Fine Particle Sources and Cardiorespiratory Morbidity: An Application of Chemical Mass Balance and Factor Analytical Source-Apportionment Methods

Jeremy A. Sarnat,¹ Amit Marmur,² Mitchel Klein,¹ Eugene Kim,³ Armistead G. Russell,² Stefanie E. Sarnat,¹ James A. Mulholland,² Philip K. Hopke,⁴ and Paige E. Tolbert¹

¹Department of Environmental and Occupational Health, Emory University, Atlanta, Georgia, USA; ²Department of Civil and Environmental Engineering, Georgia Institute of Technology, Atlanta, Georgia, USA; ³California Air Resources Board, Sacramento, California, USA; ⁴Department of Chemical Engineering, Clarkson University, Potsdam, New York, USA

BACKGROUND: Interest in the health effects of particulate matter (PM) has focused on identifying sources of PM, including biomass burning, power plants, and gasoline and diesel emissions that may be associated with adverse health risks. Few epidemiologic studies, however, have included source-apportionment estimates in their examinations of PM health effects. We analyzed a timeseries of chemically speciated PM measurements in Atlanta, Georgia, and conducted an epidemiologic analysis using data from three distinct source-apportionment methods.

OBJECTIVE: The key objective of this analysis was to compare epidemiologic findings generated using both factor analysis and mass balance source-apportionment methods.

METHODS: We analyzed data collected between November 1998 and December 2002 using positive-matrix factorization (PMF), modified chemical mass balance (CMB-LGO), and a tracer approach. Emergency department (ED) visits for a combined cardiovascular (CVD) and respiratory disease (RD) group were assessed as end points. We estimated the risk ratio (RR) associated with same day PM concentrations using Poisson generalized linear models.

RESULTS: There were significant, positive associations between same-day PM_{2.5} (PM with aerodynamic diameter $\leq 2.5 \ \mu\text{m}$) concentrations attributed to mobile sources (RR range, 1.018–1.025) and biomass combustion, primarily prescribed forest burning and residential wood combustion, (RR range, 1.024–1.033) source categories and CVD-related ED visits. Associations between the source categories and RD visits were not significant for all models except sulfate-rich secondary PM_{2.5} (RR range, 1.012–1.020). Generally, the epidemiologic results were robust to the selection of source-apportionment method, with strong agreement between the RR estimates from the PMF and CMB-LGO models, as well as with results from models using single-species tracers as surrogates of the source-apportioned PM_{2.5} values.

CONCLUSIONS: Despite differences among the source-apportionment methods, these findings suggest that modeled source-apportioned data can produce robust estimates of acute health risk. In Atlanta, there were consistent associations across methods between $PM_{2.5}$ from mobile sources and biomass burning with both cardiovascular and respiratory ED visits, and between sulfate-rich secondary $PM_{2.5}$ with respiratory visits.

KEY WORDS: acute, Atlanta, cardiovascular, chemical mass balance, emergency department visits, fine particulate matter, positive matrix factorization, respiratory, source apportionment, tracer. *Environ Health Perspect* 116:459-466 (2008). doi:10.1289/ehp.10873 available via *http://dx.doi.org/* [Online 14 January 2008]

Recent interest in the health effects of particulate matter (PM) has focused on identifying sources of PM that pose the greatest health risks. Because it is likely that not all PM is equally toxic, epidemiologic models that incorporate source-resolved PM may provide a step toward targeting the most important causal agents and refining traditional mass-based PM standards. Quantifying health risks associated with sources such as biomass burning, power plants, gasoline and diesel emissions, rather than individual pollutants, may also capture complex multipollutant interactions that more accurately reflect the etiologic relationships between PM and adverse health. Few epidemiologic studies, however, have included sourceapportionment data in their examinations of PM health effects (Ito et al. 2006; Laden et al. 2000; Mar et al. 2000, 2006; Ozkavnak and Thurston 1987; Schreuder et al. 2006). The limited application of source apportionment may be attributable partly to uncertainties regarding optimal methods for conducting PM source apportionment, as well as the lack of suitable air quality data for analysis.

Source-apportionment methods used in previous studies have generally relied on factor analytic approaches. For example, Laden et al. (2000) grouped elemental PM concentrations from six U.S. cities into a small numbers of categories, or "factors" (Laden et al. 2000). Significant associations were found between mortality and the traffic and coal combustion factors, with the largest effect size for the traffic factor. No significant associations were observed for the oil and soil factors.

A series of recent analyses examined the associations between source-resolved PM estimates and mortality in Washington, DC, and Phoenix, Arizona, using several different multivariate factor analytic methods in each city (Hopke et al. 2006; Ito et al. 2006; Mar et al. 2006; Thurston et al. 2005). In these analyses, source apportionment was conducted on samples collected twice a week, using absolute principal components analysis (PCA), UNMIX (a multivariate receptor model), and positive matrix factorization (PMF). Results showed that variability among the methods was small when compared with overall sourceapportionment model uncertainty, and suggested that these apportionment methods may be useful in discerning source-specific health effects. The authors note the relatively limited sample size for these data sets and their inability to robustly identify certain source categories (e.g., specific mobile source types). Questions also remain concerning the generalizability of these findings to other locations with different aerosol compositions, the marginal benefit of using source-apportioned data over single-species tracers, and whether analyses using other source-apportionment methods, notably chemical mass balance (CMB), will show the same pattern of agreement.

Here we present and compare results from epidemiologic analyses of emergency department (ED) visits and source-resolved $PM_{2.5}$ (PM with aerodynamic diameter $\leq 2.5 \mu m$; fine PM) obtained using PMF, modified CMB, and a single-species tracer approach.

Supplemental Material is available online at http:// www.ehponline.org/members/2008/10873/suppl.pdf We thank the Electric Power Research Institute (EP-P4353/C2124) for supporting earlier work on the emergency department study and Atmospheric Research and Analysis for assistance in using Jefferson Street air quality measurements.

This work was supported by grants to Emory University from the U.S. Environmental Protection Agency (EPA) (R82921301-0) and the National Institute of Environmental Health Sciences (NIEHS) (R01ES11294), as well as to Georgia Tech (U.S. EPA grants RD832159, RD831076, and RD830960).

The content is solely the responsibility of the authors and does not necessarily represent the official views of U.S. EPA or NIEHS.

The authors declare they have no competing financial interests.

Received 11 September 2007; accepted 10 January 2008.

Address correspondence to J. Sarnat, Rollins School of Public Health of Emory University, Department of Environmental and Occupational Health, 1518 Clifton Rd., Room 260, Atlanta, GA 30322 USA. Telephone: (404) 712-9725. Fax: (401) 727-8744. E-mail: jsarnat@sph.emory.edu

This analysis is the first to compare epidemiologic findings generated using both factor analysis and mass balance source-apportionment methods. The data used in this analysis were collected in Atlanta, Georgia, a unique location for conducting this type of healtheffects study given the existence of an extensive time-series of daily speciated PM2.5 measurements and corresponding hospital records. These data have been previously characterized in several source-apportionment and epidemiologic analyses (Kim et al. 2004; Marmur et al. 2005; Metzger et al. 2004; Peel et al. 2005). We compare results across methods and assess the robustness of health risk estimates for cardiopulmonary ED visits. The implications of using one or several methods for understanding the sources of PM₂ 5-mediated health risks are also addressed.

