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

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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 ($PM_{2.5}$),

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   (0.72μg/m^3) in $PM_{2.5}$ 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 $PM_{2.5}$ 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

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Introduction

Childhood, adolescence, and early adulthood are critical periods for the development of psychiatric disorders: worldwide, nearly two-thirds of those affected become unwell by age 25.[1] Identifying early-life risk factors is a crucial research challenge in order to develop preventative interventions and improve lifelong mental health trajectories.

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

Air pollution could negatively affect mental health via numerous pathways, including by compromising the blood-brain barrier, promoting neuroinflammation and oxidative stress, and directly entering the brain and damaging tissue therein.[12,13] However, key research gaps remain. First, the relative importance of early-life exposure, including prenatal exposure, is uncertain. Babies and children are thought to be especially vulnerable to air pollution,[14,15] but longitudinal, high-resolution pollution data spanning the early years are scarce. Second, relatively few studies have examined the association of air pollution with youth mental health problems,[16] despite youth being a critical period for intervention. Third, few longitudinal studies have investigated the role of noise pollution in mental health,[17] despite the correlation between noise and air pollution.[18] Finally, studies have often used crude pollution data and 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 increasing[22] 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-

122   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]

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128

129 *Psychotic experiences*

130 Psychotic experiences were measured at ages 13, 18, and 24, using a semi-structured

131 interview[31] 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 (NO_2) and fine particulate matter with a diameter of

158 <2.5 microns ($PM_{2.5}$). Both pollutants have well-established health impacts[10] and more recent

159 associations with psychiatric disorders.[5] These air pollutants were estimated as part of the

160 LifeCycle project[37] 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 NO_2 and $PM_{2.5}$ in 2010. The model produces annualized

164 estimates at $100m^2$ resolution, explaining 59% and 71% of measured spatial variability for

165 NO_2 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 area[29] up to age

167 12 and provided permission for geospatial linkage. Linkage was completed in 2020.

168

169 *Noise pollution*

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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 threshold[39]), high (55-60 decibels), and very high noise (>60

175 decibels). eFigure 1 in the Supplementary Materials shows the correlation between noise

176 pollution, NO_2, 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 NO_2,

207 $PM_{2.5}$, and noise pollution during these age windows. Given that NO_2 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 NO_2,

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

adjusted childhood and adolescent exposure for previous exposure (Model 4). However,

given that the high correlation between pollutants over time (eFigure 1) could introduce

multi-collinearity, we interpret Model 4 with caution. To estimate residual confounding, we

also calculated e-values[43] for Models 3 and 4, which indicate the strength of association that

an unmeasured confounder would require to nullify associations. All models accounted for

potential hierarchy in the data by clustering around the Lower Layer Super Output Area

(LSOA's contain ~1,500 residents on average) using the "cluster" command, which provides

robust standard errors adjusted for within cluster correlated data.[44] All analyses were

conducted following multiple imputation by chained equations,[45] described in the

Supplementary Materials.

We conducted three sensitivity analyses. First, we analysed NO_2, $PM_{2.5}$, and noise

pollution simultaneously, to control each for the others and address potential co-pollutant

confounding. Second, we restricted analyses to participants who did not move house from

pregnancy to age 12 (29.8%), to keep pollution levels as consistent over time as possible.

Third, we repeated main analyses for those with complete data.

Results

Sample characteristics

The study included 9,065 participants who had any mental health data, of whom 51.4%

(N=4,657) were female, 95.8% (N=7,616) were ethnically White, and 19.5% (N=1,544),

11.4% (N=947), and 9.7% (N=811) reported psychotic experiences, depression, and anxiety,

respectively (Table 1). Over half the sample (60.8%; N=4,793) had a family psychiatric

history; 21.8% (N=1,583) had mothers who worked in manual occupations; 15.7% (N=1,274)

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 NO_2 and $PM_{2.5}$ for the sample, alongside the World Health

249 Organization's (WHO) 2021 exposure thresholds.[46] Mean levels of NO_2 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 NO_2, $PM_{2.5}$, 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 ($4.47\mu g/m^3$) increases in NO_2 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 ($0.72\mu g/m^3$) 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 NO_2, noise pollution, and depression.

