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Longitudinal effects of environmental noise and air pollution exposure on autism spectrum disorder and attention-deficit/hyperactivity disorder during adolescence and early adulthood: The TRAILS study

Yiran Li [a,*], Tian Xie [a,**], Raniere Dener Cardoso Melo [a], Maaike de Vries [b,c], Jeroen Lakerveld [d], Wilma Zijlema [e,f,g], Catharina A. Hartman [a]

[a] University of Groningen, University Medical Center Groningen, Interdisciplinary Center Psychopathology and Emotion Regulation, Department of Psychiatry, Groningen, Netherlands
[b] University of Groningen, University Medical Center Groningen, Department of Epidemiology, Groningen, Netherlands
[c] University of Groningen, University Medical Center Groningen, Groningen Research Institute for Asthma and COPD (GRIAC), Groningen, Netherlands
[d] Amsterdam UMC, Vrije Universiteit Amsterdam, Department of Epidemiology and Data Science, Amsterdam Public Health Research Institute, Amsterdam, Netherlands
[e] The Barcelona Institute for Global Health (ISGlobal), Doctor Aiguader 88, 08003, Barcelona, Spain
[f] Universitat Pompeu Fabra (UPF), Doctor Aiguader 88, 08003, Barcelona, Spain
[g] CIBER Epidemiolo'gıa y Salud Pública (CIBERESP), Melchor Fernandez ' Almagro, 3-5, 28029, Madrid, Spain

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ABSTRACT

Background: Exposure to ambient noise and air pollution may affect the manifestation and severity of Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD). However, evidence is limited, and most studies solely assessed environmental exposures during pregnancy and early childhood.
Objective: To examine the longitudinal effects of ambient noise and air pollutants on ASD and ADHD symptom severity during adolescence and early adulthood.
Methods: Using a longitudinal design, we included 2750 children between 10 and 12 years old from the TRacking Adolescents' Individual Lives Survey (TRAILS) in the Netherlands, who were assessed in 6 waves from 2001 to 2017. ASD was measured by the Children's Social Behavior Questionnaire and the Adult Social Behavior Questionnaire. ADHD was measured by Child Behavior Checklist and the Adult Behavior Checklist. Ambient noise and air pollution exposures, including Ozone (O_3), soot, sulfur dioxide (SO_2), nitrogen dioxide (NO_2), particulate matter 2.5 ($PM_{2.5}$), and PM_{10} were modeled at the residential level according to standardized protocols. The longitudinal associations between exposures and symptom outcomes were examined using linear mixed models.
Results: We found evidence that higher levels of exposure to PM were associated with more severe ASD and ADHD symptoms. This association decreased over time. We did not observe any other consistent associations of noise or other air pollutants with ASD and ADHD severity.
Conclusion: The current study provides evidence for the negative impact of PM on ASD and ADHD symptoms. We did not find evidence of the negative health impact of other air pollutants and noise exposures on ASD or ADHD symptoms. Our study adds more evidence on the presence of associations between PM air pollution and neurodevelopmental diseases among adolescents and young adults.

1. Introduction

Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) are two common neurodevelopmental disorders with an estimated prevalence of around 1.5% and 5% among children, respectively (Lyall et al., 2017; Polanczyk et al., 2014). Both

* Corresponding author. Interdisciplinary Center Psychopathology and Emotion Regulation, Department of Psychiatry, University Medical Center Groningen, University of Groningen, Groningen, Netherlands.
** Corresponding author. Interdisciplinary Center Psychopathology and Emotion Regulation, Department of Psychiatry, University Medical Center Groningen, University of Groningen, Groningen, Netherlands.
E-mail addresses: y.li@umcg.nl (Y. Li), t.xie@umcg.nl (T. Xie).

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ASD and ADHD are associated with broad functional impairments and reduced quality of life and impose a high disease burden on the individual, the family, and society (Schoemaker and Houwen, 2021). Previous evidence indicated that both genetic and non-genetic factors contribute to the development and persistence of ASD and ADHD (Caye et al., 2016; Hallmayer et al., 2011; Lyall et al., 2014; Sciberras et al., 2017; Suades-Gonzalez et al., 2015). Still, the complicated etiologies of the disorders, as well as why some have a less severe course than others, are only partly understood.

Among the potential non-genetic risk factors, environmental exposures, such as air pollution and noise, have been related to ASD and ADHD (Costa et al., 2019). Some studies support that exposure to air pollution during pregnancy or early childhood is associated with the subsequent onset of ASD and ADHD in childhood; however, existing evidence is not conclusive (Aghaei et al., 2019; Chun et al., 2020b; Dutheil et al., 2021; Flores-Pajot et al., 2016; Fuertes et al., 2016; Gong et al., 2014; Guxens et al., 2016; Jung et al., 2013; Kalkbrenner et al., 2015; Park et al., 2020; Suades-Gonzalez et al., 2015; Fan, 2022). Air pollutants of major health concern are composed of a wide range of pollutants such as ozone (O_3), soot, sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and particulate matter (PM). PM is a mixture of solid and liquid particles of substances suspended in the air. The major components of PM are sulfate, nitrates, ammonia, sodium chloride, black carbon, mineral dust, and water. It can be classified by particle size into e.g. PM_{10} ($\leq$ 10 μm in diameter) and $PM_{2.5}$ ($\leq$ 2.5 μm in diameter) (Chun et al., 2020b). The detrimental impacts of PM and NO_2 on the risk of ASD and ADHD in children were suggested in previous systematic reviews and cohort studies (Aghaei et al., 2019; Dutheil et al., 2021; Flores-Pajot et al., 2016; Fuertes et al., 2016; Jung et al., 2013; Kalkbrenner et al., 2015; Park et al., 2020). On the contrary, a twin study in Sweden did not find an association between pre- or postnatal exposure to nitrogen oxides (NO_x) and PM_{10} and neurodevelopmental disorders in children (Gong et al., 2014). Another cohort study based on four European samples, including the Netherlands, Italy, Spain, and Sweden, also found no associations between prenatal exposure to PM and NO_2 and autism traits among children from 4 to 10 years old (Guxens et al., 2016).

