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Air pollution, traffic noise, mental health, and cognitive development: A multi-exposure longitudinal study of London adolescents in the SCAMP cohort

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ABSTRACT

Background: There is increasing evidence that air pollution and noise may have detrimental psychological impacts, but there are few studies evaluating adolescents, ground-level ozone exposure, multi-exposure models, or metrics beyond outdoor residential exposure. This study aimed to address these gaps.

Methods: Annual air pollution and traffic noise exposure at home and school were modelled for adolescents in the Greater London SCAMP cohort (N=7555). Indoor, outdoor and hybrid environments were modelled for air pollution. Cognitive and mental health measures were self-completed at two timepoints (baseline aged 11–12 and follow-up aged 13–15). Associations were modelled using multi-level multivariate linear or ordinal logistic regression.

Results: This is the first study to investigate ground-level ozone exposure in relation to adolescent executive functioning, finding that a 1 interquartile range increase in outdoor ozone corresponded to −0.06 ($p < 0.001$) z-score between baseline and follow-up, 38 % less improvement than average (median development + 0.16). Exposure to nitrogen dioxide (NO_2), 24-hour traffic noise, and particulate matter < 10 μg/m^3 (PM_{10}) were also significantly associated with slower executive functioning development when adjusting for ozone. In two-pollutant models, particulate matter and ozone were associated with increased externalising problems. Daytime and evening noise were associated with higher anxiety symptoms, and 24-hour noise with worse speech-in-noise perception (auditory processing). Adjusting for air pollutants, 24-hour noise was also associated with higher anxiety symptoms and slower fluid intelligence development.

Conclusions: Ozone's potentially detrimental effects on adolescent cognition have been overlooked in the literature. Our findings also suggest harmful impacts of other air pollutants and noise on mental health. Further

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research should attempt to replicate these findings and use mechanistic enquiry to enhance causal inference. Policy makers should carefully consider how to manage the public health impacts of ozone, as efforts to reduce other air pollutants such as NO_2 can increase ozone levels, as will the progression of climate change.

1. Introduction

Understanding the risk and resilience factors for mental health and cognitive development is of crucial importance. Mental health conditions lead to high levels of distress and disability globally (Dakić, 2020), and cognitive abilities are interrelated with mental health, with social, economic, and health impacts (Batty and Deary, 2004). Air and noise pollution are major public health concerns with known risk to physical health (Basner et al., 2014, Landrigan, 2017).

Adolescence is associated with cognitive development and the emergence of socio-emotional mental health disorders (Blakemore and Choudhury, 2006, Kessler et al., 2005). Air pollution is thought to affect the brain via systemic inflammation, oxidative stress, and, for some pollutants, direct interaction (Block et al., 2012). Until a person's mid-twenties, brain development is characterised by the establishment and fine tuning of neural connections, so adolescent brains may be more vulnerable to damage from environmental pollutants than adult brains (Fuhrmann et al., 2015). Physiological effects of noise are mainly driven by chronic arousal or stress, associated with a variety of physical and mental health problems, and distraction from education, annoyance, learned helplessness, and sleep disruption could also impact cognitive and mental health (Stansfeld and Clark, 2015).

Systematic reviews and *meta*-analyses have reported detrimental associations between exposure to transportation noise and air pollution, especially particulate matter and NO_2, and a range of mental health and cognitive outcomes (Braithwaite et al., 2019, Thompson et al., 2023, Thompson et al., 2022, Schubert et al., 2019, Dzhambov and Lercher, 2019). The existing literature has a limited number of adolescent studies, with many failing to distinguish between children and adolescents. The psychological changes undergone in adolescence are different to those undergone in childhood, so impacts of environmental stressors could differ. Moreover, ozone's cognitive impacts have not been extensively investigated in younger populations, and existing evidence around ozone and mental health is inconclusive (Zhao et al., 2018) Most studies have not used multi-pollutant or multi-exposure models, and the possibility of mutual confounding or interactive effects makes such studies a high priority (Foraster, 2013). Most studies have evaluated outdoor exposure to air pollution at the residential address only, rather than indoors and/or at other frequented settings such as schools, which may have led to exposure misclassification.

This longitudinal study of air pollution, traffic noise, and psychological outcomes in the Study of Cognition, Adolescents and Mobile Phones (SCAMP) cohort (Toledano et al., 2019) aims to address these gaps. It was hypothesised that higher levels of exposure to air pollution and noise would be associated with poorer mental health and cognitive development over time.

2. Materials and methods

2.1. Participants

SCAMP is a London-based longitudinal cohort in the United Kingdom (Fig. 1). Eligible schools were identified from the Department of Education's register (EduBase) and the 2012 school census and selected to be representative of general population adolescents in London (Toledano et al., 2019). Of 206 schools invited to take part, 35 agreed to take part and a further 8 eligibles schools asked to participate, with 4 schools subsequently dropping out (final total 39). Baseline assessments