270   Before covariate adjustment, IQR increases in NO_2 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 growth[49] 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/m^3 increase) was considerably greater than a previous meta-

15

311   analytic estimate based on exposure in adulthood (10% elevated odds for every 10μg/m^3

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 100m^2 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 NO$_2$ 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 NO$_2$. 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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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; NO_2=nitrogen dioxide; $PM_{2.5}$=particulate matter <2.5microns, unit is micrograms per metre squared; SD=standard deviation; T=t-test statistic; X^2=Chi-square.
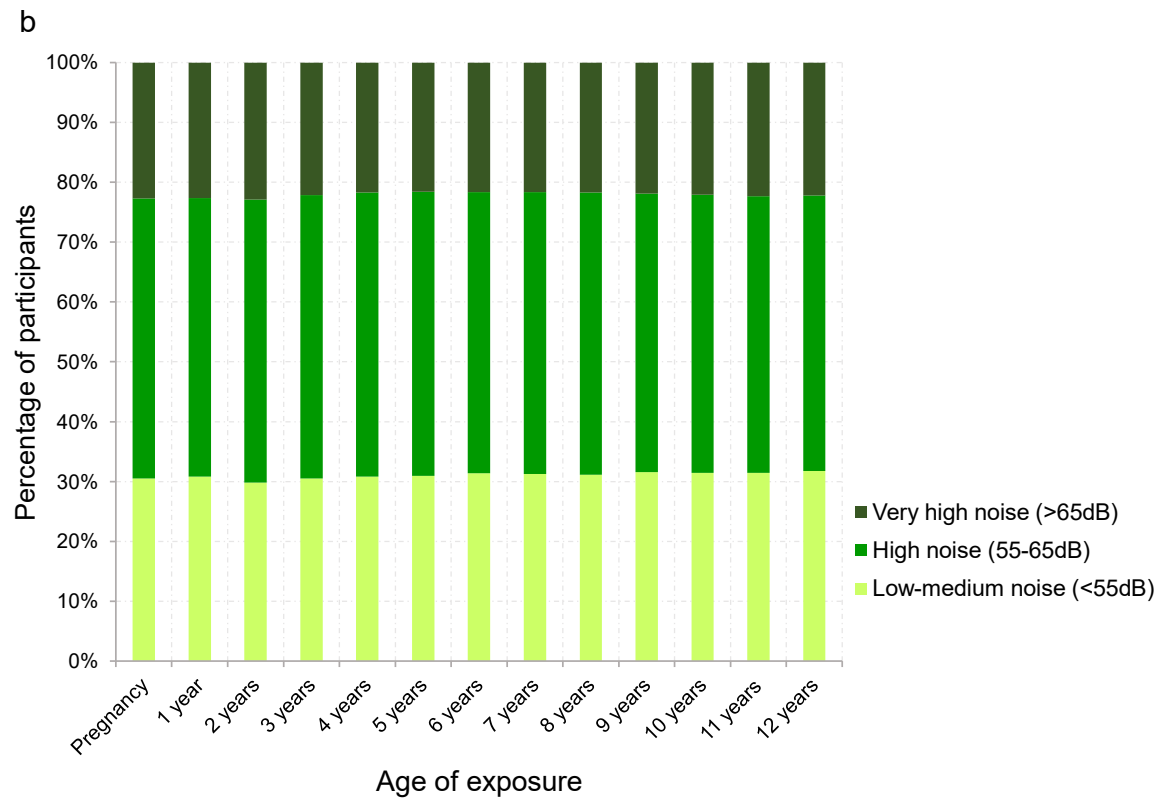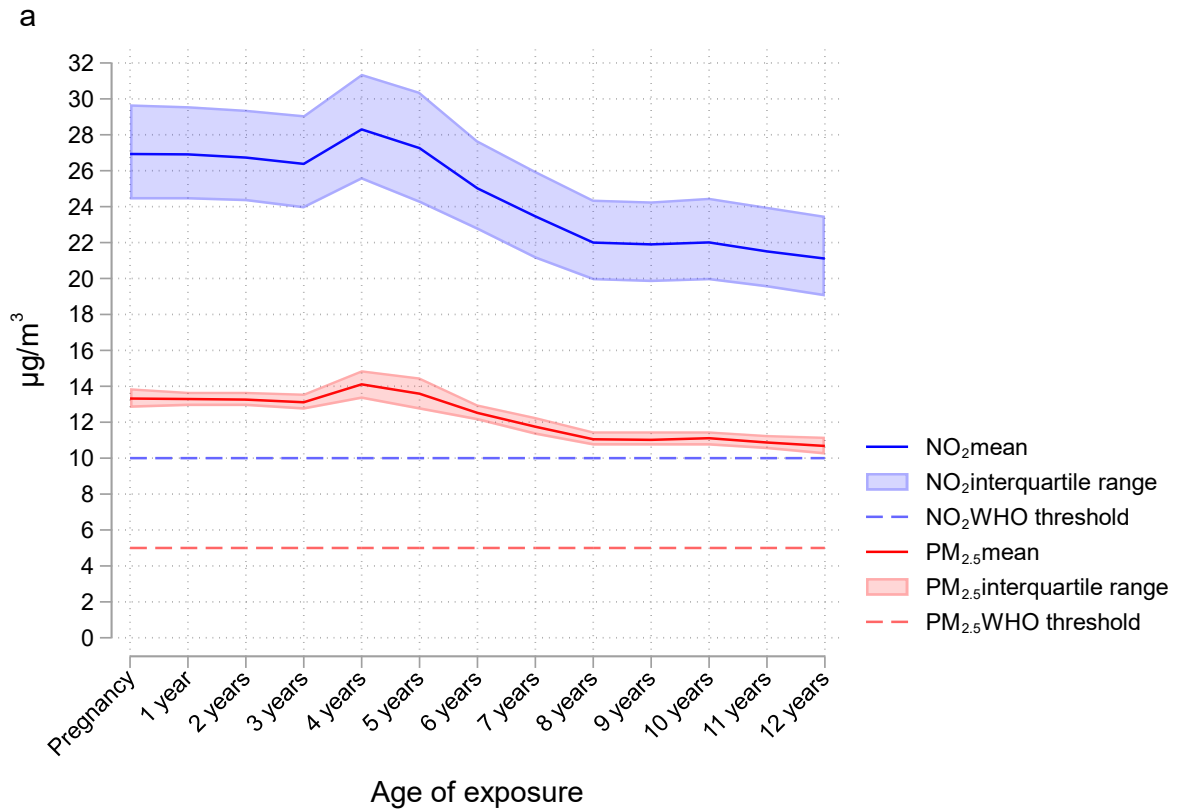
a

