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Triggering of cardiovascular hospital admissions by fine particle concentrations in New York state: Before, during, and after implementation of multiple environmental policies and a recession☆

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ABSTRACT

Background: Previous studies reported triggering of acute cardiovascular events by short-term increasedPM$_{2.5}$ concentrations. From 2007 to 2013, national and New York state air quality policies and economic influences resulted in reduced concentrations of PM$_{2.5}$ and other pollutants across the state. We estimated the rate of cardiovascular hospital admissions associated with increased PM$_{2.5}$ concentrations in the previous 1–7 days, and evaluated whether they differed before (2005–2007), during (2008–2013), and after these concentration changes (2014–2016).

Methods: Using the Statewide Planning and Research Cooperative System (SPARCS) database, we retained all hospital admissions with a primary diagnosis of nine cardiovascular disease (CVD) subtypes, for residents living within 15 miles of PM$_{2.5}$ monitoring sites in Buffalo, Rochester, Albany, Queens, Bronx, and Manhattan from 2005 to 2016 (N = 1,922,918). We used a case-crossover design and conditional logistic regression to estimate the admission rate for total CVD, and nine specific subtypes, associated with increased PM$_{2.5}$ concentrations.

Results: Interquartile range (IQR) increases in PM$_{2.5}$ on the same and previous 6 days were associated with 0.6%–1.2% increases in CVD admission rate (2005–2016). There were similar patterns for cardiac arrhythmia, ischemic stroke, congestive heart failure, ischemic heart disease (IHD), and myocardial infarction (MI). Ambient PM$_{2.5}$ concentrations and annual total CVD admission rates decreased across the period. However, the excess rate of IHD admissions associated with each IQR increase in PM$_{2.5}$ in previous 2 days was larger in the after period (2.8%; 95%CI = 1.5%–4.0%) than in the during (0.6%; 95% CI = 0.0%–1.2%) or before periods (0.8%; 95%CI = 0.2%–1.3%), with similar patterns for total CVD and MI, but not other subtypes.

Conclusions: While pollutant concentrations and CVD admission rates decreased after emission changes, the same PM$_{2.5}$ mass was associated with a higher rate of ischemic heart disease events. Future work should confirm these findings in another population, and investigate whether specific PM components and/or sources trigger IHD events.

1. Introduction

Previously, we and others have reported that short-term increases in ambient fine particle concentrations were associated

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with triggering of acute cardio- and cerebrovascular events, including myocardial infarction (Mustafić et al., 2012; Pope et al., 2015), ischemic stroke (Shah et al., 2015), cardiac arrhythmia (Link et al., 2013; Peters et al., 2000; Rich et al., 2006a, 2005; 2006b), and heart failure (Shah et al., 2018). For example, in Rochester, New York, each 7.1 $\mu g/m^3$ increase in $PM_{2.5}$ in the previous hour was associated with an 18% increase in the risk of a ST-elevation myocardial infarction (OR = 1.18; 95% CI = 1.01, 1.38) (Gardner et al., 2014). Others have reported similar findings (Akbarzadeh et al., 2018; Argacha et al., 2016; Liu et al., 2018; Pope et al., 2015; Zhang et al., 2016).

Since the early-to mid-2000s, policy initiatives to improve air quality have been implemented nationally and across New York State (NYS). These initiatives include use of ultra-low sulfur on-road diesel fuel starting in October 2006, the requirement for particle regenerative traps on new heavy-duty diesel on-road trucks and buses on July 1, 2007, the requirement for NOx control as of January 1, 2010, decreases in the sulfur content of non-road diesel fuel between 2007 and 2014, and the requirement that all No. 2 based fuels sold in NYS be ultra-low sulfur by July 1, 2012. Additionally, several actions (e.g. emission control technology retrofits in the Ohio River Valley, Cross-State Air Pollution Rule, and electricity policy changes in Ontario) have occurred during this same time to reduce SO_2 and NOx emissions from power plants in upwind source areas. During this period, there were also major economic drivers of changes in air quality including the 2008 recession and the change in the price of natural gas compared to coal and oil (Squizzato et al., 2018).

Squizzato et al. (2018) reviewed the spatial and temporal patterns of all the gaseous pollutants and particulate matter measurements made between 2005 and 2016 at every New York State Department of Environmental Conservation (NYS DEC) site in NYS. Not all pollutants are measured at every site. They reported that $PM_{2.5}$ concentrations decreased significantly at all sites for which there are data (−2.2% per year to −8.6% per year across sites). Similarly, SO_2 concentrations dropped significantly at all sites within this period, with the highest reductions observed at the urban sites (e.g., −8.5% per year at Queens, New York City). Reductions in NOx emissions contributed to the reduction in high ozone episodes during summers. However, no reductions were found for the maximal spring ozone concentrations across the state. There were observed increases in autumn and winter ozone concentrations (e.g., 6.6 ± 0.4% per year across New York City). Thus, the decrease in $PM_{2.5}$ and inorganic precursor gases (SOx and NOx) in Rochester (Emami et al., 2018) and all of NYS over the past 12 years (Squizzato et al., 2018) suggest that the composition of $PM_{2.5}$ has likely changed during this period. Thus, whether these PM composition changes affected the rate of acute cardiovascular events associated with increased ambient $PM_{2.5}$ concentrations may provide clues as to the PM sources/mixtures that most strongly trigger acute cardiovascular events.

Using the New York State Department of Health Statewide Planning and Research Cooperative System (SPARCS), a legislatively mandated database of NYS hospital discharges, and daily ambient $PM_{2.5}$ measurements made at multiple monitoring sites across NYS, we examined the association between short-term increases in $PM_{2.5}$ concentration and hospital admissions for acute cardiovascular events across NYS. We then evaluated whether that relative rate differed before (2005–2007), during (2008–2013), and after (2014–2016) these environmental policies/actions were implemented and economic impacts occurred. We hypothesized that short-term increases in $PM_{2.5}$ concentrations would be associated with an increased rate of total and cause-specific cardiovascular outcomes. We also hypothesized that these relative rates would be smaller after implementation of these environmental actions and policies, the influence of the recession and cost-driven changes in the fuel used for electricity generation, and the resulting reductions in $PM_{2.5}$ and most other pollutant concentrations across the state.

2. Materials and methods

2.1. Study population and hospital admissions data

Information on hospital admissions were obtained from the inpatient SPARCS database, which we have used previously (Fitzgerald et al., 2014; Garcia et al., 2011; Jones et al., 2013, 2015, Lin et al., 2012, 2016, 2004, 2005, 2008a, 2008b, 2009, 2010). SPARCS is a legislatively mandated database that covers ~95% of hospitals in NYS, excluding federal facilities (e.g. Veterans Affairs Hospitals) and psychiatric facilities. It contains billing and medical information on over 2.5 million discharges for NYS hospitals, 1.5 million ambulatory surgery center visits, and 6.5 million emergency department visits per year. SPARCS data include patient information on the principal diagnoses and up to 24 other diagnoses at the time of hospital admission, as well as demographic characteristics and event/hospital information including date of birth, gender, race, ethnicity, street address, admission date, source of payment, and length of stay. Data reported by hospitals are reviewed by SPARCS administrative staff for accuracy and completeness. We geocoded the residential address for each hospitalization using the Street and Address Maintenance Program in ArcGIS 10.3.1 (The NYS GIS Program Office, 2017).

We retained those hospital admissions with a “principal” diagnoses of cardiovascular disease (defined using ICD-9 and ICD-10 codes) and an admission date from January 1, 2005 to December 31, 2016, for adult (≥18 years of age) residents of NYS. From this primary diagnosis, we defined total cardiovascular disease as ICD-9 = 393–396, 401–405, 410–415, 427–428, 430–434, 436–438, and ICD-10 = I05-I08, I10-I16, I20-I25, I47-I49, I42, I50-I51, I60-I69, I26-I27. In a similar manner, we defined cardiac arrhythmias (ICD-9 = 427 and ICD-10 = I47-I49), cerebrovascular disease (ICD-9 = 430–434 and 436–438 and ICD-10 = I60-I69), chronic rheumatic heart disease (ICD-9 = 393–396 and ICD-10 = I05-I08), congestive heart failure (ICD-9 = 428 and ICD-10 = I42 and I50-I51), hypertension (ICD-9 = 401–405 and ICD-10 = I10-I16), ischemic heart disease (ICD-9 = 410–414 and ICD-10 = I20-I25), myocardial infarction (ICD-9 = 410 and ICD-10 = I21), and ischemic stroke (ICD-9 = 434 and ICD-10 = I63). We then excluded all study subjects living more than 15 miles from any our six $PM_{2.5}$ monitoring sites in Buffalo, Rochester, Albany, Bronx, Manhattan, and Queens (described below), leaving N = 1,922,918 available for analysis. This study was reviewed and approved by the Institutional Review Board at the State University of New York at Albany.

For use in descriptive analyses, we estimated the population size within each 15-mile buffer around a monitoring station (for 18 age and 8 race categories for males and females separately) for each day during the study period, using population size estimates from each census tract within the 2000 and 2010 Census data and the 2011–2015 American Community Survey. For those census tracts not completely within the buffer, we scaled the population size from that census tract by the proportion of the census tract within the buffer. We then estimated the population size for each day by linear interpolation between the 2000 and 2010 Census and between the 2010 Census and 2011–2015 American Community Survey data; this latter trend was extrapolated until December 2016. The spatial analyses of census tracts were performed using ArcGIS 10.4.1 (ESRI), with linear interpolations computed using R 3.4.0 (https://www.r-project.org/).

