Investigating the impact of environmental pollution on adolescent mental health and cognition

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Declaration of originality

I, the candidate, declare that the submitted thesis is my own work and that, to my best knowledge, it contains no sources or contributions other than those mentioned and acknowledged.

Statement of contributions and adapted work

The literature in the systematic reviews found in chapters 2 and 3 were reviewed in duplicate by one of the following researchers, in addition to the candidate (who reviewed all literature): Dr Rachel B. Smith, Yasmin Bou Karim, Dr Chen Shen, Kayleigh Drummond, Chloe Teng. With the candidate, the following co-researchers coded the qualitative data and collaboratively produced the thematic structure found in chapter 4: Tania Hussain and Zaina Kabba. The training and methodology for co-producing this analysis was provided by Dr Lindsay H. Dewa. Dr Helen L. Fisher peer-debriefed the qualitative analyses. Modelling to produce the noise exposure data used in chapters 6 and 7 was conducted by Dr Calvin Jephcote. Modelling to produce the air pollution exposure data used in chapters 6 and 7 was conducted by Dr Shanon Lim, Dr Gregor Stewart, and Dr Tuan Vu.

Some of the work in chapters 3 and 4 has been adapted from prior work published by the candidate as first author, in the following articles:

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Abstract

There is increasing evidence that environmental pollution can impact people physiologically and psychologically. Therefore, this PhD aimed to investigate the impact of environmental air and noise pollution on adolescent mental health and cognition. Firstly, two systematic literature reviews were conducted evaluating the evidence for air pollution and noise effects, respectively, showing generally detrimental trends but with low certainty evidence. This demonstrated the need for further, good quality research. Next, interviews were conducted with adolescents aged 14-18 (N=15) about their experiences of environmental problems. Co-produced qualitative thematic analysis showed that environmental quality, including air quality and noise levels, contributes to young people's sense of wellbeing, especially feelings of stress and overwhelm. High concern was shown for environmental issues in general, particularly climate change.

Finally, an epidemiological analysis of the London based adolescent cohort SCAMP (Study of Cognition, Adolescents and Mobile Phones) was conducted. Multi-level multivariate linear regression, generalised additive modelling, moderation and mediation analyses were conducted using longitudinal data, predicting measured mental health and cognition at age 11-13, 13-15, and 15-18, from modelled annual exposure to air pollution and noise (N=7555 total, with sample sizes varying by specific exposure-outcome analysis). Analyses revealed detrimental effects of exposure to ozone and cognitive development, and particulate matter < 10 μm in diameter (PM_{10}) and mental health. However, some 'beneficial' effects were observed between pollutants and executive function, driven by indoor exposure. In multi-pollutant models, daytime noise was associated with some detrimental effects, and particulate matter with ameliorated processing speed development. Ozone and PM_{10} were associated

with greater odds of psychosis symptoms in male and low IQ students respectively, and mixed-race students presented some unique detrimental effects of air pollution and cognition.

This project contributes a comprehensive study of air and noise pollution in relation to adolescent psychological outcomes. The implications of its complex findings are discussed in relation to prior literature and possible mechanisms or sources of confounding, as well as the specific directions for future research. Young people's passion for local and global environmental problems demonstrates the necessity of policies that not only reduce personal exposure to air pollution and noise but address emissions and environmental damage in general. The risks presented to young people by environmental degradation are multi-factorial, and top-down approaches present opportunities to protect their health and wellbeing at multiple levels, scales, and timescales.

Table of Contents

Investigating the impact of environmental pollution on adolescent mental health and cognition ***1***

List of tables **9**

List of figures **10**

List of major acronyms **12**

Glossary of key terms **13**

Chapter 1: Introduction ***14***

1.1 Background **14**

1.2 Mechanisms **17**

1.2.1 Air pollution mechanisms 18

1.2.2 Noise mechanisms 23

1.2.3 Young people's perspectives 24

1.3 Covariates, moderators, and mediators **27**

1.4 Gaps in the literature, aims, and hypotheses **32**

Chapter 2: A systematic review of the literature evaluating associations between air pollution exposure and psychological outcomes in adolescence ***34***

2.1 Background **34**

2.2 Methods **34**

2.2.1 Search strategy and eligibility criteria 35

2.2.2 Review process 36

2.2.3 Risk of bias and Quality of Evidence Assessment 37

2.2.4 Meta-analysis 38

2.3 Results **39**

2.4 Discussion **77**

2.4.1 Summary 77

2.4.2 Evaluation 78

2.4.3 Future research 80

2.4.4 Strengths and limitations 83

2.4.5 Concluding comments and implications for wider project 84

Chapter 3: Systematic review of the literature studying associations between noise pollution and adolescent mental health and cognition ***86***

3.1 Background **86**

3.2 Methods **87**

3.2.1 Update Strategy 87

3.2.2 Search strategy and eligibility criteria 88

3.2.3 Review process 89

3.2.4 Risk of bias and Quality of Evidence Assessment 89

3.2.5 Meta-analysis 89

3.3 Results **90**

3.4 Discussion **113**

3.4.1 Summary 113

3.4.2 Future research 115
3.4.3 Strengths and limitations 116
3.4.4 Concluding comments and implications for wider project 117

Chapter 4: Qualitative interview study on adolescents' environmental perspectives 119

4.1 Introduction 119

4.2 Methods 120

4.3 Results 123

4.4 Discussion 136
4.4.1 Summary 136
4.4.2 Implications for research and practice 139
4.4.3 Strengths and limitations 141
4.4.4 Implications for wider project 143

Chapter 5: Methodology for epidemiological analyses 145

5.1 Introduction 145
5.1.1 Background 145
5.1.2 Contextualisation with rest of thesis and prior research 146
5.1.3 Aims and Hypotheses 148

5.2 Methods 149
5.2.1 Data 149
5.2.2 Statistical analysis 163

Chapter 6: Epidemiological associations between exposure to air pollution and noise and psychological outcomes in adolescents 171

6.1 Results 171
6.1.1 Participant characteristics 171
6.1.2 Main model results 182
6.1.3 Backward step multiple regression of processing speed and CAPE outcomes 190
6.1.4 Sensitivity analyses 191
6.1.5 Non-linear relationships 193

6.2 Discussion 199
6.2.1 Summary 199
6.2.2 Interpretation 199
6.2.3 Strengths and limitations 204
6.2.4 Summary 206

Chapter 7: Moderation and mediation of epidemiological associations 207

7.1 Results 208
7.1.1 Moderation analyses 208
7.1.2 Mediation analyses 215

7.2 Discussion 216
7.2.1 Summary 216
7.2.2 Interpretation 217
7.2.3 Strengths and limitations 220
7.2.4 Concluding comments 221

Chapter 8: Discussion 222

8.1 Summary and Interpretation 222

8.2 Strengths & Limitations 226

8.3 Implications for Future Research 230
8.4 Policy and public health implications 232
8.5 Concluding Comments 237
References 239
Appendices 261

List of tables

Table 1a: Characteristics of eligible air pollution and cognition studies included in systematic review

Table 1b: Characteristics of eligible air pollution and mental health studies included in systematic review

Table 2a: GRADE assessed certainty of collective evidence for air pollution and cognition associations investigated in the literature

Table 2b: GRADE assessed certainty of collective evidence for air pollution and mental health associations investigated in the literature

Table 3a: Characteristics of eligible noise pollution and cognition studies included in systematic review

Table 3b: Characteristics of eligible noise pollution and mental health studies included in systematic review

Table 4a: GRADE assessed certainty of collective evidence for noise pollution and cognition associations investigated in the literature

Table 4b: GRADE assessed certainty of collective evidence for noise pollution and mental health associations investigated in the literature

Table 5. Participant background and demographic information

Table 6. Summary of air pollution and noise exposure models

Table 7. Cohort sociodemographics after imputation (N=7744)

Table 8. Comparing Baseline sociodemographic characteristics of the target population SCAMP cohort, and imputed analytical sample, adapted from Toledano et al., (2019)

Table 9. Descriptive statistics of mental health and cognitive variables measured at baseline (2014-2016), follow-up 1 (2016-2018), and follow-up 2 (2020-2021)

Table 10. Descriptive statistics for exposures in main analytical sample (living within M25, N=3530)

Table 11a. Comparison between those living within the M25 (air pollution models) and the wider sample (noise only models)

Table 11b. Comparison of analytical sample (providing at least one address) in total, baseline, follow-up, lost to follow up, and those new to follow-up I

Table 12. Primary model results reflecting associations between exposure to $PM_{2.5}$ or PM_{10} and continuous psychological outcomes at baseline and follow-up

Table 13. Primary model results reflecting associations between exposure to NO_2 or O_3 and continuous psychological outcomes at baseline and follow-up.

Table 14. Logistic regression at follow up-2 (2020-2021) of odds of psychotic-like experiences at age 15-18 at different levels of average exposure to pollutants and noise from 2013-2018

Table 15. Primary models reflecting associations between exposure to noise and psychological outcomes in adolescence

Table 16. Multi-pollutant models where the direction or significance of the single-exposure effect was altered

Table 17. Results of backward step multiple regression predicting processing speed and risk for psychosis status

Table 18. Correlations between air pollution metrics

Table 19. Comparison of GLMs with GAMs, when GAM improved model fit

Table 20. Subgroup analyses of exposure to air pollution or noise and psychological outcomes where a subgroup effect reached significance

Table 21. Mediation analyses of associations between air pollution and adolescent psychological outcomes by sleep quality, atopic disease, mental health, and cognition

Table 22. Overall hypotheses tested by thesis and the corresponding findings

List of figures

Figure 1. DAG of causal pathways involved in exposure-response relationships

Figure 2. PRISMA diagram of search results (air pollution review)

Figure 3. Pooled beta coefficient of the relationship between 1μg/m3 increase prenatal NO_2 exposure and early psychomotor development.

Figure 4. Pooled beta coefficient of the relationship between 1μg/m3 increase prenatal $PM_{2.5}$ exposure and early psychomotor development. Note, TE= beta coefficient, seTE= standard error of beta coefficient.

Figure 5. PRISMA diagram of search results (noise review)

Figure 6. The pooled mean difference in reading comprehension scores between children in high noise (average Leq 59-69.9dBA, Max Leq 89-96.5) and low noise exposed (average Leq 54.4-57 dBA, Max Leq 73.2-74.3) classrooms.

Figure 7. The pooled beta coefficients reflecting the association between environmental noise (impact of 1dB increase, from mixed transport sources) and reading and language abilities.

Figure 8. Co-produced thematic map depicting adolescents' responses to the environment

Figure 9. Illustrative model structure of cross-classified multilevel mixed-effects linear models

Figures 10a and 10b. Histograms of participant ages at baseline and follow-up

Figure 11. Participant locations and attrition from baseline (BL) and follow-up (F1)

Figure 12. Participants, schools, and area-level socioeconomic status

Figure 13. Participants, schools, and area-level ethnicity

Figure 14. Participants, schools, and area-level estimated mental disorder prevalence (%)

Figure 15. Participants, schools, and area-level access to mental health treatment (% waiting >6 weeks for first treatment from NHS talking therapies services)

Figure 16. Predicted relationship between NO_2 in the year prior to baseline (BL) assessment and baseline SDQ-assessed internalising problems (a) in an adjusted linear model, (b) in an adjusted GAM

Figure 17. Predicted relationship between NO_2 in the year prior to baseline (BL) assessment and baseline executive function (a) in an adjusted linear model, (b) in an adjusted GAM

Figure 18. Predicted relationship between Lden in the year prior to baseline (BL) assessment and baseline SDQ-assessed externalising problems (a) in an adjusted linear model, (b) in an adjusted GAM

Figure 19. Predicted relationship between Lday in the year prior to baseline (BL) assessment and baseline SPIN threshold (a) in an adjusted linear model, (b) in an adjusted GAM

Figure 20. Causal mediation analysis of the association between PM10 exposure and follow-up anxiety, explained by baseline externalising problems, arrows' annotations showing β(p)

List of major acronyms

$PM_{2.5}$	Particulate Matter < 2.5 μg in diameter
PM_{10}	Particulate Matter < 10 μg in diameter
PM	Particulate Matter
UFP	Ultrafine particles, Particulate Matter < 0.1 μg in diameter
PM_{coarse}	Particulate Matter >2.5 μg & ≤ 10μg in diameter
NO_2	Nitrogen Dioxide
NOx	Nitrogen Oxides
NO	Nitric oxide
O_3	Ozone
VOC	Volatile Organic Compound
PAH	Polycyclic Aromatic Hydrocarbon
SO_2	Sulphur Dioxide
CO	Carbon monoxide
CO_2	Carbon dioxide
BC	Black carbon
EC	Elemental carbon
ECAT	Elemental carbon attributable to traffic
μgm^3	Micrograms per cubic metre
ppb	Parts per billion
AP	Air pollution
TRAP	Air pollution attributable to traffic
dB	Decibels
dBA	A-weighted decibels
Leq	Equivalent continuous sound level
Laeq	A-weighted equivalent continuous sound level
Lden	A-weighted 24 day-evening-night nosie level, with penalty for evening and night noise
Lday	A-weighted day noise level (07:00 - 19:00)
Leve	A-weighted evening noise level (19:00 - 23:00)
Lnight	A-weighted night noise levels (23:00 - 07:00)
LUR	Land use regression
TE	Treatment effect
SE	Standard error
seTE	Standard error of treatment effect
IQR	Interquartile Range
GLM	Generalised linear model
GAM	Generalised additive model
CI	Confidence Interval
OR	Odds Ratio
RR	Risk Ratio
IRR	Increased Risk Ratio
SCAMP	Study of Cognition, Adolescents and Mobile Phones
SES	Socioeconomic Status
EF	Executive Function
IQ	Intelligence Quartile
BL	Baseline
F1	Follow-up I
F2	Follow-up II

Glossary of key terms

Adolescence is defined as young people aged 10-19 years, according to the World Health Organisation (WHO) and United Nations (UN) definition, though it is acknowledged that a universally recognised definition has not been established (Sawyer et al., 2018, WHO, 2005).

Psychological outcomes in this thesis refer to mental health and cognition.

Mental health in this thesis refers to psychological and emotional wellbeing, including the spectrum of positive and non-clinical levels of negative affect (e.g. distress, worry, sadness) found in the general population, as well as symptoms and diagnoses of mental health disorders.

Cognition refers to the faculties involved in acquiring, processing, using, and producing information and understanding, and includes attention, memory, learning, memory, executive function, reasoning, computation, and language (Harvey, 2019, Sternberg, 1999).

Air pollution is broadly defined as contamination of the atmosphere modifying its natural composition**,** with a particular focus on agents presenting risk to physical health or the environment (e.g. particulate matter, ozone, nitrogen oxides, carbon dioxide).

Noise pollution is broadly defined as any excessive (unwanted or harmful) noise level with the potential to disturb human populations.

Chapter 1: Introduction

1.1 Background

The identification of factors contributing to psychological outcomes, particularly mental health and cognition, is an important undertaking in the field of public health. Mental health conditions are associated with a great degree of unmet need, unanswered questions, and social stigmas (Dakić, 2020, Saraceno, 2020, Wainberg et al., 2017). For instance, depression is the leading cause of disability worldwide (Friedrich, 2017, Vos et al., 2017), and mental health has wide ranging consequences for quality of life, physical health, individual achievement, and economic outcomes (Firth et al., 2019, McDaid et al., 2019). This is especially concerning for young people who are at much higher risk of mental health problems compared to other areas of health. For example, suicide is the second leading cause of death in 15-29 year olds (World Health Organization, 2019) and around 50% of adult mental health disorders arise by the age of 14 (Kessler et al., 2005, Kim-Cohen et al., 2003). Therefore, it is imperative to investigate which factors during childhood and adolescence increase (and decrease) the likelihood of developing mental health problems to inform the development of early preventive interventions. Cognitive abilities are interrelated with mental health and also have social, economic, and health consequences (Batty et al., 2007, Bortolato et al., 2015, Bukodi et al., 2014, Carneiro et al., 2007, Firkowska-Mankiewicz, 2011) so understanding the risk and resilience factors for cognitive ability is also of importance to public health. Cognitive abilities also continue to develop into adolescence (Sanders, 2013). Therefore, the factors contributing to cognition and mental health and their interplay are particularly important to understand in adolescence.

The foremost psychological theories of mental health and cognition emphasise the role of the social environment, genetic factors, how the two relate over time, and how thoughts and behaviours of the individual contribute to and perpetuate their psychological development and outcomes (Beauchaine and Hinshaw, 2017, Sroufe, 2009). However, environmental psychology is focussed upon the relationships between people and their environments, including built and natural environments (Gifford, 2014). This field is grounded in the concept that people truly engage with their physical environments, not just acting upon them and being affected by them physically but interacting with them psychologically. This means that the features in people's environments influence their state of mind, their cognition, and particularly their wellbeing (Bonnes et al., 2018). For example, there is a wide literature on nature and greenspace, which is thought to have a soothing and beneficial impact (Barton and Rogerson, 2017, Martin et al., 2020, McMahan and Estes, 2015, Vanaken and Danckaerts, 2018, Wells, 2000). Further, research in this field suggests that our perceptions and attitudes towards our environments influence how we feel and behave towards them. For example, nature-connectedness has been shown to enhance wellbeing, and sense of place (attachment to environments) is associated with pro-environmental and sustainable behaviours (Capaldi et al., 2014, Martin et al., 2020, Pritchard et al., 2020, Stedman, 2002, Vaske and Kobrin, 2001).

Environmental epidemiology is the study of the relationships between people's environments and their health. Particularly, it estimates the impact of environmental exposures such as air pollution, noise, radiation, chemicals, and pathogens on someone's proclivity to disease, illness, good health, injury, or mortality (Baker and Nieuwenhuijsen, 2008, Morgenstern and Thomas, 1993). This field studies a range of risk and resilience factors, often on a population level, grounded in the understanding that health and disease are made up of a wide variety of

factors and rarely consist of a linear bivariate cause-and-effect relationship(Rothman and Greenland, 2005). There are an increasing number of studies that use these methods in relation to psychological and social factors.

Air and noise pollution are two major health concerns in increasingly urbanised populations, with consequences for physical health and mortality (Basner et al., 2014, Basner and McGuire, 2018, Van Kempen et al., 2018, Chen and Kan, 2008). The World Health Organisation (WHO) estimated that 2,975,400 deaths in 2012 globally were attributable to ambient air pollution (2016), and that 1,000,000 healthy life years (based on disability-adjusted life-years, that is years of ill-health, disability, or death) are lost every year in Western Europe from traffic-related noise (2011).

Air pollution arising from transport, industry, and community activity (e.g. cooking and heating) is pervasive in many places containing human populations, especially in urban areas where an increasing proportion of the global population live. Secondary pollutants are also of increasing concern, arising from the interactions of primary pollutants (directly emitted from human activity) and other substances in the atmosphere. Noise pollution is also pervasive, arising from transport, industry, and community activity (e.g. technology, machinery, nightlife). People can be exposed to excess noise at home, school, work, and other settings, and whilst noise exposure can disrupt concurrent activities and performance (Hughes and Jones, 2001, Szalma and Hancock, 2011) there are additional concerns that noise, as an environmental exposure, can carry overall risks for human health and development beyond any immediate interference (e.g. increased heart disease). Researchers are increasingly interested in how these exposures can influence psychological as well as physical health. For example, researchers are exploring whether increased air and noise pollution could increase

psychological disorders and worsen cognition (Clark et al., 2020, Tzivian et al., 2015a). This literature will be explored further in chapters 2 and 3.

Additionally, noise and air pollution often covary; in cities, traffic is the main common source of human exposure to noise and air pollution, and so there could be mutual confounding effects (Foraster, 2013). In London, modelled noise and air pollution correlations are moderate (0.34-0.55) and stable over time (Fecht et al., 2016). Therefore, studies of air pollution that do not evaluate the role of noise cannot eliminate the possibility that noise, being correlated with air pollution and sharing common sources, is the actual causal variable impacting mental health or cognition, and vice versa.

Exposure to pollution and noise may perpetuate health inequalities because less privileged socioeconomic status and ethnicity are associated with higher levels of exposure to noise and air pollution and worse outcomes (Fecht et al., 2015, Huang et al., 2019, Bilenko et al., 2015, Braga et al., 2001, Chen and Kan, 2008, Cohen et al., 2005, Clark et al., 2020). Further, these social inequalities and inequalities in exposures might intersect to produce worse outcomes, as has been observed in other effects found in environmental epidemiology (Cakmak et al., 2011, Clougherty et al., 2014, Glad et al., 2012, Gray et al., 2014). Therefore, the psychological impacts of pollution and noise must be understood to inform best policy and practice in public health that protects wellbeing, quality of life, opportunity in life, and equality in urban and exposed populations.

1.2 Mechanisms

Although our physical and psychological experiences have different tangible qualities to them and there is a conceptual and theoretical dichotomy between the two (and a

corresponding philosophical debate beyond the scope of this thesis), it is understood in the fields of environmental psychology and social ecology that physical factors can have psychological effects. Sometimes this might be moderated by psychological, social, cognitive or behavioural mechanisms (for example, one's opinion of a landscape eliciting disgust and stress). However, physical and mental health are highly intertwined, with each related to the other; for example, chronic pain, asthma, allergies, and obesity are all comorbid with mental health conditions (Goodwin et al., 2012, Mallen et al., 2008, Russell-Mayhew et al., 2012, Ferro et al., 2016, Gatchel, 2004). Further, early life psychological stress such as adverse events and social disadvantage have been linked to neuroimmunologic pathways of inflammation which may make some people especially vulnerable to external stressors like air pollution, thus conferring an interactively higher level of disease risk (Alvarez et al., 2018).

It is generally accepted in medicine, psychology and psychiatry that mental health and cognition have a basis in the brain, and therefore that the factors influencing the brain, as part of the body, can influence psychological outcomes (Brendel, 2000, Emery et al., 2016, Kristiansson et al., 2015). For example, dementia has a physical basis in the brain, which corresponds to psychological outcomes such as memory loss, and brain damage due to stroke influences a myriad of psychological outcomes depending on where the lesion is localised (Bessey and Walaszek, 2019, Merriman et al., 2019). Therefore, it is plausible that physical exposures like air pollution and noise can influence psychological outcomes.

1.2.1 Air pollution mechanisms

One way in which air pollution could affect mental health is through its effects on the brain, and the young brain is thought to be especially vulnerable to pollutants. Adolescence is a

high-risk period for mental health; around 50% of adult mental health disorders arise by the age of 14 (Kessler et al., 2005) but even healthy adolescence is characterised by emotional unrest and dysfunction (Dahl, 2004, Steinberg, 2005). Until a person's mid-twenties, brain development is characterised by cell proliferation, neuronal migration and the establishment and fine tuning of specific connections (Fuhrmann et al., 2015). With high rates of neuronal proliferation and differentiation as well as a weaker blood brain barrier and immature metabolism, the child and adolescent brain is more vulnerable to damage from environmental pollutants than the adult brain (Grandjean and Landrigan, 2006, Sunyer, 2008). In the developing brain, the myelin sheath is especially sensitive to inflammation and oxidative stress, which are thought to be key processes in pollutant neurotoxicity (French et al., 2009, Thorburne and Juurlink, 1996). Myelination occurs in a very precise way up until adulthood meaning it is likely white matter would be more affected by air pollution during development than after (Dubois et al., 2014). Indeed, particulate matter <2.5 μm in diameter ($PM_{2.5}$) has been shown to impair rat neurobehaviour through this mechanism (Zhang et al., 2018a).

There are multiple mechanisms through which inhaled pollutants can affect the brain. First, by directly interacting with the brain; a range of nanoparticles have been shown to cross the barrier between the lungs and the circulatory system (Miller et al., 2017) and circulating pollutants reaching the brain can induce neuro-inflammatory responses (activating the brain's immune system) by stimulating afferent nerves (Banks and Robinson, 2010, Siso et al., 2010). Direct entry into neuronal cells by small particulate matter (e.g., $PM_{2.5}$) can also occur. A point of vulnerability is the circumventricular organs (the third and fourth ventricles/lateral ventricles of the cerebellum) which are especially vulnerable as they do not have a blood brain barrier (BBB) and are highly vascularised, allowing for direct uptake of circulating pollutants into neuronal cells (Siso et al., 2010). Very small ultra-fine particles can cross most

cell membranes easily so the usual barriers protecting the lungs and brain do not prevent their translocation (Block and Calderon-Garciduenas, 2009, Valavanidis et al., 2008). Additionally, in the nose epithelium, olfactory receptor neurons and the trigeminal nerve sit directly against air, which may allow small particles to reach the brain this way (Shiga et al., 2011).

Secondly, the brain can be affected indirectly by pollutants triggering the release of inflammatory agents. Circulating particles can cause systematic inflammation through exerting pro-inflammatory effects elsewhere (Cunningham et al., 2009) and this systemic inflammation will lead to the production of cytokines circulating in the blood, which can activate the brain's endothelial cells, disrupt the blood brain barrier, stimulate the brain's surface immune cells, activate neuronal afferents at the surface of the brain, or actually enter the brain (Dantzer et al., 2008, Teeling and Perry, 2009). Cytokines also activate the hypothalamic-pituitary-adrenal axis (HPA-axis), which is an important component of the body's reaction to stress (Besedovsky and Del Rey, 2000). Air pollution's stimulation of inflammatory cytokines and the HPA-axis could impact cognitive and mental health issues (Dantzer et al., 1999, Makhija and Karunakaran, 2013, Raymond et al., 2018, Young, 2014). A recent study found that $PM_{2.5}$ was associated with heightened HPA-axis sensitivity in girls with existing anxiety, but not in the general (all female) sample (Miller et al., 2020). Additionally, it has been observed by theorists that socio-economic status and psychosocial stressors have similar effects to air pollution on inflammatory activity (chronic low-level inflammation), which could create a prior increased inflammatory reactivity to environmental stressors like air pollution or they could serve to compound one another, creating especial vulnerability to health problems (Alvarez et al., 2018, Kristiansson et al., 2015). Something

parallel or indeed intertwined could occur with the development of mental health or cognitive issues.

Following air pollution's interaction with the brain and its pro-inflammatory effects, neuronal injury can occur by many mechanisms. Particles that are taken up by neuronal cells or that interact with surface proteins can cause potential cell death and disruption through a wide variety of mechanisms: cause a cell to release inflammatory markers (Peters et al., 2006), damage the mitochondria (Hou et al., 2010), alter reactive oxidative species and antioxidant mechanisms (Hou et al., 2010), cause DNA damage (Bolognin et al., 2009, Yauk et al., 2008), affect enzyme activity and antigen formation (Nel et al., 2006), increase protein fibrillation rate (Colvin and Kulinowski, 2007, Lewis and Lieberman, 2000), induce oxidative stress (Higgins et al., 2010, Orrenius et al., 2011), disrupt endothelium (Hartz et al., 2008), damage the blood brain barrier (Pun et al., 2009), reduce brain repair (Rivas-Arancibia et al., 2010), provoke reactive astrogliasis (Araneda et al., 2008), activate microglia (Cameron and Landreth, 2010, Long-Smith et al., 2009), and exert epigenetic effects (Cao et al., 2007, Yauk et al., 2008). Additionally, irritants to the CNS or systemic inflammation from irritants elsewhere will lead to neuroinflammation - the composition of what is released will vary by trigger but this inflammasome can induce cell death and trigger the release of toxins from glial and endothelial cells (Chakraborty et al., 2010). Systemic inflammation is found in many mental health problems (Bauer and Teixeira, 2019, Wirtz and von Kanel, 2017), as is oxidative stress (Ng et al., 2008, Smaga et al., 2015). As a result of these mechanisms, air pollutants can have many toxic effects on brain cells.

These mechanisms also translate to functional and structural effects beyond the cellular level. In epidemiological studies on humans, white matter volume reduction has been consistently

related to air pollution (particularly Polycyclic Aromatic Hydrocarbons (PAHs) and $PM_{2.5}$) in the frontal, parietal, and temporal areas (Casanova et al., 2016, Chen et al., 2015, Peterson et al., 2015). Alterations in white matter found in school children exposed to air pollution parallel those found in many neurological and psychiatric disorders (Lubczyńska et al., 2018), and effects on the sensory and cognitive systems could also affect mental health indirectly (Shoham et al., 2019). In mice, prenatal exposure to diesel exhaust particles have reduced locomotor activity, dopamine activity in the striatum, and monoamine metabolism (Suzuki et al., 2010, Yokota et al., 2016). The frontal lobe and striatum (specifically their activity, structure, and connectivity) are implicated in anxiety and depression (Alcaro et al., 2010, Carlisi and Robinson, 2018, Phillips et al., 2015). Human exposure to $PM_{2.5}$ has also been associated with smaller total cerebral volume and brain infarcts (Wilker et al., 2015). Prenatal ozone exposure in rats and mice has been shown to permanently damage the cerebellum and its monoaminergic system (Gonzalez-Pina et al., 2008, Rivas-Manzano and Paz, 1999) with more oxidative stress injury found in younger rats than older rats (Rivas-Arancibia et al., 2000). Brainstem auditory evoked potentials show significant delays in highly exposed children (Calderón-Garcidueñas et al., 2011), with associated auditory and vestibular impairments, which can be associated with cognition and mental health problems (Antoine et al., 2017, Bigelow and Agrawal, 2015). Acute exposure of diesel exhaust particles was associated with significantly decreased fast wave activity and median power frequency under electroencephalogram (EEG) recording in 10 participants (Cruts et al., 2008). Additionally, dopaminergic neurons are selectively vulnerable to diesel exhaust particles (Block et al., 2004, Levesque et al., 2011) and are thought to play an important role in psychiatric disorders (Dunlop and Nemeroff, 2007, Kesby et al., 2018), as well as attention, behavioural control, and motor control (Cools, 2011, Longo et al., 2021, Nieoullon, 2002)

1.2.2 Noise mechanisms

Environmental noise exposure is thought to affect human psychology through a variety of mechanisms. While exposure to noise has a direct and immediate psychological effect, which means it can impact mood and cognition when concurrently exposed (e.g. distraction, arousal), chronic noise exposure in one's day-to-day real-life environment is also thought to influence psychological outcomes in the longer term (Clark and Paunovic, 2018b, Clark and Paunovic, 2018a).

The auditory system remains active throughout one's life, consciously and unconsciously, and alerts us to what may be going on in our surroundings. Noise information is transmitted as electro-chemical activity through two distinct pathways in the brain: through subcortical structures such as the amygdala, and through the auditory thalamus to the auditory cortex and back to subcortical areas (the former is mostly involved when asleep) (Basner et al., 2014). Physiological effects of noise are mainly driven by the Sympathetic-Adrenal- Medullary (SAM) axis and the HPA-axis. The SAM axis is activated by stressors to the central nervous system, leading to the release of adrenaline and noradrenaline, preparing the body for fight or flight, increasing blood pressure, mobilising glucose, and aggregating thrombocytes (blood platelets) (Spreng, 2000a, Babisch, 2003). The HPA-axis response leads to the secretion of cortisol and other glucocorticoids, elevating blood glucose and blood pressure and suppressing the immune system (Majzoub, 2006, Spreng, 2000b). Such responses are necessary in certain situations, but chronic arousal or stress can lead to hyperactivity of these systems, and this is associated with a variety of physical and mental health problems (Evans et al., 1998, Martinac et al., 2014, Björntorp, 1996). Therefore, noise influences the mind and

body unconsciously through physiological mechanisms of stress and arousal, inducing physical stress responses involving cortisol and adrenaline (Babisch, 2002).

As one is phenomenologically aware of some of the noise in one's environment (in a way that we are not always directly aware of air quality at lower levels - though high levels of some pollutants can be smelt or seen, e.g. diesel or smog) scientists hypothesise it can also affect psychological outcomes through psychological mechanisms. For example, through distraction from education or work, noise could have detrimental effects on the learning process and therefore impair cognitive development (Chere and Kirkham, 2021, Connolly et al., 2019). Sleep quality is crucial for good physical and mental functioning because it is when the body rests and repairs itself (Wells and Vaughn, 2012). Therefore, noise which keeps a person awake or reduces sleep quality will affect cognitive function and development, reduce mental wellbeing, and increase the risk of emotional problems (Muzet, 2007, Sygna et al., 2014). Noise has also been linked to learned helplessness, which is a person's sense of control and self-efficacy, so a noisy environment could reduce a person's confidence, motivation, and ultimately mental health (Van Kamp and Davies, 2013). Additionally, a noisy environment can be frustrating and annoying, which reduces wellbeing and impacts sleep (Basner et al., 2014, Hahad et al., 2019, Hatfield et al., 2002, Hume et al., 2012, Stansfeld and Clark, 2015).

1.2.3 Young people's perspectives

The mechanisms outlined in the prior two sections are considered direct mechanisms because the individual's level of exposure to pollution or noise induces a response in the exposed person's body or mind which changes psychological outcomes. Theoretical mechanisms linking air pollution and noise to psychological effects tend to focus on a direct biological or

psychological mechanism, but understanding young people's experiences will contextualise and elucidate these relationships. Further, it is possible that young people's perspectives on or experiences of these environmental characteristics could constitute part of the mechanisms through which they are affected psychologically by them.

Subjective or experiential factors might contribute to or help us understand these relationships; human mental states exist on multiple levels where they can be aware of other mental states, be about other mental states, and influence other mental states, similarly to how environmental factors such as greenspace influence other environmental factors like air and noise pollution (Rhodes, 2019, Northoff et al., 2006). Therefore, a person's mind and environments interact on multiple levels where mental states can themselves be mechanistic, rather than exclusively being the outcomes of linear physical relationships. Many academic fields take human experience, processing, and response, to be important factors for understanding human-environment, or psychological-physical, interactions. For example, in environmental psychology, the mind is considered an important mediator between the physical environment and psychological states; people's beliefs, emotions, perceptions, awareness, etc. of these exposures can influence how they respond to them (Gifford, 2014). In social ecology, the relationships between social factors and the environment are appraised (Haberl et al., 2016). For example, young people are collectively becoming increasingly environmentally aware and engaged, meaning that a social body influences and is influenced by the environment (Nissen et al., 2021). This engagement could influence how young people perceive themselves and their spaces, in turn affecting their direct responses to their surroundings. In social epidemiology, research investigates how social non-physical factors, like social support, can influence outcomes of physical health and disease (Galea and Link, 2013). While there is a literature on people's perspectives on urban and rural environments

(Buttazzoni et al., 2021, Hokka et al., 1999, Lekies et al., 2015, Soga et al., 2016, Stokas et al., 2017), the specific literature on young people's thinking about air pollution, noise, and other environmental exposures is fairly limited.

This means that it is important to collect qualitative as well as quantitative data, to understand the 'how' of a statistical association and its meaning to those effected by comprehending their worldview. This could advance the field by addressing several aspects of the question - shedding light on mechanisms, context, and policy implications. Mechanistically, does concern and engagement with environmental issues factor in how environmental features affect a person? This could offer alternative explanations - the predominant hypothesis is that air pollution effects people psychologically via the biological impacts of inhalation (Block and Calderon-Garciduenas, 2009, Braithwaite et al., 2019b, Brockmeyer and D'Angiulli, 2016, Buoli et al., 2018, Calderon-Garciduenas et al., 2015, Fluegge, 2016, Plusquellec and Lanoix, 2013, Suades-González et al., 2015, Tzivian et al., 2015b) but if young people are aware of and concerned about air quality in their area, triggered by their knowledge about local air quality and/or their experience of it (e.g. the sight or smell of cars), then the worry or concern could be an indirect mechanism of impact. The context of the association could also provide useful information - for example, what else is going on in adolescents' lives besides these effects? This may inform why some people are more affected than others or more vulnerable, and tells us what else is important to them and their wellbeing. Finally, qualitative data is crucial for understanding the correct policy implications of environmental health impacts. What solutions would satisfy the people who are affected most? Which would fit in the context of their other needs and priorities? Do they want to be involved? Do they trust government and environmental communications? How can we be more impactful, get them on board with changes or policies, and convince them they are worthwhile? Young people are

politically disenfranchised, so how can we support them to improve things and empower them?

Therefore, a deeper analysis to supplement epidemiological study of the associations between noise and air pollution and psychological outcomes in young people should bring a variety of benefits to our understanding of the current literature and new findings, by going beyond the objective transaction of exposure and outcome, to understand what it is like for young people in their experience. An interpretivist theoretical approach underlies this thinking, as these experiences are assumed to be changeable and context-dependent, where people's realities are actively created by the individual as they interact with the world (Ezzy, 2013, Holloway, 1997).

1.3 Covariates, moderators, and mediators in the relationships between air pollution, noise, and psychological outcomes

Therefore, there are plausible mechanisms through which air pollution and noise can impact adolescent psychological outcomes. It is proposed that air pollution and noise exposure affect mental health and cognitive outcomes in adolescence, which will be tested during this thesis. According to predominant thinking in the field, effects of air pollution are attributable to neuronal injury from exposure to fine particles and systemic oxidative stress (Costa et al., 2017) and effects of noise are due to chronic stress and arousal, and disruption of learning, sleep, and development (Basner et al., 2014). Qualitative research gathering the perspectives of young people will further our understanding of these relationships in terms of additional or alternative levels of explanation and contextualisation. To address questions of confounding, moderation, and mediation, modelling the associations of pollution or noise with

psychological outcomes in adolescence will involve the inclusion of several additional key variables.

Interrelationships between cognition and mental health

Cognition and mental health are deeply interconnected, and it is theorised that problems with each can exacerbate the other during development (Bortolato et al., 2015, Goodall et al., 2018, Moran, 2016). For example, a recent meta-analysis found that depressed youth (12-25 years old) have poorer attention, verbal memory, visual memory, verbal reasoning/ knowledge, and Intelligence Quotient (IQ) (Goodall et al., 2018). Further, global cognitive impairment (across many domains) is a key established feature of schizophrenia and bipolar disorder (Bortolato et al., 2015). Schizophrenia is particularly associated with worse processing speed and episodic memory, as well as general intellectual function, working memory, learning, language, and executive function (Bowie and Harvey, 2006). This set of deficits precedes illness onset (and does not particularly decline after) and is present but attenuated in first degree relatives (Owen et al., 2011). Bipolar disorder is associated with deficits in attention, processing speed, episodic memory, executive functions, verbal learning and memory (Bortolato et al., 2015). These deficits tend to be less severe than in schizophrenia. Both sets of deficits are present even in euthymic periods (asymptomatic periods), so are trait-markers rather than state-markers of disorder (Bortolato et al., 2015). Source-monitoring difficulties are particularly associated with hallucinations (Brookwell et al., 2013). Evidence has also shown that cognitive deficits are an important signifier of anxiety, specifically working memory (Moran, 2016), and the evaluation of risk (Maner and Schmidt, 2006). However, some of these cognitive patterns associated with mental health could be the result of it rather than the cause. Additionally, they could reflect an aspect of task-performance other than trait ability (for example, motivation or distraction). However,

bi-causal relationships between cognition and mental health are entirely plausible, in fact this is the foundational principle of cognitive behavioural therapies (CBT). In CBT, which has good efficacy for treating anxiety disorders other mental health conditions, the focus is on reframing thoughts (cognition) and behaviours, which will in turn inform mood and mental health (Hofmann et al., 2012). Therefore, cognition is an important variable to test in analyses of mental health. On one hand, its interactions with mental health could lead to effect modification. Additionally, air pollution or noise could affect mental health through its effects on cognition (mediation).

Concurrent exposures

Real life exposures are not presented in isolation and many environmental exposures are correlated, so it is important to evaluate whether exposures are confounding one another – either a spurious effect being produced due to positive correlation or an existing effect being masked due to negative correlation. Therefore, correlations between exposures will be measured and multi-pollutant models run to further understand effects in the data. Further, there is evidence that contexts of high/low exposure to noise can modify effects of air pollution, and vice versa (Tzivian et al., 2017, Wurth et al., 2018). Therefore, effect modification by high/low noise or air pollution exposure should be tested in studies on this subject. The areas that adolescents occupy, most notably school and neighbourhood can vary almost infinitely with their visual, auditory, olfactory, gustatory, tactile, social, and conceptual features, which will have a psychological effect as they are processed. The settings and their components also broadly determine the level of exposure adolescents are subject to. Therefore, this is an important confounder to consider, although any given setting will share much variance with the exposures of interest themselves.

Sociodemographic factors and lifestyle

Age, sex, socioeconomic status (SES), ethnicity, school type (independent/state) and first language are demographic factors that may act as confounders of the association between amount of exposure to air pollution or noise and psychological outcomes (Fecht et al., 2015, Glad et al., 2012, Hyde, 2016, McCarthy and Coleborne, 2012) and thus should be controlled for in epidemiological analyses. These could also be moderators – acting as vulnerability factors – which has not been widely investigated in the literature, although a recent study by Zhang and colleagues (Zhang et al., 2017) reported that both age and gender moderated effects of air pollution on adult happiness and mental health. Religion could be protective (Ganga and Kutty, 2013, Hayward and Krause, 2014) so potential effect modification by religious belief will be explored. Smoking and environmental tobacco smoke (ETS) should additionally be controlled for as these will affect air quality, indoor/outdoor behaviours, and are associated with psychological outcomes (Hwang et al., 2012, Pagani, 2014).

Health

Sleep quality, Body Mass Index (BMI), breathing capacity (Forced vital capacity [FVC], forced expiratory volume [FEV]), asthma/ eczema/ allergies, low Intelligence Quotient (IQ), are associated with the mental outcomes and/or could be influenced by environmental exposures, so are most likely to be mediators or moderators (de Bont et al., 2019, Glad et al., 2012, Goodwin et al., 2012, Huang et al., 2017, Russell-Mayhew et al., 2012, Sygna et al., 2014, Takano and Inoue, 2017, Tarokh et al., 2016, Viner, 2015).

The aforementioned causal relationships are outlined in the directed acyclic graph below (DAG, Figure 1). Based on the variables, and their probable bivariate causal relationships, DAGs show which variables are potential mediators on the causal pathway and which

variables are potential confounders (variables influencing the exposure and outcome which pose the risk of masking a real relationship or creating an illusory association if not controlled for). DAGs can also be used to identify colliders (variables affected by both exposure and outcome, which pose the risk of distorting associations if adjusted for). The necessity of controlling for wider environment, sociodemographics and smoking/ETS, as ancestors of exposures (green) and outcomes (blue), and therefore potential confounders, is exemplified by the pink variables and pathways (which indicate a biasing risk). Some of the mechanisms and potential moderators represented in the DAG will also be explored in this project.

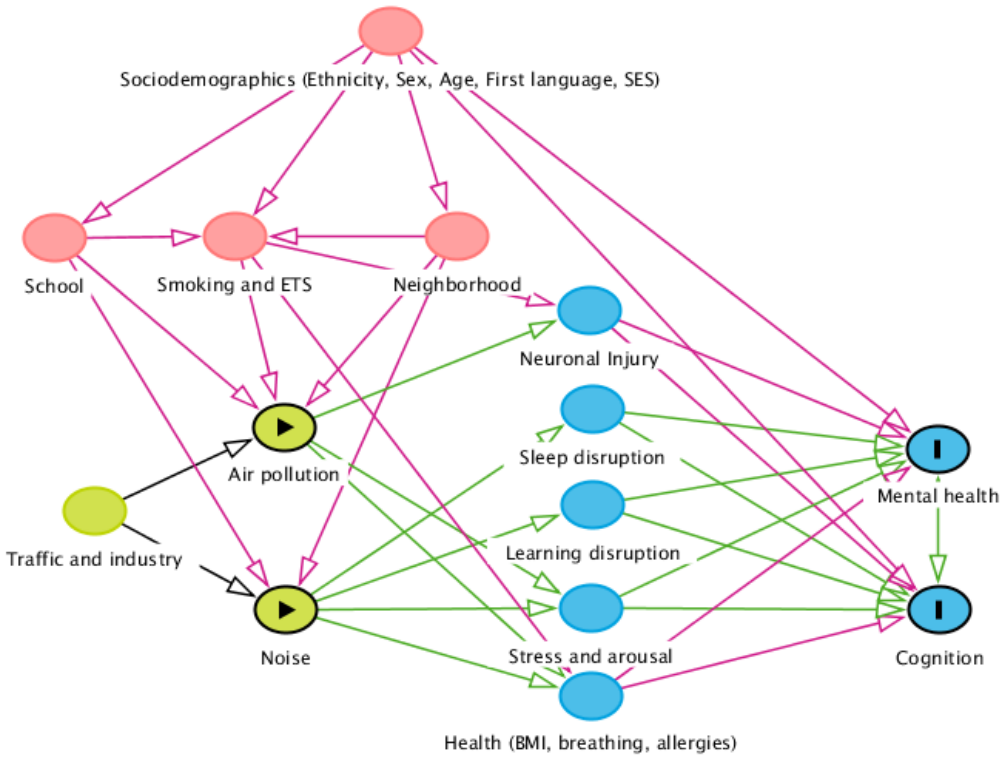


Figure 1. DAG of causal pathways involved in exposure-response relationships, created using R package "dagitty". Abbreviations: SES- socioeconomic status. ETS- environmental tobacco smoke. BMI- body mass index.

1.4 Gaps in the literature, aims, and hypotheses

The overarching aim of this thesis is to better understand how exposure to air pollution and noise influence psychological outcomes, specifically mental health and cognition, in adolescents. There are several gaps in the existing literature on this question which I seek to address in this thesis. In the overarching question of exposure to air pollution and noise and their association with mental health outcomes, the adolescent literature is small, despite this being a developmentally vulnerable group. Further, there have not been recent comprehensive reviews (since 2015) on air pollution or noise exposure in relation to cognitive outcomes. Nor has there been a vast literature evaluating how these outcomes might moderate or mediate one another. Additionally, understanding this age groups' subjective experiences of their physical environments will help us to contextualise results, assist with explaining unexplained variance, inform modelling choices, interpret findings, and make policy recommendations. Further, the mechanisms of these associations are not greatly understood, nor whether these effects are moderated by sociodemographic factors or effect everyone unilaterally.

Therefore, this thesis will make a significant and original contribution to the literature through the following aims, by chapter:

1. To carry out a comprehensive up-to-date review of the evidence regarding exposure to air pollution and noise and adolescent cognitive and mental health outcomes (Chapters 2 & 3)

2. To interview UK adolescents about how their environments affect them, including the involvement of young people to improve the validity of what these responses are interpreted to mean (Chapter 4)

3. To use environmental epidemiological methods to evaluate the associations between a range of air pollutants and noise exposure and psychological outcomes in adolescence (Methods described in Chapter 5, results in Chapter 6)
4. To evaluate whether such effects are especially pronounced in certain sociodemographic groups (Chapter 7)
5. To evaluate the mechanisms of sleep, mental, and physical health outcomes as mediators of these relationships (Chapter 7)
6. To discuss the findings altogether, interpreting the evidence and implications for future research, policy and practice (Chapter 8).

Hypotheses

1. The existing literature will be supportive of associations between higher levels of air pollution and noise exposure and poorer psychological outcomes in adolescence.
2. The qualitative element to this mixed methods project will be without hypotheses, allowing themes to emerge from the data inductively.
3. Higher levels of exposure to air pollution and noise will be associated with poorer cognitive abilities and worse mental health outcomes.
4. The psychological outcomes of more socially disadvantaged groups - by socioeconomic status, ethnicity, and gender - will be especially negatively associated with exposure to air pollution and noise, reflected in interaction effects.
5. Sleep, physical health factors, and mental health/cognition will mediate the relationships between air pollution and/or noise exposure and psychological outcomes in adolescence.

Chapter 2: A systematic review of the literature evaluating associations between air pollution exposure and psychological outcomes in adolescence

2.1 Background

In conducting an epidemiological study of psychological outcomes and air pollution in adolescence, it is important to gauge the state of the field and findings of prior literature, to guide the research and ensure: (i) methods are consistent enough with prior studies in the area that the results are comparable; (ii) that the research adds novelty; (iii) and that it is well-conducted in line with current best practice. Conducting a systematic literature review and meta-analysis of existing research also serves as evidence in answering the question regarding the current state of the evidence available pertaining to the psychological effects of air pollution. A previous review synthesised the evidence for cognitive associations with air pollution across the lifespan prior to November 2015 (Clifford et al., 2016) and another synthesised the evidence for air pollution and mental health in adults (Braithwaite, 2019). However, these reviews did not meta-analyse the evidence, were largely focussed on other age ranges, and impactful studies are known to have been published since, warranting an updated review of the literature.

Therefore. the objective of this chapter is to provide an updated review of the literature and evaluation of the evidence base regarding the associations between air pollution and cognition and mental health in adolescents.

2.2 Methods

This review has been written in accordance with the PRISMA (The Preferred Reporting Items for Systematic reviews and Meta-Analyses) checklist for reporting systematic reviews

and meta-analyses (Page et al., 2021). The wider systematic review project, which evaluated evidence across all ages as opposed to just adolescents, was registered on PROSPERO prior to commencing (CRD42019151923). The PECOS (Morgan et al., 2018) statement for this research is as follows:

- Participants: outcomes measured in human populations aged 10-19 years, exposure anytime during lifetime including prenatally.
- Exposure: environmental air pollution. Specifically, PM_{10}, $PM_{2.5}$, PM_{coarse} (PM >2.5 μm and ≤ 10 μm), Ultrafine Particles (UFP), and other particulate matter, NO_2 (Nitrogen Dioxide), NOx (Nitrogen Oxides), ozone, hydrocarbons (PAH's, benzene), SO_2 (sulphur dioxide), VOC's (Volatile Organic Compounds), CO (carbon monoxide), CO_2 (carbon dioxide), BC (black carbon), EC (elemental carbon), ECAT (elemental carbon attributable to traffic)
- Control/Comparison: persons less exposed (continuously or in high/low exposure groups)
- Outcomes: general population cognitive abilities, specifically general cognitive ability and IQ, executive function (EF), processing speed, academic ability, attention, memory and learning, language/reading abilities, reaction times, and visuospatial skills. Mental health outcomes, specifically depression, anxiety disorders, Obsessive Compulsive Disorder (OCD), bipolar disorder, psychosis, internalising symptoms, externalising symptoms, Post-Traumatic Stress Disorder (PTSD), self-harm, eating disorders, conduct disorder.
- Study Design: epidemiological study designs - cohort, case-control, and observational studies (observational studies of exposure to air pollution in day-to-day life, as opposed to pollutants experimentally presented in a lab).

2.2.1 Search strategy and eligibility criteria

The online databases EMBASE (via Ovid), PubMed, PsycINFO (via Ovid) and Web of Science were searched for epidemiological studies of the relationships between air pollution exposure up to and including adolescence (including prenatally) on human cognitive functioning and mental health assessed during adolescence. Specific search terms can be

found in Appendix I, and included searches for studies involving children though they were not included in the selection criteria for this review. These searches were conducted on 26/09/2019 and 07/04/2022 for cognition, and 01/08/19 and 28/03/22 for mental health by title/abstract. Reference lists of studies were also scanned for any further literature.

The eligibility criteria were as follows:

1. Studies reporting cognitive and mental health outcomes in adolescence. However, studies of neurodevelopmental, learning, or neurological disorders were not included (autism, learning disability, attention deficit hyperactivity disorder (ADHD)).

2. Measured and modelled air pollutants included PM_{10}, $PM_{2.5}$, PM_{coarse}, UFP, and other particulate matter, NO_2/NOx, ozone, hydrocarbons (PAH's, benzene), SO_2, VOC's, CO, CO_2, BC, EC, and ECAT . Airborne metals (e.g. copper, lead) were not included. Studies using proxies for air pollution exposure (e.g. air quality construction works, hard fuel use, distance to traffic/industry) were not included.

3. Conference abstracts and grey literature were eligible for inclusion if they reported an independent effect of exposure on outcome that could not be found in a published academic paper.

4. Publications written in English, as there were not resources available for translation.

5. Outcomes measured in human populations age 10-19. Studies of childhood with age 10 as the upper range and studies of adulthood with 19 as the lower range were excluded. Exposure could occur anytime before or during outcome measurement (including prenatally).

2.2.2 Review process

For the cognition searches, the candidate and a second reviewer (YBK or CT) carried out the search and screened titles, abstracts, then full texts, against the above eligibility criteria. Any disagreements as to paper eligibility were resolved by an independent third reviewer (RBS). Reviewers independently extracted the fields Title, Authors, Year, Study location, sample

size, age, gender, study design, exposure, exposure period, method of exposure assessment, cognitive outcome, measure of cognitive outcome, confounders measured and adjusted for, statistical analysis conducted, outcome/results, conflict of interests and funding. The mental health search (2018-2022) and data extraction was unfortunately not conducted in duplicate due to limitations on resources, but the same process was applied. Where a publication was not available through institutional access, it was requested from authors.

2.2.3 Risk of bias and Quality of Evidence Assessment

The OHAT Risk of Bias Rating Tool for Human and Animal Studies (OHAT, 2015) was used to evaluate risk of bias across the following six domains: selection bias (were participants a representative sample who are comparable across exposure levels?); confounding bias (were key socioeconomic covariates and possible other exposures controlled for?); attrition bias (low levels of exclusion or attrition, or demonstration it should not appreciably bias results); exposure characterisation (was the characterisation of air pollution a valid approach? i.e. measured, a biomarker of exposure, or a validated model); outcome characterisation (was the characterisation of cognition a valid approach? i.e. a standardised and validated cognitive test, mental health questionnaire, or clinically documented mental health outcome); and selective reporting (were all measured outcomes and planned analyses reported in results?). Ratings of 'definitely high', 'probably high', 'probably low', and 'definitely low' risk of bias were applied in duplicate and agreed between two reviewers, using criteria for the ratings from OHAT Risk of Bias Rating Tool (NTP, 2015). These specific criteria for each rating for each domain are provided in Appendix II. The ratings for each field by paper are provided in the main summary tables (1a and 1b).

These fields were then used in application of the GRADE (The Grading of Recommendations Assessment, Development and Evaluation) approach for evaluating the overall quality of evidence for different cognitive outcomes (Morgan et al., 2016). In accordance with OHAT Approach for evidence integration, the initial GRADE confidence level was based upon the following key features of the study design (moderate confidence for 3 features, low for 2 features, very low for 1 feature): exposure prior to outcome (longitudinal evidence), individual outcome data, comparison group used (or participant outcomes compared over a range of continuous exposure), or controlled exposure, which did not arise in this review due to its focus on observational epidemiological evidence (Rooney et al., 2014). Evidence for associations could then be downgraded on the basis of Risk of Bias concerns (ascribed when ≥ 50% of studies rated 'probably high' or 'definitely high' risk of bias in ≥2 Risk of Bias domains) imprecision of effects, inconsistency, indirectness, and publication bias. Finally, ratings could be upgraded if a large or very large effect was observed, if any plausible biases would have reduced the observed effect (or increased the effect if no effect was observed), or a dose-response gradient was observed. All GRADE quality of evidence decisions and reasons are specified in Table 2. Exposure-outcome associations only studied once could not be evaluated in this way (listed with direction of effect in Appendix III).

2.2.4 Meta-analysis

All outcomes and exposures from identified studies were tabulated to identify effects which (i) reflected valid measurements of the same cognitive faculty (e.g. neurodevelopment, general cognition, verbal fluency, executive function), (ii) did not come from overlapping study populations, and (iii) had compatible (the same or equivalent) effect statistics, performing meta-analysis if at least three estimates met these criteria. Heterogeneity in

methods generally precluded pooling effects which predicted the exact same outcome (i.e., estimates arising from the same task on the same scale), therefore most meta-analyses reflect trends in the literature rather than specifying an exact dose-response relationship in terms of impact on real world behaviour. Where estimates were modelled at different resolutions of exposure (e.g. μgm^3, parts per billion (ppb), IQR(Interquartile range), tertile increments) they were transformed to the same scale to improve interpretability. If further details were required from authors to perform meta-analysis these were requested. DerSimonian and Laird's inverse-variance, random effects models was estimated using the package "meta" in R version 3.6.1 (Balduzzi et al., 2019, Core, 2019).

2.3 Results

We identified 43,262 records in the initial searches, which after exclusions left 54 studies to be included in the qualitative synthesis, 26 evaluating cognition and 28 evaluating mental health respectively (see Figure 2). The level of agreement on paper selection between the first two reviewers was high (Cohen's Kappa 0.94, 95% Confidence Interval (CI): 0.88-1). There were 10 cognitive studies from Europe, 10 from the USA, 3 from China, and 1 from each of Kenya, Iran, and Indonesia, respectively. Characteristics of these individual studies are summarised in Table 1(a). There were 13 mental health studies from Europe, 8 from the US and Canada, 6 from East Asia, and 1 from Australia. Characteristics of these individual studies are summarised in Table 1(b).

The most common approach used to estimate exposure to air pollution was to predict exposure at residential (or occasionally school) addresses using air quality models, though some studies measured air pollution at home or school, or measured biomarkers of exposure. When assessing risk of bias across the 54 studies, the majority of studies (83%) raised some

concerns about bias (domains with ‘probably high’ or ‘definitely high’ risk of bias), with 3 cognitive and 6 mental health studies devoid of bias concerns. This was largely due to attrition or exclusion bias and selection bias.

Table 2 shows the GRADE-assessed collective certainty of evidence for each exposure-outcome association investigated at least two times in this literature. Appendix III lists the associations investigated in only a single study, thus highlighting gaps in the literature. Only two associations ($PM_{2.5}$ and executive function skills, including attention, working memory and inhibition, and $PM_{2.5}$ and psychiatric emergency) were assessed as high-quality evidence, with 5 sets of evidence for associations of moderate quality and 23 low quality. Nineteen of these low-quality associations were for mental health domains, often because there were only two studies evaluating that outcome (imprecision), whereas a smaller number of domains were evaluated in the cognitive literature.

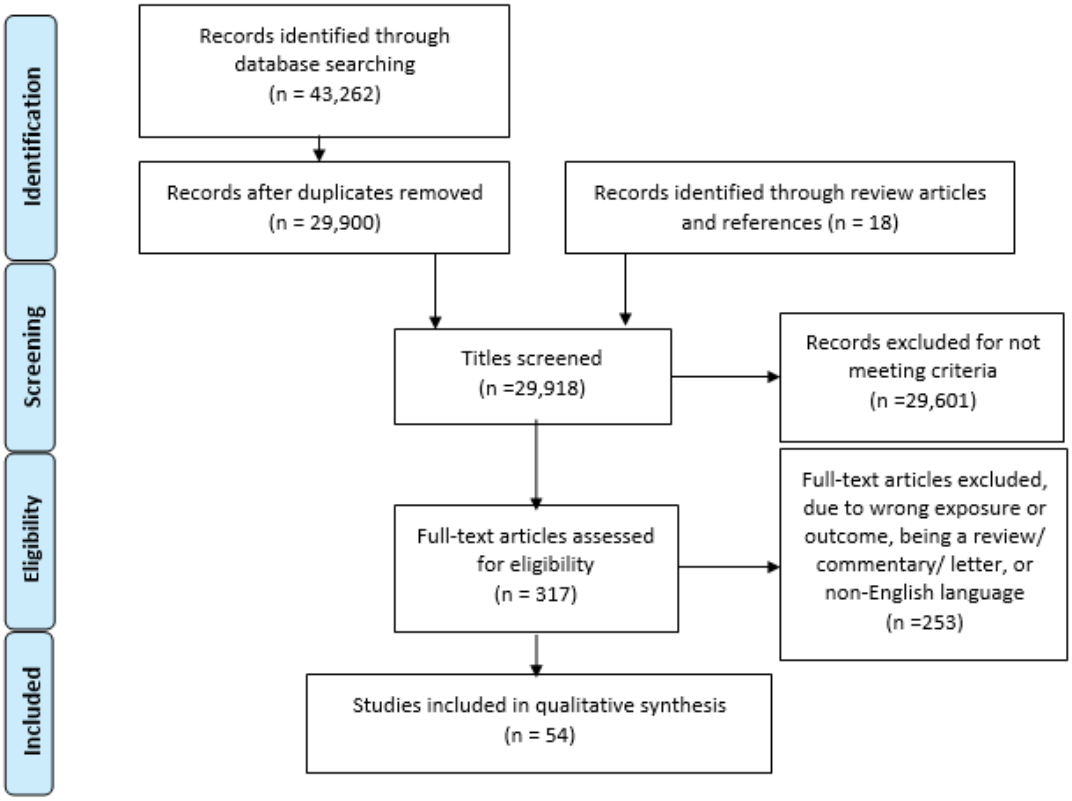


Figure 2. PRISMA diagram of search results[1]

[1] *From:* Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). *P*referred *R*eporting *I*tems for *S*ystematic Reviews and *M*eta- *A*nalyses: The PRISMA Statement. PLoS Med 6(7): e1000097. doi:10.1371/journal.pmed1000097

Table 1a: Characteristics of eligible cognition studies included in this systematic review

Author and year	Analytical sample:	Measurement of exposure	Measure of cognition	Confounders considered (controlled for or demonstrated it was unrelated)	Outcomes- most adjusted model	Selection bias for comparison	Accounts for important confounding and modifying variables	Outcome data complete without attrition or exclusion?	Exposure characterisation	Outcome characterization	Were all measured outcomes reported? [2]
	n, age (μ and σ unless stated otherwise), gender, location				*= p<0.05 DL=definitely low PL=probably low PH=probably high DH=definitely high						
(Alemany et al., 2018a)	N= 1488 inattentiveness/ 1591 working memory, Age 7-11 (mean 8.52±0.87), 47.70% female, Barcelona	Average EC, PAHs and NO_2 measured 4x across one year at school (outdoor)	ANT and n-back test *Inattentiveness and working memory*	Age, sex, maternal education, maternal prenatal smoking, ETS at home, neighbourhood SES, Air Pollution at home	1 IQR increase PAH: inattentiveness mean ratio 4.44 (95% CI: 0.48, 8.40), n-back numbers beta −0.08 (95% CI: −0.14, −0.02), n-back words beta −0.02 (−0.07, 0.04). Adverse associations with PAHs and NO_2 on inattentiveness trajectories were stronger or limited to Apolipoprotein E (APOE) e4 carriers (P-interaction 0.15 and 0.08, respectively). EC similar	PL	PL	PH	DL	DL	DL

[2] PL: Probably Low
DL: Definitely Low
PH: Probably High
DH: Definitely High

(Amanzadeh et al., 2020)	N= 79,000, 56% female, High School examinees, Tehran and Mashhad (Iran)	Averaged hourly measurement of particulate matter, CO, nitrogen oxides, sulphur dioxide, ground-level ozone within the city 4h before and during the exam	Subject-specific concour mock test score from 2012 to 2017	Student ability, parental education, family income, education quality	One standard deviation increase in PM_{10} and $PM_{2.5}$ is associated with 0.047 and 0.029 standard deviation decrease in test scores. Other pollutants, except for SO_2 which was positively related, do not show a robust relationship with test scores.	PH	PH	PH	PL	PL	DL
(Chiu et al., 2013a)	N-=174, Age 7-14, median age 10, 53.4% female, Boston, USA	Spatiotemporal model for 24-hr measures of BC based on 6,021 observations 82 locations. Used to calculate quartiles of lifetime exposure for each participant	Completed the Continuous Performance Test (CPT), giving omission errors, commission errors and Hit Reaction Time (HRT) calculated *Inattentiveness*	Maternal race and educational status and child's IQ, sex, age at CPT assessment, exposure to prenatal maternal smoking and postnatal secondhand smoke, log-transformed peak blood lead level, and community violence	Compared to lowest quartile, 4th quartile of black carbon exposure In 7-14 year olds association with increased: Omission error (β=0.62, -8.57 to 9.81). Commission error (β = 3.32, -0.87 to 7.51) Slower RT (β=1.75, -4.44 to 7.94). Latter two did differ significantly between first and second and/or first and third quartile.	PL	DL	PH	PL	DL	PL

(Clark-Reyna et al., 2016c)	N= 1895 Age 8-13(mean 10.4±0.8), 50.08% female U.S.A	Modelled 2005 residential-census tract total respiratory risk (RR), on-road mobile respiratory risk; point respiratory risk; non-road mobile respiratory risk; non-point respiratory risk; total diesel particulate matter (PM), on-road diesel PM, non-road diesel PM	Parent reported grade point average (GPA) in 2012 *Academic ability*	Free school meals, mothers education, ethnicity, teenage motherhood, mothers English proficiency, child sex, child's current age, school clustering	Controlling for covariates, total respiratory risk (beta -0.26, p=.004), and total diesel risk (-0.023, p=.008) predicted GPA. So did on-road RR, non-road RR, non-point RR, on road diesel risk, and non-road diesel risk (but not point RR)	PL	DL	PL	PL	DH	DL
(Clark-Reyna et al., 2016b)	N= 1895 Age 8-13(mean 10.4±0.8), 50.08% female U.S.A	Modelled 2005 residential-census tract total respiratory risk, on-road mobile respiratory risk; point respiratory risk; non-road mobile	Parent reported GPA *Academic ability*	Free school meals, mothers education, ethnicity, teenage motherhood, mothers English proficiency, child sex, child's current age, school clustering, children's caretaker-rated subjective health status	Controlling for subjective health status, both total respiratory risk (beta −0.023, p= 0.004) and total diesel PM risk (beta −0.021 p=.009) significantly predicted GPA	PL	DL	PL	PL	DH	DL

		respiratory risk; non-point respiratory risk; total diesel particulate matter, on-road diesel PM, non-road diesel PM									
(Clark-Reyna et al., 2016a)	N= 1319 Age 9-13 (mean 10.4 ±0.8), 50.40% female U.S.A	Modelled 2005 residential census-tract known metabolic disruptors (arsenic, benzene, cadmium chloride, chlordane, dibutyl phthalate, diesel exhaust, ethylene glycol, and naphthalene)	Parent reported GPA *Academic ability*	Free school meals, mothers education, ethnicity, teenage motherhood, mothers English proficiency, child sex, child's current age, school , child health, child BMI	After adjustment, known MDS predicted GPA (r=-.0114, p=.03), mediated by indirect effects of BMI on GPA	PL	DL	PL	PL	DH	PL

(Dorizas et al., 2015)	N=93 Age 11-12, Greece	Contemporaneously measured $PM_{0.5}$, PM_1, $PM_{2.5}$, PM_5, PM_{10}, TPM, UFP, CO, CO_2, VOCs, in classroom	Attention/concentration tests from the SINPHONIE project *Attention and Concentration*	None	17.01 % increase indoor CO_2 concentrations associated with reduction in the performance by 16.13 % between days; CO_2 and performance correlated r=-0.305--0.5	PH	DH	DL	DL	PL	PH
(Forns et al., 2017)	N= 1439 Mean age 11.4(±0.6), 48.4% female, Barcelona	Measured school indoor and outdoor Traffic Related Air Pollution (TRAP): $PM_{2.5}$, EC, UFP, NO_2, averaged for year of baseline assessment	N-back test in 2012(baseline) and 2015 (follow-up) *Working memory*	age, sex, maternal education, ETS, teacher reported ADHD, home neighbourhood urban vulnerability index , BC at home, school and residential greenness, noise (measured at same time as AP) hour of test, temp on day of test, humidity on day of test	All pollutants associated with WM. E.g. IQR increase outdoor NO_2 on annual change in 3-back d' coef =− 4.22, 95% CI: − 6.22, − 2.22. Indoor UFP coef =− 4.12, 95%CI: − 5.68, − 1.83, equivalent to − 20% (95%CI: − 30.1, − 10.7) and − 19.9% (95%CI: − 31.5, − 8.4) less annual working memory development respectively	DL	DL	PL	DL	DL	DL
(Grineski et al., 2016)	N= 1888 Age 8-13(mean 10.39 ±0.77), 50% female U.S.A	Modelled school-census tract total respiratory risk, on-road mobile respiratory risk, non-road mobile respiratory risk, total diesel particulate matter, on-road diesel	Parent reported GPA *Academic ability*	Individual level: free or reduced price meals, maternal education, teen mother, race/ethnicity, mother's english, child's age, child sex. School level: total enrolment, percentage qualifying for free or reduced meals, teachers with a masters, special education, race/ethnicity.	Drop in GPA per IQR increase in total respiratory risk(RR)- 0.403 (-0.635, -0.173), total diesel PM risk -0.222 (-0.371, -0.072). Also significant were effects of non-road RR, on road RR, on-road diesel PM risk, non-road diesel PM risk.	PL	PL	PL	PL	DH	DL

		PM, non-road diesel PM, 2005									
(Gui et al., 2020a)	BRIEF questionnaire (metacognition and behavioural regulation): N= 5028, Age 6-12 (9.0±1.7), 46.3% female. Computerised testing (working memory and executive function): N= 522, Age 9.7±1.6, 48.2% female. China.	Daily concentration of $PM_{2.5}$, PM_{10}, NO_2, SO_2, ozone (O_3) at residential address assigned using inverse distance weighting interpolation modelling to find one-year mean exposure level	BRIEF parent report questionnaire, CBTT, SCWT, WCST *Working memory, inhibitory control, cognitive flexibility, metacognition*	Sex, age, only child, delivery mode, birth weight, physical activity, screen time, paternal and maternal education, parental smoking, child's IQ, monthly household income, screen time	Working memory: Negatively associated with 1IQR increase PM2.5 (forward recall -0.72 points, backward recall -0.26) and PM10 exposure (forward -0.55, backward -0.67) Inhibitory control: one IQR increment in PM2.5 exposure associated with 48.04ms (95%CI:2.18 to 93.89) slower response. Metacognition: associated with SO2 (0.73 higher scores, 0.40-1.05), and O3 (0.87, 0.45-1.27). Behavioural regulation: associated with SO2 (0.69, 0.37-1.02). Cognitive flexibility not associated.	PL	PL	PL (BRIEF questionnaire)/ DH (computerised testing)	PL	PH (BRIEF questionnaire)/ DL (computerised testing)	DL
(Jalaludin et al.)	N= 16591, Age 10.4±2.2, 49.2% female, Indonesia	Annual concentration of ambient $PM_{2.5}$ at subdistrict level, and participant's residence in forest fire-	Raven's Coloured Progressive Matrices test *Reasoning, general intelligence*	Age, gender, urban/rural residence	Positive association between $PM_{2.5}$ levels and Raven's scores for mean $PM_{2.5}$ levels in the year of survey (0.1; 95%CI 0.01,0.19) and for lifetime $PM_{2.5}$ levels (0.1; 95%CI 0.003,0.2). Although currently living in forest-fire prone area (forest fires release high levels of fine particles) not associated, those who had lived in one across their lifetime had	PL	PH	DH	PH	DL	PL

		prone province			significantly lower scores than those who had never lived in a forest-fire prone province (-1.5%, -2.94%to -0.07%).						
(Julvez et al., 2021b)	N= 1298 (87 exposures measured during pregnancy, 122 during childhood), Age 6-11 (mean 8), 45.3% female, Europe	Estimated $PM_{2.5}$, NO_2 levels from geo-coding home address for pregnancy period and home and school addresses for childhood period	Raven's Coloured Progressive Matrices test, Attention Network Test, N-Back task *Fluid intelligence, attention, working memory*	Cohort inclusion, maternal age, maternal education level, trimester of year of child conception, child age at cognitive examination, child sex	ETS associated with lower fluid intelligence (-0.886, 95%CI -1.419;-0.353, $p<0.001$), indoor $PM_{2.5}$ exposure associated with lower working memory scores (-0.093, 05%CI -0.161:-0.024, $p<0.008$)	PL	PL	PH	PL	DL	PL
(Kicinski et al., 2015a)	N=606 9th Graders, (mean=14. 9 ± 0.7), 46.5% female, Belgium	Traffic exposure factor (composed based on a benzene biomarker t,t-Muconic Acid (t,t-MA-U), time spent in traffic, and both residential and school traffic density)	Neurobehavioral Evaluation System computerized battery of tests (NES3) *sustained attention, short-term memory, manual motor speed*	Gender, age, smoking, passive smoking, level of education of the mother, socioeconomic status of the family, time of the day, and the day of the week	1sd increase in associated with reduced: Sustained attention: -0.26sd (-0.51, -0.02) Short term memory: -0.17sd, (-0.38, 0.04) and Motor speed: -0.10sd (-0.27, 0.07).	DL	DL	DL	PL	DL	PL

(Kicinski et al., 2016)	N= 895, Age 14-15 (mean 14.88±0.63), 48.60% female, Belgium	Measured t-t-MA-U (a Benzene metabolite) and lead in urine up to 30 days before assessment	continuous performance test and digit span test *Attention and working memory*	Urinary creatinine, day of the week, time of the day, sex, age, passive smoking, the highest level of education of either parent, ethnicity	10fold increased t,t-MA-U: 0.14 SD lower sustained attention (95% CI: 0.26, 0.019); 0.17 SD lower short-term memory (0.31, 0.030); 12.2ms higher reaction time (4.86, 19.5); 0.51 more errors of omission (0.057,0.97); 0.2 less digits in forward digit span task (0.38 to 0.026) and 0.15 digits fewer in backward digit span (0.32, 0.022) .	PL	DL	PL	DL	DL	DL
(Margolis et al., 2021)	N=200, 8-14 years (mean age 10.4 and 13.7), 59% female, New York, USA	PAH exposure of mother measured during third trimester	Inhibitory control (NEPSY test Inhibition) at mean age = 10.4 years, and Woodcock-Johnson Tests of Achievement-III (WJ-III) at mean age = 13.7 *Inhibition, spelling, reading, maths*	ethnicity, maternal country of birth, language of prenatal interview, maternal marital status, maternal years of education, material hardship, quality of home caregiving environment, and early life stress	higher prenatal PAH was significantly associated with worse spelling skills (WJ-III Spelling, β = - 0.16, 95%Confidence Interval [CI]: -0.30,-0.02, p=0.02), trends for reading and maths. higher PAH was significantly associated with worse inhibitory control (β =-0.15, -0.29-, -0.01,p=0.03).	DL	DL	DH	DL	DL	DL
(Powdthavee and Oswald, 2020b)	N= 34,000, Age 15 or older, UK	NO_2 and PM_{10} levels using regression models	Delayed-recall test of 10 words *Memory*	Age, sex, ethnicity, average income by Local Authority District (LAD), relative deprivation rank index by LAD in 2010, log of equivalent household income, highest qualification, unemployment status, marital status, health status, number of children	NO_2: beta -0.011 (95%CI -0.017 to -0.0005) PM_{10}: -0.031 (95%CI -0.05 to -0.012)	DL	PL	PH	PL	PL	DL

(Russ et al., 2021)	N=572, mean age 10.92±0.27 (baseline), then 70-79 (follow-up), 46.9% female, Scotland	Modelled residential $PM_{2.5}$ to estimate in utero exposure, and lifetime exposure (at specific intervals, and accumulation of risk)	Moray House Test *IQ*	Sex, father's occupation, smoking, age 11 IQ (at age 70+)	Higher exposure in utero was associated with worse change in IQ from 11 to 70 years (beta = -0.006, p=0.03) but not from 70-79. Other windows of exposure and accumulation of exposure were not associated with IQ.	DL	PH	DH	PL	DL	DL
(Saenen et al., 2016)	N= 310, Age 8-12 (mean 10.2±1.3), 49.10% female, Belgium	Modelled residential $PM_{2.5}$, PM_{10}, and BC for 2 days before assessment (recent) and for year before (chronic) assessment; measured air pollution In classroom on day of testing; assessed residential proximity to major roadways (RPMR)	Stroop test + four tests of Neurobehavioral evaluation system 3 (NES3): continuous performance test, digit span test, digit-symbol test, and pattern comparison test *Working memory/attention, short-term memory, processing speed*	Sex, age, education of mother, highest rank of either parent, passive smoking, out of school sport, traffic noise, computer time, learning effect, day of the week and month of exam.	1IQR increase in recent $PM_{2.5}$ exposure worsened pattern comparison by 0.087s (SE: ±0.034, p=0.01). 1IQR increase chronic $PM_{2.5}$ slowed continuous performance task 9.45msec (SE: ±3.47, p = 0.007) and Stroop Test 59.9(± 26.5 msec, p = 0.02). IQR increased $PM_{2.5}$ in class on test day slowed digit symbol task 2.05s(95% CI: 0.43-3.66), p=.01. All similar for PM_{10}. BC and distance to major roadways not consistently associated with cognition.	DH	PL	PL	PL	DL	DL

(Sears et al., 2020)	N=221, mean age 11 ±2.5, 47% female, USA	Indoor residential measurement of PM_{10} for one week	Behavioural Assessment and Research System (BARS), CPT, and Selective Attention Test (SAT). *Attention and cognitive control*	age, maternal education, race, sex, ETS, socioeconomic status, age of housing unit	Higher indoor PM_{10} associated with more commission errors on the CPT (increased risk ratio (IRR)= 1.22 per IQR increase log-transformed PM_{10}, 95% 1.01-1.46), which was more strongly associated in girls. CPT and SAT response latency were not associated.	PH	PL	DL	DL	DL	DL
(Shier et al., 2019a)	N= 9550, Age 8-11years, 49% female, U.S.A	Modelled residential $PM_{2.5}$, PM_{10} and Ozone from kindergarten to 5th grade (outcome being the number of years where levels exceeded NAAQS standards)	Reading and maths tests in 3rd and 5th grade *Academic ability*	Asthma, exercise, school absences, SES, race-ethnicity, gender, age in months, birthweight, number of siblings, percentage of ethnic minority at school, urbanacity, census-level white population, census-level vacant houses, census-level home ownership, temperature, weather, school size, private/ public school, % students eligible for free lunch, region	Most Ozone and $PM_{2.5}$ associations significant. For example, having 2+ years of Ozone above standard (as opposed to 0) predicted 3rd grade maths, coef -2.45(0.96)* and 3rd grade reading -2.14(1.09)*. Having 2+ years of $PM_{2.5}$ above standard related to reading in third (-1.25(0.4), P<.01), reading in 5th (-1.94(0.67), p<.01), and maths in 5th (-1.37(0.57)*. PM_{10} not important.	PL	DL	DH	PL	DL	DL
(Suglia et al., 2008)	N=202, age 8-11 (mean age = 9.7±1.7), 52% female, Boston, USA	Spatiotemporal land-use regression model to predict 24-hour measures of traffic exposure, computing average lifetime	Cognitive battery including the Kaufman Brief Intelligence Test (K-BIT) and The Wide Range Assessment of Memory and Learning (WRAM-L) *General cognition / intelligence,*	age at cognitive assessment, gender, race/ethnicity, and maternal education (as a marker of socioeconomic status), ETS, birth weight, blood lead	An interquartile range (0.4 µg/m3) increase in residential black carbon exposure predicted decreased scores in children's K-BIT performance (mean difference): Vocabulary (-2.2, -5.5 to 1.1); Matrices (- 4.0, -7.6 to -0.5); Composite intelligence (-3.4, -6.6 to -0.3). Also associated with subscales on the WRAM-L (mean difference): Verbal (-1.3, -4.8 to 2.2); Visual (-5.4, -8.9 to -1.9); Learning (-2.8, -6.6	PH	DL	DH	PL	DL	DL

		black carbon exposure	*vocabulary, visual, learning*		to 1.1); General index (-3.9, -7.5 to - 0.3).						
(Suter et al., 2018)	N= 94, Age 5-12(6.65±1.11), 54.24% female, Kenya	Measured contemporaneous PAH metabolite (1-OHP) in urine and personally monitored mothers CO (proxy for child exposure)	Kaufman assessment battery for children (KABC-II) *General cognition*	Age at testing, household monthly rent, human immunodeficiency virus (HIV) status	In HIV uninfected and unexposed children, pollution was not related to neurocognition; in HIV positive children, high 1-OHP associated with lower global cognitive ability, delayed memory and attention.	PH	PL	PH	DL	DL	DL
(Vahedi, 2020a)	N=unclear (851-5302 in 1997), aged 3-15, USA	Measured ozone at nearest monitor to household location in 1997 (time of testing), environmental protection agency (EPA)	Letter-word (LW) test *Symbol learning and reading abilities*	Family income, family labour hours, active parents time with child, time spent studying alone	1 SD increase Ozone associated with a 0.07 SD reduction in LW test scores. Older age and lower household income mitigated effects.	DL	PH	nk	PL	PL	PH
(Van Kempen et al., 2012)	N=485 9-11 year olds (mean 10.5±0.6), 48.8% female, Amsterdam, The Netherlands	School and home addresses linked to modelled nitrogen dioxide (NO_2) and particulate matter (PM_{10})	Battery from the Neurobehavioural evaluation system (NES) *Reaction time, attention, task switching, hand-eye coordination, perceptual coding, digit span*	Age, sex, socio-economic status, longstanding illness (y/n), parental support, main language spoken at home is Dutch (y/n), and type of window glazing at home/school	NO_2 levels at school (9-11 year olds) associated with: digit memory span test (DMST) span (β = -0.16, -0.29 to -0.02); Simple Reaction Time test (SRT) (-1.18, -11.73 to 9.36); SAT block errors (-0.01, -0.22 to 0.20); SAT block RT (7.38, -8.52 to 23.28); SAT arrow errors (-0.16, -0.49 to 0.16); SAT arrow RT (11.42, -10.45 to 33.29); Switch errors (-0.63, -1.92 to 0.67); Switch RT (11.40, - 23.94 to 46.74);	DL	DL	PH	PL	DL	DL

					HECT (0.04, - 0.04 to 0.13); SDST (0.02, -0.4 to 0.16). NO2 levels at home associated with: DMST span (0.09, -0.04 to 0.22); SRT (-1.12, -11.11 to 8.86); SAT block errors (-0.02, -0.21 to 0.17); SAT block RT (8.40, -5.98 to 22.77); SAT arrow errors (-0.18, -0.50 to 0.14); SAT arrow RT (-1.76, -22.77 to 19.26); Switch errors (-0.65, -1.76 to 0.46); Switch RT (-10.71, -39.71 to 18.29); HECT (0.03, -0.04 to 0.11); SDST (-0.03, -0.14 to 0.08).						
(Xu et al., 2022)	N=1555,age 9-18 (mean 13.3 ±/ 2.3), 45.4% female, China,	Average value of $PM_{2.5}$ during pregnancy was modelled for Mother using MEIC-CMAQ model	Standardised cognitive ability module from the China Family Panel study (CFPS), a nationally representative survey *General cognitive function*	Age interview, gender, years of education, premature birth, low birth weight, material years of education, maternal age, per capita GDP, urban residence, household per capita income quantile	$PM_{2.5}$ exposure during pregnancy was associated with cognitive ability score of adolescents ($\beta = -0.040$, $p < 0.05$).	DL	DL	DH	PL	DL	PL
(Zhang et al., 2018b)	N= 31959, Age 10-69, 52.07% female, China	County-level Air Pollution Index (API) (calculated based on readings of PM_{10} SO_2 and NO_2) for 1d, 7d, 30d, 90d, 1year, 2year, 3 year (prior to testing)	Standardised cognitive ability module from the CFPS, a nationally representative survey *Mathematical and verbal ability*	Demographic characteristics (gender, age, household capita, years of education), weather conditions, county-level characteristics (GDP, population density, industrial value)	1 SD reduction in mean API on verbal test score across 30 days 0.599*, 3 years 1.132* (all lags but 1 day significant). 1SD reduction in mean API on math test score only significant after 90 days (0.146*, 1 year 0.146*, 2 years 0.157*, 3 years 0.211*.)	DL	PL	PH	PL	DL	PL

Table 1b: Characteristics of eligible mental health studies included in this systematic review

Author and year	Sample	Exposure	Outcome	Covariates considered	Outcomes-most adjusted model *= p<0.05 DL=definitely low PL=probably low PH=probably high DH=definitely high	Selection bias for comparison	Accounts for important confounding and modifying variables	Outcome data complete without attrition or exclusion?	Exposure characterisation	Outcome characterization	Were all measured outcomes reported? [3]
Ahmed, 2022	N=5471, Age 2-12, (mean 7.39±2.9). 47.66% female, Australia	Land-use regression (LUR) models used to predict annual mean concentrations NO_2 (ppb) and $PM_{2.5}$ (µg/m3), used to calculate exposure prenatally, for the first year, in years 1-4, and lifetime, separated into low, moderate, high	Strengths & Difficulties Questionnaire (SDQ) assessed total behavioural difficulties, completed by parents or teachers- score >17 (high/very high problems)	Child age at survey, sex, maternal education, maternal income, family composition	OR emotional and behavioural problems higher for those exposed to moderate and high levels of $PM_{2.5,}$ though only significant for moderate exposure first year (OR 1.30, 95% CI 1.05,1.60) or lifetime (OR 1.36, 95%CI 1.09,1.70). NO2 not associated or protective.	PL	PL	DH	PL	PL	PH

[3] PL: Probably Low
DL: Definitely Low
PH: Probably High
DH: Definitely High

		tertiles (0.5–6.1, 6.1–7.3, 7.3–14.3 µg/m3 PM2.5, 1.3–5.4, 5.4–7.9, and 7.9–19.0 ppb NO2)									
Alemany, 2018	N=1,589 children aged 7-11(8.5± 0.88), 46.7% female, Barcelona	PAHs, EC and NO_2 measured at school	SDQ by parents	age, sex, maternal education, smoking during pregnancy, smoking at home, residential neighbourhood socioeconomic status , air pollution at home based on LUR models	IQR increases in all three air pollutants associated with higher behaviour problem scores (significant for EC, Mean Ratio (MR) =1:06; 95% CI: 1.01, 1.11).	PL	PL	PH	DL	PL	PL
Bloemsma, 2022	Age 11: N=2624, 49.7% female. Age 14: N= 2506, 49.9% female. Age 17: N=2085, 51.5% female. Age 20: N=2189, 52.6% female. The Netherlands	LUR models used to estimate annual average concentrations of PM_{10}, $PM_{2.5}$, NO_2, $PM_{2.5}$ absorbance (marker of BC), and the oxidative potential of $PM_{2.5}$ (electron spin resonance (OPESR) and dithiothreitol (OPDTT)).	At ages 11, 14, 17 and 20 years, participants of the PIAMA study were requested to complete the MHI-5. Score ≤60 classified as poor mental wellbeing	Age, sex, parental education, smoking at home, season, neighbourhood SES	Higher exposure associated with higher odds of poor mental wellbeing. For example, OR 1.23 (95% CI 1.08–1.40) per 0.28 nmol DTT/min/m3 increase in OPDTT and OR 1.23 (95% CI 1.09–1.38) per 9.11 µg/m3 increase in NO_2. All pollutants were significant predictors at age 20, none at age 11 (NO_2 sig at 14, $PM_{2.5}$, NO_2, OPESR age 17).	PL	PL	PL	PL	DL	PL
Brokamp, 2019	N=13,812, 0-18 year olds, median age 14.4 (25^{th} perc 11.7,	Spatiotemporal random forest model used to produce daily ambient concentrations of $PM_{2.5}$ at residential address	Paediatric psychiatric emergency department (ED) utilisation	Daily temperature, humidity, holidays,	a 10-lg/m3 increase in $PM_{2.5}$ and any psychiatric ED visit at 1 d (Odds Ratio (OR)= 1.07 (95% CI: 1.02, 1.12)) and 2 d later (OR= 1.05 (95% CI: 1.00, 1.10))	DL	PH	DL	PL	DL	DL

