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Original Contribution

Fine Particulate Matter Air Pollution and Cognitive Function Among Older US Adults

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Existing research on the adverse health effects of exposure to pollution has devoted relatively little attention to the potential impact of ambient air pollution on cognitive function in older adults. We examined the cross-sectional association between residential concentrations of particulate matter with aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$) and cognitive function in older adults. Using hierarchical linear modeling, we analyzed data from the 2004 Health and Retirement Study, a large, nationally representative sample of US adults aged 50 years or older. We linked participant data with 2000 US Census tract data and 2004 census tract–level annual average $PM_{2.5}$ concentrations. Older adults living in areas with higher $PM_{2.5}$ concentrations had worse cognitive function ($\beta = -0.26$, 95% confidence interval: −0.47, −0.05) even after adjustment for community- and individual-level social and economic characteristics. Results suggest that the association is strongest for the episodic memory component of cognitive function. This study adds to a growing body of research highlighting the importance of air pollution to cognitive function in older adults. Improving air quality in large metropolitan areas, where much of the aging US population resides, may be an important mechanism for reducing age-related cognitive decline.

air pollution; cognitive function; communities; health

Abbreviations: CI, confidence interval; EPA, Environmental Protection Agency; HRS, Health and Retirement Study; PM, particulate matter; $PM_{2.5}$, particulate matter with aerodynamic diameter of 2.5 μm or less.

Research suggests that exposure to ambient air pollution is associated with a number of adverse health effects. Small-particle air pollution (<10 μm in diameter) is especially harmful to health because, once inhaled, particles can travel through the circulatory system and ultimately cause damage to organs, such as the heart, lungs, and brain (1). Chronic exposure to particulate matter (PM) air pollution has been linked to elevated risks of cardiovascular diseases, respiratory diseases, and death (2–4). Emerging research suggests that exposure to air pollution may also be harmful to brain health and functioning (5). Studies of animals and humans have found an association between exposure to high concentrations of PM air pollution and both increased brain inflammation and accumulation of β-amyloid, a marker of neuronal dysfunction (6–8). Individuals experiencing air pollutant–induced inflammation and neurodegeneration may be more likely to develop cognitive deficits (9) and may have greater risk for experiencing progressive neurodegenerative diseases such as Alzheimer's disease.

Older adults are especially susceptible to the adverse cardiovascular and respiratory outcomes associated with particulate air pollution (10, 11). However, relatively little is known about the relationship between PM exposure and cognitive function in older adults. Prior studies have found that older adults living in close proximity to a major road, a measure of exposure to traffic-related PM, have worse cognitive function (12, 13). Studies of particulate matter with an aerodynamic diameter of 10 μm or less reported no association with cognitive function (12, 14). However, in a study that examined the coarse (aerodynamic diameter 2.5–10 μm) and fine (aerodynamic diameter ≤2.5 μm) components of PM separately, higher levels of residential exposure to both components were associated with worse cognitive decline (15). Furthermore, exposure to higher concentrations of black

carbon, a component of $PM_{2.5}$, has been linked to worse cognitive function in older adults (13, 16).

There is increasing evidence that particulate air pollution, such as $PM_{2.5}$, may play a significant role in neurodegeneration. To our knowledge, there is no existing research on the association between $PM_{2.5}$ exposure and cognitive function in a national sample of older US men and women. In this paper, we use a nationally representative sample of community-dwelling older adults to explore the hypothesis that living in areas with higher concentrations of $PM_{2.5}$ is associated with worse cognitive function, even after adjustment for individual-level risk factors and community-level social and economic characteristics.

