Ozone Health Risk Assessment for Selected Urban Areas: Draft Report

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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). Sections 108 and 109 of the Clean Air Act (Act) 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.[1] 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).

EPA's overall plan and schedule for this O_3 NAAQS review is presented in a *Plan for Review of the National Ambient Air Quality Standards for Ozone* (EPA, 2005a), which is available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_pd.html . That plan discusses the preparation of two key documents in the NAAQS review process: an Air Quality Criteria Document (AQCD) and a Staff Paper. The AQCD provides a critical assessment of the latest available scientific information upon which the NAAQS are to be based, and the Staff Paper evaluates the policy implications of the information contained in the AQCD and presents staff conclusions and recommendations for standard-setting options for the Administrator to consider. In conjunction with preparation of the Staff Paper, staff in EPA's Office of Air Quality Planning and Standards (OAQPS) conducts various policy-relevant assessments, including in this review a quantitative exposure analysis and a human health risk assessment. Both the exposure analysis and the risk assessment require a quantitative analysis of O_3 air quality. The methods and results of this analysis are described in Chapter 2 of the draft Staff Paper (EPA, 2005b) (hereafter "draft Staff Paper") and in Fitz-Simons et al. (2005). The methods and results of the modeling of personal exposures are discussed in Chapter 4 of the draft Staff Paper and in an accompanying technical support document (EPA, 2005c). The methods and results of the human health risk assessment are described in this draft document.

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[1]Section 109(b)(1) [42 U.S.C. 7409] of the Act defines a primary standard as one "the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health."

As part of the last O_3 NAAQS review, EPA conducted exposure analyses for the general population; children, who spend more time outdoors; and outdoor workers. Exposure estimates were generated for 9 urban areas for existing (referred to as "as is") air quality and for just meeting the existing 1-hour standard and several alternative 8-hour standards. Several reports (Johnson et al., 1996a,b,c; Johnson, 1997) that describe these analyses can be found at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr_td.html. EPA also conducted a health risk assessment that produced risk estimates for the number and percent of children experiencing lung function and respiratory symptoms associated with the exposures estimated for these same 9 urban areas. This portion of the risk assessment was based on exposure-response relationships developed from analysis of data from several controlled human exposure studies. The risk assessment for the last review also included risk estimates for excess respiratory-related hospital admissions related to O_3 concentrations for New York City based on a concentration-response relationship reported in an epidemiology study. Risk estimates for lung function decrements, respiratory symptoms, and hospital admissions were developed for "as is" air quality and for just meeting the existing 1-hour standard and several alternative 8-hour standards. Reports describing the health risk assessment (Whitfield et al., 1996; Whitfield, 1997) can be found at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr_td.html.

The health risk assessment described in this report builds upon the methodology and lessons learned from the exposure and risk work conducted for the last review. The current draft of this report is also based on the information currently available in the second external review draft of the O_3 AQCD (EPA, 2005d) (hereafter draft O_3 AQCD); as such, some aspects of the analysis may change based on changes that may be incorporated in the final O_3 AQCD.

The O_3 health risk assessment currently estimates the health effects associated with short-term exposures to O_3 under existing ("as is") air quality levels and upon just meeting the current O_3 primary NAAQS in selected sample urban areas. A subsequent draft will also include estimates of the health effects associated with short-term exposures to O_3 upon meeting various alternative standards in these sample urban areas. These assessments cover a variety of health effects for which there is adequate information to develop quantitative risk estimates. However, there are several health endpoints for which there currently is insufficient information to develop quantitative risk estimates. These additional health endpoints will be discussed qualitatively in the O_3 draft Staff Paper. The risk assessment is intended as a tool that, together with other information on these health endpoints and other health effects evaluated in the draft O_3 AQCD and draft Staff Paper, can aid the Administrator in judging whether the current primary standard protects public health with an adequate margin of safety, or whether revisions to the standard are appropriate.

The basic structure of the risk assessment reflects the two different types of studies on which the health risk assessment for O_3 is based: controlled human exposure studies, and epidemiological studies. This basic structure is described in Section 2. Section 3 describes the methods and results of that portion of the risk assessment based on controlled human exposure studies. Section 4 describes the methods and results of that portion of the risk assessment based on epidemiological studies.

2 BASIC STRUCTURE OF THE RISK ASSESSMENT

The health risk assessment described in this report estimated various health effects associated with O_3 exposures for recent ("as is") O_3 levels, based on 2004 air quality data, as well as the reduced risks for one O_3 season associated with just meeting the current 8-hour daily maximum O_3 NAAQS. Reduced risks associated with just meeting alternative O_3 standards will be estimated at a later time. Risk estimates were developed for 12 urban areas located throughout the U.S. Health endpoints examined in the risk assessment include: lung function decrements, respiratory- and cardiac-related hospital admissions, and mortality. Additional health endpoints, such as respiratory symptoms in asthmatic children, may be added at a later time.

At this time, two general types of human studies are particularly relevant for deriving quantitative relationships between O_3 levels and human health effects: controlled human exposure studies and epidemiological studies. Controlled human exposure 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 (FEV_1), respiratory symptoms, airway hyperresponsiveness, and inflammation. As noted above, 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. For the current health risk assessment, we used the exposure-response relationships developed during the last review, which was based on analysis of individual data that describes the relationship between a measure of personal exposure to O_3 and the measure(s) of lung function recorded in the study. 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, a 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 were derived through exposure modeling, as described in the accompanying draft technical support document (EPA, 2005c).

In contrast to the exposure-response relationships derived from controlled human exposure studies, epidemiological studies provide estimated concentration-response (C-R) relationships based on data collected in real world settings. Ambient O_3 concentration is typically measured as the average of monitor-specific measurements. Population health responses for O_3 have included hospital admissions for respiratory and cardiac illness and premature mortality. As described more fully below, a risk assessment based on epidemiological studies requires baseline incidence rates and population data for the risk assessment locations.

The characteristics that are relevant to carrying out a risk assessment based on controlled human exposure studies versus one based on epidemiology studies can be summarized as follows:

- A risk assessment based on controlled human exposure studies uses exposure-response functions, and therefore requires as input (modeled) personal exposures to O_3. A risk assessment based on epidemiology studies uses C-R functions, and therefore requires as input (monitored) ambient O_3 concentrations.

- Epidemiological studies are carried out in specific real world locations (e.g., specific urban areas). A risk assessment focused on locations in which the epidemiologic studies providing the C-R functions were carried out will minimize uncertainties. 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 location for which there are adequate air quality data on which to base the modeling of personal exposures.

- The adequate modeling of hourly personal exposures associated with ambient concentrations requires more complete ambient monitoring data than are necessary to estimate average ambient concentrations used to calculate risks based on C-R relationships. Therefore, there may be some locations in which an epidemiological studies-based risk assessment could appropriately be carried out but a controlled human exposure studies-based risk assessment would introduce significant additional uncertainty.
- To derive estimates of risk from C-R relationships estimated in epidemiological studies, it is usually necessary to have estimates of the baseline incidences of the health effects involved. Such baseline incidence estimates are not needed in a controlled human exposure studies-based risk assessment.

The methods and results for the two parts of the risk assessment – the part based on controlled human exposure studies and the part based on epidemiological studies – are discussed in Sections 3 and 4 below.

3 ASSESSMENT OF RISK BASED ON CONTROLLED HUMAN EXPOSURE STUDIES

3.1 Methods

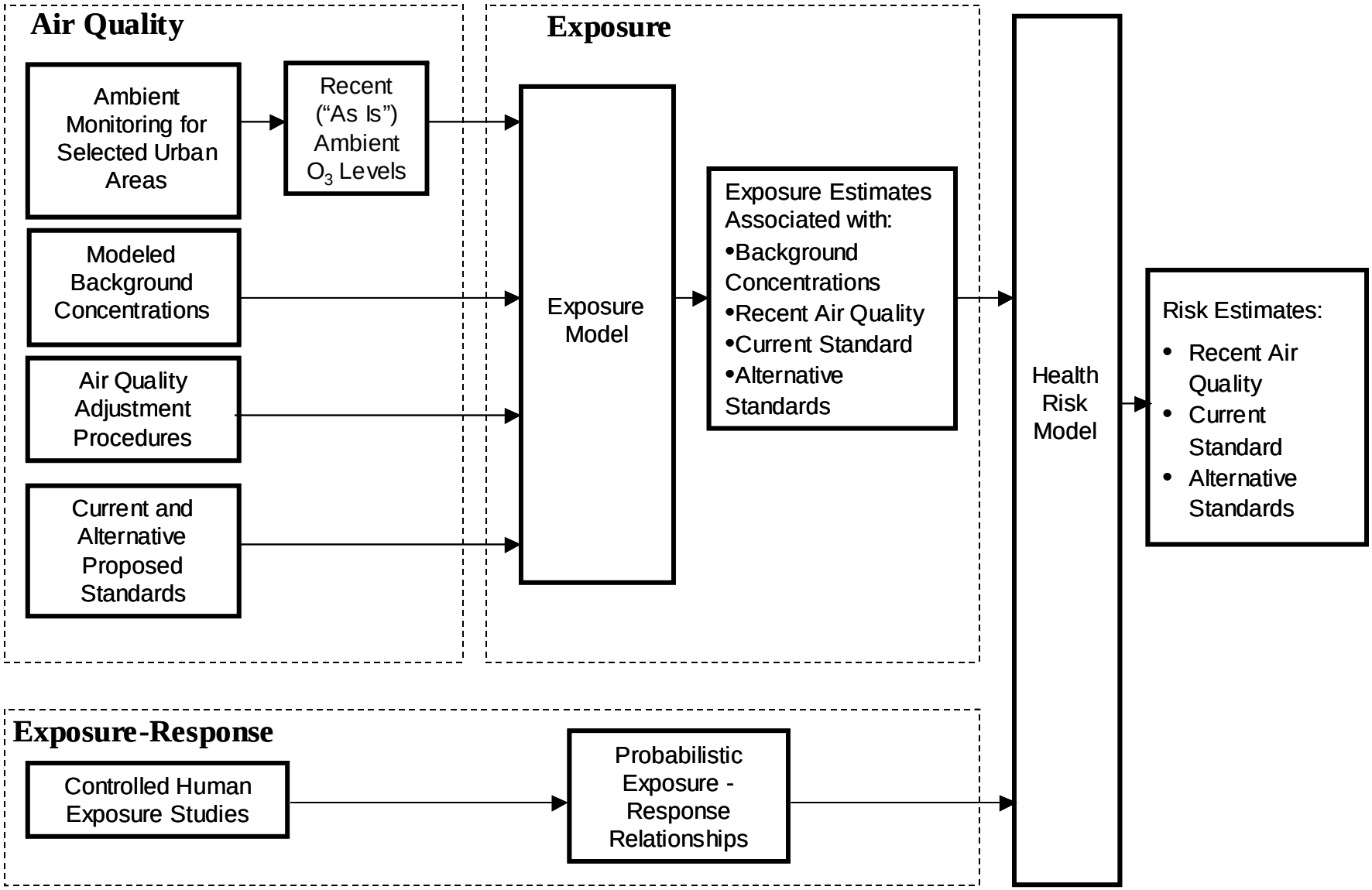
The major components of the part of the health risk assessment based on data from controlled human exposure studies are illustrated in Figure 3-1. The air quality and exposure analysis components that are integral to this part of the risk assessment are discussed in Chapters 2 and 4, respectively, of the draft Staff Paper. As described in the draft O_3 AQCD, there are numerous controlled human exposure studies reporting lung function decrements (as measured by changes in FEV_1), other measures of lung function, airway responsiveness, respiratory symptoms, and various markers of inflammation. Most of these studies have involved voluntary exposures with healthy adults, although a few studies have been conducted with mild and moderate asthmatics and one study reported lung function decrements for children 8-11 years old (McDonnell et al., 1985a) at a single exposure level.

3.1.1 Selection of health endpoints

In the last review, the health risk assessment estimated both lung function decrements ($\geq$10, $\geq$15, and $\geq$20% changes in FEV_1) and respiratory symptoms in children 6-18 years old associated with 1-hour exposures at moderate and heavy exertion and 8-hour exposures at moderate exertion. At that time EPA staff and the CASAC O_3 Panel judged that it was reasonable to estimate the exposure-response relationships for children 6-18 years old based on data from adult subjects (18-35 years old). As discussed in the 1996 O_3 Staff Paper (EPA, 1996a) and 1996 O_3 AQCD (EPA, 1996b), findings from other chamber studies (McDonnell et al., 1985a) for children 8-11 years old for a single exposure level and summer camp field studies involving children exposed to ambient O_3 in at least six different locations in the United States and Canada found lung function changes in healthy children similar to those observed in healthy adults exposed to O_3 under controlled chamber conditions. We are using the same approach in this assessment.

In the prior risk assessment, EPA estimated risk for lung function decrements associated with 1-hour heavy exertion, 1-hour moderate exertion, and 8-hour moderate exertion exposures. Since the 8-hour moderate exertion exposure scenario clearly resulted in the greatest health risks in terms of lung function decrements, EPA staff has chosen to include only the 8-hour moderate exertion exposures in the current risk assessment for this health endpoint. As discussed in Chapter 4 of the draft Staff Paper, levels of physical activity were categorized by a daily Physical Activity Index (PAI). Children were characterized as active if their median daily PAI over the period modeled was 1.75 or higher, a level characterized by exercise physiologists as being "moderately active" or "active."

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



Although respiratory symptoms in healthy children were estimated in the last review, EPA staff has decided not to estimate respiratory symptoms in healthy children given the lack of symptoms found in field studies examining responses in healthy children published since the prior review. The draft O_3 AQCD concludes that "collectively, these studies indicate that there is no consistent evidence of an association between O_3 and respiratory symptoms among healthy children" (p. 7-48). While a number of controlled human exposure studies have been published since the last review reporting various other acute effects, including airway responsiveness and increases in inflammatory indicators, none of these studies were conducted at multiple concentration levels within the range of greatest interest (i.e., below 0.12 ppm). Thus, EPA staff has decided to limit this portion of the risk assessment to lung function decrements in children and to again base the exposure-response relationships on data obtained for 18-35 year old subjects.

3.1.2 Development of exposure-response functions

We used a similar methodology to that used in the prior risk assessment (see Appendices A and B in Whitfield et al., 1996) to estimate probabilistic exposure-response relationships for lung function decrements associated with 8-hour moderate exertion exposures. The combined data set from the Folinsbee et al. (1988), Horstman et al. (1990), and McDonnell et al. (1991) studies provide three data points – lung function decrements associated with each of three O_3 concentrations (0.08, 0.10, and 0.12 ppm) – for each of the three measures of lung function decrement listed above ($\geq$10, $\geq$15, and $\geq$20% changes in FEV_1). Before being used to estimate exposure-response relationships for 8-hour exposures, the data from these controlled human exposure studies were corrected for the effect of exercise in clean air to remove any systematic bias that might be present in the data attributable to an exercise effect. Generally, this correction for exercise in clean air is small relative to the total effects measures in the O_3-exposed cases. Regression techniques were then used to fit a function to the data for each of the three measures of lung function decrement. In each case, a linear function provided a good fit.[2]

3.1.3 Approach to calculating risk estimates

We have generated several risk measures for this portion of the risk assessment. In addition to the estimates of the number of school age children and active children experiencing 1 or more occurrences of a lung function decrement $\geq$ 10%, $\geq$ 15% and $\geq$ 20% in an O_3 season, risk estimates have been developed for the total number of occurrences of these lung function decrements in school age children and active school age children. The mean number of occurrences per child has been calculated to provide an indicator of the average number of times that a responder would experience the specified effect during an O_3 season.

[2] As noted in Whitfield et al., 1996, the response data point associated with 0.12 ppm for the response measure FEV1 $\geq$ 15% appeared to be inconsistent with the other data points (see Whitfield et al., 1996, Table 10, footnote c). Because of this, we estimated the probability of a response of FEV1 $\geq$ 15% at an O_3 concentration of 0.12 ppm by interpolating between the FEV1 $\geq$ 10% and FEV1 $\geq$ 20% response rates at that O_3 concentration.

A headcount risk estimate for a given lung function decrement (e.g., ≥20% change in FEV_1) is an estimate of the expected number of people who will experience that lung function decrement. To obtain risk estimates associated with ozone concentrations in excess of policy relevant background (PRB) concentrations, we have (1) estimated expected risk, given the personal exposures associated with "as is" ambient O_3 concentrations, (2) estimated expected risk, given the personal exposures associated with estimated background ambient O_3 concentrations, and (3) subtracted the latter from the former. The headcount risk is then calculated by multiplying the resulting expected risk by the number of people in the relevant population. Because response rates are calculated for 21 fractiles, estimated headcount risks are similarly fractile-specific.

The risk (i.e., expected fractional response rate) for the k^{th} fractile, R_k is:

$$R_k = \sum_{j=1}^{N} P_j \; x \; (RR_k \mid e_j) \quad - \quad \sum_{i=1}^{N_b} P_i^b \; x \; (RR_k \mid e_i^b) \quad \text{(Equation 3-1)}$$

where:

e_j = (the midpoint of) the jth category of personal exposure to ozone, given "as is" ambient O_3 concentrations;

e_i^b = (the midpoint of) the ith category of personal exposure to ozone, given background ambient O_3 concentrations;

P_j = the fraction of the population having personal exposures to O_3 concentration of e_j ppm, given "as is" ambient O_3 concentrations;

P_i^b = the fraction of the population having personal exposures to O_3 concentration of e_i^b ppm, given background ambient O_3 concentrations;

$RR_k \mid e_j$ = k-fractile response rate at O_3 concentration e_j;

$RR_k \mid e_i^b$ = k-fractile response rate at O_3 concentration e_i^b; and

N = number of intervals (categories) of O_3 personal exposure concentration, given "as is" ambient O_3 concentrations; and

N_b = number of intervals of O_3 personal exposure concentration, given background ambient O_3 concentrations.

For example, if the median expected response rate given "as is" ambient concentrations is 0.065 (i.e., the median expected fraction of the population responding is 6.5%) and the median expected response rate given background ambient concentrations is 0.001 (i.e., the median

expected fraction of the population responding is 0.1%), then the median expected response rate associated with "as is" ambient concentrations above PRB concentrations is 0.065 – 0.001 = 0.064. If there are 300,000 people in the relevant population, then the headcount risk is 0.064 x 300,000 = 19,200.

3.1.4 Selection of urban areas

EPA staff chose to develop lung function decrement risk estimates for school age children and active school age children living in 12 urban areas in the U.S. Since the exposure-response functions for lung function decrements based on the controlled human exposure studies were based on controlled laboratory conditions, the location of these studies played no role in selecting urban locations for the risk assessment. Instead, several criteria and considerations guided the selection of urban areas for the risk assessment, including the following:

- The overall set of urban locations should represent a range of geographic areas, urban population demographics, and climatology, and be focused on areas that do not meet the current 8-hour O_3 NAAQS.
- The largest areas with major O_3 nonattainment problems should be included.
- There must be sufficient air quality data for the three-year period (2002 - 2004).

Several additional criteria, which apply to the epidemiology-based portion of the risk assessment, are discussed below in Section 4.1.4. Because the same 12 urban areas were used in both the controlled human studies- and the epidemiological studies-based portions of the risk assessment, these additional criteria further limited the choice of urban areas for which to develop lung function decrement risk estimates.

For the purposes of estimating population exposure and the risk of lung function decrements associated with these population exposure estimates, the 12 urban areas were defined based on consolidated statistical areas (CSAs). In contrast, for the risk estimates for premature mortality and excess hospital admissions based on C-R relationships estimated in epidemiological studies, the urban areas were defined to be generally consistent with the geographic boundaries used in those studies. Risk estimates in both the controlled human studies-based portion and the epidemiology-based portion of the O_3 risk assessment are based on the months of April through September, rather than the actual location-specific O_3 seasons. The CSAs and their actual O_3 seasons are shown in Table 3-1. The populations of school age and active school age children in these areas are shown in Table 3-2.

3.1.5 Addressing variability and uncertainty

Any estimation of risk and reduced risks associated with just meeting the current O_3 standards should address both the variability and uncertainty that generally underlie such an analysis. *Uncertainty* refers to the lack of knowledge regarding the actual values of model input variables (parameter uncertainty) and of physical systems or relationships (model uncertainty – e.g., the shapes of exposure-response and concentration-response functions). The goal of the analyst is to reduce uncertainty to the maximum extent possible. Uncertainty can be reduced by

Table 3-1. Urban Areas Used in the Controlled Human Studies-Portion of the O_3 Risk Assessment and Their O_3 Seasons

Urban Area (CSA)	O_3 Season
Atlanta-Sandy Springs-Gainesville, GA-AL	March 1 to Oct. 31
Boston-Worcester-Manchester, MA-NH	April 1 to Sept. 30
Chicago-Naperville-Michigan City, IL-IN-WI	April 1 to Sept. 30
Cleveland-Akron-Elyria, OH	April 1 to Oct. 31
Detroit-Warren-Flint, MI	April 1 to Sept. 30
Houston-Baytown-Huntsville, TX	Jan. 1 to Dec. 30
Los Angeles-Long Beach-Riverside, CA	Jan. 1 to Dec. 30
New York-Newark-Bridgeport, NY-NJ-CT-PA	April 1 to Sept. 30
Philadelphia-Camden-Vineland, PA-NJ-DE-MD	April 1 to Oct. 31
Sacramento--Arden-Arcade--Truckee, CA-NV	Jan. 1 to Dec. 30
St. Louis-St. Charles-Farmington, MO-IL	April 1 to Oct. 31
Washington-Baltimore-N. Virginia, DC-MD-VA-WV	April 1 to Oct. 31

Table 3-2. Population Coverage of Modeled Areas

Urban Area (CSA)	Modeled population (thousands)	Modeled children (thousands)	Active children (thousands)
Atlanta	4,548	942	519
Boston	5,714	1,098	529
Chicago	9,311	1,946	933
Cleveland	2,945	582	295
Detroit	5,357	1,110	553
Houston	4,815	1,076	598
Los Angeles	16,349	3,594	1,951
New York	21,357	4,084	2,009
Philadelphia	5,832	1,179	609
Sacramento	1,930	418	226
St. Louis	2,754	572	309
Washington, DC	7,572	1,473	759

improved measurement and improved model formulation. In a health risk assessment, however, significant uncertainty often remains.

The degree of uncertainty can be characterized, sometimes quantitatively. For example, the statistical uncertainty surrounding the estimated O_3 coefficients in the exposure-response functions is reflected in confidence or credible intervals provided for the risk estimates.

A Bayesian approach was used to characterize uncertainty attributable to sampling error based on sample size considerations at each of the three O_3 concentrations for which there were data from the underlying studies (0.08, 0.10, and 0.12 ppm). Using diffuse Beta distributions as prior distributions, the resulting posterior distributions are also Beta distributions (see Appendix A in Whitfield et al., 1996). We calculated response rates for 21 fractiles (for cumulative probabilities from 0.05 to 0.95 in steps of 0.05, plus probabilities of 0.01 and 0.99) using these posterior Beta distributions.

Because there are no response data for O_3 concentrations other than 0.08, 0.10, and 0.12 ppm, we had to use a different approach to derive response rates for the 21 fractiles at other O_3 concentrations. For each of the 21 fractiles, we used regression techniques to fit a linear function through the three points generated at O_3 concentrations of 0.08, 0.10, and 0.12 ppm. If the probability of response thus estimated for a given fractile at a given O_3 concentration was less than 0.0, it was set equal to 0.0. Similarly, if the probability of response thus estimated was greater than 1.0, it was set equal to 1.0. For example, for the exposure-response function for $\geq$20% changes in FEV_1, the tenth percentile (fractile) responses at O_3 concentrations of 0.08, 0.10, and 0.12 ppm (generated using the Bayesian approach described above) were 0.042, 0.081, and 0.112, respectively. A linear regression using the three data points (0.08, 0.042), (0.10, 0.081), and (0.12, 0.112) generated the linear function:

$$\text{Response} = 1.7665*(O_3 \text{ concentration}) - 0.09837.$$

For an O_3 concentration of 0.09 ppm, the response is 1.7665*(0.09) -0.09837 = 0.0606 – i.e., the tenth fractile probability of a response of $\geq$20% in FEV_1 associated with an exposure to an O_3 concentration of 0.09 ppm is about 6 percent. The tenth fractile response to an O_3 exposure of 0.04 ppm is 1.7665*(0.04) -0.09837 = -0.0277, which was set equal to 0.0. Response rates can similarly be calculated for all 21 fractiles at any specified O_3 concentration.

In addition to uncertainties arising from sample size considerations, other uncertainties associated with the use of the exposure-response relationships for lung function responses are briefly summarized below. Additional uncertainties with respect to the exposure inputs to the risk assessment are described in Chapter 4 of the draft Staff Paper and in the draft Exposure Assessment TSD (EPA 2005e). The main additional uncertainties with respect to the approach used to estimate exposure-response relationships include:

- Length of exposure. The 8-hour moderate exertion risk estimates are based on a combined data set from three controlled human exposure studies conducted using 6.6-hr exposures. The use of these data to estimate responses associated with an 8-hour exposure seem

reasonable, however, because lung function response appears to level off after exposure for 4 to 6 hours. It is unlikely that the exposure-response relationships would have been appreciably different had the studies been conducted over an 8-hour period.

- Extrapolation of exposure-response relationships. It was necessary to estimate responses at O_3 levels below the lowest exposure levels used in the controlled human studies (i.e., 0.08 ppm). In the prior review, the CASAC O_3 Panel supported EPA staff's decision to use a linear extrapolation approach down to background levels. Similarly, in the current assessment, the response has been extrapolated down to background levels.

- Reproducibility of O_3 induced responses. The risk assessment assumed that the O_3-induced responses for individuals are reproducible. This assumption is supported by the evaluation in the draft O_3 AQCD (see section AX6.4) which cites studies by McDonnell et al. (1985b) and Hazucha et al. (2003) as showing significant reproducibility of response.

- Age and lung function response. As in the prior review, exposure-response relationships based on controlled human exposure studies involving 18-35 year old subjects were used in the risk assessment to estimate responses for school age children (ages 5-18). This approach is supported by the findings of McDonnell et al. (1985a) who reported that children 8-11 year old experienced FEV_1 responses similar to those observed in adults 18-35 years old when both groups were exposed to concentrations of 0.12 ppm at an EVR of 35 L/min/m^2. In addition, a number of summer camp studies of school age children exposed in outdoor environments in the Northeast also showed O_3-induced lung function changes similar in magnitude to those observed in controlled human exposure studies.

- Exposure history. The risk assessment assumed that the O_3-induced response on any given day is independent of previous O_3 exposures. As discussed in Chapter 3 of the draft Staff Paper and in the draft O_3 AQCD, O_3-induced responses can be enhanced or attenuated as a result of recent prior exposures. The possible impact of exposure history on the risk estimates is an additional source of uncertainty that is not quantified in this assessment.

- Interaction between O_3 and other pollutants. Because the controlled human exposure studies used in the risk assessment involved only O_3 exposures, it was assumed that estimates of O_3-induced health responses would not be affected by the presence of other pollutants (e.g., SO_2, $PM_{2.5}$, etc). Some evidence exists that other pollutants may enhance the respiratory effects associated with exposure to O_3, but the evidence is not consistent across studies.

Variability refers to the heterogeneity in a population or parameter. Even if there is no uncertainty surrounding inputs to the analysis, there may still be variability. For example, there may be variability among exposure-response functions describing the relationship between O_3 and lung function across urban areas. Similarly, there may be variability among C-R functions describing the relationship between O_3 and mortality across urban areas. This variability does not imply uncertainty about the exposure-response or C-R function in any of the urban areas, but only that these functions are different in the different locations, reflecting differences in the populations and/or other factors that may affect the relationship between O_3 and the associated

health endpoint. In general, it is possible to have uncertainty but no variability (if, for instance, there is a single parameter whose value is uncertain) or variability but little or no uncertainty (for example, people's heights vary considerably but can be accurately measured with little uncertainty).

The current controlled human exposure studies portion of the risk assessment incorporates some of the variability in key inputs to the analysis by using location-specific inputs for the exposure analysis (e.g., location-specific population data, air exchange rates, air quality and temperature data). Although spatial variability in these key inputs across all U.S. locations has not been fully characterized, variability across the selected locations is imbedded in the analysis by using, to the extent possible, inputs specific to each urban area. Temporal variability is more difficult to address, because the risk assessment focuses on some unspecified time in the future. To minimize the degree to which values of inputs to the analysis may be different from the values of those inputs at that unspecified time, we have used the most current inputs available – for example, year 2004 air quality data for all of the urban locations, and the most recent available population data (from the 2000 Census). However, future changes in inputs have not been predicted (e.g., future population levels).

3.2 Results

Section 3.2.1 presents the results of the assessment of lung function decrement associated with exposure to "as is" O_3 concentrations (representing levels measured in 2004 for all of the assessment locations) over PRB levels, based on controlled human exposure studies. The corresponding results when O_3 concentrations just meet the current 8-hour daily maximum standard are presented in Section 3.2.2. All estimated numbers (of children and of occurrences) were rounded to the nearest 1000, and all percentages were rounded to one decimal place. These rounding conventions are not intended to imply confidence in that level of precision, but rather to avoid the confusion that can result when a greater amount of rounding is used.

