**Supplementary Material**

**Air Pollution and Dementia: A Systematic Review**

**Search Strategy and Methods**

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Database: Embase from 1974, Ovid MEDLINE(R) In-Process & Other Non-Indexed Citations and Ovid MEDLINE(R) from 1946 and PsycINFO from1806: to 20 September 2018

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Alzheimer Disease or cognitive decline or cognitive impairment or Dementia or cognition disorders or Cognition Disorders or cognitive function or dementia or Dementia, Vascular or Dementia, Multi-Infarct or alzheimer disease or alzheimers disease or Alzheimers disease or dementia vascular.mp. or vascular dementia or multi infarct dementia

and

air pollution or Air Pollution or particulate matter or Particulate Matter or exp Air Pollutants or PM10 or Particle Size or PM\* or nitrogen dioxide or Nitrogen Dioxide or nitrates or Nitrates or nitric oxide or Nitric Oxide or ozone or Ozone or "03" or O3 or ultrafine particulate matter or UFPM or Vehicle Emissions or diesel or vehicle emissions or Environmental Exposure or environmental exposure or road proximity or traffic or Sulfur Dioxide or sulphur dioxide

limit to English language, human and adulthood (≥18 years)

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| **Supplementary Table 1.**Exposure and outcome measures | | | | | |  |  | |
| **Authors** | **Primary years of exposure** | **Pollutants** | | **Exposure measures** | |  | **Cognitive outcome measures** | |
| Weuve et al., 2012 [1] | 1995-2008 | PM2.5 | | Averaged month-specific exposure over five intervals (1 month, 1, 2, 5 y and from 1988 through preceding month) preceding cognitive interview. Derived from EPA's AQS meteorological data and GIS smoothing models for geocoded residential location per participant. PM2.5-10 calculated as difference between PM2.5 and PM10.  1-month average: exposure 1 month preceding baseline 1-y average: exposure 1 y preceding baseline 2-y average: exposure 2 y baseline 5-y average: exposure 5 y baseline Average since 1988: long-term exposure (1988-baseline) | | Quintiles, highest to lowest Q5: 16.9-25.5 μg/m3  Q4: 15.1-16.8 μg/m3 Q3: 13.3-15.0 μg/m3 3 Q2: 11.6-13.2 μg/m3  Q1: 1.9-11.5 μg/m3  1-month average: 12.5 µg/m3 (*SD*=3.7; range: 2.1-33.7 µg/m3) 1-y average: 12.7 µg/m3 (*SD*=2.8; range: 2.3-23.9 µg/m3) 2-y average: 13.1 µg/m3 (*SD*=2.8; range:2.1-24.8 µg/m3) 5-y average: 13.1 µg/m3 (*SD*=2.8; range: 1.9-24.0 µg/m3) Average since 1988: 14.2 µg/m3 (*SD*=3.0; range: 19.2-25.5 µg/m3) | Global score s created by averaging z-scores from six tests, and individuals z-scores on each (TICS 10-word list, EBMT, immediate recall, delayed recall, Digit Span Backward test, category fluency)  Note. tests added to the initial TICS when participation in cognitive interviews became apparent and thus sample for each test varied | |
|  |  | PM2.5-10 | |  | | Quintiles, highest to lowest Q5: 11.9-50.2 μg/m3 Q4: 9.6-11.8 μg/m3 Q3: 7.9-9.5 μg/m3 Q2: 6.7-7.8 μg/m3 Q1: 1.1-6.6 μg/m3  1-month average: 8.6 µg/m3 (*SD*=5.2; range: -4.0-69.0 µg/m3) 1-y average: 8.3 µg/m3 (*SD*=4.1; range: -0.2-56.1 µg/m3) 2-y average: 8.2 µg/m3 (*SD*=3.9; range: 0.1-52.2 µg/m3) 5-y average: 8.5 µg/m3 (*SD*=3.8; range: 0.0-48.8 µg/m3) Average since 1988: 9.6 µg/m3 (*SD*=4.1; range: 1.0-50.2 µg/m3) |  | |
| Loop et al., 2013 [2] | 2003-2009 | PM2.5 | | Annual average exposure using an algorithm combining EPA's AQS ground level monitoring data and NASA's MODIS aerosol optical depth satellite data to calculate daily PM2.5 exposure per participant according to residence up to and including the date of the baseline visit. | |  | Six Item Screening telephone assessment (3-item recall and orientation in time) cognitively intact: scores ≥5/6; incident cognitive impairment: scores ≤4 | |
| Tonne et al., 2014 [3] | 2002-2009 (5-y window in this time frame) | PM2.5 | | Annual average concentration for years 2003-2009 modelled at resolution 20 x 20 m using the KCLurban dispersion modelling system which incorporates meteorological data, empirically derived NO-NO2-O3 and PM relationships and emission from the London Atmospheric Emission Inventory. Exposure at residence based on average concentration at model grid points within 25 m of the postcode center. Averaging periods: 1-y average (concentration during year of the 2007-2009 assessment) yearly lag 0 1-4-y lag (i.e., 1-4-y prior to 2007-2009 assessment) 3-y average (average concentration of 3 years prior to 2007-2009 assessment) 5-y average (i.e., 1-y average plus 4 preceding year of assessment in 2002-2009) | | 5-y average: 14.9 µg/m3 (*SD*=0.9; IQR: 1.1 µg/m3) 4-y lag: 16.2 µg/m3 (*SD*=1.4; IQR: 1.3 µg/m+Q83) | Tests of reasoning (Alice Hein 1-I test) Short term verbal memory (20-word free-recall test) Semantic verbal fluency Phonemic verbal fluency  Note. good test-retest reliability (range 0.60-0.89) | |
|  |  | PM2.5 from traffic exhaust only | | 5-y average: 0.64 µg/m3 (*SD*=0.25; IQR: 0.27 µg/m3)  4-y lag: 0.81 µg/m3 (*SD*=0.32; IQR: 0.34 µg/m3) |  | |
|  |  | PM10 | | 5-y average: 23.4 µg/m3 (*SD*=1.5; IQR: 1.8 µg/m3) 4-y lag: 24.7 µg/m3 (*SD*=1.9; IQR: 2.2 µg/m3) |  | |
|  |  | PM10 from traffic exhaust only | | 5-y average: 0.72 µg/m3 (*SD*=0.27; IQR: 0.3 0µg/m3) 4-y lag: 0.91 µg/m3 (*SD*=0.36; IQR: 0.38 µg/m3) |  | |
| Carey et al., 2018 [4] | 2004-2010 | NO2, O3, PM2.5, distance from a major roadway (based on central point of postcode area) | Modelled annual concentrations of air pollutants in the year prior to baseline estimated using the KCLurban dispersion modelling system at a resolution of 20\*20 m incorporating hourly meteorological measurements and empirically derived concentrations on emissions from the London Atmospheric Emissions Inventory | | NO2 Mean 37.1 μg/m3 (SD5.7) O3, Mean 38.0 μg/m3 (SD 3.9) PM2.5 Mean 15.7 μg/m3 (SD 0.8) | | | Incident dementia from general practice and healthcare records | |
| Chen et al., 2017 [5] | 1994-2012, 7-y lag | PM2.5 | | Annual mean concentration of PM2.5 (1 x 1 km) yearly between 1998 and 2012. Derived from satellite data, global atmospheric chemistry transport model (GEOS-Chem CTM) outputs, and calibrated with land cover, elevation, aerosol composition information using geographically-weighted regression. Postal code represented centroids or blocks or residence. | | 5-y cumulative exposure with 2-y lag: 10.4 µg/m3 (range: 1.1-49.7 µg/m3; IQR: 4.8 µg/m3) | Incident dementia   Note. validated algorithm applied to health insurance database. Cases defined as dementia related hospital admission, physician claims, prescriptions | |