METHODS

Data

We used data from the Health and Retirement Study (HRS), a longitudinal and nationally representative study of US adults aged 50 years or older. The study sample was obtained using a stratified, multistage area probability sample design with oversampling of African Americans, Hispanics, and Floridians. Respondent information was obtained from the RAND HRS data file, version L, which is produced by the RAND Center for the Study of Aging (Santa Monica, California), with funding from the National Institute on Aging (Baltimore, Maryland) and the Social Security Administration (Woodlawn, Maryland). We used data from the 2004 HRS survey, which included 18,575 community-dwelling adults aged 50 years or older. We excluded 1,441 respondents with a history of stroke, as well as 86 respondents who were missing data on individual-level covariates. Respondents who lived in census tracts that were not within 60 km of an air monitoring station ($n = 1,718$) or who could not be geocoded to a census tract ($n = 297$) were not included in the analysis, because their exposures to $PM_{2.5}$ could not be ascertained. In addition, 1,222 respondents who had a proxy complete the interview, and thus whose cognitive function was not directly assessed, were excluded from the analytical sample. Implications of missing $PM_{2.5}$ and cognitive data are discussed in more detail in the results section. The final analytical sample consisted of 13,996 men and women aged 50 years or older.

Air pollution data came from the US Environmental Protection Agency's (EPA's) Air Quality System, a national network of air monitoring stations that collects data on air pollutants. Data on census tract–level air pollution for 2004 were obtained from RAND's Center for Population Health and Health Disparities Data Core Series (17). Measures of census tract–level social and economic characteristics are from the 2000 census. Pollution and census data were linked to HRS respondents using census tract identifiers.

Measures

Cognitive function. The HRS measure of cognitive function is based on the Telephone Interview for Cognitive Status (18) and has been validated for use in population surveys conducted in person and by telephone (19). Approximately 72% of the sample received the cognitive assessment in face-to-face interviews, and the remainder completed the assessment by telephone. Cognitive function was assessed on a 35-point scale that sums scores on immediate and delayed 10-noun free-recall tests to measure memory, a serial 7 subtraction test to measure working memory, a backwards counting test to measure processing speed, recall of the date and name of the president and vice president to measure orientation, and an object naming test to measure knowledge and language. Participants under age 65 years were not assessed on the orientation and object naming tests because the "young old" generally answer all items correctly. To construct a total cognitive function score for all respondents, we assigned full points on these tests to those younger than 65 years.

We also created measures for 2 separate components of cognitive function measured in the HRS: episodic memory and mental status. The numbers of correctly recalled words in the immediate and delayed recall tests were summed to create a total episodic memory score that ranges from 0 to 20. Correct answers on the remaining tests were summed to create a mental status score that ranges from 0 to 15. Previous analysis of cognition in the HRS suggests that episodic memory and mental status represent 2 distinct factors and should be investigated separately (19).

Air pollution. Census tract–specific concentrations of $PM_{2.5}$ in 2004 were derived using 24-hour daily means reported by monitors within a 60-km radius of each census tract centroid. Values from each monitoring station were weighted using the inverse of the distance from the census tract centroid to the location of the monitor to give greater importance to values reported by monitors closer to the respondents' census tracts. The daily means were then aggregated to create the tract-level 2004 annual average $PM_{2.5}$ concentration.

Covariates. Individual-level covariates include sociodemographic and socioeconomic characteristics, as well as smoking status. Sociodemographic information included age, sex, and race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, other). Socioeconomic measures included educational attainment, which was assessed as the highest completed grade of school or year of college; current employment status; and logged household income and wealth. Smoking behavior was included (never smoker, former smoker, current smoker). Census tract–level covariates include the proportion of residents aged 25 years or older without a high school degree and median household income (logged).

Analytical strategy

We used multilevel linear regression models to examine the association between $PM_{2.5}$ and cognitive function. On average, there were 3 sample members living in a tract (13,996 respondents living in 4,577 tracts), with only 13% of sample members living in a singleton tract (i.e., a tract with no other sample members). Thus, most sample members were clustered in tracts and, therefore, had similar ambient air pollution exposures. We used multilevel models to account for the clustering of respondents within census tracts (20). For

Table 1. Characteristics of 13,996 Adults Aged 50 Years or Older in the US Health and Retirement Study, 2004

Individual-Level Variable	Mean (SD)	Range	%
Age, years	64.0 (10.4)	50–102	
Female			56.08
Race/ethnicity			
White			81.09
Black			9.49
Hispanic			6.57
Other			2.86
Education, years	13.0 (3.0)	0–17	
Household income, $US	70,369 (110,733)	0–3,532,388	
Household wealth, $US	401,687 (1,467,617)	−20,000–3,238,335	
Not employed			51.73
Smoking status			
Never			42.01
Former			41.65
Current			16.03
Cognitive function			
Episodic memory score	10.3 (3.4)	0–20	
Mental status score	13.4 (2.0)	0–15	
Total score	23.7 (4.6)	0–35	

