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Associations of Trauma Type, Timing and Frequency from Infancy to Adolescence with Psychotic Experiences in Early Adulthood

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25 Word count: 2,996

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27 **Key Points**

28 **Question** Does exposure to trauma during childhood and adolescence increase the risk of

29 developing psychotic experiences?

30 **Findings** In a cohort study of 4,433 adolescents, we find strong evidence that all types of

31 trauma, at any time from early childhood through adolescence, are associated with

32 subsequent psychotic experiences after adjusting for a number of plausible confounders.

33 Effect-sizes were larger for repeated exposure, exposure to multiple types of trauma, and for

34 more proximal exposure to trauma.

35 **Meaning** These findings are consistent with the thesis that trauma has a causal effect on

36 psychotic experiences and highlights the need to identify modifiable mediators in this

37 relationship to inform prevention strategies.

38

39 **Abstract**

40 **Importance** Cross-sectional and longitudinal studies have consistently reported associations between

41 childhood trauma and psychotic experiences and disorders. However, few studies have been able to

42 examine whether timing of exposure or specific trauma-types have differential effects on risk.

43 **Objectives** To examine whether exposure to trauma, assessed at multiple time-points between 0 and

44 17 years of age, is associated with increased risk of psychotic experiences by age 18 years, and

45 whether this association varies according to type, timing and frequency of exposure

46 **Design** Birth cohort study using The Avon Longitudinal Study of Parents and Children, with

47 participants recruited between April 1, 1991, and December 31, 1992. Analyses were carried out from

48 January to November 2017.

49 **Setting** Population-based

50 **Participants** Participants who completed an assessment of psychotic experiences at age 18 years old.

51 **Exposure** Exposure to 6 different types of trauma (covering inter-personal violence and neglect),

52 measured contemporaneously during three age-periods (early childhood, mid-childhood, adolescence)

53 **Main Outcome** Suspected or definite psychotic experiences (9.3%) assessed using the semi-

54 structured PLIKSi interview at age 18 years.

55 **Results** We analysed data from 4,433 participants (56.5% female). All trauma-types across ages 0-17

56 years were associated with an increased odds of psychotic experiences, with little attenuation when

57 adjusting for confounding (OR_{crude} for exposure to any trauma 3.13; 95%CI 2.32, 4.22; OR_{adj} 2.91,

58 95% CI 2.15, 3.93). Assuming this estimate is accurate and causal, the population attributable fraction

59 for childhood and adolescent trauma on psychotic experiences was 45% (95%CI 25%, 60%). Effect

60 sizes for most trauma-types were greater for exposure that was more proximal to the outcome, though

61 confidence intervals overlapped with those for more distal trauma. There was strong evidence to

62 support dose-response associations for exposure to multiple trauma-types and for exposure at multiple

63   timepoints. In an analysis aimed at minimising reverse causality, adolescent trauma was also

64   associated with past-year incident psychotic experiences at age 18 years.

65   **Conclusions and Relevance** Our findings are consistent with the thesis that trauma has a causal effect

66   on psychotic experiences, and highlight the need to identify modifiable mediators of this relationship

67   to inform prevention strategies for psychotic experiences and related adverse mental health outcomes.

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Introduction

Meta-analyses show that exposure to childhood trauma is associated with a 2-3 fold increase in risk of psychotic outcomes[1–4]. Increasing severity or chronicity of trauma, and the presence of multiple different types of trauma exposure (e.g. physical and emotional abuse), which frequently co-occur[5], further elevates this risk[6–10].

However, there is substantial heterogeneity in effect sizes across studies[11–13], with methodological issues including small sample sizes, cross-sectional data, variation in how trauma and psychotic experiences are assessed, and the influence of confounding. As a result, whether the association between trauma and psychosis is causal and, if it is, the size of the causal effect, remain uncertain.