|  |  | NO2 | | Annual measurement of NO2 in 2006.Developed National land-use regression model which included observations at fixed site monitors from the national air pollution surveillance network, satellite data, industrial land use, distance decay gradient. Postal code represented centroids or blocks or residence. | | 5-y cumulative exposure with 2-y lag: 16.2 ppb (range: 2.4-65.3 ppb; IQR: 14.2 ppb or 26.7 µg/m3) |  | |
|  |  | O3 | | Long-term annual mean of O3. Derived using optimal interpolation technique combining true observations with benefits of physically based air quality prediction models that account for meteorological and chemical patterns. Postal code represented centroids or blocks or residence. | | 5-y cumulative exposure with 2-y lag: 45.8 ppb (range: 23.3-58.9 ppb; IQR: 6.3 ppb or 12.4 µg/m3) |  | |
| Cleary et al., 2018 [6] | 2004-2008, 1-y lag | PM2.5 | | Annual mean concentrations derived daily 24-hour PM2.5 concentrations in µg/m3 starting year before baseline. EPA’s hierarchical Bayesian model data derived from ground-level monitoring data from the AQS and simulated ozone and from the CMAQ model (estimates available in 12x12 m resolution covering eastern states and 24 x 24 m nationwide) used. Yearly exposure estimates based on to ZIP codes of residence, or via interpolation. | | Annual mean concentrations (2004-2008): 9.7 µg/m3 (range: 3.8-14.4 µg/m3)  tertiles:  low <9.1 µg/m3 medium 9.1-10.6 µg/m3 high >10.6 µg/m3 | MMSE (<24 considered cognitively intact) CDR-SB to assess cognitive trajectory | |
|  |  | O3 | | Annual mean concentrations derived from daily 8-hour maximum ozone concentration in ppb starting year before baseline. EPA’s hierarchical Bayesian model data derived from ground-level monitoring data from the AQS and simulated ozone and from the CMAQ model (estimates available in 12x12 m resolution covering eastern states and 24 x 24 m nationwide) used. Yearly exposure estimates based on to ZIP codes of residence, or via interpolation. | | Annual mean concentrations (2004-2008): 37.8 ppb (range: 30.4-47.5 ppb)  tertiles: low <36.7 ppb medium 36.7-40 ppb high >40 ppb |  | |
| Chen et al., 2017 [7] | 1996-2012, 5-y lag | Residential proximity to a major roadway or highway | | Exposure based on residential proximity to a major roadway or highway based on postcode in 1996 (5-y lag). Median distance (continuous) and five categories of distance calculated using ArcGIS: <50 m, 50-100 m, 101-200 m, 201-300 m, >300 m.   Note. Pollutant concentration (4-y average) was analyzed a covariate (calculated as exposure 1988-2001, at 1 x 1 km spatial resolution covering all North America below 70°N, derived from satellite observations of aerosol optical depth in combination with outputs from a global atmospheric chemistry transport model and adjusted for urban land cover, elevation and aerosol composition using geographically weighted regression) | | Average concentration PM2.5 5-y lag: 9.7 μg/m3 (1.3-19.8 μg/m3)  Average concentration NO2 5-y lag: 15.4 ppb (2.2-62.0 ppb) | Incident diagnoses of dementia   Note. databases validated with chart-review, with 78-84% sensitivity and 99-100%; created using hospital discharge abstracts, physician and prescription medication claims. | |
| Oudin et al., 2016 [8] | 1993-1995 | NO2 | | Mean annual NO2 concentrations estimated with developed land-use regression model derived from 4 weeklong measurements obtained from November 2009 to June 2010, at 36 sites around Umea. Concentration grid of 50 x 50 m created using geocoded from baseline addresses obtained from the Swedish Population Register. | | Quartiles, highest to lowest Q4: ≥26.0 μg/m3 Q3: 17.0.3-26.0 μg/m3 3 Q2: 9.0-16.9 μg/m3  Q1: <9.0 μg/m3 | Incident dementia diagnoses    Note. from medical records data documenting assessment for dementia every 5 y using the DSM-IV, hospital and primary care visits, MRI, computerized topography scans record, in neuropsychological evaluation for participants who scored MMSE≤23, showed cognitive of function decline between tests phases | |
| Jung et al., 2015 [9] |  | PM2.5 | | Taiwan data on PM2.5 only available after 2006, hence this was extrapolated backwards using the mean ratio between PM2.5 and PM10 during 2006-2010. Annual average PM10 according to guidance from USA EPA. Data from 70 EPA sites across Taiwan at postcode level, interpolated using inverse distance weight method. | | Mean annual average of PM2.5 concentration, during 2006-2010: 33.56 μg/m3 (*SD*=9.20; range 10.36-61.76) Baseline PM2.5 IQR: 13.21-4.34 μg/m3 | Incident AD (newly diagnosed)  Note. validated database diagnosis based on ICD-9-CM; incident AD defined as individuals who had received at least 2 consensus diagnosis between baseline and follow-up, which is assigned by physician based on history, physical examination, laboratory and imaging investigations, MMSE, DSM-IV and NINCDS-ADRDA, or Hachinski ischemic scores | |
|  |  | O3 | | Annual fourth-highest daily maximum 8-hour average based on 8-h standard of Ozone. Data from 70 EPA sites across Taiwan at postcode level, interpolated using inverse distance weight method. | | Mean annual 4th daily maximum 8-hour average of O3: concentration: 92.64 ppb (*SD* =13.47); range: 34.75-137.65 ppb)  baseline IQR: 9.63-10.91ppb |  | |
| Chang et al., 2014 [10] | 2000 - ? | NO2 | | Annual average concentration per residential district containing the clinic where there people most frequently sought treatment for acute upper respiratory tract infection based on daily measures of NO2 and CO from 74 air quality monitoring stations. Data were from between 1998 to 2010.  Yearly average concentrations calculated per patient from baseline to date of dementia diagnosis, withdrawal or end of study. Exposure categorized into quartiles | | Quartiles, highest to lowest Q4: >9825.5 ppb Q3: 8349.1-9825.5 ppb Q2: 6652.3-8349.0 ppb  Q1: < 6652.3 ppb | Incident dementia   Note. validated database diagnosis based on ICD-9-CM | |
|  |  | CO | |  | | Quartiles, highest to lowest Q4: >296.9 ppb Q3: 241.7-296.9 ppb Q2: 196.2-241.6 ppb  Q1: <196.2 ppb |  | |
