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Triggering of ST-elevation myocardial infarction by ambient wood smoke and other particulate and gaseous pollutants

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Abstract

Background—We previously observed increased odds of ST-elevation myocardial infarctions (STEMI) associated with increased ambient fine particulate matter ($PM_{2.5}$) in the previous hour. However, data are lacking on the effects of specific PM sources.

Methods—Using data from 362 patients, a case-crossover design, and conditional logistic regression, we estimated the relative odds of STEMI associated with increased Delta-C (wood smoke), black carbon (BC; traffic), $PM_{2.5}$, and gaseous pollutants in the previous 1-72 hours.

Results—We did not observe increased odds of STEMI associated with increased Delta-C or BC. We did observe increased odds associated with each 7.1 $\mu g/m^3$ increase in $PM_{2.5}$ (OR [95% CI]: 1.17 [0.99, 1.39]) and each 19.9 ppb increase in ozone (O_3; 1.27 [1.00, 1.63]) in the previous hour, and each 0.22 ppm increase in 48-hour carbon monoxide (CO) concentrations (1.32 [1.00, 1.73]). Larger relative odds were associated with $PM_{2.5}$ in May-October, and O_3 and CO in November-April.

Conclusions—Increased $PM_{2.5}$, O_3, and CO, but not wood smoke or black carbon, were associated with increased odds of STEMI, and effects may differ by season. Studies using spatially-adjusted pollution estimates are needed, as well as studies further examining O_3 and CO effects on the risk of STEMI.

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Keywords

wood smoke; fine particles; myocardial infarction; STEMI; air pollution

INTRODUCTION

Short term increases in ambient particulate matter (PM) concentrations over hours and days have been repeatedly associated with various manifestations of cardiovascular disease (CVD) (1), including the onset of myocardial infarctions (MI) (2, 3). Previously, we observed a significant 18% increase in the odds of ST-elevation myocardial infarction (STEMI), but not non-ST-elevation myocardial infarction (NSTEMI), associated with each 7.1 μg/m^3 increase in fine particulate pollution (<2.5 μm diameter; $PM_{2.5}$) concentrations in the previous hour (3) among patients in Monroe County, NY. However, examination of the odds of STEMI associated with source-specific PM (e.g. PM from traffic or from wood smoke, etc.) has not been reported. Further, a recent workshop on the public health impacts of wood burning for heat and power summarized the evidence for wood smoke effects on respiratory and cardiovascular health, and noted the need for research on the cardiovascular effects of wood smoke (4).

Most ambient $PM_{2.5}$ in the eastern United States is comprised of secondary particles formed from the oxidation of precursor gases such as sulfur dioxide, nitrogen dioxide, and volatile organic compounds (5). $PM_{2.5}$ is also a byproduct of combustion activities, including motor vehicle operation, power plants, and the burning of wood and other biomass for residential heating and other purposes (6). Several previous studies have examined which specific component(s) of PM is/are more strongly associated with health effects. Two reviews by Rohr (7) and Wyzga (8) summarized studies of short and long-term exposure to elevated concentrations of certain PM components and cardiovascular endpoints, including heart rate variability, carotid artery intima media thickness (CIMT), coronary artery calcification, inflammatory and coagulation biomarkers, and CVD hospitalizations and mortality. They reported consistent effects for organic, elemental, and black carbon, sulfate, and certain metals. Lippmann et al. (9) also noted associations between CVD hospitalizations and increased concentrations of elemental and organic carbon, sulfate, and some metals (copper, iron, selenium, silicon, and zinc), but not others (arsenic, potassium, nickel, or lead). Vedal et al. (10) reported greater risks of cardiovascular and cerebrovascular death associated with increased organic carbon (OC) concentrations than with total $PM_{2.5}$, and stronger associations of sulfur with CIMT and coronary artery calcification. Thus, some PM components or pollutant mixtures/sources may be more strongly associated with cardiovascular disease and acute cardiovascular events than total $PM_{2.5}$ and other PM components.

In source apportionment analyses of data from the New York State Department of Environmental Conservation (DEC) pollution monitoring site in Rochester, NY, Wang et al. (11,12) estimated that ~ 9% of the local annual average $PM_{2.5}$ concentration is contributed by wood smoke, with up to 30% contribution in the winter months. These estimates are substantially assisted by light absorption measurements of black carbon (BC), ultraviolet

black carbon (UVBC), and the difference between UVBC and BC, termed Delta-C. Although there is evidence that the respiratory toxicity of particulates from wood smoke is similar to that from other sources (13), it is not clear whether the same is true for cardiovascular toxicity. Some studies have shown wood smoke exposure to be associated with systemic inflammation and markers of CV pathophysiology (14-17) and increased CV hospital admissions and mortality (18, 19). Additionally, localized reductions in wood burning have been associated with decreases in CV mortality and markers of CV pathology (20-22). However, there is a lack of studies specifically examining the association of ambient concentrations of wood smoke with the triggering of MI.

