OPEN ACCESS

Long-term exposure to ambient fine particulate matter originating from traffic and residential wood combustion and the prevalence of depression

Sara Allaouat ¹,² Tarja Yli-Tuomi,² Pekka Tiittanen,² Anu W Turunen,² Taina Siponen,² Jaakko Kukkonen,^{3,4,5} Leena Kangas,³ Mari Kauhaniemi,³ Mia Aarnio,⁶ Tiia Ngandu,⁷ Timo Lanki^{1,2,8}

► Additional supplemental material is published online only. To view, please visit the journal online (<http://dx.doi.org/10.1136/jech-2021-216772>).

For numbered affiliations see end of article.

Correspondence to

Sara Allaouat, Institute of Public Health and Clinical Nutrition, University of Eastern Finland, Kuopio, Finland; sara.allaouat@uef.fi

Received 8 March 2021
Revised 19 April 2021
Accepted 24 April 2021
Published Online First
13 May 2021

ABSTRACT

Introduction Air pollution has been suggested to be associated with depression. However, current evidence is conflicting, and no study has considered different sources of ambient particulate matter with an aerodynamic diameter below 2.5 µm (PM_{2.5}). We evaluated the associations of long-term exposure to PM_{2.5} from road traffic and residential wood combustion with the prevalence of depression in the Helsinki region, Finland.

Methods We conducted a cross-sectional analysis based on the Helsinki Capital Region Environmental Health Survey 2015–2016 (N=5895). Modelled long-term outdoor concentrations of PM_{2.5} were evaluated using high-resolution emission and dispersion modelling on an urban scale and linked to the home addresses of study participants. The outcome was self-reported doctor-diagnosed or treated depression. We applied logistic regression and calculated the OR for 1 µg/m³ increase in PM_{2.5}, with 95% CI. Models were adjusted for potential confounders, including traffic noise and urban green space.

Results Of the participants, 377 reported to have been diagnosed or treated for depression by a doctor. Long-term exposure to PM_{2.5} from road traffic (OR=1.23, 95% CI 0.86 to 1.73; n=5895) or residential wood combustion (OR=0.78, 95% CI 0.43 to 1.41; n=5895) was not associated with the prevalence of depression. The estimates for PM_{2.5} from road traffic were elevated, but statistically non-significant, for non-smokers (OR=1.38, 95% CI 0.94 to 2.01; n=4716).

Conclusions We found no convincing evidence of an effect of long-term exposure to PM_{2.5} from road traffic or residential wood combustion on depression.

atrophy, have been shown to correlate with exposure to particulate matter. Indeed, a few studies have reported that particulate matter is associated with inflammation and oxidative stress in animal brain.^{4–5} Particulate matter has also been associated with stress hormone release in animals and humans, and with decreased hippocampal nerve growth factors along with depressive symptoms in animals.^{4,6} Short-term exposure to gasoline exhaust has caused changes in neurotransmitter levels in favour of depression in rats.⁷

Epidemiological studies have also suggested that exposure to air pollution could be associated with depression. Both short-term (hourly or daily) and long-term exposures to ambient particulate matter with an aerodynamic diameter below 2.5 µm (PM_{2.5}) have shown a correlation with depression. However, the evidence base is limited due to the scarcity of available studies and the shortcomings in their design.^{8–9} Moreover, results have been conflicting, especially for areas with lower concentrations of PM_{2.5}.¹⁰ To our knowledge, no research has considered the associations between depression and different source categories of PM_{2.5}.

This study evaluates the associations of long-term exposure to PM_{2.5} from road traffic and residential wood combustion with the prevalence of depression in the Helsinki region, Finland, an area with relatively low PM_{2.5} levels (annual average in most locations ≤10 µg/m³). Road traffic and wood combustion are the main local sources of fine particles in the study area.

METHODS

Study design and population

We conducted a cross-sectional study based on the Helsinki Capital Region Environmental Health Survey. The survey was conducted by the Finnish Institute for Health and Welfare in Finland, in Helsinki in 2015 and in Vantaa and Espoo in 2016, as described earlier.¹¹ Participants (n=7321), aged 25 years and above, were sampled randomly from the general population living in the Helsinki region using the population register. The survey response rate was 47% in 2015 and 45% in 2016.

Exposure assessment

We used residential outdoor levels of fine particulate matter as estimates of personal exposure.

