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A multicity study of air pollution and cardiorespiratory emergency department visits: comparing approaches for combining estimates across cities

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Abstract

Determining how associations between ambient air pollution and health vary by specific outcome is important for developing public health interventions. We estimated associations between twelve ambient air pollutants of both primary (e.g. nitrogen oxides) and secondary (e.g. ozone and sulfate) origin and cardiorespiratory emergency department (ED) visits for 8 specific outcomes in

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five U.S. cities including Atlanta, GA; Birmingham, AL; Dallas, TX; Pittsburgh, PA; St. Louis, MO. For each city, we fitted overdispersed Poisson time-series models to estimate associations between each pollutant and specific outcome. To estimate multicity and posterior city-specific associations, we developed a Bayesian multicity multi-outcome (MCM) model that pools information across cities using data from all specific outcomes. We fitted single pollutant models as well as models with multipollutant components using a two-stage chemical mixtures approach. Posterior city-specific associations from the MCM models were somewhat attenuated, with smaller standard errors, compared to associations from time-series regression models. We found positive associations of both primary and secondary pollutants with respiratory disease ED visits. There was some indication that primary pollutants, particularly nitrogen oxides, were also associated with cardiovascular disease ED visits. Bayesian models can help to synthesize findings across multiple outcomes and cities by providing posterior city-specific associations building on variation and similarities across the multiple sources of available information.

Keywords

Air pollution; Bayesian hierarchical models; Cardiorespiratory morbidity; Health associations; Time-series models

1. INTRODUCTION

Short-term exposure to ambient air pollution has been associated with adverse health outcomes including emergency department (ED) visits, hospitalizations, and mortality due to cardiorespiratory diseases (Dominici et al. 2006; Stafoggia et al. 2013; Environmental Protection Agency 2009, 2013; Bell 2004; Burnett et al. 1997). Air pollutants known to be associated with either cardiovascular (CVD) or respiratory (RD) diseases include particle pollutants, for example particulate matter less than 2.5 μm in aerodynamic diameter ($\text{PM}_{2.5}$), and gaseous pollutants such as ozone (Environmental Protection Agency 2009, 2013). Associations with cardiorespiratory hospitalizations or ED visits have been identified for asthma (Strickland et al. 2010; Halonen et al. 2008), chronic obstructive pulmonary disease (COPD) (Qiu et al. 2012; Malig et al. 2015), congestive heart failure (CHF) (Wellenius 2005; S. E. Sarnat et al. 2015), and others (Darrow et al. 2014; Wellenius, Schwartz, and Mittleman 2005; Malig et al. 2015), where these outcomes are based on specific diagnosis codes from hospital billing records. Frequently, these outcomes are combined into broad categories such as all RD ED visits, which may include ED visits for asthma, COPD, upper respiratory infection (URI), and pneumonia. However, identifying the most susceptible individuals, e.g. adults with COPD, requires understanding how associations between air pollution and health vary by specific outcome.

Multicity studies are necessary to robustly estimate air pollution associations for national regulations and also to examine effect modification across *cities* (Environmental Protection Agency 2009). In multicity studies, previously observed differences across cities in estimated associations for total CVD and RD could potentially be driven by differences across cities in the proportion of specific outcomes, such as the proportion of pneumonia. Most previous studies of air pollution and cardiorespiratory ED visits are single-city studies

that examine only a few pollutants or a few specific outcomes (Metzger et al. 2004; Jennifer L. Peel et al. 2005). Several multicity studies conducted in Canada, California, and four U.S. cities have found significant associations between pollutants and ED visits (Stieb et al. 2009; Malig et al. 2015; Krall et al. 2017); however the pollutant-outcome associations that were the strongest varied between studies. By examining associations in multiple cities across the U.S., we can more robustly explore how air pollution is associated with cardiorespiratory ED visits.

A challenge in conducting studies of air pollution and health is that daily counts for specific outcomes can be small, leading to greater uncertainty in estimated health associations compared with examining combined, broad cardiorespiratory categories (Winqvist et al. 2012). When overdispersed Poisson time-series regression models (Ito et al. 2011; S. E. Sarnat et al. 2015; Peng et al. 2009; Ostro et al. 2006) are applied to city-specific data with short time series or with small daily health outcome counts, large effects are required in order to detect statistically significant associations due to large standard errors which are driven by the relatively small sample sizes. In multicity studies, instead of using only the data within the city to estimate associations, Bayesian approaches can borrow information across cities to estimate city-specific health associations using posterior means (Carlin and Louis 2009).

