Sonoma Technology, Inc.

1360 Redwood Way, Suite C
Petaluma, CA 94954-1169
707/665-9900
FAX 707/665-9800
www.sonomatech.com

ASSESSMENT OF THE HEALTH BENEFITS OF IMPROVING AIR QUALITY IN HOUSTON, TEXAS

FINAL REPORT
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By:
Frederick W. Lurmann[a]
Jane V. Hall[b]
Michael Kleinman[c]
Lyle R. Chinkin[a]
Victor Brajer[b]
Dianne Meacher[c]
Francis Mummery[b]
Richard L. Arndt[a]
Tami H. Funk[a]
Siana H. Alcorn[a]
Naresh Kumar[a]

[a]Sonoma Technology, Inc.
1360 Redwood Way, Suite C
Petaluma, CA 94954-1169

[b]Institute for Economic and Environmental Studies
California State University
Fullerton, CA 92634

[c]Department of Community and Environmental Medicine
University of California
Irvine, CA 92717

Prepared for:
City of Houston
Office of the Mayor
P.O. Box 1562
Houston, TX 77251

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EXECUTIVE SUMMARY

ES-1. INTRODUCTION

ES-1.1 BACKGROUND

Unhealthful air quality conditions in the Houston area are a public health concern. The concentrations of ozone in ambient air are greater than the health-based National Ambient Air Quality Standards (NAAQS) on 30 to 50 days per year. While significant progress has been made in reducing ozone concentration in the last twenty years, the rate of improvement in ozone air quality is slower than in other major urban areas such as Los Angeles. Recent monitoring information indicates the concentrations of fine particulate matter ($PM_{2.5}$) are likely to exceed the new annual $PM_{2.5}$ NAAQS in Houston. Fine particulate matter includes the mass of particles with aerodynamic diameters less than 2.5 μm. There is also concern for human exposure to other hazardous air pollutants (HAPs) that are not covered by NAAQS.

Improving air quality conditions is a difficult and usually expensive undertaking. Emission reductions are needed on an on-going basis just to offset emission increases associated with population growth and economic growth in most urban areas. The Texas Natural Resource and Conservation Commission (TNRCC) performed extensive analysis of the Houston area ozone problem and modeled potential mitigation strategies for the 1998 State Implementation Plan (SIP). The TNRCC found that large emissions reductions are needed to achieve compliance with the 1-hr ozone standard. They estimated that an 85 percent reduction in nitrogen oxides (NO_x) emissions and 15 percent reduction in volatile organic compound (VOC) emissions would be needed to achieve compliance with the 1-hr standard by the year 2007. Such large reductions in region-wide NO_x emissions would probably require changes in the emission control technologies employed and possibly the fuels used in most stationary and mobile combustion sources. Emissions from cars, trucks, buses, trains, ships, power plants, refineries, chemical plants, and other commercial and industrial facilities may have to be reduced significantly to meet these reduction targets. Attainment of the new 8-hr ozone standard may be more difficult than attainment of the older 1-hr standard.

Characterization of the $PM_{2.5}$ problem is in its early stages and much work is needed to understand the source contributions and options for mitigation of the problem. Ambient $PM_{2.5}$ in the Houston area comes from local primary PM emission sources, local gaseous VOC, NO_x, sulfur dioxide (SO_2), and ammonia (NH_3) emissions sources that react to make secondary $PM_{2.5}$, and regional-scale transport of $PM_{2.5}$ from sources outside of the region. The relative contributions of these sources are not well known at this time. However, it is important to note that there are some commonalities in the precursor emissions for ozone, secondary $PM_{2.5}$, and HAPs. VOCs and NO_x emissions contribute to both secondary $PM_{2.5}$ and ozone. VOC and primary PM emissions also contribute to the ambient concentrations of HAPs.

Urban air pollution is a mixture of many unhealthful chemicals. As the understanding of the urban air pollution problem expands, there is greater recognition of the need to develop

control strategies using "integrated multi-species approaches". A key air quality concern is how emissions changes will affect the whole mixture of particles and gases in the urban atmosphere. Likewise, there is concern for the public health risks associated with human exposure to this mixture of compounds. In fact, numerous health effects studies find more consistent evidence for associations of human health effects with urban air pollution as a whole than with the individual chemical components of urban air pollution.

Achieving compliance with the NAAQS may be disruptive and expensive. In order to obtain broad-based support for control plans, the approach to compliance needs to be practical, based on the best available science, and optimized to meet the public health objectives. The regulatory authorities responsible for air quality resource management face challenging questions about how best to achieve compliance with the Federal Clean Air Act requirements. In addition to uncertainties regarding emissions and air quality, little is known regarding the health benefits of different approaches for compliance in the Houston area. For several years, the **H**ouston **A**ir e**X**cellence and **L**eadership (HAXL) group has expressed strong interest in assessment of the health benefits of alternate and more holistic air quality control strategies. This study was commissioned by the City of Houston, partially funded by the U.S Environmental Protection Agency (EPA), and guided by the HAXL committee to address the concerns regarding the health benefits of alternate air pollution control programs.

Detailed control plans have not been fully developed to address the ozone problem in the Houston area. No control strategy has been developed for $PM_{2.5}$ since the Federal regulatory deadlines for having plans in place are after 2005. The 1998 SIP does not list the specific control measures that would have to be adopted to meet the large emissions reduction targets. The absence of defined control plans provides an opportunity for careful selection of the best control plans to deal with both the ozone and $PM_{2.5}$ problems in the area.

ES-1.2 GOALS OF THIS STUDY

The overall purpose of this study is to provide information that will assist decision-makers in setting priorities for emissions reductions based on the relative health benefits of different emission control strategies. The specific objectives are to:

- Identify and economically value the health benefits of achieving compliance with the ozone and $PM_{2.5}$ NAAQS.
- Identify and economically value the health benefits of alternate control strategies that would make progress towards compliance with the ozone and $PM_{2.5}$ NAAQS.
- Investigate relationships between ozone and $PM_{2.5}$ control strategies.
- Explore the health benefits of reductions in selected toxic air contaminants that arise from emissions of substances that also contribute to ozone and/or $PM_{2.5}$.
- Identify control strategies that maximize health-related economic benefits.

An approach was developed to address these objectives with existing data, models, and knowledge for the Houston area. The approach involved:

- Compiling air pollution emission inventory information for baseline conditions (1993-1995) and projected future-year (2007) conditions for ozone and PM precursor compounds.
- Defining a range of alternate emission control scenarios.
- Characterizing the air pollution concentrations to which the Houston area residents were exposed in a baseline period (based on observed air quality data) and the concentrations to which the Houston area residents would be exposed in the future under alternate scenarios (based on the projected emission inventories and air quality models).
- Reviewing air pollution / human health effects literature and selecting the most scientifically sound air pollution exposure / human health effects response functions.
- Estimating the health effects (symptoms) that the Houston area residents would experience under alternate control scenarios.
- Reviewing the economics literature and selecting the most appropriate dollar values for the air pollution-related health effects.
- Estimating the economic value of the health effects that the Houston area residents would experience under alternative control scenarios. A comparative analysis was conducted using the morbidity and mortality results for ozone and $PM_{2.5}$.

The methods and results from each of the major elements of the study are summarized below.

ES-2. EMISSION INVENTORIES

Emission inventories are needed to estimate the likely changes in air quality conditions between the baseline period (1993) and the future year adopted for planning purposes (2007). Baseline and future-year emission inventories were compiled for the compounds influencing ozone and $PM_{2.5}$ concentrations. These include emissions of VOCs, NO_x, CO, SO_2, NH_3, and PM. The geographic area inventoried included the City of Houston, Harris County, and the 17 surrounding counties. The emission estimates were based on information obtained from the TNRCC's revised ozone SIP modeling database and the EPA's National Particulate Inventory (NPI). The projections for the 2007 base case (Scenario 1) account for the effects of currently adopted control programs and estimated growth. The growth factors were based on the EPA's Economic Growth and Analysis System (E-GAS) which provides region-specific growth projections for economic factors, employment, energy production and consumption, and demographic factors.

Forecasts of future-year emissions are inherently uncertain. Concerns arise for the accuracy of the base-period inventory, the estimated effects of current control programs, and

the growth projections. Our analysis included an investigation of the sensitivity of the 2007 emission estimates to different assumptions regarding growth, transportation sources, and control measures. The principal emission scenarios considered in our analysis are:

- Scenario 1 – the 2007 base-case inventory developed from E-GAS projections.
- Scenario 2 – the lower growth rate 2007 inventory. This inventory is the same as Scenario 1 except it was developed using the lower of E-GAS growth factors or the population growth rate. It reflects slower growth of the fastest growing subcategories of emissions.
- Scenario 3 – the higher mobile source and lower dust source inventory. This inventory is the same as Scenario 1 except it reflects recent findings from the Northern Front Range Air Quality Study (NFRAQS) regarding possible biases in the baseline PM emission inventories.
- Scenario 4 – full implementation of a clean diesel fuel program. This inventory is the same as Scenario 1 except it assumes a new control program requiring cleaner burning diesel fuel (similar to that in California) is fully implemented for mobile and stationary diesel-fueled sources by 2007.
- Ozone SIP Scenario – implementation of region-wide emission control measures sufficient to reduce NO_x emissions by 85 percent and VOC emissions by 15 percent in 2007. The PM, SO_2, and NH_3 emissions are the same as Scenario 1.

Table ES-1 summarizes the emission estimates for the 1993 baseline period and for 2007 under these scenarios. Comparison of the 2007 Scenario 1 emissions with 1993 emissions indicates that reductions in NO_x and VOC emissions, and increases in SO_2, NH_3, and $PM_{2.5}$ emissions are projected to occur without new control measures. These trends are consistent with the historical focus on ozone precursor emissions (NO_x and VOC) in the Houston area. Scenario 2 has slightly lower emissions for all species, while Scenario 3 has significantly lower PM emissions. The clean diesel fuel control measure (Scenario 4) would reduce emissions of NO_x, VOC, PM, and SO_2, while the Ozone SIP control measures would primarily reduce emissions of NO_x and VOCs. In addition, future-year cases with partial implementation of the Ozone SIP control program's NO_x reductions were considered.

ES-3. AIR QUALITY CONDITIONS

Baseline air quality conditions were characterized using measurements from the Houston area air-monitoring network. Baseline ambient ozone concentrations were derived from hourly ozone measurements for 1993-1995. Baseline $PM_{2.5}$ conditions were derived from 24-hr average measurements made every sixth day from March 1997 to March 1998. Annual average concentrations of selected HAPs were determined from 24-hr average measurements made between 1992 and 1997 in the Houston area. The ambient ozone and $PM_{2.5}$ data were spatially mapped to 4 km x 4 km grids for use in the population exposure modeling. Future-year air quality conditions were projected for ozone and $PM_{2.5}$. The spatial coverage of the

HAPs data was insufficient to support spatial mapping over the greater Houston area. Future-year projection of HAP concentrations were not compiled because of the large uncertainties in their emissions and the limited means that we have to quantify the health benefits of reductions in HAPS.

Projections of future-year ozone conditions were developed using photochemical modeling and rollback assumptions. The effects of changes in NO_x, VOC, and CO emissions on ozone were simulated using the grid-based CAMx photochemical model and the meteorological conditions of the September 6-11, 1993 ozone episode that was used in the Ozone SIP modeling. Future base-case peak ozone concentrations are estimated to be only slightly lower than 1993 conditions under Scenario 1. With a clean diesel fuel program (Scenario 4), peak ozone levels are estimated to be about 4 percent lower in 2007 than for Scenario 1. Modeling results with new mobile source and biogenic emissions information (provided by TNRCC) confirmed the 1998 SIP finding that emission reductions of about 85 percent for NO_x and 15 percent for VOC were needed to reduce the peak ozone levels enough to achieve compliance with the 1-hr NAAQS. Modeling calculations made with lower NO_x emission reductions (25, 45, 65, and 75 percent instead of 85 percent) did not show sufficient reductions in peak ozone concentrations to achieve NAAQS compliance.

For quantification of health benefits, we considered ozone concentrations on all days in a 3-year period with moderate or high concentrations, not just on the three or four worst days of a year. We applied the average relative changes in peak hourly ozone determined from the CAMx photochemical model simulations of the September episode to the concentration patterns on 336 days in 1993-1995 to estimate the future-year concentration patterns under different scenarios.

The 1997-1998 $PM_{2.5}$ data were projected backwards (to 1993) and forward (to 2007) using emissions information and the relatively simple speciated PM rollback model. Insufficient air quality and emissions information was available for the Houston area to support the application of more sophisticated and detailed grid-based PM models. Recall that the $PM_{2.5}$ NAAQS is a standard for the mass of $PM_{2.5}$ particles in ambient air. $PM_{2.5}$ is a complex mixture of many chemical species including sulfate, nitrate, ammonium, organic material, elemental carbon, water, and crustal material (or dirt). Virtually all $PM_{2.5}$ models must build up estimates of $PM_{2.5}$ mass from estimates of the chemical components. The speciated rollback model associates changes in specific chemical components of $PM_{2.5}$ with changes in the emissions that produce those chemical components. The speciated rollback model also takes into account that there are significant background concentrations of $PM_{2.5}$ contributed by transport from sources outside the region of interest. The rollback model assumes there are linear relationships between local emissions and ambient $PM_{2.5}$ concentration in excess of the background $PM_{2.5}$ concentrations. For example, the 85 percent NO_x and 15 percent VOC emission reductions in the 1998 Ozone SIP are estimated to reduced the $PM_{2.5}$ ammonium nitrate (in excess of the background) by 85 percent and reduce the secondary $PM_{2.5}$ organic material (in excess of the background) by 15 percent.

Compliance with the $PM_{2.5}$ NAAQS in the Houston area will be determined by the highest annual average concentration measured at any monitoring station. **Table ES-2** shows the estimated annual average $PM_{2.5}$ mass concentrations under different scenarios. These estimates were made using an 11 $\mu g/m^3$ background $PM_{2.5}$ concentration, which is a fairly high background level, and a large fraction of the 15 $\mu g/m^3$ annual NAAQS. This high background level is based on the 1997-1998 $PM_{2.5}$ measurements and indicates that sources outside of the study region make significant contributions. The $PM_{2.5}$ projections show slightly higher concentrations in 2007 under Scenarios 1, 2, and 3 (18.8 to 19.0 $\mu g/m^3$) than in 1997-1998 (18.3 $\mu g/m^3$) or in 1993 (17.9 $\mu g/m^3$). With implementation of a clean diesel program or the Ozone SIP control program, the annual average $PM_{2.5}$ concentrations are estimated to decrease to 18.6 $\mu g/m^3$ in 2007. Given the high background levels, substantial reductions in local $PM_{2.5}$ precursor emissions will be needed to reach the standard.

ES-4. ASSESSMENT OF POPULATION AIR POLLUTION EXPOSURE

To quantify the health benefits of air quality improvements, we need to know the population's air pollution exposure in the baseline and future-year period. For an annual assessment, we need to know not only the distribution of air pollutant concentrations but also how many people were exposed to those concentrations and for how many days. Thus, the primary measure of exposure for this study is the number of person-days of exposure to concentrations above the NAAQS. For annual average exposures, this reduces to the number of people exposed to concentrations above the NAAQS.

A spatially resolved, grid-based approach was used to estimate the exposure of Houston area residents in this study. Population data and ambient air quality data were compiled on 4 km x 4 km grids covering the Houston area. The residents living within 35 km of ozone monitoring stations were included in the study. The total population considered in 1993 and 2007 were 3.76 million and 4.81 million. Over 93 percent of the residents living in the 180 km x 188 km region centered on Houston were included.

The Regional Human Exposure (REHEX-II) Model was applied to estimate the population exposure to ozone and $PM_{2.5}$ in the baseline period and future-year periods under a variety of scenarios. The population's exposure to ozone was estimated with and without consideration of the modifying effects of buildings on exposure. Human beings spend most of their lives inside of buildings, and concentrations of ozone and $PM_{2.5}$ are usually lower indoors than outdoors (in the absence of indoor sources of $PM_{2.5}$). Indoor-outdoor differences are generally smaller for $PM_{2.5}$ than ozone. We expect Houston area residents spend more time in air-conditioned buildings than residents of other cities. Air-conditioned buildings tend to have very low ozone concentrations. The differences between ambient ozone concentrations and the actual personal exposure concentrations are probably larger, on average, in the Houston area than in others because of the extensive use of air conditioning.

Despite the recognized effects of buildings on exposure concentrations, we have quantified the benefits of improved air quality based on "all outdoor" exposures estimates.

The reason for this is that health effect studies (epidemiologic studies) used "all outdoor exposure" as a surrogate for actual exposure. In order to make the quantitative linkage between exposure and health effects for these pollutants, the exposure assessment must quantify population exposure to ambient air pollutant concentrations.

The key results from this assessment are estimates of the population exposure to outdoor pollution levels in 1993 and in 2007 under different scenarios. These key results are as follows:

- There are almost three times as many exposures to levels above the 8-hr NAAQS as above the 1-hr NAAQS in the Houston area. The "all outdoors" ozone exposure estimates for 1993-1995 indicate that residents experienced 26.2 million person-days of exposure to 1-hr maximum concentrations above the 120 ppb NAAQS and 72.6 million person-days of exposure to 8-hr maximum concentrations above the 80 ppb NAAQS.
- As shown in **Figure ES-1**, there are 26.7 million person-days of exposure to 1-hr maximum concentrations above the 120 ppb NAAQS. Seven alternative scenarios were considered for ozone in 2007. The 2007 base-case has about the same number of exposures as the 1993 case. The results for the "all outdoors" control cases are as follows. Implementation of a 15 percent VOC emissions reduction and a 25 percent NO_x emissions reduction (across the board) is estimated to reduce the number of person-days of exposure to 1-hr maximum concentrations above 120 ppb from 26.7 million to 24.2 million. Full implementation of a clean diesel fuel program on top of the 15 percent VOC emissions reductions is estimated to be slightly more effective than the 25 percent NO_x reduction: 22.9 million compared to 24.2 million person-days of exposure. NO_x emissions reductions of 45, 65, 75, and 85 percent are estimated to reduce the number of person-days of exposure to 1-hr maximum concentrations above 120 ppb to 19.6, 10.1, 3.8, and 0.24 million per year, respectively.
- The future-year "all outdoors" exposure estimates for the daily 8-hr maximum ozone concentrations indicate there would be 84.2 million person-days per year of exposures to 8-hr maximum concentrations above 80 ppb. The number of 8-hr exposures above the standard are estimated to be 16 percent higher in the 2007 base case than in 1993-1995. The air quality improvements incorporated into the 2007 base case are not sufficient to offset the 28 percent population growth. These results confirm the general belief that achieving compliance with the 8-hr standard will be more difficult than with the 1-hr standard.
- The 8-hr exposure results for the 85 percent NO_x emission reduction case show the estimated person-days of exposure to 8-hr ozone concentrations above 80 pbb decrease from 84 million to 11 million per year in 2007. Achieving compliance with the 1-hr NAAQS reduces the 2007 exposures above the level of the 8-hr NAAQS by about 87 percent, which is a large improvement. However, it still may not be enough improvement to fully mitigate the 8-hr exposures of concern.
- As shown in **Figure ES-2**, the estimated portion of the population exposed to annual average $PM_{2.5}$ above the 15 $\mu g/m^3$ annual NAAQS is 62 percent in 1993 (or 2.36

million people) and 79 percent in 2007 (or 3.85 million people) without additional control measures. The spatial pattern of annual $PM_{2.5}$ exposures above 15 $\mu g/m^3$ is similar to the pattern for ozone exposures above 120 ppb. The estimated portion of the population exposed to more than 15 $\mu g/m^3$ of $PM_{2.5}$ mass under the Ozone SIP Scenario is 74 percent or 3.5 million people. Full implementation of the clean diesel fuel control scenario is estimated to have about the same effect as the Ozone SIP control program, which is an 11 percent reduction in the number of people exposed to concentrations greater than 15 $\mu g/m^3$.

- All of the $PM_{2.5}$ 24-hr average exposures are well below the 65 $\mu g/m^3$ NAAQS. For the 1993 base case, there are approximately 51 million person-days of exposures per year to daily concentrations above 30 $\mu g/m^3$. For the 2007 base cases (Scenarios 1-3), the estimates are 115 to 125 million person-days of exposures per year to daily concentrations above 30 $\mu g/m^3$.
- The gaseous VOCs of principal concern for toxicity in the Houston area are benzene, 1,3-butadiene, and formaldehyde. The maximum annual concentrations between 1992 and 1997 were 3.5 ppb of benzene, 6.5 ppb of 1,3-butadiene, and 4.7 ppb of formaldehyde. Only the benzene level exceeds the TNRCC screening level (1 ppb). The Houston area concentrations are quite similar to those found in other urban areas.

ES-5. HEALTH EFFECTS

The EPA conducts comprehensive reviews of the health effects associated with Criteria air pollutants every five years as part of their review of the NAAQS. These EPA Criteria Documents provide the scientific basis for the health-based NAAQS. In this study, the health effects literature that has been published since the last EPA Criteria Documents for ozone and PM were reviewed. In addition, we briefly evaluated the potential health impacts of toxic air pollutants important for the Houston area. Although laboratory-based studies are discussed in the review, the emphasis is placed on environmental epidemiological studies because these studies represent the 'real-world' exposures of people living in communities impacted by airborne contaminants. Based on the Criteria Documents and our review of the more recent literature, end-points (symptoms) that can be quantitatively associated with changes in concentrations were identified, and the most scientifically appropriate exposure-response functions were selected for use with the exposure estimates.

A number of factors were considered in assessing the suitability of health effects studies for determination of exposure response functions. In order to be considered, the studies needed to:

- Have been subjected to scientific peer-review.
- Have appropriately considered the effects of potential confounding factors such as other pollutants and meteorological variables.
- Have employed a mix of pollutants relevant for the Houston area.

- Have employed a study population with relevant socioeconomic status and ethnicity for Houston area residents.

However, because of the very limited number of Houston-specific studies in the published literature, studies based in other geographic regions were also considered as candidates.

ES-5.1 OZONE HEALTH EFFECTS

Ozone has been extensively evaluated in laboratory animal and human clinical studies, as well as in epidemiological studies. There is little doubt that ozone can damage lung tissue, cause symptoms in children and adults, and exacerbate diseases such as asthma and bronchitis. The role of ozone as an agent that causes (rather than exacerbates) lung or heart disease is less clear, despite the fact that some specific mechanisms by which ozone injures the lung could also be related to the development of disease. The association of ozone with human mortality is definitely being investigated, but at this time the available evidence does not convincingly provide for effects that can be clearly separated from those of other pollutants (especially fine particles).

Approximately 60 epidemiological studies that investigated the effects of ozone on adult populations and 15 epidemiological studies that investigated ozone effects on children were evaluated in this study. Most of the studies report acute health effects for ozone. We found sufficient evidence and were able to establish concentration-response functions for evaluating benefits related to:

- Chest symptoms (wheeze and phlegm production)
- Eye irritation
- Cough
- Chest congestion
- Throat irritation
- Headache

Current studies suggest ozone may also play a role in more serious health effects (as described for PM) but there is no credible approach for disassociating the effects of ozone from those of its co-pollutants (primarily fine particle PM). To avoid over-estimation of ozone health effects, the potentially more serious health effects have not been quantified separately from those of PM. The potentially more serious health effects of ozone may be included in the effects of PM. However, numerous studies suggest total pollutant-related mortality is primarily attributable to PM rather than ozone. Current studies indicate ozone only explains a small amount of the total pollutant-associated mortality.

ES-5.2 PM HEALTH EFFECTS

Airborne particles are classified with respect to size for regulatory purposes. Fine particles are designated as ones with 2.5 μm or smaller in aerodynamic diameter. Particles

10 μm or smaller in diameter are classified as PM_{10} (which includes the $PM_{2.5}$ class). There is often strong correlation between ambient PM_{10} and $PM_{2.5}$ mass concentrations and in many cases it is possible to infer one from the other by using appropriate ratios. Ambient PM_{10} mass has been monitored on a routine basis in most urban areas since 1986 when the first PM_{10} NAAQS was promulgated. However, prior to 1998, ambient $PM_{2.5}$ levels were not routinely monitored in most parts of the country. Consequently there are many more environmental epidemiology studies that examine associations of health effects with PM_{10} than with $PM_{2.5}$.

Although $PM_{2.5}$ is more strongly associated with human health effects than PM_{10}, it is not appropriate to assume all of the PM health effects are attributable to fine PM. In fact, EPA still maintains a NAAQS for PM_{10} and the literature supports both fine particle and coarse particle health effects. Thus, we have used the best available health-related studies and developed quantitative assessments for either PM_{10} or $PM_{2.5}$ (when the data were directly available) but then estimated the effect that might be due to $PM_{2.5}$ by adjusting the measured PM_{10} concentrations to approximate the $PM_{2.5}$ concentrations.

More than 50 published studies that examined the health effects of PM in adults and about 15 studies that examined PM health effects in children were reviewed. Most of the studies reported health outcomes in terms of relative risk or in terms of a concentration-response function that was associated with relative risk. The health outcomes associated with exposures to particles tend to be chronic in nature rather than acute. Quantitative evaluations were made for the following health outcomes:

- Days of restricted activities due to air pollutant-induced symptoms or illness.
- Days of minor restricted activities.
- Cases of chronic bronchitis.
- Hospital admissions for respiratory and other relevant diseases.
- Premature death.

ES-5.3 AIR TOXICS HEALTH EFFECTS

Three HAPs—benzene, formaldehyde, and 1,3-butadiene—were present in the Harris County air samples at concentrations that exceeded currently accepted risk levels for cancer. Exposures to each of these three compounds are related to increases of specific types of cancers. The EPA has tried, over the years, with limited success, to develop more definitive data sets with which to evaluate the potential health risks associated with ambient air toxics. At the present time, the air monitoring data are geographically quite limited and the number of suitable studies that provide credible estimates of concentration-response function is very small. The former limitation makes it difficult to assess the effects of future emission reductions on environmental exposures to air toxics and definitive air toxics reduction strategies are still to be developed. EPA provided some preliminary estimates of the value of reducing air toxics exposures on a nation-wide basis. EPA's estimates suggest that the health benefits associated with air toxics reductions are small compared to those for ozone and PM. However, it is entirely possible that as better concentration-response functions become available, this issue may need to be revisited.

ES-6. ECONOMIC VALUATION AND BENEFITS ESTIMATION

ES-6.1 ECONOMIC VALUATION

Why do we assign dollar values at all, and what is the basis for those values? The most basic answer to the first question is that society does not have the material resources to do all of the good things that we would like to be able to do. Therefore, we must choose among competing uses for our scarce resources. This is true of individuals and of society as a whole. The objective of placing dollar values on reduced health risks is to help determine whether resources are being used well. If we cannot do all good things, we can at least attempt to do as many and as much as possible, which implies a need to know the relative value of different actions.

Regarding the second question, we want a systematic way to make collective (social) choices, so we need to decide which environmental protections are the most worthwhile, that is, which ones will return the greatest value in improved health. We therefore need a means of comparison that is transparent, uses the best available information, and reflects social preferences. We also need a common denominator - a basis for comparison to weight each option, and we use dollars as the denominator.

To decide what dollar value to place on an adverse health effect, we begin with the premise that, with limited exceptions, we accept individual choices as valid. Other than restrictions like speed limits and proscriptions against murder and some environmentally harmful actions, we assume that what individuals choose to do accurately represents what is best for them, and by reference, for society. In economic benefit assessment, the sum of value to individuals equals social value. We use prices when available (or implied prices), and survey results to value changes in air pollution-related risks to health.

The ultimate objective of this study is a dollar measurement of the benefits of better health associated with lower levels of air pollution. This requires determining how much people value avoiding pollution-related health effects ranging from the most serious consequence (premature death) to relatively minor symptoms such as eye irritation. Value is gained from reducing:

- Direct medical costs and loss of work and school days that result from avoiding or treating adverse health effects.
- Discomfort, inconvenience, and fear resulting from adverse health effects, their treatment, or efforts to avoid them.
- Loss of enjoyment and leisure time.
- Impacts on others as a result of an individual's adverse health effects.

Specific Values

Premature mortality is one of the most significant effects of exposure to elevated levels of fine particles. Consequently, the value that society attaches to reducing pollution-related early deaths is high in comparison to the value of reducing less severe effects. It is important to recognize that we are not attributing this value to the life of any identified individual, or group of individuals. We do not know whose life expectancy will be shortened if pollutant concentrations do not decline. What we are doing is valuing the benefits of reducing a small risk to a large population, and so we refer to the value of a statistical life (VSL).

Most estimates of the value of statistical life that are both well-supported by research and appropriate for valuing environmental risks cluster between $3 million and $7 million. For the purpose of assessing the value to society of reducing risk from air pollution, we want a value that is based on risk of a similar scale and is derived from the preferences of a population not unlike the population at risk from pollution exposure. The value currently used by the EPA is $5.9 million in 1997 dollars. This value meets the criteria noted above and best represents the results of nearly 30 studies.

Overall, morbidity values range from as little as $5.80, to avoid a case of mild cough, to over $600,000 for a new case of moderately severe adult chronic bronchitis. In between, a case of hospitalization for respiratory or cardiac conditions has been valued in the thousands, and other effects in the tens or hundreds of dollars. Basically, costs tend to vary with the extensiveness of medical treatment and the degree to which an effect impairs normal activities.

The values used to estimate economic benefits of declining air pollution levels in the Houston region are summarized in **Table ES-3**. These values are generally used and accepted in economic studies.

ES-6.2 BENEFITS ESTIMATION

The results reported here characterize the magnitude of expected health benefits that would result from alternative emissions reduction scenarios. They should be regarded as indicative of scale rather than as precise measures. Nonetheless, they do represent the best estimates that can be constructed with information now available.

Overall benefit estimates are shown in **Table ES-4**. The total quantifiable economic benefits expected to result from compliance with the annual $PM_{2.5}$ NAAQS and the 1-hr ozone NAAQS in the year 2007 are between $2.9 billion and $3.1 billion annually.[1] More than 400 people each year are estimated to die each year because of pollution levels above the national standards. The adverse health effects of ozone are far more frequent than is the case for fine particles, ranging to over 4 million person-days each year of eye irritation and more than a million days of chest symptoms in asthmatic children; but they carry much less substantial economic value because they are less severe and sometimes more reversible than fine particle-related effects.

[1] The range of benefits represents uncertainty about the relative potency of $PM_{2.5}$ (compared to PM_{10}) in association with chronic bronchitis, hospitalizations, and MRADs.

Several points are important in viewing these results.

- Many well-studied and documented effects associated with urban air pollution are not quantified here. Physiological changes that make people more susceptible to illness are not represented. This omission may be especially important for children, whose lung capacity is still developing.

- The effects of hazardous air pollutants are not represented, nor is initiation of chronic disease other than bronchitis, such as asthma and emphysema.

- We presently have no basis to estimate how much underestimation of benefits is introduced by ignoring the unquantified effects.

- The health science literature has developed primarily to support setting air quality standards for single pollutants. Consequently, the scientific basis for quantifying effects focuses on teasing out the impact of discrete compounds and not the overall impact of the complex mixture that people breathe. While results are reported for ozone and for fine particles, these pollutants in fact occur together, and in combination with other pollutants such as NO_2.

- Mortality (premature death) and chronic bronchitis represent the effects of longer term pollutant exposure on health, while the other effects are acute and occur in relation with one-day elevated concentrations.

- Finally, as reflected in **Table ES-5**, emission control programs designed to reduce ozone and that are included in the SIP will also reduce fine particle concentrations. Consequently, the benefits of ozone controls are greater than just the benefits of reducing ozone-related morbidity quantified in this study. Estimates of the benefits of the ozone SIP, for example, show 44 fewer PM-related deaths annually. Implementation of a clean diesel fuel program similarly reduces ozone exposures as well as PM exposures.

Who Is Most At Risk?

Certain groups of people are more likely to suffer adverse health effects than others following exposure to air pollution. Children are more vulnerable because of their physical immaturity, tendency to be more physically active, and tendency to spend more time out of doors. The air pollution health risks for children is an area of great concern to policy makers. Traditionally, most of the emphasis in developing data for setting air quality standards has been on adults. More recently, however, awareness has grown that children cannot be considered "scaled-down" adults, and that more sophisticated approaches are necessary in order to accurately account for the effects of air pollution on them. Moreover, lungs grow until the late teens or early twenties. If air pollution influences the growth process, resulting in smaller lungs, vitality is at risk.

Those aged 65 and older are at greatest risk of dying prematurely. This effect results from years of exposure, possibly beginning in childhood. Given the lack of a quantified database of health effects on children, the effects on those 65 and older is the most serious of the consequences of elevated pollution concentrations assessed in this study.

Unquantified Effects

The adverse health effects whose frequency in association with air pollution can be quantified, and whose avoidance can also be valued in dollars, is a limited subset of the total set of adverse effects that have been observed or measured in epidemiological, human exposure, or animal studies. **Tables ES-6 and ES-7** list examples of unquantifiable and quantifiable air pollution health effects. Limitations of both health science and economics contribute to our inability to quantify these effects, as does lack of ambient monitoring data for some pollutants. Reduced lung function, for example, which has been linked to fine particles and ozone in a number of health studies, cannot be easily valued in economic terms. Moreover, we simply have no way to calculate the relative importance of what is measurable and what is not, so it is impossible to determine how large the unquantified effects may be in economic terms. In addition, some of the unquantified and quantified effects may overlap. For example, changed pulmonary function accompanies chronic bronchitis. Nonetheless, it is important to recognize the number and nature of such unquantified effects to provide perspective when interpreting the meaning and comprehensiveness of estimates of the economic value of improved air quality.

Currently, some unquantified ozone-related effects are: lower respiratory symptoms, changes in the immune system, chronic respiratory damage and disease, increased rates of hospitalization, inflammation of the lung, increased airway sensitivity, and changes in lung function. Fine particle-related effects that cannot be quantified include: initiation of chronic respiratory disease other than bronchitis, cancer, altered human defense mechanisms (which may be related to vulnerability to disease or injury), changes in the structure of the respiratory system, and changes in lung function.

An important unquantified effect is premature death in children, and, more generally, people under age 30. This is because the study used to determine the quantitative association between pollution exposure and mortality did not include the under-30 population. Consequently mortality risk to that population is not represented here, although other studies suggest that while the risk is smaller to younger people, it is not zero.

ES-7. CONCLUSIONS

This study of health benefits associated with air quality improvements is interdisciplinary in nature. The key conclusions from the study regarding emissions, air quality, exposure, health effects, and economic valuation are listed below.

ES-7.1 EMISSIONS, AIR QUALITY, AND EXPOSURE

Noteworthy characteristics and results of the analysis of emissions, air quality, and exposure are as follows:

- Updated VOC and NO_x emissions estimates used in the photochemical modeling produced comparable results to those produced with previous emissions estimates. Given the high biogenic VOC emission rates in the Houston area, the simulated future ozone levels are still primarily controlled by the regional NO_x emissions, rather than the anthropogenic VOC emissions.
- Revised PM and SO_x emissions from the National Particle Inventory were used, along with VOC and NO_x emission estimates, in a speciated PM rollback model to estimate $PM_{2.5}$ conditions in earlier and later years. The PM and PM-related emissions (SO_x and ammonia) were much less well characterized than the VOC and NO_x emissions. The uncertainty in PM and PM-related emissions remains high. However, with the high background $PM_{2.5}$ concentrations and the linear modeling approach used for this study, the future-year $PM_{2.5}$ concentrations estimates were not strongly sensitive to alternate base-case emission assumptions. The maximum annual average $PM_{2.5}$ concentration in 2007 was about 19 $\mu g/m^3$ regardless of assumptions.
- The ozone exposure estimates were made mostly assuming Houston area residents were exposed to outdoor concentrations at all times. Population exposure estimates that accounted for human time-activity and the modifying effects of enclosures (buildings and vehicles) suggested there would be 85 percent fewer exposures above the 1-hr NAAQS than in the "all outdoors" or ambient exposure simulations. There may be greater disparity between personal ozone exposure levels and outdoor levels in the Houston area than in other less air-conditioned cities.
- A relatively large fraction of the Houston area residents are exposed to high concentrations of $PM_{2.5}$ and ozone. We estimate 62 percent of the population in 1993 and 79 percent of the population in 2007 (without additional control measures) would be exposed to annual $PM_{2.5}$ concentrations above the NAAQS. For the 1993-1995 time frame, we estimate there were 26 million and 72 million person-days per year with exposures above the 1-hr and 8-hr NAAQS levels, respectively. In 2007 without additional controls, we estimate there will be 27 million and 84 million person-days per year with exposures above the 1-hr and 8-hr NAAQS levels, respectively. The 1998 Ozone SIP emissions reductions are likely to reduce ozone exposures above the 1-hr and 8-hr standards by 99 and 90 percent, respectively. The geographic distribution of exposures above the standards are similar, suggesting the same individuals are exposed to high ozone and $PM_{2.5}$ concentrations.

ES-7.2 HEALTH EFFECTS

The review of health effects literature published before and after the last EPA Ozone and PM Criteria Documents reaffirmed concerns for ozone and PM health effects at the

NAAQS levels. Health effects of benzene, 1,3-butadiene, and formaldehyde at ambient levels are less clear. Key findings of this part of the study area are that:

- There is coherence across multiple types of studies (animal toxicological, human clinical, and epidemiological) which supports the conclusion that significant adverse health effects in humans result from exposure to ozone and fine particles at levels typical of the Houston area atmosphere.
- There is coherence of the cascade of effects, including changes in lung structure and function, acute symptoms such as cough and exacerbation of asthma, respiratory illness, and mortality, which strengthens the conclusion that effects observed in epidemiological studies result from pollutant exposure.
- Recent studies suggest an association between ozone and premature mortality. Until this body of evidence expands to include more independent studies demonstrating consistency of this result, we have chosen not to quantify this effect.
- Annual average concentrations of formaldehyde, 1,3-butadiene, and benzene are above accepted risk levels at some Houston area monitors, however, the location of those monitors is not representative of the general population's exposure, so we do not currently have an appropriate basis from which to quantify any potential effects in the overall population. Based on national studies, the benefits of reducing concentrations of these toxic compounds to the risk threshold are likely to be much smaller than achieving compliance with the NAAQS in the Houston area.

ES-7.3 ECONOMIC BENEFITS

- Total annual economic benefits of reduced exposure associated with achieving compliance with the 1-hr ozone and $PM_{2.5}$ NAAQS in 2007 are in the range of $2.9 billion to $3.1 billion in the Houston area.
- The estimated benefits of lower exposure to fine particles far outweigh the benefits of reduced ozone exposure. This is not because ozone is benign, but because the consequences of fine particle exposure are severe and are therefore associated with very large economic benefits to avoid even a relatively small number of effects. For example, we estimate there will be over 4 million fewer days of ozone-related eye irritation with ozone NAAQS compliance and 435 fewer premature deaths at lower fine particle levels. Given that a day of eye irritation is valued at less than $10 and a life lost is valued at nearly $6 million, particle-related benefits dominate the benefits analysis.
- There are significant unquantified effects. Multiple studies have found that lung function is affected by exposure to ozone and to fine particles, but we have no basis on which to quantify the economic benefit of avoiding this effect. We do not yet know the consequences later in life, for example, that might result from short-term impairments in lung function experienced as a child.

- The health studies that support quantification of hospital admissions, chest symptoms in asthmatic children, and restricted activity days are all based on short-term exposure. They do not capture any effects on days when pollution levels are not as elevated but that occur as the cumulative result of longer-term exposure at lower levels. In contrast, mortality and chronic bronchitis estimates are based on longer-term exposures and therefore reflect more completely the effects of exposure over time.
- The study used to determine the association between fine particle exposure and premature mortality did not assess deaths in the under 30 age group. Consequently, mortality risk to that population is not represented in the economic benefit estimates presented here.
- Apart from the aggregate benefits to the region's population, there are significant benefits to some sub-groups. Asthmatic children, who represent a sensitive population, would each experience more than 50 fewer days a year of chest symptoms, on average, with NAAQS compliance.
- The ozone benefits estimated in this study are for compliance with the 1-hr standard. Work is currently underway in Texas to evaluate control requirements for compliance with the 8-hr standard. No attempt was made to estimate the emissions reductions that would be required to meet the 8-hr standard or the benefits derived from meeting the standard. There is concern that even the 15 percent VOC reduction and an 85 percent NO_x emission reduction may be insufficient to attain compliance with the 8-hr NAAQS.

ES-8. RECOMMENDATIONS FOR FURTHER RESEARCH

The process of assessing emissions, air quality, exposure, health effects, and economic valuation helps identify numerous areas where further research could improve the understanding and accuracy of the air pollution effects. Salient areas in need of further research are listed below.

- Given the large benefits estimated for improvements in $PM_{2.5}$ air quality and the significant deficiencies in the current understanding of the $PM_{2.5}$ problem, further research is needed on many aspects of the problem. Research should be conducted to improve the characterization of ambient concentrations, background concentrations, and PM chemical composition in order to understand what is actually in the Houston area air. Likewise, better information on PM and PM-related emissions and control options as well as better air quality models (especially annual models with reasonable physics and chemistry) are needed to develop an understanding of the contribution of different sources to the problem. Emissions and meteorological information suitable for long-term applications of models would be particularly useful given that the annual NAAQS is the controlling standard in this region. Development of an understanding of the seasonal variations in emissions would be particularly useful. In addition, it is important to develop PM emission inventories that have chemical compositions that are consistent with ambient observations of primary PM components.

- Further research on the relationships between ozone and $PM_{2.5}$ precursor emissions is needed to begin exploring multi-species control strategies. Ultimately, a high quality modeling system and database will be needed to assess the effects of actual control measures on both ozone and $PM_{2.5}$ for the appropriate averaging times. The modeling system should be able to account for potential nonlinearities in the gas-phase and aqueous-phase chemistry as well as in the aerosol thermodynamics. At present, the contribution of NO_x emissions to $PM_{2.5}$ ammonium nitrate, the extent of ammonia emission limitations on aerosol nitrate levels and aerosol acidity, and the contribution of VOC emissions to secondary $PM_{2.5}$ organic material are not well known. Given that sulfate and organic material are the two most abundant chemical constituents of Houston area $PM_{2.5}$, this research should include investigation of the roles of SO_2 and VOC emissions in aerosol formation.

- There is little information on the time-activity patterns of Houston area residents and indoor/ outdoor pollutant concentrations of ozone and $PM_{2.5}$ in different types of buildings or with different types of heating/air-conditioning/ventilation systems. This type of information is needed to make better estimates of personal exposure to these pollutants.

- As described in the report, there are myriad adverse physiological responses to pollutant exposure. Many of the physiological responses, such as structural changes in the lungs and airways, reduced lung function, and immune system responses, are logically related to the occurrence of disease in some portion of the population. We presently have no economic knowledge about what the benefits would be of preventing these responses and, thus, have not included the benefits in our analysis. Our inability to value these responses contributes to underestimation of the probable benefits of air quality improvements. Therefore, one important area of further research is interdisciplinary work by health scientists and economists to better model the link between physiological change and adverse health outcomes and to design contingent valuation studies to then determine the value of avoiding the more subtle pre-clinical effects.

- The preponderance of mortality appears to be among the elderly. Given this emerging health science finding, it is important to more clearly determine whether and by how much society values the lives of older people differently than lives in the age groups that current values are drawn from. This is also true for the lives of children, as there is preliminary research indicating higher risk of death in young infants in polluted environments.

- Society places significant weight on protecting children, but we have almost no basis to determine how to value, for example, chest symptoms in children relative to the adults on whom values are now based. Given the demonstrated sensitivity of asthmatic children, and the higher doses of pollution experienced by children in general, relative to adults, this is a key area of investigation.

- Emissions reductions will often reduce multiple pollutants simultaneously, but we typically assess the benefits of reducing one pollutant at a time. More investigation is needed of the joint value of reducing a set of adverse effects, rather than single effects.

- None of the health effects data are specific to the Houston area. It would be helpful to investigate and confirm the types of symptoms and other effects prevalent in the Houston area.

GLOSSARY

Acute = Short-term
Ambient air = Outdoor air
CAMx = Comprehensive Air Quality Model with Extensions
Chronic = Long-term
Criteria Pollutants = Ozone, PM, CO, NO_2, and SO_2
E-GAS = Economic Growth and Analysis System
EPA = United States Environmental Protection Agency
HAPs = Hazardous air pollutants
HAXL = **H**ouston **A**ir e**X**cellence and **L**eadership
Morbidity = Health effects or diseases other than death
Mortality = Deaths
MRAD = Minor reduced activity day
NAAQS = National Ambient Air Quality Standards
NFRAQS = Northern Front Range Air Quality Study
NH_3 = Ammonia
NPI = National Particle Inventory
NO_x = Nitrogen oxides
PPB = Parts per billion (in air)
PM = Airborne particulate matter or the mass of airborne particles
$PM_{2.5}$ = Airborne particulate matter with aerodynamic diameters less than 2.5 µm
PM_{10} = Airborne particulate matter with aerodynamic diameters less than 10 µm
RAD = Reduced activity day
REHEX-II = Regional Human Exposure Model, Version 2
Rollback = A simple air quality model that assumes air concentrations (above their background concentrations) are proportional to emissions
SIP = State Implementation Plan
SO_2 = Sulfur dioxide
Spatial Pattern = Geographic pattern
Speciated = Separated into different chemical components
TNRCC = Texas Natural Resource and Conservation Commission
TNRCC Screening Level = A level of concentration used to determine whether further analysis of health risk should be carried out
VOC = Volatile organic compounds
VSL = Value of a Statistical Life
µm = micrometer (or one millionth of a meter)
$\mu g/m^3$ = microgram of pollutant per cubic meter of air
18 Counties = Austin, Brazoria, Brazos, Chambers, Fort Bend, Galveston, Grimes, Hardin, Harris, Jefferson, Liberty, Matagorda, Montgomery, Polk, San Jacinto, Waller, Washington, Wharton

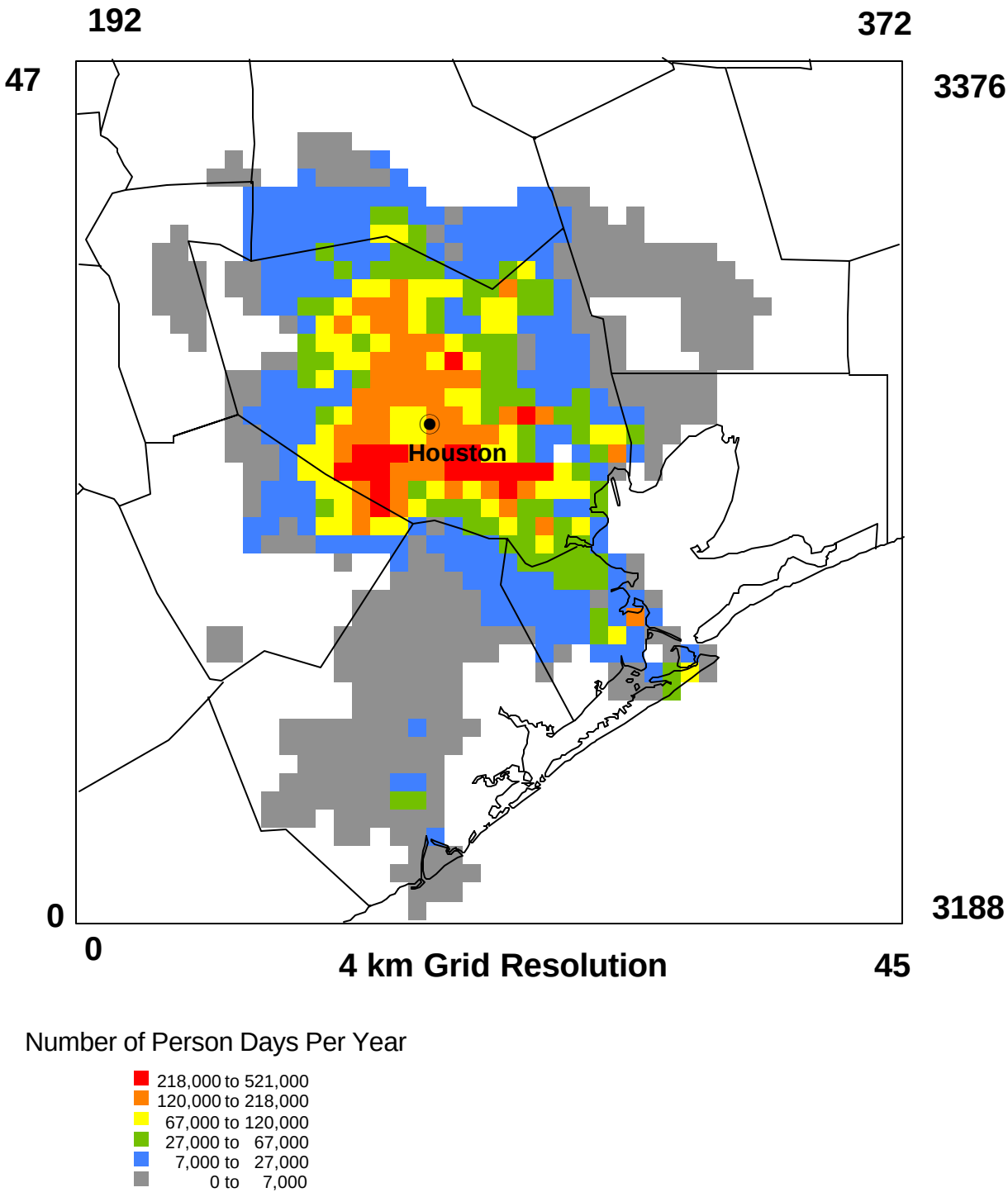

Figure ES-1. Spatial distribution of person-days per year of exposures to 1-hr maximum ozone concentrations above 120 ppb in the 2007 base case with the "all outdoors" approach.

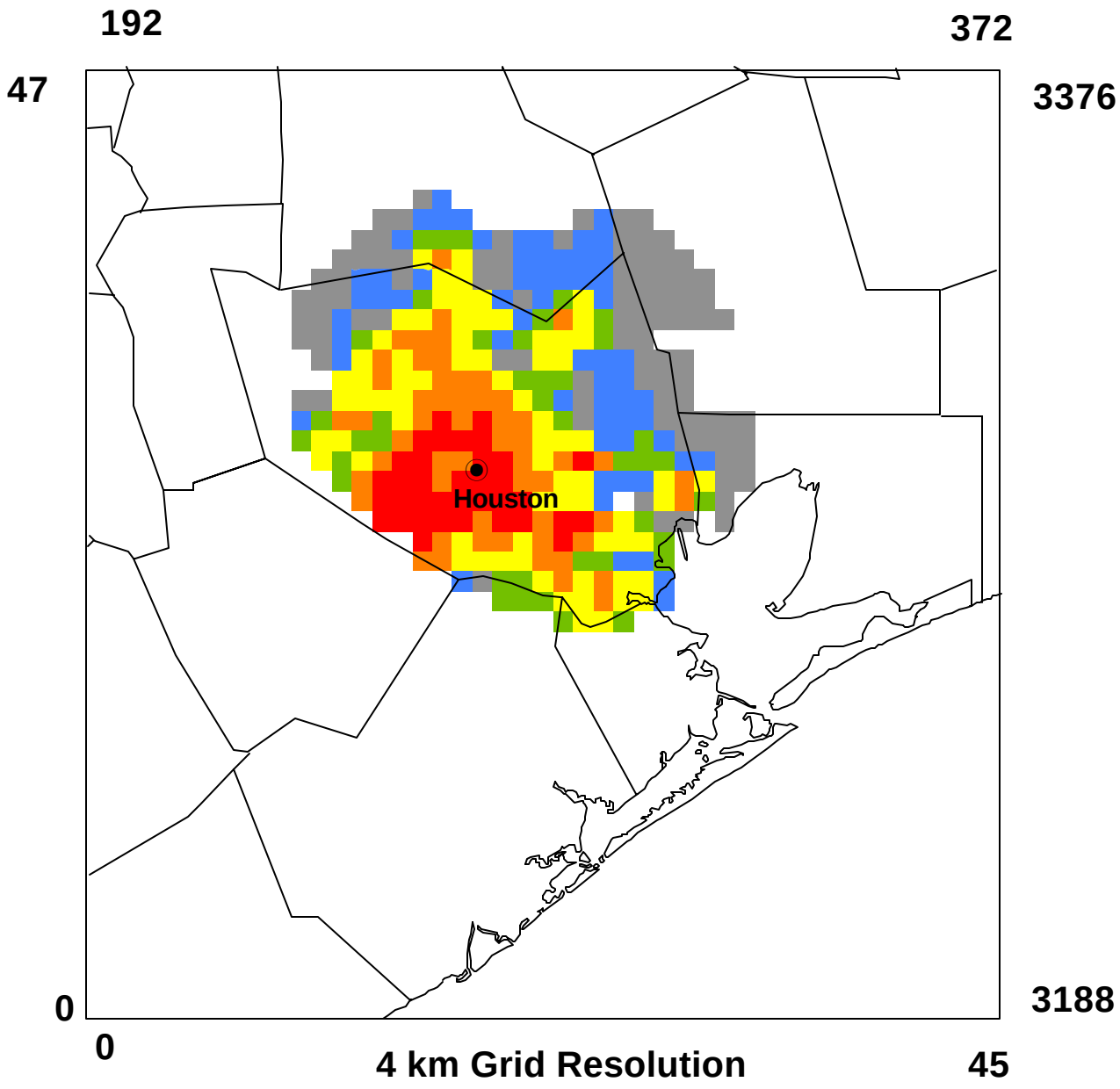

Figure ES-2. Estimated number of people exposed to annual average $PM_{2.5}$ concentrations greater than 15 $\mu g/m^3$ in 2007 under Scenario 1.

Table ES-1. Estimated man-made emissions[a] (tons per day) in the Houston area.

Case	NO_x	VOCs[b]	SO_2	NH_3	$PM_{2.5}$
1993	2,819	1,952	697	149	420
2007 – Scenario 1	2,155	1,562	768	174	483
2007 – Scenario 2	1,970	1,562	749	174	469
2007 – Scenario 3	2,023	1,557	768	174	190
2007 – Clean Diesel Fuel	1,972	1,631	677	173	463
2007 – Ozone SIP	323	1,328	677	173	463

[a] Emission totals are for the 18 counties.

[b] Natural or biogenic NO_x and VOC emissions are approximately 100 and 10,000 tons per day, respectively, on a typical warm summer day. Thus, VOC emissions are dominated by natural rather than man-made emissions.

Table ES-2. Estimated maximum annual average $PM_{2.5}$ concentration in the Houston area.

Case	$PM_{2.5}$ Concentration ($\mu g/m^3$)
1993	17.9
1997-1998[a]	18.3
2007 – Scenario 1	19.0
2007 – Scenario 2	18.8
2007 – Scenario 3	18.9
2007 – Clean Diesel Fuel	18.6
2007 – Ozone SIP	18.6
NAAQS Compliance	15.0

[a] The 1997-1998 maximum was estimated from the spatially mapped observations.

Table ES-3. Unit economic values of health effects in 1997 dollars.

Effect	Dollar Value per Event
Value of a Statistical Life	$5,900,000
Value of Life Year Lost	$360,000
New Case of Chronic Bronchitis	$319,000
Hospitalization for Respiratory Conditions	$8,790
Hospitalization for COPD	$11,670
Hospitalization for Pneumonia	$11,380
Hospitalization for Congestive Heart Failure	$11,960
Hospitalization for Ischemic Heart Disease	$14,840
Lower Respiratory Symptoms	$15/day
Minor Restricted Activity Day	$47/day
Restricted Activity Day	$87.79/day aged 20-64 $53.75/day aged over 64
Cough	$5.80/day
Throat Irritation	$9.75/day
Eye Irritation	$8.45/day
Headache	$9.75/day
Chest Congestion	$9.10/day

Table ES-4. Aggregate economic benefits of reduced fine particle and ozone exposures associated with achieving the annual $PM_{2.5}$ NAAQS and 1-hr ozone NAAQS in 2007.

Health Effect	Affected Group	Number of Health Effects[a]		Dollar Values (in millions of 1997 dollars)	
		Unadjusted[a]	Adjusted	Individual Group	Total
Mortality	Ages 30-64	-	132	$778.8	
	Ages > 64	-	303	$1,787.7	
Total Mortality	All	-	435	-	$2,566
Chronic Bronchitis	Ages 20-64	637	1071	$203.2 - 341.6	
	Ages > 64	74	125	$23.6 - 39.9	
	All	711	1,196	-	$227 – 381
All Respiratory Hospitalizations	Ages < 20	56	92	$ 0.5 - 0.8	
	Ages 20-64	118	194	$1.0 - 1.7	
	Ages > 64	14	23	$0.1 - 0.2	
	All	188	309	-	$1.6 -2.7
Cardiac Hospitalizations	Ages > 64	6	9	-	$0.1 - 0.1
Minor Restricted Activity Days	Ages 20-64	-	606,000	$28.5	
	Ages > 64	-	70,600	$3.3	
	All	-	676,600	-	$31.8
Restricted Activity Days	Ages 20-64	-	182,500	$16.0	
	Ages > 64	-	21,200	$1.1	
	All	-	203,700	-	$17.1
Cough	Ages > 20	345,800	345,800	-	$2.0
Chest Discomfort	All Ages	344,000	344,000	-	$3.1
Headache	All Ages	580,800	580,800	-	$5.7
Irritated Throat	All Ages	957,400	957,400	-	$9.3
Eye Irritation	All Ages	4,350,000	4,350,000	-	$36.8
Chest Symptoms	Asthmatic Children, Ages 7-13	1,100,000	1,100,000	-	$16.7
Total Morbidity[b]	All	-	-	-	$351 - $507[b]
TOTAL BENEFITS					$2,917 - $3,073

[a] Health effects are estimated assuming all of the particle-induced effects found in PM_{10} epidemiologic studies are due to fine particles (adjusted estimates) and due to fine and coarse particles (unadjusted).

[b] Approximately $74 million of the total morbidity is attributable to ozone exposures.

Table ES-5. Relative reductions in fine particle-related effects from compliance with the annual $PM_{2.5}$ NAAQS and 1-hr ozone NAAQS in 2007.

Effect	Benefits of annual $PM_{2.5}$ NAAQS compliance starting from			Benefits of clean diesel fuel[d]	Benefits of the 1998 Ozone SIP[e]
	Scenario 1[a]	Scenario 2[b]	Scenario 3[c]		
Reduction in Number of Deaths Per Year	435	409	418	44	44
Reduction in Number of Chronic Bronchitis Cases Per Year	711	668	687	81	81
Reduction in Number of Reduced Activity Days Per Year	203,700	201,300	197,000	24,000	24,000

[a] 2007 baseline projected inventory

[b] Emissions growth rates limited to population growth rate

[c] Dust content of PM inventory limited

[d] Scenario 1 + clean diesel fuel

[e] Scenario 1 + ozone SIP

Table ES-6. Known health effects and suspected health effects of air pollutants which are currently unquantifiable.

Known Air Pollution Health Effects	Suspected Air Pollution Health Effects
Changes in lung structure	Acute myeloid leukemia (HAPs)
Respiratory tract damage	Other cancers (HAPs)
Accelerated “aging” of lung	Cardiac arrhythmias (HAPs)
Heart rhythm and rate anomalies	Premature death (Ozone)
Respiratory illness in children	Emergency room visits (Ozone)
Exacerbation of existing lung disease	Lung cancer
Reduced pulmonary function	Prevalence of asthma
Increased respiratory symptoms	Altered lung defense mechanisms

Table ES-7. Types of known health effects of air pollutants that are currently quantifiable.

Known Health Effect	Exposure Quantified	Number of Effects Quantified	Economic Value Quantified
Reduced pulmonary function	✓		
Increased respiratory symptoms	✓	✓	✓
Increased asthma symptoms	✓	✓	✓
Hospital admissions	✓	✓	✓
Initiation of chronic bronchitis	✓	✓	✓
Minor restricted activity days	✓	✓	✓
Restricted activity days	✓	✓	✓
Headache	✓	✓	✓
Eye irritation	✓	✓	✓
Premature death (PM-related)	✓	✓	✓

1. INTRODUCTION

1.1 BACKGROUND

Air quality conditions in the Houston area are not in compliance with the National Ambient Air Quality Standards (NAAQS). The area is known to exceed the 1-hr and 8-hr ozone standards, and new monitoring information indicates the area is likely to exceed the new annual NAAQS for fine particulate matter ($PM_{2.5}$). Extensive analysis of the ozone problem, including detailed photochemical modeling for the State Implementation Plan (SIP), indicates large emission reductions are needed to achieve compliance with the 1-hr ozone standard. Attainment of the new 8-hr ozone standard will probably be more difficult than attainment of the older 1-hr standard. Characterization of the $PM_{2.5}$ problem is in its early stages and much work is needed to understand the source contributions and options for mitigation of the problem. There is a clear need to characterize the relationship between ozone and $PM_{2.5}$ in the Houston area and understand the likely effects of changes in common precursor emissions on these species. In addition, there is concern for human exposure to other hazardous air pollutants (HAPs) that are not covered by NAAQS. The sources that contribute to ozone formation and ambient $PM_{2.5}$ may also contribute to the ambient concentrations of toxic compounds.

The understanding of emissions and atmospheric processes has progressed to a state where integrated multi-species approaches are needed for development of control strategies. While many compounds are emitted into ambient air and many connections exist between species via complex reactions that may produce additional species, there is still only "one atmosphere." A key air quality concern is how emissions changes will affect the whole interactive mixture of particles and gases in the "one atmosphere". Furthermore, given our health-based air quality standards, a key public health concern is reducing the health risks of exposure to this mixture of compounds in the most effective manner.

Achieving compliance with the NAAQS may be disruptive and expensive. In order to obtain broad-based support for control plans, the approach to compliance needs to be practical, based on the best available science, and optimized to meet the public health objectives. The regulatory authorities responsible for air quality resource management face challenging questions about how best to achieve compliance with the Federal Clean Air Act requirements. In addition to uncertainties regarding emissions and air quality, little is known regarding the health benefits of different approaches for compliance in the Houston area. For at least three years, the **H**ouston **A**ir e**X**cellence and **L**eadership (HAXL) group has expressed strong interest in assessment of the health benefits of alternate air quality control strategies. This report describes a study commissioned by the City of Houston and guided by the HAXL committee to address the concerns regarding the health benefits of alternate air pollution control programs.

Detailed control plans have not been fully developed to address the ozone problem in Houston. No control strategy has been developed for $PM_{2.5}$ since the Federal regulatory deadlines for having plans in place are after 2005. The 1998 ozone SIP update indicates that a

15 percent VOC emission reduction and an 85 percent NO_x emission reduction are needed to bring the area into compliance with the 1-hr standard. The 1998 SIP does not list the specific control measures that would have to be adopted to reduce emissions by these amounts. The absence of defined control plans provides an opportunity for careful selection of the best control plans to deal with both the ozone and $PM_{2.5}$ problems in the area.

1.2 OBJECTIVES

The overall purpose of the study is to provide information that will assist decision-makers in setting priorities for emission reductions based on the relative health benefits of different emission control strategies. The specific objectives are to:

- Identify and economically value the health benefits of achieving compliance with the NAAQS
- Identify and economically value the health benefits of alternate control strategies that would make progress towards compliance with the NAAQS
- Investigate relationships between ozone and $PM_{2.5}$ control strategies
- Explore the health benefits of reductions in selected toxic air contaminants that arise from emissions of substances that also contribute to ozone and/or $PM_{2.5}$
- Identify control strategies that maximize health-related economic benefits.

An approach was devised to address these objectives with existing data, models, and knowledge for the Houston area. While new data would be helpful to address these objectives in a more refined manner, the time frame for this study did not permit collection of new data.

1.3 OVERVIEW OF THE APPROACH

An integrative and multi-disciplinary approach is needed to address these objectives. Quantitative links must be made between emissions, air quality, human exposure, health effects, and economics in order to identify and value the health benefits of meeting the NAAQS and implementing alternate emission control plans. Missing links between any of the disciplines inhibits the ability to make quantitative estimates for the final outcomes. For example, one common problem is that there are no valid means to economically value certain widely reported health effects, such as decrements in lung function. Our approach focuses on health effects that can be quantified and economically valued. However, by neglecting certain symptoms that cannot be valued, the resulting health benefit estimates may underestimate actual benefits.

The technical approach and sections of this report are organized into the five disciplines noted above. The approach for each element is summarized below.

- **Emissions –** Baseline (1993) and projected future-year (2007) emission inventories were compiled for VOC, NO_x, CO, SO_x, NH_3, and PM in the Houston area. The emission estimates were based on information obtained from the Texas Natural

Resource Conservation Commission's (TNRCC) revised ozone SIP modeling database and the U.S. Environmental Protection Agency's (EPA) National Particulate Inventory (NPI). The analysis includes investigation of the sensitivity of the 2007 emission estimates to different assumptions regarding growth, transportation sources, and control measures.

- **Air Quality** – Baseline air quality conditions were characterized using ozone measurements for 1993-1995, $PM_{2.5}$ measurements for 1997-1998, and toxic compound measurements for 1992-1997 in the Houston area. These ambient data were spatially mapped to 4 x 4 km grids for use in the population exposure modeling. Projections of future-year ozone conditions were developed using photochemical modeling and rollback assumptions. Projections of future-year $PM_{2.5}$ conditions were made using a speciated PM rollback model. No projections were made for toxic compounds.

- **Population Air Pollution Exposure Assessment** – The Regional Human Exposure (REHEX) Model was applied to estimate the population exposure to ozone and $PM_{2.5}$ in the baseline period and future-year periods under a variety of scenarios. The population's exposure to ozone was estimated with and without consideration of the modifying effects of buildings on exposure. The key results from this element are estimates of the population exposure to outdoor pollution levels in 2007 under different scenarios.

- **Health Effects –** Considerable health effects research has been published since the EPA's last assessment in the ozone and PM Criteria Documents. Under this element, recent health effects literature was surveyed, end-points (symptoms) that can be quantitatively associated with changes in concentrations were identified, and the most scientifically appropriate exposure-response functions were selected for use with the exposure estimates. The rationale for selection of specific exposure-response functions is documented.

- **Benefits Estimation –** The EPA has expended considerable effort during the past few years to assemble and evaluate the health effects and economics databases for assessing the benefits of air quality improvements. That work, with more recently published studies, provides an established basis for estimating changes in the frequency of illness and other adverse health effects, and for calculating the economic benefit of reducing those effects. Under this element, the literature on economic valuation of the health effects was reviewed and the most appropriate values for morbidity and mortality were selected. The response functions and monetary valuations were applied to the exposure modeling results to estimate reductions in the number of symptoms and the dollar values of reduced symptoms associated with various air quality improvements and emission control strategies. The estimated benefits of improved air quality in Houston were compared with previous estimates for other parts of the country.

The methodology and results from each element of the study are described in the following sections of the report.

2. EMISSIONS COMPILATION

The analysis techniques used to assess the air pollution exposures in the base year and future years required the compilation and projection of ozone precursor emissions [i.e., volatile organic compounds (VOC), nitrogen oxides (NO_x), and carbon monoxide (CO)], primary PM emissions, and other secondary PM precursor emissions (ammonia and sulfur dioxide) for the Houston study area. The emissions assessment task included five major elements: (1) acquisition, assessment, and compilation of existing emissions data for VOC, NO_x, CO, SO_2, PM_{10}, $PM_{2.5}$, and NH_3 for the Houston area, (2) assembly of a 1993 base-year emission inventory, (3) projection of the 1993 base-year inventory to 2007, (4) seasonal allocation of the emissions to characterize summer and winter differences, and (5) chemical speciation of the fine and coarse PM emissions. The approach and results of each element are described below.

Other analyses in this study involve hazardous air pollutants, such as benzene, 1,3-butadiene, and formaldehyde. No attempt was made to quantify emissions of hazardous air pollutants in this study. The EPA has compiled a National Toxics Inventory (NTI) for 1993 that contains countywide annual average emission estimates for the 118 HAPs listed in the Clean Air Act (CAA) (Pope et al., 1996). Due to the large uncertainties in these data, the EPA suggests that they are not suitable for use in detailed air quality assessments at this time (Rosenbaum et al., 1998).

2.1 DATA ACQUISITION

A variety of data sources were used to compile the various components of the emission inventories. The VOC, NO_x, and CO emission inventories that were used in the Houston/Galveston State Implementation Plan (Texas Natural Research Conservation Commission, 1998) were acquired. These were hourly, gridded, day-specific emissions files formatted for use in the UAM-V or CAMx photochemical models. The TNRCC supplied these emission inventories for the September 6-11, 1993 baseline episode days and provided projections for 2007. Sources in the inventory were categorized as area, point, on-road mobile, or biogenic emission sources. Area sources include commercial, small industrial, and residential sources, evaporative emissions, and off-road mobile sources (e.g., lawn mowers and construction equipment). Point sources are comprised of power plants, large coating operations, storage tanks, and large-scale industrial processes including fugitive emissions. On-road mobile sources include startup emissions, exhaust emissions, and fuel evaporation for motor vehicles traveling on roadways. Biogenic sources are made up of natural and cultivated plants. During the course of the study, the TNRCC supplied new estimated biogenic emissions (for any year) and new on-road mobile estimates for the future year, which were used in this study.

The most complete and readily available emission inventory of SO_x, PM_{10}, $PM_{2.5}$, and NH_3 for the Houston area is the EPA NPI for 1990. The 1990 NPI contains countywide annual average SO_x, PM_{10}, $PM_{2.5}$, and NH_3 estimates resolved into three major source types: (a) point sources (e.g., power plants and refineries); (b) area sources (e.g., wind blown and entrained road dust, and off-road activities); and (c) on-road mobile sources. The NPI also

contains estimates of VOC, NO_x, and CO, which we compared with the emissions obtained from the TNRCC. Several other data sources were used to obtain emissions estimates including the AIRS Texas point source inventory, the National Toxics Release Inventory (TRI), and the Houston/Galveston State Implementation Plan (Texas Natural Resource Conservation Commission, 1998). Ratios between pollutants from the same source (e.g., SO_x and NO_x from power plants) in NPI were used to determine the reasonableness of the corresponding emissions estimates for each source category.

The most complete and readily available emission inventory for toxic substances for the Houston area is the EPA National Toxics Inventory (NTI) for 1993. The 1993 NTI contains countywide annual average emission estimates for the 118 HAPs listed in the CAA. The toxic emissions in the NTI database are resolved into point sources, area sources, and on-road mobile sources. Data from the 1993 Toxic Release Inventory (TRI) were used by the EPA as the foundation of the NTI for major sources, along with emission estimates developed using HAP emission factors and corresponding activity data for area and mobile source categories (U.S. Environmental Protection Agency, 1996).

2.2 OVERVIEW OF THE EMISSIONS DATA

2.2.1 Ozone Precursor Emissions

Ozone precursor emissions rates exhibit significant day-to-day variations as a result of variations in human activity (e.g., weekday rates differ from weekend rates) and environmental factors such as temperature and solar radiation. For illustration purposes, we report here the emission estimates for September 8, 1993, which were typical of the weekday emissions for September 6–11 (note the last day modeled is a Saturday). The area, point, on-road mobile, and biogenic VOC, NO_x, and CO emissions for September 8, 1993, estimated by the TNRCC, are listed in **Table 2-1** for the 18-county domain included in the Houston/Galveston SIP. Biogenics were the major source of VOC emissions, accounting for over 81 percent of the total VOC emissions in the study domain. Point sources were the largest source of NO_x emissions, accounting for over half of the NO_x inventory. Point and on-road mobile sources were the two largest CO sources, accounting for 48 percent and 31 percent, respectively, of the September 8 CO emissions.

2.2.2 PM and PM-related Emissions

Point, area, and on-road mobile source emissions, as reported in the 1990 NPI, are listed in **Table 2-2** for Harris County and the 17 surrounding counties (the 18-county domain – see **Figure 2-1**). Harris County contains the city of Houston and accounts for more than half of the PM emissions in the domain. **Figure 2-2** shows the contributions of point, area, and on-road mobile sources to total SO_x, PM, and NH_3 for the 18-county domain as reported in the 1990 NPI.

2.3 INVENTORY QUALITY ASSESSMENT AND ADJUSTMENTS

2.3.1 Ozone Precursor Emissions

The 1993 emission inventory provided by the TNRCC was compared with the values reported in the Houston/Galveston SIP to ensure that the correct emission inventory was used. The intention of this assessment was to ensure that the baseline inventory used in our ozone modeling was the same as that used in the baseline study reported in the Houston/Galveston SIP. Small differences between the SIP published component source emissions and values calculated by STI, using the TNRCC provided inventory, were assumed to be numerical calculation rounding errors.

2.3.2 PM and PM-related Emissions

The emissions reported in the 1990 NPI were assessed by comparing emissions estimates in the 1990 NPI to independent sources of emissions data, census data, and emissions estimates for regions of the country similar to Houston. The objective of this emissions assessment was to check the data for reasonableness and to arrive at emissions estimates that are realistic and consistent among several sources of data.

2.3.3 Point Source Emissions

Point sources are major contributors to SO_x, VOC, and NO_x emissions and only minor contributors to PM and NH_3 in the study area. Point source emission estimates of PM_{10} and SO_x reported in the 1990 NPI for Harris County were compared to PM_{10} and SO_x reported in an alternative point source inventory available on the EPA's AIRS web site. The values reported in the 1990 NPI are approximately 35 percent higher than those reported in the AIRSData web site. This is not surprising since some of the data in the AIRSData web site have not been updated in several years. As a second check, the 1990 NPI point source VOC and NO_x estimates were compared to point source emissions data reported in the Houston/Galveston SIP for 1993. The VOC and NO_x estimates reported in the 1990 NPI agree with the 1993 SIP values within about ±25 percent, not an unreasonable difference between two independent data sources.

Estimates of NH_3 emissions reported in the NPI were compared to those in the TRI. The TRI is an inventory maintained by the EPA that includes self-reported toxic emissions for major point sources. Point source NH_3 estimates reported in the NPI are 1.8 tons/day, while point source NH_3 emissions in the TRI are approximately 4.3 tons/day. Because the TRI database relies on emissions data reported by individual facilities, it is thought to be more accurate than the estimates in the NPI, suggesting that the point source NH_3 data in the NPI are likely underestimated by at least a factor of 2.

A strategy was developed to revise the original NPI point source NH_3 inventory. Revised NH_3 emissions were computed by applying emission factors for NH_3 and NO_x for the major sources of NH_3 including: natural gas, oil, and coal combustion processes as well as fluid catalytic cracking, a process used during petroleum refining. The emission factors were obtained from EPA's pollutant emission factors from AP-42 (U.S. Environmental Protection Agency, 1997). The point source NH_3 estimates in the 1990 NPI were revised by using source-specific NO_x emission factors to back-calculate fuel consumption. The fuel consumption estimates were then multiplied by source-specific NH_3 emission factors to calculate the NH_3 emissions. The methodology used to revise the point source NH_3 emissions is shown in Equation 2-1.

$$E_{NH3} = \left(\frac{E_{NOx}}{EF_{NOx}} \right) EF_{NH3} \qquad \textbf{(2-1)}$$

where:

E_{NOx} = Existing NO_x emissions from combustion source (tons NO_x/year)
EF_{NOx} = Fuel-specific NO_x emission factor (tons NO_x/ton fuel burned)
E_{NH3} = Adjusted NH_3 emission estimate (tons NH_3/year)
EF_{NH3} = Fuel specific NH_3 emission factor (tons NH_3/ton fuel burned)

The revised NH_3 emissions for combustion processes and catalytic cracking increased the point source 1990 NPI NH_3 estimates from 1.8 to 5.3 tons/day, resulting in an estimate which is more consistent with the value reported in the TRI (i.e., 4.3 tons/day).

2.3.4 Discussion of Area Source Emissions

Area sources are responsible for approximately 95 percent of the total PM_{10} emissions in the domain. The main sources of PM_{10} are wind blown dust (erosion) and entrained road dust from both paved and unpaved roads. The 1990 NPI area source inventory includes off-road mobile sources such as construction and heavy-duty diesel equipment, which also emit substantial amounts of PM. Area source categories of livestock, wastewater treatment, and industrial fuel combustion are the main sources of NH_3 emissions in the study area. A cursory review of the 1990 NPI NH_3 emissions suggests that they are too low. **Table 2-3** presents a comparison of several "vital statistics" affecting ammonia emissions such as geographic area and cattle population for a recently compiled NH_3 emission inventory in central California (Coe et al., 1998) and the 18-county area centered in Houston. The central California NH_3 emission inventory was based on recent activity data and emission factors that were derived from a critical review of the relevant literature. The comparisons suggest that the area source NH_3 emissions for the Houston area may be underestimated by a factor of 2 or more.

Emissions reported for the major area sources of NH_3 were investigated. Emissions of NH_3 from wastewater treatment appeared to be reasonable for the Houston area on a per capita basis; therefore, emissions from this source category were not revised. However, livestock

and industrial combustion emissions appeared to be low throughout the 18-county domain. Emission factors for these categories were obtained from AP-42 and combined with activity data to generate new estimates. The effect of adjusting the NH_3 emissions for livestock, cattle feedlots, and industrial combustion sources was an increase in the total area source NH_3 emission estimates from 55 to 127 tons/day (with most of the emissions coming from livestock-cattle feedlots). The revised NH_3 estimates for Houston (127 tons/day) now closely match what would be expected for a region with the human and cattle populations of the Houston study area.

It is important to note that fertilizer application was not revised due to lack of information about fertilizer composition and application in the Houston region, but could potentially be a significant contributor to NH_3 emissions. Also, naturally occurring NH_3 emissions from soil, which can be a major source of NH_3 emissions, were not included in the inventory.

2.3.5 On-Road Mobile Source Emissions

Motor vehicles emit PM and NH_3 in their exhaust and re-suspend PM road dust. Review of the NH_3 emissions in the NPI, however, in our judgement for the 18-county Houston area, appear to be too large by a factor of 3 to 5. Preliminary calculations were performed using countywide vehicle miles of travel (VMT) estimates compiled by the EPA for use in its upcoming revised NPI inventory for 1996 and emission factors published in the technical documentation for the 1990 NPI. The transportation data indicate that 70, 15, and 15 percent of the VMT was associated with catalyst-equipped gasoline-fueled vehicles, non-catalyst gasoline-fueled vehicles, and diesel-fueled vehicles, respectively. For each county, ammonia emissions calculated using the 1996 VMT and EPA emission factors were a factor of 3 less than those published in the 1990 NPI emission inventory. In addition, the on-road mobile NH_3 emissions from the NPI inventory for Houston were compared to those for central California. The on-road mobile source ammonia emissions for the 18-county Houston area were more than double those in the recently updated central California inventory even though the Houston VMT was only half of that in the central California estimates. The Houston NPI mobile ammonia emissions were two times larger, instead of two times smaller than those in the California study. While fuel and vehicle fleets are not identical in the two study areas, it seems unlikely that small differences in vehicles and/or fuel could account for a factor of 4 difference in the overall on-road mobile source NH_3 emissions.

Because of these discrepancies, the existing mobile source NH_3 emissions estimates in the 1990 NPI were revised for light-duty gasoline vehicles and diesel vehicles using the ammonia emission factors published in the NPI technical documentation combined with best-available VMT data. The Houston-Galveston Area Council (HGAC) provided VMT data for Harris County and seven adjacent counties. The VMT data were generated as part of the Vision 2020 Conformity Analysis and include VMT estimates for 1990, 1996, 1999, 2007, 2010, and 2020. Data provided by the HGAC were used to adjust the mobile source emissions for counties with available VMT data, and the 1996 National Emissions Trend (NET) VMT data were used for all remaining counties.

Further investigation of the on-road mobile source categories in the NPI inventory showed that emissions estimates of SO_x, PM_{10}, $PM_{2.5}$, and NH_3 were completely missing for heavy-duty diesel vehicles (HDDV). However, estimates of VOC, NO_x, and CO were reported for HDDV. Because HDDVs can contribute significantly to PM emissions, it is important that these categories are not omitted from the inventory. The missing emissions were estimated using NO_x estimates reported for HDDVs in the NPI and NO_x/SO_x and NO_x/PM pollutant ratios as reported in the California Air Resources Board (ARB) emission inventory (California Air Resources Board, 1996). Heavy-duty diesel NH_3 emissions were estimated using the emission factor for diesel vehicles as reported in the 1990 NPI technical documentation.

The NPI and revised on-road mobile source emission estimates are summarized in **Table 2-4**. The revised emission estimates are 35 percent lower than the NPI estimates for NH_3 and 35 percent higher than the NPI estimates for $PM_{2.5}$ and PM_{10}. The on-road mobile source SO_x emissions are the same in both inventories.

2.4 DEVELOPMENT OF THE 1993 BASE-YEAR AND 2007 FUTURE-YEAR INVENTORIES

2.4.1 Ozone Precursor Emissions

As stated previously, the TNRCC provided emission inventories for September 6-11, 1993 and projected inventories for the same dates in 2007. They provided the VOC, NO_x, and CO emission files used for the SIP and updated files for biogenics and future-year mobile sources. **Table 2-5** presents the Houston/Galveston SIP emissions and the revised emission totals used in this study. STI's totals for the 1993 area, point, and on-road mobile emissions are very close to those in the SIP documentation (but not exactly the same). The "STI" totals reflect the emissions as received from TNRCC. Differences may be attributed to numerical rounding. Note that although the TNRCC did not report CO emissions totals in the SIP, they are used in the modeling and reported here.

Subsequent to the 1998 SIP, the TNRCC revised the biogenic VOC emission inventory for the Houston/Galveston area using updated species emission factors and improved biomass data for the urban subregions. The new biogenic emission estimates for September 8 are 20 percent higher than the previous estimates (10,212 versus 8,496 tons/day). Biogenic emissions are assumed to be the same in future years as in the base year (i.e., assuming no discernable changes occur in plant populations). The biogenic emissions are by far the largest source of VOCs in the 1993 and 2007 inventories.

Unlike comparisons made with the 1993 data in which STI's calculated totals closely matched the totals reported in the SIP, the area and point emission totals for the year 2007 varied significantly between what was reported in the Houston/Galveston SIP and what STI calculated using the TNRCC provided inventories. For example, September 8 NO_x point source emissions totaling 1620 tons/day were reported in the SIP. STI calculated point source

emissions totaled 1288 tons/day. Similarly, September 8 area VOC emission totals reported in the SIP were 1041 tons/day while STI calculated VOC emission totals equaled 846 tons/day. It should be noted that area NO_x emissions reported in the SIP for 2007 were very close to those calculated by STI. TNRCC staff were contacted, and they confirmed STI's totals for the point source files. The TNRCC updated the on-road emission inventory from that used in the Houston/Galveston SIP. The new mobile source emission estimates for 2007 are substantially lower than the former estimates. The VOC emission estimates for September 8, 2007, were reduced from 400 to 120 tons/day. Likewise, NO_x emission estimates were reduced from 675 to 282 tons/day and the CO emissions were reduced from 3,345 to 901 tons/day. TNRCC staff were contacted, and they confirmed STI's totals for the new mobile source files.

2.4.2 PM and PM-related Emissions

The revised 1990 inventory was used to generate a 1993 base-year inventory using default growth factors from the EPA's Economic Growth and Analysis System (E-GAS) for the 18-county Houston domain. E-GAS was developed to project emission inventories for ozone non-attainment areas. E-GAS estimates region-specific economic growth projections, employment growth projections, growth in production and energy consumption, and changes in demographic variables. **Table 2-6** summarizes the 1993 base-year inventory for point, area, and on-road mobile source emissions in the 18-county domain. It also summarizes the emissions for a smaller 8-county domain and for Harris County. The 8-county domain includes Harris, Brazoria, Chambers, Fort Bend, Galveston, Liberty, Montgomery, and Waller Counties. **Figure 2-3** shows the contributions of point, area, and on-road mobile source emissions to total SO_x, PM_{10}, and NH_3 in the 1993 base-year inventory for the 18-county domain.

As calculated in the revised inventory, projected to 1993, Harris County accounts for 32 percent of the SO_x emissions and more than half of the PM emissions. NH_3 emissions from Harris County are a small fraction of the NH_3 emissions in the entire domain, because NH_3 emissions from livestock (area source subcategory) are the main contributor to NH_3 emissions. U.S. Agriculture Census data were used to adjust the NH_3 livestock emissions based on the number of cattle per county. Harris County has about half as many cattle (48,000) as Washington County which has the highest number (87,000) in the domain. However, Harris County has higher NH_3 emissions from combustion and wastewater treatment than does Washington County. The source category contributions to SO_x and PM_{10} in the 1993 inventory are consistent with those in the published 1990 NPI (see Figure 2-2). However, area sources contribute more to the revised inventory NH_3 emissions (about 90 percent), mobile sources contribute less (about 7 percent), and point source contributions remain the same.

The 1993 inventory was projected to 2007 using a combination of E-GAS default growth factors and other growth factors for point, area (including off-road mobile equipment), and on-road mobile sources. The point and area source strengths were projected to 2007 using the E-GAS default growth factors for all sources except windblown dust. We assumed the windblown dust emissions would not grow with time. On-road mobile emissions were

projected using 2007 VMT data provided by HGAC for Harris and surrounding counties, and E-GAS default VMT growth factors for the remaining counties in the domain. On-road HDDV emissions were projected by accounting for both VMT growth and fleet turnover effects expected due to the recently adopted PM emissions standards which are expected to decrease the PM_{10} emission factors for HDDV by about 80 percent by 2007 (U.S. Environmental Protection Agency, 1997). Note that specific growth scenarios to account for possible increases in HDDV emissions from trucks from Mexico were not performed. **Table 2-7** summarizes the 2007 future-year inventory for point, area, and on-road mobile source emissions in the 18-county and 8-county domains. **Table 2-8** summarizes the percent growth in emissions from 1993 to 2007. These projections suggest area sources are likely to remain the dominant sources of PM and NH_3 emissions in the region.

2.5 SEASONAL ALLOCATION OF EMISSIONS

Most pollutants are emitted in varying quantities at different times of the year. In order to investigate potential seasonal variability of PM emissions, summer average and winter average emissions were estimated from the annual average estimates and readily available allocation factors. Seasonal allocation factors for area and mobile sources were obtained from EPA's Emissions Processor System 2.0 (EPS 2.0). This database includes season emission adjustment factors for sources like residential fuel combustion (for space heating) which are typically higher in the winter than in the summer. Point sources were seasonally allocated based on seasonal activity factors contained in the point source records of the 1990 NPI. Seasonal factors for the ten largest area and mobile source emissions categories were reviewed and adjusted for Texas conditions, while the remainder of the area and mobile source categories were assigned EPS 2.0 default profiles. **Table 2-9** shows the average winter and summer emissions for point, area, and on-road mobile sources in 1993 for the 18-county domain.

2.6 SPECIATION

Ozone precursor emissions need to be chemically resolved for use in photochemical modeling. The emissions provided by TNRCC were already split into the Carbon Bond IV mechanism species (NO, NO_2, PAR, OLE, ETH, TOL, XYL, ISOP, FORM, ALD2, CO) used in the CAMx photochemical model. Speciated PM emissions are needed to apply almost any PM air quality model. For this study, speciated PM emissions data were needed for use with a speciated PM rollback model that backcasted and forecasted $PM_{2.5}$ concentrations in the Houston area. The 1993 and 2007 PM mass emission estimates were divided into the 13 individual chemicals or chemical classes listed in **Table 2-10**. These compounds and classes were selected because they are the most abundant PM species. PM emission speciation information from several databases was reviewed in order to select the most appropriate profiles for Houston.

The EPA SPECIATE database contains many PM speciation profiles for various industrial emissions source categories. However, these data are mostly limited to point sources and are not region-specific. Much of these data are outdated and appear to be incorrect.

There are limited existing PM speciation data for area source emissions categories. Other sources considered in this study included a Desert Research Institute (DRI) database recently updated as part of the Northern Front Range Air Quality Study (NFRAQS), a DRI database from the 1989-1990 Phoenix Urban Haze Study, and an ARB database. The sources of the speciation profiles selected for the top ten area and on-road mobile source categories are listed in **Table 2-11**. PM emissions from wind erosion and entrained road dust were assigned PM profiles from DRI's Phoenix database, assuming that the soil composition in Houston would be more similar to that in Phoenix than EPA's default soil composition. Furthermore, the DRI profiles are likely to be more accurate than those contained in the EPA SPECIATE database for entrained road dust. The authors recognize this limitation and strongly recommend that in future studies Houston-specific soil and entrained road dust samples be collected.

The sources listed in Table 2-11 are responsible for more than 90 percent of the total PM emissions in the 18-county Houston domain. All other emissions sources were assigned EPA SPECIATE default profiles. However, the profile assignments for other large PM contributors were reviewed and the profile assignments were corrected when necessary. Note that due to a lack of data, identical PM speciation profiles were assigned to both fine and coarse PM without modifications. **Tables 2-12 and 2-13** show the 1993 speciated area, point, and on-road mobile source estimates for the 8-county and 18-county domains, respectively.

2.7 ALTERNATE EMISSIONS PROJECTIONS

In order to examine the influence of changes to ozone precursor emission rates on resultant ozone concentrations, 2007 emission inventories were adjusted downward for several different emissions scenarios. Six different emission reduction scenarios were generated. In each scenario, biogenic emissions were held constant. All six scenarios included a region-wide 15 percent reduction in VOC emissions for all anthropogenic sources. Five of the scenarios reflected an across-the-board NO_x reduction applied to all anthropogenic sources (e.g., 85, 75, 65, 45, and 25 percent reduction of the 2007 baseline NO_x emission inventory). The sixth scenario reflected a clean diesel fuel reformulation policy (described in detail below). That scenario resulted in a 6 percent reduction in area source NO_x emissions and a 2 percent reduction in on-road mobile NO_x emissions.

Sensitivity of the PM and PM-related emission estimates to alternate assumptions was also investigated. Here the emphasis was on alternate growth assumptions between 1993 and 2007. For purposes of this discussion, the PM emission inventory described above is referred to as Scenario 1. Three other future-year emissions scenarios were generated. The emissions scenarios are summarized in **Table 2-14. Figures 2-4 and 2-5** compare the emissions for the 1993 and 2007 emission estimates for these scenarios. The methodology used to develop each new scenario is described below.

2.7.1 Scenario 2 – Population-limited growth

Emissions Scenario 2 was developed using exactly the same procedures and data as used for Scenario 1 except the area source category growth rates were limited to the population growth rate in Scenario 2. This scenario was motivated by some high E-GAS projected growth rates for 1993-2007. The 8-county population growth rate for 1993 to 2007 is estimated as 26 to 28 percent (Houston/Galveston Area Council, personal communication, 1998; Texas Cancer Data Center, 1999, see Section 4). The 28 percent population growth rate was used in Scenario 2. Growth factors from E-GAS were applied to the 1993 inventory to make the 2007 projections. For the emissions source categories that have E-GAS growth factors greater than population growth, the factor was changed to 1.28 (equal to population growth).

The emissions corresponding to Scenario 2 (2007 future year) are listed in **Table 2-15**. Limiting area source growth rates to the level of population growth decreases the area source PM and SO_x emissions by 5 and 17 percent, respectively, compared to Scenario 1 in 2007. The total emissions estimates from all categories and species are still within 5 percent of those for Scenario 1.

2.7.2 Scenario 3 – Mobile and area source adjustments based on chemical mass balance data

PM source apportionment studies in Denver and Phoenix strongly suggest that emission inventories developed using standard EPA emission factors over-estimate dust emissions and under-estimate mobile source emissions. The Scenario 3 alternate emission inventories for 1993 and 2007 were developed to reflect these findings. Scenario 3 projections were carried out using newly available chemical mass balance (CMB) source apportionment estimates from NFRAQS. The results of ambient sampling and CMB analysis in Denver and Phoenix indicate that dust emissions from wind erosion and mechanical processes such as construction activities, paved and unpaved road dust, and agricultural processes, account for 5 to 10 percent of total $PM_{2.5}$ in ambient air. Assuming about half of the ambient $PM_{2.5}$ is primary, then no more than 20 percent of the $PM_{2.5}$ emission inventory should be dust. The NPI estimated $PM_{2.5}$ dust emissions constitute 40 percent of the total $PM_{2.5}$ emissions in the 18-county Houston domain (i.e., in Scenario 1).

The results of NFRAQS also suggest that $PM_{2.5}$ emission factors for light-duty vehicles are significantly higher than those currently used to develop on-road mobile source emission estimates (Watson et al., 1998). They found significant $PM_{2.5}$ contributions from high-emitting vehicles.

The 1993 base-year and 2007 future-year mobile source emissions were adjusted using fleet age distribution data for 1993 and the PM emission factors for pre-1980, post-1980, and high emitting vehicles. It was assumed that 10 percent of the vehicle fleet are high-emitters based on the results of an international remote sensing study (Zhang et al., 1995). The 1993 base-year and 2007 future-year mechanically generated and wind blown dust emissions were adjusted to constitute 20 percent of the total $PM_{2.5}$ emissions. PM_{10} emissions from the

same dust categories were calculated using the 20 percent adjusted $PM_{2.5}$ mass and the ratio of $PM_{2.5}$ to PM_{10} from the categories.

Tables 2-16 and 2-17 list the 1993 base-year and 2007 future-year emissions, respectively, for Scenario 3 reflecting these adjustments. Overall, the emissions are lower. The 1993 base-year and 2007 future-year Scenario 3 mobile source PM emissions are slightly lower than in Scenarios 1 and 2. However, Scenario 3 area source PM emissions are significantly lower due to the large downward adjustment of the dust categories.

2.7.3 Scenario 4 – Mobile and area source adjustments for clean-diesel fuel

Emissions for Scenario 4 are identical to those for Scenario 1 in 2007 except they assume emissions reductions from full implementation of a clean diesel-fuel program similar to the current program in California. The ARB's clean diesel-fuel regulation was implemented in 1993 and has worked quite well (California Air Resources Board, 1997). The ARB estimates that the California clean-diesel fuel program reduces SO_2, PM, and NO_x emissions by 82, 25, and 7 percent, respectively, for engines burning the cleaner diesel fuel. This includes both on- and off-road diesel engines. The relative emissions reductions expected from California's clean-diesel program were applied to the 2007 future-year emission inventory in Houston in order to examine the potential pollution reduction effects of implementing a clean-diesel program in the region.

The 2007 future-year mobile source diesel SO_2, NO_x, PM_{10}, and $PM_{2.5}$ emissions were adjusted according to the emissions reductions for the corresponding pollutants. The 2007 future-year area source emissions were also adjusted for those sources that would be impacted by the fuel change (i.e., off-road construction equipment and farming equipment). **Table 2-18** lists the 2007 future-year emissions corresponding to the Scenario 4 adjustments.

The 2007 future-year Scenario 4 mobile and area source SO_x emission estimates are slightly lower than the other scenarios. However, since point sources are responsible for most of the SO_x emissions, large reductions in area and mobile source SO_x emissions only result in about a 10 percent total reduction in SO_x emissions. On a mass basis, the effects of implementing a clean-diesel fuel program in the Houston domain would amount to a 2 to 5 percent decrease in total PM emissions as compared to Scenario 1. It is important to note that in the Scenario 4 projections, both growth and reduction in PM emissions are occurring simultaneously. While there are reductions in PM from the clean diesel program, the emissions are also growing with time. Based on the emissions totals, the growth predicted in PM emissions outweighs the reductions from the clean diesel program between 1993 and 2007.

2.8 DISCUSSION AND RECOMMENDATIONS

Estimation of emissions on a regional scale is an evolving engineering practice that improves each year, yet still has serious limitations. The baseline and future emission estimates presented here are uncertain and should be used with caution. Most of the inputs to

the emissions calculations are not measured quantities, but rather uncertain estimates of activity levels, emission factors, spatial distributions, temporal variations, seasonal variations, and chemical composition of sources. Future-year emissions are especially uncertain because of obvious uncertainties in future growth rates, economic conditions, and technologies. The alternative emission scenarios developed for PM and PM-related compounds demonstrate potential uncertainties in these estimates. For example, both the amount of the $PM_{2.5}$ mass emissions and relative contribution of major source categories vary by factors of two or more in our Scenarios 1 and 3. Further research is needed to characterize what is actually emitted into the air. Even after twenty years of inventory development and control program implementation, the ozone precursor inventories undergo significant revisions every few years. For PM, the process is just starting and there is probably a long way to go until PM inventories can be relied upon in an absolute sense and for reliable source attribution.

