110	0.0991	0.1475
Yes	South	Female	Above Poverty Level	13	0.1368	0.0113	0.1138	0.1635
Yes	South	Female	Above Poverty Level	14	0.1437	0.0111	0.1210	0.1699
Yes	South	Female	Above Poverty Level	15	0.1448	0.0104	0.1235	0.1690
Yes	South	Female	Above Poverty Level	16	0.1395	0.0113	0.1166	0.1661
Yes	South	Female	Above Poverty Level	17	0.1283	0.0172	0.0952	0.1709
Yes	South	Female	Below Poverty Level	0	0.0496	0.0153	0.0250	0.0962
Yes	South	Female	Below Poverty Level	1	0.0682	0.0123	0.0458	0.1004
Yes	South	Female	Below Poverty Level	2	0.0893	0.0116	0.0670	0.1181
Yes	South	Female	Below Poverty Level	3	0.1111	0.0141	0.0838	0.1459
Yes	South	Female	Below Poverty Level	4	0.1319	0.0171	0.0987	0.1740
Yes	South	Female	Below Poverty Level	5	0.1473	0.0181	0.1120	0.1914
Yes	South	Female	Below Poverty Level	6	0.1553	0.0183	0.1193	0.1997
Yes	South	Female	Below Poverty Level	7	0.1592	0.0183	0.1231	0.2035
Yes	South	Female	Below Poverty Level	8	0.1650	0.0188	0.1277	0.2104
Yes	South	Female	Below Poverty Level	9	0.1766	0.0198	0.1374	0.2241
Yes	South	Female	Below Poverty Level	10	0.1825	0.0216	0.1398	0.2347
Yes	South	Female	Below Poverty Level	11	0.1805	0.0219	0.1373	0.2336
Yes	South	Female	Below Poverty Level	12	0.1837	0.0221	0.1401	0.2371
Yes	South	Female	Below Poverty Level	13	0.1932	0.0218	0.1499	0.2453
Yes	South	Female	Below Poverty Level	14	0.1891	0.0202	0.1487	0.2374
Yes	South	Female	Below Poverty Level	15	0.1760	0.0181	0.1398	0.2192
Yes	South	Female	Below Poverty Level	16	0.1560	0.0195	0.1178	0.2037
Yes	South	Female	Below Poverty Level	17	0.1298	0.0271	0.0810	0.2015
Yes	South	Male	Above Poverty Level	0	0.0335	0.0089	0.0186	0.0596
Yes	South	Male	Above Poverty Level	1	0.0629	0.0093	0.0453	0.0867
Yes	South	Male	Above Poverty Level	2	0.0985	0.0094	0.0797	0.1212
Yes	South	Male	Above Poverty Level	3	0.1306	0.0116	0.1073	0.1581
Yes	South	Male	Above Poverty Level	4	0.1472	0.0133	0.1204	0.1787
Yes	South	Male	Above Poverty Level	5	0.1523	0.0130	0.1259	0.1831

Appendix 5C, Attachment C, Table 1. Smoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	South	Male	Above Poverty Level	6	0.1539	0.0128	0.1278	0.1842
Yes	South	Male	Above Poverty Level	7	0.1485	0.0125	0.1231	0.1782
Yes	South	Male	Above Poverty Level	8	0.1461	0.0123	0.1212	0.1752
Yes	South	Male	Above Poverty Level	9	0.1517	0.0124	0.1265	0.1810
Yes	South	Male	Above Poverty Level	10	0.1639	0.0129	0.1375	0.1943
Yes	South	Male	Above Poverty Level	11	0.1772	0.0134	0.1496	0.2085
Yes	South	Male	Above Poverty Level	12	0.1794	0.0128	0.1530	0.2093
Yes	South	Male	Above Poverty Level	13	0.1752	0.0127	0.1491	0.2049
Yes	South	Male	Above Poverty Level	14	0.1705	0.0120	0.1458	0.1984
Yes	South	Male	Above Poverty Level	15	0.1652	0.0108	0.1428	0.1902
Yes	South	Male	Above Poverty Level	16	0.1600	0.0118	0.1358	0.1876
Yes	South	Male	Above Poverty Level	17	0.1562	0.0190	0.1189	0.2026
Yes	South	Male	Below Poverty Level	0	0.0629	0.0140	0.0383	0.1016
Yes	South	Male	Below Poverty Level	1	0.0922	0.0118	0.0694	0.1215
Yes	South	Male	Below Poverty Level	2	0.1253	0.0123	0.1008	0.1547
Yes	South	Male	Below Poverty Level	3	0.1578	0.0156	0.1265	0.1951
Yes	South	Male	Below Poverty Level	4	0.1852	0.0186	0.1479	0.2294
Yes	South	Male	Below Poverty Level	5	0.1975	0.0190	0.1592	0.2424
Yes	South	Male	Below Poverty Level	6	0.2038	0.0198	0.1639	0.2506
Yes	South	Male	Below Poverty Level	7	0.2087	0.0204	0.1675	0.2570
Yes	South	Male	Below Poverty Level	8	0.2078	0.0203	0.1669	0.2558
Yes	South	Male	Below Poverty Level	9	0.2080	0.0206	0.1664	0.2567
Yes	South	Male	Below Poverty Level	10	0.2122	0.0203	0.1711	0.2601
Yes	South	Male	Below Poverty Level	11	0.2137	0.0202	0.1727	0.2612
Yes	South	Male	Below Poverty Level	12	0.2192	0.0214	0.1759	0.2698
Yes	South	Male	Below Poverty Level	13	0.2199	0.0220	0.1755	0.2718
Yes	South	Male	Below Poverty Level	14	0.2059	0.0209	0.1639	0.2554
Yes	South	Male	Below Poverty Level	15	0.1946	0.0186	0.1571	0.2385
Yes	South	Male	Below Poverty Level	16	0.1827	0.0177	0.1471	0.2246
Yes	South	Male	Below Poverty Level	17	0.1709	0.0246	0.1235	0.2317
Yes	West	Female	Above Poverty Level	0	0.0131	0.0067	0.0042	0.0400
Yes	West	Female	Above Poverty Level	1	0.0188	0.0057	0.0096	0.0365
Yes	West	Female	Above Poverty Level	2	0.0264	0.0053	0.0171	0.0407
Yes	West	Female	Above Poverty Level	3	0.0361	0.0064	0.0245	0.0531
Yes	West	Female	Above Poverty Level	4	0.0469	0.0083	0.0317	0.0689
Yes	West	Female	Above Poverty Level	5	0.0647	0.0105	0.0451	0.0919
Yes	West	Female	Above Poverty Level	6	0.0857	0.0130	0.0611	0.1189
Yes	West	Female	Above Poverty Level	7	0.1008	0.0144	0.0733	0.1372
Yes	West	Female	Above Poverty Level	8	0.1032	0.0151	0.0746	0.1412
Yes	West	Female	Above Poverty Level	9	0.1063	0.0144	0.0786	0.1424
Yes	West	Female	Above Poverty Level	10	0.1166	0.0140	0.0893	0.1509
Yes	West	Female	Above Poverty Level	11	0.1181	0.0129	0.0927	0.1494
Yes	West	Female	Above Poverty Level	12	0.1196	0.0131	0.0938	0.1513
Yes	West	Female	Above Poverty Level	13	0.1202	0.0130	0.0945	0.1519
Yes	West	Female	Above Poverty Level	14	0.1241	0.0127	0.0987	0.1548
Yes	West	Female	Above Poverty Level	15	0.1389	0.0125	0.1136	0.1687
Yes	West	Female	Above Poverty Level	16	0.1665	0.0152	0.1358	0.2025
Yes	West	Female	Above Poverty Level	17	0.2118	0.0305	0.1525	0.2864
Yes	West	Female	Below Poverty Level	0	0.0250	0.0138	0.0073	0.0819
Yes	West	Female	Below Poverty Level	1	0.0309	0.0099	0.0152	0.0618
Yes	West	Female	Below Poverty Level	2	0.0387	0.0082	0.0243	0.0612
Yes	West	Female	Below Poverty Level	3	0.0488	0.0099	0.0312	0.0757
Yes	West	Female	Below Poverty Level	4	0.0602	0.0129	0.0374	0.0955
Yes	West	Female	Below Poverty Level	5	0.0843	0.0169	0.0538	0.1296
Yes	West	Female	Below Poverty Level	6	0.1143	0.0197	0.0776	0.1652
Yes	West	Female	Below Poverty Level	7	0.1295	0.0191	0.0930	0.1775
Yes	West	Female	Below Poverty Level	8	0.1195	0.0175	0.0861	0.1636
Yes	West	Female	Below Poverty Level	9	0.0950	0.0151	0.0666	0.1338
Yes	West	Female	Below Poverty Level	10	0.0786	0.0139	0.0530	0.1150
Yes	West	Female	Below Poverty Level	11	0.0812	0.0150	0.0537	0.1209
Yes	West	Female	Below Poverty Level	12	0.0979	0.0179	0.0651	0.1447
Yes	West	Female	Below Poverty Level	13	0.1278	0.0221	0.0866	0.1848
Yes	West	Female	Below Poverty Level	14	0.1324	0.0211	0.0925	0.1859
Yes	West	Female	Below Poverty Level	15	0.1188	0.0176	0.0853	0.1631

Appendix 5C, Attachment C, Table 1. Smoothed prevalence for children "EVER" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	West	Female	Below Poverty Level	16	0.0917	0.0164	0.0615	0.1347
Yes	West	Female	Below Poverty Level	17	0.0600	0.0186	0.0300	0.1163
Yes	West	Male	Above Poverty Level	0	0.0057	0.0035	0.0014	0.0229
Yes	West	Male	Above Poverty Level	1	0.0191	0.0067	0.0084	0.0428
Yes	West	Male	Above Poverty Level	2	0.0479	0.0092	0.0306	0.0743
Yes	West	Male	Above Poverty Level	3	0.0903	0.0114	0.0673	0.1201
Yes	West	Male	Above Poverty Level	4	0.1300	0.0149	0.0993	0.1685
Yes	West	Male	Above Poverty Level	5	0.1437	0.0158	0.1110	0.1842
Yes	West	Male	Above Poverty Level	6	0.1374	0.0157	0.1050	0.1779
Yes	West	Male	Above Poverty Level	7	0.1290	0.0148	0.0985	0.1671
Yes	West	Male	Above Poverty Level	8	0.1365	0.0148	0.1058	0.1743
Yes	West	Male	Above Poverty Level	9	0.1560	0.0154	0.1236	0.1950
Yes	West	Male	Above Poverty Level	10	0.1794	0.0160	0.1454	0.2193
Yes	West	Male	Above Poverty Level	11	0.1980	0.0175	0.1608	0.2413
Yes	West	Male	Above Poverty Level	12	0.1948	0.0180	0.1566	0.2396
Yes	West	Male	Above Poverty Level	13	0.1818	0.0175	0.1449	0.2256
Yes	West	Male	Above Poverty Level	14	0.1771	0.0164	0.1423	0.2183
Yes	West	Male	Above Poverty Level	15	0.1801	0.0148	0.1484	0.2167
Yes	West	Male	Above Poverty Level	16	0.1897	0.0149	0.1577	0.2264
Yes	West	Male	Above Poverty Level	17	0.2081	0.0248	0.1567	0.2709
Yes	West	Male	Below Poverty Level	0	0.0258	0.0126	0.0087	0.0738
Yes	West	Male	Below Poverty Level	1	0.0442	0.0124	0.0237	0.0812
Yes	West	Male	Below Poverty Level	2	0.0700	0.0119	0.0479	0.1013
Yes	West	Male	Below Poverty Level	3	0.1005	0.0144	0.0729	0.1370
Yes	West	Male	Below Poverty Level	4	0.1323	0.0190	0.0959	0.1799
Yes	West	Male	Below Poverty Level	5	0.1609	0.0218	0.1186	0.2147
Yes	West	Male	Below Poverty Level	6	0.1663	0.0213	0.1247	0.2184
Yes	West	Male	Below Poverty Level	7	0.1582	0.0205	0.1182	0.2086
Yes	West	Male	Below Poverty Level	8	0.1536	0.0204	0.1140	0.2040
Yes	West	Male	Below Poverty Level	9	0.1543	0.0214	0.1128	0.2075
Yes	West	Male	Below Poverty Level	10	0.1630	0.0240	0.1168	0.2228
Yes	West	Male	Below Poverty Level	11	0.1746	0.0270	0.1230	0.2420
Yes	West	Male	Below Poverty Level	12	0.1828	0.0270	0.1306	0.2498
Yes	West	Male	Below Poverty Level	13	0.1809	0.0276	0.1280	0.2495
Yes	West	Male	Below Poverty Level	14	0.1800	0.0259	0.1298	0.2440
Yes	West	Male	Below Poverty Level	15	0.1828	0.0233	0.1371	0.2396
Yes	West	Male	Below Poverty Level	16	0.1881	0.0242	0.1405	0.2471
Yes	West	Male	Below Poverty Level	17	0.1964	0.0396	0.1234	0.2978

Appendix 5C, Attachment C, Table 2. Smoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	Midwest	Female	Above Poverty Level	0	0.0082	0.0051	0.0021	0.0319
Yes	Midwest	Female	Above Poverty Level	1	0.0168	0.0064	0.0073	0.0382
Yes	Midwest	Female	Above Poverty Level	2	0.0289	0.0070	0.0169	0.0490
Yes	Midwest	Female	Above Poverty Level	3	0.0420	0.0086	0.0267	0.0655
Yes	Midwest	Female	Above Poverty Level	4	0.0509	0.0103	0.0326	0.0788
Yes	Midwest	Female	Above Poverty Level	5	0.0573	0.0108	0.0378	0.0859
Yes	Midwest	Female	Above Poverty Level	6	0.0611	0.0109	0.0412	0.0897
Yes	Midwest	Female	Above Poverty Level	7	0.0624	0.0107	0.0427	0.0902
Yes	Midwest	Female	Above Poverty Level	8	0.0629	0.0100	0.0443	0.0886
Yes	Midwest	Female	Above Poverty Level	9	0.0663	0.0096	0.0481	0.0907
Yes	Midwest	Female	Above Poverty Level	10	0.0737	0.0108	0.0533	0.1012
Yes	Midwest	Female	Above Poverty Level	11	0.0889	0.0126	0.0649	0.1206
Yes	Midwest	Female	Above Poverty Level	12	0.1056	0.0151	0.0768	0.1435
Yes	Midwest	Female	Above Poverty Level	13	0.1157	0.0163	0.0845	0.1565
Yes	Midwest	Female	Above Poverty Level	14	0.1191	0.0160	0.0882	0.1588
Yes	Midwest	Female	Above Poverty Level	15	0.1177	0.0144	0.0896	0.1530
Yes	Midwest	Female	Above Poverty Level	16	0.1107	0.0143	0.0831	0.1461
Yes	Midwest	Female	Above Poverty Level	17	0.0999	0.0205	0.0632	0.1544
Yes	Midwest	Female	Below Poverty Level	0	0.0381	0.0164	0.0146	0.0956
Yes	Midwest	Female	Below Poverty Level	1	0.0620	0.0160	0.0349	0.1076
Yes	Midwest	Female	Below Poverty Level	2	0.0875	0.0160	0.0581	0.1295
Yes	Midwest	Female	Below Poverty Level	3	0.1079	0.0183	0.0738	0.1550
Yes	Midwest	Female	Below Poverty Level	4	0.1187	0.0202	0.0811	0.1704
Yes	Midwest	Female	Below Poverty Level	5	0.1117	0.0194	0.0758	0.1616
Yes	Midwest	Female	Below Poverty Level	6	0.0940	0.0188	0.0602	0.1439
Yes	Midwest	Female	Below Poverty Level	7	0.0974	0.0187	0.0634	0.1469
Yes	Midwest	Female	Below Poverty Level	8	0.1144	0.0205	0.0765	0.1676
Yes	Midwest	Female	Below Poverty Level	9	0.1237	0.0220	0.0830	0.1805
Yes	Midwest	Female	Below Poverty Level	10	0.1196	0.0237	0.0766	0.1821
Yes	Midwest	Female	Below Poverty Level	11	0.1074	0.0225	0.0672	0.1673
Yes	Midwest	Female	Below Poverty Level	12	0.1025	0.0199	0.0664	0.1551
Yes	Midwest	Female	Below Poverty Level	13	0.1096	0.0211	0.0712	0.1649
Yes	Midwest	Female	Below Poverty Level	14	0.1236	0.0229	0.0815	0.1830
Yes	Midwest	Female	Below Poverty Level	15	0.1412	0.0266	0.0924	0.2099
Yes	Midwest	Female	Below Poverty Level	16	0.1633	0.0413	0.0914	0.2746
Yes	Midwest	Female	Below Poverty Level	17	0.1906	0.0779	0.0722	0.4158
Yes	Midwest	Male	Above Poverty Level	0	0.0122	0.0064	0.0038	0.0384
Yes	Midwest	Male	Above Poverty Level	1	0.0268	0.0083	0.0135	0.0525
Yes	Midwest	Male	Above Poverty Level	2	0.0480	0.0091	0.0315	0.0725
Yes	Midwest	Male	Above Poverty Level	3	0.0710	0.0113	0.0500	0.1001
Yes	Midwest	Male	Above Poverty Level	4	0.0842	0.0134	0.0591	0.1187
Yes	Midwest	Male	Above Poverty Level	5	0.0934	0.0138	0.0673	0.1282
Yes	Midwest	Male	Above Poverty Level	6	0.1056	0.0144	0.0779	0.1416
Yes	Midwest	Male	Above Poverty Level	7	0.1117	0.0149	0.0829	0.1489
Yes	Midwest	Male	Above Poverty Level	8	0.1111	0.0152	0.0820	0.1489
Yes	Midwest	Male	Above Poverty Level	9	0.1138	0.0155	0.0840	0.1525
Yes	Midwest	Male	Above Poverty Level	10	0.1126	0.0153	0.0831	0.1507
Yes	Midwest	Male	Above Poverty Level	11	0.1108	0.0146	0.0826	0.1472
Yes	Midwest	Male	Above Poverty Level	12	0.1129	0.0137	0.0861	0.1466
Yes	Midwest	Male	Above Poverty Level	13	0.1139	0.0132	0.0880	0.1462
Yes	Midwest	Male	Above Poverty Level	14	0.1128	0.0127	0.0878	0.1438
Yes	Midwest	Male	Above Poverty Level	15	0.1054	0.0118	0.0822	0.1343
Yes	Midwest	Male	Above Poverty Level	16	0.0935	0.0133	0.0682	0.1269
Yes	Midwest	Male	Above Poverty Level	17	0.0782	0.0184	0.0462	0.1292
Yes	Midwest	Male	Below Poverty Level	0	0.0402	0.0177	0.0151	0.1028
Yes	Midwest	Male	Below Poverty Level	1	0.0824	0.0213	0.0463	0.1425
Yes	Midwest	Male	Below Poverty Level	2	0.1338	0.0225	0.0917	0.1911
Yes	Midwest	Male	Below Poverty Level	3	0.1774	0.0255	0.1282	0.2401
Yes	Midwest	Male	Below Poverty Level	4	0.1949	0.0267	0.1429	0.2601
Yes	Midwest	Male	Below Poverty Level	5	0.1867	0.0237	0.1402	0.2443
Yes	Midwest	Male	Below Poverty Level	6	0.1807	0.0222	0.1371	0.2344
Yes	Midwest	Male	Below Poverty Level	7	0.1734	0.0221	0.1301	0.2273
Yes	Midwest	Male	Below Poverty Level	8	0.1739	0.0248	0.1260	0.2350
Yes	Midwest	Male	Below Poverty Level	9	0.1814	0.0269	0.1297	0.2478

Appendix 5C, Attachment C, Table 2. Smoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	Midwest	Male	Below Poverty Level	10	0.1813	0.0282	0.1275	0.2514
Yes	Midwest	Male	Below Poverty Level	11	0.1749	0.0282	0.1214	0.2454
Yes	Midwest	Male	Below Poverty Level	12	0.1702	0.0298	0.1143	0.2457
Yes	Midwest	Male	Below Poverty Level	13	0.1499	0.0296	0.0959	0.2268
Yes	Midwest	Male	Below Poverty Level	14	0.1366	0.0269	0.0876	0.2066
Yes	Midwest	Male	Below Poverty Level	15	0.1484	0.0268	0.0987	0.2169
Yes	Midwest	Male	Below Poverty Level	16	0.1846	0.0359	0.1185	0.2761
Yes	Midwest	Male	Below Poverty Level	17	0.2590	0.0740	0.1306	0.4484
Yes	Northeast	Female	Above Poverty Level	0	0.0153	0.0089	0.0042	0.0537
Yes	Northeast	Female	Above Poverty Level	1	0.0281	0.0096	0.0132	0.0589
Yes	Northeast	Female	Above Poverty Level	2	0.0437	0.0090	0.0276	0.0683
Yes	Northeast	Female	Above Poverty Level	3	0.0584	0.0098	0.0402	0.0840
Yes	Northeast	Female	Above Poverty Level	4	0.0657	0.0112	0.0449	0.0950
Yes	Northeast	Female	Above Poverty Level	5	0.0668	0.0111	0.0461	0.0958
Yes	Northeast	Female	Above Poverty Level	6	0.0678	0.0111	0.0471	0.0967
Yes	Northeast	Female	Above Poverty Level	7	0.0696	0.0114	0.0482	0.0993
Yes	Northeast	Female	Above Poverty Level	8	0.0737	0.0124	0.0506	0.1062
Yes	Northeast	Female	Above Poverty Level	9	0.0840	0.0147	0.0569	0.1224
Yes	Northeast	Female	Above Poverty Level	10	0.0807	0.0144	0.0541	0.1187
Yes	Northeast	Female	Above Poverty Level	11	0.0710	0.0134	0.0466	0.1068
Yes	Northeast	Female	Above Poverty Level	12	0.0629	0.0116	0.0416	0.0938
Yes	Northeast	Female	Above Poverty Level	13	0.0680	0.0113	0.0469	0.0976
Yes	Northeast	Female	Above Poverty Level	14	0.0786	0.0117	0.0564	0.1085
Yes	Northeast	Female	Above Poverty Level	15	0.0913	0.0120	0.0681	0.1214
Yes	Northeast	Female	Above Poverty Level	16	0.1095	0.0165	0.0781	0.1513
Yes	Northeast	Female	Above Poverty Level	17	0.1328	0.0330	0.0753	0.2234
Yes	Northeast	Female	Below Poverty Level	0	0.0234	0.0142	0.0061	0.0856
Yes	Northeast	Female	Below Poverty Level	1	0.0564	0.0190	0.0266	0.1157
Yes	Northeast	Female	Below Poverty Level	2	0.1040	0.0219	0.0648	0.1627
Yes	Northeast	Female	Below Poverty Level	3	0.1466	0.0272	0.0964	0.2167
Yes	Northeast	Female	Below Poverty Level	4	0.1618	0.0304	0.1056	0.2400
Yes	Northeast	Female	Below Poverty Level	5	0.1441	0.0280	0.0928	0.2168
Yes	Northeast	Female	Below Poverty Level	6	0.1124	0.0238	0.0698	0.1761
Yes	Northeast	Female	Below Poverty Level	7	0.0751	0.0174	0.0447	0.1234
Yes	Northeast	Female	Below Poverty Level	8	0.0633	0.0157	0.0364	0.1078
Yes	Northeast	Female	Below Poverty Level	9	0.0838	0.0188	0.0507	0.1355
Yes	Northeast	Female	Below Poverty Level	10	0.1288	0.0270	0.0802	0.2004
Yes	Northeast	Female	Below Poverty Level	11	0.1778	0.0336	0.1154	0.2638
Yes	Northeast	Female	Below Poverty Level	12	0.2073	0.0349	0.1410	0.2941
Yes	Northeast	Female	Below Poverty Level	13	0.2063	0.0328	0.1435	0.2873
Yes	Northeast	Female	Below Poverty Level	14	0.1929	0.0287	0.1375	0.2637
Yes	Northeast	Female	Below Poverty Level	15	0.1703	0.0235	0.1248	0.2281
Yes	Northeast	Female	Below Poverty Level	16	0.1414	0.0234	0.0974	0.2009
Yes	Northeast	Female	Below Poverty Level	17	0.1108	0.0327	0.0567	0.2051
Yes	Northeast	Male	Above Poverty Level	0	0.0225	0.0108	0.0078	0.0633
Yes	Northeast	Male	Above Poverty Level	1	0.0368	0.0105	0.0195	0.0682
Yes	Northeast	Male	Above Poverty Level	2	0.0562	0.0104	0.0373	0.0838
Yes	Northeast	Male	Above Poverty Level	3	0.0797	0.0127	0.0559	0.1123
Yes	Northeast	Male	Above Poverty Level	4	0.1035	0.0162	0.0730	0.1449
Yes	Northeast	Male	Above Poverty Level	5	0.1289	0.0187	0.0931	0.1757
Yes	Northeast	Male	Above Poverty Level	6	0.1472	0.0190	0.1102	0.1938
Yes	Northeast	Male	Above Poverty Level	7	0.1423	0.0181	0.1070	0.1868
Yes	Northeast	Male	Above Poverty Level	8	0.1290	0.0163	0.0973	0.1690
Yes	Northeast	Male	Above Poverty Level	9	0.1251	0.0159	0.0943	0.1641
Yes	Northeast	Male	Above Poverty Level	10	0.1288	0.0155	0.0985	0.1668
Yes	Northeast	Male	Above Poverty Level	11	0.1262	0.0139	0.0989	0.1598
Yes	Northeast	Male	Above Poverty Level	12	0.1246	0.0139	0.0971	0.1584
Yes	Northeast	Male	Above Poverty Level	13	0.1230	0.0149	0.0939	0.1594
Yes	Northeast	Male	Above Poverty Level	14	0.1207	0.0144	0.0925	0.1560
Yes	Northeast	Male	Above Poverty Level	15	0.1114	0.0126	0.0868	0.1420
Yes	Northeast	Male	Above Poverty Level	16	0.0983	0.0124	0.0743	0.1291
Yes	Northeast	Male	Above Poverty Level	17	0.0823	0.0171	0.0518	0.1285
Yes	Northeast	Male	Below Poverty Level	0	0.0930	0.0402	0.0347	0.2262
Yes	Northeast	Male	Below Poverty Level	1	0.1202	0.0280	0.0710	0.1964

Appendix 5C, Attachment C, Table 2. Smoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	Northeast	Male	Below Poverty Level	2	0.1475	0.0256	0.0997	0.2130
Yes	Northeast	Male	Below Poverty Level	3	0.1714	0.0311	0.1134	0.2508
Yes	Northeast	Male	Below Poverty Level	4	0.1860	0.0335	0.1232	0.2708
Yes	Northeast	Male	Below Poverty Level	5	0.2060	0.0276	0.1519	0.2732
Yes	Northeast	Male	Below Poverty Level	6	0.2256	0.0276	0.1708	0.2919
Yes	Northeast	Male	Below Poverty Level	7	0.2496	0.0317	0.1866	0.3255
Yes	Northeast	Male	Below Poverty Level	8	0.2727	0.0387	0.1964	0.3653
Yes	Northeast	Male	Below Poverty Level	9	0.2579	0.0395	0.1810	0.3535
Yes	Northeast	Male	Below Poverty Level	10	0.2318	0.0366	0.1611	0.3216
Yes	Northeast	Male	Below Poverty Level	11	0.1902	0.0310	0.1311	0.2678
Yes	Northeast	Male	Below Poverty Level	12	0.1624	0.0268	0.1116	0.2302
Yes	Northeast	Male	Below Poverty Level	13	0.1641	0.0254	0.1155	0.2278
Yes	Northeast	Male	Below Poverty Level	14	0.1699	0.0251	0.1216	0.2323
Yes	Northeast	Male	Below Poverty Level	15	0.1797	0.0244	0.1321	0.2396
Yes	Northeast	Male	Below Poverty Level	16	0.1933	0.0276	0.1397	0.2612
Yes	Northeast	Male	Below Poverty Level	17	0.2097	0.0451	0.1274	0.3253
Yes	South	Female	Above Poverty Level	0	0.0131	0.0059	0.0048	0.0349
Yes	South	Female	Above Poverty Level	1	0.0228	0.0063	0.0124	0.0415
Yes	South	Female	Above Poverty Level	2	0.0352	0.0064	0.0236	0.0522
Yes	South	Female	Above Poverty Level	3	0.0495	0.0074	0.0355	0.0685
Yes	South	Female	Above Poverty Level	4	0.0633	0.0089	0.0464	0.0857
Yes	South	Female	Above Poverty Level	5	0.0740	0.0092	0.0561	0.0969
Yes	South	Female	Above Poverty Level	6	0.0826	0.0096	0.0638	0.1063
Yes	South	Female	Above Poverty Level	7	0.0888	0.0099	0.0695	0.1129
Yes	South	Female	Above Poverty Level	8	0.0860	0.0100	0.0666	0.1105
Yes	South	Female	Above Poverty Level	9	0.0791	0.0095	0.0606	0.1025
Yes	South	Female	Above Poverty Level	10	0.0747	0.0088	0.0576	0.0963
Yes	South	Female	Above Poverty Level	11	0.0736	0.0085	0.0570	0.0944
Yes	South	Female	Above Poverty Level	12	0.0776	0.0087	0.0606	0.0989
Yes	South	Female	Above Poverty Level	13	0.0851	0.0093	0.0669	0.1078
Yes	South	Female	Above Poverty Level	14	0.0871	0.0093	0.0688	0.1099
Yes	South	Female	Above Poverty Level	15	0.0876	0.0087	0.0702	0.1087
Yes	South	Female	Above Poverty Level	16	0.0859	0.0091	0.0681	0.1080
Yes	South	Female	Above Poverty Level	17	0.0819	0.0136	0.0567	0.1169
Yes	South	Female	Below Poverty Level	0	0.0396	0.0135	0.0186	0.0823
Yes	South	Female	Below Poverty Level	1	0.0573	0.0113	0.0371	0.0876
Yes	South	Female	Below Poverty Level	2	0.0772	0.0109	0.0564	0.1048
Yes	South	Female	Below Poverty Level	3	0.0963	0.0136	0.0704	0.1306
Yes	South	Female	Below Poverty Level	4	0.1120	0.0165	0.0805	0.1536
Yes	South	Female	Below Poverty Level	5	0.1206	0.0174	0.0874	0.1641
Yes	South	Female	Below Poverty Level	6	0.1219	0.0173	0.0888	0.1652
Yes	South	Female	Below Poverty Level	7	0.1152	0.0162	0.0842	0.1556
Yes	South	Female	Below Poverty Level	8	0.1131	0.0157	0.0829	0.1524
Yes	South	Female	Below Poverty Level	9	0.1190	0.0161	0.0880	0.1591
Yes	South	Female	Below Poverty Level	10	0.1208	0.0175	0.0874	0.1646
Yes	South	Female	Below Poverty Level	11	0.1195	0.0178	0.0857	0.1642
Yes	South	Female	Below Poverty Level	12	0.1275	0.0192	0.0910	0.1757
Yes	South	Female	Below Poverty Level	13	0.1405	0.0197	0.1026	0.1893
Yes	South	Female	Below Poverty Level	14	0.1394	0.0184	0.1037	0.1848
Yes	South	Female	Below Poverty Level	15	0.1296	0.0166	0.0973	0.1706
Yes	South	Female	Below Poverty Level	16	0.1136	0.0184	0.0791	0.1605
Yes	South	Female	Below Poverty Level	17	0.0923	0.0249	0.0503	0.1634
Yes	South	Male	Above Poverty Level	0	0.0228	0.0070	0.0116	0.0443
Yes	South	Male	Above Poverty Level	1	0.0476	0.0082	0.0325	0.0693
Yes	South	Male	Above Poverty Level	2	0.0793	0.0089	0.0619	0.1011
Yes	South	Male	Above Poverty Level	3	0.1076	0.0109	0.0859	0.1341
Yes	South	Male	Above Poverty Level	4	0.1193	0.0123	0.0949	0.1490
Yes	South	Male	Above Poverty Level	5	0.1194	0.0117	0.0960	0.1475
Yes	South	Male	Above Poverty Level	6	0.1145	0.0111	0.0924	0.1411
Yes	South	Male	Above Poverty Level	7	0.1071	0.0105	0.0861	0.1323
Yes	South	Male	Above Poverty Level	8	0.1011	0.0099	0.0813	0.1251
Yes	South	Male	Above Poverty Level	9	0.1000	0.0098	0.0806	0.1236
Yes	South	Male	Above Poverty Level	10	0.1059	0.0102	0.0855	0.1305
Yes	South	Male	Above Poverty Level	11	0.1122	0.0106	0.0910	0.1376

Appendix 5C, Attachment C, Table 2. Smoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	South	Male	Above Poverty Level	12	0.1103	0.0105	0.0893	0.1356
Yes	South	Male	Above Poverty Level	13	0.1052	0.0105	0.0843	0.1305
Yes	South	Male	Above Poverty Level	14	0.0983	0.0094	0.0795	0.1210
Yes	South	Male	Above Poverty Level	15	0.0899	0.0081	0.0737	0.1093
Yes	South	Male	Above Poverty Level	16	0.0811	0.0089	0.0636	0.1028
Yes	South	Male	Above Poverty Level	17	0.0727	0.0136	0.0479	0.1089
Yes	South	Male	Below Poverty Level	0	0.0499	0.0126	0.0285	0.0860
Yes	South	Male	Below Poverty Level	1	0.0749	0.0110	0.0542	0.1027
Yes	South	Male	Below Poverty Level	2	0.1033	0.0116	0.0805	0.1316
Yes	South	Male	Below Poverty Level	3	0.1305	0.0149	0.1012	0.1666
Yes	South	Male	Below Poverty Level	4	0.1519	0.0177	0.1171	0.1948
Yes	South	Male	Below Poverty Level	5	0.1595	0.0180	0.1240	0.2029
Yes	South	Male	Below Poverty Level	6	0.1598	0.0185	0.1234	0.2045
Yes	South	Male	Below Poverty Level	7	0.1540	0.0180	0.1186	0.1977
Yes	South	Male	Below Poverty Level	8	0.1466	0.0170	0.1130	0.1879
Yes	South	Male	Below Poverty Level	9	0.1457	0.0170	0.1122	0.1870
Yes	South	Male	Below Poverty Level	10	0.1504	0.0171	0.1167	0.1917
Yes	South	Male	Below Poverty Level	11	0.1508	0.0171	0.1171	0.1921
Yes	South	Male	Below Poverty Level	12	0.1506	0.0184	0.1146	0.1955
Yes	South	Male	Below Poverty Level	13	0.1470	0.0192	0.1097	0.1943
Yes	South	Male	Below Poverty Level	14	0.1345	0.0179	0.0999	0.1788
Yes	South	Male	Below Poverty Level	15	0.1215	0.0159	0.0907	0.1607
Yes	South	Male	Below Poverty Level	16	0.1080	0.0164	0.0770	0.1494
Yes	South	Male	Below Poverty Level	17	0.0948	0.0227	0.0555	0.1573
Yes	West	Female	Above Poverty Level	0	0.0077	0.0049	0.0019	0.0306
Yes	West	Female	Above Poverty Level	1	0.0122	0.0046	0.0053	0.0278
Yes	West	Female	Above Poverty Level	2	0.0181	0.0045	0.0105	0.0310
Yes	West	Female	Above Poverty Level	3	0.0248	0.0055	0.0153	0.0401
Yes	West	Female	Above Poverty Level	4	0.0305	0.0068	0.0186	0.0494
Yes	West	Female	Above Poverty Level	5	0.0382	0.0077	0.0245	0.0590
Yes	West	Female	Above Poverty Level	6	0.0482	0.0091	0.0318	0.0724
Yes	West	Female	Above Poverty Level	7	0.0573	0.0098	0.0393	0.0829
Yes	West	Female	Above Poverty Level	8	0.0628	0.0106	0.0432	0.0904
Yes	West	Female	Above Poverty Level	9	0.0697	0.0106	0.0497	0.0970
Yes	West	Female	Above Poverty Level	10	0.0768	0.0099	0.0577	0.1016
Yes	West	Female	Above Poverty Level	11	0.0786	0.0094	0.0603	0.1018
Yes	West	Female	Above Poverty Level	12	0.0808	0.0100	0.0615	0.1056
Yes	West	Female	Above Poverty Level	13	0.0829	0.0108	0.0621	0.1100
Yes	West	Female	Above Poverty Level	14	0.0845	0.0111	0.0632	0.1121
Yes	West	Female	Above Poverty Level	15	0.0908	0.0110	0.0694	0.1179
Yes	West	Female	Above Poverty Level	16	0.1016	0.0129	0.0766	0.1337
Yes	West	Female	Above Poverty Level	17	0.1180	0.0236	0.0753	0.1803
Yes	West	Female	Below Poverty Level	0	0.0244	0.0144	0.0066	0.0862
Yes	West	Female	Below Poverty Level	1	0.0270	0.0091	0.0128	0.0561
Yes	West	Female	Below Poverty Level	2	0.0306	0.0074	0.0179	0.0518
Yes	West	Female	Below Poverty Level	3	0.0354	0.0090	0.0201	0.0615
Yes	West	Female	Below Poverty Level	4	0.0407	0.0112	0.0221	0.0738
Yes	West	Female	Below Poverty Level	5	0.0577	0.0146	0.0328	0.0996
Yes	West	Female	Below Poverty Level	6	0.0807	0.0185	0.0483	0.1319
Yes	West	Female	Below Poverty Level	7	0.0954	0.0181	0.0624	0.1434
Yes	West	Female	Below Poverty Level	8	0.0876	0.0159	0.0583	0.1296
Yes	West	Female	Below Poverty Level	9	0.0648	0.0127	0.0419	0.0989
Yes	West	Female	Below Poverty Level	10	0.0495	0.0107	0.0306	0.0792
Yes	West	Female	Below Poverty Level	11	0.0473	0.0110	0.0282	0.0781
Yes	West	Female	Below Poverty Level	12	0.0606	0.0137	0.0366	0.0988
Yes	West	Female	Below Poverty Level	13	0.0845	0.0179	0.0526	0.1329
Yes	West	Female	Below Poverty Level	14	0.0931	0.0180	0.0603	0.1411
Yes	West	Female	Below Poverty Level	15	0.0846	0.0154	0.0562	0.1253
Yes	West	Female	Below Poverty Level	16	0.0629	0.0143	0.0379	0.1026
Yes	West	Female	Below Poverty Level	17	0.0376	0.0146	0.0158	0.0868
Yes	West	Male	Above Poverty Level	0	0.0007	0.0007	0.0001	0.0067
Yes	West	Male	Above Poverty Level	1	0.0052	0.0027	0.0014	0.0192
Yes	West	Male	Above Poverty Level	2	0.0225	0.0063	0.0112	0.0447
Yes	West	Male	Above Poverty Level	3	0.0596	0.0095	0.0398	0.0884

