Home outdoor models for traffic-related air pollutants do not represent personal exposure measurements in Southern California

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Abstract. Recent studies have used measurements or estimates of traffic-related air pollutants at home or school locations to link associations between exposure and health. However, little is known about the validity of these outdoor concentrations as an estimate for personal exposure to traffic. This paper compares modelled outdoor concentrations at home with personal exposure to traffic air pollution of 63 children in two areas in Los Angeles in 2003/2004. Exposure monitoring consisted of sixteen 10-day monitoring runs, with each run monitoring 4 subjects concurrently with the active personal DataRAM for particulate matter <2.5 µm (PM₂.₅), elemental carbon (EC) and organic carbon (OC). One child per run had concurrent indoor/outdoor home monitoring. Measurements at central sites (24-hr PM₂.₅, EC, OC) were taken daily and concentrations of PM₂.₅, EC, and OC from traffic sources were calculated using the CALINE4 model for individual residences. We modelled outdoor concentrations of PM₂.₅, EC and OC with multilinear regression including GIS and meteorological parameters and adjusted for auto-correlation between repeated measurements. The model fit (R²) for home outdoor estimates was 0.94, 0.74 and 0.80 for PM₂.₅, EC and OC, respectively. Comparisons between these outdoor estimates and the personal measurements showed a good agreement for PM₂.₅ (R²=0.65-0.70) with a mean bias of −0.7±11.8µg for the smog receptor area, and 18.9±16.2µg for the traffic impacted area. However the outdoor estimates were not related to personal exposure for EC (R²=0.01-0.29) and OC (R²=0.03- 0.14). Conclusions: Predictions of outdoor concentrations can be used as approximations of personal exposure to PM₂.₅. However, they are not appropriate for estimating personal exposure to traffic-related air pollutants including EC and OC in studies of acute exposure-response relationships.

1. Introduction
Many children living in the traffic dominated Los Angeles area have an increased exposure to traffic air pollution due to their homes and schools being located close to major roads or highways [1]. A birth cohort study in this area found an increased risk of low birth weight and preterm birth in
association with residential proximity to heavy-traffic roadways [2]. Positive associations between exposure to traffic-related air pollution and asthma [3] and negative effects on lung growth independent of background air pollution levels [4, 5] were found in the Southern California Children’s Health Study.

Assessing effects or exposure of traffic exhaust is more complicated than assessing ambient air pollution in general due to the significant intra-urban spatial variation of traffic air pollution [6-8]. Therefore individual traffic index or dispersion models were used in many air pollution epidemiologic studies to link traffic-related air pollution to health effects [2-5, 9-11]. Only few studies used personal measurements to link acute respiratory effects to personal measurements of PM$_{2.5}$ [12-16]. Defino et al. [17, 18] presented one of the few studies associating personal exposure to traffic related pollutants to respiratory effects. He found associations between acute asthma outcomes and personal exposure to traffic pollutants but not with ambient PM$_{2.5}$ or OC. There may be other more toxic pollutants from traffic-related sources or other PM constituents, which are not sufficiently represented by home outdoor measurements that might be attributed to these acute asthma outcomes.

The current state-of-the-art exposure assessment using measured or modeled concentrations of NO$_2$, black smoke, and/or PM$_{2.5}$ outside homes or schools remains as the most affordable exposure assessment option for studies linking traffic pollution to health effects [4, 9, 11, 19-23]. Although these outdoor concentration estimates are widely used, only one study had examined the validity of using home or school outdoor concentrations to represent personal exposure to traffic pollution [24-26]. Van Roosbroeck and coworkers reported that the indices “living on a busy road” and “traffic intensity” at the street of residence predicted reasonably well long-term personal exposure to soot, but not exposure to PM$_{2.5}$ or nitrogen oxides. It remains unknown whether personal exposure to traffic exhaust can be represented by the outdoor estimates from the land-use regression or dispersion models.

This study evaluates the validity of using home outdoor measurements and estimates from land-use regression models for personal short-term exposure to PM$_{2.5}$, EC, and OC. Exposure models for PM$_{2.5}$, EC, and OC outside homes were constructed using geographic parameters and outdoor measurements. Predictions from these outdoor models were then compared to personal exposure measurements from children with asthma in two communities with different ambient pollution source profiles.

