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Fine particulate matter exposure and olfactory dysfunction among urban-dwelling older US adults

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Abstract

Objectives—The olfactory nerve is anatomically susceptible to injury from pollution in inspired air, but there are no large-scale epidemiologic studies investigating this relationship.

Methods—Cross-sectional study using data from the National Social Life, Health, and Aging Project, a representative sample of home-dwelling US adults age 57–85 years. Olfactory function was tested using a validated 5-item odor identification test (Sniffin' Sticks). Exposure to fine particulate matter ($PM_{2.5}$) at each respondent's home was estimated as 1–12 month moving averages prior to olfactory assessment using validated spatio-temporal models.

Results—Olfactory dysfunction was significantly associated with $PM_{2.5}$ exposures averaged over 3–12 months in urban-dwelling respondents. The strongest effect was for 6 month average exposure (per 1-IQR increase in $PM_{2.5}$: OR 1.28, 95% CI 1.05, 1.55) adjusting for age, gender, race/ethnicity, education, cognition, comorbidity, smoking, and the season. Interestingly, the most deleterious effects were observed among the youngest respondents, 57–64 years old, and those living in the northeast and south.

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Study of Human Subjects

The Institutional Review Boards of The University of Chicago and NORC approved this study and all respondents provided written, informed consent.

Conclusions—We show for the first time that air pollution exposure is associated with poor olfaction among urban-living, older US adults.

Keywords

air pollution; fine particulate matter ($PM_{2.5}$); olfactory dysfunction; smell; elderly

1. Introduction

Loss of olfactory function poses a huge burden to older adults, with a prevalence of approximately 24% (Murphy et al., 2002; Jayant M Pinto et al., 2014; Schubert et al., 2009). Olfactory dysfunction has a major human impact, in terms of decreased quality of life (Smeets et al., 2009), impaired nutrition and enjoyment of foods (Schiffman and Graham, 2000), a decreased ability to detect hazards (e.g., gas leaks or fires) (Santos et al., 2004), decreased sex drive (Toller, 1999), and increased feelings of depression and distress (Smeets et al., 2009). Olfactory dysfunction may also be an indicator of the development of neurodegenerative conditions, such as Parkinson's (Ross et al., 2008) or Alzheimer's diseases (Devanand et al., 2000), and a predictor of mortality (Wilson et al., 2011; Jayant M. Pinto et al., 2014b; Devanand et al., 2015). Loss of olfactory function therefore represents a significant public health problem, particularly among older adults.

Anatomically, the olfactory neurons comprise the first cranial nerve, which is directly exposed to the outside environment due to its position in the roof of the nasal cavity (Cullen and Leopold, 1999; Pinto, 2011). Harmful airborne pollutants may come into direct contact with olfactory neurons and may thus play a role in olfactory decline. Additionally, air pollution may explain mechanisms through which loss of olfaction serves as an indicator of future neurocognitive decline; the olfactory nerve can serve as a route of transportation for inhaled particles between the environment and the brain that bypasses the blood-brain barrier (Lucchini et al., 2012; Oberdörster et al., 2004). While occupational exposures have also been associated with impaired olfaction (Doty, 2006; Gobba, 2006), few (if any) large-scale studies have examined the impact of air pollution on olfactory loss in the general population.

Of the common air pollutants, the impacts of fine particulate matter ($PM_{2.5}$, diameter < 2.5 μm) on olfactory function may be particularly important to examine. $PM_{2.5}$ is a class of pollutant with well-documented impacts on mortality, cardiovascular disease, and to a lesser extent cognitive health (Ailshire and Crimmins, 2014; Brunekreef and Holgate, 2002). Exposure to $PM_{2.5}$ has also been linked to poor olfaction in studies of younger adults in Mexico City, a city with substantially elevated $PM_{2.5}$ concentrations, compared to those living in nearby cities with lower pollution levels (Hudson et al., 2006; Calderon-Garciduenas et al., 2010). Indirect support for these findings was provided by a study of older German women (ages 68–79 years), which found olfactory dysfunction to be associated with distance to the nearest roadway, a proxy for $PM_{2.5}$ exposure (Ranft et al., 2009).

