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Source-specific pollution exposure and associations with pulmonary response in the Atlanta Commuters Exposure Studies

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Abstract

Concentrations of traffic-related air pollutants are frequently higher within commuting vehicles than in ambient air. Pollutants found within vehicles may include those generated by tailpipe exhaust, brake wear, and road dust sources, as well as pollutants from in-cabin sources. Source-specific pollution, compared to total pollution, may represent regulation targets that can better protect human health. We estimated source-specific pollution exposures and corresponding pulmonary response in a panel study of commuters. We used constrained positive matrix factorization to estimate source-specific pollution factors and, subsequently, mixed effects models to estimate associations between source-specific pollution and pulmonary response. We identified four pollution factors that we named: crustal, primary tailpipe traffic, non-tailpipe traffic, and secondary. Among asthmatic subjects (N=48), interquartile range increases in crustal and secondary pollution were associated with changes in lung function of −1.33% (95% confidence interval (CI): −2.45, −0.22) and −2.19% (95% CI: −3.46, −0.92) relative to baseline, respectively. Among non-asthmatic subjects (N=51), non-tailpipe pollution was associated with pulmonary response only at 2.5 hours post-commute. We found no significant associations between pulmonary response and primary tailpipe pollution. Health effects associated with traffic-related

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pollution may vary by source, and therefore some traffic pollution sources may require targeted interventions to protect health.

Keywords

source apportionment; pulmonary health; air pollution; traffic pollution; commuting; on-road exposures

1. INTRODUCTION

Short-term exposure to traffic-related air pollution has been associated with adverse health outcomes including mortality (1), hospitalizations (2,3), and pediatric asthma (4). On average, US adults spend approximately one hour within a vehicle each day (5), and previous studies have found in-vehicle concentrations of harmful pollutants, such as fine particulate matter ($PM_{2.5}$), frequently exceed ambient concentrations (6–9). Regulation of traffic pollution has focused on reducing tailpipe emissions (6); however, emissions from other traffic-related sources may also be associated with adverse health outcomes. As tailpipe regulations continue to result in lower combustion-related primary emissions from vehicles, pollutants generated by processes such as tire wear and brake wear will grow in their proportion of total mobile source pollution (6). Determining whether tailpipe and non-tailpipe traffic-related pollution are both individually associated with adverse health outcomes will help develop more targeted regulation to better protect public health.

Traffic pollution is a highly heterogeneous mixture containing both volatile and semi-volatile gases, as well as organic and inorganic particulate species that contribute to total ambient particulate matter (PM). Some traffic-related chemical components of PM, such as organic carbon (OC), elemental carbon (EC), and zinc, have been implicated in epidemiologic studies as either direct or indirect indicators of adverse health outcomes (10–12), though results have not been consistent across studies examining PM components (13,14). Transition metals, in particular, have been implicated as potential chemical drivers of internal oxidative stress and inflammation, both biological processes hypothesized to play a role in acute adverse response to air pollution (15,16). Furthermore, short-term exposure to pollution from diesel and gasoline vehicles has been associated with pediatric asthma emergency department visits (4) and asthma symptoms (17).

Traffic-related pollutants can be generated by gasoline tailpipe emissions, diesel emissions, road dust, tire wear, or other sources, and each of these sources can be characterized by the pollutants emitted. For example, combustion of fossil fuels associated with both gasoline and diesel engines emits OC and EC (6). Tire wear and brake wear contribute metal particles including zinc and iron to total traffic-related pollution (18,19). Resuspended road dust may also contribute to traffic-related pollution and contains both transition metal species and crustal particles including aluminum and calcium (6,19).

Conducting studies of source-specific health effects is challenging because concentrations of source-specific pollution are generally not directly measured. Commonly, source categories are estimated by applying source apportionment models to concentrations of multiple

pollutants measured at stationary monitors, also known as receptor modeling. Source apportionment approaches have been extensively applied in epidemiologic studies of ambient pollution to estimate unobserved factors that may be indicative of one or more pollution sources (2,12,20,21). When applied to ambient monitoring data, these models represent observed daily pollutant concentrations as the product of daily source-specific pollution concentration and the amount each pollutant contributes to each source factor. Factors obtained from source apportionment models are named (e.g. road dust or tailpipe emissions) based on the pollutant combination associated with each factor, though may truly represent one or more known sources of pollution.

In studies of ambient $PM_{2.5}$, often only one or two traffic-related sources, such as road dust or gasoline emissions, can be separated using source apportionment models applied to available data (4,12,22). Moreover, pollutant distributions of traffic-related components (e.g. OC and EC) are frequently spatially heterogeneous (23), and therefore data from ambient monitors located far from roadways may not represent on-road traffic pollution well. Instead of estimating sources of traffic pollution using ambient monitoring data, another approach is to measure pollution closer to the source, such as within vehicles, and to incorporate prior information in source apportionment models that can help distinguish sources that emit some of the same pollutants. This approach will also help to determine what non-traffic pollution sources, such as secondary sources that are mixtures of pollutants formed through chemical reactions in the air, may impact health during commuting.

In this work, we estimated associations between source-specific pollution factors and pulmonary response in a panel study of commuters. To estimate source factors corresponding to traffic-related pollution, we incorporated prior information in a constrained source apportionment model.