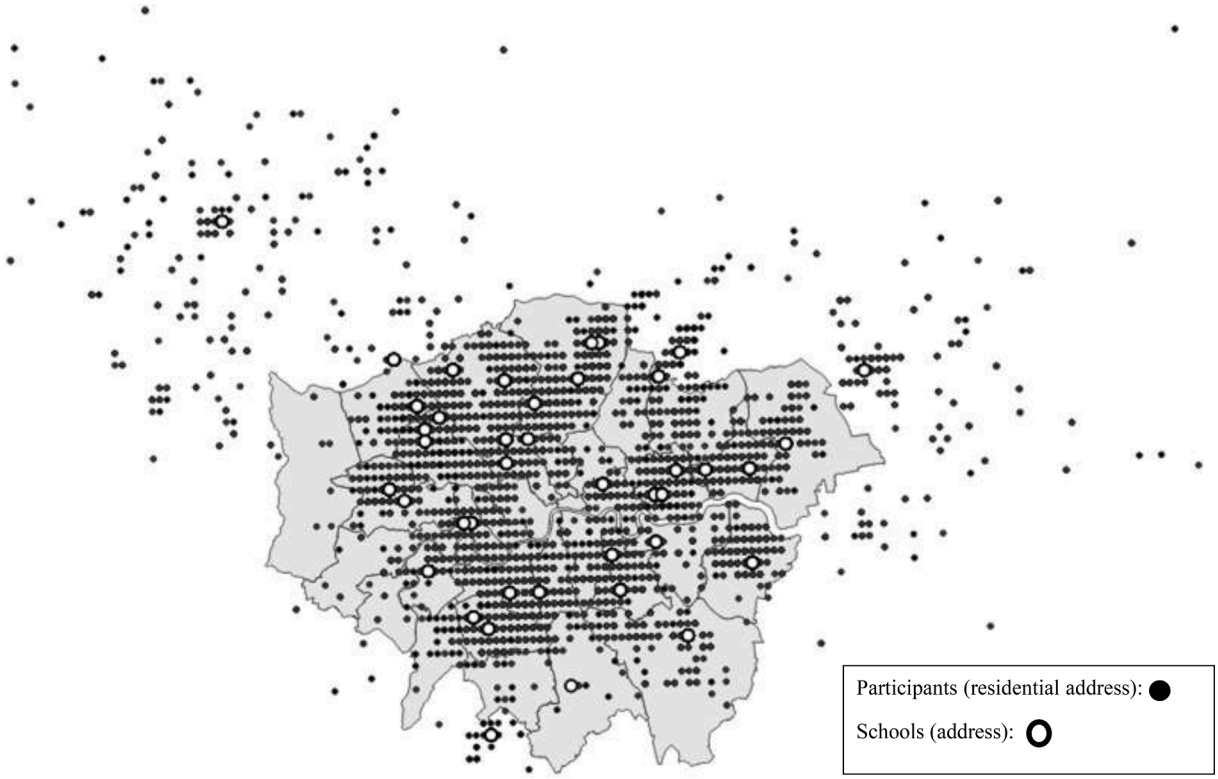

Fig. 1. Location of SCAMP participants and participating schools (1 km resolution).

took place at age 11–12 years (2014–2016), and follow-up assessments at age 13–15 years (2016–2018). The sample was roughly balanced for sex (51.5 % female) and represented a wide range of ethnic and socio-demographic groups (Toledano et al., 2019).

2.2. Exposures

2.2.1. Air pollution

Yearly exposure estimates (2013–2018) were produced for NO_2, $PM_{2.5}$, PM_{10} (all in $\mu g/m^3$) and ozone (in ppb) in the following settings: outdoors (at home and school, time-weighted at school 6 h/day, 190 days/year), indoors (at home and school), and hybrid (indoors and outdoors, at home, school, and whilst travelling). Two estimates were generated for each of indoor and hybrid exposure, one including indoor sources of air pollution (from cooking) and one excluding indoor sources (only including the ingress of outdoor air pollutants). Table 1 shows the different settings, sources and models contributing to the exposure estimates. Indoor and hybrid models were enhanced with air pollution measurements and time-activity data (collected during 2015–2018 from N=193 sub-study participants), as well as cooking fuel data and questionnaire data about cooking behaviours. More information about how air pollution exposure was estimated can be found in supplementary information 1 (SI1).

2.2.2. Noise

Based on home and school addresses, annual average road traffic noise outdoors was modelled for 2013–2018 using the Common noise assessment methods in Europe (CNOSSOS-EU) model. Traffic noise is pervasive to all urban residents (most of the cohort were not highly exposed to aircraft or rail noise) and is the type of noise pollution most concerning to public health researchers and professionals at present. The noise metrics Lden (24-hour A-weighted Leq [equivalent noise level], including + 5 dB penalty during the evening, and + 10db penalty during the night), Lday (daytime A-weighted Leq from 7am-7 pm), Leve (A-weighted Leq from 7 pm-11 pm), and Lnight (A-weighted Leq from 11 pm-7am) were used. Lden and Lday were time-weighted as 6 h/day, 190 days/year at school and only home exposure was relevant for Leve and Lnight. A-weighting accounts for human exposure by weighting towards noise levels within the spectrum of typical human hearing.

Noise exposure was estimated for the whole cohort providing address data, whereas air pollution exposure could only be estimated for residents within the M25, a 117-mile orbital motorway encompassing London (45.6 % of cohort), the geographical extent of the CMAQ-urban/LHEM model. Individual-level exposure windows were calculated to reflect baseline exposure (annual average for the year prior to baseline assessment), and long-term follow-up exposure (average annual exposure between baseline and follow-up).

Table 1
Air pollution sources/settings and models contributing to the different exposure estimates.

Air pollution sources/ settings	**Exposure estimate**				
	Outdoors	Hybrid with cooking sources	Hybrid without cooking sources	Indoors with cooking sources	Indoors without cooking sources
Home outdoor	x	x	x		
Home indoor (infiltration from outdoors only)		x	x	x	x
Home indoor cooking sources		x		x	
School outdoor	x	x	x		
School indoor (infiltration from outdoors only)		x	x		
Travelling		x	x		
Model:	CMAQ-urban	London Hybrid Exposure Model	London Hybrid Exposure Model	Indoor mass balance model	Indoor mass balance model

2.2.3. Outcomes

Cognitive and mental health tasks and questionnaires were self-completed by adolescents on a computer, tablet or smartphone using the Psytools (Delosis Ltd) online platform. These measured emotional and behavioural problems (SDQ total score), externalising problems (SDQ), internalising problems (SDQ), executive functioning, fluid intelligence, processing speed, speech-in-noise perception, depression (PHQ-9) and anxiety (GAD-7) symptoms. The specific metrics are described in SI1.

2.2.4. Covariates

Plausible confounders were selected a priori based on the literature (personal characteristics self-reported via questionnaire): age, parental National Statistics Socio-economic classification (NS-SEC), ethnicity (white, black, Asian, mixed or other ethnicity), sex (male or female), first language (English, not English, or bilingual including English), parental education (mother attended university, yes/no), school type (independent/state), neighbourhood (postcode sector), and school (data collection site).