Appendix 5C, Attachment C, Table 2. Smoothed prevalence for children "STILL" having asthma.								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	West	Male	Above Poverty Level	4	0.0989	0.0140	0.0691	0.1397
Yes	West	Male	Above Poverty Level	5	0.1070	0.0147	0.0754	0.1496
Yes	West	Male	Above Poverty Level	6	0.0959	0.0141	0.0660	0.1372
Yes	West	Male	Above Poverty Level	7	0.0830	0.0126	0.0565	0.1203
Yes	West	Male	Above Poverty Level	8	0.0877	0.0124	0.0613	0.1239
Yes	West	Male	Above Poverty Level	9	0.1029	0.0135	0.0737	0.1419
Yes	West	Male	Above Poverty Level	10	0.1189	0.0140	0.0883	0.1584
Yes	West	Male	Above Poverty Level	11	0.1292	0.0153	0.0955	0.1724
Yes	West	Male	Above Poverty Level	12	0.1214	0.0154	0.0879	0.1653
Yes	West	Male	Above Poverty Level	13	0.1050	0.0139	0.0749	0.1452
Yes	West	Male	Above Poverty Level	14	0.0981	0.0127	0.0707	0.1346
Yes	West	Male	Above Poverty Level	15	0.0997	0.0116	0.0742	0.1327
Yes	West	Male	Above Poverty Level	16	0.1091	0.0128	0.0810	0.1454
Yes	West	Male	Above Poverty Level	17	0.1290	0.0231	0.0814	0.1984
Yes	West	Male	Below Poverty Level	0	0.0263	0.0130	0.0088	0.0761
Yes	West	Male	Below Poverty Level	1	0.0374	0.0101	0.0204	0.0673
Yes	West	Male	Below Poverty Level	2	0.0518	0.0086	0.0358	0.0742
Yes	West	Male	Below Poverty Level	3	0.0681	0.0105	0.0483	0.0952
Yes	West	Male	Below Poverty Level	4	0.0871	0.0143	0.0604	0.1240
Yes	West	Male	Below Poverty Level	5	0.1074	0.0173	0.0749	0.1517
Yes	West	Male	Below Poverty Level	6	0.1167	0.0183	0.0820	0.1635
Yes	West	Male	Below Poverty Level	7	0.1138	0.0186	0.0789	0.1615
Yes	West	Male	Below Poverty Level	8	0.1073	0.0177	0.0741	0.1529
Yes	West	Male	Below Poverty Level	9	0.0964	0.0164	0.0659	0.1389
Yes	West	Male	Below Poverty Level	10	0.0830	0.0149	0.0557	0.1221
Yes	West	Male	Below Poverty Level	11	0.0745	0.0151	0.0474	0.1152
Yes	West	Male	Below Poverty Level	12	0.0825	0.0165	0.0527	0.1268
Yes	West	Male	Below Poverty Level	13	0.1000	0.0197	0.0643	0.1524
Yes	West	Male	Below Poverty Level	14	0.1074	0.0200	0.0707	0.1600
Yes	West	Male	Below Poverty Level	15	0.1120	0.0193	0.0760	0.1620
Yes	West	Male	Below Poverty Level	16	0.1127	0.0222	0.0724	0.1714
Yes	West	Male	Below Poverty Level	17	0.1084	0.0340	0.0531	0.2088

Appendix 5C, Attachment C, Table 3. Smoothed prevalence for adults "EVER" having asthma								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age_group**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	Midwest	Female	Above Poverty Level	18-24	0.1642	0.0141	0.1219	0.2176
Yes	Midwest	Female	Above Poverty Level	25-34	0.1341	0.0063	0.1142	0.1568
Yes	Midwest	Female	Above Poverty Level	35-44	0.1193	0.0058	0.1012	0.1402
Yes	Midwest	Female	Above Poverty Level	45-54	0.1204	0.0057	0.1025	0.1409
Yes	Midwest	Female	Above Poverty Level	55-64	0.1246	0.0066	0.1040	0.1486
Yes	Midwest	Female	Above Poverty Level	65-74	0.1165	0.0062	0.0971	0.1392
Yes	Midwest	Female	Above Poverty Level	75+	0.0980	0.0089	0.0719	0.1322
Yes	Midwest	Female	Below Poverty Level	18-24	0.2014	0.0153	0.1531	0.2603
Yes	Midwest	Female	Below Poverty Level	25-34	0.1812	0.0114	0.1445	0.2248
Yes	Midwest	Female	Below Poverty Level	35-44	0.1782	0.0130	0.1370	0.2284
Yes	Midwest	Female	Below Poverty Level	45-54	0.2104	0.0146	0.1638	0.2662
Yes	Midwest	Female	Below Poverty Level	55-64	0.2295	0.0164	0.1770	0.2920
Yes	Midwest	Female	Below Poverty Level	65-74	0.1892	0.0145	0.1435	0.2453
Yes	Midwest	Female	Below Poverty Level	75+	0.1176	0.0173	0.0690	0.1933
Yes	Midwest	Male	Above Poverty Level	18-24	0.1705	0.0149	0.1249	0.2284
Yes	Midwest	Male	Above Poverty Level	25-34	0.1209	0.0063	0.1008	0.1444
Yes	Midwest	Male	Above Poverty Level	35-44	0.0886	0.0053	0.0719	0.1087
Yes	Midwest	Male	Above Poverty Level	45-54	0.0727	0.0046	0.0583	0.0904
Yes	Midwest	Male	Above Poverty Level	55-64	0.0770	0.0054	0.0602	0.0980
Yes	Midwest	Male	Above Poverty Level	65-74	0.0828	0.0058	0.0647	0.1053
Yes	Midwest	Male	Above Poverty Level	75+	0.0847	0.0106	0.0545	0.1292
Yes	Midwest	Male	Below Poverty Level	18-24	0.1654	0.0175	0.1122	0.2370
Yes	Midwest	Male	Below Poverty Level	25-34	0.1143	0.0109	0.0808	0.1593
Yes	Midwest	Male	Below Poverty Level	35-44	0.1066	0.0122	0.0703	0.1585
Yes	Midwest	Male	Below Poverty Level	45-54	0.1376	0.0146	0.0936	0.1979
Yes	Midwest	Male	Below Poverty Level	55-64	0.1643	0.0164	0.1141	0.2309
Yes	Midwest	Male	Below Poverty Level	65-74	0.1396	0.0160	0.0918	0.2068
Yes	Midwest	Male	Below Poverty Level	75+	0.0853	0.0205	0.0353	0.1920
Yes	Northeast	Female	Above Poverty Level	18-24	0.1791	0.0176	0.1265	0.2474
Yes	Northeast	Female	Above Poverty Level	25-34	0.1423	0.0076	0.1183	0.1701
Yes	Northeast	Female	Above Poverty Level	35-44	0.1256	0.0072	0.1029	0.1525
Yes	Northeast	Female	Above Poverty Level	45-54	0.1246	0.0071	0.1024	0.1509
Yes	Northeast	Female	Above Poverty Level	55-64	0.1281	0.0076	0.1043	0.1565
Yes	Northeast	Female	Above Poverty Level	65-74	0.1151	0.0070	0.0934	0.1412
Yes	Northeast	Female	Above Poverty Level	75+	0.0879	0.0098	0.0598	0.1273
Yes	Northeast	Female	Below Poverty Level	18-24	0.1646	0.0182	0.1104	0.2383
Yes	Northeast	Female	Below Poverty Level	25-34	0.1705	0.0110	0.1356	0.2123
Yes	Northeast	Female	Below Poverty Level	35-44	0.1842	0.0126	0.1442	0.2323
Yes	Northeast	Female	Below Poverty Level	45-54	0.2084	0.0143	0.1629	0.2627
Yes	Northeast	Female	Below Poverty Level	55-64	0.2180	0.0156	0.1684	0.2773
Yes	Northeast	Female	Below Poverty Level	65-74	0.1695	0.0118	0.1321	0.2149
Yes	Northeast	Female	Below Poverty Level	75+	0.0960	0.0125	0.0603	0.1495
Yes	Northeast	Male	Above Poverty Level	18-24	0.1728	0.0210	0.1126	0.2560
Yes	Northeast	Male	Above Poverty Level	25-34	0.1163	0.0081	0.0914	0.1469
Yes	Northeast	Male	Above Poverty Level	35-44	0.0932	0.0070	0.0721	0.1197
Yes	Northeast	Male	Above Poverty Level	45-54	0.0901	0.0063	0.0710	0.1139
Yes	Northeast	Male	Above Poverty Level	55-64	0.0963	0.0072	0.0744	0.1237
Yes	Northeast	Male	Above Poverty Level	65-74	0.0874	0.0073	0.0656	0.1155
Yes	Northeast	Male	Above Poverty Level	75+	0.0708	0.0118	0.0398	0.1229
Yes	Northeast	Male	Below Poverty Level	18-24	0.1734	0.0193	0.1138	0.2552
Yes	Northeast	Male	Below Poverty Level	25-34	0.1323	0.0138	0.0896	0.1911
Yes	Northeast	Male	Below Poverty Level	35-44	0.1182	0.0135	0.0772	0.1768
Yes	Northeast	Male	Below Poverty Level	45-54	0.1254	0.0144	0.0816	0.1879
Yes	Northeast	Male	Below Poverty Level	55-64	0.1361	0.0198	0.0786	0.2253
Yes	Northeast	Male	Below Poverty Level	65-74	0.1305	0.0195	0.0743	0.2191
Yes	Northeast	Male	Below Poverty Level	75+	0.0988	0.0255	0.0373	0.2366
Yes	South	Female	Above Poverty Level	18-24	0.1533	0.0114	0.1185	0.1959
Yes	South	Female	Above Poverty Level	25-34	0.1235	0.0054	0.1065	0.1429
Yes	South	Female	Above Poverty Level	35-44	0.1114	0.0050	0.0956	0.1295
Yes	South	Female	Above Poverty Level	45-54	0.1149	0.0047	0.0998	0.1320
Yes	South	Female	Above Poverty Level	55-64	0.1261	0.0058	0.1077	0.1472
Yes	South	Female	Above Poverty Level	65-74	0.1188	0.0058	0.1004	0.1400
Yes	South	Female	Above Poverty Level	75+	0.0959	0.0087	0.0701	0.1297
Yes	South	Female	Below Poverty Level	18-24	0.1491	0.0122	0.1107	0.1978

Appendix 5C, Attachment C, Table 3. Smoothed prevalence for adults "EVER" having asthma								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age_group**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	South	Female	Below Poverty Level	25-34	0.1365	0.0066	0.1149	0.1614
Yes	South	Female	Below Poverty Level	35-44	0.1414	0.0078	0.1159	0.1714
Yes	South	Female	Below Poverty Level	45-54	0.1686	0.0097	0.1369	0.2059
Yes	South	Female	Below Poverty Level	55-64	0.1881	0.0115	0.1505	0.2324
Yes	South	Female	Below Poverty Level	65-74	0.1651	0.0101	0.1325	0.2039
Yes	South	Female	Below Poverty Level	75+	0.1125	0.0124	0.0755	0.1644
Yes	South	Male	Above Poverty Level	18-24	0.1445	0.0095	0.1147	0.1805
Yes	South	Male	Above Poverty Level	25-34	0.1086	0.0050	0.0926	0.1269
Yes	South	Male	Above Poverty Level	35-44	0.0860	0.0044	0.0720	0.1025
Yes	South	Male	Above Poverty Level	45-54	0.0742	0.0040	0.0616	0.0891
Yes	South	Male	Above Poverty Level	55-64	0.0733	0.0045	0.0594	0.0902
Yes	South	Male	Above Poverty Level	65-74	0.0790	0.0048	0.0639	0.0974
Yes	South	Male	Above Poverty Level	75+	0.0900	0.0102	0.0606	0.1316
Yes	South	Male	Below Poverty Level	18-24	0.1433	0.0144	0.1000	0.2013
Yes	South	Male	Below Poverty Level	25-34	0.1031	0.0087	0.0766	0.1376
Yes	South	Male	Below Poverty Level	35-44	0.0934	0.0090	0.0664	0.1300
Yes	South	Male	Below Poverty Level	45-54	0.1055	0.0101	0.0751	0.1462
Yes	South	Male	Below Poverty Level	55-64	0.1072	0.0108	0.0750	0.1510
Yes	South	Male	Below Poverty Level	65-74	0.0942	0.0092	0.0666	0.1314
Yes	South	Male	Below Poverty Level	75+	0.0712	0.0123	0.0385	0.1279
Yes	West	Female	Above Poverty Level	18-24	0.1571	0.0135	0.1163	0.2089
Yes	West	Female	Above Poverty Level	25-34	0.1415	0.0067	0.1201	0.1660
Yes	West	Female	Above Poverty Level	35-44	0.1373	0.0070	0.1150	0.1631
Yes	West	Female	Above Poverty Level	45-54	0.1423	0.0067	0.1207	0.1670
Yes	West	Female	Above Poverty Level	55-64	0.1497	0.0071	0.1268	0.1758
Yes	West	Female	Above Poverty Level	65-74	0.1445	0.0070	0.1220	0.1704
Yes	West	Female	Above Poverty Level	75+	0.1266	0.0112	0.0929	0.1702
Yes	West	Female	Below Poverty Level	18-24	0.1434	0.0164	0.0945	0.2117
Yes	West	Female	Below Poverty Level	25-34	0.1318	0.0092	0.1026	0.1678
Yes	West	Female	Below Poverty Level	35-44	0.1440	0.0117	0.1074	0.1903
Yes	West	Female	Below Poverty Level	45-54	0.1806	0.0144	0.1350	0.2374
Yes	West	Female	Below Poverty Level	55-64	0.1713	0.0136	0.1284	0.2248
Yes	West	Female	Below Poverty Level	65-74	0.1511	0.0117	0.1141	0.1974
Yes	West	Female	Below Poverty Level	75+	0.1292	0.0177	0.0785	0.2054
Yes	West	Male	Above Poverty Level	18-24	0.1566	0.0173	0.1067	0.2240
Yes	West	Male	Above Poverty Level	25-34	0.1233	0.0069	0.1019	0.1485
Yes	West	Male	Above Poverty Level	35-44	0.1025	0.0060	0.0839	0.1247
Yes	West	Male	Above Poverty Level	45-54	0.0908	0.0054	0.0741	0.1107
Yes	West	Male	Above Poverty Level	55-64	0.0955	0.0059	0.0774	0.1174
Yes	West	Male	Above Poverty Level	65-74	0.1067	0.0068	0.0860	0.1318
Yes	West	Male	Above Poverty Level	75+	0.1265	0.0152	0.0834	0.1871
Yes	West	Male	Below Poverty Level	18-24	0.1521	0.0204	0.0938	0.2373
Yes	West	Male	Below Poverty Level	25-34	0.0942	0.0095	0.0660	0.1327
Yes	West	Male	Below Poverty Level	35-44	0.0885	0.0102	0.0590	0.1308
Yes	West	Male	Below Poverty Level	45-54	0.1133	0.0130	0.0753	0.1670
Yes	West	Male	Below Poverty Level	55-64	0.1237	0.0156	0.0789	0.1888
Yes	West	Male	Below Poverty Level	65-74	0.1134	0.0142	0.0726	0.1727
Yes	West	Male	Below Poverty Level	75+	0.0961	0.0190	0.0474	0.1849

Appendix 5C, Attachment C, Table 4. Smoothed prevalence for adults "STILL" having asthma								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age_group**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	Midwest	Female	Above Poverty Level	18-24	0.1046	0.0121	0.0703	0.1528
Yes	Midwest	Female	Above Poverty Level	25-34	0.0888	0.0057	0.0714	0.1100
Yes	Midwest	Female	Above Poverty Level	35-44	0.0835	0.0052	0.0675	0.1030
Yes	Midwest	Female	Above Poverty Level	45-44	0.0893	0.0050	0.0738	0.1077
Yes	Midwest	Female	Above Poverty Level	55-64	0.0909	0.0057	0.0736	0.1118
Yes	Midwest	Female	Above Poverty Level	65-74	0.0811	0.0051	0.0654	0.1002
Yes	Midwest	Female	Above Poverty Level	75+	0.0630	0.0067	0.0438	0.0898
Yes	Midwest	Female	Below Poverty Level	18-24	0.1327	0.0139	0.0907	0.1899
Yes	Midwest	Female	Below Poverty Level	25-34	0.1280	0.0095	0.0980	0.1656
Yes	Midwest	Female	Below Poverty Level	35-44	0.1315	0.0114	0.0961	0.1772
Yes	Midwest	Female	Below Poverty Level	45-44	0.1600	0.0134	0.1181	0.2132
Yes	Midwest	Female	Below Poverty Level	55-64	0.1777	0.0146	0.1318	0.2352
Yes	Midwest	Female	Below Poverty Level	65-74	0.1488	0.0128	0.1091	0.1998
Yes	Midwest	Female	Below Poverty Level	75+	0.0940	0.0157	0.0513	0.1659
Yes	Midwest	Male	Above Poverty Level	18-24	0.0807	0.0115	0.0491	0.1299
Yes	Midwest	Male	Above Poverty Level	25-34	0.0584	0.0045	0.0448	0.0758
Yes	Midwest	Male	Above Poverty Level	35-44	0.0479	0.0040	0.0359	0.0637
Yes	Midwest	Male	Above Poverty Level	45-44	0.0472	0.0038	0.0358	0.0620
Yes	Midwest	Male	Above Poverty Level	55-64	0.0522	0.0042	0.0395	0.0687
Yes	Midwest	Male	Above Poverty Level	65-74	0.0528	0.0045	0.0393	0.0706
Yes	Midwest	Male	Above Poverty Level	75+	0.0481	0.0081	0.0268	0.0847
Yes	Midwest	Male	Below Poverty Level	18-24	0.0912	0.0136	0.0542	0.1496
Yes	Midwest	Male	Below Poverty Level	25-34	0.0683	0.0091	0.0430	0.1067
Yes	Midwest	Male	Below Poverty Level	35-44	0.0694	0.0109	0.0402	0.1173
Yes	Midwest	Male	Below Poverty Level	45-44	0.1015	0.0141	0.0624	0.1610
Yes	Midwest	Male	Below Poverty Level	55-64	0.1338	0.0165	0.0866	0.2010
Yes	Midwest	Male	Below Poverty Level	65-74	0.1202	0.0161	0.0751	0.1869
Yes	Midwest	Male	Below Poverty Level	75+	0.0709	0.0210	0.0250	0.1850
Yes	Northeast	Female	Above Poverty Level	18-24	0.1098	0.0134	0.0721	0.1638
Yes	Northeast	Female	Above Poverty Level	25-34	0.0965	0.0065	0.0765	0.1210
Yes	Northeast	Female	Above Poverty Level	35-44	0.0899	0.0063	0.0708	0.1136
Yes	Northeast	Female	Above Poverty Level	45-44	0.0901	0.0060	0.0718	0.1124
Yes	Northeast	Female	Above Poverty Level	55-64	0.0917	0.0062	0.0727	0.1151
Yes	Northeast	Female	Above Poverty Level	65-74	0.0862	0.0059	0.0681	0.1085
Yes	Northeast	Female	Above Poverty Level	75+	0.0726	0.0093	0.0467	0.1110
Yes	Northeast	Female	Below Poverty Level	18-24	0.1212	0.0166	0.0744	0.1915
Yes	Northeast	Female	Below Poverty Level	25-34	0.1199	0.0093	0.0914	0.1559
Yes	Northeast	Female	Below Poverty Level	35-44	0.1338	0.0106	0.1013	0.1747
Yes	Northeast	Female	Below Poverty Level	45-44	0.1655	0.0127	0.1260	0.2143
Yes	Northeast	Female	Below Poverty Level	55-64	0.1824	0.0143	0.1381	0.2370
Yes	Northeast	Female	Below Poverty Level	65-74	0.1273	0.0098	0.0972	0.1650
Yes	Northeast	Female	Below Poverty Level	75+	0.0529	0.0086	0.0300	0.0917
Yes	Northeast	Male	Above Poverty Level	18-24	0.0922	0.0154	0.0509	0.1616
Yes	Northeast	Male	Above Poverty Level	25-34	0.0600	0.0058	0.0428	0.0836
Yes	Northeast	Male	Above Poverty Level	35-44	0.0488	0.0050	0.0340	0.0696
Yes	Northeast	Male	Above Poverty Level	45-44	0.0483	0.0051	0.0334	0.0693
Yes	Northeast	Male	Above Poverty Level	55-64	0.0563	0.0065	0.0376	0.0834
Yes	Northeast	Male	Above Poverty Level	65-74	0.0576	0.0063	0.0393	0.0837
Yes	Northeast	Male	Above Poverty Level	75+	0.0554	0.0106	0.0281	0.1062
Yes	Northeast	Male	Below Poverty Level	18-24	0.0791	0.0128	0.0430	0.1409
Yes	Northeast	Male	Below Poverty Level	25-34	0.0800	0.0119	0.0459	0.1360
Yes	Northeast	Male	Below Poverty Level	35-44	0.0805	0.0135	0.0427	0.1465
Yes	Northeast	Male	Below Poverty Level	45-44	0.0857	0.0162	0.0419	0.1672
Yes	Northeast	Male	Below Poverty Level	55-64	0.1064	0.0224	0.0475	0.2211
Yes	Northeast	Male	Below Poverty Level	65-74	0.1040	0.0200	0.0501	0.2035
Yes	Northeast	Male	Below Poverty Level	75+	0.0771	0.0236	0.0241	0.2203
Yes	South	Female	Above Poverty Level	18-24	0.0891	0.0083	0.0649	0.1212
Yes	South	Female	Above Poverty Level	25-34	0.0735	0.0039	0.0615	0.0876
Yes	South	Female	Above Poverty Level	35-44	0.0684	0.0036	0.0571	0.0817
Yes	South	Female	Above Poverty Level	45-44	0.0732	0.0037	0.0617	0.0866
Yes	South	Female	Above Poverty Level	55-64	0.0846	0.0046	0.0705	0.1012
Yes	South	Female	Above Poverty Level	65-74	0.0817	0.0047	0.0674	0.0987
Yes	South	Female	Above Poverty Level	75+	0.0641	0.0070	0.0443	0.0920
Yes	South	Female	Below Poverty Level	18-24	0.0948	0.0105	0.0641	0.1380

Appendix 5C, Attachment C, Table 4. Smoothed prevalence for adults "STILL" having asthma								
Smoothed	**Region**	**Gender**	**Poverty Status**	**Age_group**	**Prevalence**	**SE**	**LowerCI**	**UpperCI**
Yes	South	Female	Below Poverty Level	25-34	0.0942	0.0059	0.0758	0.1166
Yes	South	Female	Below Poverty Level	35-44	0.1086	0.0073	0.0859	0.1365
Yes	South	Female	Below Poverty Level	45-44	0.1446	0.0095	0.1149	0.1806
Yes	South	Female	Below Poverty Level	55-64	0.1618	0.0112	0.1267	0.2043
Yes	South	Female	Below Poverty Level	65-74	0.1379	0.0095	0.1082	0.1742
Yes	South	Female	Below Poverty Level	75+	0.0881	0.0109	0.0570	0.1337
Yes	South	Male	Above Poverty Level	18-24	0.0600	0.0073	0.0392	0.0907
Yes	South	Male	Above Poverty Level	25-34	0.0490	0.0035	0.0381	0.0629
Yes	South	Male	Above Poverty Level	35-44	0.0421	0.0033	0.0322	0.0550
Yes	South	Male	Above Poverty Level	45-44	0.0386	0.0031	0.0292	0.0510
Yes	South	Male	Above Poverty Level	55-64	0.0384	0.0034	0.0282	0.0520
Yes	South	Male	Above Poverty Level	65-74	0.0457	0.0038	0.0343	0.0607
Yes	South	Male	Above Poverty Level	75+	0.0627	0.0089	0.0382	0.1013
Yes	South	Male	Below Poverty Level	18-24	0.0583	0.0080	0.0358	0.0937
Yes	South	Male	Below Poverty Level	25-34	0.0443	0.0053	0.0290	0.0672
Yes	South	Male	Below Poverty Level	35-44	0.0492	0.0067	0.0303	0.0790
Yes	South	Male	Below Poverty Level	45-44	0.0720	0.0090	0.0460	0.1112
Yes	South	Male	Below Poverty Level	55-64	0.0771	0.0096	0.0492	0.1188
Yes	South	Male	Below Poverty Level	65-74	0.0608	0.0075	0.0390	0.0937
Yes	South	Male	Below Poverty Level	75+	0.0353	0.0082	0.0154	0.0787
Yes	West	Female	Above Poverty Level	18-24	0.0842	0.0115	0.0522	0.1328
Yes	West	Female	Above Poverty Level	25-34	0.0876	0.0054	0.0708	0.1080
Yes	West	Female	Above Poverty Level	35-44	0.0931	0.0062	0.0742	0.1163
Yes	West	Female	Above Poverty Level	45-44	0.0981	0.0065	0.0781	0.1226
Yes	West	Female	Above Poverty Level	55-64	0.1028	0.0067	0.0820	0.1281
Yes	West	Female	Above Poverty Level	65-74	0.0984	0.0061	0.0795	0.1213
Yes	West	Female	Above Poverty Level	75+	0.0825	0.0090	0.0565	0.1189
Yes	West	Female	Below Poverty Level	18-24	0.0863	0.0121	0.0524	0.1387
Yes	West	Female	Below Poverty Level	25-34	0.0934	0.0078	0.0695	0.1243
Yes	West	Female	Below Poverty Level	35-44	0.1091	0.0100	0.0789	0.1489
Yes	West	Female	Below Poverty Level	45-44	0.1332	0.0120	0.0967	0.1806
Yes	West	Female	Below Poverty Level	55-64	0.1292	0.0120	0.0929	0.1770
Yes	West	Female	Below Poverty Level	65-74	0.1169	0.0104	0.0854	0.1580
Yes	West	Female	Below Poverty Level	75+	0.1021	0.0148	0.0609	0.1662
Yes	West	Male	Above Poverty Level	18-24	0.0597	0.0092	0.0351	0.0998
Yes	West	Male	Above Poverty Level	25-34	0.0569	0.0046	0.0432	0.0745
Yes	West	Male	Above Poverty Level	35-44	0.0549	0.0045	0.0414	0.0723
Yes	West	Male	Above Poverty Level	45-44	0.0525	0.0046	0.0389	0.0704
Yes	West	Male	Above Poverty Level	55-64	0.0562	0.0053	0.0407	0.0770
Yes	West	Male	Above Poverty Level	65-74	0.0660	0.0058	0.0487	0.0889
Yes	West	Male	Above Poverty Level	75+	0.0783	0.0131	0.0437	0.1364
Yes	West	Male	Below Poverty Level	18-24	0.0720	0.0125	0.0389	0.1295
Yes	West	Male	Below Poverty Level	25-34	0.0484	0.0068	0.0294	0.0787
Yes	West	Male	Below Poverty Level	35-44	0.0539	0.0084	0.0311	0.0919
Yes	West	Male	Below Poverty Level	45-44	0.0784	0.0115	0.0465	0.1293
Yes	West	Male	Below Poverty Level	55-64	0.0936	0.0155	0.0517	0.1635
Yes	West	Male	Below Poverty Level	65-74	0.0758	0.0129	0.0413	0.1350
Yes	West	Male	Below Poverty Level	75+	0.0489	0.0136	0.0182	0.1250

Figure 1. Smoothed asthma 'EVER' prevalence rates and confidence intervals
region=Midwest pov_rat=Above Poverty Level

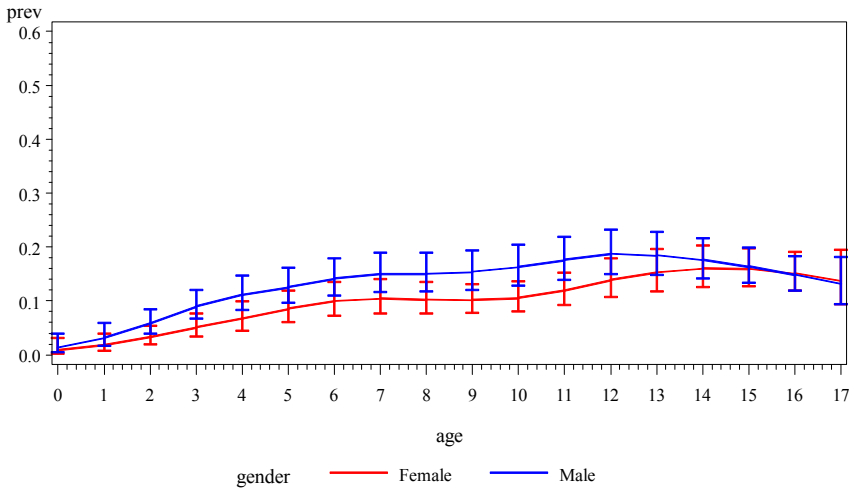


Figure 1. Smoothed asthma 'EVER' prevalence rates and confidence intervals
region=Northeast pov_rat=Above Poverty Level

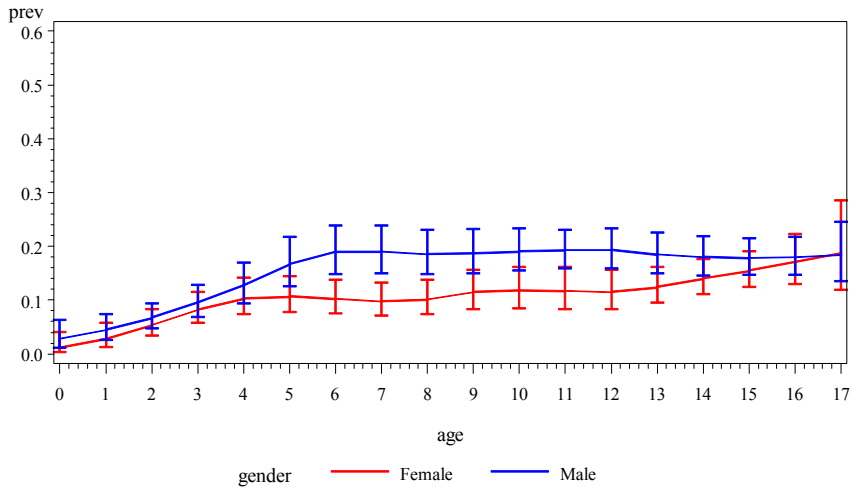


Figure 1. Smoothed asthma 'EVER' prevalence rates and confidence intervals
region=Midwest pov_rat=Below Poverty Level

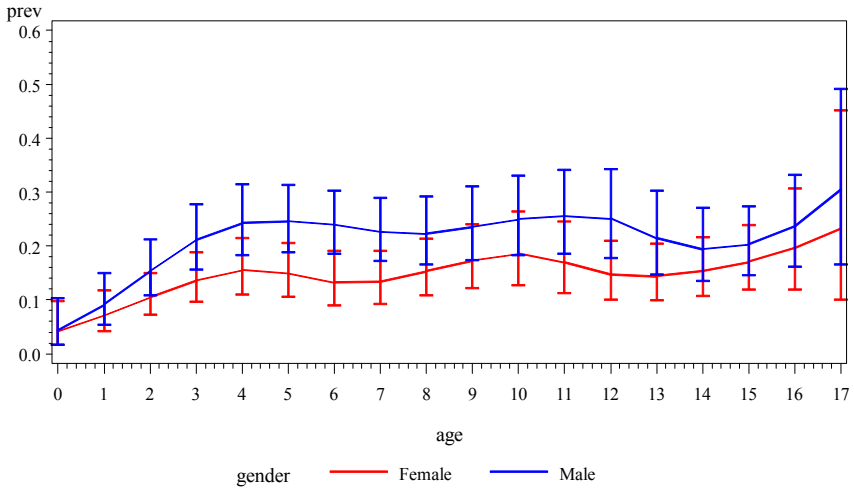
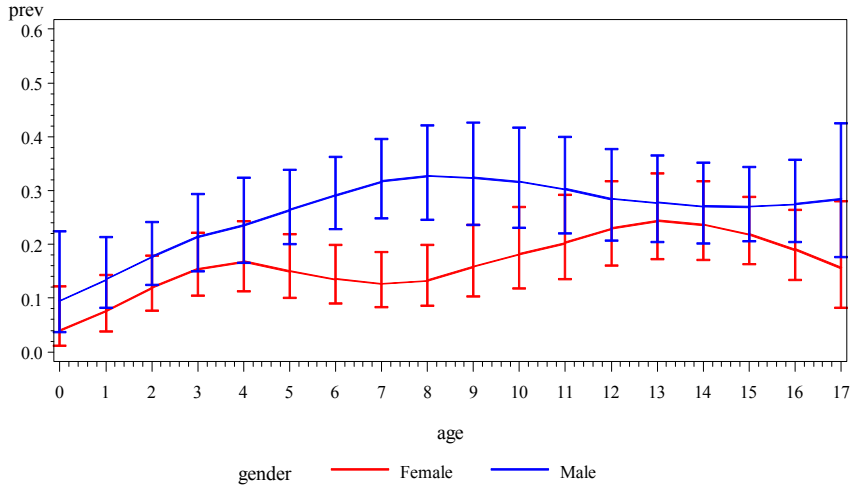


Figure 1. Smoothed asthma 'EVER' prevalence rates and confidence intervals
region=Northeast pov_rat=Below Poverty Level



Appendix 5C, Attachment C, Figure 1. Smoothed prevalence and confidence intervals for children 'EVER' having asthma.