2. MATERIAL AND METHODS

2.1 Data

We used pollution and health data collected as part of the Atlanta Commuters Exposure (ACE) studies (24,25). Briefly, the ACE-1 and ACE-2 studies measured in-vehicle pollution during scripted two-hour commutes. In ACE-1, 42 adults completed 81 scripted highway commutes, where most participants completed two commutes on two separate days to allow repeated measure assessment of highway exposure. In ACE-2, 59 adults completed scripted highway commutes and a subset (n=29) also completed a scripted surface street commute. To control for possible diurnal patterns in traffic and pulmonary response, all commutes were scheduled during the two-hour morning rush hour period (~7AM-9AM). The pollutants measured during each commute consisted of 25 chemical components of $PM_{2.5}$ including concentrations of metals such as zinc, lead, and nickel, as well as OC, water soluble OC (WSOC), and black carbon (BC), which is a surrogate measure of EC (Supplementary Material, Table S1). In addition to $PM_{2.5}$ chemical components, we also included particle-bound polycyclic aromatic hydrocarbons (pbPAH), particle number concentration (PNC), and noise. Although noise is not traditionally used to identify air pollution sources, we chose to include it as an additional means of differentiating sources associated with sound (e.g. vehicle emissions) from background ambient pollution. We did not include total $PM_{2.5}$ mass

because individual $PM_{2.5}$ component concentrations are frequently correlated with total $PM_{2.5}$ mass, which can lead to poor source estimates. Details about the pollution data collection methodology, including information about the filters, can be found in the supplementary material of Greenwald et al., (2014) (24).

Prior to and following each two-hour commute, pulmonary response was measured on each participant including exhaled nitric oxide (eNO) in parts per billion (ppb), a measure of oxidative stress and a biomarker for airway inflammation (26–28), and lung function (forced expiratory volume (FEV1) and forced vital capacity (FVC)). FEV1 and FVC were adjusted for age, sex, and race and were reported as percent (%) predicted values (29). FEV1 and FVC were measured using an OHD KoKo spirometer (Occupational Health Dynamics, Birmingham, AL, USA) and eNO was measured using a portable NIOX MINO analyzer (Aerocrine, New Providence, NJ, USA) (25). Health measurements were obtained for each participant at baseline as well as at hourly intervals following the two-hour commute (0 hours (baseline), 2.5, 3.5, 4.5, 5.5). We also collected gender, age, body mass index (BMI), pre-commute cortisol, and asthma status for each commuter. The details of the ACE-1 (24,25) and ACE-2 studies (*Golan et al., in preparation*) are described elsewhere. All participants provided informed consent prior to enrollment and both the ACE-1 and ACE-2 studies were approved by the Emory University Institutional Review Board.

2.2 Traffic-related pollution estimation

We first imputed missing values in the pollutant data with sequential regression (30), which uses a series of regression models to predict missing values starting with the pollutant with the least missingness and ending with the pollutant with the most missingness. Sequential regression can incorporate categorical variables, such as commute type.

Next, we employed positive matrix factorization (PMF) (31) to estimate source-specific pollution matrices $\boldsymbol{G}$ and $\boldsymbol{F}$ that form observed pollutant concentrations x_{ip} for $\boldsymbol{I}$ observations for P pollutants. PMF minimizes Q where

$$Q = \sum_{i=1}^{I} \sum_{p=1}^{P} \left(\frac{x_{ip} - \sum_{l=1}^{L} g_{il} f_{lp}}{u_{ip}} \right)^2 \quad (1)$$

for L source categories subject to $g_{il} \geq 0$ and $f_{lp} \geq 0$ for all i,l,p. In our dataset, each row i of the matrix $\boldsymbol{G}$ represents impacts of sources on observation i, and each row l of $\boldsymbol{F}$ is a source profile that represents the composition of source category l, specifically how much each pollutant p contributes to that source. For health studies, the columns of $\boldsymbol{G}$, which correspond to each source category, can be associated with health outcomes. The $\boldsymbol{u}_{ip}$ are observation- and pollutant-specific uncertainties that downweight observations with large errors.

A constrained PMF approach that incorporates prior information can help to resolve source factors that better match known sources of pollution. PMF resolves source factors using equation (1) as the basic source apportionment model and can also incorporate constraints

by minimizing $Q + Q^{aux}$, where Q is defined by equation (1) and Q^{aux} constrains elements of $\boldsymbol{F}$ or $\boldsymbol{G}$. To develop the constraints in Q^{aux}, we selected those pollutants known to be emitted, or known to not be emitted, by each source based on previous studies of traffic-related pollution (6,18,19). We used inequality constraints that "pull" these pollutants up or down in the source profiles $\boldsymbol{F}$ and these penalties generally help to obtain traffic pollution factors that better match information about the sources (32). The constrained PMF model was fitted using the multilinear engine (ME-2) (33). The results from the PMF approach were scaled based on observed $PM_{2.5}$ to represent $\mu g/m^3$. More information about our constrained PMF approach can be found in the Supplementary Material, Part A.

We included observation- and species-specific uncertainties $\boldsymbol{u}_{ip}$ in equation (1) using the PMF framework that computes uncertainties based on the concentrations x_{ip} (34). We compared estimated sources from models for $L=4$, $L=5$, and $L=6$ source factors. To select the final source apportionment model, we compared the PMF results to sources known to be associated with traffic pollution, namely brake wear, tire wear, road dust, crustal pollution, and primary tailpipe emissions, as well as sources identified in ambient air, such as secondary sulfate.