b

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; NO$_2$=nitrogen dioxide; PM$_{2.5}$=particulate matter under 2.5 microns; WHO=World Health Organization; µg/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)

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	OR (95% CI)	p	E-value	OR (95% CI)	p	E-value	OR (95% CI)	p	E-value
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; NO$_2$=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).

Psychotic experiences

Depression

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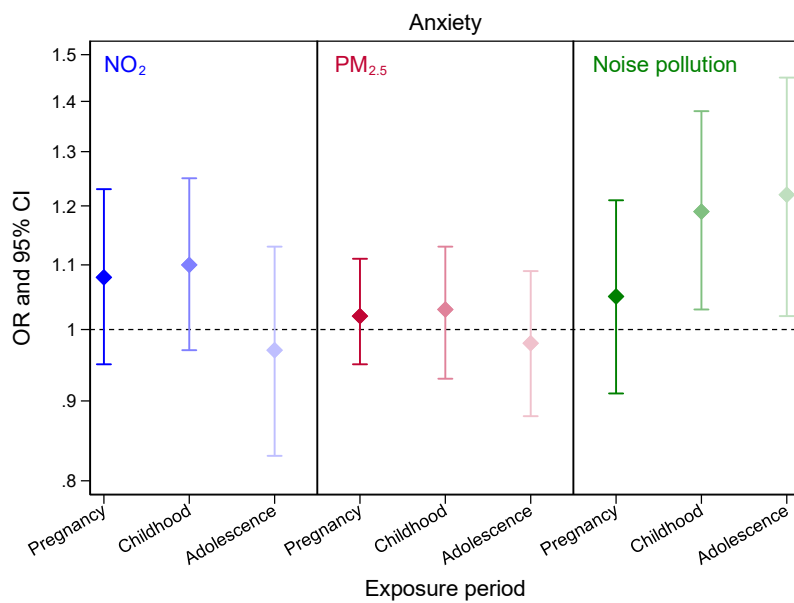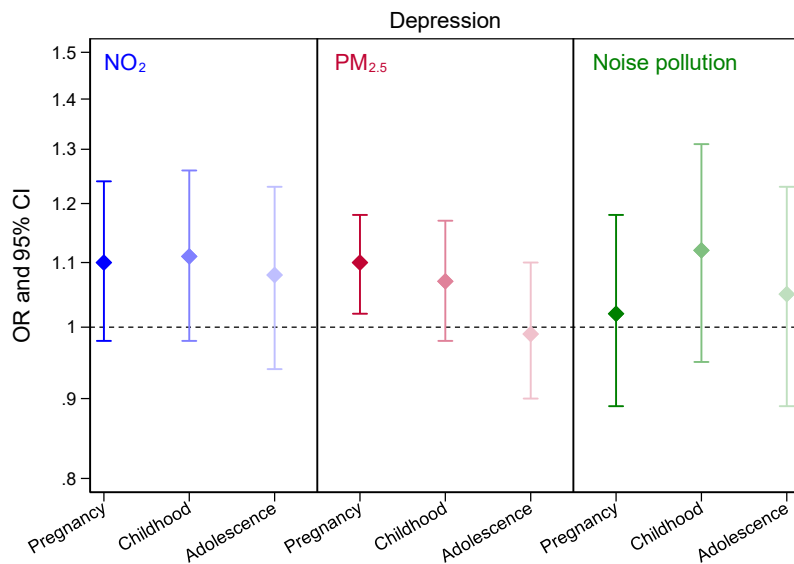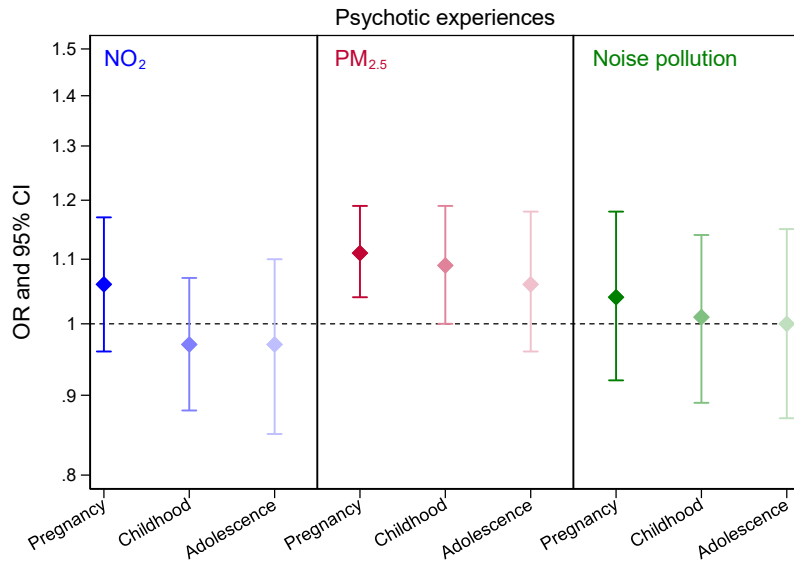
Figure 2. Adjusted associations of early-life air and noise pollution exposure with youth mental health problems

Note: CI=confidence intervals; NO$_2$=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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