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Source-apportioned $PM_{2.5}$ and cardiorespiratory emergency department visits: accounting for source contribution uncertainty

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Abstract

Background: Despite evidence suggesting that air pollution-related health effects differ by emissions source, epidemiologic studies on fine particulate matter ($PM_{2.5}$) infrequently differentiate between particles from different sources. Those that do rarely account for the uncertainty of source apportionment methods.

Methods: For each day in a 12-year period (1998 to 2010) in Atlanta, GA, we estimated daily $PM_{2.5}$ source contributions from a Bayesian ensemble model that combined four source apportionment methods including chemical transport and receptor-based models. We fit Poisson generalized linear models to estimate associations between source-specific $PM_{2.5}$ concentrations and cardiorespiratory emergency department visits (n=1,598,117). We propagated uncertainty in the source contribution estimates through analyses using multiple imputation.

Results: Respiratory emergency department visits were positively associated with biomass burning and secondary organic carbon. For a 1 $\mu g/m^3$ increase in $PM_{2.5}$ from biomass burning during the past 3 days, the rate of visits for all respiratory outcomes increased by 0.4% (95% CI 0.0%, 0.7%). There was less evidence for associations between $PM_{2.5}$ sources and cardiovascular

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Computing Code and Data Availability: The computing code can be obtained by contacting the first or last author. The data are not available because they include medical information that cannot be released.

outcomes, with the exception of ischemic stroke, which was positively associated with most $PM_{2.5}$ sources. Accounting for the uncertainty of source apportionment estimates resulted, on average, in an 18% increase in the standard error for rate ratio estimates for all respiratory and cardiovascular emergency department visits, but inflation varied across specific sources and outcomes, ranging from 2% to 39%.

Conclusions: This study provides evidence of associations between $PM_{2.5}$ sources and some cardiorespiratory outcomes and quantifies the impact of accounting for variability in source apportionment approaches.

Keywords

$PM_{2.5}$; air pollution; fine particulate matter; source apportionment; uncertainty; respiratory health; cardiovascular health

INTRODUCTION

Fine particulate matter ($PM_{2.5}$) is a mixture of aerosols from heterogeneous sources such as biomass burning, gasoline-fueled vehicles, coal combustion, and atmospheric reactions. Despite evidence suggesting that the health effects of $PM_{2.5}$ may vary depending on pollutant source,[1-5] the vast majority of epidemiologic studies on $PM_{2.5}$ do not differentiate between particles from different sources.

While we cannot directly identify the sources of particles using ambient pollutant monitors, we can use models to relate ambient $PM_{2.5}$ back to its sources (i.e. emissions-based chemical transport models and receptor-based models). However, there is no gold standard method of source apportionment; different studies have applied different methods, all of which have limitations and source uncertainties. Even though uncertainty estimates are available for different methods, they are rarely accounted for in health studies of source-apportioned $PM_{2.5}$, which may underestimate the uncertainty of the resulting health associations of $PM_{2.5}$ sources.[6]

One approach for estimating the mass contribution from specific $PM_{2.5}$ sources is to combine the concentrations from several source apportionment methods. Compared to using only one source apportionment technique, ensemble averaging has been found to reduce variability in source attribution estimates, improve fit statistics,[7] and provide uncertainty estimates for comparison across methods.[8] In 2013, Balachandran and colleagues implemented a Bayesian-based ensemble source apportionment method for $PM_{2.5}$ using three receptor-based source apportionment models and one chemical transport model.[9] Rather than providing a single estimate of source concentration for each day, their method resulted in a distribution of daily source-specific $PM_{2.5}$ concentrations reflecting the source-specific uncertainty in the PM estimate on that day. This uncertainty can be propagated into the uncertainty estimates of the health associations in epidemiologic analyses. In 2015, Gass and colleagues used such an approach to estimate associations between ensemble-based $PM_{2.5}$ source contributions and pediatric asthma emergency department visits.[1] In this study, we extend the Gass et al. work to estimate associations between source-apportioned $PM_{2.5}$ concentrations and all-ages emergency department visits for a range of cardiovascular and

respiratory outcomes in Atlanta, Georgia during the period from 1 August 1998 to 23 December 2010.

METHODS

Data sources

Daily counts of emergency department visits, aggregated from individual-level data, with billing zip codes in the five-county (DeKalb, Clayton, Cobb, Fulton, Gwinnett) Atlanta metropolitan area for 1 August 1998 to 23 December 2010 (4,528 days of data) were created for the following health outcomes: asthma and wheeze, chronic obstructive pulmonary disease (COPD), pneumonia, upper respiratory infection, ischemic heart disease, dysrhythmia, congestive heart failure, ischemic stroke, as well as for combinations of these respiratory disease and cardiovascular disease outcomes. Outcomes were identified via ICD-9 codes (Table 1) listed as the primary reason for the emergency department visit. These data were collected from individual hospitals and the Georgia Hospital Association as part of the Study of Particles and Health in Atlanta (SOPHIA)[10-12] and capture over 90% of visits for the area. Emory University's Institutional Review Board approved this study.

We used daily measurements of pollutant gases (SO_2, CO, and NO_X), total $PM_{2.5}$ and $PM_{2.5}$ components (major ions, carbon fractions, and trace metals) from the Jefferson Street monitor in midtown Atlanta[13] for long-term $PM_{2.5}$ source apportionment. Daily Bayesian ensemble source-apportioned $PM_{2.5}$ concentration estimates at Jefferson Street were available for 1 August 1998 to 23 December 2010 for five primary $PM_{2.5}$ sources: biomass burning, primary coal combustion, dust/resuspended soil, diesel-fueled vehicles and non-road engines, and gasoline-fueled vehicles and engine sources; and one secondary source: secondary organic carbon. Ammonium sulfate, ammonium bisulfate, and ammonium nitrate are derived directly from the monitor measurements and were not examined because our focus was on chemical mass balance-derived $PM_{2.5}$ sources and the uncertainty inherent in quantifying source concentrations that cannot be directly measured. Organic carbon (OC) that is not attributed to one of the source categories above is expected to be largely secondary OC and is included to demonstrate the impact of separating the OC into different sources. The methodology for Bayesian-based ensemble source apportionment is described in detail elsewhere[1,9] and summarized below.

