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Impact of air pollution control policies on cardiorespiratory emergency department visits, Atlanta, GA, 1999–2013

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ABSTRACT

Background: Air pollution control policies resulting from the 1990 Clean Air Act Amendments were aimed at reducing pollutant emissions, ambient concentrations, and ultimately adverse health outcomes.
Objectives: As part of a comprehensive air pollution accountability study, we used a counterfactual study design to estimate the impact of mobile source and electricity generation control policies on health outcomes in the Atlanta, GA, metropolitan area from 1999 to 2013.
Methods: We identified nine sets of pollution control policies, estimated changes in emissions in the absence of these policies, and employed those changes to estimate counterfactual daily ambient pollutant concentrations at a central monitoring location. Using a multipollutant Poisson time-series model, we estimated associations between observed pollutant levels and daily counts of cardiorespiratory emergency department (ED) visits at Atlanta hospitals. These associations were then used to estimate the number of ED visits prevented due to control policies, comparing observed to counterfactual daily concentrations.
Results: Pollution control policies were estimated to substantially reduce ambient concentrations of the nine pollutants examined for the period 1999–2013. We estimated that pollutant concentration reductions resulting from the control policies led to the avoidance of over 55,000 cardiorespiratory disease ED visits in the five-county metropolitan Atlanta area, with greater proportions of visits prevented in later years as effects of policies became more fully realized. During the final two years of the study period, 2012–2013, the policies were estimated to prevent 16.5% of ED visits due to asthma (95% interval estimate: 7.5%, 25.1%), 5.9% (95% interval estimate: −0.4%, 12.3%) of respiratory ED visits, and 2.3% (95% interval estimate: −1.8%, 6.2%) of cardiovascular disease ED visits.
Discussion: Pollution control policies resulting from the 1990 Clean Air Act Amendments led to substantial estimated reductions in ambient pollutant concentrations and cardiorespiratory ED visits in the Atlanta area.

1. Introduction

Ambient air pollution is a substantial contributor to cardiovascular and respiratory morbidity. Federal policies aimed at limiting air pollution cost over $80 billion per year (Bell et al., 2011; US Environmental Protection Agency, 2011). As efforts to improve air quality have grown in scope and sophistication, attention has turned to evaluating the effectiveness of these policies. Air pollution accountability research aims to quantify the impact of pollution control policies on ambient pollutant levels and health endpoints (Health Effects Institute Accountability Working Group, 2003).

Many accountability studies of air pollution utilize a pre-post study design comparing outcomes before and after an intervention. This approach has been used to measure health effects of distinct events such as the closure of a steel mill and short-term traffic restrictions during the Olympics and the Asian Games (Friedman et al., 2001; Lee et al., 2007; Pope, 1989; Wong et al., 1999). Pre-post studies can suffer from temporal confounding as changes in occurrence of health outcomes

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before and after an intervention could be due to many known or unknown factors other than the intervention (Clancy et al., 2002; Dockery et al., 2013). Potential for such confounding can increase with study length; therefore, pre-post studies are best suited for short-term events with distinct target and control periods. However, many pollution control policies are gradually implemented over long periods of time, and appropriate accountability research requires alternative study designs.

Additional challenges in accountability research stem from the fact that the ultimate goal is generally to improve health outcomes, but the policies act upon sources of pollutant emissions. Studies aimed at capturing consequences of pollution control policies therefore are tasked with quantifying the effect of policies on emissions, then assessing downstream effects of emissions reductions on ambient pollutant levels, which are then used to estimate impacts on public health (Health Effects Institute Accountability Working Group, 2003; Henneman et al., 2017a, 2017b) These types of studies require collection of substantial amounts of data as well as complex modeling to link changes at each level. Consequently, many accountability studies are unable to assess impacts of pollution control policies on all three levels: emissions, ambient pollutant concentrations, and health endpoints (Peters et al., 2009; US Environmental Protection Agency, 1997, 2011).

In the current study, we utilized a counterfactual study design to estimate the effect on air quality and health in Atlanta, Georgia, of pollution control policies that were largely implemented to bring the region into compliance with the National Ambient Air Quality Standards (NAAQS) and with other regulations related to the 1990 Clean Air Act Amendments. In this design, observed outcomes after an intervention are compared to estimates of unobserved outcomes that would have occurred in the absence of the intervention; all external factors are held constant. We assessed the impact of numerous pollution control policies implemented in the 1990s and 2000s, each of which took effect over long time periods, such that there were no clear-cut reference or intervention periods. In our counterfactual analysis, we modeled continuous changes in emissions, ambient pollutant levels, and health outcomes during 1999–2013 to quantify the impacts of these policies. Through the use of multipollutant modeling, we were able to more fully capture the joint benefits of multipollutant reductions to public health.

2. Methods

The overall study approach is summarized in Fig. 1 and further described below. Additional detail is provided in the full research report on this project published by the Health Effects Institute (HEI), as well as the accompanying paper by Henneman et al. (2019).