Methods

Source-apportionment methods, including PMF, an extended CMB method, and singlespecies tracers, were applied to PM2.5 concentrations collected daily at an urban Atlanta site, Jefferson Street, between November 1998 and December 2002. The Jefferson Street site is located 4 km northwest of downtown and has served as a primary measurement site for the Southeastern Aerosol Research and Characterization Study (SEARCH), the Aerosol Research Inhalation Epidemiology Study (ARIES), and several ongoing epidemiologic studies as part of the extensive Studies of Particles and Health in Atlanta (SOPHIA) (Hansen et al. 2006; Metzger et al. 2004; Peel et al. 2005; Wade et al. 2006). Detailed particulate speciation was conducted to obtain particulate ionic, trace metals, and temperature-resolved carbonaceous concentrations, via ion chromatography, X-ray fluorescence, thermal-optical reflectance (TOR), respectively. The source-resolved PM_{2.5} was subsequently used in daily time-series analyses to estimate the risk ratio (RR) of respiratory disease (RD) and cardiovascular disease (CVD) ED visits.

Source apportionment of the Atlanta aerosol. Several source-apportionment studies have been conducted on speciated PM2.5 data collected at Jefferson Street. For the current analyses, PM_{2.5} source apportionment was conducted using PMF with temperatureresolved carbon fractions (Kim et al. 2004); an extended CMB approach using the Lipschitz global optimizer (LGO) program, CMB-LGO (Marmur et al. 2005); and single-species source-indicative tracers. The PMF and CMB-LGO analyses on the current data set were conducted, independent of each other, by researchers from Clarkson University and the Georgia Institute of Technology, respectively. Complete descriptions of the specific source-apportionment methods have been published elsewhere (Kim et al. 2004, 2003a,

2003b; Marmur et al. 2005), but brief summaries for each method are presented below.

PMF. PMF is a factor analytic method that distinguishes correlation patterns among speciated $PM_{2.5}$ measurements in a given location. As such, it does not rely on *a priori* knowledge concerning chemical profiles of sources to generate source contribution estimates. An often-noted limitation of using factor analysis methods is the inability to link observed factors in the analysis directly with actual sources. Because these methods are based on statistical patterns of correlations, rather than empirical chemical sources is somewhat subjective.

CMB-LGO. CMB receptor models are a common tool for apportioning ambient levels of pollutants among the major contributing sources. CMB combines the chemical and physical characteristics of particles measured at sources and receptors to quantify the source contributions to the receptor. The quantification is based on the solution to a set of linear equations that express each receptor's ambient chemical concentration as a linear sum of products of source-profile abundances and source contributions. In the enhanced CMB-LGO model, source-indicative sulfur dioxide/ PM2.5, carbon monoxide/PM2.5, and nitrogen oxides/PM2.5 ratios are used as constraints, in addition to the commonly used particulatephase source profiles. A limitation of CMB approaches is the assumption that profiles characterized at the source remain unchanged between source and receptor. For this comparison, both estimated source contributions from CMB-LGO and factor contributions from PMF will be referred to as "source categories."

Tracer method. Species that are characteristic of a given source profile and present in samples above their respective limits of detection may, in some cases, serve as suitable tracers of that source. Several source-indicative tracers were selected *a priori*, based on results from previously published studies (Koutrakis et al. 1992; Laden et al. 2000). For the current analyses, silicon was used as a tracer for soil, potassium for biomass or wood burning, zinc as a tracer for gasoline vehicles, elemental carbon (EC) for diesel sources, organic carbon (OC) for the CMB-LGO source "other OC," sulfate ion (SO42-) for sulfate-dominated secondary aerosol, nitrate ion (NO₃⁻) for nitratedominated secondary aerosol, and selenium for primary emissions from coal-fired power plants (Reddy et al. 2005). None of these elements should be considered unique to a given source.

To facilitate comparisons within and among the methods, most analyses were restricted to days when estimates for all source categories were available. Over the time period analyzed, a total of 1,018 daily measurements were included in the analyses [492 measurements during the cool season (October 15–April 14); 526 measurements during the warm season (April 15–October 14)]. Sensitivity analyses conducted comparing the restricted and complete data sets and their corresponding epidemiologic results showed little difference in the RR estimates and resulting interpretation.

Emergency department data. Information on individual-level ED visits was collected via electronic billing data from acute care hospitals serving the 20-county Atlanta metropolitan area. For the period November 1998 through December 2002, a subset of the ongoing SOPHIA study period, 27 of 42 hospitals serving the area were able to provide useable electronic data. We categorized ED visits into a combined RD case group using the following primary International Classification of Disease, 9th Revision (ICD-9; World Health Organization 1975) diagnostic codes: asthma (493, 786.09), chronic obstructive pulmonary disease (491, 492, 496), upper respiratory infection (460-466, 477), and pneumonia (480-486). A combined CVD group was also created that combined the following primary ICD-9 codes: ischemic heart disease (410-414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451-453). ED visits for each outcome group were aggregated by day for use in epidemiologic analyses. Repeat visits within a day by a specific patient were counted as a single visit.

Data analysis. Source impact comparisons. We compared source impacts within and between source-apportionment methods. Pollutant data were non-normally distributed, so we used Spearman's correlation coefficients. Many of the analyses were conducted using seasonally stratified data, given the differences in pollutant concentrations, distribution, and meteorology occurring in warm compared with cool seasons.

Epidemiologic analyses. We estimated the relative risk of daily RD and CVD ED visits associated with 24-hr integrated source impacts using Poisson generalized linear models (McCullagh and Nelder 1989). These analyses are similar to those used in our previous analyses of Atlanta data (Metzger et al. 2004; Peel et al. 2005). The basic form of the model is

```
\begin{split} \log[E(Y_t)] &= \alpha + \beta \text{ source category}_t \\ &+ \Sigma_{\kappa} \lambda_k \text{ DOW}_{kt} \\ &+ \Sigma_m \upsilon_m \text{ hospital}_{mt} \\ &+ g(\gamma_1, \dots, \gamma_N; \text{ time}_t) \\ &+ g(\sigma_1, \dots, \sigma_N; \text{ temp}_t) \\ &+ g(\eta_1, \dots, \eta_N; \text{ dewpoint}_t), \end{split}
```

where Y_t is the count of ED visits on day t for the outcome of interest. The model also included indicator variables for day of week and holidays (DOW), and hospital indicator variables (hospital) to account for the entry and exit of hospitals into and from the database during the study period. Long-term trends in case presentation rates (time) were controlled using semiparametric cubic splines, $g(\gamma_1,...,$ γ_{N} ; x) with monthly knots in both CVD and RD models. Cubic splines were used to control mean temperature (temp) and mean dewpoint temperature (dewpoint), with knots placed at the 25th and 75th percentiles. The first and second derivatives of g(x) were continuous allowing time trends and meteorology to be modeled as smooth functions. Variance estimates were scaled to account for Poisson overdispersion. In this study, we calculated all RRs for an increase of the approximate interquartile range (IQR) of the source category of interest. Relationships were examined with 8 of the 9 CMB sources and 10 of the 11 PMF factors. Relationships with the CMB ammonium bisulfate source and the PMF bus and highway factor were not obtained because of instability resulting from the large number of days in which there were no estimated source contributions from these categories.