3.2.1 Assessment of lung function decrement associated with exposure to "as is" O_3 concentrations in excess of policy relevant background levels

The estimated number and percent of occurrences of lung function decrement associated with exposure to "as is" O_3 concentrations over PRB concentrations among all school age children (ages 5 – 18) engaged in moderate exercise for at least one 8-hour period from April through September, 2004, is given in Table 3-3. The number and percent of these children estimated to experience at least one lung function decrement associated with exposure to "as is" O_3 concentrations over PRB concentrations is given in Table 3-4. Tables 3-5 and 3-6 give the corresponding results for active children. Results for all three measures of lung function decrement being considered in this analysis – decrements in FEV_1 of $\geq$10%, $\geq$15%, and $\geq$20% -- are shown in each table.

Table 3-3. Estimated Number and Percent of Occurrences of Lung Function Response Associated with Exposure to "As Is" O_3 Concentrations Over Background O_3 Concentrations Among All Children (Ages 5-18) Engaged in Moderate Exercise: April - September, 2004*

Location	Response = Decrease in FEV_1 Greater Than or Equal to:					
	10%		15%		20%	
	Number (1000s)	Percent	Number (1000s)	Percent	Number (1000s)	Percent
Atlanta-Sandy_Springs-Gainesville__GA-AL	2165 (1094 - 4258)	3.1% (1.6% - 6.1%)	738 (320 - 1735)	1.1% (0.5% - 2.5%)	52 (8 - 218)	0.1% (0% - 0.3%)
Boston-Worcester-Manchester__MA-NH	1829 (860 - 3855)	2.4% (1.1% - 5%)	533 (223 - 1350)	0.7% (0.3% - 1.7%)	27 (4 - 126)	0% (0% - 0.2%)
Chicago-Naperville-Michigan_City__IL-IN-WI	3066 (1432 - 6388)	2.2% (1% - 4.6%)	847 (340 - 2244)	0.6% (0.2% - 1.6%)	22 (2 - 142)	0% (0% - 0.1%)
Cleveland-Akron-Elyria__OH	1153 (556 - 2349)	2.8% (1.3% - 5.7%)	351 (147 - 875)	0.9% (0.4% - 2.1%)	17 (2 - 82)	0% (0% - 0.2%)
Detroit-Warren-Flint__MI	1902 (892 - 3978)	2.4% (1.1% - 5%)	544 (225 - 1398)	0.7% (0.3% - 1.8%)	24 (3 - 118)	0% (0% - 0.2%)
Houston-Baytown-Huntsville__TX	2069 (1152 - 3428)	2.6% (1.5% - 4.4%)	853 (382 - 1851)	1.1% (0.5% - 2.4%)	85 (16 - 310)	0.1% (0% - 0.4%)
Los_Angeles-Long_Beach-Riverside__CA	15323 (8311 - 27075)	5.8% (3.2% - 10.3%)	6171 (2802 - 13306)	2.3% (1.1% - 5.1%)	670 (126 - 2401)	0.3% (0% - 0.9%)
New_York-Newark-Bridgeport__NY-NJ-CT-PA	8236 (3998 - 16581)	2.8% (1.4% - 5.6%)	2552 (1077 - 6303)	0.9% (0.4% - 2.1%)	135 (18 - 628)	0% (0% - 0.2%)
Philadelphia-Camden-Vineland__PA-NJ-DE-MD	2893 (1456 - 5573)	3.5% (1.7% - 6.7%)	972 (419 - 2307)	1.2% (0.5% - 2.8%)	61 (8 - 269)	0.1% (0% - 0.3%)
Sacramento-Arden-Arcade-Truckee__CA-NV	1212 (623 - 2362)	4% (2.1% - 7.9%)	420 (180 - 990)	1.4% (0.6% - 3.3%)	24 (3 - 111)	0.1% (0% - 0.4%)
St._Louis-St._Charles-Farmington__MO-IL	1065 (529 - 2110)	2.6% (1.3% - 5.2%)	339 (142 - 836)	0.8% (0.4% - 2.1%)	13 (1 - 75)	0% (0% - 0.2%)
Washington-Baltimore-Northern_Virginia__DC-MD-VA-WV	3243 (1627 - 6455)	3.1% (1.5% - 6.1%)	1090 (472 - 2577)	1% (0.4% - 2.4%)	78 (13 - 320)	0.1% (0% - 0.3%)

*Numbers are median (0.5 fractile) numbers of occurrences. Numbers in parentheses below the median are 95% confidence intervals based on statistical uncertainty surrounding the O_3 coefficient. Numbers are rounded to the nearest 1000. Percents are rounded to the nearest tenth.

Table 3-4. Number and Percent of All Children (Ages 5-18) Engaged in Moderate Exercise Estimated to Experience At Least One Lung Function Response Associated with Exposure to "As Is" O_3 Concentrations Over Background O_3 Concentrations: April - September, 2004*

Location	Response = Decrease in FEV_1 Greater Than or Equal to:					
	10%		15%		20%	
	Number (1000s)	Percent	Number (1000s)	Percent	Number (1000s)	Percent
Atlanta-Sandy_Springs-Gainesville__GA-AL	154 (101 - 197)	16.3% (10.7% - 21%)	96 (48 - 164)	10.2% (5.1% - 17.5%)	24 (5 - 68)	2.6% (0.6% - 7.2%)
Boston-Worcester-Manchester__MA-NH	149 (96 - 195)	13.5% (8.7% - 17.7%)	88 (44 - 157)	8% (4% - 14.3%)	16 (3 - 52)	1.5% (0.3% - 4.8%)
Chicago-Naperville-Michigan_City__IL-IN-WI	229 (149 - 296)	11.8% (7.6% - 15.2%)	132 (64 - 243)	6.8% (3.3% - 12.5%)	15 (1 - 64)	0.8% (0.1% - 3.3%)
Cleveland-Akron-Elyria__OH	80 (52 - 105)	13.8% (8.9% - 18%)	48 (24 - 85)	8.2% (4.1% - 14.5%)	9 (1 - 29)	1.5% (0.2% - 5%)
Detroit-Warren-Flint__MI	148 (96 - 192)	13.3% (8.6% - 17.3%)	87 (43 - 156)	7.9% (3.9% - 14.1%)	14 (2 - 50)	1.3% (0.2% - 4.5%)
Houston-Baytown-Huntsville__TX	183 (121 - 231)	17% (11.2% - 21.5%)	119 (61 - 197)	11% (5.7% - 18.3%)	37 (10 - 93)	3.4% (0.9% - 8.7%)
Los_Angeles-Long_Beach-Riverside__CA	686 (449 - 874)	19.1% (12.5% - 24.3%)	456 (239 - 735)	12.7% (6.7% - 20.4%)	164 (47 - 393)	4.6% (1.3% - 10.9%)
New_York-Newark-Bridgeport__NY-NJ-CT-PA	596 (386 - 776)	14.6% (9.4% - 19%)	358 (179 - 631)	8.8% (4.4% - 15.4%)	73 (15 - 229)	1.8% (0.4% - 5.6%)
Philadelphia-Camden-Vineland__PA-NJ-DE-MD	184 (119 - 239)	15.6% (10.1% - 20.3%)	112 (57 - 195)	9.5% (4.8% - 16.6%)	26 (6 - 77)	2.2% (0.5% - 6.5%)
Sacramento-Arden-Arcade-Truckee__CA-NV	57 (37 - 73)	13.6% (8.9% - 17.4%)	37 (20 - 61)	8.9% (4.7% - 14.5%)	9 (2 - 28)	2.3% (0.5% - 6.7%)
St._Louis-St._Charles-Farmington__MO-IL	76 (50 - 98)	13.3% (8.7% - 17.1%)	46 (23 - 81)	8% (3.9% - 14.2%)	7 (1 - 26)	1.3% (0.2% - 4.6%)
Washington-Baltimore-Northern_Virginia__DC-MD-VA-WV	242 (157 - 313)	16.4% (10.7% - 21.2%)	149 (75 - 257)	10.1% (5.1% - 17.4%)	39 (9 - 107)	2.6% (0.6% - 7.3%)

*Numbers are median (0.5 fractile) numbers of children. Numbers in parentheses below the median are 95% confidence intervals based on statistical uncertainty surrounding the O_3 coefficient. Numbers are rounded to the nearest 1000. Percents are rounded to the nearest tenth.

Table 3-5. Estimated Number and Percent of Occurrences of Lung Function Response Associated with Exposure to "As Is" O_3 Concentrations Over Background O_3 Concentrations Among Active Children (Ages 5-18) Engaged in Moderate Exercise: April - September, 2004*

Location	Response = Decrease in FEV_1 Greater Than or Equal to:					
	10%		15%		20%	
	Number (1000s)	Percent	Number (1000s)	Percent	Number (1000s)	Percent
Atlanta-Sandy_Springs-Gainesville__GA-AL	1368 (700 - 2612)	3.5% (1.8% - 6.6%)	479 (209 - 1113)	1.2% (0.5% - 2.8%)	35 (5 - 145)	0.1% (0% - 0.4%)
Boston-Worcester-Manchester__MA-NH	977 (468 - 1992)	2.6% (1.3% - 5.4%)	296 (124 - 737)	0.8% (0.3% - 2%)	15 (2 - 72)	0% (0% - 0.2%)
Chicago-Naperville-Michigan_City__IL-IN-WI	1657 (789 - 3341)	2.5% (1.2% - 5%)	475 (192 - 1240)	0.7% (0.3% - 1.9%)	13 (1 - 82)	0% (0% - 0.1%)
Cleveland-Akron-Elyria__OH	641 (314 - 1269)	3.1% (1.5% - 6.1%)	202 (85 - 496)	1% (0.4% - 2.4%)	10 (1 - 49)	0% (0% - 0.2%)
Detroit-Warren-Flint__MI	1052 (502 - 2129)	2.7% (1.3% - 5.4%)	312 (129 - 790)	0.8% (0.3% - 2%)	14 (2 - 70)	0% (0% - 0.2%)
Houston-Baytown-Huntsville__TX	1301 (736 - 2097)	2.9% (1.6% - 4.6%)	551 (247 - 1185)	1.2% (0.5% - 2.6%)	56 (11 - 202)	0.1% (0% - 0.4%)
Los_Angeles-Long_Beach-Riverside__CA	9536 (5231 - 16504)	6.4% (3.5% - 11.1%)	3935 (1796 - 8387)	2.6% (1.2% - 5.6%)	440 (83 - 1567)	0.3% (0.1% - 1.1%)
New_York-Newark-Bridgeport__NY-NJ-CT-PA	4584 (2259 - 8953)	3.1% (1.5% - 6.1%)	1464 (621 - 3570)	1% (0.4% - 2.4%)	80 (11 - 369)	0.1% (0% - 0.3%)
Philadelphia-Camden-Vineland__PA-NJ-DE-MD	1695 (866 - 3179)	3.8% (2% - 7.2%)	588 (255 - 1376)	1.3% (0.6% - 3.1%)	39 (6 - 169)	0.1% (0% - 0.4%)
Sacramento-Arden-Arcade-Truckee__CA-NV	749 (390 - 1421)	4.4% (2.3% - 8.4%)	267 (115 - 622)	1.6% (0.7% - 3.7%)	15 (2 - 72)	0.1% (0% - 0.4%)
St._Louis-St._Charles-Farmington__MO-IL	655 (331 - 1258)	2.9% (1.5% - 5.6%)	216 (91 - 525)	1% (0.4% - 2.4%)	9 (1 - 49)	0% (0% - 0.2%)
Washington-Baltimore-Northern_Virginia__DC-MD-VA-WV	1907 (971 - 3676)	3.4% (1.8% - 6.6%)	660 (287 - 1541)	1.2% (0.5% - 2.8%)	48 (8 - 198)	0.1% (0% - 0.4%)

*Numbers are median (0.5 fractile) numbers of occurrences. Numbers in parentheses below the median are 95% confidence intervals based on statistical uncertainty surrounding the O_3 coefficient. Numbers are rounded to the nearest 1000. Percents are rounded to the nearest tenth.

Table 3-6. Number and Percent of Active Children (Ages 5-18) Engaged in Moderate Exercise Estimated to Experience At Least One Lung Function Response Associated with Exposure to "As Is" O_3 Concentrations Over Background O_3 Concentrations: April - September, 2004*

Location	Response = Decrease in FEV_1 Greater Than or Equal to:					
	10%		15%		20%	
	Number (1000s)	Percent	Number (1000s)	Percent	Number (1000s)	Percent
Atlanta-Sandy_Springs-Gainesville__GA-AL	90 (59 - 115)	17.3% (11.4% - 22.1%)	57 (29 - 96)	10.9% (5.5% - 18.6%)	15 (4 - 42)	2.9% (0.7% - 8.1%)
Boston-Worcester-Manchester__MA-NH	75 (48 - 97)	14.1% (9.2% - 18.3%)	45 (22 - 79)	8.5% (4.2% - 15%)	9 (2 - 28)	1.7% (0.3% - 5.3%)
Chicago-Naperville-Michigan_City__IL-IN-WI	115 (75 - 148)	12.3% (8% - 15.8%)	67 (33 - 123)	7.2% (3.5% - 13.2%)	8 (1 - 35)	0.9% (0.1% - 3.7%)
Cleveland-Akron-Elyria__OH	43 (28 - 55)	14.5% (9.4% - 18.7%)	26 (13 - 45)	8.7% (4.4% - 15.4%)	5 (1 - 16)	1.7% (0.3% - 5.5%)
Detroit-Warren-Flint__MI	77 (50 - 99)	14% (9.1% - 18%)	46 (23 - 82)	8.4% (4.2% - 14.9%)	8 (1 - 28)	1.5% (0.3% - 5.1%)
Houston-Baytown-Huntsville__TX	108 (71 - 136)	18% (11.9% - 22.7%)	71 (37 - 116)	11.8% (6.1% - 19.4%)	23 (6 - 57)	3.9% (1.1% - 9.6%)
Los_Angeles-Long_Beach-Riverside__CA	391 (257 - 498)	20.1% (13.2% - 25.5%)	264 (139 - 420)	13.5% (7.1% - 21.5%)	100 (29 - 235)	5.1% (1.5% - 12%)
New_York-Newark-Bridgeport__NY-NJ-CT-PA	311 (203 - 400)	15.5% (10.1% - 19.9%)	191 (96 - 332)	9.5% (4.8% - 16.5%)	42 (9 - 127)	2.1% (0.4% - 6.3%)
Philadelphia-Camden-Vineland__PA-NJ-DE-MD	101 (66 - 130)	16.6% (10.8% - 21.4%)	63 (32 - 108)	10.3% (5.2% - 17.7%)	16 (4 - 45)	2.6% (0.6% - 7.4%)
Sacramento-Arden-Arcade-Truckee__CA-NV	31 (21 - 40)	13.9% (9.1% - 17.8%)	21 (11 - 34)	9.4% (4.9% - 14.9%)	6 (1 - 17)	2.5% (0.5% - 7.3%)
St._Louis-St._Charles-Farmington__MO-IL	43 (28 - 56)	14.1% (9.2% - 18%)	26 (13 - 47)	8.5% (4.3% - 15.1%)	5 (1 - 16)	1.5% (0.2% - 5.2%)
Washington-Baltimore-Northern_Virginia__DC-MD-VA-WV	132 (86 - 169)	17.3% (11.3% - 22.2%)	82 (42 - 141)	10.8% (5.5% - 18.5%)	22 (5 - 61)	3% (0.7% - 8%)

*Numbers are median (0.5 fractile) numbers of children. Numbers in parentheses below the median are 95% confidence intervals based on statistical uncertainty surrounding the O_3 coefficient. Numbers are rounded to the nearest 1000. Percents are rounded to the nearest tenth.

The estimated occurrence of lung function decrement among all school age children exercising moderately while exposed to "as is" O_3 concentrations (Table 3-3) varied across the locations for each of the three lung function response measures. For all three lung function response measures (decrements in $FEV_1 \geq 10\%$, $\geq 15\%$, and $\geq 20\%$), Los Angeles had the greatest percentage of child-days with occurrences of lung function response, and Chicago had the smallest percentage. Decrements of $FEV_1 \geq 10\%$, for example, were estimated to occur on 2.2% of child-days in Chicago versus 5.8% of child-days in Los Angeles. Not surprisingly, absolute numbers of occurrences of lung function decrement were also largest in Los Angeles. They were smallest in St. Louis. For example, there were fewer than 13,500 child-days in St. Louis versus almost 670,000 child-days in Los Angeles with decrements in $FEV_1 \geq 20\%$.

The patterns were similar for occurrences of lung function decrement among active school age children (Table 3-5). Once again, the greatest percentages of child-days with occurrences, for each of the three lung function response measures, were in Los Angeles. For example, the percentage of child-days (for active children) on which decrements of $FEV_1 \geq 15\%$ were estimated to occur ranged from 0.7% in Chicago to 2.6% in Los Angeles. The absolute numbers of occurrences were also largest in Los Angeles for all three lung function response measures (ranging from almost 440,000 occurrences of decrements in $FEV_1 \geq 20\%$ to over 9.5 million occurrences of decrements in $FEV_1 \geq 10\%$). They were smallest in Cleveland for decrements in $FEV_1 \geq 10\%$ and $\geq 15\%$, and smallest in St. Louis for decrements in $FEV_1 \geq 20\%$.

When we considered the number of children experiencing at least one lung function response during the period from April through September (Tables 3-4 and 3-6), the patterns were similar to those observed when occurrence of lung function responses was estimated. Among all school age children and among active school age children, the percentage experiencing at least one lung function response was largest in Los Angeles and smallest in Chicago – for each of the three lung function response measures. For example, 19% of all school age children and 20% of active school age children in Los Angeles experienced at least one decrement in $FEV_1 \geq 10\%$ during the ozone season. The corresponding percentages for Chicago were 11.8% and 12.3%, respectively.

3.2.2 Assessment of lung function decrement associated with exposure to O_3 concentrations that just meet the current daily maximum 8-hour standard

The estimated number and percent of occurrences of lung function response associated with exposure to O_3 concentrations that just meet the current daily maximum 8-hour standard among all school age children (ages 5 – 18) engaged in moderate exercise for at least one 8-hour period from April through September, is given in Table 3-7. The number and percent of these children estimated to experience at least one lung function response associated with exposure to O_3 concentrations that just meet the current standard is given in Table 3-8. Tables 3-9 and 3-10 give the corresponding results for active school age children. Results for all three measures of lung function response being considered in this analysis – decrements in FEV_1 of $\geq 10\%$, $\geq 15\%$, and $\geq 20\%$ -- are shown in each table.

Table 3-7. Estimated Number and Percent of Occurrences of Lung Function Response Associated with Exposure to O_3 Concentrations That Just Meet the Current Daily Maximum 8-Hour Standard Among All Children (Ages 5-18) Engaged in Moderate Exercise: April - September*

Location	Response = Decrease in FEV_1 Greater Than or Equal to:					
	10%		15%		20%	
	Number (1000s)	Percent	Number (1000s)	Percent	Number (1000s)	Percent
Atlanta-Sandy_Springs-Gainesville__GA-AL	1723 (836 - 3558)	2.5% (1.2% - 5.1%)	528 (220 - 1318)	0.8% (0.3% - 1.9%)	24 (3 - 121)	0% (0% - 0.2%)
Boston-Worcester-Manchester__MA-NH	1455 (656 - 3201)	1.9% (0.8% - 4.1%)	378 (152 - 1022)	0.5% (0.2% - 1.3%)	11 (1 - 66)	0% (0% - 0.1%)
Chicago-Naperville-Michigan_City__IL-IN-WI	2216 (964 - 4915)	1.6% (0.7% - 3.6%)	509 (191 - 1493)	0.4% (0.1% - 1.1%)	5 (0 - 50)	0% (0% - 0%)
Cleveland-Akron-Elyria__OH	861 (389 - 1876)	2.1% (0.9% - 4.5%)	222 (88 - 606)	0.5% (0.2% - 1.5%)	5 (0 - 34)	0% (0% - 0.1%)
Detroit-Warren-Flint__MI	1487 (660 - 3274)	1.9% (0.8% - 4.2%)	371 (146 - 1027)	0.5% (0.2% - 1.3%)	9 (1 - 57)	0% (0% - 0.1%)
Houston-Baytown-Huntsville__TX	1424 (770 - 2421)	1.8% (1% - 3.1%)	526 (223 - 1233)	0.7% (0.3% - 1.6%)	27 (3 - 130)	0% (0% - 0.2%)
Los_Angeles-Long_Beach-Riverside__CA	7258 (3396 - 15342)	2.8% (1.3% - 5.8%)	1974 (778 - 5325)	0.8% (0.3% - 2%)	33 (1 - 271)	0% (0% - 0.1%)
New_York-Newark-Bridgeport__NY-NJ-CT-PA	5388 (2414 - 11709)	1.8% (0.8% - 4%)	1362 (537 - 3760)	0.5% (0.2% - 1.3%)	33 (3 - 209)	0% (0% - 0.1%)
Philadelphia-Camden-Vineland__PA-NJ-DE-MD	2096 (991 - 4307)	2.5% (1.2% - 5.2%)	599 (244 - 1556)	0.7% (0.3% - 1.9%)	20 (2 - 114)	0% (0% - 0.1%)
Sacramento-Arden-Arcade-Truckee__CA-NV	823 (391 - 1771)	2.7% (1.3% - 5.9%)	233 (93 - 615)	0.8% (0.3% - 2.1%)	5 (0 - 36)	0% (0% - 0.1%)
St._Louis-St._Charles-Farmington__MO-IL	903 (436 - 1845)	2.2% (1.1% - 4.6%)	267 (108 - 687)	0.7% (0.3% - 1.7%)	7 (0 - 47)	0% (0% - 0.1%)
Washington-Baltimore-Northern_Virginia__DC-MD-VA-WV	2265 (1080 - 4761)	2.1% (1% - 4.5%)	671 (279 - 1698)	0.6% (0.3% - 1.6%)	32 (5 - 151)	0% (0% - 0.1%)

*Numbers are median (0.5 fractile) numbers of occurrences. Numbers in parentheses below the median are 95% confidence intervals based on statistical uncertainty surrounding the O_3 coefficient. Numbers are rounded to the nearest 1000. Percents are rounded to the nearest tenth.

Table 3-8. Number and Percent of All Children (Ages 5-18) Engaged in Moderate Exercise Estimated to Experience At Least One Lung Function Response Associated with Exposure to O_3 Concentrations That Just Meet the Current Daily Maximum 8-Hour Standard: April - September*

Location	Response = Decrease in FEV_1 Greater Than or Equal to:					
	10%		15%		20%	
	Number (1000s)	Percent	Number (1000s)	Percent	Number (1000s)	Percent
Atlanta-Sandy_Springs-Gainesville__GA-AL	130 (85 - 167)	13.8% (9% - 17.7%)	78 (39 - 138)	8.3% (4.1% - 14.7%)	14 (2 - 47)	1.4% (0.2% - 5%)
Boston-Worcester-Manchester__MA-NH	124 (80 - 164)	11.3% (7.3% - 14.9%)	70 (34 - 131)	6.4% (3.1% - 11.9%)	8 (1 - 32)	0.7% (0.1% - 3%)
Chicago-Naperville-Michigan_City__IL-IN-WI	179 (116 - 233)	9.2% (6% - 12%)	97 (45 - 190)	5% (2.3% - 9.8%)	4 (0 - 28)	0.2% (0% - 1.4%)
Cleveland-Akron-Elyria__OH	64 (41 - 84)	11% (7.1% - 14.5%)	36 (17 - 67)	6.1% (2.9% - 11.5%)	3 (0 - 15)	0.6% (0% - 2.7%)
Detroit-Warren-Flint__MI	122 (79 - 159)	11% (7.1% - 14.3%)	68 (33 - 128)	6.2% (3% - 11.6%)	7 (1 - 30)	0.6% (0.1% - 2.7%)
Houston-Baytown-Huntsville__TX	136 (90 - 172)	12.7% (8.3% - 16%)	84 (42 - 147)	7.8% (3.9% - 13.7%)	15 (3 - 52)	1.4% (0.2% - 4.8%)
Los_Angeles-Long_Beach-Riverside__CA	349 (227 - 450)	9.7% (6.3% - 12.5%)	208 (103 - 371)	5.8% (2.9% - 10.3%)	19 (1 - 93)	0.5% (0% - 2.6%)
New_York-Newark-Bridgeport__NY-NJ-CT-PA	447 (288 - 588)	10.9% (7% - 14.4%)	250 (120 - 470)	6.1% (2.9% - 11.5%)	25 (3 - 108)	0.6% (0.1% - 2.7%)
Philadelphia-Camden-Vineland__PA-NJ-DE-MD	145 (94 - 190)	12.3% (8% - 16.1%)	84 (41 - 153)	7.1% (3.5% - 13%)	11 (1 - 44)	1% (0.1% - 3.7%)
Sacramento-Arden-Arcade-Truckee__CA-NV	40 (26 - 51)	9.5% (6.2% - 12.2%)	25 (13 - 42)	5.9% (3% - 10.1%)	3 (0 - 13)	0.7% (0% - 3.2%)
St._Louis-St._Charles-Farmington__MO-IL	68 (44 - 87)	11.8% (7.7% - 15.2%)	39 (19 - 72)	6.9% (3.4% - 12.6%)	4 (0 - 19)	0.8% (0.1% - 3.4%)
Washington-Baltimore-Northern_Virginia__DC-MD-VA-WV	198 (128 - 257)	13.4% (8.7% - 17.5%)	117 (58 - 209)	7.9% (3.9% - 14.2%)	21 (4 - 69)	1.4% (0.3% - 4.7%)

*Numbers are median (0.5 fractile) numbers of children. Numbers in parentheses below the median are 95% confidence intervals based on statistical uncertainty surrounding the O_3 coefficient. Numbers are rounded to the nearest 1000. Percents are rounded to the nearest tenth.

Table 3-9. Estimated Number and Percent of Occurrences of Lung Function Response Associated with Exposure to O_3 Concentrations That Just Meet the Current Daily Maximum 8-Hour Standard Among Active Children (Ages 5-18) Engaged in Moderate Exercise: April - September*

Location	Response = Decrease in FEV_1 Greater Than or Equal to:					
	10%		15%		20%	
	Number (1000s)	Percent	Number (1000s)	Percent	Number (1000s)	Percent
Atlanta-Sandy_Springs-Gainesville__GA-AL	1093 (538 - 2187)	2.8% (1.4% - 5.6%)	345 (145 - 851)	0.9% (0.4% - 2.2%)	16 (2 - 82)	0% (0% - 0.2%)
Boston-Worcester-Manchester__MA-NH	779 (358 - 1656)	2.1% (1% - 4.5%)	211 (85 - 560)	0.6% (0.2% - 1.5%)	7 (1 - 38)	0% (0% - 0.1%)
Chicago-Naperville-Michigan_City__IL-IN-WI	1201 (534 - 2576)	1.8% (0.8% - 3.9%)	288 (109 - 830)	0.4% (0.2% - 1.2%)	3 (0 - 30)	0% (0% - 0%)
Cleveland-Akron-Elyria__OH	482 (222 - 1019)	2.3% (1.1% - 4.9%)	129 (51 - 347)	0.6% (0.2% - 1.7%)	3 (0 - 20)	0% (0% - 0.1%)
Detroit-Warren-Flint__MI	825 (374 - 1755)	2.1% (1% - 4.5%)	214 (85 - 584)	0.5% (0.2% - 1.5%)	6 (0 - 34)	0% (0% - 0.1%)
Houston-Baytown-Huntsville__TX	897 (495 - 1476)	2% (1.1% - 3.3%)	342 (146 - 794)	0.8% (0.3% - 1.8%)	18 (2 - 86)	0% (0% - 0.2%)
Los_Angeles-Long_Beach-Riverside__CA	4593 (2188 - 9444)	3.1% (1.5% - 6.3%)	1293 (513 - 3439)	0.9% (0.3% - 2.3%)	22 (1 - 182)	0% (0% - 0.1%)
New_York-Newark-Bridgeport__NY-NJ-CT-PA	3016 (1375 - 6338)	2.1% (0.9% - 4.3%)	789 (313 - 2146)	0.5% (0.2% - 1.5%)	20 (2 - 124)	0% (0% - 0.1%)
Philadelphia-Camden-Vineland__PA-NJ-DE-MD	1233 (593 - 2457)	2.8% (1.3% - 5.6%)	366 (150 - 934)	0.8% (0.3% - 2.1%)	13 (1 - 73)	0% (0% - 0.2%)
Sacramento-Arden-Arcade-Truckee__CA-NV	514 (249 - 1073)	3% (1.5% - 6.4%)	151 (61 - 392)	0.9% (0.4% - 2.3%)	3 (0 - 24)	0% (0% - 0.1%)
St._Louis-St._Charles-Farmington__MO-IL	559 (274 - 1105)	2.5% (1.2% - 4.9%)	171 (70 - 433)	0.8% (0.3% - 1.9%)	5 (0 - 32)	0% (0% - 0.1%)
Washington-Baltimore-Northern_Virginia__DC-MD-VA-WV	1341 (650 - 2721)	2.4% (1.2% - 4.9%)	410 (171 - 1025)	0.7% (0.3% - 1.9%)	20 (3 - 95)	0% (0% - 0.2%)

*Numbers are median (0.5 fractile) numbers of occurrences. Numbers in parentheses below the median are 95% confidence intervals based on statistical uncertainty surrounding the O_3 coefficient. Numbers are rounded to the nearest 1000. Percents are rounded to the nearest tenth.