We estimated distributions of speciated $PM_{2.5}$ emissions distributions of two seasonal sets of ensemble-trained source profiles using Bayesian ensemble averaging, three receptor-based source apportionment methods (chemical mass balance with organic molecular markers, chemical mass balance with gas-based constraints, and positive matrix factorization),[14-16] and one chemical transport model (Community Multiscale Air Quality [CMAQ]).[17] We conducted the ensemble averaging and source profile estimation for 2 months representing summer and winter. These profiles are then used to estimate long-term $PM_{2.5}$ source impacts and uncertainties as measured by the root mean square error (RMSE) between each method's estimated source concentration and the average source concentration across methods (the ensemble average) using a Monte Carlo framework. An estimate of uncertainty for each source apportionment method for each source concentration for each day was calculated using a Bayesian framework accounting for the uncertainties in the RMSEs

themselves. Our approach is to describe uncertainties in source concentration estimates using posterior distributions derived from a data likelihood that assumes estimates from individual methods are normally distributed around the ensemble mean. To estimate daily source concentrations for use in this epidemiologic analysis, we sampled the seasonal source profiles 10 times for each day in the time series, and calculated deterministically 10 separate time series of daily source-apportioned $PM_{2.5}$ concentrations from these realizations using the chemical mass balance with gas constraints approach. We present the correlations between $PM_{2.5}$ source concentrations and specific $PM_{2.5}$ compounds (tracers) commonly used in $PM_{2.5}$ source apportionment in each technique and sometimes analyzed directly in relation to health outcomes.[5,18-20]

Statistical Analysis

We used Poisson log-linear models, with unconstrained distributed lags that accounted for overdispersion, to estimate associations between cumulative lag 0–2 (same day and 2 previous days) exposure to $PM_{2.5}$ sources and respiratory emergency department visits, and lag 0 (same day) exposure to $PM_{2.5}$ sources and cardiovascular visits. *A priori* we chose shorter lags for cardiovascular outcomes based on past evidence that the impact of pollution is more acute for these outcomes[21,22] than for respiratory outcomes.[12,23] Rate ratios were calculated by exponentiating the sum of the betas for the source of interest (3 betas for respiratory models ($RR_{0-2} = \exp(\beta_{lag\ 0}+\beta_{lag\ 1}+\beta_{lag\ 2})$) and 1 beta for cardiovascular models ($RR_0 = \exp(\beta_{lag\ 0})$)). The covariance matrix from the genmod procedure in SAS was used to calculate standard errors (SAS Institute Inc., Cary, NC). Rate ratios for lag 0–2 exposure estimate the impact of a cumulative 1 $\mu g/m^3$ increase of $PM_{2.5}$ source concentration on all 3 days; rate ratios for lag 0 exposure estimate the impact of only a same day 1 $\mu g/m^3$ increase. As a sensitivity analysis, we also calculated the association between lag 0–7 exposure to $PM_{2.5}$ sources and respiratory emergency department visits.

To control for temporal and meteorologic factors all models included parametric cubic splines with monthly knots,[24] cubic polynomials on lag 0 maximum temperature, interaction between lag 0 maximum temperature and season, cubic polynomials for the 2-day (lags 1–2) moving average of minimum temperature, cubic polynomials for lag 0 dewpoint, an indicator for each hospital taking the value 1 if the hospital contributes on a given day and 0 if the hospital does not contribute, an indicator representing season of emergency department visits (December–February [winter], March–May [spring], June–August [summer], September–November [autumn]), and indicators for day of week and holidays and their interaction with season.[25] We first estimated associations for each source and each outcome group, and then fit models that included all sources to control for potential confounding by other sources. For respiratory outcomes we also examined models adjusting for ozone due to previous findings of associations between ozone and respiratory outcomes.[26,27]

We fit ten models for each source–outcome combination, one for each of the 10 time series of daily source-apportioned $PM_{2.5}$ concentrations. The combined point estimate was calculated as an average of the 10 point estimates from the models. Imputation-corrected variance was calculated to incorporate the average variance from the ensemble runs and variance between ensemble run coefficients using the method presented by Rubin[28] and

described in more detail by Gass[1]. To quantify the increase in variance attributable to source method uncertainty, we calculated ratios of the imputation-corrected standard errors to the average standard error from the 10 model runs. To compare the results for specific sources to associations obtained for total $PM_{2.5}$ mass, we also fit one model for each outcome with the exposure of total $PM_{2.5}$ using the same lag structure and covariate control as for the source concentrations models.

We performed sensitivity analyses for select models based on our primary analysis results. We examined associations between GV and respiratory outcomes stratified by season. For models of BURN, we completed analyses to evaluate the extent to which days with extreme $PM_{2.5}$ levels (days above 25 $\mu g/m^3$ [88^{th} percentile]) were driving the analysis. Because some previous analyses have used $PM_{2.5}$ tracer species as indicators of sources instead of source apportionment models, we also examined adjusting for measured gasoline vehicle tracers from the monitor instead of model-based gasoline vehicle source concentrations estimated from the source apportionment in the all-sources model. All analyses were completed in SAS 9.4 (SAS Institute Inc., Cary, NC).

RESULTS

Over the 4,528 day time series, there was an average of 286 respiratory outcome emergency department visits and 67 cardiovascular outcome emergency department visits per day (Table 1). The individual outcome with the highest number of visits was upper respiratory infections, with an average of 163 visits per day, constituting over half of the all respiratory outcome group.

Due to missing data on source concentrations (due to incomplete $PM_{2.5}$ speciation on those days), $PM_{2.5}$, and meteorologic variables, 3,531 days of data were used in respiratory models and 4,060 days of data were used in cardiovascular models. Fewer days were available for respiratory outcomes because they required non-missing source concentrations for 3 days (lag 0, lag 1, lag 2), whereas cardiovascular outcomes only required concentrations for 1 day (lag 0).

