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Characterization of Particulate and Gas Exposures of Sensitive Subpopulations Living in Baltimore and Boston

Petros Koutrakis, Helen H Suh, Jeremy A Sarnat, Kathleen Ward Brown, Brent A Coull, and Joel Schwartz

Includes a Commentary by the Institute's Health Review Committee

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HEI STATEMENT
Synopsis of Research Report 131

Characterization of Particulate and Gas Exposures of Sensitive Subpopulations Living in Baltimore and Boston

Epidemiologic studies conducted in a variety of locations have reported that short-term increases of particulate matter (PM) at low concentrations are associated with short-term increases in morbidity and mortality. The strongest associations have been found for individuals with compromised cardiac or respiratory function. However, personal exposure for some groups (particularly those considered to be susceptible to PM, such as children, older individuals, or those with cardiorespiratory conditions) is largely influenced by time spent indoors. This fact increases the uncertainty in using values from a fixed-site outdoor monitor as surrogate estimates of personal exposure. To adequately resolve this uncertainty, the nature of the association between outdoor particle concentrations and personal exposure levels must be more clearly assessed.

In 1998, HEI issued Request for Applications 98-1, "Characterization of Exposure to and Health Effects of Particulate Matter." At that time, most environmental epidemiologic studies had assessed exposure on the basis of ambient PM monitors at central sites. One objective of RFA 98-1 was to characterize personal exposure to PM in a variety of indoor and outdoor microenvironments and in geographic locations that differ in types and sources of particles, topography, and climate in order to determine the kind of exposure information necessary for epidemiologic studies. To address this objective, proposed studies would determine particle characteristics (eg, concentration, size, and composition) and describe the relation between personal exposure and the outdoor measurements of PM and other pollutants that typically have been used in epidemiologic time-series studies.

APPROACH

HEI funded Dr Koutrakis and his colleagues to assess the correlations between personal exposure to PM less than or equal to 2.5 μm in aerodynamic diameter ($PM_{2.5}$) and gaseous copollutants and compare these measurements with those taken at central-site monitors. Three groups of possibly susceptible individuals (children, seniors, and individuals with chronic obstructive pulmonary disease) were recruited in two cities (Boston and Baltimore) in two seasons (summer and winter). Integrated 24-hour personal exposures were measured over 8 or 12 consecutive days by using pump-driven multipollutant personal exposure monitors equipped with filters for collecting PM and by using passive Ogawa samplers for collecting gaseous pollutants. Concentrations of ambient pollutants were obtained at central monitoring sites in Baltimore and Boston by using a variety of monitors. Participants in each city, season, and group completed questionnaires that provided information about housing characteristics and about activities they were engaged in at different locations throughout the day.

Ambient and personal pollutant levels were characterized by using summary statistics, graphic analyses, and analysis of variance. Measurements below the limit of detection, including negative values, were included in all analyses. Mixed-model regression analysis was used to evaluate the associations between the concentrations of (1) ambient $PM_{2.5}$ and ambient gaseous pollutants, (2) ambient pollutants and their corresponding personal exposure concentrations, (3) other ambient pollutants (gaseous and sulfate [SO_4^{2-}]), and (4) personal $PM_{2.5}$, and personal $PM_{2.5}$ (including SO_4^{2-} as a surrogate for personal $PM_{2.5}$ of ambient origin) and personal gaseous exposures.

RESULTS

The results indicate that the relation between ambient $PM_{2.5}$ levels and personal exposures to $PM_{2.5}$ varies by season, location, and home characteristics. Surprisingly, groups did not appear to differ in their exposures to the fraction of ambient $PM_{2.5}$ that contributes to personal $PM_{2.5}$, despite the presence of indoor $PM_{2.5}$ sources. Ambient concentrations of SO_4^{2-}, a component of $PM_{2.5}$, were strongly associated with personal exposures to SO_4^{2-} and also with personal exposures to $PM_{2.5}$.

Continued

This Statement, prepared by the Health Effects Institute, summarizes a research project funded by HEI and conducted by Dr Petros Koutrakis at Harvard School of Public Health, Boston MA. The following Research Report contains both the detailed Investigators' Report and a Commentary on the study prepared by the Institute's Health Review Committee.

The investigators concluded that, for a given season or city, ambient $PM_{2.5}$ and SO_4^{2-} are strong predictors of respective personal exposures and that ambient SO_4^{2-} is also a strong predictor of personal exposure to $PM_{2.5}$. Because $PM_{2.5}$ has substantial indoor sources and SO_4^{2-} does not, the investigators concluded that personal exposure to SO_4^{2-} accurately reflects exposure to ambient $PM_{2.5}$ and therefore the ambient component of personal exposure to $PM_{2.5}$ as well. This conclusion is tempered, however, by the different techniques used to measure ambient concentrations and personal exposures to $PM_{2.5}$ and SO_4^{2-}. How these differences may influence the findings is unclear.

For some gaseous pollutants measured in the study, most measurements were below the limit of detection. Particularly problematic were ozone (O_3) and sulfur dioxide (SO_2) measurements, more than 90% of which were below the limit of detection in both cities, and nitrogen dioxide (NO_2) measurements in Baltimore, 80% of which were below the limit of detection. Although the investigators handled the values below the limit of detection by using currently accepted methods, care should be taken in interpreting results with many such values.

The investigators also concluded that ambient concentrations of gaseous pollutants serve as a better surrogate for personal exposure to $PM_{2.5}$ than for personal exposure to gaseous pollutants. However, this conclusion is weakened by the high percentage of gaseous pollutant samples below the limit of detection in this study. Nevertheless, even with more sensitive measurements, the variability of removal mechanisms for gaseous pollutants in indoor environments may continue to result in weak correlations between ambient and personal gaseous data, since a major component of personal air includes indoor air. In contrast, most measurements of $PM_{2.5}$ and SO_4^{2-} were above the limits of detection, giving more confidence to analyses that depend on them.

INTERPRETATION

This study provides important information for understanding the relation between ambient measures of urban air pollution and personal exposures to the same pollutants. Ambient concentrations and personal exposures to a variety of urban air pollutants were measured in two cities, in two seasons, and in three groups of possibly susceptible individuals. The study design involved examination of important personal–ambient exposure issues for both $PM_{2.5}$ and gaseous air pollutants: differences by city, season, and sensitive subpopulations, as well as by day of the week. The study design was of high quality, with standardized measurements taken at consistent times of day. However, although investigators employed appropriate and state-of-the-art methods to monitor both personal and ambient concentrations of pollutants, some of these methods differed between cities or between measurements of ambient concentrations and personal exposures.

The investigators collected valuable information to better understand the use of ambient air pollution concentrations obtained from central monitors in the evaluation of health effects. The key results of the study and its implications are:

1. Ambient concentrations of $PM_{2.5}$ were strongly associated with personal exposure to $PM_{2.5}$, supporting the use, in epidemiologic studies, of exposure information for $PM_{2.5}$ from central monitoring sites.
2. The strength of the associations between personal exposures to $PM_{2.5}$ and ambient concentrations of $PM_{2.5}$ appeared to vary somewhat by city and season, as investigators hypothesized. In contrast, the variation among groups was not as great as was anticipated. The HEI Health Review Committee agreed that studying groups thought to be sensitive is valuable. The Committee cautioned, however, that results of this study should not be extended generally to other locations and populations, given that the groups studied did not represent the general population or even the subpopulations of which they are a part.
3. For these data, ambient concentrations of gaseous copollutants such as O_3, NO_2, and SO_2 correlated more strongly with measures of personal exposure to $PM_{2.5}$ than with personal measures of the same gaseous pollutants. On the basis of these results, the investigators recommended caution when interpreting results from previous time-series studies that included both gaseous and particulate pollutant concentrations. This caution is noteworthy. The high percentage of nonmeasurable data in this study precludes the ability to discern whether strong correlations actually do exist between ambient and personal gaseous data. On the other hand, even with a more sensitive measurement technique, the correlations observed may still be weak because of the variability in removal mechanisms for gases in indoor spaces.

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Research Report 131

HEALTH EFFECTS INSTITUTE

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HEI Statement

This Statement is a nontechnical summary of the Investigators' Report and the Health Review Committee's Commentary.

Investigators' Report

When an HEI-funded study is completed, the investigators submit a final report. The Investigators' Report is first examined by three ouside technical reviewers and a biostatistician. The report and the reviewers' comments are then evaluated by members of the HEI Health Review Committee, who had no role in selecting or managing the project. During the review process, the investigators have an opportunity to exchange comments with the Review Committee and, if necessary, revise the report.

Continued

COMMENTARY Health Review Committee

The Commentary about the Investigators' Report is prepared by the HEI Health Review Committee and staff. Its purpose is to place the study into a broader scientific context, to point out its strengths and limitations, and to discuss remaining uncertainties and implications of the findings for public health.

RELATED HEI PUBLICATIONS

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INVESTIGATORS' REPORT

Characterization of Particulate and Gas Exposures of Sensitive Subpopulations Living in Baltimore and Boston

Petros Koutrakis, Helen H Suh, Jeremy A Sarnat, Kathleen Ward Brown, Brent A Coull, and Joel Schwartz

ABSTRACT

Personal exposures to particulate and gaseous pollutants and corresponding ambient concentrations were measured for 56 subjects living in Baltimore, Maryland, and 43 subjects living in Boston, Massachusetts. The 3 Baltimore cohorts consisted of 20 healthy older adults (seniors), 21 children, and 15 individuals with physician-diagnosed chronic obstructive pulmonary disease (COPD*).The 2 Boston cohorts were 20 healthy seniors and 23 children. All children were 9 to 13 years of age; seniors were 65 years of age or older; and the COPD participants had moderate to severe physician-diagnosed COPD. Personal exposures to particulate matter with aerodynamic diameters less than 2.5 µm ($PM_{2.5}$), sulfate (SO_4^{2-}), elemental carbon (EC), ozone (O_3), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2) were measured simultaneously for 24 hours/day. All subjects were monitored for 8 to 12 consecutive days.

The primary objectives of this study were (1) to characterize the personal particulate and gaseous exposures for individuals sensitive to PM health effects and (2) to assess the appropriateness of exposure assessment strategies for use in PM epidemiologic studies.

Personal exposures to multiple pollutants and ambient concentrations were measured for subjects from each cohort from each location. Pollutant data were analyzed using correlation and mixed-model regression analyses. In Baltimore, personal $PM_{2.5}$ exposures tended to be comparable to (and frequently lower than) corresponding ambient concentrations; in Boston, the personal exposures were frequently higher. Overall, personal exposures to the gaseous pollutants, especially O_3 and SO_2, were considerably lower than corresponding ambient concentrations because of the lack of indoor sources for these gases and their high removal rate on indoor surfaces. Further, the impact of ambient particles on personal exposure (the *infiltration factor*) and differences in infiltration factor by city, season, and cohort were investigated. No difference in infiltration factor was found among the cohorts, which suggests that all subjects were exposed to the same fraction of ambient $PM_{2.5}$ for a given ambient concentration. In addition, the results show significant correlations between ambient $PM_{2.5}$ concentrations and corresponding personal exposures over time and provide further indication that ambient gaseous pollutant concentrations may be better surrogates for personal $PM_{2.5}$ exposures, especially personal exposures to $PM_{2.5}$ of ambient origin, than their respective personal exposures. These results have important implications for PM health effects studies that use regression models including both ambient $PM_{2.5}$ and gaseous pollutant concentrations as independent variables, because both parameters may be serving as surrogates for $PM_{2.5}$ exposures.

* A list of abbreviations and other terms appears at the end of the Investigators' Report.

This Investigators' Report is one part of Health Effects Institute Research Report 131, which also includes a Commentary by the Health Review Committee and an HEI Statement about the research project. Correspondence concerning the Investigators' Report may be addressed to Dr Petros Koutrakis, Exposure, Epidemiology and Risk Program, Harvard School of Public Health, Landmark Center, PO Box 15677, 401 Park Dr, Room 410, Boston MA 02215.

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INTRODUCTION

In 1997, the US Environmental Protection Agency (EPA) proposed revisions to the Clean Air Act, which, for the first time, included standards for fine PM ($PM_{2.5}$). Numerous epidemiologic studies have provided evidence of $PM_{2.5}$ toxicity, showing associations between ambient concentrations of $PM_{2.5}$ and a variety of adverse health outcomes (increased hospital admissions, increased emergency room visits, exacerbation of asthma, higher rates of lung cancer, and increased mortality; Dockery et al 1993;

Schwartz et al 1999; Sheppard et al 1999; Sarnat et al 2000; Pope et al 2002). The consistency of these findings was remarkable considering the diverse study populations, meteorologic conditions, particle composition, and variety of study designs.

Despite this consistency, there are still considerable areas of uncertainty. For example, weak correlations between outdoor PM concentrations and total personal PM exposures reported in various cross-sectional exposure studies support the notion that the ambient $PM_{2.5}$ concentrations used in epidemiologic studies are poor indicators of personal exposure (Gamble 1998; Green et al 2002). Also, the variability of personal PM exposure across individuals has led to speculation that ambient concentrations may be better estimates of exposure for some individuals or subpopulations than others (Rojas-Bracho et al 2000a).

There have also been suggestions that estimates of health risks associated with exposure to $PM_{2.5}$ may be confounded by gaseous copollutants, because ambient concentrations of $PM_{2.5}$ and gaseous copollutants are frequently correlated with each other (Lipfert and Wyzga 1999; Green et al 2002). A gaseous pollutant can be a confounder of ambient particles only if it correlates with both the exposure of interest (ie, $PM_{2.5}$) and the health outcome of interest. Investigation of the possible confounding effect of gaseous copollutants was one of the research priorities on PM health effects recommended by the US National Research Council (1998).

To date, many researchers have examined the potential for confounding using models that include ambient $PM_{2.5}$ as well as ambient concentrations of one or more gaseous pollutants (O_3, NO_2, and SO_2; Burnett et al 1999; Samet et al 2000; Schwartz 2000). Models provide an appropriate method if the pollutant measures are actual personal exposures. Because the measures used are typically ambient concentrations, however, these models rely on two assumptions: (1) the ambient pollutant concentrations included within the model are good surrogates for (ie, are well correlated with) their respective personal exposures and are poorly correlated with personal exposures to other pollutants; and (2) gaseous pollutants and $PM_{2.5}$ included in the models will capture both the independent effects of these pollutants and effectively control for any residual confounding. These previous investigations used ambient pollutant concentrations exclusively as model inputs because information concerning personal exposures to the multiple air pollutants was not available.

The recent development of a multiple-pollutant personal sampler (Demokritou et al 2001) has enabled simultaneous measurement of $PM_{2.5}$, O_3, NO_2, and SO_2, providing researchers with an opportunity to examine confounding using personal exposures. In addition, because indoor SO_4^{2-} sources are generally limited, SO_4^{2-} measurements from the $PM_{2.5}$ sampler filters provide a means of assessing exposures to particles of ambient origin.

We previously investigated associations among multiple-pollutant personal exposures and ambient concentrations for cohorts of healthy seniors, children, and individuals with COPD living in Baltimore, Maryland (Sarnat et al 2001). The 3 Baltimore cohorts were 20 healthy seniors, 21 children, and 15 individuals with physician-diagnosed COPD. Ambient 24-hour concentrations and personal exposure data were collected for $PM_{2.5}$, O_3, NO_2, and SO_2 over 8 to 12 consecutive days for each subject. The results showed significant associations between ambient $PM_{2.5}$ concentrations and corresponding ambient gaseous concentrations. Associations were weak between measures of personal exposure and ambient concentrations of the gases, however. This dissociation suggested that ambient concentrations of O_3, NO_2, and SO_2 were poor surrogates for personal exposure. Associations between personal $PM_{2.5}$ exposures and corresponding personal gaseous exposures were also weak. Together, these results suggest that confounding of $PM_{2.5}$-related health effects by these gaseous pollutants may not be likely for this location.

The results from the Baltimore multipollutant study may not be representative. Therefore, it is important to examine whether similar associations exist for subjects living in other locations, residing in homes with various building characteristics, and being exposed to different mixtures and levels of air pollutants.

In this report, we further evaluate the role of ambient O_3, NO_2, SO_2, and carbon monoxide (CO) as surrogates of personal $PM_{2.5}$ exposures and of personal exposures to $PM_{2.5}$ of ambient origin. These analyses used data from an additional exposure assessment study conducted in Boston, Massachusetts. Similarities and differences in the findings from Baltimore and Boston are also discussed.

REVIEW OF PERSONAL EXPOSURE STUDIES

The earliest personal PM exposure studies entailed measurement of a relatively large number of individuals over a very limited time. These cross-sectional studies treated each personal exposure measurement as an independent event that was influenced by individual, ambient concentration, and microenvironmental sources. Three of these studies characterized personal exposures to PM with aerodynamic diameters less than 3.5 μm ($PM_{3.5}$, or respirable PM) using similar study designs (Dockery and Spengler 1981; Sexton et al 1984; Spengler et al 1985). All three studies reported that indoor concentrations were better predictors of personal $PM_{3.5}$ exposures than outdoor concentrations, which

underscored the importance of nonambient source contributions to personal PM exposures. These studies also reported high variability in personal $PM_{3.5}$ exposures and provided preliminary information about specific PM source contributions, such as environmental tobacco smoke (ETS) and emissions from wood-burning stoves.

Dockery and Spengler (1981) measured personal $PM_{3.5}$ and SO_4^{2-} exposures (over 12 or 24 hours) for 37 healthy adults from Steubenville, Ohio, and Watertown, Massachusetts. Corresponding concentrations from stationary ambient monitoring and from indoor monitors were also measured. Stronger correlations were observed for personal versus ambient SO_4^{2-} associations ($r = 0.81$) than for personal versus ambient $PM_{3.5}$ ($r = 0.69$). Because SO_4^{2-} is primarily associated with ambient sources, the difference in the personal–ambient correlations between SO_4^{2-} and $PM_{3.5}$ was attributed to the presence of indoor $PM_{3.5}$ sources.

Sexton and colleagues (1984) measured 24-hour $PM_{3.5}$ exposures for 48 nonsmokers during winter in Waterbury, Vermont. $PM_{3.5}$ concentrations were measured inside and outside the subjects' residences. No association was observed between $PM_{3.5}$ outdoor concentrations and corresponding personal exposures ($r = 0.00$). Mean personal exposures were found to exceed corresponding outdoor concentrations by an average of 19 $\mu g/m^3$, which was attributed to emissions from burning wood, which most subjects used as a major heating source. Further, indoor $PM_{3.5}$ concentration was a stronger predictor of personal exposure than the corresponding outdoor concentration. Similar results were found by Spengler and collaborators (1985) in a study of 97 healthy subjects living in two rural Tennessee communities, where measured ambient concentrations explained none of the variance observed in personal exposure. The variability of indoor concentrations, however, accounted for half of the variability in personal exposures, again suggesting that indoor sources (ETS in particular) are the primary determinants of variability in personal $PM_{3.5}$ exposures.

The first large-scale attempt to quantify source contributions to personal exposures to PM with aerodynamic diameters less than 10 μm (PM_{10}) was the Particle Total Exposure Assessment Methodology (PTEAM) study by the EPA (Clayton et al 1993; Ozkaynak et al 1993, 1996; Thomas et al 1993). As part of this study, personal PM_{10} exposures for 178 individuals from Riverside, California, were measured for 2 consecutive 12-hour periods (1 daytime period and 1 nighttime period). The exposure distribution of this selected group was intended to represent the 139,000 residents of Riverside (excluding children under 10 years of age). In addition, indoor and outdoor $PM_{2.5}$ and PM_{10} were sampled simultaneously. As with earlier cross-sectional studies, personal exposures exceeded both indoor and outdoor concentrations. This pattern was especially pronounced during the daytime, when personal PM_{10} exposures exceeded both indoor and outdoor concentrations by more than 50%. These higher PM_{10} exposures, which could not be explained by either outdoor or indoor concentrations, led investigators to consider the existence of a personal particulate cloud. The personal cloud was found to be mostly due to participant motion, resulting in PM resuspension, and/or the participant's proximity to particle sources. Both types of exposures may not be fully captured by the indoor samplers. Elemental analysis of the PM filters indicated that for 14 of the 15 measured elements, personal exposures were higher than corresponding indoor concentrations. Sulfur was the only element that did not appear to have any indoor or personal sources. It was hypothesized that simple physical movement, such as walking across a carpet or sitting on an upholstered piece of furniture, was a source of personal PM exposure through resuspension and that $PM_{2.5–10}$ was more easily resuspended than $PM_{2.5}$ (which includes all of the SO_4^{2-}). During relative inactivity, such as at night, the contribution of this personal cloud was far smaller, leading to personal exposure levels comparable to outdoor and indoor levels.

Data from the PTEAM activity diaries were used in multiple regression models to estimate activity-specific changes in personal exposure. Indoor PM_{10} concentrations were found to be the best predictor of personal PM_{10} exposures. In turn, outdoor PM_{10} concentrations, smoking, and cooking were shown to predict corresponding indoor PM_{10} levels. Cigarettes contributed on average 4% of indoor PM_{10} mass, cooking contributed about 5%, and other unidentified indoor sources or activities contributed 26%. At night, lower indoor concentrations were observed in homes with high air-exchange rates. Owing to the substantial contribution of indoor PM_{10} sources to personal exposures, correlations between outdoor concentrations and personal exposures were relatively weak ($r^2 = 0.37$). Correlations between personal sulfur exposures and outdoor sulfur concentrations, however, were consistently high ($r^2 = 0.77$).

Both the Dockery and Spengler (1981) and the PTEAM studies showed strong personal–ambient associations for SO_4^{2-} (or PM sulfur). A similar result was found for an exposure panel study of 24 children living in Uniontown, Pennsylvania (Suh et al 1992). Despite the cross-sectional design of the study, correlations between personal and ambient SO_4^{2-} concentrations were extremely strong ($r = 0.90$).

The collective results of these investigations have shown strong associations between particles originating

from ambient sources and corresponding personal exposures. Weak associations between daily outdoor PM concentrations and corresponding personal exposures were largely attributed to intersubject variability and the limited number of measurements conducted per subject (over 1 to 2 days). In contrast, as mentioned previously, many cross-sectional studies found strong associations between indoor and personal fine particle concentrations, underscoring the importance of indoor and personal source contributions to total personal PM exposures (Sexton et al 1984; Spengler et al 1985). Consequently, the existing PM exposure database was considered inadequate for investigating associations between personal exposures and outdoor concentrations, and longitudinal exposure studies were proposed. As a result, in the last 5 years several personal PM exposure studies have been conducted to examine the association between personal exposures and ambient PM concentrations over time. These studies built on the experience of previous longitudinal cohort studies (Lioy and Waldman 1990; Tamura et al 1996; Janssen et al 1998).

LONGITUDINAL STUDIES

Lioy and Waldman (1990) conducted 14 repeated 24-hour measurements of personal PM_{10} exposure and corresponding indoor and outdoor concentrations for 14 healthy, nonsmoking adults living in an industrial community. As was the case with the cross-sectional studies, personal PM_{10} exposures were higher than corresponding outdoor and indoor levels. Personal–outdoor associations were weak ($r^2 = 0.27$) when data were pooled and analyzed cross-sectionally. Subsequent reanalysis of the data by individual found that the personal–ambient associations were stronger (median $r^2 = 0.46$), with 6 of the 14 subjects having significant personal–ambient associations (Wallace 2000).

Janssen and colleagues (1998) examined longitudinal associations between personal PM_{10} exposures and outdoor concentrations in a study of 37 adults living in Amsterdam, the Netherlands. For each of these individuals, personal, indoor, and outdoor PM_{10} levels were measured for 7 consecutive days. To simulate a cross-sectional personal–ambient correlation coefficient, the investigators randomly selected 1 measurement per person for 7 days. This process was repeated 1000 times, yielding a median r value of 0.50. In comparison, the median, subject-specific longitudinal correlation coefficient equaled 0.71. In both analyses, days with exposure to ETS were excluded. Finally, the strength of the individual personal–ambient associations varied considerably between subjects, with correlation coefficients ranging from -0.41 to 0.94.

STUDIES INVOLVING SENSITIVE SUBPOPULATIONS

Epidemiologic studies of morbidity and mortality related to PM have indicated that older adults, children, and individuals with respiratory disease may be at greater risk than healthy adults are. For this reason, several personal exposure studies have focused on characterizing exposures for populations considered to be especially sensitive to PM health effects (Janssen et al 1997; Bahadori 1998; Bahadori et al 1999; Rojas-Bracho et al 2000b). A primary objective of these studies was to examine whether exposures for these cohorts differed from those for healthy cohorts. Some investigators have suggested that activity patterns for these cohorts may differ from that for the general population, leading to personal exposures that may, in turn, differ in concentration or composition (Bahadori et al 1999; Wallace 2000). Tamura and associates (1996) studied these factors in 7 retired, older adults with limited exposure to indoor PM sources. Personal PM_{10} exposure, home indoor, home outdoor, and central site ambient measurements were conducted every 48 hours for up to 11 consecutive days. Personal–ambient correlations were strong for all subjects, with r values ranging from 0.77 to 0.96. These results provided further evidence that when the impact of indoor and personal sources is minimized, the association between personal PM exposures and corresponding ambient PM becomes stronger.

Children have been cited in epidemiologic studies as being more susceptible to PM exposures (Pope and Dockery 1992; Schwartz et al 1994). Janssen and colleagues (1997) measured repeated personal PM_{10} exposures of 45 children (10 to 12 years old) in the Netherlands. Each child was sampled for 4 to 8 consecutive periods. Ambient PM_{10} concentrations were collected from a centrally located fixed monitoring site. Additional indoor measurements were also collected in the children's classroom. Personal exposures were, on average, 67 $\mu g/m^3$ higher than ambient concentrations as a result of exposure to ETS, high indoor PM_{10} concentrations, and PM resuspended by physical activity. The median longitudinal correlation coefficient for associations between personal exposures and ambient concentrations was 0.63.

Bahadori (1998) conducted a pilot study in which personal, indoor, and outdoor $PM_{2.5}$ and PM_{10} levels were measured for 10 individuals with COPD in Nashville, Tennessee. Indoor and outdoor $PM_{2.5}$ and PM_{10} concentrations were measured for 6 consecutive daytime (12-hour) periods. Personal sampling was also conducted every day, with personal $PM_{2.5}$ and PM_{10} exposures measured on alternate days. Results from this study showed that monitored individuals were exposed to few sources of indoor PM; mean daytime indoor concentrations were approximately two

thirds of the corresponding outdoor concentrations for both $PM_{2.5}$ and PM_{10}. Likewise, personal $PM_{2.5}$ and PM_{10} exposures were higher than corresponding indoor concentrations yet comparable to outdoor concentrations. Owing to the limited number of sampling days per participant, longitudinal correlations between personal exposure and outdoor concentrations were not determined.

Building upon the pilot design employed by Bahadori and colleagues (1998), Rojas-Bracho and coworkers (2000a) measured personal, indoor, and outdoor $PM_{2.5}$ and PM_{10} exposures of 18 individuals with COPD for multiple 12-hour periods (between 6 and 18 samples per individual). Of the 18 subjects, 9 had significant associations between personal $PM_{2.5}$ exposures and corresponding outdoor $PM_{2.5}$ concentrations (median $r = 0.60$). In contrast, only 3 of 18 subjects had significant associations between personal PM_{10} exposures and outdoor concentrations (median $r = 0.12$). These data provided further evidence of the individual-specific nature of the personal–ambient association and the stronger personal–ambient associations for $PM_{2.5}$ as compared to PM_{10}.

GASEOUS POLLUTANT PERSONAL EXPOSURE STUDIES

Numerous studies have characterized personal exposure to O_3, NO_2, and SO_2 (Liu et al 1993, 1997; Brauer and Brook 1995, 1997; Raaschou-Nielsen et al 1997; Alm et al 1998; Levy et al 1998). Ambient concentrations of gaseous pollutants and $PM_{2.5}$ are often correlated owing to common sources and the impact of meteorologic conditions (Chen et al 1999). As with personal PM exposure, efforts to characterize personal exposure to gaseous pollutants have been motivated primarily by epidemiologic findings showing associations between ambient gaseous pollutants and adverse health outcomes. Few published studies have characterized simultaneous personal exposures to both PM and gaseous copollutants (Sarnat et al 2000, 2001; Mosqueron et al 2002). Further, the potential for confounding by gaseous and particulate copollutants in particular has not been fully examined.

A copollutant may be a confounder of a $PM_{2.5}$-associated health effect if it correlates with exposure to $PM_{2.5}$ and if exposure to this copollutant can itself cause a health effect. Because exposure studies have focused on single pollutants, it has not been possible to determine the level of correlation between exposure to $PM_{2.5}$ and its copollutants. However, confounding by gaseous pollutants has been addressed by epidemiologic studies, which have provided conflicting results (Burnett et al 1999; Fairley 1999). For example, Fairley (1999) observed significant associations between health effects and numerous ambient pollutant concentrations (including $PM_{2.5}$, CO, O_3, and NO_2) when pollutants were examined individually. When the gaseous pollutants were examined along with $PM_{2.5}$, however, the significant associations with the gases disappeared, whereas the association for $PM_{2.5}$ became stronger, suggesting that fluctuations in ambient $PM_{2.5}$ concentrations were driving the observed associations with health effects. Burnett and colleagues (1999) examined the association between ambient $PM_{2.5}$, CO, O_3, NO_2, and SO_2 concentrations and a number of morbidity outcomes over 14 years in Toronto, Canada. In contrast to previous studies, They found attenuated associations between respiratory infections and $PM_{2.5}$ when gaseous copollutants were included in the model.

STUDY OBJECTIVES

The primary objectives of this study were to characterize personal particulate and gaseous exposures for cohorts of older adults (seniors), children, and individuals with COPD and to assess the appropriateness of exposure assessment strategies for use in PM epidemiologic studies.

Specifically, this report addresses 3 hypotheses:

- Ambient $PM_{2.5}$ concentrations are appropriate surrogates of corresponding personal $PM_{2.5}$ exposures.
- Associations between personal $PM_{2.5}$ exposures and ambient $PM_{2.5}$ concentrations vary by city, season, and cohort.
- Ambient concentrations of O_3, NO_2, and SO_2 correlate more strongly with measures of personal exposure to $PM_{2.5}$ than with measures of personal exposure to O_3, NO_2, and SO_2.

METHODS

Personal multipollutant exposures and corresponding ambient concentrations were measured for 56 and 43 subjects living in Baltimore, Maryland, and Boston, Massachusetts, respectively (Table 1). Several subjects from both cities participated in both summer and winter sampling seasons. Both studies were approved by the Harvard School of Public Health Institutional Review Board. Additionally, subjects completed and returned informed consent forms prior to their participation in the study.

The Baltimore cohorts consisted of 20 healthy seniors, 21 children, and 15 individuals with physician-diagnosed COPD. The Boston cohorts consisted of 20 healthy seniors and 23 children. All subjects were nonsmokers and lived in residences (either single-family houses or apartments)

Table 1. Study Design

City / Season	Seniors *N*	Seniors Sampling Days per Subject	Children *N*	Children Sampling Days per Subject	COPD *N*[a]	COPD Sampling Days per Subject[a]
Baltimore						
Summer 1998	15[b]	12	10	8	—	—
Winter 1999	15[b]	12	15	12	15	12
Boston						
Summer 1999	15	12	15	12	—	—
Winter 2000	15	12	15	12	—	—
Total sampling days		720		620		180

Pollutant	Total Samples Targeted for Collection: Seniors	Children	COPD
$PM_{2.5}$	720	620	180
O_3	720	620	180
NO_2	720	620	180
SO_2	540[c]	540[c]	180
SO_4^{2-}	720	360[d]	180
EC	360[e]	620	180

[a] — is not applicable.

[b] Data from senior cohort in Baltimore are included in the analyses but were funded by the American Petroleum Institute (API).

[c] SO_2 was not collected during summer in Baltimore.

[d] SO_4^{2-} was not analyzed for Baltimore children.

[e] EC was not collected for the API-funded senior cohort.

with nonsmokers. Although the subjects were from a range of socioeconomic backgrounds and geographic locations within each city, subject selection was not random and was not intended to be representative of sensitive populations in general. All children who participated in the study were between the ages of 9 and 13 years at the start of the sampling period. Members of the senior cohort were recruited on the basis of a self-reported age of 65 or older. The COPD cohort consisted of individuals with physician-diagnosed moderate to severe COPD. Only 1 participant, a senior in Baltimore during the summer, dropped out of the study after the first day of sampling and was replaced.

Seniors were recruited mainly from senior centers and newspaper advertisements. Children were recruited primarily from local community centers (eg, Young Men's Christian Association [YMCA] and Police Athletic League centers) in Baltimore and Boston and through a science education program in Baltimore. Word of mouth and referral by other participants were also important means of recruiting many of the participants. COPD subjects were recruited mainly through exercise groups and newspaper advertisements.

Personal exposures for all subjects were measured for 12 consecutive days (henceforth referred to as a *sampling session*) in winter and then again in summer except for the summer sampling session for children in Baltimore, which was only for 8 consecutive days (Appendix A). Sampling sessions consisted of 4 to 16 subjects per group. Personal exposures of subjects from all cohorts were sampled concurrently, except for the summer sampling in Baltimore, in which personal exposures were measured successively in seniors and children. There were 3 sessions during each sampling period, with the exception of 4 sessions during the summer in Baltimore. A total of 1520 person-days of personal exposure data were collected for $PM_{2.5}$, O_3, NO_2, and SO_2 (Table 1). All $PM_{2.5}$ filters were analyzed for SO_4^{2-} concentration except for children's filters in Baltimore. Personal SO_4^{2-} exposures were used as surrogates of personal exposure to $PM_{2.5}$ of ambient origin.

Personal exposure samples of $PM_{2.5}$, O_3, NO_2, and SO_2 were collected using a specially designed multipollutant sampler (Demokritou et al 2001). $PM_{2.5}$ was collected using personal environmental monitors (PEMs) and 37-mm Teflon filters (Teflo, Gelman Sciences, Ann Arbor MI). The O_3, NO_2, and SO_2 concentrations were measured with passive samplers. Each passive sampler contained a single filter coated with either nitrite to collect O_3 (Koutrakis et al 1993) or triethanolamine to collect NO_2 and SO_2 (Ogawa & Co 1998). Because no accurate personal CO monitors exist, no personal CO measurements were conducted.

Subjects were permitted to remove the sampler during prolonged periods of inactivity (eg, sleeping and watching television) and during activities when the sampler could be damaged (eg, showering and intense physical activity). When the sampler was removed from the subject's body, subjects were instructed to keep the sampling inlets as close as possible to their breathing zone.

Field staff visited the residences of the seniors and the COPD participants each morning of sampling between approximately 7:00 and 11:00 AM to change sampling equipment, replace pump batteries, record and adjust flows, and review the completed time–activity diaries from the previous 24 hours. Daily visits to the children's

residences in Baltimore during winter took place between 3:00 and 7:00 PM.

ACTIVE SAMPLERS

The main sampler components of the active samplers included a pump and battery that were carried in the pouch of a backpack or shoulder pack and an elutriator, which was used as a support structure for the various active and passive samplers (Appendix B). Active flows to the pump were split into 2 or 3 subflows using flow restrictors or valves. For all Baltimore cohorts and the Boston summer cohorts, the $PM_{2.5}$ target flow rate was 3.2 L/min and the EC target flow rate was 2.0 L/min. For the winter Boston cohorts, a 3-way flow split was used to collect $PM_{2.5}$ and EC at 2.0 L/min and SO_4^{2-} at 0.8 L/min.

