# A Study of Ambient Air Contaminants and Asthma in New York City 

Final Report 06-02
May 2006


Center for Environmental Health

## 全

The New York State Energy Research and Development Authority (NYSERDA) is a public benefit corporation created in 1975 by the New York State Legislature. NYSERDA's responsibilities include:

- Conducting a multifaceted energy and environmental research and development program to meet New York State's diverse economic needs.
- Administering the New York Energy \$mart ${ }^{\text {sM }}$ program, a Statewide public benefit R\&D, energy efficiency, and environmental protection program.
- Making energy more affordable for residential and low-income households.
- Helping industries, schools, hospitals, municipalities, not-for-profits, and the residential sector, including low-income residents, implement energy-efficiency measures.
- Providing objective, credible, and useful energy analysis and planning to guide decisions made by major energy stakeholders in the private and public sectors.
- Managing the Western New York Nuclear Service Center at West Valley, including: (1) overseeing the State's interests and share of costs at the West Valley Demonstration Project, a federal/State radioactive waste clean-up effort, and (2) managing wastes and maintaining facilities at the shut-down StateLicensed Disposal Area.
- Coordinating the State's activities on energy emergencies and nuclear regulatory matters, and monitoring low-level radioactive waste generation and management in the State.
- Financing energy-related projects, reducing costs for ratepayers.

NYSERDA administers the New York Energy \$mart ${ }^{\text {sM }}$ program, which is designed to support certain public benefit programs during the transition to a more competitive electricity market. Some 2,700 projects in 40 programs are funded by a charge on the electricity transmitted and distributed by the State's investor-owned utilities. The New York Energy $\$ m a r t^{\text {sM }}$ program provides energy efficiency services, including those directed at the low-income sector, research and development, and environmental protection activities.

NYSERDA derives its basic research revenues from an assessment on the intrastate sales of New York State's investor-owned electric and gas utilities, and voluntary annual contributions by the New York Power Authority and the Long Island Power Authority. Additional research dollars come from limited corporate funds. Some 400 NYSERDA research projects help the State's businesses and municipalities with their energy and environmental problems. Since 1990, NYSERDA has successfully developed and brought into use more than 170 innovative, energy-efficient, and environmentally beneficial products, processes, and services. These contributions to the State's economic growth and environmental protection are made at a cost of about $\$ .70$ per New York resident per year.

Federally funded, the Energy Efficiency Services program is working with more than 540 businesses, schools, and municipalities to identify existing technologies and equipment to reduce their energy costs.

For more information, contact the Communications unit, NYSERDA, 17 Columbia Circle, Albany, New York 12203-6399; toll-free 1-866-NYSERDA, locally (518) 862-1090, ext. 3250; or on the web at www.nyserda.org

State of New York
George E. Pataki
Governor

Energy Research and Development Authority
Vincent A. DeIorio, Esq., Chairman
Peter R. Smith, President and Chief Executive Officer

# A Study of Ambient Air Contaminants and Asthma in New York City <br> Final Report 

Prepared for the<br>New York State<br>Energy Research and<br>Development Authority

Albany, NY
www.nyserda.org
Ellen Burkhard
Project Manager
and

# The US Department of Health and Human Services Agency for Toxic Substances and Disease Registry <br> (Cooperative Agreement V50/ATV200002-11) 

Mohammed Uddin, MD, MPH
Project Manager

Prepared by

## New York State Department of Health Center for Environmental Health

Troy, NY

## EXECUTIVE SUMMARY

Many previous studies of acute asthma exacerbations and ambient air pollution have examined effects of only a few of the many contaminants that are found in urban air, making it difficult to determine which specific air pollutant or group of pollutants is most important in triggering hospital visits. In particular, ambient particulate matter is usually characterized based only on mass concentration, despite the knowledge that many particulate matter components such as acidity, metals or different carbon fractions might have different effects on asthma morbidity. In addition, whereas numerous studies have reported associations between daily air pollution concentrations and counts of hospital visits for asthma or other respiratory diseases, few studies have evaluated whether risks for air pollution-related hospital visits vary across communities that differ in their baseline health status. To investigate these issues, we conducted the study reported below. The study's primary goals were to assess whether ambient air quality differed in two New York City locations and to relate daily variation in the ambient concentrations of various air contaminants to daily variation in acute asthma exacerbations in both communities.

Mid-town Manhattan and the South Bronx are separated by less than 5 miles. However, the two regions of New York City differ greatly in levels of asthma morbidity. Although these differences are likely to be caused by multiple factors, including differential access to primary care for asthma, the present study was not designed to investigate these differences. Rather, we investigated whether day-to-day variations in air pollution were associated with asthma emergency department (ED) visits in each community and compared the magnitude of the air pollution effect between the two communities. To investigate this question, we analyzed daily counts of ED asthma visits to hospitals serving two distinct communities, one in Manhattan and the other in the South Bronx, and related those data to daily enhanced air monitoring data in each community.

We analyzed air quality and weather data collected over about a two year period, from January 1999 through November 2000, at two centrally located measurement stations sampling a broad range of contaminants (Figure 1). In addition to data on many commonly measured chemical air pollutants, information was collected on several components of airborne particulate matter that had not previously been assessed for their possible association with asthma exacerbations. Emergency department data on asthma visits for the corresponding dates were collected from the 22 hospitals throughout New York that served the communities surrounding the air monitoring stations. Data for hospital patients who lived in zip code areas within approximately 1.5 miles of either measurement site were extracted.

The study measured 24-hour average ambient air concentrations of acetone, aldehydes, chromium, iron, nickel, manganese, hydrogen ion, sulfate, pollen and mold spores. One-hour average concentrations were measured for ozone $\left(\mathrm{O}_{3}\right)$, sulfur dioxide $\left(\mathrm{SO}_{2}\right)$, nitrogen oxides $\left(\mathrm{NO}_{\mathrm{x}}\right)$, number of particles measuring 0.007


Figure 1. Air monitoring locations in Manhattan and Bronx (squares). Shaded zip code areas indicate communities where emergency department cases resided. Emergency department records were obtained from hospitals throughout New York City.
to 2.5 micrometers, particulate matter $\leq 2.5$ micrometers $\left(\mathrm{PM}_{2.5}\right)$ and particulate matter $\leq 10$ micrometers $\left(\mathrm{PM}_{10}\right)$. Three-hour average concentrations were measured for $\mathrm{PM}_{2.5}$ elemental and organic carbon. The hourly data were used for calculating daily averages, maximum concentrations and, for ozone, eight-hour moving averages. Meteorological data (temperature, wind speed and direction, humidity) were also collected. Ambient air data were collected from one site in Manhattan from January 1999 through November 2000, from one site in the Bronx from January 1999 through August 1999 and from a second nearby site in the Bronx from September 1999 through November 2000.

Table 1. Mean Concentrations of Air Pollutants and Bioaerosols Measured in Bronx and Manhattan. The values are summary statistics of all daily observations from January 1999 through November 2000, including days with missing values that were imputed by regression modeling for the time-series analysis of health data.

| Air Contaminant | Bronx | Manhattan |
| :--- | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}(\mathrm{ppm})^{*}$ | 0.027 | 0.021 |
| $\mathrm{NO}_{2}(\mathrm{ppm})^{*}$ | 0.031 | 0.036 |
| $\mathrm{SO}_{2}(\mathrm{ppm})^{*}$ | 0.011 | 0.012 |
| $\mathrm{PM}_{2.5}\left(\mu \mathrm{~g} / \mathrm{m}^{3}\right)^{*}$ | 14.5 | 16.6 |
| Max PM $_{2.5}\left(\mu \mathrm{~g} / \mathrm{m}^{3}\right)$ | 27.3 | 27.5 |
| Coarse $\mathrm{PM}\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)^{\dagger}$ | 7.69 | 7.10 |
| Sulfate $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)^{*}$ | 3.6 | 4.0 |
| $\mathrm{pH} *$ | 5.15 | 5.04 |
| Elemental Carbon $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | 1.19 | 1.32 |
| Organic Carbon $\left(\mu \mathrm{mg} / \mathrm{m}^{3}\right)$ | 3.17 | 3.09 |
| Total Metals $\left(\mathrm{ng} / \mathrm{m}^{3}\right)^{* *}$ | 101 | 94.0 |
| Total Aldehdes $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | 16.6 | 16.2 |
| Total Pollen $\left(\# / \mathrm{m}^{3}\right)^{\dagger \dagger}$ | 22.3 | 13.2 |
| Total Mold $\left(\# / \mathrm{m}^{3}\right)^{\dagger \dagger}$ | 448 | 490 |

* Mean levels significantly different ( $\mathrm{P}<0.05$, paired t-test) between the two communities over the entire study period.
** Nickel was significantly higher in Manhattan compared to Bronx over entire study period
${ }^{\dagger}$ Coarse PM ( $\left.=\mathrm{PM}_{10}-\mathrm{PM}_{2.5}\right)$ was not included in statistical comparisons of air quality in Bronx and Manhattan, but was included as a key pollutant variable in the asthma ED visit analysis
${ }^{\dagger}$ Bronx was significantly higher than Manhattan for two of three pollen sub-categories; Manhattan was significantly higher than Bronx for one of seven mold spore sub-categories.

Mean levels of $\mathrm{PM}_{2.5}, \mathrm{PM}_{2.5}$ acidity, $\mathrm{PM}_{2.5}$ sulfate, $\mathrm{PM}_{2.5}$ nickel, acid gases, ammonia, sulfur dioxide and nitrogen oxides were significantly higher in Manhattan than in the Bronx over the entire study period (Table 1). Mean levels of ozone, ragweed pollen and grass pollen were significantly higher in the Bronx. Statistical tests had power to detect small mean differences because of large sample sizes. Therefore, although several mean comparisons were significantly different, the absolute differences in analyte concentrations between the two sites were generally not large. For example, for most comparisons, the higher mean was no more than about 1.6 -fold larger than the lower mean, and many of the significant mean differences were less than 1.2-fold.

Exploratory temporal analyses of certain air contaminants were conducted. $\mathrm{PM}_{10}$ and $\mathrm{PM}_{2.5}$, organic carbon and elemental carbon were evaluated by the hour and day of week. Both sites exhibited a daily temporal pattern in $\mathrm{PM}_{10}$ and $\mathrm{PM}_{2.5}$ levels. Lowest levels were seen in the middle of the night (2 A.M.). The highest levels were seen in the morning, with a smaller peak in the early evening. Particulate matter elemental carbon concentrations peaked at 9 A.M. at both sites. The particulate organic carbon fraction increased modestly in concentration from early in the morning to a high in the evening for Manhattan, whereas the Bronx organic carbon levels remained nearly constant throughout the day. Acetone, elemental carbon, nitrogen oxides, $\mathrm{PM}_{10}$ and particulate Fe were the only variables showing a noticeable day-of-week trend, with somewhat lower daily means on Sundays, increasing through the week to Thursdays.

Table 2. Relative Risks* and 95\% Confidence Intervals for Asthma ED Visits as Function of 5-Day Mean Air Pollution and Bioaerosols from Single-Pollutant Models. Bold text indicates statistical significance at the 0.05 level.

| Air Contaminant | Bronx | Manhattan | Pollutant Concentration Increment** |
| :---: | :---: | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}$ | 1.06 (1.01, 1.10) | 1.06 (0.94, 1.19) | 0.024 |
| Max 8-hour $\mathrm{O}_{3}$ (warm season) | 1.08 (1.03, 1.12) | 1.04 (0.91, 1.19) | 0.024 |
| $\mathrm{NO}_{2}$ | 1.10 (1.01, 1.18) | 0.95 (0.72, 1.25) | 0.034 |
| $\mathrm{SO}_{2}$ | 1.11 (1.06, 1.17) | 0.99 (0.88, 1.12) | 0.011 |
| $\mathrm{PM}_{2.5}$ | 1.05 (1.01, 1.10) | 1.04 (0.94, 1.15) | 15.9 |
| Max PM 2.5 | 1.09 (1.03, 1.15) | 1.04 (0.91, 1.18) | 27.6 |
| Coarse PM | 1.02 (1.00, 1.04) | 1.02 (0.98, 1.07) | 7.4 |
| Sulfate | 1.03 (1.00, 1.06) | 1.05 (0.98, 1.13) | 3.9 |
| pH | $0.99(0.98,1.00)$ | 0.99 (0.95, 1.02) | 5.07 |
| Elemental Carbon | 1.04 (0.99, 1.09) | 1.06 (0.94, 1.19) | 1.25 |
| Organic Carbon | 1.05 (0.93, 1.17) | 1.20 (0.96, 1.49) | 3.14 |
| Total Metals | 1.02 (0.99, 1.05) | 1.02 (0.91, 1.15) | 93.5 |
| Total Aldehydes | $1.02(1.00,1.04)$ | 1.03 (0.96, 1.10) | 16.1 |
| Total Pollen | $1.00(1.00,1.00)^{\dagger}$ | 1.01 (1.00, 1.02) | 17.0 |
| Total Mold | 1.01 (0.99, 1.03) | 1.01 (0.97, 1.06) | 504 |

* A mean Relative Risk of 1.10 indicates that an increase in the daily pollutant concentration equal to the Pollutant Concentration Increment is associated, on average, with a $10 \%$ increase in daily asthma ED visits. ** Increment value used to calculate relative risks in Tables 2 and 3 were based on the mean pollutant level combining all data from both communities. Same units as in Table 1.
${ }^{\dagger}$ When RR and CI bounds appear equal, it is due to rounding.

The air monitoring study was not designed to attribute air contaminant variability to particular sources. However, air mass back-trajectory analysis was used to compare local and long-distance transport
contributions to total contaminant levels. ${ }^{1}$ On an annual average basis, 39-47 percent of measured sulfate concentrations was associated with long-distance transport from the west and southwest of New York. In comparison, long-distance transport from those directions contributed 26-32 percent of $\mathrm{PM}_{2.5}$ and 11 - 17 percent of sulfur dioxide. Nitrous acid (HONO) and ammonia levels appeared unrelated to long-distance air trajectories, suggesting that atmospheric transport did not contribute significantly to their concentrations.

Mean daily crude rates of asthma ED visits were over eight fold higher in the Bronx study area (16.9 per 100,000 persons) than in the Manhattan area ( 2.02 per 100,000 persons). Exploring reasons for these differences was beyond the scope of the present study. Among 14 key pollutants examined individually in regression analyses, five had statistically significant effects on asthma ED visits in the Bronx, including daily eight-hour maximum $\mathrm{O}_{3}$, mean daily $\mathrm{NO}_{2}, \mathrm{SO}_{2}, \mathrm{PM}_{2.5}$ and maximum one-hour $\mathrm{PM}_{2.5}$ (Table 2). No statistically significant pollution effects were observed in the Manhattan community.

In two-pollutant and three-pollutant regression models, $\mathrm{O}_{3}$ and $\mathrm{SO}_{2}$, and to a lesser extent maximum onehour $\mathrm{PM}_{2.5}$, were the most robust pollutants (Table 3). In other words, these pollutants exhibited less change in their effect estimates as additional pollutants were added to the models. It is of particular interest that we observed more robust health impacts of the daily maximum $\mathrm{PM}_{2.5}$ concentration than for the 24hour mean, suggesting that peak exposures may have larger health impacts.

In analyses restricted to the warm season (April through October), $\mathrm{O}_{3}$ effects in the Bronx were larger and more significant than in the full-year analysis, and they were approximately double those seen in Manhattan, suggesting greater susceptibility and/or exposure to this airway irritant and pro-inflammatory agent in the Bronx. Ozone effects in the Bronx also remained significant after removing daily maximum 8hour average concentrations that exceeded the ozone National Ambient Air Quality Standard (NAAQS) from the data set ( $<1 \%$ of all observations). Analyses by sex suggested that the air pollution effects in the Bronx were greater among females than males. No strong differences in effects were observed with age strata, though there was some indication of larger effects in older adults.

Our findings of significant air pollution effects in the Bronx, but not Manhattan, are likely to relate in part to greater statistical power for identifying effects in the Bronx where baseline ED visits were greater, but they may also reflect greater sensitivity to air pollution effects in the Bronx. For example, the mean effect estimates (expressed as relative risks) for the associations of average daily ozone with asthma ED visits were the same in the Bronx and Manhattan, although the Bronx relative risk was statistically significant,

[^0]Table 3. Relative Risks (95\% Confidence Intervals) for Asthma ED Visits as Function of 5-Day Mean Air Pollution from Two-Pollutant Models. Note: Pollutants included here were those that were significant predictors of ED visits in single-pollutant models. Exposure increments used to compute RRs were the same as in Table 2. Bold text indicates statistical significance at the 0.05 level.

| Contaminant | Controlled with | RR, Bronx | RR, Manhattan |
| :---: | :---: | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}$ | $\mathrm{PM}_{2.5}$ | 1.06 (1.01, 1.10) | 1.05 (0.93, 1.19) |
|  | Max $\mathrm{PM}_{2.5}$ | 1.04 (1.00, 1.09) | 1.05 (0.93, 1.19) |
|  | $\mathrm{NO}_{2}$ | 1.05 (1.01, 1.10) | 1.07 (0.94, 1.21) |
|  | $\mathrm{SO}_{2}$ | 1.05 (1.01, 1.10) | 1.06 (0.93, 1.20) |
| $\mathrm{PM}_{2.5}$ | Max 8-hour $\mathrm{O}_{3}$ | 1.05 (1.01, 1.10) | 1.03 (0.94, 1.14) |
|  | Max $\mathrm{PM}_{2.5}$ | 0.99 (0.92, 1.06) | 1.04 (0.89, 1.23) |
|  | $\mathrm{NO}_{2}$ | 1.03 (0.98, 1.09) | 1.08 (0.95, 1.23) |
|  | $\mathrm{SO}_{2}$ | 1.01 (0.96, 1.06) | 1.05 (0.94, 1.17) |
| Max $\mathrm{PM}_{2.5}$ | Max 8-hour $\mathrm{O}_{3}$ | 1.07 (1.02, 1.13) | 1.02 (0.89, 1.17) |
|  | $\mathrm{PM}_{2.5}$ | 1.09 (1.00, 1.20) | 0.99 (0.79, 1.23) |
|  | $\mathrm{NO}_{2}$ | 1.07 (1.01, 1.14) | 1.10 (0.92, 1.31) |
|  | $\mathrm{SO}_{2}$ | 1.05 (0.99, 1.11) | 1.05 (0.90, 1.21) |
| $\mathrm{NO}_{2}$ | Max 8-hour $\mathrm{O}_{3}$ | 1.08 (1.00, 1.17) | 0.91 (0.68, 1.21) |
|  | $\mathrm{PM}_{2.5}$ | 1.06 (0.97, 1.16) | 0.83 (0.59, 1.17) |
|  | Max $\mathrm{PM}_{2.5}$ | 1.04 (0.96, 1.14) | 0.84 (0.59, 1.20) |
|  | $\mathrm{SO}_{2}$ | 1.02 (0.94, 1.12) | 0.95 (0.69, 1.30) |
| $\mathrm{SO}_{2}$ | Max 8-hour $\mathrm{O}_{3}$ | 1.11(1.05, 1.17) | 0.99 (0.88, 1.12) |
|  | $\mathrm{PM}_{2.5}$ | 1.11 (1.04, 1.18) | 0.97 (0.85, 1.11) |
|  | Max $\mathrm{PM}_{2.5}$ | 1.09 (1.03, 1.16) | 0.98 (0.85, 1.12) |
|  | $\mathrm{NO}_{2}$ | 1.11 (1.04, 1.17) | 1.01 (0.87, 1.16) |

while the Manhattan estimate was not. In contrast, Bronx relative risks for average daily $\mathrm{NO}_{2}$ and $\mathrm{SO}_{2}$ and maximum hourly $\mathrm{PM}_{2.5}$ were statistically significant in the Bronx and were also substantially larger than the corresponding Manhattan effect estimates.

To evaluate the specificity of the air pollution effects observed for asthma visits, we analyzed the relationships between air pollutants and ED visits for outcomes thought a priori to be unrelated to air pollution (e.g., urinary tract infections, acute gastroenteritis). Of the five pollutants that had significant univariate effects on asthma in the Bronx, one, 24-hour $\mathrm{PM}_{2.5}$, had significant effects on the control outcome. Positive but non-significant effects were seen for the remaining pollutants, except ozone. There was no evidence of any effect of ozone on control ED counts. These results could suggest some degree of overestimating risk in the analysis.

The observed associations between specific pollutants and asthma ED visits do not necessarily indicate cause and effect. It is possible that unmeasured confounders related to indoor environmental exposures or socio-economic status variables might be contributing to variability in acute asthma exacerbations. However, within each study area, the time-series design at least partially controls for unmeasured
confounders because each case acts essentially as its own control. The analysis detects marginal changes in the outcome variable relative to the baseline rate that are associated with the measured exposure variables, and the baseline rate would include effects due to unmeasured variables, such as local or indoor exposures.

Estimating exposure based on centrally located ambient monitors also adds some uncertainty to the results reported here due to potential exposure misclassification compared to actual personal exposure. However, the relatively high population density of the Bronx and Manhattan allowed for the central monitors to be used as an indicator for exposure for a relatively small area (i.e., the population residing within approximately 1.5 miles of the monitoring site). Furthermore, the correlation between the two monitoring sites was relatively high (i.e., greater than 0.6 ) and mean levels were very similar for most analytes, perhaps partially mitigating against exposure misclassification biases that might occur because of movement of residents throughout the greater New York City area.

## CONCLUSIONS AND RECOMMENDATIONS

Mean levels of most air contaminants did not differ substantially between the two New York City monitoring sites over the course of the study. When differences were observed, mean levels in Manhattan tended to be modestly higher than mean levels in the Bronx for most pollutants. Mean ozone and pollen levels were somewhat higher in the Bronx.

The health analysis results suggest that the criteria pollutants $\mathrm{PM}_{2.5}, \mathrm{SO}_{2}, \mathrm{O}_{3}$ and $\mathrm{NO}_{2}$ had a statistically detectable impact on acute asthma ED visits in a community with a relatively high baseline rate of acute asthma exacerbations. In two-pollutant and three-pollutant regression models, $\mathrm{O}_{3}$ and $\mathrm{SO}_{2}$, and to a lesser extent maximum one-hour $\mathrm{PM}_{2.5}$, were the most robust pollutants. Robust effects of $\mathrm{O}_{3}$ have been seen in previous ED asthma studies and in hospital admissions studies of asthma and other respiratory diseases. It is of particular interest that we observed more robust health impacts of daily maximum $\mathrm{PM}_{2.5}$ concentration than of the 24-hour mean, suggesting that peak exposures may have larger health impacts.

The following recommendations are suggested based on the study results:

1. EPA should consider the findings in this study and others identifying respiratory health effects associated with $\mathrm{SO}_{2}$ concentrations below current standards during their review of the $\mathrm{SO}_{2}$ NAAQS. The results of this study were submitted in response to a Call for Information issued by EPA in May, 2006 to initiate review of the $\mathrm{SO}_{2}$ NAAQS.
2. Future time-series studies examining associations between ambient air pollutants and health outcomes would benefit from direct evaluation of the relationship between personal exposure and regional monitoring data.
3. More research should be conducted to try to determine if peak, short-term (e.g. hourly) elevated concentrations of $\mathrm{PM}_{2.5}$ are more strongly associated than daily average concentrations with asthma and other health endpoints. If the science is sufficiently strong, consideration should be given to the effects of short-term $\mathrm{PM}_{2.5}$ excursions in future reviews of the particulate matter NAAQS.
4. The high correlations between pollutants (including components of $\mathrm{PM}_{2.5}$ ) make it difficult in these epidemiologic studies to confidently identify critical compounds. Alternative strategies to address this question should be considered in the future.
5. Further evaluation of the statistical methods employed in time-series epidemiological studies is warranted, based on the suggestion of possible model bias indicated by our analysis of control outcomes.
6. To the extent that targeted community based asthma interventions are planned with respect to air pollution messages, higher priority should be given to communities with larger asthma burdens.

Part A:
A Comparison of Ambient Air Quality
in the Bronx and Manhattan

## CONTENTS

LIST OF TABLES ..... iv
LIST OF FIGURES ..... v
SUMMARY ..... 9
INTRODUCTION ..... 13
OBJECTIVES ..... 15
BACKGROUND ..... 17
METHODS ..... 21
RESULTS ..... 29
DISCUSSION ..... 37
CONCLUSIONS AND RECOMMENDATIONS ..... 43
REFERENCES ..... 45
AUTHORS AND ACKNOWLEDGMENTS ..... 49
TABLES ..... 51
FIGURES ..... 63
APPENDICES ..... 135

## LIST OF TABLES

Table 1. U.S. Census and NYS motor vehicle registration data ..... 52
Table 2. Business data ..... 53
Table 3. Average particulate matter concentrations ..... 54
Table 4. Average particle count ..... 54
Table 5. Average pH , sulfate, elemental carbon and organic carbon concentrations ..... 54
Table 6. Average particulate metals concentrations ..... 55
Table 7. Average pollen concentrations ..... 55
Table 8. Average mold concentrations ..... 55
Table 9. Average acetone and aldehyde concentrations ..... 56
Table 10. Average acidic and basic gas concentrations ..... 56
Table 11. Average ozone, sulfur dioxide and nitrogen oxide concentrations ..... 56
Table 12. Comparison of average particulate matter concentrations between two Bronx sampling locations ..... 57
Table 13. Comparison of average pH , sulfate, elemental carbon and organic carbon concentrations between two Bronx sampling locations ..... 57
Table 14. Comparison of average particulate metals concentrations between two Bronx sampling locations ..... 57
Table 15. Comparison of average pollen and mold concentrations between two Bronx sampling locations ..... 58
Table 16. Comparison of average acetone and aldehyde concentrations between two Bronx sampling locations ..... 58
Table 17. Comparison of average acidic and basic gas concentrations between two Bronx sampling locations ..... 59
Table 18. Comparison of average ozone, sulfur dioxide and nitrogen oxide concentrations between two Bronx sampling locations ..... 59
Table 19. Daily maximum concentrations for analytes measured as one-hour or three-hour time-weighted averages ..... 60
Table 20. Pearson correlation coefficients for corresponding observations at the Bronx and Manhattan sampling locations ..... 61
Table 21. Pearson correlation coefficients within sampling location for corresponding daily average and daily maximum observations ..... 61
Table 22. Pearson correlation coefficients for corresponding observations at the Bronx and Manhattan sampling locations, stratified by year ..... 62

## PREFACE

The New York State Energy Research and Development Authority is pleased to publish "A Study of Ambient Air Contaminants and Asthma in New York City, Parts A and B." The report was prepared by the principal investigator, Daniel Luttinger of the New York State Department of Health, Center for Environmental Health.

This study was conducted in the Bronx and Manhattan, two boroughs in New York City. Previous studies of acute asthma exacerbations and ambient air pollution have examined effects of only a few of the many contaminants that are found in urban air. This study was supported to improve the understanding of exposure to a large variety of pollutants (ozone, sulfur dioxide, nitrogen oxides, aldehydes, nitrous acid, nitric acid, particulate metals, organic particulate, sulfate, nitrate) in ambient air and their effects on respiratory health.

The work was funded by the New York Energy Smart ${ }^{\text {SM }}$ Environmental Monitoring, Evaluation, and Protection (EMEP) Program. This study is one of a broader portfolio of research projects characterizing particulate matter (PM), performing source apportionment on PM datasets, and addressing policy-relevant questions for PM control strategies in New York State.

## NOTICE

This report was prepared by the New York State Department of Health’s Center for Environmental Health, the New York State Department of Environmental Conservation and Columbia University in the course of performing work contracted for and sponsored by the New York State Energy Research and Development Authority and the U.S. Agency for Toxic Substances and Disease Registry (hereafter "the Sponsors"). The opinions expressed in this report do not necessarily reflect those of the Sponsors or the State of New York, and reference to any specific product, service, process, or method does not constitute an implied or expressed recommendation or endorsement of it. Further, the Sponsors and the State of New York make no warranties or representations, expressed or implied, as to the fitness for particular purpose or merchantability of any product, apparatus, or service, or the usefulness, completeness, or accuracy of any processes, methods, or other information contained, described, disclosed, or referred to in this report. The Sponsors, the State of New York, and the contractors make no representation that the use of any product, apparatus, process, method, or other information will not infringe privately owned rights and will assume no liability for any loss, injury, or damage resulting from, or occurring in connection with, the use of information contained, described, disclosed, or referred to in this report.

## LIST OF TABLES

Table 1. U.S. Census and NYS motor vehicle registration data ..... 52
Table 2. Business data ..... 53
Table 3. Average particulate matter concentrations ..... 54
Table 4. Average particle count ..... 54
Table 5. Average pH , sulfate, elemental carbon and organic carbon concentrations ..... 54
Table 6. Average particulate metals concentrations ..... 55
Table 7. Average pollen concentrations ..... 55
Table 8. Average mold concentrations ..... 55
Table 9. Average acetone and aldehyde concentrations ..... 56
Table 10. Average acidic and basic gas concentrations ..... 56
Table 11. Average ozone, sulfur dioxide and nitrogen oxide concentrations ..... 56
Table 12. Comparison of average particulate matter concentrations between two Bronx sampling locations ..... 57
Table 13. Comparison of average pH , sulfate, elemental carbon and organic carbon concentrations between two Bronx sampling locations ..... 57
Table 14. Comparison of average particulate metals concentrations between two Bronx sampling locations ..... 57
Table 15. Comparison of average pollen and mold concentrations between two Bronx sampling locations ..... 58
Table 16. Comparison of average acetone and aldehyde concentrations between two Bronx sampling locations ..... 58
Table 17. Comparison of average acidic and basic gas concentrations between two Bronx sampling locations ..... 59
Table 18. Comparison of average ozone, sulfur dioxide and nitrogen oxide concentrations between two Bronx sampling locations ..... 59
Table 19. Daily maximum concentrations for analytes measured as one-hour or three-hour time-weighted averages ..... 60
Table 20. Pearson correlation coefficients for corresponding observations at the Bronx and Manhattan sampling locations ..... 61
Table 21. Pearson correlation coefficients within sampling location for corresponding daily average and daily maximum observations ..... 61
Table 22. Pearson correlation coefficients for corresponding observations at the Bronx and Manhattan sampling locations, stratified by year ..... 62

## LIST OF FIGURES

Figure 1. Bronx sampling locations ..... 64
Figure 2. Manhattan sampling location ..... 65
Figure 3. Daily averages and daily average differences between Bronx and Manhattan:
$\mathrm{PM}_{2.5}$ (TEOM) ..... 66
Figure 4. Daily averages and daily average differences between Bronx and Manhattan:
$\mathrm{PM}_{10}$ (TEOM) ..... 67
Figure 5. Daily averages and daily average differences between Bronx and Manhattan: particle count ..... 68
Figure 6. Daily averages and daily average differences between Bronx and Manhattan:
pH ..... 69
Figure 7. Daily averages and daily average differences between Bronx and Manhattan: sulfate ..... 70
Figure 8. Daily averages and daily average differences between Bronx and Manhattan: organic carbon ..... 71
Figure 9. Daily averages and daily average differences between Bronx and Manhattan: elemental carbon ..... 72
Figure 10. Daily averages and daily average differences between Bronx and Manhattan: iron ..... 73
Figure 11. Daily averages and daily average differences between Bronx and Manhattan: nickel ..... 74
Figure 12. Daily averages and daily average differences between Bronx and Manhattan: total pollen ..... 75
Figure 13. Daily averages and daily average differences between Bronx and Manhattan: tree pollen ..... 76
Figure 14. Daily averages and daily average differences between Bronx and Manhattan:
grass pollen ..... 77
Figure 15. Daily averages and daily average differences between Bronx and Manhattan: ragweed pollen ..... 78
Figure 16. Daily averages and daily average differences between Bronx and Manhattan: total mold ..... 79
Figure 17. Daily averages and daily average differences between Bronx and Manhattan: basidiospores ..... 80
Figure 18. Daily averages and daily average differences between Bronx and Manhattan: ascospores ..... 81

Figure 19. Daily averages and daily average differences between Bronx and Manhattan:
mitospores .......................................................................................................................................... 82
Figure 20. Daily averages and daily average differences between Bronx and Manhattan: dark mitospores ..... 83
Figure 21. Daily averages and daily average differences between Bronx and Manhattan: non-dark mitospores ..... 84
Figure 22. Daily averages and daily average differences between Bronx and Manhattan: mold spores $<10 \mu \mathrm{~m}$ ..... 85
Figure 23. Daily averages and daily average differences between Bronx and Manhattan: mold spores > $10 \mu \mathrm{~m}$ ..... 86
Figure 24. Daily averages and daily average differences between Bronx and Manhattan: acetone ..... 87
Figure 25. Daily averages and daily average differences between Bronx and Manhattan: acetaldehyde. ..... 88
Figure 26. Daily averages and daily average differences between Bronx and Manhattan: formaldehyde ..... 89
Figure 27. Daily averages and daily average differences between Bronx and Manhattan: hydrochloric acid. ..... 90
Figure 28. Daily averages and daily average differences between Bronx and Manhattan: nitrous acid ..... 91
Figure 29. Daily averages and daily average differences between Bronx and Manhattan: nitric acid ..... 92
Figure 30. Daily averages and daily average differences between Bronx and Manhattan: ammonia ..... 93
Figure 31. Daily averages and daily average differences between Bronx and Manhattan: ozone ..... 94
Figure 32. Daily averages and daily average differences between Bronx and Manhattan: sulfur dioxide ..... 95
Figure 33. Daily averages and daily average differences between Bronx and Manhattan: nitrogen dioxide ..... 96
Figure 34. Daily averages and daily average differences between Bronx and Manhattan: nitrogen oxide ..... 97
Figure 35. Daily averages and daily average differences between Bronx and Manhattan: total nitrogen oxides ..... 98
Figure 36. Multidimensional scaling results: all seasons combined ..... 99
Figure 37. Multidimensional scaling results: January-March ..... 100
Figure 38. Multidimensional scaling results: April-June ..... 101

Figure 39. Multidimensional scaling results: July-September .................................................................. 102
Figure 40. Multidimensional scaling results: October-December............................................................. 103
Figure 41. Hierarchical clustering results: all seasons combined ............................................................. 104
Figure 42. Hierarchical clustering results: January-March ...................................................................... 105
Figure 43. Hierarchical clustering results: April-June .............................................................................. 106
Figure 44. Hierarchical clustering results: July-September ..................................................................... 107
Figure 45. Hierarchical clustering results: October-December................................................................. 108
Figure 46. Day of week averages: $\mathrm{PM}_{2.5}$ and $\mathrm{PM}_{10}$ (TEOM) ................................................................... 109
Figure 47. Hour of day averages: $\mathrm{PM}_{2.5}$ and $\mathrm{PM}_{10}$ (TEOM) .................................................................... 110
Figure 48. Day of week averages: particle count...................................................................................... 111
Figure 49. Hour of day averages: particle count........................................................................................ 112
Figure 50. Day of week averages: pH ....................................................................................................... 113
Figure 51. Day of week averages: sulfate.................................................................................................. 114
Figure 52. Day of week averages: organic carbon and elemental carbon................................................. 115
Figure 53. Hour of day averages: organic carbon and elemental carbon.................................................. 116
Figure 54. Day of week averages: particulate metals ................................................................................ 117
Figure 55. Day of week averages: total pollen .......................................................................................... 118
Figure 56. Day of week averages: tree, grass and ragweed pollen ........................................................... 119
Figure 57. Day of week averages: total mold ............................................................................................. 120
Figure 58. Day of week averages: ascospores, basidiospores and mitospores ......................................... 121
Figure 59. Day of week averages: dark and non-dark mitospores............................................................ 122
Figure 60. Day of week averages: small and large mold spores............................................................... 123
Figure 61. Day of week averages: acetone, acetaldehyde and formaldehyde........................................... 124
Figure 62. Day of week averages: hydrochloric acid ................................................................................ 125
Figure 63. Day of week averages: nitrous acid and nitric acid................................................................. 126
Figure 64. Day of week averages: ammonia ............................................................................................ 127
Figure 65. Day of week averages: ozone.................................................................................................... 128
Figure 66. Hour of day averages: ozone..................................................................................................... 129
Figure 67. Day of week averages: sulfur dioxide ...................................................................................... 130
Figure 68. Hour of day averages: sulfur dioxide ....................................................................................... 131
Figure 69. Day of week averages: nitric oxide, nitrogen dioxide and total nitrogen oxides..................... 132
Figure 70. Hour of day averages: nitric oxide, nitrogen dioxide and total nitrogen oxides....................... 133

## SUMMARY

This report compares ambient levels of certain hazardous air pollutants, criteria pollutants, and bioaerosols in two New York City neighborhoods that have different rates of hospital admissions for asthma and different socio-economic status characteristics. Chemical and biological analytes were chosen for this study based on existing information suggesting that exposure to these analytes may be related to acute asthma exacerbations. In addition to data on many commonly measured chemical air pollutants, information was collected on several components of airborne particulate matter that have not previously been assessed for their possible association with asthma exacerbations. The primary goal was to assess whether ambient air quality differed in two New York City locations. A separate report presents the results of the analysis evaluating the effects of various air contaminants on acute asthma exacerbations.

The study measured 24-hour average ambient air concentrations of acetone, aldehydes, chromium, iron, nickel, manganese, hydrogen ion, sulfate, pollen and mold spores. One-hour average concentrations were measured for ozone, sulfur dioxide, nitrogen oxides, number of particles measuring 0.007 to 2.5 micrometers, particulate matter $\leq 2.5$ micrometers $\left(\mathrm{PM}_{2.5}\right)$ and particulate matter $\leq 10$ micrometers $\left(\mathrm{PM}_{10}\right)$. Three-hour average concentrations were measured for elemental and organic carbon. The hourly data were used for calculating daily averages, maximum concentrations and for ozone, eight-hour moving averages. Meteorological data (temperature, wind speed and direction, humidity) were also collected. Ambient air data were collected from one site in Manhattan from January 1999 through November 2000, from one site in the Bronx from January 1999 through August 1999 and from a second nearby site in the Bronx from September 1999 through November 2000.

Statistical analyses comparing ambient air concentrations between the Bronx and Manhattan sites were conducted using a paired t-test adjusted for autocorrelation. Comparisons were made on a seasonal basis (quarterly) and for the entire study period. Mean levels of fine particulate matter, particulate acidity, particulate sulfate, particulate nickel, acid gases, ammonia, sulfur dioxide and nitrogen oxides were significantly higher in Manhattan than in the Bronx over the entire study period. Mean levels of ozone, ragweed pollen and grass pollen were significantly higher in the Bronx. Statistical tests had power to detect small mean differences because of large sample sizes. Therefore, although several mean comparisons were significantly different, the absolute differences in analyte concentrations between the two sites were generally not large. For example, for most comparisons, the higher mean was no more than about 1.6 -fold larger than the lower mean, and many of the significant mean differences were less than 1.2-fold.

Most of the variables were correlated (Pearson $r>0.6$ ) over the entire study period between the Manhattan and Bronx sites. In general, low correlations were due to a few outliers. Weak correlations between the two sites were found for particle count, iron, nickel, acetone and non-dark mitospores.

Exploratory temporal analyses of certain air contaminants were conducted. $\mathrm{PM}_{10}$ and $\mathrm{PM}_{2.5}$, organic carbon and elemental carbon were evaluated by the hour and day of week. Both sites exhibited a daily temporal pattern in $\mathrm{PM}_{10}$ and $\mathrm{PM}_{2.5}$ levels. Lowest levels were seen in the middle of the night (2 A.m.). The highest levels were seen in the morning, with a smaller peak in the early evening. Particulate matter elemental carbon concentrations peaked at 9 A.M. at both sites. The particulate organic carbon fraction increased modestly in concentration from early in the morning to a high in the evening for Manhattan, whereas the Bronx organic carbon levels remained nearly constant throughout the day. Acetone, elemental carbon, nitrogen oxides, $\mathrm{PM}_{10}$ and particulate Fe were the only variables showing a noticeable day-of-week trend, with somewhat lower daily means on Sundays, increasing through the week to Thursdays.

Two multivariate statistical procedures (multidimensional scaling and hierarchical cluster analysis) were used in exploratory analyses of associations among chemical analytes within each sampling site. Very robust patterns of clustering among variables were not observed in these analyses, but some modest associations were found. Ozone tended to be negatively associated with all other analytes, particularly during the coldweather months. The strongest positive associations tended to be between or among variables measuring closely related chemical species. That is, all nitrogen oxide variables tended to cluster together, two different measures of sulfur dioxide were closely associated and particulate matter variables tended to be closely associated with each other.

Larger clusters of analytes varied somewhat by season, but in general, nitrogen oxides, sulfur dioxide, elemental carbon and some metals tended to form a relatively consistent aggregation of variables. A second aggregation usually included the particulate matter variables, some aldehydes, organic carbon, sulfate and in some instances inorganic acid measures. Patterns of associations among analytes did not differ noticeably between the Bronx and Manhattan.

## CONCLUSIONS AND RECOMMENDATIONS

Ambient air quality measured with rooftop monitors at two locations in New York City found that, for most analytes, either the two sites did not differ or mean air levels were higher at the Manhattan location than at the Bronx location. Analyte measurements from both locations were subject to large temporal variations on hourly, daily, and often seasonal time scales. When statistically different average pollutant levels were detected between the two locations, they differed by less than twofold. Average ozone and pollen levels tended to be higher in the Bronx, with mean differences of about $30 \%$ to $70 \%$ between the two sites. These results, representing approximately two years of hourly or daily observations on nearly three dozen analytes
from two locations in New York City, provide a more detailed characterization of ambient air pollutants, especially particulate matter constituents, than has been previously reported for a large urban area. We recommend that future studies investigating ambient air pollutant exposures on an urban neighborhood scale collect additional data to better characterize spatial variability of ambient pollutants in urban areas, particularly for noncriteria pollutants.

## Section 1 <br> INTRODUCTION

Asthma is a serious chronic disease that in 1999 affected roughly four percent of the U.S. population (approximately 11 million total cases of diagnosed asthma with an acute asthma episode in the previous 12 months). Its prevalence has been increasing over the past few decades (Mannino et al. 1998, 2002; IOM 2000). Lifetime prevalence (i.e., ever-diagnosed asthma) in the United States was approximately $10 \%$ in the 1997-1999 National Health Interview Survey, which is consistent with the adult lifetime prevalence estimated in the 2000 Behavioral Risk Factor Surveillance System data (CDC 2006). Asthma disproportionately affects African American communities, with higher rates of asthma emergency department visits, asthma hospitalizations and asthma mortality (Mannino et al. 2002).

The New York State Department of Health (NYSDOH) received many letters from students, teachers, community groups and environmental organizations requesting an environmental health investigation in the South Bronx. The South Bronx is a densely populated, inner-city area with high traffic volume, multifamily residential developments and a variety of industrial operations. The Bronx is the site of a city water pollution control plant, a sludge pelletization plant that handles over $70 \%$ of the city's sewage sludge, a large wholesale food market and distribution center and many small industries. Bronx residents and elected officials raised concerns that high asthma rates in the borough were related to ambient air pollution exposures from these sources.

