Exposure Assessment of Particulate Matter for Susceptible Populations in Seattle

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In this article we present results from a 2-year comprehensive exposure assessment study that examined the particulate matter (PM) exposures and health effects in 108 individuals with and without chronic obstructive pulmonary disease (COPD), coronary heart disease (CHD), and asthma. The average personal exposures to PM with aerodynamic diameters < 2.5 µm (PM2.5) were similar to the average outdoor PM2.5 concentrations but significantly higher than the average indoor concentrations. Personal PM2.5 exposures in our study groups were lower than those reported in other panel studies of susceptible populations. Indoor and outdoor PM2.5, PM10 (PM with aerodynamic diameters < 10 µm), and the ratio of PM2.5 to PM10 were significantly higher during the heating season. The increase in outdoor PM10 in winter was primarily due to an increase in the PM2.5 fraction. A similar seasonal variation was found for personal PM2.5. The high-risk subjects in our study engaged in an equal amount of dust-generating activities compared with the healthy elderly subjects. The children in the study experienced the highest indoor PM2.5 and PM10 concentrations. Personal PM2.5 exposures varied by study group, with elderly healthy and CHD subjects having the lowest exposures and asthmatic children having the highest exposures. Within study groups, the PM2.5 exposure varied depending on residence because of different particle infiltration efficiencies. Although we found a wide range of longitudinal correlations between central-site and personal PM2.5 measurements, the longitudinal r is closely related to the particle infiltration efficiency. PM2.5 exposures among the COPD and CHD subjects can be predicted with relatively good power with a microenvironmental model composed of three microenvironments. The prediction power is the lowest for the asthmatic children. Key words: asthma, CHD, COPD, infiltration efficiency, longitudinal correlation, personal cloud, PM2.5, wood smoke, Environ Health Perspect 111:909–918 (2003). doi:10.1289/ehp.6011 available via http://dx.doi.org/[Online 4 February 2003]

Many epidemiologic studies have reported associations between daily morbidity and mortality and daily particulate matter (PM) air pollution concentrations [U.S. Environmental Protection Agency (EPA) 2001]. Most of these studies have relied on central-site PM monitors for information on concentration of PM and other pollutants. Effects have been seen with all size ranges of PM, from total suspended particulates [PM with aerodynamic diameters ≤ 10 µm (PM10) or < 2.5 µm (PM2.5)] to ultrafine particles (aerodynamic diameters < 0.1 µm). Several studies indicate that PM2.5 may be more strongly associated with some adverse health endpoints than are the larger size fractions (Katsouyanni et al. 1997; Schwartz et al. 1996; Schwartz and Neas 2000; Woodruff et al. 1997), although other studies suggest that coarse particles are more closely associated with asthma (Lin et al. 2002; Zhang et al. 2002). Premature mortality is usually found in individuals with preexisting cardiopulmonary disease (Goldberg et al. 2001; Samet et al. 2000; Schwartz 2000; Sunyer et al. 2000). Morbidity (measured as hospital admissions, lung function decrements, airway inflammation, respiratory symptoms or medication use, or cardiac dysfunction) is found in individuals with chronic obstructive pulmonary disease (COPD) or heart disease. Children with asthma appear to be more susceptible than adults to air pollution–induced aggravation (Koenig 1999). Despite the wealth of data supporting associations between health outcomes and PM exposures, there are many gaps in our knowledge.

One concern is whether the particle concentration measured at an outdoor monitoring site is, in fact, related to the exposure of people in the community. This concern has been the focus of several panel studies in susceptible subpopulations (Ebel et al. 2000; Evans et al. 2000; Jansen et al. 2000; Rodes et al. 2001; Rojas-Bracho et al. 2000; Williams et al. 2000a, 2000b). More accurate assessment of exposure to PM, particularly among individuals shown to be susceptible to PM exposure in epidemiologic studies, is a crucial research need (e.g., Moolgavkar et al. 1997; National Research Council 2001; Reichhardt 1995; Styer et al. 1995).

This Seattle panel study is one of four panel studies of high-risk subpopulations sponsored by the U.S. Environmental Protection Agency (EPA) that monitored PM and related air pollutants. In these panel studies, groups of subjects were monitored and followed for several seasons to characterize their exposure to PM. Our study included four susceptible study groups: elderly persons who a) were healthy, b) had COPD, or c) had coronary heart disease (CHD), and d) children with asthma. We collected personal, indoor, and outdoor samples for PM10, PM2.5, carbon monoxide, sulfur dioxide, and nitrogen dioxide during seasons with high and low wood smoke (1999–2001). In this article we focus on characterizing the PM exposure among these four study groups.

Study Design

Subjects and monitoring sites. We recruited elderly subjects through distribution of flyers throughout the community at such sites as clinics, senior centers, and retirement homes. Children with asthma were recruited from one large asthma and allergy clinic. Our panel included 34 with COPD, 27 with CHD, 28 without any signs or symptoms of cardiopulmonary disease (healthy), all elderly, and 19 children with asthma. These subjects were volunteers and were not selected using probability-based sampling; therefore, our results cannot be extrapolated to larger populations. All but one of the elderly subjects were more than 65 years of age; 85% were between 71 and 90 years of age. The children were between 6 and 13 years of age. About 55% of these subjects were reenrolled for monitoring in different seasons within a year. All COPD subjects had physician-diagnosed COPD and had a forced expiratory volume in the first second (FEV1) between 40% and 70% predicted value. All CHD subjects had a history of heart disease. The authors declare they have no conflict of interest. Received 20 September 2002; accepted 3 February 2003.
of myocardial infarction, angina, or congestive heart failure. All asthmatic children had physician-diagnosed mild to moderate asthma and had intermittent use of rescue medication (albuterol). All subjects were nonsmokers living with nonsmokers, and they usually spent more than 30 min a day outdoors. Most of the COPD and healthy subjects lived in either group homes or private residences. Most of the cardiac subjects lived in private homes or apartments. All but one of the children lived in private homes.