In our previous analyses (Metzger et al. 2004; Peel et al. 2005), we observed associations for both cardiovascular and respiratory ED visits with same-day levels of total $PM_{2.5}$. Because an objective of the current analysis was to disaggregate the source-specific health effects associated with total $PM_{2.5}$, for each exposure variable (*source category*) in this analysis, the zero-day lag was chosen as the *a priori* exposure window. Secondary analyses also included models stratified by warm (April 15–October 14) and cool (October 15–April 14) seasons.

Results

The CMB-LGO and PMF analyses quantified impacts from 9 sources and 11 factors, respectively, for the Atlanta PM_{2.5} concentrations (Table 1). Complete summary statistics for the measured PM2.5 concentrations and source categories are presented in Table 2. Six comparable source categories-gasoline vehicles, diesel vehicles, biomass burning or wood smoke, soil, sulfate-rich secondary aerosols, and nitrate-rich secondary aerosols-were identified by both methods. Despite the similar category names and the fact that many of the source categories are driven by similar species, differences exist in the chemical profiles used by each method [Supplemental Material, Table 1 (online at http://www.ehponline.org/members/ 2008/10873/suppl.pdf)]. To assist in the identification of the factors as source types for PMF, the explained variation provides an indication of which sources are most responsible for explaining the variation in each particular chemical species used in the PMF analysis. A description of explained variation and the results for these data are presented in the Supplemental Material [Table 2 (online at http://www.ehponline.org/members/2008/10873/suppl.pdf)].

A substantial fraction of the Atlanta $PM_{2.5}$ was identified as sulfate-rich secondary aerosol, comprising approximately 40% of the total $PM_{2.5}$ mass during the study period (Table 2, Figure 1). Both the PMF and CMB-LGO models identified separate diesel and gasoline engine source categories. Both methods indicated that roughly 20% of the $PM_{2.5}$ mass in Atlanta originated from these two mobile source categories, although PMF apportioned a greater fraction of the total

 $PM_{2.5}$ mass to diesel as compared with CMB-LGO (13 vs. 9%, respectively). There were other differences regarding the identification of specific categories, such as primary $PM_{2.5}$ from coal-fired power plants, identified in CMB-LGO only, and cement, railroad, bus/highway, and metals-processing factors, identified in PMF only.

Associations within and between sourceapportionment methods. Several source categories within each method were correlated with each other, as expected given the strong influence of meteorologic factors on pollutant temporal patterns [Supplemental Material,

Table 1	Source-apportionment	category	/ names h	v method
Table L		Category	11011163 0	y methou.

PMF factors	CMB-LGO sources	Tracer	
Gasoline	Gasoline	PM _{2.5} Zn	
Diesel	Diesel	PM _{2.5} EC	
Wood smoke	Biomass burning	PM _{2.5} K	
Soil	Soil	PM _{2.5} Si	
Secondary sulfate I	Ammonium sulfate	PM _{2.5} sulfate	
Secondary nitrate	Ammonium nitrate	PM _{2.5} nitrate	
Secondary sulfate II	_		
Metal processing	_	_	
Railroad	—	_	
Bus and highway	—	_	
Cement kiln	—	_	
_	Power plants (1°)	PM _{2.5} Se	
_	Other OC	PM _{2.5} OC	
	Ammonium bisulfate		

Comparable source categories are listed on the same row.

Table 2. Mean, median, and selected percentiles (10th, 90th) of daily PM _{2.5} source contributions by season
from the ARIES monitoring station at Jefferson Street, Atlanta, GA (November 1998–December 2002).

	Cool season (n = 492 days)					Warm season (<i>n</i> = 526 days)			
	Mean	10th	Median	90th	Mea	in 10th	Median	90th	
Total PM _{2.5}	15.8	7.5	14.3	25.5	18.	2 9.1	17.0	29.0	
PMF diesel	2.6	0.5	1.8	6.3	1.	9 0.3	1.5	3.9	
CMB diesel	1.5	0.4	1.2	3.3	1.	4 0.5	1.2	2.6	
PM _{2.5} EC	1.7	0.6	1.4	3.3	1.	4 0.6	1.3	2.5	
PMF gasoline	1.7	0.3	1.2	3.6	1.	1 0.3	0.9	2.3	
CMB gasoline	1.5	0.5	1.3	2.9	1.	0 0.3	0.9	1.9	
$PM_{25}Zn (ng/m^3)$	15.7	4.6	11.7	30.2	10.	9 3.3	8.5	20.2	
PMF wood smoke	1.6	0.5	1.3	2.8	0.	8 0.1	0.7	1.4	
CMB biomass burning	1.1	0.5	1.0	2.0	0.	9 0.4	0.8	1.7	
PM _{2.5} K (ng/m ³)	63.0	24.3	53.9	114.2	52.	7 23.2	43.3	93.5	
PMF soil	0.3	0.0	0.3	0.7	0.	8 0.2	0.6	1.3	
CMB soil	0.2	0.0	0.1	0.4	0.	4 0.1	0.3	0.7	
PM _{2.5} Si (ng/m ³)	67.7	24.3	54.1	123.5	110.	9 32.9	89.0	186.3	
PMF secondary sulfate I	4.4	1.4	3.7	8.4	8.	7 3.0	7.6	16.4	
CMB ammonium sulfate	4.2	1.6	3.7	7.5	8.	2 3.3	7.3	14.3	
PM _{2.5} SO ₄ ²⁻	3.4	1.5	0.6	5.8	6.	0 2.3	5.2	10.8	
PMF secondary nitrate	1.4	0.4	1.2	2.6	0.	6 0.2	0.5	1.1	
CMB ammonium nitrate	2.0	0.6	1.7	3.6	0.	9 0.4	0.7	1.6	
PM _{2.5} NO ₃	1.4	0.5	1.2	2.6	0.	7 0.3	2.9	1.2	
CMB power plants ^a	0.1	0.0	0.1	0.3	0.	1 0.0	0.1	0.3	
$PM_{2.5}$ Se (ng/m ³)	1.4	0.4	1.1	3.0	1.	2 0.4	0.9	2.7	
CMB other OC	2.6	0.9	2.1	4.8	2.	5 1.1	2.3	4.2	
PM _{2.5} OC	4.6	1.9	3.9	8.0	4.	0 2.1	3.7	6.4	
CMB ammonium bisulfate	0.4	0.0	0.0	1.5	0.	4 0.0	0.0	1.1	
PMF secondary sulfate II	1.4	0.0	1.3	2.6	1.	7 0.4	1.5	3.2	
PMF cement kiln	0.4	0.1	0.3	0.8	0.	4 0.1	0.3	0.8	
PMF bus and highway	0.1	0.0	0.0	0.3	0.	1 0.0	0.0	0.2	
PMF railroad	0.5	0.1	0.5	0.9	0.	7 0.2	0.7	1.3	
PMF metal processing	0.8	0.1	0.6	1.8	0.	7 0.1	0.6	1.5	

Values reported in milligrams per cubic meter unless otherwise specified. Cool season is October 15–April 14; warm season is April 15–October 14. Secondary sulfate II contains a greater organic fraction than secondary sulfate I, which is mainly sulfate and ammonia.