2.3. Statistical analysis

Missing covariate data were imputed using non-parametric random forest imputation (using missForest package in R 4.0.3). Linear regression was used for all outcomes except the PHQ-9 and GAD-7, which (due to skewedness) were categorised into mild, moderate, severe or no depression/anxiety and analysed using ordinal logistic regression. Adjusted associations were estimated using multi-level multivariate regression models (lme4 package in R 4.0.3, and ologit function in Stata 13). Air pollution exposure was analysed per 1 interquartile (IQR) increase and noise exposure in quartiles. Individual-level covariates were entered as fixed effects, and school and neighbourhood as random intercepts. Baseline outcomes were predicted from year-prior exposure, follow-up outcomes from exposure between baseline and follow-up. Longitudinal changes in outcome were indexed by adjusting for baseline outcome score as a covariate predictor of follow-up absolute score, however depression and anxiety were only reported at follow-up so not adjusted for baseline score. Two-exposure models were run where collinearity allowed (Pearson's r between exposures < 0.80). Substantial missing data created greater uncertainty for indoor and hybrid models than outdoor models, so outdoor exposure was used for the main analysis.

2.4. Sensitivity analyses

Outdoor exposure effects were compared to effects using the other exposure metrics (hybrid and indoor air pollution). For comparability with air pollution analyses in terms of geographical spread and sample size, noise exposure analyses were repeated for participants within the M25 only. Finally, analyses were run excluding those who had provided ambiguous address data (that is, geocodes differing at 3dp between methods that prioritised first line of address or postcode).

2.5. Role of the funding source

The study sponsors had no role in study design; in the collection,

analysis, and interpretation of data; in the writing of the report; or in the decision to submit the paper for publication.

3. Results

Sample characteristics for the analytical sample are found in Table 2. A total of 7744 participants took part in baseline, follow-up, or both, of whom 7603 provided address data, and 7555 of these provided data on at least one outcome (the analytical sample). Noise exposure was estimated for 7555, whereas air pollution exposure was estimated for 3529 resident within the M25. Sample sizes varied between analyses by exposure, outcome, and timepoint (N=3792 baseline participants retained at follow-up, N=1112 joined at follow-up, follow-up time range 18–42 months, mean 26.74 months). Fig. 2 (flow diagram) shows the analytical sample and participation at each timepoint. The sociodemographic differences between baseline and follow-up were small. Mental health and behavioural difficulties tended to increase between baseline and follow-up, and cognitive skills tended to improve (supplementary information tables SI2 and SI3 provide descriptive statistics of mental health, cognitive, and exposure variables).

Table 3 shows associations between exposure to outdoor air pollution and psychological outcomes at follow-up. After adjustment for covariates and ozone, higher PM_{10} exposure between baseline and follow-up was associated with increased total emotional and behavioural problems ($\beta = 0.76$, $p = 0.030$), and externalising problems ($\beta = 0.44 p = 0.036$). In a multi-exposure model containing $PM_{2.5}$ and ozone, higher levels of both were associated with increased externalising problems ($PM_{2.5}$ $\beta = 0.55$, $p = 0.011$ & ozone $\beta = 0.45$, $p = 0.026$). In single exposure models, higher ozone exposure was associated with slower executive functioning development between baseline and follow-up ($\beta = -0.06$, $p < 0.001$), but higher levels of $PM_{2.5}$ and NO_2 were associated with faster executive functioning development ($\beta = 0.04$, $p = 0.045$ & $\beta = 0.04$, $p = 0.007$, respectively). However, adjusted for ozone all other pollutants were associated with slower executive function development ($\beta = -0.06$ to -0.12, $p = 0.010$ to 0.062) and higher ozone remained strongly associated with slower executive function development ($\beta = -0.11$ to -0.18, $p \leq 0.001$). At baseline, higher levels of PM_{10} and NO_2 over the previous year were associated with more emotional and behavioural problems at baseline, and ozone with less, particularly for externalising problems (see Tables SI4, SI6b, SI6c, Supplementary information). Baseline executive functioning was not associated with air pollution in single-exposure models (SI4), however in combined models of PM_{10} and ozone, both were associated with significantly worse executive functioning (SI6b), with the same pattern for NO_2 and ozone (SI6c).

Table 4 shows adjusted associations between exposure to traffic noise and psychological outcomes at follow-up. Higher levels of evening noise between baseline and follow-up were associated with greater follow-up anxiety symptoms (4th quartile $\beta = 0.24$, $p = 0.046$), as was day-time noise (3rd quartile $\beta = 0.33$, $p = 0.010$) with 24-hour (4th quartile $\beta = 0.20$, $p = 0.090$) and night-time noise approaching significance (4th quartile $\beta = 0.21$, $p = 0.074$). More 24-hour noise between baseline and follow-up was associated with worse speech-in-noise perception between baseline and follow-up (3rd quartile $\beta = 0.49$, $p = 0.035$). Consistent with the findings for anxiety, higher levels of baseline 24-hour noise, evening noise, and night-time noise were associated with worse mental health (emotional and behavioural problems), especially internalising problems (see Supplementary Information Table SI5).

Table 5 shows adjusted associations between co-exposure to outdoor air pollution and traffic noise and psychological outcomes at follow-up. Adjustment for ozone revealed a significant association between more 24-hour noise and slower executive functioning development (ozone $\beta = -0.07$, $p < 0.001$, Lden 4th quartile $\beta = -0.06$, $p = 0.044$) and with adjustment for any pollutant, daytime noise was non-significantly associated with slower executive function development. Adjustment for air pollution revealed associations between more 24-hour noise and slower fluid intelligence development (3rd quartile $\beta = -0.31$ to -0.32, $p = 0.047$ to 0.053). Associations between more 24-hour noise and increased anxiety were strengthened with adjustment for air pollutants (e.g. adjusted for NO_2, Lden 4th quartile $\beta = 0.28$, $p = 0.035$) whereas associations with daytime noise were slightly weakened (e.g. adjusted for PM_{10}, Lday 3rd quartile $\beta = 0.26$, $p = 0.053$).