INTRODUCTION

The physical environment has been suggested to play an important role in mental well-being, but relevant environmental components remain unclear. Emerging evidence suggests that air pollution could be associated with a mental health decline.¹

The hypothesis that particulate matter affects the biological mechanisms behind depressive disorders is plausible. Several theories on the pathophysiology of depression have been presented.^{2,3} Some of the covered pathophysiological processes, such as inflammation and oxidative stress, neurotransmitter imbalances including monoamine and serotonin decrease, elevated glucocorticoids, and brain

© Author(s) (or their employer(s)) 2021. Re-use permitted under CC BY-NC. No commercial re-use. See rights and permissions. Published by BMJ.

To cite: Allaouat S, Yli-Tuomi T, Tiittanen P, et al. *J Epidemiol Community Health* 2021;**75**:1111–1116.

Estimates of traffic-originated $PM_{2.5}$ concentrations in the Helsinki region were based on emission and dispersion modelling. The dispersion model used was the 'Contaminants in the Air from a Road-Finnish Meteorological Institute'.¹² Vehicular exhaust emission factors originated from 2014, and meteorological data for years 2012–2014 were used in the modelling. The distances between the receptor points varied from 25 m near roads to 200 m in rural areas. In the present study, the $PM_{2.5}$ concentration assigned to a participant's home was evaluated as the road traffic concentration at the nearest outdoor modelling point. The highest annual average of 2010–2014 was used as the exposure estimate.

The outdoor concentrations of $PM_{2.5}$ have been modelled globally, in Europe and in the Helsinki region for a period of 35 years, from 1980 to 2014.¹³ In the Helsinki region, the emissions originating from domestic wood combustion were assumed to be uniformly distributed in area sources of size $100 \times 100 m^2$. The corresponding concentrations were evaluated using the Urban Dispersion Model of the Finnish Meteorological Institute on a horizontal resolution of $100 \times 100 m^2$.¹³ In the present study, the concentration of $PM_{2.5}$ for each participant's home was evaluated as the residential wood combustion concentration at the nearest outdoor modelling point. The average values for the years from 2010 to 2014 were used as the exposure estimates.

The description of how exposures to traffic noise and green and blue spaces were estimated is available in online supplemental materials and methods.

Primary outcome measure

We used a binary outcome measure for depression that was based on self-report. The question in the survey was 'has a doctor diagnosed or treated you for [depression] during the last year (12 months)?' with response options (yes/no).

Statistical analysis

We performed logistic regression analyses using the function *glm()* in R V.3.6.0 software.¹⁴ We calculated the ORs for $1 \mu g/m^3$ increase in $PM_{2.5}$ and their 95% CIs.

In the crude model, we included residential wood smoke and road traffic $PM_{2.5}$ simultaneously and adjusted air pollutant effects for road traffic noise (L_{den}), green space within 1 km, age and sex. In the main model, we included variables of the crude model but further adjusted for marital status, employment status, annual household income, education, alcohol intake in the last 7 days, daily smoking, weekly physical exercise, area-level unemployment (rate, %) and area-level income (mean, €). In the single-pollutant models, we considered either road traffic $PM_{2.5}$ or residential wood combustion $PM_{2.5}$, and we adjusted for the same covariates as in the main model. Covariate categories are described in online supplemental table S1.

Using the main model, we tested the linearity of the exposure–response function for air pollution and for the confounders used on a continuous scale, namely road traffic noise, green space within 1 km, area-level unemployment and area-level income. We used the spline function *s()* from the *mgcv* package V.1.8–33.¹⁵

To test the consistency and robustness of the results, we conducted several sensitivity analyses, each on the main model: in separate models, we considered potential confounding by green space within 300 m; nature space within 1 km (including green and blue spaces and excluding field and pasture, %); use of summer cottage in summer; body mass index (kg/m^2); own wood combustion; comorbidities; and road traffic noise annoyance.

A detailed description of these variables is available in online supplemental table S1.

We also used information on depression medication intake as an alternative outcome measure. It was defined by the survey question 'when did you last take [antidepressants]?' and the possible answers were 'during the past week', 'one to four weeks ago', 'one to 12 months ago', 'over a year ago' and 'never'. We then dichotomised this outcome measure by categorising the three first possible answers as 'depression medication use' and the two last possible answers as 'no depression medication use'.

