

## Particle Concentrations in Inner-City Homes of Children with Asthma: The Effect of Smoking, Cooking, and Outdoor Pollution

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Inner-city children have high rates of asthma. Exposures to particles, including allergens, may cause or exacerbate asthma symptoms. As part of an epidemiologic study of inner-city children with asthma, continuous (10-min average) measurements of particle concentrations were made for 2-week periods in 294 homes drawn from seven cities. Measurements were made using an optical scattering device that is most sensitive to fine particles. The concentrations recorded by these devices were corrected to agree with colocated outdoor gravimetric PM<sub>2.5</sub> monitors. Indoor concentrations in the homes averaged 27.7 (standard deviation = 35.9) µg/m<sup>3</sup>, compared with concurrent outdoor concentrations of 13.6 (7.5) µg/m<sup>3</sup>. A multivariate model indicated that outdoor particles penetrated indoors with an efficiency of 0.48 and were therefore responsible for only 25% of the mean indoor concentration. The major indoor source was smoking, which elevated indoor concentrations by 37 µg/m<sup>3</sup> in the 101 homes with smokers. Other significant sources included frying, smoky cooking events, use of incense, and apartment housing, although the increases due to these events ranged only from 3 to 6 µg/m<sup>3</sup>. The 10-min averaging time allowed calculation of an average diurnal variation, showing large increases in the evening due to smoking and smaller increases at meal times due to cooking. Most of the observed variance in indoor concentrations was day to day, with roughly similar contributions to the variance from visit to visit and home to home within a city and only a small contribution made by variance among cities. The small variation among cities and the similarity across cities of the observed indoor air particle distributions suggest that sources of indoor concentrations do not vary considerably from one city to the next, and thus that simple models can predict indoor air concentrations in cities having only outdoor measurements. *Key words:* continuous monitors, environmental tobacco smoke, gravimetric measurements, indoor air, MIE pDR, optical scattering, PM<sub>10</sub>, PM<sub>2.5</sub>. *Environ Health Perspect* 111:1265–1272 (2003). doi:10.1289/ehp.6135 available via <http://dx.doi.org/> [Online 1 April 2003]

Particulate pollution in outdoor air has been associated with a number of adverse health effects [U.S. Environmental Protection Agency (U.S. EPA) 1996, 2002]. Time-series studies have revealed that short-term increases in the concentration of particles in outdoor air are associated with increased mortality in the overall population (Schwartz 1994), and limited data have also suggested adverse effects of outdoor particles on emergency room use (Norris et al. 1999; Schwartz et al. 1993), symptoms (Yu et al. 2000), and lung function (Peters et al. 1997a, 1997b) among persons with asthma. Fine particles, those with an aerodynamic diameter < 2.5 µm (PM<sub>2.5</sub>), may produce most of these harmful effects (Schwartz and Neas 2000), although coarse particles (those with diameters 2.5–10 µm) have also been implicated in some studies of childhood asthma (Lin et al. 2002; Sheppard et al. 1999). The concentrations of combined fine and coarse particles (PM<sub>10</sub>) in outdoor air, estimated on the basis of gravimetric measurements made at geographically dispersed monitoring sites, have been well described across the United States (U.S. EPA 2001), providing essential exposure data for research and regulatory purposes [Aerometric

Information Retrieval System (AIRS)]. More recently, limited data are becoming available from outdoor PM<sub>2.5</sub> monitoring stations in several cities.

Fewer data have been reported on the concentration of particles in the indoor air of the home, where the average American spends an estimated 70% of the time (Klepeis et al. 1995). Indoor concentrations of particles vary substantially among homes and over time in a given home as a result of variation in sources of combustion products, such as smoking (Dockery and Spengler 1981; Spengler et al. 1980), cooking (Özkaynak et al. 1996; Pellizzari et al. 1993), and heating (Sheldon et al. 1989), as well as variation in ventilation and air filtration (Sarnat et al. 2000; Wallace et al. 2002). The variation in these factors among different homes produces a low correlation between the indoor and outdoor levels of particles across homes (Dockery and Spengler 1981; Pellizzari et al. 1993; Spengler et al. 1980). A limitation of most previous studies of indoor particles is their reliance on 24- or 12-hr daily average levels that lack the temporal resolution needed to assess peak exposures that may be important determinants of adverse health effects.

Asthmatic children residing in the inner city are a subgroup of particular interest with regard to indoor particulate air pollution, but their exposure to particles in indoor air has not been well characterized. Hyperresponsive airways make these children especially susceptible to the adverse respiratory health effects of particles (Pope 2000). In addition, the high prevalence of parental smoking, poor ventilation, and other environmental factors found in many inner-city homes (Crain et al. 2002; Kattan et al. 1997) may expose asthmatic children in this setting to relatively high levels of particles. For these reasons, we investigated the concentration of particles in indoor air and other characteristics of the indoor home environments of 328 asthmatic children living in low-income census tracts of seven U.S. cities. We employed portable nephelometers providing continuous measurements and electronic data recording to optimize the temporal resolution of indoor particle levels in these homes.

### Study Design

The Inner-City Air Pollution (ICAP) study was an enhancement of the Inner-City Asthma Study (ICAS), sponsored by the National Institute of Allergy and Infectious Diseases and the National Institute of Environmental

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Investigators in the Inner-City Asthma Study are listed in the Appendix. We thank the participants for making their homes available to us for 2-week periods. We also thank our excellent staff for performing the many tasks involved in a multiyear, multicity field study.

This work was supported by grants AI-39769, AI-39900, AI-39902, AI-39789, AI-39901, AI-39761, AI-39785, and AI-39776 from the National Institute of Allergy and Infectious Diseases and the National Institute of Environmental Health Sciences, with partial support provided by the U.S. Environmental Protection Agency through an inter-agency agreement with the National Institute of Allergy and Infectious Diseases. The manuscript has been subjected to agency review and approved for publication. The opinions expressed here do not necessarily reflect U.S. EPA policy. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

The authors declare they have no conflict of interest. Received 28 November 2002; accepted 1 April 2003.