Table 3-10. Number and Percent of Active Children (Ages 5-18) Engaged in Moderate Exercise Estimated to Experience At Least One Lung Function Response Associated with Exposure to O_3 Concentrations That Just Meet the Current Daily Maximum 8-Hour Standard: April - September*

Location	Response = Decrease in FEV_1 Greater Than or Equal to:					
	10%		15%		20%	
	Number (1000s)	Percent	Number (1000s)	Percent	Number (1000s)	Percent
Atlanta-Sandy_Springs-Gainesville__GA-AL	76 (50 - 97)	14.7% (9.6% - 18.8%)	47 (23 - 82)	9% (4.5% - 15.8%)	9 (2 - 29)	1.7% (0.3% - 5.7%)
Boston-Worcester-Manchester__MA-NH	63 (41 - 82)	11.9% (7.7% - 15.5%)	36 (18 - 67)	6.9% (3.3% - 12.6%)	4 (1 - 18)	0.8% (0.1% - 3.4%)
Chicago-Naperville-Michigan_City__IL-IN-WI	90 (59 - 117)	9.7% (6.3% - 12.5%)	50 (23 - 96)	5.3% (2.5% - 10.3%)	2 (0 - 16)	0.3% (0% - 1.7%)
Cleveland-Akron-Elyria__OH	34 (22 - 44)	11.5% (7.5% - 15%)	19 (9 - 36)	6.6% (3.2% - 12.2%)	2 (0 - 9)	0.6% (0% - 3%)
Detroit-Warren-Flint__MI	64 (42 - 83)	11.6% (7.5% - 15%)	37 (18 - 68)	6.7% (3.2% - 12.3%)	4 (0 - 17)	0.7% (0.1% - 3.1%)
Houston-Baytown-Huntsville__TX	80 (53 - 101)	13.4% (8.8% - 16.9%)	50 (26 - 87)	8.4% (4.3% - 14.5%)	10 (2 - 32)	1.7% (0.3% - 5.4%)
Los_Angeles-Long_Beach-Riverside__CA	200 (130 - 257)	10.2% (6.7% - 13.2%)	123 (62 - 213)	6.3% (3.2% - 10.9%)	12 (1 - 60)	0.6% (0% - 3.1%)
New_York-Newark-Bridgeport__NY-NJ-CT-PA	233 (152 - 301)	11.6% (7.5% - 15%)	134 (65 - 248)	6.7% (3.2% - 12.3%)	15 (2 - 61)	0.7% (0.1% - 3.1%)
Philadelphia-Camden-Vineland__PA-NJ-DE-MD	80 (52 - 103)	13.2% (8.6% - 17%)	47 (23 - 85)	7.8% (3.8% - 14%)	7 (1 - 26)	1.1% (0.1% - 4.3%)
Sacramento-Arden-Arcade-Truckee__CA-NV	22 (14 - 28)	9.7% (6.4% - 12.5%)	14 (7 - 23)	6.3% (3.3% - 10.4%)	2 (0 - 8)	0.8% (0.1% - 3.6%)
St._Louis-St._Charles-Farmington__MO-IL	39 (25 - 50)	12.6% (8.2% - 16.1%)	23 (11 - 41)	7.4% (3.6% - 13.4%)	3 (0 - 12)	0.9% (0.1% - 3.9%)
Washington-Baltimore-Northern_Virginia__DC-MD-VA-WV	109 (71 - 140)	14.4% (9.3% - 18.5%)	66 (33 - 116)	8.7% (4.3% - 15.3%)	13 (3 - 41)	1.7% (0.3% - 5.4%)

*Numbers are median (0.5 fractile) numbers of children. Numbers in parentheses below the median are 95% confidence intervals based on statistical uncertainty surrounding the O_3 coefficient. Numbers are rounded to the nearest 1000. Percents are rounded to the nearest tenth.

When O_3 concentrations just meet the current daily maximum 8-hour standard, the decrease in occurrence of lung function response, relative to risk estimated under "as is" O_3 concentrations, is estimated to be greater the larger the decrement being measured. Among all school age children, the numbers of occurrences of decrements in $FEV_1 \geq 10\%$ were estimated to be, on average, 28 percent of what they were under "as is" O_3 concentrations (compare Tables 3-7 and 3-3). The corresponding average percent decreases in occurrence for decrements in $FEV_1 \geq 15\%$ and $\geq 20\%$ were 39 percent and 68 percent, respectively. A similar pattern was evident among active school age children (compare Tables 3-9 and 3-5). Average estimated percent decreases in occurrence of decrements in $FEV_1 \geq 10\%$, $\geq 15\%$, and $\geq 20\%$ (from what they were estimated to be under "as is" O_3 concentrations) among active school age children were 28 percent, 38 percent, and 68 percent, respectively.

The extent of reduction in the population estimated to experience lung function response going from "as is" O_3 concentrations to levels just meeting the current 8-hour standard varied from one location to another. While Los Angeles had the greatest percent of child-days with lung function responses under "as is" O_3 concentrations, for all three lung function response measures, it also was estimated to have the greatest *decrease* in occurrence of lung function response when O_3 concentrations just meet the current standard – with estimated percent decreases in occurrence of decrements in $FEV_1 \geq 10\%$, $\geq 15\%$, and $\geq 20\%$ among all school age children of 53 percent, 68 percent, and 95 percent, respectively. The same patterns were evident for active school age children.

When we considered the number of children experiencing at least one lung function response during the months of April through September (Tables 3-8 and 3-10), the patterns were similar to those observed when occurrence of lung function responses was estimated. Among all school age children and among active school age children, the percent experiencing at least one lung function response decreased (from the percentages under "as is" O_3 concentrations) more as the severity of the lung function decrement increased. Among all school age children, the number with at least one decrement in $FEV_1 \geq 10\%$ was estimated to be, on average, 23 percent of what it was under "as is" O_3 concentrations (compare Tables 3-8 and 3-4). Among active school age children, the corresponding percent decrease was 22 percent. The corresponding percent decreases in numbers of children, when we consider decrements in $FEV_1 \geq 15\%$ and $\geq 20\%$, are 27 percent and 59 percent, respectively, for all school age children, and 26 percent and 58 percent, respectively, for active school age children.

4 ASSESSMENT OF RISK BASED ON EPIDEMIOLOGICAL STUDIES

As discussed in the draft O_3 AQCD, a significant number of epidemiological 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 studies and air quality information, EPA staff decided to expand the O_3 risk assessment to include an assessment of selected health risks attributable to ambient O_3 concentrations over PRB concentrations and the reduced health risks associated with just meeting the current O_3 standard in selected urban locations in the U.S. The methods and results of this portion of the risk assessment are discussed below.

4.1 Methods

4.1.1 General approach

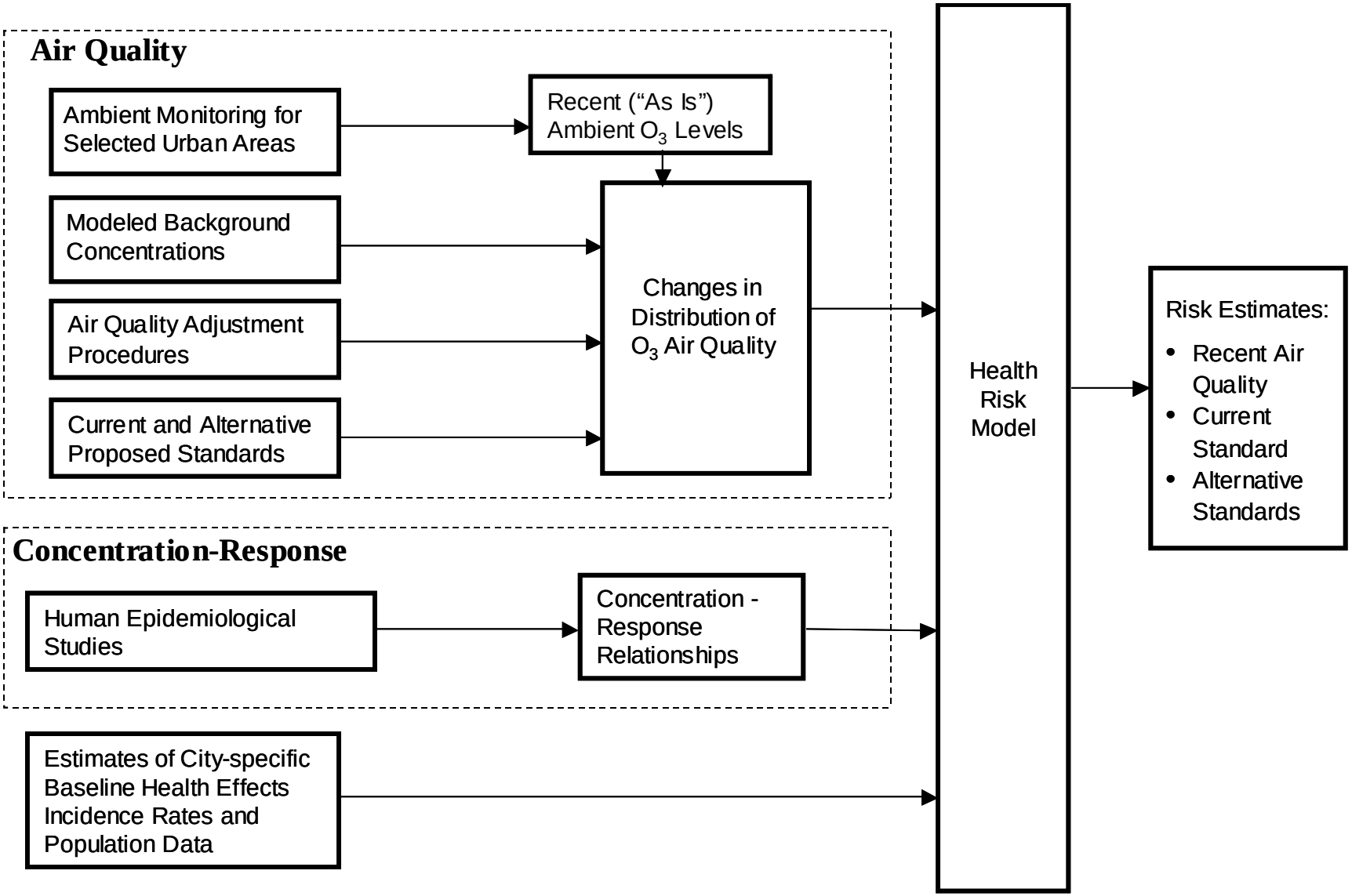
As in the recently completed particulate matter (PM) risk assessment (see EPA, 2005e, Chapter 4, and Abt Associates 2005), the general approach used in this part of the O_3 risk assessment relies upon C-R functions which have been estimated in epidemiological studies. Since these studies estimate C-R functions using ambient air quality data from fixed-site, population-oriented monitors, the appropriate application of these functions in a risk assessment similarly requires the use of ambient air quality data at fixed-site, ambient monitors. The general O_3 health risk model combines information about O_3 air quality for specific urban areas with C-R functions derived from epidemiological studies and baseline health incidence data for specific health endpoints and population estimates to derive estimates of the incidence of specified health effects attributable to ambient O_3 concentrations during the period examined. Although the O_3 season varies somewhat from one location to another, in most locations it coincides roughly with spring and summer. To allow comparisons across locations, all analyses were carried out for the same time period, April through September. The analyses are conducted for both "as is" air quality (using 2004 data) and for air quality simulated to reflect just meeting the current O_3 ambient standard. At a later time, analyses for air quality simulated to reflect just meeting alternative O_3 ambient standards will be added. The major components of the portion of the health risk assessment based on data from epidemiological studies are illustrated in Figure 4-1.

In the first part of the epidemiology-based portion of the risk assessment, we estimated health effects incidence associated with "as is" O_3 levels. In the second part, we estimated the reduced health effects incidence associated with those O_3 concentrations that would result if the current O_3 standard was just met in the assessment locations. In both parts, we considered only the incidence of health effects associated with O_3 concentrations in excess of estimated PRB O_3 levels.

Both parts of the epidemiology-based portion of the risk assessment may be viewed as assessing the change in incidence of the health effect associated with a change in O_3 concentrations from some upper levels to specified (lower) levels. The important operational difference between the two parts is in the upper O_3 levels. In the first part, the upper O_3 levels

are "as is" concentrations. In contrast, the upper O_3 levels in the second part are the estimated O_3 levels that would occur when the current 8-hour daily maximum O_3 standard are just met in the assessment locations. The second part therefore requires that a method be developed to simulate just meeting the current standard. This method is described in Chapter 4 of the draft Staff Paper and in Rizzo (2005).

Figure 4-1. Major Components of Ozone Health Risk Assessment Based on Epidemiology Studies



To estimate the change in incidence of a given health effect resulting from a change in ambient O_3 concentrations from "as is" levels to PRB levels, or from O_3 concentrations that just meet the current standard to PRB levels, in an assessment location, the following analysis inputs are necessary:

- **Air quality information** including: (1) "as is" air quality data for O_3 from ambient monitors in the assessment location, (2) "as is" concentrations adjusted to reflect patterns of air quality estimated to occur when the area just meets the specified standard, and (3) estimates of PRB O_3 concentrations appropriate to this location. (These air quality inputs are discussed in more detail in Chapters 2 and 4 of the draft Staff Paper.

- **Concentration-response function(s)** which provide an estimate of the relationship between the health endpoint of interest and O_3 concentrations (preferably derived in the assessment location, although functions estimated in other locations can be used at the cost of increased uncertainty -- see Section 4.1.9.1.3).

- **Baseline health effects incidence rate and population**. The baseline incidence rate provides an estimate of the incidence rate (number of cases of the health effect per O_3 season, usually per 10,000 or 100,000 population) in the assessment location corresponding to "as is" O_3 levels in that location. To derive the total baseline incidence per O_3 season, the baseline incidence rate must be multiplied by the corresponding population number (e.g., if the baseline incidence rate is number of cases per O_3 season per 100,000 population, it must be multiplied by the number of 100,000s in the population). (Section 4.1.8 summarizes considerations related to the baseline incidence rate and population data inputs to the risk assessment).

These inputs are combined to estimate health effect incidence changes associated with specified changes in O_3 levels. Although some epidemiological studies have estimated linear or logistic C-R functions, by far the most common form is the exponential (or log-linear) form:

$$y = Be^{\beta x}, \tag{4-1}$$

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 of ambient O_3 concentration, 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

$$y_0 = Be^{\beta x_0}. \tag{4-2}$$

Because the log-linear form of C-R function (equation (4-1)) is by far the most common form, we use this form to illustrate the "health impact function" used in this portion of the risk assessment.[3]

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 a relationship between the change in x, Δx, and the corresponding change in y, Δy, from equation (4-1). If $\Delta x > 0$ – i.e., if $\Delta x = (x_0 - x_1)$ – then the relationship between Δx and Δy can be shown to be

$$\Delta y = (y_0 - y_1) = y_0[1 - e^{-\beta \Delta x}]. \quad (4\text{-}3)$$

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:

$$\Delta y = (y_1 - y_0) = y_0[e^{\beta \Delta x} - 1] \ . \quad (4\text{-}4)$$

Alternatively, the difference in health effects incidence can be calculated indirectly using relative risk. Relative risk (RR) is a measure commonly used by epidemiologists to characterize the comparative health effects associated with a particular air quality comparison. The risk of mortality at ambient 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 (4-1) 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

$$\Delta y = (y_0 - y_1) = y_0[1 - (1 / RR_{\Delta x})] \quad (4\text{-}5)$$

if $\Delta x > 0$, and

$$\Delta y = (y_1 - y_0) = y_0[(1 / RR_{\Delta x}) - 1] \quad (4\text{-}6)$$

if $\Delta x < 0$.

Equations (4-3) and (4-5) 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. The same is true for equations (4-4) and (4-6), when $\Delta x < 0$. 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.

[3] The derivations of health impact functions from concentration-response functions for all three functional forms found in the epidemiological literature – the log-linear, the linear and the logistic – are given in section B.2 of Appendix B.

Note that if we use equations (4-3) or (4-5), in which $\Delta x > 0$, Δy will similarly be positive; and if we use equations (4-4) or (4-6), in which $\Delta x < 0$, Δy will similarly be negative. However, the *magnitude* of Δy will be the same – i.e., the absolute value of Δy does not depend on which equation is used. If Δx and Δy are defined to be negative, we interpret Δy as the number of cases of the health effect that would be avoided by reducing O_3 levels to lower levels; if Δx and Δy are defined to be positive, we interpret Δy as the number of cases of the health effect that would occur that are associated with O_3 levels at the higher level above the lower level. The number of cases is the same, however, regardless of the interpretation used.

4.1.2 Air quality considerations

Air quality considerations are discussed in detail in Chapters 2 and 4 of the draft Staff Paper. 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.

In the first part of the epidemiology-based portion of the risk assessment, we estimated the change in health effect incidence, Δy, associated with a change in O_3 concentrations from current levels of O_3 ("as is" levels) to PRB levels. In the second part, we estimated the change in health effect incidence associated with a change in O_3 concentrations from the levels simulated to just meet the current 8-hour daily maximum standards to PRB levels.[4]

To estimate the change in incidence of a health effect associated with a change in O_3 concentrations from "as is" levels to PRB levels in an assessment location, we need two time series of O_3 concentrations for that location: (1) hourly "as is" O_3 concentrations, and (2) hourly PRB O_3 concentrations. In order to be consistent with the approach generally used in the epidemiological studies that estimated O_3 C-R functions, the (spatial) average ambient O_3 concentration on each hour for which measured data are available is deemed most appropriate for the risk assessment. Consistent with the approach used in the recently completed PM risk assessment (see EPA, 2005e, Chapter 4, and Abt Associates 2005), a composite monitor data set was created for each assessment location. The concentration at the composite monitor in a given hour on a given day is simply the average of the monitor-specific concentrations for that hour on that day.

Two different exposure metrics, the 24-hour average and the daily 1-hour maximum, have been used in epidemiological O_3 studies. We therefore calculated daily changes at the composite monitor in the O_3 exposure metric appropriate to a given C-R function. For example, if a C-R function related daily mortality to daily 1-hour maximum O_3 concentrations, we

[4] In both parts of the risk assessment, both $\Delta x = (x_0 - x)$ and $\Delta y = (y_0 - y)$, as defined in equation (3) above, are negative (or zero). We could have alternatively defined Δx to be positive (i.e., the change from a higher O_3 level to a lower one), in which case Δy would also have been positive, and the relationship between Δx and Δy would be slightly different from the relationship shown in equation (3). The results, however, would be the same. If Δx and Δy are defined to be negative, we interpret Δy as the number of cases of the health effect that would be avoided by reducing O_3 levels to lower levels; if Δx and Δy are defined to be positive, we interpret Δy as the number of cases of the health effect that exist that are associated with O_3 levels at the higher level above the lower level. The number of cases is the same, however, in both cases.

calculated the daily changes in 1-hour maximum O_3 concentrations at the composite monitor. In the first part of the epidemiology-based risk assessment, in which we estimated risks associated with the recent levels of O_3 ("as is" levels) above PRB levels, this required the following steps:

- Using the monitor-specific input streams of hourly "as is" O_3 concentrations, calculate a stream of hourly "as is" O_3 concentrations at the composite monitor. The "as is" O_3 concentration at the composite monitor for a given hour on a given day is the average of the monitor-specific "as is" O_3 concentrations for that hour on that day.
- Using the stream of "as is" hourly O_3 concentrations at the composite monitor, just created, calculate the 1-hour maximum "as is" O_3 concentration for each day at the composite monitor.
- Using the monitor-specific input streams of hourly PRB O_3 concentrations, calculate a stream of hourly PRB O_3 concentrations at the composite monitor.
- Using the stream of PRB hourly O_3 concentrations at the composite monitor, just created, calculate the 1-hour maximum PRB O_3 concentration for each day at the composite monitor.
- For each day, calculate Δx = (the 1-hour maximum "as is" O_3 concentration for that day at the composite monitor) - (the 1-hour maximum PRB O_3 concentration for that day at the composite monitor).[5]

The calculations for the second part of the epidemiology-based risk assessment, in which we estimated risks associated with estimated O_3 levels that just meet the current standard above PRB levels were done analogously, using the monitor-specific series of adjusted hourly concentrations rather than the monitor-specific series of "as is" hourly concentrations. Similarly, calculations for C-R functions that used a different exposure metric (e.g., the 24-hour average) were done analogously, using the exposure metric appropriate to the C-R function.

4.1.3 Selection of health endpoints

EPA staff has carefully reviewed the epidemiological evidence evaluated in Chapter 7 and in Chapter 7 Annex of the draft O_3 AQCD. Tables AX7-1 through AX7-5 summarize the available U.S. and Canadian studies of the effects of acute (short-term) exposures for various health effect categories. Given the substantial number of health endpoints and studies addressing O_3 effects, we included in this quantitative O_3 risk assessment only the better- understood (in terms of health consequences) health endpoint categories for which the weight of the evidence supports the inference of a likely causal relationship between O_3 and the effect category. In addition, we included only those categories for which there are studies that satisfy the study selection criteria discussed below.

Based on its review of the evidence evaluated in the draft O_3 AQCD, EPA staff included in the portion of the O_3 risk assessment based on epidemiology studies the following broad categories of health endpoints associated with short-term exposures:

[5] Note that the maximum-concentration hour for a given day in the "as is" series is not necessarily the same hour as the maximum-concentration hour for that day in the PRB series.

- hospital admissions for respiratory and cardiovascular illnesses; and
- premature total, respiratory, and cardiovascular mortality.

4.1.4 Selection of urban areas

Several objectives were considered in selecting potential urban areas for which to conduct the epidemiology-based O_3 risk assessment. An urban area was considered for inclusion only if it satisfied the following criteria:

- It has sufficient air quality data for the 3-year period (2002-2004).
- It is the same as or close to the location where at least one C-R function for one of the recommended health endpoints (see above) has been estimated by a study that satisfies the study selection criteria (see below).
- For the hospital admission categories, relatively recent location-specific baseline incidence data, specific to International Classification of Disease (ICD) codes, or an equivalent illness classification system, are available.[6]

Because baseline mortality incidence data are available at the county level, this is not a constraint in the selection of urban areas for the O_3 risk assessment. Data on hospital admissions for recent years, however, specific to ICD codes, are available in some cities but not others. The availability of this type of incidence data was therefore a consideration in the selection of urban areas to include in the analysis.

In addition, we took into account the following considerations in selecting from among those urban locations that satisfied the above selection criteria:

- Locations with more health endpoints were preferred to those with fewer.
- The overall set of urban locations should represent a range of geographic areas and population demographics among those areas not meeting the current O_3 8-hour daily maximum standard within the U.S.

Based on the selection criteria and additional considerations listed above, we included the following urban areas in our assessment of risk based on epidemiological studies:

- Atlanta
- Boston
- Chicago
- Cleveland
- Detroit
- Houston
- Los Angeles
- New York City

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

- Philadelphia
- Sacramento
- St. Louis
- Washington, D.C.

4.1.5 Selection of epidemiological studies

As discussed above, we included in the O_3 risk assessment only the better understood health effects for which the weight of the evidence supports a likely causal inference. Thus, in cases where none of the available studies reported a statistically significant relationship, the effect endpoint was not included. Once it had been determined that a health endpoint would be included in the analysis, however, inclusion of a study on that health endpoint was not based on statistical significance. That is, consistent with the approach taken in the particulate matter (PM) risk assessment (see EPA, 2005e, Chapter 4, and Abt Associates, 2005), no credible study on an included health endpoint was excluded from the analysis on the basis of lack of statistical significance.

We applied the following selection criteria for any study that estimated one or more O_3 C-R functions for a selected health endpoint in an urban location to be used for the O_3 risk assessment:

- It is a published, peer-reviewed study that has been evaluated in the draft O_3 AQCD and judged adequate by EPA staff for purposes of inclusion in this risk assessment based on that evaluation.

- It directly measured, rather than estimated, O_3 on a reasonable proportion of the days in the study.

- It either did not rely on Generalized Additive Models (GAMs) using the S-Plus software to estimate C-R functions or has appropriately re-estimated these functions using revised methods.[7]

- For studies of mortality associated with short-term exposure to O_3, the study reported results for the O_3 season.

We note that the draft O_3 AQCD is currently under review by the CASAC O_3 Panel and the general public. Accordingly, the final group of studies to be included in the risk assessment may change based on the advice and recommendations resulting from this review.

[7] The GAM S-Plus problem was discovered prior to the recent PM risk assessment that was carried out as part of the PM NAAQS review. It is discussed in the PM Criteria Document (EPA, 2004), PM Staff Paper (EPA, 2005e), and PM Health Risk Assessment Technical Support Document (Abt Associates, 2005).

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

Based on applying the criteria and considerations discussed above, the health endpoints, urban locations, and epidemiology studies that were included in the O_3 risk assessment are given in Table 4-1. As noted above, additional health endpoint categories, and therefore additional studies, may be included at a later time.

Table 4-1. Locations and Health Endpoints Included in the O_3 Risk Assessment Based on Epidemiological Studies*

Urban Area	Premature Mortality	Hospital Admissions for Respiratory and Cardiovascular Illnesses
Atlanta	Bell et al. (2004) Bell et al. (2004) – 95 cities Huang et al. (2004)** Huang et al. (2004) – 19 cities**	
Boston	Bell et al. (2004) – 95 cities	
Chicago	Bell et al. (2004) – 95 cities Huang et al. (2004) Huang et al. (2004) – 19 cities Schwartz (2004) Schwartz (2004) – 14 cities	
Cleveland	Bell et al. (2004) Bell et al. (2004) – 95 cities Huang et al. (2004) Huang et al. (2004) – 19 cities	Schwartz et al. (1996)
Detroit	Bell et al. (2004) Bell et al. (2004) – 95 cities Huang et al. (2004) Huang et al. (2004) – 19 cities Schwartz (2004) Schwartz (2004) – 14 cities Ito (2003)	It0 (2003)
Houston	Bell et al. (2004) Bell et al. (2004) – 95 cities Huang et al. (2004) Huang et al. (2004) – 19 cities Schwartz (2004) Schwartz (2004) – 14 cities	
Los Angeles	Bell et al. (2004) Bell et al. (2004) – 95 cities Huang et al. (2004) Huang et al. (2004) – 19 cities	Linn et al. (2000)

Urban Area	Premature Mortality	Hospital Admissions for Respiratory and Cardiovascular Illnesses
New York	Bell et al. (2004) – 95 cities Huang et al. (2004) Huang et al. (2004) – 19 cities	Thurston et al. (1992)
Philadelphia	Bell et al. (2004) – 95 cities Huang et al. (2004) Huang et al. (2004) – 19 cities Moolgavkar et al. (1995)	
Sacramento	Bell et al. (2004) Bell et al. (2004) – 95 cities	
St. Louis	Bell et al. (2004) Bell et al. (2004) – 95 cities	
Washington, D.C.	Bell et al. (2004) – 95 cities	

*Studies listed for a given assessment location reported a C-R function specifically for that location unless otherwise specified. A study reporting a multi-city C-R function is listed for a given assessment location only if that location is included among the cities used to estimate the multi-city C-R function.
**This study estimated C-R functions for cardiovascular and respiratory mortality.

4.1.7 Selection of concentration-response functions

Studies often report more than one estimated C-R function for the same location and health endpoint. Sometimes models including different sets of co-pollutants are estimated in a study; sometimes different lags are estimated. In some cases, two or more different studies estimated a C-R function for O_3 and the same health endpoint in the same location (this is the case, for example, with O_3 and mortality associated with short-term exposures). For some health endpoints, there are studies that estimated multi-city O_3 C-R functions, while other studies estimated single-city functions.

All else being equal, a C-R function estimated in the assessment location is preferable to a function estimated elsewhere, since it avoids uncertainties related to potential differences due to geographic location. That is why the urban areas selected for the epidemiological studies-based O_3 risk assessment are those locations in which C-R functions have been estimated. There are several advantages, however, to using estimates from multi-city studies versus studies carried out in single cities. Multi-city studies are applicable to a variety of settings, since they estimate a central tendency across multiple locations. When they are estimating a single C-R function based on several cities, multi-city studies also tend to have more statistical power and provide effect estimates with relatively greater precision than single city studies due to larger sample sizes, reducing the uncertainty around the estimated coefficient. Because single-city and multi-city studies have different advantages, if a single-city C-R function has been estimated in a risk assessment location and a multi-city study that includes that location is also available for the same health endpoint, we used both functions for that location in the risk assessment.

Some O_3 epidemiological studies estimated C-R functions in which O_3 was the only pollutant entered into the health effects model (i.e., single pollutant models) as well as other C-R functions in which O_3 and one or more co-pollutants (e.g., PM, nitrogen dioxide, sulfur dioxide, carbon monoxide) were entered into the health effects model (i.e., multi-pollutant models). To the extent that any of the co-pollutants present in the ambient air may have contributed to the health effects attributed to O_3 in single pollutant models, risks attributed to O_3 might be overestimated where C-R functions are based on single pollutant models. However, if co-pollutants are highly correlated with O_3, their inclusion in an O_3 health effects model can lead to misleading conclusions in identifying a specific causal pollutant. When collinearity exists, inclusion of multiple pollutants in models often produces unstable and statistically insignificant effect estimates for both O_3 and the co-pollutants. Given that single and multi-pollutant models each have both potential advantages and disadvantages, with neither type clearly preferable over the other in all cases, we report risk estimates based on both single- and multi-pollutant models where both are available.