Therefore, the primary purpose of this study was to examine the association of markers of wood smoke (Delta-C) and traffic pollution (BC) with the onset of STEMI. Using data on STEMI patients treated at the University of Rochester Medical Center and hourly measurements of Delta-C, BC, and $PM_{2.5}$ made in Rochester, New York, we hypothesized that increases in concentrations of these pollutants in the previous 1 to 72 hours would be associated with increases in the relative odds of STEMI. We also explored associations between STEMI onset and increased concentrations of gaseous pollutants (nitrogen dioxide [NO_2], sulfur dioxide [SO_2], carbon monoxide [CO], and ozone [O_3]) in the previous few hours and days, and explored whether season modified these associations.

METHODS

Study population and outcomes

The study sample was drawn from a database of acute coronary syndrome (ACS; STEMI, NSTEMI, or unstable angina) patients treated at the Cardiac Catheterization Laboratory (Cath Lab) at the University of Rochester Medical Center in Rochester, NY. The current analysis included all patients residing in Monroe County, NY who presented to the Cath Lab between January 1, 2007 and September 30, 2012 with a STEMI and for whom symptom onset date and time were recorded in the database (n=362). If a patient was admitted to the Cath Lab for multiple MI during the study period, each STEMI was included in the analysis if it was at least three days after that patient's previous MI. The Cath Lab database also contained data on patient demographic and clinical characteristics, including smoking, history of MI, and other comorbidities (peripheral artery disease, heart failure, diabetes, dyslipidemia, and hypertension) that were also used in the current analysis.

American College of Cardiology/American Heart Association guidelines (23) were used at the time of Cath Lab admittance to define the study outcome. STEMI was diagnosed as ST segment elevation on the electrocardiogram of > 1 mm in ≥2 contiguous precordial leads, or ≥2 adjacent limb leads, or new or presumed-new left bundle branch block in the presence of angina or angina equivalent. All study activities were approved by the University of Rochester Research Subjects Review Board.

Air pollution and meteorology measurements

We used measurements of particulate and gaseous pollutant concentrations, temperature, and relative humidity collected between January 1, 2007 and September 30, 2012 at a New York

State Department of Environmental Conservation (DEC) site on the eastside of Rochester, NY. This site is adjacent to two major highways (I-490 and I-590) as well as a major state route (Route 96) that carries traffic to and from downtown Rochester.

Black carbon is a measurement of light absorption at various wavelengths by particles collected on a filter, and semi-continuous measurements can be made using an aethalometer (24). The Rochester DEC site had a single-wavelength (880 nm) aethalometer until 2008, and a two-wavelength unit (370 and 880 nm) from July 2008 to the present (Magee Scientific, Inc., Berkeley, CA). Delta-C is the difference between the BC measured at 370 and 880 nm. We previously identified Delta-C as a marker of wood smoke because it was highly correlated with levoglucosan (25), the compound commonly used as a marker of wood combustion particles (26, 27).

Hourly average $PM_{2.5}$ mass concentrations was measured with a tapered element oscillating microbalance (TEOM, model 1400ab; Thermo Fisher Scientific, Inc., Waltham, MA). Pollutant gases (NO_2, SO_2, O_3, CO) were measured with standard Federal Equivalent Method gas monitors (Thermo Fisher Scientific, Inc., Waltham, MA). Wind speed and direction, ambient temperature, and relative humidity were also measured in 5 minute increments at the DEC site and provided as hourly averages. Hourly average concentrations of each pollutant and weather variables were used in the statistical analyses described below.

Study design

We used a time-stratified case-crossover design (28, 29) that has previously been used in studies of ambient air pollution and MI (3, 30-34). In this design, each patient contributes information as a case during the period before their STEMI and as a matched control during times when they did not experience a STEMI. The case-crossover design is analogous to a matched case-control study, but instead of estimating the relative odds of STEMI by comparing exposure between persons (i.e. cases versus controls), we estimate the relative odds of STEMI by comparing exposure during different time periods within the follow-up time of each STEMI case. Because case periods and their matched control periods are derived from the same person and a conditional analysis is conducted, non-time varying confounders such as age, chronic comorbidities, and long-term smoking history are controlled by design.

However, variables that may be related to both air pollution and the relative odds of STEMI that vary over short time periods (e.g. weather conditions) are possible confounders that must be included in our analytic models. Case periods for this analysis were defined as the 1, 12, 24, 48, and 72-hour periods before the time of STEMI symptom onset, while control periods (3-4 per case, depending on the number of days in the calendar month) were matched to the case period by day of the week, time of the day, year, and month. For example, if a patient's symptom onset date and time were Friday, March 20, 2009 at 12:00 a.m., then the 24-hour case period was the 24 hours prior to that date and time (12:00 a.m. to 11:59 p.m. on Thursday, March 19, 2009), and control periods were 12:00 a.m. to 11:59 p.m. on Thursdays, March 5th, 12th, and 26th in 2009. Pollutant concentrations corresponding to these case and control periods were then contrasted in the statistical analyses described below.

Statistical analysis

We first calculated the mean concentrations of each pollutant (Delta-C, BC, $PM_{2.5}$, NO_2, SO_2, O_3, and CO) for the 1 hour, 12 hours, 24, 48, and 72 hours prior to each case and control date. We then used conditional logistic regression models, stratified on each case-control set, to estimate the relative odds (and 95% confidence intervals) of STEMI associated with interquartile range (IQR) increases in pollutant concentrations during each lag period. To determine which time lags of weather variables and which functional form of that weather variable should be included in the models, we first fit separate conditional regression models using 1, 12, 24, 48, and 72-hour mean temperature and relative humidity with natural splines with 1 to 4 degrees of freedom (df). From this group of models, we chose the time lag/df with the lowest value of Akaike's Information Criterion. This selection process resulted in the inclusion of linear terms (1 df) for 3-hour mean temperature and relative humidity in all models.

