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Fine Particulate Air Pollution and Premature Atrial Contractions: The REasons for Geographic And Racial Differences in Stroke Study

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Abstract

Background—Several reports have suggested that particulate matter (PM) exposure increases the risk for atrial arrhythmias. However, data from large-scale epidemiologic studies supporting this hypothesis are lacking.

Methods—We examined the association of PM <2.5 micrometers in diameter (PM_{2.5}) concentration with premature atrial contractions (PACs) in 26,609 (mean age=65 ± 9.4 years; 55% female; 41% black) participants from the REasons for Geographic And Racial Differences in Stroke (REGARDS) study. Estimates of short- (2-week) and long-term (1-year) PM_{2.5} exposure were computed prior to each participant's baseline visit using geographic information system data

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DISCLOSURES

None.

on the individual level at the coordinates of study participants' residences. PACs were identified from baseline electrocardiograms.

Results—A total of 2,140 (8.2%) had evidence of PACs on the baseline electrocardiogram. Short-term PM_{2.5} (per 10 µg/m³) exposure was not associated with PACs (OR=1.09, 95%CI=0.98, 1.23). Increases in long-term PM_{2.5} (per 10 µg/m³) were associated with PACs (OR=1.40, 95%CI=1.10, 1.78). Interactions were not detected for short- and long-term PM_{2.5} exposure by age, sex, or race.

Conclusion—Long- but not short-term PM_{2.5} exposure is associated with PACs. This suggests a role for long-term PM_{2.5} exposure in initiating supraventricular arrhythmias that are triggered by PACs.

Keywords

particulate matter; epidemiology; population based studies

INTRODUCTION

Several reports have suggested that particulate matter (PM) exposure increases the risk for the development of arrhythmias. This is supported by data that have linked short-term (24-hour) PM exposure with an increased risk for ventricular arrhythmias (ventricular tachycardia and ventricular fibrillation) in patients with implantable cardioverter-defibrillators (ICD) (1, 2), and among persons with coronary heart disease (3). Additionally, a number of studies have suggested that air pollution is associated with the development of atrial arrhythmias. Two studies among patients with ICDs have shown that increases in ozone and PM_{2.5} (PM<2.5 micrometers in diameter) concentrations are associated with atrial fibrillation events (4, 5). Other reports have linked short-term PM_{2.5} exposure with premature atrial contractions (PACs) (3, 6, 7).

Data from large-scale epidemiologic studies to support an association between PM exposure and atrial arrhythmias are lacking. The findings of the aforementioned studies are limited in generalizability as they were conducted in specific patient populations (e.g., patients with ICDs) with small sample sizes. Furthermore, the impact of long-term PM exposure on the risk of atrial arrhythmias has not been explored. Therefore, the purpose of this study was to examine the associations of short- and long-term PM_{2.5} exposure with non-sustained atrial rhythm disorders (PACs) in the REasons for Geographic And Racial Differences in Stroke (REGARDS) study.

PATIENTS AND METHODS

Study Population and Design

Details of REGARDS have been published previously (8). Briefly, REGARDS is a prospective cohort study designed to identify causes of regional and racial disparities in stroke mortality. The study over sampled blacks and persons residing in the stroke belt (North Carolina, South Carolina, Georgia, Alabama, Mississippi, Tennessee, Arkansas, and Louisiana) between January 2003 and October 2007. This included participants from the

stroke buckle (coastal plains of North Carolina, South Carolina and Georgia), as this region experiences a stroke mortality rate considerably higher than the rest of the United States (9). A total of 30,239 participants were recruited from a commercially available list of residents using postal mailings and telephone data. Demographic information and medical histories were obtained using a computer-assisted telephone interview (CATI) system that was conducted by trained interviewers. Additionally, a brief in-home physical examination was performed 3 to 4 weeks after the telephone interview. During the in-home visit, trained staff collected information regarding medications, blood and urine samples, and a resting electrocardiogram. All participants provided written informed consent and the study was approved by all participating institutional review boards.

For the purpose of this analysis, participants were excluded if they were missing PM_{2.5} exposure or baseline covariate data. Additionally, participants who had evidence of pacemakers or sustained atrial rhythm disorders (e.g., atrial fibrillation) were excluded.