Few studies have examined whether different types of trauma have a differential impact on the risk of psychotic experiences (PEs). Trauma that involves neglect or interpersonal violence appears to be associated with a greater risk of PEs compared to exposure to accidental injury, parental loss or economic adversity[14–16]. However, whether a specific type of interpersonal trauma is more strongly associated with psychosis risk than other types is unclear. In studies that have examined a range of trauma types using multivariable models, sexual abuse has usually been reported to be more strongly associated with psychosis risk than other interpersonal trauma exposures[9,15,17,18], although confidence intervals often overlap with those for other types of trauma exposure[19].

There are also a limited number of studies that have examined whether a sensitive or critical period of risk exists during which exposure to trauma is particularly likely to be associated with psychosis. One study reported a stronger effect of earlier trauma (before age 7), but with overlapping confidence intervals for trauma after this age[14], another found no evidence of difference for exposure pre- and post-13 years[16], and another[20] examined adverse exposures that were differently defined at separate time-points and were thus not directly comparable. Further investigation is therefore required to establish whether there are sensitive periods of risk for exposure to maltreatment.

The present study investigates the role of trauma type, developmental timing, frequency, and influence of confounding in the relationship between trauma and PEs. Using data from a well-

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96   characterised UK birth cohort we examine: i) whether a comprehensive measure of trauma exposure,

97   using both child and parent-reported data during childhood and adolescence, is associated with PEs at

98   age 18 and if this is attenuated after adjusting for a comprehensive range of potential confounders, or

99   explained by reverse causation ii) whether there is evidence to support a 'dose-response' association

100   with exposure to multiple types of trauma, iii) whether specific types of trauma are more strongly

101   associated with risk of PEs than others, and iv) whether sensitive or critical periods of exposure to

102   trauma exist between 0-17 years of age.

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104 **Methods**

105 Sample
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107 We used data from a prospective cohort study, the Avon Longitudinal Study of Parents and Children

108 (ALSPAC). The initial cohort consisted of 14,062 children born to women residing in the former

109 Avon Health Authority area with expected delivery dates between April 1991-December 1992. The

110 total sample, including later enrolment phases, is 14,775 live births[21]. A fully searchable data

111 dictionary is available: http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary/.

112 Ethical approval for this study was obtained from the ALSPAC Law and Ethics Committee and the

113 Local Research Ethics Committees.

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115 Measures
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117 *Psychotic Experiences*

118 PEs were assessed using the Psychosis-like Symptoms semi-structured interview (PLIKSi) at age 12[22]

119 and 18 years[23]. The assessment at age 12 years rated PEs present in the previous six months. The

120 assessment at age 18 years rated PEs occurring since age 12 (outcome used for primary analyses), and

121 PEs that were incident in the previous 12 months (outcome used for sensitivity analysis addressing

122 potential reverse causation effects; see below). The interviews were carried out by trained

123 psychologists and rated following SCAN guidelines.

124 The questions assessed the presence of 12 PEs including hallucinations, delusions and experiences of

125 thought interference. PEs were coded as present if one or more experiences were rated as "suspected"

126 or "definitely present" (see eMethods).

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129

130 *Trauma*

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132 Trauma variables were derived from 121 questions relating to traumatic events from 49 assessments

133 completed by the parents or self-reported by the participants. 48 of these assessments assessed data

134 contemporaneously from participant ages 0 to 17 years. However, as there was no participant self-

135 reported assessment of sexual abuse during adolescence, and limited self-report information on

136 emotional neglect and physical abuse at this age, data were supplemented with information from a

137 questionnaire completed at age 22 years, where participants were asked about these experiences, and

138 the age period during which these had occurred (see below for sensitivity analyses omitting data from

139 this assessment). Selection of questions used to inform each trauma type (physical abuse, sexual

140 abuse, emotional abuse, emotional neglect, domestic violence, bullying), and responses relating to

141 severity and frequency, were carefully considered to ensure that a coding of 'exposed' reflected

142 experiences that would likely be highly upsetting to anyone who experienced them.