2.1. Air pollution and meteorological data

Data on air quality and meteorology for the period 1999–2013 were obtained from the Southeastern Aerosol Research and Characterization Study (SEARCH) site at Jefferson Street, roughly two miles northwest of downtown Atlanta; details on these measurements have been previously reported (Edgerton et al., 2005; Hansen et al., 2003, 2006). This site is located in approximately the geographic and population center of the 5-county metropolitan area. Hourly data were converted to daily values using previously established metrics (Henneman et al., 2017a, 2017b, 2015). Observed concentrations were obtained for nine pollutants of interest: ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), fine particulate matter ($PM_{2.5}$), and the following $PM_{2.5}$ components: sulfate (SO_4), nitrate (NO_3), organic carbon (OC), and elemental carbon (EC). For periods of missing meteorological data, observations from Hartsfield-Jackson International Airport (roughly 10 miles from Jefferson Street) were used.

2.2. Selection of relevant pollution control policies

Based on input from air quality stakeholders (i.e., government policy-makers and persons involved in regulatory implementations), we identified six sets of pollution control policies that affected emissions in the Atlanta area over the 1999–2013 period. For a given policy scenario, national programs related to mobile sources and electricity generating units (EGUs) were considered in tandem with state or municipal regulatory rules employed to meet the appropriate standards. Three sets of policies regulated emissions from EGUs:

- Acid Rain Program (ARP) and the Georgia Rules for Air Quality Control state program yy ($GRAQC_{yy}$)
- NO_x Budget Trading Program (NBP) and associated State Implementation Plan (SIP) Call and $GRAQC_{jjj}$
- Clean Air Interstate Rule (CAIR) and $GRAQC_{ss}$

Three additional sets of policies regulated emissions from mobile sources:

- Inspection and Maintenance programs
- Tier 2 Gasoline Program and Georgia Gasoline Marketing Rule ($GRAQC_{bbb}$)
- Heavy Duty Diesel Rule

For the counterfactual analyses, we considered nine scenarios: the six scenarios representing the non-implementation of each individual set of policies; the non-implementation of all EGU pollution control policies together; the non-implementation of all mobile pollution control policies together; and the non-implementation of all selected pollution control policies together. Dates when the policies included in each scenario were considered to impact pollutant concentrations are shown in Supplementary Table 1. Additional description of each policy is has been published previously.

2.3. Estimation of counterfactual daily air pollutant concentrations

Methods for modeling of counterfactual daily air quality levels in Atlanta during 1999–2013 have been previously described. Briefly, this involves three steps: 1) estimating counterfactual emissions in the absence of pollution control policies based on observed EGU emissions and modeled mobile source emissions; 2) evaluating relationships between emissions and ambient pollutant levels by regression analysis involving emission and meteorology variables and interaction terms to account for chemical transformation of pollutants; and 3) combining emissions-air quality relationships and counterfactual emissions to estimate daily counterfactual ambient pollutant levels that would have occurred in the absence of pollution control policies accounting for meteorology and other factors. For each of these steps, we took care to account for the uncertainty inherent in the use of predictive models to estimate counterfactual outcomes.

2.4. Hospital emergency department (ED) data

Data were collected from 42 acute care hospitals on ED visits for residents of Atlanta between January 1, 1999 and December 31, 2013. Patient-level computerized billing records were pulled; information included date of admission, the primary International Classification of Diseases 9th Revision (ICD-9) diagnostic code, date of birth, sex, race, and 5-digit residential ZIP code. ED visits were included if the patient ZIP code was wholly or partially within the five central counties of metropolitan Atlanta (Fulton, DeKalb, Gwinnett, Cobb, Clayton).

Daily counts of ED visits were calculated for combined categories for respiratory and cardiovascular disease, associated with pollution levels in previous reports using the same Atlanta ED data (Krall et al., 2016; Metzger et al., 2004; Peel et al., 2005, 2007; Sarnat et al., 2013).

