





Associations between air pollution and mental health service use in dementia: a retrospective cohort study

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ABSTRACT

Background Little is known about the role of air pollution in how people with dementia use mental health services.

Objective We examined longitudinal associations between air pollution exposure and mental health service use in people with dementia.

Methods In 5024 people aged 65 years or older with dementia in South London, high resolution estimates of nitrogen dioxide (NO₂) and particulate matter (PM_{2.5} and PM₁₀) levels in ambient air were linked to residential addresses. Associations between air pollution and Community Mental Health Team (CMHT) events (recorded over 9 years) were examined using negative binomial regression models. Cognitive function was measured using the Mini Mental State Examination (MMSE) and health and social functioning was measured using the Health of the Nation Outcomes Scale (HoNOS65+). Associations between air pollution and both MMSE and HoNOS65+ scores were assessed using linear regression models.

Findings In the first year of follow-up, increased exposure to all air pollutants was associated with an increase in the use of CMHTs in a dose-response manner. These associations were strongest when we compared the highest air pollution quartile (quartile 4: Q4) with the lowest quartile (Q1) (eg, NO₂: adjusted incidence rate ratio (aIRR) 1.27, 95% CI 1.11 to 1.45, p<0.001). Dose-response patterns between PM_{2.5} and CMHT events remained at 5 and 9 years. Associations were strongest for patients with vascular dementia. NO₂ levels were linked with poor functional status, but not cognitive function.

Conclusions Residential air pollution exposure is associated with increased CMHT usage among people with dementia.

Clinical implications Efforts to reduce pollutant exposures in urban settings might reduce the use of mental health services in people with dementia, freeing up resources in already considerably stretched psychiatric services.

INTRODUCTION

Dementia is now the leading cause of death in the UK, with an estimated 850 000 individuals living with the condition. As the UK population ages, this number is projected to increase to 1 million

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ There are known associations between air pollution exposure and increased health service use in people with dementia.
- ⇒ However, these studies have largely focused on dementia-related admissions in general hospital settings.

WHAT THIS STUDY ADDS

- ⇒ In a large cohort of people with dementia in South London, we showed that exposure to higher levels of both PM_{2.5} and NO₂ was associated with increased use of mental health services, particularly in people with vascular dementia.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ Efforts to reduce pollutant exposures in urban settings, especially those related to diesel emissions, may benefit the health and well-being of patients living with dementia.
- ⇒ Reducing pollutant exposure might reduce the use of mental health services in people with dementia, freeing up resources in already considerably stretched psychiatric services.

by 2025, and 2 million by 2050.¹ Identifying modifiable risk factors relating to illness severity and relapse following onset is therefore a crucial research challenge that could inform early intervention efforts, reducing human suffering and the associated high healthcare and social costs associated with dementia. One potential modifiable population level risk factor is air pollution, which is a major public health concern globally, known to negatively impact health across multiple body systems throughout the life course, with consequences for both physical and mental health.²⁻⁴ A considerable amount of research has focused on the role of air pollution in older age, and there is a growing appreciation of potential links between exposure to pollutants and dementia.^{5 6} The UK Committee on the Medical Effects of Air Pollutants (COMEAP) recently reviewed a large number of studies and concluded that it is likely that ambient



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air pollution contributes to both the risk of developing dementia and the acceleration of cognitive decline.⁷

If exposure to air pollution contributes to accelerated cognitive decline and the progression of dementia, it is possible that exposure to air pollution could also increase the use of health and care services. To date, evidence suggests that long-term exposure to particulate matter (PM) is associated with time to first hospitalisation for dementia,^{8,9} raising the possibility that air pollution might accelerate the progression of dementia after clinical onset. PM has also been found to be associated with an increased risk of dementia-related hospital admissions¹⁰ and Alzheimer's disease-specific emergency hospital admissions.¹¹ However, in the UK, dementia care is usually managed outside of general hospital settings with most patients with late-onset dementia receiving diagnostic assessment and treatment initiation, if indicated, from Memory Assessment Services and hospital- and primary care-based multidisciplinary dementia services.¹² Subsequent care for behavioural/psychological symptoms is provided by psychiatric services, particularly Community Mental Health Teams (CMHTs) for older adults. A previous London-based study found that exposure to both PM_{2.5} and nitrogen dioxide (NO₂) was associated with increased CMHT use in people who had recently been diagnosed with psychotic or mood disorders.¹³ However, to date, there has been no investigation of the impact of air pollution on mental health service use in people with dementia.

Therefore, in the current study we aimed to examine longitudinal associations between air pollution and CMHT usage in people with dementia. Effects of air pollution on the brain have been proposed as a consequence of its established impact on the cardiovascular system,⁷ implicating air pollution particularly in vascular dementia, so we also examined whether the strength of associations between air pollution and CMHT usage differed according to dementia subtype (ie, Alzheimer's disease, vascular dementia, other dementia). We hypothesised that higher long-term air pollution exposures would be associated with increased CMHT events over short- (1 year), medium- (5 years) and long-term (9 years) follow-up periods, and that these associations would be strongest among patients with vascular dementia. We also investigated the extent to which air pollution was associated with routine measures of cognitive function and measures of health and social functioning, in order to shed light on underlying reasons for any associations with CMHT usage among people with dementia.

METHODS

Study design and participants

We assembled a retrospective cohort study drawn from a large volume of healthcare information. The South London and Maudsley National Health Service (NHS) Foundation Trust (SLaM) is one of Europe's largest secondary mental healthcare providers.¹⁴ SLaM provides comprehensive secondary mental healthcare, including dementia assessment and management services, to an urban catchment area of approximately 1.36 million people within the London boroughs of Southwark, Lambeth, Lewisham and Croydon. Since 2006, SLaM has deployed fully electronic health records. The Clinical Record Interactive Search (CRIS) system, established in 2008, is a platform and governance framework that allows researchers to access complete, de-identified data from SLaM records for research.¹⁴ CRIS holds all information documented by professionals involved in the provision of specialist mental healthcare for all people in contact with SLaM services from 1 January 2007 to date, in addition to earlier

legacy data.^{14,15} CRIS has received research ethics committee approval as an anonymised data resource for secondary analyses (Oxford Research Ethics Committee C, reference 18/SC/0372). The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments.

