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1 **Air and noise pollution exposure in early life and mental health from**
2 **adolescence to young adulthood**

3
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28 **Key Points**

29 **Question:** Is exposure to air and noise pollution in pregnancy, childhood, and adolescence
30 associated with the development of psychotic experiences, depression, and anxiety between
31 ages 13 to 24?

32 **Findings:** In this longitudinal birth cohort followed into adulthood (original sample:
33 $N > 14,000$; sample with mental health data: $N > 9,000$), higher $PM_{2.5}$ exposure in pregnancy
34 and childhood was associated with psychotic experiences and depression, and higher noise
35 pollution exposure in childhood and adolescence was associated with anxiety.

36 **Meaning:** The findings build on evidence associating air and noise pollution with mental
37 health, by highlighting a role of early-life pollution exposure in youth mental health
38 problems.

39

40 **Abstract**

41 **Importance:** Growing evidence associates air pollution exposure with various psychiatric
42 disorders. However, the importance of early-life (e.g., prenatal) exposure to youth mental
43 health is poorly understood. Moreover, few longitudinal studies have investigated the
44 association of noise pollution with youth mental health.

45 **Objectives:** To examine the longitudinal associations of air and noise pollution exposure in
46 pregnancy, childhood, and adolescence with psychotic experiences, depression, and anxiety
47 from ages 13-24. We hypothesized that participants exposed to higher air and noise pollution
48 would subsequently have more psychotic experiences, depression, and/or anxiety.

49 **Design:** The Avon Longitudinal Study of Parents and Children (ALSPAC) is an ongoing
50 longitudinal birth cohort founded in the 1990s.

51 **Setting:** A population-based study in Southwest England, United Kingdom.

52 **Participants:** The cohort includes over 14,000 babies with due dates between 1 April 1991
53 and 31 December 1992; subsequently followed into adulthood.

54 **Exposures:** A novel linkage (completed in 2020) was performed to link high-resolution
55 (100m^2) estimates of nitrogen dioxide (NO_2), fine particulate matter <2.5 microns ($\text{PM}_{2.5}$),
56 and noise pollution to home addresses from pregnancy to age 12.

57 **Main outcomes and measures:** Psychotic experiences, depression, and anxiety were
58 measured at ages 13, 18, and 24. Logistic regression models controlled for key individual-,
59 family-, and area-level confounders.

60 **Results:** The study included 9,065 participants who had any mental health data, of whom
61 51.4% ($N=4,657$) were female, 95.8% ($N=7,616$) were of White ethnicity, and 19.5%
62 ($N=1,544$), 11.4% ($N=947$), and 9.7% ($N=811$) reported psychotic experiences, depression,

63 and anxiety, respectively. After covariate adjustment, interquartile range increases
64 ($0.72\mu\text{g}/\text{m}^3$) in $\text{PM}_{2.5}$ during pregnancy and childhood were associated with 11% and 9%
65 elevated odds for psychotic experiences (pregnancy: adjusted [a]OR=1.11, 95% CI=1.04-
66 1.19, $p=0.002$; childhood: aOR=1.09, 95% CI=1.00-1.10, $p=0.04$). Pregnancy $\text{PM}_{2.5}$ exposure
67 was also associated with depression (aOR=1.10, 95% CI=1.02-1.18, $p=0.01$). Conversely,
68 higher noise pollution exposure in childhood (aOR=1.19, 95% CI=1.03-1.38, $p=0.02$) and
69 adolescence (aOR=1.22, 95% CI=1.02-1.45, $p=0.03$) was associated with elevated odds for
70 anxiety.

71 **Conclusions and relevance:** In this longitudinal cohort study, early-life air and noise
72 pollution exposure were prospectively associated with three common mental health problems
73 from adolescence to young adulthood. There was a degree of specificity in terms of pollutant-
74 timing-outcome associations.

75

76

77 **Introduction**

78 Childhood, adolescence, and early adulthood are critical periods for the development of
79 psychiatric disorders: worldwide, nearly two-thirds of those affected become unwell by age
80 25.¹ Identifying early-life risk factors is a crucial research challenge in order to develop
81 preventative interventions and improve lifelong mental health trajectories.

82 Growing evidence suggests that air pollution exposure may contribute to the onset of
83 psychiatric problems, including mood, affective, and psychotic disorders.²⁻⁶ Air pollution
84 comprises toxic gases and particulate matter (i.e., organic and inorganic solid and liquid
85 aerosols), of mostly anthropogenic origin.⁷ Understanding the potential impact of air
86 pollution on mental health is increasingly crucial, given the human and societal cost of poor
87 mental health,⁸ given the global shift towards urban living,^{9,10} and given the backdrop of
88 emissions-induced climate change.¹¹

89 Air pollution could negatively affect mental health via numerous pathways, including
90 by compromising the blood-brain barrier, promoting neuroinflammation and oxidative stress,
91 and directly entering the brain and damaging tissue therein.^{12,13} However, key research gaps
92 remain. First, the relative importance of early-life exposure, including prenatal exposure, is
93 uncertain. Babies and children are thought to be especially vulnerable to air pollution,^{14,15} but
94 longitudinal, high-resolution pollution data spanning the early years are scarce. Second,
95 relatively few studies have examined the association of air pollution with youth mental health
96 problems,¹⁶ despite youth being a critical period for intervention. Third, few longitudinal
97 studies have investigated the role of noise pollution in mental health,¹⁷ despite the correlation
98 between noise and air pollution.¹⁸ Finally, studies have often used crude pollution data and
99 lacked adequate controls for potential confounders..

100 We aimed to advance understanding on this topic by capitalizing on a novel linkage
101 between high-resolution outdoor air and noise pollution data and a cohort of over 14,000
102 babies born in Southwest England in 1991-1993 and followed into adulthood. We examined
103 the association of air and noise pollution exposure from pregnancy to age 12 with mental
104 health problems from ages 13-24. Based on previous evidence, we focussed on psychotic
105 experiences (e.g., subclinical hallucinations and delusions), depression, and anxiety. These
106 problems are common^{1,19-21} and increasing²² among youth, and strongly predict future
107 psychopathology,^{23,24} making them useful and important targets. We hypothesized that
108 participants exposed to higher air and noise pollution would subsequently experience worse
109 mental health.

