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 ($PM_{2.5}$) were similar to the average outdoor $PM_{2.5}$ concentrations but significantly higher than the average indoor concentrations. Personal $PM_{2.5}$ exposures in our study groups were lower than those reported in other panel studies of susceptible populations. Indoor and outdoor $PM_{2.5}$, PM_{10} (PM with aerodynamic diameters < 10 μm), and the ratio of $PM_{2.5}$ to PM_{10} were significantly higher during the heating season. The increase in outdoor PM_{10} in winter was primarily due to an increase in the $PM_{2.5}$ fraction. A similar seasonal variation was found for personal $PM_{2.5}$. 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 $PM_{2.5}$ and PM_{10} concentrations. Personal $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ measurements, the longitudinal *r* is closely related to the particle infiltration efficiency. $PM_{2.5}$ 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, $PM_{2.5}$, 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 (PM_{10}) or < 2.5 μm ($PM_{2.5}$)] to ultrafine particles (aerodynamic diameters < 0.1 μm). Several studies indicate that $PM_{2.5}$ may be more strongly associated with some adverse health end points 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 cardiorespiratory 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 (Ebelt et al. 2000; Evans et al. 2000; Janssen 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 PM_{10}, $PM_{2.5}$, 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 cardiorespiratory 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 (FEV_1) between 40% and 70% predicted value. All CHD subjects had a history

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We thank L. Tuttle, T. Gould, J. Sullivan, C. Trenga, J.C. Slaughter, and the field/laboratory technicians who worked on this project. We owe a great deal to our study subjects.

This work was funded by the U.S. EPA (CR82717701), the Northwest Center for Particulate Air Pollution and Health (U.S. EPA grant CR827355), and National Institute of Environmental Health Sciences grant P30 ES07033.

This report has been subjected to U.S. EPA review and approved for publication. Mention of trade names or commercial products does not constitute an endorsement or recommendation for use.

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°F; 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 $PM_{2.5}$ exposures were determined using the Harvard Personal Environmental Monitor for $PM_{2.5}$ ($HPEM_{2.5}$; Harvard School of Public Health, Boston, MA). The small $HPEM_{2.5}$ is a single-stage inertial impactor with a 50% cut point of 2.4 ± 0.1 µm (Sioutas et al. 1999). The $HPEM_{2.5}$ was connected to a personal pump (AFC 400S; BGI, Inc., Waltham, MA) with a mass flow controller operated 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 $HPEM_{2.5}$ 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 PM_{10} and $PM_{2.5}$ (Marple et al. 1987). One $HI_{2.5}$–HI_{10} 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 $HI_{2.5}$–HI_{10} 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. $HI_{2.5}$, HI_{10}, and $HPEM_{2.5}$ were also collocated with the federal reference method monitor for $PM_{2.5}$ ($FRM_{2.5}$) at the central Beacon Hill site, which is located in a semiresidential 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 (Goswami et al. 2002). Duplicate sets of central-site $HPEM_{2.5}$, $HI_{2.5}$, and HI_{10} were running at the same schedule as those at home sites (1600 hr to 1600 hr) for estimating precision. One central-site $HI_{2.5}$–$HPEM_{2.5}$ pair ran from midnight to midnight to coincide with the $FRM_{2.5}$ 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 time, activity, 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	Study group				Total
			Asthmatics	CHD	COPD	Healthy	
1999	October 26	1	—	0	5	3	8
	November 8	2	—	0	5	4	9
	November 29	3	—	0	5	3	8
2000	January 10	4	—	0	3	6	9
	February 7	5	—	1	2	3	6
	February 21	6	—	0	3	3	6
	March 6	7	—	1	3	3	7
	March 27	8	—	0	4	4	8
	April 10	9	—	0	3	2	5
	May 1	10	—	0	5	3	8
	May 15	11	—	0	4	0	4
	July 10	12	—	1	4	2	7
	July 31	13	—	1	1	2	4
	September 25	1	—	2	4	—	6
	October 16	2	—	3	5	—	8
	November 6	3	—	6	—	—	6
	November 27	4	2	4	—	—	6
	December 25	5	4	1	—	—	5
2001	January 8	6	5	1	—	—	6
	January 22	7	3	3	—	—	6
	February 5	8	2	3	—	—	5
	February 26	9	4	4	—	—	8
	March 29	10	5	3	—	—	8
	April 16	11	3	2	—	—	5
	April 30	12	4	1	—	—	5
	May 14	13	1	3	—	—	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 calculated as three times the standard deviation of the field blanks. The LOD for the 24-hr integrated HI was 1 μg/m^3; for the 24-hr integrated $HPEM_{2.5}$, the LOD was 6.2 μg/m^3 for the first four sessions and 4.5 μg/m^3 afterward. This reduction was achieved by replacing the oiled 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/m^3 LOD for $HPEM_{2.5}$ is similar to values (2.6–4.0 μg/m^3) 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/m^3 for HI and 2.2 μg/m^3 for $HPEM_{2.5}$.