Figure 1 shows the average proportion of total $PM_{2.5}$ mass contributed by each source and other measured components. The sources examined in this analysis make up 45 percent of total $PM_{2.5}$ mass; of these, the largest contributors in terms of mass were biomass burning (daily average of 2.74 $\mu g/m^3$) and secondary organic carbon (1.73 $\mu g/m^3$) and the smallest was primary coal combustion (0.10 $\mu g/m^3$). It should be noted that the “other” in Figure 1 is likely substantially the oxygen (and some hydrogen, nitrogen, and sulfur) in secondary organic matter. Descriptive statistics for the source-specific $PM_{2.5}$ concentrations are shown in Table 2. Within source, there were high correlations (r_s>0.7) between estimates in the 10 ensemble datasets (Table 2). Between sources, correlation was highest between diesel vehicles and non-road engines and secondary OC (r_s=0.41) and between biomass burning and gasoline vehicles and engines (r_s=0.39) (Table 3). Correlations between sources and tracers are provided in eTable 1.

Figure 2 presents associations between source concentrations and emergency department visits, with numeric results included in eTable 2. We note that the magnitude of a 1 $\mu g/m^3$ increase relative to each source's mass contribution differs markedly between sources. For example, for biomass burning, with the largest mass contribution, a 1 $\mu g/m^3$ increase represents 38% of its standard deviation, but for coal combustion a 1 $\mu g/m^3$ represents 725% of its standard deviation. Further, the interpretation of estimates differs for respiratory and cardiovascular outcomes due to the use of different lag structures (lag 0–2 for respiratory outcomes and lag 0 for cardiovascular outcomes).

Emergency department visits for respiratory outcomes were consistently and positively associated with biomass burning (Figure 2). For example, for a 1 $\mu g/m^3$ increase in biomass burning for lags 0–2 the rate of emergency department visits for all respiratory outcomes combined increased 0.4% (95% CI 0.0%, 0.7%) in the single-source model and 0.8% (95% CI 0.4%, 1.1%) when adjusting for other sources. Consistent positive associations across respiratory outcomes were also observed for secondary OC. Among the respiratory outcomes, association estimates were the most uncertain for COPD due to the small number of emergency department visits (an average of 12 per day). Rate ratios for the association between gasoline vehicle source emissions and respiratory outcomes were generally below the null in single-source models and further below the null when adjusting for other sources. Results for the associations between lag 0–7 $PM_{2.5}$ sources and respiratory emergency department visits are provided in eTable 3 and show similar patterns but wider confidence intervals. Respiratory model results were similar when adjusting for ozone (results not shown).

Compared to respiratory outcomes, we observed less evidence of associations between $PM_{2.5}$ source concentrations and cardiovascular outcomes. One exception was elevated rate ratios for the association between several $PM_{2.5}$ sources and ischemic stroke. For example, the rate ratio for the association between an increase of 1 $\mu g/m^3$ in same-day diesel vehicle emissions and ischemic stroke was 1.013 (95% CI 1.001, 1.025) in single-source models, and 1.009 (95% CI 0.995, 1.023) when adjusting for other sources. Rate ratios were elevated for associations between secondary OC and cardiovascular outcomes, but with less evidence of an association than between secondary OC and respiratory outcomes.

For the same mass concentrations (1 $\mu g/m^3$), the magnitude of associations between total $PM_{2.5}$ and respiratory and cardiovascular outcomes were smaller than for $PM_{2.5}$ sources. We observed positive associations between total $PM_{2.5}$ and asthma and wheeze (RR (95% CI): 1.001 (1.000, 1.002) per 1 $\mu g/m^3$), upper respiratory infections (RR (95% CI): 1.001 (1.000, 1.002)), and the combined respiratory outcome group (RR (95% CI): 1.001 (1.000, 1.002)) (Figure 3). We also observed a positive association between total $PM_{2.5}$ and ischemic stroke (RR (95% CI) 1.002 (1.000, 1.003)) (Figure 3). Results were more consistent with no association for other cardiovascular outcomes.

The impact on standard errors due to accounting for the uncertainty in $PM_{2.5}$ source apportionment techniques through multiple imputation varied notably across sources (Table 4). Across all sources, for all respiratory and cardiovascular emergency department visits, the average increase in SE was 18%. Accounting for this uncertainty had the smallest impact

on associations with dust emission sources, where the SE increased 2.8% for the all respiratory outcome and 2.0% for the all cardiovascular outcome. The greatest increase in SE when accounting for between model uncertainty was seen for biomass burning and secondary OC reflecting more variability across methods in the estimated source concentrations and resulting health effect estimates from the different source apportionment methods (increase in SE for all respiratory emergency department visits 34.1% for biomass burning and 38.7% for secondary OC). When grouping outcomes, the relative inflation of the standard error was generally larger for all respiratory outcomes than for all cardiovascular outcomes, but this pattern was not seen within disease subgroups (eTable 4).

For coal combustion, dust, diesel vehicle, and secondary OC sources, results from single-source and all-source models were similar. For biomass burning, adjusting for other $PM_{2.5}$ sources increased rate ratios for respiratory outcomes compared to results from single-source models. The opposite was true for gasoline vehicles where adjusting for other sources resulted in smaller rate ratios for respiratory outcomes than the already below the null rate ratios from single-source models. Inclusion of biomass burning and gasoline vehicles in the same model drove the higher estimates for biomass burning and the lower estimates for gasoline vehicles. Analyses stratified by season showed that this sensitivity of results based on inclusion of biomass burning and gasoline vehicles together was most pronounced during winter and autumn. We explored adjusting for zinc, a tracer of gasoline vehicle sources (eTable 1), in biomass burning models and observed similar results, i.e., that in comparison to single-source models point estimates for biomass burning increased and zinc estimates decreased. Additional analyses on single-source biomass burning models showed that high-PM days (days above 25 $\mu g/m^3$) were not driving the biomass burning associations (the RR for all respiratory outcomes was unchanged when excluding 384 days where $PM_{2.5}$ is more than 25).