As part of the response to these concerns, NYSDOH undertook to compare the air quality in the South Bronx with that of another area in New York City, and to evaluate potential associations between measured air pollutants and emergency department visits for asthma. The study involved continuous ambient air monitoring in the South Bronx and Manhattan for criteria air pollutants, pollutants categorized by the U.S. Environmental Protection Agency (EPA) as hazardous air pollutants (HAPs) and bioaerosols, including pollen and fungal spores. The chemical and biological analytes chosen for the study were selected based on existing information suggesting that exposure to these ambient air pollutants may be associated with acute asthma exacerbations. In addition to mass concentration, ambient particulate matter was chemically characterized in terms of elemental and organic carbon fractions, acidity and metals content. The study utilized centralized monitoring stations that were expected to be representative of air quality in the two communities. Attribution of measured pollutant concentrations to specific point sources was not a goal of the study, and this was not technically feasible with the type of data collected.

A comparison of the ambient air monitoring results from the South Bronx and Manhattan monitoring sites is reported here. A separate study component investigated associations between ambient air monitoring results and asthma emergency-department visits in the two areas. Those results are presented in Part B of this report.

## Section 2

## OBJECTIVES

The purpose of the study was to evaluate and compare ambient concentrations of several air pollutants in two areas of New York City and to evaluate temporal associations between these air pollutants and acute asthmatic symptoms as measured by emergency department visits for asthma by residents in parts of the Bronx and Manhattan. Ultimately, this study should contribute to the body of knowledge about the effects of components of ambient air on asthma in urban areas.

Specific objectives were as follows:

1. to evaluate whether ambient levels of certain hazardous air pollutants, criteria pollutants or bioaerosols differ in two New York City neighborhoods that have different rates of hospital admissions for asthma and different socio-economic status characteristics;
2. to compute the overall rates of air-contaminant-attributable asthma emergency department visits among residents of the two communities over a one-year period, and test whether the magnitude of the air pollution effect differs in the two communities; and
3. to investigate which air contaminants are most associated with acute asthma exacerbations in each community.

This report focuses on the first objective-evaluating whether ambient levels of certain hazardous air pollutants, criteria pollutants or bioaerosols differ in two New York City neighborhoods. More specifically, this report compares air concentrations on a seasonal basis between sites and describes the correlation between the sites for the air contaminants, the correlations among contaminants within each site, and temporal contaminant patterns.

## Section 3 <br> BACKGROUND

Asthma is a multi-factorial disease with a complicated and still not completely understood etiology and physiological basis. Genetic factors and environmental exposures are both thought to play a role in asthma development. However, it has been argued that the recent increase in asthma prevalence has occurred too rapidly to be the result of genetic changes and is therefore assumed to be largely due to changes in environmental exposures (e.g., Ronchetti et al. 2001). Laboratory studies and studies looking at human populations that have found associations between air quality and different asthma outcomes suggest that ambient air exposures may be one important factor influencing asthma morbidity.

Ambient air contaminants, including ozone, sulfur dioxide, nitrogen dioxide, acid particulates (hydrogen ion), sulfates, $\mathrm{PM}_{2.5}$ and $\mathrm{PM}_{10}$, total particulates, wood smoke and bioaerosols (pollen and fungal spores), have all been associated with increased asthma symptoms (Boman et al. 2003; Brunekreef and Holgate 2002; Burnett et al. 1994; Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society 1996; Dales et al. 2000; Delfino et al. 1996; Gavitt and Koren 2001; Peden 2002; Schwela 2000). Evaluating these associations for individual contaminants, however, is complicated by the temporal correlations among air contaminants and weather factors. A detailed review of the epidemiological literature on the relationship between ambient air pollution and asthma morbidity is beyond the scope of this report. However, brief examples of associations between ambient air contaminant exposures and asthma morbidity are discussed below.

## PARTICULATE MATTER AND OTHER AEROSOLS

Many epidemiological studies have suggested that increases in particulate air contaminant levels can cause an increase in acute asthmatic episodes (see Dockery and Pope 1994 for review). Currently, there is no agreement among scientists as to whether a specific characteristic or component of PM is responsible for the observed health effects. Among the possibilities proposed are the physical characteristics of the particle or droplet (e.g., its size, shape or density), the number of particles present (i.e., particle number), its surface area, surface chemistry, surface charge or acidity. The specific chemical makeup of the particle or droplet is also thought to potentially contribute to health effects (e.g., elemental or organic carbon, volatile organic compounds, sulfates, nitrates, and metals such as iron, cadmium, cobalt, copper, manganese, nickel, lead, titanium, vanadium, zinc). Also of interest are particles of biological origin, such as fungal spores and pollen. The consistent finding of increased respiratory effects associated with increasing PM across areas with widely differing types of PM supports the hypothesis that more than one type of PM may be capable of producing the observed effects. Information about the potential for each of the various components of PM to worsen asthma or produce other respiratory symptoms is incomplete.

Diesel exhaust particulates (DEP) make up a significant portion of the $\mathrm{PM}_{10}$ in New York City (NYSDEC 1995). Diesel exhaust particles are generally composed of an elemental carbon core that may have a variety of organic compounds, metals, trace elements, sulfates and nitrates associated with its surface. Studies looking at DEP exposure and subsequent exposure to ragweed have associated increased allergic response with increased DEP exposure (Diaz-Sanchez et al. 1997). Studies in rodents have reported increases in airway hyper-responsiveness and inflammation following DEP and allergen challenge. These responses were reported to be greater than those observed with either DEP or antigen challenge alone (Takano et al.1998; Miyabara et al. 1998 as referenced in U.S. EPA 2002).

Several metals that can be associated with particulate matter have been found to affect lung function, including chromium, manganese and nickel. Nickel compounds have been associated with occupational asthma and can also act as a primary irritant (Agency for Toxic Substances and Disease Registry 1995). Chromium compounds have been associated with occupational asthma and decreases in forced expiratory volume at 1 second ( $\mathrm{FEV}_{1}$ ) and forced expiratory flow (Agency for Toxic Substances and Disease Registry 1993). Manganese compounds have been reported to cause an inflammatory response in the lung and reductions in lung function, and there has been some evidence of respiratory effects in residential populations near ferromanganese factories (Agency for Toxic Substances and Disease Registry 1992).

Both nitrous and sulfuric acids can be present in ambient air as acid aerosols, and strong acids such as these are known irritants. Nitrous acid is an irritant that is capable of producing symptoms in asthmatics (WHO 2000). Sulfuric acid, although a recognized irritant and corrosive at high concentrations, has not, by itself, been found to significantly affect lung function at environmentally relevant concentrations. Naturally occurring ammonia in the respiratory system is able to neutralize some inhaled acids, reducing the opportunity for acidic particles to contact tissues. However, if acid aerosol concentrations are elevated, or if underlying respiratory conditions diminish the system's ability to neutralize acids, the potential for respiratory irritation may be increased.

Airborne biological particles, or bioaerosols, carry protein allergens and inflammatory agents (such as $\beta-1,3-$ glucans) that can contribute to asthma exacerbations in sensitized patients. The common allergen bioaerosols in ambient air are pollen and fungal spores. In a study of asthma symptoms and air quality in Southern California, Delfino et al. (1997) found that exposure to fungal spores adversely affected respiratory status as increased asthma symptoms, inhaler use, and reduced peak expiratory flow rate. An earlier study by Delfino et al. (1996) found that personal ozone and fungal exposures were associated with increased asthma symptoms and inhaler use. Higgins et al. (2000) reported that increasing spore counts were associated with a drop in mean peak expiratory flow and an increase in its variability. These effects were reportedly greater when ozone levels were elevated prior to the increase in the spore counts. Dales et al. (2000) reported that
increases in ascomycete spores in air were associated with a $2.8 \%$ increase in pediatric emergency department visits for asthma. Dales et al. (2000) also reported that increases in basidiomycete spores in air were associated with a $4.1 \%$ increase in pediatric emergency department visits for asthma. Sensitization and exposure to grass pollen are risk factors for asthma prevalence and exacerbations (e.g., Schappi et al. 1999; Soriano et al. 1999; Basagana et al. 2001).

## GASES

Short-term exposures to high concentrations of sulfur dioxide in laboratory settings have produced respiratory symptoms (decrease in mean $\mathrm{FEV}_{1}$, increase in specific airway resistance, wheezing and shortness of breath) in healthy and asthmatic subjects (e.g., Linn et al. 1984a, b; Horstman et al. 1986, 1988; Heath et al. 1994; Gong et al. 1996 ). Epidemiological studies looking at populations exposed to sulfur dioxide as part of the ambient pollutant mixture have reported mixed results, perhaps due to the presence of other pollutants having similar effects on health (Schwela 2000).

Results from health effect studies of exposure to nitrogen dioxide are not consistent. However, relatively high concentrations of $\mathrm{NO}_{2}$ have been shown to increase bronchial reactivity, and in several studies they have been shown to enhance the response to aeroallergens when exposures to the gas and the allergen occur within a short time frame (Schwela 2000; Jenkins et al. 1999; D’Amato et al. 2002; Brunekreef and Holgate 2002).

In contrast to the other gaseous pollutants studied, laboratory and epidemiological studies of ozone exposure consistently show increases in respiratory symptoms and a variety of measures of asthma exacerbation as ozone concentrations increase (Schwela 2000; Peden 2002; Weisel et al. 1995). In addition, studies looking at combined or sequential exposures to ozone and allergens have noted an enhanced respiratory response compared with either exposure alone (D’Amato et al. 2002; Jenkins et al.1999). These studies may indicate that ozone exposures could create conditions within the respiratory system that might lower the threshold of effect for allergens or irritants.

Aldehydes (e.g., acetaldehyde, acrolein, formaldehyde, propionaldehyde) represent a class of HAPs that could negatively affect asthmatics. Formaldehyde has been reported to induce asthma in some individuals exposed in occupational settings (e.g., Feinman 1988). Acute, small decreases in respiratory function ( $\mathrm{FEV}_{1}$ ) have been reported after formaldehyde exposure in occupational settings (e.g., Alexandersson et al. 1982). Studies of asthmatics suggest that they may not be sensitive to formaldehyde at concentrations below those seen in occupational settings (e.g., Harving et al. 1986). Other aldehydes have not been as well studied, and potential interactions of aldehydes with other ambient contaminants have not been explored. Leikauf et al. (1995) point out that recent epidemiological studies suggest that pollutant interactions may potentiate respiratory responses.

## ASTHMA AND AIR POLLUTANTS IN NEW YORK CITY

A limited number of studies have investigated the association of air contaminants with acute asthma attacks in New York City. Thurston et al. (1992) studied the relationship between hospital admissions for asthma (and all respiratory admissions) and ambient acidic particulate matter and ozone concentrations during the summer in three regions in New York State. The researchers did not have air contaminant data for New York City, and they used ambient air data from the less urbanized suburbs. They found that higher concentrations of ozone, aerosol strong acidity (hydrogen ion) and sulfate were associated with increases in asthma admissions in the summer in Buffalo and New York City. However, they found the associations were weaker in Albany and the less urbanized New York City suburbs. This may be due, in part, to some chemical or physical difference in the composition or mix of air contaminants in the more densely populated areas.

In an older study conducted in New York, Greenburg et al. (1964) did not find an association between emergency clinic visits for asthma and sulfur dioxide, carbon monoxide, or coefficient of haze during September and October. Goldstein and Dulberg (1981) also found no significant relationship between hospital emergency department visits and sulfur dioxide or coefficient of haze measurements during the late summer and early fall. Jamason et al. (1997) found an association between asthma hospital admissions and air pollution in New York City during the spring and summer seasons but not during fall and winter. A recent study of asthma hospitalizations and ambient sulfur dioxide monitoring data in New York City found a consistent positive association between sulfur dioxide air levels and risk of asthma hospitalization in children, after adjusting for race, age and season (Lin et al. 2004).

Considering the limited information available regarding ambient air pollutants and asthma in New York City, and considering the state of the science on specific air pollutants and asthma in general, a better characterization of those air contaminants that may be associated with acute asthma attacks is needed. This study selected a set of chemical and biological factors that have been shown or are thought to have the potential to aggravate asthma and are likely to be present in urban air. The types of factors assessed were gases and vapors $\left(\mathrm{SO}_{2}, \mathrm{O}_{3}, \mathrm{NO}_{2}, \mathrm{NO}, \mathrm{NO}_{\mathrm{x}}\right.$ and a limited range of volatile organic compounds), particulates, particulate components (including sulfate, metals, carbon and hydrogen ion) and bioaerosols (pollen and fungal spores). These chemical and biological agents were measured in ambient air in two New York City locations, the South Bronx and Manhattan, over a period of nearly two years. Average air levels of the measured pollutants and patterns of change in pollutant levels over time were compared between the two sites.

## Section 4 <br> METHODS

## SAMPLING LOCATIONS

Two neighborhood sampling sites -I.S. 155 in the South Bronx and Mabel Dean Bacon School in Lower Manhattan—were selected for the study (Figures 1 and 2). These two monitoring sites were long-standing, EPA-approved air quality monitoring sites operated by New York State Department of Environmental Conservation (NYSDEC) for certain criteria air pollutants. They were located approximately 6.7 miles apart. The adequacy of these monitoring sites was evaluated by Lippman (1998). He concluded that both monitoring sites were "very well situated as regional urban sites." He further stated, "In fact, as urban monitoring sites go, these two currently have fewer complicating factors related to topography, major thoroughfares, major construction or demolition sites, etc., than most sites."

Partway through the project, a change in sampling location in the Bronx was necessary due to a construction project at IS 155. Working with EPA, NYSDEC and the New York City School Construction Authority, a new site was established at M.S. 52 ( 681 Kelly Street) in the South Bronx. As with the other monitoring sites, this site was evaluated and approved by EPA as an acceptable site. M.S. 52 is approximately 0.5 miles northeast of I.S. 155. Sampling occurred at I.S. 155 from January 1999 through August 1999, and at M.S. 52 from September 1999 through November 2000 (Figure 1). The Manhattan site at the Mabel Dean Bacon School (also known as Manhattan Comprehensive Night and Day High School), remained the same during the study period (January 1999 through November 2000). Sampling height in Manhattan was approximately seven stories and approximately four stories in the Bronx.

Sampling equipment was set up both on rooftops and indoors. Some outdoor equipment had climatecontrolled housing units (described below). A glass manifold attached to the building's exterior provided ambient air to equipment operating indoors. At the Bronx locations, the manifold's inlet was situated at approximately the same sampling height as, and located within 15 feet of, the rooftop instruments. Manhattan's manifold was located approximately 10 feet higher than, and 30 feet from, the outdoor sampling equipment.

## QUALITATIVE ENVIRONMENTAL AND ECONOMIC INFORMATION

Information on the population size, housing stock, traffic characteristics and number and types of businesses was collected for the two communities. Information sources included the U.S. Census Bureau, NYC TransitMTA, New York State and New York City Departments of Motor Vehicles, NYSDEC permits and the EPA Toxic Release Inventory. The information was used only as part of a qualitative description and comparison
of the two communities with respect to broad classes of potential air pollution sources. The study design did not include a detailed analysis of pollutant point sources, mobile sources or source apportionment.

## ANALYTICAL METHODS

A brief description of the analytical methods for ambient air analytes follows. Details, including quality assurance and quality control protocol references, are provided in Appendix 1.

## $\mathrm{PM}_{10}$ and $\mathrm{PM}_{2.5}$

Two TEOM ${ }^{\circledR}$ Series 1400a Ambient Particulate Monitors (Rupprecht and Patashnick Co., Inc., Albany, NY) were deployed at each location, one measuring $\mathrm{PM}_{10}$ and the other measuring $\mathrm{PM}_{2.5}$. Hourly average data were logged by the instruments and downloaded weekly by project staff. A supplemental system was attached to the $\mathrm{PM}_{2.5}$ units at each location for the measurement of metals (described below).

## FRM PM $\mathbf{1 0}_{10}$ and $\mathrm{PM}_{2.5}$

Twenty-four-hour particulate samples were collected for gravimetric measure of $\mathrm{PM}_{10}$ and $\mathrm{PM}_{2.5}$ using Federal Reference Method (FRM) protocols. PM $_{2.5}$ was collected using R\&P 2025 sequential samplers with WINS impactors. $\mathrm{PM}_{10}$ samples were collected using Wedding high-volume samplers with 8 - by 10 -inch quartz filters.

## Particle Number

A TSI Model 2022A condensation counter was used to measure the total number of airborne particles between 0.007 and 2.5 micrometers in diameter. The TSI instrument detects and counts particles using an optical detector. A computer linked to the counter logged data and data were downloaded once per week. Hourly and daily (24-hour) average values were calculated.

## Organic and Elemental Carbon

A Series 5400 Ambient Carbon Particulate Monitor (Rupprecht \& Patashnick Co., Inc., Albany, NY) was used for the measurement of organic and elemental carbon. The instrument uses a direct thermal- $\mathrm{CO}_{2}$ measurement to provide an indirect measure of the amount of carbon in the collected $\mathrm{PM}_{2.5}$ sample. The fraction volatilized or oxidized to $\mathrm{CO}_{2}$ between $250^{\circ} \mathrm{C}$ and $340^{\circ} \mathrm{C}$ was considered the volatile organic fraction, and the amount oxidized to $\mathrm{CO}_{2}$ between $340^{\circ} \mathrm{C}$ and $750^{\circ} \mathrm{C}$ was considered the elemental carbon fraction. Samples analyzed by the instrument represented three-hour averages. The instrument reports data to $0.1 \mu \mathrm{~g} / \mathrm{m} 3$. The results were logged by the instrument and downloaded weekly.

## Metals

An R\&P AccuSystem was installed on the TEOM collecting $\mathrm{PM}_{2.5}$ and used to collect particulate on filters for 24 hours each day (midnight to midnight) for metal analysis. The samples (filters) were gathered each week
and brought to the laboratory. The samples were analyzed at the Wadsworth Laboratory using inductively conductive plasma/mass spectrometry (ICP/MS). The following metals thought to have a possible relationship with asthma exacerbation or respiratory irritancy, based on existing information, were included in the analysis (detection limits): $\mathrm{Cr}\left(5\right.$ nanograms $\left./ \mathrm{m}^{3}\right)$, $\mathrm{Fe}\left(22 \mathrm{ng} / \mathrm{m}^{3}\right)$, $\mathrm{Pb}\left(12 \mathrm{ng} / \mathrm{m}^{3}\right)$, $\mathrm{Mn}\left(3 \mathrm{ng} / \mathrm{m}^{3}\right), \mathrm{Ni}\left(4 \mathrm{ng} / \mathrm{m}^{3}\right)$ and $\mathrm{Zn}(77$ $n g / m^{3}$ ).

## Acid Aerosols, Ammonia, and Acid Gases

Daily samples were collected on filters and denuders to characterize five reactive gases $\left(\mathrm{NH}_{3}, \mathrm{HCl}, \mathrm{HNO}_{2}\right.$, $\mathrm{HNO}_{3}$, and $\mathrm{SO}_{2}$ ), particulate $\left(\mathrm{PM}_{2.5}\right)$ sulfate and pH (U.S. EPA Method IO-4.2). The five gases were not part of the original study plan and were analyzed for only approximately one year of the study. Samples represent 24-hour averages. Samples were collected on a URG-2000-01J Weekly Air Particulate Sampler (URG, Chapel Hill, NC). The gases were collected on denuders and the aerosols on a Zeflour filter supported by a PTFE-coated stainless steel screen. Ion chromatography was used to measure concentrations. The detection limits for the various analytes were $\mathrm{NH}_{3}\left(0.19 \mathrm{micrograms} / \mathrm{m}^{3}\right), \mathrm{HCl}\left(0.10 \mu \mathrm{~g} / \mathrm{m}^{3}\right), \mathrm{HNO}_{2}\left(0.16 \mu \mathrm{~g} / \mathrm{m}^{3}\right)$, $\mathrm{HNO}_{3}\left(0.10 \mu \mathrm{~g} / \mathrm{m}^{3}\right)$, and $\mathrm{SO}_{2}\left(0.18 \mu \mathrm{~g} / \mathrm{m}^{3}\right)$.

Particulate nitrate was originally included in the analyte list but was later dropped due to concerns about the accuracy of the reported concentrations. During the study, research was published that called into question particulate nitrate concentrations collected on Teflon filters, especially at higher temperatures (U.S. EPA 1999). The particulate nitrate samples were collected on Teflon filters, and temperature measurements made inside the sampler enclosure for about one month showed a high reading of $108^{\circ} \mathrm{F}$. Because the sampler was serviced only once per week, samples collected after servicing were potentially subject to more hightemperature periods than those collected just prior to servicing, likely increasing the potential for particulate nitrate volatilization. This information, along with inconsistencies found in the concentrations of some colocated samples, led to the removal of particulate nitrate from the analyte list.

## Bioaerosols

Bioaerosol samples for enumeration of pollen and fungal spores were collected into the wind on adhesivecoated tape that was mounted on a clock-driven drum inside a low-volume sampler (Burkard seven-day recording spore trap). The clock allowed a seven-day, non-integrated, time-ordered sample to be collected. After removal of the drum, the tape was sectioned into seven equal parts, mounted on microscope slides, stained and viewed microscopically. Bioaerosol results were reported as daily (24-hour) averages.

Pollen and fungal spores were categorized into several large (in some cases overlapping) groups for statistical analyses, based on taxonomic and/or morphologic similarities. For pollen, the categories were tree, grass, ragweed, and total pollen. For fungal spores, the categories were basidiospores, ascospores, dark color
mitospores, non-dark mitospores, small spores (< 10 micrometers in the largest dimension), large spores (> 10 micrometers in the largest dimension) and total spores (see Appendix 1, Table A1).

## Acetone and Aldehydes

An automated sampler was used in the collection of daily (24-hour average) samples for acetone and aldehyde analysis, according to U.S. EPA Method TO-11. The analytes measured were acetone, acetaldehyde, acrolein, benzaldehyde, butyraldehyde, crotonaldehyde, 2,5-dimethylbenzaldehyde, formaldehyde, hexaldehyde, isovaleraldehyde, propionaldehyde, m-tolualdehyde, o-tolualdehyde, p-tolualdehyde and valeraldehyde. Detection limit for each was $1 \mu \mathrm{~g} / \mathrm{m}^{3}$. During the study, questions were raised about the validity of the acrolein data from this method due to poor recovery and possible dimerization of this analyte on sample cartridges.

## Criteria Pollutant Gases and Other Nitrogen Oxides

$\mathrm{SO}_{2}, \mathrm{NO}, \mathrm{NO}_{2}, \mathrm{NO}_{\mathrm{x}}$ and $\mathrm{O}_{3}$ were measured by EPA-approved methods (40 CFR Chapter I Part 50 and DEC web page, www.dec.state.ny.us/website/dar/reports/99annrpt/99ar_mtd.html). Data for all of these analytes were analyzed on an hourly and daily (24-hour) average basis. $\mathrm{O}_{3}$ was also analyzed on an eight-hour moving average basis, following the National Ambient Air Quality Standards calculation algorithm (40 CFR Chapter I Part 50; see below).

## Meteorological Data

Temperature, relative humidity, wind speed and wind direction were logged from a roof-mounted meteorological station at each site. The unit logged the data from wind monitor Model 05305 and relative humidity and temperature probe Model 41372LC (R.M. Young Co., Traverse City MI).

## DATA QUALITY

Data cleaning beyond the quality assurance and quality control protocols developed for the instruments was conducted to ensure that data importation had been correctly implemented. Any observations associated with known instrument malfunctions (e.g., power loss or incorrect airflow) were marked as rejected. To identify more subtle potential reporting problems with the pollutants, time series plots of some pollutants were examined for unusual observations or abnormal fluctuations. Differences between the two sites were calculated, and the data for time periods with large differences were further investigated. Screening criteria were developed to identify observations that required review. Observations were further examined for data quality if any of the following obtained:

- a value was considered a statistical outlier (i.e., more than two standard deviations from the mean);
- the data did not follow previous patterns often identified from inspection of graphs of the data; or
- an unusual trend in the data was found (e.g., a low value every third day).

Possible causes of such observations were explored. If instrument error (e.g., airflow or temperature outside specifications) was not determined to be the cause, the data were assumed to be accurate; otherwise, the result was marked as suspicious. Suspicious and rejected observations were removed from the dataset and not included in any descriptive statistics or analyses.

## STATISTICAL ANALYSIS

## Summary Statistics

Summary statistics were compiled for each pollutant at each site. For sulfate, aldehydes, and metals, observations below the limit of detection were estimated at half the detection limit. No non-detects occurred for the other chemical analytes. Bioaerosol samples where non-detects occurred were entered as zeros. The summaries included mean, standard deviation, sample percentiles, sample size ( N ), number of suspicious results (SR), number of rejected results (RJ), number of observations below detection limit (LT), number of observations present but less than detection limit (PL) and number of missing observations. Detailed data summaries for all analytes are provided in Appendix 2.

Analytical chemistry results were reported as one-hour, three-hour or 24-hour time-weighted averages (TWA), depending on the sampling methodology for each analyte. Therefore, summary statistics for each analyte could be calculated for up to three averaging times (24-hour, seasonal and the entire study period). In the presentation of results, daily mean refers to 24 -hour averages from either 24-hour time-weighted-average sample results or from averaging hourly or three-hour TWA observations across 24-hour intervals. Seasonal mean is used to refer to observations averaged over three-month intervals (described below) and overall mean is used to refer to observations averaged over the entire study period. Seasonal and overall summary statistics were calculated from daily means.

Exploratory analyses were conducted for all analyte data sets to evaluate whether the distribution shape for each was approximately normal. Distributions were characterized informally using histograms and normal probability plots. The Anderson-Darling goodness-of-fit test for the normal distribution was used to formally test distributions for their deviation from normality (D’Augostino and Stevens 1986). Since statistical comparisons were of meaningfully paired observations, the differences between paired observations were the data subjected to statistical analysis. Although differences between paired observations tended to deviate from normality, based on formal goodness-of-fit tests, their distributions deviated less from normality than did the original observations and were generally symmetric and bell-shaped, similar to a normal distribution.

Therefore, it was felt that, since the t- and F-tests are robust to deviations from the normality assumption (e.g., Neter et al. 1990), these tests could be applied to non-transformed differences.

## Site Comparisons

Analyte air concentrations in the two communities were compared using daily (24-hour) mean analyte levels and daily maximum analyte levels at the Manhattan and Bronx sampling sites. Hourly observations or threehour average observations (elemental and organic carbon variables) were averaged together on a 24-hour basis to obtain daily averages. A daily maximum value was identified from hourly and three-hour average observations if at least 75\% of that day's hourly (three-hour) observations were available. Daily maximum comparisons were not made for those variables collected only on a daily (24-hour) average basis.

For ozone, moving eight-hour averages were calculated from the original hourly observations by applying the EPA National Ambient Air Quality Standards (NAAQS) guidelines for evaluating moving eight-hour averages against the eight-hour ambient air standard. Eight-hour moving averages for ozone were assigned to the first hour of the eight-hour window. If six or more hourly observations were valid for an eight-hour segment, the non-missing observations were averaged; if less than six but at least one hourly observation was valid for the eight-hour segment, missing values were estimated at half the detection limit ( $0.002 / 2=0.001$ ) and all eight values were averaged; if none of the eight observations were valid, the eight-hour average is missing. Twenty-four-hour average ozone concentrations were calculated from the original hourly average data. Daily maximum hourly ozone observations were based on original hourly average data and on eighthour moving-average data.

There was substantial seasonal variation for many analytes in the study, so seasonally stratified statistical analyses as well as unstratified analyses were performed. The data were divided into eight seasonal categories:

- Winter 1999: January 1-March 20
- Spring 1999: March 21-June 20
- Summer 1999: June 21-September 22
- Fall 1999: September 23-December 21
- Winter 2000: December 22, 1999-March 19
- Spring 2000: March 20-June 19
- $\quad$ Summer 2000: June 20-September 21
- Fall 2000: September 22-November 22

The analytes were measured at the same times for the same duration at each site. For this reason, the pollutant data for the two sites were considered paired data. Daily differences were calculated and analyzed for each analyte. The mean differences were computed seasonally and for the entire study period. The analyses of the daily differences used paired t-tests with an autocorrelation adjustment. The variance of the differences is adjusted to account for the non-independence of autocorrelated time-series data. The adjustment given by Gilbert (1987), taking the sample variance as an estimator of the population variance, is as follows:

$$
\hat{s}_{d}^{2}=\frac{s_{d}^{2}}{n}\left[1+\frac{2}{n} \sum_{l=1}^{n-1}(n-l) \rho_{l}\right]
$$

where $S_{d}^{2}$ is the original sample variance of the differences, $\hat{S}_{d}^{2}$ is the adjusted sample variance of the differences, $n$ is the sample size, $l$ is the lag distance between two observations in the series and $\rho_{l}$ is the autocorrelation coefficient for lag $l$. The adjustment was applied assuming that the only contribution to the sum comes from statistically significant autocorrelation coefficients. That is, if the first $m$ autocorrelations are significant (and therefore $n-m$ autocorrelations are not significant), then for $l>m, \rho_{l}=0$.

Daily differences were calculated for daily average and for daily maximum hour for those contaminants with hourly data and daily three-hour maximum for carbon measures. For pollutant data collected hourly, daily maximums were generated for days considered $75 \%$ complete. Daily differences of the maximums were analyzed seasonally in the same way as daily mean differences, using a paired t-tests adjusted for autocorrelation. Detailed results of all statistical comparisons, analyzed for the entire study period and by season, are presented in Appendices 3 and 4, respectively.

The relocation of the Bronx monitoring site during the study brought into question whether the two Bronx sites were sufficiently similar in their representation of local air quality that their results could be combined. This question led to an additional analysis to evaluate the comparability of the two locations in terms of air quality. A direct comparison of Bronx Site A with Site B was not possible because data could not be collected at the two places simultaneously. Instead, data from each site were compared with data for the corresponding period at the Manhattan location using an adjusted paired t-test to try to control, at least to some extent, for temporal differences. By comparing the relationship between analyte levels at the Manhattan site and the two Bronx sites, a qualitative assessment could be made as to whether the two Bronx sites provided comparable results regarding the differences between pollutant levels in the Bronx and in Manhattan. However, if different trends were observed in results relating Manhattan and the two Bronx sites, it would not be possible to determine whether they were due to differences in the Bronx monitoring sites or to differences in the relationship between pollutants in the Bronx and pollutants in Manhattan over time.

## Correlation Between Monitoring Sites.

The correlation between the two sampling sites for each analyte was estimated using the Pearson correlation coefficient. This statistic measures the degree to which the same variable at the two sites followed a similar pattern of fluctuations through time, whether or not the mean levels were different.

## Correlation Among Pollutant Variables at a Monitoring Site.

Non-metric multidimensional scaling (MDS) analysis and complete-linkage hierarchical clustering (HC) were employed in an exploratory analysis to characterize associations among chemical analytes (Mardia et al. 1979). Data from each sampling site were analyzed separately. In both analyses, correlation matrices for 21 pollutant variables were summarized graphically to explore patterns of associations among variables. In both analyses, the pH variable was recoded as hydrogen ion concentration (by taking the anti-log of -pH ), so that increasing hydrogen-ion values would indicate increasing concentration, similar to the other pollutant variables. Details of the implementation of these techniques are provided in Appendix 1.

Pearson correlation estimates were also obtained for all pairwise analyte combinations within each sampling location as part of the initial exploratory analysis of the data. The detailed raw Pearson correlation matrices are presented in Appendix 5.

## Temporal Analyses

To characterize the temporal patterns of the pollutants, data from the entire study for each pollutant were averaged on a day-of-week basis and, when applicable, on an hour-of-day basis. For pollutant concentrations collected more than once per day, daily averages were used for day-of-week trends. Daily averages were calculated for days in which at least $75 \%$ of the available data were collected. All available hourly data were included in the hour-of-day averages. Day-of-week and hour-of-day averages $\pm$ two standard errors were plotted and temporal patterns were inferred from these graphs.

## Section 5

## RESULTS

## QUALITATIVE ENVIRONMENTAL AND ECONOMIC INFORMATION

The 2000 U.S. Census data show that about 100,000 more people live in the Manhattan study area than in the Bronx study area (Table 1A). The Manhattan study area also has about 120,000 more occupied housing units, so the average occupancy per housing unit in the Bronx study area is almost twice that in the Manhattan area (Table 1B). Renters in both communities occupy most of the housing units.

The number of motor vehicles registered in 2001 with the New York State Department of Motor Vehicles is about equal between New York County (i.e., Manhattan) and Bronx County (Table 1C). An evaluation of axle counts on selected roads showed that the number of vehicles is about equal. Both communities are adjacent to major highways— FDR Drive for the Manhattan study area and the Major Deegan and Bruckner Boulevard for the Bronx community. Although the total amount of vehicle traffic on these highways is about the same, FDR Drive does not allow commercial traffic while the Major Deegan and Bruckner Boulevard are major commercial traffic routes. The number of MTA buses in the two communities is similar but the routes in Manhattan are traveled with greater frequency.

Manhattan has one hazardous waste site on NYSDEC’s New York State Registry of Inactive Hazardous Waste Sites; the Bronx has three. No NYSDEC-permitted waste-handling facilities were located in Manhattan in 2000, but there were 15 in the Bronx.

Both communities have industrial sources of urban air contaminants. The Toxic Release Inventory (TRI) program tracks some industrial chemical emissions to the environment. TRI facilities are manufacturing and other industrial operations required to report chemical emissions or transfers to air, water, soil and waste treatment facilities under Section 313 of the federal Emergency Planning and Community Right to Know Act. In 2000, two TRI facilities submitted reports in Manhattan compared with eight in the Bronx. However, the total quantity of air emissions reported under the TRI program in 2000 was greater in Manhattan than in the Bronx (approximately 30,000 pounds versus 15,500 pounds). All but about 6.5 pounds of the Manhattan TRI releases (i.e., 99.98\%) were sulfuric acid. The remainder included less than 0.5 pound of dioxin and dioxinlike compounds and 6 pounds of polycyclic aromatic hydrocarbons. All the Manhattan releases were reported from a single facility (Consolidated Edison, East River Facility). The other Manhattan facility submitting a TRI report had no air releases in 2000. Almost $90 \%$ of the Bronx releases were trichloroethylene from a single facility (G.A.L. Manufacturing Corp.), with the remainder consisting of small amounts of toluene, xylene, zinc, glycol ethers and 1,2,4-trimethylbenzene. Three of the eight Bronx facilities submitting TRI reports had no air releases in 2000.

A review of the 2000 U.S. Census Bureau data suggested that the Manhattan study area had more businesses and that the types of businesses differed between the two communities (Table 2). Information was not available to assess whether businesses enumerated in these data sources actually represent activities that would be associated with air emissions. For example, many businesses recorded as agricultural or manufacturing in the Census data may only represent corporate offices, without significant agricultural or manufacturing activity.

Based on anecdotal NYSDOH staff observations, the Manhattan study area generally had taller buildings and more pedestrian and vehicular traffic than the Bronx study area. Prior to the study, Manhattan community members expressed concern about an electricity-generating plant as an air pollution point source. Members of the Bronx community expressed concerns about impacts on air quality from a large sewage treatment facility (Hunts Point), rotting produce at the Hunts Point markets, and a sewage sludge pelletization plant (New York Organic Fertilizer Co., NYOFCO).

## DATA COLLECTION AND LABORATORY ANALYSIS QUALITY CONTROL

Data collection was generally successful, despite some intermittent equipment malfunctions. The equipment to count particle number was the most problematic and a large amount of data from both sites was dropped because it did not meet data quality standards. Intermittent equipment breakdowns also caused loss of nitrogen dioxide, nitric oxide and nitrogen oxides data from the Bronx (and to a lesser degree, Manhattan) for the winter of 1999. Details of data completeness are provided in Appendix 2.

Some additional analytes (hydrochloric acid, nitrous acid, nitric acid and ammonia) were evaluated for a more limited time period (approximately a year, from June 23, 1999, to July 11, 2000). The period for ammonia samples was more limited, from June 23 to August 31, 1999 and from December 29, 1999, to May 16, 2000. These analytes were not included in the original study design and were added to the analysis as limited resources allowed.

The laboratory analysis for acetone and aldehydes could have measured up to 14 compounds. However, most were generally below the detection limit of 1 microgram $/$ meter $^{3}$ and were therefore not included in the analyses comparing the ambient air levels in the Bronx and Manhattan. Acetone was detected in $99.2 \%$ of the samples in the Bronx and 97.2\% in Manhattan. Acetaldehyde was detected in $98.8 \%$ of the samples in the Bronx and $98.2 \%$ in Manhattan. Formaldehyde was detected in 99.2\% of the samples in the Bronx and 99.1\% in Manhattan. The remaining aldehydes were detected in less than $35 \%$ of the samples.

Four of the metals analyzed were only detected in a limited set of the samples. Chromium, manganese, lead and zinc were detected in less than $11 \%$ of the samples and were not analyzed further. Iron and nickel were
detected in enough samples to allow comparison between the two sites. Iron was detected in $77.7 \%$ of the samples in the Bronx and $79.7 \%$ in Manhattan. Nickel was detected in $66.8 \%$ of the samples in the Bronx and 74.1\% in Manhattan.

## COMPARISON OF AMBIENT AIR QUALITY

The comparisons detailed in this section consider the two Bronx sites as one; the appropriateness of this treatment is discussed in the next section.

The daily average air concentration data are graphically summarized in Figures 3 to 35 . The top panel in each figure shows the values for the Bronx and Manhattan monitoring sites and the lower panel shows the difference in concentration between the two sites (Manhattan - Bronx). A negative number in the lower panel indicates that the average concentration was greater in the Bronx on that day. Generally the data for the two sites look quite similar in most figures. Daily concentrations at both sites varied substantially, with ranges often varying by 10 -fold or more. Some analytes (e.g., pollen, fungal spores, ozone, sulfur dioxide) showed marked seasonal variation. Many contaminants had no consistent trend showing higher levels in one sampling area or the other. For other compounds, however, the trend is consistently higher in one location. For instance, ozone was fairly consistently higher in the Bronx (Figure 31), whereas nitrogen dioxide was higher in Manhattan (Figure 33).

The daily average results for particulate matter are presented in Table 3. Two size fractions (less than 2.5 micrometers and less than 10 micrometers) were measured, each by two different methods. In all cases the overall mean concentration was higher at the Manhattan monitoring site than at the Bronx monitoring site. The differences in concentrations ranged from $3 \%$ to $11 \%$. The differences in mean values using the two methods are due to several factors, including differences in how the mass is measured, missing data for one method but not the other and slight variations possibly due to differences in location of the air intakes. In most seasons, the concentration of $\mathrm{PM}_{2.5}$ was significantly greater in Manhattan. Similarly, significant differences in seasonal results were also generally observed for $\mathrm{PM}_{10}$ measured with the automated mass measurement method. However, this was not generally the case for measurements made using the FRM. The FRM PM 10 collected data only once every sixth day and so had less statistical power to discern a given difference between sites than the automated mass measurement method.

The number of particles less than 2.5 micrometers was not significantly different over the study period at the two sites (Table 4). Because of technical problems, data were not collected for winter, spring and summer 1999, limiting particle count data to only five seasons.

Results for pH , sulfate and organic and elemental carbon constituents of $\mathrm{PM}_{2.5}$ are summarized in Table 5, and $\mathrm{PM}_{2.5}$ metals results are summarized in Table 6. Overall, the pH was slightly lower (more acidic) at the

Manhattan monitoring site than at the Bronx monitoring site. In only three of the eight study seasons was the difference statistically significant, and the difference was never statistically significant in the winter. Overall, sulfate was higher at the Manhattan monitoring site; the differences were statistically different in four of the eight study seasons. Overall, organic carbon was not consistently different between the two sites. Average elemental carbon concentrations were slightly greater in Manhattan, although the differences were statistically different in only three of the eight study seasons. Overall, iron concentrations did not vary between the two sites. Although in some seasons there were significant differences, they were not consistently in one direction. Overall, nickel was higher at the Manhattan monitoring site. The differences were statistically different in four of the eight study seasons.

Pollen counts tended to be higher at the Bronx monitoring sites than at the Manhattan monitoring site (Table 7). For ragweed pollen and grass pollen, these differences were statistically significant over the entire study period, although seasonal differences were generally not significant. For tree pollen and total pollen, some seasonal mean comparisons were statistically significant, but the overall comparisons were not significant.

Seasonal variability in tree pollen levels during the entire study period was large compared with variability between the study areas, such that overall study means were not significantly different. The variance estimate for the overall tree pollen comparison was also increased compared with the individual seasonal comparisons because more lag periods were included in the autocorrelation adjustment for the overall comparison. Total pollen levels were dominated by tree pollen levels, and thus site differences over the study period in total pollen were also not significant, despite significant seasonal differences. All statistically significant seasonal differences in tree pollen and total pollen were greater in the Bronx.

Overall, mean fungal spore levels were not different between the two sites (Table 8). The only statistically significant difference between sites for the entire study period was for large spores. On a seasonal basis, most mean differences between the sites were not statistically significant, and one site did not have consistently higher mean levels among those seasonal comparisons where significant differences were observed.

Over the entire study period, no statistically significant differences between the mean concentrations of acetone, formaldehyde or acetaldehyde were found at the two sites (Table 9). Slightly more seasonal differences were in the direction of higher levels in Manhattan than in the Bronx.

Mean hydrochloric acid, nitrous acid, nitric acid, denuder sulfur dioxide and ammonia levels all were significantly higher over the entire study period at the Manhattan monitoring site compared with the Bronx site (Table 10). Most statistically significant seasonal mean differences were also in the direction of higher mean levels in Manhattan for these analytes, with the exception of one seasonal difference for hydrochloric acid.

The daily average results for ozone, sulfur dioxide, nitric oxide, nitrogen dioxide and total nitrogen oxides are summarized in Table 11. Mean ozone concentrations were higher at the Bronx monitoring site. Mean concentrations for the other pollutant gases over the entire study period were all significantly higher in Manhattan. The same pattern of statistically significant differences between the two sites for these five analytes was seen on a seasonal basis. All significant seasonal ozone differences were in the direction of higher mean levels in the Bronx, while higher mean levels for the sulfur and nitrogen oxide variables were observed in Manhattan.

## COMPARISON OF THE TWO DIFFERENT MONITORING SITES IN THE BRONX TO MANHATTAN

The results of the comparison of daily average concentrations for each Bronx site to the Manhattan site are summarized in Tables 12 to 18 . For 24 of 34 analytes, the monitoring site with the higher mean was the same in 1999 and in 2000. In 10 cases, the direction of the mean difference reversed between 1999 and 2000, although only four of the 10 comparisons that reversed direction involved significant differences in at least one of the comparisons. Although some variation in the relative levels of air contaminants between Bronx and Manhattan was observed between the two Bronx sites, strong evidence indicating that it would be inappropriate to combine data from the two Bronx sites was not found.