In further sensitivity models, we excluded one by one some population groups: smokers, participants who had lived in the same address for less than 1 year or less than 3 years, and participants living in floors higher than the fifth floor. To test for potential effect modification, we applied a multiplicative function for road traffic noise, by adding an interaction term *road traffic noise*road traffic $PM_{2.5}$* in the model. Finally, we tested the effect of influential data points, including outliers, and we checked whether the variables used on a continuous scale were collinear.¹⁶

RESULTS

The total number of study participants with full exposure and confounder data was 5895. Of these participants, those diagnosed or treated for depression by a doctor represented 6.4% ($n=377$) and those using depression medication 6.6% ($n=385$). The mean residential outdoor concentration of $PM_{2.5}$ attributable to road traffic was $0.59 \mu g/m^3$ and that attributable to residential wood combustion was $0.52 \mu g/m^3$.

These estimates correspond to an urban background concentration of ambient $PM_{2.5}$ of $8 \mu g/m^3$ in the Helsinki region, as measured in 2014 at the station of Kallio.¹⁷ The station is located in central Helsinki, in a park area which is not immediately influenced by any road. The concentration value at this station in 2014 consisted mainly of regional and long-range transported particulate matter, with smaller contributions from urban traffic and residential wood combustion.^{18 19}

The modelled concentrations of $PM_{2.5}$ in the Helsinki region in 2013, originating from all sources and from the two considered urban source categories, are presented in figure 1A–C. The concentrations originating from residential wood combustion are focused mainly on the suburbs situated north and north-west from the centre of Helsinki. The predicted spatial distributions of the yearly $PM_{2.5}$ concentrations in this region during various years and originating from various source categories have been discussed in more detail.^{18 19}

Tables 1 and 2 present the key characteristics of the study participants and their environment.

The mean age of the study participants was 53.6 years. The prevalence of high alcohol intake (ie, more than six units per week) was 18.3% ($n=1076$). The prevalence of participants who reported to burn wood themselves at least once a week was 11.9% ($n=680$). The mean coverage of nature space within 1 km was 29.4% ($SD=12.53$) and of green space within 300 m 19.45% ($SD=14.17$). Participants who experienced some to extremely high road traffic noise annoyance represented 9.1% ($n=539$). Participants using summer cottage for 2 weeks at least, during summertime, counted for 33.9% ($n=1981$). Participants who had lived in the same address for less than 1 year represented 7.5% ($n=439$), those living in the same address for less than 3 years 28.0% ($n=1651$), and participants living in the fifth floor or lower 87.4% ($n=5139$). Online supplemental figure S1

Figure 1 Predicted concentrations of $PM_{2.5}$ in the Helsinki metropolitan area in 2013, originating from (A) all urban sources and the long-range and regionally transported background, (B) residential wood combustion, and (C) vehicular traffic. The figures also show the location of the main street and road network and the sea areas. Map data copyright: OpenStreetMap contributors. $PM_{2.5}$, particulate matter with an aerodynamic diameter $\leq 2.5 \mu m$.

presents the correlation matrix for the continuous covariates of exposure and outcomes.

In the crude model, long-term exposure to road traffic $PM_{2.5}$ was associated with the prevalence of depression (OR=1.54, 95%CI 1.10 to 2.14). Using the main model, we still found an elevated effect estimate for $PM_{2.5}$ from traffic, but it was not statistically significant (OR=1.23, 95%CI 0.86 to 1.73). Similarly, in the single-pollutant model we found somewhat elevated but not statistically significant effect estimate for $PM_{2.5}$ from traffic (OR=1.29, 95%CI 0.93 to 1.79). We found no

Table 1 Key characteristics of the study participants (N=5895)