2.3 Estimating associations with pulmonary response

We estimated associations between source-specific pollution and pulmonary response using the PMF-estimated source factors. We applied longitudinal mixed effects models controlling for temporal trends in pulmonary response post-exposure,

$$y_{jc}(t) = \beta_0 + g_{jcl}\beta_1 + x_j\beta_2 + g_{jcl}x_j\beta_3 + t\,\beta_4 + \sum_{m=1}^{5} z_{jcm}\gamma_m + v_{jc}(t) + \varepsilon_{jc}(t) \quad (2)$$

where $y_{jc}(t)$ is the difference in health from baseline (e.g., FEV) for individual subject j during commute c at post-exposure time point t, for $t = 2.5, 3.5, 4.5, 5.5$ hours post-commute. We included g_{jcl}, the estimated pollution concentration from source category l for subject j during commute c. We use c and j to represent commutes nested within individuals respectively, instead of observations i as in equation (1), to explicitly indicate that commutes are nested within individuals. This notation differs slightly from equation (1), which was written to be consistent with the source apportionment literature. We also included asthma status (asthmatic or non-asthmatic) for subject j as x_j. We allowed an interaction between pollution and asthma status to account for possible differential health effects on asthmatic compared with non-asthmatic subjects. Other potential confounders included as z_{jcm} were commute type (surface street or highway), age, gender, pre-commute cortisol, and BMI.

The random effects $v_{jc}(t) = b_{j0} + b_{j1}t + u_{jc0} + u_{jc1}t$ included both random intercepts and (time) slopes for each subject ($b_{j0} + b_{j1}t$) and each commute within subject ($u_{jc0} + u_{jc1}t$) for time t. These account for differences between subjects and between commutes within subjects in $y_{jc}(t)$ at the first time point, as well as differences over time. The last term, $\varepsilon_{jc}(t)$, represents measurement error. The main models were fitted separately for each health outcome and each source category l. We also fitted multi-source models by incorporating multiple source factors simultaneously into equation 2. To examine nonlinear associations

between source-specific pollution exposure and pulmonary response over time, we fitted random intercept models with interactions between source, asthma status, and a categorical time variable.

To determine the sensitivity of our results to the imputed data, we compared source factors estimated using imputed data to those using complete case data only. We also determined whether source factors were similar (1) using data from ACE-1 and ACE-2 separately and (2) excluding noise, pbPAH, WSOC, and PNC to determine whether these measures had an impact on the estimated source factors. Last, we compared source factors estimated using the constrained PMF approach and using unconstrained PMF, which does not include prior information and may not estimate source factors that match known sources of pollution.

3. RESULTS

3.1 Traffic-related pollution estimation

The mean (minimum, maximum) of the observed pollutant data, along with missingness, can be found in the Supplementary Material, Table S1. Of the N=169 commutes, there were 7 commutes where all $PM_{2.5}$ elemental data were not available and therefore we were unable to use these commutes to estimate source factors. For the remaining N=162 commutes, the most missingness was for pbPAH (14.2% missing), noise (12.3%), and PNC (9.3%). The remaining pollutants exhibited less than 5% missing observations. Missing data were due, exclusively, to loss of instrument power during sampling. We imputed pollutants using sequential regression on the logged pollutant data because the pollutants were approximately log-normally distributed.

Using PMF, we identified $L=4$ source factors whose compositions roughly aligned with crustal pollution, secondary pollution, primary tailpipe emissions, and non-tailpipe emissions. The primary tailpipe source was dominated by tailpipe emissions but may contain particles from other sources. This source possibly represents commutes with free-flowing traffic. Similarly the non-tailpipe source may represent “stop-and-go” commutes with a higher proportion of brake and tire wear (24) relative to primary tailpipe emissions. In source apportionment studies, the naming of source factors is subjective, but these names were chosen based on sources identified in the literature (6,18,19). When we examined unconstrained PMF solutions for $L=5$ and $L=6$ source factors, the additional factors did not resemble known sources of traffic-related or ambient pollution. We named the four source factors using the source compositions in our estimated $\boldsymbol{F}$; however, the estimated source factors may include impacts from other sources that emit similar pollutants. Particulate matter levels are subject to complex and nonlinear processing including mixing, chemical transformation, resuspension and removal dynamics. Brake wear components, for example, may be immediately emitted from vehicle braking or be present within road dust following deposition and resuspension.

The source profiles $\boldsymbol{F}$ are shown in Figure 1. Our non-tailpipe traffic source was high in metals and likely contained pollutants emitted from lubricating oils, brake pads through brake wear, as well as tire wear and resuspended road dust (6,18,19). Brake wear and tire wear are highly correlated within commutes and are therefore difficult to separate using the

available data. This source also contained some BC, which is associated with tailpipe emissions. Our crustal source was dominated by aluminum and calcium, but also included some elements that may be found in resuspended road dust (6,18,19). The secondary pollution factor represents other ambient pollution not emitted by the other sources and is dominated by sulfur. The fourth factor, which we named primary tailpipe, was high in BC, OC, pbPAH, and PNC, all of which are related to primary tailpipe emissions. This factor was also strongly associated with noise, consistent with being present in high traffic areas.