Many daily time-series epidemiological studies estimated C-R functions in which the O_3-related incidence on a given day depends only on same-day O_3 concentration or previous-day O_3 concentration (or some variant of those, such as a two-day average concentration). Such models necessarily assume that the longer pattern of O_3 levels preceding the O_3 concentration on a given day does not affect incidence of the health effect on that day. To the extent that an O_3-related health effect on a given day is affected by O_3 concentrations over a longer period of time, then these models would be mis-specified, and this mis-specification would affect the predictions of daily incidence based on the model.

A few recent studies (e.g., Bell et al., 2004; Huang et al., 2004) have estimated distributed lag models, in which health effect incidence is a function of O_3 concentrations on several days – that is, the incidence of the health endpoint on day t is a function of the O_3 concentration on day t, day (t-1), day (t-2), and so forth. Such models can be reconfigured so that the sum of the coefficients of the different O_3 lags in the model can be used to predict the changes in incidence on several days. For example, corresponding to a change in O_3 on day t in a distributed lag model with 0-day, 1-day, and 2-day lags considered, the sum of the coefficients of the 0-day, 1-day, and 2-day lagged O_3 concentrations can be used to predict the sum of incidence changes on days t, (t+1) and (t+2).

The extent to which time-series studies using single-day O_3 concentrations may underestimate the relationship between short-term O_3 exposure and mortality is unknown; however, there is some evidence, based on analyses of PM_{10} data, that mortality on a given day may be influenced by prior PM exposures up to more than a month before the date of death (Schwartz, 2000b). The extent to which short-term exposure studies (including those that consider distributed lags) may not capture the full impact of long-term exposures to O_3 is similarly not known. Currently, there is insufficient information to adequately adjust for the impact of longer-term exposure on mortality associated with O_3 exposures, and this is an important uncertainty that should be kept in mind as one considers the results from the short-term exposure O_3 risk assessment.

Epidemiological studies sometimes present several C-R functions, each incorporating a different lag structure. The question of lags and the problems of correctly specifying the lag structure in a model have been discussed extensively [see, for example, the PM AQCD (EPA, 2004, section 8.4.4); the PM Staff Paper (EPA, 2005e, sections 3.5.5.2 and 4.2.6.3); the draft O_3 AQCD (EPA, 2005d, section 7.1.3.3); and Schwartz, 2000)]. The draft O_3 AQCD notes that "analyzing a large number of lags and simply choosing the largest and most significant results may bias the air pollution risk estimates away from the null." (EPA, 2005d, section 7.1.3.3). On the other hand, there is recent evidence (Schwartz, 2000) that the relationship between PM and health effects may best be described by a distributed lag (i.e., the incidence of the health effect on day n is influenced by PM concentrations on day n, day n-1, day n-2 and so on). If this is true for O_3 as well, then a model with only a single lag may bias air pollution risk estimates towards the null. For mortality associated with short-term exposure to O_3, Bell et al. (2004) and Huang et al. (2004) present the results for distributed lag models that take into account exposure from the previous 6 days. When a study reported several single lag models for a health effect, we based our initial selection of the appropriate lag structure for each health effect on the overall assessment provided in the draft O_3 AQCD (EPA, 2005d), based on all studies reporting C-R functions for that health effect.

In summary:

- if a single-city C-R function was estimated in a risk assessment location and a multi-city function which includes that location was also available for the same health endpoint, we used both functions for that location in the risk assessment;

- risk estimates based on both single- and multi-pollutant models were used when both were available;

- distributed lag models were used, when available; when a study reported several single lag models for a health effect, we based our initial selection of the appropriate lag structure for the health effect on the overall assessment in the draft O_3 AQCD (EPA, 2005d), based on all studies reporting C-R functions for that health effect.

The locations, health endpoints, studies, and C-R functions included in that portion of the risk assessment based on epidemiological studies are summarized in Table 4-2.

Table 4-2. Summary of Locations, Concentration-Response Functions, Months Included and Counties Included

Risk Assessment Location	Ozone Season in Risk Assessment Location	Study/C-R Function	Health Endpoint	Other Pollutants in Model	Exposure Metric	Months Included for C-R Functions[1]	Counties Included for C-R Functions
Atlanta	March - October	Bell et al. (2004) - 95 cities	non-accidental mortality	none[2]	24-hr avg.	April - October	---
		Bell et al. (2004) - Atlanta	non-accidental mortality	none	24-hr avg.	April - October	Fulton, De Kalb [3]
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	PM_{10}	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	NO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	SO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	CO	24-hr avg.	June - September	---
		Huang et al. (2004) - Atlanta	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	Fulton, De Kalb
Boston	April - September	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
Chicago	April - September	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	PM_{10}	24-hr avg.	June - September	---

Risk Assessment Location	Ozone Season in Risk Assessment Location	Study/C-R Function	Health Endpoint	Other Pollutants in Model	Exposure Metric	Months Included for C-R Functions[1]	Counties Included for C-R Functions
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	NO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	SO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	CO	24-hr avg.	June - September	---
		Huang et al. (2004) - Chicago	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	Cook
		Schwartz (2004) - 14-city	non-accidental mortality	none	1-hr max.	May - September	---
		Schwartz (2004) - Chicago	non-accidental mortality	none	1-hr max.	May - September	Cook [4]
Cleveland	April - October	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
		Bell et al. (2004) - Cleveland	non-accidental mortality	none	24-hr avg.	April - October	Cuyahoga
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	PM_{10}	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	NO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	SO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	CO	24-hr avg.	June - September	---

Risk Assessment Location	Ozone Season in Risk Assessment Location	Study/C-R Function	Health Endpoint	Other Pollutants in Model	Exposure Metric	Months Included for C-R Functions[1]	Counties Included for C-R Functions
		Huang et al. (2004) - Cleveland	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	Cuyahoga
		Schwartz et al. (1996)	hosp. adms. for resp. illness	none	1-hr max.	"warm season"	Cuyahoga
Detroit	April - October	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
		Bell et al. (2004) - Detroit	non-accidental mortality	none	24-hr avg.	April - October	Wayne
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	PM_{10}	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	NO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	SO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	CO	24-hr avg.	June - September	---
		Huang et al. (2004) - Detroit	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	Wayne
		Schwartz (2004) - 14-city	non-accidental mortality	none	1-hr max.	May - September	---
		Schwartz (2004) - Detroit	non-accidental mortality	none	1-hr max.	May - September	Wayne [4]
		Ito (2003) – GAM stringent[5]	non-accidental mortality	none	24-hr avg.	April - October	Wayne

Risk Assessment Location	Ozone Season in Risk Assessment Location	Study/C-R Function	Health Endpoint	Other Pollutants in Model	Exposure Metric	Months Included for C-R Functions[1]	Counties Included for C-R Functions
		Ito (2003) – GAM stringent	circulatory mortality	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GAM stringent	respiratory mortality	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GAM stringent	unscheduled hospital adms. For pnuemonia	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GAM stringent	unscheduled hospital adms. For COPD	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GAM stringent	unscheduled hospital adms. for ischemic heart disease	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GAM stringent	unscheduled hospital adms. For heart failure	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GAM stringent	unscheduled hospital adms. For dysrhythmias	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GLM[6]	unscheduled hospital adms. For pnuemonia	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GLM	unscheduled hospital adms. For COPD	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GLM	unscheduled hospital adms. for ischemic heart disease	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GLM	unscheduled hospital adms. For heart failure	none	24-hr avg.	April - October	Wayne
		Ito (2003) – GLM	unscheduled hospital adms. For dysrhythmias	none	24-hr avg.	April - October	Wayne
Houston	All year	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---

Risk Assessment Location	Ozone Season in Risk Assessment Location	Study/C-R Function	Health Endpoint	Other Pollutants in Model	Exposure Metric	Months Included for C-R Functions[1]	Counties Included for C-R Functions
		Bell et al. (2004) - Houston	non-accidental mortality	none	24-hr avg.	All year	Harris
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	PM_{10}	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	NO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	SO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	CO	24-hr avg.	June - September	---
		Huang et al. (2004) - Houston	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	Harris
		Schwartz (2004) - 14-city	non-accidental mortality	none	1-hr max.	May - September	---
		Schwartz (2004) - Houston	non-accidental mortality	none	1-hr max.	May - September	Harris [4]
Los Angeles	All year	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
		Bell et al. (2004) - Los Angeles	non-accidental mortality	none	24-hr avg.	All year	Los Angeles
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	PM_{10}	24-hr avg.	June - September	---

Risk Assessment Location	Ozone Season in Risk Assessment Location	Study/C-R Function	Health Endpoint	Other Pollutants in Model	Exposure Metric	Months Included for C-R Functions[1]	Counties Included for C-R Functions
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	NO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	SO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	CO	24-hr avg.	June - September	---
		Huang et al. (2004) - Los Angeles	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	Los Angeles
		Linn et al. (2000)	unscheduled hosp. adms. for cardiovascular illness	none	24-hr avg.	All year; separately by season	Los Angeles, Riverside, San Bernardino, Orange[7]
		Linn et al. (2000)	unscheduled hosp. adms. for pulmonary illness	none	24-hr avg.	All year; separately by season	Los Angeles, Riverside, San Bernardino, Orange[7]
		Linn et al. (2000)	unscheduled hosp. adms. for cerebrovascular illness	none	24-hr avg.	All year; separately by season	Los Angeles, Riverside, San Bernardino, Orange[7]
New York	April - September	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	PM_{10}	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	NO_2	24-hr avg.	June - September	---

Risk Assessment Location	Ozone Season in Risk Assessment Location	Study/C-R Function	Health Endpoint	Other Pollutants in Model	Exposure Metric	Months Included for C-R Functions[1]	Counties Included for C-R Functions
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	SO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	CO	24-hr avg.	June - September	---
		Huang et al. (2004) - New York	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	Bronx, Kings, New York, Richmond, Queens, Westchester
		Thurston et al. (1992)	unscheduled hosp. adms. for respiratory illness	none	1-hr max.	June - August	Bronx, Kings, New York, Richmond, Queens[8]
		Thurston et al. (1992)	unscheduled hosp. adms. for asthma	none	1-hr max.	June - August	Bronx, Kings, New York, Richmond, Queens
Philadelphia	April - October	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	PM_{10}	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	NO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	SO_2	24-hr avg.	June - September	---
		Huang et al. (2004) - 19 cities	cardiovascular and respiratory mortality	CO	24-hr avg.	June - September	---

Risk Assessment Location	Ozone Season in Risk Assessment Location	Study/C-R Function	Health Endpoint	Other Pollutants in Model	Exposure Metric	Months Included for C-R Functions[1]	Counties Included for C-R Functions
		Huang et al. (2004) - Phila.	cardiovascular and respiratory mortality	none	24-hr avg.	June - September	Philadelphia
		Moolgavkar et al. (1995)	non-accidental mortality	none	24-hr avg.	June - August	Philadelphia
		Moolgavkar et al. (1995)	non-accidental mortality	TSP, SO_2	24-hr avg.	June - August	Philadelphia
Sacramento	All year	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
		Bell et al. (2004) - Sacramento	non-accidental mortality	none	24-hr avg.	All year	Sacramento
St. Louis	April - October	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---
		Bell et al. (2004) - St. Louis	non-accidental mortality	none	24-hr avg.		St. Louis city (FIPS 29510)
Washington, D.C.	April - October	Bell et al. (2004) - 95 cities	non-accidental mortality	none	24-hr avg.	April - October	---

[1] The months listed here are the months for which the C-R function was estimated. However, all C-R functions were *applied* in the risk assessment to April – Sept.
[2] The authors report that the results were robust to adjustment for PM_{10}, but do not report the multi-pollutant functions.
[3] Counties used by Bell et al. and Huang et al. are provided at http://www.ihapss.jhsph.edu/data/NMMAPS/documentation/counties.htm and in the June 2000 NMMAPS report (Number 94, Part II) are given in Appendix A, Table A.1.
[4] Personal communication via email (6-12-05) from J. Schwartz.
[5] Generalized Additive Model, using a stringent convergence criterion.
[6] Generalized Linear Model.
[7] Excluding mountain and desert regions of the first three counties.
[8] The paper doesn't list the counties, but notes that, in the case of New York City, surrounding counties were not included; this implies that only the five counties of which New York City is comprised are included in the analysis. This was confirmed in a personal communication from the author (G. Thurston).

4.1.8 Baseline health effects incidence considerations

The most common epidemiologically-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 changes in 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 "as is" air quality conditions) in each location is therefore needed.

Incidence rates express the occurrence of a disease or event (e.g., asthma episode, hospital admission, premature death) in a specific period of time, usually per year. Rates are expressed either as a value per population group (e.g., the number of cases in Philadelphia County) or a value per number of people (e.g., number of cases per 10,000 population), and may be age and sex specific. Incidence rates vary among geographic areas due to differences in population characteristics (e.g, age distribution) and factors promoting illness (e.g., smoking, air pollution levels). The sizes of the populations in the assessment locations that are relevant to the risk assessment (i.e., the populations for which the O_3 C-R functions are estimated and to which the baseline incidences refer) are given in Table 4-3.

We obtained estimates of location-specific baseline mortality rates for each of the O_3 risk assessment locations for 2002 from CDC Wonder, an interface for public health data dissemination from the Centers for Disease Control (CDC).[8] Rates were calculated for the specific sets of counties for which C-R functions were estimated. The mortality rates are derived from U.S. death records and U.S. Census Bureau post-censal population estimates, and are reported in Table 4-4. National rates are provided from CDC Wonder for 2002 for comparison. The epidemiological studies used in the risk assessment reported causes of mortality using the ninth revision of the International Classification of Diseases (ICD-9) codes. However, the tenth revision has since come out, and baseline mortality incidence rates for 2002 shown in Table 4-4 use ICD-10 codes. The groupings of ICD-9 codes used in the epidemiological studies and the corresponding ICD-10 codes used to calculate year 2002 baseline incidence rates are given in Table 4-5.

[8] United States Department of Health and Human Services (US DHHS), Centers for Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS), Compressed Mortality File (CMF) compiled from CMF 1968-1988, Series 20, No. 2A 2000, CMF 1989-1998, Series 20, No. 2E 2003 and CMF 1999-2002, Series 20, No. 2H 2004 on CDC WONDER On-line Database. See http://wonder.cdc.gov/.

Table 4-3. Relevant Population Sizes for O_3 Risk Assessment Locations

City	Counties	Population*		
		Total	Ages ≥30	Ages ≥ 65
Boston	Suffolk	690,000	---	---
Philadelphia	Philadelphia	1,517,000	---	---
New York	Bronx, Kings, Queens, New York, Richmond, Westchester	8,930,000	---	---
New York	Bronx, Kings, Queens, New York, Richmond	8,006,000	---	---
Washington, D.C.	Washington, D.C.	572,000	---	---
Atlanta	Fulton, DeKalb	1,482,000	---	---
St. Louis	St. Louis City	348,000	---	---
Chicago	Cook	5,376,000	---	---
Houston	Harris	3,400,000	---	---
Los Angeles	Los Angeles	9,518,000	---	---
Los Angeles	Los Angeles, Riverside, San Bernardino, Orange	---	8,378,000	---
Sacramento	Sacramento	1,223,000	---	---
Detroit	Wayne	2,061,000	---	---
Cleveland	Cuyahoga	1,394,000	---	217,000

* Total population and age-specific population estimates taken from the 2000 U.S. Census. Populations are rounded to the nearest thousand. The urban areas given in this table are those considered in the studies used in the O_3 risk assessment.

Table 4-4. Baseline Mortality Rates (per 100,000 Population) for 2002 for O_3 Risk Assessment Locations*

City	Counties	Type of Mortality (ICD-9 Codes)			
		Non-accidental (<800)	Cardiovascular and Respiratory (390-448; 490-496; 487; 480-486; 507)	Circulatory (390-459)	Respiratory (460-519)
Boston	Suffolk	736	---	---	---
Philadelphia	Philadelphia	1,057	242	---	---
New York	Bronx, Kings, Queens, New York, Richmond, Westchester	704	199	---	---
Washington, D.C.	Washington, D.C.	942	---	---	---
Atlanta	Fulton, DeKalb	623	131	---	---
St. Louis	St. Louis City	1147	---	---	---
Chicago	Cook	781	189	---	---
Houston	Harris	533	123	---	---
Los Angeles	Los Angeles	569	155	---	---
Sacramento	Sacramento	686	---	---	---
Detroit	Wayne	913	234	135	76
Cleveland	Cuyahoga	1,058	268	---	---
National	---	790	196	108	80

* Data from United States Department of Health and Human Services (US DHHS), Centers for Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS), Compressed Mortality File (CMF) compiled from CMF 1968-1988, Series 20, No. 2A 2000, CMF 1989-1998, Series 20, No. 2E 2003 and CMF 1999-2002, Series 20, No. 2H 2004 on CDC WONDER On-line Database. See http://wonder.cdc.gov/.

Table 4-5. ICD-9 Codes used in Epidemiological Studies and Corresponding ICD-10 Codes

Causes of Death	ICD-9 Codes	ICD-10 Codes
Non-accidental	<800	A00-R99
Cardiovascular and respiratory	390-448; 490-496; 487; 480-486; 507	G45.0-G45.2, G45.4-G45.9, G54.0, G93.6, G93.8, G93.8, G95.1, I00-I13.9, I20.0-I22.9, I24.1-I64, I67.0-I78.9, M21.9, M30.0-M31.9, R00.1, R00.8, R01.2, J40-J47, J67, J10-J18, J69
Cardiovascular	390-448	G45.0-G45.2, G45.4-G45.9, G54.0, G93.6, G93.8, G95.1, I00-I13.9, I20.0-I22.9, I24.1-I64, I67.0-I78.9, M21.9, M30.0-M31.9, R00.1, R00.8, R01.2
Circulatory	390-459	G45.0-G45.2, G45.4-G45.9, G54.0, G90.3, G93.6, G93.8, G95.1, I00-I13.9, I20.0-I22.9, I24.1-I64, I67.0-I87.9, I89.0-I95.9, I99, K66.1, K92.2, M21.9, M30.0-M31.9, R00.1, R00.8, R01.2, R58
Respiratory	460-519	J00-J01.9, J02.8-J02.9, J03.8-J64, J66.0-J94.9, J98.0-J98.9, P28.8, R06.5, R09.1

Hospital admissions studies included in the O_3 risk assessment were conducted in Los Angeles, Cleveland, and New York City. Because Thurston et al. (1992) estimated a linear C-R function for New York City, a baseline incidence rate is not required to estimate risks. However, a baseline incidence rate is needed to calculate hospital admissions as a percent of the total (baseline) hospital admissions. Baseline rates of unscheduled hospital admissions for respiratory illnesses and for asthma in New York City (the five boroughs) were calculated from the year 2001 data provided to us by the New York Statewide Planning and Research Cooperative. Baseline rates of unscheduled hospital admissions for Los Angeles (Los Angeles, Riverside, San Bernardino, and Orange Counties) were calculated from patient discharge data for 1999, obtained from California's Office of Statewide Health Planning and Development, which also provided records of hospital admissions for the study by Linn et al. (2000). The records provided for the Linn study included both ICD codes and All-Patient-Refined Diagnosis-Related Group (APR-DRG). Because Linn et al. (2000) used diagnosis categories based on the APR-DRG, we made sure that the records we obtained from California's Office of Statewide Health Planning and Development also contained the APR-DRG so that baseline incidence rates could be calculated for hospital admissions categories that matched those used in the Linn study. In addition, we used a flag in the dataset indicating whether an admission was scheduled or unscheduled to ensure that the rates we calculated were for unscheduled admissions only.

Schwartz et al. (1996) report several percentiles as well as the mean of the distribution of daily hospital admissions for respiratory illness (ICD-9 codes 460-519) among people ages 65 and older in Cuyahoga County, which contains Cleveland, Ohio, during the years 1988-90. The mean daily hospital admissions in this age group in Cuyahoga County was 22 in 1988-90. To

estimate a daily rate, we obtained the population age 65 and older in Cuyahoga County in 1990[9] and divided the mean daily hospital admissions for respiratory illness by that population. We will investigate the possibility of updating this baseline incidence rate. Baseline incidence rates for hospital admissions used in the risk assessment are shown in Table 4-6.

Table 4-6. Baseline Rates for Hospital Admissions

	Rate per 100,000 Relevant Population			
	Los Angeles[1]	**New York[2]**	**Detroit[3]**	**Cleveland[4]**
Relevant Population:	**Ages 30+**	**All Ages**	**Ages 65+**	**Ages 65+**
Admissions for:				
Cardiovascular illness (DRG Codes 103 – 144) - spring	431	---	---	---
Cardiovascular illness (DRG Codes 103 – 144) - summer	421	---	---	---
Pulmonary illness (DRG Codes 75 – 101) – spring	208	---	---	---
Pulmonary illness (DRG Codes 75 – 101) – summer	174	---	---	---
Respiratory illness (ICD codes 466, 480-486, 490, 491, 492, 493)	---	800	---	---
Asthma (ICD code 493)	---	327	---	---
Pneumonia (ICD codes 480-486)	---	---	2,068	---
Ischemic heart disease (ICD codes 410-414)	---	---	4,030	---
Heart failure (ICD code 428)	---	---	2,822	---
Respiratory illness ((ICD codes 460-519)	---	---	---	3,632

[1] Rates of unscheduled hospital admissions were calculated from patient discharge data for 1999, obtained from California's Office of Statewide Health Planning and Development, which also provided records of hospital admissions for the study by Linn et al. (2000).
2 Rates of unscheduled hospital admissions were calculated from patient discharge data for 2001, obtained from the New York Statewide Planning and Research Cooperative.
[3] Rates were calculated from hospitalization data for Wayne County for the year 2000, obtained from the Michigan Health and Hospital Association in April 2002.
[4] Based on mean daily hospital admissions for ages 65+ for ICD-9 codes 460-519 -- Table 1 in Schwartz et al. (1996).

4.1.9 Addressing uncertainty and variability

Any estimation of "as is" risk and reduced risks associated with just meeting the current O_3 standards should address both the variability and uncertainty that generally underlie such an analysis. In Section 3.1.5 we discussed the difference between uncertainty and variability, and gave examples of each. The discussion in that section is applicable to the uncertainty and variability to be addressed in the portion of the risk assessment based on epidemiological studies as well.

[9] 1990 U.S. Census, at: http://factfinder.census.gov/servlet/BasicFactsServlet

As with the controlled human exposure studies portion of the risk assessment, the epidemiology-based portion incorporates some of the variability in key inputs to the analysis by using location-specific inputs (e.g., location-specific population data and baseline incidence rates). Although spatial variability in these key inputs across all U.S. locations has not been fully characterized, variability across the selected locations is imbedded in the analysis by using, to the extent possible, inputs specific to each urban area. As in the controlled human exposure studies portion of the risk assessment, temporal variability is more difficult to address, because the risk assessment focuses on some unspecified time in the future. To minimize the degree to which values of inputs to the analysis may be different from the values of those inputs at that unspecified time, we have used the most current inputs available – for example, year 2004 air quality data for all of the urban locations, and the most recent available population data (from the 2000 Census). However, future changes in inputs have not been predicted (e.g., future population levels).

A number of important sources of uncertainty in the epidemiology-based portion of the risk assessment were addressed where possible. The following are among the major sources of uncertainty:

- Uncertainties related to estimating the C-R functions, including
    - uncertainty about the extent to which the association between O_3 and the health endpoint actually reflects a causal relationship.
    - uncertainty surrounding estimates of O_3 coefficients in C-R functions used in the analyses.
    - uncertainty about the specification of the model (including the shape of the C-R relationship), particularly whether or not there are thresholds below which no response occurs.
    - uncertainty related to the transferability of O_3 C-R functions from study locations and time periods to the locations and time periods selected for the risk assessment. A C-R function in a study location may not provide an accurate representation of the C-R relationship in the analysis location(s) and time periods because of
        - the possible role of associated co-pollutants, which vary from location to location and over time, in influencing O_3 risk,
        - variations in the relationship of total ambient exposure (both outdoor and ambient contributions to indoor exposure) to ambient monitoring in different locations (e.g, due to differences in air conditioning use in different regions of the U.S. or changes in usage over time),
        - differences in population characteristics (e.g., the proportions of members of sensitive subpopulations) and population behavior patterns across locations or over time in the same location.
- Uncertainties related to the air quality data, including

- the adjustment procedure that was used to simulate just meeting the current O_3 standard.

- uncertainties about estimated background concentrations for each location.

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

The specific sources of uncertainty in the O_3 risk assessment are described in detail below and are summarized in Table 4-7.

Table 4-7. Key Uncertainties in the Risk Assessment

Uncertainty	Comments
Causality	Statistical association does not prove causation. However, the risk assessment considers only health endpoints for which the overall weight of the evidence supports the assumption that O_3 is likely causally related.
Empirically estimated C-R relations	Because C-R functions are empirically estimated, there is uncertainty surrounding these estimates. Omitted confounding variables could cause bias in the estimated O_3 coefficients. However, including potential confounding variables that are highly correlated with one another can lead to unstable estimators. Both single- and multi-pollutant models were used where available. In addition, for those studies which provided both single-location and multiple-location estimates, single-location estimates were adjusted, using a Bayesian adjustment procedure, to make more efficient use of the data in the study. This is explained more fully below.
Functional form of C-R relation	Statistical significance of coefficients in an estimated C-R function does not necessarily mean that the mathematical form of the function is the best model of the true C-R relation.
Lag structure of C-R relation	There is some evidence that a distributed lag might be the most appropriate model for O_3 effects associated with short-term exposures. Most studies, however, included only one lag in their models. (Two important exceptions are Bell et al. (2004) and Huang et al. (2004).) Omitted lags could cause downward bias in the predicted incidence associated with a given reduction in O_3 concentrations.
Transferability of C-R relations	C-R functions may not provide an adequate representation of the C-R relationship in times and places other than those in which they were estimated. For example, populations in the analysis locations may have more or fewer members of sensitive subgroups than locations in which functions were derived, which would introduce additional uncertainty related to the use of a given C-R function in the analysis location. However, in the majority of cases, the risk assessment relies on C-R functions estimated from studies conducted in the same location.
Extrapolation of C-R relations beyond the range of observed O_3 data	A C-R relationship estimated by an epidemiological study may not be valid at concentrations outside the range of concentrations observed during the study.

Uncertainty	Comments
Adequacy of ambient O_3 monitors as surrogate for population exposure	Possible differences in how the spatial variation in ambient O_3 levels across each urban area are characterized in the original epidemiological studies compared to the more recent ambient O_3 data used to characterize current air quality would contribute to uncertainty in the health risk estimates.
Adjustment of air quality distributions to simulate just meeting current O_3 standards.	The pattern and extent of daily reductions in O_3 concentrations that would result if current O_3 standard were just met is not known. There remains uncertainty about the shape of the air quality distribution of hourly levels upon just meeting the current O_3 standard which will depend on future air quality control strategies.
Background O_3 concentrations	The calculation of O_3 risk associated with "as is" air quality and of reduced risks that would result if the current standard were just met requires as inputs the background O_3 concentrations in each of the assessment locations. Background concentrations were estimated based on the GEOS-CHEM model simulations for each location for all hours of an "average day" in a given month, for each of the months from April through September. There is uncertainty about these estimated background levels.
Baseline health effects data	Data on baseline incidence is uncertain for a variety of reasons. For example, location- and age-group-specific baseline rates may not be available in all cases. Baseline incidence may change over time for reasons unrelated to O_3.

We handled uncertainties in the risk assessment as follows:

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

- The uncertainty resulting from the statistical uncertainty associated with the estimate of the O_3 coefficient in a C-R function was characterized either by confidence intervals or by Bayesian credible intervals around the corresponding point estimate of risk. Confidence intervals and credible intervals express the range within which the true risk is likely to fall if the uncertainty surrounding the O_3 coefficient estimate were the only uncertainty in the analysis. They do not, for example, reflect the uncertainty concerning whether the O_3 coefficients in the study location and the assessment location are the same.

- Where possible, we made use of multi-city information to adjust location-specific estimates to make more efficient use of the data (see Section 4.1.9.1.2 below).

Although the O_3 risk assessment considered mortality as well as morbidity health effects, not all health effects which may result from O_3 exposure were included. Only those for which there was sufficient epidemiological evidence from studies which met the study selection criteria (see Section 4.1.5) were included in the risk assessment. Other possible health effects reported to be associated with exposure to O_3 are considered qualitatively in the draft O_3 Staff Paper. Thus, the draft O_3 risk assessment does not represent all of the health risks associated with O_3 exposures.

In addition, we limited application of a C-R function to only that portion of the population on which estimation of the function was based. For example, unscheduled hospital admissions for pneumonia, ischemic heart disease, and heart failure were examined in Ito (2003) for people ages 65 and older. It is likely that the effect of O_3 on hospital admissions for these illnesses and conditions does not begin at age 65; however, data are not available to estimate the number of cases avoided for younger age groups for the urban area examined by Ito (2003). Therefore, some number of potentially avoided health effects was likely not captured in this analysis.