Figure 1. Smoothed asthma 'EVER' prevalence rates and confidence intervals
region=South pov_rat=Above Poverty Level

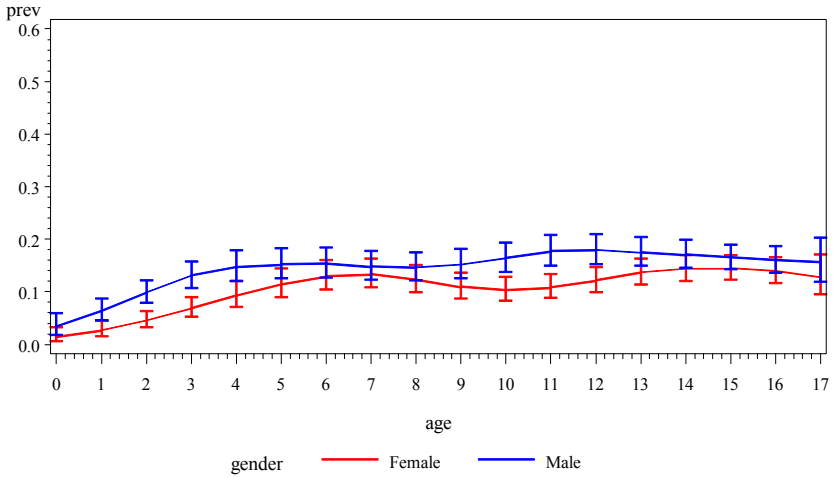


Figure 1. Smoothed asthma 'EVER' prevalence rates and confidence intervals
region=West pov_rat=Above Poverty Level

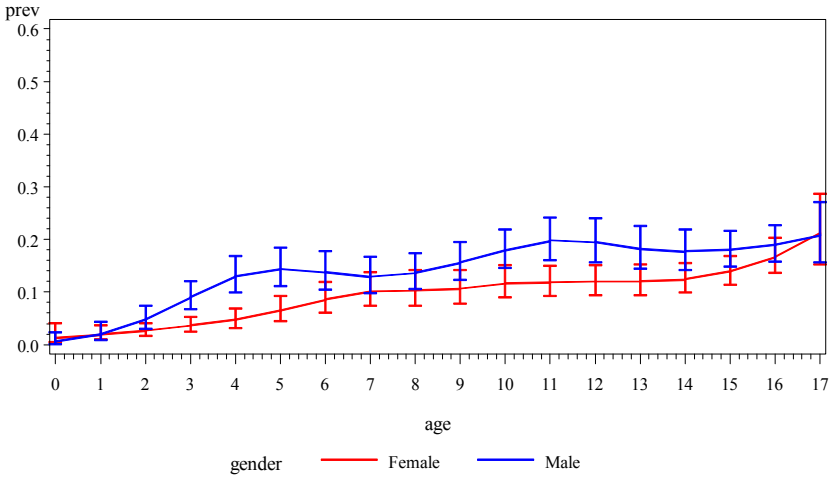


Figure 1. Smoothed asthma 'EVER' prevalence rates and confidence intervals
region=South pov_rat=Below Poverty Level

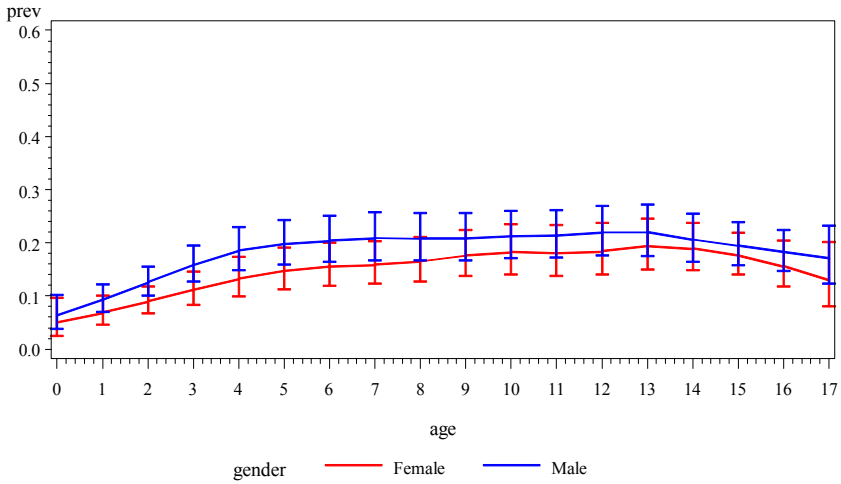
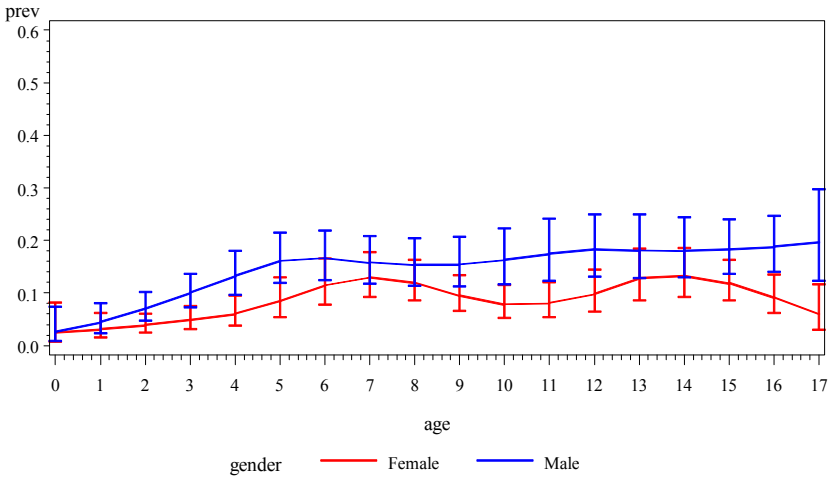


Figure 1. Smoothed asthma 'EVER' prevalence rates and confidence intervals
region=West pov_rat=Below Poverty Level



Appendix 5C, Attachment C, Figure 1, cont. Smoothed prevalence and confidence intervals for children 'EVER' having asthma.

Figure 2. Smoothed asthma 'STILL' prevalence rates and confidence intervals
region=Midwest pov_rat=Above Poverty Level

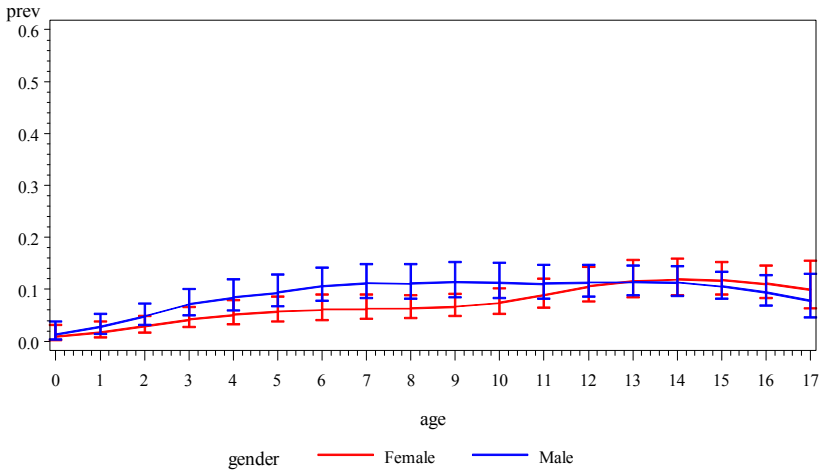


Figure 2. Smoothed asthma 'STILL' prevalence rates and confidence intervals
region=Northeast pov_rat=Above Poverty Level

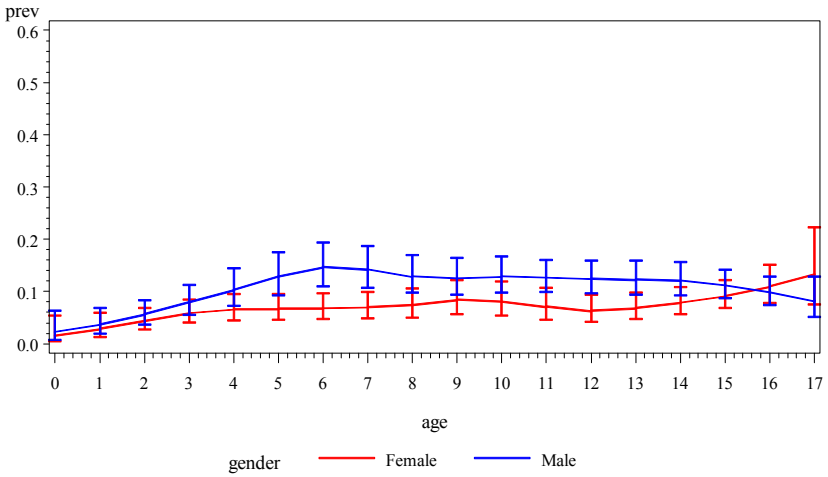


Figure 2. Smoothed asthma 'STILL' prevalence rates and confidence intervals
region=Midwest pov_rat=Below Poverty Level

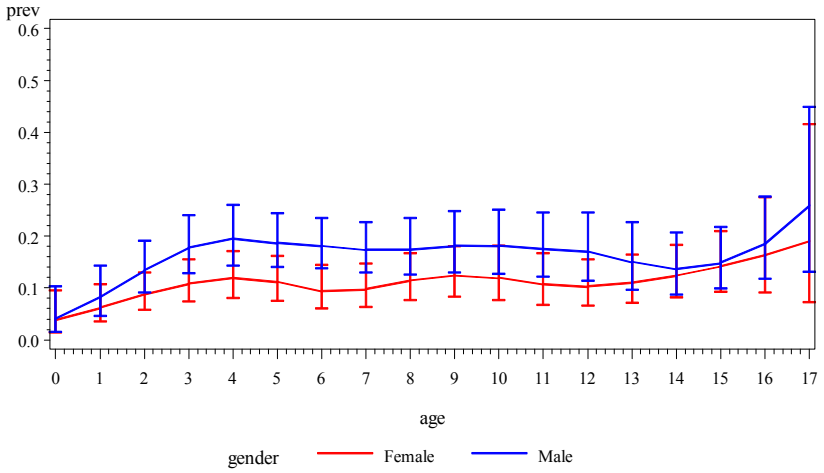
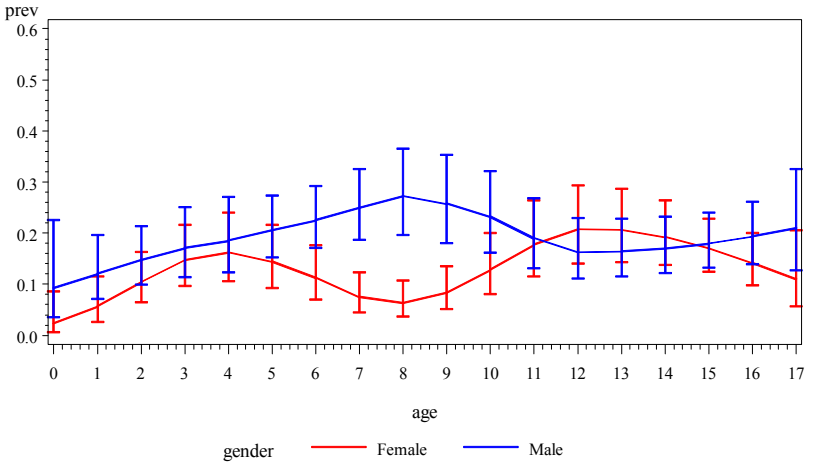


Figure 2. Smoothed asthma 'STILL' prevalence rates and confidence intervals
region=Northeast pov_rat=Below Poverty Level



Appendix 5C, Attachment C, Figure 2. Smoothed prevalence and confidence intervals for children 'STILL' having asthma.

Figure 2. Smoothed asthma 'STILL' prevalence rates and confidence intervals
region=South pov_rat=Above Poverty Level

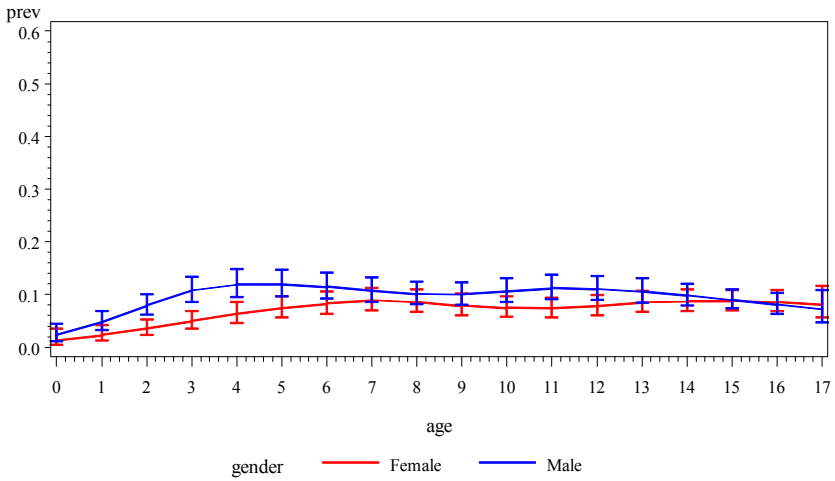


Figure 2. Smoothed asthma 'STILL' prevalence rates and confidence intervals
region=West pov_rat=Above Poverty Level

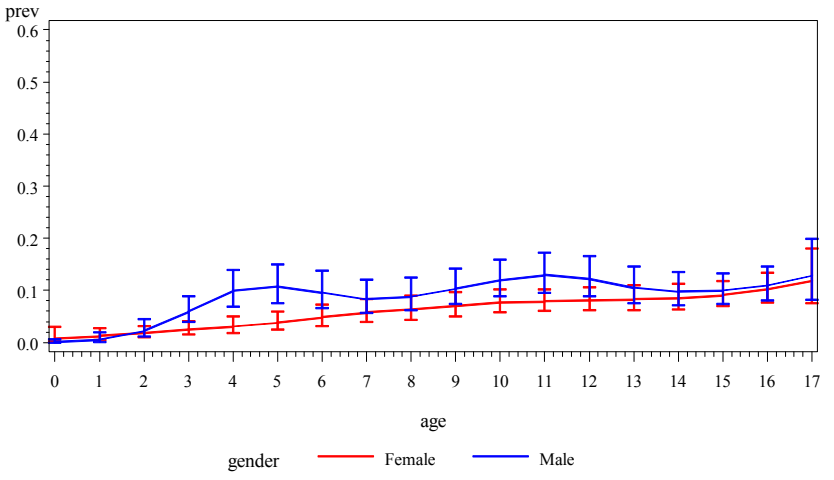


Figure 2. Smoothed asthma 'STILL' prevalence rates and confidence intervals
region=South pov_rat=Below Poverty Level

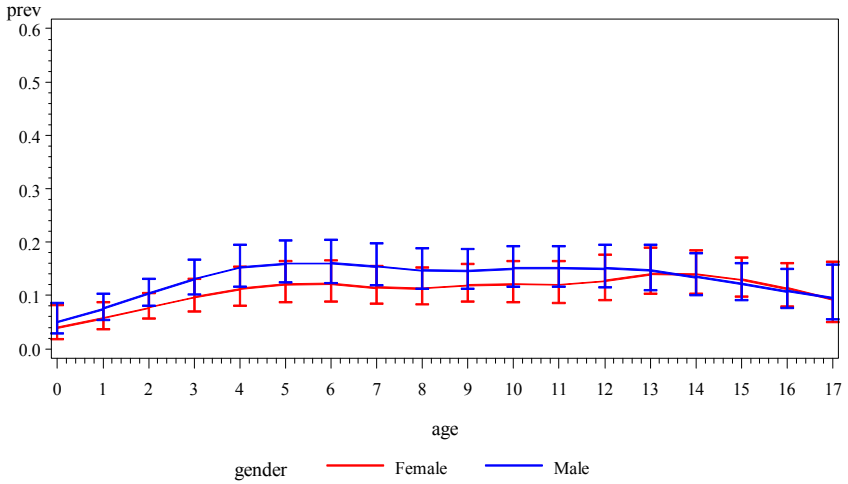
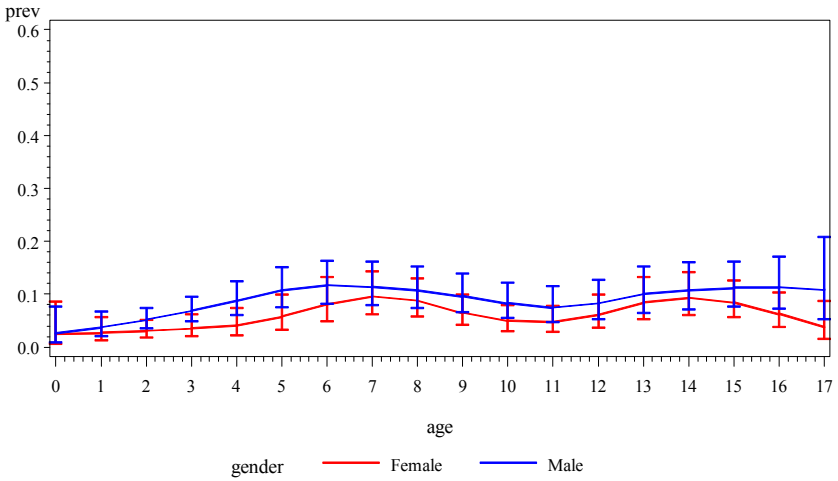


Figure 2. Smoothed asthma 'STILL' prevalence rates and confidence intervals
region=West pov_rat=Below Poverty Level



Appendix 5C, Attachment C, Figure 2, cont. Smoothed prevalence and confidence intervals for children 'STILL' having asthma.

Figure 3. Smoothed adult asthma 'EVER' prevalence rates and confidence intervals
region=Midwest pov_rat=Above Poverty Level

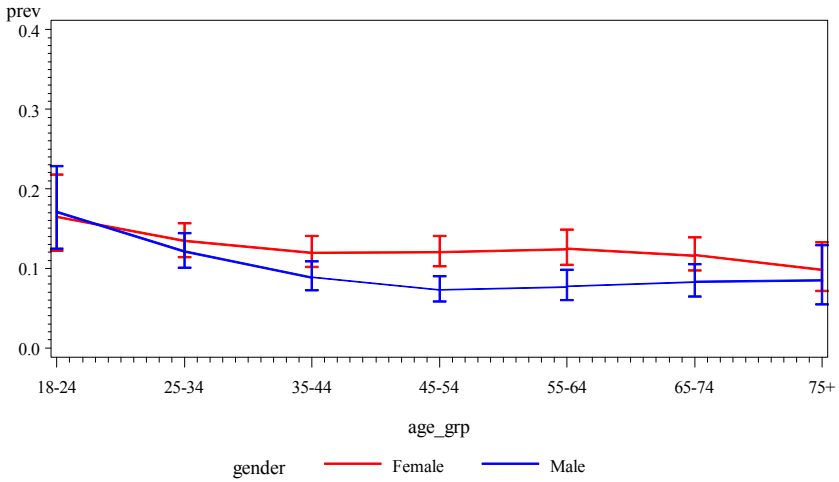


Figure 3. Smoothed adult asthma 'EVER' prevalence rates and confidence intervals
region=Northeast pov_rat=Above Poverty Level

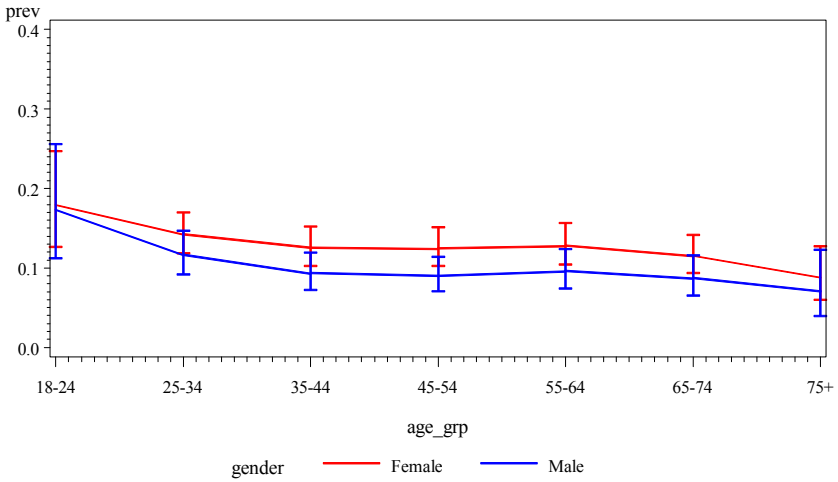


Figure 3. Smoothed adult asthma 'EVER' prevalence rates and confidence intervals
region=Midwest pov_rat=Below Poverty Level

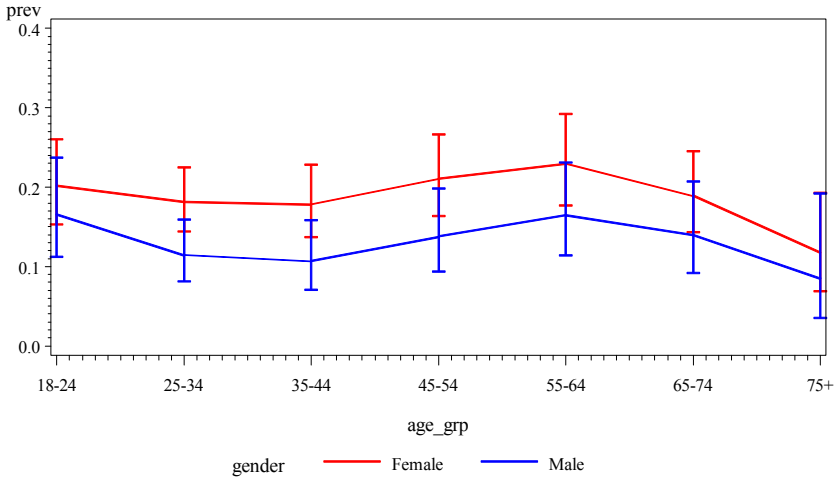
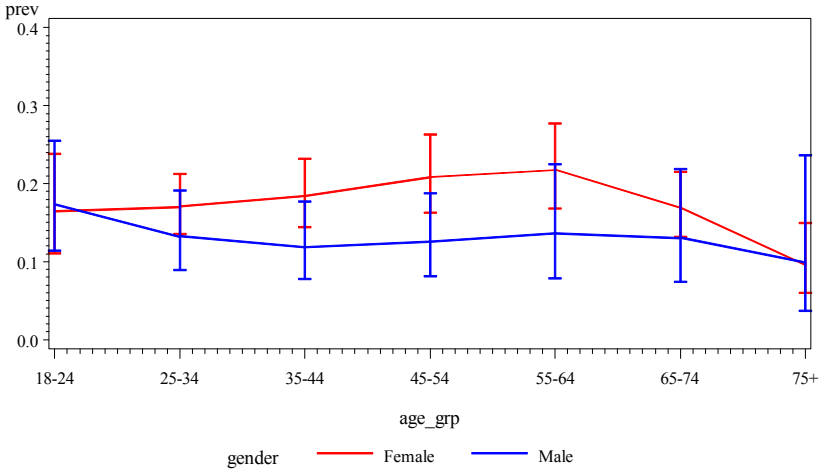


Figure 3. Smoothed adult asthma 'EVER' prevalence rates and confidence intervals
region=Northeast pov_rat=Below Poverty Level



Appendix 5C, Attachment C, Figure 3. Smoothed prevalence and confidence intervals for Adults 'EVER' having asthma.

Figure 3. Smoothed adult asthma 'EVER' prevalence rates and confidence intervals
region=South pov_rat=Above Poverty Level

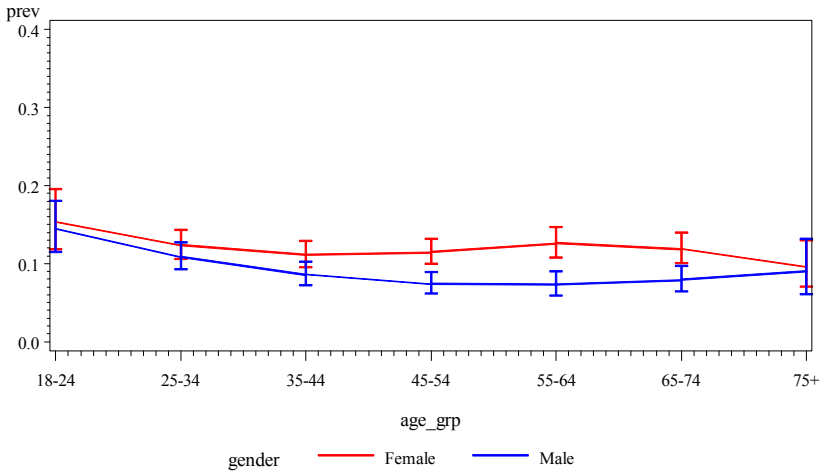


Figure 3. Smoothed adult asthma 'EVER' prevalence rates and confidence intervals
region=West pov_rat=Above Poverty Level

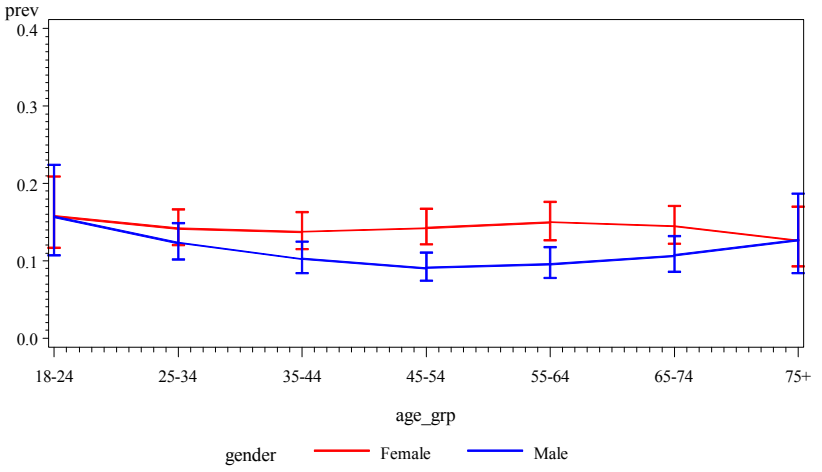


Figure 3. Smoothed adult asthma 'EVER' prevalence rates and confidence intervals
region=South pov_rat=Below Poverty Level

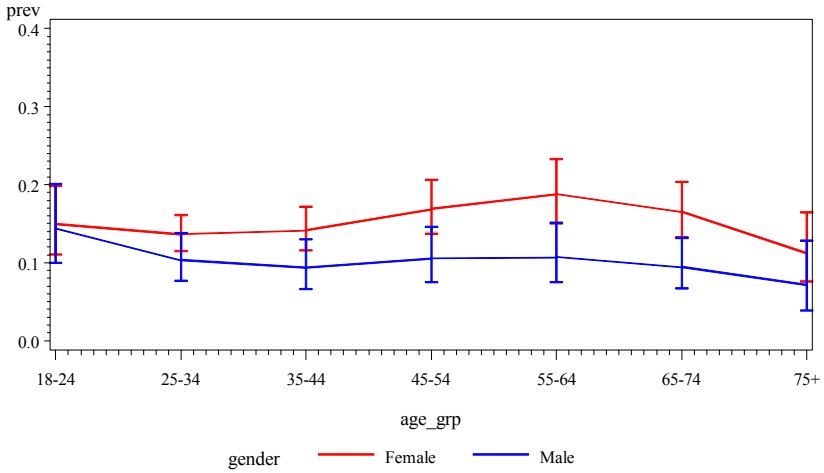
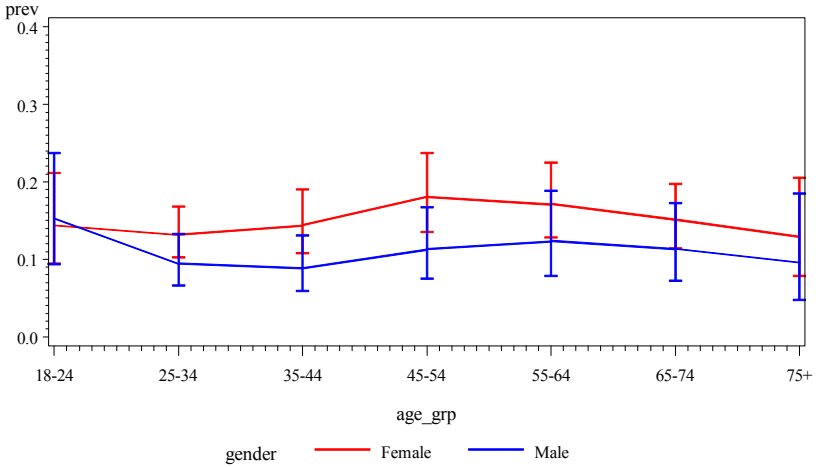


Figure 3. Smoothed adult asthma 'EVER' prevalence rates and confidence intervals
region=West pov_rat=Below Poverty Level



Appendix 5C, Attachment C, Figure 3, cont. Smoothed prevalence and confidence intervals for Adults 'EVER' having asthma.

Figure 4. Smoothed adult asthma 'STILL' prevalence rates and confidence intervals
region=Midwest pov_rat=Above Poverty Level

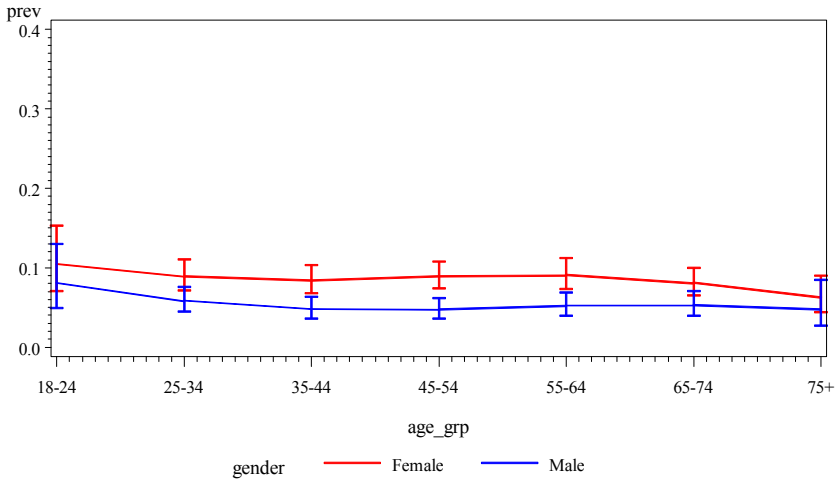


Figure 4. Smoothed adult asthma 'STILL' prevalence rates and confidence intervals
region=Northeast pov_rat=Above Poverty Level

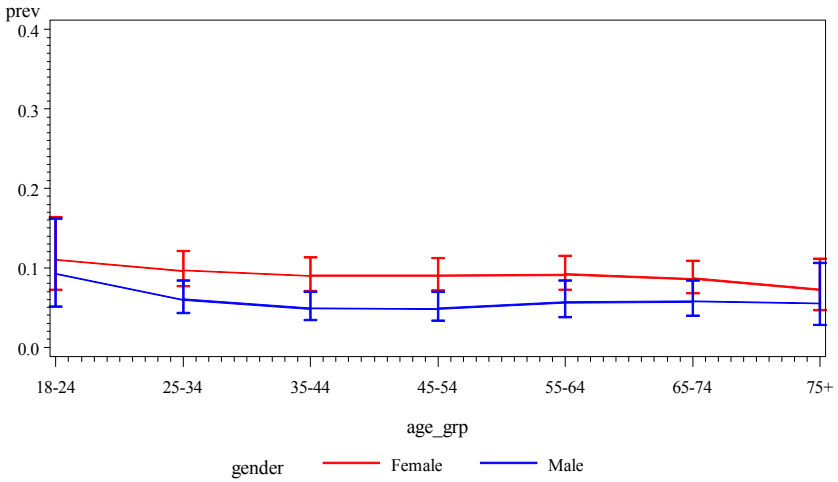


Figure 4. Smoothed adult asthma 'STILL' prevalence rates and confidence intervals
region=Midwest pov_rat=Below Poverty Level

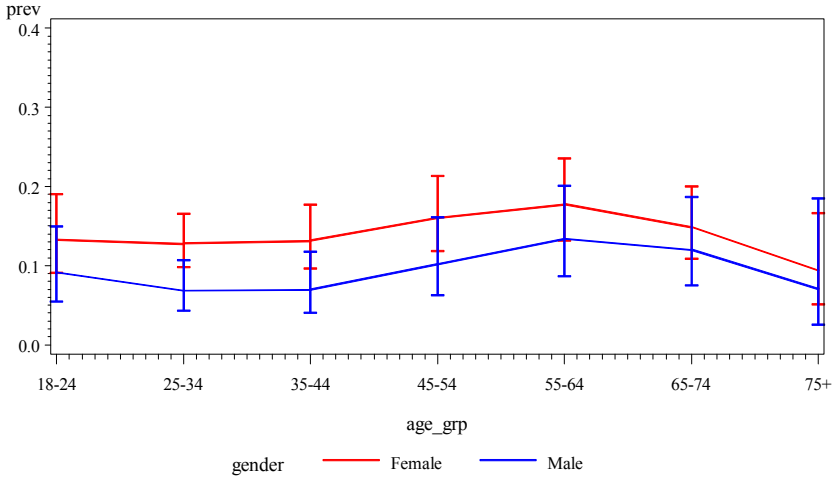
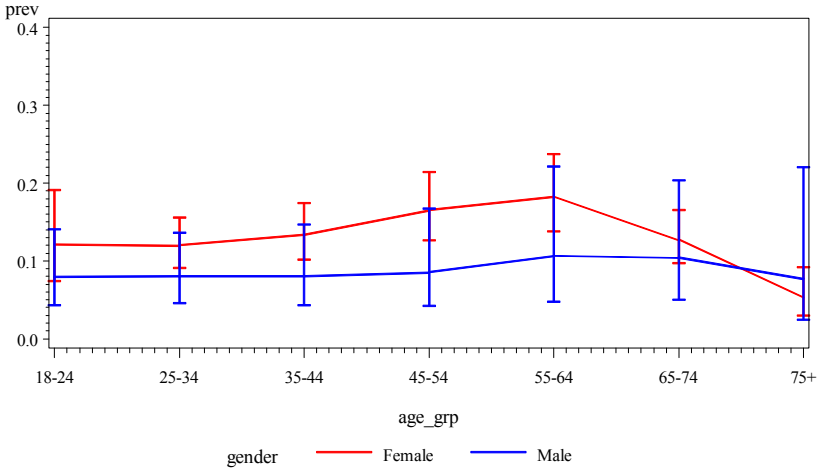


Figure 4. Smoothed adult asthma 'STILL' prevalence rates and confidence intervals
region=Northeast pov_rat=Below Poverty Level



Appendix 5C, Attachment C, Figure 4. Smoothed prevalence and confidence intervals for Adults 'STILL' having asthma.

Figure 4. Smoothed adult asthma 'STILL' prevalence rates and confidence intervals
region=South pov_rat=Above Poverty Level

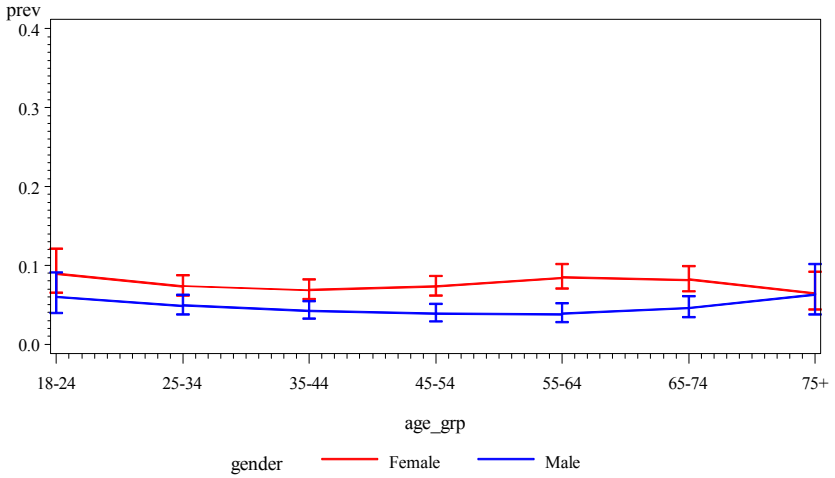


Figure 4. Smoothed adult asthma 'STILL' prevalence rates and confidence intervals
region=West pov_rat=Above Poverty Level

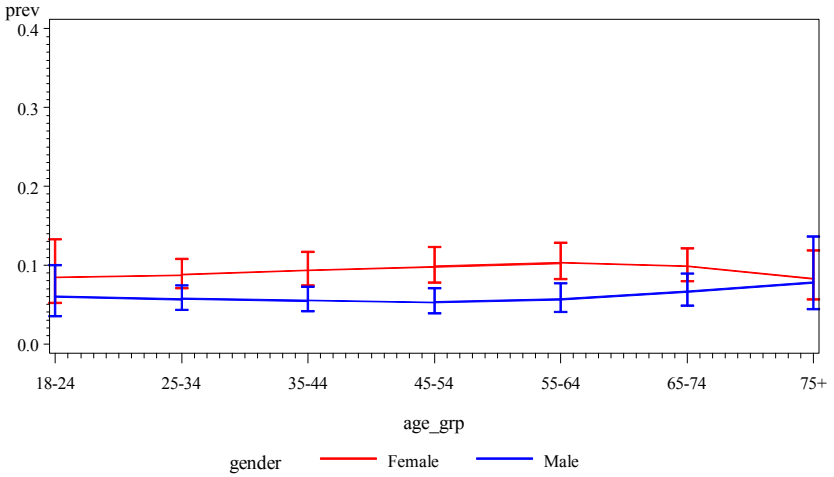


Figure 4. Smoothed adult asthma 'STILL' prevalence rates and confidence intervals
region=South pov_rat=Below Poverty Level

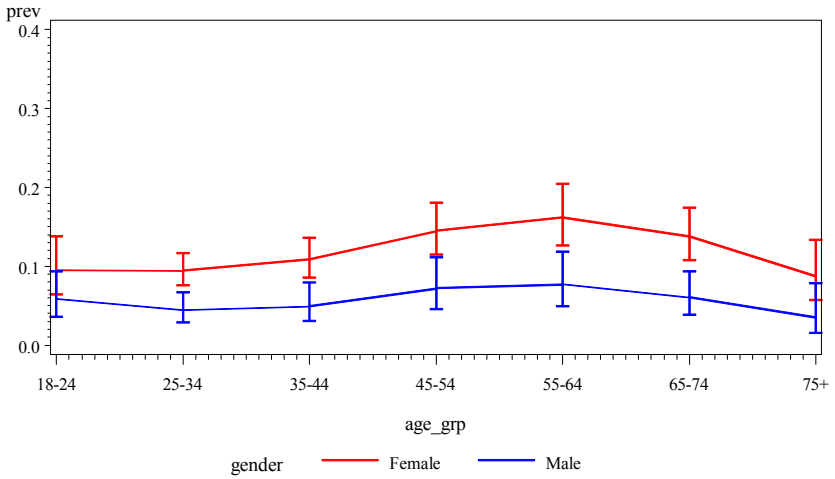
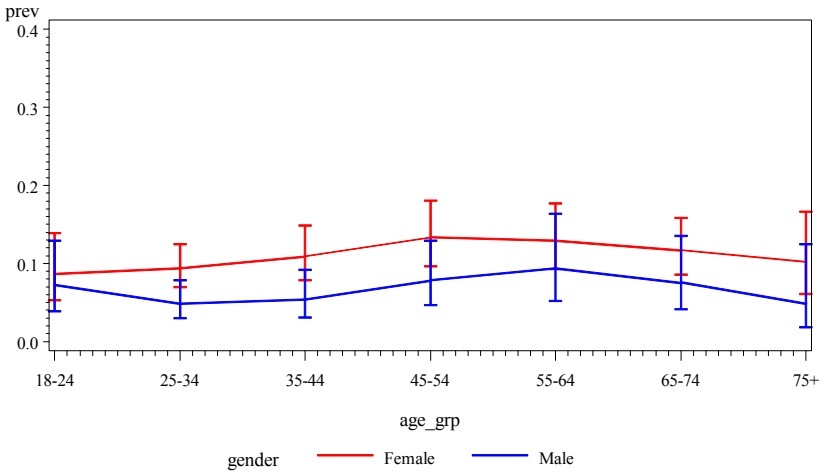


Figure 4. Smoothed adult asthma 'STILL' prevalence rates and confidence intervals
region=West pov_rat=Below Poverty Level



Appendix 5C, Attachment C, Figure 4, cont. Smoothed prevalence and confidence intervals for Adults 'STILL' having asthma.