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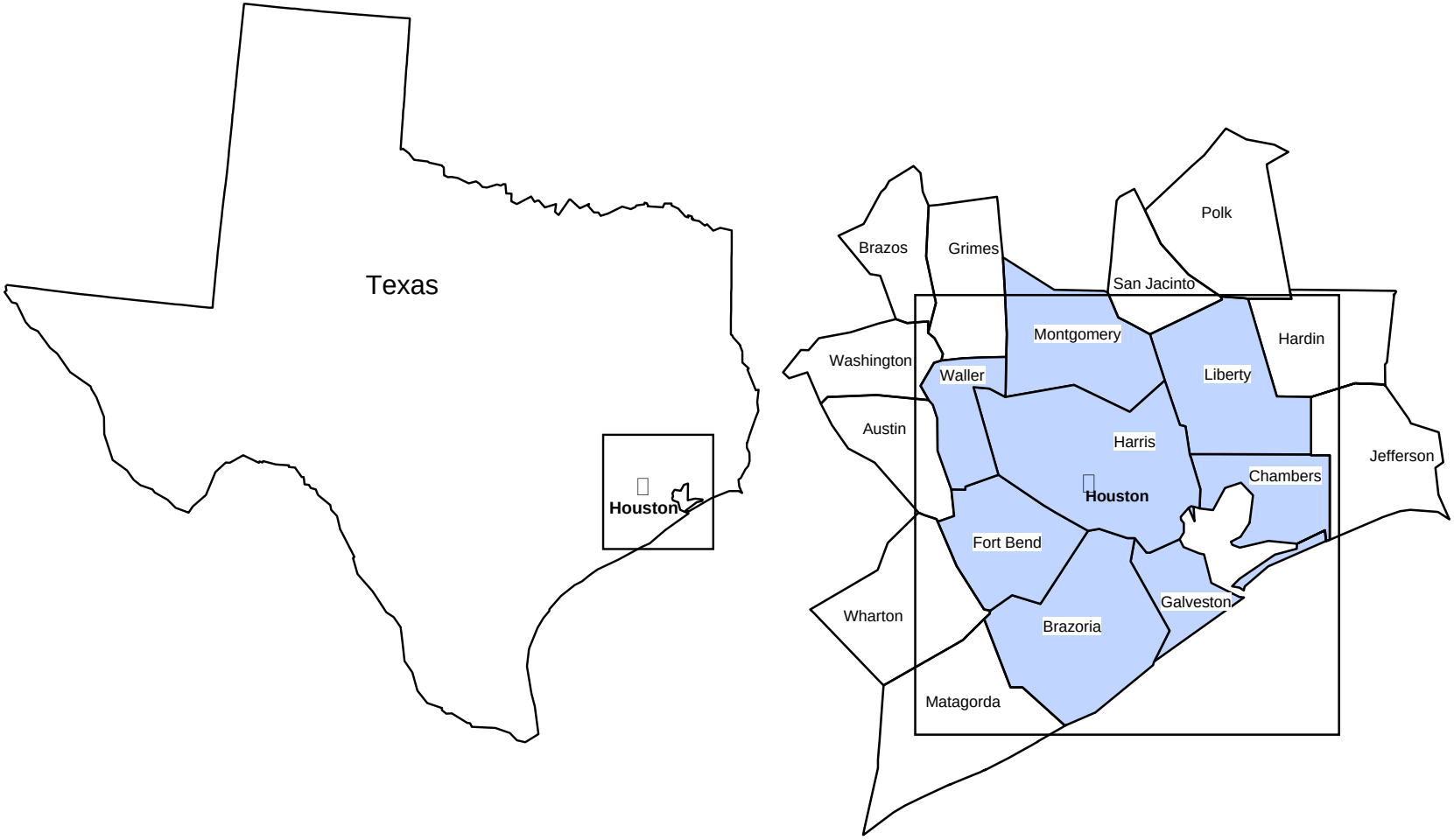

Figure 2-1. Map depicting the 8 counties (shaded ones) and 18 counties included in the Houston area emission inventories. The square depicts the boundary of the exposure modeling domain.

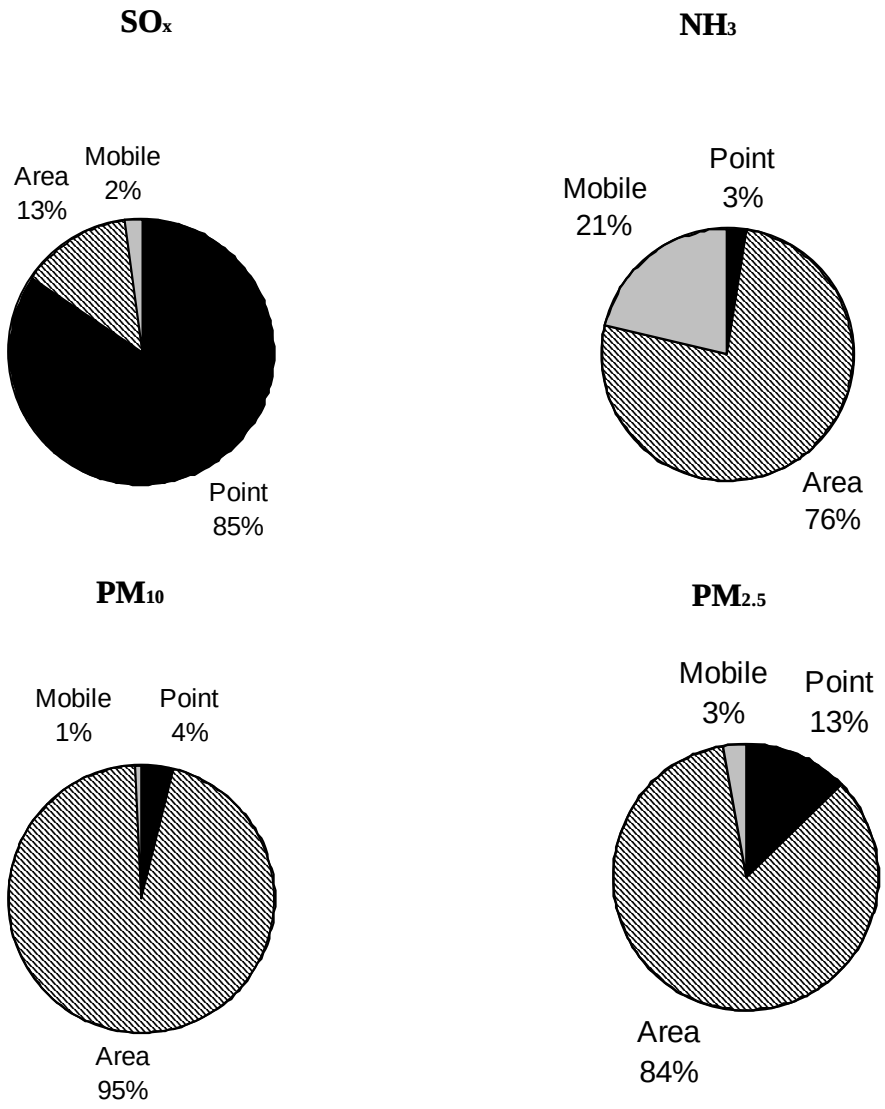

Figure 2-2. Point, area, and on-road mobile source contributions of SO_x, PM_{10}, $PM_{2.5}$, and NH_3 for the 18-county domain surrounding Houston (1990 NPI).

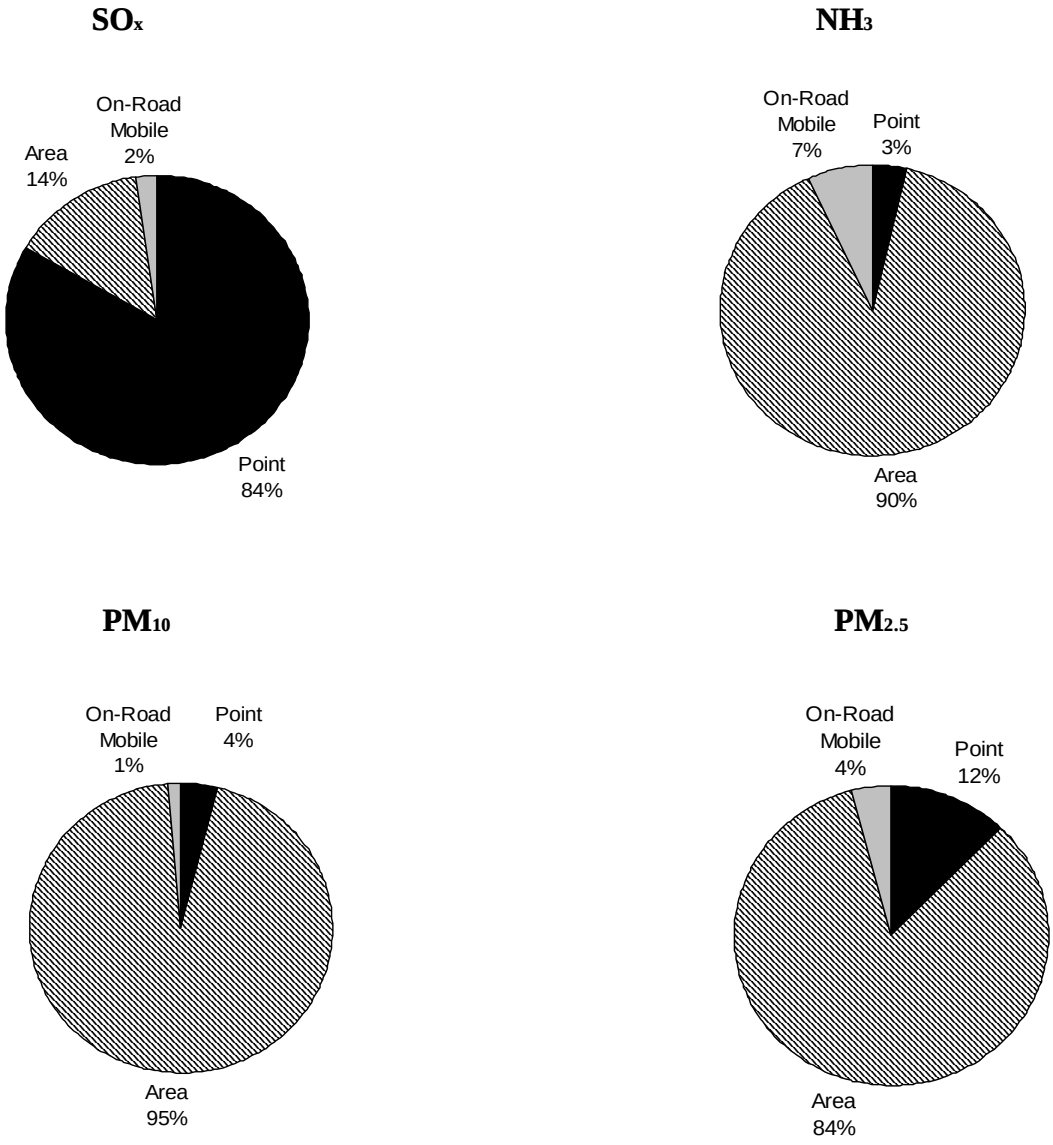

Figure 2-3. Point, area, and on-road mobile source contributions of SO_x, PM_{10}, $PM_{2.5}$, and NH_3 in the revised 1990 NPI grown to 1993 for the 18-county domain.

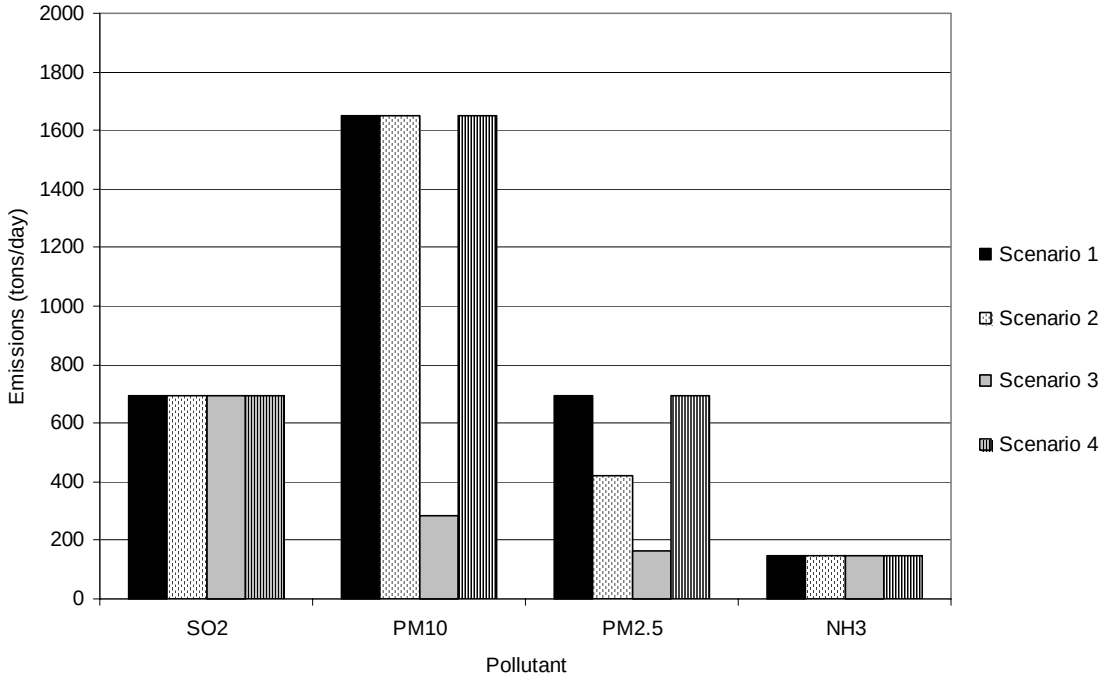

Figure 2-4. Comparison of the 1993 SO_2, PM_{10}, $PM_{2.5}$, and NH_3 emission estimates for the 18-county domain under four scenarios.

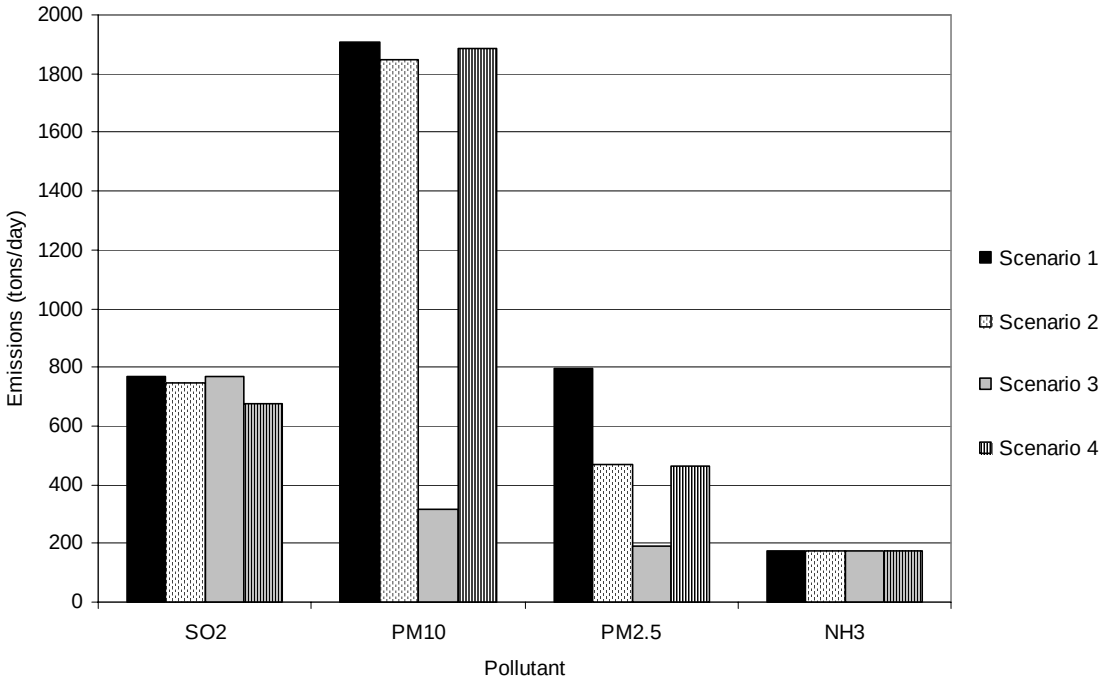

Figure 2-5. Comparison of the 2007 SO_2, PM_{10}, $PM_{2.5}$, and NH_3 emission estimates for the 18-county domain under four scenarios.

Table 2-1. Houston/Galveston area VOC, NO_x, and CO emissions for September 8, 1993, as used in the 1998 SIP.

Species	Emission Inventory (Tons/Day) : September 8				
	Area	Point	Mobile	Biogenics	Totals
VOCs	767	783	410	8,496	10,456
NO_x	501	1,540	768	114	2,931
CO	2,729	6,513	4,155	-	13,397

Table 2-2. Total SO_x, PM_{10}, $PM_{2.5}$, and NH_3 estimates for the 18-county domain and for Harris County alone (1990 NPI).

Region	SO_x (tons/day)	PM_{10} (tons/day)	$PM_{2.5}$ (tons/day)	$PM_{2.5-10}$ (tons/day)	NH_3 (tons/day)
All Counties in Domain	677	1586	400	1186	71
Harris County	217	867	224	643	19
Harris Co. Percent of Total	32%	55%	56%	54%	27%

Table 2-3. Comparison of parameters influencing NH_3 area source emissions in central California and the 18-county Houston region.

Parameter	Central California	Houston (18 counties)	Ratio
Geographic area	65.5 thousand sq. mi.	16.2 thousand sq. mi.	4.0
Cattle population	3.3 million	786 thousand	4.2
Area Source Emissions	518 tons/day	55 tons/day	9.4

Table 2-4. Estimated on-road mobile source emissions for SO_x, PM_{10}, $PM_{2.5}$, and NH_3 in the 18-county area including Houston.

Inventory	Regional Emissions (tons per day)			
	SO_x	PM_{10}	$PM_{2.5}$	NH_3
1990 NPI On-Road Mobile	15	14	11	15
Revised On-Road Mobile	15	21	17	10
Percent Change	0%	35%	35%	-35%

Table 2-5. Houston area VOC, NO_x, and CO emissions (tons per day) for 1993 and 2007.

Species	Area	Point	Mobile	Biogenics	Totals
1993 (SIP Inventory)					
VOCs	767	783	410	8,496	10,456
NO_x	501	1,540	768	114	2,931
CO	2,729	6,513	4,155	-	13,397
1993 (Revised Inventory)					
VOCs	787	724	441	10,248	12,200
NO_x	505	1,545	769	118	2,938
CO	2,729	6,513	4,155	-	13,397
2007 (SIP Inventory)					
VOCs	1,041	887	400	9,894	11,735
NO_x	581	1,620	675	118	2,666
CO	3,222	6,248	3,348	-	12,818
2007 (Revised Inventory)					
VOCs	846	595	121	10,248	11,810
NO_x	585	1,288	282	118	2,273
CO	3,222	6,248	901	-	10,371

Table 2-6. Point, area, and on-road mobile source emissions in 1993 for the 18-county, 8-county, and Harris County domains projected from the 1990 revised inventory using E-GAS default growth factors.

Source Category	Region	Emissions (tons per day)				
		SO_x	NH_3	$PM_{2.5}$	$PM_{2.5-10}$	PM_{10}
Point	18 County	581	5	52	12	64
Area	18 County	100	134	350	1,232	1,563
On-Road Mobile	18 County	16	10	18	4	22
Total	18 County	697	149	420	1,229	1,649
Point	8 County	446	4	41	11	51
Area	8 County	77	29	290	993	1283
On-Road Mobile	8 County	13	10	15	3	18
Total	8 County	537	43	346	1,007	1,353
Point	Harris County	165	2	20	3	23
Area	Harris County	51	12	207	665	873
On-Road Mobile	Harris County	10	7	10	2	12
Total	Harris County	226	21	237	670	908
Harris County Emissions Percent of 18-County Emissions		32%	14%	56%	54%	54%

Table 2-7. Point, area, and on-road mobile source emissions for the 2007 future-year inventory for the 18-county and 8-county domains.

Source Category	Region	Emissions (tons per day)[a]				
		SO_x	NH_3	$PM_{2.5}$	$PM_{2.5-10}$	PM_{10}
Point	18 County	620	5	55	13	68
Area	18 County	128	156	409	1409	1,818
On-Road Mobile	18 County	20	13	19	5	24
Total	18 County	768	174	483	1427	1,910
Point	8 County	489	4	44	11	55
Area	8 County	98	33	335	1130	1465
On-Road Mobile	8 County	17	12	16	4	20
Total	8 County	605	49	395	1,145	1,540

[a] These are the emission estimates for Scenario 1 (see Section 2.7).

Table 2-8. Percent growth in point, area, and on-road mobile source emissions from 1993 to 2007 for the 18-county and 8-county domains.

Source Category	Region	1993 to 2007 Emissions Growth (%)				
		SO_x	NH_3	$PM_{2.5}$	$PM_{2.5-10}$	PM_{10}
Point	18 County	7%	0%	6%	6%	6%
Area	18 County	29%	14%	17%	16%	16%
On-Road Mobile	18 County	28%	23%	6%	17%	8%
Total	18 County	10%	14%	15%	16%	16%
Point	8 County	10%	0%	8%	3%	7%
Area	8 County	27%	12%	15%	14%	14%
On-Road Mobile	8 County	29%	17%	7%	15%	8%
Total	8 County	13%	12%	14%	14%	14%

Table 2-9. Seasonal average point, area, and on-road mobile source emissions in 1993 for the 18-county domain.

Source Category	Season	Seasonal Emissions (tons/day)				
		SO_x	NH_3	$PM_{2.5}$	$PM_{2.5-10}$	PM_{10}
Point	Oct – Mar.	288	3	26	6	32
Area	Oct – Mar.	54	37	130	469	599
On-Road Mobile	Oct – Mar.	8	5	9	2	11
Total	Oct – Mar.	350	45	165	477	642
Point	Apr-Sept.	293	3	26	6	32
Area	Apr-Sept.	46	96	220	744	964
On-Road Mobile	Apr-Sept.	8	5	9	2	11
Total	Apr-Sept.	347	105	255	752	1007
Grand Total	Annual	697	149	420	1229	1649

Table 2-10. Individual PM chemical species selected for the PM speciation database.

Species Name	Species Abbreviation
PM_{10}	PM_{10}
PM Coarse Fraction	PMC
PM Fine Fraction	PMF
Sulfate	SO_4
Organic Carbon	OC
Elemental Carbon	EC
Sodium	Na
Chlorine	Cl
Aluminum	Al
Silicon	Si
Iron	Fe
Calcium	Ca
Potassium	K
Other (all other species in profile)	OTR

Table 2-11. Speciation profile assignments for the top ten emissions source categories in the 1993 base-year and 2007 future-year emission inventories for the 18-county Houston region.

Source Category	Profile Assignment	Profile ID	Reference
Windblown dust	Phoenix desert soil	PHDSSOIL	Watson et al., 1998
Unpaved road dust	Phoenix unpaved road dust (w/low Ca)	PHDUPRD2	Watson et al., 1998
Paved road dust	Phoenix paved road dust	PHPVRD	Watson et al., 1998
Non-road diesel	Vehicular sources-diesel	118	California Air Resources Board, 1994; Lurmann et al., 1997
Open burning	Agricultural burning	136	California Air Resources Board, 1994; Lurmann et al., 1997
Non-road diesel	Vehicular sources-diesel	118	California Air Resources Board, 1994; Lurmann et al., 1997)
Railroad	Vehicular sources-diesel	118	California Air Resources Board, 1994; Lurmann et al.,1997
Marine vessels	Marine vessels	00000	U.S. Environmental Protection Agency, 1998
Non-road gas	Motor vehicle	PHAUTOC	Watson et al., 1998

Table 2-12. Speciated area, point, and on-road mobile source emissions in 1993 for the 8-county domain.

Species	Size	Area	Point	Mobile	Total
PM_{10}	Both	1283	51	18	1353
$PM_{2.5-10}$	Coarse	993	11	3	1007
$PM_{2.5}$	Fine	290	41	15	346
SO4	Coarse	2.2	1.0	0.1	3.4
SO4	Fine	3.6	6.3	0.7	10.6
OC	Coarse	145.3	1.0	1.5	147.9
OC	Fine	70.1	6.1	4.2	80.4
EC	Coarse	3.9	0.2	1.1	5.1
EC	Fine	14.2	5.9	8.3	28.3
NA	Coarse	0.1	0.3	0.0	0.4
NA	Fine	0.2	0.9	0.1	1.2
CL	Coarse	5.9	0.4	0.0	6.2
CL	Fine	1.9	1.4	0.0	3.4
AL	Coarse	133.4	1.6	0.1	135.0
AL	Fine	31.4	2.3	0.1	33.9
SI	Coarse	382.5	3.4	0.1	386.0
SI	Fine	90.1	5.4	0.3	95.8
OTR	Coarse	313	2.8	0.4	316.2
OTR	Fine	77	12	1.1	90.1

Table 2-13. Speciated area, point, and on-road mobile source emissions in 1993 for the 18-county domain.

Species	Size	Area	Point	Mobile	Total
PM_{10}	Both	1587.27	64.28	21.70	1673.25
$PM_{2.5-10}$	Coarse	1232.77	12.09	3.86	1248.71
$PM_{2.5}$	Fine	354.97	52.21	17.84	425.02
SO4	Coarse	2.84	1.2	0.13	4.17
SO4	Fine	4.03	11.59	0.85	16.47
OC	Coarse	182.93	1.16	1.81	185.89
OC	Fine	85.05	7.09	4.97	97.11
EC	Coarse	4.57	0.19	1.31	6.08
EC	Fine	16.91	5.96	10.03	32.90
NA	Coarse	0.10	0.38	0.01	0.49
NA	Fine	0.30	0.97	0.12	1.39
CL	Coarse	7.48	0.39	0.01	7.88
CL	Fine	3.02	1.46	0.01	4.49
AL	Coarse	164.86	1.79	0.09	166.75
AL	Fine	38.16	2.93	0.18	41.27
SI	Coarse	473.84	3.74	0.08	477.66
SI	Fine	109.63	6.27	0.35	116.25
OTR	Coarse	380.69	3.21	0.42	384.32
OTR	Fine	92.63	15.74	1.33	109.70

Table 2-14. Summary of the four alternative emission inventory scenarios.

Scenario ID	1993 Base-Year Emissions	2007 Future-Year Emissions
Scenario 1 (Base-year inventories for 1993 and 2007)	**Point**: Developed using E-GAS default growth from 1990-1993. **Area**: Developed using E-GAS default growth from 1990-1993. **Mobile**: Developed using VMT growth for 1990-1993 data from HGAC for Harris and adjacent counties and E-GAS default VMT growth for remaining counties.	**Point**: Projected from 1993 using E-GAS default growth factors. **Area**: Projected from 1993 using E-GAS default growth factors, natural dust categories assigned a growth factor of 1 (no growth). **Mobile**: Projected using VMT growth for 1993-2007 data from HGAC for Harris and adjacent counties and E-GAS default VMT growth for remaining counties. On-road heavy-duty diesel emissions adjusted based on new Federal PM emission factor standards.
Scenario 2 (Area source growth limited to population growth)	Same as Scenario 1 base-year emissions.	**Point**: Same as Scenario 1 future-year emissions. **Area**: Projected from 1993 emissions growth limited to 28% population growth, natural dust categories assigned a growth factor of 1 (no growth). **Mobile**: Same as Scenario 1 future-year emissions.
Scenario 3 (Adjustments to $PM_{2.5}$ based on NFRAQS work; lowering dust emissions and adjusting on-road mobile source $PM_{2.5}$ using new emission factors.)	**Point**: Same as Scenario 1 base-year emissions. **Area**: Developed using E-GAS default growth from 1990-1993. Dust category $PM_{2.5}$ emissions adjusted to 20% of total $PM_{2.5}$ emissions. **Mobile**: Developed using VMT growth for 1990-1993 data from HGAC for Harris and adjacent counties and E-GAS default VMT growth for remaining counties. On-road emissions adjusted using newly available $PM_{2.5}$ emission factors and fleet turnover for 1993.	**Point**: Same as Scenario 1 future-year emissions. **Area**: Developed using E-GAS default growth from 1993-2007. Dust category $PM_{2.5}$ emissions grown and adjusted to 20% of total $PM_{2.5}$ emissions. **Mobile**: Developed using VMT growth for 1993-2007 data from HGAC for Harris and adjacent counties and E-GAS default VMT growth for remaining counties. On-road emissions adjusted using newly available $PM_{2.5}$ emission factors and fleet turnover for 2007.
Scenario 4 (Emissions effects of a clean diesel fuel program)	Same as Scenario 1 base-year emissions.	**Point**: Same as Scenario 1 future-year emissions. **Area**: Developed using E-GAS default growth from 1993-2007. Categories using diesel fuel adjusted based on clean diesel-fuel program. **Mobile**: Developed using VMT growth for 1993-2007 data from HGAC for Harris and adjacent counties and E-GAS default VMT growth for remaining counties. On-road diesel vehicle emissions adjusted based on the emissions reductions from a clean diesel-fuel program.

Table 2-15. Scenario 2 point, area, and on-road mobile source emissions for the 2007 future-year inventory for the 18-county and 8-county domains.

Source Category	Region	Emissions (tons per day)				
		SO_x	NH_3	$PM_{2.5}$	$PM_{2.5-10}$	PM_{10}
Point	18 County	620	5	55	13	68
Area	18 County	109	65	395	1,362	1,757
On-Road Mobile	18 County	20	13	19	5	24
Total	18 County	749	83	469	1,380	1,849
Point	8 County	489	4	44	11	55
Area	8 County	84	21	324	1097	1422
On-Road Mobile	8 County	17	12	16	4	20
Total	8 County	591	37	384	1,112	1,496

Table 2-16. Scenario 3 point, area, and on-road mobile source emissions for the 1993 base-year inventory for the 18-county and 8-county domains.

Source Category	Region	Emissions (tons per day)				
		SO_x	NH_3	$PM_{2.5}$	$PM_{2.5-10}$	PM_{10}
Point	18 County	581	5	52	12	64
Area	18 County	100	134	94	102	196
On-Road Mobile	18 County	16	10	16	8	24
Total	18 County	697	149	162	122	284
Point	8 County	446	4	41	11	51
Area	8 County	77	55	75	75	150
On-Road Mobile	8 County	14	10	18	2	20
Total	8 County	537	69	134	88	222

Table 2-17. Scenario 3 point, area, and on-road mobile source emissions for the 2007 future-year inventory for the 18-county and 8-county domains.

Source Category	Region	Emissions (tons per day)				
		SO_x	NH_3	$PM_{2.5}$	$PM_{2.5-10}$	PM_{10}
Point	18 County	620	5	55	13	68
Area	18 County	128	156	114	110	224
On-Road Mobile	18 County	20	13	21	3	24
Total	18 County	768	174	190	126	316
Point	8 County	489	4	44	11	55
Area	8 County	98	63	92	82	174
On-Road Mobile	8 County	17	12	18	2	21
Total	8 County	605	79	154	95	249

Table 2-18. Scenario 4 point, area, and on-road mobile source emissions for the 2007 future-year inventory for the 18-county and 8-county domains.

Source Category	Region	Emissions (tons per day)				
		SO_x	NH_3	$PM_{2.5}$	$PM_{2.5-10}$	PM_{10}
Point	18 County	620	5	55	13	68
Area	18 County	40	155	393	1,407	1,800
On-Road Mobile	18 County	17	13	15	4	19
Total	18 County	677	173	463	1,424	1,887
Point	8 County	489	4	44	11	55
Area	8 County	33	63	322	1129	1450
On-Road Mobile	8 County	15	12	13	3	16
Total	8 County	537	79	378	1,143	1,521

3. AIR QUALITY CONDITIONS

Houston specific air quality information is essential for characterization of the population's exposure to air pollution exposure. In this section, the baseline and estimated future-year air quality conditions for ozone, $PM_{2.5}$, and selected toxic air contaminants are presented.

3.1 BASELINE OZONE CONCENTRATIONS

Ambient air quality conditions in the Houston area exceed the 1-hr and 8-hr ozone NAAQS on numerous days in the March through October smog season. On the worst days of the year, 1-hr maximum ozone concentrations are approximately twice the level of the standard. The frequency of occurrence of concentrations above the 1-hr NAAQS (at one or more air monitoring stations) was between 31 and 59 days per year in the 1992-1995 time period (see **Table 3-1**). The 8-hr NAAQS was exceeded between 15 and 45 days per year in this same period. In 1995, there were 45 days with 1-hr maximum concentrations above 160 ppb and one day with a 1-hr maximum of 244 ppb. Both the frequency and severity of ozone standard exceedances are of concern to public health officials in Texas.

Figure 3-1 compares the frequency of high ozone days in Houston and Los Angeles. Los Angeles has the worst ozone air quality conditions in the United States. Although Los Angeles has more 1-hr NAAQS exceedances than Houston, ozone air quality conditions in the two areas appear to be converging. The Houston data show only a modest downward trend in the number of 1-hr exceedance days between 1985 and 1997, whereas the data for Los Angeles show a strong downward trend. The slow rate of improvement in Houston ozone air quality is of concern given the Federal Clean Air Act's requirement to achieve compliance with the ozone NAAQS by 2007.

The Texas Natural Resource Conservation Commission (TRNCC) carried out air quality analyses for the 1998 SIP that characterized the ozone problem in the Houston area and estimated the amounts of VOC and NO_x emission reductions that are required in order to achieve compliance with the 1-hr NAAQS by 2007 (Texas Natural Resources and Conservation Commission, 1998). The photochemical modeling database and applications developed for the Houston SIP represent the most reliable methods for estimating ozone concentrations in future years under alternate emission scenarios in the area. The approach we adopted for this benefits study involves contrasting benefits of improved air quality conditions in future years. Because the photochemical modeling was well developed for the Houston area, we designed the benefits study to use the tools and databases established by TNRCC for Houston. For consistency, we used the same years for planning as did the TNRCC: 1993 for baseline conditions and 2007 for the future year.

The Houston SIP modeling focused on the September 6-11, 1993, ozone episode for the attainment demonstration. This was one of the two worst ozone episodes in 1993 and represents an extreme in population exposure to ozone. By design, attainment demonstration

analyses must focus on the worst case conditions. However, unhealthful ozone concentrations occur on many days of the year in Houston and the approach used for the benefits study was designed to capture a broader set of days when the population was exposed to moderate and high levels of ozone. Because of year-to-year variability in meteorological and ozone conditions, we sought to pick three consecutive years that included 1993 as a baseline. All days in 1992-1995 with a 1-hr maximum ozone concentration equal to or greater than 80 ppb at one or more stations in the Houston/Galveston nonattainment area were identified. There were 322 and 336 days that met the criteria in 1992-1994 and 1993-1995, respectively. The 1993-1995 period was selected because it has a slightly higher frequency of potentially unhealthful days than the 1992-1994 period. Only the days on which concentrations were equal to or greater than 80 ppb at one or more stations in 1993-1995 were included in the baseline ozone air quality database. The database included all days with exceedances of the 1-hr and 8-hr ozone NAAQS, plus some cleaner days.

Urban ozone concentrations tend to have high spatial and temporal variability. The Houston air monitoring network is able to capture a reasonable amount of this variability. For the period of interest, the monitoring network consisted of 14 routine monitoring stations that collected data for the whole period (1993-1995) and 12 special monitoring stations that collected data only for the summer of 1993 (as part of the COAST Study). The locations of the stations are shown in **Figure 3-2**. Houston's routine monitoring network was designed for monitoring compliance with the ozone NAAQS and is probably sufficient for that purpose. However, the network was not designed for general population exposure assessment and lacks spatial coverage in certain areas. It certainly does not provide uniform coverage over the whole area. The sparsity of observational data may limit the accuracy of ambient exposure assignments in portions of the study area. Exposure assignments were made for residents that lived within 35 km of a routine or special study ozone monitoring station. The estimates of air quality conditions may be quite uncertain for residents living outside of the perimeter of the monitoring network or within the perimeter, but more than 20 km from a station.

Several methods are available for assignment of ambient exposure concentrations to the population and spatial mapping of the ambient air quality concentrations. In most epidemiologic studies, an average concentration determined from two or three "representative" monitoring stations is assigned to an entire urban population. This approach ignores the strong spatial gradients in urban ozone concentrations and is not appropriate for this study. In numerous exposure model applications (Johnson et al., 1990; Lurmann et al.,1989), the population is assigned the concentrations from the nearest monitor. This method at least uses the data from all stations, but often produces fields with sharp discontinuities. In most applications of the Regional Human Exposure (REHEX) Model, the ambient monitoring data were spatially interpolated to 10 x 10 km grids using inverse distance squared weighting (Lurmann et al., 1991; Lurmann and Korc, 1994a,b; Korc, 1996). For this study, we elected to use finer resolution (4 x 4 km) than previous applications and to use Kriging to spatially map the ambient concentration fields. Kriging is generally recognized as a more accurate spatial mapping technique than inverse distance squared interpolation.

Hopkins et al. (1999) investigated different spatial mapping techniques for ozone in the Houston area and found that three-dimensional Kriging (x, y, and time) of the square root of ozone with exponential variograms was slightly more accurate than simple two-dimensional Kriging. The third dimension, time, incorporated data for the 3 hrs before and 4 hrs after the hour of interest for a particular field. We felt the space and time Kriging was somewhat impractical for our application that required the generation of 8064 hourly concentration fields. Hence, the mapping was performed by Kriging the two-dimensional hourly fields of ozone square root using an exponential variogram and an inverse multi-quadratic basis function. The SURFER® mapping software was used to map and display the fields. For quality assurance purposes, all fields were visually examined prior to transformation and use. The Kriged square root fields were then transformed (squared) for use in the exposure modeling database. The mapping was performed for a larger domain (320 x 240 km) than the 180 x 188 km domain used for the Houston exposure assessment. This allowed for data from outlying monitoring stations to be included in the mapping inputs. There were no data offshore. A background ozone level of 50 ppb was assumed to exist about 30 km offshore. The estimated ozone concentrations in the populated coastal areas (Galveston and Texas City) are not significantly affected by the assumed offshore ozone level when coastal measurements are available (i.e., in 1993 for Galveston and 1993-95 for Texas City). The estimated ozone in the Galveston area in 1994 and 1995 are less certain than other regions because of the lack of measurements. Samples of the hourly fields are shown in **Figure 3-3**. The average 1-hr daily maximum concentrations on these days are shown in **Figure 3-4**. The latter figure illustrates that the area north and northwest of central Houston experiences the highest daily maximum ozone, on average.

3.2 FUTURE OZONE CONCENTRATIONS

Ozone concentration estimates for several future-year scenarios are needed for quantification of benefits associated with improved air quality. We primarily seek to assess the benefits of reducing ozone concentrations from the future-year baseline conditions to the NAAQS level. The future-year baseline scenario represents the conditions that will occur without implementation of additional emission control measures beyond those accepted by 1996 (see TNRCC, 1998). The future-year compliance scenario represents conditions with sufficient (but unspecified) emissions reductions to achieve compliance with the NAAQS. As in the 1998 SIP, this compliance scenario focuses on the 1-hr NAAQS rather than the 8-hr NAAQS.

The HAXL oversight committee also expressed interest in quantification of the benefits of specific levels of reductions in region-wide emissions. The additional 2007 scenarios considered here include the:

- 85 percent anthropogenic NO_x emissions reduction plus 15 percent anthropogenic VOC emissions reduction, which are the 1998 SIP estimates of the amounts of reductions needed to achieve compliance with the 1-hr NAAQS,

- Four lower levels of anthropogenic NO_x emission reductions (75, 65, 45, and 25 percent) in combination with the 15 percent anthropogenic VOC emissions reductions, and
- Substitution of clean burning diesel fuel in all stationary and mobile diesel engines in combination with the 15 percent anthropogenic VOC emissions reductions.

These scenarios represent a reasonable range of conditions for future ozone levels.

3.2.1 Photochemical Modeling of Episodic Conditions

The most scientifically accepted method of estimating future ozone concentrations involves application of a regional photochemical model with future-year emissions and historical meteorological conditions. For the 1998 SIP, the TNRCC applied the UAM-V photochemical model for baseline and future-year conditions. They modeled two episodes and obtained acceptable baseline model performance only for the September 6-11, 1993, episode. Hence, all future-year scenarios were evaluated using the conditions for the September episode in the 1998 SIP. Since the release of the SIP, the TNRCC has indicated a preference for using the CAMx photochemical model rather than the UAM-V model, and has made improvements in Houston emission inventories.

The TNRCC provided STI with the modeling database and updated emissions files for use in this study. New 1993 baseline simulations were made using the CAMx model and the improved biogenic emission estimates. As shown in **Table 3-2**, the maximum ozone estimates for the last four days of the 1993 episode are similar in the UAM-V and CAMx simulations. Note that the results from the first two days of the simulation are not used because these are "spin-up days" required to initialize the model properly and assure that the simulation is "emissions-driven" rather than "initial concentrations-driven." The estimates agree with one another within ± 5 ppb and show a tendency for slightly lower ozone in the new CAMx simulations with improved biogenic emission rates. Both simulations underestimated the highest ozone value observed during this episode (214 ppb on September 8, 1993). The other measures of model performance, such as the normalized mean bias and normalized mean error, indicated the new simulations do not agree with observations quite as well as the original SIP simulations, yet still meet EPA's model performance goals. The CAMx model was also run with the original SIP emission inputs and the results showed that the UAM-V and CAMx models gave very similar estimates with identical inputs. The spatial patterns of estimated ozone concentrations in the UAM-V and CAMx runs showed slightly larger differences than those shown for the peak values.

New 2007 baseline simulations were made using the same estimated point and area source emission estimates used for the 1998 SIP and with revised mobile source and biogenic emission estimates (all provided by the TRNCC). The new mobile source emission estimates for 2007 are lower than the ones used in the 1998 SIP. The rural biogenic emissions are higher and urban biogenic emissions are lower in the new inventory than in the former inventory. **Table 3-3** compares the maximum estimated ozone concentrations in the new

CAMx simulations with those reported in the 1998 SIP. Table 3-3 shows that the new simulations have slightly lower future-year ozone concentrations than the older simulations. The maximum ozone estimates for the 2007 baseline are 2 to 11 ppb lower and 1 to 5 percent lower than the 1993 estimates in the new CAMx runs. In the older simulations, the 2007 baseline estimates were both higher and lower than the corresponding estimates for 1993. The simulated 2007 baseline spatial patterns of ozone concentrations in the new simulations are similar to those for 1993. These results indicate that the expected increase in ozone due to population growth (28 percent) between 1993 and 2007 is approximately offset by the effects of the baseline emission control program.

Substantial emission reductions will be needed to bring the Houston area into compliance with the ozone standards. To achieve compliance with the 1-hr standard, the fourth highest ozone value in 1993-1995 of 204 ppb would need to be reduced to 124 ppb. This represents a 39 percent decrease in the maximum ozone concentration or, if you consider the background tropospheric ozone to be 50 ppb in summer, this represents a 52 percent decrease in ozone in excess of the background.

Table 3-4 shows the estimated maximum ozone concentrations for various 2007 emissions scenarios. **Tables 3-5 and 3-6** show the percentage reduction in maximum ozone concentrations for the various scenarios compared to the 2007 baseline and compared to 1993, respectively. The results suggest that a region-wide VOC emissions reduction of 15 percent combined with an 85 percent NO_x reduction would reduce the maximum ozone by 31, 35, 35, and 41 percent on September 8, 9, 10, and 11, respectively, from the 2007 baseline estimates. Comparing the simulated ozone maxima for the 85 percent NO_x reduction case to the 1993 simulated maxima shows decreases of 32, 38, 37, and 44 percent on September 8, 9, 10, and 11, respectively. The simulated effects of the large emissions reduction increase with time during the episode and achieve the 39 percent reduction needed for compliance only on the last day of the episode.

The results for the other scenarios show smaller decreases in maximum ozone. The results for September 11 suggest that a region-wide VOC emissions reduction of 15 percent combined with a 75, 65, 45, and 25 percent NO_x reduction would reduce the maximum ozone by 33, 25, 15, and 9 percent from the 1993 estimates and by 30, 21, 10, and 4 percent from the 2007 baseline estimates, respectively. The estimated reductions in maximum ozone associated with the use of clean diesel fuel are comparable to those for 25 percent NO_x reduction; reductions of up to 4 percent compared to 2007 baseline levels are estimated. Overall, these results confirm the 1998 SIP analyses indicating that upwards of 85 percent NO_x emission reductions, along with the 15 percent VOC reductions that are already planned, appear to be needed to reach compliance.

3.2.2 Estimation of Future Ozone Throughout the Smog Season

For the purpose of assessing the benefits of ozone air quality improvements, one would like to apply a regional photochemical model to the whole smog season and probably for

multiple years. Specifically, we wanted a method to estimate hourly, spatially resolved future concentrations on the 336 days included in the 1993-1995 database. Research is underway exploring methods to simulate ozone for the entire smog season (Wheeler et al., 1999), but such modeling is not well established. Month-long simulations with coarse resolution (16 x 16 km) have been made for the Texas area (ENVIRON, 1997), however, the model performance was not adequate to establish the simulation's credibility for estimating ozone on the urban scale (the results were only used for boundary conditions). Currently, there are no regional photochemical modeling systems that have demonstrated acceptable model performance for seasonal simulations in the Houston area (or any other area of the county). The development of such a system would take years and substantial resources to complete.

Simpler methods must be used to estimate future ozone concentrations throughout the smog season. We chose to apply a rollback model that relies on changes in the maximum concentration in excess of the background concentration to estimate future concentrations. For the case of compliance with the 1-hr NAAQS, the future concentrations were estimated from the 1993-1995 baseline concentrations using the rollback equations shown below with a factor (F) of 0.48 for all hours of the day. This factor represents the reduction of the fourth highest observed concentration in 1993-1995 (204 ppb) to 124 ppb assuming a background concentration of 50 ppb. The rollback method is only applied to concentrations equal to or greater than the background concentrations. Concentrations below the background are assumed to be the same in the future year as in the baseline period.

$$C_{future}^{x,y,t} = C_{backgrd} + F\left(C_{baseline}^{x,y,t} - C_{backgrd}\right) \quad \textbf{(3-1)}$$

$$F = \frac{C_{future}^{\max} - C_{backgrd}}{C_{baseline}^{\max} - C_{backgrd}} \quad \textbf{(3-2)}$$

where:

$C_{backgrd}$ = background concentration

$C_{baseline}^{x,y,t}$ = hourly gridded concentration in baseline time period

$C_{future}^{x,y,t}$ = hourly gridded concentration in future-year time period

$C_{baseline}^{\max}$ = ozone compliance design value for baseline period

$C_{future}^{\max}$ = ozone standard compliance concentration in future years

The future concentrations for the various 2007 emissions scenarios were also estimated with the rollback approach using rollback factors derived from the photochemical simulation results for September 8-11. In the absence of a seasonal ozone model, we believed a rollback approach based on the relative response of an episodic model to emission changes is probably the next best approach for projecting air quality changes throughout the smog season. Again,

we relied on estimated changes in the domain-wide maximum concentration to project future-year conditions. However, instead of using a single rollback factor for all hours of the day, we used hourly factors. The maximum simulated ozone in excess of the background for a particular hour in the future run was compared to that in the 1993 run and an average ratio for each hour was determined by averaging the daily ratios. **Table 3-7** lists the average ratio of future-year ozone maxima to base-year ozone maxima by hour for the seven 2007 scenarios. **Table 3-8** lists the average ratio of future-year ozone in excess of the background to that of the base year for the seven scenarios. While the hourly factors have little variability for the 2007 baseline case, they show significant variability for the 2007 scenarios with substantial emission reductions. The factors listed in Table 3-8 were applied to the spatially mapped hourly 1993-1995 baseline concentrations.

It is important to emphasize that the rollback approach is overly simplistic and was selected because suitable modeling databases were not available for whole ozone seasons. There is potential for this rollback method to either underestimate and overestimate the benefits of emission reductions. Often the peak ozone is less sensitive than the average ozone to emission changes, especially with VOC dominated control programs. Application of the rollback method based on changes in the peak concentration may be more likely to underestimate the benefits of controlling NO_x and VOC emissions in the Houston area than more scientifically robust methods. Also, local emission control programs may be more effective if background ozone concentrations decrease in future years as a result of region-wide emissions controls. Our approach has been to estimate the benefits of local controls conservatively and avoid assumptions about effective of regional emission controls. Future research is needed to establish reliable databases and models to estimate the effects of emission control strategies on ozone over relevant time periods of interests for health effects assessment.

3.3 BASELINE $PM_{2.5}$ CONCENTRATIONS

Most of the ambient PM concentration data for the Houston area are PM_{10} rather than $PM_{2.5}$. Ambient PM_{10} concentrations have been monitored since the promulgation of the PM_{10} standard in 1986. During this period the PM_{10} concentrations have been less than the 150 $\mu g/m^3$ 24-hr NAAQS and the 50 $\mu g/m^3$ annual average NAAQS. Hence, the area is in compliance with the standards and classified as a PM_{10} attainment area. There has been little attention given to PM air pollution in the region other than requiring new or modified sources to use the best available control technology to minimize PM emissions.

The Federal government promulgated new standards for $PM_{2.5}$ in 1997 and also revised the PM_{10} standards. The new standards require that:

- 24-hr average $PM_{2.5}$ concentrations do not exceed 65 $\mu g/m^3$ for a 3-year average of annual 98^{th} percentiles at any population-oriented site in a monitoring area, and

- 3-year annual average $PM_{2.5}$ concentrations do not exceed 15 $\mu g/m^3$ from a single community-oriented site or the spatial average of eligible community-oriented sites in a monitoring area.

These health-based $PM_{2.5}$ standards are more stringent than the prior PM_{10} standards for most areas. Annual average $PM_{2.5}$ concentrations are typically 45 to 65 percent of the PM_{10} concentration, yet the new annual standard is only 30 percent of the old annual PM_{10} standard. Many urban areas (like Houston) that are in compliance with the PM_{10} standard may exceed the new $PM_{2.5}$ standards.

In anticipation of the new $PM_{2.5}$ standards, a consortium of interested parties [City of Houston, TNRCC, and the Houston Regional Monitoring Corporation (HRM)] initiated a 1-year $PM_{2.5}$ monitoring study in 1997 to identify areas of Texas that might have compliance problems with the new standards. $PM_{2.5}$ mass and chemical components were measured at seven stations in the Houston area from March 1997 through February 1998 (Price et al., 1998). The ambient $PM_{2.5}$ mass data were collected once every sixth day and the $PM_{2.5}$ chemical composition was analyzed in about one third of the samples collected (or 1 out of every 18 days). Data were also collected less frequently at Galveston and Mauriceville to characterize $PM_{2.5}$ in outlying areas from Houston. The locations of the $PM_{2.5}$ monitoring sites are shown in **Figure 3-5**.

There is no gold standard for $PM_{2.5}$ measurements. For purposes of assessing compliance with the NAAQS, measurements must be obtained using a Federal Reference Method (FRM) sampler. These samplers provide an operational definition of $PM_{2.5}$, which may or may not agree with research samplers. FRM samplers were not available at the start of the special 1-year monitoring program. Desert Research Institute (DRI) samplers, that were configured to emulate the expected FRM mass samplers (no backup filters), were used at all sites and additional roving collocated DRI samplers with backup filters were operated at two sites. The standard samplers were operated without a denuder to remove condensable gases and the collocated samplers were operated with and without anodized aluminum denuders on an alternating month basis. The sponsors provided early access to these data for use in this study.

Prior to using the 1997-1998 $PM_{2.5}$ data, the mass concentrations were adjusted for volatilization losses. The standard samplers lose part of the $PM_{2.5}$ mass due to volatilization of ammonium nitrate and/or organic species from the main filter (or front filter). Fortunately, the backup filters from the two collocated samplers were analyzed for nitrate on a once-every-18-days basis. These data show that the front filters only retained 41 percent of the total nitrate on average. It is safe to assume the backup filter nitrate is ammonium nitrate, because other nitrates (such as sodium nitrate) are usually not sufficiently volatile to be lost from the front filter. On average, the collocated samplers' backup filter ammonium nitrate is 7.2 percent of the front filter $PM_{2.5}$ mass. Hence, all of the $PM_{2.5}$ mass, nitrate, and ammonium data from the standard samplers were adjusted for these losses (using the 7.2 percent of mass adjustment). The magnitude of these ammonium nitrate volatilization losses are similar to those found in other warm areas (e.g., Los Angeles in the summer) and

are significant enough to warrant quantification in future sampling programs. No data were available for characterization of organic species losses and no adjustments were made, even though the sampling methods are subject to positive artifacts and negative volatilization losses. Also, no adjustments were made for artifacts arising from the absence of denuders because their effects were small.

There is concern that the upward adjustment of the $PM_{2.5}$ data to account for NH_4NO_3 volatilization losses may exaggerate $PM_{2.5}$ conditions in Houston relative to what might be measured with an FRM sampler. Our view is that the backup filters clearly show that the extra NH_4NO_3 was in the air and, considering the importance of NO_x emission controls for ozone in the area, omission of the adjustment for volatilization losses would be misleading and result in significant underestimation of the benefits of the NO_x emission controls on $PM_{2.5}$ in the region.

The special study $PM_{2.5}$ data were also collected without corresponding nitric acid and ammonia gas measurements. Given the importance of NO_x as an ozone precursor and the potential importance of NO_x as a $PM_{2.5}$ precursor, it would have been particularly useful to measure these species in order to determine whether conditions for ammonium nitrate aerosol formation were nitric acid-limited or ammonia-limited in the Houston area.

The adjusted annual average and 24-hr maximum $PM_{2.5}$ mass concentrations are shown in **Table 3-9**. The annual concentrations range from a low of 12.4 $\mu g/m^3$ at Galveston to a high of 18.6 $\mu g/m^3$ at Clinton. The 24-hr maximum concentrations range from 42 $\mu g/m^3$ at the HRM03 site to 25 $\mu g/m^3$ at Mauriceville in Beaumont. The annual average concentrations exceed the level of the annual NAAQS (15 $\mu g/m^3$), whereas the 24-hr maximum concentrations are well below the level of the 24-hr NAAQS (65 $\mu g/m^3$). It is important to recognize that these data are insufficient to assess compliance with the NAAQS because they were not collected with FRM samplers and they were not collected for a long enough time period (i.e., less than 3 years). Nevertheless, they show two important characteristics. First, the data indicate that there is a relatively high background concentration in and around Houston (and elsewhere in Texas). Second, the data indicate that the Houston area is likely to exceed the annual $PM_{2.5}$ NAAQS.

Assessing the background concentration is important for understanding the $PM_{2.5}$ problem and estimating how changes in PM and PM-related emissions may affect ambient $PM_{2.5}$ levels. When the network was designed, the Galveston and Mauriceville sites were expected to collect data that would be most useful for assessing the Houston background $PM_{2.5}$. The lowest annual average concentrations were observed at Galveston in the study. Annual concentrations at Mauriceville were almost as high as at Shell Westhollow. However, direct comparison of the background site averages with the main network site averages is confounded by differences in sampling days; fewer days were sampled at Galveston and Mauriceville than at the other sites. Another way to examine background levels is to consider the lowest concentration observed at any site on a given day to represent the background or the $PM_{2.5}$ level in air blowing into the region. The average lowest daily concentration at these sites is 12.4 $\mu g/m^3$, which is the same as the average based on fewer days at Galveston. For mapping and projection purposes, we used 90 percent of the lowest daily concentration. For annual average assessment, we used 11.2 $\mu g/m^3$ which is 90 percent of the average lowest

concentration (and also 90 percent of the Galveston level). The rationale for using 90 percent rather than 100 percent is that even the site with the lowest concentration on any given day may be influenced by air recirculation and local sources within the greater Houston domain of interest for this study.

The daily and annual average 1997-1998 $PM_{2.5}$ mass concentrations were spatially mapped to a 4 x 4 km resolution grid for the greater Houston area. This is the same 180 x 188 km exposure domain and grid that was used for the baseline ozone concentrations. The mapping was performed by Kriging the two-dimensional daily and annual fields using an exponential variogram and an inverse multi-quadratic basis function. The SURFER® mapping software was used. The background value (daily or annual) was assumed to occur at locations about 30 km beyond the boundaries of the domain, which produced spatial patterns that tapered-off gradually between the network and the boundaries of the domain. **Figure 3-6** shows isopleths of the annual average $PM_{2.5}$ concentrations. It shows a somewhat bulls-eye like pattern centered on the Clinton and H3 sites, with sharper gradients to the south than to the north. It indicates that the region with concentrations above the 15 $\mu g/m^3$ standard overlaps the industrial and heavily populated areas of Houston. Similar displays of spatially mapped daily $PM_{2.5}$ concentrations are presented in Appendix A. They show that a variety of patterns occur in the region, however, quite often the area around the Clinton site has the highest levels in the area.

The average chemical composition of $PM_{2.5}$ on 1997-1998 chemistry sampling days is shown in **Figures 3-7 and 3-8**. The concentrations of organic material have been estimated as 140 percent of the measured organic carbon concentrations, which is a conservative estimation procedure. Data from unusual events were also excluded from this profile. Specifically, the data from June 21-22, 1997 and July 3, 1997 were unrepresentative and were excluded because they have large amounts of crustal material (about 50 percent of the mass) that is probably due to long-range transport of dust from Africa (Price et al., 1998). It is also important to note that the chemistry sampling days tended to be the higher concentration days and, therefore, these averages may reflect a potentially biased picture of the average PM composition.

The chemical composition data indicate that sulfate and organic material are the most abundant components. On average, sulfate comprises 26 to 29 percent of $PM_{2.5}$ mass and organic material comprises 26 to 32 percent of $PM_{2.5}$ mass at the Houston monitors (i.e., excluding Galveston and Mauriceville). On average, nitrate accounts for 7 to 9 percent and ammonium accounts for 10 to 11 percent of the $PM_{2.5}$ at these monitors. Other constituents account for 17 to 22 percent of the mass, on average, at these stations. Based on the silica, aluminum, iron, calcium, and potassium elemental data and reasonable assumptions regarding their oxidation states, we estimate that only 3 to 6 percent of $PM_{2.5}$ mass is crustal in origin (dust), on average. The sea salt accounts for less than one percent of the $PM_{2.5}$ mass. It is quite possible that a significant portion of the "other" mass should actually be associated with the organic material. Turpin et al. (1997) suggest that the mass of many condensable organic compounds may be 2 to 3 times the mass of their carbon rather than 1.4 times. This factor has a large influence on estimates of relative composition. If one assumes organic material is 2.0 times the measured organic carbon concentrations, organic material comprises 33 to

46 percent of the $PM_{2.5}$ mass and other compounds comprise only 5 to 13 percent, on average, at the Houston monitors.

The ammonium data indicate that, on average, there is insufficient ammonium to fully neutralize the sulfate and nitrate (i.e., for all of sulfate to exist as ammonium sulfate and all of the nitrate to exist as ammonium nitrate). Small portions of the sulfate are likely to be associated with calcium, potassium, and sodium, and a small portion of the nitrate is associated with sodium. A sample-by-sample analysis indicates there are at least three types of cases:

1. Ammonia-rich cases were there is sufficient ammonium to fully buffer the sulfate and nitrate;
2. Ammonia-lean cases with little or no nitrate and only sufficient ammonia for sulfate to exist as a mixture of ammonium bisulfate and ammonium sulfate;
3. Warm cases with sufficient ammonium to fully buffer the sulfate but little or no nitrate probably because warm temperatures inhibit the formation of ammonium nitrate.

Without corresponding nitric acid and ammonia data, it is not possible to definitively determine the extent of ammonium-limitations of aerosol nitrate formation. These types of data are needed to accurately assess the effects of NO_x, SO_x, and NH_3 emission changes on $PM_{2.5}$. It is especially important to understand this issue because under ammonia-limited conditions, reductions in NO_x or SO_x emissions may not reduce ambient $PM_{2.5}$ mass. In the interim, we assumed conditions were predominantly ammonia-rich for our projection of $PM_{2.5}$ in this study.

3.4 PROJECTED $PM_{2.5}$ CONCENTRATIONS

For consistency, we wanted to use the same planning years for $PM_{2.5}$ as for ozone. Emissions information was available for 1993 and 2007, and ambient concentrations were available for 1997-1998. The number of air quality models available for projecting $PM_{2.5}$ forward or backward in time (from 1998) is quite limited. The Houston aerometric database (i.e., meteorology, emissions, and ambient concentration data) is insufficient to support detailed three-dimensional dispersion modeling of aerosol species at this time. In fact, there are no areas of the country with adequate databases to support annual dispersion of both primary and secondary $PM_{2.5}$. Detailed aerosol dispersion models have only been demonstrated for episodic conditions in a few areas (Lurmann et al., 1997; Kumar and Lurmann, 1997; Meng et al., 1998; Lu et al., 1997a,b; Kumar et al., 1996; Dabdub et al., 1997). The most sophisticated model that the Houston database would support is the speciated PM rollback model (Trijoinis et al., 1975; Kumar and Lurmann, 1996).

In the speciated PM rollback model, simple linear associations are made between the region-wide emissions of specific chemical species and the ambient PM concentrations of specific chemical components. The pairs of independent emissions and ambient chemical components that are associated with one another in the speciated PM rollback model are listed in **Table 3-10**. The model assumes the ambient concentration of each chemical component in excess of the background concentration changes in proportion to the precursor emissions for

that component. For example, ammonium sulfate concentrations in excess of the background are assumed to be proportional to SO_2 emissions. Ammonium nitrate concentrations in excess of the background are assumed to be proportional to NO_x emissions. Changes in ammonia emissions are ignored because they are not assumed to be independent of sulfate and nitrate under the ammonia-rich assumption. Secondary organic aerosol (SOA) concentrations in excess of the background are assumed to be proportional to gaseous VOC emissions. In our implementation, we have assumed the ambient organic PM is 80 percent primary and 20 percent secondary. We did not identify any studies of the organic PM species distributions for Houston or Texas and elected to use values within the range estimated from other studies (Turpin and Huntzicker, 1995; Stradler et al., 1999). Primary emissions of elemental carbon, organic material, crustal material, and other $PM_{2.5}$ are assumed to be proportional to their primary emissions. The crustal material concentrations are estimated from the elemental components and assumptions regarding the most probable oxidation states of aluminum, silica, potassium, calcium, and iron, as shown in Equation 3-3.

$$[\text{Crustal PM}] = 1.89[\text{Al}] + 1.57[\text{Si}] + 1.28[\text{K}] + 1.4[\text{Ca}] + 1.43[\text{Fe}] \qquad \textbf{(3-3)}$$

Recall that chemical composition information is not available for all of the sampling days. We elected to apply the speciated rollback model to the average conditions at each monitoring station on the non-transport chemistry sampling days and use the seven-station average relative average change in $PM_{2.5}$ mass as the characteristic parameter for projecting $PM_{2.5}$ concentrations to 1993 and 2007. First, the average composition of $PM_{2.5}$ (by site) was expressed in terms of the modeled species as shown in **Table 3-11**. The average speciated background concentration for the region was calculated using the composition profile from Galveston and the 11 $\mu g/m^3$ background $PM_{2.5}$. The background concentration was assumed to be time invariant (i.e., applies to 1993-2007) because we have no data for earlier periods and there is no assurance that upwind areas will implement sufficient emission controls to reduce the future $PM_{2.5}$ levels coming into Houston. The speciated emissions data for the eight-county domain described in Section 2 were assumed to change at a constant rate between 1993 and 2007. The ratios of emissions in 1998 to 1993 and 1998 to 2007 for various scenarios are shown in **Table 3-12**.

The average concentrations in other years (1993 or 2007) were calculated from the following equations where PM is the $PM_{2.5}$ mass in a particular grid on a specific day (or annual average), C is the concentration of a specific chemical component (j) at a specific monitoring site (i), and F is the seven-station average ratio of PM mass in excess of the background in the other year to that in the base year. As indicated above, the F factors were calculated for average conditions on the non-transport chemistry sampling days, and may have some undesirable sampling bias.

$$C^{i,j}_{other\ yr} = C^{j}_{backgrd} + \frac{E^{j}_{other\ yr}}{E^{j}_{1998}} \left(C^{i,j}_{1998} - C_{backgrd} \right) \quad \textbf{(3-4)}$$

$$PM^{x,y,t}_{other\ yr} = PM_{backgrd} + F_{other\ yr} \left(PM^{x,y,t}_{1998} - PM_{backgrd} \right) \quad \textbf{(3-5)}$$

$$F_{other\ yr} = \frac{1}{M} \sum_{j}^{M} \sum_{i}^{Species} \left[\frac{C^{i,j}_{other\ yr} - C^{j}_{backgrd}}{C^{i,j}_{1998} - C^{j}_{backgrd}} \right] \quad \textbf{(3-6)}$$

Gridded fields of daily and annual average $PM_{2.5}$ under the various scenarios were generated using these procedures.

The maximum estimated annual average $PM_{2.5}$ mass concentrations for 1993 and 2007 under alternate emission scenarios are shown in **Figure 3-9** and **Table 3-13**. These are based on the spatially mapped concentrations and show a slightly lower maximum value for 1997-1998 than was observed at the Clinton monitoring site (18.3 versus 18.6 $\mu g/m^3$ observed). Only one projection (Scenario 1) is shown for 1993 because they were all very similar. Notice that with the relatively high background concentrations in the region, the maximum concentrations estimated for other years are quite similar to the 1997-1998 estimation. The maximum annual concentrations for Scenarios 1, 2, 3, and 4 in 2007 are 19, 18.8, 18.9, and 18.6 $\mu g/m^3$, respectively. Assuming full implementation of the 85 percent NO_x and 15 percent VOC emission reductions associated with the ozone SIP, the estimated maximum annual $PM_{2.5}$ is 18.6 $\mu g/m^3$. All of the future-year scenarios fall significantly short of the 15 $\mu g/m^3$ annual NAAQS. Hence, we also created daily and annual fields using the factor needed to bring the maximum concentration into compliance with the annual average standard [F_{NAAQS} = (15-11)/(18.3-11)].

It should be noted that the high background $PM_{2.5}$ levels assumed for these future year calculations greatly diminish the estimated effectiveness of the these emission control measures. The current background levels are 60 percent of maximum annual average $PM_{2.5}$ and 70 to 75 percent of the $PM_{2.5}$ NAAQS. Clearly, the background $PM_{2.5}$ is the greatest contributor to the PM problem. Without some reductions in the regional background $PM_{2.5}$, large reductions in PM-related emissions will be needed to achieve the NAAQS in Houston. Alternately, if the background concentration is significantly reduced by implementation of regional emission control measures across the eastern United State (or perhaps in smaller areas upwind of Houston), then control measures implemented in Houston would be more effective in bringing the area into compliance with the standard. Thus, it is critically important to understand the factors that control both the local-scale and regional-scale components of $PM_{2.5}$ in the region.

3.5 AIR TOXICS CONCENTRATIONS

There is concern for population exposure to noncriteria hazardous air pollutants (HAPs) in the Houston area. The 1990 Clean Air Act (CAA) lists 118 hazardous air pollutants in addition to the criteria pollutants. The HAPs include gaseous VOCs, semi-volatile compounds, and specific constituents of PM. The concentration of the petroleum and chemical industries in Houston along with the usual transportation, industrial, commercial, and residential sources suggest Houston residents could be exposed to higher concentrations of toxic compounds than residents of other areas.

The characterization of baseline air quality conditions for toxic compounds was determined from ambient VOC data collected between 1992 and 1997 at 13 monitors in the Houston area. **Figure 3-10** shows a map of the VOC monitoring locations during this period. Long-term exposure to toxic compounds is the primary health concern, so only the annual average concentrations are reported here. **Tables 3-14 through 3-17** list the annual average concentrations of 12 HAPs of particular concern, including benzene, 1,3-butadiene, acetaldehyde, formaldehyde, carbon-tetrachloride, chloroform, ethylbenzene, n-hexane, styrene, toluene, xylenes, and trichloroethylene. These data are a combination of government agency monitoring data reported in AIRS and the Houston Regional Monitoring Corporation's (HRM) monitoring data. They were not necessarily collected with identical sampling procedures, however, we believe they are comparable for the purpose of assessing annual average concentrations. Of the HAPs measured in Houston, exposure to benzene, 1,3-butadiene and formaldehyde probably have larger health risks than exposure to other compounds. These are also compounds that may be reduced in VOC control programs implemented to achieve compliance with the ozone NAAQS. Overall, despite the high concentration of industries that may emit toxic compounds, the annual average ambient concentrations do not show unusually high concentrations. Most of the measured VOCs are comparable to those measured in other urban areas (such as Los Angeles). Concentrations at these urban levels may be associated with elevated cancer risks. Despite our concern for these potential effects and interests in quantifying potential impacts, no attempt was made to spatially map the toxic compound concentrations because the toxics monitoring network does not provide sufficient spatial coverage to assign exposures to the whole population in Houston.

As part of our review of potentially toxic compounds, the elemental concentration data collected in the 1997-1998 ambient $PM_{2.5}$ measurement program were examined. The average concentrations on chemistry sampling days are shown in **Table 3-18**. The concentrations of $PM_{2.5}$ lead, arsenic, vanadium, and chromium are less than 0.005 $\mu g/m^3$, which is very low. This size cut may exclude some of the potentially toxic components that are contained in coarse particles. Nevertheless, the approach we used for assessing the health effects of PM exposures is not chemical component specific. The reason for this is that none of the known chemical components of PM are present in sufficient quantities in ambient air to explain the observed associations of PM mass with mortality and morbidity. These elemental data basically confirm that none of the measured PM components are unusually high in the Houston area.

3.6 AIR QUALITY SUMMARY AND RECOMMENDATIONS

Air quality data for the Houston area have been used to establish baseline ambient conditions and simple models have been used in conjunction with baseline data to project future ambient conditions for use in subsequent assessment of human exposure. The principal issues for ozone are the spatial representativeness of the monitoring data for the baseline period and the adequacy of the methods used to estimate future conditions on all of the days in the ozone season. Ambient ozone conditions and the ozone response to emission changes are complex, yet are probably much better understood than those for $PM_{2.5}$. The ambient conditions and source attribution of $PM_{2.5}$ are not well characterized. While good use has been made of the 1997-1998 special $PM_{2.5}$ study data, there are significant limitations regarding the duration of sampling, extent of chemical characterization, spatial representativeness, background levels, and fidelity of the projection methods. Clearly, more speciated $PM_{2.5}$ data are needed to characterize conditions and determine the sources responsible for high $PM_{2.5}$ levels in the region. Refinement of the source attribution of PM is important not only for selection of effective $PM_{2.5}$ control programs, but also for understanding the contributions of common precursor emissions to ozone and $PM_{2.5}$. In particular, further work is need to characterize the contribution of NO_x, anthropogenic VOCs, and biogenic VOCs to ambient $PM_{2.5}$ as well as ozone, in order to design emission control programs to comply with the ozone and $PM_{2.5}$ NAAQS.

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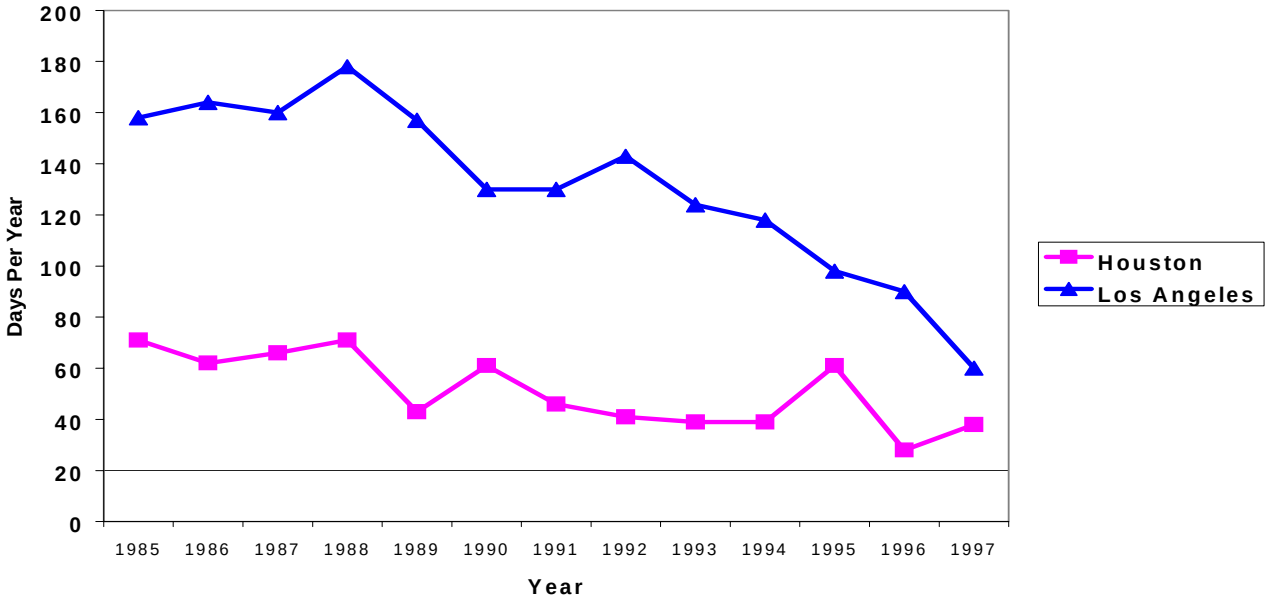

Figure 3-1. Number of days with ozone concentrations greater than 120 ppb at one or more monitoring stations in the Houston and Los Angeles nonattainment areas between 1985 and 1997.

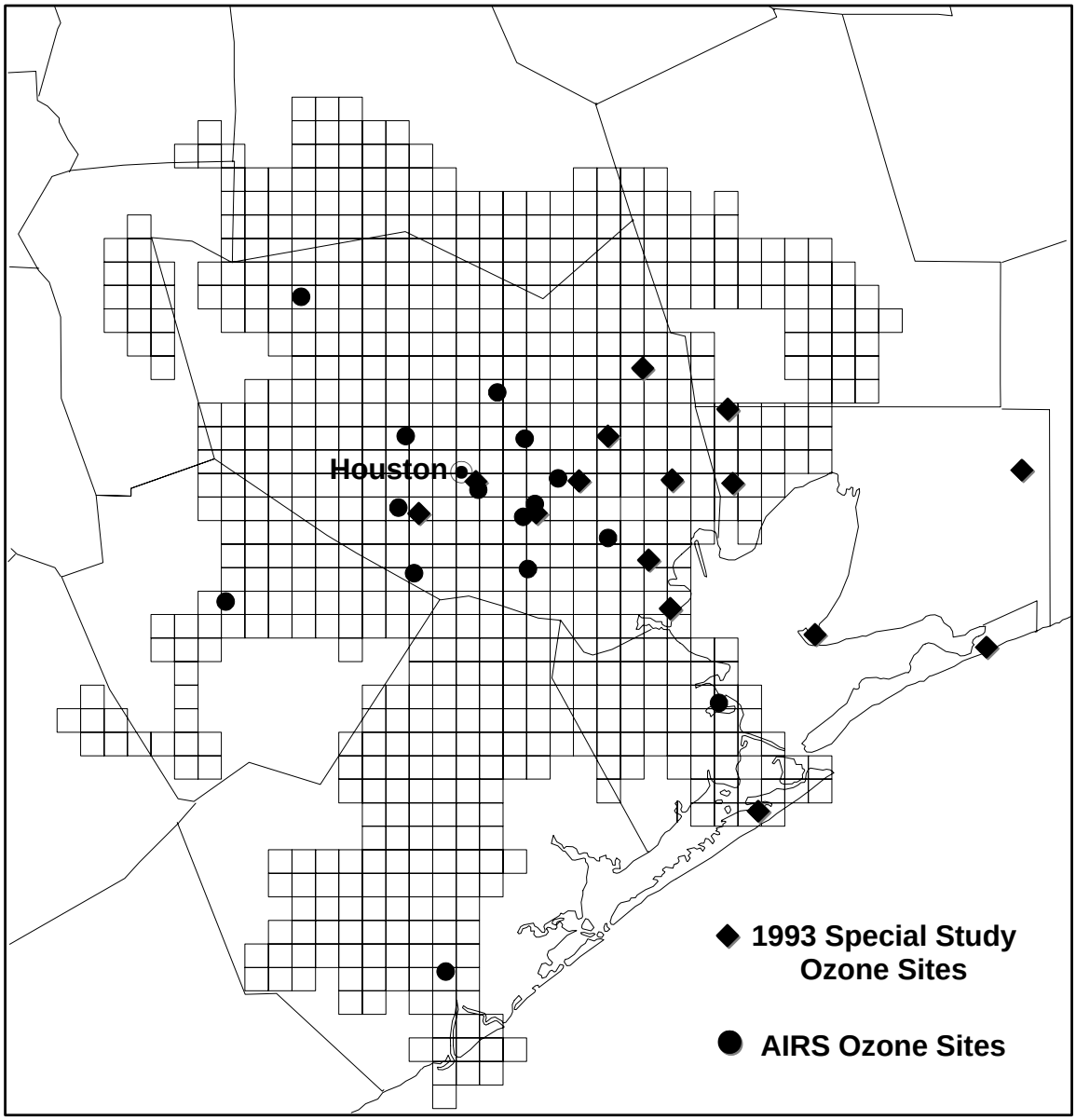

Figure 3-2. Locations of 1995 special study and AIRS ozone monitoring sites in Houston.

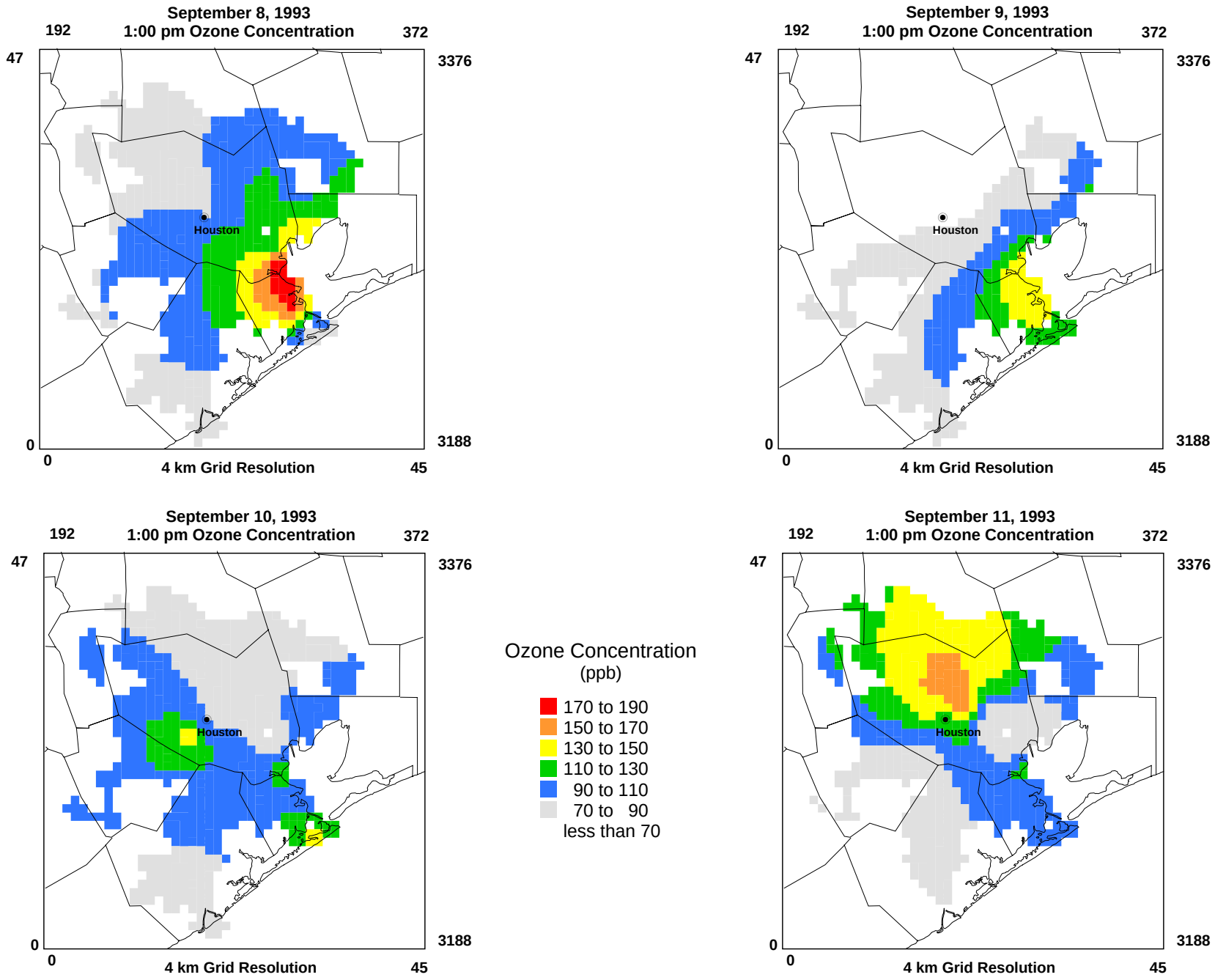

Figure 3-3. Ozone concentrations at 1:00 pm on September 8 through 11, 1993.

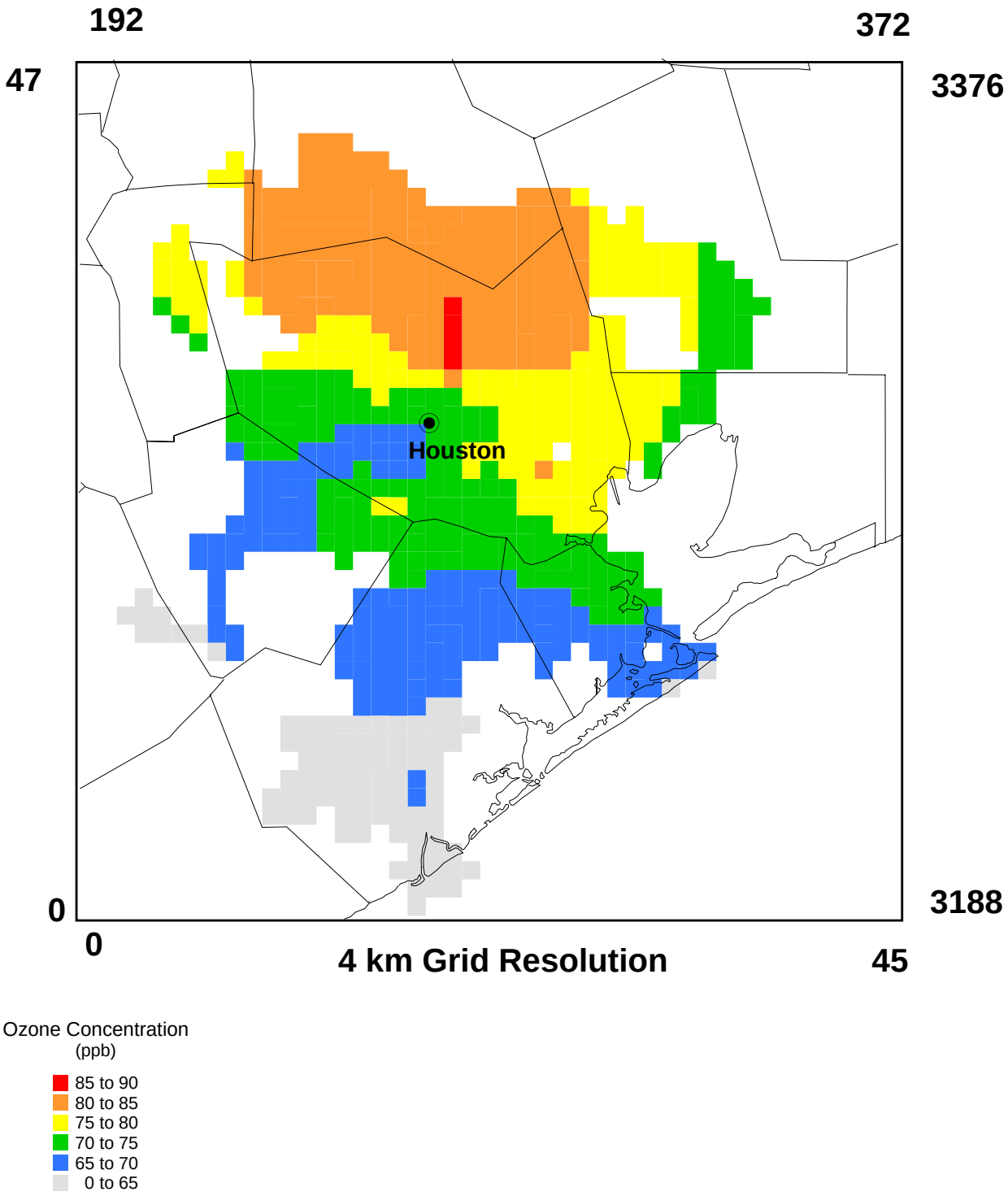

Figure 3-4. Average daily 1-hr maximum ozone for the 1993 to 1995 days included in the exposure modeling database.

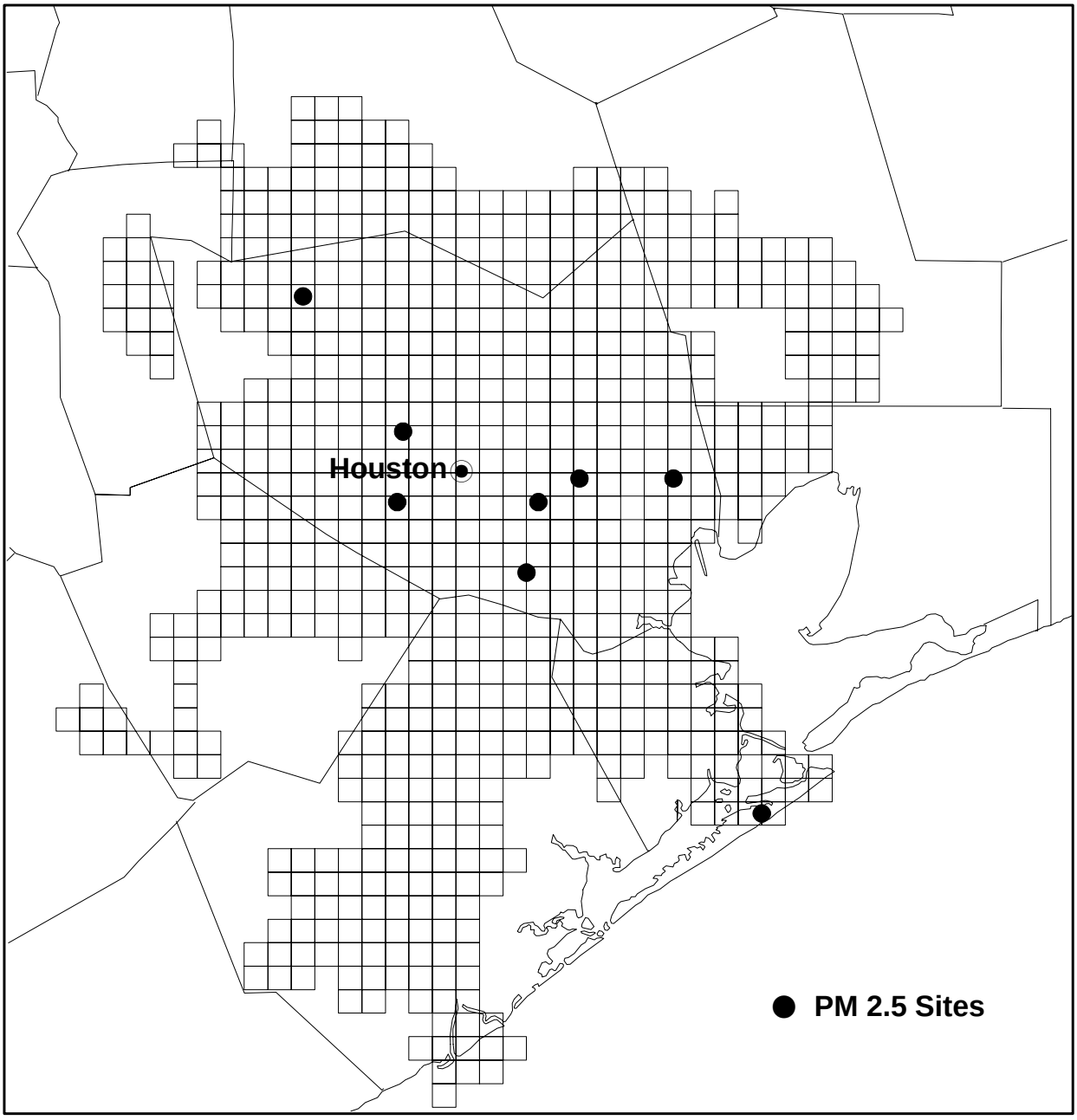

Figure 3-5. Locations of $PM_{2.5}$ monitoring sites in Houston.

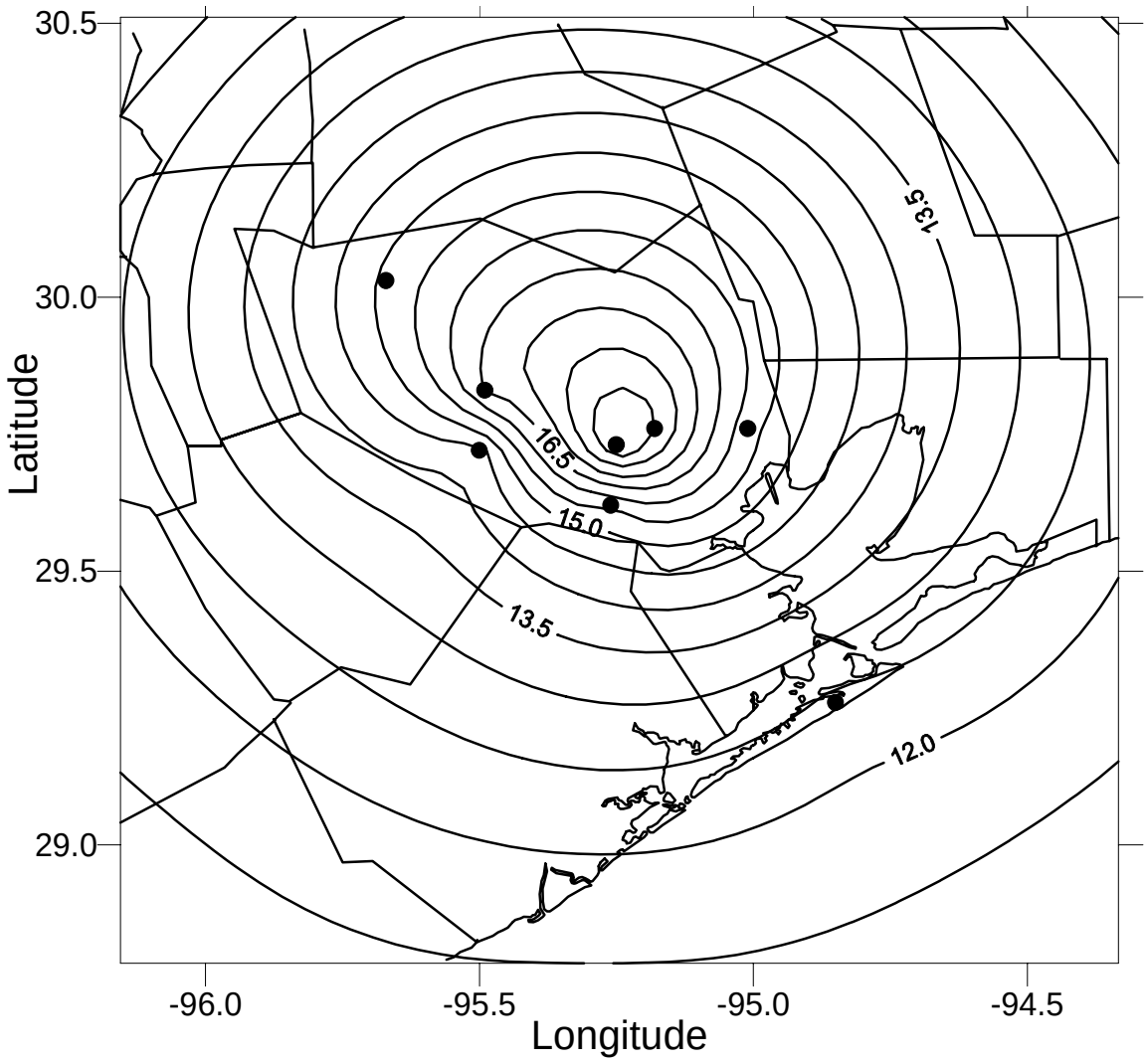

Figure 3-6. Spatial distribution of 1997-1998 annual average $PM_{2.5}$ mass concentrations in Houston. Site abbreviations are shown in Table 3-9.

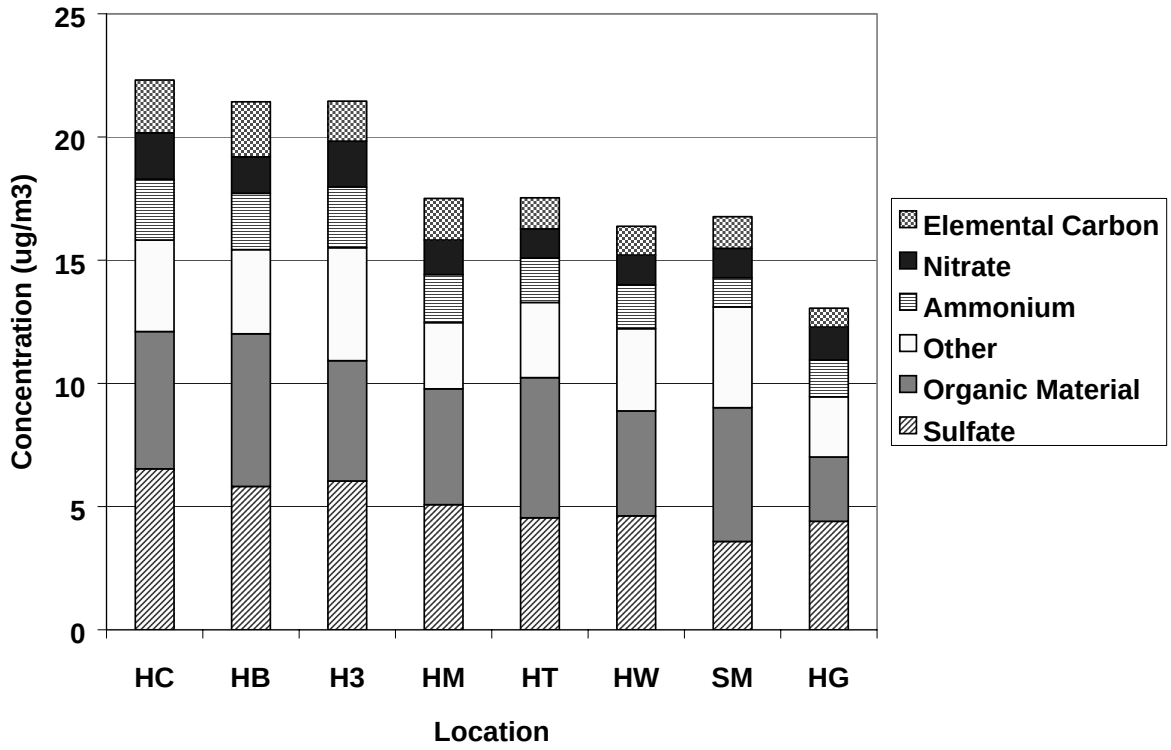

Figure 3-7. Average $PM_{2.5}$ chemical component concentrations at Houston area monitors on non-transport chemistry sampling days in 1997-1998. Site abbreviations are shown in Table 3-9.

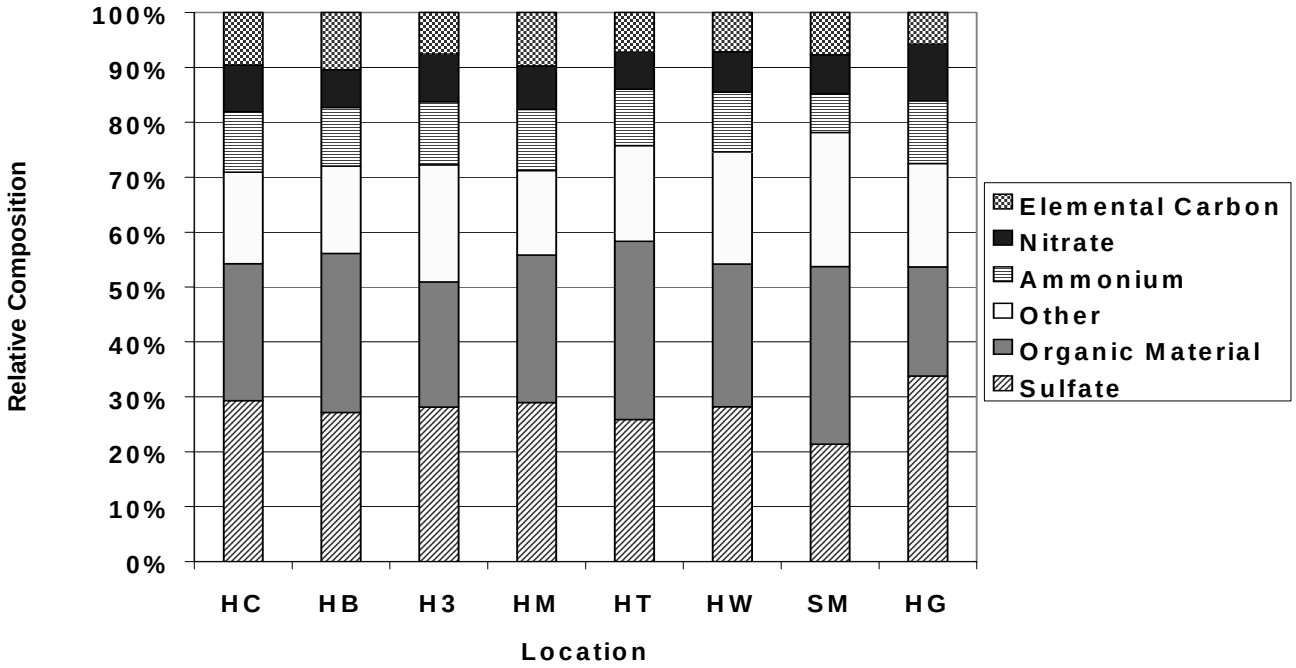

Figure 3-8. Average relative amounts of sulfate, nitrate, ammonium, organic material (1.4 times organic carbon), elemental carbon, and other material at Houston area monitors on non-transport chemistry sampling days in 1997-1998. Site abbreviations are shown in Table 3-9.