Abbreviation: SD, standard deviation.

both the total cognitive function score and the 2 subcomponents, 3 models are presented. Model 1 includes adjustments for tract-level covariates and individual sociodemographic characteristics. Model 2 adds individual socioeconomic status. Model 3 adds smoking status. Analyses were conducted using Stata, version 12, software (StataCorp LP, College Station, Texas) and HLM, version 6.06, software (Scientific Software International, Inc., Skokie, Illinois). In all analyses, we used sample weights that adjust for differential sampling probabilities and nonresponse and make the sample representative of the older US population.

RESULTS

Table 1 shows individual-level characteristics of the sample. The average age of the sample was 64 years. There were more women than men, most were non-Hispanic white, and their average educational attainment was just above high school level. Average income and wealth showed considerable variability in this sample. Just more than half of the sample was not working, and most did not smoke. The average cognitive function score was approximately 24, which is in the normal range.

Because the association between cognitive function and $PM_{2.5}$ appeared to be nonlinear, we divided $PM_{2.5}$ into quartiles. $PM_{2.5}$ ranged from 4.5 to 20.7 $\mu g/m^3$ (mean = 12.0 $\mu g/m^3$) in the tracts in which respondents lived. $PM_{2.5}$ ranged from 4.500 to 9.942 $\mu g/m^3$ in the first and lowest quartile, 9.943 to 12.184 $\mu g/m^3$ in the second quartile, 12.185 to 13.796 $\mu g/m^3$ in the third quartile, and 13.797 to 20.661 $\mu g/m^3$ in the fourth and highest quartile. The national ambient air quality standard, which is determined by the EPA (Washington, DC) to be the level at which there is increased risk to human health, is 12 $\mu g/m^3$ for $PM_{2.5}$. Nearly half of sample members live in areas where $PM_{2.5}$ concentrations exceed the air quality standard.

The distribution of sample characteristics is shown separately for each $PM_{2.5}$ quartile in Table 2. The proportion of residents with less than a high school degree is higher in more polluted areas. However, residents in more polluted areas also have higher average income than those in less polluted areas. Those living in more polluted areas were younger and less likely to be white. Education and income show a curvilinear relationship with $PM_{2.5}$, suggesting that those with the highest levels of education and income lived in both the least and most polluted areas. A higher percentage of those living in the most polluted quartiles were not working and were current smokers. Cognitive function was lower among those living in areas with higher exposure.

Table 3 presents the multilevel regression models of cognitive function. Model 1 shows the association between $PM_{2.5}$ and cognitive function after adjustment for tract-level covariates and individual sociodemographic characteristics. Cognitive function is lower in the top 3 quartiles of exposure compared with the lowest quartile. However, the largest difference is seen between the lowest and third quartiles ($\beta = -0.58$, 95% confidence interval (CI): −0.80, −0.36). Model 2 further adjusts for individual socioeconomic characteristics. The difference between the first and second quartiles of exposure is no longer statistically significant, and the size

Table 2. Weighted Means and Percentages of Covariates by Quartiles of Annual Mean $PM_{2.5}$ Exposure of 13,996 Adults Aged 50 Years or Older in the US Health and Retirement Study, 2004