Besides air pollution, environmental noise is also considered a potential environmental risk factor for ASD and ADHD (Schubert et al., 2019, van Kamp and Davies, 2013), but there are only a few studies available, which have shown heterogeneous results. Some studies suggested the potential detrimental roles of environmental noise in the development of ADHD-related symptoms (Forns et al., 2016; Hjortebjerg et al., 2016; Tiesler et al., 2013; Weyde et al., 2017). For instance, one cohort study suggested that increased road traffic noise exposure from birth to 7 years old was associated with higher abnormal hyperactivity/inattention symptoms (Hjortebjerg et al., 2016). However, this association was not observed in two other studies (Essers et al., 2021; Zijlema et al., 2021). So far, there are no studies on the relationship between ASD and exposure to environmental noise, although decreased sound tolerance is one of the most common sensory difficulties among individuals with ASD (Scheerer, 2021). For both ASD and ADHD, it is conceivable that symptom severity is enhanced under conditions of noise in the environment and this needs further study.

Existing literature has focused primarily on exposures during pregnancy or early childhood. Few studies explored the effects in adolescents or adulthood in relation to the developmental course. Although ASD and ADHD have their onset in childhood, symptoms may persist or fluctuate over the life course (Hartman et al., 2016). Adolescence is a critical period with rapid growth and development in the brain (Fuhrmann et al., 2015), therefore, adolescents may be particularly vulnerable to the impact of environmental exposures on their neurocognitive development (Karmiloff-Smith et al., 2014). For example, $PM_{2.5}$ could induce oxidative stress, potentially increasing inflammation of the central nervous system, thus potentially changing brain development (D'Angiulli, 2018). From adolescence to young adulthood, the course of ASD and ADHD may be altered (Chun et al., 2020a; Hartman et al., 2016; Newbury et al., 2019). Some children with milder forms of ASD in their youth no longer meet the criteria for clinical ASD in adulthood (Horwitz et al., 2020a). For ADHD, an approximate estimate is that at least one-third of the children with ADHD will keep the clinical diagnosis, many others will remain symptomatic and impaired in daily functioning but at a subthreshold level, while a minority will fully remit (Biederman et al., 2010; Vos et al., 2022). Individual differences in symptom improvement during adolescence are not well understood; it is important to establish if these different trajectories of ASD and ADHD symptoms may be modulated by environmental exposures such that those who are exposed show less improvement. Therefore, it is important to extend existing research beyond pregnancy and early childhood and examine the (potentially time-varying) associations between environmental exposures and ASD or ADHD in other parts of the lifespan, including adolescence.

In sum, environmental exposures, including noise and air pollution, may play a role in the development and severity of ASD and ADHD symptoms, but the evidence is inconsistent and limited. Further, most studies solely assessed environmental exposures during pregnancy and early childhood, while these exposures may also explain the variable course of ASD and ADHD during adolescence and adulthood. To address these gaps, this study focuses on the longitudinal associations between two key environmental exposures including noise and air pollution, and the course of ASD and ADHD symptom severity during adolescence and early adulthood.

2. Methods

2.1. Study design and participants

The study was embedded in the TRacking Adolescents' Individual Lives Survey (TRAILS; www.trails.nl), an ongoing prospective cohort study focusing on the psychological, physical, and social development of Dutch (pre)adolescents. The design of the TRAILS study was published elsewhere (Huisman et al., 2008; Oldehinkel et al., 2014). In brief, the TRAILS study recruited 10- to 12-year-old children in the north of the Netherlands. TRAILS consists of a cohort representative of the general population (N = 2230) and a high-risk cohort (N = 543), the latter being defined by contact with mental health outpatient clinics before age 11, implying substantial oversampling of children with ASD or ADHD. Participants were examined through questionnaires, interviews, and physical measurements from 2001 every 2–3 years. To examine the longitudinal relationships between environmental exposures and ASD/ADHD symptoms, data from the first (T_1, ages 10–12 years) to the sixth measurement wave (T_6, ages 24–28 years) were used. Our analytical sample included participants who had available measurements of ASD or ADHD for at least one measurement wave. TRAILS was approved by the Dutch Central Committee on Research Involving Human Subjects (CCMO), and informed consent was obtained from participants and parents (when participants were underage) for the different measurement waves.

2.2. The outcome measures: ASD and ADHD severity

2.2.1. Autism spectrum disorder symptoms

ASD symptoms were measured by the 49-item parent-report Children's Social Behavior Questionnaire (CSBQ) from T_1 to T_4 and the 44-item Adult Social Behavior Questionnaire (ASBQ) at T_5 and T_6 (Hartman et al., 2006, 2007). For wave 5, ASD was only collected for the high-risk cohort samples. CSBQ and ASBQ are validated questionnaires to quantify ASD symptoms in both milder and more severe forms for children and adults, respectively (de Bildt et al., 2009; Horwitz et al., 2016, 2020b). In the current study, we used four comparable sub-scales across CSBQ and ASBQ, including reduced contact, difficulties in understanding social information, fear of and resistance to changes, and stereotyped behavior. In total, 30 items were used from the CSBQ and 29 items from

the ASBQ to have the best possible match in terms of consistency over time. To have comparable outcomes over time (given the different number of items for childhood and adulthood), the item mean score on a scale from 0 to 2 was used for the analyses, with higher scores indicating more severe ASD symptoms.

2.2.2. Attention-deficit/hyperactivity disorder symptoms

ADHD symptoms were measured with the parent-report Child Behavior Checklist (CBCL) from T_1 to T_3 and Adult Behavior Checklist (ABCL) at T_5 (Achenbach and Rescorla, 2001, 2003). We used the subscales covering inattention problems and hyperactivity-impulsivity, consisting of 7 items in the CBCL and 13 in the ABCL. To have comparable outcomes over time (given the different number of items for childhood and adulthood), the item mean score on a scale from 0 to 2 was used for the analyses, with higher scores indicating more severe ADHD symptoms.

2.3. Linked environmental exposure data

Environmental exposure data were obtained from the Geoscience and Health Cohort Consortium (GECCO) database (Lakerveld et al., 2020; Timmermans et al., 2018), a centralized collection of longitudinal environmental data on different geospatial levels in the Netherlands. Nationwide environmental-level data on noise and air pollutants exposures were linked to longitudinal individual-level data using postal code information from TRAILS participants at the level of 6-digit postcodes. A 6-digit postcode area is the smallest geographical unit available in the Netherlands. It includes 10–20 households and is approximately 50 by 50 m. Environmental exposure data was mapped to the year in which the participants participated in a particular wave of the cohort study. If there was no available cohort wave in the exact year of exposure, we used the nearest available year wave. The maximum difference between exposure and cohort wave was 3 years.