2.2. Air pollution and weather data

$PM_{2.5}$ concentrations were retrieved from the USEPA Air Quality System (https://aqs.epa.gov/api) for each of the six urban sites where measurements were made: Buffalo, Rochester, Albany, and New York City (Bronx, Manhattan, Queens). At each site, hourly $PM_{2.5}$ concentrations were measured using tapered element oscillating microbalance (TEOM) monitors (DEC, 2017). In this study, 24-h daily averages were computed for each site and each day for which measurements of at least 75% of the hours that day in that site were available (18 h). TEOM data showed a seasonal bias compared to the $PM_{2.5}$ FRM (federal reference method) measurements. Thus, a non-linear correction based on the Julian Day was applied to adjust the TEOM concentrations to the FRM ones (Felton, 2005; Masiol et al., 2017; Schwab et al., 2006). Missing TEOM data were substituted with FRM concentrations if available. Summary statistics for these sites are presented in Table S1 & S2 in the supplemental material. Data below the detection limit were replaced with detection limit/2.

Hourly temperature and relative humidity data were obtained from the National Weather Service (National Climate Data Center, https://www.ncdc.noaa.gov/cdo-web/datatools/lcd) for the nearest major airport (BUF - Buffalo, ROC - Rochester, ALB - Albany, LGA - Bronx, and JFK - Queens) or the closest weather station (Central Park for Manhattan). For each study subject living within 15 miles of our six monitoring stations, we assigned $PM_{2.5}$, temperature, and relative humidity measurements from the nearest site. If a person lived <15 miles from more than one monitor (e.g. Bronx vs. Manhattan), we assigned concentrations/values to that person from the closest monitor.

2.3. Study design and statistical analyses

In descriptive analyses, we examined the change in $PM_{2.5}$ concentration and cardiovascular hospitalization rates before, during and after policy implementations and the economic changes. The incidence rate was estimated by dividing the number of cases over the estimated population for each year and site.

To estimate the relative rate of cardiovascular disease hospital admissions associated with short term increases in $PM_{2.5}$ concentration, we used the same time-stratified, case-crossover design and conditional logistic regression analyses (Levy et al., 2001; Maclure, 1991) used in previous studies of acute cardiovascular events and PM air pollution (Gardner et al., 2014; Mustafić et al., 2012; Pope et al., 2015; Shah et al., 2015) (Rich et al., 2018; Wellenius et al., 2005a, 2005b). We used this design, instead of a time-series analysis, over concerns that we could perfectly define the source population from which all CV hospitalizations arose for adults residing within 15 km of a monitoring station. Thus, this analysis could have led to biased effect estimates. The case-crossover design, in which this limitation does not lead to biased effect estimates, contrasts pollutant concentrations immediately before the cardiovascular disease admission (case-periods) to other times when the subject did not have a cardiovascular disease admission (control periods), matched to the case-period by calendar month and weekday. First, including all admissions from the six sites in the same model, we fit a conditional logistic regression model for total cardiovascular disease admissions, in which we regressed case–control status (i.e., case = 1, control = 0) against the mean $PM_{2.5}$ concentration, temperature, and relative humidity on the case and control days (lag day 0). To achieve the optimal model fit, we included natural splines for temperature with 4 degrees of freedom (df) and relative humidity with 3 degrees of freedom, which were determined using the Akaike's information criterion (Aho et al., 2014). As is standard in case-crossover studies, from this model we estimated the rate ratio and its 95% confidence interval. The excess rate is the percent increase in the rate per unit of exposure (i.e. [rate ratio − 1.0] * 100%). We then re-ran this model to estimate the rate of total cardiovascular disease admissions associated with increased mean $PM_{2.5}$ concentrations on lag days 0–1, 0–2, 0–3, 0–4, 0–5, 0–6 and for each of the cause-specific cardiovascular outcomes described above, separately. From each model, we scaled the relative rate and 95% confidence intervals by the lag specific interquartile range for the Albany site for the entire study period. Interquartile ranges were from the control periods for each outcome.

Next, using the same data, we estimated the associations between $PM_{2.5}$ and the cardiovascular disease admission rates by period (before = 2005–2007, during = 2008–2013, after = 2014–2016). Specifically, we fit a similar conditional logistic regression model as described above, but now also included indicator variables for the main effect of 'period' (before, during, after) and the 'period' by $PM_{2.5}$ interaction, to allow the $PM_{2.5}$ slope to differ by period. To facilitate a comparison of the relative rate associated with a specified unit increase in $PM_{2.5}$ across periods, we scaled each period-specific relative rate and its 95% confidence interval by the same Albany interquartile range for the entire study period described above. The significance of the difference in the $PM_{2.5}$ slopes across periods was evaluated with the 2 df test for the 'period' by $PM_{2.5}$ interaction. We re-ran this again for each cause-specific cardiovascular outcome, and then again separately for only the 'Upstate' monitoring locations (Buffalo, Rochester, and Albany) and for only the New York City monitoring locations (Bronx, Manhattan, and Queens).

2.4. Sensitivity analyses

To confirm that relative rate estimates from these models using data from all six sites together were robust, we ran separate models for each site to obtain site-specific relative rate estimates and their standard errors. We then pooled the six site-specific estimates into an overall relative rate estimate using fixed-effect models with an inverse-variance weighting method (i.e. meta-analysis method). We also pooled these estimates using random-effect models using a maximum-likelihood estimator (Viechtbauer, 2010). Since results from these two methods were essentially the same, we present pooled estimates using the "meta-analysis" method below. Second, we re-ran the analyses comparing excess rates estimates across periods, scaling those period-specific relative rates by the interquartile range for that period and conducting the test for interaction on these period-specific scaled estimates. Third, we evaluated whether inference differed when excluding 'elective' hospitalizations (i.e. those hospitalizations listed as a 'scheduled admission'). Last, we repeated our main analysis using individual lag days (lag day 0, 1, 2, 3, 4, 5, and 6) rather than averages of multiple lag days (lag days 0, 0–1, 0–2, 0–3, 0–4, 0–5, 0–6) to evaluate whether similar lag patterns were observed. All data management and statistical analyses were done using R version 3.0.1 (https://www.r-project.org/).

3. Results

Subject characteristics, by site, are shown in Table 1. Of the 1,922,918 cardiovascular hospital admissions during the study period, 82.1% were in New York City (Manhattan, Queens, Bronx), with the largest proportion in Manhattan (29.6%; n = 568,933) and smallest proportion in Albany (4.2%; n = 81,250). Across sites, subjects were slightly more often male (50.3%–54.5%), and predominantly 60 years of age and older (69.1%–76.6%). However, in the 3 Upstate sites, 77.2%–80.3% of subjects were white, while only

32.1%–51.6% were white in the 3 New York City sites. The most common co-morbidities were essential hypertension (46.1%–51.0% across sites), disorders of lipoid metabolism (39.2%–48.7%), and other forms of chronic ischemic heart disease (31.2%–46.4%). The largest proportion of admissions occurred in the spring, followed by winter, autumn, and summer. The number of admissions decreased over time across all sites (Table 1).

Across the study period, the incidence rate/year decreased for total cardiovascular disease (before: 15.6 admissions/1000 people per year; during 13.6 admissions/1000 people per year; after: 11.8 admissions/1000 people per year) and cardiovascular disease subgroups including cardiac arrhythmia, congestive heart failure, ischemic heart disease and myocardial infarction (Table 2). Daily $PM_{2.5}$ concentrations also generally decreased over time at all sites for both case and control days, but case day concentrations were slightly higher than control days across all sites (Table 3).

When assessing all six sites together, interquartile range increases in $PM_{2.5}$ concentration in the previous 0–6 days were associated with 0.6%–1.2% increases in the rate of total cardiovascular disease hospital admissions (Fig. 1; Table 4). Similarly, interquartile range increases in $PM_{2.5}$ concentrations across most lag times were associated with small but significantly increased excess rates of hospital admissions for cardiac arrhythmia (largest excess rate for lag days 0–6: 1.5%; 95% CI = 0.9%, 2.2%), ischemic stroke (largest excess rate for lag days 0–1: 1.1%; 95% CI = 0.3%, 1.9%), congestive heart failure (largest excess rate for lag days 0–4: 2.4%; 95% CI = 1.9%, 3.0%), ischemic heart disease (largest excess rate for lag days 0–4 and 0–6: 1.3%; 95% CI = 0.8%, 1.7%), and myocardial infarction (largest excess rate for lag days 0–4: 1.0%; 95% CI = 0.3%, 1.8%), but not cerebrovascular, chronic rheumatic heart disease, hypertension, or pulmonary embolus admissions (Fig. 1; Table 4). In the sensitivity analyses estimating this relative rate separately for each of the six sites and then pooling them via meta-analysis, excess rate estimates were little changed from the main analysis

Table 1
Subject and hospital admission characteristics, by study site.