Covariate	Quartile[a] of Annual Mean $PM_{2.5}$ Exposure							
	First ($n = 3,744$)		Second ($n = 3,598$)		Third ($n = 3,318$)		Fourth ($n = 3,336$)	
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%
Census Tract–Level Variables								
Annual $PM_{2.5}$ exposure in 2004	8.9 (0.8)		11.1 (0.7)		13.0 (0.5)		15.4 (1.6)	
Residents without HS degree		16.9		18.5		19.0		20.1
Median household income, $US	46,180 (18,696)		46,761 (17,189)		50,930 (24,751)		47,312 (21,034)	
Individual-Level Variables								
Age, years	64.8 (10.5)		63.8 (10.4)		63.5 (10.3)		63.9 (10.2)	
Female		55.9		55.0		57.3		56.3
Race/ethnicity								
White		86.0		84.0		77.5		76.1
Black		2.8		8.9		13.4		13.8
Hispanic		8.4		5.3		5.0		7.3
Other		2.8		1.8		4.0		2.9
Education, years	13.1 (2.9)		12.0 (2.9)		12.9 (3.0)		13.0 (3.0)	
Household income, $US	71,564 (125,970)		63,480 (85,159)		71,864 (121,608)		74,566 (104,051)	
Household wealth, $US	444,857 (1,184,514)		365,572 (1,497,100)		425,651 (2,133,574)		367,155 (766,326)	
Not employed		46.2		48.3		49.5		49.4
Smoking status								
Never		41.8		40.8		43.0		42.5
Former		44.6		40.8		40.1		40.7
Current		13.2		18.0		16.6		16.5
Cognitive function								
Episodic memory	10.4 (3.3)		10.3 (3.4)		10.1 (3.4)		10.3 (3.4)	
Mental status	13.5 (1.9)		13.5 (1.9)		13.3 (2.1)		13.3 (2.1)	
Total score	23.9 (4.4)		23.8 (4.5)		23.4 (4.6)		23.6 (4.7)	

Abbreviations: HS, high school; $PM_{2.5}$, particulate matter with aerodynamic diameter of 2.5 μm or less; SD, standard deviation.

[a] Census tract–specific concentrations of $PM_{2.5}$ in 2004 were derived using 24-hour daily means reported by Environmental Protection Agency air quality monitors within a 60-km radius of each census tract centroid. Quartile 1 is 4.500–9.942 $\mu g/m^3$; quartile 2 is 9.943–12.184 $\mu g/m^3$; quartile 3 is 12.185–13.796 $\mu g/m^3$; and quartile 4 is 13.797–20.661 $\mu g/m^3$.

of the coefficient for the third quartile is reduced by about 26%. The difference between the association of $PM_{2.5}$ with cognitive function was further reduced in model 3 after accounting for smoking status, but statistically significant differences remain between those in the lowest quartile of exposure and those in the third ($\beta = -0.43$, 95% CI: −0.63, −0.23) and highest ($\beta = -0.26$, 95% CI: −0.47, −0.05) quartiles. Although the coefficient for the fourth quartile of exposure is smaller than that for the third quartile, the difference is not statistically significant ($\beta = -0.17$, 95% CI: −0.37, 0.03).

We also examined the association between $PM_{2.5}$ and 2 subcomponents of cognitive function: episodic memory and mental status. Results are shown in Table 4, but for brevity, we show only coefficients for quartiles of $PM_{2.5}$ exposure (full model results are available upon request). Each model in Table 4 includes the covariates of the corresponding model in Table 3. Living in more polluted areas is associated with worse episodic memory after adjustment for individual- and neighborhood-level factors. Those living in more polluted areas also had worse performance on tasks representing mental status, but the association is only marginally significant ($P < 0.10$) after adjustment for individual socioeconomic characteristics.

Secondary analyses

We conducted several additional analyses that are not shown here but are available upon request. First, because residents' exposure to ambient air pollution may depend on the total number of years they have lived in an area, we examined models that adjusted for the length of time at residence. We observed the same pattern of results as those presented in Tables 3 and 4. The results were also unchanged with the inclusion of indicators of health that could be potentially

Table 3. Multilevel Regressions of Cognitive Function (β) Among 13,996 Adults Aged 50 Years or Older in the US Health and Retirement Study, 2004