Table 2
Sociodemographics of analytical sample after imputation (N=7555).

Characteristic	Total analytical sample	Baseline sample	Follow-up sample
Age		*M*=11.62 (*SD*=0.48)	*M*=13.81 (*SD*=0.56)
N	7555	6443	4904
Sex			
Female	3889 (51.5 %)	3387 (52.6 %)	2677 (54.6 %)
Male	3666 (48.5 %)	3056 (47.4 %)	2227 (45.4 %)
Parental National Statistics Socio-economic Classification (NS-SEC)[1]			
1–2: Managerial, administrative, and professional occupations	2215 (29.3 %)	1855 (28.8 %)	1560 (31.8 %)
3–4: Intermediate occupations	1857 (24.6 %)	1601 (24.8 %)	1221 (24.9 %)
5–7: Routine and manual occupations	2912 (38.5 %)	2584 (40.1 %)	1552 (31.6 %)
8: never worked or long-term unemployed	571 (7.6 %)	403 (6.3 %)	571 (11.6 %)
Ethnicity			
White	3356 (44.4 %)	2853 (44.3 %)	2253 (45.9 %)
Black	1260 (16.7 %)	1076 (16.7 %)	788 (16.1 %)
Asian	2105 (27.9 %)	1800 (27.9 %)	1349 (27.5 %)
Mixed race	771 (10.2 %)	660 (10.2 %)	476 (9.7 %)
Other ethnic group	63 (0.8 %)	54 (0.8 %)	38 (0.8 %)
State school	5858 (77.5 %)	4979 (77.3 %)	3623 (73.9 %)
Mother attended university	4837 (64.0 %)	4151 (64.4 %)	3042 (62.0 %)
First Language			
Was English	4433 (58.7 %)	3823 (59.4 %)	2931 (59.8 %)
Was not English	1612 (21.3 %)	1365 (21.2 %)	1089 (22.2 %)
Learnt English at the same time as another language	1510 (20.0 %)	1255 (19.5 %)	884 (18.0 %)

Missing data on age, parental education, first language, socioeconomic status, and ethnicity was imputed using non-parametric missing value random forest imputation. Max imputed = 35.7 %, min imputed = 0.5 %, mean imputed = 19.4 %.

[1] If multiple parental occupations reported, highest taken.

3.1. Sensitivity analyses

Results of noise analyses excluding participants outside the M25 were consistent with the wider sample (table SI7). In contrast with outdoor exposure, hybrid PM_{10} exposure including indoor sources (SI8) was associated with higher follow-up anxiety symptoms (adjusted $\beta = 0.10$, $p = 0.042$) but not increased emotional and behavioural problems. Findings for executive functioning were consistent with the main analysis and comparable findings were found for hybrid exposure without indoor sources (SI9). For indoor exposure including indoor sources (SI10), higher PM_{10} was associated with follow-up depression and anxiety symptoms ($\beta = 0.12$, $p = 0.024$ and $\beta = 0.12$, $p = 0.028$, respectively) but not emotional and behavioural problems, and associations between air pollutants and executive function development were no longer significant. For indoor air pollution without indoor sources

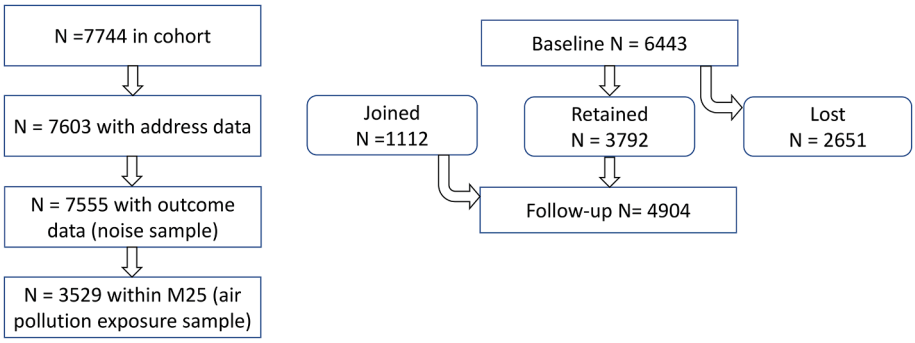

Fig. 2. Flow diagram of analytical sample and participation by timepoint.

Table 3
Adjusted associations between outdoor $PM_{2.5}$, PM_{10}, NO_2, and ozone (per interquartile range (IQR) increase) and psychological outcomes at follow-up, in single and multi-exposure models.

			$PM_{2.5}$			PM_{10}			NO_2			Ozone		
Follow-up outcomes	***Model***	**N**	β	**SE**	***p***	**β**	**SE**	***p***	**β**	**SE**	***p***	**β**	**SE**	***p***
SDQ emotional and behavioural problems[a]	**Single exposure**	2937	*0.331*	*0.180*	*0.067*	−0.024	0.145	0.871	−0.087	0.134	0.519	0.134	0.153	0.385
	$PM_{2.5}$ + ozone	2937	*0.616*	*0.359*	*0.091*							0.314	0.341	0.360
	PM_{10} + ozone	2937				**0.758**	**0.343**	**0.030**				0.540	0.364	0.144
	NO_2 + ozone	2937							0.293	0.536	0.585	0.109	0.624	0.861
SDQ externalising problems[a]	**Single exposure**	2937	0.139	0.106	0.188	0.097	0.094	0.305	0.049	0.088	0.574	−0.017	0.101	0.865
	$PM_{2.5}$ + ozone	2937	**0.545**	**0.207**	**0.011**							**0.450**	**0.198**	**0.026**
	PM_{10} + ozone	2937				**0.444**	**0.208**	**0.036**				*0.416*	*0.222*	*0.065*
	NO_2 + ozone	2937							0.446	0.312	0.154	0.480	0.360	0.184
Executive functioning[b]	**Single exposure**	2785	**0.036**	**0.018**	**0.045**	*0.030*	*0.016*	*0.057*	**0.038**	**0.014**	**0.007**	**−0.058**	**0.016**	**<0.001**
	$PM_{2.5}$ + ozone	2785	*−0.063*	*0.033*	*0.062*							**−0.111**	**0.032**	**0.001**
	PM_{10} + ozone	2785				**−0.085**	**0.032**	**0.010**				**−0.140**	**0.034**	**<0.001**
	NO_2 + ozone	2785							**−0.117**	**0.050**	**0.020**	**−0.188**	**0.057**	**0.001**