143 Variables were derived to represent i) exposure to any trauma type between ages 0 and 17, ii)

144 exposure to any trauma type within distinct age periods: early childhood (0-4.9 years), middle

145 childhood (5-10.9 years), and adolescence (11-17 years), iii) exposure to specific trauma types

146 between ages 0 and 17, and iv) exposure to specific trauma types within distinct age periods: early

147 childhood, middle childhood and adolescence. All trauma variables were coded as binary measures.

148 Variables reflecting the number of trauma-types exposed to during the different age periods were also

149 derived, each ranging from 0 to 6 (see eMethods).

150

151 *Confounding variables*

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153 A range of variables were examined as potential confounders, based on the literature in this field, and

154 included: parental information (psychiatric history, genetic risk for schizophrenia, drug use, criminal

155 history, income, smoking during pregnancy, marital status, living conditions; all assessed around the

156 participants' birth), and participant information (sex, ethnicity, genetic risk for different mental health

157 disorders, temperament (at 6 months), developmental delay (at 18 months), and IQ (at 8 years; though

158   this could also be a potential mediator of early trauma). Only confounders that changed unadjusted

159   estimates by ≥5% were included in the final model (see eMethods).

160

161   Statistical Analysis

162

163   Data analysis was carried out in STATA version 14 (Stata Corp LP, College Station, TX USA).

164   Logistic regression was used to calculate odds ratios (ORs) and 95% confidence intervals (95%CI) for

165   PEs in relation to exposure to trauma both before, and after, adjusting for confounding. We examined

166   the independent association of specific trauma types by additionally adding all trauma types to the

167   confounder-adjusted model, and dose-response associations by comparing categorical variables

168   modelled as dummy variables to modelling them as linear terms.

169   We conducted a series of sensitivity analyses to examine the robustness of our findings. To minimise

170   reverse causation, whereby associations between trauma and PEs might arise from childhood PEs

171   leading to trauma, we examined the association between: i) pre-adolescent trauma (0-10.9 years) and

172   PEs by age 18 years in a subgroup of individuals who did not report PEs at age 12, and ii) adolescent

173   trauma and past-year incident PEs at age 18 years. To address possible lack of measurement

174   invariance across rater-types we conducted separate analyses of parent-reported and child-reported

175   trauma. To examine the association between trauma and more severe PEs we used a narrower

176   outcome of 'definite' vs 'suspected or no' PEs at 18 years old. To further examine proximal versus

177   distal trauma exposure we compared the association between trauma in early childhood and PEs at 12

178   years old with that for trauma in mid-childhood. Finally, to rule out potential recall bias in the

179   measures of trauma that included data from the age 22 questionnaire we repeated the analyses after

180   omitting this data.

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184   Study Sample

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186   The complete sample with data on exposure, outcomes and confounders was 3,758 (Supplementary

187   Figure 1). We conducted multiple imputation for the sample that had completed the PLIKSi at age 18

188   (n=4,433) by creating 50 imputed datasets (see eMethods). Our primary results are presented using

189   the sample with imputed confounder and exposure data (n=4,433). Results of analyses using non-

190   imputed data were similar to those using imputed data (eTables 3-4, 6 and 10).

191

192 **Results**

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194 Study Sample

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196 As summarised in Table 1, those included in the analytic sample were more likely to be female, come

197 from a higher socio-economic position and less likely to report parental history of drug use or mental

198 health problems. Trauma in early childhood was associated with non-completion of the PLIKSi at 18

199 years old.