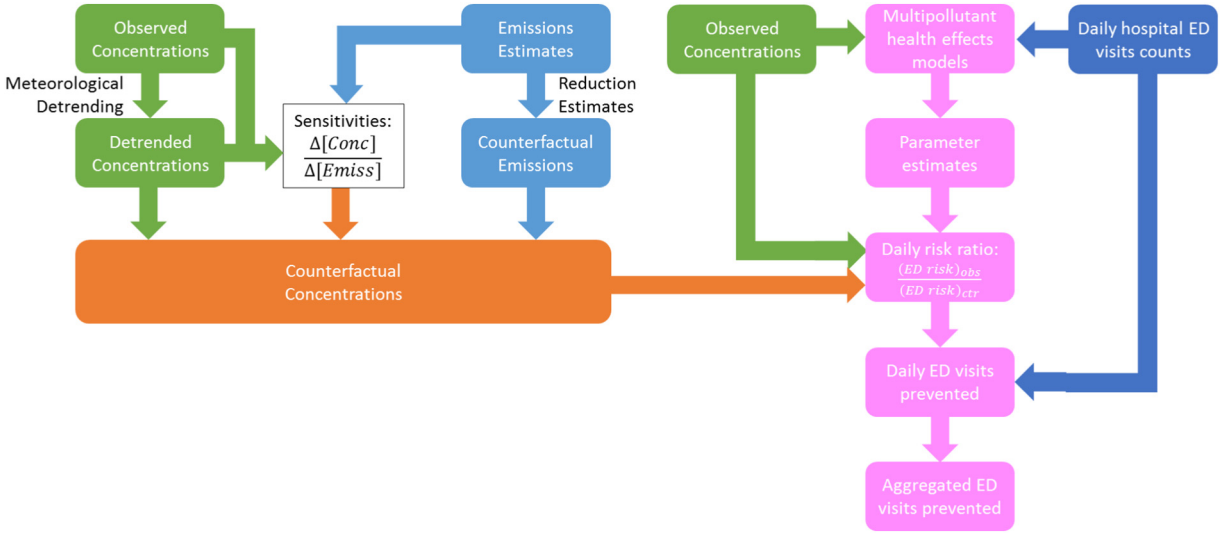

Fig. 1. Outline of steps used in the study. Information from detrended pollutant concentrations and emissions are combined to estimate empirical sensitivities and create daily counterfactual pollutant concentration time series. The associations between daily observed ambient pollutant concentrations and daily counts of hospital emergency department (ED) visits were determined using a multipollutant health effects models. From these models, parameter estimates for relationships between pollutants and ED visits were obtained. These parameter estimates were combined with daily observed and counterfactual pollutant concentrations to produce daily risk ratios representing the observed risk of ED visits compared to the counterfactual risk of ED visits in the absence of pollution control policies. Daily counts of ED visits were then incorporated to generate daily estimates of ED visits prevented, and those were combined to produce aggregated results of ED visits prevented due to pollution control policies.
(Adapted from Russell et al., 2018).

The respiratory disease (RD) outcome group included ED visits for upper respiratory infection (ICD-9 codes: 460–465, 466.0, 477), bronchiolitis (ICD-9 codes: 466.1, 466.11, 466.19), pneumonia (ICD-9 codes: 480–486), chronic obstructive pulmonary disease (ICD-9 codes: 491, 492, 496), and asthma (ICD-9 codes: 493, 786.07). The cardiovascular disease (CVD) outcome group included ED visits for ischemic heart disease (ICD-9 codes: 410–414), cardiac dysrhythmia (ICD-9 code: 427), congestive heart failure (ICD-9 code: 428), and peripheral and cerebrovascular disease (ICD-9 codes: 433–437, 440, 443–445, 451–453). We also examined daily ED visits two specific outcomes of prior interest within the combined outcome groups, asthma and congestive heart failure (CHF).

2.5. Multipollutant health model

We used Poisson log-linear models accounting for overdispersion to estimate the joint effect of multiple pollutants on ED visits in a time-series framework, controlling for potential temporal confounders. Seven pollutants were included in the model: $PM_{2.5}$, O_3, CO, SO_2, NO_2, OC, and NO_3. EC and SO_4 were not included due to concerns of collinearity: daily EC levels were highly correlated with OC ($r = 0.80$) and SO_4 was highly correlated with $PM_{2.5}$ ($r = 0.79$); models containing OC and $PM_{2.5}$ fit better than ones with EC and SO_4 (as measured by the Akaike Information Criterion). To account for potential nonlinear relationships and interaction between pollutants, we used cubic polynomials for each pollutant along with pairwise interactions between all linear pollutant terms.

In prior work, we have analyzed the association between ambient pollution and ED visits using the same Atlanta ED data (Darrow et al., 2011; Gass et al., 2015; Krall et al., 2016; Metzger et al., 2004; Peel et al., 2005, 2007; Sarnat et al., 2010, 2013; Strickland et al., 2010, 2011, 2016; Tolbert et al., 2007; Winquist et al., 2012, 2014, 2016). Based on previous results, we selected 3-day moving averages (lag 0–2: average of pollutant levels same-day, 1 day prior, and 2 days prior) *a priori* as the relevant exposure values for RD and asthma ED visits (Peel et al., 2005; Sarnat et al., 2013; Strickland et al., 2010; Tolbert et al., 2000, 2007). For CVD and CHF ED visits, same-day pollutant values (lag 0) were used based on previous work (Metzger et al., 2004; Ye et al., 2016). Previous analyses also identified important covariates and model parameterizations to provide sufficient control of potential temporal confounders, and these were included *a priori*. We included indicator variables for year, month, and the interaction term year*-month to control for long-term time trends and seasonality. We also included indicator variables for day-of-week and its interaction with month. Meteorology covariates included temperature (cubic polynomial of the daily maximum temperature and the cubic polynomial of the lag 1–2 daily minimum temperature), dew point (cubic polynomial of the lag 0–2 moving average of daily mean dew point), and interaction terms between the same-day maximum temperature cubic polynomials and season. As data were not available for all 42 hospitals over the entire study period, an indicator variable was included for each hospital to account for changes to ED visit totals attributable to hospital data availability. Indicator variables were used as well for holidays and other dates that may have distinct pollutant or ED profiles such as the day after Thanksgiving. The equation for the health impact model is shown in Supplemental Fig. 1.