2.3.1. Assessment of noise exposure

The data from three sources of noise exposure were used, capturing 1) mixed noise from roads, rail, air, industry, and wind turbines, 2) traffic noise from mixed roads, rail, and air, and 3) traffic noise only from roads. The first noise exposure was the cumulative yearly average measurement of mixed noise, which was collected by the Dutch National Georegister (Dutch National Georegister, 2022). The National Institute for Public Health and the Environment (RIVM) modeled mixed noise levels using the standard method (RMV2012) (Benders, 2012). Available records for 2006, 2011, 2017, and the aggregated period 2018–2012 and 2016–2020 were used in the current study. The other two traffic noise exposures were daily average measurements and were gathered by the Netherlands Environmental Assessment Agency. The noise levels for each address were estimated with Empara Noisetool (cell size: 25 m by 25 m). The European Union (EU) standard noise metric of the Level day-evening-night (Lden) was used. Considering the noise in the evening and night is reported as more disturbing, it averaged the noise level of a 24-h period but increased 5 and 10 dB(A) for the values of the evening (19.00–23.00 h) and night (23.00–7.00 h), respectively as a penalty (Wirth, 2004). Traffic noise from mixed roads, rail, and air in 2000, 2004, 2005, 2007, 2008, and 2012, and traffic noise from roads in 2000, 2004, 2007, 2008, 2010, and 2011 were used in the current study.

2.3.2. Assessment of air pollution exposure

The annual average value of outdoor air pollution exposure was predicted based on Land Use Regression (LUR) models following a standardized protocol developed for the European Study of Cohorts for Air Pollution Effects (ESCAPE-project) (Beelen et al., 2013; Cyrys et al., 2012). Data from six air pollutants were used in the current study, including O_3, soot, and SO_2 from 2011 to 2018, NO_2 from 2000 to 2018, $PM_{2.5}$ from 2000 to 2018, and PM_{10} from 2007 to 2018. Detailed information on the assessment of air pollution exposure was described elsewhere (Beelen et al., 2013; Cyrys et al., 2012; Eeftens et al., 2012a, Eeftens et al., 2012b).

2.4. Covariates

The following covariates were accounted for based on previous literature (Zijlema et al., 2021). Time-invariant covariates included sex, age at baseline (Morrell, 2008), parental socioeconomic status (SES) at baseline, problematic pregnancy or child delivery, maternal smoking during pregnancy, mothers' age at child delivery, pregnancy duration, and lifetime parental psychopathology at baseline. Time-variant covariates included follow-up time since enrollment (in years) and children's use of psychostimulant medication. SES was created by averaging standardized indicators, including parental educational level, parental income, and parental occupational level (Amone-P'Olak et al., 2011). The participants were classified to a "lowest 25%", "middle 50%", or "highest 25%" SES status, with the lowest category used as the reference category. Lifetime parental psychopathology was assessed for five psychiatric disorders, including depression, anxiety, severe antisocial behavior, ASD, and ADHD. Fathers or mothers who reported having had depression or anxiety and who had been treated for either (or both) or who reported severe antisocial behavior, ASD, or ADHD, were coded as having had lifetime parental psychopathological problems.

2.5. Statistical analyses

2.5.1. Main analyses

2.5.1.1. Single-pollutant models. First, descriptive analyses were used on demographic characteristics, covariates, exposures, ASD and ADHD scores. The continuous variables were described using mean and standard deviation (SD), and categorical variables were described using frequencies and percentages. Second, for each environmental exposure (three sources of noise exposure and six sources of air pollutants exposure), linear mixed models (LMM) were applied on repeated measures of ASD or ADHD symptoms (T_1-T_6), respectively. Thus, we modeled follow-up time, environmental exposures, and the interaction between follow-up time and exposures. Models were adjusted for the covariates in two steps. First, we adjusted for age at baseline and sex (minimally adjusted models). Second, all other covariates described above were added (fully adjusted models).

2.5.1.2. Multi-pollutant models. Multi-pollutant models were fitted to the data to investigate co-pollutant confounding. Because of the high correlations among three noise exposures, and between two PM exposures, we performed multi-pollutant analyses with one of the noise exposures (e.g., the most comprehensive): mixed noise exposure, and separately analysed the two PM exposures (e.g., $PM_{2.5}$ and PM_{10}) in a multipollutant model with the other air pollutants (in total two multipollutant models for both ASD and ADHD symptoms). We used variance inflation factors (VIFs) to quantify multicollinearity between the exposures. A VIF of 5 was used as a threshold, and the exposures which had VIF greater than 5 were removed.

2.5.2. Imputation procedure

We only imputed waves in which no information on ASD or ADHD was collected (wave 5 for ASD in the general population and waves 4 and 6 for ADHD in both the general population and the high-risk cohort). These waves were missing by design (the particular instrument was not administered at that wave), and therefore they were missing at random. To ensure that the imputed score was valid, we imputed only for participants who had at most one missing value across the measurement waves (4 waves of available data of ASD and 3 of ADHD) (see Supplement for the imputation method). Multiple imputation was conducted for the missing data of covariates. A total of 20

imputed data sets were obtained and subsequently the mean value was used to impute the missing data.

2.5.3. Sensitivity analyses

We performed three sensitivity analyses: (1) using comparable items between children and adult versions of questionnaires for outcomes measurement; (2) using unimputed raw data; (3) adding cohort group as a covariate in the mixed models.

First, children and adult versions of parent-rated questionnaires for ASD and ADHD were used in TRAILS across different waves. For the main analyses, the original scores from these different versions were used. However, the versions of child and adult questionnaires included a number of different items that measured behaviors relevant to different stages of development. For example, the item "does not seek comfort when he/she is hurt or upset" is only included in the children's version measuring ASD behaviors, and the item "only contact he/she has with others is when he/she has to buy something or arrange something" is only included in the adult version. Thus, potential changes in findings may be due to these differences in measurement. Therefore, we re-ran the linear mixed models using only the overlapping items between questionnaires of children and adult versions. The sensitivity analyses were performed to examine whether the main results would be influenced by removing these different developmentally applicable items across different waves. For ASD, we used 18 overlapping items between childhood and adult versions. For ADHD, we used six overlapping items between childhood and adult versions. Second, the analyses were conducted using the raw data to examine if the results were robust after imputation. Third, the study samples consist of one general population cohort and one high-risk cohort. Cohort group might be a confounding factor, and was additionally adjusted in the mixed models.