All source concentration distributions were right-skewed with larger mean concentrations of secondary pollution and primary tailpipe compared with other source factors (results not shown). The largest difference in source concentration by commute type was seen with primary tailpipe, where pollution concentrations were larger for highway commutes compared to surface street commutes. The means and standard deviations of the source factors, as well as the interquartile ranges (IQR), are shown in Table 1. Across commutes, non-tailpipe and crustal concentrations were highly correlated (R=0.74) and secondary pollution was moderately correlated with both non-tailpipe (R=0.58) and crustal (R=0.54) pollution (Supplementary Material, Table S2).

3.2 Estimating associations with pulmonary response

Demographic information on the commuters is summarized in Table 2. There were 99 individual commuters contributing to a total of 161 commutes with demographic or health data. Of the 99 commuters, 52 were male (52.5%) and 48 were asthmatic (48.5%). There were slightly more asthmatics among women (57.4%) compared to men (40.4%), though this difference was not statistically significant. BMI was missing for three commutes (1.9% missing), and pre-commute cortisol was missing for 12 commutes (7.5% missing). Pulmonary response for the commuters across five time points is shown in Table 3. The number of commutes with complete health measurements varied by outcome (FEV1, FVC, eNO) and time point.

We estimated health effects associated with source-specific pollution using the model in equation 2. The results are shown in Figure 2 as the change in pulmonary response relative to baseline for an IQR increase in source-specific pollution (measured in $\mu g/m^3$). Because eNO was highly right-skewed, we fitted all regression models using log(eNO). Exposure to crustal and secondary pollution was associated with decreased lung function only among asthmatics, with a change in FEV1 of −1.33% (95% confidence interval (CI): −2.45, −0.22) for an IQR increase in crustal pollution and −2.19% (95% CI −3.46, −0.92) for an IQR increase in secondary pollution, relative to baseline. In non-asthmatic subjects, non-tailpipe pollution was associated with decreased lung function with a change from baseline of −0.84% in FEV1 (95% CI: −2.27,0.58) and increased airway inflammation with a change from baseline of 0.04 log ppb (95% CI: 0.00,0.08) for log(eNO). However, non-tailpipe pollution was not associated with pulmonary response in asthmatic subjects. In general, associations with FVC were similar to those for FEV1. We found little evidence of associations between source-specific traffic pollution and log(eNO) among asthmatic subjects.

We also fitted multi-source models to determine whether one or several source factors could explain associations identified in the single source factor models (Figure 2). Because non-tailpipe pollution was highly correlated with crustal and moderately correlated with secondary pollution, we first fitted multi-source models by simultaneously including crustal, secondary, and primary tailpipe pollution. Then, we separately fitted multi-source models for non-tailpipe pollution adjusting for primary tailpipe. Among asthmatic subjects, associations between lung function and crustal were attenuated in multi-source models, while associations with secondary pollution were robust to adjustment for other sources. Among non-asthmatic subjects, associations with non-tailpipe pollution in multi-source models were similar to results from single source models. In multi-source models, we did not find significant associations between primary tailpipe pollution and pulmonary response.

In models allowing for non-linear associations over time, we found some indication among asthmatic commuters that the association between secondary pollution and pulmonary response was somewhat “u-shaped”, with the largest effect occurring at 4.5 hours post-exposure (Figure 3). Additionally, we found that among non-asthmatic subjects, non-tailpipe pollution was only associated with pulmonary response 2.5 hours post-commute (Figure 4).

3.3 Sensitivity analysis

In our sensitivity analysis, we found estimated source factors using the imputed data were similar to results using only the complete case data, with high correlations between source contributions and similar source profiles. We found estimated source factors were similar when the sources were estimated for ACE-1 and ACE-2 separately and were also similar for models restricted to commonly used $PM_{2.5}$ components. Using unconstrained PMF instead of constrained PMF for source apportionment did not provide interpretable results and led to source categories that were less well-resolved than those generated using the constrained model, such as a combined secondary/crustal source (results not shown).

4. DISCUSSION

We conducted one of the first studies to estimate health effects associated with source-specific pollution factors among commuters. The present study builds on the traffic pollution health effects literature in showing components of traffic emissions to be associated with acute pulmonary response in adults, but that associations may vary by source and the asthma status of adults. Estimating source factors using in-vehicle pollution compared with using ambient pollution allowed us to identify potential sources of traffic pollution, while modeling multipollutant exposures within a panel-based epidemiologic study. Typically, modeling multipollutant exposures and health response is challenging because these pollutants are commonly highly correlated across observations (35). In this analysis, we used source apportionment to effectively reduce the dimensionality of the complex, on-road multipollutant exposures. Moreover, we believe this approach is useful in identifying groups of pollutants associated with adverse health outcomes for future targeted studies that may focus on a smaller subset of potentially harmful pollutants or sources.

In the present study, we identified four source factors that allowed the estimation of pulmonary response associated with multipollutant exposures. Our primary tailpipe factor

contained pollutants generated by tailpipe emissions, such as BC, OC, pbPAH, and PNC, though also contained some Ni, V, and WSOC. Notably, Atlanta is a location characterized by relatively little fuel oil use (36), a common source of Ni and V (37). Ni and V have also been reported to be present in lubricating oil (e.g., (38,39)) and enriched in tunnel studies (e.g., (40)). The presence of enriched WSOC may be due to the partitioning of secondary organic aerosols on primary OC, which is elevated in on-road settings. WSOC has been previously found to be present in traffic-related sources (41). Our non-tailpipe source is a mixture of pollutants generated by road dust, tire wear, and brake wear. Although this indicator is useful for the present epidemiologic panel study, exposure studies that can precisely estimate individual non-tailpipe sources are needed. Our source identification from chemical composition data was based on previous work in Atlanta by this research team and others (42–45).