Initial analyses showed that there were stronger observed concentration-response relationships between pollutants and ED visits in the first half of the study period (roughly 1999–2005) compared to the latter half. If the change in observed associations between pollutants and ED visits in Atlanta over the study period was in part attributable to regulatory actions, then the impact of pollution control policies is more appropriately estimated using associations from the early study period. For example, there has been a significant reduction in anthropogenically-derived $PM_{2.5}$, leaving a larger fraction due to secondary organic aerosol (SOA) from biogenic emissions, e.g., isoprene; recent studies have found isoprene-derived SOA to have a lower oxidative potential than mobile-source derived organic aerosol (Tuet et al., 2017a, 2017b). For our primary analyses, we therefore used parameter estimates from health models of 1999–2005 data to estimate daily risk ratios and health benefits, as described below.

2.6. Health impact analysis

The goal for the health impact analysis was to estimate the number and percentage of cardiorespiratory ED visits that the implementation of air pollution policies prevented, accounting for uncertainty. These estimates were specific to the policy, the ED outcome, and the time period of interest. The daily counterfactual time series allowed us to estimate health effects assuming that only pollutant levels differed from observed levels while all other factors (e.g., meteorology, temporal trends, and total population at risk) remained the same. Using the parameter estimates derived from the multipollutant pollutant health model, the steps to obtain estimates for the health impact analysis were as follows:

1) Apply the relevant parameter estimates to the difference between observed daily pollutant levels and counterfactual daily pollutant levels to produce estimated daily risk ratios for the joint effect on ED visits from multiple pollutants (the risk ratios represent risk of ED visits in the presence of policies to the risk of ED visits in the absence of the policies).
2) Divide the number of daily observed ED visits by the corresponding daily risk ratio to obtain an estimated daily count of ED visits that would have been observed in the absence of the air pollution policies under study.
3) Sum the daily counts obtained in the previous step over the time period of interest to obtain the total counterfactual counts of ED visits over that time period.
4) Estimate the percentage of ED visits prevented over the time period by comparing the total ED counts actually observed for the specified ED outcome with the estimated ED counts under the counterfactual scenario.

The selected air pollution policies were initiated at various points during the study period (1999–2013) and gradually implemented; none were realized instantaneously. In addition to estimating the growing health impacts with time over the entire study period, we provide additional focus on ED visits prevented over the end of the study period, when the air pollution policies were more fully implemented. Since estimated effects over individual years were not highly stable, we used ED visits prevented over the final two years of the study (2012 − 2013) as a measure of the most fully realized impact of these policies.

2.7. Accounting for uncertainty

While models may provide reasonable approximations of real-world scenarios, responsible research involves quantifying uncertainty in estimates based on such models. This study involves multiple modeling steps: estimating pollutant emissions under different policy scenarios, estimating counterfactual daily time series of ambient concentrations for each pollutant under modified emissions levels, and estimating health outcomes for these counterfactual pollutant levels. Typically, estimates of uncertainty of air pollution health effects (i.e., confidence interval estimates) are based solely on the estimation of the model parameters representing the health effects. In this study, we quantified uncertainties at each step in the accountability chain and passed the uncertainties to each proceeding link. For three scenarios – the non-implementation of all EGU pollution control policies; the non-implementation of all mobile pollution control policies; and the non-implementation of all selected pollution control policies – we were able to account for uncertainty in the estimation of pollutant concentrations. We used Monte Carlo simulations to incorporate these sources of uncertainty in health impact analyses.

Henneman et al. (2019) describes the varied sources of uncertainty addressed in the estimation of counterfactual pollutant concentrations and identifies two primary sources of uncertainty: uncertainty in the base year emissions factors and uncertainty in the emissions-concentration relationships developed in statistical models. Daily counterfactual EGU emissions were estimated by using the mean base year emissions ratios (Russell et al., 2018); therefore, we accounted for uncertainty by sampling from a normal distribution with the mean and variance of measured daily emissions ratios during the base year. For daily counterfactual mobile emissions uncertainty, we utilized the established approach of sampling from a uniform distribution between ± 50% estimated change in emissions due to pollution control policies (Napelenok et al., 2008). For uncertainty in the model linking emissions to pollutant concentrations, we used the estimated variances of the modeled regression coefficients as well as the covariances between each variable to repeatedly sample from a multivariate normal distribution of possible coefficient values. An additional potential source of uncertainty, the attribution of emissions changes to specific regulations, is discussed by Henneman et al. (2019); they conclude that mobile emissions change attribution generally leads to more uncertainty than EGU due to uncertainties in the MOVES model, but that the uncertainties imparted on both EGU and mobile counterfactuals are not larger than the uncertainty estimates applied in the final analysis.