We also conducted stratified analyses to determine whether there were seasonal differences in the effects of pollutants on the odds of STEMI. We fit similar models to those described above including only the events that occurred from November through April (colder months), and then again including only those that occurred from May through October (warmer months). We also fit models including all subjects and a term for the interaction between pollutants and season (warm vs. cold).

To explore whether observed responses to pollutants differed depending on patient characteristics, we also separately fit models that included interactions between pollutant concentrations and the following variables: sex; age (< 65 vs. ≥ 65 years old); race/ethnicity (non-Hispanic white vs. other); smoking; hypertension, diabetes, dyslipidemia, prior MI, and prior cardiovascular disease (CVD). We defined prior CVD by a “yes” response to “Prior CVD” in a patient's Cath Lab record, or documentation of a prior MI, coronary artery bypass graft, or percutaneous coronary intervention. We used SAS version 9.3 (SAS Institute, Inc., Cary, NC) to construct all data sets and perform descriptive analyses, and R version 3.0.1 (R Foundation for Statistical Computing, Vienna, Austria) for all conditional logistic regression models.

RESULTS

A total of 366 STEMI events among 362 patients were included in the analysis. Most patients (57%) were between 50 and 69 years old (mean age ±SD = 62.3 ±12.9 yrs). Two thirds (67%) of subjects were male, most were non-Hispanic white (70%), nearly half were smokers (44%), and most (74%) were either overweight or obese (BMI ≥25 kg/m^2; Table 1).

The distributions of hourly pollutant concentrations during the whole study period and by season are shown in Table 2. Delta-C concentrations were, on average, more than twice as high in the colder months compared to the warmer months of the year. $PM_{2.5}$, NO_2, SO_2, and CO concentrations were also higher in the colder months as would be expected with the lower atmospheric mixed layer depth. Delta-C was moderately correlated with whole-year NO_2 (Table 3; r=0.52) and NO_2 in the colder months (r=0.56). Delta-C was also positively correlated with whole-year and seasonal BC concentrations (r=0.37-0.57), whole-year and

seasonal CO (r=0.32-0.41), and whole-year and cold season $PM_{2.5}$ concentrations (r=0.31-0.48). There were moderately strong negative correlations of hourly concentrations of Delta-C and BC with O_3 over the whole year and in the colder months (r= −0.32 to −0.54). Black carbon was also positively correlated with NO_2 (r=0.70-0.77) and $PM_{2.5}$ (r=0.57-0.63), and moderately correlated with CO (r=0.41-0.46). $PM_{2.5}$ and NO_2 were positively correlated for the whole year (r=0.38) and in the colder months (r=0.49).

Increases in Delta-C and BC concentrations were not associated with increases in the relative odds of STEMI over any of the time lags examined (Table 4). However, each interquartile range (IQR; 7.1 μg/m^3) increase in $PM_{2.5}$ was associated with a 17% (95% CI: −1%, 39%) increase in the relative odds of STEMI. There were small decreases in the odds of STEMI associated with each IQR (0.002 ppb) increase in 1 to 72-hour concentrations of SO_2, though only the association with SO_2 concentration in the previous hour was statistically significant (OR=0.94; 95% CI: 0.90, 0.99).

Interquartile range increases in 1 to 72-hour concentrations of O_3 were generally associated with increases in the odds of STEMI. Each 19.9 ppb increase in O_3 concentration in the previous hour was associated with a 27% (95% CI: 0%, 63%) increase in the odds of STEMI. Similarly, each 0.222 ppm increase in CO concentration in the previous 48 hours was associated with a 32% (95% CI: 0%, 73%) increase in the relative odds of STEMI. We did not observe increased odds of STEMI associated with increased NO_2 concentrations at any time lag.

We did not observe significant differences in the odds of STEMI associated with increases in concentrations of Delta-C or BC between the warm (May-October) and cold seasons (November-April; Table 5). However, the relative odds of STEMI associated with each 7.1 μg/m^3 increase in $PM_{2.5}$ concentration in the previous hour was slightly larger during the warmer months (OR=1.27; 95% CI: 0.99, 1.62) than during the colder months of the year (OR=1.14; 95% CI: 0.89, 1.45). Increases in O_3 concentrations during the colder months were associated with increased odds of STEMI across all time lags, with the largest relative odds associated with increased O_3 concentrations in the previous 72 hours (OR=1.60; 95% CI: 1.05, 2.46). We observed no such increased relative odds associated with O_3 in the warmer months. We also observed larger relative odds of STEMI associated with increased CO concentration in the colder months than the warmer months, with a 43% (95% CI: 5%, 95%) increase in the relative odds of STEMI associated with each 0.22 ppm increase in CO concentration in the previous 48 hours (Table 5).