110

111 **Methods**

112 *Participants*

113 The Avon Longitudinal Study of Parents and Children (ALSPAC) is a UK birth cohort,²⁵⁻²⁸
114 described further in the Supplementary Materials. Briefly, pregnant women residing in and
115 around the City of Bristol (population ~714,000 in 2024) in Southwest England with due
116 dates between 01/04/91 and 31/12/92 were approached to take part in the study. The initial
117 number of pregnancies enrolled was 14,551, resulting in 13,988 children alive at 1 year of
118 age. At age 7, the initial sample was bolstered with additional eligible cases, resulting in
119 14,901 babies alive at 1 year of age. The catchment area has a mix of urban, suburban, and
120 rural environments.²⁹ The study website contains details of all the data and a fully searchable
121 data dictionary and variable search tool: [http://www.bristol.ac.uk/alspac/researchers/our-](http://www.bristol.ac.uk/alspac/researchers/our-data/)
122 [data/](http://www.bristol.ac.uk/alspac/researchers/our-data/). Ethical approval for the study was obtained from the ALSPAC Ethics and Law
123 Committee and the Local Research Ethics Committees. Informed consent for the use of data

124 collected via questionnaires and clinics was obtained from participants following the
125 recommendations of the ALSPAC Ethics and Law Committee at the time. The present study
126 is reported according to STROBE guidelines.³⁰

127

128

129 *Psychotic experiences*

130 Psychotic experiences were measured at ages 13, 18, and 24, using a semi-structured
131 interview³¹ which consisted of 12 core items about hallucinations, delusions, and thought
132 interference, rated against the Schedule for Clinical Assessment in Neuropsychiatry version
133 2.0 (SCAN 2.0).³² Consistent with previous ALSPAC studies,^{33,34} psychotic experiences were
134 defined such that 0=none and 1=suspected/definite. The reporting period at each phase was
135 since the participant's 12th birthday. At ages 13, 18, and 24, 13.6% (N=926), 9.2% (N=432),
136 and 12.6% (N=491), reported psychotic experiences, respectively. We summed psychotic
137 experiences across timepoints and dichotomized the variable for analyses, such that
138 participants were scored as 1=suspected/definite psychotic experiences if they reported
139 psychotic experiences at any age.

140

141 *Depression and anxiety*

142 Depression and anxiety were measured at age 13 via parent-completed Development and
143 Well-being Assessments (DAWBA).³⁵ Responses were classified into probabilistic bands
144 according to DSM-IV criteria for major depressive disorder (MDD) and generalized anxiety
145 disorder (GAD), and dichotomized for analysis (bands 0-2=0, bands 3-5=1). At ages 18 and
146 24, depression and anxiety were measured using the Clinical Interview Schedule Revised

147 (CIS-R),³⁶ a self-administered computerized interview that gave ICD-10 diagnoses of
148 moderate-severe depression and GAD. The reporting period at each phase was the past
149 month, although a 6-month reporting period was used for anxiety at age 13. At ages 13, 18,
150 and 24, 5.6% (N=386), 7.9% (N=359), and 7.7% (N=304) reported depression; and 3.6%
151 (N=254), 5.8% (N=262), and 9.8% (N=386) reported anxiety, respectively. We summed
152 depression and anxiety across timepoints and dichotomized the variables for analysis, such
153 that participants were scored as 1=depression/anxiety if they had depression/anxiety at any
154 age.

155

156 *Air pollution*

157 Air pollutants included nitrogen dioxide (NO₂) and fine particulate matter with a diameter of
158 <2.5 microns (PM_{2.5}). Both pollutants have well-established health impacts¹⁰ and more recent
159 associations with psychiatric disorders.⁵ These air pollutants were estimated as part of the
160 LifeCycle project³⁷ using the Effects of Low-Level Air Pollution: A Study in Europe
161 (ELAPSE) model, which is described elsewhere and further in the Supplementary
162 Materials.³⁸ Briefly, the ELAPSE model is a hybrid land-use regression model for Europe
163 which derived concentrations of NO₂ and PM_{2.5} in 2010. The model produces annualized
164 estimates at 100m² resolution, explaining 59% and 71% of measured spatial variability for
165 NO₂ and PM_{2.5}, respectively.³⁸ Estimates were linked to residential geocodes from pregnancy
166 to age 12 for participants who had lived in the original ALSPAC catchment area²⁹ up to age
167 12 and provided permission for geospatial linkage. Linkage was completed in 2020.

168

169 *Noise pollution*

170 Residential noise pollution exposure was also estimated as part of the LifeCycle project,³⁷
171 based on the UK Government's Department for Environment, Food & Rural Affairs
172 (DEFRA) 2006 road traffic noise map. Data represent an annualized average of day and night
173 noise pollution, categorized according to low-medium (<55 decibels: the European
174 Environment Agency's threshold³⁹), high (55-60 decibels), and very high noise (>60
175 decibels). eFigure 1 in the Supplementary Materials shows the correlation between noise
176 pollution, NO₂, and PM_{2.5} across timepoints.

177

178 *Covariates*

179 Potential confounders were informed by the literature and formally selected using a directed
180 acyclic graph (DAG: eFigure 2). We considered individual-/family-level covariates that could
181 be associated with mental health problems and with downward mobility into more polluted
182 neighborhoods. These included ethnicity, family psychiatric history, maternal social class,
183 maternal education, and housing tenure. Area-level covariates included population density,
184 neighborhood deprivation, social fragmentation, and greenspace, and were time-varying,
185 corresponding to the timing of pollution exposure. Covariates are described fully in the
186 Supplementary Materials and briefly below.

