Journal of Exposure Analysis and Environmental Epidemiology (2005) 15, 17–28
© 2005 Nature Publishing Group All rights reserved 1053-4245/05/$30.00
www.nature.com/jea

Influence of ambient (outdoor) sources on residential indoor and personal $PM_{2.5}$ concentrations: Analyses of RIOPA data

QING YU MENG,[a] BARBARA J. TURPIN,[a,b] LEO KORN,[b] CLIFFORD P. WEISEL,[b] MARIA MORANDI,[c] STEVEN COLOME,[d,e] JUNFENG (JIM) ZHANG[b], THOMAS STOCK,[c] DALIA SPEKTOR,[f] ARTHUR WINER,[e] LIN ZHANG,[b] JONG HOON LEE,[a] ROBERT GIOVANETTI,[a] WILLIAM CUI,[a] JAYMIN KWON,[a,b] SHAHNAZ ALIMOKHTARI,[b] DEREK SHENDELL,[e] JENNIFER JONES,[e] CORICE FARRAR[e] AND SILVIA MABERTI[c]

[a]*Department of Environmental Sciences, Rutgers University, 14 College Farm Rd, New Brunswick, New Jersey 08901, USA*
[b]*Environmental and Occupational Health Sciences Institute, 170 Frelinghuysen Road, Piscataway, New Jersey 08854, USA*
[c]*School of Public Health, Houston Health Sciences Center, University of Texas, 1200 Hermann Pressler, Houston, Texas 77030, USA*
[d]*Integrated Environmental Sciences, 5319 University Drive #430, Irvine, California 92612, USA*
[e]*Environmental Science and Engineering Program, School of Public Health, University of California, Los Angeles, 650 Charles E. Young Drive, 46-081 CHS, Los Angeles, California 90095, USA*
[f]*Rand Co., 1700 Main St., Santa Monica, California 90401, USA*

The Relationship of Indoor, Outdoor and Personal Air (RIOPA) study was designed to investigate residential indoor, outdoor and personal exposures to several classes of air pollutants, including volatile organic compounds, carbonyls and fine particles ($PM_{2.5}$). Samples were collected from summer, 1999 to spring, 2001 in Houston (TX), Los Angeles (CA) and Elizabeth (NJ). Indoor, outdoor and personal $PM_{2.5}$ samples were collected at 212 nonsmoking residences, 162 of which were sampled twice. Some homes were chosen due to close proximity to ambient sources of one or more target analytes, while others were farther from sources. Median indoor, outdoor and personal $PM_{2.5}$ mass concentrations for these three sites were 14.4, 15.5 and 31.4 $\mu g/m^3$, respectively. The contributions of ambient (outdoor) and nonambient sources to indoor and personal concentrations were quantified using a single compartment box model with measured air exchange rate and a random component superposition (RCS) statistical model. The median contribution of ambient sources to indoor $PM_{2.5}$ concentrations using the mass balance approach was estimated to be 56% for all study homes (63%, 52% and 33% for California, New Jersey and Texas study homes, respectively). Reasonable variations in model assumptions alter median ambient contributions by less than 20%. The mean of the distribution of ambient contributions across study homes agreed well for the mass balance and RCS models, but the distribution was somewhat broader when calculated using the mass balance model with measured air exchange rates.
Journal of Exposure Analysis and Environmental Epidemiology (2005) **15,** 17–28. doi:10.1038/sj.jea.7500378
Published online 12 May 2004

Keywords: *$PM_{2.5}$, RIOPA, exposure, ambient-generated PM.*

Introduction

Numerous epidemiologic studies have shown a positive association between ambient concentrations of airborne particulate matter (PM) and excess respiratory and cardiovascular mortality and morbidity (US EPA, 1996; Norris et al., 1999; Schwartz et al., 1999; Goldberg et al., 2001). This implies an association between community exposure to PM of ambient (outdoor) origin and adverse health effects. Several personal exposure studies have found poor correlations between ambient $PM_{2.5}$ concentrations and personal exposure measurements (Sexton et al., 1984; Spengler et al., 1985; Morandi et al., 1988; Wallace, 1996; Pellizzari et al., 1999; Lachenmyer and Hidy, 2000; Oglesby et al., 2000). This seeming contradiction is partly due to the fact that measured personal exposure includes not only contributions from ambient PM sources, but also contributions from indoor and in-vehicle sources and personal activities, which vary with time and from person-to-person. Estimates of exposure to PM of ambient origin are needed to guide the development of control strategies for ambient PM and to evaluate exposure error introduced by using central-site ambient PM as a surrogate for exposure to PM of ambient origin in epidemiological studies.

Epidemiologic and exposure studies have generated great interest in characterization of indoor particles since people spend most of their time indoors. US residents spend approximately 87% of their day indoors, 7% in vehicles and only 6% outside (Robinson and Nelson, 1995). Typically, indoor PM consists of ambient particles that infiltrate indoors and remain suspended, particles emitted

1. Address all correspondence to: Dr. B.J. Turpin, Department of Environmental Sciences, Rutgers University, 14 College Farm Road, New Brunswick, NJ 08901, USA. Tel.: +1-732-932-9540.
Fax: +1-732-932-8644. E-mail: Turpin@aesop.rutgers.edu
Received 1 October 2002; accepted 2 February 2004; published online 12 May 2004

indoors (primary), and sometimes PM formed indoors through reactions of gas-phase precursors emitted both indoors and outdoors (Wallace, 1996; Weschler and Shields, 1997; Wainman et al., 2000). When indoor sources are present, indoor PM can be substantially higher than outdoor PM concentrations (Weschler and Shields, 1997). Indoor PM sources include smoking, cooking, gas stoves, cleaning, washing and walking (Lefcoe and Inculet, 1975; Yocom, 1982; Ozkaynak et al., 1996; Chao et al., 1997; Abt et al., 2000a; Jones et al., 2000). Notably, little is known about the health effects of particles generated indoors.

Outdoor $PM_{2.5}$ is also a main contributor to indoor particle concentrations in both naturally and mechanically ventilated structures (Thatcher and Layton, 1995; Weschler et al., 1997; Abt et al., 2000a). Outdoor particles can enter indoor environments by convective flow (e.g. through an open window) or by diffusional flow (i.e. infiltration) through cracks and fissures in the barrier of the building envelope. Contributions of outdoor sources to indoor $PM_{2.5}$ concentrations of 23–67% have been estimated in a handful of studies (USEPA, 1996; Wallace, 1996).

Several mechanistic and statistical models have been applied to describe important factors affecting indoor PM. These include steady state, single-compartment mass balance and random component superposition statistical models (Wallace, 1996; Ott et al., 2000). The single-component mass balance model assumes that homes can be described as completely mixed flow reactors, where the indoor PM concentration depends on the outdoor PM concentration, air exchange rate, the rate of generation and resuspension of PM indoors, the rate of removal of PM by reaction or surface deposition, and the home volume. The basic mass balance model has two assumptions: perfect instantaneous mixing and factors affecting indoor concentrations are steady or change slowly throughout the monitoring period. The penetration of particles through the building envelope, particle decay rate, and strengths of various indoor sources have been estimated in this way in PTEAM and a small number of other studies (Ozkaynak et al., 1996).

The Random Component Superposition (RCS) statistical model uses the linear regression of indoor or personal concentration on the outdoor PM concentration to estimate means and distributions of ambient (outdoor) and nonambient contributions to indoor or personal PM concentrations (Ott et al., 2000). The main assumption of this model is that the statistical distribution of nonambient concentrations is spatially and temporally invariant. The RCS model has been applied to PM_{10} data from PTEAM, THEES and Toronto (Ott et al., 2000). Other statistical methods used to apportion indoor and outdoor sources of indoor PM concentrations and to identify factors affecting indoor air quality include multivariate linear regression and receptor models using chemical species and/or time-activity information (Ozkaynak et al., 1996; Yakovleva et al., 1999).

A comprehensive assessment of the impact of ambient sources on indoor air quality would require detailed consideration of the physical and chemical mechanisms of PM formation, transformation, transport and deposition. Airborne PM consists of a mix of organic and inorganic substances, such as sulfates, nitrates, water, trace elements, oxidants and organic compounds with a variety of functional groups and spanning a wide range of vapor pressures and polarities (Ansari and Pandis, 1998; Seinfeld and Pandis, 1998; Turpin et al., 2000). Particles emitted from different sources have different compositions, and their toxicities as well are expected to be distinct. This means it is quite possible that PM emitted indoors and PM emitted outdoors have different health effects. This reason, the need to reduce uncertainties in epidemiology studies, and the need to differentiate between indoor and outdoor sources in the design of control strategies provide strong motivation for separation of PM exposure into its ambient (outdoor) and nonambient contributions.