We also examined whether the odds of STEMI associated with Delta-C, BC, and O_3 concentrations in the previous hour were modified by patient characteristics (Table 6). We did not observe differences in the odds of STEMI associated with increased concentrations of Delta-C in the previous hour between strata of age, sex, race/ethnicity, smoking status, history of MI or CVD, or other comorbidities. However, we did observe larger increases in the relative odds of STEMI per IQR increase in O_3 concentration in the previous hour in patients with a prior MI (OR=1.78; 95% CI: 0.97, 3.28) or prior CVD (OR=1.72; 95% CI: 1.02, 2.90), but not among those without a history of MI (OR=1.20; 0.92, 1.58) or CVD (OR=1.15; 95% CI: 0.81, 1.64), although the interactions were not statistically significant.

Discussion

Using a case-crossover design and conditional logistic regression, we did not observe an increase in the odds of STEMI associated with increases in 1 to 72-hour ambient concentrations of Delta-C (marker of wood smoke) or BC (marker of traffic pollution) as hypothesized. However, we did confirm an increased relative odds of STEMI associated with each IQR increase in $PM_{2.5}$ concentration in the previous hour (OR=1.17; 95% CI: 0.99, 1.39), as well as an increased odds of STEMI associated with increases in ambient O_3 and CO concentrations in exploratory analyses.

To our knowledge, this is the first study to specifically examine the association between ambient concentrations of wood smoke and the risk of an MI. Our findings are consistent with those that have observed no association between forest/vegetation fire air pollution and CV events (22, 35-37). However, some controlled exposure studies (14,17) and studies of occupational wood smoke exposure in firefighters (15,16) have observed adverse effects of wood smoke on biomarkers that are associated with acute coronary events, including coagulation factors, inflammatory proteins, arterial stiffness, and heart rate variability. However, measured and estimated total $PM_{2.5}$ concentrations during controlled and occupational wood smoke exposure studies have generally exceeded 300 $\mu g/m^3$. These values are in contrast with a maximum ambient $PM_{2.5}$ concentration in the current study of only 79 $\mu g/m^3$ and a median concentration of only 6.3 $\mu g/m^3$. It is possible that the local wood smoke PM concentrations in this study were not high enough to elicit the acute changes in CV function necessary to trigger a STEMI.

The observed 17% increase in the relative odds of STEMI associated with a 7.1 $\mu g/m^3$ increase in $PM_{2.5}$ concentrations in the previous hour (using STEMI from 2007 – 2012) is consistent with our previous work using a subset of these data (STEMI from 2007-2010). That study showed an 18% increase in the odds of STEMI for each 7.1 $\mu g/m^3$ increase in $PM_{2.5}$ in the previous hour (3), and a 10% increase in the odds of a transmural MI associated with each 10.8 $\mu g/m^3$ increase in $PM_{2.5}$ in the 24 hours before emergency room arrival for that MI (34). Our null findings for the effects of 1 to 72-hour BC concentrations, on the other hand, are in contrast to previous studies which have observed adverse effects of elevated BC on cardiac function and MI risk (38-40). Zanobetti and Schwartz (39) found increased BC concentrations in the previous two days to be associated with significant increases in the risk of hospitalization for an MI. The difference between those findings and the current study results may be attributable to differences in sample size (N>15,000 vs. 366), age (all >65 yrs. vs. 31-95 yrs.), the approximately 50% lower BC concentrations in our study area (24-hour median [IQR] = 0.51 [0.44] vs. 1.15 [0.98]), and/or a higher proportion of the BC arising from wood smoke in Rochester (11,12) compared to Boston (41) where it is primarily emitted by traffic.

In our main analysis including events in the entire year, we observed a 27% increase in the odds of STEMI (95% CI: 0%, 63%) associated with each 19.9 ppb increase in O_3 concentrations in the previous hour. Previous work offers conflicting evidence regarding the association between O_3 and MI risk. A 2005 meta-analysis (42) of 25 U.S. and non-U.S. studies showed a small (1.11%) but significant increase in cardiovascular mortality

associated with 1 to 2-day increases in ambient O_3 concentrations, which was greater than the effects of O_3 on either total or respiratory mortality. A more recent meta-analysis (2), however, showed an association between MI risk and increased concentrations of fine and ultrafine particles, CO, NO_2, and SO_2, but no association with O_3. Nuvolone et al. (43) observed a significant 6.3% increase in coronary deaths (but not MI hospitalizations) per 0.005 ppm increase in 6-day O_3 concentrations. They also found a greater effect of O_3 on coronary mortality among patients with a history of vascular diseases, which is consistent with the greater relative odds of STEMI that we observed among patients with a prior MI or other indication of prior CVD. Unexpectedly, our seasonal analyses showed larger O_3 effects in the colder months of the year, compared to the warmer months. This finding is in contrast to the results of previous work showing a small (<1%) but significant increase in CV deaths associated with each 10 $\mu g/m^3$ (0.005 ppm) increase in 1- and 8-hour O_3 concentrations in the warmer (April-September) but not colder (October-March) months of the year (44).

