Longitudinal variability in outdoor, indoor, and personal PM$_{2.5}$ exposure in healthy non-smoking adults

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Abstract

Multiple 24-h average outdoor, indoor and personal PM$_{2.5}$ measurements were made in a population of healthy non-smoking adults from the Minneapolis-St. Paul metropolitan area between April and November 1999. Personal (P) PM$_{2.5}$ concentrations were higher than indoor (I) concentrations, which were higher than outdoor (O) concentrations. For 28 adults with a median of 9 (range 5–11) measurements per person, the distribution of longitudinal (i.e., within-person) correlation coefficients between P and I was moderate (median $r = 0.45$). The distribution of longitudinal correlation coefficients between I and O concentrations showed that these variables were less strongly associated (median $r = 0.25$; 28 residences; measurement median $n = 10$ per residence, range 7–13), and the distribution of P and O correlation coefficients (median $r = 0.02$; 29 subjects; measurement median $n = 11$ per subject, range 7–15) showed little statistical relation between these two variables for a majority of participants. A sensitivity analysis indicated that correlations did not increase if days with exposure to environmental tobacco smoke or occupational exposures were excluded. On average these adults spent 91% of their time indoors, and the mean of the average PM$_{2.5}$ "personal cloud" was 15.3 μg/m^3. Participants who had the largest personal cloud values tended to work outside the home and spent more time outdoors than subjects who did not work outside the home. In this population of healthy non-smoking adults, personal exposure to PM$_{2.5}$ does not correlate strongly with outdoor central site PM$_{2.5}$ concentrations.
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1. Introduction

Time-series epidemiological studies have shown a statistical association between mortality and morbidity and day-to-day variability in outdoor particulate matter < 10 μm (PM$_{10}$) measured at central sites (Pope et al., 1992; Dockery et al., 1993; USEPA, 1996; Vedal, 1997; Samet et al., 2000). Scientists have hypothesized that fine particles < 2.5 μm (PM$_{2.5}$) may be more closely linked with health effects, especially in children and adults with chronic lung disease. Statistical associations exist between PM$_{10}$ (and PM$_{10}$ in concert with other pollutants) and hospital admissions for chronic lung diseases for the elderly in the Minneapolis-St. Paul Metropolitan area (Schwartz, 1994; Moolgavkar et al., 1997), which has relatively low ambient PM$_{2.5}$ concentrations compared to other major urban areas in the United States (Adgate et al., 2002).

Epidemiological studies examining the statistical associations between PM exposure and health outcomes assume that central site outdoor monitors provide a reasonable estimate of personal exposure across the population. These studies hypothesize that particles of ambient origin penetrate indoors where people spend a

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majority of their time. Therefore, ambient particles are correlated over time with personal exposure despite the existence of indoor sources and the "personal cloud", i.e., exposure to airborne particles resulting from personal activities (e.g., occupation, hobbies, etc.) or physical activities (e.g., jogging, operating a vacuum cleaner, etc.) by the monitored subject (Rodes et al., 1991; McBride et al., 1999). For any 24 h period total personal exposure to PM is the sum of particles from three sources: personal activity (including occupational), indoor generated particles, and outdoor PM (Wilson et al., 2000), although the commonly used estimation of the personal cloud in particle exposure studies does not distinguish between particles from personal activity and indoor generated particles (Ozkaynak et al., 1996a, b). Studies have indicated that the correlation between ambient PM_{10} and personal exposure is much stronger if the analysis is conducted longitudinally, i.e., within participants over time, as opposed to cross-sectional correlation coefficients that treat each measure as independent (Janssen et al., 1997, 1998, 1999; Wallace, 2000). More recent longitudinal personal monitoring studies have indicated that personal $PM_{2.5}$ is even more strongly associated with ambient $PM_{2.5}$ (Ebelt et al., 2000; Janssen et al., 2000; Rojas-Bracho et al., 2000; Sarnat et al., 2000; Williams et al., 2000a). All these studies examined what are thought to be sensitive populations, but there is less data on $PM_{2.5}$ exposure in healthy adults (Ozkaynak et al., 1996a, b; Brauer et al., 2000).

The objective of this study was to examine the statistical associations between outdoor, indoor, and personal $PM_{2.5}$ concentrations measured over multiple days and seasons in healthy non-smoking adults from the Minneapolis-St. Paul metropolitan area. We also explore the implications of these results for assessing $PM_{2.5}$ exposures in the general population.

2. Methods

2.1. Study design, population, and data collection

This study obtained repeat measurements of $PM_{2.5}$ at outdoor central sites, within households, and near the breathing zone of non-smoking adult participants between April and November 1999. It was conducted as part of research examining exposure to a suite of hazardous air pollutants in three communities in the Minneapolis-St. Paul metropolitan area (Pratt et al., 1998). The study rationale, design, and sampling methods are briefly summarized here, and a detailed description has been published (Adgate et al., 2002).

In this study healthy adults were recruited from the Battle Creek (BCK), East St. Paul (ESP), and Phillips (PHI) communities by house-to-house canvassing and direct solicitation. After informed consent was obtained,

participants completed a baseline questionnaire to determine smoking status, socio-demographics, occupation, and housing characteristics. All participants were non-smokers, and only one reported living with a smoker (who did not report smoke inside their residence). A total of 32 healthy non-smoking adult participants (23 females, 9 males; mean age 42 ± 10, range: 24–64 years) were monitored during the spring (26 April–20 June), summer (21 June–11 August), and fall (23 September–21 November) of 1999. Outdoor central site samples (O) were collected near the approximate geographic center of each neighborhood, and monitors ran from midnight to midnight for two consecutive 24-h periods, followed by a day to change filters. Thus two sequential 24-h average O concentrations were obtained and a new sampling session was started every third day. For each participant a monitoring session consisted of 2 consecutive days, and was conducted so that the two 24-h average matched indoor (I) and personal (P) measurements were collected in concert with O samples in each community. Up to 15 days of P and I monitoring were collected for each participant.