**Monitoring period.** This study was conducted in 26 monitoring sessions, including 13 sessions in each monitoring year: Year 1 (October 1999–August 2000) and Year 2 (September 2000–May 2001) (Table 1). Each session consisted of 10 consecutive monitoring days, starting at 1600 hr (± 2 hr) on Tuesdays and ending at 1600 hr (± 2 hr) on Fridays. Up to nine subjects (mean ± SD, 6 ± 2) per session were monitored simultaneously. The average temperature, relative humidity, and wind speed were slightly higher in Year 1 (temperature = 9.9 ± 4.6°C; relative humidity = 79.3 ± 8.4%; wind speed = 5.5 ± 0.9 m/sec) than in Year 2 (temperature = 7.9 ± 4.2°C; relative humidity = 78.7 ± 10.6%; wind speed = 4.7 ± 1.9 m/sec), whereas the daily average hours of stagnation (wind speed < 1.8 m/sec) was higher in Year 2 (11.6 ± 6.2 hr) than in Year 1 (8.8 ± 6.2 hr).

**Personal monitoring.** Personal PM2.5 exposures were determined using the Harvard Personal Environmental Monitor for PM2.5 (HPEM2.5; Harvard School of Public Health, Boston, MA). The small HPEM2.5 is a single-stage inertial impactor with a 50% cut point of 2.4 ± 0.1 µm (Sioutas et al. 1999). The HPEM1.5 was connected to a personal pump (AFC 400S; BGI, Inc., Waltham, MA) with a mass flow controller operating at 4 L/min. Particles > 2.5 µm in diameter were originally collected on a porous metal impaction plate coated with silicon oil immediately downstream of the inlet; particles < 2.5 µm bypassed the impaction plate and were collected on a 37-mm Teflon filter (polytetrafluoroethylene with support ring, model 225-1709; SKC, Inc., Eighty Four, PA). Because of an oil contamination problem, the entire porous metal plate was replaced with silicon vacuum grease after the first four sessions (Demokritou et al. 2001).

Each subject carried an HPEM2.5 in the breathing zone for 24 hr, except while sleeping, showering, or using the restroom. The monitor was attached to the shoulder strap of either a backpack or a fanny pack that contained the air pump. When the monitor was not worn, it was placed at an elevation of 3–5 feet (e.g., on a table) close to the subjects. Our field technicians visited the subjects daily to calibrate the pumps with a digital piston flow meter (Drycal, DC-Lite; SKC Inc.), and later with a rotameter (model 92-04; Cole-Parmer Instrument Co., Vernon Hills, IL), and to record on and off flow rates and change samplers.

**Fixed-site monitoring.** The indoor and outdoor PM concentrations were measured with single-stage inertial Harvard Impactors (HI) (Air Diagnostics and Engineering, Inc., Naples, ME) and 37-mm Teflon filters for PM10 and PM2.5 (Marple et al. 1987). One HI2.5–HI10 pair was located inside each home in the main activity room and connected to a Medo pump (model vp0935A; Medo USA, Inc., Hanover Park, IL). Concurrently, one HI2.5–HI10 pair was located outside each home and connected to a Gast pump (model DOA-V191-AA; Gast Manufacturing, Inc., Benton Harbor, MI). The on and off flow rates were calibrated and recorded daily with the flow meter and later with a Cole-Parmer rotameter (model 34-39). All HI sampling periods were for 24 hr at a flow rate of 10 L/min. HI2.5, HI10, and HPEM2.5 were also collocated with the federal reference method HI2.5 (FRM2.5) at the central Beacon Hill site, which is located in a semi-identical area (elevation, 300 feet) and is maintained by the Washington State Department of Ecology. This site has been validated as representative of the regional air quality in urban Seattle (Gowaswi et al. 2002). Duplicate sets of central-site HPEM2.5, HI2.5, and HI10 were running at the same schedule as those at home sites (1600 hr to 1600 hr) for estimating precision. One central-site HI2.5–HPEM2.5 pair ran from midnight to midnight to coincide with the FRM2.5 measurements.

**Filter analysis.** All filter weights were measured in either duplicate or triplicate using a seven-place electronic ultramicrobalance (model UMT2; Mettler Toledo, Greifensee, Switzerland). The filters were equilibrated for at least 24 hr before weighing. Both equilibration and weighing were performed inside a controlled environmental chamber with constant relative humidity (34.7 ± 2.5%) and temperature (22.4 ± 1.9°C) (Allen et al. 2001). Before weighing, the filters were passed between two polonium-210 strips (500 µCi) to eliminate any electrostatic charge on the filter. Each day, before the weighing sessions, the microbalance was calibrated internally and externally with four certified stainless steel weights (20, 50, 100, and 200 mg) to further validate the internal calibration.

**Other information.** At the beginning of each sampling session, technicians gathered information on the dwelling (apartment, home, etc.), proximity to a busy roadway, type of parking garage, and type of heating (forced air, radiator, fireplace, etc.). During the study, each subject kept a diary of activity, location, and location with a 15-min resolution. The diary provided sufficient room to specify minutes used for each activity if more than one activity was conducted within the 15-min interval. In addition, technicians recorded occurrence of events that would potentially affect PM concentrations at homes, including window opening, type of cooking, incense burning, and house cleaning.

**Table 1. Number of subjects by study group and session in the Seattle panel.**

| Year | Starting date | Session | Asthmatics | CHD | COPD | Healthy | Total |
|------|---------------|---------|------------|-----|------|---------|-------|
| 1999 | October 26    | 1       | 0          | 5   | 5    | 3       | 8     |
|      | November 8    | 2       | 0          | 5   | 5    | 4       | 9     |
|      | November 29   | 3       | 0          | 5   | 5    | 3       | 8     |
| 2000 | January 10    | 4       | 0          | 3   | 3    | 6       | 9     |
|      | February 7    | 5       | 1          | 2   | 3    | 4       | 6     |
|      | February 21   | 6       | 0          | 3   | 3    | 6       | 6     |
|      | March 6       | 7       | 1          | 3   | 3    | 7       | 7     |
|      | March 27      | 8       | 0          | 4   | 4    | 5       | 6     |
|      | April 10      | 9       | 0          | 3   | 2    | 5       | 8     |
|      | May 1         | 10      | 0          | 5   | 5    | 3       | 8     |
|      | May 15        | 11      | 0          | 4   | 2    | 7       | 4     |
|      | July 10       | 12      | 1          | 4   | 2    | 7       | 4     |
|      | July 31       | 13      | 1          | 1   | 2    | 4       | 4     |
|      | September 25  | 1       | 2          | 4   | 5    | 6       | 6     |
|      | October 18    | 2       | 3          | 5    | 8    | 6       | 6     |
|      | November 6    | 3       | 6          | 6    | 6    | 6       | 6     |
|      | November 27   | 4       | 2          | 4    | 6    | 6       | 6     |
|      | December 25   | 5       | 4          | 1    | 6    | 5       | 5     |
| 2001 | January 8     | 6       | 5          | 1    | 6    | 6       | 6     |
|      | January 22    | 7       | 3          | 3    | 6    | 6       | 6     |
|      | February 5    | 8       | 2          | 5    | 6    | 6       | 6     |
|      | February 26   | 9       | 4          | 4    | 6    | 6       | 6     |
|      | March 10      | 10      | 5          | 3    | 8    | 6       | 6     |
|      | April 16      | 11      | 3          | 2    | 8    | 5       | 5     |
|      | April 30      | 12      | 4          | 1    | 6    | 5       | 5     |
|      | May 14        | 13      | 1          | 3    | 8    | 4       | 4     |
|      | Total         | 33      | 40         | 56   | 38   | 167     |       |