Ground-level O_3 is formed primarily from the reaction of sunlight with pollutants such as volatile organic compounds and nitrogen oxides (45). Although O_3 levels are generally lower during the winter months, higher cold-weather O_3 concentrations can be observed on brighter, clearer days with less cloud cover and more sunlight. Safieddine et al. (46) found that NO_2 concentrations in major cities in the northern hemisphere are consistently higher during winter because of the high anthropogenic sources and longer lifetimes. Thus, bright sunny days, particularly with the sunlight reflecting from snow on the ground, will produce high rates of NO_2 photolysis and rapid O_3 production. In this study, O_3 levels were not substantially lower in the winter than in the summer, and thus, this increased odds of STEMI associated with increased winter O_3 concentrations may reflect real pollutant exposures. However, clear winter days (bright sunny days with a high ceiling) may also be associated with higher physical activity levels among those at risk for an MI. Given the increased risk of MI that has been associated with moderate to strenuous physical exertion (47-49), which may be greater with outdoor versus indoor exertion (49), the greater odds of STEMI associated with cold-weather O_3 may also be due to residual confounding by an increase in patients' physical activity levels on the clear sunny days associated with high wintertime O_3. However, it is not clear whether the observed association between cold-weather O_3 concentrations and odds of STEMI is due to a real effect of O_3, confounding by physical activity, or residual confounding by some other factor.

Our results also suggest an association between ambient CO levels and odds of STEMI, particularly over the longer time lags and during the colder months of the year. A meta-analysis by Mustafic et al. (2) including estimates from 20 studies provided a significant 5% increase in MI risk associated with each 1 mg/m^3 (~0.81 ppm) increase in 24-hour CO concentrations. Though a much smaller effect size, this is in agreement with our results showing a 32% (95% CI: 0%, 73%) increase in the odds of STEMI per 0.222 ppm increase in 48-hour CO. Although it is not clear if or how CO and O_3 exposures trigger a STEMI, these effects may be mediated by the same mechanisms believed to mediate the relationship between STEMI and $PM_{2.5}$, including increases in inflammation, coagulation, and thrombosis (50-56).

A major strength of this analysis is the inclusion of symptom onset time in the Cath Lab database from which patients were selected, whereas only the date of an MI or hospital admission has often been available from the data sources used in previous studies. This likely resulted in reduced exposure error and less bias towards the null in our effect estimates, compared to studies using only hospital or emergency room arrival date/time. However, there were study limitations to consider when making inference. First, we used measurements of hourly Delta-C, BC, and other pollutant concentrations from a single monitoring station for all subjects, regardless of the distance between the monitor and where they lived, worked, and spent time. This allowed us to examine triggering of STEMI by air pollutant concentrations in specific hours, not just days, before STEMI symptom onset. However, in 2009-2010, we used a mobile pollution monitor to assess spatial variations in BC and Delta-C in Rochester (57) and detected “hotspots” for both pollutants, indicating high spatial heterogeneity in BC and Delta-C concentrations across the area. This suggests that the hourly Delta-C and BC concentrations assigned to subjects in the current analysis based on the DEC monitor measurements may not accurately approximate individual subject's exposures (i.e. exposure error). However, this exposure error should be non-differential with regard to case/control times, resulting a bias towards the null and underestimates of risk. This limitation highlights the need to develop local spatial-temporal models of Delta-C, BC, and other pollutants to reduce exposure error and bias, providing better estimates of the odds of STEMI associated with increased Delta-C, BC, and other pollutant concentration in the previous few hours and days.

In conclusion, although this study adds to the literature supporting a role of elevated $PM_{2.5}$ in increasing the odds of STEMI, concentrations of Delta-C and BC (markers of wood smoke and perhaps traffic), as measured by our local monitoring site, do not appear to be associated with the triggering of STEMI. In addition to obtaining more locally accurate pollution measurements via spatial-temporal modeling, future work should also aim to confirm and clarify the mechanisms behind our observed O_3/STEMI and CO/STEMI associations, and the seasonal variations in those associations.

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Table 1

Subject characteristics (total N=366 STEMI among 362 subjects)

	N	%
Age		
<50 years	61	17
50-59 years	106	29
60-69 years	102	28
70-79 years	57	16
≥ 80 years	40	11
Sex (n=365)[1]		
Male	249	68
Female	116	32
Missing	1	<1
Race/ethnicity (n=351)		
Non-Hispanic white	258	74
Non-Hispanic black	51	15
Hispanic	8	2
Other	34	10
Clinical presentation		
Prior cardiovascular disease (n=190)	79	42
Prior myocardial infarction (n=363)	59	16
Prior percutaneous intervention (n=356)	58	16
Prior coronary artery bypass (n=363)	14	4
Prior peripheral artery disease (n=356)	24	7
Prior heart failure (n=355)	9	3
Smoker (n=355)	160	45
Hypertension (n=361)	256	71
Dyslipidemia (n=363)	214	59
Diabetes (n=334)	63	19
BMI (n=339)		
Overweight (BMI 25.0-29.9)	145	43
Obesity: class I (BMI 30.0-34.9)	84	25
Obesity: class II (BMI 35.0-39.9)	31	9
Obesity: class III (BMI ≥40)	11	3
Mean (SD)	29.0 (5.1)	-

[2] As indicated by “Prior CVD” in Cath Lab record, or a prior MI, PCI, or CABG

[1] Number of events with subject data available for a given characteristic

Table 2

Distribution of hourly pollutant concentrations, temperature, and humidity, January 1, 2007 – September 30, 2012