Adjusted for baseline score, follow-up age, sex, ethnicity, maternal education, first language, parental NS-SEC, school type (fixed effects), neighbourhood, school (random effects). exposure Effects are per IQR increase in exposure. Average annual exposure baseline to follow-up IQR $PM_{2.5} = 1.60\,\mu g/m^3$, $PM_{10} = 2.12\,\mu g/m^3$, $NO_2 = 3.65\,\mu g/m^3$, $O_3 = 3.11$ ppb.
Bold: $p < 0.05$. *Italics:* $p < 0.10$.
SDQ: Strengths and difficulties questionnaire.
[a] Higher values reflect worse mental health or cognition
[b] Higher values reflect better mental health or cognition

Table 4
Adjusted associations between traffic noise (in quartiles, reference first quartile) and psychological outcomes at follow-up, in single exposure models.

				Lden			Lday			Leve			Lnight		
Follow-up outcomes	***Model***	**N**	**Quartile**	**β**	**SE**	***p***	**β**	**SE**	***p***	**β**	**SE**	***p***	**β**	**SE**	***p***
GAD-7 anxiety symptoms[a,b]	**Single exposure**	2622	**2nd**	0.071	0.121	0.560	0.161	0.132	0.221	0.146	0.121	0.227	0.093	0.121	0.442
			3rd	0.061	0.120	0.614	**0.329**	**0.128**	**0.010**	0.039	0.121	0.745	0.073	0.120	0.543
			4th	*0.203*	*0.120*	*0.090*	0.074	0.142	0.602	**0.240**	**0.120**	**0.046**	*0.214*	*0.120*	*0.074*
Speech-in-noise perception threshold[a,c]	**Single exposure**	3162	**2nd**	0.261	0.232	0.261	*0.475*	*0.274*	*0.083*	0.017	0.030	0.567	0.014	0.030	0.654
			3rd	**0.487**	**0.231**	**0.035**	0.434	0.289	0.134	0.040	0.031	0.190	0.010	0.030	0.740
			4th	0.246	0.233	0.293	0.138	0.311	0.658	−0.027	0.029	0.363	−0.035	0.029	0.234

Ordinal logistic (GAD-7) and linear (speech-in-noise perception) regressions were used. Categorical GAD-7 cut-offs: no or minimal anxiety, 0 to 4; mild anxiety, 5 to 9; moderate anxiety, 10 to 14; and moderately severe and severe anxiety, 15 + . Effects are for each quartile of exposure (ref: 1st quartile).
Bold: $p < 0.05$. *Italics:* $p < 0.10$.
GAD-7: General Anxiety Disorder-7 (questionnaire).
[a] Higher values reflect worse mental health or cognition
[b] adjusted for follow-up age, sex, ethnicity, maternal education, first language, parental NS-SEC, school type (fixed effects), neighbourhood, school (random effects).
[c] adjusted for baseline score, follow-up age, sex, ethnicity, maternal education, first language, parental NS-SEC, school type (fixed effects), neighbourhood, school (random effects).

(SI11), no significant adjusted associations were observed for follow-up outcomes. Models did not include indoor sources of ozone so both sets of results for indoor ozone are the same. The results excluding uncertain geocode data (SI12 and SI13, air pollution N=3037, noise N=3276) were consistent with results from the wider cohort, although some effects did not retain significance in this smaller sample.

Table 5
Adjusted associations between exposures to outdoor air pollution (per interquartile range (IQR) increase) and traffic noise (in quartiles, reference first quartile) and psychological outcomes at follow-up, in multi-exposure models.