2. Methods

2.1. Study Design

This work was part of a panel study evaluating acute health effects of 63 children with asthma living in two areas of the Los Angeles air basin [17, 18]. Between August and December 2003 31 subjects were followed in Riverside, a smog receptor site, and in Whittier, a site immediately downwind of vehicular emission sources, 32 subjects were monitored from July to November 2004. Each child completed a time-place-activity diary every two waking hours during the 56 days of follow-up. Eight 10-day exposure monitoring runs were conducted in each area. Each run consisted of 4 subjects with concurrent personal monitoring of PM$_{2.5}$, EC and OC. Concurrent PM$_{2.5}$, EC and OC measurements were also taken at one child’s residence (indoor and outdoor) as well as at the central site. The CALINE4 dispersion model [27] was used to estimate traffic-source related PM$_{2.5}$, EC and OC at residences.

2.2. Exposure Measurements

The personal DataRAM (MIE pDR-1200; Thermo Electron Corp., Franklin, MA), an integrated nephelometer, was carried in a backpack during waking hours to measure one-minute average PM$_{2.5}$. 24-hr integrated PM$_{2.5}$ was collected on a 37 mm quartz filter (Whatman Inc, Florham Park, NJ) using a 2.5-μm sharp-cut cyclone ( BGI model GK 2.05, KTL cyclone, GI Inc., Waltham MA) operated at 4 L/min upstream to the pDR. Relative humidity (RH), and temperature loggers were used at 1-min intervals (Onset Computer Corp., Pocasset, MA). Data were downloaded and checked for quality and
compliance daily. All pDR data were adjusted for RH effect [28,29]. Detailed description and validation of the pDR can be found elsewhere [28].

One subject’s home per run had simultaneous indoor and outdoor monitoring concurrent with the personal measurements. Harvard Impactors (Air Diagnostics and Engineering, Inc., Naples, ME) [30] were used to collect 24-hour PM$_{10}$ and PM$_{2.5}$ on Teflon and quartz filters at home and central sites. The central site in Riverside was the South Coast Air Quality Management District (SCAQMD) monitoring site, while the one in Whittier was located at one subject’s residence. All quartz filters were analyzed for EC and OC using the thermal manganese dioxide oxidation technique [31].

Residences were geocoded using the TeleAtlas Eagle Geocoding service (TeleAtlas, Redwood City, CA). Distance to different types of roadways (freeway, arterial, and collector roads) were calculated in ArcGIS 8.3 (ESRI, Redlands, CA) based on TeleAtlas MultiNet™ USA roadway network. Annual average daily traffic (AADT) count data in 2000 were obtained from the California Department of Transportation (Caltrans), assigned to TeleAtlas roadway links, and adjusted to represent the years of 2003 and 2004 based on a statewide vehicle-miles-traveled growth (2.4% per year from 2000 to 2004) [32].

Traffic-source related PM$_{2.5}$, EC and OC at residences were estimated using CALINE4, a Gaussian dispersion model which predicts traffic-specific pollutant concentrations for receptors near roadways [27, 33]. The original CALINE4 model was modified to include contributions from road segments within 5 km to a receptor.

2.3. Analysis

For personal, home indoor, home outdoor and central site traffic air pollution concentrations summary characteristics and the Pearson correlation coefficients were calculated by area. Separate models were constructed for estimating 24-h averages of home outdoor PM$_{2.5}$, EC and OC using pooled data from both areas and stepwise linear regression with a 0.1 significance level for entering and retaining predictors. A random effect was added to the final models to account for data clustering within homes. In order to minimize the temporal variation we also used the same modeling approach to predict the ratios of home outdoor to central site measurements.

All models were examined for reliability using the leave-one-out cross validation. Each observation was removed from the dataset and evaluated against its model prediction, which was calculated using the remaining observations. To compare predicted home outdoor concentrations with measured personal exposure, R$^2$ from the linear regression and the bias (the differences between the measured and modeled values) were reported. All statistical analyses were computed with SAS 9.1 (SAS Institute Inc., Cary, NC).

3. Results

3.1. Quality control

The data collection rate for personal measurements of PM$_{2.5}$, EC and OC ranged between 76% to 89% in Riverside and 94% to 95% in Whittier. Data from 4 of the 31 subjects in Riverside were excluded from analysis, including one with home monitoring, due to inadequate geocoding data. From the 16 runs, outdoor measurements from 7 Riverside homes (excluding one above) were pooled with those from 8 Whittier homes. A total of 131 PM$_{2.5}$ and 129 EC/OC measurements at the home outdoor sites with matched central site measurements were valid for modeling.