Correlations were also estimated for corresponding observations from each Bronx sampling location and the Manhattan location and were qualitatively compared (Table 22). Most correlations were of similar magnitude. A few pollutants (acetone, nitrogen oxides, $\mathrm{PM}_{2.5} \mathrm{FRM}$ ) had notably different correlation coefficients when comparing the two years. In all cases, a small number of unusually high or low observations at one site, not paralleled by similar extreme observations at the other site, substantially lowered the overall correlation coefficient. This correlational analysis also failed to provide strong evidence that it would be inappropriate to combine data from the two Bronx sites.

## DAILY MAXIMUM VALUES

For $\mathrm{PM}_{2.5}$ and $\mathrm{PM}_{10}$ (by automated samplers), particle number, organic and elemental carbon, ozone, sulfur dioxide and nitrogen oxides, multiple measurements were made throughout the day, making possible a daily maximum observation (one-hour or three-hour, depending on analyte). Over the entire study period, most of the mean differences in daily maximum value were in the same direction as for the daily averages; however, fewer of the differences were statistically significant (Table 19). The only contaminant where the direction of the difference changed between the overall means and the daily maximum means was organic carbon. Mean daily maximum organic carbon was slightly higher in Manhattan for the entire study period, in contrast to the overall mean comparison for this analyte, which was slightly higher in the Bronx. Neither difference was statistically significant.

## CORRELATION BETWEEN THE BRONX AND MANHATTAN MONITORING SITES

Although daily average concentrations may be statistically significantly different between Manhattan and the Bronx, the daily averages at the sites may tend to fluctuate in a similar pattern over time. This can be seen graphically in Figures 3-35. To evaluate this, correlations between the two monitoring sites were estimated for each analyte. Most between-site correlations were relatively strong, with correlation estimates falling below 0.6 for only five analytes (non-dark mitospores, formaldehyde, acetone, iron and nickel; Table 20).

## CORRELATION BETWEEN DIFFERENT AIR CONTAMINANTS WITHIN MONITORING SITES

## Daily Mean versus Daily Maximum

For analytes where a daily maximum value could be obtained, correlations of daily maximum and daily mean values were estimated within each sampling location (Table 21). Not surprisingly, the correlations between daily maximums and daily average were fairly high. Pearson r values were $\geq 0.85$ for all analytes except particle number. This is consistent with the strong influence of large values on the arithmetic daily mean.

## Multidimensional Scaling

Special tests, referred to as diagnostics, were included in the MDS analyses to ensure that models of the associations among variables were not based on non-degenerate solutions (e.g., Wilkinson 1999; see Appendix 1). None of the MDS solutions produced diagnostics that would indicate a degenerate model solution. Similar patterns of associations among variables were observed from MDS results for the two sampling locations.

Striking patterns of variables-with points very close together in the MDS plots and clearly separated from other distinct clusters-were generally not observed (Figures 36-40), although in most configurations the two measures of sulfur dioxide $\left(\mathrm{SO}_{2}\right.$ and denuder- $\left.\mathrm{SO}_{2}\right)$ did appear closely associated and relatively isolated from all other variables. This indicates a strong positive correlation between these two variables and a tendency to weak or negative correlations of those two with most other variables. During the two seasonal periods spanning the fall and winter months (especially January-March), ozone $\left(\mathrm{O}_{3}\right)$ tended to be widely separated from all other variables in the MDS plots (Figures 37, 40), indicating a strong negative correlation with most other pollutant variables during those periods. The large negative association between $\mathrm{O}_{3}$ and most other variables during these periods obscured any other patterns of association among the remaining variables.

In the combined-seasons plots (Figure 36) and to a lesser degree in the spring and summer plots (Figures 38, 39), two loose aggregations of variables appeared to fall on opposites sides of the first MDS dimension, although the resolution of these two aggregations as distinct clusters was not strong. One aggregation usually included all nitrogen oxide variables ( $\mathrm{NO}, \mathrm{NO}_{2}, \mathrm{NO}_{\mathrm{x}}$ ), $\mathrm{SO}_{2}$, denuder- $\mathrm{SO}_{2}$, elemental carbon and nitrous acid $\left(\mathrm{HNO}_{2}\right)$. The other aggregation generally included the two particulate-matter variables $\left(\mathrm{PM}_{25}, \mathrm{PM}_{10}\right)$, sulfate
( $\mathrm{SO}_{4}^{--}$), formaldehyde, acetaldehyde, acetone and organic carbon. Iron ( Fe ), nickel ( Ni ), hydrochloric ( HCl ) and nitric $\left(\mathrm{HNO}_{3}\right)$ acids, hydrogen ion $(\mathrm{H}+)$, ammonia $\left(\mathrm{NH}_{3}\right)$ and ozone $\left(\mathrm{O}_{3}\right)$ tended to be less consistently associated with either of the two main aggregations. As noted above, these aggregations tended to be obscured during the fall and winter seasons, when $\mathrm{O}_{3}$ tended to be strongly negatively associated with all other variables.

## Hierarchical Clustering

The HC results (Figures 41-45) were generally consistent with the MDS results. In most cases, the pairs of variables that clustered together with the lowest distances (highest correlations) were $\mathrm{NO}_{\mathrm{X}} / \mathrm{NO}, \mathrm{PM}_{25} / \mathrm{PM}_{10}$, $\mathrm{SO}_{2} /$ denuder- $\mathrm{SO}_{2}$ and acetaldehyde/formaldehyde. $\mathrm{SO}_{2}$, elemental carbon, metals and $\mathrm{NO}_{2}$ or $\mathrm{NO}_{\mathrm{X}}$ were frequently clustered together at relatively low distances. $\mathrm{SO}_{4}{ }^{--}$(either alone or clustered with hydrogen ion concentration), aldehydes, acetone, organic carbon, inorganic acids and PM variables were closely associated in several trees. Especially in the fall and winter seasons, $\mathrm{O}_{3}$ tended to diverge from the other clusters containing all other variables at large distances-indicating strong negative associations—at both sampling locations.

## TEMPORAL ANALYSES

Measurements for most variables did not vary noticeably by day of the week (Figures 46, 48, 50-52, 54-65, 67,69 ). $\mathrm{PM}_{10}$, acetone, elemental carbon, $\mathrm{NO}, \mathrm{NO}_{2}, \mathrm{NO}_{\mathrm{x}}$ and particulate Fe were the only variables showing a noticeable day-of-week trend, with somewhat lower daily means on the weekends (especially Sundays) increasing during the week. Day-of-week variation was similar between the two monitoring areas.

Time-of-day trends were more pronounced than day-of-week trends for many of the analytes where hourly or three-hour-average observations were available (Figures 47, 49, 53, 66, 68). $\mathrm{SO}_{2}, \mathrm{NO}, \mathrm{NO}_{2}, \mathrm{NO}_{\mathrm{x}}, \mathrm{PM}_{2.5}, \mathrm{PM}_{10}$ (automated mass monitors) and, to a lesser degree, elemental carbon all showed daily peaks in the morning hours (approximately 6-8 A.m.). $\mathrm{O}_{3}$ showed a tendency toward daily minimum values at the same morning hours and a daily afternoon (2 P.m.) peak. These trends were consistent between the two monitoring areas. The time-of-day trends in hourly average particle number differed between Bronx and Manhattan, with somewhat elevated hourly averages in the Bronx from midnight to 4 A.m., whereas Manhattan particle counts during those hours were somewhat lower than during the rest of the day (Figure 49). Little time-of-day variation was observed in three-hour-average organic carbon levels at either site (Figure 53).

Seasonally, the concentrations of nitric acid, hydrochloric acid, ammonia, and sulfate were higher during summer than winter. The summer-winter ratios for nitric acid, hydrochloric acid and sulfate in Manhattan were 3.9,3.1 and 1.9, respectively. The concentrations of nitrous acid and sulfur dioxide were higher during winter than summer; the summer-winter ratios in Manhattan were 0.48 and 0.44 , respectively. Gaseous nitrous acid was the predominant form compared with nitric acid except in summer. The annual mean
concentrations of $\mathrm{PM}_{2.5}$ were 15.2 and $15.5 \mu \mathrm{~g} / \mathrm{m}^{3}$ in the Bronx and in Manhattan, respectively. The monthly mean concentrations in Manhattan ranged from 13.2 to $21.7 \mu \mathrm{~g} / \mathrm{m}^{3}$; they were highest in June and July and lowest in March and April. The monthly mean fraction of $\mathrm{PM}_{2.5}$ as sulfate ranged from 0.17 to 0.31 ; the highest fraction values were observed during June-September.

An analysis of the air monitoring data for sulfate, $\mathrm{SO}_{2}, \mathrm{HCl}$, ammonia, nitric acid, nitrous acid and $\mathrm{PM}_{2.5}$ has been published (Bari et al. 2003b).

## WIND TRAJECTORY ANALYSES

Although detailed source attribution was not a focus of the study design, the data were amenable to evaluating the relative contributions of long-distance pollutant transport versus local pollutant emissions by backtrajectory analysis. This was a secondary analysis that did not apply directly to the main objective of this report-that is, the air quality comparison between the two communities.

Air trajectories were used to study the effect of upwind emissions on the observed concentrations in New York City. Episodes of high concentrations of chemical species were observed in both the Bronx and Manhattan throughout the year, although they were more prominent during summer. The highest concentrations were invariably associated with the air flow from southwest to west of New York City.

Three-hour HYSPLIT4 air trajectories were used to apportion the daily measured concentrations of seven analytes- $\mathrm{PM}_{2.5}$, sulfate, $\mathrm{SO}_{2}, \mathrm{HCl}$, nitric acid, nitrous acid and ammonia—and as a function of direction. Comparison of the air trajectories with the measured concentrations suggested that a fraction of sulfate, $\mathrm{SO}_{2}$, HCl , nitric acid, and $\mathrm{PM}_{2.5}$ is transported from west and southwest of New York. Nitrous acid and ammonia concentrations appeared unrelated to the air trajectories. Air trajectories were used to evaluate contributions from the regional emission sources to the observed levels of $\mathrm{SO}_{2}$, sulfate, PM2.5, nitric acid and HCl . On an annual basis, $\sim 40 \%$ of sulfate was transported from the Midwest and $\sim 60 \%$ from nearby ( $\sim 150 \mathrm{~km}$ ) sources. On the other hand, only $\sim 14 \%$ of $\mathrm{SO}_{2}, 30 \%$ of $\mathrm{PM}_{2.5}, 27 \%$ of HCl and $24 \%$ of nitric acid were transported, with the remainder coming from the nearby sources. During the third quarter of 1999, about $26 \%$ and $40 \%$ of HCl and nitric acid, respectively, were transported from the distant sources. The modeled contributions from regional sources and transport were generally similar in Manhattan and the Bronx. The complete details are reported in Bari et al. (2003a).

## Section 6 DISCUSSION

Most analytes measured in the study either did not show a statistically significant difference between levels at the Manhattan site and the Bronx site (most mold categories, iron, aldehydes, elemental carbon and organic carbon) or had mean levels in Manhattan that were significantly higher than those in the Bronx (PM, particulate acidity and sulfate, nickel, nitric, nitrous and hydrochloric acids, ammonia, sulfur dioxide and nitrogen oxides). Mean levels for certain kinds of pollen and ozone were significantly higher in the Bronx than in Manhattan.

The study's large sample sizes resulted in statistical power to detect small mean differences as statistically significant, such that even some modest mean differences in analyte concentrations between the two sites were considered "significant." The largest relative differences were for ozone and pollen, where Bronx means exceeded Manhattan means by $30 \%$ to $70 \%$, depending on the analyte, and for ammonia, nitric oxide and nickel levels, where Manhattan means exceeded Bronx means by about $30 \%$ to $60 \%$. For all other analytes, the relative mean differences over the entire study period (percentage increase of the higher over the lower mean) were about $25 \%$ or less between the two sites, and in most cases were less than $10 \%$. Nearly half (10/21) of the statistically significant mean differences between the two sites over the entire study period were relative differences of about $10 \%$ or less.

Even though this study was not designed to address whether or not these two communities were meeting federal National Ambient Air Quality Standards (NAAQS), comparisons can be made to provide an assessment on the overall air quality. For $\mathrm{SO}_{2}, \mathrm{NO}_{2}$, and $\mathrm{PM}_{10}$, the values were well below the corresponding NAAQS levels in both communities, as were the 24 -hour average $\mathrm{PM}_{2.5}$ concentrations. However, the overall average $\mathrm{PM}_{2.5}$ measured concentrations- $14.5 \mu \mathrm{~g} / \mathrm{m}^{3}$ at the Bronx site and $16.6 \mu \mathrm{~g} / \mathrm{m}^{3}$ at the Manhattan sitewere both near the annual NAAQS level of $15 \mu \mathrm{~g} / \mathrm{m}^{3}$. For ozone, the eight-hour moving average exceeded the NAAQS level of 0.08 ppm five times in Bronx and three times in Manhattan over the course of the study, or less than $1 \%$ of the study days. These results cannot be used to evaluate compliance with federal air quality standards, since non-attainment of the NAAQS involves consideration of a longer measurement period over a larger region not restricted to these two communities. The US EPA currently considers the entire New York City metropolitan region (including the five New York City boroughs, plus adjacent counties in Long Island, the lower Hudson Valley, Connecticut and New Jersey) to be in non-attainment status for the ozone and fine particle NAAQS.

One possible source of the modest differences in air pollutant levels seen between the two sampling areas could be differences in the overall level of commercial and industrial activity. As an initial screening, we
attempted to assess this by counting the numbers of certain business types in the Bronx and Manhattan as reported in U.S. Census data. However, we were not able to determine whether Census business listings represented activities that actually contributed to air pollutant emissions in either borough. These listings are based on mailing addresses and in many cases could represent corporate offices or post office boxes. Also, the number of industrial facilities in an area does not necessarily imply a particular level of environmental chemical emissions. For example, air emissions from a single facility in Manhattan during 2000, as reported under the federal Toxic Release Inventory program, exceeded the total air emissions reported from five TRI facilities in the Bronx.

Other possible contributors to pollutant level differences in the two communities include traffic differences and the influence of more distant industrial emissions. Overall vehicle use does not appear to differ greatly in Manhattan and the Bronx, based on limited information regarding vehicle registrations and axle counts. However, local traffic patterns, such as commercial traffic and bus routes, could have a significant effect on pollutant differences between the two monitors. The industrial development in northern New Jersey, west of New York City, is substantial, and emissions related to those facilities could make different contributions to local air pollutant levels. However, data were not collected that allow those hypotheses to be evaluated.

Two analyte categories, ozone and pollen, tended toward higher average levels in the Bronx. Ozone is formed when nitrogen oxides (related to fuel combustion, especially vehicle emissions) and volatile organic compounds (VOCs) react together in the presence of sunlight. Mean nitrogen oxide levels were higher in Manhattan than in the Bronx during the study period. Although nitrogen oxides contribute to daytime ozone production, they can reduce ozone levels at night because of scavenging of oxygen atoms from ozone by nitric oxide to form nitrogen dioxide. This phenomenon, NO titration, could have the effect of decreasing daily average ozone levels in Manhattan below those in the Bronx. If this were true, overnight ozone and nitric oxide levels would be expected to decrease more and nitrogen dioxide levels would be expected to be proportionately higher overnight in Manhattan compared with the Bronx. However, hour-of-day trends for ozone, nitric oxide and nitrogen dioxide do not differ between the two study locations. Steady or increasing ozone levels in urban areas on weekends, despite reduced nitrogen oxide emissions on weekends, have been hypothesized to occur because of increased VOC-to- $\mathrm{NO}_{\mathrm{x}}$ ratios in a VOC-limited regime (e.g., Fujita et al. 2003). This is another mechanism that could be contributing to higher average ozone levels in the Bronx, where the reduced $\mathrm{NO}_{\mathrm{x}}$ levels could be causing increased VOC- $\mathrm{NO}_{\mathrm{x}}$ ratios.

The higher pollen levels in the Bronx may be a reflection of that community's larger areas of green space. They could also be an indication of sampling height differences or relative proximity of the samplers to wooded areas, giving wooded areas a stronger influence on the Bronx monitoring site than Central Park had on the Manhattan monitoring site.

An important limitation of the air monitoring data is that only a single monitoring site was operated in each borough. The monitors were sited to be representative of general area air quality. However, because of this, they may not reflect the effects of particular emissions sources, such as the Hunts Point wastewater treatment plant, on air quality in localized areas of the Bronx or Manhattan. The degree to which this may have affected the monitoring results is uncertain. However, the hour-of-day analysis (discussed below) suggests that local, ground-level traffic emissions did appear to be reflected in the monitoring results. The Bronx monitoring sites were located closer to ground level than the Manhattan site, and so could have been somewhat more influenced by local, street-level emissions sources.

The study was also limited to some degree by the choice of pollutants analyzed. Although the number of analytes was larger than in many previous studies, particular emissions sources may not have been reflected in the sampling results. For example, a very limited range of VOC pollutants was analyzed that may not have been particularly reflective of most industrial air emissions or odorous emissions from solid-waste or wastewater treatment facilities.

The extensive longitudinal database allows characterization of temporal trends in air contaminants on hourly, daily and seasonal scales. Several analytes that were measured on an hourly basis showed marked variation by hour of day, including both PM size fractions, elemental carbon, sulfur dioxide and nitrogen oxides. All of these contaminants had peak hourly concentrations occurring at 7-9 A.M. and in some cases also had a less distinct peak around 7-8 P.M. One-hour time-weighted ozone averages showed a reversed trend, with a midafternoon hourly peak and low hourly means during the morning, consistent with many previous studies (U.S. EPA 1996). Hourly temporal patterns were generally similar at the two sampling sites and could be related to traffic-volume patterns, changes in vertical mixing of air due to daytime heating and/or changes during the day in demand for heat and electricity and corresponding changes in emissions from power sources.

A tendency toward lower day-of-week means on Sundays, increasing through the week to Thursdays, was found for $\mathrm{PM}_{10}$, elemental carbon and $\mathrm{NO}_{\mathrm{x}}$. Ozone showed a slight trend toward higher weekend levels, as has been found previously in some U.S. locations (e.g., Fujita et al. 2003; Pun et al. 2003; Heuss et al. 2003). Except for ozone, these results might be hypothesized to reflect a buildup of traffic and perhaps industrial emissions during the work week. In some locations, higher weekend peak levels of ozone have been correlated to reduced $\mathrm{NO}_{\mathrm{x}}$ levels, relative to VOC levels, in areas where tropospheric ozone production is VOC-limited (e.g., Pun et al. 2003; Huess et al., 2003). However, the significance of these apparent trends for all analytes is unclear because the variance estimates for the day-of-week means are large, at least in part due to substantial seasonal variation. $\mathrm{PM}_{2.5}$, organic carbon and $\mathrm{SO}_{2}$ did not show a tendency toward day-of-week differences.

Many of the analytes (pollen, mold spores, ozone, $\mathrm{SO}_{2}$, nitrogen oxide, $\mathrm{HNO}_{2}, \mathrm{HNO}_{3}, \mathrm{HCl}, \mathrm{NH}_{3}, \mathrm{pH}$ and $\mathrm{SO}_{4}{ }^{2-}$ ) showed marked seasonal variations. For instance, the concentrations of $\mathrm{HNO}_{3}, \mathrm{HCl}, \mathrm{NH}_{3}$ and $\mathrm{SO}_{4}{ }^{2-}$ were higher during summer than in winter. The summer-winter ratios for $\mathrm{HNO}_{3}, \mathrm{HCl}$, and $\mathrm{SO}_{4}{ }^{2-}$ in Manhattan were 3.9, 3.1 and 1.9, respectively. The concentrations of $\mathrm{HNO}_{2}$, and $\mathrm{SO}_{2}$ were higher during winter than in summer, with summer-winter ratios in Manhattan 0.48 and 0.44 , respectively. Seasonal trends were similar at the Bronx sampling site.

Another indication of the similarity in pollutant trends in the two monitoring areas is the consistency observed in descriptive multivariate statistical results between the Bronx and Manhattan. In both areas, ozone levels tended to be strongly negatively associated with most other analytes, especially during the fall and winter. Similar patterns of positive associations among analytes were also seen in the two monitoring areas, with PM usually associated with sulfate and organic carbon; $\mathrm{SO}_{2}$, nitrogen oxides and elemental carbon formed another cluster of associated analytes.

Limited studies of urban air toxics have been conducted in some of the boroughs of New York City. The most extensive data have been collected on Staten Island. Ambient volatile organic compounds, benzo(a)pyrene, formaldehyde and metals were monitored in a joint EPA-New York-New Jersey study in 1987-1989. Nickel, manganese and iron were routinely detected in total suspended particulate samples and tended to range in concentration by approximately threefold between seasons and monitoring sites. Nickel was detected in more than $70 \%$ of the $\mathrm{PM}_{10}$ samples analyzed. The NYSDEC also conducted aldehyde sampling at a station in the North Bronx in summer 1995. Sampling duration of three hours in that study resulted in detectable levels of acetaldehyde, formaldehyde, and propionaldehyde in more than $99 \%$ of the samples collected.

Since 1992, NYSDEC has analyzed every-sixth-day total suspended particulates samples for five trace metals-arsenic, cadmium, mercury, nickel, and vanadium-from one monitoring station each in Brooklyn and Manhattan, two stations in Staten Island and three stations upstate. The trace metals data show regional differences in concentrations, with nickel being elevated in Manhattan compared with the other sites. Similarly, in the current study, the overall mean $\mathrm{PM}_{2.5}$ nickel level from Manhattan was higher than the overall Bronx mean. This consistency could suggest that particulate nickel is largely associated with the fine fraction. Or, nickel levels could be higher in all particulate fractions from Manhattan, compared with the other boroughs.

In conjunction with the implementation planning process for its mid-town Manhattan street-level $\mathrm{PM}_{10}$ site, which was classified moderate non-attainment in January 1994, NYSDEC has studied particulate characterization and $\mathrm{PM}_{10}$ emissions inventory data for this portion of Manhattan (NYSDEC 1995). Microscopic and chemical characterization of $\mathrm{PM}_{10}$ at the street-level Manhattan monitor indicated 53\% from diesel emissions, $13 \%$ ammonium nitrate, and $9 \%$ ammonium sulfates, with smaller contributions from road
dust, automobile emissions, sea salt, iron sources and residual fuel oil. The emissions inventory for the entire county indicates that $70 \%$ of $\mathrm{PM}_{10}$ emissions comes from area combustion sources, $19 \%$ from road dust, $6 \%$ from all vehicle emissions and smaller amounts from other sources. These results may indicate that streetlevel exposure to PM is more heavily influenced by vehicle emissions than emissions inventories would indicate. Although the current study results were obtained from rooftop monitors (four to seven stories above street level), the strong morning rush-hour peak in many of the analytes with hourly data suggests that vehicle emissions may be an important PM contributor up to at least 20 meters above ground level.

In the current study, we measured several $\mathrm{PM}_{2.5}$ components (elemental and organic carbon, sulfate, hydrogen ion and metals) and found that, on average, about $60 \%$ of FRM PM $_{2.5}$ measured at our sampling locations was accounted for by the simultaneously measured components. $\mathrm{PM}_{2.5}$ in our data set accounted for about $65 \%$ to $85 \%$ of $\mathrm{PM}_{10}$, depending on the measurement method used and the sampling location.

Data from previous studies suggest there are discernible differences in ambient concentrations of some air contaminants in urban areas, including New York City, for sites separated by as little as three to five miles. For example, Suh et al. (1995) collected 24-hour samples of sulfate, hydrogen ion and ammonia simultaneously at seven locations in Philadelphia and an upwind monitor during the summers of 1992 and 1993. Based on their assessment of spatial variation, they concluded that a single monitoring station was adequate for sulfate (consistent with the assumption that long-range transport is the dominant source), but multiple sites were necessary to determine local outdoor hydrogen ion concentrations, although variation in hydrogen ion over time was highly correlated across sites.

Goldstein and Landovitz (1977) found that for certain air contaminants (e.g., sulfur dioxide) there is a poor correlation among air monitoring sites within a metropolitan area. This suggests that the validity of exposure measures for certain contaminants can depend strongly on monitoring them within the community being studied. However, no study has determined precise limits on the area of validity of measurements for specific contaminants, and it is probably not possible to do so on a general basis. In the current study, and contrasting with Goldstein and Landovitz's results, between-site correlations were high for many of the analytes, including $\mathrm{PM}_{2.5}, \mathrm{PM}_{10}$, sulfate, $\mathrm{SO}_{2}$, nitrogen oxides, ozone, inorganic acids, ammonia and most bioaerosols. Between-site correlations within a large metropolitan area may depend on several factors, such as local topography, canyon effects, monitor height, prevailing meteorology, seasonality and local source strength.

Even when contaminant data are generally well correlated between monitoring sites, the strength of any correlation may not persist when monitored concentrations are at the high end of the range. The higher concentrations are those that are most likely to have health effects. For instance, an exploratory analysis of contemporaneous concentrations at pairs of NYSDEC ambient air monitoring sites in New York City, conducted prior to this study, found that temporal variation was strongly correlated among sites for ozone,
sulfur dioxide, nitrogen oxides and $\mathrm{PM}_{10}$. However, the temporal correlations between high contaminant levels (defined as upper quartile observations) were weaker, especially for ozone (unpublished data). Greater spatial heterogeneity in temporal patterns of high excursions in contaminant concentrations might contribute to spatial differences in acute asthma exacerbations, even if temporal patterns for all contaminant levels appear very similar across locations.

## Section 7

## CONCLUSIONS AND RECOMMENDATIONS

Ambient air quality measured with rooftop monitors at two locations in New York City found that, for most analytes, either the two sites did not differ or mean air levels were higher at the Manhattan location than at the Bronx location. Analyte measurements from both locations were subject to large temporal variations on hourly, daily and often seasonal time scales. When statistically different average pollutant levels were detected between the two locations, they differed by less than two fold. Average ozone and pollen levels tended to be higher in the Bronx, with mean differences of about $30 \%$ to $70 \%$ between the two sites. These results, representing approximately two years of hourly or daily observations on nearly three dozen analytes from two locations in New York City, provide a more detailed characterization of ambient air pollutants, especially particulate matter constituents, than has been previously reported for a large urban area. We recommend that future studies investigating ambient air pollutant exposures on an urban neighborhood scale collect additional data to better characterize spatial variability of ambient pollutants in urban areas, particularly for non-criteria pollutants.

## REFERENCES

Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for manganese and compounds. U.S. Department of Health and Human Services.
Agency for Toxic Substances and Disease Registry. 1993. Toxicological profile for chromium. U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for nickel - draft. U.S. Department of Health and Human Services.
Alexandersson R, Kolomodin-Hedman B, Hedenstierna G. 1982. Exposure to formaldehyde: effects on pulmonary function. Arch. Environ. Health 37:279-84.

Bari A, Dutkiewicz VA, Judd CD, Wilson LR, Luttinger D, Husain L. 2003a. Regional sources of particulate sulfate, $\mathrm{SO}_{2}, \mathrm{PM}_{2.5}, \mathrm{HCl}$ and $\mathrm{HNO}_{3}$ in New York, NY. Atmos. Environ. 37: 2837-44.
Bari A, Ferraro V, Wilson LR, Luttinger D, Husain L. 2003b. Measurements of gaseous HONO, $\mathrm{HNO}_{3}$, $\mathrm{SO}_{2}, \mathrm{HCl}, \mathrm{NH}_{3}$, particulate sulfate and $\mathrm{PM}_{2.5}$ in New York, NY. Atmos. Env. 37: 2825-35.

Basagana X, Sunyer J, Zock JP, Kogevinas M, Urrutia I, Maldonado JA, Almar E, Payo F, Anto JM. 2001. Incidence of asthma and its determinants among adults in Spain. Am. J. Respir. Crit. Care Med. 64(7):1133-7. Related Articles, Links

Boman BC, Forsberg AB, Jarvholm, BG. 2003. Adverse health effects from ambient air pollution in relation to residential wood combustion in modern society. Scand. J. Work Environ. Health 29(4): 251-60.

Brunekreef B, Holgate ST. 2002. Air pollution and health. Lancet 360:1233-42.
Burnett, RT, Dales RE, Raizienne ME, Krewski D., Summers, PW, Roberts GR, Raad-Young M, Dann T, Brook J. 1994. Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals. Environ. Res. 65:172-94.

Centers for Disease Control (CDC). 2006.Behavioral Risk Factor Surveillance System web site prevalence data. http://apps.nccd.cdc.gov/brfss/index.asp. Accessed May, 2006.
Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. 1996. Health effects of outdoor air pollution. Am. J. Respir. Crit. Care Med.153:3-50.

Dales RE, Cakmak S, Burnett RT, Judek S, Coates F, Brook J. 2000. Influence of ambient fungal spores on emergency visits for asthma to a regional children's hospital. Am. J. Resp. Crit. Care Med. 162:2087-90.

D’Amato GD, Liccardi G, D'Amato M, Cazzola M. 2002. Outdoor air pollution, climatic changes and allergic bronchial asthma. Eur. Resp. J. 20:763-76.

D’Augostino RB, Stevens MA (eds.). 1986. Goodness-of-Fit Techniques. Marcel Dekker, New York.
Delfino RJ, Coate BD, Zaiger RS, Seltzer JM, Street DH, Koutrakis P. 1996. Daily asthma severity in relation to personal ozone exposure and outdoor fungal spores. Am. J. Respir. Crit. Care Med.154:633-41.

Delfino RJ, Zeiger RS, Seltzer JM, Street DH, Matteucci RM, Anderson PR, Koutrakis P. 1997. The effect of outdoor fungal spore concentrations on daily asthma severity. Environ Health Perspect. 105(6):62235.

Diaz-Sanchez D, Tsien A, Fleming J, Saxon A. 1997. Combined diesel exhaust particulate and ragweed allergen challenge markedly enhances human in vivo nasal ragweed-specific IgE and skews cytokine production to a T helper cell 2-type pattern. J Immunol. 158(5):2406-13.
Dockery DW, Pope III CA. 1994. Acute respiratory effects of particulate air pollution. Annu. Rev. Pub. Health 15:107-32.

Feinman SE. 1988. Respiratory effects from formaldehyde. In Feinman SE (ed.), Formaldehyde sensitivity and toxicity, 135-48. CRC Press, Boca Raton.
Fujita EM, Stockwell WR, Campbell DE, Keislar RE, Lawson DR. 2003. Evolution of the magnitude and spatial extent of the weekend ozone effect in California’s South Coast Air Basin, 1981-2000. J. Air Waste. Manage. Assoc. 53(7):802-15.
Gavett SH, Koren HS. 2001. The role of particulate matter in exacerbations of atopic asthma. Int. Arch. Allergy Immunol. 124:109-12.

Gilbert R. 1987. Statistical methods for environmental pollution monitoring. John Wiley \& Sons, Inc., New York.
Goldstein IF, Landovitz L. 1977. Analysis of air pollution patterns in New York City: I. Can one station represent the large metropolitan area? Atmos. Environ. 11:47-52.
Goldstein IF, Dulberg EM. 1981. Air pollution and asthma: Search for a relationship. J. Air Pollut. Control Assoc. 31:370-76.

Gong H Jr, Linn WS, Shamoo DA, Anderson KR, Nugent CA, Clark KW, Lin AE. 1996. Effect of inhaled salmeterol on sulfur dioxide-induced bronchoconstriction in asthmatic subjects. Chest 110(5):122935.

Greenburg L, Field F, Reed JI, Erhardt CL. 1964. Asthma and temperature change. Arch. Environ. Health 8:642-47.
Harving H, Korsgaard J, Dahl R, Pedersen OF, Molhave L. 1986. Low concentrations of formaldehyde in bronchial asthma: a study of exposure under controlled conditions. Brit. Med. J. 293:310.

Heath SK, Koenig JQ, Morgan MS, Checkoway H, Hanley QS, Rebolledo V. 1994. Effects of sulfur dioxide exposure on African-American and Caucasian asthmatics. Environ Res. 66(1):1-11.
Higgins BG, Francis HC, Yates C, Warburton CJ, Fletcher AM, Pickering CA, Woodcock AA. 2000. Environmental exposure to air pollution and allergens and peak flow changes. Eur. Respir. J. 16(1):61-66.

Horstman D, Roger LJ, Kehrl H, Hazucha M. 1986. Airway sensitivity of asthmatics to sulfur dioxide. Toxicol. Ind. Health 2(3):289-98.

Horstman DH, Seal E Jr, Folinsbee LJ, Ives P, Roger LJ. 1988. The relationship between exposure duration and sulfur dioxide-induced bronchoconstriction in asthmatic subjects. Am. Ind. Hyg. Assoc. J. 49(1):38-47.

Heuss JM, Kahlbaum DF, Wolff GT. 2003. Weekday/weekend ozone differences: What can we learn from them? J. Air Waste. Manage. Assoc. 53(7):772-88.
Institute of Medicine (IOM). 2000. Clearing the Air. Asthma and Indoor Air Exposures. National Academy Press. Washington, DC.

Jamason PF, Kalkstein LS, Gergen PJ. 1997. A synoptic evaluation of asthma hospital admissions in New York City. Am J Respir Crit Care Med. 156(6):1781-8.
Jenkins HS, Devalia JL, Mister RL, Bevan AM, Rusznak C, Davies RJ. 1999. The effect of exposure to ozone and nitrogen dioxide on the airway response of atopic asthmatics to inhaled allergen: dose- and timedependent effects. Am J Respir Crit Care Med. 160(1):33-9.

Leikauf GD, Kline S, Albert RE, Baxter AC, Bernstein DI, Bernstein J, Buncher CR. 1995. Evaluation of a possible association of urban air toxics and asthma. Environ. Health Perspect. 103 (Suppl. 6):25371.

Lin S, Hwang SA, Pantea C, Kielb C, Fitzgerald E. 2004. Childhood asthma hospitalizations and ambient air sulfur dioxide concentrations in Bronx County, New York. Arch Environ Health. 59(5):266-75.

Linn WS, Avol EL, Shamoo DA, Venet TG, Anderson KR, Whynot JD, Hackney JD. 1984a. Asthmatics’ responses to 6-hr sulfur dioxide exposures on two successive days. Arch. Environ Health 39(4):31319.

Linn WS, Shamoo DA, Vinet TG, Spier CE, Valencia LM, Anzar UT, Hackney JD. 1984b. Combined effect of sulfur dioxide and cold in exercising asthmatics. Arch. Environ Health 39(5):339-46.
Lippmann M. 1998. Final Report. Re: adequacy of air monitoring for ATSDR, NYSDOH study of the association of neighborhood air pollution and asthma attacks. Submitted to the U.S. Centers for Disease Control and Prevention.

Mannino DM, Homa DM, Pertowski CA, Ashizawa A, Nixon LL, Johnson CA, Ball LB, Jack E, Kang DS. 1998. Surveillance for asthma--United States, 1960-1995. MMWR CDC Surveill Summ. 47(1):1-27.

Mannino DM, Homa DM, Akinbami LJ, Moorman JE, Gwynn C, Redd SC. 2002. Surveillance for asthma-United States, 1980-1999. MMWR Surveill Summ. 51(1):1-13.

Mardia KV, Kent JT, Bibby JM. 1979. Multivariate Analysis. Academic Press. San Diego, CA.
Neter J, Wasserman W, Kutner MH. 1990. Applied linear statistical models: Regression, analysis of variance and experimental designs. Third edition. Irwin, Inc. Homewood, IL.

New York State Department of Environmental Conservation (NYSDEC). 1994. New York State air quality report air monitoring system: Annual 1993. DAR-94-1.

New York State Department of Environmental Conservation (NYSDEC). 1995. New York State implementation plan: Inhalable particulate ( $\mathrm{PM}_{10}$ ).

Peden, D. 2002. Pollutants and asthma: Role of air toxics. Environ. Health Perspect. 110 (Suppl. 4):565-68.

Pun BK, Seigneur C, White W. 2003 Day-of-week behavior of atmospheric ozone in three U.S. cities. J. Air Waste Manage. Assoc. 53(7):789-801.

Ronchetti R, Villa MP, Barreto M, Rota R, Pagani J, Martella S, Falasca C, Paggo B, Guglielmi F, Ciofetta G. 2001. Is the increase in childhood asthma coming to an end? Findings from three surveys of schoolchildren in Rome, Italy. Eur. Resp. J. 17: 881-86.
Schappi GF, Taylor PE, Pain MC, Cameron PA, Dent AW, Staff IA, Suphioglu C. 1999. Concentrations of major grass group 5 allergens in pollen grains and atmospheric particles: Implications for hay fever and allergic asthma sufferers sensitized to grass pollen allergens. Clin. Exp. Allergy 29(5):633-41.

Schwela D. 2000. Air pollution and health in urban areas. Rev. Environ. Health 15(1-2):13-42.
Soriano JB, Anto JM, Sunyer J, Tobias A, Kogevinas M, Almar E, Muniozguren N, Sanchez JL, Palenciano L, Burney P. 1999. Risk of asthma in the general Spanish population attributable to specific immunoresponse. Spanish Group of the European Community Respiratory Health Survey. Int. J. Epidemiol. 28(4):728-34.
Suh HH, Allen GA, Koutrakis P, Burton RM. 1995. Spatial variation in acidic sulfate and ammonia concentrations within metropolitan Philadelphia. J. Air Waste Manage. Assoc. 45:442-52.

Takano H, Ichinose T, Miyabara Y, Yoshikawa T, Sagai M. 1998. Diesel exhaust particles enhance airway responsiveness following allergen exposure in mice. Immunopharmacol Immunotoxicol. 20(2):32936.

Thurston GD, Ito K, Kinney PL, Lippmann M. 1992. A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers. J. Expos. Anal. Environ. Epidem. 192:429-50.
U.S. Environmental Protection Agency (EPA). 1996. Air quality criteria for ozone and related photochemical oxidants.
U.S. Environmental Protection Agency (EPA). 1999. Particulate matter ( $\mathrm{PM}_{2.5}$ ) speciation guidance document. Third draft. U.S. EPA. Monitoring and Quality Assurance Group Emissions, Monitoring, and Analysis Division, Office of Air Quality Planning and Standards, Research Triangle Park, NC.
U.S. Environmental Protection Agency (EPA). 2002 [Health Assessment Document for Diesel Exhaust, section 3]

Wilkinson L. 1999 SYSTAT v. 9 manual. SPSS. Chicago, Il.
World Health Organization (WHO). 2000. Air Quality Guidelines, $2^{\text {nd }}$ edition. Geneva.

## AUTHORS AND ACKNOWLEDGEMENTS

Daniel Luttinger (NYSDOH) was the study's principal investigator, and Lloyd Wilson, Edward Fitzgerald, Laiquat Husain, Kenneth Aldous (NYSDOH), Phillip Galvin (NYSDEC), Larry Syzdek (private aerobiology consultant) and Patrick Kinney (Columbia University) were co-investigators. Additional study-design and statistical-analysis consultation was provided by John Hawley (NYSDOH), Frank Buckman (NYSDEC), Tracey Holloway (University of Michigan) and Ken Demerjian (NYSDEC). The study report was authored by Daniel Luttinger, Lloyd Wilson, Gregg Recer and Kim Mazor (NYSDOH). Field sampling staff included Dan Lince, Pat Palmer, Mike Rivara, Lloyd Wilson, Stephanie Selmer, James Kamara, Ellen Fitzsimmons, Dan Sharron and Stan House (NYSDOH), and Ed Marion, Mike Christophersen, Bob Murway, Bob Elburn and Frank Buckman (NYSDEC). Laboratory staff included Abdul Bari, Vincent Ferraro and Amarjit Narang (NYSDOH). Data analysis support staff included Jeff Hughes, Ying Wang, Valerie Haley, James Kamara, Kim Mazor, Karen Nolan, Dan Luttinger, Gregg Recer, Lloyd Wilson and Laiquat Husain (NYSDOH), and Phil Galvin and Frank Buckman (NYSDEC).

The study was funded with partial support from the Agency for Toxic Substances and Disease Registry (ATSDR) as part of Cooperative Agreement V50/ATV200002-11 and from the New York State Energy and Research Development Authority (NYSERDA).

TABLES

Table 1A. Population Characteristics of the Bronx and Manhattan Study Areas

| Population | Bronx Study Area | Manhattan Study Area |
| :--- | :---: | :---: |
| 2000 | 254,167 | 355,655 |
| 1990 | 234,478 | 343,006 |
| Percent Change | $+8 \%$ | $+4 \%$ |

Source: U.S. Bureau of Census

Table 1B. Housing Characteristics of the Bronx and Manhattan Study Areas

| Housing, 2000 | Bronx Study Area | Manhattan Study Area |
| :--- | :---: | :---: |
| Units | 85,807 | 215,016 |
| Occupied | 79,584 | 201,656 |
| Unoccupied | 6223 | 13,360 |
| Owner Occupied | 6750 | 42,532 |
| Renter Occupied | 72,834 | 159,124 |

Source: U.S. Bureau of Census.

Table 1C. Motor Vehicle Registrations in the Bronx and Manhattan Study Areas

| Vehicle Registrations, 2001 | Bronx County | Manhattan County |
| :--- | :---: | :---: |
| Total | 269,577 | 257,531 |
| Standard Series | 249,785 | 229,715 |
| Commercial | 9340 | 13,655 |
| Taxi | 5394 | 6722 |
| Bus | 624 | 230 |
| Other | 4434 | 7209 |

Source: New York State Department of Motor Vehicles.

