Fine Particulate Matter (PM$_{2.5}$) and the Risk of Stroke in the REGARDS Cohort

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Abstract

Background & Purpose—Ambient particulate matter has been shown to be associated with declining human health, although the association between fine particulate matter (PM$_{2.5}$) and stroke is uncertain.

Methods—We utilized satellite-derived measures of PM$_{2.5}$ to examine the association between exposure and stroke in the REasons for Geographic And Racial Differences in Stroke (REGARDS) study. We used a time-stratified case cross-over design, with exposure lags of 1-day, 2-days and 3-days. We examined all strokes, as well as ischemic and hemorrhagic separately.

Results—Among 30,239 participants in the REGARDS study, 746 incident events were observed: 72 hemorrhagic, 617 ischemic, and 57 of unknown type. Participants exposed to higher levels of PM2.5 more often resided in urban areas compared to rural, and in the southeastern US. After adjustment for temperature and relative humidity, no association was observed between PM2.5 exposure and stroke, regardless of the lag (1-day lag OR=0.99, 95% CI: 0.83–1.19; 2-day

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lag OR=0.95, 95% CI: 0.80–1.14; 3-day lag OR=0.95, 95% CI=0.79–1.13). Similar results were observed for the stroke subtypes.

Conclusions—In this large cohort of African Americans and whites, no association was observed between $PM_{2.5}$ and stroke. The ability to examine this association with a large number of outcomes and by stroke subtype helps to fill a gap in the literature examining the association between fine particulate matter and stroke.

Keywords

stroke; epidemiology; air pollution; REGARDS

Introduction

Ambient particulate matter has been shown to be associated with declining human health[1–3], and recent work has indicated that fine particulate matter, characterized as particles less than 2.5 μm in diameter ($PM_{2.5}$), may impact cardiovascular [4, 5]. In fact, a recent AHA Scientific health Statement has suggested that the relationship between $PM_{2.5}$ and cardiovascular morbidity and mortality is causal[6]. The proposed mechanisms through which $PM_{2.5}$ exposure is thought to impact cardiovascular and cerebrovascular health include systematic inflammation mediated by oxidative stress and endothelial dysfunction[7, 8], which would suggest that the impact of $PM_{2.5}$ on stroke would be cumulative. However, direct effects can occur through increases in heart rate and blood pressure[9, 10], arguing for the plausibility of shorter term associations. Several studies have examined both long- and short-term exposure to $PM_{2.5}$ and risk of stroke, and results have been conflicting.[11, 12] Most of the studies have examined small samples and have used sparse ground-level measures for exposure estimates, or have meta-analyzed several studies with varying approaches to measure exposure.

One promising method for characterizing $PM_{2.5}$ exposure for public health practice and epidemiologic research is integration of remote sensing satellite systems data with air monitoring network data.[13–18] Remotely-sensed data have been used to detect and track particulate matter plumes from major events such as dust storms, volcanic emissions, and fires. The aerosol optical properties measured by space-borne sensors can also be useful in filling the temporal and spatial gaps found with ground-level monitor data. Satellite data cover large geographic areas at moderate spatial resolution for multiple years and with reliable repeated measurements. Research has shown that aerosol optical depth (AOD) is indirectly related to ground-level $PM_{2.5}$, with the correlation between the two being strongest on days with low cloud cover, low relative humidity, and good vertical mixing within the atmospheric column. [14, 19–23]

In this paper, we used measures of $PM_{2.5}$ that have been estimated using a national version of the algorithm of Al-Hamdan et al. (2009), which combines data from remotely-sensed satellite data and EPA ground-level data to determine the impact of $PM_{2.5}$ exposure on stroke in participants from the REasons for Geographic and Racial Differences in Stroke (REGARDS) study, using a time-stratified case-crossover design.[24, 25] We hypothesized that among REGARDS participants who have suffered a stroke, the level of $PM_{2.5}$ exposure will be higher in the 24-hour period prior to their stroke, than on other similar days. We further

hypothesized that these results will be similar among REGARDS participants suffering from either ischemic or hemorrhagic strokes.

Methods

Study procedures

This analysis used a subset of the REGARDS cohort who have experienced an incident stroke. REGARDS is a prospective cohort study of black and white participants of both sexes, in order to study geographic and racial differences in stroke incidence and mortality, the details of which can be found elsewhere.[26] In brief, participants were recruited by mail from across the continental US, with oversampling in the Stroke Belt (North Carolina, South Carolina, Georgia, Tennessee, Alabama, Mississippi, Louisiana, and Arkansas) and Stroke Buckle (coastal plains of North Carolina, South Carolina, and Georgia), as well as oversampling of black residents. At baseline (between 2003–207), participants completed a computer-assisted telephone interview (CATI), followed by an in-home visit by a trained health professional, during which time blood and urine samples were collected, as well as several anthropomorphic traits (e.g., height and weight). Blood pressure was measured during the in-home visit by taking the average of two blood pressure measurements after the participant had been seated for five minutes. Participants were followed by CATI every six months to ascertain suspected stroke events. All participants provided written informed consent, and IRB approval was obtained by all participating institutions.