3.2. Exposure data

In Table 1 daily averages of PM$_{2.5}$, EC and OC concentrations are summarized. Central-site and home outdoor PM$_{2.5}$, EC and OC concentrations were higher in Riverside than in Whittier, whereas average personal PM$_{2.5}$ EC and OC were higher in Whittier than in Riverside. Indoor PM$_{2.5}$ concentrations at
Table 1. Daily averages of air pollution measurements and GIS parameters at subjects’ homes by sample type and location.

|                      | Riverside Panel (N=27)                  | Whittier Panel (N=32)                  |
|----------------------|-----------------------------------------|----------------------------------------|
|                      | N (missing) | Mean (Std Dev) | Median | Min / Max | N (missing) | Mean (Std Dev) | Median | Min / Max |
| **Daily air measurements (24-hr)** |             |                |        |           |             |                |        |           |
| Central Site         |             |                |        |           |             |                |        |           |
| PM$_{2.5}$ (µg/m$^3$)$^b$ | 76 (3)   | 28.5 (20.1)    | 22.1   | 6.1 / 87.2 | 74 (5)     | 16.7 (10.2)    | 14.9   | 2.8 / 66.8 |
| PM$_{2.5}$ EC (µg/m$^3$) | 70 (9)   | 1.59 (0.82)    | 1.35   | 0.36 / 5.04 | 76 (3)     | 0.69 (0.44)    | 0.59   | 0.14 / 2.95 |
| PM$_{2.5}$ OC (µg/m$^3$) | 70 (9)   | 6.09 (2.74)    | 5.89   | 1.55 / 19.73 | 76 (3)     | 3.89 (1.50)    | 3.73   | 1.64 / 8.82 |
| **Personal Exposure** |             |                |        |           |             |                |        |           |
| PM$_{2.5}$ (µg/m$^3$)$^c$ | 196 (70) | 26.8 (19.0)    | 21.1   | 5.8 / 98.4 | 299 (17)   | 36.4 (26.8)    | 29.1   | 7.6 / 220.0 |
| PM$_{2.5}$ EC (µg/m$^3$) | 234 (32) | 0.38 (0.98)    | 0.24   | 0.00 / 14.64 | 295 (21)   | 0.76 (1.32)    | 0.47   | 0.00 / 17.20 |
| PM$_{2.5}$ OC (µg/m$^3$) | 237 (29) | 4.81 (2.82)    | 3.86   | 0.98 / 17.32 | 301 (15)   | 6.83 (3.41)    | 6.43   | 2.18 / 31.50 |
| **Outdoor home**      |             |                |        |           |             |                |        |           |
| PM$_{2.5}$ (µg/m$^3$)$^b$ | 65 (4)   | 23.2 (16.4)    | 18.0   | 3.8 / 71.8 | 70 (9)     | 17.5 (10.4)    | 15.8   | 3.2 / 64.6 |
| PM$_{2.5}$ EC (µg/m$^3$) | 63 (6)   | 1.01 (0.44)    | 0.97   | 0.25 / 2.23 | 78 (1)     | 0.84 (0.51)    | 0.70   | 0.21 / 3.10 |
| PM$_{2.5}$ OC (µg/m$^3$) | 63 (6)   | 5.14 (1.82)    | 5.44   | 1.19 / 9.19 | 78 (1)     | 4.50 (1.87)    | 4.22   | 2.05 / 10.28 |
| **Indoor home**       |             |                |        |           |             |                |        |           |
| PM$_{2.5}$ (µg/m$^3$)$^b$ | 66 (3)   | 12.6 (7.6)     | 10.4   | 2.9 / 33.8 | 69 (10)    | 16.2 (7.3)     | 16.3   | 3.6 / 46.9 |
| PM$_{2.5}$ EC (µg/m$^3$) | 61 (8)   | 0.66 (0.32)    | 0.67   | 0.07 / 1.55 | 78 (1)     | 0.79 (0.92)    | 0.62   | 0.14 / 7.75 |
| PM$_{2.5}$ OC (µg/m$^3$) | 61 (8)   | 5.35 (2.11)    | 5.02   | 1.42 / 11.57 | 78 (1)     | 5.96 (2.39)    | 5.38   | 2.64 / 13.53 |
| **CALINE4 estimates (24-hr)** |             |                |        |           |             |                |        |           |
| **Home outdoor**      |             |                |        |           |             |                |        |           |
| PM$_{2.5}$ (µg/m$^3$)  | 266 (0)   | 4.4 (3.1)      | 3.8    | 0.4 / 17.0 | 316 (0)    | 5.1 (2.6)      | 4.4    | 1.8 / 17.2 |
| PM$_{2.5}$ EC (µg/m$^3$) | 266 (0)  | 0.46 (0.32)    | 0.38   | 0.04 / 1.69 | 316 (0)    | 0.51 (0.28)    | 0.43   | 0.18 / 1.79 |
| PM$_{2.5}$ OC (µg/m$^3$) | 266 (0)  | 2.28 (1.62)    | 2.06   | 0.22 / 9.68 | 316 (0)    | 2.63 (1.19)    | 2.37   | 0.93 / 7.67 |
| **GIS variables**     |             |                |        |           |             |                |        |           |
| Population density   | 27 (0)    | 1701 (1072)    | 1696   | 141 / 3782 | 32 (0)     | 3577 (1551)    | 2952   | 978 / 6575 |
| (1/km$^2$)            | 27 (0)    | 2684 (1993)    | 2175   | 460 / 6938 | 32 (0)     | 1372 (937)     | 1084   | 121 / 3339 |
| Minimal distance to   | 27 (0)    | 487 (543)      | 310    | 6 / 2593    | 32 (0)     | 335 (320)      | 248    | 20 / 1130  |
| highways (m)          | 27 (0)    | 496 (347)      | 401    | 26 / 1275   | 32 (0)     | 291 (328)      | 144    | 4 / 1238   |
| Minimal distance to   | 27 (0)    | 14 (11)        | 12     | 2 / 67      | 32 (0)     | 19 (19)        | 12     | 10 / 97    |
| arterial roads (m)    | 27 (0)    | 14 (11)        | 12     | 2 / 67      | 32 (0)     | 19 (19)        | 12     | 10 / 97    |
| Collectors (m)        | 27 (0)    | 14 (11)        | 12     | 2 / 67      | 32 (0)     | 19 (19)        | 12     | 10 / 97    |
| Local roads (m)       | 27 (0)    | 14 (11)        | 12     | 2 / 67      | 32 (0)     | 19 (19)        | 12     | 10 / 97    |