Table 2. U.S. Census Bureau Zip Code Pattern

| Zip Code Business Patterns (1997 Sector Summary) |  |  |
| :--- | :---: | :---: |
| Total | Bronx Study Area | Manhattan Study Area |
| Agricultural Services, Forestry, Fishing | 3121 | 47,340 |
| Construction | 1 | 62 |
| Mining | 159 | 897 |
| Manufacturing | 1 | 16 |
| Transportation and Public Utilities | 185 | 4090 |
| Wholesale Trade | 402 | 1388 |
| Retail Trade | 876 | 8789 |
| Finance, Insurance, and Real Estate | 443 | 5909 |
| Services | 785 | 18,108 |
| Unclassified | 50 | 536 |

Table 3. Summary of Daily Average Concentrations for Particulate Matter

| Analyte | Overall Mean |  | \# of Seasons <br> Statistically <br> Greater M/B | Range of Seasonal <br> Differences |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx |  |  |
| $\mathrm{PM}_{2.5}(\mathrm{TEOM})^{*}$ | 16.2 | 15.3 | $6 / 0$ | $0.3-1.2$ |
| $\mathrm{PM}_{2.5}(\mathrm{FRM})^{*}$ | 16.6 | 14.5 | $5 / 0$ | $0.8-2.0$ |
| $\mathrm{PM}_{10}(\mathrm{TEOM})$ | 23.1 | 22.3 | $5 / 1$ | $-6.3-3.4$ |
| $\mathrm{PM}_{10}(\mathrm{FRM})^{*}$ | 22.0 | 20.9 | $1 / 0$ | $-0.2-3.0$ |

*Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units = micrograms per cubic meter $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx
${ }^{\dagger} \mathrm{PM}_{10}$ (FRM) was collected every six days
Table 4. Summary of Particle Counts in $\mathrm{PM}_{2.5}$ Fraction

| Analyte | Overall Mean $^{\mathrm{a}}$ |  | \# of Seasons <br> Statistically <br> Greater M/B | Range of Seasonal <br> Differences $^{\mathrm{c}}$ |  |
| :--- | :---: | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx | 1560780 | $1 / 1^{\ddagger}$ | $-450936-221627$ |
| Particle Counts | 1463152 |  |  |  |  |

${ }^{\text {a }}$ Units = count
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\mathrm{c}}$ Difference $=$ Manhattan - Bronx
${ }^{\ddagger}$ Total particle counts were not available for winter 1999, spring 1999, or summer 1999

Table 5. Summary of Daily Averages for pH , Sulfate, and Carbon in Particulate Matter $\left(\mathrm{PM}_{2.5}\right)$

| Analyte | Overall Mean $^{\mathrm{a}}$ |  | \# of Seasons <br> Statistically <br> Greater M/B | Range of Seasonal <br> Differences $^{\mathrm{c}}$ |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx |  |  |
| $\mathrm{pH}^{*}$ | 5.04 | 5.15 | $0 / 3$ | $-0.12--0.02$ |
| Sulfate* $_{\text {Organic Carbon }}$ | 4.0 | 3.6 | $4 / 0$ | $0.0-0.3$ |
| Elemental Carbon | 3.09 | 3.17 | $2 / 3$ | $-0.57-0.94$ |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{\mathrm{a}}$ Units = micrograms per cubic meter $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)($ except pH$)$
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx

Table 6. Summary of Daily Averages for Selected Metals in Particulate Matter ( $\mathrm{PM}_{2.5}$ )

| Analyte | Overall Mean $^{\text {a }}$ |  |  | \# of Seasons <br> Statistically <br> Greater M/B |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx | Range of Seasonal <br> Differences |  |
| Iron |  |  |  |  |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{\mathrm{a}}$ Units = nanograms per cubic meter ( $\mathrm{ng} / \mathrm{m}^{3}$ )
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx
Table 7. Summary of Daily Averages for Pollen

| Analyte | Overall Mean $^{\mathrm{a}}$ |  | \# of Seasons <br> Statistically <br> Greater M/B | Range of Seasonal <br> Differences $^{\mathrm{c}}$ |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx |  |  |
| Total Pollen | 13.17 | 22.32 | $0 / 4$ | $-41.72-0.28$ |
| Tree | 12.18 | 20.53 | $0 / 2$ | $-41.50-0.27$ |
| Ragweed* | 0.37 | 0.45 | $0 / 1$ | $-0.74-0.01$ |
| Grasses* $^{2}$ | 0.38 | 0.59 | $0 / 0$ | $-0.36-0.01$ |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{\mathrm{a}}$ Units $=\# / \mathrm{m}^{3}$
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx

Table 8. Summary of Daily Averages for Mold

| Analyte | Overall Mean ${ }^{\mathrm{a}}$ |  | \# of Seasons <br> Statistically <br> Greater M/B | Range of Seasonal <br> Differences $^{\mathrm{c}}$ |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx | $0 / 2$ | $-208.8-112.3$ |
| Total Mold | 490.3 | 447.8 | $1 / 2$ | $-101.5-99.6$ |
| Basidiospores | 186.0 | 184.0 | $0 / 1$ | $-17.1-3.4$ |
| Ascospores | 39.0 | 43.2 | $1 / 2$ | $-89.4-117.3$ |
| Mitospores | 259.9 | 212.5 | $1 / 2$ | $-83.7-108.0$ |
| Dark Mitospores | 254.1 | 208.1 | $0 / 1$ | $5.7-9.3$ |
| Non-Dark Mitospores | 5.8 | 4.4 | $0 / 2$ | $-204.8-111.6$ |
| Small Spores $(<10 \mu \mathrm{~m})$ | 470.4 | 427.8 | $0 / 0$ | $-17.7-0.4$ |
| Large Spores $(>10 \mu \mathrm{~m})^{*}$ | 12.5 | 9.9 |  | 0.9 |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{\mathrm{a}}$ Units $=\# / \mathrm{m}^{3}$
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx

Table 9. Summary of Daily Averages for Acetone and Selected Aldehydes

| Analyte | Overall Mean $^{\mathrm{a}}$ |  | \# of Seasons <br> Statistically <br> Greater M/B | Range of Seasonal <br> Differences $^{\mathrm{c}}$ |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx |  |  |
| Acetaldehyde | 2.7 | 2.5 | $4 / 1$ | $-1.0-0.5$ |
| Acetone | 6.9 | 6.8 | $3 / 2$ | $-2.6-1.2$ |
| Formaldehyde | 4.4 | 4.2 | $3 / 1$ | $-1.9-0.5$ |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units = micrograms per cubic meter $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx

Table 10. Summary of Daily Averages for Acidic and Basic Gases

| Analyte | Overall Mean |  |  | \# of Seasons <br> Statistically <br> Greater M/B |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx | Range of Seasonal <br> Differences ${ }^{\text {c }}$ |  |
| Hydrochloric Acid $(\mathrm{HCl})^{*}$ | 0.51 | 0.47 | $0 / 1^{\dagger}$ | $-0.16-0.09$ |
| Nitrous Acid $(\mathrm{HONO})^{*}$ | 3.21 | 3.06 | $3 / 0^{\dagger}$ | $0.14-0.50$ |
| Nitric Acid $\left(\mathrm{HNO}_{3}\right)^{*}$ | 1.74 | 1.11 | $2 / 0^{\dagger}$ | $0.02-0.50$ |
| Ammonia $\left(\mathrm{NH}_{3}\right)^{*}$ | 3.536 | 2.273 | $2 / 0^{\ddagger}$ | $0.551-1.485$ |
| Sulfur Dioxide $\left(\mathrm{SO}_{2}\right)^{*}$ | 26.4 | 25.8 | $2 / 0^{\dagger}$ | $1.0-3.8$ |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units = micrograms per cubic meter $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# seasons Manhattan < Bronx
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx
${ }^{\dagger}$ Gases were collected from 6/23/99 to 7/11/00
${ }^{\ddagger}$ Ammonia results were not available from 9/1/99 to 12/28/99 and from 5/17/00 to 7/11/00

Table 11. Summary of Daily Average Concentrations for U.S. EPA Criteria Pollutant Gases and Other Nitrogen Oxides

| Analyte | Overall Mean ${ }^{\text {a }}$ |  | \# of Seasons <br> Statistically <br> Greater M/B | Range of Seasonal <br> Differences |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx | $0 / 8$ | $-0.011--0.002$ |
| Ozone $\left(\mathrm{O}_{3}\right)^{*}$ | 0.012 | 0.016 | $5 / 0$ | $0.000-0.006$ |
| Sulfur Dioxide $\left(\mathrm{SO}_{2}\right)^{*}$ | 0.012 | 0.011 |  | $7 / 0^{\dagger}$ |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units = parts per million (ppm)
${ }^{\mathrm{b}}$ \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx
${ }^{\dagger}$ Nitrogen oxide results were not available for Bronx for winter 1999

Table 12. Summary of Daily Averages Concentrations for Particulate Matter: Comparison of the Two Bronx Monitoring Sites

| Analyte $^{\mathrm{a}}$ | Bronx Site A (1999) |  | Bronx Site B (2000) |  |
| :--- | :---: | :---: | :---: | :---: |
|  | $\frac{\text { Manhattan }^{\text {Bronx }}}{}$ | Mean <br> Difference | $\frac{\text { Manhattan }}{\text { Bronx }^{\mathrm{b}}}$ | Mean <br> Difference |
| $\mathrm{PM}_{2.5}$ (TEOM) | $15.9 / 15.2$ | $0.7^{*}$ | $15.5 / 14.8$ | $0.7^{*}$ |
| $\mathrm{PM}_{2.5}$ (FRM) | $15.2 / 14.3$ | 0.8 | $16.7 / 15.2$ | $1.6^{*}$ |
| $\mathrm{PM}_{10}$ (TEOM) | $21.3 / 22.3$ | -1.0 | $24.2 / 22.5$ | $1.7^{*}$ |
| $\mathrm{PM}_{10}$ (FRM) | $23.7 / 22.8$ | 0.9 | $21.8 / 21.9$ | -0.1 |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units $=$ micrograms per cubic meter $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)($ except pH$)$
${ }^{\mathrm{b}}$ Means are from paired data
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx
${ }^{\dagger} \mathrm{PM}_{10}$ (FRM) was collected every six days

Table 13. Summary of Daily Averages for pH , Sulfate, and Carbon in Particulate Matter ( $\mathrm{PM}_{2.5}$ ): Comparison of the Two Bronx Monitoring Sites

| Analyte ${ }^{\text {a }}$ | Bronx Site A (1999) |  | Bronx Site B (2000) |  |
| :---: | :---: | :---: | :---: | :---: |
|  | $\frac{\text { Manhattan }}{\text { Bron }^{\mathrm{b}}}$ | Mean Difference ${ }^{\text {c }}$ | $\frac{\text { Manhattan }}{\text { Bronx }^{\mathrm{b}}}$ | Mean <br> Difference ${ }^{\text {c }}$ |
| pH | 5.20 / 5.26 | -0.06 | 5.05 / 5.13 | -0.08* |
| Sulfate | 3.5 / 3.4 | 0.1* | 3.9 / 3.7 | 0.2* |
| Organic Carbon | 2.84 / 2.97 | -0.13 | 3.03 / 3.53 | -0.51* |
| Elemental Carbon | 1.58 / 1.44 | 0.14 | 1.26 / 1.12 | 0.14* |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units = micrograms per cubic meter $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)($ except pH$)$
${ }^{\mathrm{b}}$ Means are from paired data
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx

Table 14. Summary of Daily Averages for Selected Metals in Particulate Matter ( $\mathrm{PM}_{2.5}$ ): Comparison of the Two Bronx Monitoring Sites

| Analyte $^{\mathrm{a}}$ | Bronx Site A (1999) |  | Bronx Site B (2000) |  |
| :--- | :---: | :---: | :---: | :---: |
|  | $\frac{\text { Manhattan }}{\text { Bronx }^{\mathrm{b}}}$ | Mean <br> Difference | $\frac{\text { Manhattan }^{B^{\mathrm{b}}}}{}$ | Mean <br> Difference $^{\mathrm{c}}$ |
| Iron | $86 / 86$ | 0 | $51 / 64$ | -13 |
| Nickel | $20 / 16$ | 4 | $18 / 12$ | $6^{*}$ |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{\mathrm{a}}$ Units = nanograms per cubic meter ( $\mathrm{ng} / \mathrm{m}^{3}$ )
${ }^{\mathrm{b}}$ Means are from paired data
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx

Table 15. Summary of Daily Averages for Pollen and Mold: Comparison of the Two Bronx Monitoring Sites

| Analyte ${ }^{\text {a }}$ | Bronx Site A (1999) |  | Bronx Site B (2000) |  |
| :---: | :---: | :---: | :---: | :---: |
|  | $\frac{\text { Manhattan }}{\text { Bron }^{\mathrm{b}}}$ | Mean Difference ${ }^{\text {c }}$ | $\frac{\text { Manhattan }}{\text { Bron }^{\mathrm{b}}}$ | Mean Difference ${ }^{\text {c }}$ |
| Total Pollen | 11.8 / 16.7 | -4.9* | 32.6 / 52.7 | -20.1 |
| Tree | 11.4 / 16.0 | -4.6* | 32.2 / 52.1 | -19.9 |
| Ragweed | 0.0 / 0.0 | 0.0 | 0.0 / 0.0 | 0.0 |
| Grasses | 0.4 / 0.6 | -0.2 | 0.4 / 0.6 | -0.2* |
| Total Mold | 307.6 / 308.1 | -0.5 | $\begin{gathered} 336.8 / \\ 289.6 \end{gathered}$ | 47.2 |
| Basidiospores | 36.2 / 39.3 | -3.1 | $\begin{gathered} 146.1 / \\ 100.1 \end{gathered}$ | 46.0 |
| Ascospores | 36.8 / 38.7 | -1.8 | 27.7 / 29.2 | -1.5 |
| Mitospores | 230.6 / 228.1 | 2.5 | $\begin{gathered} 161.0 / \\ 158.5 \end{gathered}$ | 2.6 |
| Dark Mitospores | 228.2 / 225.5 | 2.7 | $\begin{gathered} 156.3 / \\ 154.5 \end{gathered}$ | 1.9 |
| Non-ark Mitospores | 2.5 / 2.6 | -0.2 | 4.7 / 4.0 | 0.7 |
| Small Spores ( $<10 \mu \mathrm{~g}$ ) | 293.2 / 292.3 | 0.8 | $\begin{gathered} 325.4 / \\ 278.0 \end{gathered}$ | 47.4 |
| Large Spores (> $10 \mu \mathrm{~g}$ ) | 9.9 / 12.9 | -3.0 | 6.6 / 6.8 | -0.2 |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{\mathrm{a}}$ Units $=\# / \mathrm{m}^{3}$
${ }^{\mathrm{b}}$ Means are from paired data
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx

Table 16. Summary of Daily Averages for Acetone and Selected Aldehydes: Comparison of the Two Bronx Monitoring Sites

| Analyte ${ }^{\text {a }}$ | Bronx Site A (1999) |  | Bronx Site B (2000) |  |
| :---: | :---: | :---: | :---: | :---: |
|  | $\frac{\text { Manhattan }}{\text { Bronx }^{\mathrm{b}}}$ | Mean Difference ${ }^{\text {c }}$ | $\frac{\text { Manhattan }}{\text { Bron }^{\mathrm{b}}}$ | Mean Difference ${ }^{\text {c }}$ |
| Acetaldehyde | 2.4 / 2.2 | 0.2 | 2.7 / 3.0 | -0.3 |
| Acetone | 7.7 / 8.6 | -0.9 | 6.5 / 6.3 | 0.2 |
| Formaldehyde | 4.1 / 3.8 | 0.4* | 4.1 / 4.8 | -0.8 |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units = micrograms per cubic meter $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$
${ }^{\mathrm{b}}$ Means are from paired data
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx

Table 17. Summary of Daily Averages for Acidic and Basic Gases: Comparison of the Two Bronx Monitoring Sites (June 23 to July 14)

| Analyte $^{\mathrm{a}}$ | Bronx Site A (1999) |  | Bronx Site B (2000) |  |
| :--- | :---: | :---: | :---: | :---: |
|  | $\frac{\text { Manhattan }}{\text { Bronx }}$ | Mean <br> Difference | $\frac{\text { Manhattan }}{\text { Bronx }}$ | Mean <br> Difference |
| ${\text { Hydrochloric Acid }(\mathrm{HCl})^{\dagger}}^{\dagger}$ | $1.30 / 1.21$ | 0.09 | $0.83 / 1.02$ | $-0.18^{*}$ |
| Nitrous Acid $(\mathrm{HONO})^{\dagger}$ | $1.33 / 1.06$ | 0.27 | $1.38 / 1.15$ | 0.23 |
| Nitric Acid $\left(\mathrm{HNO}_{3}\right)^{\dagger}$ | $3.91 / 3.53$ | $0.38^{*}$ | $3.32 / 3.25$ | 0.07 |
| Ammonia $\left(\mathrm{NH}_{3}\right)^{\dagger \ddagger}$ | $5.299 /$ | 0.551 | NA | NA |
| ${\text { Sulfur Dioxide }\left(\mathrm{SO}_{2}\right)^{\dagger}}^{4.748}$ |  | $4.7^{*}$ | $17.81 /$ | $2.0^{*}$ |
|  | $20.19 /$ |  | 15.77 |  |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units = micrograms per cubic meter $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$
${ }^{\mathrm{b}}$ Means are from paired data
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx
${ }^{\dagger}$ Gases were collected from 6/23/99 to 7/11/00
${ }^{\ddagger}$ Ammonia results were not available from 9/1/99 to 12/28/99 and from 5/17/00 to 7/11/00

Table 18. Summary of Daily Averages Concentrations for U.S. EPA Criteria Pollutant Gases and Other Nitrogen Oxides: Comparison of the Two Bronx Monitoring Sites

| Analyte ${ }^{\text {a }}$ | Bronx Site A (1999) |  | Bronx Site B (2000) |  |
| :---: | :---: | :---: | :---: | :---: |
|  | $\frac{\text { Manhattan }}{\text { Bron }^{\mathrm{b}}}$ | Mean Difference ${ }^{\text {c }}$ | $\frac{\text { Manhattan }}{\text { Bron }^{\mathrm{b}}}$ | Mean <br> Difference ${ }^{\text {c }}$ |
| Ozone ( $\mathrm{O}_{3}$ ) | 0.016 / 0.022 | -0.006* | $\begin{gathered} \hline 0.012 / \\ 0.017 \end{gathered}$ | -0.005* |
| Sulfur Dioxide ( $\mathrm{SO}_{2}$ ) | 0.014 / 0.010 | 0.004* | $\begin{gathered} 0.013 / \\ 0.012 \end{gathered}$ | 0.001* |
| Nitrogen Dioxide ( $\left.\mathrm{NO}_{2}\right)^{\dagger}$ | 0.037 / 0.027 | 0.010* | $\begin{gathered} 0.038 / \\ 0.033 \end{gathered}$ | 0.005* |
| Nitric Oxide (NO) ${ }^{\dagger}$ | 0.017 / 0.009 | 0.008* | $\begin{gathered} 0.030 / \\ 0.024 \end{gathered}$ | 0.007* |
| Nitrogen Oxides ( $\left.\mathrm{NO}_{\mathrm{X}}\right)^{\dagger}$ | 0.054 / 0.037 | 0.017* | $\begin{gathered} 0.067 / \\ 0.057 \end{gathered}$ | 0.010* |

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
${ }^{a}$ Units = parts per million (ppm)
${ }^{\mathrm{b}}$ Means are from paired data
${ }^{\text {c }}$ Difference $=$ Manhattan - Bronx
${ }^{\dagger}$ Nitrogen oxides were not available for Bronx for winter 1999

Table 19. Summary of Comparison of Daily Maximum Concentrations

| Analyte | Overall Mean |  | \# of Seasons <br> Statistically <br> Greater M/B | Range of Seasonal <br> Differences |
| :--- | :---: | :---: | :---: | :---: |
|  | Manhattan | Bronx |  |  |
| $\mathrm{PM}_{2.5}(\mathrm{TEOM})\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | 27.5 | 27.3 | $2 / 1$ | $-1.47-2.25$ |
| $\mathrm{PM}_{10}(\mathrm{TEOM})\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | 38.4 | 37.3 | $2 / 2$ | $-10.72-6.32$ |
| Total Particles $(\#)^{*}$ | 2294848 | 2696751 | $0 / 2^{\ddagger}$ | $-937376--44048$ |
| Organic Carbon $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | 3.71 | 3.66 | $2 / 2$ | $-0.378-0.944$ |
| Elemental Carbon $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | 2.04 | 1.94 | $1 / 1$ | $-0.254-0.354$ |
| Ozone $\left(\mathrm{O}_{3}\right)-1$ hour $(\mathrm{ppm})^{*}$ | 0.028 | 0.033 | $0 / 8$ | $0.016-0.005$ |
| Ozone $\left(\mathrm{O}_{3}\right)-8$ hour $(\mathrm{ppm})^{*}$ | 0.021 | 0.027 | $0 / 8$ | $0.012-0.004$ |
| Sulfur Dioxide $\left(\mathrm{SO}_{2}\right)(\mathrm{ppm})$ | 0.024 | 0.023 | $2 / 0$ | $-0.002-0.004$ |
| Nitrogen Dioxide $\left(\mathrm{NO}_{2}\right)(\mathrm{ppm})^{*}$ | 0.050 | 0.049 | $1 / 0^{\dagger}$ | $0.000-0.014$ |
| Nitric Oxide $(\mathrm{NO})(\mathrm{ppm})$ | 0.083 | 0.075 | $1 / 0^{\dagger}$ | $-0.004-0.021$ |
| Nitrogen Oxides $\left(\mathrm{NO}_{\mathrm{x}}\right)(\mathrm{ppm})$ | 0.127 | 0.119 | $1 / 0^{\dagger}$ | $0.004-0.032$ |

Elemental and organic carbon are based on 3-hour concentrations; the rest are based on 1-hour concentrations.

* Significantly different over entire study period ( $\mathrm{P} \leq 0.05$ )
a \# Manhattan > Bronx / \# Manhattan < Bronx
${ }^{\mathrm{b}}$ Difference $=$ Manhattan - Bronx
${ }^{\dagger}$ Nitrogen oxide results were not available for Bronx for winter 1999
₹ Total particle counts were not available for winter 1999, spring 1999, or summer 1999

Table 20. Correlations (Pearson r) between Bronx and Manhattan Monitoring Sites for the Same Air Contaminants at the Two Sites

| Particles |  | Gases |  |
| :---: | :---: | :---: | :---: |
| $\mathrm{PM}_{2.5}$ (TEOM) | 0.97 | Acetaldehyde* | 0.81 |
| $\mathrm{PM}_{2.5}$ (FRM) | 0.90 | Acetone* | 0.23 |
| $\mathrm{PM}_{10}$ (TEOM) | 0.92 | Formaldehyde* | 0.80 |
| $\mathrm{PM}_{10}$ (FRM) | 0.96 | Ozone ( $\mathrm{O}_{3}$ ) | 0.92 |
| Particle Count | 0.22 | Nitrogen Oxides ( $\mathrm{NO}_{\mathrm{x}}$ ) | 0.87 |
| pH | 0.69 | Nitric Oxide (NO) | 0.88 |
| Sulfate | 0.96 | Nitrogen Dioxide ( $\mathrm{NO}_{2}$ ) | 0.77 |
| Organic Carbon | 0.62 | Sulfur Dioxide ( $\mathrm{SO}_{2}$ ) | 0.90 |
| Elemental Carbon | 0.77 | Hydrochloric Acid (HCl) | 0.84 |
| Iron | 0.37 | Nitrous Acid (HONO) | 0.84 |
| Nickel | 0.38 | Nitric Acid ( $\mathrm{HNO}_{3}$ ) | 0.93 |
| Total Pollen | 0.98 | Ammonia ( $\mathrm{NH}_{3}$ ) | 0.92 |
| Tree Pollen | 0.98 | Sulfur Dioxide ( $\mathrm{SO}_{2}$ ) (denuder) | 0.90 |
| Ragweed | 0.86 |  |  |
| Grasses | 0.75 | Meteorological |  |
| Total Mold | 0.84 | Temperature | 1.00 |
| Basidiospores | 0.71 | Relative Humidity | 0.98 |
| Ascospores | 0.68 |  |  |
| Mitospores | 0.87 |  |  |
| Mitospores (Dark) | 0.88 |  |  |
| Mitospores (Non-Dark) | 0.05 |  |  |
| Small Spores ( $<10$ um) | 0.83 |  |  |
| Large Spores (> 10 um) | 0.79 |  |  |

*Correlations between sites were calculated excluding data from April 20 to April 30, 2000. If these dates are included, the correlations between sites for acetaldehyde, acetone, and formaldehyde would be $0.66,0.21$, and 0.19 , respectively.

Table 21. -Correlations (Pearson r) between Daily Average and Daily Maximum

| Pollutant | Bronx | Manhattan |
| :--- | :---: | :---: |
| Organic Carbon $\left(\mathrm{ug} / \mathrm{m}^{3}\right.$ ) | 0.91 | 0.90 |
| Elemental Carbon $\left(\mathrm{ug} / \mathrm{m}^{3}\right)$ | 0.93 | 0.93 |
| Ozone - (1 hr max) (ppm) | 0.90 | 0.92 |
| Ozone - (8 hr max) (ppm) | 0.94 | 0.95 |
| $\mathrm{NO}_{\mathrm{Xx}}$ (ppm) | 0.89 | 0.89 |
| NO (ppm) | 0.89 | 0.88 |
| $\mathrm{NO}_{2}$ (ppm) | 0.86 | 0.85 |
| $\mathrm{SO}_{2}$ (ppm) | 0.88 | 0.85 |
| $\mathrm{PM}_{2.5}$ (TEOM) (ug/m ${ }^{3}$ ) | 0.88 | 0.90 |
| $\mathrm{PM}_{10}$ (TEOM) (ug/m ${ }^{3}$ ) | 0.90 | 0.81 |
| Total Particulates (\#) | 0.68 | 0.64 |
| Temperature (deg F) | 0.98 | 0.99 |
| Relative Humidity (\%) | 0.89 | 0.89 |

Table 22. Correlations (Pearson r) between Bronx and Manhattan Monitoring Sites for the Same Air
Contaminants at the Two Sites, Stratified by Year, for Comparable Date Ranges between the Two Bronx sites
(January 1-July 14)

| Pollutant | 1999 Pearson r | 2000 Pearson r |
| :---: | :---: | :---: |
| pH | 0.56 | 0.77 |
| Sulfate | 0.96 | 0.98 |
| Formaldehyde | 0.79 | 0.81 |
| Acetaldehyde | 0.61 | 0.86 |
| Acetone | 0.029 | 0.66 |
| Organic carbon | 0.80 | 0.86 |
| Elemental carbon | 0.74 | 0.76 |
| Nitric oxide (NO) | 0.55 | 0.91 |
| Nitrogen oxides ( $\mathrm{NO}_{\mathrm{x}}$ ) | 0.41 | 0.92 |
| Nitrogen dioxide ( $\mathrm{NO}_{2}$ ) | 0.40 | 0.88 |
| Ozone ( $\mathrm{O}_{3}$ ) | 0.85 | 0.93 |
| Sulfur dioxide ( $\mathrm{SO}_{2}$ ) | 0.87 | 0.94 |
| $\mathrm{PM}_{2.5}$ (TEOM) | 0.96 | 0.96 |
| $\mathrm{PM}_{2.5}$ (FRM) | 0.34 | 0.99 |
| $\mathrm{PM}_{10}$ (TEOM) | 0.92 | 0.97 |
| $\mathrm{PM}_{10}$ (FRM) | 0.99 | 0.95 |
| Hydrochloric acid (HCl) | 0.95 | 0.79 |
| Nitrous acid (HONO) | 0.92 | 0.79 |
| Nitric acid ( $\mathrm{HNO}_{3}$ ) | 0.98 | 0.91 |
| Sulfur dioxide ( $\mathrm{SO}_{2}$ ) (denuder) | 0.72 | 0.88 |
| Ammonia (NH3) | 0.61 | 0.92 |
| Iron (Fe) | 0.30 | 0.31 |
| Nickel (Ni) | 0.29 | 0.58 |
| Total pollen | 0.89 | 0.98 |
| Tree pollen | 0.89 | 0.98 |
| Ragweed pollen | 0.023 | 0.0075 |
| Grass pollen | 0.81 | 0.85 |
| Total mold | 0.92 | 0.73 |
| Basidiomycetes | 0.74 | 0.49 |
| Ascomycetes | 0.55 | 0.70 |
| Mitospores | 0.94 | 0.82 |
| Dark mitospores | 0.94 | 0.83 |
| Non-dark mMitospores | 0.014 | 0.19 |
| Small spores | 0.91 | 0.72 |
| Large spores | 0.76 | 0.89 |
| Total particle number | - | 0.049 |
| Temperature | 1.0 | 1.0 |
| Relative humidity | 0.99 | 0.97 |

FIGURES

Figure 1. Bronx Sampling Locations


Figure 2. Manhattan Sampling Location


## Figure 3. (A) Daily averages and (B) difference in daily averages for PM2.5 (TEOM)




## Figure 4. (A) Daily averages and (B) difference in daily averages for PM10 (TEOM)

Panel A



## Figure 5. (A) Daily vererges and (B) difference in daily averages for particle count



Figure 6. (A) Daily averages and (B) difference in dally averages for pH


# Figure 7. (A) Daily averages and (B) difference in dally averages for sulfate 

Panel A



## Figure 8. (A) Daily averages and (B) difference in daily averages for organic carbon



# Figure 9. (A) Daily averages and (B) difference in dalily averages for elemental carbon 




Figure 10. (A) Daily averages and (B) difference in dally averages for iron
Panel A



## Figure 11. (A) Daily averages and (B) difference in daily averages for nickel

## Panel A



## Panel B



## Figure 12. (A) Daily averages and (B) difference in dally averages for total pollen

Panel A


## Panel B



## Figure 13. (A) Daily averages and (B) difference in daily averages for tree pollen

## Panel A




## Figure 14. (A) Daily averages and (B) difference in daily averages for grass pollen

## Panel A



## Panel B



## Figure 15. (A) Daily averages and (B) difference in daily averages for ragweed pollen



## Panel B



## Figure 16. (A) Daily averages and (B) difference in daily averages for total mold

Panel A



## Figure 17. (A) Daily averages and (B) difference in daily averages for basidospores




## Figure 18. (A) Daily averages and (B) difference in daily averages for ascospores



## Panel B



Figure 19. (A) Daily averages and (B) difference in dally averages for mitospores
Panel A



Figure 20. (A) Daily averages and (B) difference in dally averages for dark mitospores



## Figure 21. (A) Daily averages and (B) difference in daily averages for non-dark mitospores



## Figure 22. (A) Daily averages and (B) difference in daily averages for mold spores <10um



## Figure 23. (A) Daily averages and (B) difference in daily averages for mold spores >10um



## Figure 24. (A) Daily averages and (B) difference in dally averages for acetone

Panel A


## Panel B



## Figure 25. (A) Daily averages and (B) difference in daily averages for acetaldehyde

Panel A


## Panel B



## Figure 26. (A) Daily averages and (B) difference in dalily averages for formaldehyde

## Panel A



## Panel B



Figure 27. (A) Daily averages and (B) difiference in daily averages for hydrochloric acid


## Panel B



## Figure 28. (A) Daily averages and (B) difiference in dally averages for nitrous acid

Panel A


## Panel B



## Figure 29. (A) Dally averages and (B) difference in dalily averages for nitric acid

Panel A



## Figure 30. (A) Daily averages and (B) difference in daily averages for ammonia

## Panel A




## Figure 31. (A) Dally averages and (B) difference in daily averages for ozone

Panel A


## Panel B



## Figure 32. (A) Daily averages and (B) difference in dalily averages for sulfur dioxide




## Figure 33. (A) Daily averages and (B) difference in daily averages for nitrogen dioxide



Figure 34. (A) Daily averages and (B) difference in dalily averages for nitrogen oxide



# Figure 35. (A) Daily averages and (B) difference in dally averages for nitrogen oxides 



Figure 36. Multidimensional scaling results for (A) Bronx and (B) Manhattan
air-monitoring data. Results for all seasons combined.


[^1]Figure 37. Multidimensional scaling results for (A) Bronx and (B) Manhattan
air-monitoring data. Results for January - March.


[^2]Figure 38. Multidimensional scaling results for (A) Bronx and (B) Manhattan
air-monitoring data. Results for April - June.


[^3]Figure 39. Multidimensional scaling results for (A) Bronx and (B) Manhattan
air-monitoring data. Results for July - September.


[^4]Figure 40. Multidimensional scaling results for (A) Bronx and (B) Manhattan
air-monitoring data. Results for October - December.


[^5]Figure 41. Hierarchical clustering results for (A) Bronx and (B) Manhattan air-monitoring data. Results for all seasons combined.


Figure 42. Hierarchical clustering results for (A) Bronx and (B) Manhattan air-monitoring data. Results for January - March.


Figure 43. Hierarchical clustering results for (A) Bronx and (B) Manhattan air-monitoring data. Results for April - June.


Figure 44. Hierarchical clustering results for (A) Bronx and (B) Manhattan air-monitoring data. Results for July - September.


Figure 45. Hierarchical clustering results for (A) Bronx and (B) Manhattan air-monitoring data. Results for October - December.












عu/\#
















APPENDICES

## APPENDIX 1. DETAILS OF ANALYTICAL AND STATISTICAL METHODS

## QA/QC Protocols

The quality assurance and quality control measures instituted for this sampling program followed standard laboratory and field practices for calibrations, running blanks, flow audits, servicing of equipment, etc. The schedule for performing the various QA/QC measures was at least as rigorous as that required in EPA protocols; where no EPA protocol existed, the schedule was as rigorous as the most widely accepted protocol. A list of the various approved methods and associated protocols used for each of the measurements is provided in Table A1.

Table A1. Measurement Technologies and Associated Protocols

| Measurement Technology/Field Instrument | EPA-Approved Method/Protocol |
| :---: | :---: |
| Acid Aerosols, Ammonia and Acid Gases | EPA Method IO-4.2 |
| Aldehydes | EPA Method TO-11 |
| Elemental Carbon, Organic Carbon, Total Carbon | Rupprecht and Patashnick 5400 Series Carbon analyzer |
| FRM10 | Wedding \&Assoc PM10 High Vol Sampler RFPS-1087062 |
| FRM2.5 | Rupprecht and Patashnick Partisol Plus Model 2025 RFPS-0498-118 |
| Metals | Inductively Couple Plasma/Mass Spectrometry/ <br> Swami et al (2001) Journal of Analytical Chemistry (2001) 369:63-70 |
| Molds and Pollen | Burkard Bioaerosol Sampler/No EPA Protocol Issued |
| $\mathrm{NO} / \mathrm{NO}_{2} / \mathrm{NO}_{\mathrm{x}}$ | Thermo Environmental Instruments Model 42 EPA Equivalence Number (RFNA-1289-074) |
| Ozone | Thermo Environmental Instruments Model -49, EPA Equivalence Number (EQOA-0880-047) |
| Particle Number | TSI Inc. Model 1022 Condensation Particle Counter |
| $\mathrm{PM}_{10}$ (particulate matter 10 microns or less) | Rupprecht and Patashnick TEOM Particulate Analyzer EPA Equivalence Number (EQPM-10900079) |
| $\mathrm{PM}_{2.5}$ (particulate matter 2.5 microns or less) | Rupprecht and Patashnick TEOM Particulate Analyzer EPA Equivalence Number (EQPM-10900079) |
| $\mathrm{SO}_{2}$ | Thermo Environmental Instruments Model $43 \mathrm{C} \mathrm{SO}_{2}$ <br> Pulsed Fluoresence.Analyzer <br> EPA Equivalence Number (EQSA-0486-060)_ |

Our study implementation required staff to travel every Wednesday from Albany to New York City to collect samples, download data, and service equipment. Every piece of equipment associated with the study was reviewed and serviced to make sure that it was performing to pre-established QA/QC standards. All of the self-diagnostics tools in the various pieces of equipment were reviewed. After being downloaded, the data were reviewed to see if any noticeable issues could be identified. All flow audits were performed at least as frequently as required by EPA protocols and manufacturers' recommendations with a NIST traceable flow meter. All of the work required was documented on field forms as well as many of the parameters from the self-diagnostics. At the conclusion of each sampling event on Wednesday, a supervisor reviewed the work documented on each field form.

Because the monitoring stations were also part of the DEC air monitoring network, DEC staff were on-site more frequently than once a week. They serviced the $\mathrm{NO}_{x}, \mathrm{SO}_{2}$ and ozone meters as required by EPA. DEC staff also reported to us any problems with the additional equipment, and staff were then deployed to make the appropriate corrections.

More detail on the methodology used for each measurement appears in the narrative for each analyte.

## Analytical Methods

$P M_{10}$ and $P M_{2.5}$
Two TEOM ${ }^{\circledR}$ Series 1400a Ambient Particulate Monitors (Rupprecht \& Patashnick Co., Inc., Albany, NY) were deployed at each location, with one unit measuring $\mathrm{PM}_{10}$ and the other measuring $\mathrm{PM}_{2.5}$. The TEOM ${ }^{\circledR}$ Series 1400a was used to measure particulate mass concentrations continuously. The instrument incorporates the patented tapered element oscillating microbalance (TEOM), a microweighing technology. Using a choice of sample inlets (either inertial or cyclonic), the same hardware can be configured to measure either $\mathrm{PM}_{10}$ or $\mathrm{PM}_{2.5}$. This microprocessorbased unit provides internal data storage and advanced analog and serial data input/output capabilities. The TEOM ${ }^{\circledR}$ Series 1400a monitor has received the EPA PM ${ }_{10}$ equivalency approval EQPM-1090-079. PM $_{2.5}$ measurements are within the context of a EPA-correlated acceptable continuous monitor (40 CFR 58).

The Series 1400a monitor incorporates an inertial balance that directly measures the mass collected on an exchangeable Teflon ${ }^{\circledR}$-coated borosilicate glass filter cartridge by monitoring the corresponding frequency changes of a tapered element. The sample flow passes through the filter, where particulate matter collects, and then continues through the hollow tapered element on its way to an active volumetric flow control system and vacuum pump. Active volumetric flow control is maintained by mass flow controllers whose set points are constantly adjusted in accordance with the measured ambient temperature and pressure. Both the mass and the flow rate measurements are verifiable using NIST-traceable standards. R\&P $\mathrm{PM}_{10}$ and Teflon ${ }^{\circledR}$ coated $\mathrm{PM}_{2.5}$ size-selective inlets were used for particle cutoff. Sample inlet flow was $16.7 \mathrm{l} / \mathrm{min}$, with the main flow rate through the sensor unit maintained at 3.0 $1 / \mathrm{min}$. Sample stream temperature was heated to $50^{\circ} \mathrm{C}$, and the filter unit was held at $50^{\circ} \mathrm{C}$ to prevent condensation. A measure of change in the mass concentration was made every two seconds and used to calculate hourly averages.

Data were logged by the instruments and downloaded every Wednesday by project staff. Sample filters were exchanged when the filter's percent loading (capacity) reached $75 \%$ or greater, which was about every three weeks. Approximately every two months, inlet heads were either cleaned on-site or replaced with clean heads. TEOM ${ }^{\circledR}$ units were kept in temperature- controlled rooftop enclosures. A supplemental ACCU system was attached to the $\mathrm{PM}_{2.5}$ units at each location (described below).

FRM 10 and 2.5

## Particle Number

The Model 3022A Condensation Particle Counter (TSI Inc., Shoreview, MN) was used to measure the number of airborne particles between 0.007 and 2.5 micrometers in diameter. This instrument detects and counts particles with an optical detector. The butanol vapor is introduced into the air stream and condenses on particulates. This condensation enlarges the particle so that it can be measured with the optical detector. Approximate sampling flow was $300 \mathrm{~cm}^{3} / \mathrm{min}$. Data were logged by the instrument at one-minute intervals and downloaded once per week. Maintenance of the instrument included weekly draining of the interior butanol reservoir, as well as replacement of old butanol to prevent interference due to saturation of the reservoir wick by water. Wicks were replaced at sixmonth intervals.

## Organic and Elemental Carbon

A Series 5400 Ambient Carbon Particulate Monitor (Rupprecht \& Patashnick Co., Inc., Albany, NY) was used to measure organic and elemental carbon. The instrument uses a direct thermal- $\mathrm{CO}_{2}$ measurement to provide an indirect measure of the amount of carbon in the collected particulate. Outdoor air was drawn from the glass manifold (described earlier) at 16.7 lpm through a Teflon ${ }^{\circledR}$ coated, $\mathrm{PM}_{2.5}$ size-selective inlet. The particulate was collected for three hours on a filter, which was then heated. The instruments were programmed to heat the filter to 250, 340, 550 and $750^{\circ} \mathrm{C}$. The fraction volatilized or oxidized to $\mathrm{CO}_{2}$ at $250^{\circ} \mathrm{C}$ is considered the volatile organic fraction. The semi-volatile organic fraction is oxidized at $340^{\circ} \mathrm{C}$, and the elemental carbon is the difference in the amount oxidized to $\mathrm{CO}_{2}$ at $750^{\circ} \mathrm{C}$ minus that oxidized to $\mathrm{CO}_{2} 340^{\circ} \mathrm{C}$. Data were logged by the instrument and downloaded weekly.

EPA's Environmental Technology Verification Program reviewed R\&P’s 5400 Carbon Analyzer in 2000-2001 and issued a verification statement, which reads, in part,

The program evaluates the performance of innovative technologies by developing test plans that are responsive to the needs of stakeholders, conducting field or laboratory tests (as appropriate), collecting and analyzing data, and preparing peer-reviewed reports. All evaluations are conducted in accordance with rigorous quality assurance protocols to ensure that data of known and adequate quality are generated and that the results are defensible.

Field testing was conducted in two phases. The first took place at the DOE National Energy Technology Laboratory in Pittsburgh, from August 1 through September 1, 2000. The second phase was at the California Air Resources Board's ambient air monitoring station in Fresno from December 18, 2000, through January 17, 2001. Performance characteristics verified included inter-unit precision, agreement with and correlation to time-integrated reference methods, effect of meteorological conditions, and influence of precursor gases. OC, EC, and TC results from the 5400 were compared with laboratory thermal/optical reflectance (TOR) analysis of filter-based reference samples.

## Technological Description

See report at http://www.epa.gov/etv/verifications/vcenter1-3.html.

## Verification of Performance

Inter-unit precision
PHASE I RESULTS

| Linear Regression | Organic Carbon | Elemental Carbon | Total Carbon |
| :--- | :---: | :---: | :---: |
| Hourly Average | $(\mathrm{OC})$ | $(\mathrm{EC})$ | $(\mathrm{TC})$ |
| r2 | 0.94 | 0.93 | 0.95 |
| Slope (95\% C.I.) | $1.063(0.021)$ | $1.037(0.022)$ | $1.069(0.020)$ |
| Linear Regression 24-hr | Organic Carbon | Elemental Carbon | Total Carbon |
| Average | $(\mathrm{OC})$ | $(\mathrm{EC})$ | $(\mathrm{TC})$ |
| r2 | 0.97 | 0.94 | 0.97 |
| Slope (95\% C.I.) | $1.094(0.081)$ | $1.038(0.113)$ | $1.098(0.088)$ |

## PHASE II RESULTS

| Linear Regression Hourly | Organic Carbon | Elemental Carbon | Total Carbon |
| :--- | :---: | :---: | :---: |
| Average | $(\mathrm{OC})$ | $(\mathrm{EC})$ | $(\mathrm{TC})$ |
| r2 | 0.94 | 0.92 | 0.86 |
| Slope (95\% C.I.) | $0.971(0.019)$ | $1.029(0.024)$ | $1.074(0.035)$ |
| Linear Regression 24-hr | Organic Carbon | Elemental Carbon | Total Carbon |
| Average | $(\mathrm{OC})$ | $(\mathrm{EC})$ | $(\mathrm{TC})$ |
| r2 | $>0.97$ | $>0.97$ | $>0.97$ |
| Slope (95\% C.I.) | $1.027(0.072)$ | $1.164(0.083)$ | $1.090(0.070)$ |

Comparability and Predictability
In both Phase I and Phase II, 24-hour averages from the 5400 showed a negative bias when compared with OC, EC, and TC reference measurements. Phase I regression slopes were below 0.4 for the OC, EC, and TC, and r2 values were between 0.43 and 0.52. Phase II regression slopes fell between 0.2 and 0.7 and between 0.2 and 0.9 for monitors 1 and 2, respectively, for all three carbon fractions, and r2 values were between 0.65 and 0.90 .