To examine the relationship between olfaction and $PM_{2.5}$ in the general population, we used data from the National Social Life, Health and Aging Project (NSHAP), a nationally

representative sample of older US adults (age 57–85) living at home (Suzman, 2009). In 2005–6, NSHAP performed olfactory testing and collected information on a wide range of health conditions and social measures (Schumm et al., 2009). We linked data for each NSHAP respondent to monthly $PM_{2.5}$ exposures estimated for each respondent at his/her home address using previously validated GIS-based spatio-temporal models (Yanosky et al., 2014). Using these data, we examined the association between individual-specific $PM_{2.5}$ exposures and olfactory function in an effort to explore pollution exposure as a risk factor for olfactory decline.

2. Methods

2.1. Study population

In 2005–6, professional interviewers from NORC at The University of Chicago conducted in-home interviews with 3,005 community-dwelling older adults (1,454 men and 1,551 women), a representative sample of the US community-dwelling population 57–85 years of age (O'Muircheartaigh et al., 2009; Suzman, 2009). Numerous measures were obtained for each respondent, including olfactory, demographic, social, psychological, and biological measures, as described below. Our main analyses was restricted to respondents who were determined to live in urban areas based on rural-urban commuting area (RUCA) codes, given that a majority of previous work on pollution and olfaction has centered on residents of Mexico City, a highly exposed urban group (Calderon-Garciduenas et al., 2010; Calderón-Garcidueñas et al., 2003). Others have compared the olfactory ability of residents of major European urban centers to that of people dwelling in entirely non-industrialized regions (Sorokowska et al., 2015, 2013). RUCA codes 1–3 were considered urban areas and codes 4–10 were considered rural (Hall et al., 2006). To evaluate whether any observed association between $PM_{2.5}$ and olfaction in urban respondents was different in rural respondents, we separately expanded our analyses to all NSHAP respondents and treated urban/rural dwelling as a potential modifier.

The Institutional Review Boards of The University of Chicago and NORC approved this study and all respondents provided written, informed consent.

2.2. Olfactory Assessment

Olfactory function was measured using a validated, odor identification test comprising a shortened version of the Sniffin' Sticks (Mueller and Renner, 2006; Schumm et al., 2009). Robust associations have been identified using the data obtained from this 5-item test (Jayant M. Pinto et al., 2014b, 2014a). Five felt-tipped pens containing different odorants were presented one at a time to respondents. After smelling the tip of the pen, respondents were given a card with four labeled pictures and asked to identify the odorant via a forced choice protocol. The odorant response sets were as follows (*correct odor in italics*): (1) chamomile, raspberry, *rose*, cherry; (2) smoke, glue, *leather*, grass; (3) *orange*, blueberry, strawberry, onion; (4) bread, *fish*, cheese, ham; and (5) chive, *peppermint*, pine, onion. Refusals to provide an answer to a given odorant were treated as incorrect.

A score of four or five correct answers was classified as normosmic, and a score of three or fewer correct answers was classified as olfactory dysfunction, a standard threshold (Jayant M Pinto et al., 2014; Schumm et al., 2009) which yields a prevalence of olfactory dysfunction consistent across studies (Murphy et al., 2002; Schubert et al., 2009). Changing the cutoff for olfactory dysfunction to ≤ 2 or ≤ 4 odors correct yielded similar results (data not shown).

2.3. Air pollution exposure assessment

Geographic Information Systems (GIS)-based spatio-temporal models predicting monthly $PM_{2.5}$ concentrations have been previously developed and validated for the conterminous US with high accuracy ($R^2 = 0.77$) (Yanosky et al., 2014). Models used measured $PM_{2.5}$ concentrations, monitoring site locations, location-specific site characteristics, location- and month-specific meteorology data, and spatial smoothing of monthly- and long-term average levels to describe small and large-scale spatial and temporal variability in these concentrations. From these models, 1, 3, 6, 9, and 12 month $PM_{2.5}$ exposures were estimated for each respondent as moving averages based on their home address and date of olfactory assessment.