Health Sciences. ICAS is a seven-site cooperative study designed to evaluate the effectiveness of interventions to reduce asthma morbidity among inner-city children. The study is a randomized controlled trial of two interventions to reduce asthma morbidity among urban children with moderate to severe asthma: an environmental intervention and a physician feedback intervention. The physician feedback intervention consisted of a novel communication/physician education system that provides the children's primary care physicians with current information on the child's clinical status, medication use, and health care use. The environmental intervention included education and remediation of environmental triggers, including cockroaches, dust mites, environmental tobacco smoke, mold, furry pets, and rodents.

The research protocol was approved by the institutional review board (IRB) of each research site and the Data Coordinating Center (Rho, Inc., Chapel Hill, NC). Written informed consent was obtained from the children's parent or legal guardian before enrollment. On the basis of local IRB requirements, children at several research sites were also given assent forms explaining the study and acknowledging their willingness to participate.

The ICAP study focused on a subgroup of 328 of the 947 ICAS children and was conducted after the ICAS interventions were completed. The ICAS children were recruited from inner-city census tracts in seven metropolitan areas, including the Bronx and Manhattan, New York; Boston, Massachusetts; Chicago, Illinois; Dallas, Texas; Seattle, Washington; and Tucson, Arizona. Children were eligible for the study if they had asthma, were 5 through 11 years old, and resided in metropolitan census tracts where approximately 20–40% of the residents were below the federal poverty guidelines. Recruitment criteria required that the children have at least one hospitalization in the prior 6 months or two hospital emergency department visits for asthma, and sleep in the study residence at least 5 nights per week. After completing the ICAS intervention, the ICAP subset was recruited for the home environmental air quality assessment. Indoor  $PM_{2.5}$  was measured for up to three 2-week periods at 6-month intervals during the follow-up year of ICAS. On collecting the nephelometer after the 2-week measurement period, a questionnaire was given to each ICAP study participant to assess those home and behavioral characteristics that could potentially influence the indoor air quality.

A new questionnaire was developed for this study; however, many of the questions have been employed in previous studies of particles indoors, such as the Particle Total Exposure Assessment Methodology (PTEAM) Study (Özkaynak et al. 1996). One set of questions concerned likely sources of combustion-related particles in the home: smoking, cooking, use of

a wood stove or fireplace, use of candles or incense, and use of a gas or kerosene space heater. The smoking questions asked about the number of smokers in the home and also asked for an estimate of the number of cigarettes smoked. The cooking questions differentiated between frying/sautéing/grilling and other types of cooking (baking, broiling, oven or toaster oven use) based on previous studies showing the importance of the frying mode compared with other modes (Kelly 2001; Wallace 2000). We also asked whether a gas stove was ever used to heat the house and whether cooking had produced unusually smoky conditions in the past week. A second set of questions concerned possible modifiers of particle concentrations in the home: open windows, use of air conditioning, air cleaners, and exhaust fans. A third set of questions included possible sources of noncombustion particles: sweeping, dusting, vacuuming, and use of humidifiers (because ultrasonic humidifiers are powerful sources of particles; Highsmith et al. 1988). Most questions concerned the full 2-week period of monitoring, although questions concerning cooking covered only the final week because of concerns about memory accuracy.

**Measurement methods.** The MIE personal DataRAM (pDR) 1000 (Thermo Electron Corp., Franklin, MA) was employed to measure the concentration of indoor airborne particulate matter, providing direct and continuous readout as well as electronic recording of the information. The pDR samples the air passively; air freely accesses the sensing chamber by convection, diffusion, and advective air motion. The pDR is an integrating nephelometer (scattering coefficient range =  $1.5 \times 10^{-6}$  to  $0.6 \text{ m}^{-1}$  at 880 nm wavelength). It records in units of micrograms per cubic meter, as calibrated by the manufacturer using a fine International Organization for Standardization (ISO) test dust (specific gravity, 2.6; index of refraction = 1.5–0i). Readings can be converted to light scattering units using the equation  $\mu\text{g}/\text{m}^3 = 1.023 \times (\text{light scattering units in } 1 \times 10^{-3} \text{ m}^{-1})$  (MIE 1998). The pDRs were programmed to record 10-min averages. The pDR has its greatest sensitivity to particles  $0.6 \mu\text{m}$  in diameter, with the sensitivity falling off to about 16% at diameters of about  $5 \mu\text{m}$  and even lower at higher diameters. The sensitivity also falls off steeply at diameters lower than  $0.6 \mu\text{m}$ , falling below 10% at diameters below  $0.15 \mu\text{m}$ . Therefore, the pDR would not respond or respond only minimally to ultrafine ( $< 0.10 \mu\text{m}$ ) particles. However, ultrafine particles, despite their great number, have only minimal impact on mass. For this reason, the pDR is expected to have greater correlation with  $PM_{2.5}$  measurements than with  $PM_{10}$ .

Outdoor air data for  $PM_{2.5}$  were acquired from the AIRS database for the comparable

dates of ICAP study indoor measurements using data for all monitors for the counties in which the seven research sites were located. In counties with more than one active monitor, an algorithm described by Zanobetti et al. (2000) was implemented. For each monitor, daily concentrations were obtained, and monitor-specific means and standard deviations (SDs) were computed for the entire sample period. These means and SDs were used to assign a monitor-specific Z-score for each sampling day based on that day's deviation from the calculated mean. Next, the Z-scores for a day were averaged across the monitors within a given county. Finally, the estimate of the site-specific outdoor  $PM_{2.5}$  concentration for each day was calculated by multiplying this averaged Z-score by the overall SD for that county and adding the overall county mean. This method permits use of all the available data for a given day while preventing undue bias when readings for a typically low or high monitor may be missing.

**Field operations.** No more than 24 hr before a device was taken to a participant's home, it was adjusted to measure a zero value correctly at the study center in a hand-inflatable particle-free pouch (zero bag) provided with the pDR. The device was placed in the zero bag. A hand-pump/in-line high-efficiency particulate arresting (HEPA) filter unit, attached to a nipple on the bag, was used to inflate the bag. Air was slowly released from the bag through the zippered top to flush the zero bag of any excess dust. Then, while the device was zeroing itself with the bag closed, the technician continued to pump air slowly into the bag. The unit indicated whether or not the calibration was successful.