## Meteorological Effects

For Phase I, the multivariable model ascribed a small but significant effect on the 5400's readings relative to the reference for vertical and horizontal wind speed, wind direction, and ambient air temp at 2 and 10 meters. In general, the combined effect of these parameters was small. (For example, the model predicts a Phase I average OC value that differs from the linear regression model by about 5\%.) For Phase II, small but significant effects were ascribed to wind speed, wind direction, standard deviation of wind direction, solar radiation, relative humidity, and barometric pressure.

Influence of Precursor Gases
For Phase I, the model ascribed statistical influence to $\mathrm{O}_{3}, \mathrm{H}_{2} \mathrm{~S}$, and $\mathrm{NO}_{2}$ on the readings of one or both 5400 monitors relative to the reference results. For Phase II, NO and total $\mathrm{NO}_{x}$ were ascribed a statistical influence to both monitors relative to the reference EC and TC , and to $\mathrm{NO}_{2}$ an influence on one monitor relative to the reference OC. The combined effect of the multiple parameters was typically a few percent, relative to the linear regression of the 5400 and reference results.

Other Parameters
In general, these monitors required little maintenance and could be largely operated unattended. Data recovery was about $90 \%$ over both phases of testing.

## Metals

In conjunction with the $\mathrm{TEOM}^{\circledR} \mathrm{PM}_{2.5}$ systems at each location, an Automatic Cartridge Collection Unit, or ACCU (Rupprecht \& Patashnick Co., Inc., Albany, NY), was used to collect particulates for metals analysis. The ACCU attached to the $13.7 \mathrm{l} / \mathrm{min}$ bypass flow line of the $\mathrm{TEOM}^{\circledR}$ monitor and permitted filter-based sampling. The system's eight internal flow channels allowed for daily collection of particulate samples through the use of a bank of solenoid valves. These valves were electronically controlled by the Series 1400a monitor. The airflow was directed through filter holders fitted with 47-mm, 2.0- $\mu \mathrm{m}$ pore size Zeflour ${ }^{\mathrm{TM}}$ supported PTFE filters (Pall Corp., Ann Arbor, MI). The following metals were included in the analysis (detection limits are in parentheses): Cr ( $5 \mathrm{ng} / \mathrm{m} 3$ ), Fe ( 22 $\mathrm{ng} / \mathrm{m} 3), \mathrm{Pb}(12 \mathrm{ng} / \mathrm{m} 3)$, $\mathrm{Mn}(3 \mathrm{ng} / \mathrm{m} 3)$, $\mathrm{Ni}(4 \mathrm{ng} / \mathrm{m} 3)$, and $\mathrm{Zn}(77 \mathrm{ng} / \mathrm{m} 3)$.

## Acid Aerosols, Ammonia, and Acid Gases

The URG-2000-01J Weekly Air Particulate Sampler (URG, Chapel Hill, NC), an 8-channel annular denuder system, was used to characterize five reactive gases $\left(\mathrm{NH}_{3}, \mathrm{HCl}, \mathrm{HNO}_{2}, \mathrm{HNO}_{3}\right.$, and $\left.\mathrm{SO}_{2}\right)$, particulate sulfate, and aerosol pH (EPA Method IO-4.2). Each channel was fitted with two 120-mm glass heavy-wall annular denuders connected in series, followed by a $47-\mathrm{mm}, 2.0-\mu \mathrm{m}$ supported PTFE filter (Pall Corp., Ann Arbor, MI). The first annular denuder was coated with sodium carbonate to collect acid gases, and the second with citric acid to collect $\mathrm{NH}_{3}$. The flush end of the citric acid-coated denuder was attached directly to the filter module. The filters were positioned on the Teflon-
coated stainless steel screen such that the air stream particulates were trapped on the Teflon-coated side of the filter. The denuders were coated with appropriate coating solutions (citric acid: 1\%weight/volume in methanol; sodium carbonate: $1 \% \mathrm{w} / \mathrm{v}, 1 \% \mathrm{w} / \mathrm{v}$ glycerol in a $1: 1$ methanol/water solution). The coated tubes were dried with "zero" air at a rate of $3 \mathrm{~L} / \mathrm{min}$. The denuder trains were assembled and leak-checked in clean laboratory conditions. A blank denuder assembly was included with each batch of seven denuder assemblies sent out in the field. It was left for seven days inside the sampler but was not connected to the airflow.

Ambient air was drawn through aluminum, Teflon ${ }^{\circledR}$-coated $\mathrm{PM}_{10}$ and $\mathrm{PM}_{2.5}$ size-selective inlet, then through the denuder and filter. Daily (24-hour) samples were collected beginning at midnight, at a flow rate of $10 \mathrm{~L} / \mathrm{min}$. Inlets were cleaned and replaced when necessary. After exchanging the denuders, leak checks were performed to assure system integrity.

The coated annular denuders from the exposed assemblies and field blanks were extracted with 10 ml ultra-pure water (Millipore, Milli-Q UV Plus water systems), and stored at $4^{\circ} \mathrm{C}$ for analysis. The water extract from sodium carbonate-coated denuders was used for the determination of $\mathrm{HONO}, \mathrm{HNO}_{3}$, and HCl . For $\mathrm{SO}_{2}$ analysis, 5 ml of the water extracts from the sodium carbonate-coated denuders were oxidized with 0.05 ml of $30 \%$ aqueous $\mathrm{H}_{2} \mathrm{O}_{2}$ solution to completely oxidize the collected $\mathrm{SO}_{2}$ to $\mathrm{SO}_{4}$ before analysis. The water extract from citric acid-coated denuders was used to determine ammonia. The measurement of chloride, nitrite, nitrate, sulfate, and ammonium was made with a DIONEX 500 Ion Chromatography System. The results were calculated for gaseous HCl, HONO, $\mathrm{HNO}_{3}, \mathrm{SO}_{2}$, and $\mathrm{NH}_{3}$. The separation of chloride, nitrite, nitrate and sulfate was accomplished using an IonPac AS $14(4 \times 250 \mathrm{~mm})$ analytical column, AG 14 guard column, with a $10 \mu \mathrm{l}$ sample loop, and an anion self-regenerating suppressor-ultra. A solution of $3.5 \mathrm{mM} \mathrm{Na} \mathrm{CO}_{3} / 1.0 \mathrm{mM} \mathrm{NaHCO}_{3}$ was used as eluent at a flow rate of $1 \mathrm{ml} / \mathrm{min}$. The separation of ammonium was accomplished using an IonPac CS14 (4 x 250 mm ) analytical column and a CG 14 guard column with a $50 \mu \mathrm{l}$ sample loop, and a cation self-regenerating suppressor-ultra. A solution of 10 mM methanesulfonic acid was used as eluent at a flow rate of $1 \mathrm{ml} / \mathrm{min}$.

The Zefluor filters were ultrasonically extracted for one hour in 5 ml of ultra-pure water, the pH was measured, and the samples were stored at $4^{\circ} \mathrm{C}$ for analysis of particulate sulfate. The filter extracts were analyzed for particulate sulfate by ion chromatography using the DIONEX 100 Ion Chromatography System. Selenium was also determined in some of the filter extracts using inductively coupled plasma mass spectrometry (ICP-MS). Concentrations in the field blanks for the target species were subtracted on a batch-to-batch basis. Accuracy of calibration curves was checked by analyzing the quality control samples containing the analytes of interest at a concentration in the low and high concentration range provided by an independent QA/QC laboratory within the Wadsworth Center. For all the analytes, the controls were within $\pm 10 \%$. The percent standard deviation of measurements, evaluated on duplicate runs of several samples, was found to be better than $\pm 3.0 \%$.

Particulate nitrate was originally included in the analyte list but was later dropped because of concerns about the accuracy of the reported concentrations. During the study, research was published that called into question particulate nitrate concentrations collected on Teflon filters. (The ADS used in the study collected samples on Teflon filters.) Higher temperatures experienced during the daytime in the summer months may lead to a loss of particulate nitrate from the sample. Temperatures inside the ADS enclosure on some days exceeded $108^{\circ} \mathrm{F}$. Because the ADS was serviced only once per week, samples collected after servicing were subject to more high-temperature periods than those collected the day prior to servicing, likely increasing the potential for particulate nitrate volatilization. This information, along with inconsistencies found in the concentrations of some co-located samples, led to the removal of particulate/aerosol nitrate from the analyte list.

## Pollen and Mold

Weekly pollen and mold samples were collected with a Burkard Recording Volumetric Spore Trap (Burkard Manufacturing Co., Ltd, Rickmansworth, England). Particles were impacted on adhesive-coated Melinex transparent plastic tape, supported on a clockwork-driven drum. After a thin film of $10 \%$ Gelvatol was applied to the tape and allowed to dry, the adhesive (Vaseline and 10\% paraffin wax in toluene) was then applied. The clockwork drum allowed for a seven-day sample to be collected, with the sampling volume ranging between 9 and 12 lpm . After removal of the drum, the tape was sectioned and viewed as individual days. Each slide was mounted with glycerin jelly and phenosafranin stain.

Individual bioaerosol categories were grouped into larger aggregations of pollen or mold types based on taxonomic or aerodynamic relationships. The pollen and spore aggregations used in statistical analyses are as follows:

Table A2. Bioaerosol Aggregate Categories

| Pollen | Mold |
| :---: | :---: |
| Tree Pollens <br> Abies, Acer, Alnus, Betula, Carya, Cupressa, Fagus, Fraxinus, Gingko, Juglans, Liquidaum, Morus, Olea, Picea, Pinus, Platanus, Populus, Quercus, Salix, Tilia, Tsuga, Ulmus | Basidiospores <br> Ganoderma, Coprinus, unidentified basidiospores |
| Grass Pollens Graminea | Ascospores <br> Diatrype, Leptosphaeria, Sporormiella, unidentified ascospores |
| Ragweed Pollen Ambrosia | Dark Mitospores <br> Alternaria, Arthrinium, Cladosporium, Curvularia, Epicoccum, Helminthosporium, Nigrospora, Periconium, Pithomyces, Torula, Stemphylium |
| Total Pollens <br> Tree pollen + Grass pollen + Ragweed pollen + <br> Unidentified pollens | Non-dark Mitospores <br> Penicillium/Aspergillus, Botrytis, <br> Cercospora, Fusarium, Oidium, <br> Peronospora, Pestalotiopsis, Polythrincium |
|  | Small spores all fungal spores $<10 \mu \mathrm{~m}$ |
|  | Large spores <br> all fungal spores > $10 \mu \mathrm{~m}$ |
|  | Total Molds <br> Basidiospores + Ascospores + Dark mitospores <br> + Non-dark mitospores + Unidentified mold spores |

## Acetone and Aldehydes

An ATEC Model 1600 automated multi-port sampler (Atmospheric Technology, Calabasas, CA) was used in the collection of samples for acetone and aldehyde analysis, according to EPA Method TO-11. The ATEC was programmed with a week-long run schedule to collect seven daily 24 -hour samples. Channels ran consecutively from midnight to midnight. Air was drawn through cartridges containing 2,4-dinitrophenylhydrazine- (DNPH-) coated silica (Waters Corp., Milford, MA). Following collection, the samples were eluted from the cartridge as the DNPH derivative, then analyzed by HPLC with UV detection. Flows varied between 0.28 and 0.29 lpm , yielding
approximate sample volumes of 403 to 417 liters. Actual sample volumes and run times were recorded by the instrument and were used for concentration calculations. After the installation of the new cartridges, and prior to resumption of the sampling run, all ports were checked for leaks. A denuder box was attached to the inlet port to remove ozone from the sample stream (using a potassium iodide-coated copper coil). These boxes were replaced at three- to four-week intervals. The analytes measured were acetaldehyde, acetone, acrolein, benzaldehyde, butyraldehyde, crotonaldehyde, 2,5-dimethylbenzaldehyde, formaldehyde, hexaldehyde, isovaleraldehyde, propionaldehyde, m-tolualdehyde, o-tolualdehyde, p-tolualdehyde and valeraldehyde. Detection limit was $1 \mu \mathrm{~g} / \mathrm{m}^{3}$.

## $\mathrm{SO}_{2}$ Determination

The Thermo Environmental Instruments (TEI) Model 43C SO 2 Pulsed Fluorescence Analyzer has been designated by EPA as Equivalent $\mathrm{SO}_{2}$ Analyzer (No. EQSA-0486-060). Pulsating UV light is focused through a narrow band pass of 190 nanometers that directs it into the fluorescence chamber. Sampled ambient air containing $\mathrm{SO}_{2}$ flows continuously through the chamber, where the UV light excites the $\mathrm{SO}_{2}$ molecules causing them to emit their characteristic decay radiation. This $\mathrm{SO}_{2}$-specific radiation passes through a second filter and onto a sensitive photomultiplier tube. Incoming light energy is transformed electronically into a $0-5 V D C$ output signal that is directly proportional to the concentration of $\mathrm{SO}_{2}$ in the sample air.

## $\mathrm{NO} / \mathrm{NO}_{2} / \mathrm{NO}_{x}$ Determination

The Thermo Environmental Instruments (TEI) Model $42 \mathrm{NO} / \mathrm{NO}_{2} / \mathrm{NO}_{x}$ analyzers utilize the technique of photometric detection of chemiluminescent light resulting from the flameless reaction of nitric oxide (NO) with ozone $\left(\mathrm{O}_{3}\right)$ for interference-free measurement of $\mathrm{NO}_{2}$. The analyzer includes a $\mathrm{NO}_{\mathrm{x}}$-to- NO heated molybdenum converter to change $\mathrm{NO}_{2}$ into NO for subsequent measurement via the chemiluminescent detection method. The ambient air sample enters Model 42 through a single flow-control capillary and is directed to a solenoid valve. The solenoid valve routes the sample either through the $\mathrm{NO}_{2}$-to- NO converter $\left(\mathrm{NO}_{\mathrm{x}}\right.$ mode) or around the converter ( NO mode). When flowing through the converter, the chemiluminescence measured within the reaction chamber represents the $\mathrm{NO}_{\mathrm{x}}$ concentration. Bypassing the converter allows measurement of the NO level only. The signals generated in the two modes are stored and held in memory by the instrument's microcomputer, where the difference between them is used to generate a $\mathrm{NO}_{2}$ signal. The digital-to-analog converter then converts the three stored values into analog signals that are output to the rear of the instrument. The NO and $\mathrm{NO}_{\mathrm{x}}$ concentrations calculated in the NO and $\mathrm{NO}_{\mathrm{x}}$ modes are stored in memory. The difference between the concentrations is used to calculate the $\mathrm{NO}_{2}$ concentration.

## Ozone Determination

The Thermo Environmental Instruments (TEI) Model 49-Ultraviolet Photometer ozone analyzer has been designated by U.S. EPA as an equivalent method for the measurement of ambient concentration of ozone pursuant to the requirements defined in 40 CFR Part 53. Its designated equivalence method number is EQQA-0880-047. The UV photometer determines ozone concentrations by measuring the attenuation of light due to ozone in the absorption
cell, at a wavelength of 254 nanometers. The concentration of ozone is directly related to the magnitude of the attenuation. The reference ozone-free gas passes into the absorption cell to establish a "zero" light intensity reading ( $\mathrm{I}_{\mathrm{o}}$ ). The solenoid then switches, and the sample passes through the absorption cell to establish a "sample" light intensity reading (I). The ratio of these readings $\left(I / I_{0}\right)$ is a measure of the light absorbed by ozone in the sample at 254 nm . It is directly related to the concentration of ozone in the sample through the Beer-Lambert Law. A second detector is used to monitor the changes in light intensity and to correct for these changes. This system is basically two photometers utilizing two separate but similar absorption cells and detector systems. They share the same source. These two photometers operate 180 degrees out of phase but synchronously and integrate the signals simultaneously: thus I in cell $B(I(B))$ is determined at the identical time $I_{0}$ in cell $A\left(I_{0}(A)\right)$ is determined. The solenoids then switch, and after an appropriate flush time (approximately 7 seconds), $I_{0}(B)$ and $I(A)$ are determined. Taking the average value of these two readings factors out the fluctuation in lamp intensity. The microcomputer in the TEI Model 49 solves the Beer-Lambert equation directly for each cell and outputs the average concentration in both the front panel digital display and the recorder analog output.

## Meteorological Data

Temperature, relative humidity, and wind speed and direction were logged with a Young 27600 Programmable Translator (R.M. Young Co., Traverse City, MI). The unit logged the data from the roof-mounted wind monitor and relative humidity/temperature probe (Models 05305 and 41372LC, respectively, from R.M. Young Co.).

## Flow Rates

Flow rates for the TSI, URG, and TEOM-ACCU were checked and calibrated with a DryCal DC-1 digital flow calibrator (BIOS International, Pompton Plains, NJ). The NIST-traceable DryCal DC-1 has an accuracy of $\pm 1 \%$, with a worst-case resolution of $0.2 \%$.

## Statistical Methods

## Multivariate procedures

Square Spearman correlation matrices were used as input to the MDS procedure implemented in SYSTAT v. 9 (SPSS Inc.). The SYSTAT procedure creates dissimilarity matrices from correlation matrices by taking the negative of all correlation coefficients. MDS distances are then computed from dissimilarities. Two-dimensional MDS configurations were generated for each correlation matrix using SYSTAT defaults for number of iterations and for convergence criteria. Among the three possible loss functions (Kruskal, Guttman, Young) available in the SYSTAT procedure, the Guttman loss function (Wilkinson 1999) generally explained the greatest proportion of variance in preliminary analyses and therefore was used throughout. Shepard diagrams and output of the Guttman coefficient of alienation at each iteration step were used as diagnostics for degenerate solutions.

MDS configuration plots were constructed for each correlation matrix. Non-metric MDS re-scales measures of dissimilarity between variables so that the rank order of distances between variables in the MDS plot correspond as
closely as possible to the rank order of dissimilarities between variables in the original multi-dimensional space. When dissimilarities between variables are measured with correlation coefficients, the distance between variables in the MDS plot indicates the strength of their correlation. The plots were interpreted qualitatively by observing whether points representing the pollutant analytes clustered closely together (indicating strong positive correlation among variables) or whether points were far apart (indicating large negative correlations). Intermediate distances between variables were indicative of relatively weak associations.

Rectangular data matrices were used as input to the HC procedure implemented in SYSTAT v. 9 (SPSS Inc.). Pearson correlations ( $r$ ) were used to calculate the distance metric ( $d$ ) between variables, where $d=1-r$. Complete-linkage hierarchical clustering was used to construct a tree diagram representing distances between clusters of variables. As in MDS, the tree diagrams were interpreted qualitatively by observing which variables tended to be strongly associated with each other and whether consistent clustering of variables could be observed. In the cluster trees, distances between variables or clusters near zero represent strong positive correlations, while distances near two represent strong negative correlations. Intermediate distances represent weak correlations between variables or clusters.

Appendix 2 - Detailed Data Summary

Appendix 2 - Summary of Data (continued)

Appendix 2 - Summary of Data (continued)











| Appendix 2 - Summary of Data (continued) |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Descriptive Statistics |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
|  | Season | Site | SR's | RJ's | PL's | LT's | Missing | N | Mean | Std. Dev. | Max | 95th | 75th | Median | 25th | 5th | Min |
|  | Winter99 | Bronx | --- | --- | --- | --- | 79 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | Manhattan | --- | --- | --- | --- | 79 | 0 | --- | --- | --- | --- | --- | --- | -- | -- | --- |
|  | Spring99 | Bronx | --- | --- | --- | --- | 92 | 0 | - | --- | - | - | --- | --- | -- | -- | --- |
|  |  | Manhattan | --- | --- | --- | --- | 92 | 0 | --- | --- | - | --- | --- | --- | --- | --- | --- |
|  | Summer99 | Bronx | --- | --- | --- | --- | 60 | 34 | 1.9 | 1.6 | 5.3 | 5.0 | 2.8 | 1.4 | 0.5 | 0.1 | 0.1 |
|  |  | Manhattan | --- | --- | --- | --- | 8 | 86 | 1.9 | 1.5 | 6.4 | 4.8 | 3.0 | 1.6 | 0.6 | 0.3 | 0.1 |
|  | Fall99 | Bronx | --- | - | --- | --- | 2 | 88 | 4.0 | 2.5 | 12.7 | 9.0 | 5.2 | 3.4 | 2.2 | 1.1 | 0.5 |
|  |  | Manhattan | --- | --- | -- | --- | 1 | 89 | 4.4 | 2.2 | 10.4 | 8.3 | 5.7 | 4.1 | 2.7 | 1.6 | 1.2 |
|  | Winter00 | Bronx | --- | -- | --- | --- | 5 | 84 | 3.7 | 2.6 | 12.5 | 8.6 | 5.1 | 2.9 | 1.6 | 0.9 | 0.6 |
|  |  | Manhattan | --- | --- | --- | --- | 4 | 85 | 4.0 | 2.7 | 16.8 | 8.8 | 4.6 | 3.2 | 2.2 | 1.6 | 1.2 |
|  | Spring00 | Bronx | --- | --- | --- | --- | 0 | 92 | 2.4 | 1.9 | 11.8 | 6.4 | 2.9 | 2.1 | 1.2 | 0.5 | 0.4 |
|  |  | Manhattan | --- | -- | --- | --- | 0 | 92 | 2.9 | 2.0 | 11.8 | 7.3 | 3.7 | 2.7 | 1.6 | 0.7 | 0.5 |
|  | Summer00 | Bronx | --- | - | --- | --- | 72 | 22 | 1.4 | 1.2 | 5.0 | 2.8 | 2.2 | 0.9 | 0.5 | 0.3 | 0.2 |
|  |  | Manhattan | --- | --- | - | --- | 72 | 22 | 1.5 | 1.1 | 3.9 | 3.5 | 2.4 | 1.1 | 0.6 | 0.5 | 0.5 |
|  | Fall00 | Bronx | --- | --- | --- | --- | 62 | 0 | --- | --- | - | --- | --- | --- | --- | --- | -- |
|  |  | Manhattan | --- | --- | --- | --- | 62 | 0 | --- | - | --- | --- | --- | --- | - | -- | --- |
|  | Winter99 | Bronx | --- | --- | --- | --- | 79 | 0 | --- | --- | --- | --- | --- | - | --- | --- | --- |
|  |  | Manhattan | --- | --- | --- | --- | 79 | 0 | - | --- | - | --- | --- | --- | --- | -- | --- |
|  | Spring99 | Bronx | --- | --- | --- | --- | 92 | 0 | - | --- | --- | - | --- | --- | --- | -- | --- |
|  |  | Manhattan | --- | --- | - | --- | 92 | 0 | - | --- | --- | --- | --- | --- | --- | --- | -- |
|  | Summer99 | Bronx | --- | --- | --- | --- | 60 | 34 | 2.5 | 2.0 | 6.4 | 6.4 | 3.7 | 1.8 | 0.7 | 0.3 | 0.1 |
|  |  | Manhattan | --- | --- | -- | -- | 9 | 85 | 3.9 | 3.1 | 14.7 | 10.9 | 5.6 | 2.9 | 1.6 | 0.4 | 0.1 |
|  | Fall99 | Bronx | --- | --- | - | --- | 2 | 88 | 0.6 | 0.6 | 3.1 | 1.6 | 0.6 | 0.4 | 0.2 | 0.1 | 0.0 |
|  |  | Manhattan | --- | --- | --- | --- | 1 | 89 | 0.6 | 0.5 | 2.5 | 1.8 | 0.8 | 0.5 | 0.3 | 0.1 | 0.1 |
|  | Winter00 | Bronx | --- | --- | --- | --- | 5 | 84 | 0.5 | 0.3 | 1.8 | 1.0 | 0.6 | 0.5 | 0.3 | 0.2 | 0.1 |
|  |  | Manhattan | --- | --- | --- | --- | 4 | 85 | 1.0 | 0.7 | 3.0 | 2.3 | 1.5 | 0.8 | 0.4 | 0.2 | 0.1 |
|  | Spring00 | Bronx | --- | --- | --- | --- | 0 | 92 | 1.2 | 1.4 | 7.2 | 4.4 | 1.1 | 0.7 | 0.4 | 0.2 | 0.1 |
|  |  | Manhattan | --- | --- | --- | --- | 0 | 92 | 1.2 | 1.4 | 6.9 | 4.7 | 1.2 | 0.8 | 0.4 | 0.2 | 0.1 |
|  | Summer00 | Bronx | --- | --- | --- | --- | 72 | 22 | 3.1 | 1.1 | 5.0 | 4.9 | 3.9 | 3.2 | 2.3 | 1.4 | 1.3 |
|  |  | Manhattan | --- | --- | --- | --- | 72 | 22 | 3.2 | 1.0 | 5.3 | 4.9 | 4.0 | 3.1 | 2.6 | 1.8 | 1.5 |
|  | Fall00 | Bronx | -- | --- | - | - | 62 | 0 | - | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | Manhattan | --- | - | --- | - | 62 | 0 | --- | --- | --- | --- | --- | --- | - | --- | --- |
|  | Winter99 | Bronx | --- | --- | --- | --- | 79 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | Manhattan | --- | - | --- | --- | 79 | 0 | - | --- | --- | --- | --- | --- | - | --- | --- |
|  | Spring99 | Bronx | --- | --- | --- | -- | 92 | 0 | - | --- | --- | --- | --- | --- | -- | - | -- |
|  |  | Manhattan | --- | --- | --- | --- | 92 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | Summer99 | Bronx | --- | -- | -- | -- | 74 | 20 | 4.5 | 1.2 | 6.7 | 6.4 | 5.4 | 4.6 | 3.5 | 2.7 | 2.7 |
|  |  | Manhattan | --- | - | --- | -- | 43 | 51 | 4.3 | 0.9 | 6.0 | 5.5 | 5.0 | 4.3 | 3.7 | 3.0 | 1.6 |
|  | Fall99 | Bronx | --- | --- | --- | --- | 90 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | Manhattan | --- | --- | -- | - | 90 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | Winter00 | Bronx | --- | --- | - | - | 11 | 78 | 1.3 | 1.4 | 6.7 | 4.8 | 1.7 | 0.9 | 0.2 | 0.1 | 0.0 |
|  |  | Manhattan | --- | - | --- | - | 9 | 80 | 2.8 | 1.8 | 8.3 | 7.0 | 3.7 | 2.3 | 1.2 | 0.8 | 0.6 |
|  | Spring00 | Bronx | --- | --- | -- | - | 34 | 58 | 2.8 | 1.7 | 6.9 | 6.3 | 3.8 | 2.3 | 1.4 | 0.8 | 0.5 |
|  |  | Manhattan | --- | - | --- | --- | 37 | 55 | 4.0 | 2.2 | 10.8 | 7.2 | 5.3 | 3.4 | 2.2 | 1.5 | 1.1 |
|  | Summer00 | Bronx | --- | --- | --- | --- | 94 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | Manhattan | --- | --- | --- | --- | 94 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | Fall00 | Bronx | --- | --- | --- | --- | 62 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | Manhattan | --- | --- | --- | --- | 62 | 0 | --- | --- | --- | --- | --- | --- | --- | --- | --- |






Appendix 2 - Summary of Data (continued)


Appendix 3 - Detailed Statistical Results, Entire Study Period

| Statistics and Analyses - Daily Averages ${ }^{\text {a }}$ |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Analyte | Pearson Correlation | $\begin{aligned} & \text { Detection } \\ & \text { Limit } \end{aligned}$ | Manhattan |  |  |  | Bronx |  |  |  | Difference ${ }^{\text {b }}$ |  |  |  |  |  |  |
|  |  |  | N | Missing <br> (\%) | NonDetects (\%) | Mean ${ }^{\text {c }}$ | N | Missing <br> (\%) | NonDetects (\%) | Mean ${ }^{\text {c }}$ | N | Missing (\%) | Mean ${ }^{\text {c }}$ | Mean (\%) ${ }^{\text {d }}$ | Paired T-test with Autocorrelation Adjustment |  |  |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  | \# of lags | T | p-value |
| pH | 0.6853 | --- | 680 | 1.7\% | --- | 5.04 | 627 | 9.4\% | --- | 5.15 | 622 | 10.1\% | -0.07 | -1.4\% | 1 | -4.32 | <0.0001 |
| Sulfate | 0.9647 | 0.24 | 674 | 2.6\% | 0.0\% | 4.0 | 617 | 10.8\% | 0.3\% | 3.6 | 607 | 12.3\% | 0.1 | 3.4\% | 0 | 3.90 | 0.0001 |
| Carb250 | 0.6155 | --- | 650 | 6.1\% | --- | 3.09 | 590 | 14.7\% | --- | 3.17 | 556 | 19.7\% | -0.15 | -4.9\% | 44 | -0.79 | 0.4293 |
| Soot | 0.7671 |  | 592 | 14.5\% | --- | 1.32 | 582 | 15.9\% | --- | 1.19 | 498 | 28.0\% | 0.08 | 6.5\% | 18 | 1.49 | 0.1380 |
| Ozone | 0.9235 | --- | 630 | 9.0\% | --- | 0.012 | 518 | 25.1\% | --- | 0.016 | 482 | 30.3\% | -0.004 | -33.3\% | 9 | -12.01 | $<0.0001$ |
| NOX | 0.8652 | --- | 625 | 9.7\% | --- | 0.066 | 425 | 38.6\% | --- | 0.053 | 367 | 47.0\% | 0.012 | 18.8\% | 8 | 6.51 | <0.0001 |
| NO | 0.8794 | --- | 625 | 9.7\% | --- | 0.031 | 425 | 38.6\% | --- | 0.022 | 367 | 47.0\% | 0.008 | 27.6\% | 3 | 7.72 | $<0.0001$ |
| NO2 | 0.7704 | --- | 625 | 9.7\% | --- | 0.036 | 425 | 38.6\% | --- | 0.031 | 367 | 47.0\% | 0.005 | 13.9\% | 7 | 6.11 | <0.0001 |
| SO2 | 0.8967 | --- | 648 | 6.4\% | --- | 0.012 | 608 | 12.1\% | --- | 0.011 | 566 | 18.2\% | 0.002 | 15.4\% | 27 | 2.94 | 0.0034 |
| PM2.5 (TEOM) | 0.9659 | --- | 631 | 8.8\% | --- | 16.2 | 567 | 18.1\% | --- | 15.3 | 517 | 25.3\% | 0.8 | 4.8\% | 0 | 8.43 | $<0.0001$ |
| PM10 (TEOM) | 0.9185 | --- | 609 | 12.0\% | --- | 23.1 | 497 | 28.2\% | --- | 22.3 | 444 | 35.8\% | 0.9 | 4.1\% | 14 | 1.87 | 0.0616 |
| Acetaldehyde* | 0.8086 | 1.0 | 674 | 2.6\% | 1.2\% | 2.7 | 577 | 16.6\% | 0.4\% | 2.5 | 568 | 17.9\% | 0.0 | 1.3\% | 21 | 0.23 | 0.8198 |
| Acetone* | 0.2342 | 1.0 | 674 | 2.6\% | 2.2\% | 6.9 | 577 | 16.6\% | 0.1\% | 6.8 | 568 | 17.9\% | 0.0 | -0.1\% | 2 | -0.03 | 0.9786 |
| Acrolein* | ---- | 1.0 | 674 | 2.6\% | 96.2\% | 0.6 | 577 | 16.6\% | 83.2\% | 0.5 | 568 | 17.9\% | 0.0 | 0.0\% | -- | --- | --- |
| Benzaldehyde* | 0.4232 | 1.0 | 674 | 2.6\% | 93.9\% | 0.5 | 577 | 16.6\% | 76.2\% | 0.5 | 568 | 17.9\% | 0.0 | -1.8\% | --- | --- | -- |
| Butyraldehyde* | -0.0285 | 1.0 | 674 | 2.6\% | 68.9\% | 0.8 | 577 | 16.6\% | 66.0\% | 0.7 | 568 | 17.9\% | 0.1 | 14.9\% | --- | --- | - |
| Crotonaldehyde* | 0.5875 | 1.0 | 674 | 2.6\% | 69.5\% | 0.8 | 577 | 16.6\% | 63.4\% | 0.7 | 568 | 17.9\% | 0.0 | 2.0\% | --- | --- |  |
| Formaldehyde* | 0.7982 | 1.0 | 674 | 2.6\% | 0.3\% | 4.4 | 577 | 16.6\% | 0.1\% | 4.2 | 568 | 17.9\% | -0.1 | -2.8\% | 27 | -0.47 | 0.6391 |
| Hexaldehyde* | 0.3813 | 1.0 | 674 | 2.6\% | 81.1\% | 0.8 | 577 | 16.6\% | 70.4\% | 0.6 | 568 | 17.9\% | -0.1 | -12.9\% | --- | -- | --- |
| Isovaleraldehyde* | 0.0614 | 1.0 | 674 | 2.6\% | 90.5\% | 0.6 | 577 | 16.6\% | 78.5\% | 0.5 | 568 | 17.9\% | 0.0 | -1.2\% | --- | --- |  |
| m -Tolualdehyde* | ---- | 1.0 | 674 | 2.6\% | 97.4\% | 0.5 | 577 | 16.6\% | 83.4\% | 0.5 | 568 | 17.9\% | 0.0 | 0.0\% | --- | - | -- |
| o-Tolualdehyde* | -0.0038 | 1.0 | 674 | 2.6\% | 96.8\% | 0.5 | 577 | 16.6\% | 82.7\% | 0.5 | 568 | 17.9\% | 0.0 | -0.8\% | -- | --- | -- |
| p-Tolualdehyde* | 0.6907 | 1.0 | 674 | 2.6\% | 87.1\% | 0.5 | 577 | 16.6\% | 71.1\% | 0.6 | 568 | 17.9\% | 0.0 | -1.6\% | --- | --- | -- |
| Propionaldehyde* | 0.4719 | 1.0 | 674 | 2.6\% | 67.1\% | 0.9 | 577 | 16.6\% | 55.3\% | 0.8 | 568 | 17.9\% | -0.1 | -9.7\% | --- | --- | -- |
| Valeraldehyde* | 0.0271 | 1.0 | 674 | 2.6\% | 95.4\% | 0.5 | 577 | 16.6\% | 78.9\% | 0.5 | 568 | 17.9\% | 0.0 | -0.6\% | --- | --- | -- |
| 2,5-dimethylbenzaldehyde* | --- | 1.0 | 674 | 2.6\% | 97.3\% | 0.5 | 577 | 16.6\% | 83.4\% | 0.5 | 568 | 17.9\% | 0.0 | 0.0\% | --- | --- | --- |
| Total Aldehydes* | 0.5322 | --- | 673 | 2.7\% | --- | 16.2 | 588 | 16.6\% | --- | 16.6 | 567 | 18.1\% | -0.1 | -0.7\% | 18 | -0.11 | 0.9129 |
| Chromium | -0.0068 | 5 | 661 | 4.5\% | 93.9\% | 3 | 602 | 13.0\% | 85.0\% | 3 | 575 | 16.9\% | 0 | -13.8\% | --- | -- | --- |
| Iron | 0.3656 | 22 | 661 | 4.5\% | 19.9\% | 72 | 602 | 13.0\% | 19.9\% | 75 | 575 | 16.9\% | -4 | -6.1\% | 1 | -0.96 | 0.3369 |
| Lead | 0.0541 | 12 | 661 | 4.5\% | 90.6\% | 7 | 602 | 13.0\% | 81.6\% | 7 | 575 | 16.9\% | 0 | -5.8\% | --- | --- | --- |
| Manganese | 0.1166 | 3 | 661 | 4.5\% | 85.4\% | 2 | 602 | 13.0\% | 81.5\% | 2 | 575 | 16.9\% | 0 | 2.7\% | --- | --- | --- |
| Nickel | 0.3835 | 4 | 640 | 7.5\% | 22.4\% | 15 | 575 | 16.9\% | 25.9\% | 12 | 548 | 20.8\% | 4 | 24.3\% | 0 | 4.43 | $<0.0001$ |
| Zinc | 0.1283 | 77 | 661 | 4.5\% | 93.6\% | 40 | 602 | 13.0\% | 84.4\% | 41 | 575 | 16.9\% | -2 | -3.9\% | --- | - | --- |
| Total Metals | 0.3283 | --- | 615 | 11.1\% | --- | 94 | 532 | 23.1\% | --- | 101 | 489 | 29.3\% | -4 | -4.6\% | 1 | -0.68 | 0.4976 |
| SO2 | 0.8973 | --- | 374 | 46.0\% | --- | 26.4 | 320 | 53.8\% | --- | 25.8 | 310 | 55.2\% | 1.9 | 7.0\% | 0 | 3.93 | 0.0001 |
| HCl | 0.8364 | --- | 375 | 45.8\% | --- | 0.51 | 320 | 53.8\% | --- | 0.48 | 311 | 55.1\% | -0.05 | -10.8\% | 3 | -2.21 | 0.0278 |
| HONO | 0.8350 | --- | 374 | 46.0\% | --- | 3.21 | 320 | 53.8\% | --- | 3.07 | 311 | 55.1\% | 0.38 | 10.9\% | 0 | 4.85 | <0.0001 |
| HNO3 | 0.9293 | --- | 373 | 46.1\% | --- | 1.75 | 320 | 53.8\% | --- | 1.11 | 310 | 55.2\% | 0.19 | 15.0\% | 14 | 2.52 | 0.0124 |
| NH3 | 0.9150 | --- | 186 | 73.1\% | --- | 3.536 | 156 | 77.5\% | --- | 2.274 | 135 | 80.5\% | 1.331 | 39.8\% | 1 | 15.98 | <0.0001 |
| Total Pollen | 0.9792 | --- | 691 | 0.1\% | --- | 13.2 | 632 | 8.7\% | --- | 22.3 | 632 | 8.7\% | -8.2 | -58.1\% | 5 | -1.42 | 0.1572 |
| Tree Pollen | 0.9795 | -- | 691 | 0.1\% | --- | 12.2 | 637 | 7.9\% | --- | 20.5 | 637 | 7.9\% | -7.4 | -56.4\% | 5 | -1.29 | 0.1981 |
| Ragweed | 0.8619 | --- | 691 | 0.1\% | --- | 0.4 | 637 | 7.9\% | --- | 0.4 | 637 | 7.9\% | -0.1 | -48.8\% | 2 | -2.45 | 0.0147 |
| Grasses | 0.7479 | -- | 691 | 0.1\% | --- | 0.4 | 637 | 7.9\% | --- | 0.5 | 637 | 7.9\% | -0.1 | -31.3\% | 1 | -2.15 | 0.0322 |
| Total Mold | 0.8381 | --- | 691 | 0.1\% | --- | 490.3 | 632 | 8.7\% | --- | 447.8 | 632 | 8.7\% | -35.0 | -8.5\% | 4 | -1.39 | 0.1660 |
| Basidospores | 0.7090 | -- | 691 | 0.1\% | --- | 186.0 | 637 | 7.9\% | --- | 184.0 | 637 | 7.9\% | -16.7 | -10.0\% | 3 | -1.10 | 0.2708 |
| Ascospores | 0.6789 | -- | 691 | 0.1\% | --- | 39.1 | 637 | 7.9\% | --- | 43.2 | 637 | 7.9\% | -4.1 | -10.4\% | 0 | -1.46 | 0.1454 |
| Mitospores | 0.8709 | --- | 691 | 0.1\% | --- | 259.9 | 637 | 7.9\% | --- | 212.5 | 637 | 7.9\% | -12.5 | -6.2\% | 3 | -1.02 | 0.3075 |
| Mitospores (Dark) | 0.8753 | --- | 691 | 0.1\% | --- | 254.1 | 637 | 7.9\% | --- | 208.1 | 637 | 7.9\% | -13.5 | -7.0\% | 3 | -1.14 | 0.2547 |
| Mitospores (Non-Dark) | 0.0538 | --- | 691 | 0.1\% | --- | 5.8 | 637 | 7.9\% | --- | 4.4 | 637 | 7.9\% | 1.1 | 19.4\% | 0 | 0.84 | 0.4036 |
| Small Spores (<10 um) | 0.8304 | -- | 691 | 0.1\% | --- | 470.4 | 637 | 7.9\% | --- | 427.8 | 637 | 7.9\% | -31.4 | -7.9\% | 3 | -1.31 | 0.1904 |
| Large Spores (>10 um) | 0.7879 | --- | 691 | 0.1\% | --- | 12.5 | 637 | 7.9\% | --- | 9.9 | 637 | 7.9\% | -1.7 | -20.0\% | 0 | -2.14 | 0.0330 |
| Particle Count | 0.2231 | -- | 308 | 55.5\% | --- | 1463152 | 329 | 52.5\% | --- | 1560780 | 288 | 58.4\% | -100940 | -7.0\% | 16 | -1.01 | 0.3155 |
| PM2.5 (FRM) | 0.8979 | -- | 571 | 17.5\% | --- | 16.6 | 489 | 29.3\% | --- | 14.5 | 413 | 40.3\% | 1.5 | 9.7\% | 3 | 8.83 | <0.0001 |
| PM10 (FRM) | 0.9559 | --- | 102 | 11.3\% | --- | 22.0 | 80 | 30.4\% | --- | 20.9 | 74 | 35.7\% | 0.8 | 3.8\% | 6 | 2.17 | 0.0329 |
| Average Temperature | 0.9989 | --- | 649 | 6.2\% | --- | 56.5 | 610 | 11.8\% | --- | 53.9 | 593 | 14.3\% | 0.7 | 1.3\% | 7 | 12.01 | $<0.0001$ |
| Average Relative Humidity | 0.9773 | --- | 658 | 4.9\% | --- | 63 | 603 | 12.9\% | --- | 70 | 596 | 13.9\% | -7 | -10.8\% | 7 | -19.49 | <0.0001 |

Appendix 4 - Detailed Statistical Results by Season
Appendix 4 - Statistical Analyses - By Season

Appendix 4 - Statistical Analyses - By Season (continued)

Appendix 4 - Statistical Analyses - By Season (continued)

Appendix 4 - Statistical Analyses - By Season (continued)

Appendix 4 - Statistical Analyses - By Season (continued)

Appendix 4 - Statistical Analyses - By Season (continued)

Appendix 4 - Statistical Analyses - By Season (continued)

Appendix 4 - Statistical Analyses - By Season (continued)

Appendix 4 - Statistical Analyses - By Season (continued)