Monitors were placed inside each participant's residence in the room where he/she reported spending the majority of their waking hours to obtain I measurements. Participants also carried personal pumps in small bags to obtain P measurements. For participants' convenience and logistical reasons I and P monitors were distributed and collected from participant homes in the evening (usually between 5 and 9 pm). Start times for indoor and personal monitors were always within a few minutes of each other. The average overlap between P/I and O measurements was 72% ($\sim$17 h) (Adgate et al., 2002). On sampling days participants completed a time-activity diary, recording time spent in seven primary microenvironments and documenting data related to exposure to tobacco smoke and other potential modifiers of PM exposure, such as occupation, hobbies and household ventilation.

Gravimetric O $PM_{2.5}$ concentrations were obtained using a federal reference method sampler and EPA site requirements for ambient sampling. Gravimetric concentrations for P and I samples were collected using $PM_{2.5}$ inertial impactor environmental monitoring inlets and air sampling pumps. The detection limit, defined as three times the standard deviation of the field blanks divided by the average sampled air volume, was $0.8\,\mu g/m^3$ for O central site samples, $3.6\,\mu g/m^3$ for I samples, and $7.5\,\mu g/m^3$ for P samples. One hundred percent of O, 95% of I, and 90% of P concentrations were greater than their respective detection limits.

2.2. Statistical analysis

SAS® (Version 8.01, SAS Institute, Inc., Cary, NC) was used for statistical analyses. Summary statistics

were first calculated by pooling all samples by type (P, I, and O), and then by calculating summary statistics for each participant, including ratios (P/O, P/I, and I/O), differences ($P - O$, $P - I$, and $I - O$), and longitudinal correlation coefficients (PO, PI, and IO). Concentrations less than the detection limit were used in calculations of summary statistics (as opposed to substituting an arbitrary value), and invalid samples (e.g., due to pump failures, etc.) were treated as missing values. Although significant differences in mean O $PM_{2.5}$ concentrations were observed between the ESP and BCK communities during the study period, in general O concentrations among the three communities had high correlation coefficients and relatively small absolute differences (Adgate et al., 2002). Therefore, missing O values in any community were estimated using the mean value from the other two communities ($n = 28$ days) or by the single community for which a valid sample was available ($n = 17$ days).

PO, IO, and PI regressions and correlation coefficients were calculated for each participant in a manner similar to previously published studies (Janssen et al., 1997). The models used were:

$$PO: \quad C_{P,it} = \alpha_{iPO} + \beta_{iPO} \times C_{Ot} + \varepsilon_{PO}, \tag{1}$$

$$PI: \quad C_{P,it} = \alpha_{iPI} + \beta_{iPI} \times C_{Ot} + \varepsilon_{PI}, \tag{2}$$

$$IO: \quad C_{I,it} = \alpha_{iIO} + \beta_{iIO} \times C_{Ot} + \varepsilon_{IO}, \tag{3}$$

where C is measured $PM_{2.5}$ concentration, i is participant, t is day, and α, β, and ε represent the intercept, slope estimate, and error term, respectively, in the regression model. Most of the distributions of regression parameters and outputs across individuals were skewed, so summary statistics present the median and range of values. To test the effect of periodic occupational and environmental tobacco smoke (ETS) exposures on these correlation coefficients in this non-smoking population, a sensitivity analysis was conducted by excluding days with recorded exposure to occupational PM sources or to ETS. The same process was used to test the effect of including personal and indoor measurements below the detection limit. Estimates of the "personal cloud" (PC) were modeled using $PM_{2.5}$ concentrations measured indoors and outdoors and time activity patterns as described previously (Adgate et al., 2002).

3. Results

3.1. Sample capture

Pooled 24-h average O, I, and P $PM_{2.5}$ concentrations for all participants and days show that the mean and variability of P was greater than I, and that the mean and variability of I was greater than O (Table 1). An average of 9 I and 10 P samples were collected for each participant. The I average is lower due to pump failures early in the study (Adgate et al., 2002). Twenty-eight of the 32 recruited participants had at least 6 days of valid I samples: one participant who was monitored in all three seasons had 4 valid I samples, and three participants (with 2, 3, and 5 days of monitoring) left the study before completion. At least one valid I measurement was collected for 23, 27, and 29 participants in the spring, summer, and fall seasons, respectively. All 29 of the participants who completed the study had at least 6 days of valid P samples. At least one valid P measurement was collected for 29 participants in the spring, 28 in the summer, and 28 in the fall.

3.2. Summary statistics within participants

Average ratios (P/O, P/I, and I/O) and differences between measurements ($P - I$, $P - O$, and $I - O$) were calculated for each participant and then summary statistics calculated for all participants. The mean of the average P/O ratio was 4.6 ± 4.3 (range 1.3–21.2), the mean of the average P/I ratio was 2.8 ± 2.1 (range 1.1–9.7), and the mean of the average I/O ratio was 1.6 ± 1.1 (range 0.3–5.6). For each participant the mean of the average $P - O$ value was $22.1 \pm 19.3\,\mu g/m^3$ (range 0–75.0), the mean of the average $P - I$ value was $15.2 \pm 17.8\,\mu g/m^3$ (range 0.8–67.4), and the mean of the average $I - O$ value was $3.2 \pm 8.0\,\mu g/m^3$ (range –11.0–33.9).