	Albany		Buffalo		Bronx		Queens		Rochester		Manhattan	
	N	%	N	%	N	%	N	%	N	%	N	%
DISEASE												
Total cardiovascular disease	81,250	100.0	153,643	100.0	486,251	100.0	523,389	100.0	109,452	100.0	568,933	100.0
Cardiac arrhythmia	15,446	19.0	27,022	17.6	75,122	15.5	93,772	17.9	19,339	17.7	90,641	15.9
Cerebrovascular disease	15,369	18.9	28,368	18.5	66,115	13.6	72,460	13.8	18,999	17.4	76,493	13.4
Ischemic stroke	8934	11.0	17,411	11.3	42,334	8.7	45,864	8.8	11,712	10.7	47,332	8.3
Chronic rheumatic heart disease	424	0.5	486	0.3	2158	0.4	3255	0.6	395	0.4	2630	0.5
Congestive heart failure	19,465	24.0	36,389	23.7	120,197	24.7	114,274	21.8	29,580	27.0	128,317	22.6
Hypertension	5514	6.8	7681	5.0	60,215	12.4	43,682	8.4	5742	5.3	54,847	9.6
Ischemic heart disease	21,519	26.5	46,989	30.6	148,325	30.5	182,831	34.9	30,165	27.6	201,364	35.4
Myocardial infarction	11,319	13.9	23,873	15.5	51,971	10.7	60,850	11.6	14,872	13.6	65,607	11.5
Pulmonary embolus	3513	4.3	6708	4.4	14,119	2.9	13,115	2.5	5232	4.8	14,641	2.6
GENDER												
Male	41,296	50.8	77,280	50.3	246,373	50.7	285,279	54.5	58,180	53.2	296,266	52.1
AGE												
Years	70.4 (15.2)		70.7 (14.9)		67.2 (15.3)		69.0 (14.9)		70.3 (15.2)		68.5 (14.9)	
18–39	2381	2.9	4286	2.8	20,026	4.1	15,753	3.0	3409	3.1	18,510	3.3
40–49	5504	6.8	9819	6.4	43,868	9.0	39,160	7.5	7511	6.9	43,902	7.7
50–59	12,140	14.9	21,942	14.3	86,176	17.7	85,724	16.4	16,068	14.7	94,038	16.5
60–69	16,122	19.8	29,595	19.3	110,501	22.7	113,980	21.8	21,628	19.8	127,097	22.3
70–79	18,140	22.3	36,695	23.9	110,330	22.7	121,395	23.2	24,837	22.7	135,315	23.8
>=80	26,963	33.2	51,306	33.4	115,350	23.7	147,377	28.2	35,999	32.9	150,071	26.4
RACE												
White	63,847	78.6	123,296	80.3	155,938	32.1	269,990	51.6	84,544	77.2	262,751	46.2
Black or African American	7976	9.8	23,486	15.3	145,658	30.0	120,274	23.0	18,271	16.7	169,772	29.8
Native American or Alaskan Native	73	0.1	241	0.2	2210	0.5	3810	0.7	114	0.1	3394	0.6
Asian	213	0.3	387	0.3	6809	1.4	30,773	5.9	564	0.5	18,707	3.3
Native Hawaiian or Other Pacific Islander	58	0.1	10	0.0	154	0.0	272	0.1	19	0.0	133	0.0
ETHNICITY												
Hispanic	4231	5.2	1963	1.3	98,388	20.2	37,740	7.2	2902	2.7	49,442	8.7
Non-Hispanic	77,019	94.8	151,680	98.7	387,863	79.8	485,649	92.8	106,550	97.3	519,491	91.3
YEAR OF ADMISSION												
2005	8103	10.0	14,647	9.5	45,018	9.3	50,779	9.7	10,079	9.2	54,268	9.5
2006	8096	10.0	14,184	9.2	45,305	9.3	49,901	9.5	9963	9.1	55,008	9.7
2007	7635	9.4	13,734	8.9	44,121	9.1	46,812	8.9	9794	9.0	52,936	9.3
2008	7195	8.9	13,932	9.1	43,546	9.0	46,676	8.9	9666	8.8	52,612	9.3
2009	6757	8.3	13,175	8.6	42,976	8.8	46,459	8.9	10,031	9.2	52,012	9.1
2010	6479	8.0	12,848	8.4	41,155	8.5	45,450	8.7	9277	8.5	49,691	8.7
2011	6354	7.8	12,204	7.9	39,142	8.1	42,578	8.1	8995	8.2	45,784	8.1
2012	6030	7.4	11,823	7.7	38,731	8.0	40,116	7.7	8511	7.8	44,519	7.8
2013	5789	7.1	11,747	7.7	36,960	7.6	39,244	7.5	8210	7.5	41,968	7.4
2014	5987	7.4	11,683	7.6	35,186	7.2	37,501	7.2	7790	7.1	39,583	7.0
2015	6050	7.5	11,491	7.5	35,985	7.4	38,052	7.3	8194	7.5	40,086	7.1
2016	6775	8.3	12,175	7.9	38,126	7.8	39,821	7.6	8942	8.2	40,466	7.1
SEASON OF ADMISSION												
Fall	20,006	24.6	37,585	24.5	120,313	24.7	128,594	24.6	26,809	24.5	140,374	24.7
Spring	20,823	25.6	39,846	25.9	127,056	26.1	136,715	26.1	28,535	26.1	149,337	26.3
Summer	19,897	24.5	37,469	24.4	118,692	24.4	128,072	24.5	26,568	24.3	137,991	24.3
Winter	20,524	25.3	38,743	25.2	120,190	24.7	130,008	24.8	27,540	25.2	141,231	24.8
Length of hospital stay (Days)												
Mean (Standard Deviation)	5.6 (9.0)		5.5 (8.1)		5.7 (9.2)		5.6 (8.8)		5.1 (9.0)		5.6 (8.9)	

Table 2
Mean annual incidence rate (admissions/1000 persons per year) of total cardiovascular disease and cardiovascular disease subgroup hospital admissions, by period.

Outcome	Periods	Overall	Albany	Bronx	Buffalo	Manhattan	Queens	Rochester
Total Cardiovascular Disease	Before	15.56	14.53	15.36	15.92	15.30	16.63	13.67
	During	13.61	11.60	13.60	14.33	13.26	14.46	12.47
	After	11.82	11.21	11.97	13.38	10.90	12.55	11.31
Cardiac Arrhythmia	Before	2.36	2.68	2.14	2.38	2.15	2.74	2.43
	During	2.42	2.30	2.24	2.77	2.25	2.76	2.34
	After	1.92	2.03	1.81	2.28	1.74	2.15	1.72
Cerebrovascular Disease	Before	1.96	2.55	1.82	2.54	1.76	2.04	2.02
	During	1.88	2.12	1.78	2.54	1.69	1.90	2.10
	After	2.16	2.46	2.02	3.08	1.93	2.19	2.43
Ischemic stroke	Before	1.19	1.51	1.14	1.47	1.07	1.23	1.15
	During	1.17	1.19	1.16	1.55	1.04	1.20	1.28
	After	1.39	1.49	1.28	2.00	1.22	1.44	1.62
Chronic Rheumatic Heart Disease	Before	0.07	0.07	0.06	0.03	0.06	0.09	0.06
	During	0.06	0.06	0.05	0.04	0.06	0.09	0.04
	After	0.08	0.07	0.08	0.06	0.08	0.09	0.03
Congestive Heart Failure	Before	3.67	3.52	4.00	4.03	3.48	3.61	3.26
	During	3.14	2.80	3.32	3.38	2.94	3.14	3.37
	After	2.77	2.60	2.85	2.93	2.53	2.79	3.48
Hypertension	Before	1.24	0.77	1.58	0.69	1.30	1.25	0.56
	During	1.23	0.80	1.70	0.67	1.24	1.15	0.66
	After	1.33	0.95	1.76	0.87	1.29	1.28	0.74
Ischemic Heart Disease	Before	5.92	4.48	5.44	5.73	6.27	6.58	4.83
	During	4.46	2.99	4.12	4.28	4.74	5.06	3.32
	After	3.11	2.52	2.99	3.43	2.95	3.63	2.29
Myocardial Infarction	Before	1.85	1.90	1.67	2.51	1.76	1.88	1.97
	During	1.59	1.63	1.45	2.18	1.49	1.66	1.63
	After	1.46	1.65	1.26	2.13	1.34	1.56	1.55
Pulmonary Embolus	Before	0.34	0.48	0.32	0.52	0.29	0.32	0.50
	During	0.41	0.53	0.40	0.64	0.34	0.36	0.63
	After	0.46	0.57	0.46	0.73	0.39	0.41	0.62

Table 3
Distribution of $PM_{2.5}$ concentrations ($\mu g/m^3$) for case periods and control periods, by study site and time period.