PM_{2.5} Exposure

PM_{2.5} particles are air pollutants with an aerodynamic diameter less than 2.5 micrometers and measures are in micrograms per cubic meter ($\mu\text{g}/\text{m}^3$). Spatial surfaces of estimated PM_{2.5} exposures on a 10-km grid were generated using United States Environmental Protection Agency (EPA) ground observation data and National Aeronautics and Space Administration's (NASA) MODerate-resolution Imaging Spectroradiometer (MODIS) data between 2003 and 2008. Estimates were spatially linked with REGARDS participants using geographic information system (GIS) data on the individual level at the geographic coordinates of study participants' residences. Short-term PM_{2.5} exposure was computed as the 2-week average value prior to the baseline visit and long-term exposure was computed as the 1-year average value prior to the baseline visit. Details of the methods used to compute PM_{2.5} exposure and linkage with REGARDS participants have been reported (10).

Data from the NASA product of the North American Land Data Assimilation System Phase 2 (NLDAS-2) forcing dataset were used to determine 2-week and 1-year temperature averages prior to the baseline visit. This NLDAS-2 product is based on model re-analysis data, as well as remotely-sensed and ground observations, and consists of a grid surface with ~14 km resolution over North America (11).

Premature Atrial Contractions

Electrocardiograms of study participants were read centrally at the Epidemiological Cardiology Research Center (EPICARE) located at the Wake Forest School of Medicine (Winston-Salem, NC). PACs were ascertained from baseline electrocardiograms using Minnesota code criteria (Minnesota codes 8.1.1 and 8.1.3) (12).

Covariates

Age, sex, race, income, education, and smoking status were self-reported. Education was categorized into "high school or less," or "some college or more." Smoking was defined as self-reported current cigarette use. Exercise was classified as none or 1 times per week. Fasting blood samples were obtained and assayed for high-density lipoprotein cholesterol,

total cholesterol, and serum glucose. Diabetes was defined as a fasting glucose level ≥ 126 mg/dL (or a non-fasting glucose ≥ 200 mg/dL among those failing to fast) or by self-reported diabetes medication use. The current use of aspirin, antihypertensive medications, and lipid-lowering therapies was self-reported. After the participant rested for 5 minutes in a seated position, blood pressure was measured using a sphygmomanometer. Two values were obtained following a standardized protocol and averaged. Body mass index was computed as the weight in kilograms divided by the square of the height in meters. Using baseline electrocardiogram data, left ventricular hypertrophy was defined by the Sokolow-Lyon Criteria (13). Coronary heart disease was ascertained by self-reported history of myocardial infarction, coronary artery bypass grafting, coronary angioplasty or stenting, or if evidence of prior myocardial infarction was present on the baseline electrocardiogram. Prior stroke was ascertained by participant self-reported history. Cardiovascular disease was defined as the composite of coronary heart disease and stroke.

Statistical Analyses

Baseline characteristics were examined by the presence of PACs. Categorical variables were reported as frequency and percentage while continuous variables were reported as mean $\pm$ standard deviation. Statistical significance for categorical variables was tested using the chi-square method and the student's t-test procedure for continuous variables. Logistic regression was used to compute odds ratios (OR) and 95% confidence intervals (CI) for the associations of short- and long-term PM_{2.5} exposure with PACs, separately. The association was examined per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{2.5} concentration. Multivariable models were adjusted as follows: Model 1 adjusted for age, sex, race, education, income, geographic region, and average temperature (2-week average for short-term and 1-year average for long-term); Model 2 included Model 1 covariates, with the addition of known PAC risk factors (current smoking, height, physical inactivity, high-density lipoprotein cholesterol, and cardiovascular disease) (14). Additionally, subgroup analyses were performed to evaluate effect modification by age (<65 years vs. ≥ 65 years), sex, and race (black vs. white) using a stratification technique and comparing interaction terms in Model 2. A sensitivity analysis also was performed in which the association between short-term PM_{2.5} exposure and PACs was examined using the average 24-hour PM_{2.5} concentration prior to the baseline visit. Statistical significance for all comparisons including interactions was defined as $p < 0.05$. SAS[®] Version 9.4 (Cary, NC) was used for all analyses.