For crustal and secondary source factors, we observed associations with decreased lung function only among asthmatic subjects. Previous studies have also found associations between secondary sources with respiratory hospitalizations in older adults (46), and road dust, which contains crustal elements, with asthma symptoms in children (17). The non-asthmatic commuters in our study ranged from 22 to 58 years old, and this age demographic is very different than that of previous studies of respiratory health and pollution that focused on children (4,17) or older adults (46,47). It is possible that associations with pulmonary response are stronger in populations that are sensitive to respiratory stressors such as children, older adults, and individuals with asthma. As in previous studies, we found some evidence of a “u-shaped” association between secondary pollution and pulmonary response among asthmatic commuters, even after adjustment for other source factors (48) (Figure 3). This shape may indicate a possible delay in biological response following pollution exposure.

In this study, we did not find statistically significant associations between primary tailpipe pollution and pulmonary response, and we only observed significant associations with non-tailpipe pollution among non-asthmatic commuters 2.5 hours post-commute. Our non-tailpipe pollution source contains metallic and transition metal species (Figure 1), some of which have been correlated with measures of oxidative potential (49) and also have been associated with adverse health outcomes in previous studies of pollution (50–52). Our study includes healthy individuals and asthmatic individuals, who were otherwise healthy, and therefore our study subjects may not represent those populations most susceptible to traffic-related pollution. Additionally, this study was a quasi-experimental design that aimed to capture pollution exposures experienced while commuting. It is possible that for asthmatic commuters, the effect of exposure to secondary pollution in the morning before their scripted commute dominated the effect of exposure to traffic-related pollution during the commute. Future studies could control for pollution exposure prior to the commute start by exposing subjects to only filtered air for several hours before the study. Previous epidemiologic studies of traffic-related pollutants have not consistently identified the same pollutant or pollutants most associated with pulmonary response (13,14,48,53). In a previous study of healthy individuals, stronger associations have been observed between traffic-related $PM_{2.5}$ and markers of systemic inflammation compared with lung function (54);

however other epidemiologic studies of inflammatory biomarkers have also found inconsistent associations between traffic pollution exposure and health (55–57).

4.1 Limitations

In this study, although we aimed to estimate sources of traffic-related air pollution using in-vehicle exposures, pollution experienced while commuting may not be limited to traffic-related pollution. For example, secondary and crustal pollution are not directly emitted by vehicles, but their presence within vehicles indicates that other sources of pollution still impact commuting populations. Further, pollutants generated in the vehicle cabin, for example volatile organic compounds (VOCs) emitted by upholstery and carpet, can also contribute to commuter exposures, potentially increasing OC concentrations. We did not measure specific secondary organic aerosols, but future work could potentially measure these to help distinguish in-vehicle sources.

Frequently, PMF is applied to estimate source factors using ambient data, where each sample represents one day with pollutant data. In our study, we applied PMF to in-vehicle exposure data, where each sample represented one commute with pollutant data. Unlike ambient monitoring data, these commutes took place across the city of Atlanta, and so the samples are not geographically fixed. However, our commutes took place within a two-hour time window whereas ambient monitoring data are generally averaged over 24 hours. Therefore, our in-vehicle pollution data are more temporally specific, and may better capture traffic-related pollution sources compared with ambient data. Previous studies have applied PMF to estimate source factors across multiple locations, where the sources can be assumed to be the same across sites (58,59).

We selected four factors for our constrained PMF source apportionment model that best matched known sources of pollution in Atlanta. Choosing the number of source factors in source apportionment modeling is challenging and various methods have been proposed to select this number (60,61). In a comparison of source estimation approaches across Phoenix, AZ and Washington, DC, groups of researchers selected between approximately 3 and 10 sources for each city (62). Despite varying the numbers of source factors, estimated health effects were generally consistent across research groups (1,63). Using source-specific pollution exposure allowed us to focus on subgroups of pollutants that might be most harmful to commuters. This work does not eliminate the possibility that other harmful sources, such as wildfires, also impact commuting populations.

When source apportionment models are applied to pollutant data, source factors can be approximately named based on their chemical compositions. These names are approximate because source factors are estimated and may represent combinations of one or more known sources that emit the same pollutants. For example, our non-tailpipe source contains both processed metals and crustal elements, and so is likely a mix of brake wear, tire wear, and road dust (6,18,19), as well as tailpipe emissions. Our crustal source also contains some processed metals and may contain some road dust. It is worth emphasizing that the issue of properly identifying and naming source factors in source apportionment remains subjective (62) and therefore our specific source factor names should be viewed cautiously. It is possible that our source estimation could be sensitive to the specific days sampled. Future

work could sample a larger number of commutes, and incorporate a more detailed assessment of exposure including ionic aerosols, such as ammonium and nitrate, and hydrophilic and hydrophobic WSOC fractions (41), which may aid in separation of traffic-related sources. However, previous research has not found the specific source apportionment model applied strongly influences health effect estimation (1,20,63).