The accuracy of our $PM_{2.5}$ measurements was calculated by comparing them with the collocated $FRM_{2.5}$ measurements at the central site (Figure 1A). We also collocated $HI_{2.5}$ and $HPEM_{2.5}$ 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/m^3; $p < 0.001$) for $HPEM_{2.5}$ with an oiled impaction plate; with a greased impaction plate, the bias is negligible (0.4 μg/m^3; $p = 0.08$). All HI and HPEM measurements were corrected for average blank values. The $HPEM_{2.5}$ 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.

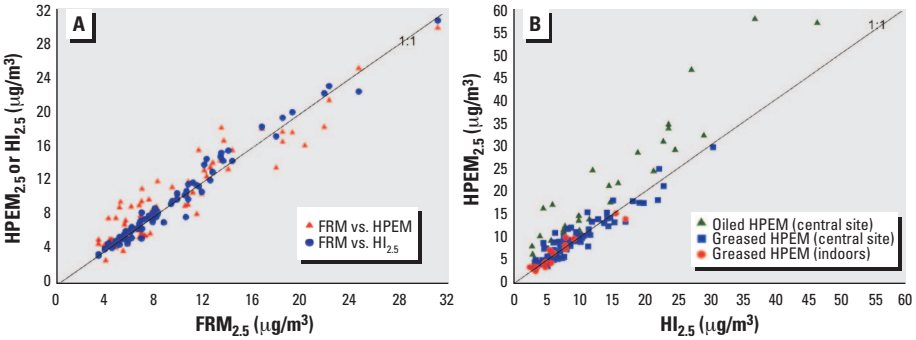

Figure 1. Comparison of HI, HPEM, and FRM measurements for $PM_{2.5}$. (*A*) HI and HPEM versus FRM at central sites [HPEM = 1.64 + (0.88 × FRM); R^2 = 0.87. HI = 0.09 + (0.97 × FRM); R^2 = 0.97]. (*B*) HI versus HPEM at central and home indoor sites.

Table 2. Summary of PM concentrations (μg/m^3) between October 1999 and May 2001 by study group.