4.1.9.1 Concentration-response functions

The C-R function is a key element of the O_3 risk assessment. The quality of the risk assessment depends, in part, on (1) whether the C-R functions used in the risk assessment are good estimates of the relationship between the population health response and ambient O_3 concentration in the study locations, (2) how applicable these functions are to the analysis periods and locations, and (3) the extent to which these relationships apply beyond the range of the O_3 concentrations from which they were estimated. These issues are discussed in the subsections below.

4.1.9.1.1 Uncertainty associated with the appropriate model form

The relationship between a health endpoint and O_3 can be characterized in terms of the form of the function describing the relationship – e.g., linear, log-linear, or logistic – and the value of the O_3 coefficient in that function. Although most epidemiological studies estimated O_3 coefficients in log-linear models, there is still substantial uncertainty about the correct functional form of the relationship between O_3 and various health endpoints – especially at the low end of the range of O_3 values, where data are generally too sparse to discern possible thresholds. While there are likely biological thresholds in individuals for specific health responses, the available epidemiological studies do not support or refute the existence of thresholds at the population level for O_3 exposures within the range of air quality observed in the studies.

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

The uncertainty associated with an estimate of the O_3 coefficient in a C-R function reported by a study depends on the sample size and the study design. The draft O_3 AQCD has evaluated the substantial body of O_3 epidemiological studies. In general, critical considerations in evaluating the design of an epidemiological study include the adequacy of the measurement of ambient O_3, the adequacy of the health effects incidence data, and the consideration of potentially important health determinants and potential confounders and effect modifiers such as:

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

The selection of studies included in the O_3 risk assessment was guided by the evaluations in the draft O_3 AQCD. One of the criteria for selecting studies addresses the adequacy of the measurement of ambient O_3. This criterion was that O_3 was directly measured, rather than estimated, on a reasonable proportion of the days in the study. This criterion was designed to minimize error in the estimated O_3 coefficients in the C-R functions used in the risk assessment.

To the extent that a study did not address all relevant factors (i.e., all factors that affect the health endpoint), there is uncertainty associated with the C-R function estimated in that study, beyond that reflected in the confidence or credible interval. It may result in either over- or underestimates of risk associated with ambient O_3 concentrations in the location in which the study was carried out. Techniques for addressing the problem of confounding factors and other study design issues have improved over the years, however, and the epidemiological studies currently available for use in the O_3 risk assessment provide a higher level of confidence in study quality than ever before.

When a study is conducted in a single location, the problem of possible confounding co-pollutants may be particularly difficult, if co-pollutants are highly correlated in the study

location. Single-pollutant models, which omit co-pollutants, may produce overestimates of the O_3 effect, if some of the effects of other pollutants (omitted from the model) are falsely attributed to O_3. Statistical estimates of an O_3 effect based on a multi-pollutant model can be more uncertain, and even statistically insignificant, if the co-pollutants included in the model are highly correlated with O_3. As a result of these considerations, we report risk estimates based on both single-pollutant and multi-pollutant models, when both are reported by a study.

As noted above, the uncertainty resulting from the statistical uncertainty associated with the estimate of the O_3 coefficient in a C-R function was characterized either by confidence intervals (if the coefficient was estimated using a classical statistical approach) or by Bayesian credible intervals (if the coefficient was estimated using a Bayesian approach) around the corresponding point estimate of risk.

Two studies, Bell et al. (2004) and Huang et al. (2004), reported both multi-location and single-location C-R functions in a variety of locations, using a Bayesian two-stage hierarchical model. In these cases, the single-location estimates can be adjusted to make more efficient use of the data from all locations. The resulting "shrinkage" estimates are so called because they "shrink" the location-specific estimates towards the overall mean estimate (the mean of the posterior distribution of the multi-location C-R function coefficient). The greater the uncertainty about the estimate of the location-specific coefficient relative to the estimate of between-study heterogeneity, the more the location-specific estimate is "pulled in" towards the overall mean estimate. Bell et al. (2004) calculated these shrinkage estimates, which were presented in Figure 2 of that paper. These location-specific shrinkage estimates, and their adjusted standard errors were provided to us by the study authors and were used in the risk assessment.

The location-specific estimates reported in Table 1 of Huang et al. (2004) are not "shrinkage" estimates. However, the study authors provided us with the posterior distribution for the heterogeneity parameter, τ, for their distributed lag model, shown in Figure 4(b) of their paper. Given this posterior distribution, and the original location-specific estimates presented in Table 1 of their paper, we calculated location-specific "shrinkage" estimates using a Bayesian method described in DuMouchel (1994) (see Section B-3 in Appendix B for a complete explanation of the calculation of these "shrinkage" estimates). As with the shrinkage estimates presented in Bell et al. (2004), the resulting Bayesian shrinkage estimates use the data from all of the locations considered in the study more efficiently than do the original location-specific estimates. The calculation of these shrinkage estimates is thus one way to address the relatively large uncertainty surrounding estimates of coefficients in location-specific C-R functions.

Several meta-analyses addressing the impact of various factors on estimates of mortality associated with short-term exposures to O_3 were just published in June 2005. We plan to review these analyses and explore whether they provide additional information that can be used to assist in characterizing the uncertainties associated with risk estimates for this health outcome.

4.1.9.1.3 Applicability of concentration-response functions in different locations

As described in Section 4.1.4, risk assessment locations were selected on the basis of where C-R functions have been estimated, to avoid the uncertainties associated with applying a C-R function estimated in one location to another location. However, multi-city C-R functions were also applied to any risk assessment location contained in the set of locations used to estimate the C-R function. The accuracy of the results based on a multi-location C-R function rests in part on how well this multi-location C-R function represents the relationship between ambient O_3 and the given population health response in the individual cities involved in the study.

The relationship between ambient O_3 concentration and the incidence of a given health endpoint in the population (the population health response) depends on (1) the relationship between ambient O_3 concentration and personal exposure to ambient-generated O_3 and (2) the relationship between personal exposure to ambient-generated O_3 and the population health response. Both of these are likely to vary to some degree from one location to another.

The relationship between ambient O_3 concentration and personal exposure to ambient-generated O_3 will depend on patterns of behavior, such as the amount of time spent outdoors, as well as on factors affecting the extent to which ambient-generated O_3 infiltrates into indoor environments. The relationship between personal exposure to ambient-generated O_3 and the population health response will depend on the population exposed.

Exposed populations differ from one location to another in characteristics that are likely to affect their susceptibility to O_3 air pollution. For instance, people with pre-existing conditions such as chronic bronchitis are probably more susceptible to the adverse effects of exposure to O_3, and populations vary from one location to another in the prevalence of specific diseases. Also, some age groups may be more susceptible than others, and population age distributions also vary from one location to another. Closely matching populations observed in studies to the populations of the assessment locations is not possible for many characteristics (for example, smoking status, workplace exposure, socioeconomic status, and the prevalence of highly susceptible subgroups).

Other pollutants may also play a role in either causing or modifying health effects, either independently or in combination with O_3 (see Section 8.1.3.2 in the 2004 PM CD and Section 7.1.3.5 in the draft O_3 AQCD). Inter-locational differences in these pollutants could also induce differences in the O_3 C-R relationship between one location and another.

In summary, the C-R relationship is most likely not the same everywhere. Even if the relationship between personal exposure to ambient-generated O_3 and population health response were the same everywhere, the relationship between ambient concentrations and personal exposure to ambient-generated O_3 differs among locations. Similarly, even if the relationship between ambient concentrations and personal exposure to ambient-generated O_3 were the same everywhere, the relationship between personal exposure to ambient-generated O_3 and population health response may differ among locations. In either case, the C-R relationship would differ.

4.1.9.1.4 Extrapolation beyond observed air quality levels

Although a C-R function describes the relationship between ambient O_3 and a given health endpoint for all possible O_3 levels (potentially down to zero), the estimation of a C-R function is based on real ambient O_3 values that are limited to the range of O_3 concentrations in the location in which the study was conducted. Thus, uncertainty in the shape of the estimated C-R function increases considerably outside the range of O_3 concentrations observed in the study.

Because we are interested in the effects of anthropogenic O_3, in this initial analysis, the O_3 risk assessment assumes that the estimated C-R functions adequately represent the true C-R relationship down to PRB O_3 levels in the assessment locations. Because those studies that reported the minimum O_3 levels observed all reported levels below PRB O_3 levels, the problem of extrapolation to levels below those air quality levels observed in a study does not arise.

The C-R relationship may also be less certain towards the upper end of the concentration range being considered in a risk assessment, particularly if the O_3 concentrations in the assessment location exceed the O_3 concentrations observed in the study location. Even though it may be reasonable to model the C-R relationship as log-linear over the ranges of O_3 concentrations typically observed in epidemiological studies, it may not be log-linear over the entire range of O_3 levels at the locations considered in the O_3 risk assessment.

4.1.9.2 The air quality data

4.1.9.2.1 Adequacy of O_3 air quality data

The method of averaging data from monitors across a metropolitan area in the risk assessment is similar to the methods used to characterize ambient air quality in most of the epidemiology studies. Ideally, the measurement of average hourly ambient O_3 concentrations in the study location is unbiased. In this case, unbiased risk predictions in the assessment location depend, in part, on an unbiased measurement of average hourly ambient O_3 concentrations in the assessment location as well. If, however, the measurement of average hourly ambient O_3 concentrations in the study location is biased, unbiased risk predictions in the assessment location are still possible if the measurement of average hourly ambient O_3 concentrations in the assessment location incorporates the same bias as exists in the study location measurements. Because this is not known, however, the errors in the O_3 measurements in the assessment locations are a source of uncertainty in the risk assessment.

O_3 air quality data were not available for all hours of the ozone season in the year chosen for the risk assessment in all of the assessment locations. Missing O_3 concentrations were filled in, as described in section 3.2 of the draft Exposure Assessment TSD.

Because the O_3 data in each assessment location were limited to a specific year (2004), the results of the risk assessment are generalizable to other years only to the extent that ambient O_3 levels in the available data are similar to ambient O_3 levels in those locations in the other years. A substantial difference between O_3 levels in the year used in the risk assessment and O_3 levels in the other years could imply a substantial difference in predicted incidences of health

effects. O_3 levels in 2004 in most of the 12 urban areas were somewhat lower than in other recent years, possibly due to both meteorological conditions that were not conducive to O_3 formation and lower emissions of NOx due to newly implemented regional controls on major power plants in the eastern U.S.

4.1.9.2.2 Estimation of PRB O_3 concentrations

The PRB O_3 concentrations that were used in the risk assessment are monthly averaged GEOS-CHEM model predictions, and the measured ambient O_3 concentrations are frequently lower than these PRB values. This raises the question of how best to deal with this in our estimation of risk above PRB. We considered two different approaches, described in Appendix E, calculating the bias expected in each case. As described in Appendix E, the relative magnitudes of the expected biases from the two approaches depends on whether we have overestimated or underestimated the monthly average PRB. The frequency with which the measured ambient O_3 concentrations are lower than our estimated PRB values suggests that these monthly PRB averages were overestimated. Fiore et al. (2002a) noted that the GEOS-CHEM model tends to overpredict O_3 concentrations in highly populated coastal areas, lending additional support for this hypothesis in Houston, where the frequency of estimated PRB concentrations above monitored "as is" concentrations was the greatest. On the assumption that monthly PRB averages were overestimated, the lowest-bias method to estimating risk above PRB is to set negative ΔO_3 (= "as is" O_3 concentration – PRB O_3 concentration) to zero. We believe this approach minimizes bias.

4.1.9.2.3 Simulation of reductions in O_3 concentrations to just meet the current standard

The pattern of hourly O_3 concentrations that would result if the current O_3 standards were just met in any of the assessment locations is, of course, not known. This therefore adds uncertainty to estimates of reduced risk when O_3 concentrations just meet the current standard.

As noted above, the current health risk assessment focuses on a single year and does not incorporate year-to-year variability, except in its use of design values based on the most recent three-year period available to determine the amount of adjustment to apply to the 2004 O_3 air quality data. If O_3 levels in the most recent year are the lowest of the three most recent years in a location, applying a design value based on the most recent three-year period available will result in a greater percent reduction in O_3 and greater reductions in risk and lower remaining risk than would be the case if the highest year of the three year period was evaluated in the assessment. We anticipate that in the next draft of the risk assessment we will examine the risk estimates for a different year within the three-year period on which the design value is based – e.g., 2002, in which O_3 levels were higher in most of the locations.

4.1.9.3 Baseline health effects incidence rates

Most of the C-R functions used in the O_3 risk assessment are log-linear (see equation 4-1 in Section 4.1.1). Given this functional form, the percent change in incidence of a health effect corresponding to a change in O_3 depends only on the change in O_3 levels (and not the actual

value of either the initial or final O_3 concentration). This percent change is multiplied by a baseline incidence, y_0, in order to determine the change in health effects incidence, as shown in equations (4-3) or (4-4) in Section 4.1.1:

$$\Delta y = y_0[1 - e^{-\beta\Delta x}] \quad \text{if } \Delta x \text{ is positive}$$

or

$$\Delta y = y_0[e^{\beta\Delta x} - 1] \quad \text{if } \Delta x \text{ is negative.}$$

Predicted changes in incidence therefore depend on the baseline incidence of the health effect.

4.1.9.3.1 Quality of incidence data

County-specific incidence data were available for mortality for all counties. We have also obtained hospital admissions baseline incidence data for all the urban areas for which we have hospital admissions C-R functions for O_3 (Detroit, Los Angeles, and Cleveland). This is clearly preferable to using non-local data, such as national or regional incidence rates. As with any health statistics, however, misclassification of disease, errors in coding, and difficulties in correctly assigning residence location are potential problems. These same potential sources of error are present in most epidemiological studies. In most cases, the reporting institutions and agencies utilize standard forms and codes for reporting, and quality control is monitored.

Data on hospital admissions are actually hospital discharge data rather than admissions data. Because of this, the date associated with a given hospital stay is the date of discharge rather than the date of admissions. Therefore, there may be some hospital admissions in an assessment location that are within the ozone season that are not included in the baseline incidence rate, if the date of discharge was after the ozone season ended, even though the date of admissions was within the ozone season. Similarly, there may be some hospital admissions that preceded the ozone season that are included in the baseline incidence rate because the date of discharge was within the ozone season. This is a very minor problem, however, partly because the percentage of such cases is likely to be very small, and partly because the error at the beginning of the ozone season (i.e., admissions that should not have been included but were) will largely cancel the error at the end of the ozone season (i.e., admissions that should have been included but were not).

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

Regardless of the data source, if actual incidence rates are higher than the incidence rates used, risks will be underestimated. If actual incidence rates are lower than the incidence rates used, then risks will be overestimated.

Both morbidity and mortality rates change over time for various reasons. One of the most important of these is that population age distributions change over time. The old and the extremely young are more susceptible to many health problems than is the population as a whole. The most recent available data were used in the risk assessment. However, the average age of the population in many locations will increase as post-World War II children age. Consequently, the baseline incidence rates for some endpoints may rise, resulting in an increase in the number of cases attributable to any given level of O_3 pollution. Alternatively, areas which experience rapid in-migration, as is currently occurring in the South and West, may tend to have a decreasing mean population age and corresponding changes in incidence rates and risk. Temporal changes in incidence are relevant to both morbidity and mortality endpoints. However, the most recent available data were used in all cases, so temporal changes are not expected to be a large source of uncertainty.

4.1.9.3.2 Lack of daily health effects incidence rates

Both ambient O_3 levels and the daily health effects incidence rates corresponding to ambient O_3 levels vary somewhat from day to day. Those analyses based on C-R functions estimated by short-term exposure studies calculate daily changes in incidence and sum them over the days of the O_3 season to predict a total change in health effect incidence during the O_3 season (standardized in this analysis to April through September). However, only annual baseline incidence rates are available. Average daily baseline incidence rates, necessary for short-term daily C-R functions, were calculated by dividing the annual rate by the number of days in the year for which the baseline incidence rates were obtained. To the extent that O_3 affects health, however, actual incidence rates would be expected to be somewhat higher than average on days with high O_3 concentrations; using an average daily incidence rate would therefore result in underestimating the changes in incidence on such days. Similarly, actual incidence rates would be expected to be somewhat lower than average on days with low O_3 concentrations; using an average daily incidence rate would therefore result in overestimating the changes in incidence on low O_3 days. Both effects would be expected to be small, however, and should largely cancel one another out.

4.2 Results

The results of the assessment of health risks associated with "as is" O_3 concentrations (representing levels measured in 2004 for all of the assessment locations) over PRB levels are presented in Section 4.2.1. The results of the assessment of the reduced health risks associated with O_3 concentrations that just meet the current 8-hour daily maximum standard are presented in Section 4.2.2. In both portions of the risk assessment, all estimated incidences were rounded to the nearest whole number, and all estimated incidences per 100,000 relevant population and all percentages were rounded to one decimal place. These rounding conventions are not intended to imply confidence in that level of precision, but rather to avoid the confusion that can result when a greater amount of rounding is used (for example, when the central tendency estimate and

both the lower and upper bounds of the 95 confidence or credible interval of incidence per 100,000 relevant population are all less than 0.5.)

There is uncertainty surrounding almost all estimates of incidence associated with "as is" O_3 concentrations in any location. Because we had to simulate the profile of O_3 concentrations that just meet the current 8-hour daily maximum O_3 standard in each location, there is additional uncertainty surrounding estimates of the reduced incidence associated with O_3 concentrations that just meet the current O_3 standard. We tried to minimize the extent of this uncertainty by avoiding the application of a C-R function estimated in one location to another location as much as possible. As discussed in Section 4.1.9, however, there are other sources of uncertainty. The uncertainty surrounding risk estimates resulting from the statistical uncertainty of the O_3 coefficients in the C-R functions used is characterized by ninety-five percent confidence or credible intervals around estimates of incidence, incidence per 100,000 relevant population, and the percent of total incidence that is O_3-related. In some cases, the lower bound of a confidence interval falls below zero. This does not imply that additional exposure to O_3 has a beneficial effect, but only that the estimated O_3 coefficient in the C-R function was not statistically significantly different from zero. Lack of statistical significance could mean that there is no relationship between O_3 and the health endpoint or it could mean that there wasn't sufficient statistical power to detect a relationship that exists.

4.2.1 Assessment of the health risks associated with "as is" O_3 concentrations in excess of policy relevant background levels

The results of the assessment of mortality risks associated with "as is" O_3 concentrations (representing levels measured in 2004 for all of the assessment locations) are summarized across urban areas in Figures 4-2a and b through 4-5a and b, and in Tables 4-8 and 4-9. Only one study, Ito (2003) for hospital admissions in Detroit, provided different lag models. The results from these different lag models are shown in Figures 4-6a and b. All results are for health risks associated with short-term exposures to O_3 concentrations in excess of PRB levels from April through September 2004. The percent of total incidence that is O_3-related is shown in Figures 4-2a through 4-6a; the incidence per 100,000 relevant population is shown in Figures 4-2b through 4-6b.

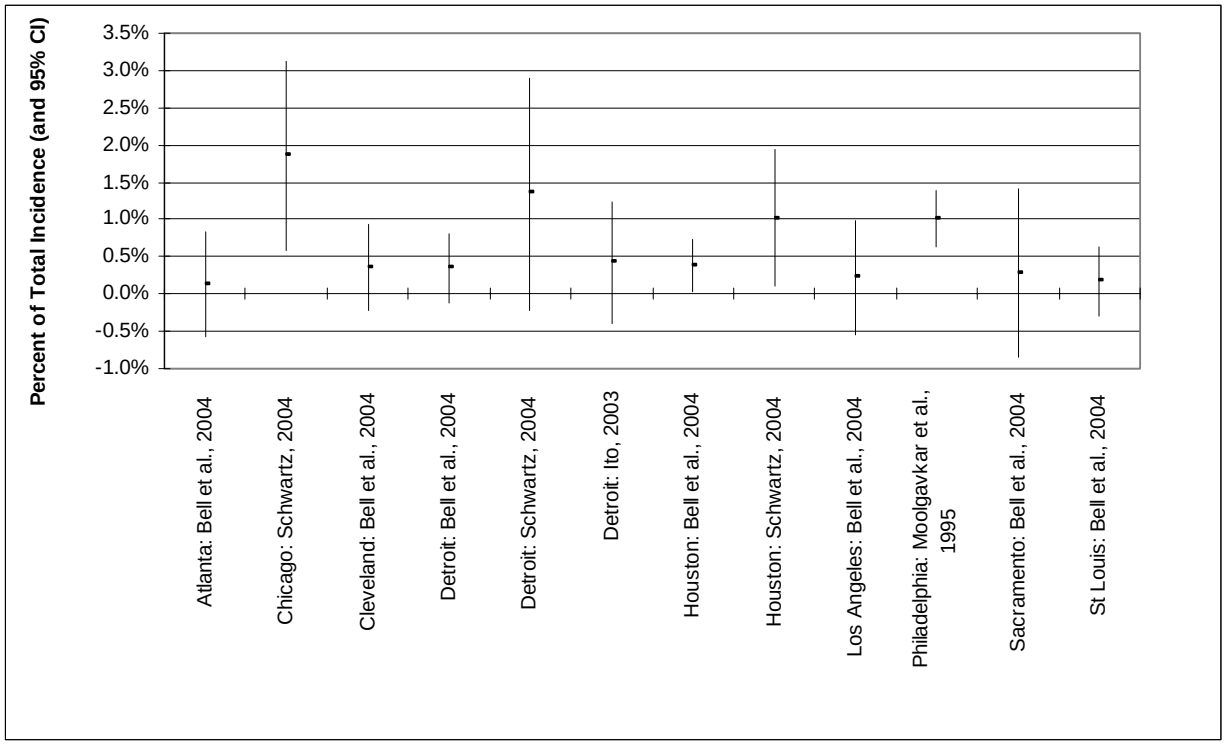
Although we carried out the analysis in each of the assessment locations, to reduce the number of tables in this section of the report, we selected one location (New York City) to include here for illustrative purposes. Table 4-10 shows results in New York for health endpoints associated with short-term exposure to "as is" O_3 concentrations in excess of estimated PRB concentrations. Results for the other locations corresponding to those shown for New York in Table 4-10 are shown in Appendix C, in Tables C-1 through C-11.

The central tendency estimates in all of the figures and in Tables 4-8 through 4-10 and C-1 through C-11 are based on the O_3 coefficients estimated in the studies, or, in the case of the location-specific estimates from Huang et al. (2004), on "shrinkage" estimates based on the O_3 coefficients estimated in the study (see Section 4.1.9.1.2). The ranges are based either on the 95 percent confidence intervals (CIs) around those estimates (if the coefficients were estimated

using classical statistical techniques) or on the 95 percent credible intervals (if the coefficients were estimated using Bayesian statistical techniques).

Figure 4-2. Estimated (Non-Accidental) Mortality Associated with Short-Term Exposure to O_3 Above Background: Single-Pollutant, Single-City Models (April – September, 2004)

4-2a. Estimated Percent of Total Incidence that is O_3-Related



4-2b. Estimated O_3-Related Cases per 100,000 Relevant Population

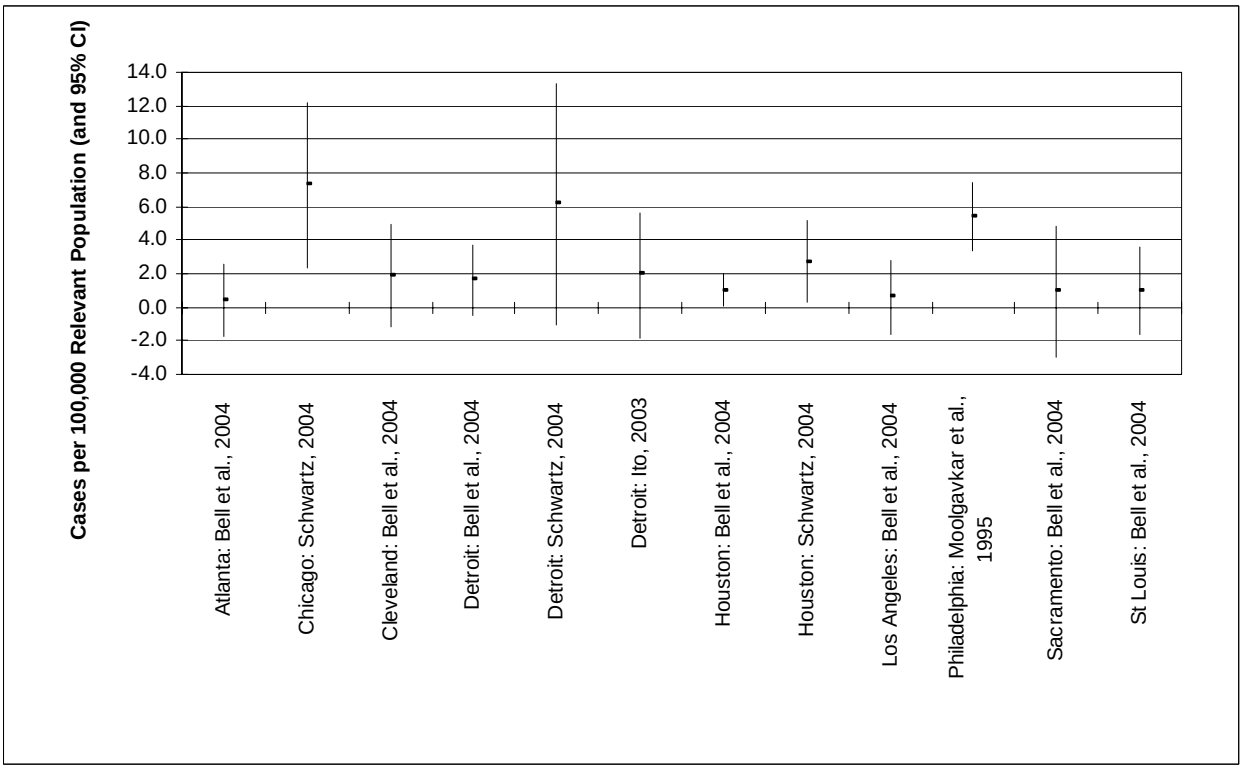


Figure 4-3. Estimated Cardiovascular and Respiratory Mortality Associated with Short-Term Exposure to O_3 Above Background (April – September, 2004): Single-Pollutant vs. Multi-Pollutant Models [Huang et al. (2004), additional pollutants, from left to right: none, PM_{10}, NO_2, SO_2, CO]