Appendix 4-Statistical Analyses - By Season (continued)

| Seasonal Statistics and Analyses - Daily Averages ${ }^{\text {a }}$ |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Season | Pearson Correlation | Detection Limit | Manhattan |  |  |  | Bronx |  |  |  | Difference ${ }^{\text {b }}$ |  |  |  |  |  |  |
|  |  |  |  | N | Missing(\%) | NonDetects (\%) | Mean ${ }^{\text {c }}$ | N | Missing(\%) | NonDetects (\%) | Mean ${ }^{\text {c }}$ | N | Missing(\%) | Mean ${ }^{\text {c }}$ | Mean (\%) ${ }^{\text {d }}$ | Paired T-test with Autocorrelation Adjustment |  |  |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  | \# of lags | T | p-value |
|  | Winter99 | 0.9973 | --- | 79 | 0.0\% | --- | 37.0 | 79 | 0.0\% | --- | 36.0 | 79 | 0.0\% | 1.0 | 2.6\% | 0 | 13.93 | <0.0001 |
|  | Spring99 | 0.9984 | --- | 87 | 5.4\% | --- | 59.8 | 87 | 5.4\% | --- | 59.2 | 87 | 5.4\% | 0.6 | 1.0\% | 0 | 10.06 | <0.0001 |
|  | Summer99 | 0.9904 | --- | 73 | 22.3\% | --- | 76.8 | 32 | 66.0\% | --- | 76.1 | 31 | 67.0\% | 0.4 | 0.5\% | 0 | 2.10 | 0.0438 |
|  | Fall99 | 0.9983 | --- | 86 | 4.4\% | - | 53.5 | 90 | 0.0\% | --- | 52.4 | 86 | 4.4\% | 0.7 | 1.3\% | 0 | 11.03 | <0.0001 |
|  | Winter00 | 0.9977 | --- | 82 | 7.9\% | --- | 37.8 | 89 | 0.0\% | --- | 35.8 | 82 | 7.9\% | 1.1 | 2.8\% | 0 | 13.10 | <0.0001 |
|  | Spring00 | 0.9978 | --- | 92 | 0.0\% | --- | 58.8 | 92 | 0.0\% | --- | 58.1 | 92 | 0.0\% | 0.7 | 1.3\% | 0 | 9.41 | <0.0001 |
|  | Summer00 | 0.9819 | --- | 89 | 5.3\% | --- | 72.6 | 87 | 7.4\% | --- | 72.6 | 82 | 12.8\% | 0.1 | 0.2\% | 2 | 0.82 | 0.4120 |
|  | Fall00 | 0.9970 | --- | 61 | 1.6\% | --- | 55.1 | 54 | 12.9\% | --- | 53.8 | 54 | 12.9\% | 0.8 | 1.4\% | 0 | 8.66 | <0.0001 |
|  | Winter99 | 0.9925 | --- | 79 | 0.0\% | --- | 60 | 79 | 0.0\% | --- | 66 | 79 | 0.0\% | -5 | -8.7\% | 1 | -16.27 | <0.0001 |
|  | Spring99 | 0.9934 | --- | 87 | 5.4\% | --- | 55 | 82 | 10.9\% | --- | 61 | 82 | 10.9\% | -6 | -10.3\% | 0 | -22.07 | <0.0001 |
|  | Summer99 | 0.9025 | --- | 73 | 22.3\% | --- | 62 | 31 | 67.0\% | --- | 67 | 31 | 67.0\% | -5 | -8.7\% | 0 | -4.01 | 0.0004 |
|  | Fall99 | 0.9956 | --- | 90 | 0.0\% | - | 65 | 90 | 0.0\% | --- | 71 | 90 | 0.0\% | -6 | -9.0\% | 1 | -24.18 | <0.0001 |
|  | Winter00 | 0.9870 | --- | 82 | 7.9\% | --- | 61 | 89 | 0.0\% | --- | 67 | 82 | 7.9\% | -6 | -9.7\% | 0 | -16.88 | $<0.0001$ |
|  | Spring00 | 0.9482 | --- | 92 | 0.0\% | --- | 66 | 92 | 0.0\% | --- | 73 | 92 | 0.0\% | -7 | -11.2\% | 3 | -5.57 | $<0.0001$ |
|  | Summer00 | 0.9903 | --- | 94 | 0.0\% | --- | 70 | 84 | 10.6\% | --- | 78 | 84 | 10.6\% | -9 | -13.7\% | 5 | -15.90 | <0.0001 |
|  | Fall00 | 0.9806 | --- | 61 | 1.6\% | --- | 65 | 56 | 9.7\% | --- | 75 | 56 | 9.7\% | -9 | -14.1\% | 1 | -13.46 | <0.0001 |

[^6]Appendix 5 - Pearson Correlations Among All Analytes Within Sampling Location
 OOO OO O 0000000000000
侯








## C0000000000000000000


























## 



讙荌呂
웅ㅁㅇㅇ․․․


웅

0,0000000000000000000000000000000000000 O

































 OO

|  | F\%om |
| :---: | :---: |
|  | \% |
|  | \% \% |
|  |  |
|  | - |
|  |  |
|  |  |
|  |  |
|  |  |
|  | - |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |
|  | \%otide |
|  | - |
|  | -0000000 |
|  |  |


 0.00000000000000009








 $\stackrel{0}{0}$




o o o o o o o o o o o o o o

0 O．



















 0 on o す。




「영훙․․ ． 응엉
 ONO
$\stackrel{\otimes}{8} \underset{\sim}{0}$ $\rightarrow 0$
 $\stackrel{\circ}{\circ} \stackrel{\circ}{0}_{\circ}^{\circ}$ N゙ゥ

$\bigcirc$
$\wedge_{0}^{\infty}$ 옹

－7 $\bigcirc$

N్ HN N N
꿍욱 국
응국구
웅
으응
NO 욱
CNJ

৪운 국
\％
웅
An N No
겅
엉 긍
，



긍N․

○ONO
O






















## 

膍氟
























$000000000000000 \%$
















| -1 |  |
| :---: | :---: |
| 0 |  |
| 0 | 0 |
| 0 | 0 |


-00
$-0_{0}^{\circ}$
${ }^{-1} 0$

ON N్N N్N

$\bigcirc 00$

벙 꾹
స్ 끙
갱N웅
웅 운 구
응 겡
OO．
No


N゙ NO
웅 9
응 걱 겅
った
ल⿵人一⿰工凡
ल 응 귱
Nָస్సN
on Nô
응응
© 앙 긍
ㅇㅇ
o웅웅
or



$\bigcirc$



















 moon oito
人




 ～mo






# Part B: <br> Air Contaminants and Emergency <br> Department Visits for Asthma in the Bronx and Manhattan 

LIST OF TABLES ..... iv
LIST OF FIGURES ..... v
SUMMARY ..... 7
INTRODUCTION ..... 11
METHODS ..... 13
RESULTS ..... 21
DISCUSSION ..... 27
CONCLUSIONS AND RECOMMENDATIONS ..... 37
REFERENCES ..... 39
AUTHORS AND ACKNOWLEDGEMENTS ..... 45
TABLES ..... 47
FIGURES ..... 63

## LIST OF TABLES

Table 1. Hospital Emergency Department Visits by Residents of Bronx and Manhattan Study Areas ..... 48
Table 2. Mean daily emergency department visits for asthma and control conditions ..... 49
Table 3. Mean concentrations of air pollutants and bioaerosols measured in the Bronx and Manhattan. ..... 50
Table 4. Relative risks for asthma ED visits as a function of 5-day mean air pollution and bioaerosols from single-pollutant models ..... 51
Table 5. Relative risks from regressions based on daily maximum hourly exposures ..... 53
Table 6. Relative variance in asthma ED visits explained by variables included in the model for daily 8-hour maximum $\mathrm{O}_{3}$. ..... 54
Table 7. Relative risks for mean change in contaminant concentrations for models that did not include temperature as a covariate ..... 55
Table 8. Results from Poisson regressions of asthma ED visits on pollens and molds. ..... 56
Table 9. Relative risks for asthma ED visits as a function of 5-day mean air pollution from two-pollutant models ..... 57
Table 10. Correlations among key air pollutants in the Bronx study community. ..... 58
Table 11. Relative risks for control ED visits in relation to the five pollutants that showed significant associations with asthma ED visits in the Bronx. ..... 59
Table 12. Relative risks for asthma ED visits from single-pollutant models, stratified by gender ..... 60
Table 13. Relative risks for asthma ED visits from single-pollutant models, stratified by age. ..... 61

## LIST OF FIGURES

Figure 1. Air Monitoring Locations in Manhattan and Bronx ........................................................................ 64
Figure 2. Map of Study Areas and Hospitals Contributing Emergency Department Data ................................ 65
Figures 3. Seasonal Patterns of Hospital ED Admissions for Asthma Fitted with 18 DF Natural Spline, for (a)
Bronx and (b) Manhattan
Figure 4. Day-of-Week Patterns Plotted for Hospital ED Admissions for Asthma for (a) Bronx and (b) Manhattan67

Figure 5. Asthma ED Visits Plotted against Temperature for (a) Bronx and (b) Manhattan............................ 68
Figure 6. Age Distributions of Study Communities (U.S. Census 2000) .......................................................... 69
Figure 7. Relative Risk for Asthma ED Visits in Bronx and Manhattan for 14 key Contaminants for Primary
Analysis with Base-Case Model.
Figure 8. Lag Dependency of Relative Risk for Asthma in Bronx and Manhattan for Example Pollutants
$\left(\mathrm{PM}_{2.5}, \mathrm{SO}_{2}\right.$ and $\left.\mathrm{O}_{3}\right)$.

## SUMMARY

Most previous studies of acute asthma exacerbations and ambient air pollution have examined effects of only a few of the many contaminants that are found in urban air, making it difficult to determine which specific air pollutant or group of pollutants is most important in triggering hospital visits. In addition, whereas numerous studies have reported associations between daily air pollution concentrations and counts of hospital visits for asthma or other respiratory diseases, few studies have evaluated whether risks for air pollution-related hospital visits vary across communities that differ in their baseline health status.

Mid-town Manhattan and the South Bronx are separated by less than 5 miles. However, the two regions of New York City differ greatly in levels of asthma morbidity. Although these differences are likely to be caused by multiple factors, including differential access to primary care for asthma, the present study was not designed to investigate these differences. Rather, we investigated whether day-to-day variations in air pollution were associated with asthma emergency department (ED) visits in each community and compared the magnitude of the air pollution effect between the two communities. To investigate this question, we analyzed daily counts of emergency asthma visits to hospitals serving two distinct communities, one in Manhattan and the other in the South Bronx, along with daily enhanced air monitoring in each community.

We analyzed air quality and weather data collected over approximately a two year period, from January 1999 through November 2000, at two centrally located measurement stations sampling a broad range of contaminants. Emergency department data on asthma visits for the corresponding dates were collected from the 22 hospitals throughout New York that served the communities surrounding the air monitoring stations. Data for hospital patients who lived in zip code areas within approximately 1.5 miles of either measurement site were extracted. Figure 1 depicts the location of the monitoring stations and adjacent areas for health data. (Note that in the Bronx, the measurement site was moved during the study period; Figure 1 shows both sites.)

Using these data, we compared the magnitude of the relationships between daily asthma ED visits and air pollution and bioaerosol concentrations across the two communities, and examined relative impacts of multiple pollutants. In addition, we explored the lag-dependency of the asthma response, age and sex stratification, and whether effects were evident for control outcomes (i.e., ED visits for causes not likely to be related to air quality). We used Poisson regression to test for effects of 14 key air contaminants on daily ED visits, with control for temporal cycles, temperature, and day-of-week effects. The core analysis utilized the average exposure for the zero- to four-day lags. Sensitivity analyses examined individual lag effects.

Mean daily crude rates of asthma ED visits were over eight fold higher in the Bronx study area (16.9 per 100,000 persons) than in the Manhattan area ( 2.02 per 100,000 persons; Table 2). Exploring reasons for these differences was beyond the scope of the present study. Concentrations of air contaminants were generally similar in the two communities (Table 3), with mean levels tending to be slightly higher in Manhattan in most cases. Mean ozone and total pollen levels were significantly higher in the Bronx. Among 14 key pollutants examined individually in regression analyses, five had statistically significant effects on asthma ED visits in the Bronx, including daily eight-hour maximum ozone $\left(\mathrm{O}_{3}\right)$, mean daily nitrogen dioxide $\left(\mathrm{NO}_{2}\right)$, sulfur dioxide $\left(\mathrm{SO}_{2}\right)$, particulate matter with aerodynamic diameter less than 2.5 micrometers $\left(\mathrm{PM}_{2.5}\right)$ and maximum one-hour $\mathrm{PM}_{2.5}$ (Table 4). No statistically significant pollution effects were observed in the Manhattan community.

Our findings of more significant air pollution effects in the Bronx are likely to relate in part to greater statistical power for identifying effects in the Bronx where baseline ED visits were greater, but they may also reflect greater sensitivity to air pollution effects in the Bronx.

In analyses restricted to the warm season (April through October), $\mathrm{O}_{3}$ effects in the Bronx were larger and more significant than in the full-year analysis, and they were approximately double those seen in Manhattan, suggesting greater susceptibility and/or exposure to this airway irritant and pro-inflammatory agent in the Bronx. Analyses by sex suggested that the air pollution effects in the Bronx were greater among females than males (Table 12). No strong differences in effects were observed with age strata, though there was some indication of larger effects in older adults (Table 13).

In two-pollutant and three-pollutant regression models, $\mathrm{O}_{3}$ and $\mathrm{SO}_{2}$, and to a lesser extent maximum onehour $\mathrm{PM}_{2.5}$, were the most robust pollutants (Table 9). In other words, these pollutants exhibited less change in their effect estimates as additional pollutants were added to the models. It is of particular interest that we observed more robust health impacts of the daily maximum $\mathrm{PM}_{2.5}$ concentration than for the 24hour mean, suggesting that peak exposures may have larger health impacts.

Analysis of ED visits for control outcomes (largely for digestive diseases) revealed positive or zero effects for all five of the pollutants that had been shown to be associated with asthma visits. In one case, 24-hour mean $\mathrm{PM}_{2.5}$, the control effect was statistically significant. The analysis of ED visits for control outcomes may suggest the possibility of overestimates of the observed associations.

## CONCLUSIONS AND RECOMMENDATIONS

The results suggest that the criteria pollutants $\mathrm{PM}_{2.5}, \mathrm{SO}_{2}, \mathrm{O}_{3}$ and $\mathrm{NO}_{2}$ had a statistically detectable impact on acute asthma ED visits in a community with a relatively high baseline rate of acute asthma exacerbations. In two-pollutant and three-pollutant regression models, $\mathrm{O}_{3}$ and $\mathrm{SO}_{2}$, and to a lesser extent
maximum one-hour $\mathrm{PM}_{2.5}$, were the most robust pollutants. Robust effects of $\mathrm{O}_{3}$ have been seen in previous ED asthma studies (Stieb et al. 1996; Martins et al. 2002) and in hospital admissions studies of asthma and other respiratory diseases (Burnett et al. 1997). It is of particular interest that we observed more robust health impacts of daily maximum $\mathrm{PM}_{2.5}$ concentration than of the 24-hour mean, suggesting that peak exposures may have larger health impacts. These associations with health effects in the Bronx occurred at ambient air levels that are below the current short-term National Ambient Air Quality Standards (NAAQS).

The following recommendations are suggested based on the study results:

1. EPA should consider the findings in this study and others identifying respiratory health effects associated with $\mathrm{SO}_{2}$ concentrations below current standards during their review of the $\mathrm{SO}_{2}$ NAAQS.
2. Future time-series studies examining associations between ambient air pollutants and health outcomes would benefit from direct evaluation of the relationship between personal exposure and regional monitoring data.
3. More research should be conducted to try to determine if peak, short-term (e.g. hourly) elevated concentrations of $\mathrm{PM}_{2.5}$ are more strongly associated than daily average concentrations with asthma and other health endpoints. If the science is sufficiently strong, consideration should be given to the effects of short-term $\mathrm{PM}_{2.5}$ excursions in future reviews of the particulate matter NAAQS.
4. The high correlations between pollutants (including components of $\mathrm{PM}_{2.5}$ ) make it difficult in these epidemiologic studies to confidently identify critical compounds. Alternative strategies to address this question should be considered in the future.
5. Further evaluation of the statistical methods employed in time-series epidemiological studies is warranted, based on the suggestion of possible model bias indicated by our analysis of control outcomes.
6. To the extent that targeted community based asthma interventions are planned with respect to air pollution messages, higher priority should be given to communities with larger asthma burdens.

## Section 1 <br> INTRODUCTION

Asthma is a serious and growing health problem. An estimated 14.9 million persons in the United States have asthma (NHLBI 1999). The number of people with asthma increased by $102 \%$ between 1979-80 and 1993-94 (NCHS 1997). The greatest increase in prevalence and severity has been among children and young adults living in poor inner-city neighborhoods (Eggleston et al. 1999). The U.S. Department of Health and Human Services has acknowledged the seriousness of the problem by declaring asthma and environmental pollution as two of the Healthy People 2010 focus areas.

Past studies have found discernible differences in ambient concentrations of some but not all air contaminants in urban areas for sites as close as three to five miles apart. Suh et al. (1995) collected 24hour samples of sulfate, hydrogen ion, and ammonia simultaneously at seven locations in Philadelphia and an upwind monitor during the summers of 1992 and 1993. Based on an assessment of spatial variation, they concluded that a single monitoring station was adequate for sulfate (consistent with long-range transport being the dominant source); however, multiple sites were necessary to determine local outdoor hydrogen ion concentrations. Goldstein and Landovitz (1977) found a poor correlation among air monitoring sites within a metropolitan area for certain air contaminants (e.g., sulfur dioxide). Recent work by Kinney and colleagues indicates that elemental carbon particle concentrations exhibit marked spatial variations within New York City as a function of local traffic density (Kinney et al. 2000; Lena et al. 2002). These studies suggest that for certain air contaminants it is very important to measure the air contaminants within the community being studied. In the present study, all subjects resided within approximately 1.5 miles of the monitoring sites used to characterize community air quality.

Both particulate matter and $\mathrm{O}_{3}$ have been associated with respiratory impacts among asthmatics. For example, a study conducted in Seattle found a correlation between hospital emergency room visits for asthma and particulate $\left(\mathrm{PM}_{10}\right)$ air concentrations (Schwartz et al. 1993). This effect was noted even though daily $\mathrm{PM}_{10}$ concentrations never exceeded current U.S. ambient air quality standards. Among 15 studies of asthma ED visits that incorporated adequate controls for seasonal patterns, all reported at least one significant positive association involving $\mathrm{O}_{3}$ or particulate matter (Cassino et al. 1999; Delfino et al. 1996, 1998; Hernandez-Garduno et al. 1997; Jaffe et al. 2003; Jones et al. 1995; Martins et al. 2002; Stieb et al. 1996; Tenias et al. 1998; Tobias et al. 1999; Tolbert et al. 2000).

Few previous studies have investigated the association of air contaminants with acute asthma attacks in New York City. Thurston et al. (1992) studied the relationship between hospital admissions for asthma (and all respiratory admissions) and ambient acidic particulate matter and $\mathrm{O}_{3}$ concentrations during the summer in several regions in New York State. The researchers did not have air contaminant data for New York

City, but rather used data from the nearby and less urbanized city of White Plains. They found that elevation of $\mathrm{O}_{3}$, aerosol strong acidity (hydrogen ion) and sulfate were associated with increases in asthma admissions in the summer in Buffalo and New York City. However, they found the associations were weaker in Albany and the less urbanized New York City suburbs. Potential reasons for this difference may be some chemical or physical difference in the composition or mix of air contaminants in the more densely populated areas or differences in susceptibility of the populations studied. Other older studies conducted in New York City did not report an air contaminant effect on hospital visits for asthma. Greenburg et al. (1964) did not find an association between sulfur dioxide, carbon monoxide, or particulate coefficient of haze and emergency clinic visits during September and October. Goldstein and Dulberg (1981) also found no significant relationship between hospital emergency department visits for asthma and sulfur dioxide or coefficient of haze measurements during the late summer and early fall. Many studies have evaluated the correlations between asthma attacks and ambient air contaminants during only one season, which may not be representative of the impact of various air contaminants throughout the year. In addition, studies may have had limited power to detect effects.

One important factor in identifying a causal association between air contaminants and asthma is biological plausibility, such as that exhibited by contaminants known to irritate the respiratory tract. Aldehydes (e.g., acetaldehyde, acrolein, formaldehyde and propionaldehyde) represent an important class of hazardous air pollutants (HAPs) that could negatively affect asthmatics. Formaldehyde has been reported to induce asthma in some individuals exposed in occupational settings (e.g., Feinman 1988). Acute, small decreases in respiratory function (forced expiratory volume at 1 second, $\mathrm{FEV}_{1}$ ) have been reported after exposure in occupational settings (e.g., Alexandersson et al. 1982). Studies of asthmatics suggest that they may not be sensitive to formaldehyde at concentrations below those seen in occupational exposures (e.g., Harving et al. 1986). Other aldehydes, and the potential interactions of aldehydes with other ambient contaminants, have not been as well studied.

The health study presented here was designed to address two overall objectives. First, we sought to examine whether the magnitude of acute air pollution effects on acute asthma ED visits differed in two communities that had different baseline ED rates for asthma. Second, we wanted to investigate which air contaminants or mix of air contaminants was most associated with acute asthma exacerbations in each community. The study design focuses on acute exacerbations of existing asthma and does not address factors influencing asthma prevalence or development of newly-diagnosed asthma. Part A of this report presents the results of the ambient air sampling study that were used to explore the association between asthma ED visits and air pollutant concentrations. Part A compares air pollutant concentrations on a seasonal basis between sites and describes the correlation between the sites for the air contaminants, the correlations among contaminants within each site, and temporal contaminant patterns.

## Section 2

## METHODS

To address the study objectives, we developed and analyzed an approximately two-year record of daily observations on emergency department visits and air contaminant measurements in two areas of New York City. The study design was a time-series analysis of air pollutant concentrations and acute asthma exacerbations (as assessed by asthma emergency department visits). The primary hypothesis was that temporal changes in individual ambient air pollutant concentrations were associated with temporal variation in asthma emergency department visits. These associations were tested separately in each study area. A secondary hypothesis was that the nature and/or strength of associations between ambient air pollutant patterns and asthma emergency department visits differed between the two study communities.

A brief summary of the methods used for collection of air quality data and details on the methods used to collect and analyze the health data are presented in this section. A complete description of the methods used to collect and analyze ambient air contaminants is given in Part A.

## COLLECTION OF AIR QUALITY DATA

Multiple air contaminants were monitored at a centrally located site in each community. Monitored air contaminants included real-time one-hour particulate matter (PM) less than 10 micrometers ( $\mu \mathrm{m}$ ) in aerodynamic diameter $\left(\mathrm{PM}_{10}\right)$ and $\mathrm{PM}_{2.5}$ by tapered element oscillating microbalance (TEOM); daily 24hour average $\mathrm{PM}_{2.5}$ on filters using the Federal Reference Method (FRM); particle number concentrations from 0.007 to $2.5 \mu \mathrm{~m}$ aerodynamic diameter using a condensation particle counter; three-hour average organic and elemental (i.e., soot) carbon by thermal analysis; $\mathrm{PM}_{2.5}$ metals ( $\mathrm{Cr}, \mathrm{Fe}, \mathrm{Pb}, \mathrm{Mn}, \mathrm{Ni}$, and Zn ); aerosol pH (expressed as $\mathrm{H}+$ concentration, i.e., $[\mathrm{H}+]=10^{-\mathrm{pH}}$ ); aerosol sulfate; the criteria gases ozone $\left(\mathrm{O}_{3}\right)$, nitrogen dioxide $\left(\mathrm{NO}_{2}\right)$ and sulfur dioxide $\left(\mathrm{SO}_{2}\right)$ using standard real-time methods; and bioaerosols including pollen and fungal spores. Pollen and fungal spores were categorized into several large (in some cases overlapping) groups for statistical analyses, based on taxonomic and/or morphologic similarities. For pollen, the categories were tree, grass, ragweed and total pollen. For fungal spores, the categories were basidiospores, ascospores, dark mitospores, non-dark mitospores, small spores ( $<10 \mu \mathrm{~m}$ in the largest dimension), large spores (> $10 \mu \mathrm{~m}$ in the largest dimension) and total spores.

Figure 1 shows a map of the study areas and air monitoring sites. The air sampling locations were US Environmental Protection Agency (EPA) approved air monitoring stations operated by the New York State Department of Environmental Conservation (NYSDEC), augmented by additional sampling equipment operated by the New York State Department of Health (NYS DOH). In the Bronx, two sites were used in a sequential fashion. The initial Bronx sampling site was at Intermediate School (IS) 155, located at 470

Jackson Avenue. This site operated from January through July 1999, after which a construction project was initiated at the school. Accordingly, the Bronx study site was moved to Middle School (MS) 52, located at 681 Kelly Street, which provided study data from September 1999 through November 2000. The MS 52 site was approximately 0.5 miles northeast of the original IS 155 site. A comparison of results from both sites with corresponding Manhattan data (for January-July 1999 for the initial site and January-July 2000 for the new site) suggested that the results from the two sites were comparable. In Manhattan, monitoring was carried out from January 1999 through November 2000 at the Manhattan Comprehensive Night and Day High School (also known as the Mabel Dean Bacon High School), located at 240 Second Avenue. Instruments in Manhattan sampled from a rooftop approximately seven stories high; those in the Bronx sampled from a rooftop approximately four stories above the ground. For further details on data collection methods and findings, see Part A.

To perform the health analyses, it was necessary to replace missing values in the air data with estimates. Values were estimated by regression on a seasonal basis, first on the same analyte at the other site, then on correlated analytes (from either same site or other site, in order of decreasing strength of correlation).

The first regression performed was across sites. For example, sulfate values at the Manhattan site were used to predict values for missing sulfate data at the Bronx site, and vice versa. To fill in remaining missing values, predictor variables were selected by ranking the variables from strongest to weakest correlation. For ranking purposes the correlation over the entire study period was used; the correlation was not compared on a seasonal basis. Regression on a seasonal basis was performed on the original data using the different predictor variables until all missing values had been replaced. For example, 67 of the 75 missing values for sulfate at the Bronx site were estimated by regression on sulfate at the Manhattan site; the eight remaining missing values were estimated by additional regression models to fill in the remaining missing values. Correlation coefficients utilized for filling in missing values were generally greater than 0.5 . Aside from filling in the summer Bronx 1999 data, when the site was shut down and relocated, only a relatively few values had to be filled in; they generally changed the mean concentration estimates by less than $10 \%$.

## COLLECTION OF HEALTH DATA

As noted on Figure 1, the two study areas comprised six zip codes in the Bronx (10451, 10454, 10455, 10456, 10459, and 10474) and 12 zip codes in Manhattan (10001, 10003, 10009, 10010, 10011, 10012, $10014,10016,10017,10018,10020$, and 10036). To select hospitals from which to extract ED data, we first identified hospitals that served residents living in the zip codes. During the planning phase of the study, data on asthma hospital admissions from the Statewide Planning and Research Cooperative System (SPARCS) were used to identify potential study hospitals. We used SPARCS data from 1996 and 1997 to determine the number of hospital admissions for asthma at each hospital that would service the study areas. We identified 24 hospitals throughout NYC that recorded an average of at least 10 asthma admissions per
year by residents from the study area zip codes. One eligible facility (Union Hospital) was excluded because it had closed in early 1998, and another (St. Clare's Hospital and Health Center) was excluded because we were unable to obtain the necessary data from this facility. Therefore, 22 hospitals were included in the study (Figure 2).

Eight of the 22 hospitals were located in the Bronx and 14 were located in Manhattan (Figure 2). Sixteen hospitals were privately owned and operated; six were public hospitals (three in the Bronx and three in Manhattan) administered by the New York City Health and Hospitals Corporation (HHC). When several hospitals were jointly owned or merged during the course of the study, data were collected from a single source rather than from each hospital individually. The study was originally designed to collect data on all emergency department visits during one year, from January 1, 1999, through December 31, 1999, but to enhance study power and to capture data from a summer season in the Bronx, this time frame was extended to include additional data through November 2000.

The study was given 206(1)(j) designation by the New York State Commissioner of Health in late 1996. This designation allowed NYSDOH to collect the data needed for the study and facilitated the cooperation of the study hospitals. This designation confers protection on the information and reports collected, maintains confidentiality, and guarantees that the data will be used solely for the purposes of scientific research with respect to this study. By February 2002, data had been received from all 22 hospitals.

The data elements requested from the hospitals included medical record number; patient's name, date of birth, sex, race, social security number and residential street address (including zip code); source of payment; emergency department visit date; principal diagnosis code; additional diagnosis codes and hospital admission and discharge dates (if applicable). The data essential for the study were medical record number, residential street address (including zip code), emergency department visit date and principal diagnosis code. In some cases additional diagnosis codes were also provided.

SAS statistical software and SQL (Structured Query Language) were used to process the data into a consistent format. The datasets were concatenated into a master SAS dataset containing 629,227 observations and 19 data fields. Twelve fields were from the ED data provided by the hospitals (including hospital identification number, sex, ED visit date, admission and discharge dates, principal diagnosis and secondary diagnoses). Seven fields were created, including the patient's age (from the date of birth and ED visit date) and fields to identify the study areas, asthma cases and controls. The dataset used for statistical analysis did not contain any personal identifying information.

Asthma cases were obtained from ED records with a principal diagnosis ICD-9 code of 493 and, in addition, for children less than one year old, codes 466.1 (acute bronchiolitis) and 786.09 (other dyspnea
and respiratory abnormalities, including wheezing and shortness of breath). The latter were included because of the difficulty of diagnosing asthma in infants.

We also counted ED visits for a set of "control" health conditions assumed a priori to be unrelated to air pollution. By analyzing these data in relation to air pollution, we hoped to verify the absence of significant associations, thereby inferring a lack of bias in our asthma analyses. These included cases with principal diagnosis ICD-9 codes 365 (glaucoma), 366.0-366.3 (cataract), 531.0-531.3 (acute gastric ulcer), 532.0532.3 (acute duodenal ulcer), 533.0-533.3 (acute peptic ulcer), 534.0-534.3 (acute gastrojejunal ulcer), 535 (gastritis and duodenitis), 537 (disorders of stomach and duodenum), 540-543 (appendicitis or diseases of the appendix), 558 (non-infectious gastroenteritis and colitis), 574-575 (cholelithiasis), 590 (infections of the kidney) and 599 (other disorders of urethra and urinary tract).

Secondary diagnoses were not used to identify asthma cases and controls for two reasons. First, New York City HHC could only provide the primary diagnosis and the number of secondary diagnoses varied among the remaining hospitals. Second, these diagnoses could be co-existing conditions but not necessarily acute conditions related to the primary diagnosis.

## STATISTICAL ANALYSIS

S-Plus software was used to analyze associations between daily air quality and asthma ED visit counts, controlling for season, day-of-week and temperature ("confounding variables"). Although humidity was another potential confounder, it did not appear to be necessary to control separately for humidity, since it is highly correlated with season and temperature. We assessed the associations between the asthma admissions data and air pollutants both individually and in multi-pollutant models. Appropriately controlling for important confounding variables is critical to isolate the influence of air contaminants on asthma response. We used the general linear model (GLM) to perform Poisson regression. We used natural splines to control for date and temperature; we also controlled for day-of-week effects. This approach fits smooth functions (natural splines) of the asthma counts as a function of each confounding variable, which in effect should leave intact the shorter-term fluctuations in asthma counts that may be explainable in part by the air quality parameters. The GLM approach using spline smoothing has been recommended by Dominici et al. (2002) as an alternative to LOESS smoothing using generalized additive models (GAM).

Although we did not constrain the shape of the spline fitted by S-plus, we selected the number of degrees of freedom (DF) for the curve. The choice of DF affects the final result, and we made efforts to test the sensitivity of results to a range of DF choices. An appropriate choice of DF captures the variability of the response variable with regard to the confounding variable but does not "over-fit" the data at risk of erroneously attributing too much variability to the confounding variable and underestimating the risk due to air pollution.

Hospital visits for asthma vary over the year. Although some of this variation may be due to air quality variations (the subject of the present study), behavioral, physiological and other causes are thought to play a dominant role in driving seasonal patterns. For instance, there is an increase in the asthma attack rate in the fall from unknown factors (Blaisdell et al. 2002), although there is some suggestion that viral infections play an important role (e.g., Johnston et al. 1996, 2005). Thus, fitting the seasonal variability with natural spline functions is aimed at removing temporal correlations between exposure and outcome that are most likely not related to any causal relationship involving air pollution. Figures 3 a and 3 b show the relationship of asthma to day of year for the two study sites and illustrates the natural spline fit to the data using 18 degrees of freedom, which was deemed adequate to capture the observed seasonal variability in asthma.

Hospital utilization, including ED visits, is known to vary with day of week. To ensure that weekly patterns in hospital ED visits were not erroneously attributed to air pollutants (some of which also exhibit day-ofweek patterns), day-of-week effects were controlled as a class variable in GLM. Figures 4a and 4 b show the relationship of asthma to day of week for the two study sites. Note that peak visits occurred on Monday at both sites.

Temperature may also influence asthma exacerbations. Scatterplots of the raw asthma and temperature data are shown in Figures 5a and 5b. We see that asthma visits tended to be highest at lower temperatures, and lower as the temperature rose. Some or most of this relationship may reflect the same seasonal factors already controlled by the spline on date. However, because $\mathrm{O}_{3}$ and other pollutants are correlated with temperature, we included temperature as a confounding variable, smoothed with a natural spline using 3 DF, which was adequate to capture the smooth curve.

A Poisson regression model was selected to quantify the relationships between asthma and air quality. Poisson regression is a standard model for dealing with a dependent variable of counts. The Poisson regression assumes a log-linear response between the dependent variable and the linear predictor. In this case, the dependent variable is asthma ED counts, and the linear predictor is the sum of air quality and confounding variables included in the model.

For a simple Poisson model with outcome $Y$ regressed on one pollutant $X$, the assumption of a log-linear response implies that

$$
\log (Y)=\alpha+\beta^{*} \mathrm{X}
$$

$$
\begin{gathered}
\mathrm{Y}=\operatorname{EXP}(\alpha) * \operatorname{EXP}(\beta * X)=\mathrm{C} * \mathrm{e}^{\beta \mathrm{x}} \\
\mathrm{RR}=\operatorname{EXP}(\beta * \mathrm{X}) \\
\mathrm{Y}=\mathrm{C} * \mathrm{RR}
\end{gathered}
$$

where
Y is the outcome variable (e.g., daily asthma ED counts);
X is the level of the air pollution variable;
$\alpha$ is the intercept term;
$\beta$ is the slope relating changes in asthma ED counts to changes in pollutant concentration;
C is the baseline level of daily asthma ED counts in the absence of air pollution; and
$R R$ is relative risk, or the proportional increase in daily asthma ED counts for an increase of X in pollutant concentration.

So, for an increase in pollutant concentration of a value $X$, the ED count increases by a factor of RR. Thus, the model assumes a constant proportional increase in asthma counts per unit increase in pollution.

## ANALYSIS STRATEGY

Although a large number of air quality parameters were measured in the study, we chose to examine 14 key parameters or groups of parameters that, by consensus among the co-investigators, were considered a priori to carry the greatest potential risk with respect to asthma exacerbations (Table 3). To minimize multicolinearity as well as excessive statistical testing, we kept the list as short as possible. We included daily maximum eight-hour moving average $\mathrm{O}_{3}$, daily mean $\mathrm{NO}_{2}$ and $\mathrm{SO}_{2}$, daily 24-hour average $\mathrm{FRM} \mathrm{PM}{ }_{2.5}$, daily one-hour maximum $\mathrm{PM}_{2.5}$, daily 24-hour average $\mathrm{PM}_{10-2.5}$ (i.e., coarse PM , the particulate matter fraction between $\mathrm{PM}_{10}$ and $\mathrm{PM}_{2.5}$ ), $\mathrm{PM}_{2.5}$ sulfate, $\mathrm{PM}_{2.5}, \mathrm{PM}_{2.5}$ acidity $\left(\mathrm{H}^{+}\right), \mathrm{PM}_{2.5}$ elemental carbon ("soot"), $\mathrm{PM}_{2.5}$ organic carbon, total $\mathrm{PM}_{2.5}$ metals (predominately nickel and iron), total carbonyl compounds (predominately formaldehyde, acetaldehyde and acetone), total pollen and total mold spores. Each "pollutant" was tested individually in the Poisson regression model to assess the independent health impacts of each air quality parameter.

At issue early on was whether separate models would be fit for the two study sites, or whether a consistent model form (e.g., the choices of degrees of freedom for splines on confounding variables) should be applied to both sites. For instance, in comparing the response of pollutants between Los Angeles and New York City, it would be expected that seasonal patterns and temperature dependencies would differ and could thus require separate models. In contrast, for two communities within New York City, it is not obvious why separate models would be required. Further, the goal of comparing air pollution effects across
the two communities argued for using a consistent model. Accordingly, for the main analyses, an identical model form was used in both communities, with confounding variables handled as noted above, and with the air pollutant expressed as the mean of lags zero through four. In other words, we expressed air pollution exposures as the five-day mean ending on the corresponding day of asthma data. We chose to use the mean of lags zero through four based in part on previous studies that suggested that most asthma ED visits occur 24 to 72 hours following the onset of symptoms (Canny et al. 1989). In addition, exploratory analyses indicated that positive associations tended to exist within this lag range but the pattern of lags differed somewhat across locations. By averaging across relevant lags, we sought to smooth out these patterns and thereby provide a consistent basis for comparison across locations. Details on the exploratory analyses of lag dependency are presented below, under Results.

## Section 3

RESULTS

The number of asthma and control ED visits and the total number of visits by hospital are enumerated in Table 1. The hospital-specific ED data presented here may include data for residents from both study areas, since residents from the Bronx may have visited a Manhattan hospital and vice versa. In constructing analytical datasets, these data were separated by residential location into two separate ED data files.

Average daily asthma ED visits differed substantially for residents of the two study communities (Table 2). Overall, daily asthma ED visits were six times higher in the Bronx study area (43 per day) than in the Manhattan study area ( 7.2 per day). To put these numbers in perspective, Table 2 also gives the Census 2000 population counts in the two study areas. By dividing the daily asthma counts by the population, we can estimate crude daily rates of asthma ED visits overall and by sex and age for each community. The crude daily asthma ED rates for all ages were 16.9 per 100,000 persons for the Bronx and 2.02 per 100,000 persons for Manhattan. Population age structures were quite different in the two communities, with larger proportions of younger persons in the Bronx versus Manhattan (Figure 6).

Means and standard deviations for the 14 key air contaminants are given in Table 3. In general, mean concentrations were fairly similar across the two communities. Exceptions included maximum eight-hour $\mathrm{O}_{3}$, which was $33 \%$ higher in the Bronx ( 28 parts per billion, ppb) than in Manhattan ( 21 ppb ), and total pollen, which was almost $60 \%$ higher in the Bronx ( 20.8 grains $/ \mathrm{m}^{3}$ ) than in Manhattan ( 13.1 grains $/ \mathrm{m}^{3}$ ). The distributions of pollen and mold concentrations were highly skewed (data not shown), with many days of zeros and brief periods of very high levels. More detailed analysis of the air quality data and the differences across communities is presented in Part A.

## SINGLE-POLLUTANT MODELS

Table 4 and Figure 7 present relative risks (RRs) and 95\% confidence intervals (CIs) for Manhattan and the Bronx for the 14 air contaminants. Relative risks are computed relative to a fixed "increment" in contaminant concentration. The CIs on the RRs were computed based on taking plus or minus $1.96 \times \mathrm{SD}$ (regression slope) and then re-computing RRs at each of the CI bounds. For the results presented in Table 4a, we have used the two-community mean concentrations given in Table 3 as the exposure increment. It should be noted that the choice of concentration increments used to compute the RRs is an arbitrary one. The mean is a common choice. However, RRs based on variability metrics, such as the standard deviation of daily pollution concentrations, may be more appropriate for expressing health impacts associated with typical day-to-day changes in contaminant concentrations and for comparing the strength of effects among pollutants whose absolute air concentrations differ. To illustrate this, we re-computed the

RRs and $95 \%$ confidence intervals for the five pollutants with significant RRs in the Bronx based on the two-community average standard deviation of the respective air pollutant concentration (Table 4 b ). Changing the scaling increment in this way does not affect the statistical significance of the RRs.

The results in Table 4a indicate that the individual contaminants with statistically significant effects (based on the $95 \%$ CI excluding $\mathrm{RR}=1.00$ ) in the Bronx were Max $8 \mathrm{hr} \mathrm{O}_{3}$ (RR 1.06; 95\% CI 1.01-1.10), $\mathrm{NO}_{2}$ (RR $1.10 ; 95 \%$ CI 1.01-1.18), $\mathrm{SO}_{2}\left(\operatorname{RR} 1.11 ; 95 \%\right.$ CI 1.06-1.17), $\mathrm{FRM} \mathrm{PM}_{2.5}$ (RR 1.05; 95\% CI 1.01-1.10), and Max $\mathrm{PM}_{2.5}$ (RR 1.09; 95\% CI 1.03-1.15). Although the magnitudes of the RR estimates in Manhattan were often similar to those observed in the Bronx, no statistically significant air pollution effects were observed for Manhattan.

When the standard deviation increment was used for the five pollutants with significant RRs in Table 4a, the relative magnitudes of the pollutant-specific RRs decreased compared with the RRs based on the mean increment (Table 4b). When standard deviation increments were used, the $\mathrm{SO}_{2}$ effect stands out as the largest of those pollutants that were statistically significant in the Bronx.

In additional exploratory analyses, we examined whether maximum hourly concentrations of $\mathrm{NO}_{2}$ or $\mathrm{SO}_{2}$ or maximum three-hour elemental (soot) carbon or organic carbon (again, averaged over lag zero to four days) yielded substantially different results than were observed above for 24-hour mean concentrations. Table 5 shows these results. Slightly stronger associations were observed for these daily maximum results for $\mathrm{NO}_{2}$ and elemental carbon in the Bronx than were observed using the 24 hour means (Table 4 a ). In contrast with the daily-mean elemental carbon effect, the maximum three-hour elemental carbon association attained statistical significance.

## CONFOUNDER EFFECTS

As noted above, the basic Poisson regression model included a single pollutant along with three "confounder" variables: a natural spline function of date with 18 degrees of freedom, a natural spline of temperature with 3 degrees of freedom and a weekday term. To assess the importance of these confounder variables, we examined the contribution of each variable to the model in terms of its ability to explain variations in ED visits. As an example, we present these results for the $\mathrm{O}_{3}$ model in the Bronx in Table 6. In the generalized linear modeling framework of S-Plus, the variance explained by a variable is characterized by the deviance divided by the degrees of freedom (Kaz Ito, personal communication). As seen in Table 6, for the single-pollutant model including $\mathrm{O}_{3}$, the date and weekday variables were the strongest predictors of asthma ED visits, followed by $\mathrm{O}_{3}$ itself.

Temperature had a very low explanatory power in the $\mathrm{O}_{3}$ model, implying that it was probably not necessary to be included as a covariate. To examine what influence the inclusion of temperature had on key
air pollution regression results, we re-ran the regressions of asthma ED visits on $\mathrm{SO}_{2}$, maximum $\mathrm{PM}_{2.5}$ and $\mathrm{O}_{3}$ without temperature in the models. There were no important changes in the RR estimates for these pollutants without temperature in the models (Table 7). However, in the interest of being conservative, temperature was retained as a covariate in all other results presented here.