Site	Time Period	CASE PERIODS							CONTROL PERIODS						
		Min.	5th %tile	25th %tile	50th %tile	75th %tile	95th %tile	Max.	Min.	5th %tile	25th %tile	50th %tile	75th %tile	95th %tile	Max.
Albany	Overall	−2.0	1.7	4.4	7.2	11.4	21.1	50.6	−2.0	1.7	4.4	7.2	11.3	21.1	50.6
	Before	−1.1	2.1	5.4	8.6	14.6	26.3	50.6	−1.1	2.1	5.5	8.7	14.5	26.2	50.6
	During	−1.6	1.7	4.3	7.0	10.9	19.5	35.6	−1.6	1.7	4.3	7.0	10.8	19.2	35.6
	After	−2.0	1.5	3.8	6.2	9.6	15.8	45.4	−2.0	1.5	3.8	6.2	9.5	15.8	45.4
Bronx	Overall	−3.3	2.6	5.7	8.8	13.8	24.9	58.2	−3.3	2.6	5.7	8.8	13.8	24.7	58.2
	Before	−0.2	3.8	7.0	10.9	17.2	29.7	58.2	−0.2	3.8	7.0	10.8	17.2	29.6	58.2
	During	−3.3	2.2	5.6	8.8	13.4	23.2	53.4	−3.3	2.2	5.6	8.8	13.3	23.1	53.4
	After	−0.1	2.7	4.8	7.0	10.3	16.8	30.2	−0.1	2.7	4.8	7.0	10.2	16.6	30.2
Buffalo	Overall	−2.7	2.5	5.6	8.5	12.6	22.8	56.1	−2.7	2.5	5.6	8.5	12.6	22.9	56.1
	Before	−0.8	3.2	6.8	10.1	14.9	29.3	56.1	−0.8	3.3	6.8	10.1	14.9	29.4	56.1
	During	−2.7	2.6	5.5	8.4	12.2	21.6	44.0	−2.7	2.6	5.5	8.4	12.2	21.4	44.0
	After	−2.0	1.9	4.5	7.3	10.7	16.4	29.1	−2.0	1.9	4.5	7.3	10.6	16.4	29.1
Manhattan	Overall	−2.0	4.1	7.2	10.5	15.3	25.7	59.6	−2.0	4.0	7.2	10.5	15.3	25.4	59.6
	Before	0.6	4.8	8.2	12.1	18.0	31.4	59.6	0.6	4.8	8.1	12.0	17.9	30.9	59.6
	During	−2.0	4.1	7.2	10.5	15.2	24.8	46.2	−2.0	4.1	7.2	10.5	15.2	24.8	46.2
	After	0.6	3.4	6.0	9.0	12.2	18.4	33.5	0.6	3.4	6.0	9.0	12.1	18.2	33.5
Queens	Overall	0.0	3.2	5.2	8.0	12.5	22.4	51.9	0.0	3.2	5.2	7.9	12.5	22.2	51.9
	Before	1.6	3.5	6.0	9.8	15.9	28.4	51.9	1.6	3.5	6.0	9.6	15.8	27.8	51.9
	During	0.6	3.3	5.2	7.9	12.1	21.1	39.0	0.6	3.3	5.2	7.9	12.0	21.0	39.0
	After	0.0	2.7	4.4	6.5	9.5	15.9	29.0	0.0	2.7	4.4	6.5	9.5	15.7	29.0
Rochester	Overall	−2.6	1.9	4.5	7.2	11.0	21.0	49.8	−2.6	1.9	4.5	7.1	10.9	20.9	49.8
	Before	−2.1	2.3	5.4	8.7	13.8	27.3	49.8	−2.1	2.3	5.3	8.5	13.6	27.6	49.8
	During	−1.4	1.9	4.3	6.9	10.5	19.8	34.1	−1.4	1.9	4.3	6.9	10.6	19.6	34.1
	After	−2.6	1.7	4.0	6.4	9.3	15.5	23.9	−2.6	1.7	4.0	6.2	9.3	15.4	23.9

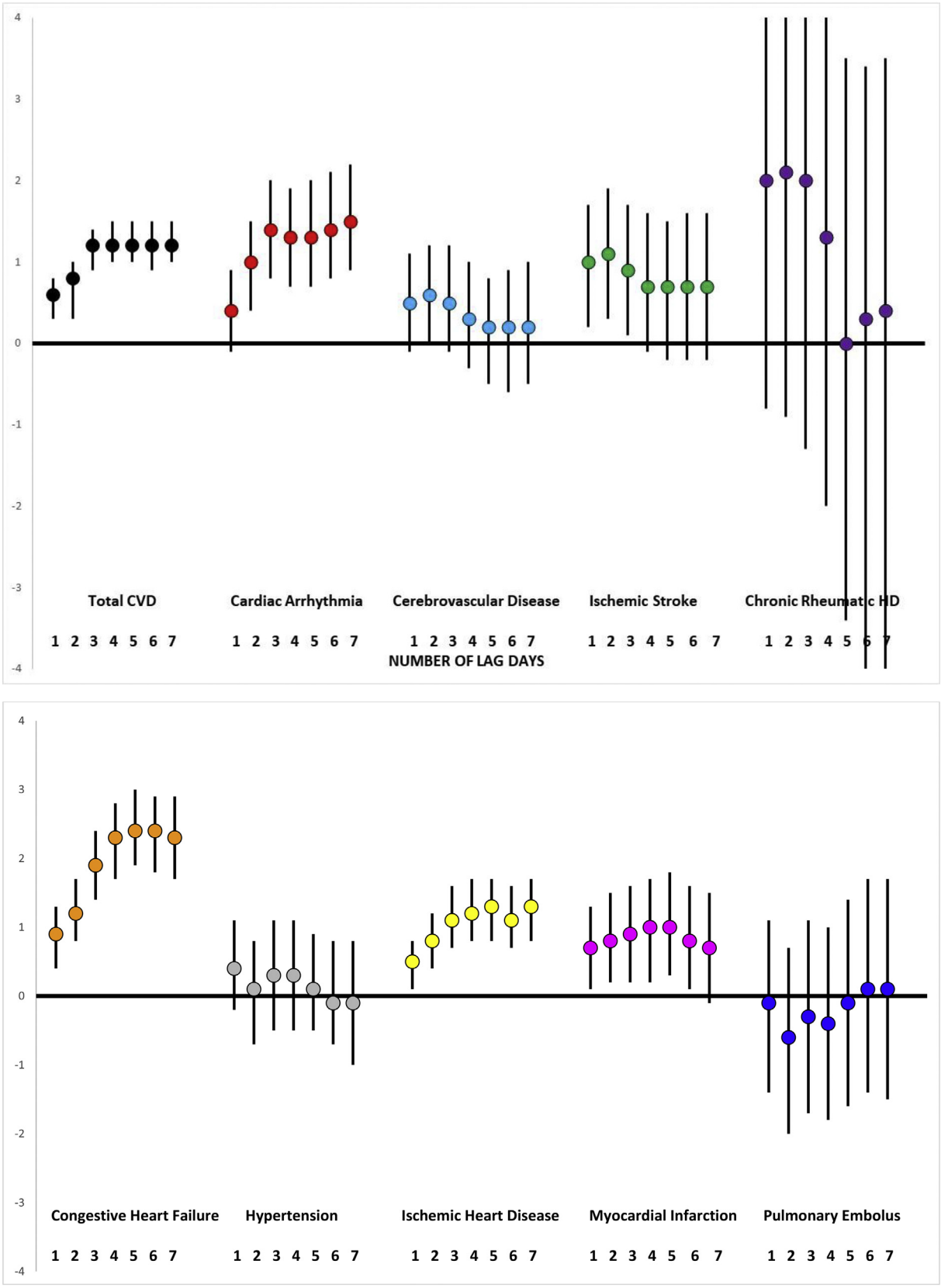

Fig. 1. Excess rate (and 95% confidence intervals) of cardiovascular disease hospital admissions associated with each interquartile range increase in $PM_{2.5}$ concentration, by lag day(s): a) Total cardiovascular disease (CVD), cardiac arrhythmia, cerebrovascular disease, ischemic stroke, chronic rheumatic heart disease (HD); b) congestive heart failure, hypertension, ischemic heart disease, myocardial infarction, pulmonary embolus.

Table 4
Excess rate (%) of total and cause specific cardiovascular hospital admissions associated with each interquartile range (IQR) increase in $PM_{2.5}$ concentration, by lag time and outcome.