200 Of the sample of 4,433 participants, 410 (9.3%) were rated as having had suspected or definite PEs

201 atthe age 18 year assessment. The frequency of specific trauma-types within each age period were

202 higher in the imputed compared to the complete case data (eTable 1); 64.5% of the imputed sample

203 reporting exposure to trauma between 0 to age 17 years. Correlations between trauma types at each

204 time-point ranged from 0.01 to 0.72 (eTable 2). Of the candidate confounding variables examined,

205 sex, parental drug use, crowded living conditions, income, and maternal education were included in

206 the final adjusted model. Individuals exposed to different types of trauma were, in general, more

207 likely to report more adverse family characteristics, though sex showed differential patterns of

208 association with different trauma types (Table 1).

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211 Is trauma exposure associated with psychotic experiences?

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213 In those with PEs at aged 18, 83.8% reported exposure to trauma, compared to 62.6% without PEs

214 (imputed data). Exposure to any trauma experienced up to age 17 years was associated with increased

215 odds of PEs at age 18 years (OR 3.13; 95%CI 2.32, 4.22; *p*<0.001; Table 3). Adjusting for

216 confounders attenuated the OR by approximately 10% (adjusted OR 2.91; 95%CI 2.15, 3.93; p<.001).

217 The population attributable fraction for any trauma experienced up to age 17 on PEs at age 18 was

218 45% (95%CI 25%, 60%).

219

220   Is there a dose-response relationship?

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222   We observed an increase in effect size with exposure to a greater number of trauma types between

223   ages 0 to 17 years (linear trend; adjusted OR 1.70; 95%CI 1.54, 1.87; p<.001; Table 3). Reporting

224   more than 3 types of trauma exposure between 0 to 17 years was associated with a 4.7-fold increase in

225   odds of PEs (95%CI 3.40, 6.59; $p<.001$).

226   There was also clear evidence that exposure to trauma in all 3 age periods was associated with higher

227   risk of developing PEs than exposure within only 1 or 2 timepoints (linear trend: OR_{adj} 1.51; 95%CI

228   1.36, 1.68) (eTables 5 & 6).

229

230   Are specific types of trauma more strongly associated with psychotic experiences than others?

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232   There was strong evidence to support increased odds of PEs for all trauma types exposed to between

233   ages 0-17 years of age (adjusted ORs 1.69 to 2.50; all p<.001; Table 3).

234   The confidence intervals for associations between specific trauma types and PEs all overlapped

235   substantially. In the multivariable model adjusting for all trauma types, strong evidence of association

236   with PEs persisted for physical abuse, sexual abuse, bullying, emotional neglect; associations for

237   exposure to domestic violence and emotional abuse were substantially attenuated.

238

239   Are there sensitive or critical periods of risk?

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241   Exposure to trauma during any of the age periods we examined was associated with increased odds of

242   PEs (Table 4). Adjusting for confounding had slightly stronger attenuating effect on the estimate for

243   trauma exposure during early childhood than on trauma exposure during adolescence (approximately

244   20% and 10% attenuation respectively). Effects sizes were greater for exposure to trauma that was

245   more proximal to the outcome, although confidence intervals overlapped with more distal exposure.

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247

248   Sensitivity analyses

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250   Results of association between exposure to both pre-adolescent and adolescent trauma and subsequent

251   PEs were substantively the same when excluding participants that reported PEs at age 12 years

252   (eTable 7), or only examined PEs at age 18 years incident in the last year (eTable 8). Estimations of

253   effect sizes were similar when using a narrower definition of PEs at age 18 years (eTable 9) and

254   comparing effect sizes in mid-childhood and adolescence between trauma reported by parents and

255   children (eTable 10). Similarly to our main analysis, exposure to trauma in mid-childhood was more

256   strongly associated with PEs at age 12 years than exposure in early childhood (OR_{adj} 1.80; 95%CI

257   1.45, 2.16; and OR_{adj} 1.33; 95%CI 1.08, 1.65 respectively), although confidence intervals overlapped.

258   Finally, when excluding trauma data collected at 22 years, effect sizes were smaller, though the

259   strength of evidence remained similar, for most trauma variables (e.g. OR_{adj} for any trauma age 0-17

260   years = 2.62; 95%CI 2.02, 3.41; $p < 0.001$; etable 11).