Location	Pollutant	Group	No.[a]	Mean ± SD	GM	GSD	Min	Max
Personal	$PM_{2.5}$	COPD	307	10.5 ± 7.2	8.6	1.9	0.8	45.6
		Healthy	183	9.3 ± 8.4	7.7	1.8	0.8	96.2
		Asthmatic	263	13.3 ± 8.2	11.1	1.9	1.0	49.4
		CHD	325	10.8 ± 8.4	8.8	1.9	1.4	66.6
Indoor	$PM_{2.5}$	COPD	443	8.5 ± 5.1	7.3	1.7	1.0	49.9
		Healthy	193	7.4 ± 4.8	6.1	1.9	0.4	38.0
		Asthmatic	276	9.2 ± 6.0	7.9	1.7	2.2	36.3
		CHD	329	9.5 ± 6.8	8.0	1.8	1.6	65.3
	PM_{10}	COPD	437	14.1 ± 6.6	12.7	1.6	2.5	40.1
		Healthy	206	12.6 ± 7.8	10.6	1.9	0.6	62.2
		Asthmatic	274	19.4 ± 11.1	16.8	1.7	2.2	107.7
		CHD	324	16.2 ± 11.3	13.6	1.8	0.6	110.6
Outdoor	$PM_{2.5}$	COPD	437	9.2 ± 5.1	8.0	1.7	–0.2	28.9
		Healthy	194	9.0 ± 4.6	7.9	1.7	0.7	24.5
		Asthmatic	272	11.3 ± 6.4	9.8	1.7	2.8	40.4
		CHD	323	12.6 ± 7.9	10.6	1.8	1.3	41.5
	PM_{10}	COPD	435	14.3 ± 6.8	12.8	1.6	2.9	41.4
		Healthy	200	14.5 ± 7.0	13.0	1.6	2.9	54.9
		Asthmatic	269	16.4 ± 7.4	14.7	1.6	1.2	47.3
		CHD	324	18.0 ± 9.0	16.1	1.6	3.3	54.3
Central site	$PM_{2.5}$		222	10.1 ± 5.7	8.6	1.8	1.0	29.5
	PM_{10}		221	17.3 ± 9.1	14.9	1.8	0.4	49.9

Abbreviations: GM, geometric mean; GSD, geometric SD; Max, maximum; Min, minimum.
[a]Number of daily samples.

Summary statistics. Table 2 summarizes concentrations of $PM_{2.5}$ and PM_{10} 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 $PM_{2.5}$ were below the new National Ambient Air Quality Standard (NAAQS) (U.S. EPA 2001) for the annual $PM_{2.5}$ average (15 μg/m^3), individual 10-day exposures exceeded the annual NAAQS for 12% of the elderly and 42% of the child subjects. The average indoor $PM_{2.5}$ levels were < 10 μg/m^3, whereas average indoor PM_{10} levels were between 10 and 20 μg/m^3. Personal $PM_{2.5}$ concentrations were similar to outdoor $PM_{2.5}$ concentrations (mean difference ± SD = 0.3 ± 8.3; $p = 0.29$, paired t-test) but significantly higher than indoor concentrations ($p < 0.0001$). Indoor $PM_{2.5}$ concentrations were significantly lower than those outdoors ($p < 0.0001$). The difference between PM_{10} and $PM_{2.5}$ measurements (coarse particles, $PM_{2.5–10}$) was approximately 5 μg/m^3 for both indoor and outdoor environments for all study groups, except inside asthmatic children's residences, where the mean $PM_{2.5–10}$ was double (10.2 μg/m^3).

$PM_{2.5}$ was on average 61% of the PM_{10} 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 $PM_{2.5}$:PM_{10} ratios were significantly higher ($p < 0.001$), whereas the central-site $PM_{2.5}$:PM_{10} ratio remained the same across years (Table 3). Both the indoor and outdoor $PM_{2.5}$:PM_{10} 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 $PM_{2.5}$ accounting for 70% of the outdoor PM_{10} mass.

Figure 2A shows $PM_{2.5}$ 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[a]	No.[b]	Mean ± SD	Min	Max[c]
Year 1 (October 1999–August 2000)	Indoor	561	0.59 ± 0.13	0.22	0.98
	Outdoor	553	0.63 ± 0.12	0.08	1.05
	Central	103	0.59 ± 0.12	0.27	0.84
Year 2 (September 2000–May 2001)	Indoor	644	0.56 ± 0.19	0.09	1.39
	Outdoor	628	0.67 ± 0.16	–0.01	1.23
	Central	113	0.60 ± 0.16	0.07	0.98
Heating season (October–February)	Indoor	708	0.60 ± 0.17	0.09	1.17
	Outdoor	690	0.70 ± 0.13	0.08	1.10
	Central	119	0.66 ± 0.12	0.20	0.98
Nonheating season (March–September)	Indoor	497	0.55 ± 0.15	0.18	1.39
	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	1,181	0.65 ± 0.14	–0.01	1.23
	Central	216	0.59 ± 0.14	0.07	0.98

Abbreviations: Max, maximum; Min, minimum.
[a]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 ($p = 0.55$). [b]Number of daily samples. [c]Some maximum ratios are > 1 due to the measurement error at low PM concentrations.