APPENDIX 5D: VARIABILITY ANALYSIS AND UNCERTAINTY CHARACTERIZATION

5D-1. OVERVIEW

An important issue associated with any population exposure or risk assessment is the characterization of variability and uncertainty. *Variability* refers to the inherent heterogeneity in a population or variable of interest (e.g., residential air exchange rates). The degree of variability cannot be reduced through further research, only better characterized with additional measurement. *Uncertainty* refers to the lack of knowledge regarding the values of model input variables (i.e., *parameter uncertainty*), the physical systems or relationships used (i.e., use of input variables to estimate exposure or risk or *model uncertainty*), and in specifying the scenario that is consistent with purpose of the assessment (i.e., *scenario uncertainty*). Uncertainty is, ideally, reduced to the maximum extent possible through improved measurement of key parameters and iterative model refinement. The approaches used to assess variability and to characterize uncertainty in this REA are discussed in the following two sections. The primary purpose of this characterization is to provide a summary of variability and uncertainty evaluations conducted to date regarding our O_3 exposure assessments and APEX exposure modeling and to identify the most important elements of uncertainty in need of further characterization. Each section contains a concise tabular summary of the identified components and how, for elements of uncertainty, each source may affect the estimated exposures.

5D-2. TREATMENT OF VARIABILITY AND CO-VARIABILITY

The purpose for addressing variability in this REA is to ensure that the estimates of exposure and risk reflect the variability of ambient O_3 concentrations, population characteristics, associated O_3 exposure and intake dose, and potential health risk across the study area and for the simulated at-risk populations. In this REA, there are several algorithms that account for variability of input data when generating the number of estimated benchmark exceedances or health risk outputs. For example, variability may arise from differences in the population residing within census tracts (e.g., age distribution) and the activities that may affect population exposure to O_3 and the resulting intake dose estimate (e.g., time spent outdoors, performing moderate or greater exertion level activities outdoors). A complete range of potential exposure levels and associated risk estimates can be generated when appropriately addressing variability in

exposure and risk assessments; note however that the range of values obtained would be within the constraints of the input parameters, algorithms, or modeling system used, not necessarily the complete range of the true exposure or risk values.

Where possible, staff identified and incorporated the observed variability in input data sets rather than employing standard default assumptions and/or using point estimates to describe model inputs. The details regarding variability distributions used in data inputs are described in Appendix 5B, while details regarding the variability addressed within its algorithms and processes are found in the APEX TSD (US EPA, 2012).

Briefly, APEX has been designed to account for variability in most of the input data, including the physiological variables that are important inputs to determining exertion levels and associated ventilation rates. APEX simulates individuals and then calculates O_3 exposures for each of these simulated individuals. The individuals are selected to represent a random sample from a defined population. The collection of individuals represents the variability of the target population, and accounts for several types of variability, including demographic, physiological, and human behavior. In this assessment, we simulated 200,000 individuals to reasonably capture the variability expected in the population exposure distribution for each study area. APEX incorporates stochastic processes representing the natural variability of personal profile characteristics, activity patterns, and microenvironment parameters. In this way, APEX is able to represent much of the variability in the exposure estimates resulting from the variability of the factors effecting human exposure.

We note also that correlations and non-linear relationships between variables input to the model can result in the model producing incorrect results if the inherent relationships between these variables are not preserved. That is why APEX is also designed to account for co-variability, or linear and nonlinear correlation among the model inputs, provided that enough is known about these relationships to specify them. This is accomplished by providing inputs that enable the correlation to be modeled explicitly within APEX. For example, there is a non-linear relationship between the outdoor temperature and air exchange rate in homes. One factor that contributes to this non-linear relationship is that windows tend to be closed more often when temperatures are at either low or high extremes than when temperatures are moderate. This relationship is explicitly modeled in APEX by specifying different probability distributions of air

exchange rates for different ambient temperatures. In any event, APEX models variability and co-variability in two ways:

- **Stochastically**. The user provides APEX with probability distributions characterizing the variability of many input parameters. These are treated stochastically in the model and the estimated exposure distributions reflect this variability. For example, the rate of O_3 removal in houses can depend on a number of factors which we are not able to explicitly model at this time, due to a lack of data. However, we can specify a distribution of removal rates which reflects observed variations in O_3 decay. APEX randomly samples from this distribution to obtain values which are used in the mass balance model. Further, co-variability can be modeled stochastically through the use of conditional distributions. If two or more parameters are related, conditional distributions that depend on the values of the related parameters are input to APEX. For example, the distribution of air exchange rates (AERs) in a house depends on the outdoor temperature and whether or not air conditioning (A/C) is in use. In this case, a set of AER distributions is provided to APEX for different ranges of temperatures and A/C use, and the selection of the distribution in APEX is driven by the temperature and A/C status at that time. The spatial variability of A/C prevalence is modeled by supplying APEX with A/C prevalence for each Census tract in the modeled area.
- **Explicitly**. For some variables used in modeling exposure, APEX models variability and co-variability explicitly and not stochastically. For example, hourly-average ambient O_3 concentrations and temperatures are used in model calculations. These are input to the model for every hour in the time period modeled at different spatial locations, and in this way the variability and co-variability of hourly concentrations and temperatures are modeled explicitly.

Important sources of the variability and co-variability accounted for by APEX and used for this exposure analysis are summarized in Tables 5D-1 and 5D-2 below, respectively.

Table 5D-1. Components of exposure variability modeled by APEX.

Component	Variability Source	Comment
Simulated Individuals	Population data	Individuals are randomly sampled from US census tracts used in each model study area, stratified by age (single years), gender, and employment status probability distributions (US Census Bureau, 2007a).
	Commuting data	Employed individuals are probabilistically assigned ambient concentrations originating from either their home or work tract based on US Census derived commuter data (US Census Bureau, 2007a).
	Activity patterns	Data diaries are randomly selected from CHAD master (>38,000 diaries) using six diary pools stratified by two day-types (weekday, weekend) and three temperature ranges (< 55.0 °F, between 55.0 and 83.9 °F, and ≥84.0 °F). The CHAD diaries capture real locations that people visit and the activities they perform, ranging from 1 minute to 1 hour in duration (US EPA, 2002).
	Longitudinal profiles	A sequence of diaries is linked together for each individual that preserves both the inter- and intra-personal variability in human activities (Glen et al., 2008).
	Asthma prevalence	Asthma prevalence is stratified by two genders, single age years (0-17), seven age groups, (18-24, 25-34, 35-44, 45-54, 55-64, 65-74, and, ≥75), four regions (Midwest, Northeast, South, and West), and US census tract level poverty ratios (CDC, 2011; US Census Bureau, 2007b).
Ambient Input	Measured ambient O_3 concentrations	Temporal: 1-hour concentrations for an entire O_3 season or year predicted using ambient monitoring data. Spatial: Several monitors are used to represent ambient conditions within each study area; each monitor was assigned a 30 km zone of influence, though value from closest monitor is used for each tract. Four US study areas assess regional differences in ambient conditions.
	Meteorological data	Spatial: Values from closest available local surface National Weather Service (NWS) station were used. Temporal: 1-hour temperature data input for each year; daily values calculated by APEX.
Microenvironmental Approach	Microenvironments: General	Twenty-eight total microenvironments are represented, including those expected to be associated with high exposure concentrations (i.e., outdoors and outdoor near-road). Where this type of variability is incorporated within particular microenvironmental algorithm inputs, this results in differential exposure estimates for each individual (and event) as persons spend varying time frequency within each microenvironment and ambient concentrations vary spatially within and between study areas.
	Microenvironments: Spatial Variability	Ambient concentrations used in microenvironmental algorithms vary spatially within (where more than one site available) and among study areas. Concentrations near roadways are adjusted to account for titration by NO.

Component	Variability Source	Comment
	Microenvironments: Temporal Variability	All exposure calculations are performed at the event-level when using either factors or mass balance approach (durations can be as short as one minute). In addition, for the indoor microenvironments, using a mass balance model accounts for O_3 concentrations occurring during a previous hour (and of ambient origin) to calculate a current event's indoor O_3 concentrations.
	Air exchange rates	Several lognormal distributions are sampled based on five daily mean temperature ranges, study area, and study-area specific A/C prevalence rates.
	Proximity factors for on- and near roads	Three distributions are used, stratified by road-type (urban, interstate, and rural), selected based on VMT to address expected ozone titration by NO near roads.
Physiological Factors and Algorithms	Resting metabolic rate (RMR)	Regression equations for three age-group (18-29, 30-59, and 60+) and two genders were used with body mass as the independent variable (see Johnson et al. (2000) and section 5.3 of APEX TSD).
	Maximum normalized oxygen consumption rate (NVO_2)	Single year age- and gender-specific normal distributions are randomly sampled for each person (Isaacs and Smith, 2005 and section 7.2 of APEX TSD). This variable is used to calculate maximum metabolic equivalents (METS).
	Maximum oxygen debt (MOXD)	Normal distributions for maximum obtainable oxygen, stratified by 3 age groups (ages 0-11, 12-18, 19-100) and two genders (Isaacs and Smith, 2007 and section 7.2 of APEX TSD). Used when adjusting METS to address fatigue and EPOC.
	Recovery time	One uniform distribution randomly sampled to estimate the time required to recover a maximum oxygen deficit (Isaacs and Smith, 2007 and section 7.2 of APEX TSD).
	METS by activity	Values randomly sampled from distributions developed for specific activities (a few are age-group specific) (McCurdy, 2000; US EPA, 2002).
	Oxygen uptake per unit of energy expended (UCF)	Values randomly sampled from a uniform distribution to convert energy expenditure to oxygen consumption (Johnson et al., 2000 and section 5.3 of APEX TSD).
	Body mass	Randomly selected from population-weighted lognormal distributions with age- and gender-specific geometric mean (GM) and geometric standard deviation (GSD) derived from the National Health and Nutrition Examination Survey (NHANES) for the years 1999-2004 (Isaacs and Smith (2005) and section 5.3 of APEX TSD).
	Height	Values randomly sampled from distributions used are based on equations developed for each gender by Johnson (1998) using height and weight data from Brainard and Burmaster (1992) (also see Appendix B of 2010 CO REA).
	Body surface area	Point estimates of exponential parameters used for calculating body surface area as a function of body mass (Burmaster, 1998)

Component	Variability Source	Comment
	Ventilation rate	Event-level activity-specific regression equations stratified by four age groups, using age, gender, body mass normalized oxygen consumption rate as independent variables, and accounting for intra and interpersonal variability (Graham and McCurdy, 2005).
	Fatigue and EPOC	APEX approximates the onset of fatigue, controlling for unrealistic or excessive exercise events in each persons activity time-series while also estimating excess post-exercise oxygen consumption (EPOC) that may occur following vigorous exertion activities (Isaacs et al., 2007 and section 7.2 of APEX TSD).

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93 **Table 5D-2. Important components of co-variability.**

Type of Co-variability	Modeled by APEX?	Treatment in APEX / Comments
Within-person correlations [1]	Yes	Sequence of activities performed, microenvironments visited, and general physiological parameters (body mass, height, ventilation rates).
Between-person correlations	No	Judged as not important.
Correlations between profile variables and microenvironment parameters	Yes	Profiles are assigned microenvironment parameters.
Correlations between demographic variables (e.g., age, gender) and activities	Yes	Age and gender are used in activity diary selection.
Correlations between activities and microenvironment parameters	No	Perhaps important, but do not have data. For example, frequency of opening windows when cooking or smoking tobacco products.
Correlations among microenvironment parameters in the same microenvironment	Yes	Modeled with joint conditional variables.
Correlations between demographic variables and air quality	Yes	Modeled with the spatially varying demographic variables and air quality input to APEX.
Correlations between meteorological variables and activities	Yes	Temperature is used in activity diary selection.
Correlations between meteorological variables and microenvironment parameters	Yes	The distributions of microenvironment parameters can be functions of temperature.
Correlations between drive times in CHAD and commute distances traveled	Yes	CHAD diary selection is weighted by commute times for employed persons during weekdays.
Consistency of occupation/school microenvironmental time and time spent commuting/busing for individuals from one working/school day to the next.	No	Simulated individuals are assigned activity diaries longitudinally without regard to occupation or school schedule (note though, longitudinal variable used to develop annual profile is time spent outdoors).
[1] The term correlation is used to represent linear and nonlinear relationships.		

5D-3. CHARACTERIZATION OF UNCERTAINTY

While it may be possible to capture a range of exposure or risk values by accounting for variability inherent to influential factors, the true exposure or risk for any given individual within a study area is largely unknown. To characterize health risks, exposure and risk assessors commonly use an iterative process of gathering data, developing models, and estimating exposures and risks, given the goals of the assessment, scale of the assessment performed, and limitations of the input data available. However, significant uncertainty often remains and emphasis is then placed on characterizing the nature of that uncertainty and its impact on exposure and risk estimates.

In the final 2008 O_3 NAAQS rule,[1] EPA staff performed such a characterization and at that time, identified the most important uncertainties affecting the exposure estimates. The key elements of uncertainty were 1) the modeling of human activity patterns over an O_3 season, 2) the modeling of variations in ambient O_3 concentrations near roadways, 3) the modeling of air exchange rates that affect the amount of O_3 that penetrates indoors, and 4) the characterization of energy expenditure (and related ventilation rate estimates) for children engaged in various activities. Further, the primary findings of a quantitative Monte Carlo analysis also performed at that time indicated that the overall uncertainty of the APEX estimated exposure distributions was relatively small: the percent of children or asthmatic children with exposures above 0.06, 0.07, or 0.08 ppm-8hr under moderate exertion have 95% were estimated by APEX to have uncertainty intervals of at most ±6 percentage points. Details for these previously identified uncertainties are discussed in the 2007 O_3 Staff Paper (section 4.6) and in a technical memorandum describing the 2007 O_3 exposure modeling uncertainty analysis (Langstaff, 2007).

The REA's conducted for the most recent NO_2 (US EPA, 2008), SO_2 (US EPA, 2009), and CO (US EPA, 2010) NAAQS reviews also presented characterizations of the uncertainties associated with APEX exposure modeling (among other pollutant specific issues), albeit mainly qualitative evaluations. Conclusions drawn from all of these assessments regarding exposure modeling uncertainty have been integrated here, following the standard approach used by EPA staff since 2008 and outlined by WHO (2008) to identify, evaluate, and prioritize the most important uncertainties relevant to the estimated potential health effect endpoints used in this O_3

[1] Federal Register Vol. 73, No. 60. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/fr/20080327.pdf

REA. Staff selected the qualitative approach used for this first draft O_3 REA as a step towards developing an appropriate probabilistic uncertainty analysis, perhaps similar to that performed at the time of the 2007 O_3 REA by Langstaff (2007).

The qualitative approach used in this first draft O_3 REA varies from that described by WHO (2008) in that a greater focus was placed on evaluating the direction and the magnitude[2] of the uncertainty; that is, qualitatively rating how the source of uncertainty, in the presence of alternative information, may affect the estimated exposures and health risk results. In addition and consistent with the WHO (2008) guidance, staff discuss the uncertainty in the knowledge base (e.g., the accuracy of the data used, acknowledgement of data gaps) and decisions made where possible (e.g., selection of particular model forms), although qualitative ratings were assigned only to uncertainty regarding the knowledge base.

First, staff identified the key aspects of the assessment approach that may contribute to uncertainty in the exposure and risk estimates and provided the rationale for their inclusion. Then, staff characterized the *magnitude* and *direction* of the influence on the assessment results for each of these identified sources of uncertainty. Consistent with the WHO (2008) guidance, staff subjectively scaled the overall impact of the uncertainty by considering the degree of uncertainty as implied by the relationship between the source of uncertainty and the exposure concentrations.

Where the magnitude of uncertainty was rated *low*, it was judged that changes within the source of uncertainty would have only a small effect on the exposure results. For example, we have commonly employed statistical procedure to substitute missing concentration values to complete the APEX ambient input data sets. Staff has consistently compared the air quality distributions and found negligible differences between the substituted data set and the one with missing values (e.g., Tables 5-13 through 5-16 of US EPA, 2010), primarily because of the infrequency of missing value substitutions needed to complete a data set. There is still uncertainty in the approach used, and there may be alternative, and possibly better, methods available to perform such a task. However, in this instance, staff judged that the quantitative comparison of the ambient concentration data sets indicates that there would likely be little influence on exposure estimates by the data substitution procedure used.

[2] This is synonymous with the "level of uncertainty" discussed in WHO (2008), section 5.1.2.2.

A magnitude designation of *moderate* implies that a change within the source of uncertainty would likely have a moderate (or proportional) effect on the results. For example, the magnitude of uncertainty associated with using the quadratic approach to represent a hypothetical future air quality scenario was rated as *low-moderate*. While we do not have information regarding how the ambient O_3 concentration distribution might look in the future, we do know however what the distribution might look like based on historical trends and the emission sources. These historical data and trends serve to generate algorithms used to adjust air quality. If these trends in observed concentrations and emissions were to remain constant in the future, then the magnitude of the impact to estimated exposures in this assessment would be judged as likely *low* or having negligible impact on the estimated exposures. However, if there are entirely new emission sources in the future or if the approach developed is not equally appropriate across the range of assessed study areas, the magnitude of influence might be judged as greater. For example, when comparing exposure estimates for one year that used three different 3-year periods to adjust that year's air quality levels to just meet the current standard, staff observed mainly proportional differences (e.g., a factor of two or three) in the estimated number of persons exposed in more than half of the twelve study areas (Langstaff, 2007). Assuming that these types of ambient concentration adjustments could reflect the addition of a new or unaccounted for emission source in a particular study area, staff also judged the magnitude of influence in using the quadratic approach to adjust air quality data to represent a hypothetical future scenario as *moderate*. A characterization of *high* implies that a small change in the source would have a large affect on results, potentially an order of magnitude or more. This rating would be used where the model estimates were extremely sensitive to the identified source of uncertainty.

In addition to characterizing the magnitude of uncertainty, staff also included the direction of influence, indicating how the source of uncertainty was judged to affect estimated exposures or risk estimates; either the estimated values were possibly *over-* or *under-estimated*. In the instance where the component of uncertainty can affect the assessment endpoint in either direction, the influence was judged as *both*. Staff characterized the direction of influence as *unknown* when there was no evidence available to judge the directional nature of uncertainty associated with the particular source. Staff also subjectively scaled the knowledge-base uncertainty associated with each identified source using a three-level scale: *low* indicated

significant confidence in the data used and its applicability to the assessment endpoints, *moderate* implied that there were some limitations regarding consistency and completeness of the data used or scientific evidence presented, and *high* indicated the extent of the knowledge-base was extremely limited.

The output of the uncertainty characterization is a summary describing, for each identified source of uncertainty, the magnitude of the impact and the direction of influence the uncertainty may have on the exposure and risk characterization results. At this point we have identified a total of 28 sources of uncertainty associated with our approach to model O_3 population exposure, each broadly summarized in Table 5D-3, including newly identified elements. We then judged whether these results from our historical characterizations were an appropriate characterization of the elements within our current exposure assessment, while also, considering our new analysis of the attributes contributing to those persons highly exposed. The most influential elements of uncertainty in need of further investigation are:

- Activity Patterns
  - In general, with a focus on representation of time spent outdoors
  - Longitudinal Activity Profiles (e.g., investigation of alternative approaches and assignment of more rigid schedules)
- Spatial Variability in O_3 Concentrations (as the outdoor microenvironment is the most important determinant for 8-hour exposure benchmark exceedances, most elements should be systematically re-evaluated)
- Physiological Processes
  - Metabolic equivalents (METs) distributions (updated information availability, short-term activity evaluations)
  - Ventilation rate equations

Newly identified elements would also be a part of this new uncertainty characterization in future drafts. These include:

- The new modeling approach used to simulate ambient air quality that just meets the current standard (if done for future next drafts)
- Poverty Status (US Census) Weighted Asthma Prevalence (CDC)
- Commuting (CHAD drive times linked with Census commute distances)
- Resting Metabolic Rate (RMR) equations

214 • At-risk population (effect of averting behavior on activity pattern data)

Table 5D-3. Characterization of key uncertainties in historical and current APEX exposure assessments.

Sources of Uncertainty		Historical Uncertainty Characterization				Is rating appropriate for current APEX O_3 exposure assessment?
		Influence of Uncertainty on Exposure/Intake Dose Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
Ambient Monitoring Concentrations	Database Quality	Over	Low	Low	All ambient pollutant measurements available from AQS are both comprehensive and subject to quality control.	Yes. No further characterization needed.
	Instrument Measurement Error	Over	Low	Low	Mean bias estimated as 1.2% (CV of 4.4%). See Table 2 and Figure 6 of Langstaff (2007).	Yes. No further characterization needed.
	Missing Data Substitution Method	Both	Low	Low	Overall completeness of data yield negligible mean bias (~0) along with an estimated standard deviation of 4 ppb when replacing missing values. See Table 3 of Langstaff (2007).	Yes. No further characterization needed.
	Temporal Representation	Both	Low	Low	Appropriately uses 1-hour time-series of O_3 concentrations for 5 years.	Yes. No further characterization needed.
	Spatial Representation: Large Scale	Both	Low	Low	Tens of monitors used in each study area.	Yes. No further characterization needed.
	Spatial Representation: Neighborhood Scale (1)	Both	Low	Low	Spatial interpolation using jackknife method (removal of a single monitor) yielded generally unbiased observed/predicted ratios (mean 1.06), having an estimated standard deviation of 0.2. Langstaff (2007).	Yes. For the uncertainties characterized, the historical rating is appropriate. However local-

Sources of Uncertainty		Historical Uncertainty Characterization				Is rating appropriate for current APEX O_3 exposure assessment?
		Influence of Uncertainty on Exposure/Intake Dose Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Spatial Representation: Neighborhood Scale (2)	Over	Low	Low	When reducing the APEX radius setting from an unlimited value (actual value used) to 10 km (i.e., the tendency would be to more accurately represent exposure), a smaller fraction (1-3 percentage points) of population exceeds benchmark levels. See Figures 7 – 9 of Langstaff (2007).	scale spatial representation (not characterized) may result in a different characterization.
	Spatial Representation: Vertical Profile	Both	Moderate	Moderate	Differences between ground-level (0-3 meters) and building rooftop sited (25 meters) monitor concentrations can be significant. Most importantly, use of higher elevation monitors would tend to overestimate ground-level exposures (i.e., persons outdoors).	Yes. Given judged impact to exposure, additional characterization is needed.
Adjustment of Air Quality to Simulate Just Meeting the Current Standard	Quadratic Approach	Both	Low - Moderate	Moderate	Variable differences (e.g., none to a factor of two or three) in the estimated number of persons exposed across study areas when using differing 3-year roll-back periods for a single year of air quality (Langstaff, 2007).	Yes. Uncertainty in the approach has resulted in plans to use alternative approach.
	New Model Simulation Approach	nc	nc	nc	New approach developed for this REA, newly identified, not evaluated.	New. Needs characterization.
APEX: General Input Databases	Population Demographics and Commuting (US Census)	Under	Low	Low	Comprehensive and subject to quality control. Differences in 2000 versus modeled years (2006-10) likely small when estimating percent of population exposed.	Yes. No further characterization needed.

Sources of Uncertainty		Historical Uncertainty Characterization				Is rating appropriate for current APEX O_3 exposure assessment?
		Influence of Uncertainty on Exposure/Intake Dose Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Activity Patterns (CHAD)	Unknown	Low - Moderate	Moderate	Comprehensive and subject to quality control. However, comprised of multiple studies, varying survey techniques, historical data, broad location/activity code assignments, among other issues, add to difficulties in assessing uncertainties.	Yes. Given judged impact to exposure, additional characterization is needed.
	Meteorological (NWS)	Both	Low	Low	Comprehensive and subject to quality control, few missing values. Limited application in selecting CHAD diaries and AERs.	Yes. No further characterization needed.
	Poverty Status (US Census) Weighted Asthma Prevalence (CDC)	nc	nc	nc	New data set generated for this REA, newly identified, not evaluated.	New. Needs characterization.
APEX: Microenvironmental Concentrations	Outdoor Near-Road and Vehicular: Proximity Factors	Both	Low	Low-Moderate	Uncertainty in mean value used approximated as 15 percentage points. See Figure 10 and Table 7 of Langstaff (2007). May be of greater importance in certain study areas.	Yes. No further characterization needed.
	Indoor: Near-Road	Over	Low	Low	Expected reduction in O_3 for persons residing near roads not modeled here, but when included, there is a small reduction (~3%) in the number of persons experiencing exposure above benchmark levels (Langstaff, 2007).	Yes. No further characterization needed.

Sources of Uncertainty		Historical Uncertainty Characterization				Is rating appropriate for current APEX O_3 exposure assessment?
		Influence of Uncertainty on Exposure/Intake Dose Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Indoor: Air Exchange Rates	Both	Low	Moderate	Uncertainty due to random sampling variation via bootstrap distribution analysis indicated the AER GM and GSD uncertainty for a given study area tends range to at most from fitted ±1.0 GM and ± 0.5 GSD hr^{-1}. Non-representativeness remains an important issue as city-to-city variability can be wide ranging (GM/GSD pairs can vary by factors of 2-3) and data available for city-specific evaluation are limited (US EPA, 2007). Also, indoor exposures are estimated as not important to 8-hour average daily maximum O_3 exposure.	Yes. No further characterization needed.
	Indoor: A/C Prevalence (AHS)	Both	Low	Low	Comprehensive and subject to quality control, estimated 95^{th} percentile confidence bounds range from a few to just over ten percentage points, though some cities use older year data (Table 9 of Langstaff, 2007). Note, variable indicates presence/absence not actual use. Also, indoor exposures are estimated here as limited in importance to 8-hour average daily maximum exposures and sensitivity analyses in NO2 REA (in-vehicle was most influential exposure ME) concluded prevalence variable was of limited importance.	Yes. No further characterization needed.

Sources of Uncertainty		Historical Uncertainty Characterization				Is rating appropriate for current APEX O_3 exposure assessment?
		Influence of Uncertainty on Exposure/Intake Dose Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Indoor: Removal Rate	Both	Low	Low	Greatest uncertainty in the input distribution regarded representativeness, though estimated as unbiased but correct to within 10%.	Yes. No further characterization needed.
	Vehicular: Penetration Factors	Both	Low	Moderate	Input distribution is from an older measurement study though consistent with recent, albeit limited data.	Yes. No further characterization needed.
APEX: Simulated Activity Profiles	Longitudinal Profiles	Under	Low - Moderate	Moderate	Depending on the longitudinal profile method selected, the number of persons experiencing multiple exposure events at or above a selected level could differ by about 15 to 50% (see Appendix B, Attachment 4 of NO2 REA). Long-term diary profiles (i.e., monthly, annual) do not exist for a population, limiting the evaluation. Modeling does not assign rigid schedules for workers or children attending school.	Yes. Given judged impact to exposure, additional characterization is needed.
	Commuting	nc	nc	nc	New method used in this assessment designed to link Census commute distances with CHAD vehicle drive times, newly identified, not evaluated. Note while vehicle time accounted for through diary selection, not rigidly scheduled.	New. Needs evaluation

Sources of Uncertainty		Historical Uncertainty Characterization				Is rating appropriate for current APEX O_3 exposure assessment?
		Influence of Uncertainty on Exposure/Intake Dose Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	At-Risk Population	Both	Low	Low – Moderate	Asthmatics activity patterns are similar to that of non-asthmatics (both types of diaries are used in our simulations, regardless of health status). See discussion in SO2 REA (section 8.11.2.2.5).	Yes. For the uncertainties characterized, the historical rating is appropriate. However, averting behavior (where present in input data and currently undesignated) may result in a different characterization.
APEX: Physiological Processes	Body Mass (NHANES)	Unknown	Low	Low	Comprehensive and subject to quality control, though older (1999-2004) than current simulated population, possible small regional variation is not represented by national data.	Yes. No further characterization needed.
	NVO2max	Unknown	Low	Low	Upper bound control for unrealistic activity levels rarely used by model, thus likely not very influential.	Yes. No further characterization needed.
	RMR	nc	nc	nc	Approach from older literature (Schofield, 1985), linked to estimated ventilation rates, not previously evaluated.	New. Needs characterization.
	METS distributions	Over	Low - Moderate	Low - Moderate	APEX estimated daily mean METs range from about 0.1 to 0.2 units (between about 5-10%) higher than independent literature reported values (Table 15 of Langstaff, 2007). Shorter-term values are of greater importance in this assessment.	Yes. Given judged impact to exposure, additional characterization is needed

Sources of Uncertainty		Historical Uncertainty Characterization				Is rating appropriate for current APEX O_3 exposure assessment?
		Influence of Uncertainty on Exposure/Intake Dose Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Ventilation rates	Over	Low - Moderate	Low - Moderate	APEX estimated daily ventilation rates can be greater (2-3 m3/day) than literature reported measurement values (Table 25 of Langstaff, 2007), though accounting for measurement bias minimizes the discrepancy (Graham and McCurdy, 2005). Also, a shorter-term comparison (for hours rather than daily), while more informative, is lacking due to limited data.	Yes. Given judged impact to exposure, additional characterization is needed.

5D-4. REFERENCES

Brainard J and Burmaster D. (1992). Bivariate distributions for height and weight of men and women in the United States. *Risk Analysis*. 12(2):267-275.

Burmaster DE. (1998). Lognormal distributions for skin area as a function of body weight. *Risk Analysis*. 18(1):27-32.

CDC. (2011). Summary Health Statistics for U.S. Adults: National Health Interview Survey, years 2006-10. U.S. Department of Health and Human Services, Hyattsville, MD. Data and documentation available at: http://www.cdc.gov/nchs/nhis.htm (accessed October 4, 2011).

Glen G, Smith L, Isaacs K, McCurdy T, Langstaff J. (2008). A new method of longitudinal diary assembly for human exposure modeling. *J Expos Sci Environ Epidem*. 18:299-311.

Graham SE and T McCurdy. (2005). Revised ventilation rate (VE) equations for use in inhalation-oriented exposure models. Report no. EPA/600/X-05/008. Report is found within Appendix A of US EPA (2009). Metabolically Derived Human Ventilation Rates: A Revised Approach Based Upon Oxygen Consumption Rates (Final Report). Report no. EPA/600/R-06/129F. Appendix D contains "Response to peer-review comments on Appendix A", prepared by S. Graham (US EPA). Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=202543

Issacs K and Smith L. (2005). New Values for Physiological Parameters for the Exposure Model Input File Physiology.txt. Technical memorandum to Tom McCurdy, NERL WA10. December 20, 2005. Provided in Appendix A of the CO REA (US EPA, 2010).

Isaacs K, Glen G, McCurdy T., and Smith L. 2007. Modeling energy expenditure and oxygen consumption in human exposure models: Accounting for fatigue and EPOC. *J Expos Sci Environ Epidemiol*. 18(3):289-98.

Johnson T. (1998). Memo No. 5: Equations for Converting Weight to Height Proposed for the 1998 Version of pNEM/CO. Memorandum Submitted to U.S. Environmental Protection Agency. TRJ Environmental, Inc., 713 Shadylawn Road, Chapel Hill, North Carolina 27514.

Johnson T, Mihlan G, LaPointe J, Fletcher K, Capel J, Rosenbaum A, Cohen J, Stiefer P. (2000). Estimation of carbon monoxide exposures and associated carboxyhemoglobin levels for residents of Denver and Los Angeles using pNEM/CO. Appendices. EPA constract 68-D6-0064.

Langstaff JE. (2007). OAQPS Staff Memorandum to Ozone NAAQS Review Docket (OAR-2005-0172). Subject: Analysis of Uncertainty in Ozone Population Exposure Modeling. [January 31, 2007]. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html

McCurdy T. (2000). Conceptual basis for multi-route intake dose modeling using an energy expenditure approach. *J Expos Anal Environ Epidemiol*. 10:1-12.

Schofield WN. (1985). Predicting basal metabolic rate, new standards, and review of previous work. *Hum Nutr Clin Nutr*. 39C(S1):5-41.

US Census Bureau. (2007a). Employment Status: 2000- Supplemental Tables. Available at: http://www.census.gov/population/www/cen2000/phc-t28.html.

US Census Bureau. (2007b). 2000 Census of Population and Housing. Summary File 3 (SF3) Technical Documentation, available at: http://www.census.gov/prod/cen2000/doc/sf3.pdf. Individual SF3 files '30'

(for income/poverty variables pct49-pct51) for each state were downloaded from: http://www2.census.gov/census_2000/datasets/Summary_File_3/.

US EPA. (2002). EPA's Consolidated Human Activities Database. Available at: http://www.epa.gov/chad/.

US EPA. (2007). Ozone Population Exposure Analysis for Selected Urban Areas. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. Available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html

US EPA. (2008). Risk and Exposure Assessment to Support the Review of the NO_2 Primary National Ambient Air Quality Standard. Report no. EPA-452/R-08-008a. November 2008. Available at: http://www.epa.gov/ttn/naaqs/standards/nox/data/20081121_NO2_REA_final.pdf.

US EPA. (2009). Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standard. Report no. EPA-452/R-09-007. August 2009. Available athttp://www.epa.gov/ttn/naaqs/standards/so2/data/200908SO2REAFinalReport.pdf.

US EPA. (2010). Quantitative Risk and Exposure Assessment for Carbon Monoxide – Amended. EPA Office of Air Quality Planning and Standards. EPA-452/R-10-009. July 2010. Available at: http://www.epa.gov/ttn/naaqs/standards/co/data/CO-REA-Amended-July2010.pdf

US EPA. (2012). Total Risk Integrated Methodology (TRIM) - Air Pollutants Exposure Model Documentation (TRIM.Expo / APEX, Version 4.4) Volume I: User's Guide. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA-452/B-12-001a. Available at: http://www.epa.gov/ttn/fera/human_apex.html

WHO. (2008). Harmonization Project Document No. 6. Part 1: Guidance document on characterizing and communicating uncertainty in exposure assessment. Available at: http://www.who.int/ipcs/methods/harmonization/areas/exposure/en/.