Follow-up outcomes	*Model*	Quartile	Lden			Lday			$PM_{2.5}$			PM_{10}			NO_2			Ozone		
			β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*
G AD-7 anxiety symptoms [a,c]	**Single exposure**	**2**	0.071	0.121	0.560	0.161	0.132	0.221	0.096	0.148	0.521	0.135	0.133	0.316	0.079	0.131	0.544	−0.140	0.150	0.352
	effect	**3**	0.061	0.120	0.614	**0.329**	**0.128**	**0.010**												
Air pollution single exposure and multi-exposure N: 2433		**4**	*0.203*	*0.120*	*0.090*	0.074	0.142	0.602												
	$PM_{2.5}$ + Lden	**2**	0.193	0.125	0.123				0.038	0.071	0.587									
		3	0.085	0.127	0.500															
Noise single exposure N: 2622		**4**	*0.251*	*0.131*	*0.055*															
	$PM_{2.5}$ + Lday	**2**				0.173	0.136	0.206	0.069	0.069	0.317									
		3				**0.276**	**0.136**	**0.042**												
		4				0.046	0.155	0.767												
	PM_{10} + Lden	**2**	0.194	0.125	0.123							0.044	0.065	0.503						
		3	0.084	0.127	0.508															
		4	*0.241*	*0.133*	*0.070*															
	PM_{10} + Lday	**2**				0.170	0.136	0.213				0.074	0.063	0.238						
		3				*0.264*	*0.137*	*0.053*												
		4				0.029	0.157	0.856												
	NO_2 + Lden	**2**	0.194	0.125	0.122										−0.017	0.065	0.796			
		3	0.096	0.127	0.447															
		4	**0.282**	**0.134**	**0.035**															
	NO_2 + Lday	**2**				0.176	0.136	0.198							0.021	0.063	0.744			
		3				**0.284**	**0.138**	**0.039**												
		4				0.067	0.159	0.675												
	Ozone + Lden	**2**	0.194	0.125	1.540													−0.023	0.072	0.754
		3	0.089	0.126	0.481															
		4	*0.258*	*0.132*	*0.051*															
	Ozone + Lday	**2**				0.173	0.136	0.205										−0.050	0.071	0.476
		3				**0.273**	**0.137**	**0.047**												
		4				0.052	0.157	0.739												
Executive functioning [b,d]	**Single exposure**	**2**	0.029	0.026	0.262	0.004	0.030	0.903	**0.036**	**0.018**	**0.045**	*0.030*	*0.016*	*0.057*	**0.038**	**0.014**	**0.007**	**−0.058**	**0.016**	**<0.001**
	effect	**3**	−0.005	0.026	0.849	−0.015	0.032	0.635												
Air pollution single exposure and multi-exposure N: 2785		**4**	−0.013	0.026	0.621	0.004	0.035	0.912												
	$PM_{2.5}$ + Lden	**2**	0.007	0.027	0.796				**0.045**	**0.018**	**0.016**									
		3	−0.009	0.027	0.738															
Noise single exposure N: 3011		**4**	−0.039	0.028	0.167															
	$PM_{2.5}$ + Lday	**2**				−0.006	0.031	0.842	**0.037**	**0.018**	**0.049**									
		3				−0.025	0.033	0.439												
		4				−0.007	0.037	0.858												
	PM_{10} + Lden	**2**	0.007	0.027	0.794							**0.041**	**0.017**	**0.014**						
		3	−0.009	0.027	0.735															
		4	−0.043	0.028	0.128															
	PM_{10} + Lday	**2**				−0.007	0.031	0.825				*0.031*	*0.017*	*0.060*						
		3				−0.027	0.033	0.409												
		4				−0.010	0.038	0.787												
	NO_2 + Lden	**2**	0.006	0.027	0.819										**0.053**	**0.015**	**0.001**			
		3	−0.012	0.027	0.669															
		4	*−0.056*	*0.029*	*0.051*															
	NO_2 + Lday	**2**				−0.009	0.030	0.772							**0.043**	**0.015**	**0.006**			
		3				−0.035	0.033	0.282												
		4				−0.025	0.038	0.505												
	Ozone + Lden	**2**	0.006	0.027	0.832													**−0.072**	**0.017**	**<0.001**
		3	−0.014	0.027	0.593															
		4	**−0.057**	**0.028**	**0.044**															

(*continued on next page*)

Table 5 (*continued*)

Follow-up outcomes	*Model*	Quartile	Lden			Lday			$PM_{2.5}$			PM_{10}			NO_2			Ozone		
			β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*
	Ozone + Lday	**2**				−0.012	0.03	0.702										**−0.065**	**0.017**	**<0.001**
		3				−0.042	0.032	0.195												
		4				−0.032	0.036	0.387												
	Single exposure	**2**	−0.132	0.154	0.392	−0.248	0.171	0.146	0.077	0.098	0.429	0.092	0.087	0.292	0.093	0.081	0.254	−0.092	0.092	0.322
Fluid intelligence [b,d]	**effect**	**3**	−0.193	0.155	0.212	−0.131	0.180	0.468												
		4	−0.157	0.155	0.312	−0.142	0.197	0.470												
Air pollution single exposure and multi-exposure N: 2953	**$PM_{2.5}$ + Lden**	**2**	−0.205	0.160	0.201															
		3	*−0.314*	*0.162*	*0.053*															
		4	−0.274	0.168	0.102															
Noise single exposure N: 3162	**$PM_{2.5}$ + Lday**	**2**				0.048	0.198	0.808	−0.055	0.088	0.534									
		3				−0.335	0.215	0.119												
		4				0.261	0.235	0.266												
	PM_{10} + Lden	**2**	−0.205	0.160	0.200							0.137	0.094	0.144						
		3	**−0.320**	**0.162**	**0.049**															
		4	*−0.305*	*0.171*	*0.075*															
	PM_{10} + Lday	**2**				−0.217	0.178	0.224				0.106	0.093	0.252						
		3				−0.130	0.191	0.497												
		4				−0.175	0.214	0.414												
	NO_2 + Lden	**2**	−0.207	0.16	0.196										0.142	0.089	0.110			
		3	**−0.322**	**0.162**	**0.047**															
		4	*−0.323*	*0.173*	*0.062*															
	NO_2 + Lday	**2**				−0.218	0.178	0.221							0.110	0.088	0.209			
		3				−0.142	0.191	0.460												
		4				−0.195	0.216	0.367												
	Ozone + Lden	**2**	−0.206	0.160	0.198													−0.136	0.098	0.169
		3	**−0.319**	**0.162**	**0.049**															
		4	*−0.296*	*0.170*	*0.082*															
	Ozone + Lday	**2**				−0.216	0.178	0.225										−0.106	0.098	0.281
		3				−0.134	0.192	0.484												
		4				−0.171	0.213	0.424												
	Single exposure	**2**	0.261	0.232	0.261	*0.475*	*0.274*	*0.083*	−0.114	0.147	0.439	−0.081	0.134	0.545	−0.080	0.124	0.522	0.140	0.140	0.322
Speech-in-noise perception threshold [a, d]	**effect**	**3**	**0.487**	**0.231**	**0.035**	0.434	0.289	0.134												
		4	0.246	0.233	0.293	0.138	0.311	0.658												
Air pollution single exposure and multi-exposure N: 1338	**$PM_{2.5}$ + Lden**	**2**	0.277	0.243	0.253				−0.145	0.155	0.351									
		3	**0.489**	**0.245**	**0.046**															
		4	0.207	0.256	0.420															
Noise single exposure N: 1443	**$PM_{2.5}$ + Lday**	**2**				*0.521*	*0.287*	*0.070*	−0.116	0.151	0.445									
		3				0.339	0.307	0.270												
		4				0.234	0.334	0.485												
	PM_{10} + Lden	**2**	0.275	0.243	0.257							−0.107	0.144	0.460						
		3	**0.483**	**0.245**	**0.049**															
		4	0.203	0.261	0.436															
	PM_{10} + Lday	**2**				*0.520*	*0.287*	*0.071*				−0.083	0.139	0.552						
		3				0.337	0.309	0.275												
		4				0.226	0.337	0.504												
	NO_2 + Lden	**2**	0.279	0.243	0.251										−0.103	0.135	0.448			
		3	**0.483**	**0.244**	**0.049**															
		4	0.214	0.265	0.420															
	NO_2 + Lday	**2**				*0.518*	*0.287*	*0.071*							−0.080	0.131	0.545			
		3				0.344	0.310	0.267												
		4				0.233	0.340	0.495												
	Ozone + Lden	**2**	0.283	0.243	0.243													0.175	0.150	0.247
		3	**0.498**	**0.245**	**0.042**															
		4	0.235	0.259	0.364															