PEMs were used to collect $PM_{2.5}$ and EC. Their inlets were placed nozzle-down within the Teflon-coated aluminum elutriator. The PEMs contained small inertial impactors designed specifically for personal monitoring to collect $PM_{2.5}$ on 37-mm Teflon filters placed downstream of an oil-coated sintered stainless steel impactor plate (Clayton et al 1993; Thomas et al 1993). EC was collected by the PEMs using a single quartz filter. Personnel at the Desert Research Institute (Reno NV) analyzed the EC collected using a thermal/optical reflectance method (Chow et al 1993). SO_4^{2-} concentrations were determined by extracting the $PM_{2.5}$ filters and analyzing the aqueous extract by ion chromatography. Originally, $PM_{2.5}$ samples were to be analyzed for elemental composition using x-ray fluorescence, but insufficient mass was collected using the split-flow sampler design. As a result, an additional subset of filters was analyzed for SO_4^{2-} instead.

Sampler flows were measured both before and after sampling, in duplicate, using a soap bubble flow meter (AP Buck, Orlando FL). Particle mass was estimated using gravimetric analysis. The filters used to collect $PM_{2.5}$ were weighed in a room with controlled temperature (18–24°C) and relative humidity (40% ± 5%). Filters were left to equilibrate 24 hours before the initial weighing and 48 hours prior to postsampling weighing. Each filter was weighed, in duplicate, both before and after sampling using a microbalance (model MT5, Mettler-Toledo, Columbus OH). The average of the two weights was used as the filter weight. When the two weights differed by more than 5 µg, the filter was weighed a third time, with the final value being the average of the two closest weights.

$PM_{2.5}$ blank samples were treated exactly as the actual samples prior to deployment in the field, including leak testing in the lab. In the field, the PEMs were removed from the plastic bag used to transport the sample, put into the elutriator, removed, placed back in the plastic bag, and placed in the front pocket of the backpack or shoulder bag until sample collection the next day.

In situations where mean field-blank values differed significantly from zero, masses were corrected by subtracting the median field-blank weights from the sample weights. Teflon filter weights were also corrected for barometric pressure during each weighing session (ie, before and after sampling; Koistinen et al 1999). During all sampling periods, with the exception of summer sampling sessions in Baltimore, particle filters were stored in refrigerated environments after sampling to reduce potential volatilization from the filters.

PASSIVE SAMPLERS

In addition to active sampling methods, the multipollutant samplers also used passive samplers (Ogawa & Company, Pompano Beach FL) to measure exposures to O_3, SO_2, and NO_2 (Ogawa et al 1998). Passive samplers used to measure SO_2 and NO_2 contained cellulose filters coated with triethanolamine. Aqueous extracts of filters were then analyzed for SO_4^{2-} and nitrite (NO_2^-) by ion chromatography. O_3 samplers contained a glass-fiber filter coated with potassium nitrate and sodium carbonate. O_3 oxidizes the NO_2^- to form nitrate (NO_3^-), which was extracted and analyzed by ion chromatography (Koutrakis et al 1994).

The O_3 sampler was tested for its accuracy and precision (Koutrakis et al 1993), because previous research indicated that these samplers provide variable results when airflow across the sampler face (face velocity) is not as consistent as would be expected with personal sampling (Liu et al 1993). Use of aluminum elutriators facilitated a constant face velocity at the boundaries of the passive samplers and maintained a constant passive sampler collection rate. The effective sampling rate of these passive samplers under various temperatures and relative humidities were reviewed. The effective sampling rate of these passive samplers did not vary dramatically: flow rates were 2.0 to 3.2 L/min (Chang et al 1999).

All passive samplers were assembled in the Harvard School of Public Health research laboratory using a positive flow hood and kept refrigerated until approximately 30 minutes before being deployed in the field. All passive samplers were shipped, stored, and transported during the study in plastic resealable bags that were placed in opaque plastic bottles. After sample collection, the plastic bottles containing the bagged passive samplers were placed in insulated, cooled bags for transport. Once back in the laboratory, bagged passive samplers were stored in a refrigerator until they were disassembled.

SAMPLING OF AMBIENT POLLUTANT CONCENTRATIONS

Ambient pollutant parameters, site locations, and sampling seasons are presented in Appendix C. Maps showing the locations of participants' homes and ambient monitors in each city are presented in Appendix D. Efforts were made to obtain all ambient monitoring data from both cities during the study sampling periods.

Baltimore Ambient Monitors

Field staff operated 2 Harvard impactors at centrally located monitoring sites in Baltimore to measure 24-hour (8 AM to 8 AM) integrated ambient $PM_{2.5}$ data. Particles were collected by the Harvard impactors at a flow rate of 10 L/min. SO_4^{2-} was extracted from the $PM_{2.5}$ filters and amounts of SO_4^{2-} were determined by ion chromatography. Personal-exposure SO_4^{2-} samples were also processed by extraction and ion chromatography. Integrated ambient $PM_{2.5}$ sampling was not conducted for the Baltimore children's samples that were collected in the afternoon; therefore no ambient SO_4^{2-} and EC samples were available for comparison with corresponding personal exposures. Instead, two continuous tapered element oscillating microbalance (TEOM) $PM_{2.5}$ monitors (model 1400A, Rupprecht & Patashnick, East Greenbush NY) operated by the Maryland Department of the Environment were used to provide ambient $PM_{2.5}$ concentrations. All of the subjects lived within a 25-mile radius of the integrated monitoring site.

Continuous O_3, NO_2, and SO_2 data were obtained from stationary ambient monitoring sites located throughout Baltimore and operated by the Maryland Department of the Environment. Ambient concentrations of O_3, NO_2, and SO_2 were measured using a UV photometric analyzer, a chemiluminescence monitor, and a pulsed fluorescent monitor, respectively. Average 24-hour gas data were calculated to coincide with the start and end of the integrated $PM_{2.5}$ samples (8 AM to 8 AM or 4 PM to 4 PM). Where data were available from multiple sites in the city, the hourly average was calculated across sites, and daily averages were calculated from those hourly averages.

Boston Ambient Monitors

For sampling in Boston, integrated monitors were operated at a central monitoring site located at Harvard School of Public Health. Harvard impactors were used to collect $PM_{2.5}$; ChemComb samplers (model 3500, Rupprecht & Patashnick) were used to collect EC; and Harvard-EPA annular denuders (HEADs) were used to collect integrated SO_4^{2-} samples. All ambient integrated samples were run from 9 AM to 9 AM to coincide with the average start times for personal samples. In addition, a TEOM was used to measure continuous $PM_{2.5}$ at the Harvard School of Public Health site. TEOM data were used on 4 days when integrated $PM_{2.5}$ was found to be invalid. All of the subjects lived within an 8-mile radius of the integrated monitoring site. City-specific seasonal corrections were applied to all TEOM data using integrated methods.

In Boston, ambient gas concentrations were obtained from all state monitors within Suffolk County (with the exception of a single site that was excluded owing to its remote location). All monitors were operated throughout the year with the exception of 1 in Chelsea that measured NO_2, SO_2, and O_3 only during summer. In addition to this site, year-round measurements were collected at 3 sites for NO_2, 4 sites for SO_2, and 1 site for O_3. Ambient monitors in Boston employed the same sampling methods as those discussed for Baltimore.

Correlation Across Ambient Monitoring Sites

Averaging pollutant concentrations across sites may introduce noise into models examining associations involving these values, especially for pollutants that are spatially heterogeneous. $PM_{2.5}$ and SO_4^{2-} have been shown to be homogeneously distributed over urban areas in the eastern United States (Burton et al 1996; Ito et al 2001), but local sources for some gaseous pollutants may result in greater differences among monitoring sites. To examine the variability among gaseous monitoring sites in our study, daily coefficients of variation (CVs) were calculated.

In Boston, the average CV among the sites measuring NO_2 was 25% and 30% during summer and winter, respectively (mean ambient NO_2: summer, 24.9 ppb; winter, 28.0 ppb). Variability across sites measuring SO_2 was greater, with average CVs of 59% and 44% during summer and winter, respectively. Mean concentrations of ambient SO_2 were much lower, however (summer, 3.7 ppb; winter, 8.1 ppb), which likely accounts for some of the higher observed CV values.

In Baltimore, the summer CV for NO_2 measurements was 22% (21.2 ppb average). During winter in Baltimore, NO_2 measurements were obtained from 2 ambient sites located approximately 5 miles apart. Good agreement was found between these sites (Appendix E).

ADDITIONAL DATA

In addition to wearing the personal sampler, all but 3 subjects completed a closed-form, time-based activity diary on each monitoring day. The diary was divided into 30-minute recording intervals for all cohorts, except for seniors in Baltimore during the winter sampling session, who recorded activities and locations in 15-minute increments. The

closed-form activity diary included several columns to allow subjects to indicate whether a specific pollutant-generating activity was being performed (eg, cooking, cleaning, or exposure to ETS). Three seniors in Baltimore used an open-form, activity-based diary: each page corresponded to a specific activity with space available to indicate location and any special conditions likely to affect exposure. This diary was found to be too cumbersome for most participants and was discontinued in subsequent sampling sessions. The activity diary used for the children was simpler than that used for the other 2 cohorts: it requested limited information on locations and particle-generating activities only.

Field staff recorded characteristics of the subjects' residences that could influence personal exposures. This information included type of cooking fuel and type of garage (attached vs freestanding); presence or absence of carpeting, air cleaning devices, and pets; and number of individuals residing in the household.

QUALITY ASSURANCE

Standard quality assurance and quality control procedures were followed for this study (Harvard School of Public Health 1999). Field-blank collection rate was approximately 10% of the total sample size. Collected data were assessed for bias, precision, and completeness. Precision and bias of the multipollutant sampler methods were calculated by collocating replicate, fully configured personal-exposure sampling backpacks at stationary ambient monitoring sites equipped with reference sampling methods. The samplers were operated for 24 hours ± 10%. For a given pollutant, precision was estimated as the root mean squared difference between the collocated personal samplers, divided by the square root of 2. This method of quantifying precision yielded the best, albeit imperfect, estimates of sampling measurement error. Precision calculated for extremely low pollutant concentrations is likely to be lower than precision calculated for collocation sampling, which was generally higher than the integrated personal exposure levels. Bias for a given method was determined using the mean relative difference between the multipollutant sampler concentration and the corresponding mean reference method concentration.

Method limits of detection (LODs) were estimated as 3 times the mean of the field blanks for the respective sampling method as well as 3 times the absolute precision of the duplicate samples. Completeness was calculated as the number of samples collected divided by the target number of samples. Table 2 lists the precision, bias, completeness, and LOD results for samplers used in both cities. Table 3 lists the percentage of samples that were below the relevant method LODs.

DATA ANALYSIS

The main components of the data analysis presented in this report are:

- characterization of particulate and gaseous pollutant exposures for the cohorts of children, seniors, and individuals with COPD who participated in the study;
- examination of the contribution of ambient $PM_{2.5}$ concentrations to corresponding personal exposures, including an analysis of differences by city, season, and cohort and of the influence of specific factors on these results; and
- assessment of the relations between $PM_{2.5}$ and its copollutants and evaluation of the implications for epidemiologic studies.

Characterization of Personal Exposures and Ambient Concentrations

Personal pollutant exposures and ambient pollutant concentrations were characterized using descriptive statistics, graphical analysis, and analysis of variance. Means, medians, and standard deviations are reported for all pollutant concentrations and exposures. Concentrations for $PM_{2.5}$ and SO_4^{2-} are reported in micrograms per cubic meter. Concentrations for O_3, NO_2, and SO_2 are reported in parts per billion. Because there is no consensus among air-quality statisticians regarding the most appropriate means of statistically treating data values below the LOD, negative pollutant concentration values and values below the LOD were included in data analyses as measured in order to reduce possible bias in estimating relations among measurements (McBean and Rovers 1998). Quantitative analyses were conducted for pollutants that were predominantly distributed below their respective LODs. Therefore, analyses for these pollutants may have been highly influenced by noise from the measurement methods. Exposures were tested for normality using Shapiro-Wilks tests and stem-and-leaf plots. Several pollutant concentrations and personal exposure distributions were found to be moderately nonnormal. For this reason, nonparametric measures of association, including Spearman correlation coefficients (r_s), were used.

Contribution of Ambient $PM_{2.5}$ and Exposure Factors

Mixed-model regression analysis was used to examine the contribution of ambient $PM_{2.5}$ to corresponding personal $PM_{2.5}$. Mixed models were used in order to model covariance among the pollutants and to pool information across individuals while accounting for repeated measures on the same individual. Mixed models also allow each subject to serve effectively as his/her own control by inherently

Table 2. Quality Assurance Data[a]

		Precision		Bias[c]		
	LOD[b]	Absolute	Relative (%)	MRD[d] (SD)	Ref Mean[e]	Completeness
Baltimore Winter						
$PM_{2.5}$ (μg/m^3)	3.5 (3.6)	1.2	5.6	1.3 (2.3)	20.5	494/540 (91.5%)
SO_4^{2-}(μg/m^3)	2.6[f]	0.7[f]	—	NA	NA	326/360 (90.6%)
EC (μg/m^3)	0.9 (1.2)	0.4	15.1	—	—	327/360 (91.8%)
O_3 (ppb)	5.5 (11.2)	3.7	17.9	−1.0 (5.4)	21.2	456/540 (84.4%)
NO_2 (ppb)	11.7 (26.7)	8.9	39.1	6.4 (20.5)	23.5	492/540 (91.1%)
SO_2 (ppb)	6.5 (6.9)	2.3	30.5	−3.5 (1.9)	8.1	494/540 (91.5%)
Baltimore Summer						
$PM_{2.5}$ (μg/m^3)	2.3 (8.7)	2.9	8.3	3.3 (2.5)	32.1	238/258 (92.2%)
SO_4^{2-}(μg/m^3)	2.6[f]	0.7[f]	—	NA	NA	168/180 (93.3%)
EC (μg/m^3)	—	—	—	—	—	—
O_3 (ppb)	6.6 (10.3)	3.4	8.9	−0.2 (5.3)	38.4	211/258 (81.8%)
NO_2 (ppb)	5.7 (6.8)	2.3	14.0	0.0 (2.8)	16.0	232/258 (89.9%)
SO_2 (ppb)	—	—		—	—	—
Boston Winter						
$PM_{2.5}$ (μg/m^3)	6.8 (4.9)	1.6	17.6	0.0 (2.4)	9.4	278/360 (72.2%)
SO_4^{2-}(μg/m^3)	0.2 (0.7)	0.2[g]	8.0	−0.2 (0.3)	2.5	312/360 (86.7%)
EC (μg/m^3)	0.9 (1.5)	0.5	40.7	0.0 (0.5)	1.1	328/360 (91.1%)
O_3 (ppb)	4.9	—	—	—	—	311/360 (86.4%)
NO_2 (ppb)	4.2	—	—	—	—	323/360 (89.7%)
SO_2 (ppb)[h]	3.2	—	—	—	—	323/360 (89.7%)
Boston Summer						
$PM_{2.5}$ (μg/m^3)	3.1 (5.9)	2.0	11.4	1.4 (2.4)	15.2	351/360 (97.5%)
SO_4^{2-}(μg/m^3)	0.1 (0.6)	0.2	3.6	0.5 (3.2)	5.8	351/360 (97.5%)
EC (μg/m^3)	—	—	—	—	—	—
O_3 (ppb)	7.0 (9.4)	3.1	8.6	−0.4 (4.5)	36.3	344/360 (95.6%)
NO_2 (ppb)	6.4 (1.6)[i]	0.5	4.0	0.5 (5.7)	14.3	353/360 (98.1%)
SO_2 (ppb)[h]	2.3 (2.3)	0.8	69.5	—	—	347/360 (96.4%)

[a] — is not measured.

[b] LOD value calculated as 3 times the standard deviation of blanks. LOD value in parentheses calculated as 3 times the method precision.

[c] NA is not available.

[d] Mean relative difference between the multipollutant sampler concentration and the corresponding reference method concentration.

[e] Mean concentration of the reference method.

[f] Estimated LOD and precision from the Harvard-EPA annular denuder system.

[g] Excludes 1 extreme value. With it included, absolute precision = 0.7 μg/m^3, relative precision = 27.0%, and LOD = 2.1 μg/m^3.

[h] SO_2 values were not adjusted for collection efficiency.

[i] Calculated using median values, owing to 1 extreme value.

controlling for subject-specific characteristics that do not change over the course of the study.

The general form of the model used for this analysis can be expressed as follows:

$$Y_{ijkl} = \alpha_k + \beta_k X_{ijkl} + b_{il} + \epsilon_{ijkl} \quad (1)$$

where Y_{ijkl} represents the personal $PM_{2.5}$ exposure for subject i on day j in sampling session l (1 of the 13 sampling sessions in the study) for the k levels of the city–season data group (k is a 4-level categorical variable: 1 = Baltimore winter, 2 = Baltimore summer, 3 = Boston winter, 4 = Boston summer); α_k represents the regression intercept for

Table 3. Percentage of Samples Below Relevant Method LOD[a]

	Baltimore		Boston	
	Summer (%)	Winter (%)	Summer (%)	Winter (%)
$PM_{2.5}$	5.4	4.3	0.3	5.1
SO_4^{2-}	—	—	3.5	2.6
EC	—	17.1	—	59.1
O_3	93.4	99.8	76.2	96.8
NO_2	38.8	80.3	10.2	2.2
SO_2	—	99.2	96.5	95.4

[a] Where LOD values were calculated as both 3 times the standard deviation of blanks and 3 times the method precision, the higher of the 2 values was used. — is not measured.

city–season data group k; βk represents the slope of the regression of personal exposure on ambient $PM_{2.5}$ concentration for city–season data group k; and X_{ijkl} represents the ambient concentration for subject i on day j of the study during sampling session l for city–season data group k. The error terms represent the between-subject random effect (b_{il}) and the within-subject effect (ϵ_{ijkl}) for each subject in sampling session l in the study. Both of these error terms are assumed to be normally distributed, with the b_{il} independently distributed with mean 0 and variance $\sigma^2_{b,l}$ and the ϵ_{ijkl} following a first-order autoregressive process with autoregressive correlation p_l and residual error $\sigma^2_{e,l}$ (Diggle et al 2002).

For this analysis, model slopes were interpreted as the degree of influence of an ambient concentration on a corresponding personal exposure, with values from 0 to 1.0. Slopes (henceforth referred to as *infiltration factor* or β_k) were calculated for each city, season, and cohort. The standard error of β_k indicates the amount of noise in the relation between dependent and independent variability in lieu of a coefficient of determination (r^2), which is not available in the output for mixed models. For this analysis, model intercepts, α_k, can be interpreted as an indication of the nonambient source contributions to personal exposures.

Similar models were used to examine differences in the impact of ambient $PM_{2.5}$ on personal exposures by cohort and differences in exposure on weekends versus weekdays. The weekend–weekday variable was not significant. As a result, a factor for day-of-week effects was not included in the final model. Likewise, an identical modeling approach was used to determine the infiltration factors for SO_4^{2-}. Because SO_4^{2-} is a component of $PM_{2.5}$ and is mostly ambient in origin, these models were aimed at examining various infiltration factors, β_k, unaffected by nonambient sources of $PM_{2.5}$.

Mixed models were also used to estimate the effects of various activity or housing factors on personal $PM_{2.5}$ exposures while controlling for the corresponding ambient concentration. A generalized form of the model used for these analyses can be written as:

$$Y_{ijl} = \alpha + \beta_0 X_{ijl} + \beta_1(\text{factor}_{ijl}) + b_{il} + \epsilon_{ijl} \quad (2)$$

where Y_{ijl} represents an observed personal particle exposure for subject i on day j in sampling session l; α represents the regression intercept; β_0 represents the fixed effect of X on Y; X_{ijl} represents the observed ambient particle concentration for subject i on day j in sampling session l; β_1 represents the fixed effect of a dichotomous variable (such as cooking or cleaning) on Y; factor_{ijl} is a categorical variable (with 0 indicating that this factor did not occur during the exposure period and 1 indicating at least some occurrence of this factor) for subject i on day j of the study during sampling session l; b_{il} represents the between-subject random effect ~ $N(0,\sigma^2_{bl})$; and ϵ_{ijl} represents the within-subject random error ~ $N(0,\sigma^2_{el})$. This model was used to test the effects of 5 dichotomous variables—cooking, cleaning, living near a busy road, living in a house (versus an apartment), and having a gas stove in the home. In addition, the impact of gas stoves on personal NO_2 exposures was examined using this form of the model.

Finally, a variance component analysis was conducted to determine the differential effects on the slope estimates for each subject by city and season. Analysis of variance was used to determine whether subject-specific regression slopes differed by city, season, and cohort. The mean sums of squares were subsequently used to partition the total model variance into the fraction attributable to city, season, and model components of error.

Relations Between $PM_{2.5}$ and Its Copollutants and Implications for Epidemiologic Studies

The following four models were used to assess associations between $PM_{2.5}$ and its copollutants:

- **Model 1.** Associations between ambient $PM_{2.5}$ concentrations and ambient gaseous concentrations (ambient–ambient associations).
- **Model 2.** Associations between ambient pollutant ($PM_{2.5}$, SO_4^{2-}, and gas) concentrations and their respective personal exposures (personal–ambient associations).
- **Model 3.** Associations between ambient gaseous concentrations and personal $PM_{2.5}$ exposures, including SO_4^{2-} as a surrogate of personal exposure to $PM_{2.5}$ of ambient origin (cross-pollutant associations).

- **Model 4.** Associations between personal $PM_{2.5}$ exposures, including SO_4^{2-} as a surrogate of personal exposure to $PM_{2.5}$ of ambient origin, and personal gaseous exposures (personal–personal associations).

For model 1, analyses were examined using time-series regression analysis assuming a first-order autoregressive structure for the error. For models involving personal exposures (ie, models 2 to 4), mixed-model regression analysis was conducted using a generalized form similar to equation 1:

$$Y_{ijl} = \alpha + \beta X_{ijl} + b_{il} + \epsilon_{ijl} \quad (3)$$

where Y_{ijl} represents an observed personal particle or gas exposure for subject i on day j in sampling session l; α represents the regression intercept; X_{ijl} represents either an observed personal gas exposure or an ambient particle or gas concentration for subject i on day j of the study during sampling session l; β represents the fixed effect of X on Y; b_{il} represents the between-subject random effect $\sim N(0,\sigma^2_{bl})$ and ϵi_{jl} represents the within-subject random error $\sim N(0,\sigma^2_{el})$. The l index in the variance components denotes the fact that the models allow for different between-subject and within-subject error in the different sampling sessions (Diggle et al 2002).

Because gas stoves constitute a potential indoor source of NO_2 and $PM_{2.5}$, mixed models were also used to assess whether associations differed between subjects living in residences with gas and electric stoves. A generalized form of this model can be expressed as:

$$Y_{ijl} = \alpha + \beta_0 X_{ijl} + \beta_1(\text{stove\_type}_i) + \beta_2(X_{ijl} \times \text{stove\_type}_i) + b_{il} + \epsilon_{ijl} \quad (4)$$

where stove_type_i is a categorical variable for either a gas or electric stove in the residence and $\beta_2(X_{ijl} \times \text{stove\_type}_i)$ is the interaction term characterizing the effect of cooking fuel. Analyses examining stove type do not address actual gas stove use during sampling. Models investigating the influence of day of week or weekend versus weekday exposure patterns did not yield significant effects and thus were not used for the final model specification.

All of the mixed models for models 2 through 4 were fitted using a compound symmetry (ie, random intercepts) covariance matrix with a nested panel variable. This covariance structure yielded the lowest Akaike information criteria diagnostic values compared with other covariance matrices examined for this analysis.[1] Compound symmetric models assume that correlations between repeated measures arise because each subject has an underlying level of response over time. This so-called subject effect can then be modeled as a random effect within the model. In addition to having the lowest Akaike information criteria values, the random intercepts models were found to be the most appropriate models given that subjects were measured during different 12-day sampling sessions. Therefore, the variability of mean longitudinal exposures may not reflect true variability but rather the variability for all subjects measured on day j. The random intercepts model provides an estimate of mean personal exposure on day j while effectively controlling for not measuring each person on every day. Similar analyses using models that did not nest each subject within their respective panel were also conducted to evaluate model robustness. Results for models 2 through 4 are presented in Appendices F through H, respectively. The strength of association was assessed by the size and corresponding t statistics of the estimated slope of the mixed models.

In addition to the time-series and mixed model regression analyses, subject-specific r_s values were reported as a secondary indicator of the strength of correlation in the observed relations by individual. Subjects having less than 7 of 12 valid person-days of observations were excluded from the analysis. The subject-specific correlation analysis provided a more general means of examining the pollutant relations because it does not assume the same association between response and predictor for all individuals. Rather, it looks at how these associations vary across individuals.

Relations described by alternative distributions might provide a more accurate model to better understand interactions and associations among the pollutants. Therefore, an additional pilot analysis was conducted using a subset of the Baltimore data to examine exclusively pollutant associations using joint distributions (Appendix I).

All of the above analyses were computed using SAS software (version 8.2, SAS Institute, Cary NC). Statistical significance is reported at the 0.05 level. Time–activity data indicated that several subjects experienced exposure to high concentrations of ETS or prolonged exposure to ETS during sampling. As a result, a total of 57 person-days (approximately 5% of total) were not included in the analyses (from 1 senior during summer and winter and 1 child during winter). These samples were not typical for a nonsmoker or someone living in a residence with nonsmokers (Appendix J).

1. Epidemiologic models typically do not include autoregressive information in models of associations involving dependent health outcomes. Therefore, using autoregressive covariance components for analyses investigating epidemiologic modeling practices would not be appropriate. Because the measures were in fact likely to be serially correlated, however, the sensitivity of the model inferences was checked using the sandwich (empirical) standard errors for the β coefficients. This approach yields an estimate of the variance for β that is robust to misspecification of the covariance model for the repeated measurements taken on the same subject. Interpretation of the presented results did not change when using either the model-based or empirical standard errors.

RESULTS

HOUSEHOLD CHARACTERISTICS AND TIME–ACTIVITY PATTERNS

The participants' time–activity diaries focused on activities or microenvironments that in previous studies have been linked with increased pollutant exposures, such as gas stoves in a residence or time spent outdoors. Much of the time–activity information is anecdotally useful. A challenge of the current study was assuring participant compliance in completing the time–activity diaries. Balancing the desire for as much information as possible against the need for convenient and easy-to-use diaries was not entirely successful. Keeping a record of location and activity every half-hour for up to 12 consecutive days required extreme diligence. Field technicians were unable to validate the diaries and relied on daily reviews with the participants for verification. Despite these limitations, key lessons were learned regarding collection of this type of information with implications for future exposure studies.

During both seasons in Boston, a substantially higher percentage of children (compared to seniors) lived in residences with gas stoves, a potential source of both NO_2 and $PM_{2.5}$ (Palmes et al 1977; Leaderer et al 1999). In Boston during the winter, 71% of the children and 13% of the seniors lived in residences with gas stoves (Appendix K). During the summer, 87% of the children and 13% of the seniors lived in homes with gas stoves. In Baltimore during the summer, 50% of children and 27% of seniors lived in residences with gas stoves. During the winter in Baltimore, 53% of seniors, 57% of children, and 60% of COPD participants lived in homes with gas stoves. When the cohorts were aggregated by city and season, the distribution of gas and electric stoves in the Boston homes sampled was essentially even, whereas in Baltimore approximate 60% of stoves were gas in the winter and 40% in summer.

The use of air conditioners during the summer was also identified as modifier of PM exposures in a previous air pollution study (Suh et al 1992). During the summer in Boston, 57% of the children and 80% of seniors lived in homes equipped with at least 1 air conditioning unit. In Baltimore, all of the children and all but 1 senior had at least 1 air conditioner in their homes. These data should be interpreted with caution, however, because no information on actual daily use of air conditioning or, alternatively, open-window status was obtained for the children or COPD cohorts in Baltimore or for any of the Boston cohorts. The time–activity diary used for the seniors in Baltimore included a question about open-window status during the day. Table 4 lists selected housing characteristics and activities for each city, season, and cohort.

In previous studies, cooking and cleaning have been linked to increases in PM exposures (Ozkaynak et al 1996). In the current study, cooking and cleaning events were rare, particularly among children who did not record participating in any cooking events. Cleaning events were also uncommon, with a minimum of 10 observations for children in Baltimore during summer and a maximum of 57 observations for seniors in Boston during winter. Participants may

Table 4. Selected Housing Characteristics and Activities

	Baltimore[a]					Boston[a]			
	Winter			Summer		Winter		Summer	
	Seniors	Children	COPD	Seniors	Children	Seniors	Children	Seniors	Children
Housing characteristic (number of houses)									
Location near busy road	5	8	6	5	2	13	10	11	10
Attached garage	1	2	1	0	0	0	1	0	0
Storm windows	10	9	8	—	—	7	8	—	—
At least 1 air conditioner in home	—	—	—	15	9	—	—	11	9
House	7	13	11	5	9	2	6	5	4
Apartment	8	0	4	10	0	12	8	9	11
Activity (number of days)									
Cooking	—	2	48	—	1	29	0	34	2
Cleaning	—	13	39	—	10	54	17	57	25

[a] — is not known.

have altered their activities during the study (a phenomenon known as the *Hawthorne effect*) and cooked or cleaned less while wearing the sampling apparatus. Likewise, younger adults may engage in these activities more frequently than children, retired seniors, and COPD patients.

Subjects from all cohorts in both cities spent the majority of their time indoors, from 65% to 82% of the 24-hour sampling periods (Table 5). The fraction of time spent in transit was relatively consistent between city, season, and cohort, from 4.3% for the Baltimore COPD patients and Boston children during the winter to 6.5% for the Boston children during the summer. Summarized below are additional patterns that emerged from the time–activity data.

- Children in Boston spent significantly more time outdoors during the summer (11.3%) as compared to the winter (2.1%) (*t* test: $P < 0.0001$).
- During the winter in Boston, children and seniors spent a similar fraction of time outdoors (2.1% and 3.3%, respectively).
- Children in Baltimore spent more time indoors during the summer (89.1%) compared to children in Boston (82.3%).
- In Baltimore during summer, children and seniors spent a similar fraction of time outdoors (5.4% and 5.7% of the day, respectively).

CHARACTERIZATION OF PERSONAL EXPOSURES AND AMBIENT CONCENTRATIONS

Subject-specific results are presented in Appendix L. The results show similar personal $PM_{2.5}$ exposure distributions among the cohorts within a given season and city. Complete boxplots showing pollutant distributions by cohort, city, and season are presented in Appendix M.[2]

$PM_{2.5}$

During both seasons, median personal $PM_{2.5}$ exposures for all cohorts in Baltimore were typically lower than corresponding median ambient concentrations (Table 6). In Boston, median personal $PM_{2.5}$ exposures were higher than corresponding ambient $PM_{2.5}$ concentrations for both seniors and children during both seasons. During the winter in Baltimore and during both seasons in Boston, children had slightly higher and more variable $PM_{2.5}$ exposures than those for seniors or individuals with COPD. This was partially due to differences in the corresponding ambient $PM_{2.5}$ concentrations while the children were sampled. When mixed models were used to

2. In Appendix M, the ambient concentrations correspond to the distribution of ambient concentration for all days of monitoring, usually approximately 36 days. For summer sampling in Baltimore, there are 2 ambient plots—1 pertaining to the seniors' sampling sessions and 1 to the children's sampling session. During summer in Baltimore, seniors were measured over 37 days during June and July and children were measured during one 10-day period in August. In Boston during summer, there are also additional plots for the ambient concentration for each cohort because 10 seniors were measured during the first 12-day sampling session in mid June, 5 seniors and 5 children were measured during the second 12-day sampling session in early July, and 10 children were measured during the third 12-day sampling session in mid July.

Table 5. Percentage of Time Spent in 6 Microenvironments by City, Season, and Cohort

	Baltimore					Boston			
	Winter			Summer		Winter		Summer	
Microenvironment	Seniors (%)[a]	Children (%)	COPD (%)	Seniors (%)[a,b]	Children (%)	Seniors (%)	Children (%)	Seniors (%)	Children (%)
Indoors at home	NA	64.7	82.3	—	72.8	79.6	66.9	73.3	65.9
Indoors at work	NA	0.0	4.8	—	0.0	1.5	0.0	2.2	0.0
Indoors away from home	NA	26.8	6.2	—	16.3	10.9	26.4	10.9	16.3
Outdoors near home	NA	1.2	0.5	—	1.6	0.2	0.4	1.9	2.4
Outdoors away from home	NA	1.6	1.9	—	3.8	3.1	1.7	5.3	8.9
In transit	NA	5.8	4.3	—	5.4	4.6	4.6	6.4	6.5
Total indoors	NA	91.5	93.3	NA	89.1	92.1	93.3	86.4	82.3
Total outdoors	NA	2.7	2.4	NA	5.5	3.3	2.1	7.3	11.3

[a] NA is not known or not available.

[b] A dash indicates that during summer, this cohort spent on average 94.3% of their time indoors (at home or away from home) or in transit and 5.7% of their time outdoors (near home or away from home).

Table 6. Summary Statistics for Personal Exposures to and Ambient Concentrations of $PM_{2.5}$, EC, and SO_4^{2-}

Season / Cohort	Baltimore[a]						Boston[a]					
	N	Mean	SD	Median	Min	Max	*N*	Mean	SD	Median	Min	Max
$PM_{2.5}$: Personal Exposure												
Winter												
Seniors	160	15.1	14.6	11.7	1.0	88.6	132	14.1	6.0	13.6	2.8	34.8
Children	162	24.0	21.8	16.5	5.2	116.9	120	18.5	12.8	15.4	5.2	96.5
COPD	165	16.4	12.6	13.0	0.8	71.4	—	—	—	—	—	—
Summer												
Seniors	160	22.1	10.1	20.6	6.9	77.9	165	18.8	9.7	17.0	4.8	66.2
Children	64	18.6	8.1	16.4	7.6	44.3	174	30.3	14.2	26.9	8.7	76.6
$PM_{2.5}$: Ambient Concentration												
Winter												
All	40	20.1	9.4	20.1	5.6	49.0	38	11.6	6.8	9.6	2.6	32.3
Summer												
Seniors	37	25.2	11.5	22.5	9.1	47.1	24	12.6	5.4	12.9	4.9	22.5
Children	11	23.2	14.0	19.3	10.2	59.5	24	17.0	11.5	18.1	4.9	46.8
EC: Personal Exposure												
Winter												
Seniors	—	—	—	—	—	—	154	1.4	0.9	1.2	0.3	8.9
Children	162	2.8	1.8	2.3	0.1	11.6	148	1.6	1.6	1.3	0.1	16.8
COPD	163	2.0	1.2	1.7	0.3	6.1	—	—	—	—	—	—
Summer												
Seniors	—	—	—	—	—	—	—	—	—	—	—	—
Children	—	—	—	—	—	—	—	—	—	—	—	—
EC: Ambient Concentration												
Winter												
All	—	—	—	—	—	—	38	1.2	0.6	1.2	0.3	3.5
Summer												
Seniors	—	—	—	—	—	—	—	—	—	—	—	—
Children	—	—	—	—	—	—	—	—	—	—	—	—
SO_4^{2-}: Personal Exposure												
Winter												
Seniors	159	1.9	1.1	1.7	0.3	6.2	149	1.9	1.2	1.5	0.1	6.5
Children	—	—	—	—	—	—	139	2.3	1.7	1.6	0.1	8.8
COPD	160	1.5	0.8	1.5	0.3	4.3	—	—	—	—	—	—
Summer												
Seniors	160	5.7	3.5	5.3	0.5	17.9	163	2.9	1.9	2.6	0.3	11.5
Children	—	—	—	—	—	—	176	5.5	5.6	3.7	0.5	24.9
SO_4^{2-}: Ambient Concentration												
Winter												
All	35	4.0	1.7	3.7	1.2	8.3	38	3.1	1.8	2.3	0.8	7.4
Summer												
Seniors	35	10.5	7.1	8.0	1.7	29.6	20	4.0	2.0	3.8	0.9	8.8
Children	—	—	—	—	—	—	22	6.5	6.0	4.5	0.9	21.4

[a] All data except *N* are micrograms per cubic meter. — is not measured.

control for differences in ambient $PM_{2.5}$ concentrations during the sampling periods, no significant cohort-specific differences in $PM_{2.5}$ exposures were found.

