Most particulate matter (PM) health effects studies use outdoor (ambient) PM as a surrogate for personal exposure. However, people spend most of their time indoors exposed to a combination of indoor-generated particles and ambient particles that have infiltrated. Thus, it is important to investigate the differential health effects of indoor- and ambient-generated particles. We combined our recently adapted recursive model and a predictive model for estimating infiltration efficiency of air conditioning.

The data came from a large panel study in Seattle, Washington, that collected indoor, outdoor, and personal exposure data on 107 subjects over a 2-year period (Liu et al. 2003). The Seattle study also collected various health end points that included lung function and exhaled nitric oxide (eNO), a marker of airway inflammation, in a subset of children with asthma. In a previous article we reported eNO associations with 24-hr PM2.5 concentrations measured outside the home (4.3 ppb increase in eNO per 10-µg/m3 increase in PM2.5; 95% confidence interval [CI], 1.4 to 7.2), inside the home (4.2 ppb; 95% CI, 1.0 to 7.4), and on subjects (4.5 ppb; 95% CI, 1.0 to 7.9) (Koenig et al. 2003). In this article we describe the results of analyzing further the health data to test the associations between health outcomes and estimates of indoor-generated exposure (Eig) and ambient-generated exposure (Eag) based on subject time–location data and estimated particle infiltration efficiency (Fag) for the fraction of the outdoor concentration that penetrates indoors and remains suspended. We hypothesize that PM2.5 of outdoor origin has more effect on respiratory outcomes per unit mass than particles of indoor origin.
Materials and Methods
This study was conducted between winter 2000–2001 and spring 2001 in Seattle, Washington, as part of a larger exposure assessment and health effect panel study (Liu et al. 2003). Nineteen children, 6–13 years of age, were recruited from a local asthma and allergy clinic. All had physician-diagnosed asthma and were prescribed asthma medications daily or regularly. Ten of the subjects were not using inhaled corticosteroid (ICS) medication; nine were. Each subject in the panel was asked to participate for a 10-day monitoring session. Trained technicians made daily home visits to subjects between 1700 and 2000 hr to take air and health effect measurements.

Pollutant concentration measurements. PM measurements were taken inside and outside of each subject’s residence using the Harvard impactors for integrated PM$_{2.5}$ (H$_{2.5}$) concentrations and using the Radiance nephelometer (model 903; Radiance Research, Seattle, WA) at eight residences for continuous light-scattering measurements. Personal PM$_{2.5}$ measurements were collected from each subject using the Harvard personal environmental monitors. Detailed descriptions and evaluation of these samplers can be found in Liu et al. (2002). All integrated measurements were collected over 24 hr (1600 to 1600 hr) for 10 consecutive days. In addition, NO concentrations were monitored continuously at the Beacon Hill central site using a chemiluminescence monitor operated by the Washington State Department of Ecology (Olympia, WA).

Measurement of NO. Exhaled breath measurements were collected offline daily in the children’s homes into an NO inert and impermeable Mylar balloon for up to 10 consecutive days. Samples were collected in the afternoon or early evening at the child’s residence. Children were asked to forgo food intake for 1 hr before collection of exhaled breath. Exhaled breath was collected before lung function measurements, because deep inspirations affect NO concentration (Deykin et al. 1998). NO was quantified within 24 hr of collection using a chemiluminescence detector (model 903). NO concentrations were also measured in the outdoor air near the subjects’ homes, and NO concentrations were determined by subtraction of the indoor NO concentration from the outdoor NO concentration.

Measurement of lung function. During the daily visits, coached spirometry values consistent with American Thoracic Society criteria (American Thoracic Society 1995) were obtained with MicroDL spiroeters (Micro Medical, Lewiston, ME). Spirometry measurements included forced expiratory volume in 1 sec (FEV$_1$), forced vital capacity (FVC), and mid-expiratory flow (MEF). In addition, symptom forms were completed by the subjects during the previous 24 hr was reviewed and collected. Subjects also filled out a time-location-activity diary (TAD) with a 15-min resolution.