DISCUSSION

Due to the relevance of examining $PM_{2.5}$ source impacts for regulation, examining impacts by source is of interest[6], but the uncertainty of source apportionment methods is rarely accounted for in the epidemiologic analysis. In this 12-year time series, we applied four source apportionment approaches to obtain uncertainty distributions of daily source specific $PM_{2.5}$ concentrations, with wider distributions on days where the source apportionment methods showed more disagreement. We then used these source-specific $PM_{2.5}$ concentration estimates in epidemiologic analyses that propagated the uncertainty of the source apportionment techniques. Our results allow direct comparison of the associations of 1 $\mu g/m^3$ of $PM_{2.5}$ from different sources. They provide evidence for associations between some respiratory outcomes and biomass burning and secondary organic carbon source concentrations in the past 3 days and respiratory outcomes, and associations between several $PM_{2.5}$ sources and ischemic stroke on the same day. Compared to an approach not accounting for the variability between source apportionment methods, our approach widened confidence intervals around point estimates, more accurately representing the uncertainty in these model-based approaches to source apportionment.

A contribution of this approach is to add to our insight regarding the possible impacts of different source apportionment techniques on associations between $PM_{2.5}$ sources and health outcomes. The ensemble approach accounts for the fact that there is no preferred source apportionment technique. The variability in estimated health associations between the four source apportionment methods that we used is directly incorporated into our results. Accounting for this uncertainty results in a more accurate estimate of the true uncertainty in $PM_{2.5}$ source-specific health effects. Previous studies have compared associations based on different source apportionment approaches,[5] but to our knowledge, there is only one other method presented in the literature that has a formal incorporation of uncertainty from multiple source apportionment techniques in health models. The other method, presented by Kioumourtzoglou and colleagues, uses a block bootstrapping approach to combine analyses with inputs from two receptor-based source apportionment methods (both factor-analytic approaches).[29] Our approach differs from theirs in our accounting for between-method uncertainty and the broader range of source apportionment methods combined in our ensemble, which include two chemical mass balance approaches (utilizing empirically derived source profiles), one factor analytic approach, and an emissions-based chemical transport model.

In this analysis, we propagated the uncertainty in daily estimated source concentration attributable to source apportionment technique through the epidemiologic models by repeat sampling (10 imputations) from a distribution of source concentrations on each day. When there was more disagreement among methods about source concentration on a given day, that sampling distribution had a higher variance, and when there was less disagreement among methods, that distribution had lower variance. The amount of added uncertainty in the estimated associations (shown in Table 4, eTable 4) due to accounting for this variability between source apportionment techniques varied by source because the source apportionment methods agreed more for some sources than others. For example, for dust and resuspended soil there was high correlation between estimates between the different ensemble runs (Spearman's r_s=0.976, Table 2) reflecting strong agreement between the source apportionment techniques. This high correlation translated to little additional uncertainty when accounting for variability between the health effect estimates, with an inflation of the standard error of approximately 2% for all respiratory outcomes and all cardiovascular outcomes (Table 4). For other sources there was less agreement in daily source concentrations between the ensemble estimates, which in turn led to more uncertainty and higher standard errors. For example, for biomass burning the Spearman correlation between ensemble runs was 0.747 (Table 2), and accounting for the variability between point estimates increased the standard error by 34% for all respiratory outcomes and 16% for all cardiovascular outcomes (Table 4). The average inflation in standard errors shown in Table 4 was 18%. Differences in variance inflation by outcome (for a given source) could be explained by differences in sample size between outcomes, with smaller case groups having larger standard errors to begin with and thus smaller relative increase due to the added uncertainty. Additionally, differential impacts on precision by outcome may reflect differences in associations between each source apportionment method and outcome; for example, if the individual source apportionment methods all yielded identical associations

with an outcome, then variability in the ensemble weighting across methods should not inflate the standard errors for this outcome.

Our approach accounts for uncertainty from using an ensemble of source apportionment methods, but does not account for the uncertainty due to exposure measurement error of the pollution components themselves. We used measurements from a single monitoring site for all receptor-based methods, which is not representative of PM for spatially heterogeneous sources. As a result, the measurement error could differ by source in our analysis. Associations of outcomes with sources with more spatial homogeneity (e.g. SOC) may be more strongly detected due to less spatial error in exposure estimates. We note that one of the four source apportionment approaches is emissions-based, and thus would be less affected by measurement error at the monitors. These limitations apply to all of our results regardless of direction and magnitude of association.

Results from this study add to the growing evidence that biomass burning is of interest for respiratory health. Examining the association between biomass burning and respiratory disease emergency department visits in four U.S. cities, Krall and colleagues observed elevated relative risks for lag 0 to lag 3 exposure in many of the cities with magnitudes that were often larger than those of the other sources examined.[2] Ostro and colleagues demonstrated an excess risk of respiratory disease emergency department visits in California with lag 0 and lag 1 biomass burning, but not with lag 2.[3] Additional time series studies have observed elevated rate ratios between biomass burning and respiratory disease and pediatric asthma visits in Atlanta, but provide less evidence of an association.[1,5] There are also studies emerging suggesting that wildfire smoke, one type of biomass burning, is particularly harmful for respiratory disease.[30] We scaled results for all sources, and total $PM_{2.5}$ mass, to a 1 $\mu g/m^3$ increase despite variable distributions for each source concentration. Our results suggest that for all respiratory outcomes the per unit impact of biomass burning is more harmful than the per unit impact of total $PM_{2.5}$ (RR (95% CI) 1.004 (1.000, 1.007), 1.001 (1.000, 1.002) respectively). Primary coal combustion and dust/resuspended soil were relatively small contributors to the total $PM_{2.5}$ concentration (contributing 0.9% and 2.4% respectively) resulting in high levels of uncertainty in estimates per 1 $\mu g/m^3$ increase. Variability in mass due to these sources may be dwarfed by the larger contributors to $PM_{2.5}$. We note that correlations between the sources we examined and the other major contributors to $PM_{2.5}$ (ammonium sulfate, ammonium bisulfate, and ammonium nitrate) were low, suggesting that these other $PM_{2.5}$ components excluded from the “all source” models are unlikely to be confounders of the observed associations. With the exception of associations between several sources and ischemic stroke, we observed little evidence of associations between same-day $PM_{2.5}$ source concentrations and cardiovascular disease outcomes.