Categorical variable	n (%)
Age (years)	
<55	3027 (51.4)
55–69	1800 (30.5)
≥ 70	1068 (18.1)
Sex	
Female	3361 (57.0)
Male	2534 (43.0)
Marital status	
Single	1025 (17.4)
Married/cohabiting	3878 (65.8)
Divorced/widow	992 (16.8)
Annual household income (€)	
$\leq 30\ 000$	1453 (24.65)
30 001–50 000	1489 (25.26)
50 001–90 000	1862 (31.59)
$> 90\ 000$	1091 (18.51)
Employment status	
Full-time job	3073 (52.1)
Part-time job	358 (6.07)
Retired	1933 (32.8)
Unemployed	298 (5.06)
Other	233 (3.95)
Education	
Comprehensive school	789 (13.4)
Vocational or high school	1361 (23.1)
College level or polytechnic training	1890 (32.1)
Academic training	1855 (31.5)
Smoking	
No	4850 (82.3)
≤ 2 units per week	218 (3.70)
> 2 units per week	827 (14.0)
Weekly physical exercise	
Less than once	1151 (19.53)
One to two times	1935 (32.82)
More than two times	2809 (47.66)
Comorbidities	
None	3191 (54.13)
One comorbidity	1544 (26.19)
Two comorbidities	687 (11.65)
Three comorbidities	352 (5.97)
NA	121 (2.05)
Doctor-diagnosed/treated depression	
No	5518 (93.6)
Yes	377 (6.40)
Depression medication use	
No	5450 (93.4)
Yes	385 (6.53)
NA	60 (1.02)

NA, not available.

association between long-term exposure to residential wood combustion $PM_{2.5}$ and depression in the crude model, main model (OR=0.78, 95%CI 0.43 to 1.41) or single-pollutant model (table 3).

Table 2 Descriptive statistics for the environmental exposures of study participants (N=5895)

Continuous variable	Mean	SD	Median	Minimum	Maximum
Unemployment rate (%)	5.97	1.59	6.00	1.60	14.1
Area-level income (€/year)	27 482	6009	26 061	12 345	69 066
Long-term road traffic PM _{2.5} (µg/m ³)	0.59	0.37	0.49	0.00	2.97
Long-term PM _{2.5} from residential wood combustion (µg/m ³)	0.52	0.21	0.52	0.08	1.12
Green space within 1 km (%)	24.8	11.4	23.9	1.88	81.6
Long-term road traffic noise, L _{den} (dB)	50.5	7.83	50.1	30.0	75.5

dB, decibels; L_{den}, day-evening-night equivalent level; PM_{2.5}, particulate matter with an aerodynamic diameter ≤2.5 µm.

Adding income to the crude model diminished the effect estimate for traffic-related PM_{2.5} to a statistically non-significant level (OR=1.27, 95% CI 0.90 to 1.77). The income variable included four categories (online supplemental table S1) and correlated with both the mean PM_{2.5} from road traffic and depression: individuals with the lowest incomes (ie, earning up to €30 000 per year) were exposed to the highest levels of PM_{2.5} from road traffic (0.62 µg/m³) and had the highest prevalence of depression with 48.5%.

We concluded all confounders to have a linear relationship with the outcome based on the shape of the exposure-response functions (online supplemental figures S2–S5). Similarly, the exposure-response functions for air pollution effects could be considered linear (online supplemental figures S6 and S7).

In further sensitivity analyses, the results remained essentially the same as in the main analyses. However, when only non-smokers were included, effect estimate for the association between PM_{2.5} from road traffic and depression increased, but was still statistically non-significant (OR=1.38, 95% CI 0.94 to 2.01; n=4716) (online supplemental table S2). The test on the effect modification by road traffic noise was inconclusive (p=0.12). Removing influential data points with the highest hat value (>0.0225) had no effect on the main model, either for road traffic PM_{2.5} (OR=1.28, 95% CI 0.89 to 1.82; n=5883) or residential wood combustion PM_{2.5} (OR=0.80, 95% CI 0.44 to 1.46; n=5883). Finally, we found that the continuous variables used in the analysis were not collinear (data not shown).

Table 3 Associations of long-term exposure to road traffic PM_{2.5} and residential wood combustion PM_{2.5} with the prevalence of depression, calculated for a 1 µg/m³ increase in PM_{2.5} (N=5895)

Model	Exposure	OR (95% CI)
Crude model*	Residential wood smoke PM _{2.5}	0.76 (0.44 to 1.31), p=0.32
	Road traffic PM _{2.5}	1.54 (1.10 to 2.14), p=0.01
Main model†	Residential wood smoke PM _{2.5}	0.78 (0.43 to 1.41), p=0.41
	Road traffic PM _{2.5}	1.23 (0.86 to 1.73), p=0.26
Single-pollutant model‡	Residential wood smoke PM _{2.5}	0.69 (0.39 to 1.19), p=0.19
	Road traffic PM _{2.5}	1.29 (0.93 to 1.79), p=0.12

*Crude model adjusted for road traffic noise, green space within 1 km, age and sex; both residential wood smoke and road traffic PM_{2.5} included in the model as exposures.