| Cacciottolo et al., 2017 [11] | 1999-2010 | PM2.5 | Yearly time series of PM2.5 exposure generated from statistically validated BME estimates applied to geocoded residential location and combined with residential histories to calculate the 3-y moving average exposure. BME method used to construct spatiotemporal models to estimate ambient concentrations of PM2.5 which integrates nationwide monitoring data from the US EPA AQS and output of chemical transport models to characterize spatiotemporal interdependence of environmental data to estimate mean trends and covariance of the air pollution field over space and time. | | Increased time-varying PM2.5  high 3-y average: >12µg/m3  low 3-y average: ≤12µg/m3 | | | Incident accelerated decline in global cognitive function (operationally defined as having an 8-point loss in the Modified MMSE in two consecutive assessments)  Note. global cognitive function, neuropsychological and functional assessment, and collection of clinical data to rule out possible reversible causes of cognitive impairment | |
|  |  | APOE x PM2.5 |  | | Effect by APOE status ε3/3 ε3/4  ε4/4 | | |  | |
|  |  | PM2.5 |  | | Increased time-varying PM2.5  high 3-y average: >12 µg/m3  low 3-y average: ≤12 µg/m3 | | | incident all-cause dementia   Note. global cognitive function, neuropsychological and functional assessment, and collection of clinical data to rule out possible reversible causes of cognitive impairment | |
|  |  | APOE x PM2.5 |  | | Effect by APOE status ε3/3 ε3/4  ε4/4 | | |  | |
| Oudin et al., 2017 [12] | 1988-2010 | NOX | Land Use Regression model used to estimate the annual mean of NOx at each geocoded address, utilizing data from 40 monitoring sites that represented a wide range of traffic conditions in residential, industrial, commercial and rural locations. | | Mean level of NOX: 20.9µg/m3 (*SD=*16.1µg/m3)Quartiles, highest to lowest Q4: 24.0 µg/m3  Q3: 15.4 µg/m3  Q2: 8.4 µg/m3  Q1: reference | | | Episodic memory measure (EMM) (consisting of immediate free recall and delayed cured recall task) | |
| Oudin et al., 2018 [13] | 1988-2010 | PM2.5 from traffic exhaust | | Used annual mean concentration of PM2.5 for 1990, 2000, 20210 calculated by SMHI which estimated concentrations using a wind model and a Gaussian air quality dispersion model. Traffic flow for vehicles collected for most major roads and modelled for elsewhere. Vehicle fleet composition derived from national vehicle registry, and emission factors for exhaust calculated based on the Handbook Emission Factors for Road Transport. Model grids were of 3200 m x3200 m spatial resolution and 50 m x 50 m in urban areas. | | Mean annual average of PM2.5 concentration: 0.18 µg/m3 (*SD*=0.17 µg/m3)  Quartiles, highest to lowest Q4: 0.24 1.81 μg/m3 Q3: 0.14 0.24 μg/m3 Q2: 0.086-0.14 μg/m3 Q1: reference: 0.017-0.086 μg/m3  per 1 μg/m3 increase in exposure | Incident dementia  Note. Blinded re-evaluation was made of medical records of those with stablished dementia diagnosis, DSM-IV diagnosis, supplemented with medical record data | |
|  |  | PM2.5 from residential wood burning | | SMHI used detailed emission inventory according to chimney sweepers allowing point-source representation of emissions, which was validated through a monitoring campaign and survey regarding wood consumptions and firing habits. In the study area emissions were dominated by wood burning. Model grids were of 3200m x3200m spatial resolution and 50m x 50m in urban areas. | | Mean annual average of PM2.5 concentration: 0.77 µg/m3 (*SD*=0.30 µg/m3)  Quartiles, highest to lowest Q4: 0.91 3.34 μg/m3 Q3: 0.72-0.91 μg/m3 Q2: 0.54-0.72 μg/m3 Q1: reference: 0.21-0.54 μg/m3  per 1 μg/m3 increase in exposure |  | |
| AD, Alzheimer's disease; VaD, Vascular dementia; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders 4th Edition, EBMT, East Boston Memory Test; ICD-9-CM, International Classification of Diseases, 9th revision, Clinical Modification; MMSE, Mini-Mental Status Examination; NINCDS-ADRDA, National Institute of Neurological and Communicative Disorders and Stroke-Alzheimer's Disease and Related Disorders Association; TICS, Telephone Interview for Cognitive Status; CO, carbon monoxide NO2, nitrogen dioxide; O3, ozone; PM2.5, particulate matter ≤ 2.5 µm in diameter; PM10, particulate matter ≤ 10 µm in diameter; SO2, sulphur dioxide; ppb, parts per billion, y; year; AQS, Air Quality System; CMAQ, Community Multi-Scale Air Quality; EPA, Environmental Protection Agency; GIS, geographic information system; NASA, National Aeronautics and Space Administration; MODIS, Moderate Resolution Imaging Spectroradiometer; SMHI, Swedish meteorological and hydrological institute; IQR, interquartile range; Q, quintile; SD, standard deviation. | | | | | | | | |

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| **Supplementary Table 2.** Analysis and results | |  |  |  |
| **Authors** | **Analysis** | **Adjustments** | **Pollutants** | **Results** |
| Weuve et al., 2012 [1] | Generalized estimating equations regression models, which included time since baseline assessment (continuous), exposure, exposure x time interaction to compared trajectories in cognitive function over three repeated measures across exposure levels. Separate analysis for each PM measure, exposures as quintiles and continuous variables. Sensitivity analyses restricted to women who did not move between 1988 and first assessment (62%). | age, cognitive assessment, education, husband's education, long-term physical activity, long-term alcohol consumption, time x covariate interactions (BMI, diabetes, smoking, aspirin use, ibuprofen use adjustments had no effect) Further adjustment in secondary analyses found similar results. Secondary analyses: SES measures (percentage of adults who have less than high school education, median home value, median income) Additional analyses: self-reported emphysema and indicators of cardiovascular and cerebrovascular disease (high blood pressure, coronary heart disease, congestive heart failure, coronary artery bypass graft, transient ischemic attack, carotid endarterectomy) | PM2.5 | Adjusted difference in 2-y change in global cognitive z-scores per quintile of exposure Q5: highest exposure: -0.018 (-0.034, -0.002)\* Q4: -0.003 (-0.013, 0.019) Q3: -0.006 (-0.022, 0.010) Q2: -0.004 (-0.020, 0.012) Q1: comparator, 0  Adjusted difference in 2-y change in global cognitive score z-scores per 10 µg/m3 increase 1 month: -0.002 (-0.016, 0.012) 1-y: -0.016 (-0.034, 0.003) 2 y: -0.015 (-0.034, 0.003) 5 y: -0.020 (-0.038, 0.002) long-term (since 1988): -0.018 (-0.035, -0.002)\*  Sensitivity and secondary analyses did not materially affect results. |