187 *Individual-/family-level covariates.* Ethnicity of the child was reported by mothers during
188 pregnancy. Family psychiatric was reported by mothers and fathers during pregnancy and
189 defined as the presence of any psychiatric problem affecting the mother, father, or any
190 biological grandparent. Maternal social class was reported by mothers during pregnancy
191 based on occupation. Maternal education was reported by mothers when babies were around
192 8 months. Home ownership was reported by mothers during pregnancy.

193 *Neighborhood-level covariates.* Population density was derived from 1991/2001 census
194 data.³⁴ Area-level deprivation was based on the 2000 Index of Multiple Deprivation.⁴⁰ Social
195 fragmentation was based on a z-scored sum of census data on residential mobility, marital
196 status, single person households, and home ownership.³⁴ Greenspace was assessed based on
197 the Normalized Difference Vegetation Index.⁴¹

198

199 *Statistical analysis*

200 Analyses were performed in Stata v18.0. Code can be found at
201 <https://github.com/JBNewbury/bris-phs-pollution-mental-health.git>. The characteristics of the
202 sample with versus without mental health data were described according to percentages,
203 means, and standard deviations. Group differences were explored using Chi-square and t-
204 tests. To explore the importance of different exposure periods, we derived exposure estimates
205 for three developmental stages: a) pregnancy, b) childhood (birth to age 9), and c)
206 adolescence (ages 10-12),⁴² which were calculated using mean exposure values for NO₂,
207 PM_{2.5}, and noise pollution during these age windows. Given that NO₂ and PM_{2.5} had very
208 different absolute ranges, scores were standardized by dividing by the interquartile range
209 (IQR). To aid comparison between air and noise pollution, we treated noise pollution as a
210 continuous variable, assuming a normal distribution underlying the categorical variable.
211 Results treating noise as categorical are reported in the Supplementary Materials.

212 For main analyses, logistic regression was used to examine the associations of NO₂,
213 PM_{2.5}, and noise pollution in pregnancy, childhood, and adolescence with the mental health
214 outcomes. We ran an unadjusted model (Model 1), then adjusted for individual-/family-level
215 covariates (Model 2), and then additionally adjusted for area-level covariates (Model 3). To
216 better understand the independent associations from different exposure periods, we then

217 adjusted childhood and adolescent exposure for previous exposure (Model 4). However,
218 given that the high correlation between pollutants over time (eFigure 1) could introduce
219 multi-collinearity, we interpret Model 4 with caution. To estimate residual confounding, we
220 also calculated e-values⁴³ for Models 3 and 4, which indicate the strength of association that
221 an unmeasured confounder would require to nullify associations. All models accounted for
222 potential hierarchy in the data by clustering around the Lower Layer Super Output Area
223 (LSOA's contain ~1,500 residents on average) using the "cluster" command, which provides
224 robust standard errors adjusted for within cluster correlated data.⁴⁴ All analyses were
225 conducted following multiple imputation by chained equations,⁴⁵ described in the
226 Supplementary Materials.

227 We conducted three sensitivity analyses. First, we analysed NO₂, PM_{2.5}, and noise
228 pollution simultaneously, to control each for the others and address potential co-pollutant
229 confounding. Second, we restricted analyses to participants who did not move house from
230 pregnancy to age 12 (29.8%), to keep pollution levels as consistent over time as possible.
231 Third, we repeated main analyses for those with complete data.

232

233 **Results**

234 **Sample characteristics**

235 The study included 9,065 participants who had any mental health data, of whom 51.4%
236 (N=4,657) were female, 95.8% (N=7,616) were ethnically White, and 19.5% (N=1,544),
237 11.4% (N=947), and 9.7% (N=811) reported psychotic experiences, depression, and anxiety,
238 respectively (Table 1). Over half the sample (60.8%; N=4,793) had a family psychiatric
239 history; 21.8% (N=1,583) had mothers who worked in manual occupations; 15.7% (N=1,274)
240 had mothers with degrees; and 81.6% (N=6,670) lived in homes owned by their parent(s).

241 Mean population density was 34 persons per hectare (SD=20.7) and 23.7% (N=1,754) lived
242 in the most deprived neighborhoods. The sample with and without mental health data differed
243 for most variables: those with data were more likely to be female, White, have a family
244 psychiatric history, and have more advantaged characteristics across the other variables.
245 These differences should be borne in mind when interpreting the results.

246

247 **Air and noise pollution exposure**

248 Figure 1 shows estimated levels of NO₂ and PM_{2.5} for the sample, alongside the World Health
249 Organization's (WHO) 2021 exposure thresholds.⁴⁶ Mean levels of NO₂ and PM_{2.5} decreased
250 slightly over time. However, average exposure at age 12 remained above the WHO's
251 thresholds for both pollutants. Additionally, over two-thirds of participants were exposed to
252 high/very high noise pollution,³⁹ which changed little over time (Figure 1).

253

254 **Associations of air and noise pollution with mental health**

255 Associations of NO₂, PM_{2.5}, and noise pollution with psychotic experiences, depression, and
256 anxiety are shown in Table 2, which shows unadjusted and adjusted results alongside e-
257 values; and Figure 2, which is fully adjusted.

258 Before covariate adjustment, IQR (4.47µg/m³) increases in NO₂ during pregnancy
259 were associated with 8% elevated odds for psychotic experiences (OR=1.08, 95% CI=1.00-
260 1.17, p=0.04). However, this was attenuated to the null after adjusting for area-level
261 covariates. In contrast, following covariate adjustment, IQR (0.72µg/m³) increases in PM_{2.5}
262 during pregnancy and childhood were associated with 11% and 9% elevated odds for
263 psychotic experiences, respectively (pregnancy: adjusted [a]OR=1.11, 95% CI=1.04-1.19,

264 p=0.002); childhood: aOR=1.09, 95% CI=1.00-1.19, p=0.04); although childhood exposure
265 was attenuated to the null after adjusting for pregnancy exposure. There was little evidence of
266 an association between noise pollution and psychotic experiences.

267 Following covariate adjustment, IQR increases in PM_{2.5} during pregnancy were
268 associated with 10% elevated odds for depression (aOR=1.10, 95% CI=1.02-1.18, p=0.01).
269 There was little evidence of associations between NO₂, noise pollution, and depression.