This study presents indoor, outdoor and personal $PM_{2.5}$ mass concentrations, and estimates the ambient and nonambient contributions to indoor and personal $PM_{2.5}$ mass concentrations using data collected in the study entitled "Relationship of Indoor, Outdoor and Personal Air" (RIOPA).

Methods

Sample Collection

The RIOPA study was designed, in part, to test and refine the current mechanistic understanding of the relationships between indoor, outdoor and personal exposure concentrations for several air pollutants. Of specific interest to the RIOPA study is the contribution of ambient outdoor sources to indoor and personal concentrations. In order to vary the pollution mix and the air exchange rate, approximately 100 nonsmoking homes from each of these three geographically distinct locations (Houston, TX; Los Angeles County, CA; and Elizabeth, NJ) were sampled across all four seasons (summer 1999–spring 2001). $PM_{2.5}$ samples were collected in more than half of these homes. The sampled homes included a variety of ages and types of home construction and ventilation practices. Homes in close proximity and farther away from primary sources (mobile sources/industry in New Jersey, mobile sources in California, and refineries in Texas) of volatile organic compounds (VOCs) and/or $PM_{2.5}$ were sampled. Homes were sampled one to four at a time, and most of the homes were sampled a second time approximately 3 months later. All integrated samples were collected for 48 h, to improve detection of trace species (e.g., PM elements and gas-phase carbonyls). Home volume, air exchange rate, time-activity information, temperature, relative humidity, VOC, aldehyde, PM mass and several PM

species concentrations were measured. Communication between participants and RIOPA field team members was conducted in English or Spanish. Additional details concerning the RIOPA study plan, sample collection and other analyses are provided by Weisel et al (2004) and Naumova et al. (2002, 2003) and subsequent publications.

A total of 212 homes were sampled for $PM_{2.5}$; 162 of these homes were sampled a second time. Residential indoor and outdoor $PM_{2.5}$ mass was measured on Teflon filters (37 mm, 2 μm pore, Pallflex Gelman Scientific, Ann Arbor, MI, USA) collected downstream of a single-jet, 2.5 μm diameter cut-point impactor (Harvard Impactor) at 10 lpm for 48 h.

Personal $PM_{2.5}$ mass concentrations were measured on Teflon filters (25 mm, 3 μm pore; Pallflex Gelman Scientific) collected for 48 h in modified MSP personal environmental monitors (PEM; MSP Co., Minneapolis) at 3.2 lpm. The PEM has a 10-jet impactor inlet designed to provide a 2.5 μm diameter cut-point when 0.4 lpm is maintained through each jet. In this study, two jets were blocked to achieve a 2.5 μm cut-point at 3.2 lpm. PEMs were also modified to hold a 25 mm, rather than 37 mm, filter to reduce detection limits for mass and other species. Flow was drawn through the PEM, and in some cases through an active carbonyl sampler connected in parallel, using an AFC 400S pump (B.G.I. Inc., Waltham, MA, USA). PEMs were placed on the front strap of the sampling bag near the breathing zone of the participant. The pump, battery pack and motion sensor (HOBO, Onset Computer Corp., Bourne, MA, USA) were worn in the sampling bag on the participant's hip or back. Since the study design called for PM sampling in only 50% of homes, participants who expressed concern about the sampler burden generally were not selected for PM sampling. Among the participants who carried the $PM_{2.5}$ sampler ($\sim$2 kg) the added burden did not appear to affect compliance.

A field blank of each filter type was transported with substrates to the field, kept near the indoor or outdoor sampler during sampling, and stored and analyzed with field samples from concurrently measured homes. Duplicate samples were collected with pairs of Harvard Impactors collocated inside or outside 35 study homes. In addition, 14 samples were collected with PEMs mounted next to the indoor Harvard Impactors for comparison of sampler performance.

Filters were loaded, unloaded and leak checked in the laboratory. Flow rates were measured at the beginning and end of each sampling period, and samplers were leak-checked at the end of the sampling period. Collected samples and field blanks were returned to the laboratory/shipped in coolers with blue ice packs and stored frozen until analysis.

Analysis

Teflon filters were weighed on a Cahn C-30 (Cahn Instruments, Inc., Cerritos, CA, USA) or a Mettlar MT5 (Mettler Toledo Inc., Columbus, OH, USA) microbalance in an EPA-audited laboratory at the Environmental and Occupational Health Sciences Institute according to EPA protocols for $PM_{2.5}$ mass. Filters were equilibrated before and after sampling for 24 h at 30–40% relative humidity (RH) and 20–23°C. Conditions for post-collection analysis were within 5% RH and 2°C of those for precollection analysis for each filter. Pre- and post-collection analyses were conducted by the same analyst on the same balance. Temperature and RH were recorded continuously in the weighting room. The balance was calibrated daily prior to filter weighing with a 200 ± 0.025 mg primary mass standard traceable to NIST mass standards. An independent standard (50 mg) was analyzed after every 10 filters to evaluate analytical accuracy. At least one laboratory blank was also weighed daily. All filters were weighed twice.

The average air exchange rate of each home over the 48-h sampling period was determined by measuring the house volume and the concentration of an inert nontoxic tracer. The tracer, perfluorinated methylcyclohexane, was emitted at a constant rate beginning approximately 48 h before sample collection. The tracer concentration was measured with a passive capillary adsorption tube and gas chromatography-electron capture detection, as described by Dietz et al. (1986). The maximum measurable air exchange rate was approximately 5 air changes/h.

Results and discussion

Quality Control and Quality Assurance

A total of 364 indoor, 369 outdoor and 383 personal $PM_{2.5}$ samples were collected. The present analysis includes only valid samples: validation required that flow rate changes were less than 15% during sampling and collection times exceeded 42 h (87.5% of target duration). Field and analytical comments, including comments regarding compliance with personal sampler protocols, were also used to identify invalid samples. Valid data comprise 91%, 82% and 83% of the samples collected in California, New Jersey and Texas, respectively.

The limits of detection for $PM_{2.5}$ mass concentrations, calculated as three times the standard deviation of the field blanks, are 0.55 $\mu g/m^3$ for indoor and outdoor samples and 1.4 $\mu g/m^3$ for personal samples. Field blank weights were not significantly different before and after transport to the field according to a paired t-test with $\alpha = 0.05$ ($N = 452$, $P = 0.24$). Therefore, no blank subtraction was performed for $PM_{2.5}$ mass measurements. All $PM_{2.5}$ mass concentrations were above detection limits.

Uncertainties in mass concentrations are clearly dominated by sampling uncertainties, as evidenced by very good analytical precision (better than 1% based on replicate sample analysis) and analytical accuracy (replicate analyses

of the 50 mg standard had a standard deviation of 0.002 mg). Overall measurement precision for indoor and outdoor mass concentrations is 17%, as a coefficient of variation, based on analysis of 35 pairs of collocated Harvard Impactor samples inside and outside of study homes and shown in Figure 1a. (The coefficient of variation is given by the pooled standard deviation (σ_{pooled}) divided by the mean value of the pairs. For paired data, $\sigma_{pooled} = [\Sigma d_i^2/2n]^{1/2}$, where d is the difference between pair i values and n is the number of pairs). Overall measurement accuracy for atmospheric PM is limited by sampling artifacts. During collection of fine PM on a sampling substrate, changes in relative humidity and changes in temperature alter the equilibrium partitioning of semi-volatile PM species such as ammonium nitrate, semivolatile organic compounds and water. Changes in conditions during weighing can also affect measurement accuracy. These changes are minimized in the EPA filter weighing protocol, which was used in this study.