For uncertainty in linking concentrations with health outcomes, we sampled from a multivariate normal distribution of regression coefficients based on the estimated variance-covariance structure. These samples were used to generate daily risk ratios and daily numbers of ED visits prevented, which were then aggregated to produce overall estimates of ED visits prevented through pollution control policies. We obtained 5000 samples at each step and took the 2.5th and 97.5th percentiles of ED visits prevented to represent the 95% interval estimate that incorporates both the uncertainty in the health impact model parameters and the uncertainty in the estimation of the counterfactual time series for each pollutant in the health model.

2.8. Sensitivity analyses

To assess the influence of modeling choices on estimated ED visits prevented, we conducted several sensitivity analyses. First, we ran the multipollutant health model using the entire 1999–2013 dataset to estimate regression coefficients for the concentration-response relationships. Also, for comparison with our seven-pollutant primary model which contained both cubic polynomials and interaction terms, we ran the health model with only linear pollutant terms, with cubic polynomials but no interaction terms, and with linear terms and interaction terms. We additionally ran health models with different sets of pollutants included: a full model with all nine pollutants; a single-pollutant model with $PM_{2.5}$ (since this pollutant measure is a mixture affected by a number of different sources and was strongly associated with cardiorespiratory outcomes in our data); and a model with five EPA NAAQS pollutants included in this study ($PM_{2.5}$, O_3, CO, SO_2, and NO_2). Finally, we ran health models using ED visits from the full 20-county ANAA to evaluate health impacts over a larger geographic area.

2.9. Statistical programs utilized

All analyses in the health impact modeling were performed using SAS version 9.3 (SAS Institute, Cary, NC) and R version 3.01 (The R Foundation for Statistical Computing, 2013).

3. Results

Observed annual average concentrations decreased for all measured pollutants from 1999 to 2013 (Fig. 2a). The greatest drop was for SO_2, with 2013 concentrations being only 9.2% of 1999 levels, while O_3 changed the least with 2013 concentrations being 82.6% of 1999 levels; summertime decreases in O_3 concentrations were accompanied by wintertime increases (Henneman et al., 2015). For all other pollutants examined, 2013 concentrations ranged from 27.8% - 53.8% of 1999 levels. Counterfactual 2013 concentrations under the non-

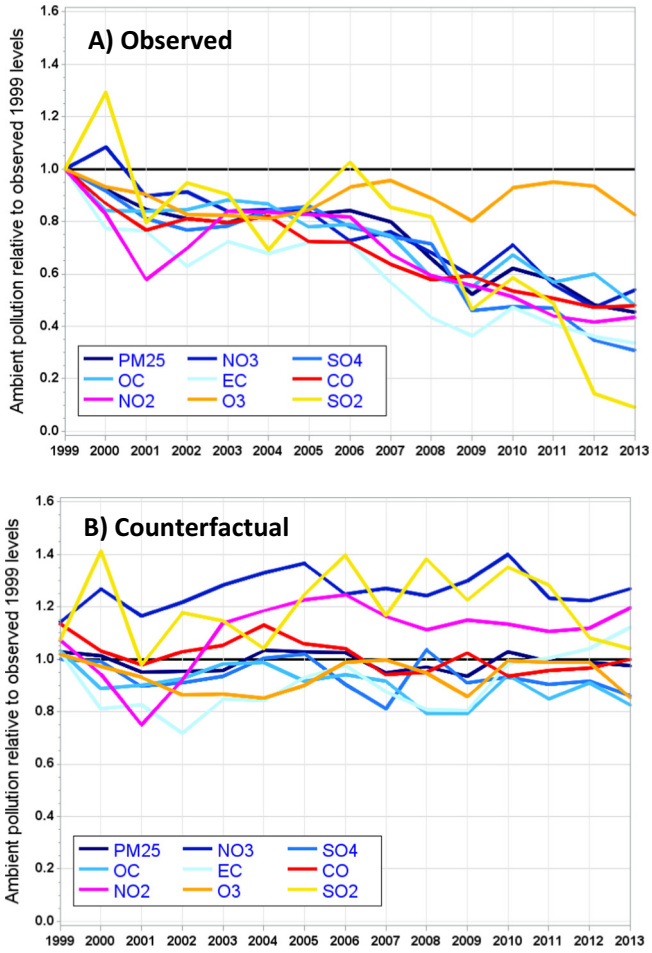

Fig. 2. Annual mean ambient levels for nine pollutants, relative to observed 1999 levels, A) observed and B) counterfactual scenario of non-implementation of all selected pollution control policies.

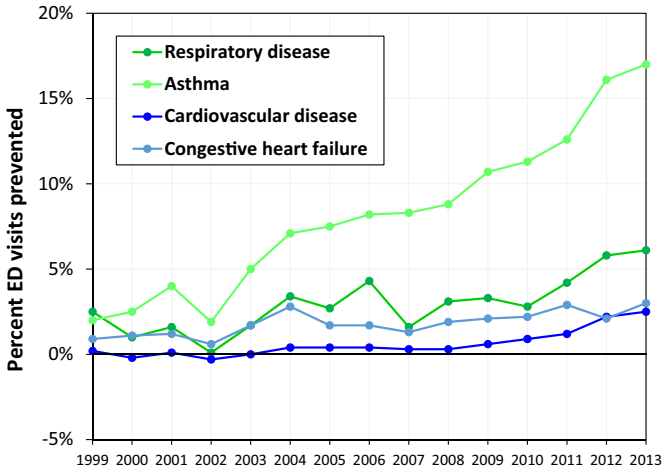

Fig. 3. Percent emergency department (ED) visits prevented by all selected pollution control policies by outcome and year in the 5-county Atlanta metropolitan area, 1999–2013.