Estimation of PM exposure components. We previously described the use of a recursive mass balance model (RM) to estimate the average $F_{\text{inf}}$ for individual residences (Allen et al. 2003). The RM estimates of $F_{\text{inf}}$ agreed well with those estimated with the sulfur tracer method ($R^2 = 0.78$; $n = 14$ residences) (Sarnat et al. 2002). We also published estimates of $E_{\text{ig}}$ and $E_{\text{ig}}$ for PM$_{2.5}$ among a subset of the Seattle panel study subjects (Allen et al. 2004). We estimated the 24-hr average $E_{\text{ig}}$ and $E_{\text{ig}}$ for each subject using the RM $F_{\text{inf}}$ estimates from the indoor/outdoor nephelometer measurements, the indoor ($C_i$) and outdoor ($C_o$) PM$_{2.5}$ concentrations measured with H$_{2.5}$, and the fraction of the day ($F_d$) that the subjects reported being outdoors or in transit based on the TAD:

$$E_{\text{ig}} = (F_o)C_o + (1 - F_d)(C_i 	imes F_{\text{inf}})$$
$$E_{\text{ig}} = (1 - F_d)(C_i - C_o 	imes F_{\text{inf}})$$

Because nephelometer measurements were only valid at 8 of the 19 subjects’ residences, a predictive model based on RM $F_{\text{inf}}$ estimates from 62 residences in the Seattle panel study, residence type, outdoor temperature, average daily rainfall, and the use of air cleaners was constructed to estimate $F_{\text{inf}}$ in the remaining 11 homes (Table 1). The estimated $F_{\text{inf}}$ values from the predictive model were compared against those from the RM and validated against the conventional sulfur method (Allen et al. 2003), which uses the regression slope of indoor versus outdoor sulfur concentrations for each residence as the estimated $F_{\text{inf}}$. As a result of calculating $F_{\text{inf}}$ using both the RM and the predictive model, three groups of $E_{\text{ig}}$ and $E_{\text{ig}}$ estimates were created: a) those using the RM $F_{\text{inf}}$ values ($n = 8$ unique subjects), b) those using the predictive model $F_{\text{inf}}$ values ($n = 11$ unique subjects), and c) a combination of the above two—that is, RM $F_{\text{inf}}$ values when available and the predictive model $F_{\text{inf}}$ for the remaining subjects (henceforth called the combined model; $n = 8 + 11 = 19$ subjects).

Statistical analysis. We used a linear mixed effects model with random intercept to test for within-subject associations between eNO and various PM$_{2.5}$ exposure estimates. The model was:}

$$F_{\text{inf}} = \beta_0 + \beta_1 \text{Residence type} + \epsilon$$

where $\beta_0$ is the intercept, $\beta_1$ is the slope for the residence type variable, and $\epsilon$ is the error term. The results of the regression analysis are presented in Table 1.

Table 1. Results of regression analysis for $F_{\text{inf}}$ ($n = 62$ residences).

| Parameter | Estimate | SE   | 95% CI | p-Value |
|-----------|----------|------|--------|---------|
| Intercept | 0.41     | 0.07 | 0.28 to 0.54 | < 0.001 |
| Residence type | | | | |
| Private home (reference) | 0.03 | 0.05 | -0.08 to 0.14 | 0.61 |
| Group home | 0.19 | 0.06 | 0.07 to 0.31 | < 0.001 |
| Air cleaner | | | | |
| None (reference) | | | | |
| Ion generator | -0.07 | 0.05 | -0.16 to 0.02 | 0.14 |
| Filter | -0.08 | 0.07 | -0.22 to 0.05 | 0.23 |
| Electrostatic precipitator | -0.11 | 0.06 | -0.22 to 0.00 | 0.05 |
| Average outdoor temperature ($^\circ$C) | | | | |
| < 4 (reference) | 0.19 | 0.07 | 0.06 to 0.32 | < 0.01 |
| ≥ 4 | 0.32 | 0.07 | 0.18 to 0.45 | < 0.001 |
| Average daily rainfall (inches) | | | | |
| < 0.5 (reference) | 0.45 | 0.07 | 0.31 to 0.58 | < 0.001 |
| > 0.5 | 0.05–0.1 | 0.05 | -0.16 to 0.02 | 0.13 |
| > 0.1 | -0.15 | 0.06 | -0.29 to -0.04 | < 0.01 |

The regression coefficients are used to predict $F_{\text{inf}}$ in residences without nephelometer data (“predictive model”).

*At Beacon Hill Central Site. At Sand Point Way National Weather Service station.