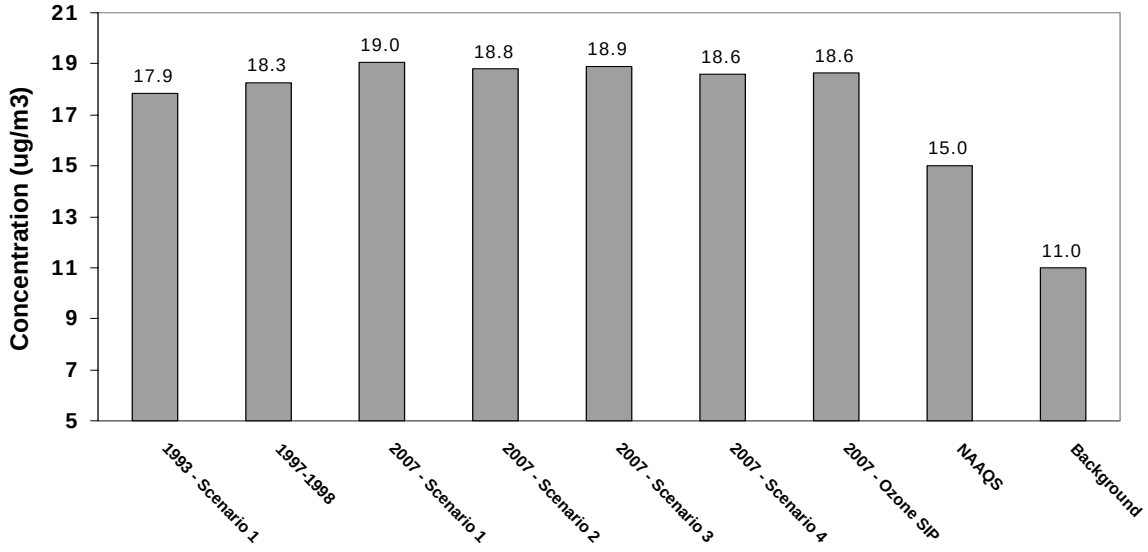

Figure 3-9. Estimated maximum annual average $PM_{2.5}$ in 1993, 1997-1998, and for 2007 under alternate emission scenarios.

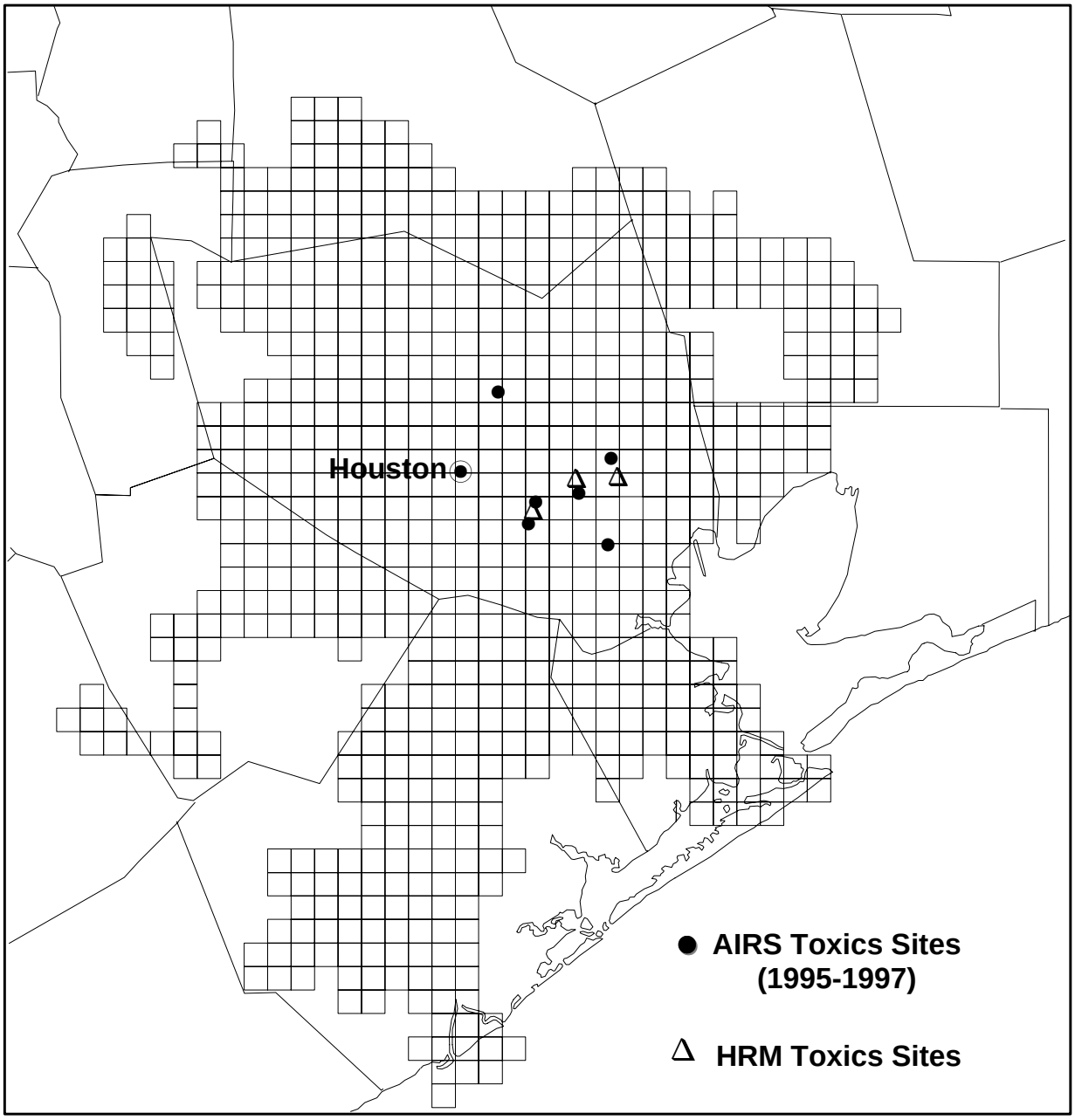

Figure 3-10. Locations of AIRS and Houston Regional Monitoring Corporations (HRM) air toxics monitoring sites in Houston.

Table 3-1. Number of days with ozone NAAQS exceedances in Houston.

Year	Number of Days With Exceedances	
	120 ppb 1-hr NAAQS	80 ppb 8-hr NAAQS
1992	40	17
1993	31	15
1994	36	15
1995	59	45

Table 3-2. September 8-11, 1993, maximum ozone concentrations observed and simulated in the UAM-V SIP simulations and the CAMx simulations with improved biogenic emission estimates.

Day	Observed Maximum 1-hr Concentration (ppb) for 1993	Estimated Maximum 1-hr Concentration (ppb)	
		UAM-V With SIP Emissions	CAMx With New Biogenic Emissions
September 8	214	186	181
September 9	195	179	178
September 10	162	179	176
September 11	189	185	185

Table 3-3. Maximum simulated ozone concentrations for the 2007 baseline conditions in the UAM-V SIP simulations and the CAMx simulations with new mobile source and biogenic emission estimates. Maximum observed 1-hr ozone concentrations for 1993 are shown for comparison.

Day	Maximum Observed 1-hr Ozone in 1993 (ppb)	Estimated Maximum 1-hr Ozone Concentration (ppb) for 2007 Baseline	
		UAM-V With SIP Emissions	CAMx With New Emissions
September 8	214	191	179
September 9	195	172	169
September 10	162	177	170
September 11	189	182	176

Table 3-4. CAMx estimated maximum 1-hr ozone for various 2007 emissions scenarios.

Day	Maximum 1-hr Ozone (ppb) for Various 2007 Scenarios						
	Baseline	25% NO_x Reduction[1]	45% NO_x Reduction[1]	65% NO_x Reduction[1]	75% NO_x Reduction[1]	85% NO_x Reduction[1]	Clean Diesel Fuel[1]
September 8	179	176	170	152	136	123	179
September 9	169	169	163	145	130	110	167
September 10	170	166	156	138	127	110	168
September 11	176	169	158	139	124	103	169

[1] Plus a 15 percent reduction in anthropogenic VOC emissions in all 2007 scenarios.

Table 3-5. Estimated percent reduction from the 2007 baseline results for maximum 1-hr ozone for various 2007 emissions scenarios.

Day	Percentage Reduction in 2007 Maximum 1-hr Ozone Compared to 2007 Baseline					
	25% NO_x Reduction[1]	45% NO_x Reduction[1]	65% NO_x Reduction[1]	75% NO_x Reduction[1]	85% NO_x Reduction[1]	Clean Diesel Fuel[1]
September 8	2%	5%	15%	24%	31%	0%
September 9	0%	4%	14%	23%	35%	1%
September 10	2%	8%	19%	25%	35%	1%
September 11	4%	10%	21%	30%	41%	4%

[1] Plus a 15 percent reduction in anthropogenic VOC emissions in all 2007 scenarios.

Table 3-6. Estimated percent reduction from the 1993 results for maximum 1-hr ozone for various 2007 emissions scenarios.

Day	Percentage Reduction in 2007 Maximum 1-hr Ozone Compared to 1993						
	Baseline	25% NO_x Reduction[1]	45% NO_x Reduction[1]	65% NO_x Reduction[1]	75% NO_x Reduction[1]	85% NO_x Reduction[1]	Clean Diesel Fuel[1]
September 8	1%	3%	6%	16%	25%	32%	1%
September 9	5%	5%	8%	19%	27%	38%	6%
September 10	3%	6%	11%	22%	28%	38%	5%
September 11	5%	9%	15%	25%	33%	44%	9%

[1] Plus a 15 percent reduction in anthropogenic VOC emissions in all 2007 scenarios.

Table 3-7. Ratio of CAMx estimated hourly maximum ozone in 2007 simulations to 1993 simulations for the September 8-11 episode with revised biogenic emissions and revised 2007 mobile source emissions.

Hour	2007 Scenario						
	Baseline	25% NO_x Reduction[1]	45% NO_x Reduction[1]	65% NO_x Reduction[1]	75% NO_x Reduction[1]	85% NO_x Reduction[1]	Clean Diesel Fuel[1]
0	0.97	0.94	0.91	0.86	0.83	0.78	0.90
1	0.97	0.94	0.91	0.86	0.83	0.78	0.90
2	0.97	0.94	0.91	0.87	0.83	0.79	0.90
3	0.97	0.94	0.91	0.87	0.84	0.81	0.90
4	0.97	0.95	0.93	0.88	0.86	0.83	0.91
5	0.98	0.96	0.94	0.90	0.88	0.85	0.93
6	0.98	0.95	0.93	0.90	0.88	0.85	0.93
7	0.98	0.96	0.93	0.90	0.88	0.85	0.93
8	0.98	0.96	0.94	0.91	0.88	0.85	0.94
9	0.98	0.96	0.93	0.90	0.87	0.84	0.95
10	0.97	0.95	0.93	0.90	0.87	0.83	0.95
11	0.96	0.95	0.93	0.89	0.85	0.77	0.94
12	0.97	0.94	0.93	0.89	0.82	0.71	0.93
13	0.96	0.96	0.93	0.87	0.79	0.67	0.93
14	0.96	0.95	0.91	0.83	0.75	0.64	0.94
15	0.96	0.95	0.91	0.81	0.73	0.61	0.95
16	0.96	0.94	0.90	0.80	0.71	0.60	0.95
17	0.95	0.93	0.88	0.79	0.71	0.59	0.93
18	0.95	0.91	0.86	0.78	0.70	0.60	0.88
19	0.95	0.91	0.86	0.78	0.71	0.62	0.86
20	0.95	0.91	0.87	0.78	0.72	0.63	0.85
21	0.96	0.93	0.88	0.81	0.74	0.66	0.86
22	0.97	0.93	0.88	0.80	0.75	0.67	0.84
23	0.96	0.93	0.88	0.80	0.75	0.67	0.85
Min	0.95	0.91	0.86	0.78	0.70	0.59	0.84
Mean	0.96	0.94	0.91	0.84	0.79	0.72	0.91
Max	0.98	0.96	0.94	0.91	0.88	0.85	0.95

[1] Plus a 15 percent reduction in anthropogenic VOC emissions in all 2007 scenarios.

Table 3-8. Ratio of CAMx estimated hourly maximum ozone in excess of assumed 50 ppb background in 2007 simulations to 1993 simulations for the September 8-11 episode with revised biogenic emissions and revised 2007 mobile source emissions.

Hour	2007 Scenario						
	Baseline	25% NO_x Reduction[1]	45% NO_x Reduction[1]	65% NO_x Reduction[1]	75% NO_x Reduction[1]	85% NO_x Reduction[1]	Clean Diesel Fuel[1]
0	0.95	0.90	0.84	0.75	0.68	0.59	0.82
1	0.94	0.89	0.83	0.74	0.67	0.59	0.82
2	0.94	0.89	0.82	0.74	0.67	0.59	0.81
3	0.94	0.88	0.82	0.73	0.68	0.60	0.80
4	0.94	0.89	0.84	0.75	0.70	0.63	0.81
5	0.96	0.90	0.86	0.78	0.72	0.66	0.84
6	0.95	0.89	0.85	0.77	0.72	0.66	0.82
7	0.95	0.90	0.85	0.77	0.72	0.65	0.83
8	0.96	0.90	0.86	0.80	0.73	0.66	0.87
9	0.95	0.91	0.87	0.80	0.75	0.70	0.90
10	0.95	0.92	0.88	0.83	0.78	0.71	0.91
11	0.94	0.92	0.89	0.82	0.76	0.63	0.91
12	0.95	0.91	0.89	0.83	0.73	0.56	0.90
13	0.94	0.93	0.90	0.81	0.70	0.52	0.90
14	0.94	0.93	0.88	0.76	0.65	0.48	0.92
15	0.95	0.93	0.87	0.73	0.62	0.45	0.93
16	0.95	0.92	0.86	0.72	0.60	0.44	0.93
17	0.94	0.90	0.84	0.70	0.59	0.43	0.90
18	0.92	0.88	0.80	0.68	0.57	0.42	0.82
19	0.92	0.86	0.80	0.66	0.56	0.43	0.78
20	0.92	0.86	0.79	0.66	0.56	0.42	0.76
21	0.94	0.88	0.80	0.67	0.57	0.44	0.77
22	0.95	0.88	0.79	0.64	0.54	0.41	0.71
23	0.93	0.86	0.77	0.61	0.51	0.38	0.72
Min	0.92	0.86	0.77	0.61	0.51	0.38	0.71
Mean	0.94	0.90	0.84	0.73	0.65	0.54	0.84
Max	0.96	0.93	0.90	0.83	0.78	0.71	0.93

[1] Plus a 15 percent reduction in anthropogenic VOC emissions in all 2007 scenarios.

Table 3-9. Annual average $PM_{2.5}$ mass concentrations in 1997-1998 in the Houston area.

$PM_{2.5}$ Monitoring Station	Site Abbreviation	Annual Average $PM_{2.5}$ Mass ($\mu g/m^3$)	Maximum 24-hr Average $PM_{2.5}$ Mass ($\mu g/m^3$)
Clinton	HC	18.6	39.2
HRM03	H3	18.1	41.6
Bingle	HB	16.6	38.9
HRM07	H7	15.7	38.5
Swiss & Monroe	HM	15.5	34.6
NW Harris Co. – Tomball	HT	15.2	35.1
Shell Westhollow	HW	14.3	35.9
Galveston	HG	12.4	27.4
Mauriceville	SM	13.9	24.7

Table 3-10. Associations of emitted and ambient $PM_{2.5}$ chemical components.

Emission Species	Ambient $PM_{2.5}$ Components
SO_2 (gaseous)	Ammonium Sulfate
NO_x (gaseous)	Ammonium Nitrate
VOC (gaseous)	Secondary Organic Material
Primary Organic Material	Primary Organic Material
Primary Elemental Carbon	Elemental Carbon
Primary Crustal PM	Crustal PM
Primary Other PM	Other PM

Table 3-11. Average $PM_{2.5}$ composition at Houston area monitoring sites in 1997-1998 expressed in terms of the rollback species.

Location	$(NH_4)_2SO_4$	NH_4NO_3	Primary OM	Secondary OM	EC	Crustal	Other	Mass
HC	8.98	2.44	4.46	1.11	2.13	1.28	1.91	22.31
HB	8.00	1.91	4.96	1.24	2.23	0.72	2.36	21.42
H3	8.30	2.41	3.91	0.98	1.62	1.22	3.01	21.45
H7	7.90	1.98	3.83	0.96	1.34	0.77	3.19	19.98
HM	6.98	1.80	3.77	0.94	1.70	0.59	1.74	17.51
HT	6.24	1.53	4.56	1.14	1.27	0.48	2.33	17.54
HW	6.35	1.55	3.41	0.85	1.17	0.54	2.51	16.38
SM[1]	4.93	1.54	4.34	1.08	1.28	1.04	2.54	16.76
HG[1]	6.06	1.73	2.08	0.52	0.75	0.47	1.43	13.04
Background	5.11	1.46	1.75	0.44	0.64	0.40	1.21	11.00

[1] Not included in the seven-station average factors used to project PM.

Table 3-12. Relative changes in $PM_{2.5}$ and PM-related emission between 1993, 1998, and 2007.

Parameter	Scenario 1[a]	Scenario 2[a]	Scenario 3[a]	Scenario 4[a]	Ozone SIP[a]
1993/1998 SO_x Emissions Ratio	0.958	0.967	0.958		
2007/1998 SO_x Emissions Ratio	1.079	1.063	1.079	0.99	1.079
1993/1998 NO_x Emissions Ratio	0.971	0.971	0.971		
2007/1998 NOx Emissions Ratio	1.055	1.055	1.055	1.085	0.158
1993/1998 VOC Emissions Ratio	0.957	0.957	0.957		
2007/1998 VOC Emissions Ratio	1.082	1.082	1.082	1.156	0.920
1993/1998 Primary OM Emissions Ratio	0.936	0.949	0.938		
2007/1998 Primary OM Emissions Ratio	1.126	1.098	1.123	1.057	1.126
1993/1998 EC Emissions Ratio	0.948	0.957	0.952		
2007/1998 EC Emissions Ratio	1.102	1.082	1.092	0.985	1.102
1993/1998 Crustal Emissions Ratio	0.964	0.972	0.991		
2007/1998 Crustal Emissions Ratio	1.068	1.053	1.015	1.064	1.068
1993/1998 Other PM Emissions Ratio	0.913	0.966	0.995		
2007/1998 Other PM Emissions Ratio	1.178	1.065	1.086	1.074	1.178

[a] See Section 2.5 for a description of the alternate future-year emission scenarios.

Table 3-13. Estimated maximum $PM_{2.5}$ concentrations in 1993 and 2007.

Case	Projection Factor[1]	Maximum Annual Average $PM_{2.5}$ ($\mu g/m^3$)	Maximum 24-hr Average $PM_{2.5}$ ($\mu g/m^3$)
1993 Scenario 1	0.9445	17.85	39.45
1997-1998 Base	1.0000	18.25	41.12
2007 Scenario 1	1.1089	19.04	44.40
2007 Scenario 2	1.0762	18.80	43.42
2007 Scenario 3	1.0906	18.91	43.85
2007 Scenario 4	1.0441	18.57	42.45
2007 Ozone SIP	1.0515	18.62	42.67
Rollback to NAAQS[2]	0.5517	15.00	27.62

[1] The scale factor applied to the concentrations in excess of the background concentration.

[2] Assumes all concentrations above the background are reduced by the amount needed to reduce the annual average concentrations at the single highest monitoring location to the level of the annual NAAQS.

Table 3-14. Annual average concentrations (ppb) of 1,3-butadiene, acetaldehyde, formaldehyde, and benzene in the Houston area for 1992-1997.

Site	Parameter	1992	1993	1994	1995	1996	1997
HRM01	1,3-Butadiene	2.87	2.66	2.89	1.47	1.46	1.46
HRM03	1,3-Butadiene	0.68	0.59	0.80	0.92	0.45	0.56
HRM04	1,3-Butadiene	1.76	0.89	1.21	1.56	0.76	0.62
HRM07	1,3-Butadiene	1.67	3.46	4.06	3.25	3.21	**6.47***
HRM08	1,3-Butadiene	0.48	0.39	0.51	0.58	0.22	0.28
HRM10	1,3-Butadiene	0.40	0.27		0.40	0.16	0.25
HRM11	1,3-Butadiene	0.15	0.29	0.42	0.28	0.12	0.26
Clinton	1,3-butadiene	na	na	na	0.66	0.69	0.85
Aldino	1,3-butadiene	na	na	na	0.16	0.10	0.17
Haden	1,3-butadiene	na	na	na	0.94	0.39	0.68
Galveston	1,3-butadiene	na	na	na	1.02	0.84	0.84
Sheldon	1,3-butadiene	na	na	na	0.38	0.33	0.67
Durant St	1,3-butadiene	na	na	na	na	0.05	0.31
HRM01	Acetaldehyde	na	na	na	na	5.90	**10.02***
HRM03	Acetaldehyde	na	na	na	na	6.57	6.04
HRM04	Acetaldehyde	na	na	na	na	5.17	5.83
HRM07	Acetaldehyde	na	na	na	na	5.55	7.46
HRM08	Acetaldehyde	na	na	na	na	5.40	7.99
HRM10	Acetaldehyde	na	na	na	na	4.72	6.39
HRM11	Acetaldehyde	na	na	na	na	5.11	5.47
Clinton	Acetaldehyde	na	na	na	na	1.55	2.17
Clinton	Formaldehyde	na	na	na	na	4.00	**4.67***
HRM01	Benzene	2.26	1.92	1.77	1.69	1.34	1.51
HRM03	Benzene	**3.44***	2.48	2.85	2.52	2.93	1.00
HRM04	Benzene	2.40	1.98	1.34	1.13	1.04	1.32
HRM07	Benzene	1.82	1.60	1.40	1.15	1.00	0.78
HRM08	Benzene	2.97	2.26	1.78	1.83	2.00	1.00
HRM10	Benzene	0.65	0.68	0.55	0.49	0.41	0.42
HRM11	Benzene	0.70	0.73	0.61	0.43	0.34	0.40
Clinton	Benzene	na	na	na	1.49	1.12	1.01
Aldino	Benzene	na	na	na	0.81	0.69	0.66
Haden	Benzene	na	na	na	2.70	1.18	1.42
Galveston	Benzene	na	na	na	1.03	0.73	1.21
Sheldon	Benzene	na	na	na	1.04	1.13	0.97
Durant St	Benzene	na	na	na		0.77	0.69

* Maximum annual average concentration.

Table 3-15. Annual average concentrations (ppb) of carbon-tetrachloride, chloroform, and ethylbenzene in the Houston area for 1992-1997.

Site	Parameter	1992	1993	1994	1995	1996	1997
HRM01	Carbon tetrachloride	0.10	0.12	0.15	0.09	0.12	0.17
HRM03	Carbon tetrachloride	0.40	0.42	0.48	0.46	0.27	0.31
HRM04	Carbon tetrachloride	0.12	0.11	0.14	0.09	0.12	0.12
HRM07	Carbon tetrachloride	0.11	0.10	0.10	0.09	0.11	0.10
HRM08	Carbon tetrachloride	0.10	0.10	0.11	0.08	0.11	0.10
HRM10	Carbon tetrachloride	0.08	0.09	0.12	0.07	0.11	0.10
HRM11	Carbon tetrachloride	0.17	0.14	0.14	0.08	0.13	0.10
Clinton	Carbon tetrachloride	na	na	na	0.04	**0.73***	0.13
Aldino	Carbon tetrachloride	Na	na	na	0.02	0.10	0.14
Haden	Carbon tetrachloride	Na	na	na	0.48	0.38	0.43
Galveston	Carbon tetrachloride	Na	na	na	0.08	0.12	0.16
Sheldon	Carbon tetrachloride	Na	na	na	0.12	0.11	0.14
Durant St	Carbon tetrachloride	Na	na	na	na	0.09	0.13
HRM01	Chloroform	0.12	0.74	0.06	0.06	0.06	**0.16***
HRM03	Chloroform	0.10	0.57	0.08	0.08	0.07	0.08
HRM04	Chloroform	0.04	0.06	0.06	0.08	0.09	0.10
HRM07	Chloroform	0.05	0.07	0.04	0.07	0.04	0.04
HRM08	Chloroform	0.04	0.04	0.04	0.06	0.07	0.06
HRM10	Chloroform	0.05	3.05	na	0.17	0.03	0.04
HRM11	Chloroform	0.03	0.03	0.03	0.05	0.04	0.04
Clinton	Chloroform	Na	na	na	0.06	0.03	0.04
Aldino	Chloroform	Na	na	na	0.02	0.01	0.05
Haden	Chloroform	Na	na	na	0.07	0.05	0.10
Galveston	Chloroform	Na	na	na	0.04	0.03	0.04
Sheldon	Chloroform	Na	na	na	0.03	0.03	0.06
Durant St	Chloroform	Na	na	na	na	0.01	0.03
HRM01	Ethylbenzene	**0.91***	0.50	0.52	0.45	0.31	0.38
HRM03	Ethylbenzene	0.71	0.67	0.78	0.50	0.35	0.29
HRM04	Ethylbenzene	0.47	0.45	0.34	0.29	0.15	0.44
HRM07	Ethylbenzene	0.42	0.27	0.29	0.26	0.18	0.21
HRM08	Ethylbenzene	0.30	0.32	0.27	0.30	0.17	0.20
HRM10	Ethylbenzene	0.22	0.56	0.19	0.45	0.04	0.09
HRM11	Ethylbenzene	0.25	0.18	0.33	0.19	0.05	0.09
Clinton	Ethylbenzene	na	na	na	0.39	0.27	0.20
Aldino	Ethylbenzene	na	na	na	0.04	0.12	0.16
Haden	Ethylbenzene	na	na	na	0.49	0.25	0.21
Galveston	Ethylbenzene	na	na	na	0.27	0.12	0.15
Sheldon	Ethylbenzene	na	na	na	0.56	0.20	0.56
Durant St	Ethylbenzene	na	na	na	na	0.08	0.10

* Maximum annual average concentration.

Table 3-16. Annual average concentrations (ppb) of n-hexane, styrene, and toluene in the Houston area for 1992-1997.

Site	Parameter	1992	1993	1994	1995	1996	1997
HRM01	n-Hexane	**3.08***	2.78	2.03	2.37	1.44	1.75
HRM03	n-Hexane	1.24	1.03	1.04	1.15	0.89	0.89
HRM04	n-Hexane	0.87	0.63	0.70	0.64	0.61	0.51
HRM07	n-Hexane	1.98	1.77	1.23	1.22	1.01	1.30
HRM08	n-Hexane	1.04	0.99	1.09	1.01	0.74	0.76
HRM10	n-Hexane	1.01	0.77	0.74	0.67	0.64	0.62
HRM11	n-Hexane	0.57	0.52	0.41	0.47	0.25	0.30
Clinton	n-Hexane	Na	na	na	1.60	1.74	0.91
Aldino	n-Hexane	Na	na	na	0.52	0.52	0.62
Haden	n-Hexane	Na	na	na	0.93	1.09	1.00
Galveston	n-Hexane	Na	na	na	0.81	0.59	0.49
Sheldon	n-Hexane	Na	na	na	0.73	0.53	0.56
Durant St	n-Hexane	Na	na	na	na	0.43	0.59
HRM01	Styrene	1.33	**2.04***	1.35	0.63	0.79	0.52
HRM03	Styrene	1.25	1.30	1.48	1.11	0.84	0.45
HRM04	Styrene	0.73	1.37	0.21	0.15	0.10	0.09
HRM07	Styrene	0.52	0.60	0.23	0.21	0.08	0.09
HRM08	Styrene	0.36	0.57	0.36	na	0.05	0.09
HRM10	Styrene	0.24	0.91		0.30	0.02	0.06
HRM11	Styrene	0.23	0.65	0.15	na	0.03	0.05
Clinton	Styrene	Na	na	na	0.16	0.12	0.09
Aldino	Styrene	na	na	na	0.00	0.04	0.07
Haden	Styrene	na	na	na	1.52	0.77	0.33
Galveston	Styrene	na	na	na	0.29	0.11	0.11
Sheldon	Styrene	na	na	na	0.28	0.12	0.83
Durant St	Styrene	na	na	na	na	0.01	0.03
HRM01	Toluene	3.76	2.59	2.38	2.12	2.04	2.18
HRM03	Toluene	**4.60***	2.92	2.73	3.00	3.40	1.67
HRM04	Toluene	1.89	1.35	1.02	0.83	0.64	0.81
HRM07	Toluene	2.79	2.08	1.91	1.44	1.85	1.10
HRM08	Toluene	1.75	1.53	3.54	1.38	3.11	2.57
HRM10	Toluene	0.69	0.52	0.48	0.43	0.37	0.56
HRM11	Toluene	1.37	1.14	1.02	0.57	0.48	0.73
Clinton	Toluene	na	na	na	2.07	2.39	1.45
Aldino	Toluene	na	na	na	0.91	1.00	1.16
Haden	Toluene	na	na	na	3.16	1.57	1.59
Galveston	Toluene	na	na	na	1.41	1.13	0.97
Sheldon	Toluene	na	na	na	1.22	0.81	1.21
Durant St	Toluene	na	na	na	na	0.89	0.99

* Maximum annual average concentration.

Table 3-17. Annual average concentrations (ppb) of total xylenes and trichloroethylene in the Houston area for 1992-1997.

Site	Parameter	1992	1993	1994	1995	1996	1997
HRM01	Total Xylenes	2.49	1.80	1.45	1.37	1.48	1.47
HRM03	Total Xylenes	**2.58***	1.70	1.56	1.83	1.40	1.08
HRM04	Total Xylenes	1.00	0.94	0.76	0.60	0.40	0.52
HRM07	Total Xylenes	2.22	1.26	1.11	0.86	1.10	0.77
HRM08	Total Xylenes	1.87	2.69	1.65	1.85	1.36	1.44
HRM10	Total Xylenes	0.74	0.75	0.59	0.91	0.16	0.29
HRM11	Total Xylenes	0.65	0.56	0.65	0.39	0.18	0.30
Clinton	Total Xylenes	na	na	na	1.56	1.27	0.66
Aldino	Total Xylenes	na	na	na	0.33	0.57	0.57
Haden	Total Xylenes	na	na	na	1.97	1.19	0.90
Galveston	Total Xylenes	na	na	na	1.22	0.62	0.57
Sheldon	Total Xylenes	na	na	na	0.78	0.44	0.64
Durant St	Total Xylenes	na	na	na	na	0.31	0.38
HRM01	Trichloroethylene	0.06	0.06	0.05	0.05	0.05	**0.20***
HRM03	Trichloroethylene	0.06	0.08	0.05	0.04	0.04	0.14
HRM04	Trichloroethylene	0.06	0.05	0.04	0.05	0.06	0.12
HRM07	Trichloroethylene	0.06	0.06	0.06	0.05	0.05	0.13
HRM08	Trichloroethylene	0.07	0.04	0.04	0.04	0.04	0.35
HRM10	Trichloroethylene	0.06	0.03	0.03	0.32	0.02	0.10
HRM11	Trichloroethylene	0.03	0.10	0.03	0.04	0.04	0.11
Clinton	Trichloroethylene	na	na	na	0.01	0.00	0.00
Aldino	Trichloroethylene	na	na	na	0.00	0.00	0.02
Haden	Trichloroethylene	na	na	na	0.01	0.00	0.01
Galveston	Trichloroethylene	na	na	na	0.01	0.00	0.00
Sheldon	Trichloroethylene	na	na	na	0.01	0.00	0.01
Durant St	Trichloroethylene	na	na	na	na	0.00	0.01

* Maximum annual average concentration.

Table 3-18. Selected $PM_{2.5}$ elemental concentrations at Houston area monitors in 1997-1998 determined by x-ray fluorescence.

Element	Elemental $PM_{2.5}$ concentrations ($\mu g/m^3$) on non-transport, chemistry sampling days by site							
	HRM Site 7	HRM Site 3	Bingle	Clinton	Swiss & Monroe	Shell Westhollow	NW Harris-Tomball	Galveston Airport
NA	0.1363	0.0789	0.0823	0.0793	0.0724	0.0693	0.0751	0.1607
MG	0.0365	0.0288	0.0370	0.0330	0.0306	0.0316	0.0306	0.0543
AL	0.2304	0.2068	0.1861	0.1760	0.1768	0.2142	0.1571	0.2236
SI	0.5578	0.5719	0.4570	0.4766	0.4327	0.5153	0.3757	0.5106
P	0.0007	0.0030	0.0005	0.0003	0.0006	0.0002	0.0003	0.0002
S	1.9737	1.9242	2.0965	2.1510	1.7464	1.7198	1.6964	1.4656
CL	0.0216	0.0138	0.0112	0.0346	0.0050	0.0161	0.0080	0.0905
K	0.1772	0.1176	0.1314	0.1056	0.1064	0.1273	0.1151	0.0905
CA	0.1431	0.1785	0.1034	0.3949	0.0929	0.0896	0.0556	0.0838
TI	0.0194	0.0158	0.0180	0.0146	0.0159	0.0177	0.0115	0.0158
V	0.0050	0.0057	0.0027	0.0050	0.0032	0.0015	0.0015	0.0027
CR	0.0024	0.0011	0.0014	0.0013	0.0040	0.0013	0.0009	0.0008
MN	0.0045	0.0081	0.0062	0.0078	0.0052	0.0049	0.0034	0.0029
FE	0.2091	0.2225	0.1992	0.2104	0.1696	0.1915	0.1247	0.1554
CO	0.0001	0.0002	0.0002	0.0001	0.0024	0.0002	0.0001	0.0002
NI	0.0019	0.0025	0.0017	0.0023	0.0021	0.0012	0.0007	0.0012
CU	0.0140	0.0295	0.0042	0.0109	0.0089	0.0049	0.0119	0.0033
ZN	0.0139	0.0303	0.0256	0.0297	0.0170	0.0211	0.0126	0.0083
GA	0.0003	0.0002	0.0000	0.0003	0.0003	0.0001	0.0001	0.0001
AS	0.0012	0.0012	0.0014	0.0013	0.0019	0.0018	0.0019	0.0005
SE	0.0009	0.0010	0.0008	0.0009	0.0007	0.0009	0.0009	0.0006
BR	0.0056	0.0055	0.0055	0.0050	0.0045	0.0044	0.0039	0.0035
RB	0.0004	0.0003	0.0003	0.0003	0.0003	0.0003	0.0003	0.0004
SR	0.0012	0.0020	0.0010	0.0016	0.0008	0.0012	0.0008	0.0011
Y	0.0001	0.0002	0.0001	0.0002	0.0001	0.0002	0.0001	0.0001
ZR	0.0008	0.0007	0.0017	0.0006	0.0008	0.0008	0.0006	0.0007
MO	0.0003	0.0004	0.0004	0.0006	0.0006	0.0005	0.0002	0.0003
PD	0.0005	0.0009	0.0012	0.0006	0.0012	0.0008	0.0007	0.0008
AG	0.0013	0.0012	0.0011	0.0007	0.0019	0.0012	0.0010	0.0007
CD	0.0005	0.0011	0.0010	0.0009	0.0017	0.0011	0.0009	0.0024
IN	0.0004	0.0012	0.0009	0.0010	0.0008	0.0007	0.0004	0.0008
SN	0.0009	0.0020	0.0012	0.0012	0.0014	0.0014	0.0021	0.0015
SB	0.0030	0.0028	0.0034	0.0025	0.0032	0.0012	0.0023	0.0011
BA	0.0080	0.0251	0.0039	0.0034	0.0058	0.0056	0.0068	0.0143
LA	0.0029	0.0054	0.0052	0.0055	0.0035	0.0080	0.0054	0.0036
AU	0.0002	0.0002	0.0003	0.0003	0.0004	0.0003	0.0002	0.0002
HG	0.0001	0.0001	0.0001	0.0003	0.0003	0.0002	0.0001	0.0001
TL	0.0001	0.0001	0.0002	0.0001	0.0000	0.0001	0.0001	0.0002
PB	0.0024	0.0047	0.0042	0.0039	0.0046	0.0037	0.0031	0.0045

4. HUMAN AIR POLLUTION EXPOSURE ASSESSMENT

4.1 OVERVIEW OF THE EXPOSURE MODELING APPROACH

Estimation of the health benefits of air quality improvements requires characterization of the population's exposure to the air pollutants of concern under baseline and future conditions. For annual assessment, one needs to know not only the distribution of air pollutant concentrations but also how many people were exposed to those concentrations and for how many days. Thus, the primary measure of exposure in this study is the number of person-days of exposure above the NAAQS. For annual average exposures, this reduces to the number of people exposed to concentrations above the NAAQS.

A spatially resolved, grid-based approach is used to estimate the exposure of Houston residents in this study. The Regional Human Exposure (REHEX-II) model is used to quantify both microenvironmental and "all outdoors" exposure estimates (Lurmann et al., 1989; Lurmann and Korc, 1994). Microenvironmental and "all outdoors" exposure estimates are reported for ozone. For $PM_{2.5}$, only estimates of exposure to outdoor pollution levels are reported. The microenvironmental method incorporates human time-activity data and the modifying effects of enclosures, such as buildings and vehicles, on exposure estimates. The amount of time spent in various microenvironments and estimates of the air pollutant concentrations in those microenvironments, which are often different than ambient concentrations, are combined to estimate the time-weighted total human exposure for the average times of interest. The REHEX-II model was developed to facilitate microenvironmental exposure estimations, but it also provides a convenient framework to carry out the simpler grid-based "all outdoors" calculations.

Ozone concentrations tend to be lower indoors than outdoors because of the absence of indoor sources, the rapid deposition of ozone on indoor surfaces, and the reduced or nonexistent photochemistry indoors (Weschler, 1989; Weschler et al., 1991). Buildings with open windows and good cross ventilation have indoor levels that are typically 60 to 90 percent of outdoor levels. Buildings with low air exchange rates (i.e., tightly built with closed windows) have low indoor-to-outdoor (I/O) ozone concentration ratios (less than 0.3). Ozone concentrations are particularly low in air-conditioned buildings because, in order to conserve energy and costs, they have low air exchange rates and pass air through ducts and sometimes filters, which enhance ozone losses.

Houston is a heavily air-conditioned city. The highest daily ozone concentrations tend to occur when the maximum temperature occurs, so maximum air-conditioning use and ozone are often coincident in time. The extensive use of air-conditioning in Houston during the warm ozone season suggests that most residents are likely to be inside air-conditioned buildings when the high outdoor concentrations occur. As in any city, there will of course be some individuals (children, workers, joggers, etc.) exposed to high outdoor concentrations as well. These are the individuals that the NAAQS seek to protect. Nevertheless, on average, one expects to see larger differences between ambient concentrations and actual personal exposure concentrations in Houston than in other less air-conditioned cities.

Indoor particle concentrations result from infiltration of ambient air to the indoor environment and from indoor sources. Offices and residences without significant indoor sources tend to have indoor $PM_{2.5}$ mass levels somewhat below the corresponding outdoor levels (Pellizari et al., 1992; Clayton et al., 1993). Indoor and outdoor levels of $PM_{2.5}$ sulfate are usually quite similar presumably because there are few indoor sources of sulfate and it occurs in a size range that has a long lifetime with respect to deposition loss (Dockery and Spengler, 1981; Suh et al., 1992, 1993). The principal indoor source of $PM_{2.5}$ is environmental tobacco smoke (ETS) (Wallace, 1996). Food preparation, especially of fried or blackened foods, vacuuming, wood stoves, and fireplaces can also contribute to indoor $PM_{2.5}$ concentrations (Samet and Spengler, 1991). Personal PM exposure monitoring studies have consistently reported that individuals are exposed to higher levels than are observed at ambient monitoring sites (Lioy et al., 1990; Ozkaynak et al., 1996; Janssen et al., 1995; Janssen, 1998). The high personal PM exposures are usually associated with being near traffic, being in a vehicle, and/or ETS exposure. The NAAQS are principally designed to protect the population from exposure to pollutants of outdoor origin. The current understanding of exposure to pollutants of outdoor origin suggests the indoor and outdoor $PM_{2.5}$ concentrations are more highly correlated and closer in magnitude, than indoor and outdoor ozone (Wilson and Suh, 1997; U.S. Environmental Protection Agency, 1996; Alzona et al., 1979).

If the majority of the population spends 90 percent of their time indoors where concentrations may be different than outdoors, why do we bother with "all outdoor" exposure estimates? The reason is that virtually all of the air pollution epidemiological studies have used ambient concentrations as surrogates for actual personal exposure in the development of exposure-response functions. Clinical health effects studies report their findings using actual exposure concentrations, but their health-end points are often more difficult to value economically. Thus, as explained in Section 5, in order to use the most recent and relevant health effects studies, one must apply epidemiological exposure-response functions to estimates of ambient exposure.

4.2 POPULATION

Population data for the two planning years associated with the air quality data, 1993 and 2007, are needed for the exposure assessment. In order to maximize the spatial resolution of the assessment, the population data were compiled with the same 4 x 4 km spatial resolution that was used to map the air quality data. Based on human time-activity considerations, we felt it was important to separate the population by gender and age. The 1990 census track population data were first aggregated into 4 x 4 km grid squares separately for males and females, and ages 0-4 years, 5-19 years, 20-64 years, and 65+ years old. MAPINFO® software was used to grid the census tract population data. Population growth to 1993 and 2007 was accomplished using the county-wide ratios of 1993 to 1990 and 2007 to 1990 populations for our age and gender groups. County populations by age and gender for 1990, 1993 and 2007 were obtained from the Texas Cancer Data Center (Texas Cancer Data Center, 1999). These were compiled on a 45 x 47 grid encompassing the greater Houston area. They include residents of Harris County and nine surrounding counties (Brazoria, Chambers, Fort Bend, Galveston, Grimes, Liberty, Montgomery, Waller, and Wharton).

The approach employed in this study for exposure assessment relies heavily on ambient air quality monitoring data. We sought to include the population for which we could justifiably assign exposure. The ozone monitoring network, shown in Figure 3-2, has the best spatial coverage in the Houston area. We chose to include all 4 x 4 km grids (or exposure districts) that were located within 30 km of ozone monitoring stations. This consists of 721 exposure districts that include 94 percent of the population on the full grid. **Figures 4-1 and 4-2** show the Houston area gridded total population data used in the study. The total population estimates are 3.76 million for 1993 and 4.81 million for 2007 for the exposure districts. **Table 4-1 and Figure 4-3** show the population age and gender distributions.

The same population data and exposure districts were used for assessing ozone and $PM_{2.5}$ exposures even though the PM monitoring network, shown in Figure 3-5, provides less spatial coverage than that for ozone. It was essential to use the same population for comparison of the health benefits of reductions in PM and ozone exposures. Limiting the exposure districts to those close to PM monitors may have biased the ozone results. Hence, while our approach involved greater extrapolation of the PM observations than one would like, we felt it probably introduced less bias into the overall study than possibly ignoring portions of the high ozone exposure region.

4.3 HUMAN TIME-ACTIVITY

Profiles of human time-activity are needed for the microenvironmental ozone exposure simulations. In the simulations, the population is subdivided into groups that follow 100 to 1,000 different diurnal patterns of locations each day. The diurnal patterns are resolved in 10 min increments and are derived from time-activity survey data. Several human time-activity databases were initially considered for use in the study. The databases were the 1992-1994 National Human Activity Pattern Survey (NHAPS), the 1988-1990 California Activity Pattern (CAP) Survey, and city-specific surveys from the early 1980s for Denver, Washington DC, and Cincinnati. All of these surveys were designed to collect time-activity data and other information that would be useful in human exposure assessment. We chose the NHAPS database because it was the only one that included data for residents of Texas.

The NHAPS was designed to obtain seasonally and nationally representative profiles of human activity using the 24-hr recall diary methodology (Nelson et al., 1994; Robinson and Blaire, 1995). Telephone interviews of approximately 9000 individuals were conducted over two years to obtain minute-by-minute diaries of their activities and the locations on the day prior to the telephone interview, plus supplemental demographic information that could be used in exposure assessment. The survey data are very detailed and include the times of day when individuals are engaged in different activities (see Tsang and Klepeis, 1996). We elected to use the data aggregated into the locations and activity categories shown in **Table 4-2**. **Figure 4-4** shows the mean duration of time spent in the different locations by NHAPS residents of four regions of the contiguous 48 states (from Klepeis et al., 1996). It shows that time use is quite similar in the different regions, on average.

The amount of time spent outdoors is a critically important parameter for assessing the exposure of individuals to ozone and the outdoor-generated component of PM. Time-activity data almost always show a high degree of individual-to-individual variability in this parameter. Most surveys also show that the average amount of time spent outdoors varies by season, day of week, age, and gender. We examined the variability in time spent outdoors by region (48 states, southern states, non-southern states, and Texas), season (summer and non-summer), weekday/weekend, age, and gender. **Tables 4-3 through 4-6** show the mean percent of time spent outdoors and the number of profiles (or individuals with 24-hr diaries) on which the means are based.

The NHAPS data indicate that, on average, people in Texas, the southern states, and the contiguous 48 states spend 91, 97, and 100 minutes per day outdoors, respectively. In summer, the survey participants in Texas, the southern states, and the contiguous 48 states spent 135, 138, and 151 min per day outdoors, respectively. Likewise, in the non-summer months, the survey participants in Texas, the southern states, and the contiguous 48 states spent 77, 76, and 75 min per day outdoors, respectively. The mean values for all days are based on relatively large sample sizes for this type of survey: 536 profiles for Texas, 3106 profiles for the southern states, and 8901 profiles for contiguous 48 states. Overall, these data suggest the average time spent outdoors is similar (within 16 min per day) in the different regions.

When the data are stratified into smaller subsets, the time outdoors data show larger variations. Unfortunately, when the Texas data alone are stratified into the 32 groups that we planned to use for the Houston exposure assessment (two seasons, two day types, four age groups and two gender groups), there are an insufficient number of profiles in the smaller strata. Similarly, even the database for the southern states alone is too small to be representative in the summer season for the smaller sized aged groups. The data indicate that when there are fewer than 20 or 30 profiles for a strata, the mean amount of time outdoors often differs considerably (up to a factor of two) from the "all region" mean for that strata. We believe these differences represent insufficient numbers of samples rather than true regional differences. Thus, we used the NHAPS profiles collected in all 48 contiguous states for age groups 0-4, 5-19, and 65+ years in the summer season, and the NHAPS profiles collected in the southern states for the 20-64 years age group in summer and for all age groups in the non-summer period. **Table 4-7** shows the mean amount of time outdoors per day in the final database. With this approach, the amount of time outdoors in summer for the Houston simulations is slightly higher than the southern states average: 148 versus 138 min per day.

The subset of NHAPS data that we used (3,667 profiles) is probably the best available data set for current exposure assessment in Houston. Clearly, there is a need to collect region-specific time-activity data for the Houston area to improve characterization of exposure in the area. The NHAPS database does not include enough data for Houston residents. It also lacks information on the types and operational status of heating, ventilating, and air-conditioning (HVAC) systems in the microenvironments occupied by individuals, which we believe are quite important for accurate ozone exposure assessment.

4.4 MICROENVIRONMENTAL FACTORS

Few measurements of indoor ozone levels have been made in the Houston area. Houston residences were sampled in the 1980s and indoor ozone levels were found to be very low regardless of outdoor levels (Contant et al., 1987). The low indoor ozone levels were attributed to the use of central and room air-conditioners in the Houston residences. Avol et al. (1998) measured ozone inside and outside of 125 southern California homes and reported an average indoor/outdoor ratio of 0.37. Lurmann et al. (1994) measured ozone at 48 southern California schools and found an average indoor/outdoor ratio of 0.31 for the hours of the day in which the rooms were used. Air-conditioned homes and schools had consistently lower I/O ratios than non-air-conditioned buildings in the two southern California studies. Druzik et al. (1990) measured ozone in public buildings and found indoor/outdoor ratios of 0.1 to 0.2 in buildings with air-conditioning and low air exchange rates. Nasaroff and Cass (1986) and Weschler (1989, 1991) have found a wide range of I/O ratios in commercial buildings and shown that the differences can be modeled reasonably well if the air exchange rate and type of filtration system is known. Carbon filters are known to remove ozone quite effectively. As a practical matter, we don't have enough information on building air exchange rates or filtration systems to apply detailed models on a widespread basis. What can be determined from the measurements and models is that most buildings with air-conditioning and low air exchange rates will have low I/O ratios for ozone.

Indoor concentrations were estimated from the outdoor concentrations and fixed I/O ratios for the Houston microenvironmental ozone simulations. **Table 4-8** lists the factors used in the simulations. In most indoor environments the ozone was estimated as 20 percent of the outdoor concentration. This factor is conservatively high for air-conditioned buildings. We assumed virtually all schools, offices, and public buildings would use air-conditioning in the ozone season. However, residential air-conditioning probably is not affordable for the economically disadvantaged residents. We extracted and gridded the household income data from the 1990 Census and chose $17,500 as the cutoff for air-conditioning affordability. This level was slightly above the government-defined poverty level for a family of four in 1990. The gridded data show significant spatial variation in the household income and these variations were accounted for in the model. The grid-average percentage of households with income below this level ranges from 1 to 23 percent. An indoor/outdoor ozone ratio of 0.70 was used for the non-air-conditioned homes, which is a fairly high ratio for ozone. The population in 1993 and 2007 was apportioned into groups with air-conditioned and non-air-conditioned homes based on the portion of households with 1990 income below the cutoff. Overall, the approach used to estimate indoor ozone concentrations is simplistic, yet it is consistent with available data and conservative in the sense that it is likely to overestimate concentrations, on average.

4.5 OZONE EXPOSURE ESTIMATES

Distributions of ozone exposure were estimated using the REHEX-II model for several averaging times and emissions scenarios, and with all outdoor and microenvironmental approaches. The exposure calculations produce large amounts of detailed information, which we have condensed and summarized in terms of three exposure metrics for comparison

purposes. These metrics are the number of person-days per year with 1-hr maximum, 8-hr maximum, and 24-hr average concentrations above various concentration thresholds. The estimates for the 1-hr maximum and 24-hr average are used with the ozone exposure-response functions (see Section 5) to estimate the symptoms experienced by the Houston population. The estimates for the 8-hr maximum are presented because this is the metric for the newest ozone NAAQS.

4.5.1 All Outdoor Ozone Exposure Estimates

Tables 4-9 and 4-10 list the estimated person-days per year of exposure to 1-hr maximum and 8-hr maximum daily ozone concentrations using the "all outdoors" approach. **Figures 4-5 and 4-6** show the estimated population exposure distributions for these averaging times. Recall the regional population is 3.7 million in this period. The results for 1993-1995 indicate that residents experienced 26.2 million person-days of exposure to 1-hr maximum concentrations above the 120 ppb NAAQS and 72.6 million person-days of exposure to 8-hr maximum concentrations above the 80 ppb NAAQS. That is, we estimate almost three times as many exposures to levels above the 8-hr NAAQS as above the 1-hr NAAQS in Houston. The figures show that the estimates of exposures decrease rapidly as the concentration threshold increases; nevertheless, the estimates show millions of person-days of exposure at higher levels. For the 1-hr daily maximum, the estimates are 8.2 million, 2.1 million, and 340,000 exposures per year above 140, 160, and 180 ppb, respectively. For the 8-hr daily maximum, the estimates are 37.4 million, 18.5 million, 2.7 million, and 174,000 exposures per year above 90, 100, 120, and 140 ppb, respectively. The number of exposures to levels just below the NAAQS levels is also quite large. The estimates indicate 69.9 million exposures to 1-hr daily maximum concentrations above 100 ppb and 125 million exposures to 8-hr maximum concentrations above 70 ppb.

Table 4-9 and **Figure 4-7** show 1-hr daily maximum exposure results for the future-year cases. For the "all outdoors" 2007 base case, where the population is 4.8 million, the model estimates residents would experience 1.6 million, 26.7 million, and 77.7 million person-days per year of exposures to 1-hr maximum concentrations above 160, 120, and 100 ppb, respectively. Thus, even though the population is 28 percent higher in 2007 than 1993, the number of exposures to concentrations above 120 ppb is about the same as in the baseline period. In other words, the emission control measures already adopted and, therefore, included in the 2007 base case air quality projection produce just enough improvement in ozone air quality to offset the population growth. The estimates show benefits at the highest levels in 2007; the estimates are 26,000 and 1.58 million person-days per year above 200 and 180 ppb in the 2007 base case compared to 58,000 and 2.14 million in the 1993-1995 case.

Seven alternate scenarios are considered for 2007. Implementation of the 15 percent VOC emission reductions and 25 percent NO_x emission reductions (across the board) is estimated to reduce the number of person-days of exposure to 1-hr maximum concentrations above 120 ppb from 26.7 million to 24.2 million. Full implementation clean diesel fuel on top of the 15 percent VOC emission reductions is estimated to be slightly more effective than the 25 percent NO_x reduction: 22.9 million compared to 24.2 million person-days of exposure. NO_x emission reductions of 45, 65, 75, and 85 percent are estimated to reduce the number of

person-days of exposure to 1-hr maximum concentrations above 120 ppb to 19.6, 10.1, 3.8, and 0.24 million per year, respectively. The exposure results for the "rollback to the 1-hr NAAQS" scenario and the 85 percent NO_x emissions reduction (with 15 percent VOC emission reduction) are very similar; less than 239,000 person-days of exposure to concentrations between 120 and 130 ppb are estimated to occur under these controlled conditions based on the "all outdoors" exposure assumption. The ozone NAAQS are statistical standards that allow infrequent exceedances and both of these latter scenarios essentially represent compliance with the standard.

Table 4-10 and **Figure 4-8** show the future-year "all outdoors" exposure distributions for the daily 8-hr maximum concentrations. For the 2007 base case, we estimate there would be 84.2 million, 18.7 million, and 2.1 million person-days per year of exposures to 8-hr maximum concentrations above 80, 100, and 120 ppb, respectively. The number of 8-hr exposures is lower in the 2007 base case than in 1993-1995 at the highest concentration levels, however, at the level of the standard we estimate there will be 16 percent more exposures in the 2007 base case than in 1993-1995. The air quality improvements incorporated into the 2007 base case are not sufficient to offset the 28 percent population growth. These results confirm the general belief that achieving compliance with the 8-hr standard will be more difficult than with the 1-hr standard.

The 8-hr exposure results for the 25 percent NO_x emission reduction and the full implementation of clean diesel fuel are similar. They indicate that the number of person-days of exposure to 8-hr concentrations above 80 ppb would be 80.2 million and 78.5 million per year, which are only slightly lower than the 84.2 million estimate for the 2007 base case. However, with 45, 65, and 75 percent NO_x emission reductions, the estimated person-days of exposure to 8-hr concentrations above 80 ppb are 71.7 million, 52.3 million, and 34 million per year. The largest improvements are estimated for the 85 percent NO_x emission reduction and "rollback to 1-hr NAAQS" cases where the estimated person-days of exposure to 8-hr concentrations above 80 ppb decrease to 10.9 million and 7.87 million per year, respectively, in 2007. Achieving compliance with the 1-hr NAAQS reduces the 2007 exposures above the level of the 8-hr NAAQS by about 90 percent, which is a large improvement. It still may not be enough improvement to fully mitigate the 8-hr exposures of concern (i.e., there are still 8 million person-days per year above 80 ppb).

The 24-hr average ozone exposure estimates are summarized in **Table 4-11**. There is no standard for this averaging time. The results for 1993-1995 indicate there were about 1.7 million, 9.1 million, 35 million, and 98 million person-days per year with 24-hr average ozone exposures above 70, 60, 50, and 40 ppb, respectively, in the Houston area. For the 2007 base case, the estimates are for about 1.6 million, 9.5 million, 40 million, and 118 million person-days per year with 24-hr average ozone exposures above 70, 60, 50, and 40 ppb, respectively. As for the other metrics, the upper tail of the exposure distribution shows similar numbers of events in the 2007 base case and in 1993-1995, while below 60 ppb there are more exposures in the future base case. The effects of the emission control scenarios on the 24-hr exposures are similar to those for the 8-hr exposures. The 25 percent NO_x emission reduction and the full implementation of clean diesel fuel are estimated to reduce

exposures above 60 ppb from 9.5 million to 8.4 million and 7.8 million person-days per year, respectively. The 45, 65, and 75 percent NO_x emission reductions are estimated to reduce exposures to 24-hr concentrations above 60 ppb by 26, 58, and 75 percent, respectively. The 85 percent NO_x emissions reduction case and the "rollback to the 1-hr NAAQS" case are estimated to have 1.2 and 1.0 million exposures per year above 60 ppb, which are reductions of 88 and 90 percent from the 2007 base case.

4.5.2 Microenvironmental Ozone Exposure Estimates

The microenvironmental exposure simulations include the estimated effects of human time-activity and the modifying effects of enclosures on exposure concentrations. With the exception of the time-activity profiles and the indoor/outdoor ratios, all of the inputs for the microenvironmental simulations were identical to those for the "all outdoors" simulations. **Tables 4-12 and 4-13** compare the microenvironmental and "all outdoors" results for 1993-1995 conditions. **Figures 4-9 and 4-10** show the results for both 1-hr and 8-hr maximum daily concentrations, respectively. The microenvironmental estimates of exposures to concentrations above the NAAQS levels are dramatically lower than the "all outdoors" estimates. The microenvironmental simulation estimates 4 million person-days per year with exposures to daily 1-hr maximum concentrations above 120 ppb, compared to 26 million in the "all outdoors" simulation. The microenvironmental simulation estimates 3.9 million person-days per year with exposures to daily 8-hr maximum concentrations above 80 ppb, compared to 72 million in the "all outdoors" simulation. Thus, at the levels of the standards, the microenvironmental estimates are factors of 6 and 20 lower than the 1-hr and 8-hr "all outdoors" estimates, respectively.

Larger differences are evident at higher exposure thresholds. For example, the microenvironmental simulation estimates 5,800 person-days per year with exposures to daily 1-hr maximum concentrations above 200 ppb, compared to 58,000 in the "all outdoors" simulation. The microenvironmental simulation estimates 62,000 person-days per year with exposures to daily 8-hr maximum concentrations above 120 ppb, compared to 2.7 million in the "all outdoors" simulation. The differences in the upper tails of the 8-hr exposure distributions can be as large as a factor of 50 between the "all outdoors" and microenvironmental estimates.

Table 4-14 and Figure 4-11 compare microenvironmental and "all outdoors" exposure results for future-year cases. The large differences evident in the 1993-1995 results are also shown in the future-year results (as expected). For example, with 45 percent NO_x emissions reduction in 2007, the model estimates 2.8 million and 19.6 million exposures to 1-hr daily maximum concentrations above 120 ppb with the microenvironmental and "all outdoors" approaches, respectively.

The microenvironmental exposure simulations provide estimates for different demographic groups. **Table 4-15** shows the average per capita exposures to 1-hr maximum concentrations above 80, 120, and 160 ppb in 1993-1995 by gender-age groups. On a per

capita basis, female adults have fewer exposures than female children and males of all ages. Male children are estimated to have the highest per capita exposures, more than double those of adult females. On average, the estimates suggest that female and male children have 14 and 43 percent, respectively, higher frequency of exposures to 120 ppb than the average resident. Female and male adults have about 46 percent lower and 23 percent higher frequency of exposures to 120 ppb than the average resident. These exposure differences primarily result from time-activity pattern differences.

The microenvironmental results are consistent with the time-activity data and ozone I/O ratios used in the simulations. During the daylight hours, 10 to 20 percent of the population is outdoors at any particular hour. Only the fraction of the population that is outdoors or in well-ventilated buildings is likely to receive ambient exposures; the majority are likely to be exposed to levels well below the ambient levels due to ozone losses indoors. At high ambient concentrations, only those individuals that are outdoors for the whole hour are assigned the high level in the microenvironmental simulations. The number of people that are outdoors for long time periods (6-8 hrs) is small. The subset of NHAPS data used in this study indicates that 2.4 hrs are spent outdoors, on average, in the summer. Hence, it is not surprising that the simulation results for 8-hr exposure intervals are much lower than the corresponding results for "all outdoors" conditions.

These microenvironmental simulations were made with I/O ratios and time-activity data that may still overestimate exposures in the Houston area. Ozone in air-conditioned buildings is probably lower than 20 percent of outdoor levels in most cases and, given the Houston climate, fewer residents may be outside during the warm and humid afternoon hours than suggested by the NHAPS data. Both of these factors should be investigated in the Houston region. If these suspicions are true, the microenvironmental estimates are probably conservatively high.

The time-use patterns and heavily air-conditioned buildings may mean that Houston residents would receive fewer exposures to ozone levels of concern than residents of other cities with exactly the same ambient air quality conditions. This factor should be considered when interpreting the results for ozone symptoms because the ratio of actual-to-ambient exposures may be lower in Houston than in the cities where the ambient exposure-health response functions were derived. Exposure-response functions developed in other cities will likely overestimate ozone symptoms in Houston.

4.5.3 Spatial Patterns of Ozone Exposures

The spatial pattern of 1-hr maximum exposures above 120 ppb and 8-hr maximum exposures above 80 ppb in the Houston area in 1993-1995 are shown in **Figures 4-12 and 4-13**, respectively. These results are based on the "all outdoors" exposure assumption. The patterns show that the central and northern portions of Houston have the highest number of exposures. The spatial pattern of exposures with the microenvironmental approach is shown in **Figures 4-14 and 4-15**. The microenvironmental and "all outdoors" patterns are similar, but the magnitudes are a lot lower for the microenvironmental case.

Figures 4-16, 4-17, and 4-18 show the estimated future-year pattern of 1-hr maximum ozone exposures above 120 ppb for the 2007 base case, the 45 percent NO_x plus 15 percent VOC emission reductions case, and the 85 percent NO_x plus 15 percent VOC emission reduction case, respectively. The spatial pattern of exposures for the 2007 base case is very similar to the 1993-1995 pattern (as expected). As precursor emissions are reduced, the areal extent of exposures above the levels of the standard shrinks. These results are consistent with the TNRCC's estimates for reductions in children's exposures based on photochemical modeling of the September 1993 episode (Smith et al., 1998).

Table 4-16 shows the average per capita exposure to 1-hr concentrations above 80, 120, and 160 ppb by county. Grimes, Liberty, Waller, and Wharton counties have virtually no exposures above 160 ppb. However, Grimes has relatively high per capita exposures to ozone above 120 ppb. Galveston, Montgomery, and Harris counties have higher per capita exposures above 160 ppb than other counties. Grimes, Montgomery, and Harris counties have higher per capita exposures above 120 ppb than other counties. Grimes and Montgomery counties have higher per capita exposures above 80 ppb than other counties. Overall, these patterns are consistent with the population distribution and ambient ozone concentration distribution in Houston.

4.6 $PM_{2.5}$ EXPOSURE ESTIMATES

$PM_{2.5}$ exposure estimates for the Houston area were performed with the REHEX-II model. The model was applied to estimate the frequency of exposures to daily and annual average $PM_{2.5}$ concentrations in 1993 and 2007. The simulations were made with the "all outdoors" exposure assumption. No diurnal variation of ambient concentration was represented since the underlying data were collected as 24-hr integrated samples. Only exposures to $PM_{2.5}$ mass were simulated; separate exposures to the chemical components were not carried out.

Results of the annual average simulation are shown in **Table 4-17 and Figures 4-19 through 4-21**. Figure 4-19 shows the distribution of annual average $PM_{2.5}$ exposures in 1993 and for Scenario 1 in 2007. Scenario 1 is one of three alternate base case projections for 2007; it has slightly higher estimated $PM_{2.5}$ concentrations than Scenarios 2 or 3. The results indicate a fairly narrow distribution of concentrations occur in 1993. There are 1.4 million people exposed to concentrations between 11.5 and 15 $\mu g/m^3$, about 2 million people exposed to concentrations between 15 and 17 $\mu g/m^3$, and the remaining 375,000 people are exposed to concentrations between 17 and 18 $\mu g/m^3$. The distribution for Scenario 1 in 2007 is shifted to slightly higher concentrations. We estimate 960,000 annual exposures between 11.5 and 15 $\mu g/m^3$, 2.3 million exposures between 15 and 17 $\mu g/m^3$, 1.08 million between 17 and 18 $\mu g/m^3$, 454,000 between 18 and 19 $\mu g/m^3$, and 18,000 between 19 and 19.5 $\mu g/m^3$ in 2007. Figure 4-20 shows the spatial pattern of estimated exposures in 2007 under Scenario 1. These results suggest that the majority of the Houston population is exposed to levels above the 15 $\mu g/m^3$ annual NAAQS.

Figure 4-21 shows the estimated exposures to annual average $PM_{2.5}$ above the 15 $\mu g/m^3$ annual NAAQS for various cases. We estimate 2.3 million of the 3.7 million residents were exposed to more than 15 $\mu g/m^3$ in 1993. The estimates for Scenarios 1, 2, and 3 in 2007 range from 3.68 million to 3.84 million people exposed to more than 15 $\mu g/m^3$ in the 2007 base case. That is, about 80 percent of the residents are likely to be exposed to $PM_{2.5}$ mass concentrations above the NAAQS in 2007 if no new control programs are implemented. With implementation of the 85 percent NO_x emissions reduction and 15 percent VOC emissions reduction specified in the 1998 ozone SIP, the ammonium nitrate and secondary organic material components of $PM_{2.5}$ are expected to decrease from their projected levels. These components are not the largest part of the $PM_{2.5}$ mass, so the effects are fairly small. The estimated number of people exposed to more than 15 $\mu g/m^3$ of $PM_{2.5}$ mass under the O_3 SIP Scenario is 3.56 million. Full implementation of the clean diesel fuel control scenario is estimated to have about the same effect as the O_3 SIP control program, which is an 11 percent reduction in the number of people exposed to concentrations greater than 15 $\mu g/m^3$.

Figure 4-22 shows the effects of the alternate scenarios on the number of people exposed to annual average concentrations above various thresholds (i.e., not just 15 $\mu g/m^3$). Only the simulated annual NAAQS rollback case brings the exposures anywhere close to the standard. As indicated in Section 3, the annual NAAQS rollback case assumes sufficient emission reductions are implemented to reduce the maximum annual concentration from 19 to 15 $\mu g/m^3$ in 2007. If the atmospheric system behaved linearly and the background remains at 11 $\mu g/m^3$, the simple rollback model estimates that a 50 percent reduction in Houston area emissions would be needed to achieve compliance with the annual NAAQS. Thus, even though it seems that ambient concentrations are not very much higher than the standard, the background is high and this means significant emissions reductions are likely to be needed to reach compliance. The ozone SIP control program and the clean diesel fuel control measure are each the equivalent of about a 5 percent reduction in $PM_{2.5}$ mass emissions.

Table 4-18 and Figure 4-23 show the estimated daily exposure modeling results for $PM_{2.5}$. All of the $PM_{2.5}$ 24-hr average exposures are well below the 65 $\mu g/m^3$ NAAQS. For the 1993 base case, there are approximately 51 million person-days of exposures per year to daily concentrations above 30 $\mu g/m^3$. For the 2007 base cases (Scenarios 1-3), the estimates are 115 to 125 million person-days of exposures per year to daily concentrations above 30 $\mu g/m^3$. It decreases to 107 million person-days above 30 $\mu g/m^3$ for the ozone SIP and clean diesel fuel scenarios. The estimated exposures to daily $PM_{2.5}$ levels above 40 $\mu g/m^3$ range from 4 to 9 million person-days per year for all of the 2007 scenarios except the annual NAAQS compliance. Using simplistic assumptions regarding the relationship between daily maximum and annual average PM concentrations, we estimate that there would be no daily exposures above 30 $\mu g/m^3$ and a 69 percent reduction in daily exposures above 20 $\mu g/m^3$ from the baseline conditions (Scenario 1) with $PM_{2.5}$ levels rolled back to the annual NAAQS.

4.6.1 Air Toxins Exposure Estimates

No attempt was made to estimate exposure to toxic compounds in a manner comparable to those for ozone and $PM_{2.5}$. There is linkage between the ozone, $PM_{2.5}$, and toxic compound precursor emissions. VOC emissions contribute to toxic gas concentrations as well as organic PM. Primary PM emissions also include toxic metals and other toxic trace compounds [e.g., polycyclic aromatic hydrocarbons (PAHs)]. The gaseous VOCs of principal concern for toxicity in Houston are benzene, 1,3-butadiene, and formaldehyde. The maximum annual concentrations between 1992 and 1997 were 3.5 ppb of benzene, 6.5 ppb of 1,3-butadiene, and 4.7 ppb of formaldehyde. These occurred at three different sites in Houston (benzene at HRM03, 1,3-butadiene at HRM07, and formaldehyde at Clinton). Only the benzene level exceeds the TNRCC screening level (1 ppb). The Houston area concentrations are quite similar to those found in other urban areas. For example the maximum benzene level is comparable to those in Los Angeles before introduction of California's reformulated gasoline. The maximum annual 1,3-butadiene level in Houston is higher than in Los Angeles; however, the formaldehyde level is lower than in Los Angeles. As discussed in the following sections, the health benefits of reductions from these levels are small even if the whole population was exposed to the maximum annual concentration. While reductions in the ambient concentrations of any toxic compound are desirable from a public health perspective (because they may contribute to cumulative risk), the health benefits of complete elimination of these three compounds are likely to be much smaller than the benefits of attaining the ozone and/or $PM_{2.5}$ NAAQS.

The 15 percent VOC emission reductions described in the ozone SIP may affect these compounds. All three compounds are emitted by gasoline-fueled vehicles. Benzene and 1,3-butadiene are emitted by some stationary sources. Formaldehyde is both emitted and photochemically produced. Formaldehyde and 1,3-butadiene also react fairly rapidly in the atmosphere. Given the current ozone control strategy's emphasis (NO_x emissions reductions rather than VOC emissions reductions), the effects are likely to be small. It should be noted that ambient benzene levels in the Los Angeles area were approximately halved by the introduction of California's reformulated gasoline, which is low in aromatics and other reactive components.

4.7 SUMMARY

Key findings of the exposure assessment are summarized below.

- The "all outdoors" ozone exposure estimates for 1993-1995 indicate that residents experienced 26.2 million person-days of exposure to 1-hr maximum concentrations above the 120 ppb NAAQS and 72.6 million person-days of exposure to 8-hr maximum concentrations above the 80 ppb NAAQS. That is, we estimate almost three times as many exposures to levels above the 8-hr NAAQS as above the 1-hr NAAQS in Houston.

- Seven alternate scenarios were considered for ozone in 2007. The results for the "all outdoors" cases are as follows. For the 2007 base-case, we estimate 26.7 million person-days of exposure to 1-hr maximum concentrations above 120 ppb (per year) which is almost identical to our estimates for 1993-1995. Implementation of a 15 percent VOC emissions reduction and a 25 percent NO_x emissions reduction (across the board) is estimated to reduce the number of person-days of exposure to 1-hr maximum concentrations above 120 ppb from 26.7 million to 24.2 million. Full implementation clean diesel fuel on top of the 15 percent VOC emissions reductions is estimated to be slightly more effective than the 25 percent NO_x reduction: 22.9 million compared to 24.2 million person-days of exposure. NO_x emissions reductions of 45, 65, 75, and 85 percent are estimated to reduce the number of person-days of exposure to 1-hr maximum concentrations above 120 ppb to 19.6, 10.1, 3.8, and 0.24 million per year, respectively. The later suggests the Ozone SIP strategy will reduce unhealthful 1-hr ozone exposures by 99 percent in 2007.

- The future-year "all outdoors" exposure estimates for the daily 8-hr maximum concentrations indicate there would be 84.2 million person-days per year of exposures to 8-hr maximum concentrations above 80 ppb. The number of 8-hr exposures above the standard are estimated to be 16 percent higher in the 2007 base case than in 1993-1995. The air quality improvements incorporated into the 2007 base-case are not sufficient to offset the 28 percent population growth. These results confirm the general belief that achieving compliance with the 8-hr standard will be more difficult than with the 1-hr standard.

- The 8-hr exposure results for the 85 percent NO_x emissions reduction and "rollback to 1-hr NAAQS" cases show the estimated person-days of exposure to 8-hr concentrations above 80 pbb decrease to 10.9 million and 7.9 million per year in 2007. Achieving compliance with the 1-hr NAAQS reduces the 2007 exposures above the level of the 8-hr NAAQS by about 90 percent, which is a large improvement. It still may not be enough improvement to fully mitigate the 8-hr exposures of concern (i.e., there are still 8 million person-days per year above 80 ppb).

- The microenvironmental estimates of exposures to concentrations above the NAAQS levels are dramatically lower than the "all outdoors" estimates. The microenvironmental simulation estimates 4 million person-days per year with exposures to daily 1-hr maximum concentrations above 120 ppb, compared to 26 million in the "all outdoors" simulation. The microenvironmental simulation estimates 3.9 million person-days per year with exposures to daily 8-hr maximum concentrations above 80 ppb, compared to 72 million in the "all outdoors" simulation. Thus, at the levels of the standards, the microenvironmental estimates are factors of 6 and 20 lower than the 1-hr and 8-hr "all outdoors" estimates, respectively.

- The estimated portion of the population exposed to annual average $PM_{2.5}$ above the 15 $\mu g/m^3$ annual NAAQS is 62 percent in 1993 and 79 percent in 2007 with additional control measures. The estimated portion of the population exposed to more than 15 $\mu g/m^3$ of $PM_{2.5}$ mass under the O_3 SIP Scenario is 74 percent or 3.5 million people.

Full implementation of the clean diesel fuel control scenario is estimated to have about the same effect as the O_3 SIP control program, which is an 11 percent reduction in the number of people exposed to concentrations greater than 15 $\mu g/m^3$.

- The area of Houston with exposure to annual average $PM_{2.5}$ concentrations above the NAAQS is almost the same area that experiences a relatively high ozone exposures. This suggests many of the same residents may be subjected to unhealthful concentrations of ozone and $PM_{2.5}$ over the course of the year.
- All of the $PM_{2.5}$ 24-hr average exposures are well below the 65 $\mu g/m^3$ NAAQS. For the 1993 base case, there are approximately 51 million person-days of exposures per year to daily concentrations above 30 $\mu g/m^3$. For the 2007 base cases (Scenarios 1-3), the estimates are 115 to 125 million person-days of exposures per year to daily concentrations above 30 $\mu g/m^3$.
- The gaseous VOCs of principal concern for toxicity in Houston are benzene, 1,3-butadiene, and formaldehyde. The maximum annual concentrations between 1992 and 1997 were 3.5 ppb of benzene, 6.5 ppb of 1,3-butadiene, and 4.7 ppb of formaldehyde. Only the benzene level exceeds the TNRCC screening level (1 ppb). The Houston area concentrations are quite similar to those found in other urban areas. The monitoring data are too geographically restricted to estimate the Houston area population exposure to these toxic compounds in a manner analogous to the ozone and $PM_{2.5}$ exposures.

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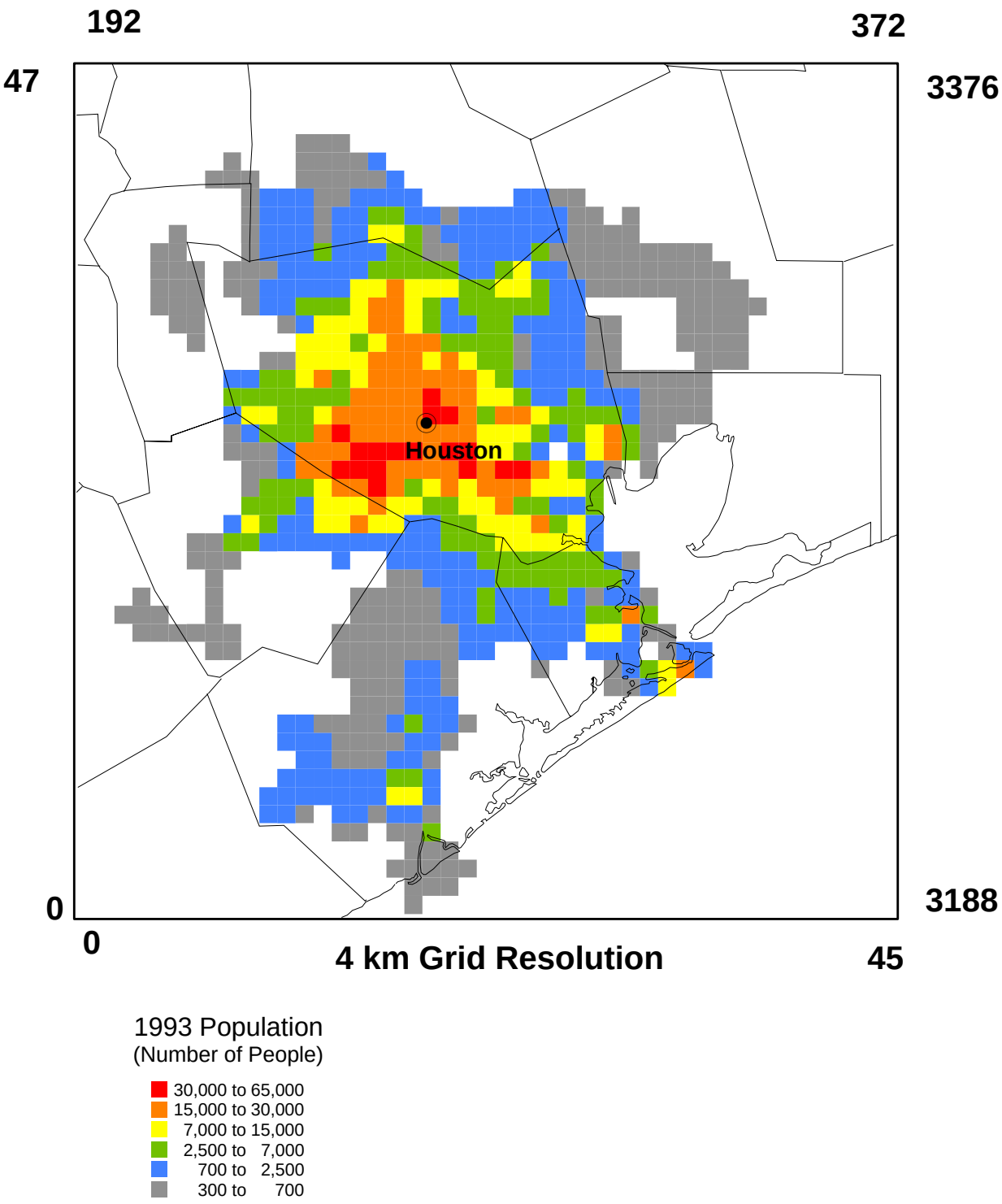

Figure 4-1. 1993 gridded population data for the Houston area.

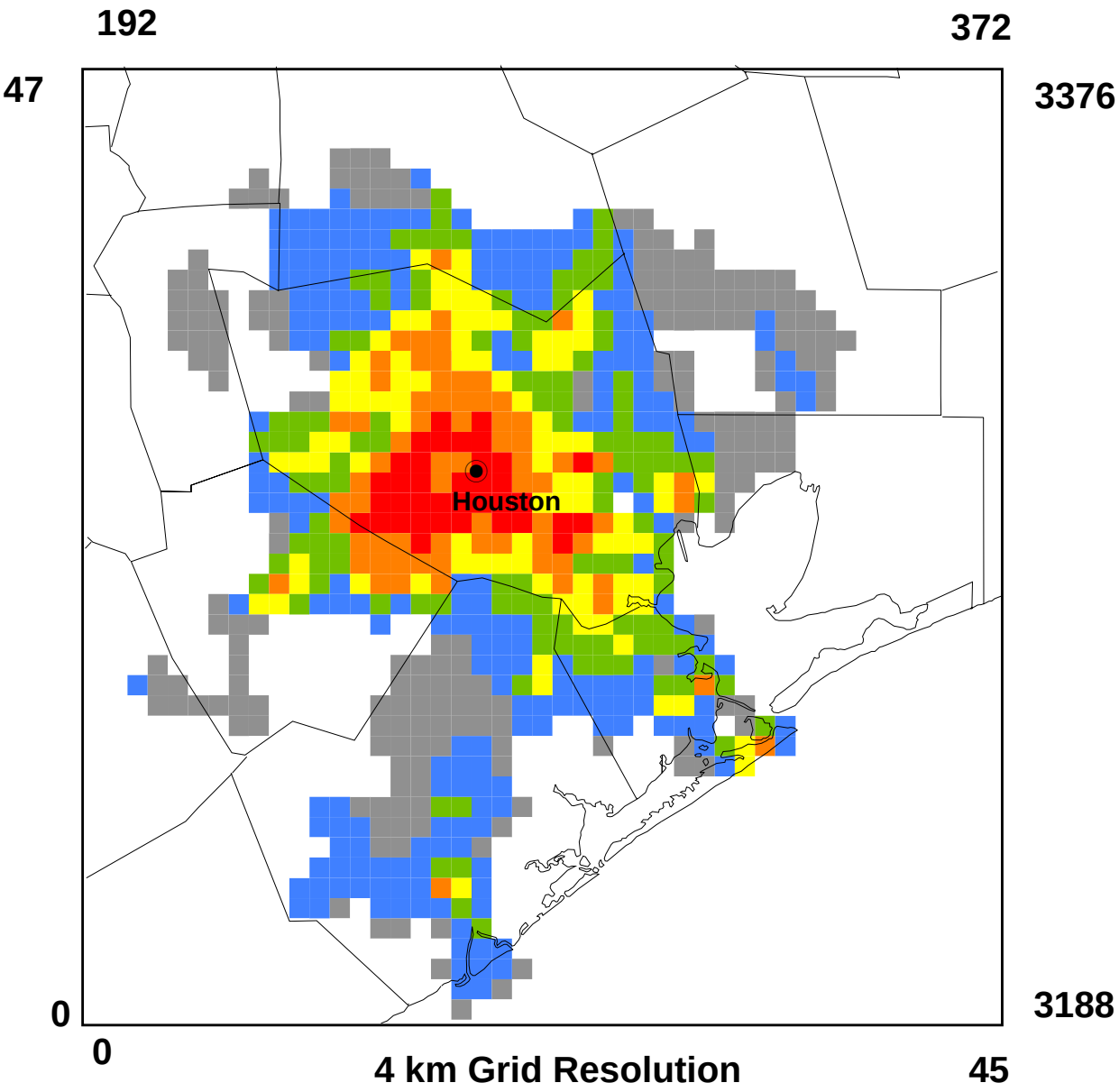

Figure 4-2. Estimated 2007 gridded population data for the Houston area.

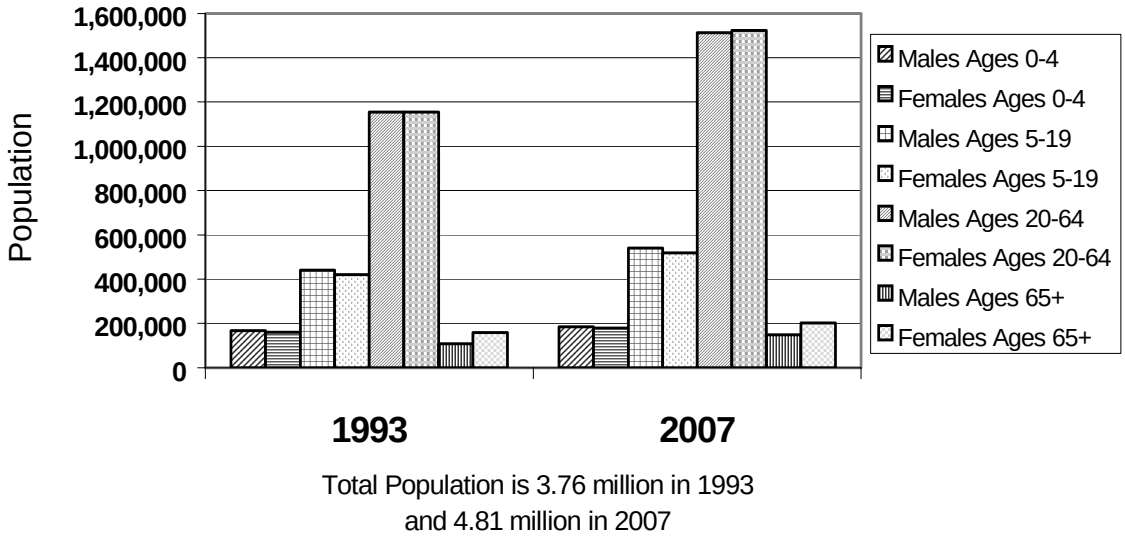

Figure 4-3. The population age and gender distribution in 1993 and estimated for 2007 in the Houston area.

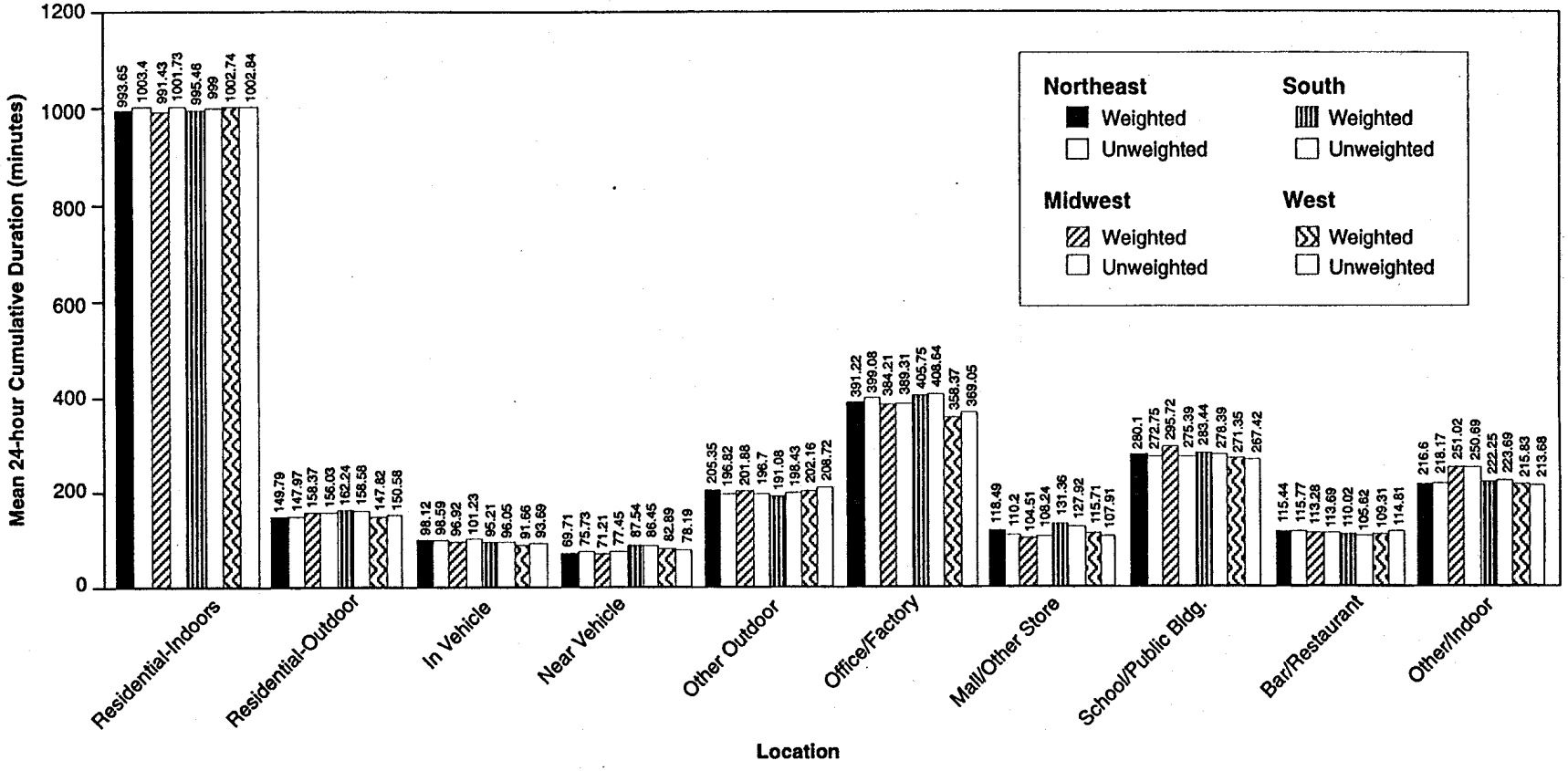

Figure 4-4. The weighted and unweighted mean 24-hr cumulative durations in each location for the different regions of the United States (from Klepeis et al., 1996).

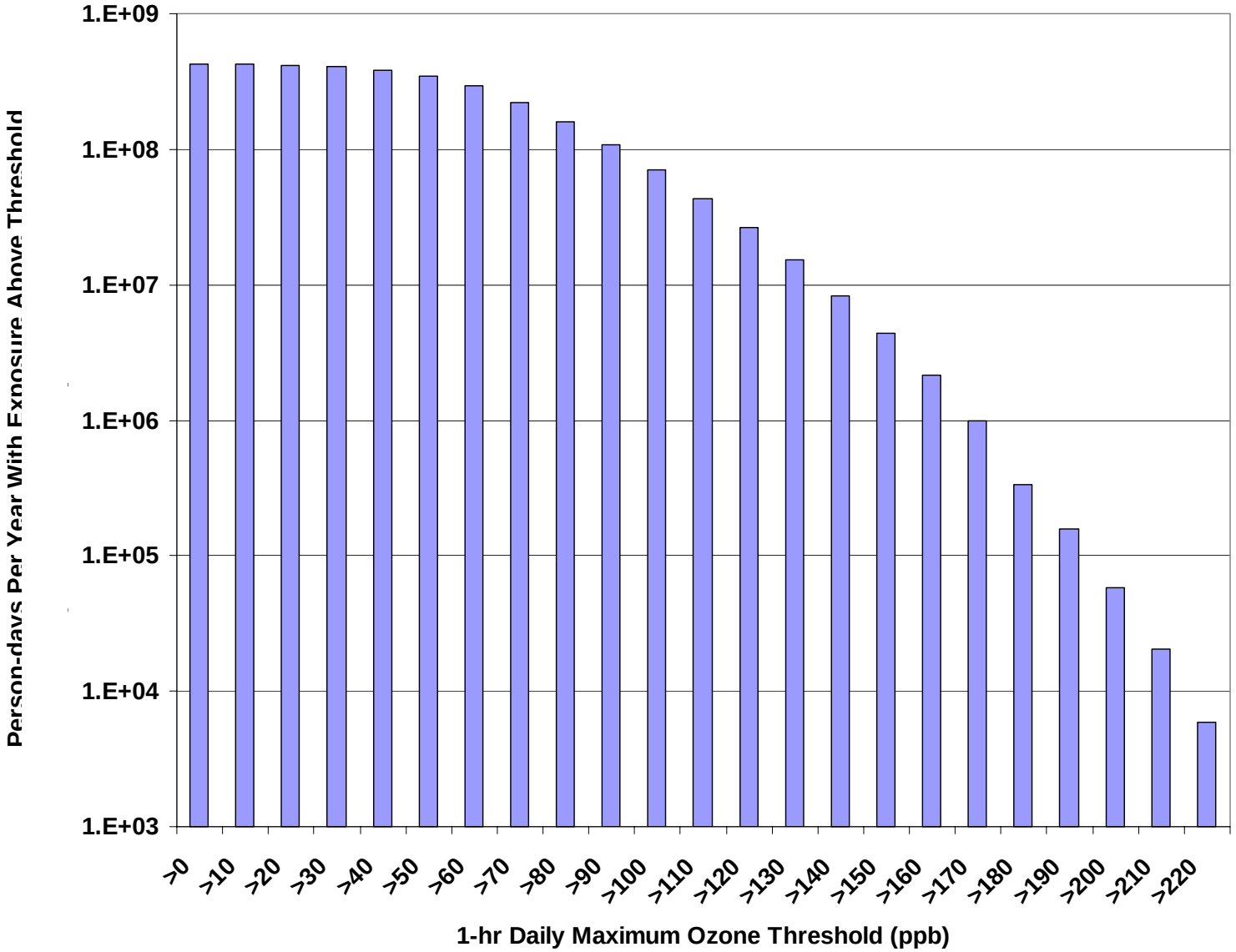

Figure 4-5. Estimated exposures of Houston residents to 1-hr daily maximum ozone in 1993-1995 under the "all outdoors" assumption.

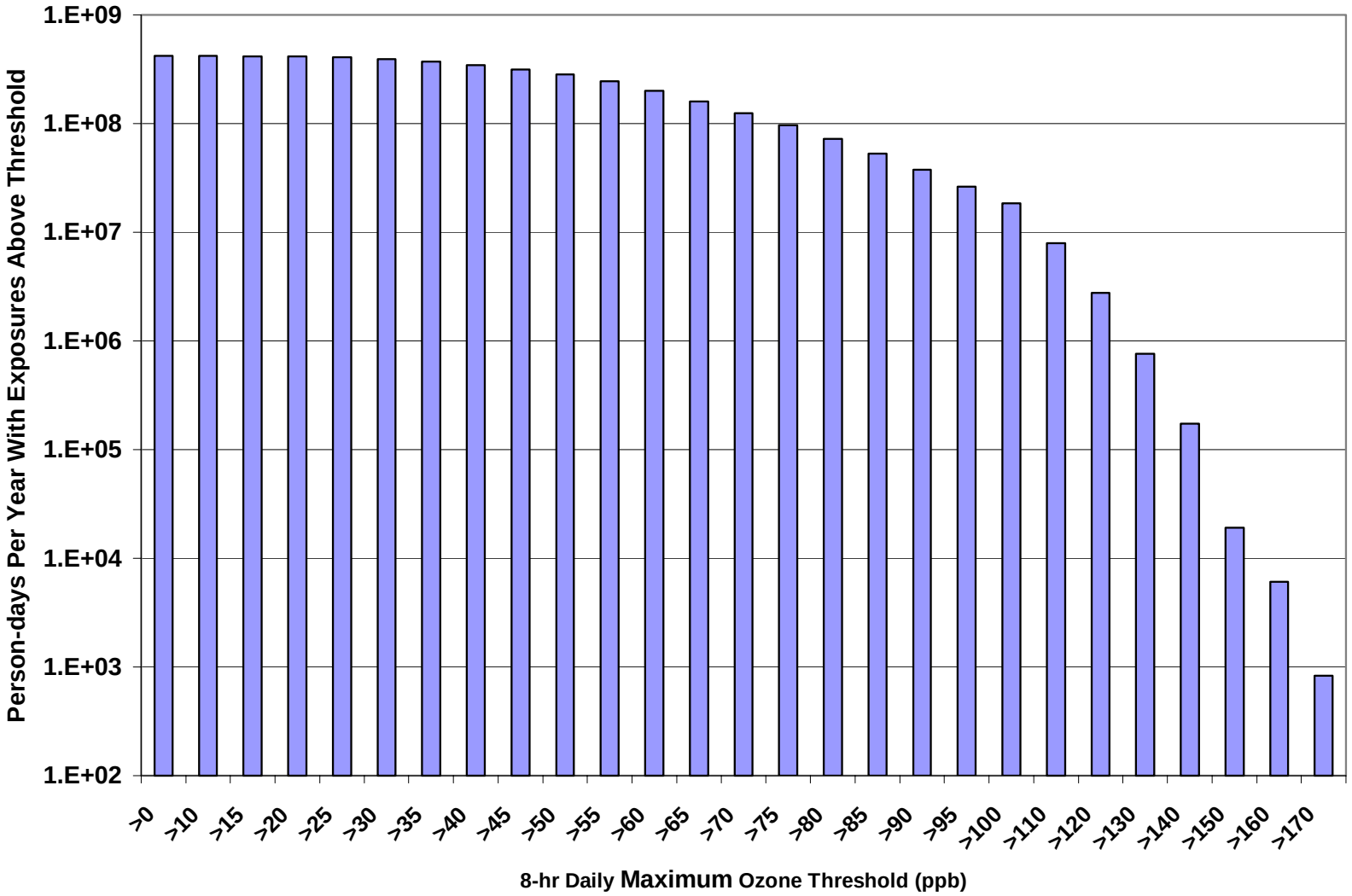

Figure 4-6. Estimated exposures of Houston residents to 8-hr daily maximum ozone in 1993-1995 under the "all outdoors" assumption.