Figure 1b shows $PM_{2.5}$ mass concentrations measured with PEM and Harvard samplers placed side-by-side in the indoor sampling rack of 14 RIOPA study homes. The Shapiro–Wilks tests suggest that both data sets are approximately lognormally distributed ($\alpha = 0.05$, $P = 0.40$ for Harvard and $P = 0.05$ for PEM). Concentrations measured by collocated Harvard and personal samplers are highly correlated (R^2 = 97%; 92% without highest point), suggesting that the PEM sampler has good precision. However, mass concentrations measured with the PEMs are significantly greater at the 95% confidence level than those measured with the Harvard samplers according to a t-test on the log-transformed data. The median concentrations measured by the Harvard and PEM samplers during collocated sampling were 11.6 and 13.5 $\mu g/m^3$, respectively. The linear least-squares regression of PEM mass measurements on collocated Harvard Impactor (HI) measurements is:

$$[PEM] = 0.92[HI] + 4.33 \quad (1)$$

where the 95% confidence intervals for slope and intercept are (0.81, 1.02) and (2.03, 6.63), respectively. Note that the confidence intervals for the slope include 1.0. These data were not used to "calibrate" one sampler against the other, because the scarcity of PEM-Harvard data above 30 $\mu g/m^3$ would make the accuracy of this correction uncertain for high-end exposures. Additionally, the regression equation is fairly sensitive to individual data points. For example, without the highest point the regression is

$$[PEM] = 1.15[HI] + 1.51 \quad (2)$$

where the 95% confidence intervals for slope and intercept are (0.92, 1.37) and (−1.66, 4.67), respectively. By Dixon's test ($\alpha = 0.05$), three outliers were identified. The regression excluding the three outliers yields:

$$[PEM] = 0.89[HI] + 4.42 \quad (3)$$

where the 95% confidence intervals for slope and intercept are (0.85, 0.92) and (3.57, 5.27), respectively. The size of the relative bias between samplers at the mean RIOPA personal exposure of 37.6 $\mu g/m^3$ is 1.4, 6.2 or 0.32 $\mu g/m^3$ (4%, 16% or 0.9%) according to Equations (1), (2) and (3), respectively. This accuracy is quite reasonable, considering $PM_{2.5}$ measurement precision. Intersampler differences of this size are not unusual for collocated measurements of $PM_{2.5}$. Differences can result from differences in the shapes of the collection efficiency curves for the 2.5 μm impactor precut, differences in bounce from the impaction plates, and/or differences in volatile losses. The Harvard Impactor has a single jet impactor inlet and a face velocity of 16 cm/s, whereas the modified PEM was operated with an 8 jet impactor inlet and a face velocity of 11 cm/s. Low face velocity samples are less susceptible to volatilization and bounce (Turpin et al., 2000). We expect examination of PM species concentrations to yield further insights into the inter-sampler differences (i.e. in a subsequent publication). Personal, indoor and outdoor mass concentrations reported below are measured values. The Harvard Impactor–PEM inter-sampler discrepancy introduces uncertainty in the

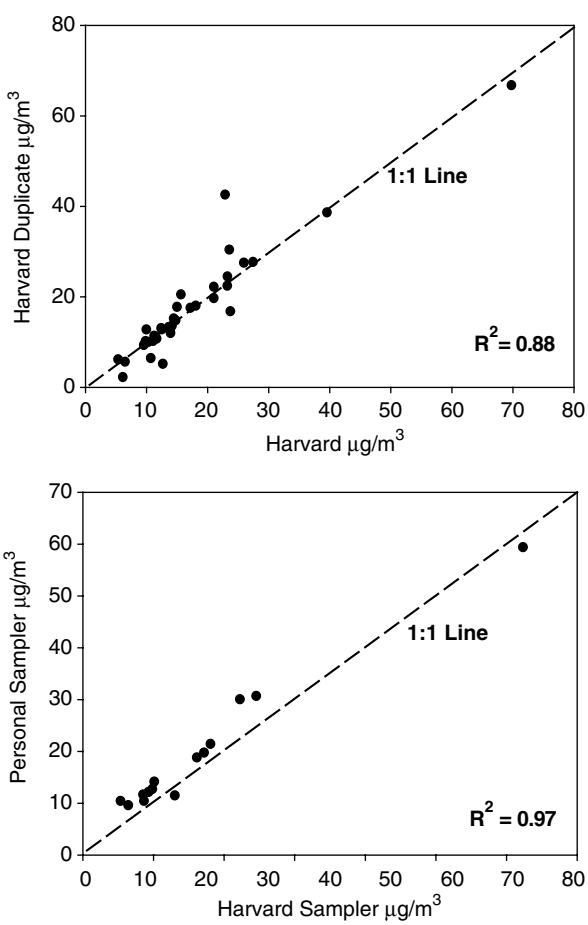

Figure 1. $PM_{2.5}$ concentrations from collocated samplers.

Table 1. Indoor, outdoor and personal $PM_{2.5}$ mass concentrations ($\mu g/m^3$) and air exchange rates (AER; h^{-1}).

Group	Category	Sample size	Mean	Median	STD
Overall study	Indoor	326	17.6	14.4	12.6
	Outdoor	334	18.1	15.5	10.7
	Personal	307	37.6	31.4	24.6
	AER	349	1.06	0.78	0.89
California	Indoor	124	16.2	14.5	9.4
	Outdoor	121	19.2	16.1	13.3
	Personal	106	29.3	26.6	14.8
	AER	131	1.22	0.93	0.87
New Jersey	Indoor	96	20.1	15.7	15.5
	Outdoor	103	20.4	18.2	10.7
	Personal	100	46.9	38.6	30.5
	AER	111	1.22	0.88	0.97
Texas	Indoor	106	17.1	13.4	12.7
	Outdoor	110	14.7	13.2	5.7
	Personal	101	37.2	31.8	23.4
	AER	107	0.71	0.46	0.73

magnitude of the difference between personal exposures and microenvironmental concentrations, rather than the correlation between these measurements. The effect of this uncertainty on subsequent analyses is noted below.

Indoor, Outdoor and Personal $PM_{2.5}$ Mass Concentrations
Summary statistics for the measured residential indoor, outdoor and personal $PM_{2.5}$ mass concentrations, and corresponding air exchange rates for the overall study and for each city are presented in Table 1. Median indoor, outdoor and personal $PM_{2.5}$ mass concentrations are 14.4, 15.5, and 31.4 $\mu g/m^3$, respectively. $PM_{2.5}$ mass concentrations and air exchange rates are approximately lognormally distributed, as suggested by a Shapiro–Wilks test on the log-transformed data ($\alpha = 0.05$). Personal $PM_{2.5}$ concentrations are significantly greater than indoor and outdoor concentrations as determined by one-way ANOVA and Sheffe's test ($\alpha = 0.05$, $P < 0.0001$) performed on the log-transformed data. This would still be true if PEM and Harvard Impactor measurements were "harmonized" using Equations (1), (2) or (3). Personal concentrations are also more variable than both indoor and outdoor concentrations according to a Levene's test ($\alpha = 0.05$) for the overall study data and data segregated by city, with the exception of California outdoor concentrations. Indoor concentrations for Texas homes are more variable than outdoor concentrations, while no significant difference in the variance of indoor and outdoor concentrations was found for California and New Jersey homes (Levene's test; $\alpha = 0.05$).

Within-home indoor, outdoor and personal concentrations were compared using an incomplete randomized block mixed model (SAS, version 8) by treating "home ID" as a random effect. The error correlations between each pair of samples were allowed to differ by including a repeated statement with an unstructured covariance matrix in the SAS script. The added power obtained by "pairing" indoor, outdoor and personal concentrations from the same home verified that personal concentrations are higher than indoor and outdoor concentrations for all three cities, and revealed that outdoor concentrations are higher than indoor concentrations for New Jersey and California homes, as well as for the overall data set. The same conclusions were drawn when only the first sample from each home was used in the analysis, confirming that the conclusions are not artifacts of within-home correlation.

Personal concentrations could be higher than residential indoor and outdoor concentrations because the participant spent time in another, higher-concentration microenvironment (e.g., a smoky bar or restaurant). In addition, higher personal concentrations could result if the participant was located in closer proximity to indoor sources than the indoor monitor (e.g., while cooking). It must be noted that while smokers were effectively excluded from this study (as validated by the personally administered activity questionnaire and technician observation), passive exposure to environmental tobacco smoke is a potential contributor to personal exposures. As part of the activity questionnaire, participants were asked if they had been in an area where smoking occurred during the sample collection period. Questionnaire responses suggest that passive tobacco smoke exposure influenced few samples (i.e., less than 15). It is unlikely that environmental tobacco smoke (ETS) exposure influenced the median personal exposures for the study, but ETS could be a significant contributor to the highest exposures. For example, two subjects reporting ETS exposure had personal exposure concentrations of 96.5 and 66.0 $\mu g/m^3$; these concentrations are greater than the 95th and 90th percentiles of measured personal exposure concentrations. Other personal activities can also have a large influence on personal exposures. These will be examined in a future publication through the use of questionnaires and chemically speciated data.