			Percentiles				
	Mean (SD)	Min	25th	50th	75th	Max	IQR
Delta-C (μg/m^3)							
Whole year	0.13 (0.26)	−1.94	0.02	0.06	0.15	5.40	0.13
November-April	0.20 (0.32)	−1.94	0.05	0.11	0.23	5.40	0.18
May-October	0.07 (0.17)	−1.43	0.004	0.03	0.08	4.60	0.08
Black carbon (μg/m^3)							
Whole year	0.58 (0.51)	−0.22	0.24	0.44	0.77	11.74	0.53
November-April	0.51 (0.47)	−0.22	0.22	0.38	0.64	5.80	0.43
May-October	0.65 (0.53)	−0.07	0.27	0.52	0.89	11.74	0.61
Fine particles ($PM_{2.5}$; μg/m^3)							
Whole year	7.62 (4.46)	−4.90	3.20	6.30	10.30	79.20	7.10
November-April	8.18 (5.18)	−4.30	4.64	7.30	10.60	79.20	5.96
May-October	7.08 (7.45)	−4.90	2.00	4.90	9.80	64.01	7.80
Nitrogen dioxide (ppb)							
Whole year	8.11 (6.78)	−2.40	3.60	6.10	10.50	58.00	6.90
November-April	9.44 (7.77)	−0.90	4.10	7.00	11.90	58.00	7.80
May-October	7.01 (5.62)	−2.40	3.10	5.40	9.30	46.50	6.20
Sulfur dioxide (ppb)							
Whole year	0.003 (0.008)	−0.0001	0.001	0.002	0.003	0.144	0.002
November-April	0.003 (0.007)	−0.0001	0.001	0.002	0.003	0.053	0.002
May-October	0.003 (0.009)	−0.0001	0.001	0.001	0.002	0.144	0.001
Ozone (ppb)							
Whole year	27.4 (15.1)	0.0	17.0	27.0	36.9	104.0	19.9
November-April	25.4 (12.7)	0.0	17.0	26.0	34.0	92.0	17.0
May-October	29.4 (17.0)	0.0	17.0	28.0	40.3	104.0	23.3
Carbon monoxide (ppm)							
Whole year	0.35 (0.19)	0.01	0.20	0.30	0.42	2.20	0.22
November-April	0.37 (0.21)	0.05	0.20	0.32	0.50	1.62	0.30
May-October	0.32 (0.16)	0.01	0.20	0.30	0.40	2.20	0.20
Temperature (°F)							
Whole year	52.1 (20.1)	1.2	35.7	52.8	67.9	100.5	32.2
November-April	36.7 (13.9)	1.2	27.2	35.5	44.9	91.0	17.7
May-October	67.0 (12.7)	29.9	58.4	67.0	75.9	100.5	17.5
Relative humidity (%)							
Whole year	64.7 (19.5)	5.0	50.0	67.0	81.0	96.4	31.0
November-April	66.1 (18.7)	11.9	54.0	69.0	81.0	96.4	27.0
May-October	63.2 (20.1)	5.0	47.0	65.5	80.8	96.0	33.8

Table 3

Whole year and seasonal Pearson correlations among hourly pollutant concentrations, temperature, and relative humidity, January 1, 2007 – September 30, 2012

	Black carbon	$PM_{2.5}$	Nitrogen dioxide	Sulfur dioxide	Ozone	Carbon monoxide	Temp.	Relative humidity
Delta-C (μg/m^3)								
Whole year	0.43	0.31	0.52	−0.01	−0.36	0.40	−0.33	0.17
Nov – April	0.57	0.48	0.56	−0.02	−0.44	0.41	−0.20	0.19
May – Oct	0.37	0.11	0.37	0.00	−0.29	0.32	−0.32	0.17
BC (μg/m^3)								
Whole year	-	0.57	0.70	−0.01	−0.32	0.41	0.12	0.22
Nov - April		0.63	0.77	−0.01	−0.54	0.46	0.11	0.18
May – Oct		0.58	0.75	−0.01	−0.22	0.44	−0.05	0.27
$PM_{2.5}$ (μg/m^3)								
Whole year	-	-	0.38	−0.01	0.11	0.29	0.06	0.06
Nov - April			0.49	−0.04	−0.29	0.30	0.01	0.13
May - Oct			0.31	0.01	0.34	0.28	0.33	0.01
NO_2 (ppb)								
Whole year	-	-	-	0.10	−0.57	0.79	−0.30	0.29
Nov - April				0.12	−0.72	0.81	−0.21	0.25
May – Oct				0.04	−0.47	0.74	−0.32	0.34
SO_2 (ppb)								
Whole year	-	-	-	-	0.03	0.02	0.02	−0.01
Nov - April					0.06	0.04	0.03	−0.01
May – Oct					0.01	0.01	0.03	−0.01
O_3 (ppb)								
Whole year	-	-	-	-	-	−0.36	0.43	−0.59
Nov - April						−0.47	0.33	−0.55
May – Oct						−0.25	0.67	−0.62
CO (ppm)								
Whole year	-	-	-	-	-	-	−0.26	0.27
Nov - April							−0.25	0.28
May – Oct							−0.19	0.24
Temp. (°F)								
Whole year	-	-	-	-	-	-	-	−0.38
Nov - April								−0.41
May – Oct								−0.58

Table 4

Relative odds of STEMI associated with interquartile range (IQR) increases in mean pollutant concentrations, by lagged averaging time. All models adjusted for 3-hour temperature and relative humidity.