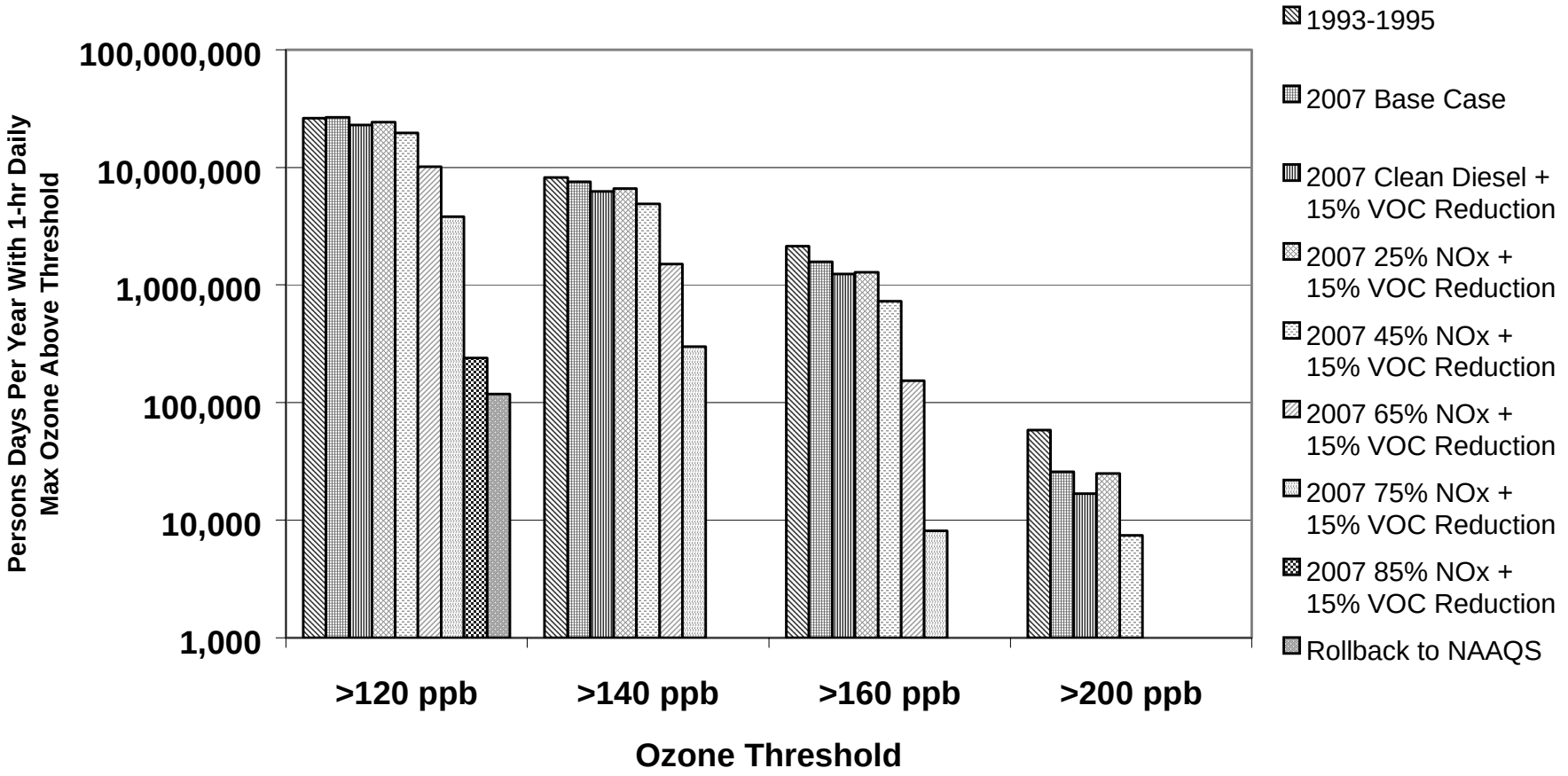

Figure 4-7. Distribution of "all outdoors" 1-hr daily maximum exposures.

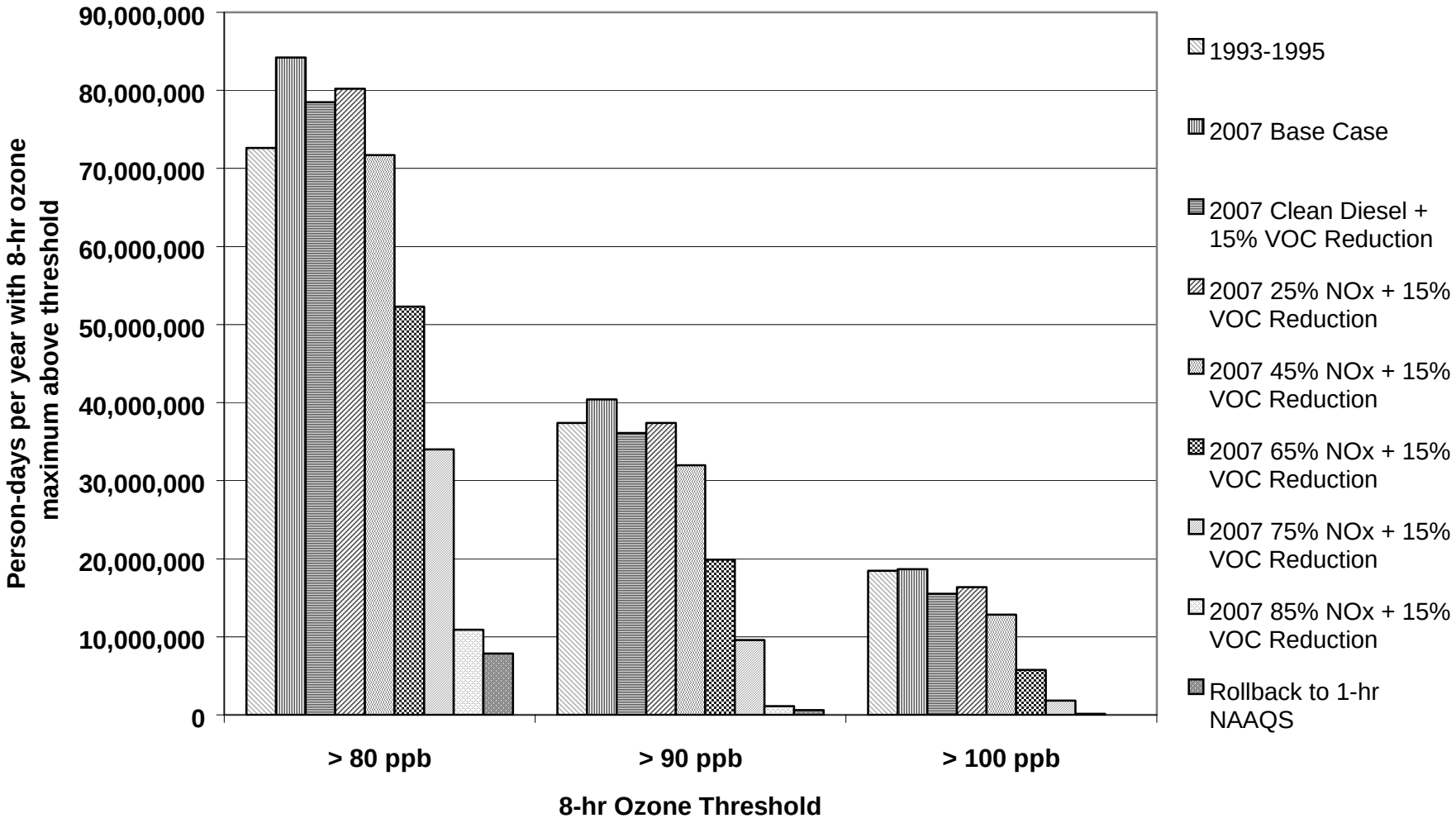

Figure 4-8. Distribution of "all outdoors" 8-hr daily maximum exposures.

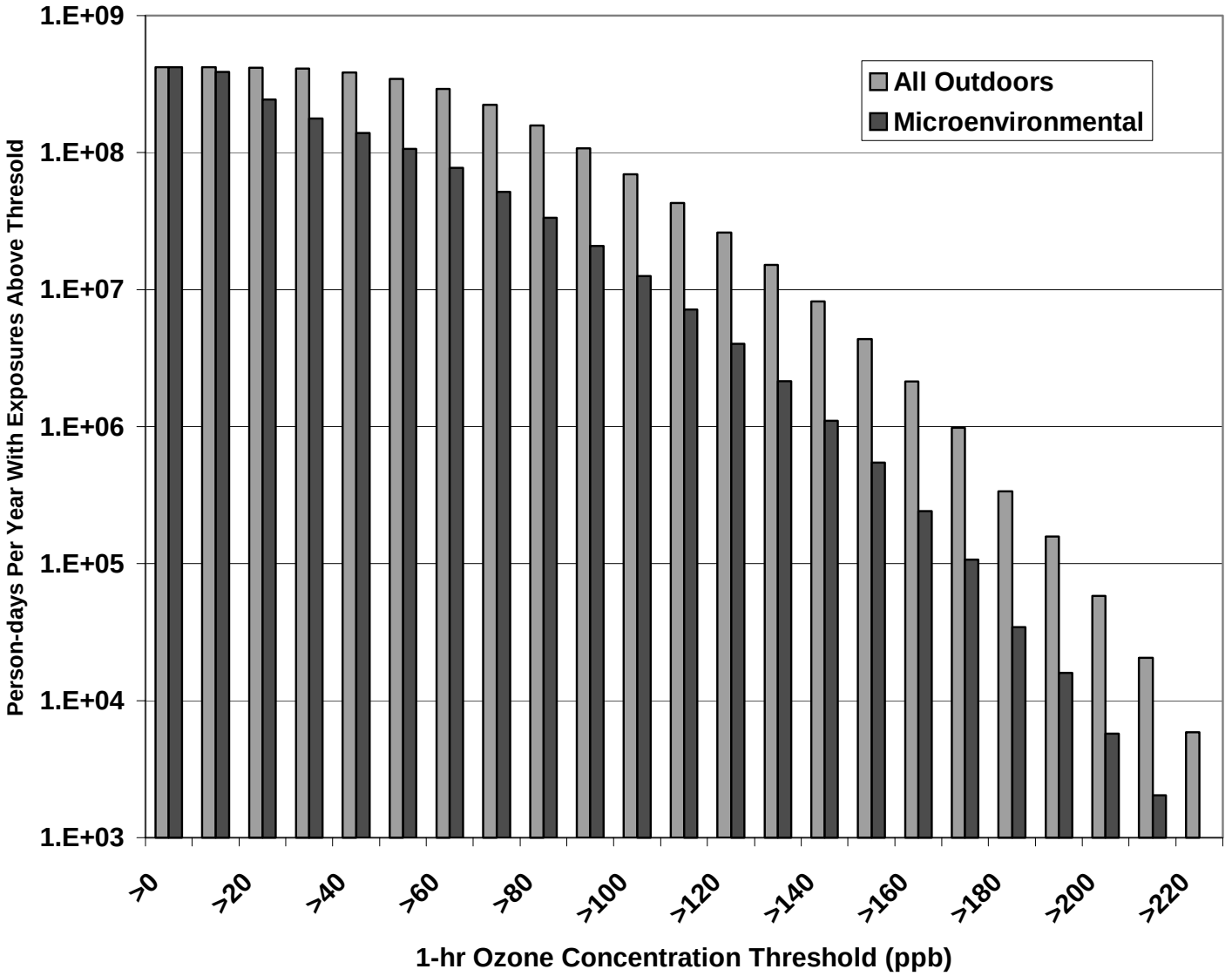

Figure 4-9. Comparison of Houston microenvironmental and "all outdoors" exposure estimates for 1-hr maximum daily exposures in 1993-1995.

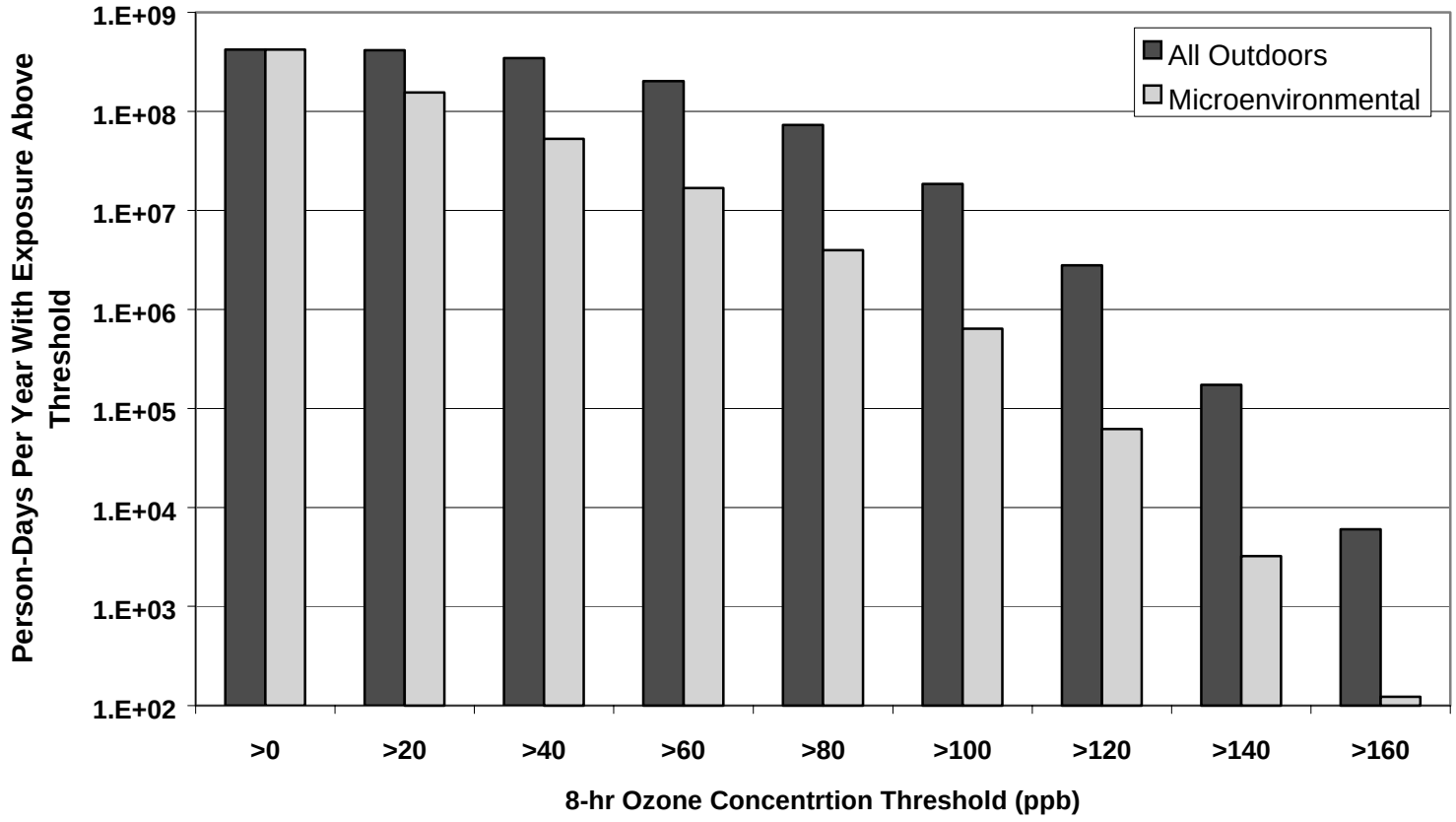

Figure 4-10. Comparison of Houston microenvironmental and "all outdoors" 8-hr maximum exposure estimates for 1993-1995.

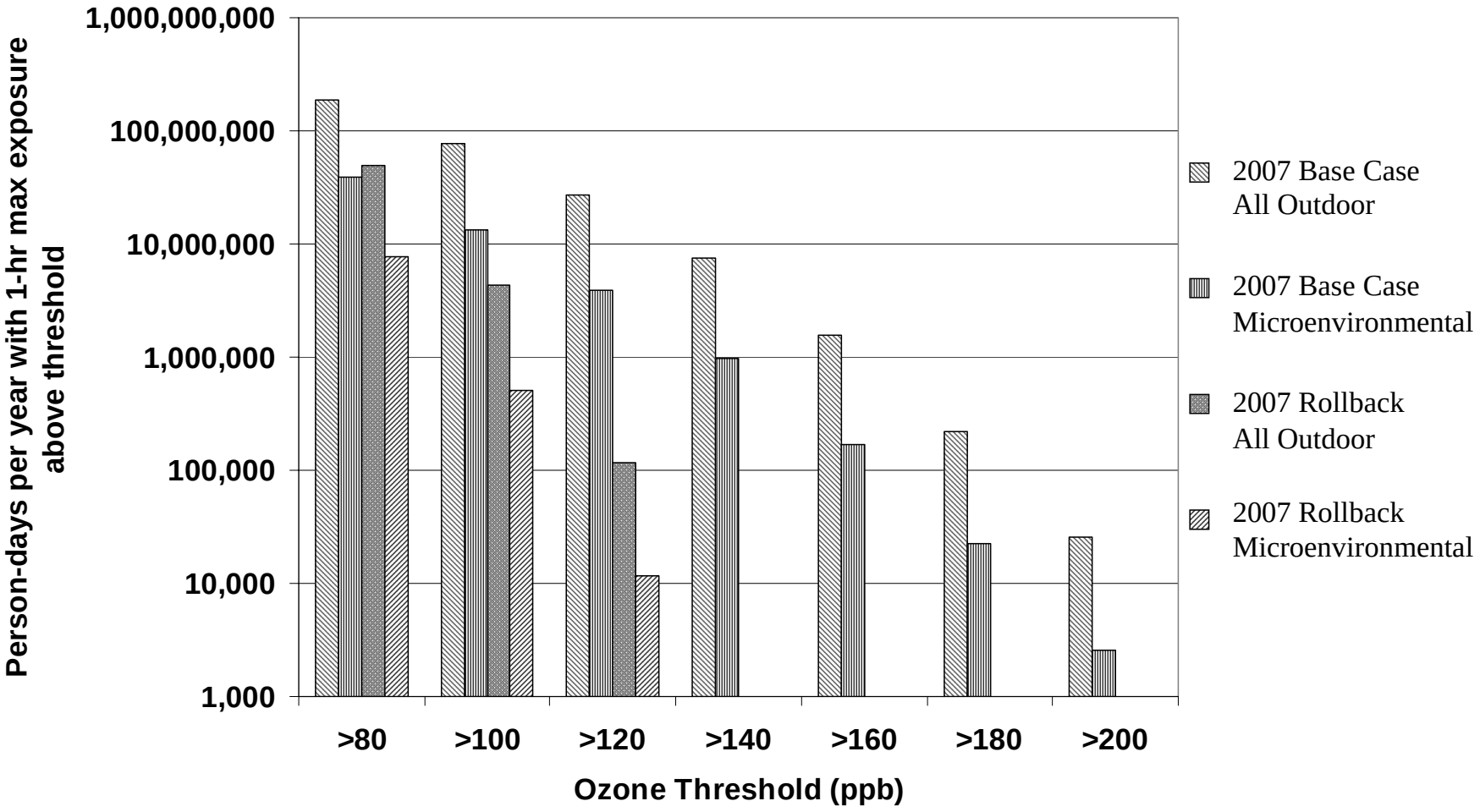

Figure 4-11. Comparison of microenvironmental and "all outdoors" 1-hr maximum exposure estimated in 2007.

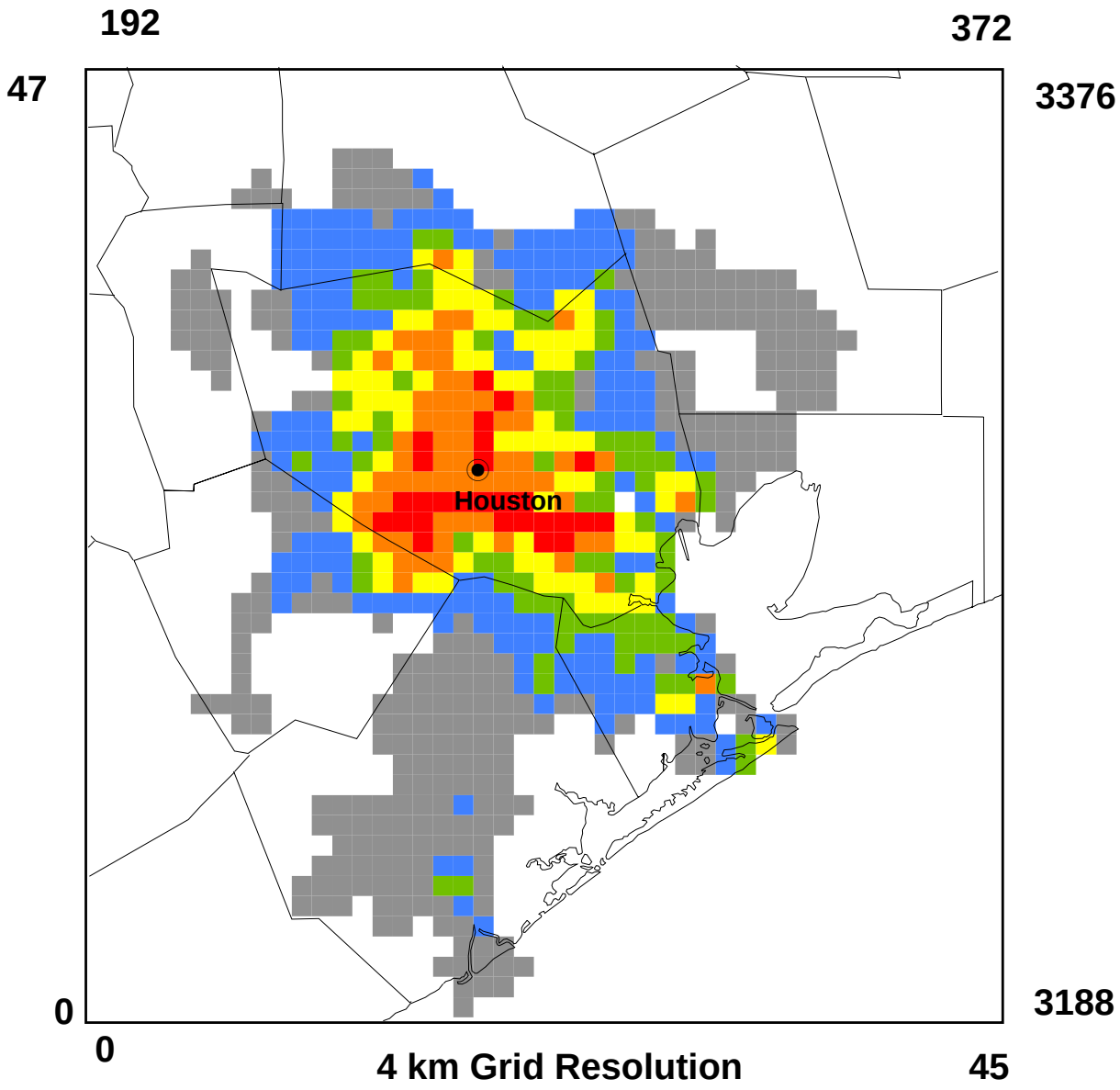

Figure 4-12. Spatial distribution of person-days per year of exposures to 1-hr maximum ozone concentrations above 120 ppb in 1993-1995 with the "all outdoors" approach.

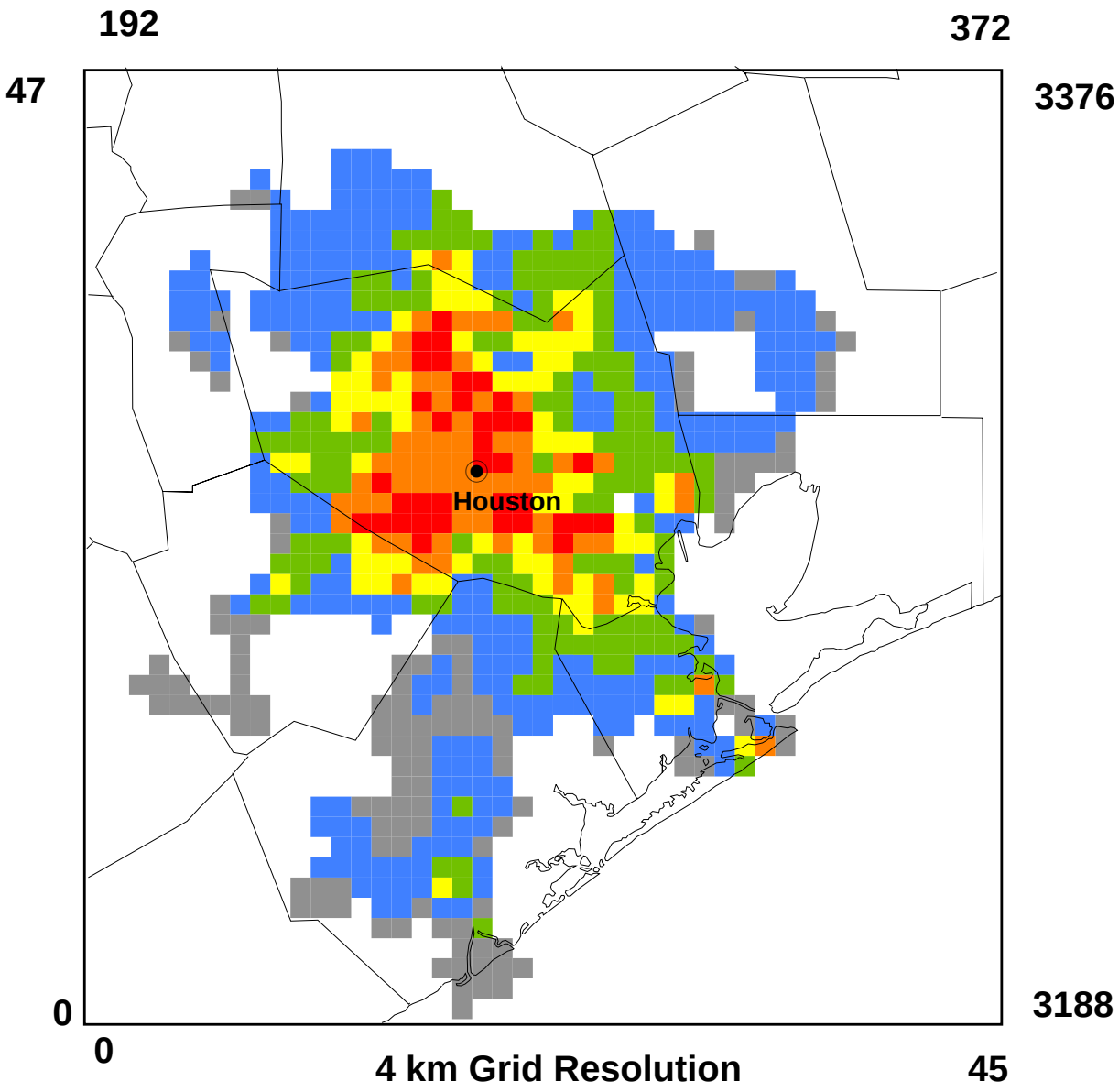

Figure 4-13. Spatial distribution of person-days per year of exposures to 8-hr maximum ozone concentrations above 80 ppb in 1993-1995 with the "all outdoors" approach.

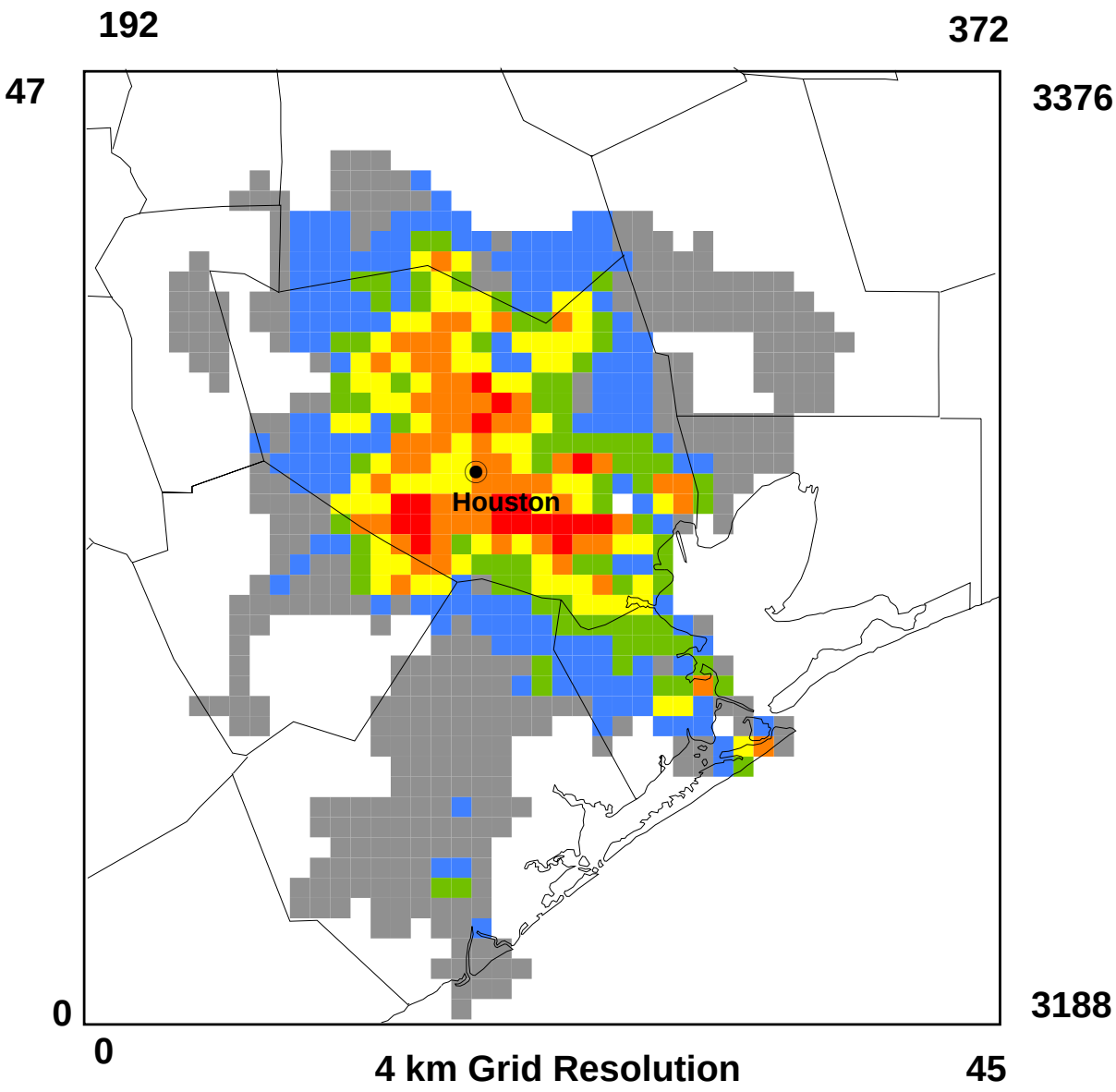

Figure 4-14. Spatial distribution of person-days per year of exposures to 1-hr maximum ozone concentrations above 120 ppb in 1993-1995 with the microenvironmental approach.

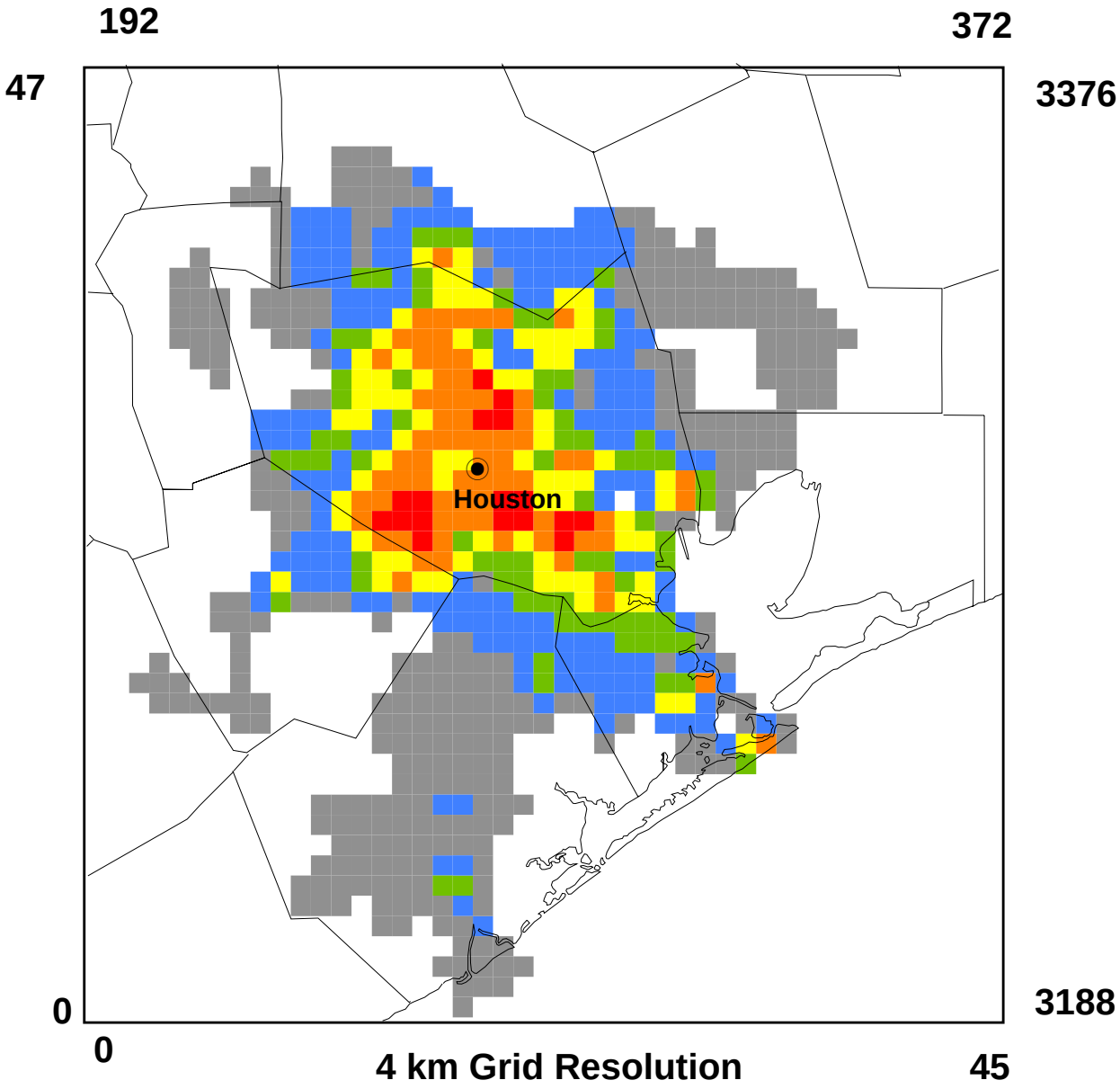

Figure 4-15. Spatial distribution of person-days per year of exposures to 8-hr maximum ozone concentrations above 80 ppb in 1993-1995 with the microenvironmental approach.

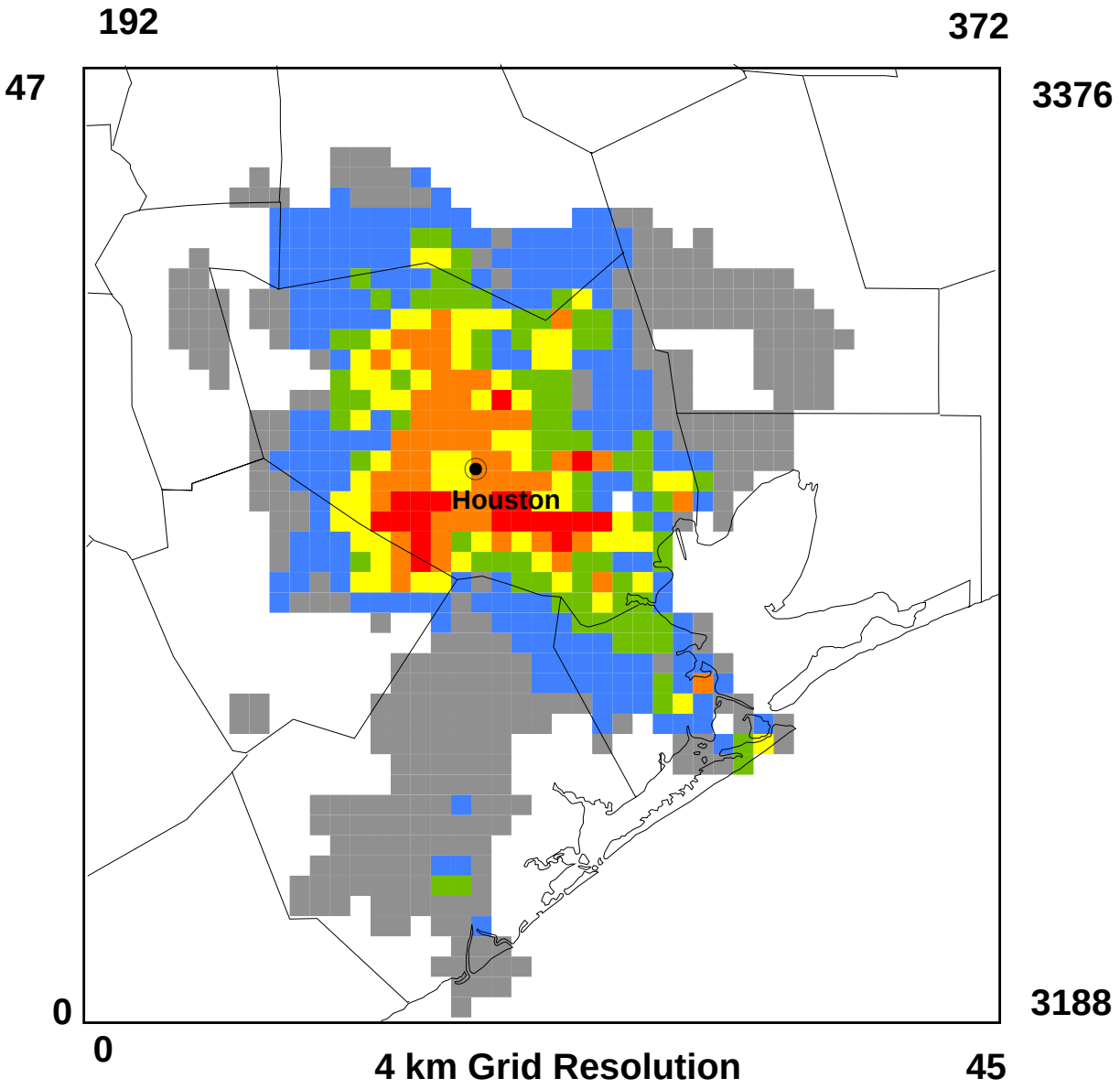

Figure 4-16. Spatial distribution of person-days per year of exposures to 1-hr maximum ozone concentrations above 120 ppb in the 2007 base case with the "all outdoors" approach.

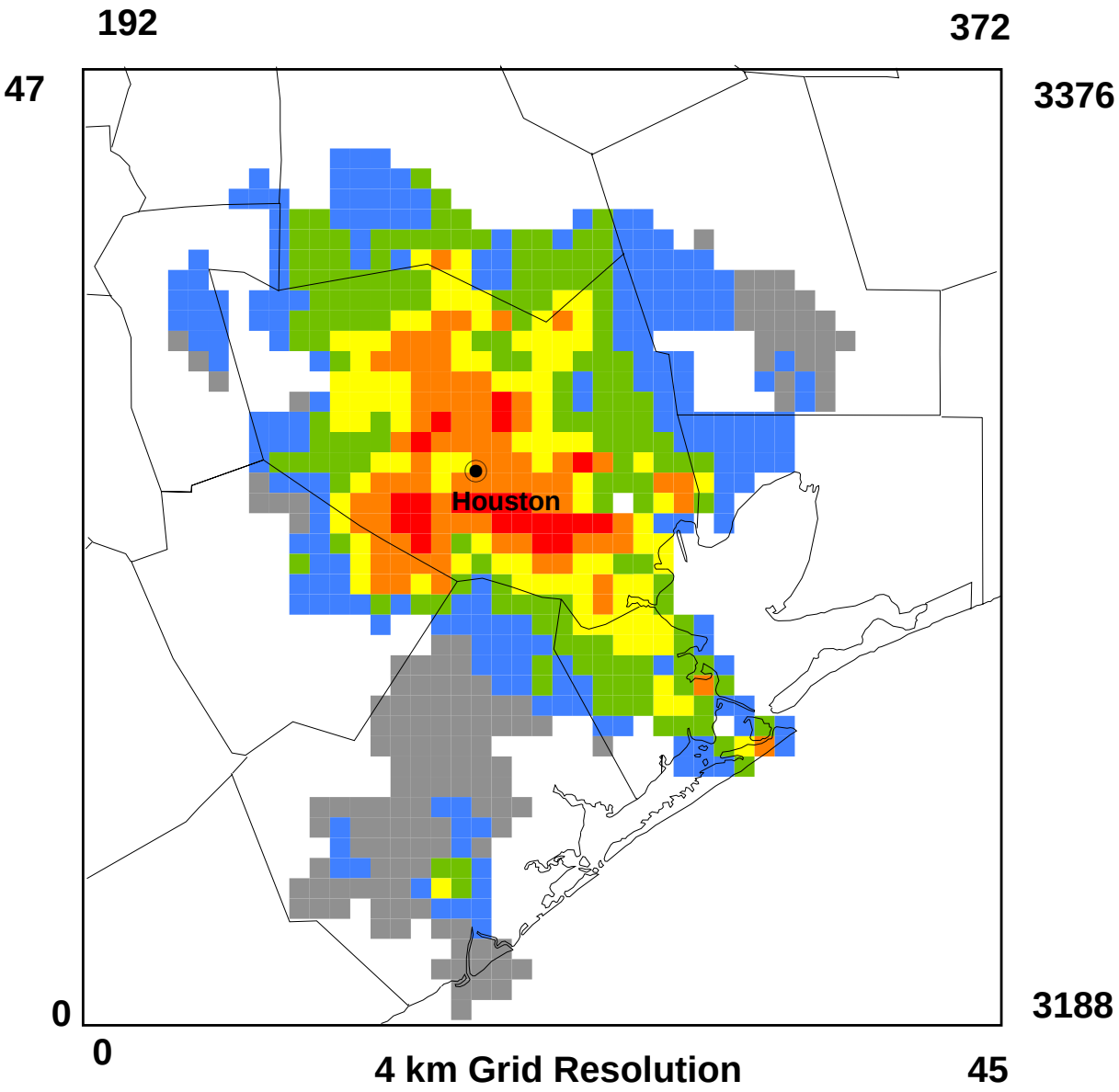

Figure 4-17. Spatial distribution of person-days per year of exposures to 1-hr maximum ozone concentrations above 120 ppb in the 2007 base case with 45 percent NO_x and 15 percent VOC emissions reductions with the "all outdoors" approach.

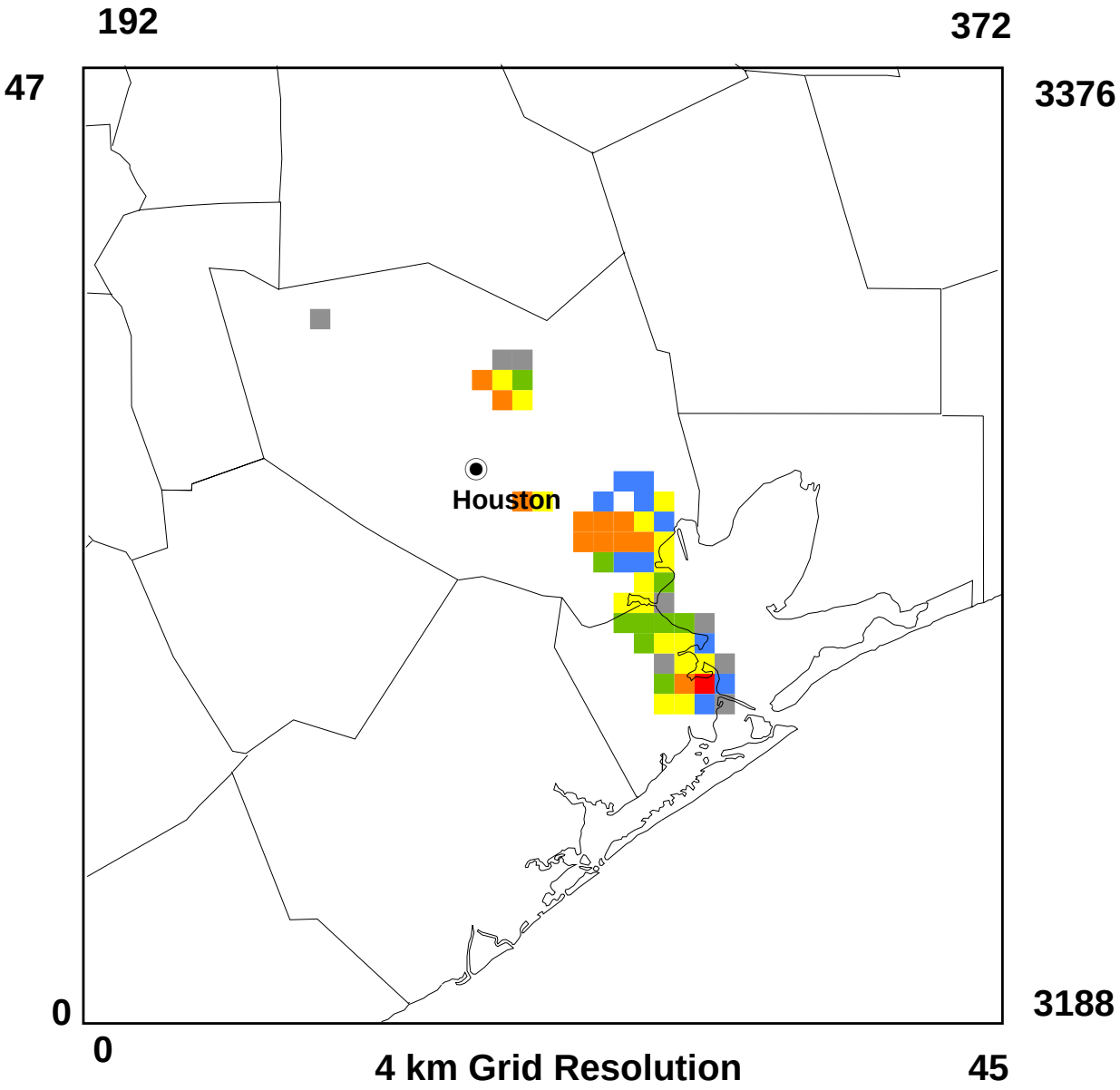

Figure 4-18. Spatial distribution of person-days per year of exposures to 1-hr maximum ozone concentrations above 120 ppb in the 2007 base case with 85 percent NO_x and 15 percent VOC emissions reductions with the "all outdoors" approach.

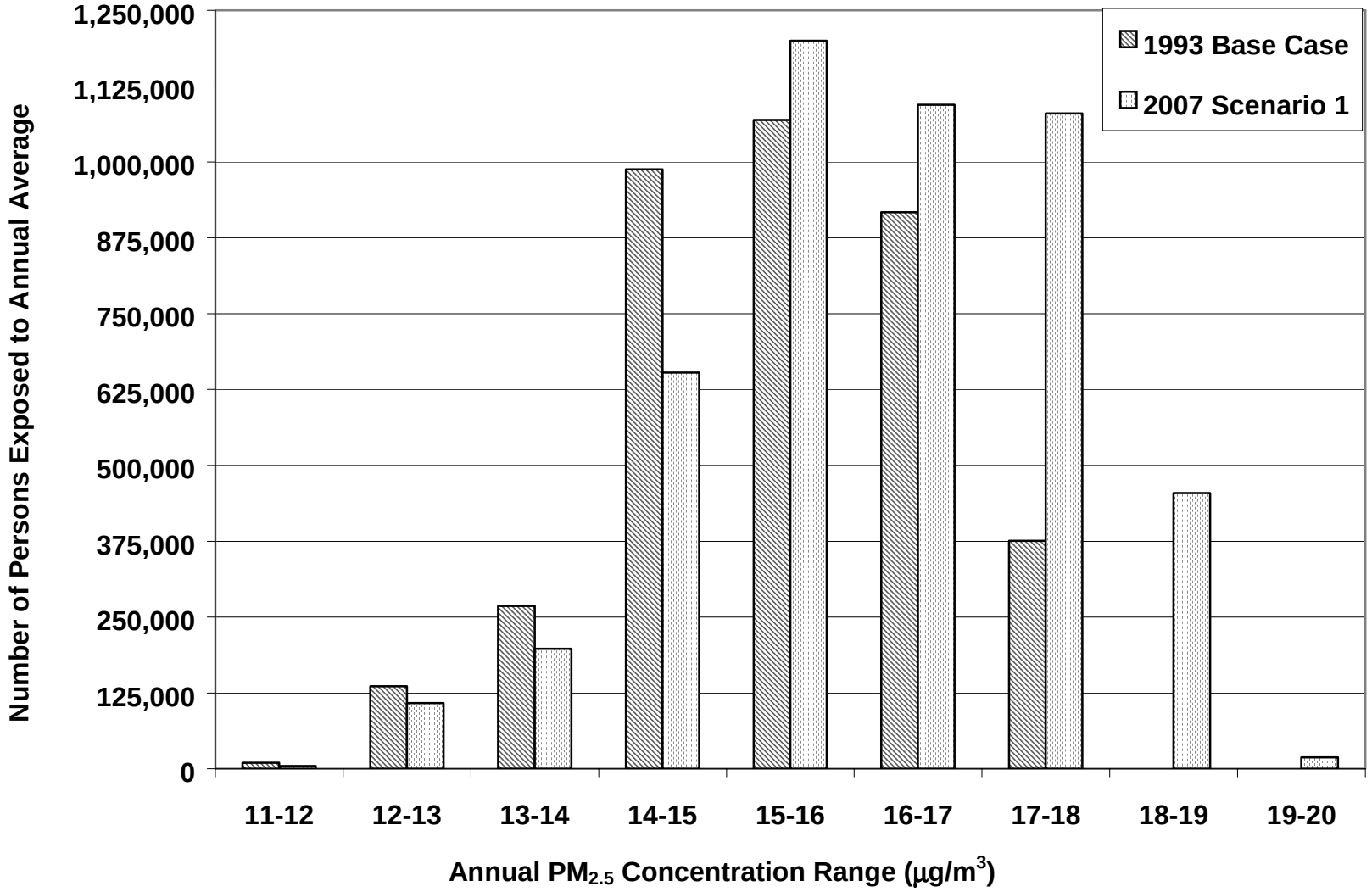

Figure 4-19. Estimated distribution of annual average $PM_{2.5}$ exposures in 1993 and 2007 (under Scenario 1)

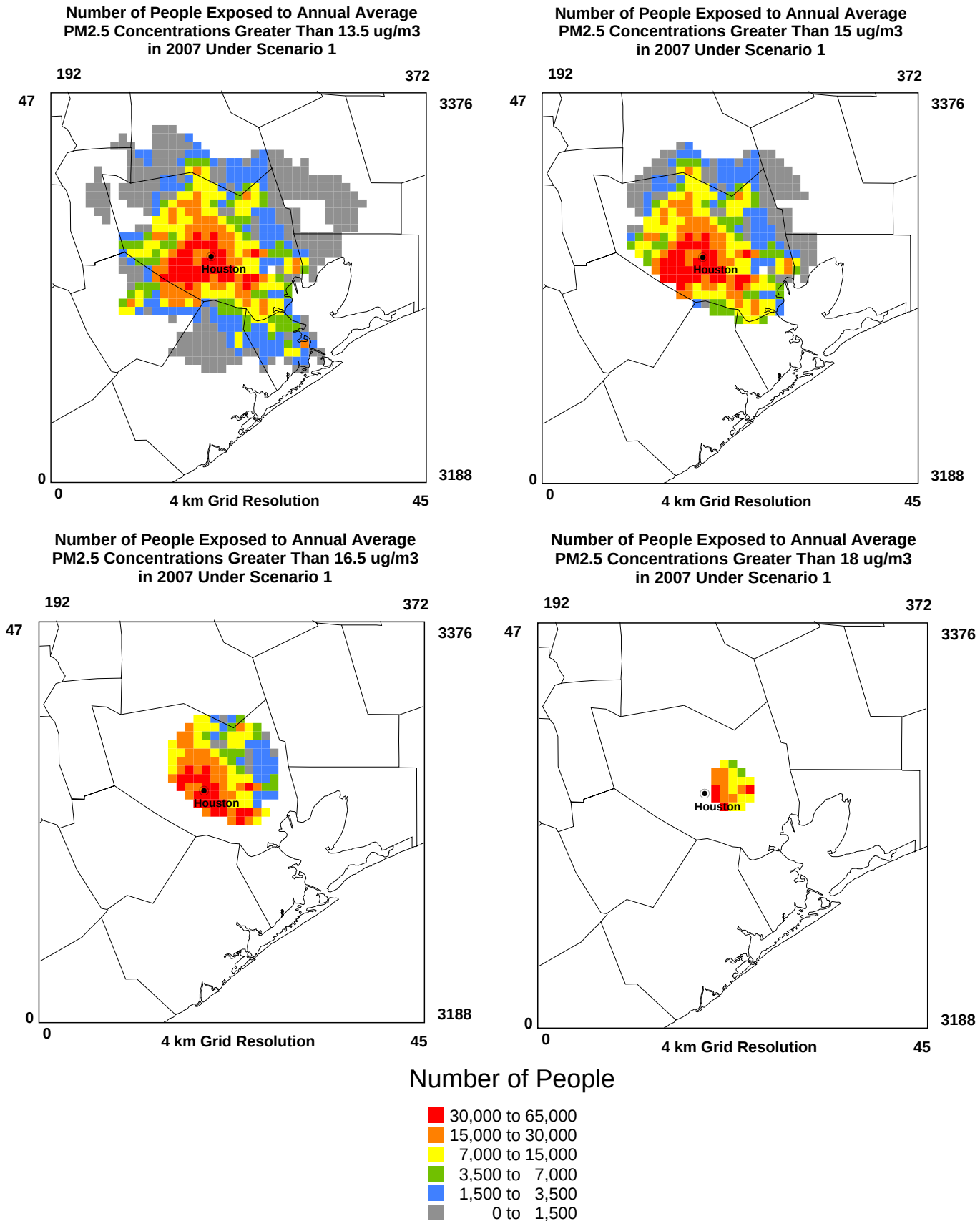

Figure 4-20. Estimated number of people exposed to various annual average $PM_{2.5}$ concentration thresholds in Houston in 2007 under Scenario 1.

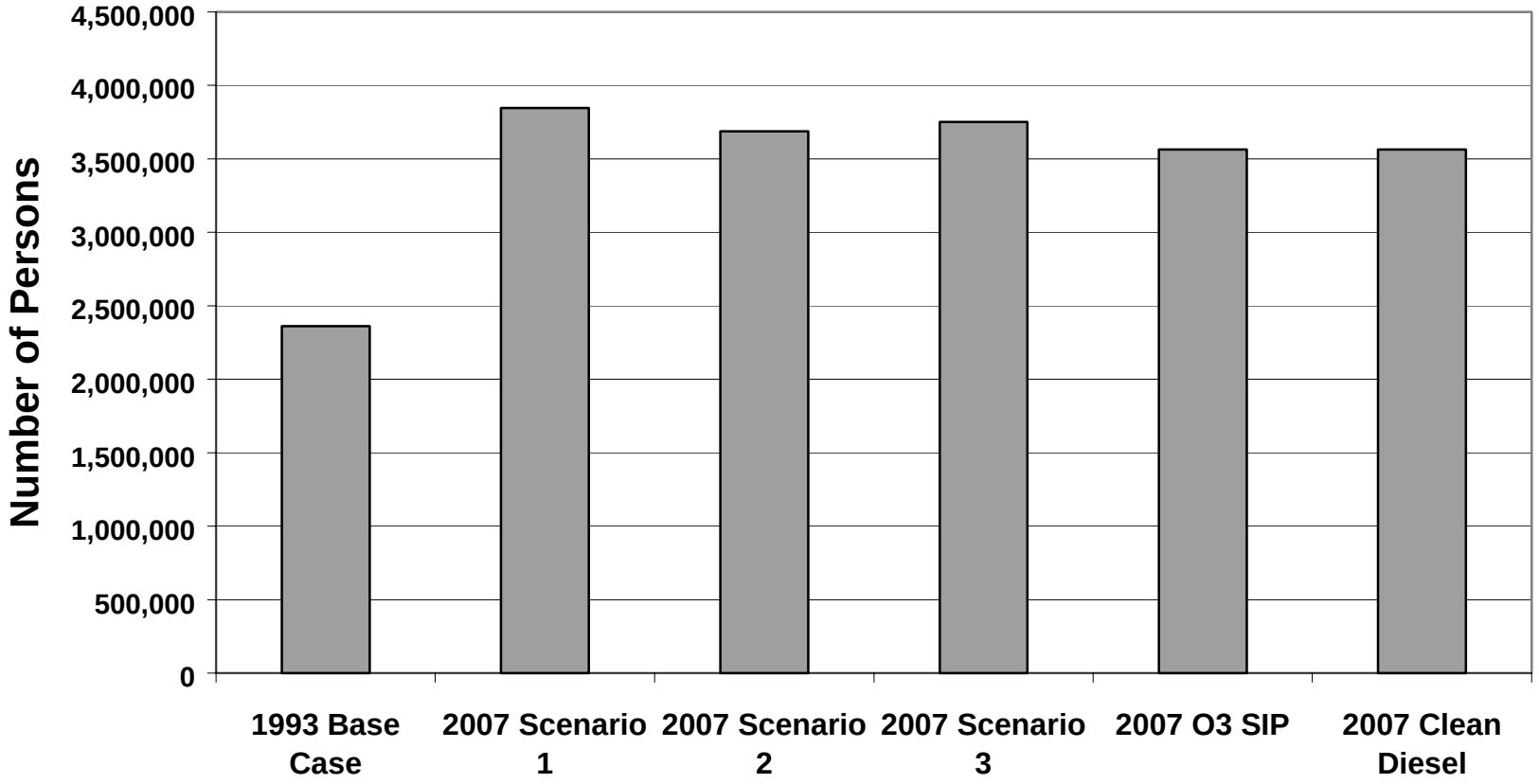

Figure 4-21. Number of people exposed to annual average $PM_{2.5}$ greater than 15 $\mu g/m^3$.

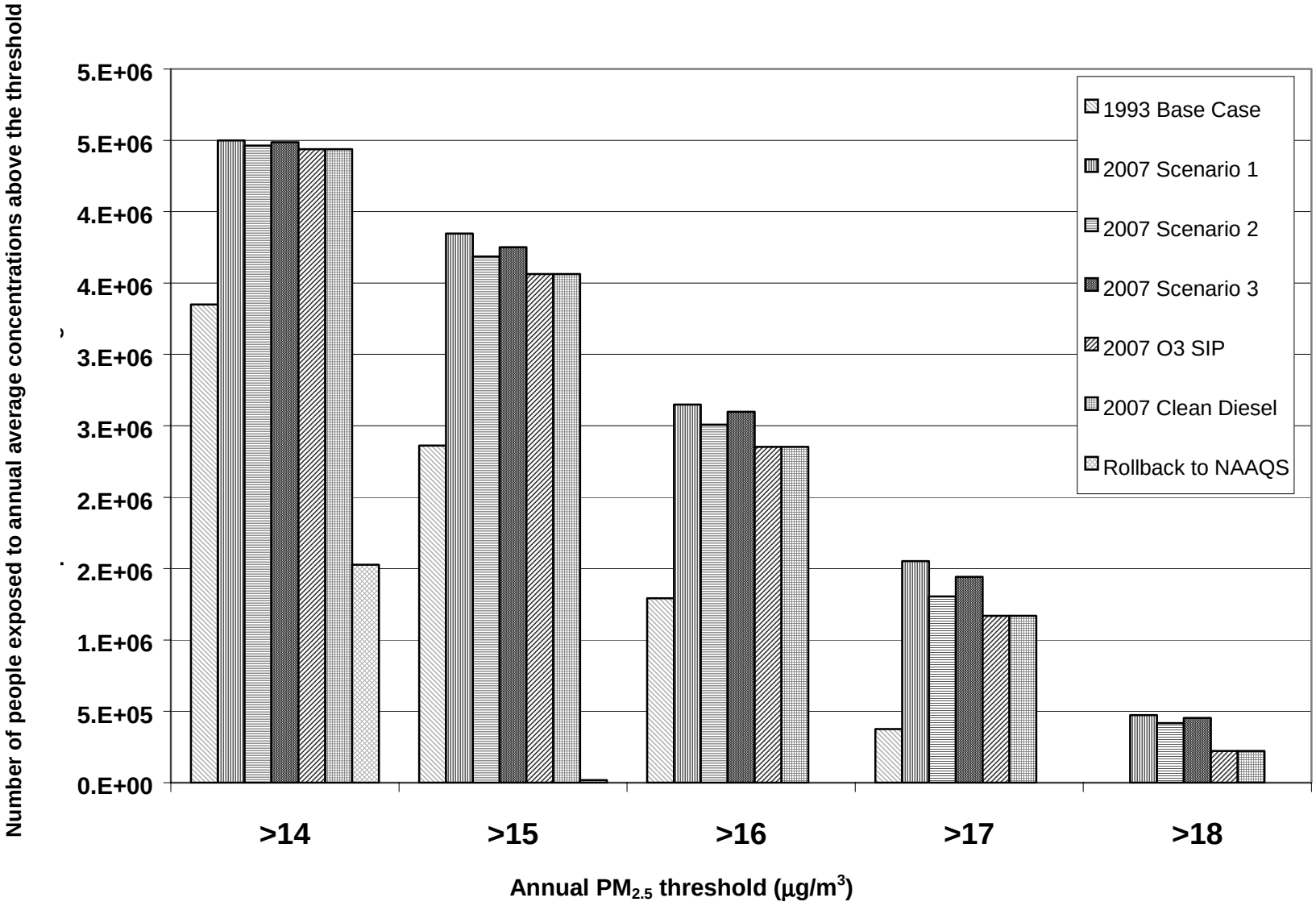

Figure 4-22. Number of people exposed to various annual average $PM_{2.5}$ concentrations in Houston.

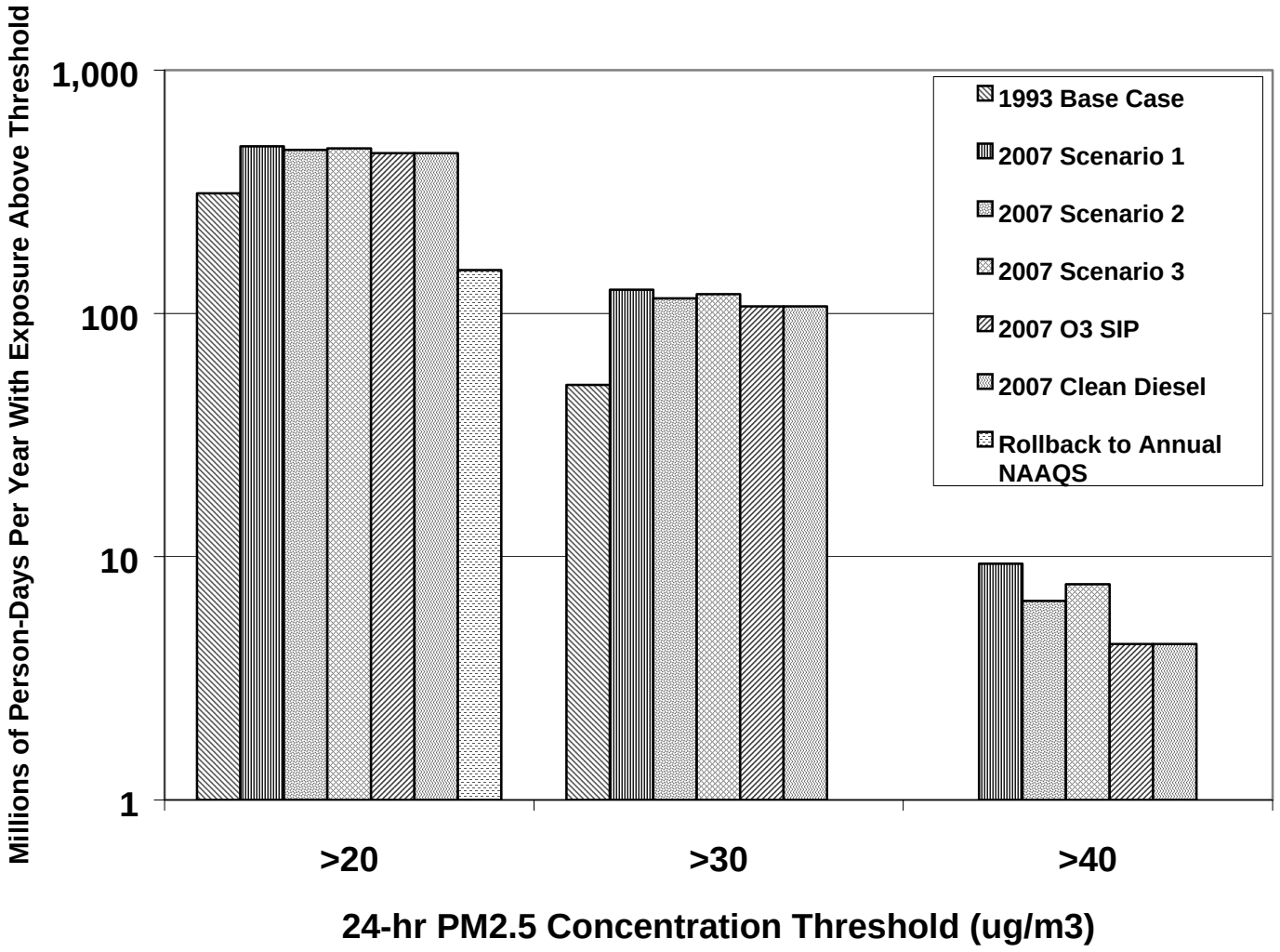

Figure 4-23. Estimated daily exposures to $PM_{2.5}$ in Houston.

Table 4-1. Houston area population in 1993 and 2007 by demographic group.

Demographic Group	1993	2007
Males – Ages 0-4 years	166,012	185,402
Males – Ages 5-19 years	440,702	539,594
Males – Ages 20-64 years	1,155,655	1,513,872
Males – Ages >64 years	108,530	148,569
Females – Ages 0-4 years	159,389	178,425
Females – Ages 5-19 years	419,591	519,008
Females – Ages 20-64 years	1,155,581	1,523,685
Females – Ages >64 years	158,712	201,954
Total	3,764,172	4,810,509

Table 4-2. Categories of locations and activities in the time-activity database.

Locations	Activities
Residential – Indoor	Sports/Exercise
Residential – Outdoor	Yard work/Maintenance
In Vehicle	Eating/Drinking
Near Vehicle(s)	Bathing
Other Outdoor	Housekeeping
Office/Factory	Laundry/Dishes
Mall/Store	Food Preparation
School/Church/Public Building	Other Activities
Bar/Restaurant	
Other Indoor	

Table 4-3. Mean percentage of time spent outdoors based on NHAPS data for the 48 contiguous states. Number of samples are shown in parentheses.

Day Type	Males					Females					Both				
	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages
Summer Weekday	9.3 (47)	14.2 (126)	12.8 (472)	11.1 (92)	12.7 (737)	11.1 (38)	12.4 (137)	5.8 (555)	5.8 (133)	7.7 (863)	10.1 (85)	13.3 (263)	9.0 (1027)	7.9 (225)	10.0 (1600)
Summer Weekend	8.6 (28)	14.6 (64)	13.8 (206)	13.3 (38)	13.5 (336)	10.8 (17)	14.9 (65)	9.1 (259)	5.5 (60)	10.2 (401)	9.5 (45)	14.8 (129)	11.2 (465)	8.5 (98)	11.7 (737)
Summer All	9.1 (75)	14.3 (190)	13.1 (678)	11.7 (130)	12.9 (1073)	11 (55)	13.1 (202)	6.7 (814)	5.7 (193)	8.4 (1264)	9.9 (130)	13.7 (392)	9.6 (1492)	8.1 (323)	10.5 (2337)
Non-Summer Weekday	4.3 (118)	5.9 (375)	8.0 (1336)	8.2 (213)	7.2 (2042)	3.6 (115)	4.4 (329)	3.2 (1485)	2.9 (406)	3.5 (2335)	4.0 (233)	5.2 (704)	5.5 (2821)	4.7 (619)	5.2 (4377)
Non-Summer Weekend	7.8 (72)	11.4 (167)	8.4 (637)	8.1 (117)	9.0 (993)	3.7 (62)	6.9 (164)	5.1 (735)	2.6 (233)	5.2 (1194)	5.9 (134)	9.2 (331)	6.6 (1372)	4.5 (350)	7.0 (2187)
Non-Summer All	5.3 (190)	7.5 (542)	8.1 (1973)	8.2 (330)	7.7 (3035)	3.6 (177)	5.1 (493)	3.7 (2220)	2.8 (639)	4.0 (3529)	4.5 (367)	6.3 (1035)	5.8 (4193)	4.6 (969)	5.7 (6564)
All Days	6.3 (265)	9.2 (732)	9.4 (2651)	9.1 (460)	9.0 (4108)	5.5 (232)	7.1 (695)	4.5 (3034)	3.5 (832)	5.1 (4793)	5.9 (497)	8.2 (1427)	6.8 (5685)	5.5 (1292)	6.9 (8901)

Table 4-4. Mean percentage of time spent outdoors based on NHAPS data for the southern states. Number of samples are shown in parentheses.

Day Type	Males					Females					Both				
	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages
Summer Weekday	4.8 (18)	14.6 (40)	12.5 (182)	8.8 (27)	12.1 (267)	12.6 (8)	11.2 (50)	5.2 (215)	6.5 (43)	7.3 (316)	7.2 (26)	12.7 (90)	8.5 (397)	7.4 (70)	9.3 (583)
Summer Weekend	6.1 (11)	11.7 (22)	15.1 (74)	11.4 (14)	13.3 (121)	8.5 (6)	9.2 (25)	8.2 (105)	8.5 (20)	8.5 (156)	6.9 (17)	10.4 (47)	11.1 (179)	9.7 (34)	10.5 (277)
Summer All	5.2 (29)	13.8 (62)	13.2 (256)	9.5 (41)	12.4 (388)	11.4 (14)	10.6 (75)	6.1 (320)	7.1 (63)	7.6 (472)	7.1 (43)	12.0 (137)	9.2 (576)	8.1 (104)	9.6 (860)
Non-Summer Weekday	3.7 (43)	5.8 (142)	9.0 (443)	6.7 (73)	7.6 (701)	5.0 (40)	4.5 (125)	2.6 (494)	3.0 (151)	3.3 (810)	4.3 (83)	5.2 (267)	5.6 (937)	4.2 (224)	5.3 (1511)
Non-Summer Weekend	13.5 (25)	12.1 (64)	8.1 (194)	9.1 (35)	9.6 (318)	4.1 (18)	5.6 (58)	4.3 (252)	3.2 (89)	4.5 (417)	9.5 (43)	9 (122)	6 (446)	4.8 (124)	6.9 (735)
Non-Summer All	6.5 (68)	7.6 (206)	8.7 (637)	7.4 (108)	8.2 (1019)	4.7 (58)	4.8 (183)	3.1 (746)	3.1 (240)	3.6 (1227)	5.8 (126)	6.3 (389)	5.7 (1383)	4.4 (348)	5.8 (2246)
All Days	6.2 (97)	9.1 (268)	9.9 (893)	7.9 (149)	9.3 (1407)	6.4 (72)	6.3 (258)	3.8 (1066)	4.1 (303)	4.6 (1699)	6.1 (169)	7.7 (526)	6.6 (1959)	5.3 (452)	6.7 (3106)

Table 4-5. Mean percentage of time spent outdoors based on NHAPS data for Texas. Number of samples are shown in parentheses.

Day Type	Males					Females					Both				
	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages
Summer Weekday	0 (1)	10.5 (8)	13.2 (39)	1 (3)	10.7 (51)	3.5 (1)	14.4 (14)	2.7 (31)	12.3 (5)	6.2 (51)	1.7 (2)	13 (22)	8.6 (70)	8.1 (8)	9.0 (102)
Summer Weekend	12.2 (4)	10.7 (9)	11.5 (10)	19.4 (2)	11.8 (25)	0 (0)	10.8 (4)	7.5 (13)	16.9 (5)	8.4 (22)	12.2 (4)	10.7 (13)	9.3 (23)	17.6 (7)	10.5 (47)
Summer All	3.5 (5)	10.6 (17)	12.7 (49)	6.3 (5)	11.0 (76)	2.5 (1)	13.4 (18)	4.1 (44)	13.6 (10)	6.8 (73)	4.7 (6)	12.3 (35)	8.8 (93)	10.8 (15)	9.4 (149)
Non-Summer Weekday	5.3 (8)	4.7 (29)	5.6 (73)	8.2 (10)	5.5 (120)	2.9 (11)	4.8 (28)	2.9 (79)	4.4 (27)	3.4 (145)	3.9 (19)	4.8 (57)	4.2 (152)	5.5 (37)	4.4 (265)
Non-Summer Weekend	14.2 (7)	11.9 (14)	7.7 (31)	6.9 (7)	9.2 (59)	5.7 (4)	12.4 (8)	4.7 (44)	2.2 (7)	6.3 (63)	11.1 (11)	12 (22)	5.9 (75)	4.6 (14)	7.7 (122)
Non-Summer All	7.8 (15)	6.8 (43)	6.2 (104)	7.8 (17)	6.6 (179)	3.7 (15)	7.0 (36)	3.4 (123)	3.8 (34)	4.3 (208)	6.0 (30)	6.9 (79)	4.7 (227)	5.2 (51)	5.3 (387)
All Days	6.8 (20)	7.7 (60)	7.8 (153)	7.4 (22)	7.7 (255)	3.4 (16)	8.6 (54)	3.6 (167)	6.2 (44)	4.9 (281)	5.6 (36)	8.2 (114)	5.7 (320)	6.6 (66)	6.3 (536)

Table 4-6. Mean percentage of time spent outdoors based on NHAPS data for the northern, eastern, and western states. Number of samples are shown in parentheses.

Day Type	Males					Females					Both				
	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages
Summer Weekday	12 (29)	14 (86)	13 (290)	12.1 (65)	13.1 (470)	10.7 (30)	13.2 (87)	6.2 (340)	5.4 (90)	8.1 (547)	11.3 (59)	13.6 (173)	9.4 (630)	8.2 (155)	10.4 (1017)
Summer Weekend	10.3 (17)	16.1 (42)	13 (132)	14.4 (24)	13.6 (215)	12.1 (11)	18.5 (40)	9.7 (154)	4.0 (40)	11.4 (245)	11 (28)	17.3 (82)	11.2 (286)	7.9 (64)	12.3 (460)
Summer All	11.5 (46)	14.6 (128)	13 (422)	12.8 (89)	13.2 (685)	11.1 (41)	14.7 (127)	7.2 (494)	5.0 (130)	9.0 (792)	11.2 (87)	14.7 (255)	9.9 (916)	8.1 (219)	11.0 (1477)
Non-Summer Weekday	4.7 (75)	5.9 (233)	7.5 (893)	8.9 (140)	6.9 (1341)	2.8 (75)	4.4 (204)	3.5 (991)	2.8 (255)	3.6 (1525)	3.8 (150)	5.2 (437)	5.4 (1884)	5.0 (395)	5.2 (2866)
Non-Summer Weekend	4.8 (47)	11 (103)	8.5 (443)	7.7 (82)	8.7 (675)	3.6 (44)	7.6 (106)	5.5 (483)	2.3 (144)	5.5 (777)	4.2 (91)	9.3 (209)	7.0 (926)	4.3 (226)	7.1 (1452)
Non-Summer All	4.7 (122)	7.4 (336)	7.8 (1336)	8.6 (222)	7.5 (2016)	3.0 (119)	5.3 (310)	4.1 (1474)	2.7 (399)	4.1 (2302)	3.9 (241)	6.4 (646)	5.9 (2810)	4.8 (621)	5.7 (4318)
All Days	6.4 (168)	9.2 (464)	9.1 (1758)	9.6 (311)	8.9 (2701)	5.0 (160)	7.7 (437)	4.9 (1968)	3.2 (529)	5.4 (3094)	5.7 (328)	8.4 (901)	6.9 (3726)	5.6 (840)	7.0 (5795)

Table 4-7. Mean percentage of time spent outdoors based on NHAPS data used in the Houston exposure modeling. Number of samples are shown in parentheses.

Day Type	Males					Females					Both				
	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages	0-4 years	5-19 years	20-64 years	65 + years	All Ages
Summer Weekday	9.3 (47)	14.2 (126)	12.5 (182)	11.1 (92)	12.5 (447)	11.1 (38)	12.4 (137)	5.2 (215)	5.8 (133)	7.4 (523)	10.1 (85)	13.3 (263)	8.5 (397)	7.9 (225)	9.7 (970)
Summer Weekend	8.6 (28)	14.6 (64)	15.1 (74)	13.3 (38)	14.3 (204)	10.8 (17)	14.9 (65)	8.2 (105)	5.5 (60)	9.7 (247)	9.5 (45)	14.8 (129)	11.1 (179)	8.5 (98)	11.6 (451)
Summer All	9.1 (75)	14.3 (190)	13.2 (256)	11.7 (130)	13.0 (651)	11.0 (55)	13.1 (202)	6.1 (320)	5.7 (193)	8.0 (770)	9.9 (130)	13.7 (392)	9.2 (576)	8.1 (323)	10.2 (1421)
Non-Summer Weekday	3.7 (43)	5.8 (142)	9.0 (443)	6.7 (73)	7.6 (701)	5.0 (40)	4.5 (125)	2.6 (494)	3.0 (151)	3.3 (810)	4.3 (83)	5.2 (267)	5.6 (937)	4.2 (224)	5.3 (1511)
Non-Summer Weekend	13.5 (25)	12.1 (64)	8.1 (194)	9.1 (35)	9.6 (318)	4.1 (18)	5.6 (58)	4.3 (252)	3.2 (89)	4.5 (417)	9.5 (43)	9.0 (122)	6.0 (446)	4.8 (124)	6.9 (735)
Non-Summer All	6.5 (68)	7.6 (206)	8.7 (637)	7.4 (108)	8.2 (1019)	4.7 (58)	4.8 (183)	3.1 (746)	3.1 (240)	3.6 (1227)	5.8 (126)	6.3 (389)	5.7 (1383)	4.4 (348)	5.8 (2246)
All Days	7.2 (143)	9.3 (396)	9.9 (893)	8.5 (238)	9.4 (1670)	6.3 (113)	6.9 (385)	3.8 (1066)	3.7 (433)	4.7 (1997)	6.8 (256)	8.1 (781)	6.6 (1959)	5.3 (671)	6.9 (3667)

Table 4-8. Indoor/outdoor ratios of ozone concentrations used in the microenvironmental ozone exposure simulations.

Microenvironment	Ozone I/O Ratio
Residential – Indoor[a]	0.2 for residents with 1990 household income > $17,500 0.7 for residents with 1990 household income < $17,500
Office/Factory	0.2
Mall/Store	0.2
School/Church/Public Building	0.2
Bar/Restaurant	0.2
Other Indoor	0.2
In Vehicle	0.2
Near Vehicle(s)	0.5
Residential – Outdoor	1.0
Other Outdoor	1.0

[a] The spatial variation in household income was accounted for in the simulations.

Table 4-9. The estimated number of person-days per year with 1-hr maximum daily ozone concentrations above various thresholds for 1993-1995 and for alternate 2007 scenarios under the "all outdoors" exposure assumption.

Case	Million person-days per year with 1-hr maximum daily ozone concentrations above thresholds						
	1-hr ozone threshold (ppb)						
	>200	>180	>160	>140	>120	>100	> 80
1993-1995	0.058	0.340	2.14	8.20	26.2	69.9	157
2007 Base Case	0.026	0.220	1.58	7.55	26.7	77.7	189
2007 25% NO_x + 15% VOC Reduction	0.025	0.179	1.29	6.61	24.2	73.0	184
2007 Clean Diesel + 15% VOC Reduction	0.017	0.162	1.23	6.25	22.9	70.7	181
2007 45% NO_x + 15% VOC Reduction	0.007	0.086	0.727	4.89	19.6	64.0	173
2007 65% NO_x + 15% VOC Reduction	0	0.008	0.154	1.51	10.1	44.5	146
2007 75% NO_x + 15% VOC Reduction	0	0	0.008	0.299	3.81	25.7	115
2007 85% NO_x + 15% VOC Reduction	0	0	0	0	0.239	5.71	60.5
Rollback to 1-hr NAAQS	0	0	0	0	0.118	4.28	49.0

Table 4-10. The estimated number of person-days per year with 8-hr maximum daily ozone concentrations above various thresholds for 1993-1995 and for alternate 2007 scenarios under the "all outdoors" exposure assumption.

Case	Million person-days per year with 8-hr maximum daily ozone concentrations above thresholds							
	8-hr ozone threshold (ppb)							
	>140	>120	>110	>100	>90	>85	>80	>70
1993-1995	0.174	2.77	7.94	18.5	37.4	53.2	72.6	125
2007 Base Case	0.093	2.11	6.99	18.7	40.4	59.1	84.2	151
2007 25% NO_x + 15% VOC Reduction	0.038	1.70	5.70	16.4	37.4	55.0	80.2	146
2007 Clean Diesel + 15% VOC Reduction	0.025	1.51	5.30	15.5	36.1	53.6	78.5	145
2007 45% NO_x + 15% VOC Reduction	0.012	0.914	3.88	12.8	32.0	48.2	71.7	139
2007 65% NO_x + 15% VOC Reduction	0.005	0.238	1.22	5,74	19.9	32.6	52.3	117
2007 75% NO_x + 15% VOC Reduction	0	0.012	0.299	1.83	9.59	19.0	34.0	94.0
2007 85% NO_x + 15% VOC Reduction	0	0	0.005	0.144	1.13	3.77	10.9	53.1
Rollback to 1-hr NAAQS	0	0	0	0.012	0.601	2.47	7.87	42.3

Table 4-11 The estimated number of person-days per year with 24-hr daily average ozone concentrations above various thresholds for 1993-1995 and for alternate 2007 scenarios under the "all outdoors" exposure assumption.

Case	Millions of person-days per year with daily average ozone concentrations above thresholds					
	24-hr ozone threshold (ppb)					
	>80	>70	>60	>50	>40	>30
1993-1995	0.336	1.70	9.06	34.8	97.8	220
2007 Base Case	0.294	1.58	9.51	40.2	118	277
2007 25% NO_x + 15% VOC Reduction	0.252	1.39	8.48	38.4	115	275
2007 Clean Diesel + 15% VOC Reduction	0.234	1.22	7.84	37.3	114	274
2007 45% NO_x + 15% VOC Reduction	0.205	1.09	6.99	35.0	111	272
2007 65% NO_x + 15% VOC Reduction	0.084	0.614	3.95	27.6	98.8	263
2007 75% NO_x + 15% VOC Reduction	0.003	0.303	2.42	21.1	88.0	254
2007 85% NO_x + 15% VOC Reduction	0	0.121	1.17	12.6	73.9	238
Rollback to 1-hr NAAQS	0	0.026	0.982	11.2	70.3	234

Table 4-12. Comparison of the "all outdoors" and "microenvironmental" exposure estimates for 1-hr maximum daily ozone exposures for 1993-1995.

1-hr ozone threshold[a]	Person-days per year with 1-hr maximum daily ozone concentrations above various thresholds	
	All outdoors[a]	Microenvironmental[a]
> 0	421 million	421 million
> 80	157 million	33.6 million
> 100	69.9 million	12.5 million
> 120	26.2 million	4.02 million
> 140	8.20 million	1.11 million
> 160	2.14 million	0.241 million
> 180	336,100	34,400
> 200	58,400	5,800
> 220	5,910	670

[a] Estimates of person-days above 0 ppb may be biased low due to omission of some lower ozone days.

Table 4-13. Comparison of the "all outdoors" and "microenvironmental" exposure estimates for 8-hr maximum daily ozone exposures for 1993-1995.

8-hr ozone threshold[a]	Person-days per year with 8-hr maximum daily ozone concentrations above various thresholds	
	All outdoors[a]	Microenvironmental[a]
> 0	421 million	421 million
> 20	414 million	154 million
> 40	344 million	52.8 million
> 60	201 million	16.8 million
> 80	72.6 million	3.96 million
> 100	18.5 million	0.64 million
> 120	2.77 million	0.062 million
> 140	174,000	3,240
> 160	6,050	122

[a] Estimates of person-days above 0, 20, and 40 ppb may be biased low due to omission of some lower ozone days

Table 4-14. Comparison of the "all outdoors" and "microenvironmental" exposure estimates for 1-hr maximum daily ozone exposures for 1993-1995 and three alternate 2007 scenarios.

Case		Million person-days per year with 1-hr maximum daily ozone concentrations above various thresholds							
		1-hr ozone threshold (ppb)							
		>220	>200	>180	>160	>140	>120	>100	>80
1993-1995	All Outdoors	0.00591	0.0584	0.3361	2.14	8.20	26.2	69.9	157.8
1993-1995	Microenvironmental	0.00067	0.0058	0.0344	0.241	1.11	4.02	12.5	33.6
2007 Base Case	All Outdoors	0.00745	0.0258	0.2209	1.58	7.55	26.7	77.7	189.0
2007 Base Case	Microenvironmental	0.00084	0.0026	0.0223	0.169	0.968	3.92	13.5	39.1
2007 45% NOx + 15% VOC Reduction	All Outdoors	0	0.0074	0.0862	0.727	4.89	19.6	64.0	173.0
2007 45% NOx + 15% VOC Reduction	Microenvironmental	0	0.00084	0.00802	0.0734	0.590	2.79	10.8	34.6
2007 Rollback to 1-hr NAAQS	All Outdoors	0	0	0	0	0	0.118	4.28	49.0
2007 Rollback to 1-hr NAAQS	Microenvironmental	0	0	0	0	0	0.0117	0.503	7.64

Table 4-15. Average per capita exposure to 1-hr daily maximum ozone in 1993-1995 by demographic group under the microenvironmental assumption.

Gender-age group	Average per capita exposure to 1-hr daily maximum ozone per year		
	> 80 ppb	> 120 ppb	> 160 ppb
Males 0-4 yrs	12.5	1.40	0.077
Males 5-19 yrs	13.0	1.57	0.087
Males 20-64 yrs	10.2	1.33	0.090
Males 64 + yrs	10.2	1.16	0.073
Female 0-4 yrs	10.7	1.21	0.070
Female 5-19 yrs	10.1	1.23	0.077
Female 20-64 yrs	5.3	0.54	0.027
Female 64 + yrs	5.5	0.58	0.033
All groups – Microenvironmental	8.9	1.07	0.063
All groups – All outdoors	41.7	7.05	0.577

Table 4-16. Average per capita exposure to 1-hr daily maximum ozone in 1993-1995 by county under the "all outdoors" assumption.

County	Average per capita exposure to 1-hr daily maximum ozone per year		
	> 80 ppb	> 120 ppb	> 160 ppb
Brazoria	31.9	3.32	0.183
Chambers	40.6	4.88	0.793
Fort Bend	36.9	5.34	0.213
Galveston	32.6	5.87	0.777
Grimes	46.6	8.16	0
Harris	43.1	7.34	0.593
Liberty	43.1	4.36	0.010
Montgomery	53.5	8.94	0.677
Waller	43.0	4.32	0
Wharton	25.2	0	0
Whole Region	43.2	6.96	0.570

Table 4-17. Estimated number of Houston residents with annual average exposure to $PM_{2.5}$ concentrations above various thresholds in 1993 and 2007 under alternate scenarios.

Annual $PM_{2.5}$ threshold ($\mu g/m^3$)	Millions of people above annual average $PM_{2.5}$ thresholds						
	1993 Base case	2007 Scenario 1	2007 Scenario 2	2007 Scenario 3	2007 O_3 SIP	2007 Clean Diesel	Rollback to NAAQS
> 11	3.76	4.81	4.81	4.81	4.81	4.81	4.81
> 12	3.75	4.81	4.80	4.81	4.80	4.80	4.70
> 13	3.62	4.70	4.69	4.70	4.69	4.69	3.76
> 14	3.35	4.50	4.46	4.49	4.44	4.44	1.53
> 15	2.36	3.85	3.69	3.75	3.56	3.56	0.0189
> 16	1.29	2.65	2.51	2.60	2.35	2.35	0
> 17	0.376	1.55	1.30	1.44	1.17	1.17	0
> 18	0	0.473	0.418	0.454	0.223	0.223	0

Table 4-18. Estimated number of person days per year with daily exposure to $PM_{2.5}$ concentrations above various thresholds in 1993 and 2007 under alternate scenarios.

Daily $PM_{2.5}$ Threshold ($\mu g/m^3$)	Millions of person-days per year above daily $PM_{2.5}$ thresholds						
	1993 Base Case	2007 Scenario 1	2007 Scenario 2	2007 Scenario 3	2007 O_3 SIP	2007 Clean Diesel	Rollback to NAAQS
> 6	1301	1662	1662	1662	1662	1662	1662
> 10	1041	1329	1329	1329	1329	1329	1329
> 14	701	935	925	931	919	919	718
> 18	424	629	611	619	600	600	249
> 22	228	371	354	361	343	343	67.5
> 26	129	221	210	215	202	202	5.74
> 30	50.8	125	115	120	107	107	0
> 34	12.9	48.8	37.8	41.6	33.4	33.4	0
> 38	0.963	16.3	13.4	15.0	10.7	10.7	0
> 42	0	2.66	0.812	1.45	0.492	0.492	0
> 44	0	0.452	0	0	0	0	0

5. HEALTH EFFECTS

Urban air pollution, or smog, has been studied for several decades. It is a complex mixture of particles and gases many of which have documented effects on human health. In the laboratory it is common to study the health effects of one or a few of these contaminants. In the real world, however, humans are exposed to pollutants in complex mixtures containing several different unhealthful contaminants. There is wide agreement that exposure to the mixture of components that we characterize as urban air pollution can be unhealthy and that such exposures worsen the frequency and severity of lung disease episodes (including respiratory tract infections, chronic diseases such as bronchitis or emphysema and inflammatory diseases such as asthma). There is also some agreement that exposure to air pollutants at high concentrations can directly or indirectly affect people's hearts. Environmental epidemiological studies have been major contributors to our understanding of air pollution health effects, but these studies in general demonstrate probable associations and not direct causal relationships to identify the pollutant components responsible.

Regulatory agencies and the scientific community have wrestled with the problem of identifying the key harmful components of air pollution but the problem is a difficult one and although there is certainty that air pollution mixtures are related to adverse health effects, there is less certainty when it comes to which components are primarily responsible for these unhealthful effects. An important reason for this is that because many pollutants are strongly correlated in urban air pollution, it is not always possible to separate the effects of one pollutant from another (or to establish definitively that the observed association is not possibly related to some other, unmeasured, pollutant that is strongly correlated with the measured pollutants. This is not the case for laboratory studies, where many acute effects of specific compounds have been documented in controlled experiments. However, laboratory studies with human volunteers are ethically limited to the study of responses that are relatively minor and readily reversible.

In this review we mainly focus on analyses of real world exposures, as documented through environmental epidemiological studies, as these best represent actual human exposure. We have used laboratory-based studies to help establish the credence of some findings (i.e., associations between specific pollutant exposures and respiratory symptoms), however the major valuations are based upon response functions derived from the epidemiological literature.

A wide array of adverse heath effects is associated with exposure to ozone and fine particles. Much of the research that establishes these associations has been done to determine where to set ambient air quality standards. Consequently, this research was designed primarily to establish thresholds for safety, rather than to investigate the association between changes in pollutant concentrations and changes in the frequency of effects. In this section, we describe:

- The process used to determine which exposure-response relationships to use for economic valuation.

- The detailed health science literature that supports setting of standards and the scientific basis for accepting the association between ozone, fine particle exposure, and adverse health effects more generally.
- The specific studies and exposure-response relationships used to quantify the frequency of adverse health effects in Houston.

5.1 ESTABLISHMENT OF EXPOSURE-RESPONSE RELATIONSHIPS

Several issues were considered in selecting studies for inclusion in this health benefits analysis. The papers selected had been published in peer-reviewed literature and had also been reviewed through public scientific review processes including the EPA's Clean Air Science Advisory Committee (CASAC) of the Science Advisory Board (SAB), the National Academy of Science, and the Health Effects Institute. Inclusion of epidemiological studies was restricted to those that gave adequate consideration to potentially confounding variables. Such confounders include weather-related variables, age and gender mix of the subject population, and pollutants other than ozone or PM that might reasonably have effects on the health response being evaluated.

The degree to which uncertainty in the analyses was determined varied from study to study. There are very few instances of a formal quantitative analysis, such as a Monte Carlo procedure, being included in the results of the selected papers and such an analysis was beyond the scope of the present project. In this analysis we included estimates of uncertainty as provided within the papers and further evaluated adequacy of treatment of confounders and the strength of exposure-response relationships (where they were provided) in developing a level of confidence for the inclusion of any specific study. The magnitude of exposures and the characteristics of the pollution mix were important considerations, especially for applying results from foreign studies to the Houston area population.

In some studies multiple analyses were performed. Some of these analyses yielded statistically significant findings at the 95 percent confidence level, while other analyses in the same publication did not. When one performs several analyses of the same data set it is possible for some findings to be statistically significant by random chance (i.e., at the 95 percent confidence level, one false positive, or false negative, finding out of 20 could be expected). Each study in which multiple analyses were performed was examined to determine whether there was consistency between the findings; a lack of consistency lowered the degree of confidence in the study.

Many studies reported results in terms of a relative risk (RR) value. The RR is the ratio of the incidence of disease (or health outcome) in two population groups exposed to different concentrations of pollutant. The health benefits analysis required that RR be converted to a corresponding coefficient (β) using the functional form of the regression equation used to estimate the RR. Some of the dose response functions are expressed as change relative to some baseline condition. Where incidence data for health outcomes were not available specifically for Houston area populations (the majority of cases) data from the National Center for Health Statistics were used to supplement the information provided by the studies themselves.

5.2 SCIENTIFIC BASIS FOR HEALTH-BASED STANDARDS FOR SELECTED POLLUTANTS

5.2.1 Ozone

Recent medical studies have uncovered statistically significant relationships between adverse health effects in humans (e.g., as measured by hospital admissions) and ambient O_3 concentrations well below 100 ppb (see, for example, the U.S. EPA Ozone Criteria Document, 1996, and the references cited therein). These studies have precipitated a major reappraisal of the air-quality goals, standards, and policies related to O_3. In the United States, the NAAQS for O_3 has been changed from a 1-hr, 0.12-ppm standard to an 8-hr, 0.08-ppm standard.[1]

5.2.1.1 Biological Responses to Ozone

The effects of ozone have been extensively evaluated in both human clinical studies and in studies with laboratory animals. The deleterious effects include: a) damage to respiratory tract tissues especially in the very sensitive gas-exchange (alveolar) region of the lung (Guth et al., 1986); b) killing of lung cells and increased rates of cell replication (hyperplasia) (Chang et al., 1991; Harkema et al., 1997); c) decreases in pulmonary function (McDonnell et al., 1983); d) inflammation of airways (Devlin et al., 1991); and e) increases in respiratory symptoms, such as cough, chest (substernal) soreness, difficulty in taking a deep breath, and in some cases headaches or nausea (Folinsbee, 1981; Hoek and Brunekreef, 1995; Hoek et al., 1993; Lag and Schwarze, 1997; Romieu et al., 1997). The effects of ozone have been demonstrated to increase with increasing dose and studies have clearly demonstrated effects at concentrations of 0.08 parts per million (ppm; in this case, a part per million is one molecule of ozone per 1,000,000 molecules of air). It has also been demonstrated that in laboratory animals, exposure to mixtures of ozone with either particles (Kleinman et al., 1995; Kleinman et al., 1993) or other pollutant gases (such as NO_2) (Gelzleichter et al., 1992) were more toxic than ozone alone, causing damage to lung tissue after short-term exposures. Intermittent exposure of monkeys to ozone increased the adverse pulmonary effects. Concurrent exposure to nitrogen dioxide (0.8 ppm) and ozone (0.2 ppm) increased the susceptibility of mice to bacteria-induced pneumonia (Gardner, 1984).

At the biochemical level, ozone can alter metabolic enzyme activity and, thus, influence the subsequent effect of another pollutant, or vice versa. On the other hand, the modulating effect can be exerted on the dosimetry of the inhaled pollutants and, indirectly, on the observed adverse effects. For example, O_3-induced changes in the lung can result in modifications of inhaled particle dose, and can influence alterations in pulmonary function and lung defense mechanisms such as mucociliary transport of particles, alveolar macrophage phagocytic activity, and alveolar macrophage translocation of ingested particles (Gerrity, 1995). Inhaled particles, however, can change the properties of airway mucus, making it thicker and perhaps

[1] The NAAQS are stated in units of ppm and to two significant figures. Thus, 1-hr and 8-hr exceedances are technically defined as a concentration of at least 0.125 and 0.085 ppm, respectively; (i.e., concentrations that, when rounded to two significant figures, yield values equal to or greater than 0.13 and 0.9 ppm).

more viscous, thus limiting the ability of O_3 to diffuse through the mucus to reach the underlying epithelium and perhaps diminishing the ozone effect (Graham, 1989). Several papers have, however, described increased toxicity of ozone-particle mixtures versus that of either component alone (Anderson et al., 1997; Graham, 1989; Jakab and Hemenway, 1994; Vincent et al., 1997). In the atmosphere, ozone can catalyze, or augment, the conversion of gas phase pollutants to particle phase pollutants. Kleinman et al. (1989) showed that, for example, sulfur dioxide could be converted to acid sulfate particles in the presence of droplet aerosols, and that this conversion was accelerated if the droplets contained reactive transition metal ions, such as iron or manganese.