Routine clinical diagnoses are recorded in UK dementia assessment services, including SLaM, according to codes from the International Classification of Diseases, 10th Edition (ICD-10). Individuals with diagnoses of dementia in Alzheimer's disease (ICD-10 codes F00*), vascular dementia (F01*), dementia in other disease classified elsewhere (F02*), and unspecified dementia (F03*) were identified from pre-structured fields. Our sample comprised individuals aged 65 years and over who had a first face-to-face contact in SLaM between 1 January 2008 and 31 December 2012 and received a primary diagnosis of dementia either at or after this initial contact. Individuals were followed from the date of their first face-to-face contact (baseline) for up to 9 years (data extraction). Air pollution was modelled for Southwark, Lambeth, Lewisham and Croydon; therefore, we only included individuals who resided within these four boroughs at the time of first face-to-face contact. Participants of no fixed abode were also excluded by design.

Measures

Air pollution

High-resolution (20×20 m) air pollution models for Greater London were produced for 2008–2012 using KCLurban, based on the Atmospheric Dispersion Modelling System model v4 and Road Source model v2.3 (Cambridge Environmental Research Consultants), hourly meteorological data, empirically derived atmospheric pollutant relationships and emission estimates recorded in the London Atmospheric Emissions Inventory. Exposure data were outputted as quarterly (3-monthly) mean concentrations of nitrogen dioxide (NO₂), particulate matter with an aerodynamic diameter <2.5 μm (PM_{2.5}) and particulate matter with an aerodynamic diameter <10 μm (PM₁₀). Quarterly outputs covered the 3-month period in which the first face-to-face contact took place. As a de-identified data resource, CRIS holds no address data on patients. Therefore, linkage of patients' addresses to air pollution data was undertaken within a pre-CRIS data processing pipeline, while maintaining de-identification at the researcher interface. Pollution exposure estimates used the bilinear interpolation method using the four 20×20 m points around each address. A comprehensive description of the model and its validation against ground-based measurements has been published previously.¹⁶ For instance, the model's predictions perform well against actual observations, with correlations in 2008 exceeding $r^2=0.93$.¹⁶

Mental health service use: Community Mental Health Team (CMHT) events

CMHTs for older adults provide community-based treatment and care for people aged ≥65 years who are living in the community with mental health difficulties or dementia. CMHT entry criteria for people with dementia include the presence of comorbid depression or psychosis, or significant behavioural and/or psychological symptoms of dementia (eg, aggression, agitation, distress).

CMHT events were measured over 1-, 5-, and 9-year follow-up periods following first face-to-face contact with SLaM. As median survival time from diagnosis ranges from approximately 4 to 6 years depending on dementia type,¹⁷ this allowed us to

assess both shorter-, mid- and long-term associations with air pollution. Events were estimated as the number of distinct face-to-face attended appointments including, for example, outpatient visits and appointments with specialist teams.

Cognitive function

Cognitive function was measured from recorded Mini Mental State Examination (MMSE) scores¹⁸ in the patients' records. The MMSE is a cognitive test used in clinical settings routinely and has been extensively validated. It is scored out of 30, with lower scores indicating more severe cognitive impairment. In line with previous analyses using the CRIS database, we restricted analyses to patients with at least three individual MMSE scores in the observation period.¹⁹ We examined three time periods in line with our outcome: mean MMSE score from first face-to-face contact with SLAM services to 12 months (Year 1: short-term follow-up), 12 months to 5 years (Year 5: medium-term follow-up) and 5 years to 9 years (Year 9: long-term follow-up).

Health and social functioning

Health and social functioning were measured using the Health of the Nations Outcome Scale for older people (HoNOS65+). The HoNOS65+ is a set of 12 subscales measuring health and social functioning in secondary care mental health services across domains of behaviour, functional impairment, symptoms and social functioning.²⁰ Each item is scored on a scale ranging from 0 (no problem) to 4 (severe problem). The HoNOS65+ items are often summed to form a total score ranging from 0 to 48, with higher scores indicative of poorer health and social functioning. For the analyses reported here, total HoNOS65+ scores and scores on each separate HoNOS65+ subscale were used. As with the MMSE, we restricted analyses to patients with at least three HoNOS65+ scores in the observation period and assessed associations between air pollution and the mean HoNOS65+ score at short-term, medium-term and long-term follow-up.

Confounders

Quarter and year of first face-to-face contact were included as covariates to control for seasonal fluctuations and annual trends in air pollution concentrations.

Individual-level demographic and clinical covariates were selected based on potential associations with mental health service use. These included age at first presentation (calculated using year of birth),²¹ gender,²² ethnicity,²³ marital status at initial presentation²⁴ and number of comorbid mental health conditions recorded over the 9-year follow-up period.²⁵ The number of comorbid mental health conditions was a count variable generated from summing the number of conditions participants had a record of in the 9-year study period. These included depression (F32, F33), schizophrenia (F20) and schizoaffective disorders (F25), bipolar affective disorder (F30, F31), and mental and behavioural disorders due to psychoactive substance use (F1*).

Area-level covariates included population density, neighbourhood deprivation and social fragmentation.²⁴ Population density was based on the number of people per hectare and was determined using 2011 census data. Neighbourhood deprivation was measured using Index of Multiple Deprivation (IMD) scores at the lower-layer super output area (LSOA) level. LSOAs are geographical areas designed to support the reporting of small area statistics, and each area includes approximately 1000–3000 people. IMD scores are based on seven domains: income,

employment, education, skills and training, health and disability, crime, barriers to housing and services, and living environment. Social fragmentation was measured at the LSOA level and composite scores were created based on four domains from the 2011 census: number of private renters, number of single people, number of single-person households and population turnover.

Statistical analysis

Sample characteristics were described using means and standard deviations (SD), medians and interquartile ranges (IQR), and frequencies. One-way ANOVAs, Kruskal–Wallis tests and χ^2 tests were used to examine differences in characteristics between dementia subtypes. Moreover, we used these tests to investigate whether there were differences between the analytical sample and those excluded from the study.