Table 1
Twenty-four hour average outdoor, indoor, and personal $PM_{2.5}$ samples. All values in $\mu g/m^3$, except GSD

Location	N	GM[a]	GSD[a]	Mean	SD	Range
Outdoor (O)	270[b]	8.6	1.8	10.1	6.2	1.0–41.6
Indoor (I)	294[c]	10.7	2.0	13.9	14.5	1.3–130
Personal (P)	332[c]	19.0	2.1	26.4	30.2	2.2–297

[a] Geometric mean ($\mu g/m^3$) and standard deviation (unitless).
[b] Number of independent samples collected outdoors in 3 communities over 112 calendar days.
[c] Number of valid indoor and personal samples in 32 participants over 112 calendar days.

A box plot of all I measurements and the number of samples collected in each household is shown in Fig. 1. The mean of the average I concentration for each household was $13.5 \pm 8.0 \, \mu g/m^3$ (range 4.5–43.3). A box plot of all P measurements and the number of samples collected for each participant are shown in Fig. 2. The mean of the average P concentration for each participant was $27.8 \pm 15.5 \, \mu g/m^3$ (range 11.6–82.9). There was

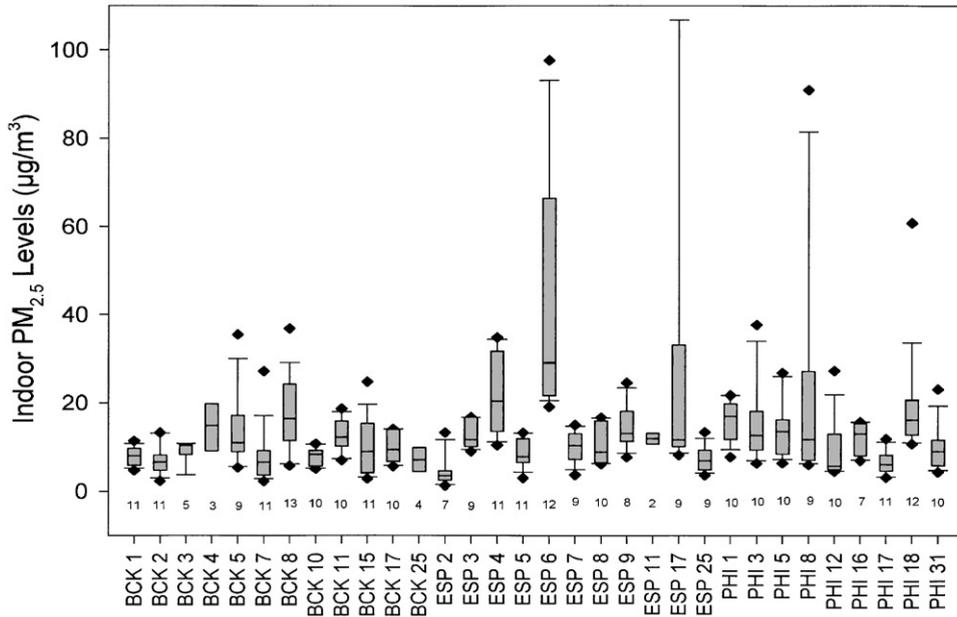Fig. 1. Box lot of indoor $PM_{2.5}$ concentrations ($\mu g/m^3$) for all participants. Number below each box indicates the number of valid measurements in that household.
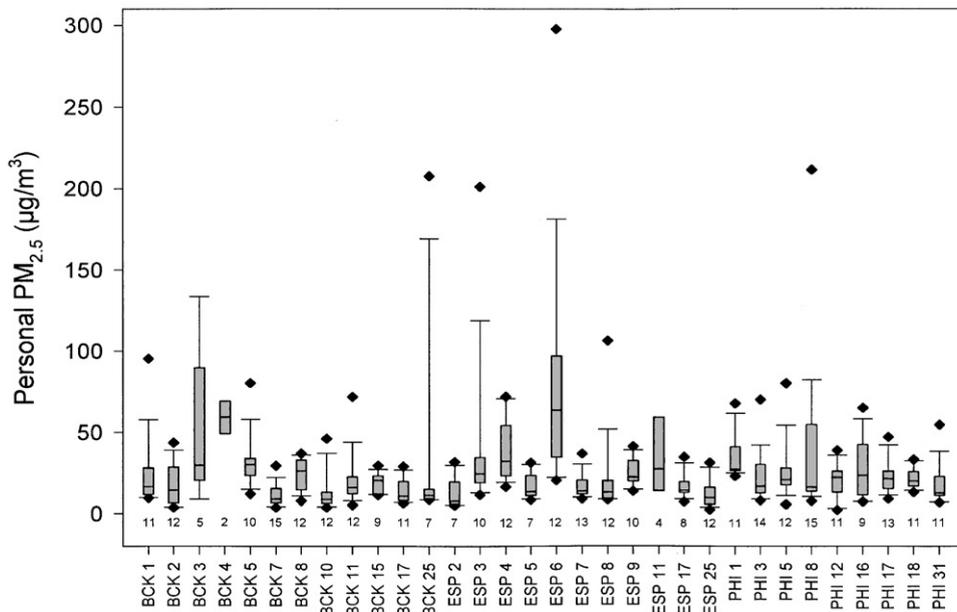

Fig. 2. Box lot of personal $PM_{2.5}$ concentrations ($\mu g/m^3$) for all participants. Number below each box indicates the number of valid measurements for that participant.

only one I measurement $> 100\,\mu g/m^3$, and P levels $> 100\,\mu g/m^3$ coincided with occupational or ETS exposure recorded on participant time-activity diaries.