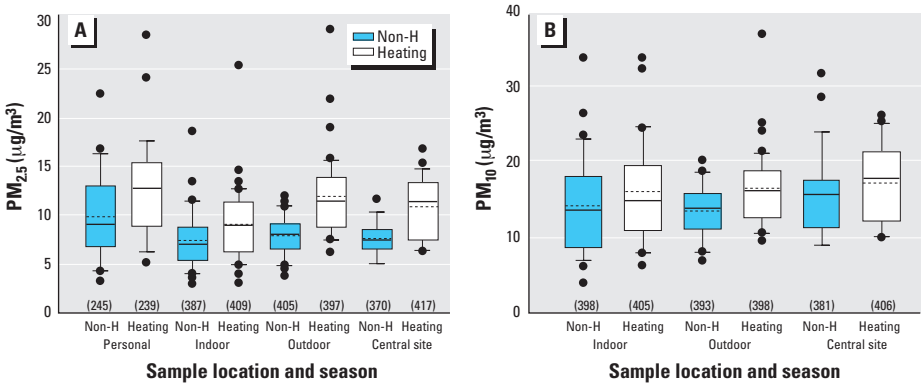

Figure 2. PM measurements during heating and nonheating (non-H) seasons. (*A*) $PM_{2.5}$. (*B*) PM_{10}. The number of daily samples is shown in parentheses. Boxes, 25th–75th percentiles; whiskers, 10th–90th percentiles; solid lines, median; dotted lines, mean; data points, outliers.

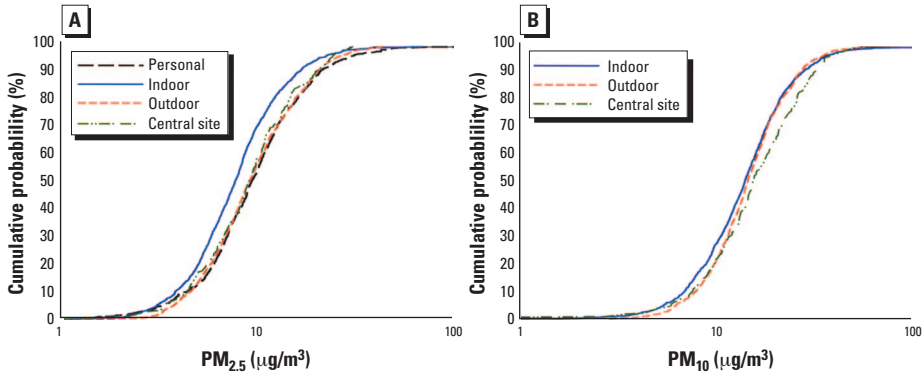

Figure 3. Cumulative distribution functions for indoor, outdoor, personal, and central-site PM measurements. (*A*) $PM_{2.5}$. (*B*) PM_{10}.

We estimated F_{inf} 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_{inf} estimates, available for 55 home sites, ranged between 0.07 and 1.00, with a mean ± SD of 0.57 ± 0.23 and a median of 0.64 (Allen R, Larson T, Wallace L, Liu L-JS. Unpublished data). F_{inf} is the only important predictor for the longitudinal r (r = 0.30; n = 33; p = 0.09).