|  |  |  | PM2.5-10 | Adjusted difference in 2-y change in global cognitive z-scores per quintile of exposure Q5: highest exposure: -0.024 (-0.040, -0.008)\* Q4: -0.004 (-0.020, 0.012) Q3: -0.013 (-0.030, 0.003) Q2: -0.006 (-0.022, 0.010) Q1: comparator, 0  Adjusted difference in 2-y change in global cognitive score z-scores per 10µg/m3 increase  1-month: -0.007 (-0.017, 0.003)  1-y: -0.017 (-0.029, -0.005)\* 2-y: -0.016 (-0.029, -0.003)\* 5 y: -0.019 (-0.032, -0.006)\* Long-term (since 1988): -0.020 (-0.032, -0.008)\*  Sensitivity and secondary analyses did not materially affect results. |
| Loop et al., 2013 [2] | Logistic regression modelling. Four models fitted and tested:  1. PM2.5, length of follow-up, confounders temperature, season, incident stroke  2. Model 1 + demographics 3. Models 1-2 + behavioral factors 4. Models 1-3 + known comorbidities of cognitive impairment Post-hoc sensitivity analysis where cognitive impairment defined as impairment on two last assessments. | Length of follow up, temperature, season, incident stroke, age, race, region, education, income, behavioral factors (alcohol, smoking, exercise, body mass index), depression, dyslipidemia, diabetes, hypertension. | PM2.5 | Effect of 10 µg/m3 increase in PM2.5 Model 1: OR =1.26 (0.97, 1.64) Model 2: OR =1.02 (0.76, 1.37) Model 3: OR =0.97 (0.72, 1.31) Model 4: OR =0.98 (0.72, 1.34)  Sensitivity analysis - exposure >12 months, n=18180 Model 1: OR =1.02 (0.61, 1.70) Model 2: OR =0.75 (0.42, 1.33) Model 3: OR =0.72 (0.39, 1.30) Model 4: OR =0.71 (0.38, 1.32) |
| Tonne et al., 2014 [3] | Cognitive test scores were converted to z scores and standardized using distribution of that wave. Linear mixed models used to evaluate relationship between cognitive change and pollutant exposure. Data was re-analyzed excluding the 213 participants who moved out of London between study waves. | Age, sex, ethnicity, marital status, education, SES (civil service employment grade), alcohol use, physical activity, time, age x time interaction, main effects of exposures. | PM2.5 | Cognitive change on reasoning, memory, semantic and phonemic fluency per IQR increase 5-y average: ns for all tests 4-y lag: ns for all tests  Re-analyses excluding participants who relocated: Mean change in memory per IQR increase 5-y average: ns 4-y lag: -0.041 (-0.079, -0.003)\* Mean change on reasoning, memory, semantic and phonemic fluency per IQR increase 5-y average: ns for all tests 4-y lag: ns for all tests |
|  | PM2.5 from traffic exhaust only | Cognitive change on reasoning, memory, semantic and phonemic fluency per IQR increase 5-y average: ns for all tests 4-y lag: ns for all tests |
|  | PM10 | Cognitive change on reasoning, memory, semantic and phonemic fluency per IQR increase 5-y average: ns for all tests 4-y lag: ns for all tests  Re-analyses excluding participants who relocated: Mean change in memory per IQR increase 5-y average: ns 4-y lag: -0.039 (-0.073, -0.005)\* Mean change on reasoning, memory, semantic and phonemic fluency per IQR increase 5-y average: ns for all tests 4-y lag: ns for all tests |
|  | PM10 from traffic exhaust only | Cognitive change on reasoning, memory, semantic and phonemic fluency per IQR increase 5-y average: ns for all tests 4-y lag: ns for all tests |
| Carey et al., 2018 [4] | Cox proportional Hazard regression analysis per IQR increase in pollutant. Four models with increasing levels of adjustments. | age, sex, ethnicity, smoking, body mass index, Index of Multiple Deprivation (area socioeconomic status), ischemic heart disease, stroke, diabetes, heart failure, night time noise. Each pollutant also adjusted for exposure to others. | PM2.5 O3 NO2  distance to a major roadway. | Model 1 (adjusted demographics and behavioral risk factors) NO2 per 7.471 µg/m3 increase HR1.17 (1.06, 1.28) PM2.5 per 0.95 µg/m3 increase HR1.07 (1.02, 1.12) O3 per 5.56 µg/m3 increase HR0.84 (0.75, 0.93) Distance to major roadway per 310 m closer HR1.02 (0.97, 1.08)  Model 4 (additional adjustment for socioeconomic status, clinical risk factors, pollutants other than the one reported, night-time noise) NO2 per 7.471 µg/m3 increase HR1.15 (1.04, 1.28) PM2.5 per 0.95 µg/m3 increase HR1.06 (1.01, 1.13) O3 per 5.56 µg/m3 increase HR0.85 (0.76, 0.96) Distance to major roadway per 310 m closer HR1.00 (0.95, 1.05)  Similar patterns for AD and VaD |
| Chen et al., 2017a [5] | Multilevel random-effects Cox proportional hazards models, accounting for similarities per areas and including exposure to pollution as a time-varying covariate. Sensitivity analyses conducted for associations of incident dementia. | Age sex, stratified region baseline SES (neighborhood-level income, education, unemployment rate, % of recent immigrants pre-existing), urban residency and a North/South indicator, comorbidities (diabetes, hypertension, coronary heart disease, stroke, heart failure, arrhythmias, traumatic brain injury), region-scale spatial patterns, urban residence, density of neurologist, geriatricians, internist, and family physicians. Secondary analysis: access to neurological care, neighborhood deprivation, linear term for time, excluded urban residency and North/South indicator. | PM2.5 | Adjusted individual pollutant model: HRIQR=1.04 (1.03, 1.05)\* Three pollutant model: HRIQR=1.02 (1.01, 1.03)\* 5-y lag: HRIQR=1.03 (1.02, 1.05)\* 10-y lag: HRIQR=1.03 (1.01, 1.06)\* |
|  |  |  | NO2 | Adjusted individual pollutant model: HRIQR=1.10 (1.08, 1.12)\* Three pollutant model: HRIQR=1.09 (1.07, 1.11)\* 5-y lag: HRIQR=1.08 (1.06, 1.09)\* 10-y lag: 1.06 (1.03, 1.08)\* |
|  |  |  | O3 | Adjusted individual pollutant model: HRIQR=0.98 (0.96, 1.00) Three pollutant model: HRIQR=0.99 (0.97, 1.01) 5-y lag: HRIQR=0.99 (0.96, 1.02) 10-y lag: HRIQR=0.99 (0.95, 1.03) |
| Cleary et al., 2018 [6] | Multi-level mixed regression, accounting for clustering within zip codes, with the highest tertile as the comparator. | Age, gender, education, race, APOE genotype, smoking, B12 deficiency, population density  (assumed no interaction terms). | PM2.5 | All compassion ns at p<0.5 Dose-dependent relationship between *APOE4* \*PM2.5 interaction and cognitive decline. Lowest decline in those without *APOE4* allele and lowest exposure. |