3.3. Longitudinal correlation within participants

A summary of the longitudinal correlation coefficients between measurements (PO, PI, and IO) for participants with 7 or more days of monitoring are presented in Table 2. Pearson and Spearman correlation coefficients gave similar results in most cases. We chose to present the median and range of Pearson correlation coefficients along with the slope and intercept so these results could be compared with other published studies. The median PI correlation coefficient (0.45) across all individuals was higher than the median IO (0.25) or PO (0.02) correlation coefficients. The overall PI regression model was statistically significant ($p < 0.05$) for 7 of 29 participants, higher than for IO (3 of 28) or for PO (1 of 29) participants. This is likely a consequence of the large percentage of time most participants spent indoors at home: on average participants reported spending 91% of their time indoors (range 70–99%), and much of that time at home (Table 3).

To test the influence of periodic elevated concentrations on the overall correlation within participants we performed a sensitivity analysis in which we excluded days with reported ETS or occupational exposures (Table 2). Exposure to ETS was reported on 13.5% of P monitoring days, and exposure to fumes (diesel or occupational) was reported on 17.8% of P monitoring days. The effect of excluding these days reduced the number of participants with at least 6 days of matched P and O measurements to 23 and shifted the distribution of PO correlation coefficients to the left, but it did not change median correlation coefficients substantially (Fig. 3). The effect of excluding these days also reduced

the number of participants with at least 6 days of matched P and I and I and O measurements. It did not change the median or range of IO correlation coefficients substantially, but did lower the median PI correlation coefficient from 0.45 to 0.17 and reduce the range of values slightly. Finally, removing the days in which P and I samples were below the limit of detection had a minimal effect on the overall distribution of correlation coefficients.

3.4. Personal cloud calculations

The mean of the average PC was $15.3\,\mu g/m^3$ (range 0.7–67.8) and large positive and negative values were associated with occupational or ETS exposures or activity patterns. Table 3 links PC with PO, PI, IO correlation coefficients for each participant and sorts them by PC. PC levels were highest in participants who worked outside the home and had more active lifestyles, as indicated by the correlation coefficient between the percentage of time spent outdoors and the PC ($r = 0.32$, $p < 0.0001$) (Adgate et al., 2002). The top three mean PC values (all $> 60\,\mu g/m^3$) were in male participants who led active lifestyles, but these values represent 6 monitored days. Five of the 6 lowest mean PC values were in female participants, 4 of whom did not work outside the home. Three of these subjects had negative PO correlation coefficients (-0.31, -0.32, -0.52), and the single subject from this group who had a relatively high positive PO correlation coefficient spent 2–3 fold more time outdoors than other subjects in this group.

4. Discussion

This examination of the longitudinal temporal variability between P, I, and O PM$_{2.5}$ concentrations in a

Table 2
Within-participant regression analysis for personal (P), indoor (I), and outdoor (O) PM$_{2.5}$ measurements

Model (No. of participants)	$P = O$ ($n = 29$; Eq. (1))		$I = O$ ($n = 28$; Eq. (2))		$P = I$ ($n = 28$; Eq. (3))	
	Median	Range	Median	Range	Median	Range
Days monitored	11	7–15	10	7–13	9	5–11
Intercept ($\mu g/m^3$)	20.6	1.40–111	8.4	−1.5–65.9	10.4	−146–92.7
Slope	0.09	−3.5–2.7	0.31	−2.2–2.1	0.72	−8.1–15.2
Pearson's r	0.02	−0.52–0.94	0.25	−0.45–0.88	0.45	−0.55–0.98
Results if days with tobacco and occupational exposures excluded						
	$P = O$ ($n = 23$)		$I = O$ ($n = 22$)		$P = I$ ($n = 18$)	
Days monitored	8	6–15	9	6–12	8	6–10
Intercept ($\mu g/m^3$)	20.9	−1.1–49.9	7.5	−3.5–129	11.0	−256–92.7
Slope	−0.11	−1.8–2.5	0.30	−7.5–2.1	0.41	−8.1–25.3
Pearson's r	−0.03	−0.67–0.83	0.28	−0.66–0.87	0.17	−0.55–0.87

Table 3
Correlation coefficients (Pearson r), mean personal cloud, and percent time spent outdoors and indoors for all study participants sorted from highest to lowest personal cloud