†Main model and single-pollutant models adjusted for marital status, employment status, annual household income, education, daily smoking, weekly physical exercise, area-level unemployment and area-level income, in addition to adjustments of the crude model; both residential wood smoke and road traffic PM_{2.5} included in the model as exposures.

‡

PM_{2.5}, particulate matter with an aerodynamic diameter ≤2.5 µm.

DISCUSSION

We conducted a cross-sectional analysis to study the associations of long-term exposure to PM_{2.5} from road traffic and residential wood combustion with depression in the Helsinki region, Finland. Although the crude model showed an association between long-term exposure to road traffic PM_{2.5} and the prevalence of depression, no clear associations were found after controlling for additional confounders. In non-smokers, there was a suggestive association between PM_{2.5} from road traffic and depression. We found no association between long-term exposure to residential wood combustion PM_{2.5} and the prevalence of depression.

No other study has evaluated the effects of source-specific PM_{2.5} on depression. However, comparisons can be made with studies that have included total PM_{2.5}. The present study is in line with the evidence from two systematic reviews and meta-analyses. One meta-analysis found no association of PM_{2.5} with depression after either short-term or long-term exposure.⁸ Another meta-analysis found no association of long-term exposure to PM_{2.5} with depressive symptoms for levels of PM_{2.5} less than 15 µg/m³.¹⁰ The urban background level of ambient PM_{2.5} in the Helsinki region was 8 µg/m³ in 2014.¹⁷ One more meta-analysis reported an association related to long-term exposure, but included only studies published in English up to September 2017, while the literature has grown rapidly afterwards.⁹

Similarly to our findings, several cohort studies found no clear associations between long-term exposure to ambient PM_{2.5} and depression in areas with low (<10 µg/m³) or moderate (10–25 µg/m³) concentrations. These studies include three European cohorts and one prospective cohort from the USA.^{20 21} One other cohort from South Korea also found no association between exposure to PM_{2.5} and depression.²²

Of the three European cohorts, one was Finnish. However, it was different from this study in terms of setting, study years, outcome definition and exposure modelling. The cohort included data from the city of Turku in addition to Helsinki and Vantaa, and the outcome was based on self-reported depressed mood. Health data were collected in 2007 and air pollution modelled for the years 2010–2011 using land use regression methodology. The sample was also smaller than the sample in the present study (n=1367), and 11.3% participants had depressed mood.²⁰

Some other cross-sectional and longitudinal studies suggest that there could yet be an association between long-term exposure to ambient PM_{2.5} even in areas with low (<10 µg/m³) or moderate (10–25 µg/m³) levels and depression.^{23–28} That said, the results of one longitudinal study suggested that long-term exposure to PM_{2.5} in an area with low concentration is associated with the onset of depression in the elderly, but may not be associated with its continuation or exacerbation.²⁹

Most of the studies showing no clear associations tend to have increased ORs/risk estimates but also large CIs crossing the null-effect line.^{20 21 23 30} Therefore, an effect cannot be ruled out. In areas with long-term levels of PM_{2.5} exceeding 15 µg/m³, stronger and more consistent associations with depression have been reported.^{10 31–35}

To our knowledge, the present study is the first to consider road traffic and residential wood combustion as source categories of PM_{2.5} in relation with depression. These sources may have different health effects due to differences in the chemical composition; for example, a higher concentration in polycyclic aromatic hydrocarbons has often been found in residential wood smoke, which may indicate a higher toxicity.^{36–38}

The present study is also one of the rare studies that control for the confounding effect of green space and road traffic noise. Most of the available studies described above did not adjust for these important confounders.^{21 22 24 26 27 29 30 39} The three European cohorts controlled for noise, and the association between PM_{2.5} exposure and depression was not clear.²⁰ One cross-sectional study for which noise data were available for a small sample found no effect of noise on depression, but did not include noise in the models to assess the association between PM_{2.5} exposure and depression.²⁴ One cross-sectional study adjusting for noise and green space found no clear associations between PM_{2.5} and depression.²³

One of the strengths of this study is the assessment of exposure. The models used to estimate PM_{2.5} in the Helsinki region are sophisticated and have been shown to predict measured PM_{2.5} reasonably well. The annual average concentration of PM_{2.5} from road traffic did not change significantly in the period 2010–2014.¹² Thus, these estimates are expected to reflect long-term exposure of the study participants. In 2008, 72%–92% of PM_{2.5} in the Helsinki region originated from outside of Finland.⁴⁰

The present study was based on an a priori protocol, from which we only deviated to conduct more sensitivity analyses than planned initially. The extensive sensitivity analyses supported the initial findings, which increased confidence in the results.