Outcome	Lag days	IQR[a] (μg/m^3)	N Cases	Excess Rate %[b] (95% CI)	p-value
Total Cardiovascular Disease	0	6.9	1,878,501	0.6 (0.3, 0.8)	<0.001
	0–1	6.3	1,893,582	0.8 (0.6, 1.0)	<0.001
	0–2	6.1	1,908,881	1.2 (0.9, 1.4)	<0.001
	0–3	5.6	1,912,293	1.2 (1.0, 1.5)	<0.001
	0–4	5.3	1,914,744	1.2 (1.0, 1.5)	<0.001
	0–5	5.1	1,916,601	1.2 (0.9, 1.5)	<0.001
	0–6	4.9	1,917,823	1.2 (1.0, 1.5)	<0.001
Cardiac Arrhythmia	0	6.8	313,134	0.4 (−0.1, 0.9)	0.135
	0–1	6.2	315,863	1.0 (0.4, 1.5)	<0.001
	0–2	6.0	318,671	1.4 (0.8, 2.0)	<0.001
	0–3	5.6	319,328	1.3 (0.7, 1.9)	<0.001
	0–4	5.3	319,758	1.3 (0.7, 2.0)	<0.001
	0–5	5.1	320,132	1.4 (0.8, 2.1)	<0.001
	0–6	4.9	320,365	1.5 (0.9, 2.2)	<0.001
Cerebrovascular	0	6.8	271,115	0.5 (−0.1, 1.1)	0.074
	0–1	6.2	273,418	0.6 (−0.0, 1.2)	0.070
	0–2	6.0	275,758	0.5 (−0.1, 1.2)	0.114
	0–3	5.6	276,257	0.3 (−0.3, 1.0)	0.343
	0–4	5.3	276,615	0.2 (−0.5, 0.8)	0.668
	0–5	5.1	276,882	0.2 (−0.6, 0.9)	0.670
	0–6	4.9	277,057	0.2 (−0.5, 1.0)	0.562
Ischemic stroke	0	6.8	169,345	1.0 (0.2, 1.7)	0.009
	0–1	6.2	170,814	1.1 (0.3, 1.9)	0.005
	0–2	5.9	172,273	0.9 (0.1, 1.7)	0.033
	0–3	5.5	172,592	0.7 (−0.1, 1.6)	0.083
	0–4	5.2	172,832	0.7 (−0.2, 1.5)	0.142
	0–5	5.0	173,010	0.7 (−0.2, 1.6)	0.145
	0–6	4.8	173,112	0.7 (−0.2, 1.6)	0.148
Chronic Rheumatic Heart Disease	0	6.2	9121	2.0 (−0.8, 4.8)	0.170
	0–1	5.8	9194	2.1 (−0.9, 5.3)	0.169
	0–2	5.6	9276	2.0 (−1.3, 5.4)	0.247
	0–3	5.1	9298	1.3 (−2.0, 4.7)	0.449
	0–4	4.9	9313	−0.0 (−3.4, 3.5)	0.997
	0–5	4.8	9320	−0.3 (−4.0, 3.4)	0.861
	0–6	4.6	9327	−0.4 (−4.2, 3.5)	0.829
Congestive Heart Failure	0	6.9	438,038	0.9 (0.4, 1.3)	<0.001
	0–1	6.3	441,534	1.2 (0.8, 1.7)	<0.001
	0–2	6.0	445,099	1.9 (1.4, 2.4)	<0.001
	0–3	5.6	445,859	2.3 (1.7, 2.8)	<0.001
	0–4	5.3	446,438	2.4 (1.9, 3.0)	<0.001
	0–5	5.1	446,852	2.4 (1.8, 2.9)	<0.001
	0–6	4.9	447,123	2.3 (1.7, 2.9)	<0.001
Hypertension	0	6.6	173,743	0.4 (−0.2, 1.1)	0.208
	0–1	6.1	175,065	0.1 (−0.7, 0.8)	0.851
	0–2	5.7	176,408	0.3 (−0.5, 1.1)	0.484
	0–3	5.3	176,744	0.3 (−0.5, 1.1)	0.525
	0–4	5.0	176,986	0.1 (−0.7, 0.9)	0.831
	0–5	4.8	177,175	−0.1 (−0.9, 0.8)	0.857
	0–6	4.6	177,272	−0.1 (−1.0, 0.8)	0.849
Ischemic Heart Disease	0	7.2	617,395	0.5 (0.1, 0.8)	0.018
	0–1	6.5	622,058	0.8 (0.4, 1.2)	<0.001
	0–2	6.2	626,731	1.1 (0.7, 1.6)	<0.001
	0–3	5.8	627,772	1.2 (0.8, 1.7)	<0.001
	0–4	5.5	628,522	1.3 (0.8, 1.7)	<0.001
	0–5	5.3	629,090	1.1 (0.7, 1.6)	<0.001
	0–6	5.1	629,497	1.3 (0.8, 1.7)	<0.001
Myocardial Infarction	0	6.9	223,090	0.7 (0.1, 1.3)	0.029
	0–1	6.3	225,010	0.8 (0.2, 1.5)	0.016
	0–2	6.1	226,876	0.9 (0.2, 1.6)	0.015
	0–3	5.6	227,258	1.0 (0.2, 1.7)	0.010
	0–4	5.3	227,550	1.0 (0.3, 1.8)	0.008
	0–5	5.1	227,785	0.8 (0.1, 1.6)	0.035
	0–6	4.9	227,933	0.7 (−0.1, 1.5)	0.089
Pulmonary Embolus	0	6.6	55,955	−0.1 (−1.4, 1.1)	0.829
	0–1	6.2	56,450	−0.6 (−2.0, 0.7)	0.352
	0–2	5.8	56,938	−0.3 (−1.7, 1.1)	0.685
	0–3	5.3	57,035	−0.4 (−1.8, 1.0)	0.563
	0–4	5.0	57,112	−0.1 (−1.6, 1.4)	0.889

Table 4 (*continued*)

Outcome	Lag days	IQR[a] (μg/m^3)	N Cases	Excess Rate %[b] (95% CI)	p-value
	0–5	4.9	57,150	0.1 (−1.4, 1.7)	0.849
	0–6	4.7	57,182	0.1 (−1.5, 1.7)	0.890

[a] Albany IQR's used to scale all OR's and 95% confidence intervals.
[b] Models adjusted for temperature (4df) and relative humidity using natural splines (3df).

(Supplementary Table S3). Thus, the sensitivity analysis suggests that our estimates from these models are robust.

Next, we evaluated whether the excess rate of total cardiovascular and cause-specific hospital admission associated with each interquartile range increase in $PM_{2.5}$ concentration in the previous 0–6 days differed by period (i.e. before: 2005–2007; during: 2008–2013; after: 2014–2016). For cardiac arrhythmia, cerebrovascular, ischemic stroke, chronic rheumatic heart disease, congestive heart failure, hypertension, and pulmonary embolus admissions, there was no difference between period specific excess rate estimates (Table 5, with common interquartile ranges of $PM_{2.5}$ for controls in all sites used). However, there was a significant difference in the excess rate of ischemic heart disease admissions associated with each interquartile range increase in $PM_{2.5}$ concentration in previous 2 days (before: 0.8%; during: 0.6%; after: 2.8%; $p = 0.004$), with similar patterns in the previous 1, 3, 4, and 5 days. Although not statistically significant, both total cardiovascular disease and myocardial infarction admissions followed similar patterns on the same lag days (Table 5). These patterns in total cardiovascular disease, ischemic heart disease, and myocardial infarction were similar when scaling period-specific excess rate estimates to the interquartile ranges from each period, but differences between periods were somewhat smaller (Supplementary Table S4).

Next, we examined whether there were similar changes in the relative rates of admissions for these three disease subgroups (total cardiovascular disease, ischemic heart disease, and myocardial infarction) associated with interquartile range increases in $PM_{2.5}$ concentrations across periods in the Upstate sites alone (Buffalo, Rochester, and Albany), and in the New York City sites alone (Manhattan, Queens, Bronx) (Table 6). In the New York City sites, the excess rates of ischemic heart disease admissions associated with each interquartile range increase in $PM_{2.5}$ concentrations in the previous 2 days, within each period, were significantly different, and largest in the after period (before: 1.1%, during: 0.6%, after: 3.1%; $P = 0.012$). In the Upstate sites, the pattern was similar, but period specific excess rates were not significantly different. For myocardial infarction, the excess rates associated with each interquartile range increase in $PM_{2.5}$ concentrations in the previous 2 days, within each period, were also significantly different and also largest in the after period (before: 0.9%, during: 0.8%; after: 2.1%). Again, although not significantly different, there was a similar myocardial infarction admission pattern across periods in the Upstate sites (before: −0.9%, during: 1.9%; after: 3.9%, Table 6). Mean $PM_{2.5}$ concentrations across different lag day periods (0, 0–1, 0–2, 0–3, 0–4, 0–5, and 0–6) were well correlated ($r = 0.56$–0.97) (Supplementary Table S5), as were concentrations across individual lag days (0, 1, 2, 3, 4, 5, and 6) (Supplementary Table S6). The pattern of relative rates of admissions associated with increased $PM_{2.5}$ concentrations on individual lag days (Supplementary Table S7) was similar to the pattern observed in our main analysis across different lag periods (Table 4).

4. Discussion

From 2005 to 2016 in NYS, short-term increases in ambient $PM_{2.5}$ concentrations were associated with small but significant increases in the rate of hospital admissions for total cardiovascular disease, cardiac arrhythmia, ischemic stroke, congestive heart failure, ischemic heart disease, and myocardial infarction. These associations were independent of acute changes in temperature and relative humidity, season, weekday, and long-term time trends. Although the incidence rates of all disease categories decreased across the study period, there was no difference in the relative rate of most cardiovascular disease subgroups associated with each interquartile range increase in $PM_{2.5}$ concentration after the recession and implementation of environmental policies (2014–2016), compared to before (2005–2007) and during implementation/recession (2008–2013). However, the rate of hospital admissions for ischemic heart disease associated with each interquartile range increase in $PM_{2.5}$ concentration was higher after the recession and implementation of environmental policies and actions. This pattern was inconsistent with our a priori hypothesis, but was observed in both Upstate sites (Buffalo, Rochester, and Albany) and New York City sites (Manhattan, Queens, and the Bronx). Further, the same conclusion was made when using period specific interquartile ranges to scale effect estimates. This suggests that changes in the particle composition resulting from these policies and the co-occurring recession may differentially trigger these acute cardiovascular outcomes. However, as discussed below, we did not have a "control" community that was similar in other ways to the study area, but was not impacted by these policies and changes. Thus, this finding needs to be confirmed in another population.

Our findings of acute associations between hospital admissions for acute cardiovascular events and short term increases in ambient $PM_{2.5}$ concentrations (Excess Rate = 2.19%; 95% CI = 1.74%, 2.65% per 10 μg/m^3 increase in $PM_{2.5}$ in lag days 0–3) are consistent with Dominici et al., 2006, who reported a 1.28% (0.78%–1.78%) increase in the risk of heart failure hospitalization per 10-μg/m^3 increase in same-day $PM_{2.5}$ concentration in 204 U.S. urban counties. They are also consistent with prior studies' findings of acute associations between short term increases in ambient $PM_{2.5}$ concentrations and myocardial infarction (Evans et al., 2017; Gardner et al., 2014; Mustafić et al., 2012; Pope et al., 2015), ischemic stroke (Shah et al., 2015), heart failure (Shah et al., 2018), and cardiac arrhythmia (Link et al., 2013; Peters et al., 2000). We also reported a 23.6% (1.3%–49.7%) increase in the risk of ventricular arrhythmia associated with each 10 μg/m^3 increase in 24-h moving average $PM_{2.5}$ concentration in a study based in Boston (Rich et al., 2005). Multiple interactive pathophysiologic mechanisms such as direct translocation of $PM_{2.5}$ into blood and then to remote target organs, as well as vascular, inflammatory, thrombotic, oxidative stress, and other mechanisms described in depth previously (Brook et al., 2010; Pope et al., 2004), may explain these associations between $PM_{2.5}$ concentrations and adverse cardiovascular outcomes (Du et al., 2016).

