Out of Breath

Health Effects from Ozone in the Eastern United States

Report

Clear the Air

National Campaign Against Dirty Power

October 1999

Clear the Air: National Campaign Against Dirty Power is a joint project of the Clean Air Task Force, National Environmental Trust, and the U.S. PIRG Education Fund. Clear the Air works with grassroots organizations throughout the country. Its mission is to safeguard public health and the environment by reducing power plant air pollution.

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Adverse Health Effects Associated with Ozone In the Eastern United States

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Abt Associates' Environmental Research Area provides multi-disciplinary scientific research and environmental policy analysis to the EPA, the U.S. Agency for International Development, the Inter-American Development Bank, the World Bank, and directly to foreign, state and local governments. Abt Associates has extensive experience in estimating the potential public health improvements and economic costs and benefits from improving ambient air quality. The Environmental Research Area conducted extensive health analysis for the U.S. EPA in support of the 1997 revisions to both the ozone and the particulate matter National Ambient Air Quality Standards. They also prepared the health and economic analyses for EPA's 1997 Report to Congress *The Benefits and Costs of the Clean Air Act: 1970 to 1990*, and conducted similar policy, health and economic analyses for EPA of regulations on the electric generating industry, automobile exhaust, and potential policies for climate change mitigation strategies. Abt Associate's Environmental Research Area conducts public health analysis projects worldwide, including air pollution health assessment projects with the environmental and health ministries in Argentina, Brazil, Canada, Chile, Korea, Russia, Thailand, the Ukraine and the World Health Organization.

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1. INTRODUCTION

This report describes the methods and results of an analysis estimating adverse human health effects due to ground-level exposures to ozone. In particular, the analysis estimates the incidence of hospital admissions attributable to ozone exposures in the 37 eastern states and the District of Columbia, that form the "OTAG" region.[1]

Ozone is a strong oxidant that inflames the lungs, alters their mechanical functions, and reduces their ability to expel foreign material. It can result in decreased lung capacity, and relatively minor symptoms, such as cough and pain on deep inspiration. In addition, it may cause severe respiratory-related adverse effects, such as asthma attacks, hospital admissions, emergency room (ER) visits, and possibly even premature mortality. Long-term ozone exposure appears to be associated with the development of chronic asthma, and perhaps other chronic health problems.[2]

Chapter 2 describes the methods for estimating ambient ozone concentrations, and Chapter 3 describes the methods used to estimate the adverse health effects associated with exposures to ambient concentrations of ozone. Chapter 4 presents results for the OTAG region as well as for 34 metropolitan areas within the OTAG region (Exhibit 1-1).[3]

Exhibit 1-1 Thirty-four Metropolitan Areas in the OTAG Region Considered in this Analysis

Atlanta	Dayton	Milwaukee	Raleigh/Durham
Baltimore	Detroit	Minneapolis/St. Paul	Richmond
Birmingham	Evansville, IN	Mobile	Savannah
Charleston	Grand Rapids	Montgomery	St. Louis
Charlotte	Hartford	Muskegon, MI	Tampa/St. Petersburg
Chicago	Indianapolis	Nashville	Toledo
Cincinnati	Kalamazoo, MI	New York	Washington
Cleveland	Memphis	Philadelphia	
Columbia	Miami/Ft. Lauderdale	Pittsburgh	

[1]The Ozone Transport Assessment Group (OTAG) was a partnership between the U.S. EPA, the Environmental Council of the States (ECOS) and various industry and environmental groups, that focused on ground-level ozone and the pollutants that cause ground-level ozone. OTAG explicitly addressed ozone transport over the Eastern United States. (For more background on OTAG, see: http://www.epa.gov/ttn/rto/otag/aboutotg.html.)

[2]The health impacts of ozone are summarized in EPA (1996a).

[3]Appendix A presents a list of the counties comprising each area and a map of the OTAG region with these areas highlighted.

2. OZONE AIR QUALITY ESTIMATION

This chapter describes the methods used to model ozone concentrations, from which reductions in health effect incidences are then estimated. Section 1 briefly describes ozone formation. Section 2 describes the available ozone monitoring data, and Section 3 discusses the extrapolation of these data to areas without ambient air quality monitors.

2.1 OZONE FORMATION

Ozone forms as the result of complex atmospheric and chemical processes involving volatile organic compounds (VOCs) and nitrogen oxides (NO_x). This highly nonlinear process is moderated by factors including temperature, sunlight, atmospheric mixing, the concentrations of ozone precursors and the ratio between VOC and NO_x, as well as the reactivity of the VOCs (U.S. EPA, 1996a, p. 3-1).

The basic process for the formation of ozone in the troposphere, the lowest layer of the atmosphere, starts with the photolysis of nitrogen dioxide (NO_2) to nitric oxide (NO) and an oxygen atom:[4]

$$NO_2 + light \rightarrow NO + O \ .$$

The oxygen atom then reacts with molecular oxygen (O_2) to form ozone (O_3):

$$O + O_2 + M \rightarrow O_3 + M \ , where \ M = air \ .$$

The ozone may be scavenged by NO:

$$NO + O_3 \rightarrow NO_2 + O_2 \ ,$$

however, reactive VOCs can mediate the conversion of NO to NO_2 without O_3, thus allowing O_3 levels to build:

$$NO \xrightarrow{VOC} NO_2 \ .$$

Ozone levels tend to peak in the early afternoon, and gradually decline to almost undetectable levels around dawn. Some variation in this diurnal pattern of ozone levels can be expected between different areas, depending on the balance among the many factors affecting ozone formation, transport, and destruction (U.S. EPA, 1996a, p. 4-46).

Anthropogenic emissions of ozone precursors cause the bulk of the ozone found in the troposphere. However, a certain amount of ozone forms naturally. Ozone forms in the stratosphere from the photolysis of oxygen (O_2) and is an important protective shield from ultraviolet radiation.[5] Some of this stratospheric ozone

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[4]The following description of the formation of ozone is based on EPA (1996a, p. 3-2).

[5]EPA (1996a, p. 3-4) provides more detail on the formation of ozone from the photolysis of O_2, the interactions of O_3 with NO, derived from the photolysis of nitrous oxide (N_2O) to NO, as well as the impact of chlorine and bromine compounds on stratospheric O_3 levels.

is transported downward into the troposphere, and contributes about 5 to 15 parts per billion (ppb) to ambient concentrations (U.S. EPA, 1996a, p. 4-28). A certain amount of ozone also forms in the troposphere itself, from natural emission sources. Methane from swamps, wetlands, and ruminants, non-methane VOCs from plants, and NO_x emitted from soils, lightning strikes, and NO transported from the stratosphere into the troposphere, all can contribute to tropospheric ozone formation (U.S. EPA, 1996a, p. 4-14).

2.2 OZONE MONITORING DATA

For this study, ozone concentrations were considered only in the 37 eastern states plus the District of Columbia (known as the OTAG region) for the period April through October, 1997. Hourly ozone concentrations for 1997 were extracted from the Aerometric Information Retrieval System (AIRS) and input into a single AMP350-format datafile. For the ozone data, a monitor record was considered to be complete if data were available for 50 percent of days in a given season (April 1-October 31 in this analysis). A monitor day was considered valid if 75% or more of the hours between 9am and 8:59pm were available.[6] There were 698 unique ozone monitoring locations found in AIRS, of which 687 passed the completeness criteria (Exhibit 2-2).

2.3 MODELING AMBIENT OZONE

For this analysis, we model ambient ozone levels in 1997 using the Criteria Air Pollutant Modeling System (CAPMS), a population-based system for modeling exposures to criteria air pollutants. CAPMS has been used extensively to estimate air pollution control benefits in the United States. As a first step in the modeling process, CAPMS divides the OTAG region into eight kilometer by eight kilometer grid cells, and then estimates air quality for each cell.

2.3.1 Interpolation of Air Quality Monitoring Data to CAPMS Grid Cell Centers

Modeling air quality throughout the U.S. has some problems, since air quality data are only available from limited monitor sites. We needed a method to extrapolate to unmonitored locations, in order to estimate the impact of air pollution on the health and welfare effects considered in this analysis.[7] Given the available air monitoring data, we extrapolated from all available monitor locations to a grid of eight km by eight km population grid-cells throughout the OTAG region, using a Voronoi Neighbor Averaging (VNA) spatial interpolation procedure.[8]

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[6]The choice of using at least 75% of the hours between 9:00am and 8:59pm is consistent with the criterion defined in the Code of Federal Regulations (U.S. Office of the Federal Register, 1995, Part 50 Appendix H).

[7]Appendix A has a map of the location of ozone monitors in the U.S. The map shows that some areas of the country do not have many ozone monitors in close proximity to each other.

[8]Interpolation between monitors is conducted using the same method as used by Abt Associates (1998) for the NO_x SIP call analysis; previously termed the "convex polygon" method, it is more accurately described as Voronoi Neighbor Averaging (VNA) spatial interpolation, which will be used throughout this document.

The VNA procedure interpolates air quality estimates from air quality monitors to the center of each population grid-cell. The VNA procedure is a generalization of planar interpolation. Rather than limit the selection of monitors to, say, three, VNA identifies the set of monitors that best "surrounds" the center of each grid-cell. The result of VNA is illustrated in Exhibit 2-1. VNA determines the set of monitors that best surround the grid-cell by identifying which monitor is closest (considering both angular direction and horizontal distance) in each direction from the grid-cell center. Each selected monitor will likely be the closest monitor for multiple directions. The set of monitors found using this approach forms a polygon around the grid-cell center.

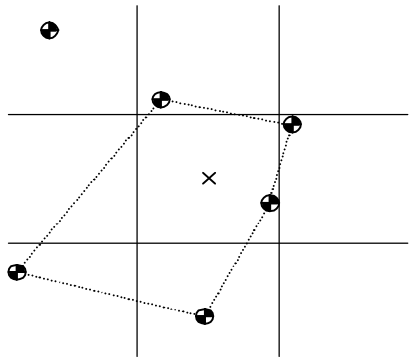

Exhibit 2-1 VNA Spatial Interpolation

For each grid cell, CAPMS calculates the distance to each of a set of monitors surrounding that grid cell. Monitors close to the grid cell are assumed to yield a more accurate air quality description of that grid cell, and are given a larger weight when calculating the average air quality for that grid cell. Conversely, monitors that are further away contribute less to the average. After determining the final set of surrounding monitors, the grid cell's air quality level is calculated as an inverse, distance-weighted average of the air quality levels at the selected monitors.

Note that the air quality inputs to CAPMS must be in the form (averaging time) required by the concentration-response (C-R) functions being used. For example, a C-R function relating respiratory hospital admissions to daily maximum ozone concentrations requires that daily maximum ozone concentrations be available at CAPMS grid cell centers. Although the input ozone data must be in the form of daily maxima, the input data need not be at CAPMS grid cell centers. Given any set of location-specific air quality data, CAPMS interpolates the corresponding air quality values at each CAPMS grid cell center. To reduce computational effort, CAPMS typically "bins" air quality data being used to calculate air levels, and bins the resulting air quality at CAPMS grid cell centers.

2.3.2 Binning Input Air Quality Data

To reduce computational time and effort when estimating the change in health effects associated with daily pollution levels, CAPMS approximates a season's (or year's) worth of daily pollutant concentrations at each input location (i.e., location for which air quality data are available) by n "bins" of pollutant concentrations. For the ozone analysis n = 20, so that each bin represents five percent of the daily pollutant concentrations in the ozone season, April through October in this analysis. Each bin is set at the midpoint of the percentile range it represents. For n = 20 and a season's worth of ozone observations, the first bin represents the first (lowest) five percent of the distribution of 214 pollutant concentrations at the given location, and is set at the 2.5th percentile value; the second bin represents the next five percent of the distribution of daily values, and is set at the 7.5th percentile value, and so on. Each of the twenty bins therefore represents 10.7 (=214/20) days. Interpolation of air quality levels at CAPMS grid cell centers is based on these input location-specific bins, so that the seasonal incidence changes in each grid cell are calculated for twenty pollutant concentrations (the 20 bins of air quality) rather than for 214 pollutant concentrations. The resulting incidence change is then multiplied by 10.7 to reconstruct an entire season's worth of incidence change in the CAPMS grid cell.

Exhibit 2-2
Ozone Monitor Locations in the OTAG Region in 1997

3. CALCULATING OZONE-RELATED ADVERSE HEALTH EFFECTS

This chapter describes the methods for estimating the adverse health effects associated with ambient ozone concentrations present throughout the U.S. Section 1 discusses the CAPMS approach to calculating point estimates of health effects. Section 2 discusses issues affecting the estimation of health effects. Section 3 discusses the epidemiological studies used to estimate incidence estimates for each of the health effects. Section 4 discusses the uncertainty in the estimation of air pollution-related health effects.

3.1 CAPMS APPROACH TO CALCULATING ADVERSE HEALTH EFFECTS

In each eight kilometer by eight kilometer grid cell in the OTAG region, CAPMS estimates the incidence of adverse health associated with the presence of air pollutants in each grid cell. The regional incidence estimate (or the estimate within an individual state or county) is then calculated as the sum of grid-cell-specific incidence estimates.

3.1.1 Calculation of Point Estimates of Incidence Changes

For this analysis, CAPMS estimates the adverse health effects attributable to levels of 1997 ambient ozone. To make this estimation, CAPMS interpolates the air quality at the CAPMS grid cell center. Since the daily values have been binned at the monitors, the resulting air quality data at the CAPMS grid cell center are also binned. It then accesses the selected C-R functions being used, the required baseline incidence rates, and the grid cell population. CAPMS then calculates the incidence associated with 1997 ozone levels for each adverse health effect for which a C-R function has been accessed. For example, if the functional form of the C-R relationship is log-linear (the most commonly used form), the relationship between a change in pollutant level,[9] e.g., ΔO_3, and the change in incidence of the health effect, Δy, is:

$$\Delta y = population \cdot baseline\,incidence\,rate \cdot [e^{b \cdot \Delta O3} - 1] ,$$

where β is the coefficient of O_3 in the C-R function. The changes in incidence corresponding to the changes in air quality in each of the 20 bins of pollutant concentrations are summed and multiplied by 10.7 (the number of days per bin) to produce a seasonal ozone-related incidence estimate in that grid cell.

The resulting seasonal incidence estimate is stored, and CAPMS proceeds to the next grid cell, where the above process is repeated. Total ozone-related incidence (or the incidence that occurs in any designated geographical area) is calculated at the end of the process by summing the grid cell-specific estimates.

3.1.2 Calculation of Distributions of Incidence Estimates

The C-R functions used in this analysis are simply estimates of the relationship between ozone and adverse health effects. To reflect the uncertainty surrounding the pollutant coefficients in the C-R functions used, CAPMS can produce a *distribution* of possible incidence estimates attributed to ozone for each adverse

[9]The change in this case is simply equal to the 1997 ozone level, since the effect of all ozone is estimated.

health effect.

To produce this distribution, CAPMS uses both the estimate of the pollutant coefficient (β) and the standard error of the coefficient estimate to produce a normal distribution with mean equal to the estimate of β and standard deviation equal to the standard error of the estimate. CAPMS takes the n^{th} percentile value of β from this normal distribution -- for n = 0.5, 1.5, ..., 99.5 – and produces an estimate of the incidence, given that value of β. The resulting distribution of values for each CAPMS grid cell is then stored, and CAPMS proceeds to the next grid cell, where the process is repeated. A distribution of the region-wide incidence is calculated by summing the n^{th} percentile grid cell-specific estimates, for n = 0.5, 1.5, ..., 99.5.

3.2 CONCENTRATION-RESPONSE FUNCTIONS

A number of issues arise in choosing health effects studies to use as the basis for concentration-response (C-R) functions, and in choosing the parameters that are included in a given C-R function.

3.2.1 Chamber Versus Epidemiological Studies

The available human health studies that could be used to estimate the impact of ozone on human health can be categorized into chamber studies and epidemiology studies. Chamber studies involve examination of human responses to controlled conditions in a laboratory setting, while epidemiological studies investigate the association between exposure to ambient air pollution and observed health effects in a study population. This analysis relies on epidemiological studies, rather than chamber studies, to develop C-R functions.

Chamber studies of air pollution involve exposing human subjects to various levels of ozone in a carefully controlled and monitored laboratory situation. One advantage of chamber studies is that they can potentially establish cause-effect relationships between ozone and certain human health effects. However, health effects measured in some well-designed chamber studies are selected on the basis of the ability to precisely measure an effect, for example forced expiratory volume, rather than a larger symptom. Some of these measurable but relatively minor health effects, such as reduced lung function, have an unclear impact on future medical condition and lifestyle. Ethical considerations preclude exposing people for extended periods of time, and limit experimental ozone concentrations to relatively modest exposure levels. This confines studies to examining only mild health effects that are believed to do no permanent damage, and prevents studying the relationship between ozone and chronic conditions, hospital admissions, and mortality.

Epidemiological studies present the results of a statistical analysis of the relationship between ambient ozone exposure and adverse health effects, unlike chamber studies, which often monitor endpoints that do not result in observable health effects (e.g. forced expiratory volume). The data for epidemiological studies includes ambient air quality monitoring data and adverse health effects data such as mortality incidence (e.g., National Center for Health Statistics, 1994), hospital admissions (e.g., Graves and Gillum, 1997), questionnaires (e.g., Adams and Marano, 1995), and diaries that are kept by study participants over a period of time (e.g., Ostro et al., 1991). Epidemiological studies typically involve a large number of people and may not suffer as much from the extrapolation problems common to chamber studies, which often have a limited number of subjects. Perhaps most importantly, they allow the study of relatively serious adverse health effects.

3.2.2 Criteria for Choosing Epidemiological Studies

We use a number of criteria in choosing studies to estimate C-R functions and to estimate the impact of ozone on adverse health effects. These criteria include:

(1) Peer review. Whenever possible, peer-reviewed research rather than unpublished information has been used.

(2) Representative populations. Studies that are representative of the general population are more desirable than studies focusing on a narrow subpopulation, because they allow application of the C-R functions to larger numbers of persons without introducing additional uncertainty.

(3) Long-term studies. Studies examining a relatively longer period of time (and therefore having more data) are more desirable, because they have greater statistical power to detect effects. More recent studies are also more desirable because of possible changes in pollution mixes, medical care, and life style over time.

(4) Multiple pollutants. In many cases, several pollutants in a "pollutant mix" are correlated with each other -- that is, their concentrations tend to change together. Although there may be an association between an adverse health effect and this mix, it may not be clear which pollutant is causally related to the health effect -- or whether more than one pollutant is causally related. Using separate regressions (from single pollutant models) for each pollutant may overstate the effect of each pollutant alone. Models that consider pollutants simultaneously are therefore preferred.[10]

(5) A measure of particulate matter. Because PM has been acknowledged to be an important pollutant, models which include some measure of PM are highly preferred to those which do not.