All air pollutant exposure estimates were rescaled to IQR increments which allows effect estimates to be calculated to permit comparison across different pollutants. Air pollutants were also categorised into quartiles to permit assessment of dose-response associations. Associations between air pollution exposure and number of CMHT events were assessed using negative binomial regression in order to handle skewed count data and to account for the overdispersion (ie, the observed variance was greater than the expected variance) of the outcome variable. Time in contact with SLAM services ('active SLAM days') was included as the offset variable to account for differences in follow-up periods (eg, due to death or moving out of the catchment area). Associations between air pollution and number of CMHT events were examined in all dementia types, and then separately for each dementia subtype (Alzheimer's disease, vascular dementia, other or unspecified dementia). We examined the regression coefficients to determine which associations were strongest.

All models were fitted separately for each air pollutant and adjusted for season and year (model 1), plus age, gender, ethnicity, marital status and number of comorbid mental health conditions (model 2). Area-level covariates (IMD, social fragmentation, population density) were added in model 3. All models only included participants who had complete data for model 3.

Associations between air pollutants and MMSE and HoNOS65+ scores were assessed using fully adjusted (model 3) linear regressions due to the continuous distribution of these variables.

As recommended for observational research, we calculated E-values.²⁶ E-values represent the minimum strength of association that an unmeasured confounder would need to have with both air pollution and CMHT events in order to explain away the significant effects reported in this study.

To estimate the percentage of mental health service use that could be attributable to air pollution, we calculated population attributable fractions (PAFs) at the Year 1 follow-up period for two exposure scenarios (London averages and UK urban traffic area averages). We calculated PAFs for NO₂ and PM_{2.5}. Further details are provided in online supplemental methods.

All analyses were performed using STATA 17.0 (Stata Corp LLP, College Station, Texas, USA).

Sensitivity analysis

Six sensitivity analyses were performed:

1. We examined differences between the analytical sample and the sample who were excluded based on missing data.
2. To address biases due to missing data on air pollution and covariate data, we repeated the analysis following multiple

- imputation by chained equations (further details provided in online supplemental methods).
- We examined whether associations between air pollution and CMHT events were modified by neighbourhood deprivation by including an interaction term with IMD in regression models.
 - We examined whether associations between air pollution and CMHT events were modified by borough of residence (Croydon, Lambeth, Lewisham or Southwark) by including an interaction term with borough in regression models.
 - To examine co-pollutant confounding, we ran fully adjusted two-pollutant models where each pollutant was included as a covariate with every other pollutant.
 - It is possible that there were some clustering effects based on people with dementia in the current study being resident in

the same care home. As information about care home residency was not available, we examined associations between air pollution and CMHT events using mixed effects negative binomial regression where exposure to air pollution was considered at level 1 (fixed effects) and LSOA was considered at level 2 (random effects).

RESULTS

Sample characteristics

The current study included participants with complete data for the exposure variables of interest to ensure that any differences between models were not due to selection bias (see online supplemental figure S1 for a flowchart depicting sample selection). The sample comprised 5024 people aged ≥ 65 years with dementia

Table 1 Sample characteristics

	Overall (n=5024)	Alzheimer's (n=2718, 54.1%)	Vascular dementia (n=1022, 20.3%)	Other/unspecified dementia (n=1330, 26.5%)	P value
	Mean \pm SD, N (%)	Mean \pm SD, N (%)	Mean \pm SD, N (%)	Mean \pm SD, N (%)	
Age, years	81.4 \pm 7.2	81.2 \pm 7.1	81.6 \pm 6.9	81.7 \pm 7.6	0.051
Female	3132 (62.3)	1803 (66.3)	581 (56.8)	778 (58.5)	<0.001
Ethnicity					0.004
White	3934 (78.3)	2170 (79.8)	762 (74.6)	1035 (77.8)	
Mixed	31 (0.6)	11 (0.4)	8 (0.8)	12 (0.9)	
Asian	262 (5.2)	147 (5.4)	55 (5.4)	62 (4.7)	
Black	718 (14.3)	346 (12.7)	181 (17.7)	202 (15.2)	
Other	69 (1.4)	37 (1.4)	13 (1.3)	19 (1.4)	
Not stated	10 (0.2)	7 (0.3)	3 (0.3)	–	
Marital status					<0.001
Married/cohabiting	1696 (33.8)	988 (36.4)	327 (32.0)	396 (29.8)	
Divorced/separated	396 (7.9)	206 (7.6)	85 (8.3)	104 (8.6)	
Widowed	2034 (40.5)	1092 (40.2)	416 (40.7)	543 (40.8)	
Single	740 (14.7)	371 (13.6)	145 (14.2)	228 (17.1)	
Not disclosed	158 (3.1)	61 (2.2)	49 (4.8)	49 (3.7)	
Mental health comorbidities, median (IQR)	0 (0–0) Range: 0–3	0 (0–0) Range: 0–3	0 (0–0) Range: 0–2	0 (0–0) Range: 0–2	0.020
Neighbourhood deprivation	26.0 \pm 11.6	24.6 \pm 11.8	27.7 \pm 11.1	27.5 \pm 11.2	<0.001
Population density	92.7 \pm 52.7	86.8 \pm 52.4	101.4 \pm 53.7	98.9 \pm 51.4	<0.001
Social fragmentation	2.3 \pm 2.5	2.0 \pm 2.5	2.7 \pm 2.2	2.7 \pm 2.3	<0.001
CMHT events					
Year 1, median (IQR)	4 (2–8.5)	4 (2–8)	4 (2–9)	5 (2–10)	0.478
Year 5, median (IQR)	8 (3–17)	9 (4–18)	6 (3–15)	7 (3–16)	<0.001
Year 9, median (IQR)	9 (4–19)	10 (4–20)	7 (3–16)	8 (3–18)	<0.001
Active SLAM days					
Year 1, median (IQR)	246 (87.5–366)	330 (121–366)	167 (51–356)	191 (65–366)	<0.001
Year 5, median (IQR)	394.5 (140.5–899)	553.5 (199–1084)	245 (87–563)	282 (109–646)	<0.001
Year 9, median (IQR)	428 (151–960.5)	613 (228–1156)	262.5 (94–615)	299.5 (116–680)	<0.001
Mortality					<0.001
Year 1	594 (11.8)	219 (8.1)	167 (16.3)	208 (15.6)	
Year 5	2660 (52.9)	1225 (45.1)	632 (61.8)	803 (60.4)	
Year 9	3965 (78.9)	2024 (74.5)	867 (84.8)	1074 (80.8)	
NO ₂ (μ g/m ³)	38.6 \pm 10.2	37.8 \pm 10.2	39.8 \pm 10.3	39.3 \pm 10.0	<0.001
PM _{2.5} (μ g/m ³)	14.4 \pm 3.0	14.3 \pm 3.0	14.6 \pm 3.1	14.4 \pm 2.9	0.009
PM ₁₀ (μ g/m ³)	21.1 \pm 4.3	20.8 \pm 4.3	21.5 \pm 4.4	21.3 \pm 4.3	<0.001

Note: Overlap between dementia subtypes means sample size will be greater than 100%.