The statistical analyses were performed using R Statistical Software version 4.0.5 with packages "lme4" (Bates et al., 2011) and a two-sided $p < 0.05$ was considered statistically significant. The environmental exposures were scaled by an interquartile range (IQR) in all the models. As multiple comparisons (18 models in total) were conducted, adjusted *p*-values by false discovery rate (FDR) using the Benjamini-Hochberg procedure were used. The R package "mice" (R Foundation) was used to impute covariates.

3. Results

3.1. Study population

Participant characteristics are shown in Table 1. About 52.4% (1440/2750) were male, and the average age at baseline was 11.11 years (SD: 0.54). The sample consisted of 2750 participants for ASD, and 2481 participants for ADHD. Specifically, there are 11.1% (306/2750), 10.7% (293/2750), 16.0% (441/2750), 44.0% (1211/2750), and 9.8% (269/2750) participants having two, three, four, five, and six waves data of ASD symptoms. There are 12.8% (318/2481), 19.7% (489/2481), and 62.9% (1560/2481) participants having two, three, and four waves data of ADHD symptoms. Following imputation for wave 5 (ASD) and waves 4 and 6 (ADHD), there are 11.1% (306/2750), 10.7% (293/2750), 2.9% (79/2750), 16.1% (443/2750), and 50.9% (1399/2750) participants having two, three, four, five, and six waves data of ASD symptoms. There are 12.9% (319/2481), 2.5% (63/2481), 4.6% (114/2481), 16.6% (411/2481), and 58.9% (1461/2481) participants having two, three, four, five, and six waves data of ADHD symptoms. These missing values were not imputed as they might not be missing at random. Levels of ambient noise and air pollutants in different waves are shown in Table 2.

Table 1
Characteristics of the study population.

	Value
Sex (male), n (%)	1440 (52.4)
Age (year), mean (SD)	
T_1	11.11 (0.54)
T_2	13.44 (0.61)
T_3	16.21 (0.72)
T_4	19.08 (0.63)
T_5	22.23 (0.68)
T_6	25.73 (0.65)
ASD scores, mean (SD)	
T_1 (N = 2714)	0.26 (0.28)
T_2 (N = 2366)	0.25 (0.28)
T_3 (N = 1919)	0.24 (0.28)
T_4 (N = 2062)	0.23 (0.27)
T_5 (N = 1864)	0.22 (0.23)
T_6 (N = 1721)	0.21 (0.26)
ADHD scores, mean (SD)	
T_1 (N = 2368)	0.66 (0.52)
T_2 (N = 2311)	0.52 (0.47)
T_3 (N = 1863)	0.47 (0.45)
T_4 (N = 1959)	0.44 (0.33)
T_5 (N = 1916)	0.37 (0.36)
T_6 (N = 1800)	0.32 (0.30)
Problematic pregnancy or child delivery	
Not at all	1272 (46.3)
A little	1352 (49.2)
Quite some or a lot	126 (4.6)
Maternal smoking during pregnancy (yes), n (%)	815 (29.6)
Mothers' age at child delivery (year), mean (SD)	29.3 (4.1)
Pregnancy duration (week), mean (SD)	39.7 (2.8)
Lifetime parental psychopathology in mother or father (yes), n (%)	1258 (45.7)
Children's use of psychostimulant medication (yes), n (%)	
T_1 (N = 2567)	297 (10.8)
T_2 (N = 2377)	335 (12.2)
T_3 (N = 1926)	241 (8.8)
T_4 (N = 2233)	198 (7.2)
T_5 (N = 1794)	132 (4.8)
T_6 (N = 1580)	144 (5.2)

Note. SD: standard deviation; ASD: autistic spectrum disorder; ADHD: attention-deficit/hyperactivity disorder.
Table 1 shows results after imputation.

Table 2
Environmental exposures across different measurement waves.

Environmental exposures	Value across different measurement waves, mean (SD)					
	T_1	T_2	T_3	T_4	T_5	T_6
Noise (dB (A))						
Mixed noise	NA	NA	54.37 (5.29)	55.47 (5.78)	56.17 (6.22)	54.90 (6.34)
Mixed traffic noise	51.60 (5.67)	51.85 (5.72)	52.24 (5.59)	54.01 (5.05)	NA	NA
Traffic noise road only	50.81 (6.08)	51.10 (6.12)	51.73 (5.69)	53.44 (5.07)	NA	NA
Air pollutants ($\mu g/m^3$)						
O_3	NA	NA	45.32 (2.21)	44.46 (3.40)	46.14 (3.42)	45.80 (3.02)
Soot	NA	NA	0.66 (0.15)	0.76 (0.26)	0.67 (0.26)	0.62 (0.22)
SO_2	NA	NA	0.73 (0.17)	0.80 (0.30)	0.76 (0.39)	0.70 (0.48)
NO_2	17.25 (3.04)	17.74 (3.76)	15.52 (3.37)	16.68 (4.50)	16.63 (4.83)	15.87 (5.41)
$PM_{2.5}$	NA	11.98 (0.57)	12.39 (0.92)	13.41 (1.81)	10.40 (1.78)	8.80 (1.80)
PM_{10}	27.43 (2.15)	26.58 (3.40)	23.17 (1.38)	20.86 (2.18)	17.65 (2.19)	15.81 (2.12)

Note. NA: not available; SD: standardized deviation; PM: particular matter.

3.2. Main results

3.2.1. Single-pollutant models

3.2.1.1. Ambient noise exposure. Exposure to ambient noise was not significantly associated with ASD or ADHD symptoms in either the minimally or fully adjusted models (Tables 3–4).