Table 2. Distributions of residential indoor and outdoor concentrations and personal $E_{\text{ig}}$ and $E_{\text{ig}}$ ($\mu$g/m$^3$).

| Model        | Concentration | Total no. of monitoring events$^*$ | No. (days) | Mean | Minimum | 25% | Median | 75% | Maximum |
|--------------|---------------|-----------------------------------|------------|------|---------|-----|--------|-----|---------|
| Home indoor  | $E_{\text{ig}}$ | 248                               | 11.1       | 2.8  | 6.3     | 9.5 | 14.6   | 40.4| 8.8     |
| Home outdoor | $E_{\text{ig}}$ | 248                               | 7.0        | 1.8  | 4.2     | 5.9 | 9.2    | 22.6| 10.8    |
| Recursive    | $E_{\text{ig}}$ | 101                               | 2.1        | 0.0  | 0.0     | 1.2 | 2.3    | 17.2| 10.8    |
| Predictive   | $E_{\text{ig}}$ | 147                               | 6.0        | 1.3  | 3.4     | 5.0 | 7.5    | 22.6| 7.8     |
| Combined     | $E_{\text{ig}}$ | 248                               | 4.0        | 0.0  | 0.9     | 2.2 | 4.9    | 33.0| 8.8     |

| Model        | Concentration | Total no. of monitoring events$^*$ | No. (days) | Mean | Minimum | 25% | Median | 75% | Maximum |
|--------------|---------------|-----------------------------------|------------|------|---------|-----|--------|-----|---------|
| Home indoor  | $E_{\text{ig}}$ | 248                               | 6.4        | 1.3  | 3.7     | 5.5 | 7.8    | 22.6| 7.8     |
| Home outdoor | $E_{\text{ig}}$ | 248                               | 3.2        | 0.5  | 1.7     | 4.2 | 8.8    | 33.0| 8.8     |

Abbreviations: 25%, 25th percentile; 75%, 75th percentile.

*Number of unique subjects in parentheses.
included an interaction term between medication use and PM, a term for the within-subject, within-session (10-day monitoring period) effects, and a term for the subject between-session effects. We adjusted for the confounding variables of temperature, relative humidity, and, in the model for eNO, ambient NO measured at the Beacon Hill site. We also adjusted for subject age and body mass index (BMI). Our primary interest was the within-subject and within-session effect of PM. Analyses were conducted with all children from both winter and spring sessions. STATA 7.0 (Stata Corp., College Station, TX) was used for all health analyses, and SAS statistical package (version 8.0; SAS Institute, Cary, NC) using PROC Genmod with a repeated statement was used for the predictive model $F_{\text{int}}$ modeling. All three $E_{\text{ag}}$, $E_{\text{ig}}$, data sets (recursive, predictive, and combined) were examined with a focus on the combined data set.

The model used for the eNO analysis was as follows:

$$E[Y] = B_0 + B_1(X_{\text{ag}} - \overline{X}_d) + B_2(X_{\text{ig}} - \overline{X}_d) + B_3(X_{\text{ig}} - \overline{X}_d) + B_4(X_{\text{ig}} - \overline{X}_d) + B_5(X_{\text{ig}} - \overline{X}_d) + B_6(X_{\text{ig}} - \overline{X}_d) + B_7(X_{\text{ig}} - \overline{X}_d) + B_8(X_{\text{ig}} - \overline{X}_d) + B_9(X_{\text{ig}} - \overline{X}_d) + B_{10}(\text{Age}) + B_{11}(\text{Temp}) + B_{12}(\text{RH}),$$

where RH is relative humidity and BMI is body mass index. This basic model was used previously in the original analysis of the relationship between eNO and PM in the children with asthma (Koenig et al. 2003), where $X_{\text{ag}}$ is the PM$_{2.5}$ reading for individual $i$ on day $d$ during session $s$, $\overline{X}_d$ is the mean PM$_{2.5}$ reading for a subject during a session, $\overline{X}_s$ is the mean PM$_{2.5}$ reading for a subject during one or two sessions, $\text{med}$ is an indicator for medication use (constant for each subject), $Z_{\text{ig}}$ is the ambient NO reading for individual $i$ on day $d$ during session $s$, $\overline{Z}_d$ is the mean ambient NO reading for a subject during a session, and $\overline{Z}_s$ is the mean ambient NO reading for a subject during all sessions.

We also analyzed the data using generalized estimating equations (GEE) with an exchangeable working correlation matrix and robust SEs to adjust for autocorrelation in the data. The GEE model produced similar effect estimates.