Once at the home, the device was placed in the participant's living area if possible. Alternative location choices included the child's bedroom or the dining room. The device was not placed in a room where a HEPA or other air filter unit was routinely operated unless there was no other suitable choice. It was placed at least 6–8 inches away from any wall, and not directly in front of a window. A single layer of clean mosquito netting was placed around the device and secured with a wire tie at the end opposite the air intake area. This was to ensure that no small insects such as cockroaches or spiders would crawl into the device. The pDR was laid flat inside a coarse mesh container (Nalgene Polypropylene Autoclaving Baskets, item 6917-0230; Nalge Nunc International, Rochester, NY) that allowed a free flow of air past the monitor.

The device remained in the home for a minimum of 14 days. As soon as feasible after this time, the technician returned to the home to disconnect the device and bring it back to the study center, where the accumulated data were downloaded. The device was checked for zero drift within 24 hr after completion of sampling.

Zero drift is the change (positive or negative) in the pDR zero value over time. First, the device was set to measure ambient conditions in the study center. Then the device was placed in the zero bag. A hand-pump/in-line HEPA filter unit, attached to a nipple on the bag, was used to inflate the bag. Air was slowly released from the bag through the zippered top to flush the zero bag of any excess dust and particles. The process was repeated twice. Then the bag was zipped closed and slowly filled with air for at least one additional minute while the concentration reading was watched. When it reached and maintained its lowest reading, this reading was recorded on the data collection forms.

**Data analysis methods.** We estimated the limit of detection (LOD) of the pDR by taking repeated measurements at low concentrations ( $\leq 5 \mu\text{g}/\text{m}^3$ ). Precision of the pDRs was determined by running between two and eight colocated monitors for a number of days at each site. All 10-min averages were compared, and also all 1-hr averages, because the 10-min averages might be affected by nonsynchronous clock times on the monitors. The relationship of the pDR to gravimetric monitors was determined by colocating the pDR with gravimetric samplers employing the Federal Reference Method (FRM 1997) for  $\text{PM}_{2.5}$  at two official U.S. EPA monitoring sites for 12–16 consecutive days. The pDR 10-min averages were combined into 24-hr averages to compare with the FRM results. The average ratio of the two methods was used to correct the pDR readings for the difference in density between the mineral dust used in the manufacturer's calibration and the less dense indoor and outdoor particles. It would have been desirable to calibrate the pDR against the FRM using indoor aerosols, because the indoor aerosol may differ in some respects from the outdoor aerosol; however, the FRM is a noisy, bulky, high-volume method that is unsuitable for monitoring indoors.

The data were analyzed using SAS (version 8.2; SAS Institute, Inc., Cary, NC). Univariate statistics, Spearman correlations, and regressions were performed on the indoor and outdoor measurements using standard SAS procedures. Bivariate comparisons of smoking versus nonsmoking homes, nonsmoking homes with cooking versus nonsmoking homes without cooking, and other variables were performed. A multivariate model relating indoor concentrations to outdoor concentrations and certain home and occupant characteristics was developed using mixed-model techniques. Other independent variables considered in the model included any smoking in the household in the past 2 weeks, number of times frying was reported during the last week of the 2-week monitoring period, number of times other types of cooking occurred, number of times a smoky cooking event (e.g., burned

toast) occurred, number of hours per day with windows open, presence of a space heater, use of a HEPA filter, frequent dusting, use of a wood stove or fireplace, use of a gas oven or gas stove for heating, use of a humidifier, use of an air conditioner, burning of incense and candles, whether the participant lived in an apartment, and the absolute value of the indoor–outdoor temperature difference. The outdoor level and smoking variable were included in the model *a priori*, and then an empirical model based approach was employed to select from the additional variables. The absolute value of the indoor–outdoor temperature difference was added as both a linear and quadratic term, because of its complex relationship with air exchange rates.

For homes with lower indoor than outdoor concentrations, increased air exchange results in increased particle concentrations because dirty outdoor air is replacing clean indoor air. A study of air exchange versus temperature in several hundred California homes (Wilson et al. 1996) found that for moderate outdoor temperatures, people tended to open their windows and increase air exchange. However, for somewhat larger temperature differences, windows tended to be closed and air exchange was reduced. Then air exchange rates increase again as still larger indoor–outdoor temperature differences cause increased pressure differences between indoor and outdoor air. Thus, there is a quadratic dependence of air exchange on the absolute indoor–outdoor temperature difference. Further complication is provided by the relation between indoor and outdoor concentrations. Homes with strong indoor sources of particles show reduced concentrations with increased air exchange, whereas the reverse is true for homes with few or weak indoor sources. This was shown for a single home monitored for 18 months—indoor concentrations increased by about a factor of 2 for air exchange rates above  $0.8 \text{ hr}^{-1}$  when no sources were active, and decreased by about the same factor for air exchange rates below this level when cooking or candle burning was occurring (Wallace et al. 2002).

The sources of variability of the indoor particle concentrations were investigated using a four-level nested variance components model applied to the full set of repeated visits. This mixed model yielded estimates of variation associated with four different sources. They were site-to-site variability (the estimate of the variance between site averages); variability between households within a site (the estimate of the expected variance of household averages within each site); variability between visits for a given household (the estimate of the expected variance of visit averages for each household) and the day-to-day variance (the estimate of the expected variance of daily averages within each household). Homes with and without

smoking were modeled separately using the significant fixed effects from the mixed model mentioned above.

## Results

Of a total of 751 installations of pDR devices in the homes, 701 resulted in some recorded data and a calibration record. The data from these 701 visits were subjected to careful quality control. Samples were flagged when *a*) zero drift was greater than  $2 \mu\text{g}/\text{m}^3$  and *b*) the 14-day average pDR value at any site was more than  $2 \mu\text{g}/\text{m}^3$  greater than the internal time-weighted average computed by the pDR software. A total of 120 visits were eliminated based upon these screening criteria. From the remaining 581 visits, any visit with less than 10 days of complete data was removed, eliminating an additional 60 visits. (A day was considered complete if at least 18 hr of data were collected, each hour having at least four recorded 10-min averages.) From this total of 521 visits with valid data for at least 10 days, the earliest visit with acceptable data was chosen for each participant. This was done to give each home equal weight. This provided a final sample size of 294.