^aReflects impacts from primary power plant emissions solely.

Table 3a–b (online at http://www.ehponline. org/members/2008/10873/suppl.pdf)]. In general, the PMF factors were more orthogonal (i.e., less correlated) than the CMB-LGO sources, which was expected given that PMF factors are identified in part on the basis of their lack of collinearity. Moderate temporal correlation (i.e., Spearman's r > 0.40-0.60) existed between the gas, diesel, and biomass burning source categories in CMB-LGO. Similarly, moderate correlation existed among the gas, diesel, and wood smoke PMF categories.

Correlations between PMF and CMB-LGO source estimates were generally strong for the six comparable categories that were identified by both methods (Table 3). For the diesel, biomass burning or wood smoke, soil, and sulfate-rich and nitrate-rich secondary aerosol source categories, correlations exceeded 0.80 during both seasons, except for the warm season correlation between PMF and CMB-LGO soil ($r_{\rm S} = 0.69$). The gasoline source category was a notable point of contrast, with only weak to moderate correlations between the methods during both seasons $(r_{\rm S} = 0.45 \text{ and } 0.24 \text{ in cool and warm seasons},$ respectively). Mean absolute deviations between values estimated by both sources were generally $< 1 \ \mu g/m^3$, which for specific source categories represented large relative differences [Supplemental Material, Table 4 (online at http://www.ehponline.org/members/ 2008/10873/suppl.pdf)]. Differences between methods were typically greatest for gasoline and soil impacts relative to their respective means, with the sulfate- and nitrate-rich secondary aerosols exhibiting the least relative deviation.

Many of the elemental species were also strongly correlated with specific source categories, supporting their *a priori* selection as source tracers (Table 4). This was not true for



Figure 1. Fractional contribution to PM_{2.5} from (*A*) PMF and (*B*) CMB-LGO source categories at Jefferson Street, Atlanta, GA (November 1998–December 2002).

all tracer species. For example, Zn was strongly correlated with CMB gasoline source contributions ($r_{\rm S} = 0.96$), but only moderately to weakly correlated with the PMF gasoline estimates ($r_{\rm S} = 0.42$). Se was not a strong tracer for power plant source contributions ($r_{\rm S} \approx 0.4$), likely because of its relatively low signal-to-noise ratio.

Associations with RD and CVD visits. Thirty-seven of 42 hospitals in the metropolitan Atlanta area provided data on > 4.5 million emergency department visits for the time period included in these analyses. There was an average (± SD) of 324 ± 123 (range, 105–1,061) and 75 ± 17 (range, 32–123) visits per day for RD and CVD visits, respectively. In the restricted data set, same-day increases in total PM2.5 (i.e., a 0-day lag) were significantly associated with increases in CVD visits [RR = 1.022; 95% confidence interval (CI), 1.007-1.038], but not RD visits (RR = 1.005; 95% CI, 0.996-1.015). In the nonrestricted data set for this time period, PM2.5 was also significantly associated with RD visits (RR = 1.016; 95%) CI, 1.007–1.024) for a 0-day lag.

Associations between the same-day concentration of the PM_{2.5} source categories and RD visits were consistent with the null for all models using PMF, CMB-LGO, and the tracers, except for the source categories characterizing sulfate-rich PM_{2.5} [Figure 2; Supplemental Material, Table 5 (online at http://www.ehponline.org/members/ 2008/10873/suppl.pdf)]. An IQR increase in sulfate-rich secondary PM_{2.5}, formed mainly from primary power plant precursors, was associated with 1.2–2.0% increases in respiratory visits. In contrast, we found a significant negative association between respiratory visits

Table 3. Spearman's correlation coefficient between PMF and CMB-LGO source categories during the cool season (10/15–4/14) and warm season (4/15–10/14) (November 1998–December 2002).

						PMF					
			Wood		Secondary	Secondary	Secondary	Metal		Bus and	
CMB	Gas	Diesel	smoke	Soil	sulfate I	nitrate	sulfate II	processing	Railroad	highway	Cement kiln
Cool season											
Gas	0.45	0.72	0.12	0.61	0.20	0.29	0.00	0.69	-0.15	0.44	0.37
Diesel	0.57	0.88	0.16	0.45	0.20	-0.09	0.04	0.43	0.13	0.46	0.21
Biomass burning	0.23	0.33	0.89	0.28	0.25	-0.21	0.03	0.20	0.10	0.27	0.30
Soil	0.67	0.73	0.29	0.83	0.31	0.14	0.10	0.42	-0.26	0.33	0.25
Ammonium sulfate	0.20	0.31	0.30	0.40	0.85	0.04	0.21	0.16	-0.03	0.00	0.15
Ammonium nitrate	0.13	0.26	-0.03	0.29	0.18	0.89	0.11	0.27	-0.18	0.14	0.15
Power plants (1°)	-0.08	0.01	0.38	0.04	0.00	0.01	0.20	-0.01	0.13	-0.01	0.76
Ammonium bisulfate	-0.13	-0.12	-0.12	-0.08	0.09	0.28	-0.01	-0.05	-0.06	0.02	-0.10
Other OC	0.80	0.76	0.11	0.45	0.16	-0.10	0.16	0.27	-0.01	0.30	0.07
Warm season											
Gas	0.24	0.58	0.06	0.44	0.24	0.31	0.17	0.62	0.02	0.25	0.56
Diesel	0.28	0.81	0.15	0.30	0.34	0.22	0.23	0.31	0.29	0.27	0.46
Biomass burning	0.23	0.34	0.94	0.19	0.04	-0.09	0.14	0.06	-0.01	0.06	0.39
Soil	0.60	0.62	0.49	0.69	0.29	0.16	0.20	0.21	-0.25	0.02	0.44
Ammonium sulfate	0.10	0.40	0.00	0.27	0.96	0.25	0.41	0.21	0.11	-0.03	0.26
Ammonium nitrate	0.15	0.44	-0.05	0.28	0.28	0.87	0.12	0.29	0.05	0.17	0.27
Power plants (1°)	0.11	0.30	0.37	0.28	0.28	0.01	0.19	0.17	0.06	0.11	0.66
Ammonium bisulfate	0.10	0.02	0.07	0.09	0.26	-0.05	0.06	-0.10	-0.16	-0.09	0.00
Other OC	0.61	0.68	0.11	0.30	0.43	-0.04	0.48	0.04	0.06	0.10	0.26

Secondary sulfate II contains a greater organic fraction than secondary sulfate I, which is mainly sulfate and ammonia.

and primary emissions from coal-fired power plants in CMB-LGO.