2.4. Other covariates

Our analyses controlled for numerous potential confounders, including age and gender due to their previously observed, consistent associations with olfactory function (Brämerson et al., 2004; Murphy et al., 2002; Jayant M Pinto et al., 2014; Schubert et al., 2009); race/ethnicity; cognitive function; education, as a proxy for socioeconomic status; smoking; comorbidity; and season. Race/ethnicity was coded by self-report using standard categories: White, Black, Hispanic (non-Black), and Other. Education was defined as the highest degree or certification completed. These basic demographic factors were also considered as potential modifiers of any pollution-olfaction association, to identify potentially vulnerable subgroups of the population. For this interaction analysis only, age was treated as a categorical variable with respondents grouped into ages 57–64 years, 65–74 years, and 75–85 years, as in prior work.

Cognitive function was measured using the Short Portable Mental Status Questionnaire (SPMSQ, scores from 0–10) (Pfeiffer, 1975). Although smoking has an unclear association with olfactory dysfunction (Brämerson et al., 2004; Frye et al., 1990; Ranft et al., 2009; Vennemann et al., 2008), current smoking (based on either positive self-report or a salivary cotinine ≥ 15 ng/mL) was included as a potential confounder because of its mechanistic relevance to air pollution exposure. Further, smoking was evaluated as a potential modifier of the associations between $PM_{2.5}$ exposure and olfaction. A modified Charlson comorbidity index was calculated for each respondent based on occurrence of mortality-associated conditions (Charlson et al., 1987; Pham-Kanter, 2009) and also considered as a potential modifier. Because of known seasonal variation in PM levels (Bell et al., 2007) and plausible seasonal differences in olfaction, we also included a season variable to compare cooler (October–March) versus warmer (April–September) months.

Additional variables evaluated as potential modifiers included: physical activity, considered as either high activity (1+ times per week) or low activity (< 1 time per week); region of the country (West, Midwest, South, or Northeast; states included in each region are listed in Supplemental Material, Table S1); and current employment status.

2.5. Statistical Analysis

Multivariate logistic regression was used to estimate the relationship between $PM_{2.5}$ and olfactory dysfunction, adjusting for potential confounders. Wald tests were used to determine p-values and 95% confidence intervals (CI). For each variable considered as an effect modifier, the corresponding model included both its main effect and interaction with $PM_{2.5}$. We note that missing data for some covariates reduced the sample size in the multivariate models slightly.

To ensure that the results were not dependent on the chosen threshold between normosmia and olfactory dysfunction, multivariate linear regression models including all covariates were fit, treating the number of odors correctly identified (0–5) as the dependent variable. To ensure that a single odor was not significantly impacting the results, the multivariate logistic regression model including all covariates was re-fit after excluding one odor at a time. Separately, we broadened the analysis to the entire NSHAP cohort (urban- and rural-dwelling), including treating urban vs. rural-dwelling as an effect modifier.

Analyses were performed using person-level weights, accounting for non-response. Design-based standard errors were calculated using the linearization method together with the strata and primary sampling unit indicators. All statistical analyses were conducted using Stata Version 14.0 (StataCorp, 2015).

3. Results

Of the 3,005 respondents interviewed, 2,940 completed olfactory testing. Of these, 719 respondents were classified as rural-dwelling and excluded from the main analyses, leaving 2,221 respondents for our primary analysis. Cohort demographic characteristics, including olfactory function, are presented in Table 1. Olfaction scores ranged from 0 to 5, with 52% of the urban-dwelling older population estimated to measure scores of 4 and below. When dichotomized as normosmia (4 or 5 correct) and olfactory dysfunction (3 or less correct), 23% of the population had olfactory dysfunction. Of note, respondents had lived in their current location for an average of 20.8 years (Table 1), with less than 3% living there for less than 1 year. The yearly average $PM_{2.5}$ exposure for NSHAP respondents equaled 13.9 ± 4.3 $\mu g/m^3$ (Table 2). Geographic variation in $PM_{2.5}$ exposure was observed, with respondents in the Midwest and Northeast having higher 1-year exposures on average (17.4 and 16.2 $\mu g/m^3$, respectively) as compared to respondents in the West and South (13.0 and 11.5 $\mu g/m^3$, respectively). Seasonal variability in the monthly exposures was also observed, with higher average 1 month exposures in warmer months (17.0 $\mu g/m^3$) compared to cooler months (13.5 $\mu g/m^3$).