APPENDIX 7A.
INPUT DATA USED IN MODELING RISK FOR THE 12 URBAN STUDY AREAS

This appendix presents data used in modeling risk for the 12 urban study areas (in Table 7A-1). In some cases (as noted below) data are presented in an aggregated fashion. If the reader would like the dis-aggregated data, they can consult the original data sources cited in the relevant sections of Chapter 7. Table 7A-1 is organized by health endpoint. The specific types of data provided for each endpoint are described below (note, only those data fields requiring additional clarification are described here, many are self explanatory).

- *Study information (C-R function):* these fields provide information on the C-R functions used in modeling endpoints covered in the risk assessment including (a) ozone metric and risk modeling period, (b) age range of the population modeled, the effect estimate (including statistical fit information), the model form and additional details related to the model (e.g., lag structure, copollutants control if relevant) (see section 7.3.2).

- *Baseline incidence*: annual incidence per 100,000 general population for the specific risk period modeled for that health endpoint in the risk assessment (i.e., these are not annual values, but rather incidence rates for the risk modeling period). Incidence rates are provided for both simulation years (2007 and 2009) (see section 7.3.4).

- *Population:* count of individuals matched to the population being modeled for the particular health endpoint (provided for 2007 and 2009 – see section 7.3.5).

- *Surrogate LMLs*: These are the LMLs obtained from the composite monitor distributions used to model each health endpoint (for a given urban study area and simulation year) (see section 7.3.3).

Table 7A-1 Selected Model Inputs Used in Generating Risk Estimates for the First Draft REA

			Study information (C-R function)								Baseline incidence[b]		Population		Surrogate LMLs (ppb)	
Endpoint	Study	Urban study area	Air metric	Risk assessment modeling period	Age range	Lag	Additional study details	Statistical Model	Effect estimate (Beta)	SE (effect estimate)[a]	2007	2009	2007	2009	2007	2009
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Atlanta, GA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0002954	0.0002886	138	134	3,856,357	3,972,395	24	21
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Baltimore, MD	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.000515	0.000314	219	211	2,146,632	2,194,116	13	24
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Boston, MA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0006816	0.0003284	190	183	4,021,878	4,048,879	19	17
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Cleveland, OH	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0005962	0.0003546	267	256	1,328,261	1,317,928	6	16
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Denver, CO	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0003518	0.0004088	183	173	558,817	559,791	21	22
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Detroit, MI	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0010459	0.0003441	230	218	1,986,360	1,969,826	19	11
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Houston, TX	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0001629	0.0002628	140	135	3,780,576	3,850,328	10	15
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Los Angeles, CA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0002737	0.0002134	152	148	9,981,639	10,042,327	31	22
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	New York, NY	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0010925	0.0002357	181	174	11,043,330	11,108,750	10	12
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Philadelphia, PA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0006246	0.0003146	264	250	1,456,148	1,444,164	12	14
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	Sacramento, CA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0005691	0.0003885	182	175	1,405,744	1,453,703	30	30
Mortality, All Cause	Zanobetti and Schwartz (b), 2008	St. Louis, MO	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0005444	0.0003334	213	204	2,198,242	2,211,259	22	22
Mortality, Non-Accidental	Bell et al., 2004	Atlanta, GA	D8HourMax	March-October (8)	0-99	distributed lag 0-6 d	-	log-linear	0.0007053	0.0002252	332	324	3,856,357	3,972,395	17	5
Mortality, Non-Accidental	Bell et al., 2004	Baltimore, MD	D8HourMax	April-October (7)	0-99	distributed lag 0-6 d	-	log-linear	0.0003428	0.0002539	474	456	2,146,632	2,194,116	13	9
Mortality, Non-Accidental	Bell et al., 2004	Boston, MA	D8HourMax	April-September (6)	0-99	distributed lag 0-6 d	-	log-linear	0.0006887	0.000267	360	347	4,021,878	4,048,879	12	12
Mortality, Non-Accidental	Bell et al., 2004	Cleveland, OH	D8HourMax	April-October (7)	0-99	distributed lag 0-6 d	-	log-linear	0.0004353	0.000308	590	564	1,328,261	1,317,928	12	15
Mortality, Non-Accidental	Bell et al., 2004	Denver, CO	D8HourMax	March-September (7)	0-99	distributed lag 0-6 d	-	log-linear	0.0003862	0.0003105	380	355	558,817	559,791	4	16
Mortality, Non-Accidental	Bell et al., 2004	Detroit, MI	D8HourMax	April-September (6)	0-99	distributed lag 0-6 d	-	log-linear	0.0003307	0.000259	424	402	1,986,360	1,969,826	13	14
Mortality, Non-Accidental	Bell et al., 2004	Houston, TX	D8HourMax	January-December (12)	0-99	distributed lag 0-6 d	-	log-linear	0.0004422	0.0003018	500	484	3,780,576	3,850,328	6	7
Mortality, Non-Accidental	Bell et al., 2004	Los Angeles, CA	D8HourMax	January-December (12)	0-99	distributed lag 0-6 d	-	log-linear	0.0004168	0.0002963	562	548	9,981,640	10,042,327	9	8
Mortality, Non-Accidental	Bell et al., 2004	New York, NY	D8HourMax	April-October (7)	0-99	distributed lag 0-6 d	-	log-linear	0.0005284	0.0003191	401	385	11,043,332	11,108,750	10	8
Mortality, Non-Accidental	Bell et al., 2004	Philadelphia, PA	D8HourMax	April-October (7)	0-99	distributed lag 0-6 d	-	log-linear	0.0003736	0.0003219	562	530	1,456,148	1,444,164	13	9
Mortality, Non-Accidental	Bell et al., 2004	Sacramento, CA	D8HourMax	January-December (12)	0-99	distributed lag 0-6 d	-	log-linear	0.0004242	0.0002985	661	636	1,405,744	1,453,703	13	5
Mortality, Non-Accidental	Bell et al., 2004	St. Louis, MO	D8HourMax	April-October (7)	0-99	distributed lag 0-6 d	-	log-linear	0.0004113	0.0003141	463	444	2,198,242	2,211,259	8	7

Endpoint	Study	Urban study area	Study information (C-R function)								Baseline incidence[b]		Population		Surrogate LMLs (ppb)	
			Air metric	Risk assessment modeling period	Age range	Lag	Additional study details	Statistical Model	Effect estimate (Beta)	SE (effect estimate)[a]	2007	2009	2007	2009	2007	2009
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Atlanta, GA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0006124	0.0003358	37	37	3,856,357	3,972,395	24	21
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Baltimore, MD	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0009665	0.000353	82	82	2,146,632	2,194,116	13	24
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Boston, MA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0007666	0.0003617	58	58	4,021,878	4,048,879	19	17
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Cleveland, OH	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0007935	0.0003711	99	99	1,328,261	1,317,928	6	16
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Denver, CO	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0008258	0.0004083	49	49	558,817	559,791	21	22
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Detroit, MI	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0014243	0.0003663	90	90	1,986,360	1,969,826	19	11
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Houston, TX	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0006319	0.0003116	41	41	3,780,576	3,850,328	10	15
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Los Angeles, CA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0003071	0.0002432	56	56	9,981,639	10,042,327	31	22
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	New York, NY	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0012475	0.0002648	77	77	11,043,330	11,108,750	10	12
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Philadelphia, PA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0009588	0.0003563	84	84	1,456,148	1,444,164	12	14
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	Sacramento, CA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.000754	0.0003961	60	60	1,405,744	1,453,703	30	30
Mortality, Cardiovascular	Zanobetti and Schwartz (b), 2008	St. Louis, MO	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0007753	0.0003646	84	84	2,198,242	2,211,259	22	22
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Atlanta, GA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0010642	0.0003746	11	11	3,856,357	3,972,395	24	21
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Baltimore, MD	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0009325	0.0003763	20	19	2,146,632	2,194,116	13	24
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Boston, MA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0010557	0.0003765	20	19	4,021,878	4,048,879	19	17
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Cleveland, OH	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0009481	0.0003905	21	20	1,328,261	1,317,928	6	16
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Denver, CO	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0008799	0.0004032	19	18	558,817	559,791	21	22
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Detroit, MI	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0010421	0.000388	18	17	1,986,360	1,969,826	19	11
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Houston, TX	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0004702	0.0003578	10	10	3,780,576	3,850,328	10	15
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Los Angeles, CA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0004179	0.0003101	14	14	9,981,639	10,042,327	31	22
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	New York, NY	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0008568	0.0003505	16	15	11,043,330	11,108,750	10	12
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Philadelphia, PA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0007869	0.0003787	21	20	1,456,148	1,444,164	12	14
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	Sacramento, CA	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.000793	0.0003976	20	19	1,405,744	1,453,703	30	30
Mortality, Respiratory	Zanobetti and Schwartz (b), 2008	St. Louis, MO	D8HourMean	June-August	0-99	distributed lag 0-3 d	-	log-linear	0.0008746	0.000383	19	18	2,198,242	2,211,259	22	22

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Endpoint	Study	Urban study area	Study information (C-R function)								Baseline incidence[b]		Population		Surrogate LMLs (ppb)	
			Air metric	Risk assessment modeling period	Age range	Lag	Additional study details	Statistical Model	Effect estimate (Beta)	SE (effect estimate)[a]	2007	2009	2007	2009	2007	2009
Asthma Exacerbation, Chest Tightness	Gent et al., 2003	Boston, MA	D1HourMax	April-September (6)	0-12	Lag 1d	-	logistic	0.0007609	0.0020002	19,541	19,546	662,064	669,219	14	15
Asthma Exacerbation, Chest Tightness	Gent et al., 2003	Boston, MA	D8HourMax	April-September (6)	0-12	Lag 1d	-	logistic	0.0057036	0.0020217	19,541	19,546	662,064	669,219	12	12
Asthma Exacerbation, Chest Tightness	Gent et al., 2003	Boston, MA	D1HourMax	April-September (6)	0-12	Lag 1d	PM2.5	logistic	0.0077052	0.0022666	19,541	19,546	662,064	669,219	14	15
Asthma Exacerbation, Chest Tightness	Gent et al., 2003	Boston, MA	D1HourMax	April-September (6)	0-12	Lag 1d	PM2.5	logistic	0.0070131	0.0022734	19,541	19,546	662,064	669,219	14	15
Asthma Exacerbation, Shortness of Breath	Gent et al., 2003	Boston, MA	D1HourMax	April-September (6)	0-12	Lag 1d	-	logistic	0.003977	0.0017947	24,426	24,432	662,064	669,219	14	15
Asthma Exacerbation, Shortness of Breath	Gent et al., 2003	Boston, MA	D8HourMax	April-September (6)	0-12	Lag 1d	-	logistic	0.0052473	0.0021808	24,426	24,432	662,064	669,219	12	12
Asthma Exacerbation, Wheeze	Gent et al., 2003	Boston, MA	D1HourMax	April-September (6)	0-12	Lag 0d	PM2.5	logistic	0.0060021	0.0020225	45,595	45,607	662,064	669,219	14	15
Emergency Room Visits, Asthma	Ito et al., 2007	New York, NY	D8HourMax	April-October (7)	0-99	average of lag 0 and lag 1	-	log-linear	0.0052134	0.0009087	686	686	11,043,332	11,108,750	10	8
Emergency Room Visits, Asthma	Ito et al., 2007	New York, NY	D8HourMax	April-October (7)	0-99	average of lag 0 and lag 1	PM2.5	log-linear	0.0039757	0.0009789	686	686	11,043,332	11,108,750	10	8
Emergency Room Visits, Asthma	Ito et al., 2007	New York, NY	D8HourMax	April-October (7)	0-99	average of lag 0 and lag 1	NO2	log-linear	0.0032337	0.0009359	686	686	11,043,332	11,108,750	10	8
Emergency Room Visits, Asthma	Ito et al., 2007	New York, NY	D8HourMax	April-October (7)	0-99	average of lag 0 and lag 1	CO	log-linear	0.0055437	0.0008939	686	686	11,043,332	11,108,750	10	8
Emergency Room Visits, Asthma	Ito et al., 2007	New York, NY	D8HourMax	April-October (7)	0-99	average of lag 0 and lag 1	SO2	log-linear	0.004115	0.0009226	686	686	11,043,332	11,108,750	10	8
Emergency Room Visits, Respiratory	Darrow et al., 2011	Atlanta, GA	D8HourMax	March-October (8)	0-99	Lag 1d	-	log-linear	0.0006852	0.0001385	2,889	2,902	3,856,358	3,972,395	17	5
Emergency Room Visits, Respiratory	Strickland et al., 2010	Atlanta, GA	D8HourMax	March-October (8)	5-17	distributed lag 0-7 d	-	log-linear	0.0047864	0.0007602	5,464	5,464	697,690	714,368	17	5
Emergency Room Visits, Respiratory	Strickland et al., 2010	Atlanta, GA	D8HourMax	March-October (8)	5-17	average of lags 0-2	-	log-linear	0.002699	0.0006456	5,464	5,464	697,690	714,368	17	5
Emergency Room Visits, Respiratory	Tolbert et al., 2007	Atlanta, GA	D8HourMax	March-October (8)	0-99	average of lags 0-2	-	log-linear	0.001286	0.0002062	2,889	2,902	3,856,358	3,972,395	17	5
Emergency Room Visits, Respiratory	Tolbert et al., 2007	Atlanta, GA	D8HourMax	March-October (8)	0-99	average of lags 0-2	CO	log-linear	0.0011408	0.0002283	2,889	2,902	3,856,358	3,972,395	17	5
Emergency Room Visits, Respiratory	Tolbert et al., 2007	Atlanta, GA	D8HourMax	March-October (8)	0-99	average of lags 0-2	NO2	log-linear	0.0010287	0.0002506	2,889	2,902	3,856,358	3,972,395	17	5
Emergency Room Visits, Respiratory	Tolbert et al., 2007	Atlanta, GA	D8HourMax	March-October (8)	0-99	average of lags 0-2	PM10	log-linear	0.0008032	0.000267	2,889	2,902	3,856,358	3,972,395	17	5
Emergency Room Visits, Respiratory	Tolbert et al., 2007	Atlanta, GA	D8HourMax	March-October (8)	0-99	average of lags 0-2	PM10, NO2	log-linear	0.0007749	0.0002672	2,889	2,902	3,856,358	3,972,395	17	5
HA, All Respiratory	Katsouyanni et al., 2009	Detroit, MI	D1HourMax	June-August	65-99	average of lag 0 and lag 1	penalized splines	log-linear	0.00056	0.000352	1,348	1,336	221,636	218,112	23	17

Endpoint	Study	Urban study area	Study information (C-R function)								Baseline incidence[b]		Population		Surrogate LMLs (ppb)	
			Air metric	Risk assessment modeling period	Age range	Lag	Additional study details	Statistical Model	Effect estimate (Beta)	SE (effect estimate)[a]	2007	2009	2007	2009	2007	2009
HA, All Respiratory	Katsouyanni et al., 2009	Detroit, MI	D1HourMax	June-August	65-99	average of lag 0 and lag 1	natural splines	log-linear	0.00054	0.0003571	1,348	1,336	221,636	218,112	23	17
HA, All Respiratory	Linn et al., 2000	Los Angeles, CA	D24HourMean	June-August	30-99	Lag 0d	-	log-linear	0.0006	0.0007	269	273	5640233.5	5730434.5	18	18
HA, Asthma	Silverman and Ito, 2010	New York, NY	D8HourMax	April-October (7)	6-18	average of lag 0 and lag 1	-	log-linear	0.007907	0.0037862	192	192	1,852,727	1,869,528	10	8
HA, Asthma	Silverman and Ito, 2010	New York, NY	D8HourMax	April-October (7)	6-18	average of lag 0 and lag 1	PM2.5	log-linear	0.0055553	0.0036926	192	192	1,852,727	1,869,528	10	8
HA, Chronic Lung Disease	Lin et al. (a), 2008	New York, NY	D1HourMax	April-October (7)	0-17	Lag 2 d	-	log-linear	0.0007609	0.000163	234	233	2,593,597	2,593,341	16	11
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Atlanta, GA	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	367	365	301,812	325,379	24	21
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Baltimore, MD	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	314	312	263,211	272,392	13	24
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Boston, MA	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	256	254	509,862	519,854	19	17
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Cleveland, OH	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	320	317	195,957	192,596	6	16
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Denver, CO	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	204	204	55,918	53,947	21	22
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Detroit, MI	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	375	372	221,636	218,112	19	11
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Houston, TX	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	261	259	291,477	307,353	10	15
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Los Angeles, CA	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	187	187	1,015,099	1,048,772	31	22
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	New York, NY	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	196	195	1,364,875	1,375,434	10	12
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Philadelphia, PA	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	265	262	183,785	179,364	12	14
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	Sacramento, CA	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	161	162	152,074	158,266	30	30
HA, Chronic Lung Disease (less Asthma)	Medina-Ramon et al, 2006	St. Louis, MO	D8HourMean	June-August	65-99	distributed lag 0-1 d	-	logistic	0.00054	0.000199	279	277	275,828	281,535	22	22

a-all Beta distributions assumed to be normal
b-Gent et al., 2003 also uses the following prevalence rates: 0.028 (wheeze), 0.015 (shortness of breath), 0.012 (chest tightness) (from study)

APPENDIX 8A.
SUPPLEMENT TO THE REPRESENTATIVENESS ANALYSIS OF THE 12 URBAN STUDY AREAS

Following the analysis discussed in Chapter 8, this appendix provides graphical comparisons of the empirical distributions of components of the risk function, and additional variables that have been identified as potentially influencing the risk associated with ozone exposures. In each graph, the blue line represents the cumulative distribution function (CDF) for the complete set of data available for the variable. In some cases, this many encompass all counties in the U.S., while in others it may be based on a subset of the U.S., usually for large urban areas. The black squares at the bottom of each graph represent the specific value of the variable for one of the case study locations, with the line showing where that value intersect the CDF of the nationwide data.

8-A.1. ELEMENTS OF THE RISK EQUATION

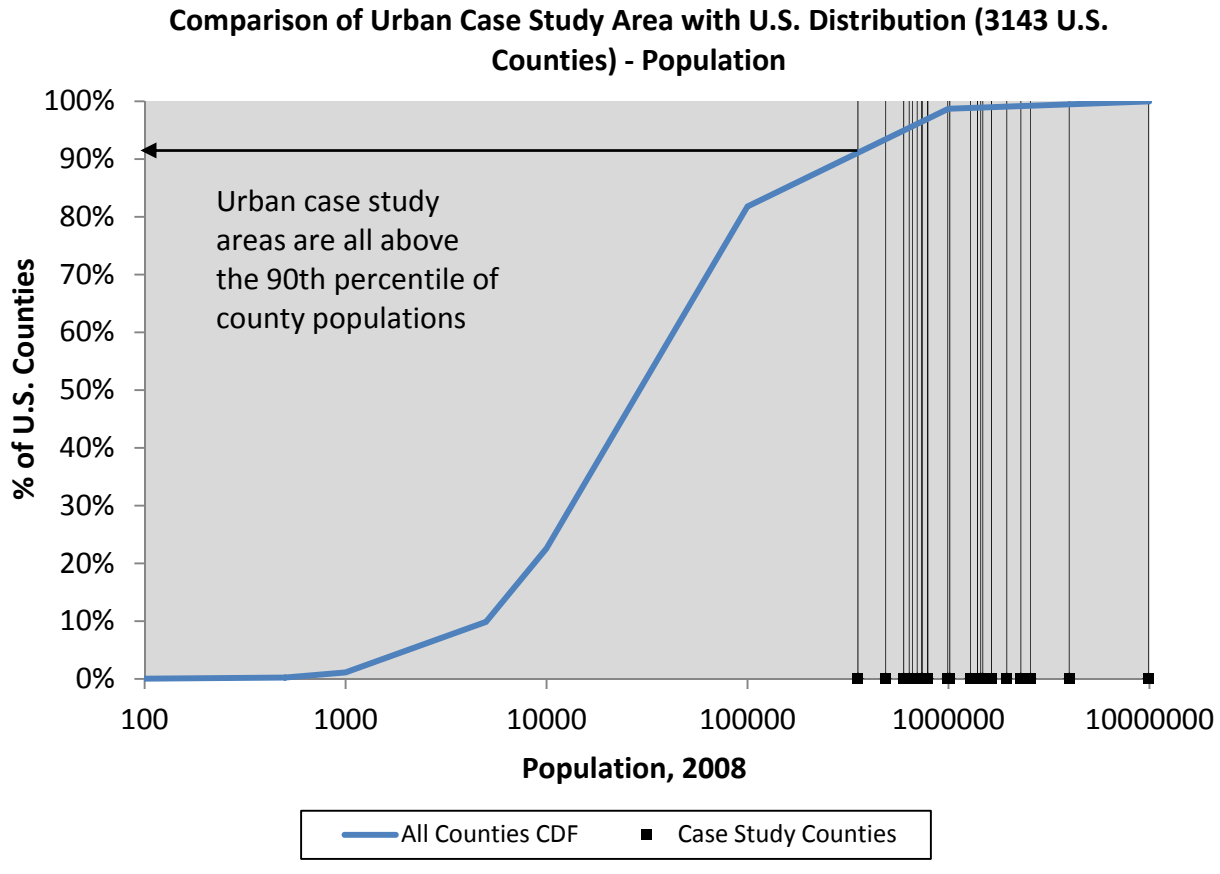

Figure 8-0.1 Comparison of distributions for key elements of the risk equation: Total population

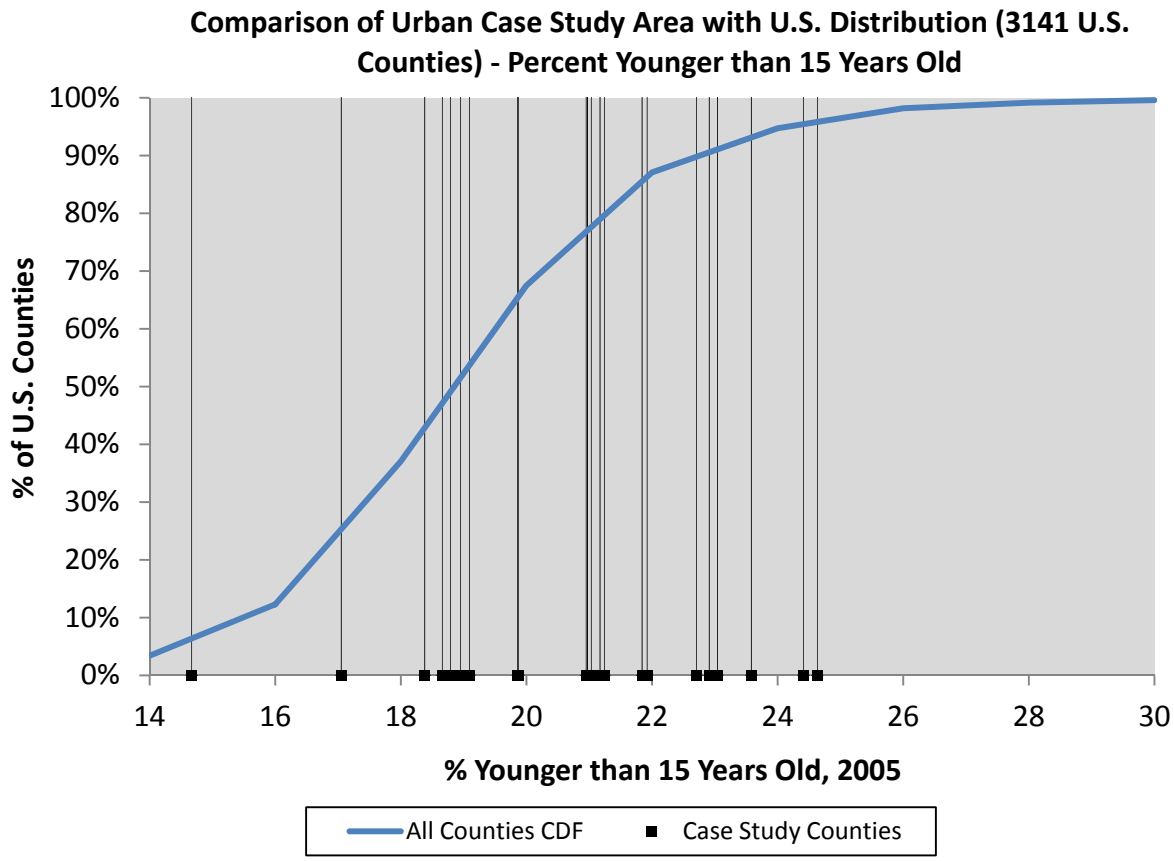

Figure 8-0.2 Comparison of distributions for key elements of the risk equation: Percent of population younger than 15 years old

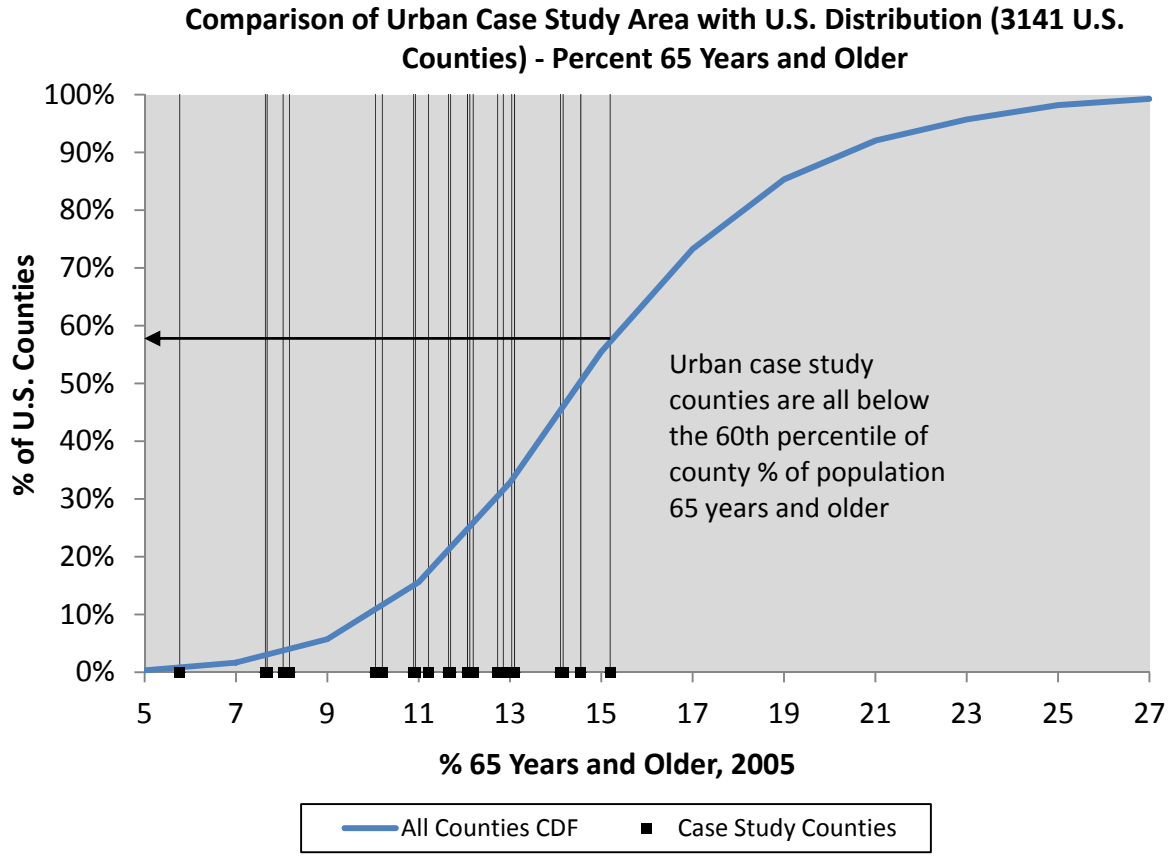

Figure 8-0.3 Comparison of distributions for key elements of the risk equation: Percent of population 65 and older

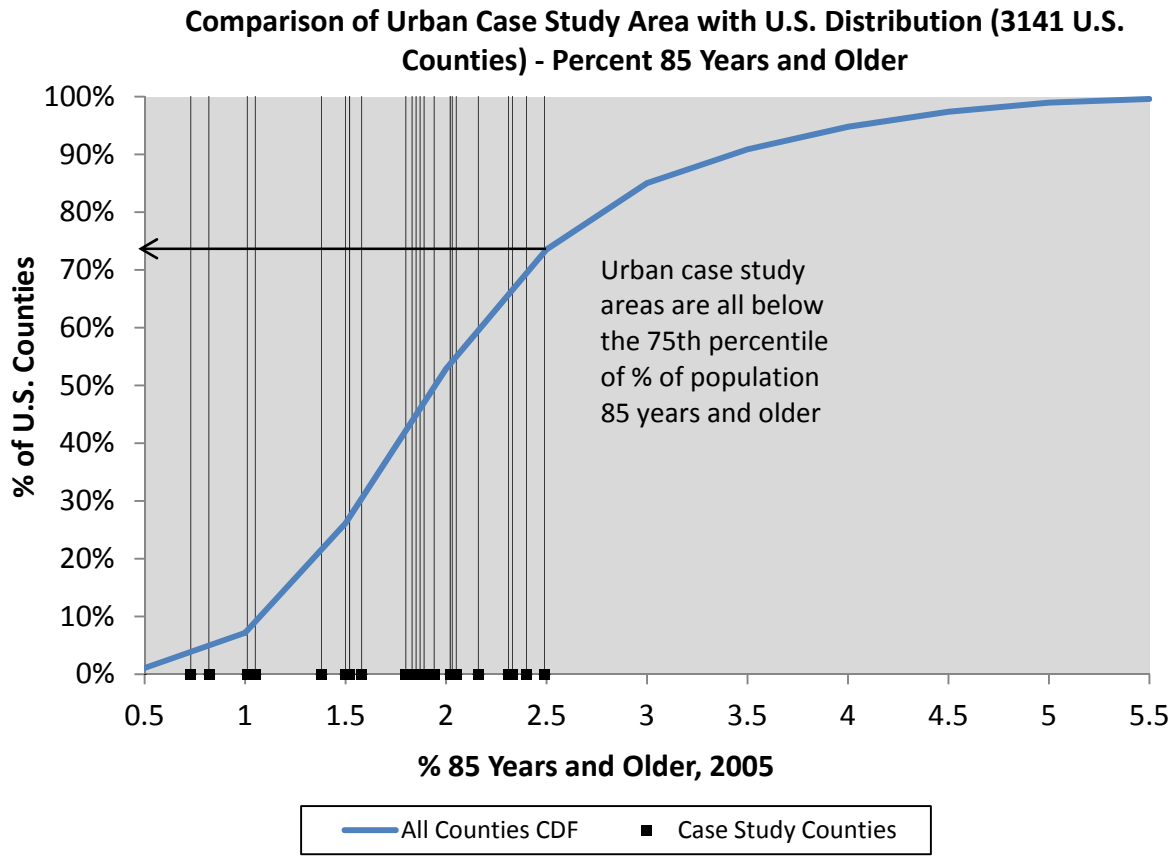

Figure 8-0.4 Comparison of distributions for key elements of the risk equation: Percent of population 85 and older

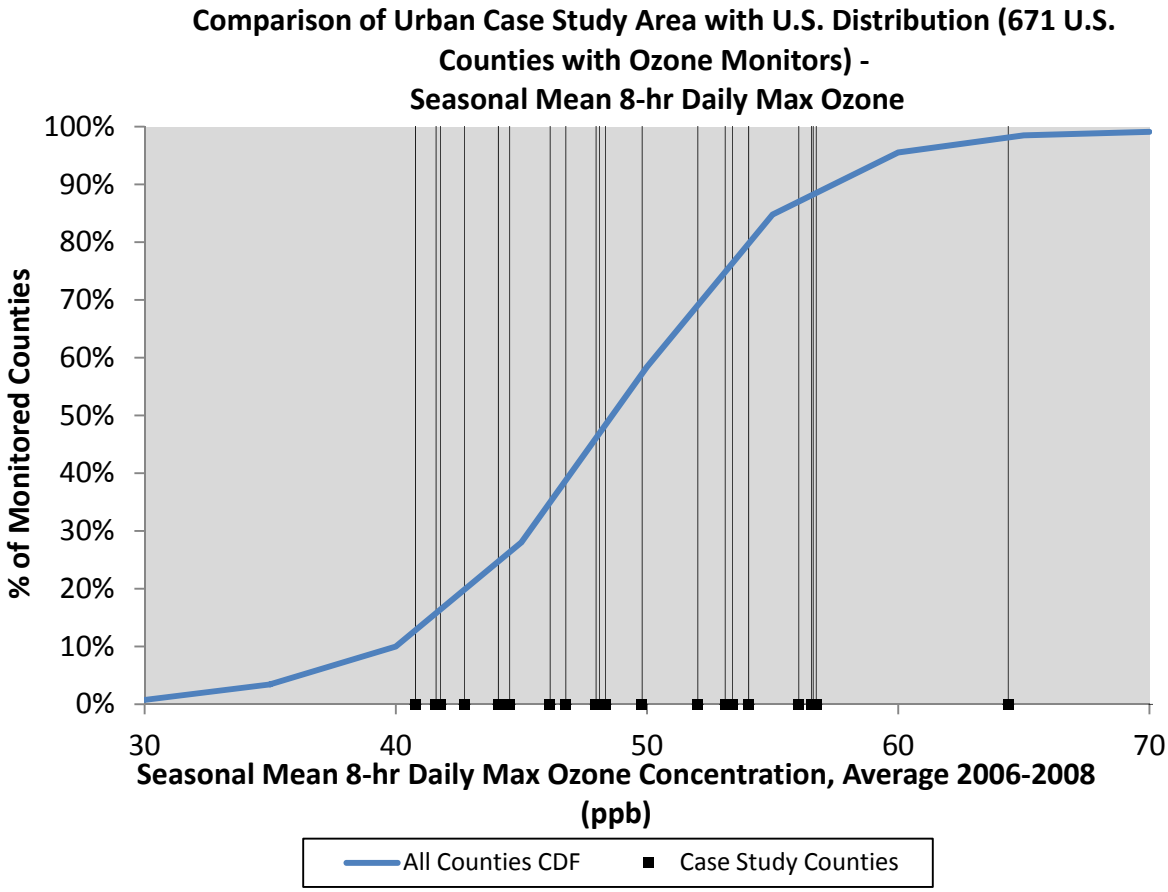

Figure 8-0.5 Comparison of distributions for key elements of the risk equation: Seasonal mean 8-hr daily maximum ozone concentration

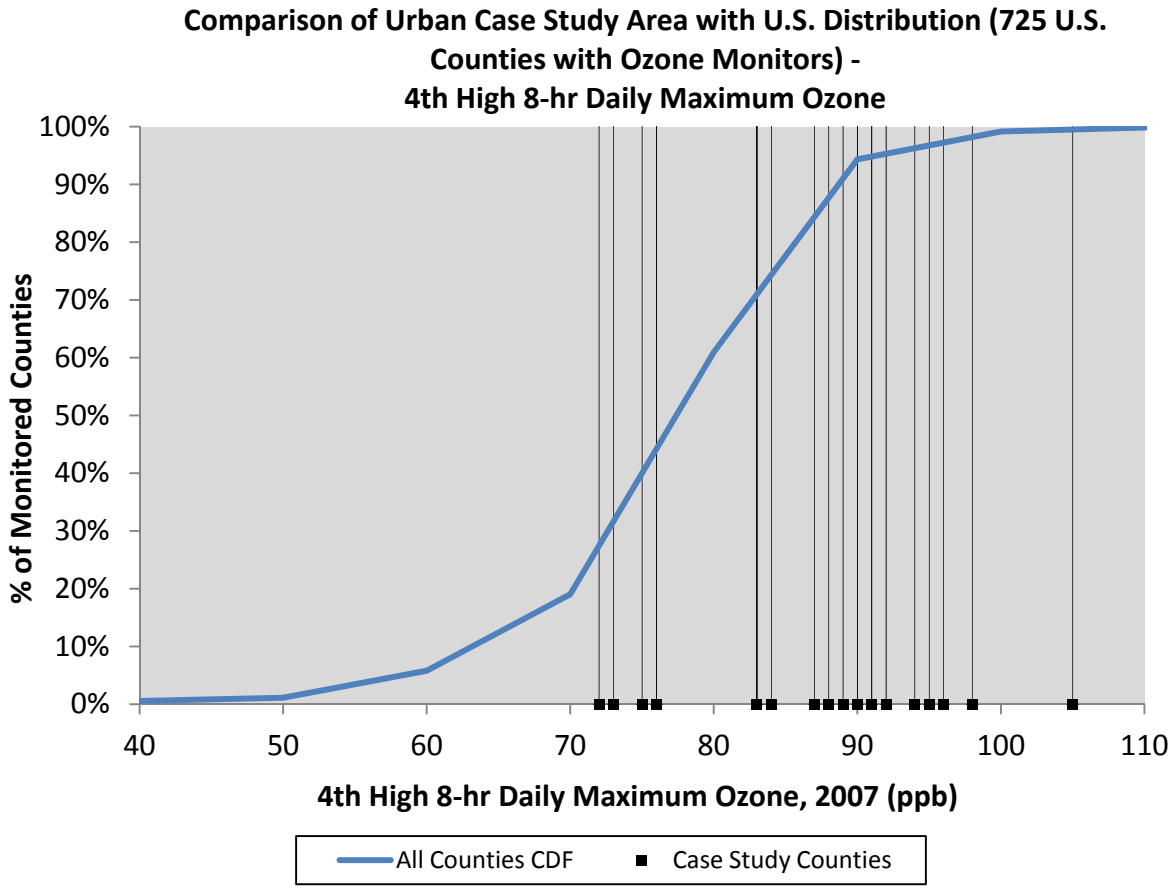

Figure 8-0.6 Comparison of distributions for key elements of the risk equation: 4th highest 8-hr daily maximum ozone concentration