The cross-sectional design of our research is an obvious limitation and longitudinal studies controlling for green space and

traffic noise are necessary to further confirm these findings. The participation rate of the survey was 45%–47%. If persons with depression were less likely to participate, this may have led to a loss of power. It would have also diluted potential associations if, in addition, PM_{2.5} was higher among non-participants. Further, the use of self-reported data on depression may have led to underdiagnosis/overdiagnosis. This may have led to some loss of statistical power. However, it is unlikely that the misclassification would be linked to PM_{2.5} exposure and thus bias the results of the study.

Author affiliations

- ¹Institute of Public Health and Clinical Nutrition, University of Eastern Finland, Kuopio, Finland
- ²Department of Health Security, Finnish Institute for Health and Welfare, Kuopio, Finland
- ³Department of Atmospheric Composition Research, Finnish Meteorological Institute, Helsinki, Finland
- ⁴Centre for Atmospheric and Climate Physics Research, University of Hertfordshire, Hatfield, UK
- ⁵Centre for Climate Change Research, University of Hertfordshire, Hatfield, UK
- ⁶Department of Air Quality Research, Finnish Meteorological Institute, Helsinki, Finland
- ⁷Department of Public Health and Welfare, Finnish Institute for Health and Welfare, Helsinki, Finland
- ⁸Department of Environmental and Biological Sciences, University of Eastern Finland, Kuopio, Finland

Acknowledgements The Academy of Finland funding for the project 'Global health risks related to atmospheric composition and weather (GLORIA)' is acknowledged. We thank Mr Kari Riikonen for processing the geographical concentration data.

Contributors SA wrote the manuscript and analysed the data. TY-T, JK, LK, MK and MA produced the exposure data. PT designed the questionnaire and supervised the data analyses. AWT designed the questionnaire. TS and TN interpreted the results. TL designed the study and the questionnaire, drafted the analysis plan, and supervised the work. He is the guarantor. All authors revised the draft and approved the manuscript before submission.

Funding SA received a doctoral research grant from the Finnish Cultural Foundation – North Savo Regional Fund, and additional intramural funding from the University of Eastern Finland.

Map disclaimer The depiction of boundaries on this map does not imply the expression of any opinion whatsoever on the part of BMJ (or any member of its group) concerning the legal status of any country, territory, jurisdiction or area or of its authorities. This map is provided without any warranty of any kind, either express or implied.

Competing interests None declared.

Patient consent for publication Not required.

Ethics approval According to Finnish legislation, no ethical permission is needed for a questionnaire which only includes adults.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. Health data are not available due to confidentiality issues. Reuse of the concentration and meteorological data is permitted upon request, without limitations. Contact: Professor Jaakko Kukkonen, Finnish Meteorological Institute, Erik Palmenin aukio 1, PO Box 503, FI-00101 Helsinki (email: jaakko.kukkonen@fmi.fi, ORCID identifier <https://orcid.org/0000-0002-5943-7069>).

Supplemental material This content has been supplied by the author(s). It has not been vetted by BMJ Publishing Group Limited (BMJ) and may not have been peer-reviewed. Any opinions or recommendations discussed are solely those of the author(s) and are not endorsed by BMJ. BMJ disclaims all liability and responsibility arising from any reliance placed on the content. Where the content includes any translated material, BMJ does not warrant the accuracy and reliability of the translations (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages), and is not responsible for any error and/or omissions arising from translation and adaptation or otherwise.

Open access This is an open access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is

What is already known on this subject

- ▶ Previous studies have shown inconsistent results on the association between long-term exposure to ambient fine particulate matter (PM_{2.5}; aerodynamic diameter below 2.5 µm) and depression.
- ▶ No epidemiological evidence exists for different sources of PM_{2.5} such as road traffic and residential wood combustion.