Part. ID	PO		PI		IO		Personal cloud ($\mu g/m^3$)		Mean percent time spent	
	N^a	Corr. (r)	N^a	Corr. (r)	N^a	Corr. (r)	Mean	SD	Outdoors[b] (%)	Indoors (%)
BCK25	3	0.10	3	0.56	4	0.72	67.8	113.0	29.6	70.4
BCK3	2	NA	2	NA	5	0.49	64.2	83.7	9.9	90.1
BCK4	1	NA	1	NA	3	0.99	61.3	NA	16.7	83.3
ESP3	7	−0.06	7	0.65	9	−0.35	36.1	66.6	15.7	84.3
BCK5	8	0.08	8	−0.35	9	0.88**	22.2	23.4	11.9	88.1
BCK1	8	−0.35	8	−0.55	11	0.20	20.7	29.4	5.4	94.6
ESP4	9	0.35	9	0.33	11	0.09	20.3	17.1	7.7	92.3
PHI1	7	−0.15	9	0.07	10	0.49	19.3	13.5	15.9	84.1
ESP8	6	0.18	6	0.72	10	0.03	18.3	35.6	10.8	89.2
PHI5	9	0.02	9	0.77*	10	−0.08	16.2	18.3	8.5	91.5
PHI17	11	−0.17	11	0.07	11	0.19	15.5	12.2	13.7	86.3
ESP9	8	0.51	8	0.40	8	0.70	10.8	9.4	10.9	89.1
PHI16	5	0.41	5	−0.32	7	0.48	10.6	25.8	8.8	91.3
ESP5	6	−0.29	6	0.64	11	0.52	9.9	7.4	6.3	93.8
PHI12	9	−0.08	9	0.66	10	0.36	9.9	9.5	8.4	91.6
BCK2	9	−0.18	10	0.13	10	0.29	9.7	15.3	6.3	93.8
ESP6	8	−0.23	8	0.98**	12	−0.29	9.0	6.0	5.7	94.3
BCK15	8	−0.46	8	−0.06	11	0.34	8.8	9.7	10.9	89.1
ESP2	6	0.72	6	0.88*	7	0.81*	8.2	5.7	14.4	85.6
BCK8	9	0.41	9	0.69*	13	0.20	7.7	8.2	14.5	85.5
PHI31	9	−0.07	9	−0.04	10	−0.05	7.1	16.1	5.3	94.7
ESP7	9	0.21	9	0.49	9	0.43	6.4	6.4	3.9	96.1
PHI18	11	0.12	11	0.28	12	0.17	5.9	6.8	4.3	95.7
BCK17	9	0.02	9	0.09	10	0.29	3.9	9.0	0.6	99.4
BCK7	9	0.48	10	0.87**	10	0.70	3.8	4.4	6.3	93.8
PHI8	8	0.07	8	0.24	9	0.49	3.7	37.4	4.8	95.2
ESP11	1	NA	1	NA	2	NA	3.4	NA	4.0	96.0
BCK10	8	−0.32	8	0.51	10	−0.21	2.6	8.5	3.1	96.9
BCK11	10	−0.52	10	0.42	10	−0.45	2.4	6.3	3.3	96.7
PHI3	10	0.38	10	0.67*	10	0.13	2.2	6.7	7.1	92.9
ESP17	7	−0.31	7	0.70	9	−0.45	0.9	3.7	4.1	95.9
ESP25	9	0.94**	9	0.74*	9	0.73*	0.7	2.7	13.8	86.2
MEAN	7.5	0.06	7.6	0.39	9.1	0.29	15.3		9.1	90.9
STD	2.6	0.34	2.7	0.40	2.5	0.39	17.8		5.7	5.7
MEDIAN	8	0.01	8	0.49	10	0.29	9.4		8.1	91.9
MIN	1	−0.52	1	−0.55	2	−0.45	0.7		0.6	70.4
MAX	11	0.94	11	0.98	13	0.99	67.8		29.6	99.4

NA—Not applicable.
*Overall model statistically significant at $p < 0.05$ (*) or $p < 0.01$ (**).
[a] Number of paired measurements.
[b] Includes time spent in transit.

population of healthy non-smoking adults found moderate correlation between P and I (median $r = 0.45$), a modest correlation between I and O (median $r = 0.25$), and a minimal (median $r = 0.02$) correlation between P and O PM$_{2.5}$ measurements. These PO correlation results do not change substantively if days with ETS, occupational exposures, or below detection limit measurements are excluded.

A related manuscript investigating cross-sectional relationships found that there was relatively little spatial and temporal variability in O PM$_{2.5}$ concentrations for the Minneapolis-St. Paul metropolitan area in 1999, and that concentrations were low compared to other major metropolitan areas in the United States (Adgate et al., 2002). While I levels were higher than O levels, the distribution of I PM$_{2.5}$ concentrations observed in this
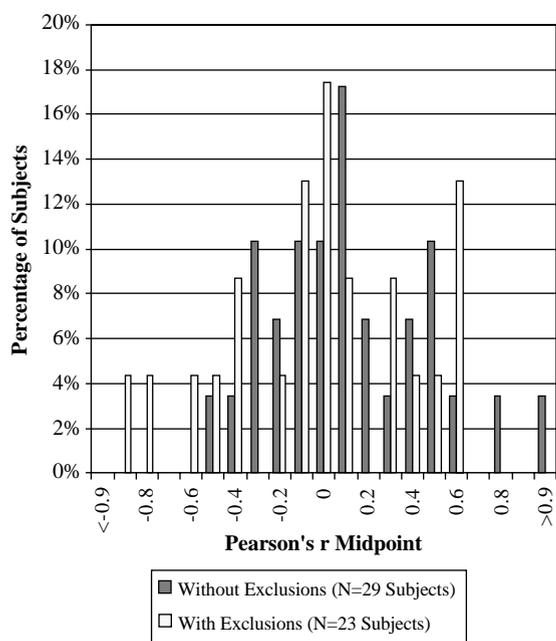
Fig. 3. Distribution of Personal to Outdoor Pearson's correlation coefficients: the "without exclusions" series has all 29 participants with 7–15 days of monitoring, and the "with exclusions" series has 23 participants with 6–15 days of monitoring, after excluding days with recorded tobacco or occupational exposures.