Figure 2 shows scatter diagrams of indoor, outdoor and personal $PM_{2.5}$ concentrations, and Table 2 provides coefficients of determination. Pooled indoor, outdoor and personal $PM_{2.5}$ mass concentrations are only poorly to moderately correlated ($R^2 = 1$–19% for NJ and TX; $R^2 = 21$–44% for CA), reflecting daily and home-to-home variations in indoor source strength, air exchange rates and personal activities. As one would expect, correlations between indoor and outdoor concentrations are much stronger for homes where the ratio of the indoor-to-outdoor mass concentration is less than 1 ($R^2 = 43$–80%; $I/O < 1$ in 54–71% of homes by state). The higher correlations occur presumably because of low indoor source strengths and/or

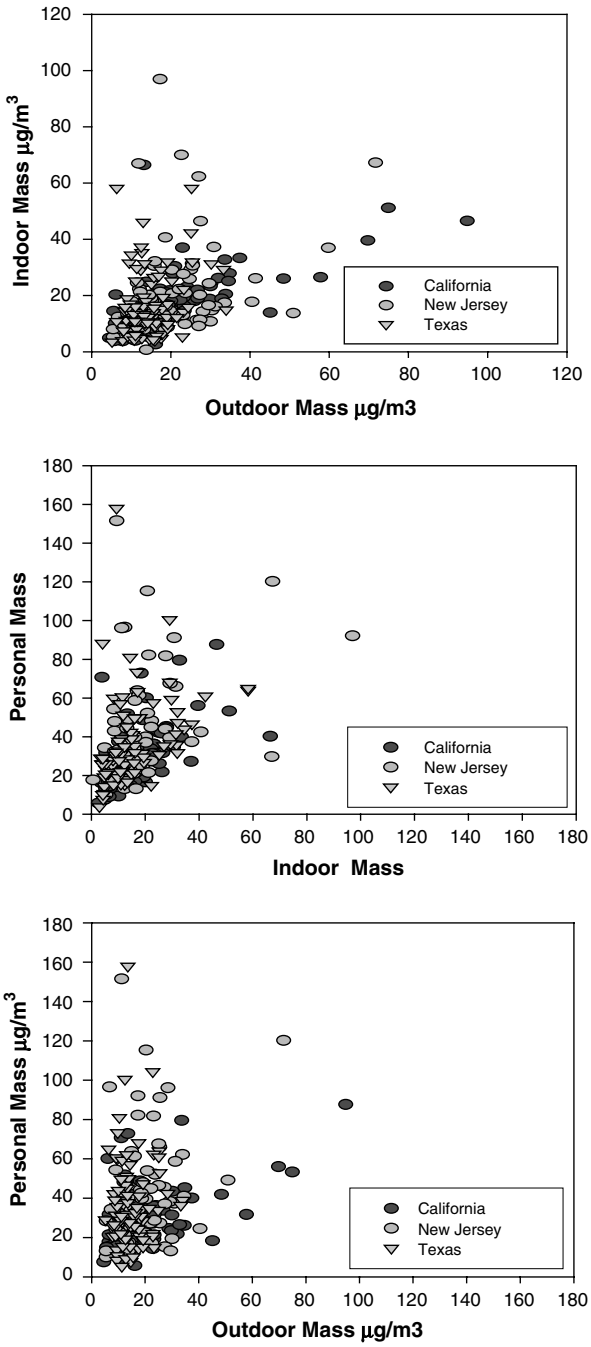

Figure 2. Indoor, outdoor and personal $PM_{2.5}$ mass concentrations.

high air exchange rates in these homes. Correlations of outdoor or indoor $PM_{2.5}$ concentrations with personal $PM_{2.5}$ concentrations are not much greater for these homes than for all homes.

The mean outdoor $PM_{2.5}$ concentration for the Los Angeles County, California samples (19.2 $\mu g/m^3$) is similar to that measured in the 1999 wintertime Fresno $PM_{2.5}$ exposure studies (20.5 $\mu g/m^3$; Vette et al., 2001). While the mean (19.2 $\mu g/m^3$) and median (16.1 $\mu g/m^3$) are much smaller than $PM_{2.5}$ mass concentrations in PTEAM (Riverside, CA, USA; 1990; mean = 48.9 $\mu g/m^3$ for daytime and 50.5 $\mu g/m^3$ for nighttime, median = 35.5 $\mu g/m^3$ for daytime and 35.0 $\mu g/m^3$ for nighttime). Also the outdoor mass concentrations for RIOPA California samples are less variable than PTEAM samples (σ = 13.3 $\mu g/m^3$ or 69% in RIOPA; σ = 37.6 $\mu g/m^3$ or 77% for the daytime and 40.3 $\mu g/m^3$ or 80% for the nighttime in PTEAM; Clayton et al., 1993). The differences between RIOPA and PTEAM are likely to result from differences in sampling strategies, study locations and study years. Riverside is at the eastern edge of the Los Angeles Basin, a receptor of aged pollution transported across the basin. In contrast, RIOPA homes are located in the western half of the air basin, closer to primary sources. Air quality in the Los Angeles Basin has also improved over the last 10 years, although declines in PM concentrations are more modest than declines in ozone concentrations. The annual average central monitor $PM_{2.5}$ mass concentration was 16.4 $\mu g/m^3$ in Elizabeth, New Jersey for the period July 1997 to June 1998 (Chuersuwan et al., 2000), which is close to the RIOPA outdoor residential median concentration in Elizabeth of 18.2 $\mu g/m^3$, and somewhat lower than the mean of 20.4 $\mu g/m^3$.

Comparisons can also be drawn with studies conducted in other locations. Lachenmyer and Hidy (2000) reported 48-h average $PM_{2.5}$ mass concentrations in Birmingham, AL of 12.2 $\mu g/m^3$ for outdoors and 11.2 $\mu g/m^3$ for indoors in the winter 1998, and 26.5 $\mu g/m^3$ for outdoors and 16.1 $\mu g/m^3$ for indoors in the summer 1997. Median indoor, outdoor and personal concentrations in the Toronto exposure study are 15.4, 13.2 and 18.7 $\mu g/m^3$. Median indoor, outdoor and personal concentrations in the EXPOLIS study (Helsinke, Finland; 1996–1998; Koistinen et al., 2001) are 11.7 $\mu g/m^3$ for indoor, 7.3 $\mu g/m^3$ for outdoor and 21.6 $\mu g/m^3$ for personal. It should be noted that EXPOLIS included smokers, while RIOPA did not.

Air exchange rates for California RIOPA homes (with a mean, median and standard deviation of 1.22, 0.93, 0.87 h^{-1}) are similar to those measured in a 1984–1985 survey in Los Angeles, which reported mean, median and standard deviations of 1.51, 1.07 and 1.47 h^{-1}, respectively (Wilson et al., 1996). In a wintertime 1991 ~ 1992 air exchange rate study in the Los Angeles Area, mean, median and standard deviations of air exchange rates were 0.79, 0.64 and 0.57 h^{-1}, respectively, whereas the wintertime mean, median and standard deviations of CA air exchange rates for RIOPA samples are 0.83, 0.76, 0.47 h^{-1}, respectively. Pandian et al. (1998) summarized nationwide residential air exchange rates based on 4590 measurements from different studies. New Jersey and Texas are included in northeast and southeast regions, respectively. Mean, median and standard deviations were 0.55, 0.42 and 0.47 h^{-1}, respectively, for the northeast region, and 0.71, 0.62 and 0.56 h^{-1} for the southeast region (after removal of two outliers). RIOPA air exchange rates in

Table 2. Coefficients of determination (R^2) for $PM_{2.5}$ concentrations.

State	Group	Indoor vs. outdoor	Personal vs. indoor	Personal vs. outdoor
Overall study	All homes	0.18	0.20	0.05
	I/O <1 homes	0.71	0.15	0.10
California	All homes	0.44	0.27	0.21
	I/O <1 homes	0.80	0.40	0.33
New Jersey	All homes	0.12	0.19	0.05
	I/O <1 homes	0.66	0.16	0.09
Texas	All homes	0.06	0.13	0.007
	I/O <1 homes	0.43	0.03	0.02

I/O indicates R^2 for homes where indoor/outdoor $PM_{2.5}$ ratio is less than 1.