Individual variables

Age (years), race (black or white), gender, income, and education were all self-reported during the CATI. Region and urbanicity were assigned using the participant's geocoded residential address at baseline. Total cholesterol (mg/dL) was measured from a blood sample collected during the in-home visit. Participants were determined to have hypertension if their systolic blood pressure was 140 mmHg, diastolic blood pressure was 90 mmHg, or they self-reported taking medication to control blood pressure. Participants were determined to have dyslipidemia if their total cholesterol was 240 mg/dL, their low density lipoprotein cholesterol (LDLC) was 160 mg/dL, their high density lipoprotein cholesterol (HDLC) was 40 mg/dL, or they self-reported taking lipid-lowering medications. Participants were determined to have diabetes if their fasting glucose was 126 mg/dL, their non-fasting glucose was 240 mg/dL, or they self-reported using diabetes medication or insulin. Smoking status (current, past, never) was self-reported.

Environmental variables

Ambient $PM_{2.5}$ concentration was the exposure of interest. Previous studies have used Environmental Protection Agency (EPA) ground-level monitoring stations to estimate the concentrations of $PM_{2.5}$ for participants.[27] In order for measurements from these monitoring stations to accurately reflect the exposure of the participants, the study participants are usually restricted to those living in metropolitan statistical areas (MSAs). Since approximately 20% of REGARDS participants do not live in urban areas, and excluding them might have biased our sample, we used an estimated $PM_{2.5}$ concentration that integrated ground-level measurements and satellite measurements of the total amount of

aerosols within the air column, called aerosol optical depth (AOD), from the Moderate Resolution Imaging Spectroradiometer (MODIS) instrument on the NASA Aqua satellite.[13, 14] Briefly, regression equations for each combination of EPA region of the US and season were used to estimate ground-level $PM_{2.5}$ from AOD measurements,[28] then these measurements were integrated with ground-level monitoring station measurements and spatially smoothed using a b-spline to a 10 km × 10 km resolution across the continental US. Daily estimates of $PM_{2.5}$ concentration were created for REGARDS participants from 2003 to 2011, which was based upon the 10 km by 10 km grid cell of the $PM_{2.5}$ surfaces containing the participant's geocoded residential address.

Ambient temperature and relative humidity were included as potential confounders of the relationship between $PM_{2.5}$ and incident stroke. Mean daily temperatures were computed from the North American Land Data Assimilation System (NLDAS) hourly air temperature dataset on a 12 km × 12 km grid across the continental US[13, 29]. Relative humidity was derived according to Iribarne and Godson (1981)[30] using NLDAS air temperature, atmospheric pressure and specific humidity on the NLDAS grid. Mean daily temperatures and relative humidity were assigned for REGARDS participants based upon which 12 km by 12 km NLDAS grid cell contained the participant's residence location.

Stroke Ascertainment

Potential events were ascertained during the follow-up calls, and those reported as possible stroke, transient ischemic attack (TIA), death, hospitalization or emergency department visit for brain aneurysm, brain hemorrhage, stroke symptoms, or unknown reason prompted a request for medical records. Initial review by a stroke nurse excluded obvious non-strokes; the remaining medical records were centrally adjudicated by physicians. Death certificates were examined and adjudicated for deaths without a medical record, and proxy interviews undertaken to ascertain potential events. Stroke events were defined following the World Health Organization (WHO) definition.[31] Those events not meeting the WHO definition but with symptoms lasting >24 hours and neuroimaging consistent with acute ischemia or hemorrhage were classified as clinical strokes, and were included in analyses.

Statistical analysis

The analysis used a case-crossover design, where the $PM_{2.5}$ exposure 1, 2, or 3 days previous to the stroke was compared with the exposure on three other days in the same month as the stroke. We chose the three other days to be the same days of the week as the lagged day before the stroke.[24] Conditional logistic regression models were used to determine whether short-term $PM_{2.5}$ exposure was associated with stroke. Thus, each participant served as their own control, removing the need to control for potential confounders that did not vary during the month, such as age, race, or sex. $PM_{2.5}$ exposure was dichotomized as "good" (12 μg/m^3) or "moderate/unhealthy" (12 – 150.4 μg/m^3), which reflects 2012 EPA guidelines for 24-hour exposure.[32] We included mean temperature and relative humidity as potential confounders, since these covariates did vary within a month. We also investigated potential interactions between category of $PM_{2.5}$ concentration and urbanicity (rural, mixed, or urban) and between category of $PM_{2.5}$ concentration and region (Stroke Belt, Stroke Buckle, or rest of US). Sensitivity analyses included using $PM_{2.5}$

concentration as a continuous variable, as well as stratifying by stroke subtype (ischemic or hemorrhagic).