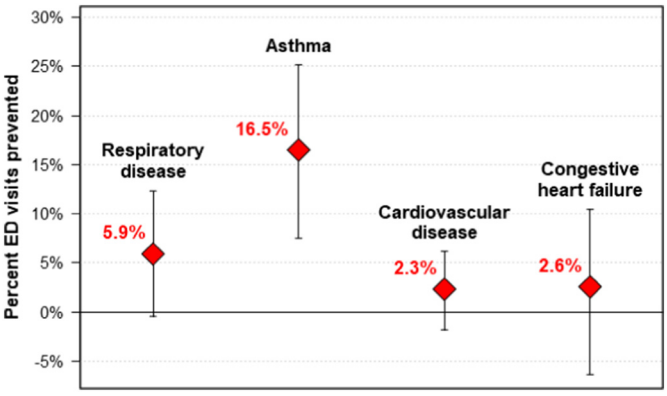

Fig. 4. Percent emergency department (ED) visits prevented by all selected pollution control policies by outcome, 5-county Atlanta metropolitan area, 2012–2013.

implementation of all pollution control policies ranged from 77.2% - 125.3% of observed 1999 levels (Fig. 2b). In all instances, observed annual average concentrations from 2000 to 2013 were lower than counterfactual levels under the non-implementation of all pollution control policies.

There were 16,191,785 total ED visits in our database for the 5-county Atlanta metropolitan area from 1999 to 2013, an average of just over a million ED visits per year. The RD group included 1,637,338 ED visits, of which 374,126 were for asthma; the CVD group included 416,392 ED visits, of which 105,561 were for CHF.

Following the steps outlined in Fig. 1, we estimated the percent of ED visits prevented by the implementation of pollution control policies. The estimated percent of ED visits prevented by all selected pollution control policies gradually increased over time as the impacts of pollution control policies became more fully realized (Fig. 3). Using model parameters from the first half of the study period (1999–2005), the estimated annual percent of RD ED visits prevented increased from 2.5% in 1999 to 6.1% in 2013; for asthma, the annual percent of visits prevented increased from 2.0% to 17.0%; for CVD, the annual percent of visits prevented increased from 0.2% to 2.5%; and for CHF, and annual percent of visits prevented increased from 0.9% to 3.0%.

Together, the selected air pollution control policies were estimated to have prevented over 55,000 cardiorespiratory ED visits in the five-county metropolitan area over the entire study period (1999–2013), compared to what would have occurred in the absence of the pollution control policies. For the final two years of the study period (2012, 2013), when the control policies were most fully implemented, we estimated that these policies prevented 5.9% of all RD ED visits, 95% CI (−0.4%, 12.3%); 16.5% of asthma ED visits, 95% CI (7.5%, 25.1%); 2.3% of CVD ED visits, 95% CI (−1.8%, 6.2%); and 2.6% of CHF ED visits (−6.3%, 10.4%) (Fig. 4).

EGU policies, particularly the NO_x Budget Trading program and the Acid Rain program, generally had a greater estimated health impact in preventing asthma and RD ED visits than mobile source policies (Supplemental Fig. 2). For CVD ED visits, the Clean Air Interstate Rule/multipollutant program had the largest estimated impact of any set of policies. For all outcomes, there was substantial overlap in the interval estimates for the majority of pollution control scenarios. The uncertainty in the health effect results was driven largely by the uncertainty estimation from the health impact model, suggesting that uncertainty in attributing changes in emissions and pollutant levels to individual regulations was of smaller consequence to the overall results.

In sensitivity analyses, estimates for ED visits prevented were lower for all four outcomes when using the entire 1999–2013 period to fit concentration-response parameter estimates (Supplemental Fig. 3). Using the entire study period for model fitting, we estimated that these policies prevented 0.6% of all RD ED visits, 95% CI (−3.7%, 6.1%); 6.8% of asthma ED visits, 95% CI (−0.3%, 13.9%); 1.8% of CVD ED visits, 95% CI (−1.0%, 4.6%); and 1.8% of CHF ED visits (−3.5%, 7.7%). The inclusion of cubic polynomial terms and interaction terms improved model fit and increased the estimates of ED visits prevented for RD and asthma, but results for CVD and CHF were less affected (Supplemental Fig. 4). The 1-pollutant, 5-pollutant, and 9-pollutant

models all estimated fewer ED visits prevented than the primary 7-pollutant model, though all models showed significant reduction in asthma ED visits due to pollution control policies (Supplemental Fig. 5). Slightly smaller percentages of ED visits were estimated to be prevented over the 20-county ANAA compared to the 5-county Atlanta metropolitan area (Supplemental Fig. 6).