270 Before covariate adjustment, IQR increases in NO₂ in pregnancy and childhood were
271 associated with 14% (OR=1.14, 95% CI=1.04-1.26, p=0.006) and 15% (OR=1.15, 95%
272 CI=1.03-1.27, p=0.009) elevated odds for anxiety, respectively, but associations were
273 attenuated to the null after adjusting for area-level covariates. There was little evidence
274 associating PM_{2.5} with anxiety. In contrast, participants exposed to higher noise pollution in
275 childhood and adolescence had 19% and 22% elevated odds for anxiety, respectively
276 (childhood: aOR=1.19, 95% CI=1.03-1.38, p=0.02; adolescence: aOR=1.22, 95% CI=1.02-
277 1.45, p=0.03); although adolescent exposure was attenuated to the null after controlling for
278 pregnancy and childhood exposure. eTable 1 displays results when noise pollution was
279 treated as categorical. This highlighted several dose-response associations, though no
280 difference in model fit compared to the main results.

281

282 **E-values**

283 In eTables 2 and 3 in the Supplementary Materials, we take as examples the adjusted
284 associations of a) pregnancy PM_{2.5} with psychotic experiences and b) adolescent noise
285 pollution with anxiety; and compare the e-values to the associations from included covariates.
286 E-values were a) 1.46 (lower confidence limit [LCL]=1.24), and b) 1.74 (LCL=1.16),
287 respectively. These were larger in magnitude than the associations of the covariates with the

288 exposures and outcomes, indicating that an unmeasured confounder would require a
289 relatively strong confounding influence to nullify associations.

290

291 **Sensitivity analyses**

292 Results from sensitivity analyses are presented in eTables 4-6 and are discussed in the
293 Supplementary Materials. Briefly, point estimates were generally similar after adjusting
294 pollutants for each other; similar (and often higher) for those who did not move house; and
295 similar for complete cases: though confidence intervals were often less precise.

296

297 **Discussion**

298 In this longitudinal birth cohort study followed up over ~25 years, participants exposed to
299 higher PM_{2.5} during pregnancy and childhood subsequently experienced more psychotic
300 experiences and (for pregnancy exposure only) depression. In contrast, participants exposed
301 to higher noise pollution in childhood and adolescence subsequently experienced more
302 anxiety. These associations were not explained by numerous potential individual-, family-,
303 and area-level confounders.

304 Our findings suggest an important role of early-life (including prenatal) exposure to
305 air pollution in the development of youth mental health problems. Early-life exposure could
306 be detrimental to mental health given the extensive brain development and epigenetic
307 processes that occur *in utero* and during infancy.^{13,15,47,48} Air pollution exposure could also
308 lead to restricted foetal growth⁴⁹ and preterm birth,⁵⁰ which are both risk factors for
309 psychopathology. Notably, the point estimate for pregnancy PM_{2.5} and depression (10%
310 elevated odds for every 0.72µg/m³ increase) was considerably greater than a previous meta-

311 analytic estimate based on exposure in adulthood (10% elevated odds for every 10 μ g/m³
312 increase).² These contrasting findings are in keeping with a particularly detrimental role of
313 early-life air pollution exposure. However, our findings could also have arisen if early-life
314 exposure data provide a proxy for cumulative exposure over a longer period, given that
315 families often settle when children are young.

316 For noise pollution, evidence was strongest for childhood and adolescent exposure.
317 Childhood and adolescent noise pollution exposure could increase anxiety by increasing
318 stress and disrupting sleep; with high noise potentially leading to chronic physiological
319 arousal and disruption to endocrinology.⁵¹ Noise pollution could also impact cognition,⁵²
320 which could increase anxiety by impacting concentration during school years. It was
321 interesting that noise pollution was associated with anxiety but not with psychotic
322 experiences or depression. However, our measure of noise pollution estimated only decibels
323 (i.e., intensity) from road sources. Other qualities of noise, such as pitch, could be relevant to
324 mental health.

325

326 *Study Limitations*

327 We acknowledge several limitations. First, the causality of the findings is uncertain given that
328 data were observational. Despite comprehensive covariate adjustment, residual confounding
329 is inevitable given imperfect selection and measurement of covariates. The relatively large e-
330 values strengthened our confidence in the findings, but future studies should consider other
331 methods to address confounding, such as quasi-experimental designs. Second, ALSPAC
332 families are more affluent and less diverse than the UK population.⁵³ The extent that our
333 findings generalize to other populations and locations is uncertain. Our findings likely
334 generalize to cities and surrounds in other high-income countries; but may be less

335 generalizable to urban settings in lower-income countries, which can have more extreme
336 pollution concentrations (<https://www.iqair.com/world-air-quality-ranking>). Third, modelled
337 pollution data are subject to various sources of measurement error,³⁸ particularly Berkson-like
338 error whereby estimates are smoother (less variable) than reality, leading to less precise,
339 though unbiased, exposure-outcome estimates.^{54,55} For instance, the 100m² resolution, though
340 an improvement over many previous studies, would have masked hyperlocal variation (e.g.,
341 differences between participants living on adjacent streets), to which NO₂ is especially prone
342 due to its short decay function.⁵⁶ Additionally, the model estimated residential exposure,
343 which would have masked variation due to behaviour and time spent away from home. Finer-
344 resolution data, including personal exposure estimates, would enable more precise exposure-
345 outcome estimates, particularly for NO₂. Fourth, we could not apply life-course models to
346 investigate sensitive periods versus cumulative effects, as there was limited within-person
347 variation in exposure over time. Larger datasets (e.g., national registries) and quasi-
348 experimental designs would be required to further tease out this question.