Texas (mean, median and standard deviation of 0.71, 0.46, and 0.73 h^{-1}, respectively) are similar to the southeast region survey data, but RIOPA air exchange rates in New Jersey (mean, median and standard deviations of 1.22, 0.88, and 0.97 h^{-1}) are considerably higher than the northeast region survey data. One possible reason for this difference is that the maximum measurable air exchange rate for RIOPA (five changes per hour) is considerably greater than for previous studies. The highest air exchange rates reported by Pandian are approximately two changes per hour. Also, air exchange rates in New Jersey are expected to vary considerably from area to area since some areas, like Elizabeth, have primarily older homes (built 1910–1940) and other areas have primarily homes built after 1940 or after 1970. (Only valid air exchange rates for homes with PM sampling are included in this paper.)

Ambient and Nonambient Contributions to Indoor and Personal $PM_{2.5}$

Assuming perfect instantaneous mixing and assuming that factors affecting indoor concentrations are constant or change slowly throughout the monitoring period, the indoor $PM_{2.5}$ mass concentration can be described with a single compartment mass balance model:

$$V(\mathrm{d}C_\mathrm{i}/\mathrm{d}t) = PVaC_\mathrm{a} - VaC_\mathrm{i} + Q_\mathrm{i} - kC_\mathrm{i}V \qquad (4)$$

At steady state $\mathrm{d}C_\mathrm{i}/\mathrm{d}t = 0$, and this equation becomes:

$$C_\mathrm{i} = \frac{PaC_\mathrm{a}}{a+k} + \frac{Q_\mathrm{i}/V}{a+k} = F_\mathrm{INF}C_\mathrm{a} + C_\mathrm{pig} = C_\mathrm{ai} + C_\mathrm{pig} \qquad (5)$$

where C_i and C_a are the measured indoor and outdoor concentrations of $PM_{2.5}$ ($\mu g/m^3$), P is the penetration coefficient (dimensionless), a is the air exchange rate (h^{-1}), k is the particle loss coefficient (h^{-1}), Q_i is the strength of indoor sources ($\mu g/h$), and V is the house volume (m^3). The first term describes the contribution of outdoor-generated $PM_{2.5}$ to the indoor $PM_{2.5}$ concentration (ambient contribution, C_ai, $\mu g/m^3$), and the second term is the contribution of indoor sources to the indoor concentration (nonambient contribution, C_pig, $\mu g/m^3$). The quantity $Pa/(a+k)$ is called the infiltration factor (F_INF) and describes the fraction of C_a that is found indoors.

The random component superposition statistical (RCS) model proposed by Ott et al. (2000) derives a constant F_INF across all homes from the linear regression of measured values of C_i on C_a. The product of the constant F_INF and C_a from each home provides an estimate of the mean and distribution of the ambient contributions (C_ai) for the population of study homes. The mean and distribution of indoor contributions (C_pig) are given by the difference C_i–C_ai on a home-by-home basis. This model is not used to estimate ambient and nonambient contributions for individual homes, in part because the use of a single infiltration factor does not account for the large home-to-home variations in actual air exchange rates. This model assumes a linear superposition of the ambient and nonambient components of exposure and lack of correlation between these two components.

The mass balance approach can also be used to estimate personal exposure to PM of ambient and nonambient origin:

$$\begin{aligned} E_\mathrm{t} &= \{y + (1-y)[Pa/(a+k)]\}C_\mathrm{a} + E_\mathrm{nonag} \\ &= \alpha C_\mathrm{a} + E_\mathrm{nonag} = E_\mathrm{ag} + E_\mathrm{nonag} \end{aligned} \qquad (6)$$

where E_t is the time-averaged personal $PM_{2.5}$ exposure ($\mu g/m^3$) and y is the fraction of time a person spent outdoors (dimensionless). The first term describes the time-averaged personal exposure to $PM_{2.5}$ of ambient origin (E_ag, $\mu g/m^3$) and the second is the time-averaged personal exposure to $PM_{2.5}$ of nonambient origin (E_nonag, $\mu g/m^3$). The fraction of C_a that contributes to exposure is called the attenuation factor (α). In this case the RCS model derives a constant α across all homes from the linear regression of measured values of E_t on C_a. The RCS-predicted distribution of E_ag across the population of study homes is given by the product of the constant α and C_a from each home.

In this work, the mass balance model and RCS statistical model were used to estimate the ambient and nonambient contributions to indoor $PM_{2.5}$ concentrations and personal $PM_{2.5}$ exposures. Both models use measured $PM_{2.5}$ concentrations. The primary difference is that the mass balance model takes into consideration the measured air exchange rate, which varies considerably from home to home. Mass balance model results were obtained by fitting measured

Table 3. Parameter estimation by NLIN regression.

State	N	Boundary condition[a]	P	95% CI of P	k (h^{-1})	95% CI of k (h^{-1})
Overall	268	Y	0.91	(0.71, 1.12)	0.79	(0.18, 1.41)
		N	0.91	(0.71, 1.12)	0.79	(0.18, 1.41)
California	112	Y	1.00	(1.00, 1.00)	0.90	(0.53, 1.28)
		N	1.04	(0.75, 1.33)	0.98	(0.28, 1.69)
New Jersey	80	Y	0.73	(0.42, 1.05)	0.46	(−0.44, 1.36)
		N	0.73	(0.42, 1.05)	0.46	(−0.44, 1.36)
Texas	76	Y	1.00	(1.00, 1.00)	0.99	(−1.38, 3.35)
		N	1.35	(0.46, 2.23)	1.18	(−1.57, 3.92)

[a]means parameters are estimated with boundary condition $P \in [0,1]$; N means no boundary conditions for parameter estimation.

$PM_{2.5}$ concentrations and air exchange rates to the mass balance equation using nonlinear regression (NLIN in SAS). This results in the estimation of a single particle penetration coefficient (P) and loss rate coefficient (k) for the data set. Ambient and nonambient contributions to indoor $PM_{2.5}$ concentrations and personal exposures were then calculated for each home or participant using these population estimates of P and k and the measured concentrations and air exchange rates for that residence.

Table 3 shows mass balance model estimates of P and k with no constraints (N) and with the constraint $P \in [0,1]$, forcing a physically meaningful value of P (Y). Based on its physical meaning, P should be less than unity. Population-average $P = 0.91$ and $k = 0.79$ were estimated for the overall data set (all 268 homes with available a, C_i and C_a). P and k were also estimated for California, New Jersey and Texas homes separately. P and k values estimated in RIOPA study are in reasonable agreement with other $PM_{2.5}$ studies, which have estimated statistically and through experimentation P values of 0.84 to 1.0 for fine particles (Koutrakis et al., 1992; Thatcher and Layton, 1995; Ozkaynak et al., 1996; Lachenmyer and Hidy, 2000; Winkle and Scheff, 2001). P depends on the particle size distribution and house structure, so it is likely to vary from home to home, with season, and with geographic location (Long et al., 2001; Mosley et al., 2001; Riley et al., 2002).

The estimated particle loss rate for $PM_{2.5}$ for the RIOPA homes is $0.79\,h^{-1}$ (95% CI = 0.18, $1.4\,h^{-1}$). Ozkaynak et al. (1996) estimated a particle loss rate of $0.39\,h^{-1}$ (95% CI = 0.22, $0.55\,h^{-1}$) for the PTEAM study. Lachenmyer and Hidy (2000) estimated a loss rate of $0.6\,h^{-1}$ with a range of $2.0\,h^{-1}$. Abt et al. (2000b) and Vette et al. (2001) estimated k-values as a function of particle size using real-time particle monitors. In Fresno, CA, k was estimated to be $0.5\,h^{-1}$ for 0.1 μm diameter particles and $3.5\,h^{-1}$ for 2.5 μm diameter particles; in Boston, MA the lowest k was $0.7\,h^{-1}$ for 0.4–0.5 μm diameter particles, and the highest was $1.2\,h^{-1}$ for 2–3 μm diameter particles.

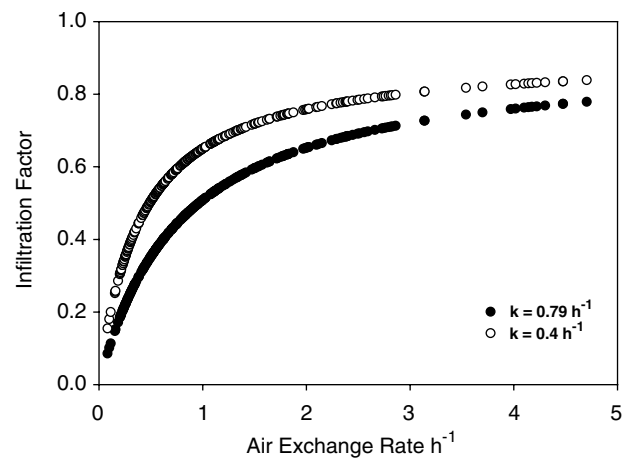

Figure 3. Infiltration factor as a function of air exchange rate at $k = 0.79\,h^{-1}$ (the RIOPA estimate) and $0.4\,h^{-1}$ (half the estimated value).