Furthermore, it is possible that the overarching biological pathways that lead from pollution exposure to cardiorespiratory ED visits, such as those involving oxidative stress and inflammation, are similar across outcomes. Therefore, we can also consider using information for multiple *specific outcomes* as well as *cities* to improve estimates. For pollutant-outcome associations, we can compare standard time-series estimates, which do not use information for multiple cities and outcomes, with Bayesian posterior city-specific estimates, which (with the proper structure) use information for multiple cities and outcomes. This comparison can guide interpretation of uncertain time-series estimates as well as quantify potential added value in framing a more complex model. Our results identify and highlight where estimates are both uncertain and different from other estimates across cities.

Associations with cardiorespiratory ED visits also vary by *pollutant*. Some studies have found that pollutants of primary origin, such as nitrogen dioxide (NO₂) and elemental carbon (EC), were strongly associated with CVD ED visits including for myocardial infarction, heart failure (Stieb et al. 2009; J. L. Peel et al. 2007; S. E. Sarnat et al. 2015), and stroke (Villeneuve et al. 2006). For RD ED visits, associations have been identified for pollutants of both primary and secondary origin (Stieb et al. 2009; Alhanti et al. 2016; Orazzo et al. 2009; Halonen et al. 2008). While most studies have examined associations between pollution and health using a single-pollutant framework, a multipollutant framework may better explain some of these differences across previous studies.

As part of ongoing efforts in multicity air pollution investigations (Krall et al. 2017; Alhanti et al. 2016; Friberg et al. 2017; O' Lenick et al. 2017), here we estimated associations between short-term exposure to major air pollutants and cardiorespiratory ED visits in five US cities: Atlanta, GA; Birmingham, AL; Dallas, TX; Pittsburgh, PA; St. Louis, MO. In

these five cities, we first estimated city-specific associations between twelve major air pollutants and specific cardiorespiratory outcomes using standard time-series regression models. Then, to combine estimated health associations across cities using data for all specific outcomes, we applied a Bayesian multicity multi-outcome (MCM) model.

2. MATERIAL AND METHODS

2.1 Data

For each of the five cities, we obtained ED visits using electronic billing data from: the 20-county Atlanta, GA metropolitan area from 2002–2008; the 7-county Birmingham, AL metropolitan area from 2004–2008, the 12-county Dallas-Fort Worth metropolitan area from 2006–2008, the 3-county Pittsburgh area (including Allegheny, Washington, and Westmoreland counties) from 2002–2008, and the 16 counties (8 in Missouri and 8 in Illinois) in the St. Louis metropolitan area from 2002–2007.

ED visits were compiled based on their primary *International Classification of Diseases, 9th Revision* (ICD-9) codes (Web Appendix, Table S1). Outcomes included CHF, cardiac dysrhythmia (DYS), ischemic heart disease (IHD), and stroke for CVD ED visits and asthma and/or wheeze, COPD, pneumonia, and URI for RD ED visits. ED visits for chronic conditions such as COPD represent ED visits for exacerbations or symptoms associated with the condition. The ED visit data in this study were used in accordance with our data use agreements with the Georgia Hospital Association, the Dallas-Fort Worth Hospital Council Foundation, the Missouri Hospital Association, and individual hospitals and/or hospital systems in Birmingham and Pittsburgh. This study was conducted under approval by the Emory University Institutional Review Board; we were exempted from informed consent requirements, given the minimal risk nature of the study and the infeasibility of obtaining informed consent from individual patients for > 1.8 million billing records.

Concentrations of ozone (parts per billion, ppb), CO (parts per million, ppm), NO₂ (ppb), nitrogen oxides (NO_x; ppb), SO₂ (ppb), PM₁₀ (PM < 10 µm, measured in µg/m³), PM_{2.5} (µg/m³), and PM_{2.5} chemical constituents (µg/m³) including sulfate, nitrate and ammonium ions, OC, and EC were obtained from ambient monitoring stations located within each of the metropolitan areas. To create population-weighted average estimates of the 24-hour average (all PM measures), one-hour maximum (all gases except ozone), or the 8-hour maximum (ozone only) ambient pollution concentrations, monitor concentrations were fused with Community Multi-Scale Air Quality model estimates as described in previous work (Friberg et al. 2017, 2016). The fused estimates better reflect population exposure for our large metropolitan areas compared with monitoring data alone (Friberg et al. 2017). To control for possible confounding by meteorological factors, we also obtained temperature and dew-point temperature from the National Climatic Data Center from automated surface observing stations at the major airport in each city.