Visits to the ED for CVD were significantly and positively associated with CMB-LGO diesel, gas, biomass burning, and other OC source categories [Figure 3, Supplemental Material, Table 5 (online at http://www. ehponline.org/members/2008/10873/suppl. pdf)]. IQR increases in these sources were associated with RR estimates ranging from 1.014 (other OC) to 1.033 (biomass). PMF diesel, gas, wood smoke, and metal processing categories were also significantly and positively associated with CVD visits, with the magnitude of RRs per IQR ranging from 1.013 (metal processing) to 1.025 (diesel). Similarly, EC and K, selected as single-species tracers for the diesel and biomass burning source categories, were also significantly associated with CVD visits.

Analyses comparing the observed RR estimates for source categories identified by all three methods indicated strong agreement among the three source-apportionment methods. Scatterplots of the RR values from CMB-LGO predicting the RR values from PMF, the RR values from PMF predicting the RR values from the tracer method, and the RR values from CMB-LGO predicting the RR values from the tracer method showed approximate one-to-one associations (Figure 4). Variability in observed RR estimates within the CMB-LGO model output, for example, explained 87% of the corresponding variability in the PMF-based RRs.

There was some indication that associations with CVD outcomes were stronger during the cooler months in Atlanta, whereas associations with respiratory visits were stronger during the warmer months from models stratified between cool and warm months [Supplemental Material, Table 6 (online at http://www.ehponline.org/ members/2008/10873/suppl.pdf)]. Pollutant variables found to be associated with ED visits throughout the year were PMF diesel, EC, CMB-LGO gas, Zn, and the three biomass combustion source categories (i.e., CMB-LGO biomass burning, PMF wood smoke, and K). Diesel and gas sources, from both PMF and CMB-LGO, which were not associated with respiratory visits in the overall models, were associated with significant increases in respiratory visits (1.2-2.1% per IQR) during the warm season.

Discussion

The current results indicate that specific sources are associated with PM_{2.5}-related health effects in Atlanta. Additionally, these findings raise new questions concerning the current limitations and resulting value of the source apportionment–epidemiology approach. During this 4-year time-series in Atlanta, there were clear, positive associations between same-day PM_{2.5} concentrations attributed to OC-dominated source categories, such as mobile sources and biomass burning, and ED visits for CVDrelated causes. Respiratory visits were also associated with sulfate-rich secondary PM2 5 and, during the warmer months, with mobile source-related source impacts. Each of these results was robust to the selection of sourceapportionment method, with strong agreement between the RR estimates from the PMF and CMB-LGO models, as well as with results from models using single-species tracers as surrogates of the source-apportioned PM_{2.5} values. Together, these findings indicate a link between these sources or specific chemical components from these sources and acute, adverse cardiopulmonary responses in Atlanta.

Despite the substantial methodologic differences and sources of uncertainty that differentiate PMF from CMB-LGO, the epidemiologic model results were strikingly similar in direction and magnitude. This analysis was the first, to our knowledge, to compare epidemiologic results incorporating both factor analytical and chemical mass balance source impacts as exposure terms. Several previous studies examined results generated from factor analytic-epidemiologic models exclusively and found generally consistent estimates across methods (Ito et al. 2006; Mar et al. 2006; Schreuder et al. 2006; Thurston et al. 2005). Ito et al. (2006), for example, examined time-series mortality risks associated with PM2.5 apportioned impacts from four PCA/UNMIX and two PMF models in Washington, DC. These models identified up to nine factors, with sea salt, oil combustion, and soil having the greatest between-method heterogeneity in factor estimates with secondary sulfate having the least. Analysis of variation showed that variability across source types was approximately 10 times that of variability across methods. In Atlanta, as well, source contribution estimates from PMF and CMB-LGO differed most for soil and least for secondary sulfate, yet showed generally strong agreement.

It is likely that some homogeneity among the risk estimates across methods was

Table 4. Spearman's correlation coefficients between single species tracers, CMB, and PMF by season.

		Cool s correlati	eason ion with	Warm season correlation with	
Source category	Tracer	CMB	PMF	CMB	PMF
Gas	Zn	0.96	0.42	0.94	0.19
Diesel	EC	0.96	0.95	0.96	0.88
Wood smoke/biomass burning	K	0.99	0.82	0.98	0.62
Soil	Si	0.83	0.82	0.96	0.92
Secondary sulfate I/ammonium sulfate	S04 ²⁻	0.87	0.97	0.96	0.99
Secondary nitrate/ammonium nitrate	N03 ⁻	0.94	0.95	0.95	0.92
Power plant	Se	0.39		0.50	_
Other OC	00	0.91	—	0.89	_

Cool season is October 15-April 14; warm season is April 15-October 14.



Figure 2. RRs and 95% CIs per IQR increase from same-day lag models for the association of ED visits for all respiratory disease with daily source-apportioned ambient PM_{2.5} (Atlanta, GA, November 1998–December 2002).

attributable to the fact that in Atlanta, many of the PMF factors and CMB-LGO source impacts were driven by similar species (e.g., K with biomass burning or wood smoke, Si with soil, ionic sulfate with secondary sulfate). This fact also explains a key observed finding that the epidemiologic model results using source indicative tracers generated similar RRs to those from the CMB-LGO and PMF models. Together, the results raise the question of whether source-indicative tracers provide an accurate and less analytically intensive alternative to conducting source apportionment. We recommend a cautious approach in using tracers exclusively, however, because many of these species are emitted from numerous sources (e.g., K, used here as a tracer for biomass burning or wood smoke, is also found in soil) and it is likely that the same species may not serve as tracers to the same sources in different locations (e.g., Zn, an additive to lubricating oil, is indicative of gasoline vehicles in a non-heavily industrial city as Atlanta, but is also associated with industrial metals processing in some locations). An alternative approach, therefore, would be to conduct a sensitivity analysis of source-apportionment results, to mathematically identify and quantify the effect of each species on the apportionment process source-indicative tracers for a given local source (Marmur et al. 2006, 2007). Such an analysis is very useful in determining the most influential tracers for each source category in a given location, and can assist in selecting specific tracers to be investigated as part of an epidemiologic analysis. Although correlations between sources and species can provide such initial estimates, these are descriptive rather than conclusive.