$^a$ missing and excluded together.

$^b$ 24-hr PM$_{2.5}$ collected with Harvard Impactors.

$^c$ 24-hr averages of 1-min pDR readings.

Riverside homes were lower than outdoor and central-site concentrations, while home outdoor and indoor concentrations were comparable to the central site levels in Whittier. The CALINE4 model estimates for PM$_{2.5}$, EC and OC from local mobile sources were expectedly lower than measurements from all sources. Based on the CALINE4 estimates, about 30% of outdoor PM$_{2.5}$ and 60% of EC/OC came from local traffic in Whittier, while in Riverside the local traffic contribution was only 20% and
45% for PM$_{2.5}$ and EC/OC, respectively. This is in accordance with the GIS data showing that subjects in Whittier lived closer to major roads than subjects in Riverside (Table 1).

3.3. Correlation between personal, central site and home measurements

Home outdoor and personal concentrations of PM$_{2.5}$ showed good correlations to those at the central, home outdoor and home indoor sites (Table 2). Weaker but still strong correlations were found for OC between home outdoor and central site measurements. Correlations were smaller between indoor and outdoor OC measurements and between personal and central site OC measurements. The smallest correlation coefficients were found for EC, between the home outdoor and central sites and between home indoor and outdoor sites. Personal EC measurements in Riverside were significantly correlated with only home outdoor measurements, whereas in Whittier a strong correlation was observed with measurements at both the home indoor and the central sites.