study is similar to those observed in non-smoking households in North America (Ozkaynak et al., 1996a, b; Pellizzari et al., 1999; Rojas-Bracho et al., 2000; Williams et al., 2000b). Although there are relatively few PM$_{2.5}$ P monitoring studies (Table 4), they consistently find that median P levels are higher than I levels. The distribution of P PM$_{2.5}$ concentrations observed in this study was higher than those reported for the populations summarized in Table 4. Values are similar to levels observed in a cross-sectional study that sampled California adults (Ozkaynak et al., 1996a, b) and lower than levels observed in working adults and high school students from Slovakia, who likely experienced more ETS exposure than our population (Brauer et al., 2000).

Of the three correlation coefficients calculated for this analysis, the PO correlation is of particular special interest in time series epidemiological studies because robust statistical associations have been documented between mortality/morbidity and ambient PM$_{10}$ levels in the United States (Samet et al., 2000). It has been hypothesized that the PO correlation should be even stronger for PM$_{2.5}$ because this size fraction more readily penetrates indoors (Vedal, 1997). The longitudinal P PM$_{2.5}$ monitoring studies in the peer-reviewed literature (summarized in Table 4) have shown relatively consistent results: strong longitudinal correlation coeffi-

cients that are higher than cross-sectional correlation coefficients. The seven studies in Table 4 were conducted in cohorts of 10 to 47 subjects presumed to be more sensitive to PM-associated health effects. The number of days monitored ranged from 5 to 20, with median longitudinal PO correlation coefficients ranging from 0.35 to 0.86.

Our study was similar to or larger in size and number of repeat measurements within each participant than many other studies, but there are at least three potential overlapping reasons that may explain why observed median PO correlation coefficients are lower in this study. First, this healthy adult population most likely has substantially different activity patterns than the populations in Table 4, which were older adults with chronic obstructive pulmonary/cardiovascular disease or children. Our subjects were active adults, who likely have higher and more varied exposures as a result of being closer to indoor particle sources, such as cooking and cleaning (Abt et al., 2000). The only other study with repeat PM$_{2.5}$ measurements in healthy adults had the lowest median PO correlation coefficient (0.35) among those summarized in Table 4 (although it lumps adult and children's results) (Wallace, 2000). Second, the relatively low median correlation coefficients observed in this study may result from the relatively low variability in O measurements in Minneapolis-St. Paul (Adgate et al., 2002). Lastly, our measurements were spread out over three seasons, whereas most other studies employed a "panel" study design, where subjects are measured repeatedly over a relatively short time, i.e., days or weeks within a season.

PI correlation coefficients in this study were relatively high, most likely because (1) participants spent a majority of their time indoors at home, (2) because I PM$_{2.5}$ is likely to be spatially homogenous as a result of relatively long airborne residence times, and (3) because indoor PM$_{2.5}$ sources are typically associated with human activities, such as cleaning, cooking, and movement (Ozkaynak et al., 1996a, b; Abt et al., 2000; Rojas-Bracho et al., 2000). Support for this interpretation is provided by the PC levels observed for these participants, which where higher than those observed for an elderly population in Baltimore (Williams et al., 2000a), but in the range of those observed in other studies of more active participants (Wallace, 2000). Our PI correlation coefficients are similar to those observed in other studies that have examined this parameter (Janssen et al., 2000; Rojas-Bracho et al., 2000; Williams et al., 2000a). Our IO correlation coefficients were lower than observed in many studies, likely due to the combination of indoor sources and relatively low variability in observed O PM$_{2.5}$ concentrations in the Minneapolis-St. Paul metropolitan area.

In this study some uncertainty about the strength of PO and IO correlation coefficients is introduced by the

Table 4
Summary of PM$_{2.5}$ longitudinal personal monitoring studies in non-smoking participants: study populations, concentrations and correlation coefficients between personal and outdoor concentrations (after Wallace, 2000, and Ebelt et al., 2000)

Study population (location)	Mean age (range) (years)	N (participants) times # (or range) of measurements per participant	Mean/median personal exposure (range)(μg/m³)	Personal to outdoor cross-sectional correlation coefficient	Median (range) of personal to outdoor longitudinal correlation	Study
Healthy adults (Minneapolis-St. Paul)	42 (24–64)	29 × 7–15	26.4/19.0[a] (2.2–297)	0.06	0.02 (−0.52–0.94)	This study; (Adgate et al., 2002)
Adults >64 years (Baltimore)	75±7 (NR)	15 × (9–12)	26.7/23.1 (summer) 18.5/15.4 (winter)	0.53 (both seasons)[b]	0.76 (−0.21–0.95) 0.25 (−0.38–0.81)	(Sarnat et al., 2000)
COPD patients (Vancouver)	74 (54–86)	16 × 7	18.2/14.3[a] (2.2–90.9)	0.15	0.48	(Ebelt et al., 2000)
Angina Pectoralis and/or coronary heart disease	NR (50–84)	37 × 5–13 (Amsterdam)	24.3/15.3 (8.5–133.7)	NR	0.79 (−0.41–0.98)	(Janssen et al., 2000)
		47 × 6–9 (Helsinki)	10.8/10.0 (3.8–32.7)	NR	0.76 (−0.12–0.97)	
COPD patients (Boston)	NR (38–60)	18 × 6–17 (12 h samples)	21.6/17.3 (0.6–127.7)	NR	0.61 (−0.1 or 0.1 − 0.93)[c]	(Rojas-Bracho et al., 2000)
Elderly adults (Baltimore)	65+ (NR)	19 × 16–20	12.9/NR (2.4–47.8)	NR	0.79 (0.37–0.89)	(Williams et al., 2000a)
Children (Amsterdam)	11 (10–12)	13 × 8	28/NR (18.7–60)	0.41[d] 0.82[e]	0.86[d] (−0.11–0.99) 0.92[e] (0.63–0.97)	(Janssen et al., 1999)
Children/adults (PTEAM pre-pilot)	⩾11 (NR)	18 × 5–7	70/NR (NR)	0.04	0.12 (NR)	(Wallace, 2000)