SO_4^{2-} and EC

Personal SO_4^{2-} exposures were measured for seniors in both cities during both seasons, for the COPD subjects in Baltimore during winter only, and for children in Boston during both seasons. Summary statistics are presented in Table 6. Ambient SO_4^{2-} concentrations were higher in summer than in winter in both cities, with generally higher levels in Baltimore than in Boston during both seasons. Seasonal differences in median ambient SO_4^{2-} concentrations were more pronounced in Baltimore (3.7 $\mu g/m^3$ in winter and 8.0 $\mu g/m^3$ in summer) than in Boston (2.3 $\mu g/m^3$ in winter and 3.8 $\mu g/m^3$ in summer).

Personal SO_4^{2-} exposures were higher during the summer than during the winter, reflecting the corresponding higher levels outdoors (Table 6). In Baltimore, median personal SO_4^{2-} exposures for seniors were 1.7 and 5.3 $\mu g/m^3$ during winter and summer, respectively. In Boston, median winter and summer personal SO_4^{2-} exposures for seniors were 1.5 and 2.6 $\mu g/m^3$, respectively. In Boston, the personal SO_4^{2-} exposures for children were comparable to those of the seniors during winter (1.6 $\mu g/m^3$) but higher than those of the seniors during summer (3.7 $\mu g/m^3$). During winter, median personal exposures were comparable between cities, although the ambient levels were slightly higher in Baltimore. During the summer, the median personal SO_4^{2-} exposure for seniors in Baltimore was twice that of Boston, likely due to the twofold higher ambient levels in Baltimore during summer (Suh et al 1994; Sarnat et al 2000).

Owing to cost constraints, EC analysis from the personal exposure filters was conducted only during winter in both cities (children and individuals with COPD in Baltimore and children and seniors in Boston; Table 6). In addition, ambient EC concentrations were not available in Baltimore. In Boston and Baltimore, personal EC exposures were low, with approximately two thirds and less than one third of the samples below the LOD, respectively. Children had slightly higher median EC exposures (2.3 $\mu g/m^3$) than individuals with COPD (1.7 $\mu g/m^3$) in Baltimore. In Boston, children and seniors had similar median personal EC exposures: 1.3 and 1.2 $\mu g/m^3$, respectively. The low personal EC concentrations were comparable to the corresponding ambient levels. The median subject-specific Spearman correlation coefficient between personal EC exposure and ambient EC concentration during winter in Boston was 0.41, approximately half that for SO_4^{2-} ($r_s = 0.82$). EC is associated with diesel combustion and traffic in general, which tend to vary spatially within a metropolitan area (Kinney et al 2000). There are also several indoor sources of EC, making it a poor surrogate for ambient PM, as shown by the low personal–ambient Spearman correlation coefficients for EC compared to SO_4^{2-}. A more detailed spatial analysis of EC within the metropolitan areas studied may allow for assessment of the contribution of traffic-related pollution to personal PM.

O_3, NO_2, and SO_2

During each of the sampling seasons and in both cities, personal exposures to O_3 and SO_2 were generally low and frequently below their respective LODs (Table 7). During winter in particular, subjects were exposed to extremely low levels of O_3 and SO_2 due to the lack of indoor sources for these gases and their high removal rate on indoor surfaces. In Boston, for example, winter O_3 and SO_2 personal exposures were below their LODs 96.8% and 95.4% of the time, respectively, even when corresponding ambient levels were well above their LODs. For comparison, less than 6% of the personal $PM_{2.5}$ exposures were below their LODs during both seasons in both cities (Table 3).

Distributions of personal exposures and ambient concentrations of O_3, NO_2, and SO_2 are presented in Appendix M, Figures M.4 through M.6. During each of the sampling periods for all cohorts, personal exposures to O_3 were extremely low. Almost all personal O_3 exposures were lower than corresponding ambient levels, as well as below their respective method LOD. During winter, the median ambient O_3 concentration was 16.9 ppb in Baltimore and 15.8 ppb in Boston, with median winter personal O_3 exposure less than or equal to 1.0 ppb for all cohorts in both cities (Table 7). Summer median personal O_3 exposures were slightly higher than winter exposures: 2.6 ppb for seniors in Baltimore to 5.1 ppb for children in Boston. During summer, the median ambient concentration was approximately 36 ppb in Baltimore and approximately 23 ppb in Boston. The observed O_3 personal exposures and ambient concentrations are consistent with those found by other researchers (Liu et al 1995). O_3 is a highly reactive gas with a high deposition rate on indoor surfaces, resulting in substantially lower indoor concentrations and personal exposures (Liu et al 1995). Time spent outdoors has been shown to be an important predictor of personal O_3 exposure (Brauer and Brook 1995). Individuals spent a greater fraction of time outdoors during summer in both cities (Table 5).

A rather different pattern was found for personal exposures to NO_2 (Table 7). In Boston, summer personal NO_2 exposures were somewhat higher than winter exposures for seniors and much higher for children. In Baltimore, the seniors' NO_2 exposures were considerably lower during

Table 7. Summary Statistics for Personal Exposures to and Ambient Concentrations of O_3, NO_2, and SO_2[a]

Season / Cohort	Baltimore						Boston					
	N	Mean	SD	Median	Min	Max	*N*	Mean	SD	Median	Min	Max
O_3: Personal Exposure												
Winter												
Seniors	160	−0.1	3.0	0.3	−8.5	10.6	150	1.2	1.7	1.0	−3.9	6.7
Children	135	0.4	2.2	0.6	−7.2	8.7	137	1.3	2.1	1.0	−3.0	11.6
COPD	154	1.1	2.3	0.9	−7.7	11.6	—	—	—	—	—	—
Summer												
Seniors	137	3.8	4.5	2.6	−3.2	21.1	156	5.1	6.6	3.4	−4.5	34.5
Children	59	2.1	4.8	3.0	−10.6	10.7	176	7.0	6.9	5.1	−0.8	48.5
O_3: Ambient Concentration												
Winter												
All	40	18.1	10.0	16.9	1.3	38.5	38	16.8	6.1	15.8	4.9	29.7
Summer												
Seniors	37	37.6	7.9	36.3	21.7	53.6	25	22.9	9.8	23.0	6.8	46.5
Children	11	32.9	11.6	34.7	17.8	58.0	24	27.4	15.3	25.5	5.4	64.9
NO_2: Personal Exposure												
Winter												
Seniors	158	16.2	13.3	14.6	−4.5	94.4	155	13.2	5.5	12.8	1.8	32.4
Children	163	18.0	16.7	13.7	−1.9	105.4	141	20.4	12.5	19.2	0.1	75.8
COPD	164	16.6	16.6	12.3	−2.9	109.0	—	—	—	—	—	—
Summer												
Seniors	159	8.4	9.0	7.1	−5.0	65.8	165	15.2	9.6	13.6	−7.3	48.0
Children	58	13.3	8.3	12.0	0.8	49.0	176	24.5	11.3	26.3	−7.2	59.1
NO_2: Ambient Concentration												
Winter												
All	40	23.0	6.9	24.0	9.6	39.2	38	28.0	8.4	26.9	8.8	45.0
Summer												
Seniors	37	21.4	4.8	20.4	13.5	30.8	25	23.5	6.6	23.1	12.7	37.2
Children	11	20.4	8.1	18.7	11.7	36.9	24	24.9	5.6	22.7	12.7	37.8
SO_2: Personal Exposure												
Winter												
Seniors	155	−0.1	1.6	0.2	−3.3	5.0	155	0.8	1.1	0.7	−1.7	6.2
Children	166	0.3	2.3	0.3	−3.3	16.3	143	0.6	2.5	0.2	−1.8	13.1
COPD	166	0.0	1.4	0.0	−3.4	4.1	—	—	—	—	—	—
Summer												
Seniors	—	—	—	—	—	—	161	0.4	2.0	0.2	−1.3	20.8
Children	—	—	—	—	—	—	174	0.5	1.3	0.2	−1.3	11.9
SO_2: Ambient Concentration												
Winter												
All	40	9.0	4.4	8.4	2.0	18.8	38	8.1	3.7	8.6	2.0	18.2
Summer												
Seniors	—	—	—	—	—	—	25	3.4	1.3	3.0	1.8	7.8
Children	—	—	—	—	—	—	24	3.7	1.2	5.3	1.9	5.9

[a] All data except *N* are parts per billion. — is not measured.

summer than during winter, with children's exposures slightly lower during summer than during winter. The difference in seasonal effect on personal NO_2 exposures by city is somewhat surprising, given the fact that ambient NO_2 concentrations in both cities were lower during summer than during winter. The children's median personal NO_2 exposure in Boston during winter (19.2 ppb) was significantly lower than the median ambient level (26.9 ppb), but during summer the median personal NO_2 exposure for Boston children (26.3 ppb) was higher than the median ambient NO_2 level (22.7 ppb). In contrast, seniors living in Boston had lower personal exposures than ambient levels for both seasons. For Baltimore, all median personal NO_2 exposures were less than corresponding ambient levels.

Distributions of personal NO_2 exposures were essentially the same among the 3 winter cohorts in Baltimore (seniors, children, and COPD), possibly due to the relatively balanced percentage of homes with gas stoves in those cohorts. During the summer in Baltimore and both seasons in Boston, children had higher NO_2 exposures than seniors; these children also had a larger fraction of homes with gas stoves. Mixed models were used to examine the influence of gas stoves in the home on personal NO_2 exposures (Table 8). Results showed higher exposures to NO_2 for participants living in homes with gas stoves than in homes with electric stoves, and all but two cohort differences were found to be statistically significant. Numerous other studies have shown higher home indoor and personal NO_2 exposures for homes with gas stoves (Dockery et al 1981; Brauer et al 1990; Levy et al 1998; Lee et al 2000). The apparent cohort-specific differences in exposures to NO_2 may reflect the use of gas stoves.

Personal exposures to SO_2 were low, mainly due to relatively low ambient concentrations of this pollutant during sampling, lack of indoor or local sources of SO_2, and high deposition rate of SO_2 on indoor surfaces (Table 7). Median personal SO_2 exposures were close to 0 ppb for all of the cohorts in the study, with more than 95% of the samples found to be below their respective LODs. Median ambient SO_2 concentrations in winter were similarly low, 8.4 ppb for Baltimore and 8.6 ppb for Boston. The very low personal SO_2 exposures are consistent with results found by Brauer and collaborators (1989), where mean ambient SO_2 concentrations were 5 times higher than the corresponding personal and home indoor levels measured. The pronounced differences in concentrations among microenvironments are likely due to the deposition of SO_2 on indoor surfaces (Koutrakis et al 1991).

Exposure Factors for Personal $PM_{2.5}$

Several factors (cooking, cleaning, living near a busy road, living in a house versus an apartment, and the presence of a gas stove in the residence) were examined for their influence on personal $PM_{2.5}$ exposures. Given the generally low and less variable distribution of personal exposures to the gases, the exposure factor analyses focused exclusively on quantifying the contribution to personal $PM_{2.5}$ exposures. Mixed regression models (see equation 2) were used to examine the influence of these factors as well as to control for different ambient $PM_{2.5}$ levels on a given day. Because each of the exposure factors is categorical, differences in the model intercepts can be interpreted as the $PM_{2.5}$ exposure contribution from that factor. Table 9 presents results from the models as well as summary statistics for $PM_{2.5}$ stratified by each factor.

Because activity data for Baltimore seniors was unavailable and children were not studied for cooking, analysis of the effects of cooking on personal exposures was only conducted for the COPD cohort in Baltimore and the senior cohort in Boston during both seasons (Table 9). For the COPD subjects, personal exposures were found to be 1.7 $\mu g/m^3$ lower on days when cooking occurred as compared to other days ($P = 0.0382$). For Boston seniors during summer, personal exposures were lower by 2.5 $\mu g/m^3$ on days when participants cooked ($P = 0.0127$). These counterintuitive results are likely due to the small number of cooking days ($n = 43$ for cooking and $n = 122$ for no cooking for the COPD cohort; $n = 34$ for cooking and $n = 131$ for no cooking for the Boston summer seniors).

Similarly, lower exposures to $PM_{2.5}$ were measured on days when cleaning took place for 4 of the 7 groups measured (Table 9). Comparable intercepts were found for children in Baltimore during winter (19.4 $\mu g/m^3$ for cleaning and 19.3 $\mu g/m^3$ for no cleaning) and for seniors in Boston during summer (10.7 $\mu g/m^3$ for cleaning and 10.0 $\mu g/m^3$ for no cleaning). For children in Boston during winter, cleaning activities were associated with a 3 $\mu g/m^3$ increase in $PM_{2.5}$ exposures. All other groups showed slightly lower exposures associated with cleaning. As with cooking, the infrequency of cleaning activities, especially for children, and the unbalanced nature of the data may have led to these unusual results. For example, the Baltimore children's results during the summer were based on only 10 occurrences of cleaning, suggesting a reduced power to assess significant trends. In addition, some misclassification of cleaning activities is likely (eg, cleaning occurred in the homes but was not conducted by the participant).

Living near a busy road was associated with both higher and lower $PM_{2.5}$ exposures, depending on the city, season, and group (Table 9). Of the 9 groups, 3 had higher exposures

Table 8. Mixed-Model Regression Results for Personal NO_2 Exposures by Stove Type

City / Season / Cohort / Stove Type	Stoves (*n*)	NO_2 (ppb) Mean ± SD	NO_2 (ppb) Median	Slope	Slope 95% CI	Slope *P*	Intercept (ppb)	Intercept 95% CI	Intercept *P*	**diff *P*[a]
Baltimore										
Winter										
Seniors										
Electric	78	13.1 ± 14.9	6.8	0.06	(−0.10,0.22)	0.4832	11.1	(4.6,17.6)	0.001	
Gas	80	19.2 ± 10.8	17.8				17.6	(13.5,21.7)	< 0.0001	0.0113
Children										
Electric	64	11.3 ± 14.0	6.9	−0.02	(−0.21,0.17)	0.8269	13.1	(6.7,19.5)	< 0.0001	
Gas	88	23.9 ± 16.9	19.8				22.7	(15.4,29.9)	< 0.0001	0.0024
COPD										
Electric	67	11.2 ± 14.2	5.4	0.15	(−0.04,0.34)	0.1157	7.9	(0.7,15.1)	0.0322	
Gas	97	20.4 ± 17.1	14.5				17.3	(12.1,22.4	< 0.0001	0.0068
Summer										
Seniors										
Electric	124	7.7 ± 9.7	6.1	0.17	(0.0,0.34)	0.0397	4.4	(0.3,8.6)	0.0374	
Gas	35	11.2 ± 5.5	9.9				7.0	(2.8,11.2)	0.0014	0.1896
Children										
Electric	25	9.5 ± 6.1	8.2	0.04	(−0.10,0.18)	0.5619	9.3	(2.4,16.2)	0.0095	
Gas	33	16.2 ± 8.6	14.6				15.5	(10.9,20.1)	< 0.0001	0.0671
Boston										
Winter										
Seniors										
Electric	131	12.0 ± 4.9	12.0	0.18	(0.11,0.24)	< 0.0001	7.9	(5.2,10.6)	< 0.0001	
Gas	24	19.5 ± 4.2	18.8				15.2	(13.1,17.4)	< 0.0001	< 0.0001
Children										
Electric	42	10.2 ± 4.6	8.7	−0.07	(−0.23,0.10)	0.4258	11.1	(6.1,16.1)	< 0.0001	
Gas	82	25.3 ± 12.8	22.2				24.4	(19.5,29.3)	< 0.0001	< 0.0001
Summer										
Seniors										
Electric	141	13.4 ± 8.2	12.8	0.19	(0.04,0.33)	0.0107	8.8	(4.4,13.2)	0.0001	
Gas	24	25.9 ± 10.2	22.8				21.1	(17.0,25.3)	< 0.0001	< 0.0001
Children										
Electric	23	15.1 ± 9.8	11.3	0.28	(0.06,0.50)	0.0124	14.4	(7.0,21.8)	0.0002	
Gas	143	25.5 ± 11.0	26.6				21.8	(15.8,27.8)	< 0.0001	0.002

[a] **diff *P* = statistical difference between intercepts for each cohort. Significance is reported at the 0.05 level.

Table 9. Mixed-Model Regression Results for Personal $PM_{2.5}$ Exposures by Factor[a]

City / Season / Cohort / Factor	*n*	$PM_{2.5}$ ($\mu g/m^3$) Mean ± SD	Median	Slope	Slope 95% CI	Slope *P*	Intercept	Intercept 95% CI	Intercept *P*	**diff *P*[b]
Cooking Events										
Baltimore										
Winter										
COPD										
No Cooking	122	18.0 ± 13.7	13.9	0.19	(0.10,0.27)	< 0.0001	9.8	(6.6,13.0)	< 0.0001	
Cooking	43	11.8 ± 6.6	9.8				8.1	(6.1,10.1)	< 0.0001	0.0382
Summer										
—										
Boston										
Winter										
Seniors										
No Cooking	109	14.3 ± 6.3	13.4	0.25	(0.10,0.41)	0.0019	7.6	(4.7,10.6)	< 0.0001	
Cooking	23	13.5 ± 4.5	13.7				6.5	(4.2,8.9)	< 0.0001	
Summer										
Seniors										
No Cooking	131	19.4 ± 10.2	17.2	0.73	(0.55,0.90)	< 0.0001	10.4	(7.7,13.1)	< 0.0001	
Cooking	34	16.5 ± 6.7	15.0				7.9	(5.4,10.5)	< 0.0001	0.0127
Cleaning Events										
Baltimore										
Winter										
Children										
No Cleaning	151	23.7 ± 20.9	16.7	0.28	(0.21,0.35)	< 0.0001	19.3	(14.2,24.4)	< 0.0001	
Cleaning	11	28.1 ± 32.7	10.3				19.4	(11.6,27.2)	< 0.0001	
COPD										
No Cleaning	130	16.3 ± 13.0	13.1	0.19	(0.11,0.27)	< 0.0001	9.3	(6.5,12.1)	< 0.0001	
Cleaning	35	16.6 ± 11.1	12.3				8.6	(5.9,11.2)	< 0.0001	
Summer										
Seniors	—	—	—	—	—	—	—	—	—	
Children										
No Cleaning	54	19.4 ± 8.3	17.5	0.74	(0.55,0.92)	< 0.0001	5.8	(1.8,9.9)	0.0059	< 0.0001
Cleaning	10	14.2 ± 5.6	12.3				0.8	(−4.1,5.7)	0.7464	
Boston										
Winter										
Seniors										
No Cleaning	91	14.5 ± 5.9	13.8	0.24	(0.08,0.41)	0.0042	8.0	(5.0,11.0)	< 0.0001	
Cleaning	41	13.2 ± 6.2	12.1				7.3	(4.1,10.4)	< 0.0001	
Children										
No Cleaning	109	17.9 ± 11.5	15.3	0.39	(0.18,0.60)	0.0005	12.5	(9.5,15.4)	< 0.0001	
Cleaning	11	24.7 ± 21.8	17.0				15.5	(5.9,25.2)	0.0019	
Summer										
Seniors										
No Cleaning	109	18.1 ± 9.0	16.8	0.71	(0.53,0.88)	< 0.0001	10.0	(7.5,12.5)	< 0.0001	
Cleaning	56	20.0 ± 10.9	18.2				10.7	(8.0,13.4)	< 0.0001	

Table continues on next page

[a] — is not available.

[b] **diff *P* = statistical difference between intercepts for each cohort. Significance is reported at the 0.05 level.

[c] Busy road = home is located within 100 yards of a busy road.

Table 9 (*continued*). Mixed-Model Regression Results for Personal $PM_{2.5}$ Exposures by Factor[a]

City / Season / Cohort / Factor	*n*	$PM_{2.5}$ (μg/m^3) Mean ± SD	Median	Slope	Slope 95% CI	Slope *P*	Intercept	Intercept 95% CI	Intercept *P*	**diff *P*[b]
Cleaning Events / Boston (*continued*)										
Summer (*continued*)										
Children										
No Cleaning	150	30.5 ± 14.5	27.0	0.89	(0.76,1.03)	< 0.0001	14.7	(11.2,18.2)	< 0.0001	
Cleaning	24	28.9 ± 12.1	25.9				11.0	(7.5,14.6)	< 0.0001	< 0.0044
Home Proximity to Busy Road[c]										
Baltimore										
Winter										
Seniors										
No busy road	104	15.0 ± 16.7	10.2	0.21	(0.09,0.34)	0.0009	4.0	(0.5,7.5)	0.0254	
Busy road	56	15.4 ± 9.8	14.5				3.4	(0.0,6.8)	0.0471	
Children										
No busy road	67	15.0 ± 16.8	15.5	0.28	(0.21,0.35)	< 0.0001	18.1	(10.2,25.9)	< 0.0001	
Busy road	85	27.8 ± 25.8	17.7				20.2	(13.1,27.3)	< 0.0001	
COPD										
No busy road	92	13.7 ± 8.0	12.9	0.19	(0.12,0.27)	< 0.0001	9.1	(5.8,12.4)	< 0.0001	
Busy road	73	19.9 ± 16.0	13.3				10.0	(7.1,12.8)	< 0.0001	
Summer										
Seniors										
No busy road	107	20.7 ± 8.0	20.2	0.52	(0.37,0.66)	< 0.0001	7.9	(4.6,11.2)	< 0.0001	
Busy road	53	24.9 ± 13.1	21.8				14.1	(8.4,19.8)	< 0.0001	0.0074
Children										
No busy road	50	19.0 ± 8.7	18.0	0.70	(0.47,0.94)	< 0.0001	6.1	(1.1,11.0)	0.0171	
Busy road	14	16.9 ± 5.7	15.1				4.1	(−0.7,9.0)	0.0943	
Boston										
Winter										
Seniors										
No busy road	16	21.3 ± 6.4	20.2	0.23	(0.07,0.39)	0.0052	18.1	(13.4,22.8)	< 0.0001	
Busy road	116	13.1 ± 5.2	12.9				7.9	(5.1,10.7)	< 0.0001	< 0.0001
Children										
No busy road	29	21.4 ± 16.4	19.0	0.39	(0.19,0.59)	0.0002	14.0	(9.9,18.1)	< 0.0001	
Busy road	84	18.1 ± 11.8	15.5				12.9	(9.0,16.7)	< 0.0001	
Summer										
Seniors										
No busy road	47	20.4 ± 13.8	17.0	0.72	(0.55,0.88)	< 0.0001	11.9	(8.7,15.1)	< 0.0001	
Busy road	118	18.1 ± 7.4	17.0				9.3	(6.6,12.1)	< 0.0001	
Children										
No busy road	43	30.5 ± 12.7	28.0	0.87	(0.71,1.02)	< 0.0001	18.2	(14.0,22.5)	< 0.0001	
Busy road	103	24.2 ± 8.8	23.2				13.4	(10.5,16,4)	< 0.0001	0.008

Table continues on next page

[a] — is not available.

[b] **diff *P* = statistical difference between intercepts for each cohort. Significance is reported at the 0.05 level.

[c] Busy road = home is located within 100 yards of a busy road.

Table 9 (*continued*). Mixed-Model Regression Results for Personal $PM_{2.5}$ Exposures by Factor[a]

City / Season / Cohort / Factor	*n*	$PM_{2.5}$ ($\mu g/m^3$) Mean ± SD	Median	Slope	Slope 95% CI	Slope *P*	Intercept	Intercept 95% CI	Intercept *P*	**diff *P*[b]
Home Type										
Baltimore										
Winter										
Seniors										
Apartment	88	11.2 ± 11.0	7.7	0.21	(0.09,0.33)	0.0005	2.5	(0.6,4.4)	0.0112	
House	72	19.9 ± 16.9	14.9				8.4	(7.1,9.7)	< 0.0001	< 0.0001
Children										
Apartment	12	76.8 ± 25.5	75.9	—	—	—	—	—	—	
House	140	20.3 ± 15.3	15.5				—	—	—	
COPD										
Apartment	44	26.8 ± 16.6	25	0.19	(0.11,0.27)	< 0.0001	10.7	(4.2,17.2)	0.0015	
House	121	12.6 ± 7.9	12.6				9.5	(6.6,12.4)	< 0.0001	
Summer										
Seniors										
Apartment	112	22.8 ± 9.7	21.1	0.52	(0.37,0.66)	< 0.0001	8.1	(4.5,11.7)	< 0.0001	
House	48	20.3 ± 11.0	17.5				3.3	(−0.3,6.9)	0.0698	0.0409
Children										
Apartment	—	—	—	—	—	—	—	—	—	
House	64	18.6 ± 8.1	16.4							
Boston										
Winter										
Seniors										
Apartment	116	14.6 ± 6.1	13.8	0.25	(0.08,0.42)	0.0037	8.4	(5.8,11.0)	< 0.0001	
House	16	10.7 ± 3.8	10.4				7.0	(3.6,10.4)	< 0.0001	
Children										
Apartment	56	19.7 ± 17.1	14.3	0.37	(0.18,0.57)	0.0002	15.8	(10.5,21.0)	<0.0001	
House	56	16.5 ± 6.9	15.4				11.3	(8.2,14.4)	<0.0001	0.0256
Summer										
Seniors										
Apartment	106	17.8 ± 7.2	16.9	0.71	(0.55,0.88)	< 0.0001	9.2	(6.3,12.0)	< 0.0001	
House	59	20.4 ± 12.9	17.4				11.7	(8.8,14.6)	< 0.0001	0.1013
Children										
Apartment	120	31.4 ± 14.9	27.6	0.89	(0.74,1.03)	< 0.0001	15.3	(12.1,18.6)	< 0.0001	
House	44	27.1 ± 13.4	23.3				11.6	(8.1,15.2)	< 0.0001	0.0285
Stove Type										
Baltimore										
Winter										
Seniors										
Electric stove	78	12.6 ± 11.2	9.9	0.22	(0.09,0.34)	0.0008	2.4	(0.4,4.3)	0.0190	
Gas stove	82	17.5 ± 16.9	14.2				5.2	(1.2,9.1)	0.0104	0.0495

Table continues on next page

[a] — is not available.

[b] **diff *P* = statistical difference between intercepts for each cohort. Significance is reported at the 0.05 level.

[c] Busy road = home is located within 100 yards of a busy road.

Table 9 (*continued*). Mixed-Model Regression Results for Personal $PM_{2.5}$ Exposures by Factor[a]

City / Season / Cohort / Factor	*n*	$PM_{2.5}$ (μg/m^3) Mean ± SD	Median	Slope	Slope 95% CI	Slope *P*	Intercept	Intercept 95% CI	Intercept *P*	**diff *P*[b]
Stove Type / Baltimore (*continued*)										
Winter (*continued*)										
Children										
Electric stove	66	19.8 ± 14.7	15.5	0.28	(0.21,0.35)	< 0.0001	17.8	(11.2,24.5)	< 0.0001	
Gas stove	86	28.6 ± 26.1	17.8				20.0	(11.4,28.5)	< 0.0001	
COPD										
Electric stove	66	17.4 ± 15.4	11.9	0.19	(0.11,0.27)	< 0,0001	9.8	(7.3,12.3)	< 0.0001	
Gas stove	99	15.7 ± 10.3	13.3				8.6	(5.2,12.1)	< 0.0001	
Summer										
Seniors										
Electric stove	124	21.4 ± 9.5	20.4	0.52	(0.38,0.67)	< 0.0001	7.8	(3.9,11.8)	0.0001	
Gas stove	36	24.2 ± 11.9	21.0				10.4	(6.1,14.7)	< 0.0001	
Children										
Electric stove	29	17.2 ± 7.1	15.6	0.70	(0.47,0.94)	< 0.0001	5.0	(−0.9,10.8)	0.0935	
Gas stove	35	19.7 ± 8.8	19.0				6.1	(0.8,11.5)	0.0247	
Boston										
Winter										
Seniors										
Electric stove	111	14.5 ± 6.1	13.8	0.25	(0.08,0.42)	0.0036	7.4	(4.4,10.4)	< 0.0001	
Gas stove	21	12.0 ± 4.8	11.3				8.3	(6.1,10.4)	< 0.0001	
Children										
Electric stove	37	16.7 ± 9.0	14.2	0.39	(0.19,0.59)	0.0002	13.3	(10.5,16.2)	< 0.0001	
Gas stove	68	19.5 ± 15.2	16.0				12.9	(8.4,17.5)	< 0.0001	
Summer										
Seniors										
Electric stove	142	18.7 ± 9.7	16.9	0.71	(0.55,0.88)	< 0.0001	10.2	(7.8,12.6)	< 0.0001	
Gas stove	23	19.1 ± 9.4	17.5				10.1	(7.2,12.9)	< 0.0001	
Children										
Electric stove	23	24.2 ± 10.8	20.7	0.88	(0.73,1.02)	< 0.0001	8.8	(4.9,12.7)	< 0.0001	
Gas stove	141	31.3 ± 14.9	26.9				16.1	(12.3,20.0)	< 0.0001	< 0.0001

[a] — is not available.

[b] **diff *P* = statistical difference between intercepts for each cohort. Significance is reported at the 0.05 level.

[c] Busy Road = home is located within 100 yards of a busy road.

associated with living near a busy road: Baltimore children and COPD subjects in winter and Baltimore seniors in summer had 2.1, 0.9, and 6.2 μg/m^3 higher exposures when living near a busy road. For the Baltimore summer seniors, participants living near a busy road had exposures that were almost twice as high as those not living near a busy road (14.1 and 7.9 μg/m^3). Conversely, living near a busy road was associated with lower $PM_{2.5}$ exposures for the other groups. As with the results for the cooking and cleaning, the distribution between those living near versus those not living near a busy road was unbalanced, especially in Boston.

For 5 of the 7 groups, $PM_{2.5}$ exposures were higher for participants living in apartments as compared to single-family houses (Table 9). No results are provided for children in Baltimore because only 1 lived in an apartment during winter and none lived in apartments during summer. The largest differences in exposures were seen for Baltimore summer seniors, with an intercept of 8.1 μg/m^3 for apartments and 3.3 μg/m^3 for houses. In contrast, Baltimore winter seniors living in houses had a much higher intercept, 8.4 μg/m^3, compared to an intercept of 2.5 μg/m^3 for those living in houses. Boston summer seniors also had higher exposures associated with living in

houses, with an intercept of 11.7 μg/m^3 for houses and 9.2 μg/m^3 for apartments.

Living in a home with a gas stove was associated with moderately higher $PM_{2.5}$ exposures for 6 of the 9 groups (Table 9). The electric stove intercepts ranged from 2.4 μg/m^3 for Baltimore winter seniors to 17.8 μg/m^3 for Baltimore winter children. The gas stove intercepts ranged from 5.2 μg/m^3 for Baltimore winter seniors to 20.0 μg/m^3 for the Baltimore winter children. Differences in the electric stove and gas stove intercepts ranged from 0.9 μg/m^3 for Boston winter seniors to 7.3 μg/m^3 for Boston summer children. For Boston children in summer, 141 observations were associated with gas stoves, whereas 23 observations were associated with gas stoves for seniors in Boston during summer. Therefore, other confounding factors may be attributable for the observed gas stove results.

IMPACT OF AMBIENT PARTICLES ON PERSONAL EXPOSURES

Mixed-Model Analyses

Mixed-model regression analysis was used to assess the influence of ambient sources on personal $PM_{2.5}$ exposures by city, season, and cohort. Infiltration factors (as estimated by mixed-model slopes) for ambient $PM_{2.5}$ attenuation ranged from 0.23 (95% confidence interval [CI]: 0.15,0.30) in Baltimore and Boston during winter to 0.81 (95% CI: 0.70,0.91) in Boston during summer (Table 10, Figure 1).

For the infiltration factor, one model was used that included all data from both cities and seasons. For this model, a variable called *city–season* was created that was comprised of a number from 1 to 4 (ie, 1 was Baltimore winter, 2 was Baltimore summer, 3 was Boston winter, and 4 was Boston summer). Personal $PM_{2.5}$ exposure was then regressed on an interaction term with this city–season variable and the ambient $PM_{2.5}$ concentration. A simplified version of the regression model is: y = city_season + city_season•x, where y is personal $PM_{2.5}$ or SO_4^{2-} and x is ambient $PM_{2.5}$ or SO_4^{2-}. All infiltration factors were less than 1, indicating that subjects were exposed to less than 100% of the ambient pollution levels. Contributions from nonambient sources ranged from 8.1 μg/m^3 (95% CI: 5.1,11.0) for subjects in Baltimore during summer to 15.5 μg/m^3 (95% CI: 12.5,18.6) for subjects in Baltimore during winter.

To examine differences in infiltration factors by season, the slopes (β_k) from mixed-model regressions were calculated and compared in each city separately. Season was a highly significant determinant of the influence of ambient $PM_{2.5}$ on personal exposures in both cities ($P < 0.05$; Table 10). Similarly, in both cities contributions from ambient $PM_{2.5}$ to personal exposures were higher during the summer than the winter, presumably due to home ventilation and/or more time spent outdoors during summer.

Differences in infiltration factors by city were estimated separately during the winter and summer. The relative contribution of ambient $PM_{2.5}$ sources on corresponding personal exposures did not vary by city during the winter ($P = 0.98$), with observed β_k values of 0.23 both in Baltimore and Boston. In contrast, during the summer, ambient $PM_{2.5}$ comprised a significantly greater fraction of personal $PM_{2.5}$ exposures in Boston (81%) than in Baltimore (55%) ($P < 0.003$). Nonambient $PM_{2.5}$ contributions were also higher during winter than during summer in both cities.

Similar analyses of infiltration factors were conducted using SO_4^{2-} exposures and concentrations (Table 11). In contrast to $PM_{2.5}$, nonambient contributions to SO_4^{2-} (ie, the model intercepts) were expected to be negligible and thereby to provide a means of comparison with the previous results. The intercepts were all quite low: Baltimore winter and Boston summer values were approximately 0.0 and 0.2, respectively, whereas the Baltimore summer value was 1.3 μg/m^3 and the value for Boston winter was 0.5 μg/m^3. The observed infiltration factors for SO_4^{2-} were higher in Boston (0.60 in winter and 0.83 in summer) than in Baltimore (0.42 in winter and 0.41 in summer) ($P < 0.0001$ for both seasons), with no difference in infiltration factors between seasons in Baltimore ($P = 0.95$). The infiltration factors in Boston showed a strong seasonal trend: 0.60 in winter (95% CI: 0.54,0.66) and 0.83 in summer (95% CI: 0.74,0.91).

