Journal of the Air & Waste Management Association

ISSN: 1096-2247 (Print) (Online) Journal homepage: http://www.tandfonline.com/loi/uawm20

Associations between Personal, Indoor, and Residential Outdoor Pollutant Concentrations: Implications for Exposure Assessment to Size-Fractionated Particulate Matter

Mohammad Arhami , Andrea Polidori , Ralph J. Delfino , Thomas Tjoa & Constantinos Sioutas

To cite this article: Mohammad Arhami , Andrea Polidori , Ralph J. Delfino , Thomas Tjoa & Constantinos Sioutas (2009) Associations between Personal, Indoor, and Residential Outdoor Pollutant Concentrations: Implications for Exposure Assessment to Size-Fractionated Particulate Matter, Journal of the Air & Waste Management Association, 59:4, 392-404

To link to this article: http://dx.doi.org/10.3155/1047-3289.59.4.392

View supplementary material

Published online: 24 Jan 2012.

Submit your article to this journal

Article views: 247

View related articles

Citing articles: 10 View citing articles

Full Terms & Conditions of access and use can be found at
http://www.tandfonline.com/action/journalInformation?journalCode=uawm20

Download by: [University of North Texas] **Date:** 05 January 2017, At: 18:45

TECHNICAL PAPER

ISSN:1047-3289 *J. Air & Waste Manage. Assoc.* **59**:392–404
DOI:10.3155/1047-3289.59.4.392
Copyright 2009 Air & Waste Management Association

Associations between Personal, Indoor, and Residential Outdoor Pollutant Concentrations: Implications for Exposure Assessment to Size-Fractionated Particulate Matter

Mohammad Arhami and Andrea Polidori
University of Southern California, Department of Civil and Environmental Engineering, Los Angeles, CA

Ralph J. Delfino and Thomas Tjoa
Department of Epidemiology, School of Medicine, University of California–Irvine, Irvine, CA

Constantinos Sioutas
University of Southern California, Department of Civil and Environmental Engineering, Los Angeles, CA

ABSTRACT
The physical and chemical characteristics of indoor, outdoor, and personal quasi-ultrafine (<0.25 μm)-, accumulation (0.25–2.5 μm)-, and coarse (2.5–10 μm)-mode particles were studied at four different retirement communities in southern California between 2005 and 2007. Linear mixed-effects models and Spearman's correlation coefficients were then used to elucidate the relationships among size-segregated particulate matter (PM) levels, their particle components, and gaseous co-pollutants. Seasonal and spatial differences in the concentrations of all measured species were evaluated at all sites on the basis of *P* values for product terms. Outdoor quasi-ultrafine (UF) and, to a lesser extent, accumulation-mode particles were the two fractions that best correlated with outdoor concentrations of carbon monoxide (CO), nitrogen dioxide (NO_2), nitrogen oxides (NO_x; during both phases of the study), and ozone (O_3; only during the warmer months). Outdoor and indoor concentrations of CO, NO_2, and NO_x were more positively correlated to personal quasi-UF particles than larger size fractions. Despite these findings, it seems unlikely that these gaseous co-pollutants could confound epidemiologic associations between quasi-UF particles and adverse health effects. Overall, measured gaseous co-pollutants were weak surrogates of personal exposure to accumulation-mode PM, at least for subjects with similar exposure profiles and living in similar urban locations. Indoor sources were not significant contributors to personal exposure of accumulation and quasi-UF PM, which is predominantly influenced by primary emitted pollutants of outdoor origin. Correlations between personal coarse-mode PM and both outdoor and indoor gaseous co-pollutant concentrations were weak at all sites and during all seasons.

IMPLICATIONS
Health risks associated with exposure to PM can be confounded by the presence of gaseous co-pollutants. Thus, modeling results from time-series epidemiologic studies that include both gaseous and particulate pollutant concentrations should be interpreted with caution. A better understanding of the relationships among indoor, outdoor, and personal size-fractionated PM, the corresponding particle components, and gaseous co-pollutants of both indoor and outdoor origin is needed to elucidate the health risks associated with PM exposure.

INTRODUCTION
Several epidemiological and toxicological studies have found positive associations between levels of atmospheric fine particulate matter ($PM_{2.5}$; aerodynamic diameter [D_p] ≤ 2.5 μm) and acute or chronic adverse health effects.[1] The PM components that are believed to be responsible for the adverse health outcomes include transition metals, organic species (often reported using surrogate measurements such as organic and elemental carbon [EC], or organic carbon [OC] and EC, respectively), sulfate and nitrate salts, and bioaerosols.[2,3] In addition, PM properties such as mass, number, surface area, and especially size are important in evaluating particle toxicity. In addition to having the highest potential to penetrate deeply into the human lungs, ultrafine (UF) particles ($D_p \leq 0.10$ μm) collected in urban environments include particularly toxic species such as polycyclic aromatic hydrocarbons (PAHs). In a recent study conducted by Ntziachristos et al.,[4] size-fractionated ambient particulate matter (PM) samples (i.e., quasi-UF- [$D_p \leq 0.25$ μm], accumulation- [$D_p \leq 2.5$ μm], and coarse- [2.5 μm $\leq D_p \leq$ 10 μm] mode particles) were collected at four different locations in the Los Angeles Basin, and analyzed for their chemical composition and redox potential (an indicator of particle toxicity). The quasi-UF fraction had the highest redox activity on a per-PM mass basis, which was correlated with the higher mass fractions of particulate OC and PAHs (some of which are well-known carcinogens[5,6]) for this size range.

Although the link between PM exposure and adverse health effects has become widely accepted, ascertaining the true risk associated with exposure to PM is difficult, mainly because the concentrations of ambient particles and those of their gaseous co-pollutants {e.g., carbon monoxide (CO), nitrogen oxides (NO_x = nitric oxide [NO] + nitrogen dioxide [NO_2]), ozone (O_3), and sulfur dioxide (SO_2)} are often well correlated, and estimates of the health risks associated with PM exposure may be confounded by these gaseous species.[7–9] The National Research Council[10] listed the investigation of the potential confounding effect of gaseous co-pollutants on PM health effects as one of their research priorities.

Sarnat et al.[8] explored these confounding effects in Baltimore, MD,[9] and in Boston, MA, using integrated (24-hr) ambient and personal exposure data (i.e., $PM_{2.5}$, O_3, NO_2, and SO_2 concentrations) collected in summer and winter. In both cities, ambient gaseous levels were more strongly correlated with personal exposure to $PM_{2.5}$ and sulfate (SO_4^{2-}) than with their respective personal gas exposures. Only the results obtained in Boston showed an occasional association between ambient $PM_{2.5}$ and personal O_3 and NO_2. Although the strength of these crosspollutant associations was not as substantial as between ambient and personal $PM_{2.5}$, these findings suggest that at times, ambient pollutant gases can also serve as surrogates for personal exposures to $PM_{2.5}$. In addition, these studies showed that between-subject differences might exist in the strength of the personal-ambient association for gases, probably because of differences in house characteristics and ventilation conditions.[9,11,12] Whereas in Boston personal-ambient gaseous correlations were moderately strong, in Baltimore ambient gas concentrations were not associated with their respective personal exposures. This implies that changes over time for some gaseous pollutants (e.g., O_3) measured at central sites only reflect corresponding changes in personal exposures at some locations.

In this study, we expanded the work by Sarnat et al.[8] by including size-segregated PM data in our analysis. Associations between indoor, outdoor, and personal size-fractionated PM and OC, EC, particle number (PN), O_3, CO, NO, NO_x, and other important pollutants of both indoor and outdoor origin were evaluated, and the role of gaseous co-pollutants as surrogates of personal size-fractionated PM exposures was assessed. Data were collected at four retirement communities of the Los Angeles Basin during the Cardiovascular Health and Air Pollution Study (CHAPS), a multidisciplinary project with goals of investigating the effects of microenvironmental exposures to PM on cardiovascular outcomes in elderly retirees affected by coronary artery disease.[13]

METHODS

Study Design

The physical and chemical characteristics of indoor, outdoor, and personal quasi-UF, accumulation, and coarse PM were studied at four different retirement communities in southern California between 2005 and 2007. Three of these communities were in the San Gabriel Valley, CA (here referred to as site San Gabriel 1, San Gabriel 2, and San Gabriel 3) and the fourth in Riverside, CA. Site San Gabriel 1 was located approximately 50 km east of downtown Los Angeles, in a residential area, approximately 3 km away from any major freeways and close to a construction site. Site San Gabriel 2 was situated approximately 8 km east of Los Angeles, approximately 300 m south of a major freeway (see Polidori et al.,[11] for details). Site San Gabriel 3 was approximately 55 km east of downtown Los Angeles, 2.5 km away from 2 busy freeways and in close proximity (150 m) of a major street. The Riverside site was approximately 110 km east of Los Angeles and 15 km southeast of downtown Riverside. The closest freeway and a major street were approximately 3 and 1 km away, respectively, and downwind of the site. The abundant vegetation surrounding this last community may be a potential source of precursors of biogenically generated PM (e.g., organic particles from the photo-oxidation of terpenes).

Two 6-week sampling campaigns were conducted at each location. Phase 1 (P1) of each campaign was conducted during a warmer season (including summer and early fall), whereas phase 2 (P2) was conducted during a cooler season (including late fall and winter). Site San Gabriel 1 was operated from July 6, 2005 to August 20, 2005 (P1) and from October 19, 2005 to December 10, 2005 (P2); sampling at site San Gabriel 2 was conducted from August 24, 2005 to October 15, 2005 (P1) and from January 4, 2006 to February 18, 2006 (P2); site San Gabriel 3 was operated from July 5, 2006 to August 17, 2006 (P1) and from October 18, 2006 to December 1, 2006 (P2); sampling at site Riverside was conducted from August 23, 2006 to October 13, 2006 (P1) and from January 4, 2007 to February 16, 2007 (P2). Thus, we were able to study the seasonal variations in indoor, outdoor, and personal relationships between size-segregated PM and its components.