RESULTS

A total of 26,609 participants (mean age = 65 ± 9.4 years; 55% female; 41% black) were included in this analysis. A total of 2,140 (8.2%) had evidence of PACs on the baseline electrocardiogram. A higher 1-year average PM_{2.5} concentration was observed for those with PACs compared with those without PACs. No difference was observed between the mean 2-week PM_{2.5} concentration for those with and without PACs (Table 1). Baseline characteristics stratified by the presence of PACs are shown in Table 1.

Short-term PM_{2.5} (per 10 $\mu\text{g}/\text{m}^3$) exposure was not associated with PACs (Table 2). The results were similar when we used the mean 24-hour PM_{2.5} concentration to define short-

term exposure (OR=1.04, 95%CI=0.98, 1.11). In contrast, increases in long-term PM_{2.5} concentration (per 10 µg/m³) were associated with PACs (Table 3). No interactions were detected in the short- (Table 2) and long-term (Table 3) analyses when stratified by age, sex, or race.

DISCUSSION

In this analysis from REGARDS, a large-scale epidemiologic study, short-term PM_{2.5} exposure was not associated with PACs. In contrast, we observed a significant association of long-term PM_{2.5} exposure with PACs. The data presented demonstrate that the supraventricular arrhythmogenesis associated with PM_{2.5} concentration likely depends on the duration of PM_{2.5} exposure.

Several reports have suggested that air pollution triggers the development of atrial arrhythmias. A study of 203 patients with ICDs in Boston, Massachusetts has shown a positive association between episodes of paroxysmal atrial fibrillation and increases in ozone concentration in the hour before the development of the arrhythmia (4). Similarly, an examination of 176 patients with ICDs in Boston, Massachusetts reported that the odds of atrial fibrillation increased by 26% for each 6.0 µg/m³ increase in PM_{2.5} in the 2 hours prior to the event (5). Other reports have linked short-term PM_{2.5} exposure with supraventricular ectopy. An examination of 57 males with coronary heart disease has shown that PM exposure (over 5 days) is associated with runs of supraventricular tachycardia (defined as at least 3 consecutive beats) and a study of 32 non-smoking adults in Steubenville, Ohio observed a similar association with increases in PM_{2.5} exposure over a 10-day period (3, 6). Additionally, a small study of 9 highway patrolmen in North Carolina found an association between 4-day PM_{2.5} exposure and supraventricular ectopic beats (7). In contrast, our data from a large population-based cohort do not support an association between short-term PM_{2.5} exposure and PACs. This is consistent with data from the Women's Health Initiative and the Air Pollution and Cardiac Risk and Its Time Course (APACR) study, as these studies did not show an association between short-term PM_{2.5} exposure and supraventricular ectopy (15, 16).

Possible explanations for the differences observed with prior reports and the current study include the populations examined, the definition of short-term PM_{2.5} exposure, and the methods of short-term PM_{2.5} detection. The current study included nearly 26,000 black and white participants and was not limited to individuals with heart disease who had ICDs, a group with an inherent risk for arrhythmia. Short-term exposure was defined using 2-week average PM_{2.5} concentrations and a sensitivity analysis was performed using the average 24-hour PM_{2.5} concentration. It is possible that the relationship between PACs and PM_{2.5} varies with different durations to define short-term exposure (e.g., hourly). Similar variation also is possible with long-term PM_{2.5} exposure (e.g., 3 and 6 months). Furthermore, PM_{2.5} estimates were spatially linked at the geographic coordinates of each participant's residence rather than relying on local monitoring stations.

To our knowledge, there are no studies that have attempted to link long-term PM_{2.5} exposure with PACs. Possibly, atrial cardiomyocytes require a longer duration of PM_{2.5} exposure

before ectopic beats are apparent, as we did not observe an increased risk of PACs with short-term exposure. This would suggest that significant atrial remodeling likely occurs with PM_{2.5} exposure and a threshold of cumulative PM_{2.5} exists for which the risk of PACs is highest. Additionally, it is possible that long-term PM_{2.5} exposure decreases the threshold for PACs by altering the autonomic tone of atrial cardiomyocytes. Both mechanisms possibly lead to an increased risk for non-sustained supraventricular arrhythmias (17). In aggregate, the data presented in this study suggest a potential role for long-term PM_{2.5} exposure in initiating supraventricular arrhythmias that are triggered by PACs.