Using both factor analytical and chemical mass balance methods in the same analysis may partially compensate for weaknesses in each method. For example, the process of naming factors and linking them with specific sources in PMF analyses is generally subjective. This is not true for CMB-LGO, which only includes sources in a model for which a known source profile exists, though collinearity and exclusion of sources might cause "misplacement" of emissions between the various sources examined. Conversely, CMB processes are constrained by information available for specific source profiles. If information about a specific source profile does not exist, it will not be apportioned as its own source impact within the CMB model. PMF, conversely, empirically examines temporal patterns to identify factors that fit the data set. Typically, modelers will employ several means of optimizing the number of factors selected, including the physical validity of the model (Reff et al. 2007). The mathematical identification of factors within a data set, therefore, may highlight sources that are potentially missing from a CMB model in a given location. Therefore, more certainty is associated with a source category that is identified by both PMF and CMB-LGO.

During this time period in Atlanta, there were consistent associations between $PM_{2,5}$ emitted from both gasoline and diesel sources and CVD and, to a lesser degree, respiratory ED visits. A recent study examining health effects associated with 19 $PM_{2,5}$ components in six California counties also found substantially elevated mortality risks associated with EC and Zn with CVD and respiratory mortalities (Ostro et al. 2007). As such, the



Figure 3. RRs and 95% CIs per IQR increase from same-day lag models for the association of ED visits for all CVD with daily source-apportioned ambient $PM_{2.5}$ (Atlanta, GA, November 1998–December 2002).

Atlanta results contribute to growing observations that mobile source $PM_{2.5}$ is, in part, a key agent responsible for acute cardiovascular outcomes in many locations (Adar et al. 2007; Bigert et al. 2003; Finkelstein et al. 2004; Hoek et al. 2002; Mills et al. 2005; Ostro et al. 2007; Pekkanen et al. 2000; Peters et al. 2001, 2004; Riediker et al. 2004; Salvi et al. 1999; Schwartz 2001; Schwartz et al. 2005; Sioutas et al. 2005). Although the exact biological mechanisms responsible for traffic-mediated cardiovascular effects remain uncertain, several plausible hypotheses have emerged related to autonomic system and inflammatory response pathways (Brook et al. 2004; Donaldson et al. 2001; Pope et al. 2004; Stone and Godleski 1999).

Sulfate-rich secondary PM_{2.5}, formed largely from photochemical reactions involving SO₂ from primary power plant emissions, was the only source category significantly associated with respiratory visits in the primary models. Although other studies have reported similar effects examining particulate sulfate concentrations (Burnett et al. 1997; Fairley 1999; Schwartz et al. 1996), evidence to support this finding is still inconclusive (Reiss et al. 2007). The sulfate finding is interesting given the observed negative association between respiratory visits and primary PM_{2.5} from power plants. Moreover, there was almost no correlation between secondary sulfate aerosol and primary power plant source contributions, reflecting the different atmospheric processes controlling these primary and secondary source impacts. Given the lack of correlation and different composition of these source impacts [Supplemental Material, Table 2 (online at http://www. ehponline.org/members/2008/10873/suppl. pdf)], the difference in the observed health response is not surprising.

The negative association between primary coal-fired power plant emissions and health outcomes should not be viewed as protective. Primary emissions from coal-fired power plants have the most significant impact on the Jefferson Street monitoring site under very specific conditions (i.e., when the wind is blowing from a power plant that is 7 km away, and when the mixed layer is high enough to entrain those emissions). However, the width of the power plant plume beyond 7 km is quite narrow, and a rather small fraction of the sampled area is being affected at that same time. On the other hand, having relatively stronger winds and a higher mixed depth will decrease many other primary emissions.

Associations of $PM_{2.5}$ components and sources with ED visits for CVD were typically stronger during the cooler months, and associations of respiratory visits stronger during the warmer months, which is consistent with findings from our previous Atlanta analyses

(Metzger et al. 2004; Peel et al. 2005). The impact of season on specific source impacts may be explained by atmospheric conditions that predominate during specific times of the year. During the cooler months, reduced photochemistry and greater atmospheric stability typically leads to the enhanced impact from local sources of primary PM_{2.5}, such as that from gasoline and diesel vehicles. Conversely, photochemistry during the warmer months will enhance the contribution of secondary PM_{2.5} from regional sources, such as sulfaterich secondary aerosols and secondary organic aerosol. Biomass burning or wood smoke was the sole source category that showed consistent year-round associations that, in the current analyses, were with CVD visits. This may be explained by the fact that, among the sources examined, it is one of the few that has considerable local (e.g., residential or industrial wood burning) and regional contributions (e.g., prescribed agricultural burning) (Marmur et al. 2006; Zheng et al. 2007). Alternatively, these results could reflect seasonal variation in risk factors not associated with air pollution, for which the analyses are not adequately accounting. Respiratory illnesses, for example, are higher in the cold and flu season, which makes potential air pollution effects more difficult to ascertain during the cold season. Heat stress is a risk factor for CVD illnesses and may lead to a reduced ability to detect air pollution effects in the warm season.

In Atlanta, biomass burning, due to prescribed forest management burning in the summer and residential wood burning in the winter, contributed a sizeable fraction of the PM_{2.5}. Associations between PM_{2.5} attributed to biomass burning and CVD visits were somewhat unexpected, however. Several previous PM epidemiologic studies examining PM_{2.5} from this source have found little or no effect on CVD outcomes (Ito et al. 2006; Mar et al. 2006; Schreuder et al. 2006), yet fairly consistent associations related to a variety of respiratory end points (Larson and Koenig 1994; Schreuder et al. 2006; Zelikoff et al. 2002). A few studies have noted similar associations that lend plausibility to the Atlanta findings. Ostro et al. (2007) recently

presented results showing significant associations between ambient K concentrations and CVD-, but not respiratory-, related mortality in California. Additionally, several controlled experiments have shown relationships between wood smoke inhalation and markers of acute systemic inflammatory response in animal and human models (Barregard et al. 2006; Barrett et al. 2006). Barrett et al. (2006) found increased levels of serum amyloid A, a cardiovascular risk factor, as well as other markers of plasma coagulability in 13 human subjects exposed to PM wood smoke concentrations over a 4-hr duration. Evidence also suggests that pulmonary inflammation, more commonly associated with biomass burning, may precede a systemic autonomic and inflammatory response as a potential step in the biological pathway of PM-mediated cardiovascular health effects (Brook et al. 2002; Gold et al. 2000).