About 50% of the subjects were monitored twice.
among others. These questionnaires were developed especially for the four panel studies sponsored by the U.S. EPA and were approved by its Office of Management and Budget.

Data reduction. All data were examined for irregularity and noncompliance with our standard operating procedures. Samples were flagged and removed when the flow rates fell outside 10% of the designated flow rate. Most flagged samples, including 4.6% of HI samples and 9% of HPEM samples, were due to pump or battery failure, broken filters, or disconnected tubing.

Results

Quality control. The total number of field blanks was between 10% and 26% of the total sample size. The limit of detection (LOD) was 1 µg/m³; for the 24-hr integrated HI was 1 µg/m³; for the 24-hr integrated HPEM2.5, the LOD was 6.2 µg/m³ for the first four sessions and 4.5 µg/m³ afterward.

This reduction was achieved by replacing the oil porous impaction plate with vacuum grease to reduce contamination from silicon oil (Demokritou et al. 2001), and adding a drain disk downstream of the Teflon filter. This 4.5 µg/m³ LOD for HPEM2.5 is similar to values (2.6–4.0 µg/m³) reported by Sarnat et al. (2000). The total number of field duplicates ranged between 18% and 29% of total sample size. All duplicates were highly correlated with each other, with a Pearson’s r of ≥ 0.96. The mean difference between the duplicates was not significantly different from zero. The precision, calculated as the standard deviation of duplicate differences divided by the square root of 2, was 1.2 µg/m³ for HI and 2.2 µg/m³ for HPEM2.5.

The accuracy of our PM2.5 measurements was calculated by comparing them with the collocated FRM2.5 measurements at the central site (Figure 1A). We also collocated HI2.5 and HPEM2.5 whenever possible: 77 pairs at the stationary ambient monitoring sites and 17 pairs at subjects’ homes (Figure 1B).

The Pearson’s r between samplers was ≥ 0.93. There is a positive bias (7.7 µg/m³; p < 0.001) for HPEM2.5 with an oiled impaction plate; with a greased impaction plate, the bias is negligible (0.4 µg/m³; p = 0.08). All HI and HPEM measurements were corrected for average blank values. The HPEM2.5 measurements with oiled impaction plates during the first four monitoring sessions (n = 269 out of 1,347 personal filters) were removed from the following analysis because of the oil contamination problem.

Summary statistics. Table 2 summarizes concentrations of PM2.5 and PM10 for the four study groups and four microenvironments. Because all measurements are skewed to the right, geometric means (GMS) and geometric standard deviations (GSDs) are reported along with the arithmetic means. Although for all study groups the mean personal exposures and indoor and outdoor concentrations of PM2.5 were below the new National Ambient Air Quality Standard (NAAQS) (U.S. EPA 2001) for the annual PM2.5 average (15 µg/m³), individual 10-day exposures exceeded the annual NAAQS for 12% of the elderly and 42% of the child subjects. The average indoor PM2.5 levels were < 10 µg/m³, whereas average indoor PM10 levels were between 10 and 20 µg/m³. Personal PM2.5 concentrations were similar to outdoor PM2.5 concentrations (mean difference ± SD = 0.3 ± 8.3; p = 0.29, paired t-test) but significantly higher than indoor concentrations (p < 0.0001). Indoor PM2.5 concentrations were significantly lower than those outdoors (p < 0.0001). The difference between PM10 and PM2.5 measurements (coarse particles, PM2.5–10) was approximately 5 µg/m³ for both indoor and outdoor environments for all study groups, except inside asthmatic children’s residences, where the mean PM2.5–10 was double (10.2 µg/m³).

PM2.5 was on average 61% of the PM10 mass both indoors and outdoors in Year 1. In Year 2, when more homes were located in wood-smoke–affected neighborhoods (Larson et al. 1989), the mean home outdoor and indoor PM2.5:PM10 ratios were significantly higher (p < 0.001), whereas the central-site PM2.5:PM10 ratio remained the same across years (Table 3). Both the indoor and outdoor PM2.5:PM10 ratios were significantly higher (p < 0.001) during the heating season (October through February), when wood smoke was dominant (Maykut et al. 2001), with home outdoor PM2.5 accounting for 70% of the outdoor PM10 mass.

Figure 2A shows PM2.5 measurements obtained from same subjects and locations in both the heating and nonheating seasons. A significant seasonal effect was detected for all locations (p < 0.0001, paired t-test), with the outdoor locations showing the most prominent seasonal effect. Higher variability in
PM$_{2.5}$ measurements was observed at fixed locations (indoor, outdoor, and the central site) during the heating season than during the nonheating season. In contrast, the variability in personal PM$_{2.5}$ measurements was similar during both seasons. PM$_{10}$ concentrations also elevated during the heating season ($p < 0.01$) for all locations (Figure 2B). For outdoor particles, the increase in PM$_{10}$ during the heating season was accompanied by a significant increase in the coarse fraction ($p < 0.0001$). For indoor particles, the increase in PM$_{10}$ was entirely due to the increase in the fine fraction because the PM$_{2.5-10}$ levels were identical in both seasons ($p = 0.25$).