3.1. Olfaction and $PM_{2.5}$

Older adults who experienced higher $PM_{2.5}$ exposure levels tended to face greater odds of olfactory dysfunction: a 20% increase in odds per IQR increase in 1 month exposure (OR 1.20, 95% CI 0.98, 1.46), controlling for age, gender, race/ethnicity, and education (Table 3, Model 1). This relationship persisted despite further accounting for cognition, comorbidities, smoking, and season (Table 3, Model 2).

The association between $PM_{2.5}$ exposure and olfaction was stronger for longer-term exposures. An IQR increase in 3 or 6 month $PM_{2.5}$ exposure, for example, was significantly associated with increased odds of impaired olfaction after controlling for basic demographic factors (respectively, OR 1.24, 95% CI 1.04, 1.48; OR 1.24, 95% CI 1.02, 1.51; Table 3, Model 1) and in the full model which also controlled for cognition, comorbidity, smoking, and season (respectively, OR 1.22, 95% CI 1.02, 1.45; OR 1.28, 95% CI 1.05, 1.55; Table 3, Model 2). Similar results were seen in the fully adjusted model for 9 month and 1-year exposures (respectively, OR 1.24, 95% CI 1.01, 1.54; OR 1.24, 95% CI 1.01, 1.52; Table 3, Model 2). Effect sizes for all covariates for 6 month $PM_{2.5}$ exposure are presented in the supplementary material (see Supplemental Material, Table S2).

3.2. Effect Modification

Full results of effect modification across different variables are presented in Table 4. As our primary research question concerned effects of urban pollution based on prior literature, these models were restricted to the urban-dwelling NSHAP cohort. The association between $PM_{2.5}$ and olfaction was strongest for the 6 month exposure window, tests for interactions are limited to this window for brevity. Similar results were obtained among all exposure windows from 1 month to 1 year (see Supplemental Material, Table S3). Significant interaction effects were observed in three variables: age, region of the country, and employment status (Table 4). These three interaction terms were then combined in a single model to evaluate the independent effect of each interaction variable (Figure S1).

Pollution had a significantly stronger effect on the youngest age group, 57–64 years old, as compared to older individuals (65–74 or 75–85 years old) (Table 4, interaction $p = 0.002$). For the youngest age group, the odds of olfactory dysfunction was substantially higher per IQR increase in 6 month $PM_{2.5}$ exposures (OR 2.15, 95% CI 1.45, 3.20), adjusting for relevant covariates (Table 4). In contrast, the association between $PM_{2.5}$ exposures and olfaction was not significant for 6 month moving averages for either of the older age groups, although it was elevated in those 65–74 years (Table 4). This interaction remained robust after including the regional and employment status interactions as well ($p = 0.050$, Figure S1a). In fact, the model predicted that for $PM_{2.5}$ exposures around the 75^{th} percentile, the proportion with olfactory dysfunction would be approximately equal in the 57–64 and 65–74 year age groups.

There was significant regional variation in the effect of $PM_{2.5}$ such that 6 month exposure was strongly associated with olfactory dysfunction in the Northeast (OR 2.12, 95% CI 1.17, 3.83) with a similar trend in the South (OR 1.43, 95% CI 0.97, 2.10), but no effects were seen in the Midwest (OR 1.00, 95% CI 0.62, 1.61) and the West (OR 0.95, 95% CI 0.81,

1.12). This trend was largely unchanged after accounting for age and employment status interactions (p = 0.071, Figure S1b).