The biological effects of the combination of nitrogen dioxide and ozone have also been studied. Here, in addition to possible biochemical and dosimetric factors, the toxic interactions can also include the formation of reactive intermediate compounds that can have an effect on health, in this case nitric oxide and nitrate-free radicals. There are several studies that suggest a synergistic interaction between ozone and nitrogen dioxide (Gelzleichter et al., 1992; Last et al., 1984; Mautz et al., 1988; Oehme et al., 1996). There can also be interactions with materials derived from biological components of ambient aerosols. For example, pre-exposure to endotoxin, a component of the cell walls of bacteria, can alter the response to ozone in animal studies. Whether it augments or reduces the ozone effect depends in part on the order of exposure and possibly to the dose (Peavy and Fairchild 1987; Pendino et al., 1996).

5.2.1.2 Benefits Assessment for O_3

Controlled laboratory studies with several species of animals have clearly shown significant and persistent damage to the lung. The changes seen include thickening of lung tissue structures (Tyler et al., 1988)(seen in light microscopy sections of lung tissue) which would be consistent with "stiffening" of the lung (Barry et al., 1985; Tepper et al., 1993). Functional changes in the lung which are also indicative of "stiffer" lungs (Costa et al., 1995; Harkema and Mauderly, 1994), and changes that are characteristic of those seen in the acute stages of experimentally caused pulmonary fibrosis (Last et al., 1994) are also adverse effects of ozone. The long-term effects of ozone exposure, therefore, seem to promote accelerated “aging” of the lung and persistent, and possibly irreversible, changes in lung structure. In summary, ozone is a respiratory irritant that causes physiological effects, lung inflammation, and disruption or death of cells along the respiratory tract. Adaptation, or diminution of responses, has been observed in both human subjects and laboratory animals after repeated exposures on sequential days. This phenomenon is mainly associated with responses related to sensory irritation and respiratory symptoms. This is not to say that adaptation leads to the elimination of adverse health effects. There is credible evidence that repeated exposures continue to produce changes in the lung at the tissue level, despite the apparent reduction of pulmonary function effects. The effects of other pollutants can modify the effects of ozone due to chemical interactions in the aerosol, biochemical interactions in the target, or changes in the dose to the target. The deleterious effects of the ozone-particle mixture can, in some circumstances, be underestimated from the effects of the mixture components taken individually.

5.2.2 PM

Fine particles (including $PM_{2.5}$) are defined as atmospheric particulate matter with diameters less than 2.5 μm[2]. Because of their small size, such particles can penetrate deep into the lungs of a respiring individual and, thus, have increased potential to cause respiratory and other health effects. Recent epidemiological studies (National Research Council, 1998) show a statistical association between adverse health effects and relatively low concentrations of $PM_{2.5}$. As a result, a new NAAQS for $PM_{2.5}$ has been promulgated in the United States (Federal Register, 1997). The U.S. NAAQS for PM_{10} (defined as particles with aerodynamic diameters less than 10 μm) remains in effect. These actions imply that our knowledge is sufficient to accept that fine particles are associated with adverse health effects, and that the $PM_{2.5}$ is more harmful than an equivalent mass of PM_{10}. There is, however, controversy with respect to this implication. There is evidence that some pollutants by themselves have clear human health effects (e.g., the results of O_3 chamber work that demonstrate respiratory, acute, reversible, irritant, etc. effects are nearly irrefutable). Strictly speaking this is not the case for PM. However, much of the past research did not address biological mechanisms associated with PM effects or the role of particle physical and chemical characteristics because the research was designed primarily to identify any harmful effects (and determine thresholds), not to explain why they occurred. It is not known precisely what aspect(s) of particles may contribute to causing adverse health effects, what role the gases play, and how the interaction among the pollutants (in terms of the time sequence of exposure and magnitude of concentrations) affects health outcomes.

5.2.2.1 Clinical and Toxicological Studies

For most air pollutants other than PM (e.g., O_3 or NO_2), controlled laboratory studies with human volunteers or animal models have been able to demonstrate convincingly biologically plausible mechanisms by which that pollutant causes a given effect on health. In contrast, most controlled laboratory studies of PM_{10}-related aerosols have shown small effects at exposure levels comparable to ambient concentrations, even when the studies are restricted to those components of PM_{10} which are the most toxic (e.g., sulfuric acid particles). Recent studies with laboratory animals and human volunteers exposed to concentrated ambient fine particles have demonstrated mortality in seriously compromised laboratory animals and heart rhythm and heart rate anomalies in human subjects. These effects may be suggestive of the mechanisms by which PM may exert deleterious effects during pollution episodes. Acute exposures to particles have been demonstrated to elicit release of inflammatory mediators and stress proteins, and laboratory-generated particles in combination with ozone can cause increased proliferation of cells in the epithelium and interstitium of the lung. However, the specific components of ambient PM_{10} that may be responsible for deleterious effects are not known.

[2] The regulatory definition of $PM_{2.5}$ is "particles that behave aerodynamically as if they had diameters of 2.5 μm or less."

It may be that these laboratory studies have not employed the correct mixture of other air pollutants in addition to particles to elicit responses. It also may be that it is necessary to perform studies with subjects that more closely resemble the actual population at risk in order to observe effects and establish biological plausibility for PM_{10} health effects.

Laboratory studies are intrinsically less subject to confounding factors than are epidemiological studies, but they are limited with respect to how realistically they duplicate the true ambient exposure conditions of human populations and model the physical characteristics of the members of the human population who are susceptible to PM_{10}. The aerosols that have been studied in the laboratory, with the important exceptions of diesel exhaust and furnace effluent particles, have not been combustion generated. Nevertheless, some of these studies have demonstrated biological effects that relate to some of the postulated mechanisms by which PM_{10} causes illness. For example, changes in mucociliary clearance, macrophage functions, and airway hyper reactivity have been demonstrated after exposures to acidic sulfate aerosols (Schlesinger, 1995). Others have demonstrated changes in macrophage functions and lung permeability in rats exposed to acid-coated diesel exhaust particles and carbon particles (Kleinman et al., 1988; Prasad et al., 1988). Sulfuric acid, when coated onto the surface of zinc oxide particles (Amdur, 1989) produced significant decreases in pulmonary function in guinea pigs, a laboratory animal that is often used as an asthma model. Greater effects were observed in animals exposed to the 0.05 µm aerodynamic diameter sulfuric acid-coated particles than were seen in animals exposed to the acid alone (as 0.3 µm particles). This finding might be suggestive of the importance of particle size, or particle number, in the relationship between PM_{10} exposures and health effects.

5.2.2.2 Epidemiological Studies

Epidemiological studies, conducted in diverse communities having different climates, different pollutant characteristics, and involving a broad spectrum of socioeconomic groups, have associated PM_{10} exposure with increased rates of respiratory illnesses, hospitalization, and increased incidences of premature deaths. These relationships have been reviewed extensively in recently published articles (Dockery and Pope 1994; Ostro et al., 1993). Time series analyses of daily mortality and 24-hr average PM_{10} concentrations associate an approximate 1 percent increase in mortality with a 10 $\mu g/m^3$ increase in PM_{10}. These associations have been statistically significant, in some instances, even though the PM_{10} daily (24-hr) average concentrations were below the current U.S. PM_{10} NAAQS level of 150 $\mu g/m^3$. Typically, the time lag between pollution increases and health effect manifestations is on the order of one to a few days. Long-term effects of PM_{10} exposure have been documented in cross-sectional studies (Evans et al., 1984). These studies also predict excess mortality in association with increased PM_{10} concentrations, and the magnitude of this effect agrees with integrated mortality estimates made from the time series analyses.

Most of the chronic effects studies are cross-sectional studies, rather than longitudinal studies. If PM_{10} caused acute mortality only in individuals who were near death and would have died within a short time, even without the advent of the episode, these deaths would be

detected in longitudinal studies but not in cross-sectional analyses. This so-called "harvesting" effect has been discussed and may offset some (20 percent) of the particle-related mortality (Spix et al., 1993; Thurston et al., 1989). On the other hand, if PM_{10} had little acute effect, but if long-term exposure degraded cardiopulmonary functions and, thus, contributed to the development and eventual terminal effects of chronic cardiopulmonary disease, these effects would be observed in cross-sectional studies but not in longitudinal studies. If, however, the mortality associated with PM_{10} episodes was related to acute deaths, which otherwise would have been delayed for months or years, then the effects of those episodes would be observed in both types of epidemiological study. In this latter case, the longitudinal and cross-sectional studies may or may not be addressing different phenomena (Evans et al., 1984).

There has been some inconsistency between epidemiological evidence and the findings of laboratory studies (using human or laboratory animal subjects) regarding the impact of inhaled particles on health. If the epidemiological evidence is correct, then the potency of PM_{10} (or some unspecified component) could be astoundingly high. Furthermore, if there is a threshold (or no observable effect level) for deleterious effects of PM_{10}, that threshold would appear to be lower than the typical ambient concentrations in even relatively "clean" areas. On the other hand, early laboratory studies, using components of PM_{10} thought to be toxic, generally require exposures at concentrations much higher than the average ambient levels in polluted cities in order to demonstrate relatively subtle changes in biological endpoints. This has led some to express skepticism regarding the potential impacts of particles on health. However, recent laboratory studies have demonstrated responses akin to acutely life-threatening human responses to ambient particles. Some possible explanations for the discrepancy between epidemiology and earlier laboratory studies might include: (1) unidentified or incompletely controlled confounders such as weather-related physiological stresses or aeroallergens; (2) highly toxic species in ambient PM_{10} that have not been included in laboratory studies; (3) failure to identify and test vulnerable populations in laboratory studies; (4) interactions among key PM_{10} components in ambient air that have not been tested in laboratory studies; and (5) the fact that the biological endpoints measured so far in studies of healthy versus compromised animals are not related closely enough to the mechanisms of acute PM_{10}-related deaths in communities. The more recent studies using concentrated ambient fine particles and animal models with lung or heart diseases have confirmed that particle exposures can cause death and seriously exacerbate existing lung diseases (Godleski et al., 1996).

5.2.2.3 Benefits Assessment for PM

The evidence supporting the association between PM_{10} and health effects is compelling because it suggests both ***consistency*** of effects across independent studies performed by different investigators in communities with different characteristics and ***coherence*** of effects across related health outcomes. For example, if mortality is increased in a population, one would expect that some possibly larger fraction of the population would become clinically but not mortally ill, that hospital and emergency room visits would increase, that pre-existing respiratory disease states might be exacerbated, that respiratory symptoms would increase, and that measures of lung function would show impairment (Dockery and Pope, 1994). In the case

of PM_{10} exposures, an example for such coherence can be seen in **Table 5-1**, which is adapted from data presented by Dockery and Pope (1994).

5.2.3 Air Toxics

Polluted urban air contains an extremely complex mixture of gases and particles consisting of thousands of inorganic and organic compounds. The gaseous constituents include carbon dioxide, carbon monoxide, nitric oxide, nitrogen dioxide, oxides of sulfur, hydrocarbons (e.g., ethylene, formaldehyde, methane, benzene, phenol, 1,3-butadiene, acrolein [Health Effects Institute, 1995]), polycyclic aromatic hydrocarbons (PAHs) (e.g., napthalenenes, benzopyrenes, benzanthracenes, and fluoranthrenes), oxygenates (Choudhury, 1982), and nitroaromatic species (Newton et al., 1982; Pitts et al., 1982). Combustion processes, industrial sources, and emissions from internal combustion engines (gasoline and diesel) are the major contributors of these compounds. Three of these compounds (benzene, formaldehyde, and 1,3-butadiene) are present in Harris County air at concentrations that exceed currently accepted risk levels. The health-related aspects of exposures to these compounds (in the context of ambient exposures), and potential benefits from exposure minimization are discussed in this section of the report.

5.2.3.1 Benzene

Recent studies of benzene exposure found that personal exposures exceeded indoor concentrations, which, in turn, exceeded outdoor concentrations of this toxic contaminant and also determined that food is not an important pathway for benzene exposure (Wallace, 1996). The major sources of exposure are active and passive smoking, auto exhaust, and driving or riding in automobiles. Data from waste sites show that industrial wastes may be another important source of exposure. Recently analyzed data identify the most frequently found contaminant combinations in waste site samples were perchloroethylene (PERC) and trichloroethylene (TCE) in water (23.5 percent of sites); chromium (Cr) and lead (Pb) in soil (20.5 percent); benzene and toluene in air (3.5 percent); PERC, 1,1,1-trichloroethane (1,1,1-TCA), and TCE in water (11.6 percent); Cr, cadmium (Cd), and Pb in soil (12.0 percent); and benzene, and PERC in air (2.2 percent) (Fay and Mumtaz, 1996). In terms of emissions in the Houston area, the largest contributions in the state of Texas come from the petrochemical industries along the Gulf Coast (Pendleton, 1995). Benzene is mutagenic and carcinogenic, and occupational exposures have been clearly associated with acute myeloid leukemia (AML). However, the epidemiologic evidence linking benzene to leukemia in aggregate, as well as for subtypes other than AML, is no less persuasive than that for AML alone (Savitz and Andrews, 1997). Several reports suggest an association of benzene exposure with multiple myeloma, malignant lymphoma, and lymphatic leukemia (Kaneko et al., 1997). Some reports suggest positive associations between gasoline exposures and the development of myeloleukemia and other diseases (Caprino and Togna, 1998; Kaneko et al., 1997; Nordlinder and Jarvholm, 1997), however methodological differences between studies and changes in gasoline composition over the years make it difficult to quantitatively assess the risk. There is sufficient epidemiological evidence indicating that benzene is a human carcinogen. Benzene can also exert an effect on the heart. An increased risk of spontaneous abortion was found in women

exposed to petrochemicals including benzene and gasoline (Xu et al., 1998). Acute doses of benzene can also contribute to cardiac arrhythmias (abnormal heartbeats) and, in fact, benzene is used to develop animal models of heart disease for cardiac research.

5.2.3.2 1,3-Butadiene

The International Agency for Research on Cancer (IARC) has given to 1,3-butadiene the designations of "sufficient evidence" of carcinogenicity in experimental animals and "limited evidence" of carcinogenicity in humans. In a recent cohort mortality study (Ward et al., 1996) conducted among 364 men who worked in one of three 1,3-butadiene production units, neither benzene nor ethylene oxide was present at the facilities. The study found a significantly elevated standardized mortality ratio (SMR) for lymphosarcoma and reticulosarcoma based on four observed cases (SMR = 577; 95 percent confidence interval (CI) = 157-1480). An excess of lymphosarcoma among workers with routine exposure to 1,3-butadiene was also observed in a previous study (Devine et al., 1993).

Another cohort mortality study of 2795 male workers employed at least 6 months between 1942 and 1994 at a 1,3-butadiene facility showed 42 deaths from lymphohematopoietic cancers (LHC) with 28.6 expected (SMR = 147, 95 percent CI = 106-198), 9 observed versus 4.7 expected deaths for lymphosarcoma (SMR = 191, 95 percent CI = 87-364), 13 observed versus 11.5 expected deaths for leukemia (SMR = 113, 95 percent CI = 60-193), and 15 observed versus 9.9 expected deaths from cancer of other lymphatic tissue (SMR = 152, 95 percent CI = 85-250) (Divine and Hartman, 1996).

Most of the epidemiological studies just described used surrogate measures of dose out of necessity. Some extrapolations have been made using animal toxicology data. Estimated workplace cancer risks that are based on the assumption that humans are as responsive as mice would suggest added risks of 200 or more out of 1000 workers (1 in 5) exposed to 2 ppm butadiene (assuming 40 years of exposure). These estimates are clearly much greater than the level of risk suggested by the human studies (Cagan et al., 1996). Other estimates of elevated risks to workers exposed to butadiene at a concentration of 2 ppm range from 0.2 per 10,000 workers (based on female mouse heart hemangiosarcomas) to 600 per 10,000 workers (based on female mouse lung tumors). Depending on the assumptions, estimates of lifetime excess risk derived from the female mouse lung model ranged from 60 per 10,000 to 1600 per 10,000 (Dankovic et al., 1993; Melnick and Huff, 1993). Following an extensive review of the available literature, Landrigan (1993) concludes that (1) the epidemiological data strongly suggest an etiological association between exposure to butadiene and human cancer; (2) the data amply fulfill all of Bradford Hill's criteria for causality; and (3) it is therefore reasonable to conclude that there is convincing evidence for the carcinogenicity of butadiene to humans. Tornqvist and Ehrenberg (1994) estimated that ambient butadiene exposures (average exposures in Sweden) could cause 20 deaths per 100,000 exposed.

5.2.3.3 Formaldehyde

Both the EPA and the Occupational Safety and Health Administration (OSHA) now regard formaldehyde as a possible human carcinogen, based upon their consideration of available epidemiologic and toxicological data (Anon., 1989). However, the evidence is far from clear and there is some lack of consistency among studies of site-specific cancer risk. Uncertainty resulting from unresolved confounding by known risk factors (Anon., 1988) and lack of clear dose-response relationships in human studies were identified as significant obstacles to more definitive conclusions (Starr, 1990). The most definitive relationships are derived from animal toxicology data, but there are significant species and site-specific differences in tumorigenic responses of animals and people. Environmental exposures to formaldehyde have been clearly associated with respiratory irritation and allergic sensitization and therefore can contribute to the exacerbation of airway diseases such as asthma (Liu et al., 1991; Schnuch and Geier 1997; Smedje et al., 1997).

5.2.3.4 Benefits Assessment for Air Toxics

The effects of air toxics are difficult to quantify for several reasons. Most of the adverse effects of interest are irreversible and involve painful and/or protracted diseases. Therefore they cannot be studied in human clinical settings (in contrast to PM or ozone). Epidemiological studies need to cover extended periods of time and are often confounded by simultaneous exposures to several pollutants. The health effects associated with air toxics include cancer incidence and mortality as well as non-cancer effects such as neurological, respiratory, cardiovascular, reproductive, hematopoetic, developmental and immunological diseases, and toxicity. EPA has estimated that mobile source emissions account for about 60 percent of the air toxics cancer risk, stationary sources contribute about 15 percent and area sources contribute about 25 percent. About 95 percent of the 1700 to 2700 excess annual air toxics-related cancer cases nationally can be attributed to 14 specific pollutants (arsenic, asbestos, benzene, 1,3-butadiene, carbon tetrachloride, chloroform, hexavalent chromium, dioxin, ethylene dichloride, ethylene dibromide, formaldehyde, gasoline vapors, products of incomplete combustion, and vinyl chloride). Of these 14 pollutants, three (benzene, 1,3-butadiene, and formaldehyde) may be of significance with respect to exposures in the Houston area based on data from atmospheric monitoring.

Quantification of any health benefits would entail evaluation of measures that would reduce emissions from these contributors. Since the institution of air quality standards for air toxics there have been substantial reductions in emissions. Overall, however, the EPA has estimated that benefits related to reductions in air toxics emissions (other than asbestos and vinyl chloride) were relatively small on the national scale, when computed as a function of reduced incidences of cancer (~ $19.5 million dollars per year from 1982-1990).

5.3 SELECTION OF STUDIES FOR QUANTITATIVE ESTIMATION

5.3.1 Ozone Effects on Adults

More than 60 epidemiological studies that addressed the health effects of ozone on adults were examined in the process of choosing appropriate studies to use in the health benefits assessment. The studies are described individually in **Tables 5-2 and 5-3**. The studies are arranged by year with the most recent studies listed first and are in alphabetical order by author for each of the years. Overall, the results of the studies point to an association between ozone exposure and morbidity and mortality. Assigning a health effect to a single component in a mixture of ambient air pollutants is difficult, however, because the pollutant may be acting as a surrogate for the toxicity of the mixture. Nevertheless, the finding of independent ozone effects in multiple locations that have different levels of various pollutants increases confidence in the hypothesis that relatively low levels of ozone in ambient air have measurable adverse health consequences.

We have chosen several acute health effect time-series studies as sources of data for the ozone health benefits analyses. Time-series studies are population-based studies; that is, the relationship of exposure and outcome is analyzed for a population in the aggregate rather than for individuals. An important feature of time-series studies is the ability for the study population to act as its own control, making a separate control population unnecessary. Critical to time-series analysis, however, is controlling for the regular pattern of changes in variables over time. A major confounder in correlational time-series studies is long-wavelength patterns. Effects of season may be the most important long-wavelength pattern that needs addressing. Long-term trend (i.e., over the years) also may need to be removed from the data. In addition, calendar-specific days (e.g., day-of-week, holidays), influenza epidemics, and unusual events (e.g., strikes) may cause confounding. We have attempted to choose studies that have dealt adequately with these possible confounders as well as with autocorrelation (the tendency of two observations close together in time to be more similar to one another than observations spaced more distantly).

We also have selected those studies that have included in the assessment all components of the mix of ambient air pollutants for which sufficient measurements were available, as well as meteorological variables, in order to isolate the effects of environmental factors that may be serially correlated with ozone. These variables are limited to those that may produce a biologically plausible health effect. Furthermore, increased confidence in the study results for ozone is possible when the intercorrelation of ozone with other pollutants is low.

Failure, in some studies we reviewed, to adequately consider confounding by co-pollutants was a major reason for not including a study in the health benefits analyses. An exploratory study of the effects of air pollution on lung function and respiratory symptoms failed to consider particulate matter as a co-pollutant (Higgins et al., 1995). In another study of respiratory admissions (Hernandez-Garduno et al., 1997), sufficient particulate matter data were not available to include in the analysis. Several studies of hospital admissions by Schwartz analyzed only PM_{10} as a potentially confounding pollutant (Schwartz 1994a, 1994b,

1994c). In addition to the association between total mortality and ozone, Sunyer et al. (1996), found associations between ozone and both cardiovascular and respiratory mortality, but these latter relationships were only evident for elderly (≥ 70 years) individuals during summer months. Unfortunately, carbon monoxide, a potential confounder, was not reported. Morris and others analyzed relationships between carbon monoxide (and other gaseous pollutants) and cardiovascular mortality but didn't consider particles (Morris et al., 1995). Studies by Burnett and others examined the effects of exposure to ozone, sulfates, and gaseous pollutants on cardiorespiratory admissions, but did not include analyses of particle-related effects (Burnett et al., 1997a, 1994). Cakmak and colleagues examined various methods of temporal filtering of time series data on the relationship between environmental variables and health outcomes (Cakmak et al., 1998). Although carefully conducted for such a purpose, the study considered no pollutants other than ozone in the analysis and, therefore, we choose not to use the data in the health benefits analysis.

A number of the studies could not be used quantitatively in the benefits analysis because the effects of ozone were not separable from those of the covariates (Abbey et al., 1993; Holmen et al., 1997; Ito and Thurston 1996; Schwartz 1996; Stieb et al., 1996; Sunyer et al., 1991; Thurston et al., 1994, 1992). An independent effect of ozone from particulate matter was unable to be shown in a couple of studies because of the limited number of days of sampling for airborne particulate matter (Schwartz, 1996; Stieb et al., 1996).

In some studies, results were inconsistent and not clearly explainable for differences between or among age groups (Jones et al., 1995), cities (Burnett et al., 1997b; Moolgavkar et al., 1997; Schouten et al., 1996; Schwartz 1995), seasons (Castellsague et al., 1995), or years (Delfino et al., 1998, 1997). For example, Jones et al. (1995) found a significant effect of ozone exposure on emergency department visits by adults but not by pediatric or geriatric patients. Small data sets may have led to inconsistencies in the results of Schouten et al. (1996). Other studies were eliminated from consideration for use in the health effects assessment for methodological reasons. The aggregation of respiratory symptoms into only two categories (upper or lower respiratory tract symptoms) restricted interpretation of the results of the study on air pollution by Ostro et al. (1993). Methodological problems limited the interpretation of the study of ozone effects on lung function and symptoms by Krzyzanowski et al. (1992). Long-wave variations in health outcome data may have confounded the results of several studies (Moolgavkar et al., 1997; Pönkä, 1991; Weisel et al., 1995). The results of the study of house calls by Medina et al. (1997) was not applicable to the United States. The findings of the cohort study of ambient air pollutants and lung cancer need to be investigated in a larger study in order to better separate the independent effects of ozone (Beeson et al., 1998).

Fourteen of the studies found no significant independent association between health effects and ozone (Borja-Aburto et al., 1997; Dab et al., 1996; Delfino et al., 1994; Dockery et al., 1992; Kinney et al., 1995; Morgan et al., 1998; Ostro et al., 1996; Pönkä and Virtanen, 1994, 1996a; Saldiva et al., 1995; Sunyer et al., 1997; Verhoeff et al., 1996; Voigt et al., 1998; Zmirou et al., 1996). Many of these studies looked at total mortality (Dockery et al., 1992; Kinney et al., 1995; Saldiva et al., 1995; Ostro et al., 1996; Verhoeff et al., 1996;

Zmirou et al., 1996; Borja-Aburto et al., 1997; Morgan et al., 1998). Others investigated hospital admissions for respiratory diseases (Delfino et al., 1994; Pönkä and Virtanen, 1994; Dab et al., 1996; Schouten et al., 1996) or asthma only (Pönkä and Virtanen, 1996a; Sunyer et al., 1997). Dab et al. (1996) also examined respiratory mortality. In some of these studies (Pönkä and Virtanen, 1994; Delfino et al., 1994; Dab et al., 1996; Verhoeff et al., 1996; Voight et al., 1998), the lack of a significant positive association was due perhaps to relatively low ambient ozone levels. Other studies lacked sufficient power to detect associations because of the short study period (Dockery et al., 1992) or the small data set (Kinney et al., 1995; Zmirou et al., 1996). A negative association between ozone exposure and mortality was found in a study conducted in Sao Paulo, Brazil (Saldiva et al., 1995). Modeling difficulties were reported in the study of asthma hospital admissions and air pollution by Pönkä and Virtanen (1996a). Heterogeneity among cities and seasons in the association of ozone levels with asthma prevented a significant finding in the meta-analysis by Sunyer et al. (1997).

The results of investigations of the effects of ozone on cardiovascular health outcomes are mixed. Some studies of cardiovascular hospital admissions and ozone exposure have shown an association (Burnett et al., 1997a; Pönkä and Virtanen, 1996b), whereas, others have not (Morris et al., 1995; Schwartz and Morris, 1995). In their study of cardiovascular mortality, Pönkä et al. (1998) found both positive and negative associations depending on the lag structure for ozone. In Mexico City, (Borja-Aburto et al., 1998) found an association between ozone and cardiovascular, but not respiratory, mortality. Simpson et al. (1997) found no significant association between ozone and cardiovascular mortality in Brisbane, Australia.

The time-series studies of ozone exposure and health effects in which we have the most confidence include studies of respiratory hospital admissions (Anderson et al., 1997; Burnett et al., 1997a; Schwartz 1996; Spix et al., 1998), daily mortality (Hoek et al., 1997; Kelsall et al., 1997; Simpson et al., 1997; Touloumi et al., 1997), and respiratory symptoms in asthmatic adults (Hiltermann et al., 1998). Two of the studies used the method of meta-analysis to study the short-term effects of air pollution on hospital admissions for respiratory diseases (Spix et al., 1998) and mortality (Touloumi et al., 1997) in several European cities. Three additional studies were conducted in Europe (Anderson et al., 1997; Hoek et al., 1997; Hiltermann et al., 1998), two were conducted in the United States (Schwartz et al., 1996; Kelsall et al., 1997), and one each was conducted in Canada (Burnett et al., 1997b) and Australia (Simpson et al., 1997). A well-performed study of the effects of air pollution on respiratory hospital admissions in London (Ponce et al., 1996) was not chosen to be used in the health effects analysis because the data were included in the meta-analysis by Spix et al. (1998).

The studies chosen as suitable for use in the health benefits analysis appeared to control adequately for potential confounders such as long-term trends, long-wavelength cycles, calendar effects, influenza epidemics, and weather, as well as co-pollutants. Associations from the studies of hospital admissions for respiratory diseases by persons at least 65 years of age include a 3 percent increase in admissions for an increase of 25 ppb ozone (1-hr maximum) (Anderson et al., 1997; Spix et al., 1998) and a 9 percent increase in admissions for a 50 ppb increase in ozone (1-hr maximum) (Schwartz et al., 1996). The study of respiratory hospital admissions by all ages in summer found a 6 percent increase in admissions for a 12 ppb

increase in ozone (1-hr maximum) (Burnett et al., 1997b). In three of the mortality studies, increases of 10-25 ppb ozone (1-hr maximum or 24-hr mean) were associated with a 2 percent increase in the number of daily deaths (Kelsall et al., 1997; Simpson et al., 1997; Touloumi et al., 1997). In the study by Hoek et al. (1997), an increase of 33.5 ppb ozone (24-hr mean) was associated with a 6 percent increase in daily mortality. An increase in 25 ppb ozone (mean 7 day) was associated with a 16 percent increase in bronchodilator use by asthmatic adults (Hiltermann et al., 1998).

In general, the results of studies on the acute effects of air pollutants in which we have the most confidence suggest that ambient levels of ozone can cause small but statistically significant increases in exacerbation of asthma, hospital admissions for respiratory diseases, and mortality. As shown in Tables 5-1 and 5-2, considerable evidence exists for an association between ozone levels and daily hospital admissions or emergency room visits for respiratory diseases. Fewer studies have investigated the relationship between ozone levels and daily mortality and many of them contained weaknesses that prevented unambiguous conclusions. Nevertheless, several carefully performed studies provide evidence that increases in ozone levels are related to increases in total mortality. The plausibility of the effects of ozone on admissions and mortality is strengthened by the consistency of the findings across cities on different continents and with different weather patterns.

5.3.2 Ozone Effects on Children

We examined approximately 40 studies that addressed the health effects of ozone on children. The studies are summarized in **Table 5-4**. The study categories include panel, time series, summer camp, exercise, and daily life studies. The health outcome categories assessed include respiratory symptoms (**Table 5-5**), lung function (**Table 5-6**), and hospital admissions (**Table 5-7**). The studies in each of these four tables are arranged in alphabetical order by author.

Some of studies that we reviewed report no significant association between symptoms and ozone exposure in children (Spektor et al., 1991; Cuijpers et al., 1994; Stern et al., 1994; Hoek and Brunekreef, 1995; Linn et al., 1996; Braun-Fahrlander et al., 1997; Avol et al., 1998). None of these studies examined asthmatic children exclusively although asthmatic children were one of the subject groups examined by Avol et al. (1998). Braun-Fahrlander et al. (1997) found a small positive association with the number of hours per year that the children were exposed to > 80 ppb ozone. Neas et al. (1995) found a positive but nonsignificant association of ozone exposure and cough episodes in schoolchildren. Other studies report, however, significant increases in symptoms following exposure to ozone in schoolchildren (Castillejos et al., 1992; Schwartz et al., 1994; Neas et al., 1995), day camp children (Berry et al., 1991), asthmatic children (Ostro et al., 1995; Delfino et al., 1996, 1998; Romieu et al., 1996, 1997; Gielen et al., 1997; Thurston et al., 1997), and wheezy children (Buchdahl et al., 1996). In the study by Berry et al. (1991), only ozone levels $\geq$120 ppb ozone (1-hr maximum) were significantly associated with symptoms. Studies that

report a significant association between ozone exposure and respiratory symptoms are described in Table 5-5.

A few studies found no significant decrements in lung function following ozone exposure. These include a summer camp study (Berry et al., 1991), an exercise study (Hoek et al., 1993a), and two panel studies (Romieu et al., 1996; Avol et al., 1998). Avol et al. (1998) found significant decrements in lung function only in healthy children during high ozone exposures and not in asthmatic or wheezy children, but limitations in the study methods and design may have compromised the results. Numerous studies found significant decrements in at least one type of lung function test, however. These include daily life studies (Castillejos et al., 1992; Krzyzanowski et al., 1992; Hoek et al., 1993b; Stern et al., 1994; Neas et al., 1995; Studnicka et al., 1995; Thurston et al., 1997), panel studies (Gielen et al., 1997; Romieu et al., 1997; Ulmer et al., 1997;), summer camp studies (Higgins et al., 1990; Kinney et al., 1990; Spektor et al., 1991), and an exercise study (Braun-Fahrlander et al., 1994). Studies that found a significant effect of ozone on symptoms are described in Table 5-6.

Inconsistent results have been reported for the association between ozone exposure and hospital admissions for children. A number of studies found no association between hospital admissions for children and ozone exposure (Bates et al., 1990; Jones et al., 1995; Keiding et al., 1995; Ponce de Leon et al., 1996; Delfino et al., 1997; Hernández-Garduño et al., 1997; Holmen et al., 1997; Sunyer et al., 1997). Almost the same number of studies found significant increases in hospital admissions for children following ozone exposure (Pönkä, 1991; Tseng et al., 1992; Burnett et al., 1994; Romieu et al., 1995; Buchdahl et al., 1996; Pönkä and Virtanen, 1996; White et al., 1994). The studies that reported significant increases in hospital admissions in the form of rate ratios are described in Table 5-7.

5.3.3 PM Effects on Adults

The studies used in assessing the health effects of PM on adults are summarized in **Tables 5-8 and 5-9**. The issues that were considered in selecting PM studies for inclusion in this health benefits analysis were similar to those used for ozone and described earlier. The papers used were peer-reviewed. Included epidemiological studies gave adequate consideration to potentially confounding variables such as weather-related variables, age and gender mix of the subject population, and pollutants other than PM that might reasonably have effects on the health response being evaluated. The exposure metric was of special importance. Studies that did not assess particle mass were not used in the quantitative evaluation. Thus, studies in which BS or visibility were used as PM surrogates were discussed but given low confidence levels. In this study, the health benefits were assessed with respect to meeting the $PM_{2.5}$ standard. Some of the studies that were evaluated provided a β factor based on PM_{10} and not one for $PM_{2.5}$. In those instances, $PM_{2.5}$ was estimated by multiplying the PM_{10} concentration by 0.6, and the β factor was not modified.

More than 50 published studies that addressed health effects of airborne particulate matter (PM) in adults were reviewed and summarized (see Tables 5-8 and 5-9). The design

and quality of the studies were assessed for use in establishing dose-response relationships. All of the studies that were reviewed were observational epidemiologic studies, and very few were chronic studies. By far, the majority of the investigations were group-level (ecological) daily time-series studies. These time-series studies examined the acute effects of air pollution by using ambient concentrations of air pollutants measured at stationary monitors as a surrogate for individual-level exposures. A major advantage of time-series studies as compared with cross-sectional studies is that there is little need to control for risk factors such as age, cigarette smoking, and occupational exposure. The reason these factors have diminished importance as predictors of health effects in time-series studies is that the age distribution as well as the smoking and occupational histories of the population are unlikely to change from day to day. Because of the design of time-series studies, however, it is not possible to determine whether the observed short-term effects are associated with the individuals who received the highest exposures.

Similar criteria that were used to select studies of the adverse health effects of ozone were used to select studies of the adverse health effects of PM. Once again, special attention was paid as to whether the effects of potential confounders such as co-pollutants, weather, and factors that change over time were controlled adequately. Additionally, in order to be useful in the health benefits assessment, the health outcomes needed to be those to which a dollar value could be assigned.

The health outcomes evaluated in the majority of these studies were mortality (including all-cause, respiratory, and cardiovascular) and hospital admissions or emergency room visits for respiratory and cardiac diseases. A few studies examined lung function, lung cancer, prevalence of respiratory symptoms, or new cases of airway obstructive disease and chronic bronchitis. We sought studies in which $PM_{2.5}$ or PM_{10} were used as measures of ambient PM, but we also looked at effects (presented in Table 5-8 as relative risks) for PM measures such as total suspended particulates (TSP) and black smoke (BS). Neither TSP nor BS include a measurement of particle size. TSP is measured using a high volume sampler that collects PM up to approximately 25-45 μm in diameter. BS is measured using the British black smoke filter method, which collects particles with diameters less than 4 μm with high efficiency and measures their light reflectance. Time-series studies that did not use the direct metrics of PM, but estimated PM exposure using TSP, BS, and other alternative measures, were included in the review to better assess the overall consistency of the results; however, these time-series studies were not used in the final analysis. In addition, we did not quantify health effects for specific PM components, such as sulfate.

Indicators of visibility such as coefficient of haze (COH), nephelometry (b_{sp}), and local visual distance (LVD) were used as indexes of PM in a number of the reviewed studies. Like BS, these indexes are optical measures and they do not directly measure PM mass. Mass can be estimated from the optical readings using a calibration curve. COH samplers have entry cut points of about 4.5 μm and measure transmission of light through particle-loaded filters. COH and BS measurements are greatly affected by airborne carbon particles. Nephelometry measures light scattering by particles of the size range 0.01-2 μm (Morgan et al., 1998), and LVD measures particles in the size range 0.1-2.5 μm (Voight et al., 1998). Because the

particle components associated with mortality and morbidity may be contained in the finer particles, the PM effects in Table 5-8 are provided for the finer particles when more than one measure of PM was evaluated in a study.

The studies of PM and health outcomes selected for use in the health benefits assessment are as follows. Two studies of indicators of acute morbidity included in the valuation are the study by Ostro and Rothschild (1989) of minor restricted activity days and the study by Ostro (1987) of restricted activity days. Other acute-effects studies of PM used in the assessment include those analyzing respiratory hospital admissions of the elderly (Schwartz, 1994a, 1994b, 1994c, 1996) and cardiovascular disease hospital admissions of the elderly (Schwartz and Morris, 1995). Further support for the association between PM exposure and respiratory hospital admissions of the elderly in the United States was provided by Schwartz (1995) and Schwartz et al. (1996). The study by Pope (1991) was used to estimate associations between respiratory hospital admissions for all ages and PM exposure. New cases of definite symptoms of chronic bronchitis were estimated from the long-term study Abbey et al. (1993). The association between PM exposure and increased risk of definite symptoms of chronic bronchitis was confirmed by Abbey et al. (1995).

Hiltermann et al., (1998) found significant associations between prevalence of asthma symptoms in adults and PM_{10}. These results were not quantified in the health benefits assessment. Several studies of the short-term effects of PM exposure found significant associations between mortality and $PM_{2.5}$, PM_{10} or TSP (Saldiva et al., 1995; Ito and Thurston, 1996; Ostro et al., 1996; Borja-Aburto et al., 1997, 1998). These studies appeared to be carefully conducted and their results were used directly to confirm the consistency of effects and dose-response relationships. The long-term prospective cohort study (Pope et al., 1995) provided highly reliable estimates of mortality responses for adults over 30 years of age. This study was used in the benefits analysis to quantify the relationship of $PM_{2.5}$ exposure and premature mortality in adults.

In a number of other studies that appeared to be well conducted, health effects of PM were assessed using a measure other than $PM_{2.5}$, PM_{10}, or TSP. We did not attempt to estimate $PM_{2.5}$ or PM_{10} levels from the alternative PM measures, or use these studies for developing dose-response relations for the health benefits assessment. These studies do, however, support the coherence and consistency of PM-induced health effects, including mortality. Findings included: significant effects of PM on respiratory hospital admissions in Europe (Dab et al., 1996; Anderson et al., 1997) and Australia (Simpson et al., 1997); an independent association of COH, but not $PM_{2.5}$ or PM_{10} with respiratory and cardiac hospital admissions (Burnett et al., 1997b); an effect of sulfate, but not $PM_{2.5}$, on respiratory hospital admissions (Thurston et al., 1994); and an association in southern California between sulfate, but not COH, and respiratory morbidity (Ostro et al., 1993). Also not included in the health benefits assessment were two mortality studies that reported significant effects of PM. One of these studies estimated $PM_{2.5}$ from b_{sp} (Morgan et al., 1998) and, in the other, the association was significant for BS but not for PM_{10} (Verhoeff et al., 1996).

Diminished confidence in some of the reviewed studies was due to unexplainable results between age groups (Pönkä et al., 1998), years (Delfino et al., 1997), seasons (Castellsague et al., 1995), or cities (Dockery et al., 1992), or if the PM results were of borderline significance (Zmirou et al., 1996; Hoek et al., 1997). In the study by Pönkä et al. (1998), significant associations were found between PM_{10} and total cardiovascular mortality for persons < 65 years of age but not for persons > 65 years. Castellsague et al. (1995) found a significant effect of BS on asthma emergency room visits in summer and not in winter, when the BS levels were slightly higher. Studies in which PM effects could not be distinguished clearly from those of co-pollutants also did not receive a high confidence rating. These include studies of mortality (Kinney et al., 1995; Moolgavkar et al., 1995; Sunyer et al., 1996; Kelsall et al., 1997), all respiratory hospital admissions (Moolgavkar et al., 1997; Spix et al., 1998), asthma hospital admissions (Delfino et al., 1994), and incident lung cancer (Beeson et al., 1998). Determination of an independent effect of PM on mortality in the meta-analysis by Spix et al. (1998) was hindered by the variety of PM measurement methods used.

Statistically nonsignificant relative risks for adverse health effects and PM exposure were reported in some of the reviewed studies. These include studies of respiratory emergency hospital admissions and BS (Ponce de Leon et al., 1996; Schouten et al., 1996) or $PM_{2.5}$ (Delfino et al., 1998); asthma emergency hospital admissions and BS (Sunyer et al.,1997) or TSP (Pönkä, 1991; Pönkä and Virtanen, 1996a; Stieb et al., 1996); cardiac and cerebrovascular hospital admissions and TSP (Pönkä and Virtanen, 1996b); and chronic bronchitis or emphysema hospital admissions and TSP (Pönkä and Virtanen, 1994). Negative confounding of BS by ozone was probable in the study by Ponce de Leon et al. (1996). The small number of daily hospital admissions may explain the lack of significant findings by Schouten et al. (1996), and temperature and ozone confounded the $PM_{2.5}$ effects on respiratory hospital admissions (Delfino et al., 1998).

Other studies were excluded from consideration for use in the health benefits assessment because responses for PM were not reported in a useful form (Sunyer et al., 1991; Burnett et al., 1997a; Voight et al., 1998). The study of house calls conducted in Paris (Medina et al., 1997) was excluded because the results were not applicable to the United States, and the study of lung function was not included in the health benefits assessment (Abbey et al., 1998) because of the difficulty in placing a dollar value on this health outcome.

As noted previously, the vast majority of the reviewed studies addressed the short-term effects of PM. Overall, these studies provide strong consistent evidence that acute exposure to PM increases risk of mortality and morbidity, especially of hospitalization for respiratory diseases in the elderly. Individuals aged 65 years and older represent a sensitive subpopulation to the harmful effects of air pollution. Based on the studies of the elderly in which we have the most confidence, relative risks for hospital admissions for all respiratory diseases for a 50 $\mu g/m^3$ increase in PM_{10} range from 1.06 to 1.10 (i.e., a 6 to 10 percent increase over the mean level of hospitalizations is estimated) (Schwartz, 1995, 1996; Schwartz et al., 1996). The range of relative risk estimates for hospital admissions for pneumonia is essentially the same as that for admissions for all respiratory diseases (1.06-1.09 per 50 $\mu g/m^3$ increase in PM_{10}) (Schwartz, 1994a, 1994b, 1994c). For chronic obstructive lung disease, the increase in admissions for a 50 $\mu g/m^3$ increase in PM_{10} is about twice that for pneumonia (relative risk, 1.12-1.25) (Schwartz, 1994a, 1994b, 1994c). The evidence for an acute adverse effect of PM

on hospital admissions for cardiovascular disease is weaker and the estimated effects are smaller than those for respiratory admissions. Only one reviewed study found a significant association between PM and cardiovascular disease hospital admissions. For a 32 $\mu g/m^3$ increase in PM_{10}, relative risks of 1.02 for ischemic heart disease admissions and 1.03 for congestive heart failure admissions were found (Schwartz and Morris, 1995). All of the foregoing short-term exposure studies took place in the United States. Their results are comparable in that the same primary investigator conducted all of the studies and similar analysis techniques were used. Nevertheless, the ability of relatively low levels of PM to adversely impact health is supported by air pollution studies conducted in foreign countries as well.

5.3.4 PM Effects on Children

Fifteen studies that addressed the acute health effects of PM on children were reviewed. The studies are summarized in **Table 5-10**. The study categories include daily life, daily time series, daily diary, panel, and summer camp studies. The health outcomes assessed in the studies are hospital admissions, respiratory symptoms, and lung function.

The following studies reported statistically significant associations between PM (PM_{10} in all but one study) and respiratory and allergic symptoms. Symptom rates for chronic cough, nocturnal dry cough, bronchitis, and conjunctivitis were associated with PM_{10} in a cross-sectional study conducted in 10 communities in Switzerland (Braun-Fahrlander et al., 1997). Incidence of cough and incidence of lower respiratory symptoms were associated with PM_{10} in a daily diary study conducted in six United States cities (Schwartz et al., 1994), and incidence of evening cough episodes was associated with $PM_{2.5}$ in a daily life study of nonasthmatic schoolchildren in Uniontown, Pennsylvania (Neas et al., 1995). The next four significant findings are from panel studies. PM_{10} was associated with increased symptom severity in asthmatic children in southern California (Delfino et al., 1998), with the prevalence of lower respiratory symptoms and bronchodilator use in asthmatic children in Amsterdam (Gielen et al., 1997), and with shortness of breath in African-American asthmatics in Los Angeles (Ostro et al., 1995). Finally, both $PM_{2.5}$ and PM_{10} were associated with increases in cough, phlegm, and lower respiratory illness in asthmatic children in Mexico City (Romieu et al., 1996).

Two studies found no significant relationship between PM and daily respiratory symptoms in schoolchildren. The study by Hoek and Brunekreef (1995) took place in two rural towns in the Netherlands, and the PM index was PM_{10}. Linn et al. (1996) conducted their study in three communities in southern California using PM_5 levels measured outside of schools. In a study of asthmatic children living southwest of Mexico City, the significant association of PM_{10} and difficulty breathing became nonsignificant when ozone, PM_{10}, and minimum temperature were included simultaneously in a model (Romieu et al., 1997).

Five of the six reviewed studies that tested the effects of PM on lung function found significant negative relationships. Decrements in forced vital capacity (FVC) and change from morning to afternoon forced expiratory volume in one second (FEV_1) were associated with PM_5 in schoolchildren in southern California even though symptoms were not associated (Linn

et al., 1996). Decreases in FVC and FEV_1 in one of the three panels of children at a summer camp in the Austrian Alps were associated with PM_{10} (Studnicka et al., 1995). The association was found for the panel for which the highest pollutant levels were recorded. A decrease in peak expiratory flow rate (PEFR) was associated with PM_{10} in schoolchildren in Uniontown, Pennsylvania (Neas et al., 1995). Associations were also found between morning or evening peak expiratory flow (PEF) and BS, but not PM_{10}, in asthmatic children in Amsterdam (Gielen et al., 1997). Lastly, decreases in PEFR were related to $PM_{2.5}$ and PM_{10} in asthmatic children in northern Mexico City (Romieu et al., 1996) but not in asthmatic children living southwest of Mexico City where the PM_{10} levels were much lower (Romieu et al., 1997).

Hospital admission of children was significantly associated with PM in two of the reviewed studies. Significant correlations were found between PM_{10} and respiratory admissions for preschool children in three mountain valleys in Utah (Pope, 1991) and between TSP and hospitalization for childhood asthma in Hong Kong (Tseng et al., 1992). No association was found for BS and contacts with the Copenhagen Emergency Medical Service for respiratory illnesses by children (Keiding et al., 1995) and for TSP and asthma hospital admissions in children living in Helsinki (Pönkä, 1991). A positive but insignificant association between BS and emergency admissions for asthma in children living in Barcelona, London, and Paris was found in a meta-analysis (Sunyer et al., 1997). The collinearity of pollutants NO_2 and BS may have limited separation of their effects in this latter study.

In summary, the results of these studies provide evidence that even relatively low levels of PM can cause adverse health effects in both asthmatic and nonasthmatic children. Significant increases in respiratory symptoms as well as decrements in lung function were found in the majority of the reviewed studies that tested for these effects. Associations between PM and hospital admissions for children with pneumonia, bronchitis, and asthma also have been shown.

5.4 SUMMARY

Section 5 summarizes the literature since the last NAAQS criteria documents were published for ozone and particulate matter, and briefly evaluates the potential health impacts of toxic air pollutants important for the Houston area. Although laboratory-based studies are discussed, the emphasis is on environmental epidemiology studies because these studies represent the 'real-world' exposures of people living in urban communities impacted by a complex of airborne contaminants.

Studies were evaluated with respect to suitability for estimating health benefits in this study based on: whether they had been peer-reviewed, if they dealt adequately with potential confounding factors (such as copollutants and meteorological variables), if the mix of pollutants was reasonably relevant to exposures in the Houston area, and if the socioeconomic factors and various ethnic characteristics were sufficiently similar.

Ozone: Ozone has been extensively evaluated in laboratory animal and human clinical studies, as well as in epidemiological studies. There is little doubt that ozone can damage lung

tissue, cause symptoms in children and adults and exacerbate diseases such as asthma and bronchitis. The role of ozone as an agent that causes (rather than exacerbates) lung or heart disease is less clear, despite the fact that some specific mechanisms by which ozone injures the lung could also be related to the development of disease. The association of ozone with human mortality is being investigated, but at this time the available evidence does not convincingly demonstrate this effect as clearly separated from those of other pollutants (especially fine particles). Approximately 60 epidemiology studies that investigated the effects of ozone on adult populations and about 40 studies that investigated ozone effects on children were evaluated for this analysis. We found sufficient evidence and were able to establish concentration-response functions for evaluating benefits related to: (a) chest symptoms (wheeze and phlegm production) in asthmatic children; and (b) symptoms such as eye irritation, chest congestion, throat irritation and headache in the overall population, and (c) cough in adults. Based on the available literature, ozone may also play a role in more serious health effects (as described for PM) but there is no well accepted way to disassociate the effects of ozone from those of its co-pollutants (primarily fine particle PM). To avoid mis-specifying and overcounting these possible ozone effects, they have not been called out specifically but may well be included in the effects of PM. However, based on the available data, the amount of an effect (i.e. mortality) attributed to PM that is actually due to ozone is small in comparison to the total pollutant-associated mortality.

Particulate Matter (PM): Airborne particles, for regulatory purposes are classified with respect to sizes. Fine particles are designated as those of 2.5 μm or smaller in aerodynamic diameter. Particles 10 μm or smaller in diameter are classified as PM_{10} (which includes the $PM_{2.5}$ class). In general there is a strong correlation between PM_{10} and $PM_{2.5}$ such that for some purposes it is possible to convert from one to the other by using appropriate ratios. Until recently, most air quality programs monitored PM_{10} on a more regular basis, but monitored $PM_{2.5}$ sporadically or not at all. Consequently there are many more environmental epidemiology studies that examine associations with PM_{10} than there are that examine $PM_{2.5}$. Although $PM_{2.5}$, as a measure of particulate air pollution, is thought to be more closely associated with human health effects than is PM_{10}, there are limitations in the data base that preclude one from making the assumption that all of the effects on human health associated with particle exposures are attributable to the $PM_{2.5}$ fraction. Thus, we have used the best available health-related studies and developed quantitative assessments for either PM_{10} or $PM_{2.5}$ (when the data were directly available) but then estimated the effect that might be due to $PM_{2.5}$ by adjusting the measured PM_{10} concentrations to approximate the $PM_{2.5}$ concentration. More than 50 published studies that examined the health effects of PM in adults and about 15 studies that examined PM health effects in children were reviewed. Most of the studies reported health outcomes in terms of relative risk or in terms of a concentration-response function that was associated with relative risk. The health outcomes for particles tended to be of a more chronic nature than did those for ozone. Valuations were made for the following health outcomes: (a) days of restricted activities because of pollutant-induced symptoms or illness; (b) minor restricted activity days, (c) cases of chronic bronchitis; (d) hospital admissions for respiratory and other relevant diseases; and (e) premature deaths.

Air Toxics: Three compounds that are listed as air toxics were present in the Harris County air samples at concentrations that exceeded currently accepted risk levels for cancer. Exposures to each of these three compounds (benzene, formaldehyde and 1.3-butadiene) are related to increases of specific types of cancers. The USEPA has tried, over the years, with limited success, to develop more definitive data sets with which to evaluate the potential health risks associated with ambient air toxics. At the present time, the available air monitoring data are quite limited and the number of suitable studies that provide credible estimates of concentration-response function are scant. The former limitation makes it difficult to assess the effects of future emission reductions on environmental exposures to air toxics and definitive air toxics reduction strategies are still to be developed. EPA has published some cursory estimates of the value of reducing air toxics exposures on a nation-wide basis. Using their estimates one could conclude that the benefits associated with air toxics reductions are small, however as better estimates of concentration-response function and total human exposure become available, this issue may need to be revisited.

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Table 5-1. Coherence of PM_{10} health effect measures.

	Percent change in health measure per each 10 $\mu g/m^3$ increase in PM_{10}
Increase in daily mortality	
Total deaths	1.0
Respiratory deaths	3.4
Cardiovascular deaths	1.4
Increase in hospital visits (respiratory)	
Admissions	0.8
Emergency Room	1.0
Exacerbation of asthma	
Attacks	3.0
Increased medication use	2.9
Emergency room visits*	3.4
Hospital Admissions	1.9
Increased reports of respiratory symptoms	
Lower respiratory	3.0
Upper respiratory	0.7
Cough	1.2
Impairment of lung function	
Decreased forced expiratory volume	0.15
Decreased peak expiratory flow	0.08

* One study only

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
California	Abbey et al., 1998	Nonsmokers ≥25 y	Lung Function	See Table 5-3	8-h Mean	PM_{10} SO_2 SO_4^{2-} TSP	Medium
California	Beeson et al., 1998	Nonsmokers ≥25 y	Incident Lung Cancer in Males	3.56 (1.35-9.42)/Annual Avg h > 100 ppb	Interquartile Range	PM_{10} SO_2 NO_2	Medium
Mexico City, Mexico	Borja-Aburto et al., 1998	All ages	Daily Cardiovascular Mortality	1.028 (1.000-1.055)/10 ppb	24-h Avg, Lag 1-2	$PM_{2.5}$ NO_2 NO_x NO SO_2	Medium
Toronto, Canada	Cakmak et al., 1998	All ages	Daily Respiratory Hospital Admissions	1.017-1.020, All Year 1.046-1.052, Summer	Interquartile Range, Lag 2	None	Medium
Montreal, Canada	Delfino et al., 1998a	≥65 y	Daily Respiratory ER Visits	1.187 (1.065-1.309)/44 ppb (1989) 1.218 (1.097-1.338)/38 ppb (1989)	1-h Max, Lag 1 8-h Max, Lag 1	$PM_{2.5}$	Medium
The Netherlands	Hiltermann et al., 1998	Non-Smoking Asthmatic Adults	Shortness of Breath Pain on Deep Inspiration Bronchodilator Use	1.18 (1.02-1.36)/50 ppb, Lag 0 1.44 (1.10-1.88)/50 ppb, Lag 0 1.16 (1.02-1.33)/25 ppb	8-h Max, Lag 0 8-h Max, Lag 0 Mean 7 Day	PM_{10} BS NO_2 SO_2	High

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Sydney, Australia	Morgan et al., 1998	All ages	Daily Total Mortality	1.02 (1.004-1.037)	10^{th} to 90^{th} Percentile 1-h Max, Lag 0	b_{sp} NO_2	Medium
			Daily Cardiovascular Mortality	1.025 (0.998-1.054)			
			Daily Respiratory Mortality	No association			
Helsinki, Finland	Pönkä et al., 1998	< 65 y	Daily Cardiovascular Mortality	1.099 (1.011 to 1.195)/10 ppb	24-h Avg, Lag 0	SO_2 NO_2 & NO CO PM_{10} TSP	Low
				-1.117 (-1.039 to -1.189)/10 ppb	24-h Avg, Lag 5		
London, Amsterdam, Rotterdam, and Paris	Spix et al., 1998	15-64 y	Daily Respiratory Hospital Admissions (mainly emergency) All Year	1.031 (1.013-1.049)/ 25 ppb for 15-64 y	8-h Mean	SO_2 BS TSP NO_2	Medium
		≥65 y		1.038 (1.018-1.058)/ 25 ppb for ≥65 y			
				1.019 (1.005-1.033)/ 25 ppb for 15-64 y	1-h Max		
				1.031 (1.015-1.047)/ 25 ppb for ≥65 y			
			Daily Respiratory Hospital Admissions Warm Season Only	1.04 (1.02-1.07)/ 25 ppb for ≥65 y	8-h Mean		
				1.04 (1.02-1.05)/ 25 ppb for ≥65 y	1-h Max		

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Latrobe Valley, Victoria, Australia	Voight et al., 1998	All ages	Daily Hospital Admissions for Asthma and COPD	No association	24-h Avg	SO_2 NO_2 LVD	Low
5 Central and Western Europe Cities	Anderson et al., 1997	All ages (48-70% ≥65 y)	Daily COPD Hospital Admissions (mainly emergency)	1.043 (1.022-1.065)/25 ppb 1.029 (1.011-1.047)/25 ppb	8-h Mean, Lag 1 1-h Max, Lag 1	SO_2 BS TSP NO_2	Medium
Mexico City, Mexico	Borja-Aburto et al., 1997	All ages	Daily Total Mortality	1.024 (1.011-1.039)/100 ppb No independent effect	1-h Max, Lag 0	SO_2 TSP CO	Medium
16 Canadian Cities	Burnett et al., 1997a	All ages	Daily Respiratory Hospital Admissions	1.031/30 ppb (t-ratio, 3.04), All Cities	1-h Max, Lag 1	SO_2 NO_2 CO COH	Medium
Toronto, Canada	Burnett et al., 1997b	All ages	Daily Respiratory Hospital Admissions	1.064 (t–ratio, 5.13), 3-d Avg and 1-d Lag 1.059 (t–ratio, 4.73), 3-d Avg and 1-d Lag, (Coregressed with NO_2 and SO_2)	Inter-Quartile Range for 12-h Mean (11.50 ppb)	SO_2 NO_2 PM_{10} CP $PM_{2.5}$ COH CO SO_4^{2-} H^+	Medium

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Toronto, Canada (continued)	Burnett et al., 1997b	All ages	Daily Cardiac Hospital Admissions	1.074 (t-ratio, 3.85), 3-d Avg and 2-d Lag 1.064 (t-ratio, 3.80), 3-d Avg and 2-d Lag (Coregressed with NO_2 and SO_2)	Inter-Quartile Range for 12-h Mean (11.50 ppb)	SO_2 NO_2 PM_{10} CP $PM_{2.5}$ COH CO SO_4^{2-} H^+	Medium
Montreal, Canada	Delfino et al., 1997	> 64 y	Daily Respiratory ER Visits	1.214 (1.084-1.343)/36 ppb (1993) 1.222 (1.091-1.354)/22 ppb (1993)	1-h Max, Lag 1 8-h Max, Lag 1	PM_{10} $PM_{2.5}$ SO_4^{2-} H^+	Medium
Mexico City, Mexico	Hernández-Garduño et al., 1997	All ages	Daily Respiratory Hospital Admissions	1.23 ± 0.09 No independent effect	Max/Mean 1-h Max, Lag 5	SO_2 NO_2 CO NO_x	Low
Rotterdam, the Netherlands	Hoek et al., 1997	All ages > 78 y	Daily Total Mortality	1.06 (1.01-1.11) 1.13 (1.06-1.20)	5^{th} to 95^{th} Percentile 24-h Avg, Lag 1	TSP Fe in TSP BS SO_2 CO	Medium
Halmstad, Sweden	Holmén et al., 1997	> 15 y	Daily Asthma ER Visits	RR not reported	24-h Avg	NO_2 SO_2 Toluene	Low

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Philadelphia, PA	Kelsall et al., 1997	All ages	Daily Total Mortality	1.023 (1.011-1.035)/20.2 ppb (Single-pollutant model) 1.020 (1.007-1.032) 20.2 ppb (Multipollutant model)	Inter-quartile Range 24-h Avg, Lag 1	TSP SO_2 NO_2 CO	Medium
Paris, France	Medina et al., 1997	All ages	Daily House Calls for Cardiovascular Conditions	1.10 (1.04-1.16)/ 49.5 ppb	5^{th} to 95^{th} Percentile 8-h Max, Lag 3	BS PM_{13} SO_2 NO_2	Medium
			Daily House Calls for Angina pectoris/MI	1.63 (1.10-2.41)/ 49.5 ppb	5^{th} to 95^{th} Percentile 8-h Max, Lag 3		
			Daily House Calls for Eye Conditions	1.17 (1.02-1.33)/ 49.5 ppb	5^{th} to 95^{th} Percentile 8-h Max, Lag 2		
Minneapolis-St. Paul, MN; Birmingham, AL	Moolgavkar et al., 1997	≥65 y	Daily Respiratory Hospital Admissions, Minneapolis-St. Paul	1.052 (1.024-1.079)/15 ppb	Interquartile Range 24-h Avg, Lag 1	PM_{10} SO_2 NO_2 CO	Medium

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Brisbane, Australia	Simpson et al., 1997	All ages ≥65 y All ages ≥65 y	Daily Total Mortality	1.016 (1.006-1.026/10 ppb 1.016 (1.004-1.028/10 ppb 1.024 (1.008-1.040)/10 ppb 1.024 (1.005-1.042)/10 ppb	1-h Max, Lag 0 8-h Mean, Lag 0	b_{sp} NO_2 SO_2	High
4 European Cities	Sunyer et al., 1997	Adults	Daily Asthma Emergency Admissions	No association	1-h Max	SO_2 BS NO_2	Low
7 European Cities	Touloumi et al., 1997	All ages	Daily Total Mortality	1.029 (1.01-1.049)/25 ppb (Single-pollutant model) 1.018 (1.009-1.027)/25 ppb (Coregressed with BS) 1.025 (1.015-1.034)/25 ppb (Coregressed with NO_2)	1-h Max	NO_2 BS	Medium

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Paris, France	Dab et al., 1996	All ages	Daily Respiratory Hospital Admissions Daily Respiratory Mortality	No association No association	1-h Max, 8-h Mean	BS SO_2 NO_2 PM_{13}	Medium
Cook County, IL	Ito and Thurston, 1996	All ages	Daily Total Mortality	1.07 (1.01-1.12)/ 100 ppb	24-h Avg, Avg of Lags 0 and 1	PM_{10} CO SO_2	Low
Santiago, Chile	Ostro et al., 1996	All ages	Daily Total Mortality	1.02 (1.00-1.05)/ 100 ppb for Summer No independent effect	1-h Max, Lag 1	PM_{10} NO_2 SO_2	Medium
London, United Kingdom	Ponce de Leon et al., 1996	All ages 15-64 y ≥65 y	Daily Respiratory Emergency Hospital Admissions	1.0483 (1.0246-1.0726)/29 ppb 1.0751 (1.0354-1.1163)/29 ppb 1.0616 (1.0243-1.1003)/29 ppb	10^{th}-90^{th} Percentile 8-h Mean, Lag 1 (Apr.-Sept.)	BS SO_2 NO_2	Medium
Helsinki, Finland	Pönkä and Virtanen, 1996a	≥15 y	Daily Asthma ER Hospital Admissions	No association	8-h Mean	SO_2 NO_2 TSP	Low
Helsinki, Finland	Pönkä and Virtanen, 1996b	All ages	Daily Ischemic Cardiac Hospital Admissions	1.10 (1.03-1.18)/log scale unit (about a 2.7-fold change)	24-h Avg, Lag 1	SO_2 NO_2 NO TSP	Low

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Amsterdam and Rotterdam, The Netherlands	Schouten et al., 1996	≥65 y	Daily Emergency Respiratory Hospital Admissions	1.272 (1.089-1.487)/50 ppb (Rotterdam, 1977-1981) 1.346 (1.133-1.598)/50 ppb (Rotterdam, Summer, 1977-1981)	1-h Max, Lag 2	SO_2 NO_2 BS	Low
Spokane, WA	Schwartz, 1996	≥65 y	Daily Respiratory Hospital Admissions	1.244 (1.002-1.544)/25 ppb	1-h Max, Lag 2	PM_{10}	Medium
Cleveland, OH	Schwartz et al., 1996	≥65 y	Daily Respiratory Hospital Admissions	1.09 (1.02-1.16)/ 50 ppb	1-h Max, Lag 2	PM_{10} SO_2	High
New Brunswick, Canada	Stieb et al., 1996	All ages	Daily Asthma ER Visits	1.33 (1.10-1.56)	1-h Max $> 95^{th}$ Percentile, Lag 2	SO_2 NO_2 SO_4^{2-} TSP	Medium
Barcelona, Spain	Sunyer et al., 1996	All ages	Daily Total Mortality, Summer Daily Mortality ≥70 y, Summer Daily Cardiovascular Mortality, Summer	1.058 (1.017-1.101)/50 ppb 1.059 (1.016-1.105)/50 ppb 1.088 (1.028-1.152)/50 ppb	1-h Max, Lag 0 1-h Max, Lag 1 1-h Max, Lag 1	BS SO_2 NO_2	Medium

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Amsterdam, the Netherlands	Verhoeff et al., 1996	All ages	Daily Total Mortality	1.049 (1.001-1.100)/50 ppb No independent effect	1-h Max	BS TSP PM_{10} SO_2 CO	Medium
Lyon, France	Zmirou et al., 1996	All ages	Daily Total Mortality	No association	1-h Max 24-h Avg	SO_2 PM_{13} NO_2	Low
Barcelona, Spain	Castellsague et al., 1995	< 65 y	Daily Asthma ER Visits	No association	1-h Max	SO_2 BS NO_2	Low
United Kingdom	Higgins et al., 1995	75 Adults (18-82 y) with Asthma or COPD	Wheeze, Reactors	1.50 (1.14-1.97)/ 5 ppb	24-h Avg, Lag 2	SO_2 NO_2	Low
			Dyspnea, All Subjects	1.20 (1.02-1.41)/ 5 ppb	24-h Avg, Lag 2		
			Dyspnea, Reactors	1.36 (1.08-1.72)/ 5 ppb	24-h Avg, Lag 2		
			Throat Symptoms, All Subjects	1.19 (1.01-1.40)/ 5 ppb	24-h Avg, Lag 1		
			Eye Symptoms, All Subjects	1.22 (1.03-1.46)/ 5 ppb	24-h Avg, Lag 1		
			Bronchodilator Use, All Subjects	1.29 (1.02-1.62)/ 5 ppb	24-h Avg, Lag 0		

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
United Kingdom (continued)	Higgins et al., 1995	75 Adults (18-82 y) with Asthma or COPD	Bronchodilator Use, All Subjects	1.44 (1.14-1.82)/ 5 ppb	24-h Avg, Lag 1	SO_2 NO_2	Low
			Bronchodilator Use, Reactors	1.44 (1.05-1.97)/ 5 ppb	24-h Avg, Lag 1		
Baton Rouge, LA	Jones et al., 1995	Adults	Daily Respiratory ER Visits	RR not reported	24-h Avg	None	Low
Los Angeles, CA	Kinney et al., 1995	All ages	Daily Total Mortality	1.02 (1.00-1.05)/ 143 ppb No independent effect	95^{th} percentile 1-h Max, Lag 1	PM_{10} CO	Low
Philadelphia, PA	Moolgavkar et al., 1995	All ages	Daily Total Mortality, Summer	1.15 (1.07-1.24)/ 100 ppb	24-h Avg, Lag 1	TSP SO_2	Medium
7 U.S. cities	Morris et al., 1995	Elderly	Daily Congestive Heart Failure Hospital Admissions	No association	1-h Max for cities, Lag 0	CO NO_2 SO_2	Medium
Sao Paulo, Brazil	Saldiva et al., 1995	≥65 y	Daily Total Mortality	Negative association	1-h Max 24-h Avg	PM_{10} SO_2 NO_x CO	Low
New Haven, CT	Schwartz, 1995	≥65 y	Daily Respiratory Hospital Admissions	1.07 (1.00-1.15)/ 25 ppb	24-h Avg, Lag 2	SO_2 PM_{10}	Medium
Tacoma, WA	Schwartz, 1995	≥65 y	Daily Respiratory Hospital Admissions	1.20 (1.06-1.37)/ 25 ppb	24-h Avg, Lag 2	SO_2 PM_{10}	Medium
Detroit, MI	Schwartz and Morris, 1995	≥65 y	Daily Cardiovascular Hospital Admissions	No association	1-h Max, Lag 0	PM_{10} SO_2 CO	Medium

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Central New Jersey	Weisel et al., 1995	All ages	Daily Asthma ER Visits	1.26 for ≥60 ppb	10:00 a.m. to 3:00 p.m. Mean, Lag 0	SO_4^{2-} NO_2 SO_2	Low
Ontario, Canada	Burnett et al., 1994	All ages	Daily Urgent or Emergency Hospital Respiratory Admissions	1.10 (1.06-1.14)/ 100 ppb	1-h Max, Lag 1	SO_4^{2-} SO_2 NO_2	Medium
Montreal, Canada	Delfino et al., 1994	All ages	Daily Urgent Respiratory Hospital Admissions	No association independent of temperature	8-h Max, Lag 4 (July/Aug.)	PM_{10} SO_4^{2-}	Low
Helsinki, Finland	Pönkä and Virtanen, 1994	All ages	Daily Chronic Bronchitis or Emphysema Hospital Admissions	No association	24-h Avg	SO_2 NO_2 TSP	Low
Birmingham, AL	Schwartz, 1994a	≥65 y	Daily Pneumonia or COPD Hospital Admissions	No association	24-h Avg or 1-h Max	PM_{10}	Medium
Detroit, MI	Schwartz, 1994b	≥65 y	Daily Pneumonia Hospital Admissions Daily COPD Hospital Admissions	1.026 (1.013-1.040)/ 5 ppb 1.028 (1.007-1.049)/ 5 ppb	24-h Avg, Lag 1	PM_{10}	Medium
Minneapolis-St. Paul, MN	Schwartz, 1994c	≥65 y	Daily Pneumonia Hospital Admissions Daily COPD Hospital Admissions	1.19 (1.02-1.40)/ 50 ppb No association	24-h Avg, Lag 1	PM_{10}	Medium

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Toronto, Ontario, Canada	Thurston et al., 1994	All ages	Daily Total Respiratory Hospital Admissions Daily Asthma Hospital Admissions	1.37 ± 0.15 (Max/Mean O_3) 1.31 ± 0.15 (Max/Mean O_3)	1-h Max, Lags not reported for these RRs	SO_4^{2-} H^+ $PM_{2.5}$ CP PM_{10} TSP SO_2 NO_2	Medium
California	Abbey et al., 1993	Nonsmokers ≥25 y	Development of Asthma (Males Only)	1.95 (1.00-3.94)/ 500 h	Hours Avg Annual Level in excess of > 100 ppb O_3	TSP SO_2	Low
Southern California	Ostro et al., 1993	Nonsmokers ≥18 y	Daily Upper Respiratory Tract Symptoms	1.13 (1.01-1.28)/ 100 ppb	7-h Mean, Lag 0	SO_4^{2-} COH NO_2 SO_2	Low
St. Louis, MO and Eastern Tennessee	Dockery et al., 1992	All ages	Daily Total Mortality	No association	24-h Avg	PM_{10} $PM_{2.5}$ SO_2 NO_2 SO_4^{2-} H^+	Medium
Tucson, AZ	Krzyzanowski et al., 1992	All ages	Peak Expiratory Flow Rates	See Table 5-3	8-h Max	PM_{10}	Medium

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI) or ± SE	Daily Ozone Exposure Metric	Co-Pollutants Considered	Confidence in Study
Buffalo, NY	Thurston et al., 1992	All ages	Daily Total Emergency Respiratory Hospital Admissions	1.22 ± 0.12 (Max/Mean O_3)	1-h Max, Lag 2	SO_4^{2-} H^+	Low
			Daily Asthma Hospital Admissions	1.29 ± 0.12 (Max/Mean O_3)	1-h Max, Lag 3		
New York City, NY	Thurston et al., 1992	All ages	Daily Total Emergency Respiratory Hospital Admissions	1.19 ± 0.04 (Max/Mean O_3)	1-h Max, Lag 3	SO_4^{2-} H^+	Low
			Daily Asthma Hospital Admissions	1.23 ± 0.10 (Max/Mean O_3)	1-h Max, Lag 1		
Helsinki, Finland	Pönkä, 1991	All ages	Daily Asthma Hospital Admissions	RRs not reported	8-h Mean	SO_2 NO NO_2 CO TSP	Low
Barcelona, Spain	Sunyer et al., 1991	> 35 y	Daily COPD ER Admissions	No conclusions possible	1-h Max	BS CO NO_2	Low
Vancouver, Canada	Bates et al., 1990	15-60 y > 60 y	Daily ER Visits	No conclusions possible	1-h Max	SO_2 NO_2 COH SO_4^{2-}	Low

Table 5-2. Summary of effect estimates for ozone in adults in recent studies.

Abbreviations:

1-h Max = daily maximum hourly level
8-h Max = daily maximum value for an 8-h moving average
8-h Mean = mean value for a fixed daily 8-h time period
24-h Avg = average value for a 24-h period
Avg = average
BS = black smoke
b_{sp} = scattering coefficient of particles measured by nephelometry
CI = confidence interval
CO = carbon monoxide
COH = coefficient of haze; an optical measure of airborne particulates
COPD = chronic obstructive pulmonary disease
CP = coarse particles (2.5 µm < aerodynamic diameter < 10 µm)
d = day
ER = emergency room
Fe = iron
h = hour
H^+ = hydrogen ion
Interquartile Range = 3rd minus 1st quartile
Lag = delay (in days) of the manifestation of the effect following exposure
LVD = local visual distance; a measure of particles of size range 0.1-2.5 µm
Max/Mean = RR calculated as the maximum O_3 level deviation from the mean, multiplied by its corresponding regression coefficient, divided by the mean hospital admission, plus one.
Mean 7-Day = mean concentration of the previous week
MI = myocardial infarction
NO = nitric oxide
NO_2 = nitrogen dioxide
NO_x = nitrogen oxides
No association = no association at $p < 0.05$.
O_3 = ozone
OR = odds ratio
p = probability
$PM_{2.5}$ = particulate matter ≤2.5 µm aerodynamic diameter
PM_{10} = particulate matter ≤10 µm aerodynamic diameter
PM_{13} = particulate matter ≤13 µm aerodynamic diameter
ppb = parts per billion (factor of 0.5 used to convert $\mu g/m^3$ to ppb)
RR = relative risk, given per unit increase in O_3 level
SE = standard error
SO_2 = sulfur dioxide
SO_4^{2-} = sulfate
t–ratio = ratio of log-linear regression coefficient to SE
TSP = total suspended particulates
y = year

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Abbey et al., 1998	Cohort	1977-1993	49 (Mean 8-h Mean) 17.6-73.8 (Range 8-h Mean)	Sex-specific multiple linear regressions adjusted for covariates. In single pollutant models, an increase of 23 ppb O_3 was associated with a 6.3% decrement in FEV_1 (as % of predicted) in males whose parents had asthma, bronchitis, emphysema, or hay fever.	No
Beeson et al., 1998	Cohort	1977-1992	26.2 ± 7.7 (Avg Annual Mean ± SD)	Used time-dependent, gender-specific Cox proportional hazards regression models adjusting for potential confounders. Single-pollutant models plus multipollutant analyses of all pairwise comparisons of annual avg number of h > 100 ppb O_3 and mean levels of the other 3 pollutants. PM_{10} and SO_2 associated with lung cancer in males and females.	No
Borja-Aburto et al., 1998	Time series	1993-1995	44.0 (Mean 24-h Avg) 4.1-127.1 (Range 24-h Avg)	Poisson regression employing a generalized additive model and controlling for temperature and periodic cycles. Accounted for autocorrelation. Considered lags. O_3 positively correlated with $PM_{2.5}$ in contrast to the typical negative correlations seen in Europe and the US. RR given in Table 5-2 is for 3-pollutant model. $PM_{2.5}$ consistently associated with mortality.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Cakmak et al., 1998	Time series	1981-1991	Not reported	Compared 4 temporal filtering techniques for time series analyses and found no important differences. Adjusted for long-wave cycles ≥1 mo, day of week, temperature, and dew point. Examined lag times. Quantitative details of collection of data used in the analysis not reported.	No
Delfino et al., 1998a	Time series	June-Aug., 1989-1990	44.1/66.4 (Mean/90th Percentile 1-h Max, 1989) 29.9/45.5 (Mean/90th Percentile 1-h Max, 1990) 37.5/58 (Mean/90th Percentile 8-h Max, 1989) 35.4/52.3 (Mean/90th Percentile 8-h Max, 1990)	Ordinary least squares regressions of the normally distributed filtered ER visit variables. Autoregressive parameters added if needed. Time series regressions controlled for season, day of week, autocorrelation, temperature, and RH. No significant correlations for the summer of 1990 after taking day of week into account. Reason for the difference in O_3 findings between the years was unclear.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Hiltermann et al., 1998	Panel	July 3 to Oct. 6, 1995	5.8-92.6 (8-h Moving Avg)	60 patients with intermittent to severe asthma studied using time series analysis. Medication and symptom daily prevalence data analyzed using a logistic regression model. Confounders considered: trends in symptom prevalence, medication use, mean morning and evening PEF, exposure to aeroallergens and environmental tobacco smoke, day of week, and temperature. O_3 was the most prominent air pollutant present and the one most strongly associated with symptom prevalences. No significant associations demonstrated between ΔPEF and O_3. Authors concluded that severity of asthma is not an indicator for sensitivity to air pollution.	No
Morgan et al., 1998	Time series	Jan. 1989 to Nov. 1993	24 (Mean 1-h Max) 3-102 (Range 1-h Max)	Poisson regression using GEE to allow for overdispersion and autocorrelation. Controlled for weather, season, day of the week, public holidays, and influenza epidemics using dummy variables. Lags examined. SO_2 levels negligible. PM (measured by nephelometry) showed strongest association with mortality. RRs given in Table 5-2 are for single-pollutant models. Few daily respiratory deaths. In multiple-pollutant models, effects of O_3 were marginally reduced but became nonsignificant.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Pönkä et al., 1998	Time series	1987-1993	9 (Median 24-h Avg) 1.5-25.5 (Range 24-h Avg)	Poisson regression controlled for temperature, RH, day of week, month, year, long-term trend, holidays, and influenza epidemics. Investigated lags. Single- and multi-pollutant models analyzed. PM_{10} also significantly associated with total and cardiovascular mortality. No control admissions category analyzed.	No
Spix et al., 1998	Meta-analysis	London, 1987-1991 Amsterdam, 1977-1989 Rotterdam, 1977-1989 Paris, 1987-1992	7 (Mean 8-h Mean) 34 (Mean 8-h Mean) 30 (Mean 8-h Mean) 10 (Mean 8-h Mean)	Each APHEA center performed Poisson time series regressions of their data controlling for trend, seasonality, calendar effects, unusual events, meteorology, and autoregression. Lags 0 or 1 most commonly used. For the meta-analysis, all days with pollution levels $> 200\ \mu g/m^3$ were deleted. Meta-analysis was performed using a pooled regression coefficient obtained as a weighted mean of local regression coefficients. A fixed-effects model was used when no heterogeneity was detected. Otherwise, a random-effects model was used and explanations for the heterogeneity was sought. The most consistent and strong finding was for O_3 and respiratory admissions. Different measurement methods of particles hindered analysis of their effects. No control admissions category analyzed.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Voight et al., 1998	Time series	1988	15.8 (Mean 24-h Avg) 3.05-82.0 (Range 24-h Avg)	Poisson distribution adjusted for day-of-week and seasonal variation. Asthma and all respiratory admissions correlated with pollutants individually and in combination. Lags 0 and 1 and autocorrelation examined. O_3 levels low. NO_2 and PM associated with COPD. No control admissions category analyzed.	No
Anderson et al., 1997	Meta-analysis	1977-1992 (dates varied among cities)	10-34.5 (Range Median 8-h Mean) (18-38.5 (Range Median 1-h Max)	As part of APHEA project, this prospective study used parametric Poisson regression controlling for trend, seasonal and other cycles, day of week, holidays, influenza epidemics, temperature, humidity, and autocorrelation. Individual city (Amsterdam, Barcelona, London, Paris, and Rotterdam) results combined. Single-pollutant models analyzed. The most consistent and significant findings were for O_3. Stronger effect of O_3 in warm season than in cold. No control admissions category analyzed.	No
Borja-Aburto et al., 1997	Time series	1990-1992	155 (Median 1-h Max) 25-285 (Range 1-h Max)	Various Poisson regression models all fit the mortality data well. Adding indicator variables for season, month, and day of week did not significantly reduce the variance. The model that used only temperature was chosen. Overdispersion and autocorrelation were accounted for. In multipollutant models, only TSP remained associated with mortality.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Burnett et al., 1997a	Time series	Apr. to Dec., 1981-1991	32.9/64 (Mean/95th Percentile 1-h Max All Cities)	Random effects relative risk regression model controlled for SO_2, NO_2, CO, COH, dew point temperature, and seasonal, subseasonal, and day-of-week effects. Fine PM and acid aerosol data not available. Lags examined. Considerable variation in O_3 RR among cities. O_3 association with respiratory hospitalizations eliminated in 2nd and 3rd largest cities after adjustment for CO and dew point temperature. No association of O_3 with admissions in winter.	No
Burnett et al., 1997b	Time series	Summers of 1992-1994	41.2 (Mean 1-h Max) 9-91 (Range 1-h Max)	Poisson regression adjusted for long-term trends, season, influenza epidemics, day of week, temperature, and dew point temperature. Compared two methods of removing temporal trends. GEE used to estimate log-relative risks. Lags examined. Only unscheduled admissions were considered, but no control admission category was analyzed. O_3 weakly correlated to all other pollutants except SO_4^{2-} (r = 0.53). O_3 was the least sensitive of the air pollutants to adjustment for gaseous and particulate pollutants. Strongest associations with both respiratory and cardiac diseases observed for COH and O_3. Association between O_3 and hospitalization stable over the 3 y of observation.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Delfino et al., 1997	Time series	June-Sept. 1992-1993	33.2 ± 12.6 (Mean 1-h Max ± SD, 1992) 79/49 (Max/90th Percentile 1-h Max, 1992) 36.2 ± 13.8 (Mean 1-h Max ± SD, 1993) 67/57 (Max/90th Percentile 1-h Max, 1993)	Separate analyses performed by year because yearly differences in means and day-of-week trends were found for most variables. Ordinary least squares regression and multiple regression analyses used. Controlled for temporal trends including day of week, weather, and autocorrelation. Lags examined. For 1992, no significant associations ($p < 0.02$) between air pollutants and ER visits were found. For 1993, no significant associations found with ER visits for patients 2-64 y of age. RR in Table 5-2 is for single-pollutant model. When O_3 was coregressed with $PM_{2.5}$, association remained significant; variance inflation was due to moderately strong covariation of the two. Effects for O_3 were larger than those of PM. Unclear why O_3 findings differed between years.	No
Hernández-Garduño et al., 1997	Time series	June 1992 to Jan. 1993	About 20-290 (1-h Max)	Ordinary least squares regression models controlled for long-wave trends, day of week, and autocorrelation. Autoregressive models used when necessary. TSP and PM_{10} not analyzed due to inconsistent availability. RR in Table 5-2 given for model that included temperature, lag 5. O_3 and NO_2 coefficients nonsignificant when both pollutants were included in same autoregressive model.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Hoek et al., 1997	Time series	1983-1991	13.5 (Median 24-h Avg) 0.5-34 (5^{th}–95^{th} Percentile 24-h Avg)	Poisson regression analysis using generalized additive models to adjust for long-term and seasonal trends, day of week, weather, and influenza. Lags examined. Conducted sensitivity analyses using different model specifications for seasonal trends. Significant associations between mortality and all pollutants except Fe were found when O_3 lag 1 was included in the model (O_3 negatively correlated with all other pollutants). O_3 and TSP associated most consistently with mortality. RRs are given in Table 5-2 for model that included TSP lag 1; no change when either SO_2 or CO was added to the model. O_3 RR remained stable after exclusion of all 24-h O_3 levels > 20 ppb. Most consistent association across seasons was O_3 lag 1.	No
Holmén et al., 1997	Time series	Jan. 1990 to May 1993	22.4 ± 8.9 (Mean 24-h Avg ± SD) 0.2/48.0 (Min/Max 24-h Avg)	Pearson correlation coefficient and *t*-test for analysis of linear relationship and differences between groups. ANOVA for analysis of effect of independent variables. O_3 levels significantly higher on days with 4-12 asthma visits than on those with 0-3 asthma visits. Temperature and air pollutants, as well as O_3 and NO_2, strongly correlated. No control admissions category analyzed.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Kelsall et al., 1997	Time series	1974-1988	19.8 (Mean 24-h Avg) 0-90.0 (Range 24-h Avg)	Age-stratified Poisson regression models developed using generalized additive models controlling for long-term trends, season, and weather. Models fitted by maximum likelihood with assumption of constant overdispersion over time. Lags examined. Correlations of O_3 with other pollutants were lower relative to correlations among the others. Investigated seasonal and age interactions with pollutant effects. Little evidence of seasonal effects and no overall evidence of modification of pollutant effects by age were found. Increasing TSP effect found with age. Strong independent effect of O_3 was shown that was consistent across seasons and various models that included other pollutants. TSP and SO_2 also significantly associated with mortality.	No
Medina et al., 1997	Time series	1991-1995	22 (Mean 1-h Max) 0.5-116 (Range 1-h Max) 17 (Mean 8-h Max) 0.5-93 (Range 8-h Max)	Poisson regressions using nonparametric smoothing functions (generalized additive model) controlled for time trend, season, pollen, influenza epidemics, and weather. Lags tested. Controlled for autocorrelation. O_3 weakly correlated with other pollutants; highest correlation was with SO_2. Single-pollutant models. No control admission category analyzed. House calls for headache significantly associated with all air pollutants but O_3.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Moolgavkar et al., 1997	Time series	1986-1991	26.2 (Mean 24-h Avg Minneapolis-St. Paul) 13.5-40.1 (10^{th}-90^{th} Percentile 24-h Avg Minneapolis-St. Paul) 25.1 (Mean 24-h Avg Birmingham) 13.5-37.6 (10^{th}-90^{th} Percentile 24-h Avg Birmingham)	Semiparametric Poisson regression models (generalized additive models) adjusted for temperature, day of week, season, and temporal trends. Examined lags. In Minneapolis, the strongest association with respiratory admissions was with O_3 when the 4 pollutants were regressed simultaneously. In Birmingham, no consistent association of respiratory admissions with any of the 3 available air pollutants was found (NO_2 levels not available and insufficient SO_2 data available). No control admission category analyzed.	No
Simpson et al., 1997	Time series	1987-1993	24.2 (Mean 1-h Max) 2.5-101.5 (Range 1-h Max) 18.1 (Mean 8-h Mean) 1.7-63.4 (Range 8-h Mean)	GEE analysis. Used autoregressive Poisson models and APHEA methods to control for weather, temporal trends, and interaction effects. Lags examined. Alternative approaches tested. Analyzed for effects of extreme events. Strong positive correlations in summer between O_3, NO_2, and b_{sp}. Absence of significant interaction effects between pollutants. No significant association between O_3 and mortality for < 65 y. Positive but insignificant associations found between O_3 and cardiovascular and respiratory mortality categories; low counts may have caused the insignificant association with respiratory mortality. PM also associated with mortality.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Sunyer et al., 1997	Meta-analysis	Barcelona, 1986-1989 Helsinki, 1987-1989 London, 1987-1991 Paris, 1987-1992	36 (3.5-141.5), Median (Range)1-h Max 13.5 (0.5-39) 20 (0.5-94) 18 (0.5-115)	As part of the APHEA project, Poisson regression models controlling for long wavelength patterns, seasonal patterns, day of week, weather, epidemics, and autocorrelation were built by each city. A combined estimate was computed using a fixed effects model except for O_3, which required a random effects model because heterogeneity of coefficients was present. Association of asthma with O_3 varied with cities and seasons possibly due to local variation in the O_3 levels. NO_2 was significantly associated with admissions. No control admissions category analyzed.	No
Touloumi et al., 1997	Meta-analysis	Athens, 1987-1991 Barcelona, 1986-1992 London, 1987-1991	46.9 (Mean 1-h Max) 36.2 (Mean 1-h Max) 20.6 (Mean 1-h Max) 23.0 (Mean 1-h Max)	Poisson autoregressive models allowing for overdispersion fitted to remove seasonal and long-term trends, day of week, and holidays, influenza epidemics, and other unusual events. For each of the 4 APHEA centers, the best lags were chosen. Correlation between O_3 and BS relatively low and in most cases negative. Significant positive associations found between daily deaths and both NO_2 and O_3, but NO_2 may be a surrogate of other pollutants. No significant difference between warm and cold seasons for either pollutant. In Table 5-2, RR for single-pollutant model is given for random effects	Yes

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Touloumi et al., 1997 (continued)				model; RRs for two-pollutant models are for fixed effects model. For meta-analysis of main effects of O_3 and NO_2, O_3 data were contributed by 7 cities (the 4 APHEA centers plus Amsterdam, Basel, and Zurich); RR for O_3 unchanged from that of single-pollutant model. No modification of O_3 effects by high or low levels of BS or NO_2.	
Dab et al., 1996	Time series	1987-1992	22.0 (Mean 1-h Max) 3.0-73.5 (5^{th}-99^{th} Percentile 1-h Max)	Followed APHEA protocol using an autoregressive Poisson model controlled for long-term trends, seasonal, weekly and daily patterns, weather, influenza, holidays, and strikes of medical staff. Lags examined. Emergency and planned hospital admissions not distinguishable. PM and SO_2 associated with respiratory hospital admissions and mortality. O_3 levels low.	No
Ito and Thurston, 1996	Time series	1985-1990	38.1 (Mean 24-h Avg) 17-65.6 (10^{th}-90^{th} Percentile 24-h Avg)	Poisson regression models controlled for long-wave cycles, day of week, and temperature. RR given in Table 5-2 for two-pollutant model. Unlike PM_{10}, O_3 showed no cause-specificity in mortality associations. Possible O_3-PM_{10} and O_3-temperature confounding.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Ostro et al., 1996	Time series	1989-1991	52.8 (Mean 1-h Max) 11-264 (Range 1-h Max)	Ordinary least squares regression and parametric tests used. Basic model (included only PM_{10}) controlled for temperature, day of week, month, and year. Additional controls for cyclical nature of total mortality counts and effects of temperature explored through several techniques. Other pollutants, unusual events, and lags examined. Autocorrelation corrections applied. High covariation between O_3 and PM_{10} led to nonsignificant associations with mortality when the two pollutants were coregressed in the summer model, but O_3 had the stronger association. O_3 not associated with mortality in winter. Of the pollutants examined, PM_{10} was associated most consistently with mortality.	No
Ponce de Leon et al., 1996	Time series	Apr. 1987 to Feb. 1992	15.6 ± 12 (Mean 8-h Mean ± SD) 2-37 (5^{th}–95^{th} Percentile 8-h Mean)	As part of the APHEA project, the following approach was followed: Poisson regression analysis adjusting for trend, seasonal and other cyclical factors, day of week, holidays, influenza epidemic, temperature, humidity, and autocorrelation. Lags examined. The most consistent association with respiratory admissions was with O_3. Warm season effects were larger than those for cool season. Evidence for a threshold at 50 ppb (8-h Mean). Very few significant associations found for other pollutants. O_3 coefficients little affected by controlling for other pollutants. No control admission category analyzed.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Pönkä and Virtanen, 1996a	Time series	1987-1989	11 (Mean 8-h Mean)	Poisson regression analysis adjusted for long-term trend, season, epidemics, day of week, holidays, temperature, and RH using protocol of APHEA project. Corrected for autocorrelation. Modeling was problematic. Significant associations seen with control admission category and O_3.	No
Pönkä and Virtanen, 1996b	Time series	1987-1989	11 ± 6.6 (Mean ± SD 1-h Max) 0-45 (Range 1-h Max)	Poisson regression models controlled for long-term trends, season, and day of week, and influenza epidemics with dummy variables. Weather controlled using a smoothing technique. Lags examined. Autocorrelation addressed. RRs in Table 5-2 given for model that included all 5 pollutants; association remained significant in one-pollutant model. Significant associations of ER ischemic cardiac admissions with NO and ER cerebrovascular admissions with NO_2. Temperature, RH, and SO_2 had no significant effects on admissions.	No
Schouten et al., 1996	Time series	1977-1989	79 (Mean 1-h Max, Amsterdam) 76 (Mean 1-h Max, Rotterdam)	Following APHEA approach, used autoregressive Poisson regression allowing for overdispersion and controlling for season, weather, and influenza epidemics. Lags investigated. Nonsignificant positive O_3 effect on respiratory emergency admissions in summer for ages ≥65 y in Amsterdam. Low daily number of emergency admissions may explain inconsistent results between the two cities. No control admission category analyzed.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Schwartz, 1996	Time series	1988-1990	79 (Mean 1-h Max) 58-107 (10^{th}-90^{th} Percentile 1-h Max, Apr.-Oct. only)	Poisson regression analysis (using the generalized additive model) controlled for long wavelength patterns, weather, and day of week. Lags considered. SO_2 levels trivial. Both PM_{10} and O_3 significantly associated with respiratory hospital admissions. Were too few days when both pollutants were measured to fit a two-pollutant model, but correlation between PM_{10} and O_3 was low (0.26). Examined sensitivity analyses to control for weather. No control admission category analyzed.	No
Schwartz et al., 1996	Time series	1988-1990	56 (Mean 1-h Max) 30-88 (10^{th}-90^{th} Percentile 1-h Max)	Analysis of Cleveland data was used as an example in a review paper of methodologies in time series studies. Long-term trends in the data were removed by including linear and quadratic terms in the model. Seasonality removed by including sinusoidal terms. Dummy variables for categories of temperature and RH and for day of week included. Individual pollutants then tested. O_3 and PM_{10} significantly associated with respiratory hospital admissions. No control admission category analyzed.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Stieb et al., 1996	Time series	1984-1992 (May-Sept.)	41.6 (Mean 1-h Max) 75 (95^{th} Percentile 1-h Max)	Daily ER visit frequencies filtered to remove day-of-week and long-wave trends. Filtered values regressed on air pollution and weather variables allowing for Poisson over- and underdispersion. Lags examined. First-order autocorrelation assessed. O_3 weakly and positively correlated with TSP and SO_4^{2-}. Only O_3 appeared to be related nonlinearly to ER visits. Single- and two-pollutant models used. Only O_3 exhibited a consistently positive association with asthma visits. Effect of O_3 not influenced by addition of co-pollutants into multivariate models, but a particulate effect could not be ruled out because of the limited number of sampling days for SO_4^{2-} and TSP. Subgroup analysis for O_3 effect on children not significant. No control ER visit category analyzed.	No
Sunyer et al., 1996	Time series	1985-1991	43.2 (Median 1-h Max) 4.8-141.8 (Range 1-h Max)	APHEA protocol followed using separate Poisson regression models for each pollutant and mortality category. Controlled for temperature, RH, year, season, day of week, influenza, and autocorrelation. Lags assessed. O_3 negatively correlated with BS. Association between O_3 and total mortality, mortality ≥70 y, and cardiovascular mortality significant for all the year, but stronger for summer. Positive but not significant association between O_3 and respiratory mortality. BS, SO_2, and NO_2 also related to mortality. CO, a possible confounder for the cardiovascular effect, not measured.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Verhoeff et al., 1996	Time series	1986-1992	21.5 (Mean 1-h Max) 8-81 (10^{th}-90^{th} Percentile 1-h Max)	Poisson regression analysis to control for seasonal and other long-term temporal patterns using indicators for year, month, day of week, influenza epidemics, and weather. Lags considered. Overdispersion and autocorrelation addressed. BS and PM_{10} were also positively associated with mortality. The associations of both PM_{10} and O_3 with mortality became nonsignificant when the two pollutants were coregressed. When BS and O_3 were coregressed, only the association of BS remained significant. The RR for O_3 in high-temperature months was nonsignificant. O_3 levels were relatively low.	No
Zmirou et al., 1996	Time series	1985-1990	7.6 (Mean 1-h Max) 0-76 (Range 1-h Max)	Followed Poisson time series regression protocol established by APHEA members including controls for long-wave seasonality, day of week, temperature, RH, influenza epidemics, and autocorrelation. Lags examined. SO_2 was only pollutant that showed a clear association with mortality. Daily mortality counts and measured O_3 levels were very low (the only O_3 monitoring station was located downtown, where NO emitted by traffic scavenges O_3).	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Castellsague et al., 1995	Time series	1985-1989	84.3 (Mean 1-h Max, Summers 1986-89) 56.6 (Mean 1-h Max, Winters 1986-89)	Poisson regression controlled for weather, long-term and shorter trends, day of week, autocorrelation and overdispersion. Analyses conducted separately for summers and winters. Lags examined. Pollutants analyzed individually because of colinearity. Small but significant association of PM and NO_2 with asthma ER visits. O_3 showed greater positive (but not significant) associations with asthma visits in winter than in summer although O_3 levels were higher in summer. No control admission category analyzed.	No
Higgins et al., 1995	Daily life	4 weeks	27.5 (Max 24-h Avg)	Subjects kept records of PEF measurements, symptoms, and bronchodilator use. Subjects classified as reactors (36) were those in whom a provocative dose of methacholine causing a 20% fall in FEV_1 was below 12.25 μmol. Multiple regression analysis was used to determine the relation between PEF and pollution levels; lags examined. The analysis of symptom and bronchodilator use was performed using multiple logistic regression, and the results for O_3 are given as odds ratios in Table 5-2. Falls in mean and minimum peak flow were significantly related to O_3 and SO_2. The study is described as exploratory in nature. Airborne PM not considered.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Jones et al., 1995	Time series	June 1990 to Aug. 1990	28.2 ± 11.7 (Mean 24-h Avg ± SD) 9.3-57.9 (Range 24-h Avg)	Separate time series analyses using multiple linear regression techniques were conducted for pediatric, adult, and geriatric ER visits. Day of week determined to be nonsignificant using ANOVA. Tested for autocorrelation. Significant association between O_3 and ER visits for adults 18-60 y but not for children or geriatrics. O_3 remained significant in simultaneous multiple linear regression with temperature, RH, mold spore counts, and pollen counts.	No
Kinney et al., 1995	Time series	1985-1990	70 ± 41 (Mean 1-h Max ± SD) 3-201 (Range 1-h Max)	Evaluation of methods used in controlling for temporal cycles and weather, examining effects of co-pollutants, and regression models. Sensitivity of RR for mortality/PM_{10} to the various methods and models was low, but data set was small (n = 364). RR in Table 5-2 given for single-pollutant model. O_3 not significant in two-pollutant model. PM_{10} effect appeared stronger than that of O_3 because the PM_{10} RR was the same whether PM_{10} was regressed singly or with O_3. CO also associated with mortality. PM_{10} and O_3 substantially correlated.	No
Moolgavkar et al., 1995	Time series	1973-1988	35.5 (Mean 24-h Avg, June-Aug.) 1.3-159 (Range 24-h Avg, June-Aug.)	Poisson regression. Controlled for effect of weather by analyzing seasons separately and including quintiles of temperature. Checked for autocorrelation. Investigated lags. RR given in Table 5-2 for 3-pollutant model (RR unchanged from single-pollutant model). SO_2 in spring and winter, but not TSP, also significantly associated with mortality.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Morris et al., 1995	Time series	1986-1989	39-75 (Mean 24-h Avg of 1-h Max, Each City)	Single-pollutant and multipollutant models with adjustments for temperature, season, and weekly cycles used to conduct negative binomial regression analyses. Autocorrelation addressed. Lags examined. CO showed consistent association with congestive heart failure admissions. Significant association of congestive heart failure admissions with an increase of 120 ppm O_3 only for Los Angeles in single-pollutant model. Other cities analyzed: Chicago, Philadelphia, New York, Detroit, Houston, Milwaukee. No control admission category examined.	No
Saldiva et al., 1995	Time Series	1990-1991	38.3 ± 29.7 (Mean 1-h Max ± SD) 12.5 ± 11.5 (Mean 24-h Avg ± SD)	Gaussian regression models controlling for month, temperature, RH, day of week, and autocorrelation. Lags examined. Pollutants analyzed individually and together. Various sensitivity analyses performed. The regression model was re-estimated using Poisson regression. Of the air pollutants studied, PM_{10} was the most strongly associated with mortality.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Schwartz, 1995	Time series	1988-1990	28.6 (Mean 24-h Avg), 15.8-45.4 (10^{th}-90^{th} Percentile 24-h Avg), New Haven 24.5 (Mean 24-h Avg), 13.3-55.7 (10^{th}-90^{th} Percentile 24-h Avg), Tacoma	Multiple regression analysis on temperature, dew point temperature, and air pollutants. A moving average filter removed seasonal and subseasonal patterns from the data. Poisson regression using single- and two-pollutant models. Autoregression examined. Strongest evidence for an independent association was for PM_{10} followed by O_3. RRs given in Table 5-2 are for the coregression on O_3 and PM_{10}. Although summer O_3 levels in Tacoma were 61% of those in New Haven and the number of respiratory admissions/day were about half that in New Haven, the RR for O_3 in Tacoma was greater and more stable than that for New Haven. No control admission category analyzed.	No
Schwartz and Morris, 1995	Time series	1986-1989	41 (Mean 1-h Max) 17-71 (10^{th}-90^{th} Percentile 1-h Max)	Poisson regressions controlled for seasonal and other long-term temporal trends, day of week, temperature, dew point temperature, and autoregression. Several sensitivity analyses to weather performed. Lags up to 2 days examined. Single- and two-pollutant models analyzed. PM_{10} and CO showed strongest associations with cardiovascular disease. No control admission category analyzed.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Weisel et al., 1995	Time series	Summers, 1986-1990	46-57 (Range of Yearly Means for 10:00 a.m. to 3:00 p.m.)	Forward stepwise multiple regression for each year showed only O_3 and temperature to be correlated with asthma visits; rate of temperature change, RH, SO_4^{2-}, NO_2, SO_2, and visibility not correlated. Autocorrelation assessed. Lags examined. Analysis of covariance conducted using an O_3 cutoff of 60 ppb and adjusted for temperature. Long-wave confounding not directly addressed. No control admission category analyzed.	No
Burnett et al., 1994	Time series	May-Aug. 1983-1988	About 50 (Mean 1-h Max, Summer)	Removed effects of day of week, season, and variations among hospitals. Addressed RH, temperature, lags, autocorrelation. Used Poisson regression and GEE. Single- and two-pollutant and stepwise regression models. Data from each of 168 hospitals assessed separately. SO_2 and NO_2 not analyzed because of too few monitoring stations and relatively low correlation with SO_4^{2-} and O_3. Both O_3 and SO_2 were significantly associated with respiratory admissions. O_3 stronger predictor than SO_4^{2-}.	No
Delfino et al., 1994	Time series	1984-1988	34.4 ± 15.4 (8-h Max ± SD, July-Aug.)	Regression analysis controlled for season, day of week, autocorrelation, temperature, and RH. Examined lags. Used single- and two-pollutant models. PM_{10} and SO_4^{2-} estimated from meteorological variables. Pollutants highly correlated. Relatively small sample size and low O_3 levels.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Pönkä and Virtanen, 1994	Time series	1987-1989	11 (Mean 24-h Avg) 0-45 (Range 24-h Avg)	Poisson regressions controlled for weather, season, time trends, day of week, and influenza. Autocorrelation assessed. Lags 0 to 7 days included. No significant association between all admissions and pollutants. Significant association of SO_2 with ER admissions for ages < 65 y and of NO_2 with hospital admissions for ages > 65 y. No control admission category analyzed. Correlations among predictor variables not given. Fairly low O_3 levels.	No
Schwartz, 1994a	Time series	1986-1989	25 ppb (Mean 24-h Avg) 14-37 (10^{th}-90^{th} Percentile 24-h Avg)	Poisson regression analysis controlling for time trends, seasonal fluctuations, and weather (but not day of week) using several alternative approaches. If serial correlation was found, autoregressive Poisson models were estimated using GEE. Used only single-pollutant models. PM_{10} was significantly associated with pneumonia and COPD admissions. No potentially confounding pollutants other than PM_{10} and no control admission category were analyzed.	No
Schwartz, 1994b	Time series	1986-1989	21 (Mean 24-h Avg) 7-36 (10^{th}-90^{th} Percentile 24-h Avg)	Regression analysis controlling for weather and long-term temporal and seasonal patterns (two methods compared), but not day of week. Autocorrelation tested for and, if present, autoregressive Poisson models estimated using	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Schwartz, 1994b (continued)				GEE. Lags considered. Both PM_{10} and O_3 significantly associated with pneumonia and COPD. RRs given in Table 5-2 for two-pollutant models. No potentially confounding pollutants other than PM_{10}, and no control admission category analyzed.	
Schwartz, 1994c	Time series	1986-1989	26 (Mean 24-h Avg) 11-41 (10^{th}-90^{th} Percentile 24-h Avg)	Poisson regression analysis controlled for time trends, season, and weather. Lags examined. Results not sensitive to any of several approaches examined to control for confounders. RR given in Table 5-2 for single-pollutant model. When both PM_{10} and O_3 were considered simultaneously, the O_3 effect became insignificant. No potentially confounding pollutants other than PM_{10} and no control admission category analyzed.	No
Thurston et al., 1994	Time series	July-Aug., 1986-1988	49.3/86.0 (Mean/Max 1-h Max, 1986) 53.4/111.0 (Mean/Max 1-h Max, 1987) 69.7/159.0 (Mean/Max 1-h Max, 1988)	Ordinary least squares regression analysis controlled for long-wave trends, day of week, and temperature. Lags and autocorrelation examined. Single- and two-pollutant models employed. Sensitivity analyses conducted for O_3 and H^+, the pollutants most strongly associated with respiratory admissions. RRs reported for peak pollution days only.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Abbey et al., 1993	Cohort	1977-1987	337 (Mean avg annual h in excess of 1 ppb) 966 (Max avg annual h in excess of 1 ppb)	Separate multiple logistic regression models used to study associations between long-term cumulative ambient concentrations of air pollutants and new cases of definite symptoms for airway obstructive disease, chronic bronchitis, and asthma. Used multivariate models to adjust simultaneously for a number of covariates. Ran stepwise regression analyses separately for each pollutant, then fit multi-pollutant models. Both TSP and O_3 appeared associated with new cases of asthma and change in asthma severity score, but the two pollutants were highly correlated.	No
Ostro et al., 1993	Time series	Sept. 1978 to Mar. 1979	98.6 ± 89.4 (Mean 1-h Max ± SD) 13.6-277.1 (Min/Max 1-h Max)	Multiple logistic regression of pooled data accounting for heterogeneity. Each pollutant modeled separately. Other covariates: sex, day of study, gas stove use, and presence of chronic respiratory disease. Odds ratio given in Table 5-2 is for model that included maximum temperature; the OR for upper respiratory symptoms was nonsignificant when maximum temperature was excluded. OR for lower respiratory symptoms became nonsignificant when maximum temperature was included in the model.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Dockery et al., 1992	Time series	Sept. 1985 to Aug. 1986	22.5/0-64 (Mean/Range 24-h Avg, St. Louis) 23/0-49 (Mean/Range 24-h Avg, Eastern Tennessee)	Poisson regression controlled for seasonal and weather effects. Examined lags. Models estimated by GEE. Single-pollutant models used. Power to detect associations limited because of short study period.	No
Thurston et al., 1992	Time series	1988-1989 Summers	75/164, 65/128 (Buffalo Mean/Max 1988, 1989, respectively) 69/206, 53/111 (New York City Mean/Max 1988, 1989, respectively)	Conducted separate least squares regressions of admissions on one pollutant plus temperature. Data prefiltered to remove long-wave cycles and day-of-week effects. Time lags considered. Autocorrelation examined. High temporal intercorrelations among pollutants prevented separation of the effects of the various pollutants.	No
Krzyzanowski et al., 1992	Daily life	May 1986 to Nov. 1988	46 ± 13 (Mean 8-h Max ± SD) 9-82 (Range 8-h Max)	Analyzed relationship of PEFR values to O_3 using a random-effects longitudinal model that included an autoregressive component. Analyzed day-to-day changes in PEFRs and symptoms in relation to O_3 using multifactorial ANCOVA. Examined lags. For adults, evening PEFRs were decreased in asthmatics who spent more time outdoors on days when O_3 was higher. Significant effects of interactions of O_3 with PM_{10} and temperature on daily PEFR found after adjustment for covariates.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Reference	Study Type	Study Period	O_3 Levels (ppb)	Statistical Analysis and Remarks	Included in Valuation
Krzyzanowski et al., 1992 (continued)				For all ages, allergic-irritant symptoms increased 7% with previous day O_3 > 56 ppb. The increase was much greater with interactions of O_3, maximum temperature, and PM_{10}. Methodological difficulties limit interpretation.	
Pönkä, 1991	Time series	1987-1989	11.0 ± 6.6 (Mean 8-h Mean ± SD) 0-45.0 (Range 8-h Mean)	Multiple regression analysis standardized for minimum temperature. In the step-wise analysis, NO and O_3 were the pollutants most strongly associated with admissions. Long-wave patterns and autocorrelation not examined.	No
Sunyer et al., 1991	Time series	1985-1986	31.6 (Mean 1-h Max) 0.75-126.5 (Range 1-h Max)	Multivariate linear regression models controlled for weather, season, day of week, and autocorrelation. Lags tested. Air pollutants analyzed separately because of their colinearity. O_3 not analyzed in a multivariate linear regression model because of its negative correlation with COPD admissions.	No
Bates et al., 1990	Time series	July 1984 to Nov. 1986	About 0-85 (1-h Max)	Controlled for day-of-week effects only. Lags examined. Calculated Pearson correlation coefficients between environmental variables and total visits, total respiratory visits, and asthma visits separately for summer and winter. Only total visits correlated with O_3 and temperature.	No

Table 5-3. Supporting information for studies of the effects of ozone on adults shown in Table 5-2.