Relationships among measurements. Figure 3A shows the cumulative probability plots of PM$_{2.5}$ measurements at four microenvironments with indoor PM$_{2.5}$ consistently lower than personal and outdoor PM$_{2.5}$. The personal median is about the same as that of the outdoor or central-site measurements. For PM$_{10}$, the home indoor and outdoor measurements are very similar above the 50th percentile (Figure 3B). Above the 90th percentile, indoor PM$_{10}$ often exceeded outdoor PM$_{10}$. The highest correlation for the PM$_{2.5}$ measurements was between the home outdoor and central-site measurements ($r = 0.84$) (Table 4). Personal PM$_{2.5}$ correlated best with indoor PM$_{2.5}$ ($r = 0.65$) and less so with outdoor and central-site PM$_{2.5}$. Indoor PM correlated with both outdoor and central-site PM, with higher correlations found for PM$_{2.5}$ than for PM$_{10}$. Outdoor PM$_{10}$ and PM$_{2.5}$ showed comparable correlations between sites (0.82 vs. 0.84) because the majority of the PM$_{10}$ consists of PM$_{2.5}$. All PM$_{2.5}$ measurements are highly correlated with the collocated PM$_{10}$ measurements, again indicating a predominant portion of PM$_{2.5}$ in PM$_{10}$.

The longitudinal (Pearson’s) correlation between personal PM$_{2.5}$ exposures and central-site measurements for each subject, calculated over the 10 consecutive 24-hr monitoring days and for at least six valid pairs of measurements, ranged between −0.57 and 0.98 (Figure 4A), with a median of 0.34. One issue in presenting such longitudinal correlations is the limited number of observations per subject and thus an overly broad distribution compared with the true distribution, particularly at the low end. The shrunk correlation estimates (Lumley T, Liu L-JS. Unpublished data), by modifying slightly the upper end and significantly the lower end of the crude correlations by an appropriate amount (Figure 4B), give a more representative underlying distribution. The shrunk $r$ estimates ranged between 0.10 and 0.82, with a median of 0.43. The correlation between the crude and shrunk correlations is 0.98. There were no significant differences among study groups in either the crude or shrunk longitudinal $r$ ($p = 0.43$).

We used analysis of variance (ANOVA) to examine factors that may affect the crude longitudinal $r$, including age, sex, activity pattern (e.g., time spent outdoors), home type, and the estimated particle infiltration efficiency ($F_{inf}$) (Allen R, Larson T, Wallace L, Liu L-JS. Unpublished data). $F_{inf}$, a unitless quantity defined as the equilibrium fraction of ambient PM that penetrates indoors and remains suspended (Wilson et al. 2000), is one of the most important parameters for estimating personal exposure to ambient PM. It is a function of air exchange rate ($a$), particle penetration ($p$), and particle decay rate ($k$):

$$F_{inf} = \frac{pa}{a + k}. \quad [1]$$

### Table 3. Ratio of PM$_{2.5}$ to PM$_{10}$, stratified by year or heating season.

| Group          | Location | No. | Mean ± SD | Min | Max  |
|----------------|----------|-----|-----------|-----|------|
| Year 1         | Indoor   | 561 | 0.59 ± 0.13 | 0.22 | 0.98 |
| (October 1999–August 2000) | Outdoor  | 553 | 0.63 ± 0.12 | 0.08 | 1.05 |
| Central        | 103      | 0.59 ± 0.12 | 0.27 | 0.84 |
| Year 2         | Indoor   | 644 | 0.56 ± 0.19 | 0.09 | 1.39 |
| (September 2000–May 2001) | Outdoor  | 628 | 0.67 ± 0.16 | −0.01 | 1.23 |
| Central        | 113      | 0.60 ± 0.16 | 0.07 | 0.98 |
| Heating season | Indoor   | 708 | 0.60 ± 0.17 | 0.09 | 1.17 |
| (October–February) | Outdoor  | 691 | 0.70 ± 0.13 | 0.08 | 1.10 |
| Central        | 119      | 0.66 ± 0.12 | 0.20 | 0.98 |
| Nonheating season | Indoor  | 497 | 0.55 ± 0.15 | 0.18 | 1.39 |
| (March–September) | Outdoor  | 491 | 0.57 ± 0.13 | −0.01 | 1.23 |
| Central        | 97       | 0.50 ± 0.11 | 0.07 | 0.73 |
| Combined       | Indoor   | 1,205 | 0.58 ± 0.16 | 0.09 | 1.39 |
|                 | Outdoor  | 1,191 | 0.65 ± 0.14 | −0.01 | 1.23 |
|                 | Central  | 216   | 0.59 ± 0.14 | 0.07 | 0.98 |

### Abbreviations:
- Max: maximum; Min: minimum.
- *Indoor, outdoor, and central-site ratios significantly different during heating/nonheating seasons ($p < 0.001$); indoor ($p = 0.002$) and outdoor ($p < 0.001$) differ by year, but central site does not ($p = 0.55$). *Number of daily samples. *Some maximum ratios are > 1 due to the measurement error at low PM concentrations.

### Figure 2. PM measurements during heating and nonheating (non-H) seasons. (A) PM$_{2.5}$. (B) PM$_{10}$. The number of daily samples is shown in parentheses. Boxes, 25th–75th percentiles; whiskers, 10th–90th percentiles; solid lines, median; dotted lines, mean; data points, outliers.

### Figure 3. Cumulative distribution functions for indoor, outdoor, personal, and central-site PM measurements. (A) PM$_{2.5}$. (B) PM$_{10}$.

### Figure 4. Non-H vs. Heating plots of PM$_{2.5}$. (A) The representative underlying distribution. The shrunk correlation estimates (Lumley T, Liu L-JS. Unpublished data), by modifying slightly the upper end and significantly the lower end of the crude correlations by an appropriate amount (Figure 4B), give a more representative underlying distribution. The shrunk $r$ estimates ranged between 0.10 and 0.82, with a median of 0.43. The correlation between the crude and shrunk correlations is 0.98. There were no significant differences among study groups in either the crude or shrunk longitudinal $r$ ($p = 0.43$).
We estimated $F_{\text{int}}$ using the recursive model (Switzer and Ott 2001) with continuous nephelometer measurements taken concurrently with HI measurements (Allen R, Larson T, Wallace L, Liu L-JS. Unpublished data). The $F_{\text{int}}$ estimates, available for 55 home sites, ranged between 0.07 and 1.00, with a mean $\pm$ SD of 0.57 $\pm$ 0.23 and a median of 0.64 (Allen R, Larson T, Wallace L, Liu L-JS. Unpublished data). $F_{\text{int}}$ is the only important predictor for the longitudinal model (Switzer and Ott 2001) with continuous nephelometer measurements taken concurrently with HI measurements (Allen R, Larson T, Wallace L, Liu L-JS. Unpublished data).