3.2.1.2. Air pollutants

3.2.1.2.1. $PM_{2.5}$. The minimally adjusted model showed that a higher level of $PM_{2.5}$ exposure was associated with more severe ASD symptoms ($b = 0.037$, $p < 0.001$, adjusted $p = 0.002$; Table 3) and that the effect decreased over time, indicated by the significant interaction effect with time ($b = -0.004$, $p < 0.001$, adjusted $p < 0.001$; Table 3). Similar results were shown in the fully adjusted model, although the main effect was not statistically significant after adjusting for multiple testing ($b = 0.023$, $p = 0.035$, adjusted $p = 0.105$; Table 3).

3.2.1.2.2. Other air pollutants. The results of single-pollutant models showed significant associations of NO_2 and PM_{10} with ASD and O_3 and soot with ADHD. Higher levels of NO_2 or PM_{10} exposures were associated with less ASD symptoms (for NO_2, $b = -0.012$, $p = 0.007$, adjusted $p = 0.032$; for PM_{10}, $b = -0.021$, $p < 0.001$, adjusted $p = 0.003$; Table 3). Regarding ADHD, higher levels of O_3 exposure were associated with more severe ADHD symptoms only in the fully adjusted model ($b =$

Table 3
Effects of environmental exposures on ASD symptoms.

Environmental exposures	Model 1				Model 2			
	b [95% CI]	*SE*	*p*	adjusted *p*[a]	*b* [95% CI]	*SE*	*p*	adjusted *p*[a]
Noise								
Mixed noise								
Main effects	−6.7u[b] [−21.0u, 7.9u]	7.5u	0.366	0.545	−2.1u [−18.0u, 14.0u]	8.1u	0.800	0.901
Interaction with time	0.3u [−1.0u, 1.6u]	0.7u	0.614	0.862	0.1u [−1.4u, 1.5u]	0.7u	0.916	0.963
Mixed traffic noise								
Main effects	−3.1u [−13.0u, 6.9u]	5.1u	0.544	0.653	−1.2u [−11.0u, 8.7u]	5.0u	0.810	0.901
Interaction with time	0.3u [−1.5u, 2.1u]	0.9u	0.735	0.842	0.1u [−1.7u, 1.9u]	0.9u	0.944	0.963
Traffic noise from the road								
Main effects	−1.7u [−12.0u, 8.4u]	5.2u	0.741	0.741	−1.0u [−11.0u, 8.9u]	5.0u	0.851	0.901
Interaction with time	0.5u [−1.3u, 2.3u]	0.92	0.605	0.778	0.3u [−1.5u, 2.1u]	0.9u	0.736	0.946
Air pollutants								
O_3								
Main effects	−24.0u [−46.0u, −1.9u]	11.0u	0.031*	0.080	−13.0u [−36.0u, 11.0u]	12.0u	0.278	0.455
Interaction with time	**3.4u [1.3u, 5.4u]**	**1.0u**	**0.001****	**0.006****	2.2u [0.1u, 4.4u]	1.1u	0.044*	0.113
Soot								
Main effects	9.0u [−12.0u, 30.0u]	10.0u	0.391	0.587	0.6u [−21.0u, 22.0u]	11.0u	0.957	0.957
Interaction with time	−2.0u [−3.9u, −0.1u]	0.9u	0.034*	0.108	−1.1u [−3.1u, 0.9u]	1.0u	0.261	0.470
SO_2								
Main effects	−4.4u [−16.0u, 7.6u]	6.4u	0.470	0.645	−7.8u [−20.0u, 4.8u]	6.4u	0.222	0.455
Interaction with time	−0.1u [−1.1u, 0.8u]	0.48u	0.796	0.843	0.2u [−0.8u, 1.2u]	0.5u	0.714	0.946
NO_2								
Main effects	**−19.0u [−28.0u, −10.0u]**	**4.5u**	**<0.001*****	**<0.001*****	**−12.0u [−21.0u, −3.2u]**	**4.6u**	**0.007****	**0.032***
Interaction with time	1.0u [0.1u, 1.8u]	0.4u	0.026*	0.108	0.4u [−0.5u, 1.2u]	0.4u	0.425	0.695
$PM_{2.5}$								
Main effects	**37.0u [17.0u, 58.0u]**	**10.0u**	**<0.001*****	**0.002****	23.0u [1.6u, 44.0u]	11.0u	0.035*	0.105
Interaction with time	**−4.2u [−6.1u, −2.3u]**	**1.0u**	**<0.001*****	**<0.001*****	**−2.7u [−4.7u, −0.7u]**	**1.0u**	**0.007****	**0.042***
PM_{10}								
Main effects	**−29.0u [−40.0u, −17.0u]**	**5.8u**	**<0.001*****	**<0.001*****	**−21.0u [−33.0u, −9.7u]**	**6.0u**	**<0.001*****	**0.003****
Interaction with time	0.3u [−0.7u, 1.3u]	0.5u	0.559	0.774	−0.1u [−1.1u, 0.9u]	0.5u	0.839	0.963

Note. Model 1 adjusted for follow-up time, the interaction between follow-up time and environmental exposures, age at baseline, and sex.
Model 2 further adjusted for socioeconomic status, children's use of psychostimulant medication, the interaction between follow-up time and sex, the interaction between follow-up time and children's use of psychostimulant medication, problematic pregnancy or child delivery, maternal smoking during pregnancy, mothers' age at child delivery, pregnancy duration, and lifetime parental psychopathology.
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.
ASD: autistic spectrum disorder; CI: confidence interval; PM: particular matter; SE: standard error.
[a] Adjusted *p:* adjust *p*-values were calculated by false discovery rate using the Benjamini-Hochberg procedure.
[b] u: 10^{-3}. Because the estimations are small, we used a unit (10^{-3}) to represent the results. The environmental exposures were scaled by an interquartile range (IQR) in all the models.

Table 4
Effects of environmental exposures on ADHD symptoms.