Neighbourhood deprivation: Index of Multiple Deprivation (IMD) score; population density: persons per hectare; social fragmentation: z-scored composite of unmarried adults, single-person households, housing tenure and population turnover.

CMHT, Community Mental Health Teams; NO₂, nitrogen dioxide; PM₁₀, particulate matter with a diameter of <10 μ m; PM_{2.5}, particulate matter with a diameter of <2.5 μ m; SLAM, South London and Maudsley National Health Service (NHS) Foundation Trust.

who had their first face-to-face contact with SLAM services between 2008 and 2012 (table 1). Dementia diagnosis was the first diagnosis made after the initial face-to-face contact. Of these, 54.1% had a diagnosis of Alzheimer's disease (n=2718), 20.3% had vascular dementia (n=1022) and 26.5% had other or unspecified dementia (n=1330). The total exceeding 100% reflects some diagnostic overlap.

In the overall sample, the mean age at first face-to-face contact was 81.4 years, 62.3% were female, 78.3% were of white ethnicity and 40.5% were widowed. A higher proportion of people with Alzheimer's disease were female (66.3%, $p<0.001$), of white ethnicity (79.8%, $p=0.004$) and married/cohabiting (36.4%, $p<0.001$) relative to those with vascular or other/unspecified dementia. People with vascular dementia resided in areas with higher levels of neighbourhood deprivation (27.7 ± 11.1 , $p<0.001$), population density (101.4 ± 53.7 , $p<0.001$) and social fragmentation (2.7 ± 2.2 , $p<0.001$) compared with the other dementia types, with starkest differences observed with Alzheimer's disease. Exposure to all air pollutants was highest in people with vascular dementia and lowest in people with Alzheimer's disease.

The number of CMHT events in the first year of follow-up did not differ between dementia subtypes, but at Year 5 and Year 9 people with Alzheimer's disease had a higher number of events. People with Alzheimer's disease also had a higher number of active SLAM days at each time point. This may be because people with Alzheimer's disease had the lowest mortality rates at 1, 5 and 9 years.

Air pollution and mental health service use

Associations between air pollution exposure and mental health outcomes for Year 1, Year 5 and Year 9 are shown in figure 1 (and online supplemental table S1). In fully adjusted models, clear dose-response associations emerged between all air pollutants and the number of CMHT events in the first year of follow-up. The most pronounced consistent dose-response association emerged for NO_2 exposure, particularly when we compared the highest quartile (Q4) with the lowest (Q1) (adjusted incidence rate ratio (aIRR) 1.27, 95% CI 1.11 to 1.45, $p<0.001$). Higher levels of $\text{PM}_{2.5}$ and PM_{10} exposure were also associated with more CMHT events in the first year of follow-up, but associations were only significant among the higher quartiles (eg, $\text{PM}_{2.5}$ – Q4 vs Q1: aIRR 1.33, 95% CI 1.16 to 1.53, $p<0.001$).

Dose-response associations between $\text{PM}_{2.5}$ and CMHT events at Year 5 and Year 9 became slightly stronger (Year 5 – Q4 vs Q1: aIRR 1.45, 95% CI 1.20 to 1.75, $p<0.001$; Year 9 – Q4 vs Q1: aIRR 1.46, 95% CI 1.19 to 1.80, $p<0.001$). At Year 5, significant associations remained only between the highest level of PM_{10} exposure and number of CMHT events (aIRR 1.20, 95% CI 1.01 to 1.43, $p=0.036$), but this relationship disappeared by Year 9 (aIRR 1.18, 95% CI 0.98 to 1.42, $p=0.081$). Associations between NO_2 and CMHT events at Year 5 and Year 9 were less clear, with dose-response relationships disappearing at these time points. For example, the relationship between NO_2 exposure and CMHT events was strongest for the second highest quartile (Q3) versus the lowest quartile (Q1) (Year 5 – Q3 vs Q1: aIRR 1.35, 95% CI 1.16 to 1.57, $p<0.001$; Year 9 – Q3 vs Q1: aIRR 1.27, 95% CI 1.08 to 1.49, $p=0.003$).

Associations between each air pollutant measured continuously and the number of CMHT events are also provided in online supplemental table S1. Across all follow-up periods, the strongest associations were consistently seen between NO_2 exposure and CMHT events (Year 1: aIRR 1.18, 95% CI 1.09

to 1.28, $p<0.001$; Year 5: aIRR 1.18, 95% CI 1.10 to 1.28, $p<0.001$; Year 9: aIRR 1.18, 95% CI 1.10 to 1.27, $p<0.001$).

E-values indicated that unmeasured confounding was unlikely to explain away a substantial proportion of the observed associations for $\text{PM}_{2.5}$, PM_{10} and NO_2 (online supplemental table S2A–C).

Population attributable fractions (PAFs)

PAFs are shown in online supplemental table S3. Our analysis suggests that, if the annual (2019) $\text{PM}_{2.5}$ exposure in London ($11.6 \mu\text{g}/\text{m}^3$) was reduced to the WHO's recommended annual limit ($5 \mu\text{g}/\text{m}^3$), then the annual number of CMHT events in people with dementia could be reduced by 13% (95% CI 7% to 18%). Reducing annual (2019) NO_2 levels in London ($39.0 \mu\text{g}/\text{m}^3$) to the WHO's recommended limits ($10 \mu\text{g}/\text{m}^3$) could bring about a 38% (95% CI 22% to 51%) reduction in CMHT events in those with dementia. For other UK urban traffic areas (2021), reductions in air pollution to WHO recommendations could also result in a considerable reduction in the number of CMHT events ($\text{PM}_{2.5}$: 6%; NO_2 : 22%).