	75th perc 16.1), 50% female, USA	(24hr averages), as well as 1,2, and 3 day lags			(same day and 3 days ns). Stronger for those in high-deprivation communities.						
Brunst, 2019	145 adolescents aged 12, 41.4% female, Cincinnati	LUR estimated ECAT at addresses at birth, for 12 months prior to assessment, and average childhood exposure	Spence Children's Anxiety Scale	race, maternal age, birth weight, duration of breastfeeding, mothers education, household income, caregiver depression, caregiver relational frustration, serum cotinine levels, use of depression and /or anxiety medications	Where recent ECAT exposure was high (compared to low) significantly higher generalised anxiety symptoms (beta = 4.71; 95% CI 0.95, 8.45) with the same direction of effect for anxiety subscales	PH	PL	DH	PL	DL	DL
Casas, 2017	21,231 suicides (population of Belgium 10,295,724); 71 events by children age 5-14, 482 by adolescents age 15-19	Daily average PM_{10} and O_3 by municipality (event days vs matched control days)	Suicides over 10 years, stratified by age group (5-14, 15-19, 20-64, 64-84, 85+)	average temperature, duration of sunshine, day of week, seasons, age, sex	10 $\mu g/m^3$ increase in PM_{10} significantly associated with a 45% increase in odds of children's suicide (95% CI 1.03–2.04). Among adolescents, a 10 $\mu g/m^3$ increase in O_3 in previous 0–1 days associated with an 8% increased odds of suicide (95% CI: 1–16%)	DL	PL	DL	PL	DL	DL
Dales, 2016	1,883 aged 6-17, Canada	Closest air monitors to examination site day of testing- 8hr average O_3 and 24 hour average $PM_{2.5}$ and NO_2	Would you describe yourself as happy; Do you have a mood disorder; parent responded emotional difficulties scale of SDQ	None in air pollution mental health associations (compared mean air pollution values for	Mean O_3 concentrations 2ppb greater in those with some vs no SDQ symptoms (significant), otherwise air pollution and mental health outcomes unrelated but mood disorder exacerbated	DL	DH	PL	PL	PH	PL

				presence/absence of difficulties groups). In models of physical health and air pollution, stratified by mental health: age, gender, bmi, ethnicity, household education, household income, smoking status, household smoking, alcohol consumption, mean day temperature, relative humidity	the negative association between air pollution and general health					
Fan, 2019	N=21,780, 15.4 (1.9), 52.7% female, China	Annual average $PM_{2.5}$ concentration at school – satellite data based LUR estimates	Suicide attempts (self-reported)	age, sex, nationality, living arrangement, household SES, classmate relations, lifetime drinking, lifetime smoking, sleep disturbance (PSQI), depressive symptoms	Ambient $PM_{2.5}$ associated with suicide attempts after adjusting for covariates and depressive symptoms, (Adjusted Odds Ratio (aOR)=1.25, 95% CI=1.03–1.56). It also significantly interacted with sleep disturbance.	DL	PL	DL	PL	DH	DL
Jorcano, 2019	N=8494, aged 7-11, 41.3-52% female, Europe (The Netherlands, Germany, Poland, France, Spain)	LUR models – prenatal annual concentrations NOx, PM_{10}, $PM_{2.5}$, $PM_{2.5abs}$ (EC proxy), residential addresses	Parent reported Child behaviour Checklist(CBCL) (aged 7-10) or SDQ (age 7-11)-anxious/depressed and aggressive problems scales	parental age, parental cohabitation, parental education, tobacco in pregnancy, parity, maternal BMI, child sex, child age	In gen R study, 10μg/m^3 higher prenatal NO_2 associated with OR 1.15 [95%CI 1.01 to 1.30] for depression-anxiety symptoms and In EDEN study 10μg/m3 higher prenatal NO_2 OR 3.04 (95%CI: 1.56, 16.25) for aggressive symptoms. However, overall effects ns. Postnatal exposure ns (overall direction of l*ower* symptoms with higher exposure).	DL	PL	PH	PL	PL	DL

Karamanos, 2021	N= 4775 adolescents age 11-16, UK	KCL-urban modelled annual mean NO_2, $PM_{2.5}$, PM_{10} and ozone at residential address	Conduct problems subscale of SDQ	Age, racism, sex, ethnicity, maternal mental health, parental care, parental control, cigarette smoking, alcohol, family affluence, Index of Multiple Deprivation (IMD) income domain	$PM_{2.5}$ was significantly associated with conduct problems – 0.11 (− 0.17 to – 0.05, $p<0.001$), as was NO_2 – 0.02 (− 0.03 to 0.00, $p<0.05$). $PM_{2.5}$ and racism significantly interacted to exacerbate conduct problems. In a two-pollutant model, NO_2 no longer associated.	PL	DL	PL	PL	PL	PH
Kim, 2019	N=67,561 15+ year olds (out of 8,734,321 citizens), 49.3% female, South Korean	Hourly measured $PM_{2.5}$, $PM_{10,}$ CO, NO_2, SO_2, and O_3 in Seoul, from Korean National Institute of Environmental Research	Patients with psychiatric emergencies, Emergency department visits with psychiatric disease codes (F00-F99 in ICD10 codes), the National Emergency Department Information System (NEDIS) of the National Emergency Medical Center (NEMC)	Daily mean temperature, relative humidity, mean air pressure, day of the week	$PM_{2.5}$ not associated in single-pollutant models. The absolute risk reduction (ARR) (95%CI) in the model adjusted for SO_2 was 1.011 (1.002–1.021) by 10 μg/m3 of $PM_{2.5}$ on Lag 1 for all psychiatric diseases. The ARR (95% CI) in the model adjusted for O_3 was 1.015 (1.003–1.029) by 10 μg/m3 of $PM_{2.5}$ on Lag 1 for Neurotic, stress-related and somatoform disorders. SO_2, CO, ozone, NO_2 not related.	DL	PL	DL	DL	DL	DL
Latham, 2021	N=2066, age 18, 51% female, UK	CMAQ-urban used to model residential address exposure to NO_2, NOx, $PM_{2.5}$, PM_{10}, in 2004 (age 10), evaluated in quartiles	Diagnostic interview schedule to identify clinically significant depression (major depressive disorder) at 18	Non-independence twin observations, sex, neighbourhood, SES, smoking status, family SES, family psychiatric history, age 10 depressive symptoms	Trends observed for highest quartile NO_2 (OR 1.43, 95% CI: 0.96-2.13) and $PM_{2.5}$ (1.35, 0.95-1.92) (and unadjusted associations significant) .	DH	DL	PL	PL	DL	PL
Liu, 2019	N=158,634 (11727 under	hourly $PM_{2.5}$ concentration was recorded by the ambient	Emergency Ambulance dispatch for psychiatric emergencies (EPE),	Calendar time, day of week, public holidays, weather conditions	10μg/m3 increase $PM_{2.5}$ associated with 0.12% increase of the same day	DL	PL	DL	DL	DL	DL

	18s), 40.2% female, China	air quality monitoring station located at the U.S. Embassy building, used to calculate 24 hour averages	Beijing Emergency Medical Center and Beijing Red Cross Emergency Center from January 1, 2008 to December 31, 2014		overall EPE (95% CI: 0.03–0.22%, p=0.013) and a 0.12% increase of the suicide-related EPE at lag 2 (95%CI: 0.01–0.24%, p=0.041). Children (age<18) showed a higher risk of suicide-related EPE after $PM_{2.5}$ exposure compared to adults (18≤age≤64)						
Liu, 2018	54,923 students, 48.08% female, mean age 16.2(8.8- 24.6), China	Inverse-distance weighted air monitoring station measurements averaged school exposure for year prior to event for $PM_{2.5}$, CO, NO_2, O_3 and SO_2	Function of Self-Mutilation measure (Non Suicidal Self Injury (NSSI) behaviours over last 12 months)	gender, household income, mothers education, maltreatment, pocket money, school level	10 µg/m3 $PM_{2.5}$ & O3 associated 13.9% & 10.5% NSSI odds increase. 0.1 µg/m3 increase CO = 5.1% higher odds. O_3 and CO significantly associated with NSSI in middle and high school students but not college. Exposure-response relationships shown for CO and O_3	DL	PL	PL	PL	PH	PL
Manczak, 2022	N=213, baseline 9-13 years, N=124 at T3 age 13-17, 57% female, USA	Daily maximum eight-hour average concentrations of ozone from air monitoring sites and inverse distance weighting used to estimate ozone exposure for the center of each census tract of residence	Self-reported questionnaire at 3 timepoints, Children's Depression Inventory-Short Form (CDI-S) and Youth Self Report (the Withdrawn/Depressed and Anxious/Depressed subscales)	Age, sex, ethnic minority status, severe life stressors, household income, parental education, neighbourhood disadvantage	ozone was not significantly related to T1 symptoms but ozone predicted steeper increases in depressive symptoms across adolescent development, but not other psychopathology symptoms: CDI-S score $ß_{slope(time)}$ = 0.18, p<0.001. Withdrawn depressed: 0.13, p=0.004	PH	PL	DH	PL	DL	PL
Margolis, 2016	462 assessed age 3-5, 7, 9 and 11, 53.46% female, U.S.A	PAH-DNA adducts in maternal blood	Mother reported CBCL; anxious/depressed + aggression, + impulsivity scores = deficient	Gender, ethnicity, maternal education, marital status, maternal	All subscales significantly associated with PAH. Significant time-PAH interaction: without	DH	PL	DH	DL	PL	DL

			emotional self-regulation (DESR) score	mental health, neighbourhood SES	detectable PAH- DNA adducts DESR score decreased by 0.38% per year, with detectable adducts 0.09%.						
Mueller, 2019	3683 10-15 year olds (mean age 12.24(SE= 1.73), 50% female, England and Wales	Bayesian spatio-temporal modelling of outdoor neighbourhood annual NO_2 from 2009-2011	Self-completed SDQ 2011-2013	gender, age, education of mother, ethnicity, greenspace, neighbourhood deprivation, urbanicity, neighbourhood noise and cohesion as reported by mothers, adolescent perceptions of neighbourhood (liking, safety)	Air Pollution predicted hyperactivity and inattention but not in fully adjusted model. Subjective perceptions of neighbourhood (safety, fear of crime, liking the area) were most important for mental health	PL	DL	DH	PL	PL	DL
Newbury, 2019	2063, 52.5% female, assessed at 18, England and Wales	CMAQ-urban model of NO_2, NOx, $PM_{2.5}$, and PM_{10} for 2012 (age 17)-based on residential + two most frequented addresses	Interviewed at 18 about 13 psychotic experiences since age 12-hallucinations, delusions, and unusual experiences found in prodrome	Urbanicity, family and neighbourhood SES , family psychiatric history, maternal psychosis, childhood psychotic symptoms, adolescent smoking, cannabis and alcohol dependence, neighbourhood crime, social conditions	Fully adjusted models remained significant for NOx (OR of 1.72 (95%CI, 1.30-2.29)), NO_2 (OR 1.71 (95% CI, 1.28- 2.28)) and $PM_{2.5}$ (OR 1.45 (95% CI, 1.11- 1.90)). 2 pollutant model implied associations of $PM_{2.5}$ driven by NOx/NO_2.	DH	PL	DL	PL	DL	DL
Oudin, 2016	552,221 people <18 registered between 2007-2010 in Stolkholm, Vastra Gotaland, Skane, or Vasterbotten	LUR annual means of NO_2, $PM_{2.5}$, and PM_{10} for year individual was included in the study (2005- 2010)	Number of dispensed medications category NO5 (neuroleptics, ataractics and sleeping pills – not antidepressants or ADHD medications).	Age at start of follow-up, sex, parental education, maternal BMI in pregnancy, maternal prenatal smoking, neighbourhood education level, family income, county	Very similar results for PM_{10} and $PM_{2.5}$ so they only present the former. Main model; Hazard Ratio (HR)=1.09, (95% CI 1.06-1.12,) associated to a 10 µg/m3 increase in NO_2. For PM_{10}, 1.04 (95% CI 1-1.08). Further adjustment did not change	DL	PL	DL	PL	PH	PL

					substantially, with some regional variation.						
Reuben, 2021	N=2039, age 18, 52.5% female, England and Wales	Long-term exposure (ag 10-18) estimated for NOx (NO_2 + NO) and $PM_{2.5}$ using CMAQ-urban	Interviewed for mental health symptoms age 18, factor analysis used to produce general psychopathology, internalising, externalising, and thought disorder scales	Sex, family, SES, familial psychiatric history, participant history of emotional and behavioural factors, tobacco smoking.	1IQR increase in NOx exposure associated with a 1.40-point increase (95% CI, 0.41-2.38; P = .005) general psychopathology. No association between continuous $PM_{2.5}$ and general psychopathology (b = 0.45; 95% CI, −0.26 to 1.11; P = .22); however highest quartile of $PM_{2.5}$ scored 2.04 points higher (95% CI, 0.36-3.72; P = .02) than bottom 3 quartiles. Copollutant models suggested Nox driving these associations. NOx exposure associated with all outcomes, although weakest for internalizing (adjusted b = 1.07; 95% CI, 0.10-2.04; P = .03), medium for externalizing (adjusted b = 1.42; 95% CI, 0.53-2.31; P = .002), and strongest for thought disorder symptoms (adjusted b = 1.54; 95% CI, 0.50-2.57; P = .004)	DH	PL	DL	PL	DL	DL
Roberts, 2019	284, 46% female, mean age 12, England	KCLurban model estimated many pollutants, $PM_{2.5}$ and NO_2 elected as focus	Age 12: self- reported Children's Depression Inventory and Multidimensional Anxiety Scale for children, conduct disorder symptoms. Age 18: self-	Sex, ethnicity, neighbourhood SES, family SES, family psychiatric history, severe victimisation exposure, participants' smoking	NO_2 and $PM_{2.5}$ significantly associated to a diagnoses of MDD at 18 even when controlling for further covariates- OR of 1.57(1.05-2.35, p= 0.029) and 1.63(1.08-2.46, p	DH	PL	PL	PL	DL	DL

			report symptoms of Major Depressive Disorder(MDD), Generalised additive disorder (GAD), Conduct disorder		=0.021) but not anxiety, conduct disorder, or ADHD. Concurrent exposure age 12 not associated with mental health						
Shen, 2021	N=20,079, Age 18.3 ± 0.8, 48.9% female, China	Modelled mean residential BC concentration during 6 years prior to assessment, adjusted for breath rate	Patient Health Questionnaire-9 (PHQ-9) and Generalised Anxiety Disorder Assessment (GAD-7); self-reported symptoms of depression and anxiety in past 2 weeks	Age, sex, family income, parental education, ETS exposure, alcohol consumption, physical activity, BMI, principal components environmental factors (humidity, temperature, UV, artificial night-time light, other pollutants)	long-term BC exposure significantly associated with symptoms of depression ($\beta = 0.17$, $P < 0.001$) but not anxiety ($\beta = 0.07$, $P = 0.125$). Driven by women and stronger in those with less education	PH	DL	DL	PL	PL	PL
Szyszkowicz, 2020	N=83,985, 51% female, Canada	Hourly recorded concentrations of NO_2, ozone, $PM_{2.5}$, averaged to daily concentrations at lags from 0-5 days to event	Visiting emergency departments for mental disorders	Temperature, time-trends, weekday, month, year	same day $PM_{2.5}$, for all (IQR = 6.03 µg/m3), Risk Ratio (RR) = 1.01 (95% CI: 1.00–1.02) and female-only patients RR = 1.02 (1.00–1.03). 1-day lag in males: and $PM_{2.5}$ RR = 1.02 (1.01–1.03), for NO_2 (IQR = 9.1 ppb) RR = 1.02 (1.00–1.04), and ozone (IQR = 16.0 ppb) RR = 1.06 (1.01–1.10) for males. Strongest in 13-18 year old age group and for $PM_{2.5}$	DL	PL	DL	DL	PL	DL
Thilakaratne, 2020	720,012 visits for CO data/ 69,192 visits for NO2 data, 10% 0-18 years old, 46% female, USA	Daily concentrations of CO, NO_2 from US EPA, nearest monitor	daily counts of mental health-related ED visits	Apparent temperature (temperature + humidity), seasonal/long-term trends, holidays, day of the week	For children 0–18 years old, an IQR increase in two-day average CO was associated with an 8.68% (95% CI: 5.15, 12.33%) increase in risk ($p < 0.001$ compared to 19–34 years	DL	PL	PL	PL	PL	DL

					old), and an IQR increase in two-day average NO_2 was associated with a 6.73% (95% CI: 4.25, 9.27%) increase in risk ($p < 0.001$ compared to 19–34 years old).						
Yolton, 2019	N=344, mean age 12.2, 44.5% female, U.S.A	LUR estimated TRAP and ECAT at addresses at birth, for 12 months prior to assessment, and average childhood exposure	Parent completed Behaviour Assessment System for Children (BASC-2). Children completed Spence Children's anxiety scale (SCAS) and children's depression inventory-2.	Parent BDI, parenting relationship questionnaire, lead exposure, tobacco smoke, age at delivery, maternal education, parental marital status, depression, household income, community level deprivation, social support, child age at assessment, sex, race, early childhood cotinine levels	0.25 μg/m3 increase in in prenatal/early life ECAT significantly associated with 1.90 point increase (95% CI 0.29, 3.51) in SCAS total anxiety and 1.64 increase (95% CI 0.33, 2.95) in generalized anxiety. Current ECAT exposure associated with generalized anxiety (β=1.90, 95% CI 0.37, 3.43) and social phobia (β=1.88, 95% CI 0.06, 3.70). 0.25 μg/m3 increase in averaged childhood exposure to ECAT associated with 2.15 increase (95% CI 0.35, 3.95) in generalized anxiety and 2.26 increase (95% CI 0.10, 4.42) in social phobia. Per 0.25 μg/m3 increase in ECAT prenatal/early life ECAT significantly associated with children's depression (β=3.01, 95% CI 1.03, 4.99).	DH	DL	PH	PL	PL	DL

Yorifuji, 2017	N=46,039, assessed yearly from 0- 14 years old, Japan	Municipality level Suspended Particulate Matter (SPM), NO_2, SO_2, and CO prenatally (measured and averaged for each area)	Parent reported CBCL behavioural problems at 8 years	Birth weight, gestational age, twin status, sex, parity, parental age, birth month, maternal smoking, maternal education, paternal income, municipality type, municipality population density, per capita income	Each air pollutant, in particular SPM and SO_2 exposure, was positively associated with unfavourable behavioural problems (e.g. 1 IQR increase in SPM adjusted ORs 1.06 (95% CI: 1.01, 1.11) for lying, and 1.07 (95% CI: 1.02, 1.13) for causing disturbances in public.)	DL	DL	DL	PL	PL	PH
Zhao, 2019	N=2,827, 15.2 (+-0.3) 50.7% female, Germany	LUR estimated long term ozone, and ozone 0, 1, 2, 3 and 7 days prior to assessment.	At 15 years self-completed Depression Screener for Teenagers (DesTeen). DesTeen	cohort, exact age, sex, parental education, maternal age at birth, household income, single parent family, maternal smoking in pregnancy, second hand exposure at home, time spent in front of a screen, time spent outside, parental psychopathology	No significant long term effects. Found protective association, withstanding confounder adjustments, for Wesel at 0 days (OR=0.76, 95% CI: (0.59, 0.98), p=0.037), especially for males	PH	PL	DH	PL	PL	PL
Zhao, 2020	N=1,126,014, Age 16+ 54.61% female, Germany	Ozone and PM_{10} modelled per quarter per district, provided by the German Environment Agency	Any F32 or F33 diagnosis from a physician was defined as a depression case, any F40 or F41 diagnosis was defined as anxiety case, converted to cases per quarter	Season, access to healthcare, sex, age	In two-pollutant model, 10 more days with a maximum 8-h average ozone concentration exceeding 120 $\mu g/m^3$ resulted in a relative risk (RR) of 1.010 with 95% confidence interval (CI) (1.005, 1.014) for depression and an RR of 1.007 (95% CI (1.000, 1.014)) for anxiety. Effects of PM_{10} for depression and anxiety were 1.180 (95%	PL	PH	DL	PL	PL	DL

					CI (1.160, 1.201)) and 1.176 (95% CI (1.148, 1.205)) per 10 μg/m³ increase in PM_{10} concentration						

AP=air pollution, MH= mental health, SES= socio-economic status, LUR=land-use regression model, IQR=Interquartile range, ETS= environmental tobacco smoke, MDD= major depressive disorder, GAD= generalised anxiety disorder, DESR= deficient emotional self-regulation, OR= odds ratio, aOR=adjusted odds ratio, NSSI= non- suicidal self-injury, SDQ= strengths and difficulties questionnaire, CBCL= Child Behaviour Checklist, BASC-2= behaviour assessment system for children, BDI= Beck's depression inventory, MRI= magnetic resonance imaging, MR= mean ratio, aMR= adjusted mean ratio, CI= confidence interval, HR= hazard ratio, $\mu g/m^3$ = microgram per cubic metre

Table 2a: GRADE assessed certainty of collective evidence for cognitive associations investigated in the literature

Exposure	**Outcome**	**Papers**	**N papers**	**Direction of evidence**	**Start rating**	**Downgrade?**	**Upgrade?**	**Finish**
$PM_{2.5}$	Attention/EF	Forns et al., 2017 Gui et al., 2020; Julvez et al., 2021; Saenen et al., 2016	4	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	No	Yes Reason: Large effect	High
PM10	Attention/EF	Gui et al., 2020; Saenen et al., 2016; Sears et al., 2020	3	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	No	No	Moderate
PM2.5	General intelligence/IQ	Jalaludin et al. 2020; Julvez et al., 2021; Russ et al., 2021; Xu, 2022	4	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	No	No	Moderate
BC	Processing speed/reaction time	Chiu 2013; Saenen, 2016	2	Unsupportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: imprecision	No	Low
Composite	Academic ability	Clark-Reyna, 2016a b and c (same people), Grineski, 2016	5 (2 populations)	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: imprecision	No	Low
Ozone	Language	Shier et al., 2019; Vahedi, 2020	2	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: Risk of bias concerns and imprecision	No	Low

PAH	Attention/EF	Margolis, 2021; Suter et al, 2018; Alemany, 2018	3	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: imprecision	No	Low
EC/BC	Attention/EF	Alemany, 2018; Chiu et al., 2013; Forns et al., 2017; Saenen et al., 2016	4	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: Imprecision and inconsistency	Yes Reason: Large effect	Low
NO2	Attention/EF	Alemany, 2018; Forns et al., 2017; Gui et al., 2020; Julvez et al., 2021; Van Kempen, 2012	5	Unsupportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: Inconsistency	No	Low

Table 2b: GRADE assessed certainty of collective evidence for mental health associations investigated in the literature

Exposure	**Outcome**	**Papers**	**N papers**	**Direction of evidence**	**Start rating**	**Downgrade?**	**Upgrade?**	**Finish**
PM2.5	Psychiatric emergency	Brokamp, 2019; Kim, 2019; Liu, 2019; Szyszkowicz, 2020	4	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	No	Yes Reason: Large effect	High
Nox	Externalising/ conduct symptoms	Reuben, 2021; Roberts, 2019; Jorcano, 2019; Karamanos, 2021; Mueller, 2019	5	No	Moderate Reason: Longitudinal evidence, individual level data, comparison used	No	No	Moderate
PM2.5	Internalising /emotional symptoms	Reuben, 2021; Jorcano, 2019; Dales, 2016	3	No	Moderate Reason: Longitudinal evidence, individual level data, comparison used	No	No	Moderate
Nox	General psychopathology/ general difficulties	Reuben, 2021; Ahmed, 2022; Alemany, 2018	3	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency	No	Low

PM2.5	General psychopathology/ general difficulties	Ahmed, 2022 Reuben, 2021	2	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: imprecision	No	Low
PAHs	General psychopathology/ general difficulties	Alemany, 2018; Margolis, 2016	2	yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: imprecision	No	Low
PM2.5	Externalising/ conduct symptoms	Reuben, 2021; Jorcano, 2019; Karamanos, 2021; Roberts, 2019	4	No	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency	No	Low
NOx	Internalising /emotional symptoms	Reuben, 2021; Dales, 2016; Jorcano, 2019; Mueller, 2019	4	No	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency	No	Low
NO2	Happiness yes no /Mental wellbeing	Dales, 2016; Bloemsma, 2022	2	50/50	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and imprecision	No	Low
PM2.5	Happiness yes no/ Mental Wellbeing	Dales, 2016; Bloemsma, 2022	2	50/50	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and imprecision	No	Low
NO2	Psychiatric emergency	Kim, 2019; Szyszkowicz, 2020; Thilakaratne, 2020	3	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency	No	Low
CO	Psychiatric emergency	Kim, 2019; Thilakaratne, 2020	2	50/50	Moderate	Yes Reason: inconsistency	No	Low

					Reason: Longitudinal evidence, individual level data, comparison used	and imprecision		
Ozone	Psychiatric emergency	Kim, 2019; Szyszkowicz, 2020	2	No	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and imprecision	No	Low
ozone	Anxiety	Manczak, 2022; Zhao, 2020	2	50/50	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and imprecision	No	Low
EC/BC/ECAT	Anxiety	Brunst, 2019; Shen, 2021; Yolton, 2019	3	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and risk of bias concerns	No	Low
BC/ECAT	Depression symptoms	Shen, 2021; Yolton, 2019	2	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and imprecision	No	Low
NO2	MDD / Mood disorder yes no	Latham, 2021; Roberts, 2019; Dales, 2016	3	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and imprecision	No	Low
PM2.5	MDD/Mood disorder yes no	Latham, 2021; Roberts, 2019; Dales, 2016	3	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and imprecision	No	Low
PM10	MDD	Latham, 2021, Zhao, 2020	2	50/50	Moderate	Yes Reason: inconsistency	No	Low

					Reason: Longitudinal evidence, individual level data, comparison used	and imprecision		
ozone	MDD/depression symptoms/ mood disorder	Zhao, 2020; Manczak, 2022; Zhao, 2019; Dales, 2016	4	50/50	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and risk of bias concerns	No	Low
PM2.5	Psychosis /Thought disorder	Newbury, 2019; Reuben, 2021	2	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency and imprecision	No	Low
NO2/Nox	Psychosis/ Thought disorder	Newbury, 2019; Reuben, 2021	2	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: imprecision	No	Low
PM2.5	Suicide /suicide attempt	Fan, 2019; Liu, 2019	2	Yes	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: imprecision	No	Low

2.3.1 Cognitive outcomes

2.3.1.1 General cognition and IQ

Six studies were identified evaluating the relationship between pollutants and general cognition or intelligence (see Table 1a). There was moderate certainty of evidence in support of a relationship between $PM_{2.5}$ and worsened adolescent general cognition/ intelligence (Jalaludin et al., 2022, Julvez et al., 2021a, Russ et al., 2020, Xu et al., 2022). One study evaluated its relationship to each of NO_2, EC/BC, and PAH's, respectively (Julvez et al., 2021a, Suglia et al., 2008, Suter et al., 2018), which were not convincingly supportive of associations. There was vast heterogeneity in outcome metrics (K-BIT, WRAMA-L, Ravens progressive matrices). Four of these studies evaluated younger adolescents (≤13), one study evaluated 10-15 year-olds, and one study evaluated 10-70 year-olds (Russ et al., 2020), so this study included but was not specific to adolescents.

2.3.1.2 Academic performance

In High schools in Texas, several studies reported pollutant composites to be associated with worse grade point average (GPA); specifically metabolic disruptors, respiratory risk indexes, and particulate matter diesel risk indexes (Clark-Reyna et al., 2016a, Clark-Reyna et al., 2016b, Clark-Reyna et al., 2016c, Grineski et al., 2016). Amanzadeh and colleagues reported that high school students' contemporaneous exposure to PM_{10} and $PM_{2.5}$ was associated with lower test scores, but not CO, NO_X, SO_2, or ozone (Amanzadeh et al., 2020) and another study reported that CO_2 was assocaited with lower academic ability in Greek 11-12 year-olds (Dorizas et al., 2015). This literature supports an association between air pollution composite metrics and academic abilities with moderate certainty, but is inconclusive for other exposures which were either not investigated or insufficiently investigated. Academic performance is a more functional outcome than performance on a standardised cognitive test,

and combines multiple cognitive domains in an applied way, so this may be particularly concerning but also subject to greater risk of confounding. The evidence here was weighted towards older adolescents (14+ years).

2.3.1.3 Language abilities (verbal, reading and writing)

Five studies included in this review evaluated language abilities and air pollution exposure in adolescents (see Table 1a). Overall there was low certainty evidence from two studies that ozone exposure is associated with adolescent language abilities (Shier et al., 2019b, Vahedi, 2020b). Both of these samples included children, with samples aged 3-15 and 8-11 years, respectively. Other associations were only investigated by one project so their evidence could not be collectively assessed, but there was evidence for detrimental associations between adolescent language ability and $PM_{2.5}$ (Shier et al., 2019b) and air pollution composites (Zhang, 2018), though the latter sample reflected a wider population that included adolescents (10-69 years) rather than focussing on them. Trend associations were observed between both EC/BC and PAHs and worsened academic ability (Margolis et al., 2021, Suglia et al., 2008) in 6-13 and 10-15 year-olds, and Shier and colleagues did not find evidence for associations with PM_{10} (Shier et al., 2019b).

2.3.1.4 Mathematical ability

Three studies evaluated mathematical skills and air pollution; reporting detrimental associations with $PM_{2.5}$ and ozone exposure (Shier et al., 2019b), trend detrimental effects with air pollution composites (Zhang et al, 2018) and PAHs (Margolis et al., 2021), and no association with PM_{10} (Shier et al., 2010b). There was not sufficient evidence for any given association to evaluate its collective certainty, and although these were good quality studies

only Margolis had an adolescent focus (10-15 year-olds, as opposed to 10-69 or 8-11 year-olds).

2.3.1.5 Executive function, attention, and working memory

This was the most investigated set of outcomes in the literature with 12 studies (see Table 1a). Four studies provided high quality evidence for an association between $PM_{2.5}$ and worsened attentional outcomes (Forns et al., 2017, Gui et al., 2020b, Julvez et al., 2021a, Saenen et al., 2016), and three studies provided moderate quality evidence for PM_{10} (Gui et al., 2020b, Saenen et al., 2016, Sears et al., 2020). However, the studies only included younger adolescents (up to 12 for $PM_{2.5}$ and up to 14 for PM_{10}). There was also evidence assessed as low quality for detrimental associations with PAHs (Alemany et al., 2018b, Margolis et al., 2021, Suter et al., 2018), and EC/BC (Alemany et al., 2018b, Chiu et al., 2013b, Forns et al., 2017, Suter et al., 2018). The evidence for NO_2 was generally unsupportive of an association but low quality due to inconsistencies between studies (Alemany et al., 2018b, Forns et al., 2017, Gui et al., 2020b, Julvez et al., 2021a, Van Kempen et al., 2012). Further studies found evidence for detrimental associations with SO_2 and ozone (Gui et al., 2020b), UFP (Forns et al., 2017), and benzene (Kicinski et al., 2015b, Kicinski et al., 2016), but these were not numerous enough to collectively assess the certainty of evidence for these exposures. These studies generally focussed on younger adolescents.

2.3.1.6 Memory and learning

Three studies evaluated memory and learning outcomes (see Table 1a). There was supportive evidence for detrimental associations with NO_2 and $PM_{2.5}$ (Powdthavee and Oswald, 2020a), a trend detrimental association observed with BC (Suglia et al., 2008) and a detrimental

effect observed in HIV-positive participants only (Suter et al., 2018). Kicinski and colleagues (2015) did not find evidence for associations with an air pollution composite.

2.3.1.7 Reaction time, processing speed, psychomotor function, and visuospatial skills

Two longitudinal studies did not find evidence of an association between reaction times and BC (Chiu et al., 2013; Saenen et al., 2016), which was assessed as evidence of low certainty against an association. However, Saenen and colleagues (2016) did report associations with $PM_{2.5}$ and PM_{10}. Kicinski and colleagues (2015) did not find associations between a benzene /traffic exposure composite and motor skills.

Based on the obtained children's literature, which was larger (76 studies were identified in the search involving 0-10 year-olds), two meta-analyses of psychomotor development and prenatal air pollution exposure were conducted. Early psychomotor development is likely to inform the trajectory of later development so given the lack of available data in adolescents to meta-analyse, this analysis was conducted. The pooled beta-coefficient from 3 studies (figure 3) evaluating the relationship between increasing prenatal NO_2 (1µg/m^3) and early psychomotor development (6 months-6 years) showed a negative but non-significant trend (figure 3; β = -0.08, 95% CI: -0.16, 0.01, p=0.08). However, an increase of 1µg/m^3 prenatal $PM_{2.5}$ was associated with significant reductions in psychomotor development score (figure 4; β = -1.54, 95% CI: -2.91, -0.17, p=0.03), meaning a 1µg/m^3 increase in each pollutant corresponded to a -0.08 and -1.54 reduction in psychomotor development score, respectively. Heterogeneity was high for the latter effect (I^2 = 90% p<0.01).

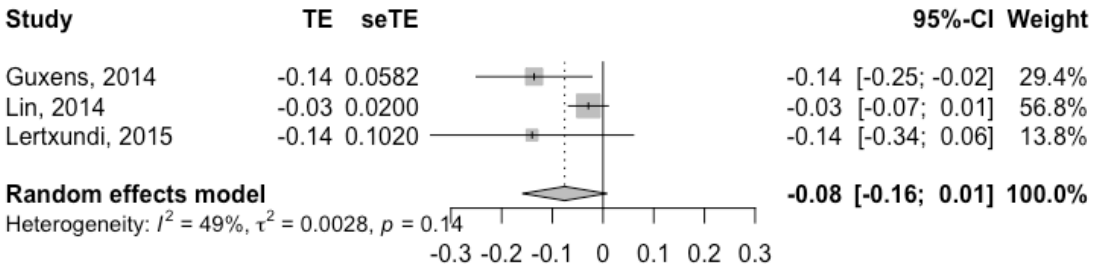


Figure 3. Pooled beta coefficient of the relationship between 1µg/m^3 increase prenatal NO_2 exposure and early psychomotor development. Note, TE= beta coefficient, seTE= standard error of beta coefficient.

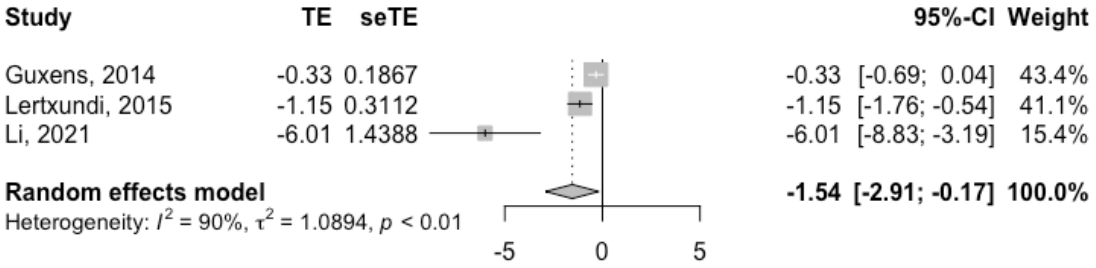


Figure 4. Pooled beta coefficient of the relationship between 1µg/m^3 increase prenatal $PM_{2.5}$ exposure and early psychomotor development. Note, TE= beta coefficient, seTE= standard error of beta coefficient.

2.3.2 Mental health outcomes

2.3.2.1 General psychopathology and mental health

Five studies evaluated the effects of general difficulties or a range of psychopathology symptoms, however these were all in high-income countries (see Table 1b). Three studies (Ahmed et al., 2022; Alemany et al., 2018; Reuben et al., 2021) evaluated the effects of Nox (NO and/or NO_2), two studies evaluated $PM_{2.5}$ (Ahmed et al., 2022; Reuben et al., 2021) and two studies evaluated PAHs (Alemany et al., 2018; Margolis et al., 2016) which all tended towards being supportive of a detrimental association but with low certainty. These studies

included either populations younger than 12 or aged 18 years, and did not evaluate middle adolescence. Oudin and colleagues (2016) evaluated the relationship between prescription of psychiatric medications to under 18s and exposure to NO_2, $PM_{2.5}$, and PM_{10}, which were all associated.

2.3.2.2 Mental wellbeing and happiness

Two studies examined associations between wellbeing or happiness across adolescence and air pollutants (NO_2 and $PM_{2.5}$) but with mixed results and low certainty evidence (Bloemsma et al., 2022; Dales et al., 2019).

2.3.2.3 Psychiatric emergencies

Five studies evaluated the associations between emergency department (ED) visits or emergency ambulance dispatch and air pollution (Kim et al., 2019; Brokamp et al., 2019; Liu et al., 2019; Szyszkowicz et al., 2020; Thilakaratne et al., 2020), providing high quality evidence in support of a detrimental association with $PM_{2.5}$, and low-quality evidence in support of a detrimental association with NO_2. Associations with CO and ozone were not supported by low certainty evidence. These studies were not adolescent specific, though their ranges (0-18, 15+ years, or all ages) included adolescence. Syzszkowicz et al. (2020) observed associations between ED psychiatric visits and $PM_{2.5}$ were strongest in the 13-18 year-old group.

2.3.2.4 Anxiety, depression, internalising difficulties, and mood disorder

Eleven studies evaluated associations of air pollution and anxiety, depression, internalising problems, or mood disorder (see Table 1b). Evidence of moderate certainty suggested a lack of an association between internalising symptoms and $PM_{2.5}$ (Reuben et al., 2021; Jorcano et

al., 2019; Dales et al., 2016) and low certainty evidence tended against associations with Nox (Reuben et al., 2021; Dales et al., 2016; Jorcano et al., 2019; Mueller et al., 2019). Evidence was low certainty but supportive of associations of EC/BC/ECAT and anxiety (Brunst et al., 2019; Shen et al., 2021; Yolton et al., 2019) and depression (Shen et al., 2021; Yolton et al., 2019), NO_2 and depression or mood disorder (Latham et al., 2021; Roberts et al., 2019; Dales et al., 2016), $PM_{2.5}$ and depression or mood disorder (Latham et al., 2021; Roberts et al., 2019; Dales et al., 2016). Evidence was split and low certainty for associations of ozone (Manczak et al., 2022; Zhao et al., 2020; Zhao et al., 2019; Dales et al., 2016) and PM_{10} (Latham et al., 2021, Zhao et al., 2020) with these outcomes.

2.3.2.5 Conduct disorder, aggression, and externalising problems

Six studies evaluated associations between air pollution and externalising problems, conduct disorder, and aggressive behaviours in adolescence (see Table 1b). There was moderate certainty evidence that NOx was not associated with these outcomes (Reuben et al., 2021; Roberts et al., 2019; Jorcano et al., 2019; Karamanos et al., 2021; Mueller et al., 2019) and evidence tended to suggest there were not associations with $PM_{2.5}$ though with low certainty (Reuben et al., 2021; Jorcano et al., 2019; Karamanos et al., 2021; Roberts et al., 2019). These studies included the whole age span of adolescence. Yorifuji and colleagues (2017) did find evidence in support of associations with Suspended Particulate Matter (SPM) and SO_2 in 0-14 year-olds, though this single study could not be evaluated with respect to certainty. Jorcano and colleagues (2019) did not find evidence for associations with PAHs in 7-11 year-olds.

2.3.2.6 Psychosis and thought disorder

Two studies from one UK-based cohort evaluated symptoms of psychosis in 18 year-olds, which was supportive of associations with $PM_{2.5}$ and Nox, though low certainty due to the limited number of studies (Newbury et al., 2019; Reuben et al., 2021).

2.3.2.7 Suicide and self-harm

Three studies evaluated air pollution and suicides or suicide attempts in adolescents (Casas et al., 2017; Fan et al., 2019; Liu et al., 2019). Two studies reported associations with $PM_{2.5}$ (Fan et al., 2019; Liu et al., 2019) though the small number meant this evidence was evaluated as low certainty, and a single study found evidence in support of associations with PM_{10} and ozone (Casas et al., 2017). Another study reported associations of $PM_{2.5}$, CO, and ozone, but not NO_2 or SO_2, with non-suicidal self-injury in 8-24 year-olds (Liu et al., 2018).

2.4 Discussion

2.4.1 Summary

The study findings had a high degree of variation but more often reported detrimental associations between the air pollutant exposures and psychological outcomes in adolescence than did not. The mechanisms through which pollutants could affect the brain are not the same for all pollutants (e.g., ozone is not thought to pass the blood-brain barrier), so the potential effects of different pollutants were evaluated individually.

Associations for which there was high certainty were $PM_{2.5}$ and worsened executive function/attentional skills, and $PM_{2.5}$ and psychiatric emergencies. There was moderate certainty evidence for detrimental associations between PM_{10} and executive function/attentional skills, and $PM_{2.5}$ and general intelligence/IQ. There was moderate

certainty evidence against associations between NO_2 and externalising problems and $PM_{2.5}$ and internalising problems.

The relatively small number of studies and heterogeneities in studies precluded meta-analysis of the adolescent literature. A vast range of outcomes were assessed in this literature, and within each outcome several metrics were employed to measure the same construct. The wide range of outcomes evaluated in the mental health literature meant that evidence for most associations was low certainty as a result of insufficient data. Far more studies were identified in child and older adult populations. Therefore, there was limited opportunity to make clear conclusions for most mental health/cognitive outcomes, warranting further research in this age group. Yet, associations were far more likely to be detrimental overall than null or beneficial.

Exposure of the world's population to pollution is increasing in prevalence and severity (Moore et al., 2003, Ritchie and Roser, 2018), and if this negatively impacts cognitive abilities, it has wider implications for exposed persons' socioeconomic conditions, health outcomes, and overall wellbeing (Batty et al., 2007, Firkowska-Mankiewicz, 2011). Therefore, these results suggest that policies mitigating such exposures could have a range of benefits for human health and wellbeing beyond cognitive performance.

2.4.2 Evaluation

Most research focussed on younger adolescents (10–12-year-olds) and a much larger body of evidence was identified for children (0-10 years, N=76). Yet, the brain, mental health, and cognitive abilities are still rapidly developing up to a person's mid-twenties (Sawyer et al.,

2018), so the mid-late adolescent period requires far more attention, thus further illustrating the need for the project detailed in this thesis.

Noise and air pollution covary, due to common sources such as road traffic, and if they have similar effects there is the risk of residual confounding of one to the other. Only three studies controlled for noise (Saenen et al., 2016, Forns et al., 2017, Van Kempen et al., 2012, Mueller et al., 2019). So, the relationships between these exposures upon cognition and mental health have not been adequately disentangled. There is also the possibility of exposure from another time window confounding the relationship between exposure during the study period and cognition. People's exposure at different periods of their life may be correlated, and if, like most of the reviewed studies, only one period is considered (say the year prior) this could be acting as a proxy for a different period (say, the prenatal period), and not itself be the critical developmental exposure period that it appears to be. Exposures at different life stages could also affect psychological development differently, via different mechanisms. For example, prenatal exposure may reduce corpus callosum volume and increase amygdala volume, which could impact verbal skills and stress response, but in adolescence exposure to air pollution might mitigate development of functional connectivity between the frontal cortex and other areas, interfering with development of advanced cognitive skills such as future-orientation, logical reasoning, and theory-of-mind (Caetano et al., 2021, Herting et al., 2019, Konrad et al., 2013, Lubczyńska et al., 2021, Mortamais et al., 2019, Nosarti et al., 2004).

Further, exposure was usually estimated at a single location, usually home, or sometimes school, which does not precisely approximate most people's real-life exposure with ecological validity and warrants more studies which evaluate exposure at multiple locations.

There was vast heterogeneity in the effect sizes even for studies of the same topic. However, a number of high-quality studies, in terms of large sample size and rigorous study design, found robust estimates, so we could tentatively conclude that air pollutants are associated with some aspects of cognition and mental health, and that the inconsistencies are attributable to complexities in the phenomena (i.e. the effect depending on the specific context and population) and the heterogeneities in the studies (e.g. different methods and tools being used, different confounders being adjusted for, different exposure windows). However, much more research needs to be done to establish if and how pollution affects adolescent cognition and mental health, and which domains are most affected.

2.4.3 Future research

Most studies could not be meta-analysed due to incompatible methodologies, and there were many inconsistent findings which may at least, in part, reflect differing study methodologies. Often researchers look for a unique angle, but scientifically this may have led to many similar studies which are not directly comparable, precluding clear conclusions. There should be a standardisation of the field where researchers seek to apply the same methods of outcome assessment and statistical analyses as past or concurrent research and thereby enable direct comparison of findings. Studies would most advance the field by either evaluating an entirely new subject area or by replicating methodology. Existing cohort studies and emerging projects should apply an integrated global approach, and initiatives which establish agreed core metrics, such as that by the International Alliance of Mental Health Research Funders for aggregating data related to mental health, are welcomed.

Risk of bias by confounding can be reduced by controlling for socioeconomic status (education and income), ethnicity, gender, age, and other covarying exposures (e.g.

environmental tobacco smoke/ETS, noise, other pollutants, neighbourhood). Birth factors are important covariates to control for studies of early life exposure (prenatal or neonatal exposure could still have adolescent impacts), as there is evidence that pollution exposure is associated with birth outcomes, which in turn are associated with brain development and cognition (Arpino et al., 2010, Batty et al., 2007, Smith et al., 2020, Smith et al., 2017). There is a great need for more literature on air pollution and psychological outcomes in adolescents and young adults, given the developmental changes that occur in this period. Processing speed, reaction times, and visuospatial skills were under-investigated.
Often studies looked at just one pollutant or looked at pollutants separately, and multi-exposure models (multi-air pollutant models or joint air pollutant and noise models) are key for several reasons: because one correlated exposure may be acting as a proxy for another; one pollutant could mask the effects of another; because their impact may be different depending on other exposures; and because mixed exposure is more representative of real life than exposure to a single isolated component. Some studies of cognition and air pollution have found that other pollutants (Wurth et al., 2018) or noise (Tzivian et al., 2017) enhanced or were even required for a significant effect, so this needs further exploration in future research. Future research should also further consider the roles of different exposure windows.

The modifying role of biological, environmental, and psychosocial factors, such as genetics and neighbourhood socioeconomic disadvantage, have only been investigated in a small number of studies of air pollution and psychological outcomes. In the adolescent literature, there was evidence of modification with stronger effects in girls (Sears et al., 2020), in HIV positive participants (Suter et al., 2018), and in high noise-exposed participants (Van Kempen et al., 2012). Research in other age groups has also found evidence for modification by

neighbourhood social stressors (Ailshire et al., 2017), *APOE* ε4 status (Alemany et al., 2018, Kulick et al., 2020) single nucleotide polymorphisms (Colicino et al., 2016), telomere length (Colicino et al., 2017), prenatal plasma folate levels (Loftus et al., 2019), demonstrate the complexity of the relationships between environmental exposures and psychological outcomes. Research should investigate the modifying circumstances further, which may help to explain differential estimates between studies, age-groups, and populations. Longitudinal studies comparing multiple exposure windows, and inter-country or inter-age studies, would also be valuable for clarifying how the air pollution-psychological outcomes relationships present in time and space. Mental health was unexplored in the cognitive literature and vice versa.

Finally, studies investigating observational associations should more often combine this with mechanistic investigations in the same population - measuring indicators of response (e.g. oxidative stress, cortisol, neuroimaging, neuroscientific markers, cognitive tests, and questionnaire data on mental health or sleep quality) to build up the evidence that these plausible mechanisms are responsible and to enhance possible causal inference. Examples in the literature have investigated oxidative stress (Sánchez-Rodríguez et al., 2006), lung function (Aretz et al., 2021, Huls et al., 2018), maternal thyroid hormones (Li et al., 2021), gut microbiota (Zhang et al., 2021), and brain structure (Crous-Bou et al., 2020), though not in adolescent groups. One adolescent study (Clark-Reyna et al., 2016) did report mediation of the relationship between known metabolic disruptors and GPA by BMI. Two studies in other ages reported that depression mediated the association between pollution exposure and cognitive outcomes in older people (Petkus et al., 2021, Tallon et al., 2017). Physical health was largely unexplored in this literature, which is associated with pollution (Bortolato et al.,

2015, Halahakoon et al., 2019, Moran, 2016), so could be an important mediator or effect modifier.

2.4.4 Strengths and limitations

This review has several strengths, it is thorough and comprehensive: providing an up-to-date review of the growing literature on the relationships between air pollution and psychological outcomes in adolescents up to 2022. Two researchers completed paper selection and data extraction for the cognition literature. That said, it was not conclusive, because there is so much evidence that could not be quantitatively synthesised. If future research is standardised to align with other research, there is no *a priori* reason meta-analyses cannot be employed in future to refine our understanding of how these exposures affect adolescent psychological outcomes.

Some shortcomings to this research should be noted. There is always the possibility, due to systematic or human error, that relevant studies are missed, although our methodology was systematic and customary. The choice of search engines also has the potential to miss studies located in low- and middle-income countries (LMICs). The focus of the review was on mental health disorders, symptoms, and cognition in the general population, as opposed to neurodevelopmental disorders including cognitive and mental health symptoms (e.g. ADHD, autism). However, it is appreciated that this represents a continuum, and it is a somewhat arbitrary distinction, and so evidence related to the question of psychological outcomes may not have been included due to where this line was drawn. Further, studies not written in English were not included, so some evidence may have been missed on this basis. Finally, the qualitative syntheses and judgements of certainty were based on a partially computable method, however there is still room for some subjectivity in the interpretation of the

collective evidence in its various combinati–ns - therefore we would recommend that, for studies of particular interest or relevance, researchers read the relevant table entries as well as the qualitative syntheses.

2.4.5 Concluding comments and implications for wider project

This review is supportive of a general association between environmental air pollution and adolescent psychological outcomes. This research has important implications for this PhD going forward. Firstly, the relatively small size of the adolescent literature and lack of certainty for most outcomes reinforces the importance and value of further research in adolescents. Secondly, processing speed as an outcome was vastly understudied, which was therefore included as an outcome when selecting cognitive metrics in the current thesis. Thirdly, the importance of studies that evaluate effect differences in various socioeconomic groups is reinforced, as only gender has been studied previously as a possible sociodemographic modifier. Fourth, the importance of multi-pollutant models and inclusion of noise in studies of exposure to air pollution was illustrated. Fifth, most studies evaluated exposure at home or school only, rather than modelling exposure throughout the day, so studies incorporating exposure at both locations may increase the validity and generalisability of findings. Sixth, many studies have not looked at the effects of exposure over time on changing outcomes, compared different exposure windows, or evaluated the impact at different stages of adolescence (usually focussing only on early adolescence), therefore this PhD will evaluate year-prior exposure to baseline outcomes, the effects of exposure over time in relation to changes in outcomes between baseline and follow-up at ages 13-15 years, and the effects of exposure from ages 11-16 years on follow-up outcomes at 16-18 years. Finally, mechanisms of association via physical, cognitive, and mental health factors were

understudied given that they present plausible mediators, which as a result was incorporated into the analysis plan for this thesis.

Chapter 3: Systematic review of the literature studying associations between noise pollution and adolescent mental health and cognition

3.1 Background

Similar to associations between air pollution exposure and psychological outcomes, the existing literature on how noise exposure relates to cognitive and mental health outcomes in adolescence has not been recently synthesised. A previous review synthesised the evidence for cognitive associations with noise across the lifespan prior to June 2015 (Clark and Paunovic, 2018a) and another systematically reviewed the evidence for neurodevelopmental and mental health outcomes in under 18's in relation to noise exposure for literature up to March 2018 (Zare Sakhvidi et al., 2018b). Clark & Paunovic (2018a) found moderate quality evidence in support of associations between aircraft noise and children's reading and oral comprehension and poorer long-term memory, with low or very low certainty evidence for other associations. Zare Sakhvidi and colleagues (2018a) reported only low or very low certainty evidence for mental health outcomes and noise, with the most consistent evidence between road traffic noise and total SDQ score, and road traffic noise and hyperactivity/inattention. These studies did not distinguish between children and adolescents. An up-to-date synthesis of the more recent literature focussed on adolescence was required for the present study, therefore this study aimed to update these reviews and synthesise their previously identified adolescent literature with recent findings.

Therefore, the objective of this study is to provide an comprehensive review of the current literature and evaluation of the evidence base, in order to inform the epidemiological study that follows and provide evidence on the question of whether noise exposure is associated with adolescent psychological outcomes.

3.2 Methods

This chapter has been written in accordance with the PRISMA checklist for reporting systematic reviews and meta-analyses (Page et al., 2021). The wider systematic review project was registered on PROSPERO prior to commencing (CRD42019151923). The PECOS (Morgan et al., 2018) statement for this research is as follows:

- Participants: human populations aged 10-19 years
- Exposure: environmental noise (exposure to noise in day-to-day life, as opposed to noise presented in a lab to measure concurrent effects on cognition and mental health).
- Control/Comparison: persons less exposed (continuously or in high/low exposure groups).
- Outcomes: general population cognitive abilities, specifically general cognitive ability and IQ, executive function, processing speed, academic ability, attention, memory and learning, language/reading abilities, reaction times, and visuospatial skills. Mental health outcomes, specifically depression, anxiety disorders, OCD, bipolar disorder, psychosis, internalising symptoms, externalising symptoms, PTSD, self-harm, eating disorders, conduct disorder.
- Study Design: epidemiological study designs - cohort, case-control and observational studies

3.2.1 Update Strategy

Using the AMSTAR 1 (A Measurement Tool to Assess Systematic Reviews) and ROBIS (Risk Of Bias In Systematic reviews) methodologies for assessing quality of systematic reviews (Whiting et al., 2016, Shea et al., 2007), the reviews by Clark & Paunovic (2018a) and Zare Sakhvidi and colleagues (2018) were assessed as sufficient quality to update rather than repeat their literature searches. Thus, the cognition search was limited to those studies published after June 2015 (when Clark & Paunovic's search took place) and the mental health

search to studies published after March 2018. I based search terms upon the ones that they utilised and used their identified studies in the qualitative syntheses. However, their reviews were only updated with respect to the literature identified in their search, screen, and select process, and the previously identified and reviewed studies of adolescents (not children) were reviewed anew for the present study using the same methodology as the more recent literature, to allow for comparability and coherent synthesis.

3.2.2 Search strategy and eligibility criteria

The online databases EMBASE (Ovid), PubMed, PsychINFO (ProQuest) and Web of Science were searched for epidemiological studies of the relationship between noise exposure across the lifespan (including prenatal period) and cognitive functioning and mental health in human adolescence. Search terms and strategy can be found in Appendix IV, and included searches for studies involving children though they were not included in the selection criteria for this review. These searches were conducted on 26/09/2019, 14/07/2021, and 28/03/2022 by title/abstract. Reference lists of studies were also scanned for any further literature.

The eligibility criteria were as follows:

1. Studies reporting cognitive and mental health outcomes in adolescence. However, studies of neurodevelopmental, learning, or neurological disorders were not included (autism, learning disability, ADHD).
2. Noise exposure only included the estimated real-life day-to-day environmental noise exposure (e.g. traffic noise, aircraft noise, day–evening–night noise level (Lden) in decibels (dB), equivalent continuous sound level in db (LAeq), night-time noise in dB (Lnight)) and not experimental manipulations of noise measuring concurrent cognition.

3. Conference abstracts and grey literature were eligible for inclusion if they reported an independent effect of exposure on outcome that could not be found in a published academic paper.
4. Publications written in English, as there were not resources available for translation.
5. Outcomes assessed in human populations aged 10-19 years, with exposure occurring anytime beforehand (including prenatally). Studies of childhood with age 10 as the upper range and studies of adulthood with 19 as the lower range were excluded.

3.2.3 Review process

The same process as described in chapter 2 was applied for this review. The cognition search was conducted by the same reviewers in duplicate, but the mental health search (2018-2022) and data extraction was unfortunately not conducted in duplicate due to limitations on resources. Where a publication was not available through institutional access, it was requested from authors.

3.2.4 Risk of bias and Quality of Evidence Assessment

The OHAT Risk of Bias Rating Tool (OHAT, 2015) was used to evaluate risk of bias in an equivalent manner to the systematic review in Chapter 2. The ratings for each field by paper are provided in the main summary tables (3a and 3b). In the same methodology as the previous chapter, these fields were then used in application of the GRADE methodology for evaluating the overall quality of evidence (Morgan et al., 2016).

3.2.5 Meta-analysis

The same methodology of chapter 2 was applied here: all outcomes, exposures and statistics were tabulated to see if pooling of estimates would be meaningful, effect estimates were

extracted and converted to the same scale or type of statistic if necessary, and random effects models were estimated using the package "meta" (Balduzzi et al., 2019, Core, 2019).

3.3 Results

We identified 4777 records in the initial searches, which after exclusions left 30 studies to be included in the qualitative synthesis (see Figure 5). The level of agreement on paper selection was initially moderate (Cohen's Kappa 0.74) but reached 100% agreement following a single round of discussion. Twenty-one studies were identified evaluating cognition and 9 evaluating mental health.

Most cognitive studies (17/21) focussed on early adolescence (<13 years), with only 3 papers from two studies including those aged 13-17 years, and one looking at 18-22 year-olds. Though there were fewer studies, the mental health studies had better coverage of the age range (ranges of 7-18, 8-11, 9-14, 10, 10-11, 10-17, 13-18 and 18-35 years). Older studies tended to be cross-sectional but most exposure-outcome associations for mental health had some longitudinal evidence, whereas the cognition literature (which also tended to be older) was often rated lower due to cross-sectionality.

Literature on cognition tended to be from the USA and Europe: N=7 papers from the USA, N= 10 Europe, N=1 Thailand, N=1 Mexico, and N= 2 (same cohort) from South Africa. The mental health literature also had a European bias: N=6 from Europe, 1 from USA, N=1 Singapore, N=1 South Korea.

Characteristics of these individual studies and their risk of bias in the domains specified in the OHAT are summarised in Tables 3a (cognition literature) and 3b (mental health

literature). Table 4 outlines the GRADE assessed certainty of collective evidence for associations investigated multiple times in the literature. Appendix V lists the associations only investigated in one paper, thus highlighting gaps in the literature.

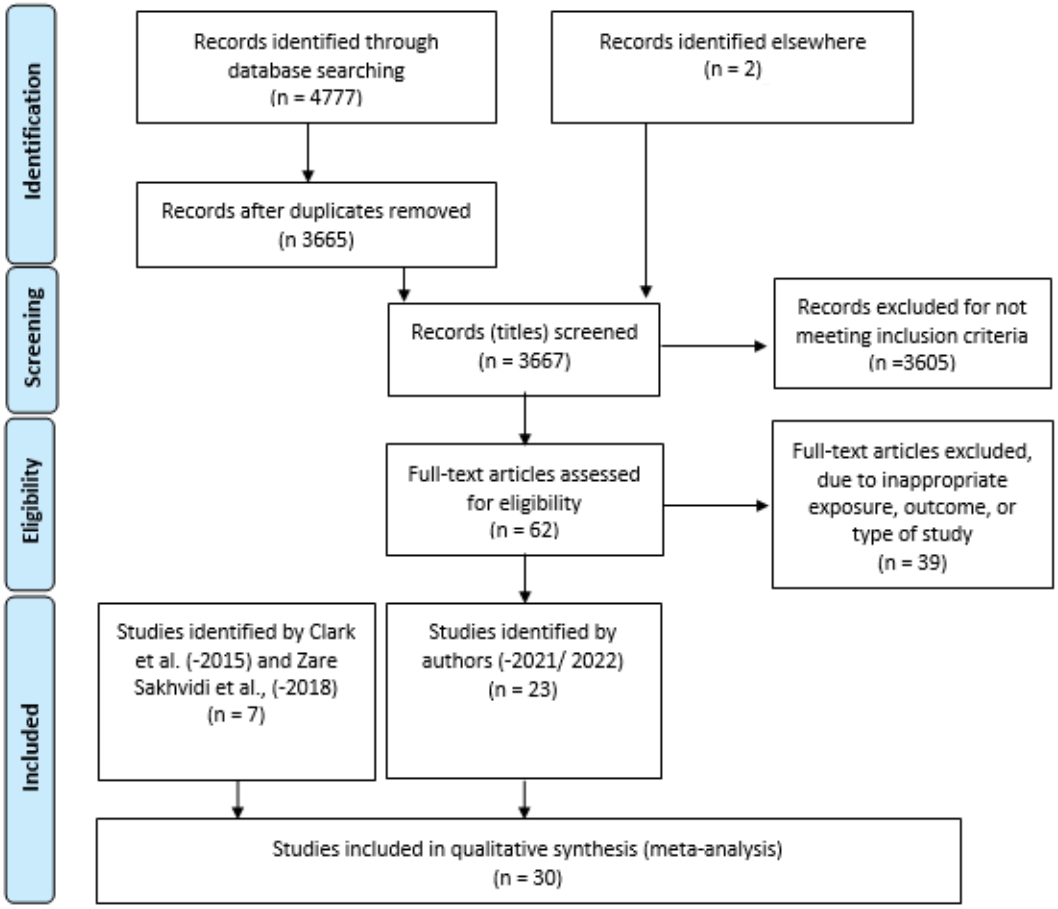


Figure 5. PRISMA diagram of search results[4]

[4] *From:* Moher D, Liberati A, Tetzlaff J, Altman DG. The PRISMA Group (2009). *P*referred *R*eporting *I*tems for *S*ystematic Reviews and *M*eta- *A*nalyses: The PRISMA Statement. PLoS Med 6(7): e1000097. doi:10.1371/journal.pmed1000097

Table 3a. Literature identified in this systematic review, findings and risk of bias assessment for cognitive outcomes

Paper	Analytical sample: n, age (μ and σ unless stated otherwise), gender, location	Measurement of exposure	Measure of cognition	Confounders considered (controlled for or demonstrated unrelated)	Outcomes- most adjusted model *p<.05	Risk of bias			DH: Definitely High PH: Probably High PL: Probably Low DL: Definitely Low		
						Selection bias	Confounding bias	Attrition/Exclusion bias	Exposure characterisation	Outcome characterisation	Selective reporting bias
Bronzaft, 1975	N=161 2nd, 4th and 6th graders (14 classes, 6-12 years old), USA	Noise exposure measured in one classroom of school, comparing noisy classrooms (measured, mean 59 dBA and max 89 dBA) to quiet classrooms (unspecified).	The California Achievement Test (CTB-McGraw Hill, 1977) based Reading achievement (word knowledge and reading comprehension)	None	Children on noisy side of school had poorer reading compared to children on quiet side of school	PH	PH	PH	PH	DL	PL
Bronzaft, 1981	Same study as above. N=350 (first testing), N=605 (second testing), 2nd-6th graders, USA	Noise exposure measured in one classroom of school, comparing noisy classrooms (measured, mean 59 dBA and max 89 dBA) to quiet classrooms	The California Achievement Test (CTB-McGraw Hill, 1977) based Reading achievement (word knowledge and reading comprehension.	None	Children on noisier side of school had poorer reading achievement, but after abatement children on both sides had similar scores	PH	PH	DL	PL	DL	PL

		(unspecified). Then compared pre and post noise-abatement measures									
Cohen, 1973	N=54 childre[n] (2[nd]-5th grade), 46.3% female, USA	Measured noise levels outside apartment buildings (75-84 dBA), inside hallways, and inside apartments to confirm floor level as noise exposure metric. Participants lived in apartment buildings of varying noise levels	The metropolitan Achievement Tests – word knowledge, reading comprehension, reading total . Wepman auditory discrimination test.	None	Children living in noisy apartments for more than 4 years had correlations between floor level and auditory discrimination and floor level and reading ability, and between auditory discrimination and reading abilities, suggesting noise exposure impaired reading through auditory discrimination.	PH	PL	PH	PH	PL	PL
Eagan et al., 2004 and Zusman, 2007 (FICAN report)	N=unspecified 6-18 year olds from 35 schools, USA	Modelled indoor aircraft noise, before and after an airport closure or school sound insulation.	Mandatory state-standardised tests (e.g. TAAS, IGAP), producing failure rate, average test score (%), top-score rate (% pupils with best score)	Cause of noise reduction, state, change in test regime, overall wealth and parental education, Spanish language, socioeconomic status, school-district size	Noise reduction associated with reduced failure rate in high school compulsory verbal and math/science tests, but not younger pupils, and an increase in top-score rates of children with disabilities.	PH	DL	PH	PL	PL	DL

Green, 1982	N=unspecified 2nd-6th graders, USA	Modelled aircraft noise contours around schools, assigned noise scores to 362 schools	Nationally standardised tests- % of students reading below grade level	% free meals, % different ethnicities, absentee admission and departure rates, pupil-teacher ratio, teacher experience, teacher education	Aircraft noise associated with % pupils below grade reading level in 3-5th but not 6th graders	DL	PL	PL	PL	DL	PL
Haines, 2001a	N=451 8–11-year-olds, 50.4% female, UK	Aircraft noise modelled around schools and measured inside classrooms. Grouped into 10 noisy (16hr Leq > 63 dBA) and 10 quiet (<57dBA) schools.	Suffolk reading scale, the children's memory scale, backward digit span, TEA-Ch (test of everyday attention for children)	Age, first language, household deprivation	High noise exposed children had poorer performance on difficult items of the Suffolk reading comprehension scale, but did not significantly differ on total reading comprehension score, sustained attention, immediate recall, delayed recall, delayed recognition, or backward digit span	DL	PH	PL	PL	DL	PL
Haines, 2001b	N= 340 8-11 year olds studied, and N=275 again a year later, 52% female, UK	Aircraft noise levels modelled around 20 schools, Noisy classrooms: 16hr Leq >66dBA, quiet: <57 dBA	Suffolk reading scale, TEA-Ch	Age, first language, household deprivation	At follow-up children from high-aircraft noise-impacted schools had worse reading comprehension and sustained attention than children from low-aircraft noise-impact schools.	DL	PH	PH	PL	DL	PL

Haines, 2001c	N=340 8-11 year olds, 50% female, UK	Aircraft noise modelled around 8 schools, and measured indoors, categorised as noisy classrooms (16hr Leq >66dBA) or quiet (<57 dBA)	Suffolk reading scale, long-tern memory of it (like Evans et al., 1995), motivation	Age, first language, household deprivation	High noise exposed children had poorer performance on reading comprehension and long-term memory recognition (in a subsample) than low noise exposed children. Aircraft noise had no significant effect on long-term memory recall performance or short-term memory and recognition.	DL	PH	PH	PL	DL	PL
Haines, 2002	N=10957-11163 (depending on test, 11 year olds, UK	Aircraft noise modelled around 123 schools, Low noise exposure Leq (16 hr) =<57 dBA; Moderate noise exposure=57>63 dBA; high noise exposure=63>72 dbA.	the National Standardised Scores (SATs) in mathematics, science, and English	Sex, age, school type, eligibility for free school meals, first language, special needs	Increased aircraft noise contour band associated with decreased SAT Key Stage 2 standardised reading and maths test performance but not after controlling for average SES of school.	DL	PL	DL	PL	DL	DL
Hygge et al., 2002	N=326 8-12 year olds, (*M* = 10.4, *SD* = 0.85, Germany	Range of mean 24 hr Leq: 53-68dBA. Measured aircraft noise levels around schools before and after relocation of an airport; 43 in the old-airport, no-	Standardised german reading test, long term memory for text read the day prior, visual search and reaction time, speech-in-noise perception	None, matched on sociodemographic characteristics	Newly exposed; decreased correct answers on long-term memory test, impairment of speech perception. After relocation of old airport, formerly exposed showed; decreased number of errors on reading test,	PL	PH	PL	DL	DL	PL

		noise group; 65 in the old-airport, noise group; 107 in the new-airport, no- noise group; and 111 in the new-airport, noise group.			increased correct answers on short-term memory test, improved speech perception. More exposed group were slower in attentional task at time 2 (after relocation) but not 1 or 3, and after the new airport opened the newly exposed group were slower than non-exposed group.						
Julvez, 2021	N=1275 mother-child pairs, Age 6-11 (Mean age 8), 45.3% female, UK France Spain Lithuania Norway Greece	Modelled 24-hour traffic noise prenatally, and at residence during childhood, categorised in dB(A): <55; 2:55-59.9; 3: 60-64.9; 4: >=65	Raven Coloured Progressive Matrices Test (CPM), Attention Network Test (ANT), and N-Back task. *Fluid intelligence, attention, working memory*	Cohort, maternal age, maternal education level, trimester of the year of child conception, child age, child sex	None of prenatal traffic noise (Lden), or traffic noise (Lden and Lnight) at home during childhood, were significantly associated with cognition. Effects of high noise (≥65dB (A)) relative to low (<55) ranging from -18.158 (night noise and ANT (HRT-SE), 95% CI: -45.395, 9.078) to 0.106(prenatal traffic noise and CPM number correct responses, 95% CI: -0.844, 1.056).	DL	PL	PH	PL	DL	PH

Lukas, 1981 (report)	N=18[26] 3rd a[nd] 6th graders, USA	Traffic noise levels measured outside schools, in community and inside classrooms, comparing noisy and quiet schools	Academic performance	Nk	Pupils had poorer reading and mathematics achievement compared to those from quieter schools (e.[g.] 6th graders were 0.7 years behind in reading).	PL	PH	DL	DL	DL	PL
Matheson, 2010	N=2844 9-13 year olds from 89 schools. UK, Spain, Netherlands	Aircraft noise measured or modelled at home and school. Road traffic noise combined from measurements and models. Aircraft noise at school ranged from 32 to 77 dB $L_{Aeq\ 16\ hour}$ and road traffic noise at school ranged from 31 to 71dB $L_{Aeq\ 16\ hour}$.	Child's memory scale, prospective memory test (Shield & Dockrell)	classroom glazing, age, sex, country, mo'her's educational attainment, socioeconomic status, crowding and home ownership, LSI, main language spoken at home and parental support for school work and the other noise exposure variable, e.g., for aircraft noise analyses, road traffic noise exposure was adjusted for, child having dyslexia, and acute noise during testing	Increased road traffic noise associated with increased information and conceptual recall but not recognition or prospective memory. Aircraft noise associated with decreased recognition memory, but not related to prospective memory, information recall, or conceptual recall.	PL	DL	PL	PL	DL	DL
Onchang, 2018	N= 786, mean age male off campus 21.16 ± 1.44/ female off campus 20.73 ± 1.08/ male on campus 20.61 ± 1.21/ female on campus	Measured noise in on and off campus areas to compare students living in and off campus, applied noise annoyance questionnaire (International	Self-reported cumulative GPA (0-4) *Academic ability*	None	Off campus students had significantly higher noise exposure, subjective perceptions did not differ. Campus group had significantly higher GPA than outside group (mean 2.80 vs 2.83, p<.001)	DH	DH	PH	PL	DH	PH

	20.51 ± 1.06, 73% female, Thailand	Commission on the Biological Effects of Noise). Mean $L_{eq, 24h}$ off-campus 64.7 ± SD 4.8, on-campus 60.3 ± 1.8.			Noise annoyance was not consistently related to GPA (ORs from 1.049-1.164 but 95% CIs crossing 0). In off campus group, self-reported noise impacting reading and mental tasks was related to poor GPA (OR 2.801).						
Sanz et al., 1993	N=136 6-11 year olds, Spain	Road traffic noise measured inside and outside classrooms for Quiet schools Outdoor Leq range 47.5 -69.1 dBA, Loud schools 58 5- 76 6 d BA.	Attention tasks: differences perception test and text test	None	Children from high noise school had poorer result on 'text test' in grade 3 (crossing out letters from a text, attention), no differences on face test (difference perception test).	PL	DH	PH	DL	PL	DL
Seabi, 2012	N=693 9-14 year olds, South Africa	Measured noise levels outside schools in mornings, high noise (near flight path, 16hr outdoor Leq 63.5 to 69.9) and low noise (50.5–57.9 Leq) schools compared	Suffolk reading scale	intellectual ability (figure analogies subtest of quantitative battery for cognitive abilities test), gender, and socioeconomic status	High aircraft noise groups had lower reading comprehension than low noise groups; the effect was stronger for those whose primary home language was not English	PL	PL	PL	DL	DL	PL
Seabi, 2015	9-15 year olds measured at three timepoints, South Africa	Measured noise levels outside schools before (2009) and after (2010-2011)	Suffolk reading scale	intellectual ability (figure analogies subtest of quantitative battery for cognitive abilities test), gender,	Reading comprehension significantly differed in 2009 between high and low exposed	PL	PL	DH	DL	DL	PL

		relocation of an airport. (Ns =732; 650; 178). Leq reduced from range 63.5-69.9, to average of 55.2dB.		and socioeconomic status	groups; no significant differences between high and low noise groups in 2010 or 2011 (adjusted) and no interaction effect of exposure and time on reading comprehension, i.e. removing aircraft noise did not improve reading but prior chronic exposure did not worsen it						
Sharp, 2014 (report)	N=unspecified 3rd, 4th, 5th graders from 6198 schools,USA	Modelled aircraft noise levels at school, evaluating effects of noise exposure and (for a subsample) sound insulation installation. Exterior Leq mean 54.2 dB, SD 5.2	school average test scores in reading and mathematics.	Free lunch program (%), ethnic minority (%), pupil-teacher ratio, average enrolment per grade	Aircraft noise associated with decreased school state ranking, based on school average test scores in reading and mathematics. Subsample (119 schools) had sound insulation installed which removed this effect of aircraft noise on ranking.	DL	PL	PL	PL	PL	PL
Shield and Dockrell (2008)	N=unspecified 7-11 year olds (142 schools external noise, 16 schools internal noise), UK	Noise levels measured inside and outside schools, External Leq range 49 to 75 dB (A).	SATs results	Free school meals, first language, special additional needs	External noise levels associated with average test scores of Key Stage 1(KS1) and Key Stage 2(KS2) in suburban schools but not urban schools, poorer mathematics and science in suburban schools, and poorer KS1 and KS2 English	PL	PH	PH	DL	DL	PL

					in urban schools. Internal noise associated with poorer KS1 maths and KS2 English.						
Van Kempen et al., 2010	N=553 9-11 year olds from 24 schools, Netherlands	Aircraft and road traffic noise modelled for home and school addresses, School $L_{Aeq, 7-23hr,}$ 36-63dB(A), Home Leq range 34-63dB(A)	Neurobehavioural evaluation system; simple reaction time test (SRTT), switching attention task (SAT), hand-eye coordination test (HECT), symbol-digit substitution test (SDST), DMST, reading comprehension, child memory scale	Age, gender, home language, chronic illness, parental support, single glazing, socio-economic status, other noise	Aircraft noise and traffic noise at school associated with more errors on Switching Attention Task (SAT), but not with other tests; simple reaction, hand-eye-coordination, symbol-digit substitution, digit memory, reading comprehension, prospective memory, episodic memory, working memory, sustained attention	PL	DL	PH	PL	DL	PL
Van Kempen et al., 2012	N= 485 children aged 9-11 in 24 schools. Netherlands (as above but combined with air pollution)	Aircraft noise and road noise at home and school modelled, Range 28-67dB (A)	Neurobehavioural evaluation system; simple reaction time test (SRTT), switching attention task (SAT), hand-eye coordination test (HECT), symbol-digit substitution test (SDST), digit memory span test (DMST)	Age, gender, home language, chronic illness, parental support, single glazing, socio-economic status, other noise	Aircraft and road noise at school associated with errors on SAT test even after adjustment for NO2 and confounders, but not with other tests (NO2 at school associated with digit memory span test); home exposures not associated. Combined NO2 and road noise at home related to reaction times in simple reaction time test (SRTT) and	PL	DL	PH	PL	DL	DL

					errors in SAT (children living in areas with high levels of road traffic noise had a shorter reaction time/more errors as NO2 increased)						

Table 3b. Literature identified in this systematic review, findings, and risk of bias assessment for mental health outcomes

Paper	Analytical sample: n, age (μ and σ unless stated otherwise), gender, location	Measurement of exposure	Measure of mental health	Confounders considered (controlled for or demonstrated unrelated)	Outcomes- most adjusted model *p<.05	Risk of bias			DH: Definitely High PH: Probably High PL: Probably Low DL: Definitely Low		
						Selection bias	Confounding bias	Attrition/Exclusion bias	Exposure characterisation	Outcome characterisation	Selective reporting bias
Benita, 2019	N= 49,526 students, Singapore	SENSg sensors measuring sound pressure level	Momentary wellbeing indicator (participants press button when happy)	Type of place, area, day of week, month, individual fixed effects should account for individual differences	Immediate noise levels associated with subjective wellbeing; Higher noise levels associated with higher odds of happy moment occurring	PL	PL	PL	DL	PH	DL
Dzhambov, 2017	N=720 18-35 year olds, Bulgaria	Residential noise exposure modelled using LUR	General health questionnaire -symptoms of common mental health disorders	Age, sex, ethnicity, duration at residence, average time spent at home, month of data collection, financial security, population density. Full model adjusted for mediators: environmental annoyance, sleep disturbance, restorative quality, social cohesion, and physical activity	Noise was significantly associated with mental ill-health in the crude β= 1.952 (95% CI 0.76-3.15) but not adjusted models. This was mediated by neighbourhood restorative quality reducing physical activity, and distinctly by sleep disturbance.	PH	DL	PL	PL	PL	DL

Haines, 2001	N= 451 8-11 year olds, age range 8 y1m–9y8m, 49% girls, UK	High aircraft noise (16 h outdoor Leq > 63 dBA) schools compard to low aircraft noise schools (<57dBA)	Lewis child stress scale child-reported, Strengths & difficulties scale (SDQ) parent-reported	Age, deprivation, language	Stress proneness, total difficulties, and hyperactivity problems associated with noise level (p=0.01-0.06) SDQ peer problems, conduct problems, emotional problems not associated.	DL	PH	PL	PL	PL	DL
Lercher, 2002	N=1280/ n=123 age 8–11, Austria	GIS calculated noise exposure (equivalent sound pressure level, dB,A,Leq) at the child's home address use to recruit two new samples of children at extreme levels of exposure (< 50 dB,A,Ldn vs > 60 dB,A,Ldn (day-night levels)).	Two self-reported subscales of the KINDL reflecting quality of life and four items on a sleep disturbance scale, teacher reported classroom behavioural adjustment	Maternal education, sex, people in household, house type, biological risk (small birth weight or short gestation)	Teacher rated adjustment was significantly associated with noise, and self-reported mental health was associated in those children at pre-existing biological risk	DL	DL	PH	PL	PL	PL
Lim, 2018	N=918, mean age 11.47 ± 1.54 years (age range, 9–14 years), 53.5% female, South Korea	Road traffic noise at exterior building, using noise prediction software CadnaA and noisemap	Parent completed CBCL	Age, sex, income, premature birth, maternal age at birth, passive smoking, disease during pregnancy, mental disorders	At all levels of adjustment Noise level (Ldn) associated with CBCL internalsing problems (model 4 OR 1.42,1.15-1.75, p<0.001), total problem (model 4 OR 1.08, 1.01-1.15,p=0.034), but not externalising problems (M4: OR 1.03, p=0.25).	PH	DL	DL	PL	PH	DL

Rudolph, 2019	N=4508, 13-18 years olds (mean age 15.3), 51.6% female (low noise)/49.6% female (high noise)	Average Ldn in residential census block group, geospatial sound models, dichotomized as high (≥55 dB) versus low (<55 dB).	Composite International Diagnostic Interview (CIDI) interviews with adolescents: Sleep pattern, history of anxiety, depression, behavioural (conduct or oppositional defiant) disorder, ADHD, substance use disorder	Air pollution, greenness, temperature, population density, neighbourhood SES, sex, age, ethnicity,	High noise exposed had significantly higher risk of substance use (OR 2.42, 1.05-5.57), but not depression/anxiety (1.58 (0.89, 2.80), ADHD (0.71 (0.38, 1.31)) or behavioural disorder (0.82 (0.31, 2.18))	DL	PL	PL	PH	DL	DL
Ristovska,2004	N=529, 10-11 years old, 50.1% female, Macedonia	average energy-equivalent sound level in residential area, compared those exposed to noise level LAeq, 8 h >55 dBA (n=266) and children exposed to noise level LAeq, 8 h <55 dBA (n=263).	Self-reported General anxiety scale Teacher reported attention, hyperactivity, social adaptability, and opposite behaviour	Education & employment of parents, housing	Noise associated with attention (p=0.03), social adaptability (p=0.03) and opposite behaviour (p<0.001) , but not anxiety (p=0.11) or hyperactivity (p=0.22).	PH	PH	PL	PH	PL	PL
Tangermann, 2022	N=887, mean age 14.0 (range: 10.4–17.0), 56.5% female , Switzerland	Modelled Lden, Lnight, lday for transportation noise (rail and traffic) at homes and schools.	Parent report SDQ-total, emotional, conduct, hyperactivity/inattention, peer problems, and prosocial behaviour	Parental education, age, sex, education level, alcohol, smoking, physical activity, screentime	Cross-sectionally, 1 10dB increase road traffic noise associated with increased peer problems by 0.15 units (95%CI: 0.02, 0.27). Longitudinally and other outcomes not associated in adjusted models.	PH	PL	DL	PL	PH	DL

Tiesler, 2013	N=872 10 year olds, 47% female, Munich	Residential road traffic noise, the Munich noise map	Parent reported SDQ-total, emotional, conduct, hyper/inattention, peer problems	Sex, age, study, parental educational level, mothers age at birth, television/computer usage, single parent status	Lden associated with hyperactivity/inattention (OR= 1.28(95%-confidence interval(CI):1.03–1.58) and emotional symptoms scale, (2.19(95% CI:1.32–3.64). Results for Lnight similar. Nocturnal noise was associated with sleeping problems.	PH	PL	DH	PL	PH	DL

Table 4: GRADE assessed quality of evidence for noise exposure-outcome associations

Exposure	**Outcome**	**Papers**	**N papers**	**Direction of evidence**	**Start rating** **3- moderate** **Long** **Comparison** **Non-ind**	**Downgrade?** **Risk of bias?** **Wide Cis?** **Inconsistencis?**	**Upgrade ?** **Dose-response, large effect**	**Finish**
Traffic noise	**General psychopathology / problems**	Lim, 2018; Tangermann, 2022; Tiesler, 2013	3	Against	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: Inconsistency, Risk of bias concerns	No	Low
General noise	**Depression/anxiety**	Rudolph, 2019; Ristovska, 2005	2	Against	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: Risk of bias concerns and imprecision	No	Low
Traffic noise	**Internalising**	Lim, 2018; Tangermann, 2022; Tiesler, 2013	3	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: Risk of bias concerns	Yes: Large effects	Moderate
General noise	**Externalising difficulties**	Rudolph, 2019; Ristovska, 2005	2	50/50	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: Inconsistency, Risk of bias concerns, imprecision	No	Low
Traffic noise	**Externalising difficulties**	Lim, 2018; Tangermann, 2022; Tiesler, 2013	3	Against	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: Inconsistency, Risk of bias concerns	No	Low
Aircraft noise	**Reading**	Haines, 2001a, Haines, 2001b, Haines, 2001c, Seabi, 2012; Seabi, 2015, Van Kempen et al., 2010,	6 (3 cohorts)	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: inconsistency	No	Low
Railway noise	**Reading**	Bronzaft, 1975, Bronzaft, 1981 (same sample)	2 (1 cohort)	Supportive	Low Reason: longitudinal evidence, individual level data, but no clear comparison used	Yes Reason: imprecision and risk of bias concerns	No	Very Low

General noise	**Academic achievement**	Shield and Dockrell (2008), Onchang, 2018	2	Supportive	Low Reason: individual level data, comparison used	Yes: imprecision, risk of bias concerns	No	Very Low
Aircraft noise	**Academic achievement**	Eagan et al., 2004/ Zusman, 2007; Green, 1982, Haines, 2002; Sharp, 2014	5 (4 cohorts)	Supportive	Low Reason: longitudinal evidence, comparison used, but non-individual level data	No	No	Low
Aircraft noise	**Attention and executive function**	Haines, 2001a, Haines, 2001b,,Hygge, 2002, Van Kempen et al., 2010, Van Kempen et al., 2012	5 (3 cohorts)	Supportive	Moderate Reason: Longitudinal evidence, individual level data, comparison used	No	No	Moderate
Traffic noise	**Attention and executive function**	Sanz, 1993, Van Kempen et al., 2010, Julvez, 2021, Van Kempen et al., 2012	4 (3 cohorts)	Supportive for sustained attention, not for other modalities	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Sustained attention: No Attention/EF generally: Yes (inconsistency)	No	Moderate for sustained attentat /Low for other
Aircraft noise	**memory**	Haines, 2001a, Haines, 2001c, Hygge, 2002, Matheson, 2010, Van Kempen et al., 2010, Van Kempen et al., 2012	6 (4 cohorts)	Against	Low Reason: individual level data, comparison used	No	No	Low
Traffic noise	**memory**	Matheson, 2010, Van Kempen et al., 2010 Van Kempen et al., 2012	3 (2 cohorts)	Against	Low Reason: individual level data, comparison used	Yes Reason: imprecision	No	Very Low
Aircraft noise	**Reaction time**	Hygge et al., 2002, Van Kempen et al., 2010, Van Kempen et al., 2012	3 (2 cohorts)	Against	Moderate Reason: Longitudinal evidence, individual level data, comparison used	Yes Reason: imprecision and inconsistency	No	Low
Traffic noise	**Reaction time**	Van Kempen et al., 2010, Van Kempen et al., 2012	2 (1 cohort)	Against	Low Reason: individual level data, comparison used	Yes Reason: imprecision, inconsistency	No	Very Low

3.3.1 Cognitive outcomes

3.3.1.1 Academic performance

Two studies were identified evaluating the relationship between general noise and academic achievement in 7-11 year-olds (Shield and Dockrell, 2008) and young adults (Onchang and Hawker, 2018), which were supportive of a detrimental association but this evidence was rated very low certainty due to its size, risk of bias concerns, and cross-sectional design. Aircraft noise was evaluated in relation to academic achievement in 5 papers from 4 cohorts (Eagan et al., 2004, Green et al., 1982, Haines et al., 2002, Sharp et al., 2014, Zusman, 2007) which was supportive of a detrimental association but considered low quality evidence due to being cross-sectional. One of these studies included the 13-18 age range, the others evaluated students up to age 12 years. Traffic noise was evaluated in one study (Lukas et al., 1981) which was supportive of a detrimental association but did not control for covariates.