What this study adds

- ▶ We evaluated whether long-term exposure to PM_{2.5} from road traffic or residential wood combustion is associated with the prevalence of depression in an urban area with fairly low air pollution concentrations.
- ▶ We found no convincing evidence that long-term exposure to low levels of road traffic PM_{2.5} or residential wood combustion PM_{2.5} would be associated with the prevalence of depression; however, an effect could not be ruled out for traffic-related PM_{2.5}, especially among non-smokers.

properly cited, appropriate credit is given, any changes made indicated, and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>.

ORCID iD

Sara Allouat <http://orcid.org/0000-0002-2850-111X>

REFERENCES

- Xue T, Zhu T, Zheng Y, et al. Declines in mental health associated with air pollution and temperature variability in China. *Nat Commun* 2019;10:2165.
- Fekadu N, Shibeshi W, Engidawork E. Major depressive disorder: pathophysiology and clinical management. *J Depress Anxiety* 2017;06:255–61.
- Hasler G. Pathophysiology of depression: do we have any solid evidence of interest to clinicians? *World Psychiatry* 2010;9:155–61.
- Thomson EM. Air pollution, stress, and allostatic load: linking systemic and central nervous system impacts. *J Alzheimers Dis* 2019;69:597–614.
- Chu C, Zhang H, Cui S, et al. Ambient PM_{2.5} caused depressive-like responses through Nrf2/NLRP3 signaling pathway modulating inflammation. *J Hazard Mater* 2019;369:180–90.
- Liu J, Yang C, Yang J, et al. Effects of early postnatal exposure to fine particulate matter on emotional and cognitive development and structural synaptic plasticity in immature and mature rats. *Brain Behav* 2019;9:e01453.
- Kinawy AA, Ezzat AR, Al-Suwaigh BR. Inhalation of air polluted with gasoline vapours alters the levels of amino acid neurotransmitters in the cerebral cortex, hippocampus, and hypothalamus of the rat. *Exp Toxicol Pathol* 2014;66:219–24.
- Fan S-J, Heinrich J, Bloom MS, et al. Ambient air pollution and depression: a systematic review with meta-analysis up to 2019. *Sci Total Environ* 2020;701:134721.
- Braithwaite I, Zhang S, Kirkbride JB, et al. Air pollution (particulate matter) exposure and associations with depression, anxiety, bipolar, psychosis and suicide risk: a systematic review and meta-analysis. *Environ Health Perspect* 2019;127:126002.
- Zeng Y, Lin R, Liu L, et al. Ambient air pollution exposure and risk of depression: a systematic review and meta-analysis of observational studies. *Psychiatry Res* 2019;276:69–78.
- Okokon EO, Yli-Tuomi T, Turunen AW, et al. Traffic noise, noise annoyance and psychotropic medication use. *Environ Int* 2018;119:287–94.
- Kukkonen J, Härkönen J, Walden J. Evaluation of the dispersion model CAR-FMI against data from a measurement campaign near a major road. *Atmos Environ* 2001;35:949–60.
- Kukkonen J, Kangas L, Kauhaniemi M. Modelling of the urban concentrations of PM_{2.5} on a high resolution for a period of 35 years, for the assessment of lifetime exposure and health effects. *Atmos Chem Phys* 2018;18:8041–64.
- R Core Team. R: a language and environment for statistical computing. R foundation for statistical computing, 2018. Available: <https://www.R-project.org/> [Accessed 10 Aug 2020].
- Wood SN. Thin plate regression splines. *Journal of the Royal Statistical Society* 2003;65:95–114.
- Fox J, Monette G. Generalized collinearity diagnostics. *J Am Stat Assoc* 1992;87:178–83.
- Malkki M, Loukkola K. *Air quality in the Helsinki metropolitan area in 2014*. Helsinki, Finland: Helsinki Region Environmental Services Authority, 2015. https://www.hsy.fi/globalassets/ilmanlaatu-ja-ilmasto/tiedostot/6_2015_ilmanlaatu_paakaupunkiseudulla_vuonna_2014.pdf
- Kauhaniemi M, Karppinen A, Härkönen J, et al. Evaluation of a modelling system for predicting the concentrations of PM_{2.5} in an urban area. *Atmos Environ* 2008;42:4517–29.
- Kukkonen J, López-Aparicio S, Segersson D. The influence of residential wood combustion on the concentrations of PM_{2.5} in four Nordic cities. *Atmos Chem and Phys* 2020;20:4333–65.