Determining how to best estimate sources of pollution remains a major challenge in studies of source categories and health. To aid in source estimation, we used prior knowledge about pollutants commonly emitted by traffic-related sources to develop auxiliary equations for constrained PMF. In our analysis, we were unable to separately estimate sources of non-tailpipe traffic pollution including road dust, tire wear, and brake wear. Many sources of traffic pollution, for example brake and tire wear, are spatially and temporally correlated and separating these sources is difficult even when prior information is available. Bayesian source apportionment models provide an alternative approach for incorporating source-specific prior information (64,65), though they can be difficult to fit to available data.

In our source estimation, we incorporated field measurement and laboratory uncertainty; however we did not propagate uncertainty from estimating sources into the estimated health effects. Incorporating uncertainty from estimating sources would somewhat increase uncertainty in subsequently estimated health effects (2). Importantly, previous studies have found that uncertainty due to source estimation is smaller than uncertainty due to the health associations (1,2,4,63). Additionally, to our knowledge few epidemiologic studies have used constrained PMF to estimate source-specific exposure, and methods for incorporating uncertainties from PMF into estimated health effects have not been extensively explored and provide an area for future study. Previous studies have incorporated uncertainty by using fully Bayesian source apportionment models (60,65), Bayesian ensemble source apportionment models (4), and block bootstrapping (2).

5. CONCLUSIONS

Using data from the Atlanta Commuters Exposure studies, we found exposures related to crustal and secondary pollution were associated with decreased lung function among asthmatic commuters. Considering multiple sources of traffic pollution and their impacts on human health is important for developing interventions to protect health while commuting.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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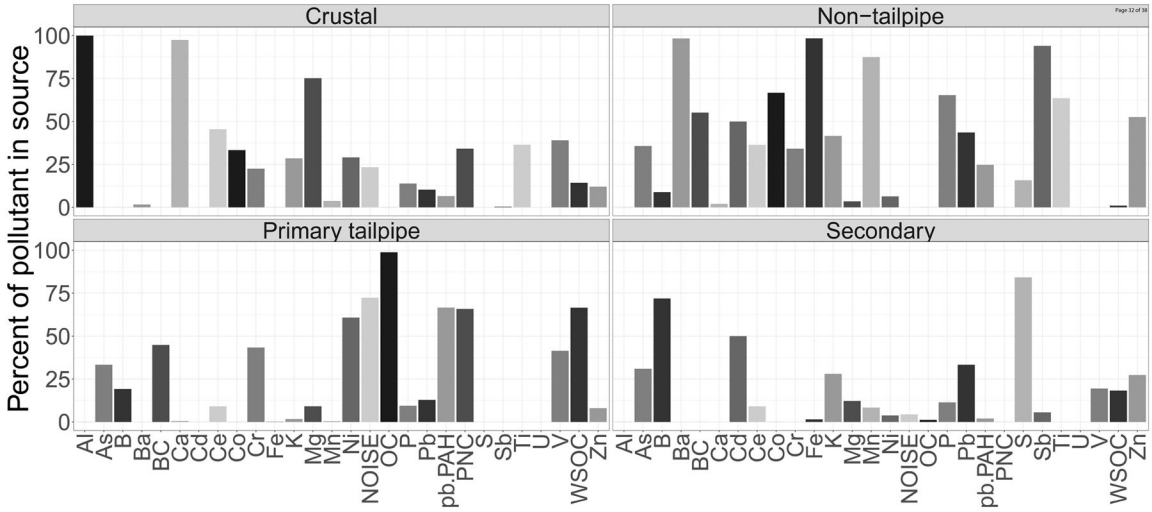

Figure 1.

Profile matrices representing the amount each pollutant contributes to each traffic-related pollutant source factor. Results are shown as the percent of pollutant in each source so that the bars for each pollutant add to 100% across the four source factors.

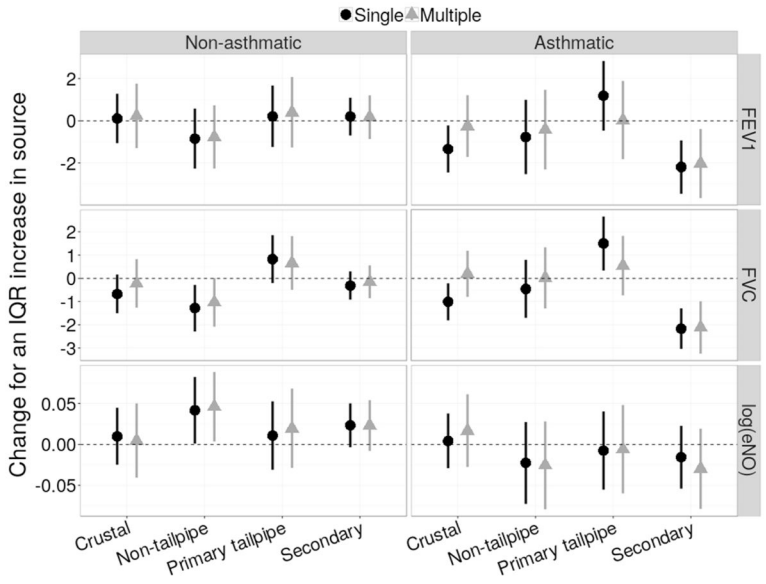

Figure 2.