Air pollution and CMHT events: dementia subtypes

Fully adjusted associations between continuous measures of air pollution and CMHT events for each dementia subtype are shown in table 2. At Year 1, exposure to all air pollutants was associated with increased CMHT events for people with vascular dementia, with the strongest association observed for NO_2 (aIRR 1.30, 95% CI 1.10 to 1.55, $p=0.003$). Findings were less consistent for patients with Alzheimer's disease and other/unspecified dementia at this time point, with associations only emerging between particulate matter exposure and increased CMHT events (eg, Alzheimer's disease – $\text{PM}_{2.5}$: aIRR 1.08, 95% CI 1.02 to 1.14, $p=0.010$).

At Year 5 and Year 9, a more consistent pattern emerged where all air pollutants were associated with increased CMHT events for people with vascular dementia and Alzheimer's disease, but not for other/unspecified dementia. These associations were consistently strongest for people with vascular dementia at both time points (eg, Year 9, NO_2 – vascular dementia: aIRR 1.25, 95% CI 1.06 to 1.46, $p=0.007$; Alzheimer's disease: aIRR 1.16, 95% CI 1.06 to 1.28, $p=0.002$), although overlap between CIs indicates that these associations were not statistically significantly stronger.

Cognitive, health and social functioning

In terms of cognitive function, fully adjusted associations between air pollution exposure and average MMSE scores at short-, medium- and long-term time points are shown in table 3. There were no significant associations between any air pollutant and MMSE score at any time point.

Fully adjusted associations between air pollution exposure and average HoNOS65+ score at each time point are also shown in table 3. At all time points, exposure to NO_2 was associated with higher HoNOS65+ scores indicating poorer health and social functioning (eg, short-term follow-up: $\beta=1.14$, 95% CI 0.75 to 1.54, $p<0.001$). At short-term ($\beta=0.53$, 95% CI 0.19 to 0.86, $p=0.002$) and medium-term ($\beta=0.63$, 95% CI 0.25 to 1.01, $p=0.001$) follow-up periods, PM_{10} was also associated with HoNOS65+ scores, but this became non-significant in the long-term. $\text{PM}_{2.5}$ was not associated with HoNOS65+ scores.

As the greatest number of people (n=4706) had HoNOS65+ scores within Year 1, we assessed associations between NO_2 (the pollutant displaying strongest associations with overall scores)

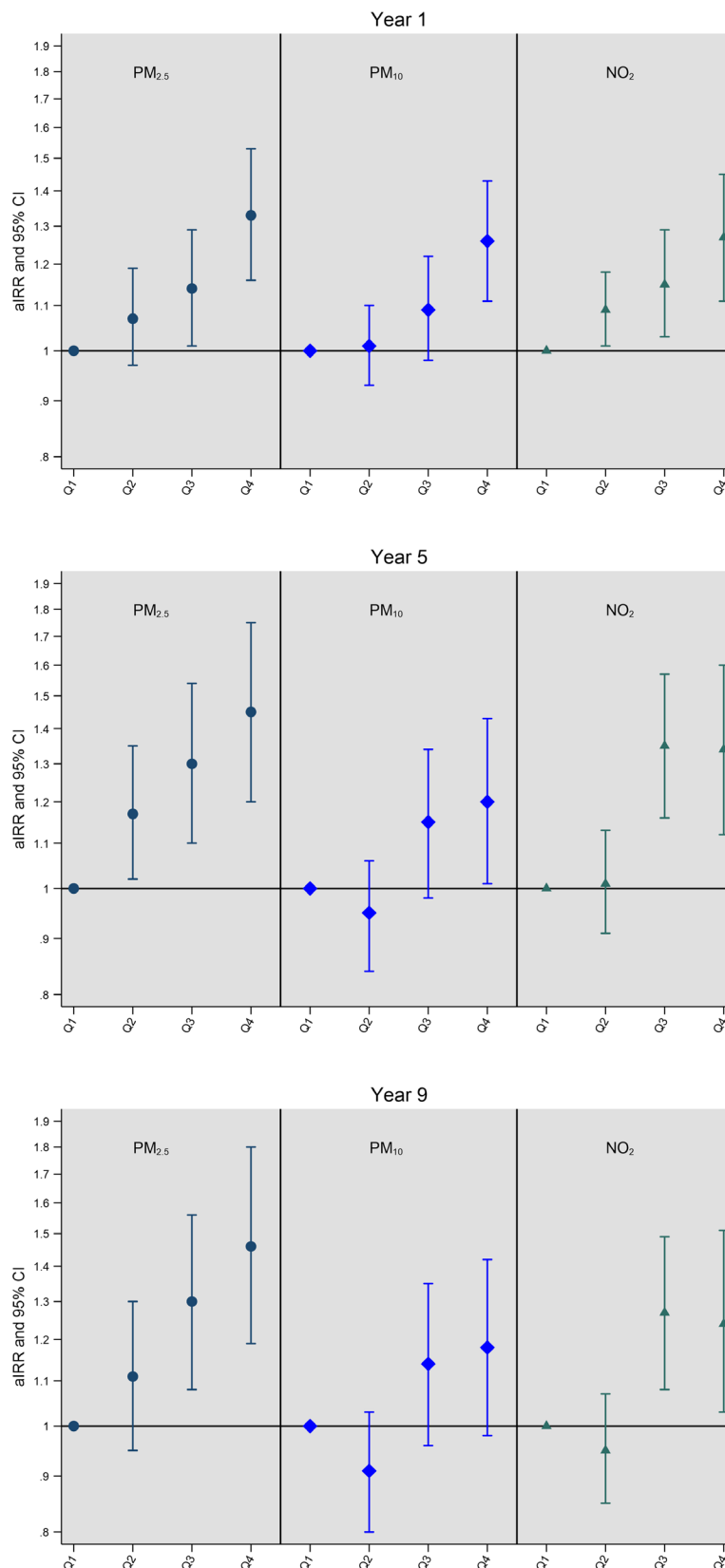


Figure 1 Adjusted incidence rate ratios (aIRR) and their corresponding 95% confidence intervals (CI) from standard negative binomial regressions looking at associations between exposure to air pollution (PM_{2.5}, PM₁₀, NO₂) and use of community mental health teams (number of events) at years 1, 5 and 9. Air pollutants (µg/m³) are categorised as quartiles to assess dose-response associations, with the lowest quartile (Q1) acting as the reference quartile. All models are adjusted for season, year, age, sex, ethnicity, marital status, number of comorbid mental health conditions, neighbourhood deprivation, social fragmentation and population density. The quartile cut-offs were: Q1 <12.55 µg/m³, Q2 <14.10 µg/m³, Q3 <15.18 µg/m³, Q4 ≥15.18 µg/m³ for PM_{2.5}; Q1 <18.20 µg/m³, Q2 <20.73 µg/m³, Q3 <24.07 µg/m³, Q4 ≥24.07 µg/m³ for PM₁₀; and Q1 <30.39 µg/m³, Q2 <37.60 µg/m³, Q3 <46.19 µg/m³, Q4 ≥46.19 µg/m³ for NO₂.