2.2 Poisson time-series regression models

To estimate pollutant-outcome associations, we used the most common approach applied within the literature (Peng et al. 2009; Krall et al. 2013): we applied overdispersed Poisson

time-series regression models to data from each city separately. We estimated associations with ED visits for an *a priori* selected exposure lag based on previous research: lag 0 exposure for CVD ED visits (Dominici et al. 2006; J. A. Sarnat et al. 2008; Metzger et al. 2004; Ye et al. 2017) and 8-day moving average exposure (mean of lag 0–7 exposure) for RD ED visits (Gass et al. 2015).

We controlled for potential confounding to be consistent with our previous studies of pollution and ED visits (Krall et al. 2017; Winquist et al. 2015). First, we included in our models weekday, season, holidays, meteorology, temporal trends, and the hospitals reporting data for each day. To control for meteorology, we used cubic polynomials of maximum temperature and dew-point temperature. For CVD ED visits, we used lag 0 maximum temperature and lag 0–2 dew-point temperature. For RD ED visits, with the longer exposure lag, we used separate cubic splines of lag 0–2 and lag 3–7 temperature and dew-point temperature, as in a previous study of pollution and RD ED visits (Gass et al. 2015). In all models, we included interaction terms of season with maximum temperature, weekdays, and federal holidays. To control for long-term trends in ED visits, we included cubic splines of time with one degree of freedom (df) per month.

We estimated associations for each specific outcome, and for combined broad categories created by adding together CHF, DYS, IHD, and stroke ED visits for CVD, and adding together asthma and/or wheeze, COPD, pneumonia, and URI ED visits for RD, as is common in studies of air pollution and cardiorespiratory ED visits (S. E. Sarnat et al. 2015; Winquist et al. 2015). To compare associations across pollutants, we reported results as the relative risk for an interquartile range increase in the pollutant using the median of city-specific interquartile ranges.

2.3 Bayesian hierarchical models

To estimate both overall and posterior city-specific associations, we applied Bayesian hierarchical models using the TLNise two-level normal independent sampling estimation software with uniform priors (Everson and Morris 2000). Commonly in air pollution and health studies, time-series models described in the previous section are first applied to city-specific data (Dominici et al. 2006; Zanobetti and Schwartz 2009). Then, the estimates are pooled in a Bayesian hierarchical framework. This approach is common because when the number of cities is large, fitting mixed models becomes challenging with the increasing size of the datasets. Therefore, we first apply the time-series regression models described in the previous section to estimate city-specific associations for each city c and outcome j , denoted $\hat{\beta}_{cj}$ and their corresponding standard errors, $\hat{\sigma}_{cj}^2$. Then for each pollutant, we fitted Bayesian hierarchical models of the form:

$$\begin{aligned}\hat{\beta}_{cj} &\sim N(\beta_{cj}, \hat{\sigma}_{cj}^2) \\ \beta_{cj} &\sim N(\mu_j, \gamma_j^2)\end{aligned}\quad 1$$

Where β_{cj} is the unobserved true city-specific association for city c and outcome j , and μ_j is the unobserved, shared multicity association for outcome j .

Traditional *multicity* models shown in equation (1) were first fitted to the five estimated city-specific associations from time-series models. However because we only have data for five cities, these models are difficult to fit to the available data. Specifically, it can be challenging to estimate γ_j^2 for each outcome using only five estimated associations across cities.

Therefore, in our Bayesian *MCM model*, we included multiple outcomes simultaneously to estimate a shared intercity variation γ^2 . Instead of fitting models separately for each outcome, the *MCM model* borrows information across cities for each pollutant, incorporating information from all outcomes, via city-specific random slopes, β_{cj} varying around the multicity association. The full Bayesian model details for both the multicity and MCM models are located in the Web Appendix: we assigned uniform priors to all parameters. Models were fitted using the *tlmise* package in R (Peng and Everson 2016).