(6) North American studies. Studies of U.S. and Canadian populations are generally preferred to foreign studies due to potential differences in factors such as activity patterns and medical care.

3.2.3 Basic Concentration-Response Model

The methods discussed in this sub-section apply to the estimation of ozone's impact on adverse health effects. For expository simplicity, the discussion below refers only to a generic "health endpoint," denoted as y, although several endpoints are associated with ozone. Additionally, the discussion refers to the estimation of incidence for a health endpoint at a single location (population cell). Region-wide incidence totals are estimated by summing ozone-related incidence estimates over all population cells in the region.

Corresponding to a change in ozone, ΔO_3, in a given population cell is an associated change in the health endpoint, Δy, in that population cell. Given a C-R function estimated by a study, and a particular change in ozone, ΔO_3, the corresponding change in the health endpoint, Δy, corresponding to the particular ΔO_3 can be calculated. This change in the health endpoint, Δy, can be thought of as the health effect incidence that is attributable to ozone exposures in a given area.

When using a particular epidemiological study to develop a C-R function and estimate the effect of ambient ozone levels, it is important to follow the study design as much as possible. Epidemiological studies

[10] Models containing several correlated pollutants are not without problems, however. High correlations among variables in a model result in coefficient estimates with large variances.

can differ in a number of ways. Some studies that measure the relationship between ozone and hospital admissions related to respiratory illnesses may include all categories of respiratory health effects, while others may exclude certain symptoms from the study. One study may have measured daily (24-hour) average ozone concentrations while another study may have used two-day averages. Some studies have assumed that the relationship between y and ozone is best described by a linear form (i.e., the relationship between y and ozone is estimated by a linear regression in which y is the dependent variable and ozone is one of several independent variables). Other studies have assumed that the relationship is best described by a log-linear form (i.e., the relationship between the natural logarithm of y and ozone is estimated by a linear regression).[11] Finally, one study may have considered changes in the health endpoint only among members of a particular subgroup of the population (e.g., individuals 65 and older), while other studies may have considered the entire population in the study location.

Estimating the relationship between ozone and a health endpoint, y, consists of (1) choosing a functional form of the relationship and (2) estimating the values of the parameters in the function assumed. The two most common functional forms in the epidemiological literature on ozone and health effects are the log-linear and the linear relationship. The log-linear relationship is of the form:

$$y = Be^{b \cdot O_3} \ ,$$

or, equivalently,

$$\ln(y) = a + b \cdot O_3$$

where the parameter B is the incidence of y when the concentration of O_3 is zero, the parameter β is the coefficient of O_3, ln(y) is the natural logarithm of y, and $\alpha = \ln(B)$.[12] If the functional form of the C-R relationship is log-linear, the relationship between ΔO_3 and Δy is:

$$\Delta y = y \cdot \left(e^{b \cdot \Delta O_3} - 1\right) \ , \qquad \textbf{(1)}$$

where y is the baseline incidence of the health effect (i.e., the incidence before the change in O_3). For a log-linear C-R function, the relative risk (RR) associated with the change ΔO_3 is:

$$RR_{\Delta O_3} = e^{\beta \cdot \Delta O_3} \ .$$

Epidemiological studies often report a relative risk for a given ΔO_3, rather than the coefficient, β, in the C-R function. The coefficient can be derived from the reported relative risk and ΔO_3, however, by solving for β:

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[11]The log-linear form used in the epidemiological literature on ozone-related health effects is often referred to as "Poisson regression" because the underlying dependent variable is a count (e.g., number of deaths), believed to be Poisson distributed. The model may be estimated by regression techniques but is often estimated by maximum likelihood techniques. The form of the model, however, is still log-linear.

[12] Other covariates besides pollution clearly affect mortality. The parameter B might be thought of as containing these other covariates, for example, evaluated at their means. That is, $B = B_o \exp\{\beta_1 x_1 + ... + \beta_n x_n\}$, where B_o is the incidence of y when all covariates in the model are zero, and $x_1, ... , x_n$ are the other covariates evaluated at their mean values. The parameter B drops out of the model, however, when changes in y are calculated (see equation (7)) and is therefore not important.

$$b = \frac{\ln(RR)}{\Delta O_3} \ .$$

The linear relationship is of the form:

$$y = a + b \cdot O_3 \ ,$$

where α incorporates all the other independent variables in the regression (evaluated at their mean values, for example) times their respective coefficients. When the C-R function is linear, the relationship between a relative risk and the coefficient, β, is not quite as straightforward as it is when the function is log-linear. Studies using linear functions usually report the coefficient directly.

If the functional form of the C-R relationship is linear, the relationship between ΔO_3 and Δy is simply:

$$\Delta y = b \cdot \Delta O_3 \ . \qquad \textbf{(2)}$$

For a given O_3 change, ΔO_3 (using a measure consistent with the O_3 measure used in the C-R function -- e.g., daily average O_3), and a value for the O_3 coefficient, β, the corresponding change in the health endpoint, Δy, is estimated -- using equation (1), if the C-R function is log-linear, or equation (2), if the C-R function is linear.

A few epidemiological studies, estimating the relationship between certain morbidity endpoints and O_3, have used functional forms other than linear or log-linear forms. Of these, logistic regressions are the most common. Abt Associates (1999) provides further details on the derivation of dose-response functions.

3.2.4 Thresholds

When conducting chamber and epidemiological studies, C-R functions may be estimated with and without explicit thresholds. Air pollution levels below the threshold are assumed to have no associated adverse health effects. When a threshold is not assumed, as is often the case in epidemiological work, any exposure level theoretically poses a non-zero risk of response to at least one segment of the population.

Thresholds may also be incorporated by a policy analyst using a C-R function derived from the original study. A threshold may be set at any point, although some points may be considered more obvious candidates than others, such as the non-anthropogenic background level of ozone, the lowest observed level in the study that estimated the C-R function, or the ozone standard. However, since there appears to be little evidence pointing to a particular threshold level, this analysis estimates ozone-related effects down to zero ambient ozone levels.

3.2.5 Baseline Incidences

Most of the C-R functions used in this analysis are log-linear, and the estimation of incidence changes based on a log-linear C-R function requires a baseline incidence. The baseline incidence rate is the fraction of a given population that incurs an adverse health effect in a given time period. For example, in 1994, people ages 65 and older had a daily incidence rate for respiratory-related hospital admissions of 1.187 E-4; in other words, out of a population of one million individuals, about 119 would be admitted to the hospital for a

respiratory condition on any given day.[13] Unfortunately, incidence rates are not available for each CAPMS grid-cell, so national rates are used, or in some cases the rate found in the (epidemiological) study population was used to develop an incidence rate.

3.2.6 Population

Many epidemiological studies focus on a particular age cohort. The age group chosen is often a matter of convenience (e.g., extensive Medicare data may be available for the elderly population) and not because the effects are necessarily restricted to the specific age group. To avoid overestimating the benefits of reduced pollution levels, this analysis applies the given C-R relationships only to those age groups corresponding to the cohorts studied.

Some people are especially sensitive to ozone, and suffer effects of a different kind or to a different degree than does the rest of the population. Factors that result in a higher risk include: (1) a person's biological response to ozone, (2) pre-existing lung disease, (3) activity patterns, (4) personal exposure, and (5) personal factors, such as age and nutritional status.[14] Asthmatics and others with pre-existing respiratory conditions are examples of potentially sensitive populations. There are a limited number of epidemiological studies, so it is not possible to capture all of the ozone-related effects for all groups of interest. In this analysis, we have C-R functions that cover asthmatics, children, adults, the elderly, and persons of all ages.

There is little doubt that ozone affects children's health, however, the type of the effect is uncertain. Studies by Avol et al (1985), Portney and Mullahy (1986), and Krupnick et al. (1990) found children did not develop the ozone-related respiratory symptoms suffered by adults. However, in a comprehensive review of the health effects literature, EPA (1996b, p. 9-36) concluded that: “Human studies have identified a decrease in pulmonary function responsiveness to ozone with increasing age, although symptom rates remain similar.” So it appears that children may indeed be sensitive to ozone, however we have a limited number of studies to estimate this effect.

Finally, some of our C-R functions are derived from epidemiological studies of relatively small populations. Nevertheless, we apply these functions to all areas in the OTAG region. Our results may be inaccurate, to the extent that the OTAG region differs from the study population used as the basis for the C-R function.

3.2.7 Pooling Study Results

When only a single study has estimated the C-R relationship between a pollutant and a given health endpoint, the estimation of a population cell-specific incidence change is straightforward. For some endpoints, however, C-R functions have been estimated by several studies, often in several locations. In this case, a pooled, “central tendency” C-R function can be derived from the multiple study-specific C-R functions in

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[13]All respiratory hospital admissions are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

[14]Populations at risk are discussed by EPA (1996b, p. 9-35).

several ways.

One potential method of pooled analysis is simply averaging the coefficients from all the studies. This has the advantage of simplicity, but the disadvantage of not taking into account the measured uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty.

An alternative approach to pooling studies is to apply subjective weights to the studies. If the analyst is aware of specific strengths and weaknesses of the studies involved, this prior information may be used as input to the calculation of weights which reflect the relative reliability of the estimates from the studies.

A third approach to pooling the estimates from different studies – and the one primarily used in this analysis – is to give more weight to estimates with little reported uncertainty and less weight to estimates with relatively more uncertainty. The exact way in which weights are assigned to estimates of ozone coefficients from different studies in a pooled analysis depends on the underlying assumption about how the different estimates are related to each other. If, for example, there is actually a distribution of true ozone coefficients, or β's, that differ by location (referred to as the "random effects" model), the different coefficients reported by different studies may be estimates of *different* underlying coefficients, rather than just different estimates of the same coefficient. In contrast to what one might call a "fixed effects" model (which assumes that there is only one β everywhere), the random-effects model allows the possibility that different studies are estimating different parameters.

In deciding whether to use a fixed or random effects pooling estimate, we use the fixed effects approach as our null hypothesis, and then determine whether the data suggest that we should reject this null hypothesis. The test statistic is asymptotically chi square distributed; if the test statistic exceeds a value that would occur no more than 5 percent of the time, then a random effects pooling estimate is used. Further details on how studies are pooled are given below; in addition, Abt Associates (1999, p. A-48) discusses general issues involved with pooling in more detail.

In some cases, studies reported several estimates of the C-R coefficient, each corresponding to a particular year or particular study area. For example, Ostro and Rothschild (1989) reported six separate regression coefficients that correspond to regression models run for six separate years. This analysis combined the individual estimates using a meta-analysis on the six years of data.

3.3 OZONE-RELATED ADVERSE HEALTH EFFECTS

Health effects studies have found that ozone is associated with a variety of adverse health outcomes, ranging from relatively minor symptoms, to hospital admissions, chronic illness, and even premature mortality. Not all studies have found ozone linked to these effects. In particular, there is a significant amount of uncertainty about the relationships between ozone and both mortality and chronic illness. In this section, we discuss the studies we use to estimate ozone-related adverse health effects. In addition, we briefly discuss the ozone-mortality and ozone-chronic illness relationships (although we do not estimate them). Exhibit 3-1 summarizes the effects that we estimate, and Appendix C presents the C-R functions that are used in this analysis.

Exhibit 3-1 Ozone-related Adverse Health Effects Included in the Analysis

Adverse Health Effect	Comment
Respiratory hospital admissions	A large number of studies have linked ozone to hospital admissions for pneumonia, chronic obstructive pulmonary disease (COPD), asthma, and other ailments.
Cardiovascular hospital admissions	We estimate the link between ozone and dysrhythmias (or abnormal heartbeat patterns).
Total respiratory ER visits	We estimate ER visits for pneumonia, chronic obstructive pulmonary disease (COPD), asthma, and other respiratory ailments.
Asthma ER visits	A number of studies have reported that ozone adversely affects asthmatics. We estimate the link between ozone and emergency room (ER) visits for asthma.
Minor symptoms	Short-term exposure to ozone has been linked to a variety of symptoms, including cough, sore throat, and head cold. We estimate ozone-related minor symptoms in adults.
Asthma attacks	We estimate the number of days that juvenile and adult asthmatics suffered one or more asthma attacks
Shortness of breath	We estimate the number of days with shortness of breath in African-American asthmatics ages 7-12.

3.3.1 Hospital Admissions

The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking ozone with other types of hospital admissions; most of the evidence supports the link between ozone and respiratory admissions. In addition to hospital admissions, we consider separately the association between ozone and ER visits. Hospital admissions require the patient to be examined by a physician, and on average may represent more serious incidents than ER visits (Lipfert, 1993, p. 230).

Respiratory Hospital Admissions

To estimate the number of hospital admissions for respiratory illness, we pool the incidence estimates from a variety of U.S. and Canadian studies, using a random-effects weighting procedure. These studies may differ in two important ways: (1) some studies considered people of all ages while others considered only people ages 65 and older; and (2) the types of effects included in studies of respiratory hospital admissions and air pollution may vary substantially.

The epidemiological studies used in this analysis used version 9 of the International Classification of Diseases (ICD-9).[15] The broadest classification of respiratory admissions includes ICD-9 codes 460-519.

[15]The International Classification of Diseases is often used to define health outcomes in epidemiological studies. The Center for Disease Control provides detailed information:

Other studies, however, considered only subsets of the broader classification. For example, Burnett et al. (1997) consider ICD-9 codes 466, 480-486, 490-494, and 496. If the broadest category (460-519) is too broad, including respiratory illnesses that are not linked to ozone, we would expect the estimated pollutant coefficients to be biased downward; however, they would be used in combination with a larger baseline incidence in estimating changes in respiratory hospital admissions associated with changes in pollutant concentrations. If the broadest category is correct (i.e., if all the respiratory illnesses included are actually associated with ozone), then studies using only subsets of ICD-9 codes within that category would presumably understate the change in respiratory hospital admissions. It is likely, however, that all the studies have included the most important respiratory illnesses, and that the impact of differences in the definition of "all respiratory illnesses" may be less than that of other study design characteristics. We therefore treat each study equally, at least initially, in the pooling process, assuming that each study gives a reasonable estimate of the impact of air pollution on respiratory hospital admissions.

There are several steps in our estimation process:

(1) Develop the C-R function for ozone in a model from a given study. Note that a study may include multiple pollutants in the final model. We generally prefer this, especially if the model includes a measure of particulate matter, which has been found to be related to a number of adverse effects. For example, Burnett et al. (1997) included $PM_{10-2.5}$, O_3, NO_2, and SO_2 in their final model for respiratory admissions[16]; we just use the O_3 coefficient from this model to develop a C-R function.

(2) If a study estimated separate models for non-overlapping respiratory illness categories, we sum the estimated incidence changes across these non-overlapping categories. For example, Burnett et al. (1999) estimated separate models for asthma admissions (ICD-9 code 493) and respiratory infection admissions (ICD-9 codes 464, 466, 480-487, 494); we calculated the ozone-related incidences for these two categories, and then summed the two estimates.

(3) We then pooled studies from the same geographic location, using an inverse-variance weighted average. Three studies based on data from Toronto and two studies based on data from Minneapolis were pooled in this fashion, and these two estimates were then combined with the estimates from the studies in other locations (e.g., Buffalo, NY). We used a random effects pooling procedure for this final step.

Exhibit 3-2 summarizes the studies used in estimating respiratory admissions. Since two of the 13 studies did not include ozone in the final model, this raises the question of whether these studies should be included anyway, with the mean ozone estimate for these studies then set to zero. However, we would also need an estimated standard error, to include these in our pooled estimate, since the pooling weights are based on the estimated standard error. We considered several approaches. One approach is to use a simple average: 11 studies (out of 13) would be used to develop a pooled ozone estimate; the pooled estimate could then be given a weight of 11/13, with the remaining 2/13 weight applied to a zero estimate. Alternatively, we could simply drop those studies that do not include ozone, and base the pooling on 11 studies. We used the latter approach, however the interested reader can easily apply an alternative weighting to derive an overall estimate.

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http://www.cdc.gov/nchswww/about/otheract/icd9/icd9hp2.htm.

[16]They defined respiratory admissions using the following codes from the ninth version of the International Classification of Disease (ICD): 464-466, 480-486, 490-494, 496.

Exhibit 3-2 Studies Used to Develop Respiratory Admissions Estimates

Location	Study	Endpoints Estimated [a] (ICD-9 code)	Pollutants Used in Final Model	Study Population
Toronto, Canada	Burnett et al. (1997)	all respiratory (464-466, 480-486, 490-494, 496)	O_3, $PM_{2.5-10}$, NO_2, SO_2	all ages
Toronto, Canada	Burnett et al. (1999)	asthma (493); respiratory infection (464, 466, 480-487, 494); non-asthma COPD (490-492, 496)	O_3, CO, $PM_{2.5-10}$ (asthma); O_3, NO_2, $PM_{2.5}$ (respiratory infection); O_3, CO, $PM_{2.5-10}$ (COPD).	all ages
Toronto, Canada	Thurston et al. (1994)	all respiratory (466, 480-482, 485, 490-493)	O_3, $PM_{2.5}$	all ages
New York, NY	Thurston et al. (1992)	all respiratory (466, 480-486, 490-493)	O_3, SO_4, H^+	all ages
Buffalo, NY	Thurston et al. (1992)	all respiratory (466, 480-486, 490-493)	O_3, SO_4, H^+	all ages
Montreal, Canada	Delfino et al. (1994)	asthma (493); all respiratory non-asthma (462-466, 480-487, 490-492, 494, 496);	PM_{10} [b]	all ages
Minneapolis-St. Paul, MN	Moolgavkar et al. (1997)	pneumonia (480-487); COPD (490-496)	O_3, SO_2, NO_2, PM_{10} (pneumonia); O_3, CO, PM_{10} (COPD)	>64
Minneapolis-St. Paul, MN	Schwartz (1994c)	pneumonia (480-486); COPD (490-496)	O_3, PM_{10} (pneumonia); PM_{10} (COPD)	>64
Birmingham, AL	Schwartz (1994a)	pneumonia (480-487); COPD (490-496)	PM_{10} [b]	>64
Detroit, MI	Schwartz (1994b)	pneumonia (480-486); non-asthma COPD (491-492, 494-496)	O_3, PM_{10}	>64
Spokane, WA	Schwartz (1996)	all respiratory (460-519)	O_3 [c]	>64
New Haven, CT	Schwartz (1995)	all respiratory (460-519)	O_3, PM_{10}	>64
Tacoma, WA	Schwartz (1995)	all respiratory (460-519)	O_3, PM_{10}	>64

[a] There is some variation in the ICD-9 codes for a given endpoint; "all respiratory" admissions, in particular, varies by study.

[b] A significant association was not found for ozone and hospital admissions.

[c] Due to limited data, O_3 and PM_{10} were considered in separate models. Both pollutants were significantly related to hospital admissions, and the correlation between PM_{10} and O_3 is modest (r=0.259) (Schwartz, 1996, Table 2).