NR—Not reported.
[a] Geometric mean.
[b] Square root of reported R^2, one extreme value removed.
[c] Square root of reported R^2.
[d] All measurements.
[e] Days with environmental tobacco smoke exposure removed, longitudinal correlations for 9 subjects.

24-h average P and I samples, which typically started between 5 and 7 pm and thus had an average overlap of 72% with the 24-h average O samples, which all started at midnight. This uncertainty may be less than it appears, however, because O measurements have moderately high autocorrelation (0.45, 0.46, and 0.52 for PHI, ESP, and BCK, respectively) from day to day within a monitoring session (Adgate et al., 2002). While it is not feasible to quantify this uncertainty, the magnitude of the model uncertainty introduced by the offset is likely less than that introduced by indoor sources and activity patterns of our healthy adult subjects because the median IO correlation coefficient (0.29) was substantially higher than the median PO correlation coefficient (0.01), even though the P and I measurements have the same amount of overlap with the O measurement. In our judgment the effect of human activity patterns, the non-panel study design that spread our measurements over three seasons, and the relatively low O PM$_{2.5}$ concentrations in Minneapolis-St. Paul are likely to have a bigger influence on the longitudinal associations between P, I, and O than the uncertainty introduced by temporal offset between P/I and O.

Studies have suggested that the most important determinants of the statistical relations between P, I and O include ventilation rates and the presence of air conditioning. A study of 15 elderly subjects in Baltimore demonstrated that during the summer the PO correlation was strongest for subjects who spent a large percentage of their time in well ventilated indoor environments (Sarnat et al., 2000). A recent study showed improved statistical relations between PM$_{10}$ and hospital admissions for heart disease if the analysis includes variables representing the prevalence of central air conditioning and proportion of traffic related particles (Janssen et al., 2002). In this study 14 of 32 residences reported having central air conditioning, with 10 of the homes in BCK, and 2 in both PHI and ESP. While households in BCK reported having their windows open fewer hours than those from ESP and PHI (Adgate et al., 2002), PO and IO correlations do not appear to vary substantially by community or presence of air conditioning. Additional analysis using mixed models that combine subjects and control for the presence of air conditioning, particle sources, and other important covariates may provide additional insights into the correlations between P, I, and O PM$_{2.5}$ measurements.

5. Conclusions

In healthy non-smoking adults we observed moderate median PI, modest median IO, and minimal median PO longitudinal correlation coefficients for PM$_{2.5}$ measurements. In this population neither P nor I monitors

provided a highly correlated estimate of exposure to O PM$_{2.5}$ over time. These results suggest that the studies showing relatively strong longitudinal correlation coefficients between P and O PM$_{2.5}$ for individuals sensitive to air pollution health effects do not necessarily predict exposure to PM$_{2.5}$ in the general population.

References

Abt, E., Suh, H.H., Allen, G., Koutrakis, P., 2000. Characterization of indoor particle sources: a study conducted in the metropolitan Boston area. Environmental Health Perspectives 108 (1), 35–44.

Adgate, J.L., Ramachandran, G., Pratt, G.C., Waller, L.A., Sexton, K., 2002. Spatial and temporal variability in outdoor, indoor, and personal PM2.5 exposure. Atmospheric Environment 36 (20), 3255–3265.

Brauer, M., Hruba, F., Mihalikova, E., Fabianova, E., Miskovic, P., Plzikova, A., Lendacka, M., Vandenberg, J., Cullen, A., 2000. Personal exposure to particles in Banska Bystrica, Slovakia. Journal of Exposure Analysis and Environmental Epidemiology 10 (5), 478–487.

Dockery, D.W., Pope 3rd, C.A., Xu, X., Spengler, J.D., Ware, J.H., Fay, M.E., Ferris Jr., B.G., Speizer, F.E., 1993. An association between air pollution and mortality in six US cities. New England Journal of Medicine 329 (24), 1753–1759.

Ebelt, S.T., Petkau, A.J., Vedal, S., Fisher, T.V., Brauer, M., 2000. Exposure of chronic obstructive pulmonary disease patients to particulate matter: relationships between personal and ambient air concentrations. Journal of the Air and Waste Management Association 50 (7), 1081–1094.

Janssen, N.A., Hoek, G., Harssema, H., Brunekreef, B., 1997. Childhood exposure to PM10: relation between personal, classroom, and outdoor concentrations. Occupational and Environmental Medicine 54 (12), 888–894.

Janssen, N.A., Hoek, G., Brunekreef, B., Harssema, H., Mensink, I., Zuidhof, A., 1998. Personal sampling of particles in adults: relation among personal, indoor, and outdoor air concentrations. American Journal of Epidemiology 147 (6), 537–547.