## SEASONAL AND THRESHOLD ANALYSES

Because bioaerosols, $\mathrm{SO}_{2}$ and $\mathrm{O}_{3}$ are seasonal contaminants that reach high airborne concentrations primarily in the warm (bioaerosols and $\mathrm{O}_{3}$ ) or cold $\left(\mathrm{SO}_{2}\right)$ season in New York City, we re-estimated these effects in a data subset restricted to the relevant season. In addition to eliminating some statistical noise that may be introduced by including non-peak season data, seasonal restriction also can help reduce residual confounding by seasonal patterns (Burnett et al. 1994). For $\mathrm{SO}_{2}$, there was no change in results when we reran the regression within a winter data subset (data not shown), and these results are not discussed further.

In the case of $\mathrm{O}_{3}$, the basic regression model was re-run using data for the seven-month period April 1 through October 31, which yielded a larger and more significant RR in the Bronx (1.08; 95\% CI 1.03-1.12) but a smaller RR in Manhattan (1.04; 95\% CI 0.91-1.19) for a 24 ppb change in $\mathrm{O}_{3}$ concentration (Table $4 a)$. These results are contrasted to those obtained for $\mathrm{O}_{3}$ in the full-year analysis of the Bronx $(1.06 ; 95 \%$ CI 1.01-1.10) and Manhattan (1.06; 95\% CI 0.93-1.20) for a 24 ppb change in $\mathrm{O}_{3}$ concentration. Based on these results, it would appear that the warm-season effect of $\mathrm{O}_{3}$ on ED visits for asthma is about twice as high in the Bronx as it is in Manhattan, although the Bronx CI includes the Manhattan RR estimate. Further, because the RR represents the proportional increase in asthma ED visits associated with a fixed increase in $\mathrm{O}_{3}$ (here, 24 ppb ), and because average asthma ED visits were six times higher in the Bronx study area than in the Manhattan area, the number of $\mathrm{O}_{3}$-related ED visits in the Bronx would be estimated to be about 12 times higher than in Manhattan.

To investigate whether there was evidence for $\mathrm{O}_{3}$ effects below a daily maximum eight-hour moving average of 80 ppb -the National Ambient Air Quality Standard—we repeated the summer-season regression after eliminating days with concentrations above this level. There were only five such days in the Bronx during the study period (fewer than $1 \%$ of all days). The $\mathrm{O}_{3} \mathrm{RR}$ from this regression model (RR $1.09 ; 95 \%$ CI 1.03-1.15) was similar to that obtained above for the summer-season regression over the full concentration range (RR.1.08; 95\% CI 1.03-1.12).

We also re-ran regressions for selected pollen and mold categories within warm-season data subsets (Table 8). The results from the seasonal analysis did not differ from the results observed in the full annual analysis, with RR estimates generally close to 1.00 . Because of the highly episodic temporal patterns of pollen and mold concentrations observed in this study, regression modeling may not represent the optimal
analytical strategy for analyzing health effects of these contaminants. However, because the focus of the present report is on air pollutants and their effects on asthma, we did not pursue this issue further.

## LAG DEPENDENCY OF SINGLE-POLLUTANT MODELS

Exploratory regression analysis of the Bronx and Manhattan data revealed distinct differences between the two regions in asthma effects as a function of lag. This observation prompted a more thorough investigation of the lag structures in each community, with testing of lags from zero to four days. This approach is consistent with past studies indicating that ED hospital visits for asthma peak for patients with symptoms beginning 24 to 72 hours prior to arrival (Canny et al. 1989).

To illustrate the observed lag structures, Figure 8 plots the single-lag relative risks in the Bronx and Manhattan for three pollutants $\left(\mathrm{PM}_{2.5}, \mathrm{SO}_{2}\right.$ and $\left.\mathrm{O}_{3}\right)$, with error bars representing $95 \%$ CIs. As before, the RR for each contaminant was calculated for a concentration increment corresponding to the mean contaminant concentration in Manhattan and the Bronx, given in Table 3, with the same concentration used for both community RRs (thus, all differences in the RRs between the two areas arise from differences in the calculated regression slopes). The data suggest differences exist between the two areas and among contaminants in the lag-dependencies of the responses. $\mathrm{PM}_{2.5}$ produced a maximum response in the Bronx at a zero-day lag, but in Manhattan at a one-day lag. $\mathrm{SO}_{2}$ produced a maximum response in the Bronx at a two-day lag, and in Manhattan at a three-day lag. $\mathrm{O}_{3}$ maximum Bronx response occurred at a one-day lag, whereas in Manhattan the response decreased sharply from zero- to four-day lags.

For the 14 contaminants listed in Table 3, the analysis of lags zero to four yielded three statistically significant RRs for a specific-day lag in the Bronx, and one in Manhattan. In the Bronx, $\mathrm{NO}_{2}$ was significant at a zero-day lag, maximum $\mathrm{PM}_{2.5}$ was significant at a four-day lag and $\mathrm{SO}_{2}$ was significant at a two-day lag. In Manhattan, daily average $\mathrm{PM}_{2.5}$ was significant at a one-day lag (Figure 8, not all data shown).

Because the patterns of lag-dependency differed among pollutants and locations, choosing a single-day lag to apply uniformly to both communities would have a profound impact on the conclusions regarding the differences in air pollution effects across the two communities. In an effort to mitigate this problem, comparisons across communities presented in this report were based on a model that regressed ED visits on the multi-day average concentrations computed over lags zero to four for each air quality parameter (i.e., a five-day distributed lag giving equal weight to the day of the ED visit and the preceding four days).

## MULTI-POLLUTANT MODELS

As noted above, significant single-pollutant results were seen for $\mathrm{PM}_{2.5}, \mathrm{O}_{3}, \mathrm{NO}_{2}$ and $\mathrm{SO}_{2}$ in the Bronx dataset. We sought to investigate whether these individual pollutant effects were independent of one
another, or on the other hand, whether results for individual pollutants were confounded by omission of other pollutants. This issue can be addressed by including two or more pollutants simultaneously into the regression model and examining whether the pollutant-specific effects change compared with the singlepollutant models presented above.

Pairs of contaminants with significant effects in the single-pollutant models were tested simultaneously in the basic model that included controls for date, temperature and day of week. We report in Table 9 the relative risk and $95 \%$ CI for these results for the Bronx and Manhattan. To assist in interpretation of these results, Table 10 gives the correlations among the individual pollutant concentrations.

For the Bronx, co-pollutant regression results for $\mathrm{O}_{3}$ and $\mathrm{SO}_{2}$ were robust to all other pollutants considered (Table 9). The univariate $\mathrm{O}_{3} \mathrm{RR}$ was 1.06 (Table 4); with co-pollutants, the RR ranged from 1.04 to 1.06. The univariate $\mathrm{SO}_{2} \mathrm{RR}$ was 1.11 (Table 4); with co-pollutants, the RR ranged from 1.09 to 1.11. FRM $\mathrm{PM}_{2.5}$ was robust to $\mathrm{O}_{3}$ but not to other co-pollutants tested (RR 1.05 in both univariate and bivariate $\mathrm{O}_{3}$ models; RR reduced to approximately 1.00 with other co-pollutant models). The high correlation between FRM $\mathrm{PM}_{2.5}$ and maximum $\mathrm{PM}_{2.5}$ made it difficult to assess their relative importance. Still, the results in Table 9 do suggest that maximum $\mathrm{PM}_{2.5}$ was the stronger predictor of asthma ED visits in this study. Compared with the effects of co-pollutants on RRs for $\mathrm{FRM} \mathrm{PM}_{2.5}$, the maximum $\mathrm{PM}_{2.5}$ RRs did not diminish to the same extent when co-pollutants were factored in. This robustness argues for a greater independent impact of maximum $\mathrm{PM}_{2.5}$ concentrations compared with 24-hour average $\mathrm{PM}_{2.5} . \mathrm{NO}_{2}$ effects were robust to $\mathrm{O}_{3}$ but were not robust to the other pollutants. As was seen for the single-pollutant results presented earlier, none of the Manhattan results were statistically significant.

To summarize the results presented in Table 9, the $\mathrm{O}_{3}$ effect on daily asthma ED visits was robust to inclusion of the other pollutants into the model. The $\mathrm{SO}_{2}$ effect was also robust. $\mathrm{PM}_{2.5}$ exhibited somewhat less robustness. Of the two $\mathrm{PM}_{2.5}$ metrics, maximum hourly $\mathrm{PM}_{2.5}$ was the more robust. Note that in threepollutant models including $\mathrm{O}_{3}, \mathrm{SO}_{2}$ and maximum $\mathrm{PM}_{2.5}$, the RRs for $\mathrm{O}_{3}$, maximum $\mathrm{PM}_{2.5}$ and $\mathrm{SO}_{2}$ effects remained virtually unchanged from their univariate magnitudes. $\mathrm{NO}_{2}$ effects were robust only to inclusion of $\mathrm{O}_{3}$ in the model.

## ANALYSIS OF CONTROL VARIABLES

To evaluate the specificity of the air pollution effects observed for asthma visits, we repeated the analysis of five key air pollutants with control-cause ED visits as the outcome variable. If there was no association with air pollution, one would expect non-significant RRs centered at 1.00 for the control outcome. Results for the Bronx and Manhattan are presented in Table 11. Of the five pollutants that had significant univariate effects on asthma in the Bronx, one, FRM PM $_{2.5}$, had significant effects on the control outcome in the Bronx. Positive but non-significant effects were seen for the remaining pollutants, except $\mathrm{O}_{3}$. There was no
evidence of any effect of $\mathrm{O}_{3}$ on control ED counts. Analysis of Manhattan control outcome data showed a similar but somewhat weaker positive bias for the same five pollutants.

To determine whether patients diagnosed with one of the study control conditions also had a secondary diagnosis of asthma, additional diagnosis codes were examined for the nine hospitals from which the data were available. The nine hospitals had reported a total of 193,300 emergency department visits, including 11,451 asthma visits (i.e., asthma as principal diagnosis) and 11,087 control visits (i.e., one of the control conditions as principal diagnosis). A total of 49 ED visits were made by patients with a control condition as the principal diagnosis and a secondary diagnosis of asthma, accounting for $0.4 \%$ of patients diagnosed with one of the control conditions. The control conditions for which this was most frequently the case were non-infectious gastroenteritis $(\operatorname{ICD} 9=558 ; N=13)$, urinary tract infection $(\operatorname{ICD} 9=599 ; N=11)$, and ulcers (ICD9 $=531-535 ; N=7$ ). Secondary asthma diagnoses do not appear to be a likely contributor to the observed trend of control-outcome RRs > 1 for the five air pollutants investigated in the Bronx.

## ANALYSIS OF SEX-SPECIFIC RESPONSES

To examine whether males and females responded differently to pollution, we repeated the basic general linear modeling for five key pollutants in data subsets stratified by sex. Larger and/or more significant RRs in one sex or the other would be taken as evidence for differential responses. Results of the stratified analysis are presented in Tables 12a (the Bronx) and 12b (Manhattan). In the Bronx, the RRs were larger and more significant for females than for males for all pollutants except $\mathrm{O}_{3}$. Results in Manhattan were generally similar, with higher RRs for females (except for $\mathrm{O}_{3}$ ), though none of the RRs were statistically significant. These results suggest that female asthmatics may be more susceptible than males to the acute effects of air pollution.

## ANALYSIS OF AGE-SPECIFIC RESPONSES

Health data were broken down by age group for regression against each of five key pollutants (Tables 13a and 13b). Age was split into five strata, $0-4,5-18,19-34,35-64$, and over 65 . Because the numbers of cases in each age stratum were relatively small for these analyses (refer to Table 2), there was considerable variability in results across ages. Although some of the largest RRs occurred in the very young and very old, for most pollutants it was the older adult age group (35-64) that appeared to have larger and more significant effects. These findings should be taken as only suggestive, however, since study power was limited for testing effects within age strata. Larger studies would be needed to derive firm conclusions about age-specific effects.

## Section 4

## DISCUSSION

This study evaluated daily asthma emergency department visits in relation to a range of air contaminants over a two-year period in two communities that differed substantially in baseline asthma morbidity - Lower Manhattan and the South Bronx. Primary objectives were identifying which air pollutants were most consistently associated with asthma ED visits and comparing the magnitude of air pollution effects across the two communities. The study design did not address factors influencing asthma prevalence or development of newly diagnosed asthma.

In Poisson regression models that included controls for longer-term and day-of-week temporal cycles and temperature, five of 14 key air contaminants were significantly associated with daily asthma ED visits at the $\mathrm{P}<0.05$ level in the Bronx community only. Significant pollutants included daily eight-hour maximum $\mathrm{O}_{3}$, mean daily $\mathrm{NO}_{2}, \mathrm{SO}_{2}, \mathrm{PM}_{2.5}$ and maximum one-hour $\mathrm{PM}_{2.5}$, all expressed as the mean of lags zero to four. In secondary analyses of effects for peak hourly $\mathrm{SO}_{2}$ and $\mathrm{NO}_{2}$ concentrations or peak three-hour elemental (soot) carbon and organic carbon, all but organic carbon were significantly associated with asthma visits.

In two- and three-pollutant regression models, $\mathrm{O}_{3}, \mathrm{SO}_{2}$, and to a lesser extent, maximum one-hour $\mathrm{PM}_{2.5}$ were the most robust pollutants. In other words, these pollutants exhibited less change in their effect estimates as additional pollutants were added to the models. The relative risk for $\mathrm{O}_{3}$ did not change appreciably when we repeated the analysis after eliminating all days with maximum eight-hour moving average $\mathrm{O}_{3}$ above 80 ppb , the National Ambient Air Quality Standard. Robust effects of $\mathrm{O}_{3}$ have been seen in previous ED asthma studies (Stieb et al. 1996; Martins et al. 2002) and in hospital admissions studies of asthma and other respiratory diseases (Burnett et al. 1997). It is of particular interest that we observed more robust health impacts of the daily maximum $\mathrm{PM}_{2.5}$ concentration compared with the 24 -hour mean, suggesting that peak exposures may have larger health impacts. Prior studies have also suggested that stronger associations between particulate matter exposure and asthma morbidity are observed with shorter particulate matter averaging times (e.g., Delfino et al. 1998; Michaels and Kleinman 2000).

When the Bronx relative risks and 95\% confidence intervals were re-computed based on the pollutant standard deviations rather than on means, $\mathrm{SO}_{2}$ effects appeared more prominent than the other pollutants. RRs calculated using the standard deviation normalize all pollutant concentration increments relative to their observed variability and convey a better sense of the health effects associated with typical day-to-day variations in concentrations.

Although concentrations of air contaminants were generally similar in the two communities, health impacts of air pollution were more apparent in the Bronx than in Manhattan. Among the 14 pollutants examined individually in regression analyses, five had statistically significant effects on asthma ED visits in the Bronx. Although the magnitudes of the RR estimates in Manhattan were often similar to those observed in the Bronx, no statistically significant air contaminant effects were observed for Manhattan.

The more prominent effects in the Bronx at least partially reflect greater statistical power for identifying effects there. Because asthma ED visits follow a Poisson distribution, the greater mean daily asthma ED counts in the Bronx would lead to reduced relative uncertainty around the effect estimates. This effect can be illustrated using the relative uncertainty of the estimate of the mean of a Poisson distribution. For a Poisson variable, the variance is equal to the mean and is referred to as lambda. Therefore, by the Central Limit Theorem, the sampling distribution of the mean lambda will have variance equal to lambda/n, or a standard error of the mean equal to sqrt(lambda/n). Expressed as the ratio of the standard error of the mean to the mean, this becomes
Sqrt(lambda/n)/lambda = 1/sqrt(lambda*n)

Thus, for a fixed sample size n , as lambda (the mean) increases, the uncertainty around the mean estimate relative to the mean diminishes as $1 /$ sqrt(lambda). In the present study, the relative uncertainty of mean (or effect) estimates in Manhattan is about 2.5 times greater than in the Bronx. This translates into greater uncertainty around effect estimates and reduced power to detect effects. However, in addition to wider error bars relative to the mean, the RRs in Manhattan were also closer to 1.0 than those in the Bronx for the five pollutants that were significantly related to asthma visits, which does support the idea that effects might be larger in the Bronx.

In analyses restricted to the warm season (April through October), the $\mathrm{O}_{3} \mathrm{RR}$ in the Bronx was approximately double that of Manhattan (although the CIs overlap), suggesting greater susceptibility to this airway irritant and pro-inflammatory agent in the Bronx. Because the RR represents the proportional increase in asthma ED visits associated with a fixed increase in $\mathrm{O}_{3}$ (here, 24 ppb ), and because average asthma ED visits were six times higher in the Bronx study area than in the Manhattan area, the number of $\mathrm{O}_{3}$-related ED visits in the Bronx would be estimated to be about 12 times higher than in Manhattan.

A variety of factors could contribute to differences in susceptibility to air pollution effects as measured by asthma emergency department visits across the two study communities, if such differences exist. Factors that might play a role include differential access to primary asthma care, nutritional differences, co-morbid conditions or other factors related to general socio-economic status. Lack of adequate primary asthma care may lead to higher baseline asthma morbidity and to greater use of the ED as the first line of care during a
severe exacerbation. Along with other community-level factors, such as nutritional status and comorbidities, this could manifest as a greater proportional response to a given increase in air pollutant levels. Data were not available to evaluate these hypotheses in this report.

Variation in effects of unmeasured co-pollutants, such as indoor allergens, environmental tobacco smoke or local traffic and industrial emissions, might also influence the apparent differences in acute asthma ED responses to ambient air pollution observed in the two communities. Increased exposure to such local measured pollutants could directly increase baseline asthma morbidity and might also indirectly increase the response to changes in ambient air pollutants by increasing airway inflammation and hyperresponsiveness to acute airway irritants. Data were not available to address these possible effects in this report.

Analyses by sex suggested that the air pollution effects in the Bronx were greater among females than males. Medical utilization for acute asthma exacerbations has been observed to be greater for females among adults, and greater for males among children (e.g., Schatz and Camargo 2003; Schatz et al. 2004). Schatz et al. (2004) concluded that increased asthma hospitalization among boys was a reflection of prevalence rather than increased asthma severity in boys versus girls. However, the larger relative increase in acute ED visits observed in females in this study with fixed incremental increases in air pollutant concentrations suggests possible sex differences due to factors other than prevalence, such as differences in severity, disease management or access to care. Data were not available to further evaluate this hypothesis in this report.

No strong differences in effects were observed with age strata, though there was some indication of larger effects in older adults but not the elderly. Differences in the response of adults and children with asthma to different air pollution exposures have been observed in studies designed to investigate age-related effects (e.g., Atkinson et al. 1999; Sinclair and Tolsma 2004). In the present study, our ability to resolve agerelated differences in asthma response to air pollution exposure may have been too limited by the required stratified sub-analyses.

To evaluate the specificity of the air pollution effects observed for asthma visits, we analyzed the relationships between air pollutants and control-cause ED visits. Of the five pollutants that had significant univariate effects on asthma in the Bronx, one, FRM $\mathrm{PM}_{2.5}$, had significant effects on the control outcome. Positive but non-significant effects were seen for the remaining pollutants, except $\mathrm{O}_{3}$. There was no evidence of any effect of $\mathrm{O}_{3}$ on control ED counts. These results could suggest some degree of overestimating risk in the analysis.

We explored this apparent risk overestimation effect with additional analyses. For those hospitals where a secondary diagnosis was available, there was no indication that a diagnosis of asthma secondary to one of the control conditions contributed to the tendency toward positive associations with control outcomes. When control conditions were stratified by organ system, there was a similar tendency toward positive associations between the same five pollutant variables and control conditions grouped as gastrointestinal or urinary tract. Based on these follow-up analyses, we were not able to discern a clear explanation for the apparent positive model bias suggested by the analysis of control outcome variables.

In the current study, significant associations were observed between asthma ED visits and four criteria air pollutants- $\mathrm{O}_{3}, \mathrm{SO}_{2}$, FRM $\mathrm{PM}_{2.5}$ and $\mathrm{NO}_{2}$. The results for $\mathrm{O}_{3}$ and $\mathrm{SO}_{2}$ remained significant in models considering the simultaneous effects of two and three pollutants. Other recent studies have found similar associations using time-series methods similar to those used here. However, finding associations between any of these pollutants and acute asthma ED visits varies among studies, as does the degree to which associations are robust to inclusion of additional pollutants in the models.

Ozone has been associated with acute asthma ED visits in several recent studies (Fauroux et al. 2000; Galan et al. 2003; Cassino et al. 1999; Stieb et al. 2000; Jaffe et al. 2003), but it was not significantly associated with ED visits in single-pollutant models in a similar number of studies (Lierl and Hornung 2003; Atkinson et al. 1999; Tolbert et al. 2000; Thompson et al. 2001; Jalaludin et al. 2004; Sinclair and Tolsma 2004). The association observed in Galan et al. (2003) remained significant for $\mathrm{O}_{3}$ after the inclusion of $\mathrm{NO}_{2}$ and pollen. Stieb et al. (2000) found that an association between $\mathrm{O}_{3}$ and all respiratory ED visits persisted in a multi-pollutant model with $\mathrm{NO}_{2}$ and $\mathrm{SO}_{2}$, but a separate multi-pollutant model for asthma ED visits was not reported. Conversely, inclusion of $\mathrm{O}_{3}$ in multi-pollutant models did not modify the significant effect of $\mathrm{SO}_{2}, \mathrm{NO}_{2}, \mathrm{PM}_{10}$ or CO (Atkinson et al. 1999) or grass pollen (Lewis et al. 2000), suggesting that any association between $\mathrm{O}_{3}$ and acute asthma ED visits was small compared with the other pollutants in these studies.

In a majority of recent studies, $\mathrm{SO}_{2}$ has been significantly associated with acute asthma ED visits in singlepollutant models (Michaud et al. 2004; Atkinson et al. 1999; Stieb et al. 2000; Tolbert et al. 2000; Chew et al. 1999; Thompson et al. 2001; Jaffe et al. 2003; Norris et al. 1999), although several studies failed to observe a significant association (Fauroux et al. 2000; Galan et al. 2003; Cassino et al. 1999; Donoghue and Thomas 1999; Sinclair and Tolsma 2004). The $\mathrm{SO}_{2}$ association persisted in two-pollutant models including $\mathrm{NO}_{2}, \mathrm{O}_{3}, \mathrm{CO}, \mathrm{PM}_{10}$ and black smoke in one study (Atkinson et al. 1999) and was not modified by the inclusion of $\mathrm{PM}_{1}$ (i.e., ultrafine PM) in another study (Michaud et al. 2004), but the association was not robust to inclusion of $\mathrm{PM}_{10}$ (Galan et al. 2003) or benzene (Thompson et al. 2001) in other studies.

Compared with other criteria pollutants, $\mathrm{NO}_{2}$ and other nitrogen oxides have been included in fewer recent time-series analyses of acute asthma ED visits, but they tend to show mixed results in single-pollutant models, similar to the overall results observed for $\mathrm{O}_{3}$. Significant associations have been reported in several studies (Galan et al. 2003; Atkinson et al. 1999; Tobert et al. 2000; Thompson et al. 2001) but have not been found in others (Fauroux et al. 2000; Cassino et al. 1999; Jaffe et al. 2003; Jalaludin et al. 2004; Sinclair and Tolsma 2004; Norris et al. 1999). When $\mathrm{NO}_{2}$ was significantly associated with acute asthma ED visits in single-pollutant models and was included in multi-pollutant models in these studies, its association with asthma ED visits (Galan et al. 2003; Atkinson et al. 1999) or all respiratory ED visits (Stieb et al. 2000) has generally persisted, although the association did not persist in one study after inclusion of benzene in the model (Thompson et al. 2001).

The relationship observed in recent time-series studies between changes in ambient particulate matter and acute asthma ED visits is complicated by the diversity of exposure indicators representing airborne particulates. Ambient particulate matter has most often been assessed as $\mathrm{PM}_{10}$, but other metrics have been used, including $\mathrm{PM}_{10-2.5}$ (coarse fraction), $\mathrm{PM}_{2.5}, \mathrm{PM}_{1}$, total suspended particulates, black smoke and ultra fines assessed on a particle count, surface area or light scatter basis. Collectively, ambient particulate matter has been significantly associated with acute asthma ED visits in a majority of recent studies (Galan et al. 2003; Atkinson et al. 1999; Stieb et al. 2000; Chew et al. 1999; Thompson et al. 2001; Jaffe et al. 2003, Jalaludin et al. 2004; Sinclair and Tolsma 2004; Norris et al. 1999). A few studies that included particulate matter in models of acute asthma ED visits have not observed a significant association (Slaughter et al. 2004; Michaud et al. 2004; Lierl and Hornung 2003; Tolbert et al. 2000), although Lierl and Hornung (2003) did report that the association of acute asthma ED visits with pollen levels was stronger on high $\mathrm{PM}_{10}$ days than on low $\mathrm{PM}_{10}$ days, suggesting an enhancement of the pollen effect by $\mathrm{PM}_{10}$. The association between $\mathrm{PM}_{10}$ and acute asthma ED visits has generally persisted in the few studies that included other criteria pollutants in the model (Galan et al. 2003; Atkinson et al. 1999), although the association was not robust to inclusion of benzene in the model in one study (Thompson et al. 2001).

The studies mentioned above give no clear indication that the association of ambient particulate matter and acute asthma exacerbations can be attributed to a specific size fraction. However, there has been relatively little previous investigation of the association between acute asthma ED visits and fine-fraction $\left(\mathrm{PM}_{2.5}\right)$ particulate matter components, as was done in this study. In the current study, the association observed with acute asthma exacerbations in the Bronx was stronger for the $\mathrm{PM}_{2.5}$ fraction than for $\mathrm{PM}_{10}$. Tolbert et al. (2000) reported preliminary findings using one year of data from the Aerosol Research and Inhalation Epidemiology Study (ARIES) "supersite" air monitoring station in Atlanta. They found no significant associations between asthma ED visits and 10 particulate matter parameters- $\mathrm{PM}_{10}, \mathrm{PM}_{2.5}, \mathrm{PM}_{10-2.5}$, ultrafine particle number, ultrafine particle surface area and five $\mathrm{PM}_{2.5}$ constituents (metals, acidity, sulfates, organic carbon and elemental carbon). Sinclair and Tolsma (2004), investigated acute asthma
visits to ambulatory care clinics in a private health-care network in relation to two years of data from the Atlanta ARIES supersite. Among all the same particulate matter variables, they reported significant associations between ultrafine particle surface area and adult asthma, and between child asthma and four particulate matter variables $\left(\mathrm{PM}_{10-2.5}, \mathrm{PM}_{10}, \mathrm{PM}_{2.5}\right.$ elemental carbon, $\mathrm{PM}_{2.5}$ organic carbon). Fine fraction acidity and sulfate were significantly associated with acute asthma ED visits in single-pollutant models in another study (Stieb et al. 2000).

In the current study, changes in bioaerosol levels and acute asthma ED visits were generally unrelated or only weakly associated. This contrasts somewhat with several recent studies showing significant associations between temporal patterns of ambient pollens or fungal spores and asthma ED visits (Lierl and Hornung 2003; Stieb et al. 2000; Lewis et al. 2000; Tobias et al. 2003, 2004; Dales et al. 2000, 2003). Average daily pollen and fungal-spore counts in these studies were generally several-fold higher than levels observed in the current study. Population prevalence of allergen sensitization varies geographically, depending on the geographic distribution of plants, animals and fungi that produce allergens (e.g., Arruda et al. 1991; Call et al. 1992; Gelber et al. 1993; Platts-Mills et al. 1995). For pollen and mold exposure to potentially have an effect on acute asthma exacerbations, individuals must be both sensitized and exposed to the relevant allergens. Some evidence indicates that prevalence of sensitization to pollen and mold allergens is relatively low in inner-city children with asthma (Kattan et al. 1997; Crain et al. 2002), which could limit study power to detect bioaerosol effects in urban environments. Our study design may not have been optimal to investigate direct associations between acute asthma exacerbations and ambient bioaerosol levels or potential effects modification between bioaerosols and ambient chemical pollutants, due to the relatively low bioaerosol levels we observed and their highly skewed temporal distribution.

Associations were observed in this study between acute asthma ED visits and changes in daily air pollutant levels. For criteria air pollutants, these associations were found for levels that were generally near or below the National Ambient Air Quality Standards (NAAQS; see Part A). The annual average $\mathrm{NO}_{2}$ and $\mathrm{SO}_{2}$ standards were not exceeded during the study at either site, and neither was the 24-hour $\mathrm{SO}_{2}$ standard. The maximum 24-hour FRM $\mathrm{PM}_{2.5}$ observation during the study did not exceed the 24 -hour standard, but the annual averages at each site were approximately equal to or slightly above the standard ( $15-16 \mu \mathrm{~g} / \mathrm{m}^{3}$ versus the NAAQS of 15). More than $95 \%$ of all eight-hour $\mathrm{O}_{3}$ observations fell below the 80 ppb standard, and removing the eight-hour moving average $\mathrm{O}_{3}$ observations that exceeded 80 ppb did not alter the association between incremental $\mathrm{O}_{3}$ exposure and asthma ED visits. This is consistent with the results of other recent studies that observed significant associations between acute asthma ED visits and increments in ambient air pollutant concentrations at absolute concentrations at or below the NAAQS (e.g., Fauroux et al. 2000; Michaud et al. 2004; Galan et al. 2003; Atkinson et al. 1999; Jaffe et al. 2003).

Five-day mean contaminant concentrations were used for assessing associations of pollutants and asthma emergency department visits. This was done to provide a consistent model for all pollutants at both study areas. Using five days has biological plausibility, based on both disease mechanisms and reports on when symptoms start versus visits to the ED. For instance, exposure to pollutants capable of inducing airway inflammation, such as ozone and fine particulates (Peden 2002), may promote underlying airway inflammation or hyper-responsiveness to an extent requiring medical treatment. Rodrigo (2004) reported that when airway inflammation was predominant in the progression of an asthma attack in adults, deterioration of lung function and clinical status usually occurred over a period of days or weeks prior to presenting to the emergency department. This type of asthma progression was noted in $80 \%$ to $90 \%$ of adults presenting to the emergency department with acute asthma. Canny et al. (1989) investigated the time between when asthma symptoms were first noted in pediatric patients and when they went to the ED. The average duration of symptoms before the ED visit was 41 hours; $84 \%$ went to the hospital within 72 hours and $97 \%$ within 168 hours. Sinclair and Tolsma (2004) investigated associations of various lags between pollutants and children's ambulatory care visits for asthma and noted that most of the statistically significant associations occurred with lags of three to five days, compared with zero- to two-day lags and six- to eight-day lags.

## STUDY STRENGTHS AND LIMITATIONS

The relatively high population density of the Bronx and Manhattan allowed for the central monitors to be used as an indicator for exposure for a relatively small area (i.e., the population residing within approximately 1.5 miles of the monitoring site). Furthermore, the correlation between the two monitoring sites was relatively high (i.e., greater than 0.6 ) and mean levels were very similar for most analytes, perhaps partially mitigating against exposure misclassification biases that might occur because of movement of residents throughout the greater New York City area. Nevertheless, using a central monitoring site to estimate exposure still adds some uncertainty to exposure estimates compared with personal monitoring. Personal exposure to air pollutants can be influenced not only by ambient concentrations but also by individual activity and other indoor and microenvironmental exposures (e.g., exposure to VOCs from consumer products, smoke from tobacco, candles or cooking). For pollutants such as particulate matter, these other sources can exert significant influence on personal exposure. However, ambient $\mathrm{PM}_{2.5}$ measured at central monitoring sites has been found to be correlated with average personal exposures to $\mathrm{PM}_{2.5}$ (e.g., Liu et al. 2003; Sarnat et al. 2001, 2005).

Combining data across a five-day lag window when estimating associations between changes in pollutant concentrations and acute asthma ED visits represents a trade-off between the sensitivity of the analysis to detect effects in short time intervals versus obtaining a consistent understanding of the relationship between air pollutant changes and ED visits, given lag structures that differed among air contaminants as well as between the two communities. The five-day exposure window could capture ED visits for asthma attacks
with either a slow or a sudden progression. However, using five days could also have potentially weakened or masked associations if the pollutant has a rapid onset, short lasting effect. Since a rolling five-day lag window was employed in the analysis, effects of multi-day pollution events would be captured by accumulating cases during the duration of the pollution event and during the following four days.

The lack of consistency in statistically significant effects in the two study areas adds some uncertainty to the generality of the findings. However, as discussed above, differences in the statistical power in the two study areas may have contributed to this. Similarly, the tendency for the control conditions to have odds ratios greater than 1 adds some uncertainty to the robustness of the findings. Several of the findings, particularly $\mathrm{O}_{3}$ and $\mathrm{SO}_{2}$, are strengthened by the robustness of the findings when adding in the other pollutants.

The observed associations between specific pollutants and asthma ED visits do not necessarily indicate cause and effect. One possible reason is that the association may be due to an unmeasured pollutant that covaries with the measured pollutant. For instance, Thompson et al. (2001) observed associations between $\mathrm{PM}_{10}, \mathrm{SO}_{2}, \mathrm{NO}, \mathrm{NO}_{2}, \mathrm{NO}_{\mathrm{x}}, \mathrm{CO}$ and benzene and acute asthma exacerbations in children. When adjusting for benzene, none of the other pollutants were associated with a significant effect. In addition, many other variables that can trigger an asthma attack were not controlled for in the study. It is also possible that unmeasured confounders related to indoor environmental exposures or socio-economic status variables might be contributing to variability in acute asthma exacerbations. However, within each study area, the time-series design at least partially controls for unmeasured confounders because each case acts essentially as its own control. The analysis detects marginal changes in the outcome variable relative to the baseline rate that are associated with the measured exposure variables, and the baseline rate would include effects due to unmeasured variables, such as local or indoor exposures.

Our results suggest that increases in several ambient pollutants may be associated with increased acute asthma exacerbations in a community. Because of the community-based design used in the study, uncertainty exists regarding the precise pattern of exposure to the study analytes experienced by each asthma case and the extent to which individual exposure closely matches ambient pollutant patterns. Recent data suggest that there is variation in the degree to which personal monitoring reflects concomitant ambient pollutant patterns. In studies from Baltimore and Boston comparing urban ambient air monitoring data with personal monitoring data, ambient $\mathrm{PM}_{2.5}$ data correlated well with personal $\mathrm{PM}_{2.5}$ data, but ambient gaseous criteria pollutants did not correlate well with their corresponding personal data (Sarnat et al. 2001, 2005). Interestingly, ambient gaseous pollutants (particularly $\mathrm{SO}_{2}, \mathrm{O}_{3}$ and CO ) were correlated with personal $\mathrm{PM}_{2.5}$ data, particularly the personal monitoring data for $\mathrm{PM}_{2.5}$ components associated with ambient sources (e.g., sulfate). The authors suggest that some respiratory effects associated with ambient variation in gaseous criteria pollutants in time-series studies might actually be detecting effects of personal $\mathrm{PM}_{2.5}$
exposure, with the ambient gaseous concentrations acting as $\mathrm{PM}_{2.5}$ surrogates. Obtaining acute asthma cases through ED utilization made it impractical to consider personal exposure monitoring in this study, so we cannot investigate this potential surrogate effect further. Study designs that retain the power of communitybased time-series analyses but incorporate personal air monitoring to complement ambient monitoring data would be desirable.

Some missing data were estimated by extrapolation from the same analyte at the other monitoring site or another analyte that was correlated with the analyte for the missing data at the same monitoring site. This adds some additional uncertainty to the measurements, but the effect on the mean exposure estimates appeared to be small and therefore unlikely to change the conclusions.

## Section 5

## CONCLUSIONS AND RECOMMENDATIONS

The results suggest that the criteria pollutants $\mathrm{PM}_{2.5}, \mathrm{SO}_{2}, \mathrm{O}_{3}$ and $\mathrm{NO}_{2}$ had a statistically detectable impact on acute asthma ED visits in a community with a relatively high baseline rate of acute asthma exacerbations. In two-pollutant and three-pollutant regression models, $\mathrm{O}_{3}$ and $\mathrm{SO}_{2}$, and to a lesser extent maximum one-hour $\mathrm{PM}_{2.5}$, were the most robust pollutants. In other words, these pollutants exhibited less change in their effect estimates as additional pollutants were added to the models. Robust effects of $\mathrm{O}_{3}$ have been seen in previous ED asthma studies (Stieb et al. 1996; Martins et al. 2002) and in hospital admissions studies of asthma and other respiratory diseases (Burnett et al. 1997). It is of particular interest that we observed more robust health impacts of daily maximum $\mathrm{PM}_{2.5}$ concentration than of the 24 -hour mean, suggesting that peak exposures may have larger health impacts. These associations with health effects in the Bronx occurred at ambient air levels that are below the current short-term National Ambient Air Quality Standards.

The following recommendations are suggested based on the study results:

1. EPA should consider the findings in this study and others identifying respiratory health effects associated with $\mathrm{SO}_{2}$ concentrations below current standards during their review of the $\mathrm{SO}_{2}$ NAAQS.
2. Future time-series studies examining associations between ambient air pollutants and health outcomes would benefit from direct evaluation of the relationship between personal exposure and regional monitoring data.
3. More research should be conducted to try to determine if peak, short-term (e.g. hourly) elevated concentrations of $\mathrm{PM}_{2.5}$ are more strongly associated than daily average concentrations with asthma and other health endpoints. If the science is sufficiently strong, consideration should be given to the effects of short-term $\mathrm{PM}_{2.5}$ excursions in future reviews of the particulate matter NAAQS.
4. The high correlations between pollutants (including components of $\mathrm{PM}_{2.5}$ ) make it difficult in these epidemiologic studies to confidently identify critical compounds. Alternative strategies to address this question should be considered in the future.
5. Further evaluation of the statistical methods employed in time-series epidemiological studies is warranted, based on the suggestion of possible model bias indicated by our analysis of control outcomes.
6. To the extent that targeted community based asthma interventions are planned with respect to air pollution messages, higher priority should be given to communities with larger asthma burdens.

## REFERENCES

Alexandersson R, Kolomodin-Hedman B, Hedenstierna G. 1982. Exposure to formaldehyde: effects on pulmonary function. Arch. Environ. Health. 37:279-84.
Arruda LK, Rizzo MC, Chapman MD, Fernandez-Caldas E, Baggio D, Platts-Mills TA, Naspitz CK. 1991. Exposure and sensitization to dust mite allergens among asthmatic children in Sao Paulo, Brazil. Clin. Exp. Allergy. 21(4):433-39.

Atkinson RW, Anderson HR, Stachan DP, Bland AJM, Bremmer SA, Ponce de Leon A. 1999. Short-term associations between outdoor air pollution and visits to accident and emergency departments in London for respiratory complaints. Eur. Resp. J. 13: 257-65.

Blaisdell CJ. Weiss SR. Kimes DS. Levine ER. Myers M. Timmins S. Bollinger ME. 2002. Using seasonal variations in asthma hospitalizations in children to predict hospitalization frequency. J. Asthma. 39(7):567-75.
Burnett RT, Brook JR, Yung WT, Dales RE, Krewski D. 1997. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. Environ Res. 72(1):24-31.

Burnett, RT, Dales RE, Raizienne ME, Krewski D., Summers, PW, Roberts GR, Raad-Young M, Dann T, Brook J. 1994. Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals. Environ. Res. 65:172-94.
Call RS, Smith TF, Morris E, Chapman MD, Platts-Mills TA. 1992. Risk factors for asthma in inner city children. J. Pediatr. 121(6):862-66.

Canny, G. J., J. Reisman, R. Healy, C. Schwartz, C. Petrou, A. S. Rebuck, H. Levison. 1989. Acute Asthma: Observations Regarding the Management of a Pediatric Emergency Room. Pediatrics 83: 507-12.

Cassino C, Ito K, Bader I, Ciotolo C, Thurston G, Reibman J. 1999. Cigarette smoking and ozoneassociated emergency department use for asthma by adults in New York City. Am. J. Respir. Crit. Care Med. 159: 1773-79.
Chew FT, Goh DYT, Ooi BC, Saharom R, Hui JKS, Lee BW. 1999. Association of ambient air-pollution levels with acute asthma exacerbation among children in Singapore. Allergy. 54: 320-29.

Crain EF, Walter M, O’Connor GT, Mitchell H, Gruchalla RS, Kattan M, Malindzak GS, Enright P, Evans R III, Morgan W, Stout JW. 2002. Home and allergic characteristics of children with asthma in seven US urban communities and design of an environmental intervention: the inner-city asthma study. Environ. Health Perspect. 110: 939-45.

Dales RE, Cakmak S, Burnett RT, Judek S, Coates F, Brook, J. 2000. Influence of ambient fungal spores on emergency visits for asthma to a regional children's hospital. Am. J. Resp. Crit. Care Med. 162:2087-90.

Dales RE, Cakmak S, Judek S, Dann T, Coates F, Brook J Burnett RT. 2003. The role of fungal spores in thunderstorm asthma. Chest 123:745-50.

Delfino RJ, Coate BD, Zaiger RS, Seltzer JM, Street DH, Koutrakis P. 1996. Daily asthma severity in relation to personal ozone exposure and outdoor fungal spores. Am. J. Respir. Crit. Care Med. 154:633-41.

Delfino RJ, Zeiger RS, Seltzer JM, Street DH. 1998. Symptoms in pediatric asthmatics and air pollution: differences in effects by symptom severity, anti-inflammatory medication use and particulate averaging time. Environ. Health Perspect. 106(11): 751-61.
Dominici F, McDermott A, Zeger SL, Samet JM. 2002. On the use of generalized additive models in timeseries studies of air pollution and health. Am. J. Epidemiol. 156:193-203.

Donoghue AM, Thomas M. 1999. Point source sulphur dioxide peaks and hospital presentations for asthma. Occup. Environ. Med. 56: 232-36.
Eggleston, PA, TJ Buckley, PN Breysse, M. Wills-Karp, SR Kleeberger, and JJ Jaakkola. 1999. The environment and asthma in US inner cities. Environ. Health Perspect. 107(suppl3):439-50.
Fauroux B, Sampil M, Quenel P, Lemoullec Y. 2000. Ozone: a trigger for hospital pediatric asthma emergency room visits. Ped. Pulmonol. 30: 41-46.
Feinman SE. 1988. Respiratory effects from formaldehyde. In Feinman SE (ed.), Formaldehyde sensitivity and toxicity, 135-48. CRC Press, Boca Raton.
Galan I, Tobias A, Banegas JR, Aranguez E. 2003. Short-term effects of air pollution on daily asthma emergency room admissions. Eur. Resp. J. 22: 802-808.
Gavett SH, Koren HS. 2001. The role of particulate matter in exacerbations of atopic asthma. Int. Arch. Allergy Immunol. 124:109-12.
Gelber LE, Seltzer LH, Bouzoukis JK, Pollart SM, Chapman MD, Platts-Mills TA. 1993. Sensitization and exposure to indoor allergens as risk factors for asthma among patients presenting to hospital. Am. Rev. Respir. Dis. 147(3):573-78.

Goldstein IF, Dulberg EM. 1981. Air pollution and asthma: search for a relationship. J. Air Pollut. Control Assoc. 31:370-76.