Cardiovascular Hospital Admissions

We considered using a similar pooling procedure for cardiovascular admissions as we used for respiratory hospital admissions. However, there are only two studies that are relevant for ozone, and one of these studies (Burnett et al. (1997)) gives implausibly large estimates (Exhibit 4-4). The link between ozone and cardiovascular problems is not as well established as that between ozone and respiratory problems. Other studies have not found a link between ozone and cardiovascular problems, and instead have found associations with PM and CO (Exhibit 3-3). Acknowledging the uncertainty in our estimate, we use only the results of the Burnett et al. (1999) study, that focused on a narrow subset of cardiovascular problems, the relationship between ozone and abnormal heart rhythms or "dysrhythmias."[17]

Exhibit 3-3 Studies Used to Develop Cardiovascular Admissions Estimates

Location	Study	Endpoints Estimated (ICD-9 code)	Pollutants Used in Final Model	Study Population
Toronto, Canada	Burnett et al. (1997)	cardiac (410-414, 427-428)	O_3, $PM_{2.5-10}$	all ages
Toronto, Canada	Burnett et al. (1999)	ischemic heart disease (410-414); dysrhythmias (427); congestive heart failure (428)	NO_2, SO_2 (ischemic heart disease); $PM_{2.5}$, CO, O_3 (dysrhythmias); CO, NO_2 (congestive heart failure) [a]	all ages
Detroit, MI	Schwartz and Morris (1995)	ischemic heart disease (410-414); congestive heart failure (428)	CO, PM_{10} [b]	>64
Eight U.S. counties 1/88-12/90	Schwartz (1999)	cardiovascular disease (390-429)	CO, PM_{10} [b]	>64
Tucson, AZ 1/88-12/90	Schwartz (1997)	cardiovascular disease (390-429)	CO, PM_{10} [b]	>64

[a] Burnett et al. (1999) only found ozone associated with dysrhythmias, and did not report a significant link with ischemic heart disease or heart failure.

[b] A significant association was not found for ozone and hospital admissions.

[17]Dysrhythmias include the heart problems in ICD-9 code 427, and compose about 15 percent of hospital admissions associated with cardiovascular problems (ICD-9 codes 390-459). Dysrhythmias appear synonymous with "arrhythmias" and include problems such heartbeats that are too slow, too rapid, irregular, or too early. Rapid arrhythmias (greater than 100 beats per minute) are called tachycardias. Slow arrhythmias (slower than 60 beats per minute) are called bradycardias. Irregular heart rhythms are called fibrillations (as in atrial fibrillation). When a single heartbeat occurs earlier than normal, it is called a premature contraction. (See "arrhythmias" at: Cancer Web online medical dictionary: http://www.graylab.ac.uk/omd/index.html.)

3.3.2 Emergency Room Visits

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and ER visits. Because most ER visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

The only types of ER visit that have been explicitly linked to ozone in U.S. and Canadian epidemiological studies are asthma visits. However, it seems likely that ozone may be linked to other types of respiratory-related ER visits. So we include two separate estimates: (1) an estimate for asthma-related ER visits, and (2) an estimate for total respiratory-related ER visits based on the observed relationships between ER visits and hospital admissions.

Asthma ER Visits

We use three C-R functions for asthma-related ER visits, based on epidemiological studies by Cody et al. (1992), Weisel et al. (1995), and Stieb et al. (1996). The first two studies, Cody et al. and Weisel et al., were conducted in Northern New Jersey. The Cody et al. study examined asthma-related ER visits over a 16 month period between May, 1988 and August, 1989, and found that ozone was linked to asthma-related ER visits. No significant effect was seen for PM_{10} or SO_2. Using a one-pollutant model, Weisel et al. also found ozone linked to asthma-related ER visits in an all-age 1990 population for eight New Jersey counties. Finally, Stieb et al. examined asthma-related ER visits over an eight year period from May through September in St. John, New Brunswick, Canada. Ozone was linked to ER visits within the all-ages population, especially when ozone levels exceeded 75 ppb. No significant effect was seen for the other pollutants.

An additional study by Schwartz et al. (1993) failed to find a significant relationship between asthma-related ER visits and ozone. In this study of Seattle residents, Schwartz et al. found PM_{10} to be significantly related to asthma-related ER visits. We include the Schwartz et al. study in Exhibit 3-4, however, we do not include this result in our pooled estimate of the link between ozone and asthma-related ER visits.

Exhibit 3-4 Studies Used to Develop Asthma-related Emergency Room Visits

Location	Study	Pollutants Used in Final Model	Study Population
central and northern NJ	Cody et al. (1992)	O_3	all ages
central and northern NJ	Weisel et al. (1995)	O_3	all ages
St. John, New Brunswick, Canada	Stieb et al. (1996)	O_3	>15
Seattle, WA	Schwartz et al. (1993)	PM_{10} [a]	<65

[a] Schwartz et al. found no significant association between ozone and ER visits.

Total Respiratory ER Visits

There have been far fewer studies that examine the link between ozone and ER visits than those that examine the link between ozone and hospital admissions. This is curious, in light of the strong evidence linking ozone and a variety of respiratory ailments. However, in part, this is understandable. ER data are not easily obtainable, so epidemiologists have fewer opportunities to explore this relationship. This lack research results creates obvious problems for the present analysis, but there is a reasonable alternative. Since people that are hospitalized for ozone-related respiratory problems likely pass through the ER – i.e., ozone-related hospitalizations are presumably not planned events and need urgent treatment -- it seems reasonable to assume that ozone-related ER visits are some multiple of ozone-related hospital admissions. We estimate total respiratory-related ER visits using the observed relationship between ER visits and hospital admissions multiplied by the estimated number of ozone-related hospital admissions.

This is the same procedure used by the ALA (1996, p. 15). The ALA estimated respiratory-related ER visits by first estimating respiratory-related hospital admissions, and then multiplying the estimated hospital admissions by an observed (3:1) ratio between ER visits and hospital admissions.[18]

3.3.3 Minor Symptoms

Two studies, one by Ostro and Rothschild (1989) and the other by Krupnick et al. (1990), cover a variety of minor symptoms. To avoid double counting, we treat these two studies as alternative measures of the same health effect, and pool the incidence estimates.

Ostro and Rothschild (1989) estimated the impact of ozone and $PM_{2.5}$ on the incidence of minor restricted activity days (MRAD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.[19] Separate coefficients were reported for each year in the analysis (1976-1981). The coefficient used in this analysis is a weighted average of these coefficients using the inverse of the variance as the weight.

Krupnick et al. (1990) estimated the impact of coefficient of haze (COH, a measure of particulate

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[18]The ALA found a 3:1 ratio between all-cause ER visits and all-cause hospital admissions in Massachusetts; they reported that a sample from Philadelphia suggested a similar ratio. Due to the paucity of data on ER visits it is difficult to determine the correct ratio, however, what data are available suggest that the ratio used by the ALA seems reasonable. Smith et al. (1997, p. 789) reported that nationwide in 1987 about 37% of asthma-related ER visits end up as hospital admissions, on average for people of all ages. This works out to a ER visit to hospital admission ratio of 2.7:1 – close to the 3:1 used by ALA. The only other data of which we are aware are provided by Richards et al. (1981, p. 350), who reported that 13 percent of children coming to the ER for asthma in Los Angeles in 1979 were subsequently admitted to the hospital for treatment. This suggests a ratio of almost 8:1, however, we caution that the data from Richards et al. are a very limited sample.

[19]An MRAD is defined as a day of which a respondent was forced to alter her normal activity due to both respiratory and non-respiratory conditions. An MRAD does not result in either work loss or bed disability and therefore involves more minor conditions and reduction in activity (Ostro et al., 1989, p. 239).

matter concentrations), ozone and SO_2 on the incidence of any of 19 symptoms or conditions.[20] They used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model is that the model they used includes a lagged value of the dependent variable.

3.3.4 Asthma and Shortness of Breath

A number of studies have linked ozone to acute respiratory problems in asthmatics. Dockery et al. (1989) found ozone significantly related to the asthma rates of grade school children. Holguin et al. (1985) found ozone linked to asthma symptoms in a study of 42 Houston asthmatics ranging in age from 7 to 55 years. Following work by Whittemore and Korn (1980) – discussed below – Holguin et al. developed a sophisticated model that controlled for NO_2, pollen, whether a respondent had an asthma attack on the previous day, and other variables; however, they did not control for particulate matter. Using spectral analysis, Lebowitz et al. (1987) found ozone linked to wheeze, coughing, and reduced pulmonary functioning in asthmatics in Tucson. In subsequent work in Tucson, Krzyzanowski et al. (1992) found asthmatics were more sensitive to ozone exposure, experiencing a greater decline in pulmonary function than non-asthmatics. Ostro et al. (1995) found ozone increased symptoms of shortness of breath in a study of African-American asthmatics living in Los Angeles. Finally, Whittemore and Korn (1980) examined 443 asthmatic children and adults in six communities in southern California, for three 34-week periods in 1972-1975. Controlling for TSP levels, they found oxidants (90 percent of which is ozone) were significantly related to asthma attacks.[21]

After comprehensively reviewing the literature, EPA (1996b, p. 7-121) concluded that the epidemiological literature has "generally supported a direct association between ambient ozone/oxidant concentrations and acute respiratory morbidity in asthmatics." However, as this suggests, the published studies have not uniformly found a significant relationship between ozone and asthma symptoms. In a careful study of 24 cities in the U.S. and Canada , published concurrently with the EPA review, Dockery et al. (1996) did not report a significant relationship between the annual average of the daily peak ozone level and asthma symptoms reported in the previous year. It is unclear why Dockery et al. did not support previous findings. One possible explanation is that an annual measure is too crude, and that a daily measure of ozone is more appropriate. Alternatively, the annual average of the daily peak may yield different results than if the annual average of all hours was used as in Dockery et al. (1989).

We estimate ozone-related reductions in asthma symptoms using two studies. We present results based on the Whittemore and Korn study (1980) because it is the only study that controls for a measure of particulate matter, measures a symptom that is easily recognized, and can be easily used to develop a C-R function.[22] In addition, we present results based on the single pollutant model found in Ostro et al. (1995), because they focus on children, who may be particularly susceptible to the effects of ozone.

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[20]Krupnick et al. (1990) list 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

[21]EPA (1996b, pp. 7-115 to 7-121) nicely summarizes most of the studies presented here.

[22] The study by Krzyzanowski et al. (1992) uses technical measures of lung functioning such as peak expiratory flow rate; Lebowitz et al. (1987) is somewhat qualitative and thus difficult to translate into a C-R function.

The two studies that we use have symptom definitions and populations that overlap somewhat with each other, and the estimates based on these studies should not be added together to get an overall effect. Whittemore and Korn (1980) looked at the number of days that juvenile and adult asthmatics suffered one or more asthma attacks; and Ostro et al (1995) looked at the number of days with shortness of breath in African-American asthmatics ages 7-12. Clearly, the population in Ostro et al. are a subset of that covered by Whittemore and Korn. However, the effects examined in the study are still somewhat different and give another view of the impact of ozone on children.

3.3.5 Adult Onset of Asthma

In recent years, a number of studies have investigated the possible link between ozone and the development of chronic illness. Abbey et al. (1991; 1993) reported a significant link between ozone and the development of asthma, and Portney and Mullahy (1990) found ozone linked to sinusitis and hay fever. A review of research data by the EPA (1996b, p. 9-35) concluded that prolonged ozone exposure causes structural changes in several regions of the respiratory tract, and the available epidemiological studies are suggestive of a link between chronic health effects in humans and long-term ozone exposure.

Most recently, a study by McDonnell et al. (1999) carefully measured ozone exposure over 15 years, and found ozone exposure was linked to the onset of asthma in adult males. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures. No significant effect was found between ozone exposure and asthma in females, and no significant effect was reported for females or males due to exposure to PM, NO_2, SO_2, or SO_4. This evidence is intriguing, however, there is still enough uncertainty about the magnitude of the long-term effects of ozone, that we chose not to estimate its effects.

3.3.6 Mortality

The literature investigating the relationship between ozone and mortality has been rapidly evolving over the last several years. Of the 31 time-series epidemiology studies identified in the literature that report quantitative results on a possible association between daily ozone concentrations and daily mortality, 25 were published or presented since 1995. These studies were conducted in various urban areas throughout the world: sixteen in the United States or Canada, nine in Europe, two in Australia, and four in Latin America. Seventeen of the studies report a statistically significant relationship between ozone and mortality, with the more recent studies tending to find statistical significance more often than the earlier studies.

While the growing body of epidemiological studies suggests that there may be a positive relationship between ozone and premature mortality, there is still substantial uncertainty about this relationship. Because the evidence linking premature mortality and particulate matter is currently stronger than the evidence linking premature mortality and ozone, it is important that models of the relationship between ozone and mortality include a measure of particulate matter as well. Because of the lack of monitoring data on fine particulates or its components, however, the measure of particulate matter used in most studies was generally either particulate matter less than 10 microns (PM_{10}) or total suspended particulates (TSP) or, in some cases, Black Smoke. If a component of particulate matter (PM), such as particulates less than 2.5 microns ($PM_{2.5}$) or sulfates, is more highly correlated with ozone than with PM_{10} or TSP, and if this component is also related to premature mortality, then the apparent ozone effects on mortality could be at least partially spurious. Because of this, we adopt a conservative approach to estimating ozone-related health impacts, and do not include an estimate of ozone-related mortality.

3.4 SOURCES OF UNCERTAINTY

There are a number of sources of uncertainty in this analysis. The easiest to quantify is the uncertainty about the values of the ozone coefficients in C-R functions. Because the locations where C-R functions have been estimated (the study locations) are not necessarily the same as the locations of interest in this analysis (the assessment locations),[23] there are two sources of uncertainty about the value of the ozone coefficients in C-R functions: (1) statistical uncertainty (due to sampling error) about the true value of the coefficient in the study location, and (2) uncertainty about how well the value of an ozone coefficient in any study location approximates the value in any assessment location.

When a C-R function has been estimated in only a single location, we quantify the first type of uncertainty in CAPMS by using the reported ozone coefficient and its standard error to produce a normal distribution with the mean equal to the coefficient estimate and the standard deviation equal to the standard error. CAPMS then calculates the incidence change in adverse health effects for the nth percentile value from this normal distribution of coefficients, for n = 0.5, 1.5, ...,. 99.5. In the results section, we then present the 5^{th} percentile, mean, and 95^{th} percentile from the resulting distribution of incidence changes. If we have only a single study location, we have no information on the possible geographic variability of ozone coefficients.

When a C-R function has been estimated in several locations, we estimate a distribution of possible incidence changes that could result from a given change in ozone concentrations based on all the available C-R functions, using a Monte Carlo procedure. This distribution characterizes both the within-location uncertainty due to sampling error and the between-location variability in ozone coefficients.[24] On each iteration of the Monte Carlo procedure (1) one of the study-specific normal distributions described above is randomly selected, using either fixed effects or random effects weights (see Section 3.2.7), and (2) an estimate of the incidence change is randomly selected from the chosen distribution. This pooling procedure was used for several endpoints. For example, three C-R functions for asthma-related ER visits are used to estimate the range of avoidable ER visits.

However, there are other sources of uncertainty that are less easy to quantify, involving choices and judgements made at many points in the analysis. Therefore, the range of estimates presented here is only a partial reflection of the total uncertainty range. Exhibit 3-5 presents some of the key sources of uncertainty, the majority of which we can identify but not quantify.

The lack of actual ambient air quality monitoring data for each CAPMS grid cell adds a significant amount of uncertainty to the analysis. To estimate ozone levels at each CAPMS grid cell, we interpolate from the available monitoring data. Monitors are often in the more polluted urban areas – areas that have experienced past ozone problems. We may overestimate ozone levels in relatively unpolluted and unmonitored

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[23] Because this is a regional analysis, there are many assessment locations. A given endpoint could theoretically have a different C-R function in each location (e.g., county). Because it is not feasible to estimate county-specific C-R functions, however, a single C-R function is applied everywhere.

[24] The between-location variability implies uncertainty for any assessment location. If there is true variability in ozone coefficients across locations, then there is a corresponding uncertainty about which ozone coefficient best approximates the ozone coefficient in the assessment location.

areas when we interpolate. The overall effect on the analysis is tempered somewhat by the fact that rural areas have smaller populations, and will thus contribute less to the estimated incidence of adverse health effects.

The choice of studies clearly has an important effect on the analysis. We focused on epidemiological studies from the United States and Canada, and did not include some newly published studies from Europe, Australia, and other countries. Another choice we made was to use epidemiological studies and not use laboratory studies involving humans; nor did we attempt to extrapolate from the results of animal studies. The effect of these choices was to make the analysis more tractable – we still used a large number of studies – at the expense of examining a more limited set of adverse health effects. In addition, we chose not to estimate ozone's possible association with mortality and chronic effects.

The choice of the form of the C-R function – whether it is log-linear, logistic, or linear – depends on the choice of functional form made in the health effects study. We follow the choice made in the original study. However, if the original functional form is incorrect then our estimates will likely be incorrect. It is not uncommon for researchers to examine similar data and reach quite different conclusions. The extent of any resulting bias is unknown. We try to minimize bias by using a wide range of high quality studies.

Whether the C-R function for a given health endpoint is estimated by a single function from a single study or by a pooled function of C-R functions from several studies, that same C-R relationship is applied everywhere in the analysis. Although the C-R relationship may in fact vary somewhat from one location to another (for example, due to differences in population susceptibilities, or to activity patterns), location-specific C-R functions are available only for those locations in which studies were conducted. While a single function applied everywhere may result in overestimates of incidence changes in some locations and underestimates of incidence changes in other locations, these location-specific biases will to some extent cancel each other out when the total incidence change is calculated. It is not possible to know the extent or direction of the bias in the total incidence change based on application of a single C-R function everywhere.

We have limited the application of C-R functions to the age, and in one case, the race of the population considered in the study. The series of hospitalization studies that examined individuals 65 years and older may also be applicable to individuals 64 years of age (or younger). Similarly, Ostro et al. (1995) examined only a subset of children, however a greater number of children may suffer from ambient ozone exposures. This tends to underestimate the effects of ozone, however the overall effect on the analysis is uncertain.

As discussed in Appendix C, the hospitalization and ER visit studies by Thurston et al. (1994; 1992), Cody et al. (1992), Weisel et al. (1995), and Stieb et al. (1996), are linear functions. To apply these functions in a benefits analysis requires knowing the relevant populations in the study areas. Cody et al. and Weisel et al. gathered records from a group of hospitals in a number of counties in northern New Jersey. However, they did not gather records from all of the hospitals in these counties, so part of the population went to other hospitals. It is difficult to determine what fraction of the population was served, and we opted to include the full county population for any county that had a hospital in the study. In this case, our choice will likely lead to an underestimate of the number of ozone-related adverse health effects. For the other studies with linear functions, there may also be problems with correctly estimating the population served by the hospitals in the study.