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 inside their homes and between 6% and 8% of the time indoors away from home. As expected, asthmatic children spent more time outdoors (4.7 ± 3.5%) compared with all elderly subjects (0.9–1.7%; $p < 0.0001$, F-test). We analyzed time–activity patterns by age, sex, and health condition (Table 6). For elderly adults, the healthy group was used as the baseline for comparison. ANOVA analysis shows that percentage of time spent indoors at home was significantly affected by health condition and age among elderly subjects. The COPD group spent an average of 5% more time inside their homes than did the healthy group. For every 1-year increase in age, the time spent indoors also increased by 0.7%. For asthmatic children, the age effect is significant but negative. The COPD group spent an average of 0.7% less time outdoors than did the healthy elderly cohort, and every 1-year increase in age decreased the time spent outdoors by 0.06%. For time spent outdoors by asthmatic children, the significant predictor was session (a surrogate for time of year), not age.

We also examined minutes spent conducting various potential PM-generating activities versus health conditions, age, and sex, while controlling for the session effect (Tables 5 and 6). Indoor activities included cooking, dusting, vacuuming, sweeping, tidying up, and washing windows. Outdoor activities included exercise, yard work, painting, and so on, and in-transit included walking, riding a car, and so forth. The only activity that differed by elderly group was the time spent in transit, with the COPD group spending an average of 11 ± 2 min less than other elderly groups in transit, after controlling for the session effect ($p < 0.05$). Among the elderly groups, time spent on indoor and outdoor activities was significantly different by sex and age but not by study group. Elderly female subjects spent 18 ± 5 min more per day conducting indoor activities than did male subjects ($p < 0.001$). The older they were, the fewer indoor activities they conducted (–1.5 ± 0.4 min/year; $p < 0.001$). Minutes spent doing outdoor activities were significantly affected by sex in the opposite direction, with female elderly subjects 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 × H interaction effects. We used the following fixed-effect model:

$$C_p = \mu + \text{group} + C_o + S + H + \text{group} \times \text{H} + \varepsilon, \quad [2]$$

where μ is the overall mean and ε 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 μg/m^3 greater, and the COPD group 3.5 μg/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 had lower $PM_{2.5}$ exposure than did other COPD subjects living in private homes, whereas the reverse was true for the CHD subjects. 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 μg/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

Table 4. Spearman correlations (number of daily samples) between personal, indoor, outdoor, and central-site monitors for $PM_{2.5}$ and PM_{10}.

	Personal $PM_{2.5}$	Indoor $PM_{2.5}$	Outdoor $PM_{2.5}$	Central $PM_{2.5}$	Indoor PM_{10}	Outdoor PM_{10}	Central PM_{10}
Personal $PM_{2.5}$	1 (1,078)						
Indoor $PM_{2.5}$	0.65 (996)	1 (1,500)					
Outdoor $PM_{2.5}$	0.41 (1,009)	0.58 (1,425)	1 (1,497)				
Central $PM_{2.5}$	0.37 (974)	0.51 (1,293)	0.84 (1,297)	1 (1,408)			
Indoor PM_{10}	0.56 (1,007)	0.83 (1,454)	0.42 (1,439)	0.38 (1,303)	1 (1,514)		
Outdoor PM_{10}	0.41 (998)	0.57 (1,422)	0.91 (1,440)	0.81 (1,289)	0.41 (1,441)	1 (1,491)	
Central PM_{10}	0.37 (965)	0.5 (1,288)	0.76 (1,289)	0.9 (1,368)	0.41 (1,297)	0.82 (1,283)	1 (1,398)

All p-values are < 0.0001.