Table 10. Mixed-Model Results for Regression of Ambient $PM_{2.5}$ Concentrations on Personal $PM_{2.5}$ Exposures by City and Season Using Empirical Standard Errors

City / Season	Slope	Slope 95% CI	Intercept (μg/m^3)	Intercept 95% CI
Baltimore				
Winter[a]	0.23[b]	(0.15,0.30)	15.5[b]	(12.5,18.6)
Summer	0.55[b]	(0.42,0.68)	8.1[b]	(5.1,11.0)
Boston				
Winter	0.23[c]	(0.09,0.36)	14.2[b]	(11.7,16.8)
Summer	0.81[b]	(0.70,0.91)	12.3[b]	(10.0,14.6)

[a] These parameter estimates include data from the COPD cohort study, which only took place in Baltimore during winter. Without the COPD cohort data, the intercept is 16.3 μg/m^3 (95% CI 12.8,19.8) and the slope is 0.25 (95% CI 0.15,0.35).

[b] $P < 0.0001$.

[c] $P < 0.05$.

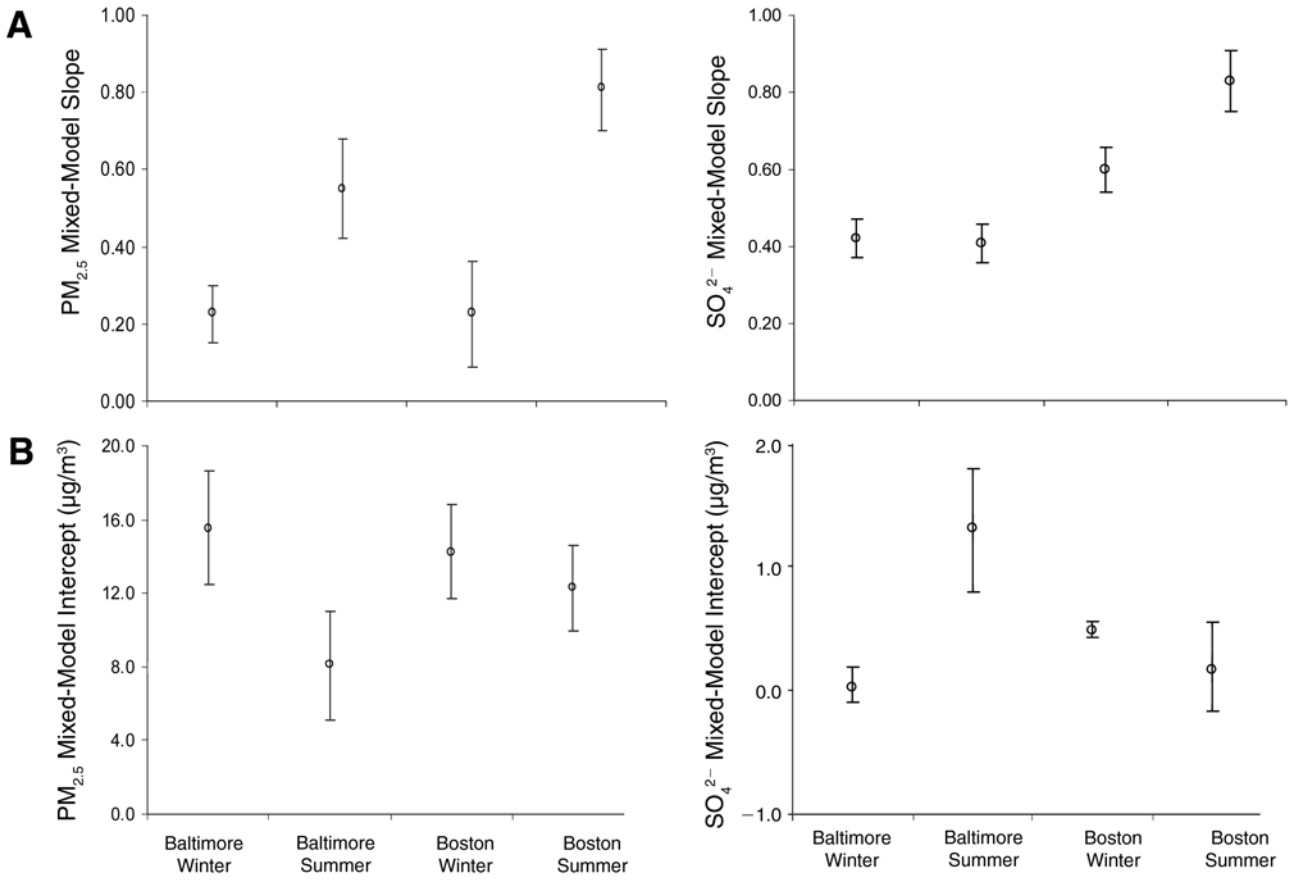

Figure 1. $PM_{2.5}$ and SO_4^{2-} mixed-model regression estimates (± 95% CI) by city–season group. *A.* Slope. ***B.*** Intercept. (Note the difference in scale for the 2 panels.)

City and season likely serve as surrogates for other factors, such as ventilation and meteorology, which may directly affect the influence of ambient PM on personal PM exposures (Suh et al 1994; Sarnat et al 2000). The 2 cities analyzed in this study represent distinct environments with different climatic conditions and pollutant profiles. Baltimore has hot summers and mild winters, whereas Boston generally has mild summers and harsh winters. In the current analysis, the seasonal difference in infiltration factors (slopes) was distinct within a given city. During the summer, $PM_{2.5}$ and SO_4^{2-} infiltration factors were higher in Boston than in Baltimore, a difference that may be attributable to the greater use of air conditioners in Baltimore homes (Suh et al 1997; Abt et al 2000). One limitation of our study design is that we did not have a record of air conditioner use, rather only the presence or absence of air conditioning units in the home. From the current data, it is difficult to determine the differential influence of geographic and seasonal effects on the ambient contribution to personal exposures.

Table 11. Mixed-Model Results for Regression of Ambient SO_4^{2-} Concentrations on Personal SO_4^{2-} Exposures by City and Season

City / Season	Slope	Slope 95% CI	Intercept (µg/m^3)	Intercept 95% CI
Baltimore				
Winter[a]	0.42[b]	(0.36,0.47)	0.0	(−0.1,0.2)
Summer	0.41[b]	(0.37,0.46)	1.3[b]	(0.7,1.9)
Boston				
Winter	0.60[b]	(0.54,0.66)	0.5[b]	(0.4,0.6)
Summer	0.83[b]	(0.74,0.91)	0.2	(−0.2,0.5)

[a] These parameter estimates include data from the COPD cohort study, which only took place in Baltimore during winter. With the COPD cohort data, the intercept is −0.1 µg/m^3 (95% CI −0.3,0.1) and the slope is 0.51 (95% CI 0.46,0.56).

[b] $P < 0.0001$.

Analyses by Cohort

Differences in infiltration factor by cohort were also examined (Table 12). The observed infiltration factors and nonambient $PM_{2.5}$ concentrations (as determined from the slopes and intercepts of the models, respectively) for $PM_{2.5}$ were statistically significant. On average, ambient $PM_{2.5}$ was a greater fraction of the children's personal exposures than other cohorts. None of these higher infiltration factors in children were significantly different, however, from those found for the other cohorts. Nonambient $PM_{2.5}$ contributions to personal exposures also tended to be higher for the children's cohort as compared to the other cohorts in both cities and during both seasons. One exception was found for children in Baltimore during the summer—nonambient $PM_{2.5}$ contributions (5.6 ± 2.7 μg/m^3) were less than that for seniors (8.5 ± 1.7 μg/m^3). Only in Boston during summer did children have a significantly higher nonambient contribution (15.3 ± 1.6 μg/m^3) than seniors (9.9 ± 1.6 μg/m^3) ($P = 0.02$). The Boston children also had significantly higher nonambient contributions during summer than the Baltimore children (5.6 ± 2.7 μg/m^3) ($P = 0.003$).

Similar to the results for $PM_{2.5}$, SO_4^{2-} infiltration factors did not differ by cohort, but the difference between slopes for the senior and COPD cohorts in Baltimore during winter was significant ($P < 0.005$) (Table 13). Seasonal differences were seen for seniors in Baltimore during winter and summer ($P < 0.02$) and between children in Boston during summer and winter ($P < 0.0001$). In terms of differences by city, seniors in Boston during summer had a significantly higher infiltration factor than seniors in Baltimore during summer.

VARIANCE COMPONENT ANALYSIS

To estimate the differential influence of explained and unexplained measurement error on the personal–ambient relations, we conducted a simple variance component analysis. Analysis of variance was used to determine how much of the variance in subject-specific regression slopes was due to the effect of city, season, and unexplained error. Table 14 presents results from the analysis of variance and variance component analyses, showing city and season to be highly significant factors in determining subject-specific regression slopes. In addition, 72% of the model variance was due to the effect of season, 25% to the city effect, and less than 3% to model error. These results suggest that city and season are important factors in determining the

Table 12. Mixed-Model Results for Regression of Ambient $PM_{2.5}$ Concentration on Personal $PM_{2.5}$ Exposures by Cohort

City / Season / Cohort	Slope	Slope 95% CI	Intercept (μg/m^3)	Intercept 95% CI
Baltimore				
Winter				
Seniors	0.25[a]	(0.06,0.43)	13.1[b]	(8.7,17.5)
Children	0.27[b]	(0.18,0.36)	18.0[b]	(12.7,23.4)
COPD	0.15[a]	(0.06,0.24)	14.7[b]	(8.6,20.8)
Summer				
Seniors	0.52[b]	(0.38,0.67)	8.5[b]	(5.0,11.9)
Children	0.70[b]	(0.48,0.93)	5.6[a]	(1.2,10.0)
Boston				
Winter				
Seniors	0.20[a]	(0.03,0.37)	12.9[b]	(9.7,16.0)
Children	0.29[b]	(0.10,0.49)	14.6[b]	(10.8,18.4)
Summer				
Seniors	0.74[b]	(0.56,0.92)	9.9[b]	(7.5,12.3)
Children	0.79[b]	(0.66,0.92)	15.3[b]	(12.0,18.7)

[a] $P < 0.05$.

[b] $P < 0.0001$.

Table 13. Mixed-Model Results for Regression of Ambient SO_4^{2-} Concentration on Personal SO_4^{2-} Exposures by Cohort[a]

City / Season / Cohort	Slope	Slope 95% CI	Intercept (μg/m^3)	Intercept 95% CI
Baltimore				
Winter				
Seniors	0.49[b]	(0.44,0.54)	−0.1	(−0.3,0.1)
Children	—	—	—	—
COPD	0.36[b]	(0.29,0.44)	0.1	(−0.1,0.3)
Summer				
Seniors	0.41[b]	(0.37,0.46)	1.3[b]	(0.7,1.9)
Children	—	—	—	—
Boston				
Winter				
Seniors	0.59[b]	(0.52,0.66)	0.5[b]	(0.4,0.6)
Children	0.61[b]	(0.51,0.71)	0.4[b]	(0.3,0.6)
Summer				
Seniors	0.80[b]	(0.56,1.04)	0.3	(−0.5,1.1)
Children	0.86[b]	(0.80,0.93)	−0.4[c]	(−0.6,−0.3)

[a] — is not analyzed.

[b] $P < 0.0001$.

[c] $P < 0.05$.

relation between personal and ambient $PM_{2.5}$ for each subject. As a result, we concluded that in future studies emphasis should be placed on evaluating seasonal changes in housing conditions (such as measuring air exchange rate or collecting ventilation information) rather than on more precise $PM_{2.5}$ measurement techniques.

ASSOCIATIONS AMONG POLLUTANT CONCENTRATIONS AND PERSONAL EXPOSURES

Model 1: Ambient–Ambient Associations

Ambient $PM_{2.5}$ concentrations were significantly associated with corresponding ambient concentrations of several gaseous copollutants in both Baltimore and Boston, although the strength and direction for several of these associations differed by city and by season (Table 15). Ambient O_3 was significantly positively associated with ambient $PM_{2.5}$ during summer (Boston slope: 0.51; 95% CI: 0.34,0.68; Baltimore slope: 0.89; 95% CI: 0.62,1.16) and significantly negatively associated with ambient $PM_{2.5}$ during winter (Boston slope: −0.53; 95% CI: −0.85,−0.22; Baltimore slope: −0.67; 95% CI: −0.91,−0.43). In Boston, associations between ambient $PM_{2.5}$ and ambient NO_2 were positive during both seasons and significantly so in winter (winter slope: 0.64; 95% CI: 0.47,0.82; summer slope: 0.44; 95% CI: −0.04,0.92).

In Baltimore, associations between ambient $PM_{2.5}$ and ambient NO_2 were positive during both seasons, but significantly so only during summer (winter slope: 0.29; 95% CI: −0.14,0.72; summer slope: 0.67; 95% CI: 0.08,1.26). Only during winter were ambient levels of SO_2 in Boston significantly associated with ambient $PM_{2.5}$ (slope: 0.80; 95% CI: 0.23,1.37), despite a summer slope value twice as large (slope: 1.58; 95% CI: −0.79,3.94). The variability in this summer association was likely due to greater scatter around this association. There was a weak negative association between ambient SO_2 and ambient $PM_{2.5}$ during the winter in Baltimore (slope: −0.38; 95% CI: −1.07,0.31). Finally, ambient $PM_{2.5}$ was positively associated with ambient CO in both cities during both seasons (Boston winter slope: 24.40; 95% CI: 17.90,30.90; Boston summer slope: 33.66; 95% CI: 6.75,45.14; Baltimore winter slope: 13.96; 95% CI: 8.63,19.29; Baltimore summer slope: 6.07; 95% CI: −16.47,28.61).

Model 2: Personal–Ambient Associations

Ambient $PM_{2.5}$ concentrations were positively associated with corresponding personal $PM_{2.5}$ exposures (Boston summer slope: 0.77; 95% CI: 0.65,0.89; Baltimore summer slope 0.51; 95% CI: 0.37,0.66; Boston winter slope: 0.33; 95% CI: 0.13,0.53; Baltimore winter slope 0.26; 95% CI: 0.18,0.34) (Table 16). During each season, these associations were highly significant. In both cities, slightly more than half of the subjects sampled during the summer (25 of 49) had significant correlation coefficients, resulting in a median r_s of 0.56 in Boston and 0.61 in Baltimore (Figure 2). Substantially fewer subjects (12 of 72) had significant correlations during winter, with a considerably

Table 14. Analysis of Variance and Variance Component Analysis Results for Subject-Specific Regressions of Personal $PM_{2.5}$ on Ambient $PM_{2.5}$

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F	$P > F$
Model	2	8.6946	4.3473	22.45	< 0.0001
Error	121	23.4260	0.1936		
Corrected Total	123	32.1206			

R-Square	Coefficient of Variation	Root Mean Square Error	Slope Mean
0.2707	94.9539	0.4400	0.4634

Source of Variation	Degrees of Freedom	Type III Sum of Squares	Mean Square	F	$P > F$
City	1	1.8800	1.8800	9.71	0.0023
Season	1	5.3853	5.3853	27.82	< 0.0001

Variance Component	Mean Square	Total Variance
City	1.8800	1.8800/7.4589 = 25.2%
Season	5.3853	5.3853/7.4589 = 72.2%
Error	0.1936	0.1936/7.4589 = 2.6%
Total	7.4589	

Table 15. Mixed-Model Regression Results for Ambient–Ambient (Model 1) Associations[a]

Dependent Variable/ Independent Variable/ Season	Baltimore					Boston				
	Slope	Slope 95% CI	Intercept (μg/m^3)	Intercept 95% CI	*n*	Slope	Slope 95% CI	Intercept (μg/m^3)	Intercept 95% CI	*n*
Ambient $PM_{2.5}$										
Ambient O_3										
Winter	**−0.67**[b]	**(−0.91,−0.43)**	32.28	(27.54,37.02)	38	**−0.53**	**(−0.85,−0.22)**	20.67	(14.73,26.60)	38
Summer	**0.89**[b]	**(0.62,1.16)**	−7.61	(−18.31,3.09)	46	**0.51**[b]	**(0.34,0.68)**	2.34	(−2.69,7.37)	36
Ambient NO_2										
Winter	0.29	(−0.14,0.72)	13.47	(2.53,24.41)	38	**0.64**[b]	**(0.47,0.82)**	−6.28	(−11.55,−1.01)	38
Summer	**0.67**	**(0.08,1.26)**	10.76	(−2.27,23.79)	46	0.44	(−0.04,0.92)	4.71	(−8.22,17.64)	36
Ambient SO_2										
Winter	−0.38	(−1.07,0.31)	23.59	(16.10,31.08)	38	**0.80**	**(0.23,1.37)**	5.20	(−0.01,10.41)	38
Summer	—	—	—	—	—	1.58	(−0.79,3.94)	9.87	(−0.37,20.10)	36
Ambient CO										
Winter	**13.96**[b]	**(8.63,19.29)**	7.39	(2.00,12.78)	38	**24.40**[b]	**(17.90,30.90)**	−7.60	(−13.01,−2.18)	38
Summer	6.07	(−16.47,28.61)	22.04	(10.26,33.82)	46	**33.66**	**(6.75,45.14)**	−1.71	(−15.01,30.90)	36
Ambient SO_4^{2-}										
Ambient O_3										
Winter	**−0.07**	**(−0.13,−0.01)**	5.22	(3.93,6.51)	33	−0.07	(−0.17,0.02)	4.29	(2.57,6.02)	38
Summer	**0.56**[b]	**(0.32,0.80)**	−10.98	(−20.43,−1.53)	33	**0.25**[b]	**(0.16,0.34)**	−1.10	(−4.04,1.84)	31
Ambient NO_2										
Winter	**0.09**[b]	**(0.01,0.17)**	1.95	(0.17,3.73)	33	**0.15**	**(0.09,0.20)**	−1.07	(−2.62,0.49)	38
Summer	0.1	(−0.39,0.59)	8.32	(−2.48,19.12)	33	0.20	(−0.08,0.47)	0.90	(−6.71,8.51)	31
Ambient SO_2										
Winter	−0.01	(−0.13,0.11)	4.01	(2.60,5.42)	33	**0.19**	**(0.03,0.34)**	1.55	(0.16,2.94)	38
Summer	—	—	—	—	—	0.23	(−1.02,1.49)	4.89	(−0.94,10.72)	31
Ambient CO										
Winter	0.10	(−1.13,1.33)	3.87	(2.42,5.32)	33	**5.40**[b]	**(3.30,7.50)**	−1.20	(−2.94,0.53)	38
Summer	−11.05	(−28.49,6.39)	15.55	(6.87,24.23)	33	**13.59**	**(1.92,25.26)**	−3.34	(−11.43,4.74)	31

[a] Bold type indicates significance at the 0.05 level. — is not measured.

[b] Significant at the 0.0001 level.

lower median winter r_s (Boston winter r_s = 0.38; Baltimore winter, r_s = 0.29).

Stronger and less variable personal–ambient associations were found for SO_4^{2-}, a component of $PM_{2.5}$ with few indoor sources (Table 16; Boston winter slope: 0.55; 95% CI: 0.49,0.61; Boston summer slope: 0.74; 95% CI: 0.67,0.80; Baltimore winter slope: 0.42; 95% CI: 0.36,0.48; Baltimore summer slope: 0.38; 95% CI: 0.31,0.46) (Mage et al 1999). Significant personal–ambient SO_4^{2-} correlations were found for 41 of 43 subjects during the summer and 37 of 56 subjects during the winter (Figure 2). Previous studies also found stronger personal–ambient associations for SO_4^{2-} as compared to $PM_{2.5}$ (Suh et al 1992; Ozkaynak et al 1996).

In Boston, ambient O_3 during both seasons and ambient NO_2 during summer were also significantly associated with their corresponding personal exposures. For these pollutants, the associations were stronger during summer (O_3 slope: 0.27; 95% CI: 0.18,0.37; NO_2 slope: 0.19; 95% CI: 0.08,0.30) as compared to winter (O_3 slope: 0.04; 95% CI: 0.00,0.07; NO_2 slope: 0.02; 95% CI: −0.1,0.15). The results for these models should be interpreted with caution. For example, the extremely small Boston winter slope for O_3 (0.04), while significant, indicates that the actual impact of ambient O_3 on corresponding personal O_3 exposures was probably minimal. In Boston, the total number of subjects having significant correlation coefficients for personal–ambient O_3 during the summer (15 of 29) was comparable to that found for $PM_{2.5}$ (Figure 2). Corresponding slopes for the personal–ambient O_3 and NO_2 associations

Table 16. Mixed-Model Regression Results for Personal–Ambient (Model 2) Associations[a]

Dependent Variable/ Independent Variable/ Season	Baltimore					Boston				
	Slope	Slope 95% CI	Intercept[b]	Intercept 95% CI	*n*	Slope	Slope 95% CI	Intercept[b]	Intercept 95% CI	*n*
Personal $PM_{2.5}$										
Ambient $PM_{2.5}$										
Winter[c]	**0.26**[d]	**(0.18,0.34)**	14.43	(11.23,17.63)	481	**0.33**	**(0.13,0.53)**	12.75	(9.34,16.15)	253
Summer	**0.51**[d]	**(0.37,0.66)**	8.91	(5.12,12.70)	224	**0.77**[d]	**(0.65,0.89)**	12.64	(9.79,15.48)	330
Personal O_3										
Ambient O_3										
Winter	0.01	(−0.01,0.03)	0.52	(0.21,0.83)	449	**0.04**	**(0.00,0.07)**	0.25	(−0.32,0.82)	288
Summer	0.03	(−0.02,0.09)	0.93	(−1.27,3.12)	196	**0.27**[d]	**(0.18,0.37)**	−1.08	(−2.74,0.57)	332
Personal NO_2										
Ambient NO_2										
Winter	−0.05	(−0.22,0.12)	18.09	(13.22,22.96)	485	0.02	(−0.1,0.15)	16.35	(10.71,21.98)	298
Summer	0.05	(−0.22,0.31)	8.1	(2.06,14.14)	217	**0.19**	**(0.08,0.30)**	12.80	(9.33,16.26)	341
Personal SO_2										
Ambient SO_2										
Winter	**−0.05**	**(−0.09,−0.02)**	0.52	(0.09,0.96)	487	−0.02	(−0.04,0.00)	0.54	(0.29,0.78)	299
Summer	—	—	—	—	—	0.00	(−0.11,0.10)	0.47	(−0.01,0.94)	335
Personal SO_4^{2-}										
Ambient SO_4^{2-}										
Winter	**0.42**[d]	**(0.36,0.48)**	0.06	(−0.14,0.26)	301	**0.55**[d]	**(0.49,0.61)**	0.55	(0.41,0.70)	289
Summer	**0.38**[d]	**(0.31,0.46)**	1.57	(0.68,2.47)	242	**0.74**[d]	**(0.67,0.80)**	0.28	(−0.02,0.57)	291

[a] Bold type indicates significance at the 0.05 level. — is not measured.

[b] Units for $PM_{2.5}$ and SO_4^{2-} are in μg/mg^3. Units for O_3, NO_2, and SO_2 are in ppb.

[c] Parameters excluding an extreme observation from a child during winter: slope = 0.42 (95% CI 0.28,0.57); intercept = 10.76 μg/m^3 (95% CI 8.32,13.19).

[d] Significant at the 0.0001 level.

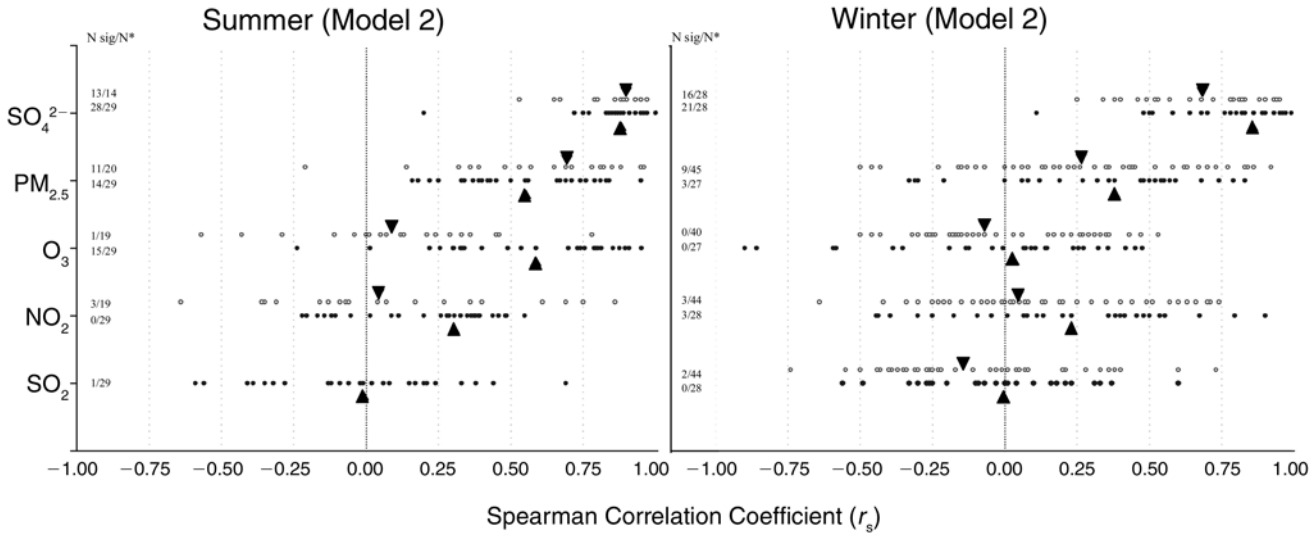

Figure 2. Subject-specific personal–ambient Spearman correlation coefficients. ●, subjects in Boston. ○, subjects in Baltimore. ▼ or ▲, median correlation coefficient. *N sig* refers to the number of significant ($P < 0.05$) correlation coefficients. *N** refers to the total number of correlation coefficients.

in Baltimore were generally lower and more variable than those found in Boston.

Slopes describing associations between personal and ambient SO_2 were close to 0 in Boston during both seasons (Table 16; winter SO_2 slope: −0.02; 95% CI: −0.04,0.00; summer SO_2 slope: 0.00; 95% CI: −0.11,0.10). For SO_2, only 1 of 57 subject-specific correlations during both seasons in Boston was found to be significant. Ambient SO_2 concentrations were negatively associated with corresponding personal SO_2 exposures during the winter in Baltimore (slope: −0.05; 95% CI: −0.09,−0.02).

The presence of a gas stove in a subject's residence did not influence the association between ambient NO_2 concentrations and corresponding personal NO_2 exposures. Mixed-model results (not presented) including a gas stove interaction term were not significant in either season for either city.

Model 3. Cross-Pollutant Associations

An argument can be made for examining cross-pollutant associations using personal SO_4^{2-} exposures instead of personal $PM_{2.5}$ exposures. Personal $PM_{2.5}$ exposures are composed of both ambient and indoor sources whereas SO_4^{2-} is primarily ambient in origin, and the objective of this analysis is to examine associations between ambient pollutant and corresponding personal exposures to a pollutant of ambient origin. Given the strong associations between personal and ambient $PM_{2.5}$, however, analyses using both $PM_{2.5}$ and SO_4^{2-} were conducted.

Ambient O_3, NO_2, and SO_2 concentrations were significantly associated with personal $PM_{2.5}$ exposures in Baltimore during both seasons but in Boston only during the summer (Table 17). The direction of these associations mirrored those of the corresponding ambient–ambient associations.

In Boston, with the exception of the ambient NO_2–personal SO_4^{2-} association during winter, the associations between each of the ambient gas concentrations and personal SO_4^{2-} exposures were stronger, with smaller and less variable slopes, than those found for $PM_{2.5}$ (Table 17). The smaller slopes are due to the smaller range of SO_4^{2-} exposures as compared to $PM_{2.5}$ exposures. These significant results for SO_4^{2-} were expected, given the lack of nonambient SO_4^{2-} contributions to personal exposures that could introduce noise into cross-pollutant relations. Correspondingly, the presence of nonambient contributions to personal $PM_{2.5}$ exposures likely resulted in the observed weaker cross-pollutant associations involving $PM_{2.5}$. Similar to the Boston results, slopes from the cross-pollutant associations involving personal SO_4^{2-} exposures in Baltimore were lower and less variable than those involving personal $PM_{2.5}$ exposures. In both cities, cross-pollutant association slopes were higher with less variability in summer than in winter. These results indicate the greater fraction of SO_4^{2-} and, therefore, ambient $PM_{2.5}$ contributions to total personal $PM_{2.5}$ exposures. Subject-specific correlation coefficients between the ambient O_3, NO_2, and SO_2 concentrations and personal $PM_{2.5}$ exposures showed considerable variation by subject, pollutant, and season (Figure 3).

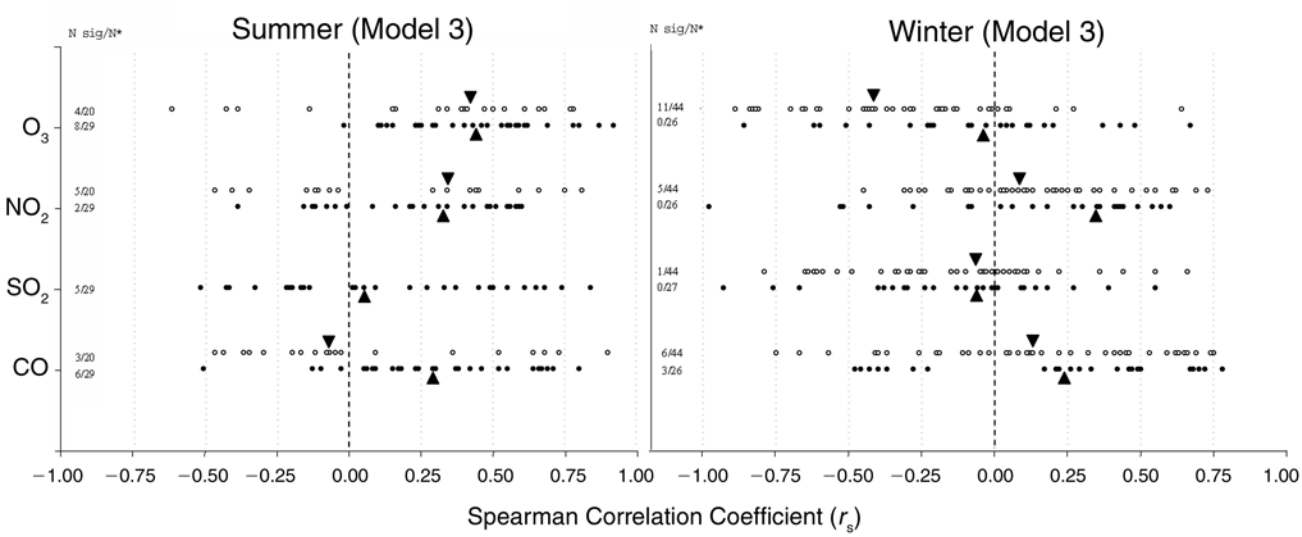

Figure 3. Subject-specific cross-pollutant (personal $PM_{2.5}$ and ambient pollutant) Spearman correlation coefficients. ●, subjects in Boston. ○, subjects in Baltimore. ▼ or ▲, median correlation coefficient. *N sig* refers to the number of significant ($P < 0.05$) correlation coefficients. *N** refers to total number of correlation coefficients.

Table 17. Mixed-Model Regression Results for Cross-Pollutant (Model 3) Associations[a]

Dependent Variable/ Independent Variable/ Season	Baltimore					Boston				
	Slope	Slope 95% CI	Intercept[b]	Intercept 95% CI	*n*	Slope	Slope 95% CI	Intercept[b]	Intercept 95% CI	*n*
Personal $PM_{2.5}$										
Ambient O_3										
Winter	**−0.28**[c]	**(−0.39,−0.17)**	24.74	(20.57,28.91)	487	0.02	(−0.24,0.27)	16.60	(12.15,21.05)	253
Summer	**0.32**	**(0.15,0.49)**	9.22	(2.37,16.07)	224	**0.47**[c]	**(0.37,0.57)**	12.35	(9.58,15.13)	339
Ambient NO_2										
Winter	**0.24**[c]	**(0.13,0.35)**	14.76	(11.30,18.22)	487	0.09	(−0.11,0.30)	14.19	(7.40,20.97)	253
Summer	**0.42**	**(0.13,0.71)**	11.94	(6.13,17.75)	224	**0.39**[c]	**(0.26,0.52)**	14.19	(10.80,17.58)	339
Ambient SO_2										
Winter	**−0.24**[c]	**(−0.36,−0.12)**	22.46	(18.44,26.48)	487	−0.13	(−0.52,0.26)	17.81	(14.09,21.54)	253
Summer	—	—	—	—	—	**1.68**[c]	**(0.98,2.38)**	18.34	(15.11,21.56)	339
Ambient CO										
Winter	**3.94**[c]	**(2.35,5.53)**	16.45	(12.55,20.35)	487	6.38	(−1.87,14.62)	11.68	(4.09,19.27)	253
Summer	5.73	(−4.63,16.09)	17.13	(12.13,22.13)	224	**30.42**[c]	**(21.69,39.15)**	4.68	(−0.76,10.11)	339
Personal SO_4^{2-}										
Ambient O_3										
Winter	**−0.04**[c]	**(−0.05,−0.03)**	2.32	(2.06,2.58)	319	−0.01	(−0.03,0.01)	1.89	(1.39,2.40)	273
Summer	**0.25**[c]	**(0.19,0.31)**	−3.66	(−5.87,−1.45)	160	**0.24**[c]	**(0.20,0.28)**	−1.73	(−2.65,−0.80)	329
Ambient NO_2										
Winter	**0.03**	**(0.00,0.06)**	0.66	(0.11,1.21)	319	**0.07**[c]	**(0.06,0.08)**	0.13	(−0.19,0.44)	280
Summer	0.10	(−0.06,0.26)	3.66	(0.19,7.12)	160	**0.09**[c]	**(0.05,0.13)**	1.34	(0.32,2.37)	338
Ambient SO_2										
Winter	**−0.04**[c]	**(−0.06,−0.02)**	1.71	(1.50,1.92)	319	**0.06**	**(0.02,0.10)**	1.39	(1.13,1.65)	282
Summer	—	—	—	—	—	**0.39**	**(0.15,0.64)**	2.20	(1.14,3.27)	332
Ambient CO										
Winter	0.18	(−0.09,0.45)	1.19	(0.96,1.42)	319	**2.73**[c]	**(2.08,3.38)**	−0.08	(−0.53,0.36)	289
Summer	−4.30	(−8.82,0.23)	7.76	(5.59,9.93)	160	**13.34**[c]	**(9.03,17.64)**	−4.21	(−7.00,−1.42)	339
Personal O_3										
Ambient $PM_{2.5}$										
Winter	0.00	(−0.01,0.02)	0.54	(0.19,0.89)	443	0.02	(0.00,0.05)	0.41	(0.01,0.80)	288
Summer	0.03	(−0.03,0.08)	1.49	(−0.19,3.17)	196	**0.27**[c]	**(0.14,0.39)**	1.85	(0.19,3.51)	323
Personal NO_2										
Ambient $PM_{2.5}$										
Winter	−0.05	(−0.18,0.07)	18.04	(14.38,21.71)	479	**0.21**	**(0.10,0.33)**	14.34	(11.52,17.16)	297
Summer	0.02	(−0.08,0.12)	8.52	(5.85,11.19)	217	−0.03	(−0.10,0.04)	17.79	(14.63,20.95)	332
Personal SO_2										
Ambient $PM_{2.5}$										
Winter	0.01	(−0.01,0.02)	−0.13	(−0.47,0.21)	481	−0.01	(−0.03,0.01)	0.51	(0.24,0.78)	299
Summer	—	—	—	—	—	0.00	(−0.02,0.01)	0.54	(0.21,0.86)	326

[a] Bold type indicates significance at the 0.05 level. — is not measured.