4-3a. Estimated Percent of Total Incidence that is O_3-Related

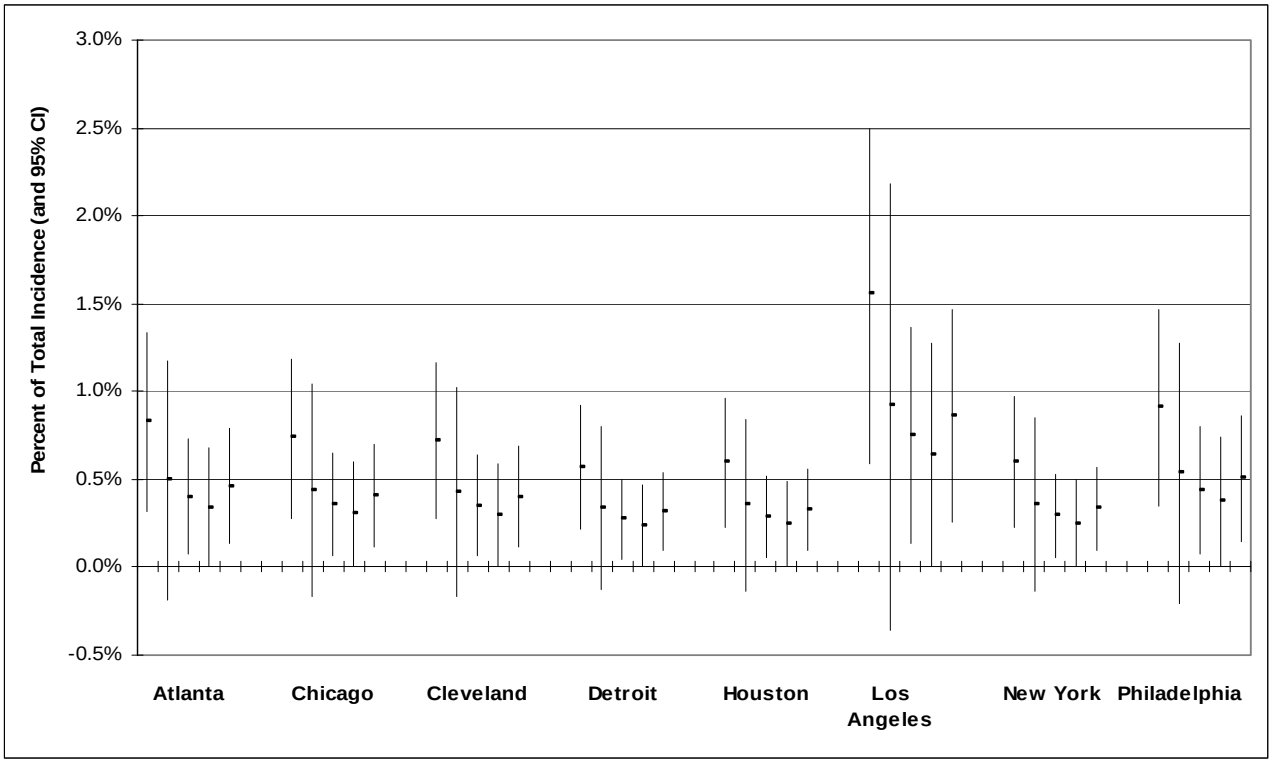


Figure 4-3b. Estimated O_3-Related Cases per 100,000 Relevant Population

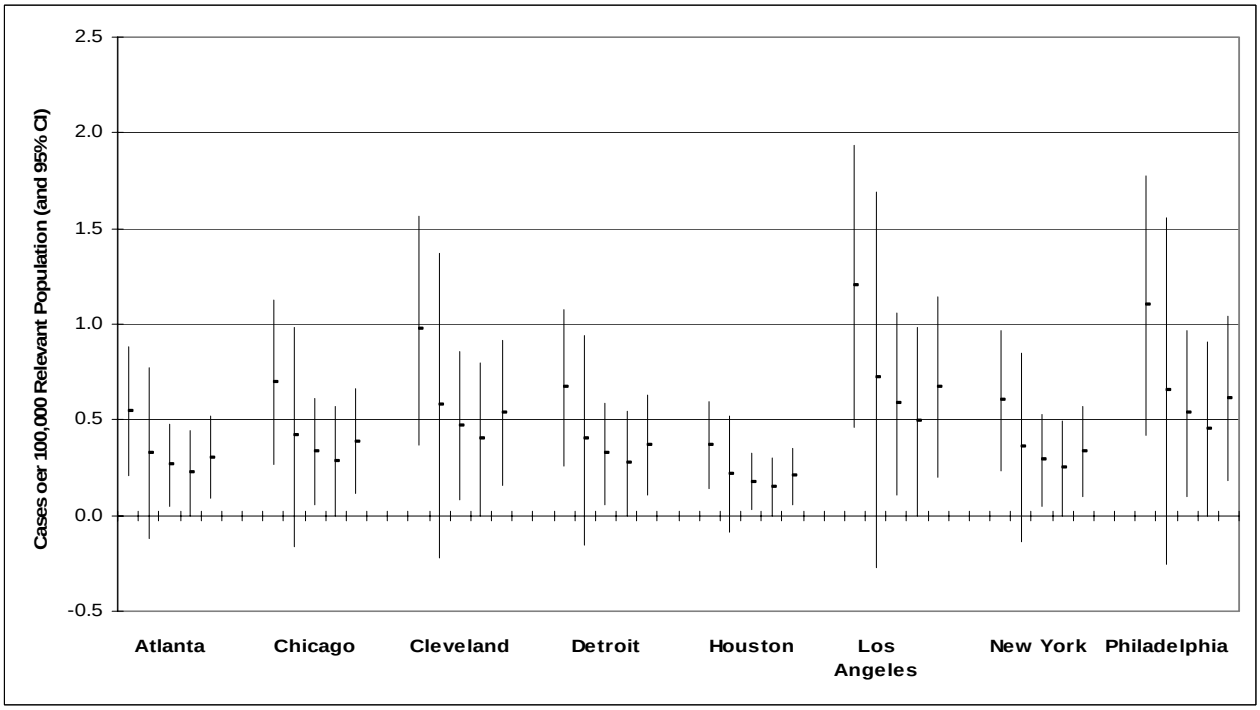


Figure 4-4. Estimated (Non-Accidental) Mortality Associated with Short-Term Exposure to O_3 Above Background (April – September, 2004): Single-City Model (left bar) vs. Multi-City Model (right bar)

4-4a. Estimated Percent of Total Incidence that is O_3-Related

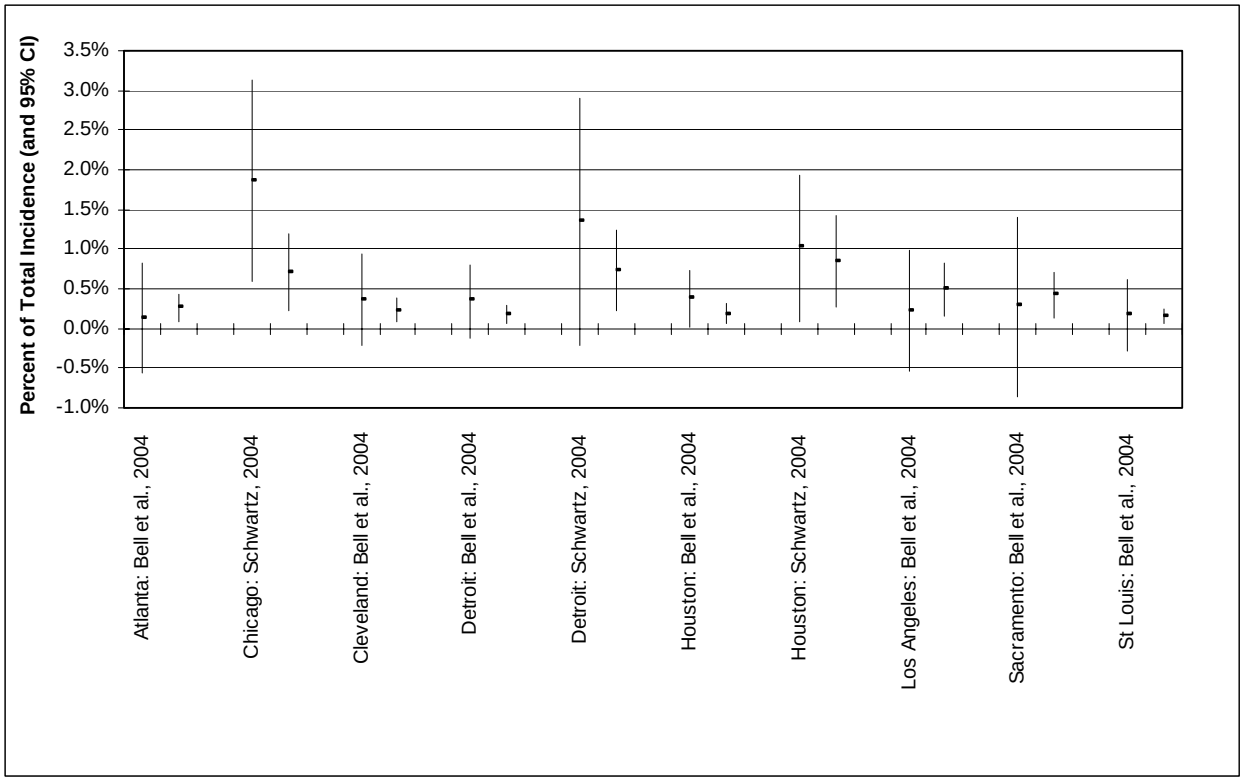


Figure 4-4b. Estimated O_3-Related Cases per 100,000 Relevant Population

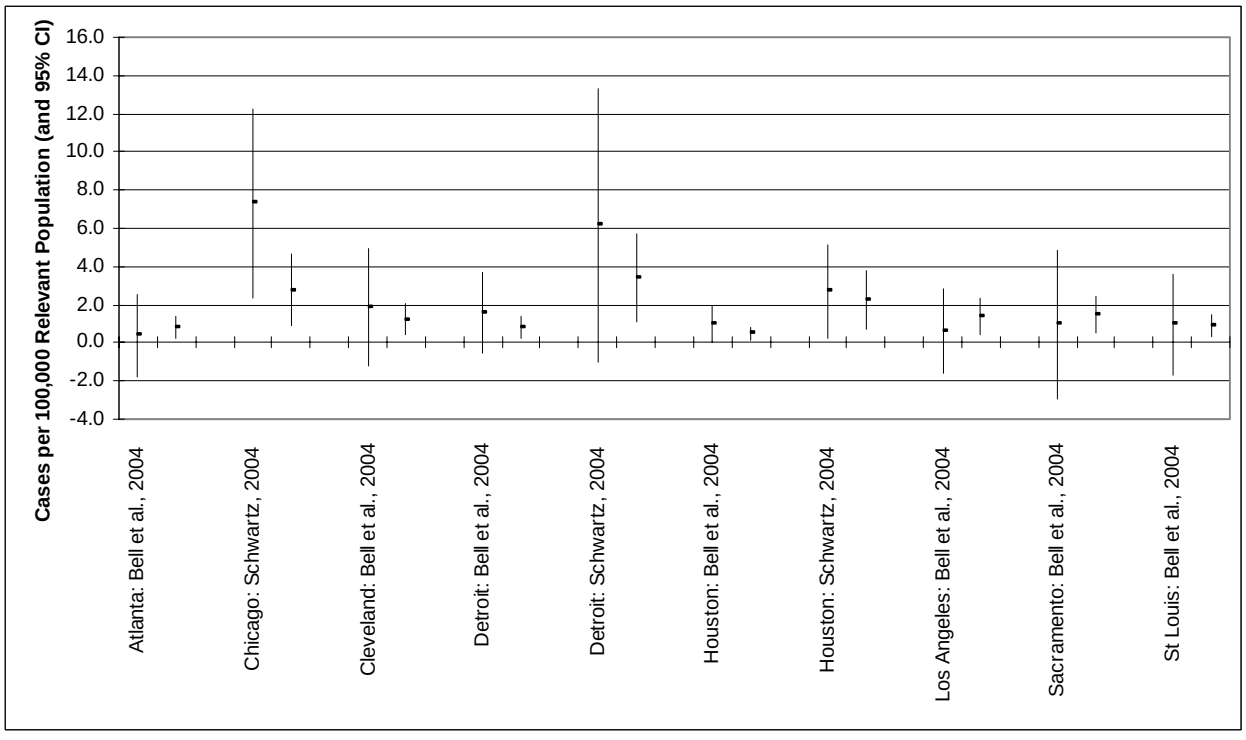


Figure 4-5. Estimated Cardiovascular and Respiratory Mortality Associated with Short-Term Exposure to O_3 Above Background (April – September, 2004): Single-City Model (left bar) vs. Multi-City Model (right bar) – Based on Huang et al. (2004)

4-5a. Estimated Percent of Total Incidence that is O_3-Related

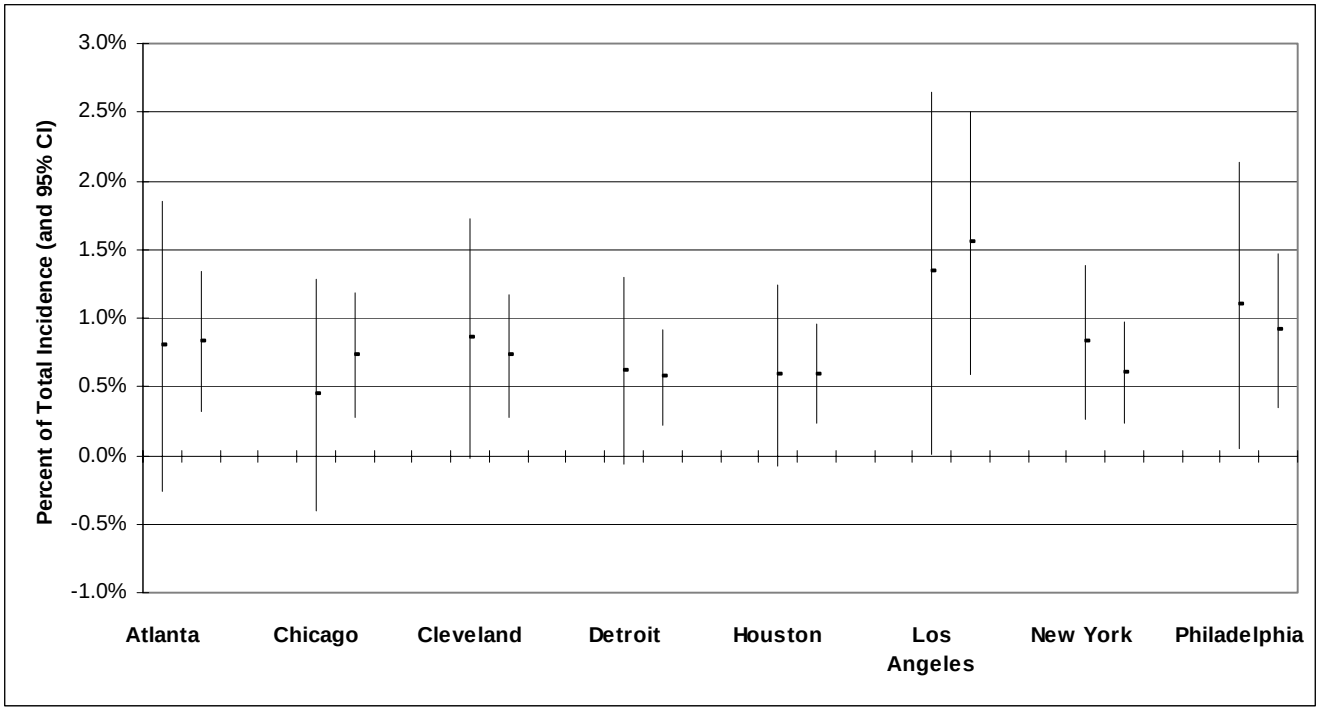


Figure 4-5b. Estimated O_3-Related Cases per 100,000 Relevant Population

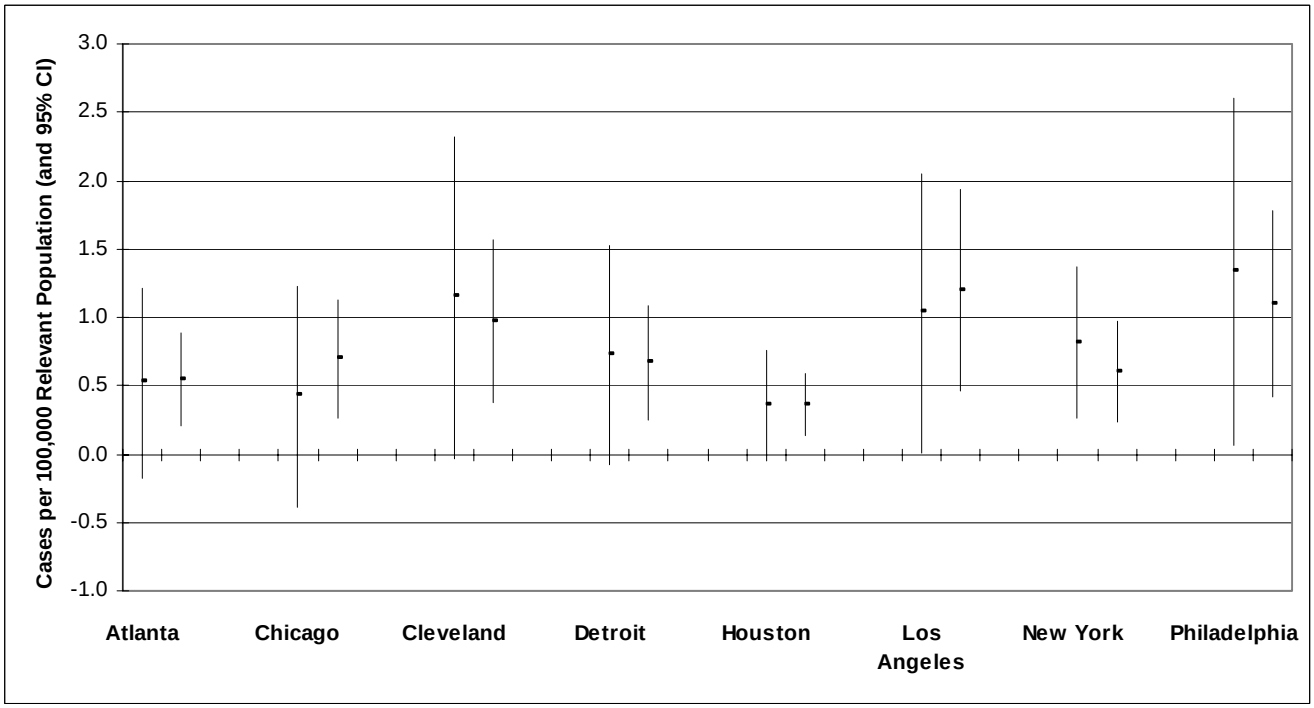


Figure 4-6. Estimated (Unscheduled) Hospital Admissions in Detroit Associated with Short-Term Exposure to O_3 Above Background (April – September, 2004): Different Lag Models – Based on Ito (2003) [bars from left to right are 0-day, 1-day, 2-day, and 3-day lag models]

4-6a. Estimated Percent of Total Incidence that is O_3-Related

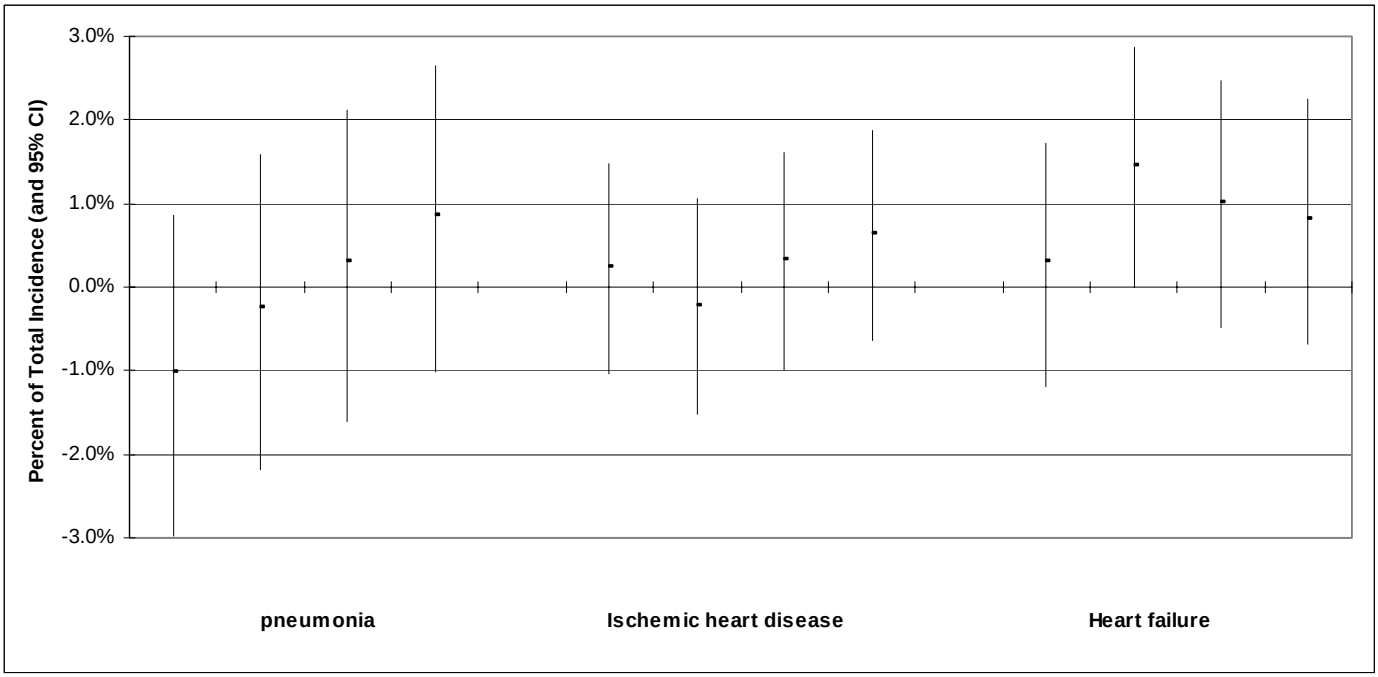


Figure 4-6b. Estimated O_3-Related Cases per 100,000 Relevant Population

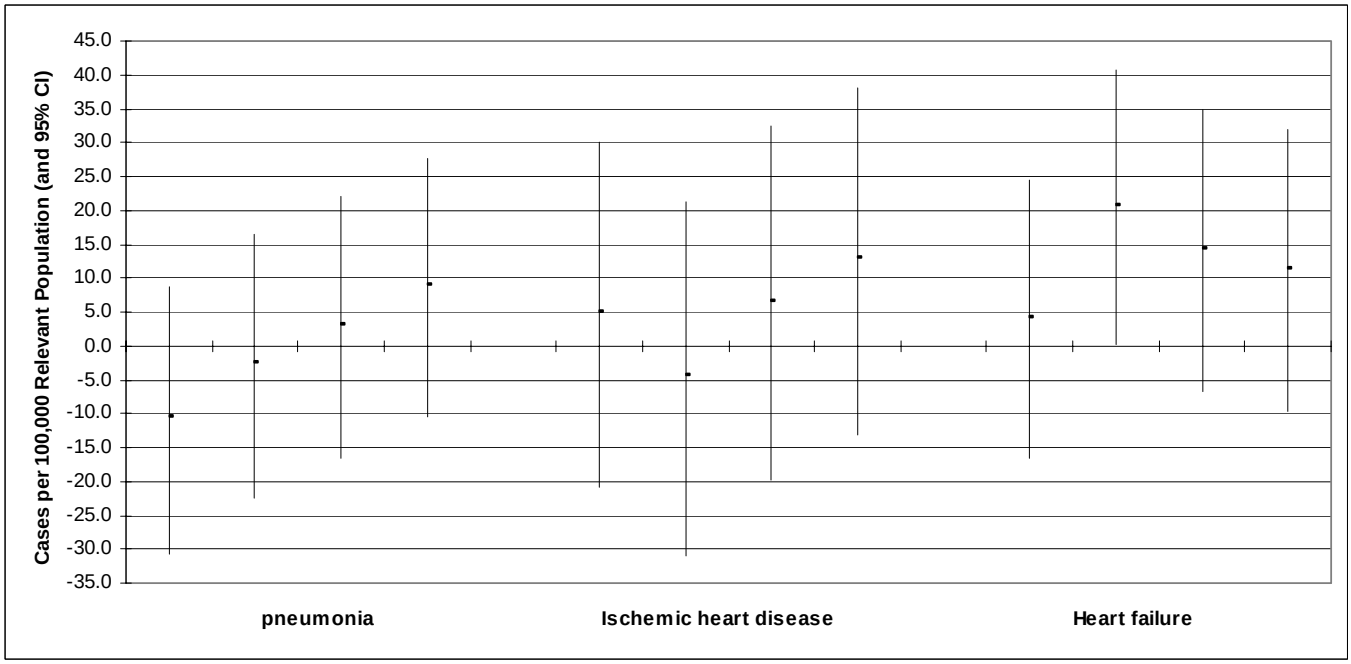


Table 4-8. Estimated Non-Accidental Mortality Associated with "As Is" O_3 Concentrations: April - September, 2004*

Location	Study	Lag	Exposure Metric	Non-Accidental Mortality Associated with O_3 Above Policy Relevant Background Levels**		
				Incidence	Incidence per 100,000 Relevant Population	Percent of Total Incidence
Atlanta	Bell et al. (2004)	distributed lag	24 hr avg.	6 (-26 - 38)	0.4 (-1.8 - 2.6)	0.1% (-0.6% - 0.8%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	12 (4 - 20)	0.8 (0.3 - 1.4)	0.3% (0.1% - 0.4%)
Boston	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	7 (2 - 12)	1.0 (0.3 - 1.7)	0.3% (0.1% - 0.5%)
Chicago	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	49 (16 - 81)	0.9 (0.3 - 1.5)	0.2% (0.1% - 0.4%)
	Schwartz (2004)	0-day lag	1 hr max.	394 (125 - 658)	7.3 (2.3 - 12.2)	1.9% (0.6% - 3.1%)
	Schwartz -- 14 US Cities (2004)	0-day lag	1 hr max.	148 (46 - 250)	2.8 (0.9 - 4.6)	0.7% (0.2% - 1.2%)
Cleveland	Bell et al. (2004)	distributed lag	24 hr avg.	27 (-17 - 69)	1.9 (-1.2 - 5)	0.4% (-0.2% - 0.9%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	17 (6 - 28)	1.2 (0.4 - 2)	0.2% (0.1% - 0.4%)
Detroit	Bell et al. (2004)	distributed lag	24 hr avg.	33 (-11 - 76)	1.6 (-0.5 - 3.7)	0.4% (-0.1% - 0.8%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	17 (6 - 28)	0.8 (0.3 - 1.4)	0.2% (0.1% - 0.3%)
	Schwartz (2004)	0-day lag	1 hr max.	128 (-21 - 274)	6.2 (-1 - 13.3)	1.4% (-0.2% - 2.9%)
	Schwartz -- 14 US Cities (2004)	0-day lag	1 hr max.	70 (22 - 117)	3.4 (1.1 - 5.7)	0.7% (0.2% - 1.2%)
	Ito (2003)	0-day lag	24 hr avg.	40 (-37 - 116)	2.0 (-1.8 - 5.6)	0.4% (-0.4% - 1.2%)
Houston	Bell et al. (2004)	distributed lag	24 hr avg.	35 (2 - 67)	1.0 (0.1 - 2)	0.4% (0% - 0.7%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	17 (6 - 28)	0.5 (0.2 - 0.8)	0.2% (0.1% - 0.3%)
	Schwartz (2004)	0-day lag	1 hr max.	93 (9 - 176)	2.7 (0.3 - 5.2)	1% (0.1% - 1.9%)
	Schwartz -- 14 US Cities (2004)	0-day lag	1 hr max.	78 (24 - 130)	2.3 (0.7 - 3.8)	0.9% (0.3% - 1.4%)
Los Angeles	Bell et al. (2004)	distributed lag	24 hr avg.	62 (-149 - 271)	0.6 (-1.6 - 2.8)	0.2% (-0.5% - 1%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	133 (45 - 221)	1.4 (0.5 - 2.3)	0.5% (0.2% - 0.8%)
New York	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	60 (20 - 100)	0.7 (0.2 - 1.1)	0.2% (0.1% - 0.3%)
Philadelphia	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	23 (8 - 38)	1.5 (0.5 - 2.5)	0.3% (0.1% - 0.5%)
	Moolgavkar et al. (1995)	1-day lag	24 hr avg.	82 (52 - 112)	5.4 (3.4 - 7.4)	1% (0.6% - 1.4%)

Location	Study	Lag	Exposure Metric	Non-Accidental Mortality Associated with O_3 Above Policy Relevant Background Levels**		
				Incidence	Incidence per 100,000 Relevant Population	Percent of Total Incidence
Sacramento	Bell et al. (2004)	distributed lag	24 hr avg.	12 (-36 - 59)	1.0 (-3 - 4.8)	0.3% (-0.9% - 1.4%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	18 (6 - 29)	1.4 (0.5 - 2.4)	0.4% (0.1% - 0.7%)
St Louis	Bell et al. (2004)	distributed lag	24 hr avg.	3 (-6 - 13)	1.0 (-1.7 - 3.6)	0.2% (-0.3% - 0.6%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	3 (1 - 5)	0.9 (0.3 - 1.5)	0.2% (0.1% - 0.3%)
Washington, D.C.	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	8 (3 - 14)	1.5 (0.5 - 2.4)	0.3% (0.1% - 0.5%)

*All results are for mortality (among all ages) associated with short-term exposures to O_3. All results are based on single-pollutant models.

**Incidence was quantified down to estimated policy relevant background levels. Incidences are rounded to the nearest whole number; incidences per 100,000 relevant population and percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence or credible intervals based on statistical uncertainty surrounding the O_3 coefficient.

Table 4-9. Estimated Cardiovascular and Respiratory Mortality Associated with "As Is" O_3 Concentrations: April - September, 2004*

Risk Assessment Location	Study Location	Cardiovascular and Respiratory Mortality Associated with O_3 Above Policy Relevant Background Levels**		
		Incidence	Incidence per 100,000 Relevant Population	Percent of Total Incidence
Atlanta	Atlanta	8 (-3 - 18)	0.5 (-0.2 - 1.2)	0.8% (-0.3% - 1.8%)
	19 U.S. Cities	8 (3 - 13)	0.5 (0.2 - 0.9)	0.8% (0.3% - 1.3%)
Chicago	Chicago	23 (-21 - 66)	0.4 (-0.4 - 1.2)	0.4% (-0.4% - 1.3%)
	19 U.S. Cities	38 (14 - 61)	0.7 (0.3 - 1.1)	0.7% (0.3% - 1.2%)
Cleveland	Cleveland	16 (0 - 32)	1.2 (0 - 2.3)	0.9% (0% - 1.7%)
	19 U.S. Cities	14 (5 - 22)	1.0 (0.4 - 1.6)	0.7% (0.3% - 1.2%)
Detroit	Detroit	15 (-2 - 31)	0.7 (-0.1 - 1.5)	0.6% (-0.1% - 1.3%)
	19 U.S. Cities	14 (5 - 22)	0.7 (0.3 - 1.1)	0.6% (0.2% - 0.9%)
Houston	Houston	12 (-2 - 26)	0.4 (0 - 0.8)	0.6% (-0.1% - 1.2%)
	19 U.S. Cities	13 (5 - 20)	0.4 (0.1 - 0.6)	0.6% (0.2% - 1%)
Los Angeles	Los Angeles	99 (1 - 195)	1.0 (0 - 2.1)	1.3% (0% - 2.6%)
	19 U.S. Cities	115 (44 - 185)	1.2 (0.5 - 1.9)	1.6% (0.6% - 2.5%)
New York	New York	73 (23 - 123)	0.8 (0.3 - 1.4)	0.8% (0.3% - 1.4%)
	19 U.S. Cities	54 (21 - 87)	0.6 (0.2 - 1)	0.6% (0.2% - 1%)
Philadelphia	Philadelphia	20 (1 - 39)	1.3 (0.1 - 2.6)	1.1% (0.1% - 2.1%)
	19 U.S. Cities	17 (6 - 27)	1.1 (0.4 - 1.8)	0.9% (0.3% - 1.5%)

*All results are for cardiovascular and respiratory mortality (among all ages) associated with short-term exposures to O_3. Results are based on single-pollutant single-city models or a single-pollutant multi-city model estimated in Huang et al. (2004).