Table 2 Air pollution and community mental health team events by dementia subtype

	NO ₂		PM _{2.5}		PM ₁₀	
	aIRR (95% CI)	P value	aIRR (95% CI)	P value	aIRR (95% CI)	P value
1-year follow-up						
Alzheimer's	1.11 (1.00 to 1.23)	0.056	1.08 (1.02 to 1.14)	0.010	1.07 (0.98 to 1.17)	0.104
Vascular	1.30 (1.10 to 1.55)	0.003	1.19 (1.09 to 1.31)	<0.001	1.26 (1.09 to 1.46)	0.002
Other/unspecified	1.16 (0.99 to 1.35)	0.073	1.09 (1.00 to 1.19)	0.044	1.14 (1.00 to 1.31)	0.048
5-year follow-up						
Alzheimer's	1.19 (1.08 to 1.31)	0.001	1.08 (1.02 to 1.13)	0.006	1.12 (1.03 to 1.22)	0.006
Vascular	1.23 (1.05 to 1.45)	0.011	1.15 (1.05 to 1.25)	0.002	1.20 (1.04 to 1.37)	0.011
Other/unspecified	1.10 (0.94 to 1.27)	0.225	1.07 (0.99 to 1.16)	0.085	1.09 (0.97 to 1.24)	0.153
9-year follow-up						
Alzheimer's	1.16 (1.06 to 1.28)	0.002	1.07 (1.02 to 1.13)	0.008	1.11 (1.03 to 1.20)	0.010
Vascular	1.25 (1.06 to 1.46)	0.007	1.15 (1.06 to 1.26)	0.001	1.21 (1.06 to 1.39)	0.006
Other/unspecified	1.12 (0.96 to 1.30)	0.136	1.08 (0.99 to 1.17)	0.066	1.10 (0.97 to 1.25)	0.126

All air pollutant exposure estimates were rescaled to IQR increments – NO₂: mean=2.45, SD=0.65; PM_{2.5}: mean=5.50, SD=1.15; PM₁₀: mean=3.60, SD=0.73. Bold text indicates p<0.05.

Covariates: Season, year, age, gender, ethnicity, marital status, comorbid mental health conditions, neighbourhood deprivation, population density, social fragmentation. aIRR, fully adjusted incident rate ratio; NO₂, nitrogen dioxide; PM₁₀, particulate matter with a diameter of <10 µm; PM_{2.5}, particulate matter with a diameter of <2.5 µm.

and individual HoNOS65+ subscales at this time point. Adjusted beta coefficients and 95% CI are shown in figure 2. These indicate that exposure to higher levels of NO₂ was most strongly associated with aspects of poor physical (activities of daily living, physical illness) and social (problems with relationships and living conditions) functioning. Similar findings emerged for PM_{2.5} and PM₁₀ (see online supplemental table S4 and figure S2).

Sensitivity analysis

Results from the six sensitivity analyses are as follows:

1. We investigated differences between the analytical sample (n=5024) and those excluded from the study on the basis of missing data (n=1418) (online supplemental table S5). Excluded participants differed from the analytical sample on

Table 3 Associations between air pollution levels and the mean MMSE score and HoNOS65+ score taken at short-term (first face-to-face contact to 12 months), medium-term (Year 1–Year 5), and long-term (Year 5–Year 9) follow-up

Short-term				
	MMSE scores (n=2488)		HoNOS65+ scores (n=4076)	
	β (95% CI)	P value	β (95% CI)	P value
Mean±SD	19.0±5.3		10.7±4.3	
NO ₂	0.03 (–0.62 to 0.68)	0.930	1.14 (0.75 to 1.54)	<0.001
PM _{2.5}	–0.00 (–0.35 to 0.35)	0.998	0.17 (–0.04 to 0.39)	0.112
PM ₁₀	–0.23 (–0.78 to 0.31)	0.398	0.53 (0.19 to 0.86)	0.002
Medium-term				
	MMSE scores (n=1975)		HoNOS65+ scores (n=2808)	
	β (95% CI)	P value	β (95% CI)	P value
Mean±SD	19.1±5.1		10.5±4.1	
NO ₂	0.04 (–0.66 to 0.74)	0.908	1.13 (0.69 to 1.58)	<0.001
PM _{2.5}	–0.01 (–0.38 to 0.37)	0.961	0.22 (–0.02 to 0.46)	0.070
PM ₁₀	–0.14 (–0.73 to 0.44)	0.630	0.63 (0.25 to 1.01)	0.001
Long-term				
	MMSE scores (n=574)		HoNOS65+ scores (n=799)	
	β (95% CI)	P value	β (95% CI)	P value
Mean±SD	20.2±4.7		10.31±22.8	
NO ₂	–0.74 (–2.07 to 0.58)	0.269	1.41 (0.54 to 2.27)	0.001
PM _{2.5}	0.13 (–0.59 to 0.85)	0.717	0.03 (–0.43 to 0.49)	0.901
PM ₁₀	–0.19 (–1.28 to 0.90)	0.729	0.57 (–0.13 to 1.27)	0.110

Bold text indicates p<0.05.

Covariates: Season, year, age, gender, ethnicity, marital status, comorbid mental health conditions, neighbourhood deprivation, population density, social fragmentation. HoNOS, Health of the Nations Outcome Scale; MMSE, Mini Mental State Examination; NO₂, nitrogen dioxide; PM₁₀, particulate matter with a diameter of <10 µm; PM_{2.5}, particulate matter with a diameter of <2.5 µm.