Because a greater proportion of younger respondents still worked (Table 1), we examined whether accounting for employment as an effect modifier could explain these findings, hypothesizing that working individuals may have greater exposures for the same ambient $PM_{2.5}$ level as compared to their older counterparts due to exposures experienced while commuting. Employment status was in fact a significant modifier such that the effect of 6 month $PM_{2.5}$ exposure was significant among those working (OR 1.74, 95% CI 1.27, 2.38), but not among those not working (OR 1.13, 95% CI 0.92, 1.40), albeit with a trend in the same direction. Thus employment status could potentially explain why pollution may have had its strongest effect among the youngest respondents. However, when including the interaction effects of age, employment status, and region in a model together, the modifying effect of employment (p = 0.226, Figure S1c) was almost entirely diminished. There were no other significant interactions with $PM_{2.5}$ exposure among the remaining covariates (Table 4).

3.3. Sensitivity Analyses

Sensitivity analyses using multivariate linear regression for the number of odors demonstrated a similar deleterious effect of pollution on olfaction consistent with the primary multivariate logistic regression analysis (see Table 3 and Supplemental Material, Table S4). In these models, increased exposure was associated with decreased olfaction, albeit generally without reaching statistical significance. The exceptions to this were the partially adjusted model including 3 month $PM_{2.5}$ exposure and the fully adjusted model including 6 month $PM_{2.5}$ exposure (see Supplemental Material, Table S4).

As a separate sensitivity analyses, we excluded one odor at a time to examine whether findings were driven primarily by one odor (see Supplemental Material, Table S5). It appeared that the strongest effects were from the rose and leather odors, as exclusion of these odorants resulted in a marginally smaller effect of $PM_{2.5}$ exposure at 3–12 month exposure windows. However, in all cases, the effect of $PM_{2.5}$ exposure remained largely intact and of similar magnitude, indicating that these results were not driven by one particular odorant.

Finally, in a separate analysis, broadened to include all NSHAP Wave 1 respondents without missing data, we tested whether the effect of 6-month $PM_{2.5}$ exposure observed in urban respondents was also present in rural-dwelling respondents. The $PM_{2.5}$-olfaction association remained significant among urban respondents as expected (OR 1.29, 95% CI 1.05, 1.60). No association was found between $PM_{2.5}$ and olfactory dysfunction among rural respondents (OR 0.82, 95% CI 0.53, 1.23). This interaction approached, but did not reach, significance (p = 0.064). For brevity, results here are limited to the 6 month exposure window, however results were similar among all exposure windows from 1 month to 1 year (see Supplemental Material, Table S6).

4. Discussion

We show here for the first time that exposure to outdoor air pollution is associated with worse olfaction in urban-dwelling older adults in the United States. This relationship persisted after adjusting for important potential confounders including age, gender, race/ethnicity, education, cognition, comorbidity, smoking, and season. Importantly, these associations were found at commonly observed $PM_{2.5}$ concentrations, which were near or above the current US EPA National Ambient Air Quality Standard (NAAQS) (12 $\mu g/m^3$, averaged over a three-year period) (US EPA, 2015a, 2015b), with 62.7% of urban-dwelling respondents having 1-year exposures above this threshold. Older people living in urban environments in the US thus face chronically high exposures to ambient $PM_{2.5}$ and attendant adverse health effects.