**Time–activity pattern.** Table 5 shows the average percentage of the 24-hr day spent in different microenvironments as reported in subjects’ time–activity diaries. On average, asthmatic subjects spent 66% of the time at home indoors and 21% indoors away from home (mostly at school). Elderly subjects spent between 83% and 88% of the time in their homes and between 6% and 8% of the time outdoors away from home. As expected, asthmatic children spent more time outdoors (4.7 $\pm$ 3.9%) compared with all elderly subjects (0.9–1.7%; $p < 0.05$). Time outdoors of asthmatic children was far more limited than that of healthy children, but was significantly affected by sex (24) (28) (24) (17) (24) (28) (24) (17). The only activity that differed by gender was spending 6 ± 3 min fewer being active outdoors ($p < 0.05$).

**Effects of health condition, age, and sex on personal PM$_{2.5}$.** Personal PM$_{2.5}$ exposures ($C_p$) were examined for all study groups while controlling for home outdoor PM$_{2.5}$ concentration ($C_o$), home type ($H$), session ($S$), and the group $\times H$ interaction effects. We used the following fixed-effect model:

$$C_p = \mu + group + C_o + S + H + group \times H + \epsilon,$$

where $\mu$ is the overall mean and $\epsilon$ is the error term. The session effect accounts for any systematic differences between sessions, such as changes in neighborhood or subject cluster that could not be accounted for by $C_o$. Age was not included in this analysis because the age range in children was far more limited (6–13 years) than that in adults. The healthy elderly group and private homes were used as the references in the model. Results show that personal PM$_{2.5}$ exposures differed significantly by group, with that of the asthmatic group 5.6 g/m$^3$ greater, and the COPD group 3.5 g/m$^3$ greater, than the healthy elderly group (Table 7). There are significant interactions between home type and group. The COPD subjects living in group homes or private apartments who had lower PM$_{2.5}$ exposure than did other COPD subjects living in private homes, whereas the reverse was true for the CHD. The interaction effects canceled each other such that the home effect is not significant. This model also estimated that an average of 39% of outdoor PM$_{2.5}$ contributed to personal PM$_{2.5}$.

Among the elderly subjects, age is an important factor affecting personal exposure. For elderly subjects, personal PM$_{2.5}$ exposure was significantly reduced by 0.23 g/m$^3$ for each year of age increase (Table 7). Age is not a significant predictor for personal PM exposure among asthmatic children, most likely because of the small age range among subjects.

**Microenvironmental modeling and personal cloud.** We used a microenvironmental model (Özkaynak et al. 1996) with three microenvironments to predict personal exposures to PM$_{2.5}$. The three microenvironments

![Figure 4. Longitudinal correlation (Pearson’s $r$) between personal and central site PM$_{2.5}$ for each subject by study group (n = 6 for each subject). (A) Crude correlation. (B) Shrunken estimates. The number of subjects is shown in parentheses. Boxes, 25th–75th percentiles; whiskers, 10th–90th percentiles; solid lines, median; dotted lines, mean; data points, outliers.](image-url)
are indoor (including home, work, and other places), outdoor near home, and outdoor away from home. The model predicts personal exposures \((C_p)\) by summing up time-weighted exposures from each microenvironment:

\[
\hat{C}_p = (C_i \times F_i) + (C_v \times F_v) + (C_{oo} \times F_{oo}),
\]

where \(C_i, C_v\), and \(C_{oo}\) are PM\(_{2.5}\) concentrations measured indoors at home, outdoors at home, and at the central site, respectively. \(F_i, F_v\), and \(F_{oo}\) are fractions of the 24-hr day spent indoors at all locations, outdoors near home, and outdoors away from home, respectively. This model does not include personal cloud (or, equivalently, the error term), whereas in the earlier model (Equation 2) the error term appears explicitly. Technically, the \(C\) values in Equation 3 should be averaged only during the time the person is in that microenvironment, but in fact only 24-hr averages were available for all three \(C\) values. This is not much of a problem for the last two terms because of slow-changing outdoor \(C\) values and very small \(F\) values. However, it may be problematic if \(F_i\) includes a fair amount of time at school or work, where the concentration is unknown, which may introduce an additional source of error. This model predicts exposures of the elderly groups relatively well (Table 8). When the model predictions were regressed against measured personal exposures, this microenvironmental model predicts between 45% and 62% of the variability in measured elderly PM\(_{2.5}\) exposures. The percentage of variation explained was the highest for elderly CHD and COPD groups, due to the limited microenvironments encountered and personal activities. The lowest prediction power was observed for the children’s asthmatic group (\(R^2 = 0.09\)), due in part to the fact that the home indoor PM\(_{2.5}\) measurements were used as a surrogate for the PM\(_{2.5}\) levels away from home.

Some of the unexplained variability is likely due to the so-called “personal cloud.” We define the personal cloud as the difference between the predicted and measured personal PM exposures. The personal cloud is a combined result of particles generated from personal activities (e.g., cooking or dusting) and exposures to local sources (e.g., next to traffic exhaust on the street) that are not captured by the stationary indoor and outdoor monitors. When using the intercepts of the regression models as estimates for the personal clouds, asthmatic children had the highest personal cloud (9.6 µg/m\(^3\)), and elderly groups had similar low personal clouds (1.1, 2.2, and 2.4 µg/m\(^3\) for CHD, COPD, and healthy elderly groups, respectively). However, when using the difference between the measured and the modeled personal exposures, the personal cloud is much lower for asthmatic children (3.9 µg/m\(^3\)) and comparable for elderly (1.7, 2.3, and 1.3 for CHD, COPD, and the healthy elderly groups, respectively).