The indoor particle loss rate coefficient, k, is determined by many factors including surface-to-volume ratio, housing structure, near-surface air flows, turbulence and particle size distribution. The use of air conditioning has been reported to increase particle decay rates. The k-value obtained from the nonlinear regression procedure is an "average" value for the RIOPA homes. Given that the mass balance results account for home-to-home differences air exchange rate, the use of a single value of k for all homes probably introduces the largest uncertainties in the mass balance results. The effects of air exchange rate (a) and decay rate (k) on the infiltration coefficient (F_{INF}) for all RIOPA data are illustrated in Figure 3. The two curves in Figure 3 show the infiltration coefficient as a function of a assuming $k = 0.79\,h^{-1}$ (the RIOPA estimate) and $0.4\,h^{-1}$ (half the estimated value). When the air exchange rate is very small, k is a very important determinant of the infiltration of ambient $PM_{2.5}$. At $1.0\,h^{-1}$, a 50% reduction in k increases the infiltration of ambient $PM_{2.5}$ (F_{INF}) from roughly 55% to nearly 70%.

Table 4. Nonambient contribution to indoor concentrations and personal exposures (Results from mass balance model).

Home category	Source category	Mean contribution ($\mu g/m^3$)	Percent contribution%
Overall study	Nonambient to indoor ($N=268$)	9.0	39.8
	Nonambient to personal exposure ($N=197$)	28.3	73.9
California homes	Nonambient to indoor ($N=112$)	6.0	33.4
	Nonambient to personal exposure ($N=85$)	21.0	66.8
New Jersey homes	Nonambient to indoor ($N=80$)	10.6	29.7
	Nonambient to personal exposure ($N=53$)	38.7	77.5
Texas homes	Nonambient to indoor ($N=76$)	11.7	59.0
	Nonambient to personal exposure ($N=59$)	28.7	78.5

Table 4 provides the mean nonambient contributions to indoor and personal $PM_{2.5}$ concentrations on a concentration and percentage basis, estimated using the mass balance model. The mean contribution of ambient outdoor sources to indoor $PM_{2.5}$ concentrations is 8.7 $\mu g/m^3$ or 60% for all study homes (67%, 70% and 41% for California, New Jersey and Texas homes, respectively). The median contribution of ambient sources to indoor $PM_{2.5}$ concentrations using the mass balance approach is estimated to be 7.2 $\mu g/m^3$ or 56% for all study homes (63%, 52% and 33% for California, New Jersey and Texas study homes, respectively).

Figure 4 shows the cumulative distributions of ambient and nonambient contributions to indoor $PM_{2.5}$ concentrations calculated by mass balance and RCS approaches for the same 268 homes. The RCS model calculated an infiltration factor (slope of the regression of C_i on C_a) of 0.46, and a mean and median ambient contribution of 8.5 and 7.2 $\mu g/m^3$, respectively. The values of "$Pa/(a+k)$" calculated from the mass balance model are approximately normally distributed (by Kolmogorov-Smirnov test with $\alpha=0.05$ and $P>0.15$) with a mean of 0.46 and a standard deviation of 0.16. Good agreement is seen between these two approaches, particularly for distribution means, which are within 1 $\mu g/m^3$ for both ambient and nonambient contributions to indoor $PM_{2.5}$ concentrations. The breadth of the distribution of ambient contributions is somewhat larger in the mass balance solution because this approach takes into consideration home-by-home variations in air exchange rate. The standard deviation of ambient contributions is 5.2 $\mu g/m^3$ for RCS and 6.4 $\mu g/m^3$ for mass balance distribution estimates.

While the RCS model is not designed to predict the ambient contribution for *individual* homes, we found that the coefficient of variance for the ambient contribution to indoors for the two models was 26% when results were compared on a home-by-home basis (268 homes). The coefficient of variance for the nonambient contribution to indoors is 24%. Results are well correlated, with a coefficient of determination (R^2) greater than 75%, for both ambient and nonambient contributions. This method comparison illustrates the degree of uncertainty introduced (26%) when estimates of ambient contributions to indoor $PM_{2.5}$ concentrations are made without measured air exchange rate, which is perhaps the most variable parameter.

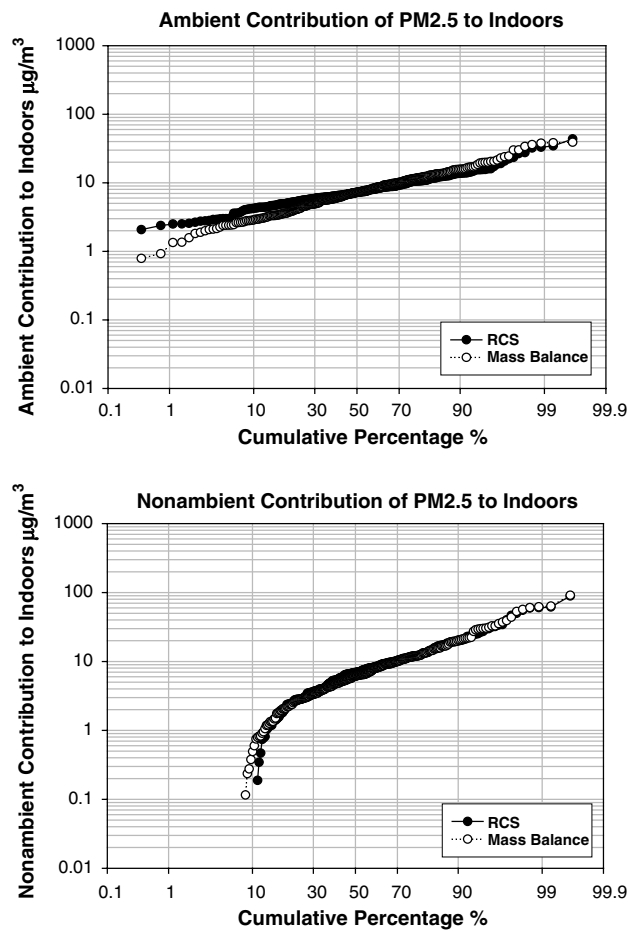

Figure 4. Ambient and nonambient contributions to indoor $PM_{2.5}$ concentration estimated by RCS and mass balance models.

The mean ambient (outdoor) contribution to personal $PM_{2.5}$ exposure, estimated using the mass balance approach and activity diary data, is 26% for all study homes, (33%, 22% and 21% for California, New Jersey and Texas homes, respectively). Means obtained by RCS were comparable. It must be noted that harmonizing PEM and Harvard measurements using Equations (1), (2) or (3) would increase

the percentage ambient contribution to personal $PM_{2.5}$ exposure by 0.1–4.0 to 26–30% for all study homes.

The RCS model calculates ambient contributions to indoor concentrations and personal exposures based on the statistical inferences of regression analysis. However, indoor–outdoor or personal–outdoor regressions could be affected by extreme values (outliers), such as a high nonambient exposure on a day with low ambient concentration or vice versa. For this reason outliers were identified and their influence on the infiltration factor or attenuation factor in the RCS model was evaluated. A value was considered as outlier if the absolute studentized residual of that data point was larger than three. In the evaluation of the ambient $PM_{2.5}$ contribution to indoor concentrations, seven outliers were identified. After removal of those outliers, the infiltration factor (F_{INF}) changed by only 0.01. Four outliers were found in the outdoor-personal regression. Elimination of these outliers changed the attenuation factor by 0.05. Elimination of outliers increased the RCS-estimated mean ambient contribution to indoor by 0.1 $\mu g/m^3$ and RCS-estimated mean ambient contribution to personal by 0.9 $\mu g/m^3$.