The relative SD (RSD) of the pDRs based on 2,300 duplicate 1-hr average measurements was 19.7%, with an SD of 21.8%. The mean RSD by site varied from 14.0% to 26.5%. The RSD based on 13,734 10-min average measurements was 20.2%, very close to the 1-hr RSD, indicating that lack of synchronicity of the pDRs was not a serious concern. These RSDs are a combined measure of precision and bias (the difference between the averages of two or more colocated monitors, which may derive from slightly different baselines established during the calibration procedure or to zero drift). When corrected for bias, the precision of the monitors averaged 16.6% with an SD of 21.7%.

The LOD of the pDRs (based on three times the SD of all colocated measurements below  $5 \mu\text{g}/\text{m}^3$ ) was determined to be approximately  $1.6 \mu\text{g}/\text{m}^3$ . The 28 days of colocated pDR and FRM measurements resulted in an FRM:pDR ratio of 0.66. All pDR data have been multiplied by this factor to agree with the gravimetric measurements.

Table 1 shows the number of homes by city, the total number of indoor data collection days, and the number of days with both indoor and outdoor data. The final column shows the total number of 10-min averages collected. In some cases, the device collected more than 14 days worth of data; data up to the 18th day were included, but additional days were removed. The number of homes monitored in each city ranged from 38 to 49; the total number of days monitored ranged from 576 to 743, and the total number of 10-min average values ranged from 82,000 to 106,000.



Table 2 provides some characteristics of the subjects and their homes, including information on smoking, cooking, use of air cleaners and air conditioners, incense, and other possible sources of airborne particles.

For each of the 294 subjects the average indoor concentration for the entire visit was calculated. Figure 1 shows the lognormal probability plot of the nominal 2-week (actually 10–18 days) averages by city. The plots are fairly linear with a hint of an upturn for the upper portion of the distribution, likely due to increased concentrations and variability in homes with smokers. Although Seattle and Tucson are slightly lower across most of the distribution, the lines for all cities have similar slopes, denoting homoscedasticity of variance across cities.

The distribution of hourly averages (Figure 2) again shows similar slopes for all cities, again with an indication of an upturn at the upper percentiles. Note that the shorter averaging time leads to higher concentrations, some exceeding  $1,000 \mu\text{g}/\text{m}^3$ , at the upper percentiles.

In Figure 3, we compare the distributions of the smoking and nonsmoking homes separately. This figure shows that each distribution is close to lognormal, and that the upturn noticed in the higher percentiles in the first two figures is probably caused by mixing two nearly lognormal distributions with greatly different means. Table 3 provides the indoor and outdoor arithmetic means and geometric means for each city as well as their associated SDs.

We calculated the subject-specific Spearman longitudinal correlations between the indoor concentration and the outdoor  $\text{PM}_{2.5}$  concentration. Table 4 shows the first, second, and third quartiles by city, separated by smoking status. As expected, the nonsmoking homes show a somewhat higher correlation of indoor with outdoor air than do smoking homes, although the median correlation of 0.42 is only moderate. The median correlation of 0.22 for smoking homes indicates that outdoor air concentrations are a poor indicator of indoor air levels in most homes with smokers. Table 4 also shows that the differences between persons at any one site are far greater than the median differences between sites; therefore these differences appear to be due to individual

**Table 1.** Number of homes and days monitored by site.

Site	No. of homes	Indoor days	Both indoor and outdoor days	Total data points
Overall	294	4,480	4,031	641,195
Boston	49	743	738	106,355
Bronx	38	605	604	86,797
Chicago	42	660	658	94,401
Dallas	40	585	585	83,687
Manhattan	45	679	679	97,283
Seattle	42	632	632	90,196
Tucson	38	576	135	82,476

differences in activities (e.g., cooking/not cooking, working/not working) rather than geographic or seasonal differences.

To investigate the diurnal variation of indoor PM and possible sources that might contribute to it, we divided the participants into four groups and plotted their daily variation. Using responses to the postvisit questionnaire, participants were coded as having smokers in their household ( $n = 79$ ), reporting frying meals 14 or more times in the previous week ( $n = 48$ ), neither of these items ( $n = 145$ ), or both ( $n = 22$ ). The pDR value for each hour of the day was averaged across the full visit for each subject, and then the hours for the subjects in each group were combined. The hourly average value for each group is plotted in Figure 4. As expected, values are lowest in the early morning hours, but then pick up rapidly starting around breakfast time. Concentrations in nonsmoking homes with frequent frying are higher than in nonsmoking homes without frequent frying and show visible increases at mealtimes. Both smoking groups are well above the nonsmoking groups, and those with both frequent frying and smokers in the household have the highest peak, which appears at approximately 1900 hours. The smokers without frequent frying have a maximum peak slightly later, at 2100 hours.

A regression of indoor versus outdoor concentrations was performed. According to the random component superposition (RCS) model (Ott et al. 2000), the slope of the regression line provides an estimate of the infiltration factor, or the fraction of the outdoor air concentration that remains airborne in the home under equilibrium conditions. The intercept is an estimate of the average contribution of indoor sources. A parallel zero-intercept line to the regression line

defines a “forbidden region” below the line in which few data points should reside, if the assumptions of the RCS model are met. Such data points below the line might indicate homes with stronger ability to reduce the impact of outdoor air particles, such as through the use of air cleaners or filtered air-conditioning systems. Regressions of indoor versus outdoor concentrations in the six cities with daily outdoor measurements resulted in a range of slopes from 0.41 to 0.82. Combining all data resulted in a slope of 0.50. The intercept in the combined regression, corresponding to the average contribution from indoor sources across all cities, was  $5.9 \mu\text{g}/\text{m}^3$ . By subtracting the calculated contribution to indoor air of the average 2-week outdoor concentration (i.e., multiplying it by 0.50), we obtained an estimated distribution of contributions from indoor sources. There was a wide variation in the distribution of the 2-week average indoor contributions, with an 84th percentile (1 SD on the log scale) concentration of  $32.7 \mu\text{g}/\text{m}^3$ , and a 97.7th percentile (2 SD) value of  $115.7 \mu\text{g}/\text{m}^3$ . These values suggest a geometric SD for the upper half of the distribution in the neighborhood of 3.5–4. This distribution of estimated concentrations due to indoor sources is similar to the distribution calculated from the indoor  $\text{PM}_{2.5}$  measurements in Riverside, California, and Toronto, Ontario, Canada (Wallace and Ott 2002).