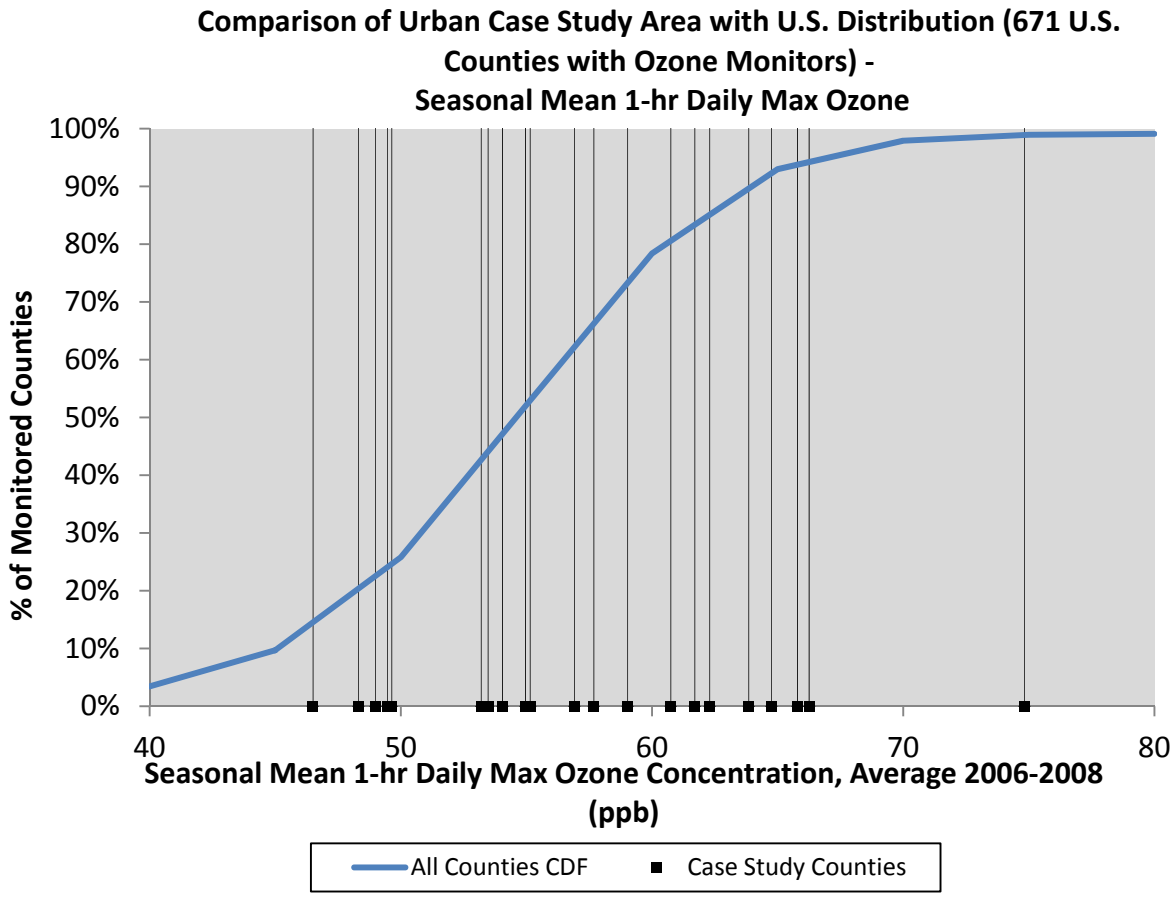

Figure 8-0.7 Comparison of distributions for key elements of the risk equation: Seasonal mean 1-hr daily maximum ozone concentration

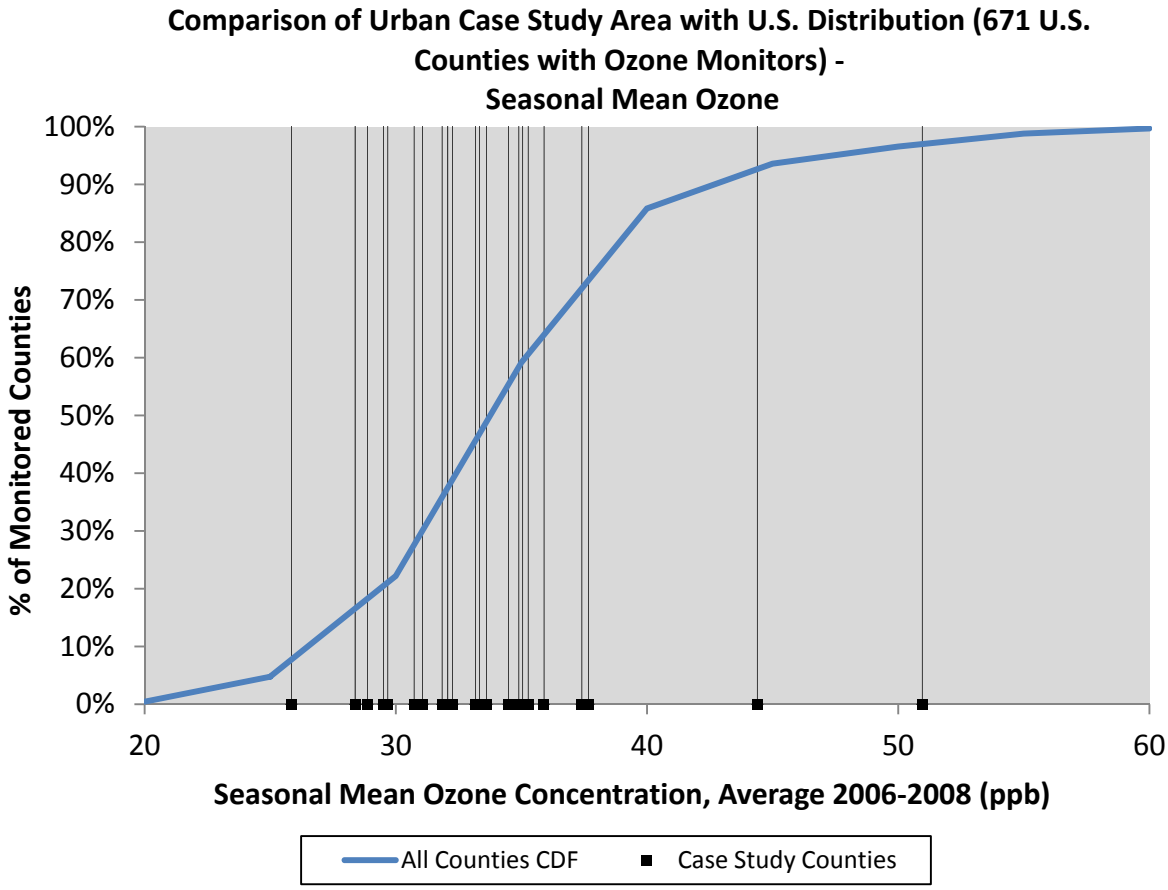

Figure 8-0.8 Comparison of distributions for key elements of the risk equation: Seasonal mean ozone concentration

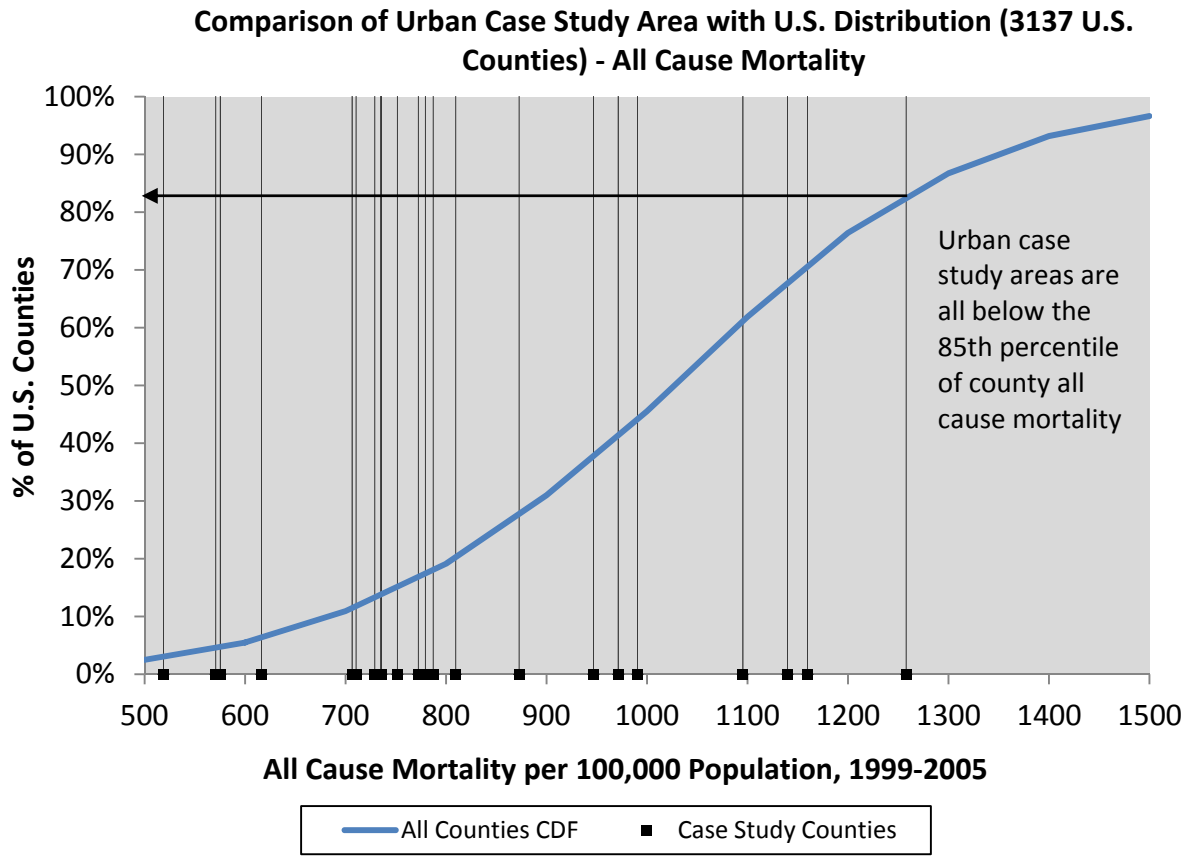

Figure 8-0.9 Comparison of distributions for key elements of the risk equation: Baseline all-cause mortality

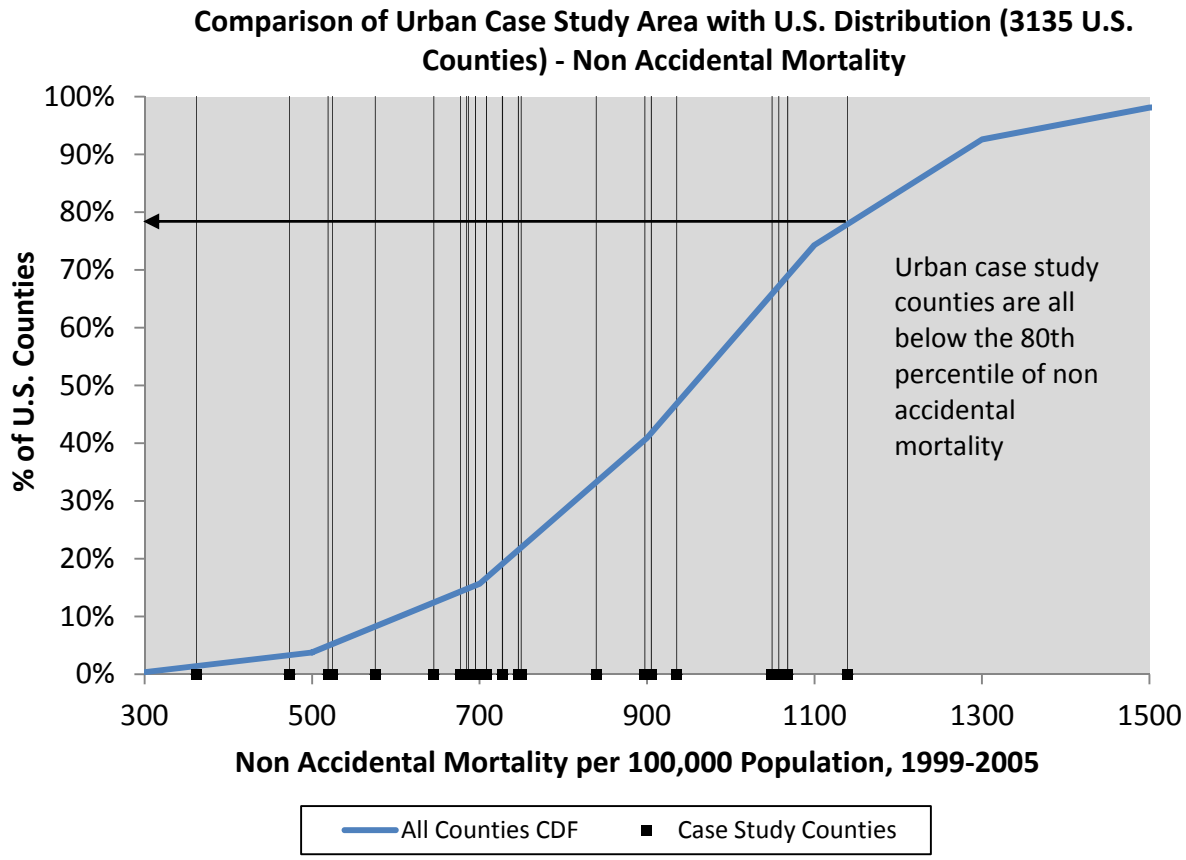

Figure 8-0.10 Comparison of distributions for key elements of the risk equation: Baseline non-accidental mortality

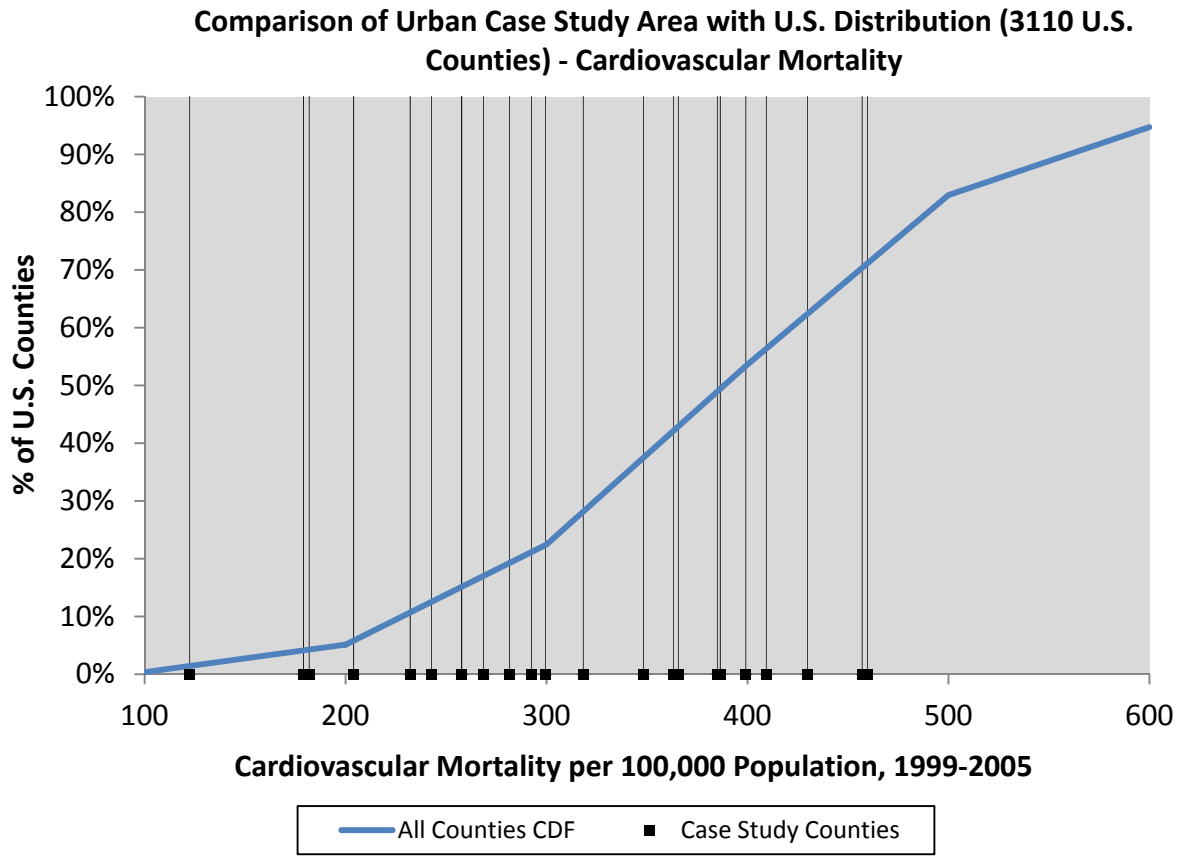

Figure 8-0.11 Comparison of distributions for key elements of the risk equation: Baseline cardiovascular mortality

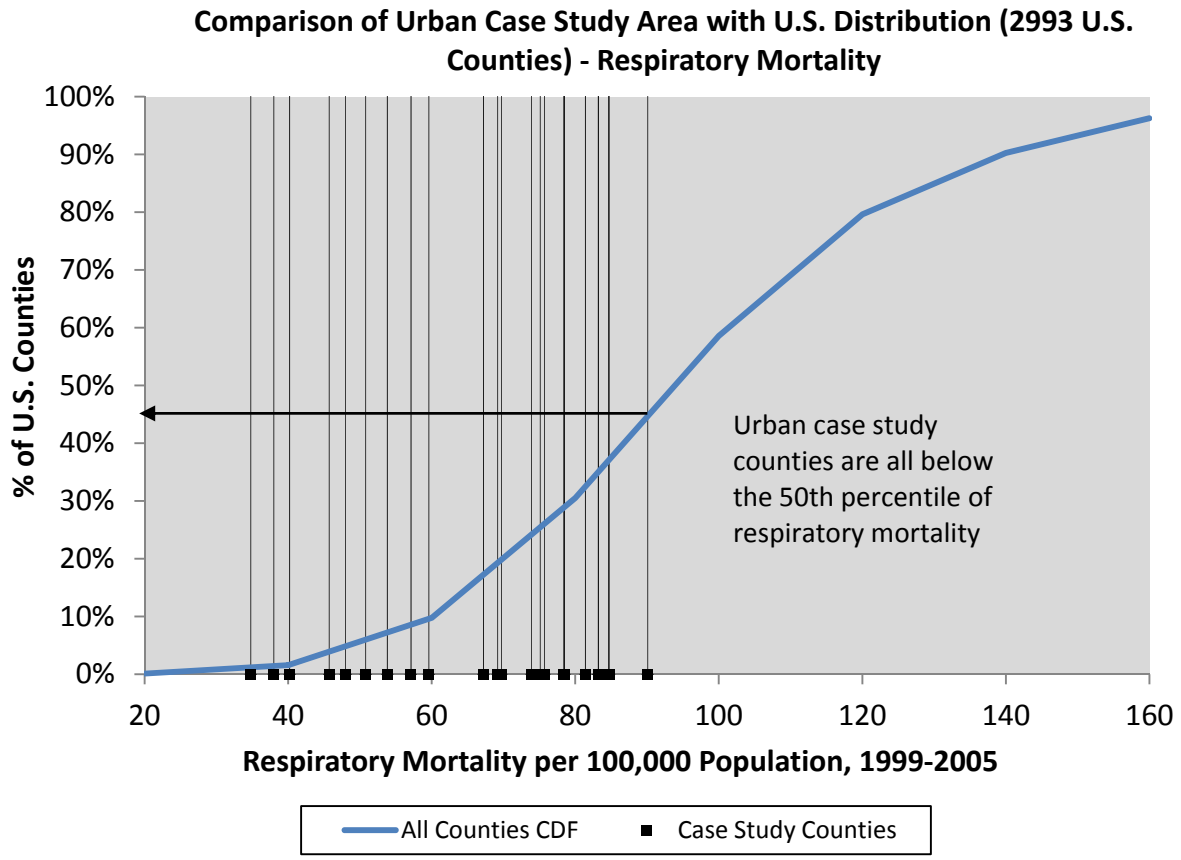

Figure 8-0.12 Comparison of distributions for key elements of the risk equation: Baseline respiratory mortality

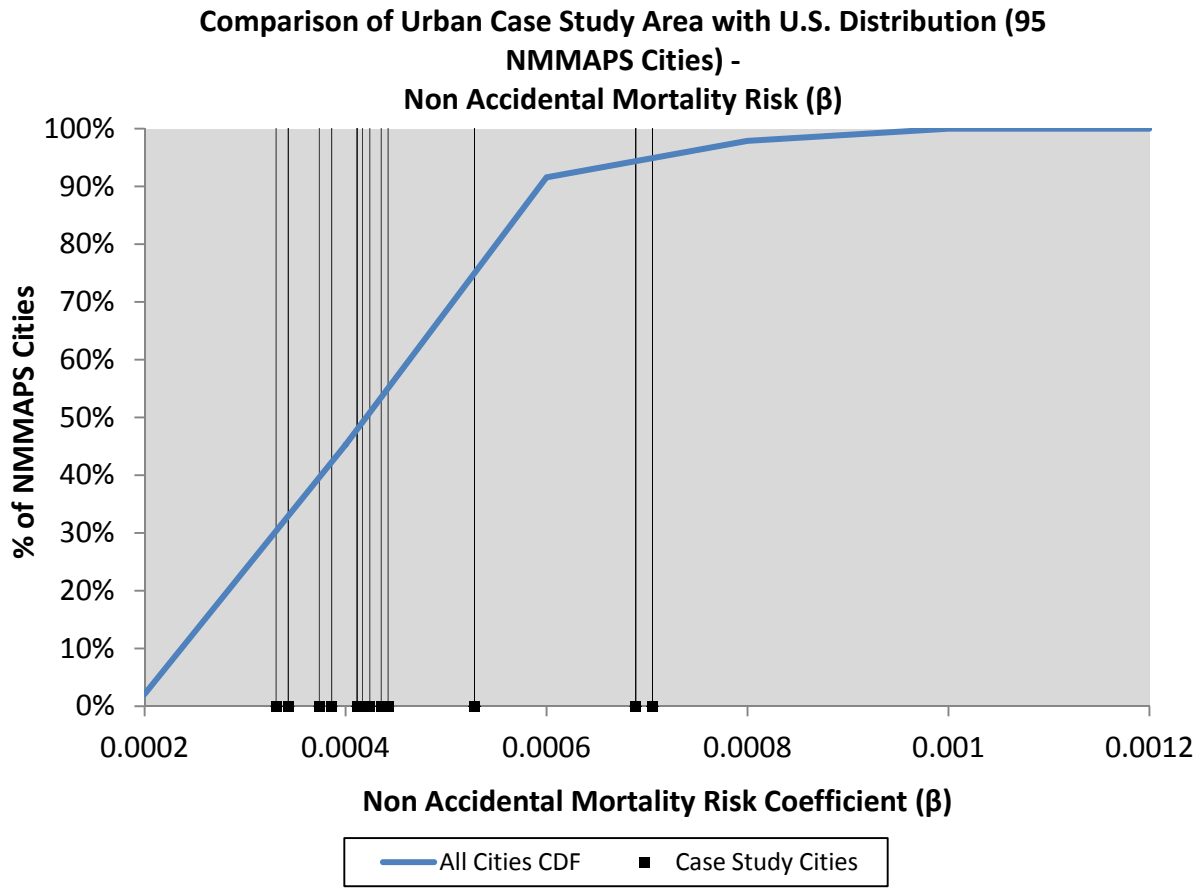

Figure 8-0.13 Comparison of distributions for key elements of the risk equation: Non-accidental mortality risk coefficient from Bell et al. (2004)

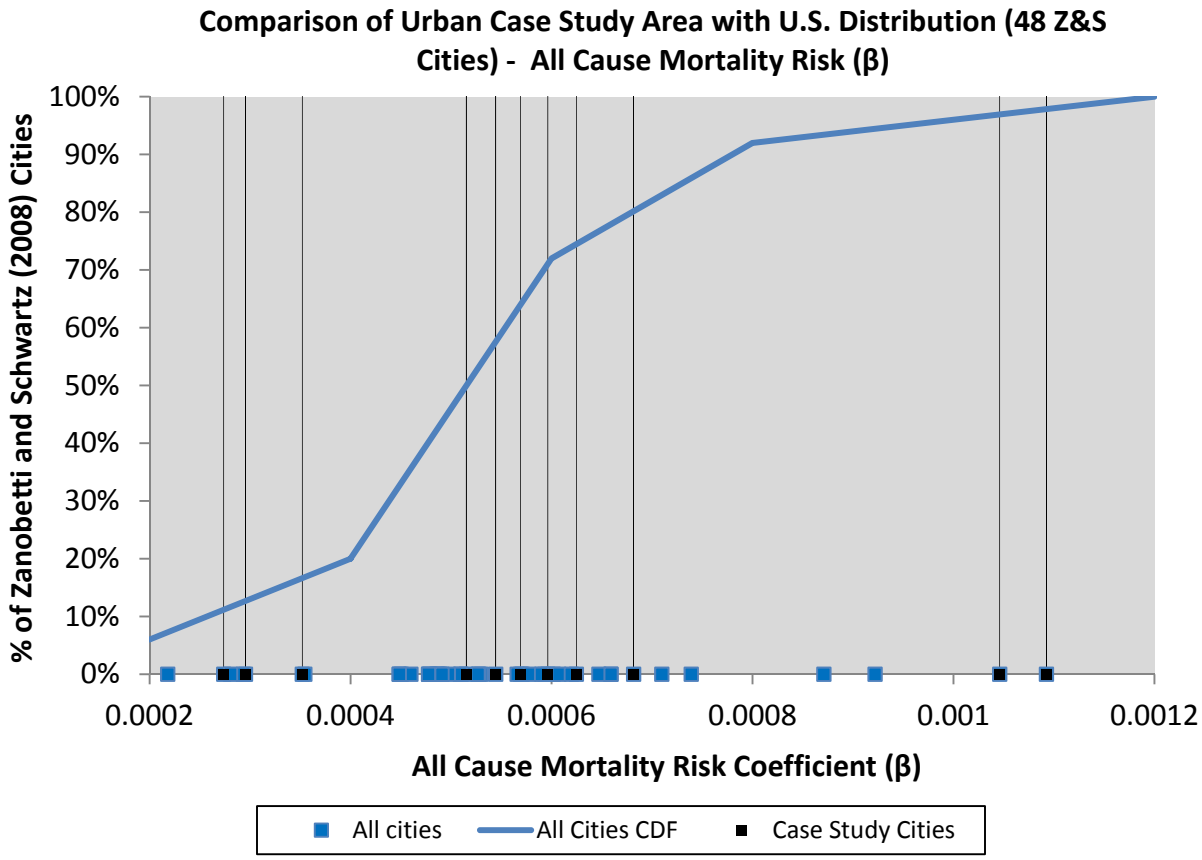

Figure 8-0.14 Comparison of distributions for key elements of the risk equation: All-cause mortality risk coefficient from Zanobetti and Schwartz (2008)

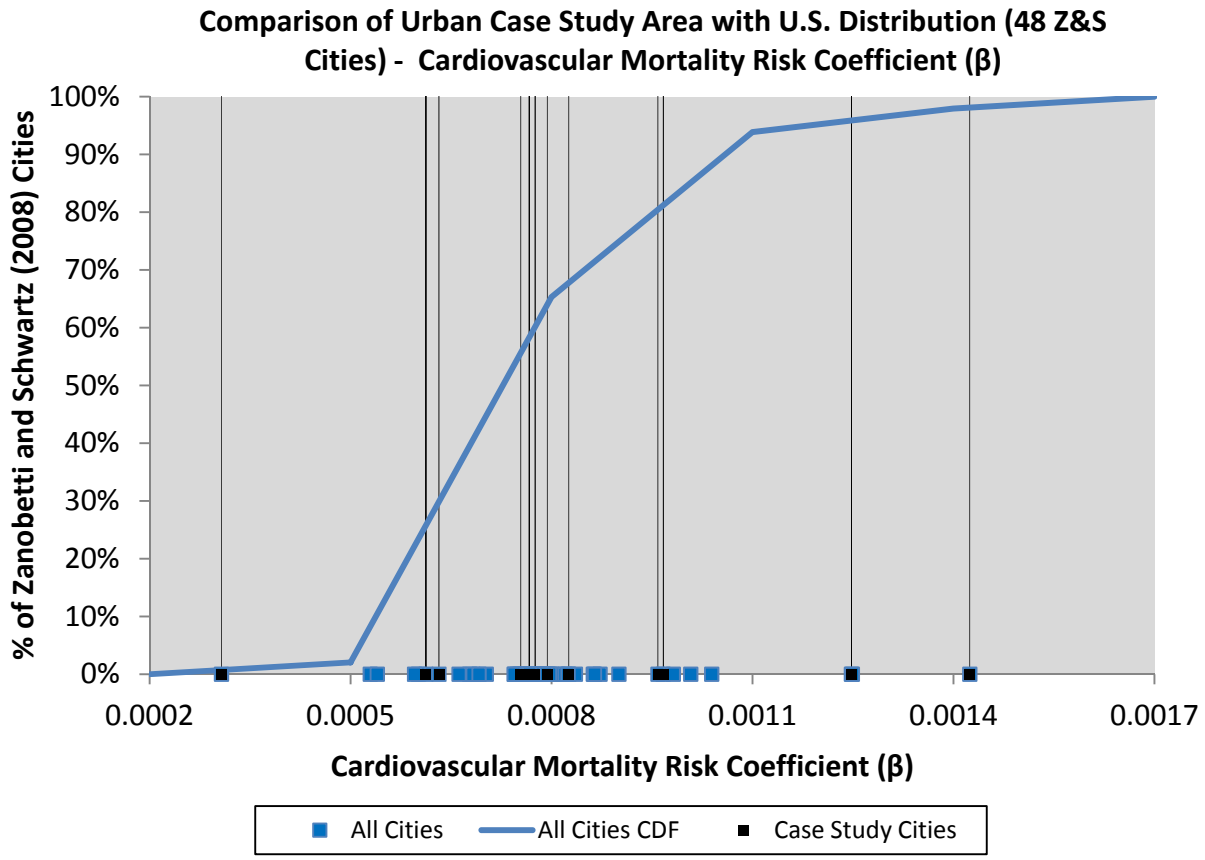

Figure 8-0.15 Comparison of distributions for key elements of the risk equation: Cardiovascular mortality risk coefficient from Zanobetti and Schwartz (2008)

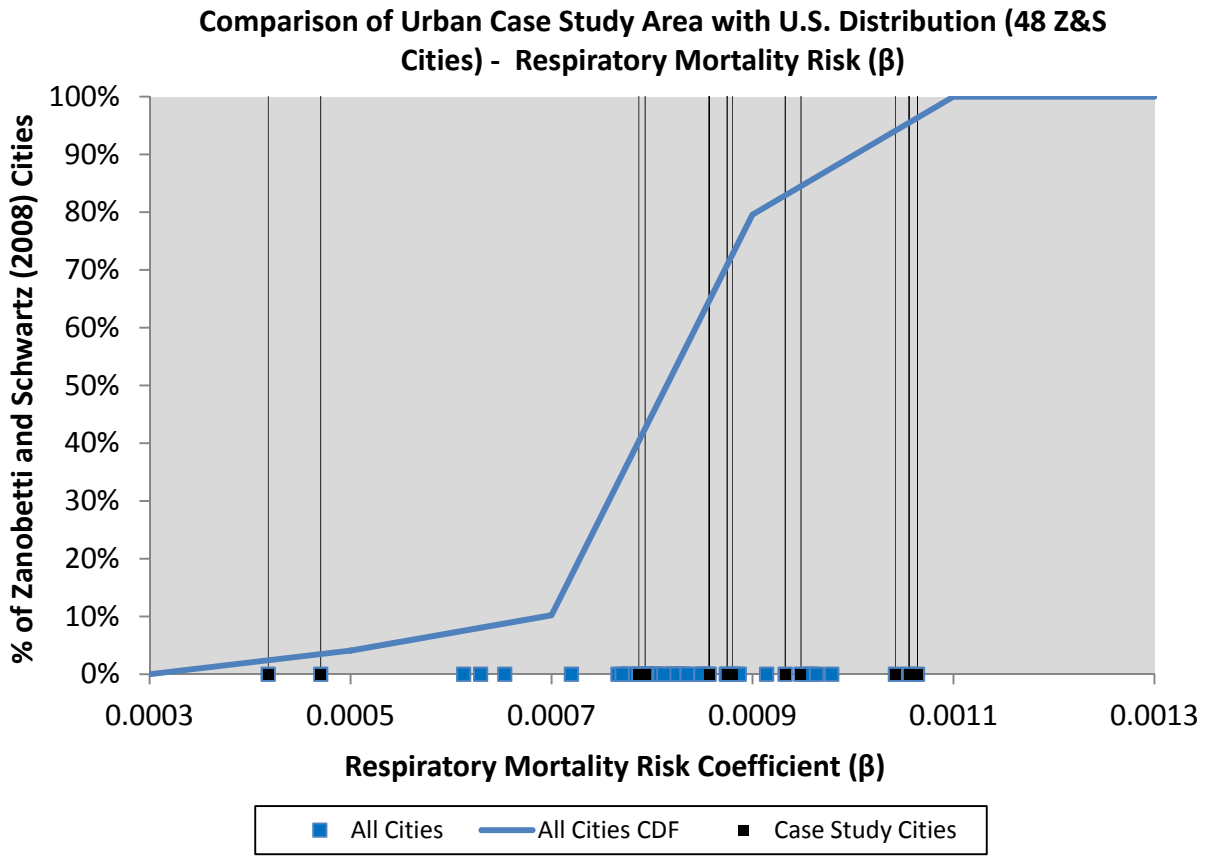

Figure 8-0.16 Comparison of distributions for key elements of the risk equation: Respiratory mortality risk coefficient from Zanobetti and Schwartz (2008)