Except for the linear C-R functions (e.g., Cody et al., 1992), the C-R functions that we use require a baseline incidence rate. We assume a single rate and apply it to the OTAG region, however, it is likely that the actual baseline incidence rate will vary by region and perhaps by the time of year. The overall effect of using a single baseline incidence rate everywhere is unclear.

Pollutants may interact in ways that magnify or counteract their individual effects. A mixture of pollutants – which is what people are exposed to – may be more or less harmful than is indicated by the simple sum of the effects of the pollutants considered in isolation. Controlled human studies (in laboratories) have generally examined two-pollutant mixtures, and have found that the effects of pollutants are additive (U.S. EPA, 1996b, p. 9-32). The large number of animal studies suggest that exposure to pollutants, each at relatively low levels, may result in significant effects (U.S. EPA, 1996b, p. 9-32). An (uncontrolled) epidemiological study by Krzyzanowski et al. (1992) found that in the presence of higher PM_{10} levels, ozone has a more detrimental effect on lung functioning. We do not account for any possible pollutant interactions in our incidence estimates, because the evidence for interactions is still too sparse to develop C-R functions. If interactions are significant, then we bias our results by excluding them, but the direction of the bias is not known.

Exhibit 3-5 Key Sources of Uncertainty

Ozone Concentrations
Ozone levels in unmonitored locations may not be well approximated by ozone values in monitored locations.
Concentration-Response Functions
Not all health effects associated with ozone may be included in this analysis. Only epidemiological studies from the United States and Canada were used. This analysis did not use studies from Europe, Australia, and other areas, nor did this analysis use controlled, laboratory studies. We did not attempt to estimate any chronic effects associated with ozone exposure, nor did we include the possibility that ozone is significantly related to mortality.
The correct functional form of each concentration-response relationship is uncertain. Poisson regression and logistic regression are often used in health effects studies; however, the choice of the functional form, as well as data filtering and other analytic techniques, may have a significant impact on the estimated result.
An epidemiological study's ozone coefficient (β) and standard error (typically based on a limited geographic area) may not be a good approximation for the OTAG region. In addition, there may be significant variation in ozone's effects within the OTAG region, due to activity patterns or other factors.
The C-R functions were applied to the same population groups in the OTAG region as were considered in the studies which estimated the C-R functions (e.g., children ages 10-12). This implicitly assumes that there is no effect for other groups in the population (e.g., children ages 0-9).
There is uncertainty about the correct population level for studies with linear C-R functions (e.g., Weisel et al., 1995).
The baseline incidence rates are not location-specific and may therefore not accurately represent the actual location-specific rates.
There is limited epidemiological evidence linking ozone with ER visits. Our estimate of total ER visits, based on the observed ratio of ER visits to hospital admissions (due to ozone and other causes), may not accurately capture the effect of ozone on ER visits.

4. RESULTS

This chapter presents estimates of the physical magnitude of changes in the selected health endpoints associated with changes in ambient 1997 ozone concentrations within the 37-state OTAG region. Incidence estimates have been calculated for the entire OTAG region, for each state present in the OTAG Region, and for selected metropolitan areas located throughout the OTAG region. All results have been rounded to two significant figures.[25]

Exhibit 4-1 presents total estimated incidence changes for each health effect aggregated across the entire OTAG region. These estimates are presented at the mean with an associated 90% confidence interval. To place these estimated incidence changes into context with predicted baseline incidences, Exhibit 4-2 displays the 1997 baseline incidence figures for all endpoints included in this analysis. In addition to baseline incidence, for each health effect, Exhibit 4-2 presents the mean estimated incidence change and the percent change in the baseline incidence rate. The estimated incidence changes are generally less than 10% of the baseline rate.

Exhibits 4-3 through 4-6 present the study-specific incidence results (for each of the three background assumptions) for those health effects that were pooled: respiratory hospital admissions, cardiovascular hospital admissions, ER visits, and minor symptoms, respectively. These tables also present the weights that were applied to each study in the pooling procedure. The final two tables included in the results section, Exhibits 4-7 and 4-8, present health effect estimates for 37 states and the District of Columbia, as well as for 34 metropolitan areas in the OTAG region. Only mean estimates for each of the health effects are presented in these tables.

4.1 Comparison with American Lung Association Study

To provide a point of comparison, we compare our results with those from a recent study by the American Lung Association (1996). Exhibit 4-9 presents estimates for ozone-related respiratory-related hospital admissions and total respiratory-related ER visits for the eight "cities" that are in both the ALA study and the current study.

Overall, the estimates for respiratory admissions are reasonably close -- in some cases exceptionally so -- while in others the estimates can differ by a factor of two. However, due to differences in the study designs the results *should* be different. In particular, the definitions of a "city" differ somewhat between the two studies. The ALA study used metropolitan statistical areas (MSAs), while the present study used ozone nonattainment areas. Two large differences are in the definition of New York and Philadelphia. This analysis included significant portions of New Jersey (Exhibit A-2) in each area; these are excluded from the ALA definition (1996, Table 1 in Appendix 1).

Other differences in the studies include: (1) the years under analysis. The present analysis estimated ozone-related effects in 1997, while the ALA study examined 1993 and 1994. (2) The epidemiological studies used in the two analyses. The present study uses some very recent studies that were not available when the ALA study was published. (3) The hospital admission rates. The ALA study devoted a significant amount of resources to collect hospital admission rates for each city; the present study used national rates.

[25] An exception is the total respiratory ER visit estimate, where we multiply the respiratory admission estimate by three.

Perhaps the biggest difference between the two studies is in the estimation of ER visits. The present analysis includes an estimate asthma-related ER visits based on the relatively limited number of epidemiological studies that specifically examined asthma-related ER visits. These studies point to a relationship between ozone and asthma-related ER visits, but not to other types of ER visits. It is unclear why there is little evidence for other types of ER visits; it could be due, in part, to the relatively limited availability of data on ER visits. Data on U.S. hospital admissions – especially those involving persons over 65 – are more widely available, and there are a number of studies linking ozone with a variety of respiratory-related hospital admissions.

In light of this, we include an estimate total respiratory ER visits based on the observed ratio between ER visits and hospital admissions. The ALA (1996, p. 15) estimated respiratory-related ER visits by first estimating respiratory-related hospital admissions, and then multiplying the estimated hospital admissions by an observed (3:1) ratio between ER visits and hospital admissions. Multiplying our estimates of respiratory admissions by three gives results that are reasonably close to those reported by ALA (Exhibit 4-9).

Exhibit 4-1 Ozone-related Adverse Health Effects (April-October 1997)

Health Effect [a]	Ages affected	5th percentile	mean	95th percentile
Respiratory hospital admissions [a]	all ages	11,000	53,000	110,000
Cardiovascular hospital admissions [a, b]	all ages	0	16,000	32,000
Total respiratory ER visits [c]	all ages	33,000	159,000	330,000
Asthma ER visits [a]	all ages	4,900	16,000	25,000
Minor symptoms [a]	18-65	34,000,000	83,000,000	130,000,000
Shortness of breath	7-12	110,000	450,000	760,000
Asthma attacks	all ages	2,500,000	6,200,000	9,900,000

Note: in two places, there is some overlap between the estimated health effects, so the effects should not be considered strictly additive: (1) about a third of the people entering the ER are subsequently admitted to the hospital, and (2) "asthma ER visits" are a subset of "total respiratory ER visits."

[a] A pooled estimate. The pooling of respiratory hospital admissions estimates, cardiovascular hospital admissions, ER visits for asthma, and minor symptoms is discussed in the text; results for each C-R function and the respective "study weights" used in pooling the results are given in Tables 4-3 to 4-6 below.

[b] Because the Burnett et al. (1999) C-R function was found to be statistically significant at the 90th percentile, the 5th percentile estimates in this analysis were negative. These negative values have been set to zero due to the implausible nature of negative incidence values associated with declines in ambient ozone concentrations.

[c] ER visits for all respiratory causes (including asthma, chronic bronchitis, pneumonia, and other respiratory problems) is estimated by multiplying the estimated number of respiratory hospital admissions by a factor of three. An estimate for the number of ER visits associated with cardiovascular problems was not included, because the evidence for the relationship between ozone and cardiovascular problems is more speculative, and not as well supported empirically as that between ozone and respiratory problems.

Exhibit 4-2 Baseline Rates and Ozone-related Adverse Health Effects (April-October 1997)

Health Effect [a]	Ages affected	Annual baseline incidence [b]	mean	% of baseline
Respiratory hospital admissions [a]	all ages	2,500,000	53,000	2.12%
Cardiovascular hospital admissions [a]	all ages	490,000	16,000	3.27%
Respiratory ER visits	all ages	7,500,000	159,000	2.12%
Asthma ER visits [a]	all ages	980,000	16,000	1.60%
Minor symptoms [a]	18-65	1,000,000,000	83,000,000	8.30%
Shortness of breath	7-12	4,400,000	450,000	10.23%
Asthma attacks	all ages	120,000,000	6,200,000	5.17%

[a] A pooled estimate. The pooling of respiratory hospital admissions estimates, cardiovascular hospital admissions, ER visits for asthma, and minor symptoms is discussed in the text; results for each C-R function and the respective "study weights" used in pooling the results are given in Tables 4-3 to 4-6 below.

[b] To estimate the baseline rate for respiratory-related hospital admissions, we used annual admissions per person of all ages for ICD-9 codes 460-519 (1.20% per year) multiplied with the population of all ages; for cardiovascular admissions, we used annual admissions per person of all ages for dysrhythmias, ICD-9 code 427 (0.24% year), multiplied with the population of all ages; for ER visits for asthma, we used the estimated asthma-related ER visit rate for persons of all ages (0.29% per year) multiplied with the population of all ages; for total respiratory-related ER visits, we simply multiplied the baseline incidence rate for respiratory hospital admissions by three; for the pooled estimate of minor restricted activity days and any-of-19 symptoms, we used the incidence rate for minor restricted activity days (7.8 per year) multiplied with the population ages 18-65; for shortness of breath we used the estimated annual incidence rate (20.4 per year) multiplied by the population of African-American asthmatics ages 7-12; for asthma attacks, we used the estimated annual number of days an asthmatic has asthma attacks (9.9 per year) multiplied with the population of asthmatics of all ages.

Exhibit 4-3 Ozone-related Respiratory Hospital Admissions (April-October 1997)

Study	Ages affected	Study weights [a]	5th %	mean	95th %
Burnett et al. (1997)	all ages	0.01	170,000	270,000	380,000
Burnett et al. (1999)	all ages	0.01	62,000	88,000	120,000
Thurston et al. (1994)	all ages	0.01	3,100	41,000	81,000
Thurston et al. (1992) [b] - New York City	all ages	0.24	13,000	34,000	55,000
Thurston et al. (1992) - Buffalo	all ages	0.02	11,000	76,000	140,000
Delfino et al. (1994) [c]	all ages	0	0	0	0
Moolgavkar et al. (1997)	>64	0.22	31,000	58,000	86,000
Schwartz (1994c)	>64	0.19	480	28,000	59,000
Schwartz (1994a)	>64	0.06	1,100	51,000	100,000
Schwartz (1994b)	>64	0.16	55,000	87,000	120,000
Schwartz (1996)	>64	<0.01	48,000	460,000	1,000,000
Schwartz (1995)	>64	0.06	5,900	60,000	110,000
Schwartz (1995)	>64	0.01	63,000	180,000	300,000
Pooled estimate of respiratory hospital admissions	all ages	--	11,000	53,000	110,000

[a] "Study weights" are the weights that are used in combining studies. For example, a weight of 0.05 suggests that a study estimate contributes about five percent to the combined estimate. Weights vary between studies because the distribution of incidence estimates generated by each of the different studies have differing variances. Those studies with a larger variance are assigned a smaller weight and those with a smaller variance are assigned a larger weight. It should also be noted that the two Burnett studies, both of which were conducted in Toronto, were combined before weights were assigned to each of the studies. This accounts for the relatively small weights assigned to them, despite an apparent lack of variance between the 5th and 95th percentiles.

[b] Thurston et al. (1992) presented separate coefficient estimates for Buffalo and New York, NY. Each is used here.

[c] Delfino et al. (1994) did not find a significant relationship between respiratory admissions and ozone, and is not included in the overall estimate.

Exhibit 4-4 Ozone-related Cardiovascular Hospital Admissions (April-October 1997)

Study	Ages affected	Study weights [a]	5th %	mean	95th %
Burnett et al. (1997)	all ages	0	220,000	430,000	640,000
Burnett et al. (1999) [b]	all ages	1.00	0	16,000	32,000
Schwartz & Morris (1995)	>64	0	0	0	0
Schwartz (1999)	>64	0	0	0	0
Schwartz (1997)	>64	0	0	0	0
Pooled estimate of cardiovascular hospital admissions	all ages	--	0	16,000	32,000

[a] "Study weights" are the weights that are used in combining studies. In this instance only a single study is used. The results from Burnett et al. (1997) strike us as implausibly large, especially given the fact that there is little other evidence that ozone is associated with cardiovascular problems. The studies by Schwartz and Morris (1995), Schwartz (1999), and Schwartz (1997) did not find a significant relationship between respiratory admissions and ozone, and are not included in the overall estimate.

[b] Because the Burnett et al. (1999) function was found to be statistically significant at the 90th percentile, the 5th percentile estimates in this analysis were negative. These negative values have been set to zero due to the implausible nature of negative incidence values associated with declines in ambient ozone concentrations.

Exhibit 4-5 Ozone-related Asthma ER Visits (April-October 1997)

Study	Ages affected	Study weights [a]	5th %	mean	95th %
Cody et al. (1992)	all ages	0.49	6,300	16,000	24,000
Weisel et al. (1995)	all ages	0.49	25,000	34,000	43,000
Stieb et al. (1996)	>15	0.02	15,000	120,000	210,000
Schwartz et al. (1993) [b]	<65	0	0	0	0
Pooled estimate asthma ER visits	all ages	--	4,900	16,000	25,000

[a] "Study weights" are the weights that are used in combining studies. In this instance, the study by Stieb et al. (1996) is given a relatively low weighting because the estimated incidence from this study has a relatively high variance.

[b] Schwartz et al. (1993) did not find a significant relationship between ozone and ER visits, and is not included in the overall estimate.

Exhibit 4-6 Ozone-related Days with Minor Symptoms (April-October 1997)

Study	Ages affected	Study weights [a]	5th %	mean	95th %
Any-of-19 symptoms	18-65	0.05	29,000,000	210,000,000	380,000,000
Minor restricted activity days (MRAD)	18-65	0.95	37,000,000	77,000,000	120,000,000
Pooled estimate of minor symptoms	18-65	--	34,000,000	83,000,000	130,000,000

[a] "Study weights" are the weights that are used in combining studies. In this instance, the estimate of any-of-19 symptoms is given a relatively low weighting because the estimated incidence has a relatively high variance.

Exhibit 4-7 Ozone-related Adverse Health Effects by State (mean cases April-October 1997)

State	respir. hosp. admis.	cardiovas. hosp. admis.	total respir. ER visits	asthma ER visits	minor symptoms	shortness of breath	asthma attacks
Alabama	1,000	310	3,000	350	1,700,000	19,000	130,000
Arkansas	760	210	2,280	200	1,000,000	8,100	79,000
Connecticut	870	250	2,610	250	1,400,000	4,200	100,000
Delaware	210	65	630	62	350,000	2,200	25,000
District of Columbia	120	36	360	39	230,000	3,800	16,000
Florida	4,200	1,000	12,600	1,000	5,200,000	31,000	400,000
Georgia	1,700	580	5,100	630	3,400,000	35,000	240,000
Illinois	2,400	740	7,200	770	4,000,000	24,000	310,000
Indiana	1,500	480	4,500	470	2,500,000	8,000	190,000
Iowa	740	210	2,220	180	910,000	770	73,000
Kansas	810	240	2,430	220	1,100,000	3,400	85,000
Kentucky	970	290	2,910	310	1,700,000	4,800	120,000
Louisiana	980	320	2,940	360	1,700,000	26,000	130,000
Maine	290	89	870	78	400,000	64	31,000
Maryland	1,300	410	3,900	450	2,500,000	21,000	180,000
Massachusetts	1,500	440	4,500	430	2,300,000	3,600	170,000
Michigan	2,100	670	6,300	660	3,600,000	20,000	280,000
Minnesota	1,100	340	3,300	300	1,600,000	1,700	120,000
Mississippi	690	210	2,070	230	1,100,000	19,000	85,000
Missouri	1,500	430	4,500	430	2,100,000	9,000	160,000
Nebraska	400	110	1,200	100	510,000	860	41,000
New Hampshire	230	75	690	75	400,000	99	30,000
New Jersey	2,000	570	6,000	600	3,400,000	15,000	240,000
New York	4,100	1,200	12,300	1,200	6,900,000	37,000	510,000
North Carolina	1,900	610	5,700	630	3,300,000	26,000	240,000
North Dakota	170	50	510	43	200,000	51	16,000
Ohio	2,800	830	8,400	870	4,700,000	19,000	350,000
Oklahoma	980	290	2,940	280	1,400,000	4,600	110,000
Pennsylvania	3,200	840	9,600	860	4,900,000	16,000	370,000
Rhode Island	270	73	810	72	380,000	580	28,000
South Carolina	830	270	2,490	300	1,500,000	18,000	110,000
South Dakota	190	57	570	50	230,000	54	19,000
Tennessee	1,500	460	4,500	470	2,500,000	16,000	180,000
Texas	4,600	1,600	13,800	1,700	8,900,000	42,000	660,000
Vermont	150	53	450	43	230,000	29	17,000
Virginia	1,600	550	4,800	560	3,000,000	19,000	220,000
West Virginia	510	140	1,530	140	760,000	900	58,000
Wisconsin	1,400	420	4,200	380	1,900,000	5,100	150,000

Exhibit 4-8 Ozone-related Adverse Health Effects by City (mean cases April-October 1997)

Metropolitan area	respir. hosp. admis.	cardiovas. hosp. admis.	total respir. ER visits	asthma ER visits	minor symptoms	shortness of breath	asthma attacks
Atlanta	580	230	1,740	260	1,500,000	13,000	100,000
Baltimore	630	200	1,890	220	1,200,000	11,000	86,000
Birmingham	170	49	510	64	320,000	4,000	24,000
Charleston	52	18	155	19	100,000	1,400	7,300
Charlotte	120	43	360	51	280,000	2,600	20,000
Chicago	1,500	470	4,500	500	2,700,000	21,000	200,000
Cincinnati	390	120	1,170	140	760,000	3,800	57,000
Cleveland	760	220	2,280	220	1,200,000	6,900	89,000
Columbia	52	18	156	22	120,000	1,700	8,200
Dayton	240	72	720	79	440,000	2,200	32,000
Detroit	930	290	2,790	310	1,800,000	14,000	130,000
Evansville, IN	58	15	174	16	80,000	260	6,100
Grand Rapids	160	56	480	53	290,000	810	22,000
Hartford	660	200	1,980	190	1,000,000	3,000	75,000
Indianapolis	210	69	630	69	370,000	3,100	27,000
Kalamazoo, MI	47	16	142	15	93,000	350	6,700
Memphis	260	91	780	97	500,000	8,500	37,000
Miami/Ft. Lauderdale	1,200	280	3,600	280	1,400,000	11,000	110,000
Milwaukee	470	150	1,410	140	730,000	4,600	55,000
Minneapolis/St. Paul	470	180	1,410	150	900,000	1,600	66,000
Mobile	88	27	264	32	160,000	2,300	12,000
Montgomery	50	16	149	18	85,000	1,600	6,600
Muskegon, MI	46	14	137	13	68,000	400	5,300
Nashville	140	43	420	47	270,000	2,200	19,000
New York	4,100	1,200	12,300	1,200	7,200,000	43,000	520,000
Philadelphia	1,600	460	4,800	480	2,700,000	17,000	200,000
Pittsburgh	730	180	2,190	180	1,100,000	2,700	79,000
Raleigh/Durham	150	57	450	61	340,000	2,600	23,000
Richmond	210	65	630	72	380,000	3,700	27,000
Savannah	60	19	180	18	92,000	1,500	7,100
St. Louis	610	180	1,830	200	1,000,000	7,400	79,000
Tampa/St. Petersburg	780	160	2,340	170	860,000	3,600	68,000
Toledo	140	43	420	42	230,000	1,100	18,000
Washington	800	310	2,400	340	2,000,000	15,000	130,000

Exhibit 4-9 Incidence Comparisons with the American Lung Association Study

City	American Lung Assoc. Study [a]		Present Analysis		
	Respiratory Admissions	Respiratory ER Visits	Respiratory Admissions	Total Respiratory ER Visits	Asthma ER Visits
Baltimore	660	2,000	630	1,900	220
Detroit	940	2,800	930	2,800	310
Hartford [b]	380	1,100	660	2,000	190
Milwaukee	270	800	480	1,400	140
New York	2,400	7,300	4,100	12,000	1,200
Philadelphia	850	2,600	1,600	4,800	480
St. Louis	530	1,600	610	1,800	200
Washington, DC	600	1,800	800	2,400	340

[a] Our mean estimates and the mean estimates from the American Lung Association (1996, Tables 15 and 32) study are presented here with two significant digits. Note that we present results for the eight cities that are in both the ALA study and the current study.