Although it is clear that building characteristics and occupant behavior have a strong effect on the infiltration of outdoor air indoors, several studies suggest that over long periods of time, the average infiltration rates among all homes are similar for a given geographic region. In one study of 38 North Carolina residents monitored for  $\text{PM}_{2.5}$  exposure for 28 days over four seasons, a model assuming identical infiltration

**Table 2.** Selected household activity frequencies.

Characteristic	Mean <sup>a</sup>	Percent <sup>b</sup>
ICAS visit month		
12		36.4
18		47.6
24		16.0
Homes in ICAS environmental intervention group		48.0
Homes with at least one smoker		34.4
Homes with at least one smoky cooking event		31.6
Homes with a space heater		
Vented		1.4
Not vented		1.0
Homes with a working HEPA air cleaner at month 12		39.5
Homes dusting $\geq 6$ times a week		37.3
Apartment housing		49.3
Number of smokers in home	1.8	34.4
Number of cigarettes smoked inside home per day	9.0	34.4
Number of frying events in past week (out of 21 possible)	9.1	91.8
Hours per day a wood stove or fireplace was used to heat the home	1.7	1.0
Hours per day a gas oven or gas stove was used to heat the home	6.1	6.1
Hours per day that at least one window was open	12.3	77.5
Hours per day that a humidifier was used	11.2	4.4
Hours per day that an air conditioner was used	13.2	28.9
Hours per day that an air cleaner was used	18.8	36.4
Total hours in 2 weeks that candles were burned	34.9	28.9
Total hours in 2 weeks that incense was burned	4.0	15.0

<sup>a</sup>For only those homes with characteristic. <sup>b</sup>Percentage of homes with that characteristic.

rates for all 38 homes performed better (under the Aikake information criterion) than all other models tested, including a model allowing each home to have its own infiltration rate (Williams et al. Unpublished data). Also, the RCS model was able to estimate the average personal exposures to PM<sub>10</sub> of Toronto residents using only the average measured infiltration rate in Toronto and the calculated indoor-generated particle concentration distribution of residents of Riverside, California (calculated by again assuming a single constant infiltration rate for all homes in Riverside) (Ott et al. 2000). Thus, although calculating a single average infiltration rate for all homes in one area does not seem to have much use, in fact it has produced useful estimates of total particle exposure given only outdoor concentrations. Because most cities have outdoor concentrations measured over long periods of time but no corresponding indoor measurements, use of this assumption is virtually the only way to estimate the distribution of total exposure to particles in such cities.

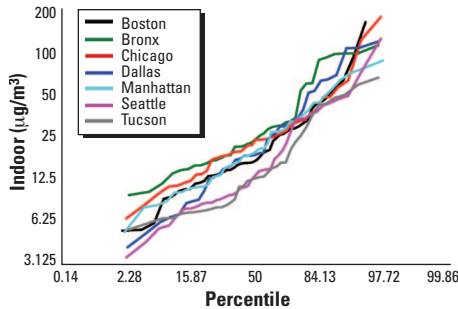
The final multivariate model, combined across all cities, is presented in Table 5. On average, 48% of the outdoor concentration infiltrated into the home. Smoking households added 37 μg/m<sup>3</sup> to the indoor concentration. However, this effect was reduced in those smoking homes with open windows, at a rate of 0.9 μg/m<sup>3</sup> per hour that a window was reported open. Indoor concentrations were 3.7 μg/m<sup>3</sup> higher in homes where at least one smoky cooking event had occurred in the prior week. Concentrations increased by 0.4 μg/m<sup>3</sup> per event involving frying or sautéing. Incense burning and apartment living also produced significant increases in indoor concentrations. Other variables considered were not selected at the 0.05 significance level. However, dusting was nearly significant (*p* = 0.071), and given the lack of sensitivity of the MIE monitor to the large particles produced by dusting, it is likely that dusting does produce a significant increase in coarse particles.

The four variance components are summarized in Table 6 for smokers and nonsmokers separately. For both groups, the day-to-day variance provides more than half the total, the visit-to-visit and person-to-person variances are roughly equal at about 14–21% of the total, and the site-to-site variance is the smallest of the four types, ranging from none to 6%. Note that smokers have much larger variance for each component. For nonsmokers, the site-to-site variance was so small compared with the other components that the model returned the lower bound, zero, for this variance component.

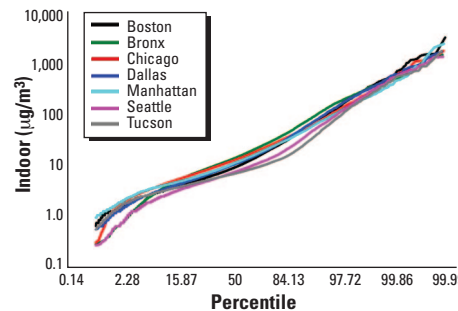
### Discussion

Earlier studies of indoor air particles have used gravimetric monitors to measure integrated concentrations over extended time intervals of 12 hr to several days (Dockery and Spengler

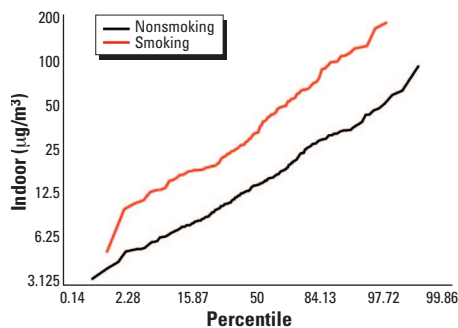
1981; Spengler et al. 1980). More recent studies have used continuous samplers such as the pDR (Howard-Reed et al. 2000; Liu et al. 2002; Quintana et al. 2001; Sioutas et al. 2000). Because the pDR employs optical scattering, which is dependent on size, shape, and refractive index of the particles, it will provide different results for different aerosol mixtures,



**Figure 1.** Log-probability graph of nominal 2-week average indoor air particle concentrations for seven locations.



**Figure 2.** Log-probability graph of hourly average indoor air particle concentrations for seven locations.



**Figure 3.** Log-probability graph comparing smoking (*n* = 101) and nonsmoking (*n* = 193) homes.

**Table 3.** Indoor and outdoor arithmetic mean (AM), geometric mean (GM), SD, and geometric standard deviation (GSD) (μg/m<sup>3</sup>).