Two identical sampling stations were installed at each site, one indoors and one outdoors. The indoor sampling station at site San Gabriel 1 was located in a recreational area of the first community's main building, adjacent to a construction site where work was ongoing. The indoor sampling area at site San Gabriel 2 was situated in the dining room of the community's central building. The indoor station at site San Gabriel 3 was set up in a recreational area of the main retirement community complex, adjacent to a gym and an activity room. The indoor area at the Riverside site was located in the hallway of the main building with a dining room, activity room, and numerous apartment units nearby. At all sites the outdoor station (set up inside a movable trailer) was positioned within 300 m from the indoor station.

Personal PM concentrations were measured for 67 elderly retirees (18, 14, 17, and 18 subjects at sites San Gabriel 1, 2, 3, and Riverside, respectively) with a history of coronary artery disease. All subjects, except one, also participated in at least some part the epidemiologic part of this study, including ambulatory electrocardiogram and blood pressure monitoring. All participants were 71 yr of age or older, nonsmokers, and with no home exposure to environmental tobacco smoke (ETS). Each subject was followed for two 5-day sampling periods during the two phases of the study. Throughout this time, size-fractionated PM levels were measured daily. The number of subjects and observations of measured pollutants

Table 1. Estimated AER and F_{inf} over the studied sites and phases of the study.

Phase	Site	AER, hr^{-1} (mean ± SD)	F_{inf} OC	EC	PN	$PM_{2.5}$
P1 (summer and fall)	San Gabriel 1	0.25 ± 0.04	0.98	0.82	0.66	0.52
	San Gabriel 2	0.28 ± 0.06	0.74	0.74	0.46	0.45
	San Gabriel 3	0.40 ± 0.12	0.67	0.68	0.41	0.42
	Riverside	0.21 ± 0.06	0.60	0.72	0.57	0.49
P2 (fall and winter)	San Gabriel 1	0.33 ± 0.07	0.87	0.79	0.78	0.52
	San Gabriel 2	0.31 ± 0.10	0.61	0.64	0.55	0.38
	San Gabriel 3	0.26 ± 0.08	0.81	0.75	0.43	0.57
	Riverside	0.31 ± 0.09	0.90	0.82	0.45	0.41

during different phases of the study is reported in supplemental data Table S1 (published at http://secure.awma.org/onlinelibrary/samples/10.3155-1047-3289.59.4.392_supplmaterial.pdf). A more detailed description of the study design can be found in Delfino et al.[13]

Instrumentation

Continuous (1-min) PN concentrations were measured using water-based condensation particle counters (CPC; Model 3785, TSI, Inc.) at both indoor and outdoor sampling stations. Indoor and outdoor particulate OC and EC were measured in hourly cycles (i.e., sampling time = 45 min, analysis time = 15 min) by means of two semicontinuous OC-EC analyzers (Model 3F, Sunset Laboratory, Inc.). A beta attenuation monitor (BAM; Model 1020, Met One Instruments, Inc.) measured hourly $PM_{2.5}$ mass concentrations. Continuous (1 min) NO and NO_2 measurements were obtained both indoors and outdoors by using Thermo Environmental NO_x analyzers (Model 42, Thermo Environmental Instruments, Inc.). Dasibi CO analyzers (Model 3008, Dasibi Environmental Corp.) were implemented to measure continuous (1 min) indoor and outdoor CO levels. Continuous (1 min) outdoor O_3 concentrations were also monitored at each sampling site by using API O_3 analyzers (Model 400A, Teledyne Technologies, Inc.). For more details about the continuous/semicontinuous instruments used during the sampling campaign, see Polidori et al.[11]

In addition, integrated (24 hr) size-segregated indoor and outdoor particle samples were collected at all sites by means of Sioutas Personal Cascade impactors (SKC Inc.)[14,15] from Monday to Friday. Coarse-, accumulation-, and quasi-UF-mode PM were sampled on Zefluor filters (3-μm pore size; Pall Life Sciences) and analyzed gravimetrically using a microbalance (Mettler-Toledo; weight uncertainty ± 2 μg). Personal environmental monitors (PEMs) were deployed concurrently with the indoor and outdoor PM samplers to obtain integrated (24 hr) personal PM exposure data. Each PEM consists of an inlet, a Sioutas impactor, and a Leland Legacy pump (SKC, Inc.) operating at 9 L/min, all enclosed in a personal carry-on bag, then assigned to each subject.

Data Analyses

To match all continuous (or semicontinuous) measurements to the corresponding filter-based data, only daily averages of the concurrently measured $PM_{2.5}$, OC, EC, PN, NO_x, CO, and O_3 concentrations were considered. The contributions to outdoor OC by primary OC (OC_{pri}; emitted directly from combustion sources such as vehicular exhaust and wood smoke) and secondary organic aerosol (SOA; formed from semi- and low-volatility products of chemical reactions involving reactive organic gases) were estimated from measured OC and EC concentrations using EC as a tracer of primary combustion-generated OC (i.e., the "elemental carbon tracer method," see Polidori et al.[11]). Because one of the aims of this study is to evaluate the effects of outdoor air pollutants on indoor and personal exposure, we estimated air exchange rates (AERs; from CO measurements during periods affected by a dominant indoor source, see Abt et al.[16]) and infiltration factors (F_{inf}; defined as the equilibrium fraction of the outdoor species of interest that penetrates indoors and remains suspended, see Long et al.[17]) at each site.

If we assume an exponential decay of particles, that AER (hr^{-1}) and outdoor concentrations are constant during the decay period, that CO is conservative, and that indoor concentrations are well mixed, then:

$$C_t = e^{-(\mathrm{AER})t}\, C_0 \tag{1}$$

where C_t is the indoor CO concentration after time t (after the decay period) and C_0 is the initial peak CO concentration (right after CO emission). F_{inf} is described by the following equation:

$$F_{inf} = P(\mathrm{AER})/(\mathrm{AER} + k) \tag{2}$$

where P is the penetration coefficient (dimensionless). F_{inf} for OC, EC, $PM_{2.5}$, and PN were calculated using the recursive model (RM) developed by Allen et al.[18] In this method, for a particular species of interest, the average indoor concentration during an hour is equal to the sum of a fraction of the average outdoor concentration during the same hour, a fraction of the average indoor concentration remaining from the previous hour, and the contribution from indoor sources. More details on the F_{inf} and AER estimation methods are described in Polidori et al.[11] The average AERs calculated during CHAPS at the four retirement communities ranged from 0.21 to 0.4 hr^{-1} (Table 1). The generally low estimated AERs are consistent with the structural characteristics of the sampling

sites, the low number of open windows and doors, and the presence of central air conditioners. The average F_{inf} results were highest for EC (0.64–0.82) and OC (0.60–0.98) compared with those of $PM_{2.5}$ (0.38–0.57) and PN (0.41–0.78). In general, the F_{inf} results were similar across P1 (summer and fall) and P2 (fall and winter), which is consistent with no seasonal changes in home dynamics and ventilation conditions as indicated by the rather constant AERs calculated throughout the study. Estimated F_{inf} and measured particle concentrations across all hours were then used in a single compartment mass balance model to assess the contributions of indoor and outdoor sources to measured indoor EC, OC_{pri}, SOA, and PN.[11,19,20] The average amounts of indoor- and outdoor-generated PM and PM species (here denoted with "ig" and "og" subscripts, respectively) inside the studied homes were determined by this approach.

The following four associations (models) were then used to study the relationships between pollutant concentrations and size-segregated PM levels:

(1) Associations between outdoor co-pollutant concentrations and outdoor size-fractionated PM levels ("outdoor-outdoor associations").
(2) Associations between indoor co-pollutant concentrations and indoor size-fractionated PM levels ("indoor-indoor associations")
(3) Associations between outdoor particle and gaseous species concentrations and personal exposure to size-fractionated PM ("outdoor-personal associations").
(4) Associations between indoor particle and gaseous species concentrations and personal exposures to size-fractionated PM ("indoor-personal associations").

Linear mixed-effects regression parameters (slopes, or S) and Spearman's correlation coefficients (R) were calculated for the above-mentioned correlations.

Mixed Models

Data were analyzed with linear mixed-effect models, which expand the capabilities of linear regression by accounting for the correlation present in repeated measures data. In matrix notation, the mixed-effects model can be expressed as:

$$Y = X\beta + Z\upsilon + \varepsilon \quad (3)$$

where Y is a vector of outcomes, X is a known matrix of covariates, β is a vector of fixed-effect parameters, and ε is a vector of normally distributed errors. These terms parallel the standard linear regression model. To account for correlation within subjects, the linear mixed-effects model includes an additional covariate matrix Z and a vector of subject-specific random effects, υ.[21] In our models, random intercepts were estimated for each subject, and an autoregressive covariance structure was selected based on best fit from Akaike's information criteria. Residual and influence diagnostics were used to identify potentially outlying and influential observations. The interquartile range (IQR) of the considered independent variable standardized all regression parameter estimates to allow for a fair comparison of the S values. Thus, the mixed model coefficients for the dependent variable correspond to an IQR change in the independent variable.

In addition to linear mixed-effects models, R values were reported as an additional indicator of the association strength among indoor, outdoor, and personal data for the studied associations. Subjects having fewer than 5 valid days of observations in any of the size fractions (out of 10 per size fraction) were excluded from the analysis. Before regression and correlation analyses, outliers were detected at 3 standard deviations beyond the mean and individually inspected for influence in regression models.