Abbreviations:

1-h Max	=	daily maximum hourly level
8-h Max	=	daily maximum value for an 8-h moving average
8-h Mean	=	mean value for a fixed daily 8-h time period
24-h Avg	=	average value for a 24-h period
ANCOVA	=	analysis of covariance
ANOVA	=	analysis of variance
APHEA	=	Air Pollution and Health, a European Approach
Avg	=	average
BS	=	black smoke
b_{sp}	=	scattering coefficient of particles measured by nephelometry
CO	=	carbon monoxide
COH	=	coefficient of haze; an optical measure of airborne PM
COPD	=	chronic obstructive pulmonary disease
ER	=	emergency room
FEV_1	=	forced expiratory volume in one second
GEE	=	generalized estimating equations
h	=	hour
H^+	=	hydrogen ion
Min	=	minimum
Max	=	maximum
n	=	number of variates in the distribution
NO	=	nitric oxide
NO_2	=	nitrogen dioxide
O_3	=	ozone
OR	=	odds ratio
p	=	probability
PEF	=	peak expiratory flow
ΔPEF	=	morning to evening change in peak expiratory flow
PEFR	=	peak expiratory flow rate
PM	=	particulate matter
$PM_{2.5}$	=	particulate matter ≤2.5 μm aerodynamic diameter
PM_{10}	=	particulate matter ≤10 μm aerodynamic diameter
ppb	=	parts per billion
r	=	Pearson correlation coefficient
RH	=	relative humidity
RR	=	relative risk
SD	=	standard deviation
significant	=	$p < 0.05$ unless stated otherwise
SO_2	=	sulfur dioxide
SO_4^{2-}	=	sulfate
Time series	=	aggregate-level daily time series studies
TSP	=	total suspended PM

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect: Hospital Admissions	Category of Health Effect: Symptom Exacerbations	Category of Health Effect: Lung Function Changes	Association	Description and Results	Statistical Methods
Avol et al., 1998	Panel	About 20-200 ppb (range 1-h max)		✓	✓	– None	195 asthmatic, wheezy, or healthy children (10-12 y) studied in spring and summer, 4 d each season, 1994, Los Angeles, CA. Activity and symptom diaries used. Significant decrement (–45 ml) in FVC only for healthy children during high-O_3 exposures (> 100 ppb ambient, > 32.4 ppb personal sampler) in summer. Baseline lung function measurements similar among the 3 groups, O_3 levels low and similar in both seasons, data missing, and quality of exposure measurements questionable. No consistent effect of O_3 on symptoms or medication use.	Logistic regression for categorical variables. Linear model with individual intercepts for continuous variables. Multiple testing not adjusted for. Co-pollutants not considered.
Bates et al., 1990	Time series	About 0-85 ppb (range 1-h max)	✓			None	Emergency visits (About 25,500/mo) to 9 acute care hospitals in Vancouver, July 1984 to Oct. 1986. O_3, SO_2, NO_2, temperature analyzed. No correlation of respiratory visits with O_3. In winter and in all age groups (0-14, 15-59, 60 + y), all respiratory visits (but not asthma alone) correlated with SO_2.	Pearson correlation coefficients calculated between emergency visits and environmental variables. Day of week and lags examined.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Berry et al., 1991	Summer day camp	56-204 ppb (range1-h max)		✓		+	14 children < 14 y, central suburban New Jersey, 19 d in July 1988. Self-reported activity levels. Increase in symptoms with increasing O_3 level for cough, chest pain, runny or stuffy nose, eye irritation, shortness of breath. Exposure to < 120 ppb O_3 not significantly associated with symptoms.	Individual linear regression analysis and analysis of daily average measures for all subjects using 1-h or 8-h mean O_3 metrics.
					✓	None	Mean PEFR negative slope of borderline significance using 8-h mean O_3. Mean FEV_1 and FVC slopes positive. Presence of high O_3 levels during 1st 2 weeks possibly obscured dose-response relationship.	
Braun-Fahrlander et al., 1994	Exercise	40-157 $\mu g/m^3$ or 20-78 ppb (range ½-h mean)			✓	–	128 children, 9-11 y, Switzerland, May-Oct. 1989. Heavy exercise on cycle ergometer once/mo for 10 min during exposure to ambient air. O_3, NO_2, NO, temperature, RH measured. O_3 correlated significantly and positively with temperature and significantly and negatively with NO_2 and RH. Using common slope regression analysis, a significant negative association found for PEFR, but not for FVC or FEV_1.	Separate linear regressions determined for each subject for ΔFVC, ΔFEV_1, ΔPEF. Weighted mean regression coefficient calculated for each lung function index. Common slope regression analysis.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Braun-Fahrlander et al., 1997	Daily life	8.5-37.5 ppb (range annual ½ h means)		✓		None	4,470 children 6-15 y in 10 communities in Switzerland during school year 1992/1993. O_3, PM_{10}, NO_2, SO_2 evaluated. Respiratory and allergic symptoms assessed. Small positive association with number of h/y > 80 ppb O_3 and nocturnal dry cough, bronchitis, and conjunctivitis.	Logistic regression. Fog included in all models. Separate models for atopy. Random area effect models adjusted for autocorrelation.
Buchdahl et al., 1996	Time series	28-48 $\mu g/m^3$ or 14-24 ppb (range seasonal 24-h means)	✓			+	1025 children (≤16 y) attending emergency department at a London hospital for acute wheezing, Mar. 1, 1992 to Feb. 28, 1993. O_3, SO_2, NO_2, weather evaluated. Most significant association was with O_3; effect U-shaped.	Spearman's rank correlation coefficients. Poisson regression models adjusted for autocorrelation. Tested for non-linearity and lag effect.
Burnett et al., 1994	Time series	About 50 ppb in summer (mean 1-h max)	✓			+	Emergency or acute respiratory admissions to 168 hospitals in Ontario analyzed May-Aug. of 1983-88 for 0-1, 2-34, 35-64, and 65+ y. O_3 stronger predictor than SO_4^{2-}. For age 0-1 y, respiratory admissions significantly associated with O_3 (1- and 3-d lags) and SO_4^{2-} (1-d lag), but O_3 and SO_4^{2-} effects not separated.	Poisson regression techniques using GEE. Adjusted for season, day of week, hospital. Addressed RH, temperature, lags. Stepwise regression analysis.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect: Hospital Admissions	Category of Health Effect: Symptom Exacerbations	Category of Health Effect: Lung Function Changes	Association	Description and Results	Statistical Methods
Castillejos et al., 1992	Daily life	14-287 ppb (range 1-h mean) 29-82 ppb (range previous 48-h mean) 51-287 (range 1-h max)		✓	✓	– +	143 children (7-9 y), SW Mexico City, Jan.-June 1988. Children followed at 1-2 week intervals. No air pollutants other than O_3 measured. In univariate analyses, previous 48-h mean O_3 rise of 53 ppb (range of values) predicted a 2% decrement in FEV_1. Previous 48-h mean O_3 increase of 53 ppb associated with increased relative odds of 1.7 of cough/phlegm.	Linear regression for lung function. Multiple regression analyses included temperature and RH. Logistic regression for symptoms.
Cuijpers et al., 1994	Daily life	50-163 $\mu g/m^3$ or 25-82 ppb (range 8-h mean)		✓	✓	None	535 primary school children in the Netherlands, during summer smog episode July 8-16, 1991. O_3, SO_2, NO_2, BS measured. No increase in respiratory symptoms after exposure to moderately high O_3. Small inconsistent changes in lung function.	Simple linear regression analysis.
Delfino et al., 1996	Panel	14-87 ppb (range 12-h mean) 0-84.8 ppb (range personal 12-h day)		✓		+	12 asthmatics, 9-16 y, in San Diego, CA, Sept.-Oct. 1993. O_3, outdoor aeroallergens, $PM_{2.5}$, SO_4^{2-}, NO_3^-, H^+ measured. Personal O_3 increase of 25 ppb led to 25% increase in symptoms and 26% increase in inhaler use. Outdoor 12-h O_3, but not 1-h max O_3, associated with inhaler use ($p < 0.03$).	Random effects autoregression models controlled for weekend days and fungal spores.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Delfino et al., 1997	Time series	90th percentile < 60 ppb	✓			None	Emergency room visits for respiratory illnesses in Montreal, Canada, June-Sept. 1992/1993 (8,564 in 1992, 10,659 in 1993, all age groups: < 2, 2-18, 19-34, 35-64, > 65 y). Evaluated O_3, PM_{10}, $PM_{2.5}$, SO_4^{2-}, H^+. Significant association found in 1993 for elderly only. None of the pollutants significantly related to emergency room visits in 1992.	Ordinary least squares regressions after filtered emergency room visits shown to be normally distributed. Lags evaluated. Multiple regression analyses. Controlled for autocorrelation, weather, day of week.
Delfino et al., 1998b	Panel	52-135 ppb (range 1-h max) 44-110 ppb (range 8-h max)		✓		+	24 asthmatics ages 9-17 y, Alpine, CA, studied for 42-91 days, Aug. 1 to Oct. 30, 1995. O_3, PM_{10} evaluated. Subjects kept daily diary reports of asthma symptom severity. ORs significant for increased symptoms in single pollutant model but not in model that adjusted for PM. In less symptomatic asthmatics, ORs significant for increased symptoms in both models.	Regression analysis utilizing GEE. Confounders tested: day of week, temperature, RH, wind speed.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Gielen et al., 1997	Panel	27.5-110.8 $\mu g/m^3$ or 13.8-55.4 ppb (range 8-h max)		✓		+	61 asthmatics, 7-13 y (77% taking asthma medication), Amsterdam, the Netherlands, Apr. 26-July 4, 1995. O_3, PM_{10}, BS evaluated. Current day O_3 positively related to upper respiratory symptoms (runny/stuffed nose, sore throat).	Individual multiple linear regression controlled for pollen, time trend, day of week. Time series analysis corrected for autocorrelation.
					✓	–	Morning PEFR negatively associated with 2-d lag O_3 ($p < 0.05$); evening PEF negatively related with 2-day lag O_3 ($p < 0.10$).	
Hernández-Garduño et al., 1997	Time series	About 20-290 ppb (range 1-h max)	✓			None	Hospital visits for respiratory illnesses for patients ≤14 y, ≥15y, and all ages, June 1, 1992 to Jan. 31, 1993, northern Mexico City (24,113 for all ages). O_3, SO_2, NO_2, NO_x, RH, temperature monitored. PM levels not available consistently. Significant effect of air pollution on respiratory visits. O_3 effects not separated from NO_2 effects.	Ordinary least squares regression corrected for long-wave trends, day of week, and autocorrelation.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Higgins et al., 1990	Summer camp	20-245 ppb (range 1-h mean)			✓	–	3-week study in San Bernadino Mountains, CA, June-July 1987. 43 children ages 7-13 y; all but 3 attended only 1 week. O_3, NO_x, SO_2, PM_{10}, TSP measured. Average regression line slopes for individual slopes and for common slope significantly negative for FEV_1 and FVC (not PEFR) for various O_3 metrics. Using expanded common slope model, slopes for FEV_1, FVC, and PEFR significantly negative. FEV_1, FVC, and PEFR significantly negative with at least one of O_3 metrics for > 120 ppb O_3 during 6 h prior to testing.	Separate linear regressions for each subject and common slope regression analysis conducted with various O_3 metrics and lung function measures. Expanded common slope models included temperature, RH, fine and coarse PM. NO_2 and SO_2 levels low.
Hoek et al., 1993a	Exercise	50-236 $\mu g/m^3$ or 25-118 ppb (range 1-h max)			✓	None	83 children, 7-12 y, in the Netherlands. O_3, NO_x, $PM_{2.5}$, SO_4^{2-}, H^+, temperature, RH measured. PEFR evaluated before and after sports activities. Negative association of borderline significance ($p < 0.10$) found for previous-day O_3 and PEFR after training.	Day of week examined. Individual linear regression analysis adjusted for maximum temperature.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Hoek et al., 1993b	Daily life	7-237 $\mu g/m^3$ or 4-118 ppb (range 1-h max)			✓	–	533 children, 7-12 y, in 3 towns in the Netherlands, spring and summer, 1989. O_3, SO_2, NO_2, PM_{10}, H^+ measured. Most lung function tests in a.m. Significant negative association of previous-day maximum O_3 and pulmonary function. Addition of previous-day NO_2, SO_2, or PM_{10} or same-day PM_{10} caused no significant change in mean O_3 slope.	Individual linear regression. Multiple linear regression.
Hoek and Brunekreef, 1995	Panel	14-114 ppb (range 1-h max)		✓		None	300 children, 7-10 y, in 2 rural towns in the Netherlands, spring and summer 1989. O_3, SO_2, NO_2, PM_{10}, H^+, SO_4^{2-}, NO_3^- measured (SO_2, NO_2, H^+ low). Acute respiratory symptoms recorded by parents in a diary. No association of symptoms with same- or previous-day O_3, PM_{10}, SO_4^{2-}, or NO_3^-.	Logistic regression model that accounted for auto-correlation. Potential confounders considered: trends in symptom prevalence, day of week, weather variables.
Holmén et al., 1997	Time series	0.4-95.9 $\mu g/m^3$ or 0.2-48.0 ppb (range 24-h Avg)	✓			None	Emergency visits (4127) by asthmatics (< 15 and > 15 y) in Halmstad, Sweden, Jan. 1990 to May 1993. No effect of O_3 on visits by children. Effect significant for asthmatic adults at high levels of O_3.	Pearson correlation coefficient and *t*-test for analysis of linear relationship and differences between groups. ANOVA for analysis of effect of independent variables.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Jones et al., 1995	Time series	9.3-57.9 ppb (range 24-h Avg)	✓			None	Respiratory-related emergency visits (1265) in Baton Rouge, LA, June 1-Aug. 31, 1990, for ages 0-17 18-60, 61 + y. Significant association for adults only. No analysis of air pollutants other than O_3.	ANOVA for day of week effect. Multiple linear regression for temperature, RH, mold, pollen. Checked for autocorrelation.
Keiding et al., 1995	Time series	About 1-19 $\mu g/m^3$ or about 0.5-10 ppb (range 24-h avg)	✓			None	Contacts with Copenhagen Emergency Medical Service (3,974), Jan. 14 to Apr. 14, 1991, for respiratory illnesses by children 0-15 y old. O_3, CO, SO_2, NO_x, BS evaluated. Only NO and NO_x were associated significantly with contacts.	Weighted multiple regression analysis controlled for temperature, RH, holidays, weekends.
Kinney et al., 1996	Reana-lysis of 6 camp studies	53-123 ppb (range of averages of 1-h means from individual studies)			✓	–	Studies included 2 in NW New Jersey, 2 in Ontario, Canada, and 2 in southern California. Five of FEV_1 study-specific slopes were significantly negative as was the combined-studies slope; slope diminished by inclusion of time-trend variables. PEFR study-specific slopes more variable and combined studies slope not significant; appeared partially confounded by time-trends in PEFR.	Linear regression models that fit subject-specific intercepts. Single, pooled O_3 slope for each study separately and all studies combined. Analyses repeated after addition of time-trend variables.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Krzyzan-owski et al., 1992	Daily life	15-92 ppb (range 1-h max)			✓	–	287 children (6-15 y) and 532 nonsmoking adults in Tuscon, AZ, May 1986-Nov. 1988. O_3, PM_{10} evaluated. In children, noon PEFRs lower on days with higher O_3; effect increased in those with asthma. Effect on evening PEFRs seen only in asthmatic children.	Regression analyses. Random effects model that accounted for autorcorrelation. Multifactorial analysis of covariance.
				✓		+	Significant increase in symptoms associated with prolonged exposure to O_3.	
Linn et al., 1996	Daily life	3-53 ppb (range 24-h avg)			✓	–	269 children during 4th and 5th grade school years in 3 southern California communities, 1992-93 and 1993-94. O_3, NO_2, PM_5, temperature evaluated. Lung function and symptoms measured twice daily for 1 week each in fall, winter, and spring; time-activity and personal exposure data collected as well. FEV_1 p.m.-a.m. for current 24 h only lung function measure significantly negatively related with O_3. PM was most strongly associated with lung dysfunction.	Repeated-measures ANOVA was the primary analytic tool. Tested only relatively simple ANOVA/ regression models that included 1 or 2 environmental variables, year, season, year-by-season interaction, and day-of-week.
				✓		None	No tested symptom/pollutant relationship was significant.	

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Neas et al., 1995	Daily life	87.5 ppb (mean 12-h max) 37.2 ppb (mean 12-h mean)		 ✓	✓	– +	83 4th and 5th graders in Uniontown, PA, summer 1990. O_3, SO_2, PM_{10}, $PM_{2.1}$, SO_4^{2-}, H^+, temperature examined. Significant negative association of O_3 with PEFR decline. Association between incidence of evening cough episodes significant only when O_3 level was weighted by proportion of h spent outdoors.	Autogressive linear regression model for mean deviation in PEFR. Autoregressive logistic regression model using a modified GEE approach for symptoms.
Ostro et al., 1995	Panel	10-160 ppb (range 1-h max)		✓		+	83 African-American asthmatics, 7-12 y, Los Angeles, CA, 13 weeks in summer 1992. O_3, PM_{10}, NO_2, SO_2, pollens, molds, weather examined. O_3 associated with increased shortness of breath.	Stepwise regression for group-level and pooled analyses. Pooled logistic regressions based on GEE. Individual regressions.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Ponce de Leon et al., 1996	Time series	2-37 ppb (5^{th} to 95^{th} percentile 8-h mean)	✓			None	Admissions for respiratory disease to London hospitals, Apr. 1987 to Feb. 1992, for all ages (125.7/d), 0-14, 15-64, 65+ y. O_3, BS, SO_2, NO_2, lags evaluated. Most consistent association was with O_3. Not significant for children. Greatest effect on 15-64 y, followed by > 64 y, and then 0-14 y. Difficulty modeling children.	Poisson regression analysis adjusted for trend, seasonal and other cyclical factors, day of week, holidays, influenza, temperature, RH, autocorrelation.
Pönkä, 1991	Time series	0-89.9 $\mu g/m^3$ or 0-45.0 ppb (range 8-h mean)	✓			+	Admissions (4,209) for asthma attacks in Helsinki, 1987 through 1989, for 0-14, 15-64, 65+ y. Evaluated O_3, SO_2, NO_2, NO, CO, TSP, temperature, wind speed, RH, lags. For 0-14 y, significant correlation with O_3 for all admissions for asthma and for emergency ward admissions for asthma.	Regression analysis. Correlations calculated separately for weekdays and weekends. Stepwise regression.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Pönkä and Virtanen, 1996a	Time series	19-41 $\mu g/m^3$ or 10-20 ppb (range 8-h mean during different seasons)	✓			+	Emergency ward admissions (2,421) of asthmatics, 1987-89 in Helsinki, for 0-14, 15-64, 65+ y. O_3, SO_2, NO_2, TSP evaluated. Same data included in analysis by Pönkä, 1991. Positive association with O_3 for children < 15 y. Modeling was problematic.	Poisson regression analysis adjusted for long-term trend, season, epidemics, day of week, holidays, temperature, and RH using protocol of APHEA project. Corrected for autocorrelation.
Romieu et al., 1995	Time series	10-250 ppb (range 1-h max)	✓			+	Emergency visits in Mexico City for asthmatics < 16 y, Jan-June 1990. Evaluated O_3, NO_2, SO_2. O_3 correlated with NO_2, but not with SO_2. O_3 and SO_2 significantly associated with number of emergency visits for asthma.	Poisson regression model adjusted for day of week, period of year, minimum temperature, SO_2, age, sex. Looked for autocorrelation.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect: Hospital Admissions	Category of Health Effect: Symptom Exacerbations	Category of Health Effect: Lung Function Changes	Association	Description and Results	Statistical Methods
Romieu et al., 1996	Panel	40-370 ppb (range 1-h max)		✓		+	71 asthmatics, 5-13 y, in northern Mexico City, Apr. 24 to July 7, 1991 and Nov. 1, 1991 to Feb. 28, 1992. None taking medication regularly. O_3, PM_{10}, $PM_{2.5}$, NO_2, SO_2, measured. Significant positive association with same day cough, phlegm production, wheezing, or difficulty breathing. Comparisons across O_3 quartiles given for PEFR and respiratory symptoms.	Autoregressive logistic-regression models or GEEs used for symptom analysis. Autoregressive linear-regression models or GEEs adjusted for minimum temperature and multivariate models used for PEFR analysis.
					✓	None	Negative association with ΔPEFR not significant.	
Romieu et al., 1997	Panel	40-390 ppb (range 1-h max)		✓		+	65 asthmatics, 5-13 y, SW of Mexico City, Apr. 24 to July 7, 1991 and Nov. 1, 1991 to Feb. 28, 1992. None taking medication regularly. O_3, NO_2, SO_2 measured. PM_{10} and O_3 evaluated. Positive association with increase in cough, phlegm, and lower respiratory tract illness in asthmatics.	Autoregressive logistic-regression models or GEEs used for symptom analysis. Autoregressive linear-regression models or GEEs adjusted for minimum temperature and multivariate models used for PEFR analysis.
					✓	–	Negative association with evening PEFR on current day and 1-d lag in asthmatics.	

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect: Hospital Admissions	Category of Health Effect: Symptom Exacerbations	Category of Health Effect: Lung Function Changes	Association	Description and Results	Statistical Methods
Schwartz et al., 1994	Diary	23-54.1 ppb (10^{th}-90^{th} percentile 24-h avg)		✓		+	1,844 children grades 2-5 in 6 cities studied for 1 y starting Sept. 1984. Analyses limited to Apr.-Aug. O_3, SO_2, NO_2, PM_{10}, $PM_{2.5}$, SO_4^{2-}, H^+, temperature evaluated. O_3 and PM_{10} independently and significantly associated with cough incidence; PM_{10} associated somewhat more strongly.	Incidence of respiratory symptoms regressed separately against each pollutant controlling for previous day temperature, day of week, city of residence. Significant pollutants analyzed in multiple pollutant models.
Spektor et al., 1991	Summer camp	40-150 ppb (range 1-h max)			✓	–	46 healthy children (8-14 y) in SW New Jersey, 1988, who produced at least 7 d of acceptable data. O_3, H^+ evaluated. Regressions of p.m. lung function on previous-h O_3, avg O_3 for day, and 1-h max O_3 significantly negative in most cases. Regressions of p.m.-a.m. lung function on the O_3 periods significantly negative. In subset of 35 subjects with ≥2 consecutive days of measurements, regressions of a.m. lung function on previous day mean or maximum O_3 significantly negative, suggesting possible carryover.	Linear regression. No models were fit that included O_3, THI, H^+ simultaneously, nor were interaction effects tested for.
				✓		None	No consistent associations of symptoms with O_3 or H^+.	

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect: Hospital Admissions	Category of Health Effect: Symptom Exacerbations	Category of Health Effect: Lung Function Changes	Association	Description and Results	Statistical Methods
Stern et al., 1994	Daily life	46.3-115.0 ppb (annual mean to 99th percentile 1-h max, Ontario); 34.1-57.0 ppb (annual mean to 99th percentile 1-h max, Saskatchewan)			✓	–	> 3300 children, 7-10 y, residing in 10 rural Canadian towns, studied Sept. 1985 to Mar. 1986 by questionnaire and one spirometric assessment/child. Central Saskatchewan = low exposure region; SW Ontario = moderate exposure region. O_3, SO_4^{2-}, PM_{10}, particulate NO_3^-, SO_2, NO_2 measured. Significant negative decrements in FVC and FEV_1 in SW Ontario children compared to Saskatchewan children.	Mixed effects regression model. Community represented random effect. Fixed effect covariates controlled for age, sex, parental smoking, gas cooking, parental education (symptoms only), standing height, weight (lung function only.)
				✓		None	No change in cough, phlegm production, wheeze.	
Studnicka et al., 1995	Summer camp	45.4-56.4 ppb (range mean ½-h max for the 3 panels)			✓	–	133 children ≥7 y old, each studied for 3 weeks, Austrian Alps. 3 panels, June 28 to Aug. 28, 1991. O_3, H^+, SO_4^{2-}, NH_4^+, PM_{10}, temperature, RH, pollen measured. Daily spirometry, symptoms, exposure recorded. For panel 1 (highest daily maximum O_3), significant negative association between O_3 and FEV_1, FVC, and PEFR.	Linear regression analyses for repeated measures adjusted for sex, height, age. Included correction for within-subject correlation.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Sunyer et al., 1997	Time series	7-283 $\mu g/m^3$ or 4-142 ppb (range 1-h max)	✓			None	Asthma admissions (50.3/d) in 4 European cities, 1986-92, for ages < 15 and 15-64 y. Emergency admissions for 3 cities. O_3, SO_2, NO_2, BS evaluated. No association for O_3 in children.	Poisson regression models (APHEA project approach). Combined associations estimated using meta-analysis.
Thurston et al., 1997	Summer camp	20-160 ppb (range 1-h max)			✓	–	52, 58, 56 children with asthma (mostly moderate to severe), ages 7-13, studied in Connecticut during last week of June 1991, 1992, and 1993 respectively. O_3, $PM_{2.5}$, H^+, SO_4^{2-}, pollen, weather evaluated. Significant negative association with a.m. to p.m. ΔPEFR.	Poisson modeling approach for analysis of symptoms and medication use. Linear regression for analysis of ΔPEFR. Autocorrelation assessed in each model.
				✓		+	Significant positive association with β-agonist use, chest symptoms.	
Tseng et al., 1992	Time series	11-48 $\mu g/m^3$ or 6-24 ppb (range quarterly means of mean monthly data)	✓			None	Quarterly hospital discharges (33,953) for asthma in Hong Kong, 1983-1989. Age groups 0-1, 1-4, 4-14, 15 + y. Examined O_3, TSP, SO_2, NO_2, NO_x. No association of discharges with O_3; significant correlation for ages 1-4 and TSP.	Univariate and stepwise multiple regression analysis. Controls lacking for day of week, season, long-wave periodicities.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Symptom Exacerbations	Lung Function Changes			
Ulmer et al., 1997	Daily life	22.5-89.8 ppb (5^{th} to 95^{th} percentile ½-h mean)			✓	-	135 children (8-10.7 y) showing no sensitization to grass or tree pollens, 2 small towns in SW Germany. No air pollutants other than O_3 measured. Lung function tests conducted on 4 occasions (Apr., June, Aug., Sept., 1994). Cross-sectional analysis showed significant negative association with FVC only in June (highest O_3 exposure). Longitudinal analysis showed significant negative correlation with FVC and FEV_1 for children from town with higher O_3 levels.	Models adjusted for national origin, gender, passive smoke exposure, and included a dummy variable for each of the 4 occasions. Time-dependent covariates included.

Table 5-4. Summary of studies relating ozone levels with health effects on children.

Reference	Study Type	Daily O_3 Level	Category of Health Effect: Hospital Admissions	Category of Health Effect: Symptom Exacerbations	Category of Health Effect: Lung Function Changes	Association	Description and Results	Statistical Methods
White et al., 1994	Time series	15-163 ppb (range 1-h max)	✓			+	Emergency visits for asthma or reactive airway disease by 543 Atlanta blacks (1-16 y), June 1 to Aug. 31, 1990. O_3, PM_{10}, SO_2 evaluated. SO_2 effect small. Significant increase in asthma visits following afternoons with maximum 1-h O_3 ≥110 ppb. No dose-response relationship at < 110 ppb O_3.	Poisson regression model adjusted for minimum temperature, day of week, PM_{10}. Autocorrelation and pollen level addressed. One Poisson model fitted using GEE.

✓ Indicates which category was studied.
\+ Indicates that the association between the health effect and O_3 exposure was significantly positive.
– Indicates that the association between the health effect and O_3 exposure was significantly negative.

Abbreviations:

24-h avg = average value for a 24-h period
½-h max = daily ½ h maximum
1-h max = daily 1-hour maximum
8-h max = maximum value for a daily 8-h time period
12-h max = maximum value for a daily 12-h time period
½-h mean = daily ½-h mean
1-h mean = daily 1-h mean
8-h mean = mean value for a daily 8-h time period
12-h mean = mean value for a daily 12-h time period
48-h mean = mean value for a 48-h period
ANOVA = analysis of variance
APHEA = Air Pollution and Health, a European Approach
Avg = average
BS = black smoke
CO = carbon monoxide
D = day
FEV_1 = forced expiratory volume in one second
ΔFEV_1 = change in forced expiratory volume in one second
FVC = forced vital capacity
ΔFVC = change in forced vital capacity
GEE = general estimating equations
h = hour
H^+ = hydrogen ion
lag = number of days (time lag) between exposure and effect
max = maximum
min = minute
mo = month
NH_4^+ = ammonium ion
NO = nitric oxide
NO_2 = nitrogen dioxide
NO_3^- = nitrate
NO_x = oxides of nitrogen
O_3 = ozone
OR = odds ratio
p = probability
PEFR = peak expiratory flow rate
ΔPEFR = change in peak expiratory flow rate
personal = personal O_3 exposures measured with passive samplers worn by subjects
PM = particulate matter
$PM_{2.1}$ = particulate matter ≤2.1 μm aerodynamic diameter
$PM_{2.5}$ = particulate matter ≤2.5 μm aerodynamic diameter
PM_5 = particulate matter ≤5 μm aerodynamic diameter
PM_{10} = particulate matter ≤10 μm aerodynamic diameter
ppb = parts per billion; $\mu g/m^3$ were converted to ppb by multiplying by ½
RH = relative humidity
significant = $p < 0.05$
SO_2 = sulfur dioxide
SO_4^{2-} = sulfate
THI = temperature-humidity index
time series = aggregate-level daily time series studies
TSP = total suspended particulate matter
y = year

Table 5-5. Effects of ozone on respiratory symptoms in children.

Reference	Symptom	Measure Odds Ratio (95% CI)	O_3 Metric	Data Analysis and Remarks
Castillejos et al., 1992	Cough or phlegm	1.88 (1.07-3.30) per 53 ppb	Increase from lowest to highest levels 48-h before spirometry	Adjusted for epidemic effects and total number of times the child reported cough or phlegm.
Delfino et al., 1998b	Asthma symptoms	1.54 (1.02-2.33) per increase to 90th percentile O_3	Current day 1-h max	$P < 0.05$. Single pollutant regression model adjusted for day of week, maximum temperature. O_3 range (minimum to 90th percentile) = 58 ppb.
		1.42 (1.00-2.00) per increase to 90th percentile O_3	Current day 8-h max	$P < 0.05$. Single pollutant regression model adjusted for day of week, maximum temperature. O_3 range (minimum to 90th percentile) = 46 ppb.
		2.15 (1.04-4.44) per increase to 90th percentile O_3	Current day 1-h max	Less symptomatic asthmatics. $P < 0.05$. Single pollutant regression model; weather was not a confounder. O_3 range (minimum to 90th percentile) = 58 ppb.
Neas et al., 1995	Evening cough	2.20 (1.02-4.75) per 30 ppb	Preceding 12-h mean	Autoregressive logistic regression model using a modified GEE approach. O_3 weighted by proportion of hours spent outdoors during previous 12 h.
Ostro et al., 1995	Shortness of breath	1.40 (1.14-1.72) per 80 ppb	Current day mean	$P < 0.001$. Pooled analysis using multiple logistic regression based on GEE. Respiratory infection on previous day was included as an explanatory variable.

Table 5-5. Effects of ozone on respiratory symptoms in children.

Reference	Symptom	Measure Odds Ratio (95% CI)	O_3 Metric	Data Analysis and Remarks
Romieu et al., 1996	Cough	1.11 (1.05-1.18) per 50 ppb	1-h max, lag 0	OR estimated using GEE models and adjusted for minimum temperature.
		1.12 (1.06-1.19) per 50 ppb	1-h max, lag 1	
	Difficulty breathing	1.10 (1.00-1.22) per 50 ppb	1-h max, lag 0	
	LRI	1.09 (1.03-1.15) per 50 ppb	1-h max, lag 0	
	Cough	1.08 (1.00-1.15) per 50 ppb	1-h max, lag 0	OR estimated using GEE multivariate models in which O_3 and $PM_{2.5}$ and minimum temperature were included.
Romieu et al., 1996	LRI	1.07 (1.02-1.14) per 50 ppb	1-h max, lag 0	OR estimated using GEE multivariate models in which O_3 and $PM_{2.5}$ and minimum temperature were included.
	Cough	1.08 (1.01-1.16) per 50 ppb	1-h max, lag 1	
Romieu et al., 1997	Cough	1.08 (1.02-1.15) per 50 ppb	1-h max, lag 0	ORs estimated using GEE models adjusted for minimum temperature.
	Phlegm	1.24 (1.13-1.35) per 50 ppb	1-h max, lag 0	
		1.13 (1.03-1.22) per 50 ppb	1-h max, lag 1	
		1.11 (1.02-1.22) per 50 ppb	1-h max, lag 2	
	LRI	1.11 (1.05-1.19) per 50 ppb	1-h max, lag 0	
		1.08 (1.01-1.15) per 50 ppb	1-h max, lag 1	
		1.07 (1.02-1.13) per 50 ppb	1-h max, lag 2	
	Bronchodilator use	1.03 (1.00-1.05 per 50 ppb	1-h max, lag 0	
	Cough	1.10 (1.00-1.20) per 50 ppb	1-h max, lag 0	ORs estimated for O_3, PM_{10}, and minimum temperature simultaneously using multivariate models.
	Phlegm	1.289 (1.17-1.43) per 50 ppb	1-h max, lag 0	
	Difficulty in breathing	1.19 (1.06-1.34) per 50 ppb	1-h max, lag 0	
	Wheezing	1.09 (0.96-1.24) per 50 ppb	1-h max, lag 0	
	LRI	1.12 (1.01-1.23) per 50 ppb	1-h max, lag 0	

Table 5-5. Effects of ozone on respiratory symptoms in children.

Reference	Symptom	Measure	O_3 Metric	Data Analysis and Remarks
Schwartz et al., 1994	Cough	Odds Ratio (95% CI) = 1.23 (0.99-1.54) per 30 ppb	Previous 24-h avg	Single-pollutant regression model.
		Odds Ratio (95% CI) = 1.22 (0.96-1.49) per 30 ppb	Previous 24-h avg	Two-pollutant (O_3 and PM_{10}) linear logistic regression model. Result reflects lower sample size caused by the greater number of days with missing PM_{10} data.
Buchdahl et al., 1996	Wheeze	Relative Risk (95% CI) = 3.01 (2.17-4.18)	2 SD < mean	Rate ratios estimated using nonlinear Poisson regression adjusted for season, temperature, wind speed.
		Relative Risk (95% CI) = 1.34 (1.09-1.66)	2 SD > mean	

Table 5-5. Effects of ozone on respiratory symptoms in children.

Reference	Symptom	Measure Relative Risk (95% CI)	O_3 Metric	Data Analysis and Remarks
Thurston et al., 1997	Use of medication prescribed for "as needed administration" (prn-β-agonist treatments)	1.46	1-h max, lag 0	Regression analysis of O_3 associations with individual subjects. Converted to relative risks from Poisson coefficients at the mean concentration (83.6 ppb). Implies a slope at the mean of 0.0020 prn-β-agonist treatments/day/child/ppb, which is very close to the value for the aggregate analysis (0.0018).
	Chest symptoms	1.50	1-h max, lag 0	Regression analysis of O_3 associations with individual subjects. Converted to relative risks from Poisson coefficients at the mean concentration (83.6 ppb). Implies a slope at the mean of 0.0031 symptoms/day/child/ppb, which is very close to the value for the aggregate analysis (0.0029).
	Use of medication prescribed for "as needed administration" (prn-β-agonist treatments)	Not significant	1-h max, lag 0	Simultaneous regressions of SO_4^{2-} and O_3. These two pollutants were highly correlated with each other over time causing their regression coefficients to be highly negatively correlated.
	Chest symptoms	Significant (nearly unchanged from single pollutant model)	1-h max, lag 0	Simultaneous regressions of SO_4^{2-} and O_3.

Table 5-5. Effects of ozone on respiratory symptoms in children.

Reference	Symptom	Measure	O_3 Metric	Data Analysis and Remarks
Gielen et al., 1997	Runny/stuffed nose, sore throat	Prevalence Ratio (95%CI) = 1.23 (1.03-1.43)	8-h current day	$P < 0.05$. Linear time series analysis model adjusted for pollen, time trend, day of week.
Berry et al., 1991	Cough	Positive Response = 12.6%	< 80 ppb	Hourly O_3 level just prior to spirometry. Daily symptom questionnaire for previous 24-h period.
		Positive Response = 23.7%	80-120 ppb	
		Positive Response = 27.5%	> 120 ppb	
	Chest pains	Positive Response = 6.9%	< 80 ppb	
		Positive Response = 7.9%	80-120 ppb	
		Positive Response = 10.0%	> 120 ppb	
	Runny or stuffy nose	Positive Response = 48.3%	< 80 ppb	
		Positive Response = 51.3%	80-120 ppb	
		Positive Response = 57.5%	> 120 ppb	
	Eye irritation	Positive Response = 12.6%	< 80 ppb	
		Positive Response = 18.4%	80-120 ppb	
		Positive Response = 27.5%	> 120 ppb	
	Shortness of breath	Positive Response = 3.4%	< 80 ppb	
		Positive Response = 3.9%	80-120 ppb	
		Positive Response = 7.5%	> 120 ppb	

Table 5-5. Effects of ozone on respiratory symptoms in children.

Reference	Symptom	Measure	O_3 Metric	Data Analysis and Remarks
Castillejos et al., 1992	Cough or phlegm	Positive Response = 13.6%	Increase from 29 to 52 ppb	48-h mean O_3. Logistic regression.
		Positive Response = 17.1%	Increase from 52 to 65.6 ppb	
		Positive Response = 18.5%	Increase from 65.7 to 82.3 ppb	
Delfino et al., 1996	Asthma	Increase (95% CI) = 25% (0-49%)	25 ppb (90^{th} percentile) increase in personal O_3	Random effects autoregression models controlled for weekend days and fungal spores.
	Inhaler use	Increase (95% CI) = 26% (3-48%)		

Abbreviations:
24-h avg = average value for a 24-h period
1-h max = daily 1-h maximum
8-h max = maximum value for a daily 8-h time period
12-h mean = mean value for a 12-h period
CI = confidence interval
d = day
GEE = general estimating equations
h = hour
lag = number of days (time lag) between exposure and effect
LRI = lower respiratory illness (presence of one or more symptoms of cough, phlegm, wheezing, or difficulty breathing)
max = maximum
personal = personal O_3 exposures measured with passive samplers worn by subjects
O_3 = ozone
OR = odds ratio, given per unit increase in O_3 level
P = probability
$PM_{2.5}$ = particulate matter ≤ 2.5 μm in diameter
PM_{10} = particulate matter ≤ 10 μm in diameter
ppb = parts per billion
PR = change in prevalence between day with highest and day with lowest O_3 level, relative to mean prevalence.
prn = *pro re nata* (as needed)
RR = rate ratio or relative risk
SD = standard deviation
SO_4^{2-} = sulfate

Table 5-6. Effects of ozone on values for PEFR (ml/s/ppb), FEV_1 (ml/ppb), or FVC (ml/ppb) in children.

Reference	Measure Slope ± SE	Description	Data Analysis
Castillejos et al., 1992	–0.069 ± 0.029	FVC. Mean O_3 1 h before spirometry. $P < 0.05$.	Multiple linear regression adjusted for temperature and RH.
	–0.488 ± 0.160	FEV_1. Mean O_3 48-h before spirometry. $P < 0.05$.	
	–0.097 ± 0.038	FVC. Daily 1-h max O_3 in 24 h preceding spirometry. $P < 0.05$.	
	–0.127 ± 0.035	FEV_1. Daily 1-h max O_3 in 24 h preceding spirometry. $P < 0.05$.	
Higgins et al., 1990	–0.76 ± 0.15 (SD)	FEV_1. Current O_3 exposure. $R^2 = 0.961$. $P < 0.0001$.	Expanded common slope regression model that included temperature, RH, and coarse and fine PM. Similar results for hourly O_3 levels 2-h previous to current exposure.
	–0.68 ± -0.16 (SD)	FVC. Current O_3 exposure. $R^2 = 0.963$. $P < 0.0001$.	
	–1.91 ± 0.63 (SD)	PEFR. Current O_3 exposure. $R^2 = 0.870$. $P < 0.01$.	
Kinney et al., 1996	–0.50 ± 0.07	FEV_1. $P < 0.0001$. 1-h mean O_3 in hour preceding lung function tests.	Data from 6 camp studies analyzed in one linear regression model that fit subject-specific intercepts and 1 pooled O_3 slope (no trend line).
	–0.26 ± 0.07	FEV_1. $P < 0.0003$. 1-h mean O_3 in hour preceding lung function tests.	Data from 6 camp studies analyzed in one linear regression model that fit subject-specific intercepts, slopes of trend functions, and one pooled O_3 slope.

Table 5-6. Effects of ozone on values for PEFR (ml/s/ppb), FEV_1 (ml/ppb), or FVC (ml/ppb) in children.

Reference	Measure	Description	Data Analysis
Linn et al., 1996	Slope ± SE = –0.58 ± 0.23	ΔFEV_1, a.m. to p.m. Current 24-h O_3. $P < 0.01$.	ANOVA/regression model incorporating one environmental variable adjusted for year, season, day of week, and year-season interaction.
Spektor et al., 1991	Slope ± SE = –1.53 ± 0.38	p.m. FVC measurement. Previous-h mean O_3.	Linear regression.
	Slope ± SE = –1.60 ± 0.30	p.m. FEV_1measurement. Previous-h mean O_3.	
	Slope ± SE = –5.38 ± 0.92	p.m. PEFR measurement. Previous-h mean O_3.	
Ulmer et al., 1997	Slope = –2.46	FVC. Maximum ½-h mean O_3 concentration in 24 h preceding lung function tests. Town with higher O_3 level (median, 50.6 ppb). $P < 0.0018$.	Longitudinal linear regression model adjusted for national origin, gender, and passive smoke exposure, height, and time of day of lung function test.
	Slope = –2.26	FEV_1. Maximum ½-h mean O_3 concentration in 24 h preceding lung function tests. Town with higher O_3 level (median, 50.6 ppb). $P < 0.0021$.	
Braun-Fahrlander et al., 1994	Coefficient = –1.14	ΔPEFR from before to after exercise. 95% CI = –2.00, –0.285. ½-h mean O_3 during exercise test.	Multiple regression model controlling for day of study, temperature, RH, study area, and sex.

Table 5-6. Effects of ozone on values for PEFR (ml/s/ppb), FEV_1 (ml/ppb), or FVC (ml/ppb) in children.

Reference	Measure Coefficient ± SE	Description	Data Analysis
Gielen et al., 1997	–0.93 (–1.79, –0.07)	Pooled individual regression coefficient and 95% CI in percent of mean PEFR over range of O_3 concentrations. Morning PEFR. $P < 0.05$. 8-h max O_3, 2-d lag.	Individual multiple linear regression adjusted for pollen, time trend, day of week. Performed time series analysis to correct for autocorrelation.
	–.94 (–1.97, 0.09)	Pooled individual regression coefficient and 95% CI in percent of mean PEFR over range of O_3 concentrations. Evening PEFR. $P < 0.10$. 8-h max O_3, 2-d lag.	
Hoek et al., 1993b	–0.10 ± 0.02	Mean ± SE of individual regression coefficients for FVC. Previous day 1-h max O_3. $P < 0.05$.	Individual linear regression adjusted for lung growth. No material influence of adding other pollutants to the model.
	–0.10 ± 0.02	Mean ± SE of individual regression coefficients for FEV_1. Previous day 1-h max O_3. $P < 0.05$.	
	–0.86 ± 0.11	Mean ± SE of individual regression coefficients for PEFR. Previous day 1-h max O_3. $P < 0.05$.	
Krzyzanowski et al., 1992	–2.30 ± 0.817	Coefficient ± SE for PEFR evening measurement in asthmatics. Daily 1-h max O_3. $P < 0.05$.	Regression analysis using a random-effects longitudinal model.
	–2.93 ± 0.967	Coefficient ± SE for PEFR evening measurement in asthmatics. Daily maximum 8-h mean O_3. $P < 0.05$.	

Table 5-6. Effects of ozone on values for PEFR (ml/s/ppb), FEV_1 (ml/ppb), or FVC (ml/ppb) in children.

Reference	Measure	Description	Data Analysis
Studnicka et al., 1995	Coefficient = –2.16 ± 1.01	Coefficient ± SE for FEV_1 for Panel 1 (highest daily maximum O_3 levels). Same day ½-h max O_3. P < 0.033.	Linear regression analyses for repeated measures adjusted for sex, height, and age. Included within-subject correlation correction.
	Coefficient = –4.11 ±0.87	Coefficient ± SE for FVC for Panel 1. Same day ½-h max O_3. P < 0.000.	
	Coefficient = –8.93 ± 2.57	Coefficient ± SE for PEFR for Panel 1. Same day ½-h max O_3. P < 0.000.	
Thurston et al., 1997	Coefficient = –1.6	ΔPEFR a.m. to p.m. Daily 1-h max O_3.	Linear regression of intercept of each subject and overall slope for the pollutant. Single pollutant model without temperature. Autocorrelation assessed..
Neas et al., 1995	Mean Deviation = –1.24	PEFR. 95% CI, –2.46 to –0.03.	Autoregressive linear regression using an autoregressive integrated moving average procedure. Mean deviation in evening PEFR associated with unweighted preceding 12-h average daytime O_3 concentration adjusted for 12-h average daytime temperature, trend, evening measurement, and 2^{nd}-order autocorrelation.
	Mean Deviation = –1.55	PEFR. 95% CI, –3.72 to –0.62.	Autoregressive linear regression using an autoregressive integrated moving average procedure. Mean deviation in evening PEFR associated with preceding 12-h average daytime O_3 concentration weighted by proportion of hours spent outdoors during prior 12-h and adjusted for 12-h average daytime temperature, trend, evening measurement, and 2^{nd}-order autocorrelation.

Table 5-6. Effects of ozone on values for PEFR (ml/s/ppb), FEV_1 (ml/ppb), or FVC (ml/ppb) in children.

Reference	Measure	Description	Data Analysis
Romieu et al., 1997	Mean Deviation = –0.60	Evening PEFR. Daily 1-h max O_3. 95% CI, –1.20 to –0.003.	Estimated using GEE regression models and adjusted for minimum temperature.
	Mean Deviation = –0.77	Evening PEFR. Lag 1 d O_3. 95% CI, –1.39 to –0.16.	
Stern et al., 1994	Percent = –1.7	FVC. $P < 0.01$. Children living in moderate compared to low exposure region.	Mixed effects regression model adjusted for age, sex, weight, standing height, parental smoking, and gas cooking.
	Percent = –1.3	FEV_1. $P < 0.01$. Children living in moderate compared to low exposure region.	

Abbreviations:

½-h max = daily ½ h maximum
1-h max = daily 1-hour maximum
8-h max = maximum value for a daily 8-h time period
ANOVA = analysis of variance
CI = confidence interval
d = day
FEV_1 = forced expiratory volume in one second
ΔFEV_1 = change in forced expiratory volume in one second
FVC = forced vital capacity
GEE = general estimating equations
lag = number of days (time lag) between exposure and effect
R^2 = coefficient of determination
max = maximum
O_3 = ozone
P = probability
PEFR = peak expiratory flow rate
ΔPEFR = change in peak expiratory flow rate
PM = particulate matter
ppb = parts per billion
h = hour
SD = standard deviation
SE = standard error
RH = relative humidity

Table 5-7. Effect of ozone on hospital admissions for children.

Reference	Rate Ratio (95% CI)	Description	Data Analysis
Buchdahl et al., 1996	3.01 (2.17-4.18)	RR for 2 SD < mean (mean, 20 ppb). Accident or emergency department visit for acute wheezy episodes.	Nonlinear Poisson regression model adjusted for season, temperature, wind speed.
	1.34 (1.09-1.66)	RR for 2 SD > mean (mean, 20 ppb). Accident or emergency department visit for acute wheezy episodes.	
Burnett et al., 1994	1.08 per 50 ppb increase in O_3	P < 0.005. 0-1 y of age. 1-h max O_3. Asthma admissions, July-Aug.	Poisson regression. Seasonal, day of week, and hospital effects removed.
Romieu et al., 1995	1.43 (1.24-1.66) per 50 ppb increase in O_3	1-h max O_3, 1-d lag. Emergency visits for asthma.	Multivariate regression model included day of week, period, daily minimum temperature.
	1.68 (1.30-2.17) per 50 ppb increase in O_3	1-h max O_3. After preceding and concurrent day of > 110 ppb O_3. Emergency visits for asthma.	
	2.33 (1.83-2.96) per 50 ppb increase in O_3	1-h max O_3. After 2 preceding days of > 110 ppb O_3. Emergency visits for asthma.	
White et al., 1994	1.37 (1.02-1.73)	Following days with max 1-h O_3 levels of ≥110 ppb. Emergency visits for asthma or reactive airway disease.	Poisson regression.

Abbreviations:
1-h max = daily 1-hour maximum
CI = confidence interval
d = day
h = hour
O_3 = ozone
max = maximum
P = probability
ppb = parts per billion
RR = rate ratio
SD = standard deviation
y = year

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
California	Abbey et al., 1998	Nonsmokers ≥25 y	Lung Function	See Table 5-9	Interquartile Range (number of d/y PM_{10} exceeded 100 µg/m^3)	O_3 SO_2 SO_4^{2-} TSP	Medium
California	Beeson et al., 1998	Nonsmokers ≥25 y	Incident Lung Cancer in Males	5.21(1.94-13.99)/ 24 µg/m^3	Interquartile Range Mean PM_{10}	O_3 SO_2 NO_2	Medium
Mexico City, Mexico	Borja-Aburto et al., 1998	All ages	Total Mortality Mortality Among ≥65 y Cardiovascular Mortality	1.017 (1.002-1.031)/10 µg/m^3 1.023 (1.003-1.042)/10 µg/m^3 1.034 (1.007-1.062)/10 µg/m^3	24-h Avg $PM_{2.5}$, Lag 4	O_3 NO_2 NO_x NO SO_2	High
Montreal, Canada	Delfino et al., 1998a	≥65 y	Respiratory ER Visits	No association	24-h Avg $PM_{2.5}$	O_3	Medium
The Netherlands	Hiltermann et al., 1998	Non-Smoking Asthmatic Adults	Shortness of Breath Woken Up with Breathing Problems Bronchodilator Use	1.17 (1.03-1.34)/ 50 µg/m^3 1.24 (1.01-1.54)/ 50 µg/m^3 1.12 (1.00-1.25)/ 25 µg/m^3	24-h Avg PM_{10}, Lag 0 24-h Avg PM_{10}, Lag 0 Mean 7-Day PM_{10}	O_3 BS NO_2 SO_2	High

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
Sydney, Australia	Morgan et al., 1998	All ages	Total Mortality	1.023 (1.002-1.044)	10^{th} to 90^{th} Percentile Estimated 24-h Avg $PM_{2.5}$ (13.8 µg/m^3), Lag 1	O_3 NO_2	Medium
Helsinki, Finland	Pönkä et al., 1998	< 65 y	Total Mortality	1.034 (1.011-1.059/ 10 µg/m^3	24-h Avg PM_{10}, Lag 5	O_3 SO_2 NO_2	Low
			Cardiovascular Mortality	1.041 (1.004-1.080)/10 µg/m^3	24-h Avg PM_{10}, Lag 5	NO CO TSP	
London, Amsterdam, Rotterdam, and Paris	Spix et al., 1998	15-64 y	Respiratory Hospital Admissions (mainly emergency) All Year	1.028 (1.006-1.051)/ 50 µg/m^3	24-h Avg BS, Lag 2 for 3 cities, Lag 0 for one	O_3 SO_2 TSP NO_2	Low
			Respiratory Hospital Admissions Cold Season	1.04 (1.02-1.07)/ 50 µg/m^3			
		≥65 y	Respiratory Hospital Admissions Warm Season	1.07 (1.00-1.15)/ 50 µg/m^3			
Latrobe Valley, Victoria, Australia	Voight et al., 1998	All ages	COPD Hospital Admissions	RR not reported	24-h Avg LVD, Lag 0	O_3 SO_2 NO_2	Low

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
5 Central and Western Europe Cities	Anderson et al., 1997	All ages (48-70% ≥65 y)	COPD Hospital Admissions (mainly emergency)	1.035 (1.010-1.060)/ 50 μg/m^3	24-h Avg BS, Lag 1	O_3 SO_2 TSP NO_2	Medium
Mexico City, Mexico	Borja-Aburto et al., 1997	All ages	Total Mortality Respiratory Mortality Cardiovascular Mortality	1.058 (1.033-1.083)/100 μg/m^3 1.095 (1.013-1.184)/ 100 μg/m^3 1.052 (1.009-1.099)/ 100 μg/m^3	24-h Avg TSP, Lag 0	O_3 SO_2 CO	High
16 Canadian Cities	Burnett et al., 1997a	All ages	Respiratory Hospital Admissions	RR not reported for COH	1-h Max COH	O_3 SO_2 NO_2 CO	Low
Toronto, Canada	Burnett et al., 1997b	All ages	Respiratory or Cardiac Hospital Admissions	No association	Interquartile Range 24-h Avg $PM_{2.5}$ (11 μg/m^3)	O_3 SO_2 NO_2 PM_{10} CP COH CO SO_4^{2-} H^+	Low

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
Montreal, Canada	Delfino et al., 1997	> 64 y	Respiratory ER Visits	1.116 (1.023-1.210)/12.15 µg/m^3 $PM_{2.5}$ (1993) 1.159 (1.042-1.276)/21.70 µg/m^3 PM_{10} (1993)	Increase in Mean ER Visit Level per Increase to Mean 24-h Avg $PM_{2.5}$ Level, Lag 1	O_3 SO_4^{2-} H^+	Medium
Rotterdam, The Netherlands	Hoek et al., 1997	All ages	Total Mortality	1.04 (1.00-1.09)/ 91 µg/m^3	5^{th} to 95^{th} Percentile 24-h Avg TSP, Lag 1	O_3 Fe in TSP BS SO_2 CO	Medium
Philadelphia, PA	Kelsall et al., 1997	All ages	Total Mortality	1.012 (1.004-1.019)/ 34.5 µg/m^3	Inter-quartile Range 24-h Avg TSP, Lag 0	O_3 SO_2 NO_2 CO	Low
Paris, France	Medina et al., 1997	All ages	House Calls for Asthma House Calls for Headache	1.09 (1.04-1.15)/ 44 µg/m^3 1.16 (1.12-1.19)/ 44 µg/m^3	5^{th} to 95^{th} Percentile 24-h Avg BS, Lag 0-3	O_3 PM_{13} SO_2 NO_2	Medium
Minneapolis-St. Paul, MN; Birmingham, AL	Moolgavkar et al., 1997	≥65 y	Respiratory Hospital Admissions, Minneapolis	1.034 (1.019-1.052)/ 20 µg/m^3	Interquartile Range 24-h Avg PM_{10}, Lag 0	O_3 SO_2 NO_2 CO	Medium

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
Brisbane, Australia	Simpson et al., 1997	All ages ≥65 y	Total Mortality	1.009 (1.003-1.015)/10 µg/m^3 PM_{10} (estimated from b_{sp}) 1.010 (1.002-1.018)/10 µg/m^3 PM_{10} (estimated from b_{sp})	24-h Avg b_{sp}, Lag 0	O_3 NO_2 SO_2	Medium
Barcelona, London, and Paris	Sunyer et al., 1997	Adults	Asthma Emergency Admissions	No association	24-h Avg BS	O_3 SO_2 NO_2	Low
Paris, France	Dab et al., 1996	All ages	Respiratory Mortality Respiratory Hospital Admissions	1.168 (1.041-1.310) 1.045 (1.004-1.087)	Increase of 100 µg/m^3 above the 5th Percentile of 24-h Avg PM_{13}	O_3 BS SO_2 NO_2	Medium
Cook County, IL	Ito and Thurston, 1996	All ages	Total Mortality Cancer Mortality Respiratory Mortality	1.04 (1.01-1.07)/100 µg/m^3 1.12 (1.06-1.18)/100 µg/m^3 1.14 (1.04-1.25)/100 µg/m^3	24-h Avg PM_{10}, Avg of Lags 0 and 1	O_3 CO SO_2	High

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
Santiago, Chile	Ostro et al., 1996	All ages	Total Mortality	1.04 (1.01-1.07)/ 115 μg/m^3	24-h Avg PM_{10}, Lag 0	O_3 NO_2 SO_2	High
			Respiratory Mortality	1.15 (1.08-1.23)/ 115 μg/m^3			
			Cardiovascular Mortality	1.09 (1.04-1.14)/ 115 μg/m^3			
			Mortality for Age ≥65 y	1.11 (1.07-1.14)/ 115 μg/m^3			
London, United Kingdom	Ponce de Leon et al., 1996	All ages	Respiratory Emergency Hospital Admissions	No association	10^{th}-90^{th} Percentile 24-h Avg BS, Lag 1	O_3 SO_2 NO_2	Low
Helsinki, Finland	Pönkä and Virtanen, 1996a	≥15 y	Asthma ER Hospital Admissions	No association	24-h Avg TSP	O_3 SO_2 NO_2	Low
Helsinki, Finland	Pönkä and Virtanen, 1996b	All ages	Cardiac and Cerebrovascular Hospital Admissions	No association	24-h Avg TSP, Lag 3	O_3 SO_2 NO_2 NO	Low
Amsterdam and Rotterdam, The Netherlands	Schouten et al., 1996	All ages	Respiratory Emergency Hospital Admissions	No association	24-h Avg BS, Lag 2	O_3 SO_2 NO_2 BS	Low

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
Spokane, WA	Schwartz, 1996	≥65 y	Respiratory Hospital Admissions	1.085 (1.036-1.136)/ 50 μg/m^3	24-h Avg PM_{10}, Lag 0	O_3	Medium
Cleveland, OH	Schwartz et al., 1996	≥65 y	Respiratory Hospital Admissions	1.12 (1.01-1.24)/ 100 μg/m^3	24-h Avg PM_{10}, Avg of Lags 1 and 2	O_3 SO_2	High
New Brunswick, Canada	Stieb et al., 1996	All ages	Asthma ER Visits	No association	24-h TSP, Lag 3	O_3 SO_2 NO_2 SO_4^{2-}	Low
Barcelona, Spain	Sunyer et al., 1996	All ages	Total Mortality Mortality ≥70 y Cardiovascular Mortality	1.070 (1.029-1.112)/ 100 μg/m^3 1.063 (1.016-1.113)/ 100 μg/m^3 1.093 (1.036-1.153)/ 100 μg/m^3	24-h Avg BS, Lag 1	O_3 SO_2 NO_2	Medium
Amsterdam, The Netherlands	Verhoeff et al., 1996	All ages	Total Mortality	1.19 (1.02-1.38)/ 100 μg/m^3	24-h Avg BS, Lag 0	O_3 TSP PM_{10} SO_2 CO	Medium
Lyon, France	Zmirou et al., 1996	All ages	Respiratory Mortality	1.04 (1.00-1.09)/ 50 μg/m^3	24-h Avg PM_{13}, Lag 0	O_3 SO_2 NO_2	Low

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
California	Abbey et al., 1995	Nonsmokers ≥25 y	New Cases of Definite Symptoms of Airway Obstructive Disease	1.17 (1.02-1.33)	Avg annual exceedance frequency of 1000 h/y for 100 μg/m^3 PM_{10}	None	Medium
			New Cases of Definite Chronic Bronchitis Symptoms	1.17 (1.01-1.35)			
Barcelona, Spain	Castellsague et al., 1995	< 65 y	Asthma ER Visits, Summer	1.082 (1.011-1.157)/25 μg/m^3	24-h Avg BS, Lag 0	O_3 SO_2 NO_2	Low
Los Angeles, CA	Kinney et al., 1995	All ages	Total Mortality	1.05 (1.00-1.11)/ 100 μg/m^3	24-h Avg PM_{10}, Lag 0	O_3 CO	Low
Philadelphia, PA	Moolgavkar et al., 1995	All ages	Total Mortality, Summer	1.068 (1.025-1.113)/ 100 μg/m^3	24-h Avg TSP, Lag 1	O_3 SO_2	Low
151 U.S. Cities	Pope et al., 1995	Adults	Total Mortality	1.17 (1.09-1.26)/ 24.5 μg/m^3	24-h Median $PM_{2.5}$, Lag 0	SO_4^{2-}	High
			Cardiopulmonary Mortality	1.31 (1.17-1.46)/ 24.5 μg/m^3			
Sao Paulo, Brazil	Saldiva et al., 1995	≥65 y	Total Mortality	1.13 (1.07-1.18)/ 100 μg/m^3	24-h Avg PM_{10}	O_3 SO_2 NO_x CO	High

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
New Haven CT and Tacoma, WA	Schwartz, 1995	≥65 y	Respiratory Hospital Admissions, New Haven Respiratory Hospital Admissions, Tacoma	1.06 (1.00-1.13)/ 50 µg/m^3 1.10 (1.03-1.17)/ 50 µg/m^3	24-h Avg PM_{10}, Lag 2	O_3 SO_2	High
Detroit, MI	Schwartz and Morris, 1995	≥65 y	Ischemic Heart Disease Hospital Admissions Congestive Heart Failure Admissions	1.018 (1.005-1.032)/ 32 µg/m^3 1.032 (1.012-1.052)/ 32 µg/m^3	Interquartile Range PM_{10}, Lag 0 Interquartile Range PM_{10}, Avg Lags 0 and 1	O_3 SO_2 CO	High
Montreal, Canada	Delfino et al., 1994	All ages	Asthma Hospital Admissions	1.025 (1.007-1.048)/ 12 µg/m^3	24-h Avg PM_{10}, Lag 3 (May-Oct.)	O_3 SO_4^{2-}	Low
Helsinki, Finland	Pönkä and Virtanen, 1994	All ages	Chronic Bronchitis or Emphysema Hospital Admissions	No association	24-h Avg TSP	O_3 SO_2 NO_2	Low
Birmingham, AL	Schwartz, 1994a	≥65 y	Pneumonia Hospital Admissions COPD Hospital Admissions	1.19 (1.07-1.32)/ 100 µg/m^3 1.27 (1.08-1.50)/ 100 µg/m^3	24-h Avg PM_{10}, Lag 0	O_3	Medium

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
Detroit, MI	Schwartz, 1994b	≥65 y	Pneumonia Hospital Admissions COPD Hospital Admissions	1.012 (1.004-1.019)/ 10 μg/m^3 1.020 (1.004-1.032)/ 10 μg/m^3	24-h Avg PM_{10}, Lag 0	O_3	Medium
Minneapolis-St. Paul, MN	Schwartz, 1994c	≥65 y	Pneumonia Hospital Admissions COPD Hospital Admissions	1.17 (1.02-1.33)/ 100 μg/m^3 1.57 (1.20-2.06)/ 100 μg/m^3	24-h Avg PM_{10}, Lag 0 24-h Avg PM_{10}, Avg of Lags 0 and 1	O_3	Medium
Toronto, Ontario, Canada	Thurston et al., 1994	All ages	Respiratory Hospital Admissions	No association	24-h Avg $PM_{2.5}$	O_3 SO_4^{2-} H^+ CP PM_{10} TSP SO_2 NO_2	Low

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
California	Abbey et al., 1993	Nonsmokers ≥25 y	New Cases of Definite Symptoms of Airway Obstructive Disease	1.36 (1.11-1.66)	Avg annual exceedance frequency of 1000 h/y for 200 µg/m^3 TSP	O_3 SO_2	Medium
			New Cases of Definite Chronic Bronchitis Symptoms	1.33 (1.07-1.65)			
			New Cases of Definite Symptoms of Asthma	1.74 (1.11-2.72)			
Southern California	Ostro et al., 1993	Nonsmokers ≥18 y	Respiratory Morbidity	No association	24-h Avg COH, Lag 0 or 1	O_3 SO_4^{2-} NO_2 SO_2	Low
Seattle, WA	Schwartz et al. 1993	All ages	Asthma Hospital ER Visits	1.12 (1.04-1.20)/ 30 µg/m^3	24-h Avg PM_{10}	SO_2 O_3	Medium
St. Louis, MO and Eastern Tennessee	Dockery et al., 1992	All ages	Total Mortality, St. Louis	1.16 (0.99-1.33)/ 100 µg/m^3	24-h Avg PM_{10}, Lag 1	O_3 $PM_{2.5}$ SO_2 NO_2 SO_4^{2-} H^+	Medium
Helsinki, Finland	Pönkä, 1991	All ages	Asthma Hospital Admissions	No association	24-h Avg TSP	O_3 SO_2 NO NO_2 CO	Low

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
Utah, Salt Lake, and Cache Valleys, UT	Pope, 1991	All ages	Respiratory Hospital Admissions	RR not reported	24-h Avg PM_{10}	None	Medium
Barcelona, Spain	Sunyer et al., 1991	> 35 y	COPD ER Admissions	RR not reported	24-h Avg BS	O_3 CO NO_2	Low
United States	Ostro and Rothschild, 1989	18-65 y	Respiratory-Related Restricted Activity Days Minor Restricted Activity Days	1.016/µg/m^3 1.008/µg/m^3	24-h Avg $PM_{2.5}$, 2-period lag	O_3	Medium

Table 5-8. Summary of effect estimates for particulate matter in adults in recent studies.

Location	Reference	Population	Health Outcome	Response RR (95% CI)	PM Exposure Metric	Co-Pollutants Considered	Confidence in Study
United States	Ostro, 1987	18-65 y	Restricted Activity Days	RR not reported	Fine PM estimated using airport visibility data	None	High

Abbreviations:

24-h Avg = average value for a 24-h period
Avg = average
BS = black smoke
b_{sp} = scattering coefficient of particles measured by nephelometry
CI = confidence interval
CO = carbon monoxide
COH = coefficient of haze; an optical measure of airborne particulate matter
COPD = chronic obstructive pulmonary disease
CP = coarse particles (2.5 µm < aerodynamic diameter < 10 µm)
d = day
ER = emergency room
Fe = iron
h = hour
H^+ = hydrogen ion
Interquartile Range = 3rd minus 1st quartile
Lag = delay (in days) of the manifestation of the effect following exposure
LVD = local visual distance; a measure of particles of size range 0.1-2.5 µm
m = meter
Mean 7-Day = mean concentration of the previous week
MI = myocardial infarction
NO = nitric oxide
NO_2 = nitrogen dioxide
NO_x = nitrogen oxides
No association = no association at $p < 0.05$.
O_3 = ozone
PM = particulate matter
$PM_{2.5}$ = particulate matter ≤2.5 µm aerodynamic diameter
PM_{10} = particulate matter ≤10 µm aerodynamic diameter
PM_{13} = particulate matter ≤13 µm aerodynamic diameter
RR = relative risk; given per unit increase in PM level
SO_2 = sulfur dioxide
SO_4^{2-} = sulfate
TSP = total suspended particulate matter
y = year

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Abbey et al., 1998	Cohort	1977-1993	31.3 (Mean d/y exceeding 100 $\mu g/m^3$ PM_{10}, Males) 0.3-112.4 (Range d/y exceeding 100 $\mu g/m^3$ PM_{10}, Males)	Sex-specific multiple linear regressions adjusted for covariates. In single pollutant models, an interquartile range increase of 54.2 d/y when PM_{10} exceeded 100 $\mu g/m^3$ was associated with a 7.2% decrement in FEV_1 (as % of predicted) in males whose parents had asthma, bronchitis, emphysema, or hay fever (95% CI, -2.7% to –11.5%).	No
Beeson et al., 1998	Cohort	1977-1992	51 ± 16.5 (Avg Annual Mean PM_{10} ± SD)	Time-dependent, gender-specific Cox proportional hazards regression models adjusted for potential confounders. Analyzed single-pollutant models plus multipollutant models of all pairwise comparisons of annual avg number of h > 100 ppb O_3 and mean levels of the other 3 pollutants. In multipollutant analyses, the avg annual d/y in excess of 100 $\mu g/m^3$ PM_{10} had the strongest association with lung cancer in males, and SO_2 had the strongest association with lung cancer in females.	No
Borja-Aburto et al., 1998	Time series	1993-1995	27 (Mean 24-h Avg $PM_{2.5}$) 4-85 (Range 24-h Avg $PM_{2.5}$)	Poisson regression employing a generalized additive model and controlling for temperature and periodic cycles. Accounted for autocorrelation. Considered lags. $PM_{2.5}$ and O_3 positively correlated. RRs given in Table 5-8 are for 3-pollutant models. In single-pollutant model, $PM_{2.5}$ was associated with a 2.5% increase in respiratory deaths; the association decreased to 2% in 2-pollutant model with NO_2, and to 1.7% in 3-pollutant model with NO_2 and O_3. CI for all models of $PM_{2.5}$ and respiratory deaths included the null value.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Delfino et al., 1998a	Time series	June-Aug., 1989-1990	18.6 ± 9.3 (24-h Avg $PM_{2.5}$ ± SD) 30.0 (90^{th} Percentile 24-h Avg $PM_{2.5}$)	Ordinary least squares regressions of the normally distributed filtered ER visit variables. Autoregressive parameters added if needed. Time series regressions controlled for season, day of week, autocorrelation, temperature, and RH. For 1989, consistent but not statistically significant correlation found between elderly respiratory visits and $PM_{2.5}$, lag 1, was confounded by temperature and O_3. No significant correlations for the summer of 1990 after taking day of week into account.	No
Hiltermann et al., 1998	Panel	July 3 to Oct. 6, 1995	39.7 (24-h Avg PM_{10}) 16.4-97.9 (Range 24-h Avg PM_{10})	60 patients with intermittent to severe asthma studied using time series analysis. Medication and symptom daily prevalence data analyzed using a logistic regression model. Confounders considered: trends in symptom prevalence, medication use, mean morning and evening PEF, exposure to aeroallergens and environmental tobacco smoke, day of week, and temperature. O_3 was the most prominent air pollutant present and the one most strongly associated with symptom prevalences. No significant associations demonstrated between ΔPEF and pollutants. Authors concluded that severity of asthma is not an indicator for sensitivity to air pollution.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Morgan et al., 1998	Time series	Jan. 1989 to Nov. 1993	9 ± 6.3 (Mean 24-h Avg $PM_{2.5}$ estimated from b_{sp} ± SD) 0.9-55.5 (Range 24-h Avg $PM_{2.5}$ estimated from b_{sp})	Poisson regression using GEE to allow for overdispersion and autocorrelation. Controlled for weather, season, day of the week, public holidays, and influenza epidemics using dummy variables. Lags examined. SO_2 levels negligible. PM (measured by nephelometry) showed strongest association with mortality. RR given in Table 5-8 is for multipollutant model. Significant association of PM and cardiovascular mortality in single-pollutant model became nonsignificant in multipollutant model. No significant association of PM with respiratory mortality. Few daily respiratory deaths.	No
Pönkä et al., 1998	Time series	1987-1993	28 (Median 24-h Avg PM_{10}) 14-55 (5^{th} to 95^{th} Percentile 24-h Avg PM_{10})	Poisson regression controlled for temperature, RH, day of week, month, year, long-term trend, holidays, and influenza epidemics. Investigated lags. Single- and multi-pollutant models analyzed. Effects of O_3 and PM_{10} were independent of each other. No pollutant was significantly related to total mortality or cardiovascular mortality in persons 64+ y. No control admissions category analyzed.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Spix et al., 1998	Meta-analysis	London, 1987-1991 Amsterdam, 1977-1989 Rotterdam, 1977-1989 Paris, 1987-1992	13 (Mean 24-h Avg BS) 6 (Mean 24-h Avg BS) 22 (Mean 24-h Avg BS) 26 (Mean 24-h Avg BS)	Each APHEA center performed Poisson time series regressions of their data controlling for trend, seasonality, calendar effects, unusual events, meteorology, and autoregression. For the meta-analysis, all days with pollution levels > 200 $\mu g/m^3$ were deleted. Meta-analysis was performed using a pooled regression coefficient obtained as a weighted mean of local regression coefficients. A fixed-effects model was used when no heterogeneity was detected. Otherwise, a random-effects model was used and explanations for the heterogeneity was sought. Different measurement methods of particles hindered analysis of their effects. The BS effect was independent of SO_2 but dependent on NO_2. No control admissions category analyzed.	No
Voight et al., 1998	Time series	1988	125.9 km ± 28.0 (Mean 24-h Avg LVD ± SD)	Poisson distribution adjusted for day-of-week and seasonal variation. Asthma and COPD correlated with pollutants individually and in combination. Lags 0 and 1 and autocorrelation examined. NO_2 and particulate matter significantly associated with COPD in multivariate analysis. No significant relationship between any of pollutants and asthma admissions. No control admissions category analyzed.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Anderson et al., 1997	Meta-analysis	1977-1992 (dates varied among cities)	6-41 (Range Median 24-h Avg BS)	As part of APHEA project, this prospective study used parametric Poisson regression controlling for trend, seasonal and other cycles, day of week, holidays, influenza epidemics, temperature, humidity, and autocorrelation. Individual city (Amsterdam, Barcelona, London, Paris, and Rotterdam) results combined. Single-pollutant models analyzed. The most consistent and significant findings were for O_3. Stronger effect of BS in cool season than in warm. Comparatively smaller PM effects in Europe compared with North America. No control admissions category analyzed.	No
Borja-Aburto et al., 1997	Time series	1990-1992	204 (Median 24-h Avg TSP) 66-456 (Range 24-h Avg TSP)	Various Poisson regression models all fit the mortality data well. Adding indicator variables for season, month, and day of week did not significantly reduce the variance. The model that used only temperature was chosen. Overdispersion and autocorrelation were accounted for. In multipollutant models, only TSP remained associated with mortality. Mortality for those > 65 y essentially same as for total mortality for all ages.	No
Burnett et al., 1997a	Time series	Apr. to Dec., 1981-1991	0.64 x 1000 ft ± 0.44 x 1000 ft (Mean 1-h Max COH ± SD, 11 Cities)	Random effects relative risk regression model controlled for SO_2, NO_2, CO, COH, dew point temperature, and seasonal, subseasonal, and day-of-week effects. Fine PM and acid aerosol data not available. Lags examined. O_3, COH, and CO positively associated respiratory admissions.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Burnett et al., 1997b	Time series	Summers of 1992-1994	16.8 (Mean 24-h Avg $PM_{2.5}$) 1-66 (Range 24-h Avg $PM_{2.5}$)	Poisson regression adjusted for long-term trends, season, influenza epidemics, day of week, temperature, and dew point temperature. Compared two methods of removing temporal trends. GEE used to estimate log-relative risks. Lags examined. Only unscheduled admissions were considered, but no control admission category was analyzed. Strongest associations with both respiratory and cardiac diseases observed for COH and O_3. When either $PM_{2.5}$ and PM_{10} were coregressed with O_3, NO_2, and SO_2, the RRs became nonsignificant.	No
Delfino et al., 1997	Time series	June-Sept. 1992-1993	18.5 ± 14.0 (Mean 24-h Avg $PM_{2.5}$ ± SD, 1992) 12.2 ± 7.1 (Mean 24-h Avg $PM_{2.5}$ ± SD, 1993) 30.1 ± 17.3 (Mean 24-h Avg PM_{10} ± SD, 1992) 21.7 ± 10.2 (Mean 24-h Avg PM_{10} ± SD, 1993)	Separate analyses performed by year because yearly differences in means and day-of-week trends were found for most variables. Ordinary least squares regression and multiple regression analyses used. Controlled for temporal trends including day of week, weather, and autocorrelation. Lags examined. For 1992, no significant associations ($p < 0.02$) between air pollutants and ER visits were found. For 1993, no significant associations found with ER visits for patients 2-64 y of age. RRs in Table 5-8 are for single-pollutant models. When O_3 was coregressed with $PM_{2.5}$, association remained significant; variance inflation was due to moderately strong covariation of the two. Effects for O_3 were larger than those for particulate matter. Unclear why findings differed between years.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Hoek et al., 1997	Time series	1983-1991	42 (Median 24-h Avg TSP) 21-112 (5^{th}–95^{th} Percentile 24-h Avg TSP)	Poisson regression analysis using generalized additive models to adjust for long-term and seasonal trends, day of week, weather, and influenza. Lags examined. Conducted sensitivity analyses using different model specifications for seasonal trends. Significant associations between mortality and all pollutants except Fe were found when O_3 lag 1 was included in the model (O_3 negatively correlated with all other pollutants). O_3 and TSP associated most consistently with mortality. RRs are given in Table 5-8 for model that included TSP, O_3, and CO. RRs for TSP in models that included TSP, O_3, and Fe or TSP, O_3, and SO_2 were also 1.04 but were not significant.	No
Kelsall et al., 1997	Time series	1974-1988	67.3 (Mean 24-h Avg TSP) 14.5-222.0 (Range 24-h Avg TSP)	Age-stratified Poisson regression models developed using generalized additive models controlling for long-term trends, season, and weather. Models fitted by maximum likelihood with assumption of constant overdispersion over time. Lags examined. Investigated seasonal and age interactions with pollutant effects. Little evidence of seasonal effects and no overall evidence of modification of pollutant effects by age were found. Increasing TSP effect found with age. Strong independent effect of O_3. In single-pollutant models, TSP and SO_2 were significantly associated with mortality. Because the two pollutants were highly correlated, their effects became nonsignificant when they were coregressed.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Medina et al., 1997	Time series	1991-1995	21 (Mean 24-h Avg BS) 3-150 (Range 24-h Avg BS)	Poisson regressions using nonparametric smoothing functions (generalized additive model) controlled for time trend, season, pollen, influenza epidemics, and weather. Lags tested. Controlled for autocorrelation. Single-pollutant models. No control admission category analyzed. PM_{13} was significantly associated with house calls for asthma and cardiovascular conditions. House calls for headache significantly associated with all air pollutants but O_3. In two-pollutant models including BS with either SO_2, NO_2, or O_3, only BS and O_3 effects remained stable.	No
Moolgavkar et al., 1997	Time series	1986-1991	33.98 (Mean 24-h Avg PM_{10}, Minneapolis-St. Paul) 43.44 (Mean 24-h Avg PM_{10}, Birmingham)	Semiparametric Poisson regression models (generalized additive models) adjusted for temperature, day of week, season, and temporal trends. Examined lags. In Minneapolis, the strongest association with respiratory admissions was with O_3 when the four pollutants were regressed simultaneously. Neither PM_{10}, SO_2, or NO_2 could not be singled out as more important than the others. In Birmingham, no consistent association of respiratory admissions with any of the three available air pollutants was found (NO_2 levels not available and insufficient SO_2 data available). No control admission category analyzed.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Simpson et al., 1997	Time series	1987-1993	2.59×10^5/m (Mean 24-h Avg b_{sp}) 26.9 (Mean 24-h Avg PM_{10})	GEE analysis. Used autoregressive Poisson models and APHEA methods to control for weather, temporal trends, and interaction effects. Lags examined. Alternative approaches tested. Analyzed for effects of extreme events. Strong positive correlations in summer between O_3, NO_2, and b_{sp}. Absence of significant interaction effects between pollutants. No significant association between b_{sp} and mortality for < 65 y. Positive but insignificant associations found between b_{sp} and cardiovascular and respiratory mortality categories; low counts may have caused the insignificant association with respiratory mortality. O_3 also associated with mortality.	No
Sunyer et al., 1997	Meta-analysis	Barcelona, 1986-1989 London, 1987-1991 Paris, 1987-1992	40 (11-258), Median (Range) 24-h Avg BS 13 (3-95) 28 (4-186)	As part of the APHEA project, Poisson regression models controlling for long wavelength patterns, seasonal patterns, day of week, weather, epidemics, and autocorrelation were built by each city. A combined estimate was computed using a fixed effects model except for O_3, which required a random effects model. NO_2 was significantly associated with admissions. For BS, a positive though nonsignificant association was found in all cities. Collinearity among pollutants generated by the same source may have limited separation of their effects. No control admissions category analyzed.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Dab et al., 1996	Time series	1987-1992	50.8 (Mean 24-h Avg PM_{13}) 19-137 (5^{th}-99^{th} Percentile 24-h Avg PM_{13})	Followed APHEA protocol using an autoregressive Poisson model controlled for long-term trends, seasonal, weekly and daily patterns, weather, influenza, holidays, and strikes of medical staff. Lags examined. Emergency and planned hospital admissions not distinguishable. BS and SO_2 also associated with respiratory hospital admissions and mortality.	No
Ito and Thurston, 1996	Time series	1985-1990	40.7 ± 19.1 (Mean 24-h Avg PM_{10} ± SD) 20-65 (10^{th}-90^{th} Percentile 24-h Avg PM_{10})	Poisson regression models controlled for long-wave cycles, day of week, and temperature. RR given in Table 5-8 is for total mortality is for two-pollutant model (PM_{10} and O_3). RRs for cancer and respiratory deaths are for single-pollutant models. Black females showed the highest RRs for total, respiratory, and cancer mortality categories.	No
Ostro et al., 1996	Time series	1989-1991	115 (Mean 24-h Avg PM_{10})	Ordinary least squares regression and parametric tests used. Basic model (included only PM_{10}) controlled for temperature, day of week, month, and year. Additional controls for cyclical nature of total mortality counts and effects of temperature explored through several techniques. Other pollutants, unusual events, and lags examined. Autocorrelation corrections applied. Of the pollutants examined, PM_{10} was associated most consistently with mortality. RRs in Table 5-8 are given for for Poisson regression models. Of the various model types, the ordinary least squares model with 36 binary monthly terms provided the lowest RR (1.04), which basically was unchanged when PM_{10} was coregressed with either SO_2, NO_2, or O_3.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Ponce de Leon et al., 1996	Time series	Apr. 1987 to Feb. 1992	14.6 ± 7.7 (Mean 24-h Avg BS ± SD) 6-27 (5^{th}–95^{th} Percentile 24-h Avg BS)	As part of the APHEA project, the following approach was followed: Poisson regression analysis adjusting for trend, seasonal and other cyclical factors, day of week, holidays, influenza epidemic, temperature, humidity, and autocorrelation. Lags examined. No control admission category analyzed. The most consistent association with respiratory admissions was with O_3. Very few significant associations found for other pollutants. Probable negative confounding of BS by O_3.	No
Pönkä and Virtanen, 1996a	Time series	1987-1989	76 (Mean 24-h Avg TSP)	Poisson regression analysis adjusted for long-term trend, season, epidemics, day of week, holidays, temperature, and RH using protocol of APHEA project. Corrected for autocorrelation. Modeling was problematic. Significant associations seen with control admission category and O_3.	No
Pönkä and Virtanen, 1996b	Time series	1987-1989	76 ± 51.6 (Mean ± SD 24-h Avg TSP) 6-414 (Range 24-h Avg TSP)	Poisson regression models controlled for long-term trends, season, and day of week, and influenza epidemics with dummy variables. Weather controlled using a smoothing technique. Lags examined. Autocorrelation addressed. Significant associations of ER ischemic cardiac admissions with NO and of ER cerebrovascular admissions with NO_2. Long-term transient ischemic attack admissions were positively but insignificantly associated with TSP. Temperature, RH, and SO_2 had no significant effects on admissions.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Schouten et al., 1996	Time series	1977-1989	11 (Mean 24-h Avg BS, Amsterdam) 26 (Mean 24-h Avg BS, Rotterdam)	Following APHEA approach, used autoregressive Poisson regression allowing for overdispersion and controlling for season, weather, and influenza epidemics. Lags investigated. BS data available for only 4 y (1986-89). BS did not show any clear effects in Amsterdam; in Rotterdam, it was positively but insignificantly related to admissions. Number of daily admissions was small. No control admission category analyzed.	No
Schwartz, 1996	Time series	1988-1990	46 (Mean 24-h Avg PM_{10}) 16-83 (10^{th}-90^{th} Percentile 24-h Avg PM_{10})	Poisson regression analysis (using the generalized additive model) controlled for long wavelength patterns, weather, and day of week. Lags considered. SO_2 levels trivial. Both PM_{10} and O_3 significantly associated with respiratory hospital admissions. Were too few days when both pollutants were measured to fit a two-pollutant model, but correlation between PM_{10} and O_3 was low (0.26). Examined sensitivity analyses to control for weather. No control admission category analyzed.	Yes
Schwartz et al., 1996	Time series	1988-1990	43 (Mean 24-h Avg PM_{10}) 19-72 (10^{th}-90^{th} Percentile 24-h Avg PM_{10})	Analysis of Cleveland data was used as an example in a review paper of methodologies in time series studies. Long-term trends in the data were removed by including linear and quadratic terms in the model. Seasonality removed by including sinusoidal terms. Dummy variables for categories of temperature and RH and for day of week included. Individual pollutants then tested. O_3 and PM_{10} significantly associated with respiratory hospital admissions. No control admission category analyzed.	Yes?