The CVD response associated with PM2.5 from biomass burning or wood smoke was similar in significance and lag structure to that attributed to the diesel source category. It is possible that the compounds responsible for the observed mobile source-related CVD effects may also be present in Atlanta biomass emissions, and that meteorology-induced temporal correlations between the sources led to similar observed responses. Indeed, controlled speciation studies of wood smoke and motor vehicles have identified multiple shared particle and gas-phase organic compounds in their emissions (Schauer and Cass 2000; Zheng et al. 2002). A previous analysis of measurement sites in the southeastern United States, including Atlanta, for example, found that both gasoline vehicles and biomass burning jointly contributed to the local distributions of several polycyclic aromatic hydrocarbon concentrations (i.e., indeno[cd]pyrene and indeno[cd]fluoranthene) (Zheng et al. 2002). Broadly, the observed CVD responses for many of the source categories characterized by high organic content may reflect the presence of shared compounds in the emissions or similar particle attributes, such as oxidation potential, common to organic carbon species. This interpretation may also explain the strong

association between the non-source-apportioned total OC concentration and CVD visits.

Multivariate modeling to control for potential confounding is particularly challenging with source-apportionment variables, given the differential uncertainties associated with each source category. In the current analysis, for example, the results showing positive associations with PM2.5 from gasoline sources may represent a true association with PM_{2.5} from this source or the gas source may be serving as a surrogate for exposure to PM_{2.5} from a co-varying source, such as PM_{2.5} from diesel, biomass burning, or other, secondary organic aerosol. In this setting, the effects of source variables measured with greater error will be transferred, in part, to those variables with less measurement error (Schwartz and Coull 2003; Zeger et al. 2000). Additionally, multiple sources often contain common pollutant constituents, further complicating the interpretation of parameter estimates obtained from multisource models. Finally, methods for adjusting model parameters based on empirical uncertainty estimates, such as regression calibration, are questionable because each source category is an aggregate of numerous pollutants each with their own error distribution and covariances.

Future analyses using data with greater pollutant resolution, especially in the organic fraction, may elucidate some of the outstanding issues raised in the current analysis. Organic fingerprints of specific sources exist for a subset of these data (Zheng et al. 2000, 2007) and will contribute toward our ability to disaggregate OC-dominated source impacts. Planned analyses involving individual ED case groups will be helpful in disaggregating nuances in health response that we found when using the aggregated health outcome categories. Specific PM_{2.5} sources or components, for example, may be associated with certain health responses but not others (e.g., congestive heart failure compared with ischemic heart disease) due to etiologic differences among these subcategories. Despite the limitations of current source-apportionment methodology, these findings collectively suggest that one can use variables describing



Figure 4. Agreement of estimated RRs (0-day lag) for all RD and CVD between (*A*) CMB and PMF ($R^2 = 0.87$), (*B*) CMB and single-species tracers ($R^2 = 0.76$), and (*C*) PMF and single-species tracers ($R^2 = 0.87$). Observations are taken from the comparable source categories.

sources in epidemiologic models to obtain estimates of $PM_{2.5}$ acute health effects. Moreover, combining multiple source-apportionment methods adds information that compensates for limitations of relying on any single method.

REFERENCES

- Adar SD, Gold DR, Coull BA, Schwartz J, Stone PH, Suh H. 2007. Focused exposures to airborne traffic particles and heart rate variability in the elderly. Epidemiology 18(1):95–103.
- Barregard L, Sallsten G, Gustafson P, Andersson L, Johansson L, Basu S, et al. 2006. Experimental exposure to woodsmoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation. Inhal Toxicol 18(11):845–853.
- Barrett EG, Henson RD, Seilkop SK, McDonald JD, Reed MD. 2006. Effects of hardwood smoke exposure on allergic airway inflammation in mice. Inhal Toxicol 18(1):33–43.
- Bigert C, Gustavsson P, Hallqvist J, Hogstedt C, Lewne M, Plato N, et al. 2003. Myocardial infarction among professional drivers. Epidemiology 14(3):333–339.
- Brook RD, Brook JR, Urch B, Vincent R, Rajagopalan S, Silverman F. 2002. Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. Circulation 105(13):1534–1536.
- Brook RD, Franklin B, Cascio W, Hong YL, Howard G, Lipsett M, et al. 2004. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. Circulation 109(21):2655–2671.
- Burnett RT, Cakmak S, Brook JR, Krewski D. 1997. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environ Health Perspect 105:614–620.
- Donaldson K, Stone V, Seaton A, MacNee W. 2001. Ambient particle inhalation and the cardiovascular system: potential mechanisms. Environ Health Perspect 109:523–527.
- Fairley D. 1999. Daily mortality and air pollution in Santa Clara County, California: 1989–1996. Environ Health Perspect 107:637–641.
- Finkelstein MM, Jerrett M, Sears MR. 2004. Traffic air pollution and mortality rate advancement periods. Am J Epidemiol 160(2):173–177.
- Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B, et al. 2000. Ambient pollution and heart rate variability. Circulation 101(11):1267–1273.
- Hansen DA, Edgerton E, Hartsell B, Jansen J, Burge H, Koutrakis P, et al. 2006. Air quality measurements for the aerosol research and inhalation epidemiology study. J Air Waste Manag Assoc 56(10):1445–1458.
- Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. 2002. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. Lancet 360(9341):1203–1209.
- Hopke PK, Ito K, Mar T, Christensen WF, Eatough DJ, Henry RC, et al. 2006. PM source apportionment and health effects. 1. Intercomparison of source apportionment results. J Expo Sci Environ Epidemiol 16(3):275–286.
- Ito K, Christensen WF, Eatough DJ, Henry RC, Kim E, Laden F, et al. 2006. PM source apportionment and health effects. 2. An investigation of intermethod variability in associations between source-apportioned fine particle mass and daily mortality in Washington, DC. J Expo Sci Environ Epidemiol 16(4):300–310.
- Kim E, Hopke PK, Edgerton ES. 2004. Improving source identification of Atlanta aerosol using temperature resolved carbon fractions in positive matrix factorization. Atmos Environ 38(20):3349–3362.