All data collected at sites San Gabriel 1–3 were combined and considered to be representative of the entire San Gabriel Valley and compared with the measurements generated at the Riverside site. This division was based on the observation that diurnal and seasonal patterns of indoor and outdoor PM pollutants in Riverside (in particular OC, OC_{pri}, and SOA) were distinctly different from those recorded at the other three sites. The San Gabriel Valley sites are closer to downtown Los Angeles, are impacted to a higher degree by fresh traffic emissions, and thus exemplify the characteristics of typical "source" sites. Conversely, site Riverside is designated as a "receptor" location, where the aerosol is mostly comprised of advected, aged, and photochemically processed particles from the central Los Angeles area as well as of some local emissions.[22]

Finally, seasonal differences in phase-average concentrations of all measured species were estimated at all sites and assessed using *P* values for product terms between phase and the predictor. These "phase interaction" terms allowed us to study possible temporal variations in the associations between the variables considered during P1 (summer and early fall) and P2 (late fall and winter). Similarly, regional interactions between the San Gabriel Valley and the Riverside sites were calculated to highlight possible spatial differences in the considered associations.

RESULTS

Data Overview

Mean personal and mean indoor PM mass concentrations were similar at all sites, during all phases, and for all size fractions (Table 2), probably because most subjects spent most of their time indoors. Mean outdoor PM levels were higher than the corresponding personal and indoor concentrations across all size modes (especially for accumulation and coarse-mode particles), sites, and phases of the study (Table 2). This suggests that the overall loss of outdoor particles during penetration through the building envelope was higher than the particle generation from indoor sources. $PM_{2.5}$ levels (Table 1) were calculated in two different ways: by combining the filter-based quasi-UF- and accumulation-mode PM gravimetric masses, and from continuous BAM measurements. The resulting $PM_{2.5}$ concentrations were always within 1.7 $\mu g/m^3$ across all sites and during all phases of the study. The estimated average indoor concentrations of outdoor origin of $PM_{2.5}$, OC, EC, and PN ($Fine_{og}$, OC_{og}, EC_{og}, and CPC_{og}, respectively) were higher than the corresponding mean indoor-generated levels ($Fine_{ig}$, OC_{ig}, Ec_{ig}, and CPC_{ig}, respectively), which confirms that outdoor sources

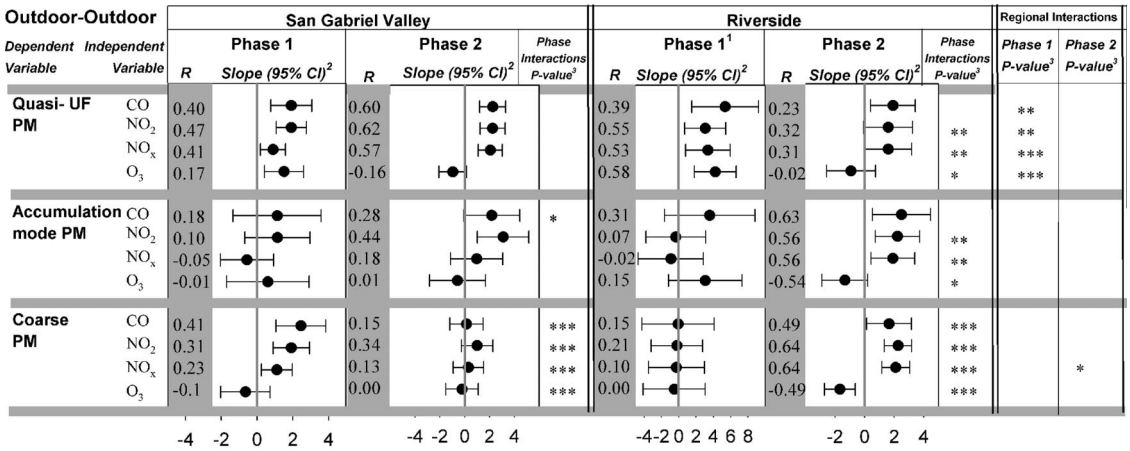
Table 2. Descriptive statistics for residential outdoor, indoor, and personal concentrations.

Pollutant	Outdoor/ Indoor/ Personal	San Gabriel Valley P1 Mean	SD	IQR1	P2 Mean	SD	IQR1	Riverside P1 Mean	SD	IQR1	P2 Mean	SD	IQR1
Gas concentration													
CO (ppm)[a]	Outdoor	0.59	0.23	0.37	0.69	0.31	0.36	0.31	0.13	0.25	0.12	0.11	0.17
	Indoor	0.64	0.23	0.29	0.78	0.35	0.37	0.46	0.11	0.16	0.60	0.11	0.17
NO_2 (ppb)	Outdoor	32.55	9.47	12.20	33.33	9.05	10.72	15.25	5.83	7.14	15.37	7.14	9.42
	Indoor	25.70	9.99	16.21	31.02	9.51	15.09	11.41	2.48	3.89	13.03	2.93	4.09
NO_x (ppb)	Outdoor	48.36	21.07	21.77	68.63	35.59	39.06	18.52	7.29	10.08	18.77	9.58	12.47
	Indoor	41.02	21.39	25.45	69.32	37.71	40.77	15.58	3.97	5.84	19.66	5.65	6.81
O_3 (ppb)	Outdoor	31.52	10.98	15.65	18.31	7.13	7.77	38.05	10.40	13.82	28.69	6.26	8.62
Particulate mass ($\mu g/m^3$)													
Quasi-UF PM	Personal	8.52	4.21	4.09	8.77	7.83	4.30	6.97	3.10	5.04	4.58	2.75	2.60
	Outdoor	9.87	3.02	4.13	9.95	4.40	5.46	11.42	4.92	7.06	7.29	3.54	6.49
	Indoor	8.93	2.86	3.77	8.89	3.66	4.62	8.99	3.26	4.24	6.05	2.95	2.47
Accumulation-mode PM	Personal	6.03	3.69	4.43	5.01	4.87	3.75	4.86	3.16	3.64	1.98	1.64	1.30
	Outdoor	13.30	6.05	8.01	10.94	9.23	10.39	9.75	6.34	8.65	5.07	3.81	4.61
	Indoor	7.31	3.41	4.42	6.28	4.72	5.14	4.55	2.36	1.49	2.21	1.68	1.17
Coarse PM	Personal	3.33	2.40	2.68	3.42	4.80	2.11	3.14	1.90	2.34	1.61	1.01	0.89
	Outdoor	11.48	3.61	4.35	8.58	5.21	5.89	12.52	6.95	7.95	4.62	2.55	3.15
	Indoor	2.62	2.91	3.16	4.14	5.49	3.93	2.88	2.64	1.94	2.85	1.40	1.54
$PM_{2.5}$[b]	Personal	14.54	6.60	7.87	13.77	10.93	8.20	11.77	5.50	8.13	6.50	3.66	3.53
	Outdoor	24.48	8.30	10.56	20.10	13.75	18.35	22.06	7.94	12.52	11.57	7.65	11.85
	Indoor	15.09	5.95	9.88	12.78	7.21	8.94	9.30	2.83	3.94	5.31	2.31	3.30
$Fine_{og}$	Indoor	12.73	6.02	10.43	9.95	7.54	8.86	7.50	2.52	4.56	3.79	2.17	3.20
$Fine_{ig}$	Indoor	3.42	2.10	1.81	4.85	3.39	5.24	3.39	1.21	1.31	2.53	1.46	1.12
PN (no./cm^3)													
PN	Outdoor	12,100	4,717	5,646	17,357	6,424	10,334	6,947	1,077	1,133	9,574	2,038	3,243
	Indoor	8,910	7,120	8,383	13866	7930	11,196	4,676	907	997	4,373	969	1,343
PN_{ig}	Indoor	3,021	4,112	2,386	4311	5,912	4,336	1,239	847	662	970	849	1,086
PN_{og}	Indoor	6,459	3,621	5757	9695	4,147	7,782	3,504	487	569	3,412	627	533
OC ($\mu g/m^3$)													
OC	Outdoor	6.24	1.90	2.60	7.70	3.55	6.94	10.50	1.14	1.31	15.39	1.74	2.90
	Indoor	5.62	1.42	1.70	6.78	3.12	5.72	8.46	1.68	2.47	14.27	1.95	3.11
OC_{ig}	Indoor	1.16	0.70	1.15	1.21	1.10	1.32	2.32	1.65	2.55	1.41	0.52	0.69
OC_{og}	Indoor	4.49	1.28	1.49	5.69	2.99	5.76	6.14	0.65	0.85	13.64	1.44	2.37
OC_{pri}	Outdoor	2.85	0.82	1.11	5.13	3.27	6.17	8.23	1.09	1.65	11.25	0.78	0.97
$OC_{pri(og)}$	Indoor	2.19	0.80	1.04	4.07	2.28	4.11	4.42	0.68	1.07	10.07	0.61	0.87
SOA	Outdoor	2.81	1.45	1.93	2.76	1.43	2.13	2.28	0.90	1.19	4.14	1.55	1.98
SOA_{og}	Indoor	2.38	1.23	1.53	2.37	1.22	1.83	1.82	0.72	1.05	3.56	1.33	1.70
EC ($\mu g/m^3$)													
EC	Outdoor	1.66	0.44	0.55	1.71	0.74	1.09	1.22	0.48	0.56	0.82	0.31	0.39
	Indoor	1.39	0.44	0.65	1.32	0.54	0.83	1.37	0.23	0.21	1.01	0.25	0.41
EC_{ig}	Indoor	0.31	0.23	0.25	0.25	0.20	0.15	0.48	0.26	0.35	0.34	0.10	0.14
EC_{og}	Indoor	1.08	0.30	0.42	1.10	0.49	0.73	0.89	0.29	0.37	0.65	0.24	0.31

Notes: [a]ppm = parts per million; [b]Calculated as the sum of quasi-UF and accumulation-mode PM integrated measurements (personal $PM_{2.5}$) and from continuous BAM data (indoor and outdoor $PM_{2.5}$).

were the most important contributors to the measured indoor pollutant concentrations.