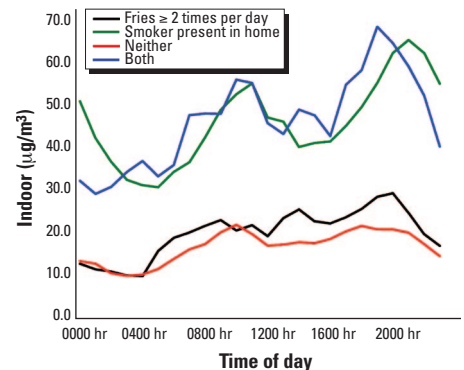
Site	Indoor AM (SD)	Outdoor AM (SD)	Indoor GM (GSD)	Outdoor GM (GSD)
Overall	27.7 (35.9)	13.6 (7.5)	17.2 (2.6)	11.8 (1.7)
Boston	28.2 (44.8)	11.3 (6.0)	16.2 (2.6)	9.9 (1.7)
Bronx	35.6 (37.3)	15.1 (7.6)	23.5 (2.4)	13.5 (1.6)
Chicago	29.1 (35.8)	17.3 (8.9)	19.5 (2.3)	15.0 (1.7)
Dallas	29.5 (36.7)	12.6 (5.4)	18.0 (2.7)	11.5 (1.6)
Manhattan	25.9 (28.7)	15.5 (8.4)	17.5 (2.4)	13.7 (1.6)
Seattle	21.0 (29.4)	11.3 (5.9)	12.5 (2.6)	10.0 (1.6)
Tucson	14.5 (17.8)	6.9 (2.5)	10.0 (2.1)	6.5 (1.4)

even if those aerosol mixtures had identical gravimetric concentrations. This will lead to increased imprecision when comparing pDR results with gravimetric results. Our estimate of the precision (mean RSD) of the pDR was 17%, somewhat larger than the values around 10% found by other studies (Liu et al. 2002; Williams et al. 2000).

**Table 4.** Longitudinal Spearman correlations between indoor and outdoor PM concentrations by site and smoking status.

Site	Q1	Median	Q3	No.
<b>Nonsmoking households</b>				
Overall	0.14	0.42	0.64	193
Boston	0.09	0.24	0.44	24
Bronx	0.26	0.49	0.73	24
Chicago	0.16	0.36	0.50	18
Dallas	0.12	0.36	0.57	30
Manhattan	0.50	0.59	0.70	27
Seattle	0.09	0.41	0.55	38
Tucson	-0.2	0.50	0.80	32
<b>Smoking households</b>				
Overall	-0.05	0.22	0.51	101
Boston	-0.04	0.14	0.33	25
Bronx	-0.05	0.25	0.58	14
Chicago	0.08	0.36	0.61	24
Dallas	-0.38	0.09	0.43	10
Manhattan	-0.01	0.33	0.52	18
Seattle	-0.24	0.05	0.30	4
Tucson	-0.80	0.40	0.50	6

Abbreviations: Q1, first quartile; Q3, third quartile.



**Figure 4.** Diurnal variation of hourly average concentrations in homes with smokers, homes reporting frequent frying (twice a day or more), homes with both characteristics, and homes with neither characteristic.

The relation between optical scattering intensity and mass is also affected by the density of the calibration aerosol. To the extent that this density differs from the density of the particles being sampled, this will lead to a bias in comparisons with gravimetric results. Several recent studies indicate that the bias associated with the pDR is on the order of 50–70% higher than gravimetric measures (Liu et al. 2002; Williams et al. 2000). This is consistent with the fact that the density of the calibration aerosol is 2.6, whereas the average density of ambient aerosols appears to be of the order of 1.55 (Sioutas et al. 2000). Our calculated value of 1.5 for the colocated pDR/gravimetric monitor ratio agrees well with these findings.

On the other hand, these disadvantages of increased imprecision and bias are counterbalanced by the information on short-term peaks and diurnal variation that can be provided by continuous monitors. Coupled with the use of activity diaries, peaks can be linked to sources in many cases. This information may be useful in suggesting ways to limit exposures.

Our evaluation of the pDR monitor suggests that it is dependable and relatively easy for lightly trained unskilled workers to deploy with valid results. Our modified calibration procedure allowed us to detect periods with positive or negative zero drift and to correct or remove the data. Also, the comparison with the internal averaging algorithm was another way to detect negative zero drift and remove invalid data. The amount of data removed by our quality assurance procedures was relatively substantial, at about 20% of all the data, but should have resulted in a much more reliable data set.

We considered the possibility that the intervention occurring in half of the homes before the ICAP study could have influenced the measured particle concentrations. Therefore, we analyzed the data separately for the intervention and control homes and observed no significant differences. Because the interventions were largely limited to the child's bedroom, whereas the measurements were made in the living rooms, this result is not unexpected.

Our results confirm the dominant contribution of smoking, when present, to indoor air concentrations. The mean indoor value in the 101 smoking homes was 46.5  $\mu\text{g}/\text{m}^3$ , compared with 17.8  $\mu\text{g}/\text{m}^3$  in the 193 nonsmoking

homes. This difference of 28.7  $\mu\text{g}/\text{m}^3$  for the smoking homes is in very good agreement with the difference of about 30  $\mu\text{g}/\text{m}^3$  reported by Spengler et al. (1980).

The results also confirm the smaller but significant contribution of cooking, as noted earlier by Pellizzari et al. (1993) and Özkaynak et al. (1996). Considering only the homes without smoking, there were 132 homes without a smoky cooking event and 58 with a smoky cooking event. There were 270 homes (92%) reporting at least one frying event. Mean indoor values were 31  $\mu\text{g}/\text{m}^3$  in homes with cooking and 23.5  $\mu\text{g}/\text{m}^3$  in homes without cooking. This difference of about 8  $\mu\text{g}/\text{m}^3$  is in reasonable agreement with the estimate of 10–15  $\mu\text{g}/\text{m}^3$  in Özkaynak et al. (1996).