**Incidence was quantified down to estimated policy relevant background levels. Incidences are rounded to the nearest whole number; incidences per 100,000 relevant population and percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% credible intervals based on statistical uncertainty surrounding the O_3 coefficient.

Table 4-10. Estimated Health Risks Associated with "As Is" O_3 Concentrations: New York, NY, April - September, 2004

Health Effects*	Study	Ages	Lag	Exposure Metric	Other Pollutants in Model	Health Effects Associated with O_3 Above Policy Relevant Background Levels**		
						Incidence	Incidence per 100,000 Relevant Population	Percent of Total Incidence
Mortality, non-accidental	Bell et al. -- 95 US Cities (2004)***	all	distributed lag	24 hr avg.	none	60 (20 - 100)	0.7 (0.2 - 1.1)	0.2% (0.1% - 0.3%)
Mortality, cardiovascular and respiratory	Huang et al. (2004)***	all	distributed lag	24 hr avg.	none	73 (23 - 123)	0.8 (0.3 - 1.4)	0.8% (0.3% - 1.4%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	none	54 (21 - 87)	0.6 (0.2 - 1)	0.6% (0.2% - 1%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	PM10	32 (-12 - 76)	0.4 (-0.1 - 0.8)	0.4% (-0.1% - 0.9%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	NO2	26 (5 - 47)	0.3 (0.1 - 0.5)	0.3% (0.1% - 0.5%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	SO2	22 (0 - 44)	0.2 (0 - 0.5)	0.2% (0% - 0.5%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	CO	30 (9 - 51)	0.3 (0.1 - 0.6)	0.3% (0.1% - 0.6%)
Hospital admissions (unscheduled), respiratory illness	Thurston et al. (1992)****	all	3-day lag	1 hr max.	none	447 (108 - 786)	5.6 (1.4 - 9.8)	1.3% (0.3% - 2.2%)
Hospital admissions (unscheduled), asthma	Thurston et al. (1992)****	all	1-day lag	1 hr max.	none	382 (81 - 683)	4.8 (1 - 8.5)	2.9% (0.6% - 5.2%)

*Health effects are associated with short-term exposures to O_3.

**Incidence was quantified down to estimated policy relevant background levels. Incidences are rounded to the nearest whole number; incidences per 100,000 relevant population and percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence or credible intervals based on statistical uncertainty surrounding the O_3 coefficient.

***New York in this study is defined as the five boroughs of New York City plus Westchester County.

****New York in this study is defined as the five boroughs of New York City.

As discussed in Section 4.1.4, assessment locations were chosen in part on the basis of whether an acceptable C-R function had been reported for that location. As a result, risks were estimated in a given assessment location only for those health endpoints for which there is at least one acceptable C-R function reported for that location. The set of health effects shown in Table 4-10 and Tables C-1 through C-11 therefore varies from one location to another. For example, hospital admissions for pneumonia, ischemic heart disease, and heart failure associated with short-term exposure to O_3 are included in Table C-5 for Detroit, but no hospital admissions endpoints are included in Tables C-1, C-2, or C-3 for Atlanta, Boston, and Chicago, respectively, because there was no study that met the selection criteria that reports a C-R function for hospital admissions reported in the O_3 epidemiological literature for any of those cities evaluated in the draft O_3 AQCD. For non-accidental mortality associated with short-term exposure to O_3, Figures 4-2a and b display estimates for only nine of the twelve risk assessment locations because acceptable (single-city) C-R functions for this health outcome were not available for the other three locations.

All results discussed below are for April through September, 2004. Figures 4-2a and b show estimated percent of non-accidental mortality and cases per 100,000 relevant population related to "as is" O_3 concentrations over PRB levels, based on single-pollutant, single-city models across all locations for which such models were available. Table 4-8 shows estimates of incidence, incidence per 100,000 relevant population, and percent of total incidence of non-accidental mortality related to "as is" O_3 concentrations over PRB levels in all locations, based on both single-city and multi-city models. Estimates of O_3-related (non-accidental) mortality ranged from 0.4 per 100,000 relevant population in Atlanta (Bell et al., 2004) to 7.3 per 100,000 relevant population in Chicago (Schwartz, 2004). Estimated O_3-related (non-accidental) mortality reported by Schwartz (2004) for Chicago, Detroit, and Houston, based on both the single-city and the multi-city C-R functions, tend to be higher than other estimates in those locations in large part because Schwartz used the 1-hr maximum O_3 concentration, rather than the 24-hour average, as the exposure metric. The changes from "as is" 1-hr maximum to PRB 1-hr maximum O_3 concentrations were generally larger in the assessment locations than the corresponding changes from "as is" 24-hr average to PRB 24-hr average O_3 concentrations. As a percent of total incidence, estimated O_3-related (non-accidental) mortality ranged from 0.1 percent in Atlanta (Bell et al., 2004) to 1.9 percent in Chicago (Schwartz, 2004). Although 7 of the 12 estimates from single-city single-pollutant models shown in Figures 4-2a and b were not statistically significant, all 12 were positive.

Figures 4-3a and b show estimated percent of cardiovascular and respiratory mortality and cases per 100,000 relevant population related to "as is" O_3 concentrations over PRB levels, based on multi-city single-pollutant versus multi-pollutant models from Huang et al. (2004) across all locations for which such models were available. Table 4-9 shows estimates of incidence, incidence per 100,000 relevant population, and percent of total incidence of cardiovascular and respiratory mortality related to "as is" O_3 concentrations over PRB levels in all risk assessment locations covered in Huang et al. (2004), based on both single-city and multi-city single-pollutant models from that study. Estimates of O_3-related cardiovascular and respiratory mortality ranged from 0.4 per 100,000 relevant population in Chicago (using the single-city C-R function) and Houston (using both the single-city and the multi-city C-R functions) to 1.3 per 100,000 relevant population in Philadelphia (using the single-city C-R

function). As a percent of total incidence, estimated O_3-related cardiovascular and respiratory mortality ranged from 0.4 percent in Chicago (using the single-city C-R function) to 1.6 percent in Los Angeles (using the multi-city C-R function). All of the estimates of O_3-related cardiovascular and respiratory mortality based on Huang et al. (2004), from both single-city and multi-city models, and from both single-pollutant and multi-pollutant models, were positive. Five of the single-city single-pollutant "shrinkage" estimates (for Atlanta, Chicago, Cleveland, Detroit, and Houston) and the estimate from the multi-city multi-pollutant model with PM_{10} were not statistically significant. All the rest of the estimates of O_3-related cardiovascular and respiratory mortality based on Huang et al. (2004) were statistically significant.

Figures 4-4a and b show estimated percent of non-accidental mortality that is O_3-related and O_3-related cases per 100,000 relevant population, based on single-city versus multi-city models across all locations for which both types of model were available. Estimates of O_3-related non-accidental mortality based on single-city models tended to have wider confidence or credible intervals than those based on multi-city models, with both multi-city models (from Bell et al., 2004 and Schwartz, 2004) producing statistically significant results. However, the choice of single-city versus multi-city model did not have a uniform affect on the magnitude of the point estimate. In some cases (Atlanta, Los Angeles, and Sacramento), the multi-city models produced larger estimates than the single-city models, while in other cases (Chicago, Cleveland, Detroit, Houston, and St. Louis) the reverse was true.

Bayesian credible intervals around the "shrinkage" estimates of O_3-related cardiovascular and respiratory mortality (see Section 4.1.9.1.2) based on single-city models in Huang et al. (2004) were uniformly larger than the corresponding credible intervals around estimates based on the multi-city model from that study. As noted above, all of the estimates were positive and, with the exception of the single-city estimate for Chicago, all were statistically significant.

The affect of O_3 lag structure on O_3-related unscheduled hospital admissions in Detroit (Ito 2003), shown in Figures 4-6a and b, depended on the illness. Estimated O_3-related pneumonia hospital admissions increased monotonically with increasing lag, with the greatest estimate predicted by a 3-day lag model. A 3-day lag model also predicted the greatest number of O_3-related ischemic heart disease hospital admissions. With the exception of the 0-day lag, O_3-related heart failure hospital admissions decreased monotonically with increasing lags. None of the estimates of O_3-related unscheduled hospital admissions in Detroit were statistically significant.

4.2.2 Assessment of the reduced health risks associated with O_3 concentrations that just meet the current 8-hour standard

The results of the assessment of the reduced mortality risks associated with O_3 concentrations that just meet the current 8-hour daily maximum standard are summarized across urban areas in Figures 4-7a and b through 4-10a and b, and in Tables 4-11 and 4-12. The results from the different lag models estimated in Ito (2003) for hospital admissions in Detroit are shown in Figures 4-11a and b. All results are for health risks associated with short-term exposures to O_3 concentrations in excess of PRB levels from April through September. The

percent of total incidence that is O_3-related is shown in Figures 4-7a through 4-11a; the incidence per 100,000 relevant population is shown in Figures 4-7b through 4-11b.

Table 4-13 shows results in New York City for health endpoints associated with short-term exposure to O_3 concentrations that just meet the current 8-hour daily maximum standard. Results for the other locations corresponding to those shown for New York in Table 4-13 are shown in Appendix D, in Tables D-1 through D-11.

As described in the previous section, the central tendency estimates in all of the figures and tables are based on the O_3 coefficients estimated in the studies, or, in the case of the location-specific estimates from Huang et al. (2004), on "shrinkage" estimates based on the O_3 coefficients estimated in the study (see Section 4.1.9.1.2). The ranges are based either on the 95 percent confidence intervals around those estimates (if the coefficients were estimated using classical statistical techniques) or on the 95 percent credible intervals (if the coefficients were estimated using Bayesian statistical techniques).

Figure 4-7. Estimated (Non-Accidental) Mortality Associated with Short-Term Exposure to O_3 Above Background When the Current 8-Hour Standard is Just Met: Single-Pollutant, Single-City Models (April – September)

4-7a. Estimated Percent of Total Incidence that is O_3-Related

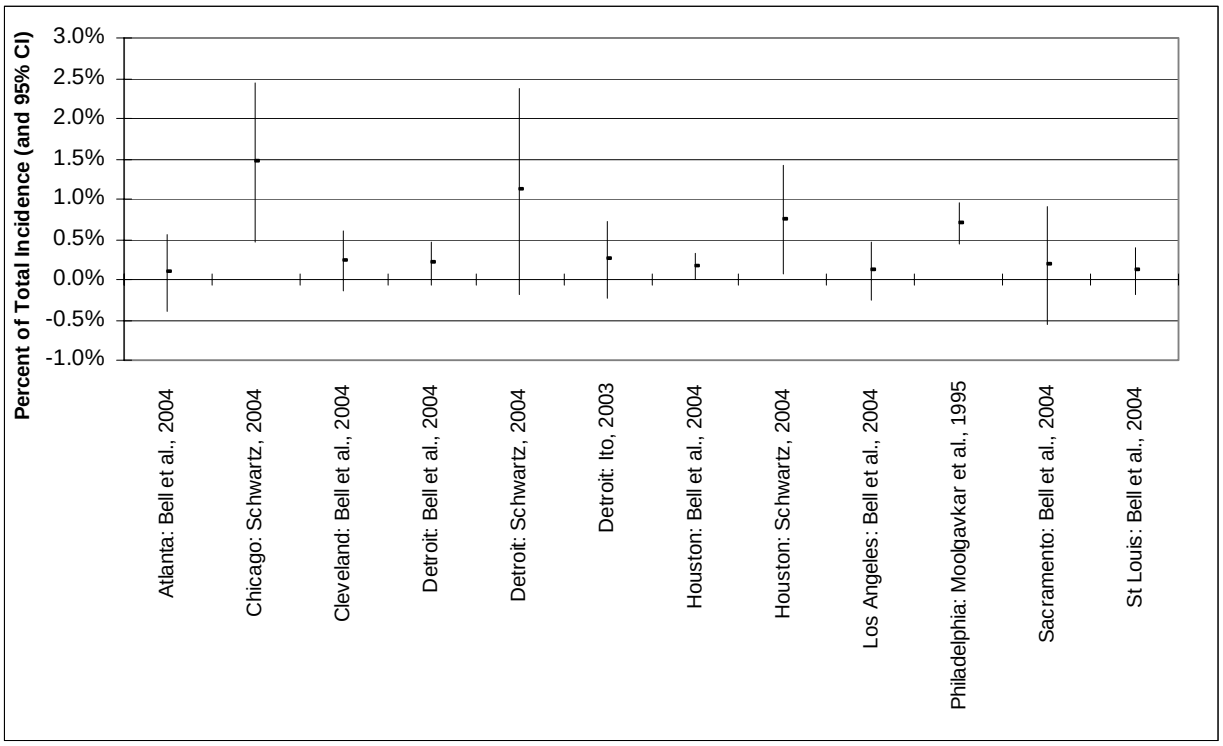


Figure 4-7b. Estimated O_3-Related Cases per 100,000 Relevant Population

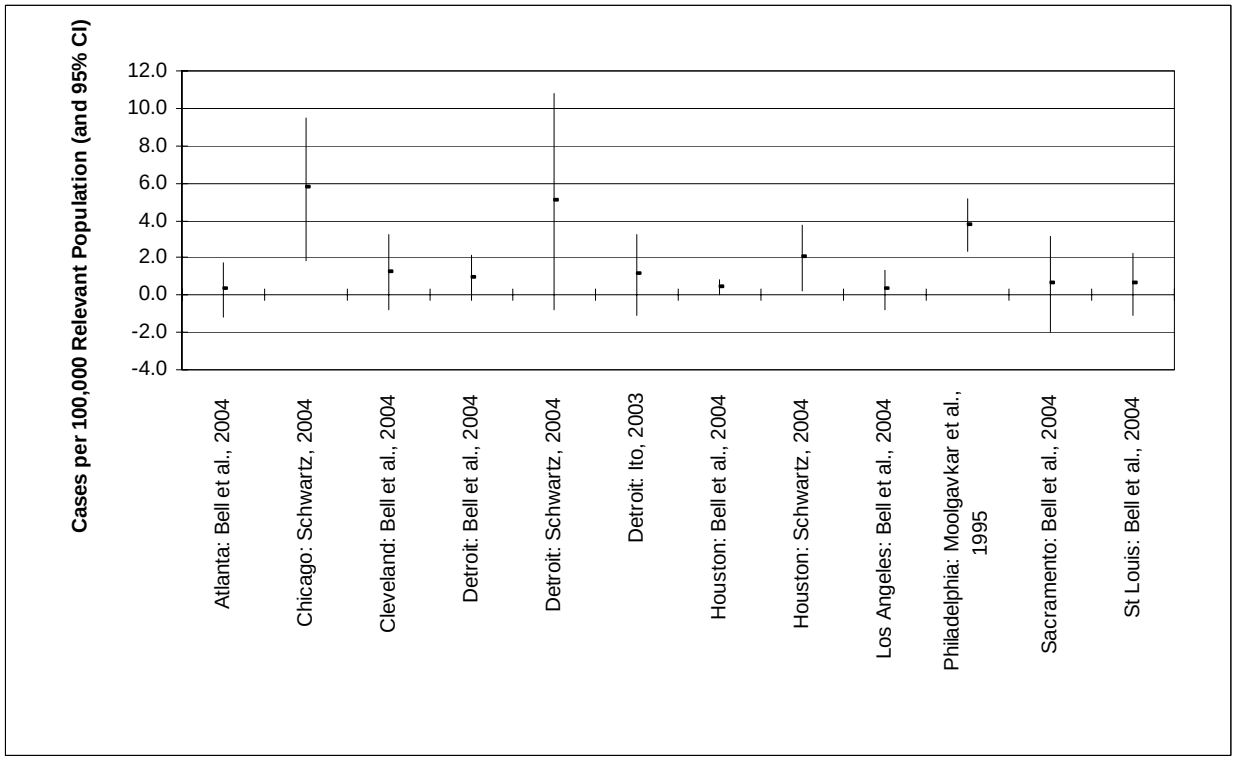


Figure 4-8. Estimated Cardiovascular and Respiratory Mortality Associated with Short-Term Exposure to O_3 Above Background When the Current 8-Hour Standard is Just Met (April – September): Single-Pollutant vs. Multi-Pollutant Models [Huang et al. (2004), additional pollutants, from left to right: none, PM_{10}, NO_2, SO_2, CO]

4-8a. Estimated Percent of Total Incidence that is O_3-Related

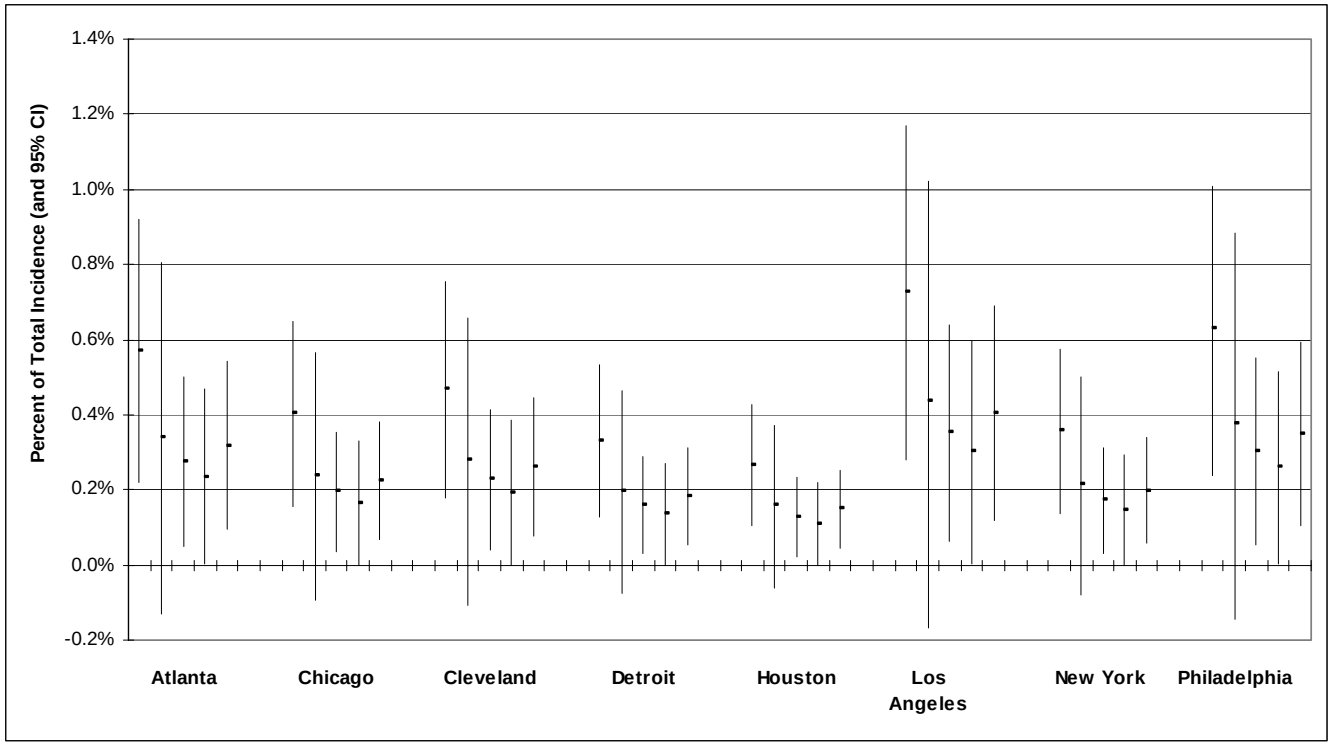


Figure 4-8b. Estimated O_3-Related Cases per 100,000 Relevant Population

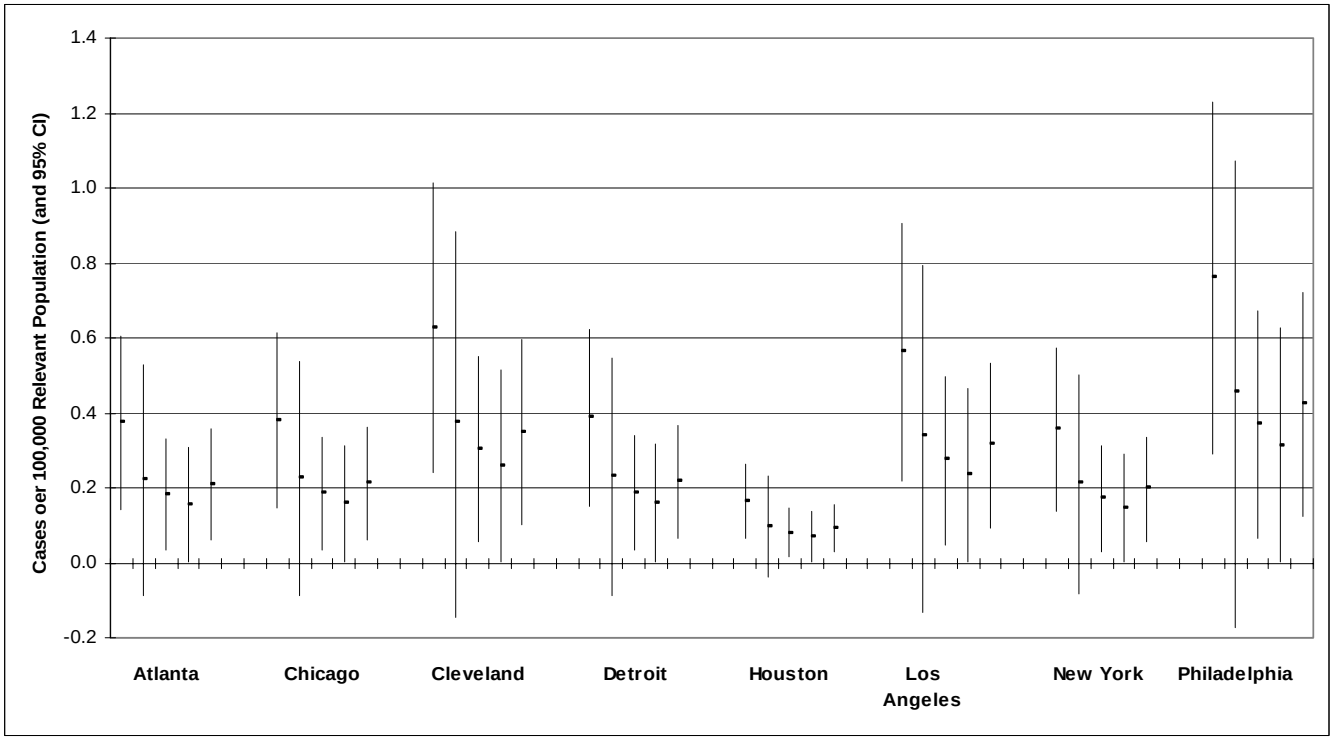


Figure 4-9. Estimated (Non-Accidental) Mortality Associated with Short-Term Exposure to O_3 Above Background When the Current 8-Hour Standard is Just Met (April – September): Single-City Model (left bar) vs. Multi-City Model (right bar)

4-9a. Estimated Percent of Total Incidence that is O_3-Related

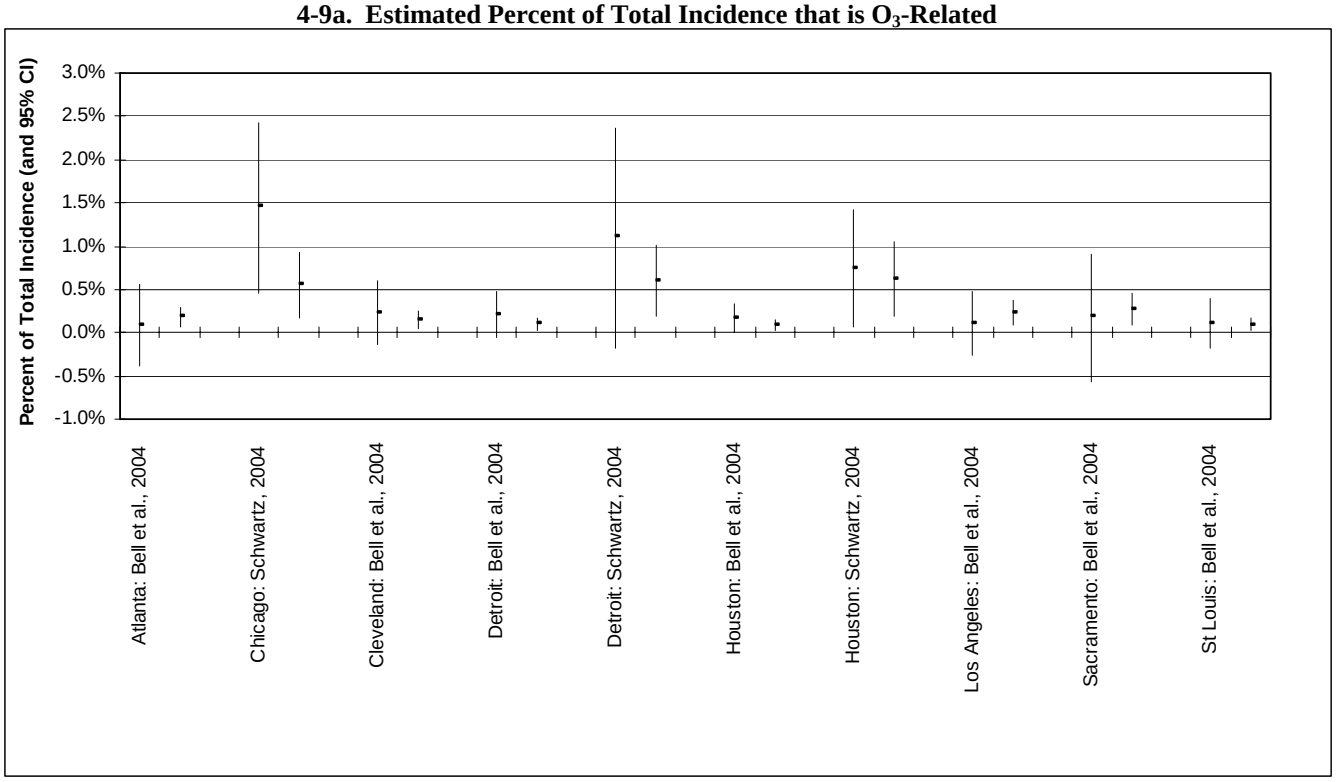


Figure 4-9b. Estimated O_3-Related Cases per 100,000 Relevant Population

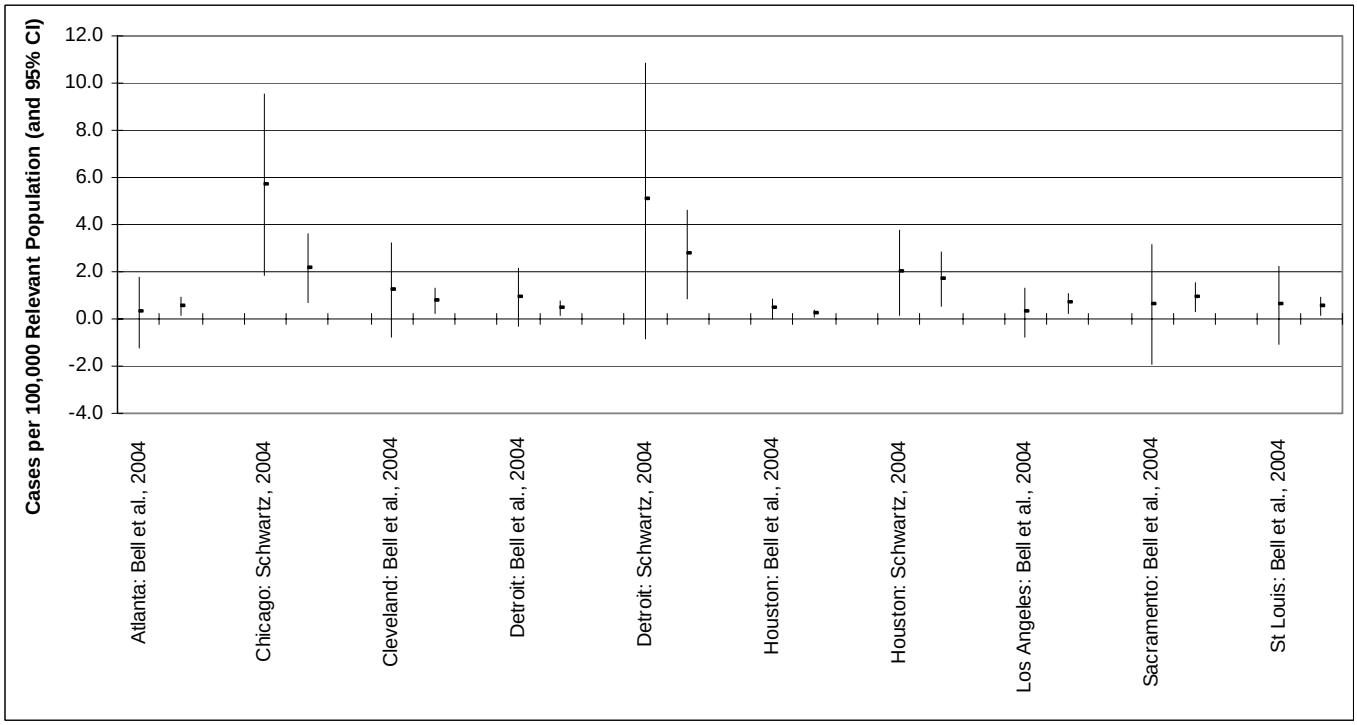


Figure 4-10. Estimated Cardiovascular and Respiratory Mortality Associated with Short-Term Exposure to O_3 Above Background When the Current 8-Hour Standard is Just Met (April – September): Single-City Model (left bar) vs. Multi-City Model (right bar) – Based on Huang et al. (2004)