Parameter	Model 1		Model 2		Model 3	
	β	95% CI	β	95% CI	β	95% CI
Census Tract–Level Variables						
Annual $PM_{2.5}$ exposure, quartiles[a]						
First (4.500–9.942 μg/m^3)	1.00	Referent	1.00	Referent	1.00	Referent
Second (9.943–12.184 μg/m^3)	−0.26	−0.49, −0.04	−0.10[a]	−0.30, 0.10	−0.10[b]	−0.30, 0.11
Third (12.185–13.796 μg/m^3)	−0.58	−0.80, −0.36	−0.43	−0.63, −0.23	−0.43	−0.63, −0.23
Fourth (13.797–20.661 μg/m^3)	−0.25	−0.48, −0.02	−0.26	−0.47, −0.05	−0.26	−0.47, −0.05
No high school degree (%)	−2.81	−3.78, −1.85	0.04	−0.87, 0.95	0.05	−0.86, 0.97
Median household income (logged)	1.07	0.78, 1.36	0.45	0.18, 0.72	0.45	0.18, 0.72
Individual-Level Variables						
Age, years	−0.19	−0.20, −0.18	−0.15	−0.16, −0.14	−0.15	−0.16, −0.14
Female	0.60	0.46, 0.74	0.86	0.73, 1.00	0.89	0.75, 1.02
Race/ethnicity						
Non-Hispanic white	1.00	Referent	1.00	Referent	1.00	Referent
Non-Hispanic black	−2.91	−3.21, −2.62	−2.31	−2.56, −2.07	−2.31	−2.56, −2.07
Hispanic	−2.98	−3.33, −2.63	−1.14	−1.48, −0.81	−1.14	−1.48, −0.81
Other	−2.14	−2.69, −1.59	−1.83	−2.29, −1.36	−1.82	−2.28, −1.35
Education, years			0.49	0.47, 0.52	0.49	0.47, 0.52
Household income (logged)			0.09	0.04, 0.14	0.09	0.04, 0.14
Household wealth (logged)			0.08	0.06, 0.10	0.08	0.06, 0.10
Not employed			−0.52	−0.68, −0.35	−0.52	−0.68, −0.36
Smoking status						
Never					1.00	Referent
Former					0.13[b]	−0.02, 0.28
Current					−0.02[b]	−0.22, 0.19
Intercept	25.43	22.10, 28.75	20.56	17.47, 23.64	20.55	17.46, 23.64

Abbreviations: CI, confidence interval; $PM_{2.5}$, particulate matter with aerodynamic diameter of 2.5 μm or less.

[a] Census tract–specific concentrations of $PM_{2.5}$ in 2004 were derived using 24-hour daily means reported by Environmental Protection Agency air quality monitors within a 60-km radius of each census tract centroid.

[b] All other coefficients are statistically different from 0 at $P < 0.05$.

important confounders in the association between $PM_{2.5}$ and cognitive function, including body mass index (weight (kg)/height (m)^2) and doctor diagnosed hypertension, heart attack, diabetes, cancer, and lung disease.

Recognizing that some individuals may be especially vulnerable to PM air pollution, we examined interactions between $PM_{2.5}$ and all covariates. We found no evidence of interactions between $PM_{2.5}$, sociodemographic factors, or socioeconomic factors (P for all interactions < 0.01). However, interactions between $PM_{2.5}$ and smoking status suggest that current smokers in the second quartile of exposure had worse cognitive function than nonsmokers in the same quartile and either smokers or nonsmokers in the bottom quartile of exposure (P for interaction = 0.04).

We also considered the potential influence of missing $PM_{2.5}$ and cognitive function data on the reported findings. First, we performed logistic regressions using individual-level sociodemographic and socioeconomic variables to predict missingness of cognitive function data and $PM_{2.5}$ data (i.e., 1 = missing, 0 = not missing). The following characteristics were associated with having significantly higher odds ($P < 0.05$) of missing cognitive data: greater $PM_{2.5}$ exposure, older age, male sex, "other" race/ethnicity, fewer years of education, greater income, and currently being employed. Those with worse cognitive function, younger age, male sex, non-Hispanic white race/ethnicity, and fewer years of education were significantly more likely to have missing $PM_{2.5}$ data ($P < 0.05$).