The effect of pollution on olfaction was greatest in individuals between 57–64 years of age. We were unable to find that $PM_{2.5}$ exposure was associated with olfactory function in older age groups who may have already experienced olfactory decline. Given that older age is the strongest risk factor for impaired olfaction, it is possible that the pronounced biological effects of aging in people over age 65 overwhelm the effects of air pollution on olfaction in the oldest respondents (Murphy et al., 2002; Jayant M Pinto et al., 2014; Schubert et al., 2009). The observed effect modification by age was somewhat attenuated with the inclusion of the pollution by employment status interaction term, but remained significant. It is possible that other, unmeasured, age-related factors and behaviors may underlie the age interaction, such as nasal inflammation or increased activity-associated respiration. The interaction with age may also explain our finding of stronger $PM_{2.5}$-olfactory associations in respondents that were currently employed vs. those who were not employed. These results indicate that employed individuals, who were more prevalent in the youngest examined age group, were more susceptible to the impact of $PM_{2.5}$ on olfaction. Additionally, we found no significant modification of the $PM_{2.5}$ effect by physical activity, thus there was no evidence that our observed age effect was driven by increased activity-associated ventilation in younger respondents or increased time spent outdoors. We acknowledge that this was an imperfect measure of activity and direct measures are needed in future work to support this idea. Further, it is possible that these effects are at least in part due to time spent outdoors, something not directly evaluated in our survey, a factor that should be considered in future work.

We previously identified a large disparity in olfaction among Blacks compared to Whites (Jayant M Pinto et al., 2014), which we hypothesized may have been driven by differences in environmental exposures. However, we found no effect modification of the $PM_{2.5}$-olfaction association by race/ethnicity, and race remained a significant predictor of olfactory dysfunction after accounting for $PM_{2.5}$ exposure, suggesting that our prior finding of a significant racial disparity in olfactory function may not be a product of $PM_{2.5}$ exposure (Jayant M Pinto et al., 2014). Additional analyses are needed of other environmental and occupational exposures that are particular to Blacks and may affect olfactory function.

Interestingly, we observed a significant interaction between region of the United States and the effect of $PM_{2.5}$ on olfaction. It is difficult to explain why the effects were strongest in the

South and Northeast and less strong or absent in the West and Midwest. As $PM_{2.5}$ is a heterogeneous pollutant, it is possible these differences are due to regional variations in pollutant composition (Bell et al., 2007).

It appeared that the pollution-olfaction association was strongest for the rose and leather odorants although notably the association was in the same direction and of similar magnitude for all odorants. Although there may be differences in susceptibility to pollution for olfactory neurons for different odorants, it is difficult to draw any major conclusions about individual odorants from our data as only 5 were tested here. Future work may be able to address the question of odorant specificity.

Finally, we could not identify an association between $PM_{2.5}$ exposure and risk for impaired olfaction among rural-dwelling respondents. This may be driven by known and significant differences in urban vs. rural particulate matter source or composition (Kundu and Stone, 2014); we speculate that PM from urban areas may have more traffic-related pollutants and may be more toxic to the olfactory system. There may also be residual confounding by factors such as differences in occupation, time spent outdoors, or prevalence of airway conditions (e.g., allergic rhinitis). These might vary significantly by location and all may plausibly impact olfaction or susceptibility of the olfactory system to airborne insults. Unfortunately, this study was a secondary data analysis, so direct measures of these conditions were not available. However, we note that although no direct measure of allergy symptoms was available, controlling for use of allergy medications did not change the results. Future prospective studies with larger samples are needed to address these issues definitively.

Importantly, these data are consistent with findings from the only other population study of air pollution and olfactory function to date, which found that closer proximity to the nearest expressway was associated with decreased olfactory function among German women ages 68–79 years (Ranft et al., 2009). Our work includes a more accurate measure of air pollution exposure and a substantially larger, more diverse, and nationally representative sample.

There are practical implications for these findings. Physicians should consider that olfactory impairment might be linked to commonly occurring air pollutants in addition to well-known occupational exposures. People who work in spaces with high PM exposure (e.g., traffic attendants, street repair workers, bus drivers, etc.) may have greater exposures and thus may have increased vulnerability to $PM_{2.5}$-induced health consequences (Han et al., 2005; Li et al., 2014). Further, the link between PM exposure and olfactory loss stresses the need for additional research into the health effects of air pollutants beyond the focus of conventional studies to date (heart, lung, and brain disease primarily), especially for sensory systems. Inhaled particles may be able to translocate through the olfactory nerve to the olfactory bulb and then on to higher brain areas (Oberdörster et al., 2004). This is consistent with the idea that pollution exposure may be one explanation for the finding that olfactory dysfunction presages neurodegenerative disease (Calderon-Garciduenas et al., 2010). Thus, the effect of PM (and other forms of pollution more broadly) on olfaction has potentially devastating public health implications across a range of upper airway, neurosensory and neurodegenerative diseases.