- Kim E, Hopke PK, Edgerton ES. 2003a. Source identification of Atlanta aerosol by positive matrix factorization. J Air Waste Manag Assoc 53(6):731–739.
- Kim E, Hopke PK, Paatero P, Edgerton ES. 2003b. Incorporation of parametric factors into multilinear receptor model studies of Atlanta aerosol. Atmos Environ 37(36):5009–5021.
- Koutrakis P, Briggs SLK, Leaderer BP. 1992. Source apportionment of indoor aerosols in Suffolk and Onondaga Counties, New York. Environ Sci Technol 26(3):521–527.
- Laden F, Neas LM, Dockery DW, Schwartz J. 2000. Association of fine particulate matter from different sources with daily mortality in six US cities. Environ Health Perspect 108:941–947.
- Larson TV, Koenig JQ. 1994. Wood smoke: emissions and noncancer respiratory effects. Annu Rev Public Health 15:133–156.
- Mar TF, Ito K, Koenig JQ, Larson TV, Eatough DJ, Henry RC, et al. 2006. PM source apportionment and health effects. 3. Investigation of inter-method variations in associations between estimated source contributions of PM_{2.5} and daily mortality in Phoenix, AZ. J Expo Sci Environ Epidemiol 16(4):311–320.
- Mar TF, Norris GA, Koenig JQ, Larson TV. 2000. Associations between air pollution and mortality in Phoenix, 1995–1997. Environ Health Perspect 108:347–353.
- Marmur A, Mulholland JA, Russell AG. 2007. Optimized variable source-profile approach for source apportionment. Atmos Environ 41(3):493–505.
- Marmur A, Park SK, Mulholland JA, Tolbert PE, Russell AG. 2006. Source apportionment of PM_{2.5} in the southeastern United States using receptor and emissions-based models: Conceptual differences and implications for timeseries health studies. Atmos Environ 40(14):2533–2551.
- Marmur A, Unal A, Mulholland JA, Russell AG. 2005. Optimization-based source apportionment of PM₂₅ incorporating gas-to-particle ratios. Environ Sci Technol 39(9):3245–3254.
- McCullagh P, Nelder JA. 1989. Generalized Linear Models. 2nd ed. New York:Chapman and Hall.
- Metzger KB, Tolbert PE, Klein M, Peel JL, Flanders WD, Todd K, et al. 2004. Ambient air pollution and cardiovascular emergency department visits. Epidemiology 15(1):46–56.
- Mills NL, Tornqvist H, Robinson SD, Gonzalez M, Darnley K, MacNee W, et al. 2005. Diesel exhaust inhalation causes vascular dysfunction and impaired endogenous fibrinolysis. Circulation 112(25):3930–3936.
- Ostro B, Feng WY, Broadwin R, Green S, Lipsett M. 2007. The effects of components of fine particulate air pollution on mortality in California: results from CALFINE. Environ Health Perspect 115:13–19.
- Ozkaynak H, Thurston GD. 1987. Associations between 1980 United-States mortality-rates and alternative measures of airborne particle concentration. Risk Anal 7(4):449–461.
- Peel JL, Tolbert PE, Klein M, Metzger KB, Flanders WD, Todd K, et al. 2005. Ambient air pollution and respiratory emergency department visits. Epidemiology 16(2):164–174.
- Pekkanen J, Brunner EJ, Anderson HR, Tiittanen P, Atkinson RW. 2000. Daily concentrations of air pollution and plasma fibrinogen in London. Occup Environ Med 57(12):818–822.
- Peters A, Frohlich M, Doring A, Immervoll T, Wichmann HE, Hutchinson WL, et al. 2001. Particulate air pollution is associated with an acute phase response in men: results from the MONICA-Augsburg Study. Eur Heart J 22(14):1198–1204.
- Peters A, von Klot S, Heier M, Trentinaglia I, Hormann A, Wichmann HE, et al. 2004. Exposure to traffic and the onset of myocardial infarction. N Engl J Med 351(17):1721–1730.
- Pope CA, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, et al. 2004. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation 109(1):71–77.

Reddy MS, Basha SB, Joshi HV, Jha B. 2005. Evaluation of the

emission characteristics of trace metals from coal and fuel oil fired power plants and their fate during combustion. J Hazard Mater 123(1-3):242-249.

- Reff A, Eberly SI, Bhave PV. 2007. Receptor modeling of ambient particulate matter data using positive matrix factorization: review of existing methods. J Air Waste Manag Assoc 57(2):146–154.
- Reiss R, Anderson EL, Cross CE, Hidy G, Hoel D, McClellan R, et al. 2007. Evidence of health impacts of sulfate- and nitrate-containing particles in ambient air. Inhal Toxicol 19(5):419–449.
- Riediker M, Cascio WE, Griggs TR, Herbst MC, Bromberg PA, Neas L, et al. 2004. Particulate matter exposure in cars is associated with cardiovascular effects in healthy young men. Am J Respir Crit Care Med 169(8):934–940.
- Salvi S, Blomberg A, Rudell B, Kelly F, Sandstrom T, Holgate ST, et al. 1999. Acute inflammatory responses in the airways and peripheral blood after short-term exposure to diesel exhaust in healthy human volunteers. Am J Respir Crit Care Med 159(3):702–709.
- Schauer JJ, Cass GR. 2000. Source apportionment of wintertime gas-phase and particle-phase air pollutants using organic compounds as tracers. Environ Sci Technol 34(9):1821–1832.
- Schreuder AB, Larson TV, Sheppard L, Claiborn CS. 2006. Ambient woodsmoke and associated respiratory emergency department visits in Spokane, Washington. Int J Occup Environ Health 12(2):147–153.
- Schwartz J. 2001. Air pollution and blood markers of cardiovascular risk. Environ Health Perspect 109:405–409.
- Schwartz J, Coull BA. 2003. Control for confounding in the presence of measurement error in hierarchical models. Biostatistics 4(4):539–553.
- Schwartz J, Dockery DW, Neas LM. 1996. Is daily mortality associated specifically with fine particles? J Air Waste Manag Assoc 46(10):927–939.
- Schwartz J, Litonjua A, Suh H, Verrier M, Zanobetti A, Syring M, et al. 2005. Traffic related pollution and heart rate variability in a panel of elderly subjects. Thorax 60(6):455–461.
- Sioutas C, Delfino RJ, Singh M. 2005. Exposure assessment for atmospheric ultrafine particles (UFPs) and implications in epidemiologic research. Environ Health Perspect 113:947–955.
- Stone PH, Godleski JJ. 1999. First steps toward understanding the pathophysiologic link between air pollution and cardiac mortality. Am Heart J 138(5 pt 1):804–807.
- Thurston GD, Ito K, Mar T, Christensen WF, Eatough DJ, Henry RC, et al. 2005. Workgroup report: workshop on source apportionment of particulate matter health effects—intercomparison of results and implications. Environ Health Perspect 113:1768–1774.
- Wade KS, Mulholland JA, Marmur A, Russell AG, Hartsell B, Edgerton E, et al. 2006. Effects of instrument precision and spatial variability on the assessment of the temporal variation of ambient air pollution in Atlanta, Georgia. J Air Waste Manag Assoc 56(6):876–888.
- World Health Organization. 1975. International Classification of Diseases, 9th Revision. Geneva:World Health Organization.
- Zeger SL, Thomas D, Dominici F, Samet JM, Schwartz J, Dockery D, et al. 2000. Exposure measurement error in time-series studies of air pollution: concepts and consequences. Environ Health Perspect 108:419–426.
- Zelikoff JT, Chen LC, Cohen MD, Schlesinger RB. 2002. The toxicology of inhaled woodsmoke. J Toxicol Environ Health B Crit Rev 5(3):269–282.
- Zheng M, Cass GR, Ke L, Wang F, Schauer JJ, Edgerton ES, et al. 2007. Source apportionment of daily fine particulate matter at Jefferson Street, Atlanta, GA, during summer and winter. J Air Waste Manag Assoc 57(2):228–242.
- Zheng M, Cass GR, Schauer JJ, Edgerton ES. 2002. Source apportionment of PM2.5 in the southeastern United States using solvent-extractable organic compounds as tracers. Environ Sci Technol 36(11):2361–2371.