Several reports have linked PM exposure with adverse cardiovascular events, implicating air pollution as a serious public health problem (18–25). The data provided in this analysis also implicate prolonged exposure to elevated levels of PM_{2.5} as a risk factor for supraventricular ectopic beats. Therefore, it is possible that air pollution is a modifiable risk factor for arrhythmia development and regulations aimed to reduce the level of ambient PM_{2.5} concentration should be considered. As we strive for more cost-effective options within our health care system, reductions in air pollution possibly have a role to reduce the burden of atrial arrhythmias, including cost.

The results of the current analysis should be interpreted in the context of certain limitations. Several baseline characteristics (e.g., current smoking) were self-reported and subjected our analysis to recall bias. PACs were detected from a single electrocardiogram recording and participants possibly will be reclassified with subsequent tracings. The PM_{2.5} exposure was presumed to be constant during the time periods examined. However, we acknowledge that temporal variation in PM_{2.5} concentration was unable to be accounted for as our estimates were measured by satellite. Additionally, we linked participants at the level of their home addresses and assumed that each participant spent enough time outdoors to expose them to the level of PM_{2.5} measured. PM_{2.5} exposure also was estimated on a 10-km grid using EPA ground observation data and MODIS data, and this spatial resolution possibly resulted in inaccurate PM_{2.5} estimates. Furthermore, we were unable to determine the components comprising the PM_{2.5} exposure and cannot differentiate which PM contributes to our results. Humidity, barometric pressure, and time of day were not collected in our cohort and we were unable to incorporate these covariates into our multivariable models. We also included several risk factors associated with PACs in our multivariable models but cannot exclude the possibility of residual confounding similar to other epidemiologic studies.

In conclusion, the findings of this study suggest that increased levels of long- but not short-term PM_{2.5} exposure are associated with PACs. These data implicate a role for long-term PM_{2.5} exposure in initiating supraventricular arrhythmias that are triggered by PACs. Further research is needed to confirm our findings and to explore the utility of air pollution reduction to decrease the incidence of supraventricular arrhythmias.

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Table 1

Baseline Characteristics (N=26,069)

Characteristic	PACs (n=2,140)	No PACs (n=23,929)	P-value*
Age, mean (SD), years	69 (9.6)	64 (9.2)	<0.0001
Male (%)	1,141 (53)	10,671 (45)	<0.0001
Black (%)	972 (45)	9,769 (41)	<0.0001
Region			
Stroke belt (%)	687 (32)	8,370 (35)	
Stroke buckle (%)	450 (21)	4,975 (21)	
Non-belt (%)	1,003 (47)	10,584 (44)	0.020
Education, high school or less (%)	900 (42)	8,982 (38)	<0.0001
Annual income			
Refused (%)	293 (14)	2,867 (12)	
<\$20,000 (%)	439 (21)	4,082 (17)	
\$20,000–\$34,999 (%)	585 (27)	5,679 (24)	
\$35,000–\$74,999 (%)	559 (26)	7,298 (31)	
>\$75,000 (%)	264 (12)	4,003 (17)	<0.0001
No exercise (%)	712 (33)	7,838 (33)	0.63
Current smoker (%)	287 (13)	3,519 (15)	0.10
Diabetes (%)	527 (25)	4,828 (20)	<0.0001
Systolic blood pressure, mean (SD), mm Hg	130 (18)	127 (16)	<0.0001
Height, mean (SD), m	1.71 (0.10)	1.69 (0.10)	<0.0001
Body mass index, mean (SD), kg/m ²	29 (6.4)	29 (6.1)	0.092
Total cholesterol, mean (SD), mg/dL	188 (39)	193 (40)	<0.0001
HDL cholesterol, mean (SD), mg/dL	52 (16)	52 (16)	0.31
Aspirin (%)	998 (47)	10,124 (42)	0.0001
Antihypertensive medications (%)	1,203 (56)	11,877 (50)	<0.0001
Lipid-lowering medications (%)	702 (33)	7,738 (32)	0.66
Left ventricular hypertrophy (%)	250 (12)	2,258 (9.4)	0.0007
Cardiovascular disease (%)	540 (25)	4,629 (19)	<0.0001
2-week PM _{2.5} , µg/m ³			
Mean (SD)	13.4 (4.0)	13.4 (4.1)	0.82
Median	12.8	12.8	
2-week Temperature, C			
Mean (SD)	16.5 (8.8)	17.1 (8.5)	0.0017
Median	17.7	18.3	
1-year PM _{2.5} , µg/m ³			
Mean (SD)	13.5 (1.9)	13.4 (1.9)	0.017
Median	13.7	13.5	
1-year Temperature, C			
Mean (SD)	15.8 (4.0)	15.9 (4.0)	0.090
Median	16.5	16.6	