| Table 2. Pearson correlations between the concentrations at different locations by pollutant. |
|---------------------------------------------------------------|
|                  | Riverside                  | Whittier                  | Pooled                  |
|                  | Central Site | Home outdoor | Home indoor | Central Site | Home outdoor | Home indoor | Central Site | Home outdoor | Home indoor |
| PM$_{2.5}$      |              |              |             |
| Home outdoor    | 0.95**       | 0.80**       | 0.97**      | 0.58**       | 0.95**       | 0.64**      |
| Personal        | 0.78**       | 0.81**       | 0.84**      | 0.83**       | 0.77**       | 0.74**      | 0.60**       | 0.65**       | 0.79**      |
| OC              |              |              |             |
| Home outdoor    | 0.83**       | 0.48**       | 0.79**      | 0.65**       | 0.79**       | 0.55**      |
| Personal        | 0.16*        | 0.48**       | 0.25*       | 0.22**       | 0.53**       | 0.54**      | 0             | 0.26**       | 0.39**      |
| EC              |              |              |             |
| Home outdoor    | 0.60**       | 0.52**       | 0.32**      | 0.38**       | 0.26**       | 0.39**      |
| Personal        | 0.05         | 0.33**       | 0.13        | 0.29**       | 0.18*        | 0.89**      | -0.01        | 0.17**       | 0.80**      |

* = p-value <0.05.  
** = p-value <0.001.

3.4. Models for home outdoor pollution

Table 3 shows the final models for home outdoor measurements of PM$_{2.5}$, OC, and EC for both areas. All 3 models were dominated by the central site measurements and included minimal distance to highways. The PM$_{2.5}$ model ($R^2=0.94$) was dominated by central site PM$_{2.5}$. The OC model ($R^2=0.80$) was also influenced by the population density and wind direction. The EC model, with the smallest $R^2$ value among all 3 models ($R^2=0.74$) was also influenced by the population density, minimal distance to highways, and wind direction. The CALINE4 estimates did not enter the OC or the EC model.

Models for the ratios of home outdoor to central site measurements had smaller $R^2$ values than the concentration models because of the adjustment for the seasonal variation. The major predictors in the model for the PM$_{2.5}$ ratio included minimal distance to highways, wind direction, and temperature ($R^2=0.28$). The major predictors in the OC ratio model were distances to highways and collector roads, wind direction, and temperature ($R^2=0.62$). For the EC ratio model minimal distance to highways to the EC model was replaced by the CALINE4 estimate, which was the major predictor, followed by wind direction and RH ($R^2=0.38$).

Predictions from the concentration and ratio models showed comparable performance for predicting home outdoor concentrations, after the ratio estimates were multiplied by the central-site measurements. Subsequently we used the concentration models for the following evaluation of their representativeness to personal exposure measurements.
Table 3. Results of multilinear regression modeling for home outdoor air pollution measurements.

| Variable | Estimate | SE | % of model $R^2$ explained | Adj. Model $R^2$ |
|----------|----------|----|----------------------------|------------------|
| **PM$_{2.5}$ Model (N=131)** | | | | 0.94 |
| Central site PM$_{2.5}$ measurement ($\mu$g/m$^3$) | 0.92 ** | 0.01 | 98 |
| Minimal distance to highway (m) | -3.4E-04 | 2.3E-04 | <1 |
| Average temperature at central site ($^\circ$F) | -0.04 | 0.03 | <1 |
| Weekly time term (sin) | 0.51 * | 0.24 | <1 |
| PM$_{2.5}$ CALINE4 estimate at home ($\mu$g/m$^3$) | 0.29 ** | 0.15 | <1 |
| **OC Model (N=129)** | | | | 0.80 |
| Central site OC measurement ($\mu$g/m$^3$) | 0.83 ** | 0.04 | 77 |
| Population density (per km$^2$) | 3.3E-04 ** | 5.1E-05 | 16 |
| Average wind direction from W to N | 0.27 | 0.21 | 0.27 |
| Minimal distance to highway (m) | -1.6E-04 ** | 5.4E-05 | <1 |
| Average wind direction from E to S | 0.02 | 0.25 | <1 |
| Average wind speed (miles/hour) | -0.20 * | 0.10 | <1 |
| Frequency of wind direction from E to S | -0.08 ** | 0.02 | <1 |
| Relative humidity at central site (%) | 0.01 | 0.01 | 0.01 |
| Daily time term (sin) | -0.19 | 0.11 | <1 |
| Frequency of wind direction from S to W | -0.03 * | 0.02 | <1 |
| **EC Model (N=129)** | | | | 0.74 |
| Central site EC measurement ($\mu$g/m$^3$) | 0.66 ** | 0.04 | 64 |
| Population density (per km$^2$) | 8.7E-05 ** | 1.5E-05 | 23 |
| Minimal distance to highway (m) | -5.0E-05 ** | 1.7E-05 | 7 |
| Frequency of wind direction from E to S | -0.03 ** | 6.8E-03 | 4 |
| Relative humidity at central site (%) | 5.8E-03 ** | 1.7E-03 | <1 |
| Frequency of wind direction from S to W | -0.01 | 4.5E-03 | <1 |
| Average wind direction from N to E | -0.20 * | 0.09 | <1 |

* $p<0.05$.