The San Gabriel Valley sites were closer to freeways than the Riverside site (located in a desert region) and were impacted by higher levels of CO, NO_2, and NO_x, mainly emitted from primary combustion sources (e.g., motor-vehicle emissions). Conversely, OC and O_3 levels were generally higher in Riverside. The latter site was approximately 110 km east (and downwind) of downtown Los Angeles, with prevailing westerly winds blowing from the Pacific Ocean. The plume of pollutants generated in the Los Angeles area includes several reactive organic species that are likely to form OC through secondary processes (i.e., SOA formation) as the air mass ages and is transported eastwards. The higher average OC, O_3, and SOA levels and the smaller diurnal OC variation in Riverside (compared with the characteristic afternoon increase in OC, O_3, and SOA in the San Gabriel Valley) confirm that the latter is a typical receptor area, where the contribution of SOA to total measured OC is substantial. In addition, the vegetation surrounding the Riverside community is a potential source of biogenic gas-phase precursors, which form SOAs through photochemical reactions (e.g., photochemical oxidation of terpenes[23]).



1 Sclae is different for phase 1 at Riverside
2 Mixed model coefficients for the dependent variable correspond to an interquartile range change in the independent variable
3 * = p-value < 0.1 (sufficient evidence for a difference)
** = p-value <0.05
*** = p-value < 0.01 (significantly different)

Figure 1. Mixed model and Spearman correlation results for outdoor–outdoor associations.

Outdoor–Outdoor Associations

Outdoor quasi-UF PM was positively (and moderately) correlated with CO, NO_2, and NO_x at all locations and during all seasons, mainly because all of these species are emitted by the same combustion sources, and also because their atmospheric transport and removal is affected by similar meteorological processes[24,25] (Figure 1). Outdoor quasi-UF PM was positively correlated with O_3 only during P1 (warmer season), both in the San Gabriel Valley (r = 0.17; regression S = 1.5 $\mu g/m^3$ [95% confidence interval = 0.4–2.6]) and, especially, in Riverside (r = 0.58; S = 4.2 $\mu g/m^3$ [1.8–6.6]). For an interquartile increase in the average O_3 concentration measured in Riverside during P1, the corresponding quasi-UF PM increased by 4.2 $\mu g/m^3$, reflecting the important contribution of photochemical processes to the production of SOA. The same associations were negative and nonsignificant during P2 (cooler months) (r = −0.16; S = −1 $\mu g/m^3$ [−2.1 to 0.2] in the San Gabriel Valley; r = −0.02; S = −1 $\mu g/m^3$ [−2.6 to 0.7] in Riverside). On the basis of *P* values for product terms, the prediction of quasi-UF particles by gaseous co-pollutants was significantly different between P1 and P2 in Riverside, and between P1 in Riverside and P1 in the San Gabriel Valley (see the "Regional Interaction" column in Figure 1 for details, and note the scale difference for S values in P1 for Riverside).

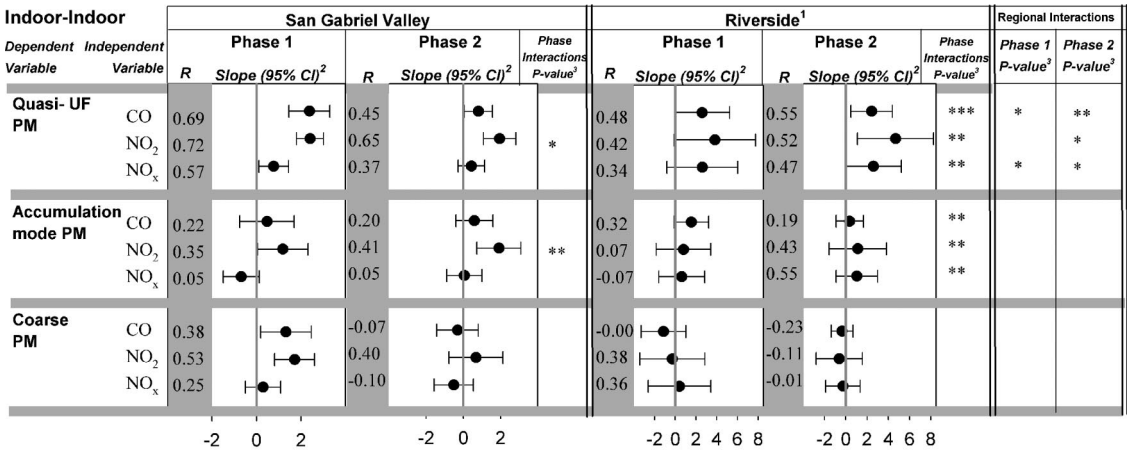
Accumulation-mode particles had generally smaller correlations and mixed model S values with CO, NO_2, and NO_x compared with quasi-UF PM, probably because their outdoor levels are influenced by a combination of primary and secondary sources. However, these associations were stronger in P2, when the production of accumulation-mode particles and their corresponding co-pollutants from primary combustion sources is typically the highest and the contribution of secondary formation mechanisms to the measured PM mass is decreased.

With the exception of P2 in the San Gabriel Valley and P1 in Riverside, positive and moderately strong correlations between coarse particles and CO, NO_2, and NO_x were observed at all sites. Although not consistent, this association is not surprising, because coarse PM is emitted from resuspended soil and road dust, and the latter mechanism shares the same anthropogenic sources responsible for the production of these gaseous co-pollutants. The product term models for phase showed a significant phase interaction at all sites ($P < 0.01$ = *** in Figure 1), indicative of the seasonal effect of primary sources on the associations between outdoor coarse PM and the corresponding gaseous co-pollutants. Interestingly, S values for the prediction of coarse particles by these gases were greater in P2 than P1 in Riverside. The opposite situation was observed in the San Gabriel Valley.

Results showing associations between outdoor fine particulate EC and OC and associations between PN and outdoor gases are presented in the online supplemental information (Table S2). Strong and positive associations between outdoor EC and outdoor CO, NO_2, and NO_x were found both in the San Gabriel Valley and in Riverside (average r = 0.45–0.91 and average S = 0.4–0.8 $\mu g/m^3$). Similarly, PN and OC concentrations were well correlated with primary gaseous co-pollutant levels measured at all sites and during all phases. EC consists of graphite-like material emitted from the incomplete combustion of organic fuels.[26,27] Combustion sources are also the major contributor to the ambient PN and OC concentrations, although a substantial amount of OC is also formed from secondary processes. A significant phase interaction for PN was observed at the San Gabriel Valley sites, but not in Riverside. PN concentrations were both higher and more strongly associated with CO, NO_2, and NO_x during the period of air stagnation (P2). This finding highlights the seasonal effect of primary sources on the associations between PN and primary gaseous co-pollutants.

Indoor–Indoor Associations

Indoor quasi-UF particles were strongly and positively associated with indoor gaseous co-pollutants at all sites and during all phases (R values ranged from 0.34 to 0.72



1 Sclae is different for Riverside graph
2 Mixed model coefficients for the dependent variable correspond to an interquartile range change in the independent variable
3 * = p-value < 0.1 (sufficient evidence for a difference)
** = p-value <0.05
*** = p-value < 0.01 (significantly different)

Figure 2. Mixed model and Spearman correlation results for indoor–indoor associations.

for this size fraction) (Figure 2). The strength and regression S values of these correlations are similar to the corresponding outdoor-outdoor associations (Figure 1).

Accumulation-mode particles were more weakly associated with gaseous co-pollutants than quasi-UF PM, probably because this size fraction contains a higher percentage by mass of SOA that might condense on existing indoor particles, or volatilize in the indoor environment (depending on the vapor pressure of the considered organic species and/or the home characteristics). It is well known that changes in the physical and chemical properties of $PM_{2.5}$ occur as they penetrate indoors from outdoors. For example, ammonium nitrate (NH_4NO_3), which accounts for 35–50% of the outdoor $PM_{2.5}$ mass in the Los Angeles Basin,[22,28,29] volatilizes upon building entry and results in altering the PM size and chemical composition indoors.

In most cases, indoor coarse PM was not correlated with indoor gaseous co-pollutants because, unlike CO, NO_2, and NO_x, the coarse fraction is characterized by very low infiltration indoors (F_{inf}) and may also be emitted from resuspension of existing particles deposited on indoor surfaces and floors.[3] The product term analysis showed a significant phase interaction at the Riverside site for indoor quasi-UF and accumulation-mode PM, implying a seasonal variability in the associations between these two fractions and the corresponding indoor gaseous co-pollutants (Figure 2).