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Stieb et al., 1996	Time series	1984-1992 (May-Sept.)	36.7 (Mean 24-h TSP) 5-108 (Range 24-h TSP)	Daily ER visit frequencies filtered to remove day-of-week and long-wave trends. Filtered values regressed on air pollution and weather variables allowing for Poisson over- and underdispersion. Lags examined. First-order autocorrelation assessed. O_3 weakly and positively correlated with TSP and SO_4^{2-}. Only O_3 appeared to be related nonlinearly to ER visits. Single- and two-pollutant models used. Only O_3 exhibited a consistently positive association with asthma visits. Effect of O_3 not influenced by addition of co-pollutants into multivariate models, but a particulate effect could not be ruled out because of the limited number of sampling days for SO_4^{2-} and TSP. No control ER visit category analyzed.	No
Sunyer et al., 1996	Time series	1985-1991	49.7 (Median 24-h Avg BS, Winter) 11.4-66.7 (Range 24-h Avg BS, Winter) 35 (Median 24-h Avg BS, Summer) 10.6-125.6 (Range 24-h Avg BS, Summer)	APHEA protocol followed using separate Poisson regression models for each pollutant and mortality category. Controlled for temperature, RH, year, season, day of week, influenza, and autocorrelation. Lags assessed. BS and SO_2 highly correlated. O_3 negatively correlated with BS. O_3, SO_2, and NO_2 also significantly related to total, elderly, and cardiovascular mortality. Association of each pollutant with respiratory mortality not significant. CO, a possible confounder for the cardiovascular effect, not measured.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Verhoeff et al., 1996	Time series	1986-1992	12 (Mean 24-h Avg BS) 4-23 (10^{th}-90^{th} Percentile 24-h Avg BS)	Poisson regression analysis controlling for seasonal and other long-term temporal patterns using indicators for year, month, day of week, influenza epidemics, and weather. Lags considered. Overdispersion and autocorrelation addressed. The association of BS with mortality remained significant when BS was coregressed separately with each of the other pollutants. PM_{10} and O_3were positively associated with mortality, but the association with PM_{10} was nonsignificant.	No
Zmirou et al., 1996	Time series	1985-1990	38.05 (Mean 24-h Avg PM_{13}) 2.67-179.81 (Range 24-h Avg PM_{13})	Followed Poisson time series regression protocol established by APHEA members including controls for long-wave seasonality, day of week, temperature, RH, influenza epidemics, and autocorrelation. Lags examined. SO_2 was only pollutant that showed a clear association with mortality. Daily mortality counts low.	No
Abbey et al., 1995	Cohort	1973-1987	55.77 (Mean 24-h Avg PM_{10}) 0-90.74 (Range 24-h Avg PM_{10})	Long-term ambient PM_{10} levels were estimated from TSP levels. Separate multivariate statistical models were developed for airway obstructive disease, chronic bronchitis, and asthma. New cases of disease were analyzed by multiple logistic regression models. The RR for new cases of asthma was not statistically significant.	Yes

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Castellsague et al., 1995	Time series	1985-1989	48.2 (Mean 24-h Avg BS, Summer) 52 (Mean 24-h Avg BS, Winter)	Poisson regression controlled for weather, long-term and shorter trends, day of week, autocorrelation and overdispersion. Analyses conducted separately for summers and winters. Lags examined. Pollutants analyzed individually because of collinearity. Small but significant association of particulate matter and NO_2 with asthma ER visits. BS was associated with asthma visits in summer but not in winter. No control admission category analyzed.	No
Kinney et al., 1995	Time series	1985-1990	58 ± 23 (Mean 24-h Avg PM_{10} ± SD) 15-177 (Range 24-h Avg PM_{10})	Evaluation of methods used in controlling for temporal cycles and weather, examining effects of co-pollutants, and regression models. Sensitivity of RR for mortality/PM_{10} to the various methods and models was low, but data set was small (n = 364). Single- and two-pollutant models analyzed. PM_{10} effect appeared stronger than that of O_3 because the PM_{10} RR was the same whether PM_{10} was regressed singly or with O_3. CO also associated with mortality. PM_{10} and O_3 substantially correlated.	No
Moolgavkar et al., 1995	Time series	1973-1988	66.3-74 (Range of Seasonal Means 24-h Avg TSP)	Poisson regression. Controlled for effect of weather by analyzing seasons separately and including quintiles of temperature. Checked for autocorrelation. Investigated lags. RR in Table 5-8 given for model in which both TSP and SO_2 were included. TSP effect became nonsignificant when O_3 was added to the model. SO_2 in spring and winter and O_3 in summer were significantly and independently associated with mortality.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Pope et al., 1995	Cohort	1982-1989	18.2 (Mean 24-h Median $PM_{2.5}$) 9.0-33.5 (Range 24-h Median $PM_{2.5}$)	Multiple regression analysis based on Cox proportional hazards models. SO_4^{2-} levels were from 151 cities in 1980; $PM_{2.5}$ levels, from 50 cities in 1979-1983. Risk ratios for $PM_{2.5}$ were adjusted for age, sex, race, cigarette smoking, exposure to passive cigarette smoke, body-mass index, drinks per day of alcohol, education, and occupational exposure. Lung cancer mortality was associated with SO_4^{2-} but not $PM_{2.5}$. Noncardiopulmonary mortality was not significantly associated with pollution levels. Results from an ecologic comparison of total mortality by the authors for the same cities were consistent with those from the prospective cohort study.	Yes
Saldiva et al., 1995	Time Series	1990-1991	82.4 ± 38.8 (Mean 24-h Avg PM_{10} ± SD)	Gaussian regression models controlling for month, temperature, RH, day of week, and autocorrelation. Lags examined. Pollutants analyzed individually and together. Seasons evaluated separately. Various sensitivity analyses performed. The regression model was re-estimated using Poisson regression. The association of mortality with PM_{10} was the most statistically significant and independent of other air pollutants. The association was consistent across various model specifications and estimation techniques. The dose-response relationship between mortality and PM_{10} was nearly linear.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Schwartz, 1995	Time series	1988-1990	41 (Mean 24-h Avg PM_{10}), 19-67 (10^{th}-90^{th} Percentile 24-h Avg), New Haven 37 (Mean 24-h Avg PM_{10}), 14-67 (10^{th}-90^{th} Percentile 24-h Avg), Tacoma	Multiple regression analysis on temperature, dew point temperature, and air pollutants. A moving average filter removed seasonal and subseasonal patterns from the data. Poisson regression using single- and two-pollutant models. Autoregression examined. Strongest evidence for an independent association was for PM_{10} followed by O_3. RRs in Table 5-8 are for single-pollutant models. The PM_{10} association was little changed by control for either O_3 or SO_2. No control admission category analyzed.	Yes
Schwartz and Morris, 1995	Time series	1986-1989	48 (Mean 24-h Avg PM_{10}) 22-82 (10^{th}-90^{th} Percentile 24-h Avg PM_{10})	Poisson regressions controlled for seasonal and other long-term temporal trends, day of week, temperature, dew point temperature, and autoregression. Several sensitivity analyses to weather performed. Lags up to 2 days examined. Single- and two-pollutant models analyzed. PM_{10} and CO showed strongest associations with cardiovascular disease. RRs in Table 5-8 are for single-pollutant models. In two-pollutant models, PM_{10} remained significant after controlling for other pollutants that were significant in single-pollutant models. No control admission category analyzed.	Yes
Delfino et al., 1994	Time series	1984-1988	29.5 ± 12.0 (24-h Avg PM_{10} ± SD, May-Oct.)	Regression analysis controlled for season, day of week, autocorrelation, temperature, and RH. Examined lags. Used single- and two-pollutant models. PM_{10} and SO_4^{2-} estimated from meteorological variables. SO_4^{2-} significantly associated with respiratory nonasthma admissions. Pollutants highly correlated. Relatively small sample size.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Pönkä and Virtanen, 1994	Time series	1987-1989	76 ± 51.6 (Mean 24-h Avg TSP ± SD) 6-414 (Range 24-h Avg TSP)	Poisson regressions controlled for weather, season, time trends, day of week, and influenza. Autocorrelation assessed. Lags 0 to 7 days included. No significant association between all admissions and pollutants. Significant association of SO_2 with ER admissions for ages < 65 y and of NO_2 with hospital admissions for ages > 65 y. No control admission category analyzed. Correlations among predictor variables not given. TSP measurements available for only 2/3 of days.	No
Schwartz, 1994a	Time series	1986-1989	45 ppb (Mean 24-h Avg PM_{10}) 19-77 (10^{th}-90^{th} Percentile 24-h Avg PM_{10})	Poisson regression analysis controlling for time trends, seasonal fluctuations, and weather (but not day of week) using several alternative approaches. If serial correlation was found, autoregressive Poisson models were estimated using GEE. Used only single-pollutant models. The PM_{10} results were not sensitive to alternative methods for controlling for seasonal patterns nor to the exclusion of very hot or cold days. No potentially confounding pollutants other than O_3 and no control admission category were analyzed.	Yes

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Schwartz, 1994b	Time series	1986-1989	48 (Mean 24-h Avg PM_{10}) 22-82 (10^{th}-90^{th} Percentile 24-h Avg PM_{10})	Regression analysis controlling for weather and long-term temporal and seasonal patterns (two methods compared), but not day of week. Autocorrelation tested for and, if present, autoregressive Poisson models estimated using GEE. Lags considered. Both PM_{10} and O_3 significantly associated with pneumonia and COPD. RRs given in Table 5-8 are for two-pollutant models. The RRs were unchanged from those for the single-pollutant models. No potentially confounding pollutants other than PM_{10} and no control admission category analyzed.	Yes
Schwartz, 1994c	Time series	1986-1989	36 (Mean 24-h Avg PM_{10}) 18-58 (10^{th}-90^{th} Percentile 24-h Avg PM_{10})	Poisson regression analysis controlled for time trends, season, and weather. Lags examined. Results not sensitive to any of several approaches examined to control for confounders. No potentially confounding pollutants other than O_3 and no control admission category analyzed. RRs in Table 5-8 are for single-pollutant models. The association of PM_{10} and pneumonia was unchanged when the pollutants were coregressed. O_3 was insignificantly associated with COPD admissions.	Yes

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Thurston et al., 1994	Time series	July-Aug., 1986-1988	17.7/66.0 (Mean/Max 24-h Avg $PM_{2.5}$, 1986) 15.8/63.0 (Mean/Max 24-h Avg $PM_{2.5}$, 1987) 22.3/50.7 (Mean/Max 24-h Avg $PM_{2.5}$, 1988)	Ordinary least squares regression analysis controlled for long-wave trends, day of week, and temperature. Lags and autocorrelation examined. Single- and two-pollutant models employed. Sensitivity analyses conducted for O_3 and H^+, the pollutants most strongly associated with respiratory admissions. SO_4^{2-} also significantly associated with respiratory admissions. None of the associations of the other pollutants remained consistently significant after controlling for temperature.	No
Abbey et al., 1993	Cohort	1977-1987	394.6 (Mean avg annual h in excess of 200 $\mu g/m^3$ TSP, 1973-87)	Separate multiple logistic regression models used to study associations between long-term cumulative ambient concentrations of air pollutants and new cases of definite symptoms for airway obstructive disease, chronic bronchitis, and asthma. Used multivariate models to adjust simultaneously for a number of covariates. Ran stepwise regression analyses separately for each pollutant, then fit multi-pollutant models. Both TSP and O_3 appeared associated with new cases of asthma and change in asthma severity score, but the two pollutants were highly correlated.	Yes
Ostro et al., 1993	Time series	Sept. 1978 to Mar. 1979	11.55 ± 5.19 (Mean 24-h Avg COH/100 ft ± SD)	Multiple logistic regression of pooled data accounting for heterogeneity. Each pollutant modeled separately. Other covariates: sex, day of study, gas stove use, and presence of chronic respiratory disease. Associations found for O_3 and SO_4^{2-} with lower respiratory tract symptoms, but no association found with COH.	No

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Schwartz et al., 1993	Time series	Sept. 1989 to Sept. 1990	29.6 ± 18 (Mean 24-h Avg PM_{10} ± SD)	Poisson regressions controlling for weather, season, time trends, age, hospital, and day of week. GEE used to adjust for serial correlation. No significant associations between asthma ER visits and SO_2, O_3 or between PM_{10} and the control admissions category.	No
Dockery et al., 1992	Time series	Sept. 1985 to Aug. 1986	27.6/1-97 (Mean/Range 24-h Avg PM_{10}, St. Louis) 30/4-67 (Mean/Range 24-h Avg PM_{10}, Eastern Tennessee)	Poisson regression controlled for seasonal and weather effects. Examined lags. Models estimated by GEE. Single-pollutant models used. Power to detect associations limited because of short study period. In Tennessee, the association of mortality and PM_{10} was not statistically significant, but the estimated increase in total mortality was similar to that for St. Louis.	No
Pönkä, 1991	Time series	1987-1989	76.3 ± 51.6 (Mean 24-h Avg TSP ± SD) 6-414 (Range 24-h Avg TSP)	Multiple regression analysis standardized for minimum temperature. In the step-wise analysis, NO and O_3 were the pollutants most strongly associated with admissions. Long-wave patterns and autocorrelation not examined.	No
Pope, 1991	Time series	April 1985 through March 1989	53, 55, and < 40 (Annual Ave PM_{10} for Utah, Salt Lake, and Cache Valleys respectively	Autoregressive regressive models estimated using maximum-likelihood estimation. Monthly hospital admissions for pneumonia, bronchitis, and asthma were regressed on a trend variable, mean monthly PM_{10}, and monthly mean low temperature. SO_2, NO_2, and O_3 low. Significant correlations between respiratory hospital admissions for all ages and previous month's mean PM_{10} for Salt Lake Valley. Significant negative correlations between hospital admissions and low temperature.	Yes

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Sunyer et al., 1991	Time series	1985-1986	72.9 (Mean 24-h Avg BS) 39-310 (Range 24-h Avg BS)	Multivariate linear regression models controlled for weather, season, day of week, and autocorrelation. Lags tested. Air pollutants analyzed separately because of their collinearity. Weak but statistically significant associations between ER COPD admissions and SO_2, BS, and CO.	No
Ostro and Rothschild, 1989	Time series	1976-1981	20.73-22.59 (Range of Mean Annual $PM_{2.5}$)	Multiple regression including an air pollution variable, age, sex, race, education, family income, quarter of the year, marital status, existence of a chronic condition, and avg of daily minimum temperatures duing the 2-week recall period. A Poisson distribution was used to model event counts over time. A fixed effects model controlled for intercity differences demonstrated an association between $PM_{2.5}$ (estimated using daily airport visibility data) and both minor restrictions in activity and respiratory conditions severe enough to result in work loss and bed disability. Acute respiratory data were obtained from the annual Health Interview Survey, a national probability survey of 50,000 households.	Yes

Table 5-9. Supporting information for studies of the effects of particulate matter in adults shown in Table 5-8.

Reference	Study Type	Study Period	PM Levels ($\mu g/m^3$)	Statistical Analysis and Remarks	Included in Valuation
Ostro, 1987	Time series	1976-1981	Not reported	A Poisson distribution was used to model health endpoints measured for a period of 2 weeks before the survey day. A fixed effects model controlled for intercity differences. Screened visual range data from 49 metropolitan cities was used to estimate fine PM, which was used in the model as 2-week averages lagged 2 periods. Control variables included age, sex, race, education, income, survey quarter, marital status, existence of a chronic condition, average 2-week minimum temperature, and working status. A unique sample of about 12,000 adults from the Health Interview Survey was studied in all 6 years. In each year, the regression coefficient of fine PM was positively and significantly related to restricted activity days.	Yes

Abbreviations:

24-h Avg = average value for a 24-h period
1-h Max = daily maximum hourly level
APHEA = Air Pollution and Health, a European Approach
Avg = average
BS = black smoke
b_{sp} = scattering coefficient of particles measured by nephelometry
CI = confidence interval
CO = carbon monoxide
COH = coefficient of haze; an optical measure of airborne particulate matter
COPD = chronic obstructive pulmonary disease
d = day
ER = emergency room
FEV_1 = forced expiratory volume in one second
GEE = generalized estimating equations
h = hour
H^+ = hydrogen ion
LVD = local visual distance; a measure of particles of size range 0.1-2.5 µm
Max = maximum
n = number of variates in the distribution
NO = nitric oxide
NO_2 = nitrogen dioxide
O_3 = ozone
p = probability
PEF = peak expiratory flow
ΔPEF = morning to evening change in peak expiratory flow
PM = particulate matter
$PM_{2.5}$ = particulate matter ≤2.5 µm aerodynamic diameter
PM_{10} = particulate matter ≤10 µm aerodynamic diameter
ppb = parts per billion
RH = relative humidity
RR = relative risk
SD = standard deviation
significant = $p < 0.05$ unless stated otherwise
SO_2 = sulfur dioxide
SO_4^{2-} = sulfate
Time series= aggregate-level daily time series studies
TSP = total suspended particulate matter
y = year

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Braun-Fahrlander et al., 1997	Cross-sectional	10-33 (range of annual means of PM_{10} among cities)		✓		+	4,470 children 6-15 y in 10 communities in Switzerland during school year 1992/1993. O_3, PM_{10}, NO_2, and SO_2 evaluated. Respiratory and allergic symptoms assessed. Strongest association was found for PM_{10}. Adjusted ORs (95% CI) for chronic cough, nocturnal dry cough, bronchitis, and conjunctivitis between the most and least polluted communities for PM_{10} were 3.07 (1.62-5.81), 2.88 (1.69-4.89), 2.17 (1.21-3.87) and 2.11 (1.29-3.44) respectively. Asthma, current wheeze, sneezing attacks during pollen season, and hay fever were not associated with PM_{10} nor was the reference symptom diarrhea. Similar results (but somewhat smaller associations) were found for NO_2 and SO_2, which were closely correlated with PM_{10}.	Logistic regression. Fog included in all models. Separate models for atopy. Random area effect models adjusted for autocorrelation.

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Delfino et al., 1998	Panel	57 ± 16 (mean 1-h max PM_{10} ± SD) 43 ± 12 (mean 8-h max PM_{10} ± SD) 31 ± 8 (mean 24-h avg PM_{10} ± SD)		✓		+	24 asthmatics ages 9-17 y, Alpine, CA, studied for 42-91 days, Aug. through Oct. 1995. O_3 and PM_{10} evaluated. Subjects kept daily diary reports of asthma symptom severity. The OR (CI) for asthma symptoms per increase to 90th percentile PM_{10} level for current day 1-h max in model adjusting for O_3 was 1.63 (1.17-2.28). The OR for current day 8-h max PM_{10} was similar, but for current day 24-h avg was not significant. The largest effect was for 5-day moving average 8-h max PM_{10}.	Regression analysis utilizing GEE. Confounders tested: day of week, temperature, RH, and wind speed.

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level (µg/m^3)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Gielen et al., 1997	Panel	30.5 ± 10.1 (mean 24-h avg PM_{10} ± SD)		✓		+	61 asthmatics, 7-13 y (77% taking asthma medication), Amsterdam, the Netherlands, Apr. 26 to July 4, 1995. O_3, PM_{10}, and BS evaluated. Five-d mean PM_{10} prevalence ratio (95% CI) for lower respiratory symptoms was 1.47 (1.00-1.94) and for bronchodilator use was 1.61 (1.16-2.06). Five-d mean BS was significantly related to upper and lower respiratory symptoms, eye irritation, nocturnal awakenings, and bronchodilator use.	Individual multiple linear regression controlled for pollen, time trend, and day of week. Time series analysis corrected for autocorrelation.
					✓	–	Significant associations were found between morning PEF and 2-d lag and 5-d mean BS, and between evening PEF and 1-d lag and 5-d mean BS. For PM_{10}, none of the associations with PEF were significant.	

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Hoek and Brunekreef, 1995	Daily life	48 ± 22 (mean 24-h avg PM_{10} ± SD), Deurne 36 ± 21 (mean 24-h avg PM_{10} ± SD), Enkhuizen		✓		None	300 children, 7-10 y, in two rural towns in the Netherlands, spring and summer 1989. O_3, SO_2, NO_2, PM_{10}, H^+, SO_4^{2-}, and NO_3^- measured (SO_2, NO_2, H^+ low). Acute respiratory symptoms recorded by parents in a diary. No association of symptoms with same- or previous-day O_3, PM_{10}, SO_4^{2-}, or NO_3^-.	Logistic regression model that accounted for autocorrelation. Potential confounders considered: trends in symptom prevalence, day of week, and weather variables.
Keiding et al., 1995	Time series	22 (yearly mean BS, 1990)	✓			None	Contacts with Copenhagen Emergency Medical Service (3,974), Jan. 14 to Apr. 14, 1991, for respiratory illnesses by children 0-15 y old. O_3, CO, SO_2, NO_x, and BS evaluated. Only NO and NO_x were associated significantly with contacts.	Weighted multiple regression analysis controlled for temperature, RH, holidays, and weekends.

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Linn et al., 1996	Daily life	24 ± 21 (mean 24-h avg PM_5 ± SD)			✓	–	269 children during 4th and 5th grade school years in 3 southern California communities, 1992-93 and 1993-94. O_3, NO_2, and PM_5, temperature evaluated. Lung function and symptoms measured twice daily for 1 week each in fall, winter, and spring; time-activity and personal exposure data collected as well. PM_5 was most strongly associated with lung dysfunction. Morning FVC for previous 24-h PM_5 and ΔFEV_1, p.m.-a.m., for current 24 h were significantly and negatively related with PM_5.	Repeated-measures ANOVA was the primary analytic tool. Tested only relatively simple ANOVA/ regression models that included one or two environmental variables, year, season, year-by-season interaction, and day-of-week.
				✓		None	No tested symptom/pollutant relationship was significant.	

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Neas et al., 1995	Daily life	35.6 (mean 24-h avg PM_{10}) 24.5 (mean 24-h avg $PM_{2.1}$		 ✓	✓	– +	83 4th and 5th graders in Uniontown, PA, summer 1990. O_3, SO_2, PM_{10}, $PM_{2.1}$, SO_4^{2-}, H^+, and temperature examined. A 20 $\mu g/m^3$ increment in 24-h avg PM_{10} was associated with a –1.23 l/min change in evening PEFR (95% CI, -2.30 to 0.17). Significant association between incidence of cough episodes and 15 $\mu g/m^3$ increments of $PM_{2.1}$ after adjusting for trend, time of observation, temperature, and autocorrelation (OR = 1.25, 95% CI = 1.04-1.50). $PM_{2.1}$ also significantly related to cough episodes when concentration was weighted by proportion of hours spent outdoors during prior 12 h.	Autogressive linear regression model for mean deviation in PEFR. Autoregressive logistic regression model using a modified GEE approach for symptoms.

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Ostro et al., 1995	Panel	55.9 ± 15.2 (mean 24-h avg PM_{10} ± SD)		✓		+	83 African-American asthmatics, 7-12 y, Los Angeles, CA, 13 weeks in summer, 1992. O_3, PM_{10}, NO_2, SO_2, pollens, molds, and weather examined. In pooled analysis, same-day mean PM_{10} significantly associated with shortness of breath (OR = 1.58, 95% CI = 1.05-2.38). PM_{10} also significantly associated with shortness of breath in individual-level analysis (OR = 2.42, 95% CI = 1.76-4.68). O_3 was associated with increased shortness of breath as well. PM_{10} and O_3 levels were correlated making their effects difficult to separate.	Stepwise regression for group-level and pooled analyses. Pooled logistic regressions based on GEE. Individual regressions.
Pönkä, 1991	Time series	76.3 ± 51.6 (24-h avg TSP ± SD)	✓			None	Admissions (4,209) for asthma attacks in Helsinki, 1987 through 1989, for 0-14, 15-64, 65+ y. Evaluated O_3, SO_2, NO_2, NO, CO, TSP, temperature, wind speed, RH, and lags. For subjects 0-14 y, only O_3 and NO correlated with admissions.	Regression analysis. Correlations calculated separately for weekdays and weekends. Stepwise regression.

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Pope, 1991	Time series	53, 55, and < 40 (annual avg PM_{10} for Utah, Salt Lake, and Cache valleys)	✓			+	Respiratory hospital admissions data in 3 mountain valleys in central and north-central Utah collected. Largest number of admissions was for preschool age group (0-5 y). Significant correlations between respiratory hospital admissions and mean monthly PM_{10} and previous month's mean PM_{10} in Utah and Salt Lake valleys and with previous month's low temperature in Salt Lake Valley. The correlations were stronger preschool age than for all ages and for lagged PM_{10} than for current month PM_{10}.	Autoregressive regession models estimated using maximum-likilihood estimation. Monthly hospital admissions for pneumonia, bronchitis, and asthma for preschool-age children regressed on a trend variable, mean monthly PM_{10}, and monthly mean low temperature.

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Romieu et al., 1996	Panel	166.8 ± 72.8 (mean 24-h avg PM_{10} ± SD) 85.7 ± 30.2 (mean 24-h avg $PM_{2.5}$ ± SD)		✓		+	71 asthmatics, 5-13 y, in northern Mexico City, Apr. 24 to July 7, 1991 and Nov. 1, 1991 to Feb. 28, 1992. None taking medication regularly. O_3, PM_{10}, $PM_{2.5}$, NO_2, and SO_2, measured. An increase of 20 $\mu g/m^3$ PM_{10}, or of 10 $\mu g/m^3$ $PM_{2.5}$, was significantly associated with increases of 8-10% in cough, phlegm, and lower respiratory illness on the same day as exposure. PEFR was strongly associated with PM_{10}. The mean deviation of evening PEFR associated with an increase of 20 $\mu g/m^3$ PM_{10} was –1.92 (95% CI = -3.20 , -0.68) l/min and with an increase of 10 $\mu g/m^3$ $PM_{2.5}$ was -1.71 (95% CI = -3.09, -0.34) l/min on the same day as exposure.	Autoregressive logistic regression models or GEEs used for symptom analysis. Autoregressive linear regression models or GEEs adjusted for minimum temperature as well as multivariate models used for PEFR analysis.
					✓	–		

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Romieu et al., 1997	Panel	54.2 ± 24.8 (mean 24-h avg PM_{10} ± SD)		✓		None	65 asthmatics, 5-13 y, SW of Mexico City, Apr. 24 to July 7, 1991 and Nov. 1, 1991 to Feb. 28, 1992. None taking medication regularly. O_3, NO_2, and SO_2 measured. PM_{10} and O_3 evaluated. Although an increase of 20 $\mu g/m^3$ PM_{10} was associated with a 5% increase in difficulty breathing, no significant effect of PM_{10} was found in models that included O_3, PM_{10}, and minimum temperature simultaneously. PM_{10} and O_3 levels were correlated (r = 0.47).	Autoregressive logistic regression models or GEEs used for symptom analysis. Autoregressive linear regression models or GEEs adjusted for minimum temperature and multivariate models used for PEFR analysis.
					✓	None	Effects of PM_{10} on PEFR were small.	

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Schwartz et al., 1994	Diary	30.0 (median 24-h avg PM_{10})		✓		+	1,844 children grades 2-5 in six US cities studied for 1 y starting Sept. 1984. Analyses limited to Apr.-Aug. O_3, SO_2, NO_2, PM_{10}, $PM_{2.5}$, SO_4^{2-}, H^+, and temperature evaluated. O_3 and PM_{10} independently and significantly associated with cough incidence; PM_{10} somewhat more strongly associated. A 30 $\mu g/m^3$ change in previous-day 24-h PM_{10} was associated with an OR of 1.53 (95% CI, 1.20-1.95) for lower respiratory symptoms and an OR of 1.22 (95% CI, 1.03-1.45) for cough. The association of PM_{10} with upper respiratory symptoms was marginally significant.	Incidence of respiratory symptoms regressed separately against each pollutant controlling for previous day temperature, day of week, and city of residence. Significant pollutants analyzed in multiple pollutant models.
Studnicka et al., 1995	Summer camp	10.3 ± 4.4 (mean 24-h avg PM_{10} ± SD, panel 1)			✓	−	133 children ≥7 y old, each studied for 3 weeks, Austrian Alps. 3 panels, June 28 to Aug. 28, 1991. O_3, H^+, SO_4^{2-}, NH_4^+, PM_{10}, temperature, RH, and pollen measured. Daily spirometry, symptoms, and exposure recorded. For panel 1 (highest pollutant levels), PM_{10} and O_3 were significantly and negatively associated with FEV_1 and FVC.	Linear regression analyses for repeated measures adjusted for sex, height, and age. Included correction for within-subject correlation.

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level ($\mu g/m^3$)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Sunyer et al., 1997	Time series	13-40 (range of median 24-h avg BS among cities)	✓			None	Asthma admissions (50.3/d) in Barcelona, Helsinki, London, and Paris, 1986-92, for ages < 15 y and 15-64 y. Emergency admissions for 1st three cities. O_3, SO_2, NO_2, and BS evaluated. BS not measured in Helsinki. In children, admissions increased significantly with SO_2 and nonsignificantly with BS and NO_2. Collinearity between BS and NO_2 may have limited separation of their effects.	Poisson regression models (APHEA project approach). Combined associations estimated using meta-analysis.

Table 5-10. Summary of studies relating particulate levels with health effects on children.

Reference	Study Type	Daily PM Level (µg/m^3)	Category of Health Effect			Association	Description and Results	Statistical Methods
			Hospital Admissions	Respiratory Symptoms	Lung Function Changes			
Tseng et al., 1992	Time series	54-136 (range quarterly means of mean monthly TSP data)	✓			+	Quarterly hospital discharges (33,953) for asthma in Hong Kong, 1983-1989. Age groups 0-1, 1-4, 5-14, and 15+ y. Examined O_3, TSP, SO_2, NO_2, NO_x, and PM_{10}. Quarterly mean TSP was strongly correlated with hospital discharge rate for children 1-4 y (r = 0.62, p < 0.001).	Univariate and stepwise multiple regression analysis. Controls lacking for day of week, season, and long-wave periodicities.

✓ Indicates which category was studied.
\+ Indicates that the association between the health effect and PM exposure was significantly positive.
– Indicates that the association between the health effect and PM exposure was significantly negative.

Abbreviations:

24-h avg = average value for a 24-h period
1-h max = daily 1-hour maximum
8-h max = maximum value for a daily 8-h time period
ANOVA = analysis of variance
APHEA = Air Pollution and Health, a European Approach
Avg = average
BS = black smoke
CI = confidence interval
CO = carbon monoxide
d = day
FEV_1 = forced expiratory volume in one second
ΔFEV_1 = change in forced expiratory volume in one second
FVC = forced vital capacity
GEE = general estimating equations
h = hour
H^+ = hydrogen ion
l = liter
Lag = number of days (time lag) between exposure and effect
Max = maximum
Min = minute
NH_4^+ = ammonium ion
NO = nitric oxide
NO_2 = nitrogen dioxide
NO_3^- = nitrate
NO_x = oxides of nitrogen
O_3 = ozone
OR = odds ratio
p = probability
PEF = peak expiratory flow
PEFR = peak expiratory flow rate
PM = particulate matter
$PM_{2.1}$ = particulate matter ≤2.1 µm aerodynamic diameter
$PM_{2.5}$ = particulate matter ≤2.5 µm aerodynamic diameter
PM_5 = particulate matter ≤5 µm aerodynamic diameter
PM_{10} = particulate matter ≤10 µm aerodynamic diameter
r = Pearson correlation coefficient
RH = relative humidity
SD = standard deviation
significant = p < 0.05
SO_2 = sulfur dioxide
SO_4^{2-} = sulfate
Time series = aggregate-level time series studies
TSP = total suspended particulate matter
y = year

6. ECONOMIC VALUATION

6.1 THE BASIS FOR VALUE

Why do we assign dollar values at all, and what is the basis for those values? The most basic answer to the first question is that society does not have the material resources to do all of the good things that we would like to be able to do. Therefore, we must choose among competing uses for our scarce resources. This is true of individuals and of society as a whole. The objective of placing dollar values on reduced health risks is to help determine whether resources are being used well. If we cannot do all good things, we can at least attempt to do as many and as much as possible, which implies a need to know the relative value of different actions.

Regarding the second question, we want a systematic way to make collective (social) choices, so we need to decide which environmental protections are the most worthwhile, that is, which ones will return the greatest value in improved health. We therefore need a means of comparison that is transparent, uses the best available information, and reflects social preferences. We also need a common denominator - a basis for comparison to weight each option, and we use dollars as the denominator.

To decide what dollar value to place on an adverse health effect, we begin with the premise that, with limited exceptions, we accept individual choices as valid. Other than restrictions like speed limits and proscriptions against murder and some environmentally harmful actions, we assume that what individuals choose to do accurately represents what is best for them, and by reference, for society. In economic benefit assessment, the sum of value to individuals equals social value. We use prices when available (or implied prices), and survey results to value changes in air pollution-related risks to health.

The ultimate objective of this study is a dollar measurement of the benefits of better health associated with lower levels of air pollution. This requires determining how much people value avoiding pollution-related health effects ranging from the most serious consequence (premature death) to relatively minor symptoms such as eye irritation. Value is gained from reducing:

- Direct medical costs and loss of work and school days that result from avoiding or treating adverse health effects.
- Discomfort, inconvenience, and fear resulting from adverse health effects, their treatment, or efforts to avoid them.
- Loss of enjoyment and leisure time.
- Impacts on others as a result of an individual's adverse health effects.

6.2 CONCEPTS AND MEASURES

Ideally, measurements of value would capture all of the losses to individuals and to society that result from adverse health effects. They would also reflect as closely as possible real preferences and decision-making processes similar to those we use daily to decide everything from where to live to what to have for dinner. Purchases of goods or services are based on which items give the most satisfaction, or utility, relative to prices and income. Prices paid are generally reasonable measures of the value of those items that can be purchased. However, clean air (like many other environmental goods) is an item for which no markets exist. Values for such goods cannot be assumed from directly observed prices. Economists have therefore developed alternative methods to measure the value of environmental benefits, including health improvements resulting from cleaner air.

Two generally accepted economic measures of changes in well-being (utility) due to the adverse health effects of air pollution are the cost of illness (COI) measure and the willingness to pay (WTP) (or willingness to accept (WTA)) measure. Both measures have drawbacks and weaknesses but, when used in conjunction, yield a reasonable range of values for the health benefits of improvements in air quality.

Actual data on health care costs and wages are used to calculate COI measures. WTA and WTP measures are calculated from either market-based (hedonic) studies or contingent valuation (survey) studies. Market-based approaches estimate value from the economic consequences of actual and observed behavior in relation to differing levels of risk. In comparison, contingent valuation studies rely on surveys of a sample of the affected population to elicit WTP or WTA for a good such as improved air quality that cannot be purchased in a market.[1]

This study combines the COI and WTP or WTA measures with estimates of human exposure, resulting adverse health impacts, and population characteristics to estimate the economic benefits of potential improvements in air quality.

6.2.1 Cost of Illness

The COI method was the first economic valuation method to be developed and described in the health and safety literature. It involves calculating direct medical costs and indirect costs (lost wages) due to illness. This method is still used to value the benefit of avoiding hospital admissions and other medical treatments. The COI method has the advantage of being based on real dollars spent to treat specific health effects and the actual market value of work time. Since it includes only monetary losses, however, and does not include losses associated with the value of leisure time, of school or unpaid work time, or of general misery, it does not capture all of the benefits of better health. The method is therefore generally

[1] The alternative pure human capital (PHC) approach has been largely avoided in studies of environmental benefits for a variety of reasons, although it is commonly applied in litigated compensation cases and related settlements.

viewed as somewhat limited. It basically defines a measure of the *financial* impact of illness, not the *change in well being* due to illness, since financial loss is only part of the effect of illness and malaise on well being.

Minimum value to avoid the adverse effects of unhealthful air quality is reflected in the estimated direct and indirect COI measures. Other factors, most notably the perception of lost utility or welfare that is associated with illness, can result in significant disparity between COI estimates and WTP or WTA estimates. As discussed in Section 6.5.3 below, the COI approach has been shown to produce a lower-bound value estimate. While medical cost and wage data are generally available, determining what portion of costs is attributable to pollution-related morbidity effects, and what should be attributed to co-factors, such as occupational exposure or smoking, is a complex undertaking. Therefore, COI measures may also capture costs unrelated to pollution. This effect is mitigated when the studies used to estimate the frequency of an effect account for such co-factors.

Overall, COI measures are used when more complete measures are unavailable for an effect. While they generally represent a lower bound of value, using them allows the valuation of some adverse effects, such as hospitalization, which would otherwise not be quantified.

6.2.2 Market-Based Measures

Willingness to pay (WTP) and willingness to accept (WTA) measures are preferred as alternatives to COI because these measures are more complete and the method is consistent with well-established economic theory about markets and how people make economic choices. Market transactions that result in changes in risks to health or life reveal, indirectly, the WTP for lower risks. Studies using this approach relate differences in price to differing degrees of risk to estimate the "demand" for reduced risk. Wage-risk and consumer-behavior studies that examine differentials in wages or consumer costs are examples of this approach. Although a safer environment is not a commodity whose price is directly observable in any market, responses to perceived risk can be observed and the costs people bear to reduce the potential harm can be measured. These costs are surrogates for market prices. If people accept lower wages or pay higher prices for a lower level of risk, or require higher pay or lower prices to accept a greater level of risk, the wage or cost differential indicates the value of the risk-related environmental improvement. Wage-risk studies typically indicate the WTA greater risk of job-related injury or death. The results of wage-risk studies must, however, be carefully interpreted. In particular, problems may arise from the presence of job attributes not directly related to the probability of harm. Job attributes unrelated to risk have been controlled for in later wage-risk studies (Leigh and Folsom, 1984; Gejax et al., 1985; Moore and Viscusi, 1988; Garen, 1988).

Early market-based studies of consumer behavior (Blomquist, 1979; Dardis,1980; Ippolito and Ippolito, 1984) produced value estimates that are generally regarded as less reliable than values from wage-risk studies because they failed to include all costs incurred by consumers to improve safety. More recent studies, such as Carlin and Sandy (1991) have introduced new analytical methods, but have not overcome this limitation. This group of

studies therefore underestimates WTP. Further, consumers often face multiple risks, compounding the difficulty of determining the value of reducing any single component of risk (Viscusi et al., 1987). One study by Atkinson and Halvorsen (1990) of WTP for automobile safety provides a more reliable measure of the general public's WTP for reduced risk of death. By carefully controlling for vehicle characteristics other than relative risk of death, this study estimated WTP for reduced risk of death comparable to the wage-risk studies (Viscusi, 1992).

6.2.3 Contingent Valuation

Willingness to pay or WTA can also be estimated from survey instruments. Personal preferences and values for certain goods that do not carry market prices (such as cleaner air), are determined from interviews and questionnaires, rather than inferred from related market choices. Interest in the contingent valuation (CV) method has increased significantly in the last decade and is accepted as a basis to assess compensation in resource damage litigation (such as the Exxon Valdez oil spill case). Like wage-risk measures of WTP, CV is conceptually preferable to COI methods because it is more inclusive. The value of lost enjoyment and life experience is included in CV responses. Because the CV method relies on surveys, however, it is costly; and the validity of results depends on careful sampling and study design and selection of an appropriate survey instrument. It is important, for example, to make the non-market good being valued as easy to understand as possible because respondents are being asked about hypothetical events. The range of values considered must also be bounded within a reasonable range.

Willingness to accept (WTA) measures, an alternative to WTP measures, involve determining what people would have to receive as compensation to accept a greater risk, rather than what they would be willing to pay to avoid a greater risk, or to receive a reduced risk. For practical reasons, WTP is a more common measure for CV assessments of the risks from hazards such as air pollution. Mitchell and Carson (1989) argue that WTA is difficult to estimate because of practical difficulties in survey design. Hanemann (1991) found, however, that WTP and WTA diverge substantially where there are few alternatives for "public goods", and that in such cases WTP is an underestimate of the true value. This may be an important consideration because cleaner air is an example of what economists call public goods. For this class of goods, non-payers cannot be excluded and one person’s consumption does not diminish another’s. It is not possible to clean up the air for one person; if the air is cleaner in a specific area, it will be cleaner for everyone who is exposed to it.

6.2.4 Comparing Methods

Willingness to pay values based on CV studies and WTA based on wage-risk studies presently constitute the most appropriate basis for valuing health risk (Viscusi, 1993 and references therein). Cost of illness is accepted when other measures of value are not available, to represent at least a lower bound of value. In this study, four criteria were used to determine which values are most appropriate:

- First, a measure of value should be appropriate for the type of risk. Risk can be differentiated by the degree of the risk (1 in 10,000 or 1 in 1,000,000), whether the risk is voluntary or involuntary, whether it is prospective or harm has already occurred, and the direction of the risk (that is, whether risk is increasing or decreasing).
- Second, the measure must provide the most comprehensive estimates possible. This means it should capture gains or losses in well being (utility) as fully as possible.
- Third, if similar values are derived from more than one type of study, from both wage-risk and CV studies, for example, such values have a greater presumption of validity than those derived solely from one method.
- Fourth, if several valid studies show results that converge for comparable adverse effects, the values reported in those studies are given greater weight.

Based on these criteria, and the preceding discussion, valuation methods are ranked. Contingent valuation results for WTP are most highly ranked, the WTA measures from wage-risk studies follow, along with WTP from one consumer behavior study, and finally COI studies. The pure human capital method used to establish damage awards in legal proceedings is not relied on, because it is a very incomplete measure and, since harm has already occurred and cannot be prevented, damage awards and settlements do not represent the value of reduced risk of morbidity or mortality. Additionally, these awards result from a complex legal process and are not independent measures of value.

6.3 SELECTION OF SPECIFIC VALUES FOR MORTALITY

Premature mortality is one of the most significant effects of exposure to elevated levels of atmospheric pollution. Consequently, the value that society attaches to reducing pollution-related early deaths is high in comparison to the value of reducing less severe effects. Before reviewing the alternative ways in which we might place an economic value on reductions in premature death, it is important to recognize that we are not attributing this value to the life of any identified individual, or group of individuals. We do not know whose life expectancy will be shortened if pollution concentrations do not decline. What we are doing is valuing the benefits of reducing a small risk to a large population. The degree of risk is not uniform, however, and varies across the population, depending largely on age and health status. The probability of early death declines as exposure to atmospheric pollution falls and it is really this declining risk – the change in the probability of death in a given year - that we are valuing. The value of reducing fine particle pollution is cognitively something like a lottery. Those who pay small sums to join the lottery do not know who will win, but someone will. By joining, they are increasing very slightly the odds that they will be the winner. One way of estimating the value of reducing mortality risks associated with air pollution is similar. What are people willing to pay to reduce the risk to the population and the number of premature deaths, and thereby reduce the risk to themselves? It is the sum that all individuals in the population at risk are willing to pay to change the level of risk, divided by the number of premature deaths expected to be averted, that represents what is often called the value of a life, or – more correctly – the value of a statistical life (VSL).

6.3.1 The Economic Value of Life

Most people know that courts and insurance companies determine how much to pay the survivors of people who die in accidents or due to another's negligence. Generally, these payments are based largely on the expected lifetime earnings of the individual who died. That is not how most risk-related social policies (including environmental regulation) relate to the value of a life, however. This is true for many reasons. Primarily, regulations are intended to *prevent* injury or death, reducing the severity of harm and number of victims, rather than to compensate survivors after the harm has been inflicted. Avoiding harm has greater social value than making after-the-fact efforts to ameliorate the loss with payment. Also, in the case of reducing environmental risk, a large population will benefit from small changes in risk. In the case of compensation for harm that has occurred, the benefits accrue solely to a small group of survivors.

For the most part, we assume that people value the things that they buy at least as much as the price they pay. A movie matinee must then be worth at least $3.50. We know how many people go to matinees, so we know the minimum value of those matinees to society - it is just the price multiplied by the number of tickets sold[2]. There is no similar market in which we can individually "purchase" changes in air pollution-related risk to health and life. Consequently, economists infer the willingness to pay (WTP) for small changes in risk to life due to environmental exposure either from studies that evaluate risk-dollar trade-offs in other settings, such as the workplace or in transportation, or from surveys.

Consider this case. There are 20,000 workers in an industry with a 1 in 10,000 annual risk of job-related death. This means that two workers [20,000 x 1/10,000] would probably die on the job annually, but no one knows who those workers will be. Now consider a similar industry, requiring the same job skills, where the risk is 1 in 20,000, reducing the likely annual deaths to one. If the workers in the relatively safer industry are each paid $250 a year less than those in the riskier industry, we can infer that the value of reducing risk enough to avert one death to that group of workers is $250 x 20,000 = $5 million. These are the basic theory and method underlying what are called wage-risk studies. Because they generally represent blue-collar workers, who typically have lower incomes than the older population most at risk of premature death due to fine particle exposure, these studies could underestimate the willingness to pay of better off individuals. Still, repeated over different time periods and large groups of workers, the results converge and represent real willingness to forgo higher income for lower risk to life. It is not explicitly a market for risk, but clearly represents economic choice in relation to risk.

A related group of studies, also based on actual market decisions that relate to risk, are based on consumer behavior and product risk. Car buyers, for example, are estimated to pay about $5 million for each life saved by the purchase of safer cars (Atkinson and Halvorsen,

[2] This is referred to as the minimum value because some of the people who paid $3.50 would be willing to pay $3.75, or $4.00, or more. They only have to pay the uniform price, however. The difference between the price paid and what some individuals were willing to pay is referred to as consumer surplus. It represents real value to the purchaser, but is not reflected in the aggregate paid for tickets.

1990). As with the wage-risk studies, this result represents a differential in car price multiplied by the number of car buyers and divided by the number of highway fatalities likely avoided by those who pay more for safer models. Again, the objective is to determine what a large group of people actually pays to reduce risk by a specific small amount. Both wage-risk and consumer product estimates are market-based measures.

An alternative way to estimate the value of a statistical life is to create a hypothetical (or contingent) market in a survey and ask a sample of the population what they are willing to pay to change risk by some amount. This is a common way to assess the benefits of safer transportation systems before design, construction, or regulation, and is referred to as the contingent valuation method (CVM). Fewer CVM studies are available, but their results generally fit into the narrower range of the wage-risk results. CVM studies are an important adjunct to the market-based measures because they offer the ability to ask about altruistic value (not just value to those directly at risk) and to investigate how value might change over a range of risks.

6.3.2 Studies That Assess the Value of a Statistical Life

A recent assessment of virtually all available estimates from published WTP and WTA studies (including wage-risk, consumer behavior, and contingent valuation studies) places most reported values of life in the range of $3.5 million to $8 million (Viscusi, 1993)[3]. The overall range is much broader, from about $1 million to over $15 million.

This range is explained by recognizing that the value of reducing risk varies with the nature of the risk. Is the risk voluntary or involuntary? What is the nature of the death that is risked - sudden, or slow and painful? What is the magnitude of the risk? Viscusi (1986) concluded that for involuntary risk or smaller risks (averaging less than a 1 in 10,000 probability of a job-related death in a year), the value of a statistical life for the "representative worker" was between $3 million and $5 million. An important caveat to this figure is that lower income groups are typical in wage-risk studies. When these wage-risk values are adjusted to account for this income bias (relative to the average income), the value of life rises significantly (Viscusi, 1993).

In later work, Moore and Viscusi (1988) determined that the value of life for a "representative worker" ranged from about $8 million to over $9 million. This conclusion is the result of comparing results from the same data used in earlier wage-risk studies with results derived from an additional data set. The original data set suffered from a serious downward bias, whereas results from the new data were statistically significant and yielded value of life estimates approximately double earlier figures. However, the 1988 results might also be compromised because the data did not allow sufficient disaggregation of industries to ensure that risks faced by workers in differing wage categories were reflected accurately. These results do demonstrate that earlier work is biased downward, but not by what degree.

[3] Throughout this report, unit values of adverse health effects have been converted to 1997 dollars.

The data set used by Viscusi and others to determine the wage premiums required to accept greater on-the-job risk was reanalyzed by Garen (1988). He used simultaneous equation analysis, a more sophisticated econometric technique than the ordinary least squares (OLS) method used by Moore and Viscusi. The Garen method values a statistical life closer to $12 million, contrasted to about half that value based on the same data but using OLS. He concluded that OLS underestimated the value of reducing risk and, therefore, the value of a statistical life.

The choices of car buyers were analyzed by Atkinson and Halvorsen (1990) to determine consumer WTP for lower risk of vehicular death. Price differences for different models were adjusted for factors other than safety, and the value of safer cars inferred from the premiums paid for safer models. Since fatality risks are known for various models, it was possible to directly associate prices and risk to estimate the value of life. This study produced an estimate of about $5 million. Because this study reflects actual market decisions of the general population, it is an important result.

Contingent valuation studies have resulted in estimates nearer the upper range of reported values. A mean value of more than $11 million to avoid an automobile-related death was reported by Viscusi (1991), with a median value closer to $3.5 million. The difference between the mean and the median suggests that some survey respondents may not have fully grasped the small degree of risk, and therefore bid very high values. Jones-Lee (1976), in one transportation-related survey, found a value of life over $19 million, in contrast to his 1986 report of highway safety values, which were closer to $5 million.

Finally, values of life above the middle of the range are supported by the assessment that the value of reducing small risks to the public, comparable to risks that result from exposure to atmospheric fine particles, ranges from about $5 million to over $11 million (Viscusi, 1986).

6.3.3 Specific Dollar Values Used in This Study

The range of reported estimates for the value of a statistical life is quite large, from around $1 million to over $15 million (Viscusi, 1993). Most estimates that are both robust and appropriate for valuing environmental risks cluster between $3 million and $7 million. For the purpose of assessing the value to society of reducing risk from air pollution, we want a value that is based on risk of a similar scale and is derived from the preferences of a population not unlike the population at risk from pollution exposure. The value currently used by the EPA is $5.9 million (in 1997 dollars). This value meets the criteria noted above and best represents the results of 26 studies, including those described in the section above (U.S. Environmental Protection Agency, 1997). Because the Houston population is not substantially different from the national population, this is also a reasonable value to use for this regional assessment.

6.4 SELECTING SPECIFIC VALUES FOR MORBIDITY

Reductions in acute or chronic illness or symptoms are important components of the welfare gain resulting from reduced risks to health. Morbidity effects are departures from "a state of physical or mental well-being, resulting from disease or injury, of which the affected individual is aware" (Peterson, 1975). Specific health conditions and symptoms such as eye irritation, cough, exacerbation of asthma, chronic bronchitis, and hospitalization for respiratory and cardiac conditions are reported by many health studies as evidence of ozone or fine particle-related morbidity effects.

Overall, morbidity values in the valuation literature range from as little as $5.80, to avoid a case of mild cough (Loehman et al., 1979), to over $600,000 for cases of moderately severe adult chronic bronchitis (Viscusi et al., 1991). In between, a case of hospitalization for respiratory or cardiac conditions has been valued in the thousands, and other effects in the tens or hundreds of dollars. Basically, costs tend to vary with the extensiveness of medical treatment and the degree to which an effect impairs normal activities.

This section reviews the COI and WTP studies that underpin the values used for specific morbidity effects. Unlike the extensive mortality literature, considerably less empirical work has been done to estimate morbidity-related values. Because morbidity incorporates a wide range of health conditions of differing severity and duration, a wide range of value estimates for different types of morbidity exists. It is therefore especially important to consider whether the values used correspond to effects of appropriate severity.

Earlier it was stated that defensible estimates of the value of reducing various air pollution-related health effects are a key component of estimating the benefits of attaining the National Ambient Air Quality Standards (NAAQS). That the nature of morbidity effects might make this undertaking difficult was also noted. A number of COI, and WTP morbidity-related studies are available as the basis for symptom valuation. Despite limitations,[4] consistent results emerge from this array of studies. Loehman et al. (1979) and Tolley et al. (1986) provide estimates for effects that are directly comparable to many of the symptoms included in this study, while Dickie and Gerking (1991) conducted a CV study that generated plausible lower bound WTP values.

Dollar values are selected for five individual symptoms: cough, headache, eye irritation, throat irritation, and chest discomfort, along with estimates for minor restricted activity days (MRADs) and restricted activity days (RADs). Chronic bronchitis, emergency room visits, and hospitalization for respiratory and cardiac conditions are also valued. All values are adjusted to 1997 dollars.

Although the cost of illness method is commonly used, the contingent valuation method of eliciting willingness to pay is generally closer to the ideal for morbidity valuation. Since most morbidity effects are well within the general population's range of experience, the likelihood of hypothetical inaccuracy in survey instruments is reduced.

[4] See Hall et al., 1989 and 1992 for a discussion of these limitations.

6.4.1 Specific Morbidity Values From Cost of Illness Studies

The cost of illness literature addresses two general categories of costs, direct and indirect. Direct costs include all medical and illness-related expenditures made by patients, insurance companies, and government agencies, and typically include: hospital care (inpatient and outpatient), services of physicians and other health professionals, and medication and related equipment. Indirect costs are calculated as some form of the wage rate multiplied by the time lost from work.

The COI-based estimates of most significance in benefit assessment are often the indirect cost represented by wages. This is because certain health outcomes, such as RADs, result in work loss, or equivalent restrictions on activity. Wages represent the payment necessary to compensate workers for giving up non-work activities, and the minimum value of a day's productivity. Sick pay prevents a direct loss of pay for many workers, but the value of foregone work output is still an important loss to the economy. We note that using foregone wages probably produces a conservative estimate of the value of a RAD, since wage rates do not tell us what compensation would be necessary to persuade an individual to be ill; they only reflect the value of one time given to one workday, not discomfort or pain.

Restricted activity days were valued by using average compensation levels specific to the Houston area. The U.S. Department of Labor (1996) reports detailed pay levels by occupation for the Houston-Galveston-Brazoria Consolidated Metropolitan Area. With this information, an average wage for the region was constructed in the form of a weighted average, by using the number of workers in each job classification, and the average wage obtained for the classification. The mean weekly wage calculation, in 1996 dollars, is $657.11, implying a daily wage of $131.42. Since the Labor Department did not include benefits in their occupational survey, this figure is a conservative estimate of the total compensation received by workers in the greater Houston area. In a further effort to remain conservative in our estimate, and because detailed medical data representative of Houston specifically were not readily available, we elected not to include any direct medical expenditures associated with RADs in our calculations. Direct medical expenditures on sick days in previous studies have typically comprised a small percentage of the total COI figure– 5 percent or less; this omission would, therefore, not distort our estimates in any appreciable way.

To generate RAD values, two adjustments are made to the reported average wage. First, we take into account that not all days of restricted activity occur during the work week, and assume that only 5/7 of all restricted activity days lead to complete work loss. Following previous valuation work (Hall et al., 1989, 1992; Rowe et al., 1986), other days of restricted activity are conservatively valued at one fourth of the average wage rate, giving an adjusted RAD value of $103.26. Second, we recognize that even this adjusted RAD value, if applied uniformly across the whole affected population, could overstate the direct cost associated with this health endpoint. If a retired person, or a student not currently working, suffers through a day of restricted activity, society obviously does not lose the output equivalent of a full-time working adult. To compensate for this factor, we construct RAD values as follows. For

working adults, the opportunity cost of time spent ill is valued using foregone daily wages; for non-working adults classified as “homemakers,” RADs are valued at 75 percent of the average daily wage; and for everyone else, a RAD is valued with our estimate for MRADs of $47. This provides a more conservative valuation of the change in occurrence of RADs, which can then be used to generate final dollar figures. Our final dollar values for restricted activity days, then, are: $87.79 for adults aged 20-64; and $53.75 for adults aged 64 and older. It should be noted that when children experience RADs, or other effects that require care, adult time at work is likely to be lost. The benefit of avoiding that loss is not estimated here, in part because RADs are not quantified for the population under age 20.

Finally, hospitalization and emergency room visits are valued on the basis of cost of treatment and loss of time. The COI for one respiratory illness admission, one chronic obstructive pulmonary disease (COPD) admission, or one pneumonia admission is $8,800, $11,700, and $11,400, respectively. The COI for a congestive heart failure admission is $12,000 rising to $14,800 for one ischemic heart disease admission (U.S. Environmental Protection Agency, 1997 adjusted to 1997 dollars). An emergency room visit is valued at $560 (Rowe et al., 1986 adjusted to 1997 dollars).

6.4.2 Specific Morbidity Values From Contingent Valuation Measures

The contingent valuation method uses surveys to place dollar values on nonmarket goods. This approach has been used to estimate values related to health as well as many other environmental amenities, including the cost of the Valdez oil spill. The contingent bidding method is the most widely used. To value morbidity, respondents are given information about the levels of morbidity, or factors that may affect morbidity. They are also presented with a hypothetical market that describes how payments might be made to reduce morbidity through, for example, taxes. They are then asked their maximum bid for a specific change in morbidity, which reveals their WTP. Respondents may alternatively be asked to estimate the minimum compensation that they would be willing to accept in order to agree to a specific change in morbidity.

Median estimates of the value of avoiding one day of mild cough and minor throat irritation from the Loehman et al. (1979) study are $5.80 and $9.75, respectively. Since these values are derived from the "minor" (as opposed to the "severe") symptom valuations given by that study's survey respondents, we concluded that they provide a reasonable range of value for the severity of these symptoms likely to be associated with ozone exposure. Similarly, Loehman et al.'s median value for avoiding one day of minor head congestion ($9.75) represents a reasonable value for mild headache.

Loehman et al.'s minor eye irritation WTP figure and Rowe et al.'s (1986) eye irritation value were averaged to produce a mid-range value of $8.45 for one less day of eye irritation. Two different symptom values from the Loehman et al. study were used to value the mid-range of chest discomfort. The WTP for the symptoms "cough" and "short of breath," which captures a reasonable range of the health effects generally construed as "chest discomfort" were averaged, resulting in a WTP figure of $9.10 for chest discomfort.

In asthmatic children, an effect referred to as "chest symptoms" has been reported in more recent health studies (e.g., Thurston et al., 1997). This effect includes some combination of cough, phlegm, and wheeze. We value this using the EPA estimate of $15 for lower respiratory symptoms (U.S. Environmental Protection Agency, 1997).

Willingness to pay to avoid a minor restricted activity day is logically higher than the WTP to avoid a single symptom, unless that symptom is quite severe. Tolley et al.'s median value for a three-symptom combination supports a WTP estimate of $47 (U.S. Environmental Protection Agency, 1997).

This MRAD value also falls within the range of WTP values developed by Farber and Rambaldi (1993), who used the CV method to determine WTP for air quality improvements in the Baton Rouge, Louisiana area.

The most highly valued morbidity effect is chronic bronchitis. Two CV studies (Krupnick and Cropper, 1989; Viscusi, 1991) have estimated the WTP to avoid one case of this illness. Those studies assessed a condition representing a more severe case of the disease. Consequently, their estimates have been adjusted by EPA to reflect the more moderate nature of the cases associated in the health literature with fine particle exposure. This results in a value of $319,000 per case (U.S. Environmental Protection Agency, 1997).

6.4.3 Summary of Values Used in This Study

All unit values used to estimate the economic benefits of reduced adverse health effects are summarized in **Table 6-1**. As discussed above, the value of avoiding an effect varies with the severity of the effect. More minor transient effects such as eye irritation carry much less value than severe consequences such as chronic bronchitis or premature mortality. All values in Table 6-1 have been adjusted to 1997 dollars. Measures based on willingness to pay were adjusted with the consumer price index (CPI-U), and hospitalization costs were adjusted with the medical component of the CPI-U[5].

6.5 ISSUES IN SELECTING SPECIFIC VALUES

It is important that the values for each effect quantified here reflect as closely as possible the value that society places on reducing the risk of ill health or death related to ambient ozone and fine particles. Because we have little direct evidence of the value individuals place on their own safety, and because we often extrapolate from the values evidenced in one group to other groups (workers to the elderly, or children for example), it is also important to be aware of the primary sources of upward and downward bias that might be introduced by use of any particular value. Some issues specific to a particular measure are discussed above. Here, we address some broader issues.

[5] The CPI-U represents changes in price levels in urban areas.

6.5.1 Groups Most At Risk

There are certain groups of people who are more likely to suffer adverse health effects than others following exposure to air pollution. Children are more vulnerable because of their physical immaturity, tendency to be more physically active, and time spent out of doors

An area of great concern to policy makers is risk to children. Traditionally, most of the emphasis in developing data for setting air quality standards has been on adults. More recently, however, awareness has grown that children cannot be considered "scaled-down" adults, and that more sophisticated approaches are necessary in order to accurately account for the effects of air pollution on them. While children inhale less mass of pollutant for a given exposure level than adults do, most physiological effects are dependent upon dose received. On this basis, the effective dose to a child's lung, in terms of pollutant received as a function of body weight, is usually greater than that for adults under similar exposure conditions. Moreover, lungs grow until the late teens or early twenties. If air pollution influences the growth process, resulting in smaller lungs, vitality is at risk. Children are more impacted by a given level of pollution because of their physiology and tendency to be active and out of doors.

At the same time that they are more vulnerable, there is evidence that both families and society place more weight on avoiding an adverse health effect in a child than in an adult. Consumers with young children report a WTP for safer consumer products that is 2 to 4 times higher than the WTP of households without children (Viscusi et al., 1987). In a later study (Viscusi et al., 1988) 75 percent of respondents indicated willingness to pay for child-risk reductions outside their own households.

Children and members of ethnic minorities might also be more at risk because they live in sections of urban areas with worse than average air quality. A number of studies suggest that exposure to riskier levels of pollution is greater for lower income groups, ethnic groups, and children (Brajer and Hall, 1992; Korc, 1996).

Poorer groups also tend to be more exposed because housing costs correlate to some degree with pollution. To the extent that minorities are more likely to be poor, they will also be differentially exposed to higher levels of pollution.

The health effects literature offers quantitative evidence that the individuals at highest risk of premature mortality are 65 or older. The concentration of premature deaths in this older age group has important implications for calculating health effects, and for monetary valuation, since the value of changes in mortality risk might be different for the older population. Extrapolating findings from one age group to another introduces further uncertainty into an already challenging process. Finally, it is useful to draw a distinction between persons who are merely "at risk" from exposure to air pollution and persons who are extremely sensitive to air pollution effects. Sensitive persons, who perhaps already have compromised heart or lung function, will react more dramatically to exposure than would individuals with no particular sensitivity.

6.5.2 Age and the Value of Life

Recently, consideration of the years of life lost, and the quality of life, as well as the number of premature deaths, has become a consideration in benefit estimation. Is the value of a statistical life for those aged 65 and older significantly less than for younger people? There are two reasons why this might be the case. First, fewer years of life are lost if an older person dies than if a child or younger adult dies. Second, the quality of life of the elderly may be poorer than the average person's quality of life. Therefore, less time is lost and the quality of that time might also be assumed to be compromised, when the risk is greatest for the elderly. While one study (Jones-Lee et al., 1985) suggests that individuals aged 65 value their lives at about 90 percent of the value forty year-olds place on themselves, other studies indicate that society places significantly greater weight on saving those who are younger (Cropper et al., 1994), with the greatest weight being given to the number of years saved per person, rather than the aggregate of years over the entire group at risk. That suggests that risks that could reduce life expectancy significantly for smaller groups are considered to be more serious than those that could reduce life expectancy by fewer years per person in a larger group, even when they reduce total life years by the same amount.

Confounding this idea is recent research (Tsevat et al., 1998) suggesting that even very old people - 80 and older - with compromised quality of life place significantly greater weight on even a limited amount of additional life than has been assumed. There are a number of reasons for this, with a main one being that, given little remaining time, the marginal benefit of what remains is very high.

Further complicating this issue, WTP for reduced risk of death is sensitive to income. For every percent increase in income, WTP for safety increases about one percent (Viscusi, 1992). Because income tends to rise with age, this implies that older individuals will be willing to pay more to reduce an equivalent risk than someone who is younger with a lower income (or less wealth). The Atkinson and Halvorsen automobile safety study, which estimated fairly high dollar amounts, illustrates this point, since the average age of their sample is greater than the age of lower income blue collar workers represented in wage-risk studies.

One technique to account for years lost in the population is the life years-lost (LYL) approach. The value of a life year is estimated at $360,000 (U.S. Environmental Protection Agency, 1997). In Section 6.7.6 below we present an alternative value of premature mortality based on this approach.

In short, the value of a statistical life for those 65 and older is almost certainly not the same as the VSL for a child or a 30 year-old, but we presently have no sound or consistent theoretical basis for adjusting that value.

6.5.3 Cost of Illness Compared to Willingness to Pay

Most economists support the use of WTP or WTA measures as alternatives or adjuncts to COI because they more completely account for loss of quality of life. A number of

valuation studies indicate that only about one-half of an individual's WTP is typically captured by the COI approach (Chestnut et al., 1988; Rowe and Neithercut, 1987). For example, Dickie and Gerking (1991) estimated that the ratio WTP to COI for reduction in ozone-related symptoms ranges from 2 to 4 for normal and respiratory-impaired individuals in California.

Other studies provide evidence that the difference between COI and WTP is much wider for more serious health endpoints, such as hospital admissions or development of chronic disease. For example, commonly accepted WTP values to *avoid* chronic bronchitis are 10 to 20 times higher than the cost of *treating* the illness. The WTP of parents of children with elevated lead levels is 2-20 times the cost of chelation therapy (Agee and Crocker, 1996). Clearly, COI measures represent an incomplete picture of the losses caused by deteriorating air quality; they are useful, at the very least, in providing lower-bound value estimates.

6.5.4 Other Valuation Issues

There will always be some uncertainty regarding the closeness of value estimates, from any method, to the "real" value of avoiding some health effects. The issues discussed below reflect other ways in which real and reported values might differ. Conclusions about the net effect on final estimates of divergences between "real" and estimated values cannot be made. This is because there is no established basis to make numerical adjustments. We know, for example, that there are important health endpoints (such as loss of lung function) that are not quantified. We also know that the value of reducing risk to life likely varies with age, but the values commonly used are based on a young to middle-aged cohort. We do not know how these factors jointly impact the degree to which aggregate estimates of value diverge from "real" values.

Unquantified effects

The adverse health effects whose frequency in association with air pollution can be quantified, and whose avoidance can also be valued in dollars, is a limited subset of the total set of adverse effects that have been observed or measured in epidemiological, human exposure, or animal studies. **Tables 6-2 and 6-3** list examples of unquantifiable and quantifiable air pollution health effects. Limitations of both health science and economics contribute to our inability to quantify these effects. Reduced lung function, for example, which has been linked to fine particles and ozone in a number of health studies, cannot be easily valued in economic terms. Moreover, we simply have no way to calculate the relative importance of what is measurable and what is not, so it is impossible to determine how large the unquantified effects may be in economic terms. In addition, some of the unquantified effects may overlap some effects that are quantified. For example, changed pulmonary function accompanies chronic bronchitis. Nonetheless, it is important to recognize the number and nature of such unquantified effects to provide perspective when interpreting the meaning and comprehensiveness of estimates of the economic value of improved air quality.

Currently, unquantified ozone-related effects include: lower respiratory symptoms, immunological changes, chronic respiratory damage and disease, inflammation of the lung, increased airway responsiveness, and changes in pulmonary function. Fine particle-related effects that cannot be quantified include: initiation of chronic asthma, chronic respiratory disease other than bronchitis, cancer, altered host defense mechanisms (which may be related to vulnerability to disease or injury), morphological changes, and changes in pulmonary function.

The effect of altruism

In addition to the downward bias introduced by omitted effects, we make no adjustment for altruism. A number of studies indicate that individuals are willing to pay more than the direct value to themselves to reduce risk to others. Jones-Lee (1992) reports that in a "caring society" the VSL of others is 110-140 percent of the VSL based on pure self-interest. As Viscusi et al. (1988) point out, however, these percentages cannot be treated as universal constants and therefore cannot be extrapolated to values for effects and circumstances not reflected in the studies that find such percentage differences between self-interested and altruistic values.

Given that both wage-risk and CV studies measure only the individual's WTP to reduce the risk of his own death (and not his value to family, friends and others, or the value he places on reducing risk to others) and the evidence that the value of avoiding an environmentally-related loss of life is greater than that of avoiding accidental death (Beggs, 1984), wage-risk values may be conservative estimates. Jones-Lee et al. (1985) found that the amount people were willing to pay to reduce risks to anonymous others was about one-third of what they were willing to pay to reduce similar risks to themselves. This result reflected, in part, what Viscusi (1986) called "the altruistic concerns of society at large." If the value of anonymous others was taken as a floor for the value those close to an individual who is at risk place on reducing risk to that individual, then it can be inferred that wage-risk based WTP studies may be at least one-third too low as measures of the full social welfare gained by reducing loss of life. WTP, therefore would capture no more than 75 percent of the value of a statistical life.

Quality adjustments for symptom values

Quality of life issues were raised above in the discussion of whether lives of the elderly should be valued less than lives of younger people because we presume that quality of life declines with aging. The question of quality of life also arises in the context of how to properly value a day when functioning is reduced to varying degrees by pollution-related symptoms. The values we use for eye irritation, headache, and respiratory symptoms are all under ten dollars a day. Recent work that pooled data across five WTP studies of symptoms (including the studies used here to establish values), and also used a quality of well-being (QWB) index to rate symptoms for their impact on mobility and physical and social activity, yields significantly higher values (Johnson et al., 1997). For example, these authors report a central value for a day of mild cough as $27, while the value we use is $5.80 for the same

symptom. The higher value results directly from indexing WTP for predicted reductions in the ability to function fully as a result of the symptom. Until these results have been repeated, or selected through the EPA's peer-review process for use in regulatory analyses, we have chosen not to use them.

Other sources of uncertainty

An important issue is the existence of health effects thresholds, that is, pollution levels below which there are no further beneficial health benefits to be realized from increased controls. The fact that most empirical studies that link pollution to adverse health effects come from epidemiological studies, where researchers cannot control exposure, makes it particularly difficult to determine whether or not there are health effect threshold levels. A consistent association between particulate matter and health effects has been found by recent health studies, even at pollution levels well below most air quality standards. The existence and level of thresholds remains an important source of uncertainty in health estimation, for if no threshold is assumed, but one exists, benefits will be overestimated. Conversely, if a threshold is imposed where one does not exist, the predicted health benefits resulting from an improvement in air quality could be seriously underestimated.

Benefit assessments depend heavily on epidemiological studies, many of which show statistical associations between pollutants and a number of common health effects, including increased hospital admissions and elevated mortality risk. What makes the use of these associations somewhat problematic is that significant overlap may exist in the estimated benefits attributed separately to reductions in particulate concentrations and in ozone concentrations. An additional source of overlap in benefit estimation involves the valuing of reductions in the common precursor emissions for fine particles and ozone. Reductions in nitrogen oxides, for example, can reduce both fine particles and ozone. Any calculation of health benefits per ton reduced, therefore, should account for reductions in effects related to both pollutants. In future valuation efforts, this overlap issue may carry further importance, as statistical associations are now being increasingly found between ozone and higher mortality risk–ozone-related mortality will increasingly be included in benefit assessments. The health studies used in this analysis were chosen in part for their efforts to address this issue and to control for multiple pollutants.

6.6 RESULTS FOR MORBIDITY

A number of upper- and lower-respiratory effects are associated with exposure to ozone. These include cough, chest congestion, irritated throat, phlegm, and wheezing. The reduced frequency of the cough is reported here for adults (age 20 and older), while chest discomfort and irritated throat are reported for the general population, along with eye irritation and headache. A symptom called "chest symptoms" is reported for asthmatic children aged 7 to 13. The results reported in this section are based on the air quality improvements associated with achieving the 1-hr NAAQS in 2007, relative to the 2007 baseline projection (Scenario 1). The economic value of reducing these adverse effects is also estimated.

The adverse health effects that have been demonstrated to result from exposure to elevated levels of fine particles are generally more severe that those associated with ozone. Their occurrence is also less frequent than most ozone-related effects, but the value per effect is high because of the extent to which PM-related effects impede daily function. Estimates of how much the frequency of each effect is expected to fall with attainment of the NAAQS, and the value of those reductions, are reported below for each effect that has been quantified. A range of values is also reported for each effect. The range is based on two different assumptions about the relative effects of $PM_{2.5}$ and PM_{10}. Many of the underlying epidemiological studies used to quantify effects report PM_{10}-related concentration-effect relationships. One of the reasons the EPA promulgated a $PM_{2.5}$ NAAQS in 1997 is increasing evidence that this fraction of fine particles is more hazardous than the total PM_{10} fraction. Applying the mathematical association between PM_{10} and adverse effects to changes in $PM_{2.5}$ concentrations is therefore likely to underestimate the actual improvements in health as $PM_{2.5}$ levels decline. However, adjusting those mathematical associations to account for the average fraction of PM_{10} that is $PM_{2.5}$ in the Houston area (0.607) would likely overestimate the effect of changing exposure to $PM_{2.5}$ by ascribing the total effect of fine particles to just this fraction. The health literature currently provides no clear basis for determining how great the upward and downward biases are with the two approaches, so we present results for both cases when the underlying health literature is based on PM_{10}. When $PM_{2.5}$ was the fine particle measure no adjustment is made. These should be taken as upper and lower bounds, with recognition that we do not know with certainty what part of the spectrum better represents reality.

6.6.1 Chest Symptoms in Asthmatic Children

Asthmatic children are more sensitive to some pollutants than children, on average, and a significant minority – seven percent – of all children are asthmatic (American Lung Association, 1998). Because of these factors some recent research has focused on the association between ozone and a set of symptoms in asthmatic children. Using the results from Thurston et al. (1997) who carefully followed groups of asthmatic 7 to 13 year olds during intervals of three successive summers, we estimate reductions in "chest symptoms" for this sub-population. This effect includes cough, phlegm, and wheeze.

For the year 2007, we estimate that chest symptoms will decline by just over 1.1 million person-days in the population of about 22,000 asthmatic children aged 7 to 13. This represents a per capita decline of over 52 days a year (from a baseline prediction of nearly 61 days a year). At a value of $15 for each day of reduced symptoms, the economic benefit of reducing this effect is $16.7 million annually.

6.6.2 Cough

Reduced experience of cough is reported for adults only, because the health literature does not document this symptom sufficiently in healthy children. For the population aged 20 and older 345,800 fewer person-days of cough are estimated to occur. At a value of $5.80 for

each day of avoided symptoms, this represents an economic benefit of over $2 million annually.

6.6.3 Eye Irritation, Chest Congestion, Irritated Throat, and Headache

These symptoms are expected to occur across the entire age range of the population. We estimate 344,000 fewer person-days of chest discomfort, over 580,000 fewer person-days of headache, a reduction of more than 957,000 person-days of irritated throat, and 4,350,000 fewer person-days of eye irritation annually. Given the economic values of $9.10, $9.75, $9.75, and $8.45, respectively, the economic value of these reductions in ozone-related symptoms in the general population, and cough in adults, is summarized in **Table 6-4**.

6.6.4 Ozone-Related Effects For Other Emission Scenarios

In addition to the rollback to the 1-hr NAAQS attainment scenario results reported above, six alternative emission reductions cases were evaluated. Five of these assume varying percentages of NO_x emissions reductions in conjunction with a 15 percent reduction in VOC emissions. The last case is for implementation of a clean diesel fuel program.

As **Table 6-5** shows, the clean diesel fuel case is very similar to the effect of 25 percent NO_x reductions, showing somewhat larger reductions in ozone-related symptoms. The alternative cases show increasing benefits of NO_x emission reductions up to the 85 percent NO_x reduction case, which is essentially equivalent to the 1-hr NAAQS rollback case.

6.6.5 Approach to PM-Morbidity Quantification

To quantify the expected changes in health effects associated with reduced exposure to fine particulate matter, we have used the basic exponential concentration-response functions developed in the EPA Retrospective Analysis (U.S. Environmental Protection Agency, 1997), which evaluates the benefits and costs of emissions controls imposed by the Clean Air Act. Specifically, the functional form used is as follows:

$$\Delta C = C\left(e^{\beta \Delta PM} - 1\right) \qquad \textbf{(6-1)}$$

where:

C = the number of cases (of a particular health endpoint)
ΔPM = the change in ambient PM concentrations
β = an exponential "slope" factor derived from the health literature pertaining to that specific endpoint.

In most of the recent health literature, "relative risk" factors are reported which relate change in pollution levels to the increased odds of developing various health effects. These risk

factors are related to the β in the EPA concentration-response functions in the following manner:

$$\beta = \frac{(1 + \text{Increased Odds})}{\text{Change in Pollution}} \qquad \textbf{(6-2)}$$

Finally, as noted in the sections below, in some cases linear concentration response functions were also used.

6.6.6 Minor Restricted Activity Days

Minor restricted activity days are days when fine particle-related symptoms reduce normal activities, but do not prevent going to work or attending school. The combination of symptoms that induces an MRAD is more restrictive than any individual symptom.

A 1989 study by Ostro and Rothschild uses six years of Health Interview Survey (HIS) data, a large cross-sectional database collected by the National Center for Health Statistics, to determine the health consequences of particulate matter in an urban environment. They find an association between fine particles and minor restrictions in activity that can be used to derive an exponential exposure-response function.

From the data included in Ostro and Rothschild's analysis, the best estimate of b = .0082, and the average number of annual MRADs is 7.8 per person. The specific linear equation used to estimate reductions in MRADs thus becomes:

$$\Delta \text{MRADs (per person)} = 0.000175\ \Delta PM_{2.5} \qquad \textbf{(6-3)}$$

The results from application of this exposure-response function to the Houston population are shown in **Table 6-6**.

6.6.7 Restricted Activity Days

Restricted activity days (RADs) are estimated for two age groups, those aged 20 to 64 and those aged 65 and older. This division is not based on any difference in the concentration-response functions used to estimate the number of RADs, but on differences in the opportunity cost of time between the age groups. The value of reducing this effect is based on wages (as described in Section 6.4.1 above. For the younger group, this is $87.79 per day and for the older one, it is $53.75 per day.

In Ostro (1987), restricted activity days occurring over a two-week recall period were related to fine particulate matter determined from visual range data for 49 different metropolitan areas in the United States. The study found a significant association between particulate matter and RADs for each of the six years of the study (1976-1981). The functional form derived from the analysis is:

$$PM_{2.5} \text{ related RADs (per person)} = b\left(RADs_{\text{annual average}}\right)\Delta PM_{2.5} \qquad \textbf{(6-4)}$$

From the six years of data included in Ostro's analysis, the best estimate of b = .0048; and according to the EPA (U.S. Environmental Protection Agency, 1997), the average number of annual RADs is 4 per person. The specific linear equation used to estimate reductions in RADs thus becomes:

$$\Delta \text{RADs (per person)} = 0.00005267\ \Delta PM_{2.5} \qquad \textbf{(6-5)}$$

Results for this effect are summarized in **Table 6-7**. They suggest that about five percent of adults in the Houston area lose at least one day of normal activity a year to elevated $PM_{2.5}$ levels. These estimates do not include RADs in children because they are not normally included in the working population, so there is nothing comparable to wage rates with which to estimate the value of a child's time. Given that a child whose activities are substantially restricted is likely to require that an adult take time from work to care for them, the totals here do not represent the total value of reducing RADs.

6.6.8 Chronic Bronchitis

This effect represents the initiation of new cases of chronic bronchitis on an annual basis, in association with $PM_{2.5}$ exposure. The value of reducing this effect is not the annual value of an on-going case, which is captured in the value assigned to each case at the outset of that case, but the value of avoiding the effect over the future life of an affected individual. Because it is a severe effect that reduces the range of normal activities over a period of years and also represents on-going discomfort, people place substantial value on avoiding chronic bronchitis. Per case, that value is $319,000. This figure is based on contingent valuation studies that estimated WTP for a more severe case and adjustment to reflect the relatively more mild cases associated with fine particles in the epidemiology literature.

A study by Abbey et al. (1993) presents associations between long-term cumulative ambient concentrations of total suspended particulate matter and new cases of chronic bronchitis in southern California. Transforming the relative risk factor of 1.36 (for a new case of bronchitis at a 10-year exposure of 60 $\mu g/m^3$) yields a one-year exponential β value of 0.00915 for PM_{10}. Adjusting this PM_{10}-based measure to correspond to $PM_{2.5}$ produces a β value of 0.01507. Reductions in the number of chronic bronchitis cases associated with air quality improvements were calculated using this β value and a baseline incidence of 710 chronic bronchitis cases per 100,000 in the adult age groups.

In the Houston area, the total number of new cases annually that could be avoided with attainment of the $PM_{2.5}$ NAAQS ranges from 711 to 1,196. The breakdown by age group is shown in **Table 6-8**. Unlike some other $PM_{2.5}$-related effects, chronic bronchitis occurs most frequently in the age group from 20-64. This is not unexpected, because this group comprises about 87 percent of the total adult population (aged 20 and up), but it is significant in that

functioning is likely reduced over a significant number of years. Again, this is why a relatively large value is attached to avoiding a case of chronic bronchitis.

6.6.9 Hospital Admissions

A number of studies have associated respiratory and cardiac hospital admissions with exposure to fine particles. Admissions associated with all respiratory-related admissions for the total population are estimated here, along with cardiac-related admissions in the group over age 64. For these effects, the economic value of reducing a case is based on COI, or medical treatment and lost time. The value of avoiding pain is not captured in these values.

Hospitalization in the 65 and older age group

Multiple studies (Schwartz, 1994a, 1994b, 1994c, 1996) have found associations between airborne particles and respiratory hospital admissions for several cities in the United States. More specifically, admission counts for pneumonia and chronic obstructive pulmonary disease (COPD) and all respiratory admissions are associated with PM_{10} concentrations for persons 65 years of age and older. Each study also generates relative risk factors, which can be transformed into β values, using the same procedure previously described in the premature mortality section. These β values appear in **Table 6-9**. For our calculations, we chose the average β value for each specific health endpoint. These average values also appear in Table 6-9, along with baseline incidence estimates (U.S. Environmental Protection Agency, 1997) for each admissions category. Together, this information allowed us to apply exponential exposure-response functions for the following health outcomes: All respiratory hospital admissions, hospital admissions for pneumonia, and hospital admissions for COPD, for the group aged 64 and older.

For hospital admissions relating to congestive heart failure and ischemic heart disease, we use the study by Schwartz and Morris (1995), which examines the association between air pollution and cardiovascular hospital admissions for persons aged 65 and older in the Detroit, Michigan area. Here, relative risk factors of 1.032 and 1.018 for congestive heart failure and ischemic heart disease translate into β values of .00098 and .00056, respectively. Results are shown in **Table 6-10**.

Hospitalization in all age groups

Pope (1991) estimates statistical associations between respiratory hospital admissions and particulate (PM_{10}) pollution for all age groups. Instead of reporting relative risk factors, Pope (1991) reports regression coefficients that describe the relationship between pollution levels and health outcomes. The coefficients provide a linear exposure-response relationship that was used as the basis for our calculations of reductions in hospitalizations from improved air quality conditions.

Pope (1991) assesses the association between respiratory hospital admissions and PM_{10} pollution across Utah, Salt Lake, and Cache counties in Utah. By performing a multiple regression analysis, the study estimates a slope coefficient of 0.8047 between lagged mean PM_{10} and monthly hospital admissions. For our calculations, this coefficient was adjusted to reflect daily rather than monthly admissions and modified to associate the effect with $PM_{2.5}$ rather than PM_{10}. The adjusted model has 5.6×10^{-8} daily per person change in admissions per unit change in $PM_{2.5}$ concentration (in $\mu g/m^3$), which is applicable to the entire population. The overall results and results by age group for all-respiratory admissions are shown in **Table 6-11**.

6.6.10 Summary of Morbidity Results

The aggregated benefits of reduced morbidity are shown in **Table 6-12.** It should again be noted that the dollar values for effects related to fine particle exposure are greater than for effects related to ozone. Yet, the number of symptoms related to ozone exposure is much greater than for PM-related symptoms. Reducing ozone exposure is predicted to substantially reduce respiratory symptoms in asthmatic children.

6.7 MORTALITY

The most severe consequence of exposure to unhealthful levels of fine particles is premature mortality – dying before the end of an individual's life expectancy has been reached. The value that individuals attach to reducing the risk of death in a variety of circumstances implies a value per deferred death of $5.9 million. (Please see Section 6.3.3 above for a discussion of the basis for this value.) Given this very large amount per life, mortality tends to overwhelm the economic benefit of reducing all other effects. This is true in Houston as it has been in every other region where economic benefits of better air quality have been analyzed.

In the EPA Retrospective Study (U.S. Environmental Protection Agency, 1997) the incidence of PM-related mortality used for estimating the benefits of the Clean Air Act is based primarily on a recent, long-term prospective (cohort) exposure study by Pope et al. (1995). This work is considered to be an important contributor to the study of particulate-related mortality, both because of its prospective design and the large number and diversity of locations included. In addition, it is supported by several earlier cross-sectional studies of annual mortality. To estimate fine particle-related premature mortality in adults, the concentration-response function developed in the Pope et al. study was determined to best represent risk to the population aged 30 and older.

In the estimations presented here, we also rely on Pope et al. (1995) to quantify changes in the number of deaths in the population aged 30 and older. Pope et al. is based on a prospective cohort-based method. Over 500,000 individuals were followed for a seven-year period. This approach has the advantage of allowing investigators to control for individual variables such as smoking and age (as well as generic variables such as temperature). The association is between long-term exposure rather than daily exposure. This is important

because it indicates that fine particle pollution increases mortality as a result of continuing exposure, rather than just a short-term insult, and, on average, years of life, rather than days, are lost for individuals who die prematurely. This research compares populations over multiple (151) locations, which reduces the influence of location-specific factors. This long-term study finds a higher mortality effect than the short-term population-based studies because the short-term studies do not capture deaths that occur outside high particle episodes, but that are associated with long-term particle exposure. (A detailed discussion concerning the merits of the Pope et al. study is provided in U.S. Environmental Protection Agency, 1997, page D-15).

It is important to note that the Pope et al. study (1995) did not include data on those under age 30. Therefore, we report this effect only for the group 30 and older. We do this because it is generally accepted that the risk is greatest for those 65 and older, and if we were to extrapolate the relationship between exposure and death for that group (or the 30-64 group) to younger adults and children we would surely overestimate (probably by a significant degree) the effect in the younger population. While there are other studies of mortality risk in a broader population group, the methods in those studies are not comparable to those in Pope et al. (1995). Using those mathematical associations to estimate risk in the younger group would therefore produce results that could not be compared reasonably with those presented here for the older group.

The Pope et al. results are used to generate the exponential concentration-response function in the following manner. Pope et al. report a relative risk factor of 1.17 for all-cause mortality associated with a 24.5 $\mu g/m^3$ increment of fine particles. (This means that mortality was 17 percent higher in the areas where average $PM_{2.5}$ concentrations were 24.5 $\mu g/m^3$ higher). The β, or slope, factor to be used in the exponential function is thus calculated as follows:

$$1.17 = e^{\beta\,(24.5\ \mu g/m3)}$$
$$\beta = 0.006408 \qquad \textbf{(6-6)}$$

To facilitate our calculations, we combined these exponential concentration-response functions with Texas-specific, baseline non-accidental death rates. (These were defined, in a manner consistent with the EPA, as Total Deaths – [Accidents + Suicides + Homicides]). The following death rates were used:

Population segment	Death rate per 1,000
Adults, Ages 30-64	3.46
Adults, Ages 65 and older	50.79

The results for mortality are shown in **Table 6-13**. To place these numbers in perspective, we compare them to the annual risk of death in a motor vehicle accident and to the

death rate for each age group. The $PM_{2.5}$ risk for those aged 20 to 64 is 5.8 in 100,000, or about one third of the risk of dying in a motor vehicle. For those aged 65 and older, the risk from $PM_{2.5}$ is much greater, 86.4 in 100,000, or about 3.5 times the risk of dying on the road. The overall death rates for these age groups are 346 in 100,000 and 5,079 in 100,000, respectively. This means that while the risk of death in association with fine particle exposure is much greater for the older group, the percentage change in baseline death rates for the two groups is nearly the same, 0.0168 percent for those aged 30 to 64 and 0.0170 percent for those aged 65 and older.

Valuing premature mortality with the life years lost method

The greatest risk of premature death is in the population aged 65 and older. Because life expectancy in that group is lower than in the younger population that underlies the wage risk studies on which VSL is largely based, consideration of years lost is a factor in valuing this effect. Countering this is the fact that wealth tends to increase with age, so the WTP for the older population could be higher than that for the wage-risk population, all else being equal. To provide some perspective, we have estimated the value of avoiding premature death using the life years lost (LYL) method as well as the VSL method reported above. Following Deck and Post (1996) as used in the EPA Retrospective analysis (U.S. Environmental Protection Agency, 1997), we assume, on average, fourteen years are lost to individuals who die prematurely due to elevated levels of fine particles. Given a value of each LYL of $360,000 and 435 premature deaths, using this method yields an economic benefit from reduced premature mortality of $2.19 billion. This is 85 percent of the value estimated using the VSL method.

6.7.1 Fine-Particle Related Effects Under Ozone-Reducing Scenarios

As described in Section 2, there were several alternate emission inventory projections for 2007 developed for PM and PM-related species. The effects described above are for the "Scenario 1" projected inventory. In addition to Scenario 1, a number of alternative emission reduction combinations were assessed, including two variations on Scenario 1 (Scenarios 2 and 3), full implementation of clean diesel fuels, and an analysis of the 1998 ozone SIP provisions. The results for all emissions scenarios and for the three most severe health effects (premature mortality, RADs, and chronic bronchitis) are shown in **Table 6-14**. Results for other fine particle-related effects are proportionate. Scenarios 1, 2, and 3 produce similar results, with less than 10 percent variation between the largest and smallest changes in effects. The clean diesel fuel and ozone SIP scenarios have approximately 10 percent of the benefits of achieving compliance with the annual $PM_{2.5}$ NAAQS.

6.8 OVERALL BENEFITS

Overall benefit estimates are shown in Table 6-12. The total benefits expected to result from compliance with the annual $PM_{2.5}$ NAAQS and the 1-hr ozone NAAQS in the year 2007

are between $2.9 billion and $3.1 billion.[6] The adverse health effects of ozone are far more frequent than is the case for fine particles, ranging to over 4 million person-days each year of eye irritation, and totaling more than 7 million person-days each year; but they carry much less substantial economic value because they are less severe and sometimes more reversible than fine particle-related effects.

Several points are important in viewing these results. First, many well-studied and documented effects associated with urban air pollution are not quantified here. Physiological changes that make people more susceptible to illness are not represented. This omission may be especially important for children, whose lung capacity is still developing. The effects of hazardous air pollutants are not represented, nor is the initiation of chronic diseases other than bronchitis, such as asthma and emphysema. We presently have no basis to estimate how much of a downward bias is introduced by the unquantified effects. Second, the health science literature has developed primarily to support setting air quality standards for single pollutants. Consequently, the scientific basis for quantifying effects focuses on teasing out the impact of discrete compounds and not the overall impact of the complex mixture that people breathe. While results are reported for ozone and for fine particles, these pollutants in fact occur together, and in combination with other pollutants such as NO_2. Third, premature death and chronic bronchitis represent the effects of longer term pollutant exposure on health, while the other effects are acute and occur in relation with one-day elevated concentrations. Finally, emissions controls designed to reduce ozone will also reduce fine particle concentrations. Consequently, the benefits of ozone controls are greater than just the benefits of reducing ozone-related morbidity quantified in this study.

Epidemiological studies find statistical associations between fine particle levels and an array of health endpoints, including hospital admissions and restricted activity days (RADs), that may be associated with one another. Obviously, days spent in a hospital are also days of restricted activity, and should therefore be subtracted from RADs before aggregating monetary benefits. For the overall results of this study, this makes no difference in the aggregate benefits, because the number of hospitalizations is so small relative to the number of RADs. Because all ages are included in the "all respiratory" related hospitalizations, the separate estimates for those aged 65 and older for COPD and pneumonia are not represented in the aggregate benefits. Again, this makes no difference in the aggregate results.

6.8.1 Who Benefits

The health literature that supports quantification of the benefits of reducing the adverse health effects included in Table 6-12 was largely designed to discover whether one or more of the criteria pollutants is discretely associated with any effect(s), and if so at what concentration and for what time interval. Consequently, there is little or no basis to estimate the benefits of reducing exposure to the complex mix of urban pollution and only a limited basis to assess the benefits to subsets of the population. Conversely, some effects can only be quantified reliably

[6] The range of benefits represents uncertainty about the relative potency of $PM_{2.5}$ (compared to PM_{10}) in association with chronic bronchitis, hospitalizations, and MRADs.

for some age groups, because the underlying health studies did not consider all age groups, or because not all effects have been observed in all age groups.

Generally, children and the elderly appear to be more vulnerable. Adults 65 and older are at the greatest risk of premature mortality. Asthmatic children experience significant numbers of days of chest symptoms – attaining the 1-hour NAAQS for ozone would reduce this effect by an average of over 50 days a year for each asthmatic child. Also, exposure during childhood logically contributes to greater risk of pollution-related illness in adulthood, but the subtle physiological effects that precede overt illness or symptoms are not quantifiable in economic terms. Children's exposure is more important than what can be quantified indicates.

6.9 RELATED HEALTH BENEFIT STUDIES

This study is specific to the air quality and population characteristics of Houston. The objective is to determine the scale of benefits expected to result from reduced exposure to air pollutants, especially ozone and fine particle pollution, which are the object of federally mandated regulatory programs. A summary of other recent regional health benefit studies, of varying geographic scale and using different assessment methods, but a similar set of concentration-response functions and economic values is provided here so that the results of the Houston study can be considered in a larger context. A more comprehensive review of related work is provided in the American Lung Association report, *The Value of Clean Air* (1997).

6.9.1 REHEX-Based Studies

In 1988, Winer et al. developed an approach to human exposure modeling that accounts for variables such as age, distribution of the population relative to air quality monitors, time and activity profiles of the population, and how pollutant concentrations differ by location (especially indoors or outdoors) (Winer et al., 1989). The model that estimates exposure and dose is called the Regional Human Exposure model (REHEX). Since that time, REHEX has been refined in multiple applications to regions within California. The capability of REHEX to estimate exposure by location, and to overlay demographic characteristics on pollutant exposure, also makes it possible to consider differential risks to older populations and children, and to evaluate the distribution of effects by income and ethnicity.

REHEX was first developed to support assessment of the potential benefits of meeting the NAAQS in the South Coast Air Basin of California (the SoCAB). In the late 1980s, the regional population approached 13 million and air quality was (and remains) the worst in the United States. Accounting for the impact of PM_{10} on mortality and RADs, and ozone-related symptoms including eye irritation, cough, chest congestion, headache, and throat irritation along with MRADs, the study concluded that the economic benefits of meeting the ozone and PM_{10} NAAQS in effect at that time[7] (relative to levels of pollution experienced in the mid-

[7] This means the pre-1997 NAAQS for ozone and fine particles.

1980s) totaled more than $11 billion annually. Of the total, $3.3 billion was due to reduced ozone exposure and $7.8 billion resulted from meeting the NAAQS for PM_{10} (Hall et al. 1989, 1992). Ozone-related effects were far more frequent, but PM_{10}-related effects, while less common, carried much more substantial values per effect avoided. The overall results were dominated by the value of reducing premature mortality.

Subsequent REHEX-based studies

In 1992 Brajer and Hall published an analysis of how ozone and fine particle-related effects relate to age, income, and ethnicity in the South Coast Air Basin. The results show a positive correlation between lower- (and middle-) income groups and ozone exposure (and a negative association with the highest income group) with a similar, but stronger pattern for PM_{10}. Higher ozone and PM_{10} levels also were correlated with age – those under 18 were more likely to be exposed to higher levels within the basin. Regarding ethnicity, both Hispanics and blacks were more likely to be exposed to higher levels of both pollutants, with a stronger association again for PM_{10} relative to ozone. The ability of REHEX to represent mobility and microenvironments supported more detailed assessment of the correlation among demographic characteristics and pollution exposure.

The San Francisco Bay Area is also nonattainment for ozone and PM_{10}. In 1994, Hall, Brajer, and Kleinman undertook an assessment of the health benefits of attaining the standards in that basin. Again, this work was supported by the REHEX model (Lurmann and Korc, 1994), now known as REHEX II to reflect the increased scope and efficiency of the model. This study compared health effects resulting from exposure in the 1990-1992 time period to predicted levels of effects if the ozone and PM_{10} standards had been met in the same interval. The regional population was about five million. The Bay Area was much closer to attainment of the ozone than to the PM_{10} standard, so the frequency of effects from PM_{10} was proportionately greater than in the SoCAB studies. Again, children experienced a disproportionate number of non-fatal effects. The overall benefit of attaining the California AQS (more comparable to the new NAAQS) was almost $2.9 billion annually, with over 95 percent of the total benefits resulting from reduced PM_{10} exposure.

Following the original study of health benefits (Hall et al., 1989, 1992), the South Coast Air Quality Management District (South Coast Air Quality Management District, 1996) has continued to use and refine the REHEX approach to assess health benefits from alternative air quality programs. In an analysis done to support the 1997 Air Quality Management Plan the benefits of reducing concentrations at the highest monitor to the pre-1997 Federal standards, and setting the threshold at the California AQS, were nearly $5 billion annually. Again, while ozone-related effects were more common, the more severe effects of elevated fine particle levels dominated the aggregate benefits.

6.9.2 Other Regional Health Benefit Studies

Utah Valley

In 1986 and 1987 a steel mill in Utah Valley was shut down due to a labor dispute. The Federal 24-hour NAAQS for PM_{10} was violated on 12.6 days a year when the mill was operating whereas there were no violations while the mill was closed. Ransom and Pope (1995) estimated the difference in hospital admissions for bronchitis and asthma, pneumonia and other respiratory diseases, cardiovascular diseases, total days of hospitalization, and changes in mortality when the mill was operating and when it was not. They also compared the pattern of these effects during the same time period in the Cache Valley, where the PM_{10} NAAQS was never violated, demographic characteristics were similar to those in Utah Valley, and the mill did not influence air quality. The results of this study show a strong statistical association of PM_{10} with bronchitis and asthma, which rose by over 20 percent and 45 percent, respectively, as well as significant associations with other respiratory-related hospital admissions and mortality.