[b] Units for Personal $PM_{2.5}$ and SO_4^{2-} are in $\mu g/mg^3$. Units for Personal O_3, NO_2, and SO_2 are in ppb.

[c] Significant at the 0.0001 level.

Table 18. Mixed-Model Regression Results for Personal–Personal (Model 4) Associations[a]

Dependent Variable/ Independent Variable/ Season	Baltimore					Boston				
	Slope	Slope 95% CI	Intercept ($\mu g/m^3$)	Intercept 95% CI	*n*	Slope	Slope 95% CI	Intercept ($\mu g/m^3$)	Intercept 95% CI	*n*
Personal $PM_{2.5}$										
Personal O_3										
Winter	−0.06	(−0.28,0.16)	20.28	(16.86,23.70)	432	1.25	(−0.03,2.54)	15.29	(13.33,17.26)	239
Summer	0.18	(−0.09,0.45)	17.80	(15.49,20.11)	193	**0.72**[b]	**(0.42,1.01)**	17.39	(15.11,19.67)	329
Personal NO_2										
Winter	−0.02	(−0.12,0.08)	20.66	(16.18,25.14)	468	**0.42**	**(0.01,0.83)**	9.70	(2.69,16.72)	245
Summer	**0.20**	**(0.08,0.32)**	16.50	(14.46,18.54)	213	**0.21**	**(0.04,0.39)**	20.31	(17.16,23.46)	338
Personal SO_2										
Winter	−0.19	(−0.67,0.29)	20.40	(16.90,23.90)	466	−0.69	(−1.93,0.55)	17.18	(15.14,19.22)	246
Summer	—	—	—	—	—	0.16	(−0.53,0.86)	22.80	(21.08,24.52)	332
Personal SO_4^{2-}										
Personal O_3										
Winter	−0.02	(−0.04,0.01)	1.32	(1.18,1.46)	300	**0.07**	**(0.01,0.13)**	1.47	(1.32,1.62)	289
Summer	0.06	(−0.08,0.20)	5.46	(4.78,6.14)	135	**0.35**[b]	**(0.22,0.47)**	1.60	(0.98,2.22)	339
Personal NO_2										
Winter	**−0.01**	**(−0.02,0.00)**	1.47	(1.29,1.65)	306	0.01	(−0.01,0.03)	1.53	(1.28,1.78)	289
Summer	**0.08**	**(0.01,0.15)**	5.14	(4.36,5.92)	156	0.01	(−0.04,0.07)	3.36	(2.26,4.46)	339
Personal SO_2										
Winter	0.04	(−0.04,0.11)	1.31	(1.18,1.44)	305	0.16	(−0.02,0.33)	1.60	(1.45,1.76)	289
Summer	—	—	—	—	—	−0.02	(−0.26,0.22)	3.58	(3.07,4.09)	339

[a] Bold type indicates significance at the 0.05 level. — is not measured.

[b] Significant at the 0.0001 level.

Models examining the opposite cross-correlations (ie, ambient $PM_{2.5}$ concentrations as predictors of personal gas exposures) showed that ambient $PM_{2.5}$ in Boston was significantly associated with personal O_3 in summer and with personal NO_2 in winter (Table 17; $PM_{2.5}$–O_3 slope: 0.27; 95% CI: 0.14,0.39; $PM_{2.5}$–NO_2 slope: 0.21; 95% CI: 0.10,0.33) (Table 17). Slopes from the other models were all close to 0, indicating little association between the measurements.

Model 4. Personal–Personal Associations

In Baltimore, no personal gas exposures were significantly associated with corresponding personal $PM_{2.5}$ exposures with the exception of the summer association between personal $PM_{2.5}$ and NO_2 (Table 18). In contrast, significant personal–personal associations were found in Boston between $PM_{2.5}$ and O_3 during summer and between $PM_{2.5}$ and NO_2 during both seasons. Slightly weaker personal–personal associations were found for models using personal SO_4^{2-} instead of personal $PM_{2.5}$.

Results from the correlation analysis showed that, in Baltimore, only 5 of the 164 subject-specific personal–personal correlations were significant (Figure 4; 3 during the summer and 1 in the winter for $PM_{2.5}$–NO_2, and 1 during the summer for $PM_{2.5}$–O_3). An additional 3 significant correlations between personal $PM_{2.5}$ and personal NO_2 were negative, an inverse relation from that observed between the ambient concentrations of these two pollutants. Similarly, only 90 of the 162 subject-specific personal–personal correlations in Boston were significant (1 during the winter for $PM_{2.5}$–NO_2; and 6 during the summer and 2 in the winter for $PM_{2.5}$–O_3).

Sensitivity Analysis

Sensitivity analyses were conducted to examine the individual impact of a given observation on personal–ambient mixed-model slopes (Appendix N). These analyses show that the observed results were generally robust to the impact of individual observations. Removing single subjects from the models did not result in substantial

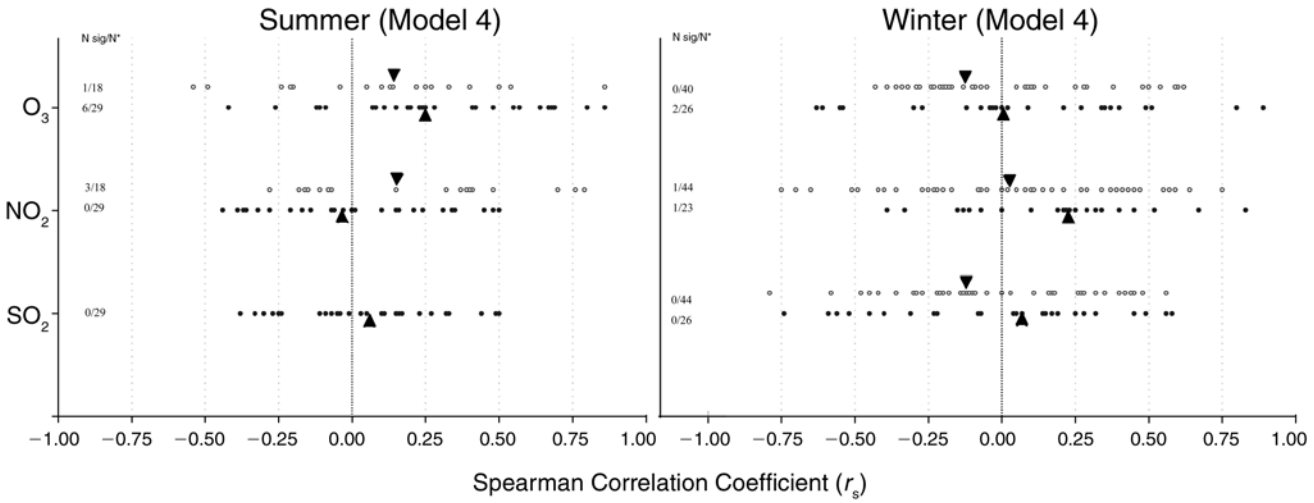

Figure 4. Subject-specific personal $PM_{2.5}$–personal gaseous pollutant Spearman correlation coefficients. ●, subjects in Boston. ○, subjects in Baltimore. ▼ or ▲, median correlation coefficient. *N sig* refers to the number of significant ($P < 0.05$) correlation coefficients. *N** refers to the total number of correlation coefficients.

changes in direction of the associations or in model *t* values. A notable exception was the winter personal–ambient associations for $PM_{2.5}$ in Boston. The analysis shows that results for subject 1 deviated substantially from other subjects during that season. Removing this subject from the analyses resulted in a upward shift of the observed slope from 0.33 (95% CI: 0.13,0.53) to 0.42 (95% CI: 0.38,0.57). The personal exposure data for this subject indicated that a single extreme $PM_{2.5}$ exposure occurred on 1 of the 12 days of sampling. Because no time–activity record was kept for ETS exposure or any other possible $PM_{2.5}$ source for this individual on this day, analyses were conducted including this observation. Concurrent personal SO_4^{2-} and gaseous exposures were within the normal range of distribution for this participant.

DISCUSSION

The current analyses were conducted to examine the consistency of findings between the Boston panel study and the original Baltimore panel study. A schematic diagram summarizing the results from both cities is presented for associations involving $PM_{2.5}$ and SO_4^{2-} (Figure 5).

SIMILARITIES BETWEEN BOSTON AND BALTIMORE

Subjects from Boston and Baltimore were generally exposed to very low levels of the gases, particularly O_3 during the winter and SO_2 during the summer, over an integrated 24 hours even when corresponding ambient levels of these pollutants were high. For epidemiologic studies using 24-hour integrated measurements, a gaseous pollutant is an unlikely confounder of ambient particles if actual exposure to that pollutant is negligible. For example, during winter sampling in Boston, a 6.1 ppb increase in ambient O_3, or 1 standard deviation, was associated with corresponding increases of 0.2 ppb in personal exposure to O_3. Therefore, inclusion of the ambient concentrations of these pollutants in multivariate models is questionable because changes in observed ambient concentrations may not be associated with exposures of biological significance.

Likewise, in both Baltimore and Boston, ambient $PM_{2.5}$ was correlated with many ambient gases in the summer and winter. Correlations among ambient pollutants are common throughout many parts of the United States and are largely due to the similar impact of meteorology on pollutant transport and removal (Schwartz et al 1994; Chen et al 1999; Wallace 2000). For epidemiology, identifying the relative importance of these pollutants for observed mortality and morbidity outcomes is made more difficult by the presence of collinearity among ambient pollutant concentrations.

Ambient concentrations of $PM_{2.5}$ were also significantly associated with personal exposures for subjects from both cities during both seasons, despite the presence of nonambient $PM_{2.5}$ sources. These findings are consistent with results from other recent, longitudinal, PM personal exposure assessments (Janssen et al 1997; Ebelt et al 2000; Williams et al 2000). Moreover, personal–ambient associations for SO_4^{2-} were stronger than those found for $PM_{2.5}$ in both cities. The personal–ambient SO_4^{2-} correlations were more striking, with significant summertime SO_4^{2-} correlations found for 41 of 43 subjects in both locations.

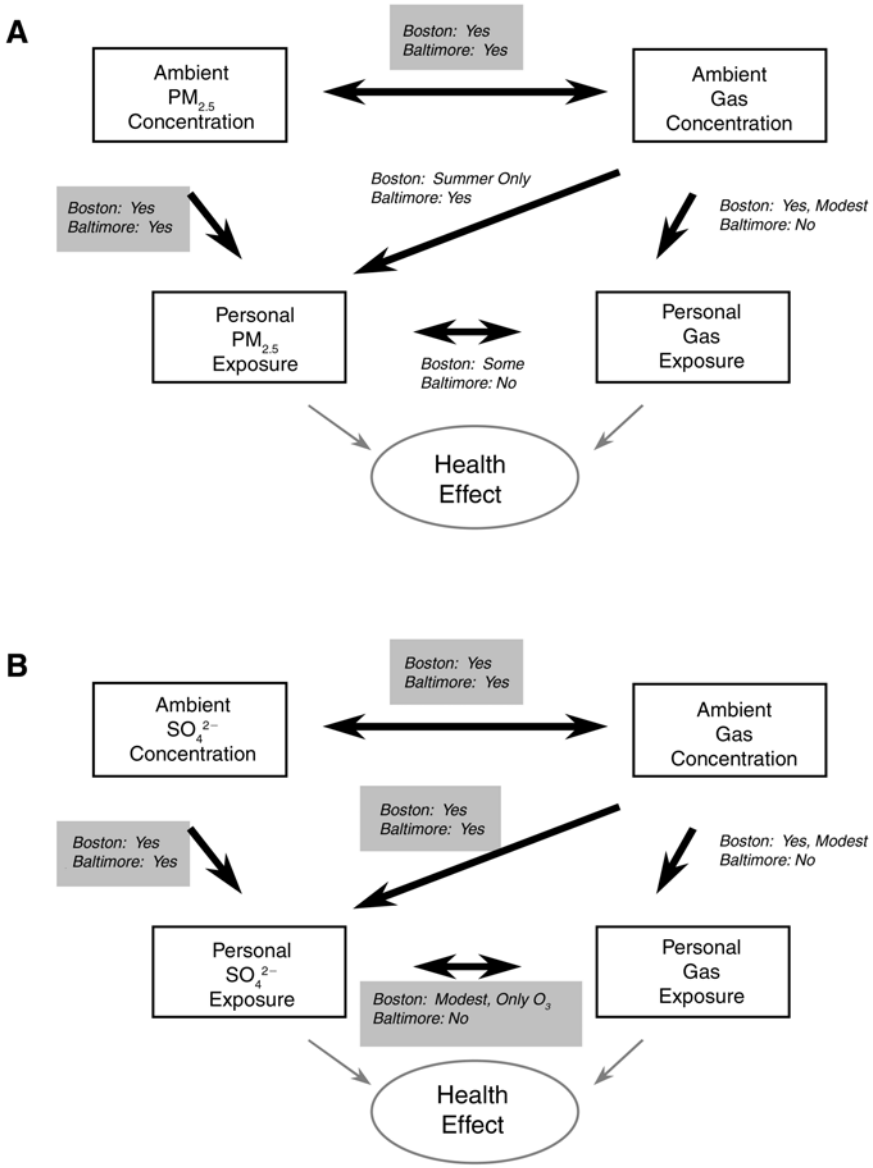

Figure 5. Summary of results involving (*A*) personal $PM_{2.5}$ and (*B*) SO_4^{2-} in the Boston and Baltimore studies. Consistent findings between the studies are shaded.

A key finding from Baltimore was the significant association between ambient gas concentrations and personal $PM_{2.5}$ and SO_4^{2-} exposures. This association was also shown to exist in Boston, particularly for SO_4^{2-}. In both cities, the associations between the ambient gas concentrations and personal SO_4^{2-} exposures were generally stronger than those observed for personal $PM_{2.5}$. This was expected because SO_4^{2-} is a component of $PM_{2.5}$ that is primarily ambient in origin. Conversely, total $PM_{2.5}$ has many nonambient sources that likely contributed to a weakening of the observed cross-pollutant associations.

DIFFERENCES BETWEEN BOSTON AND BALTIMORE

Despite their similarities, several key differences existed between the results from Boston and Baltimore. Most importantly were the significant findings in Boston for some personal–ambient gaseous associations, indicating that changes over time for some gaseous pollutants measured at central sites did, in fact, reflect corresponding changes in the personal exposures of subjects participating in this study. This was not the case in Baltimore, as ambient gas concentrations were not associated with their respective personal exposures. Although these personal–ambient

associations were significant for O_3 in both seasons and for NO_2 in summer, for 2 of the 3 models (summer O_3 and NO_2) the ambient gas measurements were more strongly associated with personal exposure to $PM_{2.5}$ than with their respective personal exposures.

For personal–ambient O_3 associations, the city-specific discrepancy in the results may be due to differences in ventilation. Previous studies have shown that the degree of indoor ventilation can influence personal–ambient associations for several pollutants, including O_3 and $PM_{2.5}$ (Liu et al 1995; Brauer and Brook 1997; Rojas-Bracho et al 2000a; Sarnat et al 2000). Although no quantitative measures of ventilation were taken in either Boston or Baltimore, average air exchange rates for the relatively older, leakier homes in Boston were probably higher than those in Baltimore, where many subjects lived in apartment complexes with central air conditioning. This assumption was also supported by the finding of higher mean personal–ambient SO_4^{2-} ratios in Boston than in Baltimore (Boston summer: 0.76, winter: 0.76; Baltimore summer: 0.54, winter: 0.45). A previous study by our group showed that SO_4^{2-} ratios can provide an estimate of ventilation, given the lack of major indoor SO_4^{2-} sources (Sarnat et al 2002).

Personal–personal associations were significant between $PM_{2.5}$ and O_3 during summer and between $PM_{2.5}$ and NO_2 during both seasons for Boston but not for Baltimore. Correlation between personal $PM_{2.5}$ and NO_2 exposures may be induced from common indoor sources such as gas stoves, which emit both $PM_{2.5}$ and NO_2 (Levy et al 1998). The likelihood that exposure to nonambient $PM_{2.5}$ and NO_2 sources drive the personal–personal associations for these pollutants is supported by the finding of weak, insignificant associations between personal NO_2 and personal SO_4^{2-} in Boston during the sampling periods.

Few published results have examined similar personal–personal associations. A study conducted on office workers in Paris found strong 48-hour correlations between ambient $PM_{2.5}$ and NO_2 ($r = 0.69$; $P < 0.001$) and weak 48-hour correlations between personal $PM_{2.5}$ and NO_2 in both the subjects' homes ($r = 0.06$; $P < 0.69$) and offices ($r = 0.05$; $P < 0.74$; Mosqueron et al 2002).

IMPLICATIONS

If gaseous pollutant exposures are responsible in part for the observed health effects, then ambient gas concentrations should be included along with $PM_{2.5}$ in multivariate epidemiologic models. This method will control for the possible confounding effect of these gases. The results from these studies indicate that associations between the ambient measurements of the gases may be more strongly associated with personal exposure to $PM_{2.5}$ and $PM_{2.5}$ of ambient origin than with their respective personal exposures. The Boston results show that the strength of the personal–ambient association for the gases may differ by location. These differences are likely due to geographic variability in housing characteristics, such as ventilation.

Given these findings, however, one should be cautious when interpreting results from previous time-series epidemiologic studies that have included gaseous and particulate pollutant concentrations in models. Numerous epidemiologic studies, for example, have shown significant associations between SO_2 concentrations measured at central monitoring sites and adverse health outcomes including hospital admissions and mortality (Schwartz et al 1994; Katsouyanni et al 1997; Samoli et al 2001). To date, however, no exposure studies have shown ambient SO_2 to be a suitable surrogate for personal SO_2 exposures. This issue has been addressed previously (Schwartz et al 1994; Katsouyanni et al 1997). Katsouyanni and colleagues speculate that findings showing ambient SO_2 associated with excess mortality in 12 European cities may be due to SO_2 serving as a "surrogate of other substances." The authors acknowledge further that SO_2 is highly reactive with a short indoor half-life. These two factors likely result in weak personal–ambient associations for this pollutant.

The results from Boston show that ambient summer O_3 and NO_2 concentrations are modestly associated with their corresponding personal exposures. These findings suggest that it is incorrect to assume that ambient gas measurements always serve as surrogates for PM exposures. Indeed, controlled exposure studies have shown associations between O_3, NO_2, and SO_2 exposures and adverse health outcomes (Sheppard et al 1981; Frampton et al 1991; Morrow et al 1992; Devlin et al 1997; Gong et al 1998). Likewise, environmental and occupational exposure assessment studies have reported higher mean personal O_3, NO_2, and SO_2 exposures than the levels reported in the current study (Brauer and Brook 1995; Geyh et al 2000; Lee et al 2002; Mosqueron et al 2002; Rotko et al 2002).

The Boston results show that, for some pollutants during specific seasons, ambient $PM_{2.5}$ was significantly associated with personal O_3 and NO_2 exposures. Although these cross-pollutant associations were not as strong as the association between ambient and personal $PM_{2.5}$, the findings suggest that ambient $PM_{2.5}$ may also serve as a surrogate for exposures to other pollutants.

LIMITATIONS

The analyses conducted for the data collected from Baltimore and Boston have numerous limitations. Several key issues, raised by colleagues and other reviewers of this analysis, warrant further discussion.

- *Differences in precision of PM and gas measurements.* A possible explanation for differences in the personal–ambient associations may be that the personal gas sampler measurements were less precise than measurements of $PM_{2.5}$ and thus resulted in greater random noise in associations involving the gases. Estimates of precision for the gas measurements taken during the Boston study indicate that they were as precise as, and in some cases more precise than, the $PM_{2.5}$ measurements. These estimates were derived from collocations under conditions with generally higher gaseous levels than those observed during actual subject sampling. Many of the personal gas exposures were below the estimated LODs and the exposure values probably contained more random noise. Despite this, we chose to use a consistent, quantitative modeling approach for all pollutants, regardless of their distributions. The fact that significant associations involving personal exposures to the gases were found, however, strongly indicates that sampler imprecision cannot completely explain the few significant personal–ambient associations for the gases.
- *Generalizability to other locations and cohorts.* Results from the current study only apply to a small, nonrandom selection of subjects living in the eastern United States. Caution should therefore be exercised in generalizing the results presented here and elsewhere to other locations and cohorts. The Boston results provide some indication that the relations between personal and ambient concentrations and among the pollutants may be influenced by a variety of factors (such as differences in ventilation or by the amount of time the subjects spent outdoors). Thus, the results reported here are likely to vary for locations where average ventilation rates differ or where subjects tend to spend more time outdoors. Additionally, associations among pollutants may vary in locations where pollutant concentrations are not the same as those observed in Boston and Baltimore. In particular, the presence of specific local sources of pollutants may impact each of the associations examined in the 4 models.
- *Limited dynamic range of gaseous pollutants.* Some of the weakness in the personal–personal gas associations may also be attributed not only to the extremely low concentrations of these pollutants but also to pollutants that varied little during the sampling periods. Limited dynamic range in the dependent variable (ie, personal exposures in personal–ambient and cross-pollutant models), however, should not impair the ability to determine significance because:

$$\mathrm{se}(\beta) = \sqrt{(x'x)^{-1}\sigma_y{}^2} \qquad (5)$$

where se(β) is the standard error of the slope of the mixed model, x is the independent variable in the model, and $\sigma_y{}^2$ is the variance in distribution of the dependent variable. Therefore, a low $\sigma_y{}^2$ would improve, and a low $x'x$ would impair, our ability to detect significance.

- *Inadequate exposure window.* Our objective was to examine the potential for confounding by gaseous pollutants within the context of the 24-hour time-series analyses commonly used to investigate associations between daily $PM_{2.5}$ concentrations and mortality and morbidity outcomes. These observed patterns of association may differ for longer or shorter exposure periods. In addition, more accurate, individual measures of exposure may exist and may need to be defined for use in future epidemiologic studies. Of particular interest will be the results of studies that examine pollutant associations for short peaks of exposure; these may provide more relevant exposure measures from a perspective of O_3 health effects (Lippmann 1989; Thurston et al 1997).
- *Spatial heterogeneity.* Among the unexamined factors that may explain some of the heterogeneity of the observed results by subject are the amount of spatial heterogeneity of the various pollutants and the proximity of the subjects' residences to the ambient monitoring sites. For example, a subject who lives close to a site measuring a pollutant that is spatially heterogeneous is more likely to have a stronger personal–ambient association than a subject living farther from the site. The ambient concentration levels used in the current analysis were derived by averaging values across sites, which is a common practice employed in epidemiologic investigations.

CONCLUSIONS

A major objective of this study was to determine the impact of ambient particles on personal exposures (infiltration factor) and to investigate the differences in the $PM_{2.5}$ and $SO_4{}^{2-}$ infiltration factors by city, season, and cohort. These results suggest that the relation between exposures to particles of ambient origin and the corresponding ambient levels can vary by season, location, and home characteristics. An initial hypothesis was that children would have higher personal exposures than seniors owing to their more active lifestyle, including more time spent outdoors. Notably, no cohort effect was found among the infiltration factors, which suggests that subjects from each of the 3 cohorts were exposed to a similar fraction of

ambient $PM_{2.5}$, given the same concentrations of ambient $PM_{2.5}$. Whereas summary statistics provided some indication of higher exposures for children, the model results showed no significant cohort differences in the relative contribution of ambient $PM_{2.5}$ on personal exposures. The lack of an observed cohort difference may be due to the fact that participants from all cohorts spent a comparatively small fraction of their time outdoors. Within a city and season, children tended to have similar levels of exposure to ambient $PM_{2.5}$ but higher nonambient contributions to their personal exposure. This was not the case for children in Baltimore during summer, who had a smaller intercept than that for the seniors. One explanation for this finding is that Baltimore children spent significantly less time outdoors (5.5%) than the Boston children during summer (11.3%). In the summer sampling session in Baltimore, children's exposures were measured during a single 10-day period in mid August. Many of these children were recruited from a science education program, which may explain their limited time spent outdoors.

Another explanation for the lack of a difference by cohort may be that cohort is not a sufficient proxy for differences in time–activity patterns or exposure-related activities. Rather, more specific information on activities and, in particular, indoor sources may better explain intersubject variability in the influence of ambient $PM_{2.5}$ on corresponding personal exposures. Finally, the within-cohort variability in personal exposures was large compared to the between-cohort variability in personal exposures. These differences in variability may have obscured cohort differences in the mixed-model regression results.

The current analysis of SO_4^{2-} infiltration appears to indicate that subjects in Boston are exposed to a greater fraction of ambient SO_4^{2-} than those in Baltimore, despite the higher absolute levels of SO_4^{2-} in Baltimore. The higher ambient SO_4^{2-} levels in Baltimore are due to the warmer climate and the city's proximity to power-plant SO_2 emissions (Suh et al 1997). Summer ambient SO_4^{2-} levels were higher than winter levels in both cities. Other studies have shown that higher summer ambient SO_4^{2-} levels are related to increased photochemical activity in the summer (Brauer et al 1989; Suh et al 1994; Burton et al 1996). On the basis of our results, differences in personal SO_4^{2-} exposures by city, season, and cohort are likely related to differences in ambient SO_4^{2-}concentrations, time–activity patterns, and housing characteristics between the cohorts in the two cities during different seasons (Suh et al 1994; Sarnat et al 2000).

The $PM_{2.5}$ infiltration factors, particularly in winter, were probably influenced by indoor $PM_{2.5}$ sources. Previous personal exposure assessment and indoor particle characterization studies have shown that SO_4^{2-} is a good surrogate for ambient particles (Suh et al 1994; Ebelt et al 2000; Sarnat et al 2000) because ambient SO_4^{2-} concentrations present little spatial variability (Suh et al 1997), lack indoor sources, and exhibit high penetration efficiencies (Brauer et al 1989). Thus, the SO_4^{2-} infiltration factors are likely to more accurately reflect the contribution of ambient PM to personal exposures. This hypothesis is further supported by the high SO_4^{2-} infiltration factor for Boston during winter (0.60), a period when the cohorts spent significantly less time outside than during summer and more than 90% of their time indoors. This information also supports the notion that building conditions, particularly home conditions, may play the greatest role in determining exposures to PM of ambient origin.

Despite regional and seasonal variations in infiltration factors, however, ambient $PM_{2.5}$ and SO_4^{2-} were shown to be strong predictors of personal exposures. On the basis of these results, therefore, it is conceivable that a single nationwide PM standard may provide a different degree of protection for different populations depending on season, regional home characteristics, and indoor ventilation patterns.

Finally, the results from Boston provide an indication from another location in the eastern United States that ambient gas concentrations may be more strongly associated with exposures to $PM_{2.5}$ than with their respective personal exposures. Additional results and analyses from other locations and future improvements in personal sampler design to characterize exposures to pollutants at shorter time intervals will provide insight into questions surrounding use of ambient measurements.

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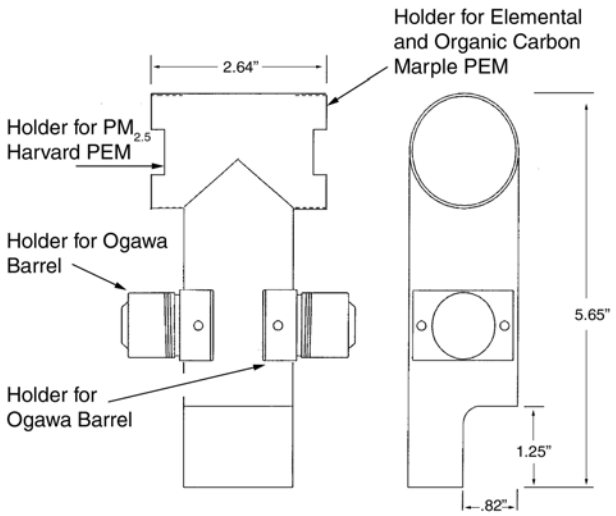
APPENDIX A. Sampling Sessions

City / Season / Session	Start Date	End Date	Consecutive Days of Sampling	Cohort
Baltimore				
Summer				
1	6/28/98	7/10/98	12	4 Seniors
2	7/13/98	7/24/98	12	6 Seniors
3	7/26/98	8/7/98	12	5 Seniors
4	8/12/98	8/22/98	8	10 Children
Winter				
5	2/1/99	2/12/99	12	5 Seniors 5 COPD 4 Children
6	2/15/99	2/26/99	12	4 Seniors 5 COPD 6 Children
7	3/1/99	3/16/99	12	6 Seniors 5 COPD 5 Children
Boston				
Summer				
8	6/13/99	6/24/99	12	10 Seniors
9	6/28/99	7/10/99	12	5 Seniors 5 Children
10	7/12/99	7/23/99	12	10 Children
Winter				
11	2/1/00	2/13/00	12	6 Seniors 4 Children
12	2/15/00	2/26/00	12	4 Seniors 5 Children
13	2/29/00	3/12/00	12	5 Seniors 6 Children

APPENDIX B. Multipollutant Sampler Used to Collect Personal Exposure Samples



APPENDIX C. Ambient Sampling Specifications

Baltimore		Boston	
Season / Pollutant	Ambient Site	Season / Pollutant	Ambient Site
	Summer		
Integrated $PM_{2.5}$	Old Town Clifton	Integrated $PM_{2.5}$, EC, SO_4^{2-} Continuous $PM_{2.5}$	Harvard School of Public Health
Continuous $PM_{2.5}$	Old Town	Continuous O_3	Dudley Square Chelsea
Continuous O_3	Essex Living Classroom	Continuous NO_2	Kenmore Square East Boston South Boston Chelsea
Continuous NO_2	Old Town Essex Living Classroom	Continuous SO_2	Kenmore Square Dorchester East Boston South Boston Chelsea
	Winter		
Integrated $PM_{2.5}$ Continuous $PM_{2.5}$	Old Town	Integrated $PM_{2.5}$, EC, SO_4^{2-} Continuous $PM_{2.5}$	Harvard School of Public Health
Continuous O_3	Essex	Continuous O_3	Dudley Square
Continuous NO_2	Old Town Essex	Continuous NO_2	Kenmore Square East Boston South Boston
Continuous SO_2	Riviera Beach	Continuous SO_2	Kenmore Square Dorchester East Boston South Boston

APPENDIX D. Locations of Participants' Homes and Ambient Monitors

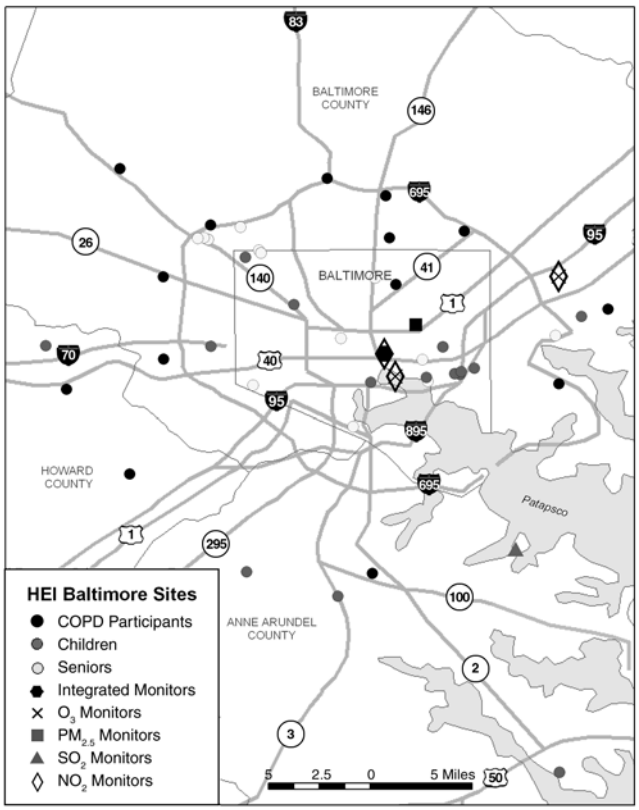


Figure D.1. Baltimore, Maryland. Data are from US Census Bureau and Harvard School of Public Health.

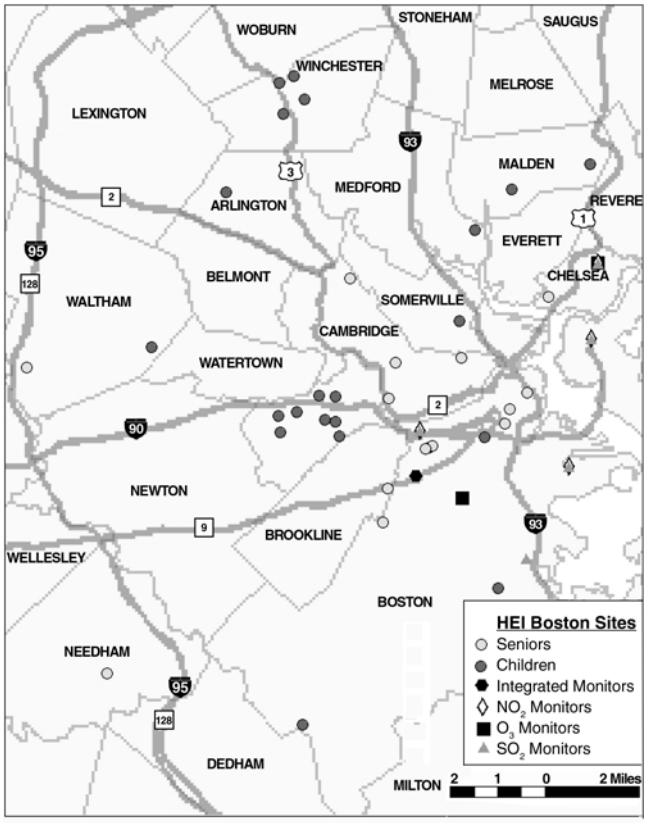
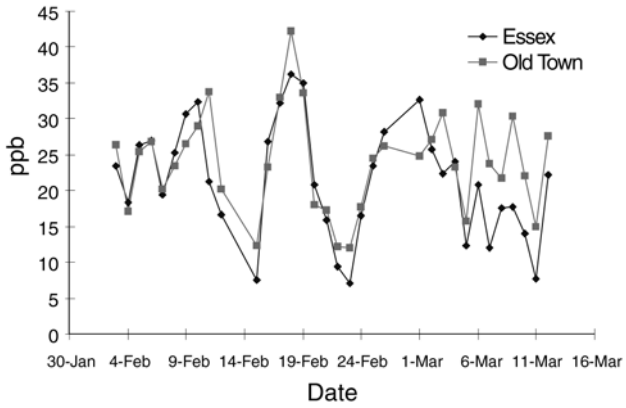


Figure D.2. Locations of participants' homes and ambient monitors in Boston, Massachusetts. Data are from MassGIS (Office of Geographic and Environment Information, Boston MA) and Harvard School of Public Health.