**Results**

Nineteen children with asthma participated in this panel study in Seattle. All subjects completed one 10-day monitoring session, and 10 subjects completed two sessions. During this study, the home indoor and outdoor PM$_{2.5}$ concentrations averaged 9.5 and 11.1 µg/m$^3$, respectively (Table 2), whereas personal exposure to total PM$_{2.5}$ averaged 13.4 µg/m$^3$. The total personal PM$_{2.5}$ exposure was then separated into indoor- and outdoor-originated components using the RM for eight residences with nephelometer measurements and a predictive model for the remaining 11 residences. The predictive model for $F_{\text{int}}$ employed two important home characteristics, residence type, and the use of air cleaner, as well as outdoor temperature and precipitation as surrogates for changes of home ventilation conditions (Table 1). This predictive model agreed well with the RM ($R^2 = 0.60$) and the sulfur tracer $F_{\text{int}}$ estimates ($R^2 = 0.66$) (Figure 1). The average $F_{\text{ag}}$ for the 19 subjects was 0.56 ± 0.15 (range, 0.23–0.86). The average $E_{\text{ag}}$ and $E_{\text{ig}}$ from the RM model were not significantly different from those estimated from the predictive model (Table 2).

Thus, we pooled the $E_{\text{ag}}$ and $E_{\text{ig}}$ estimates from both models for the following health effect assessment. We examined the $E_{\text{ag}}$ and $E_{\text{ig}}$ estimates from the combined model for their associations with increase in eNO. Table 3 shows distributions for the health end points. In this analysis we found that eNO was associated with $E_{\text{ig}}$ estimated among subjects not on prescribed ICS medication (5.0 ppb per 10-µg/m$^3$ increase in estimated exposure; 95% CI, 0.3 to 9.7; Table 4). There was no association between eNO and $E_{\text{ag}}$ (Table 4). In contrast to our findings with eNO, associations between changes in lung function and estimated exposures were found for $E_{\text{ag}}$ but not for $E_{\text{ig}}$. Furthermore, the results were not statistically significant across all lung function measures. FEV$_1$ and FVC were both significantly negatively associated with $E_{\text{ag}}$ in children not using ICS (FEV$_1$, $p = 0.01$; FVC, $p = 0.00$), whereas MEF was negatively but not significantly, associated with $E_{\text{ig}}$ ($p = 0.35$). No significant associations were seen between lung function changes and the combined model estimates of $E_{\text{ag}}$.

Table 5 shows associations between the eNO and measured PM$_{2.5}$ on subjects (Harvard personal environmental monitor) and at home indoors and outdoors in the same 19 children included in the combined model. As shown in Table 5, associations were found between eNO and measured outdoor, indoor, and personal PM$_{2.5}$ ($p = 0.01$–0.03). In all

| Health measurement | No. of subjects (nos. sessions) | Person-years | Mean | Minimum | Median | 25% | 75% | Maximum |
|--------------------|---------------------------------|--------------|------|---------|--------|-----|-----|---------|
| eNO (ppb)          | 19 (29)                         | 240          | 15.4 | 5       | 9.7    | 12.5| 18.0| 79.8    |
| FEV$_1$ (L)        | 17 (29)                         | 269          | 1.8  | 0.5     | 1.4    | 1.9 | 2.2 | 3.4     |
| MEF (L/min)        | 17 (29)                         | 269          | 113  | 21      | 71     | 107 | 149| 420     |
| FVC (L)            | 17 (29)                         | 269          | 2.3  | 0.7     | 1.9    | 2.4 | 2.7 | 3.5     |

Table 3. Descriptive statistics of health outcomes.

| Exposure           | Model        | Use of medication | Change per 10 µg/m$^3$ | 95% CI          | p-Value |
|--------------------|--------------|-------------------|------------------------|-----------------|---------|
| $E_{\text{ag}}$    | Combined     | No                | 3.29                   | -1.14 to 7.73   | 0.15    |
|                    | Yes          | -4.94             | -10.94 to 1.06         | 0.11            |
| $E_{\text{ig}}$    | Combined     | No                | 4.98                   | 0.28 to 9.69    | 0.04    |
|                    | Yes          | 1.67              | -3.77 to 7.12          | 0.55            |
| $E_{\text{ag}}$    | Recursive    | No                | -0.19                  | -8.37 to 8.00   | 0.97    |
| $E_{\text{ig}}$    | Recursive    | Yes               | -0.47                  | 12.03 to 11.10  | 0.94    |
|                    | No           | 5.63              | -0.62 to 11.88         | 0.08            |
| $E_{\text{ag}}$    | Predictive   | Yes               | -4.30                  | -14.60 to 6.01  | 0.41    |
| $E_{\text{ig}}$    | Predictive   | No                | 3.46                   | -0.90 to 7.83   | 0.12    |
|                    | Yes          | -4.99             | -11.01 to 1.04         | 0.11            |