349

350 *Conclusions*

351 Our study provides novel evidence that early-life exposure to particulate matter is
352 prospectively associated with the development of psychotic experiences and depression in
353 youth. Ours is among only a handful of longitudinal studies investigating the association
354 between noise pollution and mental health, demonstrating an association with anxiety. Our
355 findings suggest a degree of specificity in terms of pollutant-timing-outcome pathways. The
356 opportunity for intervention is potentially enormous. However, though our study addressed
357 various biases affecting observational research, the causality of the findings remains
358 uncertain. Nevertheless, previous post-mortem^{57,58} and recent quasi-experimental^{3,59} studies

359 support a causal role of air pollution in mental health problems. Thus, if we assume causality
360 from this triangulating evidence, interventions to reduce population-level exposure (e.g., low
361 emissions zones in cities) could potentially improve youth mental health. There is now a
362 pressing need for further longitudinal research using more precise measures of air and noise
363 pollution; and for replication using quasi-experimental designs.

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367

368 **Author contributions:**

369 Dr Newbury had full access to all the data in the study and takes responsibility for the
370 integrity of the data and the accuracy of the data analysis. Dr Newbury conceived the study,
371 conducted analyses, and wrote the first draft of the manuscript. Prof Zammit supervised the
372 analyses and write-up and oversaw data collection and assessment scoring in the ALSPAC
373 study. Dr Heron, Dr Bakolis, Prof Kirkbride, and Prof Fisher advised on statistical analyses.
374 Prof Kirkbride oversaw construction of the area-level variables. Mr Boyd and Mr Thomas
375 oversaw the linkage of air and noise pollution data with the ALSPAC study. All authors
376 contributed to the revisions of the manuscript, interpretation of the findings, and approval of
377 the final manuscript.

378

379

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381 The authors declare no conflict of interests.

382

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422

Table 1. Sample characteristics for participants with and without mental health data

Sample characteristics	Sample with mental health data		Sample without mental health data		X ² / T	P-value
	N/M	%/SD	N/M	%/SD		
Psychotic experiences (ages 13-24)						
No	6,579	83.2	NA	-	-	-
Yes	1,544	19.5	NA	-	-	-
Depression (ages 13-24)						
No	7,397	88.7	NA	-	-	-
Yes	947	11.4	NA	-	-	-
Anxiety (ages 13-24)						
No	7,587	90.3	NA	-	-	-
Yes	811	9.7	NA	-	-	-
Sex						
Male	4,394	48.6	3,295	55.0		
Female	4,657	51.4	2,691	45.0	60.9	<0.001
Ethnicity						
All other ethnicities ^a	338	4.3	275	6.6		
White	7,616	95.8	3,906	93.4	31.0	<0.001
Family psychiatric history						
No	3,093	39.2	2,569	80.81		
Yes	4,793	60.8	610	19.19	1600.0	<0.001
Maternal social class ^b						
1 – Professional	295	4.1	73	1.9		
2 – Managerial and technical	2,302	31.8	849	22.0		
3 – Skilled non-manual	3,068	42.3	1,656	42.9		
4 – Skilled manual	264	3.6	188	4.9		
5 – Partly skilled	1,096	15.1	867	22.4		
6 – Unskilled	223	3.1	230	6.0	258.3	<0.001
Maternal education						
1 – Degree	1,274	15.7	334	7.6		
2 – A level	2,087	25.8	706	16.1		
3 – O level	2,850	35.2	1,472	33.6		
4 – Vocational	730	9.0	499	11.4		
5 – CSE	1,152	14.2	1,373	31.3	693.1	<0.001
House tenure						
Mortgaged/owned	6,670	81.6	3,200	60.3		
Rented	1,506	18.4	2,109	39.7	744.6	<0.001
Population density ^c	33.50	20.7	35.16	19.1	4.3	<0.001
Area-level deprivation						
1 – least deprived	1,419	29.4	596	19.7		
2	830	17.2	456	15.0		
3	785	16.3	515	17.0		
4	864	17.9	529	17.4		
5 – most deprived	933	19.3	937	30.9	179.9	<0.001
Social fragmentation ^d	-0.28	2.9	-0.11	2.8	2.9	0.003
Greenspace ^e	0.41	0.1	0.42	0.1	-3.4	<0.001
NO ₂	26.93	4.2	27.08	4.0	2.0	0.047
PM _{2.5}	13.32	0.9	13.38	0.8	3.9	<0.001
Noise pollution						
Low (<55 decibels)	1,594	30.5	1,010	30.1		
Medium (55-60 decibels)	2,442	46.8	1,531	45.6		
High (>60 decibels)	1,185	22.7	817	24.3	3.1	0.213

Note: ^a due to small numbers within most ethnicities, all ethnicities other than White were grouped. These ethnicities included Bangladeshi, Black African, Black Caribbean, Chinese, Indian, Pakistani, and other ethnicities; ^b based on maternal occupation; ^c unit is persons per hectare; ^d sum of z-scored census information on population turnover, unmarried people, single person households, and privately rented households; ^e unit is the Normalized Difference Vegetation Index: range -1 to 1; CSE=certificate of secondary education; M=mean; NO₂=nitrogen dioxide; PM_{2.5}=particulate matter <2.5microns, unit is micrograms per metre squared; SD=standard deviation; T=t-test statistic; χ^2 =Chi-square.