3.3.1.2 Language abilities

Reading in adolescence in relation to railway noise was evaluated in two papers from one study, which were supportive of a detrimental association (Bronzaft, 1981, Bronzaft and McCarthy, 1975) but presented selection bias and risk of confounding concerns. Other studies reported associations with apartment noise (Cohen et al., 1973) but not traffic noise (Van Kempen et al., 2010). All of these populations were under 13 years of age. Six papers from 3 cohorts evaluated aircraft noise in relation to reading in 8-15 year-olds (Haines et al., 2001b, Haines et al., 2001d, Haines et al., 2001c, Seabi et al., 2012, Seabi et al., 2015, Van Kempen et al., 2010) which overall were supportive of a detrimental association but evaluated as low quality due to inconsistencies.

However, a meta-analysis including two of these studies in combination with a study of railway noise (studies comparing noisy to quiet classrooms) was supportive. Six effect sizes, extracted from 3 studies reflecting mean differences in reading test scores between noisy and quiet classrooms (either rail or aircraft noise, Figure 6) were meta-analysed and scores in quieter classrooms were significantly higher (by 0.80 points, 95% CI: 0.40;1.20) with significant heterogeneity ($p<.001$). TE refers to the treatment effect of individual studies (here the difference in mean reading test scores), and seTE refers to the standard errors of treatment effects. In noisy classrooms, the average Leq was 59-69.9dBA, and Max Leq 89-96.5, whereas in low noise exposed classrooms the average Leq was 54.4-57 dBA and Max Leq 73.2-74.3. The authors applied different reading tests, therefore this is not a precise estimate of how noise relates to reading but does reflect a trend in the literature which is supportive of an effect. A meta-analysis of beta coefficients (Figure 7) from linear regressions of noise (mixed sources) upon reading/writing test performance also found a trend of noise reducing abilities although it did not reach significance (-0.11, 95% CI: -0.32;0.10), with significant heterogeneity (<.01). Different tests of outcome were also applied by these authors; therefore, this again indicates the trend of the literature rather than precisely estimating dose-response.

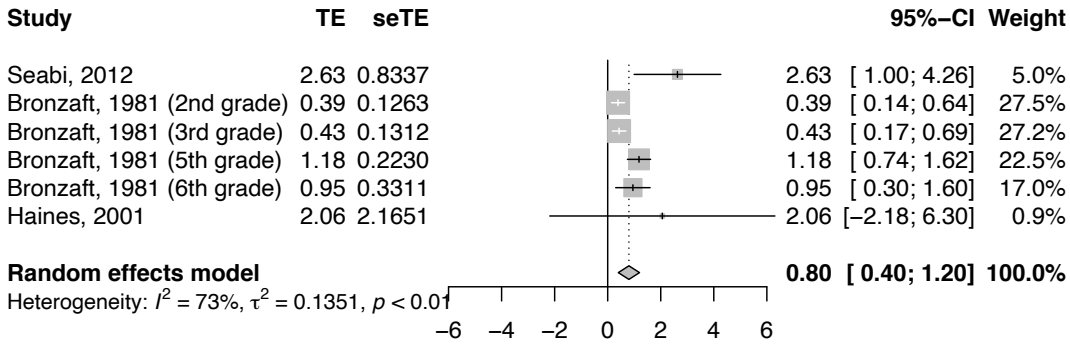


Figure 6. The pooled mean difference in reading comprehension scores between children in high noise (average Leq 59-69.9dBA, Max Leq 89-96.5) and low noise exposed (average Leq 54.4-57 dBA, Max Leq 73.2-74.3) classrooms. *Note.* TE = mean reading score of quiet classrooms – mean reading score of noisy classrooms, seTE = pooled standard error.

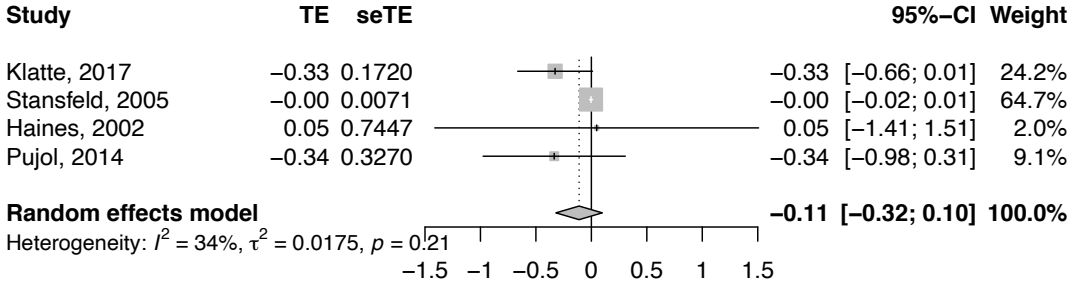


Figure 7. The pooled beta coefficients reflecting the association between environmental noise (impact of 1dB increase, from mixed transport sources) and reading and language abilities. *Note.* TE=beta coefficient (β), seTE= β standard error.

3.3.1.3 Executive function, attention, and working memory

There was moderate quality evidence in support of a detrimental relationship between aircraft noise and attention and executive function (Haines et al., 2001b, Haines et al., 2001d, Hygge et al., 2002, Van Kempen et al., 2012, Van Kempen et al., 2010), and moderate quality evidence for detrimental effects on sustained attention from traffic noise, though this wasn't the case for other attentional modalities (Sanz et al., 1993, Van Kempen et al., 2012, Van

Kempen et al., 2010, Julvez et al., 2021b). All of these were in individuals under 12 years of age.

3.3.1.4 Memory and learning

Several studies collectively suggested there was not an association between aircraft noise and memory (Haines et al., 2001b, Haines et al., 2001d, Hygge et al., 2002, Matheson et al., 2010, Van Kempen et al., 2012, Van Kempen et al., 2010) but this was cross-sectional so low certainty evidence. Similarly, evidence was against an association with traffic noise but had very low certainty (Matheson et al., 2010, Van Kempen et al., 2012, Van Kempen et al., 2010). All of this evidence was in participants aged 12 years and under.

3.3.1.5 Reaction time

Three studies evaluated reaction time in relation to aircraft and traffic noise, which were not supportive of associations and with low (Hygge et al., 2002, Van Kempen et al., 2012, Van Kempen et al., 2010) and very low (Van Kempen et al., 2012, Van Kempen et al., 2010) certainty evidence respectively.

3.3.1.6 Auditory discrimination

Two studies reported detrimental associations of apartment noise (Cohen, 1973) and aircraft noise (Hygge et al., 2002) with auditory discrimination, i.e., the ability to pick out sounds from background noise.

3.3.2 Mental health outcomes

3.3.2.1 General psychopathology and mental health

Three studies in 9-17 year-olds evaluated general psychopathology and traffic noise, which were not supportive of an association, but this evidence was low certainty due to inconsistencies and risk of bias concerns (Lim et al., 2018, Tangermann et al., 2022, Tiesler et al., 2013). One study each reported an association with aircraft noise in 8-11 year-olds (Haines et al., 2001a), a trend association with general noise in 18-35 year-olds (Dzhambov et al., 2018), and no association with railway noise in 10-17 year-olds (Tangermann, 2022). Contrary to expectation, one study of 7-18 year-olds reported that participants were more likely to report 'happy moments' in louder places (Benita et al., 2019).

3.3.2.2 Internalising difficulties

Two studies evaluated general noise and depression and anxiety and did not report evidence for associations in 10-11 (Ristovska et al., 2004) and 13-18 year-olds (Rudolph et al., 2019) but with low certainty due to the small number of studies and risk of bias concerns. Further, Tangermann and colleagues (2022) did not report associations between railway noise and internalising symptoms. However, Rudolph and colleagues (2019) did report associations with substance use, and Ristovksa (2004) with social adaptability. Further, three studies evaluated internalising symptoms and traffic noise reported associations (Lim et al., 2018, Tangermann et al., 2022, Tiesler et al., 2013), with moderate quality certainty. Haines and colleagues (2001b) reported stress proneness to be associated with aircraft noise.

3.3.2.3 Externalising difficulties

The evidence for general noise and externalising difficulties was mixed as to whether associations were present and low certainty (Rudolph et al., 2019, Ristovka et al., 2004). The evidence for traffic noise was not supportive of associations but was also low certainty (Lim et al., 2018, Tangermann et al., 2022, Tiesler et al., 2013). Tangermann and colleagues also did not find associations with railway noise (2022), and Haines and colleagues (2001b) reported associations between aircraft noise and hyperactivity but not conduct problems.

3.4 Discussion

3.4.1 Summary

The study findings had a high degree of variation but tended to indicate some associations between the different noise exposures and cognitive outcomes, with a more mixed picture for mental health outcomes. More robust studies are required particularly for mental health outcomes. The number of studies was small, and most cognition studies focussed on populations aged 12 years and under, so the mid-to-late adolescence period requires more attention. There was moderate certainty evidence for detrimental associations between aircraft noise and attentional/executive function abilities, moderate evidence for detrimental associations between traffic noise and sustained attention, and moderate evidence for associations between traffic noise and increased internalising symptoms.

If noise negatively impacts the mental health and cognition of adolescents, this suggests that, policies and interventions that reduce human noise exposure could therefore potentially improve cognition and mental health in adolescent populations in addition to supporting positive health outcomes (Brown and Van Kamp, 2017). Several studies have previously observed improvements in reading after noise abatement procedures took place in schools

(Cohen et al., 1981, Eagan et al., 2004, Seabi et al., 2015) which, given the other potential benefits for physical health, provides a mandate for better noise insulation in schools, particularly in urban areas or areas proximal to transport and industry. Exposure of the world's population to noise is increasing in prevalence and severity, and if this negatively impacts cognitive abilities and mental health in the next generation, this has knock-on implications for socioeconomic conditions, health outcomes, and overall wellbeing in a global context (Batty et al., 2007, Firkowska-Mankiewicz, 2011). Therefore, these results suggest that policies mitigating such exposures could have a range of benefits for human health and wellbeing, but most of all, policy should promote and support further good quality research in this area.

However, the quality of evidence was generally low or very low. The most common reasons for initially rating evidence as low was cross-sectional design, and downgrading occurred most frequently for inconsistency between study findings, a small number of studies, or risk of bias concerns. Risk of bias concerns were mostly due to sample attrition or exclusion, inadequate controlling for confounding, and unrepresentative population sampling. Usually, the method of exposure and outcome assessment were considered reasonable and did not often lead to downgrading, though parent-reported mental health measures were overused. Selective reporting did not emerge as a major concern. Most studies did not control for air pollution or other environmental variables, and many compared high noise schools to low noise schools, despite the fact these schools undoubtedly differ in ways other than noise level.

3.4.2 Future research

A far larger number of papers have been identified in other age groups, particularly children and older adults (Zare Sakhvidi et al., 2018a, Thompson et al., 2022b). However, mechanisms like stress and sleep could impact cognition and mental health in all age groups (Lupien et al., 2009, Wilckens et al., 2014), and perhaps especially in teenagers who are still psychologically developing and are considered especially vulnerable for this reason. There is a need for further good quality noise research in adolescents and young adults. In the studies that have been conducted, there were great heterogeneities in methods and findings. As with air pollution research, studies should endeavour to either evaluate an entirely new subject area or replicate methodology.

There were far more studies from the USA (N=8) and Europe (N=16), than Asia (3), Central and South America (1), Africa (2) or Australasia (0). The effect of noise on cognition may not be homogenous across countries (as they could be modified by sociocultural context, air pollution levels, temperature, age of population, nature of noise, times when exposure is highest, etc.) and so more research is needed in these understudied regions. Studies investigating observational associations between noise and cognition should also combine this with mechanistic investigations in the same population - measuring indicators of response (e.g. cortisol, changes in patterns of activity or structure of the brain, questionnaire data on mental health or sleep quality), which alludes to potential explanations, potential interventions, and enhances causal inference. Two studies explored effect mediation, one reporting mediation of mental health effects by neighbourhood restorative quality, physical activity, and sleep disturbance (Dzhambov, 2017), and one reporting mediation of cognitive effects by BMI (Clark et al, 2016a). One study looked at both mental health and cognition together (Haines et al., 2001a).

Future research should more clearly disentangle the impacts of noise from air pollution; noise and air pollution covary, presenting the risk of residual confounding of one to the other. Four papers from two studies (Clark et al., 2012, Tzivian et al., 2016b, Tzivian et al., 2016b; Van Kempen et al., 2012) controlled for air pollution, with Clark et al. (2012) observing no evidence of confounding but Tzivian et al. (2016a, 2016b) observing an attenuation of noise's effect when controlling for air pollution, and Van Kempen (2012) observing a relationship between school transportation noise and errors on the switching attention test after adjusting for NO_2, as well as observing effect modification where children in high noise areas were more vulnerable to the effects of pollution on attention and reaction time. Another study (Tzivian et al., 2017) also looked at effect modification by stratifying the sample into groups of high/low exposure, finding that the relationship between air pollution and general cognition was stronger when noise exposure was high, and that noise was only associated with general cognition in the context of high air pollution exposure. Future research should also consider the roles of different exposure windows (night, day, long-term, recent) and locations (comparing home, school, work, etc.). Just two studies evaluated exposure in both home and school locations (Matheson et al., 2010, Van Kempen et al., 20102012).

3.4.3 Strengths and limitations

This review has several strengths, it is thorough and comprehensive. Risk of bias and quality of evidence was assessed with transparent, replicable and customary methodology. Possibly due to the variability in how these subject matters have been studied, this was the first meta-analysis on the subject (Thompson et al., 2022). However, most evidence could not be quantitatively synthesised, and much evidence had to be downgraded in certainty.

Evidence was evaluated by noise type, following other reviews on the subject (Clark et al., 2018; Sakhvidi et al., 2018). It is plausible that some mechanisms of impact are more source-specific than others, e.g. if airplane and helicopter noise were more annoying than other sources (Miller et al., 2021) but general physiological stress responses were mainly affected by volume (Eriksson et al., 2018). Although different noise sources were combined in meta-analysis, the GRADE assessed certainty of evidence and qualitative synthesis was separated by noise type. Therefore, it is possible that a more general approach evaluating across noise sources may have been more substantial and conclusive. Additionally, the body of literature up to 2015/ 2018 was identified by prior reviews (Clark & Paunovic 2018; Zare Sadhviki et al., 2018), so if they did not capture all the literature this will be reflected in this synthesis of the literature. Further, the meta-analyses cut across multiple outcome metrics, which is not ideal as it only demonstrates the trends of effects rather than precisely estimating the exposure-response relationship. If standardised methodology is employed in future, more precise estimation of effects will be possible

The remaining limitations are shared by the systematic review of chapter 2; specifically, the choice of search engine potentially missing evidence from LMICs, the possibility for human error in judgement of study selection or quality, and the exclusion of neurodevelopmental and neurological disorders.

3.4.4 Concluding comments and implications for wider project

Detrimental trends and similar gaps in the literature were apparent for the noise literature as were observed for the air pollution literature, though the evidence base for noise exposure is smaller and the strength of evidence weaker. The evidence was strongest for attentional skills and internalising symptoms being related to noise exposure. The evidence was tentatively

supportive of general associations between environmental noise pollution and worsened psychological outcomes, but adolescence in particular has not been sufficiently investigated. In terms of the PhD, this reinforces the importance of furthering our understanding of these associations in adolescent groups. Further, most studies did not control for air pollution or see if air pollution exposure moderated associations despite its association with noise exposure, so this is an important aspect of these relationships to model in the forthcoming epidemiological analyses. Additionally, existing research has not richly explored whether sociodemographic factors moderate exposure-outcome associations, nor whether cognition and mental health may interact or mediate each other with respect to how they are each impacted by noise, which again is an important aspect of these relationships to explore in this project. Finally, studies tended to focus on home or school exposure and did not usually compare different windows or contexts of exposure, so it is important to delineate whether exposure to noise at specific times or places could have a differential impact. This is also helpful for understanding mechanisms of impact; for example, if night-time noise were especially impactful this may be due to effects on sleep, whereas if daytime noise were especially impactful this could be due to effects on learning. Therefore, the epidemiological analyses in chapters 6 and 7 will compare noise at different times and over different exposure periods. Daytime noise will largely index the school setting, and night-time/evening noise will index home exposure.

Chapter 4: Qualitative interview study on adolescents' environmental perspectives

4.1 Introduction

There are several motivations behind the qualitative element of this thesis. Firstly, gathering young people's thoughts and feelings about environmental problems may inform contemporary thinking as to how we interpret the psychological effects of environmental exposures mechanistically. Secondly, these experiences will elucidate the modelling choices that are made in the epidemiological analysis, by bringing insights as to how young people interact with and are affected by their environments (e.g., choices of covariates, exposure windows, moderation and mediation analyses, random effects). Finally, to make meaningful policy recommendations that truly advocate for this age group requires insights into what issues matter to them and what they want us to do about it, rather than making assumptions that we know what is best for them.

On the research side, it was decided to involve young people from the public in guiding the research and analysis (public involvement/PPI). This was to ensure that the research methods were effective at garnering helpful, honest, comprehensive feedback. It was also employed to make sure the methods were agreeable and acceptable for working with teenagers ethically. These two considerations are based upon the principle that teenagers are experts by experience, who know more about what it is like to be an adolescent, and therefore how to work with them, than researchers do (Caron-Flinterman et al., 2005). Further, given that the research is about young people and in principle for their benefit, it is more democratic to involve them in the decision making, operational side; to conduct the research with them rather than simply upon them (Gradinger et al., 2015).

Local environmental issues besides climate change such as greenspace, air pollution, and noise are also thought to have direct mental health impacts(Barton and Rogerson, 2017, Braithwaite et al., 2019a, Clark et al., 2020). In order to understand and contextualise these quantified exposure effects, it is important to understand young people's perspectives on how their local physical environments affect them in different contexts (for example, whether adolescents are aware of and worried about air quality in their area). Further, their attitudes to environmental issues in general are important to understand how they relate to and may be affected by their environments, and what policy and practice recommendations are most likely to be beneficial to and supported by young people.

Therefore, the aim of this chapter was to conduct qualitative interviews with UK adolescents to explore their thoughts and feelings about the environment, understand their experiences of contextual environmental issues, and inform the wider project by introducing an experience-led perspective. Some of the following material has been adapted from an earlier publication by the candidate (Thompson et al., 2022a).

4.2 Methods

This qualitative study was guided by the recommended consolidated criteria for reporting qualitative research (COREQ) checklist (Appendix VI).

4.2.1 Patient and Public Involvement

A Young People's Advisory Group (YPAG) of twelve adolescents (ranging in age from 14-18 years-old) was recruited using professional and personal networks, and they were paid for their time according to NIHR INVOLVE guidelines. Initial project planning was guided by a 3-hour in-person research design workshop with this group, and the information sheet, consent form and recruitment adverts were reviewed and edited by two of its members. From the YPAG, TH and ZK applied to be involved in more depth for the remaining research cycle

as co-researchers. The interview questions and protocol were co-produced with TH and ZK in a 2-hour online workshop. TH, ZK and RT analysed the data across two 2-hour online workshops with independent working in-between and reflexive discussions, according to previous methodology developed for involving young people in qualitative thematic analysis (Dewa et al., 2019, Dewa et al., 2020). This research was approved by the Imperial's Science Engineering Technology Research Ethics Committee (SETREC reference 20IC5842).

4.2.2 Participants

Young people were recruited during Spring 2020 by circulating a digital flyer. The advert was posted on the social media accounts of the candidate, their family and friends, on a research opportunity mailing list, and on a number of research related and youth organisation twitter accounts. Although professional and social networks were used, only two participants knew the candidate/ interviewer prior to interview. The flyer stated "*14-19 year-olds wanted to give their thoughts and feelings about environmental problems! How do issues like air pollution affect you?*" and interested participants were invited to contact the interviewing researcher by phone or email. Inclusion criteria were that the participants must be aged 14-19, based in the UK, and English-speaking. Participants who got in touch were sent an information sheet via email outlining the study aims and what to expect from taking part. Written consent and background and demographic information (e.g., age, gender) were then obtained via email, and an interview was scheduled.

4.2.3 Semi-structured interviews

The questions were broad and exploratory, thus allowing young people's perspectives and experiences to guide our understanding of what environmental problems and factors affect

their wellbeing. The interview structure varied depending on flow, but was roughly divided into sections asking about:

1. Pollution and their local environment
2. The environment more broadly and its future
3. Engagement, influences, and information (regarding environmental issues)
4. Responsibility and power (to influence the environment)

Interviews took place via videocall (Skype, Facetime, or WhatsApp) or telephone call between the interviewing researcher (23 year-old female, MSc, PhD candidate) and the participant only. Question type and order were applied flexibly, with follow-up questions or probes used as the conversation warranted (full questions and protocol in Appendix VII). Interviews were recorded using the 'eXtra Voice Recorder Lite App' and transcribed verbatim along with notes of any important additional details. After the interview, the recording was stopped, and participants were given the opportunity for questions and provided with a list of resources for support. Participants were reimbursed for their time with a £20 e-voucher.

4.2.4 Analysis

The underlying theoretical perspective of the analysis was interpretivist, as the aim of the study was to meaningfully capture and interpret the subjective experiences of a group of young people in response to these contextual issues without assuming them to be predictable or fixed (Ezzy, 2013, Holloway, 1997). Therefore, there were no specific hypotheses, and codes and themes were drawn from the data inductively.

It was decided a priori to begin with 12 interviews, conduct the initial coding analysis, and then continue in sets of three interviews until code saturation was reached (Braun and Clarke, 2006, Francis et al., 2010, Guest et al., 2006). Braun and Clarke's six stages of thematic analysis were applied to the data. Initially, the researcher (RT) and co-researchers (ZK, TH) discussed the aims of the project and analysis and familiarised themselves with the first transcript. They then generated initial codes using Microsoft Word's Highlighting and Comment functions and discussed the first transcript's codes in a two-hour virtual meeting. The three researchers then independently coded the remaining transcripts (using Word's Highlighting and Comments functions) according to their own iterative coding frameworks (RT all transcripts, ZK 8 transcripts, TH 7 transcripts). Codes were then triangulated to produce a full list. In another two-hour virtual meeting between the three researchers, the full code list was discussed, edited, and organised into an initial thematic framework and map. RT, ZK, and TH then checked all the data against each theme for referential adequacy. The referential adequacy and structure of the themes, codes, and data were reviewed by an independent researcher (peer debriefing, HLF). Feedback from the group (HLF, RT, ZK) was incorporated to finalise the thematic map and code structure, for which several themes were rephrased, or their structure re-ordered (e.g., renaming 'the future' as 'perceptions of the future').

4.3 Results

Fifteen participants aged 14-18 years from a range of demographic backgrounds took part in the interviews (Table 5). Two-thirds were female, two-thirds were from urban areas, 80% were white, and 60% went to comprehensive schools. Their experience of mental health and environmental engagement or activism also varied considerably (Table 5). Twelve interviews took place initially, which were then coded, and an additional three interviews were

conducted to reach data saturation (no new codes arose in interviews 11-15). The mean interview length was 25.02 minutes (range 16.36m - 36.18m).

Table 5. Participant background and demographic information

Demographic characteristic	**Coding**	**N (%)**
Age	14 years	2 (13.3)
	15 years	3 (20.0)
	16 years	5 (33.3)
	17 years	2 (13.3)
	18 years	3 (20.0)
Gender	Female	10 (66.7)
	Male	4 (26.7)
	Non-binary	1 (6.7)
Ethnicity	White	12 (80.0)
	Black	2 (13.3)
	Declined to answer	1 (0.7)
School type	Private	6 (40.0)
	Comprehensive	9 (60.0)
Location	Rural	5 (33.3)
	Urban	10 (66.7)
Self-reported environmental campaigning*	Yes	8 (53.3)
	No	7 (46.7)
Self-reported prior mental health difficulties	Present	2 (13.3)
	Absent	12 (80.0)
	Declined to answer	1 (6.7)

* Mostly attending the climate strikes

4.3.1 Environmental Themes

There were 52 codes in the final framework, resulting in 6 themes and 11 subthemes (see Figure 8). The themes are described with exemplar quotes below. All codes and their prevalence can be found in Appendix VIII.

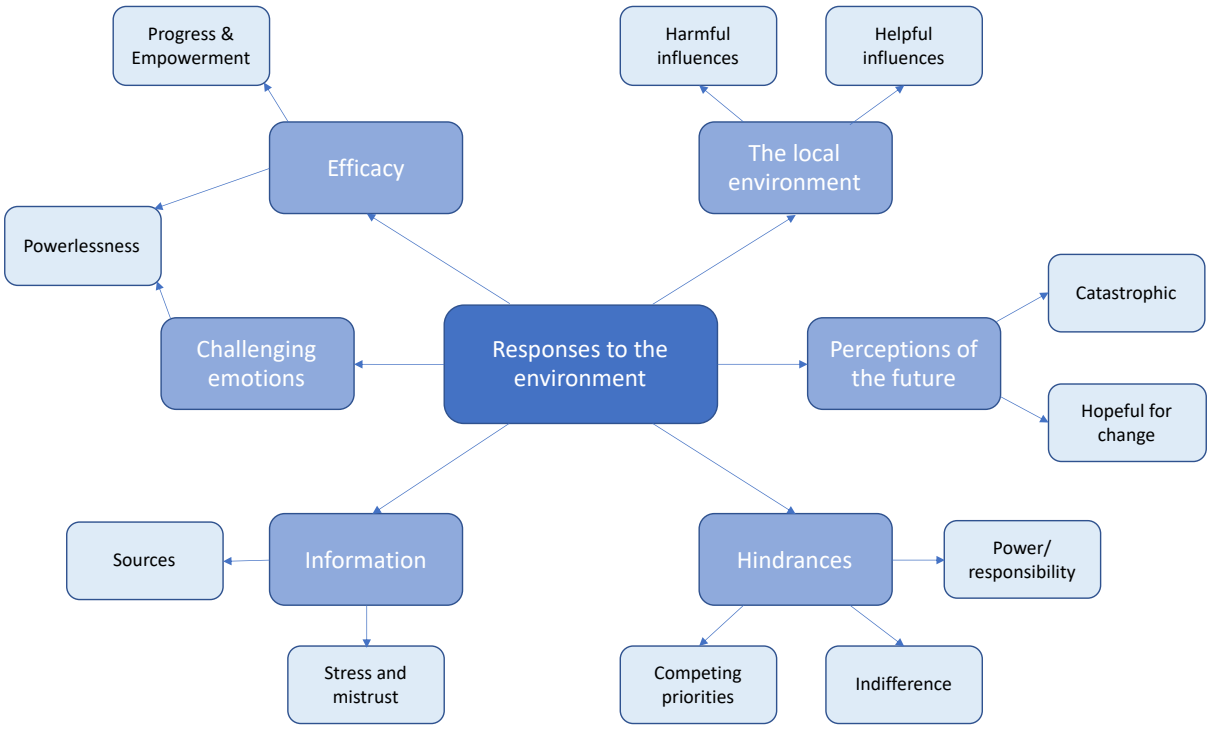


Figure 8. Co-produced thematic map depicting adolescents' responses to the environment

Theme 1: Impact of the local environment

Harmful influences

Factors described as having a harmful influence included air pollution; most talked about its impact on health and mortality and participants used many general negative terms (e.g., "bad", "concerning"). Noise was reported to have a negative impact on wellbeing; distraction and stress or anxiety were the most widely discussed effects of noise, but annoyance, feeling trapped, and impact on sleep and physical health were also mentioned. In addition,

overcrowding, traffic, litter and uncleanliness, were widely reported to have a harmful impact, because they both increase pollution and noise and are themselves stressful stimuli. Some aspects of the local environment such as litter, pollution and noise were talked about negatively in terms of their non-personal effects on the climate and on wildlife. A common thread to the sources and effects of harmful influences seemed to be a sense of feeling overwhelmed or bombarded by numerous features of the urban environment.

> *"I think it affects quite a lot of my thoughts, like sometimes when I'm walking it's just sort of, in a city, I get kind of paranoid about pollution and things and what I'm breathing"* (Participant #8, Female(F), age 14)

> *"It's too noisy, so I feel like there's nowhere to go to like relax" (Participant* #10, F, 14)

Helpful influences

Adolescents reported that influences in their local environment could be helpful to their wellbeing, in particular greenspace, nature, and fresh air. This created an overarching perspective of the natural environment as soothing in contrast to urban experiences. Accessibility to places, activities and other people was the only feature associated with the urban environment that was discussed fondly.

> *"…being able to go outside, I think that has quite a big impact on young people's mental health especially…it's nice to have parks and greenspaces to go to, just to get out of the house and not just walk around the streets all the time but walk in the park and see nature and things like that." (Participant* #15, F, 17)

Light, safety, and personal space were all described as having effects (too much light, feeling unsafe, or not having personal space as harmful and the contrary as beneficial), but these elements of the local environment were not as strongly emphasised as other factors such as greenspace and traffic.

> *"I think having lots of space, is very nice, and particularly now, but having the ability to go into a garden and spend time and you can't, you can't see people next to you, if they're in their garden like you have your own privacy" (Participant* #7, F, 15)

Theme 2: Efficacy

Young people talked about their ability to affect their environment and address their environmental concerns. This was largely talked about on a global scale (e.g., related to climate change) but also somewhat on the local level.

Progress and empowerment

Many of the young people talked about taking environmental action and this feeling good, usually describing their impact as collectively rather than individually driven and recognising group efficacy as more relevant to these issues than individual action. Another common characteristic of these discussions was references to adolescent activism such as the climate strikes, and this adolescent group described their generation as especially interested in, engaged with, and passionate about environmental issues:

> *"It makes me feel a bit more hopeful, you know if I've taken action as an individual, hopefully some other people out there are doing the same and as a collective, it will have an impact." (*#12, M, 16)

> *"Talking from my perspective, like my friends, I know most of my friends are interested in the environment"* (#13, F, 18)

Powerlessness

In contrast, a very palpable perspective which emerged was widely expressed feelings of powerlessness, and many interviewees expressed fears that climate change is out of control, that it was too late to avert a climate crisis, and that they did not know what to do to prevent it. Sometimes this lack of control was attributed to their being one individual, but often participants said that their age and the associated lack of power was further restricting their ability to exert an influence.

> *" I can feel a bit stuck a bit useless I'm like, why isn't this happening…I don't know what I can do, it feels like, I feel a bit lost in just not being able to do much because I'm just one person, I dunno, it feels like it's out of control." (Participant* #11, F, 15)

Theme 3: Challenging emotions

This theme captures the extensive challenging emotions that were expressed. The more welcomed emotions mentioned (passion, solidarity, and hope) were less numerous and therefore were conceptualised under the themes of *efficacy* and *perceptions of the future*. Positive feelings tended to be elicited by specific questions about pro-environmental action or

how participants viewed the future, whereas more open questions about environmental issues in general elicited negative responses.

The participants expressed anxiety and sadness about the environment, its present state, our treatment of it, and where they anticipated it going. Feelings of guilt about not doing enough or their own privilege in not yet being directly affected were also discussed.

> *"It's always at the back of your mind, lingering…There is a big little voice in the back of your head going, the world is dying, are you gonna make it past 50?" (*#1, F, 16*)*

> *"I'd say that I don't go out of my way to learn more things about it because I dunno, it's quite depressing in a way." (#3,* Male (M), 16*)*

Furthermore, many young people expressed frustration, anger and annoyance at societal structures or other people.

> *"I would say I feel quite frustrated that nothing, like, not that nothing is being done but I don't see as much progress as I would think there would have been, especially like people in power you would think that they would take the environmental problems seriously and try and tackle it so it's quite frustrating that there's a lack of progress or a lack of initiative taken place." (*#14, F, 16)

Theme 4: Information

The interviews touched upon the sources young people get their information about the environment from, and their main influences in terms of their environmental knowledge and opinions.

Sources

The main influences and sources of information young people discussed were social media (73.3%), the news (40.0%), friends and family (93.3%), and school (66.7%).

> *"...also my friends do similar things so when we talk about it we get the information, we all get information from various sources and then we talk about it so I get it from that as well." (*#7, F, 16)

> *"either school or Instagram, I think. Sometimes the news, but yeah it depends on what I'm doing that day, but usually school I think and media" (*#14, F, 16)

Responses

This subtheme is about responses to information about environmental issues. The most salient perspective emerging from this was a mistrust of the media and media/news stress. The news was generally not perceived as constructive but rather as stressful, negative, and catastrophizing, without offering solutions, perspective, or nuance. Some young people talked about avoiding the news, or even general content or discussion about environmental issues, for these reasons.

"you know what the media are like, they're fear mongers, a lot of media, it's very hard to get media that truly shows you the big picture and shows you the big picture as what it is, not as you know, an overdramatised scary model," (#5, M, 16)

"I stay away from the news because it's so negative, it's so depressing." (#1, F, 16*)*

Theme 5: Hindrances

Young people reported that not enough was being done to take care of the environment, especially with respect to climate change, and this theme encompasses the factors they identified as hindering this progress.

Indifference

This subtheme encompasses how participants identified people's indifference as a cause of environmental problems. This indifference was attributed to a lack of awareness or denial, people's psychological avoidance of the issue, and because many people in the UK are not affected at present. Often this indifference was discussed in the context of generational differences, with participants suggesting that younger people are especially engaged because they expect these issues to affect them in their lifetime. Education was highly cited as an important solution.

"I think where a lot of people feel relaxed is when they know that they're not going to be affected in the future whereas for a lot of young people it's, we will be affected, we will live through this. So that's where a lot of the worry I think comes from." (#11, F, 15)

Power/responsibility

This subtheme is about which people or organisations the young people held responsible for our environments, and which people or organisations they saw as holding the power to bring about change (these essentially went hand in hand). The strongest effect was that the government is capable of affecting and culpable for our environmental problems, though there was also blame geared towards big companies and older generations for damaging our local and global surroundings:

> *"Frustration with people in power, like people in government, politicians who aren't, it feels like sometimes they're not listening... And also frustration with like bigger companies like global players in that sense, who, it just, I also think it really wouldn't kill them to just make the change" (*#11, F, 15)

> *" I think we feel like we've kind of, we have to try and fix the mistakes that have been made in the generations above us, so we're kind of trying to do what we can I guess."* (#14)

Other/competing priorities

A final reason for a lack of progress on environmental problems mentioned by the young people was how other issues can take priority. On a societal level, competing policy objectives and the pursuit of profit or a strong economy were identified as reasons why governments or companies might not be eco-friendly. On the level of the individual, the young people spoke about how people do not always have the time or resources to live in an eco-friendly way, and how people may not want to give up their present lifestyles.

"It's not good enough, it's not that the people in charge don't have access to do it, they just don't because it's more efficient, it's more profitable and it's easier." (#4, Non-binary, 17)

"Unfortunately, a lot of the environmentally friendly stuff is the more expensive stuff." (#5, M, 16)

Theme 6: Perceptions of the future

How participants talked about the future was thematically organised into positive and negative perceptions.

Hopeful for change

When asked about their expectations, there were some positive expectations of increased awareness and action, and that a crisis could be preventable:

"*More pressure to be applied on the government to take action I think, from protests as well, is understanding that we won't sit and watch the world just deteriorate, I guess. I expect people to understand a lot more" (*#12, M, 16)

"I know eventually we will get there, it might take time, in a way I reckon that if things do get worse, it's not necessarily a good thing but it is almost a good thing because it will kick the government into gear… I'm fairly certain, it might take time, but I reckon it will get better, they just, we need to wake up almost" (#2, M, 16)

When asked about their hopes for the future, adolescents wished for increased awareness, increased action, and a cleaner more sustainable world for future generations:

> *"…a lot more trees, more trees being planted rather than so many being cut down, like people using charity shops more and second-hand things more, using local farmers stuff like that for food so they don't have to transport it miles and miles, and then more places, like clean water, litter cleaned up every week or two, just a nice place to be really." (*#10, F, 14)

> *"I want to see a just world where everyone's equal and that countries are not gonna, are not being exploited for their materials and we've found alternatives. There is already alternatives we're just not using them to their greatest extent, like wind power, solar power, wave power, we're just not using them, and I think those resources have to be used and we have to build upon them and create a just transition for everyone," (*#9, F, 18)

Catastrophic

Under the subtheme of catastrophic perceptions of the future, some participants discussed urgency or said that we are running out of time, and some indicated that the world is dying or becoming uninhabitable. When discussing the future of the world, there was a general sense of impending doom or catastrophe.

> *"There's a sense that the world is starting to become, not necessarily dying, but dying for us. As in we won't be able to live in it for much longer… it's kind of like ah my adulthood might not be a thing. Or, me being an adult might be in a world where we*

are getting constant weather issues, constant floods, where you have to wear a mask outside because you can't breathe" (#1, F, 16)

"I don't think you can reverse the amount of damage that's already been done." (#3, M, 18)

In terms of expectations, all but one participant had negative expectations about the future of our environment. These tended to take a catastrophic narrative that things will get worse and have serious consequences before they could stop it. Those that did expect positive change generally felt things would have to get worse beforehand:

" It's just full of litter, there'll be nowhere nice to go to, like ponds just full of water that's like polluted, got rubbish in, I think, eventually we won't have sunny days it'll just be like thick smoke clouds, just not a nice place to be." (#10, F, 14)

"I feel like the politicians and the people have got to wake up and see that something is happening when we do start to feel huge effects from it, but I don't think that will happen before effects are felt and before there is some damage done" (#7, F, 15)

4.3.2 Other results

Although it does not contribute to the thematic map which concerns environmental issues, other factors in young people's lives which contribute to their mental health were discussed. These were coded and organised thematically, giving rise to the following key areas: school/educational stress, communication and social support, health and fitness, awareness/stigma, mental healthcare, and social media. School and education were talked

about extensively as a source of stress; regarding pressure to achieve grades, fear of exams, levels of homework, and university applications, there was a real sense of being overwhelmed. Teachers were described as not being understanding and the pressures of the educational system were seen as actively harmful to young people's mental health. Having higher levels of social support, open communication, and awareness amongst peers and from adults were seen as positive influences. Better health and fitness and accessible high quality mental health care (e.g., counselling) were seen as promoting better mental health, and social media was seen as a negative influence, particularly in terms of comparison of self to others and body image.

4.4 Discussion

4.4.1 Summary

In a convenience sample of UK adolescents, this study corroborates previous findings from journalism (Bodkin, 2019, Taylor, 2019) and academic research from a range of countries (Connell et al., 1999, Hokka et al., 1999, Strife, 2012, Tucci et al., 2007, Hickman et al., 2021, Kelly, 2017) which have reported high levels of youth engagement with environmental issues, coupled with fear, anxiety, anger, and sadness. As has been observed previously in children and young adults, participants had grim expectations for the future of the world (Strife, 2012, Tucci et al., 2007). Crucially, the majority of the sample did not report any prior mental health problems. This supports the idea that 'negative responses' or coping mechanisms with regards to environmental problems should not always be pathologised and could be considered a rational anxiety or a rational anger (Lawton, 2019, Verplanken and Roy, 2013) which is even constructive for bringing about change when motivating action. When coupled with informed and hopeful action, eco-anxiety may be considered an adaptive response to a crisis (Pihkala, 2020a, Verplanken et al., 2020).

Participants reported a belief that their generation is more passionate about environmental issues than older generations but, as has been reported in previous studies, most felt powerless themselves to have a real impact (Connell et al., 1999, Kelly, 2017). The young people's perceptions of why enough action is not being taken are consistent with some previously identified factors in climate inaction; limited cognition (lack of awareness, denial), ideologies (e.g. capitalism), sunk costs (economy, profit, lifestyle), perceived risk (low perceived personal risk of climate change relative to risks associated with action), and limited behaviour (Gifford, 2011). However, these factors were largely attributed to middle-to-older aged adults and adults with power (those in government and big business), and not described as hindering young people's motivation to act to the same extent. This perception of a generational gap, associated with feelings of anger and betrayal, has been observed in prior research (Jones and Davison, 2021, Strife, 2012). When describing their personal reasons for not doing more, this was attributed to not knowing what to do, and not having the power to do everything they would want to.

However, participants also commented on progress being made and discussed the benefits of taking action for their sense of hope and their sense of personal wellbeing. This suggests a potential dual benefit of environmental action for the environment and mental health. This group of adolescents recognised the role of the collective in generating positive outcomes for the planet, presenting higher group-efficacy than self-efficacy (Fritsche and Masson, 2021), and described how observing or participating in the actions of a group enhanced their sense of hope, community, and wellbeing. This 'co-benefit' of pro-environmental behaviours for wellbeing has been observed in prior studies (Chukwuorji et al., 2017, Kaida and Kaida, 2016). This is in-keeping with Ojala's observations (Ojala, 2012b, Ojala, 2013) of adolescent

responses to climate change and corresponding conceptualisation of constructive hope and meaning-focussed coping (emphasising the capacity for pro-environmental action to influence environmental outcomes) as being the most beneficial for wellbeing and environmental action, compared to denial or a lack of hope.

Stronger levels of concern and powerlessness in relation to global issues over local issues were observed, which is consistent with prior research (Uzzell, 2000). However, participants' personal environments and spaces were also found to influence their wellbeing, particularly their level of stress and comfort, and how and where they tended to spend their time. Research that focusses upon how people interact with and are influenced (positively and negatively) by their built and natural environments has a longstanding tradition in environmental psychology (Bonnes et al., 2018, Gifford, 2014). Air pollution, water pollution, and biodiversity were found to be of concern, as a study of a previous generation of teenagers found (Hokka et al., 1999), as well as light, uncleanliness and litter, overcrowding, greenspace, fresh air, personal space, and safety. These young people seemed to associate stress with urban spaces. Aspects of the local environment which exerted a positive influence were greenspace, nature, fresh air, and accessibility to other people, places and activities, which suggests both urban and rural environments have some benefits for young people. The soothing and beneficial effects of greenspace, time in nature, and nature-connectedness are well-documented (Barton and Rogerson, 2017, Piccininni et al., 2018, Pritchard et al., 2020).

Research suggests the bonds and attachments youth and other people feel to environments (place-attachments) are associated with stronger intentions to preserve them and higher levels of pro-environmental behaviours (Stedman, 2002, Vaske and Kobrin, 2001). It has been suggested that this could be leveraged in environmental education to influence pro-

environmental practices (Kudryavtsev et al., 2012, Walker and Chapman, 2003). Given that younger people are often associated with a preference for cities, accessibility, and activity, it was interesting that both the urban and rural youth engaged in the same narrative of appreciating the healing aspects of nature and expressing concerns surrounding the complex and lurid nature of urban life, although this positive valuation and respect for nature has been observed in teenagers and young adults in other research (Lekies et al., 2015, Soga et al., 2016), and this may not hold true for all UK teenagers not represented in this sample.

4.4.2 Implications for research and practice

This research has implications for mental health research and practice, environmental policy, and methodologies for working with young people. Regarding mental health research, future research should quantify the prevalence, nature, and severity of climate change's indirect psychological impacts through large scale studies, and explore what factors could protect or alleviate environmental distress (Thoma et al., 2021). Several preprints in this area suggest this research is already emerging (Hogg et al., 2021, Wullenkord et al., 2021, Hickman et al., 2021) and the recently developed climate change anxiety scale is a valuable resource for researchers looking to address this (Clayton and Karazsia, 2020). These interviews provide preliminary insights into these factors; on the local level, the young people found time spent in clean and green spaces beneficial, and many talked about how trying to 'make a difference' was beneficial for a more optimistic outlook. It should be cautioned, however, that the relationship between action and wellbeing is unlikely to be linear, with potentially high risk of burnout for climate activists (Hoggett and Randall, 2018). Clinically, mental health practitioners and other professionals working with young people should be aware of how environmental problems might figure in their world view and impact their wellbeing, and professional bodies should produce research and guidance to support their members, e.g., recent work by the Royal College of Psychiatrists (2020).

However, environmental degradation and climate change are higher-level problems and threats to health and mental health which reducing individual distress will not solve. The young people in these interviews were informed, passionate and opinionated, but felt fundamentally disempowered and had a mistrust of, and frustration towards, government and those with power. Policy-makers and politicians should take steps to listen to young people and their grievances, aggregate their views, amplify their voices, and give them practical influence, whether it be in terms of local town planning and maintenance, government policy towards energy and transport, or international environmental strategies (Kara, 2007, Thew et al., 2021). This research also implies that supporting organisations that encourage collaborations between environmentally engaged young people and include young people in environmental projects would benefit adolescent wellbeing and the environment.

Additionally, social media was the key resource used by participants to gather information, which should be considered when communicating with adolescents about environmental information, campaigns, interventions, or projects. The importance of friends and peers as a source of information and influence suggests that youth gatekeepers and ambassadors are valuable ways of working with this age group.

The young people's experience of climate-related information, especially the news, was that it was lacking nuance, positivity, and practical advice, leading to a sense of powerlessness and 'end of the world', in turn leading to stress, sadness, and avoidance. In keeping with previous research, this suggests that news and information providers and educators have a responsibility to provide accurate information which shows the links between present events and future outcomes (Hicks, 2012, Hicks, 2013). However, some accurate information may

be intrinsically negative in what it predicts, meaning the provision of factual information that is non-distressing may not always be possible. This requires the cooperation of educators, activists, communicators, and support services to provide emotion-sensitive environmental material whilst building youth resilience to difficult information, thereby generating a sense of understanding and 'constructive hope' which encourages environmental behaviour without provoking unconstructive or excessive anxiety and distress (Ojala, 2015, Pihkala, 2020b, Moser, 2014).

4.4.3 Strengths and limitations

This study has many strengths. Adolescents were involved throughout the project, from initial brainstorming, to designing the interviews, to analysing and interpreting results, according to practices developed by NIHR INVOLVE and Imperial College's Patient Experience Research Centre (PERC). Their insights greatly affected the course of the study, improving the experience of participants, and making findings more relevant and insightful. Crucially, themes, codes, and meaning were drawn out by young people, adding validity to the perspective and experience constructed from the data. Their involvement in designing the interview protocol for greeting, explaining, question phrasing, and managing distress, will have helped to reduce the power dynamic inherent to an interview situation, as will the candidate's relatively young age at the time (23 years-old) and prior experience working with adolescents. Peer debriefing, reflexivity, and triangulation added further rigour to the process of interpreting data. Additionally, participants were from mixed socioeconomic backgrounds and urban/rural residency, and therefore a variety of adolescent voices were represented. This novel piece of research adds knowledge and understanding to a relatively underexplored but emergent field.

There are also limitations. The project took place during the COVID-19 pandemic, which will have influenced where participants spent their time and could have affected their relationship to their environment. The interviews were conducted remotely, which could have produced different results to a face-to-face interview. Additionally, discussion of these issues may have brought them to mind uncharacteristically and thus elicited a reaction which, although negative, is not typical of their day-to-day state of mind. Although measures were taken to uphold objectivity, some researcher and co-researcher bias cannot be ruled out, notably due to their female gender and their own pro-environmental views and experiences. Additionally, the views of adolescents from countries other than the UK are not represented here, the sample was not very ethnically diverse, and two-thirds of participants were female. Although participants varied from very active on environmental issues to entirely inactive, this sample did feel confident enough to respond to the advert, so they may have been especially environmentally aware and/or engaged. The recruitment strategy may also have somewhat biased the sample, as personal networks were used along with professional networks oriented towards young people interested in research. Additionally, the interviews varied in length, so some participants were more forthcoming than others which may be related to their knowledge, confidence, or engagement. Finally, the interdisciplinary nature of this topic means that it is difficult to consult all relevant resources when contextualising new research.

Reflexive observations

Although the research was not explicitly driven by particular hypotheses, the researchers and co-researchers will have brought prior impressions of good and bad (e.g., climate change and environmental issues as negative) and knowledge of prior theory (e.g., concepts of direct and indirect as distinct), which will have come together in designing the study (e.g., separating

global and local in interview questions), interviewing participants (e.g., language and tone, choice of questions to follow up on), and extracting this set of themes (e.g., anxiety and anger may have been anticipated and identified). However, whilst different approaches will have yielded superficially different responses, we believe the use of multiple coders (including other young people), peer debriefing, triangulation, and the salience of a consistent narrative across interviews means that the key themes are a valid construction of these young people's experiences and that this should be shared.

4.4.4 Implications for wider project

The experiences of these young people and their peers are concerning and deserving of attention. This group described their generation as very environmentally engaged, but also anxious, frustrated, pessimistic, and disempowered. The fact that environmental problems are negatively affecting the psychological wellbeing of young people highlights the need for greater investment in environmental policy and education, and for greater research, understanding, and support for young people suffering in relation to environmental issues, the details of which will be explored further in the Discussion chapter of this thesis.

There are several key takeaways with respect to the epidemiological analysis which follows in the thesis. Interviewees identified several psychosocial mechanisms through which noise could influence young people; they talked about distraction from school and work, stress levels when they are trying to relax, and effects on sleep. That is, there was not one predominant mechanism which was thought to be always most impactful in all settings, but instead a variety of ways in which noise could be impactful depending on the context. This suggests that it is important to delineate different times of day and/or different locations to

unpick which avenues of impact are influential for which outcomes. Therefore, different time periods of exposure (day, night, evening) will be compared in the epidemiological analysis. Although one participant expressed substantial mental health concerns about air quality, describing anxiety and paranoia around breathing in air pollution, most were more concerned about the effects on climate change and worry about their own exposure to air pollution was not a major concern. Therefore, this is not likely to be a major pathway through which it impacts mental health or explain the associations that have been observed in prior literature. However, the general quality of their environments, with respect to cleanliness, levels of industrialisation, traffic and population density, greenspace and nature, and litter, was described as having an impact on mental wellbeing, and these factors are likely to covary with the levels of air pollution and noise. Therefore, a random effect of neighbourhood/ local area, to capture these general variations between places, will be employed as a level of adjustment in modelling.

Having a garden and nice outdoor space to make use of was described as beneficial for mental health but could also indirectly increase a person's exposure to certain sources of pollution and noise (due to more time being spent outside). Yet, greenspace does ameliorate air and noise pollution levels. This implies that there could be differences in the effects of noise and air pollution on psychological outcomes between groups who are from different socioeconomic backgrounds and who are based in different places. This reinforces the potential benefits of including a random effect of neighbourhood, as well as the significance of exploring effect modification by sociodemographic factors within this thesis.

Chapter 5: Methodology for epidemiological analyses

5.1 Introduction

5.1.1 Background

Although many environmental factors have the potential to affect adolescent mental health and psychological wellbeing (as shown in chapter 4), environmental noise and air pollution are major contemporary public health concerns (WHO, 2016, Basner et al., 2014), and their shared causalities present dual opportunities for mitigation. Further, many policies mitigating air and noise pollution (such as changes to transport and industrial activity) have additional benefits for climate change. Therefore, contributions to this evidence base have high potential policy impact due to the co-benefits for climate, and the additional mental health benefits conferred through climate change mitigation.

The aim of this chapter is to specify the methodology that will be used to evaluate associations between exposure to air pollution and noise and psychological outcomes in the Study of Cognition, Adolescents and Mobile phones (SCAMP), a longitudinal London-based adolescent cohort study. SCAMP has collected data on cognitive and mental health outcomes at multiple timepoints since participants were in year 7 (2014-), and collaborators have produced exposure estimates for the traffic noise and air pollution exposure of most participants during the follow-up period. The sample is distributed in and around greater London, representing a diverse sample in terms of their sociodemographics and their exposure to varying levels of exposure and noise. The study has also collected a rich array of health and lifestyle data. Therefore, this dataset presents a unique opportunity to evaluate the effects of longitudinal exposure to both air pollution and noise in relation to both mental health and cognitive outcomes, including changes in outcomes over time.

5.1.2 Contextualisation with rest of thesis and prior research

The plausibility of air pollution and noise in the physical environment having a psychological impact on teenagers, through a combination of physiological and psychosocial mechanisms, was established in Chapter 1. The importance of mental health and cognition as facets of health impacting almost every aspect of human life, meaning that factors with a detrimental effect are of high concern to the field of public health, was also established in Chapter 1. Reviews of prior literature (Chapters 2-3) have provided useful insights which have informed the choices made in the epidemiological analyses in the next 2 chapters. Firstly, prior literature has based exposure assessment most commonly at the home address, followed by the school address; a scarcity of past literature has used a hybrid approach (combining exposure at the home and school environment together), which is more reflective of real-life exposure. Secondly, linear and logistic general linear regression (GLM) models have tended to be applied in past research. Therefore, the main models in the analyses of this chapter utilise these for comparability, though non-linearity was also investigated using Generalised Additive Models (GAMs). Thirdly, the relative neglect of adolescent age groups despite developmental vulnerability strengthens the importance of investigation in this age group.

The qualitative research outlined in Chapter 4, which investigated adolescent experiences of their physical environment, also provided important insights; general neighbourhood quality was impactful upon how young people feel in those spaces, meaning general factors like litter, cleanliness, upkeep, and busyness, and is also likely to covary with air pollution and noise. Therefore, a random effect for neighbourhood was considered in modelling, as well as for school (reflecting sampling differences as well as the quality of this environment). Young people also discussed different ways in which noise could have an impact; on work/study, relaxation, and sleep, therefore it was important to investigate different time periods of

exposure to delineate the mechanisms of impact, and to include school noise exposure in noise estimates.

Prior research has found varying psychological effects in association with air pollution and noise in different studies and populations (as summarised in chapters 2 and 3), but specific sociodemographic factors have not been extensively investigated. This is even though people of different sociodemographic backgrounds are exposed to different levels of air pollution and noise, usually in correspondence to the presence of social disadvantages, which has been shown to interact with disadvantage to produce differential health outcomes (Alvarez et al., 2018, Hicken et al., 2012). I.e., environmental exposures do not only perpetuate existing health inequalities due to disadvantaged groups being exposed to less healthy environments but may exacerbate existing health inequalities by interacting with other stress factors. London has much diversity and effects may be different for different groups, which is important not only to control for but to identify these differences via moderation analyses, thus allowing for public health recommendations to be made.

Further, only a small number of studies have looked at moderation or mediation by other psychological factors. A very small number of studies have investigated whether mental health or cognition mediates the other's relationship to exposure to air pollution and noise (Tallon et al., 2017, Petkus et al., 2021, Ren et al., 2019), and no studies known to the author have looked at whether they moderate the impact on the other. Other important potential mediators identified in Chapters 1 are health factors- atopic diseases and sleep quality- which are likely to be impacted by air pollution or noise and are associated with psychological outcomes. Heterogeneities between prior studies could be due to heterogeneities in methods or population but could also be due to real-world complexities in the associations between

environmental exposures and psychological outcomes. Investigation of moderation and mediation will shed light on the main associations, as well as the general nature of these epidemiological relationships.

5.1.3 Aims and Hypotheses

The aim of these analyses (methodology chapter 5, results chapters 6 and 7) is to investigate the longitudinal associations between exposure to air pollution and noise and psychological (mental health and cognitive) outcomes in the SCAMP cohort, including exploration of plausible moderators and mediators.

It was hypothesised that higher levels of exposure to air pollution and noise would be associated with worse cognitive and mental health outcomes and worse development in these areas (between baseline and follow-up). Specifically, 'worse development' here means they will be associated with less growth in cognitive scores (which can be expected to improve over time), and deterioration in mental health. It was also hypothesised that any adverse effects from air and noise pollution would be greater in students from relatively disadvantaged sociodemographic groups - female students, non-white students, students with below-average IQ, state school students - compared to more advantaged students. Additionally, it was hypothesised that religiosity may be protective against harmful mental health effects, and that high exposure to pollution/ noise would compound the impact of noise/air pollution. Finally, it was hypothesised that mental health or cognition, atopic disease (allergies, eczema, asthma and hayfever), BMI, and sleep quality would mediate associations between air pollution or noise and cognition or mental health.

5.2 Methods

5.2.1 Data

5.2.1.1 Participants

The SCAMP study involves pupils from 39 secondary schools (26 state, 12 independent) (Toledano et al., 2019). To recruit participating schools, eligible schools were identified using Edubase and the 2012 School Census. Schools were eligible that contained pupils in the target age range (11-15), were not special schools, pupil referral units or secure units, were in or around London, had a pupil count of >50 if an independent school or >200 if a state school. 206 schools were contacted and invited to participate, 35 of whom initially agreed, and a further 8 schools were included who approached the study asking to participate (having heard by word of mouth or other communications). Due to logistic or technical issues, four schools dropped out. Parents received information packs and participation consent was opt-out, with entire school years invited to participate (year 7 at baseline, year 9 or 10 at follow-up 1). Pupils could also voluntarily opt out at any time. Baseline and follow-up 1 were conducted in face-to-face timetabled computer sessions in schools during school hours. Due to the COVID pandemic, follow-up 2 was conducted remotely through online surveys, with links shared by teachers or student ambassadors to the study, with students themselves providing affirmative consent based on consent forms and information sheets. The North West-Haydock Research Ethics Committee approved the SCAMP study protocol and subsequent amendments.

Baseline assessments took place at age 11/12 years (N=6905 total, 2014-2016, “BL”), follow-up assessment 1 at age 13/14 years (N= 5425 total, 2016-2018, “F1”) and follow-up 2 at age 15-18 years (N≈ 1500, 2020-2022 “F2”). The sample represented a wide range of ethnic and sociodemographic groups, and was 51.5% female. Only those providing an address for at

least one timepoint were eligible for these analyses, as an address was required to estimate exposure to pollution and/or noise. Sample sizes varied depending on completeness of data for different outcomes, and what the exposure was (air pollution could only be estimated for those within the M25, noise was estimated for all participants providing an address at BL or F1).

5.2.1.2 Exposures

Collaborators in Imperial's Environmental Research Group (ERG) and Leicester University have produced yearly exposure estimates over the period of BL and F1 outcome assessment for this cohort (2013-2018). Table 6 summarises the key details for how exposure was estimated.

For a subsample (N~200) of participants, measurements of air pollution indoors, outdoors, and in transit were taken over 48-72 hours from 2016-2018. AQMesh devices were used to measure ozone and NO_2, PurpleAir and TZOA monitors were used to measure PM. Geolocation and time-activity diaries were used to map the participants movements during the time period. This information was used to estimate exposure in the different micro-environments common in this age group and calibrate the LHEM model appropriately (adolescents).

Exposure for the whole cohort living within the M25 was then estimated using home and school addresses and questionnaire data. For outdoor exposure at home and school addresses, the Community Multiscale Air Quality Modeling System (CMAQ)-urban model was used. Its predicted concentrations perform well against concentrations at measuring sites (NO_2 r = 0.80 for NO_2, 0.72 for $PM_{2.5}$ (Williams et al., 2018)). For indoor exposure at home, school,

and inside vehicles, a mass balance model was used to predict outdoor-indoor air exchange. Infiltration and loss rates were 0.6-0.7 and 0.1-0.3 $hour^{-1}$ for particles, and 0.4 and 0.5-0.8 $hour^{-1}$ for NO_2 (Vu et al., 2022). Additionally, home indoor sources of air pollution were used to estimate exposure from these indoor sources (as opposed to outdoor air pollution exchange) based on student and parent surveys reporting cooking frequency and cooking fuel (gas/electric), and Energy Performance Certificate (EPC) database information about cooking fuel (gas/electric). The London hybrid exposure model was used to estimate hybrid indoor and outdoor exposure at home, school, and in transit based on addresses, route simulation, and questionnaire data (modes of transport to school, living or commuting on busy roads, time spent outdoors, time in green and blue spaces, exercise outside). Altogether, five exposure metrics were produced per participant per pollutant per year: indoor exposure including indoor sources of air pollution, indoor exposure not including indoor sources (infiltration from outside only), outdoor exposure only, total hybrid (indoor and outdoor, home, school and in transit) exposure without indoor sources of air pollution, total hybrid (indoor and outdoor) exposure with indoor sources of air pollution. For each exposure metric, annual average estimated levels of four pollutants were determined; NO_2, $PM_{2.5}$, PM_{10}, (all in $\mu g/m^3$) and Ozone (in ppb), across the 5 years of BL and F1 assessment (2014-2018). Exposure in 2013 was assumed to be the same as 2014 when it was required to estimate exposure 1 year prior to baseline assessment for those participants taking baseline tests in 2014. The main analyses were run with hybrid exposure including indoor sources of air pollution as this was the most comprehensive metric, but the other exposure metrics were run as sensitivity analyses. As most prior research has used outdoor exposure, models were rerun looking at indoor and outdoor exposure effects only, compared to hybrid effects, and reported when the adjusted effects differed markedly in direction and/ or significance from the combined exposure.

To estimate noise exposure, based on home and school addresses and assuming hours 9am-3pm were spent at the school address, annual average road traffic noise exposure was modelled by collaborators at Leicester University for all participants providing at least one home address (school address data were complete) from 2013-2018 using the CNOSSOS-EU model (Kephalopoulos et al., 2012). A UK feasibility study reported good model performance, ranking sites in comparison to measured noise levels with correlations (Spearman's rank) of 0.75 or higher ($p < 0.001$) (Gulliver et al., 2015). This model covers the whole of the UK so was not limited to participants living within the M25. The noise metrics Lden (24 hour A-weighted equivalent noise level/Leq including day, evening and night, with a penalty of +10db for evening and night), Lday (daytime A-weighted Leq from 7am-7pm), Leve (A-weighted Leq from 7pm-11pm), and Lnight (A-weighted Leq from 11pm-7am), and $LAeq_{16}$ (A-weighted Leq from 7am-11pm) were computed. These measures were school-weighted. meaning that school address and home address exposure were combined, with the relative contribution of each weighted according to the proportion of the metric hours which included 9am-3pm (so this only affected estimates Lden and Lday). It was established that only a small proportion of participants were exposed to rail and/or aircraft noise, hence these were not included. Noise models reflected outdoor exposure only.

Table 6. Summary of air pollution and noise exposure models

Model	LHEM model for children	CNOSSOS-EU
N applied to	3530	7555
Data used	Home and school geocodes, within M25 only. LHEM also incorporates survey data to generate time-activity diaries, such as modes of transport to school, living or commuting on busy roads, time spent outdoors, time in green and blue spaces, exercise outside.	Home and school geocode, all available addresses.
Composite models (microenvironments)	LHEM model estimates exposure to air pollution as annual exposure concentrations. Different time-activity diaries generated for school, non-school, and weekend days, made up of different microenvironments. The following models were used to estimate exposure in different microenvironments: *Outdoors:* CMAQ-urban model estimated outdoor air pollution at specific times and coordinates;	N/A

	Applied to home, school, walk or cycle, other outdoor environments *Indoors at home*: EnergyPlus infiltration model (building structure, energy performance) *Indoor at school:* mass-balance model *Transportation indoors:* mass-balance model *Indoor sources (home):* cooking frequency and fuel type (survey/EPC database)	
Exposure settings	Outdoors + indoors	Outdoors only
Exposure locations	Home, school, in transit, other outdoor locations For hybrid exposure, exposure on school days, weekends, and non-school days estimated, then annual average weighted according to proportion of each type of day in a year.	Home + school Schooldays weighted with 9am-3pm exposure at school address and 3pm-9am exposure at home address, non-school days exposure assigned at home address. Annual

		exposure weighted with proportion of school vs non-school days.
Temporal resolution	Annual averages 2014-2018 (2013 imputed with 2014 exposure)	Annual averages 2013-2018
Spatial resolution	20m x 20m	Building level
Estimates produced	$PM_{2.5}$, PM_{10}, NO_2, O_3, annual averages 5 metrics: • Hybrid (home, school, in transit) with indoor sources of air pollution • Hybrid (home, school, in transit) without indoor sources • Indoor (home) without indoor sources • Indoor (home) with indoor sources • Outdoor only (home + school)	A-weighted equivalent noise levels, annual averages 5 metrics: • Lden, 24 hour period with penalty for evening and night noise • Lday, 7am-7pm • Leve, 7pm-11pm • Lnight, 11pm-7am • LAeq16h, 7am-11pm
Estimates used in main analyses	Hybrid with indoor sources, all pollutants	Lden, Lday
Estimates used in sensitivity analyses	Hybrid without indoor sources Indoor without indoor sources Indoor with indoor sources Outdoor only	Leve, Lnight

Supporting literature	Beevers, S. D., Kitwiroon, N., Williams, M. L., Kelly, F. J., Ross Anderson, H., & Carslaw, D. C. (2013). Air pollution dispersion models for human exposure predictions in London. *Journal of exposure science & environmental epidemiology*, *23*(6), 647-653. Smith, J. D., Mitsakou, C., Kitwiroon, N., Barratt, B. M., Walton, H. A., Taylor, J. G., ... & Beevers, S. D. (2016). London hybrid exposure model: improving human exposure estimates to NO2 and PM2. 5 in an urban setting. *Environmental science & technology*, *50*(21), 11760-11768. Williams, M. L., Beevers, S., Kitwiroon, N., Dajnak, D., Walton, H., Lott, M. C., ... & Holland, M. (2018). Public health air pollution impacts of pathway options to meet the 2050 UK Climate Change Act target: a modelling study.	Morley DW, de Hoogh K, Fecht D, Fabbri F, Bell M, Goodman PS, Elliott P, Hodgson S, Hansell AL, and Gulliver J. (2015). International scale implementation of the CNOSSOS-EU road traffic noise prediction model for epidemiological studies. Environmental Pollution, Volume 206, pp.332-341

Using annual average exposure for 2013, 2014, 2015, 2016, 2017, and 2018, the following individual-level exposure windows were calculated. These were transformed into 1IQR increments to increase comparability between exposures and ease interpretability.

1) **Recent exposure:** The average annual exposure for the year prior to baseline assessment (relative to the individuals' assessment date*), to be assessed in relation to baseline cognition and mental health.
2) **Longitudinal exposure from baseline to follow-up:** The average annual exposure between a person's baseline and follow-up (mean 26.74 months, range 18-42 months*) was evaluated in relation to changes in outcome between baseline and

follow-up, or absolute scores at follow-up for outcomes only reported at this timepoint (depression and anxiety symptoms). If people moved between baseline and follow-up it was assumed that they moved halfway through this period.

3) **Long-term average exposure:** The effect of average exposure over the whole study period (2013-2018) was evaluated for one outcome (psychosis symptoms) in 2020-2021, by taking the average annual exposure of all years. If people moved between baseline and follow-up, it was assumed they moved halfway.

*Yearly averages were time-weighted to reflect the month of the year in which testing took place. E.g. if baseline testing was April 2015, the year prior average would be: (2014 average * 1/3) + (2015 average * 2/3).

5.2.1.3 Outcomes

The following cognitive and mental health variables were measured at baseline and follow-up (all self-completed):

- **The Culture Fair Intelligence Test (CFT)** was completed on a computer using the Psytools (Delosis Ltd) online platform. It involves viewing a sequence of non-verbal visual panels and predicting which pattern (from multiple choice) logically comes next based upon existing patterns, and measures Fluid Intelligence (Cattell, 1973). This was evaluated as a continuous score where a higher score = better performance/higher intelligence.
- **SPIN (speech-in-noise) task** was completed on a computer using the Psytools online platform. It involves detecting speech at varying volumes against background noise, measuring a personal 'threshold' volume at which speech can be reliably detected against background noise (Taylor, 2003), which reflects auditory and speech processing. This was assessed continuously where a lower threshold = better performance.

- **A Processing speed composite** was completed on a computer using Psytools and used to measure processing speed, computed as the z-score of average reaction times in the Trail Making Task (TMT) and Enumeration tasks. The TMT involves connecting numbered and alphabetised dots in ascending order, firstly using numbers then alternating numbers and letters (i.e. 1-A-2-B, etc.), and measures visual search, attention and task switching (Reitan and Wolfson, 2004). The enumeration task measures visual attention and involves counting a cluster of dots which are presented for a small interval (Green and Bavelier, 2003). This was a continuous score where lower=quicker/better reaction times.
- **An Executive function (EF) composite** was completed on a computer using Psytools and was used to measure executive function, computed as the average Z score of performance in the TMT (including accuracy rather than just speed), backward digit span, and find-the-phone task. The backward digit span task involves repeating back an increasingly large list of numbers backwards, and a person's digit span is how many numbers they can correctly recall (Dumontheil and Klingberg, 2012). This measures working memory. The answer the phones task involves checking one 'box' at a time and figuring out which box has a phone ringing under it, using process of elimination (each box will only ring once) and remembering which boxes have been checked already that trail. This measures spatial working memory (Holst and Thorell, 2017). The composite score was assessed continuously where higher=better executive functioning.
- **The Strength and Difficulties Questionnaire (SDQ)** was completed on a computer using Psytools. This questionnaire measures behavioural functioning, specifically emotional symptoms, conduct problems, hyperactivity or inattention, peer relationship problems, prosocial behaviour, giving a total difficulties score and two subscales of

internalising and externalising problems (Goodman, 1997). These were evaluated continuously where a higher score corresponds to more mental health problems.

The following were completed at follow up I and II only

- **The Patient Health Questionnaire-9 (PHQ-9)** was completed on a computer (or phone/tablet at follow-up II, when sessions were remote rather than face-to-face) using Psytools and used to measure depression symptoms, which has been shown to have good psychometric properties in adolescent groups (Richardson et al., 2010). This was evaluated continuously where higher scores = more symptoms of depression.
- **The Generalized Anxiety Disorder 7-item scale (GAD-7)** was completed on a computer (or phone/tablet at follow-up II, when sessions were remote) using Psytools and measures anxiety symptoms. It has also been shown as a valid and reliable measure in this age group (Mossman et al., 2017). This was evaluated continuously where higher scores correspond to more symptoms of anxiety.

The following were completed at follow up II only.

- **The Community Assessment of Psychic Experiences- Positive scale (CAPE-P15)** was completed via Psytools on participants' personal devices and measures psychotic-like symptoms (hallucinatory experiences and delusional thinking) in the general population, and has been shown to be effective in adolescents (Núñez et al., 2021). Total scores were used to categorise participants according to whether they are considered at Ultra-High Risk (UHR) of psychosis, using a cut-off of mean-per-item score > 1.47 (Bukenaite et al., 2017).

5.2.1.4 Covariates

Possible confounders were selected a priori based on reviews of the literature (see Chapter 1 section 4). These were ascertained from the literature to be associated with air pollution or noise exposure, be a risk or resilience factor for psychological outcomes, to not be on the causal pathway between exposure and outcome, and to each represent distinct characteristics from one another to minimise issues of collinearity or over-adjustment. These were:

- **Age** at assessment of outcome (self-reported via Psytools questionnaire)
- **Parental National Statistics Socio-economic classification (NS-SEC)**: classified based on adolescent report of parental occupation (Psytools questionnaire). Here we used the NS-SEC three-class version: 1. Higher managerial, administrative, and professional occupations; 2. Intermediate occupations; 3. Routine and Manual Occupations; 8. Never worked and long-term unemployed
- **Adolescent's ethnicity** (self-reported via Psytools questionnaire): Here classified into five categories: 1. White; 2. Black; 3. Asian; 4. Mixed ethnicity; 5. Other.
- **Adolescent's sex** (self-reported via Psytools questionnaire): 1. Male; 2. Female
- **First language** (self-reported via Psytools questionnaire): 1. English was the first language spoken at home; 2. English was not the first language spoken at home; 3. English was spoken at home at the same time as another language.
- **Parental education** (self-reported via Psytools questionnaire): Did your mother go to University? (Yes/No) Did your father go to University? (Yes/No)
- **Smoking and Environmental Tobacco Smoke (ETS)** (self-reported via Psytools questionnaire): Does anyone in your family/home smoke when you are around? How often have you smoked cigarettes (tobacco) in the last 6 months? Converted to a binary variable (yes/no) reflecting either self-reported smoking or smoking in the home (yes) or neither (no).

- **School type** (independent/state): based on meta-data regarding participating schools
- **School** (categorical code reflecting which of 39 schools the participant attended): known for baseline and follow-up I collection based on session, self-reported at follow-up II online questionnaire.
- **Neighbourhood** (postcode sector as a categorical variable): based on self-reported address via Psytools Questionnaire.

At follow-up, the corresponding baseline outcome (if measured) was also controlled for so that the exposure effect reflected the change during that time period in relation to exposure during that time period, rather than being confounded by the associations between exposure and earlier development (which were already estimated in baseline models). Several lifestyle factors which are possible confounders- time spent on busy roads, time spent outdoors, exercise carried out outdoors, and modes of transport in school commute – have already fed into exposure prediction models and so should not confound associations.

5.2.1.5 Moderators

The variables hypothesised to potentially moderate associations and investigated using interaction/subgroup analyses were as follows:

- **Sex** (as above)
- **Ethnicity** (as above)
- **School type** (as above)
- **Below average IQ:** participants were dichotomised based on whether their baseline CFT score fell below the mean score (13.18) or was average-to-above average (binary variable)

- **Religiousness:** Based on self-reported Psytools questionnaire item "What religion group or church, if any, do you belong to?". Used to compute a binary variable (0=no religion, 1=religion)
- **High/low PM_{10}** (only evaluated in noise analyses): Participants categorise as to whether their PM_{10} exposure (baseline hybrid exposure with indoor sources) fell above or below the sample mean (14.10 µg/m3)
- **High/low Lden** (only evaluated in pollution analyses): Participants categorised as to whether their baseline Lden exposure fell above or below the sample mean (54.58dBA)

5.2.1.6 Mediators

For harmful associations, the following mediators were hypothesised to explain variance in associations:

- **Sleep problems** (self-reported by baseline Psytools Questionnaire at BL and F1): During the last 4 weeks, how often did you… …have difficulty falling asleep? …sleep restlessly (toss and turn during the night)? …wake up several times during the night? …wake up too early in the morning? (total score range 0-12, assessed continuously as a total score)
- **Atopic disease** (self-reported by Psytools Questionnaire at BL and F1): Do you have a long-term disability, illness or medical condition? Do you have any allergies or food allergies? Binary variable (yes/no) produced, affirmative when any of asthma, eczema, or allergies selected.
- **BMI** (self-reported by Psytools Questionnaire at BL and F1): self-reported height and weight were used to compute BMI.
- **Cognition or mental health:** for follow-up outcomes observed to be associated with an exposure, the baseline outcome most associated with that exposure at baseline

(with the exception of the baseline score of the outcome being investigated) was investigated as a potential mediator.

5.2.2 Statistical analysis

5.2.2.1 Imputation of covariates

Using R, baseline and follow-up covariate data for ethnicity, socioeconomic status, sex, first language, smoking (themselves or in home environment; yes/no), and parental education were used to create single fields for these covariates across timepoints; if covariate data were missing for one of baseline and follow-up, the converse data was used, if there were discrepancies between baseline and follow-up, follow-up data was used. Age, school and neighbourhood were allowed to differ between baseline and follow-up. Missing covariate data were imputed using nonparametric missing value random forest imputation (missForest-package in R). This was used in order to achieve lower imputation errors than non-model-based approaches (e.g. nearest neighbour) but also permit the imputation of both categorical and continuous variables. We also wanted to use the same data/sample for all models, so a method which doesn't involve specifying the model during iteration was helpful to create a standardised dataset (rather than different imputations for each model).

5.2.2.1 Pre-processing

Area-level demographic and mental health data were plotted against the locations of SCAMP schools and home addresses to visualise geographical characteristics of the cohort. This was mapped using QGIS. Correlations between exposures were calculated to inform modelling choices.

Exposure levels were received from collaborators at Leicester University and ICL's ERG, in long form by address (baseline home, follow-up home, school) and year (2013-2018). Using

the tidyverse package in R, these were converted to create single school-weighted (9am-3pm) estimates per year per person, on the assumption that any moves in address between baseline and follow-up occurred exactly halfway. Those with missing address data (at 1 rather than 2 timepoints) were assumed to have remained at that address throughout the study period. Personal estimates for each exposure factor relative to the individual's assessment dates were then calculated for the year prior to baseline (average of that year's average exposure and the year prior, weighted based on month of assessment), the average exposure between baseline and follow-up (average exposure over the years in-between and including the year of each assessment, weighted by month of those assessments), and the average exposure for the whole period (2013-2018).

5.2.2.3 Main models

The choice of frequentist linear and logistic regression models is based upon the approach used in most prior literature, to increase consistency and comparability with prior findings and precluding the existence of valid informative priors, as well as its suitability for testing pre-specified hypotheses. GLMS also allow for good flexibility for and comparability between terms from different models within the study.

It was decided that longitudinal changes in response between BL and F1 assessed outcomes would be assessed by controlling for the BL outcome value as a covariate predictor of F1 absolute score. This was because absolute differences were often small and may not create detectable effects. It was decided not to use nested repeated measures multi-level modelling nor latent path analysis because there were only 1-2 timepoints of assessment per participant during the exposure period, so parsimony in managing timepoints was not a major motivator. Additionally, analysing the timepoints' outcomes separately increased the sample size for

baseline assessment due to attrition, as it was decided not to use imputation for the exposures and outcome data as these are most important in terns if accuracy. Thirdly, data from earlier assessment dates were not themselves predictors of interest; that is, models were longitudinal regardless because exposure was always estimated for the time before assessment (including BL), so combining all timepoints/outcomes was not necessary in order to create a longitudinal model where the main predictor of interest preceded the outcome of interest.

Although sample sizes varied by exposure (noise or air pollution) and outcome, the equivalent single and multi-level models were run with the same three levels of adjustment for all outcomes (as well as the same models run for sensitivity, moderation and mediation analyses), meaning the same variables were entered in the three types of models except for varying the exposure and outcomes, thus easily and flexibly seeing the isolated impact of each variable changing.

Adolescents in the same school or neighbourhood are likely to share some circumstances affecting psychological outcomes and will have sampling similarities, meaning there is likely to be clustering where their data are more similar to each other than data from students in different schools or residential areas. Further, the qualitative analysis highlighted the importance of general environmental quality in young people's psychological wellbeing. Therefore, these variables were entered as random intercepts for the highest/third level of adjustment to account for non-independence of observation and potential higher-order effects of school or neighbourhood environment on mental health or cognition (see figure 9 for cross-classified multi-level model structure).

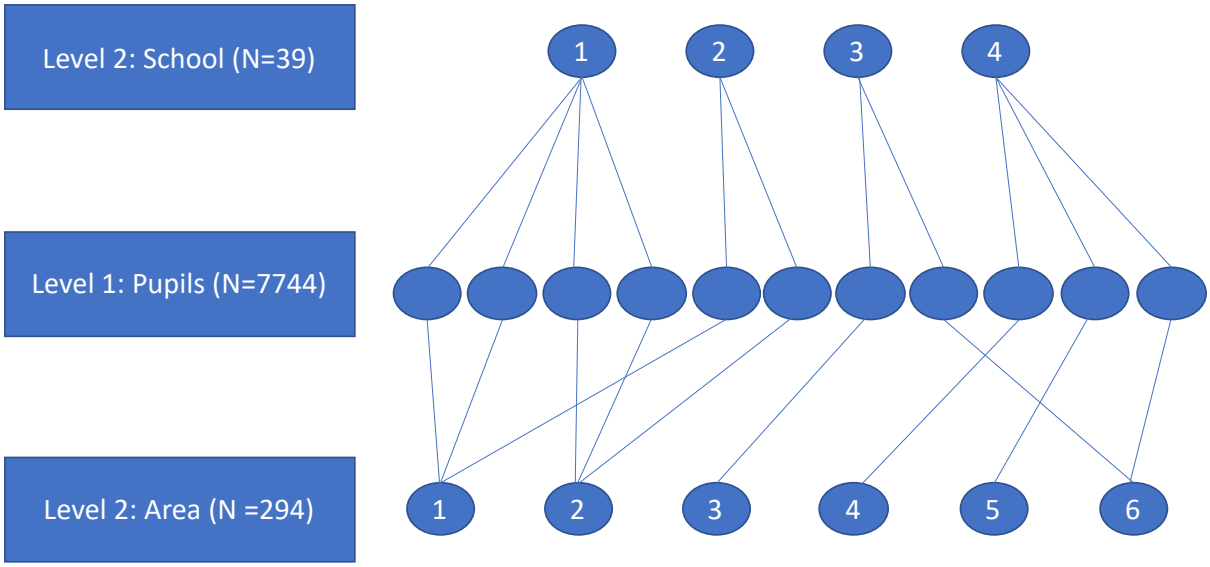


Figure 9. Illustrative model structure of cross-classified multilevel mixed-effects linear models

Other individual-level covariates were entered as fixed effects to control for them and make their impact on the response variable interpretable. The models that were used are described below:

- **Main model 1:** BL outcome ~ average exposure year-prior
- **Main model 2**: Main Model 1+ NSSEC + ethnicity + sex + BL age + first language + mother university + father university + smoke exposure (yes/no, self-reported smoking or parental smoking at home) + school type (independent/state)
- **Main model 3:** Main model 2 + school + home postcode sector (neighbourhood) (random effects)
- **Main model 4:** F1 outcome ~ average annual exposure between BL and F1
- **Main model 5:** Main Model 4 + corresponding BL outcome (if applicable) + NSSEC + ethnicity + sex + F1 age + first language + mother university + father university + smoke + school type (independent/state)

- **Main model 6:** Main model 5 + school + home postcode sector (neighbourhood) (random effects)
- **Main model 7** (CAPE only): F2 outcome ~ average exposure 2013-2018
- **Main model 8** (CAPE only): Model 7 + NSSEC + ethnicity + sex + first language + mother university + father university + smoke + school type (independent/state)
- **Main model 9** (CAPE only): Model 8 + school + home postcode sector (neighbourhood) (random effects)

Bivariate correlations and plots were inspected prior to modelling. Models were checked for collinearity using variance inflation factor (VIF) and bivariate correlations (VIF <2.5 and *r* <80%). Normality and homoscedasticity of residuals were checked using QQ plots, residuals plots, and Levene's test of equality of variances. The addition of covariates, random effects, and additional exposures was tested using likelihood ratio tests (LRTs), and comparison of Akaike information criterion (AIC) and deviance. The False discovery rate (FDR)/ Benjamini-Hochberg Procedure was applied to adjust *p* values for multiple comparisons.

5.2.2.4 Non-linear models

In order to investigate nonlinearity, the main models adjusted for socioeconomic covariates were computed as GAMs with restricted maximum likelihood (REML) estimation of smoothing parameters for the exposure variable, using the package mgcv in R. A random selection of models were run to test different smoothing terms (comparing penalised b-spines, cubic regression splines, low rank isotropic smoothers, Duchon splines, Gaussian Process smooths), with penalised b-spines the best fit for these data (based on AIC). These were compared to linear models based upon AIC, R-squared and deviance.

5.2.2.5 Multi-exposure models

Two-exposure models were run where collinearity allowed, with these multi-exposure models compared to single exposure models using goodness of fit (LRTs). As Lden, Leve and Lnight were 99% correlated (and results were similar), models adjusted for Lden were not additionally adjusted for these. Moderation by high/low noise (Lden) and high/low pollution (PM_{10}) was also explored in Chapter 7.

5.2.2.6 Sensitivity analyses

As air pollution exposure was generated as a hybrid of outdoor and indoor exposure with indoor sources, models were also run for outdoor and indoor exposure only and compared to hybrid effects. Models were also run with just random effects of school and using backward step regression from all predictor variables for outcomes where additional levels of adjustment did not add to the explanatory value of a model.

The first line of addresses was used to geocode location, which was used to predict exposure. However, use of postcode rather than first line gave different geocodes in some cases. Those who had provided ambiguous address data, differing by method (based on first line address or postcode), were excluded and models run again.

To discern if effects were attributable to spatially covariant neighbourhood stressors, quality, and degradation as opposed to air pollution or noise specifically, models were additionally adjusted for Lower-layer Super Output Area (LSOA) level English Index of Multiple Deprivation (IMD) 2019 categorical decile, ranging from most (1) to least deprived (10). This composite variable reflects area-level living conditions based on income, employment,

health, education, crime, housing and services, and physical living environment, and was obtained from the Office for National Statistics (ONS).

5.2.2.7 Moderation

Moderation of all effects by below average IQ, sex, ethnicity, school type, religiousness (yes/no), and high/low PM_{10} or Lden were investigated by adding interaction effects to adjusted models. Where significant effects were observed, subgroup analysis was conducted to disentangle the nature of the interaction.