APPENDIX E. Comparison of Ambient NO_2 Concentrations from Two Baltimore Locations in Winter



APPENDIX F. Mixed-Model Regression Results for Personal–Ambient (Model 2) Associations Without Nested Group[a]

Dependent Variable/ Independent Variable/ Season	Baltimore					Boston				
	Slope	Slope 95% CI	Intercept[b]	Intercept 95% CI	*n*	Slope	Slope 95% CI	Intercept[b]	Intercept 95% CI	*n*
Personal $PM_{2.5}$										
Ambient $PM_{2.5}$										
Winter	**0.26**[c]	**(0.18,0.35)**	13.27	(9.4,17.13)	481	**0.35**	**(0.15,0.55)**	12.87	(8.82,16.92)	253
Summer	**0.52**[c]	**(0.37,0.66)**	9.22	(5.48,12.95)	224	**0.78**[c]	**(0.66,0.9)**	12.59	(9.47,15.7)	330
Personal O_3										
Ambient O_3										
Winter	0.00	(−0.02,0.02)	0.45	(0.04,0.87)	449	**0.05**	**(0.02,0.08)**	0.42	(−0.1,0.95)	288
Summer	0.04	(−0.02,0.10)	1.84	(−0.55,4.23)	196	**0.27**[c]	**(0.18,0.37)**	−1.00	(−2.74,0.75)	332
Personal NO_2										
Ambient NO_2										
Winter	−0.05	(−0.22,0.12)	18.16	(13.27,23.05)	485	0.02	(−0.1,0.15)	16.62	(10.76,22.48)	298
Summer	0.04	(−0.23,0.31)	9.52	(3.46,15.58)	217	**0.19**	**(0.08,0.30)**	15.22	(11.38,19.05)	341
Personal SO_2										
Ambient SO_2										
Winter	**−0.05**	**(−0.09,−0.02)**	0.55	(0.11,0.98)	487	0.00	(−0.02,0.02)	0.66	(0.12,1.2)	299
Summer	—	—	—	—	—	−0.01	(−0.11,0.10)	0.46	(−0.02,0.95)	335
Personal SO_4^{2-}										
Ambient SO_4^{2-}										
Winter	**0.42**[c]	**(0.36,0.48)**	0.15	(−0.07,0.37)	301	**0.57**[c]	**(0.50,0.64)**	0.37	(0,0.73)	289
Summer	**0.38**[c]	**(0.30,0.46)**	1.7	(1.01,2.4)	242	**0.74**[c]	**(0.67,0.80)**	0.38	(0,0.76)	291

[a] Bold type indicates significance at the 0.05 level. — is not measured.

[b] Units for $PM_{2.5}$ and SO_4^{2-} are in μg/mg^3. Units for O_3, NO_2, and SO_2 are in ppb.

[c] Significant at the 0.0001 level.

APPENDIX G. Mixed-Model Regression Results for Cross-Pollutant (Model 3) Associations Without Nested Group[a]

Dependent Variable/ Independent Variable/ Season	Baltimore					Boston				
	Slope	Slope 95% CI	Intercept[b]	Intercept 95% CI	*n*	Slope	Slope 95% CI	Intercept[b]	Intercept 95% CI	*n*
Personal $PM_{2.5}$										
Ambient O_3										
Winter	**−0.29**[c]	**(−0.40,−0.18)**	23.86	(19.15,28.56)	487	0.01	(−0.23,0.26)	16.61	(12.29,20.93)	253
Summer	**0.33**	**(0.16,0.49)**	9.57	(2.33,16.81)	224	**0.47**[c]	**(0.38,0.57)**	12.52	(9.9,15.14)	339
Ambient NO_2										
Winter	**0.24**[c]	**(0.13,0.35)**	13.16	(8.88,17.44)	487	0.10	(−0.1,0.29)	14.17	(7.22,21.11)	253
Summer	**0.43**	**(0.13,0.72)**	12.37	(6.47,18.27)	224	**0.41**[c]	**(0.29,0.54)**	14.40	(11.31,17.48)	339
Ambient SO_2										
Winter	**−0.24**[c]	**(−0.36,−0.12)**	20.75	(16.26,25.24)	487	−0.13	(−0.49,0.23)	17.86	(13.77,21.95)	253
Summer	—	—	—	—	—	**1.72**[c]	**(1.03,2.4)**	18.29	(14.66,21.92)	339
Ambient CO										
Winter	**3.99**[c]	**(2.37,5.60)**	15.00	(10.41,19.59)	487	6.70	(−1.22,14.63)	11.58	(3.85,19.3)	253
Summer	5.92	(−4.59,16.43)	18.21	(13.4,23.02)	224	**30.07**[c]	**(21.42,38.71)**	4.47	(−0.72,9.66)	339
Personal SO_4^{2-}										
Ambient O_3										
Winter	**−0.04**[c]	**(−0.04,−0.03)**	2.42	(2.10,2.74)	319	0.00	(−0.02,0.01)	2.14	(1.53,2.74)	273
Summer	**0.25**[c]	**(0.19,0.31)**	−3.68	(−5.95,−1.42)	160	**0.24**	**(0.19,0.28)**	−1.80	(−2.77,−0.83)	329
Ambient NO_2										
Winter	**0.03**	**(0.00,0.06)**	1.07	(0.47,1.67)	319	**0.07**	**(0.05,0.08)**	0.26	(−0.14,0.66)	280
Summer	0.10	(−0.05,0.26)	3.51	(−0.22,7.24)	160	**0.12**	**(0.07,0.16)**	1.35	(0.45,2.25)	338
Ambient SO_2										
Winter	**−0.04**	**(−0.06,−0.02)**	2.10	(1.88,2.33)	319	**0.05**	**(0.01,0.09)**	1.73	(1.35,2.11)	282
Summer	—	—	—	—	—	**0.54**	**(0.31,0.77)**	2.23	(1.36,3.09)	332
Ambient CO										
Winter	0.16	(−0.12,0.44)	1.61	(1.27,1.95)	319	**2.56**	**(1.87,3.24)**	0.09	(−0.47,0.64)	289
Summer	−4.25	(−8.91,0.40)	7.68	(5.14,10.21)	160	**12.91**	**(8.34,17.49)**	−4.45	(−7.31,−1.58)	339
Personal O_3										
Ambient $PM_{2.5}$										
Winter	0.00	(−0.01,0.02)	0.38	(−0.1,0.86)	443	0.01	(−0.02,0.03)	1.19	(0.67,1.71)	288
Summer	0.03	(−0.03,0.09)	2.65	(0.54,4.75)	196	**0.27**[c]	**(0.14,0.39)**	1.87	(0.19,3.54)	323
Personal NO_2										
Ambient $PM_{2.5}$										
Winter	−0.05	(−0.18,0.07)	18.14	(14.47,21.81)	479	**0.21**	**(0.09,0.32)**	14.86	(11.86,17.86)	297
Summer	0.02	(−0.08,0.12)	9.83	(6.55,13.12)	217	−0.02	(−0.09,0.05)	20.50	(16.97,24.03)	332
Personal SO_2										
Ambient $PM_{2.5}$										
Winter	0.01	(−0.01,0.02)	−0.09	(−0.44,0.26)	481	0.00	(−0.03,0.02)	0.68	(0.21,1.16)	299
Summer	—	—	—	—	—	0.00	(−0.02,0.01)	0.53	(0.18,0.87)	326

[a] Bold type indicates significance at the 0.05 level. — is not measured.

[b] Units for Personal $PM_{2.5}$ and SO_4^{2-} are in $\mu g/mg^3$. Units for Personal O_3, NO_2, and SO_2 are in ppb.

[c] Significant at the 0.0001 level.

APPENDIX H. Mixed-Model Regression Results for Personal–Personal (Model 4) Associations Without Nested Group[a]

Dependent Variable/ Independent Variable/ Season	Baltimore					Boston				
	Slope	Slope 95% CI	Intercept ($\mu g/m^3$)	Intercept 95% CI	*n*	Slope	Slope 95% CI	Intercept ($\mu g/m^3$)	Intercept 95% CI	*n*
Personal $PM_{2.5}$										
Personal O_3										
Winter	−0.06	(−0.27,0.16)	18.50	(14.53,22.48)	432	**1.31**	**(0.02,2.60)**	15.43	(12.43,18.44)	239
Summer	0.21	(−0.07,0.49)	19.86	(17.06,22.66)	193	**0.73**[b]	**(0.42,1.04)**	20.17	(17.73,22.61)	329
Personal NO_2										
Winter	−0.02	(−0.13,0.08)	19.02	(14.01,24.04)	468	**0.45**	**(0.03,0.86)**	9.24	(2.4,16.08)	245
Summer	**0.18**	**(0.06,0.30)**	18.73	(16.19,21.28)	213	**0.23**	**(0.04,0.43)**	20.00	(16.14,23.85)	338
Personal SO_2										
Winter	−0.18	(−0.65,0.29)	18.66	(14.58,22.74)	466	−0.41	(−1.72,0.90)	17.22	(14.3,20.14)	246
Summer	—	—	—	—	—	0.14	(−0.56,0.84)	24.76	(22.13,27.39)	332
Personal SO_4^{2-}										
Personal O_3										
Winter	−0.01	(−0.04,0.01)	1.75	(1.50,1.99)	300	**0.09**	**(0.02,0.16)**	1.94	(1.61,2.28)	289
Summer	0.06	(−0.08,0.21)	5.14	(4.26,6.02)	135	**0.35**	**(0.22,0.48)**	2.13	(1.57,2.69)	339
Personal NO_2										
Winter	**−0.01**	**(−0.01,0.00)**	1.9	(1.60,2.19)	306	**0.02**	**(0.00,0.05)**	1.73	(1.34,2.12)	289
Summer	**0.08**	**(0.01,0.15)**	4.99	(4.09,5.88)	156	**0.05**	**(0.00,0.10)**	3.34	(2.11,4.56)	339
Personal SO_2										
Winter	0.03	(−0.05,0.11)	1.75	(1.51,1.99)	305	0.19	(−0.01,0.38)	1.99	(1.74,2.24)	289
Summer	—	—	—	—	—	−0.02	(−0.28,0.23)	4.29	(3.65,4.93)	339

[a] Bold type indicates significance at the 0.05 level. — is not measured.

[b] Significant at the 0.0001 level.

APPENDIX I. Pilot Analysis—Joint Pollutant Distributions

The analyses conducted for the current report focus on bivariate relations between pairs of concentrations and/or personal exposures. Relations described by alternative distributions might provide a more accurate model for understanding interactions and associations among the pollutants, however. Therefore, an additional pilot analysis was conducted using a subset of the Baltimore data exclusively. For this analysis, the full joint distribution of both personal and ambient measures of the multiple pollutants were considered. The implications of this joint relation on a simulated health effects analysis were also examined.

A review of the implications of pollutant measurement error in health effects analyses provides a suitable introduction to this issue. Zeger and colleagues (2000) developed a statistical framework for studying the effects of such measurement error on health effect estimates. One key finding of this paper is that previous discussions of measurement error in the exposure measurement frequently have ignored differences between classical and Berkson measurement error. The investigators showed that in the case of longitudinal studies of air pollution and health, such as those cited in this report, most of the difference between personal exposure and ambient concentrations was Berkson measurement error, which does not bias the estimate of the health effect but does reduce the power to detect significance. The remaining measurement error, which is classical, results in attenuation of the health effect estimates toward the null hypothesis of no effect. Although the coefficient estimates from models containing more than one exposure could in principle be biased upward or downward, Zeger and colleagues showed that upward bias occurs mainly in rare settings in which either ratio of the amounts of measurement error in PM and other pollutants is high and the correlations among pollutants is extreme. Hence, the authors concluded that observed PM coefficients were not likely biased away from 0 by measurement error. These investigators also noted that quantifying the effects of exposure measurement error in PM epidemiology is difficult because of the lack of data at that time on both the magnitude of these errors and how components of error covary across pollutants. As a result, they called for studies that collect daily measurements of personal exposure and ambient levels for multiple pollutants for each person.

To address these issues, Baltimore $PM_{2.5}$, O_3, and NO_2 data were used to obtain realistic estimates of the effects of measurement error in PM epidemiology. A simulation was performed to generate the distribution of health effect estimates for pollutants when the measurement errors and cross-pollutant correlations matched those observed in the Baltimore data for a variety of assumed true health effect associations with exposures to each of the pollutants. The resulting framework builds upon that of Zeger and colleagues (2000) by allowing the covariances among personal and ambient exposures to vary across subjects and by explicitly dealing with the measurement error in gaseous pollutants.

Of particular concern is the possibility that a true association with exposure to pollutant *A* may result in an observed association with pollutant *B* due to correlations among the ambient concentrations and exposures of these two pollutants. The analytic approach used for the simulation can be described in five steps:

1. Subject-specific covariance matrices, by season, were calculated between the personal exposures and corresponding ambient concentrations for all measured pollutants. Use of the covariance matrix allowed us to capture the correlation between ambient concentrations of each pollutant and personal exposure to the same pollutant as well as the cross-correlations among the ambient pollutants and exposures. On the basis of the range of these subject-specific data, a joint distribution of covariance matrices by season was created.
2. A covariance matrix was randomly selected from a given subject. This matrix generated a time series consisting of 2000 days of ambient air pollution concentrations and personal exposures to $PM_{2.5}$, O_3, and NO_2. Exposures were estimated from a multivariate normal distribution with covariance matrix matching that observed in the personal exposure study.
3. A true association, $\beta_p = 0.05$, was assumed to exist between personal exposures to one of the pollutants and an unspecified health event (either a morbid or mortal outcome). Exposures to the other pollutants were also assumed not to be associated with any health risks. Assuming further that events are Poisson distributed about their true risk, a time series of adverse events occurring on each of the 2000 days was generated, with a daily mean equal to:

$$0.1 \times e^{0.05 \times X_t} \quad \text{(D1)}$$

   where X_t is the personal exposure to the assumed causal pollutant on day *t*.
4. The events were regressed against each of the simulated ambient concentrations, producing an estimated coefficient, β_{pa}, for each of the pollutants. This coefficient was compared to β_p (ie, 0.05) for the chosen pollutant and to 0 for the other pollutants. To capture the range of possible covariances observed in Baltimore, this entire process was repeated 210 times (sampling with replacement). The mean was used as an indication of what

might be expected for each ambient pollutant given the covariance between ambient concentrations and personal exposures and its distribution in Baltimore.

The mean of these 210 sets of coefficients was interpreted to be comparable to what one might expect to find using Poisson regression analysis with ambient pollution as an exposure metric in a city with 22 events per day. Multiple simulations were conducted to allow for each pollutant to have an assumed true association with a health risk.

5. Steps 1 through 4 were repeated 200 times. The mean and empirical 95% confidence interval of the coefficients for the 200 replications were used to simulate the distribution of results that might be seen from a Poisson regression of events versus each air pollutant based on the assumption of a true association with one of the exposures. This simulation was repeated assuming a true association with each exposure in turn.

For the pollutant that was assumed to be truly associated with the adverse health risk, the observed difference between β_{pa} and β_p provided an estimate of the impact of measurement error that arises from using ambient concentrations as opposed to personal exposures as surrogates of exposures to ambient pollutants. For the other pollutants assumed not to be associated with the health outcome, the results provided an estimate of the deviation from the truth (ie, 0) that might be expected, given measurement error and the correlations among the variables. One important feature of the simulations was that covariances from the winter and summer samples were separately calculated. Because correlations between ambient $PM_{2.5}$ and O_3 were shown to be positive during summer but negative during winter, this season-specific approach seemed more reasonable than calculating 1 covariance for each subject averaging over the 2 seasons.

Results from the single-pollutant simulations showed that $PM_{2.5}$ was the only pollutant for which a true association with its personal exposures resulted in a significant association with ambient concentrations ($\beta = 0.0143$, 95% CI: 0.0097,0.0177) (Table I). Because the true coefficient was assumed to be 0.05, the observed coefficient using ambient $PM_{2.5}$ concentrations represents an attenuation of roughly 70%. The attenuation was slightly less (about 60%) than the true association when personal exposures to $PM_{2.5}$ of ambient origin were assumed to be the true exposures of health risk association ($\beta = 0.018$, 95% CI: 017,0.20).

The simulations also showed that, for O_3 and NO_2, true associations with exposure would result in no significant associations with any of their respective ambient concentrations. However, a true association with $PM_{2.5}$ would result in a significant, observed association with ambient NO_2 ($\beta = 0.0115$, 95% CI: 0.0056,0.0185). The pattern of results in these simulations is quite informative and suggests that if a true health risk associated with personal $PM_{2.5}$ exposures existed, one could expect to see a corresponding significant association using ambient $PM_{2.5}$ concentrations, albeit attenuated relative to the real association.

The O_3 health risk estimate should be interpreted carefully given that the simulations indicate that a true association with personal O_3 would not be expected to result in an observed association with any ambient pollutant concentration. We do not interpret the results of our simulation as indicating no adverse effects associated with either O_3 or the other gaseous pollutants. The results do indicate that ambient O_3 concentrations may not be adequate surrogates for corresponding personal O_3 exposures to allow the question to be investigated using ambient monitors. This may not be true in special circumstances, such as studies of summer campers (Brauer and Brook 1997), in which ambient O_3 concentrations were shown to be good surrogates for exposure to O_3.

The results observed for models using NO_2 also require caution in interpretation. A true association with exposure to NO_2 would not be expected to result in an observed association using ambient NO_2 concentrations, whereas a true association with $PM_{2.5}$ exposure would be expected to result in an observed association with ambient NO_2. For cities with exposure patterns similar to those in Baltimore, these results again suggest that associations with ambient NO_2 are much more likely to represent the results of a true association with exposure to $PM_{2.5}$ than with personal exposure to NO_2. This conclusion makes the use of two-pollutant models highly suspect in this case as well.

In summary, the simulation-based approach provided an initial investigation into the likely impacts of pollutant measurement error on epidemiologic findings. The approach is useful because it considers the full distribution of the multiple personal and ambient measurements rather than simple bivariate associations. The strategy and methods described here can be improved in several ways and will be the subject of future research. First, these results will be extended to other size fractions, such as coarse particles, and other components of PM, such as $PM_{2.5}$ of ambient origin and SO_4^{2-} particles. Second, the current analyses used the simple approach of simulating ambient and personal exposures from a multivariate normal distribution having covariance matrices matching those observed in personal exposure studies. In the future, it would be of interest to generalize this approach to allow for more flexible and realistic multivariate distributions of exposure. Even in light of these limitations, the approach used in the pilot analysis yielded insight into the impact of such associations on estimated health effects of interest.

Table I. Distribution of Coefficients Observed in Regressions Against Ambient Pollutants Based on Covariance of Personal and Ambient Exposures in Baltimore[a]

Model Scenario / Coefficient	Ambient $PM_{2.5}$	Ambient O_3	Ambient NO_2
True association with personal exposure to total $PM_{2.5}$			
Median β	0.0143	−0.0016	0.0115
95% CI	(0.0097,0.0177)	(−0.0056,0.0025)	(0.0056,0.0185)
True association with personal exposure to O_3			
Median β	0.0014	0.0010	0.0009
95% CI	(−0.0005,0.0035)	(−0.0007,0.0024)	(−0.0011,0.0034)
True association with personal exposure to NO_2			
Median β	0.0015	0.0009	0.0010
95% CI	(−0.0006,0.0036)	(−0.0008,0.0025)	(−0.0014,0.0036)

[a] All simulations assume a true coefficient of 0.05.

APPENDIX J. Samples Invalidated by Atypical Exposure to ETS[a]

City / Season / Cohort	Invalidated Samples (*n*)	Total Samples Scheduled (*n*)	Invalidated Samples (%)
Baltimore			
Winter			
Seniors	5	180	2.8
Children	0	180	0.0
COPD	2	180	1.1
Summer			
Seniors	8	180	4.4
Children	7	80	8.8
Boston			
Winter			
Seniors	10	180	5.6
Children	13	180	7.2
Summer			
Seniors	12	180	6.7
Children	0	180	0.0
Total	57	1520	3.8

[a] Exposure to high ETS concentration or prolonged exposure to ETS.

APPENDIX K. Number of Homes with Gas and Electric Stoves by City, Season, and Cohort

City / Season / Cohort	Gas Stoves		Electric Stoves	
	n	%	*n*	%
Baltimore				
Winter				
Seniors	8	53	7	47
Children[a]	8	57	6	43
COPD	9	60	6	40
Summer				
Seniors	4	27	11	73
Children	5	50	5	50
Boston				
Winter				
Seniors	2	13	13	87
Children[a]	10	71	4	29
Summer				
Seniors	2	13	13	87
Children	13	87	2	13

[a] Data for 1 child are missing.

APPENDIX L. Personal Exposures by Subject, City, and Season[a]

Session / Cohort / Subject	$PM_{2.5}$ (μg/m^3)					SO_4^{2-} (μg/m^3)					EC (μg/m^3)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Winter															
Session 5															
Seniors															
1	12	9.5	10.3	3.5	17.9	12	1.5	1.5	0.6	2.5	—	—	—	—	—
2	11	15.4	17.0	2.6	21.0	11	2.6	2.3	0.8	3.6	—	—	—	—	—
3	6	20.0	27.9	24.0	76.6	6	3.1	3.6	1.4	6.2	—	—	—	—	—
4	9	14.6	15.2	2.9	19.6	9	2.6	2.2	0.7	2.8	—	—	—	—	—
5	11	15.3	16.5	5.5	30.6	11	2.7	2.6	0.9	4.0	—	—	—	—	—
Children															
1	8	29.5	31.3	6.4	43.4	—	—	—	—	—	10	4.4	4.5	1.5	7.3
2	9	13.7	14.2	4.5	23.1	—	—	—	—	—	11	2.6	3.0	1.5	6.3
3	11	23.3	30.1	18.3	78.0	—	—	—	—	—	11	2.5	3.2	1.3	5.2
4	10	27.3	31.6	22.2	91.2	—	—	—	—	—	9	3.3	3.6	2.0	8.2
COPD															
1	7	11.2	12.4	6.6	26.8	6	1.6	1.4	0.5	1.8	8	1.7	1.9	0.8	3.5
2	10	28.5	31.7	8.0	46.1	10	1.9	1.9	0.6	2.8	8	4.7	4.5	1.0	5.9
3	12	5.6	5.7	3.1	13.3	10	0.6	0.7	0.2	1.0	12	1.4	1.4	0.5	2.3
4	12	42.0	45.4	14.4	71.4	12	2.0	2.0	0.6	3.0	12	2.1	1.9	0.7	2.9
5	10	16.4	19.8	8.1	37.1	11	2.0	1.9	0.6	2.6	10	3.0	2.9	1.0	4.9
Ambient	12	23.4	21.7	10.6	36.5	—	—	—	—	—	—	—	—	—	—
Session 6															
Seniors															
6	11	11.1	12.4	4.3	19.8	10	2.1	2.1	0.9	3.8	—	—	—	—	—
7	12	14.2	21.2	14.6	57.3	12	2.4	2.4	1.2	4.2	—	—	—	—	—
8	11	42.6	46.5	24.3	88.6	11	3.1	3.1	1.4	5.2	—	—	—	—	—
9	11	25.0	26.9	13.5	52.6	11	2.3	2.2	1.1	4.1	—	—	—	—	—
Children															
5	11	23.2	33.6	21.6	83.6	—	—	—	—	—	12	3.6	4.1	2.5	11.1
6	11	23.7	34.2	22.0	83.7	—	—	—	—	—	11	3.0	4.0	2.9	11.6
7	12	12.7	13.4	5.4	22.8	—	—	—	—	—	12	1.6	2.1	1.5	5.5
8	12	16.9	18.3	7.7	33.3	—	—	—	—	—	11	1.6	1.8	1.0	3.7
9	10	13.2	13.3	4.6	21.4	—	—	—	—	—	11	1.8	2.0	1.1	4.4
10	12	13.4	18.0	9.1	34.6	—	—	—	—	—	12	1.9	2.1	1.4	4.8
COPD															
6	10	20.9	22.2	6.5	32.8	10	1.8	2.0	1.0	3.2	10	2.9	3.3	1.3	5.5
7	12	9.9	10.6	5.3	23.1	12	1.8	2.1	1.0	3.6	12	1.5	1.7	0.8	3.6
8	11	16.7	15.2	4.5	24.1	11	1.0	1.1	0.5	2.0	11	0.8	0.9	0.5	2.0
9	11	13.9	15.2	7.6	33.6	11	1.8	2.2	1.2	4.3	11	1.7	1.9	0.9	3.3
10	12	13.9	19.2	12.0	41.0	11	1.7	2.1	0.9	3.5	12	2.0	2.5	1.4	6.1
Ambient	11	20.7	25.1	16.0	52.6	—	—	—	—	—	—	—	—	—	—

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	$PM_{2.5}$ (μg/m^3)					SO_4^{2-} (μg/m^3)					EC (μg/m^3)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Winter (*continued*)															
Session 7															
Seniors															
10	12	4.7	5.2	2.8	11.1	12	0.8	0.9	0.4	1.6	—	—	—	—	—
11	10	3.5	4.9	3.7	14.2	10	1.1	1.2	0.6	2.3	—	—	—	—	—
12	10	5.1	5.9	3.7	13.7	10	1.0	1.2	0.7	2.2	—	—	—	—	—
13	10	6.3	6.5	2.9	11.7	10	1.9	1.5	0.6	2.2	—	—	—	—	—
14	12	4.8	5.2	2.2	9.8	12	1.7	1.6	0.6	2.4	—	—	—	—	—
15	12	8.9	10.5	5.1	24.5	12	1.5	1.3	0.5	2.0	—	—	—	—	—
Children															
11	11	9.4	10.6	3.3	17.3	—	—	—	—	—	11	1.4	1.5	0.5	2.4
12	12	11.0	11.8	4.0	21.4	—	—	—	—	—	11	1.8	2.0	0.7	3.6
13	10	9.8	10.6	4.3	20.5	—	—	—	—	—	10	1.7	1.6	0.6	2.4
14	11	10.2	9.9	2.0	13.0	—	—	—	—	—	10	2.0	1.9	0.5	2.7
15	12	75.9	76.8	25.5	116.9	—	—	—	—	—	10	4.3	4.4	1.1	6.4
COPD															
11	12	13.7	13.4	4.9	21.0	12	1.6	1.4	0.6	2.4	12	1.5	1.7	0.9	3.6
12	12	5.6	6.0	2.9	13.3	11	1.3	1.2	0.5	2.0	12	1.3	1.3	0.6	2.3
13	11	7.1	8.6	4.6	20.4	11	1.1	1.1	0.5	1.9	11	1.3	1.5	0.7	3.2
14	12	9.9	9.9	3.7	16.0	11	0.9	1.0	0.6	2.1	11	1.6	1.9	0.9	3.9
15	11	8.1	12.0	8.0	32.5	11	1.3	1.2	0.4	1.6	11	1.7	1.5	0.9	3.5
Ambient	16	11.7	14.6	7.0	29.2										

Session / Cohort / Subject	O_3 (ppb)					NO_2 (ppb)					SO_2 (ppb)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Winter															
Session 5															
Seniors															
1	12	0.5	0.1	1.3	1.6	12	3.9	4.2	1.9	7.4	11	0.3	0.2	1.4	2.3
2	11	−0.3	0.0	1.1	1.7	11	16.3	17.0	3.9	24.5	10	1.1	1.2	1.8	5.0
3	6	1.6	1.7	1.6	4.1	5	19.4	21.7	6.7	29.9	4	−0.5	−0.9	0.8	−0.4
4	9	0.7	0.4	1.6	3.0	9	15.4	15.2	4.5	20.0	9	1.3	0.7	1.2	2.1
5	11	0.7	0.6	1.0	1.8	11	19.2	19.2	5.2	29.5	11	0.4	0.0	1.8	2.5
Children															
1	10	1.9	1.7	2.7	5.2	10	19.1	25.0	13.7	58.8	10	−0.3	−0.7	1.2	1.1
2	11	0.3	0.3	1.1	2.5	11	13.0	16.0	15.5	60.2	12	0.6	0.5	1.3	1.9
3	10	0.6	0.1	1.2	1.4	11	9.5	13.0	11.1	40.8	12	−0.3	−0.1	1.6	2.7
4	10	0.7	0.8	0.6	1.6	10	8.0	16.8	22.2	78.6	10	0.6	2.0	5.1	16.3

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	O_3 (ppb)					NO_2 (ppb)					SO_2 (ppb)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Winter (*continued*)															
Session 5 (*continued*)															
COPD															
1	7	0.3	0.3	1.0	1.7	9	3.1	6.4	10.2	30.5	9	0.0	0.3	0.8	2.1
2	10	0.5	−0.1	1.3	1.5	8	17.2	19.0	14.9	51.0	8	0.3	0.1	1.0	1.4
3	12	0.7	0.6	1.0	2.9	12	0.9	2.9	4.6	13.6	12	0.4	0.4	1.2	2.6
4	12	0.2	0.6	2.1	6.7	10	7.2	16.8	20.0	63.5	11	0.4	0.2	1.2	1.8
5	11	−0.1	0.1	0.9	1.2	11	12.8	26.4	25.2	72.3	11	0.1	0.2	1.5	2.4
Ambient	12	8.2	11.0	7.2	23.4	12	25.2	23.5	6.1	34.4	12	6.1	6.5	3.1	13.5
Session 6															
Seniors															
6	11	0.6	0.5	1.2	2.71	10	5.7	8.6	8.5	24.4	10	0.5	0.3	1.6	2.3
7	12	0.5	0.5	0.9	2.46	11	11.3	18.2	17.7	54.1	11	0.4	0.3	1.4	3.5
8	11	0.3	0.9	1.5	3.63	10	11.3	18.0	18.6	52.6	10	0.1	−0.5	1.7	1.7
9	11	0.9	0.8	1.6	3.7	11	6.4	14.2	16.4	42.1	11	−0.9	−0.6	1.4	1.3
Children															
5	12	0.9	1.2	1.6	4.9	11	23.5	26.6	10.9	51.9	11	0.8	0.5	1.5	2.2
6	11	0.0	0.6	1.5	3.1	11	24.8	25.7	6.8	38.1	11	0.0	1.8	4.3	11.1
7	12	0.9	0.9	1.1	2.8	11	13.3	12.2	6.3	21.9	11	0.0	−0.3	1.9	3.1
8	10	0.9	0.8	1.5	4.2	12	4.4	5.3	4.7	16.9	12	0.5	0.6	2.9	8.8
9	11	2.2	1.7	1.7	3.8	11	5.4	8.9	9.4	36.0	11	0.6	0.6	1.2	2.9
10	12	0.7	0.8	1.1	2.5	12	3.4	4.6	4.7	16.0	12	0.2	0.3	1.3	3.4
COPD															
6	10	1.1	1.1	1.3	3.1	10	17.5	21.9	14.3	56.1	10	0.6	0.3	2.2	3.2
7	12	0.9	0.6	2.0	4.6	12	10.6	10.6	3.2	15.4	12	−0.7	−0.6	1.4	2.2
8	12	1.3	1.0	1.1	2.4	12	2.9	4.4	4.9	15.0	12	−0.2	−0.2	1.2	1.8
9	11	−0.4	−0.1	3.0	3	10	13.9	14.1	2.5	19.5	11	−0.5	−0.8	1.4	1.3
10	12	1.0	1.1	1.3	3.2	11	19.2	28.4	27.2	109.0	11	0.5	0.3	1.4	2.3
Ambient	12	14.7	14.7	9.3	34.5	12	24.4	24.0	9.4	38.5	12	9.9	10.5	3.2	16.5
Session 7															
Seniors															
10	12	−2.8	−2.1	4.3	6.67	12	15.7	20.8	24.8	94.4	12	0.3	−0.1	1.5	1.8
11	10	0.0	−0.8	3.8	3.81	11	9.2	11.8	9.0	25.9	11	−0.3	−0.3	1.4	2.6
12	10	0.0	−0.2	5.0	8.51	11	12.3	13.6	9.7	32.3	11	−0.2	−0.7	1.5	1.7
13	11	−2.8	−2.5	2.1	0.3	11	15.1	18.9	9.2	36.7	11	−0.3	−0.8	1.3	0.7
14	12	−0.8	−1.3	3.1	2.78	12	25.9	30.1	11.1	54.5	12	0.8	0.5	1.6	2.7
15	11	0.4	1.5	5.7	10.55	11	10.0	13.1	10.3	40.6	11	−0.3	−0.7	1.4	1.4
Children															
11	5	0.6	1.1	1.1	2.9	11	4.6	9.5	14.9	51.3	11	−0.3	0.1	2.0	3.2
12	6	−2.9	−3.1	0.7	−2.6	12	11.4	18.4	16.3	46.8	12	0.4	−0.3	1.9	2.9
13	4	−3.7	−3.9	3.2	−1.1	8	25.2	23.4	15.0	49.2	9	−2.1	−1.0	1.9	1.8
14	4	−1.3	−1.8	2.8	1	11	16.8	31.3	28.1	105.4	11	1.0	0.2	1.4	1.5
15	7	−2.5	−0.3	4.8	8.7	11	33.6	37.0	16.7	69.4	11	0.4	−0.1	1.4	1.7

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	O_3 (ppb)					NO_2 (ppb)					SO_2 (ppb)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Winter (*continued*)															
Session 7 (*continued*)															
COPD															
11	10	3.4	3.9	3.6	11.6	12	18.3	17.9	15.3	44.1	12	0.0	0.3	0.8	1.6
12	9	2.4	1.7	2.5	5.0	12	13.6	18.8	13.4	41.2	12	−0.3	0.2	1.6	4.1
13	9	2.4	3.4	4.1	9.5	12	16.5	23.8	16.9	61.0	12	0.6	0.3	1.5	2.0
14	9	1.8	1.6	1.9	5.6	12	16.4	18.4	15.9	42.3	12	−0.7	−0.8	1.4	1.5
15	8	1.6	1.9	2.4	5.1	11	15.5	20.6	17.0	58.3	11	0.1	−0.2	1.6	1.8
Ambient	16	27.0	26.0	7.0	36.3	16	20.8	20.9	7.2	37.3	16	10.8	10.0	5.0	19.5

Session / Cohort / Subject	$PM_{2.5}$ (μg/m^3)					SO_4 (μg/m^3)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Summer										
Session 1										
Seniors										
1	12	14.8	16.4	5.5	25.6	12	2.7	3.0	1.6	6.5
2	10	27.4	29.2	8.5	43.1	10	3.7	5.0	3.5	13.8
3	3	14.3	25.3	21.3	49.8	3	2.6	6.6	7.1	14.8
4	10	24.0	28.5	12.3	49.0	10	4.8	7.5	4.9	17.9
Ambient	12	21.6	25.3	12.8	46.8	10	6.9	9.4	6.2	21.2
Session 2										
Seniors										
5	11	21.2	23.5	11.0	52.4	11	5.2	5.0	3.2	13.4
6	10	27.5	32.8	17.6	78.0	10	4.9	4.7	1.8	7.5
7	12	20.6	21.9	7.7	39.4	12	6.6	6.4	3.3	14.2
8	12	18.6	21.6	6.4	34.9	12	6.3	6.3	2.9	11.5
9	12	21.0	22.6	8.4	42.1	12	5.4	6.1	3.4	15.1
10	9	23.5	23.5	7.6	40.1	9	6.8	7.5	2.9	14.9
Ambient	12	25.0	26.4	9.5	47.1	12	12.5	11.7	7.0	29.6
Session 3										
Seniors										
11	12	19.0	20.9	10.1	35.5	12	6.0	6.8	4.6	16.5
12	12	12.8	12.9	4.1	23.0	12	3.4	3.6	1.8	6.4
13	12	18.0	19.6	8.0	31.5	12	4.5	5.5	3.7	11.3
14	11	20.4	20.0	6.7	28.8	11	7.1	6.7	3.8	14.0
15	12	20.5	18.9	6.4	29.0	12	6.5	5.8	2.8	9.3
Ambient	13	21.1	23.9	12.7	45.8	13	6.8	10.2	8.2	27.3