Second, we performed sensitivity analyses using an imputed cognitive function score for those with a proxy interview. We used an approach developed for the HRS that uses proxy assessments and interviewer observations to classify respondents as normal, cognitively impaired with no dementia, or demented, and then we assigned proxy respondents the median cognitive function score for each category (for normal cognitive function (range, 11–35), median,

Table 4. Multilevel Regressions of Episodic Memory and Mental Status Among 13,996 Adults Aged 50 Years or Older in the US Health and Retirement Study, 2004

Quartile of Annual $PM_{2.5}$ Exposure	Model 1[a]		Model 2[b]		Model 3[c]	
	β	95% CI	β	95% CI	β	95% CI
Episodic Memory						
First (4.500–9.942 μg/m^3)	1.00	Referent	1.00	Referent	1.00	Referent
Second (9.943–12.184 μg/m^3)	−0.21	−0.38, −0.04	−0.12[d]	−0.28, 0.04	−0.11[d]	−0.27, 0.04
Third (12.185–13.796 μg/m^3)	−0.44	−0.61, −0.27	−0.36	−0.51, −0.20	−0.35	−0.51, −0.19
Fourth (13.797–20.661 μg/m^3)	−0.17[d]	−0.34, 0.00	−0.18	−0.34, −0.01	−0.17	−0.33, −0.01
Mental Status						
First (4.500–9.942 μg/m^3)	1.00	Referent	1.00	Referent	1.00	Referent
Second (9.943–12.184 μg/m^3)	−0.06[d]	−0.15, 0.04	0.01[d]	−0.08, 0.10	0.01[d]	−0.07, 0.10
Third (12.185–13.796 μg/m^3)	−0.15	−0.25, −0.04	−0.09[d]	−0.18, 0.01	−0.09[d]	−0.18, 0.01
Fourth (13.797–20.661 μg/m^3)	−0.09[d]	−0.19, 0.02	−0.09[d]	−0.19, 0.00	−0.09[d]	−0.19, 0.00

Abbreviations: CI, confidence interval; $PM_{2.5}$, particulate matter with aerodynamic diameter of 2.5 μm or less.
[a] Model 1 includes percent of residents in the census tract with no high school diploma; median income in the tract; and individual age, sex, and race/ethnicity.
[b] Model 2 additionally adjusts for education, income, wealth, and employment status.
[c] Model 3 additionally adjusts for smoking status.
[d] All other coefficients are statistically different from 0 at $P < 0.05$.

23; for cognitive impairment with no dementia (range, 8–10), median = 9; and for dementia (range, 0–7), median = 6) using category cut points established in prior studies of the HRS) (21, 22). Inclusion of the proxy respondents with imputed scores did not change the findings reported here. Although we do not have information about $PM_{2.5}$ exposure for individuals who do not live in close proximity to an air monitor, we can assign these individuals to the lowest exposure quartile under the assumption that the EPA does not place monitors in areas with low $PM_{2.5}$ concentrations. Assigning individuals with missing $PM_{2.5}$ data to the lowest quartile did not change our findings.

DISCUSSION

In this cross-sectional study of older US men and women, exposure to $PM_{2.5}$ was associated with cognitive function after adjustment for community social characteristics and individual social, demographic, economic, and health characteristics. We did not find a linear association between $PM_{2.5}$ and cognitive function. Those living in areas with the highest exposure in our sample did not have the worst cognitive function. Rather, the data indicate that living in areas with annual $PM_{2.5}$ concentrations that exceed EPA air quality standards is harmful to older adults' cognition. To put our findings in perspective, we found that living in more polluted areas was associated with a decrease in cognitive function that is similar to a 1.7- to 2.8-year difference in age in our data. We did not find much support for effect modification by individual-level characteristics. However, our results suggest that, at relatively low $PM_{2.5}$ concentrations, cognition may be worse among older adults who smoke.

Separate analyses of 2 subcomponents of cognitive function suggest that it is episodic memory that is adversely affected by exposure to $PM_{2.5}$. Episodic memory impairment is 1 of the core diagnostic criteria used to determine mild cognitive impairment and dementia in older adults (23), and it has been suggested that impairments in this aspect of memory represent some of the earliest signs of dementia (24). Thus, exposure to PM may have the most significance for cognition in healthy older adults who have not yet demonstrated cognitive impairment.

To our knowledge, this is 1 of only a handful of studies to consider the association between air pollution and cognitive function in older adults and the first to do so using a nationally representative sample of older men and women. Most of the existing evidence for a link between air pollution and cognitive function comes from studies of younger populations (9, 25, 26). Yet there is now growing evidence of the importance of air pollution for the aging brain, as well. This study adds to existing research on air pollution and cognition in older adults by demonstrating that the pollution-cognition link observed in prior studies of select populations (12–16) is also present in a broader sample of older US men and women.