	Time lag	IQR	N	Events	Odds ratio (95% CI)
Delta-C (μg/m^3)	1 hr	0.129	274	277	0.99 (0.92-1.07)
	12 hrs	0.132	275	278	0.97 (0.87-1.08)
	24 hrs	0.134	275	278	0.97 (0.85-1.10)
	48 hrs	0.138	268	271	1.03 (0.87-1.22)
	72 hrs	0.137	264	267	1.00 (0.83-1.22)
Black carbon (μg/m^3)	1 hr	0.531	336	339	1.00 (0.85-1.17)
	12 hrs	0.477	337	340	1.02 (0.85-1.22)
	24 hrs	0.435	337	340	1.07 (0.88-1.30)
	48 hrs	0.374	330	333	1.07 (0.87-1.31)
	72 hrs	0.333	326	329	0.96 (0.78-1.17)
Fine particles ($PM_{2.5;}$ μg/m^3)	1 hr	7.10	334	339	1.17 (0.99-1.39)
	12 hrs	6.37	330	340	1.11 (0.93-1.33)
	24 hrs	6.03	327	340	1.17 (0.96-1.41)
	48 hrs	5.53	331	333	1.10 (0.91-1.34)
	72 hrs	5.12	324	329	0.98 (0.80-1.21)
Nitrogen dioxide (ppb)	1 hr	6.90	65	66	0.86 (0.62-1.20)
	12 hrs	5.67	58	59	1.01 (0.72-1.42)
	24 hrs	4.76	61	62	0.97 (0.68-1.36)
	48 hrs	4.25	62	63	1.04 (0.71-1.52)
	72 hrs	3.81	59	60	1.08 (0.71-1.65)
Sulfur dioxide (ppb)	1 hr	0.002	347	351	0.94 (0.90-0.99)
	12 hrs	0.002	343	347	0.95 (0.89-1.01)
	24 hrs	0.002	349	353	0.95 (0.89-1.02)
	48 hrs	0.002	349	353	0.93 (0.87-1.00)
	72 hrs	0.002	347	351	0.93 (0.86-1.00)
Ozone (ppb)	1 hr	19.9	345	349	1.27 (1.00-1.63)
	12 hrs	15.7	342	346	1.20 (0.95-1.51)
	24 hrs	13.5	348	352	1.14 (0.91-1.44)
	48 hrs	12.6	347	351	1.09 (0.84-1.41)
	72 hrs	12.2	345	349	1.17 (0.87-1.57)
Carbon monoxide (ppm)	1 hr	0.223	336	340	1.14 (0.92-1.40)
	12 hrs	0.230	334	338	1.19 (0.93-1.51)
	24 hrs	0.221	339	343	1.15 (0.90-1.48)
	48 hrs	0.222	341	345	1.32 (1.00-1.73)
	72 hrs	0.221	339	343	1.25 (0.94-1.68)

Table 5

Relative odds of STEMI associated with interquartile range (IQR) increases in mean pollutant concentrations, by season (Nov – April vs. May – Oct). All models adjusted for 3-hour temperature and relative humidity.

			November 1 – April 30		May 1 – October 31		interaction p-value
	Time lag	IQR	Events	Odds ratio (95% CI)	Events	Odds ratio (95% CI)	
Delta-C (μg/m^3)	1 hr	0.129	162	0.99 (0.91-1.07)	115	1.01 (0.81-1.27)	0.78
	12 hrs	0.132	162	0.97 (0.87-1.08)	116	0.94 (0.63-1.39)	0.88
	24 hrs	0.134	161	0.97 (0.85-1.11)	116	0.93 (0.60-1.46)	0.86
	48 hrs	0.138	159	1.04 (0.87-1.24)	112	0.91 (0.51-1.61)	0.83
	72 hrs	0.137	160	0.98 (0.79-1.20)	107	1.15 (0.65-2.03)	0.50
Black carbon (μg/m^3)	1 hr	0.531	218	0.97 (0.80-1.17)	121	1.10 (0.81-1.49)	0.77
	12 hrs	0.477	218	0.96 (0.77-1.19)	122	1.21 (0.84-1.74)	0.44
	24 hrs	0.435	218	1.01 (0.81-1.27)	122	1.24 (0.83-1.84)	0.58
	48 hrs	0.374	215	1.08 (0.85-1.37)	118	1.03 (0.69-1.55)	0.69
	72 hrs	0.333	216	0.94 (0.74-1.20)	113	0.99 (0.66-1.50)	0.95
Fine particles ($PM_{2.5;}$ μg/m^3)	1 hr	7.10	214	1.14 (0.89-1.45)	123	1.27 (0.99-1.62)	0.90
	12 hrs	6.37	210	1.13 (0.88-1.46)	123	1.16 (0.88-1.52)	0.67
	24 hrs	6.03	211	1.18 (0.91-1.54)	119	1.19 (0.90-1.58)	0.69
	48 hrs	5.53	211	1.21 (0.92-1.59)	123	1.03 (0.77-1.38)	0.24
	72 hrs	5.12	206	1.06 (0.80-1.41)	121	0.93 (0.68-1.27)	0.31
Ozone (ppb)	1 hr	19.9	220	1.33 (0.94-1.88)	129	1.28 (0.87-1.88)	0.79
	12 hrs	15.7	217	1.43 (1.03-1.98)	129	1.03 (0.72-1.46)	0.29
	24 hrs	13.5	219	1.45 (1.04-2.03)	133	0.95 (0.67-1.35)	0.14
	48 hrs	12.6	218	1.37 (0.94-2.01)	133	0.92 (0.63-1.36)	0.21
	72 hrs	12.2	216	1.60 (1.05-2.46)	133	0.93 (0.60-1.44)	0.10
Carbon monoxide (ppm)	1 hr	0.223	210	1.22 (0.96-1.54)	130	0.96 (0.59-1.57)	0.13
	12 hrs	0.230	208	1.24 (0.95-1.64)	130	1.10 (0.64-1.89)	0.35
	24 hrs	0.221	209	1.22 (0.92-1.63)	134	1.00 (0.58-1.75)	0.25
	48 hrs	0.222	211	1.43 (1.05-1.95)	134	1.02 (0.57-1.83)	0.15
	72 hrs	0.221	209	1.36 (0.97-1.91)	134	0.99 (0.55-1.78)	0.18