The observed below the null rate ratios for the association between gasoline-fueled vehicles and respiratory outcomes were unexpected; these associations were furthest below the null when adjusting for biomass burning, the source with which gasoline-fueled vehicles was most highly correlated (r_s=0.39). Results were similar from models including elemental zinc instead of gasoline-fueled vehicles, which was highly correlated with the ensemble average of gasoline-fueled vehicles (r_s=0.8) and is a tracer of tailpipe emissions. Winter and autumn were the seasons where adjusting for biomass burning had the most impact on gasoline-

fueled vehicle results. During these seasons gasoline fueled vehicle contributions are high (due to mixing height and weather influences) and there may be overlap of gasoline-fueled vehicle and biomass burning source impact estimates, which may partially explain these results. Additionally, since there is substantial anthropogenic organic carbon associated with both gasoline-fueled vehicle and biomass burning sources, they may be more difficult to differentiate as cleanly as other sources. With the exception of associations with ischemic stroke, and to some degree asthma for diesel-fueled vehicles, we observed little evidence of associations between mobile source $PM_{2.5}$ (diesel-fueled vehicles and gasoline-fueled vehicles) and emergency department visits. Evidence on the health effects of these sources from previous studies is inconsistent with some evidence suggesting associations with cardiovascular and respiratory emergency department visits, while others' results have been less convincing.[1-3,5]