4-10a. Estimated Percent of Total Incidence that is O_3-Related

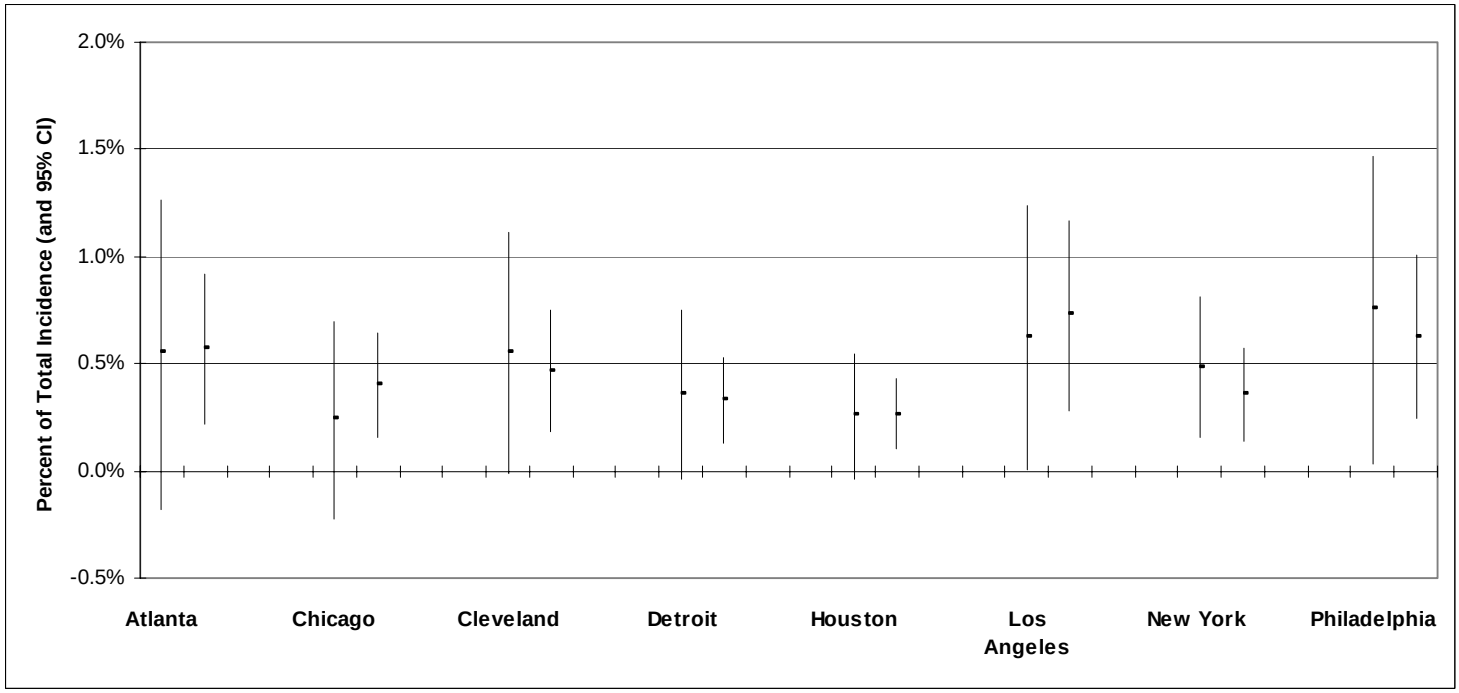


Figure 4-10b. Estimated O_3-Related Cases per 100,000 Relevant Population

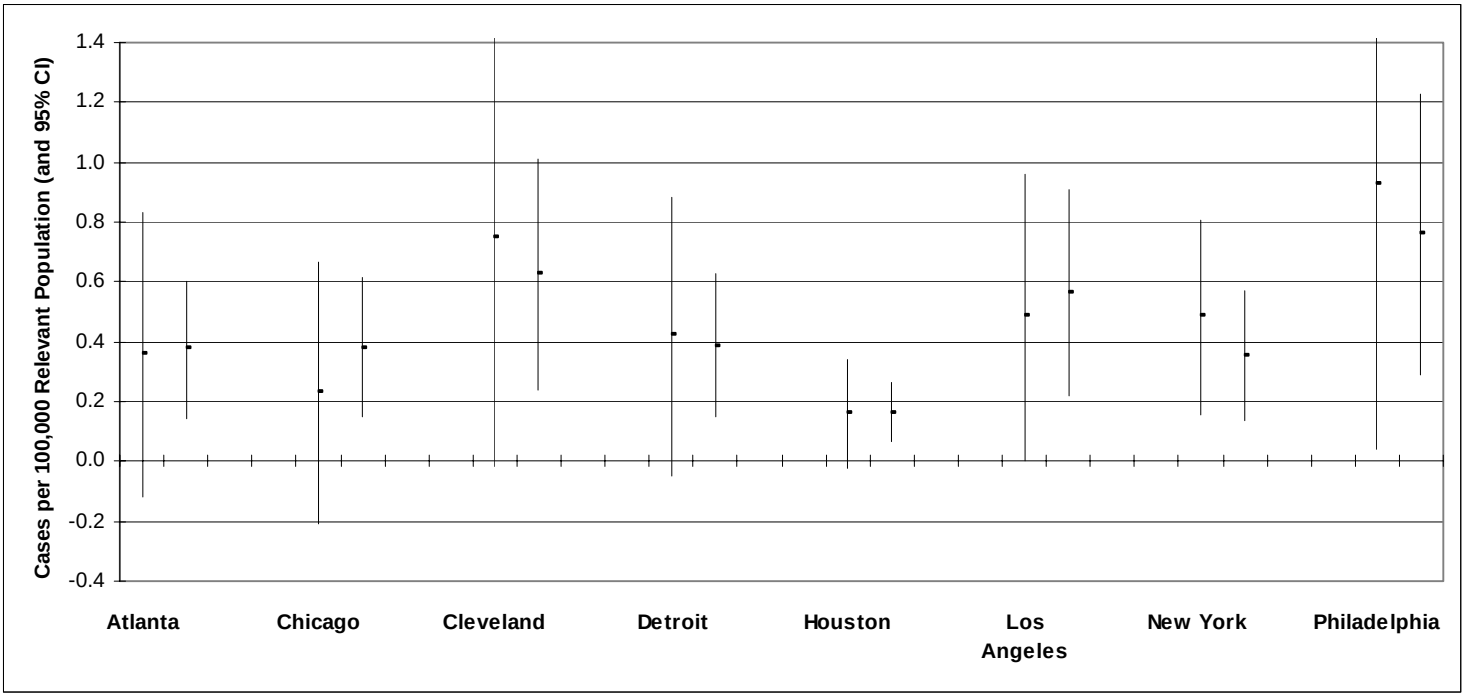


Figure 4-11. Estimated (Unscheduled) Hospital Admissions in Detroit Associated with Short-Term Exposure to O_3 Above Background When the Current 8-Hour Standard is Just Met (April – September): Different Lag Models – Based on Ito (2003) [bars from left to right are 0-day, 1-day, 2-day, and 3-day lag models]

4-11a. Estimated Percent of Total Incidence that is O_3-Related

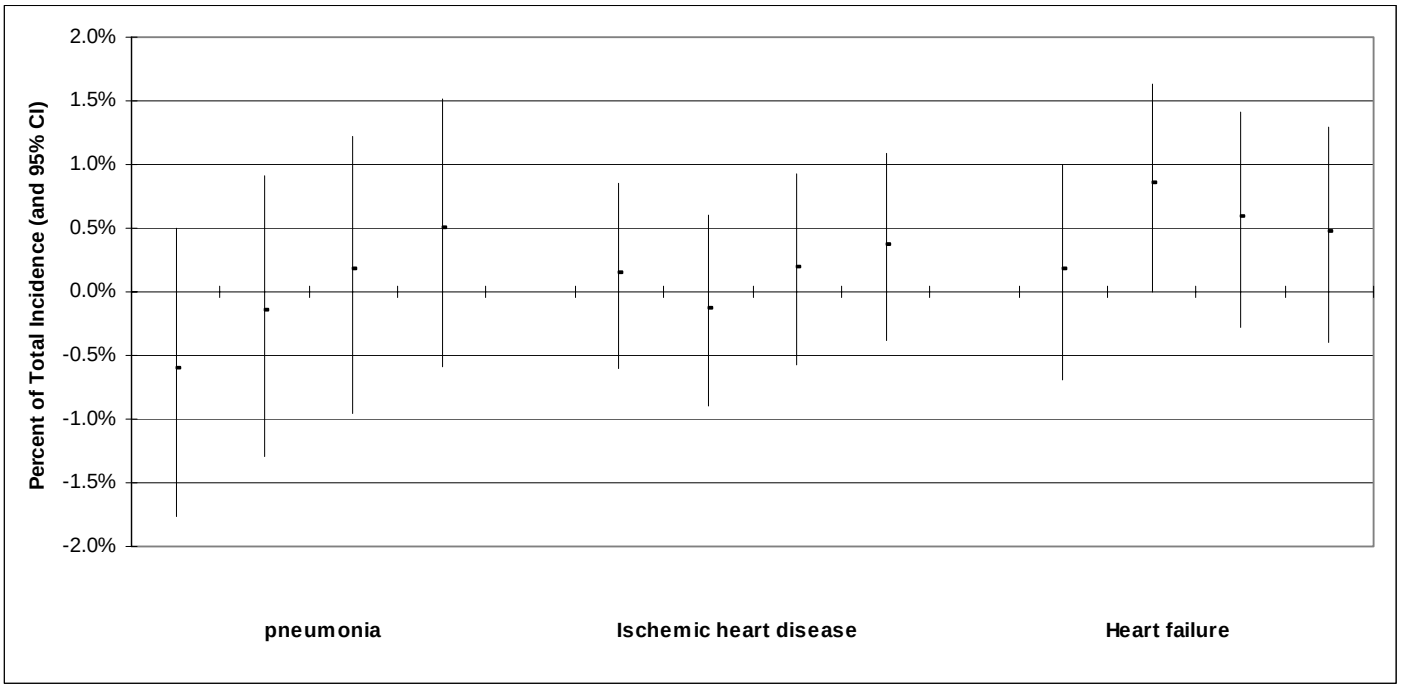


Figure 4-11b. Estimated O_3-Related Cases per 100,000 Relevant Population

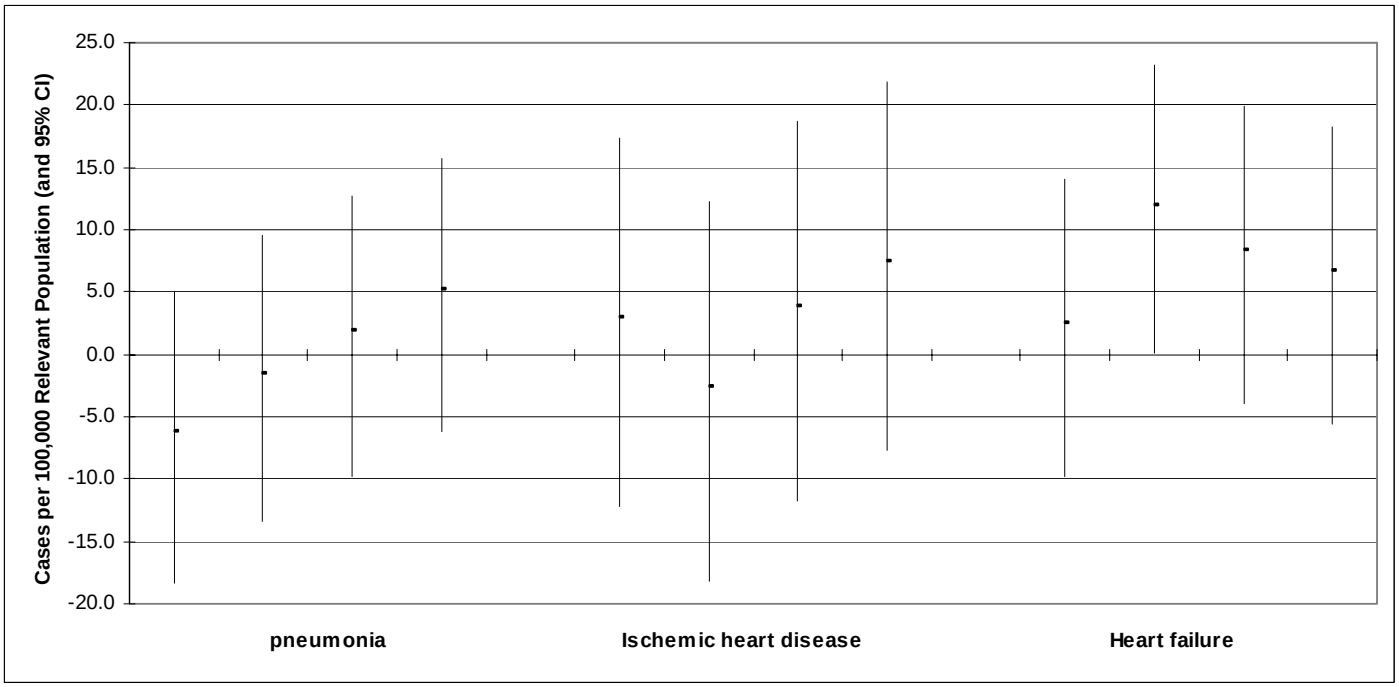


Table 4-11. Estimated Non-Accidental Mortality Associated with O_3 Concentrations that Just Meet the Current 8-Hour Daily Maximum Standard: April - September*

Location	Study	Lag	Exposure Metric	Non-Accidental Mortality Associated with O_3 Concentrations that Just Meet the Current O_3 Standard**		
				Incidence	Incidence per 100,000 Relevant Population	Percent of Total Incidence
Atlanta	Bell et al. (2004)	distributed lag	24 hr avg.	4 (-18 - 26)	0.3 (-1.2 - 1.8)	0.1% (-0.4% - 0.6%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	8 (3 - 14)	0.6 (0.2 - 0.9)	0.2% (0.1% - 0.3%)
Boston	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	5 (2 - 9)	0.8 (0.3 - 1.3)	0.2% (0.1% - 0.3%)
Chicago	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	27 (9 - 44)	0.5 (0.2 - 0.8)	0.1% (0% - 0.2%)
	Schwartz (2004)	0-day lag	1 hr max.	307 (98 - 512)	5.7 (1.8 - 9.5)	1.5% (0.5% - 2.4%)
	Schwartz -- 14 US Cities (2004)	0-day lag	1 hr max.	116 (36 - 195)	2.2 (0.7 - 3.6)	0.6% (0.2% - 0.9%)
Cleveland	Bell et al. (2004)	distributed lag	24 hr avg.	17 (-11 - 45)	1.2 (-0.8 - 3.2)	0.2% (-0.1% - 0.6%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	11 (4 - 18)	0.8 (0.3 - 1.3)	0.1% (0% - 0.2%)
Detroit	Bell et al. (2004)	distributed lag	24 hr avg.	19 (-6 - 44)	0.9 (-0.3 - 2.1)	0.2% (-0.1% - 0.5%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	10 (3 - 16)	0.5 (0.2 - 0.8)	0.1% (0% - 0.2%)
	Schwartz (2004)	0-day lag	1 hr max.	105 (-17 - 223)	5.1 (-0.8 - 10.8)	1.1% (-0.2% - 2.4%)
	Schwartz -- 14 US Cities (2004)	0-day lag	1 hr max.	57 (18 - 96)	2.8 (0.9 - 4.6)	0.6% (0.2% - 1%)
	Ito (2003)	0-day lag	24 hr avg.	23 (-22 - 67)	1.1 (-1.1 - 3.3)	0.2% (-0.2% - 0.7%)
Houston	Bell et al. (2004)	distributed lag	24 hr avg.	16 (1 - 30)	0.5 (0 - 0.9)	0.2% (0% - 0.3%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	8 (3 - 13)	0.2 (0.1 - 0.4)	0.1% (0% - 0.1%)
	Schwartz (2004)	0-day lag	1 hr max.	68 (6 - 129)	2 (0.2 - 3.8)	0.8% (0.1% - 1.4%)
	Schwartz -- 14 US Cities (2004)	0-day lag	1 hr max.	57 (18 - 96)	1.7 (0.5 - 2.8)	0.6% (0.2% - 1.1%)
Los Angeles	Bell et al. (2004)	distributed lag	24 hr avg.	29 (-71 - 127)	0.3 (-0.7 - 1.3)	0.1% (-0.3% - 0.5%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	63 (21 - 104)	0.7 (0.2 - 1.1)	0.2% (0.1% - 0.4%)
New York	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	36 (12 - 59)	0.4 (0.1 - 0.7)	0.1% (0% - 0.2%)
Philadelphia	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	16 (5 - 26)	1.1 (0.4 - 1.7)	0.2% (0.1% - 0.3%)
	Moolgavkar et al. (1995)	1-day lag	24 hr avg.	57 (36 - 78)	3.7 (2.4 - 5.1)	0.7% (0.4% - 1%)

Location	Study	Lag	Exposure Metric	Non-Accidental Mortality Associated with O_3 Concentrations that Just Meet the Current O_3 Standard**		
				Incidence	Incidence per 100,000 Relevant Population	Percent of Total Incidence
Sacramento	Bell et al. (2004)	distributed lag	24 hr avg.	8 (-24 - 39)	0.6 (-1.9 - 3.2)	0.2% (-0.6% - 0.9%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	12 (4 - 19)	0.9 (0.3 - 1.6)	0.3% (0.1% - 0.5%)
St Louis	Bell et al. (2004)	distributed lag	24 hr avg.	2 (-4 - 8)	0.6 (-1 - 2.3)	0.1% (-0.2% - 0.4%)
	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	2 (1 - 3)	0.6 (0.2 - 0.9)	0.1% (0% - 0.2%)
Washington, D.C.	Bell et al. -- 95 US Cities (2004)	distributed lag	24 hr avg.	7 (2 - 11)	1.2 (0.4 - 2)	0.3% (0.1% - 0.4%)

*All results are for mortality (among all ages) associated with short-term exposures to O3. All results are based on single-pollutant models.

**Incidence was quantified down to estimated policy relevant background levels. Incidences are rounded to the nearest whole number; incidences per 100,000 relevant population and percents are rounded to the nearest tenth.

Table 4-12. Estimated Cardiovascular and Respiratory Mortality Associated with O_3 Concentrations that Just Meet the Current 8-Hour Daily Maximum Standard: April - September*

Risk Assessment Location	Study Location	Cardiovascular and Respiratory Mortality Associated with O_3 Concentrations that Just Meet the Current O_3 Standard**		
		Incidence	Incidence per 100,000 Relevant Population	Percent of Total Incidence
Atlanta	Atlanta	5 (-2 - 12)	0.4 (-0.1 - 0.8)	0.6% (-0.2% - 1.3%)
	19 U.S. Cities	6 (2 - 9)	0.4 (0.1 - 0.6)	0.6% (0.2% - 0.9%)
Chicago	Chicago	13 (-11 - 36)	0.2 (-0.2 - 0.7)	0.2% (-0.2% - 0.7%)
	19 U.S. Cities	21 (8 - 33)	0.4 (0.1 - 0.6)	0.4% (0.2% - 0.6%)
Cleveland	Cleveland	10 (0 - 21)	0.7 (0 - 1.5)	0.6% (0% - 1.1%)
	19 U.S. Cities	9 (3 - 14)	0.6 (0.2 - 1)	0.5% (0.2% - 0.8%)
Detroit	Detroit	9 (-1 - 18)	0.4 (0 - 0.9)	0.4% (0% - 0.8%)
	19 U.S. Cities	8 (3 - 13)	0.4 (0.1 - 0.6)	0.3% (0.1% - 0.5%)
Houston	Houston	6 (-1 - 12)	0.2 (0 - 0.3)	0.3% (0% - 0.5%)
	19 U.S. Cities	6 (2 - 9)	0.2 (0.1 - 0.3)	0.3% (0.1% - 0.4%)
Los Angeles	Los Angeles	46 (0 - 91)	0.5 (0 - 1)	0.6% (0% - 1.2%)
	19 U.S. Cities	54 (21 - 86)	0.6 (0.2 - 0.9)	0.7% (0.3% - 1.2%)
New York	New York	43 (14 - 72)	0.5 (0.2 - 0.8)	0.5% (0.2% - 0.8%)
	19 U.S. Cities	32 (12 - 51)	0.4 (0.1 - 0.6)	0.4% (0.1% - 0.6%)
Philadelphia	Philadelphia	14 (1 - 27)	0.9 (0 - 1.8)	0.8% (0% - 1.5%)
	19 U.S. Cities	12 (4 - 19)	0.8 (0.3 - 1.2)	0.6% (0.2% - 1%)

*All results are for cardiovascular and respiratory mortality (among all ages) associated with short-term exposures to O3. Results are based on single-pollutant single-city models or a single-pollutant multi-city model estimated in Huang et al. (2004).

**Incidence was quantified down to estimated policy relevant background levels. Incidences are rounded to the nearest whole number; incidences per 100,000 relevant population and percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% credible intervals based on statistical uncertainty surrounding the O_3 coefficient.

Table 4-13. Estimated Health Risks Associated with O_3 Concentrations that Just Meet the Current 8-Hour Daily Maximum Standard: New York, NY, April - September

Health Effects*	Study	Ages	Lag	Exposure Metric	Other Pollutants in Model	Health Effects Associated with O_3 Concentrations that Just Meet the Current O_3 Standard**		
						Incidence	Incidence per 100,000 Relevant Population	Percent of Total Incidence
Mortality, non-accidental	Bell et al. -- 95 US Cities (2004)***	all	distributed lag	24 hr avg.	none	36 (12 - 59)	0.4 (0.1 - 0.7)	0.1% (0% - 0.2%)
Mortality, cardiovascular and respiratory	Huang et al. (2004)***	all	distributed lag	24 hr avg.	none	43 (14 - 72)	0.5 (0.2 - 0.8)	0.5% (0.2% - 0.8%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	none	32 (12 - 51)	0.4 (0.1 - 0.6)	0.4% (0.1% - 0.6%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	PM10	19 (-7 - 45)	0.2 (-0.1 - 0.5)	0.2% (-0.1% - 0.5%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	NO2	15 (3 - 28)	0.2 (0 - 0.3)	0.2% (0% - 0.3%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	SO2	13 (0 - 26)	0.1 (0 - 0.3)	0.1% (0% - 0.3%)
Mortality, cardiovascular and respiratory	Huang et al. -- 19 US Cities (2004)***	all	distributed lag	24 hr avg.	CO	18 (5 - 30)	0.2 (0.1 - 0.3)	0.2% (0.1% - 0.3%)
Hospital admissions (unscheduled), respiratory illness	Thurston et al. (1992)****	all	3-day lag	1 hr max.	none	364 (88 - 639)	4.5 (1.1 - 8)	1% (0.2% - 1.8%)
Hospital admissions (unscheduled), asthma	Thurston et al. (1992)****	all	1-day lag	1 hr max.	none	310 (66 - 555)	3.9 (0.8 - 6.9)	2.4% (0.5% - 4.2%)

*Health effects are associated with short-term exposures to O_3.

**Incidence was quantified down to estimated policy relevant background levels. Incidences are rounded to the nearest whole number; incidences per 100,000 relevant population and percents are rounded to the nearest tenth.

***New York in this study is defined as the five boroughs of New York City plus Westchester County.

****New York in this study is defined as the five boroughs of New York City.

Note: Numbers in parentheses are 95% confidence or credible intervals based on statistical uncertainty surrounding the O_3 coefficient.

The results in this portion of the risk assessment follow the same patterns as the results discussed in Section 4.2.1 for risks associated with "as is" O_3 concentrations, because they are largely driven by the same C-R function coefficient estimates and confidence or credible intervals.

Figures 4-7a and b show estimated percent of non-accidental mortality and cases per 100,000 relevant population related to O_3 concentrations that just meet the current 8-hour O_3 standard, based on single-pollutant, single-city models across all locations for which such models were available. Table 4-11 shows estimates of incidence, incidence per 100,000 relevant population, and percent of total incidence of non-accidental mortality related to O_3 concentrations that just meet the current 8-hour O_3 standard, based on both single-city and multi-city models. Estimates of O_3-related (non-accidental) mortality ranged from 0.3 per 100,000 relevant population in Atlanta (Bell et al., 2004), Houston (Bell et al., 2004 – 95 U.S. Cities), and Los Angeles (Bell et al., 2004) to 5.8 per 100,000 relevant population in Chicago (Schwartz, 2004). As was the case for the analysis of effects associated with "as is" O_3 concentrations, estimated O_3-related (non-accidental) mortality reported by Schwartz (2004) for Chicago, Detroit, and Houston, based on both the single-city and the multi-city C-R functions, tend to be higher than other estimates in those locations in large part because Schwartz used the 1-hr maximum O_3 concentration, rather than the 24-hour average, as the exposure metric. The changes from 1-hr maximum O_3 concentrations that just meet the current 8-hour O_3 standard to PRB 1-hr maximum O_3 concentrations were generally larger in the assessment locations than the corresponding changes using the 24-hr average metric. As a percent of total incidence, estimated non-accidental mortality related to O_3 concentrations that just meet the current 8-hour O_3 standard ranged from 0.1 percent in several locations (Atlanta, Chicago, Detroit, Houston, Los Angeles, New York, and St. Louis) to 1.5 percent in Chicago (Schwartz, 2004). Although 7 of the 12 estimates from single-city single-pollutant models shown in Figures 4-7a and b were not statistically significant, all 12 were positive.

Figures 4-8a and b show estimated percent of cardiovascular and respiratory mortality and cases per 100,000 relevant population related to O_3 concentrations that just meet the current 8-hour O_3 standard, based on multi-city single-pollutant versus multi-pollutant models from Huang et al. (2004) across all locations for which such models were available. Table 4-12 shows estimates of incidence, incidence per 100,000 relevant population, and percent of total incidence of cardiovascular and respiratory mortality related to O_3 concentrations that just meet the current 8-hour O_3 standard in all risk assessment locations covered in Huang et al. (2004), based on both single-city and multi-city models from that study. Estimates of O_3-related cardiovascular and respiratory mortality ranged from 0.2 per 100,000 relevant population in Houston (using both the single-city and the multi-city C-R functions) to 1.0 per 100,000 relevant population in Philadelphia (using the single-city C-R function). As a percent of total incidence, estimated O_3-related cardiovascular and respiratory mortality ranged from 0.3 percent in Chicago (using the single-city C-R function) to 0.8 percent in Los Angeles (using the multi-city C-R function) and Philadelphia (using the single-city C-R function). All of the estimates of O_3-related cardiovascular and respiratory mortality based on Huang et al. (2004), from both single-pollutant and multi-pollutant models (see Figures 9a and b) and from both single-city and multi-city models (see Table 4-12) were positive. With the exceptions of the estimates from the single-city single-pollutant model for Chicago and the multi-city multi-pollutant model with PM_{10}, all of the

estimates of cardiovascular and respiratory mortality related to O_3 concentrations that just meet the current 8-hour O_3 standard, based on Huang et al. (2004), were statistically significant.

Figures 4-9a and b show estimated percent of non-accidental mortality and cases per 100,000 relevant population related to O_3 concentrations that just meet the current 8-hour O_3 standard, based on single-city versus multi-city models across all locations for which both types of model were available. The results followed the same patterns as were observed in the analysis of effects associated with "as is" O_3 concentrations above PRB levels, discussed in Section 4.2.1 above (see also Figures 4-4a and b). Similarly, the results seen in Figures 4-10a and b, for cardiovascular and respiratory mortality, followed the same patterns as are evident in the corresponding analysis of "as is" O_3 concentrations (see Figures 4-6a and b).

The affect of O_3 lag structure on O_3-related unscheduled hospital admissions in Detroit (Ito 2003), shown in Figures 4-12a and b, followed the same patterns as were evident in the analysis of risks associated with "as is" O_3 concentrations. Estimated pneumonia hospital admissions associated with O_3 concentrations that just meet the current 8-hour O_3 standard increased monotonically with increasing lag, with the greatest estimate predicted by a 3-day lag model. A 3-day lag model also predicted the greatest number of O_3-related ischemic heart disease hospital admissions. With the exception of the 0-day lag, O_3-related heart failure hospital admissions decreased monotonically with increasing lags. None of the estimates of O_3-related unscheduled hospital admissions in Detroit were statistically significant.

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