** $p<0.01$.

SE: Standard error

3.5. Comparisons between measured personal exposure and outdoor model predictions

65% of the variation in personal PM$_{2.5}$ measurements in Riverside and 70% in Whittier were explained by the estimates of home outdoor PM$_{2.5}$ (Figure 1A). The prediction bias (measured minus predicted concentration) was larger in Whittier than in Riverside. Although the models were constructed using outdoor measurements from only 25% of the subjects’ homes, the model predicted equally well for all subjects regardless of the availability of home monitoring data (Figure 1B).

The predictions of the home outdoor OC model explained very little of the variation in the personal exposure to OC in both Riverside ($R^2=0.03$) and Whittier ($R^2=0.14$, two outliers excluded). The prediction bias was again larger in Whittier (2.2 μg/m$^3$) than in Riverside (-1.1 μg/m$^3$). Outdoor OC estimates at monitored homes seemed to explain a slightly higher percentage of the variation in personal OC ($R^2=0.06$ vs. 0.003, respectively) (results not shown).

Similarly, home outdoor EC estimates did not relate to personal exposure to EC (Figure 2). Analysis without four outliers increased the model fit to 29% (from 7%) in Whittier. The prediction bias was -0.8 μg/m$^3$ in Riverside and -0.2 μg/m$^3$ in Whittier. As with the OC model, the EC model predicted better for the subjects with home monitoring (Figure 2B).
Figure 1. Measured personal PM$_{2.5}$ vs. predicted home outdoor PM$_{2.5}$ (concentration model with random effect): (A) by city, (B) by homes monitored and not monitored ($\beta_1 =$ estimate of regression slope; **p<0.01). Statistics for measured (Meas), predicted (Pred) and the difference (Bias = Meas-Pred) PM$_{2.5}$ are also shown next to the Figure.
3.6. Sensitivity analysis
To test for an improvement of the prediction by smoothing out the temporal variation, we averaged the personal measurements and modeled home outdoor values over the 10 monitoring days for each subject/home and re-examined the relationships. The $R^2$ for PM$_{2.5}$ increased in Riverside from 0.65 (daily averages) to 0.77 (10-day averages). However, some large prediction biases in Whittier had a
larger influence on the 10-day mean and reduced the $R^2$ value from 0.70 to 0.48. Using the 10-day averaged EC data, there was a modest increase in the association from 1% to 24% in Riverside and from 7% to 17% in Whittier. Similar results were observed for OC with an increase from 3% to 21% in Riverside and from 9% to 13% in Whittier.

Analysis stratified by “Near source” days, meaning days with at least one 15-min entry of near smoking or cooking, revealed only a significant difference for PM$_{2.5}$ in Whittier. The $R^2$ for modeled home outdoor PM$_{2.5}$ and personal PM$_{2.5}$ exposure was 0.74 for the days without PM sources and 0.55 for days with any source.

4. Discussion
One finding of this study is the importance of area-specific modeling as indicated by the reduced predictive power of the home outdoor models when the two areas were pooled. Overall results for the predictions of air pollution outside homes indicate that PM$_{2.5}$ is spatially homogeneous within the two areas, and while this is the case for OC to some extend, it is not the case for EC.

The contribution from traffic to the home outdoor concentrations was higher in Whittier due to the subjects living closer to highways and arterials, although the central site and home outdoor concentrations were higher in Riverside, the smog receptor site. There PM mass concentrations were influenced by sources other than local traffic exhaust. This is in agreement with results from Kim et al. identifying Riverside as a receptor site, affected by secondary air pollution and local traffic emissions while Downey, located near central Los Angeles (10 km west of Whittier) was identified as a source site primarily affected by local traffic emissions [34].