5.2.2.8 Mediation

For those follow-up outcomes (timepoints 2 or 3) with a significant association with adjustment for covariates, random effects, or other exposures, in the whole sample or for a particular subgroup, mediation by other factors was explored. As the mediator and exposure should precede the outcome for causal inference, it was not possible to conduct mediation analysis for baseline outcomes. 'Beneficial' associations between exposure and outcomes or mediators were contrary to hypotheses and s1o a priori causal mechanisms were not specified and were not explored. For mental health outcomes, baseline cognitive outcomes were considered as mediators, and vice versa, except for the GAD-7 anxiety and CAPE psychosis risk outcomes; as these were not themselves measured at earlier timepoints, earlier mental health was considered as a potential mediator in addition to earlier cognition.

For harmful associations, the following mediators were considered at the prior timepoint: sleep problems score (0-12), asthma, eczema, or allergies (yes/no), BMI, and cognition or mental health (when an effect of that exposure was observed relating to another outcome at baseline). Therefore, the following causal associations were investigated:

- Year prior to Baseline exposure -> BL sleep/ allergies/BMI -> F1 outcome
- Year prior to Baseline exposure -> BL mental health or cognition -> F1 outcome

- Exposure between baseline and follow-up -> F1 sleep/ allergies/BMI -> F2 outcome
- Exposure between baseline and follow-up -> F1 mental health or cognition -> F2 mental health

Mediators were considered plausible mediators if associated with both the exposure and outcome in linear regression, adjusted for individual level covariates, at the $p<0.10$ significance level. Mediation was tested using the quasi-Bayesian Monte Carlo method based on normal approximation using the package mediation in R (Imai et al. 2010a), and significance testing of the average causal mediation effect (ACME) and proportion of association mediated.

Chapter 6: Epidemiological associations between exposure to air pollution and noise and psychological outcomes in adolescents

6.1 Results

6.1.1 Participant characteristics

Sample characteristics for cohort participants after multiple imputation are found in Table 7 (sample total N= 7744, BL N=6574, F1=4978). Figures 10a and 10b show the distribution of participant ages at baseline and follow-up, and figures 11-15 show participant and school (shown in red dots) locations mapped against area-level sociodemographic characteristics, prevalence of common mental health disorders, and access to psychological support. For anonymity, geocodes were rounded to 2 decimal places (about 1km^2 resolution). Figure 11 shows the locations of the sample at baseline and follow-up with good retention of spatial diversity, and figures 12-15 demonstrate that participants come from a diverse sample in terms of area-level sociodemographics and mental health. Table 8 shows the sociodemographic characteristics of the target population according to census data (Greater London school population), of baseline SCAMP, and of the analytical sample. Taken together, these data show a geographically dispersed, representative, and diverse cohort, which presents both challenges and opportunities to be discussed later in the report. The greatest deviation between the SCAMP analytical sample and census data was a 6.6% higher proportion of Asian students than in the 2015 school census (state-funded secondary schools).

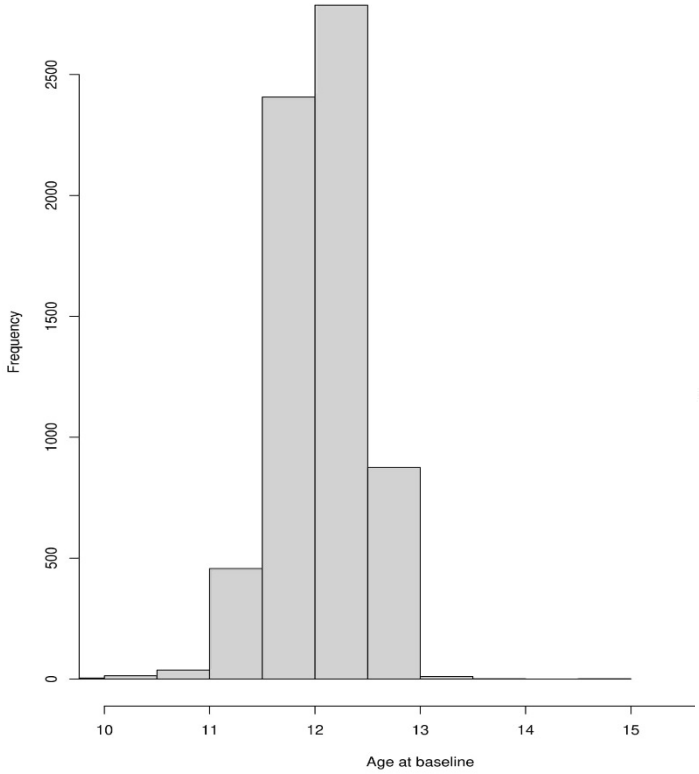
When interpreting the data, this provides confidence that the results from this cohort are generalisable to adolescents from a range of geographies, backgrounds, and with a range of experiences. The applicability to young people in areas with high prevalence of mental health disorders and low access to services has pertinence for the potential impact of policy recommendations arising from results. Table 9 shows descriptive statistics for the mental health and cognitive variables utilised in this analysis. Mental health and behavioural

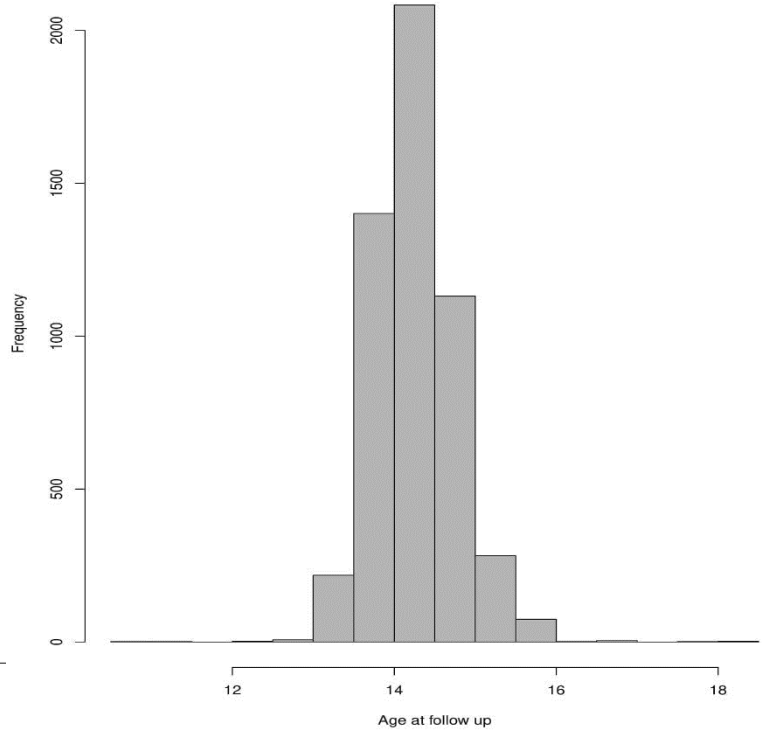
difficulties (SDQ scores) tended to increase between baseline and follow-up, and cognitive skills tended to improve (lower processing speech and speech-in-noise corresponds to better performance).

Table 7. Total cohort sociodemographics after imputation (N=7744)

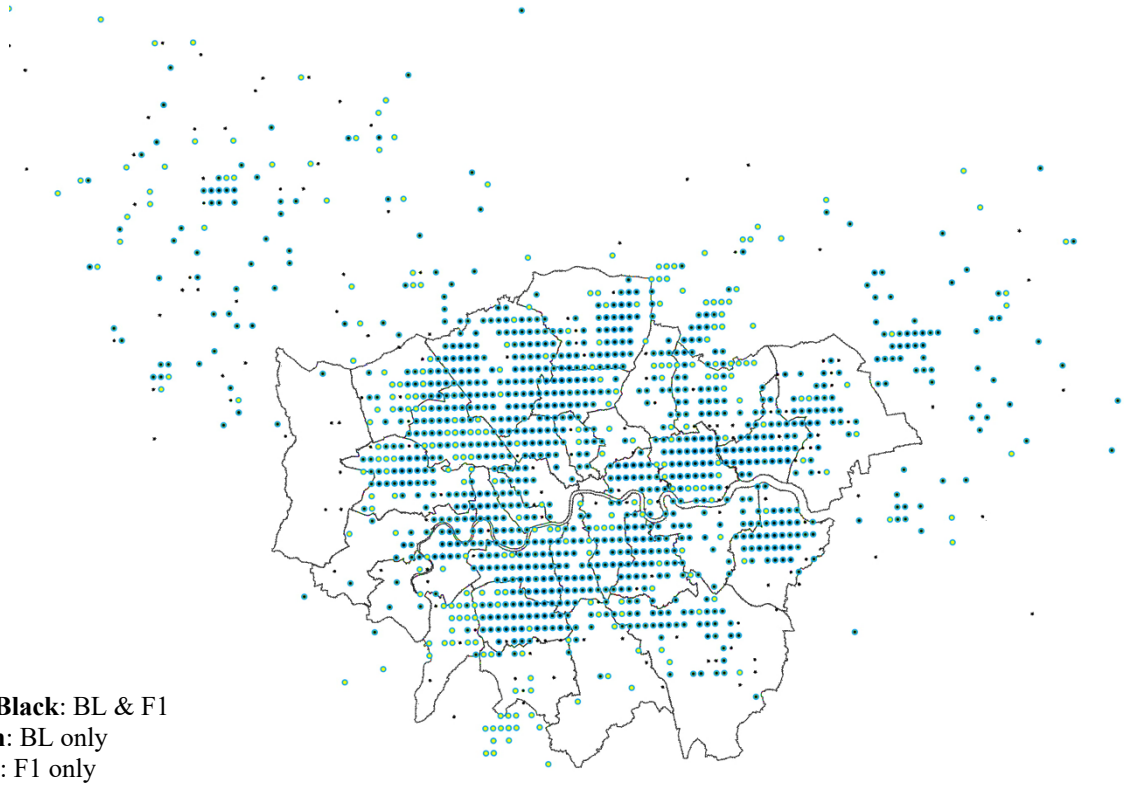
Variable	Statistic	Value
Baseline Age	μ, sd	11.59(0.48)
Follow-up Age	μ, sd	13.82(0.55)
Gender female	%	57.8
First Language was English	%	59.8
First Language was not English	%	17.3
Learnt English at the same time as another language	%	23.0
Mother attended university	%	61.4
Father attended university	%	64.02
Parental socioeconomic status	%	
NS-SEC 1 (1, 1.1, 1.2, 2)		30.0
NS-SEC 2 (3,4)		25.5
NS-SEC 3 (5, 6, 7)		33.3
NS-SEC 8		11.3
Ethnicity	%	
White		43.8
Black		16.8
Asian		28.6
Mixed race		9.9
Other ethnic group		0.8
Smoking or ETS (self-reported yes)	%	25.8
Religious belief or practice (yes)	%	68.6

NS-SEC: The National Statistics Socio-economic Classification (1-2: Managerial, administrative, and professional occupations; 3-4: Intermediate occupations; 5-7: Routine and manual occupations; 8: never worked or long-term unemployed)





Figures 10a and 10b. Histograms of participant ages at baseline and follow-up



Blue/Black: BL & F1
Green: BL only
Black: F1 only

Figure 11. Participant locations and attrition from baseline (BL) and follow-up (F1)

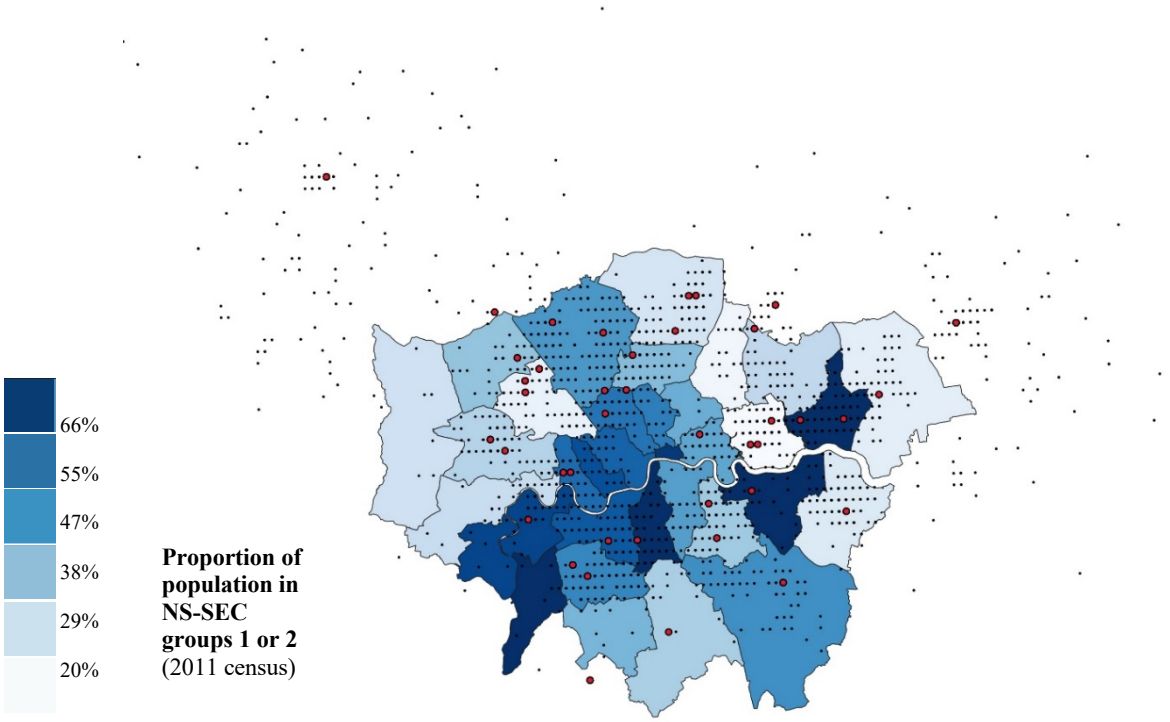


Figure 12. Participants, schools, and area-level socioeconomic status

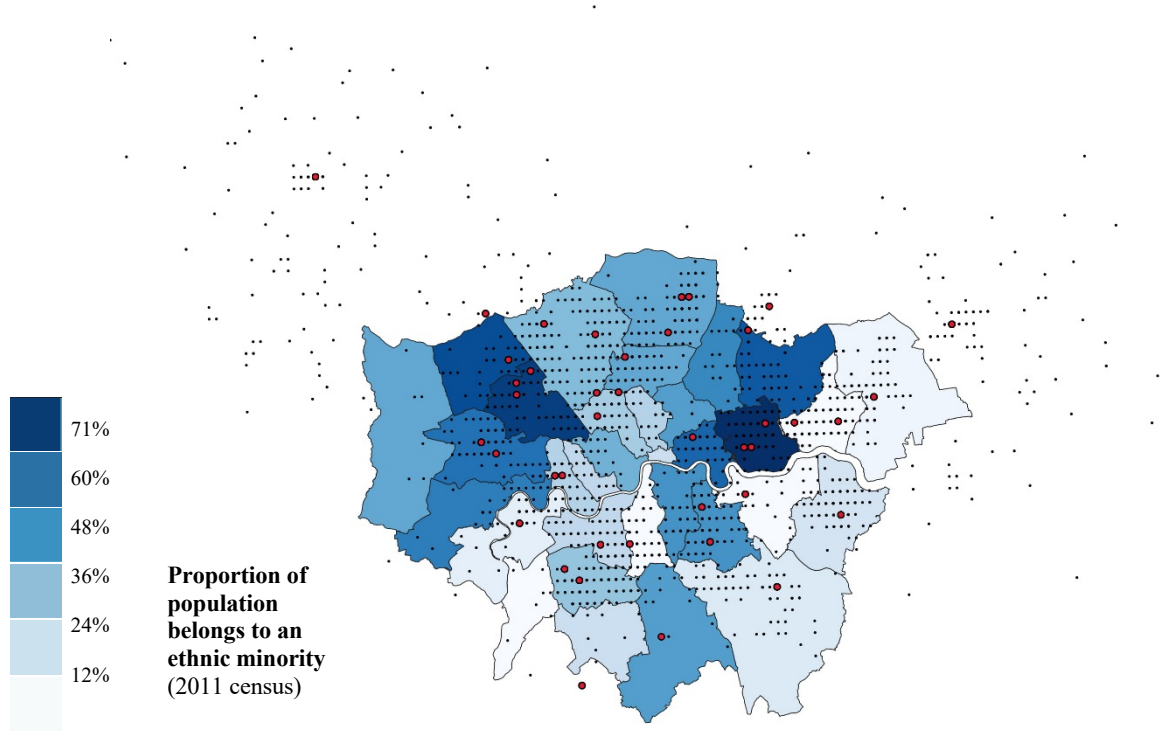


Figure 13. Participants, schools, and area-level ethnicity

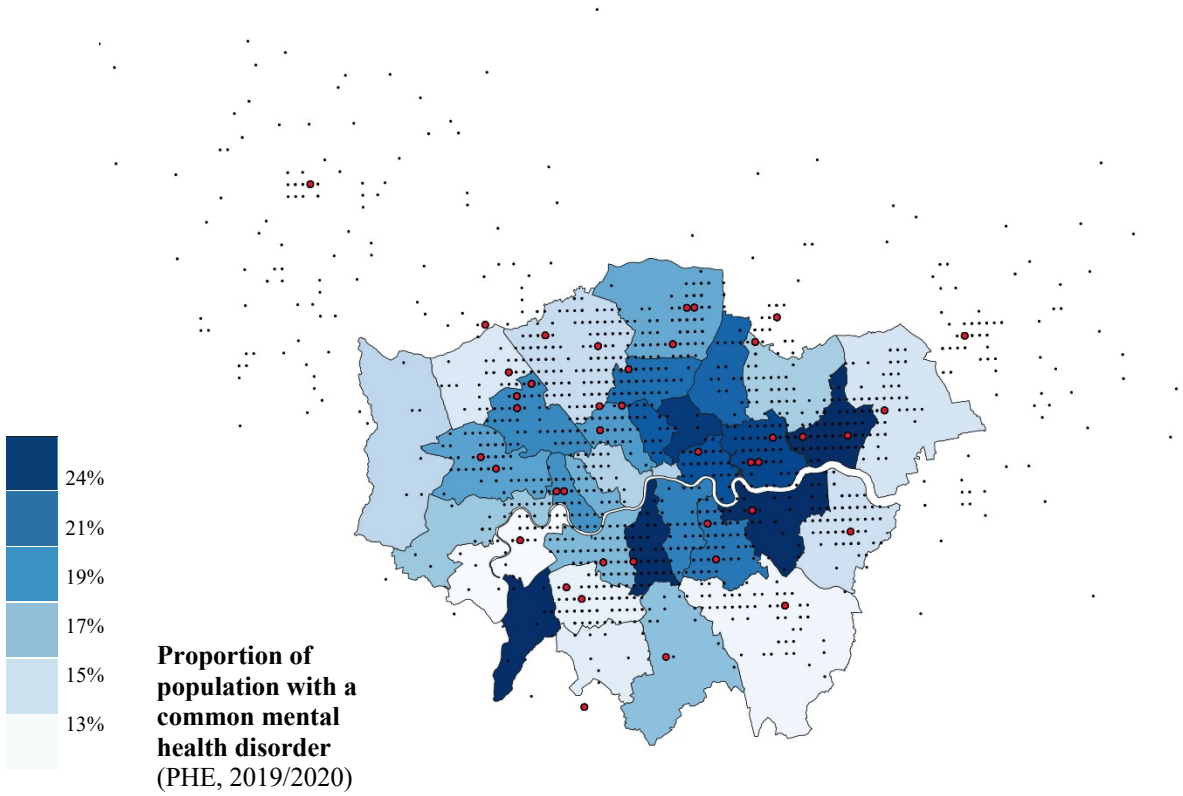


Figure 14. Participants, schools, and area-level estimated mental disorder prevalence (%)

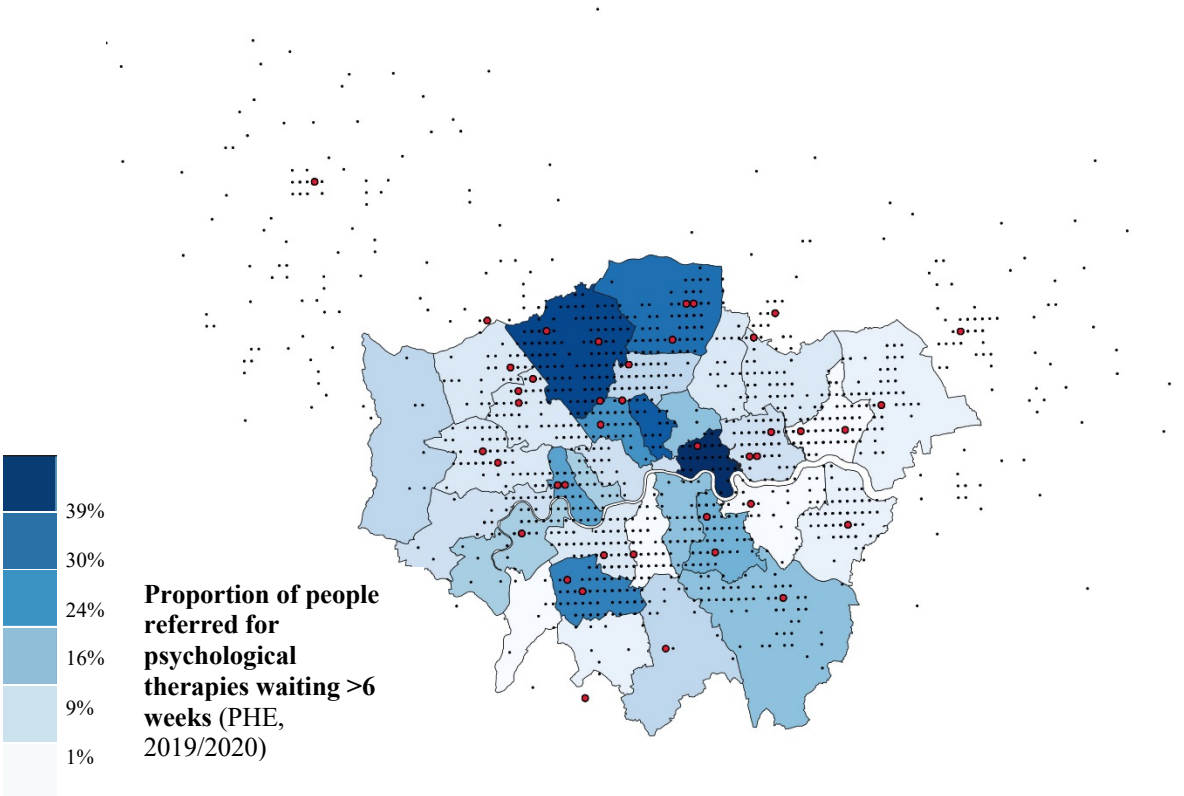


Figure 15. Participants, schools, and area-level access to mental health treatment (% waiting >6 weeks for first treatment from NHS talking therapies services)

Table 8. Comparing Baseline sociodemographic characteristics of the target population SCAMP cohort, and imputed analytical sample, adapted from Toledano et al., (2019)

	Target population	SCAMP Baseline sample	Total sample including follow-up and after imputation
	Based on census data[5]	6574	7744
Male	50.8%	47.5%	48.6%
Ethnicity:			
White	41.1	44.7	44.2
Black	21.3	16.5	16.7
Asian	21.2	27.6	27.8
Mixed	8.7	10.4	10.4
Other ethnic group	5.6	0.9	0.8
Missing	2.1	N = 277	Imputed to above
Socioeconomic classification:			
(1) Managerial/ professional occupations	35.5	33.9	29.1
(2) Intermediate occupations, Small employers and own-account workers	21.5	27.8	24.6
(3) Lower supervisory and technical occupations, Semi-routine/routine occupations	39.4	30.5	38.8
(8) Never worked, long-term unemployed	8.5	7.9	7.5
Not classified or Missing (N)	10.7	N = 2971	Imputed to above
Type of school			
State	76.8	77.7	77.5
Independent	23.2	22.3	22.5

[5] Gender, ethnicity and type of school from January 2015 School Census (www.gov.uk/government/statistics/schools-pupils-and-their-characteristics-january-2015) and data on socioeconomic classification from 2011 Census: NS-SEC in London (https://data.london.gov.uk/dataset/ns-sec-report-data).

Table 9. Descriptive statistics of mental health and cognitive variables measured at baseline (2014-2016), follow-up 1 (2016-2018), and follow-up 2 (2020-2021) in total cohort

Outcome	Baseline N for outcome	Baseline median (IQR)	Follow-up N for outcome	Follow-up median (IQR)
SDQ- total score	3300	9.00(7)	3183	10.00(7)
SDQ- internalising	3300	4.00(4)	3183	4.00(5)
SDQ- externalising	3300	5.00(4)	3183	6.00(5)
PHQ-9 (Depression)	-	-	2585	3.00(6)
GAD-7 (Anxiety)	-	-	2631	3.00(5)
Processing speed composite score	2634	-0.11(0.56)	2452	-0.44(0.49)
EF-composite score	5757	0.09(0.90)	4298	0.25(0.83)
Culture fair task total score	5787	13.00(5)	4563	15.00 (5)
Speech-in-noise threshold	4944	-6.00(3)	2273	-7.50(4.5)
				Follow-up 2 (2020-2021)
CAPE-P15 (2020-21)			236	20.0(7)

SDQ: Strengths and difficulties Questionnaire. PHQ-9: patient health questionnaire-9. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale

Air pollution estimates using the hybrid exposure model could only be produced within the M25 and where an address was provided for both timepoints, leaving a sample of 3530 with air pollution exposure estimates and 7555 with noise exposure estimates. Sample size, range, mean, and correlations between exposures can be seen in Table 10. The mean annual levels of $PM_{2.5}$ (10.5 $\mu g/m^3$) exceed WHO Air quality guideline values (5 $\mu g/m^3$), as did NO_2 (mean 10.2 $\mu g/m^3$, guideline 10 $\mu g/m^3$) but PM_{10} did not (mean 14.1 against a guideline of 15 $\mu g/m^3$). WHO ozone guidelines are for 8-hour daily maximum values or 8-hour means during peak season, so our annual mean value (mean 4.6 ppb) are not directly comparable.

To be included in the analysis complete exposure and outcome data were required, though missing covariates were imputed. Sample numbers varied by outcome, therefore the Ns for the different models are variable. Exposure assessment required a home address at baseline and/or follow-up and school address, leaving a total analytical sample of 7555 (N=141 participants in the total cohort (N=7744) did not provide any address data and so could not

have exposure estimated for analysis, and 48 of those with exposure data (N=7603) did not provide any outcome data).

Table 11a shows the differences in baseline characteristics between the two analytical samples (noise and air pollution) and 11b shows the differences between participants who were lost-to-follow-up and those that remained. The sample who contributed to the noise analyses only were significantly older, and had a significantly higher proportion of participants who were male, had parental occupations in NSSEC classifications 3 and 8, and whose first language was not English or who learnt English in tandem with another first language (table 11a). In terms of exposure to noise, only their exposure to daytime noise significantly differed, which was significantly higher within the M25. It should be noted that these participants were also included in pollution analyses, pooled with those within the M25, so they did not constitute an analytical sample in and of themselves (but significance testing between the pollution and noise analytical samples would involve comparing participants to themselves and so would not be statistically sound). A sensitivity analysis was conducted of the noise analyses only including the within M25 sample to test for sampling bias (see section 6.1.4/ appendix IX).

Comparing the sample at different timepoints (Table 11b), the proportion of female and first language English students increased between baseline and follow-up 1, then decreased to below baseline levels at follow-up II. The proportion of NSSEC 1 or 2, white ethnicity, and independent school students increased at both timepoints, whereas the proportion whose mother attended university decreased then increased between the timepoints. These samples could not be directly compared (due to being largely constituted of the same participants), but within baseline participants, chi-squared tests of the association between retention to F1 (lost

N= 2651, retained N= 3792) and sociodemographic characteristics showed significant differences in all variables. Retention to F2 (lost N= 6233, retained N= 210) was associated with NSSEC, ethnicity and independent school status. However, these comparisons had high statistical power such that even small associations would have been detectable (e.g. with sample size N= 6443 & df =4, we have 95% power to detect an effect size of 0.05). The differences between all of baseline and all of follow-up I (the overlapping populations included in analyses) were small (>3.5%) and proportions were also broadly consistent with census data, retaining much of the diversity and representativeness of the baseline sample. On the other hand, the COVID-19 pandemic presented substantial limitations to research during F2 and this sample was highly overrepresented with NSSEC 1- 2 and independently educated participants (>10% higher than baseline and > 7% higher then census data). Only the outcome 'CAPE Psychosis Risk' was analysed from follow-up II, and as it was not included for the whole survey period (only at follow-up II) and some of these participants did not provide an address at earlier timepoints for exposure assessment, this analysis had a limited sample of 235 participants for noise exposure and 123 for air pollution exposure. Limited sample size and representativeness need to be taken into consideration when interpreting these results.

Table 10. Descriptive statistics for exposures (average 2013-2018) in analytical sample livin g within M25, N=3530

Exposure	**Mean (range)**	**$PM_{2.5}$** ***r(p)***	**PM10** ***r(p)***	**NO2** ***r(p)***	**O3** ***r(p)***	**Lden** ***r(p)***	**Lday** ***r(p)***	**Lnight** ***r(p)***	**Leve** ***r(p)***
$PM_{2.5}$	10.5 µg/m^3 (7.4-14.1)	1	0.90 (<0.001)	0.87 (<0.001)	-0.48 (<0.001)	0.29 (<0.001)	0.22 (<0.001)	0.29 (<0.001)	0.29 (<0.001)
PM_{10}	14.1 µg/m^3 (8.4-18.9)		1	0.84 (<0.001)	-0.33 (<0.001)	0.27 (<0.001)	0.22 (<0.001)	0.27 (<0.001)	0.26 (<0.001)
NO_2	10.3 µg/m^3 (4.3-17.6)			1	-0.57 (<0.001)	0.35 (<0.001)	0.32 (<0.001)	0.35 (<0.001)	0.35 (<0.001)
O_3	4.6 ppb (2.7-7.0)				1	-0.28 (<0.001)	-0.22 (<0.001)	-0.28 (<0.001)	-0.28 (<0.001)
Lden	54.6 dB(A) (43.8-81.2)					1	0.68 (<0.001)	0.99 (<0.001)	0.99 (<0.001)
Lday	55.8 dB(A) (39.1-78.5)						1	0.68 (<0.001)	0.67 (<0.001)
Lnight	47.9 dB(A) (37.9-74.4)							1	0.99 (<0.001)
Leve	51.8 dB(A) (38.0-79.0)								1

$PM_{2.5}$ = particulate matter < 2.5 micrometres in diameter. PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nit rogen Dioxide. O_3 = ozone. Lden = 24-hour A-weighted equivalent noise level, +10db penalty for evening and night. Lday = A-weighted equivalent noise level 7am-7pm. Lnight = A-weighted equivalent noise level 11pm-7am. Leve = A-weighted eq uivalent noise level 7pm-11pm.

Table 11a. Comparison between participants living within the M25 (only included in air poll ution models), outside the M25 (included in noise models only), and the whole sample (comp lete noise sample, some of whom also included in air pollution analyses)

	Inside *and* outside M25 Noise analytical sample (N=7555)	Within M25 Air pollution analytical sample (N=3529)	Outside M25, Sample in noise analyses only (N=4026)	Within vs outside M25 Difference testing
	Mean and standard deviation:			*Welch Two Sample t-test*
Average age BL	11.61(0.47)	11.59(0.48)	11.63(0.46)	t = -3.9(7370.3), $\mathbf{p < 0.001}$
Average age F1	13.86(0.49)	13.82(0.55)	13.90(0.43)	t = -7.1(6621.7), $\mathbf{p < 0.001}$
	Proportion (%):			*Chi-squared test*
Female	51.5	57.9	45.9	χ^2 = 107.2(1), $\mathbf{p < 0.001}$
White ethnicity	44.4	43.8	45.0	χ^2 = 2.54(4), p = 0.64
NSSEC 1 and 2	53.9	55.5	52.5	χ^2 = 171.7(3), $\mathbf{p < 0.001}$
First language English	58.7	59.8	57.7	χ^2 = 33.9(2), $\mathbf{p < 0.001}$
Independent school	22.5	22.7	22.2	χ^2 = 0.19(1) p = 0.67
	Mean and range (average annual exposure 2013-2018):			*Welch Two Sample t-test*
$PM_{2.5}$(μg/m^3)	10.5 (7.4-14.1)		N/a	N/a
PM_{10}(μg/m^3)	14.1 (8.4-18.9)		N/a	N/a
NO_2(μg/m^3)	10.3 (4.3-17.6)		N/a	N/a
O_3 (ppb)	4.6 (2.7-7.0)		N/a	N/a
Lden (dB)	54.6(43.8-81.5)	54.6(43.8-81.2)	54.5(43.9-81.5)	t = 0.43(7535.5), p = 0.67
Lday (dB)	55.5(38.4-78.9)	55.8(39.1-78.5)	55.2(38.4-78.9)	t = 4.35(7467), $\mathbf{p < 0.001}$
Lnight (dB)	47.9(37.9-74.7)	47.9(37.9-74.4)	47.9(38.0-74.7)	t = 0.36(7534.8), p = 0.72
Leve (dB)	51.8 (38.0-79.0)	51.9(38.0-78.7)	51.8(38.0-79.0)	t = -0.67(7538.9), p = 0.50

Table 11b. Comparison of analytical sample (providing at least one address) in total, baseline (BL), follow-up I (F1), and follow-up II (F2)

Characteristic	Total analytical sample	BL	F1 sample	F2 sample	BL sample: association of retention at F1 with characteristic χ^2 (df), p	BL sample: association of retention at F2 with characteristic χ^2 (df), p
N	7555	6443	4904	235	6443	6443
%Female	51.5%	52.6%	54.6%	48.5%	84.3(1), **<0.001**	0.02(2), p=0.90
% NSSEC 1 & 2	53.9%	53.6%	56.7%	64.3%	449.9(3), **<0.001**	16.03(3), **p<0.01**
% White Ethnicity	44.4%	44.2%	45.9%	49.4%	15.1(4), **p<0.01**	15.2(4), **p<0.01**
%Independent school	22.5%	22.7%	26.1%	36.6%	132.2(1), p**<0.001**	21.7(1), **p<0.001**
% Mother university	64.0%	64.4%	62.0%	66.0%	20.95(1), **p<0.001**	0.03(1), p=0.86
% English as first language	58.7%	59.3%	59.8%	58.3%	49.50(2), **p<0.001**	0.36(2), p=0.83

6.1.2 Main model results

6.1.2.1 Air pollution associations (hybrid exposure)

Table 12 shows the results of the main models estimating associations between exposure to $PM_{2.5}$ and PM_{10} (hybrid exposure, i.e. indoor + outdoor pollution including indoor sources) and psychological outcomes at baseline and follow-up. $PM_{2.5}$ exposure between baseline and follow-up was associated with positive changes in executive function at follow-up (with baseline score controlled for, Model 2: $\beta = 0.04$, $p = 0.001$) . One-year $PM_{2.5}$ prior to assessment was associated with better baseline speech-in-noise perception. Similarly, PM_{10} was associated with better executive function at baseline and follow-up. PM_{10} was associated with higher baseline externalising problems (Model 2 $\beta = 0.14$, $p = 0.023$), higher follow-up anxiety symptoms ($\beta = 0.19$, $p = 0.048$).

Table 12. Primary model results reflecting associations between exposure to $PM_{2.5}$ or PM_{10} and continuous psychological outcomes at baseline and follow-up

Outcome N= -35 for random effects	1: Unadjusted 2: Adjusted 3: 2+ Random effects	$PM_{2.5}$ 1IQR= 1.20 β	SE	p	PM_{10} 1IQR=1.01 β	SE	p
Baseline outcomes							
SDQ Higher score=more difficulties N=3057	1	-0.269	0.107	**0.012**	0.141	0.098	0.15
	2	-0.186	0.112	*0.096*	0.176	0.099	*0.074*
	3	-0.153	0.14	0.277	0.16	0.113	0.156
SDQ internalising Higher score=more difficulties N=3057	1	-0.09	0.063	0.149	0.032	0.057	0.581
	2	-0.1	0.066	0.13	0.039	0.058	0.502
	3	-0.049	0.081	0.548	0.067	0.066	0.309
SDQ externalising Higher score=more difficulties N=3057	1	-0.179	0.066	**0.007**	0.11	0.061	*0.071*
	2	-0.087	0.069	0.206	0.138	0.06	**0.023**
	3	-0.113	0.086	0.189	0.094	0.069	0.173
EF Higher score=higher performance N=3158	1	0.048	0.012	**<0.001**	0.034	0.012	**0.004**
	2	0.021	0.013	*0.097*	0.019	0.012	0.103
	3	0.029	0.017	*0.085*	0.028	0.013	**0.033**
CFT Higher score=higher performance N=3188	1	0.002	0.067	0.977	0.071	0.063	0.259
	2	-0.084	0.069	0.223	0.001	0.062	0.986
	3	-0.036	0.094	0.701	0.03	0.073	0.684
Processing speed Higher score=slower performance N=1522	1	-0.006	0.012	0.61	-0.003	0.011	0.783
	2	-0.007	0.012	0.594	0.001	0.011	0.95
	3	-0.019	0.016	0.236	-0.013	0.012	0.29

SPIN Lower threshold= lower volume detection N=2744	1	-0.194	0.07	**0.005**	0.043	0.06	0.473
	2	-0.169	0.072	**0.019**	0.057	0.06	0.345
	3	-0.106	0.088	0.231	-0.001	0.068	0.987
Follow-up outcomes							
SDQ Higher score=more difficulties N=2654	1	0.105	0.119	0.379	-0.007	0.104	0.944
	2	0.12	0.122	0.327	-0.138	0.093	0.136
	3	0.097	0.155	0.533	-0.167	0.105	0.114
SDQ internalising Higher score=more difficulties N=2967	1	-0.008	0.072	0.908	-0.043	0.063	0.49
	2	-0.025	0.073	0.737	-0.076	0.056	0.18
	3	-0.015	0.094	0.875	-0.1	0.063	0.117
SDQ externalising Higher score=more difficulties N=2967	1	0.113	0.076	0.135	0.036	0.066	0.587
	2	0.001	0.071	0.984	-0.063	0.06	0.29
	3	-0.003	0.091	0.972	-0.063	0.068	0.356
PHQ-9 Higher score=more symptoms N=2389	1	0.096	0.127	0.453	0.075	0.112	0.504
	2	-0.06	0.135	0.656	0.009	0.112	0.935
	3	-0.031	0.161	0.849	0.027	0.125	0.827
GAD-7 Higher score=more symptoms N=2433	1	0.23	0.109	**0.035**	0.237	0.096	**0.013**
	2	0.138	0.115	0.227	0.19	0.096	**0.048**
	3	0.165	0.133	0.218	0.199	0.104	*0.056*
EF Higher score=better performance N=3095	1	0.052	0.013	**<0.001**	0.048	0.011	**<0.001**
	2	0.039	0.012	**0.001**	0.026	0.01	**0.009**
	3	0.029	0.015	*0.059*	0.016	0.012	0.171
CFT Higher score=better performance N=3285	1	0.073	0.077	0.342	0.159	0.067	**0.018**
	2	0.047	0.07	0.507	0.073	0.059	0.219
	3	0.091	0.086	0.289	0.08	0.066	0.222
Processing speed Higher=slower performance N=940	1	0.031	0.013	0.016	0.022	0.011	*0.053*
	2	0.024	0.013	*0.066*	0.02	0.011	*0.073*
	3	0.016	0.017	0.346	0.016	0.013	0.194
SPIN Lower threshold= better threshold for hearing N=1348	1	-0.005	0.098	0.962	-0.015	0.085	0.858
	2	-0.09	0.104	0.39	-0.021	0.086	0.806
	3	-0.131	0.127	0.307	-0.025	0.097	0.798

$PM_{2.5}$ = particulate matter < 2.5 micrometres in diameter. PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nitrogen Dioxide. O_3 = ozone. Lden = 24-hour A-weighted equivalent noise level, +10db penalty for evening and night. Lday = A-weighted equivalent noise level 7am-7pm. BL= baseline. F1= Follow-up-1. SDQ: Strengths and difficulties Questionnaire. EF= executive function. CFT= culture fair intelligence test. SPIN= speech-in-noise threshold. PHQ-9: patient health questionnaire-9. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale. **Bold: p<0.05**. *Italics: p<0.10*

Table 13 shows the associations between exposure to NO_2 or Ozone (hybrid exposure) and continuous psychological outcomes at baseline and follow-up. NO_2 was associated with better executive function at baseline and follow-up (follow-up Model 3: $\beta = 0.035$, $p < 0.01$) Ozone was associated with worse follow-up executive function (Model 3: $\beta = -0.05$, $p < 0.001$).

Table 13. Primary model results reflecting associations between exposure to NO_2 or O_3 and continuous psychological outcomes at baseline and follow-up.

Outcome N= -35 for random effects	1: Unadjusted 2: Adjusted 3: 2+ Random effects	NO2 1IQR= 1.91 β	SE	p	O3 1IQR = 0.79 β	SE	p
Baseline outcomes							
SDQ Higher score=more difficulties N=3057	1	-0.016	0.117	0.893	-0.051	0.125	0.682
	2	0.059	0.118	0.616	-0.091	0.127	0.474
	3	0.166	0.145	0.255	-0.158	0.15	0.292
SDQ internalising Higher score=more difficulties N=3057	1	-0.068	0.068	0.319	-0.018	0.073	0.806
	2	-0.063	0.07	0.367	0.041	0.075	0.585
	3	0.022	0.084	0.797	-0.03	0.087	0.731
SDQ externalising Higher score=more difficulties N=3057	1	0.052	0.072	0.469	-0.033	0.077	0.666
	2	0.122	0.072	*0.092*	-0.132	0.078	*0.09*
	3	0.131	0.088	0.137	-0.122	0.09	0.18
EF Higher score=higher performance N=3158	1	0.054	0.014	**<0.001**	-0.011	0.015	0.462
	2	0.031	0.014	**0.022**	-0.017	0.015	0.257
	3	0.032	0.017	*0.057*	-0.008	0.017	0.639
CFT Higher score=higher performance N=3188	1	0.101	0.074	0.17	-0.042	0.08	0.598
	2	-0.006	0.073	0.932	-0.038	0.08	0.631
	3	0.015	0.095	0.877	-0.084	0.098	0.394
Processing speed Higher score=slower performance N=1522	1	-0.006	0.013	0.645	0.006	0.015	0.689
	2	0	0.014	0.994	0.01	0.015	0.517
	3	-0.004	0.017	0.811	0.012	0.018	0.509
SPIN Lower threshold= lower volume detection N=2744	1	-0.035	0.072	0.629	-0.046	0.076	0.542
	2	-0.021	0.073	0.769	-0.019	0.078	0.811
	3	-0.009	0.088	0.915	-0.058	0.091	0.521
Follow-up outcomes							
SDQ Higher score=more difficulties N=2654	1	-0.135	0.118	0.251	-0.027	0.131	0.834
	2	-0.203	0.105	*0.053*	0.159	0.118	0.176
	3	-0.139	0.122	0.256	0.035	0.135	0.798
SDQ internalising Higher score=more difficulties N=2967	1	-0.124	0.07	*0.079*	-0.002	0.078	0.977
	2	-0.114	0.064	*0.073*	0.1	0.071	0.161
	3	-0.11	0.073	0.131	0.067	0.08	0.403
SDQ externalising Higher score=more difficulties N=2967	1	-0.011	0.075	0.883	-0.025	0.083	0.761
	2	-0.092	0.067	0.172	0.062	0.076	0.41
	3	-0.032	0.079	0.683	-0.017	0.087	0.849
PHQ-9 Higher score=more symptoms N=2389	1	0.005	0.13	0.971	-0.119	0.152	0.433
	2	-0.075	0.13	0.565	0.119	0.155	0.445
	3	-0.04	0.144	0.778	0.121	0.168	0.471
GAD-7 Higher score=more symptoms N=2433	1	0.179	0.111	0.106	-0.143	0.13	0.273
	2	0.12	0.11	0.276	0.021	0.131	0.873
	3	0.13	0.119	0.275	0.031	0.139	0.825
EF Higher score=better performance N=3095	1	0.086	0.012	**<0.001**	-0.068	0.014	**<0.001**
	2	0.052	0.011	**<0.001**	-0.067	0.012	**<0.001**
	3	0.035	0.013	**0.007**	-0.047	0.014	**0.001**
	1	0.216	0.074	**0.004**	-0.157	0.084	*0.063*

CFT Higher score=better performance N=3285	2	0.042	0.065	0.517	-0.114	0.074	0.126
	3	0.058	0.075	0.439	-0.143	0.084	0.088
Processing speed Higher=slower performance N=940	1	0.011	0.013	0.408	0.016	0.015	0.286
	2	0.007	0.012	0.594	0.014	0.014	0.312
	3	0.006	0.015	0.7	0.018	0.016	0.277
SPIN Lower threshold= better threshold for hearing N=1348	1	-0.086	0.094	0.359	-0.006	0.103	0.951
	2	-0.081	0.095	0.393	0.118	0.106	0.266
	3	-0.066	0.112	0.554	0.09	0.122	0.463

$PM_{2.5}$ = particulate matter < 2.5 micrometres in diameter. PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nitrogen Dioxide. O_3 = ozone. Lden = 24-hour A-weighted equivalent noise level, +10db penalty for evening and night. Lday = A-weighted equivalent noise level 7am-7pm. BL= baseline. F1= Follow-up-1. SDQ: Strengths and difficulties Questionnaire. EF= executive function. CFT= culture fair intelligence test. SPIN= speech-in-noise threshold. PHQ-9: patient health questionnaire-9. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale. **Bold: p<0.05**. *Italics: p<0.10*

Logistic regression was used to evaluate the impact of 1IQR increase in each exposure as an average annual exposure from 2013-2018 and odds of CAPE-assessed UHR of psychosis (Table 14), which were mostly positive but not significant in single or multi-exposure models, which could be attributable to low power (N=123, other outcomes ranged from N = 940 to N= 3285).

Table 14. Logistic regression at follow up-2 (2020-2021) of odds of psychotic-like experiences at age 15-18 at different levels of average exposure to pollutants and noise from 2013-2018

CAPE Ultra-High Risk for Psychosis (cut-off 1.47)	Models	PM2.5 1IQR: 0.96 OR(95% CI)	PM10 0.99 OR(95% CI)	NO_2 1.72 OR(95% CI)	O_3 0.78 OR(95% CI)	Lden 5.94 OR(95% CI)	Lday 8.40 OR(95% CI)
N=123 (air pollution) N=235 (noise)	1	1.28 (0.76-2.15)	1.14 (0.73-1.83)	0.85 (0.51-1.43)	0.75 (0.42-1.29)	1.07 (0.82-1.40)	0.92 (0.60-1.40)
	2	1.46 (0.82-2.62)	1.39 (0.84-2.38)	1.11 (0.59-2.11)	0.92 (0.47-1.77)	1.03 (0.69-1.52)	0.91 (0.51-1.61)
	3	1.46 (0.82-2.59)	1.39 (0.83-2.32)	1.11 (0.59-2.12)	0.91 (0.46-1.79)	1.03 (0.70-1.53)	0.91 (0.51-1.61)

$PM_{2.5}$ = particulate matter < 2.5 micrometres in diameter. PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nitrogen Dioxide. O_3 = ozone. Lden = 24-hour A-weighted equivalent noise level, +10db penalty for evening and night. Lday = A-weighted equivalent noise level 7am-7pm. BL= baseline. F1= Follow-up-1. SDQ: Strengths and difficulties Questionnaire. EF= executive function. CFT= culture fair intelligence test. SPIN= speech-in-noise threshold. PHQ-9: patient health questionnaire-9. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale. **Bold: p<0.05**. *Italics: p<0.10*

6.1.2.2 Noise associations

Due to similarities in effects and high correlation, only Lden was presented here (Leve and Lnight in Appendix XI). Exposure to noise showed trend associations with higher baseline total SDQ and internalising problems, and higher follow-up anxiety (table 15). Lden, Leve and Lnight were 99% correlated and so were associated with similar effects, however mental health associations were strongest for evening exposure.

Table 15. Primary models reflecting associations between exposure to noise and psychological outcomes in adolescence

Outcome N= -35 for random effects	1:Unadjusted 2: Adjusted 3: Random effects	Lday 1IQR=8.37 dB β	SE	p	Lden 1IQR= 5.79 dB β	SE	p
Baseline outcomes							
SDQ Higher score=more difficulties N=3288	1	-0.029	0.016	*0.06*	0.04	0.016	**0.011**
	2	0.003	0.017	0.868	0.025	0.016	0.114
	3	0.016	0.019	0.403	0.025	0.016	0.119
SDQ internalising Higher score=more difficulties N=3288	1	-0.011	0.009	0.205	0.023	0.009	**0.013**
	2	0.003	0.01	0.761	0.016	0.009	*0.078*
	3	0.01	0.011	0.37	0.017	0.009	*0.068*
SDQ externalising Higher score=more difficulties N=3288	1	-0.018	0.01	*0.064*	0.017	0.01	*0.078*
	2	0	0.01	0.983	0.009	0.01	0.373
	3	0.004	0.012	0.699	0.008	0.01	0.432
EF Higher score=higher performance N=5669	1	0.012	0.002	**<0.001**	-0.005	0.001	**<0.001**
	2	0.003	0.002	0.178	0	0.002	0.918
	3	0.002	0.002	0.395	0.001	0.002	0.776
CFT Higher score=higher performance N=5692	1	0.046	0.008	**<0.001**	-0.011	0.008	0.165
	2	0.001	0.011	0.901	0.013	0.01	0.204
	3	0.006	0.012	0.621	0.013	0.01	0.203
Processing speed Higher score=slower performance N=2612	1	0	0.001	0.895	0.003	0.001	**0.03**
	2	0	0.002	0.96	0.002	0.002	0.385
	3	0	0.002	0.943	0.001	0.002	0.658
SPIN Lower threshold= lower volume detection N=4475	1	-0.029	0.008	**<0.001**	0.017	0.008	**0.027**
	2	-0.009	0.010	0.334	0	0.01	0.994
	3	-0.009	0.011	0.447	-0.003	0.01	0.779
Follow-up outcomes							
SDQ Higher score=more difficulties N= 2838	1	-0.035	0.017	**0.036**	0.012	0.018	0.504
	2	0.002	0.017	0.915	0.009	0.017	0.571
	3	0.008	0.02	0.685	0.006	0.017	0.705
	1	-0.031	0.01	**0.002**	0	0.011	0.969

SDQ internalising Higher score=more difficulties N=2838	2	-0.008	0.01	0.444	0.001	0.01	0.894
	3	-0.004	0.012	0.757	0	0.01	0.966
SDQ externalising Higher score=more difficulties N=2838	1	-0.004	0.011	0.706	0.011	0.011	0.312
	2	0.009	0.01	0.389	0.001	0.01	0.944
	3	0.008	0.012	0.477	0.001	0.01	0.943
PHQ-9 Higher score=more symptoms N= 2575	1	-0.01	0.017	0.573	0.002	0.019	0.906
	2	0.003	0.019	0.862	-0.008	0.018	0.652
	3	-0.004	0.021	0.855	-0.008	0.019	0.654
GAD-7 Higher score=more symptoms N= 2621	1	0.015	0.015	0.307	0.031	0.016	*0.053*
	2	0.017	0.016	0.292	0.027	0.016	*0.083*
	3	0.017	0.017	0.31	0.026	0.016	*0.098*
EF Higher score=better performance N=3039	1	0.008	0.002	**<0.001**	-0.003	0.002	0.173
	2	-0.001	0.002	0.724	0	0.002	0.988
	3	0	0.002	0.993	0	0.002	0.766
CFT Higher score=better performance N= 3190	1	0.041	0.011	**<0.001**	-0.013	0.011	0.226
	2	-0.013	0.01	0.179	-0.003	0.009	0.714
	3	-0.007	0.011	0.504	-0.003	0.01	0.749
Processing speed Higher=slower performance N= 974	1	0.001	0.002	0.706	0.001	0.002	0.752
	2	0.001	0.002	0.662	-0.001	0.002	0.657
	3	0	0.002	0.981	-0.002	0.002	0.396
SPIN Lower threshold= better threshold for hearing N=1453	1	-0.034	0.013	**0.014**	0.022	0.015	0.139
	2	-0.002	0.015	0.902	0.01	0.015	0.501
	3	-0.001	0.017	0.931	0.006	0.015	0.674

$PM_{2.5}$ = particulate matter < 2.5 micrometres in diameter. PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nitrogen Dioxide. O_3 = ozone. Lden = 24-hour A-weighted equivalent noise level, +10db penalty for evening and night. Lday = A-weighted equivalent noise level 7am-7pm. BL= baseline. F1= Follow-up-1. SDQ: Strengths and difficulties Questionnaire. EF= executive function. CFT= culture fair intelligence test. SPIN= speech-in-noise threshold. PHQ-9: patient health questionnaire-9. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale. **Bold: p<0.05**. *Italics: p<0.10*

6.1.2.3 Multipollutant models

Models were further adjusted for other exposures, where collinearity allowed. Lden, Leve and Lnight were correlated 99%, therefore models suitable for adjustment by Lden were not additionally adjusted for Leve and Lnight in separate models. Multi-pollutant models which include both pollution and noise terms are limited to the sample within London for which pollution data could be generated, therefore they are not additively derived from the noise models above and reflect a smaller sample (the results of the noise exposure only models for the smaller pollution sample, which may be more directly comparable with the multi-pollutant models, can be found in Appendix IX). Where the addition of the other exposure significantly improved the model fit or altered the significance of exposure terms ($p<0.05$),

exposure effects have been reported below in table 16, with the other models tested to be found in Appendix X.

$PM_{2.5}$ and PM_{10} were no longer associated with follow-up executive functioning after controlling for ozone (β = 0.01, p = 0.37 and β = 0.01, p = 0.24, respectively) which was negatively correlated and strongly associated with worse executive function development (surviving adjustment for $PM_{2.5}$, PM_{10}, and NO_2 individually, whilst removing significance for all three effects). After controlling for ozone and Lden, $PM_{2.5}$ exposure between baseline and follow-up was associated with slower processing speed development between baseline and follow-up (controlling for ozone β = 0.04, p = 0.01, and for Lden β = 0.03, p =0.04). Similarly, PM_{10} became associated with slower processing speed at follow-up (β = 0.03, p =0.03) once controlling for ozone (which was negatively correlated with PM_{10}). A trend ($p<0.10$) association between $PM_{2.5}$ and fewer baseline SDQ problems became significant after controlling for Ozone and Lden.

NO_2's significant association with better follow-up executive function did not remain after controlling for ozone. However, ozone's association with worse follow-up executive function (Model 3: β = -0.05, $p < 0.001$), was resilient to adjustment for PM, NO_2, Lden and Lday. Ozone was also associated with slower follow-up processing speeds after adjusting for $PM_{2.5}$.

The trend association of Lden with baseline internalising problems became significant controlling for $PM_{2.5}$ or NO_2 (β = 0.12, p = 0.03). In a model containing Lden and Lday, the former became associated with better baseline intelligence and the latter with worse baseline intelligence (Lden β = 0.23 p = 0.01; Lday β = -0.32, p = 0.04). Greater follow-up

externalising problems were significantly associated with Lday exposure in models adjusted for NO_2 or Lden (adjusted for NO_2, β =0.20, p = 0.04).

Table 16. Multi-pollutant models where the direction or significance of the single-exposure effect was altered

Outcome	Additional exposure	**PM2.5** β(p) Additional exposure β(p)	**PM10** β(p) Additional exposure β(p)	**NO2** β(p) Additional exposure β(p)	**Lden** β(p) Additional exposure β(p)
Baseline outcomes					
SDQ N=3057	+ O_3 +Lden	**-0.24(<0.05)** O_3: -0.18(0.19) **-0.23(0.04)** Lden: *0.18(0.06)*			
SDQ Internalising N=3057	+Lden	*-0.13(0.06)* **Lden: 0.11(0.04)**		-0.12(0.12) **Lden: 0.12(0.03)**	
SDQ Externalising N=3057	+O_3	*-0.14(0.06)* **O_3: -0.18(0.03)**	*0.12(0.06)* O_3: -0.09(0.29)		
CFT N=3188	+Lday				**0.23(0.01)** **Lday: -0.32(0.04)**
Follow-up outcomes					
SDQ Externalising N=2967	+Lday			*-0.12(0.07)* **Lday: 0.20(0.04)**	-0.11(0.20) **Lday: 0.29(0.04)**
GAD-7 N=2433	+O_3 +Lden		**0.21(0.04)** O_3: 0.08(0.59) *0.16(0.10)* Lden: 0.12(0.20)		
EF N=3095	+O_3 +Lden	0.01(0.37) **O_3:-0.06(<0.001)** **0.03(0.04)** Lden: -0.01(0.37)	0.01(0.24) **O_3:-0.06(<0.001)**	0.02(0.10) **O_3:-0.04(0.01)**	
Processing speed N=940	+O_3 +Lden	**0.04(0.01)** **O_3: 0.03(0.04)** **0.03(0.04)** Lden: -0.01(0.35)	**0.03(0.03)** O_3: 0.02(0.12)		

$PM_{2.5}$ = particulate matter < 2.5 micrometres in diameter. PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nitrogen Dioxide. O_3 = ozone. Lden = 24-hour A-weighted equivalent noise level, +10db penalty for evening and night. Lday = A-weighted equivalent noise level 7am-7pm. BL= baseline. F1= Follow-up-1. SDQ: Strengths and difficulties Questionnaire. EF= executive function. CFT= culture fair intelligence test. SPIN= speech-in-noise threshold. PHQ-9: patient health questionnaire-9. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale. **Bold: p<0.05**. *Italics: p<0.10*
Models adjusted for sex, age, ethnicity, school type, NSSEC, first language, parental education, smoke

6.1.2.4 Model fit and robustness of effects

Overall, $PM_{2.5}$, PM_{10}, and Lden exposure conferred a combination of beneficial and harmful effects, NO_2 beneficial effects only, and Ozone and Lday harmful effects only. Most Q-Q plots were consistent with some right skewedness, but assumptions of equality of variances across the sample (levene's test) were met for all outcomes except executive function. Plots of residuals against fitted values did not show concerning levels of heteroscedasticity. The addition of covariates and then the addition of random effects significantly increased goodness of fit, with the exceptions of models predicting processing speed and odds of UHR status which were not improved. Therefore, a post-hoc analysis was run of backward step regression predicting processing speed and CAPE from all independent variables.

After FDR correction to single and multi-exposure models, only follow-up associations with executive function were significant. Ozone was significantly associated with executive function development between baseline and follow-up detrimentally, and PM_{10}, $PM_{2.5}$, and NO_2 were associated with better executive function development. However, in FDR corrected multi-exposure models, ozone remained significant whilst controlling for the 'beneficial' pollutants PM_{10}, $PM_{2.5}$, and NO_2 (which are negatively correlated to ozone), which became non-significant.

6.1.3 Backward step multiple regression of processing speed and CAPE outcomes

Processing speed and psychosis symptoms were not better predicted by additional levels of adjustment, and the latter was not predictable from exposure variables. Therefore, as a post-hoc exploratory analysis, all independent variables were entered into backward step regressions predicting processing speed and the presence of subsyndromal psychotic symptoms from all independent variables (table 17).

Table 17. Results of backward step multiple regression predicting processing speed and risk for psychosis status

Outcome	Predictors	β (p)
Processing speed baseline	Baseline Ozone + Ethnicity + School	Ozone: 0.04(0.03)
Processing speed follow-up	Follow-up sleep problems + Sex + School	
CAPE-P15 Psychosis risk (2019-2021)	Follow-up sleep problems	

6.1.4 Sensitivity analyses

The addition of the English Index of Multiple Deprivation decile as a covariate, to adjust for deprivation at neighbourhood level, made a negligible difference to results (appendix XIII). Models were also run with just school (as opposed to school + neighbourhood) as a random effect which made a negligible difference to effect sizes and model fit. When models were run limiting the sample for the noise analysis to those included in pollution models (within the M25), results were similar, although some effects were weaker due to lower power (presented in Appendix IX). The use of precise geocodes for which there was certainty of accuracy produced largely consistent results (see Appendix XIV for full tables). Although some confidence intervals were widened due to lower power, regression coefficients were largely similar in size and direction. However, a trend association between fewer baseline externalising problems and Ozone gained significance, and the trend association between NO_2 and fewer follow-up SDQ behavioural and specific internalising problems dissipated. For the data with certain/precise geocodes, compared to the broader sample, PM_{10} became more strongly associated with anxiety in model 2 ($p = 0.048$ and $p= 0.029$) and strengthened from a trend effect to a significant effect ($p = 0.056$ and $p = 0.029$) for Model 3 (including random effects).

As a sensitivity analysis and for comparability with prior research, analyses were run with outdoor and indoor estimates of exposure only (rather than exposure estimates incorporating indoor and outdoor sources of air pollution). Table 18 shows the correlations between estimates of overall hybrid exposure (including indoor, outdoor, and in transit), indoor, and outdoor only. The results of these analyses have been provided in Appendices XV-XVIII. Most effects were consistent between indoor, outdoor and hybrid exposure. Some effects that were significant in hybrid but not replicated outdoors (Appendix XV) were: $PM_{2.5}$ and baseline SPIN, PM_{10}/NO_2 and baseline executive function, PM_{10} and GAD-7. For outdoor exposure, PM_{10} became significantly associated with worse baseline SDQ problems for outdoor exposure (Model 3 $\beta = 0.25$, $p = 0.035$), remaining significant for externalising and showing a trend effect for internalising.

Follow-up associations with executive function remained associated in both outdoor and indoor models (Appendix XV and XVI), including the negative effect for ozone. Ozone was associated with worse executive function development for all five pollution metrics (hybrid with indoor sources, hybrid without indoor sources, indoor with indoor sources, indoor without indoor sources, outdoor). Baseline 'beneficial' effects for executive function were only associated for indoor exposure with indoor sources and hybrid exposure with indoor sources, suggesting these were driven by indoor sources of air pollution. The associations observed in multi-pollutant models between $PM_{2.5}/PM_{10}$ and slowed follow-up processing speed development were replicated indoors, reaching significance in models of indoor air pollution without indoor sources (single pollutant models, Model 2: $PM_{2.5}$ $\beta = 0.03$, $p = 0.037$/ PM10 $\beta = 0.03$, $p = 0.01$).

Indoor effects were more similar to the hybrid exposure than outdoor exposure (Appendix XVI). Outdoor exposure was associated with more harmful and fewer beneficial effects. Differences between indoor and outdoor air pollution effects suggest that, similar to noise (when comparing Lday at school, Leve at home), the context of exposure moderates its impact. The effects for outdoor exposure, indoor exposure without indoor sources, and overall hybrid exposure without indoor sources were more consistent with hypotheses (Appendices XVII and XVIII); the 'beneficial' association of baseline executive function and air pollution was removed, and harmful associations with follow-up processing speed ($PM_{2.5}$ and PM_{10}) and anxiety (PM_{10}) were strengthened. Associations with follow-up executive function remained mostly present, however for Ozone these were still the strongest effects and in the harmful direction (noting that multi-pollutant models suggested Ozone was driving the 'beneficial' associations found in single pollutant models). Without indoor sources, indoor $PM_{2.5}$ became associated with worse baseline intelligence (Model 2 $\beta = -0.15$, $p = 0.038$). Only outdoor and hybrid exposure were associated with mental health.

Table 18. Correlations between air pollution metrics

Average 2013-2018	Hybrid exposure & Inside exposure *r*(N)	Hybrid exposure & Outside exposure *r(*N)	Outside exposure & Inside exposure *r*(N)
$PM_{2.5}$	0.968 (3527)	0.884(3527)	0.813(3528)
PM_{10}	0.900(3527)	0.668(3527)	0.432(3528)
NO_2	0.924(3527)	0.843(3527)	0.711(3528)
Ozone	0.867(3527)	0.767(3527)	0.482(3528)
$PM_{2.5}$ = particulate matter < 2.5 micrometres in diameter. PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nitrogen Dioxide.			

6.1.5 Non-linear relationships

The relationships in table 19 were better explained with non-linear terms for exposure (-2 AIC units, or a change to or from significance of the exposure term). Models for which exposure was associated $p < 0.05$ with the outcome are presented in Figures 16-19. Remaining GAM plots can be found in Appendix XIX. NO_2 and Lday were the best suited

exposure to nonlinear modelling and baseline outcomes were better suited than follow-up outcomes. Average PM_{10} significantly better explained odds of UHR status using a bimodal distribution, which showed a trend effect although neither model was significant. Baseline total and internalising problems were better predicted by a negative association (higher exposure =fewer problems) up to 12 μg/m² NO_2 (scale of IQR * 1IQR NO_2 (1.91)) and then a positive relationship (more exposure = more problems, range 4.3-17.6) than a linear negative association, with the nonlinear exposure term for BL internalising problems being significant.

Lden better predicted baseline SDQ and baseline externalising problems in a GAM model (the exposure term for the latter was significant) with increasing problems up to around 60dB and plateauing relationship after (range 43.8-81.2). A similar distribution was observed for baseline executive function and ozone which significantly bettered the linear negative association (however in this case higher executive function scores considered beneficial, whereas higher SDQ scores are considered more problematic) however neither exposure term was significant. The relationship between Ozone and baseline intelligence was also significantly better explained using a similar GAM distribution, though neither exposure term was significant. The relationship between NO_2 and baseline executive function was significantly better explained by a GAM than a linear model (though both exposure terms were significant) where exposure up to 8.5 μg/m² was associated with worse executive function, followed by a positive association up to 14 μg/m², then a negative association (range 4.3-17.6).

For baseline SDQ score, baseline externalising problems, and follow-up processing speed, Lday was associated with a similar pattern which was a significantly better fit than a linear model where worse outcomes were observed with increasing volume up to around 50dB

(sometimes appearing as positive or negative association depending on the outcome), then broadly plateauing but with a trough around 60dB (range 39.1-78.5). Baseline SPIN thresholds were significantly associated with Lday in a non-linear model and fit the data significantly better than a linear model, however the irregular wiggle shape (4 peaks and 3 troughs) implies that the association was not perfectly suited to these modelling specifications. CAPE UHR status was significantly better explained by Lday in a GAM model than a linear model, with a peak risk around 59dB, but neither exposure term was significant.

Table 19. Comparison of GLMs with GAMs, when GAM improved model fit

	Linear model $^{adj}R_2$(n), AIC, p(exposure)	GAM model $^{adj}R_2$(n), AIC, p(exposure)
PM_{10} and CAPE-P15	12.3% (123), 160.22, 0.21	*12.4% (123), 156.74, 0.09*
NO_2 and BL SDQ	4.0% (3057), 19027.01, 0.62	*4.2% (3057), 19023.15, 0.09*
NO_2 and BL internalising	2.5%(3057),15780.8, 0.37	**2.7%(3057), 15775.19, 0.04**
NO_2 and BLEF	**10.4%(3158), 6074.33, 0.02**	**10.6% (3158), 6071.01, 0.02**
Ozone and BLEF	10.3%(3158), 6078.35, 0.26	10.5% (3158), 6076.32, 0.14
Ozone and BLCFT	6.6%(3188), 16978.1, 0.63	6.8% (3188), 16975.79, 0.18
Lden and BLSDQ	4.0% (3057), 19024.98, 0.13	*4.2% (3057), 19022.71, 0.07*
Lden and BL externalising	5.6% (3057), 16031.91, 0.39	**5.8%(3057), 16026.93, 0.04**
Lday and BLSDQ	4.0% (3057), 19026.76, 0.48	4.2%(3057), 19024.4, 0.15
Lday and Bl externalising	5.6% (3057), 16032.43, 0.63	*5.8%(3057), 16027.83, 0.07*
Lday and BLSPIN	2.6% (2483), 12524.45, 0.51	**3.9% (2483), 12498.65, <0.001**
Lday and F1 processing speed	16.4% (940), 625.89, 0.56	*17.5%(940), 619.20, 0.06*
Lday and CAPE-P15 Psychosis risk	11.9% (123) 129.49, 0.56	9.1%(123), 159.22, 0.17

PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nitrogen Dioxide. Lden = 24-hour A-weighted equivalent noise level, +10db penalty for evening and night. Lday = A-weighted equivalent noise level 7am-7pm. BL= baseline. F1= Follow-up-1. SDQ: Strengths and difficulties Questionnaire. EF= executive function. CFT= culture fair intelligence test. SPIN= speech-in-noise threshold. PHQ-9: patient health questionnaire-9. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale. **Bold: p<0.05**. *Italics: p<0.10*

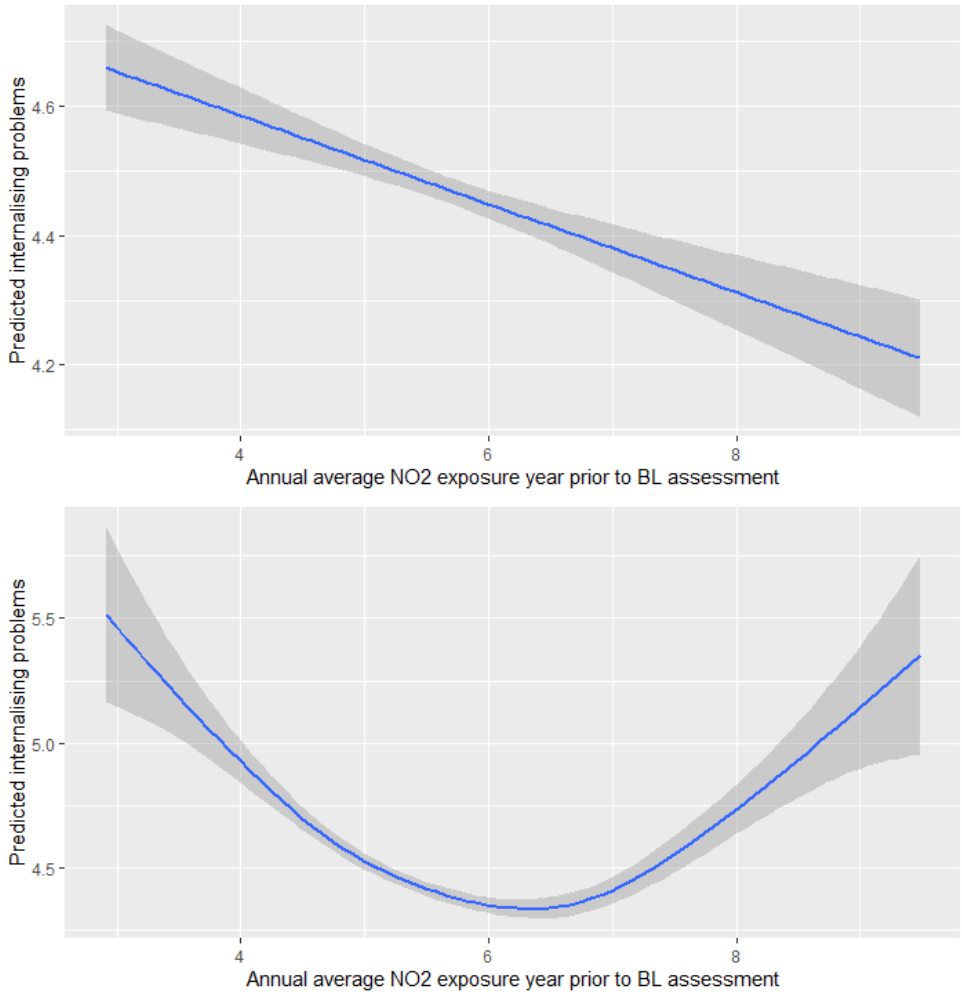


Figure 16. Predicted relationship between NO_2 in the year prior to baseline (BL) assessment and baseline SDQ-assessed internalising problems (a) in an adjusted linear model, (b) in an adjusted GAM

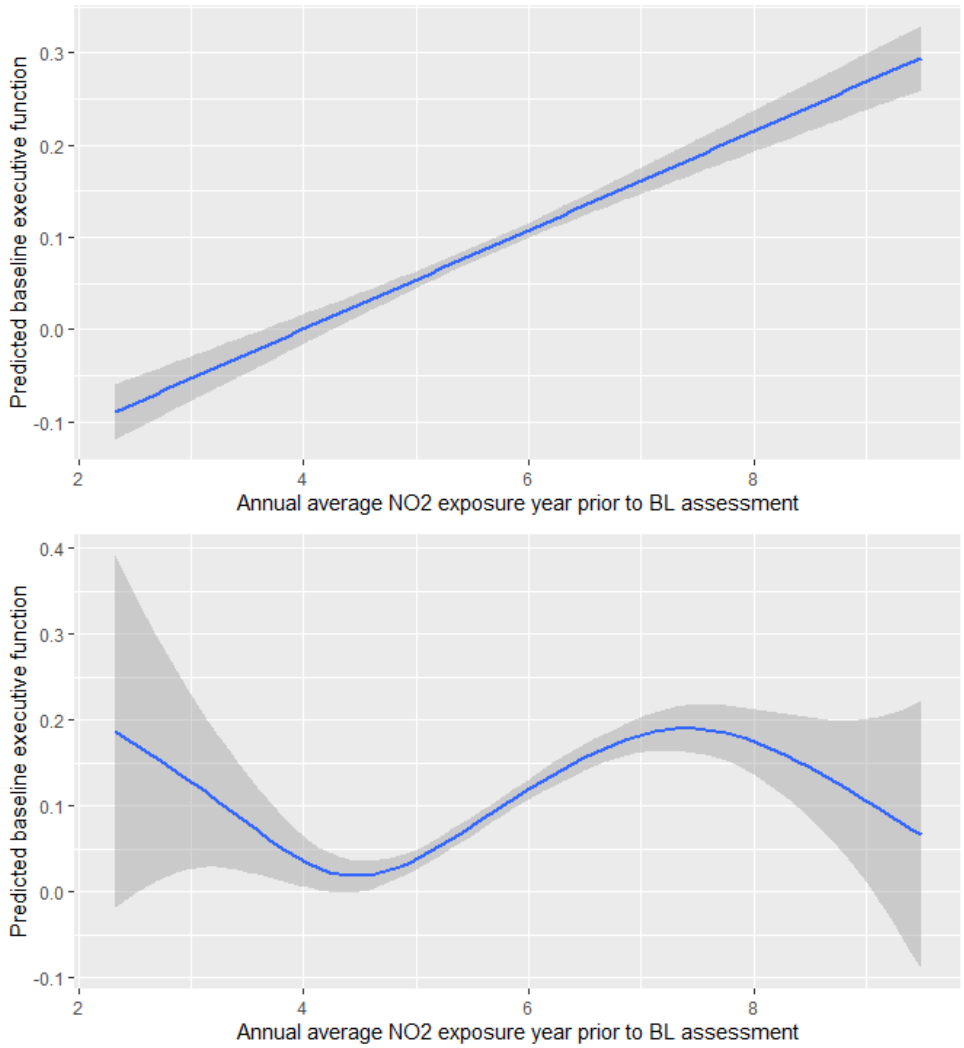


Figure 17. Predicted relationship between NO_2 in the year prior to baseline (BL) assessment and baseline executive function (a) in an adjusted linear model, (b) in an adjusted GAM

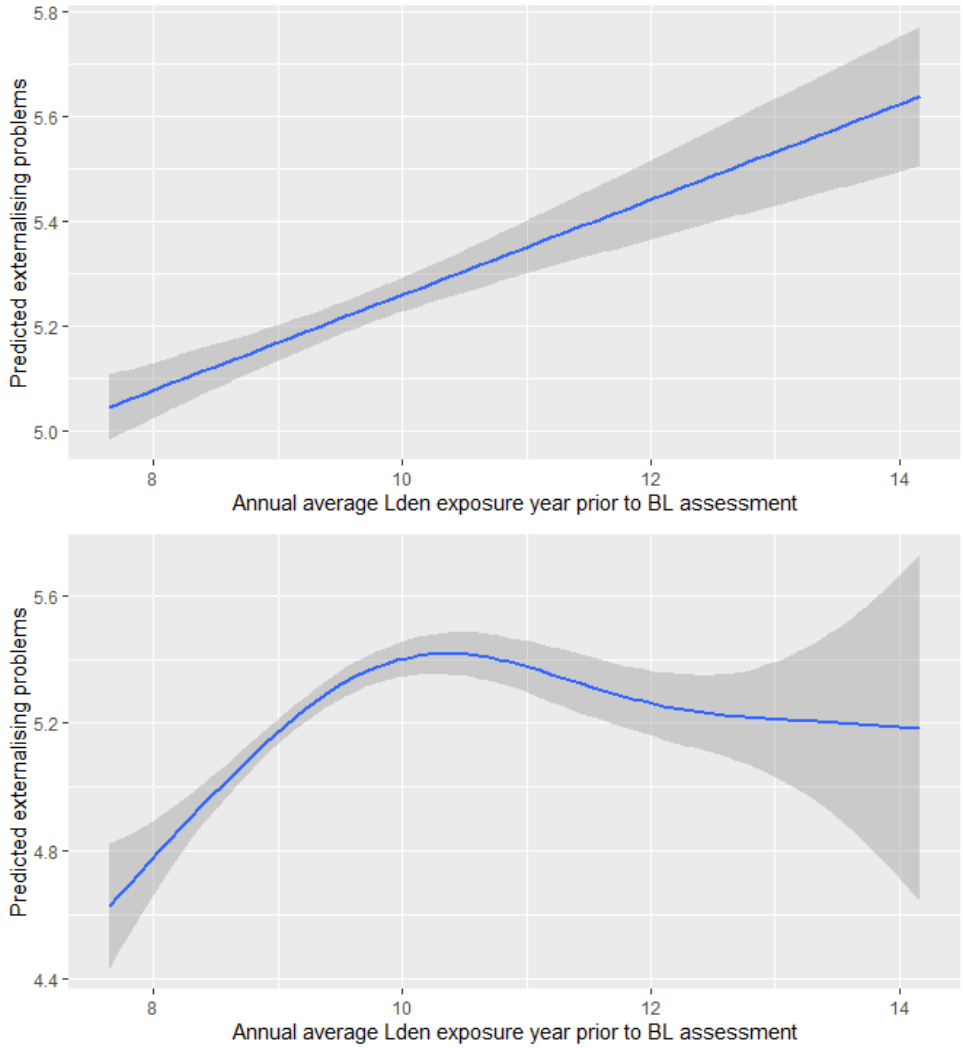


Figure 18. Predicted relationship between Lden in the year prior to baseline (BL) assessment and baseline SDQ-assessed externalising problems (a) in an adjusted linear model, (b) in an adjusted GAM

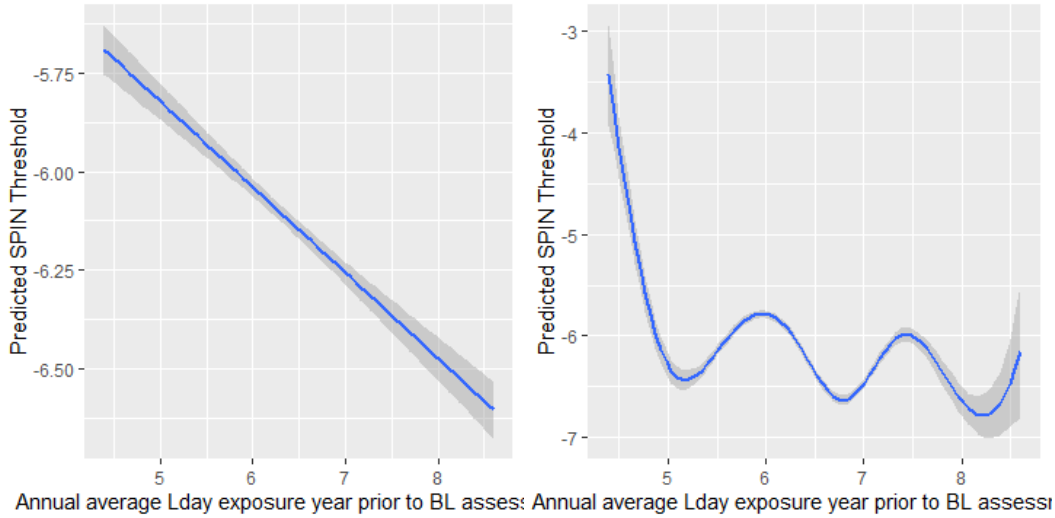


Figure 19. Predicted relationship between Lday in the year prior to baseline (BL) assessment and baseline SPIN threshold (a) in an adjusted linear model, (b) in an adjusted GAM

6.2 Discussion

6.2.1 Summary

Between baseline and follow-up, exposure to Ozone was associated with worse executive function development. PM_{10} was associated with higher baseline externalising problems and higher follow-up anxiety symptoms. In multipollutant models, particulate matter (PM_{10} and $PM_{2.5}$) were associated with slower changes in processing speed between baseline and follow-up, and Lden and Lday were associated with converse effects on baseline intelligence (Lden beneficial, Lday harmful). Adjusting for NO_2 or Lden, Lday was associated with worsening follow-up externalising problems. Adjusting for $PM_{2.5}$ or NO_2, Lden was associated with greater baseline internalising problems. However, $PM_{2.5}$ was associated with better speech-in-noise perception at baseline, and PM_{10} and NO_2 with better executive function. In multi-pollutant models $PM_{2.5}$ became associated with fewer baseline behavioural difficulties. Ozone's detrimental longitudinal effect on executive function development was the most robust finding.

Non-linear relationships were also tested. Using GAMs, Increased Lden was significantly associated with increasing baseline externalising problems up to a threshold of around 60dB. Associations with Lday exposure were most likely to be better explained in a nonlinear model, with a broadly detrimental impact on processing speed and mental health which again seemed to increase more at lower volumes and plateau or trough above a certain volume. Non-linear associations with NO_2 did the opposite, seeming beneficial with increasing exposure to a point before appearing to be detrimentally associated.

6.2.2 Interpretation

Air pollution exposure was associated with higher externalising problems, anxiety, and worse processing speed and executive function over time. However, the results somewhat differed from hypotheses with respect to some air pollutants and cognitive outcomes. Follow-up outcomes were more likely to be associated with exposure in a detrimental way, which may suggest a special vulnerability period during early-to-mid adolescence. These models are more longitudinal in design than baseline models; both results reflect exposure prior to outcome, however the follow-up models controlled for baseline effects (except PHQ-9 and GAD-7, which were measured at one timepoint) so are indicative of change in response to exposure, which is a stronger design for inferring causality and reducing confounding by the potential level of the outcome before measurement and exposure, or by effects of earlier exposure (which will be highly correlated to baseline exposure).

Some of the surprising effects were driven by indoor exposure (with indoor sources of air pollution) and most prior research has used outdoor exposure, so this may help explain how effects differed. Indoor with source and hybrid exposure were most similar, which is not surprising because most participants spent most of their time annually indoors, so this contributed more to their exposure assessment. However, most prior literature has utilised outdoor exposure assessment. This may explain why most prior literature found only harmful effects and why some of these findings deviate from expectations. Outdoor effects tended to be more detrimental and consistent with prior research, and the correlations between indoor and outdoor were weakest (<50%) for PM10 and ozone. The indoor estimations of these exposures are also considered less reliable due to the reaction of indoor ozone with other species, and technical assumptions about the size distribution of PM_{10} (which can vary more widely than $PM_{2.5}$) affecting infiltration rates. Therefore, the outdoor only air pollution results may be more reliable for PM_{10} and ozone.

Slowed processing speeds with air pollution exposure during adolescence were found for both indoor and outdoor exposure. When indoor and hybrid exposure without indoor sources of air pollution were tested, results were more in keeping with hypotheses in terms of largely detrimental effects upon cognition and mental health. However, baseline speech-in-noise and $PM_{2.5}$ remained associated as a 'beneficial' association. $PM_{2.5}$, PM_{10} and NO_2 remained associated with better follow-up executive function, but this is probably attributable to ozone, which was strongly related to worsening executive function, negatively correlated to other air pollutants, remained associated where the other pollutants did not in multi-pollutant adjusted analysis, and did not contribute to indoor air pollution via indoor sources (just infiltration) in this modelling.

Surprisingly, indoor sources of air pollution were associated with better baseline executive function, even controlling for ozone, and some of the detrimental effects observed for indoor air pollution without sources (from outdoor air exchange) and outdoor air pollution were reduced when including indoor sources. This may be due to confounding or reverse causation from having parents who cook more in the house; more mentally well or intelligent parents and cooking more often may be associated with better nutrition and therefore better development. Indeed, those living in households that cooked more frequently were more likely to have parents who went to university and be of a higher socioeconomic status. However, despite being contrary to hypotheses, it is also possible that some 'beneficial' effects of air pollution could occur; for example, an experimental study has observed acute exposure to nitrogen dioxide to lower blood pressure, which is a risk factor for brain development (Floyd et al., 2020, Lurbe et al., 2016). Further, nitrogen oxide species are highly spatially correlated (Levy et al., 2014), and nitric oxide is clinically administered for

various cardiopulmonary disorders due to its potential to relax vascular smooth muscle, lower pulmonary hypertension, and improve oxygenation (Bhatraju et al., 2015). Perhaps the patterns seen using non-linear modelling are indicative that nitrogen dioxide is beneficial to a point by lowering blood pressure, but becomes detrimental at high levels. However, there is no proven benefit of chronic ambient exposure to these air pollutants for health or brain development. The effects on mental health were more consistently detrimental, especially for noise, although $PM_{2.5}$ was associated with fewer baseline behavioural problems when controlling for Lden and ozone. Only outdoor and hybrid exposures, and not indoor exposures, were associated with mental health.

In non-linear models, lowest levels of noise were associated with worse speech-in-noise perception, which may mean very low levels do not enable the development of keen hearing that easily distinguishes sounds, or that very low levels are associated with less access to resources and stimulation. However, controlling for noise did not remove the association with $PM_{2.5}$ suggesting it is not driven by corelations with noise.