In equation (1), γ_j^2 represents the residual heterogeneity across cities. The main difference between the multicity and MCM models is that the multicity model estimates the mean μ_j and variance γ_j^2 separately for each outcome j . In contrast, the MCM model estimates the mean μ_j and shares the estimate of the residual heterogeneity γ^2 across outcomes. Therefore, the MCM model assumes that this residual heterogeneity across cities does not vary by outcome. The MCM model borrows information differently to estimate multicity health associations (Estimated associations for 5 cities across each outcome) compared with the standard multicity model (Estimated associations for 5 cities separately by outcome). Therefore, if the heterogeneity assumption of the MCM model is correct, the MCM model can leverage multiple outcomes to adjust uncertain health associations via a better estimate of the heterogeneity variance γ^2 . This is a modeling choice that defines how we borrow information across observations, and should be assessed as an assumption when comparing models. Here, we compared the MCM model to the standard multicity model using the ratio of heterogeneity variances, γ^2/γ_j^2 . This ratio will be less than one when the residual heterogeneity is smaller for the MCM model, which may suggest that γ_j^2 cannot be estimated well using data from only five cities and therefore the MCM model fits the data better than the multicity model. However, it is possible that in some cases, the residual heterogeneity is smaller for the MCM model by chance alone.

Bayesian hierarchical models implement modeling assumptions via prior distributions on the random effects yielding posterior mean estimates of the city-specific associations (Carlin and Louis 2009). For each city, the posterior mean estimate is a weighted average of 1.) the city-specific estimate from time-series regression models, $\hat{\beta}_{cj}$, and 2.) the estimated multicity average estimate, μ_j . The weights given to the time-series estimate depend on the time-series variance $\hat{\sigma}_{cj}^2$, which, in turn, depends on the data available in city c for outcome j . When there are many days of data available and large daily counts for an outcome (i.e. yielding a small value of $\hat{\sigma}_{cj}^2$), or when all time-series estimates are similar, the posterior

mean will be very similar to the time-series estimates. However, uncertain time-series estimates with large variances $\hat{\sigma}_{cj}^2$ will be pulled towards the more robust estimated average across cities, μ_j in the MCM or multicity models. While posterior estimates pulled towards the average incur some bias relative to time-series estimates, they have smaller uncertainty, i.e. the familiar bias-variance trade-off in statistical estimation, a standard motivation for hierarchical Bayesian inference. The Bayesian approach maintains some differences in associations between cities, based on the variances $\hat{\sigma}_{cj}^2$ while borrowing information across cities.

2.4 Multipollutant modeling

In our main analyses, we treated each pollutant individually in the MCM model. We also considered incorporating multipollutant exposures using a hierarchical principal component analysis approach (PCA) (Thurston and Spengler 1985; Ito, Xue, and Thurston 2004; Krall, Hackstadt, and Peng 2017). Briefly, varimax-rotated PCA was applied to the time series of pollution data in each city. The number of principal components (PCs) was chosen based on the number of eigenvalues of the correlation matrix that were greater than one. Then, for each multipollutant PC, health associations were estimated using time-series models and the MCM model as described earlier. Our multipollutant approach adds to our main results by estimating the associations between groups of pollutants and ED visits for specific outcomes. This approach does not attempt to identify sources of pollution, as in previous studies of pollution and health (Gass et al. 2015; J. A. Sarnat et al. 2008; Kioumourtzoglou et al. 2014; Mar et al. 2006). More details about our multipollutant approach can be found in the Web Appendix.

2.5 Sensitivity analysis

We tested the sensitivity of our results to the confounders included in the models by removing sequentially meteorology, holidays, weekdays, and season. We also compared results using different exposure lags for both CVD and RD: same day, 2-day moving average, and 7-day moving average exposure. Although we chose our control for time based on previous studies of ED visits (cubic spline with one df / month) (S. E. Sarnat et al. 2015; Winquist et al. 2015), we also compared this approach to using natural splines with 7 df/ year, similar to previous studies of other health outcomes (Ostro et al. 2006; Krall et al. 2013; Zanobetti et al. 2009; Samet, Dominici, et al. 2000). Last, we compared our main model results for the Bayesian MCM model to results using Jeffrey's prior for γ (Peng and Everson 2016).

3. RESULTS

Across the five cities, the number of days with complete pollution and ED visit data varied from 1,096 days in Dallas to 2,557 days in Atlanta. The average number of daily ED visits for cardiorespiratory diseases also varied across cities and outcomes (Table 1). Atlanta and Dallas had the most daily ED visits with means of 400 and 448 daily RD ED visits, and 98 and 120 daily CVD ED visits, respectively. The majority of ED visits for RD was for URI. There were fewer CVD ED visits for stroke compared to other outcomes.