[b] The ALA results for Hartford and New Haven have been combined, since these two areas are included in the "Hartford" ozone nonattainment area used in the present analysis.

5. REFERENCES

Abbey, D.E., P.K. Mills, F.F. Petersen and W.L. Beeson. 1991. Long-Term Ambient Concentrations of Total Suspended Particulates and Oxidants As Related to Incidence of Chronic Disease in California 7th-Day Adventists. Environmental Health Perspectives. 94(AUG): 43-50.

Abbey, D.E., F. Petersen, P.K. Mills and W.L. Beeson. 1993. Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. 48(1): 33-46.

Abt Associates Inc. 1998. Air Quality Estimation for the NOx SIP Call RIA. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, under contract no. 68-D-98-001. Research Triangle Park, NC. September.

Abt Associates Inc. 1999. Co-Control Benefits of Greenhouse Gas Control Policies. Prepared for U.S. EPA, Office of Policy, under contract no. 68-W4-0029. Washington, DC. February.

Adams, P.F. and M.A. Marano. 1995. Current Estimates from the National Health Interview Survey, 1994. National Center for Health Statistics. Hyattsville, MD.

American Lung Association. 1996. Ambient Ozone Exposure and Emergency Hospital Admissions and Emergency Room Visits for Respiratory Problems in Thirteen U.S. Cities. Prepared by H. Ozkaynak, J.D. Spengler, M. O'Neill, J. Xue, H. Zhou, K. Gilbert, and S. Ramstrom. Washington, DC. June.

Avol, E.L., W.S. Linn, D.A. Shamoo, L.M. Valencia, U.T. Anzar, T.G. Venet and J.D. Hackney. 1985. Respiratory effects of photochemical oxidant air pollution in exercising adolescents. Am Rev Respir Dis. 132(3): 619-22.

Burnett, R.T. 1999. Email to Donald R. McCubbin, Abt Associates Inc.

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

Burnett, R.T., M. Smith-Doiron, D. Stieb, S. Cakmak and J.R. Brook. 1999. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. Archives Environmental Health. 54(2): 130-139.

Cody, R.P., C.P. Weisel, G. Birnbaum and P.J. Lioy. 1992. The effect of ozone associated with summertime photochemical smog on the frequency of asthma visits to hospital emergency departments. Environ Res. 58(2): 184-94.

Collins, J.G. 1997. Prevalence of Selected Chronic Conditions: United States 1990-1992. National Center for Health Statistics: Hyattsville, MD.

Delfino, R.J., M.R. Becklake and J.A. Hanley. 1994. The relationship of urgent hospital admissions for respiratory illnesses to photochemical air pollution levels in Montreal. Environ Res. 67(1): 1-19.

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

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

Graves, E.J. and B.S. Gillum. 1997. Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1994. National Center for Health Statistics. Hyattsville, MD. March.

Holguin, A.H., P.A. Buffler, C.F. Contant, T.H. Stock, D. Kotchmar, B.P. Hsi, D.E. Jenkins, B.M. Gehan, L.M. Noel and M. Mei. 1985. The effects of ozone on asthmatics in the Houston area. In Transactions: Evaluation of the Scientific Basis for Ozone/Oxidants Standards. Lee, S.D., Ed. Air Pollution Control Association: Pittsburgh, PA. p. 262-280.

Krupnick, A.J. 1988. An Analysis of Selected Health Benefits from Reductions in Photochemical Oxidants in the Northeastern United States: Final Report. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Washington, DC. EPA Contract No. 68-02-4323. September.

Krupnick, A.J., W. Harrington and B. Ostro. 1990. Ambient Ozone and Acute Health Effects - Evidence From Daily Data. Journal of Environmental Economics and Management. 18(1): 1-18.

Krzyzanowski, M., J.J. Quackenboss and M.D. Lebowitz. 1992. Relation of peak expiratory flow rates and symptoms to ambient ozone. Arch Environ Health. 47(2): 107-15.

Lebowitz, M.D., L. Collins and C.J. Holberg. 1987. Time series analyses of respiratory responses to indoor and outdoor environmental phenomena. Environ Res. 43(2): 332-41.

Lipfert, F.W. 1993. A Critical Review of Studies of the Association Between Demands For Hospital Services and Air Pollution. Environmental Health Perspectives. 101(S2): 229-268.

McDonnell, W.F., D.E. Abbey, N. Nishino and M.D. Lebowitz. 1999. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG study. Environ Res. 80(2 Pt 1): 110-21.

Moolgavkar, S.H., E.G. Luebeck and E.L. Anderson. 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis St. Paul and Birmingham. Epidemiology. 8(4): 364-370.

National Center for Health Statistics. 1994. Vital Statistics of the United States, 1990, vol II, Mortality, Part B. Public Health Service: Washington, DC.

National Heart, L., and Blood Institute. 1997. Guidelines for the Diagnosis and Management of Asthma: Expert Panel Report 2. National Institutes of Health. Bethesda, MD. NIH Publication No. 97-4051. July.

Ostro, B.D., M.J. Lipsett, J.K. Mann, H. Braxtonowens and M.C. White. 1995. Air Pollution and Asthma Exacerbations Among African-American Children in Los Angeles. Inhalation Toxicology. 7(5): 711-722.

Ostro, B.D., M.J. Lipsett, M.B. Wiener and J.C. Selner. 1991. Asthmatic Responses to Airborne Acid Aerosols. Am J Public Health. 81(6): 694-702.

Ostro, B.D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity - an Observational Study of Multiple Pollutants. Environ Res. 50(2): 238-247.

Portney, P.R. and J. Mullahy. 1990. Urban Air Quality and Chronic Respiratory Disease. Regional Science and Urban Economics. 20(3): 407-418.

Richards, W., S.P. Azen, J. Weiss, S. Stocking and J. Church. 1981. Los Angeles air pollution and asthma in children. Ann Allergy. 47(5 Pt 1): 348-54.

Schwartz, J. 1994a. Air Pollution and Hospital Admissions For the Elderly in Birmingham, Alabama. American Journal of Epidemiology. 139(6): 589-598.

Schwartz, J. 1994b. Air Pollution and Hospital Admissions For the Elderly in Detroit, Michigan. American Journal of Respiratory and Critical Care Medicine. 150(3): 648-655.

Schwartz, J. 1994c. PM(10) Ozone, and Hospital Admissions For the Elderly in Minneapolis St Paul, Minnesota. Archives of Environmental Health. 49(5): 366-374.

Schwartz, J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. Thorax. 50(5): 531-538.

Schwartz, J. 1996. Air pollution and hospital admissions for respiratory disease. Epidemiology. 7(1): 20-28.

Schwartz, J. 1997. Air pollution and hospital admissions for cardiovascular disease in Tucson. Epidemiology. 8(4): 371-377.

Schwartz, J. 1999. Air pollution and hospital admissions for heart disease in eight U.S. counties. Epidemiology. 10(1): 17-22.

Schwartz, J. and R. Morris. 1995. Air Pollution and Hospital Admissions For Cardiovascular Disease in Detroit, Michigan. American Journal of Epidemiology. 142(1): 23-35.

Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista and W.B. Saunders. 1997. A national estimate of the economic costs of asthma. Am J Respir Crit Care Med. 156(3 Pt 1): 787-93.

Stieb, D.M., R.T. Burnett, R.C. Beveridge and J.R. Brook. 1996. Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. Environmental Health Perspectives. 104(12): 1354-1360.

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

Thurston, G.D., K. Ito, P.L. Kinney and M. Lippmann. 1992. A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and

1989 summers. J Expo Anal Environ Epidemiol. 2(4): 429-450.

U.S. Bureau of the Census. 1997. Statistical Abstract of the United States: 1997. 117 ed. Washington, DC.

U.S. EPA. 1996a. Air Quality Criteria for Ozone and Related Photochemical Oxidants. Volume I. U.S. EPA, Office of Research and Development. Washington, DC. EPA-/600/P-93/004aF. July.

U.S. EPA. 1996b. Air Quality Criteria for Ozone and Related Photochemical Oxidants. Volume III. U.S. EPA, Office of Research and Development. Washington, DC. EPA-/600/P-93/004cF. July.

U.S. EPA. 1996c. Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. OAQPS Staff Paper. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA-452\R-96-007. June.

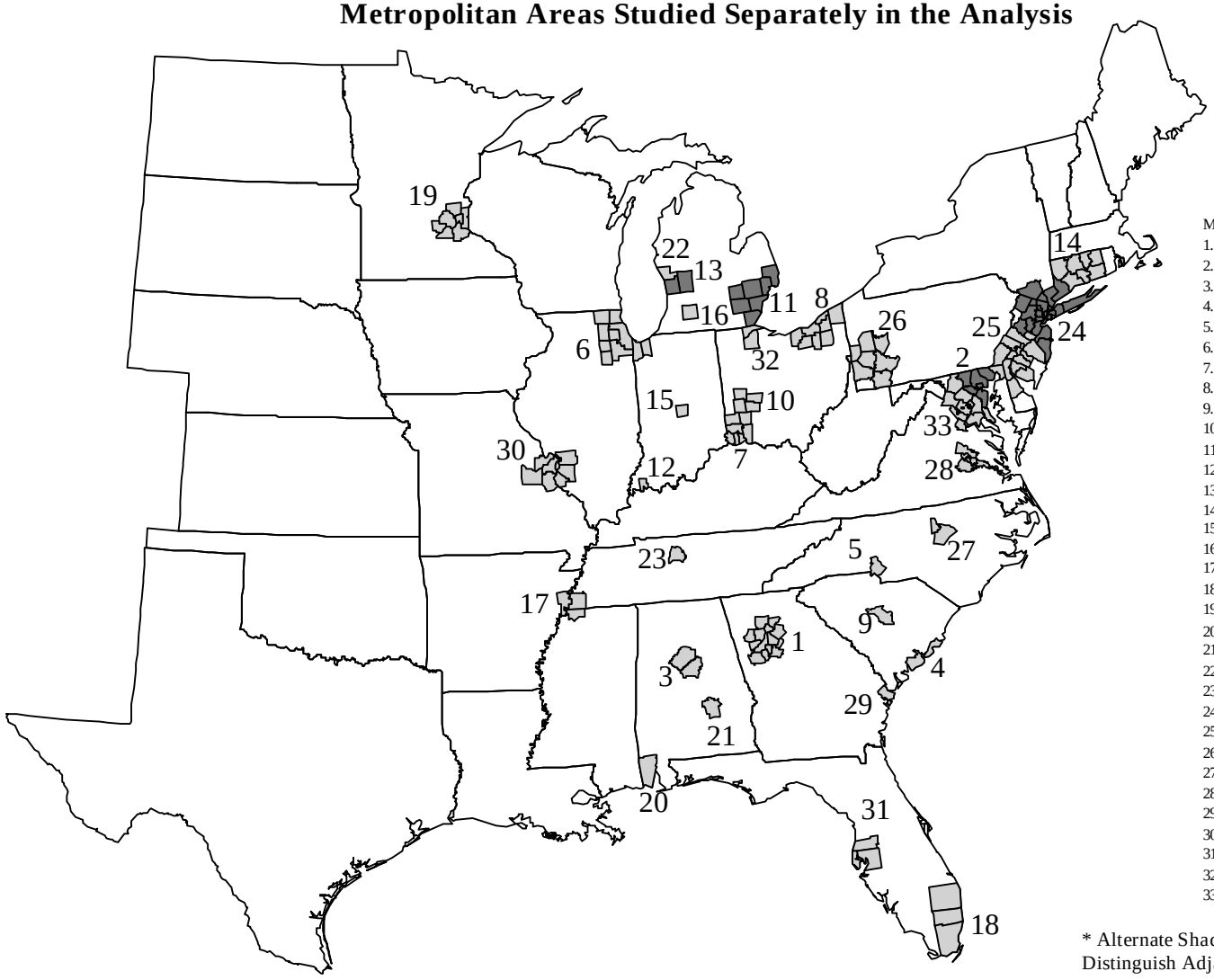
U.S. EPA. 1997. The Benefits and Costs of the Clean Air Act: 1970 to 1990. U.S. EPA, Office of Air and Radiation, Office of Policy, Planning and Evaluation. Washington, DC. October.

U.S. Office of the Federal Register. 1995. 40 CFR, Protection of Environment, revised July 1, 1995. U.S. Government Printing Office. Washington, DC.

Weisel, C.P., R.P. Cody and P.J. Lioy. 1995. Relationship between summertime ambient ozone levels and emergency department visits for asthma in central New Jersey. Environ Health Perspect. 103 Suppl 2: 97-102.

Whittemore, A.S. and E.L. Korn. 1980. Asthma and Air Pollution in the Los Angeles Area. Am J Public Health. 70: 687-696.

Exhibit A-1
Metropolitan Areas Studied Separately in the Analysis



Metropolitan Areas
1. Atlanta
2. Baltimore
3. Birmingham
4. Charleston
5. Charlotte
6. Chicago
7. Cincinnati
8. Cleveland
9. Columbia
10. Dayton
11. Detroit
12. Evansville, IN
13. Grand Rapids
14. Hartford
15. Indianapolis
16. Kalamazoo, MI
17. Memphis
18. Miami/Ft. Lauderdale
19. Minneapolis/St. Paul
20. Mobile
21. Montgomery
22. Muskegon, MI
23. Nashville
24. New York
25. Philadelphia
26. Pittsburgh
27. Raleigh/Durham
28. Richmond
29. Savannah
30. St. Louis
31. Tampa/St. Petersburg
32. Toledo
33. Washington

* Alternate Shading Used to Distinguish Adjacent Metroplitan Areas

Exhibit A-2 List of Metropolitan Counties Considered Separately in the Analysis

Area name	County	State	1997 population
Atlanta	Cherokee	GA	104,953
	Clayton	GA	211,830
	Cobb	GA	520,986
	Coweta	GA	62,658
	De Kalb	GA	635,124
	Douglas	GA	82,751
	Fayette	GA	72,622
	Forsyth	GA	51,292
	Fulton	GA	755,104
	Gwinnett	GA	410,637
	Henry	GA	68,345
	Paulding	GA	48,415
	Rockdale	GA	62,936
Baltimore	Anne Arundel	MD	460,917
	Baltimore	MD	746,697
	Carrol	MD	133,091
	City of Baltimore	MD	794,049
	Hartford	MD	196,485
	Howard	MD	202,095
Birmingham	Jefferson	AL	701,722
	Shelby	AL	107,005
Charleston	Charleston	SC	318,616
Charlotte	Mecklenburg	NC	577,573
Chicago	Cook	IL	5,332,774
	Du Page	IL	816,527
	Grundy	IL	33,773
	Kane	IL	331,625
	Kendall	IL	41,167
	Lake	IL	539,445
	McHenry	IL	191,406
	Will	IL	373,238
	Lake	IN	508,171
	Porter	IN	137,760
Cincinnati	Boone	KY	61,368
	Campbell	KY	89,375
	Kenton	KY	151,362
	Butler	OH	301,891
	Clermont	OH	155,550
	Hamilton	OH	897,195
	Warren	OH	117,975
Cleveland	Ashtabula	OH	103,379
	Cuyahoga	OH	1,462,627
	Geauga	OH	84,022
	Lake	OH	223,198
Cleveland (cont.)	Lorain	OH	280,811

Exhibit A-2 List of Metropolitan Counties Considered Separately in the Analysis (cont.)