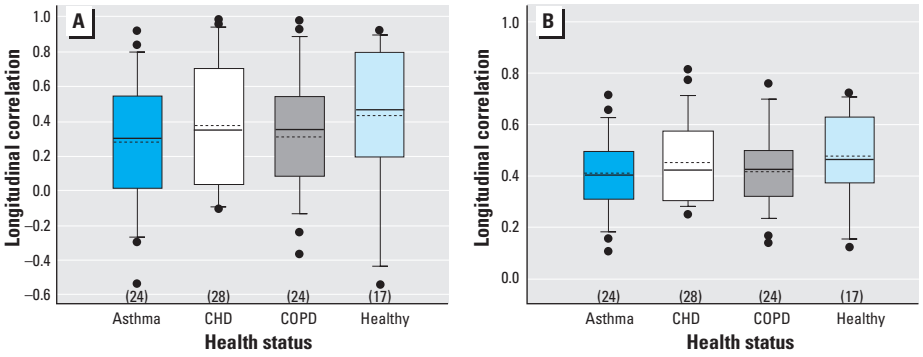

Figure 4. Longitudinal correlation (Pearson's r) between personal and central site $PM_{2.5}$ for each subject by study group ($n \geq 6$ for each subject). (*A*) Crude correlation. (*B*) Shrunk estimates. The number of subjects is shown in parentheses. Boxes, 25th–75th percentiles; whiskers, 10th–90th percentiles; solid lines, median; dotted lines, mean; data points, outliers.

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:

$$\hat{C}_p = (C_i \times F_i) + (C_o \times F_o) + (C_{oo} \times F_{oo}), \quad [3]$$

where C_i, C_o, and C_{oo} are $PM_{2.5}$ concentrations measured indoors at home, outdoors at home, and at the central site, respectively. F_i, F_o, 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	Home	66.4 ± 5.7	55.5	80.0
(n = 33)[a]	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
CHD	Home	85.5 ± 7.8	65.0	96.5
(n = 38)	Yard	1.0 ± 1.4	0.0	6.0
	In transit	3.6 ± 2.3	0.1	9.2
	Work	0.3 ± 1.7	0.0	10.6
	Outdoors	0.9 ± 1.2	0.0	4.8
	Indoors away from home	6.9 ± 5.1	0.1	20.9
	Cooking, self	1.7 ± 1.6	0.0	5.8
	Cooking, others	0.2 ± 0.3	0.0	1.5
COPD	Home	87.6 ± 6.9	71.4	100.0
(n = 56)	Yard	0.8 ± 1.0	0.0	4.3
	In transit	3.2 ± 1.9	0.0	7.3
	Work	0.1 ± 0.6	0.0	3.1
	Outdoors	1.0 ± 1.9	0.0	11.6
	Indoors away from home	6.1 ± 4.6	0.0	21.3
	Cooking, self	1.0 ± 1.3	0.0	5.6
	Cooking, others	0.2 ± 0.6	0.0	2.7
Healthy	Home	82.7 ± 8.3	66.8	99.2
(n = 39)	Yard	1.2 ± 1.6	0.0	6.7
	In transit	4.0 ± 2.5	0.5	9.3
	Work	1.0 ± 2.8	0.0	12.4
	Outdoors	1.7 ± 1.8	0.0	7.9
	Indoors away from home	8.0 ± 5.3	0.1	19.4
	Cooking, self	1.0 ± 1.0	0.0	4.4
	Cooking, others	0.3 ± 0.6	0.0	2.7

Abbreviations: Max, maximum; Min, minimum.
[a]Number of subjects in sessions.

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
				Lower	Upper	
Indoors						
Elderly adults	Intercept	0.270	0.069	0.135	0.405	0.00
(R^2 = 0.51, p < 0.001)	Female	–0.007	0.011	–0.028	0.014	0.52
	Age (per year increase)	0.007	0.001	0.005	0.009	0.00
	Health status					
	Healthy			Reference		
	CHD	0.040	0.025	–0.010	0.089	0.11
	COPD	0.049	0.012	0.025	0.073	0.00
	Session					0.45
Asthmatic children	Intercept	0.812	0.052	0.709	0.914	0.00
(R^2 = 0.47, p = 0.14)	Female	–0.011	0.024	–0.058	0.037	0.66
	Age (per year increase)	–0.012	0.005	–0.022	–0.001	0.03
	Session					0.37
Outdoors						
Elderly adults	Intercept	0.0674	0.0193	0.0295	0.1052	0.00
(R^2 = 0.23, p = 0.43)	Female	–0.0015	0.0030	–0.0074	0.0043	0.61
	Age (per year increase)	–0.0006	0.0002	–0.0010	–0.0001	0.01
	Health status					
	Healthy			Reference		
	CHD	–0.0001	0.0070	–0.0139	0.0137	0.99
	COPD	–0.0072	0.0034	–0.0139	–0.0006	0.03
	Session					0.77
Asthmatic children	Intercept	0.0100	0.0290	–0.0469	0.0669	0.73
(R^2 = 0.57, p = 0.03)	Female	–0.0130	0.0134	–0.0393	0.0133	0.33
	Age (per year increase)	0.0030	0.0029	–0.0027	0.0087	0.30
	Session					0.02