Results showing associations between outdoor fine particulate EC and OC and associations of PN with outdoor gases are presented in the online supplemental information (Table S3). Indoor OC was weakly associated with indoor gases, except during P1 (warmer months) at the San Gabriel Valley site, where strong positive associations (r = 0.69–0.81; average regression S = 0.6–1.4 $\mu g/m^3$) were found. These overall weak associations could be explained by changes in the OC characteristics as it penetrates indoors from outdoors, or by the presence of indoor OC sources, such as cooking, particularly for the retirement community here referred to as group 2 (see Polidori et al.,[11] for details). A significant phase interaction between indoor EC and indoor gaseous co-pollutants was observed at all locations, similar to the findings for outdoor-outdoor associations. This indoor correlation may be because EC, CO, NO_2, and NO_x all share the same primary combustion sources coupled with the size range of EC particles (0.1–0.3 μm)[30] that allows them to penetrate indoors with great efficiency.[31] Moreover, EC is formed indoors in negligible amounts.[11,32,33]

In Riverside, correlations between indoor PN and indoor gaseous species were weaker than the correspondent outdoor-outdoor associations, probably because of the presence of indoor submicrometer PN particles from fan heaters (particularly during P2), which tend to increase PN concentrations but not the overall PM mass level.[34]

Outdoor–Personal Associations

At all sites, correlations of personal quasi-UF particles with outdoor quasi-UF PM (Figure 3a) were weaker compared with corresponding correlations for accumulation-mode particles (discussed below). Weaker quasi-UF correlations are consistent with the relatively low penetration of <100-nm particles indoors because of diffusional losses[31,35] as well as losses because of evaporation of volatile species associated with this size range.[36]

This is further confirmed by the relatively small average regression S values for the personal-outdoor associations (S = 0.1 to 1.4 $\mu g/m^3$). Personal quasi-UF particles were not well correlated with either the larger outdoor PM fractions, or OC and most of its components (e.g., SOA and OC_{pri}), but were positively associated with EC and tracers of primary combustion (CO and NO_x). The low correlations (R) between personal quasi-UF PM and OC_{pri} might be because OC_{pri} includes components with a relatively high vapor pressure that volatilize upon building entry. Most importantly, these data suggest that personal exposures to quasi-UF PM were significantly associated with EC at all CHAPS sites except during P2 in Riverside. This has important implications for the potential health effects of personal PM because EC is a good surrogate for

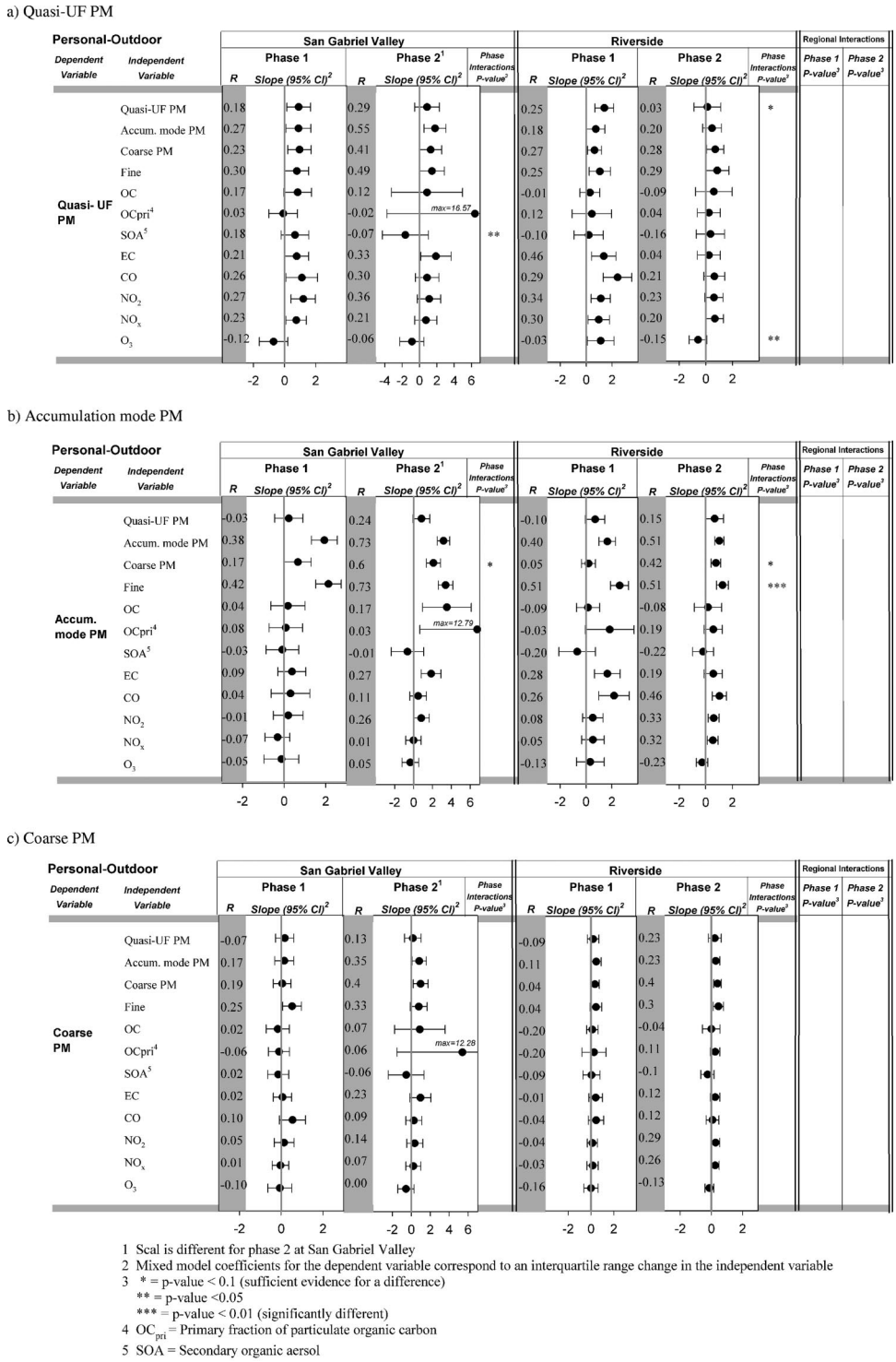


Figure 3. Mixed model and Spearman correlation results for outdoor pollutants with (a) personal quasi-UF PM, (b) personal accumulation-mode PM, and (c) coarse PM.

diesel particles and a well-known carcinogenic compound class.[37]

Outdoor accumulation-mode PM (as well as $PM_{2.5}$) were well correlated with personal accumulation-mode particles (R values ranged between 0.38 and 0.73), and the magnitude of the regression parameter estimates was also relatively high. In particular, an interquartile increase in outdoor accumulation-mode PM was associated with increases in personal accumulation-mode particles of 1.9 and 3.1 $\mu g/m^3$ (for P1 and P2, respectively) in the San Gabriel Valley, and of 1.6 and 1 $\mu g/m^3$ (for P1 and P2, respectively) in Riverside (see Figure 3b for details). This size fraction is not as easily lost by diffusion/coagulation as quasi-UF PM when entering indoors, or by inertial

deposition mechanisms (like coarse particles) and, hence, has a high penetration efficiency. Outdoor OC and its subfractions were generally not well associated with personal accumulation-mode particles. However, in the cooler seasons, stronger correlations (and generally higher regression S values) were found with EC and other combustion products, such as CO, NO_2, and NO_x. On the basis of product term models, a significant phase interaction ($p < 0.01$) was found at the Riverside site for the prediction of personal accumulation-mode particles by outdoor $PM_{2.5}$, highlighting the seasonal effect of primary sources on the associations between these two PM size fractions.

Overall, personal coarse-mode particles were not well correlated with any of the outdoor PM fractions/components or outdoor gases, suggesting that none of these species was a good surrogate for exposure to personal coarse PM. This is because of the lower F_{inf} values for larger particle sizes, coupled with the fact that indoor coarse particles are mostly generated by mechanical processes and/or resuspended from previously deposited particles on indoor surfaces. *P* values showed no significant phase or regional interactions for personal coarse particles at the San Gabriel Valley and Riverside communities (see the "Phase Interactions" and "Regional Interactions" columns in Figure 3c), which implies that factors affecting the associations between personal coarse PM and its outdoor gaseous and particle co-pollutants were similar at all sites and phases. Mixed model and Spearman correlation results for outdoor pollutants with (1) personal quasi-UF, (2) personal accumulation, and (3) personal coarse-mode PM are shown in the online supplemental information (Table S4).

Indoor–Personal Associations

Personal and indoor quasi-UF PM levels were best correlated in the San Gabriel Valley (r = 0.38; average regression S = 1.6 $\mu g/m^3$ [0.8–2.3] in the warmer season; r = 0.40; S = 2.3 $\mu g/m^3$ [0.7–4] in the cooler months) than in Riverside (Figure 4a). At the San Gabriel Valley sites, moderate correlations and significant regression parameter estimates were also observed between personal quasi-UF particles and $PM_{2.5}$. Associations between personal quasi-UF particles and indoor OC (and its subcomponents) varied with location (from strong and positive in the San Gabriel Valley, to poor and negative in Riverside). Although there are different possible mechanisms leading to the production of particulate OC (e.g., photochemical reactions, condensation of organic vapors on existing particles, and primary emission from combustion sources), organic particles in the quasi-UF mode are generally formed by combustion processes,[30,38] which are more dominant in the San Gabriel Valley than in Riverside. Indoor EC and outdoor-generated EC found indoors (EC_{og}) were better associated with quasi-UF personal PM than indoor-generated EC (EC_{ig}), indicating that indoor sources of EC were not substantial. For each interquartile increase in EC_{og}, the personal quasi-UF PM concentration increased 0.9 and 2.4 $\mu g/m^3$ (during P1 and P2, respectively) in the San Gabriel Valley, and 1.6 and 0.2 $\mu g/m^3$ (during P1 and P2, respectively) in Riverside (see Figure 4a for details). Similarly, $Fine_{og}$, OC_{og}, and $OC_{pri(og)}$ (see Figure 4a) were generally strongly and positively correlated with personal quasi-UF PM compared with $Fine_{ig}$ and OC_{ig}; $OC_{pri(ig)}$, estimates were not reported). This observation suggests that indoor sources were probably not significant contributors to personal exposure, which is predominantly influenced by primary emitted pollutants produced/emitted outdoors. Indoor gaseous co-pollutants (most likely originating outdoors) were more positively correlated to quasi-UF particles (r = 0.21–0.34; average regression S = 0.4–2.3 $\mu g/m^3$) than larger size fractions (discussed below), likely because of similarities in sources, transportation, and deposition mechanisms for these two classes of pollutants. Positive and fairly good correlation of indoor (and outdoor) concentrations of CO, NO_2, and NO_x with personal quasi-UF PM levels reflects the possibility that these gaseous co-pollutants could confound epidemiologic associations between quasi-UF particles and adverse health effects. However, this seems questionable because CO is neither a respiratory irritant nor a moderator of immune response in the respiratory tract,[9] and NO_2 is probably not responsible for adverse health effects given its low concentrations in personal and ambient air.[13,39] Thus, we speculate that the observed association between personal quasi-UF PM concentrations and measured indoor co-pollutant levels do not reflect the corresponding personal-personal relationships (personal gaseous measurements were not performed during CHAPS).