The mean concentrations for all pollutants and the median of city-specific IQRs are shown in Table 2 for five cities. Atlanta had the largest concentrations of ozone and PM_{2.5}, with mean concentrations of 42.1 ppb (standard deviation (SD) = 17.3) and 15.4 µg/m³ (SD = 7.1) respectively. Compared with other cities, Dallas had relatively lower concentrations of PM_{2.5}. Median correlations across cities between combustion-related pollutants, including OC, EC, and NO_x, were relatively large, and correlations between secondary pollutants such as ammonium ion and sulfate were also large (Web Appendix, Table S2).

3.1 Estimated health associations

We first estimated pollution-outcome associations for each of the five cities separately using overdispersed Poisson time-series regression models as in previous multicity studies (Peng et al. 2009). Then, we estimated posterior city-specific and overall associations using both the multicity and MCM Bayesian hierarchical models (Web Appendix, Figures S1-S2). We fitted the MCM model separately for CVD and RD outcomes to account for the differences in potentially complex confounding factors, such as the populations primarily affected (e.g., children and CVD vs. children and RD), as well as to match previous studies that have separately examined CVD and RD ED visits (S. E. Sarnat et al. 2015; Gass et al. 2015).

Estimated health associations across cities from the MCM model are shown in Figures 1–2. The pollutants in Figures 1–2 are roughly ordered according to primary and secondary pollutants (Web Appendix, Figure S3) for consistency across results figures. For short-term exposure to NO_x and NO₂, we found some positive and statistically significant associations for CVD ED visits, with relative risks for NO_x of 1.02 (95% posterior interval (PI): 1.01, 1.03) and 1.02 (95% PI: 1.01, 1.03) for CHF and stroke respectively. In general, primary pollutants related to combustion such as CO, NO₂, NO_x, SO₂, EC, and OC, showed positive, though not always statistically significant, associations with CVD ED visits. For RD ED visits, associations varied in magnitude across pollutants. For ozone, the largest estimated associations were for COPD ED visits, with a relative risk of 1.09 (95% PI: 1.02, 1.16). We did not fit the MCM model for the combined categories of CVD and RD because these combined categories are created by summing specific outcomes.

Comparing associations between standard time-series regression models and posterior means from the multicity and MCM models (Web Appendix, Figures S1-S2), the point estimates for the two hierarchical models were similar, with smaller standard errors associated with the MCM model, as expected. When large daily counts for an outcome were available, there were few differences across models. For example, the associations between PM_{2.5} and asthma/wheeze in Atlanta from the time-series, multicity, and MCM models were, respectively: 1.03 (95% confidence interval: 1.01, 1.06), 1.04 (95% PI: 1.02, 1.06), 1.04 (95% PI: 1.02, 1.06). However, when associations from time-series models were more uncertain, estimated health associations from Bayesian hierarchical models were pulled towards the average association across cities. In Dallas, the estimated PM_{2.5}-stroke relative risk was 1.04 (95% confidence interval: 1.01, 1.07) from the time-series model and 1.01 (95% PI: 0.996, 1.03) from the MCM model. Additionally, the ratio of residual variances γ^2/γ_j^2 was generally less than one (Web Appendix, Figure S4), with a median decrease of 69% for the MCM model compared with the multicity model. The residual variance for SO₂

and RD was larger for the MCM model compared to the multicity model, which reflects the differences across RD outcomes observed in Dallas compared to other cities (Figure 2).

3.2 Multipollutant modeling

Across cities, three multipollutant PCs displayed in the Web Appendix Figure S3 explained most of the variability in our pollutant data, defined as the number of eigenvalues greater than 1 (Krall, Hackstadt, and Peng 2017). The proportion variability explained by the PCs selected for each city ranged from 73.5% (Atlanta) to 81.0% (Pittsburgh). PC1 contained mostly primary pollutants including CO, NO₂, NO_x, SO₂, and EC, while PC2 contained mostly secondary pollutants such as ozone, ammonium, and sulfate. PC3 contained mostly ammonium and nitrate. However, in Atlanta, this PC explained little variability in the data, had an eigenvalue less than one, and was not interpretable as ammonium nitrate based on its loadings.

We then fitted health models, including the time-series model and the Bayesian models, separately for each multipollutant PC as in the main pollutant models. Because the PCs are uncorrelated, results from time-series models fitted to all multipollutant PCs simultaneously were similar to results from single-PC models. We found associations of the primary pollutant PC (PC1) with all CVD specific outcomes, though associations were not all statistically significant and varied across cities (Figure 3). For RD ED visits, both primary and secondary pollutants (PCs 1–2) were positively associated with all respiratory specific outcomes. Associations of PC1 with COPD and pneumonia were positive, but not consistently statistically significant across cities. There was some evidence of associations between PC3 (ammonium nitrate) with URI ED visits.