4. Discussion

The current study takes advantage of a 15-year record of speciated ambient air pollutant measurements and daily ED visit data to evaluate effects of regulatory actions on emissions changes, ambient air quality, and health impacts. Here, we produce counterfactual time series of daily estimated air pollutant concentrations estimated to have occurred had selected regulations not been implemented (Henneman et al., 2017a, 2017b, 2015). Multi-pollutant health modeling was used to relate pollutant concentrations with ED visits for specific health endpoints, to estimate the number of visits prevented each day, and then estimate the total number of visits prevented and the percent of ED visits prevented. We took care to account for uncertainty in the counterfactual pollutant concentrations as well as in the health model concentration-response estimates. Altogether, the extensive data available and method contribute to making this one of the most comprehensive air pollution accountability studies to date.

This approach yielded evidence that pollution control policies in the Atlanta area were effective in reducing ambient pollutant levels and, in turn, cardiorespiratory ED visits. These impacts were strongest in the later years of the study period, after the pollution control policies had been most fully implemented. Observed concentrations decreased for the nine measured pollutants that we examined from 1999 to 2013, with reductions of mean daily pollutant levels over the study period ranging from 17% for ozone to 91% for SO_2. These reductions are even more impressive when considering that the Atlanta 5-county metropolitan area grew in population by 24% between 1999 and 2013 (U.S. Census Bureau, 2017). Estimated health impacts were highest for asthma and other respiratory disease, and more modest for cardiovascular diseases. Overall, EGU policies showed the greatest impact, but the uncertainties are greater in comparing individual policies, and it should be kept in mind that some policies on mobile sources are slower to achieve impact due to dependence on fleet turnover.

Due to logistical and ethical concerns of experimental studies, assessments of the effects of pollution control interventions often utilize observational study designs framed as natural experiments (Rich, 2017). Natural experiments necessitate a spatial and/or a temporal contrast that should be comprehensively similar to the intervention group in all potentially relevant factors. To utilize a spatial contrast, researchers would need to identify two populations (or groups of populations) similar in terms of population size, population demographics, risk factors for disease, ED usage, meteorology, baseline pollution levels, etc., except that the control population would not be subject to the air pollution control policies that the intervention group experienced during the study period. Realistically, it is highly unlikely to find two populations alike in every meaningful way, thus confounding could cause biased estimates of health impacts (Chen et al., 2013; Dominici and Zigler, 2017; Parker et al., 2008). Alternatively, researchers may identify a single population exposed to a single well-defined intervention or set of interventions; such a situation could be amenable to pre-post analysis (Gauderman et al., 2015; Hedley et al., 2002; Su et al., 2015). This approach to approximate the counterfactual is also problematic, however, as effects of interventions are often not immediately fully realized. Population changes, secular changes in incidence of the outcome, and other external factors that could affect either pollution levels or health outcomes may bias study results. The current study had numerous factors that could have substantially biased pre-post analytic results: the length of the intervention period, demographic changes in the Atlanta metropolitan area, the occurrence of events such as the economic recession, and the assessment of numerous air pollution policies instigated at different time points. Importantly, these policies were gradually implemented over time (e.g., engine emissions standards for new cars were phased in over several years and realization of their impact depends on fleet turnover), and the lack of clearly defined control and intervention periods would have been extremely problematic for pre-post analysis.

In recent work, Zigler et al. (2018) employed propensity score grouping to compare areas in the Eastern United States that were in attainment to areas that were not in attainment of the 1997 NAAQS for $PM_{2.5}$. Grouping by propensity score is designed to approximate what would occur in a successfully randomized experiment by matching for a number of measured confounders. But it is precisely the control of unmeasured confounding that provides a randomized design with its key advantage over observational studies. The comparison of areas in the United States that were in attainment to those not in attainment is plausible because there is within-country variation of this factor. Our goal in the present study, on the other hand, was to estimate the consequence of national policies implemented at various geographic scales, which makes utilizing discrete geographical comparisons less feasible.

Rather than use a proxy city or proxy time period to represent the counterfactual levels of air pollution in Atlanta, our approach was to directly estimate daily counterfactual levels for selected pollutants in Atlanta during the study period, incorporating changes in emissions with meteorology and atmospheric chemistry and accounting for model uncertainty. The validity of such an approach depends upon how well the counterfactual air pollution levels are estimated. We employed Monte Carlo simulations to obtain lower and upper confidence limits to account simultaneously for the uncertainty in both the estimation of the counterfactual pollution levels and the estimation of the health effects.