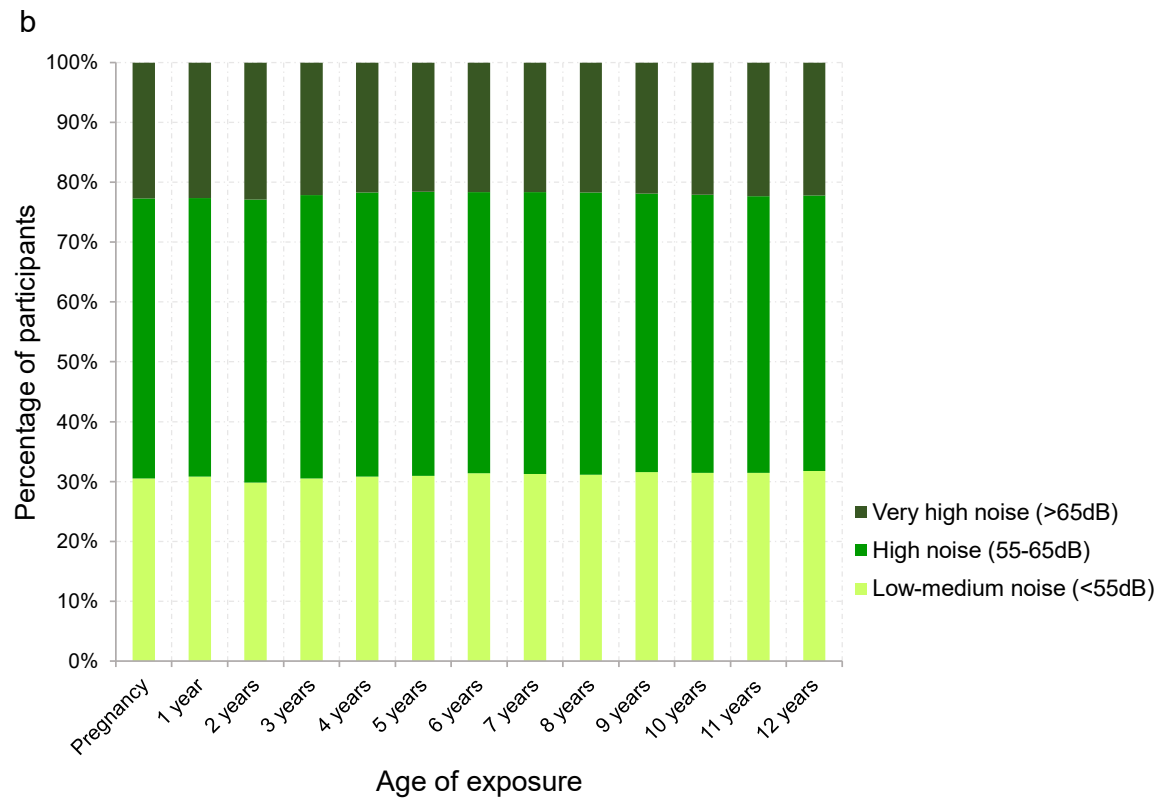
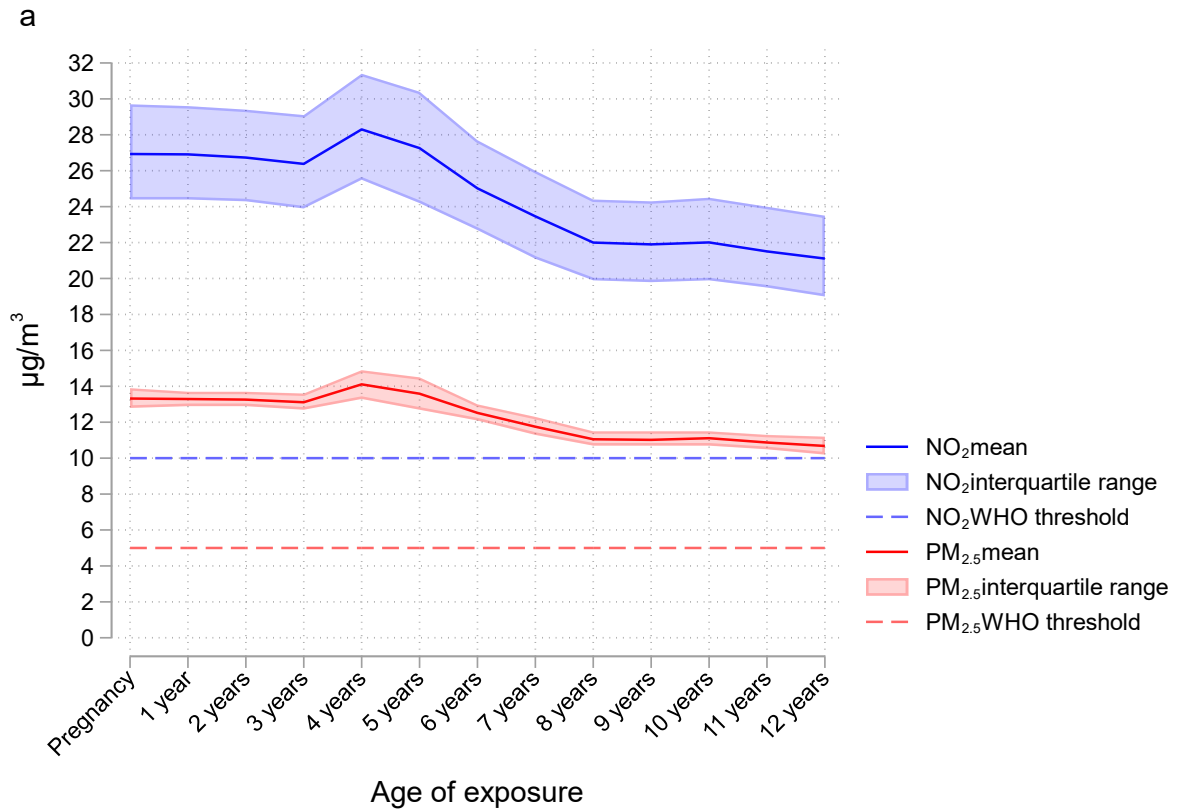


Figure 1. Air and noise pollution exposure in the ALSPAC study sample from pregnancy to age 12

Note: a=air pollution levels; b=noise pollution levels; dB=decibels; NO₂=nitrogen dioxide; PM_{2.5}=particulate matter under 2.5 microns; WHO=World Health Organization; $\mu\text{g}/\text{m}^3$ =micrograms per cubic meter

Table 2. Associations of early-life air and noise pollution exposure with youth mental health problems