Results

Of the 30,239 participants in the REGARDS sample, 1,101 had strokes between January 1, 2003 and December 31, 2011. Of these participants, 227 were excluded due to self-reported history of stroke at baseline, 128 were excluded due to low confidence in their geocoded residential location, leaving a final sample of 746 participants with incident stroke for analysis (72 hemorrhagic, 617 ischemic, and 57 of unknown type). Among the 746 incident strokes, 403 (54%) occurred following a day classified as "good", whereas the remaining 343 (46%) occurred following a day classified as "moderate" or "unhealthy".

Table 1 presents the demographic characteristics of the cohort (n=746) by level of $PM_{2.5}$ concentration on the day prior to the event, as well as data on co-morbidities. Few differences in demographics exist, although participants who live in the stroke belt or buckle and participants who live in urban areas are more often exposed to moderate levels of $PM_{2.5}$, compared to those who live in the remainder of the US. Further, those exposed to higher $PM_{2.5}$ levels more frequently have hypertension and are slightly more frequently current smokers.

The odds ratios and 95% confidence intervals for moderate compared to good $PM_{2.5}$ exposure for lags of 1, 2, and 3 days for the entire cohort, as well as the stratified by stroke subtype, are presented in Table 2 and Figure 1. These odds ratios were adjusted for mean temperature and relative humidity. $PM_{2.5}$ exposure was not associated with incident stroke or ischemic stroke, regardless of lag. We did not observe a significant association between $PM_{2.5}$ exposure and stroke, regardless of lag and regardless of stroke subtype. A sensitivity analysis using $PM_{2.5}$ concentration as a continuous variable did not change these inferences. Interaction terms for a 1-day lag between urbanicity and $PM_{2.5}$, and region and $PM_{2.5}$ were not significant (p = 0.70 and p = 0.14, respectively). These results indicate that $PM_{2.5}$ concentration is not significantly associated with incident stroke in the REGARDS cohort.

Discussion

Evidence regarding the association between $PM_{2.5}$ and stroke is mixed, and our conclusions did not support an association between short-term $PM_{2.5}$ exposure and stroke. We were able to examine this association in a large, geographically and racially diverse sample of stroke, using a novel measure of $PM_{2.5}$ exposure, and accounting for temperature and humidity. This research directly addressed gaps in the research calling for large studies that specifically examined ischemic and hemorrhagic stroke as well as used medical-record review for event ascertainment.[33] We have provided strong evidence of a lack of association.

Recent reports describing relationships between longer-term $PM_{2.5}$ exposure and stroke have drawn mixed conclusions. With respect to long-term exposures, a meta-analysis of 11 European Cohorts found that the risk of stroke increased by 19% for each 5 μg/m^3 increase in mean annual $PM_{2.5}$, although these results were not statistically significant.[12] A global

meta-analysis including 20 studies found long-term $PM_{2.5}$ exposure to be associated with stroke, but that there was geographic variability in the association.[11]

Short-term exposure data have also been conflicting. Lisabeth et al. (2008) showed marginally significant associations between same-day $PM_{2.5}$ and stroke in a community in Texas,[34] while Wellenius et al. (2012) showed that the likelihood of stroke following a 24-hour period of moderate $PM_{2.5}$ exposure ($PM_{2.5}$ 15–40 μg/m^3), was 34% higher than for periods classified as good ($PM_{2.5}$ 15 μg/m^3).[27] This research has indicated that $PM_{2.5}$ exposure within ranges classified as "good" by the EPA still result in increased risk of stroke among stroke patients in the Boston area. Conversely, a recent meta-analysis of short-term changes in $PM_{2.5}$ exposure and risk of hospital admissions for ischemic stroke including six studies found no significant association.[33] However, these studies were based on EPA ground-level data, and many were limited to single communities. A larger study in Israel, also using satellite-derived measures of exposure, found no overall association between ischemic stroke and same-day $PM_{2.5}$ exposure, but did find marginally significant association among those <55 years of age in stratified models.[35]

While our study has addressed gaps in the literature by including a large, geographically diverse US population, and with our ability to examine ischemic and hemorrhagic strokes separately, our research does suffer from some limitations. First, we used ambient $PM_{2.5}$ exposure at the participants' residences at the time of their stroke, and thus we may have misclassified exposure as the participant may have lived elsewhere during this time. Further, research considering $PM_{2.5}$ exposure was limited by the inability to measure indoor $PM_{2.5}$ exposure, and all $PM_{2.5}$ research to date assumes that outdoor levels are reasonable surrogates for total exposure levels. Additionally, we did not have data about the components of the $PM_{2.5}$ (the speciation of the fine particulate matter), and thus could not determine what comprised the $PM_{2.5}$ exposure. It is known that speciation varies regionally, and thus we could have increased variability in our exposure measure by calling it all $PM_{2.5}$.