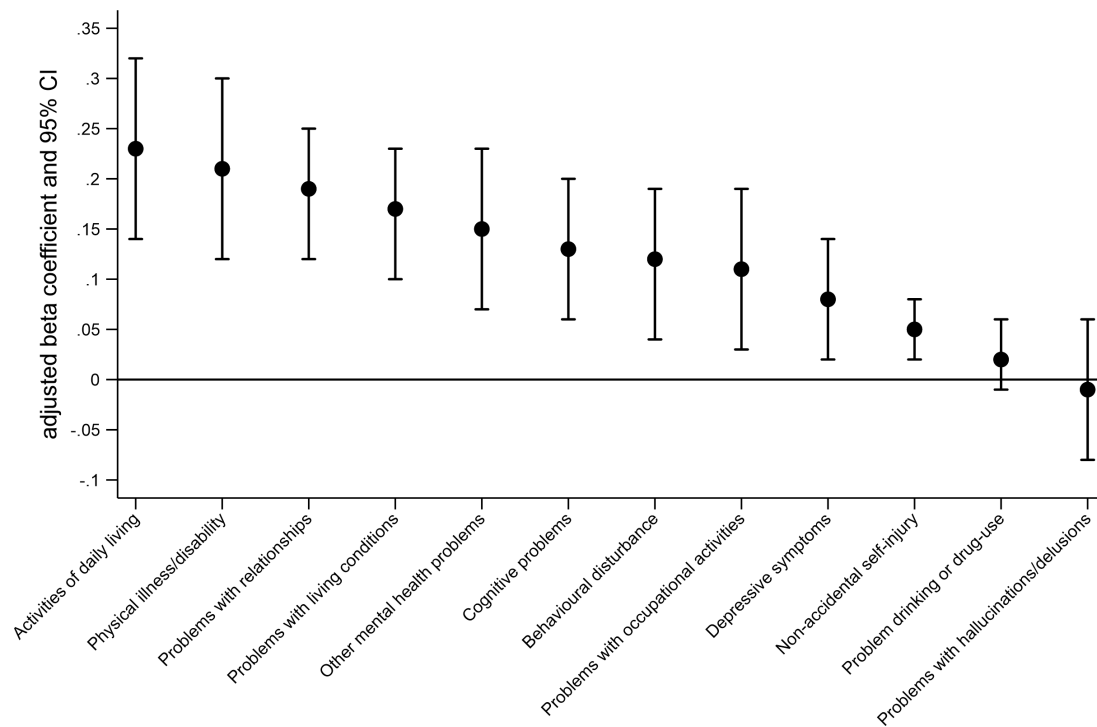


Figure 2 Adjusted beta coefficients and their corresponding 95% confidence intervals (CI) from linear regressions examining associations between NO_2 exposure and scores on individual HoNOS65+ subscales within 12 months of first face-to-face contact with SLaM services for people with dementia. All models are adjusted for season, year, age, sex, ethnicity, marital status, number of comorbid mental health conditions, neighbourhood deprivation, social fragmentation and population density. HoNOS65+, Health of the Nations Outcome Scale; NO_2 , nitrogen dioxide; SLaM, South London and Maudsley National Health Service (NHS) Foundation Trust.

a number of sociodemographic factors, had higher numbers of CMHT events, and were exposed to higher levels of NO_2 , but not particulate matter.

- Results following multiple imputation were mostly consistent with the original complete case associations (see online supplemental table S6). At Year 5, associations between PM_{10} and CMHT events became non-significant. At Year 9, NO_2 levels were no longer significantly associated with CMHT events, but associations with $\text{PM}_{2.5}$ were preserved.
- There was no evidence that associations were modified by neighbourhood deprivation (online supplemental table S7).
- There was some evidence that associations were modified by residential borough (see online supplemental table S8). We stratified fully adjusted analyses (Model 3) by borough (see online supplemental table S9) and found that air pollution was associated with CMHT events in all boroughs except Southwark. Associations were most consistent in Croydon.
- Two-pollutant models brought about some changes in the strength and magnitude of some associations. Online supplemental table S10 shows two-way exposure regression models where each air pollutant was adjusted for in turn to assess potential mutual confounding, which was due to collinear associations between the pollutants (see online supplemental table S11).
- The inclusion of LSOA as a random intercept in fully adjusted models did not affect the direction or significance of associations between air pollutants and CMHT events, but slightly attenuated the strength of these associations (see online supplemental table S12).

DISCUSSION

Statement of principal findings

In a retrospective cohort of 5024 older adults with dementia we found that long-term exposure to higher levels of air pollution was associated with more frequent use of CMHT in the months and years following first face-to-face contact with secondary mental health services. These results were particularly consistent for $\text{PM}_{2.5}$ and NO_2 , even after adjustment for a wide range of individual-level and area-level confounders. At all time points, associations were strongest for those with vascular dementia, although not significantly so. Using routinely collected measures showed that air pollution was not associated with cognitive functioning (MMSE scores), but NO_2 exposure levels were repeatedly related to health and social functioning (HoNOS65+ scores) over the months and years of follow-up. Looking at each of the HoNOS65+ domains separately showed that NO_2 was associated with worse scores on almost all aspects of functioning, and this was strongest for functional status (activities of daily living), physical illness and disability, and problems with relationships.

Strengths and weaknesses of the study

We used a state-of-the-art air quality model to estimate address-level exposure to NO_2 , $\text{PM}_{2.5}$ and PM_{10} in four South London boroughs linked to comprehensive electronic health records which allowed for mental health service use to be measured over 9 years of follow-up. Furthermore, use of electronic health records from secondary mental healthcare settings where misdiagnosis of dementia would perhaps be less likely than in an emergency admission to a general hospital means that misclassification bias was unlikely.²⁷ A further strength includes steps to address other potential biases. We included comprehensive individual-level

and area-level covariates in fully adjusted models, calculated e-values to assess the impact of unmeasured confounding, and repeated the analysis on an imputed dataset to assess bias introduced by missing data. Moreover, as well as examining associations between air pollution and CMHT events, we sought to unpick results further by examining cognitive as well as health and social functioning.