Previous studies have reported beneficial population health effects of reductions in PM concentration, such as increases in life expectancy and reduced population morbidity across the United State (Dominici et al., 2015; Laden et al., 2006; Pope et al., 2009). We observed reductions in rates of cardiovascular disease hospital

Table 5
Excess rate (%) of total and cause-specific cardiovascular hospital admissions associated with each interquartile range (IQR) increase in $PM_{2.5}$ concentration, by lag time, outcome, and period (using common IQRs across the whole period).

Outcome	Lag days	IQR ($\mu g/m^3$)	BEFORE		DURING		AFTER		Interaction P value
			N cases	Excess Rate (%)	N cases	Excess Rate (%)	N cases	Excess Rate (%)	
Total Cardiovascular Disease	0	6.9	535,966	0.6 (0.3, 0.9)	923,237	0.4 (0.1, 0.7)	419,298	1.1 (0.5, 1.7)	0.063
	0–1	6.3	537,382	0.8 (0.5, 1.1)	935,035	0.7 (0.4, 1.0)	421,165	1.5 (0.9, 2.1)	0.052
	0–2	6.1	538,680	1.0 (0.7, 1.3)	947,228	1.2 (0.9, 1.6)	422,973	1.8 (1.1, 2.5)	0.099
	0–3	5.6	538,869	1.1 (0.8, 1.5)	950,175	1.3 (0.9, 1.6)	423,249	1.6 (0.9, 2.2)	0.450
	0–4	5.3	539,074	1.2 (0.8, 1.6)	952,097	1.3 (0.9, 1.7)	423,573	1.1 (0.5, 1.8)	0.878
	0–5	5.1	539,074	1.2 (0.9, 1.6)	953,763	1.2 (0.8, 1.6)	423,764	1.0 (0.3, 1.7)	0.855
	0–6	4.9	539,074	1.4 (1.0, 1.8)	954,856	1.1 (0.7, 1.5)	423,893	1.0 (0.3, 1.7)	0.370
Cardiac Arrhythmia	0	6.8	81,121	0.2 (−0.5, 0.9)	163,968	0.5 (−0.3, 1.2)	68,045	1.1 (−0.3, 2.5)	0.511
	0–1	6.2	81,371	0.4 (−0.3, 1.2)	166,142	1.3 (0.6, 2.1)	68,350	1.6 (0.1, 3.1)	0.139
	0–2	6.0	81,566	0.7 (−0.1, 1.6)	168,472	2.0 (1.2, 2.9)	68,633	1.5 (−0.1, 3.1)	0.052
	0–3	5.6	81,587	0.6 (−0.2, 1.5)	169,062	1.9 (1.1, 2.8)	68,679	1.4 (−0.2, 3.1)	0.061
	0–4	5.3	81,621	0.8 (−0.1, 1.7)	169,404	1.8 (1.0, 2.7)	68,733	1.1 (−0.6, 2.8)	0.174
	0–5	5.1	81,621	0.9 (0.0, 1.9)	169,737	1.9 (1.0, 2.8)	68,774	1.2 (−0.5, 3.0)	0.270
	0–6	4.9	81,621	1.0 (0.1, 2.0)	169,946	2.0 (1.1, 2.9)	68,798	1.4 (−0.3, 3.3)	0.300
Cerebrovascular Disease	0	6.8	67,382	0.1 (−0.7, 0.9)	127,144	0.8 (0.0, 1.6)	76,589	0.9 (−0.4, 2.3)	0.334
	0–1	6.2	67,568	0.4 (−0.4, 1.3)	128,908	0.7 (−0.2, 1.5)	76,942	0.7 (−0.8, 2.1)	0.923
	0–2	6.0	67,774	0.5 (−0.4, 1.4)	130,689	0.7 (−0.2, 1.6)	77,295	0.1 (−1.4, 1.6)	0.793
	0–3	5.6	67,798	0.3 (−0.7, 1.2)	131,123	0.6 (−0.4, 1.5)	77,336	−0.3 (−1.8, 1.3)	0.630
	0–4	5.3	67,818	0.2 (−0.8, 1.1)	131,405	0.4 (−0.5, 1.4)	77,392	−0.7 (−2.3, 0.9)	0.478
	0–5	5.1	67,818	0.1 (−0.9, 1.2)	131,638	0.3 (−0.6, 1.3)	77,426	−0.4 (−2.0, 1.2)	0.710
	0–6	4.9	67,818	0.4 (−0.6, 1.5)	131,797	0.3 (−0.8, 1.3)	77,442	−0.5 (−2.1, 1.2)	0.668
Ischemic Stroke	0	6.8	40,738	0.4 (−0.6, 1.4)	79,415	1.5 (0.5, 2.6)	49,192	1.2 (−0.5, 2.9)	0.214
	0–1	6.2	40,846	0.7 (−0.3, 1.8)	80,545	1.5 (0.5, 2.6)	49,423	0.9 (−0.9, 2.7)	0.500
	0–2	5.9	40,978	0.8 (−0.3, 2.0)	81,656	1.2 (0.1, 2.4)	49,639	0.1 (−1.8, 2.0)	0.576
	0–3	5.5	40,996	0.7 (−0.5, 1.9)	81,931	1.0 (−0.1, 2.2)	49,665	−0.1 (−2.0, 1.8)	0.573
	0–4	5.2	41,007	0.8 (−0.5, 2.0)	82,118	0.9 (−0.3, 2.1)	49,707	−0.5 (−2.5, 1.5)	0.428
	0–5	5.0	41,007	0.8 (−0.5, 2.1)	82,275	0.9 (−0.4, 2.1)	49,728	−0.2 (−2.2, 1.8)	0.623
	0–6	4.8	41,007	1.1 (−0.3, 2.4)	82,367	0.7 (−0.6, 2.0)	49,738	−0.3 (−2.4, 1.8)	0.537
Chronic Rheumatic Heart Disease	0	6.2	2263	1.8 (−2.0, 5.7)	4158	0.9 (−3.1, 5.0)	2700	5.8 (−0.7, 12.7)	0.421
	0–1	5.8	2268	2.0 (−2.1, 6.3)	4212	0.6 (−3.7, 5.0)	2714	7.4 (0.3, 15.0)	0.243
	0–2	5.6	2272	1.2 (−3.3, 6.0)	4276	0.8 (−3.7, 5.6)	2728	7.8 (0.0, 16.2)	0.266
	0–3	5.1	2273	0.6 (−3.9, 5.4)	4293	0.3 (−4.2, 5.1)	2732	6.3 (−1.4, 14.6)	0.370
	0–4	4.9	2273	0.2 (−4.6, 5.3)	4307	−1.1 (−5.8, 3.9)	2733	2.7 (−5.1, 11.1)	0.710
	0–5	4.8	2273	0.3 (−4.8, 5.8)	4314	−1.7 (−6.7, 3.5)	2733	1.8 (−6.3, 10.6)	0.721
	0–6	4.6	2273	1.5 (−4.0, 7.2)	4321	−2.6 (−7.7, 2.8)	2733	0.8 (−7.6, 9.8)	0.524
Congestive Heart Failure	0	6.9	126,506	1.2 (0.6, 1.8)	213,338	0.5 (−0.1, 1.1)	98,194	1.2 (−0.0, 2.4)	0.216
	0–1	6.3	126,840	1.3 (0.6, 1.9)	216,041	1.1 (0.4, 1.8)	98,653	1.6 (0.3, 2.9)	0.750
	0–2	6.0	127,172	1.7 (1.0, 2.4)	218,847	2.1 (1.4, 2.8)	99,080	2.3 (1.0, 3.7)	0.518
	0–3	5.6	127,222	2.1 (1.3, 2.8)	219,492	2.4 (1.7, 3.2)	99,145	2.5 (1.1, 3.9)	0.694
	0–4	5.3	127,269	2.3 (1.6, 3.1)	219,948	2.5 (1.8, 3.3)	99,221	2.5 (1.1, 3.9)	0.898
	0–5	5.1	127,269	2.3 (1.5, 3.1)	220,323	2.4 (1.6, 3.1)	99,260	2.5 (1.0, 3.9)	0.982
	0–6	4.9	127,269	2.4 (1.6, 3.2)	220,564	2.1 (1.3, 3.0)	99,290	2.6 (1.1, 4.1)	0.806
Hypertension	0	6.6	42,849	1.1 (0.2, 2.1)	83,780	0.1 (−0.9, 1.0)	47,114	−0.6 (−2.3, 1.0)	0.102
	0–1	6.1	42,931	0.8 (−0.2, 1.9)	84,834	−0.6 (−1.6, 0.4)	47,300	−0.2 (−1.9, 1.6)	0.098
	0–2	5.7	43,001	0.9 (−0.2, 2.0)	85,928	−0.5 (−1.6, 0.5)	47,479	1.1 (−0.8, 3.0)	0.083
	0–3	5.3	43,015	0.9 (−0.2, 2.1)	86,220	−0.5 (−1.6, 0.6)	47,509	0.9 (−1.1, 2.9)	0.124
	0–4	5.0	43,031	0.8 (−0.4, 2.0)	86,418	−0.5 (−1.6, 0.6)	47,537	0.0 (−1.9, 2.0)	0.215
	0–5	4.8	43,031	0.7 (−0.5, 2.0)	86,588	−0.7 (−1.9, 0.4)	47,556	−0.3 (−2.3, 1.8)	0.180
	0–6	4.6	43,031	1.0 (−0.3, 2.3)	86,676	−1.0 (−2.2, 0.2)	47,565	−0.1 (−2.2, 2.1)	0.047
Ischemic Heart Disease	0	7.2	204,048	0.5 (0.0, 1.0)	303,085	0.2 (−0.4, 0.7)	110,262	1.8 (0.6, 3.0)	0.032
	0–1	6.5	204,563	0.8 (0.2, 1.3)	306,748	0.6 (0.0, 1.2)	110,747	2.8 (1.5, 4.0)	0.004
	0–2	6.2	205,019	0.9 (0.4, 1.5)	310,487	1.0 (0.4, 1.6)	111,225	3.0 (1.7, 4.4)	0.011
	0–3	5.8	205,097	1.1 (0.5, 1.7)	311,376	1.1 (0.5, 1.7)	111,299	2.6 (1.3, 4.0)	0.096
	0–4	5.5	205,182	1.2 (0.6, 1.9)	311,947	1.2 (0.5, 1.8)	111,393	2.0 (0.6, 3.4)	0.594
	0–5	5.3	205,182	1.3 (0.6, 1.9)	312,458	1.0 (0.3, 1.7)	111,450	1.3 (−0.2, 2.7)	0.834
	0–6	5.1	205,182	1.5 (0.9, 2.2)	312,821	1.0 (0.3, 1.7)	111,494	1.0 (−0.5, 2.4)	0.466
Myocardial Infarction	0	6.9	63,483	0.4 (−0.4, 1.3)	107,764	0.9 (−0.0, 1.8)	51,843	1.2 (−0.4, 2.9)	0.598
	0–1	6.3	63,685	0.4 (−0.5, 1.3)	109,244	0.9 (−0.0, 1.9)	52,081	2.3 (0.5, 4.0)	0.155
	0–2	6.1	63,865	0.6 (−0.3, 1.6)	110,715	0.9 (−0.1, 1.9)	52,296	2.1 (0.3, 4.1)	0.346
	0–3	5.6	63,885	0.9 (−0.1, 1.9)	111,038	0.9 (−0.1, 1.9)	52,335	1.6 (−0.3, 3.5)	0.797
	0–4	5.3	63,905	1.0 (0.0, 2.1)	111,255	1.0 (−0.0, 2.1)	52,390	1.1 (−0.9, 3.0)	0.998
	0–5	5.1	63,905	0.9 (−0.2, 2.0)	111,464	0.9 (−0.2, 2.0)	52,416	0.4 (−1.6, 2.4)	0.889
	0–6	4.9	63,905	0.9 (−0.2, 2.0)	111,593	0.7 (−0.4, 1.8)	52,435	0.1 (−1.9, 2.2)	0.804
Pulmonary Embolus	0	6.6	11,797	−0.9 (−2.6, 1.0)	27,764	−0.1 (−1.8, 1.5)	16,394	1.9 (−0.9, 4.8)	0.245
	0–1	6.2	11,841	−1.1 (−3.1, 0.8)	28,150	−0.7 (−2.5, 1.1)	16,459	1.0 (−2.0, 4.1)	0.478
	0–2	5.8	11,876	−0.5 (−2.5, 1.6)	28,529	−0.5 (−2.4, 1.4)	16,533	1.0 (−2.2, 4.2)	0.689
	0–3	5.3	11,877	−0.2 (−2.3, 2.0)	28,609	−0.6 (−2.4, 1.3)	16,549	−0.6 (−3.7, 2.7)	0.958