8-A.2. VARIABLES EXPECTED TO INFLUENCE THE RELATIVE RISK FROM OZONE

i. Demographic Variables

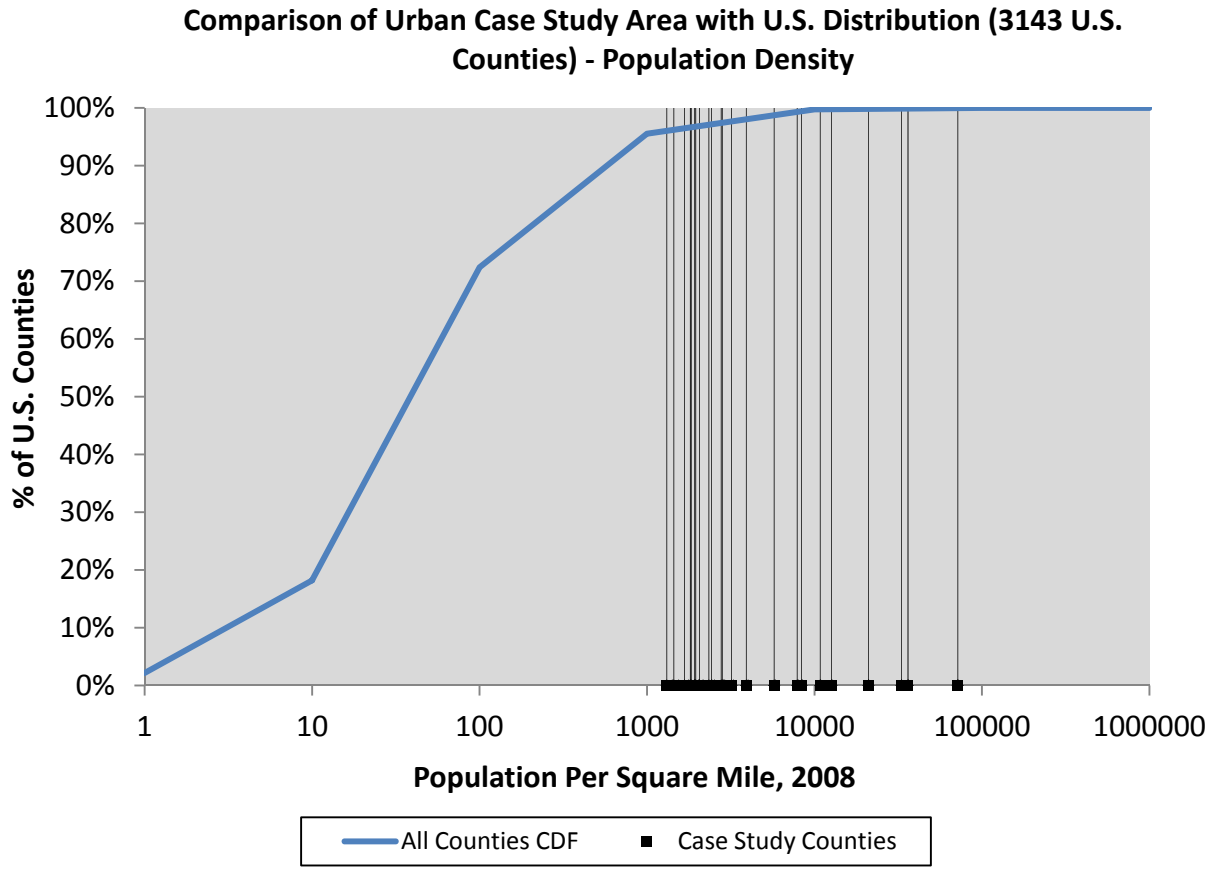

Figure 8-0.17 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Population density

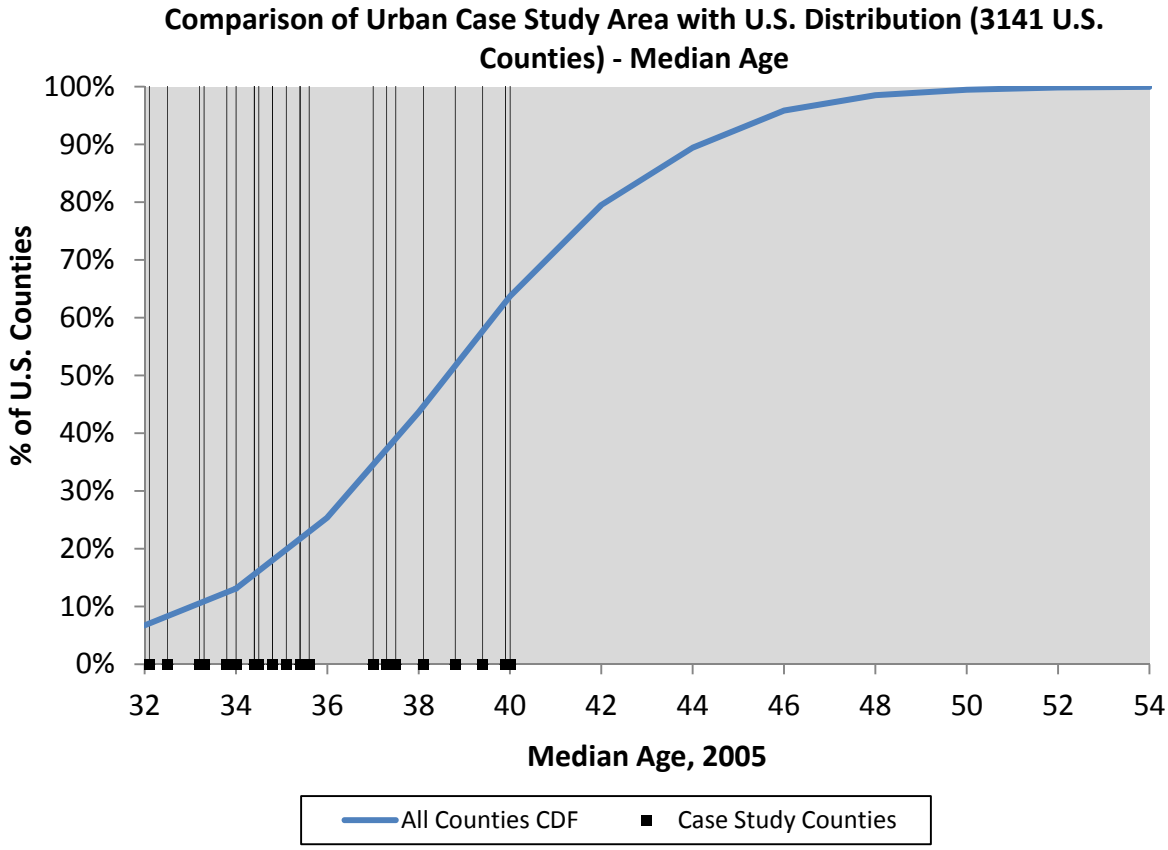

Figure 8-0.18 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Median age

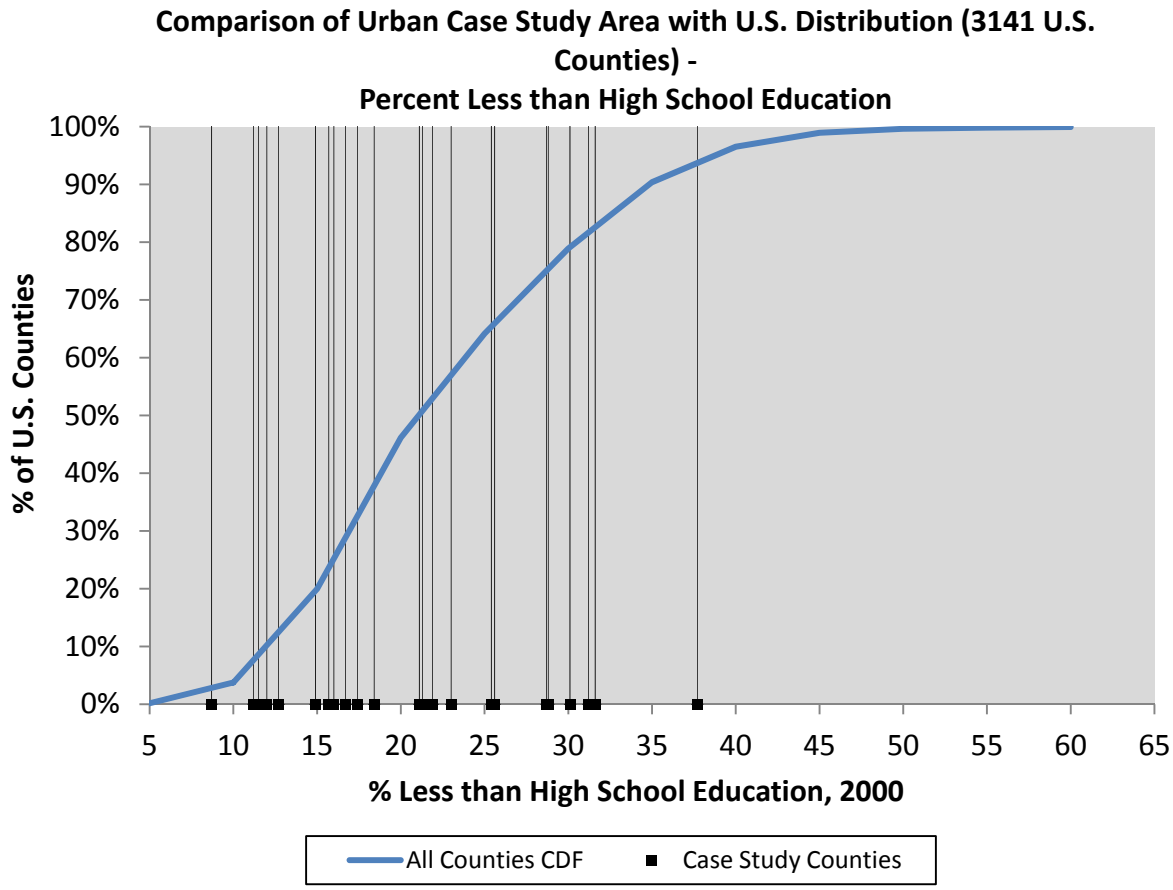

Figure 8-0.19 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Percent less than high school education

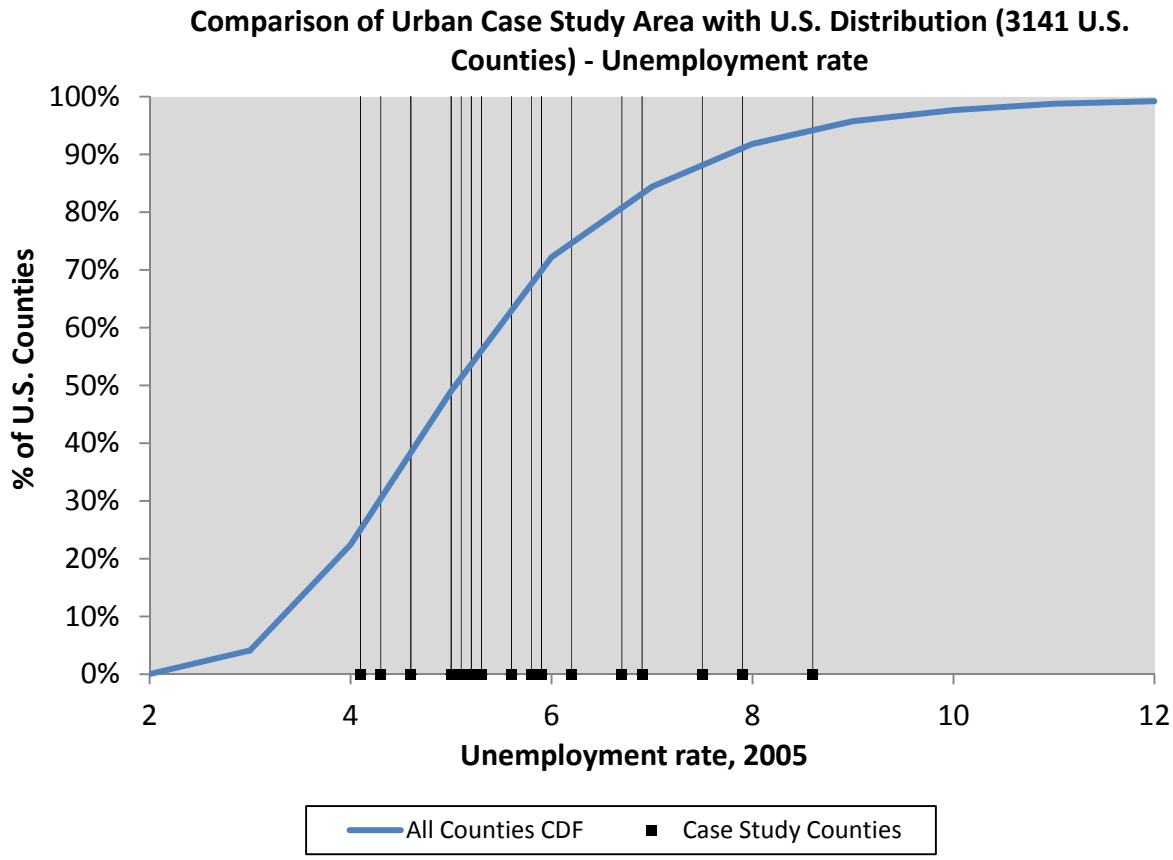

Figure 8-0.20 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Unemployment rate

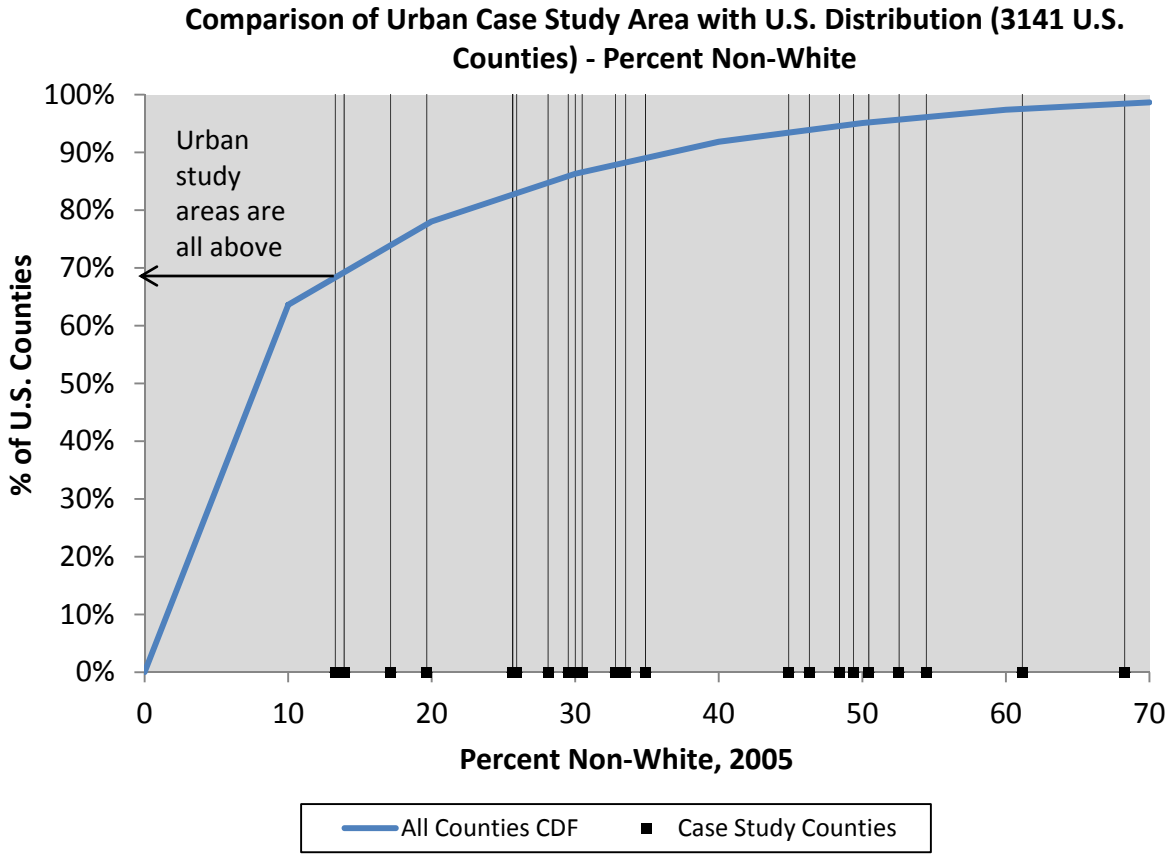

Figure 8-0.21 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Percent non-white

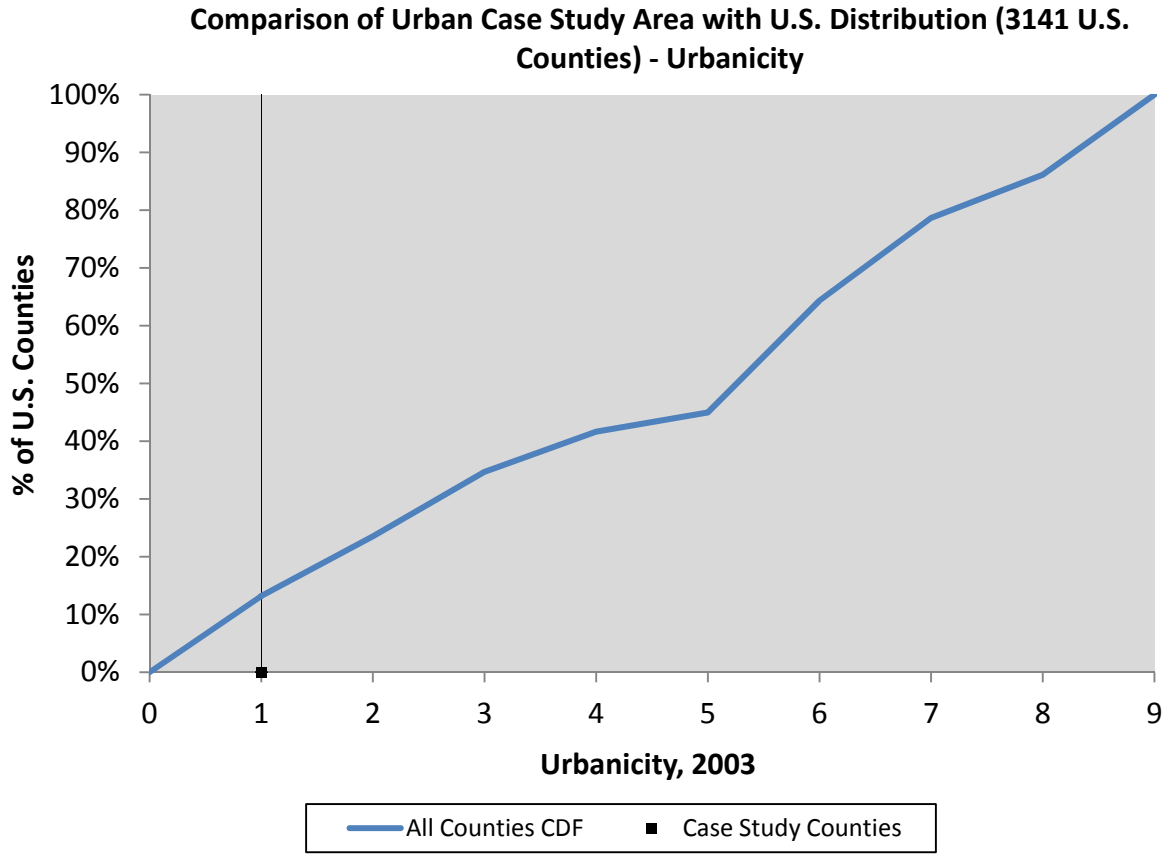

Figure 8-0.22 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Urbanicity

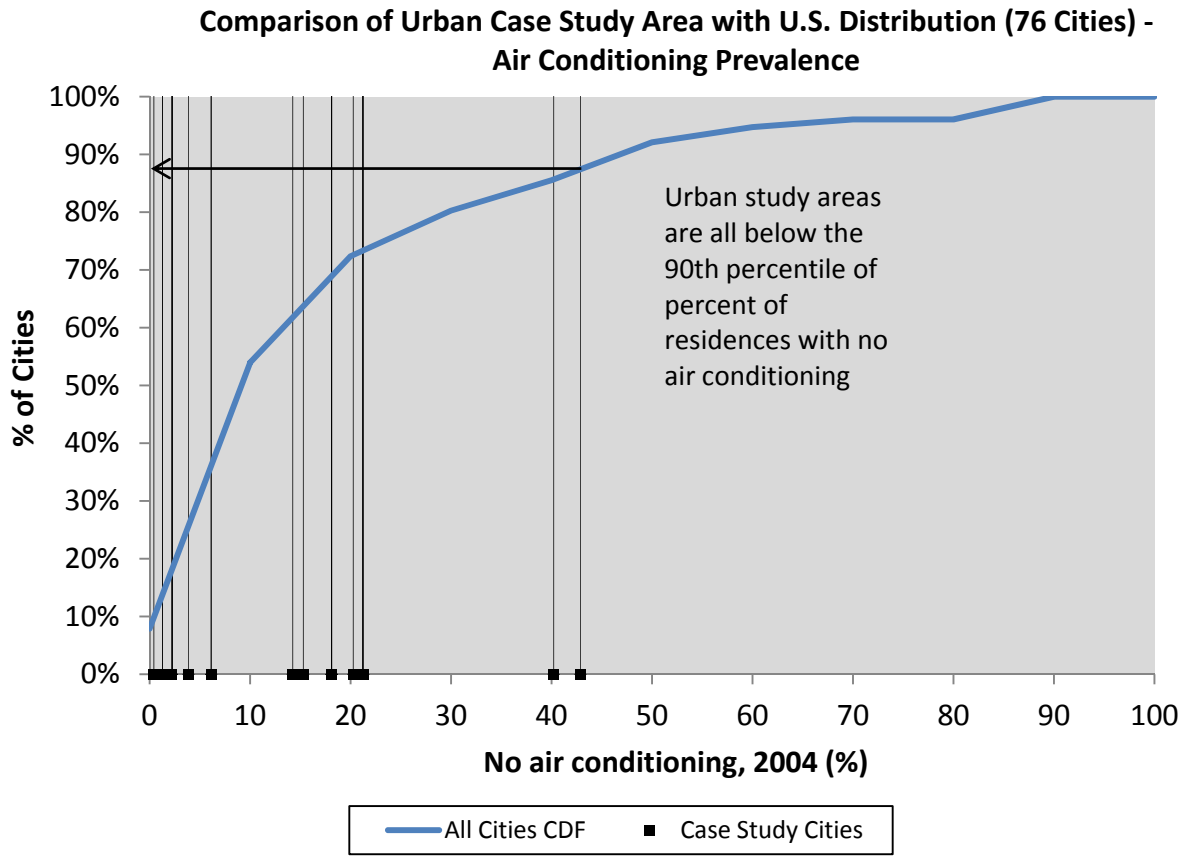

Figure 8-0.23 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Air conditioning prevalence

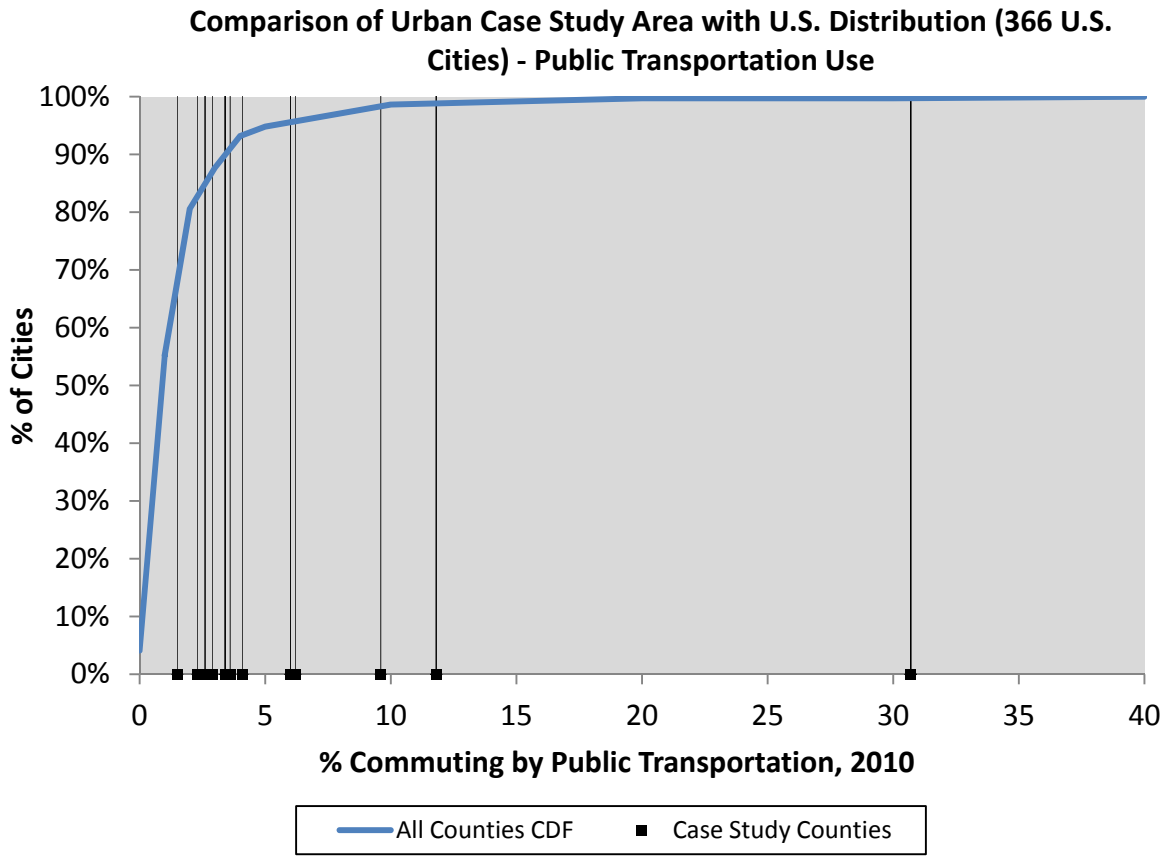

Figure 8-0.24 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Percent commuting by public transportation

ii. Health Conditions

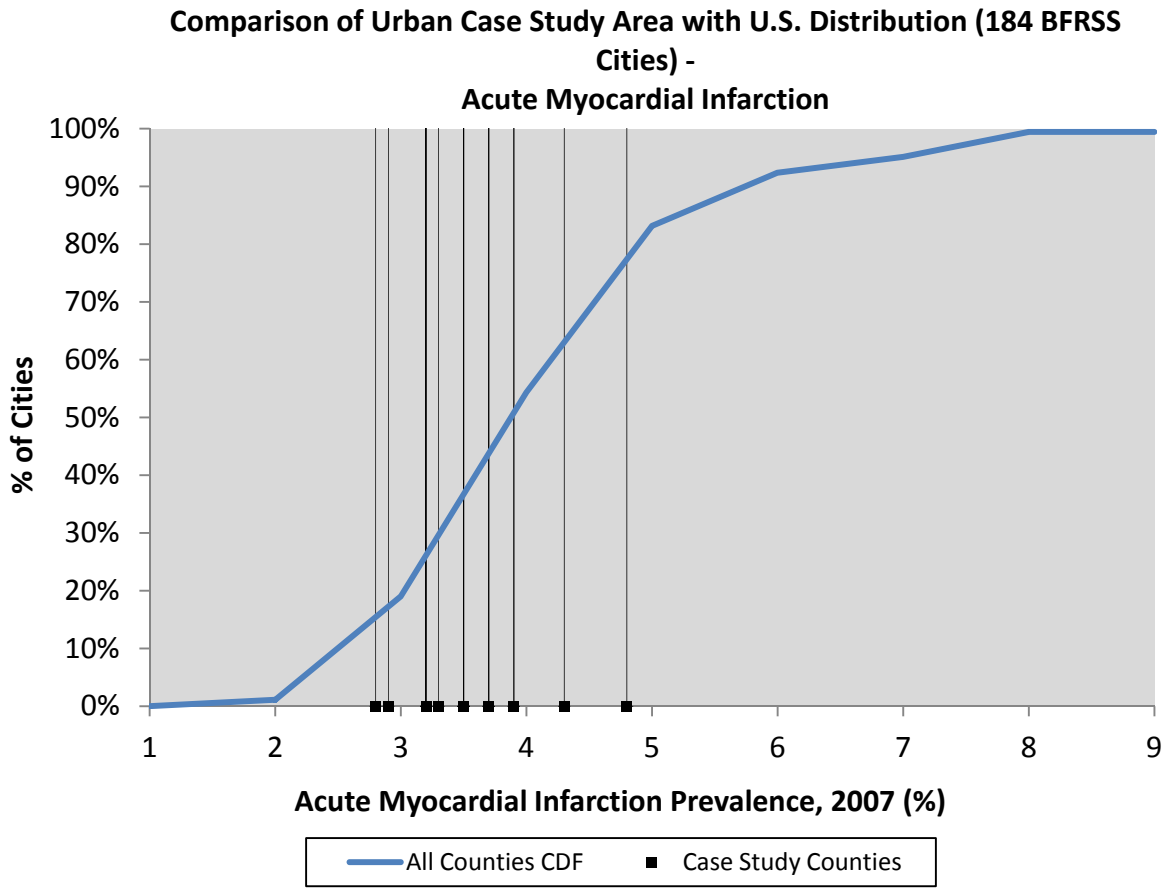

Figure 8-0.25 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Acute myocardial infarction prevalence

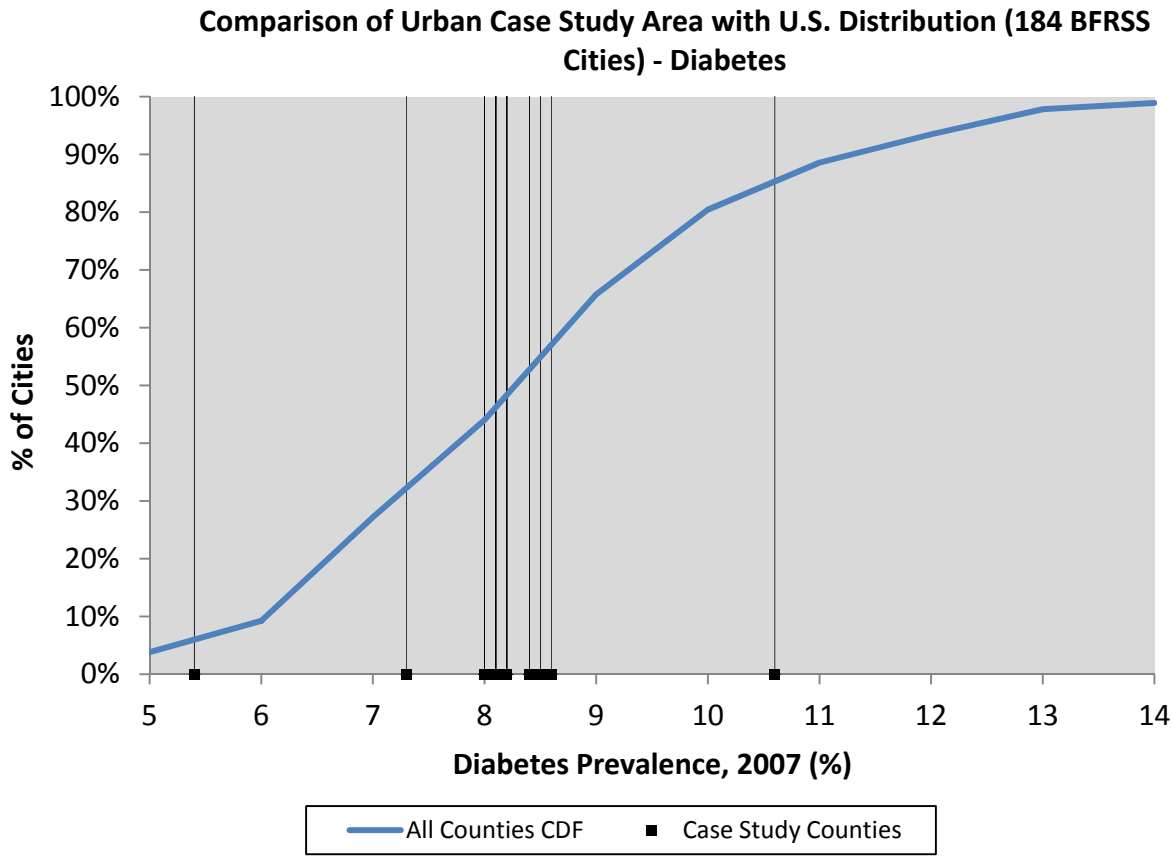

Figure 8-0.26 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Diabetes prevalence

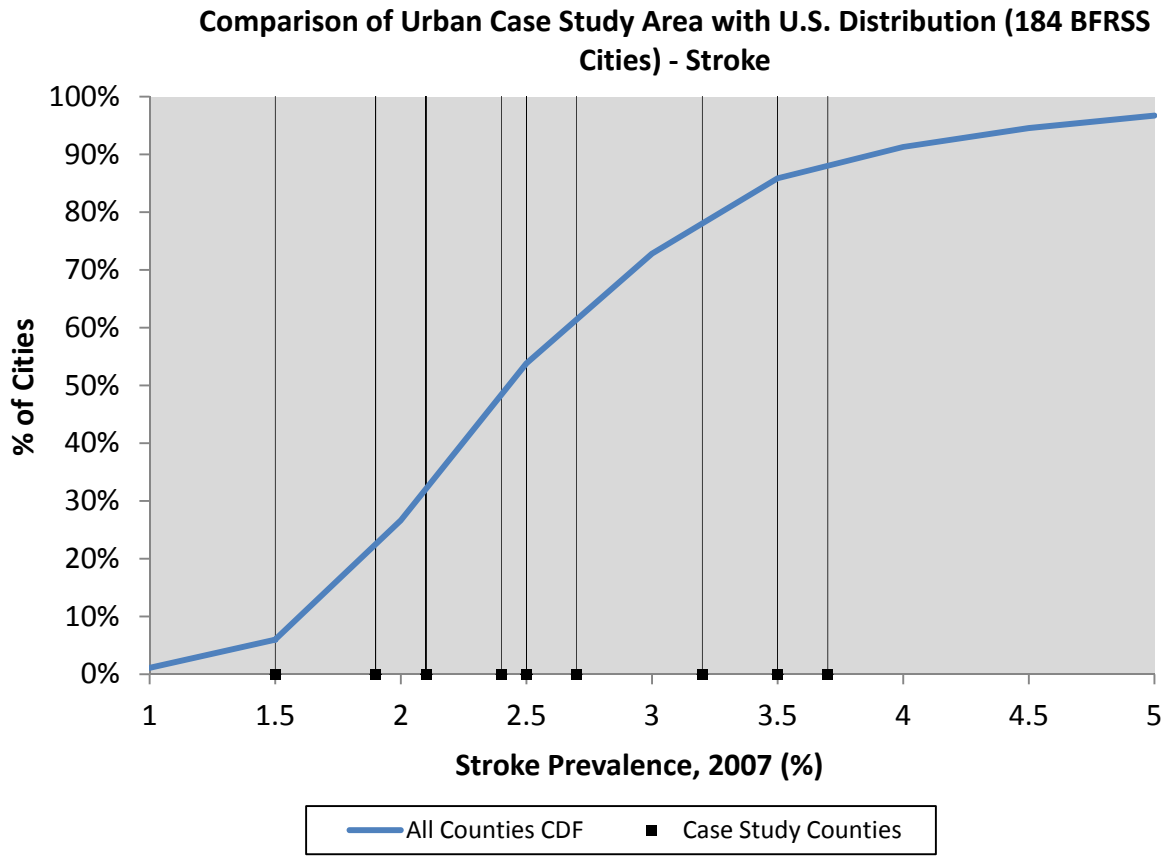

Figure 8-0.27 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Stroke prevalence

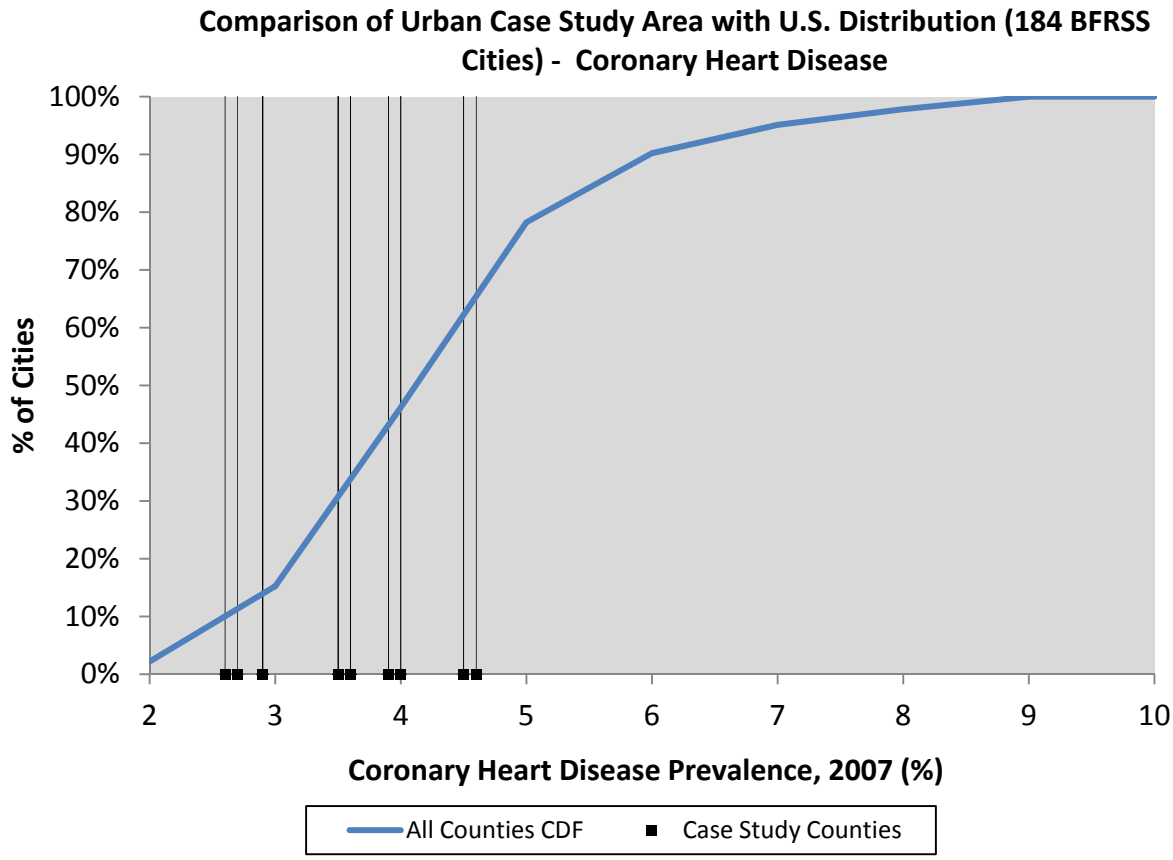

Figure 8-0.28 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Coronary heart disease prevalence