Models including random effects of school and home neighbourhood were consistent with associations at lower levels of adjustment, although most were weaker or lost significance. This suggests these higher-order effects contributed somewhat to associations between exposure and outcome, but did not entirely explain them. The fact associations did not neutralise, change in direction, or emerge, suggests there was not strong confounding by random effects. Neighbourhood deprivation did not influence results substantially, so neighbourhood quality was likely not confounding associations through covariance with pollution/noise.

Differences between different metrics of exposure (noise at day, evening, night, and overall, and hybrid, indoor and outdoor), and between these findings and prior research (which predominantly utilised home without school and outdoor exposure), could be a reflection of confounding differences or could reflect differences in the effects themselves depending on context. Differences between Lday (mostly reflecting school-based noise exposure) and Lden (mostly reflecting home exposure in the evening and night-time), show that the nature, direction, and severity of impact of noise exposure may depend on the context in which the adolescent is exposed to noise. Differences between indoor/outdoor air pollution effects could suggest the same is true for air pollution exposure, though these long-term psychological effects are generally hypothesised to mechanistically occur over time via impacts on the body and brain rather than in psychological and physiological response to contemporaneous exposure(Allen et al., 2017, Braithwaite et al., 2019a, de Prado Bert et al., 2018, Genc et al., 2012). For noise, contextually different impacts may seem more intuitively plausible; as noise is largely theorised to influence psychological outcomes through immediate psychological and physiological effects, that may disrupt other factors like learning, sleep, stress hormones, and social interactions, what someone is doing at the time of exposure and how this is affected by noise will vary (Hahad et al., 2019, Stansfeld et al., 2005, Sygna et al., 2014). This means that long term impact of disruption by noise may depend on what one is doing at the time. The fact only day noise was associated with significant negative effects (in the multi-pollutant models) suggests that school exposure during studying hours is particularly detrimental. Some negative trend mental health effects were observed for Lden, Leve and Lnight exposure (largely occurring at home) which were actually strongest in the evening, suggesting perhaps disruption of relaxing activities may be more detrimental than sleep disruption. However, given the associations between air pollution and psychological experience are not fully understood, and that there is evidence of

concurrent effects of air pollution on cognition (Chen et al., 2017, Roth, 2016) it may be that context (time/place) of exposure also matters, which could go some way to explaining differential effects between indoor and outdoor sources of the same air pollutants.

The stronger longitudinal design pertaining to follow-up outcomes means that we can hold more confidence in the results for follow-up outcomes than baseline outcomes. The fact detrimental effects were observed more consistently at follow-up than baseline could be due to moderation of the associations by age/developmental stage. Perhaps some ages are more vulnerable to effects of air pollution or noise due to the psychological or physiological changes that are occurring at that time, similar to how nutrition has an impact on height in childhood but not in adulthood (Perkins et al., 2016). Effects on anxiety at follow-up reflected static response (rather than change) in response to prior exposure, but were also associated with air pollution.

6.2.3 Strengths and limitations

This is the first study combining school and home-based exposure to air pollution and noise in relation to mental health and cognition in adolescents. The hybrid exposure to air pollution, which includes both indoor and outdoor sources of exposure in transit and leisure activities (as opposed to just school or residential address), is especially novel. Analyses were very comprehensive in the different models run for each outcome and exposure, and the SCAMP study cohort has the benefits of being longitudinal, diverse, representative, and, given this, a relatively large sample.

With this number of models, the problem of multiple comparisons arises (where some significant associations become statistically likely due to chance). Given that there were many outcomes with 3 main models per outcome, and the exploratory nature of this research

(as an undeveloped field, therefore without exact hypotheses beyond expecting 'detrimental effects') Bonferroni correction was considered excessive; however, it is important to remember 1/10 associations are likely to arise as a significant or trend effect by chance. The proportion of observed effects was higher than this, and consistency with other findings and prior research can be used as a heuristic for judging validity or spuriousness. Here, associations were interpreted as meaningful when they illustrated a consistent overall picture across models, rather than relying on one association or effect to make conclusions.

The sample sizes in the main models were larger than many studies (N=1000+) however, the CAPE outcome analyses were underpowered. In multiple imputation, missForest was chosen for accuracy, ability to predict categorical data, and suitability for entering the same sample into different models. However, unlike other methods, the uncertainty of the imputed values is not carried into the model (just the predicted value) and is therefore not reflected in the confidence intervals of results which may be narrower (Shah et al., 2014). Further, this study focussed on one city (London) in one country (UK) so the results may not generalise to other parts of the world, for example LMICs, where pollution and noise is often higher in cities and different contextual factors are operating. Additionally, although a number of plausible confounders were selected for adjustment, there may be other confounders associated with air pollution and outcomes, E.g., young people talked about greenspace and antisocial behaviour influencing their wellbeing in interviews. Noise exposure reflected traffic noise only and not factors like community noise, or aircraft/rail noise, which may be particularly prominent in parts of London and could have differential effects. Finally, although personally monitored exposure data from a sub-study of the cohort was used to validate the modelling, exposure estimates still largely rely on modelled rather than measured exposure which creates a higher level of uncertainty.

6.2.4 Summary

This study found longitudinal detrimental effects of ozone upon executive function development and of PM_{10} upon mental health. There were also surprisingly beneficial effects of air pollution on baseline executive function, the latter of which were driven by indoor sources of air pollution, for which there was a great deal of missing data and possible sociodemographic confounding. In multi-exposure models, daytime noise was associated with worse baseline intelligence and the development of follow-up externalising problems, and $PM_{2.5}$ and PM_{10} were associated with detrimental effects on processing speed development.

Chapter 7: Moderation and mediation of epidemiological associations

As established in chapters 1-3, there has been a dearth of prior literature evaluating the other factors involved in associations between air pollution and noise and adolescent psychological outcomes. Moderating factors may go some way to explaining heterogeneities in the prior literature and in the results of these analyses (chapter 6) and have important implications for health inequalities. Specifically, sociodemographic factors and other exposures (high air pollution or noise) could confer physiological and psychosocial vulnerability which is then compounded by environmental exposures (Kristiansson et al., 2015, Olvera Alvarez et al., 2018).

Although results of the main analyses were modest, several factors meant that it was sensible to explore moderation by sociodemographics and other exposures, as this could potentially add to our understanding of the associations: their apparent complexity (varying with indoor/outdoor setting, time of exposure, and the inclusion of other exposures), the existence of trend effects, the mixture of results consistent and inconsistent with past literature, and the theoretical plausibility of effect modification (see sections 1.2.1 and 1.3).

Mediation analyses are important to strengthen causal inference and design effective interventions. Health and psychological factors are associated with both exposure to air pollution and noise, and mental health and cognition, so are plausible mediators to investigate and further our understanding of the mechanistic underpinnings of these relationships (WHO, 2016, Stansfeld et al., 2005, Wells and Vaughn, 2012). As the a priori hypothesised mechanisms applied to detrimental associations, based on theory and literature, only some of

the observed associations in Chapter 5 ('detrimental' but not 'beneficial' associations) were amenable to the planned mediation analyses.

Many of the following analyses had reduced sample sizes due to utilisation of subgroups or mediators with missing data, but it was important results were comparable with the effects of primary models to discern the impact of moderation/mediation. As random effects did not substantially change the effect sizes of interest, the subgroup and mediation analyses were conducted at the second level of adjustment (individual level covariates).

7.1 Results

7.1.1 Moderation analyses

Interaction terms were added to regression models (including adjustment for fixed individual-level and random effects) to identify moderating variables. The following interaction terms were tested: baseline below average IQ (yes/no), gender, ethnicity, independent school (yes/no), religion (yes/no), high/low baseline PM_{10} (for noise analyses), and high/low baseline Lden (for air pollution analyses). The results are presented in Appendix XII. All these analyses were run using hybrid exposure with indoor sources.

When an interaction term in a model was significant (Appenix XII), indicating that the effect varied by group, stratified models were run for the individual groups in order to directly identify and compare the differential trends between them. Where a significant association was observed within a subgroup, results are reported in table 20 (where none of the subgroup effects were significant, these were removed in the interests of brevity and relevance). As results for Lden, Leve and Lnight were equivalent (with 99% correlations between them), only moderation analyses for Lden and Lday were run. This also delineates school and home

exposure, as Lday exposure largely occurs at school and Lden exposure largely occurs at home, with weighting for periods (Leve and Lnight) typically spent at home.

In subgroup analyses (table 20), PM_{10} exposure was significantly associated increased with CAPE-assessed psychosis risk in students with below average IQ (below average IQ OR = 6.23 (95% CI: 1.46, 52.0), average-above average IQ OR = 1.08 (95% CI: 0.48, 2.57)), and ozone exposure was associated with significantly higher odds of psychosis risk in boys (OR = 6.96 (95% CI: 1.66, 46.0)) but significantly lower odds in girls (OR = 0.26 (95% CI: 0.07, 0.79)). Despite a small sample of mixed-race participants (N=293), noise seemed to be associated with detrimental associations with intelligence (Lday β = -0.81, $p = 0.006$, Lday β = 0.41, $p = 0.02$) and NO_2 with detrimental processing speed effects ((β = 0.12, $p = 0.04$) specifically in these groups.

The average to above average IQ group seemed to drive a number of 'beneficial' associations observed in the wider sample: between PM_{10} and improved executive function (below average IQ β = 0.01, $p = 0.73$, average-above average IQ β = 0.04, $p = 0.001$), NO_2 and improved executive function (below average IQ β = 0.03, $p = 0.09$, average-above average IQ β = 0.07, $p < 0.001$), and NO_2 and reduced behavioural difficulties (below average IQ β = -0.04, $p = 0.81$, average-above average IQ β = -0.33, $p = 0.01$). Longitudinal changes in SPIN were not associated with exposure to $PM_{2.5}$ in the whole sample analysis (though year-prior $PM_{2.5}$ was associated with baseline SPIN) however they were associated in participants average to above average IQ (β = - 0.28, $p = 0.02$), but not those with below average IQ (β = 0.12, $p = 0.49$) (Table 20). Low noise exposure was associated with a stronger 'beneficial' effect of NO_2 on executive function. High vs low PM_{10} did not interact

with exposure to noise (Appendix XII, p360-361, p373-374) so subgroup analyses were not run.

A number of effects were observed in female students only without a clear direction in terms of benefits/harms: $PM_{2.5}$ was associated with fewer baseline behavioural problems and $PM_{2.5}$ and PM_{10} with better executive function, but ozone was associated with lower baseline intelligence (table 20). Some effects were observed in specific ethnic groups and not others, usually white students. Again, some of these were beneficial, e.g. PM_{10} or NO_2 and internalising problems, executive function, or fluid intelligence, and some were detrimental, e.g. Lday and depression, ozone and processing speed or executive function, without a clear or meaningful pattern of effects (table 20). Independent school students also drove a number of effects, some harmful and some beneficial, but without following a clear pattern (table 20). Some mental health and intelligence effects were only present in religious or non-religious students but again without a clear pattern in terms of benefits/harms (table 20).

Table 20. Subgroup analyses of exposure to air pollution or noise and psychological outcomes where a subgroup effect reached significance

Association *Hybrid air pollution exposure	Main unstratified effect* *Adjusted for individual-level covariates *β(p) unless otherwise stated	Below average IQ at baseline β(p) Average-above average IQ at baseline β(p) *F1 outcomes only	Male β(p) Female β(p)	White β(p) Black β(p) Asian β(p) Mixed β(p) Other β(p) *Subgroup analyses only run for groups with significant interaction terms	Non-independent school Independent school	No belief/ practice β(p) Religious β(p)	Low baseline Lden β(p) High baseline Lden β(p)
PM2.5 and BL SDQ	*-0.19(<0.10)*		0.13(0.45) **-0.41(0.01)**				
PM2.5 and BL EF	*0.02(<0.10)*		-0.02(0.40) **0.06(<0.001)**		-0.001(0.95) **0.09(<0.001)**		
PM2.5 and F1 SDQ	0.12(0.33)					**-0.46(0.04)** 0.02(0.87)	
PM2.5 and F1 SPIN	-0.09(0.39)	0.12(0.49) **-0.28(0.02)**					
PM10 and BL internalising	0.04(0.50)					0.09(0.50) **0.16(0.03)**	
PM10 and EF BL	0.02(0.10)		-0.01(0.22) **0.05(<0.001)**		<0.001(0.97) **0.07(<0.001)**		
PM10 and BL processing speed	0.001(0.95)				*0.02(0.06)* **-0.04(0.01)**		
PM10 and GAD7	**0.10(<0.05)**					**0.52(0.02)** 0.15(0.22)	
PM10 and F1 CFT	0.07(0.22)			**White: 0.27(0.003)** Asian: -0.23(0.19)			

PM10 and F1EF	**0.03(0.01)**	0.01(0.73) **0.04(0.001)**		**White:** **0.06(<0.001)** Asian: <0.01(0.76) Mixed race: -0.04(0.13)			
PM10 and CAPE	OR: 1.39(0.84-2.38)	OR: **6.23(1.46-52.0)** 1.08(0.48-2.57)					
NO2 and BL internalising	-0.06(0.37)			**White: -0.30(0.005)** Black: 0.19(0.29) Asian: 0.16(0.19)			
NO2 and EF BL	**0.03(0.02)**		0.01(0.75) **0.08(<0.001)**	**White:** **0.08(<0.001)** Black: -0.01(0.84)	0.01(0.65) **0.09(<0.001)**		**Low noise:** **0.05(0.02)** High noise: 0.02(0.21)
NO2 and CFT BL	-0.01(0.93)				-0.13(0.16) **0.34(0.003)**		
NO2 and F1 SDQ	*-0.20(0.05)*	-0.04(0.81) **-0.33(0.01)**					
NO2 and EF F1	**0.05(<0.001)**	*0.03(0.09)* **0.07(<0.001)**		**White:** **0.09(<0.001)** Asian: 0.02(0.38) Mixed race: -0.02(0.46)			**0.09(<0.001)** **0.04(0.004)**
NO2 and processing speed F1	0.01(0.59)			White: -0.03(0.16) **Mixed race:** **0.12(0.04)**			
Ozone and BL EF	-0.02(0.26)			**White: -0.04(0.03)** Asian: 0.02(0.57)	0.01(0.46) **-0.07(<0.001)**		
Ozone and BL CFT	-0.04(0.63)		0.21(0.13) **-0.21(0.03)**		0.13(0.22) **-0.32(0.005)**		

Ozone and F1 SDQ	0.16(0.18)			White: -0.05(0.74) **Asian: 0.68(0.007)**			
Ozone and F1 EF	**-0.07(<0.001)**	**-0.06(0.01)** **-0.08(<0.001)**		**White: -0.08(<0.001)** Black: 0.02(0.61)			
Ozone and F1 Processing speed	-0.11(0.13)			**White: 0.05(0.01)** Asian: -0.04(0.18)			
Ozone and CAPE	OR: 0.92(0.47-1.77)		OR: **6.96(1.66-46.0)** **0.26(0.07-0.79)**				
Lday and CFT BL	0.001(0.90)					0.35(0.14) **-0.45(0.003)**	
Lday and F1 externalising	0.01(0.39)				0.01(0.96) **0.50(0.02)**		
Lday and PHQ-9	<0.01 (0.86)			**White: 0.67 (0.03)** Asian: -0.39(0.21) Mixed race: -0.03(0.97)			
Lday and F1 CFT	-0.01(0.18)			White: 0.11(0.47) **Mixed race: -0.81(0.006)**			

Lden and BL CFT	0.01(0.20)					**0.29(0.03)** -0.06(0.50)	
Lden and F1 CFT	<-0.01(0.71)			White: 0.12(0.18) Black: -0.20(0.14) **Mixed race: -0.41(0.02)**			

$PM_{2.5}$ = particulate matter < 2.5 micrometres in diameter. PM_{10} = particulate matter <10 micrometres in diameter. NO_2 = Nitrogen Dioxide. O_3 = ozone. Lden = 24-hour A-weighted equivalent noise level, +10db penalty for evening and night. Lday = A-weighted equivalent noise level 7am-7pm. BL= baseline. F1= Follow-up-1. SDQ: Strengths and difficulties Questionnaire. EF= executive function. CFT= culture fair intelligence test. SPIN= speech-in-noise threshold. PHQ-9: patient health questionnaire-9. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale. **Bold: p<0.05**. *Italics: p<0.10*

7.1.2 Mediation analyses

The following mediators were related to exposure or outcome metrics (p<0.10): sleep difficulties were associated with baseline ozone exposure, follow-up anxiety, executive function, depression, and intelligence. Higher risk of asthma/eczema/allergies was associated with baseline ozone, Lday, $PM_{2.5}$ & PM_{10}, and depression, and BMI was associated with baseline Lday exposure and anxiety. Where a trend or significant association was observed between an exposure and a detrimental follow-up outcome (including multipollutant and subgroup analyses), and there existed a plausible mediator associated with exposure and outcome (p <0.10 significance level), results of mediation analyses are presented in table 21.

Most of the exposure-outcome pairings identified in earlier analyses as associated were not also both associated with a plausible mediator. However, the relationship between PM_{10} and later anxiety was significantly (31%, p = 0.02) explained by earlier externalising problems.

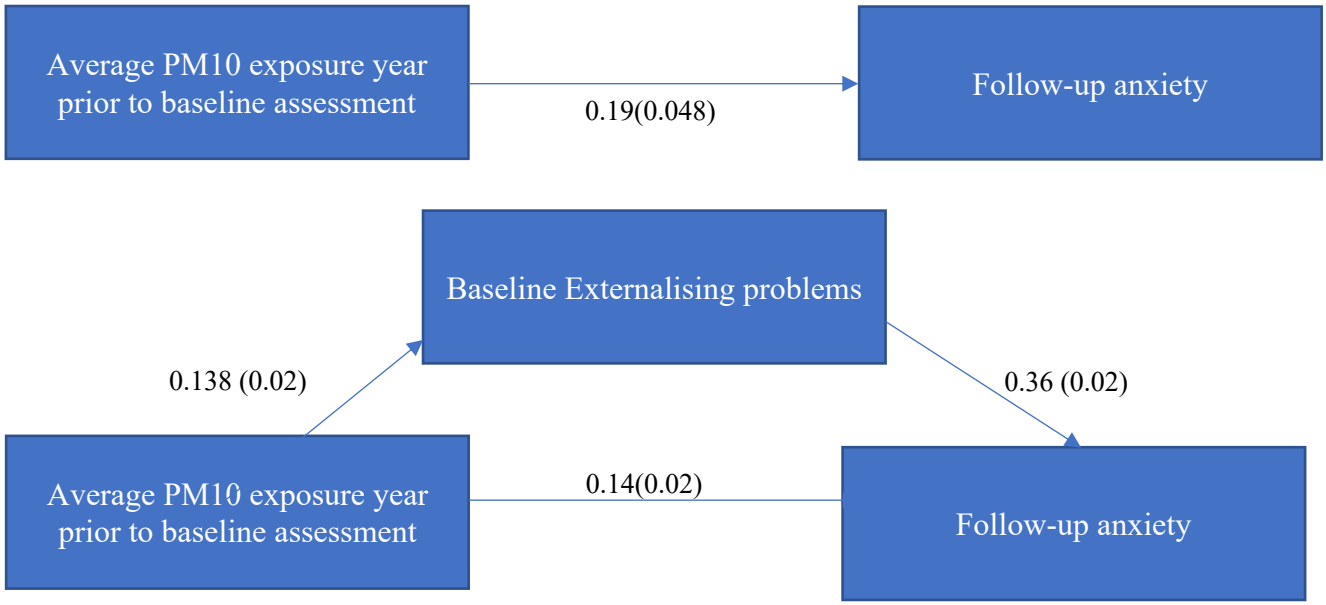


Figure 20. Causal mediation analysis of the association between PM10 exposure and follow-up anxiety, explained by baseline externalising problems, arrows' annotations showing β(p)

Although it was an underpowered sample (N=34) there was a trend effect of earlier anxiety mediating the relationship between PM_{10} and CAPE psychosis risk in those with below average intelligence, by 49% at close to significance ($p = 0.06$). There was also a trend effect where worsened sleep quality mediated the detrimental effect of ozone on follow-up executive function ($p = 0.08$).

Table 21. Mediation analyses of associations between air pollution and adolescent psychological outcomes by sleep quality, atopic disease, mental health, and cognition

Mediator:	Sleep quality score	Asthma/eczema, allergies	BMI	Prior Mental health	Prior Cognition
Association:	Indirect ACME effect estimate (*p* value), % mediated				
PM_{10} and GAD-7	Mediator not associated with exposure	Mediator not associated with exposure or outcome	Mediator not associated with outcome	**BI externalising: 0.05(0.02) 31%**	No detrimental cognitive effects at baseline
Ozone and F1 EF	*0.01(0.08), -1%*	Mediator not associated with outcome	Mediator not associated with exposure or outcome	No detrimental MH effects at baseline	
Subgroups					
PM_{10} and CAPE in those with below average IQ	Mediator not associated with exposure	Mediator not associated with exposure or outcome	Insufficient data	*GAD: 0.10(0.06), 49%*	Plausible mediator (F1 processing speed) not associated with outcome

PM_{10} = particulate matter <10 micrometres in diameter. O_3 = ozone. EF= executive function.. GAD-7: The Generalized Anxiety Disorder 7-item scale (GAD-7). EF: Executive Function. CAPE-P15: The Community Assessment of Psychic Experiences- Positive scale. MH= mental health
Bold: p<0.05. *Italics: p<0.10*
Models adjusted for sex, age, ethnicity, school type, NSSEC, first language, parental education, smoking/ETS.

7.2 Discussion

7.2.1 Summary

This chapter investigated the involvement of other factors as moderators or mediators in the relationships between air pollution or noise and psychological outcomes in teenagers. Interaction terms were tested to identify subgroups which may differ with respect to associations.

Several ‘beneficial’ cognitive associations were observed in average or above average students only, with ‘harmful’ effects of air pollution seen especially in below average IQ students. Girls presented a number of specific cognitive effects, some beneficial and some harmful. White students also presented a number of specific effects, some cognitive, some mental health, some beneficial, and some harmful. Independent school students presented a number of effects not observed in non-independent school students, again some cognitive, some mental health, some beneficial, and some harmful. Although religion was hypothesised as potentially protective for mental health, and it was mostly mental health outcomes it moderated, these students presented two negative effects, whereas non-religion was associated with two ‘beneficial’ effects and one ‘harmful one’ (PM_{10} and anxiety). Despite small power, three cognitive outcomes were detrimentally associated with air pollution or noise in mixed race pupils. Low noise environments strengthened the observed ‘beneficial’ association of NO_2 with executive functioning. Results of mediation analyses were mostly supportive of the null, however the relationship between PM_{10} and anxiety was mediated by its association with earlier externalising symptoms, psychosis risk from PM_{10} was mediated by earlier effects on anxiety, and ozone’s relationship with worsened executive function was mediated by earlier effects on sleep quality.

7.2.2 Interpretation

It was hypothesised that less privileged or advantaged students may be more vulnerable to the effects or air pollution and noise. The fact some ‘beneficial’ effects were only observed in pupils with IQ’s average or above average, and some ‘harmful’ effects only observed in students with below average IQ’s is perhaps unsurprising, as other factors contributing to students’ performance IQ may have conferred a vulnerability to the impact of other stressors (e.g. a more reactive immune system, stress response, or distractibility). Notably, psychosis

symptoms were only associated with PM_{10} in below average intelligence students. This is interesting as prior cognitive deficit is a well-documented feature of schizophrenia (Reichenberg et al., 2010), but it is debated whether it represents an aetiological risk factor (partial cause), or whether it a manifestation of the early stages of the disorder, antecedent to core symptoms but already determined. The finding of air pollution conferring risk of psychosis in only low IQ students suggests that low IQ may be an effect modifier (rather than merely a symptom of premorbid psychotic disorder), as other environmental factors can interact with it to influence eventual outcomes. Additionally, ozone was associated with increased risk of psychosis symptoms in boys. Psychosis is more common in men than women, and there is evidence of differential aetiologies (Falkenburg and Tracy, 2014, Fisher et al., 2009). Given the age of the sample, air pollution's influence during developmental changes specific to boys (pubertal or psychosocial) may have presented a particular risk to boys. The limited sample size and generalisability of this sample means these interesting results are provisional and strongly warrant further research.

For mixed race students, despite a small sample size, associations were observed between NO_2 exposure and slower processing speed development between baseline and follow-up, and between Lden and Lday exposure and reduced improvements in CFT assessed intelligence between baseline and follow-up. These associations were not observed in the overall cohort and did not significantly differ between white students and students of other ethnicities. While mixed race students generally perform better academically than White British, Other White, Pakistani, Black Caribbean or Roma students (Alexander and Shankley, 2020), there is evidence they may engage in more high risk or antisocial adolescent behaviours, which has been theorised to arise from the lack of a clear peer group and low proportions of surrounding people with the same ethnicity (Chisolm et al., 2009, Fryer et al.,

2012). Children and adolescents of mixed heritage are subject to unique social stressors (Ali, 2020), which may confer increased vulnerability to the impacts of air pollution and noise on cognitive development.

However, the fact many effects were only observed in independent schools, positive and negative, is contrary to hypotheses as it was theorised that less socioeconomically privileged pupils may be more vulnerable to the effects of noise and air pollution. The fact that a mix of beneficial and harmful effects were found specifically in independent school pupils suggests perhaps that those at independent schools provided more accurate data, which could easily arise from the resources and facilities schools provided for data collection (e.g. spacious and quiet computer rooms). Girls presenting a number of cognitive effects not observed in boys could also be due to data accuracy if girls were more likely to respond compliantly (Eisenberg et al., 2006, Feingold, 1994). A larger number of effects being observed in white students than other ethnicities, but without a clear direction, could be due to power as they were the largest group. NO_2 was more strongly associated with executive function in a low noise environment. If this was due more cooking in the home or reduced blood pressure, it is plausible that a loud environment might somewhat counteract these benefits. Although only a few mediation effects were observed, all three were mediators of negative or harmful associations as opposed to beneficial ones, and all were psychological mediators (sleep, anxiety, externalising problems). This strengthens the evidence for these harmful associations as they follow a plausible pattern consistent with cause and effect over time including partial mechanisms. However, as 'beneficial' effects were not hypothesised, potential mechanisms for such associations were not posited and explored, so these analyses were biased towards findings evidence of mediators for harmful associations.

7.2.3 Strengths and limitations

The strengths of this analysis are that most prior literature has not investigated the impact of sociodemographic, exposure, health, and psychological factors on the associations between exposure to air pollution and noise and psychological outcomes in adolescence. While these results present a complex picture, they advance the field by identifying potential vulnerabilities and warning signs (male gender, low IQ, mixed race ethnicity, poor mental health, worse sleep quality), for impacts of air pollution and noise on later psychological outcomes. The analyses were thorough, evaluating a wide range of factors in relation to numerous models. The strengths outlined in chapter 6 also apply here.

In terms of shortcomings, the categorisations used for religion (yes/no), school (independent/state), and ethnicity (white, black, asian, mixed race, other) could be considered reductive and thus lack nuance- e.g. between the experiences of different cultures- which could have affected results. Further, sleep, allergies, and BMI were assessed using questionnaire/self-report, and might have been more accurate if assessed using objective measurement. Future research should include biological measures or biomarkers of blood pressure, breathing capacity, BMI, sleep, allergies, oxidative stress, neuronal injury, and inflammation. Additionally, some subgroup analyses were underpowered, for example, evaluating the psychosis symptoms outcome in just below average IQ students (N=34) or just boys (N=41). Finally, these analyses were conducted using the hybrid exposure with source, but as discussed in the previous chapter, data on indoor sources of air pollution and on indoor infiltration of outdoor PM_{10} and ozone introduced some uncertainty to these estimates. The novelty and ecological validity of hybrid approaches has to be weighed up against the relative uncertainty of indoor estimates compared to outdoor.

7.2.4 Concluding comments

The case has been made that inequalities in early life adversity and socioeconomic privilege may impact the body's ability to withstand the impact of other physical stressors has been made by other authors with respect to physical health (Hicken et al., 2012, Kristiansson et al., 2015, Olvera Alvarez et al., 2018), and so it is plausible that this could translate to psychological health for the detrimental effects observed in less privileged groups. However, effects being more pronounced in more privileged groups, or beneficial effects being stronger in some groups than others, is harder to explain as such. It seems more likely that this is due to various biases in the data collection process (e.g., more reliable reporting by independent school students), or confounding by other associated factors (e.g. accessibility, cooking frequency). Mediation analyses yielded little evidence for a role of questionnaire-assessed atopic disease, sleep, BMI- mediating these associations, but future research should explore the role of measured biological and health factors. There was some evidence of other psychological factors moderating or mediating associations, which was consistent with hypotheses. Overall, these results show the complexity of these associations; that they do not have an equivalent effect across the population, and they interact with other psychological factors at play to influence outcomes, perhaps interacting with other circumstances which have conferred greater vulnerability or resilience to their effects. This may help us to make sense of heterogeneities in prior findings as the setting and population is likely to produce different findings, however it also shows that much more research is required to properly understand these associations.

Chapter 8: Discussion

8.1 Summary and Interpretation

This thesis investigated the question of whether there is an association between exposure to air pollution and noise and adolescent psychological outcomes. Reviews of the literature found overall supportive evidence for the associations, though there was still great heterogeneity in findings and the adolescent age group was sorely overlooked despite plausible developmental vulnerability. Cognitive outcomes in young people most strongly associated with higher levels of exposure to air pollution were executive function/attentional skills (especially working memory), general intelligence, and neurodevelopment. Reading and language abilities and executive function/attentional skills were the cognitive outcomes most strongly associated with noise pollution in young people, especially with aircraft noise. Mental health outcomes were most often associated with exposure to $PM_{2.5}$, with the strongest evidence for associations with psychiatric emergencies. The mental health outcome most strongly associated with noise pollution, specifically road traffic noise, was internalising problems. The relative number of studies in adolescents was far smaller than in children (some studies included younger teenagers categorised together with children, but few studied those aged 14+ or focussed on the adolescent period), and the literature provided no high quality evidence for associations between noise exposure and adolescent psychological outcomes, and only provided high quality evidence for two categories pertaining to air pollution ($PM_{2.5}$ and executive function/attentional skills, $PM_{2.5}$ and psychiatric emergency).

A co-produced qualitative study of young people's experiences found that some teenagers do worry about air quality and noise and find it, or indicators of it, disruptive. However, other potentially covarying features are also disruptive or protective (such as greenspace, litter, dirt, traffic, industrial and busyness of population). Young people were passionate about

environmental issues, so action ameliorating air pollution will have a dual benefit directly through reduced exposure and indirectly through benefits for the environment and climate change. Action that reduces noise pollution - largely due to transport and industry - is also likely to have benefits for climate, wildlife, and local environment, which young people indirectly benefit from through their interest in these issues and enjoyment of these spaces.

Although prior studies have occasionally found 'beneficial' effects of exposure to air pollution on mental health and cognition (Zhao et al., 2019, Chen et al., 2020a), this study finding multiple 'beneficial' associations with increased exposure to air pollution was inconsistent with hypotheses and most past literature. However, multi-pollutant models and longitudinal models were less likely to produce such associations - supporting harmful associations between exposure and executive function, processing speed, anxiety, internalising problems, externalising problems, and intelligence. This is more broadly consistent with the theoretical mechanisms posited in Chapter 1 and the literature examined in Chapters 2 and 3. The robust finding of ozone exposure as related to executive function development is particularly novel, as no identified prior literature had investigated this association in adolescence (though one study had investigated working memory and ozone, Gui et al., 2020). This study had the novel contribution of evaluating indoor sources and infiltration of air pollution (at home and school), rather than just residential outdoor air pollution, but this may have been subject to some confounding by house type, fuel type, and cooking frequency as these indoor sources drove most of the 'beneficial' associations. The comparison between different time periods of noise exposure and different sources/settings of pollution exposure suggested that context may moderate exposure effects; it is unclear why results are different for indoors vs outdoors, for day vs night, for different groups (not necessarily the most disadvantaged as was hypothesised), or compared to prior research, or

why some associations suggest positive (beneficial) outcomes with increased exposure. These results may reflect complexities in the phenomena itself (ontological limitations), or the phenomena being subject to large amounts of confounding, making them methodologically hard to approximate (epistemological limitations), or both. There were also benefits to urban spaces identified by young people in interview; access to resources, activities, institutions, other people, and higher levels of stimulation, so this may have driven some of the positive effects (as these things are likely to covary with exposure). This could also make sense of the fact benefits were often felt more by young people with higher IQs and girls, if these groups are utilising or absorbing the benefits of such opportunities or influences more than other adolescents.

There was evidence that students with low baseline IQ, male gender, or mixed-race heritage may have been more vulnerable to the detrimental effects of air pollution and noise. Girls, white students, and independent school students also drove a number of effects but without a clear pattern in terms of benefits and harms. Low noise conditions seemed to enhance the improved executive function found to be associated with NO_2 exposure. The association of PM_{10} with anxiety was mediated by its association with earlier externalising problems, the association of PM_{10} with psychosis risk in low IQ students was mediated by earlier anxiety, and the association of ozone with executive function was mediated by earlier sleep quality. Evidence of mediation is supportive of these 'harmful' associations' potential causality, though mechanisms were not investigated as thoroughly for 'beneficial' associations because they were not hypothesised to exist a priori. Each hypothesis for the project is listed in table 22 with the corresponding findings.

Table 22. Overall hypotheses tested by thesis and the corresponding findings

Hypotheses	Supported by research?	Comment
1. The existing literature will be supportive of associations between higher levels of air pollution and noise exposure and poorer psychological outcomes in adolescence.	Yes	Overall, the evidence tended to be supportive of these associations. However, the number of studies was small, there was much heterogeneity in results and the evidence for specific associations (e.g. $PM_{2.5}$ and depression) was sparse. Much further research is required to establish the nature, causality and strength of these associations.
2. The qualitative element to this mixed methods project will be without hypotheses, allowing themes to emerge from the data inductively.	N/a	N/a
3. Higher levels of exposure to air pollution and noise will be associated with poorer cognitive abilities and worse mental health outcomes.	Partially	Although ozone was associated with slower executive function development during adolescence, and several exposures were associated with detrimental effects on intelligence and processing speed, unanticipated associations were observed between air pollution and better executive function, driven by indoor sources of air pollution. Associations with mental

		health, especially for $PM_{10,}$ were more consistently detrimental.
4. The psychological outcomes of more socially disadvantaged groups - by socioeconomic status, ethnicity, intelligence, and gender - will be especially negatively associated with exposure to air pollution and noise, reflected in interaction effects.	Partially	Mixed race ethnicity seemed to be associated with stronger detrimental associations of intelligence and processing speed and air pollution and noise, and baseline IQ and male gender with detrimental effects of air pollution and psychosis risk. However, state school students and girls were not especially prone to detrimental associations (girls and independent school students drove a number of effects, but some were beneficial and some harmful).
5. Sleep, physical health factors, and mental health/cognition will mediate the relationships between air pollution and/or noise exposure and psychological outcomes in adolescence.	Partially	Sleep and mental health factors showed some evidence of mediating impacts of air pollution on anxiety, executive function, and psychosis risk, however physical health factors (BMI and atopic disease) and prior cognition were not significant mediators.

8.2 Strengths & Limitations

This study focussed on adolescents. Adolescents have been neglected in the literature, yet good psychological development is integral to their adult health and wealth (Batty et al.,

2007, Bukodi et al., 2014, Carneiro et al., 2007, Firkowska-Mankiewicz, 2011, Firth et al., 2019), meaning a good understanding of the factors influencing these outcomes developmentally is integral to our ability to apply good policy and practice. This research used a combination of systematic review, qualitative, and quantitative epidemiological methods, informing each other to form a comprehensive picture of UK adolescent responses to these issues from their subjective experience and from a more objective standpoint. Public involvement (PPI) was utilised substantially for the qualitative work (in conception, methods, analysis, and interpretation) enhancing the validity of the results. There were interestingly high levels of coherence between the responses from interviewees on some topics, such as the global environment, environmental information, and environmental policy, so data saturation was reached after interview 12. However, the local environmental features identified, and how severe an influence they were described as having, did vary substantially between individuals. Together, the studies show the importance of context and how varied the psychological impacts of air pollution and noise can be. The epidemiology of mental health and cognition is never simple, as these are multifactorial outcomes, but the nature of environmental factors, which themselves interact and carry conceptual salience, may mean these associations are especially evasive.

However, there are also shortcomings to this research. Although methods were generally robust, some systematic reviews (for mental health outcomes) were not searched, screened, selected, and reviewed in duplicate, so there is the possibility of missed or misconstrued evidence from human error. The sample size and demographics for the qualitative study mean that there are limits to the generalisability of the described experiences. And, despite a large and diverse overall sample for the quantitative analyses, the samples with eligible data for some models (mediation analyses, psychosis symptoms, some subgroups) were less

substantial, and the samples providing certain data (notably cooking frequency and fuel type) were not representative and could have biased results. PPI was thoroughly incorporated into the qualitative study, but the literature review and epidemiological analyses were conducted without much youth insight/involvement; while there is less of a precedent for involving the public in some methodologies than others, it may have benefitted these studies to identify creative ways to do this.

Although in environmental epidemiology we approximate 'noise' and 'air pollution' (and greenspace, neighbourhood deprivation, etc.,) using discrete metrics, this was not the adolescents' experience of their environment as described in interviews. Their worldview of environmental problems and environmental quality was far more fluid and holistic. That is, their general feelings about their environments were influenced by many factors, often discussed interchangeably or in tandem, and were influenced by their judgements, experiences, and the interactions of the two. Often in urban spaces there was a sense of being bombarded or overwhelmed; poor air quality and loud noise is a part of this picture, but the level of overwhelm, and how much air and noise influence it, seems likely to depend on other internal and external factors. That is, each individual is experiencing their environment holistically and uniquely, arising as an emergent experience from numerous factors, features and causes. For example, there were varying levels of concern about air quality, which again is likely to depend on other factors like wider mental health, sociodemographics, personal values, appraisals of their air quality, and other concerns (Chen et al., 2020b, Chiarini et al., 2020, Deguen et al., 2017, Dons et al., 2018, Guo et al., 2016). Real life environments, internal and external, have high levels of complexity. This may be reflected in the apparent contradictions and uncertainty in epidemiological results that attempt to cleanly isolate how much a factor is affecting human psychology statistically. When we attempt to estimate

exactly when, where, for whom, and exactly how, such factors impact people psychologically, the complexity of the human mind and behaviour may mean this is to some degree unpredictable (even if such associations 'really exist' as an absolute feature of the world where all else being equal X determines Y, which is debatable). This is because each person is a complex organism, subject to different interacting causes on the genetic, biological, psychological, sociological levels, as well as actively participating in and moderating their own experience, and therefore each person may be affected differently. What this research enables us to do is to look for general trends or patterns across the literature and make general recommendations on environmental quality, but it may not be possible to detect a precise underlying association because these associations likely reflect many causes and many unique cases summarised together.

Although the qualitative study found evidence of general youth concern with air quality, noise, local environment, and global environment, which could plausibly impact their mental health, it is also possible that the young people who are worried about air quality and noise differ socio-demographically from those who are less worried, which may not covary exactly with the levels of exposure. For example, a recent survey of UK children and adolescents reported more pro-environmental sentiment in white and higher income respondents (NaturalEngland, 2021). Therefore there is a methodological issue of disentangling impacts of exposure irrespective of awareness, from impacts of knowledge of exposure, which this project was not able to directly address, but which other studies have indicated may have a role in the exposure-response relationship (Bergstra et al., 2018, Claeson et al., 2013). Further, some pollutants may be associated with sources which are more tangible in terms of the perceivable air quality (Cantuaria et al., 2017) so the role of awareness or perception could vary by pollutant.

Finally, an important limitation to note is that the literature as it stood precluded clear and specific exposure-outcome hypotheses, beyond 'detrimental impacts of air pollution/ noise on cognition/mental health'. This meant that a broad-brush approach was necessary to be confident that associations would not be missed, however this is raised the issue of multiple comparisons and a high likelihood that some observations were spurious. Specific exposure-outcome hypotheses arising from this thesis, which should be investigated as a priority by future researchers, are outlined in the following section.

8.3 Implications for Future Research

Whilst advancing understanding within the field in several ways, this project has raised many questions and directions for future research. The differences between day, evening, and night noise exposure, and indoor, outdoor and hybrid pollution exposure warrant further investigation. It is unclear if this is due to the exposures impacting people differently in different contexts or different sources of confounding, but it shows that the usual non-specific approach of using annual residential average pollution or noise from traffic is not a specific enough indicator for evaluating how exposure will impact outcomes. Future studies should delineate exposure by home/school, indoor/outdoor sources, and different time periods. There should also be further investigation into what effects are immediate/contemporaneous, which are longer term, and the relationship between the different exposure latencies- e.g., concurrent performance (potentially experimentally, see Rom et al., 2013), 24 hours, 7 days, monthly, annually.

To help establish if a causal link is present between these exposures and outcomes, more biological responses should be measured and investigated as mediators of epidemiological associations; for example, biomarkers of inflammation, oxidative stress, neuronal injury,

blood pressure, breathing capacity, hearing ability, BMI, and epigenetic markers. Other populations of adolescents should be investigated in different countries and contexts - especially those in highly urbanised areas exposed to extreme pollution and noise; there was some evidence of threshold and nonlinear effects so it cannot be assumed that we can extrapolate from the observed levels here to higher levels of average exposure. Prenatal exposure and psychological outcomes have also been understudied, especially with respect to outcomes later than early childhood. Longitudinal studies should look at the longer-term impact of exposure. Where there is a lack of appropriate prospective data, prenatal exposure can be estimated retrospectively in relation to adolescent outcomes, and adolescent exposure can be estimated retrospectively in relation to adulthood outcomes.

Where possible, studies should measure environmental awareness and concern about air quality and noise on a local level; it is worth establishing how well adolescents can predict their relative level of exposure (there is a general population association between perception and exposure, see (Cori et al., 2020, Deguen et al., 2017)), and if so whether their concern has a role in how they are impacted by these exposures and other environmental features.

Finally, to avoid the problem of non-specific hypotheses and multiple comparisons encountered in this project, some specific hypotheses have been identified for future researchers to focus upon:

1. Ozone exposure exerts a detrimental effect upon executive function development during adolescence.
2. PM_{10} exposure is associated with detrimental effects upon adolescent mental health, specifically (from early to late adolescence) externalising problems, anxiety, and psychosis.

3. Particulate matter exerts a detrimental effect upon processing speed during adolescence.
4. Air pollution increases the risk of psychosis in specific groups (male and low IQ adolescents may be of particular interest).
5. Indoor air pollution may improve executive function.
6. Daytime noise at school can negatively impact intelligence and externalising behaviours.

8.4 Policy and public health implications

This research suggests noise and air pollution exposure in adolescence can have detrimental psychological effects, the long-term impacts of which are unknown. It is important to remember pollution has been linked to long term serious outcomes across the life course, including increased risk of dementia and Alzheimer's disease (Peters et al., 2019). Young people's health and the planet's health are the foundation of their lives and futures, and their internal and external worlds are being damaged through contemporary practices in transport, farming, and manufacturing. Yet, young people have no real power or influence with respect to how the environment is managed. Therefore, as well as the relevance of policy to the health implications of environmental exposures, there is a perceived injustice also relevant to policy on ethical grounds.

Despite the causality between environmental factors and young people's psychology being complex, it remains true that exposure to air and noise pollution affects the body and other aspects of health, it (and/or its main sources) are harmful to the environment, and that young people are passionate about the environment. Therefore, policy applied to reduce young people's exposure to pollution and noise has the opportunity to be beneficial on several levels. This complexity, or web of causation, can be taken advantage of in terms of co-

benefits; because the connectivity between air pollution / noise and psychological outcomes in youth mean that higher level intervention can trickle through to benefit youth at different levels of causation. For example, it implies that reductions of traffic in urban spaces will have benefits operating on several different levels; directly through reduced exposure to these stressors (air pollution and noise); and indirectly through impacts on local environmental quality (aesthetics, cleanliness, busyness, greenery and wildlife, creating calming rather than overwhelming environments, beneficial on the conscious level and unconscious level), approval or appreciation of the policy itself, and reductions in climate change. Although noise does not cause climate change (as air pollution does) there is a high degree of overlap between major causes of noise pollution and air pollution, so actions to reduce both are likely to benefit young people's mental health through these various pathways. For example, policies limiting flying will reduce noise pollution around airports, as well as improve air quality, as well as reduce climate change, which then has indirect benefits for young people because they are concerned and engaged when it comes to global environmental issues, and direct benefits for mental health through decreased temperatures and traumatic climate disasters (Lawrance et al., 2021). Benefits for cognition could occur through reduced exposure, with indirect effects via improved health and wellbeing. For example, noise exposure was noted in interviews as a cause of school/ work distraction, which was also described as causing stress (which can impact cognition). The relative importance of daytime noise, i.e., exposure during working hours, may reflect this. This research suggests that ozone may be particularly harmful for cognitive development so policies oriented towards reducing ozone may be beneficial on the cognitive side, though reduction of ozone precursor gases will also reduce the presence of other pollutants (Lu et al., 2019). Effects of daytime noise suggest that noise reduction policies near schools may be especially beneficial.

Further, if young people are brought onboard to guide initiatives, e.g. that incentivise greener behaviour or reimagine urban spaces, then this may benefit their wellbeing in terms of empowerment and constructive hope (Ojala, 2012a), as well as make policy which is more likely to benefit them because it is informed by their lived experience of urban spaces. As recommended by the British Youth Council (BYC) Steering Group in their review of environment and climate policy[6], a network of youth advisors and ambassadors (termed climate champions/ green ambassadors by the BYC) should be developed to inform policy on different levels and regions and develop a youth centred approach to engaging communities. Some good examples of recent practice in this regard are youth representation in the steering group for the Department for Educations' sustainability and climate change policy[7], and recent youth voice projects launched by the Department for Digital, Culture, Media and Sport.[8] Unfortunately, these are not the most instrumental departments for environmental protection and human environmental exposures and the Department for Environment, Food and Rural Affairs (Defra), the Department for Transport, the Department for Levelling Up, Housing and Communities, and the Department for Business, Energy and Industrial Strategy (BEIS) should set up equivalent programs.

Young people are disempowered politically; they don't hold influential positions in society or the workplace, and often they don't have a great deal of choice lifestyle wise in terms of transport, consumption, school, or work practices. As well as inviting in youth voices and encouraging personal action, the BYC also emphasised the importance of top-down change

[6] http://www.youthworkunit.com/wp-content/uploads/2020/07/Youth-Steering-Group-Climate-Change-Review-2020.pdf

[7] https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1031454/SCC_DRAFT_Strategy.pdf

[8] https://www.gov.uk/government/news/dcms-launches-new-youth-voice-projects

and collective responsibility, noting the disempowering and demotivating nature of seeing large organisations and people in power not behaving in a pro-environmental way. This is echoed in the qualitative study (Chapter 4) where young people expressed the sentiment that they were not the ones primarily responsible for environmental problems but felt they had been handed the responsibility to solve them. Therefore, those in power, particularly stakeholders in health and environment, should be leading by example through their institutional and personal practices. Further, adults with relative influence should engage with youth voices and educate themselves on importance of the environmental quality for youth health and wellbeing (e.g., parents, teachers, educators, youth leaders, social workers, clinicians, mental health advocates). This understanding should then be applied through voting, petitioning, lobbying, advocating, raising awareness, and making more conscious choices on personal and professional levels. There are sociodemographic inequalities and limitations in how much influence people have (e.g., control over where they and their families live, and the consumer choices they make), so messaging should empower and encourage the general population whilst not shaming them or holding them solely responsible for environmental problems like air and noise pollution, or their/their family's exposure to them.

Something that was also identified by the young people in this study was the importance of communication around environmental issues, and how environmental information is often scary and alienating. It is important to disseminate knowledge without fear. The BYC also said that clear and concise messaging from government, communicating policy and campaigns, was important to engage and empower young people.

Finally, it is important to remember that creating perfectly unpolluted environments will not eliminate other hindrances to mental health and cognitive development in adolescence. In interview, the young people identified other priorities which they felt were important to address. First of all, while policies taken to reduce air pollution and noise are likely to reduce climate change, if policies are enacted that simply reduce adolescent exposure to sources of noise and air pollution (e.g. placement of schools and airports, factories) but without reducing the industrial and transport activity that is detrimental to climate, this will still effect adolescent mental health indirectly through their climate change anxieties and the direct risks presented to mental health and psychological development by climate change.

The most commonly and emphatically identified area for young people's mental health was stress around school, exams, educational attainment, university, and career. Additionally, social support, health and fitness, mental health awareness/stigma, mental healthcare (particularly access) and social media were identified as important. Interventions that target both environmental exposure or environmental quality and promote wellbeing in these areas could therefore be manifoldly beneficial. For example, nature-based interventions for mental health, community-oriented campaigns geared towards environmental change, active transport, school-based activities engaging with nature, and development of social media platforms oriented to youth campaigning, connection, and education around environmental issues.

Finally, some actions unconnected to air pollution and noise must be addressed directly for the promotion of good adolescent psychological development; most notably the provision of good, accessible, clinical mental healthcare for adolescents from all sociodemographic backgrounds and in all NHS trusts, and investigation into the sources of pressure for young

people that mean they feel so overcome by schoolwork, exams, and their professional futures. Whilst encouraging or rewarding hard work may be a good thing, detrimental impacts on mental health are neither fair nor productive. This could be partially attributable to the extensive education system reforms which took place in 2014[9], an increasingly competitive job market[10](Berry and McDaniel, 2022), or social factors pertaining to how work and achievement is represented by teachers, parents, and peers (Sun et al., 2013). Schools should also provide training and support to young people and parents about navigating social media; whilst it is not likely to go away anytime soon, there should be curriculum-based guidance on using it skilfully, including the differential impacts of different content (e.g. following pages that promote body comparison or self-harm, in comparison to pages promoting mental health, issues of interest, or connecting with peers), the importance of life balance with other priorities, and how to look out for signs of excessive or harmful use (Best et al., 2014, de Calheiros Velozo and Stauder, 2018, Escobar-Viera et al., 2018, Vidal et al., 2020).

8.5 Concluding Comments

This thesis investigated the question of whether exposure to air pollution and noise has psychological impacts in adolescence. Systematic reviews of the prior literature were broadly supportive but showed a real need for further research, as the existing studies were not conclusive. Qualitative research with this age group showed that adolescents experience air pollution and noise is impacting them on several levels, directly through exposure and indirectly through their environmental concern. The epidemiological analyses found detrimental mental health impacts of air pollution and noise, and some detrimental effects of

[9] https://www.gov.uk/government/speeches/education-reform-new-national-curriculum-for-schools

[10] https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/404997/15.02.18._Youth_report_V17.pdf

air pollution and noise on cognition and cognitive development, though alongside some unpredicted beneficial associations. More research is required to untangle the nuances of these complex relationships in a variety of settings. This age group showed high levels of environmental engagement in general and were particularly worried about climate change. Policy responses should thus address the emissions themselves from transport and industrial activity, rather than just focussing on mitigating adolescent exposure through changes to the locations of sources and/or locations or features of adolescent occupied spaces. Young people should be involved in environmental decision making, as the quality of the physical environment impacts them now and in future.

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Appendices

Contents

	Page
I: Air pollution review search terms	262
II: OHAT risk of bias assessment domains used in risk of bias assessment for individual studies	264
III: Associations only assessed by one study in Air Pollution literature	270
IV: Noise review search terms	274
V: Associations assessed by one study in Noise literature	276
VI: Consolidated criteria for reporting qualitative research checklist, applied to research in Chapter 4	277
VII: Interview protocol: environmental thoughts and feelings in adolescents	280
VIII: All codes identified in qualitative analysis	281
IX: Noise effects for participants within the M25 (pollution analysis sample)	288
X: Associations between exposure to air pollution and/or noise and psychological outcomes in the SCAMP cohort: results for all two-exposure models	291
XI: Associations between exposure to evening noise (Leve) and night-time noise (Lnight) and psychological outcomes in the SCAMP cohort	314
XII: Associations between exposure to air pollution or noise and psychological outcomes in the SCAMP cohort: Interaction effects	316
XIII: Results for associations between exposure to air pollution or noise and psychological outcomes in the SCAMP cohort, adjusted for indices of multiple deprivation (IMD-19)	391
XIV: Results for associations between exposure to air pollution or noise and psychological outcomes, for participants with a precise (certain) geocode only	399
XV: Exposure to outdoor air pollution and psychological outcomes in the SCAMP cohort	405
XVI: Exposure to indoor air pollution (with indoor sources) and psychological outcomes in the SCAMP cohort	409
XVII: Exposure to indoor air pollution (without indoor sources) and psychological outcomes in the SCAMP cohort	414
XVIII: Exposure to hybrid air pollution (without indoor sources) and psychological outcomes in the SCAMP cohort	418
XIX: Non-linear associations between exposure to air pollution or noise and psychological outcomes in the SCAMP cohort (generalised additive model plots, presented when the non-linear association was a significantly better fit than the linear model)	422

I: Air pollution review search terms

A) *Cognition*

1) Medline/Embase/PsycInfo (all via OVID, searched separately)

1 exp Air Pollution/
2 (Air adj2 (pollut* or quality or ambient)).ti,ab.
3 (atmospher* adj2 pollut*).ti,ab.
4 (Particulate matter or ambient particulate or PM or PM1* or PM2* or PM10* or ultrafine particulate* or ultrafine particle* or UFP).ti,ab.
5 (Coarse particle* or Soot or Black smoke or Black carbon or Elemental carbon or wood smoke).ti,ab.
6 (Nitrogen dioxide or nitrogen oxide or NOx or NO2 or nitric oxide).ti,ab.
7 ozone/
8 Cognition/ or attention/ or executive function/ or memory/
9 cognit*.ti,ab.
10 ((react* adj2 (time or speed)) or (process* adj2 (time or speed))).ti,ab.
11 (intell* or IQ or intelligence quotient or reasoning).ti,ab.
12 8 or 9 or 10 or 11
13 1 or 2 or 3 or 4 or 5 or 6 or 7
14 12 and 13

2) SCOPUS

((TITLE-ABS-KEY(air w/2 ambient OR "air pollut*" OR "air quality") OR("particulate matter" OR "ambient particulate" OR "ultrafine particule*" OR "ultrafine particle*" OR UFP OR "coarse particle") OR ("black smoke" OR "black carbon" OR "elemental carbon") OR (“nitrogen dioxide” or “nitrogen oxide” or NOx or NO2 or “nitric oxide”) OR (ozone OR O3))) AND ((TITLE-ABS-KEY(cognition OR attention OR executive function OR memory OR cognit* OR “reaction time” or “processing speed” or intell* OR IQ OR reasoning)))

B) *Mental health*

1. Webof Science (not exact search)

((ALL=(air pollution) OR ALL=(particulate matter) OR ALL=(carbon monoxide) OR ALL=(ozone) OR ALL=(sulphur dioxide) OR ALL=(nitrogen dioxide) OR ALL=(nitrogen oxide) OR ALL=(black carbon) OR ALL=(PAH) OR ALL=(urban pollution) OR ALL=(traffic) OR ALL=(TRAP) OR ALL=(NO2) OR ALL=(PM) OR ALL=(NOx) OR ALL=(air quality) OR ALL=(UFP)) AND (ALL=(mental health) OR ALL=(psychiatric) OR ALL =(Neurodevelopment)) AND (ALL=(children) OR ALL=(paediatric) OR ALL=(adolescent)))

2. PubMed (terms above exploded)

(("air pollution"[MeSH Terms] OR ("air"[All Fields] AND "pollution"[All Fields]) OR "air pollution"[All Fields] OR ("particulate matter"[MeSH Terms] OR ("particulate"[All Fields] AND "matter"[All Fields]) OR "particulate matter"[All Fields]) OR ("carbon monoxide"[MeSH Terms] OR ("carbon"[All Fields] AND "monoxide"[All Fields]) OR "carbon monoxide"[All Fields]) OR ("ozonated"[All Fields] OR "ozonating"[All Fields] OR

"ozonation"[All Fields] OR "ozonations"[All Fields] OR "ozone"[MeSH Terms] OR "ozone"[All Fields] OR "ozone s"[All Fields] OR "ozonization"[All Fields] OR "ozonized"[All Fields] OR "ozonizer"[All Fields]) OR ("sulphur dioxide"[All Fields] OR "sulfur dioxide"[MeSH Terms] OR ("sulfur"[All Fields] AND "dioxide"[All Fields]) OR "sulfur dioxide"[All Fields]) OR ("nitrogen dioxide"[MeSH Terms] OR ("nitrogen"[All Fields] AND "dioxide"[All Fields]) OR "nitrogen dioxide"[All Fields]) OR ("nitrogen oxides"[MeSH Terms] OR ("nitrogen"[All Fields] AND "oxides"[All Fields]) OR "nitrogen oxides"[All Fields] OR ("nitrogen"[All Fields] AND "oxide"[All Fields]) OR "nitrogen oxide"[All Fields]) OR ("soot"[MeSH Terms] OR "soot"[All Fields] OR ("black"[All Fields] AND "carbon"[All Fields]) OR "black carbon"[All Fields]) OR "PAH"[All Fields] OR (("urban"[All Fields] OR "urbanicity"[All Fields] OR "urbanism"[All Fields] OR "urbanity"[All Fields] OR "urbanization"[MeSH Terms] OR "urbanization"[All Fields] OR "urbanizations"[All Fields] OR "urbanize"[All Fields] OR "urbanized"[All Fields] OR "urbanizes"[All Fields] OR "urbanizing"[All Fields] OR "urbans"[All Fields]) AND ("environmental pollutants"[Pharmacological Action] OR "environmental pollutants"[MeSH Terms] OR ("environmental"[All Fields] AND "pollutants"[All Fields]) OR "environmental pollutants"[All Fields] OR "pollutant"[All Fields] OR "pollutants"[All Fields] OR "pollutant s"[All Fields] OR "pollute"[All Fields] OR "polluted"[All Fields] OR "polluter"[All Fields] OR "polluters"[All Fields] OR "pollutes"[All Fields] OR "polluting"[All Fields] OR "pollution"[All Fields] OR "pollution s"[All Fields] OR "pollutional"[All Fields] OR "pollutions"[All Fields])) OR ("traffic"[All Fields] OR "traffic s"[All Fields] OR "traffics"[All Fields]) OR "TRAP"[All Fields] OR "NO2"[All Fields] OR ("precis med"[Journal] OR "phys med"[Journal] OR "pharmacogn mag"[Journal] OR "pediatr med"[Journal] OR "pm"[All Fields]) OR "NOx"[All Fields] OR ("air pollution"[MeSH Terms] OR ("air"[All Fields] AND "pollution"[All Fields]) OR "air pollution"[All Fields] OR ("air"[All Fields] AND "quality"[All Fields]) OR "air quality"[All Fields]) OR "UFP"[All Fields]) AND ("mental health"[MeSH Terms] OR ("mental"[All Fields] AND "health"[All Fields]) OR "mental health"[All Fields] OR ("psychiatrical"[All Fields] OR "psychiatrically"[All Fields] OR "psychiatrics"[All Fields] OR "psychiatry"[MeSH Terms] OR "psychiatry"[All Fields] OR "psychiatric"[All Fields]) OR "Neurodevelopment"[All Fields]) AND ("child"[MeSH Terms] OR "child"[All Fields] OR "children"[All Fields] OR "child s"[All Fields] OR "children s"[All Fields] OR "childrens"[All Fields] OR "childs"[All Fields] OR ("paediatrics"[All Fields] OR "pediatrics"[MeSH Terms] OR "pediatrics"[All Fields] OR "paediatric"[All Fields] OR "pediatric"[All Fields]) OR ("adolescences"[All Fields] OR "adolescency"[All Fields] OR "adolescent"[MeSH Terms] OR "adolescent"[All Fields] OR "adolescence"[All Fields] OR "adolescents"[All Fields] OR "adolescent s"[All Fields])))

3. Embase/ PsycInfo (both Via Ovid, searched separately)

(((("air pollution" or "particulate matter" or "carbon monoxide" or ozone or "sulphur dioxide" or "nitrogen dioxide" or "nitrogen oxide" or "black carbon" or PAH or "urban pollution" or traffic or TRAP or NO2 or PM or NOx or "air quality" or UFP) and ("mental health" or psychiatric or Neurodevelopment) and (children or paediatric or adolescent)).ab.) or ((("air pollution" or "particulate matter" or "carbon monoxide" or ozone or "sulphur dioxide" or "nitrogen dioxide" or "nitrogen oxide" or "black carbon" or PAH or "urban pollution" or traffic or TRAP or NO2 or PM or NOx or "air quality" or UFP) and ("mental health" or psychiatric or Neurodevelopment) and (children or paediatric or adolescent)).ti.)

II: OHAT risk of bias assessment domains used in risk of bias assessment for individual studies in systematic reviews

Risk of Bias	**Selection bias**	**Confounding bias**	**Attrition/Exclusion bias**	**Exposure characterisation**	**Outcome characterisation**	**Selective reporting bias**
Question (OHAT Risk of Bias Rating Tool 2015)	3. Did selection of study participants result in appropriate comparison groups?	4. Did the study design or analysis account for important confounding and modifying variables?	7. Were outcome data complete without attrition or exclusion from analysis?	8. Can we be confident in the exposure characterization?	9. Can we be confident in the outcome assessment?	10. Were all measured outcomes reported?
DH: Definitely High	There is direct evidence that subjects (both exposed and non-exposed) were not similar, recruited within very different time frames, or had the very different participation/response rates. *OR that participants were a non-representative sample (a specific or unusual group who are likely to differ from the general population of that age and place).*	There is direct evidence that the distribution of primary covariates and known confounders differed between the groups *or across exposure levels*, confounding was demonstrated, and was not appropriately adjusted for in the final analyses, OR there is direct evidence that primary covariates and confounders were assessed using non valid measurements, *or were not measured*	There is direct evidence that loss of subjects (i.e., incomplete outcome data) was unacceptably large and not adequately addressed*. Unacceptable handling of subject attrition includes: reason for missing outcome data likely to be related to true outcome, with either imbalance in numbers or reasons for missing data across study groups; or potentially inappropriate	There is direct evidence that the exposure was assessed using methods with poor validity, OR evidence of exposure misclassification (e.g., differential recall of self-reported exposure).	There is direct evidence that the outcome assessment method is an insensitive instrument, OR the length of follow up differed by study group, OR there is direct evidence for lack of adequate blinding of outcome assessors (including study subjects if outcomes were self-reported), including no blinding or incomplete blinding.	There is direct evidence that all of the study's measured outcomes (primary and secondary) outlined in the protocol, methods, abstract, and/or introduction (that are relevant for the evaluation) have not been reported. In addition to not reporting outcomes, this would include reporting outcomes based on composite score without individual outcome components or outcomes reported using measurements,

		OR there is direct evidence that there was an unbalanced provision of additional co-exposures across the primary study groups, which were not appropriately adjusted for.	application of imputation. **Unacceptably large if addressed: <60%* *Unacceptably large if unaddressed: <80%*			analysis methods or subsets of the data (e.g., subscales) that were not pre-specified or reporting outcomes not pre-specified, or that unplanned analyses were included that would appreciably bias results.
PH: Probably High	There is indirect evidence that subjects (both exposed and non-exposed) were not similar, recruited within very different time frames, or had the very different participation/response rates, OR there is insufficient information provided about the comparison group including a different rate of non-response without an explanation *OR that participants were a non-representative sample (a specific or unusual group who are likely to differ from the general population of that age and place).*	There is indirect evidence that the distribution of primary covariates and known confounders differed between the groups and was not appropriately adjusted for in the final analyses, OR there is insufficient information provided about the distribution of known confounders (record), OR there is indirect evidence that primary covariates and confounders were assessed using measurements of unknown validity, OR there is insufficient information provided about the measurement techniques used to assess primary covariates and confounders,	There is indirect evidence that loss of subjects (i.e., incomplete outcome data) was unacceptably large* and not adequately addressed, OR there is insufficient information provided about numbers of subjects lost to follow-up **Unacceptably large if addressed: <60%* *Unacceptably large if unaddressed: <80%*	There is indirect evidence that the exposure was assessed using poorly validated methods that directly measure exposure, OR there is direct evidence that the exposure was assessed using indirect measures that have not been validated or empirically shown to be consistent with methods that directly measure exposure (e.g., a job-exposure matrix or self-report without validation) (record "NR" as basis for answer), OR there is insufficient information provided about the exposure assessment, including validity and reliability, but no evidence for concern about the method used .	There is indirect evidence that the outcome assessment method is an insensitive instrument (e.g., a questionnaire used to assess outcomes with no information on validation), OR the length of follow up differed by study group, OR there is indirect evidence that it was possible for outcome assessors (including study subjects if outcomes were self-reported) to infer the study group prior to reporting outcomes, OR there is insufficient information provided about blinding of outcome assessors.	here is indirect evidence that all of the study's measured outcomes (primary and secondary) outlined in the protocol, methods, abstract, and/or introduction (that are relevant for the evaluation) have been reported, OR and there is indirect evidence that unplanned analyses were included that may appreciably bias results, OR there is insufficient information provided about selective outcome reporting.

		OR there is indirect evidence that there was an unbalanced provision of additional co-exposures across the primary study groups, which were not appropriately adjusted for, OR there is insufficient information provided about co-exposures in occupational studies or studies of contaminated sites where high exposures to other chemical exposures would have been reasonably anticipated.				
PL: Probably Low	There is indirect evidence that subjects (both exposed and non-exposed) were similar (e.g., recruited from the same eligible population, recruited with the same method of ascertainment using the same inclusion and exclusion criteria, and were of similar age and health status), recruited within the same time frame, and had the similar participation/response rates,	There is indirect evidence that appropriate adjustments were made, OR it is deemed that not considering or only considering a partial list of covariates or confounders in the final analyses would not appreciably bias results. AND there is evidence (direct or indirect) that primary covariates and confounders were assessed using valid and reliable measurements, OR it is deemed that the measures used would not appreciably bias	There is indirect evidence that loss of subjects (i.e., incomplete outcome data) was adequately addressed and reasons were documented when human subjects were removed from a study, OR it is deemed that the proportion lost to follow-up would not appreciably bias results*. This would include reports of no statistical differences in characteristics of subjects lost to follow up or with unavailable records from those of	There is indirect evidence that the exposure was consistently assessed using well-established methods that directly measure exposure, OR exposure was assessed using indirect measures (e.g., questionnaire or occupational exposure assessment by a certified industrial hygienist) that have been validated or empirically shown to be consistent with methods that directly measure exposure (i.e., inter-	There is indirect evidence that the outcome was assessed using acceptable methods (i.e., deemed valid and reliable but not the gold standard) (e.g., validity and reliability ≥0.40 Genaidy et al. 2007), OHAT Risk of Bias Tool (January 2015) 27 AND subjects had been followed for the same length of time in all study groups [Acceptable, but not ideal assessment methods will depend on the outcome, but	There is indirect evidence that all of the study's measured outcomes (primary and secondary) outlined in the protocol, methods, abstract, and/or introduction (that are relevant for the evaluation) have been reported, OR analyses that had not been planned in advance (i.e., retrospective unplanned subgroup analyses) are clearly indicated as such and it is deemed that the unplanned analyses were

	OR differences between groups would not appreciably bias results.	results (i.e., the authors justified the validity of the measures from previously published research), AND there is evidence (direct or indirect) that other co-exposures anticipated to bias results were not present or were appropriately adjusted for, OR it is deemed that co-exposures present would not appreciably bias results.	the study participants. Generally, the higher the ratio of participants with missing data to participants with events, the greater potential there is for bias. For studies with a long duration of follow-up, some withdrawals for such reasons are inevitable. **Proportion of >80% retained considered unlikely to appreciably bias results*	methods validation: one method vs. another). *This includes validated exposure models for noise and air pollution.*	examples of such methods may include proxy reporting of outcomes and mining of data collected for other purposes], OR it is deemed that the outcome assessment methods used would not appreciably bias results, AND there is indirect evidence that the outcome assessors (including study subjects, if outcomes were self-reported) were adequately blinded to the study group, and it is unlikely that they could have broken the blinding prior to reporting outcomes, OR it is deemed that lack of adequate blinding of outcome assessors would not appreciably bias results, which is more likely to apply to objective outcome measures	appropriate and selective reporting would not appreciably bias results (e.g., appropriate analyses of an unexpected effect). This would include outcomes reported with insufficient detail such as only reporting that results were statistically significant (or not).
DL: Definitely Low	There is direct evidence that subjects (both exposed and non-exposed) were similar (e.g., recruited from the same eligible population, recruited with the same method of ascertainment using	There is direct evidence that appropriate adjustments or explicit considerations were made for primary covariates and confounders in the final analyses through the use of statistical models	There is direct evidence that loss of subjects (i.e., incomplete outcome data) was adequately addressed and reasons were documented when human subjects were removed from a study.	There is direct evidence that exposure was consistently assessed (i.e., under the same method and time-frame) using well-established methods that directly measure exposure (e.g., measurement of the	There is direct evidence that the outcome was assessed using well-established methods (e.g., the "gold standard" with validity and reliability >0.70 Genaidy et al. 2007),	There is direct evidence that all of the study's measured outcomes (primary and secondary) outlined in the protocol, methods, abstract, and/or introduction (that are relevant for the

	the same inclusion and exclusion criteria, and were of similar age and health status), recruited within the same time frame, and had the similar participation/response rates	to reduce research-specific bias including standardization, matching, adjustment in multivariate model, stratification, propensity scoring, or other methods that were appropriately justified. Acceptable consideration of appropriate adjustment factors includes cases when the factor is not included in the final adjustment model because the author conducted analyses that indicated it did not need to be included, AND there is direct evidence that primary covariates and confounders were assessed using valid and reliable measurements, AND there is direct evidence that other exposures anticipated to bias results were not present or were appropriately measured and adjusted for. In occupational studies or studies of contaminated sites, other chemical exposures known to be associated with those settings were	Acceptable handling of subject attrition includes: very little missing outcome data8; reasons for missing subjects unlikely to be related to outcome (for survival data, censoring unlikely to be introducing bias); missing outcome data balanced in numbers across study groups, with similar reasons for missing data across groups, OR missing data have been imputed using appropriate methods and characteristics of subjects lost to follow up or with unavailable records are described in identical way and are not significantly different from those of the study participants. **Very little missing outcome data defined as >90%.*	chemical in air or measurement of the chemical in blood, plasma, urine, etc.), OR exposure was assessed using less-established methods that directly measure exposure and are validated against well-established methods	AND subjects had been followed for the same length of time in all study groups. Acceptable assessment methods will depend on the outcome, but examples of such methods may include: objectively measured with diagnostic methods, measured by trained interviewers, obtained from registries (Shamliyan et al. 2010), AND there is direct evidence that the outcome assessors (including study subjects, if outcomes were self-reported) were adequately blinded to the study group, and it is unlikely that they could have broken the blinding prior to reporting outcomes.	evaluation) have been reported. This would include outcomes reported with sufficient detail to be included in meta-analysis or fully tabulated during data extraction and analyses had been planned in advance.

		appropriately considered.				

III: Associations only assessed by one study in Air pollution literature

Exposure	Outcome	Study	Supportive of harmful association?
NO2	General intelligence/IQ	Julvez et al., 2021	No
BC	General intelligence/IQ	Suglia et al., 2008	Ish (trend)
PAH	General intelligence/IQ	Suter et al., 2018	Ish (HIV only)
Composite	maths	Zhang, 2018	Ish (at some time lags)
Composite	reading/language	Zhang, 2018	Yes
PAH	math	Margolis, 2021	Ish (trend)
PAH	reading/language	Margolis, 2021	Yes
PM2.5	maths	Shier et al., 2019	Yes
PM2.5	reading/language	Shier et al., 2019	Yes
PM10	maths	Shier et al., 2019	No
PM10	reading/language	Shier et al., 2019	no
Ozone	maths	Shier et al., 2019	Yes
BC	Reading/language	Suglia et al., 2008	Ish (trend)
UFP	Attention/EF	Forns et al., 2017	Yes
SO2	Attention/EF	Gui et al., 2020	Yes
Ozone	Attention/EF	Gui et al., 2020	yes
Benzene	Attention/EF	Kicinski et al., 2016	yes
Composite	Attention/EF	Kicinski et al., 2015	yes
NO2	Memory and learning	Powdthavee and Oswald, 2020	yes
BC	Memory and learning	Suglia et al., 2008	Ish (trend)
PM2.5	Memory and learning	Powdthavee and Oswald, 2020	yes
PAH	Memory and learning	Suter et al., 2018	Ish (HIV only)
Composite	Memory and learning	Kicinski et al., 2015	no
PM2.5	Processing speed/reaction time	Saenen, 2016	Yes
PM10	Processing speed/reaction time	Saenen, 2016	Yes
Composite	Motor skills	Kicinski et al., 2015	No
PM2.5	Academic ability	Amanzadeh, 2020	Yes
PM10	Academic ability	Amanzadeh, 2020	Yes
Ozone	Academic ability	Amanzadeh, 2020	No
NOx	Academic ability	Amanzadeh, 2020	No

CO	Academic ability	Amanzadeh, 2020	No
SO2	Academic ability	Amanzadeh, 2020	Positive association
CO2	Academic ability	Dorizas, 2015	yes
EC	SDQ behavioural/general difficulties	Alemany, 2018	yes
SPM	CBCL behavioural problems-attention, delinquent or aggressive behaviour.	Yorifuji, 2017	yes
SO2	CBCL behavioural problems-attention, delinquent or aggressive behavior.	Yorifuji, 2017	yes
PAH	SDQ/CBC aggressive symptoms	Jorcano, 2019;	No
ozone	SDQ /emotional symptoms	Dales, 2016	Yes
PAH	SDQ/CBCL//emotional symptoms	Jorcano, 2019	no
ozone	Happiness yes no	Dales, 2016	no
PM10	Mental Wellbeing	Bloemsma, 2022	yes
SO2	Psychiatric emergency	Kim, 2019	no
PM10	Psychiatric emergency	Kim, 2019	no
PM2.5	Anxiety	Roberts, 2019	No
NO2	Anxiety	Roberts, 2019	no
PM10	Anxiety	Zhao, 2020	yes
PM10	Suicide	Casas, 2017	Yes
ozone	Suicide	Casas, 2017	yes
PM2.5	Self-harm	Liu, 2018	Yes
CO	Self-harm	Liu, 2018	Yes
NO2	Self-harm	Liu, 2018	No
Ozone	Self-harm	Liu, 2018	Yes
SO2	Self-harm	Liu, 2018	no
PM10	psychosis	Newbury, 2019	No
PM2.5	Psychiatric medication	Oudin, 2016	Yes

PM10	Psychiatric medication	Oudin, 2016	Yes
NO2	Psychiatric medication	Oudin, 2016	Yes

IV: Noise review search terms

A) Cognition

1. PUBMED

("noise annoyance"[Title/Abstract] or "noise pollution"[Title/Abstract] or "urban noise"[Title/Abstract] or "city noise"[Title/Abstract] or "urban sound"[Title/Abstract] or "environmental noise"[Title/Abstract] or "community noise"[Title/Abstract] or "traffic noise"[Title/Abstract] or "wind turbine noise"[Title/Abstract] or " wind farm noise"[Title/Abstract] or " wind turbine sound"[Title/Abstract] or "wind farm sound"[Title/Abstract] or " aircraft noise"[Title/Abstract] or " airport noise"[Title/Abstract] or " railway noise"[Title/Abstract] or "road traffic noise"[Title/Abstract] or "transportation noise"[Title/Abstract] or " train noise"[Title/Abstract] or " leisure noise"[Title/Abstract] or " leisure-time noise"[Title/Abstract] or "neighbourhood noise"[Title/Abstract] or "neighborhood noise"[Title/Abstract] or "household noise"[Title/Abstract] or "low frequency noise"[Title/Abstract] or "classroom noise"[Title/Abstract] or "school noise"[Title/Abstract] or "high-volume music "[Title/Abstract] or "high-volume noise"[Title/Abstract] or "noise from personal electronic devices"[Title/Abstract] or "noise from mp3 players"[Title/Abstract] or "noise from childrens toys"[Title/Abstract] or "hospital noise"[Title/Abstract] or "combined noise exposure"[Title/Abstract] or "noise nuisance"[Title/Abstract] or "noise exposure"[Title/Abstract] or "truck noise"[Title/Abstract] or "motor vehicle noise"[Title/Abstract] or "noise load"[Title/Abstract] or "entertainment noise"[Title/Abstract] or "noise from mobile phones"[Title/Abstract] or "noise from personal audio devices"[Title/Abstract] or "noise from personal music players"[Title/Abstract] or "combined exposure to noise and vibration"[Title/Abstract] or "combined exposure to noise and air pollution"[Title/Abstract]) AND (cogniti*[Title/Abstract] OR intelligence[Title/Abstract] OR neuropsycholog*[Title/Abstract] OR neurobehavio*[Title/Abstract] OR neurodevelopment[Title/Abstract] OR attention[Title/Abstract] OR memory[Title/Abstract] OR executive function[Title/Abstract] OR IQ[Title/Abstract] OR psychomotor[Title/Abstract] OR verbal[Title/Abstract] OR visual spatial[Title/Abstract] OR visual motor[Title/Abstract] OR academic[Title/Abstract] OR learning[Title/Abstract] OR reading[Title/Abstract] OR reaction time[Title/Abstract] OR school[Title/Abstract] OR education*[Title/Abstract] OR reasoning[Title/Abstract] OR flexibility[Title/Abstract] OR hyperactivity[Title/Abstract] OR problem-solving[Title/Abstract])

2. WEB OF SCIENCE

((AB=("noise annoyance" OR "noise pollution" OR "urban noise" OR "city noise" OR "urban sound" OR "environmental noise" OR "community noise" OR "traffic noise" OR "wind turbine noise" OR " wind farm noise" OR " wind turbine sound" OR "wind farm sound" OR " aircraft noise" OR " airport noise" OR " railway noise" OR "road traffic noise" OR "transportation noise" OR " train noise" OR " leisure noise" OR " leisure-time noise" OR "neighbourhood noise" OR "neighborhood noise" OR "household noise" OR "low frequency noise" OR "classroom noise" OR "school noise" OR "high-volume music " OR "high-volume noise" OR "noise from personal electronic devices" OR "noise from mp3 players" OR "noise from childrens toys" OR "hospital noise" OR "combined noise exposure" OR "noise nuisance" OR "noise exposure" OR "truck noise" OR "motor vehicle noise" OR "noise load" OR "entertainment noise" OR "noise from mobile phones" OR "noise from personal audio devices" OR "noise from personal music players" OR "combined exposure to noise and vibration" OR "combined exposure to noise and air pollution")) OR TI=("noise annoyance"

OR "noise pollution" OR "urban noise" OR "city noise" OR "urban sound" OR "environmental noise" OR "community noise" OR "traffic noise" OR "wind turbine noise" OR " wind farm noise" OR " wind turbine sound" OR "wind farm sound" OR " aircraft noise" OR " airport noise" OR " railway noise" OR "road traffic noise" OR "transportation noise" OR " train noise" OR " leisure noise" OR " leisure-time noise" OR "neighbourhood noise" OR "neighborhood noise" OR "household noise" OR "low frequency noise" OR "classroom noise" OR "school noise" OR "high-volume music " OR "high-volume noise" OR "noise from personal electronic devices" OR "noise from mp3 players" OR "noise from childrens toys" OR "hospital noise" OR "combined noise exposure" OR "noise nuisance" OR "noise exposure" OR "truck noise" OR "motor vehicle noise" OR "noise load" OR "entertainment noise" OR "noise from mobile phones" OR "noise from personal audio devices" OR "noise from personal music players" OR "combined exposure to noise and vibration" OR "combined exposure to noise and air pollution")) AND (AB=("cogniti*"OR "intelligence" OR "neuropsycholog* OR "neurobehavio*"OR "neurodevelopment" OR "attention" OR "memory" OR "executive function" OR "IQ" OR "psychomotor" OR "verbal" OR "visual spatial" OR "visual motor" OR "academic" OR "learning" OR "reading" OR "reaction time" OR "school" OR "education*" OR "reasoning" OR "flexibility" OR "hyperactivity" OR "problem solving")) OR TI=("cogniti*"OR "intelligence" OR "neuropsycholog* OR "neurobehavio*"OR "neurodevelopment" OR "attention" OR "memory" OR "executive function" OR "IQ" OR "psychomotor" OR "verbal" OR "visual spatial" OR "visual motor" OR "academic" OR "learning" OR "reading" OR "reaction time" OR "school" OR "education*" OR "reasoning" OR "flexibility" OR "hyperactivity" OR "problem solving"))

3. PSYCHINFO and Embase (via Ovid)

noise annoyance" or "noise pollution" or "urban noise" or "city noise" or "urban sound" or "environmental noise" or "community noise" or "traffic noise" or "wind turbine noise" or " wind farm noise" or " wind turbine sound" or "wind farm sound" or " aircraft noise" or " airport noise" or " railway noise" or "road traffic noise" or "transportation noise" or " train noise" or " leisure noise" or " leisure-time noise" or "neighbourhood noise" or "neighborhood noise" or "household noise" or "low frequency noise" or "classroom noise" or "school noise" or "high-volume music " or "high-volume noise" or "noise from personal electronic devices" or "noise from mp3 players" or "noise from childrens toys" or "hospital noise" or "combined noise exposure" or "noise nuisance" or "noise exposure" or "truck noise" or "motor vehicle noise" or "noise load" or "entertainment noise" or "noise from mobile phones" or "noise from personal audio devices" or "noise from personal music players" or "combined exposure to noise and vibration" or "combined exposure to noise and air pollution").ti,ab.
AND
(cogniti* or intelligence or neuropsycholog* or neurobehavio* or neurodevelopment or attention or memory or executive function or IQ or psychomotor or verbal or visual spatial or visual motor or academic or learning or reading or reaction time or school or education* or reasoning or flexibility or hyperactivity or problem-solving).ti,ab.