Area name	County	State	1997 population
	Medina	OH	126,723
	Portage	OH	147,675
	Summit	OH	533,398
Columbia	Richland	SC	308,557
Dayton	Clark	OH	152,817
	Greene	OH	141,612
	Miami	OH	96,507
	Montgomery	OH	594,319
Detroit	Livingston	MI	119,600
	Macomb	MI	741,987
	Monroe	MI	138,169
	Oakland	MI	1,120,728
	St. Clair	MI	150,587
	Washtenaw	MI	292,623
	Wayne	MI	2,184,075
Evansville, IN	Vanderburgh	IN	176,362
Grand Rapids	Kent	MI	517,780
	Ottawa	MI	194,196
Hartford	Hartford	CT	849,798
	Litchfield	CT	173,674
	Middlesex	CT	142,855
	New Haven	CT	802,343
	New London	CT	254,353
	Tolland	CT	128,394
	Windham	CT	102,279
Indianapolis	Marion	IN	851,764
Kalamazoo, MI	Kalamazoo	MI	231,061
Memphis	Crittenden	AR	54,324
	De Soto	MS	72,739
	Shelby	TN	924,471
Miami/Fort Lauderdale	Broward	FL	1,426,422
	Dade	FL	2,200,829
	Palm Beach	FL	981,076
Milwaukee	Kenosha	WI	136,895
	Milwaukee	WI	1,024,518
	Ozaukee	WI	77,781
	Racine	WI	186,934
	Washington	WI	101,804
	Waukesha	WI	325,431
Minneapolis/St. Paul	Anoka	MN	262,840
	Carver	MN	51,687
	Dakota	MN	296,911
	Hennepin	MN	1,113,810
Minneapolis/St. Paul (cont.)	Ramsey	MN	524,053
	Scott	MN	62,400

Exhibit A-2 List of Metropolitan Counties Considered Separately in the Analysis (cont.)

Area name	County	State	1997 population
	Washington	MN	157,388
Mobile	Mobile	AL	407,807
Montgomery	Montgomery	AL	225,187
Muskegon, MI	Muskegon	MI	164,426
Nashville	Davidson	TN	571,447
New York	Fairfield	CT	825,713
	Bergen	NJ	860,749
	Essex	NJ	811,556
	Hudson	NJ	576,801
	Hunterdon	NJ	112,387
	Middlesex	NJ	700,565
	Monmouth	NJ	576,820
	Morris	NJ	439,401
	Ocean	NJ	451,756
	Passaic	NJ	472,471
	Somerset	NJ	250,571
	Sussex	NJ	136,545
	Union	NJ	514,979
	Bronx	NY	1,213,860
	Kings	NY	2,319,913
	Nassau	NY	1,298,112
	New York	NY	1,499,981
	Orange	NY	310,207
	Queens	NY	1,967,924
	Richmond	NY	382,145
	Rockland	NY	267,691
	Suffolk	NY	1,332,899
	Westchester	NY	882,175
Philadelphia	Kent	DE	123,704
	New Castle	DE	492,576
	Cecil	MD	76,964
	Burlington	NJ	411,986
	Camden	NJ	524,369
	Cumberland	NJ	143,961
	Gloucester	NJ	239,936
	Mercer	NJ	339,784
	Salem	NJ	68,086
	Bucks	PA	552,792
	Chester	PA	384,472
	Delaware	PA	559,416
	Montgomery	PA	692,673
	Philadelphia	PA	1,619,650
Pittsburgh	Allegheny	PA	1,365,160
	Armstrong	PA	75,047
	Beaver	PA	190,084

Exhibit A-2 List of Metropolitan Counties Considered Separately in the Analysis (cont.)

Area name	County	State	1997 population
	Butler	PA	155,267
	Fayette	PA	148,463
	Washington	PA	208,966
	Westmoreland	PA	378,261
Raleigh/Durham	Durham	NC	205,349
	Wake	NC	478,127
Richmond	Charles City	VA	6,909
	Chesterfield	VA	230,242
	Colonial Heights city	VA	17,673
	Hanover	VA	69,645
	Henrico	VA	239,713
	Hopewell city	VA	25,415
	Richmond city	VA	223,405
Savannah	Chatham	GA	252,416
St. Louis	Madison	IL	260,344
	Monroe	IL	23,418
	St. Clair	IL	274,567
	Franklin	MO	85,551
	Jefferson	MO	181,915
	St. Charles	MO	226,001
	St. Louis city	MO	421,096
	St. Louis	MO	1,054,667
Tampa-St. Petersburg	Hillsborough	FL	947,598
	Pasco	FL	319,398
	Pinellas	FL	967,608
Toledo	Lucas	OH	478,884
	Wood	OH	117,307
Washington, DC	Washington	DC	538,497
	Calvert	MD	55,416
	Charles	MD	109,121
	Frederick	MD	162,041
	Montgomery	MD	816,712
	Prince George's	MD	786,764
	Alexandria city	VA	122,325
	Arlington	VA	188,067
	Fairfax City	VA	21,588
	Fairfax	VA	900,622
	Falls Church City	VA	10,537
	Loudon	VA	94,754
	Manassas city	VA	30,759
	Manassas Park city	VA	7,409
Washington, DC (cont.)	Prince William	VA	237,295
	Stafford	VA	67,369

Counties in each area are based on EPA defined ozone attainment areas. See: http://www.epa.gov/oar/oaqps/greenbk/ozone1hr/may98/cfr81.html For many areas, only one county constitutes an area (e.g., Nashville, Evansville, Columbia SC, Mobile AL). For Raleigh-Durham, the counties containing Raleigh and Durham were included-- for others only the county containing the study city were included. For the Florida areas, Miami-Ft. Laud, Tampa-

Area name	County	State	1997 population

St. Pete's-- logical subsets of OMB defined MSAs were used. In areas where only parts of a county were in the EPA area, the whole county was included. In areas where parts of a county may appear in two areas (NY/CT), the county was placed in the area which contained more of the county in area.

Exhibit B-1
Seasonal Average Ozone (April-October, 1997)

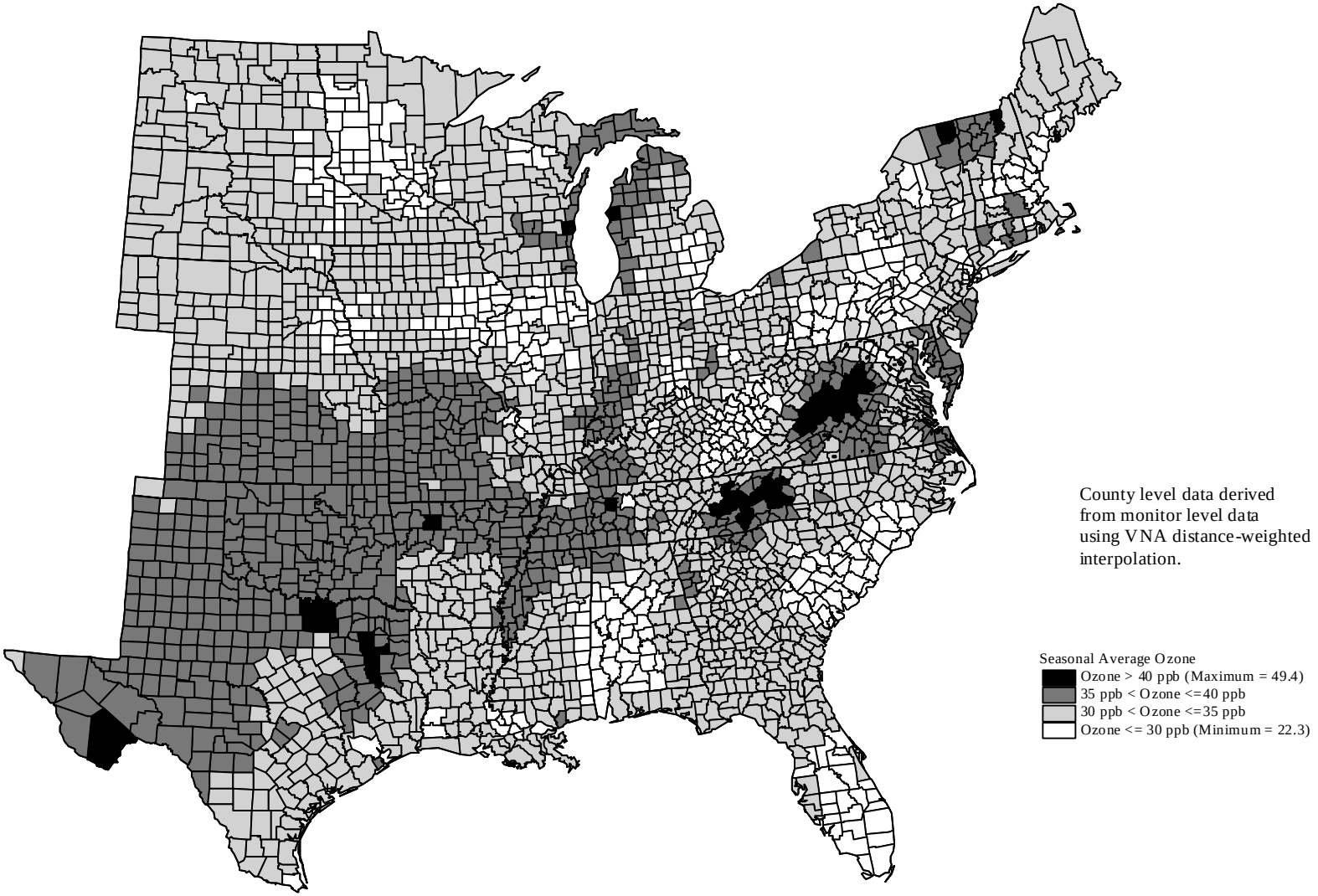


Exhibit B-2
Seasonal Average of the Daily 12-Hour Average Ozone Level (April-October, 1997)

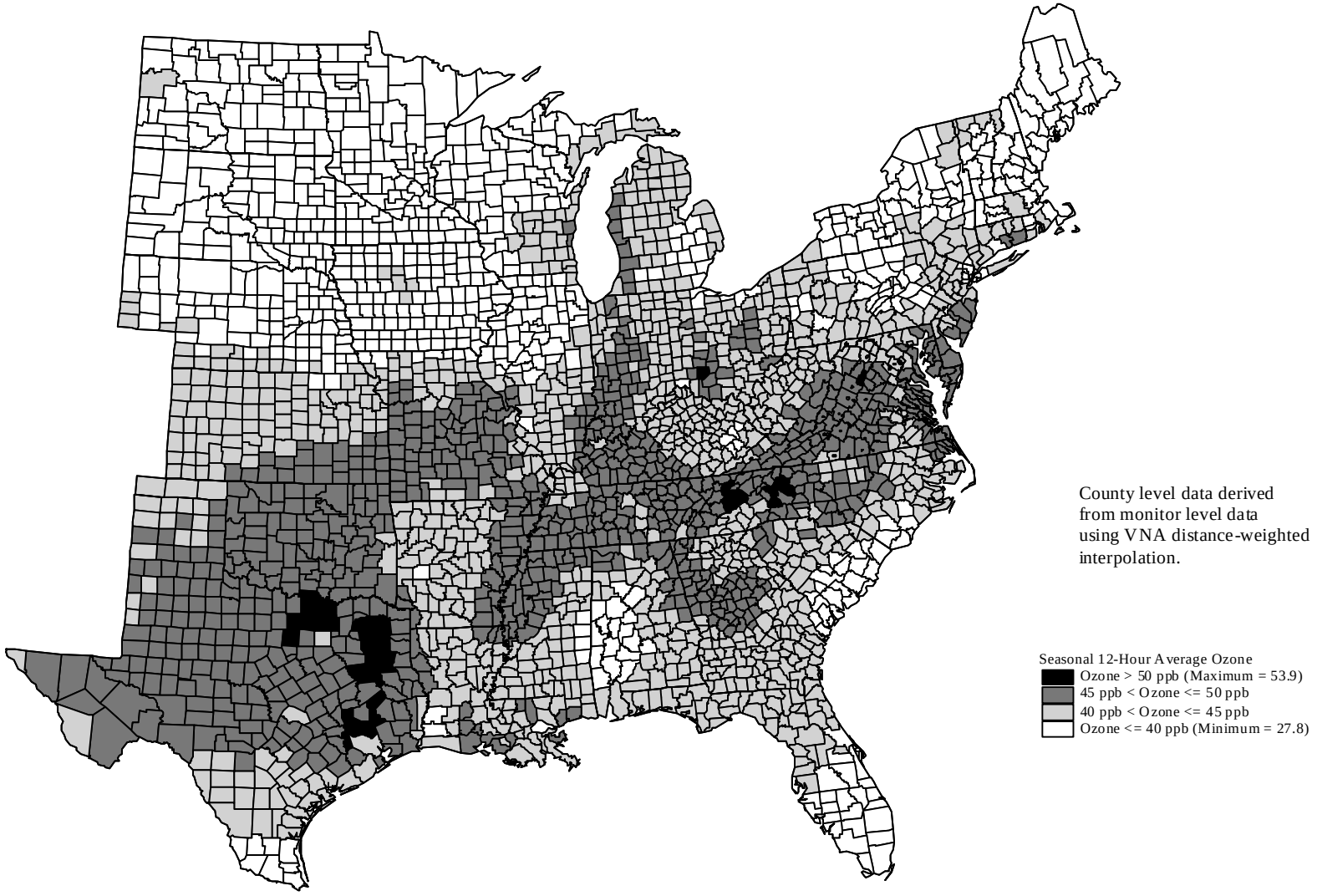


Exhibit B-3
Seasonal Average of the Daily 8-Hour Average Ozone Level (April-October, 1997)

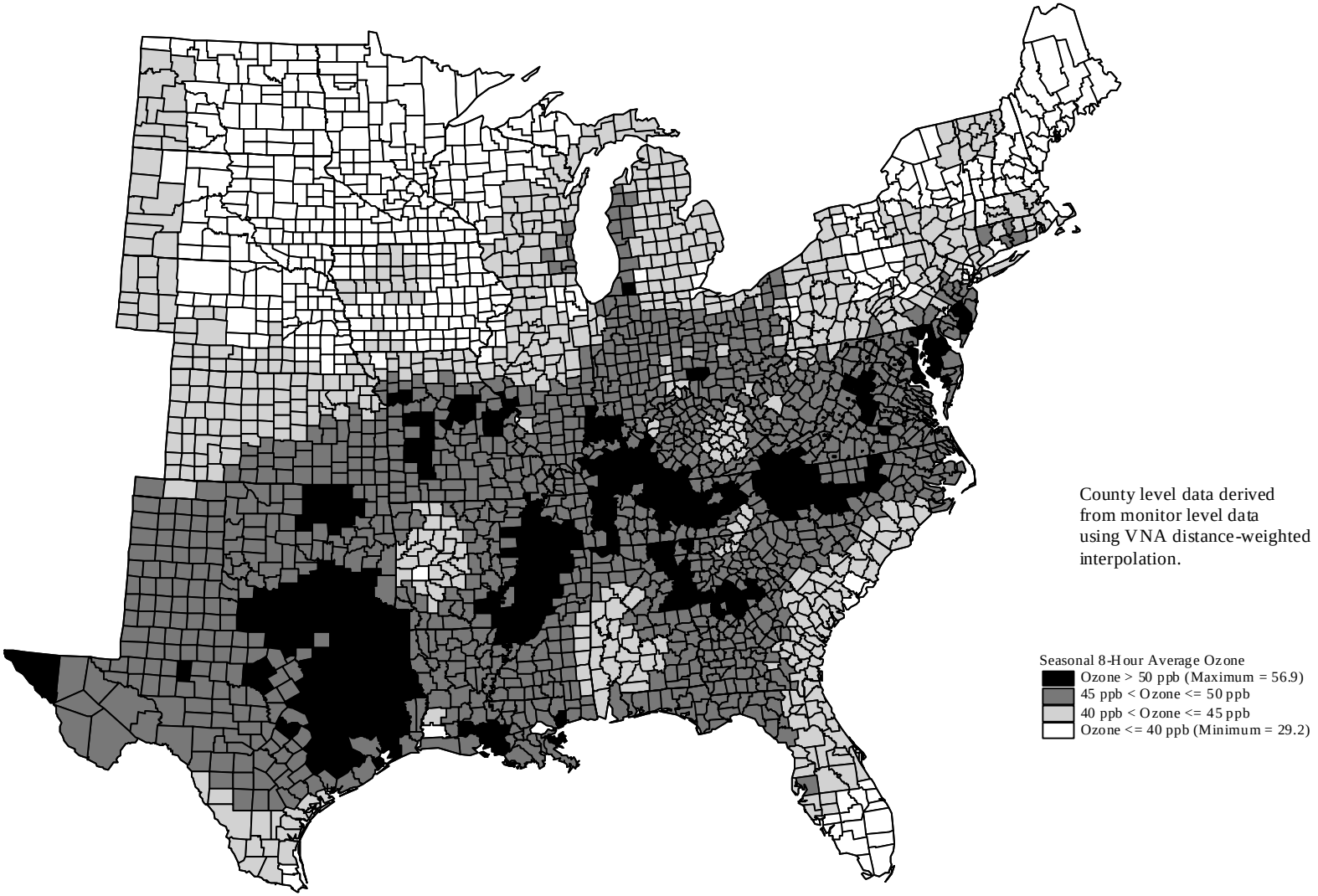


Exhibit B-4
Seasonal Average of the Daily 5-Hour Average Ozone Level (April-October, 1997)

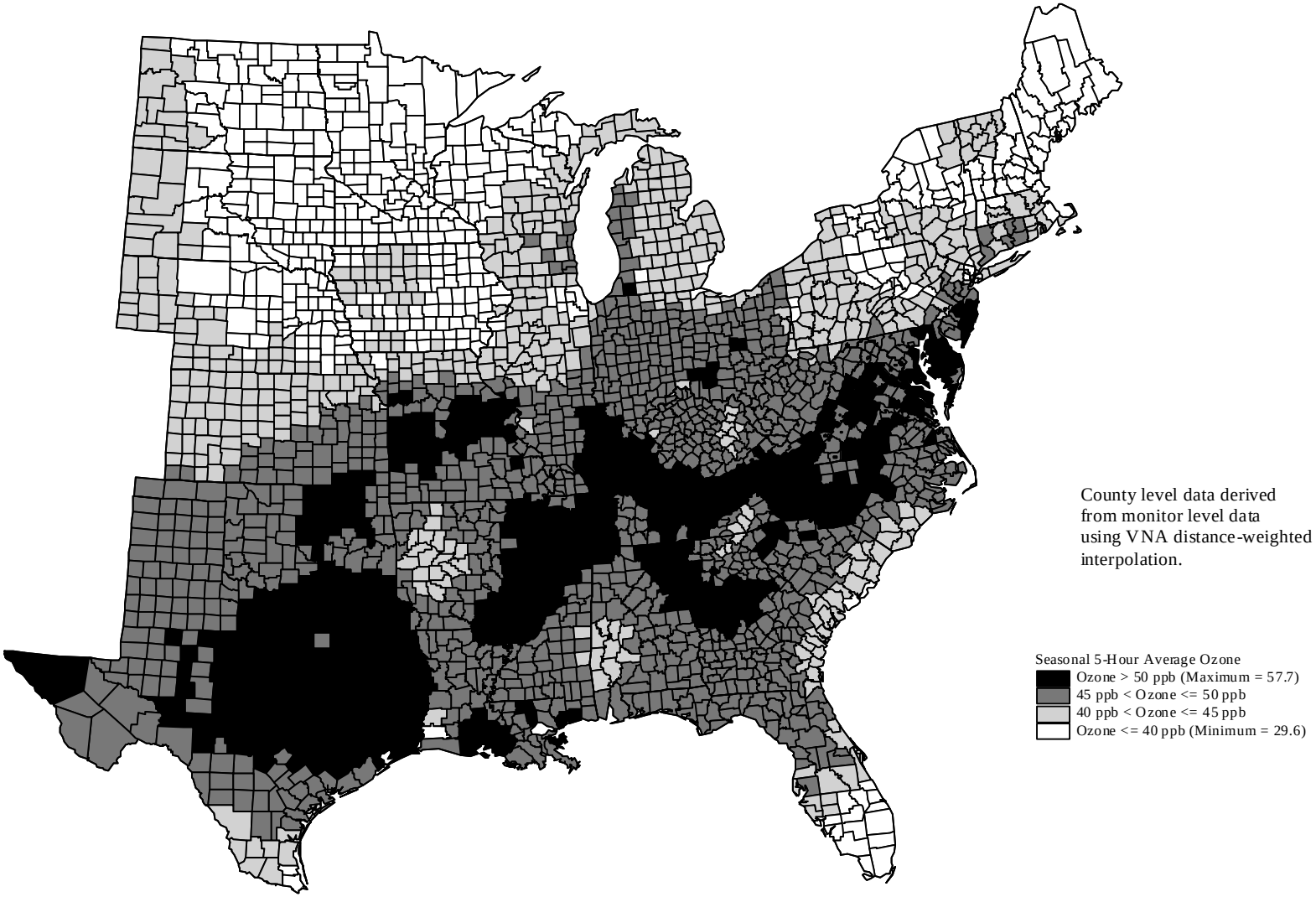


Exhibit B-5
Seasonal Average of the Daily One-Hour Maximum Ozone Level (April-October, 1997)