Table 4. Associations between eNO (ppb) and outdoor- versus indoor-generated particles in children with asthma: recursive model (n = 8), predictive model (n = 11), and combined model (n = 19).
cases, the changes were seen only in children not using ICS medications.

Discussion

Our study has shown that, for eNO, ambient-generated particles are more potent per unit mass than indoor-generated particles. This $E_{ag}$ effect on eNO using the combined model estimates also agreed well with the estimates from both the RM and the predictive model. The increases in eNO associated with $E_{ag}$ were 5.6 ppb for the RM estimates ($p = 0.08$), 5.3 ppb for the predictive model estimates ($p = 0.04$), and 5.0 ppb for the combined model ($p = 0.04$). Corresponding changes with $E_{inf}$ were not significant ($p = 0.41, 0.12, \text{and } 0.15$, respectively). In this respect, our results agree with those of Ebel et al. (in press), who found that outdoor-generated particles were associated with health outcomes, whereas nonambient particles were not in a group of subjects with COPD in Vancouver. These two studies demonstrate the usefulness of separating total personal particle exposures into indoor- and outdoor-generated components and the relative potency of indoor- and outdoor-generated particles.

Our conclusion that eNO is associated more strongly with outdoor-generated than indoor-generated particles is supported by the internal consistency of the results. For subjects with combined model estimates of $F_{inf}$, the estimated increase in eNO per 10-µg/m³ increase in PM$_{2.5}$ was 5.0 ppb ($p < 0.04$) for $E_{ag}$, which was greater than the 3.9 ppb for outdoor measured PM$_{2.5}$ ($p = 0.01$) because $E_{ag}$ takes into account personal activities and particle infiltration efficiency to arrive at a more accurate estimate of exposure to ambient-originated PM (Table 5). The effect of measured total indoor PM$_{2.5}$, a combination of indoor- and outdoor-generated particles, on eNO was 4.1 ppb/10 µg/m³ PM$_{2.5}$ ($p = 0.01$) in Table 5, which was reduced to a nonsignificant 3.3 ppb/10 µg/m³ PM$_{2.5}$ ($p = 0.15$) for $E_{inf}$ when the ambient PM contribution was removed from the total exposures. In all three exposure models, $E_{inf}$ was more strongly associated with eNO than was $E_{ag}$. Also, $E_{inf}$ showed an interaction with ICS use, as did our original study with outdoor, indoor, and personal measured PM$_{2.5}$ (Koenig et al. 2003).

Our lung function results show that exposure to particles generated indoors, but not outdoors, was associated with decrements of lung functions except for MEF. Furthermore, the association was not consistent across all three exposure models. Both combined ($n = 17$ subjects) and predictive models ($n = 9$ subjects) showed similar results for FEV$_1$ and FVC, whereas the recursive model estimates for eight subjects showed nonsignificant association between these lung function measures and $E_{inf}$. The fact that some lung function decrements were associated with indoor-generated particles indicates that the relationship between respiratory health and PM is complex. It was not surprising that the PM$_{2.5}$ associations with eNO and lung function were not consistent. This disagreement between eNO increases and lung function changes has been reported in clinical literature that consistently shows either no correlation or a negative correlation between changes in eNO and changes in FEV$_1$ among subjects with asthma (Dal Negro et al. 2003; Li et al. 2003; Nightingale et al. 1999; Steerenberg et al. 2003).

Outdoor particle concentrations are associated with a wide spectrum of respiratory health effects including respiratory symptoms in children with asthma (Delfino et al. 1998), lung function decrements in children with asthma (Delfino et al. 2002; Koenig et al. 1993), hospital admissions in the general population (Schwarz 1996; Sheppard et al. 1999), and mortality in the general population (Dockery et al. 1993; Schwarz 2000). On the other hand, there are also studies showing adverse respiratory health effects associated with indoor-generated particles including allergens, dust mites, fungal spores, endotoxins, and viruses (Long et al. 2001; Majid and Kammen 2001; Simoni et al. 2002; Smedbol et al. 2002; Wan and Li 1999).