Environmental exposures	Model 1				Model 2			
	b [95% CI]	*SE*	*p*	adjusted *p*[a]	*b* [95% CI]	*SE*	*p*	adjusted *p*[a]
Noise								
Mixed noise								
Main effects	4.4u[b] [−15.3u, 24.0u]	10.0u	0.663	0.702	9.0u [−11.2u, 29.3u]	10.3u	0.381	0.568
Interaction with time	−0.3u [−2.0u, 1.4u]	0.9u	0.748	0.842	−0.4u [−2.2u, 1.4u]	0.9u	0.674	0.946
Mixed traffic noise								
Main effects	4.3u [−12.4u, 21.0u]	8.5u	0.616	0.693	4.0u [−10.8u, 18.8u]	7.5u	0.593	0.762
Interaction with time	1.6u [−0.1u, 4.2u]	1.3u	0.217	0.355	2.5u [−0.1u, 5.1u]	1.3u	0.056	0.126
Traffic noise road only								
Main effects	7.3u [−8.4u, 22.9u]	8.0u	0.362	0.587	5.8u [−8.1u, 19.8u]	7.1u	0.410	0.568
Interaction with time	1.7u [−0.7u, 4.1u]	1.2u	0.156	0.351	2.5u [0.1u, 4.9u]	1.2u	0.041*	0.113
Air pollutants								
O_3								
Main effects	27.5u [3.7u, 51.3u]	11.9u	0.021*	0.063	**42.5u [18.1u, 66.9u]**	**12.5u**	**0.001****	**0.006****
Interaction with time	−1.3u [−3.4u, 7.8u]	1.0u	0.209	0.355	−2.8u [−5.0u, −0.6u]	1.1u	0.012*	0.054
Soot								
Main effects	**−30.8u [−52.9u, −8.6u]**	**11.1u**	**0.006****	**0.027***	**−42.8u [−65.4u, −20.2u]**	**11.5u**	**<0.001*****	**0.003****
Interaction with time	1.8u [−0.1u, 3.7u]	1.0u	0.062	0.159	**3.1u [1.0u, 5.1u]**	**1.0u**	**0.003****	**0.027***
SO_2								
Main effects	−11.2u [−24.5u, 2.2u]	6.8u	0.098	0.176	−13.7u [−27.0u, −0.4u]	6.8u	0.043*	0.111
Interaction with time	0.7u [−0.3u, 1.6u]	0.5u	0.195	0.355	0.9u [−0.1u, 1.9u]	0.5u	0.083	0.166
NO_2								
Main effects	**−18.3u [−32.3u, −4.4u]**	**7.1u**	**0.010***	**0.036***	−16.1u [−30.1u, −2.1u]	7.1u	0.024*	0.086
Interaction with time	1.3u [0.1u, 2.6u]	0.6u	0.036*	0.108	1.5u [0.2u, 2.8u]	0.6u	0.023*	0.083
$PM_{2.5}$								
Main effects	22.9u [−2.2u, 47.9u]	12.8u	0.073	0.164	16.0u [−10.3u, 42.3u]	13.4u	0.234	0.455
Interaction with time	−1.1u [−3.3u, 1.2u]	1.2u	0.364	0.546	0.1u [−2.4u, 2.5u]	1.2u	0.963	0.963
PM_{10}								
Main effects	−14.9u [−31.9u, 2.0u]	8.6u	0.082	0.164	−9.9u [−27.3u, 7.5u]	8.8u	0.262	0.455
Interaction with time	**−4.7u [−6.1u, −3.3u]**	**0.7u**	**<0.001*****	**<0.001*****	**−4.9u [−6.4u, −3.4u]**	**0.8u**	**<0.001*****	**<0.001*****

Note. Model 1 adjusted for follow-up time, the interaction between follow-up time and environmental exposures, age at baseline, and sex.
Model 2 further adjusted for socioeconomic status, children's use of psychostimulant medication, the interaction between follow-up time and sex, the interaction between follow-up time and children's use of psychostimulant medication, problematic pregnancy or child delivery, maternal smoking during pregnancy, mothers' age at child delivery, pregnancy duration, and lifetime parental psychopathology.
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.
ADHD: attention-deficit/hyperactivity disorder; CI: confidence interval; PM: particular matter; SE: standard error.
[a] Adjusted *p:* adjust *p*-values were calculated by false discovery rate using the Benjamini-Hochberg procedure.
[b] u: 10^{-3}. Because the estimations are small, we used a unit of 10^{-3} to represent the results. The environmental exposures were scaled by an interquartile range (IQR) in all the models.

0.043, $p = 0.001$, adjusted $p = 0.006$; Table 4). Oppositely, higher levels of soot exposure were associated with less ADHD symptoms ($b = -0.043$, $p < 0.001$, adjusted $p = 0.003$; Table 4), and the effect increased over time, indicated by the significant interaction effect with time ($b = 0.003$, $p = 0.003$, adjusted $p = 0.027$; Table 4). NO_2 also showed a significant association with ADHD, but it attenuated to null after adjustment for multiple testing.

3.2.2. Multi-pollutant models

We fitted two multi-pollutant models with respectively $PM_{2.5}$ and PM_{10} exposure in the models. Soot was removed as a predictor from the models due to multicollinearity (VIF > 5). In multi-pollutant models, the associations between $PM_{2.5}$ and ASD symptoms attenuated but the interaction effect with time remained significant (Table 5). The time-decreasing associations between $PM_{2.5}$ and ADHD symptoms also remained significant (Table 5). Of note is that the association of PM_{10} evaluated in the second PM exposure model was also significant and it was similar to the effect found for $PM_{2.5}$. A higher level of PM_{10} exposure was associated with more severe ADHD symptoms and the effect decreased over time (Table 6). The main effect of PM_{10} on ASD symptoms was not significant but the interaction effect with time was significant (Table 6). Noise and other air pollutants did not show significant associations with ASD and ADHD symptoms.

Table 5
Results of multi-pollutant models with $PM_{2.5}$.