In this 12-year time series we used concentration estimates from four source apportionment methods to estimate associations between $PM_{2.5}$ source concentrations and respiratory and cardiovascular emergency department visits in Atlanta. Our uncertainty propagation analytic framework allowed us to account for variability between source apportionment methods and to take into account the uncertainty of each method when determining the weighting in the ensemble averaging. Our results indicate potential associations between biomass burning and respiratory outcomes and between a number of sources and ischemic stroke.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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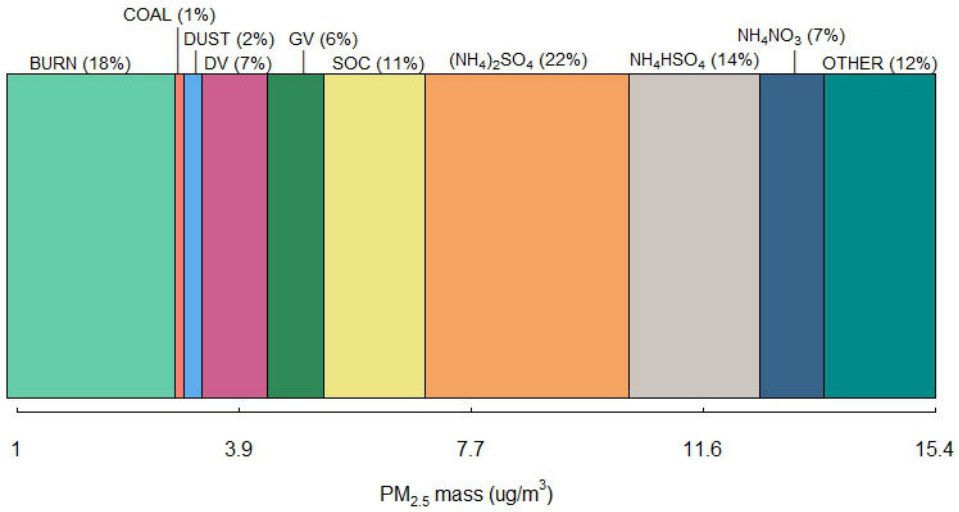
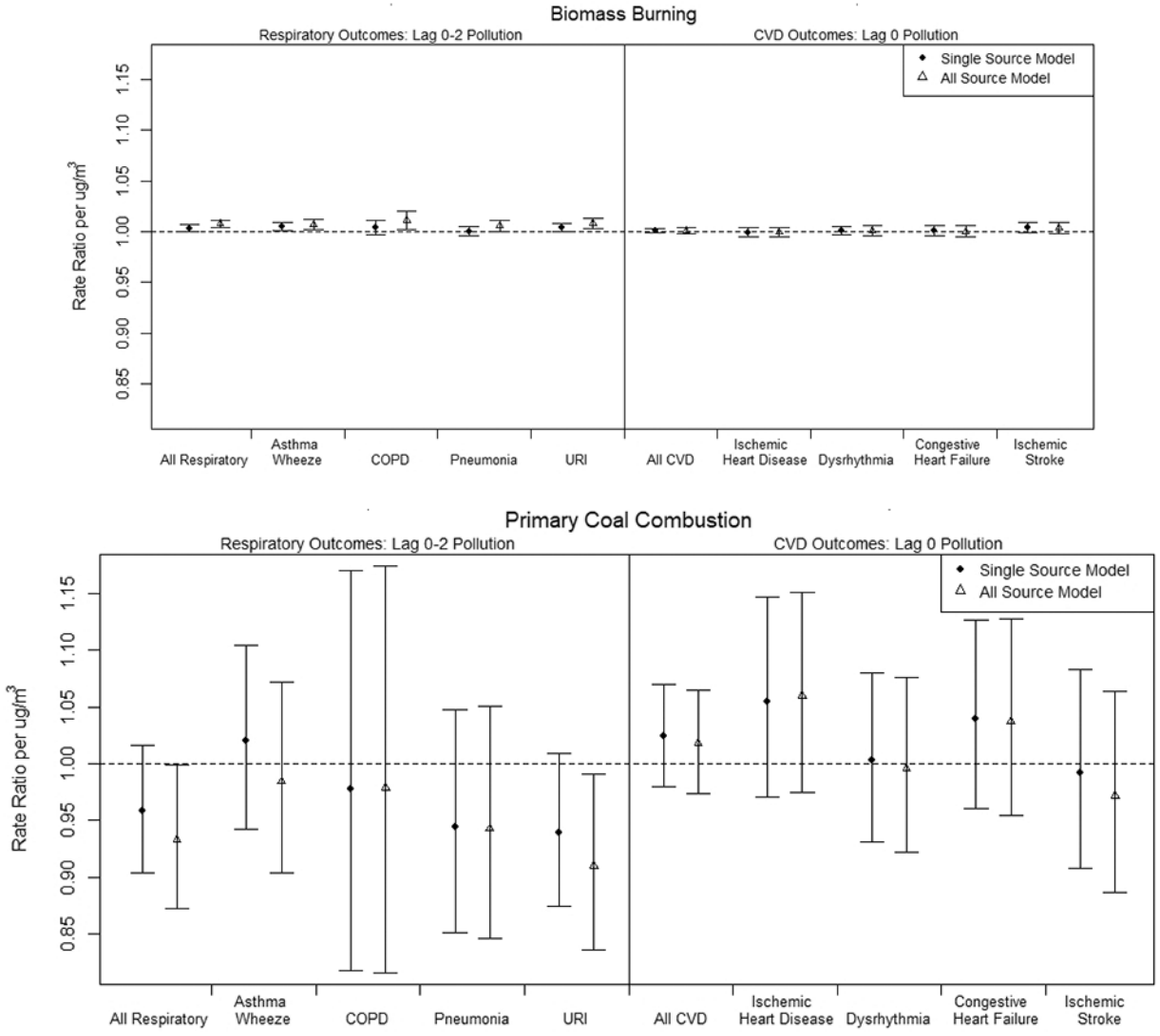
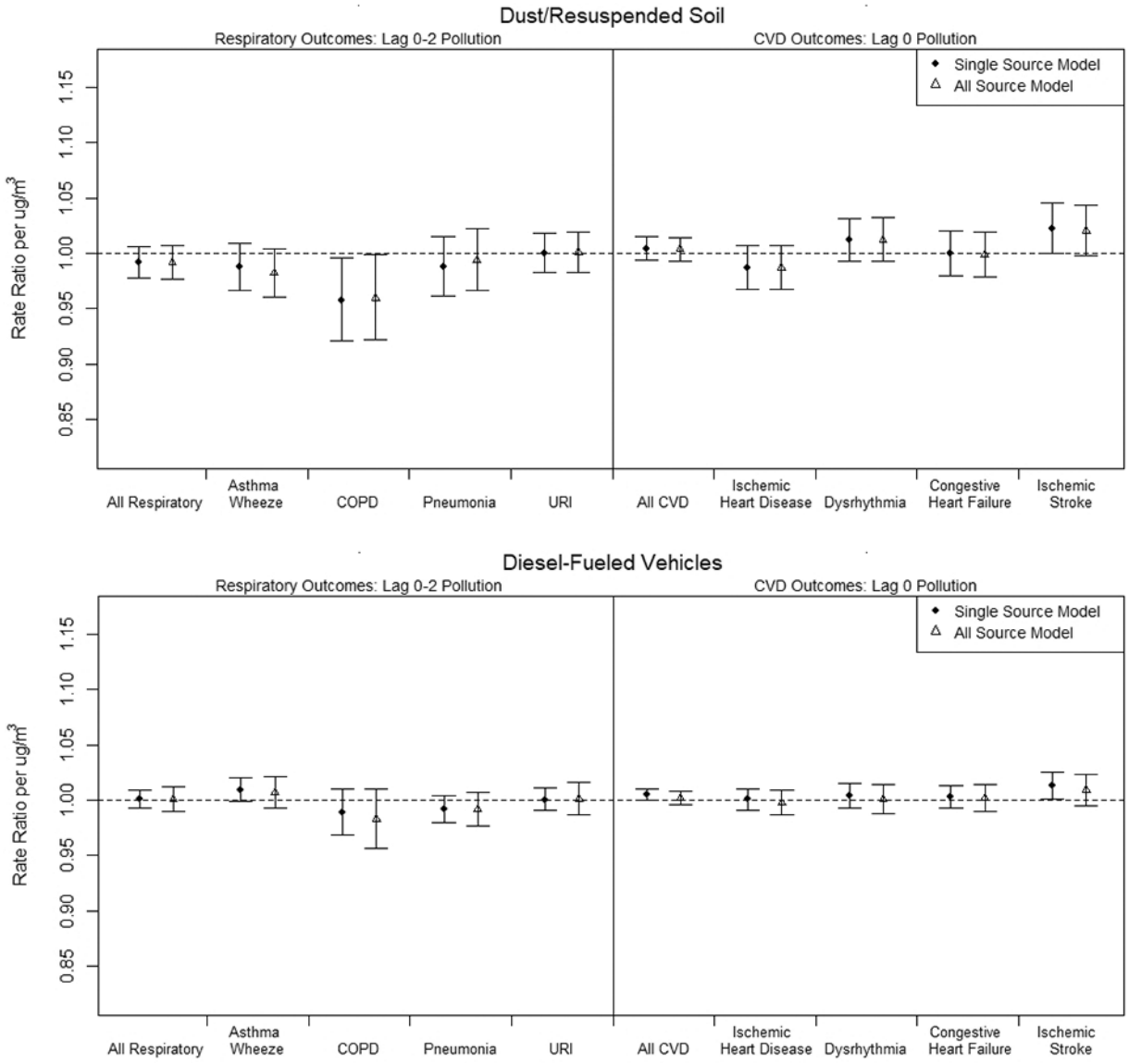


Figure 1.