Estimated changes relative to baseline in lung function measured in predicted percent (FEV1, FVC) and inflammation measured in log parts per billion (eNO) for interquartile range (IQR) increases in each of four source factors, measured in $\mu g/m^3$. Results are shown for both asthmatics and non-asthmatic commuters, using both single source and multi-source models. Airway inflammation, as measured by eNO, was right-skewed and therefore the results are shown for log(eNO).

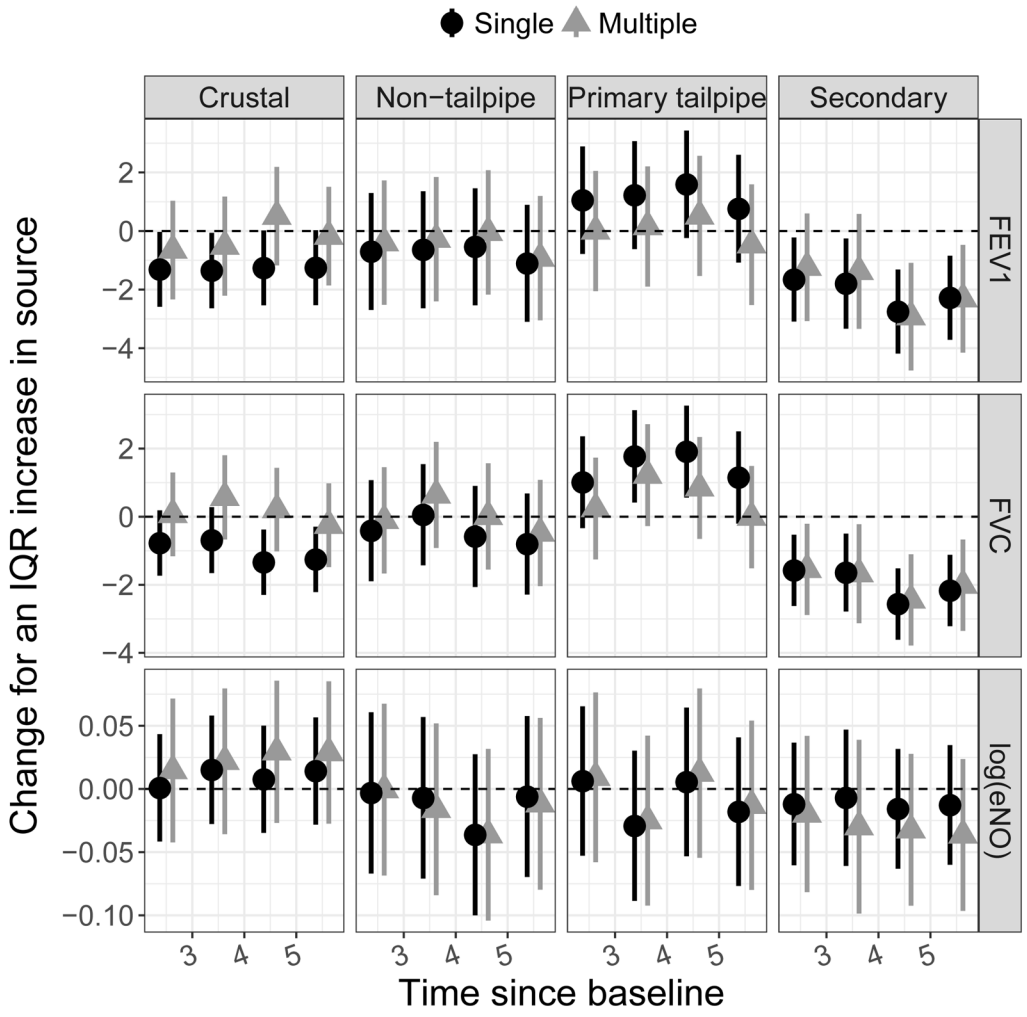

Figure 3.

Estimated changes relative to baseline in lung function measured in predicted percent (FEV1, FVC) and inflammation measured in log parts per billion (eNO) for interquartile range (IQR) increases in each of four source factors, measured in $\mu g/m^3$, where effects are allowed to vary at each time point. Results are shown for asthmatic commuters for both single and multiple source models. Airway inflammation, as measured by eNO, was right-skewed and therefore the results are shown for log(eNO).