spent at school in class/library and in the bus or shuttle (Table 9). Waiting on the roadside for buses/cars is only marginally significant. The personal cloud was also negatively correlated with the longitudinal r ($r = -0.11$, $p < 0.01$) and was affected by the type of residence. Subjects living in group homes or private apartments had 1.1 μg/m^3 or 1.5 μg/m^3 lower personal cloud, respectively, than did those living in private homes. This could be due to the fact that more active subjects lived in private homes. As expected, the particle infiltration efficiency does not affect the personal cloud ($p = 0.75$).

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 (Ebelt 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 (Ebelt 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 median of 15 healthy senior citizens in Baltimore, Maryland, was between 14.5 and 23.1 μg/m^3 (Sarnat et al. 2000); the GM of 21 elderly subjects (6 healthy, 4 COPD, 11 CHD) in Baltimore was 12.4 μg/m^3 (Williams et al. 2000a, 2000b); 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_{inf} 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	Confidence limit Lower	Confidence limit Upper	*p*-Value	Model R^2	*p*-Value
All groups							
Intercept	5.72	1.73	2.34	9.11	< 0.001	0.23	< 0.0001
Health status							
Asthma	5.57	1.57	2.49	8.64	< 0.001		
CHD	–1.02	1.55	–4.05	2.01	0.51		
COPD	3.46	1.11	1.29	5.63	< 0.001		
Outdoor $PM_{2.5}$	0.39	0.04	0.31	0.47	< 0.0001		
Home					0.14		
Session					< 0.0001		
Health × home					< 0.0001		
Elderly adults							
Intercept	22.87	3.91	15.21	30.53	< 0.0001	0.27	< 0.0001
Health status							
CHD	0.43	1.48	–2.48	3.33	0.77		
COPD	3.84	1.04	1.80	5.87	< 0.001		
Age (per year increase)	–0.23	0.05	–0.32	–0.14	< 0.0001		
Outdoor $PM_{2.5}$	0.39	0.04	0.31	0.48	< 0.0001		
Home					0.75		
Session	–0.71	1.77	–4.17	2.75	< 0.0001		
Health × home					< 0.0001		

Table 8. Regression analysis results for measured compared with microenvironmental model-predicted $PM_{2.5}$ personal exposures ($\hat{C}_p$).

Group	Variable	Estimates β	Estimates SE	*p*-Value	R^2	$\hat{C}_p$ mean
Asthmatic children	Intercept	9.57	1.01	< 0.0001	0.09	13.6
	$\hat{C}_p$	0.41	0.09	< 0.0001		
Elderly adults						
CHD	Intercept	1.07	0.57	0.06	0.62	11
	$\hat{C}_p$	1.07	0.05	< 0.0001		
COPD	Intercept	2.24	0.57	< 0.0001	0.55	10.6
	$\hat{C}_p$	1.01	0.06	< 0.0001		
Healthy	Intercept	2.38	0.63	< 0.001	0.45	8.4
	$\hat{C}_p$	0.85	0.08	< 0.0001		

C_p = intercept + (β × $\hat{C}_p$).