There are several limitations that we must acknowledge. First, due to the observational nature of the data, causal inferences could not be made and residual confounding was inevitable. However, our confidence in the associations we report was strengthened by relatively large e-values. It is likely that the results of this study would generalise to the whole of London and other large cities in high-income countries with heavy diesel traffic. However, these findings might not be applicable to rural settings. Moreover, they may not be applicable to low- and middle-income countries (LMICs) where air pollution levels are often considerably higher in urban settings than in high-income countries (<https://www.stateofglobalair.org/>), but the increased air pollution burden might mean the impact on resource use in dementia is even more pronounced in LMICs. As air pollution was measured between 2008 and 2012, we were unable to assess the impact of exposure to pollutants earlier in life, as well as the impact of fluctuations in air pollution exposure over the 9-year follow-up period. Moreover, address-level measures of air pollution meant that we could not account for changes in exposure due to residential mobility or time away from home. Unfortunately, we were unable to look at associations between air pollution and CMHT events in participants who were non-movers (ie, remained at the same residential address over the 5-year and 9-year follow-up period) as very few remained at their residential addresses over the study period. This is not surprising in a cohort of elderly people with dementia who would be likely to move to residential care or pass away over the course of the study. There was also a considerable possibility of clustering effects due to people with dementia in the current study residing in the same care home. However, in the current study we were unable to account for this clustering as we could not access information about care home residency. Instead, we performed a sensitivity analysis which took into account clustering according to LSOA, which did not notably affect associations between air pollution and CMHT events.

Strengths and weaknesses in relation to other studies, discussing important differences in results

The results of this study extend those of previous studies which have reported links between air pollution and increased dementia-related hospital admissions,^{8–11 28} suggesting that air pollution might also impact use of dementia-related care embedded in psychiatric services (ie, use of CMHTs). In the current study, stronger associations for patients with vascular dementia imply that air pollution might impact trajectories of vascular dementia more so than other dementia types. This is supported by data from Italy showing links between air pollution and hospitalisations for vascular dementia, but not Alzheimer's disease.⁹ In reviewing the evidence that air pollution was related to dementia, the UK COMEAP concluded that it was plausible, based on the pre-existing evidence of associations with cardiovascular and cerebrovascular disease, that there was a causal pathway to vascular dementia.⁷ It was felt more work was required to establish links to Alzheimer's disease and other dementias. Our findings are in line with this notion, implying that air pollution might exacerbate vascular dementia more

strongly than other dementia types, leading to more CMHT use. It may be that air pollution increases use of CMHTs in vascular dementia through the impact of air pollution on physical functioning and health, and there are already established links between frailty and this type of dementia.²⁹ More work is needed to elucidate mechanisms.

Meaning of the study: possible explanations and implications for clinicians and policymakers

The links between NO₂ exposure and poorer functional status in the current study suggest that changes in health and social functioning—more so than cognitive decline—might somewhat mediate the relationship between traffic-related air pollution (NO₂ representing a good proxy of diesel exhaust emissions) and increased usage of CMHTs in people with dementia. However, it is noteworthy that all air pollutants were associated with higher scores on the cognitive problems subscale of the HoNOS65+, indicating worse cognitive function. This might indicate that the MMSE was not the best measure of cognitive function in this instance. There are known links between air pollution and both frailty³⁰ and multimorbidity² in older adults, as well as disability-adjusted life years.³¹ It is possible that air pollution exposure might further exacerbate poor physical health, as well as social functioning, in older people with dementia, leading to increased health service use. There are several pathways through which air pollution might affect physical health and functional status. Exposure to PM_{2.5}, NO₂ or the primary combustion aerosol represented by modelled NO₂ concentrations has been associated with increased systemic oxidative stress,³² inflammation,³³ immune activation,³⁴ as well as negative respiratory³⁵ and cardiovascular³⁶ effects, thereby contributing to poor health and disability. PM_{2.5} exposure was not associated with HoNOS65+ total scores in the current study, despite being associated with increased CMHT events. This implies that exposure to this type of air pollution might impact dementia-related health service use via different pathways. The results of the current study imply that traffic-related pollution (ie, NO₂) can exacerbate aspects of negative health and social functioning in people with dementia, whereas non-exhaust emissions (eg, domestic and industrial combustion) like PM_{2.5} have less impact on this outcome. Further work should seek to elucidate the differential effects of air pollutant types on health service use in dementia. In the current study, associations between PM₁₀ and CMHT events were not as strong and did not persist for as long as the associations seen with NO₂ and PM_{2.5}. This is likely due to the larger particle size and is in line with previous research which has shown that particles smaller than 2.5 μm in diameter have the most pronounced effects on health and health outcomes.³⁷

Based on the evidence presented, we contend that air pollution could be considered an important population-level target to reduce mental health service use in people with dementia, particularly for those with vascular dementia. The reduction in air pollution and particularly NO₂ through public health interventions such as the expansion of ultra-low emission zones could potentially improve functioning and disease trajectories for people with dementia. Moreover, reducing air pollution levels might reduce CMHT demand for people with dementia. We calculated that reducing NO₂ and PM_{2.5} in London to WHO recommended thresholds could lead to a 38% and 13% reduction in dementia-related CMHT events, respectively.

Unanswered questions and future research

Future research should ascertain whether these findings extend to other types of dementia-specific health service use such as

Memory Services and multidisciplinary dementia services based in primary care. More work is also needed to understand why people with dementia exposed to higher levels of air pollution are using mental health services more. The results of this study suggest that it might be to do with poor functional status, physical illness and relationship difficulties, but more work is needed to elucidate pathways between air pollution and increased mental health service use. Multimorbidity is common in dementia, and is also associated with increased health service use.³⁸ Uncovering which comorbid conditions are most likely to drive increased mental health service use might shed some light on the involvement of air pollution.

CONCLUSIONS

In this study we have shown clear associations between air pollution exposure and CMHT events that persist over time in people with dementia. These associations were strongest for patients with vascular dementia. The lack of association between air pollution and MMSE scores indicates that declines in cognitive function might not play a role in the observed increase in CMHT events, but more work is needed to understand the role of cognitive function in this pathway. Links between NO₂ exposure and HoNOS65+ scores indicate that increased mental health service use might be somewhat related to the effects of NO₂ on functional decline. However, the pathway through which PM_{2.5} impacts CMHT usage is not clear. PAFs indicate that reducing levels of air pollution could lead to a considerable reduction in CMHT events for people with dementia in South London as well as other urban traffic areas in the UK. However, more work is needed to understand how air pollution impacts other aspects of dementia-related care, as well as the pathways involved.

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