Personal exposure to EC was correlated only slightly with home outdoor measurements and model predictions in Riverside and strongly with home indoor measurements in Whittier. However, the outdoor EC model could not predict well the exposure of subjects whose homes were not monitored. In Riverside, this might have been partially due to measuring errors. In Whittier, this points to personal exposure to EC being sensitive to local sources which may not be captured by the home outdoor measurements or estimates alone. Correlations between personal PM$_{2.5}$ and indoor concentrations were stronger than between personal PM$_{2.5}$ and home outdoor or central site concentrations due to the relatively higher fraction of time spent at home indoors (>70%). This is in accordance with results in studies in southern California [16] and other cities [30, 35-38]. However, the performance of the PM$_{2.5}$ home outdoor model for personal exposure was good, indicating that most personal exposure to PM$_{2.5}$ came from outdoor sources. The correlations between personal OC exposure and home outdoor OC were significant. However, the associations with home indoor and central site measurements differed by region. The stronger personal-home outdoor correlations likely indicated a significant influence of local outdoor sources of OC on personal exposure in both regions.

Central-site measurement was an important predictor for all our prediction models for home outdoor PM$_{2.5}$, EC and OC concentrations, although the degree of importance varied. Predictors indicating local sources and meteorological influences overwhelmed the CALINE4 estimates for the EC and OC models. CALINE4 estimates were not a significant predictor in the EC model, which is surprising as CALINE4 estimates and EC were considered specific for local traffic sources. In CALINE4 seasonal averages of meteorological parameters were used as the inputs, which might not reflect the variations in our daily measurements. In addition, heavy-duty truck count data were not available for all potentially important roadways in the study areas, which likely influenced the accuracy of the predictions. In contrast to the EC concentration model, CALINE4 became the main predictor in the EC ratio model. This indicates that the CALINE4 model is adequate in estimating spatial variations of traffic pollution in some degree.

Although many epidemiologic studies of traffic exhaust made the assumption of concentrations outside homes or schools being good indicators of personal exposure to traffic, we could show that this applies only to PM$_{2.5}$, reflecting the high correlations between personal and outdoor PM$_{2.5}$ from regional sources, and not to EC or OC, reflecting local sources. This may be partly why PM$_{2.5}$ associations with airway inflammation [17] and lung function [18] in children with asthma were seen
with personal PM$_{2.5}$ measurements but not when ambient PM$_{2.5}$ measurements were used. Although, independent effects from PM$_{2.5}$ and gaseous copollutants or from specific PM$_{2.5}$ constituents on respiratory health can not be ruled out either. Therefore these models might not be adequate for specific sources. It should also be noted that measurement errors might have been involved, especially in Riverside.

We expected an improvement of the model fit with longer averaging durations and/or simultaneous sampling across sites, as reported in longer-term exposure and chronic health effects studies [24, 26]. However, home outdoor EC and OC models averaged over 10 days still could not predict more than 25% of the variation in the personal measurements. Thus, outdoor models for EC or OC could not fully capture the temporal and spatial variation in short-term personal exposure to EC or OC.

As subjects spend most of the time indoors, we expected a negative influence of indoor sources on the performance of the home outdoor models for personal exposure. This was true for PM$_{2.5}$ in Whittier but it did not apply to PM$_{2.5}$ in Riverside or EC or OC in either region. This could be due to the limited number of records for indoor source reporting, reporting errors, and most likely, the relatively short occurrence of these indoor sources over the 24-hr sampling period. Similar results were found by [24] with no effect of indoor sources on personal soot.

5. Conclusions
Modeling of traffic-related air pollution at different outdoor locations is regularly used in epidemiological studies to represent personal exposures. In our study daily personal exposure of PM$_{2.5}$ was in good agreement with predicted PM$_{2.5}$ concentrations at home, validating the use of these outdoor estimates to assess the acute health impacts of short-term exposures to PM$_{2.5}$ for children. However, personal short-term exposure to OC or EC can not be approximated by the home outdoor estimates, mainly due to the contributions from sources other than the traffic exhaust outside the home. Future work should focus on building personal exposure models that incorporate additional information on personal activities and locations.

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Acknowledgments

The project described was supported by grant number ES11615 from the National Institute of Environmental Health Sciences (NIEHS), U.S. National Institutes of Health (NIH), and its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIEHS, NIH. The authors declare they have no competing financial interests.