A strength of our study was our individual-specific exposure assessment, where we estimated $PM_{2.5}$ exposures at each respondent's residential address, thus reducing exposure error introduced by spatial variation in outdoor $PM_{2.5}$ concentrations. It should be noted, however, that our exposure assessment method does not capture exposure variation introduced by activity patterns and indoor exposures experienced by our NSHAP respondents. Any error resulting from this variation is likely to bias our results towards the null. Similarly our survey measure of olfactory function, odor identification, may introduce error in our findings, as it is affected by both olfactory and cognitive components. We controlled for this cognitive component by including cognition in our models. However, our cognition measure (SPMSQ) was imperfect, capturing little variation in our population and potentially diminishing our ability to examine this factor. Although the SPMSQ has high sensitivity and specificity for dementia, it has limited ability to detect mild cognitive impairment. To obviate the need to control for cognition, future studies may also want to use odor sensitivity as the health measure (concentration threshold of detection of an odorant), which may allow pollution impacts on olfaction to be examined directly independent of cognition.

The cohort in this study was limited to older adults ages 57–85 years. Given our finding that the effect of pollution on olfaction may be strongest in the younger end of our age range, it will be important to consider younger age groups (i.e., younger than 57 years) and longitudinal follow-up in future studies, especially to determine onset of the effect. Finally, an analysis of outdoor activity and occupation (data which was not available here) may provide further insights into these associations. Additional work is also needed to determine timing, duration, and mechanism of this relationship.

5. Conclusions

Exposure to fine particulate matter is associated with worse olfactory function, particularly in those 57–64 years of age living in urban US locations with $PM_{2.5}$ concentrations above the current EPA standard of 12 $\mu g/m^3$. Billions of people around the world are exposed to concentrations of $PM_{2.5}$ above this standard, so the impact upon the olfactory system is significant across the globe (Apte et al., 2015). Better understanding of the connection between pollution and olfaction will provide a model for use in other areas of neurosensory and respiratory biology.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Olfactory function and demographics for study population of urban-dwelling older adults in US.

Characteristic	Weighted %	Weighted mean ± SD
Odors correctly identified (n = 2,221)		
Impaired olfaction (0–3 correct)	23.3	
0	1.3	
1	2.3	
2	5.4	
3	14.2	
Normal olfaction (4–5 correct)	76.7	
4	28.3	
5	48.4	
Age (years, n = 2,221)		67.9 ± 8.0
Gender (men, n = 2,221)	48.8	
Race/ethnicity (n = 2,210)		
White	78.0	
Black	11.2	
Hispanic, non-Black	8.4	
Other	2.4	
Education (n = 2,221)		
< High school	18.0	
High school graduate or equivalent	24.6	
Some college	30.2	
Bachelors or higher	27.2	
Cognition (SPMSQ, n = 2,221)		9.2 ± 1.2
Modified Charlson comorbidity index (n = 2,221)		1.7 ± 1.7
Smoking (current smokers, n = 2,221)	18.2	
Season (cool, n = 2,221)	30.3	
Freq. of physical activity (1+ times per week, n = 2,218)	78.3	
Employment (current employed, n = 2,220)	40.1	
Among those 57–64 years age	61.3	
Among those 65–74 years age	31.9	
Among those 75–85 years age	15.2	
Region (n = 2,221)		
West	22.7	
Midwest	20.6	
South	37.6	
Northeast	19.2	
Time lived in current neighborhood (years, n = 1,731)		20.8 ± 16.7

Table 2

$PM_{2.5}$ exposure for study population of urban-dwelling older adults in US.