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Table 5 (*continued*)

Follow-up outcomes	*Model*	Quartile	Lden			Lday			$PM_{2.5}$			PM_{10}			NO_2			Ozone		
			β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*	β	SE	*p*
	Ozone + Lday	**2**				*0.520*	*0.287*	*0.071*										0.144	0.147	0.329
		3				0.364	0.311	0.242												
		4				0.267	0.339	0.432												

Ordinal logistic (GAD-7) and linear (all other outcomes) regressions were used. Categorical PHQ-9 cut-offs: no or minimal depression, 0 to 4; mild depression, 5 to 10; moderate depression, 11 to 14; moderately severe and severe depression, 15 + . Categorical GAD-7 cut-offs: no or minimal anxiety, 0 to 4; mild anxiety, 5 to 9; moderate anxiety, 10 to 14; and moderately severe and severe anxiety, 15 + .
Air pollution effects are per IQR increase exposure: Average annual exposure baseline to follow-up IQR $PM_{2.5} = 1.60\,\mu g/m^3$, $PM_{10} = 2.12\,\mu g/m^3$, $NO_2 = 3.65\,\mu g/m^3$, $O_3 = 3.11$ ppb. Noise effects are for each quartile of exposure (ref: 1st quartile).
Bold: $p < 0.05$. *Italics:* $p < 0.10$
GAD-7: General Anxiety Disorder-7 (questionnaire).
[a] Higher values reflect worse mental health or cognition
[b] Higher values reflect better mental health or cognition
[c] adjusted for follow-up age, sex, ethnicity, maternal education, first language, parental NS-SEC, school type (fixed effects), neighbourhood, school (random effects)
[d] adjusted for baseline score, follow-up age, sex, ethnicity, maternal education, first language, parental NS-SEC, school type (fixed effects), neighbourhood, school (random effects)

4. Discussion

This large, prospective, longitudinal study of air pollution and traffic noise in relation to adolescent mental health and cognition is the first of its kind, incorporating school and home-based, indoor, outdoor, and combined (hybrid) metrics. We observed novel and robust associations between ozone exposure and slower executive functioning development, which was also associated with a range of other exposures following adjustment for ozone. Some other associations were observed between noise exposure and cognitive outcomes (speech-in-noise perception and fluid intelligence) and between air pollution, noise, and mental health outcomes (emotional and behavioural problems, externalising problems, and anxiety).

Executive functioning impacts performance and management of day to-day tasks, and is also detrimentally associated with stress responses, physical fitness, health behaviours, and academic achievement (Williams et al., 2009, Diamantopoulou et al., 2007). Experimental studies of ozone toxicology and animal models suggest that ozone induces chronic oxidative stress and neuroinflammation, disrupts microglial functioning, impairs cortical and hippocampal antioxidant activity, and reduces synaptic plasticity (Bello-Medina et al., 2022, Martínez-Lazcano et al., 2013). A recent study in young adults reported that ozone was associated with worsened cognitive performance on a video game involving executive function skills (Wyatt et al., 2023). However, executive functioning and ozone were not associated in a study of 6–12-year-olds (Gui et al., 2020). Associations between executive functioning and ozone have not been found when investigated in older adults (Thompson et al., 2022). To our knowledge, this study is the first to investigate the relationship between ozone exposure and executive functioning in adolescence (Thompson et al., 2022), finding that typical developmental improvements were slower with increasing exposure. Therefore, although biologically plausible, there was not a precedent in the literature to expect this relationship, and more research should attempt to replicate these results in this age group. Indoor ozone was not associated with executive functioning development, which could be to do with lower levels indoor, higher risk outdoors (due to higher levels of physical and respiratory activity), or challenges in modelling indoor ozone due to its reactions with other species (Environmental Protection Agency, 2023).

In multi-exposure models with ozone, associations between executive functioning and all other exposures became negative (and ozone's association became stronger), even though many were positively associated with executive functioning in single-exposure models. This suggests the negative correlation between ozone and other exposures may have confounded single-exposure associations. Systematic reviews have highlighted detrimental associations between $PM_{2.5}$, PM_{10} and NOx and executive function in children, but have not suggested associations between transportation noise and executive functioning in children (Thompson et al., 2023, Thompson et al., 2022). However, few studies investigating environmental noise and cognition have focussed on adolescents or adjusted for air pollution, and we are unaware of any such studies adjusting for ozone. Although the strongest prior evidence in children has been for impacts on executive functioning (Thompson et al., 2023), we hypothesised that air pollutants would be associated with a wider range of cognitive outcomes than we observed. However, the prior literature has focussed more on children and older adults than adolescents (Thompson et al., 2023). Noise exposure was also associated with slower development of fluid intelligence and speech-in-noise perception, and these outcomes have not been explored in depth in prior literature (Thompson et al., 2022). Impacts on language perception may help explain prior evidence that transportation noise can impact reading development (Thompson et al., 2022).