Goldstein IF, Landovitz L. 1977. Analysis of air pollution patterns in New York City-I. Can one station represent the large metropolitan area? Atmos. Environ. 11:47-52.
Greenburg L, Field F, Reed JI, Erhardt CL. 1964. Asthma and temperature change. Arch. Environ. Health 8:642-47.

Harving H, Korsgaard J, Dahl R, Pedersen OF, Molhave L. 1986. Low concentrations of formaldehyde in bronchial asthma: a study of exposure under controlled conditions. Brit. Med. J. 293:310.
Hernandez-Garduno E, Perez-Neria J, Paccagnella AM, Pina-Garcia M, Munguia-Castro M, CatalanVazquez M, Rojas-Ramos M. 1997. Air pollution and respiratory health in Mexico City. J. Occup Environ Med. 39(4): 299-307.
Jaffe DH, Singer ME, Rimm AA. 2003. Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991-1996. Environ Res. 91: 21-28.

Jalaludin BB, O’Toole BI, Leeder SR. 2004. Acute effects of urban ambient air pollution on respiratory symptoms, asthma medication use, and doctor visits for asthma in a cohort of Australian children. Environ Res. 95: 32-42.

Johnston SL, Pattemore PK, Sanderson G, Smith S, Campbell MJ, Josephs LK, Cunningham A, Robinson BS, Myint SH, Ward ME, Tyrrell DA, Holgate ST. 1996. The relationship between upper respiratory infections and hospital admissions for asthma: a time-trend analysis. Am. J. Respir. Crit. Care Med. 154(3 Pt 1):654-60.

Johnston NW, Johnston SL, Duncan JM, Greene JM, Kebadze T, Keith PK, Roy M, Waserman S, Sears MR. 2005. The September epidemic of asthma exacerbations in children: A search for etiology. J. Allergy Clin. Immunol. 115(1):132-38.
Jones GN, Sletten C, Mandry C, Brantley PJ. 1995. Ozone level effect on respiratory illness: an investigation of emergency department visits. South Med J. 88(10):1049-56.

Kattan M, Mitchell H, Eggleston P, Gergen P, Crain E, Redline S, Weiss K, Evans R, Kaslow R, Karcsmar C,Leickly F, Malveaux F, Wedner HJ. 1997. Characteristics of inner-city children with asthma: the national cooperative inner-city asthma study. Ped. Pulmonol. 24: 253-62.

Kinney PL, Aggarwal M, Northridge ME, Janssen NA, Shepard P. 2000. Airborne concentrations of PM(2.5) and diesel exhaust particles on Harlem sidewalks: a community-based pilot study. Environ Health Perspect. 108(3):213-18.
Lena TS, Ochieng V, Carter M, Holguin-Veras J, Kinney PL. 2002. Elemental carbon and PM(2.5 )levels in an urban community heavily impacted by truck traffic. Environ Health Perspect. 110(10):100915.

Lewis SA, Corden JM, Forster GE, Newlands M. 2000. Combioned effects of aerobiological pollutants, chemical pollutants and meteorological conditions on asthma admissions and A\&E attendances in Derbyshire UK, 1993-1996. Clin. Exp. Allergy 30: 1724-32.

Lierl MB, Hornung RW. 2003. Relationship of outdoor air quality to pediatric asthma exacerbations. Ann Allergy Asthma Immunol. 90: 28-33.
Liu L-JS, Box M, Kalman D, Kaufman J, Koenig JQ, Larson T, et al. 2003. Exposure assessment of particulate matter for susceptible populations in Seattle, WA. Environ Health Perspect. 111:90918.

Martins LC, Latorre Mdo R, Saldiva PH, Braga AL. 2002. Air pollution and emergency room visits due to chronic lower respiratory diseases in the elderly: an ecological time-series study in Sao Paulo, Brazil. J. Occup. Environ Med. 44(7): 622-27.

Michaels RA and Kleinman MT. 2000. Incidence and apparent health significance of brief airborne particle excursions. Aerosol Science Technol. 32: 93-105.

Michaud J-P, Sinclair Grove J, Krupitsky D. 2004. Emergency department visits and "vog"-related air quality in Hilo, Hawai'i. Environ Res. 95: 11-19.

National Center for Health Statistics (NCHS). 1997. Current estimates from the National Health Interview Survey, 1990. Vital and Health Statistics 10(194).

National Heart, Lung, and Blood Institute (NHLBI). 1999. Data Fact Sheet. Asthma Statistics. Public Health Service, National Institutes of Health, Bethesda, MD.

Norris G, YoungPong SN, Koenig JQ, Larson TV, Sheppard L, Stout JW. 1999. An association between fine particles and asthma emergency department visits for children in Seattle. Environ Health Perspect. 107: 489-93.

Peden, D. 2002. Pollutants and asthma: role of air toxics. Environ Health Perspect. 110 (Suppl 4):565-68.
Platts-Mills TA, Sporik R, Ingram JM, Honsinger R. 1995. Dog and cat allergens and asthma among school children in Los Alamos, New Mexico, USA: altitude 7,200 feet. Int. Arch. Allergy Immunol. 107(1-3):301-303.

Rodrigo GJ, Rodrigo C, Hall JB. 2004. Acute asthma in adults A review. Chest 125:1081-102.
Sarnat JA, Brown KW, Schwartz J, Coull BA, Koutrakis P. 2005. Ambient gas concentrations and personal particulate matter exposures. Implications for studying health effects of particles. Epidemiology 16(3):385-95.

Sarnat JA, Schwartz J, Catalano PJ, Suh HH. 2001. Gaseous pollutants in particulate matter epidemiology: confounders or surrogates? Environ Health Perspect. 109(10):1053-61.

Schatz M, Camargo CA Jr. 2003. The relationship of sex to asthma prevalence, health care utilization, and medications in a large managed care organization. Ann. Allergy Asthma Immunol. 91(6):553-58.

Schatz M, Clark S, Emond JA, Schreiber D, Camargo CA Jr. 2004. Sex differences among children 2-13 years of age presenting at the emergency department with acute asthma. Ped. Pulmonol. 37(6):52329.

Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JQ. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am. Rev. Respir. Dis. 147:826-31.

Sinclair AH and Tolsma D. 2004. Associations and lags between air pollution and acute respiratory visits in an ambulatory care setting: 25-month results from the aerosol research and inhalation epidemiological study. J. Air. Waste Manage. Assn. 54: 1212-18.

Slaughter JC, Kim E, Sheppard L, Sullivan JH, Larson TV, Clairborn C. 2004. Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington. J. Expos. Anal. Environ. Epidem. Advanced online publication, 9 June 2004: 1-7.
Stieb DM, Beveridge RC, Brook JR, Smith-Doiron M, Burnett RT, Dales RE, Beaulieu S, Judek S, Mamedov A. 2000. Air pollution, aeroallergens and cardiorespiratory emergency department visits in Saint John, Canada. J. Expos. Anal. Environ. Epidem. 10: 461-77.

Stieb DM, Burnett RT, Beveridge RC, Brook JR. 1996. Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. Environ Health Perspect. 104(12):135460.

Suh HH, Allen GA, Koutrakis P, Burton RM. 1995. Spatial variation in acidic sulfate and ammonia concentrations within metropolitan Philadelphia. J. Air Waste Manage. Assoc. 45:442-52.

Tenias JM, Ballester F, Rivera ML. 1998. Association between hospital emergency visits for asthma and air pollution in Valencia, Spain. Occup. Environ. Med. 55(8):541-47.

Thompson AJ, Shields MD, Patterson CC. 2001. Acute asthma exacerbations and air pollutants in children living in Belfast, Northern Ireland. Arch. Environ Health. 56: 234-41.
Thurston GD, Ito K, Kinney PL, Lippmann M. 1992. A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers. J. Expos. Anal. Environ. Epidem. 192:429-50.

Tobias A, Campbell MJ, Saez M. 1999. Modelling asthma epidemics on the relationship between air pollution and asthma emergency visits in Barcelona, Spain. Eur. J. Epidemiol. 15(9):799-803.

Tobias A, Galan I, Banegas JR, Aranguez E. 2003. Short term effects of airborne pollen concentrations on asthma epidemic. Thorax 58: 708-10.
Tobias A, Galan I, Banegas JR. 2004. Non-linear short-term effects of airborne pollen levels with allergenic capacity on asthma emergency room admissions in Madrid, Spain. Clin. Exp. Allergy 34: 871-78.

Tolbert PE, Klein M, Busico Metzger K, Peel J, Flanders WD, Todd K, Mulholland JA, Ryan PB, Frumkin H. 2000. Interim results of the study of particulates and health in Atlanta (SOPHIA). J. Expo. Anal. Environ. Epidem. 10: 446-60.

## AUTHORS AND ACKNOWLEDGEMENTS

Daniel Luttinger (NYSDOH) was the study's principal investigator, and Lloyd Wilson, Edward Fitzgerald, Laiquat Husain, Kenneth Aldous (NYSDOH), Phillip Galvin (NYSDEC), Larry Syzdek (private aerobiology consultant) and Patrick Kinney (Columbia University) were co-investigators. Additional studydesign and statistical-analysis consultation was provided by John Hawley and Syni-An Hwang (NYSDOH), Frank Buckman (NYSDEC), Tracey Holloway (University of Michigan) and Ken Demerjian (NYSDEC). The study report was authored by Daniel Luttinger and Gregg Recer (NYSDOH) and Patrick Kinney (Columbia University). Field sampling staff included Dan Lince, Pat Palmer, Mike Rivara, Lloyd Wilson, Stephanie Selmer, James Kamara, Ellen Fitzsimmons, Dan Sharron and Stan House (NYSDOH) and Ed Marion, Mike Christophersen, Bob Murway, Bob Elburn and Frank Buckman (NYSDEC). Laboratory staff included Abdul Bari, Vincent Ferraro and Amarjit Narang (NYSDOH). Data analysis support staff included Jeff Hughes, Ying Wang, Valerie Haley, James Kamara, Kim Mazor, Karen Nolan, Dan Luttinger, Gregg Recer, Syni-An Hwang, Lloyd Wilson and Laiquat Husain (NYSDOH) and Phil Galvin and Frank Buckman (NYSDEC).

The study was funded with partial support from the Agency for Toxic Substances and Disease Registry (ATSDR) as part of Cooperative Agreement V50/ATV200002-11 and from the New York State Energy and Research Development Authority (NYSERDA).

TABLES

Table 1. Hospital Emergency Department Visits by Residents of Bronx and Manhattan Study Areas

| Hospital | Asthma <br> Visits* | Control <br> Visits** | All-Cause <br> Visits |
| :---: | :---: | :---: | :---: |
| Bellevue Hospital Center $\dagger$ | 1658 | 1875 | 65,465 |
| Beth Israel Medical Center | 1808 | 1728 | 44,441 |
| Bronx Lebanon Hospital, Concourse | 7111 | 5280 | 85,316 |
| Cabrini Medical Center | 135 | 224 | 5237 |
| Harlem Hospital Center $\dagger$ | 548 | 259 | 8748 |
| Jacobi Medical Center (formerly Bronx Municipal Hospital |  |  |  |
| Center) $\dagger$ | 991 | 251 | 16,399 |
| Lenox Hill Hospital | 143 | 324 | 5202 |
| Lincoln Medical and Mental Health Center $\dagger$ | 16,754 | 9164 | 220,470 |
| Metropolitan Hospital Center $\dagger$ | 403 | 341 | 9703 |
| Montefiore-Jack D. Weiler-Albert Einstein | 119 | 177 | 4225 |
| Montefiore Medical Center | 782 | 775 | 16,617 |
| Mount Sinai Hospital | 912 | 691 | 12,109 |
| New York Hospital (Cornell) | 236 | 518 | 8991 |
| New York-Presbyterian Hospital | 302 | 292 | 6397 |
| New York University Medical Center | 195 | 908 | 14,022 |
| North Central Bronx Hospital $\dagger$ | 759 | 213 | 12,474 |
| Our Lady of Mercy Medical Center | 265 | 266 | 5759 |
| St. Barnabas Hospital | 848 | 841 | 15,256 |
| Presbyterian Hospital-Allen Pavilion | 25 | 39 | 1158 |
| St. Luke's-Roosevelt Medical Center | 114 | 152 | 3980 |
| St. Luke's-Roosevelt-St. Luke's Division | 238 | 459 | 11,316 |
| St. Vincent's Hospital and Medical Center | 655 | 1111 | 27,970 |
| TOTAL IN BRONX | 29,987 | 18,974 | 422,849 |
| TOTAL IN MANHATTAN | 5014 | 6914 | 178,406 |

*Asthma case defined as primary diagnosis ICD-9 codes 493 and, for children less than one year of age, 466.1 and 786.09.
**Control defined as primary diagnosis ICD-9 codes $365,366.0-366.3,531.0-531.3,532.0-532.3,533.0-$ 533.3, 534.0-534.3, 535, 537, 540-543, 558, 574-575, 590, 599.
$\dagger$ Managed by the New York City Health and Hospitals Corporation.

Table 2. Mean Daily Emergency Department Visits for Asthma and Control Conditions(U.S. Census 2000)

| Outcome | Subgroup | Bronx |  |  | Manhattan |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Mean <br> Daily <br> Visits | Population | Crude <br> Daily Rate per $10^{5}$ | Mean <br> Daily <br> Visits | Population | Crude Daily <br> Rate per $10^{5}$ |
| Asthma | All | 43 | 254,167 | 16.9 | 7.2 | 355,655 | 2.02 |
|  | Male | 20 | 122,686 | 16.3 | 3.6 | 174,051 | 2.07 |
|  | Female | 23 | 131,481 | 17.5 | 3.7 | 181,604 | 2.04 |
|  | Ages 0-4 | 9.6 | 22,015 | 43.6 | 0.90 | 10,661 | 8.44 |
|  | Ages 5-18 | 9.8 | 71,314* | 13.7 | 1.3 | 30,361* | 4.28 |
|  | Ages 19-34 | 7.5 | 60,199* | 12.5 | 1.4 | 127,771* | 1.10 |
|  | Ages 35-64 | 14 | 81,841 | 17.1 | 3.1 | 146,960 | 2.11 |
|  | Ages 65+ | 2.2 | 18,798 | 11.7 | 0.54 | 39,902 | 1.35 |
| Control | All | 27 | 254,167 | 10.6 | 10 | 355,655 | 2.81 |

*Census age ranges were 5-19 and 20-35 for these categories, resulting in a slight underestimate of the crude rate in the 5-18 category and a slight overestimate of the crude rate in the 19-34 category.

Table 3. Mean (SD) Concentrations of Air Pollutants and Bioaerosols Measured in Bronx and Manhattan, with Two-Community Average

Note: The two-community average concentrations were used to calculate relative risks. The values represent summary statistics of all daily observations from January 1999 through November 2000.

| Air Contaminant | Bronx | Manhattan | Two-Community <br> Average (SD)* |
| :--- | :---: | :---: | :---: |
| Max 8-hour O 3 (ppm) | $0.028(0.018)$ | $0.021(0.016)$ | $0.024(0.017)$ |
| $\mathrm{NO}_{2}(\mathrm{ppm})$ | $0.031(0.010)$ | $0.037(0.008)$ | $0.034(0.0091)$ |
| $\mathrm{SO}_{2}(\mathrm{ppm})$ | $0.010(0.007)$ | $0.012(0.008)$ | $0.011(0.0072)$ |
| FRM PM $_{2.5}\left(\mu \mathrm{~g} / \mathrm{m}^{3}\right)$ | $15.0(8.35)$ | $16.7(9.08)$ | $15.85(8.719)$ |
| Max PM $2.5\left(\mu \mathrm{~g} / \mathrm{m}^{3}\right)$ | $27.6(13.5)$ | $27.6(13.5)$ | $27.62(13.52)$ |
| Coarse PM $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | $7.69(4.84)$ | $7.10(4.08)$ | $7.394(4.459)$ |
| Sulfate $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | $3.85(3.43)$ | $4.00(3.42)$ | $3.924(3.423)$ |
| pH | $5.10(0.54)$ | $5.03(0.47)$ | $5.066(0.5074)$ |
| Elemental $(\mathrm{Soot})$ Carbon <br> $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | $1.19(0.64)$ | $1.31(0.64)$ | $1.252(0.645)$ |
| Organic Carbon $\left(\mu \mathrm{g} / \mathrm{m}^{3}\right)$ | $3.23(0.81)$ | $3.06(0.83)$ | $3.144(0.822)$ |
| Total Metals $\left(\mathrm{ng} / \mathrm{m}^{3}\right)$ | $95.9(121.1)$ | $91.0(75.1)$ | $93.45(98.10)$ |
| Total Aldehydes $\left(\mu \mathrm{gg} / \mathrm{m}^{3}\right)$ | $15.92(8.82)$ | $16.20(10.62)$ | $16.06(9.717)$ |
| Total Pollen $\left(\# / \mathrm{m}^{3}\right)$ | $20.81(135.0)$ | $13.15(84.53)$ | $16.98(110.26)$ |
| Total Mold $\left(\# / \mathrm{m}^{3}\right)$ | $518.8(814.9)$ | $489.9(786.0)$ | $504.3(800.4)$ |

*The two-community averages are computed as the average of the two community-specific means (or standard deviations).

Table 4a. Relative Risks and 95\% Confidence Intervals for Asthma ED Visits as Function of 5-Day Mean Air Pollution and Bioaerosols from Single-Pollutant Models

Note: Exposure increments used to compute RRs were the two-community average concentrations (Table 3). Bold text indicates statistical significance at the 0.05 level.

| Air Contaminant | Bronx | Manhattan |
| :--- | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}$ | $\mathbf{1 . 0 6}(\mathbf{1 . 0 1 , 1 . 1 0 )}$ | $1.06(0.94,1.19)$ |
| Max 8-hour $\mathrm{O}_{3}$ (warm season) | $\mathbf{1 . 0 8}(\mathbf{1 . 0 3 , 1 . 1 2 )}$ | $1.04(0.91,1.19)$ |
| $\mathrm{NO}_{2}$ | $\mathbf{1 . 1 0}(\mathbf{1 . 0 1 , 1 . 1 8 )}$ | $0.95(0.72,1.25)$ |
| $\mathrm{SO}_{2}$ | $\mathbf{1 . 1 1}(\mathbf{1 . 0 6 , 1 . 1 7 )}$ | $0.99(0.88,1.12)$ |
| FRM PM $_{2.5}$ | $\mathbf{1 . 0 5}(\mathbf{1 . 0 1 , 1 . 1 0 )}$ | $1.04(0.94,1.15)$ |
| Max PM 2.5 | $\mathbf{1 . 0 9}(\mathbf{1 . 0 3 , 1 . 1 5 )}$ | $1.04(0.91,1.18)$ |
| Coarse PM | $1.02(1.00,1.04)$ | $1.02(0.98,1.07)$ |
| Sulfate | $1.03(1.00,1.06)$ | $1.05(0.98,1.13)$ |
| pH | $0.99(0.98,1.00)$ | $0.99(0.95,1.02)$ |
| Elemental (Soot) Carbon | $1.04(0.99,1.09)$ | $1.06(0.94,1.19)$ |
| Organic Carbon | $1.05(0.93,1.17)$ | $1.20(0.96,1.49)$ |
| Total Metals | $1.02(0.99,1.05)$ | $1.02(0.91,1.15)$ |
| Total Aldehydes | $1.02(1.00,1.04)$ | $1.03(0.96,1.10)$ |
| Total Pollen | $1.00(1.00,1.00)^{*}$ | $1.01(1.00,1.02)$ |
| Total Mold | $1.01(0.99,1.03)$ | $1.01(0.97,1.06)$ |

*When RR and CI bounds appear equal, it is due to rounding.

Table 4b. Comparison of Relative Risks (95\% Confidence Intervals) Computed Using Alternative Concentration Increments

Note: The following air pollutants were significant in the Bronx regression models (Table 4a). In the first column of results, we use the mean pollutant concentration as the RR increment (as in Table 4a). In the second column of results, we use the standard deviation pollution concentration as the RR increment. Note the change in relative size of the five RRs. Bold text indicates statistical significance at the 0.05 level.

| Air Contaminant | Mean Increments | SD Increments |
| :---: | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}$ | 1.06 (1.01, 1.10) | 1.04 (1.01, 1.07) |
| $\mathrm{FRM} \mathrm{PM}_{2.5}$ | 1.05 (1.01, 1.10) | 1.03 (1.00, 1.05)* |
| Max PM 2.5 | 1.09 (1.03, 1.15) | 1.04 (1.02, 1.07) |
| $\mathrm{NO}_{2}$ | 1.10 (1.01, 1.18) | 1.02 (1.00, 1.05)* |
| $\mathrm{SO}_{2}$ | 1.11 (1.06, 1.17) | 1.07 (1.04, 1.11) |

*Choice of increment does not alter statistical significance at the $\alpha=0.05$ level; the appearance of $95 \%$ CI including 1 is due to rounding differences between the two increments.

Table 5. Relative Risks from Regressions Based on Daily Maximum Hourly ( $\mathrm{SO}_{2}$ and $\mathrm{NO}_{2}$ ) or Daily
Maximum 3-Hour (Elemental and Organic Carbon) Exposures
Note: Bold text indicates statistical significance at the 0.05 level.

| Contaminant | Increment used to <br> calculate RR | Bronx | Manhattan |
| :--- | :---: | :---: | :---: |
| $\mathrm{NO}_{2}(\mathrm{ppm})$ | 0.0492 | $\mathbf{1 . 1 2}(\mathbf{1 . 0 4 , 1 . 2 0 )}$ | $0.97(0.75,1.25)$ |
| $\mathrm{SO}_{2}(\mathrm{ppm})$ | 0.0227 | $\mathbf{1 . 0 7}(\mathbf{1 . 0 3 , 1 . 1 2 )}$ | $0.96(0.86,1.07)$ |
| Elemental (Soot) <br> Carbon $(\mu \mathrm{g} / \mathrm{m} 3)$ | 1.9787 | $\mathbf{1 . 0 5}(\mathbf{1 . 0 1 , 1 . 0 9 )}$ | $1.05(0.95,1.16)$ |
| Organic Carbon <br> $(\mu \mathrm{g} / \mathrm{m} 3)$ | 3.7014 | $1.05(0.95,1.16)$ | $1.10(0.92,1.32)$ |

Table 6. Relative Variance in Asthma ED Visits Explained by Variables Included in Model for Daily Maximum 8-Hour $\mathrm{O}_{3}$

Note: DEV/DF represents an estimate of variance explained.

|  | $\mathrm{O}_{3}$ | Date <br> (natural spline <br> 18 degrees of <br> freedom) | Temperature <br> (natural spline <br> 3 degrees of <br> freedom) | Day of Week |
| :--- | :---: | :---: | :---: | :---: |

Table 7. Relative Risks (95\% Confidence Intervals) for Mean Change in Contaminant Concentrations for Models Excluding Temperature as Covariate

Note: Bold text indicates statistical significance at the 0.05 level.

| Contaminant | Bronx | Manhattan |
| :--- | :---: | :---: |
| $\mathrm{SO}_{2}$ | $\mathbf{1 . 1 1}(\mathbf{1 . 0 6}, \mathbf{1 . 1 7})$ | $0.99(0.88,1.11)$ |
| Max $\mathrm{PM}_{2.5}$ | $\mathbf{1 . 0 8}(\mathbf{1 . 0 3 ,} \mathbf{1 . 1 3})$ | $1.00(0.90,1.13)$ |
| Max 8-hour $\mathrm{O}_{3}$ | $\mathbf{1 . 0 6}(\mathbf{1 . 0 2 , 1 . 1 0 )}$ | $1.04(0.93,1.16)$ |

Table 8. Relative Risks (95\% Confidence Intervals) from Poisson Regressions of Asthma ED Visits on Pollen and Mold Categories

*Annual mean for each pollen or mold category was used for the increment to calculate the RR
**When RR and CI bounds appear equal, it is due to rounding

Table 9. Relative Risks (95\% Confidence Intervals) for Asthma ED Visits as Function of 5-Day Mean Air Pollution from Two-Pollutant Models

Note: Pollutants included here were those that were significant predictors of ED visits in single-pollutant models.
Exposure increments used to compute RRs were the two-community average concentrations (Table 3). Bold text indicates statistical significance at the 0.05 level.

| Contaminant | Controlled with | RR, Bronx | RR, Manhattan |
| :---: | :---: | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}$ | FRM PM 2.5 | 1.06 (1.01, 1.10) | 1.05 (0.93, 1.19) |
|  | Max PM ${ }_{2.5}$ | 1.04 (1.00, 1.09) | 1.05 (0.93, 1.19) |
|  | $\mathrm{NO}_{2}$ | 1.05 (1.01, 1.10) | 1.07 (0.94, 1.21) |
|  | $\mathrm{SO}_{2}$ | 1.05 (1.01, 1.10) | 1.06 (0.93, 1.20) |
| FRM PM 2.5 | Max 8-hour $\mathrm{O}_{3}$ | 1.05 (1.01, 1.10) | 1.03 (0.94, 1.14) |
|  | Max $\mathrm{PM}_{2.5}$ | 0.99 (0.92, 1.06) | 1.04 (0.89, 1.23) |
|  | $\mathrm{NO}_{2}$ | 1.03 (0.98, 1.09) | 1.08 (0.95, 1.23) |
|  | $\mathrm{SO}_{2}$ | 1.01 (0.96, 1.06) | 1.05 (0.94, 1.17) |
| Max $\mathrm{PM}_{2.5}$ | Max 8-hour $\mathrm{O}_{3}$ | 1.07 (1.02, 1.13) | $1.02(0.89,1.17)$ |
|  | FRM PM ${ }_{2.5}$ | 1.09 (1.00, 1.20) | 0.99 (0.79, 1.23) |
|  | $\mathrm{NO}_{2}$ | 1.07 (1.01, 1.14) | 1.10 (0.92, 1.31) |
|  | $\mathrm{SO}_{2}$ | 1.05 (0.99, 1.11) | 1.05 (0.90, 1.21) |
| $\mathrm{NO}_{2}$ | Max 8-hour $\mathrm{O}_{3}$ | 1.08 (1.00, 1.17) | $0.91(0.68,1.21)$ |
|  | FRM PM 2.5 | 1.06 (0.97, 1.16) | 0.83 (0.59, 1.17) |
|  | Max $\mathrm{PM}_{2.5}$ | 1.04 (0.96, 1.14) | 0.84 (0.59, 1.20) |
|  | $\mathrm{SO}_{2}$ | 1.02 (0.94, 1.12) | 0.95 (0.69, 1.30) |
| $\mathrm{SO}_{2}$ | Max 8-hour $\mathrm{O}_{3}$ | 1.11(1.05, 1.17) | $0.99(0.88,1.12)$ |
|  | FRM PM ${ }_{2.5}$ | 1.11 (1.04, 1.18) | 0.97 (0.85, 1.11) |
|  | Max $\mathrm{PM}_{2.5}$ | 1.09 (1.03, 1.16) | 0.98 (0.85, 1.12) |
|  | $\mathrm{NO}_{2}$ | 1.11 (1.04, 1.17) | 1.01 (0.87, 1.16) |

Table 10. Correlations among Key Air Pollutants in Bronx Study Community

|  | Max 8-hour $\mathrm{O}_{3}$ | $\mathrm{NO}_{2}$ | $\mathrm{SO}_{2}$ | FRM PM $_{2.5}$ | Max PM 2.5 |
| :--- | :---: | :---: | :---: | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}$ | 1.00 | $\cdot$ | $\cdot$ | $\cdot$ | $\cdot$ |
| $\mathrm{NO}_{2}$ | 0.03 | 1.00 | $\cdot$ | $\cdot$ | $\cdot$ |
| $\mathrm{SO}_{2}$ | -0.35 | 0.47 | 1.00 | $\cdot$ | $\cdot$ |
| $\mathrm{FRM} \mathrm{PM}_{2.5}$ | 0.19 | 0.61 | 0.45 | 1.00 | $\cdot$ |
| ${\text { Max } \mathrm{PM}_{2.5}}$ | 0.35 | 0.55 | 0.28 | 0.78 | 1.00 |

Table 11. Relative Risks (95\% Confidence Intervals) for Control ED Visits in Relation to Five Pollutants Showing Significant Associations with Asthma ED Visits in Bronx

Note: Exposure increments used to compute RRs were the two-community average concentrations (Table 3). Bold text indicates statistical significance at the 0.05 level.

|  | Bronx |  | Manhattan |  |
| :---: | :---: | :---: | :---: | :---: |
| Air Contaminant | Asthma RRs | Control RRs | Asthma RRs | Control RRs |
| Max 8-hour $\mathrm{O}_{3}$ | 1.06 (1.01, 1.10) | $1.00(0.95,1.05)$ | 1.06 (0.94, 1.19) | $1.01(0.92,1.11)$ |
| FRM PM 2.5 | 1.05 (1.01, 1.10) | 1.08 (1.02, 1.14) | 1.04 (0.94, 1.15) | 1.00 (0.92, 1.08) |
| Max $\mathrm{PM}_{2.5}$ | 1.09 (1.03, 1.15) | 1.04 (0.97, 1.11) | 1.04 (0.91, 1.18) | 1.07 (0.96, 1.19) |
| $\mathrm{NO}_{2}$ | 1.10 (1.01, 1.18) | 1.07 (0.98, 1.18) | 0.95 (0.72, 1.25) | 1.03 (0.84, 1.28) |
| $\mathrm{SO}_{2}$ | 1.11 (1.06, 1.17) | $1.02(0.96,1.10)$ | 0.99 (0.88, 1.12) | 1.01 (0.91, 1.12) |

Table 12. Relative Risks (95\% Confidence Intervals) for Asthma ED Visits from Single-Pollutant Models, Stratified by Sex

Note: Exposure increments used to compute RRs were the two-community average concentrations (Table 3). Bold text indicates statistical significance at the 0.05 level.
(a) Bronx

| Contaminant | Male | Female | All |
| :---: | :---: | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}$ | 1.06 (0.99, 1.13) | 1.06 (1.00, 1.12) | 1.06 (1.01, 1.10) |
| $\mathrm{FRM} \mathrm{PM}_{2.5}$ | 1.01 (0.95, 1.08) | 1.08 (1.02, 1.15) | 1.05 (1.01, 1.10) |
| Max PM 2.5 | 1.06 (0.98, 1.15) | 1.13 (1.05, 1.21) | 1.09 (1.03, 1.15) |
| $\mathrm{NO}_{2}$ | 1.07 (0.95, 1.19) | 1.13 (1.01, 1.26) | 1.10 (1.01, 1.18) |
| $\mathrm{SO}_{2}$ | 1.08 (1.00, 1.17) | 1.14 (1.06, 1.23) | 1.11 (1.06, 1.17) |

(b) Manhattan

| Contaminant | Male | Female | All |
| :--- | :---: | :---: | :---: |
| Max 8-hour $\mathrm{O}_{3}$ | $1.13(0.95,1.35)$ | $0.99(0.83,1.17)$ | $1.06(0.93,1.20)$ |
| FRM PM | 2.5 | $0.95(0.82,1.10)$ | $1.12(0.98,1.29)$ |
| Max PM | $1.04(0.94,1.15)$ |  |  |
| $\mathrm{NO}_{2}$ | $1.01(0.83,1.27)$ | $1.06(0.88,1.28)$ | $1.04(0.90,1.18)$ |
| $\mathrm{SO}_{2}$ | $0.75(0.51,1.11)$ | $1.16(0.80,1.69)$ | $0.95(0.72,1.25)$ |

Table 13. Relative Risks (95\% Confidence Intervals) for Asthma ED Visits from Single-Pollutant Models, Stratified by Age

Note: Exposure increments used to compute RRs were the two-community average concentrations (Table 3). Bold text indicates statistical significance at the 0.05 level.
(a) Bronx

|  | Age Category (years) |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Contaminant | 0-4 | 5-18 | 19-34 | 35-64 | 65-up |
| Max 8-hour $\mathrm{O}_{3}$ | $1.08(0.98,1.19)$ | 0.94 (0.85, 1.03) | 1.11 (1.01, 1.23) | $1.05(0.98,1.13)$ | 1.29 (1.08, 1.53) |
| $\mathrm{FRM} \mathrm{PM}_{2.5}$ | 1.00 (0.92, 1.10) | 0.99 (0.91, 1.08) | 1.05 (0.95, 1.16) | 1.14 (1.06, 1.23) | 1.01 (0.84, 1.22) |
| Max PM 2.5 | 1.04 (0.93, 1.17) | 1.03 (0.92, 1.15) | 1.12 (0.99, 1.27) | 1.14 (1.04, 1.25) | 1.07 (0.86, 1.36) |
| $\mathrm{NO}_{2}$ | 1.13 (0.96, 1.33) | 1.14 (0.97, 1.34) | 0.99 (0.82, 1.19) | 1.13 (0.99, 1.30) | 0.85 (0.61, 1.20) |
| $\mathrm{SO}_{2}$ | 1.13 (1.01, 1.26) | 1.03 (0.92, 1.16) | 1.06 (0.93, 1.21) | 1.18 (1.07, 1.30) | 1.12 (0.88, 1.42) |

(b) Manhattan

|  | Age Category (years) |  |  |  |  |
| :--- | :---: | :---: | :---: | :---: | :---: |
| Contaminant | $\mathbf{0 - 4}$ | $\mathbf{5 - 1 8}$ | $\mathbf{1 9 - 3 4}$ | $\mathbf{3 5 - 6 4}$ | $\mathbf{6 5 - u p}$ |
| Max 8-hour $\mathrm{O}_{3}$ | $1.24(0.84,1.82)$ | $1.11(0.83,1.49)$ | $0.90(0.68,1.18)$ | $1.09(0.90,1.30)$ | $0.96(0.61,1.53)$ |
| FRM PM $_{2.5}$ | $0.96(0.73,1.27)$ | $0.88(0.70,1.11)$ | $\mathbf{1 . 2 5}(\mathbf{1 . 0 1 , 1 . 5 5 )}$ | $1.06(0.91,1.23)$ | $0.91(0.63,1.33)$ |
| $\mathrm{Max} \mathrm{PM}_{2.5}$ | $0.99(0.67,1.44)$ | $0.82(0.59,1.12)$ | $1.31(0.98,1.78)$ | $1.05(0.86,1.29)$ | $0.94(0.57,1.55)$ |
| $\mathrm{NO}_{2}$ | $0.99(0.44,2.19)$ | $0.54(0.28,1.02)$ | $1.40(0.76,2.58)$ | $0.96(0.64,1.45)$ | $0.99(0.35,2.77)$ |
| $\mathrm{SO}_{2}$ | $0.82(0.59,1.15)$ | $1.03(0.77,1.37)$ | $1.01(0.76,1.35)$ | $1.04(0.86,1.25)$ | $0.88(0.57,1.37)$ |

FIGURES

Figure 1. Air Monitoring Locations in Manhattan and Bronx (squares). Shaded zip code areas indicate communities where emergency department cases resided.


Figure 2. Map of Study Areas and Hospitals Contributing Emergency Department Data

## Key to Hospital Codes

Bronx
B1: Or Lady of Mercy Medical Center
B2: North Central Bronx Hospita
B3: Mantefiore Medical Center
B4: Jaoobi Medical Center
B5: Mbntefiore Medical Center - Weiler Hospital
B6: St. Bamabas Hospital
B7: Bronx-Lebanon Hospital Center - Conoourse Div
B8: Linooln Medical Center
Manhattan
M: Presbyterian Hospital - Alen Pavilion
MR: New York - Presbyterian Hospita
MB: HarlemHospital
M: St. Luke's Roosevelt Hospital
ME: Mbunt Sina Hospital
M6: Metropolitan Hospital Certer
MF: Lenox Hill Hospital
MB: New York Hospital
Mg: St. Luke's Roosevelt Hospital - Roosevelt Div
MO: New York University Medical Center
M11: Belleve Hospita Center
M12: Beth Israd Medical Center M13: Cabrini Medical Center M14: St. Vinoent's Hospital


Figures 3. Seasonal Patterns of Hospital ED Admissions for Asthma Fitted with 18 DF Natural Spline, for (a) Bronx and (b) Manhattan



Figure 4. Day-of-Week Patterns Plotted for Hospital ED Admissions for Asthma for (a) Bronx and (b) Manhattan Note: Central line in box $=$ median. Upper and lower lines of box $=75^{\text {th }}$ and $25^{\text {th }}$ percentiles, respectively. Ends of whiskers represent $\pm 1.5 \times$ interquartile range. Lines outside of whiskers represent outlying observations.



Figure 5. Asthma ED Visits Plotted against Temperature for (a) Bronx and (b) Manhattan


Figure 6. Age Distributions of Study Communities (U.S. Census 2000)


Figure 7. Relative Risk for Asthma ED Visits in Bronx and Manhattan for 14 key Contaminants for Primary Analysis with Base-Case Model. Note: Error bars represent $95 \%$ confidence intervals on the risk. RRs calculated for mean increase in contaminant concentration (from Table 3, last column). The RRs and confidence intervals presented here are the same as those presented numerically in Table 4a.



Figure 8. Lag Dependency of Relative Risk for Asthma in Bronx and Manhattan for Example Pollutants $\left(\mathrm{PM}_{2.5}, \mathrm{SO}_{2}\right.$ and $\left.\mathrm{O}_{3}\right)$. Note: Error bars represent $95 \%$ confidence intervals on the risk.


For information on other NYSERDA reports, contact:

New York State Energy Research and Development Authority 17 Columbia Circle Albany, New York 12203-6399
toll free: 1 (866) NYSERDA local: (518) 862-1090 fax: (518) 862-1091
info@nyserda.org www.nyserda.org

A Study of Ambient Air Contaminants and Asthma in New York City

Final Report 06-02
State of New York
George E. Pataki, Governor
New York State Energy Research and Development Authority


[^0]:    ${ }^{1}$ Bari A, Dutkiewicz VA, Judd CD, Wilson LR, Luttinger D, Husain L. 2003. Regional sources of particulate sulfate, SO2, PM2.5, HCl, and HNO3, in New York, NY. Atmospheric Environment 37: 2837-2844.

[^1]:    DSO2 = sulfur dioxide (denuder)
    HION $=$ hyrdogen ion concentration $\mathrm{NO}=$ nitrogen oxide
    $\mathrm{PM} 10=\mathrm{PM}_{10}$

    CARB = organic carbon
    HCL = hydrochloric acid
    NI = nickel
    PM25 $=$ PM $_{2.5}$
    SULF = sulfate
    $\begin{array}{lll}\text { Key: } & \text { ACTL }=\text { acetaldehyde } & \text { ACTN }=\text { acetone } \\ & \text { FE }=\text { iron } & \text { FORM }=\text { formaldehyde } \\ & \text { HNO2 }=\text { nitrous acid } & \text { HNO3 }=\text { nitric acid } \\ & \text { NOX }=\text { nitrogen oxide } & \text { NO2 }=\text { nitrogen dioxide } \\ & \text { SOOT }=\text { elemental carbon } & \text { SO2 }=\text { sulfur dioxide }\end{array}$

[^2]:    DSO2 = sulfur dioxide (denuder)
    HION = hyrdogen ion concentration $\mathrm{NO}=$ nitrogen oxide
    $\mathrm{PM} 10=\mathrm{PM}_{10}$

    CARB = organic carbon
    HCL = hydrochloric acid
    NI = nickel
    PM25 $=$ PM $_{2.5}$
    
    $\begin{array}{lll}\text { Key: } & \text { ACTL }=\text { acetaldehyde } & \text { ACTN }=\text { acetone } \\ & \text { FE }=\text { iron } & \text { FORM }=\text { formaldehyde } \\ & \text { HNO2 }=\text { nitrous acid } & \text { HNO3 }=\text { nitric acid } \\ & \text { NOX }=\text { nitrogen oxide } & \text { NO2 }=\text { nitrogen dioxide } \\ & \text { SOOT }=\text { elemental carbon } & \text { SO2 }=\text { sulfur dioxide }\end{array}$

[^3]:    DSO2 = sulfur dioxide (denuder)
    HION $=$ hyrdogen ion concentration $\mathrm{NO}=$ nitrogen oxide
    $\mathrm{PM} 10=\mathrm{PM}_{10}$

    CARB = organic carbon
    HCL = hydrochloric acid
    NI = nickel
    PM25 $=$ PM $_{2.5}$
    
    $\begin{array}{lll}\text { Key: } & \text { ACTL }=\text { acetaldehyde } & \text { ACTN }=\text { acetone } \\ & \text { FE }=\text { iron } & \text { FORM }=\text { formaldehyde } \\ & \text { HNO2 }=\text { nitrous acid } & \text { HNO3 }=\text { nitric acid } \\ & \text { NOX }=\text { nitrogen oxide } & \text { NO2 }=\text { nitrogen dioxide } \\ & \text { SOOT }=\text { elemental carbon } & \text { SO2 }=\text { sulfur dioxide }\end{array}$

[^4]:    DSO2 = sulfur dioxide (denuder)
    HION $=$ hyrdogen ion concentration $\mathrm{NO}=$ nitrogen oxide
    $\mathrm{PM} 10=\mathrm{PM}_{10}$

    CARB = organic carbon
    HCL = hydrochloric acid
    NI = nickel
    PM25 $=$ PM $_{2.5}$
    
    $\begin{array}{lll}\text { Key: } & \text { ACTL }=\text { acetaldehyde } & \text { ACTN }=\text { acetone } \\ & \text { FE }=\text { iron } & \text { FORM }=\text { formaldehyde } \\ & \text { HNO2 }=\text { nitrous acid } & \text { HNO3 }=\text { nitric acid } \\ & \text { NOX }=\text { nitrogen oxide } & \text { NO2 }=\text { nitrogen dioxide } \\ & \text { SOOT = elemental carbon } & \text { SO2 }=\text { sulfur dioxide }\end{array}$

[^5]:    DSO2 = sulfur dioxide (denuder)
    HION = hyrdogen ion concentration $\mathrm{NO}=$ nitrogen oxide
    $\mathrm{PM} 10=\mathrm{PM}_{10}$

    CARB = organic carbon
    HCL = hydrochloric acid
    $\mathrm{NI}=$ nickel
    PM25 $=\mathrm{PM}_{2.5}$
    
    $\begin{array}{lll}\text { Key: } & \text { ACTL }=\text { acetaldehyde } & \text { ACTN }=\text { acetone } \\ & \text { FE }=\text { iron } & \text { FORM }=\text { formaldehyde } \\ & \text { HNO2 }=\text { nitrous acid } & \text { HNO3 }=\text { nitric acid } \\ & \text { NOX }=\text { nitrogen oxide } & \text { NO2 }=\text { nitrogen dioxide } \\ & \text { SOOT }=\text { elemental carbon } & \text { SO2 }=\text { sulfur dioxide }\end{array}$

[^6]:    ${ }^{\text {a }}$ For analytes collected on an hourly basis, daily averages were calculated for days with at least $75 \%$ valid data ${ }^{\text {b }}$ Difference=Manhattan - Bronx
    ${ }^{\text {c }}$ Non-detects were given values of $1 / 2$ the detection limit for statistical calculations
    ${ }^{d}$ Mean Difference (\%) =Mean Difference / Manhattan (using only days with daily averages available for both sites)