The sensitivity of ambient contribution estimates obtained by mass balance to the choice of P and k is shown in Figure 5. Figure 5 gives the mean and median percentage ambient contributions to the indoor $PM_{2.5}$ concentration in California, New Jersey, Texas and the overall study using seven reasonable choices of P and k. In scenario "A", P (1.0) and k (0.39 h^{-1}) are taken from the PTEAM study. In scenario

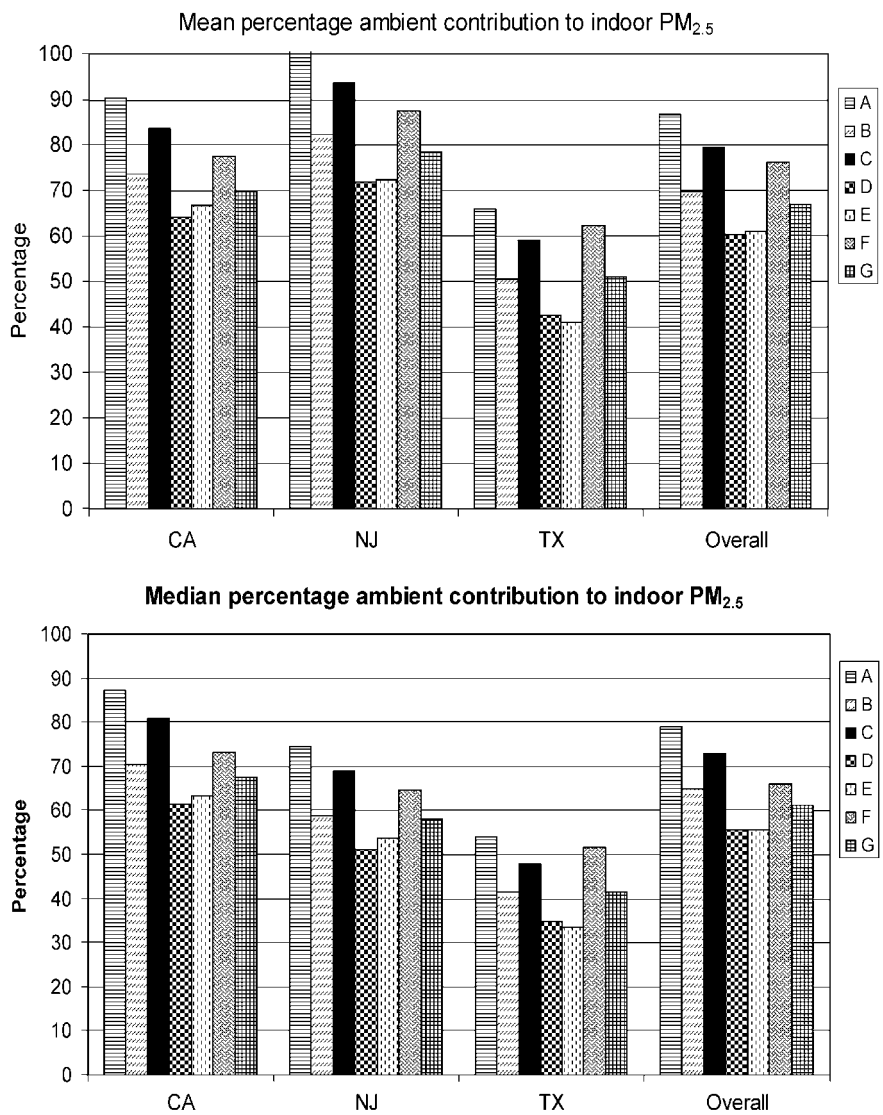

Figure 5. Sensitivity of the estimated ambient contribution to indoor $PM_{2.5}$ mass concentration to the choice of P and k. A–C use literature values from other locations, D–E are RIOPA estimates from the overall data set (D) and the dataset segregated by city (E). F and G use estimates of P and k taken from a subset of RIOPA homes expected to have small contributions from indoor sources.

"B", P (0.95) and k (0.62 h^{-1}) are the average of winter and summer values from Lachenmyer and Hidy (2000). In scenario "C", P (1.0) and k (0.5 h^{-1}) are composites from other studies (Thatcher and Layton, 1995; Abt et al., 2000b; Long et al., 2001; Thornburg et al., 2001; Vette et al., 2001). The RIOPA overall P (0.91) and k (0.79 h^{-1}) are used in scenario "D". In scenario "E", state-specified P and k from Table 3 (with boundary conditions) are used. In scenarios "F" and "G", P and k were obtained by non-linear regression of the subset of RIOPA data with few indoor sources. In "F" only homes where $C_i < C_a$ ($N = 165$ homes) were used to estimate P (0.73) and k (0.19 h^{-1}). The questionnaire data were used to identify 23 homes that had no evidence of indoor sources. This subset of data provided the P (0.78) and k (0.40 h^{-1}) estimates for scenario "G". Using literature values of P and k is likely to yield less accurate results than estimates based on RIOPA data (D and E) because P and k will vary with housing stock, climate and particle source mix. Estimate G suffers from small N. The comparison of Figure 5, which uses a variety of reasonable values for P and k, suggests that uncertainties in P and k lead to uncertainties on the order of 20% in the mean ambient contribution to indoor $PM_{2.5}$.

Conclusions and perspective

This study presents indoor, outdoor and personal $PM_{2.5}$ mass concentration results from the RIOPA study. While the poor correlations between personal, indoor and residential outdoor concentrations suggest that ambient outdoor $PM_{2.5}$ is a poor surrogate for personal exposure to total $PM_{2.5}$, it might still be a good surrogate for community exposure to "$PM_{2.5}$ of ambient origin." This quantity (i.e., the ambient contribution) varies considerably from home to home and person to person. The ambient contribution to personal $PM_{2.5}$ for RIOPA subjects is estimated to be 26% on average. The mean ambient contribution to the indoor $PM_{2.5}$ concentration is 60% when estimated using the actual home-by-home air exchange rate and a population-average P and k derived from RIOPA data. The mean obtained by assuming a population-average infiltration factor (RCS model) was similar, with a somewhat narrower distribution. The error (random) in the ambient contribution introduced by neglecting the home-by-home variation in air exchange rate (RCS model) was 26%.

Mean and median ambient contributions to indoor and personal concentrations appear to be similar for California and New Jersey homes but much lower for Texas homes. This finding is consistent with our expectations, given the prevalence of air conditioning use in Texas (more than 23% of Texas study homes used A/C during sampling) and almost lack of air conditioning in California and New Jersey study homes (less than 3% and about 18% in California and New Jersey, respectively). Air-conditioned homes tend to be tightly sealed and therefore have reduced air exchange rates. As shown in Equation (5) and Figure 3, a decrease in the air exchange rate results in a decreased ambient contribution (i.e., a decrease in the first term and increase in the second term of Equation (5).

Acknowledgements

This research was supported by the Health Effects Institute, the Mickey Leland National Urban Air Toxics Center, the NIEHS Center of Excellence, the NJ Agricultural Experiment Station, and an EPA STAR Graduate Fellowship (for D. Shendell). We gratefully acknowledge the hard work of all the students and technicians in the field and laboratories of RIOPA investigators and the hospitality of the RIOPA participants. In addition, we thank Dr. Wayne Ott of Stanford University for his advice, and acknowledge the guidance of Dr. Glen Cass, who will be missed.

Research described in this article was conducted, in part, under contract to the Health Effects Institute (HEI), an organization jointly funded by the United States Environmental Protection Agency (EPA: Assistance Agreement R828112) and automotive manufacturers. The contents of this article do not necessarily reflect the views of HEI, nor do they necessarily reflect the views and policies of EPA or of motor vehicle and engine manufacturers.

References

Abt E., Suh H.H., Allen G., and Koutrakis P. Characterization of indoor particle sources: a study conducted in the Metropolitan Boston area. *Environ Health Perspect* 2000a: 108: 35–44.

Abt E., Suh H.H., Catalano P., and Koutrakis P. Relative contribution of outdoor and indoor particle sources to indoor concentrations. *Environ Sci Technol* 2000b: 34: 3579–3587.

Ansari A.S., and Pandis S.N. Response of inorganic PM to precursor concentrations. *Environ Sci Technol* 1998: 32: 2706–2714.

Chao C.Y.H., Tung T.C.W., and Burnett J. Influence of different indoor activities on the indoor particulate levels in residential buildings. *Indoor Built Environ* 1997: 7: 110–121.

Chuersuwan N., Turpin B.J., and Pietarinen C. Evaluation of time-resolved $PM_{2.5}$ data in urban/suburban areas of New Jersey. *J Air Waste Manag Assoc* 2000: 50: 1780–1789.

Clayton C.A., Perritt R.L., Pellizzari E.D., Thomas K.W., and Whitmore R.W. Particle total exposure assessment methodology (PTEAM) study: distributions of aerosol and elemental concentrations in personal, indoor, and outdoor air samples in southern California community. *J Expos Anal Environ Epidemiol* 1993: 3: 227–250.