At all sites and during all seasons, personal accumulation-mode particles were well associated with indoor accumulation PM and with $PM_{2.5}$ (Figure 4b). Moreover, personal accumulation-mode PM was more strongly correlated with $Fine_{og}$, OC_{og} (only at the San Gabriel sites), and EC_{og} than with the corresponding indoor levels of indoor origin ($Fine_{ig}$, OC_{ig}, and EC_{ig}, respectively). In contrast, personal accumulation-mode PM was always negatively associated to SOA_{og}. These results reinforce the idea that outdoor primary emission sources are of great importance in terms of personal exposure to $PM_{2.5}$. These findings support the epidemiologic results reported by Delfino et al.[13] for the present panel of elderly people with a history of coronary artery disease. They concluded that the strongest associations were found between indoor PM of outdoor origin and increases in blood biomarkers of systemic inflammation and platelet activation, and decreases in erythrocyte antioxidant activity. In the same study, little evidence for biomarker associations with secondary $PM_{2.5}$, SOA, or total OC was found, whereas robust associations with emission sources of primary $PM_{2.5}$, OC, and quasi-UF particles were observed. This is consistent with the weak and often negative correlations between personal quasi-UF and accumulation-mode PM and indoor SOA of outdoor origin (SOA_{og}) obtained in our work.

Indoor CO, NO_2, and NO_x levels were not as well correlated to personal accumulation PM concentrations as quasi-UF-mode particles, except during P2 at Riverside (r = 0.37–0.45; see Figure 4b). Generally, indoor CO and NO_2 were better associated with personal accumulation PM exposure than indoor NO_x. Overall, this suggests that the measured gaseous co-pollutants are weak surrogates of personal exposure to accumulation-mode PM, at least for

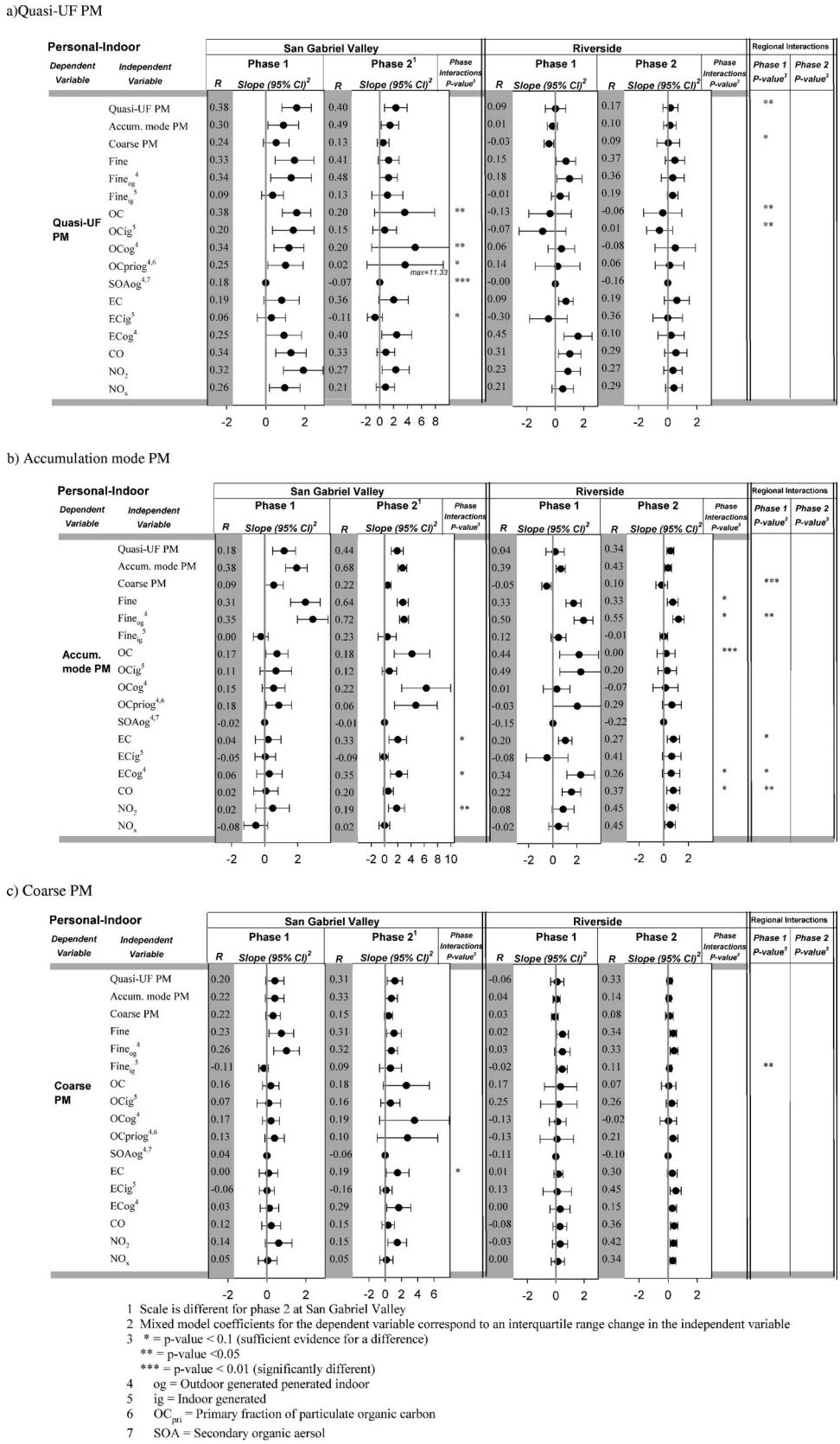


Figure 4. Mixed model and Spearman correlation results for indoor pollutants with (a) personal quasi-UF PM, (b) personal accumulation-mode PM, and (c) coarse PM.

subjects with similar exposure profiles and living in similar urban locations. It is worth emphasizing that, although quasi-UF PM is mostly produced from primary combustion sources, accumulation-mode particles originate from a combination of primary and secondary emissions. This might explain the relatively weaker correlations observed earlier between personal accumulation PM and outdoor CO (and EC, a tracer of primary combustion). For NO_2 (a rather nonreactive gas), indoor sources may also weaken its association with personal PM.

Personal coarse-mode particles were more strongly associated with all indoor PM fractions in the San Gabriel Valley than in Riverside, although the strength of this correlation was rather low at all sites (Figure 4c). Associations between personal coarse PM and indoor EC (as well as indoor OC) were generally small. Indoor CO, NO_2, and NO_x showed relatively poor associations with personal coarse PM at all sites, except in Riverside during the cooler months (r = 0.34–0.42; average regression S = 0.3–0.4 $\mu g/m^3$). Mixed model and Spearman correlation results for indoor pollutants with (1) personal quasi-UF, (2) personal accumulation, and (3) personal coarse-mode PM are shown in the supplemental data (Table S5).

Regional and Seasonal Correlations and Comparison with Other Studies

To the best of our knowledge, this study is one of the most extensive analyses to date of seasonal correlations between indoor, outdoor, and personal size-segregated PM concentrations and the corresponding gaseous levels/PM components. Despite the uniqueness of this data analysis, we considered it important to compare our results to those obtained from similar studies conducted previously in the United States in an effort to clarify the role of CO, NO_2, NO_x, and O_3 as surrogates of human exposure to $PM_{2.5}$. Figure 5 shows R values for the associations between personal $PM_{2.5}$ levels and the outdoor (residential or ambient) concentrations of $PM_{2.5}$, CO, NO_2, NO_x, and O_3 in the warmer (Figure 5a) and cooler (Figure 5b) months. R values calculated at the San Gabriel Valley and Riverside sites (personal vs. residential outdoor) were plotted against those obtained by Sarnat et al. in Baltimore[9] and Boston[8] (personal vs. ambient).

At all studied locations, personal $PM_{2.5}$ was well correlated with outdoor/ambient $PM_{2.5}$ in the warmer and cooler seasons. This is also consistent with results from other recent longitudinal studies on PM exposure.[8,9,40,41] Of particular interest are the moderately strong associations observed at all sites between personal $PM_{2.5}$ and NO_2 (in both phases; see Figure 5, a and b), personal $PM_{2.5}$ and O_3 (only in P1; Figure 5a), and $PM_{2.5}$ and CO (only in P2; Figure 5b). Variability in these correlations reflects the effects of seasonal changes on the formation mechanisms of PM.