The personal cloud, estimated using the difference between the predicted and measured values, differed significantly by group. For elderly subjects, the most important factors contributing to the personal cloud are the time (in minutes) spent outside running errands, cooking indoors, and in the yard outdoors (Table 9). For the asthmatic group, the most important factors are the time (in minutes)

### Table 5. Percentage of time spent in microenvironments by study group.

| Group | Microenvironment | Percentage of time spent in each microenvironment |
|-------|-------------------|--------------------------------------------------|
|       | Mean ± SD | Min | Max |
| Asthmatics | (n = 33) | | |
| Home | 86.4 ± 5.7 | 55.5 | 80.0 |
| Yard | 1.7 ± 2.6 | 0.0 | 8.2 |
| In transit | 4.4 ± 1.7 | 1.3 | 8.2 |
| Work | 1.1 ± 3.5 | 0.0 | 16.5 |
| Outdoors | 4.7 ± 3.5 | 0.1 | 17.5 |
| Indoors away from home | 21.0 ± 6.4 | 4.5 | 33.2 |
| Cooking, self | 0.1 ± 0.1 | 0.0 | 0.5 |
| Cooking, others | 0.7 ± 0.5 | 0.0 | 1.9 |

### Table 6. Associations between proportion of time spent indoors or outdoors (in fraction) and subject characteristics and session.

| Time spent (fraction) | Parameter | Estimate | SE | Confidence limit | p-Value |
|-----------------------|-----------|----------|----|-----------------|---------|
| Indoors | Elderly adults | Intercept | 0.270 | 0.069 | 0.135 | 0.405 | 0.00 | 0.52 | |
| | | Female | -0.007 | 0.011 | -0.028 | 0.014 | 0.52 | |
| | Age (per year increase) | Healthy | 0.007 | 0.001 | 0.005 | 0.009 | 0.00 | |
| | | CHD | 0.040 | 0.025 | -0.010 | 0.089 | 0.11 | |
| | | COPD | 0.049 | 0.012 | 0.025 | 0.073 | 0.00 | |
| | | Session | 0.049 | 0.012 | 0.025 | 0.073 | 0.00 | |
| Asthmatic children | Intercept | 0.011 | 0.024 | -0.050 | 0.037 | 0.66 | |
| | Age (per year increase) | Session | -0.012 | 0.005 | -0.022 | -0.001 | 0.03 | 0.37 | |
| Outdoors | Elderly adults | Intercept | 0.0674 | 0.0193 | 0.0295 | 0.1052 | 0.00 | |
| | | Female | -0.0015 | 0.0030 | -0.0074 | 0.0043 | 0.61 | |
| | Age (per year increase) | Healthy | -0.0006 | 0.0002 | -0.0010 | -0.0001 | 0.01 | |
| | | CHD | -0.0001 | 0.0070 | -0.0039 | 0.0137 | 0.99 | |
| | | COPD | -0.0027 | 0.0034 | -0.0139 | -0.0006 | 0.03 | |
| | | Session | -0.0001 | 0.0070 | -0.0039 | 0.0137 | 0.99 | |
| Asthmatic children | Intercept | 0.0100 | 0.0290 | -0.0469 | 0.0669 | 0.73 | |
| | | Female | -0.0130 | 0.0134 | -0.0393 | 0.0133 | 0.33 | |
| | Age (per year increase) | Session | 0.0030 | 0.0029 | -0.0027 | 0.0087 | 0.30 | 0.02 | |
Discussion
The average personal PM$_{2.5}$ exposures among sensitive subpopulations in Seattle were similar to the mean outdoor PM$_{2.5}$ concentration but significantly higher than that indoors. Our elderly subjects’ personal PM$_{2.5}$ exposure (GM = 7.7–8.8 µg/m$^3$) was lower than those observed among elderly subjects in previous studies living in nonsmoking homes (Ebel et al. 2000; Evans et al. 2000; Janssen et al. 2000; Williams et al. 2000a, 2000b). This is most likely due to the low ambient PM$_{2.5}$ in Seattle. The GM of 17 elderly COPD subjects in Vancouver, British Columbia, was 10.8 µg/m$^3$ (Ebel et al. 2000); the median of 18 elderly COPD subjects in Boston, Massachusetts, was 15.5–18.5 µg/m$^3$ (Rojas-Bracho et al. 2000); the GM was 11.4 µg/m$^3$ for five healthy elderly subjects during winter 1999 and 10.8 µg/m$^3$ for 16 elderly subjects during spring 1999 in Fresno, California (Evans et al. 2000); the median was 15.3 µg/m$^3$ for 37 CHD subjects in Amsterdam and 10.0 µg/m$^3$ for 47 CHD in Helsinki (Janssen et al. 2000). The personal PM$_{2.5}$ exposure in the asthmatic children in our study, who lived in nonsmoking households in Seattle (arithmetic mean, 13.3 µg/m$^3$), was also lower than those found elsewhere. The arithmetic mean was 24.4 µg/m$^3$ for nine children in nonsmoking households and 37.0 µg/m$^3$ for four children in smoking households in Wageningen, The Netherlands (Janssen et al. 1999).

In Seattle, both PM$_{2.5}$ and PM$_{10}$ levels were significantly elevated during the heating season at all locations, including indoors, outdoors, and around subjects (PM$_{2.5}$ only) (Figure 2). The seasonal variation was more prominent in outdoor PM levels than in indoor or personal PM measurements. Previous studies also found seasonal variation in outdoor PM$_{2.5}$ and PM$_{10}$ levels: Northeastern U.S. cities have higher PM$_{2.5}$ and PM$_{10}$ levels in the summer because of the enhanced photochemical production of sulfate and other secondary pollutants (Rojas-Bracho et al. 2000; Sarnat et al. 2000; Wilson and Suh 1997), and western U.S. cities have higher PM$_{2.5}$ and PM$_{10}$ levels in the winter because of wood burning and lack of photochemical reaction enhancement in the summer (Larson et al. 1989; Rodes et al. 2001). However, Rojas-Bracho et al. (2000) and Rodes et al. (2001) did not find significant seasonal changes in either personal or indoor PM$_{2.5}$ and PM$_{10}$ levels. We found that although $F_{int}$ varied by season in private home, it did not vary significantly in group homes or private apartments (Allen R, Larson T, Wallace L, Liu L-JS. Unpublished data). This fact, coupled with the higher outdoor PM in winter, results in higher indoor and personal PM levels in winter.