Contribution of sources to $PM_{2.5}$ mass (averaged across the study period) [Abbreviations: BURN, biomass burning; COAL, primary coal combustion; DUST, dust/resuspended soil; DV, diesel-fueled vehicles; GV, gasoline-fueled vehicles; SOC, secondary organic carbon; $(NH_4)_2SO_4$, ammonium sulfate; NH_4HSO_4, ammonium bisulfate; NH_4NO_3, ammonium nitrate; $PM_{2.5}$, fine particulate matter]





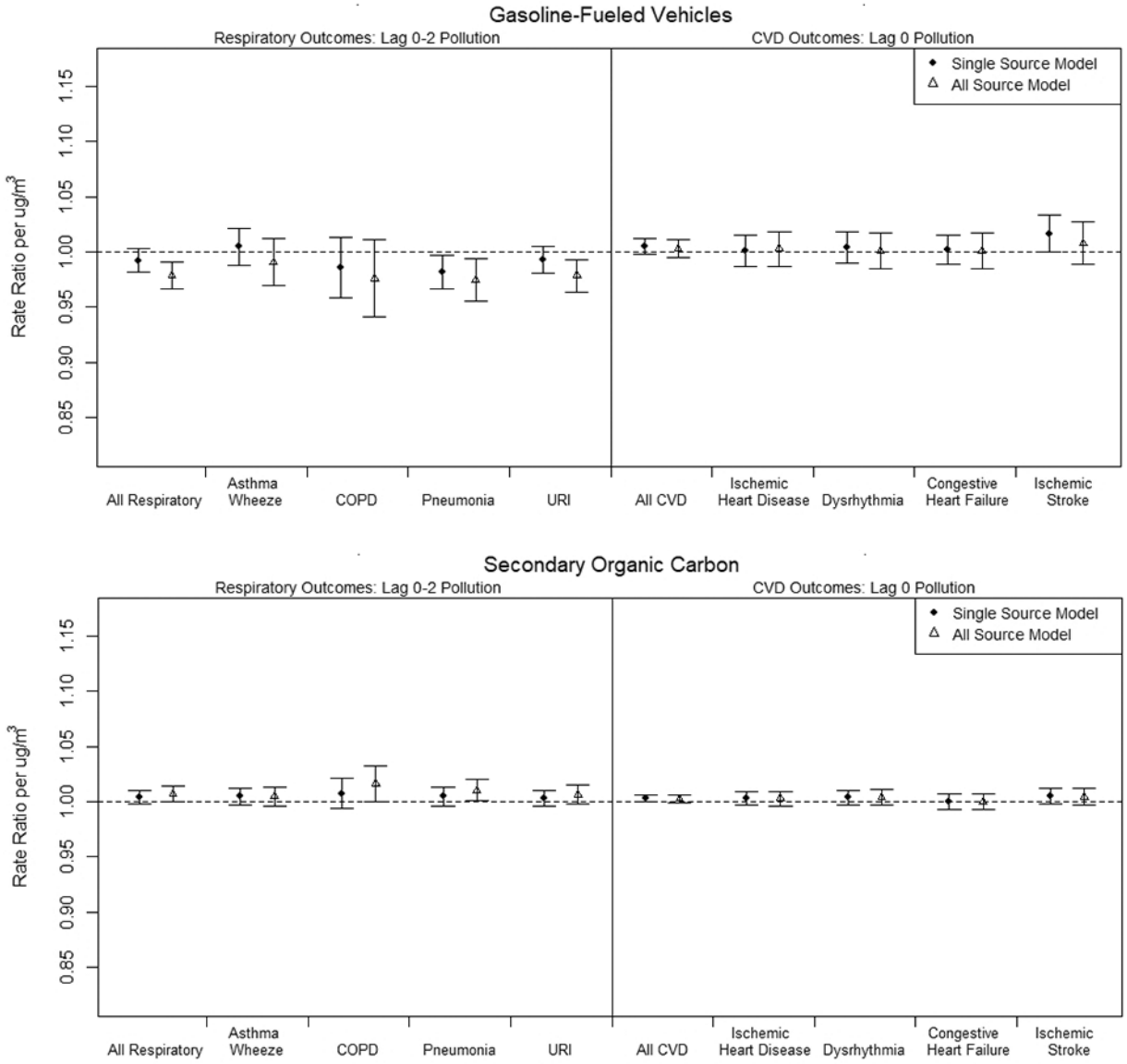


Figure 2.

Rate ratios for 1 μg/m^3 increase in source specific $PM_{2.5}$ with emergency department visits: lag 0-2 for respiratory outcomes and lag 0 for cardiovascular disease (CVD) outcomes, results from single- and all-sources models