Because the above bivariate analyses are subject to confounding by other important variables, we also developed a mixed-effects model to take into account outdoor concentrations as well as important indoor sources. Our mixed-effects model also showed an overwhelming effect of smoking. The increase due to smoking of about 37  $\mu\text{g}/\text{m}^3$  was similar to increases noted above in previous studies. Cooking also produced a significant increase in particles, with an average increase of 3.7  $\mu\text{g}/\text{m}^3$  due to a smoky cooking event plus about 3.5  $\mu\text{g}/\text{m}^3$  due to the average number of frying events (nine per week). Use of incense produced an average increase of nearly 6  $\mu\text{g}/\text{m}^3$ . Apartments averaged 4  $\mu\text{g}/\text{m}^3$  higher, possibly because smaller volumes created higher concentrations from indoor sources. There is some confounding between housing type and the study sites; however, a model including a study site covariate strengthened the relationship between the indoor level and housing type. Most previous studies have not detected the increases due to incense burning. However, one recent study has developed emission factors for many varieties of incense (Jetter et al. 2002).

The model includes an indoor–outdoor slope of 0.48 ( $\pm 0.04$ ), similar to the measured slopes of about 0.4–0.7 in previous studies (Liu et al. 2002; Pellizzari et al. 1993; Sarnat et al. 2000; Williams et al. 2000). (A slope of 0.48 indicates that 48% of the outdoor particle concentration will contribute to the indoor concentration, the other half being removed either by the building envelope or by depositing on

walls, floors, and ceilings.) Applying this factor to the overall mean outdoor concentration of 13.6  $\mu\text{g}/\text{m}^3$ , we find that outdoor particles accounted for only 25% of the average indoor concentration of 27.7  $\mu\text{g}/\text{m}^3$ .

The intercept in the model ( $4.1 \pm 1.8 \mu\text{g}/\text{m}^3$ ) is significantly greater than zero. Strictly speaking, if we had included all important indoor sources and also had no measurement error, the intercept should have been zero. Our nonzero intercept suggests that measurement variability caused by comparing optical scattering to gravimetric values may have been substantial, and also that we may have missed some important indoor sources in the model or that the participants may not have recalled some activities correctly.

Although not included in our final model, a parameter named “evidence of smoking” is of interest. This was a subjective estimate by technicians of the relative dirtiness of smoking homes. Of 101 homes with smoking, 35 had visible evidence of smoking. These 35 homes averaged 22  $\mu\text{g}/\text{m}^3$  higher concentrations than the those found in the 66 smoking homes without visible evidence of smoking, after controlling for all other significant variables. In future studies with observers but no particle measurements, this variable could be useful in estimating concentrations due to smoking.

In general, the average indoor concentrations by city tracked quite well with the outdoor ones, particularly if the geometric means are compared (Spearman correlation coefficient = 0.8). However, the absolute difference among cities—in the neighborhood of 5–10  $\mu\text{g}/\text{m}^3$ —is far smaller than the differences observed between homes and even between 2 days in the same home, as shown quantitatively by the comparison of variances (Table 6).

Because we had near-continuous measurements, we have been able to observe diurnal fluctuations in indoor particle concentrations. Peaks occur at mealtimes and during the evening after dinner (the latter ranging up to

**Table 5.** Final mixed effect model for indoor MIE measurements.

Characteristics	Estimate	SE	df	t-Value	p-Value >  t
Intercept	4.1	1.83	275	2.3	0.025
Outdoor concentration coefficient	0.48	0.04	3646	10.9	< 0.0001
Smoking household	37	5.63	3646	6.7	< 0.0001
Hours windows open (smoking households only)	-0.92	0.38	3646	-2.4	0.015
House smoky from cooking	3.7	1.76	3646	2.1	0.039
Number of frying events in last week	0.39	0.13	3646	3.0	0.0031
Burned incense in the last 2 weeks	5.4	2.43	3646	2.2	0.028
Apartment housing	4.0	1.65	3646	2.4	0.015

df, degrees of freedom.

**Table 6.** Variance components of 294 indoor measurements.

Characteristics	Variance [ $(\mu\text{g}/\text{m}^3)^2$ ]	Percent total variance
Nonsmokers comparison		
City to city	0	0
Between home (within city)	65.9	19
Between visit (within home, city)	51.3	14
Between day (within visit, home, site)	238.9	67
Smokers comparison		
City to city	64.6	6
Between home (within city)	213.6	21
Between visit (within home, city)	201.5	20
Between day (within visit, home, site)	524	52



about  $60 \mu\text{g}/\text{m}^3$ ), suggesting the power of the cooking and smoking activities. The overnight trough, reaching as low as  $20 \mu\text{g}/\text{m}^3$ , is also consistent with the cessation of activities during sleep.

The use of continuous monitors also allowed good estimates of the magnitude of short-term (1-hr) peaks. In all cities, at least 2% of all 1-hr measurements exceeded  $100 \mu\text{g}/\text{m}^3$ , and also in all cities some of these short-term peaks exceeded  $1,000 \mu\text{g}/\text{m}^3$ .

The previous studies mentioned above all showed very low correlations of indoor and outdoor particle concentrations. However, low correlations would be expected if indoor sources (e.g., smoking and cooking) were prevalent in some homes but not in others. Of more relevance to the daily mortality and morbidity time-series studies would be longitudinal correlations across multiple days within a single home. The major particle exposure studies included only 1 day per home, so only cross-sectional correlations were possible.