Table 5 (*continued*)

Outcome	Lag days	IQR (μg/m^3)	BEFORE		DURING		AFTER		Interaction P value
			N cases	Excess Rate (%)	N cases	Excess Rate (%)	N cases	Excess Rate (%)	
	0–4	5.0	11,880	−0.0 (−2.2, 2.2)	28,668	−0.0 (−1.9, 2.0)	16,564	−0.7 (−3.9, 2.6)	0.933
	0–5	4.9	11,880	0.2 (−2.1, 2.5)	28,705	0.2 (−1.8, 2.3)	16,565	−0.1 (−3.5, 3.3)	0.983
	0–6	4.7	11,880	0.2 (−2.2, 2.7)	28,731	0.2 (−1.9, 2.4)	16,571	−0.5 (−3.9, 3.1)	0.928

admissions across this period at all six sites (independent of changes in $PM_{2.5}$ concentrations), and specifically large reductions in the rates of ischemic heart disease and myocardial infarction hospital admissions. These reductions were likely primarily driven by improvements in preventive care and treatment procedures (e.g. use of statins, improvement in emergency treatment procedures) (Benjamin et al., 2018; Fang et al., 2011; McGovern et al., 2001; Moran et al., 2014; Wijeysundera et al., 2010; Yeh et al., 2010), but may also be a result of reductions in air pollutants during this time (Squizzato et al., 2018). However, an investigation into whether any change in PM composition during the study period (here generally from 2007 to 2013) impacted the rate of acute cardiovascular events associated with increased ambient $PM_{2.5}$ concentrations, may provide clues as to the PM sources/mixtures that most strongly trigger acute cardiovascular events.

Changes in $PM_{2.5}$ mass composition might be an important reason behind our finding of a larger excess rate of ischemic heart disease hospital admissions per interquartile range increase in $PM_{2.5}$ concentration in the after period, compared to the before, and during periods. To explore this possibility, inter-period differences in the concentrations of the main particulate matter species (sulfate, nitrate, organic and elemental carbon) and derived species

Table 6
Excess rate of total cardiovascular disease, ischemic heart disease, and myocardial infarction hospital admissions associated with each interquartile range (IQR) increase in $PM_{2.5}$ concentration, by lag time, outcome, period and region.