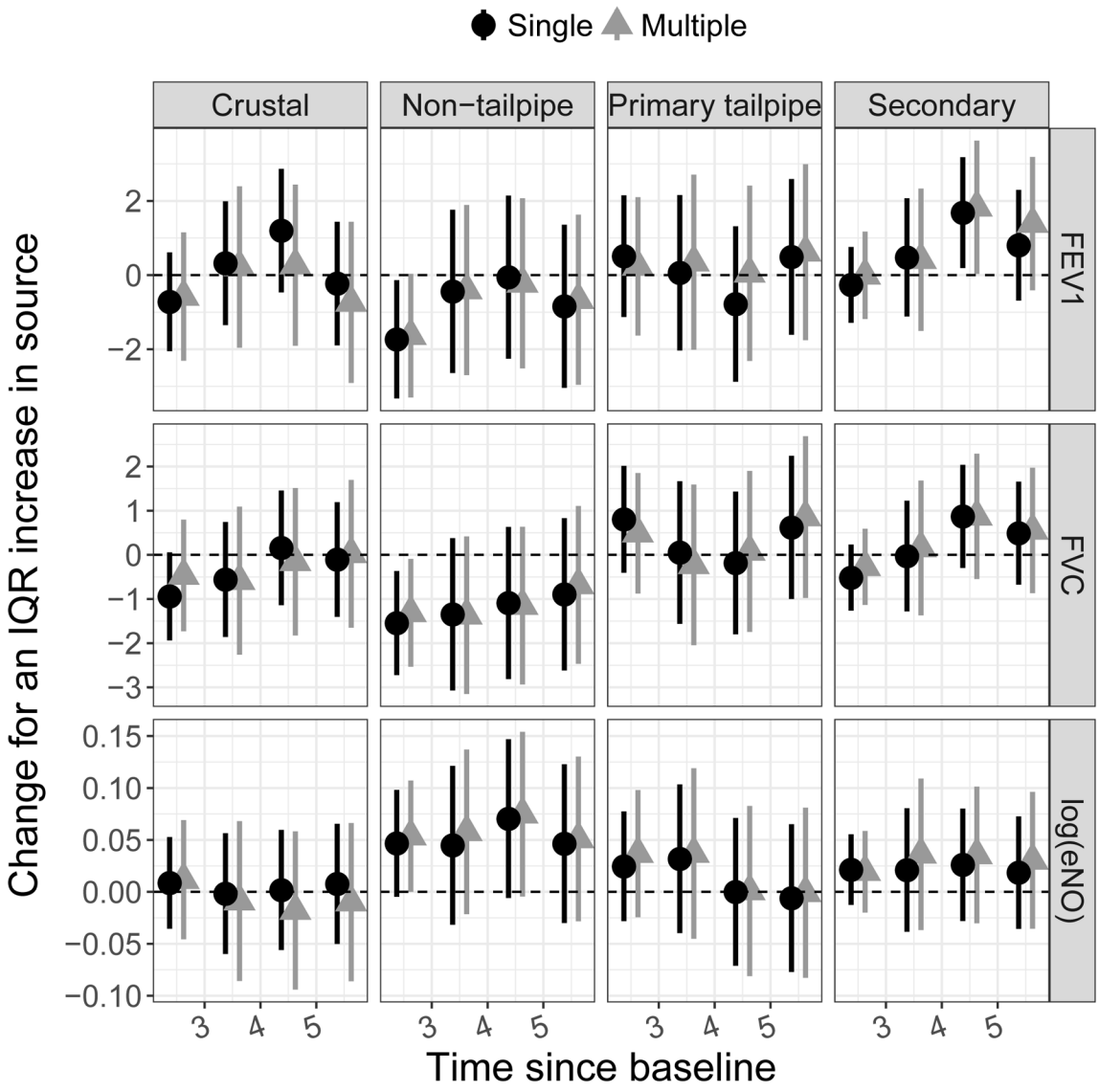

Figure 4.

Estimated changes relative to baseline in lung function measured in predicted percent (FEV1, FVC) and inflammation measured in log parts per billion (eNO) for interquartile range (IQR) increases in each of four source factors, measured in $\mu g/m^3$, where effects are allowed to vary at each time point. Results are shown for non-asthmatic commuters for both single and multiple source models. Airway inflammation, as measured by eNO, was right-skewed and therefore the results are shown for log(eNO).

Table 1

Mean (standard deviation) in μg/m^3 of source-specific traffic pollution across all commutes (Total) and by commute environment. Also shown are the interquartile ranges (IQR) in μg/m^3 for each source.

Source	IQR	Total	Surface street	Highway
Crustal	3.01	3.16 (2.99)	3.31 (2.70)	3.13 (3.06)
Non-tailpipe	2.31	2.16 (1.84)	1.40 (1.13)	2.33 (1.92)
Primary tailpipe	3.96	7.34 (3.13)	4.60 (2.50)	7.94 (2.93)
Secondary	4.30	4.71 (4.84)	5.46 (5.48)	4.55 (4.69)

Table 2

Demographic summary information including fixed (N = 99 commuters) and time-varying (N = 161 commutes) information for the study population and commutes.

Variable	N	Statistic
Fixed		
Male, N (%)	99	52 (52.5)
Asthmatic, N (%)	99	48 (48.5)
Time-varying		
Environment, N (%)	161	132 (82)
Age (years), mean (SE)	161	29.93 (0.79)
BMI, mean (SE)	158	23.68 (0.38)
Baseline cortisol (pg/mL), mean (SE)	149	736.37 (67.44)

Table 3

Mean (SE) lung function (measured in predicted percent) and airway inflammation (measured in log parts per billion (ppb)) estimated using random intercept models at each time point to account for within-subject correlation across commutes.

Hours after baseline	FEV1 (%)		FVC (%)		Log(eNO) (log(ppb))	
	N	Mean (SE)	N	Mean (SE)	N	Mean (SE)
0	157	91.90 (1.39)	157	92.65 (1.29)	156	3.01 (0.07)
2.5	157	91.33 (1.44)	157	91.57 (1.31)	154	3.08 (0.07)
3.5	157	92.67 (1.40)	157	92.29 (1.31)	154	3.12 (0.07)
4.5	157	92.50 (1.43)	157	92.04 (1.36)	156	3.11 (0.07)
5.5	155	92.41 (1.39)	155	91.91 (1.32)	156	3.10 (0.07)