Dietz R.N., Goodrich R.W., Cote E.A., and Wieser R.F. Detailed description and performance of a passive perfluorocarbon transfer system for building ventilation and air exchange measurements. In: Trechel H.R., and Laqus (eds.). *Measured Air Leakage of Buildings*. ASTM STP 904, American Society of Testing and Materials, Philadelphia, PA, 1986: pp. 203–264.

Goldberg M.S., Burnett R.T., Bailar J.C., Brook J., Bonvalot Y., Tamblyn R., Singh R., and Valois M.F. The association between daily mortality and ambient air particle pollution in montreal, Quebec. *Environ Res Sect A* 2001: 86: 12–25.

Jones N.C., Thornton C.A., Mark D., and Harrison R.M. Indoor/outdoor relationships of particulate matter in domestic homes with roadside, urban and rural locations. *Atmos Environ* 2000: 34: 2603–2612.

Koistinen K.J., Hanniene O., Rotko T., Edwards R.D., Moschandreas D., and Jantunen M.J. Behavioral and environmental determinants of personal exposures to $PM_{2.5}$ in EXPOLIS—Helsinki, Finland. *Atmos Environ* 2001: 35: 2473–2481.

Koutrakis P., Briggs S.L.K., and Leaderer B.P. Source apportionment of indoor aerosols in Suffolk and Onondaga counties New York. *Environ Sci Technol* 1992: 26: 521–527.

Lachenmyer C., and Hidy G.M. Urban measurements of outdoor–indoor $PM_{2.5}$ concentrations and personal exposure in the deep south Part I. Pilot study of mass concentrations for nonsmoking subjects. *Aerosol Sci Technol* 2000: 32: 34–51.

Lefcoe N.M., and Inculet I.I. Particulates in domestic premises II. Ambient levels and indoor, outdoor relationships. *Arch Environ Health* 1975: 30: 565–570.

Long C.M., Suh H.H., Catalano P.J., and Koutrakis P. Using time- and size-resolved particulate data to quantify indoor penetration and deposition behavior. *Environ Sci Technol* 2001: 35: 2089–2099.

Morandi M.T., Stock T.H., and Contant C.F. A comparative study of respirable particulate microenvironmental concentrations and personal exposures. *Environ Monit and Assess* 1988: 10: 105–122.

Mosley R.B., Greenwell D.J., Sparks L.E., Guo Z., Tucher W.G., Fortmann R., and Whitfield C. Penetration of ambient fine particles into the indoor environment. *Aerosol Sci Technol* 2001: 34: 127–136.

Naumova Y.Y., Totten L.A., Eisenreich S.J., Turpin B.J., Colome S.D., Morandi M.T., Weisel C.P., Stock T.H., Winer A.M., Alimokhtari S., Kwon J., Maberti S., Shendell D., Jones J., and Wall S.J. Polycyclic aromatic hydrocarbons in the indoor and outdoor air of three cities in the US.: results from the RIOPA study. *Environ Sci Technol* 2002: 36: 2552–2559.

Naumova Y.Y., Offenberg J.H., Eisenreich S.J., Polidori A., Meng Q.Y., Turpin B.J., Colome S.D., Morandi M.T., Weisel C.P., Stock T.H., Winer A.M., Alimokhtari S., Kwon J., Maberti S., Shendell D., and Jones J. An assessment of gas-particle distribution of polycyclic aromatic hydrocarbons in coupled outdoor/indoor atmospheres. *Atmos Environ* 2003: 37: 703–719.

Norris G., YoungPong S.N., Koenig J.Q., Larson T.V., Sheppard L., and Stout J.W. An association between fine particles and asthma emergency department visits for children in seattle. *Environ Health Perspectives* 1999: 107: 489–493.

Oglesby L., Kunzli N., Roosli M., Braun-Fahrlander C., Mathys P., Stern W., Jantunen M., and Kousa A. Validity of ambient levels of fine particles as surrogate for personal exposure to outdoor Air pollution—results of the European EXPOLIS-EAS Study (Swiss Center Basel). *J Air Waste Manag Assoc* 2000: 50: 1251–1261.

Ott W., Wallace L., and Mage D. Predicting particulate (PM10) personal exposure distributions using a random component superposition statistical model. *J Air Waste Manag Associ* 2000: 50: 1390–1406.

Ozkaynak H., Xue J., Spenglr J., Wallace L., Pellizzari E., and Jenkins P. Personal exposure to airborne particles and metals: results from the particle TEAM Study in Riverside, California. *J Expos Anal Environ Epidemiol* 1996: 6: 57–78.

Pandian M.D., Behar J.V., Ott W.R., Wallace L.A., Wilson A.L., Colome S.D., and Koontz M. Correcting errors in the nationwide data base of residential air exchange rates. *J Expo Anal Environ Epidemiol* 1998: 8: 577–586.

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

Riley W.J., Mckone T.E., Lai A.C.K., and Nazaroff W.W. Indoor particulate matter of outdoor origin: importance of size-dependent removal mechanisms. *Environ Sci Technol* 2002: 36: 200–207.

Robinson J., and Nelson W.C. *National Human Activity Pattern Survey Data Base*. USEPA, Research Triangle Park, NC, 1995.

Schwartz J., Norris G., Larson T., Sheppard L., Claiborne C., and Koenig J. Episodes of high coarse particle concentrations are not associated with increase mortality. *Environ Health Perspectives* 1999: 107: 339–342.

Seinfeld J.H., and Pandis S.N. *Atmospheric Chemistry and Physics*. John Wiley & Son, Inc., New York, 1998.

Sexton K., Spengler J.D., and Treitman R.D. Personal exposure to respirable particles: a case study in Waterbury, Vermont. *Atmos Environ* 1984: 18: 1385–1398.

Spengler J.D., Treitman R.D., Tostenson T.D., Mage D.T., and Soczek M.L. Personal exposures to respirable particulates and implications for air pollution epidemiology. *Environ Sci Technol* 1985: 19: 700–707.

Thatcher T.L., and Layton D.W. Deposition, resuspension, and penetration of particles within a residence. *Atmos Environ* 1995: 29: 1487–1497.

Thornburg J., Ensor D.S., Rodes C.E., Lawless P.A., Sparks L.E., and Mosley R.B. Penetration of particles into buildings and associated physical factors. Part I: Model development and computer simulations. *Aerosol Sci Technol* 2001: 34: 284–296.

Turpin B.J., Saxena P., and Andrews E. Measuring and simulating particulate organics in the atmosphere: Problems and prospects. *Atmos Environ* 2000: 34: 2983–3013.

USEPA. *Air Quality Criteria for Particulate Matter*. USEPA: Research Triangle Park, NC, 1996.

Vette A.F., Rea A.W., Lawless P.A., Rodes C.E., Evans G., Highsmith V.R., and Sheldon L. Characterization of indoor–outdoor aerosol concentration relationships during the fresno PM exposure studies. *Aerosol Sci Technol* 2001: 34: 118–126.

Wainman T., Zhang J.F., Weschler C.J., and Lioy P.J. Ozone and limonene in indoor air: a source of submicron particle exposure. *Environ Health Perspect* 2000: 108: 1139–1145.

Wallace L. Indoor particles: a review. *J Air Waste Manag Assoc* 1996: 46: 98–126.

Weisel C.P., Zhang J., Turpin B.J., Morandi M.T., Colome S., Stock T.H., Spektor D.M., Korn L., Winer A., Alimokhtari S., Kwon J., Mohan K., Harrington R., Maberti S., Shendell D., Giovanetti R., and Lee J.H. The Relationships of Indoor, Outdoor and Personal Air (RIOPA) Study: study design, methods and initial results. *J Expos Anal Environ Epidemiol* 2004, June 23 (epub ahead of print).

Weschler C.J., and Shields H.C. Potential reactions among indoor pollutants. *Atmos Environ* 1997: 31: 3487–3495.

Wilson A.L., Colome S.D., Tian Y., Becker E.W., Baker P.E., Behrens D.W., Billick I.H., and Garrison C.A. California residential air exchange rates and residence volumes. *J Expos Anal Environ Epidemiol* 1996: 6: 311–326.

Winkle M.R.V., and Scheff P.A. Volatile organic compounds, polycyclic aromatic hydrocarbons and elements in the air of ten urban homes. *Indoor Air* 2001: 11: 49–64.

Yakovleva E., Hopke P.K., and Wallace L. Receptor modeling assessment of particle total exposure assessment methodology data. *Environ Sci Technol* 1999: 33: 3645–3652.

Yocom J.E. Indoor–outdoor air quality relationships, a critical review. *J Air Pollu Control Assoc* 1982: 32: 500–520.