* Statistical significance for categorical variables tested using the chi-square method and for continuous variables the student's t-test was used.

HDL=high-density lipoprotein; PAC=premature atrial contraction; PM=particulate matter; SD=standard deviation

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Table 2Association of Short-Term PM_{2.5} Exposure with and PACs^{*}

	Model 1 [†] OR (95%CI)	P-value	Model 2 [‡] OR (95%CI)	P-value	P-interaction ^δ
No PAC	Ref	-	Ref	-	
PAC	1.10 (0.98, 1.23)	0.11	1.09 (0.98, 1.23)	0.13	
<i>Age</i>					
<65	1.09 (0.90, 1.32)	0.39	1.08 (0.89, 1.32)	0.42	0.62
65	1.09 (0.95, 1.26)	0.23	1.09 (0.94, 1.25)	0.25	
<i>Sex</i>					
Female	1.05 (0.89, 1.24)	0.54	1.05 (0.89, 1.24)	0.57	0.80
Male	1.13 (0.97, 1.33)	0.13	1.13 (0.96, 1.33)	0.14	
<i>Race</i>					
Black	1.05 (0.88, 1.25)	0.62	1.04 (0.88, 1.24)	0.64	0.71
White	1.14 (0.98, 1.33)	0.091	1.14 (0.98, 1.32)	0.10	

^{*} OR presented per 10 µg/m³ increase in PM_{2.5} exposure.[†] Model 1 adjusted for age, sex, race, education, income, geographic region, and average temperature.[‡] Model 2 adjusted for covariates in Model 1 with the addition of current smoking, height, physical inactivity, high-density lipoprotein cholesterol, and cardiovascular disease.^δ Interactions tested using Model 2.

CI=confidence interval; OR=odds ratio; PAC=premature atrial contraction; PM=particulate matter.

Table 3Association of Long-Term PM_{2.5} Exposure with PACs^{*}

	Model 1 [†] OR (95%CI)	P-value	Model 2 [‡] OR (95%CI)	P-value	P-Interaction ^δ
No PAC	Ref	-	Ref	-	
PAC	1.41 (1.11, 1.80)	0.0050	1.40 (1.10, 1.78)	0.0069	
Age					
<65	1.42 (0.92, 2.19)	0.11	1.39 (0.90, 2.15)	0.13	0.88
65	1.38 (1.03, 1.84)	0.032	1.37 (1.02, 1.83)	0.037	
Sex					
Female	1.32 (0.93, 1.88)	0.12	1.31 (0.93, 1.87)	0.13	0.50
Male	1.49 (1.07, 2.08)	0.020	1.47 (1.05, 2.05)	0.025	
Race					
Black	1.34 (0.93, 1.94)	0.12	1.34 (0.93, 1.93)	0.12	0.21
White	1.58 (1.14, 2.19)	0.0061	1.57 (1.13, 2.18)	0.0071	

^{*} OR presented per 10 µg/m³ increase in PM_{2.5} exposure.[†] Model 1 adjusted for age, sex, race, education, income, geographic region, and average temperature.[‡] Model 2 adjusted for covariates in Model 1 with the addition of current smoking, height, physical inactivity, high-density lipoprotein cholesterol, and cardiovascular disease.^δ Interactions tested using Model 2.

CI=confidence interval; OR=odds ratio; PAC=premature atrial contraction; PM=particulate matter.