Outcome	Pregnancy exposure			Childhood exposure			Adolescence exposure			
	Pollutant	OR (95% CI)	P-value	E-value (LCL)	OR (95% CI)	P-value	E-value (LCL)	OR (95% CI)	P-value	E-value (LCL)
Model										
Psychotic experiences										
NO ₂										
Model 1	1.08 (1.00-1.17)	0.04		1.05 (0.97-1.14)	0.24		1.06 (0.96-1.17)	0.28		
Model 2	1.08 (1.00-1.17)	0.05		1.04 (0.96-1.13)	0.32		1.04 (0.95-1.16)	0.39		
Model 3	1.06 (0.96-1.17)	0.28	1.31 (1.00)	0.97 (0.88-1.07)	0.55	1.21 (1.00)	0.97 (0.85-1.10)	0.58	1.21 (1.00)	
Model 4	NA	-	-	0.89 (0.77-1.03)	0.11	1.50 (1.00)	1.02 (0.81-1.28)	0.89	1.16 (1.00)	
PM _{2.5}										
Model 1	1.11 (1.04-1.18)	0.001		1.11 (1.03-1.19)	0.009		1.09 (0.99-1.21)	0.07		
Model 2	1.11 (1.04-1.18)	0.001		1.10 (1.02-1.19)	0.01		1.09 (0.98-1.20)	0.10		
Model 3	1.11 (1.04-1.19)	0.002	1.46 (1.24)	1.09 (1.00-1.19)	0.04	1.40 (1.00)	1.06 (0.96-1.18)	0.25	1.31 (1.00)	
Model 4	NA	-	-	1.00 (0.90-1.12)	0.93	1.00 (1.00)	1.02 (0.84-1.24)	0.82	1.16 (1.00)	
Noise										
Model 1	1.06 (0.94-1.20)	0.36		1.04 (0.92-1.17)	0.57		1.01 (0.89-1.15)	0.85		
Model 2	1.06 (0.93-1.20)	0.38		1.03 (0.91-1.17)	0.62		1.00 (0.87-1.14)	0.98		
Model 3	1.04 (0.92-1.18)	0.50	1.24 (1.00)	1.01 (0.89-1.14)	0.88	1.11 (1.00)	1.00 (0.87-1.15)	0.99	1.00 (1.00)	
Model 4	NA	-	-	0.95 (0.79-1.15)	0.62	1.29 (1.00)	0.99 (0.81-1.21)	0.90	1.11 (1.00)	
Depression										
NO ₂										
Model 1	1.06 (0.97-1.15)	0.19		1.09 (0.99-1.20)	0.09		1.09 (0.98-1.22)	0.12		
Model 2	1.06 (0.97-1.15)	0.19		1.08 (0.98-1.19)	0.12		1.08 (0.97-1.20)	0.18		
Model 3	1.10 (0.98-1.24)	0.10	1.43 (1.00)	1.11 (0.98-1.26)	0.09	1.46 (1.00)	1.08 (0.94-1.23)	0.28	1.37 (1.00)	
Model 4	NA	-	-	1.09 (0.89-1.33)	0.42	1.40 (1.00)	0.96 (0.72-1.28)	0.77	1.25 (1.00)	
PM _{2.5}										
Model 1	1.07 (1.00-1.15)	0.04		1.06 (0.97-1.14)	0.18		1.02 (0.93-1.12)	0.66		
Model 2	1.07 (1.00-1.15)	0.04		1.05 (0.97-1.14)	0.25		1.01 (0.92-1.11)	0.82		
Model 3	1.10 (1.02-1.18)	0.01	1.43 (1.16)	1.07 (0.98-1.17)	0.15	1.34 (1.00)	0.99 (0.90-1.10)	0.90	1.11 (1.00)	
Model 4	NA	-	-	0.97 (0.86-1.11)	0.69	1.21 (1.00)	0.89 (0.71-1.13)	0.36	1.50 (1.00)	

Noise									
Model 1	1.03 (0.90-1.19)	0.66		1.13 (0.97-1.31)	0.12		1.08 (0.92-1.26)	0.35	
Model 2	1.03 (0.90-1.18)	0.69		1.12 (0.96-1.30)	0.15		1.07 (0.91-1.25)	0.41	
Model 3	1.02 (0.89-1.18)	0.74	1.16 (1.00)	1.12 (0.95-1.31)	0.17	1.49 (1.00)	1.05 (0.89-1.23)	0.58	1.28 (1.00)
Model 4	NA	-	-	1.20 (0.97-1.49)	0.09	1.69 (1.00)	1.06 (0.80-1.40)	0.68	1.31 (1.00)
Anxiety									
NO ₂									
Model 1	1.14 (1.04-1.26)	0.006		1.15 (1.03-1.27)	0.009		1.05 (0.93-1.19)	0.40	
Model 2	1.14 (1.04-1.26)	0.007		1.14 (1.03-1.27)	0.01		1.05 (0.93-1.19)	0.40	
Model 3	1.08 (0.95-1.23)	0.27	1.37 (1.00)	1.10 (0.97-1.25)	0.15	1.43 (1.00)	0.97 (0.83-1.13)	0.73	1.21 (1.00)
Model 4	NA	-	-	0.97 (0.79-1.21)	0.81	1.21 (1.00)	0.77 (0.57-1.03)	0.08	1.92 (1.00)
PM _{2.5}									
Model 1	1.04 (0.97-1.12)	0.22		1.04 (0.96-1.13)	0.34		1.00 (0.91-1.10)	0.98	
Model 2	1.05 (0.98-1.12)	0.19		1.05 (0.96-1.14)	0.30		1.01 (0.91-1.11)	0.90	
Model 3	1.02 (0.95-1.11)	0.55	1.16 (1.00)	1.03 (0.93-1.13)	0.58	1.21 (1.00)	0.98 (0.88-1.09)	0.69	1.16 (1.00)
Model 4	NA	-	-	0.97 (0.84-1.12)	0.67	1.21 (1.00)	0.95 (0.75-1.20)	0.65	1.29 (1.00)
Noise									
Model 1	1.01 (0.88-1.16)	0.86		1.13 (0.98-1.31)	0.09		1.17 (0.98-1.39)	0.08	
Model 2	1.03 (0.90-1.18)	0.67		1.15 (1.00-1.33)	0.06		1.19 (1.00-1.43)	0.05	
Model 3	1.05 (0.91-1.21)	0.51	1.28 (1.00)	1.19 (1.03-1.38)	0.02	1.67 (1.21)	1.22 (1.02-1.45)	0.03	1.74 (1.16)
Model 4	NA	-	-	1.32 (1.04-1.68)	0.02	1.97 (1.24)	0.94 (0.68-1.29)	0.71	1.32 (1.00)

Note: CI=confidence interval; LCL=lower confidence limit. E-values do not include upper confidence limits or p-values; NO₂=nitrogen dioxide; OR=odds ratio; PM_{2.5}=particulate matter under 2.5 microns; Model 1-unadjusted; Model 2-adjusted for individual- and family-level covariates; Model 3-additionally adjusted for area-level covariates; Model 4-additional adjusted for earlier exposure. We interpret Model 4 with caution given that high correlations across timepoints could lead to multi-collinearity; Sample sizes of imputed datasets range from N=2,962 (adolescence noise pollution and psychotic experiences) to N=6,180 (pregnancy air pollution and anxiety).