The current study has several limitations. There were substantial missing data on both cognitive function and $PM_{2.5}$. Because we excluded older adults who were institutionalized or had proxy-assisted interviews, the study findings are somewhat biased toward a cognitively intact older adult population. Our analysis of the characteristics of those with and without cognitive data confirms that our analytical sample differs in several ways from those with missing cognitive data. However, our findings were unchanged after imputing cognitive function for these individuals and including them in the analytical sample. We also excluded from our analyses those individuals for whom $PM_{2.5}$ exposure could not be determined. These individuals also differed in systematic ways from the analytical sample. However, we found the same pattern of results when we included those with missing $PM_{2.5}$ data in the lowest exposure quartile.

We used a measure of outdoor pollution that may not completely capture total individual exposure. Although outdoor pollution may be an important contributor to overall exposure, pollution exposure may also occur in other contexts, such as in the workplace or during daily road travel. However, most of our sample members were not employed, so outdoor residential pollution concentrations may be a good approximation of their total exposure. In addition, previous research has found high correlations between outdoor $PM_{2.5}$ exposure and both indoor and personal $PM_{2.5}$ exposure (27). This is likely due to relatively small spatial variability in $PM_{2.5}$ concentrations within urban areas. Thus, even though we use a measure of outdoor $PM_{2.5}$ from pollution monitors, we are also likely measuring a good portion of personal exposure. Moreover, we did not have data on long-term pollution exposure, though our measure may reflect some degree of longer-term air pollution. Pollution levels are highly correlated over time. Correlations of 0.98 and 0.94 have been reported for $PM_{2.5}$ measured in a given year and levels measured 1 and 4 years prior, respectively. $PM_{2.5}$ measured in a given year has also shown a high correlation ($\rho = 0.89$) with estimated levels from 10 years prior (15).

This study examined cognition with respect to only the fine fraction of PM and did not consider the coarse fraction of PM, which has been linked to cognitive decline in prior research (15). Although we found an association between $PM_{2.5}$ and cognitive function, we are not able to determine what components of $PM_{2.5}$ are primarily responsible for the association. $PM_{2.5}$ is a complex mixture of several components, including carbon and metals (28), and it remains unclear which component, or components, are responsible for the adverse health effects of $PM_{2.5}$. Some research suggests that traffic-related components are responsible for the health effects of $PM_{2.5}$ (29). This is supported by studies of cognition that have found associations with black carbon, a traffic-related component of $PM_{2.5}$ (13, 16).

Although we included a number of community- and individual-level covariates in our models, we were unable to account for other potentially important confounders, such as diet and cognitive engagement, which are thought to be key risk factors for poor cognitive function in old age. Finally, this study examined cross-sectional associations and, thus, causal pathways between air pollution and cognitive function in older adults cannot be determined. However, an association between PM air pollution and cognitive decline has been established in previous research using data from the Nurses' Health Study (15).

The concern over the health effects of fine PM air pollution is growing. Fine particles are small enough that they can pass through the throat and nose and ultimately enter the lungs and brain (30). Inhaled particulates can affect the respiratory and cardiovascular systems, with potential consequences for vascular pathology in the brain, and they may even directly cause damage to brain structure and physiology. Studies of both humans and animals have confirmed that exposure to PM is linked to harmful changes in brain health and functioning. The biological evidence, as well as findings from population-based studies, including the study presented here, provides further indication that air pollution plays an important role in brain aging.

The population of older adults living in large metropolitan areas is growing rapidly (31). Although air pollution levels have declined in recent years (32), older residents of many US cities continue to breathe air that is harmful to their health. In 2013, the EPA lowered the air quality standard for $PM_{2.5}$ from 15 to 12 $\mu g/m^3$. Our study found that exposure to $PM_{2.5}$ concentrations below 15 $\mu g/m^3$ was associated with worse cognition in older adults. Efforts to further reduce $PM_{2.5}$ concentrations in accordance with the recent EPA standard may have beneficial consequences for the cognitive health of the aging US population.

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