In a previous *meta*-analysis, $PM_{2.5}$ was associated with anxiety and depression in adults[10]. In another UK Based adolescent study (E-risk), eight-year NOx exposure was associated with age 18 externalising and internalising symptoms (Reuben et al., 2021). Therefore, we expected to

see stronger associations for these exposures with a wider range of mental health outcomes. There has been little research in adolescents looking at the mental health impacts of PM_{10} or ozone, and the authors are not aware of studies investigating them in relation to anxiety or the SDQ, so associations between these exposures and outcomes were novel and warrant further investigation. With respect to noise, systematic reviews and *meta*-analyses of children and adolescents (under 18) have not supported associations with anxiety outcomes, which we observed (Schubert et al., 2019). However, this literature has largely focussed on children under 12, and/or measures including but not specific to anxiety. Prior work does support associations between road traffic noise and emotional and behavioural difficulties, which we observed at baseline (Schubert et al., 2019). Further, a *meta*-analysis of adults found road traffic noise (Lden) to be associated with increased anxiety (Dzhambov and Lercher, 2019). Therefore, it is possible that impacts of noise have presented as general emotional and behavioural problems at a younger age, and as increased anxiety symptoms during adolescence and adulthood.

A major strength of this study is novelty; this is the first study combining school and home-based exposure to air pollution and noise in relation to both mental health and cognition in adolescents. SCAMP is longitudinal, diverse, representative, relatively large, and is geographically dispersed within and around London, covering a range of urban, suburban, and rural areas. Cohorts concentrated in highly trafficked areas where ozone is low may not be best placed to detect the effects of high ozone concentrations; this may have contributed to the emergence of ozone as highly impactful and the lesser impact of other exposures, as compared to prior literature. However, some shortcomings should be noted. This study focussed on one city (London) in one country (UK) so the results may not be generalisable to other places with different contextual factors and exposure levels. London, like many cities, contains a diverse range of populations exposed to differing levels of air and noise pollution. In the interests of parsimony and avoiding over-adjustment, a select number of socioeconomic indicators were controlled for in this study. However, it is possible other socioeconomic or demographic factors could have contributed to the observed effects. Although personally measured exposure data from a sub-study was used in modelling, exposure estimates still largely relied on modelled rather than measured exposure, which may create higher uncertainty. We only explored associations over an 18–42-month period in 11–15-year-olds, so results are not generalisable to the whole span of childhood and adolescence. We also could not account for childhood exposure to air and noise pollution or pre-baseline cognitive and mental health difficulties, although baseline cognition/mental health was controlled for where possible. Interpretations should therefore be handled with care.

Attempts should be made to replicate these findings in other populations. As results differed somewhat between the micro-environments and noise metrics, future research should continue to model exposure to air pollution and noise in a variety of settings and times of day to disentangle if these differential impacts are due to real-world or methodological differences. More studies should include multi-exposure models, especially including ozone, which was negatively correlated with the other exposures and had the most impact on other associations when adjusted for. Adjusting for air pollution also drew out more associations with noise than single-exposure models, so studies of environmental noise exposure and psychological outcomes should adjust for air pollution as standard. Causal inference would be enhanced by studies that evaluate plausible mediators (e.g., impacts on sleep, cortisol, oxidative stress, inflammation, brain structure, and brain activity) and the role of other environmental factors like greenspace and neighbourhood quality. Future research should include careful consideration of which covariates are appropriate for a given context and explore effect modification by socioeconomic and demographic factors. As our results varied between timepoints and differed from prior research in children and adults, future research should attempt to further disentangle the psychological impacts of acute, short, and long-term exposure at different developmental stages.

Our findings suggest policies to reduce human exposure to this range of air pollutants and noise could benefit adolescent psychological health and development. However, managing human exposure to co-occurring environmental air pollutants is complex because of the interactions between these chemicals and their precursors. In our study, ozone was negatively correlated to all other exposures. The reduction of NO_2 and PM sources in urban settings, brought about by mitigations designed to reduce climate change and health impacts (such as the Ultra Low Emission Zone), will likely lead to increased ozone concentrations. In many urban environments, ozone is negatively correlated with NO_2 due to NOx titration (Yu et al., 2019). Decreases in particulate matter can also increase ozone levels, because of shared precursor chemicals (Yu et al., 2019). To avoid increasing ozone when attempting to decrease NO_2 and PM, researchers have highlighted the importance of targeted reductions in VOC (volatile organic compounds) emissions (e.g. solvent usage, burning wood and rubbish) (Yu et al., 2019). Another important consideration for environmental policy is that biogenic VOCs are a major precursor to ground level ozone, so urban greening efforts must consider species variation in biogenic VOC emissions to mitigate inadvertent ozone generation (Calfapietra et al., 2013). As heatwaves increase with climate change, co-exposure to heat stress and ozone may also increase neurocognitive and health risks (Yan et al., 2023). Climate change is likely to lead to increased ground-level ozone, especially in urban environments and polluted areas (Murazaki and Hess, 2006).

5. Conclusions

This study has found strong evidence of detrimental associations between ground-level ozone exposure and executive functioning in adolescence, and supportive evidence for relationships between a range of other exposures and psychological outcomes. However, stronger associations with a broader range of outcomes were expected for $PM_{2.5}$ and NO_2, based on prior research. Ozone has largely been overlooked in the literature, which may be due to a focus on other air pollutants or confounding with negatively correlated co-exposures. This work suggests reducing young people's exposure to air pollution and noise may improve psychological outcomes. However, acting upon air pollution requires careful consideration of the atmospheric chemistry of different air pollutants, because efforts to reduce NO_2 and particulate matter have the potential to increase ozone levels. Although the full psychological effects of air pollution and noise are yet to be understood, reducing climate change and enhancing environmental quality will have benefits for young people's psychological health by numerous other pathways in addition to the untapped benefits this study suggests (Lawrance et al., 2022). Hence, this study adds to the public health case for considered approaches to reducing human exposure to air pollution and traffic noise.

Data sharing

RT and RBS have accessed and verified the data. Data dictionary and analytical scripts are available on request to corresponding author. The Cognitive Development in the Urban Environment (CLUE) study protocol can be found at: https://scampstudy.org/wp-content/uploads/2024/04/CLUE-II-Proposal-1.pdf.

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Ethics Committee approval

The North-West Haydock Research Ethics Committee approved the SCAMP study and its subsequent amendments (#14/NW/0347).

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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.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2024.108963.

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