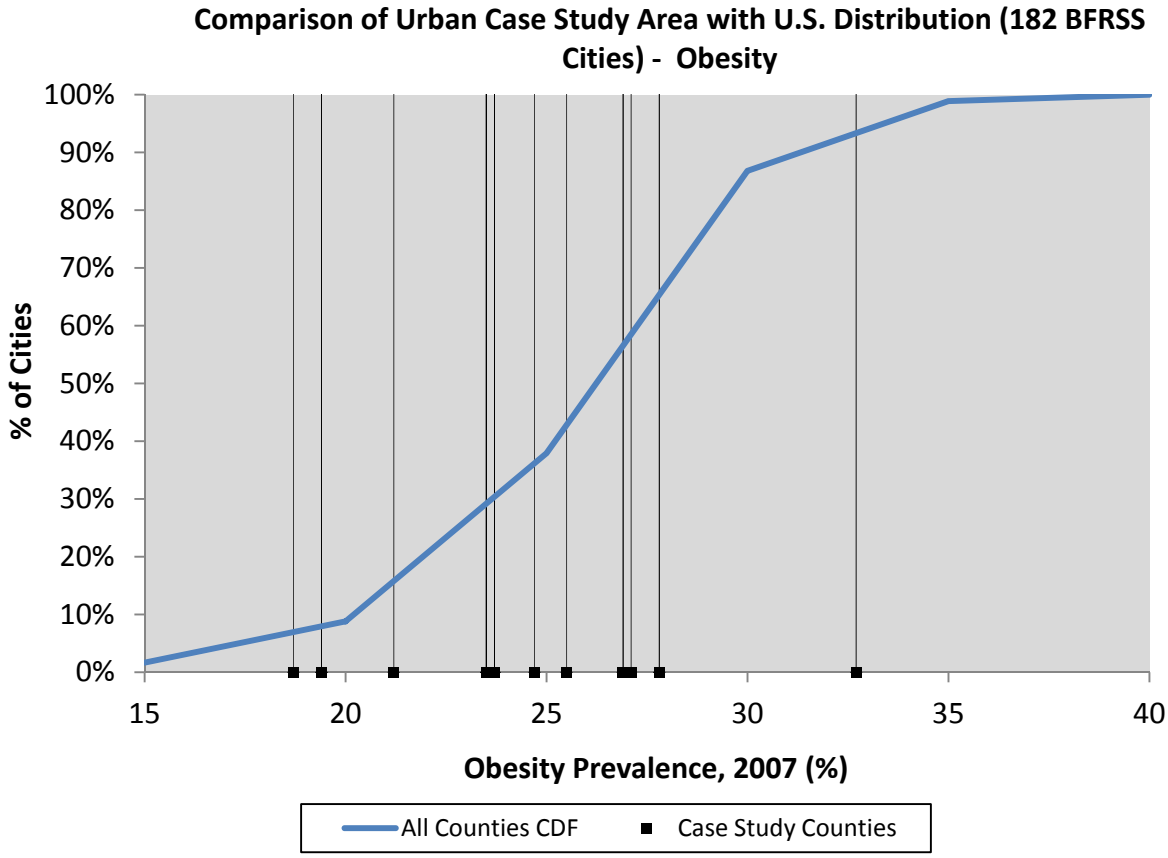

Figure 8-0.29 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Obesity prevalence

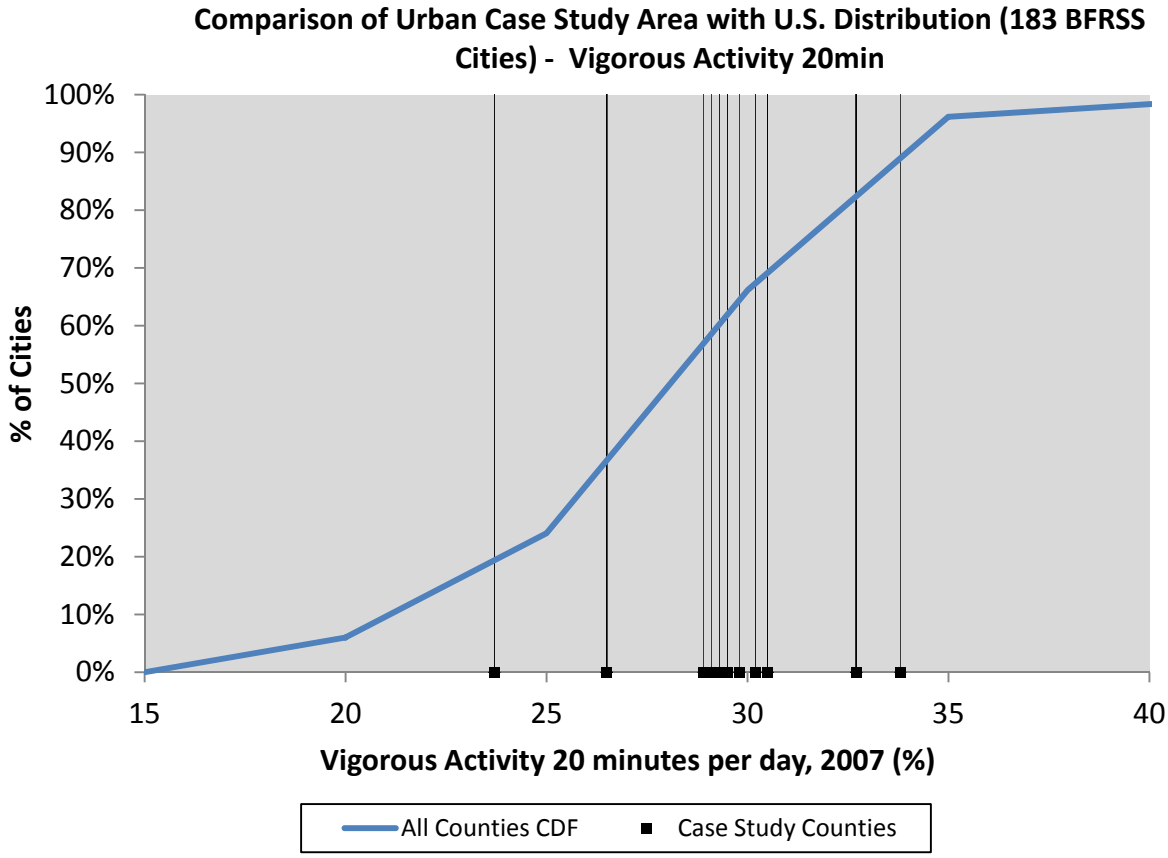

Figure 8-0.30 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Vigorous activity at least 20 minutes per day

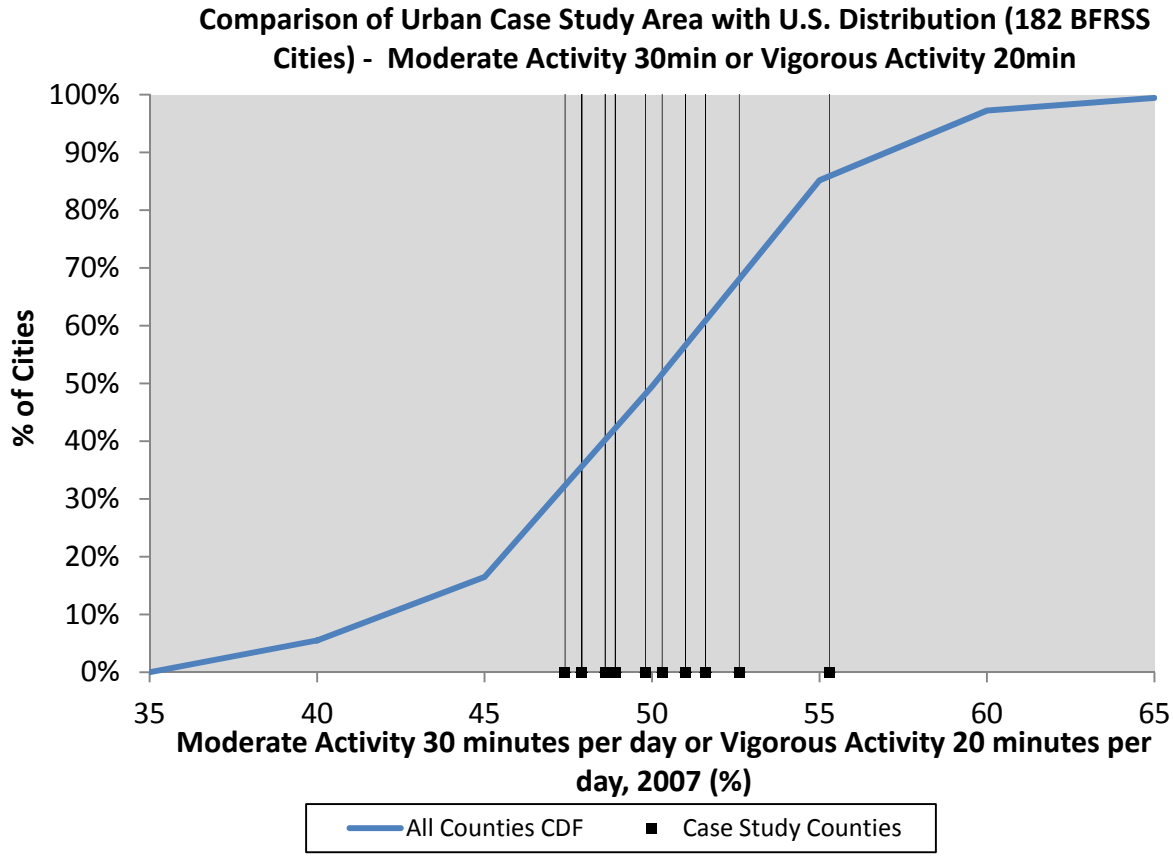

Figure 8-0.31 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Moderate activity at least 30 minutes per day or vigorous activity at least 20 minutes per day

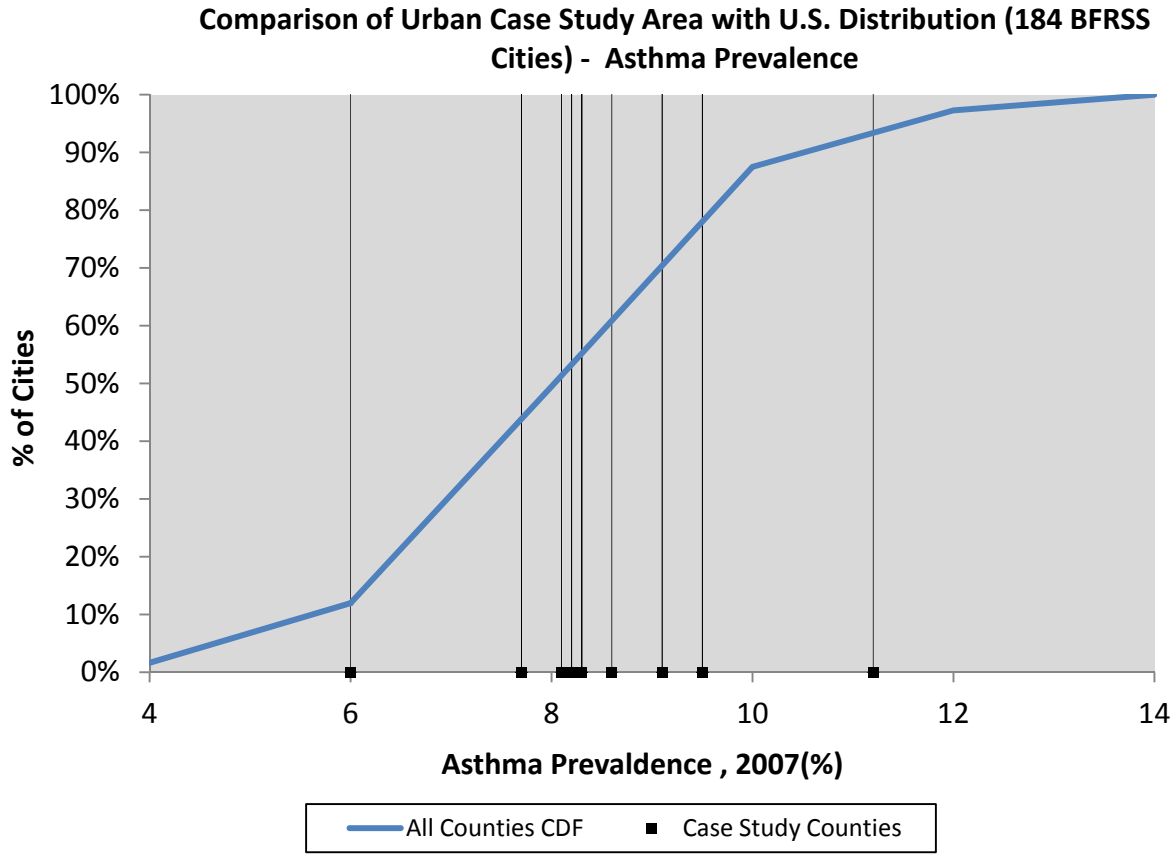

Figure 8-0.32 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Asthma prevalence

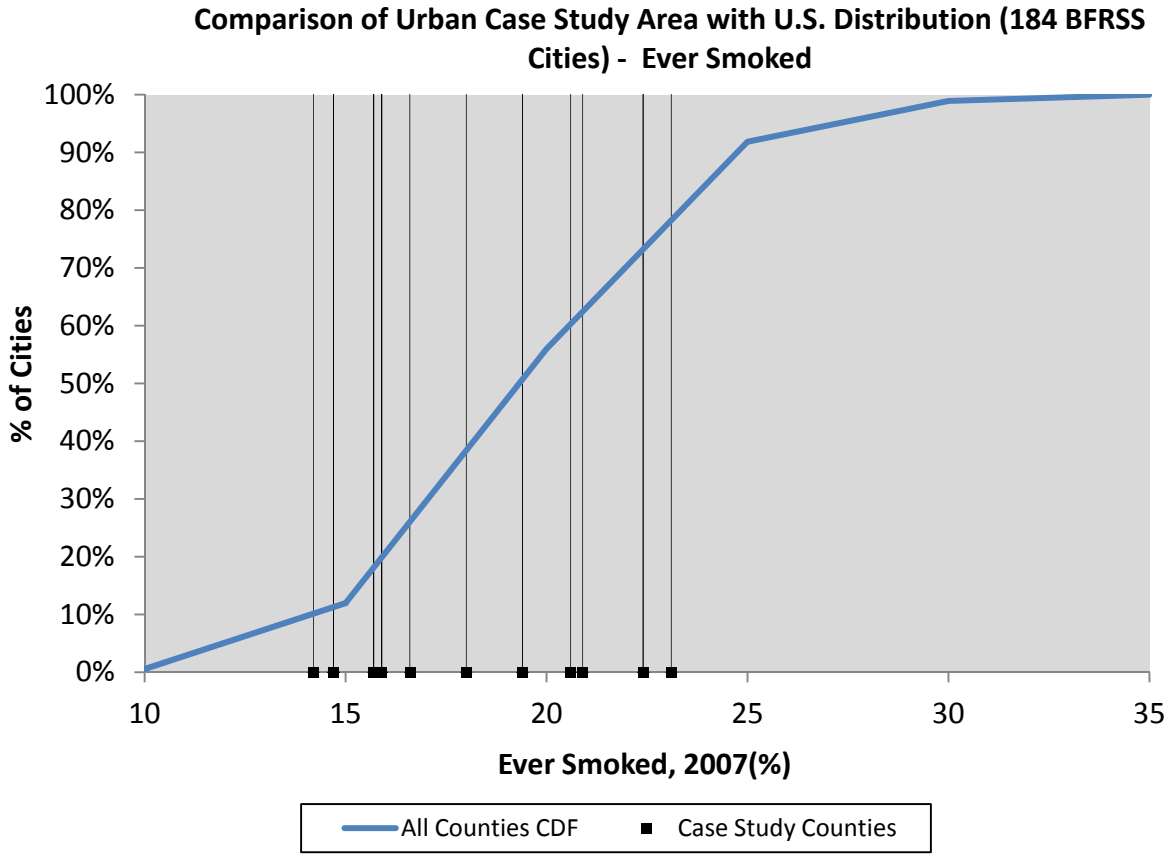

Figure 8-0.33 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Smoking prevalence

iii. Air Quality and Climate Variables

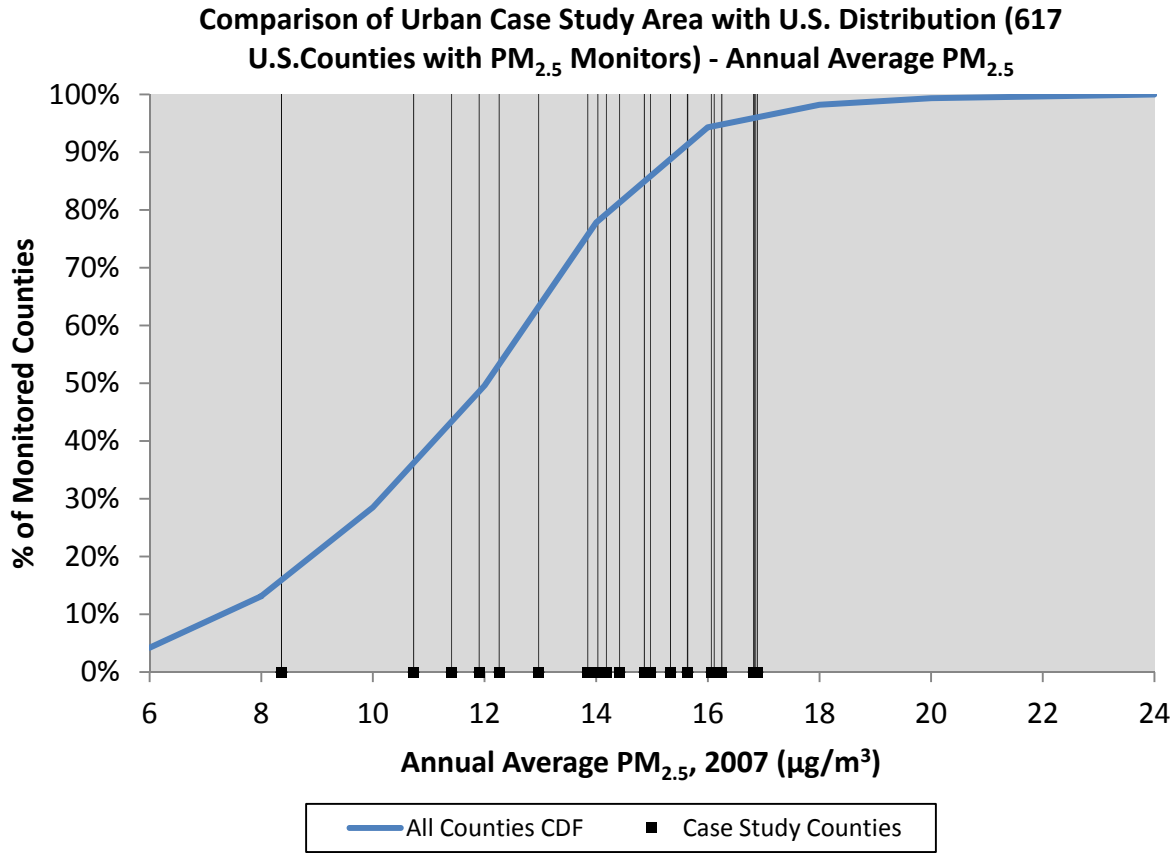

Figure 8-0.34 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Annual average $PM_{2.5}$ concentration

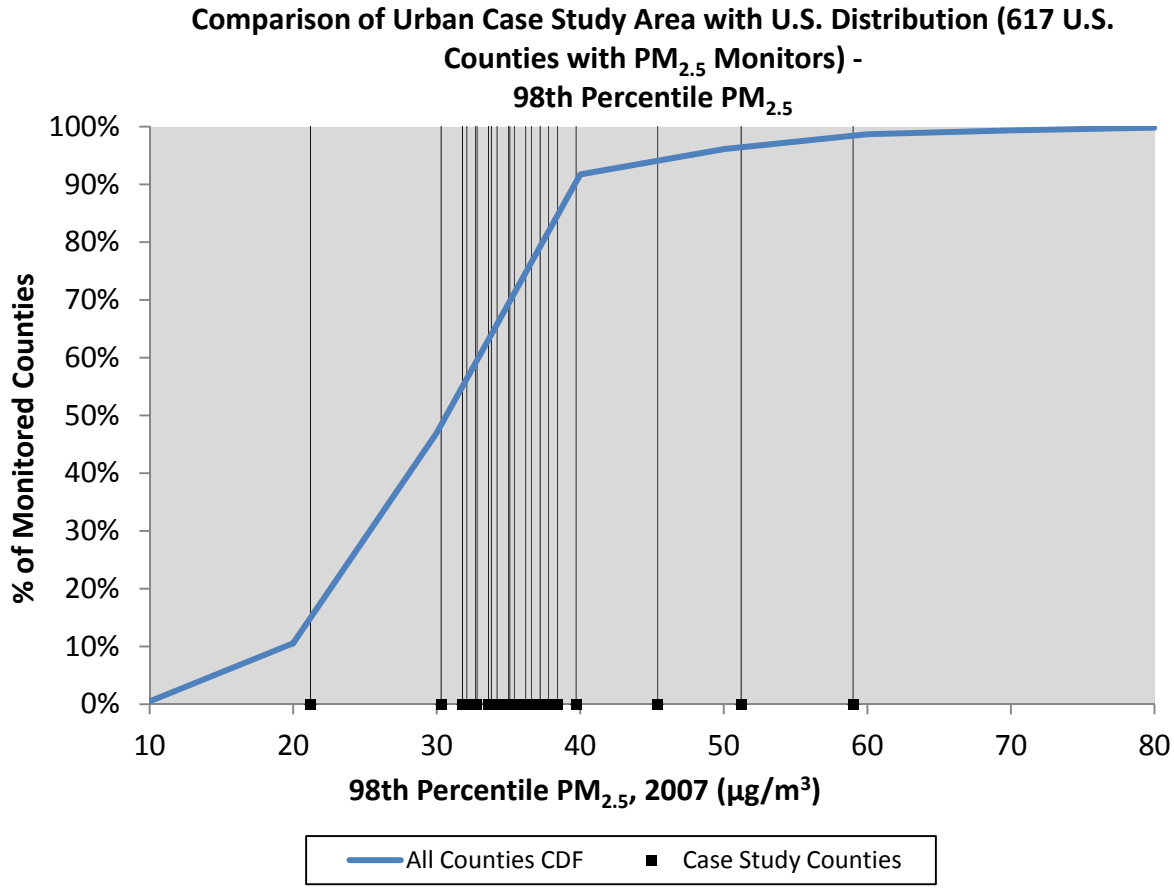

Figure 8-0.35 Comparison of distributions for selected variables expected to influence the relative risk from ozone: 98th percentile $PM_{2.5}$ concentration

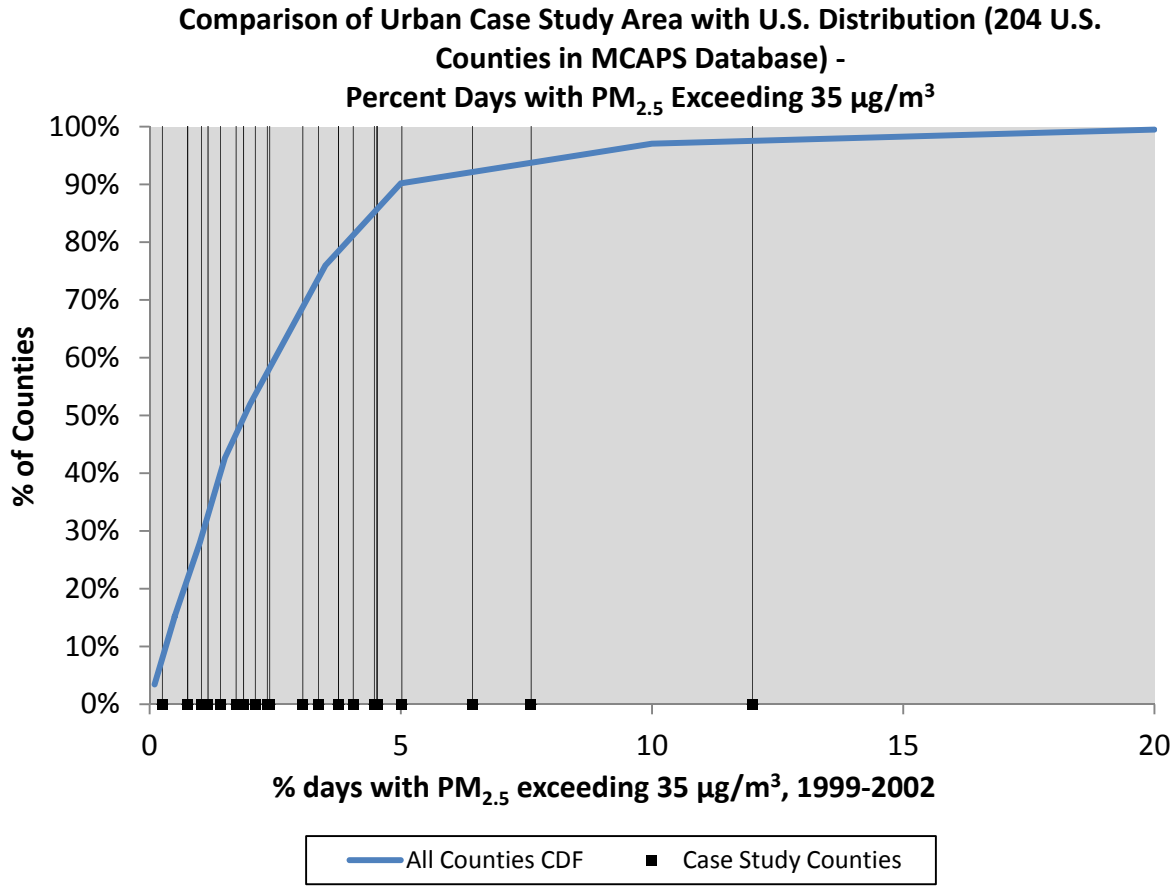

Figure 8-0.36 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Percent of days with $PM_{2.5}$ exceeding 35 µg/m^3

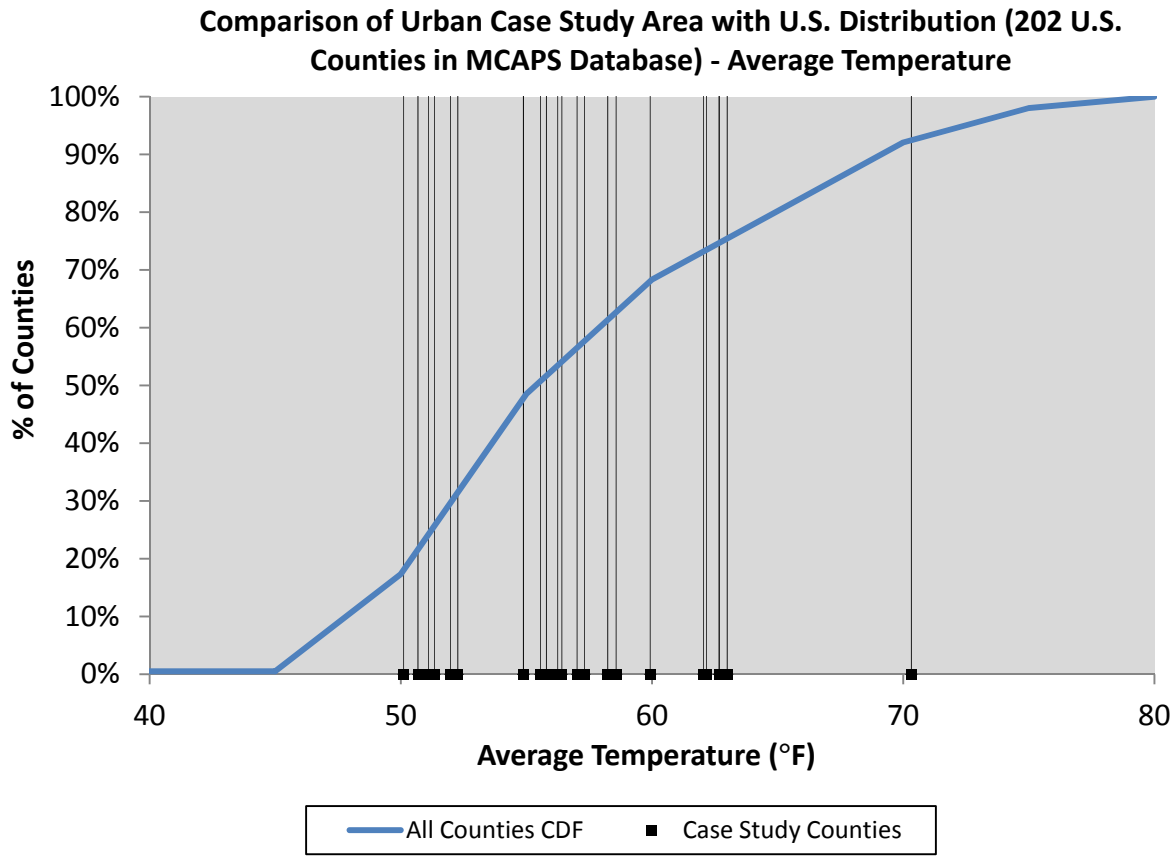

Figure 8-0.37 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Average temperature

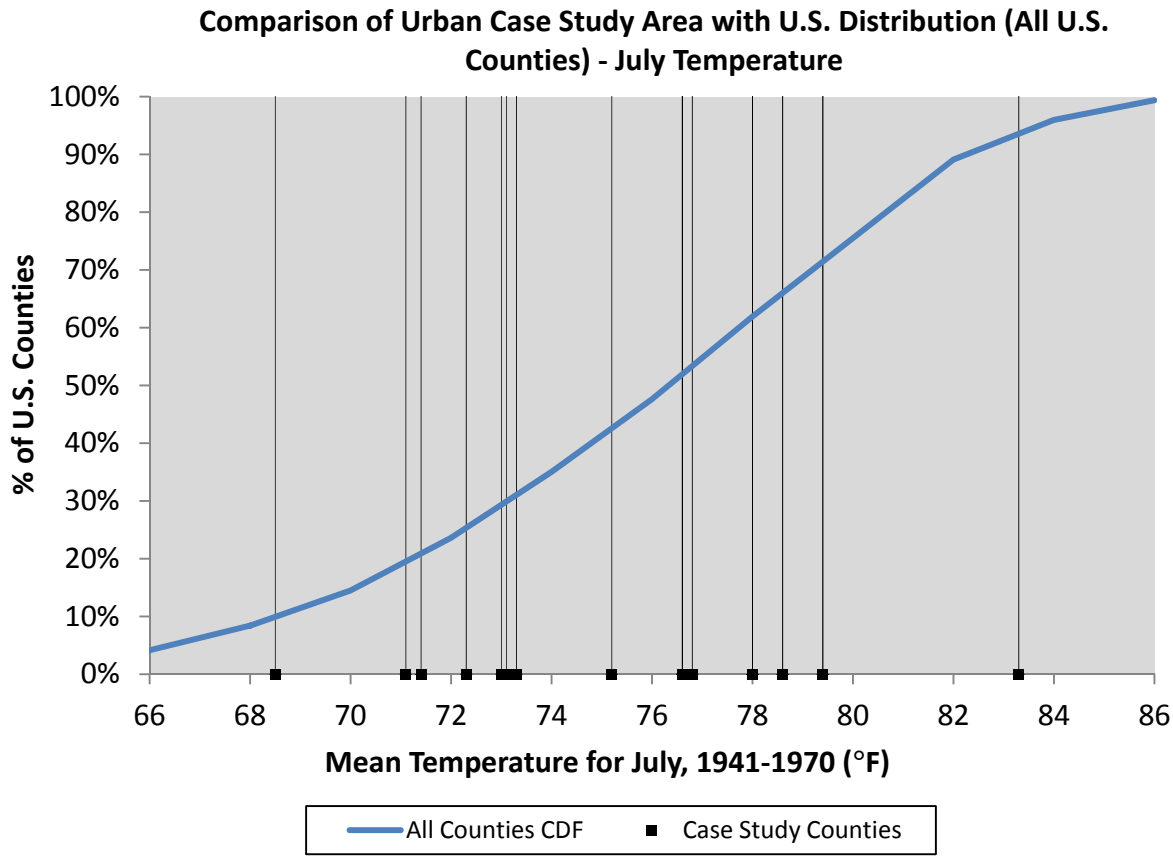

Figure 8-0.38 Comparison of distributions for selected variables expected to influence the relative risk from ozone: July temperature

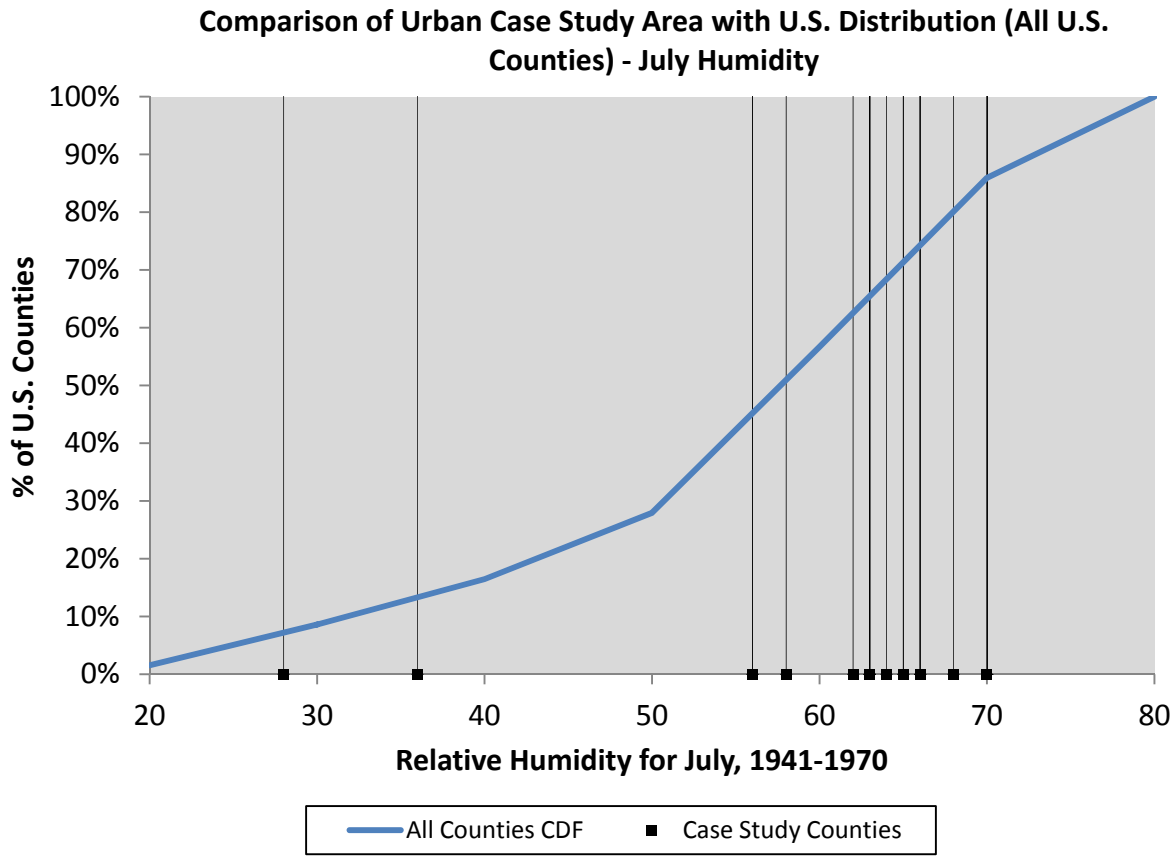

Figure 8-0.39 Comparison of distributions for selected variables expected to influence the relative risk from ozone: Relative humidity

United States
Environmental Protection
Agency

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

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