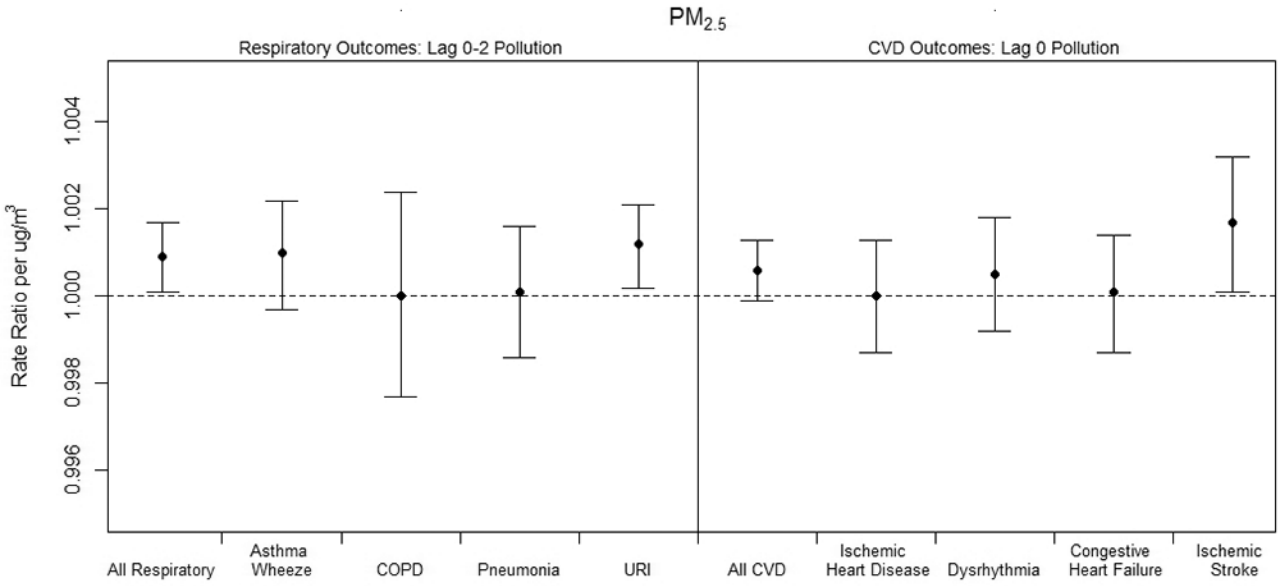


Figure 3.

Rate ratios for 1 μg/m^3 increase in $PM_{2.5}$ with emergency department visits: lag 0-2 for respiratory outcomes and lag 0 for cardiovascular disease (CVD) outcomes

Table 1.

ICD-9 codes and summary statistics of emergency department visits for respiratory and cardiovascular outcomes. SD indicates standard deviation.

Outcome	ICD-9 codes [only primary codes used]	Visits per day		
		Minimum	Mean (SD)	Maximum
Respiratory				
All respiratory	460-465, 466.0, 477, 480-486, 491-492, 493, 466.1, 466.11, 466.19, 496, 786.07	86	286.0 (102.8)	763
Asthma and wheeze	493, 786.07	13	64.7 (25.2)	191
Chronic obstructive pulmonary disease	491-492, 496	1	12.4 (5.2)	37
Pneumonia	480-486	6	35.7 (15.2)	108
Upper respiratory infection	460-465, 466.0, 477	44	162.5 (62.5)	467
Cardiovascular				
All cardiovascular	410-414, 427, 428, 433-437, 440, 443-445, 447	20	66.9 (18.7)	129
Ischemic heart disease	410-414	3	18.0 (5.4)	40
Dysrhythmia	427	3	17.2 (5.6)	40
Congestive heart failure	428	1	17.6 (7.8)	51
Ischemic stroke	433-437	0	12.8 (5.0)	34

Table 2.

Summary statistics for source-specific $PM_{2.5}$ concentrations, Atlanta, Georgia, August 1, 1998 - December 23, 2010[a]

Source	Minimum μg/m^3	Median μg/m^3	Mean μg/m^3	Maximum μg/m^3	Standard deviation	Interquartile range width	Correlation between ensemble runs[b]
Biomass burning	0.000 (0.000)	1.877(0.019)	2.737(0.012)	32.103(5.933)	2.642(0.051)	2.689(0.030)	0.747
Primary coal combustion	0.000 (0.000)	0.097(0.001)	0.134(0.002)	1.687(0.273)	0.138(0.003)	0.138(0.002)	0.722
Dust/resuspended soil	0.000 (0.000)	0.250(0.001)	0.373(0.001)	9.378(1.562)	0.474(0.010)	0.248(0.002)	0.976
Diesel vehicles	0.000 (0.000)	0.883(0.012)	1.142(0.009)	12.343(1.259)	1.089(0.020)	1.091(0.015)	0.741
Gasoline vehicles	0.008 (0.009)	0.694(0.010)	0.858(0.004)	10.534(0.884)	0.759(0.007)	0.706(0.009)	0.783
Secondary organic carbon	0.000 (0.000)	1.453(0.012)	1.727(0.007)	27.578(1.071)	1.681(0.011)	2.097(0.032)	0.745

Results presented as mean(standard deviation)

[a] Averaged across 10 ensemble runs.

[b] Mean Spearman correlation calculated from all pairwise runs

For comparison, the distribution of total $PM_{2.5}$, in μg/m, is: minimum 1.060, median 13.970, mean 15.473, maximum 72.560, standard deviation 7.936, interquartile range 9.915.

Table 3.

Spearman correlation coefficients between source-specific $PM_{2.5}$ and total $PM_{2.5}$ concentrations (averaged across 10 ensemble runs).

Pollutant	BURN	COAL	DUST	DV	GV	SOC	$PM_{2.5}$
BURN	1.00						
COAL	0.24	1.00					
DUST	-0.04	0.15	1.00				
DV	0.06	0.23	0.23	1.00			
GV	0.39	0.09	0.12	0.23	1.00		
SOC	-0.44	0.04	0.30	0.41	-0.09	1.00	
$PM_{2.5}$	0.18	0.23	0.40	0.46	0.33	0.47	1.00

Abbreviations: BURN, biomass burning; COAL, primary coal combustion; DUST, dust/resuspended soil; DV, diesel-fueled vehicles; GV, gasoline-fueled vehicles; SOC, secondary organic carbon; $PM_{2.5}$, fine particulate matter.

Table 4.

Ratios of the imputation-corrected SE (that accounts for between imputation uncertainty) to the average SE from the ensemble runs from single-source models for all respiratory and all cardiovascular ED visits

	Inflation of SE	
Source	**All respiratory**	**All cardiovascular**
Biomass burning	1.341	1.164
Primary coal combustion	1.206	1.260
Dust/resuspended soil	1.028	1.020
Diesel-fueled vehicles	1.333	1.112
Gasoline-fueled vehicles	1.248	1.068
Secondary organic carbon	1.387	1.044

SE = standard error. ED = emergency department. Inflation of SE calculated as the SE accounting for both between- and within-model uncertainty divided by the average of the SEs from the 10 models for each source-outcome combination (SE accounting for only within-model uncertainty).