### Table 7. Association between personal and indoor PM$_{2.5}$ measurements and study group, controlling for session, home type, and outdoor PM$_{2.5}$ concentration.

| Parameter                  | Estimate | SE    | Lower | Upper | p-Value | Model R$^2$   | p-Value |
|----------------------------|----------|-------|-------|-------|---------|---------------|---------|
| All groups                 | 22.87    | 3.91  | 15.21 | 30.53 | < 0.0001 | 0.27          | < 0.0001|
| Health status              | 0.43     | 1.48  | –2.48 | 3.33  | 0.77     |               |         |
| CHD                        | 3.84     | 1.04  | 1.80  | 5.87  | < 0.001  |               |         |
| Age (per year increase)    | –0.23    | 0.05  | –0.32 | –0.14 | < 0.0001 |               |         |
| Healthy                    | 0.39     | 0.04  | 0.31  | 0.48  | < 0.0001 |               |         |
| Home                       | 0.14     |       |       |       | 0.14     |               |         |
| Session                    | –0.71    | 1.77  | –4.17 | 2.75  | < 0.0001 |               |         |
| Health × home              | 0.75     |       |       |       | 0.36     |               |         |

### Table 8. Regression analysis results for measured compared with microenvironmental model-predicted PM$_{2.5}$ personal exposures ($C^p$).

| Group          | Intercept | SE    | Lower | Upper | p-Value | C$^p$ mean |
|----------------|-----------|-------|-------|-------|---------|------------|
| Asthmatic children | 9.57     | 1.01  | < 0.0001 | 0.09 | 13.6 |
| Elderly adults   | 0.41      | 0.09  | < 0.0001 |    |        |
| CHD             | 1.07      | 0.57  | 0.06  | 0.62 | 11     |
| COPD            | 1.07      | 0.05  | < 0.0001 |    |        |
| Healthy         | 2.38      | 0.63  | < 0.0001 | 0.45 | 8.4     |

### Table 9. Activities (in minutes) affecting the PM$_{2.5}$ personal cloud (µg/m$^3$).

| Parameter                  | Estimate | SE    | Wald 95% confidence limit | p-Value |
|----------------------------|----------|-------|---------------------------|---------|
| Elderly adults             | 0.70     | 0.49  | –0.26                     | 1.66    | 0.152  |
| Health status              | 0.27     | 0.52  | –0.74                     | 1.28    | 0.598  |
| CHD                        | 1.23     | 0.52  | 0.22                      | 2.24    | 0.017  |
| COPD                       | 0.02     | 0.01  | 0.01                      | 0.03    | 0.002  |
| Outdoor errands            | 0.01     | 0.01  | 0.00                      | 0.02    | 0.024  |
| Cooking, indoors           | 0.04     | 0.02  | 0.00                      | 0.07    | 0.043  |
| In yard, outdoors          | 0.12     | 0.31  | –2.27                     | 4.81    | 0.482  |
| Asthmatic children         | 1.27     | 1.81  | 0.00                      | 0.03    | 0.006  |
| At school indoors          | 0.01     | 0.01  | 0.00                      | 0.03    | 0.008  |
| In bus or shuttle           | 0.05     | 0.02  | 0.01                      | 0.59    | 0.008  |
The mean PM$_{2.5}$:PM$_{10}$ ratios indoors, outdoors, and at the central site were significantly higher during the heating season (October through February). The mean indoor PM$_{2.5}$:PM$_{10}$ ratio during the heating season (0.60) was similar to that reported by Rodes et al. (2001) in Fresno indoors between January and February 1999 (0.61), whereas the PM$_{2.5}$:PM$_{10}$ ratio in the nonheating season (0.55) was similar to that in Fresno (0.51) between April and May 1999. The home outdoor PM$_{2.5}$:PM$_{10}$ ratio during the heating season (0.70) again was similar to that reported in Fresno (0.73, January through February 1999), whereas the home outdoor nonheating PM$_{2.5}$:PM$_{10}$ ratio (0.57) was much higher than that in Fresno (0.36). The much lower ratio in Fresno is partially due to the distinct nonheating season and partially to the introduction of coarse dusts from the adjacent San Joaquin Valley during the drier spring. The higher outdoor PM$_{2.5}$ proportion during the heating seasons in Fresno and Seattle is similar to that in Baltimore during the summer (0.73). A number of studies have examined the relationship between personal exposures and central-site measurements. Results from the Particle Total Exposure Assessment Methodology (PTEAM) study (Clayton et al. 1993; Özkaynak et al. 1996) indicated that correlations of personal exposures with fixed-site outdoor concentrations were low for PM$_{10}$ (ranging between 0.37 in the daytime and 0.54 at night). The relationship could be improved considerably when longitudinal regressions were performed for each subject (Janssen et al. 1997, 1998, 2000). Outdoor sulfur or sulfate, which is predominantly in fine particles and of outdoor origin, was highly correlated with personal sulfur or sulfate exposures (Brauer et al. 1999; Ebel et al. 2000; Özkaynak et al. 1996; Stieb et al. 1998; Suh et al. 1992; Wallace 1996). For susceptible subjects in Seattle, the cross-sectional Pearson’s correlation between personal and central PM$_{2.5}$ was 0.29 over all individual days (p < 0.0001; Spearman’s r = 0.37; n = 974). The median longitudinal r between personal PM$_{2.5}$ exposure and central-site measurements was 0.34 (median shrunk r = 0.43) and does not vary much across groups. Our longitudinal correlations are in agreement with the large correlation range found in other panel studies (see, e.g., summary table 5 in Ebel et al. 2000). More recent elderly panel studies showed the median correlation ranging between 0 in Nashville, Tennessee (Bahadori et al. In press), and 0.80 in Fresno (spring; Rodes et al. 2001). Because the sample size and sampling duration vary by study, it should be interesting to compare the longitudinal shrunk estimates across all studies.

The high correlations among outdoor sites for PM$_{2.5}$ and PM$_{10}$ in Seattle are consistent with our earlier findings in Seattle. Goswami et al. (2002) found that although the PM$_{2.5}$ concentration varied by elevation and the distance from major thoroughfares to the home sites, outdoor PM$_{2.5}$ measurements were highly correlated, with a median Pearson’s r of 0.89 for 135 pairs of concurrent outdoor home sites.

Significant differences in the fraction of time spent inside, outside homes, and in transit were observed among the study groups in this study (Tables 5 and 6). The COPD and CHD study groups spent more time at home (86–88%) than did the healthy elderly group (83%), whose time at home was similar to the 81% for elderly persons (> 64 years of age) in the general population reported in the National Human Activity Pattern Survey (NHAPS) (Klepeis et al. 1996). The asthmatic children in our study spent an average of 66% of the time at home; slightly lower than the 70% reported for children between 5 and 11 years of age in NHAPS (Tsang and Klepeis 1996).