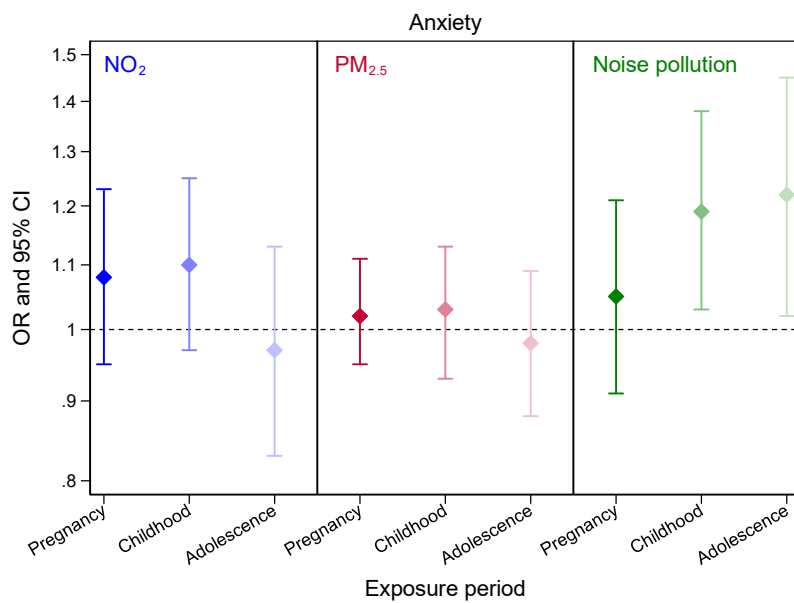
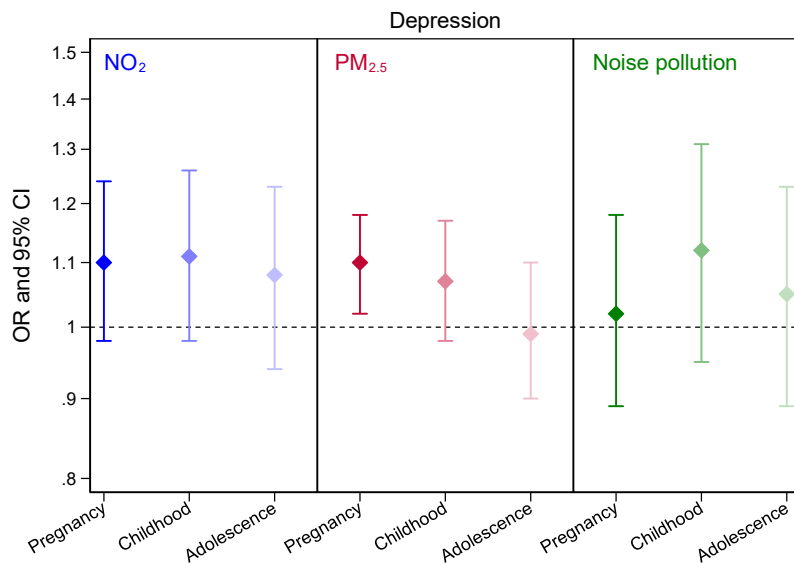
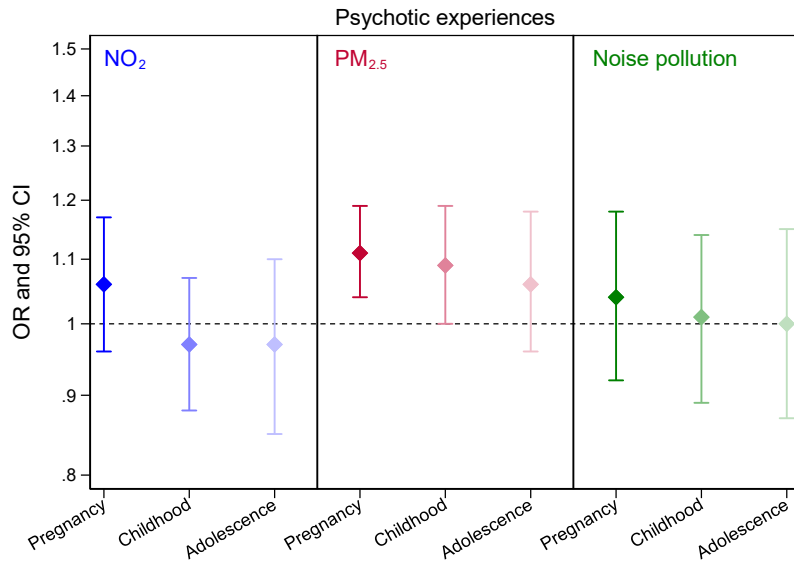


Figure 2. Adjusted associations of early-life air and noise pollution exposure with youth mental health problems

Note: CI=confidence intervals; NO₂=nitrogen dioxide; OR=odds ratio; PM_{2.5}=particulate matter <2.5 microns; Results are from Model 3, which is adjusted for ethnicity, family psychiatric history, maternal social class, maternal education, house tenure, population density, neighborhood deprivation, social fragmentation, and greenspace; Sample sizes of imputed datasets range from N=2,952 (adolescence noise pollution and psychotic experiences) to N=6,154 (pregnancy air pollution and anxiety).

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