Outcome	Region	Lag days	IQR	BEFORE		DURING		AFTER		Interaction p-value
				N cases	Excess Rate % (95% CI)	N cases	Excess Rate % (95% CI)	N cases	Excess Rate % (95% CI)	
Total Cardiovascular Disease	NYC	0	8.1	442,894	0.9 (0.6, 1.3)	759,635	0.5 (0.1, 0.9)	341,378	1.4 (0.6, 2.1)	0.066
		0–1	7.2	443,387	1.0 (0.6, 1.4)	768,903	0.9 (0.5, 1.3)	342,640	2.0 (1.2, 2.8)	0.033
		0–2	6.6	443,830	1.3 (0.9, 1.7)	778,551	1.5 (1.1, 1.9)	343,886	2.4 (1.6, 3.2)	0.036
		0–3	6.2	443,977	1.4 (1.0, 1.8)	781,319	1.5 (1.1, 1.9)	344,162	2.0 (1.2, 2.9)	0.380
		0–4	5.9	444,148	1.5 (1.1, 1.9)	783,158	1.5 (1.0, 1.9)	344,486	1.4 (0.5, 2.3)	0.975
		0–5	5.6	444,148	1.5 (1.1, 2.0)	784,776	1.3 (0.8, 1.7)	344,677	1.2 (0.3, 2.1)	0.631
		0–6	5.4	444,148	1.8 (1.3, 2.3)	785,833	1.1 (0.7, 1.6)	344,806	1.2 (0.3, 2.1)	0.081
	Upstate	0	6.9	93,072	−0.1 (−0.8, 0.6)	163,602	0.2 (−0.6, 1.0)	77,920	0.9 (−0.5, 2.3)	0.407
		0–1	6.3	93,995	0.3 (−0.5, 1.0)	166,132	0.2 (−0.6, 1.0)	78,525	0.3 (−1.1, 1.8)	0.985
		0–2	6.1	94,850	0.4 (−0.4, 1.1)	168,677	0.5 (−0.4, 1.4)	79,087	−0.0 (−1.6, 1.5)	0.810
		0–3	5.6	94,892	0.4 (−0.4, 1.2)	168,856	0.8 (−0.0, 1.7)	79,087	0.3 (−1.2, 1.9)	0.705
		0–4	5.3	94,926	0.6 (−0.3, 1.4)	168,939	1.2 (0.3, 2.1)	79,087	0.7 (−0.8, 2.3)	0.546
		0–5	5.1	94,926	0.6 (−0.3, 1.4)	168,987	1.3 (0.4, 2.3)	79,087	0.9 (−0.7, 2.5)	0.443
		0–6	4.9	94,926	0.5 (−0.3, 1.4)	169,023	1.5 (0.5, 2.5)	79,087	0.9 (−0.8, 2.5)	0.288
Ischemic Heart Disease	NYC	0	8.4	171,881	0.9 (0.3, 1.5)	257,382	0.1 (−0.5, 0.8)	92,225	2.0 (0.5, 3.5)	0.039
		0–1	7.5	172,082	1.1 (0.4, 1.7)	260,374	0.6 (−0.1, 1.3)	92,563	3.1 (1.5, 4.6)	0.012
		0–2	6.9	172,257	1.3 (0.6, 1.9)	263,429	1.1 (0.4, 1.8)	92,902	3.4 (1.8, 5.0)	0.025
		0–3	6.5	172,320	1.4 (0.7, 2.2)	264,267	1.2 (0.4, 1.9)	92,976	2.9 (1.2, 4.6)	0.160
		0–4	6.1	172,393	1.5 (0.8, 2.3)	264,811	1.2 (0.4, 1.9)	93,070	2.0 (0.3, 3.7)	0.585
		0–5	5.8	172,393	1.5 (0.8, 2.3)	265,309	0.9 (0.2, 1.7)	93,127	1.2 (−0.5, 2.9)	0.513
		0–6	5.5	172,393	1.9 (1.1, 2.7)	265,662	0.9 (0.1, 1.8)	93,171	0.9 (−0.9, 2.6)	0.151
	Upstate	0	7.2	32,167	−0.8 (−2.1, 0.4)	45,703	0.4 (−1.1, 1.9)	18,037	2.3 (−0.7, 5.4)	0.092
		0–1	6.5	32,481	−0.2 (−1.5, 1.1)	46,374	0.8 (−0.7, 2.4)	18,184	3.0 (−0.1, 6.2)	0.115
		0–2	6.2	32,762	−0.0 (−1.4, 1.3)	47,058	0.9 (−0.7, 2.6)	18,323	2.5 (−0.7, 5.8)	0.270
		0–3	5.8	32,777	0.2 (−1.3, 1.6)	47,109	1.3 (−0.3, 3.0)	18,323	2.8 (−0.5, 6.1)	0.234
		0–4	5.5	32,789	0.6 (−0.9, 2.0)	47,136	1.9 (0.2, 3.7)	18,323	2.7 (−0.6, 6.2)	0.298
		0–5	5.3	32,789	0.6 (−0.9, 2.1)	47,149	1.8 (−0.0, 3.6)	18,323	2.3 (−1.1, 5.8)	0.435
		0–6	5.1	32,789	0.5 (−1.0, 2.0)	47,159	1.8 (−0.1, 3.7)	18,323	2.1 (−1.4, 5.7)	0.438
Myocardial Infarction	NYC	0	8.0	49,804	1.2 (0.1, 2.3)	84,442	0.8 (−0.4, 1.9)	40,182	0.9 (−1.2, 3.1)	0.868
		0–1	7.2	49,866	0.9 (−0.2, 2.1)	85,554	0.8 (−0.4, 2.0)	40,327	2.1 (−0.2, 4.4)	0.576
		0–2	6.6	49,922	1.2 (0.0, 2.4)	86,656	0.8 (−0.4, 2.0)	40,463	2.0 (−0.4, 4.3)	0.638
		0–3	6.2	49,937	1.6 (0.3, 2.9)	86,949	0.8 (−0.5, 2.1)	40,502	1.1 (−1.3, 3.6)	0.623
		0–4	6.0	49,954	1.8 (0.4, 3.1)	87,154	0.8 (−0.5, 2.1)	40,557	0.4 (−2.1, 2.9)	0.461
		0–5	5.7	49,954	1.5 (0.1, 2.9)	87,359	0.7 (−0.6, 2.1)	40,583	−0.5 (−3.0, 2.1)	0.340
		0–6	5.4	49,954	1.5 (0.1, 2.9)	87,486	0.5 (−0.9, 1.9)	40,602	−0.8 (−3.3, 1.8)	0.251
	Upstate	0	6.9	13,679	−1.8 (−3.6, 0.0)	23,322	1.6 (−0.4, 3.7)	11,661	2.7 (−0.9, 6.4)	0.008
		0–1	6.3	13,819	−0.9 (−2.8, 1.0)	23,690	1.9 (−0.2, 4.0)	11,754	3.9 (0.2, 7.8)	0.018
		0–2	6.1	13,943	−1.1 (−3.1, 0.9)	24,059	1.5 (−0.7, 3.8)	11,833	3.3 (−0.7, 7.4)	0.051
		0–3	5.6	13,948	−1.0 (−3.0, 1.1)	24,089	1.6 (−0.7, 3.9)	11,833	3.7 (−0.4, 7.8)	0.051
		0–4	5.3	13,951	−0.6 (−2.7, 1.5)	24,101	2.1 (−0.2, 4.6)	11,833	3.7 (−0.4, 7.9)	0.063
		0–5	5.1	13,951	−0.5 (−2.6, 1.6)	24,105	2.0 (−0.4, 4.5)	11,833	3.3 (−0.8, 7.6)	0.116
		0–6	4.9	13,951	−0.6 (−2.8, 1.7)	24,107	1.7 (−0.9, 4.3)	11,833	3.1 (−1.2, 7.5)	0.189

(primary organic aerosol and secondary organic aerosol) were tested using the non-parametric Kruskal Wallis test and the post-hoc Dunn test. Results are reported in supplementary materials.

At each of these sites, measurements of $PM_{2.5}$ composition were made on filters that were collected every third or sixth day as part of the US EPA's Chemical Speciation Network (CSN) (Solomon et al., 2014). These data included elemental composition measured by X-ray fluorescence, cations and anions measured with ion chromatography, and organic and elemental carbon measured using thermal optical methods. As can be seen in Supplemental Figs. S1–S5, $PM_{2.5}$ and its major constituents, sulfate, nitrate, organic and elemental carbon, decreased across the three periods as demonstrated using a Kruskal-Wallis ANOVA on ranks. However, if the organic carbon (OC) is separated into primary and secondary OC as described in the supplementary material, then differences between primary OC and secondary OC can be observed (Figs. S7 and S8). Primary OC declined at all sites except Albany where it remained constant as shown by the ANOVA on ranks analysis. However, SOC showed statistically significant increases from the during period to the after period at the Albany, Bronx, Manhattan, and Rochester sites while remaining constant at Buffalo and Queens. The increased secondary OC concentrations likely result from increased oxidation of volatile organic compounds by oxidants like hydroxyl radicals that would have previously reacted with SO_2 and NO_2. The formation of secondary OC results in the formation of reactive oxygen species that allows the particles to deposit the oxidants into the respiratory tract (Hopke et al., 2015) and potentially contribute to increased oxidative stress.

Although each 7.1 $\mu g/m^3$ increase in $PM_{2.5}$ in the previous hour was associated with a significant 18% increase in the relative risk of a ST-elevation myocardial infarction in Rochester (OR = 1.18; 95% CI = 1.01, 1.38) (Gardner et al., 2014), later work found that this increased risk was observed only when the air mass in Rochester passed through the West-Southwest direction/sector any time in the previous 24 h (WSW: OR = 1.27; 95% CI = 1.08, 1.22; not WSW: OR = 0.99, 95% CI = 0.80, 1.22). This result suggested there were significant sources of gases from the WSW direction that are precursors to secondary aerosol formation (Hopke et al., 2015). Similarly, prior work by Rich et al. (2013) reported larger relative odds of myocardial infarction associated with increased $PM_{2.5}$ concentrations when the air pollution mixtures were higher in secondary PM species (sulfate, nitrate, and/or organics). Vedal et al. (2013) suggested cerebrovascular disease and stroke were associated with organic carbon, but not with elemental carbon. Thurston et al. (2016) suggested that each 0.26-$\mu g/m^3$ increase in elemental carbon might be associated with a 3.0% increase in the risk of ischemic heart disease mortality. Therefore, taken together our finding of a greater excess rate of ischemic heart disease admissions associated with the same unit increase in $PM_{2.5}$ concentration in after period, compared to the during and after periods, suggests a change in composition (e.g. increase in SOC and decrease in POC) may be responsible. However, more research is needed to explore what pollutant components and/or sources are responsible for health effects and then what source control strategies are most affective.

Our study had several strengths including a large sample size (and its resulting increased statistical power) and the use of a comprehensive statewide dataset including all hospital admissions in NYS from non-federal or psychiatric hospitals during the study period. However, several limitations of this study should also be acknowledged. First, cases within 15 miles of a $PM_{2.5}$ monitoring site were assigned the same values of $PM_{2.5}$ concentration for a specific day, regardless of how close they lived to the site, which likely resulted in exposure misclassification. However, this misclassification error is likely a combination of Berkson and classical error, resulting in a bias toward the null and underestimates of effect (Bateson et al., 2007; Zeger et al., 2000). Second, there was a change in the hospital admission diagnosis codes used in SPARCS starting Oct 1, 2015 (shift from ICD 9 to ICD 10). Certain ICD 9 codes could be divided into multiple ICD 10 codes, possibly resulting in an undercounting of cases in our study. However, this should result in only a loss of statistical power and not bias, as the case-crossover design contrasts pollutant concentrations between 2 time periods within the follow-up time of each case. Last, the air quality policies and economic changes that led to reductions in pollutant concentrations and changes in PM composition affected larger geographic regions than NYS. Thus, there is not a suitable 'control' community that was similar in other ways to the study area but was not impacted by these policies and changes. This limitation, described and discussed previously (Rich, 2017), should not result in any bias in relative rates. However, a similar analysis should be repeated in another location to confirm these findings.

5. Conclusions

Short-term increases in ambient $PM_{2.5}$ concentrations were associated with increased rates of hospital admissions for total cardiovascular disease, cardiac arrhythmias, heart failure, ischemic stroke, ischemic heart disease, and myocardial infarction in NYS. Although both ambient $PM_{2.5}$ concentrations and hospitalization rates of cardiovascular events decreased across the study period (2005–2016), the rate of hospitalizations for most cardiovascular disease subgroups associated with each interquartile range increase in $PM_{2.5}$ concentration was not different after environmental policies were implemented, compared to before and during implementation. However, there was a higher rate of ischemic heart disease and myocardial infarction associated with increased $PM_{2.5}$ concentrations after these environmental policies were implemented and an economic recession occurred (2014–2016), in both the Upstate and New York City sites, compared to previous years (i.e. 2005–2007 and 2008–2013). Given the concomitant changes in PM composition and sources occurring in NYS discussed above, strong oxidants associated with SOC particles and their ability to induce additional oxidative stress may be particularly important types of particles with regard to the triggering of acute cardiovascular events such as myocardial infarction. The impact of pollutant mixtures on acute cardiac outcomes will require further study.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.envpol.2018.08.030.

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