Janssen, N.A., Hoek, G., Harssema, H., Brunekreef, B., 1999. Personal exposure to fine particles in children correlates

closely with ambient fine particles. Archives of Environmental Health 54 (2), 95–101.

Janssen, N.A., de Hartog, J.J., Hoek, G., Brunekreef, B., Lanki, T., Timonen, K.L., Pekkanen, J., 2000. Personal exposure to fine particulate matter in elderly subjects: relation between personal, indoor, and outdoor concentrations. Journal of the Air and Waste Management Association 50 (7), 1133–1143.

Janssen, N.A., Schwartz, J., Zanobetti, A., Suh, H.H., 2002. Air conditioning and source-specific particles as modifiers of the effect of PM(10) on hospital admissions for heart and lung disease. Environmental Health Perspectives 110 (1), 43–49.

McBride, S.J., Ferro, A.R., Ott, W.R., Switzer, P., Hildemann, L.M., 1999. Investigations of the proximity effect for pollutants in the indoor environment. Journal of Exposure Analysis and Environmental Epidemiology 9 (6), 602–621.

Moolgavkar, S.H., Luebeck, E.G., Anderson, E.L., 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis-St. Paul and Birmingham. Epidemiology 8 (4), 364–370.

Ozkaynak, H., Xue, J., Spengler, J., Wallace, L., Pellizzari, E., Jenkins, P., 1996a. Personal exposure to airborne particles and metals: results from the particle team study in riverside, California. Journal of Exposure Analysis and Environmental Epidemiology 6 (1), 57–78.

Ozkaynak, H., Xue, J., Weker, R., Butler, D., Koutrakis, P., Spengler, J., 1996b. Project Summary, The Particle Team Study (PTEAM): Analysis of the Data, USEPA: EPA/600/R-95/098. Research Triangle Park, NC.

Pellizzari, E.D., Clayton, C.A., Rodes, C.E., Mason, R.E., Piper, L.L., Fort, B., Pfeifer, G., Lynam, D., 1999. Particulate matter and manganese exposures in Toronto, Canada. Atmospheric Environment 33, 721–734.

Pope 3rd, C.A., Schwartz, J., Ransom, M.R., 1992. Daily mortality and PM10 pollution in Utah Valley. Archives of Environmental Health 47 (3), 211–217.

Pratt, G., McCourtney, M., Wu, C., Bock, D., Sexton, K., Adgate, J., Ramachandran, G., 1998. Measurement and source apportionment of human exposures to toxic air pollutants in the Minneapolis-St. Paul metropolitan area. AWMA Specialty Conference: Measurement of Toxic and Related Air Pollutants, Cary, NC, Vol. 2, No. 2. Air and Waste Management Association, Pittsburgh, PA, pp. 64–72.

Rodes, C., Kamens, R., Wiener, R., 1991. The significance and characteristics of the personal activity cloud on exposure assessment measurements for indoor contaminants. Indoor Air 2, 123–145.

Rojas-Bracho, L., Suh, H.H., Koutrakis, P., 2000. Relationships among personal, indoor, and outdoor fine and coarse particle concentrations for individuals with COPD. Journal of Exposure Analysis and Environmental Epidemiology 10 (3), 294–306.

Samet, J.M., Dominici, F., Curriero, F.C., Coursac, I., Zeger, S.L., 2000. Fine particulate air pollution and mortality in 20 US cities, 1987–1994. New England Journal of Medicine 343 (24), 1742–1749.

Sarnat, J.A., Koutrakis, P., Suh, H.H., 2000. Assessing the relationship between personal particulate and gaseous exposures of senior citizens living in Baltimore, MD. Journal of the Air and Waste Management Association 50 (7), 1184–1198.

Schwartz, J., 1994. PM10, ozone, and hospital admissions for the elderly in Minneapolis-St. Paul, Minnesota. Archives of Environmental Health 49 (5), 366–374.

USEPA, 1996. Air Quality Criteria for Particulate Matter. Washington, DC, Office of Research and Development: EPA/600/P-95/001aF.

Vedal, S., 1997. Ambient particles and health: lines that divide. Journal of the Air and Waste Management Association 47 (5), 551–581.

Wallace, L., 2000. Correlations of personal exposure to particles with outdoor air measurements: a review of recent studies. Aerosol Science and Technology 32 (1), 15–25.

Williams, R., Suggs, J., Creason, J., Rodes, C., Lawless, P., Kwok, R., Zweidinger, R., Sheldon, L., 2000a. The 1998 Baltimore particulate matter epidemiology-exposure study: Part 2. Personal exposure assessment associated with an elderly study population. Journal of Exposure Analysis and Environmental Epidemiology 10 (6 Pt 1), 533–543.

Williams, R., Suggs, J., Zweidinger, R., Evans, G., Creason, J., Kwok, R., Rodes, C., Lawless, P., Sheldon, L., 2000b. The 1998 Baltimore particulate matter epidemiology-exposure study: Part 1. Comparison of ambient, residential outdoor, indoor and apartment particulate matter monitoring. Journal of Exposure Analysis and Environmental Epidemiology 10 (6 Pt 1), 518–532.

Wilson, W.E., Mage, D.T., Grant, L.D., 2000. Estimating separately personal exposure to ambient and nonambient particulate matter for epidemiology and risk assessment: why and how. Journal of the Air and Waste Management Association 50 (7), 1167–1183.