Environmental exposures	ASD				ADHD			
	b [95% CI]	*SE*	*p*	adjusted *p*[a]	*b* [95% CI]	*SE*	*p*	adjusted *p*[a]
Noise								
Mixed noise								
Main effects	−8.7u[b] [−33.6u, 16.1u]	12.7u	0.491	0.655	17.3u [−8.8u, 43.3u]	13.3u	0.193	0.436
Interaction with time	0.9u [−1.3u, 3.0u]	1.1u	0.439	0.520	−1.2u [−3.4u, 1.1u]	1.2u	0.304	0.520
Air pollutants								
O_3								
Main effects	−41.2u [−95.1u, 12.6u]	27.4u	0.133	0.278	33.8u [−27.4u, 95.0u]	31.2u	0.278	0.278
Interaction with time	5.1u [0.3u, 9.9u]	2.5u	0.038*	0.078	−0.3u [−5.8u, 5.2u]	2.8u	0.918	0.918
SO_2								
Main effects	−8.9u [−27.1u, 9.4u]	9.3u	0.341	0.682	−0.1u [−19.7u, 19.4u]	10.0u	0.989	0.989
Interaction with time	0.6u [−0.9u, 2.0u]	0.7u	0.439	0.878	−0.1u [−1.6u, 1.4u]	0.8u	0.942	0.976
NO_2								
Main effects	−18.4u [−95.1u, 12.6u]	22.1u	0.406	0.406	−37.8u [−86.8u, 11.1u]	24.9u	0.129	0.334
Interaction with time	2.9u [−1.0u, 6.9u]	2.0u	0.143	0.143	5.2u [0.8u, 9.6u]	2.2u	0.019*	0.076
$PM_{2.5}$								
Main effects	12.9u [−21.2u, 47.0u]	17.3u	0.454	0.454	**69.0u [29.3u, 108.7u]**	**20.2u**	**<0.001*****	**<0.001*****
Interaction with time	**−3.9u [−7.2u, −0.6u]**	**1.7u**	**0.018***	**0.018***	**−7.9u [−11.4u, −4.3u]**	**1.8u**	**<0.001*****	**<0.001*****

Note.
*$p < 0.05$.
**$p < 0.01$.
***$p < 0.001$.
ASD: autistic spectrum disorder; ADHD: attention-deficit/hyperactivity disorder; CI: confidence interval; PM: particular matter; SE: standard error.
[a] Adjusted *p:* adjust *p*-values were calculated by false discovery rate using the Benjamini-Hochberg procedure.
[b] u: 10^{-3}. Because the estimations are small, we used a unit of 10^{-3} to represent the results. The environmental exposures were scaled by an interquartile range (IQR) in all the models.

3.3. Sensitivity analyses

Sensitivity analyses generated similar results compared to the single-pollutant models of main analyses. Noise level was not associated with ASD or ADHD symptoms. For air pollutants, we found a similar time-decreasing effect of $PM_{2.5}$ on ASD and ADHD symptoms (Stables 3–5). Higher levels of $PM_{2.5}$ exposures were associated with more severe ASD and ADHD symptoms and the associations decreased over time. The associations of NO_2 and PM_{10} were also significant in the sensitivity analyses using raw data. Higher levels of NO_2 or PM_{10} exposures were associated with less ASD and ADHD symptoms.

4. Discussion

We investigated the potential effects of environmental exposures on ASD and ADHD symptoms over a span of 15 years among adolescents and young adults using 6-time-point repeated measurement data. We found that higher PM levels were associated with higher ASD and ADHD symptoms. However, this effect decreased with time. We did not find evidence for robust effects of other air pollutants and noise on ASD or ADHD symptoms.

Among the air pollutants involved in the study, we found robust evidence for the effect of $PM_{2.5}$ on ASD symptoms, which was consistent between the single-pollutant model and the models tested in sensitivity analyses, and which remained in the multi-pollutant analyses. Previous studies of pre- or postnatal exposure suggested stronger evidence of the detrimental effects of $PM_{2.5}$ on ASD and ADHD compared to other air pollutants (Aghaei et al., 2019; Dutheil et al., 2021; Fuertes et al., 2016). Our tentative findings were thus in line with previous studies regarding the associations between $PM_{2.5}$ and neurodevelopmental disorders (Dutheil et al., 2021; Fuertes et al., 2016). We also found some evidence of PM_{10}, which was only identified in the multi-pollutant analyses. This suggests that the null effect in the single-pollutant models potentially resulted from confounding by other exposures. $PM_{2.5}$ may be more harmful than PM_{10} because it penetrates the lung more rapidly, easily, and deeply, while PM_{10} is more likely to deposit on the surfaces of the lung (de Kok et al., 2006). Our study adds to the literature that the associations might also exist during the period of adolescents and young adults after the onset of ASD and ADHD. Mechanisms driving PM-induced detrimental effects on the development or continuation of neurodevelopmental disorders are not well understood. Some evidence suggests that PM provokes astrocyte and microglial activation, triggers inflammation, and activates oxidative stress, which may be involved in the etiology of ASD and ADHD (Ahadullah et al., 2021; Bonvicini et al., 2018; Ceylan et al., 2012), providing a plausible link (Heusinkveld et al., 2016; Onoda et al., 2017). Considering the detrimental health impacts, PM should remain to be considered a possible risk factor for neurodevelopment in general and neurodevelopmental disorders in particular. Still, more studies are needed to confirm our and previous results and to better understand the underlying mechanisms.

Except for the effect of PM, we did not find evidence for robust effects of other air pollutants on ASD or ADHD symptoms, meaning that effects were inconsistent between main and sensitivity analyses and changed to null findings in the multi-pollutant models. Therefore, the observed but inconsistent associations of other air pollutants may be chance findings or driven by other exposures, such as PM. Compared with the existing

Table 6
Results of multi-pollutant models with PM_{10}.

Environmental exposures	ASD				ADHD			
	b [95% CI]	*SE*	*p*	adjusted *p*[a]	*b* [95% CI]	*SE*	*p*	adjusted *p*[a]
Noise								
Mixed noise								
Main effects	−5.0u[b] [−28.1u, 18.2u]	11.8u	0.675	0.675	16.4u [−9.7u, 42.4u]	13.3u	0.218	0.436
Interaction with time	0.7u [−13.6u, 2.7u]	1.0u	0.520	0.520	−1.1u [−3.3u, 1.2u]	1.2u	0.347	0.520
Air pollutants								
O_3								
Main effects	−42.2u [−101.6u, 16.9u]	30.1u	0.161	0.278	41.5u [−24.8u, 107.6u]	33.6u	0.217	0.278
Interaction with time	5.5u [0.3u, 10.8u]	2.7u	0.039*	0.078	−1.1u [−6.9u, 4.8u]	3.0u	0.718	0.918
SO_2								
Main effects	−13.6u [−31.5u, 4.4u]	9.1u	0.139	0.556	−1.3u [−20.9u, 18.3u]	10.0u	0.896	0.989
Interaction with time	1.0u [−0.4u, 2.4u]	0.7u	0.174	0.696	0.1u [−1.5u, 1.5u]	0.8u	0.976	0.976
NO_2								
Main effects	−25.4u [−70.5u, 19.8u]	23.0u	0.270	0.360	−35.4u [−85.7u, 14.9u]	25.6u	0.167	0.334
Interaction with time	4.0u [−0.1u, 8.1u]	2.1u	0.057	0.076	4.8u [0.2u, 9.4u]	2.3u	0.040*	0.076
PM_{10}								
Main effects	50.3u [−21.7u, 122.0u]	36.6u	0.169	0.225	**143u [62.3u, 223.6u]**	**41.4u**	**<0.001*** **	**<0.001*** **
Interaction with time	**−10.9u [−17.1u, −4.8u]**	**3.1u**	**<0.001*** **	**<0.001*** **	**−15.0u [−21.8u, −8.2u]**	**3.5u**	**<0.001*** **	**<0.001*** **