3.3 Sensitivity analysis

In time-series regression models, our results were not very sensitive to excluding control for meteorology, holidays, day of week, and season. The lack of sensitivity to control for meteorology might be driven by the additional control for season and time in each model. For combustion-related pollutants such as EC and NO_x, estimated associations for models without control for holidays and weekdays were attenuated in RD models and greater in magnitude in CVD models, relative to results from our basic models (results not shown). Estimated associations with RD for shorter exposure periods were attenuated relative to lag 0–7 associations. For CVD, associations for lags longer than same-day exposure were not substantially different from same-day exposure. We did not find that controlling for time using a natural spline with 7 df/year substantially changed our estimated associations for CVD outcomes and most RD outcomes. We found some indication that estimated associations with this control for time were larger in magnitude for URI and for associations in Atlanta. Last, we did not find that our choice of prior for substantially impacted our estimated health associations.

4. DISCUSSION

In this study of twelve major air pollutants across five cities, we found positive associations of both primary (e.g. NO_x and EC) and secondary pollutants (e.g. ozone and sulfate) with

RD ED visits, though associations were not consistently statistically significant across cities. For CVD ED visits, there was some indication of associations for primary pollutants including NO₂ and NO_x, though these associations were smaller in magnitude compared with RD outcomes and were not consistently statistically significant across cities.

A challenge in studies of pollution and health is how to synthesize estimated health associations across pollutants, outcomes, and cities. Because some health associations are uncertain due to small daily counts, using Bayesian approaches can formalize what information our models use in order to help guide interpretation of estimated associations that are large in magnitude, but highly uncertain. Previous studies that have examined a large number of pollutants and outcomes frequently present all results for the reader to interpret (J. L. Peel et al. 2007; S. E. Sarnat et al. 2015; Halonen et al. 2008). Our MCM model leverages information across cities and outcomes to guide interpretation of a large number of estimated associations. For example, the time-series association between ozone and CHF in Dallas is statistically significant and large in magnitude, but with a large standard error. The posterior mean from the MCM model shrinks the Dallas estimate towards the average across cities, and the shrunk estimate is no longer statistically significant. In this way, the observed differences between the time-series association and MCM posterior mean can help to guide the interpretation of the association in one city (e.g. Dallas) relative to other cities. It is possible that, in some cases, the shrunk estimates from the MCM may be biased towards the null; however, these conservative estimates can still help to focus future studies where the evidence is stronger across cities.

In our Bayesian hierarchical models, γ_j^2 represents the residual variance across cities. If we apply Bayesian hierarchical models to a small number of data points, e.g. five cities in the multicity model, estimating γ_j^2 from only city-specific data is difficult. The MCM model uses more information from all cities and multiple outcomes to estimate an assumed shared value of γ^2 . A challenge with the MCM model is that with only five cities, we cannot estimate well whether the residual heterogeneity across cities varies by outcome and so this is an assumption we make. Additional data are necessary to fully evaluate whether the residual heterogeneity varies by outcome and therefore whether the MCM or multicity model is more appropriate. An advantage of the MCM model is that it provides a more robustly estimated γ^2 compared with the more commonly applied multicity model in studies of only a few cities. However, when the residual heterogeneity γ_j^2 varies by outcome, such as between SO₂ and RD, the assumptions of the MCM model are not met. Specifically, there is greater heterogeneity across cities for COPD, pneumonia, and URI compared with asthma/ wheeze (Web Appendix, Figure S2). Future work should explore whether factors that vary both by city and by outcome, such as the age distribution of ED visits, might drive this result.

In comparing our results between the multicity and MCM models (Web Appendix, Figures S1-S2), the inferences that would be drawn from the point estimates and corresponding confidence intervals are often the same. It is possible that in other scenarios, estimating a shared residual heterogeneity across outcomes, γ^2 , as in the MCM model may have a larger impact on statistical inference. Because city-specific time-series estimates are frequently

highly uncertain, posterior estimates from either the multicity or MCM model might better reflect the consensus across cities.