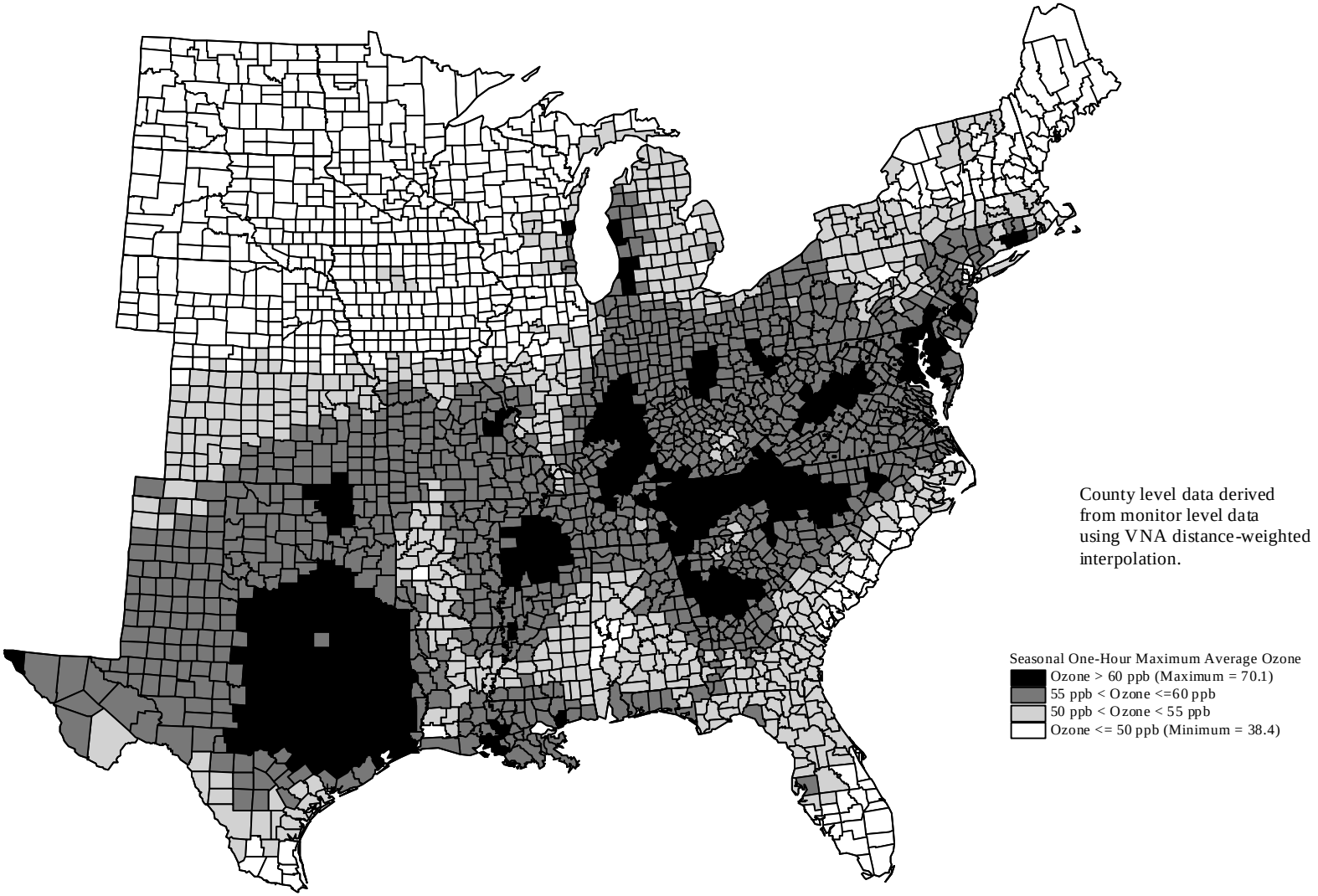
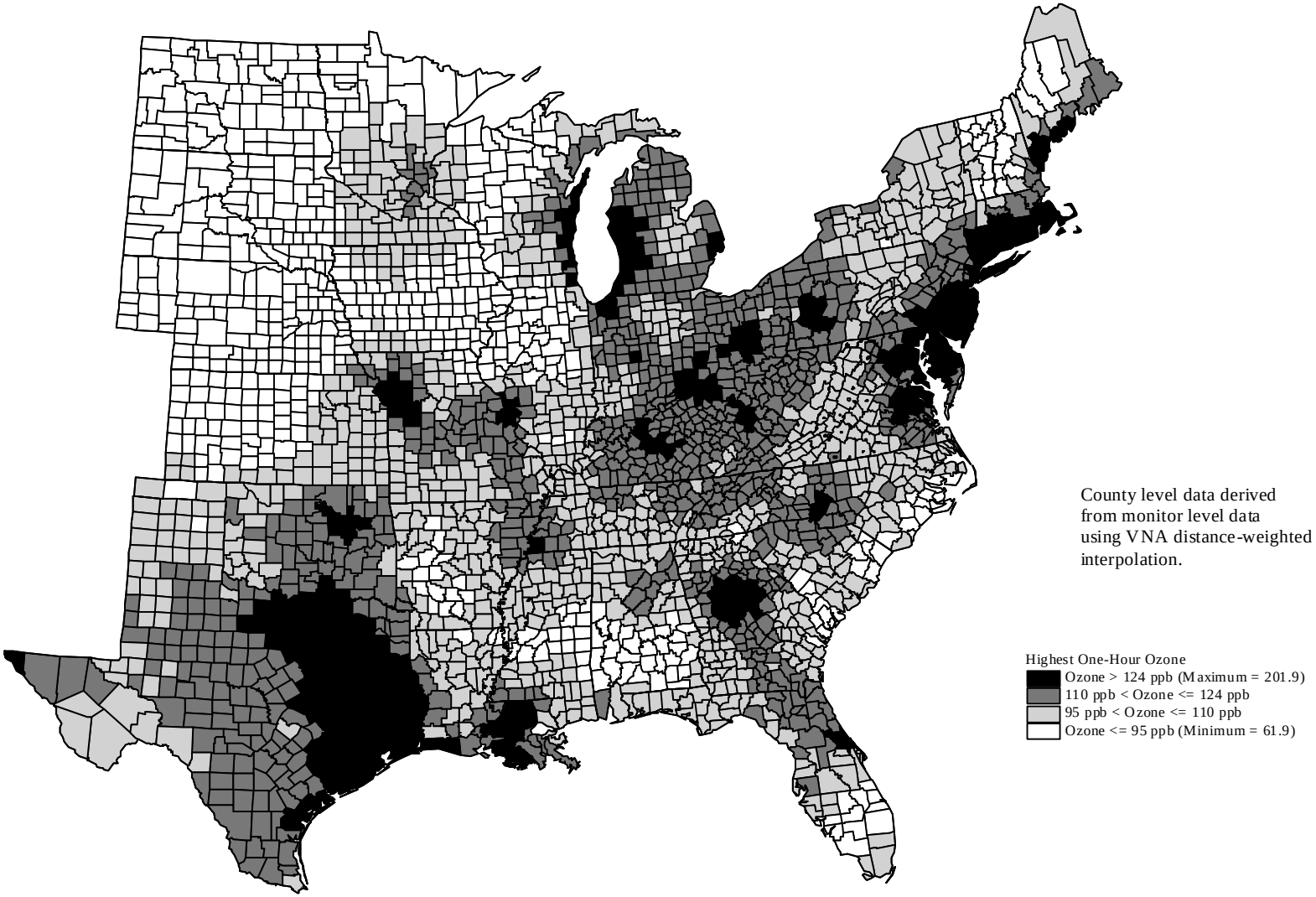


Exhibit B-6
One Hour Maximum Ozone (April-October, 1997)



APPENDIX C CONCENTRATION-RESPONSE FUNCTIONS

C.1 HOSPITAL ADMISSIONS

C.1.1 Hospital Admissions for Asthma (Burnett et al., 1999, Toronto)

The C-R function to estimate the change in hospital admissions for asthma associated with daily changes in ozone is:

$$\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for asthma per person[26] = 4.75 E-6
β = ozone coefficient = 0.00250
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
σ_β = standard error of β = 0.000718

Coefficient Estimate (β). The estimated coefficient (β) is based on a 4.99 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0499. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0499)}{19.5} = 0.00250.$$

Standard Error (σ_β). The standard error (σ_β) was calculated using the t-value (t=3.48) (Burnett, 1999):

$$s_b = \frac{0.00250}{3.48} = 0.000718.$$

[26] Hospital admissions for asthma are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.451 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

C.1.2 Hospital Admissions for Obstructive Lung Disease (Burnett et al., 1999, Toronto)

The C-R function to estimate the change in hospital admissions for obstructive lung disease associated with daily changes in ozone is:

$$\Delta Obstructive\ Lung\ Disease\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for obstructive lung disease per person[27] = 5.76 E-6
β = ozone coefficient = 0.00303
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
σ_β = standard error of β = 0.00110

Coefficient Estimate (β). The estimated coefficient (β) is based on a 6.08 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0608. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0608)}{19.5} = 0.00303.$$

Standard Error (σ_β). The standard error (σ_β) was calculated using the t-value (t=2.74) (Burnett, 1999):

$$s_b = \frac{0.00303}{2.74} = 0.00110.$$

[27] Hospital admissions for obstructive lung disease (ICD-9 490-492, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.547 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

C.1.3 Hospital Admissions for Respiratory Infection (Burnett et al., 1999, Toronto)

The C-R function to estimate the change in hospital admissions for respiratory infection associated with daily changes in ozone is:

$$\Delta \text{ Respiratory Infection Admissions} = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for respiratory infection per person[28] = 1.56 E-5
β = ozone coefficient = 0.00198
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
σ_β = standard error of β = 0.000520

Coefficient Estimate (β). The estimated coefficient (β) is based on a 3.93 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0393. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0393)}{19.5} = 0.00198.$$

Standard Error (σ_β). The standard error (σ_β) was calculated using the t-value (t=3.80) (Burnett, 1999):

$$s_b = \frac{0.00198}{3.80} = 0.000520.$$

[28]Hospital admissions for respiratory infection (ICD-9 464, 466, 480-487, 494) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (1.485 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

C.1.4 Hospital Admissions for All Respiratory (Burnett et al., 1997, Toronto)

The C-R function to estimate the change in hospital admissions for all respiratory associated with daily changes in O_3 is:

$$\Delta All\,respiratory = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for all respiratory per person[29] = 2.58 E-5
β = O_3 coefficient = 0.00498
ΔO_3 = change in daily 12-hour average O_3 concentration (ppb)[30]
pop = population of all ages
σ_β = standard error of β = 0.00106

Coefficient Estimate (β). The estimated coefficient (β) is based on a relative risk of 1.059 due to a change of 11.50 ppb in the daily average for O_3 (Burnett et al., 1997, Tables 2 and 6). The coefficient is calculated as follows:

$$b = \frac{\ln(1.059)}{11.50} = 0.00498.$$

Standard Error (σ_β). The standard error (σ_β) was calculated using the t-value (t=4.71) (Burnett et al., 1997, Table 6)

$$s_b = \frac{.00498}{4.71} = 0.00106.$$

[29] Hospital admissions for all respiratory (464-466, 480-486, 490-494, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.452 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

[30] Burnett et al. (1997, Table 2 and p. 614) reported using the daytime average ozone level from 8 A.M. to 8 P.M.

C.1.5 Hospital Admissions for All Respiratory (Thurston et al., 1992, New York City)

$$\Delta \, all\, respiratory\, admissions = b \cdot \Delta O_3 \cdot pop,$$

where:
β = ozone coefficient (Thurston et al., 1992, Table 6)[31] = 1.37 E-8
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of all ages
σ_β = standard error of β (Thurston et al., 1992, Table 6) = 5.3 E-9

C.1.6 Hospital Admissions for All Respiratory (Thurston et al., 1992, Buffalo)

$$\Delta \, all\, respiratory\, admissions = b \cdot \Delta O_3 \cdot pop,$$

where:
β = ozone coefficient (Thurston et al., 1992, Table 6)[32] = 3.09 E-8
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of all ages
σ_β = standard error of β (Thurston et al., 1992, Table 6) = 1.65 E-8

C.1.7 Hospital Admissions for All Respiratory(Thurston et al., 1994, Toronto)

$$\Delta \, all\, respiratory\, admissions = b \cdot \Delta O_3 \cdot pop,$$

where:
β = ozone coefficient = 1.68 E-8
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population all ages
σ_β = standard error of β = 9.71 E-9 .

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[31]Ozone coefficient based on a model with sulfate and hydrogen ion. The ozone levels were estimated in ppm ; the ozone coefficient has been adjusted to reflect ppb. The ICD-9 codes considered in this analysis are: 466, 480-486, 490-493 (Thurston et al., 1992, Table 1).

[32]Ozone coefficient based on a model with sulfate and hydrogen ion. The ozone levels were estimated in ppm ; the ozone coefficient has been adjusted to reflect ppb. The ICD-9 codes considered in this analysis are: 466, 480-486, 490-493 (Thurston et al., 1992, Table 1).

Coefficient Estimate (β). Based on a linear model with $PM_{2.5}$, the one-hour maximum ozone coefficient comes from an estimated coefficient of 0.0404, which estimates admissions per ppb of ozone (Thurston et al., 1994, Table 3).[33] The population of Toronto was estimated to be 2.4 million (U.S. EPA, 1997, Table D-7). We estimated a coefficient estimating admissions per person per ppb of ozone as follows:

$$b = \frac{0.0404}{2{,}400{,}000} = 1.68E-8.$$

Standard Error (σ_β). The standard error (σ_β) was calculated in a similar fashion (Thurston et al., 1994, Table 3):

$$s_b = \frac{0.0233}{2{,}400{,}000} = 9.71E-9.$$

C.1.8 Hospital Admissions for Pneumonia (Moolgavkar et al., 1997, Minneapolis)

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in O_3 is:

$$\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person[34] = 5.30 E-5
β = O_3 coefficient = 0.00370
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β = 0.00103

Coefficient Estimate (β). The estimated coefficient (β) is based on a 5.7 percent increase in admissions due to a O_3 change of 15 ppb (Moolgavkar et al., 1997, Table 4 and p. 366); the model with a 130 df smoother was reported to be optimal (p. 368). This translates to a relative risk of 1.057. The coefficient is calculated as follows:

$$b = \frac{\ln(1.057)}{15} = 0.00370.$$

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[33]The 812 Retrospective analysis (U.S. EPA, 1997, Table D-7) used an ozone coefficient based on a model with PM_{10}.