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

Parameter	Estimate	SE	Wald 95% confidence limit Lower	Wald 95% confidence limit Upper	*p*-Value
Elderly adults (model $R^2 = 0.06$, $p = 0.16$)					
Intercept	0.70	0.49	–0.26	1.66	0.152
Health status					
CHD	0.27	0.52	–0.74	1.28	0.596
COPD	1.23	0.52	0.22	2.24	0.017
Outdoor errands	0.02	0.01	0.01	0.03	0.002
Cooking, indoors	0.01	0.01	0.00	0.02	0.024
In yard, outdoors	0.04	0.02	0.00	0.07	0.043
Asthmatic children (model $R^2 = 0.19$, $p = 0.06$)					
Intercept	1.27	1.81	–2.27	4.81	0.482
At school indoors	0.01	0.01	0.00	0.03	0.006
In bus or shuttle	0.05	0.02	0.01	0.09	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 more 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; Ebelt 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 Ebelt 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_{inf}. 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 (σ^2_ε), the variance of the ambient generated PM (σ^2_x), and the attenuation from ambient PM to personal exposure (α), which is the sum of the fraction of time spent outdoors (y) and the fraction of time spent indoors ($1 - y$) times F_{inf} [i.e., $\alpha = y + (1 - y) \times F_{inf}$]. Note that α can be approximated by F_{inf} 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

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

where $R = \sigma^2_\varepsilon/\sigma^2_x$. Simulated longitudinal correlation plots for longitudinal r and F_{inf} 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, σ^2_ε, is generally smaller than the variance in ambient originated PM, σ^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 α. 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 (Ebelt 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

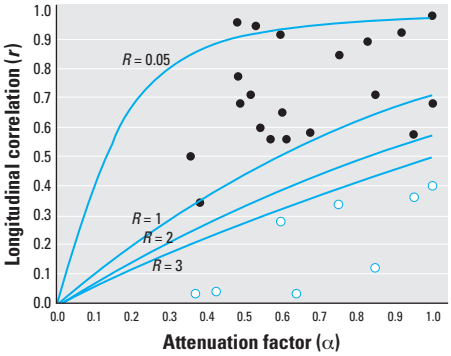

Figure 5. The F_{inf} is the only significant predictor for longitudinal correlation (r). The open circles are data points for which the variance of nonambient-originated PM (σ_ε) is three times higher than the variance of ambient-originated PM (σ_X). $r = 1/(1 + R/\alpha^2)^{1/2}$. $R = \sigma^2_\varepsilon/\sigma^2_X$. $n = 33$.

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/m^3 for the elderly groups.

Only 39% of the outdoor PM contributed to personal $PM_{2.5}$ exposure, as estimated by the fixed-effect model (Equation 2) (Table 7); this indicates that personal $PM_{2.5}$ 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 (R^2 = 0.5–0.55) for the elderly groups. Therefore, given the time–activity pattern and microenvironmental concentrations, the elderly susceptible subjects' $PM_{2.5}$ exposures are relatively predictable.

Conclusions

The average personal $PM_{2.5}$ exposures that we found among sensitive subpopulations in Seattle were similar to the average outdoor $PM_{2.5}$ concentrations but significantly higher than average indoor concentrations. The elderly subjects' personal $PM_{2.5}$ exposures were lower than those reported for other elderly subjects in other cities. The personal $PM_{2.5}$ exposure in the asthmatic children in this study, who lived in nonsmoking households, was also lower than those found elsewhere. $PM_{2.5}$ and PM_{10} concentrations, as well as the ratio of $PM_{2.5}$ to PM_{10} concentration, vary seasonally; higher concentrations were found indoors and outdoors during the heating season. A similar seasonal variation was also found for personal $PM_{2.5}$ exposures. Personal $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations than did the elderly healthy subjects. The child subjects experienced the highest indoor $PM_{2.5}$ and PM_{10} concentrations. Although a wide range of longitudinal correlations between central-site and personal $PM_{2.5}$ measurements was found, our results show that the longitudinal r is closely related to the particle infiltration efficiency of each residence. The $PM_{2.5}$ 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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