The results of our longitudinal regressions of indoor on outdoor air concentrations for each home showed fairly high median correlations of 0.3–0.6 in each city. About 30% of the correlations in nonsmoking homes and 20% of the correlations in smoking homes exceeded 0.5, a result in general agreement with the findings of the PTEAM pilot study (Wallace 2000) and a study in Phillipsburg, New Jersey (Lioy et al. 1990). These studies measured 18 persons over 5–7 days and 14 persons over 14 days, respectively. In both cases, about half the participants had fairly good longitudinal correlations ( $r > 0.5$ ). The median longitudinal personal–outdoor correlation coefficient in each case was well above the cross-sectional value (Wallace 2000). The more recent studies (Bahadori et al. In press; Janssen et al. 1997, 1998, 1999; Rojas-Bracho et al. 2000) all found similar results, again with about half the participants having relatively strong longitudinal correlations, and with median longitudinal personal–outdoor correlation coefficients ranging from 0.12 to 0.74, compared with cross-sectional coefficients ranging from  $-0.08$  to  $0.52$  (Wallace 2000). A large number of additional longitudinal studies of high-risk subpopulations (persons with respiratory or cardiovascular disease) have been sponsored by the U.S. EPA and will be reported on in the coming years (Liu et al. 2002). Several of these studies have been completed (Liu et al. 2003; Williams et al. 2000), again showing better longitudinal personal–outdoor and indoor–outdoor correlations than cross-sectional ones. Our finding of a similar range of correlations is satisfactory considering that we are comparing two different methods (optical scattering indoors vs. gravimetric outdoors).

The finding in our study and all previous studies that only a subset of participants show reasonably strong correlations of indoor with

outdoor air concentrations provides some support for the epidemiologic studies relating outdoor air to morbidity and mortality, because for this subset of the population actual exposure is correlated with the outdoor air measurements. However, the finding also suggests that many people have exposures that are not correlated with outdoor air concentrations. This would tend to produce misclassification and result in a weaker relationship between outdoor particle exposure and health effects than might actually exist. Indoor air particle exposures could thus contribute to morbidity and mortality if the toxicity of indoor air particles is comparable with that of outdoor air, as has been found in one study (Long et al. 2001).

Our questionnaire provides some interesting insights on particle-generating activities. For example, we found that in homes with cooking, the number of cooking events was very close to one per day. This may be compared with the PTEAM finding that the amount of cooking time in cooking homes averaged about 45 min per day. Both studies found comparable contributions to particle concentrations from cooking.

Our attempt to test the RCS model by running regressions of indoor versus outdoor concentrations had mixed results. One assumption of the RCS model is that the infiltration factor (given by the slope of the regression) will be similar from one city to the next; however, we found a range of slopes from 0.41 to 0.82 in the six cities with daily outdoor  $\text{PM}_{2.5}$  measurements. On the other hand, the similarity of the indoor distributions from city to city was consistent with another assumption of the RCS model: that particle-generating activities are similar in all cities. Also, the average infiltration factor of 0.50 for the combined results from all cities is similar to the range of 0.49–0.56 observed for the three major studies, each with at least 140 measurements, considered in the original RCS report (Ott et al. 2000). It may be that the number of measurements included in the individual regressions by city ( $\sim 30$ – $40$ ) was insufficient to provide the stable regression parameters that resulted from combining all 294 measurements.

With respect to our variance components model, the larger day-to-day variance compared with visit-to-visit variance would be expected, because a 14-day average should be much more stable than the daily average. An interesting finding is that for both the smokers and nonsmokers the site-to-site variance is dwarfed by the other components. This suggests that the indoor sources responsible for most indoor particle concentrations (e.g., smoking, cooking, incense burning, dusting) do not vary substantially in their frequencies from city to city, consistent with one basic assumption of the RCS model.

Although part of a larger study of asthma in children, this article has concentrated only

on the fine particle concentrations in their homes. We recognize that bioallergens such as dust mite and roach allergens are likely to have important effects on asthma exacerbation. Companion reports from ICAS will incorporate these bioallergen measurements as well as the fine particle concentrations reported in this article in models relating environmental levels to asthma health effects.

## Conclusions

The continuous sampling instrument selected for this study, the MIE pDR, appeared to work reasonably well. Positive and negative zero drift occurred in about 20% of cases, but the drift could be detected by the postcalibration step and the data corrected or discarded as appropriate.

Geometric mean values of indoor concentrations in the seven locations differed by less than a factor of 2, and the shape of the distributions was very similar across cities, both for the nominal 2-week averages and for hourly averages. The hourly averages exceeded  $100 \mu\text{g}/\text{m}^3$  for at least 2% of all measurements in all cities, and exceeded  $1,000 \mu\text{g}/\text{m}^3$  on at least a few occasions in each city.

The most important particle source in these homes was smoking. A second, less powerful source was cooking, particularly frying/sautéing or reporting a smoky cooking event. Use of incense also led to significant increases in particle concentrations. Dusting frequently also led to higher concentrations, possibly considerably higher than indicated by the pDR because of its lack of sensitivity for coarse particles. Infiltration of outdoor air added about half of the outdoor air concentration to the concentrations produced by the indoor sources, a result similar to that found by previous studies.

A new finding from this study was the observation that concentrations of fine particles peak in the late evening in homes with smoking, perhaps reflecting the influence of after-dinner smoking.

Regressions of indoor concentrations on outdoor concentrations by city resulted in a range of observed slopes from 0.41 to 0.82, inconsistent with one basic assumption of the RCS model. However, a regression using the combined data from all cities resulted in a slope and intercept quite similar to those found in other large-scale studies, and this larger regression was thus consistent with the previous RCS model results (Ott et al. 2000). Also, the resulting estimates of the average contribution of indoor sources agreed well with the mixed-effects model, which included many more parameters, and with the estimates made by the RCS model for other large-scale studies. We conclude that the combined results from all cities in this study are consistent with the RCS model.

A substantial percentage of homes (~30%) displayed relatively strong longitudinal correlations of indoor air particle concentrations with outdoor levels across multiple days. This suggests that only a portion of the population have personal exposures that are well correlated with outdoor concentrations, and therefore that epidemiologic investigations may underestimate the strength of the actual association of outdoor particles with mortality and morbidity. Depending on the relative toxicity of indoor sources compared with outdoor sources, this also opens the possibility that particles from indoor sources may have substantial effects on health.

Finally, differences across cities in 2-week mean indoor concentrations contributed very little to the total variance. This suggests that the major sources of indoor particles may have similar frequencies of occurrence in inner-city neighborhoods, making it possible to estimate the distribution of inner-city indoor concentrations in many cities other than those studied here.

**Appendix.** The Inner-City Asthma Study was a collaboration of the following institutions and investigators.

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