- Zijlema WL, Wolf K, Emery R, et al. The association of air pollution and depressed mood in 70,928 individuals from four European cohorts. *Int J Hyg Environ Health* 2016;219:212–9.
- Kioumourtoglou M-A, Power MC, Hart JE, et al. The association between air pollution and onset of depression among middle-aged and older women. *Am J Epidemiol* 2017;185:801–9.
- Zhang Z, Zhao D, Hong YS, et al. Long-term particulate matter exposure and onset of depression in middle-aged men and women. *Environ Health Perspect* 2019;127:77001.
- Klompmaier JO, Hoek G, Bloemsa LD, et al. Associations of combined exposures to surrounding green, air pollution and traffic noise on mental health. *Environ Int* 2019;129:525–37.
- Vert C, Sánchez-Benavides G, Martínez D, et al. Effect of long-term exposure to air pollution on anxiety and depression in adults: a cross-sectional study. *Int J Hyg Environ Health* 2017;220:1074–80.
- Altuğ H, Fuks KB, Hüls A, et al. Air pollution is associated with depressive symptoms in elderly women with cognitive impairment. *Environ Int* 2020;136:105448.
- Lin H, Guo Y, Kowal P, et al. Exposure to air pollution and tobacco smoking and their combined effects on depression in six low- and middle-income countries. *Br J Psychiatry* 2017;211:157–62.
- Roberts S, Arseneault L, Barratt B, et al. Exploration of NO₂ and PM_{2.5} air pollution and mental health problems using high-resolution data in London-based children from a UK longitudinal cohort study. *Psychiatry Res* 2019;272:8–17.
- Zock J-P, Verheij R, Helbich M, et al. The impact of social capital, land use, air pollution and noise on individual morbidity in Dutch neighbourhoods. *Environ Int* 2018;121:453–60.
- Pun VC, Manjourides J, Suh H. Association of ambient air pollution with depressive and anxiety symptoms in older adults: results from the NSHAP study. *Environ Health Perspect* 2017;125:342–8.
- Petkus AJ, Younan D, Widaman K, et al. Exposure to fine particulate matter and temporal dynamics of episodic memory and depressive symptoms in older women. *Environ Int* 2020;135:105196.
- Chen S, Kong J, Yu F, et al. Psychopathological symptoms under smog: the role of emotion regulation. *Front Psychol* 2017;8:2274.
- Gu X, Guo T, Si Y, et al. Association between ambient air pollution and daily hospital admissions for depression in 75 Chinese cities. *Am J Psychiatry* 2020;177:735–43.
- Wei F, Wu M, Qian S, et al. Association between short-term exposure to ambient air pollution and hospital visits for depression in China. *Sci Total Environ* 2020;724:138207.
- Qiu H, Zhu X, Wang L, et al. Attributable risk of hospital admissions for overall and specific mental disorders due to particulate matter pollution: a time-series study in Chengdu, China. *Environ Res* 2019;170:230–7.
- Wang F, Liu H, Li H, et al. Ambient concentrations of particulate matter and hospitalization for depression in 26 Chinese cities: a case-crossover study. *Environ Int* 2018;114:115–22.
- Kocbach A, Li Y, Yttri KE, et al. Physicochemical characterisation of combustion particles from vehicle exhaust and residential wood smoke. *Part Fibre Toxicol* 2006;3:1.
- Forchhammer L, Loft S, Roursgaard M, et al. Expression of adhesion molecules, monocyte interactions and oxidative stress in human endothelial cells exposed to wood smoke and diesel exhaust particulate matter. *Toxicol Lett* 2012;209:121–8.
- WHO Regional Office for Europe. *Review of evidence on health aspects of air pollution – REVIHAAP project: technical report*. Copenhagen, Denmark: WHO Regional Office for Europe, 2013.
- Petkus AJ, Younan D, Wang X, et al. Particulate air pollutants and trajectories of depressive symptoms in older women. *Am J Geriatr Psychiatry* 2019;27:1083–96.
- Aarnio MA, Kukkonen J, Kangas L. Modelling of particulate matter concentrations and source contributions in the Helsinki metropolitan area in 2008 and 2010. *Boreal Environment Research* 2016;21:445–60.