Predicted $PM_{2.5}$ Measure ($\mu g/m^3$, n = 2,221)	Weighted mean ± SD	25^{th} percentile – 75^{th} percentile (IQR)
1 month	15.9 ± 6.2	11.3 – 19.8 (8.5)
3 month	15.8 ± 5.4	12.0 –19.7 (7.7)
6 month	14.8 ± 4.7	11.3 – 18.7 (7.4)
9 month	14.4 ± 4.5	10.8 – 18.2 (7.4)
1 year	13.9 ± 4.3	10.6 – 17.4 (6.8)

Table 3

Effects of pollution exposure on olfactory dysfunction (logistic regression), controlling for demographic variables (Model 1), and other covariates (Model 2) in older adults (n = 2,210).

	Odds Ratio per 1-IQR increase in $PM_{2.5}$ exposure (95% Confidence Interval)				
	1 month $PM_{2.5}$	**3 month $PM_{2.5}$**	**6 month $PM_{2.5}$**	**9 month $PM_{2.5}$**	**1 year $PM_{2.5}$**
$PM_{2.5}$ exposure IQR	8.5 μg/m^3	7.7 μg/m^3	7.4 μg/m^3	7.4 μg/m^3	6.8 μg/m^3
Model 1[a]	1.20 (0.98, 1.46)	1.24 (1.04, 1.48)	1.24 (1.02, 1.51)	1.20 (0.97, 1.48)	1.19 (0.97, 1.47)
Model 2[b]	1.14 (0.94, 1.38)	1.22 (1.02, 1.45)	1.28 (1.05, 1.55)	1.24 (1.01, 1.54)	1.24 (1.01, 1.52)

[a] Adjusted for age, gender, race/ethnicity, and education

[b] Adjusted for age, gender, race/ethnicity, education, cognition, comorbidity, smoking, and season

Table 4

Modifiers of effect of 6 month $PM_{2.5}$ exposure on olfactory dysfunction. Models controlled for age, gender, race/ethnicity, education, cognition, comorbidity, smoking, and season.

Effect Modifier	Odds Ratio per 1-IQR (7.4 μg/m^3) increase in 6 month $PM_{2.5}$ exposure (95% Confidence Interval)	P-value for interaction
Age (years) (n = 2,210)		0.002
57–64	2.15 (1.45, 3.20	
65–74	1.11 (0.80, 1.54)	
75–85	1.00 (0.79, 1.25)	
Gender (n = 2,210)		0.578
Men	1.21 (0.91, 1.61)	
Women	1.34 (1.06, 1.68)	
Race/ethnicity (n = 2,210)		0.694
White	1.21 (0.97, 1.51)	
Black	1.59 (1.01, 2.51)	
Hispanic, non-Black	1.42 (0.83, 2.44)	
Other	1.26 (0.56, 2.80)	
Education (n = 2,210)		0.395
< High school	1.40 (1.01, 1.95)	
High school graduate or equivalent	1.59 (1.14, 2.22)	
Some college	1.06 (0.70, 1.61)	
Bachelors or higher	1.14 (0.69, 1.88)	
Comorbidity (modified Charlson index) (n = 2,210)[a]		0.867
0	1.26 (0.96, 1.67)	
5	1.31 (1.00, 1.71)	
Smoking (n = 2,210)		0.545
Not current smoker	1.31 (1.08, 1.59)	
Current smoker	1.15 (0.76, 1.75)	
Region (n = 2,210)		0.034
West	0.95 (0.81, 1.12)	
Midwest	1.00 (0.62, 1.61)	
South	1.43 (0.97, 2.10)	
Northeast	2.12 (1.17, 3.83)	
Employment status (n = 2,209)		0.013
Not working	1.13 (0.92, 1.40)	
Working	1.74 (1.27, 2.38)	
Physical Activity (n = 2,207)		0.958
Low activity (< 1 time per week)	1.28 (0.89, 1.86)	
High activity (1+ times weekly)	1.27 (1.03, 1.56)	

[a] ORs for comorbidity index are presented for 5th and 95th percentiles (0 and 5, respectively) to represent a range of values for this variable.