B) Mental health

1. WebofScience (not exact search)

(TS= (neurocognitive) OR TS= (cognition) OR TS= (cognitive) OR TS= (neuropsychological) OR TS= (neuropsychology) OR TS=(neurocognitive) OR TS= ("task performance") OR TS= (memory) OR TS= (recognition) OR TS= (attention) OR TS= (vigilance) OR TS= ("response variability") OR TS= ("motor speed") OR TS= ("naming speed") OR TS= ("interference control") OR TS= ("delay aversion") OR TS= ("choice delay") OR TS= ("reaction time") OR TS= ("response inhibition") OR TS= (psychomotor)

OR TS=(“problem solving”) OR TS= (intelligence) OR TS= (executive function)) AND (TS= (traffic) OR TS= (pollution) OR TS= (environment) OR TS= (environmental) OR TS= (occupational) OR TS= (residential) OR TS= (railway) OR TS= (airport) OR TS= (road) OR TS= (transportation) OR TS= (aircraft) OR TS= (transport) OR TS=(neighborhood)) AND (TS= (children) OR TS= (adolescent) OR TS= (school) OR TS= (teenager) OR TS= (childhood) OR TS= (preschool))

2. **Pubmed (above terms exploded)**

((((((((((((((((((((((((((((("Affective Symptoms"[Mesh]) OR "Mental Health"[Mesh]) OR "Mental Health"[tiab]) OR wellbeing[tiab]) OR emotional problems[tiab]) OR ("Strengths and Difficulties")) OR mental wellbeing[tiab]) OR psychological wellbeing[tiab]) OR "Quality of Life"[Mesh]) OR "quality of life"[tiab]) OR mental hygiene[tiab]) OR SDQ[tiab]) OR emotional symptom*[tiab]) OR prosocial behaviour*[tiab]) OR prosocial behavior*[tiab]) OR psychological distress[tiab]) OR conduct problem*[tiab]) OR hyperactivity[tiab]) OR peer relationship problem[tiab]) OR inattention[tiab]) OR psychological health[tiab]) OR "Aggression"[Mesh]) OR "Bullying"[Mesh]) OR aggressi*[tiab]) OR bullying[tiab]) OR "Behavioral Symptoms"[Mesh])) OR behavioral[tiab]) OR conduct disorder[tiab]) OR oppositional defiant disorder[tiab]) OR odd[tiab]) OR behavioural[tiab]) OR "Mood Disorders"[Mesh]) OR Impulsivity[tiab]) OR Overactivity[tiab]) AND (((noise [tiab]) AND (residential [tiab] OR environmental [tiab] OR transportation [tiab] OR traffic [tiab] OR aircraft [tiab] OR pollution [tiab] OR rail?way [tiab] OR airport [tiab]))) OR ("Noise, Transportation"[Mesh] OR "Noise"[Mesh]) AND (((((children[tiab]) OR adolescent[tiab]) OR school*[tiab]) OR child[tiab]) OR child[MeSH]) OR teenager[tiab] OR childhood[tiab] OR pre?school OR "Adolescent"[Mesh])

3. **SCOPUS**

(((TITLE-ABS-KEY (function) OR TITLE-ABS-KEY (performance) OR TITLE-ABS-KEY (functioning))) AND (TITLE-ABS-KEY (executive))) OR (TITLE-ABS-KEY (psychomotor) OR TITLE-ABS-KEY ("reaction time") OR TITLE-ABS-KEY ("task performance") OR TITLE-ABS-KEY (intelligence) OR TITLE-ABS-KEY ("response variability") OR TITLE-ABS-KEY ("motor speed") OR TITLE-ABS-KEY ("problem solving") OR TITLE-ABS-KEY ("choice delay") OR TITLE-ABS-KEY ("naming speed") OR TITLE-ABS-KEY ("response inhibition") OR TITLE-ABS-KEY ("delay aversion") OR TITLE-ABS-KEY ("interference control") OR TITLE-ABS-KEY (vigilance) OR TITLE-ABS-KEY (attention) OR TITLE-ABS-KEY (recognition) OR TITLE-ABS-KEY (memory) OR TITLE-ABS-KEY (neurocognitive) OR TITLE-ABS-KEY (neuropsychology) OR TITLE-ABS-KEY (neuropsychological) OR TITLE-ABS-KEY (cognitive) OR TITLE-ABS-KEY (cognition) OR TITLE-ABS-KEY (neurocognitive))
AND
((TITLE-ABS-KEY (traffic) OR TITLE-ABS-KEY (pollution) OR TITLE-ABS-KEY (railway) OR TITLE-ABS-KEY (airport) OR TITLE-ABS-KEY (residential) OR TITLE-ABS-KEY (transportation) OR TITLE-ABS-KEY (aircraft) OR TITLE-ABS-KEY (environmental) OR TITLE-ABS-KEY (occupational) OR TITLE-ABS-KEY (aircraft)) OR TITLE-ABS-KEY (exposure)) AND (TITLE-ABS-KEY (noise))
AND
(TITLE-ABS-KEY (preschool) OR TITLE-ABS-KEY (children) OR TITLE-ABS-KEY (adolescent) OR TITLE-ABS-KEY (school*) OR TITLE-ABS-KEY (teenager) OR TITLE-ABS-KEY (childhood))

V: Associations assessed by one study in noise literature

Exposure	Outcome	Reference	Supportive of harmful association?
Apartment noise	Auditory discrimination	Cohen, 1973	Yes
Aircraft noise	Auditory discrimination	Hygge et al., 2002	yes
Traffic noise	Academic achievement	Lukas, 1981	yes
Traffic noise	Fluid intelligence	Julvez, 2021	no
Traffic noise	reading	Van Kempen et al., 2010	no
Apartment noise	reading	Cohen, 1973	Yes
Railway noise	General psychopathology / problems	Tangermann, 2022	No
General noise	General psychopathology	Dzhambov, 2017	Ish, trend
Aircraft noise	General psychopathology / problems	Haines, 2001d	Yes
General noise	Happiness//wellbeing	Benita, 2019	Beneficial
Railway noise	Internalising	Tangermann, 2022	No
General noise	Substance use	Rudolph, 2019	Yes
General noise	Social adaptability	Ristovska, 2005	yes
Aircraft noise	Peer problems	Haines, 2001d	No
Aircraft noise	Emotional problems	Haines, 2001d	No
Aircraft noise	Stress proneness	Haines, 2001d	Yes
Railway noise	Externalising	Tangermann, 2022	No,
Aircraft noise	Hyperactivity problems	Haines, 2001d	Yes
Aircraft noise	Conduct problems	Haines, 2001d	no

VI :Consolidated criteria for reporting qualitative research checklist, applied to research in Chapter 4

Developed from:
Tong A, Sainsbury P, Craig J. Consolidated criteria for reporting qualitative research (COREQ): a 32-item checklist for interviews and focus groups. *International Journal for Quality in Health Care*. 2007. Volume 19, Number 6: pp. 349 – 357

No. Item	Guide questions/description	Reported on Page #
Domain 1: Research team and reflexivity		
Personal Characteristics		
1. Interviewer/facilitator	Which author/s conducted the interview or focus group?	122
2. Credentials	What were the researcher's credentials? E.g. PhD, MD	122
3. Occupation	What was their occupation at the time of the study?	122
4. Gender	Was the researcher male or female?	122
5. Experience and training	What experience or training did the researcher have?	121, 122
Relationship with participants		
6. Relationship established	Was a relationship established prior to study commencement?	121
7. Participant knowledge of the interviewer	What did the participants know about the researcher? e.g. personal goals, reasons for doing the research	121
8. Interviewer characteristics	What characteristics were reported about the interviewer/facilitator? e.g. Bias, assumptions, reasons and interests in the research topic	122, 142-145
Domain 2: study design		
Theoretical framework		
9. Methodological orientation and Theory	What methodological orientation was stated to underpin the study? e.g. grounded theory, discourse analysis, ethnography, phenomenology, content analysis	122
Participant selection		

10. Sampling	How were participants selected? e.g. purposive, convenience, consecutive, snowball	121
11. Method of approach	How were participants approached? e.g. face-to-face, telephone, mail, email	121
12. Sample size	How many participants were in the study?	123
13. Non-participation	How many people refused to participate or dropped out? Reasons?	N/A
Setting		
14. Setting of data collection	Where was the data collected? e.g. home, clinic, workplace	122
15. Presence of non-participants	Was anyone else present besides the participants and researchers?	122
16. Description of sample	What are the important characteristics of the sample? e.g. demographic data, date	124
Data collection		
17. Interview guide	Were questions, prompts, guides provided by the authors? Was it pilot tested?	121-122, 280 (Appendix VII)
18. Repeat interviews	Were repeat inter views carried out? If yes, how many?	N/A
19. Audio/visual recording	Did the research use audio or visual recording to collect the data?	122
20. Field notes	Were field notes made during and/or after the interview or focus group?	122
21. Duration	What was the duration of the interviews or focus group?	123
22. Data saturation	Was data saturation discussed?	123
23. Transcripts returned	Were transcripts returned to participants for comment and/or correction?	N/A
Domain 3: analysis and findings		
Data analysis		
24. Number of data coders	How many data coders coded the data?	123
25. Description of the coding tree	Did authors provide a description of the coding tree?	281 (Appendix VIII)
26. Derivation of themes	Were themes identified in advance or derived from the data?	122
27. Software	What software, if applicable, was used to	123

	manage the data?	
28. Participant checking	Did participants provide feedback on the findings?	N/A
Reporting		
29. Quotations presented	Were participant quotations presented to illustrate the themes/findings? Was each quotation identified? e.g. participant number	125-136
30. Data and findings consistent	Was there consistency between the data presented and the findings?	125-136
31. Clarity of major themes	Were major themes clearly presented in the findings?	125-136
32. Clarity of minor themes	Is there a description of diverse cases or discussion of minor themes?	125-136

VII: Interview protocol: environmental thoughts and feelings in adolescents

Introduction:
Remind participants that there are no correct answers, that the answers are completely anonymous and taking part is completely voluntary, and ask if they have any questions.
Protocol for apparent distress
Ask if they are feeling okay, if they would like to take a break, and if they wish to continue, reminding them they can stop at any point without penalty.
Debrief
Finally, a debrief takes place with an opportunity for questions and a handout with details of support for climate or general distress.

Questions	Prompts
1) Tell me about your day, how is it going?	What have you been up to?
2) What do you know about air pollution?	Its okay if you don't know much about it…
3) What do you know about noise pollution?	How do you think it affects young people, if at all?
4) What are some other aspects of city living?	Its okay if you don't know much about it… How do you think it affects young people, if at all?
5) Where do you get your information about these issues from?	How do you think these affect young people, if at all?
6) What do other people in your life think about these problems?	What are your influences? The news? Your friends?
7) How does it make you feel to think about (for example, on the news or when people around you are talking about it)?	It's okay if you don't know/ if it doesn't come up…
8) Thinking about things like air pollution, noise, and *things they have come up with*, what do you notice/ see/ experience in your life?	It's okay if it doesn't really occur to you much…
9) How does this make you feel?	
10) What is it like where you live?	
11) In what way does your environment affect you? That is, where you live, travel, study, play, socialise?	Does it feel clean and safe? Can it affect your... sleep/health? Relationships? Schoolwork? Behaviours?
12) How might a young person's environment affect their mental health?	Environment meaning, what's around you and when - it doesn't need to be noise and pollution related
13) Are there any other environmental factors that you think affect young people emotionally, maybe that they aren't directly exposed to?	E.g. on a global scale
14) Do you have any thoughts or feelings about what factors are the most important when it comes to young people's mental health?	
15) Who do you think are the ones with the power to affect your environments?	
16) Do you have any thoughts or feelings about what they are doing, or what they could or should be doing?	
17) How does this make you feel?	

VIII: All codes identified in qualitative analysis

Theme	Subtheme	Code	Example
Impact of local environment	**Harmful influences**	Air pollution-death/health	“I get quite paranoid about that and having health problems because of it” (#8, F, 14)
		Noise- health	"I’ve heard people saying there’s quite a lot of noise pollution in specific jobs and things, and how it can cause damage to you" (#8, F, 14)
		Noise- Sleep	"it definitely lowers how much sleep I get" (#9, F, 18)
		Air pollution- general negative	“in the city near us there are parts where people can’t even open their windows because the air pollution is so bad and it would affect them so badly, and that’s where I go to school, I go to school in that area” (#7, F, 15)
		Noise- annoyance	“I used to really struggle with it and it used to be really annoying” (#5, M, 16)
		Noise-Trapped	“It gets quite, I would say quite suffocating and quite saddening in a way because you can’t really get out of that environment” (#13, F, 18)
		Noise- Distraction	“it’s also quite distracting, if you’re trying to do your work or whatever and you have constant background noise” (#5, M, 16)
		Overcrowding	“a city where it feels very bustling, and if there are too many people I get really stressed and I cannot deal with it” (#7, F, 15)
		Cars and busses	“the traffic is quite bad, you’ve got to look out for roads , jammed cars” (#2, M, 16)
		Noise- Stress/anxiety	"I also don’t like walking around places where it’s really noisy because I get stressed. It’s quite overwhelming" (#8, F, 14)
		Litter/ cleanliness	“It’s just the constant thoughts that come to mind. Like avoiding air pollution and noise pollution and avoiding rubbish and touching things, because that’s just something you have to think about when you’re in a city because everything is sort of infested there. It’s sort of stressful to think about constantly” (#8, F, 14)

		Water pollution	“it’s kind of annoying when you wanna go out somewhere, to look at the flash or the canal and it’s like, it's just like full of pollution, it’s like supposed to be somewhere you go where it’s nice, but it’s all gross" (#10, F, 14)
		Harm to Wildlife	“the animals and stuff that are in the area, that it could scare away and damage the endemic population of animals” (#5, M, 16)
		Air pollution- climate	"I know about like global dimming and the warming of the atmosphere” (#6, F, 16)
		Light	“when I’m outside and its dark but it’s just light and blaring , it’s a bit much" (#12, M, 16)
		Safety	“if you’re in a situation where there are people getting beaten up or whatever” (#1, F, 16)
	Helpful influences	Personal space	“being able to have the space to do stuff by yourself, like a safe environment to be able to do stuff without constant supervision is definitely important just for like your own peace of mind” (#6, F, 16)
		Greenspace and nature	“when I go out of the city for example, it’s a massive shift, you automatically just feel a bit more peaceful , relaxed, not being overstimulated with all these things just outside.” (#12, M, 16)
		Fresh air	“it’s harder to get out, it’s almost like fresh air in a way. I know you say when you’re going out you’re getting fresh air but in the city it doesn’t necessarily feel like that," (#2, M, 16)
		Accessibility	“I like that there’s so much to see and things, and I like the fact that things are quite available here” (#8, F, 14)
Efficacy	**Progress and empowerment**	Young people- activism	“we all go and join in the climate march strikes, doing all that, which I think is fairly, sort of, that’s a sort of constant for most teenagers my age." (#5, M, 16)

		Young people- awareness/ interest	"we're probably the most conscious generation about it, the crisis that's going on in the world… there is a recurring theme in conversation and topic, which we talk about, which is the environment, and they're all quite fussed about it" (#5, M, 16)
		Passion	"Now, I'm a little bit angry but I'm more passionate about it, and I just try and be as active as possible, but definitely passion and just kind of sadness and anger and happiness and just, it's so many emotions" (#9, F, 18)
		Making a difference	"It makes me feel a bit more hopeful, you know if I've taken action as an individual, hopefully some other people out there are doing the same and as a collective, it will have an impact" (#12, M, 16)
		Community	"It kind of feels like you have some sort of input into something a lot bigger and it can be quite empowering sort of thing to be part of a global thing that others are putting their efforts toward" (#6, F, 16)
	Powerlessness	Lack of control	"I feel like it is out of our control because things are happening that, no-one can really control to an extent… I feel kind of helpless in a way" (#13, F, 13)
		Not knowing how to help	"obviously no-one knows every single thing about what's best, for the environment and there's , people think different things, so it's hard to know what to do, sort of thing sometimes." (#6, F, 15)
		Too late	"A lot of it is worrying too about whether or not it's too far gone or nothing can be done, I think that's where a lot of it comes from" (#12, M, 16)
Challenging emotions		Annoyance	"annoyed because I feel like people should be more aware of what they're putting into the earth" (#10, F, 14)
		Anger	"It's anger, a lot of anger, sadness, often there's always a sense of I could do more, I could do more" (#4, NB, 17)
		Frustration	"Just sort of frustrated. It feels like things are out of our control and people are ignoring the problems, when the people acknowledging it can't do anything." (#8, F, 14)

		Guilt	"I feel sorry for them, kind of feel really, like, guilty that you can't do anything more than sort of feel sympathy. Because we don't have any power until we can get jobs and sort of invest in helping" (#8, F, 14)
		Sadness	"Disempowered and just sad that we are doing everything we can at the moment and yet we are still being disengaged, not listened to." (#9, F, 18)
		Depression	"Us just being told that our general environment is not doing well at all, it's kind of hard to feel positive about anything" (#14, F, 16)
		Anxiety	"I feel, like, concerned, worried about the future…I think it's quite daunting" (#4, NB, 17)
Hindrances	**Indifference**	Avoidance	"It's quite, depressing in a way… I feel like I'm just gonna read more stuff about it and it's just gonna make me realise how big a problem it is, and I'd rather not to be honest" (#3, M, 18)
		Awareness/ denial	"In some cases there's a lack of education, like they don't know all of it, or they think that it's a hoax or whatever… I think we should show more coverage of places in the world where it is already having a big impact" (#7, F, 15)
		Unaffected	"it's not like you can see it, that's one of the things with humans isn't it, that's why people smoke, that's why people eat unhealthy for a long period of time and stuff, cause they can't see the effects its having onto them, they just keep on doing it." (#5, M, 16)
	Power/ responsibility	Governments	"So many people I know would do anything to have the, particularly politicians have that kind of power to make a real change, and they're just not" (#7, F, 15)
		Big companies	"it's the oil companies, it's the mining companies, it's the deforestation companies, that have the most power because they invest in the government so they make money for the governments, so obviously the government's gonna let them do things which are unmoral and unjust," (#9, F, 18)

		Blame for older generation	“I think a lot of older people, the media calls us lazy but we get a big sense that they’re not doing anything " (#1, F, 16)
	Other/ competing priorities	Policy priorities	"In a way they’ve got so much going on it’s sort of pushed aside, and that’s why I think it’s important that you know there are campaigns going on, to try and raise more awareness for the government that this is what they need to prioritise.” (#2, M, 16)
		Personal finances & lifestyle	“Unfortunately a lot of the environmentally friendly stuff is the more expensive stuff…its hard in the modern day world to do loads for it without sacrificing” (#5, M, 16)
		Profit and economy	"I think they’re concerned with the economy a lot sometimes” (#14, F, 16)
Information	**Sources**	Social influences	“all of the people I spend time with are quite like-minded” (#4, NB, 17)
		School influence	"I know that my school does, pretty well, but like my sister’s school doesn’t at all" (#6, F, 16)
		Social Media & Media	"I would say social media, mostly for example twitter and Instagram, there’s always something trending like either a hashtag or a picture that’s circulating, that catches my eye, like for example you might see a picture of melted icecaps or like a trending hashtag so that’s where I get my information from" (#13, F, 18)
		Gender differences	“Definitely seems in my friend group that the girls care more about it, which I don’t know if that’s just my group or, but people are aware, and people do care.” (#3 M, 18)
	Stress and mistrust	Mistrust of media	"there’s quite a lot on social media, which might not be that reliable, but there’s quite a lot out there” (#15, F, 17)
		Information/ news stress	“It can be bad in the sense that if you’re constantly checking up on the news it can make you feel quite stressed or like everything is going wrong” (#11, F, 15)
Perceptions of the future	**Hopeful for change**	Positive change	"I feel like the world is changing for the better in terms of that, there is awareness," (#13, F, 18)
		Hope/ reassurance	"I just take it with a pinch of salt because it’s like the world is changing and the change could be for the better it could be for the worse but if we’re all doing our little part then

			hopefully you can see it in a positive light, that you've done something to probably see the better end of it.. " (#13, F, 18)
	Catastrophic	Negative change	"all of this you kind of see, slowly going downhill, and if you follow that you can see its going to affect us, and if we have kids our kids, and if they manage to have kids, their kids. You know I think were a generation with great technology, great healthcare, but it's all gonna go downhill in our generation." (#1, F, 16)
		World uninhabitable	"we're just kind of always being bombarded with this information that our world is slowly dying and it's hard to feel like motivated for anything in our future if that makes sense, we're just kind of always being told that we don't really, specifically, have a future, like it's not gonna be one that we wanna live in" (#14, F, 16)
		Time/ urgency	"We kind of feel like we're running out of time…A lot of adults kind of don't feel the same sense of urgency, I think. And particularly the older people I find definitely don't have a sense of urgency, probably because unfortunately they're not going to be around for too much longer to see the effects" (#1, F, 16)

IX: Associations between noise exposure and psychological outcomes within the M25 only

Outcome	Model	Lden Estimate	SE	p	Lday Estimate	SE	p	Leve Estimate	SE	p	Lnight Estimate	SE	p
BL SDQ	1	0.037	0.017	0.025	-0.024	0.017	0.142	0.037	0.016	0.019	0.037	0.017	0.028
BL SDQ	2	0.025	0.016	0.132	0.013	0.018	0.481	0.026	0.016	0.104	0.025	0.017	0.142
BL SDQ	3	0.024	0.017	0.146	0.019	0.02	0.331	0.025	0.016	0.117	0.024	0.017	0.156
BL Internalising	1	0.021	0.01	0.028	-0.01	0.01	0.299	0.021	0.009	0.024	0.021	0.01	0.029
BL Internalising	2	0.016	0.01	0.096	0.007	0.011	0.489	0.016	0.009	0.087	0.016	0.01	0.099
BL Internalising	3	0.016	0.01	0.092	0.011	0.012	0.348	0.016	0.009	0.084	0.017	0.01	0.095
BL Externalising	1	0.016	0.01	0.123	-0.014	0.01	0.163	0.016	0.01	0.095	0.016	0.01	0.133
BL Externalising	2	0.009	0.01	0.391	0.005	0.011	0.628	0.01	0.01	0.314	0.008	0.01	0.418
BL Externalising	3	0.008	0.01	0.446	0.008	0.012	0.524	0.009	0.01	0.366	0.007	0.01	0.473
BL Executive Function	1	-0.003	0.002	0.109	0.012	0.002	0	-0.003	0.002	0.111	-0.003	0.002	0.109
BL Executive Function	2	0	0.002	0.957	0.001	0.002	0.805	0	0.002	0.944	0	0.002	0.959
BL Executive Function	3	0.001	0.002	0.746	0.001	0.002	0.54	0.001	0.002	0.739	0.001	0.002	0.748
BL Culture Fair Intelligence Test	1	0.003	0.01	0.778	0.046	0.01	0	0.003	0.01	0.754	0.003	0.011	0.785
BL Culture Fair Intelligence Test	2	0.015	0.01	0.154	-0.002	0.011	0.883	0.014	0.01	0.143	0.015	0.01	0.157
BL Culture Fair Intelligence Test	3	0.016	0.01	0.108	0.01	0.013	0.431	0.016	0.01	0.101	0.017	0.01	0.11
BL Processing Speed	1	0.002	0.002	0.24	-0.002	0.002	0.266	0.002	0.002	0.204	0.002	0.002	0.256
BL Processing Speed	2	0.002	0.002	0.345	0	0.002	0.951	0.002	0.002	0.304	0.002	0.002	0.362
BL Processing Speed	3	0.001	0.002	0.603	0	0.002	0.869	0.001	0.002	0.542	0.001	0.002	0.625

Outcome	Model	Lden Estimate	SE	p	Lday Estimate	SE	p	Leve Estimate	SE	p	Lnight Estimate	SE	p
BL Speech-in-noise Threshold	1	0.017	0.008	0.027	-0.002	0.002	0.266	0.002	0.002	0.204	0.002	0.002	0.256
BL Speech-in-noise Threshold	2	0	0.01	0.994	0	0.002	0.951	0.002	0.002	0.304	0.002	0.002	0.362
BL Speech-in-noise Threshold	3	-0.003	0.01	0.779	0	0.002	0.869	0.001	0.002	0.542	0.001	0.002	0.625
F1 SDQ	1	0.015	0.018	0.422	-0.029	0.018	0.101	0.016	0.018	0.369	0.015	0.019	0.437
F1 SDQ	2	0.014	0.017	0.411	0.015	0.018	0.42	0.015	0.016	0.372	0.014	0.017	0.421
F1 SDQ	3	0.01	0.017	0.546	0.016	0.021	0.434	0.011	0.017	0.496	0.01	0.018	0.557
F1 Internalising	1	0.001	0.011	0.921	-0.031	0.011	0.003	0.002	0.011	0.852	0.001	0.011	0.935
F1 Internalising	2	0.004	0.01	0.682	-0.004	0.011	0.707	0.004	0.01	0.651	0.004	0.01	0.686
F1 Internalising	3	0.003	0.01	0.775	0	0.012	0.974	0.003	0.01	0.73	0.003	0.011	0.782
F1 Externalising	1	0.014	0.012	0.242	0.002	0.011	0.856	0.014	0.011	0.215	0.014	0.012	0.251
F1 Externalising	2	0.003	0.01	0.759	0.018	0.011	0.109	0.003	0.01	0.729	0.003	0.01	0.768
F1 Externalising	3	0.003	0.01	0.8	0.013	0.012	0.308	0.003	0.01	0.776	0.003	0.01	0.806
F1 PHQ-9 Depression	1	0	0.02	0.998	-0.009	0.018	0.61	0.001	0.019	0.949	0	0.02	0.984
F1 PHQ-9 Depression	2	-0.007	0.019	0.713	0.004	0.022	0.868	-0.006	0.018	0.754	-0.007	0.02	0.703
F1 PHQ-9 Depression	3	-0.007	0.02	0.718	-0.005	0.023	0.816	-0.006	0.019	0.755	-0.007	0.02	0.709
F1 GAD-7 Anxiety	1	0.03	0.017	0.077	0.016	0.016	0.308	0.03	0.016	0.059	0.029	0.017	0.084
F1 GAD-7 Anxiety	2	0.028	0.016	0.089	0.017	0.018	0.36	0.028	0.015	0.071	0.027	0.016	0.096
F1 GAD-7 Anxiety	3	0.026	0.016	0.108	0.017	0.019	0.374	0.027	0.016	0.086	0.026	0.017	0.116
F1 Executive Function	1	-0.003	0.002	0.157	0.01	0.002	0	-0.003	0.002	0.153	-0.003	0.002	0.159
F1 Executive Function	2	0	0.002	0.88	0	0.002	0.949	0	0.002	0.838	0	0.002	0.894
F1 Executive Function	3	-0.001	0.002	0.687	0	0.002	0.885	-0.001	0.002	0.637	-0.001	0.002	0.704

Outcome	Model	Lden Estimate	SE	p	Lday Estimate	SE	p	Leve Estimate	SE	p	Lnight Estimate	SE	p
F1 Culture Fair Intelligence Test	1	-0.01	0.011	0.358	0.051	0.011	0	-0.011	0.011	0.313	-0.01	0.012	0.375
F1 Culture Fair Intelligence Test	2	-0.004	0.01	0.697	-0.008	0.011	0.438	-0.005	0.009	0.601	-0.003	0.01	0.731
F1 Culture Fair Intelligence Test	3	-0.003	0.01	0.725	-0.003	0.012	0.769	-0.005	0.009	0.625	-0.003	0.01	0.76
F1 Processing Speed	1	0.001	0.002	0.735	0.001	0.002	0.602	0.001	0.002	0.685	0.001	0.002	0.755
F1 Processing Speed	2	-0.001	0.002	0.636	0.001	0.002	0.56	-0.001	0.002	0.702	-0.001	0.002	0.613
F1 Processing Speed	3	-0.002	0.002	0.386	0	0.002	0.97	-0.001	0.002	0.441	-0.002	0.002	0.367
F1 Speech-in-noise Threshold	1	0.022	0.015	0.139	0.001	0.002	0.602	0.001	0.002	0.685	0.001	0.002	0.755
F1 Speech-in-noise Threshold	2	0.01	0.015	0.501	0.001	0.002	0.56	-0.001	0.002	0.702	-0.001	0.002	0.613
F1 Speech-in-noise Threshold	3	0.006	0.015	0.674	0	0.002	0.97	-0.001	0.002	0.441	-0.002	0.002	0.367
F2 CAPE risk	1	0.03	0.03	0.41	0.32	0.36	0.36	0.16	0.19	0.39	0.15	0.18	0.42
F2 CAPE risk	2	0.13	0.22	0.55	0.26	0.45	0.56	0.14	0.23	0.53	0.13	0.22	0.56
F2 CAPE risk	3	0.13	0.22	0.55	0.28	0.46	0.55	0.14	0.23	0.54	0.13	0.22	0.56

X: Associations between exposure to air pollution and/or noise and psychological outcomes in the SCAMP cohort: results for all two-exposure models

Exposures: PM2.5 + Ozone

Outcome	Term	Estimate	SE	p	Model	Estimate.1	SE.1	p.1	Model.1
BL SDQ	PM2.5	-0.237	0.118	0.045	2	-0.187	0.143	0.192	3
BL SDQ	Ozone	-0.178	0.134	0.186	2	-0.194	0.152	0.202	3
BL Internalising	PM2.5	-0.098	0.07	0.158	2	-0.054	0.083	0.517	3
BL Internalising	Ozone	0.005	0.079	0.951	2	-0.039	0.089	0.66	3
BL Externalising	PM2.5	-0.139	0.072	0.056	2	-0.14	0.087	0.107	3
BL Externalising	Ozone	-0.183	0.082	0.026	2	-0.151	0.092	0.101	3
BL Executive Function	PM2.5	0.018	0.013	0.173	2	0.028	0.017	0.096	3
BL Executive Function	Ozone	-0.01	0.015	0.535	2	-0.003	0.018	0.868	3
BL Culture Fair Intelligence Test	PM2.5	-0.106	0.073	0.146	2	-0.049	0.095	0.608	3
BL Culture Fair Intelligence Test	Ozone	-0.078	0.085	0.354	2	-0.091	0.099	0.359	3
BL Processing Speed	PM2.5	-0.004	0.013	0.736	2	-0.018	0.016	0.273	3
BL Processing Speed	Ozone	0.008	0.016	0.618	2	0.009	0.018	0.633	3
BL Speech-in-noise Threshold	PM2.5	-0.194	0.076	0.011	2	-0.121	0.09	0.178	3
BL Speech-in-noise Threshold	Ozone	-0.086	0.082	0.297	2	-0.082	0.092	0.374	3
F1 SDQ	PM2.5	0.15	0.136	0.272	2	0.063	0.163	0.7	3
F1 SDQ	Ozone	0.071	0.142	0.617	2	-0.107	0.156	0.493	3
F1 Internalising	PM2.5	0.013	0.081	0.872	2	-0.017	0.098	0.865	3
F1 Internalising	Ozone	0.09	0.085	0.291	2	-0.007	0.094	0.941	3
F1 Externalising	PM2.5	0.034	0.079	0.672	2	-0.009	0.095	0.922	3
F1 Externalising	Ozone	0.078	0.084	0.355	2	-0.02	0.092	0.83	3

F1 PHQ-9 Depression	PM2.5	-0.023	0.147	0.877	2	0.008	0.171	0.965	3
F1 PHQ-9 Depression	Ozone	0.108	0.169	0.522	2	0.124	0.177	0.485	3
F1 GAD-7 Anxiety	PM2.5	0.172	0.125	0.167	2	0.198	0.142	0.164	3
F1 GAD-7 Anxiety	Ozone	0.099	0.143	0.49	2	0.101	0.149	0.497	3
F1 Executive Function	PM2.5	0.012	0.013	0.366	2	0.017	0.016	0.27	3
F1 Executive Function	Ozone	-0.061	0.014	0	2	-0.042	0.015	0.005	3
F1 Culture Fair Intelligence Test	PM2.5	0	0.078	0.998	2	0.05	0.091	0.586	3
F1 Culture Fair Intelligence Test	Ozone	-0.114	0.082	0.168	2	-0.128	0.089	0.149	3
F1 Processing Speed	PM2.5	0.038	0.015	0.01	2	0.023	0.018	0.191	3
F1 Processing Speed	Ozone	0.032	0.016	0.04	2	0.024	0.017	0.157	3
F1 Speech-in-noise Threshold	PM2.5	-0.047	0.117	0.691	2	-0.112	0.135	0.408	3
F1 Speech-in-noise Threshold	Ozone	0.097	0.119	0.418	2	0.054	0.13	0.68	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis Risk	PM2.5	1.51	0.81	2.88	2	1.51	0.81	2.83	3
CAPE Psychosis Risk	Ozone	1.11	0.54	2.26	2	1.11	0.54	2.25	3

Exposures: PM2.5 + Lden

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	PM2.5	-0.23	0.114	0.044	2	-0.202	0.143	0.158	3
BL SDQ	Lden	0.181	0.096	0.06	2	0.166	0.097	0.088	3
BL Internalising	PM2.5	-0.127	0.067	0.059	2	-0.08	0.083	0.336	3
BL Internalising	Lden	0.114	0.056	0.044	2	0.105	0.057	0.066	3
BL Externalising	PM2.5	-0.103	0.07	0.142	2	-0.131	0.087	0.135	3
BL Externalising	Lden	0.067	0.059	0.255	2	0.062	0.059	0.295	3
BL Executive Function	PM2.5	0.022	0.013	0.093	2	0.029	0.017	0.091	3
BL Executive Function	Lden	-0.003	0.011	0.791	2	0	0.011	0.979	3

BL Culture Fair Intelligence Test	PM2.5	-0.108	0.07	0.125	2	-0.071	0.096	0.458	3
BL Culture Fair Intelligence Test	Lden	0.102	0.06	0.089	2	0.103	0.06	0.084	3
BL Processing Speed	PM2.5	-0.009	0.013	0.462	2	-0.021	0.016	0.189	3
BL Processing Speed	Lden	0.012	0.011	0.284	2	0.008	0.011	0.442	3
BL Speech-in-noise Threshold	PM2.5	-0.18	0.074	0.015	2	-0.11	0.09	0.225	3
BL Speech-in-noise Threshold	Lden	0.043	0.06	0.472	2	0.012	0.06	0.836	3
F1 SDQ	PM2.5	0.1	0.127	0.432	2	0.078	0.16	0.624	3
F1 SDQ	Lden	0.06	0.104	0.563	2	0.048	0.103	0.642	3
F1 Internalising	PM2.5	-0.035	0.076	0.643	2	-0.023	0.097	0.815	3
F1 Internalising	Lden	0.032	0.062	0.603	2	0.021	0.062	0.738	3
F1 Externalising	PM2.5	-0.005	0.074	0.95	2	-0.009	0.093	0.924	3
F1 Externalising	Lden	0.019	0.061	0.755	2	0.016	0.061	0.788	3
F1 PHQ-9 Depression	PM2.5	-0.05	0.14	0.72	2	-0.017	0.167	0.92	3
F1 PHQ-9 Depression	Lden	-0.03	0.116	0.798	2	-0.038	0.117	0.745	3
F1 GAD-7 Anxiety	PM2.5	0.092	0.119	0.439	2	0.117	0.137	0.394	3
F1 GAD-7 Anxiety	Lden	0.139	0.098	0.154	2	0.132	0.098	0.18	3
F1 Executive Function	PM2.5	0.042	0.013	0.001	2	0.033	0.016	0.04	3
F1 Executive Function	Lden	-0.01	0.01	0.299	2	-0.009	0.01	0.368	3
F1 Culture Fair Intelligence Test	PM2.5	0.058	0.073	0.427	2	0.105	0.089	0.238	3
F1 Culture Fair Intelligence Test	Lden	-0.034	0.059	0.559	2	-0.037	0.059	0.533	3

F1 Processing Speed	PM2.5	0.027	0.014	0.045	2	0.02	0.017	0.249	3
F1 Processing Speed	Lden	-0.011	0.012	0.354	2	-0.013	0.011	0.274	3
F1 Speech-in-noise Threshold	PM2.5	-0.112	0.109	0.306	2	-0.146	0.132	0.27	3
F1 Speech-in-noise Threshold	Lden	0.065	0.093	0.482	2	0.042	0.093	0.651	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis Risk	PM2.5	1.44	0.76	2.73	2	1.44	0.76	2.68	3
CAPE Psychosis Risk	Lden	1.03	0.62	1.66	2	1.03	0.63	1.67	3

Exposures: PM2.5 + Lday

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	PM2.5	-0.196	0.113	0.081	2	-0.179	0.143	0.211	3
BL SDQ	Lday	0.141	0.161	0.382	2	0.208	0.179	0.246	3
BL Internalising	PM2.5	-0.105	0.066	0.112	2	-0.061	0.083	0.466	3
BL Internalising	Lday	0.08	0.095	0.397	2	0.108	0.105	0.303	3
BL Externalising	PM2.5	-0.091	0.069	0.187	2	-0.125	0.087	0.15	3
BL Externalising	Lday	0.061	0.099	0.539	2	0.095	0.11	0.385	3
BL Executive Function	PM2.5	0.021	0.013	0.1	2	0.028	0.017	0.101	3
BL Executive Function	Lday	0.002	0.018	0.93	2	0.006	0.021	0.753	3
BL Culture Fair Intelligence Test	PM2.5	-0.084	0.069	0.226	2	-0.052	0.096	0.587	3
BL Culture Fair Intelligence Test	Lday	-0.002	0.1	0.985	2	0.1	0.114	0.381	3
BL Processing Speed	PM2.5	-0.007	0.012	0.591	2	-0.02	0.016	0.217	3

BL Processing Speed	Lday	0.002	0.019	0.925	2	0.008	0.021	0.708	3
BL Speech-in-noise Threshold	PM2.5	-0.165	0.073	0.023	2	-0.101	0.09	0.259	3
BL Speech-in-noise Threshold	Lday	-0.041	0.098	0.673	2	-0.029	0.11	0.788	3
F1 SDQ	PM2.5	0.107	0.124	0.387	2	0.074	0.158	0.64	3
F1 SDQ	Lday	0.108	0.164	0.508	2	0.124	0.186	0.505	3
F1 Internalising	PM2.5	-0.021	0.074	0.779	2	-0.015	0.096	0.879	3
F1 Internalising	Lday	-0.032	0.098	0.743	2	0	0.112	0.999	3
F1 Externalising	PM2.5	-0.015	0.072	0.832	2	-0.022	0.092	0.812	3
F1 Externalising	Lday	0.158	0.098	0.106	2	0.116	0.111	0.297	3
F1 PHQ-9 Depression	PM2.5	-0.064	0.136	0.637	2	-0.023	0.165	0.888	3
F1 PHQ-9 Depression	Lday	0.044	0.191	0.819	2	-0.043	0.206	0.834	3
F1 GAD-7 Anxiety	PM2.5	0.127	0.116	0.274	2	0.15	0.135	0.272	3
F1 GAD-7 Anxiety	Lday	0.122	0.161	0.447	2	0.118	0.17	0.49	3
F1 Executive Function	PM2.5	0.04	0.012	0.001	2	0.03	0.016	0.057	3
F1 Executive Function	Lday	-0.01	0.016	0.546	2	-0.005	0.018	0.773	3
F1 Culture Fair Intelligence Test	PM2.5	0.056	0.071	0.43	2	0.1	0.088	0.257	3
F1 Culture Fair Intelligence Test	Lday	-0.084	0.095	0.376	2	-0.052	0.104	0.619	3
F1 Processing Speed	PM2.5	0.024	0.013	0.075	2	0.016	0.017	0.342	3
F1 Processing Speed	Lday	0.008	0.019	0.699	2	-0.003	0.022	0.895	3
F1 Speech-in-noise Threshold	PM2.5	-0.079	0.106	0.454	2	-0.122	0.131	0.354	3

F1 Speech-in-noise Threshold	Lday	-0.098	0.144	0.493	2	-0.053	0.167	0.751	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis Risk	PM2.5	1.43	0.79	2.66	2	1.43	0.79	2.61	3
CAPE Psychosis Risk	Lday	1.1	0.4	2.86	2	1.1	0.42	2.89	3

Exposures: PM10 + Ozone

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	PM10	0.17	0.104	0.1	2	0.146	0.114	0.201	3
BL SDQ	Ozone	-0.024	0.134	0.857	2	-0.13	0.151	0.389	3
BL Internalising	PM10	0.054	0.061	0.379	2	0.066	0.067	0.327	3
BL Internalising	Ozone	0.062	0.079	0.43	2	-0.019	0.089	0.831	3
BL Externalising	PM10	0.117	0.064	0.066	2	0.081	0.07	0.244	3
BL Externalising	Ozone	-0.086	0.082	0.293	2	-0.105	0.091	0.251	3
BL Executive Function	PM10	0.016	0.012	0.179	2	0.028	0.013	0.037	3
BL Executive Function	Ozone	-0.01	0.015	0.509	2	-0.004	0.018	0.835	3
BL Culture Fair Intelligence Test	PM10	-0.009	0.065	0.892	2	0.025	0.073	0.735	3
BL Culture Fair Intelligence Test	Ozone	-0.042	0.084	0.618	2	-0.081	0.099	0.412	3

BL Processing Speed	PM10	0.003	0.011	0.794	2	-0.013	0.012	0.315	3
BL Processing Speed	Ozone	0.011	0.016	0.487	2	0.01	0.018	0.572	3
BL Speech-in-noise Threshold	PM10	0.058	0.063	0.36	2	-0.008	0.069	0.905	3
BL Speech-in-noise Threshold	Ozone	0.004	0.082	0.961	2	-0.06	0.092	0.513	3
F1 SDQ	PM10	0.037	0.107	0.729	2	-0.031	0.118	0.79	3
F1 SDQ	Ozone	0.015	0.132	0.91	2	-0.129	0.149	0.386	3
F1 Internalising	PM10	-0.009	0.064	0.892	2	-0.035	0.071	0.624	3
F1 Internalising	Ozone	0.081	0.079	0.308	2	-0.007	0.09	0.942	3
F1 Externalising	PM10	-0.054	0.062	0.385	2	-0.065	0.068	0.344	3
F1 Externalising	Ozone	0.044	0.078	0.576	2	-0.026	0.088	0.771	3
F1 PHQ-9 Depression	PM10	0.031	0.116	0.792	2	0.04	0.126	0.749	3
F1 PHQ-9 Depression	Ozone	0.128	0.16	0.421	2	0.129	0.17	0.448	3
F1 GAD-7 Anxiety	PM10	0.203	0.098	0.038	2	0.208	0.105	0.048	3
F1 GAD-7 Anxiety	Ozone	0.084	0.135	0.532	2	0.076	0.141	0.591	3
F1 Executive Function	PM10	0.012	0.01	0.241	2	0.013	0.011	0.242	3
F1 Executive Function	Ozone	-0.063	0.013	0	2	-0.045	0.014	0.002	3

F1 Culture Fair Intelligence Test	PM10	0.053	0.061	0.388	2	0.067	0.067	0.313	3
F1 Culture Fair Intelligence Test	Ozone	-0.096	0.077	0.21	2	-0.132	0.085	0.118	3
F1 Processing Speed	PM10	0.025	0.011	0.031	2	0.017	0.013	0.165	3
F1 Processing Speed	Ozone	0.023	0.015	0.117	2	0.02	0.016	0.233	3
F1 Speech-in-noise Threshold	PM10	0.009	0.09	0.924	2	-0.014	0.098	0.888	3
F1 Speech-in-noise Threshold	Ozone	0.121	0.111	0.276	2	0.087	0.124	0.483	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis Risk	PM10	1.39	0.83	2.44	2	1.39	0.82	2.36	3
CAPE Psychosis Risk	Ozone	1.02	0.51	2.01	2	1.02	0.52	2	3

Exposures: PM10 + Lden

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	PM10	0.145	0.103	0.162	2	0.126	0.117	0.284	3
BL SDQ	Lden	0.101	0.099	0.308	2	0.11	0.099	0.265	3
BL Internalising	PM10	0.011	0.061	0.855	2	0.04	0.069	0.556	3
BL Internalising	Lden	0.089	0.058	0.125	2	0.085	0.058	0.143	3
BL Externalising	PM10	0.134	0.063	0.035	2	0.086	0.071	0.23	3

BL Externalising	Lden	0.012	0.06	0.848	2	0.025	0.06	0.679	3
BL Executive Function	PM10	0.02	0.012	0.092	2	0.029	0.014	0.034	3
BL Executive Function	Lden	-0.005	0.011	0.667	2	-0.003	0.011	0.807	3
BL Culture Fair Intelligence Test	PM10	-0.028	0.065	0.671	2	-0.002	0.075	0.983	3
BL Culture Fair Intelligence Test	Lden	0.091	0.061	0.137	2	0.094	0.061	0.12	3
BL Processing Speed	PM10	-0.002	0.011	0.835	2	-0.016	0.013	0.219	3
BL Processing Speed	Lden	0.011	0.011	0.335	2	0.009	0.011	0.415	3
BL Speech-in-noise Threshold	PM10	0.059	0.063	0.353	2	0	0.071	0.999	3
BL Speech-in-noise Threshold	Lden	-0.006	0.061	0.924	2	-0.004	0.061	0.953	3
F1 SDQ	PM10	0.016	0.105	0.879	2	-0.033	0.119	0.782	3
F1 SDQ	Lden	0.079	0.102	0.442	2	0.066	0.102	0.52	3
F1 Internalising	PM10	-0.033	0.063	0.596	2	-0.039	0.072	0.586	3
F1 Internalising	Lden	0.031	0.061	0.606	2	0.023	0.061	0.707	3
F1 Externalising	PM10	-0.07	0.061	0.25	2	-0.067	0.069	0.328	3
F1 Externalising	Lden	0.033	0.06	0.587	2	0.025	0.06	0.681	3
F1 PHQ-9 Depression	PM10	0.02	0.116	0.865	2	0.037	0.127	0.769	3
F1 PHQ-9 Depression	Lden	-0.045	0.114	0.691	2	-0.048	0.115	0.68	3
F1 GAD-7 Anxiety	PM10	0.161	0.098	0.101	2	0.172	0.106	0.104	3
F1 GAD-7 Anxiety	Lden	0.124	0.096	0.199	2	0.122	0.097	0.21	3
F1 Executive Function	PM10	0.028	0.01	0.007	2	0.017	0.012	0.142	3

F1 Executive Function	Lden	-0.007	0.01	0.46	2	-0.006	0.01	0.511	3
F1 Culture Fair Intelligence Test	PM10	0.081	0.06	0.18	2	0.087	0.067	0.193	3
F1 Culture Fair Intelligence Test	Lden	-0.038	0.058	0.508	2	-0.033	0.058	0.567	3
F1 Processing Speed	PM10	0.022	0.011	0.054	2	0.018	0.013	0.146	3
F1 Processing Speed	Lden	-0.01	0.012	0.4	2	-0.012	0.011	0.276	3
F1 Speech-in-noise Threshold	PM10	-0.032	0.089	0.716	2	-0.03	0.099	0.765	3
F1 Speech-in-noise Threshold	Lden	0.047	0.092	0.612	2	0.021	0.092	0.818	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis risk	PM10	1.35	0.81	2.42	2	1.35	0.8	2.31	3
CAPE Psychosis risk	Lden	1.06	0.65	1.69	2	1.06	0.66	1.71	3

Exposures: PM10 + Lday

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	PM10	0.169	0.101	0.094	2	0.142	0.117	0.222	3
BL SDQ	Lday	0.056	0.164	0.734	2	0.118	0.183	0.52	3
BL Internalising	PM10	0.032	0.059	0.59	2	0.056	0.068	0.412	3
BL Internalising	Lday	0.054	0.096	0.572	2	0.077	0.107	0.471	3
BL Externalising	PM10	0.137	0.062	0.027	2	0.088	0.071	0.214	3
BL Externalising	Lday	0.001	0.1	0.99	2	0.035	0.111	0.755	3
BL Executive Function	PM10	0.019	0.012	0.106	2	0.028	0.014	0.041	3
BL Executive Function	Lday	-0.002	0.019	0.922	2	0.002	0.021	0.921	3

BL Culture Fair Intelligence Test	PM10	0.003	0.064	0.961	2	0.016	0.075	0.826	3
BL Culture Fair Intelligence Test	Lday	-0.016	0.102	0.878	2	0.082	0.115	0.478	3
BL Processing Speed	PM10	0.001	0.011	0.958	2	-0.014	0.013	0.258	3
BL Processing Speed	Lday	0.001	0.019	0.96	2	0.009	0.021	0.668	3
BL Speech-in-noise Threshold	PM10	0.069	0.062	0.266	2	0.008	0.07	0.913	3
BL Speech-in-noise Threshold	Lday	-0.088	0.099	0.374	2	-0.054	0.112	0.629	3
F1 SDQ	PM10	0.025	0.104	0.812	2	-0.034	0.118	0.774	3
F1 SDQ	Lday	0.126	0.163	0.439	2	0.15	0.184	0.414	3
F1 Internalising	PM10	-0.024	0.062	0.698	2	-0.035	0.071	0.627	3
F1 Internalising	Lday	-0.032	0.097	0.742	2	0.005	0.111	0.967	3
F1 Externalising	PM10	-0.074	0.06	0.219	2	-0.074	0.068	0.281	3
F1 Externalising	Lday	0.168	0.098	0.086	2	0.128	0.11	0.245	3
F1 PHQ-9 Depression	PM10	0.007	0.113	0.948	2	0.032	0.126	0.798	3
F1 PHQ-9 Depression	Lday	0.03	0.19	0.874	2	-0.056	0.205	0.786	3
F1 GAD-7 Anxiety	PM10	0.183	0.096	0.057	2	0.19	0.104	0.07	3
F1 GAD-7 Anxiety	Lday	0.117	0.16	0.465	2	0.11	0.168	0.512	3
F1 Executive Function	PM10	0.027	0.01	0.009	2	0.016	0.012	0.173	3
F1 Executive Function	Lday	-0.007	0.016	0.678	2	-0.001	0.018	0.938	3
F1 Culture Fair Intelligence Test	PM10	0.079	0.059	0.186	2	0.085	0.066	0.203	3
F1 Culture Fair Intelligence Test	Lday	-0.086	0.094	0.36	2	-0.048	0.103	0.641	3

F1 Processing Speed	PM10	0.019	0.011	0.082	2	0.017	0.013	0.192	3
F1 Processing Speed	Lday	0.008	0.019	0.694	2	-0.003	0.022	0.884	3
F1 Speech-in-noise Threshold	PM10	-0.013	0.087	0.877	2	-0.016	0.098	0.869	3
F1 Speech-in-noise Threshold	Lday	-0.112	0.143	0.435	2	-0.083	0.166	0.618	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis risk	PM10	1.36	0.82	2.4	2	1.36	0.8	2.3	3
CAPE Psychosis risk	Lday	1.15	0.42	2.95	2	1.15	0.44	2.98	3

Exposures: NO2+ Ozone

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	NO2	0.017	0.143	0.903	2	0.126	0.158	0.425	3
BL SDQ	Ozone	-0.081	0.154	0.599	2	-0.11	0.163	0.5	3
BL Internalising	NO2	-0.06	0.084	0.472	2	0.013	0.092	0.886	3
BL Internalising	Ozone	0.004	0.09	0.961	2	-0.026	0.095	0.787	3
BL Externalising	NO2	0.078	0.087	0.374	2	0.1	0.096	0.299	3
BL Externalising	Ozone	-0.085	0.094	0.365	2	-0.08	0.099	0.418	3
BL Executive Function	NO2	0.033	0.016	0.045	2	0.034	0.018	0.064	3
BL Executive Function	Ozone	0.004	0.018	0.84	2	0.004	0.019	0.823	3
BL Culture Fair Intelligence Test	NO2	-0.038	0.088	0.67	2	-0.012	0.1	0.904	3
BL Culture Fair Intelligence Test	Ozone	-0.061	0.096	0.524	2	-0.088	0.104	0.398	3
BL Processing Speed	NO2	0.007	0.016	0.681	2	0	0.018	0.98	3

BL Processing Speed	Ozone	0.014	0.018	0.443	2	0.011	0.019	0.539	3
BL Speech-in-noise Threshold	NO2	-0.045	0.088	0.608	2	-0.037	0.096	0.7	3
BL Speech-in-noise Threshold	Ozone	-0.045	0.094	0.628	2	-0.073	0.099	0.458	3
F1 SDQ	NO2	-0.056	0.135	0.68	2	0.016	0.146	0.911	3
F1 SDQ	Ozone	-0.031	0.152	0.836	2	-0.118	0.161	0.464	3
F1 Internalising	NO2	-0.069	0.081	0.392	2	-0.04	0.087	0.646	3
F1 Internalising	Ozone	0.042	0.091	0.647	2	-0.018	0.097	0.852	3
F1 Externalising	NO2	-0.087	0.08	0.274	2	-0.045	0.086	0.605	3
F1 Externalising	Ozone	0.009	0.09	0.916	2	-0.035	0.095	0.709	3
F1 PHQ-9 Depression	NO2	-0.034	0.149	0.819	2	0.003	0.158	0.984	3
F1 PHQ-9 Depression	Ozone	0.099	0.178	0.58	2	0.123	0.184	0.506	3
F1 GAD-7 Anxiety	NO2	0.169	0.126	0.181	2	0.174	0.132	0.187	3
F1 GAD-7 Anxiety	Ozone	0.119	0.151	0.429	2	0.119	0.154	0.442	3
F1 Executive Function	NO2	0.028	0.013	0.033	2	0.023	0.014	0.098	3
F1 Executive Function	Ozone	-0.05	0.015	0.001	2	-0.037	0.015	0.016	3
F1 Culture Fair Intelligence Test	NO2	-0.016	0.077	0.841	2	0.005	0.083	0.949	3
F1 Culture Fair Intelligence Test	Ozone	-0.123	0.088	0.162	2	-0.141	0.092	0.128	3
F1 Processing Speed	NO2	0.019	0.015	0.199	2	0.013	0.016	0.417	3
F1 Processing Speed	Ozone	0.026	0.017	0.122	2	0.023	0.017	0.193	3
F1 Speech-in-noise Threshold	NO2	-0.032	0.115	0.78	2	-0.038	0.123	0.755	3

F1 Speech-in-noise Threshold	Ozone	0.098	0.128	0.444	2	0.072	0.135	0.594	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis risk	NO2	0.96	0.51	1.81	2	0.97	0.51	1.82	3
CAPE Psychosis risk	Ozone	0.9	0.43	1.84	2	0.9	0.43	1.87	3

Exposures: NO2 + Lden

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	NO2	-0.001	0.126	0.994	2	0.105	0.155	0.498	3
BL SDQ	Lden	0.142	0.1	0.156	2	0.115	0.101	0.256	3
BL Internalising	NO2	-0.115	0.074	0.119	2	-0.033	0.089	0.711	3
BL Internalising	Lden	0.123	0.059	0.036	2	0.102	0.059	0.088	3
BL Externalising	NO2	0.114	0.077	0.138	2	0.122	0.093	0.192	3
BL Externalising	Lden	0.019	0.061	0.759	2	0.017	0.062	0.784	3
BL Executive Function	NO2	0.034	0.014	0.016	2	0.034	0.018	0.057	3
BL Executive Function	Lden	-0.008	0.011	0.462	2	-0.004	0.012	0.726	3
BL Culture Fair Intelligence Test	NO2	-0.047	0.078	0.544	2	-0.046	0.102	0.653	3
BL Culture Fair Intelligence Test	Lden	0.096	0.062	0.122	2	0.104	0.063	0.096	3
BL Processing Speed	NO2	-0.005	0.014	0.741	2	-0.008	0.018	0.659	3
BL Processing Speed	Lden	0.011	0.011	0.317	2	0.007	0.011	0.523	3
BL Speech-in-noise Threshold	NO2	-0.029	0.077	0.703	2	-0.009	0.094	0.927	3
BL Speech-in-noise Threshold	Lden	0.019	0.062	0.753	2	-0.002	0.062	0.98	3

F1 SDQ	NO2	-0.081	0.121	0.501	2	0.032	0.142	0.822	3
F1 SDQ	Lden	0.106	0.106	0.317	2	0.053	0.106	0.622	3
F1 Internalising	NO2	-0.111	0.072	0.124	2	-0.048	0.085	0.575	3
F1 Internalising	Lden	0.057	0.063	0.367	2	0.029	0.064	0.648	3
F1 Externalising	NO2	-0.11	0.071	0.122	2	-0.044	0.084	0.603	3
F1 Externalising	Lden	0.049	0.062	0.43	2	0.025	0.063	0.684	3
F1 PHQ-9 Depression	NO2	-0.066	0.138	0.632	2	-0.026	0.152	0.865	3
F1 PHQ-9 Depression	Lden	-0.022	0.118	0.852	2	-0.034	0.12	0.774	3
F1 GAD-7 Anxiety	NO2	0.065	0.117	0.578	2	0.076	0.125	0.544	3
F1 GAD-7 Anxiety	Lden	0.141	0.1	0.156	2	0.133	0.1	0.184	3
F1 Executive Function	NO2	0.059	0.012	0	2	0.042	0.014	0.002	3
F1 Executive Function	Lden	-0.019	0.01	0.063	2	-0.014	0.01	0.157	3
F1 Culture Fair Intelligence Test	NO2	0.057	0.069	0.412	2	0.074	0.079	0.347	3
F1 Culture Fair Intelligence Test	Lden	-0.038	0.06	0.526	2	-0.039	0.06	0.523	3
F1 Processing Speed	NO2	0.009	0.013	0.487	2	0.011	0.016	0.5	3
F1 Processing Speed	Lden	-0.008	0.012	0.515	2	-0.012	0.012	0.303	3
F1 Speech-in-noise Threshold	NO2	-0.108	0.102	0.286	2	-0.083	0.119	0.486	3
F1 Speech-in-noise Threshold	Lden	0.073	0.095	0.442	2	0.038	0.095	0.687	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis risk	NO2	0.92	0.48	1.73	2	0.92	0.49	1.72	3
CAPE Psychosis risk	Lden	1.17	0.72	1.89	2	1.17	0.73	1.89	3

Exposures: NO2 + Lday

Outcome	Term	Estimate	SE	p	Model	Estimate.1	SE.1	p.1	Model.1
BL SDQ	NO2	0.04	0.123	0.747	2	0.134	0.154	0.386	3
BL SDQ	Lday	0.098	0.167	0.555	2	0.12	0.188	0.522	3
BL Internalising	NO2	-0.082	0.072	0.257	2	-0.003	0.09	0.97	3
BL Internalising	Lday	0.096	0.098	0.33	2	0.099	0.11	0.367	3
BL Externalising	NO2	0.122	0.075	0.106	2	0.126	0.093	0.176	3
BL Externalising	Lday	0.003	0.102	0.978	2	0.018	0.114	0.876	3
BL Executive Function	NO2	0.033	0.014	0.02	2	0.032	0.018	0.073	3
BL Executive Function	Lday	-0.008	0.019	0.686	2	0	0.021	0.987	3
BL Culture Fair Intelligence Test	NO2	-0.004	0.076	0.963	2	-0.013	0.101	0.899	3
BL Culture Fair Intelligence Test	Lday	-0.013	0.104	0.898	2	0.093	0.119	0.434	3
BL Processing Speed	NO2	0	0.014	0.982	2	-0.006	0.018	0.758	3
BL Processing Speed	Lday	0.001	0.019	0.948	2	0.006	0.022	0.797	3
BL Speech-in-noise Threshold	NO2	-0.009	0.076	0.907	2	0.005	0.094	0.96	3
BL Speech-in-noise Threshold	Lday	-0.062	0.101	0.542	2	-0.053	0.115	0.645	3
F1 SDQ	NO2	-0.069	0.118	0.558	2	0.025	0.141	0.858	3
F1 SDQ	Lday	0.156	0.167	0.352	2	0.132	0.192	0.492	3
F1 Internalising	NO2	-0.088	0.07	0.208	2	-0.037	0.085	0.664	3
F1 Internalising	Lday	-0.004	0.1	0.97	2	0.012	0.115	0.919	3
F1 Externalising	NO2	-0.124	0.069	0.073	2	-0.064	0.083	0.438	3
F1 Externalising	Lday	0.198	0.1	0.048	2	0.138	0.114	0.224	3
F1 PHQ-9 Depression	NO2	-0.084	0.133	0.53	2	-0.033	0.15	0.827	3

F1 PHQ-9 Depression	Lday	0.059	0.194	0.762	2	-0.035	0.211	0.869	3
F1 GAD-7 Anxiety	NO2	0.103	0.113	0.364	2	0.111	0.123	0.37	3
F1 GAD-7 Anxiety	Lday	0.113	0.164	0.491	2	0.109	0.173	0.528	3
F1 Executive Function	NO2	0.057	0.011	0	2	0.039	0.014	0.005	3
F1 Executive Function	Lday	-0.023	0.016	0.157	2	-0.015	0.018	0.424	3
F1 Culture Fair Intelligence Test	NO2	0.058	0.067	0.387	2	0.071	0.078	0.367	3
F1 Culture Fair Intelligence Test	Lday	-0.093	0.096	0.335	2	-0.059	0.107	0.583	3
F1 Processing Speed	NO2	0.005	0.013	0.671	2	0.006	0.016	0.695	3
F1 Processing Speed	Lday	0.01	0.02	0.627	2	-0.002	0.022	0.938	3
F1 Speech-in-noise Threshold	NO2	-0.067	0.098	0.494	2	-0.053	0.118	0.655	3
F1 Speech-in-noise Threshold	Lday	-0.091	0.146	0.534	2	-0.064	0.171	0.71	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis risk	NO2	0.91	0.47	1.73	2	0.9	0.47	1.74	3
CAPE Psychosis risk	Lday	1.4	0.51	3.78	2	1.42	0.51	3.94	3

Exposures: Lden + Ozone

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	Lden	0.133	0.097	0.172	2	0.121	0.098	0.218	3
BL SDQ	Ozone	-0.045	0.132	0.734	2	-0.11	0.155	0.475	3
BL Internalising	Lden	0.107	0.057	0.062	2	0.095	0.058	0.098	3
BL Internalising	Ozone	0.078	0.077	0.313	2	0.008	0.09	0.927	3
BL Externalising	Lden	0.026	0.06	0.662	2	0.027	0.06	0.657	3

BL Externalising	Ozone	-0.123	0.081	0.128	2	-0.111	0.093	0.235	3
BL Executive Function	Lden	-0.003	0.011	0.811	2	0.002	0.011	0.832	3
BL Executive Function	Ozone	-0.017	0.015	0.247	2	-0.007	0.018	0.69	3
BL Culture Fair Intelligence Test	Lden	0.082	0.061	0.178	2	0.087	0.061	0.152	3
BL Culture Fair Intelligence Test	Ozone	-0.009	0.083	0.915	2	-0.045	0.102	0.66	3
BL Processing Speed	Lden	0.013	0.011	0.246	2	0.008	0.011	0.473	3
BL Processing Speed	Ozone	0.015	0.016	0.35	2	0.015	0.018	0.409	3
BL Speech-in-noise Threshold	Lden	0.009	0.06	0.885	2	-0.013	0.06	0.826	3
BL Speech-in-noise Threshold	Ozone	-0.016	0.08	0.844	2	-0.063	0.094	0.499	3
F1 SDQ	Lden	0.089	0.103	0.391	2	0.042	0.104	0.689	3
F1 SDQ	Ozone	0.033	0.132	0.805	2	-0.108	0.153	0.481	3
F1 Internalising	Lden	0.045	0.062	0.468	2	0.018	0.062	0.771	3
F1 Internalising	Ozone	0.099	0.079	0.211	2	0.006	0.093	0.952	3
F1 Externalising	Lden	0.033	0.061	0.591	2	0.013	0.061	0.832	3
F1 Externalising	Ozone	0.073	0.078	0.351	2	-0.012	0.09	0.897	3
F1 PHQ-9 Depression	Lden	-0.019	0.116	0.868	2	-0.021	0.117	0.86	3
F1 PHQ-9 Depression	Ozone	0.111	0.161	0.49	2	0.113	0.174	0.516	3
F1 GAD-7 Anxiety	Lden	0.176	0.097	0.071	2	0.17	0.098	0.085	3
F1 GAD-7 Anxiety	Ozone	0.086	0.136	0.526	2	0.095	0.144	0.51	3
F1 Executive Function	Lden	-0.016	0.01	0.109	2	-0.012	0.01	0.208	3
F1 Executive Function	Ozone	-0.072	0.013	0	2	-0.052	0.015	0	3

F1 Culture Fair Intelligence Test	Lden	-0.047	0.058	0.417	2	-0.048	0.059	0.416	3
F1 Culture Fair Intelligence Test	Ozone	-0.13	0.077	0.092	2	-0.161	0.086	0.064	3
F1 Processing Speed	Lden	-0.003	0.012	0.815	2	-0.007	0.012	0.53	3
F1 Processing Speed	Ozone	0.014	0.015	0.356	2	0.015	0.017	0.364	3
F1 Speech-in-noise Threshold	Lden	0.068	0.092	0.46	2	0.034	0.092	0.712	3
F1 Speech-in-noise Threshold	Ozone	0.139	0.11	0.206	2	0.101	0.126	0.421	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis risk	Lden	1.1	0.72	1.79	2	1.13	0.72	1.78	3
CAPE Psychosis risk	Ozone	0.97	0.48	1.9	2	0.97	0.49	1.91	3

Exposures: Lden + Lday

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	Lden	0.224	0.148	0.13	2	0.234	0.19	0.219	3
BL SDQ	Lday	-0.182	0.252	0.471	2	-0.206	0.353	0.562	3
BL Internalising	Lden	0.155	0.087	0.075	2	0.189	0.109	0.086	3
BL Internalising	Lday	-0.139	0.148	0.35	2	-0.204	0.202	0.315	3
BL Externalising	Lden	0.069	0.091	0.446	2	0.048	0.115	0.675	3
BL Externalising	Lday	-0.043	0.155	0.779	2	-0.008	0.213	0.969	3
BL Executive Function	Lden	-0.004	0.017	0.829	2	-0.01	0.023	0.655	3
BL Executive Function	Lday	0.009	0.029	0.746	2	0.029	0.043	0.494	3
BL Culture Fair Intelligence Test	Lden	0.23	0.093	0.014	2	0.268	0.132	0.043	3

BL Culture Fair Intelligence Test	Lday	-0.322	0.16	0.044	2	-0.371	0.252	0.142	3
BL Processing Speed	Lden	0.026	0.018	0.144	2	0.018	0.023	0.427	3
BL Processing Speed	Lday	-0.034	0.031	0.265	2	-0.027	0.043	0.534	3
BL Speech-in-noise Threshold	Lden	0.094	0.088	0.289	2	0.076	0.114	0.505	3
BL Speech-in-noise Threshold	Lday	-0.182	0.147	0.216	2	-0.172	0.211	0.417	3
F1 SDQ	Lden	0.05	0.15	0.741	2	-0.024	0.192	0.9	3
F1 SDQ	Lday	0.07	0.244	0.774	2	0.18	0.348	0.607	3
F1 Internalising	Lden	0.094	0.09	0.298	2	0.071	0.116	0.543	3
F1 Internalising	Lday	-0.15	0.146	0.303	2	-0.114	0.212	0.591	3
F1 Externalising	Lden	-0.113	0.087	0.196	2	-0.125	0.111	0.261	3
F1 Externalising	Lday	0.292	0.144	0.042	2	0.304	0.203	0.137	3
F1 PHQ-9 Depression	Lden	-0.131	0.172	0.446	2	-0.059	0.198	0.764	3
F1 PHQ-9 Depression	Lday	0.201	0.292	0.491	2	0.04	0.354	0.91	3
F1 GAD-7 Anxiety	Lden	0.226	0.146	0.121	2	0.241	0.16	0.136	3
F1 GAD-7 Anxiety	Lday	-0.148	0.248	0.55	2	-0.192	0.281	0.498	3
F1 Executive Function	Lden	-0.002	0.014	0.876	2	-0.021	0.019	0.28	3
F1 Executive Function	Lday	0.002	0.024	0.94	2	0.036	0.036	0.313	3
F1 Culture Fair Intelligence Test	Lden	0.026	0.086	0.761	2	-0.022	0.105	0.832	3
F1 Culture Fair Intelligence Test	Lday	-0.105	0.143	0.461	2	0.005	0.188	0.979	3
F1 Processing Speed	Lden	-0.022	0.017	0.184	2	-0.035	0.021	0.094	3
F1 Processing Speed	Lday	0.039	0.028	0.171	2	0.058	0.04	0.152	3

F1 Speech-in-noise Threshold	Lden	0.159	0.121	0.188	2	0.144	0.147	0.33	3
F1 Speech-in-noise Threshold	Lday	-0.285	0.192	0.139	2	-0.295	0.268	0.275	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis risk	Lden	1.08	0.49	2.5	2	1.05	0.41	2.69	3
CAPE Psychosis risk	Lday	1.15	0.21	5.66	2	1.21	0.18	8.34	3

Exposures: Lday + Ozone

Outcome	**Term**	**Estimate**	**SE**	**p**	**Model**	**Estimate.1**	**SE.1**	**p.1**	**Model.1**
BL SDQ	Lday	0.092	0.164	0.574	2	0.138	0.182	0.451	3
BL SDQ	Ozone	-0.075	0.13	0.564	2	-0.132	0.154	0.392	3
BL Internalising	Lday	0.08	0.097	0.406	2	0.095	0.107	0.375	3
BL Internalising	Ozone	0.055	0.077	0.475	2	-0.014	0.09	0.879	3
BL Externalising	Lday	0.012	0.101	0.905	2	0.037	0.111	0.738	3
BL Externalising	Ozone	-0.13	0.08	0.104	2	-0.114	0.093	0.221	3
BL Executive Function	Lday	0	0.019	0.996	2	0.011	0.021	0.605	3
BL Executive Function	Ozone	-0.017	0.015	0.268	2	-0.006	0.018	0.739	3
BL Culture Fair Intelligence Test	Lday	-0.027	0.103	0.794	2	0.069	0.115	0.551	3
BL Culture Fair Intelligence Test	Ozone	-0.043	0.082	0.598	2	-0.069	0.101	0.497	3
BL Processing Speed	Lday	0.004	0.019	0.844	2	0.007	0.021	0.739	3
BL Processing Speed	Ozone	0.01	0.015	0.5	2	0.013	0.018	0.471	3
BL Speech-in-noise Threshold	Lday	-0.073	0.099	0.463	2	-0.069	0.111	0.534	3

BL Speech-in-noise Threshold	Ozone	-0.031	0.08	0.698	2	-0.071	0.093	0.447	3
F1 SDQ	Lday	0.138	0.166	0.405	2	0.113	0.188	0.548	3
F1 SDQ	Ozone	0.027	0.131	0.835	2	-0.103	0.153	0.502	3
F1 Internalising	Lday	-0.013	0.099	0.896	2	-0.004	0.113	0.97	3
F1 Internalising	Ozone	0.081	0.078	0.299	2	-0.003	0.092	0.975	3
F1 Externalising	Lday	0.18	0.099	0.07	2	0.112	0.112	0.315	3
F1 Externalising	Ozone	0.091	0.077	0.237	2	0.005	0.09	0.954	3
F1 PHQ-9 Depression	Lday	0.068	0.195	0.727	2	-0.013	0.208	0.949	3
F1 PHQ-9 Depression	Ozone	0.131	0.159	0.41	2	0.119	0.173	0.493	3
F1 GAD-7 Anxiety	Lday	0.16	0.164	0.33	2	0.165	0.171	0.335	3
F1 GAD-7 Anxiety	Ozone	0.05	0.135	0.71	2	0.062	0.143	0.663	3
F1 Executive Function	Lday	-0.02	0.016	0.21	2	-0.012	0.018	0.5	3
F1 Executive Function	Ozone	-0.07	0.013	0	2	-0.05	0.015	0.001	3
F1 Culture Fair Intelligence Test	Lday	-0.107	0.095	0.263	2	-0.073	0.105	0.488	3
F1 Culture Fair Intelligence Test	Ozone	-0.131	0.076	0.084	2	-0.156	0.086	0.07	3
F1 Processing Speed	Lday	0.016	0.02	0.428	2	0.006	0.022	0.779	3
F1 Processing Speed	Ozone	0.017	0.014	0.253	2	0.019	0.017	0.262	3
F1 Speech-in-noise Threshold	Lday	-0.086	0.145	0.555	2	-0.064	0.167	0.703	3
F1 Speech-in-noise Threshold	Ozone	0.105	0.109	0.333	2	0.079	0.125	0.53	3
		OR	**LL**	**UL**		**OR**	**LL**	**UL**	
CAPE Psychosis risk	Lday	1.29	0.51	3.23	2	1.31	0.51	3.31	3

CAPE psychosis risk	Ozone	0.96	0.48	1.88	2	0.95	0.48	1.9	3

XI: Associations between exposure to evening noise (Leve) and night-time noise (Lnight) and psychological outcomes in the SCAMP cohort

Outcome	Model	Leve Estimate	SE	p	Lnight Estimate	SE	p
BL SDQ	1	0.041	0.015	0.007	0.04	0.016	0.013
BL SDQ	2	0.026	0.015	0.081	0.025	0.016	0.125
BL SDQ	3	0.026	0.015	0.086	0.024	0.016	0.131
BL Internalising	1	0.023	0.009	0.01	0.023	0.009	0.014
BL Internalising	2	0.016	0.009	0.066	0.016	0.009	0.082
BL Internalising	3	0.017	0.009	0.056	0.017	0.01	0.072
BL Externalising	1	0.018	0.009	0.053	0.017	0.01	0.088
BL Externalising	2	0.01	0.009	0.281	0.008	0.01	0.405
BL Externalising	3	0.009	0.009	0.334	0.007	0.01	0.465
BL Executive Function	1	-0.005	0.001	0.001	-0.005	0.002	0
BL Executive Function	2	0	0.002	0.921	0	0.002	0.917
BL Executive Function	3	0	0.002	0.799	0.001	0.002	0.77
BL Culture Fair Intelligence Test	1	-0.011	0.008	0.168	-0.011	0.008	0.165
BL Culture Fair Intelligence Test	2	0.012	0.009	0.204	0.013	0.01	0.204
BL Culture Fair Intelligence Test	3	0.012	0.009	0.216	0.013	0.01	0.2
BL Processing Speed	1	0.003	0.001	0.027	0.003	0.001	0.031
BL Processing Speed	2	0.002	0.002	0.346	0.002	0.002	0.402
BL Processing Speed	3	0.001	0.002	0.601	0.001	0.002	0.68
BL Speech-in-noise Threshold	1	0.003	0.001	0.027	0.003	0.001	0.031
BL Speech-in-noise Threshold	2	0.002	0.002	0.346	0.002	0.002	0.402
BL Speech-in-noise Threshold	3	0.001	0.002	0.601	0.001	0.002	0.68
F1 SDQ	1	0.013	0.017	0.426	0.011	0.018	0.526
F1 SDQ	2	0.01	0.016	0.517	0.009	0.017	0.583
F1 SDQ	3	0.008	0.016	0.635	0.006	0.017	0.721
F1 Internalising	1	0.002	0.01	0.876	0	0.011	0.99
F1 Internalising	2	0.002	0.009	0.859	0.001	0.01	0.897
F1 Internalising	3	0.001	0.01	0.911	0	0.01	0.974
F1 Externalising	1	0.012	0.011	0.271	0.011	0.011	0.325
F1 Externalising	2	0.001	0.009	0.913	0.001	0.01	0.951
F1 Externalising	3	0.001	0.009	0.911	0.001	0.01	0.95
F1 PHQ-9 Depression	1	0.003	0.018	0.849	0.002	0.019	0.92

F1 PHQ-9 Depression	2	-0.007	0.017	0.687	-0.009	0.019	0.646
F1 PHQ-9 Depression	3	-0.007	0.018	0.685	-0.009	0.019	0.648
F1 GAD-7 Anxiety	1	0.031	0.015	0.038	0.031	0.016	0.059
F1 GAD-7 Anxiety	2	0.028	0.015	0.062	0.027	0.016	0.09
F1 GAD-7 Anxiety	3	0.027	0.015	0.074	0.026	0.016	0.107
F1 Executive Function	1	-0.002	0.002	0.164	-0.003	0.002	0.176
F1 Executive Function	2	0	0.002	0.969	0	0.002	0.996
F1 Executive Function	3	-0.001	0.002	0.725	0	0.002	0.78
F1 Culture Fair Intelligence Test	1	-0.014	0.01	0.174	-0.013	0.011	0.245
F1 Culture Fair Intelligence Test	2	-0.005	0.009	0.61	-0.003	0.01	0.75
F1 Culture Fair Intelligence Test	3	-0.004	0.009	0.643	-0.003	0.01	0.785
F1 Processing Speed	1	0.001	0.002	0.721	0.001	0.002	0.766
F1 Processing Speed	2	-0.001	0.002	0.705	-0.001	0.002	0.639
F1 Processing Speed	3	-0.001	0.002	0.439	-0.002	0.002	0.382
F1 Speech-in-noise Threshold	1	0.001	0.002	0.721	0.001	0.002	0.766
F1 Speech-in-noise Threshold	2	-0.001	0.002	0.705	-0.001	0.002	0.639
F1 Speech-in-noise Threshold	3	-0.001	0.002	0.439	-0.002	0.002	0.382
F2 Psychosis risk	1	0.06	0.14	0.64	0.07	0.14	0.60
F2 Psychosis risk	2	0.06	0.14	0.67	0.07	0.15	0.63
F2 Psychosis risk	3	0.06	0.15	0.67	0.07	0.15	0.63

XII: Associations between exposure to air pollution or noise and psychological outcomes in the SCAMP cohort: Interaction effects

Exposure: PM2.5

Outcome	Interaction	Estimate	SE	p	Model	Estimate	SE	p	Model
BL SDQ	exposure*IQ	-0.128	0.022	0	2	-0.119	0.022	0	3
BL Internalising	exposure*IQ	-0.07	0.013	0	2	-0.065	0.013	0	3
BL Externalising	exposure*IQ	-0.058	0.013	0	2	-0.055	0.014	0	3
BL Executive Function	exposure*IQ	0.044	0.002	0	2	0.043	0.003	0	3
BL Culture Fair Intelligence Test	exposure*IQ	0.613	0.008	0	2	0.607	0.008	0	3
BL Processing Speed	exposure*IQ	0	0.003	0.882	2	0	0.003	0.911	3
BL Speech-in-noise Threshold	exposure*IQ	-0.063	0.014	0	2	-0.062	0.014	0	3
F1 SDQ	exposure*IQ	-0.044	0.022	0.045	2	-0.041	0.022	0.063	3
F1 Internalising	exposure*IQ	-0.008	0.013	0.54	2	-0.002	0.013	0.851	3
F1 Externalising	exposure*IQ	-0.005	0.013	0.707	2	-0.007	0.013	0.564	3
F1 PHQ-9 Depression	exposure*IQ	0.024	0.024	0.309	2	0.028	0.024	0.239	3

F1 GAD-7 Anxiety	exposure*IQ	0.018	0.02	0.374	2	0.019	0.02	0.358	3
F1 Executive Function	exposure*IQ	0.009	0.002	0	2	0.009	0.002	0	3
F1 Culture Fair Intelligence Test	exposure*IQ	0.005	0.02	0.784	2	0.01	0.02	0.626	3
F1 Processing Speed	exposure*IQ	0.003	0.002	0.153	2	0.004	0.002	0.131	3
F1 Speech-in-noise Threshold	exposure*IQ	-0.05	0.02	0.012	2	-0.054	0.02	0.006	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*sex	-0.525	0.216	0.015	2	-0.444	0.244	0.07	3
BL Internalising	exposure*sex	-0.288	0.127	0.023	2	-0.225	0.142	0.114	3
BL Externalising	exposure*sex	-0.237	0.132	0.074	2	-0.229	0.149	0.126	3
BL Executive Function	exposure*sex	0.072	0.024	0.003	2	0.054	0.028	0.054	3
BL Culture Fair Intelligence Test	exposure*sex	0.253	0.131	0.054	2	0.125	0.153	0.412	3
BL Processing Speed	exposure*sex	-0.039	0.025	0.121	2	-0.006	0.028	0.821	3
BL Speech-in-noise Threshold	exposure*sex	-0.155	0.143	0.278	2	0.119	0.159	0.455	3
F1 SDQ	exposure*sex	-0.18	0.229	0.431	2	-0.056	0.239	0.815	3

F1 Internalising	exposure*sex	0.118	0.137	0.389	2	0.252	0.143	0.079	3
F1 Externalising	exposure*sex	-0.199	0.133	0.134	2	-0.194	0.138	0.159	3
F1 PHQ-9 Depression	exposure*sex	0.436	0.25	0.081	2	0.457	0.257	0.075	3
F1 GAD-7 Anxiety	exposure*sex	0.209	0.212	0.325	2	0.214	0.217	0.324	3
F1 Executive Function	exposure*sex	0.02	0.023	0.379	2	0	0.024	0.999	3
F1 Culture Fair Intelligence Test	exposure*sex	-0.043	0.132	0.744	2	-0.046	0.137	0.735	3
F1 Processing Speed	exposure*sex	0.011	0.024	0.64	2	0.007	0.025	0.793	3
F1 Speech-in-noise Threshold	exposure*sex	-0.275	0.197	0.163	2	-0.219	0.203	0.28	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*ethnicity2	0.022	0.334	0.948	2	0.126	0.343	0.713	3
BL SDQ	exposure*ethnicity3	0.13	0.251	0.603	2	0.323	0.272	0.236	3
BL SDQ	exposure*ethnicity4	0.293	0.389	0.452	2	0.354	0.391	0.365	3
BL SDQ	exposure*ethnicity5	-1.697	1.521	0.265	2	-1.621	1.521	0.287	3
BL Internalising	exposure*ethnicity2	0.103	0.197	0.599	2	0.158	0.202	0.434	3
BL Internalising	exposure*ethnicity3	0.134	0.147	0.365	2	0.208	0.16	0.193	3

BL Internalising	exposure*ethnicity4	0.238	0.229	0.298	2	0.278	0.23	0.226	3
BL Internalising	exposure*ethnicity5	0.148	0.894	0.868	2	0.232	0.896	0.795	3
BL Externalising	exposure*ethnicity2	-0.082	0.205	0.691	2	-0.034	0.21	0.871	3
BL Externalising	exposure*ethnicity3	-0.003	0.154	0.984	2	0.102	0.166	0.542	3
BL Externalising	exposure*ethnicity4	0.055	0.238	0.818	2	0.074	0.239	0.755	3
BL Externalising	exposure*ethnicity5	-1.845	0.932	0.048	2	-1.858	0.932	0.046	3
BL Executive Function	exposure*ethnicity2	-0.033	0.037	0.382	2	-0.006	0.038	0.872	3
BL Executive Function	exposure*ethnicity3	0.011	0.029	0.695	2	0.018	0.031	0.561	3
BL Executive Function	exposure*ethnicity4	-0.04	0.042	0.346	2	-0.039	0.042	0.357	3
BL Executive Function	exposure*ethnicity5	-0.322	0.183	0.079	2	-0.278	0.182	0.126	3
BL Culture Fair Intelligence Test	exposure*ethnicity2	0.126	0.203	0.536	2	0.282	0.207	0.174	3
BL Culture Fair Intelligence Test	exposure*ethnicity3	-0.089	0.155	0.564	2	-0.007	0.17	0.967	3
BL Culture Fair	exposure*ethnicity4	-0.035	0.234	0.881	2	0.001	0.233	0.997	3

Intelligence Test									
BL Culture Fair Intelligence Test	exposure*ethnicity5	-0.551	0.97	0.57	2	-0.483	0.96	0.615	3
BL Processing Speed	exposure*ethnicity2	0.066	0.038	0.084	2	0.069	0.038	0.071	3
BL Processing Speed	exposure*ethnicity3	0.019	0.028	0.499	2	0.017	0.03	0.574	3
BL Processing Speed	exposure*ethnicity4	0.015	0.046	0.739	2	0.024	0.045	0.593	3
BL Processing Speed	exposure*ethnicity5	-0.073	0.133	0.582	2	-0.091	0.13	0.485	3
BL Speech-in-noise Threshold	exposure*ethnicity2	-0.248	0.217	0.253	2	-0.305	0.221	0.168	3
BL Speech-in-noise Threshold	exposure*ethnicity3	-0.151	0.163	0.354	2	-0.042	0.175	0.812	3
BL Speech-in-noise Threshold	exposure*ethnicity4	-0.138	0.25	0.581	2	-0.145	0.25	0.563	3
BL Speech-in-noise Threshold	exposure*ethnicity5	0.481	1.022	0.638	2	0.296	1.02	0.771	3
F1 SDQ	exposure*ethnicity2	0.125	0.391	0.749	2	0.069	0.401	0.863	3
F1 SDQ	exposure*ethnicity3	0.116	0.273	0.671	2	-0.01	0.29	0.972	3

F1 SDQ	exposure*ethnicity4	-0.125	0.418	0.764	2	-0.177	0.418	0.672	3
F1 SDQ	exposure*ethnicity5	0.301	1.192	0.8	2	-0.235	1.225	0.848	3
F1 Internalising	exposure*ethnicity2	0.116	0.234	0.62	2	0.059	0.24	0.805	3
F1 Internalising	exposure*ethnicity3	0.123	0.163	0.451	2	0.064	0.174	0.713	3
F1 Internalising	exposure*ethnicity4	-0.264	0.25	0.291	2	-0.295	0.251	0.239	3
F1 Internalising	exposure*ethnicity5	-0.418	0.713	0.558	2	-0.686	0.733	0.35	3
F1 Externalising	exposure*ethnicity2	-0.16	0.232	0.491	2	-0.202	0.238	0.396	3
F1 Externalising	exposure*ethnicity3	0.052	0.158	0.743	2	-0.083	0.169	0.624	3
F1 Externalising	exposure*ethnicity4	0.146	0.242	0.546	2	0.097	0.242	0.69	3
F1 Externalising	exposure*ethnicity5	1.244	0.74	0.093	2	0.836	0.762	0.272	3
F1 PHQ-9 Depression	exposure*ethnicity2	0.078	0.43	0.857	2	0.036	0.441	0.936	3
F1 PHQ-9 Depression	exposure*ethnicity3	0.163	0.304	0.593	2	0.138	0.317	0.664	3
F1 PHQ-9 Depression	exposure*ethnicity4	0.086	0.462	0.852	2	0.053	0.464	0.909	3
F1 PHQ-9 Depression	exposure*ethnicity5	-0.384	1.316	0.77	2	-0.514	1.36	0.706	3
F1 GAD-7 Anxiety	exposure*ethnicity2	-0.377	0.365	0.301	2	-0.379	0.373	0.31	3
F1 GAD-7 Anxiety	exposure*ethnicity3	-0.2	0.259	0.44	2	-0.19	0.268	0.48	3
F1 GAD-7 Anxiety	exposure*ethnicity4	0	0.392	0.999	2	-0.005	0.393	0.99	3

F1 GAD-7 Anxiety	exposure*ethnicity5	0.803	1.123	0.475	2	0.794	1.157	0.493	3
F1 Executive Function	exposure*ethnicity2	-0.058	0.038	0.128	2	-0.047	0.039	0.225	3
F1 Executive Function	exposure*ethnicity3	-0.058	0.027	0.031	2	-0.049	0.029	0.087	3
F1 Executive Function	exposure*ethnicity4	-0.126	0.04	0.002	2	-0.112	0.04	0.005	3
F1 Executive Function	exposure*ethnicity5	-0.23	0.135	0.089	2	-0.208	0.134	0.121	3
F1 Culture Fair Intelligence Test	exposure*ethnicity2	-0.511	0.222	0.022	2	-0.475	0.228	0.038	3
F1 Culture Fair Intelligence Test	exposure*ethnicity3	-0.394	0.157	0.012	2	-0.354	0.166	0.033	3
F1 Culture Fair Intelligence Test	exposure*ethnicity4	-0.48	0.239	0.044	2	-0.433	0.24	0.071	3
F1 Culture Fair Intelligence Test	exposure*ethnicity5	-2.428	0.735	0.001	2	-2.46	0.755	0.001	3
F1 Processing Speed	exposure*ethnicity2	0.034	0.045	0.45	2	-0.02	0.045	0.662	3
F1 Processing Speed	exposure*ethnicity3	0.056	0.029	0.051	2	0.023	0.031	0.459	3
F1 Processing Speed	exposure*ethnicity4	0.067	0.049	0.173	2	0.036	0.049	0.468	3

F1 Processing Speed	exposure*ethnicity5	-0.116	0.123	0.347	2	-0.164	0.121	0.175	3
F1 Speech-in-noise Threshold	exposure*ethnicity2	-0.187	0.361	0.605	2	-0.323	0.364	0.376	3
F1 Speech-in-noise Threshold	exposure*ethnicity3	0.187	0.228	0.414	2	0.096	0.24	0.689	3
F1 Speech-in-noise Threshold	exposure*ethnicity4	0.044	0.358	0.903	2	0.088	0.356	0.804	3
F1 Speech-in-noise Threshold	exposure*ethnicity5	1.521	1.08	0.159	2	1.429	1.073	0.183	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*religion	-0.06	0.03	0.05	2	-0.062	0.031	0.043	3
BL Internalising	exposure*religion	-0.041	0.018	0.024	2	-0.04	0.018	0.028	3
BL Externalising	exposure*religion	-0.019	0.019	0.308	2	-0.022	0.019	0.246	3
BL Executive Function	exposure*religion	-0.004	0.004	0.209	2	-0.006	0.004	0.107	3
BL Culture Fair Intelligence Test	exposure*religion	-0.038	0.019	0.053	2	-0.036	0.02	0.069	3
BL Processing Speed	exposure*religion	0.005	0.003	0.165	2	0.004	0.003	0.231	3
BL Speech-in-noise Threshold	exposure*religion	0.019	0.018	0.31	2	0.02	0.019	0.28	3