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	$PM_{2.5}$ (μg/m^3)					SO_4 (μg/m^3)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Summer (*continued*)										
Session 4										
Children										
1	7	10.8	14.4	8.9	31.0	—	—	—	—	—
2	6	13.8	13.3	3.8	17.8	—	—	—	—	—
3	8	11.3	12.4	4.0	21.5	—	—	—	—	—
4	8	26.1	25.5	6.6	35.7	—	—	—	—	—
5	6	25.6	24.4	5.8	33.2	—	—	—	—	—
6	8	21.1	25.2	8.4	44.3	—	—	—	—	—
7	8	15.8	16.9	4.5	25.9	—	—	—	—	—
8	6	14.0	17.0	7.5	29.6	—	—	—	—	—
9	7	14.8	17.0	9.0	34.2	—	—	—	—	—
Ambient	11	19.3	23.2	14.0	59.5	—	—	—	—	—

Session / Cohort / Subject	O_3 (ppb)					NO_2 (ppb)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Summer (*continued*)										
Session 1										
Seniors										
1	12	0.1	0.1	1.9	3.1	12	−1.2	−1.0	1.7	3.4
2	10	3.7	3.0	3.0	6.5	12	3.4	2.6	4.3	8.3
3	3	0.1	1.7	3.7	5.9	3	8.3	9.8	4.3	14.6
4	10	6.3	6.2	3.6	11.5	10	8.4	8.3	2.5	12.7
Ambient	12	34.5	37.5	9.4	53.6	12	18.7	20.7	5.2	30.6
Session 2										
Seniors										
5	10	1.2	1.1	1.8	4.3	11	7.4	8.6	4.4	16.2
6	9	0.2	0.6	1.4	2.7	10	7.1	8.2	5.4	20.5
7	11	2.7	3.2	1.8	6.9	12	5.6	5.8	3.0	11.0
8	11	1.1	1.0	1.4	3.4	12	4.8	5.4	5.1	14.4
9	10	0.8	0.9	1.5	4.0	11	3.4	9.8	17.3	59.3
10	8	3.1	4.0	4.0	11.7	9	3.9	10.4	13.4	39.6
Ambient	12	39.7	38.4	7.1	51.0	12	22.7	21.5	4.5	30.8
Session 3										
Seniors										
11	9	2.4	4.7	6.7	21.1	11	17.1	16.7	4.9	26.8
12	10	9.9	8.5	5.6	17.1	12	8.4	13.5	16.9	65.8
13	9	3.1	5.3	4.6	10.4	11	10.0	10.5	4.5	18.1
14	8	9.7	9.6	2.2	14.1	11	7.3	8.5	5.1	17.2
15	7	9.7	9.8	3.5	16.9	12	11.3	11.9	6.1	22.7
Ambient	13	36.3	36.9	7.6	48.7	13	20.4	21.8	5.2	30.5

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	O_3 (ppb)					NO_2 (ppb)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Baltimore Summer (*continued*)										
Session 4										
Children										
1	6	0.2	0.3	4.3	5.8	6	17.2	18.0	10.2	35.7
2	6	−0.5	1.2	3.5	7.9	6	16.5	16.7	4.5	24.0
3	7	4.0	2.5	4.6	6.0	6	4.3	4.9	2.6	8.2
4	7	4.7	4.1	4.0	9.2	7	14.6	19.2	13.5	49.0
5	5	3.6	2.1	5.4	5.8	4	18.7	19.3	4.2	24.8
6	6	4.5	4.2	4.5	10.3	7	18.0	18.5	4.8	27.7
7	8	3.0	2.0	5.4	8.5	8	11.1	11.8	3.4	17.4
8	7	−0.6	−1.9	4.7	3.5	7	8.8	8.9	1.9	11.8
9	7	6.3	4.1	5.7	10.7	7	5.1	5.3	2.7	8.0
Ambient	11	34.7	32.9	11.6	58.0	11	18.7	20.4	8.1	36.9

Session / Cohort / Subject	$PM_{2.5}$ (μg/m^3)					SO_4 (μg/m^3)					EC (μg/m^3)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Boston Winter															
Session 11															
Children															
1	3	26.2	45.7	44.4	96.5	10	2.2	2.3	1.6	5.9	12	1.8	1.8	0.7	3.3
2	8	8.7	10.4	3.9	17.3	11	1.8	2.1	1.0	3.9	12	1.0	1.3	1.1	4.5
3	8	25.8	25.1	5.7	34.2	11	6.3	5.9	1.9	8.8	12	1.6	1.9	1.2	4.8
4	5	20.0	22.1	7.4	32.2	8	2.9	2.8	1.6	4.9	9	0.7	2.5	5.4	16.8
Seniors															
1	7	11.5	11.4	2.4	15.7	11	0.9	1.1	0.6	2.6	9	1.2	1.0	0.4	1.6
2	8	11.9	12.1	3.4	16.3	10	1.6	1.9	1.1	3.7	9	1.1	1.3	0.9	3.5
3	8	7.7	9.2	3.8	14.8	10	1.8	2.0	1.1	3.8	11	0.9	1.0	0.6	2.6
4	7	9.1	9.8	3.5	14.8	10	1.2	1.4	0.8	2.8	10	1.0	0.9	0.3	1.3
5	10	11.9	11.6	3.6	17.0	10	1.7	1.9	1.3	4.5	12	1.1	1.1	0.5	1.7
Ambient	13	11.0	13.1	5.9	24.0	13	3.0	3.4	1.5	6.4	13	1.1	1.3	0.5	2.2
Session 12															
Children															
5	7	21.0	20.2	8.5	31.0	8	2.3	2.4	1.1	4.2	8	2.1	1.9	0.8	3.1
6	9	14.7	15.1	3.5	22.5	9	2.9	2.9	1.3	4.8	10	1.4	2.0	1.5	6.1
7	10	15.3	21.0	12.5	45.1	12	1.8	2.5	1.6	5.8	12	1.3	1.3	0.7	3.3
8	11	16.7	17.2	8.8	38.9	10	2.1	2.1	0.9	3.7	10	1.6	1.8	0.8	3.5
Seniors															
6	12	16.2	16.9	5.4	30.6	11	3.0	3.0	1.5	6.5	12	1.6	1.9	0.8	4.0
7	8	17.8	19.4	7.2	33.8	9	2.5	2.7	1.6	5.4	9	1.7	1.8	0.7	3.1
8	11	9.1	10.1	5.5	21.5	12	1.8	1.9	1.0	4.0	12	1.1	1.1	0.4	1.9
9	10	13.3	15.3	7.7	34.8	10	2.4	2.6	1.2	5.1	11	1.1	1.3	0.6	2.9
Ambient	12	13.1	15.5	7.1	32.3	12	4.1	4.2	1.9	7.4	12	1.3	1.5	0.7	3.5

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	$PM_{2.5}$ (μg/m^3)					SO_4 (μg/m^3)					EC (μg/m^3)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Boston Winter (*continued*)															
Session 13															
Children															
9	9	20.6	20.8	5.1	30.3	11	1.5	1.9	1.2	4.8	11	1.6	1.6	1.0	3.2
10	9	16.0	30.1	26.2	84.7	9	1.2	1.3	0.3	1.7	10	1.7	2.0	1.4	5.8
11	12	15.2	14.9	5.0	25.0	11	1.1	1.3	0.8	3.6	12	1.6	1.7	0.6	2.8
12	11	13.8	14.2	7.1	26.7	11	1.2	1.5	1.0	4.1	11	1.2	1.1	0.6	2.1
13	11	12.4	12.9	5.0	23.3	11	1.3	1.7	1.2	5.1	11	1.1	1.1	0.4	1.8
14	7	11.6	11.6	1.3	13.4	7	1.2	1.6	1.2	4.0	8	1.2	1.2	0.6	2.5
Seniors															
10	9	14.7	15.2	4.4	21.7	12	1.2	1.5	0.8	3.8	12	1.3	1.3	0.7	2.5
11	11	13.8	14.2	2.8	18.2	12	1.5	1.6	1.0	3.8	12	1.4	1.4	0.6	3.0
12	12	15.6	16.2	4.5	25.3	10	1.2	1.4	0.6	3.0	12	1.5	1.6	0.4	2.3
13	11	10.8	12.3	5.9	25.2	12	1.4	1.6	1.0	4.3	12	1.5	2.0	2.3	8.9
14	8	21.1	23.2	5.3	32.0	10	1.5	1.7	1.0	4.2	11	1.4	1.7	1.0	3.9
Ambient	13	5.1	6.5	3.8	15.1	13	1.6	1.7	1.1	5.1	13	0.7	0.8	0.3	1.3

Session / Cohort / Subject	O_3 (ppb)					NO_2 (ppb)					SO_2 (ppb)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Boston Winter (*continued*)															
Session 11															
Children															
1	11	0.1	0.3	1.1	2.2	12	21.4	21.9	9.1	35.4	12	−0.1	−0.2	0.9	1.4
2	10	−0.4	0.0	1.2	2.6	11	22.4	22.4	3.3	30.4	12	−0.8	−0.1	2.4	6.9
3	10	1.4	1.0	1.1	2.4	10	24.7	24.3	4.4	33.0	10	7.2	7.2	4.7	13.1
4	8	−0.2	0.1	1.0	1.6	9	6.8	7.8	4.1	18.0	9	0.5	0.5	1.1	1.9
Seniors															
1	10	−0.1	−0.6	1.8	1.3	10	6.1	6.3	2.4	10.2	10	1.1	1.0	0.6	2.1
2	10	−0.2	−0.1	0.9	1.2	10	7.2	7.3	3.4	14.0	10	0.4	0.4	1.0	2.2
3	11	0.2	0.2	1.1	1.6	11	9.2	7.9	2.7	11.6	11	0.8	0.8	0.8	2.2
4	8	0.8	0.1	1.8	1.6	10	11.9	11.8	2.1	14.8	10	1.0	1.2	1.0	3.1
5	11	0.5	1.2	2.0	6.7	12	18.3	19.4	4.3	30.9	12	1.1	1.3	0.8	3.0
Ambient	13	31.3	30.8	6.2	45.0	13	14.3	14.0	4.3	19.6	13	9.4	10.7	3.3	18.2
Session 12															
Children															
5	8	1.1	1.5	2.1	6.0	7	23.4	25.5	4.7	31.9	7	−0.5	−0.6	0.9	0.7
6	9	0.9	0.7	0.7	1.5	9	19.5	20.6	4.9	32.0	8	−0.1	−0.2	1.1	1.7
7	11	0.8	0.6	0.9	1.8	11	12.0	11.7	3.2	18.2	11	0.1	−0.1	0.8	0.9
8	11	0.9	0.6	1.7	3.1	11	20.0	21.0	4.9	29.6	11	−0.2	−0.3	0.9	0.7

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	O_3 (ppb)					NO_2 (ppb)					SO_2 (ppb)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Boston Winter (*continued*)															
Session 12 (*continued*)															
Seniors															
6	11	0.6	1.0	1.2	2.9	12	16.2	17.2	3.9	23.6	12	0.9	1.2	1.9	6.2
7	9	0.3	0.7	1.3	3.6	9	13.2	12.8	3.8	17.4	9	0.5	0.3	1.0	1.8
8	12	0.4	0.9	1.2	2.9	12	12.6	13.1	2.5	17.1	12	0.4	0.7	1.8	4.0
9	11	0.8	0.8	0.7	2.6	11	12.4	11.4	3.0	15.7	11	0.4	0.4	1.1	2.0
Ambient	12	33.7	32.6	7.3	44.1	12	14.5	14.5	5.1	22.2	12	8.8	8.6	3.2	15.0
Session 13															
Children															
9	10	1.6	1.9	1.7	5.4	10	16.3	17.1	4.4	23.9	11	0.2	0.2	1.1	1.5
10	10	1.0	2.1	3.3	10.8	10	31.0	38.3	21.8	75.8	10	0.1	−0.1	1.2	2.0
11	11	1.7	1.8	1.7	6.5	12	30.6	34.9	16.8	70.6	12	0.5	0.4	2.1	5.7
12	11	3.0	4.0	3.5	11.6	11	13.0	12.3	6.3	27.3	11	0.3	0.3	0.8	1.2
13	11	2.4	2.6	1.7	5.9	11	8.0	8.6	3.1	14.4	11	0.8	0.9	0.7	2.6
14	6	0.6	0.6	0.5	1.3	7	12.8	18.3	12.7	41.2	8	0.0	−0.2	1.3	1.4
Seniors															
10	12	2.0	1.7	0.9	2.7	12	13.4	12.6	3.9	18.3	12	0.7	0.6	1.1	2.8
11	12	3.9	3.6	0.7	4.7	12	14.6	15.9	5.8	32.4	12	0.8	0.9	0.5	1.7
12	11	2.1	2.5	1.4	5.2	12	9.6	10.0	3.4	17.9	12	0.6	0.4	0.7	1.0
13	12	1.7	1.9	1.0	4.0	12	20.1	19.6	4.2	26.3	12	0.5	0.5	0.9	1.9
14	10	2.7	2.7	1.6	4.8	10	15.4	16.6	5.4	26.0	10	1.0	1.0	0.5	1.9
Ambient	13	19.9	21.1	6.9	31.8	13	22.5	21.8	5.6	29.7	13	4.9	4.9	1.6	7.6

Session / Cohort / Subject	$PM_{2.5}$ (μg/m^3)					SO_4 (μg/m^3)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Boston Summer										
Session 8										
Seniors										
1	11	14.8	16.4	5.3	26.3	11	2.1	2.4	1.2	4.6
2	12	17.7	16.6	5.1	24.8	11	3.4	3.2	1.2	5.1
3	11	16.1	17.0	5.3	26.8	11	2.9	2.9	1.4	4.9
4	12	19.3	18.5	7.4	34.1	12	2.6	2.7	1.3	5.1
5	12	21.2	24.3	7.9	38.7	12	2.8	2.9	1.5	5.3
6	12	10.4	11.2	2.7	17.5	12	2.1	2.2	1.6	6.1
7	12	16.8	17.3	4.7	24.3	12	2.7	2.5	1.2	4.3
8	12	15.9	15.9	4.8	23.9	11	3.2	3.0	1.2	5.0
9	12	20.8	23.0	7.8	38.9	12	2.5	2.6	1.2	4.8
Ambient	11	10.2	11.9	5.1	20.9	8	3.4	3.6	1.0	5.1

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	$PM_{2.5}$ (μg/m^3)					SO_4 (μg/m^3)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Boston Summer (*continued*)										
Session 9										
Children										
1	11	29.3	29.2	6.9	40.2	12	4.7	4.5	2.5	8.4
2	12	24.4	28.7	16.0	68.7	12	5.7	4.8	2.8	9.8
3	12	21.7	21.7	7.6	37.7	12	4.7	4.6	2.9	9.2
4	12	21.4	24.0	8.8	43.7	12	3.0	3.1	2.0	7.5
5	12	22.9	23.6	9.1	44.5	12	4.3	4.1	2.6	9.1
Seniors										
10	12	17.8	21.0	12.0	55.4	12	2.9	2.8	1.4	1.2
11	12	20.3	20.7	8.9	39.2	12	5.3	4.5	2.9	9.5
12	12	9.9	18.3	19.2	61.5	12	1.6	1.8	1.3	3.8
13	12	21.9	21.9	6.8	34.8	12	6.3	5.6	3.1	11.5
14	11	14.4	20.4	17.7	66.2	11	1.8	1.8	1.1	4.2
Ambient	13	13.1	13.3	5.7	22.5	12	4.2	4.2	2.5	8.8
Session 10										
Children										
6	12	20.4	24.6	12.5	49.0	12	3.0	5.9	6.3	18.1
7	12	27.1	33.1	15.6	66.3	12	3.4	6.7	7.4	24.3
8	12	27.6	33.7	20.0	76.6	12	3.3	6.3	6.9	21.1
9	11	32.8	35.7	16.3	66.9	11	2.5	4.9	5.1	16.0
10	11	33.2	37.7	17.8	63.9	11	4.1	6.7	7.0	20.2
11	11	31.6	31.4	12.3	51.9	12	3.1	5.4	5.5	17.0
12	12	28.7	34.0	13.1	63.4	12	3.0	5.4	5.5	16.6
13	11	31.7	35.7	14.9	72.3	11	2.9	5.9	7.1	23.7
14	12	27.6	33.1	13.8	63.2	12	3.6	7.4	7.8	22.1
15	11	21.5	29.4	16.6	60.6	11	3.1	7.3	8.3	24.9
Ambient	12	11.9	21.4	14.2	46.8	11	3.7	9.0	7.5	21.4

Table continues on next page

[a] — is not measured.

APPENDIX L. Personal Exposures by Subject, City and Season[a] (*continued*)

Session / Cohort / Subject	O_3 (ppb)					NO_2 (ppb)					SO_2 (ppb)				
	N	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max	*N*	Median	Mean	SD	Max
Boston Summer (*continued*)															
Session 8															
Seniors															
1	11	0.2	0.9	2.1	4.2	11	10.8	12.5	7.6	25.4	11	−0.1	−0.1	1.0	1.9
2	12	7.1	7.9	10.2	32.5	12	12.2	14.7	11.1	31.7	12	0.4	1.7	6.1	20.8
3	12	4.2	5.7	5.3	15.7	12	30.0	28.8	12.9	48.0	12	0.0	0.0	1.0	1.8
4	12	11.3	10.5	8.6	26.6	12	14.5	16.7	7.1	28.4	12	0.3	0.1	0.9	1.6
5	12	7.6	11.0	10.4	34.5	12	10.1	11.5	6.9	22.4	12	0.0	0.0	1.5	4.0
6	11	3.9	3.4	4.3	10.9	12	8.7	8.4	4.8	13.6	11	−0.1	0.0	1.3	3.1
7	12	2.3	2.1	1.8	4.8	11	20.3	21.4	8.4	41.0	11	0.6	0.6	2.1	6.5
8	12	3.6	2.9	1.8	5.0	12	23.7	23.4	7.0	32.6	12	0.6	0.7	1.7	4.8
9	12	1.3	1.7	1.3	4.2	12	14.8	14.9	4.9	21.5	12	0.3	0.1	0.9	1.4
Ambient	12	22.8	23.1	11.2	46.5	12	23.0	25.4	8.5	37.2	12	3.9	4.0	1.7	7.8
Session 9															
Children															
1	12	3.6	4.3	3.3	10.6	12	23.7	27.6	13.2	59.1	12	0.5	0.9	1.7	6.1
2	12	10.0	10.1	5.1	20.6	12	13.1	12.3	5.7	22.3	11	0.2	0.3	0.8	1.9
3	12	8.0	9.8	6.8	27.3	12	13.7	13.4	5.8	20.3	11	−0.1	0.0	0.5	1.1
4	12	4.5	5.5	3.6	12.1	12	8.3	7.4	4.8	11.3	12	0.2	1.1	3.5	11.9
5	12	8.3	8.2	5.4	20.4	12	12.2	12.4	4.1	22.3	12	0.5	0.5	0.8	1.7
Seniors															
10	10	0.2	1.3	2.6	7.4	12	21.1	23.0	5.8	32.0	12	0.1	0.2	0.5	5.3
11	10	5.6	5.7	3.5	12.6	12	6.4	6.9	3.6	14.1	12	0.5	0.4	0.8	1.4
12	11	1.5	2.1	3.0	9.6	12	7.2	6.1	5.5	14.7	10	0.7	1.0	1.0	2.6
13	11	12.6	12.8	4.8	21.5	12	14.6	14.7	6.2	22.1	11	0.2	0.5	0.6	1.5
14	8	2.4	2.2	2.0	5.5	11	10.3	10.2	3.3	16.3	11	0.1	0.0	0.4	0.7
Ambient	13	23.0	22.7	8.8	46.3	13	23.1	21.7	3.9	29.2	13	2.9	2.8	0.7	4.4
Session 10															
Children															
6	12	5.3	7.0	7.4	24.6	12	25.2	23.9	5.8	33.2	12	0.4	0.4	0.8	1.8
7	12	4.0	6.3	5.8	17.7	12	30.9	32.8	8.6	50.3	12	0.2	0.2	0.4	1.1
8	12	3.2	6.3	6.4	17.1	12	26.1	25.7	4.9	34.4	12	0.5	0.5	0.7	1.5
9	11	2.9	4.4	4.3	13.1	11	27.5	28.2	7.6	45.7	11	0.4	1.1	2.5	8.2
10	11	2.5	3.6	3.2	9.8	11	27.1	28.6	6.9	42.6	11	0.1	0.2	0.5	1.2
11	12	4.4	5.5	3.6	11.9	12	31.9	31.0	3.8	35.8	12	0.3	0.5	0.7	1.7
12	12	5.3	6.2	4.9	17.8	12	30.4	30.5	4.3	37.9	12	0.4	0.4	0.8	1.7
13	11	2.5	4.7	4.0	13.6	11	34.6	35.4	6.2	46.8	11	0.9	0.6	0.8	1.7
14	12	4.7	6.5	6.0	21.8	12	36.1	37.0	8.4	53.8	12	0.4	0.4	0.6	1.5
15	11	11.3	17.1	16.1	48.5	11	26.6	23.0	10.4	33.0	11	0.4	0.5	0.7	2.1
Ambient	12	27.9	31.6	19.2	64.9	12	26.4	27.8	5.6	37.8	12	4.4	4.5	1.1	5.9

[a] — is not measured

APPENDIX M. Personal Exposures and Ambient Concentrations by City, Season, and Cohort

$PM_{2.5}$

Personal $PM_{2.5}$ exposures tended to be lower than ambient in Baltimore and somewhat higher than ambient in Boston (Figure M.1). In addition, personal exposures appear to differ by cohort, but these results do not take into account the effect of ambient $PM_{2.5}$ on personal exposures on a daily basis. The dotted line represents the 24-hour US National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ (65 $\mu g/m^3$), which is generally based on the 3-year average of the 98th percentile value of the 24-hour concentrations at each ambient monitor in a given area. The annual NAAQS, based on the 3-year arithmetic mean, is 15 $\mu g/m^3$.

SO_4^{2-}

Personal SO_4^{2-} exposures were lower than ambient in both cities, with SO_4^{2-} levels slightly higher in Baltimore than Boston (Figure M.2). The plots for summer in Boston show different ambient levels associated with seniors and children. This is due to differences in scheduling. The first sampling session comprised all seniors; the last sampling session comprised all children and included a 3-day pollution event.

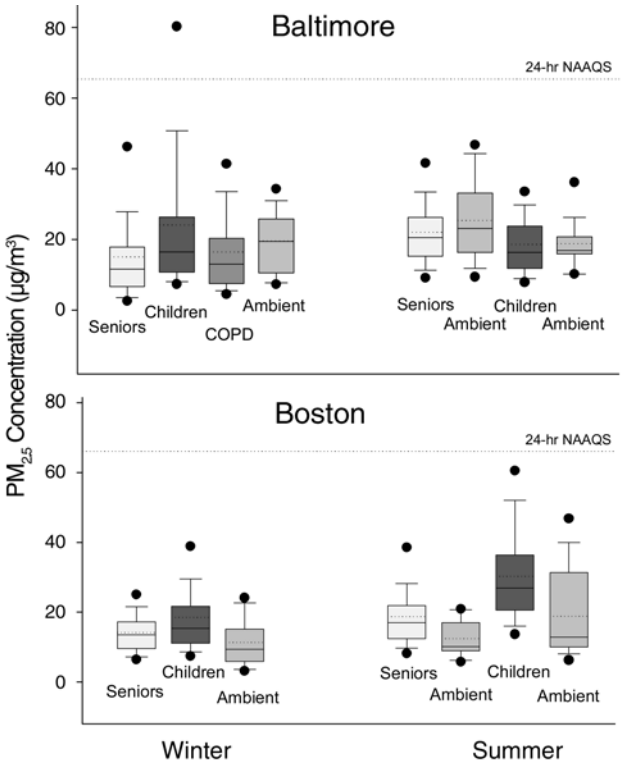


Figure M.1. Personal exposures and ambient concentrations for $PM_{2.5}$. Medians are indicated by the solid line inside each box.

EC IN WINTER

Winter EC exposures were slightly higher than ambient in Boston with children having somewhat greater variability in EC exposures (Figure M.3). Ambient EC data were not collected in Baltimore, thus no comparisons with ambient EC could be made.

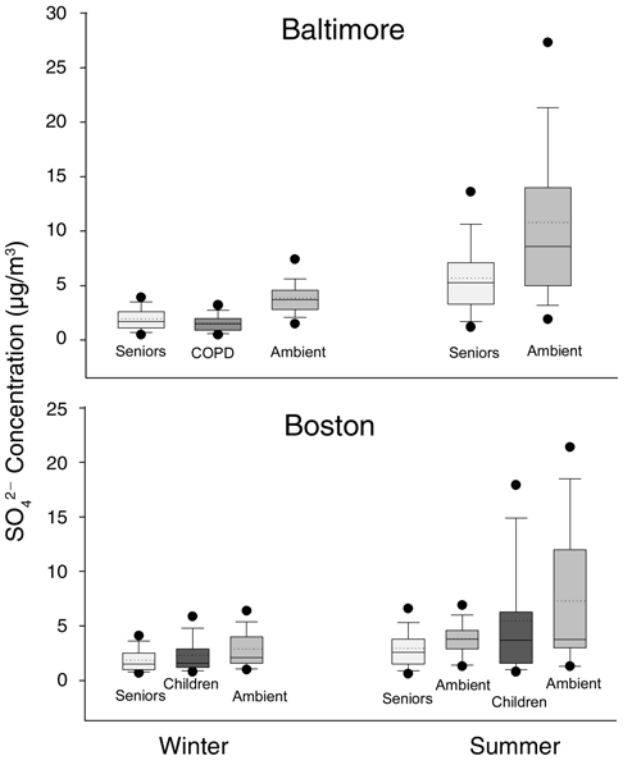


Figure M.2. Personal exposures and ambient concentrations for SO_4^{2-}. Medians are indicated by the solid line inside each box.

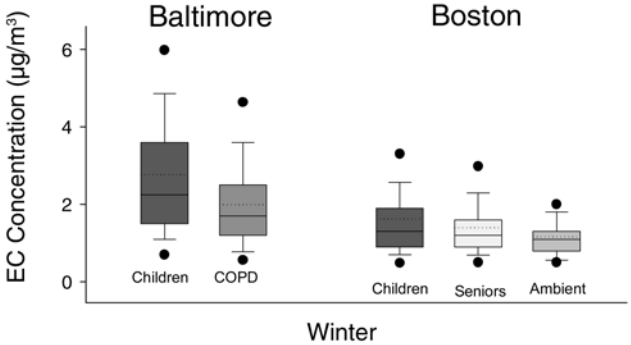


Figure M.3. Personal exposures and ambient concentrations for EC in winter. Ambient EC data were not collected in Baltimore. Medians are indicated by the solid line inside each box.

O_3

Personal O_3 exposures were extremely low in comparison to the ambient levels in both cities (Figure M.4). There was also little difference in exposures by cohort. All personal and ambient O_3 levels were below the 8-hour and 1-hour standards of 80 and 120 ppb, respectively.

NO_2

Personal NO_2 exposures tended to be somewhat lower than ambient for all cohorts with the exception of children in Boston during summer (Figure M.5). During the summer in Baltimore and both seasons in Boston, children had higher NO_2 exposures than seniors. A larger fraction of these children lived in homes with gas stoves compared with homes with electric stoves. The dotted line represents the annual NAAQS for NO_2 of 53 ppb, which is based on the annual arithmetic mean ambient NO_2 concentration. There were 25 personal exposures greater than 53 ppb, but no ambient levels exceeded the annual standard.

SO_2

Personal SO_2 exposures were extremely low in comparison to the ambient levels in both cities (Figure M.6). There was also little difference in exposures by cohort. No personal or ambient observations were greater than the annual SO_2 NAAQS of 30 ppb.

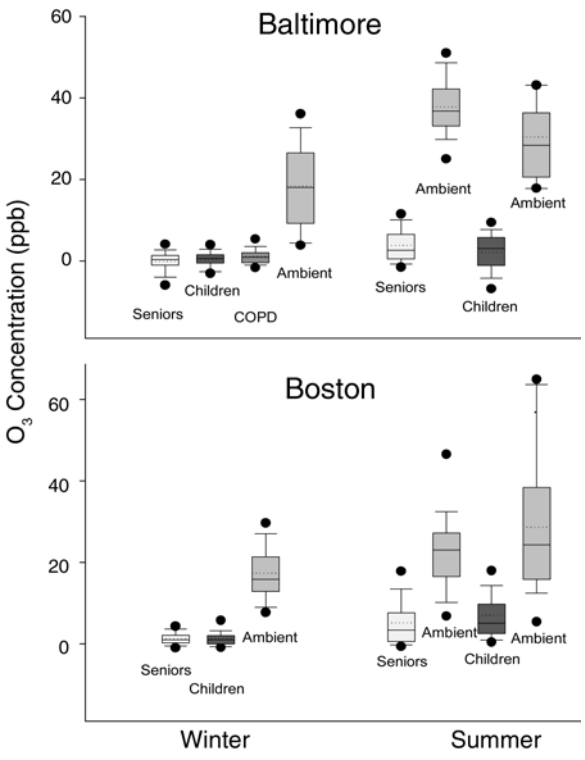


Figure M.4. Personal exposures and ambient concentrations for O_3. Medians are indicated by the solid line inside each box.

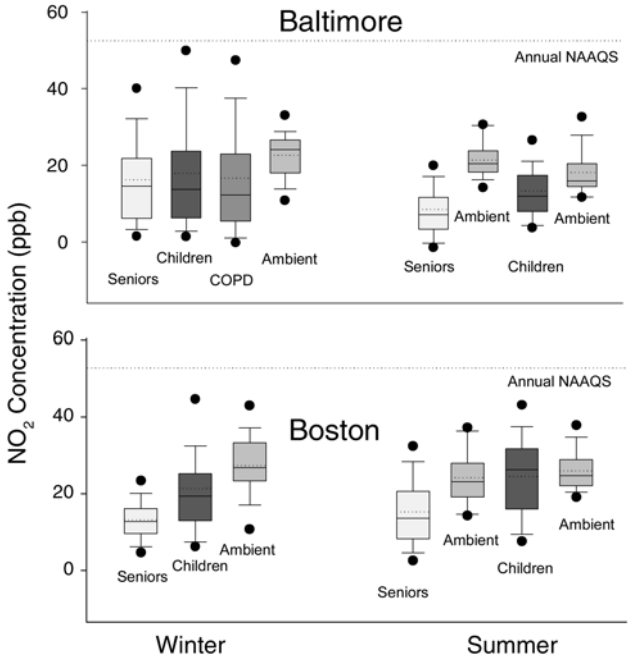


Figure M.5. Personal exposures and ambient concentrations for NO_2. Medians are indicated by the solid line inside each box.

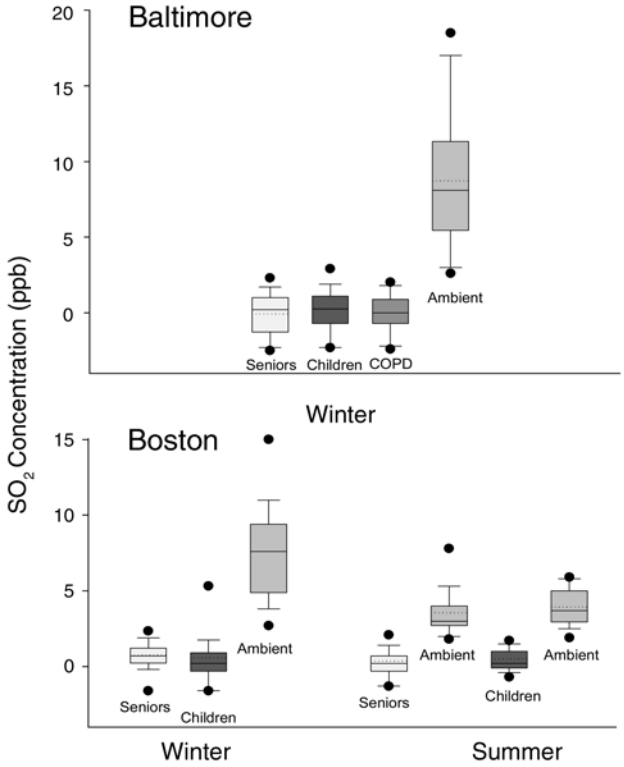


Figure M.6. Personal exposures and ambient concentrations for SO_2. Medians are indicated by the solid line inside each box.

APPENDIX N. Change in Model 2 Slopes Caused by Removal of Individual Subjects

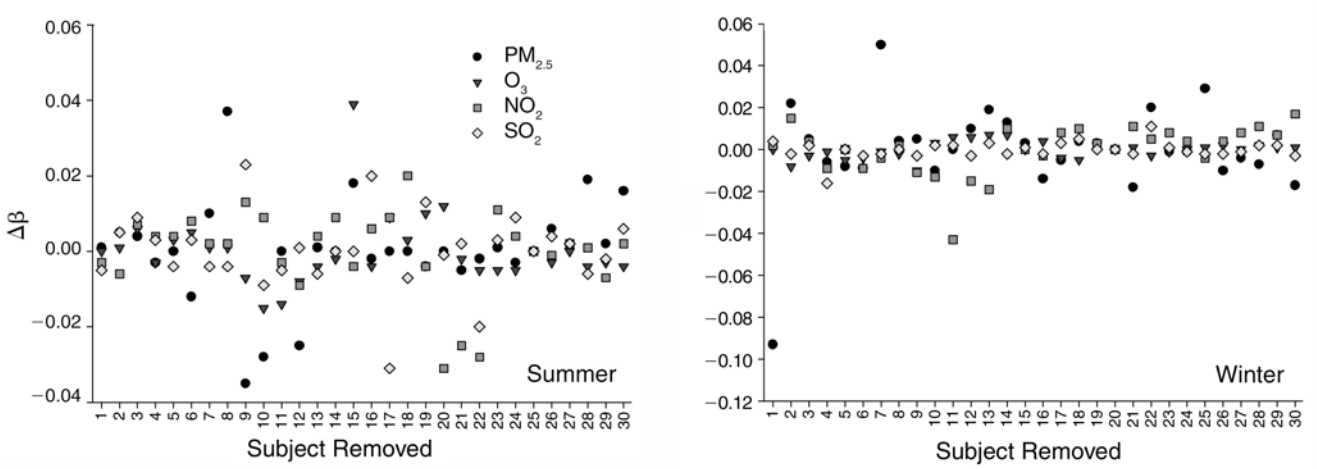


Figure N.1. Boston. Note difference in y-axis scales between panels.

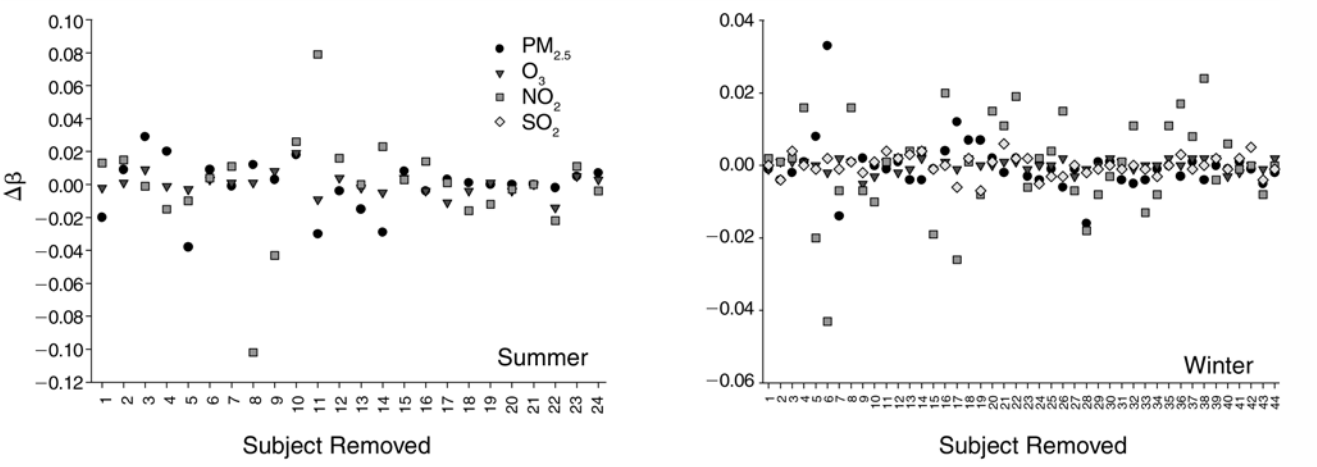


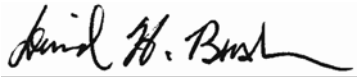
Figure N.2. Baltimore. Note difference in y-axis scales between panels.