Our results for eNO appear to be biologically plausible because asthma is an inflammatory disease and perturbations in asthma are expected to be associated with markers of airway inflammation. Several studies show relationships between eNO and outdoor exposure to PM or other air pollutants. One study found an association between exhaled NO values and high levels of outdoor carbon monoxide and NO, but not PM, in the Netherlands in healthy nonsmoking subjects (van Amsterdam et al. 1999, 2000). More recently, eNO levels were associated with exposure to PM$_{10}$, black smoke, nitrogen dioxide, and ambient NO in a panel study of children in the Netherlands (Steerenberg et al. 2001) and in a panel of adults with respiratory disease (Jansen et al. 2004). Adamkiewicz et al. (2004) presented data showing an association between measures of air pollution and eNO values in a panel of elderly nonsmoking subjects with cardiac disease in Steubenville, Ohio (USA). Their analysis found a 1.5-ppb increase in eNO (95% CI, 0.3 to 2.6) for a mean interquartile range increase in PM$_{2.5}$.

Model limitations. It is challenging to model personal exposure among children partly because of the elevated personal cloud and children’s movement between several indoor microenvironments (Liu et al. 2003; Wu et al. in press). Children in the Seattle panel study spent an average of 66% of their time indoors at home and 21% indoors away from home (primarily at school), whereas the adults in the larger panel study in Seattle spent an average of 83–88% of their time indoors at home (Liu et al. 2003). Because we only collected stationary indoor measurements and estimated $F_{inf}$ in the subjects’ residences, we made a strong assumption that all indoor environments encountered by the subject were represented by their residence. This assumption may have resulted in uncertainties in the exposure estimates because of the considerable fraction of time that this group spent in unmonitored indoor environments, especially school.

To make the most efficient use of our eNO and spirometry data, we developed a predictive model to estimate $F_{inf}$ (and therefore $E_{ag}$ and $E_{inf}$) in residences for which nephelometer data were not available (Table 1). Although the predicted $F_{inf}$ estimates were validated with an independent estimate of $F_{inf}$ (Figure 1), the predictive model is derived from the estimates produced by the recursive model, and as a result the predictive model estimates include errors introduced by a two-step modeling procedure. Nevertheless, the consistency of the associations between $E_{ag}$ and eNO for the RM and the combined model exposure estimates provides evidence of the reliability of the combined model’s $F_{inf}$ estimates.

Conclusion

Our eNO results support our hypothesis that PM$_{2.5}$ of outdoor origin could be more potent per unit mass than particles of indoor origin. However, our lung function data indicate that PM$_{2.5}$ of indoor origin might be more potent per unit mass in resulting in decrements of lung functions, although the results across functional tests were not consistent. If outdoor particles are more strongly associated with

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**Table 5. Results of eNO analyses with indoor, outdoor, and personal monitors for 19 children included in the combined model.**

| Measure | Use of medication | Change per 10 µg/m³ estimated PM$_{2.5}$ | 95% CI        | $p$-Value |
|---------|------------------|----------------------------------------|---------------|-----------|
| Personal* | No               | 4.48                                   | 0.95 to 8.00  | 0.01      |
|         | Yes              | −0.49                                  | −2.95 to 1.98 | 0.70      |
| Outdoor | No               | 3.90                                   | 0.91 to 6.88  | 0.01      |
|         | Yes              | 1.00                                   | −2.10 to 4.09 | 0.53      |
| Indoor  | No               | 4.13                                   | 0.87 to 7.38  | 0.01      |
|         | Yes              | −1.37                                  | −5.44 to 2.70 | 0.51      |

*Two sessions removed from personal PM analysis because of insufficient data.
adverse health outcomes than particles generated indoors, the fact that outdoor particles readily penetrate indoors would partially explain why epidemiologic time series studies consistently find associations between health outcomes and PM measured at outdoor fixed sites despite the fact that people spend most of their time indoors.

This is a preliminary study using a newly developed exposure source model that we hope will be useful to air pollution epidemiology. We tentatively conclude that partitioning personal exposure into indoor–versus outdoor-generated particles is useful in understanding the health effects of sources of personal PM$_{2.5}$ and that the effects of indoor–versus outdoor-generated particles differ for different health end points.

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