F1 SDQ	exposure*religion	-0.088	0.029	0.003	2	-0.086	0.029	0.003	3
F1 Internalising	exposure*religion	-0.058	0.018	0.001	2	-0.056	0.018	0.002	3
F1 Externalising	exposure*religion	-0.027	0.017	0.116	2	-0.026	0.017	0.137	3
F1 PHQ-9 Depression	exposure*religion	-0.108	0.032	0.001	2	-0.11	0.032	0.001	3
F1 GAD-7 Anxiety	exposure*religion	-0.091	0.028	0.001	2	-0.094	0.028	0.001	3
F1 Executive Function	exposure*religion	0	0.003	0.976	2	-0.001	0.003	0.777	3
F1 Culture Fair Intelligence Test	exposure*religion	-0.028	0.018	0.11	2	-0.027	0.018	0.13	3
F1 Processing Speed	exposure*religion	0.004	0.003	0.142	2	0.004	0.003	0.188	3
F1 Speech-in-noise Threshold	exposure*religion	0.012	0.023	0.591	2	0.015	0.023	0.508	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure* BL Lden (high/low)	0.046	0.021	0.028	2	0.042	0.021	0.051	3
BL Internalising	exposure* BL Lden (high/low)	0.017	0.012	0.169	2	0.015	0.013	0.236	3
BL Externalising	exposure* BL Lden (high/low)	0.029	0.013	0.024	2	0.027	0.013	0.041	3
BL Executive Function	exposure* BL Lden (high/low)	-0.002	0.002	0.433	2	-0.001	0.002	0.612	3

BL Culture Fair Intelligence Test	exposure* BL Lden (high/low)	0.016	0.013	0.232	2	0.018	0.013	0.187	3
BL Processing Speed	exposure* BL Lden (high/low)	0.003	0.002	0.179	2	0.002	0.002	0.395	3
BL Speech-in-noise Threshold	exposure* BL Lden (high/low)	0.012	0.013	0.372	2	0.006	0.013	0.667	3
F1 SDQ	exposure: F1 Lden (high/low)	0.016	0.021	0.455	2	0.012	0.021	0.562	3
F1 Internalising	exposure: F1 Lden (high/low)	0.004	0.013	0.77	2	0.002	0.013	0.876	3
F1 Externalising	exposure: F1 Lden (high/low)	0.007	0.012	0.577	2	0.005	0.012	0.712	3
F1 PHQ-9 Depression	exposure: F1 Lden (high/low)	-0.002	0.023	0.925	2	-0.006	0.023	0.813	3
F1 GAD-7 Anxiety	exposure: F1 Lden (high/low)	0.015	0.02	0.444	2	0.014	0.02	0.466	3
F1 Executive Function	exposure: F1 Lden (high/low)	-0.003	0.002	0.131	2	-0.003	0.002	0.165	3
F1 Culture Fair Intelligence Test	exposure: F1 Lden (high/low)	-0.018	0.012	0.131	2	-0.018	0.012	0.135	3
F1 Processing Speed	exposure: F1 Lden (high/low)	-0.002	0.002	0.415	2	-0.002	0.002	0.323	3

F1 Speech-in-noise Threshold	exposure: F1 Lden (high/low)	0.029	0.018	0.115	2	0.025	0.018	0.177	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*School Type (Independent yes/no)	-0.002	0.244	0.992	2	0.165	0.294	0.574	3
BL Internalising	exposure*School Type (Independent yes/no)	-0.006	0.143	0.969	2	0.061	0.17	0.721	3
BL Externalising	exposure*School Type (Independent yes/no)	0.003	0.149	0.983	2	0.091	0.179	0.611	3
BL Executive Function	exposure*School Type (Independent yes/no)	0.088	0.028	0.002	2	0.022	0.034	0.529	3
BL Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.365	0.153	0.017	2	0.01	0.196	0.959	3
BL Processing Speed	exposure*School Type (Independent yes/no)	-0.051	0.027	0.057	2	-0.036	0.033	0.271	3
BL Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	0.206	0.15	0.17	2	0.291	0.18	0.108	3
F1 SDQ	exposure*School Type	-0.323	0.273	0.236	2	-0.035	0.322	0.913	3

	(Independent yes/no)								
F1 Internalising	exposure*School Type (Independent yes/no)	-0.271	0.163	0.097	2	-0.117	0.195	0.547	3
F1 Externalising	exposure*School Type (Independent yes/no)	-0.098	0.159	0.539	2	0.068	0.188	0.719	3
F1 PHQ-9 Depression	exposure*School Type (Independent yes/no)	0.061	0.327	0.852	2	0.104	0.356	0.77	3
F1 GAD-7 Anxiety	exposure*School Type (Independent yes/no)	0.411	0.278	0.14	2	0.431	0.295	0.146	3
F1 Executive Function	exposure*School Type (Independent yes/no)	0.029	0.026	0.269	2	-0.002	0.031	0.956	3
F1 Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.149	0.16	0.351	2	0.054	0.182	0.765	3
F1 Processing Speed	exposure*School Type (Independent yes/no)	-0.035	0.029	0.217	2	0.016	0.035	0.652	3
F1 Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	-0.212	0.211	0.315	2	0	0.255	0.999	3

Exposure: PM10

Outcome	Interaction	Estimate	SE	p	Model	Estimate	SE	p	Model
BL SDQ	exposure*IQ	-0.087	0.015	0	2	-0.082	0.015	0	3
BL Internalising	exposure*IQ	-0.048	0.009	0	2	-0.044	0.009	0	3
BL Externalising	exposure*IQ	-0.04	0.009	0	2	-0.038	0.009	0	3
BL Executive Function	exposure*IQ	0.029	0.002	0	2	0.029	0.002	0	3
BL Culture Fair Intelligence Test	exposure*IQ	0.413	0.006	0	2	0.409	0.006	0	3
BL Processing Speed	exposure*IQ	0	0.002	0.907	2	0	0.002	0.937	3
BL Speech-in-noise Threshold	exposure*IQ	-0.042	0.009	0	2	-0.042	0.009	0	3
F1 SDQ	exposure*IQ	-0.031	0.015	0.045	2	-0.028	0.015	0.063	3
F1 Internalising	exposure*IQ	-0.005	0.009	0.562	2	-0.002	0.009	0.866	3
F1 Externalising	exposure*IQ	-0.003	0.009	0.715	2	-0.005	0.009	0.565	3
F1 PHQ-9 Depression	exposure*IQ	0.017	0.017	0.315	2	0.019	0.017	0.251	3
F1 GAD-7 Anxiety	exposure*IQ	0.011	0.014	0.423	2	0.012	0.014	0.401	3
F1 Executive Function	exposure*IQ	0.006	0.002	0	2	0.006	0.002	0	3
F1 Culture Fair	exposure*IQ	0.003	0.014	0.807	2	0.006	0.014	0.654	3

Intelligence Test									
F1 Processing Speed	exposure*IQ	0.002	0.002	0.166	2	0.003	0.002	0.13	3
F1 Speech-in-noise Threshold	exposure*IQ	-0.034	0.014	0.013	2	-0.037	0.014	0.006	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*sex	-0.032	0.195	0.87	2	-0.043	0.2	0.831	3
BL Internalising	exposure*sex	-0.007	0.115	0.951	2	0.015	0.118	0.899	3
BL Externalising	exposure*sex	-0.025	0.12	0.835	2	-0.056	0.122	0.648	3
BL Executive Function	exposure*sex	0.076	0.023	0.001	2	0.052	0.024	0.026	3
BL Culture Fair Intelligence Test	exposure*sex	0.332	0.122	0.007	2	0.191	0.126	0.129	3
BL Processing Speed	exposure*sex	-0.014	0.022	0.517	2	0.002	0.022	0.931	3
BL Speech-in-noise Threshold	exposure*sex	0.091	0.12	0.45	2	0.148	0.122	0.228	3
F1 SDQ	exposure*sex	-0.143	0.201	0.476	2	-0.067	0.206	0.744	3
F1 Internalising	exposure*sex	0.142	0.12	0.239	2	0.22	0.123	0.074	3
F1 Externalising	exposure*sex	-0.215	0.116	0.064	2	-0.203	0.119	0.088	3
F1 PHQ-9 Depression	exposure*sex	0.425	0.219	0.052	2	0.44	0.222	0.048	3

F1 GAD-7 Anxiety	exposure*sex	0.276	0.186	0.138	2	0.269	0.188	0.152	3
F1 Executive Function	exposure*sex	0.015	0.02	0.457	2	0.003	0.021	0.881	3
F1 Culture Fair Intelligence Test	exposure*sex	-0.057	0.115	0.622	2	-0.078	0.118	0.508	3
F1 Processing Speed	exposure*sex	-0.002	0.021	0.941	2	0	0.021	0.987	3
F1 Speech-in-noise Threshold	exposure*sex	-0.124	0.171	0.47	2	-0.108	0.174	0.537	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*ethnicity2	0.388	0.302	0.199	2	0.386	0.308	0.21	3
BL SDQ	exposure*ethnicity3	0.274	0.228	0.231	2	0.277	0.239	0.246	3
BL SDQ	exposure*ethnicity4	0.664	0.342	0.052	2	0.601	0.342	0.079	3
BL SDQ	exposure*ethnicity5	-0.241	1.213	0.843	2	-0.094	1.228	0.939	3
BL Internalising	exposure*ethnicity2	0.297	0.178	0.095	2	0.279	0.181	0.124	3
BL Internalising	exposure*ethnicity3	0.28	0.134	0.037	2	0.263	0.14	0.061	3
BL Internalising	exposure*ethnicity4	0.44	0.201	0.029	2	0.391	0.202	0.053	3
BL Internalising	exposure*ethnicity5	-0.172	0.713	0.809	2	-0.083	0.723	0.909	3
BL Externalising	exposure*ethnicity2	0.091	0.185	0.622	2	0.104	0.189	0.58	3

BL Externalising	exposure*ethnicity3	-0.007	0.14	0.962	2	0.012	0.146	0.937	3
BL Externalising	exposure*ethnicity4	0.224	0.21	0.286	2	0.212	0.21	0.313	3
BL Externalising	exposure*ethnicity5	-0.069	0.744	0.926	2	0.001	0.752	0.998	3
BL Executive Function	exposure*ethnicity2	-0.059	0.036	0.1	2	-0.028	0.036	0.431	3
BL Executive Function	exposure*ethnicity3	-0.042	0.027	0.121	2	-0.021	0.028	0.444	3
BL Executive Function	exposure*ethnicity4	-0.064	0.039	0.1	2	-0.055	0.039	0.154	3
BL Executive Function	exposure*ethnicity5	-0.339	0.161	0.035	2	-0.275	0.159	0.085	3
BL Culture Fair Intelligence Test	exposure*ethnicity2	-0.056	0.189	0.769	2	0.016	0.191	0.934	3
BL Culture Fair Intelligence Test	exposure*ethnicity3	0.089	0.144	0.535	2	0.129	0.15	0.391	3
BL Culture Fair Intelligence Test	exposure*ethnicity4	-0.115	0.215	0.592	2	-0.08	0.213	0.706	3
BL Culture Fair Intelligence Test	exposure*ethnicity5	-0.226	0.775	0.77	2	-0.201	0.776	0.796	3
BL Processing Speed	exposure*ethnicity2	0.049	0.033	0.14	2	0.039	0.033	0.24	3

BL Processing Speed	exposure*ethnicity3	0.055	0.025	0.03	2	0.037	0.026	0.154	3
BL Processing Speed	exposure*ethnicity4	0.051	0.036	0.158	2	0.046	0.036	0.203	3
BL Processing Speed	exposure*ethnicity5	-0.049	0.123	0.693	2	-0.1	0.121	0.407	3
BL Speech-in-noise Threshold	exposure*ethnicity2	-0.163	0.185	0.379	2	-0.228	0.187	0.223	3
BL Speech-in-noise Threshold	exposure*ethnicity3	0.203	0.139	0.144	2	0.157	0.145	0.277	3
BL Speech-in-noise Threshold	exposure*ethnicity4	0.165	0.211	0.435	2	0.123	0.211	0.559	3
BL Speech-in-noise Threshold	exposure*ethnicity5	1.362	0.916	0.137	2	1.191	0.921	0.196	3
F1 SDQ	exposure*ethnicity2	-0.091	0.339	0.787	2	-0.185	0.345	0.592	3
F1 SDQ	exposure*ethnicity3	0.178	0.232	0.442	2	0.027	0.241	0.912	3
F1 SDQ	exposure*ethnicity4	-0.006	0.343	0.987	2	-0.062	0.342	0.857	3
F1 SDQ	exposure*ethnicity5	0.312	1.15	0.786	2	-0.208	1.175	0.859	3
F1 Internalising	exposure*ethnicity2	0.034	0.203	0.867	2	-0.031	0.207	0.882	3
F1 Internalising	exposure*ethnicity3	0.183	0.139	0.188	2	0.103	0.145	0.476	3
F1 Internalising	exposure*ethnicity4	-0.084	0.205	0.683	2	-0.12	0.205	0.557	3

F1 Internalising	exposure*ethnicity5	-0.555	0.688	0.42	2	-0.784	0.703	0.265	3
F1 Externalising	exposure*ethnicity2	-0.246	0.198	0.212	2	-0.294	0.201	0.143	3
F1 Externalising	exposure*ethnicity3	0.015	0.134	0.91	2	-0.08	0.14	0.568	3
F1 Externalising	exposure*ethnicity4	0.036	0.197	0.856	2	-0.003	0.196	0.987	3
F1 Externalising	exposure*ethnicity5	1.17	0.69	0.09	2	0.807	0.704	0.252	3
F1 PHQ-9 Depression	exposure*ethnicity2	-0.041	0.375	0.912	2	-0.097	0.383	0.799	3
F1 PHQ-9 Depression	exposure*ethnicity3	0.121	0.256	0.638	2	0.124	0.264	0.638	3
F1 PHQ-9 Depression	exposure*ethnicity4	0.116	0.372	0.754	2	0.092	0.373	0.805	3
F1 PHQ-9 Depression	exposure*ethnicity5	-0.223	1.231	0.856	2	-0.255	1.264	0.84	3
F1 GAD-7 Anxiety	exposure*ethnicity2	-0.47	0.318	0.14	2	-0.478	0.323	0.14	3
F1 GAD-7 Anxiety	exposure*ethnicity3	-0.146	0.218	0.503	2	-0.139	0.224	0.534	3
F1 GAD-7 Anxiety	exposure*ethnicity4	-0.005	0.315	0.988	2	-0.035	0.315	0.913	3
F1 GAD-7 Anxiety	exposure*ethnicity5	0.273	1.05	0.795	2	0.299	1.075	0.781	3
F1 Executive Function	exposure*ethnicity2	-0.051	0.033	0.124	2	-0.039	0.034	0.256	3
F1 Executive Function	exposure*ethnicity3	-0.058	0.023	0.011	2	-0.046	0.024	0.052	3
F1 Executive Function	exposure*ethnicity4	-0.113	0.032	0	2	-0.096	0.032	0.003	3
F1 Executive Function	exposure*ethnicity5	-0.265	0.124	0.033	2	-0.251	0.123	0.042	3

F1 Culture Fair Intelligence Test	exposure*ethnicity2	-0.343	0.19	0.071	2	-0.331	0.193	0.087	3
F1 Culture Fair Intelligence Test	exposure*ethnicity3	-0.202	0.134	0.13	2	-0.181	0.139	0.193	3
F1 Culture Fair Intelligence Test	exposure*ethnicity4	-0.501	0.194	0.01	2	-0.471	0.195	0.016	3
F1 Culture Fair Intelligence Test	exposure*ethnicity5	-2.5	0.679	0	2	-2.53	0.693	0	3
F1 Processing Speed	exposure*ethnicity2	0.035	0.04	0.376	2	0	0.04	0.995	3
F1 Processing Speed	exposure*ethnicity3	0.027	0.025	0.269	2	0.005	0.026	0.835	3
F1 Processing Speed	exposure*ethnicity4	0.045	0.035	0.199	2	0.029	0.035	0.407	3
F1 Processing Speed	exposure*ethnicity5	-0.177	0.12	0.142	2	-0.229	0.118	0.053	3
F1 Speech-in-noise Threshold	exposure*ethnicity2	-0.317	0.319	0.32	2	-0.37	0.322	0.251	3
F1 Speech-in-noise Threshold	exposure*ethnicity3	0.22	0.19	0.247	2	0.141	0.196	0.472	3

F1 Speech-in-noise Threshold	exposure*ethnicity4	0.136	0.296	0.647	2	0.171	0.293	0.56	3
F1 Speech-in-noise Threshold	exposure*ethnicity5	0.716	1.043	0.492	2	0.69	1.036	0.506	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*religion	-0.043	0.021	0.037	2	-0.042	0.021	0.043	3
BL Internalising	exposure*religion	-0.028	0.012	0.02	2	-0.027	0.012	0.026	3
BL Externalising	exposure*religion	-0.015	0.013	0.252	2	-0.015	0.013	0.254	3
BL Executive Function	exposure*religion	-0.003	0.002	0.178	2	-0.004	0.002	0.077	3
BL Culture Fair Intelligence Test	exposure*religion	-0.025	0.013	0.057	2	-0.024	0.013	0.065	3
BL Processing Speed	exposure*religion	0.003	0.002	0.166	2	0.003	0.002	0.207	3
BL Speech-in-noise Threshold	exposure*religion	0.014	0.013	0.269	2	0.015	0.013	0.229	3
F1 SDQ	exposure*religion	-0.063	0.02	0.002	2	-0.062	0.02	0.002	3
F1 Internalising	exposure*religion	-0.042	0.012	0.001	2	-0.04	0.012	0.001	3
F1 Externalising	exposure*religion	-0.019	0.012	0.105	2	-0.018	0.012	0.125	3
F1 PHQ-9 Depression	exposure*religion	-0.075	0.022	0.001	2	-0.077	0.022	0.001	3
F1 GAD-7 Anxiety	exposure*religion	-0.063	0.019	0.001	2	-0.066	0.019	0.001	3

F1 Executive Function	exposure*religion	0	0.002	0.957	2	-0.001	0.002	0.757	3
F1 Culture Fair Intelligence Test	exposure*religion	-0.018	0.012	0.135	2	-0.017	0.012	0.155	3
F1 Processing Speed	exposure*religion	0.003	0.002	0.131	2	0.003	0.002	0.172	3
F1 Speech-in-noise Threshold	exposure*religion	0.007	0.016	0.634	2	0.01	0.016	0.533	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure* BL Lden (high/low)	0.024	0.014	0.1	2	0.025	0.015	0.089	3
BL Internalising	exposure* BL Lden (high/low)	0.009	0.009	0.306	2	0.009	0.009	0.32	3
BL Externalising	exposure* BL Lden (high/low)	0.015	0.009	0.089	2	0.016	0.009	0.07	3
BL Executive Function	exposure* BL Lden (high/low)	-0.002	0.002	0.324	2	-0.001	0.002	0.455	3
BL Culture Fair Intelligence Test	exposure* BL Lden (high/low)	0.008	0.009	0.379	2	0.01	0.009	0.271	3
BL Processing Speed	exposure* BL Lden (high/low)	0.002	0.002	0.186	2	0.002	0.002	0.338	3
BL Speech-in-noise Threshold	exposure* BL Lden (high/low)	0.002	0.009	0.808	2	0.002	0.009	0.812	3

F1 SDQ	exposure: F1 Lden (high/low)	0.013	0.014	0.367	2	0.01	0.014	0.487	3
F1 Internalising	exposure: F1 Lden (high/low)	0.002	0.009	0.794	2	0.001	0.009	0.863	3
F1 Externalising	exposure: F1 Lden (high/low)	0.006	0.008	0.459	2	0.004	0.008	0.648	3
F1 PHQ-9 Depression	exposure: F1 Lden (high/low)	-0.004	0.016	0.82	2	-0.005	0.016	0.752	3
F1 GAD-7 Anxiety	exposure: F1 Lden (high/low)	0.009	0.013	0.519	2	0.009	0.014	0.512	3
F1 Executive Function	exposure: F1 Lden (high/low)	-0.002	0.001	0.254	2	-0.002	0.001	0.252	3
F1 Culture Fair Intelligence Test	exposure: F1 Lden (high/low)	-0.012	0.008	0.129	2	-0.012	0.008	0.16	3
F1 Processing Speed	exposure: F1 Lden (high/low)	-0.001	0.002	0.485	2	-0.002	0.002	0.334	3
F1 Speech-in-noise Threshold	exposure: F1 Lden (high/low)	0.017	0.013	0.179	2	0.014	0.013	0.253	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*School Type (Independent yes/no)	0.088	0.215	0.682	2	0.055	0.24	0.819	3
BL Internalising	exposure*School Type	-0.044	0.127	0.726	2	-0.042	0.14	0.767	3

	(Independent yes/no)								
BL Externalising	exposure*School Type (Independent yes/no)	0.133	0.132	0.315	2	0.101	0.146	0.486	3
BL Executive Function	exposure*School Type (Independent yes/no)	0.068	0.025	0.006	2	0.035	0.028	0.205	3
BL Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.234	0.137	0.088	2	0.14	0.157	0.372	3
BL Processing Speed	exposure*School Type (Independent yes/no)	-0.083	0.023	0	2	-0.064	0.026	0.015	3
BL Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	0.132	0.129	0.309	2	0.306	0.144	0.034	3
F1 SDQ	exposure*School Type (Independent yes/no)	-0.186	0.221	0.4	2	0.039	0.245	0.872	3
F1 Internalising	exposure*School Type (Independent yes/no)	-0.184	0.132	0.163	2	-0.071	0.147	0.63	3
F1 Externalising	exposure*School Type (Independent yes/no)	-0.017	0.128	0.895	2	0.113	0.142	0.423	3

F1 PHQ-9 Depression	exposure*School Type (Independent yes/no)	0.063	0.255	0.806	2	0.062	0.271	0.819	3
F1 GAD-7 Anxiety	exposure*School Type (Independent yes/no)	0.302	0.217	0.165	2	0.306	0.225	0.175	3
F1 Executive Function	exposure*School Type (Independent yes/no)	0.013	0.021	0.544	2	-0.011	0.024	0.65	3
F1 Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	-0.032	0.129	0.801	2	-0.074	0.14	0.598	3
F1 Processing Speed	exposure*School Type (Independent yes/no)	-0.036	0.023	0.123	2	-0.008	0.026	0.758	3
F1 Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	-0.245	0.172	0.156	2	-0.07	0.193	0.718	3

Exposure: NO2

Outcome	Interaction	Estimate	SE	p	Model	Estimate	SE	p	Model
BL SDQ	exposure*IQ	-0.21	0.036	0	2	-0.197	0.037	0	3
BL Internalising	exposure*IQ	-0.114	0.021	0	2	-0.106	0.022	0	3
BL Externalising	exposure*IQ	-0.096	0.022	0	2	-0.092	0.022	0	3
BL Executive Function	exposure*IQ	0.072	0.004	0	2	0.07	0.004	0	3
BL Culture Fair Intelligence Test	exposure*IQ	1.007	0.014	0	2	0.997	0.014	0	3
BL Processing Speed	exposure*IQ	0.001	0.004	0.898	2	0	0.004	0.934	3
BL Speech-in-noise Threshold	exposure*IQ	-0.101	0.023	0	2	-0.1	0.023	0	3
F1 SDQ	exposure*IQ	-0.071	0.034	0.038	2	-0.066	0.035	0.056	3
F1 Internalising	exposure*IQ	-0.013	0.021	0.529	2	-0.004	0.021	0.836	3
F1 Externalising	exposure*IQ	-0.008	0.02	0.674	2	-0.012	0.02	0.535	3
F1 PHQ-9 Depression	exposure*IQ	0.034	0.037	0.36	2	0.04	0.038	0.288	3
F1 GAD-7 Anxiety	exposure*IQ	0.025	0.032	0.432	2	0.026	0.032	0.41	3
F1 Executive Function	exposure*IQ	0.014	0.004	0	2	0.014	0.004	0	3
F1 Culture Fair	exposure*IQ	0.007	0.031	0.811	2	0.013	0.031	0.667	3

Intelligence Test									
F1 Processing Speed	exposure*IQ	0.005	0.004	0.223	2	0.005	0.004	0.156	3
F1 Speech-in-noise Threshold	exposure*IQ	-0.083	0.031	0.008	2	-0.089	0.031	0.004	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*sex	0.089	0.238	0.709	2	0.062	0.255	0.808	3
BL Internalising	exposure*sex	0.001	0.14	0.995	2	0.031	0.149	0.838	3
BL Externalising	exposure*sex	0.088	0.146	0.546	2	0.036	0.155	0.819	3
BL Executive Function	exposure*sex	0.072	0.027	0.008	2	0.044	0.029	0.134	3
BL Culture Fair Intelligence Test	exposure*sex	0.411	0.147	0.005	2	0.267	0.159	0.093	3
BL Processing Speed	exposure*sex	-0.009	0.028	0.744	2	0.018	0.03	0.538	3
BL Speech-in-noise Threshold	exposure*sex	0.065	0.148	0.66	2	0.228	0.158	0.148	3
F1 SDQ	exposure*sex	-0.19	0.229	0.407	2	-0.094	0.243	0.698	3
F1 Internalising	exposure*sex	0.057	0.137	0.675	2	0.194	0.146	0.184	3
F1 Externalising	exposure*sex	-0.201	0.135	0.137	2	-0.216	0.143	0.132	3
F1 PHQ-9 Depression	exposure*sex	0.294	0.258	0.255	2	0.319	0.269	0.235	3

F1 GAD-7 Anxiety	exposure*sex	0.114	0.22	0.602	2	0.112	0.225	0.619	3
F1 Executive Function	exposure*sex	0.033	0.022	0.135	2	0.026	0.024	0.281	3
F1 Culture Fair Intelligence Test	exposure*sex	0.036	0.131	0.783	2	0.043	0.138	0.758	3
F1 Processing Speed	exposure*sex	0.015	0.026	0.556	2	0.013	0.027	0.626	3
F1 Speech-in-noise Threshold	exposure*sex	-0.232	0.199	0.244	2	-0.172	0.21	0.414	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*ethnicity2	0.587	0.352	0.095	2	0.657	0.365	0.072	3
BL SDQ	exposure*ethnicity3	0.459	0.28	0.101	2	0.525	0.299	0.079	3
BL SDQ	exposure*ethnicity4	0.646	0.428	0.132	2	0.59	0.43	0.17	3
BL SDQ	exposure*ethnicity5	0.064	1.67	0.97	2	0.473	1.73	0.784	3
BL Internalising	exposure*ethnicity2	0.501	0.207	0.015	2	0.521	0.214	0.015	3
BL Internalising	exposure*ethnicity3	0.475	0.164	0.004	2	0.479	0.175	0.006	3
BL Internalising	exposure*ethnicity4	0.438	0.252	0.082	2	0.408	0.253	0.106	3
BL Internalising	exposure*ethnicity5	0.656	0.981	0.503	2	0.887	1.018	0.384	3
BL Externalising	exposure*ethnicity2	0.086	0.216	0.689	2	0.131	0.223	0.558	3

BL Externalising	exposure*ethnicity3	-0.017	0.171	0.922	2	0.038	0.183	0.835	3
BL Externalising	exposure*ethnicity4	0.208	0.263	0.429	2	0.187	0.263	0.477	3
BL Externalising	exposure*ethnicity5	-0.593	1.023	0.562	2	-0.407	1.06	0.701	3
BL Executive Function	exposure*ethnicity2	-0.102	0.041	0.012	2	-0.054	0.042	0.195	3
BL Executive Function	exposure*ethnicity3	-0.058	0.032	0.07	2	-0.029	0.034	0.401	3
BL Executive Function	exposure*ethnicity4	-0.074	0.047	0.116	2	-0.057	0.047	0.228	3
BL Executive Function	exposure*ethnicity5	-0.319	0.215	0.138	2	-0.24	0.213	0.262	3
BL Culture Fair Intelligence Test	exposure*ethnicity2	-0.244	0.217	0.259	2	-0.111	0.225	0.62	3
BL Culture Fair Intelligence Test	exposure*ethnicity3	-0.215	0.174	0.217	2	-0.1	0.187	0.591	3
BL Culture Fair Intelligence Test	exposure*ethnicity4	-0.26	0.264	0.326	2	-0.194	0.263	0.462	3
BL Culture Fair Intelligence Test	exposure*ethnicity5	-0.322	1.066	0.763	2	-0.375	1.093	0.732	3
BL Processing Speed	exposure*ethnicity2	0.066	0.042	0.113	2	0.058	0.042	0.172	3

BL Processing Speed	exposure*ethnicity3	0.057	0.031	0.065	2	0.031	0.033	0.348	3
BL Processing Speed	exposure*ethnicity4	0.052	0.054	0.336	2	0.038	0.054	0.475	3
BL Processing Speed	exposure*ethnicity5	-0.064	0.176	0.715	2	-0.125	0.173	0.471	3
BL Speech-in-noise Threshold	exposure*ethnicity2	-0.167	0.221	0.45	2	-0.223	0.227	0.324	3
BL Speech-in-noise Threshold	exposure*ethnicity3	0.109	0.172	0.528	2	0.14	0.183	0.445	3
BL Speech-in-noise Threshold	exposure*ethnicity4	0.063	0.252	0.802	2	0.029	0.251	0.908	3
BL Speech-in-noise Threshold	exposure*ethnicity5	1.27	1.113	0.254	2	1.222	1.155	0.29	3
F1 SDQ	exposure*ethnicity2	0.093	0.343	0.786	2	0.031	0.356	0.931	3
F1 SDQ	exposure*ethnicity3	0.182	0.267	0.495	2	0.004	0.28	0.988	3
F1 SDQ	exposure*ethnicity4	0.032	0.393	0.935	2	-0.021	0.393	0.957	3
F1 SDQ	exposure*ethnicity5	0.296	1.515	0.845	2	-0.464	1.567	0.767	3
F1 Internalising	exposure*ethnicity2	0.098	0.205	0.633	2	0.055	0.213	0.796	3
F1 Internalising	exposure*ethnicity3	0.12	0.159	0.453	2	0.039	0.168	0.818	3
F1 Internalising	exposure*ethnicity4	-0.187	0.235	0.426	2	-0.214	0.235	0.363	3

F1 Internalising	exposure*ethnicity5	-0.184	0.905	0.839	2	-0.517	0.938	0.581	3
F1 Externalising	exposure*ethnicity2	-0.045	0.205	0.828	2	-0.099	0.213	0.643	3
F1 Externalising	exposure*ethnicity3	0.123	0.157	0.431	2	-0.009	0.165	0.957	3
F1 Externalising	exposure*ethnicity4	0.239	0.231	0.302	2	0.204	0.231	0.377	3
F1 Externalising	exposure*ethnicity5	0.735	0.9	0.414	2	0.216	0.934	0.817	3
F1 PHQ-9 Depression	exposure*ethnicity2	-0.096	0.389	0.805	2	-0.111	0.402	0.783	3
F1 PHQ-9 Depression	exposure*ethnicity3	-0.038	0.299	0.9	2	-0.04	0.311	0.897	3
F1 PHQ-9 Depression	exposure*ethnicity4	-0.173	0.475	0.716	2	-0.166	0.477	0.728	3
F1 PHQ-9 Depression	exposure*ethnicity5	-0.32	1.584	0.84	2	-0.432	1.646	0.793	3
F1 GAD-7 Anxiety	exposure*ethnicity2	-0.211	0.329	0.522	2	-0.179	0.338	0.597	3
F1 GAD-7 Anxiety	exposure*ethnicity3	-0.101	0.255	0.691	2	-0.094	0.263	0.721	3
F1 GAD-7 Anxiety	exposure*ethnicity4	0.011	0.401	0.978	2	0.002	0.402	0.996	3
F1 GAD-7 Anxiety	exposure*ethnicity5	1.069	1.352	0.429	2	1.031	1.402	0.462	3
F1 Executive Function	exposure*ethnicity2	-0.071	0.033	0.032	2	-0.061	0.034	0.076	3
F1 Executive Function	exposure*ethnicity3	-0.064	0.026	0.015	2	-0.054	0.027	0.047	3
F1 Executive Function	exposure*ethnicity4	-0.124	0.036	0.001	2	-0.109	0.036	0.003	3
F1 Executive Function	exposure*ethnicity5	-0.284	0.163	0.082	2	-0.259	0.162	0.112	3

F1 Culture Fair Intelligence Test	exposure*ethnicity2	-0.42	0.194	0.031	2	-0.359	0.201	0.074	3
F1 Culture Fair Intelligence Test	exposure*ethnicity3	-0.189	0.153	0.216	2	-0.137	0.16	0.394	3
F1 Culture Fair Intelligence Test	exposure*ethnicity4	-0.358	0.224	0.11	2	-0.309	0.225	0.17	3
F1 Culture Fair Intelligence Test	exposure*ethnicity5	-2.271	0.903	0.012	2	-2.213	0.937	0.018	3
F1 Processing Speed	exposure*ethnicity2	0.045	0.04	0.26	2	-0.002	0.041	0.965	3
F1 Processing Speed	exposure*ethnicity3	0.053	0.028	0.055	2	0.021	0.029	0.471	3
F1 Processing Speed	exposure*ethnicity4	0.124	0.05	0.013	2	0.09	0.049	0.069	3
F1 Processing Speed	exposure*ethnicity5	-0.156	0.157	0.321	2	-0.227	0.154	0.143	3
F1 Speech-in-noise Threshold	exposure*ethnicity2	0.087	0.319	0.785	2	0.001	0.324	0.998	3
F1 Speech-in-noise Threshold	exposure*ethnicity3	0.273	0.221	0.217	2	0.209	0.23	0.363	3

F1 Speech-in-noise Threshold	exposure*ethnicity4	0.035	0.327	0.915	2	0.063	0.324	0.846	3
F1 Speech-in-noise Threshold	exposure*ethnicity5	1.583	1.342	0.238	2	1.525	1.332	0.253	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*religion	-0.112	0.051	0.027	2	-0.111	0.051	0.029	3
BL Internalising	exposure*religion	-0.07	0.03	0.02	2	-0.068	0.03	0.023	3
BL Externalising	exposure*religion	-0.042	0.031	0.178	2	-0.042	0.031	0.178	3
BL Executive Function	exposure*religion	-0.009	0.006	0.124	2	-0.01	0.006	0.078	3
BL Culture Fair Intelligence Test	exposure*religion	-0.066	0.032	0.04	2	-0.061	0.032	0.057	3
BL Processing Speed	exposure*religion	0.009	0.006	0.112	2	0.008	0.006	0.174	3
BL Speech-in-noise Threshold	exposure*religion	0.031	0.031	0.308	2	0.033	0.031	0.283	3
F1 SDQ	exposure*religion	-0.137	0.046	0.003	2	-0.137	0.046	0.003	3
F1 Internalising	exposure*religion	-0.09	0.027	0.001	2	-0.089	0.028	0.001	3
F1 Externalising	exposure*religion	-0.037	0.026	0.167	2	-0.037	0.027	0.169	3
F1 PHQ-9 Depression	exposure*religion	-0.169	0.05	0.001	2	-0.172	0.05	0.001	3
F1 GAD-7 Anxiety	exposure*religion	-0.146	0.043	0.001	2	-0.151	0.043	0	3

F1 Executive Function	exposure*religion	-0.001	0.004	0.88	2	-0.001	0.004	0.743	3
F1 Culture Fair Intelligence Test	exposure*religion	-0.049	0.027	0.071	2	-0.047	0.027	0.087	3
F1 Processing Speed	exposure*religion	0.007	0.005	0.116	2	0.006	0.005	0.184	3
F1 Speech-in-noise Threshold	exposure*religion	0.025	0.035	0.475	2	0.029	0.035	0.414	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure* BL Lden (high/low)	0.073	0.036	0.042	2	0.065	0.036	0.073	3
BL Internalising	exposure* BL Lden (high/low)	0.032	0.021	0.126	2	0.026	0.021	0.225	3
BL Externalising	exposure* BL Lden (high/low)	0.041	0.022	0.064	2	0.04	0.022	0.075	3
BL Executive Function	exposure* BL Lden (high/low)	-0.005	0.004	0.197	2	-0.004	0.004	0.396	3
BL Culture Fair Intelligence Test	exposure* BL Lden (high/low)	0.02	0.023	0.368	2	0.025	0.023	0.261	3
BL Processing Speed	exposure* BL Lden (high/low)	0.006	0.004	0.157	2	0.004	0.004	0.382	3
BL Speech-in-noise Threshold	exposure* BL Lden (high/low)	0.014	0.022	0.519	2	0.007	0.022	0.736	3

F1 SDQ	exposure: F1 Lden (high/low)	0.038	0.033	0.25	2	0.022	0.033	0.504	3
F1 Internalising	exposure: F1 Lden (high/low)	0.012	0.02	0.534	2	0.006	0.02	0.783	3
F1 Externalising	exposure: F1 Lden (high/low)	0.019	0.019	0.331	2	0.011	0.02	0.592	3
F1 PHQ-9 Depression	exposure: F1 Lden (high/low)	-0.004	0.036	0.913	2	-0.01	0.037	0.794	3
F1 GAD-7 Anxiety	exposure: F1 Lden (high/low)	0.023	0.031	0.448	2	0.022	0.031	0.479	3
F1 Executive Function	exposure: F1 Lden (high/low)	-0.007	0.003	0.038	2	-0.006	0.003	0.076	3
F1 Culture Fair Intelligence Test	exposure: F1 Lden (high/low)	-0.029	0.019	0.127	2	-0.029	0.019	0.135	3
F1 Processing Speed	exposure: F1 Lden (high/low)	-0.002	0.004	0.595	2	-0.003	0.004	0.339	3
F1 Speech-in-noise Threshold	exposure: F1 Lden (high/low)	0.046	0.029	0.114	2	0.037	0.029	0.198	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*School Type (Independent yes/no)	0.329	0.251	0.19	2	0.271	0.31	0.383	3
BL Internalising	exposure*School Type	0.043	0.148	0.773	2	0.027	0.18	0.882	3

	(Independent yes/no)								
BL Externalising	exposure*School Type (Independent yes/no)	0.286	0.154	0.063	2	0.255	0.185	0.171	3
BL Executive Function	exposure*School Type (Independent yes/no)	0.077	0.028	0.006	2	0.041	0.036	0.248	3
BL Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.429	0.157	0.006	2	0.383	0.208	0.066	3
BL Processing Speed	exposure*School Type (Independent yes/no)	-0.065	0.028	0.021	2	-0.059	0.037	0.108	3
BL Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	0.173	0.15	0.249	2	0.332	0.185	0.074	3
F1 SDQ	exposure*School Type (Independent yes/no)	-0.333	0.234	0.156	2	-0.145	0.281	0.606	3
F1 Internalising	exposure*School Type (Independent yes/no)	-0.252	0.14	0.073	2	-0.157	0.169	0.353	3
F1 Externalising	exposure*School Type (Independent yes/no)	-0.135	0.139	0.332	2	-0.025	0.167	0.879	3

F1 PHQ-9 Depression	exposure*School Type (Independent yes/no)	-0.075	0.285	0.792	2	-0.077	0.314	0.805	3
F1 GAD-7 Anxiety	exposure*School Type (Independent yes/no)	0.134	0.243	0.582	2	0.14	0.258	0.59	3
F1 Executive Function	exposure*School Type (Independent yes/no)	0.025	0.023	0.272	2	0.013	0.027	0.624	3
F1 Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.198	0.138	0.152	2	0.14	0.158	0.376	3
F1 Processing Speed	exposure*School Type (Independent yes/no)	-0.03	0.025	0.236	2	0.024	0.032	0.464	3
F1 Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	-0.32	0.191	0.093	2	-0.185	0.222	0.406	3

Exposure: Lden

Outcome	Interaction	Estimate	SE	p	Model	Estimate	SE	p	Model
BL SDQ	exposure*IQ	-0.13	0.022	0	2	-0.121	0.022	0	3
BL Internalising	exposure*IQ	-0.073	0.013	0	2	-0.068	0.013	0	3
BL Externalising	exposure*IQ	-0.057	0.013	0	2	-0.054	0.013	0	3
BL Executive Function	exposure*IQ	0.042	0.002	0	2	0.042	0.002	0	3
BL Culture Fair Intelligence Test	exposure*IQ	0.601	0.008	0	2	0.595	0.008	0	3
BL Processing Speed	exposure*IQ	0	0.002	0.878	2	0	0.002	0.917	3
BL Speech-in-noise Threshold	exposure*IQ	-0.062	0.014	0	2	-0.062	0.014	0	3
F1 SDQ	exposure*IQ	-0.045	0.023	0.046	2	-0.042	0.023	0.063	3
F1 Internalising	exposure*IQ	-0.008	0.014	0.556	2	-0.003	0.014	0.843	3
F1 Externalising	exposure*IQ	-0.005	0.013	0.72	2	-0.008	0.013	0.548	3
F1 PHQ-9 Depression	exposure*IQ	0.027	0.025	0.273	2	0.031	0.025	0.218	3
F1 GAD-7 Anxiety	exposure*IQ	0.014	0.021	0.495	2	0.015	0.021	0.471	3
F1 Executive Function	exposure*IQ	0.009	0.002	0	2	0.008	0.002	0	3
F1 Culture Fair	exposure*IQ	0.007	0.021	0.749	2	0.01	0.021	0.617	3

Intelligence Test									
F1 Processing Speed	exposure*IQ	0.003	0.002	0.18	2	0.004	0.002	0.14	3
F1 Speech-in-noise Threshold	exposure*IQ	-0.05	0.021	0.015	2	-0.054	0.02	0.008	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*sex	0.188	0.188	0.318	2	0.192	0.188	0.307	3
BL Internalising	exposure*sex	0.174	0.111	0.117	2	0.186	0.111	0.093	3
BL Externalising	exposure*sex	0.014	0.115	0.9	2	0.008	0.115	0.947	3
BL Executive Function	exposure*sex	0.008	0.022	0.726	2	-0.001	0.022	0.96	3
BL Culture Fair Intelligence Test	exposure*sex	0.079	0.117	0.5	2	0.017	0.116	0.883	3
BL Processing Speed	exposure*sex	0.009	0.022	0.675	2	0.015	0.021	0.477	3
BL Speech-in-noise Threshold	exposure*sex	0.009	0.117	0.936	2	0	0.117	0.999	3
F1 SDQ	exposure*sex	0.064	0.2	0.751	2	0.073	0.2	0.714	3
F1 Internalising	exposure*sex	0.142	0.12	0.236	2	0.153	0.12	0.202	3
F1 Externalising	exposure*sex	0.032	0.118	0.784	2	0.029	0.118	0.805	3
F1 PHQ-9 Depression	exposure*sex	0.239	0.222	0.281	2	0.242	0.223	0.278	3

F1 GAD-7 Anxiety	exposure*sex	-0.007	0.187	0.972	2	-0.041	0.188	0.826	3
F1 Executive Function	exposure*sex	0.03	0.019	0.117	2	0.03	0.019	0.112	3
F1 Culture Fair Intelligence Test	exposure*sex	0.02	0.113	0.859	2	0.004	0.114	0.969	3
F1 Processing Speed	exposure*sex	0.034	0.023	0.148	2	0.033	0.023	0.147	3
F1 Speech-in-noise Threshold	exposure*sex	-0.11	0.18	0.544	2	-0.094	0.179	0.598	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*ethnicity2	0.105	0.259	0.684	2	0.162	0.26	0.534	3
BL SDQ	exposure*ethnicity3	-0.15	0.227	0.51	2	-0.189	0.227	0.407	3
BL SDQ	exposure*ethnicity4	-0.186	0.341	0.587	2	-0.236	0.34	0.487	3
BL SDQ	exposure*ethnicity5	-0.211	1.023	0.837	2	-0.189	1.017	0.852	3
BL Internalising	exposure*ethnicity2	0.198	0.152	0.192	2	0.216	0.153	0.158	3
BL Internalising	exposure*ethnicity3	-0.01	0.133	0.939	2	-0.022	0.134	0.871	3
BL Internalising	exposure*ethnicity4	-0.166	0.201	0.409	2	-0.182	0.2	0.362	3
BL Internalising	exposure*ethnicity5	0.233	0.602	0.699	2	0.272	0.598	0.649	3
BL Externalising	exposure*ethnicity2	-0.093	0.158	0.557	2	-0.058	0.159	0.718	3

BL Externalising	exposure*ethnicity3	-0.139	0.139	0.316	2	-0.163	0.139	0.243	3
BL Externalising	exposure*ethnicity4	-0.02	0.209	0.925	2	-0.053	0.208	0.801	3
BL Externalising	exposure*ethnicity5	-0.443	0.627	0.48	2	-0.46	0.623	0.461	3
BL Executive Function	exposure*ethnicity2	0.004	0.03	0.894	2	0.017	0.03	0.574	3
BL Executive Function	exposure*ethnicity3	-0.005	0.026	0.849	2	-0.001	0.026	0.963	3
BL Executive Function	exposure*ethnicity4	-0.035	0.039	0.371	2	-0.022	0.038	0.574	3
BL Executive Function	exposure*ethnicity5	-0.096	0.118	0.413	2	-0.099	0.116	0.392	3
BL Culture Fair Intelligence Test	exposure*ethnicity2	-0.142	0.159	0.373	2	-0.127	0.159	0.425	3
BL Culture Fair Intelligence Test	exposure*ethnicity3	-0.161	0.141	0.252	2	-0.172	0.14	0.221	3
BL Culture Fair Intelligence Test	exposure*ethnicity4	-0.043	0.211	0.837	2	0	0.208	0.999	3
BL Culture Fair Intelligence Test	exposure*ethnicity5	-0.437	0.652	0.503	2	-0.444	0.64	0.488	3
BL Processing Speed	exposure*ethnicity2	0.059	0.03	0.052	2	0.052	0.03	0.081	3

BL Processing Speed	exposure*ethnicity3	0.019	0.026	0.455	2	0.015	0.026	0.547	3
BL Processing Speed	exposure*ethnicity4	0.015	0.04	0.702	2	0.003	0.04	0.943	3
BL Processing Speed	exposure*ethnicity5	-0.022	0.099	0.821	2	-0.049	0.096	0.608	3
BL Speech-in-noise Threshold	exposure*ethnicity2	-0.256	0.162	0.114	2	-0.25	0.162	0.125	3
BL Speech-in-noise Threshold	exposure*ethnicity3	0.032	0.14	0.82	2	-0.016	0.141	0.909	3
BL Speech-in-noise Threshold	exposure*ethnicity4	-0.191	0.208	0.358	2	-0.245	0.206	0.235	3
BL Speech-in-noise Threshold	exposure*ethnicity5	-0.091	0.647	0.888	2	-0.093	0.642	0.885	3
F1 SDQ	exposure*ethnicity2	0.173	0.284	0.543	2	0.199	0.285	0.485	3
F1 SDQ	exposure*ethnicity3	-0.189	0.236	0.422	2	-0.278	0.237	0.241	3
F1 SDQ	exposure*ethnicity4	-0.198	0.352	0.574	2	-0.27	0.35	0.44	3
F1 SDQ	exposure*ethnicity5	-0.6	1.333	0.653	2	-0.651	1.321	0.622	3
F1 Internalising	exposure*ethnicity2	0.055	0.17	0.748	2	0.054	0.17	0.75	3
F1 Internalising	exposure*ethnicity3	-0.072	0.141	0.609	2	-0.115	0.142	0.417	3
F1 Internalising	exposure*ethnicity4	-0.302	0.21	0.151	2	-0.326	0.209	0.12	3

F1 Internalising	exposure*ethnicity5	-0.318	0.797	0.69	2	-0.322	0.79	0.684	3
F1 Externalising	exposure*ethnicity2	0.234	0.171	0.171	2	0.248	0.171	0.146	3
F1 Externalising	exposure*ethnicity3	-0.133	0.139	0.336	2	-0.164	0.139	0.24	3
F1 Externalising	exposure*ethnicity4	0.136	0.206	0.509	2	0.1	0.205	0.626	3
F1 Externalising	exposure*ethnicity5	-0.274	0.782	0.726	2	-0.37	0.776	0.633	3
F1 PHQ-9 Depression	exposure*ethnicity2	-0.21	0.336	0.532	2	-0.19	0.34	0.576	3
F1 PHQ-9 Depression	exposure*ethnicity3	-0.362	0.256	0.157	2	-0.36	0.259	0.165	3
F1 PHQ-9 Depression	exposure*ethnicity4	-0.283	0.406	0.486	2	-0.33	0.407	0.417	3
F1 PHQ-9 Depression	exposure*ethnicity5	-0.231	1.392	0.868	2	-0.168	1.391	0.904	3
F1 GAD-7 Anxiety	exposure*ethnicity2	0.211	0.278	0.447	2	0.239	0.28	0.394	3
F1 GAD-7 Anxiety	exposure*ethnicity3	-0.19	0.218	0.382	2	-0.199	0.219	0.364	3
F1 GAD-7 Anxiety	exposure*ethnicity4	0.236	0.34	0.488	2	0.245	0.34	0.472	3
F1 GAD-7 Anxiety	exposure*ethnicity5	1.67	1.187	0.16	2	1.734	1.184	0.143	3
F1 Executive Function	exposure*ethnicity2	-0.032	0.027	0.233	2	-0.036	0.027	0.19	3
F1 Executive Function	exposure*ethnicity3	-0.006	0.023	0.79	2	-0.009	0.023	0.688	3
F1 Executive Function	exposure*ethnicity4	0.014	0.033	0.666	2	0.009	0.033	0.783	3
F1 Executive Function	exposure*ethnicity5	-0.122	0.127	0.337	2	-0.112	0.126	0.371	3

F1 Culture Fair Intelligence Test	exposure*ethnicity2	-0.337	0.158	0.033	2	-0.325	0.159	0.041	3
F1 Culture Fair Intelligence Test	exposure*ethnicity3	0.002	0.135	0.985	2	-0.015	0.136	0.914	3
F1 Culture Fair Intelligence Test	exposure*ethnicity4	-0.469	0.198	0.018	2	-0.463	0.198	0.019	3
F1 Culture Fair Intelligence Test	exposure*ethnicity5	-1.619	0.726	0.026	2	-1.629	0.725	0.025	3
F1 Processing Speed	exposure*ethnicity2	0	0.034	0.992	2	-0.016	0.034	0.642	3
F1 Processing Speed	exposure*ethnicity3	0.039	0.026	0.132	2	0.02	0.026	0.432	3
F1 Processing Speed	exposure*ethnicity4	0.003	0.044	0.952	2	0.013	0.043	0.765	3
F1 Processing Speed	exposure*ethnicity5	0.004	0.173	0.98	2	-0.035	0.169	0.838	3
F1 Speech-in-noise Threshold	exposure*ethnicity2	0.164	0.262	0.532	2	0.109	0.261	0.677	3
F1 Speech-in-noise Threshold	exposure*ethnicity3	0	0.207	1	2	-0.007	0.207	0.972	3

F1 Speech-in-noise Threshold	exposure*ethnicity4	0.15	0.33	0.649	2	0.201	0.327	0.538	3
F1 Speech-in-noise Threshold	exposure*ethnicity5	3.541	1.392	0.011	2	3.612	1.374	0.009	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*religion	-0.067	0.031	0.028	2	-0.07	0.031	0.024	3
BL Internalising	exposure*religion	-0.042	0.018	0.02	2	-0.042	0.018	0.022	3
BL Externalising	exposure*religion	-0.025	0.019	0.186	2	-0.028	0.019	0.146	3
BL Executive Function	exposure*religion	-0.005	0.004	0.167	2	-0.007	0.004	0.06	3
BL Culture Fair Intelligence Test	exposure*religion	-0.041	0.019	0.034	2	-0.04	0.02	0.039	3
BL Processing Speed	exposure*religion	0.004	0.003	0.187	2	0.004	0.003	0.233	3
BL Speech-in-noise Threshold	exposure*religion	0.024	0.019	0.191	2	0.025	0.019	0.18	3
F1 SDQ	exposure*religion	-0.098	0.03	0.001	2	-0.096	0.03	0.002	3
F1 Internalising	exposure*religion	-0.063	0.018	0.001	2	-0.06	0.018	0.001	3
F1 Externalising	exposure*religion	-0.031	0.018	0.083	2	-0.029	0.018	0.108	3
F1 PHQ-9 Depression	exposure*religion	-0.116	0.033	0.001	2	-0.118	0.034	0	3
F1 GAD-7 Anxiety	exposure*religion	-0.096	0.029	0.001	2	-0.1	0.029	0.001	3

F1 Executive Function	exposure*religion	0	0.003	0.889	2	-0.001	0.003	0.745	3
F1 Culture Fair Intelligence Test	exposure*religion	-0.027	0.018	0.139	2	-0.026	0.018	0.151	3
F1 Processing Speed	exposure*religion	0.004	0.003	0.142	2	0.004	0.003	0.207	3
F1 Speech-in-noise Threshold	exposure*religion	0.009	0.024	0.711	2	0.014	0.024	0.568	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure* PM10 (high/low)	-0.009	0.027	0.74	2	-0.009	0.029	0.746	3
BL Internalising	exposure* PM10 (high/low)	-0.009	0.027	0.74	2	-0.009	0.029	0.746	3
BL Externalising	exposure* PM10 (high/low)	-0.009	0.027	0.74	2	-0.009	0.029	0.746	3
BL Executive Function	exposure* PM10 (high/low)	-0.009	0.027	0.74	2	-0.009	0.029	0.746	3
BL Culture Fair Intelligence Test	exposure* PM10 (high/low)	-0.009	0.027	0.74	2	-0.009	0.029	0.746	3
BL Processing Speed	exposure* PM10 (high/low)	-0.009	0.027	0.74	2	-0.009	0.029	0.746	3
BL Speech-in-noise Threshold	exposure* PM10 (high/low)	-0.009	0.027	0.74	2	-0.009	0.029	0.746	3

F1 SDQ	exposure* PM10 (high/low)	-0.001	0.022	0.957	2	-0.007	0.024	0.769	3
F1 Internalising	exposure* PM10 (high/low)	-0.007	0.013	0.578	2	-0.005	0.014	0.74	3
F1 Externalising	exposure* PM10 (high/low)	-0.013	0.013	0.301	2	-0.012	0.014	0.386	3
F1 PHQ-9 Depression	exposure* PM10 (high/low)	-0.012	0.024	0.615	2	-0.015	0.026	0.572	3
F1 GAD-7 Anxiety	exposure* PM10 (high/low)	0.013	0.021	0.535	2	0.009	0.022	0.662	3
F1 Executive Function	exposure* PM10 (high/low)	0.004	0.002	0.04	2	0.002	0.002	0.442	3
F1 Culture Fair Intelligence Test	exposure* PM10 (high/low)	-0.001	0.012	0.948	2	-0.003	0.013	0.809	3
F1 Processing Speed	exposure* PM10 (high/low)	0.007	0.002	0.005	2	0.006	0.003	0.02	3
F1 Speech-in-noise Threshold	exposure* PM10 (high/low)	-0.006	0.019	0.761	2	-0.006	0.021	0.783	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*School Type (Independent yes/no)	-0.178	0.228	0.435	2	-0.178	0.229	0.438	3
BL Internalising	exposure*School Type	-0.075	0.134	0.578	2	-0.076	0.135	0.572	3

	(Independent yes/no)								
BL Externalising	exposure*School Type (Independent yes/no)	-0.103	0.14	0.459	2	-0.104	0.14	0.458	3
BL Executive Function	exposure*School Type (Independent yes/no)	0.036	0.026	0.161	2	0.036	0.026	0.163	3
BL Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.253	0.143	0.078	2	0.253	0.143	0.076	3
BL Processing Speed	exposure*School Type (Independent yes/no)	-0.003	0.027	0.909	2	0.008	0.026	0.765	3
BL Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	0.063	0.138	0.646	2	0.117	0.139	0.401	3
F1 SDQ	exposure*School Type (Independent yes/no)	0.141	0.231	0.542	2	0.201	0.231	0.385	3
F1 Internalising	exposure*School Type (Independent yes/no)	0.003	0.138	0.985	2	0.053	0.138	0.702	3
F1 Externalising	exposure*School Type (Independent yes/no)	0.177	0.134	0.186	2	0.192	0.134	0.152	3

F1 PHQ-9 Depression	exposure*School Type (Independent yes/no)	0.283	0.268	0.291	2	0.277	0.27	0.305	3
F1 GAD-7 Anxiety	exposure*School Type (Independent yes/no)	0.326	0.228	0.152	2	0.326	0.229	0.155	3
F1 Executive Function	exposure*School Type (Independent yes/no)	-0.007	0.022	0.769	2	-0.012	0.022	0.598	3
F1 Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.096	0.135	0.476	2	0.09	0.135	0.505	3
F1 Processing Speed	exposure*School Type (Independent yes/no)	-0.027	0.024	0.275	2	-0.019	0.024	0.433	3
F1 Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	0.03	0.186	0.872	2	0.065	0.185	0.727	3

Exposure: Lday

Outcome	Interaction	Estimate	SE	p	Model	Estimate	SE	p	Model
BL SDQ	exposure*IQ	-0.19	0.033	0	2	-0.179	0.033	0	3
BL Internalising	exposure*IQ	-0.105	0.019	0	2	-0.098	0.019	0	3
BL Externalising	exposure*IQ	-0.085	0.02	0	2	-0.082	0.02	0	3
BL Executive Function	exposure*IQ	0.063	0.004	0	2	0.062	0.004	0	3
BL Culture Fair Intelligence Test	exposure*IQ	0.909	0.012	0	2	0.899	0.012	0	3
BL Processing Speed	exposure*IQ	0.001	0.004	0.881	2	0	0.004	0.911	3
BL Speech-in-noise Threshold	exposure*IQ	-0.091	0.021	0	2	-0.091	0.021	0	3
F1 SDQ	exposure*IQ	-0.066	0.033	0.048	2	-0.063	0.033	0.06	3
F1 Internalising	exposure*IQ	-0.011	0.02	0.588	2	-0.003	0.02	0.867	3
F1 Externalising	exposure*IQ	-0.009	0.019	0.66	2	-0.013	0.019	0.496	3
F1 PHQ-9 Depression	exposure*IQ	0.04	0.036	0.268	2	0.046	0.037	0.214	3
F1 GAD-7 Anxiety	exposure*IQ	0.022	0.031	0.47	2	0.023	0.031	0.453	3
F1 Executive Function	exposure*IQ	0.013	0.003	0	2	0.012	0.003	0	3
F1 Culture Fair	exposure*IQ	0.012	0.03	0.683	2	0.018	0.03	0.544	3

Intelligence Test									
F1 Processing Speed	exposure*IQ	0.005	0.004	0.157	2	0.006	0.004	0.117	3
F1 Speech-in-noise Threshold	exposure*IQ	-0.07	0.03	0.018	2	-0.075	0.03	0.012	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*sex	0.067	0.296	0.821	2	0.093	0.306	0.761	3
BL Internalising	exposure*sex	0.165	0.171	0.336	2	0.184	0.176	0.295	3
BL Externalising	exposure*sex	-0.015	0.178	0.935	2	-0.001	0.183	0.995	3
BL Executive Function	exposure*sex	0.007	0.033	0.838	2	-0.003	0.034	0.92	3
BL Culture Fair Intelligence Test	exposure*sex	0.13	0.181	0.473	2	0.105	0.186	0.571	3
BL Processing Speed	exposure*sex	-0.027	0.034	0.413	2	-0.026	0.034	0.444	3
BL Speech-in-noise Threshold	exposure*sex	-0.131	0.178	0.464	2	-0.028	0.183	0.877	3
F1 SDQ	exposure*sex	-0.318	0.293	0.278	2	-0.18	0.301	0.55	3
F1 Internalising	exposure*sex	0.023	0.175	0.897	2	0.085	0.181	0.638	3
F1 Externalising	exposure*sex	-0.259	0.173	0.134	2	-0.209	0.178	0.238	3
F1 PHQ-9 Depression	exposure*sex	0.015	0.317	0.963	2	0.021	0.326	0.948	3

F1 GAD-7 Anxiety	exposure*sex	-0.005	0.268	0.984	2	-0.004	0.273	0.988	3
F1 Executive Function	exposure*sex	0.038	0.028	0.183	2	0.026	0.029	0.376	3
F1 Culture Fair Intelligence Test	exposure*sex	-0.174	0.168	0.3	2	-0.165	0.172	0.336	3
F1 Processing Speed	exposure*sex	0.011	0.033	0.745	2	0.018	0.034	0.601	3
F1 Speech-in-noise Threshold	exposure*sex	0.111	0.254	0.662	2	0.213	0.259	0.412	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*ethnicity2	0.128	0.426	0.764	2	0.191	0.436	0.662	3
BL SDQ	exposure*ethnicity3	-0.535	0.367	0.145	2	-0.295	0.383	0.442	3
BL SDQ	exposure*ethnicity4	-0.264	0.509	0.604	2	-0.334	0.51	0.513	3
BL SDQ	exposure*ethnicity5	1.961	1.866	0.294	2	2.2	1.864	0.238	3
BL Internalising	exposure*ethnicity2	0.298	0.241	0.217	2	0.31	0.247	0.21	3
BL Internalising	exposure*ethnicity3	0.019	0.211	0.927	2	0.067	0.22	0.761	3
BL Internalising	exposure*ethnicity4	0.003	0.299	0.992	2	-0.026	0.3	0.93	3
BL Internalising	exposure*ethnicity5	1.216	1	0.224	2	1.275	0.998	0.202	3
BL Externalising	exposure*ethnicity2	-0.115	0.251	0.647	2	-0.07	0.257	0.786	3

BL Externalising	exposure*ethnicity3	-0.481	0.22	0.029	2	-0.308	0.229	0.178	3
BL Externalising	exposure*ethnicity4	-0.241	0.312	0.441	2	-0.275	0.313	0.379	3
BL Externalising	exposure*ethnicity5	0.678	1.041	0.515	2	0.839	1.039	0.42	3
BL Executive Function	exposure*ethnicity2	0.045	0.047	0.338	2	0.081	0.048	0.096	3
BL Executive Function	exposure*ethnicity3	-0.01	0.041	0.804	2	-0.019	0.043	0.656	3
BL Executive Function	exposure*ethnicity4	-0.085	0.058	0.143	2	-0.058	0.058	0.317	3
BL Executive Function	exposure*ethnicity5	-0.049	0.188	0.796	2	-0.061	0.186	0.745	3
BL Culture Fair Intelligence Test	exposure*ethnicity2	0.324	0.254	0.203	2	0.254	0.26	0.329	3
BL Culture Fair Intelligence Test	exposure*ethnicity3	-0.197	0.224	0.381	2	-0.258	0.234	0.271	3
BL Culture Fair Intelligence Test	exposure*ethnicity4	0.414	0.316	0.19	2	0.498	0.313	0.112	3
BL Culture Fair Intelligence Test	exposure*ethnicity5	-0.917	1.084	0.398	2	-0.982	1.068	0.358	3
BL Processing Speed	exposure*ethnicity2	0.044	0.049	0.377	2	0.036	0.05	0.471	3

BL Processing Speed	exposure*ethnicity3	0.045	0.042	0.281	2	0.038	0.043	0.368	3
BL Processing Speed	exposure*ethnicity4	0.048	0.058	0.412	2	0.037	0.058	0.52	3
BL Processing Speed	exposure*ethnicity5	-0.033	0.177	0.852	2	-0.084	0.174	0.627	3
BL Speech-in-noise Threshold	exposure*ethnicity2	-0.237	0.251	0.345	2	-0.234	0.258	0.364	3
BL Speech-in-noise Threshold	exposure*ethnicity3	-0.275	0.219	0.211	2	-0.051	0.23	0.825	3
BL Speech-in-noise Threshold	exposure*ethnicity4	-0.203	0.31	0.512	2	-0.263	0.31	0.397	3
BL Speech-in-noise Threshold	exposure*ethnicity5	-0.896	1.018	0.379	2	-0.815	1.013	0.421	3
F1 SDQ	exposure*ethnicity2	0.096	0.433	0.825	2	0.017	0.44	0.97	3
F1 SDQ	exposure*ethnicity3	-0.705	0.358	0.049	2	-0.611	0.375	0.103	3
F1 SDQ	exposure*ethnicity4	-0.228	0.494	0.645	2	-0.288	0.494	0.56	3
F1 SDQ	exposure*ethnicity5	0.313	1.837	0.865	2	0.109	1.83	0.953	3
F1 Internalising	exposure*ethnicity2	0.168	0.259	0.516	2	0.135	0.264	0.61	3
F1 Internalising	exposure*ethnicity3	-0.294	0.214	0.17	2	-0.214	0.225	0.342	3
F1 Internalising	exposure*ethnicity4	-0.247	0.295	0.402	2	-0.244	0.296	0.41	3

F1 Internalising	exposure*ethnicity5	0.872	1.099	0.427	2	0.692	1.095	0.527	3
F1 Externalising	exposure*ethnicity2	0.074	0.259	0.776	2	-0.001	0.264	0.998	3
F1 Externalising	exposure*ethnicity3	-0.22	0.21	0.295	2	-0.344	0.22	0.118	3
F1 Externalising	exposure*ethnicity4	0.098	0.29	0.735	2	0.031	0.291	0.916	3
F1 Externalising	exposure*ethnicity5	-1.243	1.143	0.277	2	-1.523	1.142	0.182	3
F1 PHQ-9 Depression	exposure*ethnicity2	-0.445	0.497	0.37	2	-0.483	0.506	0.34	3
F1 PHQ-9 Depression	exposure*ethnicity3	-0.824	0.386	0.033	2	-0.783	0.399	0.05	3
F1 PHQ-9 Depression	exposure*ethnicity4	-1.11	0.536	0.039	2	-1.154	0.54	0.033	3
F1 PHQ-9 Depression	exposure*ethnicity5	0.541	2.034	0.79	2	0.506	2.045	0.805	3
F1 GAD-7 Anxiety	exposure*ethnicity2	0.202	0.412	0.624	2	0.224	0.418	0.592	3
F1 GAD-7 Anxiety	exposure*ethnicity3	-0.501	0.328	0.127	2	-0.463	0.333	0.165	3
F1 GAD-7 Anxiety	exposure*ethnicity4	-0.443	0.452	0.327	2	-0.389	0.454	0.391	3
F1 GAD-7 Anxiety	exposure*ethnicity5	2.999	1.737	0.084	2	2.917	1.743	0.094	3
F1 Executive Function	exposure*ethnicity2	-0.026	0.042	0.536	2	-0.023	0.043	0.585	3
F1 Executive Function	exposure*ethnicity3	-0.011	0.035	0.755	2	-0.011	0.037	0.773	3
F1 Executive Function	exposure*ethnicity4	0.06	0.049	0.219	2	0.062	0.048	0.201	3
F1 Executive Function	exposure*ethnicity5	-0.017	0.183	0.927	2	0.026	0.181	0.888	3

F1 Culture Fair Intelligence Test	exposure*ethnicity2	-0.306	0.244	0.21	2	-0.324	0.249	0.194	3
F1 Culture Fair Intelligence Test	exposure*ethnicity3	-0.156	0.206	0.448	2	-0.104	0.214	0.627	3
F1 Culture Fair Intelligence Test	exposure*ethnicity4	-0.833	0.285	0.003	2	-0.814	0.286	0.005	3
F1 Culture Fair Intelligence Test	exposure*ethnicity5	-1.76	1.156	0.128	2	-1.71	1.162	0.141	3
F1 Processing Speed	exposure*ethnicity2	0.016	0.054	0.763	2	0.024	0.054	0.654	3
F1 Processing Speed	exposure*ethnicity3	0.014	0.04	0.733	2	0.033	0.041	0.428	3
F1 Processing Speed	exposure*ethnicity4	0.071	0.059	0.228	2	0.082	0.058	0.154	3
F1 Processing Speed	exposure*ethnicity5	0.13	0.325	0.69	2	0.104	0.319	0.744	3
F1 Speech-in-noise Threshold	exposure*ethnicity2	0.506	0.381	0.184	2	0.18	0.389	0.644	3
F1 Speech-in-noise Threshold	exposure*ethnicity3	0.233	0.304	0.443	2	0.212	0.318	0.506	3

F1 Speech-in-noise Threshold	exposure*ethnicity4	0.373	0.434	0.391	2	0.372	0.436	0.394	3
F1 Speech-in-noise Threshold	exposure*ethnicity5	4.226	1.73	0.015	2	4.097	1.712	0.017	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*religion	-0.094	0.045	0.039	2	-0.093	0.046	0.041	3
BL Internalising	exposure*religion	-0.06	0.027	0.024	2	-0.059	0.027	0.029	3
BL Externalising	exposure*religion	-0.033	0.028	0.238	2	-0.034	0.028	0.226	3
BL Executive Function	exposure*religion	-0.006	0.005	0.231	2	-0.009	0.005	0.081	3
BL Culture Fair Intelligence Test	exposure*religion	-0.062	0.029	0.032	2	-0.063	0.029	0.03	3
BL Processing Speed	exposure*religion	0.007	0.005	0.165	2	0.006	0.005	0.201	3
BL Speech-in-noise Threshold	exposure*religion	0.034	0.027	0.21	2	0.039	0.028	0.162	3
F1 SDQ	exposure*religion	-0.145	0.044	0.001	2	-0.139	0.044	0.002	3
F1 Internalising	exposure*religion	-0.092	0.026	0.001	2	-0.086	0.027	0.001	3
F1 Externalising	exposure*religion	-0.049	0.025	0.054	2	-0.046	0.026	0.076	3
F1 PHQ-9 Depression	exposure*religion	-0.162	0.048	0.001	2	-0.165	0.048	0.001	3
F1 GAD-7 Anxiety	exposure*religion	-0.134	0.041	0.001	2	-0.138	0.042	0.001	3

F1 Executive Function	exposure*religion	0	0.004	0.917	2	-0.002	0.004	0.711	3
F1 Culture Fair Intelligence Test	exposure*religion	-0.036	0.026	0.171	2	-0.036	0.027	0.18	3
F1 Processing Speed	exposure*religion	0.006	0.004	0.165	2	0.006	0.004	0.184	3
F1 Speech-in-noise Threshold	exposure*religion	0.011	0.034	0.735	2	0.019	0.034	0.583	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*religion	-0.094	0.045	0.039	2	-0.093	0.046	0.041	3
BL Internalising	exposure*religion	-0.06	0.027	0.024	2	-0.059	0.027	0.029	3
BL Externalising	exposure*religion	-0.033	0.028	0.238	2	-0.034	0.028	0.226	3
BL Executive Function	exposure*religion	-0.006	0.005	0.231	2	-0.009	0.005	0.081	3
BL Culture Fair Intelligence Test	exposure*religion	-0.062	0.029	0.032	2	-0.063	0.029	0.03	3
BL Processing Speed	exposure*religion	0.007	0.005	0.165	2	0.006	0.005	0.201	3
BL Speech-in-noise Threshold	exposure*religion	0.034	0.027	0.21	2	0.039	0.028	0.162	3
F1 SDQ	exposure*religion	-0.145	0.044	0.001	2	-0.139	0.044	0.002	3
F1 Internalising	exposure*religion	-0.092	0.026	0.001	2	-0.086	0.027	0.001	3

F1 Externalising	exposure*religion	-0.049	0.025	0.054	2	-0.046	0.026	0.076	3
F1 PHQ-9 Depression	exposure*religion	-0.162	0.048	0.001	2	-0.165	0.048	0.001	3
F1 GAD-7 Anxiety	exposure*religion	-0.134	0.041	0.001	2	-0.138	0.042	0.001	3
F1 Executive Function	exposure*religion	0	0.004	0.917	2	-0.002	0.004	0.711	3
F1 Culture Fair Intelligence Test	exposure*religion	-0.036	0.026	0.171	2	-0.036	0.027	0.18	3
F1 Processing Speed	exposure*religion	0.006	0.004	0.165	2	0.006	0.004	0.184	3
F1 Speech-in-noise Threshold	exposure*religion	0.011	0.034	0.735	2	0.019	0.034	0.583	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure:tenBB1	0.035	0.032	0.281	2	0.015	0.035	0.672	3
BL Internalising	exposure:tenBB1	-0.001	0.019	0.963	2	-0.001	0.021	0.951	3
BL Externalising	exposure:tenBB1	0.036	0.02	0.071	2	0.017	0.021	0.424	3
BL Executive Function	exposure:tenBB1	0.004	0.004	0.267	2	0.006	0.004	0.141	3
BL Culture Fair Intelligence Test	exposure:tenBB1	0.015	0.02	0.467	2	0.026	0.022	0.244	3
BL Processing Speed	exposure:tenBB1	-0.001	0.004	0.705	2	-0.006	0.004	0.173	3

BL Speech-in-noise Threshold	exposure:tenBB1	0.028	0.02	0.158	2	0.008	0.021	0.723	3
F1 SDQ	exposure* PM10 (high/low)	-0.004	0.032	0.888	2	-0.014	0.035	0.689	3
F1 Internalising	exposure* PM10 (high/low)	-0.01	0.019	0.614	2	-0.007	0.021	0.742	3
F1 Externalising	exposure* PM10 (high/low)	-0.023	0.019	0.212	2	-0.021	0.02	0.305	3
F1 PHQ-9 Depression	exposure* PM10 (high/low)	-0.021	0.035	0.558	2	-0.021	0.037	0.569	3
F1 GAD-7 Anxiety	exposure* PM10 (high/low)	0.026	0.03	0.375	2	0.021	0.031	0.498	3
F1 Executive Function	exposure* PM10 (high/low)	0.006	0.003	0.034	2	0.002	0.003	0.465	3
F1 Culture Fair Intelligence Test	exposure* PM10 (high/low)	-0.001	0.018	0.945	2	-0.006	0.019	0.755	3
F1 Processing Speed	exposure* PM10 (high/low)	0.01	0.004	0.007	2	0.009	0.004	0.018	3
F1 Speech-in-noise Threshold	exposure* PM10 (high/low)	-0.009	0.027	0.74	2	-0.009	0.029	0.746	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*School Type	-0.375	0.417	0.369	2	-0.559	0.529	0.292	3

	(Independent yes/no)								
BL Internalising	exposure*School Type (Independent yes/no)	-0.27	0.245	0.27	2	-0.253	0.303	0.405	3
BL Externalising	exposure*School Type (Independent yes/no)	-0.104	0.256	0.683	2	-0.294	0.32	0.36	3
BL Executive Function	exposure*School Type (Independent yes/no)	0.096	0.046	0.037	2	0.11	0.061	0.071	3
BL Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.376	0.267	0.158	2	0.537	0.372	0.149	3
BL Processing Speed	exposure*School Type (Independent yes/no)	-0.021	0.052	0.681	2	0.052	0.071	0.466	3
BL Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	0.316	0.242	0.192	2	0.311	0.306	0.31	3
F1 SDQ	exposure*School Type (Independent yes/no)	0.537	0.396	0.175	2	0.487	0.504	0.336	3
F1 Internalising	exposure*School Type (Independent yes/no)	0.022	0.237	0.926	2	0.064	0.307	0.836	3

F1 Externalising	exposure*School Type (Independent yes/no)	0.67	0.238	0.005	2	0.632	0.297	0.035	3
F1 PHQ-9 Depression	exposure*School Type (Independent yes/no)	0.468	0.619	0.45	2	0.392	0.689	0.571	3
F1 GAD-7 Anxiety	exposure*School Type (Independent yes/no)	0.219	0.523	0.676	2	0.201	0.555	0.718	3
F1 Executive Function	exposure*School Type (Independent yes/no)	-0.033	0.038	0.392	2	-0.023	0.051	0.651	3
F1 Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	0.197	0.241	0.415	2	0.304	0.288	0.293	3
F1 Processing Speed	exposure*School Type (Independent yes/no)	0.048	0.046	0.298	2	-0.034	0.063	0.593	3
F1 Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	-0.222	0.314	0.48	2	-0.259	0.39	0.509	3

Exposure: Ozone

Outcome	Interaction	Estimate	SE	p	Model	Estimate	SE	p	Model
BL SDQ	exposure*IQ	-0.215	0.036	0	2	-0.2	0.036	0	3
BL Internalising	exposure*IQ	-0.119	0.021	0	2	-0.111	0.021	0	3
BL Externalising	exposure*IQ	-0.097	0.022	0	2	-0.091	0.022	0	3
BL Executive Function	exposure*IQ	0.069	0.004	0	2	0.069	0.004	0	3
BL Culture Fair Intelligence Test	exposure*IQ	0.988	0.013	0	2	0.979	0.013	0	3
BL Processing Speed	exposure*IQ	0.001	0.004	0.745	2	0.001	0.004	0.774	3
BL Speech-in-noise Threshold	exposure*IQ	-0.11	0.022	0	2	-0.109	0.022	0	3
F1 SDQ	exposure*IQ	-0.07	0.037	0.057	2	-0.067	0.037	0.07	3
F1 Internalising	exposure*IQ	-0.008	0.022	0.714	2	-0.001	0.022	0.973	3
F1 Externalising	exposure*IQ	-0.009	0.021	0.689	2	-0.015	0.021	0.49	3
F1 PHQ-9 Depression	exposure*IQ	0.046	0.041	0.255	2	0.052	0.041	0.204	3
F1 GAD-7 Anxiety	exposure*IQ	0.026	0.034	0.444	2	0.028	0.035	0.422	3
F1 Executive Function	exposure*IQ	0.013	0.004	0	2	0.013	0.004	0.001	3
F1 Culture Fair	exposure*IQ	0.028	0.033	0.401	2	0.034	0.033	0.301	3

Intelligence Test									
F1 Processing Speed	exposure*IQ	0.006	0.004	0.173	2	0.006	0.004	0.145	3
F1 Speech-in-noise Threshold	exposure*IQ	-0.073	0.033	0.028	2	-0.083	0.033	0.013	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*sex	0.187	0.258	0.469	2	0.22	0.275	0.424	3
BL Internalising	exposure*sex	0.125	0.152	0.41	2	0.086	0.161	0.593	3
BL Externalising	exposure*sex	0.062	0.158	0.695	2	0.127	0.167	0.447	3
BL Executive Function	exposure*sex	-0.039	0.03	0.194	2	-0.007	0.032	0.836	3
BL Culture Fair Intelligence Test	exposure*sex	-0.435	0.162	0.007	2	-0.357	0.173	0.039	3
BL Processing Speed	exposure*sex	-0.054	0.031	0.076	2	-0.078	0.032	0.015	3
BL Speech-in-noise Threshold	exposure*sex	0.185	0.159	0.244	2	0.047	0.169	0.781	3
F1 SDQ	exposure*sex	0.238	0.258	0.356	2	0.11	0.273	0.689	3
F1 Internalising	exposure*sex	0.085	0.154	0.582	2	-0.071	0.164	0.664	3
F1 Externalising	exposure*sex	-0.032	0.151	0.831	2	-0.032	0.16	0.844	3
F1 PHQ-9 Depression	exposure*sex	0.053	0.303	0.862	2	0.093	0.311	0.765	3

F1 GAD-7 Anxiety	exposure*sex	0.266	0.257	0.3	2	0.254	0.261	0.332	3
F1 Executive Function	exposure*sex	-0.018	0.025	0.463	2	-0.008	0.026	0.753	3
F1 Culture Fair Intelligence Test	exposure*sex	0.048	0.15	0.748	2	0.084	0.157	0.59	3
F1 Processing Speed	exposure*sex	-0.016	0.029	0.574	2	-0.016	0.03	0.599	3
F1 Speech-in-noise Threshold	exposure*sex	0.165	0.225	0.463	2	0.118	0.237	0.62	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*ethnicity2	-0.273	0.399	0.494	2	-0.306	0.41	0.456	3
BL SDQ	exposure*ethnicity3	0.168	0.312	0.59	2	0.173	0.328	0.597	3
BL SDQ	exposure*ethnicity4	-0.004	0.454	0.993	2	0.053	0.456	0.907	3
BL SDQ	exposure*ethnicity5	1.231	1.373	0.37	2	1.289	1.374	0.348	3
BL Internalising	exposure*ethnicity2	-0.352	0.234	0.133	2	-0.355	0.241	0.141	3
BL Internalising	exposure*ethnicity3	-0.137	0.183	0.456	2	-0.115	0.193	0.552	3
BL Internalising	exposure*ethnicity4	-0.12	0.267	0.653	2	-0.075	0.269	0.78	3
BL Internalising	exposure*ethnicity5	-0.385	0.807	0.634	2	-0.353	0.809	0.663	3
BL Externalising	exposure*ethnicity2	0.079	0.244	0.746	2	0.049	0.251	0.844	3

BL Externalising	exposure*ethnicity3	0.305	0.191	0.11	2	0.291	0.2	0.145	3
BL Externalising	exposure*ethnicity4	0.116	0.278	0.677	2	0.12	0.279	0.668	3
BL Externalising	exposure*ethnicity5	1.616	0.84	0.055	2	1.637	0.841	0.052	3
BL Executive Function	exposure*ethnicity2	0.025	0.046	0.586	2	-0.017	0.047	0.712	3
BL Executive Function	exposure*ethnicity3	0.077	0.037	0.036	2	0.033	0.038	0.39	3
BL Executive Function	exposure*ethnicity4	0.049	0.052	0.346	2	0.023	0.052	0.657	3
BL Executive Function	exposure*ethnicity5	0.1	0.154	0.517	2	0.064	0.153	0.674	3
BL Culture Fair Intelligence Test	exposure*ethnicity2	0.323	0.25	0.196	2	0.235	0.257	0.361	3
BL Culture Fair Intelligence Test	exposure*ethnicity3	0.281	0.197	0.153	2	0.062	0.207	0.766	3
BL Culture Fair Intelligence Test	exposure*ethnicity4	0.075	0.282	0.79	2	-0.003	0.282	0.991	3
BL Culture Fair Intelligence Test	exposure*ethnicity5	-0.381	0.876	0.664	2	-0.405	0.869	0.641	3
BL Processing Speed	exposure*ethnicity2	-0.079	0.049	0.107	2	-0.072	0.05	0.147	3

BL Processing Speed	exposure*ethnicity3	-0.019	0.037	0.603	2	0.001	0.038	0.985	3
BL Processing Speed	exposure*ethnicity4	-0.005	0.057	0.929	2	0.001	0.057	0.99	3
BL Processing Speed	exposure*ethnicity5	0.036	0.188	0.849	2	0.074	0.186	0.689	3
BL Speech-in-noise Threshold	exposure*ethnicity2	0.043	0.247	0.862	2	0.055	0.253	0.828	3
BL Speech-in-noise Threshold	exposure*ethnicity3	-0.058	0.189	0.761	2	-0.195	0.199	0.328	3
BL Speech-in-noise Threshold	exposure*ethnicity4	-0.195	0.281	0.488	2	-0.202	0.281	0.471	3
BL Speech-in-noise Threshold	exposure*ethnicity5	0.38	0.895	0.671	2	0.405	0.893	0.65	3
F1 SDQ	exposure*ethnicity2	-0.417	0.413	0.313	2	-0.33	0.423	0.436	3
F1 SDQ	exposure*ethnicity3	0.597	0.313	0.057	2	0.771	0.324	0.017	3
F1 SDQ	exposure*ethnicity4	0.472	0.435	0.278	2	0.438	0.436	0.314	3
F1 SDQ	exposure*ethnicity5	-0.458	1.386	0.741	2	-0.045	1.382	0.974	3
F1 Internalising	exposure*ethnicity2	-0.355	0.247	0.151	2	-0.305	0.254	0.23	3
F1 Internalising	exposure*ethnicity3	0.282	0.187	0.132	2	0.345	0.195	0.076	3
F1 Internalising	exposure*ethnicity4	0.138	0.26	0.596	2	0.138	0.261	0.598	3

F1 Internalising	exposure*ethnicity5	0.269	0.829	0.745	2	0.472	0.829	0.569	3
F1 Externalising	exposure*ethnicity2	-0.024	0.244	0.922	2	0.066	0.251	0.793	3
F1 Externalising	exposure*ethnicity3	0.248	0.183	0.175	2	0.405	0.19	0.033	3
F1 Externalising	exposure*ethnicity4	0.192	0.26	0.459	2	0.163	0.26	0.53	3
F1 Externalising	exposure*ethnicity5	-1.145	0.793	0.149	2	-0.889	0.791	0.261	3
F1 PHQ-9 Depression	exposure*ethnicity2	-0.394	0.484	0.417	2	-0.361	0.497	0.467	3
F1 PHQ-9 Depression	exposure*ethnicity3	0.64	0.361	0.076	2	0.694	0.37	0.061	3
F1 PHQ-9 Depression	exposure*ethnicity4	-0.169	0.535	0.752	2	-0.2	0.54	0.711	3
F1 PHQ-9 Depression	exposure*ethnicity5	1.853	1.385	0.181	2	1.955	1.392	0.16	3
F1 GAD-7 Anxiety	exposure*ethnicity2	-0.262	0.41	0.523	2	-0.243	0.418	0.562	3
F1 GAD-7 Anxiety	exposure*ethnicity3	0.548	0.307	0.074	2	0.561	0.312	0.073	3
F1 GAD-7 Anxiety	exposure*ethnicity4	-0.512	0.446	0.251	2	-0.489	0.449	0.276	3
F1 GAD-7 Anxiety	exposure*ethnicity5	0.517	1.183	0.662	2	0.625	1.186	0.598	3
F1 Executive Function	exposure*ethnicity2	0.106	0.039	0.007	2	0.099	0.04	0.013	3
F1 Executive Function	exposure*ethnicity3	0.021	0.031	0.492	2	0.023	0.032	0.478	3
F1 Executive Function	exposure*ethnicity4	0.007	0.044	0.877	2	0.002	0.044	0.97	3
F1 Executive Function	exposure*ethnicity5	0.318	0.13	0.015	2	0.293	0.13	0.024	3