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J Expo Sci Environ Epidemiol. Author manuscript; available in PMC 2018 March 01.

Table 6

Relative odds of a STEMI associated with interquartile range[1] increases in Delta-C, black carbon, and $PM_{2/5}$ concentrations in the previous hour, stratified by demographic and clinical characteristics. All models adjusted for 3-hour temperature and relative humidity.

	1-hour Delta-C			1-hour black carbon			1-hour ozone		
	Events	**Odds ratio (95% CI)**	**interaction p-value**	**Events**	**Odds ratio (95% CI)**	**interaction p-value**	**Events**	**Odds ratio (95% CI)**	**interaction p-value**
Age									
< 65 yrs	172	1.00 (0.91-1.10)	0.80	204	1.09 (0.89-1.33)	0.22	207	1.23 (0.90-1.68)	0.96
≥ 65 yrs	105	0.98 (0.86-1.10)		135	0.86 (0.67-1.12)		142	1.36 0.92-2.01)	
Sex									
Male	192	1.01 (0.93-1.09)	0.55	233	0.99 (0.83-1.19)	0.89	238	1.31 (0.98-1.76)	0.97
Female	85	0.95 (0.80-1.13)		106	1.00 (0.73-1.37)		111	1.20 (0.76-1.87)	
Race									
White[2]	184	0.98 (0.89-1.07)	0.17	235	0.95 (0.78-1.16)	0.88	242	1.29 (0.95-1.74)	0.61
Other	80	1.14 (0.93-1.40)		91	1.01 (0.73-1.39)		92	1.34 (0.86-2.09)	
Smoker									
Yes	129	0.99 (0.89-1.10)	0.95	152	1.08 (0.87-1.33)	0.33	152	1.12 (0.78-1.60)	0.47
No	137	0.99 (0.89-1.11)		176	0.87 (0.68-1.14)		186	1.52 (1.06-2.17)	
Prior MI									
Yes	50	0.90 (0.72-1.14)	0.27	54	0.88 (0.60-1.29)	0.71	59	1.78 (0.97-3.28)	0.49
No	224	1.02 (0.94-1.11)		282	1.02 (0.86-1.22)		287	1.20 (0.92-1.58)	
Prior CVD									
Yes	61	0.94 (0.80-1.11)	0.51	73	0.86 (0.61-1.22)	0.48	79	1.72 (1.02-2.90)	0.44
No	158	1.00 (0.88-1.15)		158	1.12 (0.87-1.45)		162	1.15 (0.81-1.64)	
Diabetes									
Yes	42	0.99 (0.80-1.22)	0.89	55	0.99 (0.66-1.49)	0.63	60	1.12 (0.63-2.00)	0.63
No	207	1.00 (0.92-1.08)		256	1.03 (0.86-1.23)		258	1.30 (0.97-1.73)	
Hypertension									
Yes	195	0.98 (0.90-1.08)	0.82	238	1.05 (0.87-1.26)	0.24	245	1.34 (1.00-1.90)	0.60
No	77	1.00 (0.87-1.15)		96	0.81 (0.57-1.17)		99	1.26 (0.78-2.03)	
Dyslipidemia									

	1-hour Delta-C			1-hour black carbon			1-hour ozone		
	Events	**Odds ratio (95% CI)**	**interaction p-value**	**Events**	**Odds ratio (95% CI)**	**interaction p-value**	**Events**	**Odds ratio (95% CI)**	**interaction p-value**
Yes	172	0.94 (0.84-1.07)	0.89	203	1.05 (0.85-1.27)	0.63	203	1.23 (0.89-1.72)	0.63
No	102	1.03 (0.93-1.14)		133	0.92 (0.71-1.19)		143	1.35 (0.93-1.96)	

[1] Interquartile ranges: Delta-C = 0.129 μg/m^3; black carbon = 0.531 μg/m^3; ozone = 19.9 ppb

[2] Non-Hispanic