APPENDIX O. HEI QUALITY ASSURANCE REPORT

The conduct of this study was subject to independent audits by Mr David Bush of Technical & Business Systems, Inc. Mr Bush is an expert in quality assurance for air quality monitoring studies and data management. The audits included on-site reviews of study activities for conformance to the study protocol and operating procedures and selected performance audits of monitoring equipment. The dates of the audits are listed in the table below with the phase of the study examined.

Date	Phase of Study
10/25–26, 1999	The auditor conducted an on-site audit at the Harvard School of Public Health (HSPH). Staffing and internal quality assurance procedures were reviewed. Audit observations consisted primarily of recommendations for improving study documentation. An overview of laboratory operations at HSPH relevant to the study was also conducted.
2/9–11, 2000	The auditor revisited HSPH during the winter monitoring period for Boston to review the personal monitoring procedures and to conduct flow audits of the personal monitoring equipment. Ambient monitoring conducted by HSPH for use in the study was also reviewed. No problems significantly affecting the collected data were noted.
12/12–13, 2001	The auditor visited HSPH to review the data management and validation efforts, which were not fully in place during the initial audits. Study personnel addressed all problems noted.
5/10–11, 2004	The auditor reviewed the study final report. An on-site audit at the study facilities was conducted to verify the integrity of the reported data. Several data points for each parameter were traced through the entire data processing sequence to verify that the described procedures were being followed and to verify the integrity of the database. Though a problem was noted with some of the cooking data, study personnel indicated that reanalysis showed only slight changes in the results, with no changes in the interpretation of the results, and that no changes in the report were needed.

Written reports of each inspection were provided to the HEI project manager, who transmitted the findings to the Principal Investigator. These quality assurance audits demonstrated that the study was conducted by an experienced team with a high concern for data quality. Study personnel were very responsive to audit recommendations. The report appears to be an accurate representation of the study.



David H Bush, Quality Assurance Officer

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Dr Brent A Coull is an associate professor of biostatistics at the Harvard School of Public Health.

Dr Joel Schwartz is an associate professor of environmental epidemiology at the Harvard School of Public Health.

OTHER PUBLICATIONS RESULTING FROM THIS RESEARCH

Sarnat JA, Brown KW, Schwartz J, Coull BA, Koutrakis P. Relationships among personal exposures and ambient concentrations of particulate and gaseous pollutants and their implications for particle health effects studies. Epidemiology. In press.

ABBREVIATIONS AND OTHER TERMS

CI	confidence interval
CO	carbon monoxide
COPD	chronic obstructive pulmonary disease
CV	coefficient of variation
EC	elemental carbon
EPA	Environmental Protection Agency (US)
ETS	environmental tobacco smoke
HEAD	Harvard-EPA annular denuder
LOD	limit of detection
MDE	Maryland Department of the Environment
MDEP	Massachusetts Department of Environmental Protection
NAAQS	National Ambient Air Quality Standard (US)
NO_2	nitrogen dioxide
NO_2^-	nitrite
NO_3^-	nitrate
O_3	ozone
PEM	personal environmental monitor
PM	particulate matter
PM_{10}	PM with aerodynamic diameters < 10 μm
$PM_{2.5}$	PM with aerodynamic diameters < 2.5 μm
$PM_{3.5}$	PM with aerodynamic diameters < 3.5 μm
PTEAM	Particle Total Exposure Assessment Methodology
r^2	coefficient of determination for bivariate analysis
r	correlation coefficient for bivariate analysis
r_s	Spearman correlation coefficient
SO_2	sulfur dioxide
SO_4^{2-}	sulfate
TEOM	tapered element oscillating microbalance

COMMENTARY
Health Review Committee

HEI

INTRODUCTION

Epidemiologic studies conducted in a variety of locations have reported that short-term increases of particulate matter (PM*) at low concentrations are associated with short-term increases in morbidity and mortality (US Environmental Protection Agency [EPA] 1996, 2004; Health Effects Institute 2001). The strongest associations have been found for individuals with compromised cardiac or respiratory function.

To measure personal exposure to PM, most epidemiologic studies of health effects of PM have either used ambient PM measurements taken at central monitoring sites as surrogate values or have modeled the contribution of ambient PM to personal exposure from those values as a function of distance from the monitoring sites to various microenvironments (outdoors, homes, schools, workplaces) and time spent in each.

For the most part, epidemiologic studies of the correlation between outdoor or ambient exposures and health effects have not been designed to adequately address four important issues directly: (1) Temporal and spatial variations exist in PM exposure. (2) Exposure to PM includes exposure to copollutants (eg, nitrogen dioxide [NO_2] and ozone [O_3]) that may differ from PM in spatial and temporal variation and that may contribute separately to health effects. (3) Lifestyle choices and activities (eg, smoking, exercise, time spent indoors and outdoors) of study participants may contribute to PM exposure. (4) Personal exposure varies both within and between subjects.

All four issues can introduce uncertainties when interpreting changes in health effects; these uncertainties could substantially bias estimates of associations between central-site monitoring levels and personal exposure levels. In addition, data about the composition of ambient aerosol mixtures are limited. This composition must be defined in order to determine the relative contribution of indoor and outdoor pollution sources. Such source apportionment might help to identify which components of PM are directly related to observed health effects, because different sources may contribute different components to the PM mixture.

In 1998, HEI issued Request for Applications (RFA) 98-1, "Characterization of Exposure to and Health Effects of Particulate Matter," to address some of these issues. One objective of this RFA was to characterize personal exposure to PM in a variety of indoor and outdoor microenvironments and in geographic locations that differ in types and sources of particles, topography, and climate in order to determine the kind of exposure information necessary for epidemiology studies. To address this objective, studies would determine particle characteristics (eg, concentration, size, and composition) and describe the relation between personal exposure and ambient exposure to PM and other pollutants that have typically been used in epidemiologic time-series studies.

In addition, the RFA encouraged investigators to characterize personal exposure for subpopulations thought to be susceptible to particulate air pollution (Pope et al 1992; Schwartz and Dockery 1992; Schwartz 1994), including children, seniors, and individuals with chronic obstructive pulmonary disease (COPD) or other respiratory disease. Children, who at that time had been little studied, may be more sensitive than others to the effects of air pollution because they spend more time outdoors. Seniors and individuals with respiratory disease may be at greater risk than others because of compromised health.

In response to RFA 98-1, Dr Petros Koutrakis from the Harvard School of Public Health submitted an application for a three-year study, "Characterization of the Particulate and Gas Exposures of Sensitive Subpopulations Living in Eastern US Metropolitan Areas." The application proposed to characterize personal exposure of children, seniors, and individuals with COPD during summer and winter in Baltimore, Maryland, and Boston, Massachusetts. The HEI Health Research Committee recommended this study for funding because it thought the study would provide information on personal exposure to indoor and outdoor pollution useful for understanding the suitability of ambient monitoring data as a surrogate for data on personal exposure to outdoor pollution. The inclusion of two cities, two seasons, and three possibly susceptible groups was thought to be a strength of the study design.†

* A list of abbreviations and other terms appears at the end of the Investigators' Report.

This document has not been reviewed by public or private party institutions, including those that support the Health Effects Institute; therefore, it may not reflect the views of these parties, and no endorsements by them should be inferred.

† Dr Koutrakis' 3-year study, "Characterization of Particulate and Gas Exposures of Sensitive Subpopulations Living in Baltimore and Boston," began in August 1998. Total expenditures were $1,133,009. The draft Investigators' Report from Koutrakis and colleagues was received for review in January 2003. A revised report, received in April 2004, was accepted for publication in June 2004. During the review process, the HEI Health Review Committee and the investigators had the opportunity to exchange comments and to clarify issues in both the Investigators' Report and in the Review Committee's Commentary.

The investigators proposed to focus on measurements of PM less than or equal to 2.5 µm in aerodynamic diameter ($PM_{2.5}$), O_3, NO_2, and sulfur dioxide (SO_2). In response to Research Committee comments, investigators added analysis of important components of $PM_{2.5}$: Ions (sulfate [SO_4^{2-}], nitrate, and ammonia) and elemental carbon (EC). They also planned to measure trace elements. A miniature motion detector was added to the personal sampler to monitor whether children actually wore the sampler during the measurement period.

HEI funded two other studies from RFA 98-1 to investigate the association between particle concentrations measured at central monitors and indoor, outdoor, and personal exposures: "Personal, Indoor, and Outdoor Exposures to $PM_{2.5}$ and Its Components for Groups of Cardiovascular Patients in Amsterdam and Helsinki," led by Bert Brunekreef at Utrecht University and his colleagues (Brunekreef et al 2005), and "Relationship Among Indoor, Outdoor and Personal Air (RIOPA): Analysis of RIOPA PM Species Concentration," led by Barbara Turpin at Rutgers University and her colleagues as part of a series of studies jointly funded by HEI and National Urban Air Toxics Research Center (Turpin et al 2006). In addition to the research on PM, the RIOPA investigators also examined the relations among indoor, outdoor, and personal air for volatile organic carbon and for carbonyls (Weisel et al 2005).

SCIENTIFIC BACKGROUND

Although remarkably consistent associations have been documented between a range of adverse health outcomes and changes in ambient PM concentrations at central-site monitors (Dockery et al 1993; Pope et al 1995; Abbey et al 1999; Samet et al 2000), uncertainty about how well central-site measurements represent personal PM exposures affects interpretation of these associations. Accurately measuring personal exposure to PM is complex because it involves both indoor and outdoor PM sources. In addition, concentrations of pollutants vary over time and among locations. Personal exposure is also a function of a number of factors, including how much time individuals spend in different microenvironments (eg, homes, workplaces, public buildings, in traffic), characteristics of these microenvironments (including indoor ventilation rates, building type and location, and season), the proximity of microenvironments to sources of pollutants, and the individual's behavioral patterns (eg, smoking, exercise, cooking, housecleaning). Within each microenvironment, the concentrations, physical properties, and chemical compositions of PM can vary widely.

Actual personal exposure to PM is difficult to determine without direct measurement or careful consideration of the factors mentioned above. However, the complexity and high cost of obtaining direct measurements of personal exposure necessitate the use of an exposure surrogate—usually ambient concentrations at central-site monitors—even if it may not adequately reflect the full range of factors that influence personal exposure, may over- or underestimate individual exposure levels, and may represent personal exposure for some pollutants better than for others.

Prior to the current study, several studies sought to examine the fraction of ambient PM that contributes to personal exposure (Spengler et al 1985; Lioy et al 1990; Clayton et al 1993; Thomas et al 1993; Janssen et al 1995; Ozkaynak et al 1996). Correlations between outdoor and personal PM measurements reported in cross-sectional studies (in which monitoring data taken at the same time from individual participants are grouped) show no clear trend: some are fairly well correlated; some are poorly or not at all correlated (Dockery and Spengler 1981; Sexton et al 1984; Spengler et al 1985; Suh et al 1992; Clayton et al 1993; Thomas et al 1993; Ozkaynak et al 1996). When the Koutrakis study was funded, accurate assessment of personal exposures, particularly among individuals shown to be more susceptible to PM, had not been done. The investigators sought to remedy that by evaluating how well ambient measures of PM and gaseous pollutants correlate with measures of personal exposures to the same pollutants.

TECHNICAL EVALUATION

AIMS AND OBJECTIVES

The overall aim of this study was to measure the contribution of ambient $PM_{2.5}$ and associated pollutants to corresponding personal exposures in possibly sensitive cohorts in two cities and in two seasons. The study was designed to test three hypotheses: (1) ambient $PM_{2.5}$ concentrations at central monitoring sites are appropriate surrogates of personal exposures to $PM_{2.5}$; (2) associations between personal exposures to $PM_{2.5}$ and ambient concentrations of $PM_{2.5}$ vary by city, season, and cohort and are stronger if the data are analyzed longitudinally (repeated measurements for each individual); and (3) measures of ambient concentrations of gaseous copollutants (such as O_3, NO_2, and SO_2) correlate more strongly with measures of personal exposure to $PM_{2.5}$ than with personal measures of those pollutants. The investigators further postulated that the relation between ambient concentrations of gaseous copollutants and personal exposure to $PM_{2.5}$ may be sufficiently strong

that using both ambient $PM_{2.5}$ and ambient gaseous pollutant concentrations as exposure variables in epidemiologic studies may be misleading.

To address these hypotheses, Dr Koutrakis' study had the following specific objectives:

- Collect longitudinal measurements of personal exposures to $PM_{2.5}$ and gaseous pollutants (carbon monoxide, SO_2, NO_2, and O_3) of children, seniors, and individuals with COPD living in Boston and Baltimore;
- Determine the associations between personal exposures to the pollutants and corresponding ambient concentrations;
- Determine the associations between personal exposures to gaseous pollutants and ambient $PM_{2.5}$ concentrations;
- Examine the interperson, intraperson, temporal, and seasonal variability in personal PM and gas exposures as a function of activity and housing characteristics; and
- Determine the effect of city and cohort on personal PM and gas exposures and their relation to corresponding outdoor concentrations.

STUDY DESIGN AND METHODS

Several possibly sensitive subpopulations were examined in this study: children in Baltimore and Boston, seniors in Boston, and individuals with COPD in Baltimore. An earlier study by investigators examined a similar cohort of seniors in Baltimore (Sarnat et al 2000). The investigators recruited individuals living in both urban and suburban communities in Baltimore and Boston and in both apartment buildings and single-family houses. In Boston, 23 children were recruited (15 participated in summer and 15 in winter, with some overlap between the two groups); in Baltimore, 21 children were recruited (15 participated in summer and 15 in winter, with some overlap between the two groups). In addition, 20 healthy seniors were recruited in Boston in summer and winter, and 15 individuals with COPD were recruited in Baltimore (but not Boston) in winter. A previous study (Sarnat et al 2000) had examined similar exposure information for a group of seniors in Baltimore in summer (11 seniors) and winter (15 seniors); these data were used in the current study. All participants were nonsmokers and lived in residences with nonsmokers. Informed consent was obtained from each participant (from a parent or guardian for children).

Integrated 24-hour personal exposures were measured over 12 consecutive days, except summer sampling for Baltimore children, which was conducted over 8 consecutive days. Pump-driven multipollutant personal exposure monitors (PEMs), equipped with both quartz and Teflon filters for collecting PM (Demokritou et al 2001) and passive Ogawa samplers for collecting gaseous pollutants, were used to assess personal exposure to O_3, NO_2, SO_2, and $PM_{2.5}$ and associated EC and SO_4^{2-}. The Teflon filter was used to determine PM mass (by using weighing techniques) and the quartz filter was used to determine EC (by using thermal–optical reflectance techniques). SO_4^{2-} concentrations were assessed by extracting material from the Teflon filters and analyzing it by using ion chromatography. Gaseous pollutants were collected by two passive Ogawa samplers containing cellulose filters coated with nitrite (for O_3) or triethanolamine (for NO_2 and SO_2).

Ambient concentrations and personal exposures to a number of pollutants were evaluated in each city in both summer and winter. Concentrations of ambient $PM_{2.5}$ integrated over 24 hours were obtained using Harvard impactors at each of two state ambient monitoring (SAM) sites in Baltimore and from a single central monitoring site on the roof of the Harvard School of Public Health in Boston. Concentrations of SO_4^{2-} integrated over 24 hours were obtained from material extracted from $PM_{2.5}$ filters in the Harvard impactors in Baltimore and from filters from Harvard-EPA annular denuders (HEADs) in Boston.

Ambient EC concentrations were obtained in Boston, by using ChemComb samplers, but were not obtained in Baltimore. Continuous $PM_{2.5}$ data from a different site in Baltimore were obtained by using tapered element oscillating microbalance (TEOM) monitors (operated by the Maryland Department of Environmental Protection) and in Boston by using TEOM monitors at the same central monitoring site that was used to collect the integrated $PM_{2.5}$ samples. No corresponding continuous SO_4^{2-} and EC data were available from either the Baltimore or Boston sites. Daily mean O_3, NO_2, and SO_2 concentrations were calculated from continuous data obtained at stationary ambient monitoring sites operated by the Maryland Department of the Environment (Baltimore) and by the State of Massachusetts in Suffolk County (Boston) to coincide with the start and end of the $PM_{2.5}$ sample collection periods. These data were collected by using UV photometric analysis, chemiluminescence monitors, and pulsed fluorescence monitors.

Participants in each city, season, and group completed questionnaires that provided information about housing characteristics. They also completed time–activity diaries that provided information about activities such as cooking and housecleaning and about time spent in six microenvironments (indoors at home; indoors at work or school; indoors away from home, work, or school; outdoors near home; outdoors away from home; and in transit).

STATISTICAL ANALYSIS

Ambient and personal pollutant levels were characterized by using summary statistics (means, medians, standard deviations), graphic analyses (box-and-whisker plots), and analysis of variance. Measurements below the limit of detection (LOD), including negative values, were included in all analyses. For pollutants for which the exposure distribution appeared nonnormal, Spearman correlation coefficients (r_s) were used. Mixed-model regression analysis was used to evaluate the associations between the concentrations of ambient $PM_{2.5}$ and ambient gaseous pollutants, ambient pollutants and their corresponding personal exposure concentrations, ambient gaseous pollutants (and SO_4^{2-}) and personal $PM_{2.5}$, and personal $PM_{2.5}$ (including SO_4^{2-} as a surrogate for personal $PM_{2.5}$ of ambient origin) and personal gaseous exposures.

RESULTS AND INTERPRETATION

Data Quality

Investigators included a quality-control plan in their study, including sealed (unexposed) monitors that traveled between the laboratory and the field to serve as field blanks (approximately 10% of the total samples collected) and collocated monitors at each stationary monitoring site to allow determination of precision and bias in the laboratory assays. Method LODs were estimated in two ways: as 3 times the standard deviation of the field blanks and as 3 times the absolute precision of duplicate samples.

Ambient $PM_{2.5}$ Concentrations at Central Monitoring Sites Are Appropriate Surrogates of Personal Exposures to $PM_{2.5}$ (Hypothesis 1)

Mixed-model regression analyses were used to explore the contribution of ambient $PM_{2.5}$ to the corresponding personal exposures to $PM_{2.5}$ (described in this report as the *infiltration factor*). The models took advantage of the repeated measurements taken at each central monitoring site and by each PEM. Model slopes were calculated for each city, season, and cohort to estimate the infiltration factor. The model intercept for each city–season group indicates the nonambient source contributions to personal exposure.

Ambient concentrations of $PM_{2.5}$ were strongly associated with personal $PM_{2.5}$ exposures in all groups, with some differences by season and city. Slopes ranged from 0.23 to 0.81; that all slopes were less than 1.00 indicates that participants were exposed to average $PM_{2.5}$ concentrations lower than the corresponding ambient concentrations. Ambient SO_4^{2-} level and personal exposure to SO_4^{2-} were significantly correlated, with a stronger correlation in Boston in both seasons. During each sampling season (especially winter) in both cities, measurements of personal exposure to O_3 and SO_2 were generally low, often below LODs.

In general, personal exposures to O_3 and SO_2 tended to be lower than the corresponding ambient pollutant levels. The resulting associations between ambient concentrations of SO_2 and personal exposure to SO_2 were weak, which was expected because SO_2 is a reactive chemical and is thus not likely to have a large infiltration factor. The associations between ambient levels of and personal exposures to O_3 and NO_2 were strong in Boston but less strong in Baltimore.

Some activity patterns and housing characteristics may have influenced personal exposures to $PM_{2.5}$. Cooking and cleaning activities had a lesser effect on $PM_{2.5}$ levels for seniors than expected, which could reflect the low frequency of those activities in this cohort. This would be especially true for the Baltimore participants because COPD patients are expected to be more sedentary than healthy seniors. The influence of living near a busy roadway was variable. Exposure to $PM_{2.5}$ was greater near a busy roadway than far from one for Baltimore children in winter and for Baltimore seniors and COPD patients in summer. For the other participants, personal exposures to $PM_{2.5}$ were lower close to a busy roadway.

Associations Between Personal Exposures to $PM_{2.5}$ and Ambient Concentrations of $PM_{2.5}$ Vary by City, Season, and Group (Hypothesis 2)

Participants from all groups and both cities were mostly indoors during the sampling periods, spending only 2.1% to 11.3% outdoors (Table 5 of Investigators' Report). Some differences were found between cities, seasons, and cohorts: children and seniors in Boston spent less time outdoors in winter (2.1% and 3.3%, respectively) than in summer (11.3% and 7.2%, respectively); Baltimore children spent less time outdoors in summer (5.5%) than the Boston children did.

Median personal $PM_{2.5}$ exposures for all groups were higher in Baltimore (20.1 $\mu g/m^3$) than in Boston (9.6 $\mu g/m^3$), with no significant differences among cohorts in either city. In Baltimore, personal $PM_{2.5}$ exposures tended to be comparable to, or even lower than, corresponding ambient levels, whereas in Boston, personal $PM_{2.5}$ exposures tended to be higher than corresponding ambient levels. Personal EC exposures were similar in winter in the two cities and were comparable to ambient EC levels (available only in Boston). In general, personal EC exposures were low, with many individual values below the LOD.

The strongest association between ambient $PM_{2.5}$ concentrations and personal $PM_{2.5}$ exposures occurred in summer, with a stronger correlation in Boston (slope = 0.81) than in

Baltimore (slope = 0.55). The weakest correlation was between ambient $PM_{2.5}$ concentrations and personal $PM_{2.5}$ exposures in winter in both cities (slope = 0.23 for each city). Although infiltration factors were generally greater for children (summer and winter) than for other cohorts, indicating a greater association of ambient $PM_{2.5}$ level and personal $PM_{2.5}$ exposure for that group, these differences were not statistically significant. Individuals with COPD showed the weakest association between ambient $PM_{2.5}$ level and personal $PM_{2.5}$ exposure (slope = 0.15).

Median personal SO_4^{2-} levels were higher in summer than in winter and highest in summer in Baltimore, reflecting trends in ambient SO_4^{2-} concentrations. SO_4^{2-} infiltration factors did not differ by group but did differ by season and to some extent by city. Participants in Boston had higher personal exposures to SO_4^{2-} than did participants in Baltimore, even though ambient SO_4^{2-} levels were higher in Baltimore.

Personal exposures to NO_2 were different in the two cities, with Boston children (median, 19.2 and 26.3 ppb in winter and summer) and seniors (12.8 and 13.6 ppb) generally experiencing higher exposures than Baltimore children (13.7 and 12.0 ppb) and seniors (14.6 and 7.1 ppb). Children in Boston had higher personal NO_2 exposures in both seasons than seniors in the same city did; seniors' personal NO_2 exposures were lower than corresponding ambient levels. Boston children had higher-than-ambient personal exposures to NO_2 in summer but lower-than-ambient exposures in winter. Personal exposure to NO_2 was lower than ambient levels in both seasons for all other groups. These differences among groups may reflect different numbers of homes with gas stoves in the two cities. In Boston, the majority of children (87% of the summer cohort and 67% of the winter cohort) and the minority of seniors (13% of the summer cohort and 33% of the winter cohort) lived in homes with gas stoves. In Baltimore, 60% of the children, 53% of the seniors, and 60% of the COPD patients in the winter cohort and 67% of the children and 33% of the seniors in the summer cohort lived in homes with gas stoves.

Ambient Concentrations of Gaseous Copollutants Correlate More Strongly with Personal Exposures to $PM_{2.5}$ Than with Personal Exposures to Gaseous Copollutants (Hypothesis 3)

Some pollutants, including $PM_{2.5}$, originate from both indoor and outdoor sources; others, such as SO_4^{2-}, originate primarily from outdoor sources. Koutrakis and colleagues therefore examined cross-pollutant correlations to determine if one or more gaseous copollutants could predict personal $PM_{2.5}$ exposures. They found significant associations between ambient concentrations of $PM_{2.5}$ and ambient concentrations of copollutants in each city, although the degree of association varied by season and city. Ambient SO_4^{2-} concentrations were significantly associated with personal exposure to SO_4^{2-}. Likewise, ambient concentrations of $PM_{2.5}$ were significantly associated with personal exposures to $PM_{2.5}$, despite the known presence of indoor sources of $PM_{2.5}$.

Ambient–personal $PM_{2.5}$ associations were weaker than the SO_4^{2-} associations, reflecting the lack of indoor sources for SO_4^{2-}. Ambient O_3, NO_2, and SO_2 concentrations were significantly associated with personal $PM_{2.5}$ exposures in Baltimore in both seasons but in Boston only in summer. However, the high LODs for these pollutants and the high percentage of samples with values below those LODs make these latter comparisons difficult to interpret.

DISCUSSION

This is a strong study, carefully thought out and successfully executed. It is one of few field studies that have addressed an important question for understanding exposures to $PM_{2.5}$: How well do measurements of $PM_{2.5}$ (and other pollutants) taken at central monitoring sites reflect personal exposure to $PM_{2.5}$?

OVERALL RESULTS

The results indicate that the relation between ambient $PM_{2.5}$ levels and personal exposures to $PM_{2.5}$ varies by season, location, and home characteristics. Surprisingly, groups did not appear to differ in their exposures to the fraction of ambient $PM_{2.5}$ that contributes to personal $PM_{2.5}$, despite the presence of indoor $PM_{2.5}$ sources.

The investigators found that ambient concentrations of SO_4^{2-}, a component of $PM_{2.5}$, were strongly associated with personal exposures to SO_4^{2-} and also with personal exposures to $PM_{2.5}$. They concluded that, for a given season or city, ambient $PM_{2.5}$ and SO_4^{2-} are strong predictors of respective personal exposures and that ambient SO_4^{2-} is also a strong predictor of personal exposure to $PM_{2.5}$. Because $PM_{2.5}$ has substantial indoor sources and SO_4^{2-} does not, the investigators reasonably concluded that personal exposure to SO_4^{2-} accurately reflects exposure to ambient $PM_{2.5}$ and therefore the ambient component of personal exposure to $PM_{2.5}$ as well. This conclusion is tempered, however, by the different techniques used to measure ambient concentrations and personal exposures to $PM_{2.5}$ and SO_4^{2-}. How these differences may influence the findings is unclear.

Ambient $PM_{2.5}$ (and corresponding SO_4^{2-}) samples were collected by using Harvard impactors at central sites, whereas personal $PM_{2.5}$ exposures were assessed by using PEMs. Personal exposure monitors have been shown to overestimate PM mass by about 20% compared with Harvard impactors (Geyh et al 2004), which may explain the similar levels of indoor and ambient PM measured in this study. In contrast, most other studies have found levels of personal exposure to $PM_{2.5}$ to be higher than ambient levels (Clayton et al 1993; Thomas et al 1993; Ozkaynak et al 1996; Weisel et al 2005). Ambient SO_4^{2-} was assessed from material collected using different monitors in the two cities: Harvard impactors in Baltimore and HEADs in Boston. Conclusions about SO_4^{2-} collected by using different methods are limited without directly comparing the collection systems or each system against a common reference method.

A few other longitudinal studies have evaluated correlations between personal exposures and indoor and outdoor pollutant concentrations for each subject separately, with a focus on sensitive populations (eg, children and individuals with COPD). In these studies, correlations between personal exposure and ambient PM were strong (Janssen et al 1997, 1998, 1999; Ebelt et al 2000; Williams et al 2000; Liu et al 2003).

For some gaseous pollutants measured in the Koutrakis study, most measurements were below the LOD (see Table 3 of the Investigators' Report). Particularly problematic were O_3 and SO_2 measurements, more than 90% of which were below the LOD. Although in Baltimore 80% of NO_2 measurements were below the LOD in winter and 39% were below the LOD in summer, in Boston only 2% and 10% of the measurements of NO_2 were below the LOD in winter and summer, respectively.

The high percentage of nonmeasurable personal exposure data for gaseous pollutants may have precluded the opportunity to observe a strong correlation with ambient gases. Using more sensitive techniques for measuring personal gaseous pollutants may help to discern whether a correlation truly exists between ambient and personal data. Nevertheless, even with more sensitive measurements, the variability of removal mechanisms for gaseous pollutants in indoor environments may continue to result in weak correlations between ambient and personal gaseous data, since a major component of personal air includes indoor air.

Some pollution concentration data that were corrected for field-blank values were negative, a consequence of analyzing the measurements that were below the LOD and subtracting the value of the field blank, which was higher than that of the pollutant. The investigators chose to analyze those measurements in order to minimize bias. These analyses may therefore have been influenced by random noise from the measurement methods.

How to deal with values that are below the LOD is an important issue that could substantially affect the results of any analysis. It is a particular concern with the NO_2 passive monitor, for which the LOD was high and varied by season and site (eg, 11.7 ppb and 5.7 ppb, respectively, in winter and summer for Baltimore; Investigators' Report Table 2). Although the investigators handled the values below the LOD by using currently accepted methods, care should be taken in interpreting results with many such values. The conclusion that ambient concentrations of gaseous pollutants serve as a better surrogate for personal exposure to $PM_{2.5}$ than for personal exposure to gaseous pollutants is weakened by the high percentage of gaseous pollutant samples that were below the LOD in this study. In contrast, most measurements of $PM_{2.5}$ and SO_4^{2-} were above the LOD, giving more confidence to analyses that depend on them.

The conclusions of this study should be extrapolated with caution because the study was performed in only two cities and with select subpopulations. The authors note the limitations of their nonrandom recruitment strategy; their findings may or may not be relevant to the general population or to randomly selected, sensitive subpopulations from cities across the United States. Confirmation of results with other groups from other cities would be useful.

The results indicate that the children studied in both cities did not differ much in the time they spent outdoors compared with seniors and COPD patients. This conclusion may reflect the fact that the children were recruited from local community centers and a science education program; these children may be less active or spend less time outdoors than most and therefore may not be representative of physically active children.

The location of the central ambient monitoring sites, and the distribution of pollution sources, may have influenced the results of the comparisons between cities. This influence is likely for the gaseous pollutants, particularly NO_2 and carbon monoxide, the ambient concentrations of which have large spatial variability and are influenced by factors such as proximity to traffic and traffic volume (Brauer et al 2003; Gilbert et al 2005). Differences in pollution levels between the two cities may be caused more by the presence of local sources near central monitoring sites than by actual differences in pollution.

Furthermore, data from two central sites were used to assess ambient exposures in Baltimore, but only one site was used in Boston. Additional information about site-to-site variations in the data would have been useful. In addition, the Baltimore sites were set up and run by the state of Maryland for the purpose of assessing compliance with air pollution standards, whereas the Boston site was set up for

this study. We do not know how these measurements at these sites are related to those taken at the EPA sites used in many epidemiologic studies.

The results of this study were similar to those of another study funded by this research program (Brunekreef et al 2005). Brunekreef and his colleagues assessed the correlations between pairs of personal, indoor, and outdoor concentrations of $PM_{2.5}$ for two groups of seniors with cardiovascular disease living in Amsterdam, the Netherlands, and in Helsinki, Finland. Outdoor concentrations of pollutants were measured at fixed monitoring sites near the participants' residences and were measured inside residences by using Harvard impactors. Personal exposures were measured by using cyclone samplers.

These two studies share design features yet also differ. Both examined multiple cities in multiple seasons. Both investigated possibly sensitive populations, although the specific populations differed. Both involved set-up of new central monitors to assess ambient concentrations of PM. One study measured gaseous copollutants and SO_4^{2-}; the other measured trace metals. One measured indoor air; the other did not. One included homes where residents smoke; the other excluded smokers (although the homes may not have been free of environmental tobacco smoke). Despite these differences, both investigator groups found that ambient concentrations of PM are strongly associated with personal exposure to PM, even in populations that spend much of their time indoors.

STATISTICAL ANALYSIS

Estimated variance components in the Koutrakis study show that there was possibly important, unexplained variation between participants and within cities. The authors used the correlation coefficient to quantify the association between different measures of exposure. Because this metric is unit free, it does not readily convey the absolute magnitude of covariation or prediction error. Therefore, supplementing the correlation coefficient with some other metric, such as the root mean squared error of prediction, would have been useful.

The repeated measurements in the study provided sufficient information to estimate both between-individual variation and within-individual autocorrelation (and autocovariation) of exposure measurements. Taking advantage of such autocorrelation can substantially improve prediction accuracy, can be used to impute missing exposure values, and can be a key input for designing the next study. For example, it can be used to quantify information tradeoffs between number of participants and number or frequency of exposure assessments per participant.

The sensitivity analyses in this study were informative, and the authors provide a good discussion of the scientific implications of point estimates and confidence intervals. However, in addition to examining *P* values, an analysis of the magnitude of estimated effects and their plausible ranges (their confidence intervals) would have been useful.

SUMMARY AND CONCLUSIONS

This study provides important information for understanding the relation between ambient measures of urban air pollution and personal exposures to the same pollutants. Ambient concentrations and personal exposures to a variety of urban air pollutants were measured in two cities, in two seasons, and in three groups of possibly susceptible individuals. The study design involved examination of important personal–ambient exposure issues for both $PM_{2.5}$ and gaseous air pollutants: differences by city, season, and sensitive subpopulations, as well as by day of the week. The study design was high quality, with standardized measurements taken at consistent times of day. However, although investigators employed appropriate and state-of-the-art methods to monitor both personal and ambient concentrations of pollutants, some of these methods differed between cities or between measurements of ambient concentrations and personal exposures. They collected information about home characteristics and time–activity diaries for participants. Although the study design did not include indoor pollutant monitoring, complete chemical speciation of particles, or source apportionment, it did include collection of valuable information to better understand the use of ambient air pollution concentrations obtained from central monitors in the evaluation of health effects.

The key results and implications of the study are:

1. Ambient concentrations of $PM_{2.5}$ were strongly associated with personal exposure to $PM_{2.5}$, supporting the use, in epidemiologic studies, of exposure information for $PM_{2.5}$ from central monitoring sites.
2. The strength of the associations between personal exposures to $PM_{2.5}$ and ambient concentrations of $PM_{2.5}$ appeared to vary somewhat by city and season, as investigators hypothesized. In contrast, the variation among groups was not as great as was anticipated. The HEI Health Review Committee agreed that studying groups thought to be sensitive is valuable. The Committee cautioned, however, that results of this study should not be extended generally to other locations and populations, given that the groups studied did not represent the general population or even the subpopulations of which they are a part.
3. For these data, ambient concentrations of gaseous copollutants such as O_3, NO_2, and SO_2 correlated more

strongly with measures of personal exposure to $PM_{2.5}$ than with personal measures of the same gaseous pollutants. On the basis of these results, the investigators recommended caution when interpreting results from previous time-series studies that included both gaseous and particulate pollutant concentrations. This caution is noteworthy. The high percentage of nonmeasurable data in this study precludes the ability to discern whether strong correlations actually do exist between ambient and personal gaseous data. On the other hand, even with a more sensitive measurement technique, the correlations observed may still be weak because of the variability in removal mechanisms for gases in indoor spaces.

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