|  |  | O3 | Annual MMSE declines: lowest: 1.1 medium: 1.3  highest: 1.4 Annual CDR-SB declines: lowest: 0.8 medium: 1.0 highest: 1.1  MMSE: low versus highest tertile, β=0.83 (0.5, 1.2)\* medium versus highest tertile, ns low x time versus highest tertile, β=0.35 (0.2, 0.5)\* medium x time versus highest tertile, ns CDR-SB:  low versus highest tertile, β=-60 (-0.8, -0.3)\* medium versus highest tertile, ns low x time versus highest tertile, β=-0.40 (-0.5, -0.3)\* medium x time versus highest tertile, β=-0.14 (-0.2, -0.1)\*  Cognitively impaired subgroup Dose-dependent relationship between *APOE4* \*O3 interaction and cognitive decline. Lowest decline in those without *APOE4* allele and lowest exposure. |
| Chen et al., 2017 [7] | Cox proportional hazards model with age as time-scale, models stratified by region (living in Toronto or not). Adjusted HR calculated for each road way category compared to the furthest category (>300 m), and linear trend assessed with natural log of median distance. Sensitivity analyses conducted for associations of incident dementia. | age, sex, pre-existing comorbidities (traumatic brain injury, diabetes, hypertension, stroke, coronary heart disease, congestive heart failure, arrhythmia), urban residency, neighborhood-level SES (income quintile, % of population 15 or older with sub-high school education, unemployment rate, percentage of recent immigrants), regional variables Sensitivity analyses: density of neurologist, deprivation index, linear term for time, spatial dependence among participants, frailty, assumed unmeasured individual-level SES and behaviors variables (e.g., smoking, education, obesity, physical activity, long-term measures of PM2.5 and NO2 exposure (4 y-mean concentration | PM2.5 | 243611 cases of incident dementia cases between 2001-2012; ~50% lived within 200 m, 95% lived within 1000 m.  Risk of incident of dementia for distance from roadways, fully adjusted model <50 m: HR=1.07 (1.06, 1.08)\*  50-100 m: HR=1.04 (1.02, 1.05)\* 101-200 m: HR=1.02 (1.01, 1.03)\* 201-300 m: HR=1.00 (0.99, 1.01) >300 m: reference Log (distance): 0.91 (089, 0.92)\*  Sensitivity analyses:  PM2.5 and NO2 exposure modestly attenuated the association for categories of <50 m and 51-100 m <50 m: HR=1.05 (CI not reported) 50-100 m: HR=1.02  Risk of incident dementia and exposure to pollutants PM2.5 : HR=1.07 (1.06, 1.08)\* NO2: HR=1.04 (1.03, 1.05)\* Associations insensitive to additional controls; excluding first 2 and 5 y of follow up or restricting participants to >65 y old did not materially affect results. |
| Oudin et al., 2016 [8] | Cox proportional hazards models with time as underlying scale to calculate risk of long-term exposure, with censoring at date the date of dementia, date of loss to follow up, date of death or end of follow up. Baseline concertation assessed as a continuous variable and categorically in quartiles. Three models tested:  1. base line age 2. Model 1 + education physical activity, smoking, sex, wait-hip-ratio, alcohol, *APOE4*  3. Models 1-2 + baseline medical history of diabetes, hypertension, stroke Sensitivity analyses modelled exposure level back to 1993-1995. | age, education, physical activity, smoking, sex, body mass index, waist hip ratio, alcohol, *APOE4*, baseline medical diabetes, hypertension, stroke | NO2 | Incident dementia: n=301 (AD: n=191, VaD: n=111)  Risk of incident dementia Model 1 (age-adjusted) Q4: HR=1.57 (1.12, 2.19)\* Q3: HR=1.49 (1.07, 2.09)\* Q2: HR=1.10 (0.77, 1.58) Q1: reference per 10 µg/m3 increase: HR=1.04 (0.98, 1.11)  Model 2 (adjusted for genetics and behavioral factors) Q4: HR=1.43 (0.998, 2.05) Q3: HR=1.48 (1.03, 2.11)\* Q2: HR=1.11 (0.76, 1.63) Q1: reference  per 10 µg/m3 increase: HR=1.05 (0.98, 1.12)  Model 3 (fully adjusted) Q4: HR=1.60 (1.02, 2.10)\* Q3: HR=1.49 (1.04, 2.14)\* Q2: HR=1.48 (1.13, 1.66)\* Q1: reference per 10 µg/m3 increase: HR=1.05 (0.98, 1.12) |
| Jung et al., 2015 [9] | Cox proportional hazards taking to account confounds used to investigate the relationship between AD and baseline concentrations of each pollutant and between AD and change in pollutant over follow-up, with censoring at AD diagnosis, death, leaving the insurance database or end of follow-up. | age, sex, income, diabetes, diabetes mellitus, hypertension, myocardial infarction, stroke myocardial infarction, peripheral artery disease, asthma, chronic obstructive pulmonary disease, other pollutants multiple (PM10, O3, CO2, NO2, SO2) | PM2.5 | Risk of incident AD per IQR (13.21ug/m3) increment of PM2.5 Baseline: HRIQR=1.01 (0.93, 1.09) Follow-up: HRIQR=2.41 (2.24, 2.59)\*  Adjusted model Risk of incident AD per IQR (13.21 ug/m3) increment of PM2.5 Baseline: HRIQR=1.03 (0.95, 1.11) Baseline, adjustments for SO2, CO, NO2, or PM10: HRIQR remained ns Follow-up: HRIQR=2.38 (2.21, 2.56)\* Follow-up, adjustments for SO2, CO, NO2, or PM10: HRIQR increased to 2.17 to 2.43\* |
|  |  |  | O3 | Risk of incident AD per IQR (9.63 ppb) increment of O3 Baseline: HRIQR=1.06 (1.01, 1.13)\* Follow-up: HRIQR=3.12 (2.91, 3.32)\*  Adjusted models: Risk of incident AD per IQR (9.63 ppb) increment of O3  Baseline: HRIQR=1.06 (1.00, 1.12)\* Baseline, S02 adjusted: HRIQR=1.04 (0.98, 1.11) Baseline, CO adjusted: HRIQR=1.10 (1.03, 1.17)\* Baseline, N02 adjusted: HRIQR=1.06 (0.99, 1.13) Follow-up: HRIQR=3.12 (2.92, 3.33)\* Follow-up, adjustments for SO2,, CO, NO2, or PM10 : HRIQR increased to 3.23 to 3.52\* |
| Chang et al., 2014 [10] | Cox proportional hazard regression and analyses performed to examine relationship between the quartiles and incident dementia based on time to dementia diagnosis, death, leaving the insurance database or end of follow-up. Multiple models tested with controls. | age, sex, monthly income, diabetes mellitus, ischemic heart disease, hypertension, chronic obstructive pulmonary disease, alcoholism, urbanization | NO2 | Risk of incident dementia Q4: HR=1.54 (1.34, 1.77)\* Q3: HR=1.01 (0.87, 1.17) Q2: HR=1.10 (0.96, 1.26) Q1: reference  Similar patterns when they repeated the analyses by sex. |
|  |  |  | CO | Risk of incident dementia Q4: HR=1.61 (1.39, 1.85)\* Q3: HR=11.37 (1.19, 1.58)\* Q2: HR=1.07 (0.92, 1.25) Q1: reference  Similar patterns when they repeated the analyses by sex. |