F1 Culture Fair Intelligence Test	exposure*ethnicity2	0.442	0.236	0.062	2	0.391	0.243	0.107	3
F1 Culture Fair Intelligence Test	exposure*ethnicity3	0.251	0.18	0.163	2	0.139	0.186	0.455	3
F1 Culture Fair Intelligence Test	exposure*ethnicity4	0.379	0.259	0.143	2	0.294	0.261	0.26	3
F1 Culture Fair Intelligence Test	exposure*ethnicity5	1.468	0.77	0.057	2	1.348	0.772	0.081	3
F1 Processing Speed	exposure*ethnicity2	-0.032	0.05	0.52	2	0.016	0.051	0.758	3
F1 Processing Speed	exposure*ethnicity3	-0.09	0.034	0.009	2	-0.059	0.035	0.094	3
F1 Processing Speed	exposure*ethnicity4	-0.076	0.056	0.174	2	-0.052	0.055	0.346	3
F1 Processing Speed	exposure*ethnicity5	-0.114	0.365	0.755	2	-0.036	0.36	0.921	3
F1 Speech-in-noise Threshold	exposure*ethnicity2	-0.631	0.359	0.079	2	-0.419	0.365	0.252	3
F1 Speech-in-noise Threshold	exposure*ethnicity3	-0.134	0.256	0.6	2	-0.09	0.262	0.732	3

F1 Speech-in-noise Threshold	exposure*ethnicity4	0.065	0.371	0.86	2	0.078	0.371	0.834	3
F1 Speech-in-noise Threshold	exposure*ethnicity5	-0.573	0.966	0.553	2	-0.399	0.964	0.679	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*religion	-0.099	0.049	0.046	2	-0.096	0.05	0.054	3
BL Internalising	exposure*religion	-0.068	0.029	0.02	2	-0.066	0.029	0.025	3
BL Externalising	exposure*religion	-0.031	0.031	0.31	2	-0.03	0.031	0.329	3
BL Executive Function	exposure*religion	-0.006	0.006	0.334	2	-0.009	0.006	0.12	3
BL Culture Fair Intelligence Test	exposure*religion	-0.063	0.032	0.046	2	-0.064	0.032	0.043	3
BL Processing Speed	exposure*religion	0.006	0.006	0.282	2	0.006	0.006	0.305	3
BL Speech-in-noise Threshold	exposure*religion	0.035	0.03	0.236	2	0.038	0.03	0.2	3
F1 SDQ	exposure*religion	-0.148	0.048	0.002	2	-0.141	0.048	0.003	3
F1 Internalising	exposure*religion	-0.096	0.029	0.001	2	-0.091	0.029	0.002	3
F1 Externalising	exposure*religion	-0.05	0.028	0.074	2	-0.046	0.028	0.102	3
F1 PHQ-9 Depression	exposure*religion	-0.167	0.053	0.002	2	-0.17	0.054	0.002	3
F1 GAD-7 Anxiety	exposure*religion	-0.132	0.046	0.004	2	-0.136	0.046	0.003	3

F1 Executive Function	exposure*religion	0.001	0.005	0.909	2	-0.001	0.005	0.853	3
F1 Culture Fair Intelligence Test	exposure*religion	-0.03	0.029	0.295	2	-0.029	0.029	0.32	3
F1 Processing Speed	exposure*religion	0.007	0.005	0.134	2	0.007	0.005	0.166	3
F1 Speech-in-noise Threshold	exposure*religion	0.004	0.037	0.914	2	0.01	0.037	0.78	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure* BL Lden (high/low)	0.055	0.034	0.108	2	0.05	0.035	0.149	3
BL Internalising	exposure* BL Lden (high/low)	0.021	0.02	0.296	2	0.018	0.02	0.381	3
BL Externalising	exposure* BL Lden (high/low)	0.034	0.021	0.105	2	0.032	0.021	0.13	3
BL Executive Function	exposure* BL Lden (high/low)	-0.002	0.004	0.553	2	-0.001	0.004	0.781	3
BL Culture Fair Intelligence Test	exposure* BL Lden (high/low)	0.02	0.022	0.353	2	0.026	0.022	0.231	3
BL Processing Speed	exposure* BL Lden (high/low)	0.006	0.004	0.149	2	0.004	0.004	0.373	3
BL Speech-in-noise Threshold	exposure* BL Lden (high/low)	0.008	0.021	0.7	2	0.002	0.021	0.932	3

F1 SDQ	exposure: F1 Lden (high/low)	0.023	0.034	0.512	2	0.007	0.035	0.846	3
F1 Internalising	exposure: F1 Lden (high/low)	0.003	0.021	0.873	2	-0.002	0.021	0.909	3
F1 Externalising	exposure: F1 Lden (high/low)	0.011	0.02	0.599	2	0.002	0.02	0.913	3
F1 PHQ-9 Depression	exposure: F1 Lden (high/low)	-0.002	0.039	0.965	2	-0.007	0.039	0.864	3
F1 GAD-7 Anxiety	exposure: F1 Lden (high/low)	0.03	0.033	0.354	2	0.027	0.033	0.412	3
F1 Executive Function	exposure: F1 Lden (high/low)	-0.005	0.003	0.159	2	-0.004	0.003	0.258	3
F1 Culture Fair Intelligence Test	exposure: F1 Lden (high/low)	-0.034	0.02	0.087	2	-0.033	0.02	0.095	3
F1 Processing Speed	exposure: F1 Lden (high/low)	-0.001	0.004	0.801	2	-0.003	0.004	0.492	3
F1 Speech-in-noise Threshold	exposure: F1 Lden (high/low)	0.047	0.03	0.124	2	0.039	0.03	0.204	3
Outcome	**Interaction**	**Estimate**	**SE**	**p**	**Model**	**Estimate**	**SE**	**p**	**Model**
BL SDQ	exposure*School Type (Independent yes/no)	-0.425	0.258	0.099	2	-0.4	0.303	0.188	3
BL Internalising	exposure*School Type	-0.019	0.152	0.9	2	-0.001	0.177	0.994	3

	(Independent yes/no)								
BL Externalising	exposure*School Type (Independent yes/no)	-0.406	0.158	0.01	2	-0.403	0.182	0.027	3
BL Executive Function	exposure*School Type (Independent yes/no)	-0.084	0.029	0.004	2	-0.069	0.035	0.052	3
BL Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	-0.453	0.163	0.006	2	-0.387	0.202	0.056	3
BL Processing Speed	exposure*School Type (Independent yes/no)	0.03	0.031	0.33	2	0.008	0.036	0.829	3
BL Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	0.027	0.156	0.862	2	0.022	0.183	0.904	3
F1 SDQ	exposure*School Type (Independent yes/no)	-0.005	0.257	0.984	2	-0.275	0.301	0.362	3
F1 Internalising	exposure*School Type (Independent yes/no)	0.201	0.154	0.191	2	0.075	0.18	0.679	3
F1 Externalising	exposure*School Type (Independent yes/no)	-0.142	0.152	0.351	2	-0.291	0.177	0.101	3

F1 PHQ-9 Depression	exposure*School Type (Independent yes/no)	-0.341	0.329	0.3	2	-0.388	0.352	0.271	3
F1 GAD-7 Anxiety	exposure*School Type (Independent yes/no)	-0.499	0.279	0.074	2	-0.502	0.29	0.085	3
F1 Executive Function	exposure*School Type (Independent yes/no)	-0.023	0.025	0.346	2	-0.018	0.028	0.533	3
F1 Culture Fair Intelligence Test	exposure*School Type (Independent yes/no)	-0.254	0.151	0.093	2	-0.184	0.169	0.276	3
F1 Processing Speed	exposure*School Type (Independent yes/no)	0.002	0.028	0.944	2	-0.035	0.033	0.291	3
F1 Speech-in-noise Threshold	exposure*School Type (Independent yes/no)	0.158	0.217	0.466	2	-0.001	0.248	0.996	3

CAPE Psychosis risk interaction effects

Air pollution exposure

Model	Interaction term	Exposure	Effect	p	Exposure	Effect	p	Exposure	Effect	p	Exposure	Effect	p
2	IQ	PM2.5	-0.11	0.02	PM10	-0.09	0.02	NO2	-0.2	0.02	Ozone	-0.2	0.02
3	IQ	PM2.5	-0.11	0.02	PM10	-0.08	0.02	NO2	-0.2	0.02	Ozone	-0.2	0.03
2	sex	PM2.5	0.27	0.68	PM10	-0.21	0.73	NO2	0.13	0.85	Ozone	-2.2	<0.01
3	sex	PM2.5	0.27	0.68	PM10	-0.21	0.73	NO2	0.12	0.86	Ozone	-2.3	<0.01
2	ethnicity	PM2.5	-0.84	0.47	PM10	-0.58	0.57	NO2	0.15	0.89	Ozone	-0.09	0.94
2	ethnicity	PM2.5	-0.32	0.65	PM10	-0.3	0.65	NO2	0.65	0.38	Ozone	-0.31	0.67
2	ethnicity	PM2.5	-115.7	0.99	PM10	-115.92	0.99	NO2	-5.73	0.32	Ozone	-1.07	0.66
2	ethnicity	PM2.5	-122.8	>0.99	PM10	-31.14	>0.99	NO2	-63.3	0.99	Ozone	-3.68	0.59
3	ethnicity	PM2.5	-0.84	0.47	PM10	-0.58	0.57	NO2	0.15	0.89	Ozone	-0.09	-0.94
3	ethnicity	PM2.5	-0.32	0.65	PM10	-0.3	0.66	NO2	0.65	0.38	Ozone	-0.31	0.67
3	ethnicity	PM2.5	-58.01	0.85	PM10	-66.1	0.64	NO2	-5.73	0.32	Ozone	-1.07	0.66
3	ethnicity	PM2.5	-67.58	0.92	PM10	-17.38	0.73	NO2	-71.52	>0.99	Ozone	-3.68	0.59
2	religion	PM2.5	0.01	0.9	PM10	0.85	0.88	NO2	0.02	0.86	Ozone	-0.03	0.85
3	religion	PM2.5	0.01	0.9	PM10	0.85	0.88	NO2	0.02	0.86	Ozone	-0.03	0.85
2	noise/poll	PM2.5	0.05	0.31	PM10	0.04	0.23	NO2	0.11	0.18	Ozone	0.14	0.11
3	noise/poll	PM2.5	0.05	0.31	PM10	0.04	0.23	NO2	0.11	0.18	Ozone	0.14	0.11
2	school type	PM2.5	-0.27	0.68	PM10	-0.41	0.5	NO2	-0.17	0.79	Ozone	-1.12	0.13
3	school type	PM2.5	-0.27	0.68	PM10	-0.41	0.5	NO2	-0.2	0.77	Ozone	-1.16	0.13

Noise exposure

Model	Interaction term	Exposure	Exposure	Effect	p	Exposure	Effect	p
2	IQ	PM2.5	Lden	-0.11	0.03	Lday	-0.17	0.03
3	IQ	PM2.5	Lden	-0.11	0.04	Lday	-0.17	0.04

2	sex	PM2.5	Lden	-0.78	0.09	Lday	-0.93	0.3
3	sex	PM2.5	Lden	-0.78	0.09	Lday	-0.95	0.3
2	ethnicity	PM2.5	Lden	0.42	0.59	Lday	1.17	0.45
2	ethnicity	PM2.5	Lden	0.99	0.05	Lday	1.45	0.19
2	ethnicity	PM2.5	Lden	7.66	0.34	Lday	3.28	0.32
2	ethnicity	PM2.5	Lden	-51.8	0.99	Lday	12.24	0.65
3	ethnicity	PM2.5	Lden	0.42	0.59	Lday	1.22	0.45
3	ethnicity	PM2.5	Lden	0.99	0.05	Lday	1.36	0.2
3	ethnicity	PM2.5	Lden	7.66	-0.34	Lday	3.37	0.32
3	ethnicity	PM2.5	Lden	-249.91	0.86	Lday	12.51	0.63
2	religion	PM2.5	Lden	0.03	0.77	Lday	0.03	0.79
3	Religion	PM2.5	Lden	0.03	0.77	Lday	0.03	0.79
2	noise/poll	PM2.5	Lden	0.12	0.1	Lday	0.08	0.13
3	noise/poll	PM2.5	Lden	0.12	0.1	Lday	0.08	0.13
2	school type	PM2.5	Lden	-0.4	0.35	Lday	-0.17	0.84
3	school type	PM2.5	Lden	-0.4	0.36	Lday	-0.21	0.8

XIII: Results for associations between exposure to air pollution or noise and psychological outcomes in the SCAMP cohort, adjusted for indices of multiple deprivation (IMD-19)

Air pollution exposure

Outcome	Model	PM2.5 Estimate	SE	p	PM10 Estimate	SE	p	NO2 Estimate	SE	p	Ozone Estimate	SE	p
BL SDQ	1	-0.172	0.094	0.068	0.302	0.099	0.002	0.159	0.118	0.178	-0.165	0.13	0.204
BL SDQ	2	-0.13	0.098	0.184	0.262	0.102	0.01	0.154	0.119	0.196	-0.158	0.132	0.23
BL SDQ	3	-0.104	0.124	0.403	0.251	0.119	0.035	0.262	0.143	0.068	-0.349	0.17	0.041
BL Internalising	1	-0.032	0.055	0.557	0.149	0.058	0.011	0.067	0.069	0.33	-0.052	0.076	0.496
BL Internalising	2	-0.06	0.058	0.297	0.106	0.06	0.076	0.03	0.07	0.663	-0.005	0.077	0.948
BL Internalising	3	-0.019	0.072	0.797	0.128	0.07	0.067	0.129	0.084	0.128	-0.15	0.1	0.134
BL Externalising	1	-0.14	0.058	0.016	0.153	0.062	0.013	0.092	0.073	0.207	-0.114	0.08	0.158
BL Externalising	2	-0.07	0.06	0.244	0.156	0.062	0.013	0.123	0.073	0.091	-0.153	0.081	0.058
BL Externalising	3	-0.092	0.076	0.225	0.124	0.072	0.088	0.129	0.087	0.138	-0.187	0.102	0.067
BL Executive Function	1	0.015	0.011	0.182	-0.035	0.012	0.004	-0.002	0.014	0.904	-0.003	0.016	0.867
BL Executive Function	2	0.003	0.011	0.792	-0.016	0.012	0.185	0.007	0.014	0.624	-0.017	0.015	0.254
BL Executive Function	3	0.004	0.015	0.805	-0.005	0.014	0.705	0.003	0.017	0.848	-0.013	0.02	0.506

BL Culture Fair Intelligence Test	1	-0.059	0.059	0.316	-0.121	0.063	0.055	-0.02	0.074	0.79	0.026	0.083	0.755
BL Culture Fair Intelligence Test	2	-0.079	0.06	0.19	-0.049	0.064	0.438	0.001	0.074	0.993	-0.006	0.082	0.942
BL Culture Fair Intelligence Test	3	-0.014	0.083	0.862	0.037	0.076	0.625	0.038	0.092	0.675	-0.034	0.112	0.759
BL Processing Speed	1	0.001	0.01	0.955	0.015	0.011	0.178	0.003	0.014	0.809	-0.008	0.015	0.604
BL Processing Speed	2	-0.003	0.011	0.787	0.013	0.012	0.286	0.003	0.014	0.843	-0.007	0.015	0.625
BL Processing Speed	3	-0.013	0.014	0.37	-0.002	0.014	0.867	-0.002	0.017	0.919	-0.004	0.02	0.83
BL Speech-in-noise Threshold	1	-0.121	0.062	0.049	0.166	0.061	0.006	0.06	0.073	0.411	-0.085	0.08	0.291
BL Speech-in-noise Threshold	2	-0.003	0.011	0.787	0.013	0.012	0.286	0.003	0.014	0.843	-0.007	0.015	0.625
BL Speech-in-noise Threshold	3	-0.013	0.014	0.37	-0.002	0.014	0.867	-0.002	0.017	0.919	-0.004	0.02	0.83
F1 SDQ	1	0.244	0.125	0.051	0.265	0.118	0.025	0.01	0.121	0.937	0.001	0.131	0.993
F1 SDQ	2	0.229	0.13	0.077	-0.034	0.111	0.761	-0.169	0.107	0.116	0.214	0.119	0.072
F1 SDQ	3	0.238	0.166	0.153	-0.042	0.135	0.754	-0.095	0.124	0.442	0.149	0.147	0.31
F1 Internalising	1	0.046	0.075	0.54	0.079	0.071	0.266	-0.044	0.073	0.543	0.048	0.079	0.541

F1 Internalising	2	-0.002	0.078	0.978	-0.071	0.067	0.286	-0.125	0.065	0.055	0.132	0.072	0.068
F1 Internalising	3	0.004	0.101	0.97	-0.115	0.081	0.157	-0.13	0.074	0.08	0.15	0.087	0.085
F1 Externalising	1	0.198	0.079	0.013	0.186	0.075	0.013	0.054	0.077	0.484	-0.047	0.083	0.573
F1 Externalising	2	0.077	0.076	0.311	0.04	0.071	0.576	-0.044	0.069	0.525	0.084	0.076	0.272
F1 Externalising	3	0.109	0.098	0.266	0.072	0.087	0.408	0.03	0.08	0.705	0.001	0.096	0.99
F1 PHQ-9 Depression	1	0.104	0.133	0.437	0.124	0.126	0.324	0.002	0.133	0.991	-0.076	0.149	0.612
F1 PHQ-9 Depression	2	-0.1	0.144	0.485	-0.043	0.133	0.746	-0.132	0.133	0.321	0.092	0.153	0.548
F1 PHQ-9 Depression	3	-0.104	0.174	0.551	-0.054	0.156	0.728	-0.119	0.147	0.419	0.067	0.177	0.706
F1 GAD-7 Anxiety	1	0.168	0.114	0.142	0.183	0.108	0.089	0.114	0.113	0.314	-0.182	0.128	0.154
F1 GAD-7 Anxiety	2	0.051	0.122	0.676	0.097	0.113	0.387	0.043	0.113	0.702	-0.098	0.13	0.451
F1 GAD-7 Anxiety	3	0.038	0.143	0.788	0.08	0.128	0.534	0.033	0.122	0.79	-0.094	0.145	0.521
F1 Executive Function	1	0.026	0.014	0.06	0.015	0.013	0.263	0.051	0.013	0	-0.065	0.014	0
F1 Executive Function	2	0.04	0.013	0.002	0.035	0.012	0.003	0.051	0.011	0	-0.067	0.012	0
F1 Executive Function	3	0.033	0.016	0.047	0.027	0.014	0.061	0.034	0.013	0.009	-0.055	0.015	0
F1 Culture Fair Intelligence Test	1	-0.044	0.081	0.587	-0.032	0.076	0.678	0.11	0.077	0.151	-0.094	0.084	0.261
F1 Culture Fair	2	0.041	0.075	0.584	0.067	0.069	0.33	0.084	0.067	0.209	-0.084	0.074	0.257

Intelligence Test												
F1 Culture Fair Intelligence Test	3	0.085	0.092	0.36	0.096	0.082	0.244	0.099	0.075	0.192	-0.104	0.089	0.242
F1 Processing Speed	1	0.033	0.013	0.014	0.028	0.013	0.03	0.011	0.014	0.441	-0.014	0.014	0.331
F1 Processing Speed	2	0.023	0.014	0.094	0.019	0.013	0.153	0.005	0.013	0.701	-0.008	0.014	0.582
F1 Processing Speed	3	0.008	0.018	0.645	0.005	0.017	0.768	-0.003	0.016	0.834	-0.001	0.018	0.957
F1 Speech-in-noise Threshold	1	0.115	0.104	0.269	0.141	0.099	0.155	0.066	0.099	0.503	-0.068	0.105	0.522
F1 Speech-in-noise Threshold	2	0.023	0.014	0.094	0.019	0.013	0.153	0.005	0.013	0.701	-0.008	0.014	0.582
F1 Speech-in-noise Threshold	3	0.008	0.018	0.645	0.005	0.017	0.768	-0.003	0.016	0.834	-0.001	0.018	0.957
F2 CAPE psychosis risk	1	0.25	0.26	0.34	0.13	0.23	0.56	-0.15	0.26	0.56	-0.28	0.28	0.31
F2 CAPE psychosis risk	2	0.38	0.29	0.19	0.33	0.26	0.21	0.11	0.32	0.74	-0.08	0.33	0.80
F2 CAPE psychosis risk	3	0.38	0.29	0.19	0.33	0.26	0.21	0.11	0.33	0.74	-0.09	0.35	0.79

Noise exposure

Outcome	Model	Lden Estimate	SE	p	Lday Estimate	SE	p	Leve Estimate	SE	p	Lnight Estimate	SE	p
BL SDQ	1	0.04	0.016	0.011	-0.029	0.016	0.06	0.041	0.015	0.007	0.04	0.016	0.013
BL SDQ	2	0.02	0.016	0.219	-0.008	0.017	0.65	0.021	0.016	0.176	0.02	0.017	0.233
BL SDQ	3	0.022	0.016	0.179	0.009	0.019	0.645	0.023	0.016	0.14	0.022	0.017	0.192
BL Internalising	1	0.023	0.009	0.013	-0.011	0.009	0.205	0.023	0.009	0.01	0.023	0.009	0.014
BL Internalising	2	0.014	0.01	0.159	-0.001	0.01	0.894	0.013	0.009	0.142	0.014	0.01	0.163
BL Internalising	3	0.015	0.01	0.11	0.006	0.011	0.616	0.015	0.009	0.095	0.016	0.01	0.113
BL Externalising	1	0.017	0.01	0.078	-0.018	0.01	0.064	0.018	0.009	0.053	0.017	0.01	0.088
BL Externalising	2	0.007	0.01	0.517	-0.007	0.011	0.541	0.008	0.01	0.425	0.006	0.01	0.546
BL Externalising	3	0.006	0.01	0.522	0.001	0.012	0.91	0.008	0.01	0.429	0.006	0.01	0.551
BL Executive Function	1	-0.005	0.001	0	0.012	0.002	0	-0.005	0.001	0.001	-0.005	0.002	0
BL Executive Function	2	0.001	0.002	0.504	0.004	0.002	0.063	0.001	0.002	0.497	0.001	0.002	0.506
BL Executive Function	3	0.001	0.002	0.532	0.003	0.002	0.235	0.001	0.002	0.537	0.001	0.002	0.531
BL Culture Fair Intelligence Test	1	-0.011	0.008	0.165	0.046	0.008	0	-0.011	0.008	0.168	-0.011	0.008	0.165
BL Culture Fair	2	0.015	0.01	0.139	0.004	0.011	0.749	0.015	0.01	0.14	0.015	0.01	0.139

Intelligence Test												
BL Culture Fair Intelligence Test	3	0.011	0.01	0.275	0.005	0.013	0.685	0.01	0.01	0.29	0.011	0.01	0.271
BL Processing Speed	1	0.003	0.001	0.03	0	0.001	0.895	0.003	0.001	0.027	0.003	0.001	0.031
BL Processing Speed	2	0.001	0.002	0.732	-0.001	0.002	0.717	0.001	0.002	0.673	0.001	0.002	0.757
BL Processing Speed	3	0	0.002	0.959	-0.001	0.002	0.695	0	0.002	0.979	0	0.002	0.935
BL Speech-in-noise Threshold	1	0.017	0.008	0.027	0	0.001	0.895	0.003	0.001	0.027	0.003	0.001	0.031
BL Speech-in-noise Threshold	2	-0.005	0.01	0.634	-0.001	0.002	0.717	0.001	0.002	0.673	0.001	0.002	0.757
BL Speech-in-noise Threshold	3	-0.006	0.01	0.577	-0.001	0.002	0.695	0	0.002	0.979	0	0.002	0.935
F1 SDQ	1	0.012	0.018	0.504	-0.035	0.017	0.036	0.013	0.017	0.426	0.011	0.018	0.526
F1 SDQ	2	0.007	0.017	0.693	-0.007	0.018	0.71	0.008	0.016	0.638	0.007	0.017	0.703
F1 SDQ	3	0.005	0.017	0.784	0.004	0.02	0.834	0.006	0.016	0.712	0.004	0.017	0.8
F1 Internalising	1	0	0.011	0.969	-0.031	0.01	0.002	0.002	0.01	0.876	0	0.011	0.99
F1 Internalising	2	-0.001	0.01	0.936	-0.011	0.011	0.315	-0.001	0.01	0.952	-0.001	0.01	0.94
F1 Internalising	3	-0.002	0.01	0.877	-0.005	0.012	0.677	-0.001	0.01	0.912	-0.002	0.011	0.874
F1 Externalising	1	0.011	0.011	0.312	-0.004	0.011	0.706	0.012	0.011	0.271	0.011	0.011	0.325

F1 Externalising	2	0.005	0.01	0.614	0.01	0.011	0.367	0.006	0.01	0.568	0.005	0.01	0.626
F1 Externalising	3	0.004	0.01	0.687	0.011	0.012	0.386	0.005	0.01	0.632	0.004	0.01	0.702
F1 PHQ-9 Depression	1	0.002	0.019	0.906	-0.01	0.017	0.573	0.003	0.018	0.849	0.002	0.019	0.92
F1 PHQ-9 Depression	2	-0.01	0.019	0.589	-0.005	0.02	0.816	-0.009	0.018	0.613	-0.011	0.019	0.586
F1 PHQ-9 Depression	3	-0.011	0.019	0.567	-0.009	0.022	0.682	-0.01	0.018	0.594	-0.011	0.02	0.564
F1 GAD-7 Anxiety	1	0.031	0.016	0.053	0.015	0.015	0.307	0.031	0.015	0.038	0.031	0.016	0.059
F1 GAD-7 Anxiety	2	0.024	0.016	0.13	0.012	0.017	0.489	0.025	0.015	0.107	0.024	0.016	0.138
F1 GAD-7 Anxiety	3	0.024	0.016	0.136	0.013	0.018	0.464	0.025	0.015	0.112	0.024	0.016	0.145
F1 Executive Function	1	-0.003	0.002	0.173	0.008	0.002	0	-0.002	0.002	0.164	-0.003	0.002	0.176
F1 Executive Function	2	0	0.002	0.819	-0.001	0.002	0.66	0	0.002	0.799	0	0.002	0.826
F1 Executive Function	3	-0.001	0.002	0.737	0	0.002	0.993	-0.001	0.002	0.698	-0.001	0.002	0.752
F1 Culture Fair Intelligence Test	1	-0.013	0.011	0.226	0.041	0.011	0	-0.014	0.01	0.174	-0.013	0.011	0.245
F1 Culture Fair Intelligence Test	2	0.002	0.01	0.831	-0.009	0.01	0.376	0.001	0.009	0.934	0.003	0.01	0.797
F1 Culture Fair Intelligence Test	3	0.003	0.01	0.8	-0.002	0.011	0.841	0.001	0.009	0.903	0.003	0.01	0.768

F1 Processing Speed	1	0.001	0.002	0.752	0.001	0.002	0.706	0.001	0.002	0.721	0.001	0.002	0.766
F1 Processing Speed	2	-0.001	0.002	0.667	0.002	0.002	0.429	-0.001	0.002	0.704	-0.001	0.002	0.652
F1 Processing Speed	3	-0.002	0.002	0.411	0	0.002	0.884	-0.001	0.002	0.444	-0.002	0.002	0.399
F1 Speech-in-noise Threshold	1	0.022	0.015	0.139	0.001	0.002	0.706	0.001	0.002	0.721	0.001	0.002	0.766
F1 Speech-in-noise Threshold	2	0.01	0.015	0.521	0.002	0.002	0.429	-0.001	0.002	0.704	-0.001	0.002	0.652
F1 Speech-in-noise Threshold	3	0.005	0.015	0.73	0	0.002	0.884	-0.001	0.002	0.444	-0.002	0.002	0.399

XIV: Results for associations between exposure to air pollution or noise and psychological outcomes, for participants with a precise (certain) geocode only

Air pollution (N= 3037)													
Outcome	**Model**	**PM2.5 Estimate**	**SE**	**p**	**PM10 Estimate**	**SE**	**p**	**NO2 Estimate**	**SE**	**p**	**Ozone Estimate**	**SE**	**p**
BL SDQ	1	-0.289	0.115	0.012	0.088	0.105	0.405	-0.066	0.127	0.602	-0.097	0.136	0.477
BL SDQ	2	-0.198	0.121	0.101	0.132	0.106	0.214	0.035	0.129	0.786	-0.155	0.137	0.258
BL SDQ	3	-0.22	0.149	0.143	0.088	0.12	0.463	0.09	0.156	0.566	-0.228	0.16	0.154
BL Internalising	1	-0.102	0.067	0.13	-0.015	0.061	0.805	-0.114	0.074	0.125	-0.031	0.079	0.693
BL Internalising	2	-0.118	0.071	0.097	-0.004	0.062	0.952	-0.101	0.076	0.182	0.022	0.081	0.788
BL Internalising	3	-0.103	0.086	0.232	0.005	0.07	0.946	-0.054	0.09	0.551	-0.061	0.093	0.514
BL Externalising	1	-0.187	0.071	0.009	0.103	0.065	0.114	0.047	0.079	0.547	-0.065	0.084	0.435
BL Externalising	2	-0.08	0.074	0.279	0.136	0.065	0.037	0.136	0.079	0.085	-0.177	0.084	0.036
BL Externalising	3	-0.119	0.091	0.191	0.085	0.073	0.244	0.132	0.094	0.164	-0.16	0.096	0.098
BL Executive Function	1	0.058	0.013	0	0.045	0.013	0	0.071	0.015	0	-0.012	0.016	0.442
BL Executive Function	2	0.029	0.014	0.033	0.025	0.012	0.039	0.039	0.015	0.008	-0.012	0.016	0.445
BL Executive Function	3	0.033	0.018	0.058	0.035	0.014	0.012	0.04	0.018	0.031	-0.003	0.019	0.88
BL Culture Fair Intelligence Test	1	0.037	0.071	0.597	0.132	0.066	0.048	0.193	0.079	0.015	-0.033	0.086	0.702

BL Culture Fair Intelligence Test	2	-0.059	0.074	0.427	0.047	0.066	0.479	0.049	0.08	0.538	0.014	0.086	0.868
BL Culture Fair Intelligence Test	3	0.022	0.099	0.827	0.09	0.077	0.241	0.113	0.102	0.268	-0.022	0.104	0.832
BL Processing Speed	1	-0.007	0.013	0.568	-0.007	0.011	0.551	-0.008	0.014	0.573	-0.002	0.016	0.898
BL Processing Speed	2	-0.006	0.013	0.673	-0.002	0.012	0.85	0	0.015	0.995	-0.001	0.016	0.951
BL Processing Speed	3	-0.027	0.017	0.12	-0.021	0.013	0.114	-0.01	0.019	0.594	0.004	0.019	0.846
BL Speech-in-noise Threshold	1	-0.207	0.074	0.005	0.038	0.063	0.549	-0.048	0.077	0.531	-0.059	0.08	0.461
BL Speech-in-noise Threshold	2	-0.17	0.076	0.026	0.065	0.064	0.313	-0.012	0.078	0.88	-0.037	0.082	0.651
BL Speech-in-noise Threshold	3	-0.107	0.093	0.251	0.003	0.072	0.971	-0.004	0.095	0.966	-0.066	0.096	0.491
F1 SDQ	1	0.087	0.128	0.496	-0.009	0.11	0.937	-0.125	0.125	0.317	-0.028	0.14	0.843
F1 SDQ	2	0.116	0.131	0.376	-0.087	0.099	0.377	-0.128	0.112	0.254	0.137	0.126	0.276
F1 SDQ	3	0.083	0.164	0.611	-0.107	0.111	0.337	-0.056	0.131	0.67	0.021	0.144	0.885
F1 Internalising	1	-0.01	0.077	0.898	-0.048	0.066	0.471	-0.121	0.075	0.109	0.007	0.084	0.933
F1 Internalising	2	-0.038	0.079	0.631	-0.056	0.06	0.348	-0.082	0.068	0.228	0.106	0.077	0.166
F1 Internalising	3	-0.039	0.1	0.697	-0.075	0.067	0.262	-0.069	0.077	0.376	0.071	0.085	0.406
F1 Externalising	1	0.097	0.082	0.235	0.039	0.071	0.58	-0.005	0.08	0.955	-0.035	0.089	0.698
F1 Externalising	2	0.021	0.077	0.787	-0.032	0.064	0.613	-0.049	0.073	0.5	0.034	0.081	0.674
F1 Externalising	3	0.024	0.095	0.803	-0.031	0.071	0.66	0.001	0.084	0.994	-0.029	0.093	0.757
F1 PHQ-9 Depression	1	0.186	0.14	0.183	0.129	0.12	0.285	0.109	0.142	0.443	-0.243	0.167	0.146
F1 PHQ-9 Depression	2	0.004	0.148	0.977	0.057	0.122	0.638	0.021	0.144	0.884	0.005	0.171	0.975

F1 PHQ-9 Depression	3	0.008	0.17	0.963	0.059	0.131	0.656	0.038	0.155	0.804	0.017	0.18	0.926
F1 GAD-7 Anxiety	1	0.305	0.12	0.011	0.289	0.103	0.005	0.276	0.122	0.023	-0.228	0.144	0.112
F1 GAD-7 Anxiety	2	0.18	0.126	0.153	0.227	0.104	0.029	0.193	0.122	0.115	-0.042	0.145	0.774
F1 GAD-7 Anxiety	3	0.212	0.143	0.142	0.242	0.11	0.029	0.201	0.129	0.123	-0.03	0.152	0.846
F1 Executive Function	1	0.066	0.014	0	0.057	0.012	0	0.101	0.013	0	-0.081	0.015	0
F1 Executive Function	2	0.047	0.013	0	0.029	0.011	0.008	0.06	0.012	0	-0.08	0.013	0
F1 Executive Function	3	0.031	0.016	0.063	0.013	0.012	0.287	0.038	0.014	0.007	-0.056	0.015	0
F1 Culture Fair Intelligence Test	1	0.092	0.083	0.27	0.186	0.072	0.009	0.263	0.08	0.001	-0.179	0.091	0.049
F1 Culture Fair Intelligence Test	2	0.038	0.076	0.616	0.084	0.063	0.18	0.056	0.071	0.428	-0.129	0.08	0.105
F1 Culture Fair Intelligence Test	3	0.083	0.09	0.357	0.099	0.069	0.154	0.072	0.079	0.365	-0.155	0.088	0.08
F1 Processing Speed	1	0.031	0.014	0.027	0.019	0.012	0.103	0.009	0.014	0.49	0.017	0.016	0.278
F1 Processing Speed	2	0.022	0.014	0.123	0.016	0.012	0.175	0.002	0.013	0.875	0.019	0.015	0.207
F1 Processing Speed	3	0.013	0.018	0.453	0.013	0.013	0.308	0.004	0.016	0.81	0.021	0.017	0.212
F1 Speech-in-noise Threshold	1	-0.019	0.102	0.849	-0.047	0.087	0.587	-0.132	0.096	0.172	-0.003	0.106	0.979
F1 Speech-in-noise Threshold	2	-0.059	0.108	0.582	-0.024	0.088	0.782	-0.097	0.098	0.321	0.103	0.108	0.345

F1 Speech-in-noise Threshold	3	-0.1	0.131	0.449	-0.041	0.099	0.681	-0.095	0.116	0.415	0.084	0.126	0.504
		OR	**LL**	**UL**	**OR**	**LL**	**UL**	**OR**	**LL**	**UL**	**OR**	**LL**	**UL**
F2 CAPE risk	1	1.11	0.62	1.94	0.96	0.60	1.55	0.64	0.35	1.13	0.68	0.35	0.83
F2 CAPE risk	2	1.33	0.68	2.58	1.17	0.68	2.05	0.78	0.38	1.58	0.83	0.37	1.77
F2 CAPE risk	3	1.33	0.69	2.56	1.17	0.68	2.01	0.78	0.39	1.59	0.80	0.34	1.86
Noise models (N=3276)													
Outcome	**Model**	**Lden Estimate**	**SE**	**p**	**Lday Estimate**	**SE**	**p**	**Leve Estimate**	**SE**	**p**	**Lnight Estimate**	**SE**	**p**
BL SDQ	1	0.03	0.018	0.092	-0.045	0.017	0.007	0.031	0.017	0.065	0.029	0.018	0.102
BL SDQ	2	0.016	0.017	0.346	-0.008	0.018	0.662	0.018	0.017	0.276	0.016	0.018	0.368
BL SDQ	3	0.018	0.017	0.302	0.007	0.021	0.743	0.02	0.017	0.237	0.017	0.018	0.324
BL Internalising	1	0.016	0.01	0.115	-0.023	0.01	0.02	0.017	0.01	0.091	0.016	0.01	0.124
BL Internalising	2	0.009	0.01	0.364	-0.008	0.011	0.482	0.01	0.01	0.318	0.009	0.01	0.378
BL Internalising	3	0.011	0.01	0.273	0.002	0.012	0.897	0.012	0.01	0.231	0.011	0.01	0.286
BL Externalising	1	0.014	0.011	0.213	-0.023	0.01	0.03	0.014	0.01	0.164	0.013	0.011	0.231
BL Externalising	2	0.007	0.011	0.504	0	0.011	0.968	0.008	0.01	0.413	0.007	0.011	0.534
BL Externalising	3	0.007	0.011	0.536	0.004	0.013	0.781	0.008	0.01	0.445	0.006	0.011	0.566
BL Executive Function	1	-0.002	0.002	0.307	0.013	0.002	0	-0.002	0.002	0.276	-0.002	0.002	0.319
BL Executive Function	2	0.001	0.002	0.494	0.003	0.002	0.12	0.001	0.002	0.499	0.001	0.002	0.493
BL Executive Function	3	0.001	0.002	0.508	0.003	0.002	0.262	0.001	0.002	0.521	0.001	0.002	0.505
BL Culture Fair Intelligence Test	1	-0.002	0.011	0.884	0.033	0.011	0.002	-0.002	0.011	0.846	-0.001	0.011	0.897

BL Culture Fair Intelligence Test	2	0.009	0.011	0.391	-0.006	0.011	0.588	0.009	0.01	0.398	0.009	0.011	0.389
BL Culture Fair Intelligence Test	3	0.008	0.011	0.48	0.001	0.013	0.94	0.007	0.01	0.509	0.008	0.011	0.471
BL Processing Speed	1	0	0.002	0.846	-0.003	0.002	0.133	0.001	0.002	0.73	0	0.002	0.889
BL Processing Speed	2	0	0.002	0.97	-0.002	0.002	0.471	0	0.002	0.866	0	0.002	0.991
BL Processing Speed	3	-0.001	0.002	0.739	-0.001	0.002	0.582	0	0.002	0.833	-0.001	0.002	0.707
BL Speech-in-noise Threshold	1	0.014	0.011	0.171	-0.003	0.002	0.133	0.001	0.002	0.73	0	0.002	0.889
BL Speech-in-noise Threshold	2	0.004	0.011	0.688	-0.002	0.002	0.471	0	0.002	0.866	0	0.002	0.991
BL Speech-in-noise Threshold	3	0.003	0.01	0.774	-0.001	0.002	0.582	0	0.002	0.833	-0.001	0.002	0.707
F1 SDQ	1	0.01	0.019	0.612	-0.04	0.018	0.025	0.011	0.018	0.541	0.009	0.02	0.63
F1 SDQ	2	0.003	0.018	0.886	-0.005	0.019	0.782	0.004	0.017	0.822	0.002	0.018	0.9
F1 SDQ	3	0.001	0.018	0.972	0.003	0.021	0.874	0.002	0.017	0.893	0	0.018	0.99
F1 Internalising	1	-0.001	0.012	0.91	-0.034	0.011	0.002	0	0.011	0.968	-0.001	0.012	0.901
F1 Internalising	2	-0.004	0.011	0.691	-0.012	0.011	0.288	-0.004	0.01	0.711	-0.004	0.011	0.693
F1 Internalising	3	-0.005	0.011	0.637	-0.007	0.013	0.56	-0.004	0.01	0.673	-0.005	0.011	0.633
F1 Externalising	1	0.011	0.012	0.371	-0.007	0.012	0.548	0.012	0.012	0.322	0.011	0.013	0.386
F1 Externalising	2	0.003	0.011	0.809	0.011	0.011	0.327	0.003	0.01	0.764	0.002	0.011	0.821
F1 Externalising	3	0.002	0.011	0.828	0.013	0.013	0.327	0.003	0.01	0.773	0.002	0.011	0.842
F1 PHQ-9 Depression	1	0.003	0.021	0.897	-0.01	0.019	0.603	0.003	0.02	0.86	0.003	0.021	0.905
F1 PHQ-9 Depression	2	-0.009	0.02	0.651	0.007	0.021	0.733	-0.009	0.019	0.661	-0.009	0.021	0.652

F1 PHQ-9 Depression	3	-0.01	0.021	0.637	0.003	0.023	0.878	-0.009	0.02	0.649	-0.01	0.021	0.638
F1 GAD-7 Anxiety	1	0.026	0.018	0.148	0.013	0.016	0.424	0.027	0.017	0.118	0.026	0.018	0.159
F1 GAD-7 Anxiety	2	0.019	0.017	0.277	0.015	0.018	0.401	0.02	0.017	0.236	0.019	0.018	0.29
F1 GAD-7 Anxiety	3	0.018	0.018	0.299	0.016	0.019	0.405	0.019	0.017	0.254	0.018	0.018	0.314
F1 Executive Function	1	-0.003	0.002	0.201	0.009	0.002	0	-0.003	0.002	0.182	-0.003	0.002	0.208
F1 Executive Function	2	-0.001	0.002	0.688	0	0.002	0.934	-0.001	0.002	0.654	-0.001	0.002	0.7
F1 Executive Function	3	-0.001	0.002	0.474	0	0.002	0.957	-0.001	0.002	0.429	-0.001	0.002	0.49
F1 Culture Fair Intelligence Test	1	-0.008	0.012	0.511	0.042	0.011	0	-0.009	0.012	0.433	-0.008	0.012	0.539
F1 Culture Fair Intelligence Test	2	0.003	0.01	0.777	-0.01	0.011	0.34	0.002	0.01	0.864	0.003	0.011	0.749
		OR	**LL**	**UL**	**OR**	**LL**	**UL**	**OR**	**LL**	**UL**	**OR**	**LL**	**UL**
F2 CAPE risk	1	0.97	0.64	1.42	0.94	0.53	1.64	0.97	0.64	1.42	0.97	0.64	1.43
F2 CAPE risk	2	0.95	0.59	1.48	0.90	0.47	1.72	0.95	0.59	1.47	0.95	0.59	1.48
F2 CAPE risk	3	0.95	0.60	1.50	0.90	0.47	1.71	0.95	0.61	1.49	0.95	0.60	1.50

XV: Exposure to outdoor air pollution and psychological outcomes in the SCAMP cohort

Outcome	Model	PM2.5 Estimate	SE	p	PM10 Estimate	SE	p	NO2 Estimate	SE	p	Ozone Estimate	SE	p
BL SDQ	1	-0.172	0.094	0.068	0.302	0.099	0.002	0.159	0.118	0.178	-0.165	0.13	0.204
BL SDQ	2	-0.13	0.098	0.184	0.262	0.102	0.01	0.154	0.119	0.196	-0.158	0.132	0.23
BL SDQ	3	-0.104	0.124	0.403	0.251	0.119	0.035	0.262	0.143	0.068	-0.349	0.17	0.041
BL Internalising	1	-0.032	0.055	0.557	0.149	0.058	0.011	0.067	0.069	0.33	-0.052	0.076	0.496
BL Internalising	2	-0.06	0.058	0.297	0.106	0.06	0.076	0.03	0.07	0.663	-0.005	0.077	0.948
BL Internalising	3	-0.019	0.072	0.797	0.128	0.07	0.067	0.129	0.084	0.128	-0.15	0.1	0.134
BL Externalising	1	-0.14	0.058	0.016	0.153	0.062	0.013	0.092	0.073	0.207	-0.114	0.08	0.158
BL Externalising	2	-0.07	0.06	0.244	0.156	0.062	0.013	0.123	0.073	0.091	-0.153	0.081	0.058
BL Externalising	3	-0.092	0.076	0.225	0.124	0.072	0.088	0.129	0.087	0.138	-0.187	0.102	0.067
BL Executive Function	1	0.015	0.011	0.182	-0.035	0.012	0.004	-0.002	0.014	0.904	-0.003	0.016	0.867
BL Executive Function	2	0.003	0.011	0.792	-0.016	0.012	0.185	0.007	0.014	0.624	-0.017	0.015	0.254
BL Executive Function	3	0.004	0.015	0.805	-0.005	0.014	0.705	0.003	0.017	0.848	-0.013	0.02	0.506
BL Culture Fair Intelligence Test	1	-0.059	0.059	0.316	-0.121	0.063	0.055	-0.02	0.074	0.79	0.026	0.083	0.755

BL Culture Fair Intelligence Test	2	-0.079	0.06	0.19	-0.049	0.064	0.438	0.001	0.074	0.993	-0.006	0.082	0.942
BL Culture Fair Intelligence Test	3	-0.014	0.083	0.862	0.037	0.076	0.625	0.038	0.092	0.675	-0.034	0.112	0.759
BL Processing Speed	1	0.001	0.01	0.955	0.015	0.011	0.178	0.003	0.014	0.809	-0.008	0.015	0.604
BL Processing Speed	2	-0.003	0.011	0.787	0.013	0.012	0.286	0.003	0.014	0.843	-0.007	0.015	0.625
BL Processing Speed	3	-0.013	0.014	0.37	-0.002	0.014	0.867	-0.002	0.017	0.919	-0.004	0.02	0.83
BL Speech-in-noise Threshold	1	-0.121	0.062	0.049	0.166	0.061	0.006	0.06	0.073	0.411	-0.085	0.08	0.291
BL Speech-in-noise Threshold	2	-0.003	0.011	0.787	0.013	0.012	0.286	0.003	0.014	0.843	-0.007	0.015	0.625
BL Speech-in-noise Threshold	3	-0.013	0.014	0.37	-0.002	0.014	0.867	-0.002	0.017	0.919	-0.004	0.02	0.83
F1 SDQ	1	0.244	0.125	0.051	0.265	0.118	0.025	0.01	0.121	0.937	0.001	0.131	0.993
F1 SDQ	2	0.229	0.13	0.077	-0.034	0.111	0.761	-0.169	0.107	0.116	0.214	0.119	0.072
F1 SDQ	3	0.238	0.166	0.153	-0.042	0.135	0.754	-0.095	0.124	0.442	0.149	0.147	0.31
F1 Internalising	1	0.046	0.075	0.54	0.079	0.071	0.266	-0.044	0.073	0.543	0.048	0.079	0.541

F1 Internalising	2	-0.002	0.078	0.978	-0.071	0.067	0.286	-0.125	0.065	0.055	0.132	0.072	0.068
F1 Internalising	3	0.004	0.101	0.97	-0.115	0.081	0.157	-0.13	0.074	0.08	0.15	0.087	0.085
F1 Externalising	1	0.198	0.079	0.013	0.186	0.075	0.013	0.054	0.077	0.484	-0.047	0.083	0.573
F1 Externalising	2	0.077	0.076	0.311	0.04	0.071	0.576	-0.044	0.069	0.525	0.084	0.076	0.272
F1 Externalising	3	0.109	0.098	0.266	0.072	0.087	0.408	0.03	0.08	0.705	0.001	0.096	0.99
F1 PHQ-9 Depression	1	0.104	0.133	0.437	0.124	0.126	0.324	0.002	0.133	0.991	-0.076	0.149	0.612
F1 PHQ-9 Depression	2	-0.1	0.144	0.485	-0.043	0.133	0.746	-0.132	0.133	0.321	0.092	0.153	0.548
F1 PHQ-9 Depression	3	-0.104	0.174	0.551	-0.054	0.156	0.728	-0.119	0.147	0.419	0.067	0.177	0.706
F1 GAD-7 Anxiety	1	0.168	0.114	0.142	0.183	0.108	0.089	0.114	0.113	0.314	-0.182	0.128	0.154
F1 GAD-7 Anxiety	2	0.051	0.122	0.676	0.097	0.113	0.387	0.043	0.113	0.702	-0.098	0.13	0.451
F1 GAD-7 Anxiety	3	0.038	0.143	0.788	0.08	0.128	0.534	0.033	0.122	0.79	-0.094	0.145	0.521
F1 Executive Function	1	0.026	0.014	0.06	0.015	0.013	0.263	0.051	0.013	0	-0.065	0.014	0
F1 Executive Function	2	0.04	0.013	0.002	0.035	0.012	0.003	0.051	0.011	0	-0.067	0.012	0
F1 Executive Function	3	0.033	0.016	0.047	0.027	0.014	0.061	0.034	0.013	0.009	-0.055	0.015	0
F1 Culture Fair Intelligence Test	1	-0.044	0.081	0.587	-0.032	0.076	0.678	0.11	0.077	0.151	-0.094	0.084	0.261

F1 Culture Fair Intelligence Test	2	0.041	0.075	0.584	0.067	0.069	0.33	0.084	0.067	0.209	-0.084	0.074	0.257
F1 Culture Fair Intelligence Test	3	0.085	0.092	0.36	0.096	0.082	0.244	0.099	0.075	0.192	-0.104	0.089	0.242
F1 Processing Speed	1	0.033	0.013	0.014	0.028	0.013	0.03	0.011	0.014	0.441	-0.014	0.014	0.331
F1 Processing Speed	2	0.023	0.014	0.094	0.019	0.013	0.153	0.005	0.013	0.701	-0.008	0.014	0.582
F1 Processing Speed	3	0.008	0.018	0.645	0.005	0.017	0.768	-0.003	0.016	0.834	-0.001	0.018	0.957
F1 Speech-in-noise Threshold	1	0.115	0.104	0.269	0.141	0.099	0.155	0.066	0.099	0.503	-0.068	0.105	0.522
F1 Speech-in-noise Threshold	2	0.023	0.014	0.094	0.019	0.013	0.153	0.005	0.013	0.701	-0.008	0.014	0.582
F1 Speech-in-noise Threshold	3	0.008	0.018	0.645	0.005	0.017	0.768	-0.003	0.016	0.834	-0.001	0.018	0.957
F2 Psychosis Risk	1	0.31	0.23	0.17	0.17	0.21	0.19	0.06	0.25	0.82	-0.09	0.28	0.74
F2 Psychosis Risk	2	0.29	0.26	0.27	0.16	0.24	0.51	0.01	0.29	0.98	-0.03	0.32	0.94
F2 Psychosis Risk	3	0.29	0.26	0.27	0.16	0.24	0.51	0.01	0.29	0.97	-0.03	0.33	0.92

XVI: Exposure to indoor air pollution (with indoor sources) and psychological outcomes in the SCAMP cohort

Outcome	Model	PM2.5 Estimate	SE	p	PM10 Estimate	SE	p	NO2 Estimate	SE	p	Ozone Estimate	SE	p
BL SDQ	1	-0.21	0.116	0.069	0.106	0.076	0.163	0.047	0.101	0.642	-0.076	0.111	0.495
BL SDQ	2	-0.153	0.119	0.199	0.104	0.075	0.167	0.056	0.1	0.576	-0.034	0.111	0.76
BL SDQ	3	-0.079	0.143	0.583	0.102	0.08	0.199	0.139	0.109	0.204	-0.022	0.117	0.853
BL Internalising	1	-0.054	0.067	0.424	0.041	0.045	0.356	-0.009	0.059	0.882	-0.008	0.065	0.9
BL Internalising	2	-0.077	0.07	0.273	0.036	0.044	0.42	-0.019	0.059	0.743	0.051	0.065	0.435
BL Internalising	3	-0.009	0.084	0.91	0.054	0.047	0.25	0.045	0.064	0.479	0.035	0.069	0.615
BL Externalising	1	-0.156	0.071	0.029	0.065	0.047	0.165	0.056	0.062	0.371	-0.068	0.069	0.325
BL Externalising	2	-0.076	0.073	0.297	0.069	0.046	0.138	0.075	0.061	0.219	-0.084	0.068	0.213
BL Externalising	3	-0.078	0.087	0.373	0.048	0.049	0.321	0.087	0.067	0.193	-0.056	0.071	0.433
BL Executive Function	1	0.033	0.014	0.015	0.012	0.009	0.197	0.022	0.012	0.066	0.011	0.013	0.397
BL Executive Function	2	0.019	0.014	0.156	0.014	0.009	0.12	0.023	0.012	0.044	-0.008	0.013	0.532
BL Executive Function	3	0.029	0.017	0.083	0.019	0.009	0.038	0.023	0.013	0.07	-0.003	0.014	0.829

BL Culture Fair Intelligence Test	1	-0.096	0.072	0.183	-0.03	0.049	0.535	-0.029	0.063	0.647	-0.031	0.071	0.668
BL Culture Fair Intelligence Test	2	-0.129	0.074	0.082	-0.025	0.048	0.593	-0.033	0.062	0.598	-0.084	0.07	0.23
BL Culture Fair Intelligence Test	3	-0.071	0.094	0.448	-0.013	0.05	0.802	-0.026	0.068	0.704	-0.104	0.074	0.163
BL Processing Speed	1	0.003	0.013	0.824	0.002	0.008	0.783	0.004	0.011	0.748	0.008	0.013	0.546
BL Processing Speed	2	0	0.013	0.977	0.002	0.008	0.782	0.005	0.012	0.67	0.014	0.013	0.308
BL Processing Speed	3	-0.012	0.016	0.442	-0.007	0.009	0.433	0.001	0.013	0.924	0.008	0.014	0.549
BL Speech-in-noise Threshold	1	-0.167	0.074	0.024	0.01	0.046	0.834	-0.006	0.061	0.928	-0.074	0.066	0.265
BL Speech-in-noise Threshold	2	-0.176	0.076	0.021	-0.002	0.046	0.965	-0.032	0.062	0.603	-0.041	0.067	0.536
BL Speech-in-noise Threshold	3	-0.124	0.089	0.163	-0.033	0.048	0.493	-0.02	0.067	0.764	-0.059	0.07	0.399
F1 SDQ	1	0.13	0.111	0.242	0.005	0.075	0.943	-0.087	0.098	0.377	-0.049	0.116	0.671
F1 SDQ	2	0.14	0.112	0.214	-0.069	0.065	0.293	-0.144	0.085	0.092	0.088	0.101	0.386

F1 SDQ	3	0.107	0.132	0.416	-0.088	0.068	0.195	-0.099	0.091	0.277	-0.008	0.107	0.938
F1 Internalising	1	0.027	0.067	0.689	0.001	0.045	0.99	-0.062	0.059	0.295	0.029	0.07	0.682
F1 Internalising	2	0.019	0.067	0.778	-0.023	0.04	0.562	-0.061	0.052	0.242	0.099	0.062	0.107
F1 Internalising	3	0.031	0.08	0.698	-0.032	0.041	0.444	-0.052	0.055	0.343	0.073	0.065	0.261
F1 Externalising	1	0.103	0.071	0.143	0.005	0.048	0.919	-0.025	0.062	0.687	-0.078	0.074	0.291
F1 Externalising	2	-0.002	0.066	0.974	-0.045	0.042	0.279	-0.085	0.055	0.122	-0.008	0.065	0.897
F1 Externalising	3	-0.02	0.077	0.791	-0.052	0.044	0.237	-0.048	0.059	0.412	-0.068	0.069	0.322
F1 PHQ-9 Depression	1	0.086	0.119	0.469	0.037	0.08	0.643	-0.026	0.105	0.806	-0.025	0.133	0.851
F1 PHQ-9 Depression	2	-0.039	0.123	0.752	0.015	0.079	0.847	-0.052	0.103	0.611	0.124	0.132	0.345
F1 PHQ-9 Depression	3	-0.008	0.141	0.956	0.03	0.082	0.718	-0.025	0.108	0.816	0.14	0.136	0.303
F1 GAD-7 Anxiety	1	0.181	0.102	0.075	0.114	0.068	0.094	0.075	0.09	0.406	-0.015	0.114	0.894
F1 GAD-7 Anxiety	2	0.114	0.105	0.277	0.101	0.067	0.132	0.081	0.087	0.357	0.078	0.111	0.481
F1 GAD-7 Anxiety	3	0.131	0.117	0.266	0.103	0.069	0.136	0.087	0.091	0.338	0.082	0.114	0.47
F1 Executive Function	1	0.033	0.012	0.007	0.02	0.008	0.014	0.047	0.01	0	-0.02	0.012	0.101
F1 Executive Function	2	0.032	0.011	0.004	0.013	0.007	0.07	0.034	0.009	0	-0.034	0.011	0.002
F1 Executive Function	3	0.022	0.013	0.094	0.008	0.007	0.282	0.02	0.01	0.037	-0.014	0.011	0.231

F1 Culture Fair Intelligence Test	1	0.002	0.072	0.98	0.056	0.048	0.248	0.079	0.062	0.201	-0.06	0.075	0.426
F1 Culture Fair Intelligence Test	2	0.053	0.065	0.416	0.058	0.041	0.16	0.044	0.053	0.409	-0.073	0.064	0.258
F1 Culture Fair Intelligence Test	3	0.091	0.074	0.22	0.064	0.043	0.138	0.051	0.056	0.362	-0.072	0.067	0.282
F1 Processing Speed	1	0.032	0.012	0.008	0.015	0.008	0.065	0.013	0.011	0.228	0.025	0.013	0.063
F1 Processing Speed	2	0.025	0.012	0.042	0.014	0.008	0.081	0.007	0.01	0.479	0.017	0.012	0.165
F1 Processing Speed	3	0.016	0.014	0.267	0.01	0.008	0.215	0.004	0.011	0.696	0.012	0.013	0.357
F1 Speech-in-noise Threshold	1	0.057	0.092	0.537	0.036	0.062	0.565	0.018	0.081	0.823	-0.042	0.091	0.642
F1 Speech-in-noise Threshold	2	-0.063	0.098	0.517	0.005	0.062	0.933	-0.036	0.081	0.654	0.069	0.091	0.449
F1 Speech-in-noise Threshold	3	-0.082	0.111	0.461	0.007	0.065	0.912	-0.032	0.085	0.704	0.01	0.097	0.92
F2 Psychosis Risk	1	0.17	0.25	0.49	0.07	0.16	0.44	-0.25	0.20	0.22	-0.07	0.23	0.76

F2 Psychosis Risk	2	0.34	0.28	0.22	0.21	0.19	0.25	-0.12	0.22	0.59	0.10	0.27	0.71
F2 Psychosis Risk	3	0.34	0.28	0.22	0.21	0.19	0.25	-0.12	0.22	0.59	0.10	0.27	0.71

XVII: Exposure to indoor air pollution (without indoor sources) and psychological outcomes in the SCAMP cohort

Outcome	Model	PM2.5 Estimate	SE	p	PM10 Estimate	SE	p	NO2 Estimate	SE	p	Ozone Estimate	SE	p
BL SDQ	1	-0.19	0.112	0.09	0.246	0.111	0.026	0.139	0.113	0.217	-0.076	0.111	0.495
BL SDQ	2	-0.153	0.116	0.188	0.203	0.112	0.07	0.128	0.113	0.259	-0.034	0.111	0.76
BL SDQ	3	-0.087	0.144	0.548	0.231	0.13	0.077	0.249	0.129	0.055	-0.022	0.117	0.853
BL Internalising	1	-0.035	0.066	0.598	0.119	0.065	0.066	0.059	0.066	0.371	-0.008	0.065	0.9
BL Internalising	2	-0.063	0.068	0.357	0.101	0.066	0.126	0.037	0.066	0.577	0.051	0.065	0.435
BL Internalising	3	-0.001	0.084	0.994	0.146	0.077	0.057	0.126	0.076	0.098	0.035	0.069	0.615
BL Externalising	1	-0.156	0.069	0.025	0.127	0.068	0.063	0.08	0.07	0.249	-0.068	0.069	0.325
BL Externalising	2	-0.09	0.071	0.207	0.102	0.069	0.136	0.091	0.069	0.191	-0.084	0.068	0.213
BL Externalising	3	-0.094	0.088	0.282	0.081	0.079	0.308	0.114	0.079	0.146	-0.056	0.071	0.433
BL Executive Function	1	0.015	0.013	0.255	-0.026	0.014	0.053	-0.001	0.013	0.934	0.011	0.013	0.397
BL Executive Function	2	0.006	0.013	0.664	-0.009	0.013	0.495	0.008	0.013	0.526	-0.008	0.013	0.532
BL Executive Function	3	0.01	0.017	0.543	0	0.015	0.979	0.006	0.015	0.709	-0.003	0.014	0.829
BL Culture Fair	1	-0.139	0.07	0.047	-0.164	0.07	0.019	-0.088	0.071	0.211	-0.031	0.071	0.668

Intelligence Test													
BL Culture Fair Intelligence Test	2	-0.149	0.072	0.038	-0.095	0.07	0.172	-0.057	0.07	0.411	-0.084	0.07	0.23
BL Culture Fair Intelligence Test	3	-0.093	0.095	0.327	-0.059	0.083	0.476	-0.035	0.081	0.662	-0.104	0.074	0.163
BL Processing Speed	1	0.009	0.012	0.459	0.022	0.012	0.075	0.009	0.013	0.471	0.008	0.013	0.546
BL Processing Speed	2	0.007	0.013	0.59	0.023	0.013	0.066	0.01	0.013	0.437	0.014	0.013	0.308
BL Processing Speed	3	-0.001	0.016	0.951	0.01	0.015	0.493	0.007	0.015	0.649	0.008	0.014	0.549
BL Speech-in-noise Threshold	1	-0.127	0.072	0.08	0.131	0.069	0.056	0.034	0.07	0.624	-0.074	0.066	0.265
BL Speech-in-noise Threshold	2	-0.158	0.075	0.035	0.054	0.07	0.44	-0.025	0.071	0.73	-0.041	0.067	0.536
BL Speech-in-noise Threshold	3	-0.102	0.09	0.258	-0.015	0.08	0.847	-0.016	0.08	0.842	-0.059	0.07	0.399
F1 SDQ	1	0.212	0.117	0.07	0.14	0.116	0.228	-0.009	0.115	0.939	-0.049	0.116	0.671
F1 SDQ	2	0.203	0.121	0.092	-0.001	0.105	0.994	-0.128	0.102	0.21	0.088	0.101	0.386
F1 SDQ	3	0.19	0.148	0.199	-0.065	0.121	0.588	-0.075	0.113	0.505	-0.008	0.107	0.938
F1 Internalising	1	0.065	0.07	0.354	0.056	0.069	0.42	-0.018	0.069	0.799	0.029	0.07	0.682
F1 Internalising	2	0.055	0.072	0.448	0.011	0.064	0.866	-0.057	0.062	0.353	0.099	0.062	0.107

F1 Internalising	3	0.08	0.09	0.374	-0.018	0.073	0.807	-0.052	0.068	0.447	0.073	0.065	0.261
F1 Externalising	1	0.147	0.074	0.048	0.084	0.073	0.255	0.009	0.073	0.904	-0.078	0.074	0.291
F1 Externalising	2	0.044	0.07	0.534	-0.009	0.067	0.891	-0.07	0.065	0.286	-0.008	0.065	0.897
F1 Externalising	3	0.031	0.087	0.721	-0.044	0.078	0.574	-0.029	0.073	0.692	-0.068	0.069	0.322
F1 PHQ-9 Depression	1	0.107	0.123	0.386	0.099	0.12	0.411	-0.017	0.122	0.892	-0.025	0.133	0.851
F1 PHQ-9 Depression	2	-0.035	0.131	0.788	0.066	0.123	0.589	-0.082	0.122	0.503	0.124	0.132	0.345
F1 PHQ-9 Depression	3	0.003	0.155	0.985	0.123	0.141	0.383	-0.044	0.133	0.74	0.14	0.136	0.303
F1 GAD-7 Anxiety	1	0.172	0.105	0.102	0.178	0.103	0.083	0.094	0.105	0.37	-0.015	0.114	0.894
F1 GAD-7 Anxiety	2	0.105	0.111	0.346	0.187	0.104	0.073	0.078	0.103	0.45	0.078	0.111	0.481
F1 GAD-7 Anxiety	3	0.12	0.128	0.353	0.205	0.117	0.082	0.085	0.111	0.445	0.082	0.114	0.47
F1 Executive Function	1	0.018	0.013	0.165	0.004	0.013	0.736	0.038	0.012	0.002	-0.02	0.012	0.101
F1 Executive Function	2	0.027	0.012	0.02	0.012	0.011	0.287	0.036	0.011	0.001	-0.034	0.011	0.002
F1 Executive Function	3	0.021	0.014	0.156	0.012	0.013	0.333	0.023	0.012	0.055	-0.014	0.011	0.231
F1 Culture Fair Intelligence Test	1	-0.091	0.075	0.223	-0.098	0.073	0.182	0.036	0.072	0.615	-0.06	0.075	0.426
F1 Culture Fair Intelligence Test	2	-0.002	0.069	0.981	-0.014	0.065	0.825	0.034	0.063	0.585	-0.073	0.064	0.258

F1 Culture Fair Intelligence Test	3	0.028	0.082	0.737	-0.018	0.073	0.81	0.046	0.069	0.503	-0.072	0.067	0.282
F1 Processing Speed	1	0.039	0.013	0.002	0.044	0.012	0	0.02	0.013	0.121	0.025	0.013	0.063
F1 Processing Speed	2	0.027	0.013	0.037	0.03	0.012	0.014	0.011	0.012	0.353	0.017	0.012	0.165
F1 Processing Speed	3	0.014	0.016	0.378	0.017	0.014	0.222	0.004	0.014	0.758	0.012	0.013	0.357
F1 Speech-in-noise Threshold	1	0.057	0.096	0.553	0.101	0.095	0.289	0.007	0.096	0.94	-0.042	0.091	0.642
F1 Speech-in-noise Threshold	2	-0.09	0.105	0.388	0.021	0.099	0.832	-0.073	0.097	0.45	0.069	0.091	0.449
F1 Speech-in-noise Threshold	3	-0.176	0.125	0.16	-0.062	0.112	0.584	-0.129	0.107	0.225	0.01	0.097	0.92
F2 Psychosis Risk	1	0.14	0.26	0.59	0.11	0.27	0.68	-0.20	0.25	0.43	-0.07	0.232	0.76
F2 Psychosis Risk	2	0.24	0.29	0.41	0.27	0.30	0.38	-0.12	0.28	0.69	0.10	0.27	0.71
F2 Psychosis Risk	3	0.24	0.29	0.41	0.27	0.31	0.38	-0.12	0.28	0.68	0.10	0.27	0.71

XVIII: Exposure to hybrid air pollution (without indoor sources) and psychological outcomes in the SCAMP cohort

Outcome	Model	PM2.5 Estimate	SE	p	PM10 Estimate	SE	p	NO2 Estimate	SE	p	Ozone Estimate	SE	p
BL SDQ	1	-0.19	0.112	0.09	0.246	0.111	0.026	0.139	0.113	0.217	-0.076	0.111	0.495
BL SDQ	2	-0.153	0.116	0.188	0.203	0.112	0.07	0.128	0.113	0.259	-0.034	0.111	0.76
BL SDQ	3	-0.087	0.144	0.548	0.231	0.13	0.077	0.249	0.129	0.055	-0.022	0.117	0.853
BL Internalising	1	-0.035	0.066	0.598	0.119	0.065	0.066	0.059	0.066	0.371	-0.008	0.065	0.9
BL Internalising	2	-0.063	0.068	0.357	0.101	0.066	0.126	0.037	0.066	0.577	0.051	0.065	0.435
BL Internalising	3	-0.001	0.084	0.994	0.146	0.077	0.057	0.126	0.076	0.098	0.035	0.069	0.615
BL Externalising	1	-0.156	0.069	0.025	0.127	0.068	0.063	0.08	0.07	0.249	-0.068	0.069	0.325
BL Externalising	2	-0.09	0.071	0.207	0.102	0.069	0.136	0.091	0.069	0.191	-0.084	0.068	0.213
BL Externalising	3	-0.094	0.088	0.282	0.081	0.079	0.308	0.114	0.079	0.146	-0.056	0.071	0.433
BL Executive Function	1	0.015	0.013	0.255	-0.026	0.014	0.053	-0.001	0.013	0.934	0.011	0.013	0.397
BL Executive Function	2	0.006	0.013	0.664	-0.009	0.013	0.495	0.008	0.013	0.526	-0.008	0.013	0.532
BL Executive Function	3	0.01	0.017	0.543	0	0.015	0.979	0.006	0.015	0.709	-0.003	0.014	0.829
BL Culture Fair	1	-0.139	0.07	0.047	-0.164	0.07	0.019	-0.088	0.071	0.211	-0.031	0.071	0.668

Intelligence Test													
BL Culture Fair Intelligence Test	2	-0.149	0.072	0.038	-0.095	0.07	0.172	-0.057	0.07	0.411	-0.084	0.07	0.23
BL Culture Fair Intelligence Test	3	-0.093	0.095	0.327	-0.059	0.083	0.476	-0.035	0.081	0.662	-0.104	0.074	0.163
BL Processing Speed	1	0.009	0.012	0.459	0.022	0.012	0.075	0.009	0.013	0.471	0.008	0.013	0.546
BL Processing Speed	2	0.007	0.013	0.59	0.023	0.013	0.066	0.01	0.013	0.437	0.014	0.013	0.308
BL Processing Speed	3	-0.001	0.016	0.951	0.01	0.015	0.493	0.007	0.015	0.649	0.008	0.014	0.549
BL Speech-in-noise Threshold	1	-0.127	0.072	0.08	0.131	0.069	0.056	0.034	0.07	0.624	-0.074	0.066	0.265
BL Speech-in-noise Threshold	2	-0.158	0.075	0.035	0.054	0.07	0.44	-0.025	0.071	0.73	-0.041	0.067	0.536
BL Speech-in-noise Threshold	3	-0.102	0.09	0.258	-0.015	0.08	0.847	-0.016	0.08	0.842	-0.059	0.07	0.399
F1 SDQ	1	0.212	0.117	0.07	0.14	0.116	0.228	-0.009	0.115	0.939	-0.049	0.116	0.671
F1 SDQ	2	0.203	0.121	0.092	-0.001	0.105	0.994	-0.128	0.102	0.21	0.088	0.101	0.386
F1 SDQ	3	0.19	0.148	0.199	-0.065	0.121	0.588	-0.075	0.113	0.505	-0.008	0.107	0.938
F1 Internalising	1	0.065	0.07	0.354	0.056	0.069	0.42	-0.018	0.069	0.799	0.029	0.07	0.682
F1 Internalising	2	0.055	0.072	0.448	0.011	0.064	0.866	-0.057	0.062	0.353	0.099	0.062	0.107

F1 Internalising	3	0.08	0.09	0.374	-0.018	0.073	0.807	-0.052	0.068	0.447	0.073	0.065	0.261
F1 Externalising	1	0.147	0.074	0.048	0.084	0.073	0.255	0.009	0.073	0.904	-0.078	0.074	0.291
F1 Externalising	2	0.044	0.07	0.534	-0.009	0.067	0.891	-0.07	0.065	0.286	-0.008	0.065	0.897
F1 Externalising	3	0.031	0.087	0.721	-0.044	0.078	0.574	-0.029	0.073	0.692	-0.068	0.069	0.322
F1 PHQ-9 Depression	1	0.107	0.123	0.386	0.099	0.12	0.411	-0.017	0.122	0.892	-0.025	0.133	0.851
F1 PHQ-9 Depression	2	-0.035	0.131	0.788	0.066	0.123	0.589	-0.082	0.122	0.503	0.124	0.132	0.345
F1 PHQ-9 Depression	3	0.003	0.155	0.985	0.123	0.141	0.383	-0.044	0.133	0.74	0.14	0.136	0.303
F1 GAD-7 Anxiety	1	0.172	0.105	0.102	0.178	0.103	0.083	0.094	0.105	0.37	-0.015	0.114	0.894
F1 GAD-7 Anxiety	2	0.105	0.111	0.346	0.187	0.104	0.073	0.078	0.103	0.45	0.078	0.111	0.481
F1 GAD-7 Anxiety	3	0.12	0.128	0.353	0.205	0.117	0.082	0.085	0.111	0.445	0.082	0.114	0.47
F1 Executive Function	1	0.018	0.013	0.165	0.004	0.013	0.736	0.038	0.012	0.002	-0.02	0.012	0.101
F1 Executive Function	2	0.027	0.012	0.02	0.012	0.011	0.287	0.036	0.011	0.001	-0.034	0.011	0.002
F1 Executive Function	3	0.021	0.014	0.156	0.012	0.013	0.333	0.023	0.012	0.055	-0.014	0.011	0.231
F1 Culture Fair Intelligence Test	1	-0.091	0.075	0.223	-0.098	0.073	0.182	0.036	0.072	0.615	-0.06	0.075	0.426
F1 Culture Fair Intelligence Test	2	-0.002	0.069	0.981	-0.014	0.065	0.825	0.034	0.063	0.585	-0.073	0.064	0.258

F1 Culture Fair Intelligence Test	3	0.028	0.082	0.737	-0.018	0.073	0.81	0.046	0.069	0.503	-0.072	0.067	0.282
F1 Processing Speed	1	0.039	0.013	0.002	0.044	0.012	0	0.02	0.013	0.121	0.025	0.013	0.063
F1 Processing Speed	2	0.027	0.013	0.037	0.03	0.012	0.014	0.011	0.012	0.353	0.017	0.012	0.165
F1 Processing Speed	3	0.014	0.016	0.378	0.017	0.014	0.222	0.004	0.014	0.758	0.012	0.013	0.357
F1 Speech-in-noise Threshold	1	0.057	0.096	0.553	0.101	0.095	0.289	0.007	0.096	0.94	-0.042	0.091	0.642
F1 Speech-in-noise Threshold	2	-0.09	0.105	0.388	0.021	0.099	0.832	-0.073	0.097	0.45	0.069	0.091	0.449
F1 Speech-in-noise Threshold	3	-0.176	0.125	0.16	-0.062	0.112	0.584	-0.129	0.107	0.225	0.01	0.097	0.92
F2 Psychosis Risk	1	0.20	0.25	0.43	0.10	0.28	0.71	-0.05	0.27	0.85	-0.28	0.28	0.31
F2 Psychosis Risk	2	0.28	0.28	0.32	0.26	0.31	0.40	0.05	0.31	0.88	-0.08	0.33	0.80
F2 Psychosis Risk	3	0.28	0.28	0.32	0.27	0.32	0.40	0.05	0.32	0.87	-0.09	0.35	0.79

XIX: Non-linear associations between exposure to air pollution or noise and psychological outcomes in the SCAMP cohort (generalised additive model plots, presented when the non-linear association was a significantly better fit than the linear model)

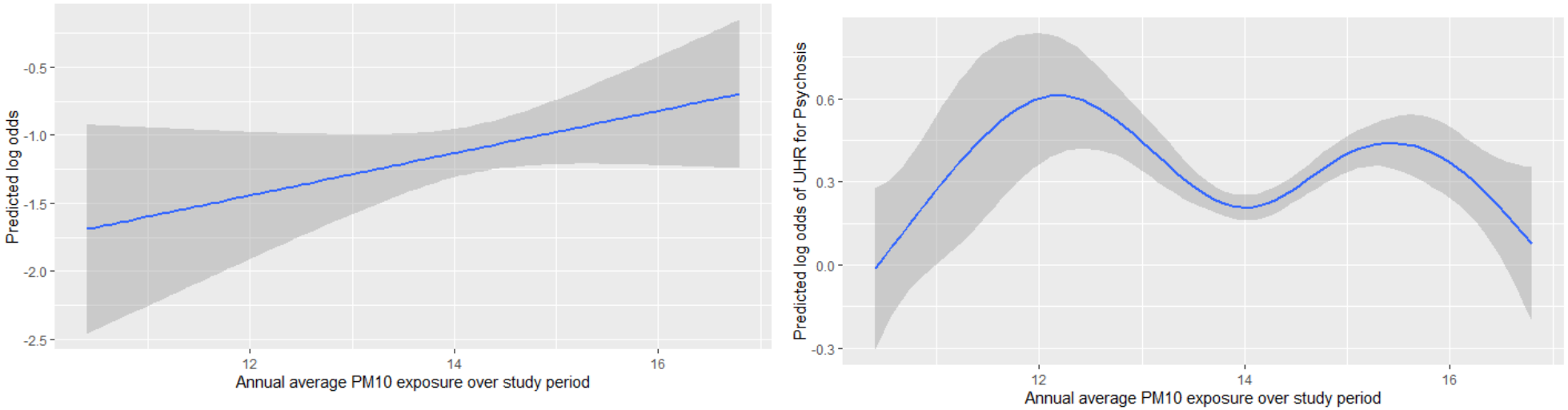


Figure A1. Predicted relationship between PM_{10} exposure across the study period and predicted risk of psychotic symptoms (a) in an adjusted linear model, (b) in an adjusted GAM

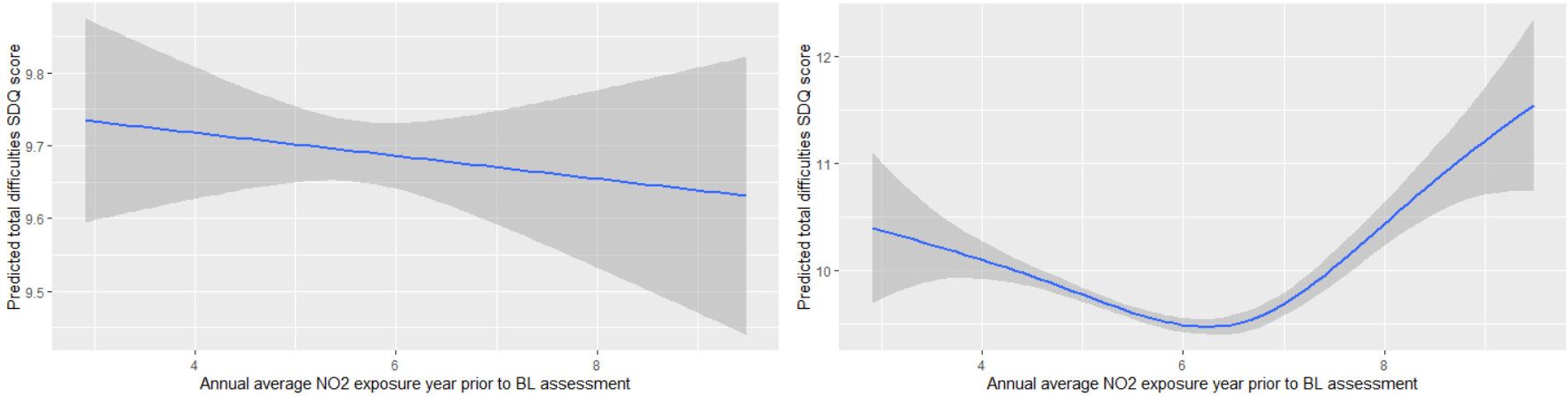


Figure A2. Predicted relationship between NO_2 in the year prior to baseline (BL) assessment and baseline SDQ-assessed behavioural problems (a) in an adjusted linear model, (b) in an adjusted GAM

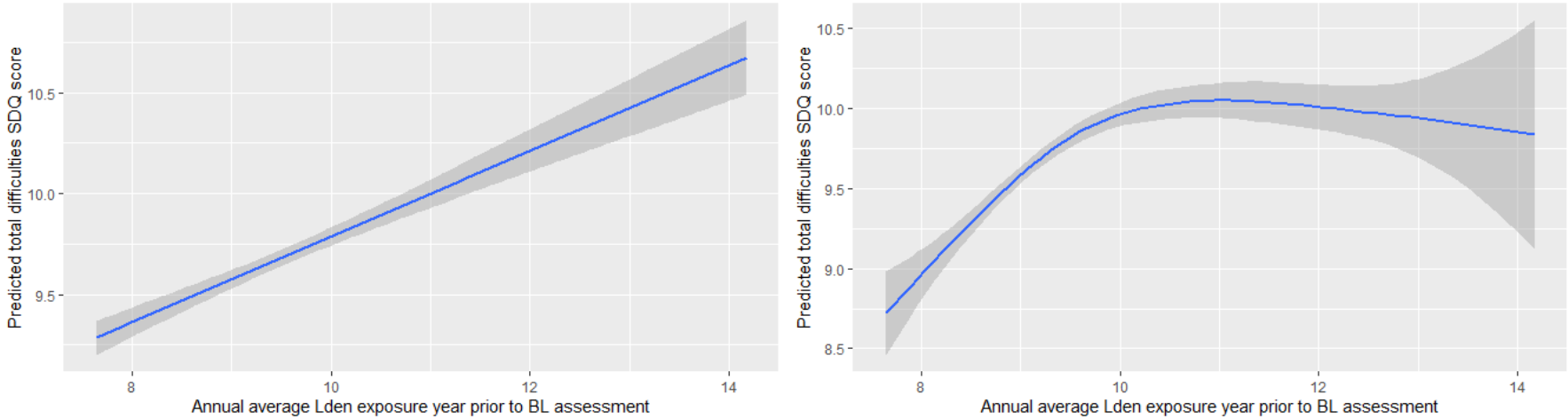


Figure A3. Predicted relationship between Lden in the year prior to baseline (BL) assessment and baseline SDQ-assessed behavioural problems (a) in an adjusted linear model, (b) in an adjusted GAM

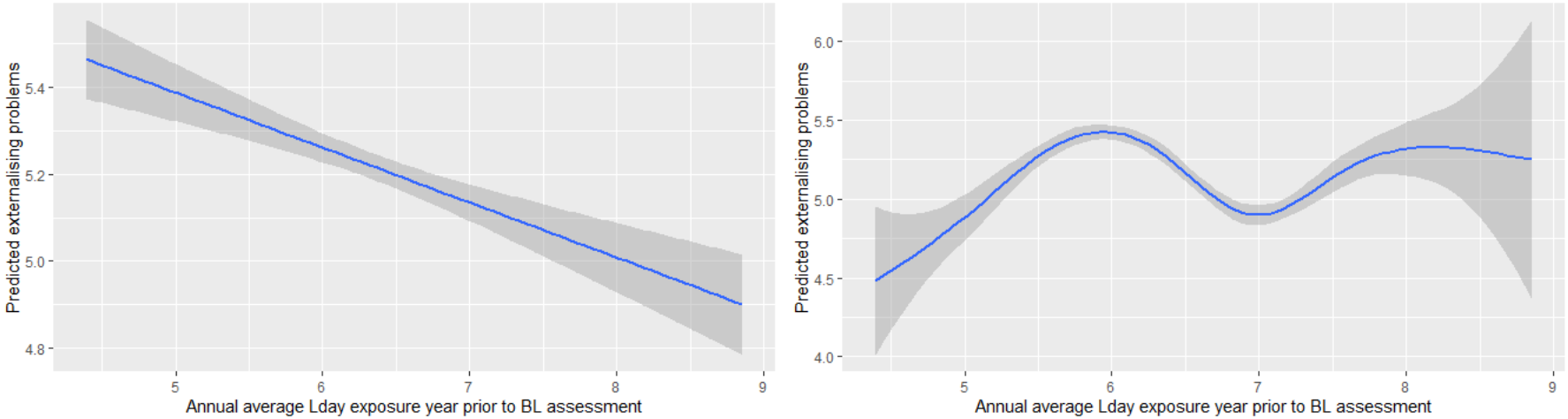


Figure A4. Predicted relationship between Lday in the year prior to baseline (BL) assessment and baseline SDQ-assessed externalising problems (a) in an adjusted linear model, (b) in an adjusted GAM

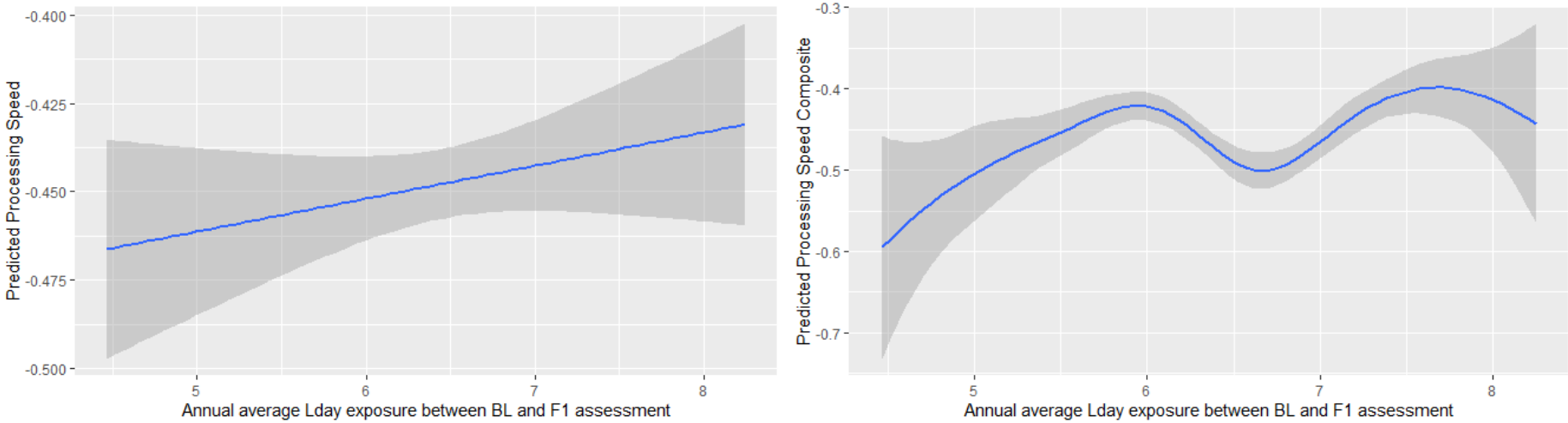


Figure A5. Predicted relationship between Lday exposure between baseline (BL) and follow-up (F1) assessment and follow-up processing speed (a) in an adjusted linear model, (b) in an adjusted GAM