In the same study, Ransom and Pope estimated the dollar value of these health effects in association with elevated PM_{10} levels at between $40 million and $525 million (depending on whether a VSL of $2 million or $7 million was used) for the residents of Utah Valley. (The population was approximately 223,000.) The authors also concluded that annual health damage in the community exceeded $17,000 per job annually when the mill was operating.

Midwestern and southern states

Krupnick et al. (1995) evaluated the health benefits expected to result from reduced PM_{10} exposure in association with NO_x and SO_x reductions resulting from Title IV of the Clean Air Act (the federal acid deposition regulatory program). Baseline exposure was based on ambient concentrations in 1992 and estimated benefits were based on forecast air quality levels for the year 2000. The geographic scope included five states: Illinois, Indiana, Mississippi, North Carolina, and South Carolina.

Across the five states (with a combined projected population of about 32 million in 2000), 659 fewer deaths and 14.4 million fewer days of respiratory symptoms were predicted to result as PM_{10} levels fell along with acid deposition precursors. Aggregate dollar benefits equaled almost $3 billion. Per capita benefits in urban areas of the region fell in the range of $80-180 per year, with a most likely value around $120. The majority of this benefit was related to mortality. These benefits do not reflect the value of attaining the PM_{10} NAAQS, because reducing acid precursors alone does not achieve attainment.

Los Angeles County hazardous air pollutants

A recently released report to the House of Representatives Committee on Government Reform (U.S. House of Representatives, 1999) concludes that residents of Los Angeles county are exposed to levels of three hazardous air pollutants at concentrations well above the EPA benchmark for a one in one million risk of cancer over a lifetime. These pollutants include 1,3-butadiene, formaldehyde, and benzene. Annual average benzene exposure in the county is estimated at 6.3 $\mu g/m^3$ from 1995 to mid-1998. The EPA benchmark for this contaminant is 0.13-0.34 $\mu g/m^3$. The study extrapolates exposure to the county population by averaging data from three monitoring sites over the entire time interval and uses the benchmark value to extrapolate the annual cancer risk to residents, finding a risk of 49 cases of benzene-related cancers per million in the population. Average 1,3-butadiene exposure is reported as 0.93 $\mu g/m^3$ annually, and formaldehyde at 5.6 $\mu g/m^3$. Given benchmark concentrations of 0.0036 µg/m3 and 0.077 $\mu g/m^3$, respectively, the report estimates related risks as 258 per million for 1,3-butadiene and 72 per million for formaldehyde. Benchmark levels, however, relate to an upper bound, not a mean effects level. The actual expected number of cases would be much lower. This report does not provide any economic estimate of the value of avoiding the estimated additional number of cancer cases.

6.10 FUTURE RESEARCH

There are significant gaps in the knowledge and information available to assess the benefits of alternative emissions reduction scenarios. Robust estimation of economic value depends first on air quality monitors that represent the exposure population reasonably well and on a good understanding of how reducing emissions would reduce exposure, while population and pollution sources are changing. Given good estimates of how exposure would change as emissions decline, we next need a health effects base that is congruent with our exposure data – assesses effects relative to equivalent chemical composition of the atmosphere, averaging times, and age distribution of the population. Finally, for each health endpoint that can be related to pollution exposure in a region we then also need an economic value that represents the dollar benefits of reducing the occurrence of that endpoint as exposure declines. Several suggestions for priority research needs follow.

- As described in Section 5, there are myriad adverse physiological responses to pollutant exposure. Many of these – structural changes in the lungs and airways, reduced lung function, and immune system responses, for example – are logically related to the occurrence of disease in some portion of the population. We presently have no economic knowledge about what the benefits would be of preventing these responses, however. Therefore, one important area of further research is interdisciplinary work by health scientists and economists to better model the link between physiological change and adverse health outcomes and to design contingent valuation studies to then determine the value of avoiding the more subtle pre-clinical effects.

- The preponderance of mortality appears to be among the elderly. Given this emerging health science finding, it is important to more clearly determine whether and by how much

society values the lives of older people differently than lives in the age groups that current values are drawn from. This is also true for the lives of children, as there is preliminary research indicating higher risk of death in infants in polluted environments.

- Society places significant weight on protecting children, but we have almost no basis to determine how to value, for example, chest symptoms in children relative to the adults on whom values are now based. Given the demonstrated sensitivity of asthmatic children, and the higher doses of pollution experienced by children in general, relative to adults, this is a key area of investigation.

- Emissions reductions will often reduce multiple pollutants simultaneously, but we typically assess the benefits of reducing one pollutant at a time. More investigation is needed of the joint value of reducing a set of adverse effects, rather than single effects.

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Table 6-1. Unit economic values of health effects in 1997 dollars.

Effect	Dollar Value per Event
Value of a Statistical Life	$5,900,000
Value of Life Year Lost	$360,000
New Case of Chronic Bronchitis	$319,000
Hospitalization for Respiratory Conditions	$8,790
Hospitalization for COPD	$11,670
Hospitalization for Pneumonia	$11,380
Hospitalization for Congestive Heart Failure	$11,960
Hospitalization for Ischemic Heart Disease	$14,840
Lower Respiratory Symptoms	$15/day
Minor Restricted Activity Day	$47/day
Restricted Activity Day	$87.79/day aged 20-64 $53.75/day aged over 64
Cough	$5.80/day
Throat Irritation	$9.75/day
Eye Irritation	$8.45/day
Headache	$9.75/day
Chest Congestion	$9.10/day

Table 6-2. Known health effects and suspected health effects of air pollutants which are currently unquantifiable.

Known Air Pollution Health Effects	Suspected Air Pollution Health Effects
Changes in lung structure	Acute myeloid leukemia (HAPs)
Respiratory tract damage	Other cancers (HAPs)
Accelerated “aging” of lung	Cardiac arrhythmias (HAPs)
Heart rhythm and rate anomalies	Premature death (Ozone)
Respiratory illness in children	Emergency room visits (Ozone)
Exacerbation of existing lung disease	Lung cancer
Reduced pulmonary function	Prevalence of asthma
Increased respiratory symptoms	Altered lung defense mechanisms

Table 6-3. Types of known health effects of air pollutants that are currently quantifiable.

Known Health Effect	Exposure Quantified	Number of Effects Quantified	Economic Value Quantified
Reduced pulmonary function	✓		
Increased respiratory symptoms	✓	✓	✓
Increased asthma symptoms	✓	✓	✓
Hospital admissions	✓	✓	✓
Initiation of chronic bronchitis	✓	✓	✓
Minor restricted activity days	✓	✓	✓
Restricted activity days	✓	✓	✓
Headache	✓	✓	✓
Eye irritation	✓	✓	✓
Premature death (PM-related)	✓	✓	✓

Table 6-4. Number and value of reduced ozone-related health effects from achieving compliance with the 1-hr ozone NAAQS in 2007.

Effect	Number of Person-Days Reduced Annually	Economic Value
Cough (adults only)	345,800	$2,000,000
Chest Discomfort (all ages)	344,000	$3,100,000
Headache (all ages)	580,800	$5,700,000
Irritated Throat (all ages)	957,400	$9,300,000
Eye Irritation (all ages)	4,350,000	$36,800,000
Chest Symptoms (asthmatic children)	1,100,000	$16,700,000
TOTAL	7,678,000	$73,600,000

Table 6-5. The estimated reductions in the number and economic value of symptoms associated with various emission scenarios in 2007.

Symptom	Emission Scenario	Number of Cases	Economic Value
Eye Irritation	25% NO_x +15% VOC	497,000	$4,199,650
	Clean Diesel Fuel	572,000	$4,833,400
	45% NO_x +15% VOC	755,000	$6,379,750
	65% NO_x +15% VOC	1,740,000	$14,703,000
	75% NO_x +15% VOC	2,730,000	$23,068,500
	85% NO_x +15% VOC	4,300,000	$36,335,000
	Rollback to 1-hr NAAQS	4,350,000	$36,757,500
Cough	25% NO_x +15% VOC	30,610	$177,538
	Clean Diesel Fuel	40,100	$232,580
	45% NO_x +15% VOC	62,520	$362,616
	65% NO_x +15% VOC	135,090	$783,522
	75% NO_x +15% VOC	208,400	$1,208,720
	85% NO_x +15% VOC	322,400	$1,869,920
	Rollback to 1-hr NAAQS	345,800	$2,005,640
Headache	25% NO_x +15% VOC	52,620	$513,045
	Clean Diesel Fuel	68,870	$671,483
	45% NO_x +15% VOC	107,430	$1,047,443
	65% NO_x +15% VOC	232,420	$2,266,095
	75% NO_x +15% VOC	358,800	$3,498,300
	85% NO_x +15% VOC	554,400	$5,405,400
	Rollback to 1-hr NAAQS	580,800	$5,662,800
Sore Throat	25% NO_x +15% VOC	87,010	$848,348
	Clean Diesel Fuel	113,890	$1,110,428
	45% NO_x +15% VOC	177,610	$1,731,698
	65% NO_x +15% VOC	384,700	$3,750,825
	75% NO_x +15% VOC	591,700	$5,769,075
	85% NO_x +15% VOC	915,200	$8,923,200
	Rollback to 1-hr NAAQS	957,400	$9,334,650
Chest Discomfort	25% NO_x +15% VOC	30,851	$280,744
	Clean Diesel Fuel	40,450	$368,095
	45% NO_x +15% VOC	63,050	$573,755
	65% NO_x +15% VOC	136,780	$1,244,698
	75% NO_x +15% VOC	211,300	$1,922,830
	85% NO_x +15% VOC	327,170	$2,977,247
	Rollback to 1-hr NAAQS	344,000	$3,130,400

Table 6-6. Number and value of reduced minor restricted activity days (MRADs) from achieving compliance with the annual $PM_{2.5}$ NAAQS in 2007.

Effect	Number of MRADs (adjusted for $PM_{2.5}$ ratio)	Range of dollar values
MRADs for ages 20 to 64	606,000	$28,480,000
MRADS for age > 64	70,600	$3,320,000
TOTAL	676,600	$31,800,000

Table 6-7. Number and value of reduced restricted activity days (RADs) from achieving compliance with the annual $PM_{2.5}$ NAAQS in 2007.

Effect	Number of RADs (adjusted for $PM_{2.5}$ ratio)	Dollar values
Restricted activity days – ages 20 to 64	182,500	$16,000,000
Restricted activity days - age > 64	21,200	$1,100,000
TOTAL	203,700	$17,100,000

Table 6-8. Number and value of reduced cases of chronic bronchitis from achieving compliance with the annual $PM_{2.5}$ NAAQS in 2007.

Effect	Number of cases (unadjusted for $PM_{2.5}$ ratio)	Number of cases (adjusted for $PM_{2.5}$ ratio)	Range of dollar values
Chronic bronchitis – ages 20 to 64	637	1071	$203,200,000 - $341,600,000
Chronic bronchitis – age > 64	74	125	$23,600,000 - $39,900,000
TOTAL	711	1,196	$226,800,000 - $381,500,000

Table 6-9. Baseline incidences and PM exposure-response coefficients (beta values) for hospital admissions.

Health Outcome	Epidemiologic Study	Beta Value	Adjusted Beta	Baseline Incidence (per 100,000)
Hospital Admissions (Pneumonia)	Schwartz (1994a)	0.00174		
	Schwartz (1994b)	0.00115		
	Schwartz (1994c)	0.00157		
	Schwartz (1996)	0.00103		
	Average	0.01373	0.00226	229
Hospital Admissions (COPD)	Schwartz (1994a)	0.00239		
	Schwartz (1994b)	0.00202		
	Schwartz (1994c)	0.00451		
	Schwartz (1996)	0.00316		
	Average	0.00302	0.005	103
Hospital Admissions (Congestive Heart Failure)	Schwartz and Morris (1995)	0.00098	0.00161	231
Hospital Admissions (Ischemic Heart Disease)	Schwartz and Morris (1995)	0.00056	.000922	450

Table 6-10. Number and value of reduced pneumonia, COPD, congestive heart failure, and ischemic heart disease from achieving compliance with the annual $PM_{2.5}$ NAAQS in 2007.

Effect (Age > 64)	Number of hospitalizations (unadjusted for $PM_{2.5}$ ratio)	Number of hospitalizations (adjusted for $PM_{2.5}$ Ratio)	Range of dollar values
Pneumonia	3	6	$34,100-68,300
COPD	3	6	$35,000-70,000
Ischemic Heart Disease	3	5	$44,500-74,200
Congestive Heart Failure	3	4	$35,900-47,800
TOTAL	12	21	$149,500-260,000

Table 6-11. Number and value of reduced hospitalizations from achieving compliance with the annual $PM_{2.5}$ NAAQS in 2007.

All respiratory-related hospitalizations by age	Number of hospitalizations (unadjusted for $PM_{2.5}$ ratio)	Number of hospitalizations (adjusted for $PM_{2.5}$ Ratio)	Range of dollar values
< Age 20	56	92	$500,000-800,000
Ages 20-64	118	194	$1,000,000-$1,700,000
> Age 64	14	23	$100,000-200,000
TOTAL	188	309	$1,600,000 - $2,700,000

Table 6-12. Aggregate economic benefits of reduced fine particle and ozone exposures associated with achieving the annual $PM_{2.5}$ NAAQS and 1-hr ozone NAAQS in 2007.

Health Effect	Affected Group	Number of Health Effects[a]		Dollar Values (in millions of 1997 dollars)	
		Unadjusted[a]	Adjusted	Individual Group	Total
Mortality	Ages 30-64	-	132	$778.8	
	Ages > 64	-	303	$1,787.7	
Total Mortality	All	-	435	-	$2,566
Chronic Bronchitis	Ages 20-64	637	1071	$203.2 - 341.6	
	Ages > 64	74	125	$23.6 - 39.9	
	All	711	1,196	-	$227 – 381
All Respiratory Hospitalizations	Ages < 20	56	92	$ 0.5 - 0.8	
	Ages 20-64	118	194	$1.0 - 1.7	
	Ages > 64	14	23	$0.1 - 0.2	
	All	188	309	-	$1.6 -2.7
Cardiac Hospitalizations	Ages > 64	6	9	-	$0.1 - 0.1
Minor Restricted Activity Days	Ages 20-64	-	606,000	$28.5	
	Ages > 64	-	70,600	$3.3	
	All	-	676,600	-	$31.8
Restricted Activity Days	Ages 20-64	-	182,500	$16.0	
	Ages > 64	-	21,200	$1.1	
	All	-	203,700	-	$17.1
Cough	Ages > 20	345,800	345,800	-	$2.0
Chest Discomfort	All Ages	344,000	344,000	-	$3.1
Headache	All Ages	580,800	580,800	-	$5.7
Irritated Throat	All Ages	957,400	957,400	-	$9.3
Eye Irritation	All Ages	4,350,000	4,350,000	-	$36.8
Chest Symptoms	Asthmatic Children, Ages 7-13	1,100,000	1,100,000	-	$16.7
Total Morbidity[b]	All	-	-	-	$351 - $507[b]
TOTAL BENEFITS					$2,917 - $3,073

[a] Health effects are estimated assuming all of the particle-induced effects found in PM_{10} epidemiologic studies are due to fine particles (adjusted estimates) and due to fine and coarse particles (unadjusted).

[b] Approximately $74 million of the total morbidity is attributable to ozone exposures.

Table 6-13. Number and value of reduced premature mortality from achieving compliance with the annual $PM_{2.5}$ NAAQS in 2007.

Mortality by age	Number of premature deaths[1]	Dollar values
Age 30-64	132	$ 778,800,000
Age > 64	303	$1,787,700,000
TOTAL	435	$2,566,500,000

[1] The original work in Pope et al., 1995 is based on $PM_{2.5}$, so there is no unadjusted value.

Table 6-14. Relative reductions in fine particle-related effects from compliance with the annual $PM_{2.5}$ NAAQS and 1-hr ozone NAAQS in 2007.

Effect	Benefits of annual $PM_{2.5}$ NAAQS compliance starting from			Benefits of clean diesel fuel[d]	Benefits of the 1998 Ozone SIP[e]
	Scenario 1[a]	Scenario 2[b]	Scenario 3[c]		
Reduction in Number of Deaths Per Year	435	409	418	44	44
Reduction in Number of Chronic Bronchitis Cases Per Year	711	668	687	81	81
Reduction in Number of Reduced Activity Days Per Year	203,700	201,300	197,000	24,000	24,000

[a] 2007 baseline projected inventory

[b] Emissions growth rates limited to population growth rate

[c] Dust content of PM inventory limited

[d] Scenario 1 + clean diesel fuel

[e] Scenario 1 + ozone SIP

7. CONCLUSIONS AND RECOMMENDATIONS FOR FURTHER RESEARCH

7.1 EMISSIONS, AIR QUALITY, AND EXPOSURE

Baseline exposure conditions in Houston were estimated from the readily available air quality and population data. These included ambient ozone data collected in 1993-1995, ambient $PM_{2.5}$ data collected in 1997-1998, and 1990 census population data. The $PM_{2.5}$ and population data were projected to 1993 (the base year for planning) and spatially mapped to 4 km x 4 km grids for use in grid-based exposure assessment.

Future-year air quality conditions were estimated using a combination of emissions information and air quality models, including a grid-based CAMx photochemical model and simple rollback models. Future-year population exposure to ozone was estimated for 2007 baseline conditions and potential 2007 controlled conditions. The population ozone exposures were estimated for cases with 15 percent VOC emissions reductions and five different NO_x emissions reduction levels: 25, 45, 65, 75, and 85 percent NO_x, as well as with hypothetical implementation of a cleaner burning diesel fuel in stationary and mobile sources. The 15 percent VOC and 85 percent NO_x emissions reductions case corresponds to the 1998 ozone SIP control requirements and represents conditions that are likely to show compliance with the 1-hr ozone NAAQS in 2007. Future-year population exposure to $PM_{2.5}$ was estimated for the future baseline case, an annual $PM_{2.5}$ NAAQS compliance case, a clean diesel fuel case, and a case with the 1998 ozone SIP emission reductions.

Noteworthy characteristics and results of the analysis are as follows:

- Updated VOC and NO_x emissions estimates used in the photochemical modeling produced comparable results to those produced with previous emissions estimates. Given the high biogenic VOC emission rates in the Houston area, the simulated future ozone levels are still primarily controlled by the regional NO_x emissions, rather than the anthropogenic VOC emissions.
- Revised PM and SO_x emissions from the National Particle Inventory were used, along with VOC and NO_x emission estimates, in a speciated PM rollback model to estimate $PM_{2.5}$ conditions in earlier and later years. The PM and PM-related emissions (SO_x and ammonia) were much less well characterized than the VOC and NO_x emissions. The uncertainty in PM and PM-related emissions remains high. However, with the high background $PM_{2.5}$ concentrations and the linear modeling approach used for the study, the future-year $PM_{2.5}$ concentrations estimates were not strongly sensitive to alternate base-case emission assumptions. The maximum annual average $PM_{2.5}$ concentration in 2007 was about 19 $\mu g/m^3$ regardless of assumptions.
- The ozone exposure estimates were made mostly assuming Houston residents were exposed to outdoor concentrations at all times. Population exposure estimates that accounted for human time-activity and the modifying effects of enclosures (buildings and vehicles) suggested there would be 85 percent fewer exposures above the 1-hr NAAQS than in the "all outdoors" or ambient exposure simulations. There may be

greater disparity between personal ozone exposure levels and outdoor levels in Houston than in other less air-conditioned cities.

- A relatively large fraction of the Houston residents are exposed to high concentrations of $PM_{2.5}$ and ozone. We estimate 62 percent of the population in 1993 and 79 percent of the population in 2007 (without additional control measures) would be exposed to annual $PM_{2.5}$ concentrations above the NAAQS. For the 1993-1995 time frame, we estimate there were 26 million and 72 million person-days per year with exposures above the 1-hr and 8-hr NAAQS levels, respectively. In 2007 without additional controls, we estimate there will be 27 million and 84 million person-days per year with exposures above the 1-hr and 8-hr NAAQS levels, respectively. The 1998 Ozone SIP emissions reductions are likely to reduce ozone exposures above the 1-hr and 8-hr standards by 99 and 90 percent, respectively. The geographic distribution of exposures above the standards are similar, suggesting the same individuals are exposed to high ozone and $PM_{2.5}$ concentrations. The overlap in population exposure regions and the commonalities of some precursor emissions points to the need for multi-pollutant control strategy development efforts.
- Regulatory agencies have been working on the Houston ozone problem for more than 20 years, while little attention has been given to $PM_{2.5}$ until the recent promulgation of the NAAQS. The number of exceedances and the severity of ozone exposure concentrations has been significantly reduced in this period. The smaller health benefits estimates for achieving the ozone NAAQS compared to the $PM_{2.5}$ NAAQS partially reflects the work that has already been done to improve ozone air quality in Houston.

7.2 HEALTH EFFECTS

The review of health effects literature published before and after the last EPA Ozone and PM Criteria Documents reaffirmed concerns for ozone and PM health effects at and above the NAAQS levels. Health effects of benzene, 1,3-butadiene, and formaldehyde at ambient levels are less thoroughly studied. Key findings of this element of the study area include:

- Coherence across multiple types of studies (animal toxicological, human clinical, and epidemiological) supports the conclusion that significant adverse health effects in humans result from exposure to ozone and fine particles in the complex urban atmosphere at levels typical of the Houston atmosphere.
- The coherence of the cascade of effects – changes in lung structure and function, acute symptoms such as cough and exacerbation of asthma, respiratory illness, and mortality – strengthens the conclusion that effects observed in epidemiological studies result from pollutant exposure.
- Recent studies suggest an association between ozone and premature mortality. Until this body of evidence expands to include more independent studies demonstrating consistency of this result, we have chosen not to quantify this effect. For assessment of the acute effects of ozone exposure, ozone concentration-response functions were

identified that provide a basis for estimating reductions in numerous symptoms, including chest symptoms (wheeze and phlegm production), eye irritation, cough, chest congestion, throat irritation, and headache.

- The health outcomes associated with exposures to particles tend to be chronic in nature rather than acute. Quantitative evaluations of changes in PM exposures and health effects were made for the following health outcomes: days of restricted activities due to air pollutant-induced symptoms or illness, days of minor restricted activities, cases of chronic bronchitis, hospital admissions for respiratory and other relevant diseases, and premature deaths.
- Annual average concentrations of formaldehyde, 1,3-butadiene, and benzene are above accepted risk levels at some Houston monitors, however, the location of those monitors is not representative of the general population's exposure, so we do not currently have an appropriate basis from which to quantify any potential effects in the overall population.

7.3 BENEFITS OF AIR QUALITY IMPROVEMENTS

The results reported here characterize the magnitude of expected health benefits that would result from alternative emissions reduction scenarios. They should be regarded as indicative of scale rather than as precise measures. Nonetheless, they do represent the best estimates that can be constructed with information now available. The key results are:

- Total annual economic benefits of reduced exposure associated with achieving compliance with the 1-hr ozone and $PM_{2.5}$ NAAQS in 2007, relative to predicted 2007 concentrations, are in the range of $2.9 billion to $3.1 billion in Houston.
- The estimated economic value of lower exposure to fine particles outweighs the benefits of reduced ozone exposure. This is not because ozone is benign, but because the consequences of fine particle exposure are severe and are therefore associated with very large economic benefits to avoid even a relatively small number of effects. For example, we estimate there will be over 4 million fewer days of ozone-related eye irritation with 1-hr ozone NAAQS compliance and 435 fewer premature deaths at lower fine particle levels. Given that a day of eye irritation is valued at less than $10 and a life lost is valued at nearly $6 million, particle-related benefits dominate the benefits analysis.
- There are significant unquantified effects. Multiple studies have found that lung function is affected by exposure to ozone and to fine particles, but we have no basis on which to quantify the economic benefit of avoiding this effect. We do not yet know the consequences later in life, for example, that might result from short-term impairments in lung function experienced as a child.
- The health studies that support quantification of hospital admissions, chest symptoms in asthmatic children, and restricted activity days are all based on short-term exposure. They do not capture any effects on days when pollution levels are not as elevated but

that occur as the cumulative result of longer-term exposure at lower levels. In contrast, mortality and chronic bronchitis estimates are based on longer-term exposures and therefore reflect more completely the effects of exposure over time.

- The study used to determine the association between fine particle exposure and premature mortality did not assess deaths in the under 30 age group. Consequently, mortality risk to that population is not represented in the economic benefit estimates presented here.
- While ozone benefits are small compared to fine particle benefits, it should be noted that emissions controls that reduce ozone also reduce particles to varying degrees. For example, the 85 percent NO_x emissions and 15 percent VOC emissions reductions proposed in the 1998 ozone SIP are estimated to reduced PM-related deaths by 44 per year in 2007.
- Estimates of the benefits of full implementation of clean diesel fuel in mobile and stationary sources also show 44 fewer PM-related deaths annually. These estimates were made assuming emissions reductions comparable to those associated with California's clean diesel fuel. Implementation of a clean diesel fuel program reduces ozone exposures as well as PM exposures.
- Apart from the aggregate benefits to the region's population, there are significant benefits to some sub-groups. Asthmatic children, who represent a sensitive population, would each experience more than 50 fewer days a year of chest symptoms, on average, with NAAQS compliance.
- The ozone benefits estimated in this study are for compliance with the 1-hr standard. Work is currently underway in Texas to evaluate control requirements for compliance with the 8-hr standard. No attempt was made to estimate the emission reductions that would be required to meet the 8-hr standard or the benefits derived from meeting the standard. There is concern that even the 15 percent VOC reduction and an 85 percent NO_x emission reduction may be insufficient to attain compliance with the 8-hr NAAQS in Houston.

7.4 RECOMMENDATIONS FOR FURTHER RESEARCH

The process of assessing emissions, air quality, exposure, health effects, and economic valuation helps identify numerous areas where further research could improve the understanding of the air pollution effects. Salient areas in need of further research are listed below.

- Given the large benefits estimated for improvements in $PM_{2.5}$ air quality and the significant deficiencies in the current understanding of the $PM_{2.5}$ problem, further research is needed on many aspects of the problem. Research should be conducted to improve the characterization of ambient concentrations, background concentrations, and PM chemical composition in order to understand what is actually in the Houston air. Likewise, better information on PM and PM-related emissions and control options as

well as better air quality models (especially annual models with reasonable physics and chemistry) are needed to develop an understanding of the contribution of different sources to the problem. Emissions and meteorological information suitable for long-term applications of models would be particularly useful given that the annual NAAQS is the controlling standard in this region. Development of an understanding of the seasonal variations in emissions would be particularly useful. In addition, it is important to develop PM emission inventories that have chemical compositions that are consistent with ambient observations of primary PM components.

- Further research on the relationships between ozone and $PM_{2.5}$ precursor emissions is needed to begin exploring multi-species control strategies. Ultimately, a high quality modeling system and database will be needed to assess the effects of actual control measures on both ozone and $PM_{2.5}$ for the appropriate averaging times. The modeling system should be able to account for potential nonlinearities in the gas-phase and aqueous-phase chemistry as well as in the aerosol thermodynamics. At present, the contribution of NO_x emissions to $PM_{2.5}$ ammonium nitrate, the extent of ammonia emission limitations on aerosol nitrate levels and aerosol acidity, and the contribution of VOC emissions to secondary $PM_{2.5}$ organic material are not well known. Given that sulfate and organic material are the two most abundant chemical constituents of Houston $PM_{2.5}$, this research should include investigation of the roles of SO_2 and VOC emissions in aerosol formation.

- There is little information on the time-activity patterns of Houston residents and indoor/outdoor pollutant concentrations of ozone and $PM_{2.5}$ in different types of buildings or with different types of heating/air-conditioning/ventilation systems. This type of information is needed to make better estimates of personal exposure to these pollutants.

- As described in the report, there are myriad adverse physiological responses to pollutant exposure. Many of the physiological responses, such as structural changes in the lungs and airways, reduced lung function, and immune system responses, are logically related to the occurrence of disease in some portion of the population. We presently have no economic knowledge about what the benefits would be of preventing these responses and, thus, have not included the benefits in our analysis. Our inability to value these responses contributes to underestimation of the probable benefits of air quality improvements. Therefore, one important area of further research is interdisciplinary work by health scientists and economists to better model the link between physiological change and adverse health outcomes and to design contingent valuation studies to then determine the value of avoiding the more subtle pre-clinical effects.

- The preponderance of mortality appears to be among the elderly. Given this emerging health science finding, it is important to more clearly determine whether and by how much society values the lives of older people differently than lives in the age groups that current values are drawn from. This is also true for the lives of children, as there is preliminary research indicating higher risk of death in young infants in polluted environments.

- Society places significant weight on protecting children, but we have almost no basis to determine how to value, for example, chest symptoms in children relative to the adults on whom values are now based. Given the demonstrated sensitivity of asthmatic children, and the higher doses of pollution experienced by children in general, relative to adults, this is a key area of investigation.
- Emissions reductions will often reduce multiple pollutants simultaneously, but we typically assess the benefits of reducing one pollutant at a time. More investigation is needed of the joint value of reducing a set of adverse effects, rather than single effects.
- None of the health effects data are specific to Houston. It would be helpful to investigate and confirm the types of symptoms and other effects prevalent in the Houston area.

APPENDIX A

Isopleth Maps of 24-hr Average $PM_{2.5}$ Concentrations in the Houston Area for March 11, 1997 through March 12, 1998

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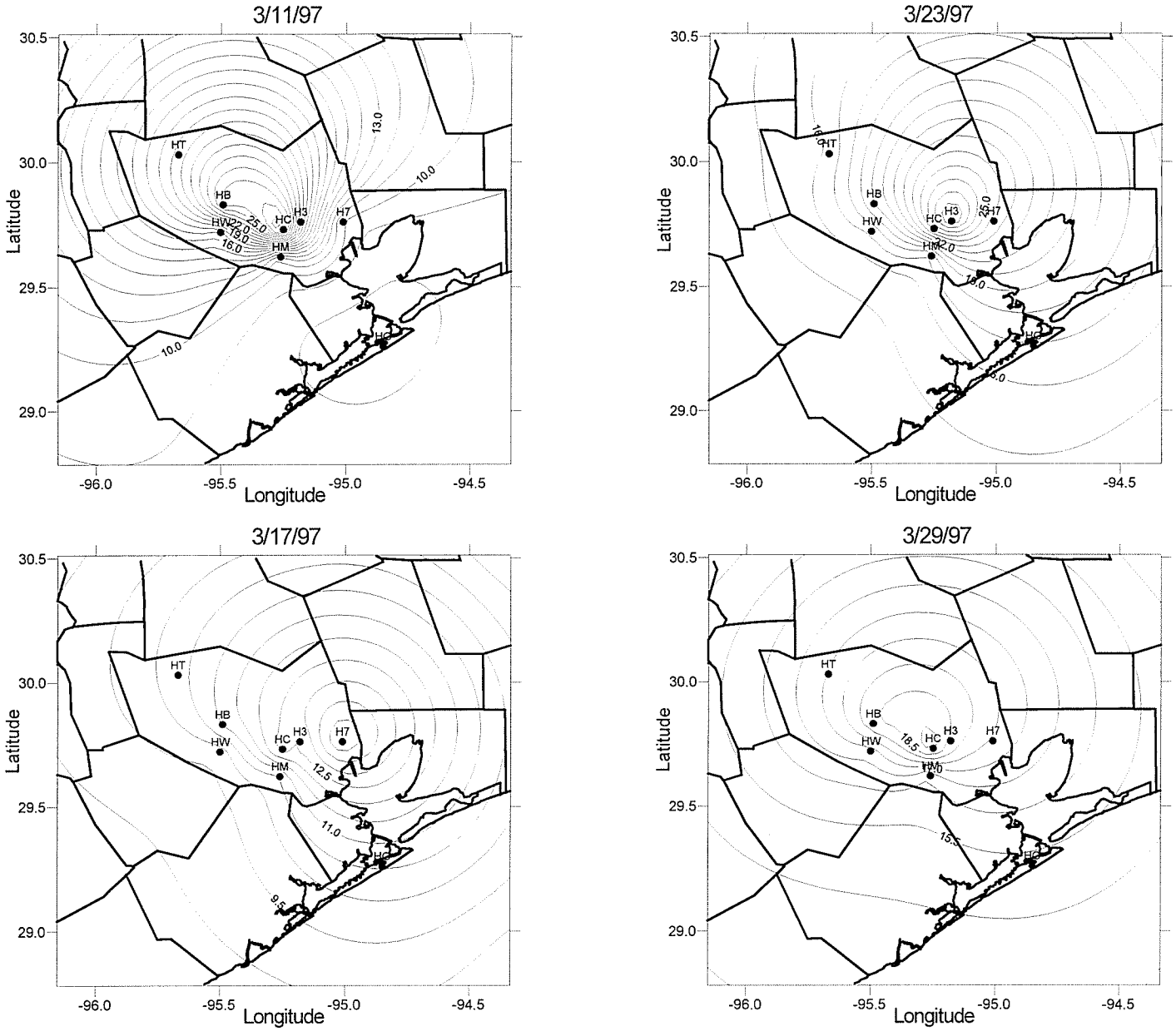
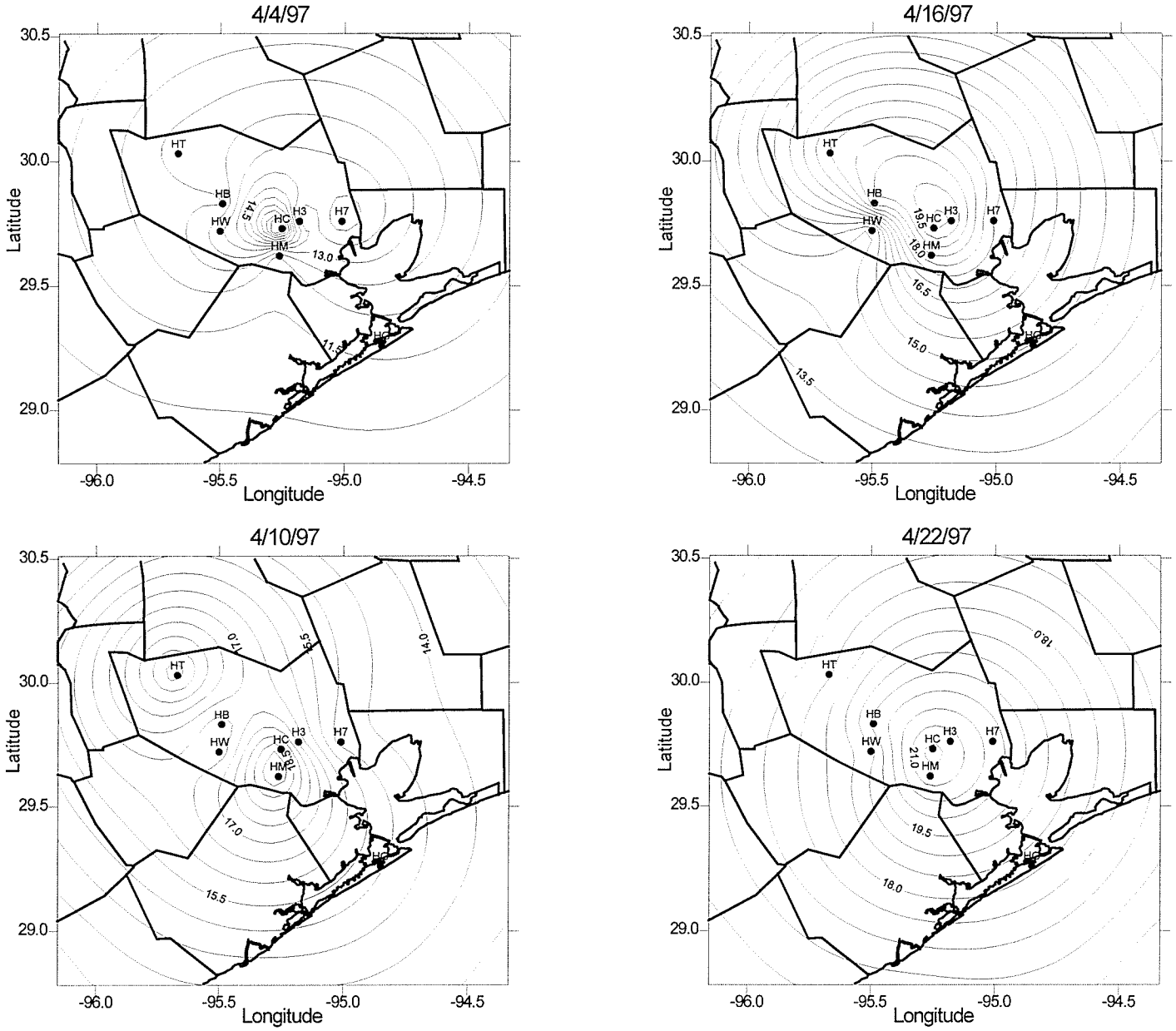


Isopleth Maps of 24-hr Average $PM_{2.5}$ Concentrations ($\mu g/m^3$) in the Houston Area



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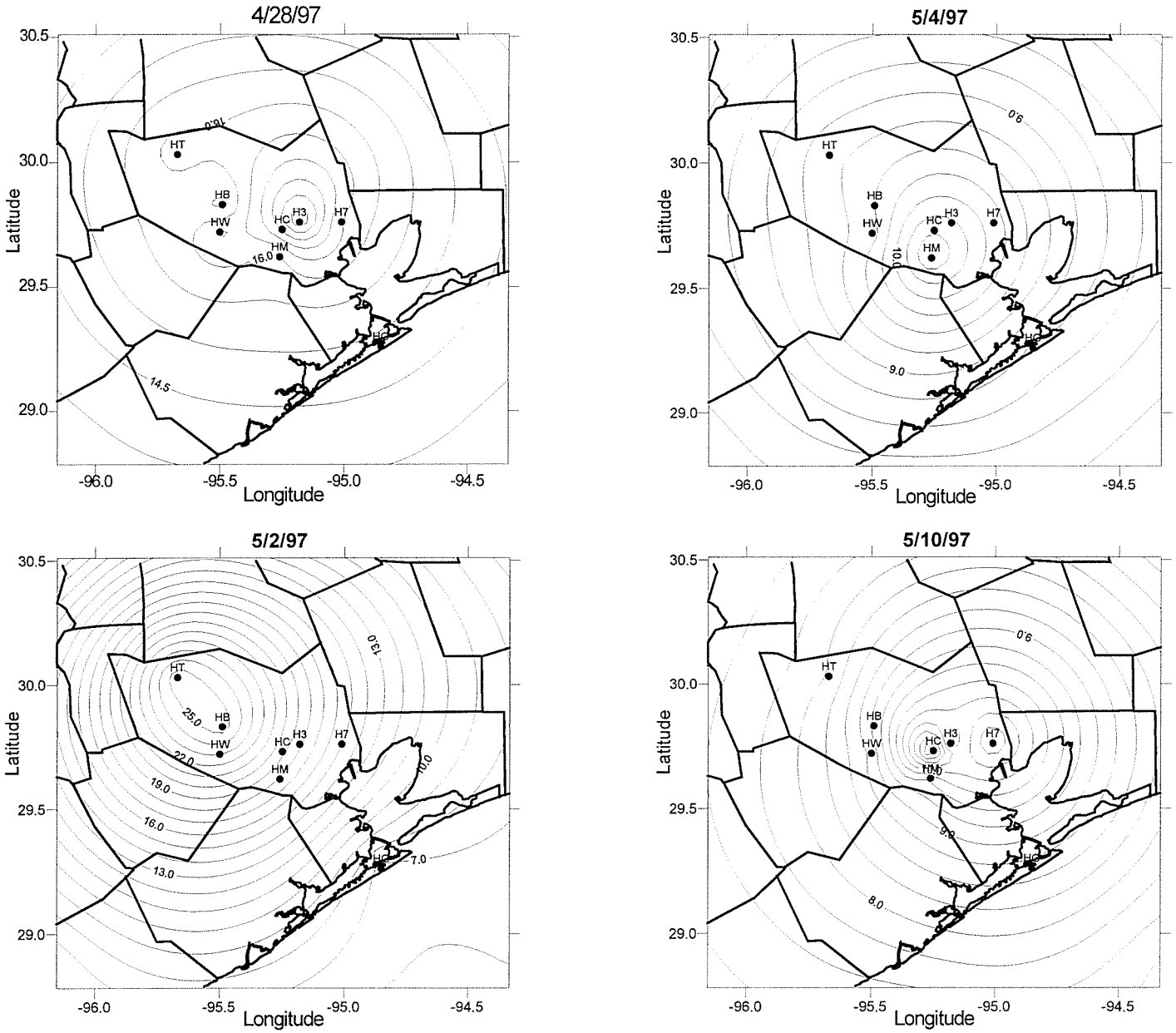

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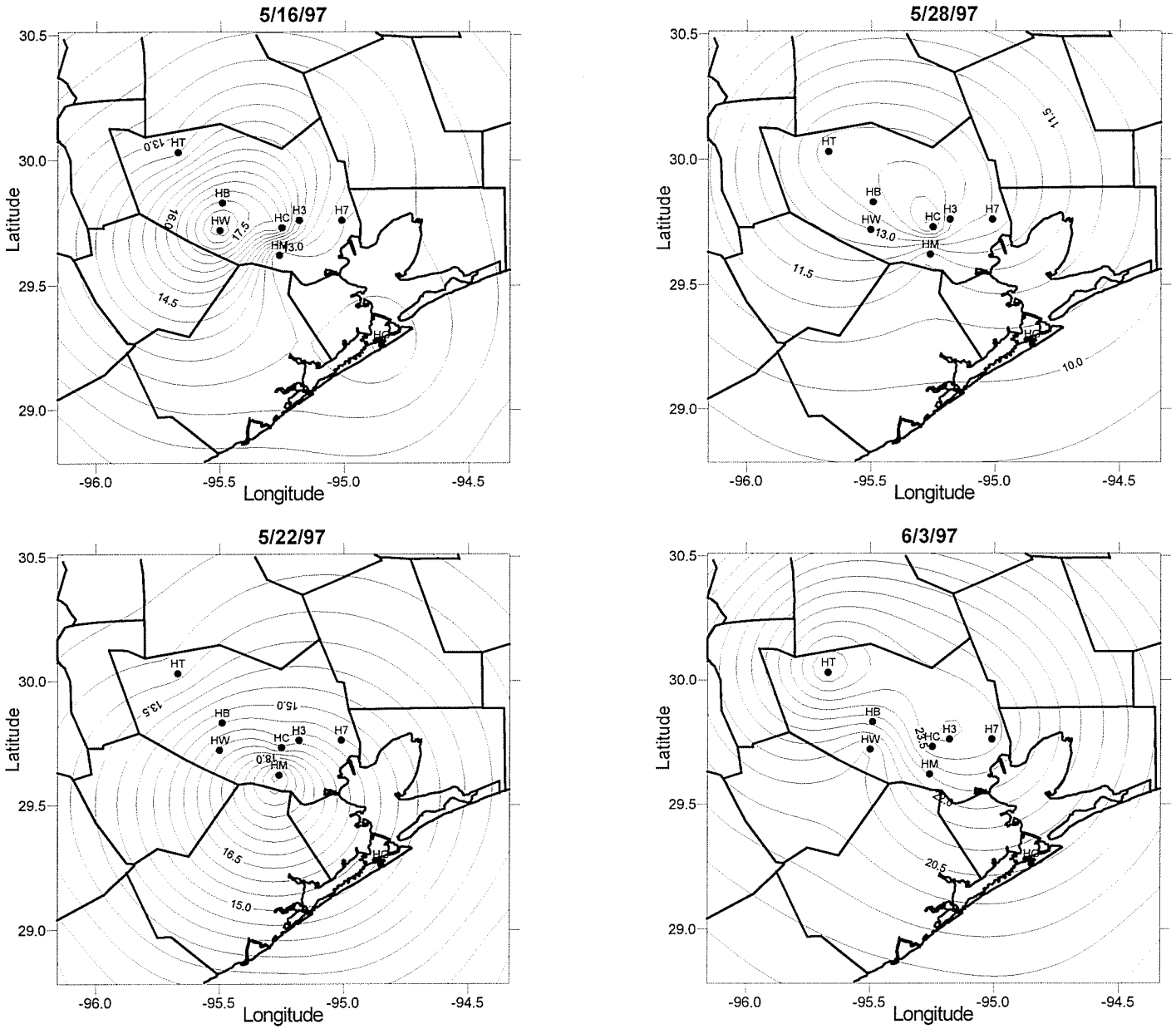
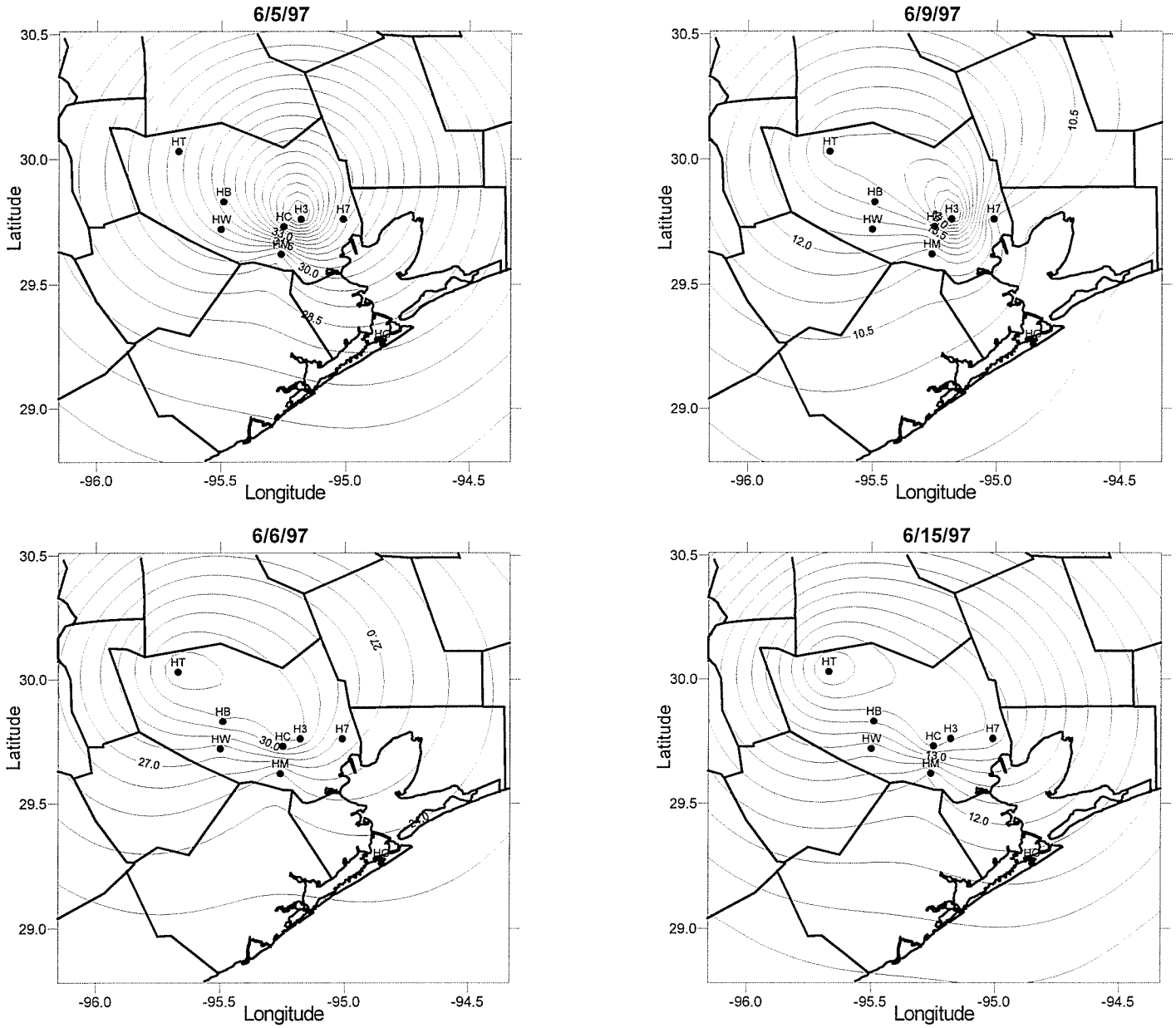


Isopleth Maps of 24-hr Average $PM_{2.5}$ Concentrations ($\mu g/m^3$) in the Houston Area



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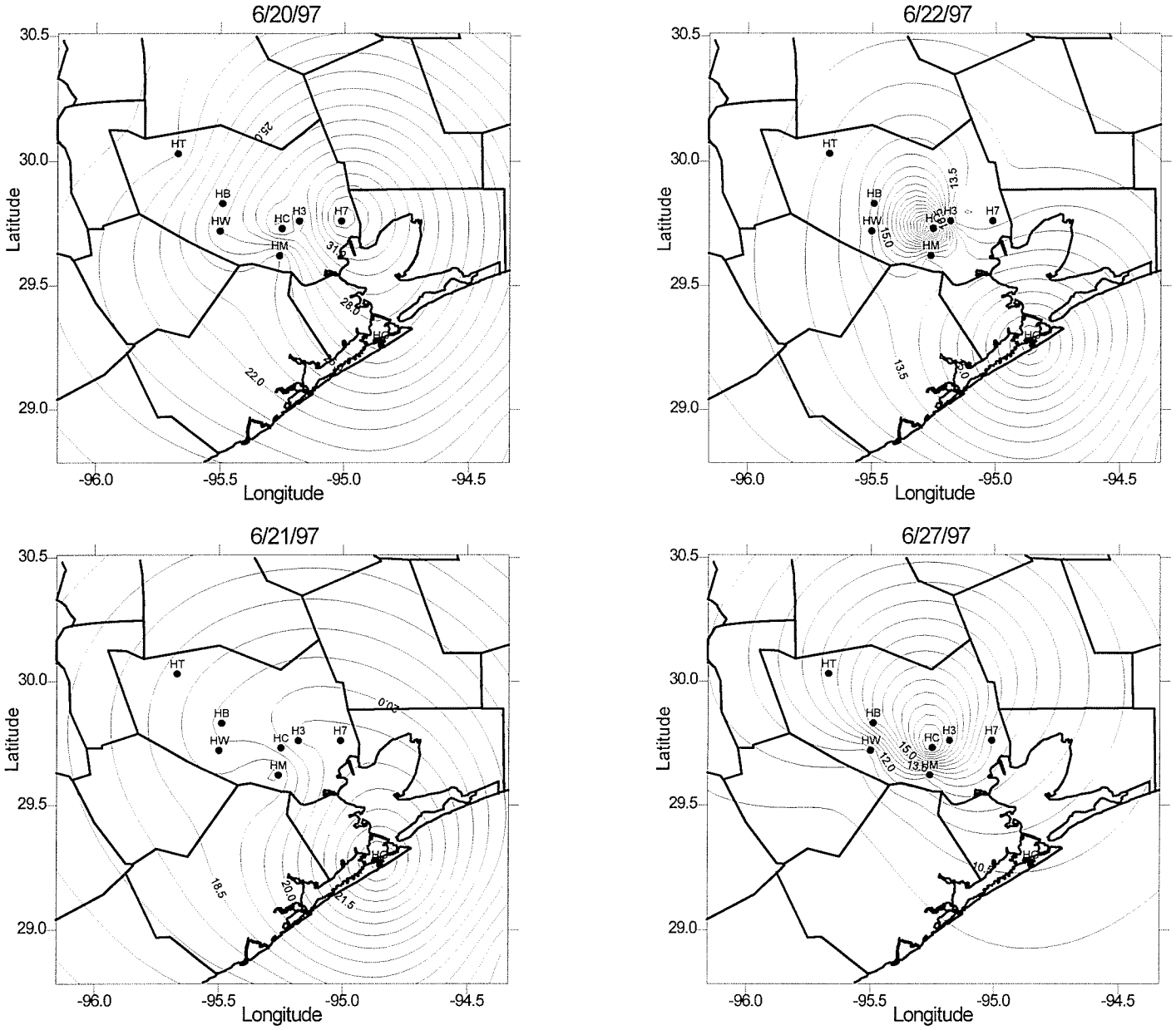

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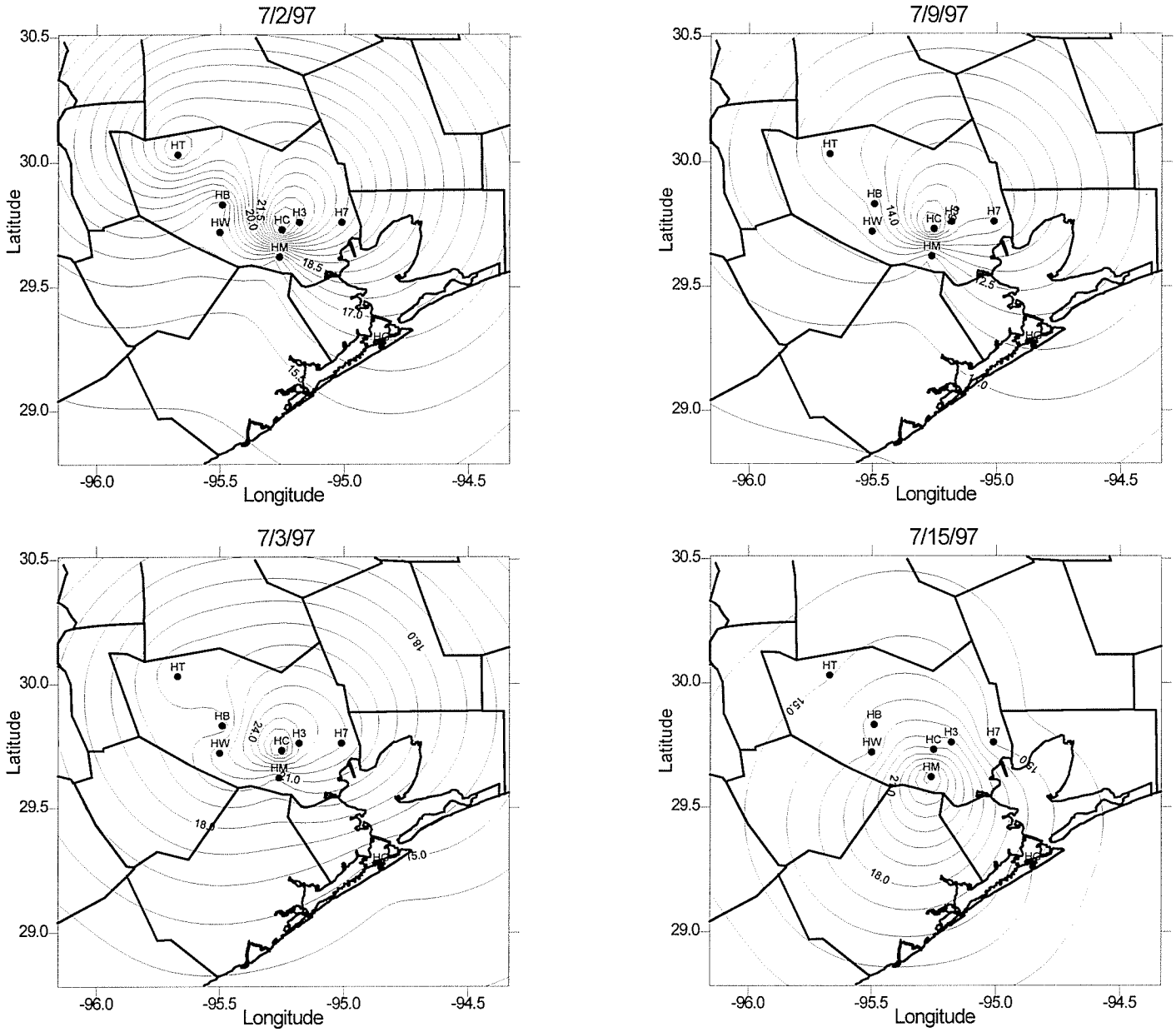
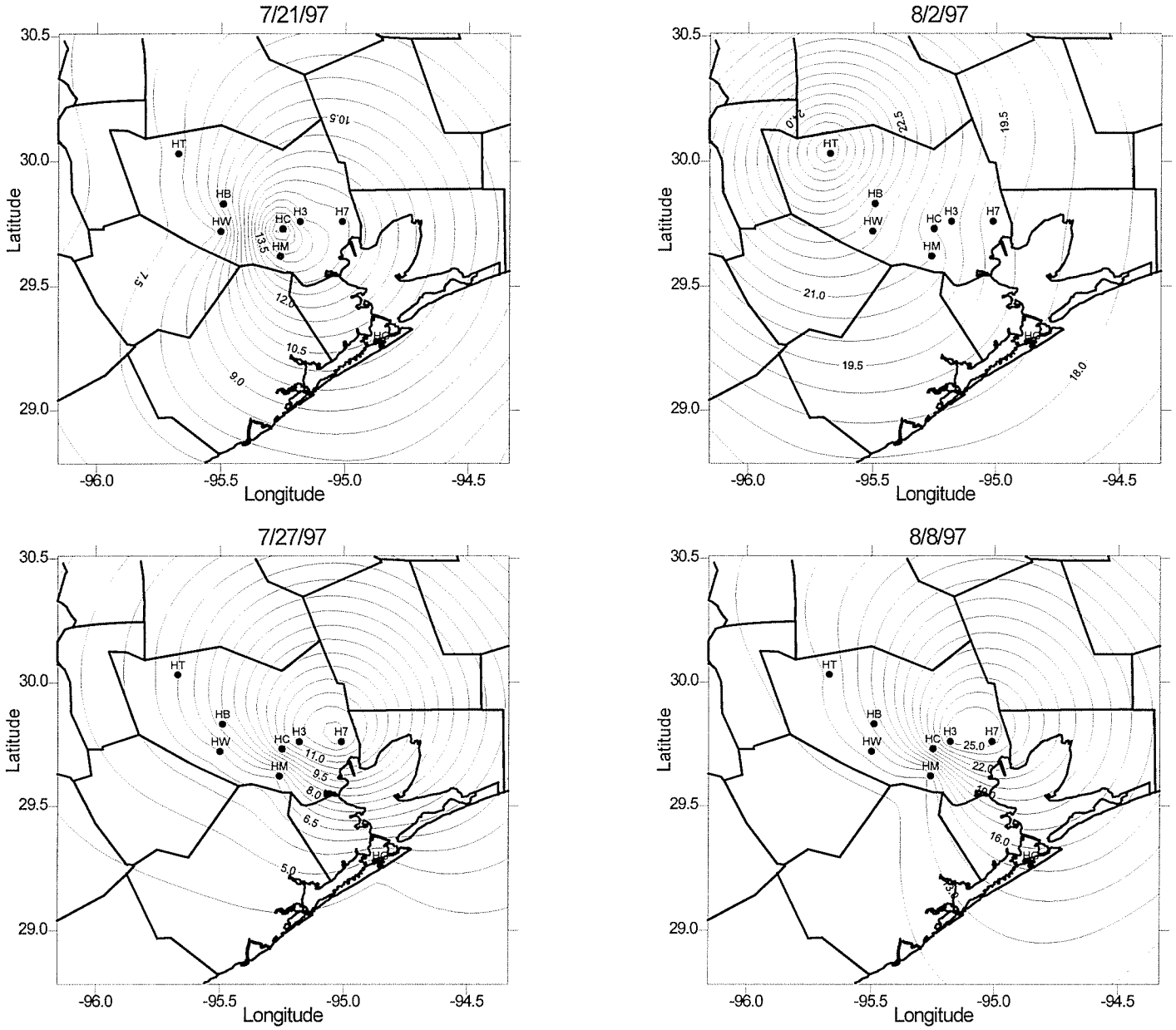


Isopleth Maps of 24-hr Average $PM_{2.5}$ Concentrations ($\mu g/m^3$) in the Houston Area



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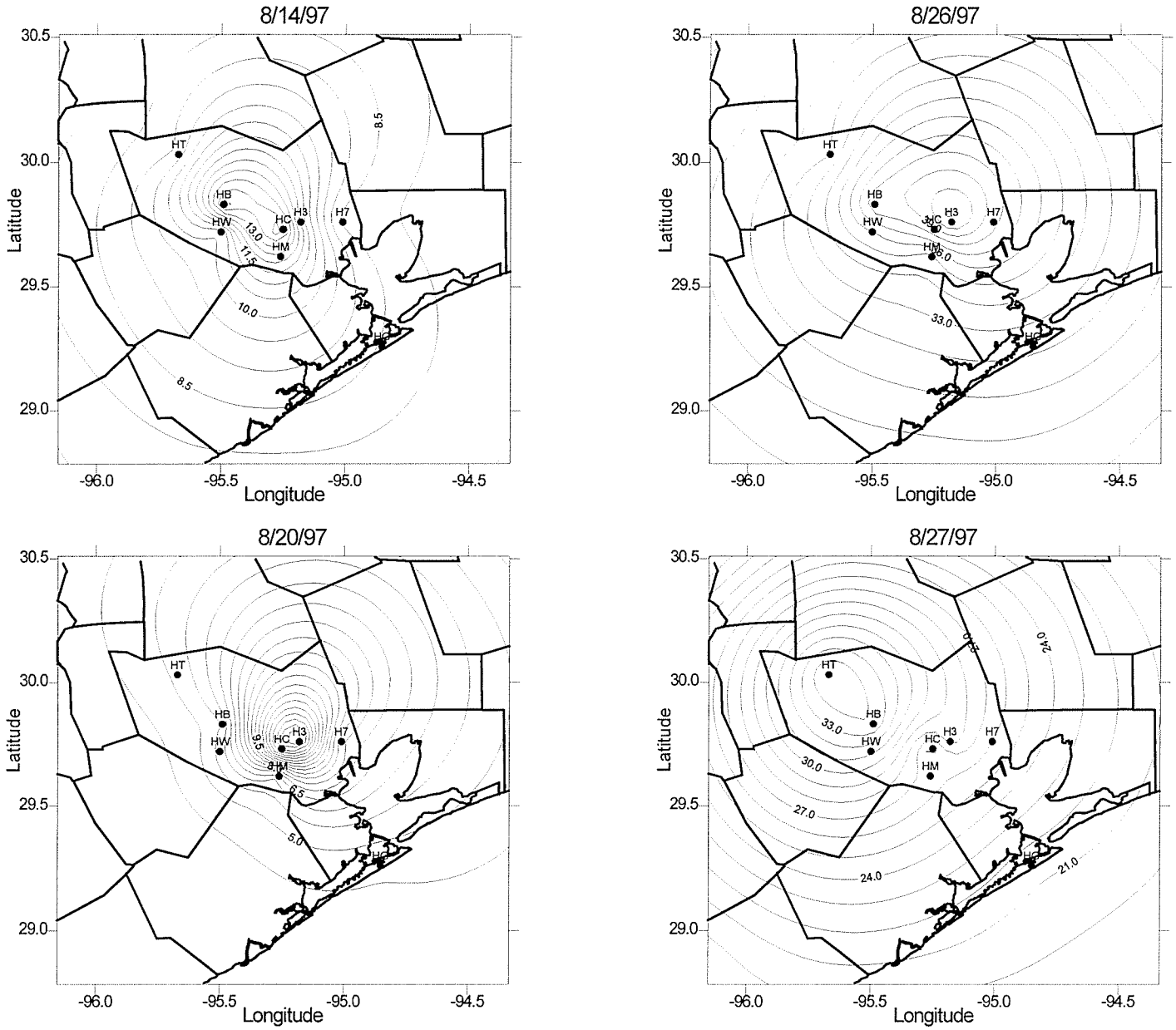

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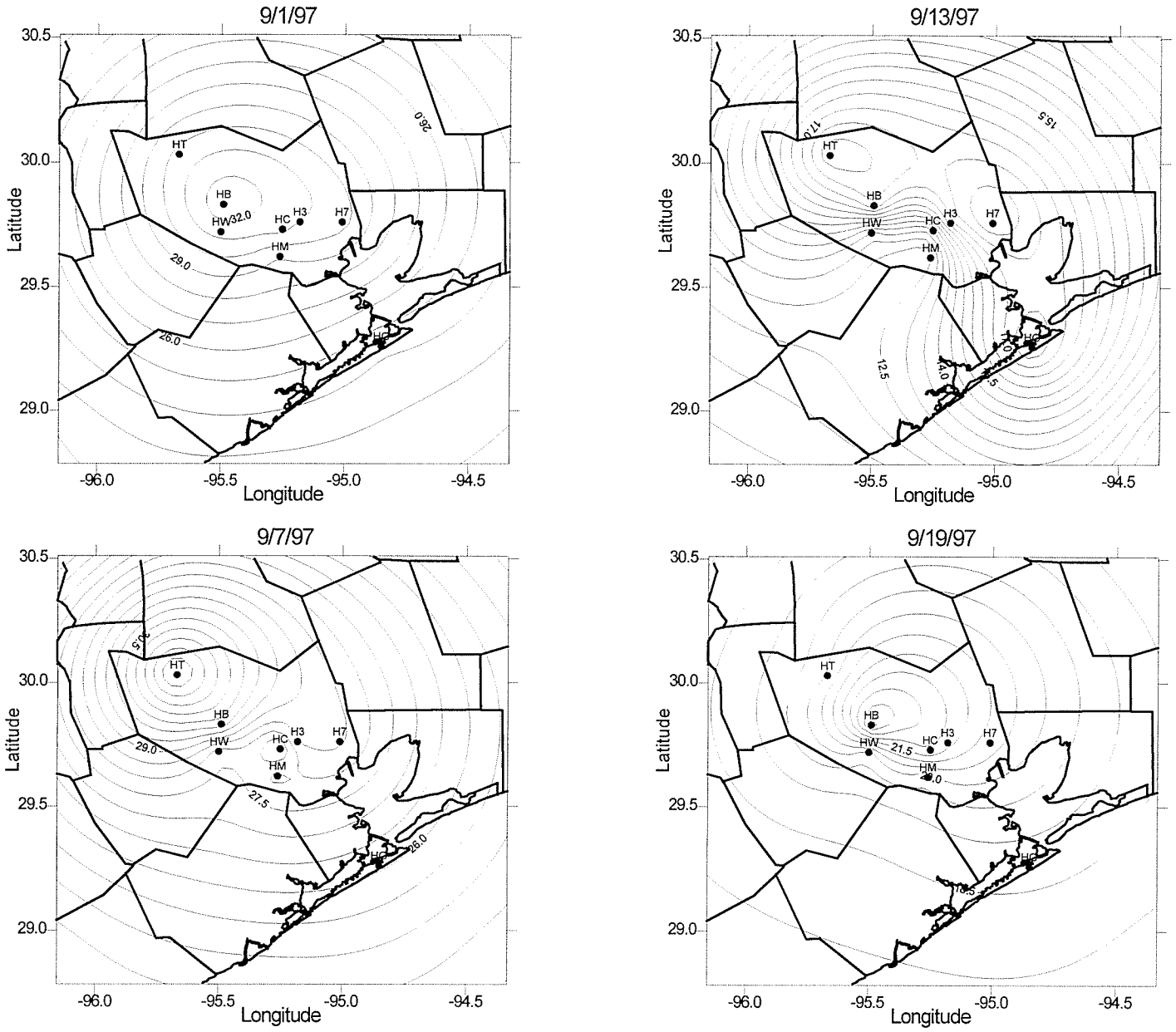

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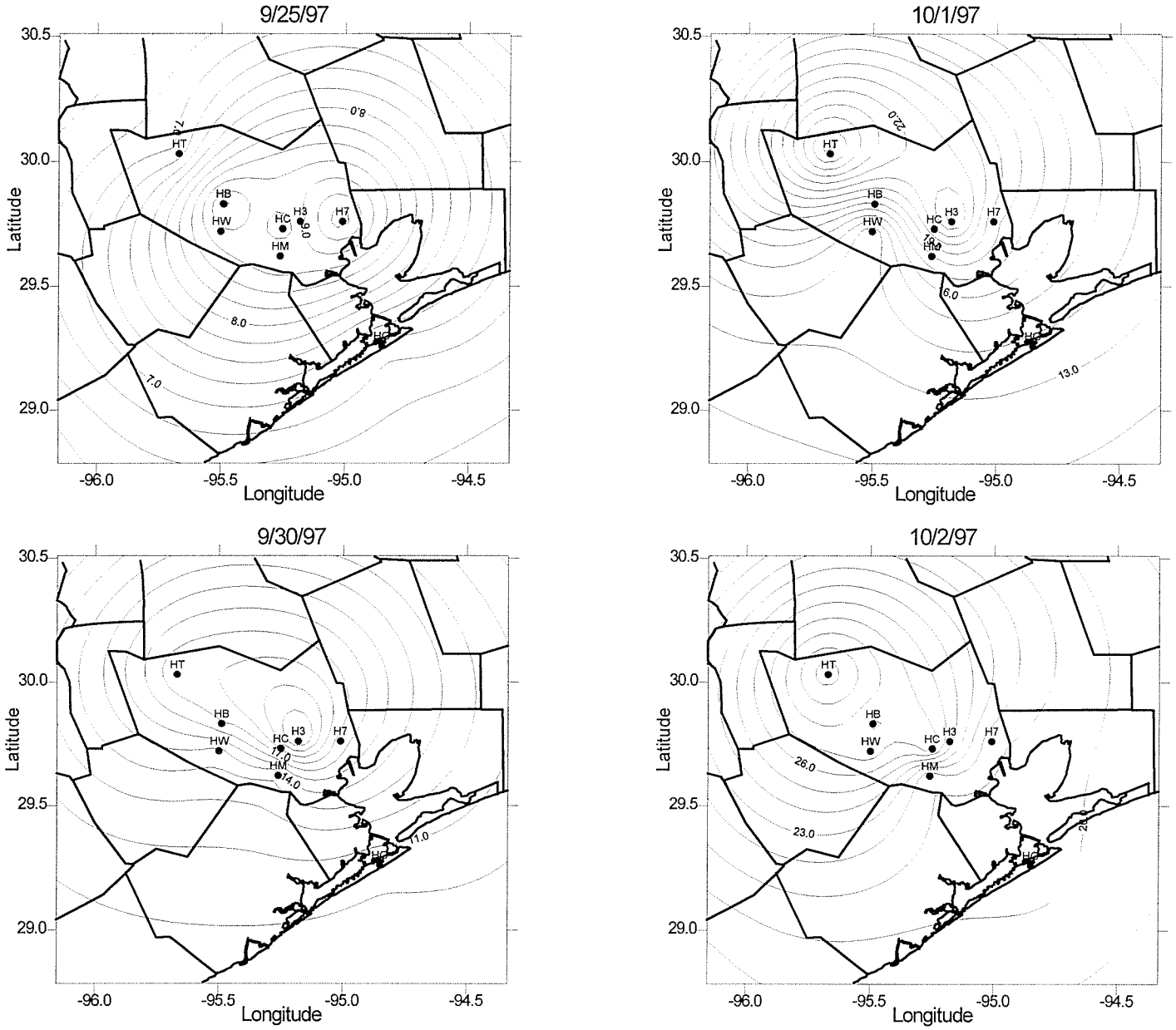

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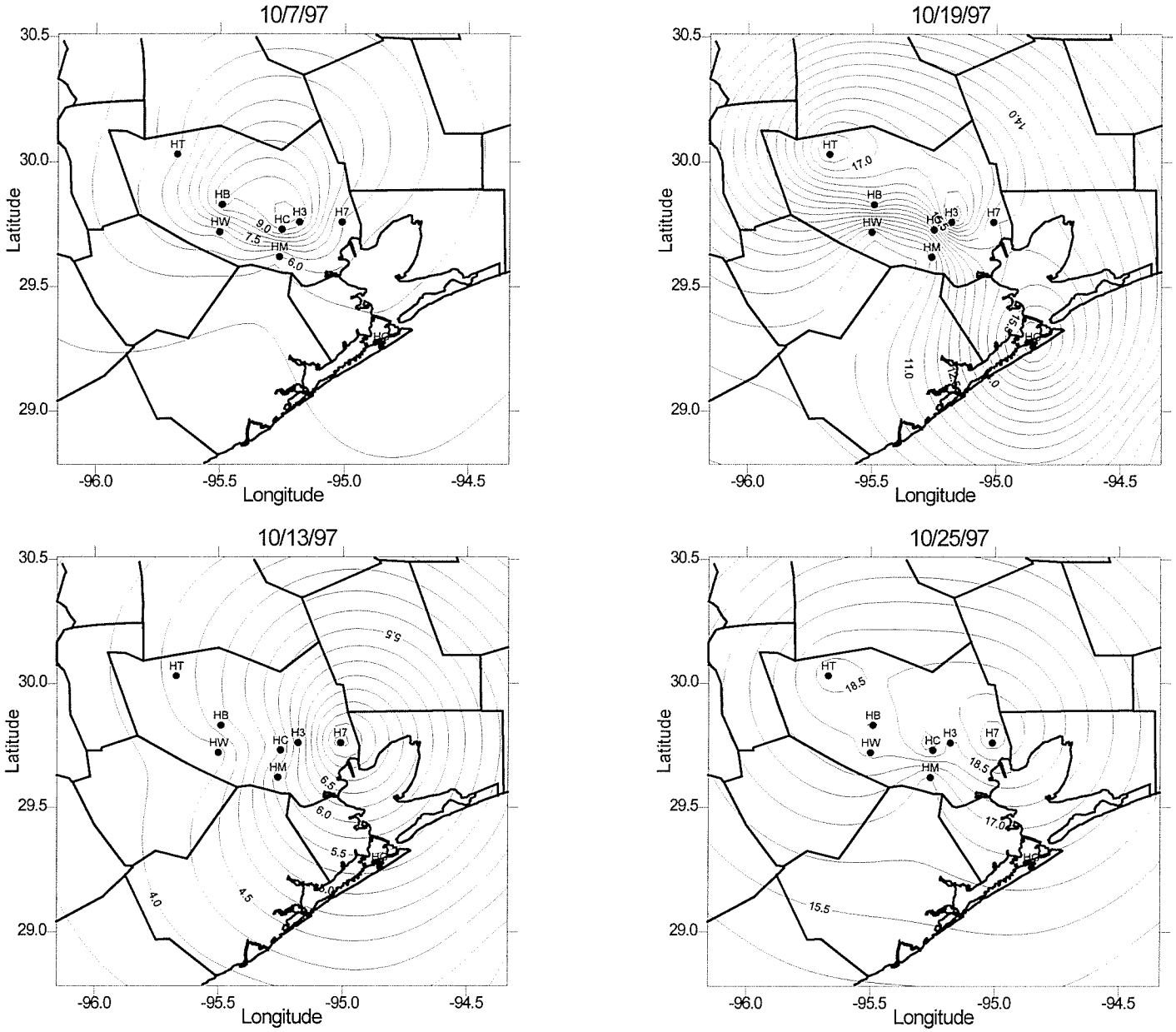

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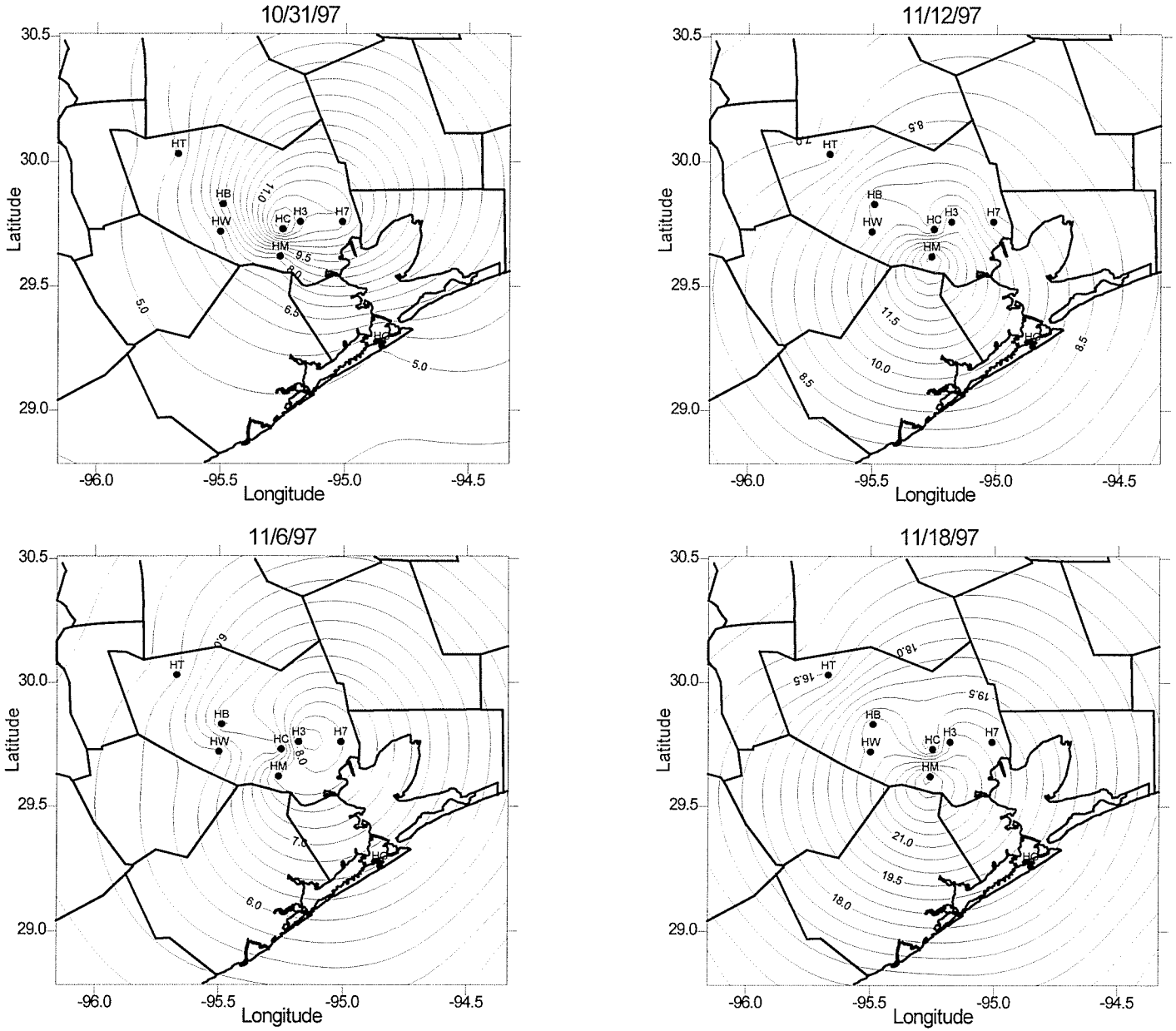

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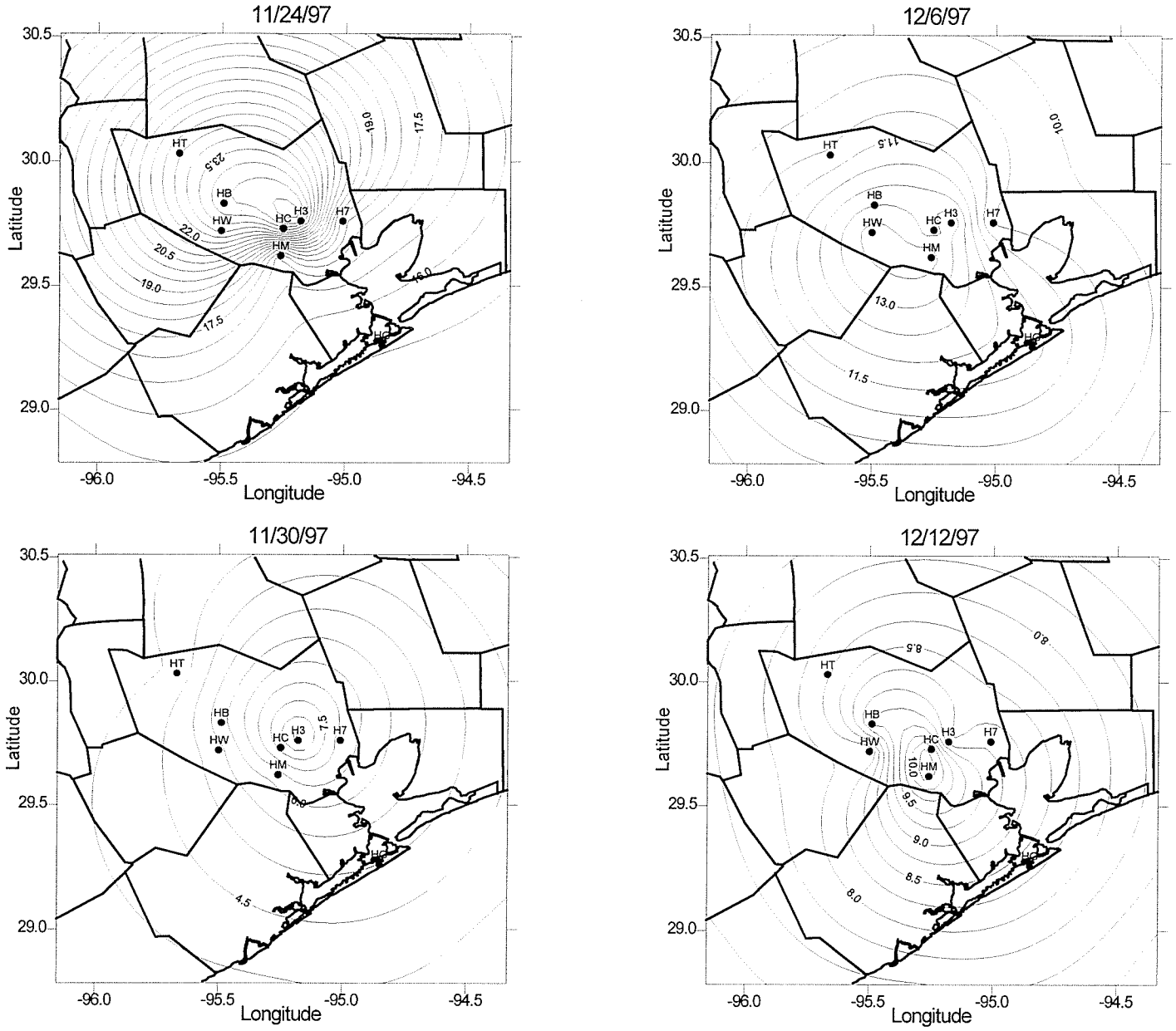

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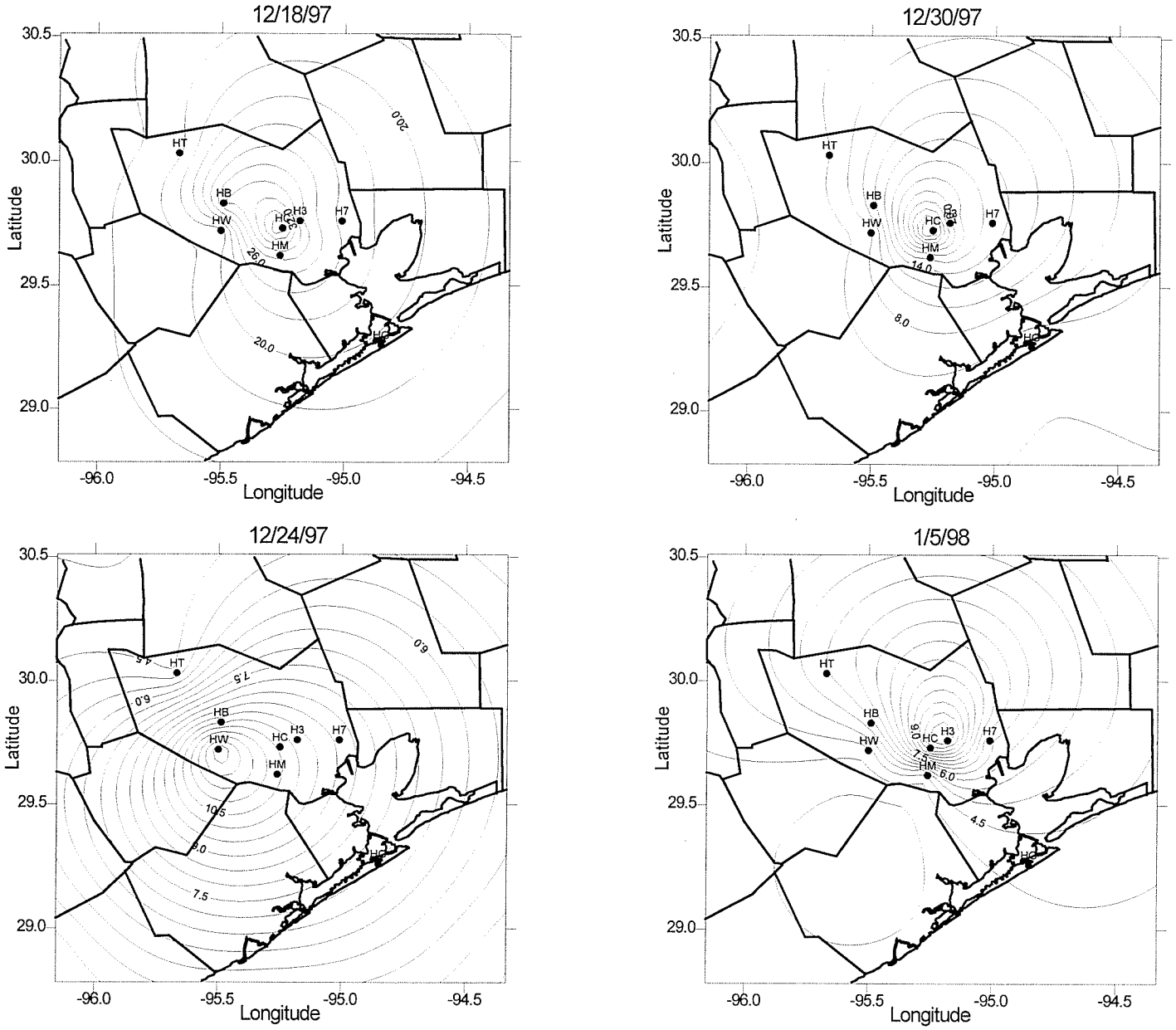

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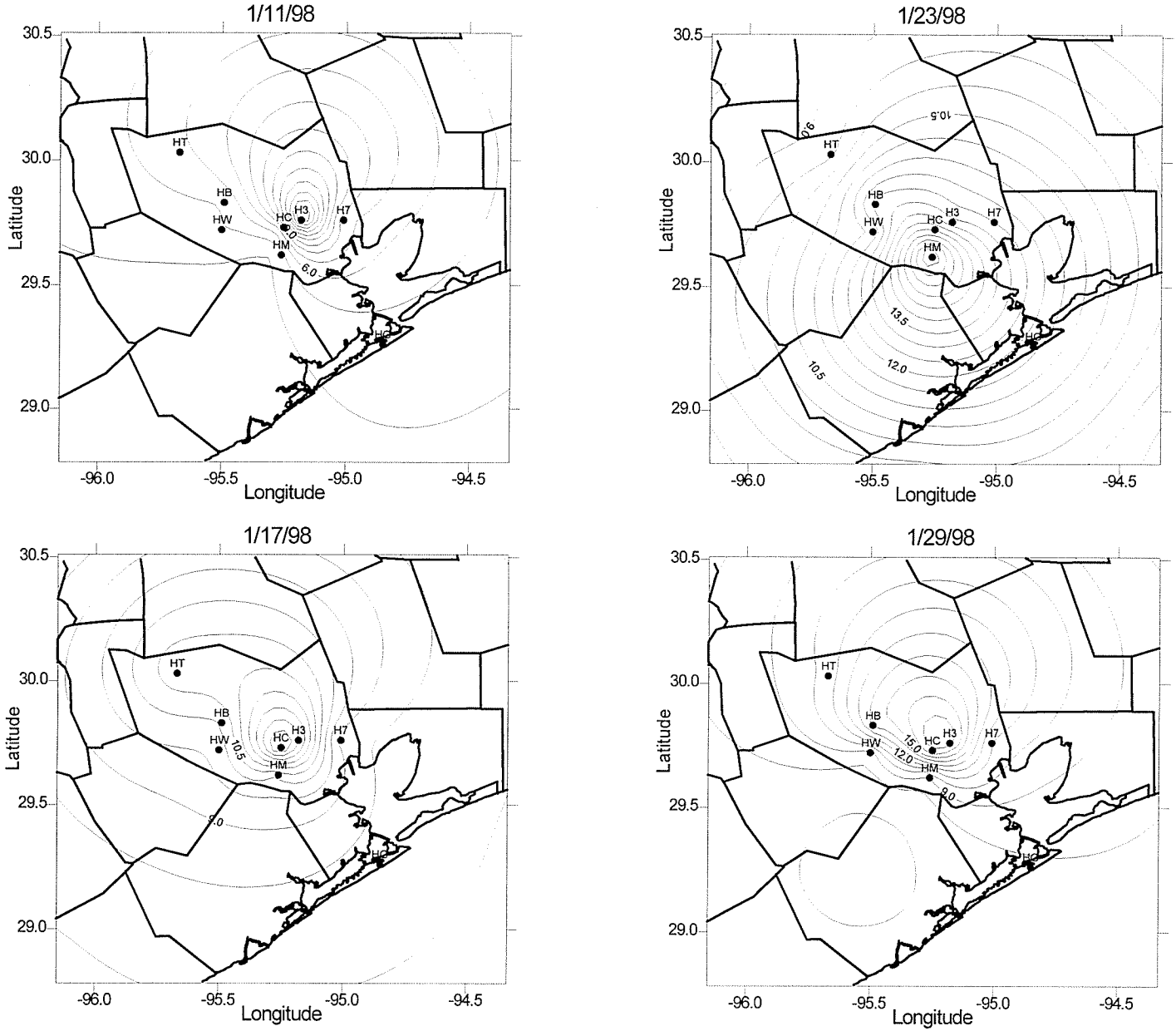

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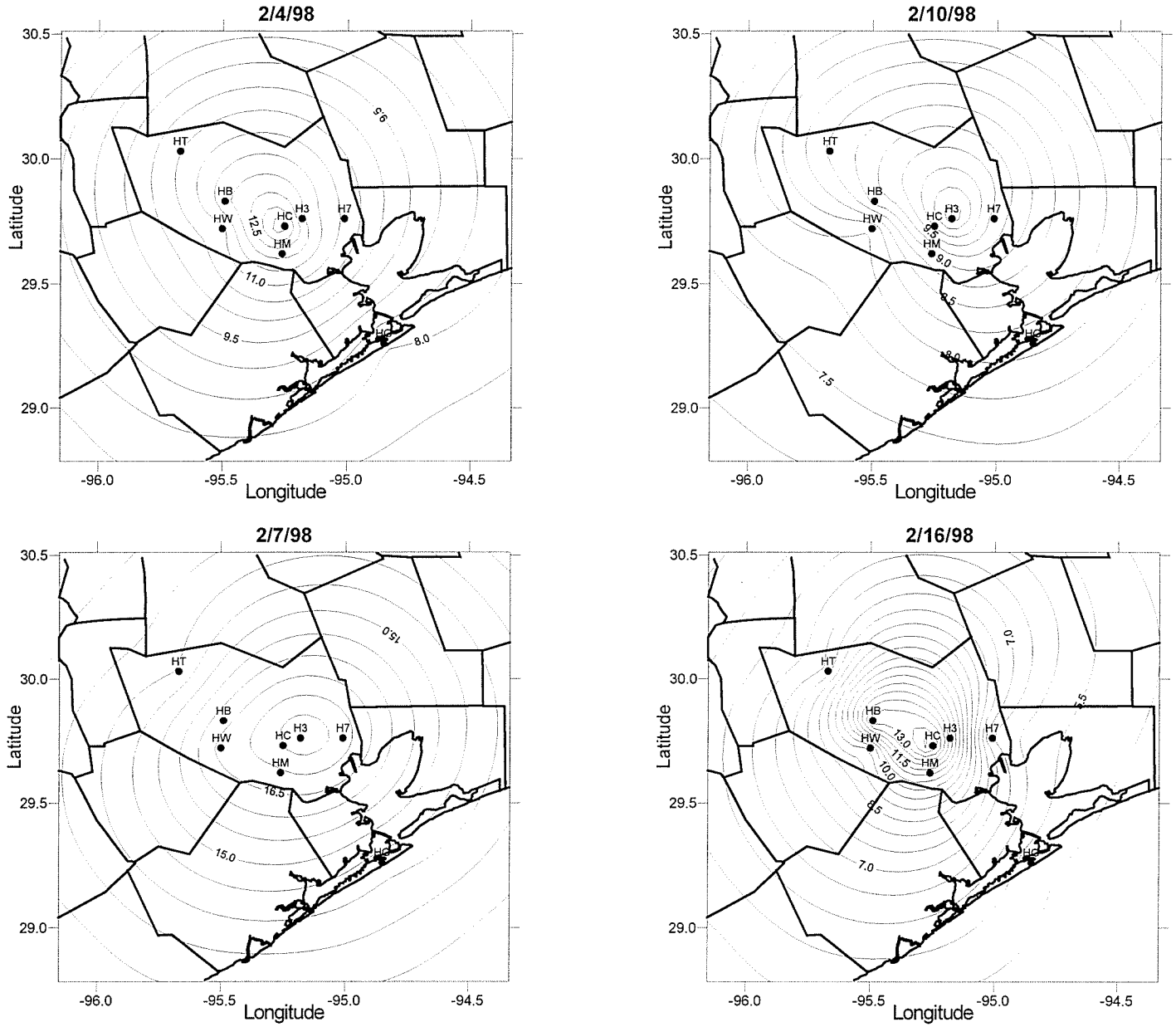

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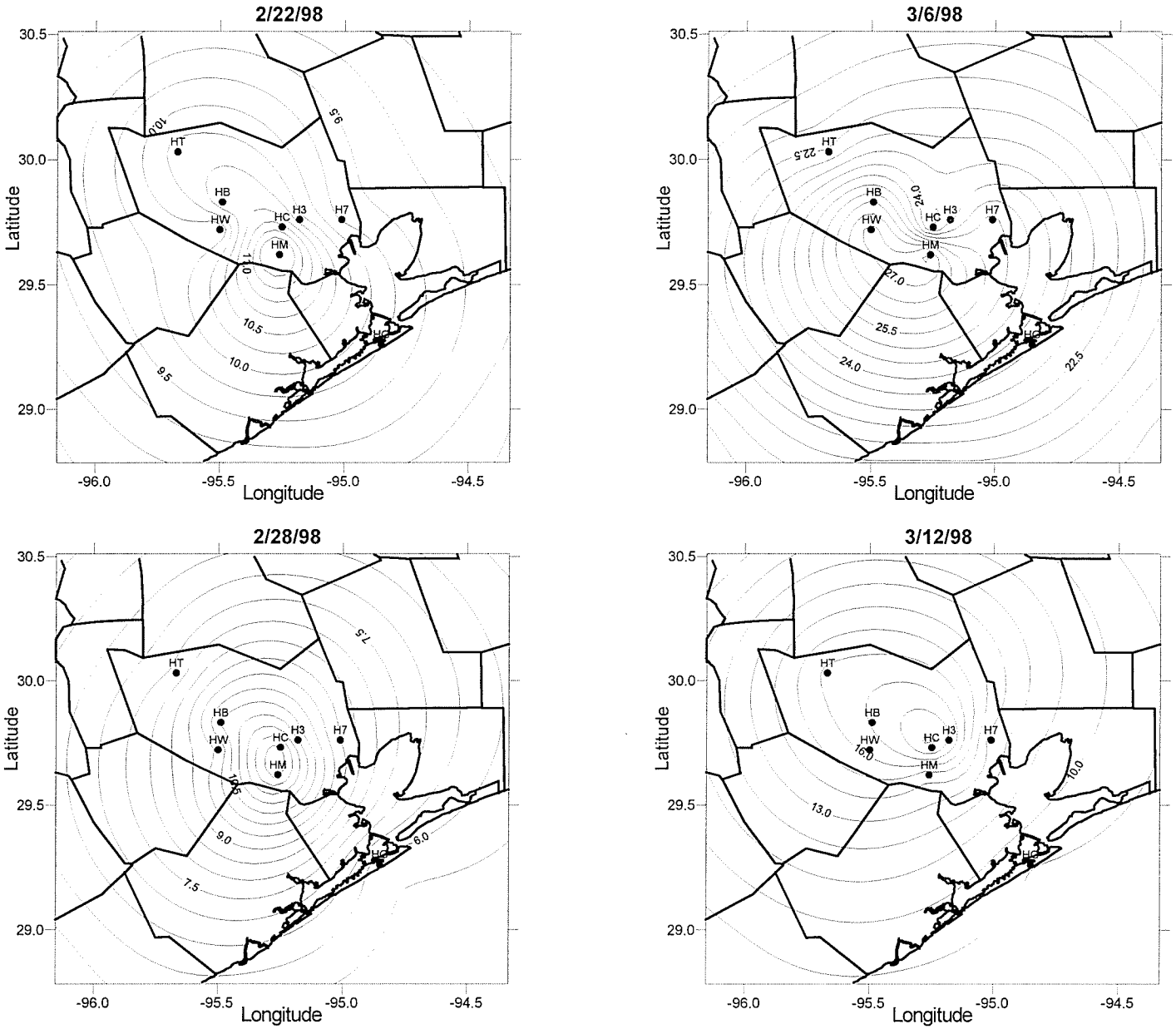

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