An advantage of our Bayesian modeling approach is that it can be extended to include more prior information. Currently, we only use non-informative priors to match previous studies that used Bayesian hierarchical models to pool information across cities. In the present study, we assume that the confounding structure of the model and the lag structure is the same across outcomes, within CVD and RD categories. While this is a strong assumption, it is common in studies of air pollution and health when outcomes are examined separately, specifically the same confounding variables and lags are specified *a priori* for all outcomes. Importantly, this is not a required assumption of the Bayesian modeling approach applied here, and future work could relax this assumption. In the present study, we did specify the confounding structures separately for CVD and RD outcomes, but in general, there is not enough prior knowledge to specify these structures separately by specific outcome within CVD and RD. We also did not estimate global means for CVD and RD using the MCM model because the focus of the present study was examining associations for specific outcomes. A three-level normal Bayesian hierarchical model could be fitted using the MCM approach to estimate the overall associations for CVD and RD ED visits. While we assumed a linear association here to be consistent with previous studies of pollution and ED visits (12,50), future work could extend our model to include non-linear associations.

Previous studies of environmental exposures have used semi-Bayes approaches to estimate city-specific associations (Stingone et al. 2014; Kalkbrenner et al. 2010; Braun et al. 2014) instead of the approach we applied in this study. Semi-Bayes approaches assume the second-level variance, γ_j^2 , is known before collecting data, whereas we have estimated γ_j^2 from the data. However, when many data points are available, the second-level variance γ_j^2 can frequently be estimated well.

In this work, we were limited to data from five cities for which we had ED visit data. These five cities represent the southeast (Birmingham, AL; Atlanta, GA), the south (Dallas, TX), and the Midwest/industrial Midwest (St. Louis, MO; Pittsburgh, PA) (Samet, Zeger, et al. 2000). Therefore, the results of this multicity study, while important for informing knowledge about associations between individual pollutants and ED visits, may not fully represent associations across the US. It is possible that specific outcomes for ED visits designated by ICD-9 codes may represent other, similar outcomes. We reduced the potential for miscategorizing specific outcomes in the present study by combining primary diagnosis ICD-9 codes (e.g. from the Web Appendix, Table S1, COPD includes 491 (chronic bronchitis), 492 (emphysema), 496 (chronic airway obstruction, not elsewhere classified)) based on similar specific outcomes (Web Appendix, Table S1).

In addition to our results examining each of the twelve pollutants individually, we also considered a multipollutant approach based on PCA to examine multipollutant exposures. In general, we found that the results were consistent between the MCM model using single pollutant exposures and multipollutant exposures. Applying the multipollutant approach allowed us to examine a smaller number of interpretable pollution components. We

employed a multipollutant approach that grouped together pollutants based on their shared temporal variation. This multipollutant method was designed to help synthesize the many estimated associations across pollutants, outcomes, and cities. This approach also helps to identify groups of pollutants most associated with adverse health outcomes. An alternative approach would attempt to identify the pollutant or pollutants that are most strongly associated with adverse health outcomes using variable selection methods.

4.1 Conclusions

This work estimated associations between twelve major ambient air pollutants, and observed multipollutant groupings, and cardiorespiratory ED visits across five US cities. Our approach is broadly applicable to future studies of multiple air pollutants and ED visits for specific outcomes, although other approaches are also necessary to develop and assess pollution interventions. We proposed the use of a multicity multi-outcome (MCM) model that incorporated information across cities and outcomes to estimate both health associations. In studies of a small number of cities, the MCM model can be used to leverage the available information to estimate health associations for multiple cities and outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations:

COPD	chronic obstructive pulmonary disease
CHF	congestive heart failure
CVD	cardiovascular diseases
DF	degrees of freedom
DYS	cardiac dysrhythmia
ED	emergency department
IHD	ischemic heart disease

MCM	multicity multi-outcome
RD	respiratory diseases
URI	upper respiratory infection

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Highlights

- Bayesian models can incorporate information across multiple cities and outcomes
- Ambient air pollutants are associated with respiratory disease ED visits.
- Pollutants of primary origin are associated with cardiovascular disease ED visits.

Figure 1.

Estimated relative risks of cardiovascular emergency department visits and 95% posterior intervals associated with an interquartile (IQR) increase in pollutant for both the average across cities and posterior mean estimates for each city based on the multicity multi-outcome (MCM) model. Pollutants are roughly ordered according to primary and secondary pollution, separated by a vertical line.

Figure 2.

Estimated relative risks of respiratory emergency department visits and 95% posterior intervals associated with an interquartile (IQR) increase in pollutant for both the average across cities and posterior mean estimates for each city based on the multicity multi-outcome (MCM) model. Pollutants are roughly ordered according to primary and secondary pollution, separated by a vertical line.