| Cacciottolo et al., 2017 [11] | Cox proportional hazard models used to estimate HRs and 95% confidence intervals for adverse events associated the estimated time varying 3-y average PM2.5 exposure  Models tested: 1. Adjusted for APOE genotype 2. Adjusted for APOE, age, geographic region/spatial random effect, SES, lifestyle factors 3. Adjusted for model 2 and BMI, depression, cardiovascular disease history and risk factors | age, geographic location, education, income, employment status, lifestyle factors (smoking, alcohol, physical activity), clinical characteristics (use of hormone treatment, depression, BMI, hypercholesterolemia, Hypertension, diabetes, history of cardiovascular disease) | PM2.5 | Accelerated global cognitive decline  Model 1 (APOE-adjusted) HR=1.83 (1.47, 2.27)\* Model 2 (adjusted APOE, age, geography, SES, lifestyle) HR=1.85 (1.45, 2.36)\* Model 3 (fully adjusted) HR=1.81 (1.42, 2.32)\* |
|  |  |  | APOE x PM2.5 | Accelerated global cognitive decline by APOE status Model 1 (APOE-adjusted) ε3/3: HR=1.83 (1.47, 2.27)\* ε3/4: HR=1.85 (1.45, 2.36)\* ε4/4: HR=1.81 (1.42, 2.32)\* interaction p=0.52 Model 2 (adjusted APOE, age, geography, SES, lifestyle) ε3/3: HR=1.71 (1.28, 2.28)\* ε3/4: HR=2.03 (1.36, 3.02)\* ε4/4: HR=2.73 (1.08, 6.94) interaction p=0.54 Model 3 (fully adjusted) ε3/3: HR=1.65 (1.23, 2.23)\* ε3/4: HR=1.93 (1.29, 2.90)\* ε4/4: HR=3.64 (1.36, 9.69)\* interaction p=0.29 |
|  |  |  | PM2.5 | Risk for all-cause dementia  Model 1 (APOE-adjusted) HR=1.67 (1.21, 2.30)\* Model 2 (adjusted APOE, age, geography, SES, lifestyle) HR=1.71 (1.20, 2.45)\* Model 3 (fully adjusted)  HR=1.92 (1.31, 2.80)\* |
|  |  |  | APOE x PM2.5 | Model 1 (APOE-adjusted) by APOE status ε3/3: HR=1.26 (0.78, 2.03) ε3/4: HR=1.89 (1.23, 2.91)\* ε4/4: HR=3.53 (1.21, 10.34)\* interaction p=0.16 Model 2 (adjusted APOE, age, geography, SES, lifestyle) ε3/3: HR=1.36 (0.81, 2.28) ε3/4: HR=1.88 (1.17, 3.01)\* ε4/4: HR=3.19 (1.06, 9.57)\* interaction p=0.31 Model 3 (fully adjusted) ε3/3: HR=1.68 (0.97, 2.92) ε3/4: HR=1.91 (1.17, 3.14)\* ε4/4: HR=3.95 (1.18, 13.19)\* interaction p=0.43 |
| Oudin et al., 2017 [12] | Change on EMM calculated as absolute difference between 2 consecutive test. Repeated measures generalized estimating equations taking repeated measurements within individuals into account.   Crude model Model 1: adjusted for age Model 2: fully adjusted  Linear model: Per 1 µg/m3 increase in NOx model adjusted for age, test occasion, no. of total test, and a cross product between NOx and test occasion | age, education, smoking, BMI, work status, cohabitation, sex, physical activity | NOX | Mean baseline EMM=33 (*SD=*9.8) Crude mean decrease in EEM score per y of age=-0.47 (-0.52, -0.46)  Crude model:  Q4: -0.91 (-1.54, -0.27)\* Q3: -0.64 (-1.32, 0.05) Q2: -0.43 (-1.11, 0.24) Q1: reference Per 1 µg/m3 increase in NOx: -0.18 (-0.32, -0.004)\* Model 1: adjusted for age Q4: -1.69 (-3.36, -0.02)\* Q3: -0.78 (-2.53, 0.97) Q2: -1.22 (-2.94, 0.49) Q1: reference Per 1 µg/m3 increase in NOx: 0.001 (-0.020, 0.02) Model 2: fully adjusted Q4: -1.45 (-3.22, -0.31) Q3: -0.72 (-2.55, 1.10) Q2: -1.33 (-3.15, 0.48) Q1: reference Per 1 µg/m3 increase in NOx: 0.005 (-0.02, 0.03) |
| Oudin et al., 2018 [13] | Cox proportional hazards models with time as underlying scale to calculate HRs and 95% confidence intervals for dementia incidence associated with annual mean concentration of PM2.5.   Model 1. crude model and unadjusted estimates Model 2. traffic exhaust, PM2.5 traffic exhaust from residential wood burning, physical activity, smoking sex, BMI, Wait-hip ratio, alcohol, age Model 3. | Education level, physical activity, smoking, sex, body mass index, waist-hip ratio (>recommended versus ≤recommended), alcohol, age. | PM2.5 from traffic exhaust | Risk of incident dementia  Model 1. Q4: HR=1.65 (1.17, 2.34)\* Q3: HR=1.70 (1.21, 2.39)\* Q2: HR=0.95 (0.65, 1.38) Q1: reference per 1 μg/m3 increase in exposure: HR=1.71 (0.94, 3.13)  Model 2. Q4: HR=1.41 (0.97, 2.04) Q3: HR=1.66 (1.16, 2.39)\* Q2: HR=1.02 (0.68, 1.53) Q1: reference per 1 μg/m3 increase in exposure: HR=1.14 (0.59, 2.23) |
|  |  |  | PM2.5 from residential wood burning | Risk of incident dementia  Model 1. Q4: HR=0.97 (0.70, 1.34) Q3: HR=0.75 (0.53, 1.06) Q2: HR=0.88 (0.63, 1.22) Q1: reference per 1 μg/m3 increase in exposure: HR=1.05 (0.70, 1.57) Q4 exposure with wood stove: 1.11 (0.73, 1.69)  Model 2. Q4: HR=1.29 (0.91–1.83) Q3: HR=0.87 (0.60–1.26) Q2: HR=0.88 (0.61–1.25) Q1: reference per 1 μg/m3 increase in exposure: HR=1.55 (1.00, 2.54) Q4 exposure with wood stove: 1.74 (1.10, 2.75)\* |
| AD, Alzheimer's disease; VaD, Vascular dementia; CDR-SB, Cognitive Dementia Rating Sum of Boxes; MMSE, Mini-Mental Status Examination; CO, carbon monoxide NO2, nitrogen dioxide; O3, ozone; PM2.5 particulate matter ≤ 2.5 µm in diameter; PM10, particulate matter ≤ 10 µm in diameter; SO2, sulphur dioxide; ppb, parts per billion, y; year; \*, statistically significant; (a , b), 95% confidence interval; HR, hazard ratio; HRIQR, hazard ratio per interquartile range increase; IQR, interquartile range; ns, non-significant; OR, odds ratio; Q, quintile; SD, standard deviation; BMI, body mass index; SES, socio-economic status. | | | | |

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| **Supplementary Table 3.** Assessment of bias | | | | | | | | | |
|  | **Risk of Bias (RoB)** | | | | | | | |
| **authors** | **clear aims *(lower risk of bias with clear aims reported)*** | **appropriate methodology *(lower risk of bias with use of appropriate methodology)*** | **generalizability *(lower risk of bias where samples are generalizable)*** | **exposure measurement *(lower risk of bias where standard methods used to assess exposure)*** | **outcome measurement**  ***(lower risk of bias where standard tools/criteria used to assess outcome)*** | **un-addressed**  **confounds *(lower risk of bias where adjustments include known confounders)*** | **adjustments** | **overall RoB rating** | | |
| Weuve et al., 2012 [1] | Low | Low | Moderate (female nurses) | Low | Low | Low | age, education, husband's education, long term physical activity, long term alcohol consumption States that secondary analyses using further adjustment found similar pattern of results | Low-moderate | | |
| Loop et al., 2013 [2] | Low | Low | Low | Low | Low | Low | length of follow up, temperature, season, incident stroke, age, race, region, education, income, behavioral factors (alcohol, smoking, exercise, body mass index), depression, dyslipidemia, diabetes, hypertension | Low | | |