In the warmer months, a considerable fraction of personal exposure to $PM_{2.5}$ might be related to particles originating from photochemical activities, whereas in the cooler months personal $PM_{2.5}$ levels are predominantly influenced by combustion processes. In addition, results from all studies indicate that outdoor/ambient NO_2 and O_3 (in the warmer season) and

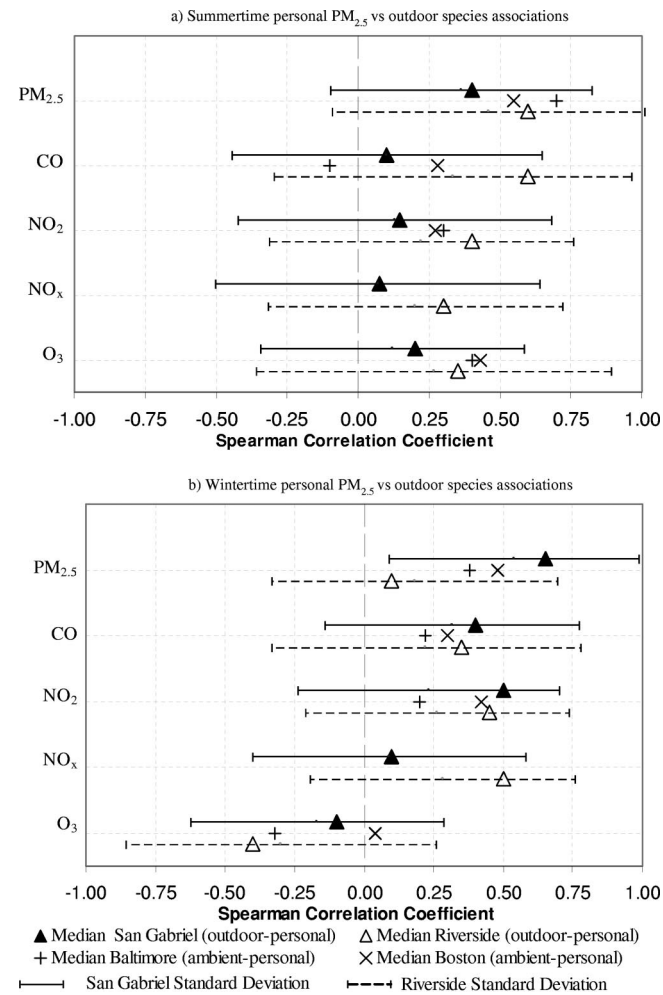


Figure 5. R values for the associations between personal $PM_{2.5}$ concentrations and outdoor (residential or ambient) particle/gaseous levels in (a) summer and (b) winter. The R values calculated in this work at the San Gabriel Valley and Riverside sites (personal vs. residential outdoor) were compared with those obtained by Sarnat et al. in Baltimore[9] and Boston[8] (personal vs. ambient). Error bars represent standard deviation of individual values.

outdoor/ambient CO and NO_2 (in the cooler season) are moderately correlated with personal $PM_{2.5}$ exposure, and in each location there may be differences in the strength of the personal-ambient associations, probably because of geographical variability in housing characteristics and ventilation conditions.[8,9,11]

Therefore, it might be incorrect to assume that outdoor/ambient gas measurements are consistent surrogates for personal $PM_{2.5}$ exposures, because correlations between personal $PM_{2.5}$ and outdoor/ambient CO, NO_2, NO_x, and O_3 vary by both season and location (Figures 5a and 5b). Results from time-series epidemiologic studies that include both gaseous and particulate pollutant concentrations in the models should be interpreted with caution. As noted by Sarnat et al.,[9] if ambient co-pollutant concentrations are surrogates (as opposed to confounders) of PM, using multiple pollutant models in epidemiologic studies of time-integrated (or size-fractionated) particles would lead to an erroneous

significant association for the collinear co-pollutants. Previous environmental and occupational studies showed that O_3 and NO_2 exposures may elicit adverse health effects,[42–46] thus having the potential to act as confounders of personal exposure to $PM_{2.5}$. However, two recent studies by Delfino et al.[13,39] have suggested that NO_2 might be a surrogate of not only particle species, but also volatile and semi-volatile compounds that may have important health effects independent of particle-bound components.

CONCLUSIONS

Our modeling results have shown that outdoor and indoor levels of CO, NO_2, and NO_x were better correlated with measured indoor, outdoor, and personal quasi-UF PM levels than accumulation- and coarse-mode PM. This better correlation is due to more similarity in sources and transportation mechanism. EC_{og}, OC_{og}, and $OC_{pri(og)}$ were more strongly correlated with personal quasi-UF and accumulation-mode PM than EC_{og}, OC_{ig}, and $OC_{pri(ig)}$. This is because indoor sources were probably not significant contributors to personal exposure of accumulation and quasi-UF PM, which is predominantly influenced by primary pollutants produced/emitted outdoors. These results are important, because other CHAPS investigators have suggested that traffic-related emission sources of $PM_{2.5}$, OC_{pri}, and quasi-UF particles lead to increases in systemic inflammation, platelet activation, and decreases in erythrocyte antioxidant activity in elderly people with a history of coronary artery disease.

Overall, our data analysis suggests that investigating the correlations among size-segregated indoor, outdoor, and personal PM; their specific components; and concurrently measured gaseous co-pollutants is a challenging endeavor. These associations depend on several factors that vary in space and time, such as the relative contribution of UF-, accumulation-, and coarse-mode PM to the measured PM concentrations; the seasonal variability of primary and secondary emission sources; the presence of indoor sources of PM and gaseous co-pollutants (e.g., cooking); home characteristics (e.g., ventilation conditions and household characteristics); and proximity to the emission sources. The analysis of these associations is further complicated by the amount of time spent indoors (highly variable among subjects, especially in the warmer season), which is also a critical component in determining exposure. Thus, results from time-series epidemiologic studies that include both gaseous and particulate pollutant concentrations in the models should be interpreted with caution. Future research should focus on how these specific factors affect the strength of between-pollutant associations for individuals living in different locations.

ACKNOWLEDGMENTS

This project was supported by grant ES12243 from the National Institute of Environmental Health Sciences, National Institutes of Health; the California Air Resources Board contract 03-329; and U.S. Environmental Protection Agency grant RD83241301. The contents are solely the responsibility of the authors and do not necessarily represent the official views of the funding agencies. The authors declare they have no competing financial interests.

REFERENCES

1. Pope, C.A.; Dockery, D.W. Health Effects of Fine Particulate Air Pollution: Lines that Connect; *J. Air & Waste Manage. Assoc.* **2006**, *56,* 709-742.
2. McClellan, R.O. Setting Ambient Air Quality Standards for Particulate Matter; *Toxicology* **2002**, *181,* 329-347.
3. *Air Quality Criteria for Particulate Matter*; U.S. Environmental Protection Agency: Research Triangle Park, NC, 2004.
4. Ntziachristos, L.; Ning, Z.; Geller, M.D.; Sheesley, R.J.; Schauer, J.J.; Sioutas, C. Fine, Ultrafine and Nanoparticle Trace Element Compositions near a Major Freeway with a High Heavy-Duty Diesel Fraction; *Atmos. Environ.* **2007**, *41,* 5684-5696.
5. Kaiser, J. Mounting Evidence Indicts Fine-Particle Pollution; *Science* **2005**, *307,* 1858-1861.
6. Nel, A. Air Pollution-Related Illness: Effects of Particles; *Science* **2005**, *308,* 804-806.
7. Green, L.C.; Crouch, E.A.C.; Ames, M.R.; Lash, T.L. What's Wrong with the National Ambient Air Quality Standard (NAAQS) for Fine Particulate Matter ($PM_{2.5}$)? *Regul. Toxicol. Pharmacol.* **2002**, *35,* 327-337.
8. Sarnat, J.A.; Brown, K.W.; Schwartz, J.; Coull, B.A.; Koutrakis, P. Ambient Gas Concentrations and Personal Particulate Matter Exposures—Implications for Studying the Health Effects of Particles; *Epidemiol.* **2005**, *16,* 385-395.
9. Sarnat, J.A.; Koutrakis, P.; Suh, H.H. Assessing the Relationship between Personal Particulate and Gaseous Exposures of Senior Citizens Living in Baltimore, MD; *J. Air & Waste Manage. Assoc.* **2000**, *50,* 1184-1198.
10. *Research Priorities for Airborne Particulate Matter. Immediate Priorities and a Long-Range Research Portfolio*; National Research Council: Washington, DC, 1998.
11. Polidori, A.; Arhami, M.; Sioutas, C.; Delfino, R.J.; Allen, R. Indoor/Outdoor Relationships, Trends, and Carbonaceous Content of Fine Particulate Matter in Retirement Homes of the Los Angeles Basin; *J. Air & Waste Manage. Assoc.* **2007**, *57,* 366-379.
12. Rojas-Bracho, L.; Suh, H.H.; Koutrakis, P. Relationships among Personal, Indoor, and Outdoor Fine and Coarse Particle Concentrations for Individuals with COPD; *J. Expo. Anal. Environ. Epidemiol.* **2000**, *10,* 294-306.
13. Delfino, R.J.; Staimer, N.; Tjoa, T.; Polidori, A.; Arhami, M.; Gillen, D.L.; Kleinman, M.T.; Vaziri, N.D.; Longhurst, J.; Zaldivar, F.; Siouta, S.C. Circulating Biomarkers of Inflammation, Antioxidant Activity, and Platelet Activation are Associated with Primary Combustion Aerosols in Subjects with Coronary Artery Disease; *Environ. Health Perspect.* **2008**, *116,* 898-906.
14. Misra, C.; Singh, M.; Shen, S.; Sioutas, C.; Hall, P.A. Development and Evaluation of a Personal Cascade Impactor Sampler (PCIS); *J. Aerosol Sci.* **2002**, *33,* 1027-1047.
15. Singh, M.; Misra, C.; Sioutas, C. Field Evaluation of a Personal Cascade Impactor Sampler (PCIS); *Atmos. Environ.* **2003**, *37,* 4781-4793.
16. Abt, E.; Suh, H.H.; Catalano, P.J.; Koutrakis, P. Relative Contribution of Outdoor and Indoor Particle Sources to Indoor Concentrations; *Environ. Sci. Technol.* **2000**, *34,* 3579-3587.
17. Long, C.; Suh, H.; Catalano, P.; Koutrakis, P. Using Time- and Size-Resolved Particulate Data to Quantify Indoor Penetration and Deposition Behavior; *Environ. Sci. Technol.* **2001**, *35,* 2089-2099.
18. Allen, R.; Larson, T.; Sheppard, L.; Wallace, L.; Liu, L.-J.S. Use of Real-Time Light Scattering Data to Estimate the Contribution of Infiltrated and Indoor-Generated Particles to Indoor Air; *Environ. Sci. Technol.* **2003**, *37,* 3484-3492.
19. Wallace, L. Indoor Particles: a Review; *J. Air & Waste Manage. Assoc.* **1996**, *46,* 98-126.
20. Meng, Q.Y.; Turpin, B.J.; Korn, L.; Weisel, C.P.; Morandi, M.; Colome, S.; Zhang, J.F.J.; Stock, T.; Spektor, D.; Winer, A.; Zhang, L.; Lee, J.H.; Giovanetti, R.; Cui, W.; Kwon, J.; Alimokhtari, S.; Shendell, D.; Jones, J.; Farrar, C.; Maberti, S. Influence of Ambient (Outdoor) Sources on Residential Indoor and Personal $PM_{2.5}$ Concentrations: Analyses of RIOPA Data; *J. Expo. Anal. Environ. Epidemiol.* **2005**, *15,* 17-28.
21. Laird, N.M.; Ware, J.H. Random-Effects Models for Longitudinal Data; *Biometrics* **1982**, *38,* 963-974.
22. Sardar, S.B.; Fine, P.M.; Sioutas, C. Seasonal and Spatial Variability of the Size-Resolved Chemical Composition of Particulate Matter (PM_{10}) in the Los Angeles Basin; *J. Geophys. Res. Atmos.* **2005**, *110,* D07SO8; doi: 10.1029/2004JD004627.
23. Kanakidou, M.; Seinfeld, J.H.; Pandis, S.N.; Barnes, I.; Dentener, F.J.; Facchini, M.C.; Van Dingenen, R.; Ervens, B.; Nenes, A.; Nielsen, C.J.; Swietlicki, E.; Putaud, J.P.; Balkanski, Y.; Fuzzi, S.; Horth, J.; Moortgat, G.K.; Winterhalter, R.; Myhre, C.E.L.; Tsigaridis, K.; Vignati, E.; Stephanou, E.G.; Wilson, J. Organic Aerosol and Global Climate Modeling: a Review; *Atmos. Chem. Phys.* **2005**, *5,* 1053-1123.
24. Chen, C.; Chock, D.P.; Winkler, S.L. A Simulation Study of Confounding in Generalized Linear Models for Air Pollution Epidemiology; *Environ. Health Perspect.* **1999**, *107,* 217-222.