A number of considerations were also given to the health modeling component of the project. Our primary health models included linear, quadratic, and cubic terms for each of seven pollutants as well as all pairwise pollutant interactions. Determining the exact nature of interactions between this many pollutants is fraught with difficulty and beyond the scope of this study; we ultimately decided on the conservative approach of including all these polynomial and interaction terms *a priori*, even if this reduced estimate precision. Such complex modeling of air pollution is unusual in air pollution-health studies and the interpretation of such pollution concentration-response functions is not straightforward. However, our goal was not to interpret pollutant concentration-response functions, nor was it to “tease out” single pollutant effects from multipollutant health models. Rather, our goal was to estimate joint pollutant effects for specified contrasts, i.e., the contrast of counterfactual vs observed pollution levels for multiple pollutants each day of the study period. This approach enabled us to most fully capture the health effects of diverse ambient pollutant profiles without making assumptions about the causal basis for each pollutant term, allowing for associations that either were null or represented surrogates for unmeasured variables.

To reduce concerns about multiple comparisons, we used *a priori* choices for key modeling decisions. We used *a priori* lag structures determined from previous research. When considering interaction, we decided to either include all possible pairwise pollutant product terms or no product terms rather than include a subset of product terms that yielded the most significant joint effect. Similarly, we decided to include all linear, quadratic, and cubic pollutant terms representing each pollutant in the model or just linear pollutant terms. We also made *a priori* choices for the control of time trend and meteorology based on previous research. We did not fix the number of pollutants in our health models *a priori* as we did not know to what degree multipollutant correlation might lead to unstable joint effect estimates. The multipollutant correlation motivated our use of multipollutant health models for valid estimation of joint pollutant effects. For transparency, we included sensitivity analyses to show the effects of our modeling decisions.

We observed stronger associations between pollutants and cardiorespiratory ED visits in the first half of the study period (roughly 1999–2005) compared to the latter half. This result led us to consider not only how air pollutant levels were affected by specified pollution control policies but also whether the policies themselves may have affected the concentration-response functions. If so, estimates of the counterfactual number of ED visits would be biased by using concentration-response parameters that were affected by air pollution control policies. In fact, there have been accountability studies in which the results were driven by the estimation of changes in risk ratios over time rather than the absolute reduction in air pollution over time, as in our study; an example of such a study is the accountability study using data from Erfurt, Germany (Peters et al., 2009). In that study, the authors speculate that changes in the associations between pollutants and mortality rates could have been at least partially caused by changes in pollutant sources or air pollution mixtures, both of which were affected by environmental controls and other industrial changes. Changes in the estimated health associations over time could also be due to extraneous factors unrelated to control policies such as changing population susceptibility, model misspecification, or chance. While we present results using the concentration-response parameters from the entire 1999–2013 period in the supplement, for our primary model we use the concentration-response parameters from the 1999–2005 period because of our concern that the change in the parameters over time were, at least in part, a consequence of the air pollution control policies under study.

The implementation of the air pollution policies of interest in Atlanta occurred gradually over the 1999–2013 period. Air pollution reduction increased substantially over the course of the study period. As such, we would expect the health impacts to increase over the study period and be greatest in the later years. One advantage of the counterfactual study design compared to a pre-post analysis is that it accounts for the gradual implementation of pollution policies. We present the estimated percent of ED visits prevented by control policies by outcome and year in the 5-county Atlanta metropolitan area from 1999 to 2013, and we also present the estimated percent of ED visits prevented by control policies by outcome for the 2012–2013 period. The 1999–2013 estimates of ED visits prevented quantify health impacts over the time period that the policies gradually became implemented. On the other hand, the 2012–2013 analyses assess health impacts during the time period when the health policies were most fully implemented. While both avenues of research are interesting, the second may be more relevant if pollution policies are sustained in future years.

The study focused on cardiorespiratory ED visits, a subset of the overall expected health benefits of air pollution reduction. Ambient air pollution has also been linked to other health endpoints such as adverse pregnancy outcomes as well as chronic diseases such as lung cancer. (Gotschi et al., 2008; Laden et al., 2006; Pope et al., 2004; Raaschou-Nielsen et al., 2013; Stafoggia et al., 2014) As such, the public health benefits of the air pollution interventions are likely underestimated in the current study. Furthermore, while this study estimated the number of ED visits in Atlanta prevented by pollution control policies from 1999 to 2013, health benefits will continue to be realized as long as the policies studied continue to be implemented.

Regulatory costs motivate air pollution accountability research, which evaluates impacts of air quality regulations on emissions, air quality, exposure, and public health – components of what has been referred to as the "accountability chain" (Health Effects Institute Accountability Working Group, 2003). The use of the accountability chain to determine the effect of pollution control policies on health necessitates that the links within the chain are truly causal. An extensive literature – both observational and experimental – provides substantial evidence in support of causal relationships between pollution control policies and pollutant emissions, between emissions and air quality, and between air quality and health outcomes. The interpretation of our results as a measure of the protective health impacts of pollution control policies is strongly supported by this existing literature.

5. Conclusion

Pollution control policies in the Atlanta area were effective in reducing ambient pollutant levels and cardiorespiratory ED visits, and these impacts were stronger in later years after the pollution control policies had been more fully implemented. The results of this study and future studies employing similar methods can help inform the development, implementation and assessment of planned pollution control strategies.

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Declaration of competing financial interests

No competing financial interests declared.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2019.01.052.

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