Figure 3. Estimated relative risks of (A.) cardiovascular and (B.) respiratory emergency department visits and 95% posterior intervals associated with an interquartile (IQR) increase in multipollutant principal components (PCs) for both the average across cities and posterior mean estimates for each city based on the multicity multi-outcome (MCM) model.

Table 1.

Data Availability and the Daily Mean (Standard Deviation) Number of Emergency Department Visits for Cardiorespiratory Diseases in Atlanta, GA; Birmingham, AL; Dallas, TX; Pittsburgh, PA; St. Louis, MO

Variable	Atlanta	Birmingham	Dallas	Pittsburgh	St. Louis
Years of data	2002–2008	2004–2008	2006–2008	2002–2008	2002–2007
# of days with complete data	2557	1827	1096	2557	2004
Cardiovascular	98(21)	32(7)	120(16)	51(10)	96(14)
CHF	26 (9)	7(3)	31 (7)	15 (5)	26 (6)
DYS	25 (6)	6(3)	30 (6)	12 (4)	21 (5)
IHD	28 (7)	11(4)	32 (7)	14 (4)	30 (7)
Stroke	18 (6)	6(3)	24 (5)	10 (3)	18 (4)
Respiratory	400 (136)	73 (26)	448 (155)	103(32)	286(84)
Asthma/Wheeze	83(28)	12(5)	76(24)	25 (8)	51(16)
COPD	20 (8)	7(3)	22 (7)	11 (4)	16 (5)
Pneumonia	51(20)	10(5)	71(28)	21 (9)	46(16)
URI	231(87)	44 (18)	258(94)	44(16)	166(54)

Abbreviations: cardiac dysrhythmia (DYS); chronic obstructive pulmonary disease (COPD); congestive heart failure (CHF); ischemic heart disease (IHD); upper respiratory infection (URI)

Table 2.

Mean (Standard Deviation) Concentration and Interquartile Ranges for Twelve Gaseous and Particle Pollutants for Atlanta, GA; Birmingham, AL; Dallas, TX; Pittsburgh, PA; St. Louis, MO

Pollutant	Units	IQR*	Atlanta	Birmingham	Dallas	Pittsburgh	St. Louis
Ozone, 8-hour max	ppb	25.34	42.1(17.3)	41.8(14.6)	41.8(14.4)	37.5(18.6)	38.4(17)
CO, 1-hour max	ppm	0.25	0.6(0.3)	0.4 (0.2)	0.4 (0.2)	0.5(0.2)	0.5(0.2)
NO ₂ , 1-hour max	ppb	9.44	21.6(7)	13.2(4.3)	19.7(8.2)	26.5(8.1)	21.3(6.3)
NOx, 1-hour max	ppb	34.54	45.6(30.5)	24.5(13.8)	41.4(32.2)	56 (35.8)	51.3(30.9)
SO ₂ , 1-our max	ppb	8.09	10 (6.9)	11 (6.5)	5.4 (3.9)	20.2(9.5)	11.6(5.4)
PM ₁₀ , 24-hour mean	µg/m ³	14.1	23.3(10.4)	26.3(10.9)	22.6(9.5)	23.7(12.2)	23 (9.4)
PM _{2.5} , 24-hour mean	µg/m ³	8.71	15.4(7.1)	14.5(7.1)	10.8(4.7)	15 (8.6)	13.8(6.7)
EC	µg/m ³	0.55	1.1(0.6)	0.9 (0.5)	0.5 (0.2)	0.9(0.5)	0.7(0.3)
OC	µg/m ³	1.94	3 (1.5)	4.2 (2.4)	2.5 (1.2)	3.8(2)	3.3(1.6)
Sulfate	µg/m ³	2.64	4.5(3)	3.9 (2.5)	2.8 (1.8)	4.9(3.4)	3.4(2.6)
Ammonium	µg/m ³	1.01	1.4(0.9)	1.5 (0.9)	1 (0.7)	1.8(0.9)	1.7(1)
Nitrate	µg/m ³	0.6	0.6(0.6)	0.7 (0.7)	0.5 (0.7)	2.1 (2)	2.1(2)

Abbreviations: carbon monoxide (CO); elemental carbon (EC); interquartile range (IQR); nitrogen dioxide (NO₂); nitrogen oxides (NOx); organic carbon (OC); particulate matter less than 2.5 µm (PM_{2.5}); particulate matter less than 10 µm (PM₁₀); sulfur dioxide (SO₂); parts per billion (ppb); parts per million (ppm)

* Median IQR across cities