| Tonne et al., 2014 [3] | Low | Low | Moderate (male civil servants) | Low | Low | Low | time, age, sex, ethnicity, socioeconomic status, physical activity, consumption of alcohol, age x time and main effect of exposure | Low-moderate | | |
| Carey et al., 2018 [4] | Low | Low | Low | Low | Moderate (used health care records, likely to be subject to bias) | Low | Age, sex, ethnicity, smoking, body mass index, Index of Multiple Deprivation (area socioeconomic status), ischemic heart disease, stroke, diabetes, heart failure, night time noise. Each pollutant also adjusted for exposure to others. |  | | |
| Chen et al., 2017 [5] | Low | Low | Low | Low | Moderate (used health care records, likely to be subject to bias) | Low | living in the Toronto area, age, sex, region, comorbidity, socioeconomic status, treatment for diabetes, hypertension, coronary heart disease, stroke, heart failure, arrhythmias, traumatic brain injury, income, urban residence, recent migration, education, unemployment rate | Low-moderate | | |
| Cleary et al., 2018 [6] | Low | Low | Moderate (selected from an ongoing longitudinal study) | Low | Moderate (used participants from an existing dementia focused study, likely to be subject to bias) | Low | age, gender, education, race, APOE genotype, smoking, B12 deficiency and population density | Moderate | | |
| Chen et al., 2017 [7] | Low | Low | Low | Moderate, based on residential distance from roadway | Moderate (used health care records, likely to be subject to bias) | Low | sex, comorbidities, urban residency, neighborhood level income, income, education, unemployment, immigration status. Additional indirect adjustment for smoking, body mass index and education. | Moderate | | |
| Oudin et al., 2016 [8] | Low | Low | Low | Low | Low-moderate (dementia assessment was periodic and cox regression requires a date of onset whereas actual onset is insidious so potential for bias) | Low | age, education, physical activity, smoking, sex, body mass index, waist hip ratio, alcohol, *APOE4* , baseline diabetes, hypertension and stroke | Low | | |
| Jung et al., 2015 [9] | Low | Low | Low | Low | Moderate (used health care records and Alzheimer’s disease only, not mixed or vascular dementia, likely to be subject to bias) | Low | age, sex, income, diabetes, hypertension, myocardial infarction, stroke, asthma | Low-moderate | | |
| Chang et al., 2014 [10] | Low | Low | Low | Moderate (unclear whether the quartiles are from baseline or totaled mean exposure for the total study follow-up) | Moderate (used health care records, likely to be subject to bias) | Low | age, sex, monthly income, diabetes, ischemic heart disease, hypertension, chronic obstructive pulmonary disease, alcoholism and urbanization | Moderate | | |
| Cacciottolo et al., 2017 [11] | Low | Low | Moderate (female only, from the Women’s Health Initiative Memory Study) | Low | Low-moderate (cognitive function and dementia diagnosis was periodic and time to event analyses requires a date of onset whereas actual onset is insidious so potential for bias) | Low | Age, geographic region, education, income, employment status, smoking, alcohol use, physical activities, use of hormone treatment, depression, body mass index, hypercholesterolemia, hypertension, diabetes, history of cardiovascular disease | Low-moderate | | |
| Oudin et al., 2017 [12] | Low | Low | Low | Low | Low  (measured change in episodic memory) | Low | Age, test occasion, number of total tests, cross-product between NOx and test occasion, education, sex, smoking, body mass index, physical activity, living with someone and work status | Low | | |
| Oudin et al., 2018 [13] | Low | Low | Low | Low | Low-moderate (dementia assessment was periodic and cox regression requires a date of onset whereas actual onset is insidious so potential for bias) | Low | Physical activity, smoking, sex, body mass index, waist hip ratio, alcohol and age. PM2.5 from residential wood burning and PM2.5 from vehicle exhaust. | Low | | |

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| **Authors** | **Funding source** |
| Weuve et al., 2013 [1] | National Institute of Environmental Health Sciences (NIEHS) grant R21ES016829; development of the air pollution model was funded by NIEHS grant R01ES017017, and the Nurses’ Health Study is supported by National Cancer Institute (NCI) grant P01CA87969; Dr Schwartz’s contributions were additionally supported by Environmental Protection Agency (EPA) grant RD832416. |
| Loop et al., 2013 [2] | Federal grants from National Institute of Neurological Disorders and Stroke (U01NS41588, http://www.ninds.nih.gov/), National Institutes of Health Heart, Lung and Blood Institute (T32HL079888, http://www.nhlbi.nih.gov/), and National Aeronautics and Space Administration (NNX09AV81G, http://www.nasa.gov/). |
| Tonne et al., 2014 [3] | C.T. funded by Economic and Social Research Council (RES-064-27-0026); exposure modelling was funded through grant NE/I008039/1 as part of the Environmental Exposures and Health Initiative; A.S.M. is funded by the US National Institutes of Health (R01AG013196, R01AG034454). |
| Carey et al., 2018 [4] | UK Natural Environment Research Council, Medical Research Council, Economic and Social Research Council, Department for the Environment, Food and Rural affairs, Department of Health and the National Institute of Health Research Health Protection Research Unit |
| Chen et al., 2017 [5] | Health Canada [MOA-4500314182]. |
| Cleary et al., 2018 [6] | NACC database is funded by NIA/NIH Grant U01 AG016976; NACC data are contributed by the NIA-funded ADCs: P30 AG019610 (PI Eric Reiman, MD), P30 AG013846 (PI Neil Kowall, MD), P50 AG008702 (PI Scott Small, MD), P50 AG025688 (PI Allan Levey, MD, PhD), P50 AG047266 (PI Todd Golde, MD, PhD), P30 AG010133 (PI Andrew Saykin, PsyD), P50 AG005146 (PI Marilyn Albert, PhD), P50 AG005134 (PI Bradley Hyman, MD, PhD), P50 AG016574 (PI Ronald Petersen, MD, PhD), P50 AG005138 (PI Mary Sano, PhD), P30 AG008051 (PI Steven Ferris, PhD), P30 AG013854 (PI M. Marsel Mesulam, MD), P30 AG008017 (PI Jeffrey Kaye, MD), P30 AG010161 (PI David Bennett, MD), P50 AG047366 (PI Victor Henderson, MD, MS), P30 AG010129 (PI Charles DeCarli, MD), P50 AG016573 (PI Frank LaFerla, PhD), P50 AG016570 (PI Marie-Francoise Chesselet, MD, PhD), P50 AG005131 (PI Douglas Galasko, MD), P50 AG023501 (PI Bruce Miller, MD), P30 AG035982 (PI Russell Swerdlow, MD), P30 AG028383 (PI Linda Van Eldik, PhD), P30 AG010124 (PI John Trojanowski, MD, PhD), P50 AG005133 (PI Oscar Lopez, MD), P50 AG005142 (PI Helena Chui, MD), P30 AG012300 (PI Roger Rosenberg, MD), P50 AG005136 (PI Thomas Montine, MD, PhD), P50 AG033514 (PI Sanjay Asthana, MD, FRCP), P50 AG005681 (PI John Morris, MD), and P50 AG047270 (PI Stephen Strittmatter, MD, PhD). |