[34] Hospital admissions for pneumonia (ICD-9 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.089)}{15} - \frac{\ln(1.057)}{15}\right)}{1.96} = 0.00101$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.057)}{15} - \frac{\ln(1.025)}{15}\right)}{1.96} = 0.00105$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00103.$$

C.1.9 Hospital Admissions for COPD (Moolgavkar et al., 1997, Minneapolis)

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in O_3 is:

$$\Delta\, COPD admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person[35] = 3.75 E-5
β = O_3 coefficient = 0.00274
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β = 0.00170

Coefficient Estimate (β). The estimated coefficient (β) is based on a 4.2 percent increase in admissions due to a O_3 change of 15 ppb (Moolgavkar et al., 1997, Table 4 and p. 366); the model with a 100 df smoother was reported to be optimal (p. 368). This translates to a relative risk of 1.042. The coefficient is calculated as follows:

$$b = \frac{\ln(1.042)}{15} = 0.00274.$$

[35] Hospital admissions for COPD (ICD-9 490-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.454 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.094)}{15} - \frac{\ln(1.042)}{15}\right)}{1.96} = 0.00166$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.042)}{15} - \frac{\ln(0.99)}{15}\right)}{1.96} = 0.00174$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00170.$$

C.1.10 Hospital Admissions for Pneumonia (Schwartz, 1994c, Minneapolis)

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in O_3 is:

$$\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person[36] = 5.30 E-5
β = O_3 coefficient = 0.00280
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β = 0.00172

Coefficient Estimate (β). Based on a model with ozone, the coefficient (β) is estimated from the relative risk (1.15) associated with a 50 ppb change in the daily average ozone level (Schwartz, 1994c, Table 4 and p. 369):

$$b = \frac{\ln(1.15)}{50} = 0.00280.$$

[36] Hospital admissions for pneumonia (ICD-9 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994c, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.36)}{50} - \frac{\ln(1.15)}{50}\right)}{1.96} = 0.00171$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.15)}{50} - \frac{\ln(0.97)}{50}\right)}{1.96} = 0.00174$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00172 .$$

C.1.11 Hospital Admissions for Pneumonia (Schwartz, 1994a, Birmingham)

$$\Delta\, pneumonia\, admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop ,$$

where:

y_0 = daily hospital admission rate for pneumonia[37] per person 65 and older = 5.30 E-5
β = ozone coefficient = 0.00262
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β = 0.00196

Coefficient Estimate (β). Based on a single pollutant model, the coefficient (β) is estimated from the relative risk (1.14) associated with a change in ozone exposure of 50 ppb (Schwartz, 1994a, Table 6):

$$b = \frac{\ln(1.14)}{50} = 0.00262 .$$

[37]Pneumonia hospital admissions (ICD-9 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves et al., 1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994a, Table 6):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.38)}{50} - \frac{\ln(1.14)}{50}\right)}{1.96} = 0.00195$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.14)}{50} - \frac{\ln(0.94)}{50}\right)}{1.96} = 0.00197$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00196.$$

C.1.12 Hospital Admissions for COPD (Schwartz, 1994a, Birmingham)

$$\Delta COPD admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for COPD[38] per person 65 and older = 3.75 E-5
β	= ozone coefficient = 0.00314
ΔO_3	= change in daily average ozone concentration (ppb)
pop	= population age 65 and older
σ_β	= standard error of β = 0.00317

Coefficient Estimate (β). Based on a single pollutant model, the daily average ozone coefficient (β) is estimated from the relative risk (1.17) associated with a change in ozone exposure of 50 ppb (Schwartz, 1994a, Table 7):

$$b = \frac{\ln(1.17)}{50} = 0.00314.$$

[38]COPD hospital admissions (ICD-9 490-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.454 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves et al., 1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994a, Table 7):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.60)}{50} - \frac{\ln(1.17)}{50}\right)}{1.96} = 0.00319$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.17)}{50} - \frac{\ln(0.86)}{50}\right)}{1.96} = 0.00314$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00317.$$

C.1.13 Hospital Admissions for Pneumonia (Schwartz, 1994b, Detroit)

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in O_3 is:

$$\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person[39] = 5.18 E-5
β = O_3 coefficient (Schwartz, 1994b, Table 4) = 0.00521
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β (Schwartz, 1994b, Table 4) = 0.0013

[39] Hospital admissions for pneumonia (ICD-9 480-486) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.627 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

C.1.14 Hospital Admissions for COPD (Schwartz, 1994b, Detroit)

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in O_3 is:

$$\Delta\, COPD admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person[40] = 3.05 E-5
β = O_3 coefficient (Schwartz, 1994b, Table 4) = 0.00549
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β (Schwartz, 1994b, Table 4) = 0.00205

C.1.15 Hospital Admissions for All Respiratory (Schwartz, 1996, Spokane)

$$\Delta\, all\ respiratory\ related\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory per person 65 and older[41] = 1.187 E-4
β = ozone coefficient = 0.00856
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β = 0.00432

[40] Hospital admissions for COPD (ICD-9 491-492, 494-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.369 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

[41] All respiratory hospital admissions (ICD-9 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a single pollutant model, the coefficient (β) is estimated from the relative risk (1.244) associated with a change in ozone exposure of 50 μg/m^3 (Schwartz, 1996, Table 3):[42]

$$b = \frac{\ln(1.244)}{\left(\frac{50}{1.96}\right)} = 0.00856.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1996, Table 3):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.544)}{50/1.96} - \frac{\ln(1.244)}{50/1.96}\right)}{1.96} = 0.00432$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.244)}{50/1.96} - \frac{\ln(1.002)}{50/1.96}\right)}{1.96} = 0.00433$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00432.$$

C.1.16 Hospital Admissions for All Respiratory (Schwartz, 1995, New Haven)

$$\Delta all\ respiratory\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory per person 65 and older[43] = 1.187 E-4
β = ozone coefficient = 0.00265
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β = 0.00140

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[42] A conversion of 1.96 μg/m^3 per ppb is assumed.

[43] All respiratory hospital admissions (ICD-9 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with PM_{10}, the coefficient (β) is estimated from the relative risk (1.07) associated with a change in ozone exposure of 50 μg/m^3 (Schwartz, 1995, Table 3 and p. 535):[44]

$$b = \frac{\ln(1.07)}{\left(\frac{50}{1.96}\right)} = 0.00265.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1995, Table 3).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.15)}{50/1.96} - \frac{\ln(1.07)}{50/1.96}\right)}{1.96} = 0.00144$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.07)}{50/1.96} - \frac{\ln(1.00)}{50/1.96}\right)}{1.96} = 0.00135$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00140.$$

C.1.17 Hospital Admissions for All Respiratory (Schwartz, 1995, Tacoma)

$$\Delta\, all\ respiratory\ related\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory conditions[45] per person 65 and older = 1.187 E-4
β = ozone coefficient = 0.00715
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population age 65 and older
σ_β = standard error of β = 0.00257

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[44] A conversion of 1.96 μg/m^3 per ppb is assumed.

[45] All respiratory hospital admissions (ICD-9 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with PM_{10}, the coefficient (β) is estimated from the relative risk (1.20) associated with a change in ozone exposure of 50 μg/m^3 (Schwartz, 1995, Table 6 and p. 535):[46]

$$b = \frac{\ln(1.20)}{\left(\frac{50}{1.96}\right)} = 0.00715.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1995, Table 6):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.37)}{50/1.96} - \frac{\ln(1.20)}{50/1.96}\right)}{1.96} = 0.00265$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.20)}{50/1.96} - \frac{\ln(1.06)}{50/1.96}\right)}{1.96} = 0.00248$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00257.$$

[46]A conversion of 1.96 μg/m^3 per ppb is assumed.

C.1.18 Hospital Admissions for Cardiac (Burnett et al., 1997, Toronto)

The C-R function to estimate the change in hospital admissions for cardiac associated with daily changes in O_3 is:

$$\Delta cardiac = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for cardiac per person[47] = 3.81 E-5
β = O_3 coefficient = 0.00531
ΔO_3 = change in daily 12-hour average O_3 concentration (ppb)[48]
pop = population of all ages
σ_β = standard error of β = 0.00142

Coefficient Estimate (β). The estimated coefficient (β) is based on a relative risk of 1.063 due to a O_3 change of 11.50 ppb (Burnett et al., 1997, Tables 2 and 5). The coefficient is calculated as follows:

$$b = \frac{\ln(1.063)}{11.50} = 0.00531.$$

Standard Error (σ_β). The standard error (σ_β) was calculated using the t-value (t=3.74) (Burnett et al., 1997, Table 5)

$$s_b = \frac{.00531}{3.74} = 0.00142.$$

[47] Hospital admissions for cardiac (410-414, 427-428) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (3.617 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

[48] Burnett et al. (1997, Table 2 and p. 614) reported using the daytime average ozone level from 8 A.M. to 8 P.M.

C.1.19 Hospital Admissions for Dysrhythmias (Burnett et al., 1999, Toronto)

The C-R function to estimate the change in hospital admissions for dysrhythmias associated with daily changes in ozone is:

$$\Delta Dysrhythmias\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for dysrhythmias per person[49] = 6.46 E-6
β = ozone coefficient = 0.00168
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
σ_β = standard error of β = 0.00103

Coefficient Estimate (β). The estimated coefficient (β) is based on a 3.34 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0334. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0334)}{19.5} = 0.00168.$$

Standard Error (σ_β). The standard error (σ_β) was calculated using the t-value (t=1.63) (Burnett, 1999):

$$s_b = \frac{0.00168}{1.63} = 0.00103.$$

[49] Hospital admissions for dysrhthmias (ICD-9 427) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.614 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data is from U.S. Bureau of the Census (1997, Table 14).

C.2 EMERGENCY ROOM VISITS

C.2.1 Emergency Room Visits for Asthma (Cody et al., 1992, Northern NJ)

$$\Delta asthma\,related\,ER\,visits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop\,,$$

where:
β = ozone coefficient (Cody et al., 1992, Table 6) = 0.0203
BasePop = baseline population in northern New Jersey[50] = 4,436,976
ΔO_3 = change in daily five-hour average ozone concentration (ppb)[51]
pop = population all ages
σ_β $_v$= standard error of β (Cody et al., 1992, Table 6) = 0.00717

C.2.2 Emergency Room Visits for Asthma (Weisel et al., 1995, Northern NJ)

$$\Delta asthma\,related\,ER\,visits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop\,,$$

where:
β = ozone coefficient = 0.0443
BasePop = baseline population in northern New Jersey[52] = 4,436,976
ΔO_3 = change in daily five-hour average ozone concentration (ppb)[53]
pop = population all ages
σ_β = standard error of β = 0.00723

[50]The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. (1992, Figure 1) presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

[51]The coefficients in the study were based on the five-hour (10:00 am to 2:59 pm) ozone average in ppm; they have been converted to ppb.

[52]The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. (1992, Figure 1) presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

[53]The coefficients in the study were based on the five-hour (10:00 am to 2:59 pm) ozone average in ppm; they have been converted to ppb.

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Weisel et al. (1995, Table 2) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{s_{b_i}^2}} \right) = 0.0443.$$

Standard Error (σ_β). The standard error of the coefficient (σ_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{s_{b_i}^2}} \right) = \left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{g} \right) = \sum_{i=1986}^{1990} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g} \right).$$

This eventually reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00723.$$

C.2.3 Emergency Room Visits for Asthma (Stieb et al., 1996, New Brunswick)

$$\Delta asthma\,related\,ER\,visits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop\ ,$$

where:

β = ozone coefficient (Stieb et al., 1996, Table 2 linear model) = 0.0035
BasePop = baseline population in Saint John, New Brunswick (Stieb et al., 1996, p. 1354) = 125,000
ΔO_3 = change in the daily one-hour maximum ozone concentration (ppb)[54]
pop = population all ages
σ_β = standard error of β (Stieb et al., 1996, Table 2 linear model) = 0.0018

[54]The coefficients in the study were based on the five-hour ozone average in ppm; they have been converted to ppb. The five-hour period is defined as 10:00 am to 2:59 pm.

C.3 ACUTE MORBIDITY

C.3.1 Any of 19 Respiratory Symptoms: Krupnick (1990)

Krupnick et al. (1990) estimated the impact of coefficient of haze (COH), ozone and SO_2 on the incidence of any of 19 respiratory symptoms or conditions, including head cold, cough with phlegm, and doctor-diagnosed flu; this complex of 19 symptoms or conditions is termed ARD2.[55]

The C-R function used to estimate the change in ARD2 associated with a change in daily one-hour maximum ozone is based on Krupnick et al. (1990, p. 12):[56]

$$\Delta ARD2 \cong b^{*}_{PM_{10}} \cdot \Delta O_3 \cdot pop\,,$$

where:

β^* = first derivative of the stationary probability = 0.000137
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)[57]
pop = population aged 18-65 years old[58]
σ_β = standard error of β^* = 0.0000697

Coefficient Estimate (β^*). The logistic regression model used by Krupnick et al. (1990) takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$probability(ARD2 \mid sickness\,or\,not_{t-1}) = p_i = \frac{1}{1 - e^{b_0 + b_1 \cdot ARD2_{t-1} + X \cdot b}}\,,\ for\ i = 0,1.$$

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[55]It is not clear what the 19 symptoms or conditions are. Krupnick et al. (1990, p. 3) listed 13 "symptoms or conditions" in the definition of ARD1: head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. ARD1 is a subset of ARD2. The other symptoms or conditions are not specified clearly.

[56]Krupnick and Kopp (1988, p. 2-24) and ESEERCO (1994, p. V-32) used the same C-R functional form as that used here.

[57]Krupnick et al. (1990) used parts per hundred million (pphm) to measure ozone; the coefficient used here is based on ppb.

[58]This analysis uses the coefficient estimates based on the sample of "adults," and assumes that individuals 18 and older were considered adult. According to Krupnick et al. (1990, Table 1), about0.6 percent of the study sample was over the age of 60. This is a relatively small fraction, so it is further assumed that the results do not apply to individuals over the age of 65.

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of ozone (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in ozone and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before} .$$

However the full suite of coefficient estimates are not available.[59] Rather than use the full suite of coefficient values, the impact of ozone on the probability of probability of ARD2 may be approximated by the derivative of ARD2 with respect to ozone:[60]

$$\frac{\partial probability(ARD2)}{\partial O_3} = \frac{p_0 \cdot (1 - p_1) \cdot b \cdot \left[p_1 + (1 - p_0)\right]}{(1 - p_1 + p_0)^2} = b^* ,$$

where β is the reported logistic regression coefficient for ozone. The change in the incidence of ARD2 associated with a given change in ozone is then estimated by:

$$\frac{\partial ARD2}{\partial O_3} \cong \frac{\Delta ARD2}{\Delta O_3}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta O_3} \cong b^*$$

$$\Rightarrow \Delta ARD2 \cong b^* \cdot \Delta O_3 .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO (1994, p. V-32), for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

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[59]The model without NO_2 (i.e., Krupnick et al., Table V, equation 3) was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., Table V). Both the ozone and COH coefficients dropped by about a factor of two or more.

[60]The derivative result is reported by Krupnick et al. (1990, p. 12).

$$b^* = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00055 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000137.$$

Standard Error (σ_β). The standard error for the coefficient (σ_β) is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. (1990, Table V):

$$b_{high} = 0.00055 + (1.96 \cdot 0.00027) = 0.00108$$

$$\Rightarrow b^*_{high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00108 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000268$$

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{(0.000268 - 0.000137)}{1.96} = 0.0000668$$

$$b_{low} = 0.00055 - (1.96 \cdot 0.00027) = 0.0000208$$

$$\Rightarrow b^*_{low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.0000208 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 5.17 \cdot 10^{-6}$$

$$\Rightarrow s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(0.000137 + 5.17 \cdot 10^{-6}\right)}{1.96} = 0.0000725$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.0000697.$$

C.3.2 Minor Restricted Activity Days: Ostro and Rothschild (1989)

Ostro and Rothschild (1989) used a log-linear regression to estimate the impact of O_3 on the incidence of minor restricted activity days (MRAD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled, as discussed below.

The C-R function to estimate the change in the number of MRADs associated with a change in daily O_3 is:

$$\Delta MRAD = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily MRAD daily incidence rate per person[61] = 0.02137
β = inverse-variance weighted O_3 coefficient = 0.00220
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)[62]
pop = adult population aged 18 to 65[63]
σ_β = standard error of β = 0.000658

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild (Ostro et al., 1989, Table 4) using the inverse of the variance as the weight:[64]

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.00220.$$

[61]The annual incidence rate (7.8) provided by Ostro and Rothschild (1989, p. 243) was divided by 365 to get a daily rate of 0.02137.

[62]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation. The study used ozone measurements in $\mu g/m^3$; a conversion of 1.96 $\mu g/m^3$ = 1 ppb is assumed here.

[63]The study is based on a "convenience" sample of individuals aged 18-65. Applying the C-R function to this age group is likely to produce a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. A number of studies have found that hospital admissions for the elderly are related to ozone exposure (e.g., Schwartz, 1994b; Schwartz, 1994c).

[64]The calculation of the MRAD coefficient and its standard error is exactly analogous to the calculation done for the work-loss days coefficient based on Ostro (1987).

Standard Error (σ_β). The standard error of the coefficient (σ_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g}\right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g}\right).$$

This reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.000658.$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.0158.$$

C.3.3 Shortness of Breath: Ostro et al. (1995)

The C-R function to estimate the change in shortness of breath days is:

$$\Delta Shortness of\ Breath = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta O_3 \cdot b} + y_0} - y_0\right] \cdot pop,$$

where:

y_0 = daily shortness of breath incidence rate per person (Ostro et al., 1995, p. 715) = 0.056
β = estimated O_3 logistic regression coefficient = 0.00383
ΔO_3 = change in daily one-hour maximum O_3 concentration (ppb)
pop = asthmatic African-American population[65] ages 7 to 12 = 6.91% of African-American population ages 7 to 12
σ_β = standard error of β = 0.00186

[65]Adams (1995, Table 57) reported that in 1994, 6.91% of individuals under the age of 18 have asthma. It has been reported that African-Americans have a higher prevalence of asthma (e.g., see U.S. EPA, 1996c). Ostro et al. (1995, p. 711) noted that "Although prevalence is only somewhat greater among African-Americans than among whites, rates of morbidity are markedly higher." Indeed, the asthma rates for whites and African-Americans were almost identical in 1994 (1995, Table 59), so no correction is made to the estimated prevalence rate for asthma in African-Americans.

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio of 1.36 (Ostro et al., 1995, Table 3) associated with a change in mean O_3 of 80.2 ppb (Ostro et al., 1995, Table 2).[66] The coefficient is calculated as follows:

$$b = \frac{\ln(1.36)}{(80.2)} = 0.00383.$$

Standard Error (σ_β). The standard error for the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (Ostro et al., 1995, Table 2):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.83)}{80.2} - \frac{\ln(1.36)}{80.2}\right)}{1.96} = 0.00189$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.36)}{80.2} - \frac{\ln(1.02)}{80.2}\right)}{1.96} = 0.00183$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00186.$$

C.3.4 Asthma Attacks: Whittemore and Korn (1980)

$$\Delta asthma\,attacks = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta O_3 \cdot b} + y_0} - y_0\right] \cdot pop,$$

where:

y_0 = daily incidence of asthma attacks = 0.027 (Krupnick, 1988, p. 4-6)[67]
β = ozone coefficient = 0.00184
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of asthmatics of all ages = 5.61% of the population of all ages (Adams et al., 1995 Table 57).
σ_β = standard error of β = 0.000714

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[66]The results in the study have been converted from parts per hundred million to parts per billion.

[67]The annual rate of 9.9 asthma attacks per asthmatic is divided by 365 to get a daily rate. A figure of 9.9 is roughly consistent with the recent statement that "People with asthma have more than 100 million days of restricted activity" each year (National Heart, 1997). This 100 million incidence figure coupled with the 1996 population of 265,557,000 (U.S. Bureau of the Census, 1997, Table 2) and the latest asthmatic prevalence rate of 5.61% (Adams et al., 1995, Table 57), suggest an annual asthma attach rate per asthmatic of 6.7.

Coefficient Estimate (β). Based on a model with particulate matter, the daily one-hour ozone coefficient is based on an oxidant coefficient (1.66) estimated from data expressed in ppm (Whittemore et al., 1980, Table 5):[68]

$$b = \frac{1.66 \cdot 1.11}{1000} = 0.00184 .$$

Standard Error (σ_β). The standard error (σ_β) is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

$$s_b = \frac{b}{t} = \frac{0.184}{2.576} = 0.000714 .$$

[68]The study used oxidant measurements in ppm (Whittemore et al., 1980, p. 688); these have been converted to ozone measurements in ppb, assuming ozone comprises 90% of oxidants (i.e., 1.11*ozone=oxidant). It is assumed that the harm of oxidants is caused by ozone. The view expressed in the Ozone Staff Paper (U.S. EPA, 1996c, p.164) is consistent with assuming that ozone is the oxidant of concern at normal ambient concentrations: "Further, among the photochemical oxidants, the acute-exposure chamber, field, and epidemiological human health data base raises concern only for O_3 at levels of photochemical oxidants commonly reported in ambient air. Thus, the staff recommends that O_3 remain as the pollutant indicator for protection of public health from exposure to all photochemical oxidants found in the ambient air."