Note.
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.
ASD: autistic spectrum disorder; ADHD: attention-deficit/hyperactivity disorder; CI: confidence interval; PM: particular matter; SE: standard error.
[a] Adjusted *p:* adjust *p*-values were calculated by false discovery rate using the Benjamini-Hochberg procedure.
[b] u: 10^{-3}. Because the estimations are small, we used a unit of 10^{-3} to represent the results. The environmental exposures were scaled by an interquartile range (IQR) in all the models.

literature examined during early life, our study is in agreement with most studies on other air pollutants conducted in some European countries, including the Netherlands, Sweden, Italy, and Spain (Gong et al., 2014; Guxens et al., 2016). In contrast, some studies in the United States, Korea, and Taiwan did find associations of other air pollutants with both ASD and ADHD (Jung et al., 2013; Kalkbrenner et al., 2015; Park et al., 2020). The differences may be related to different sources and levels of air pollutants between the Netherlands and elsewhere. For example, previous studies reported mean levels of NO_2 in California and Taiwan ranged from 32 to 43 μg/m^3 (Becerra et al., 2013; Jung et al., 2013; Volk et al., 2013), which is higher compared with our study (16 μg/m^3 on average). Possibly we did not capture the associations that may only exist in higher levels of air pollutants. Alternatively, the identified (absence of) associations may be false positives or negatives.

With regard to finding no associations between noise exposure with ASD and ADHD symptoms, our result is consistent with two previous studies reporting on ADHD. One study examined the relationship in children from a Spanish and Dutch birth cohort using 3- and 4-time-point longitudinal data. They did not observe an association between prenatal or childhood exposures to noise and ADHD (Essers et al., 2021). Also, a previous cross-sectional study based on the TRAILS baseline data reported an unexpected small protective association between residential or school traffic noise and ASD diagnosis and no association with ADHD symptoms in Dutch children aged 10–12 years (Zijlema et al., 2021). However, a review paper summarizing the association between transportation noise and behavioral and emotional disorders suggested a negative health effect of exposure to road traffic noise on increased hyperactivity/inattention symptoms in children (Schubert et al., 2019). All of the previous studies in this review were conducted on children (age range: 9–10), and most of them examined noise exposure at school instead of the residential address, which may have played a role in the different findings compared to ours. With limited evidence available in adolescents and adults for ADHD, and no previous studies done so far on ASD, future longitudinal studies are warranted. That said, our current, thorough, study unequivocally indicated that this symptom severity is not enhanced under conditions of noise in the environment.

4.1. Strengths and limitations

This study was based on a well-designed cohort and a centralized collection of high-resolution longitudinal exposure data that enabled us to assess the longitudinal associations with 6-wave repeated measurements of both environmental exposures and outcomes. We assigned exposure predictors based on the residential address in different waves, providing precise measurements of exposures at the individual level during the whole period. The environmental exposures and symptom outcomes were included as continuous variables, allowing us to examine the associations on a wide spectrum. We adjusted for many socio-demographic and perinatal covariates potentially associated with environmental exposures and ASD and ADHD symptom severity. Additionally, using LMM, we modeled ASD and ADHD symptoms and time-varying exposures in the best possible way.

However, there are some limitations in the current study. First, only exposures at the residential address were evaluated in our study. Including multiple sources of exposure, such as school and work, may provide a more comprehensive and accurate estimation of the exposure levels. Second, we cannot rule out the possibility of bias due to the loss to follow-up. Although TRAILS has high retention rates (73%–96%), we already know that attrition in TRAILS is not random (Oldehinkel et al., 2015). Those who dropped out had a higher ASD score at baseline.

Possibly, this may explain why the effect of PM was reduced over time. Third, the participants in the study were all from the north of the Netherlands. Generalization of the results to other geographical locations with different levels of exposure should be applied with caution. Future studies from different countries are needed to further examine the effects of ambient noise and air pollution on ASD and ADHD.

5. Conclusion

To conclude, our study found evidence for a positive time-decreasing association between PM exposure and ASD and ADHD symptoms. It suggests that PM air pollution (especially $PM_{2.5}$) is a risk factor for neurodevelopmental symptoms. We did not find evidence of the negative health impact of other air pollutants and noise exposures on ASD or ADHD. Our study adds more evidence on the presence of associations between PM air pollution and neurodevelopmental diseases among adolescents and young adults.

Credit author statement

Yiran Li: Conceptualization, Methodology, Formal analysis, Writing – original draft, Writing – editing. **Tian Xie**: Methodology, Writing – review & editing, Supervision. **Raniere Dener Cardoso Melo**: Methodology, Writing – review & editing. **Maaike de Vries**: Writing – review & editing. **Jeroen Lakerveld**: Resources, Writing – review & editing. **Wilma Zijlema**: Methodology, Writing – review & editing. **Catharina A. Hartman**: Conceptualization, Methodology, Writing – review & editing, Supervision.

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TRAILS was approved by the Dutch Central Committee on Research Involving Human Subjects (CCMO), and informed consent was obtained from participants and parents (when participants were underage) for the different measurement waves.

Ethics approval

This study used the data from the TRAILS, which was approved by the Dutch Central Committee on Research Involving Human Subjects (CCMO).

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The authors do not have permission to share data.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2023.115704.

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