The longitudinal correlations are a function of the particle $F_{int}$. The personal PM exposure consists of the ambient originated PM, indoor originated PM, and the personal cloud (Wilson et al. 2000). Therefore, the longitudinal r for the personal exposure and the central-site measurements (i.e., ambient originated PM) is a function of the sum of the variances of the indoor and personal (or nonambient) originated PM ($\sigma^2_x$), the variance of the ambient generated PM ($\sigma^2_a$), and the attenuation from ambient PM to personal exposure ($\alpha$), which is the sum of the fraction of time spent outdoors ($y$) and the fraction of time spent indoors (1 – y) times $F_{int}$ [i.e., $\alpha = y + (1 - y) \times F_{int}$]. Note that $\alpha$ can be approximated by $F_{int}$ because most people spend very little time outdoors and therefore $y$ is negligible. Based on the definition of correlation, the longitudinal r can be written as

$$ \text{Longitudinal } r = \frac{1}{\sqrt{1 + \frac{R}{\alpha^2}}} \quad [4] $$

where $R = \sigma^2_x / \sigma^2_a$. Simulated longitudinal correlation plots for longitudinal r and $F_{int}$ based on different R values are shown as curves in Figure 5. Our measurements show that most longitudinal correlations fall between $R = 0.05$ and 1 (black circles), indicating that for most individuals, the variance of the nonambient-originated PM, $\sigma^2_a$, is generally smaller than the variance in ambient originated PM, $\sigma^2_x$. A much smaller group of data points falls beyond the line $R = 3$, (blue circles), which has smaller longitudinal r values (< 0.4) even when $\alpha = 1$. For this group of individuals, the variance in nonambient-originated PM is greater than the variance of ambient-originated PM such that the longitudinal r is small (and most likely insignificant) regardless of $\alpha$. These results show exactly why ambient PM concentrations were significantly associated with corresponding personal exposures for only about one-half to two-thirds of the monitored populations in past panel studies (Ebel et al. 2000; Sarnat et al. 2000).

The three-microenvironmental model (Table 8) predicts personal exposures relatively well for the elderly subjects. Levels of the personal cloud in our elderly groups are lower than those reported in other elderly groups: 3.7 µg/m$^3$ (12-hr average) in 18 COPD patients in Boston (Rojas-Bracho et al. 2000; Wallace 2000), 3.1 µg/m$^3$ for COPD subjects in Baltimore (Rodes et al. 2001), and 3.4 µg/m$^3$ (24-hr average) for elderly subjects in Fresno (Rodes et al. 2001). The elderly subjects’ personal cloud in this study was much lower than the 27 µg/m$^3$ reported for 18 healthy subjects in Azusa, California (Wallace 2000), or 6 µg/m$^3$ for 10 COPD patients in Nashville (Bahadori et al. In press). The personal cloud for the asthmatic children in our study, 3.9 µg/m$^3$, is also smaller than the 11 µg/m$^3$ reported for 13 children in Amsterdam (Janssen et al. 1997). Our regression results showed that the personal cloud in the elderly groups can be attributed to running errands outdoors, cooking, and activities conducted in the yard, whereas the personal cloud among the asthmatic children can be attributed to time spent away from home (e.g., inside the school and riding the bus or shuttle). It is possible that the PM$_{2.5}$ concentration differs between the children’s homes and other indoor environments (Rea et al. 2001), where children spent about 21% of their time during the day, usually at school, in transit, and in extracurricular activities. Therefore, using home indoor measurements to represent PM$_{2.5}$ concentrations in these “away-from-home” environments resulted in an artificially larger personal cloud

![Figure 5](image-url)
for children. The microenvironmental model (Equation 3) also does not include the personal cloud, so we are left with an ambiguity about what exactly accounts for the difference in observed versus expected exposure values that are on the order of 1–2.5 μg/m³ for the elderly groups. Only 39% of the outdoor PM contributed to personal PM2.5 exposure, as estimated by the fixed-effect model (Equation 2) (Table 7); this indicates that personal PM2.5 exposure is mostly attributed to nonambient sources, resulting in a low prediction power when using the outdoor or central-site measurements to predict personal exposures. However, a three-microenvironmental model that includes indoor home, outdoor home, and other outdoor environments resulted in relatively good prediction power (R² = 0.5–0.55) for the elderly groups. Therefore, given the time–activity pattern and micro-environmental concentrations, the elderly susceptible subjects’ PM2.5 exposures are relatively predictable.

Conclusions

The average personal PM2.5 exposures that we found among sensitive subpopulations in Seattle were similar to the average outdoor PM2.5 concentrations but significantly higher than average indoor concentrations. The elderly subjects’ personal PM2.5 exposures were lower than those reported for other elderly subjects in other cities. The personal PM2.5 exposure in the asthmatic children in this study, who lived in nonsmoking households, was also lower than those found elsewhere. PM2.5 and PM10 concentrations, as well as the ratio of PM2.5 to PM10 concentrations, vary seasonally; higher concentrations were found indoors and outdoors during the heating season. A similar seasonal variation was also found for personal PM2.5 exposures. Personal PM2.5 exposures varied by study group, with elderly healthy and CHD subjects having similar exposures, elderly COPD subjects experiencing slightly higher exposures, and asthmatic children having the highest exposures. The PM2.5 exposure varied within the study groups, depending on the type of residences, most likely due to the differences in particle infiltration rates among residences. In addition, we found that the high-risk subjects engaged in an equal amount of dust-generating activities as did the healthy elderly subjects. The elderly COPD and CHD subjects had higher indoor PM2.5 concentrations than did the elderly healthy subjects. The child subjects experienced the highest indoor PM2.5 and PM10 concentrations. Although a wide range of longitudinal correlations between central-site and personal PM2.5 measurements was found, our results show that the longitudinal r is closely related to the particle infiltration efficiency of each residence. The PM2.5 exposures among the COPD and CHD subjects can be predicted with a relatively good prediction power using a microenvironmental model with three microenvironments. The prediction power is the lowest for the asthmatic children in our study, whose in-school exposure was not accounted for in this microenvironmental model.

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