| Chen et al., 2017 [7] | Health Canada (MOA-4500314182). |
| Oudin et al., 2016 [8] | The Swedish Research Council for Health, Working Life and Welfare under grant agreement 2011-1218 (B.F.). |
| Jung et al., 2015 [9] | China Medical University (CMU#100-AWARD-07). |
| Chang et al., 2014 [10] | China Medical University (CMU102-BC-2), Taiwan Ministry of Health and Welfare Clinical Trial and Research Centre of Excellence (MOHW103-TDU-B-212-113002), Taiwan Ministry of Health and Welfare Cancer Research Centre for Excellence (MOHW103-TD-B-111-03);  International Research-Intensive Centres of Excellence in Taiwan (I-RiCE) (NSC101-2911-I-002-303). |
| Cacciottolo et al., 2017 [11] | WHI program is funded by the National Heart, Lung, and Blood Institute (NIH) through contracts HHSN268201100046C, HHSN268201100001C, HHSN268201100002C, HHSN268201100003C, HHSN268201100004C and HHSN271201100004C. WHIMS was funded by Wyeth Pharmaceuticals, St Davids, PA, USA, and Wake Forest University. Study was supported by NIH awards R01AG033078, R01AG051521, R21AG040753, R21AG040683 and R00AG032361, by awards to J.C. Chen (R01AG033078; RF1AG054068), to I. Driscoll (R00AG032361) and to C.E. Finch (R01AG051521, R21AG040753, R21AG040683, R21AG0500201 and by the Cure Alzheimer's Fund). It is also supported by the Southern California Environmental Health Sciences Center (5P30ES007048). |
| Oudin et al., 2017 [12] | - |
| Oudin et al., 2018 [13] | Svenska ForskningsrådetFormas grant no. 2017-00898to AO. The funder had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. |
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**Reasons for Exclusion**

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|  |  | **Decision** | **Reason** | **Reason** |
| 1 | Seo et al., 2014 [14] | Reject | No appropriate longitudinal data. | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. Occupational exposure to lead. |
| 2 | Sun 2017 [15] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 3 | Wilker et al., 2016 [16] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 4 | Colicino et al., 2017 [17] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 5 | Colicino et al., 2014 [18] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  Aim was to look at mitochondrial haplotype relationship between black carbon and cognitive function. Cognitive change or mean cognitive change over multiple visits was examined as part of sensitivity analyses but unclear how cognitive change was calculated/used in the analyses. |
| 6 | Colicino et al., 2016 [19] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 7 | Giacoppo et al., 2014 [20] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 8 | Fehsel et al., 2016 [21] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 9 | Reed et al., 2014 [22] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 10 | Ailshire et al., 2016 [23] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 11 | Bowler et al., 2015 [24] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of cognitive decline (change in cognitive function) or incident dementia. |
| 12 | Eum et al., 2013 [25] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 13 | Tzivian et al., 2017 [26] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 14 | Peng et al., 2017 [27] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. Alzheimer Disease mortality only. |
| 15 | Linares et al., 2017 [28] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 16 | Schikowski et al., 2015 [29] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 17 | Bos et al., 2013 [30] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  Less than six months follow up. |
| 18 | Kioumourtzoglou et al., 2016 [31] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 19 | Tallon et al., 2017 [32] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 20 | Wellenius et al., 2012 [33] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 21 | Sun & Gu, 2008 [34] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 22 | Zeng et al., 2010 [35] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 23 | Ranft et al., 2009 [36] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 24 | Chen & Schwartz 2009 [37] | Reject | No appropriate longitudinal data | No appropriate longitudinal data.  No analysis of exposure to air pollution and cognitive decline (change in cognitive function) or incident dementia. |
| 25 | Min, 2016 [38] | Reject | Not clear whether solely related to air pollution/irrelevant measures. | Not clear whether solely related to air pollution/irrelevant measures. Assessed cadmium levels. |
| 26 | Prada et al., 2016 [39] | Reject | Not clear whether solely related to air pollution/irrelevant measures. | Not clear whether solely related to air pollution/irrelevant measures. Assessed bone lead levels. |
| 27 | Farooqui et al., 2017 [40] | Reject | Not clear whether solely related to air pollution/irrelevant measures. Assessed bone lead levels. | Not clear whether solely related to air pollution/irrelevant measures. Assessed bone lead levels. |
| 28 | Power et al., 2013 [41] | Reject | Not clear whether solely related to air pollution/irrelevant measures. | Not clear whether solely related to air pollution/irrelevant measures. Assessed bone and blood lead levels. |
| 29 | Gonzalez et al., 2017 [42] | Reject | Not clear whether solely related to air pollution/irrelevant measures. | Not clear whether solely related to air pollution/irrelevant measures.  Assessed dog and human autopsy data. |
| 30 | Norlen et al., 2004 [43] | Reject | Not clear whether solely related to air pollution/irrelevant measures. | Not clear whether solely related to air pollution/irrelevant measures. Assessed blood lead levels. |
| 31 | Weisskopf et al., 2012 [44] | Reject | Review | A review. Not original research. |
| 32 | Andersson et al., 2018 [45] | Reject | No appropriate longitudinal data | Reported results for the same cohort and outcomes as Oudin et al., 2016 No numerical results reported for dementia and air pollution in Andersson et al., focus is on noise and air pollution combined. |

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