25. Wallace, L. Correlations of Personal Exposure to Particles with Outdoor Air Measurements: a Review of Recent Studies; *Aerosol Sci. Technol.* **2000**, *32,* 15-25.
26. Chow, J.C.; Watson, J.G.; Pritchett, L.C.; Pierson, W.R.; Frazier, C.A.; Purcell, R.G. The DRI Thermal Optical Reflectance Carbon Analysis System—Description, Evaluation and Applications in United-States Air-Quality Studies; *Atmos. Environ.* **1993**, *27,* 1185-1201.
27. Birch, M.E.; Cary, R.A. Elemental Carbon-Based Method for Monitoring Occupational Exposures to Particulate Diesel Exhaust; *Aerosol Sci. Technol.* **1996**, *25,* 221-241.
28. Christoforou, C.S.; Salmon, L.G.; Hannigan, M.P.; Solomon, P.A.; Cass, G.R. Trends in Fine Particle Concentration and Chemical Composition in Southern California; *J. Air & Waste Manage. Assoc.* **2000**, *50,* 43-53.
29. Kim, S.; Sioutas, C.; Chang, M.C.; Gong, H. Factors Affecting the Stability of the Performance of Ambient Fine-Particle Concentrators; *Inhal. Toxicol.* **2000**, *12,* 281-298.
30. Sardar, S.B.; Fine, P.M.; Mayo, P.R.; Sioutas, C. Size-Fractionated Measurements of Ambient Ultrafine Particle Chemical Composition in Los Angeles Using the NanoMOUDI; *Environ. Sci. Technol.* **2005**, *39,* 932-944.
31. Long, C.M.; Suh, H.H.; Catalano, P.J.; Koutrakis, P. Using Time- and Size-Resolved Particulate Data to Quantify Indoor Penetration and Deposition Behavior; *Environ. Sci. Technol.* **2001**, *35,* 4584-4584.
32. Geller, M.D.; Chang, M.H.; Sioutas, C.; Ostro, B.D.; Lipsett, M.J. Indoor/Outdoor Relationship and Chemical Composition of Fine and Coarse Particles in the Southern California Deserts; *Atmos. Environ.* **2002**, *36,* 1099-1110.
33. Na, K.; Cocker, D.R. Organic and Elemental Carbon Concentrations in Fine Particulate Matter in Residences, Schoolrooms, and Outdoor Air in Mira Loma, California; *Atmos. Environ.* **2005**, *39,* 3325-3333.
34. He, C.R.; Morawska, L.D.; Hitchins, J.; Gilbert, D. Contribution from Indoor Sources to Particle Number and Mass Concentrations in Residential Houses; *Atmos. Environ.* **2004**, *38,* 3405-3415.
35. Sarnat, S.E.; Coull, B.A.; Schwartz, J.; Gold, D.R.; Suh, H.H. Factors Affecting the Association between Ambient Concentrations and Personal Exposures to Particles and Gases; *Environ. Health Perspect.* **2006**, *114,* 649-654.
36. Zhu, Y.F.; Hinds, W.C.; Krudysz, M.; Kuhn, T.; Froines, J.; Sioutas, C. Penetration of Freeway Ultrafine Particles into Indoor Environments; *J. Aerosol Sci.* **2005**, *36,* 303-322.
37. *Overall Evaluations of Carcinogenicity to Humans (World Health Organization*); International Agency for Research on Cancer: Lyon, France, 2005.
38. Fine, P.M.; Chakrabarti, B.; Krudysz, M.; Schauer, J.J.; Sioutas, C. Diurnal Variations of Individual Organic Compound Constituents of Ultrafine and Accumulation Mode Particulate Matter in the Los Angeles Basin; *Environ. Sci. Technol.* **2004**, *38,* 1296-1304.
39. Delfino, R.J.; Staimer, N.; Gillen, D.; Tjoa, T.; Sioutas, C.; Fung, K.; George, S.C.; Kleinman, M.T. Personal and Ambient Air Pollution Is Associated with Increased Exhaled Nitric Oxide in Children with Asthma; *Environ. Health Perspect.* **2006**, *114,* 1736-1743.
40. Ebelt, S.T.; Petkau, A.J.; Vedal, S.; Fisher, T.V.; Brauer, M. Exposure of Chronic Obstructive Pulmonary Disease Patients to Particulate Matter: Relationships between Personal and Ambient Air Concentrations; *J. Air & Waste Manage. Assoc.* **2000**, *50,* 1081-1094.
41. Williams, R.; Creason, J.; Zweidinger, R.; Watts, R.; Sheldon, L.; Shy, C. Indoor, Outdoor, and Personal Exposure Monitoring of Particulate Air Pollution: the Baltimore Elderly Epidemiology-Exposure Pilot Study; *Atmos. Environ.* **2000**, *34,* 4193-4204.
42. Delvin, E.E.; Lopez, V.; Levy, E.; Menard, D. Control of Apolipoprotein Synthesis by Calcitriol and Clofibrate in Human Fetal Jejunum; *Gastroenterol.* **1997**, *112,* A870-A870.
43. Geyh, A.S.; Xue, J.P.; Ozkaynak, H.; Spengler, J.D. The Harvard Southern California Chronic Ozone Exposure Study: Assessing Ozone Exposure of Grade-School-Age Children in Two Southern California Communities; *Environ. Health Perspect.* **2000**, *108,* 265-270.
44. Mosqueron, L.; Momas, I.; Le Moullec, Y. Personal Exposure of Paris Office Workers to Nitrogen Dioxide and Fine Particles; *Occup. Environ. Med.* **2002**, *59,* 550-555.
45. Devlin, R.B.; Folinsbee, L.J.; Biscardi, F.; Hatch, G.; Becker, S.; Madden, M.C.; Robbins, M.; Koren, H.S. Inflammation and Cell Damage Induced by Repeated Exposure of Humans to Ozone; *Inhal. Toxicol.* **1997**, *9,* 211-235.
46. Gong, H.; Wong, R.; Sarma, R.J.; Linn, W.S.; Sullivan, E.D.; Shamoo, D. A.; Anderson, K.R.; Prasad, S.B. Cardiovascular Effects of Ozone Exposure in Human Volunteers; *Am. J. Respir. Crit. Care Med.* **1998**, *158*, 538-546.

About the Authors

Mohammad Arhami just completed his Ph.D. at the University of Southern California (USC), Andrea Polidori is a research assistant professor at USC, and Constantinos Sioutas is the Fred Champion professor at the Department of Civil and Environmental Engineering at USC. Ralph J. Delfino is an associate professor at the Department of Medicine, Epidemiology Division, at the University of California–Irvine (UCI), and Thomas Tjoa is a statistician at UCI. Please address correspondence to: Dr. Andrea Polidori, Department of Civil and Environmental Engineering, University of Southern California, 3620 South Vermont Avenue, Los Angeles, CA 90089; phone: +1-213-740-6134; fax: +1-909-396-2099; e-mail: polidori@usc.edu.