Conclusions

In a racially and geographically diverse cohort of participants in the REGARDS study, including ischemic and hemorrhagic stroke events, we were unable to identify an association between short-term exposure to $PM_{2.5}$ and stroke. Future work should consider other environmental pollutants, as well as particular components of $PM_{2.5}$, and focus on longer term exposure.

Acknowledgments

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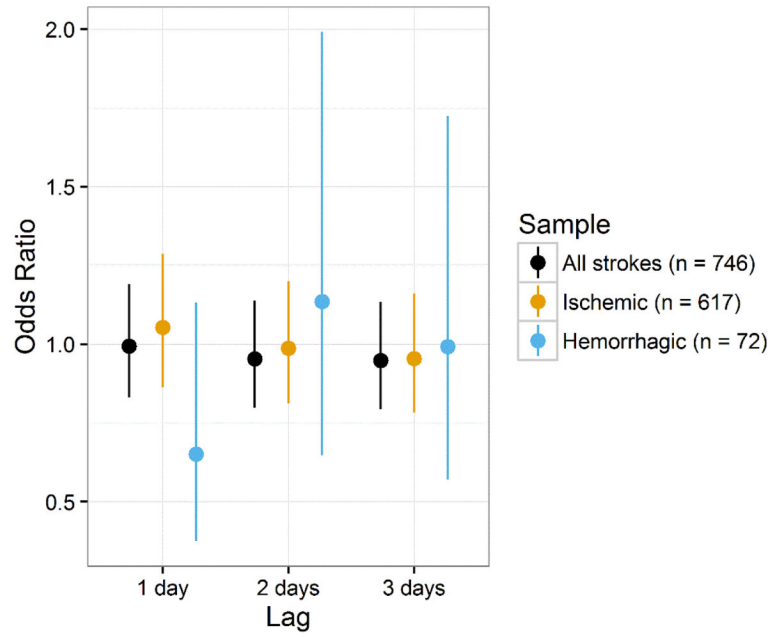
Figure 1.
Odds ratios and 95% CIs for likelihood of stroke for moderate/unhealthy to good $PM_{2.5}$ exposure with 1- to 3-day lag, for all strokes and by stroke subtype

Table 1

Demographics and comorbidities of cohort, by category of PM$_{2.5}$ exposure on day before stroke. Values are n (%) unless otherwise noted.

	PM$_{2.5}$ concentration on day before stroke	
Variable	**Good (0 – 12 μg/m³) (n=403)**	**Moderate or unhealthy (12.1 – 150.4 μg/m³) (n=343)**
Age *, years	70(9)	70(9)
Black race	186(46)	155(45)
Female	206(51)	159(46)
Income		
< $20,000	98(24)	66(19)
$20,000 – $34,000	128(32)	105(31)
$35,000 – $74,000	98(24)	93(27)
$75,000 and above	31(8)	31(9)
Refused	48(12)	48(14)
Education		
Less than high school	68(17)	58(17)
High school graduate	130(32)	98(29)
Some college	85(21)	88(26)
College graduate and above	120(30)	98(29)
Region		
Stroke Belt	134(33)	126(37)
Stroke Buckle	67(17)	81(24)
Rest of US	202(50)	136(40)
Total cholesterol *, mg/dL	191(43)	189(41)
Hypertension	289(72)	248(72)
Dyslipidemia	245(61)	217(63)
Diabetes	107(27)	98(29)
Smoking status at baseline		
Current	71(18)	63(18)
Past	160(40)	148(43)
Never	171(42)	128(37)
Mean temperature *, Celsius	16(9)	19(9)
Relative humidity *, %	59(19)	55(17)

*
mean(standard deviation)

Table 2

Model results for likelihood of stroke for moderate/unhealthy to good $PM_{2.5}$ exposure with 1- to 3-day lag, for all strokes and by stroke subtype.

Outcome	Lag	Odds Ratio (95% CI)
All Strokes	**1 day**	0.99 (0.83, 1.19)
	2 day	0.95 (0.80, 1.14)
	3 day	0.95 (0.79, 1.13)
Ischemic Stroke	**1 day**	1.05 (0.86, 1.29)
	2 day	0.99 (0.81, 1.20)
	3 day	0.95 (0.78, 1.16)
Hemorrhagic Stroke	**1 day**	0.65 (0.37, 1.13)
	2 day	1.14 (0.65, 1.99)
	3 day	0.99 (0.57, 1.73)