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Discussion

In this large, population-based, birth cohort we found that exposure to traumatic experiences during childhood and adolescence was strongly associated with development of PEs by early adulthood. This was not explained by a more comprehensive range of confounders than adjusted for in any previous study, including genetic risk for psychiatric disorders, family characteristics, socio-economic adversity, and markers of childhood development. Associations for adolescent trauma were also not explained by reverse causation, providing perhaps the strongest observational evidence to date of a causal association between trauma on PEs. That confounding is not an adequate explanation for this association is consistent with findings from other studies[9,24,25,14].

Exposure to any type of trauma was strongly associated with PEs, with little evidence that specific types of trauma increase the risk of PEs more than others. The risk of PEs was stronger following exposure to multiple types of trauma or to repeated episodes of trauma at multiple time-points, consistent with a dose-response relationship, as found in other studies[26].

We found that adolescence was the age-period during which exposure to trauma was most strongly associated with risk of PEs. Possible explanations for this include: i) temporal proximity to the outcome is more influential on risk than age of exposure, and that natural resolution of trauma-related psychopathology occurs over time, consistent with findings from two other studies[24,15]; ii) adolescence represents a particularly sensitive period of risk for the effects of interpersonal trauma on psychosis, support for which comes from animal and human studies showing increasing HPA activation and anxiety following exposure to stress in adolescence compared to other time-points [27–30]; iii) weaker effects for earlier trauma measures in our study result from greater measurement error, perhaps as they were obtained from parental reports only, although this seems unlikely given results from our sensitivity analyses addressing informant-related measurement-variance (supplementary eResults). Our findings are consistent with another[20], but not all[14,16,20] studies that have examined differential effects of age of trauma exposure on PEs.

288

Possible Mechanisms

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Our results are consistent with trauma having a causal role in the aetiology of PEs, and indicate that the mechanism underlying this is not dependent on the type of trauma, but more on the severity, chronicity, and perhaps recency of exposure. Biological models of stress show clear overlap with the dysregulation of dopaminergic and glutamatergic systems[31] that are the most widely-supported aetiological models of psychosis[32]. Cognitive and perceptual biases that can arise post exposure to trauma[33], that are observed more frequently in people with psychosis[34,35], and that have been associated with dopaminergic and glutamatergic dysfunction[36] are strong candidates as mediators of the trauma-PE relationship and, whilst further evidence of this is required[37], might be potential target for interventions.

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Strengths and Limitations

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Our study has several strengths including use of a large, population-based birth cohort with multiple measures of trauma collected contemporaneously to minimise measurement error and recall bias, a wealth of relevant data to allow rigorous testing of confounding, and repeated measures of PEs to minimise reverse causation. Furthermore, we used semi-structured interviews to assess PEs, as used in clinical practice, hence increasing the validity of our outcome and allowing us to greater confidence in inferring information about the aetiology of such phenomena.

309

However, there are also a number of limitations. First, as with most cohort studies, there was substantial attrition over time that may have led to selection bias when using complete-case data. We therefore used multiple imputation, using data from a range of relevant variables associated with our

313   exposure and with missingness, to make the missing-at-random assumption more plausible and thus

314   minimise potential attrition bias.

315   Second, whilst the majority of our exposure data was collected prior to age 18 years, we had no such

316   data on sexual abuse in adolescence, whilst we also lacked self-report measures of physical abuse and

317   emotional neglect during this developmental period. This information was therefore obtained from an

318   assessment at age 22 years, and hence may have been subject to recall bias. Our sensitivity analyses

319   omitting data from this questionnaire led, in the main, to smaller effect sizes in the association

320   between exposure to trauma and PEs, which could either support the influence of recall bias leading to

321   an over-estimation in our main reported analyses, or greater measurement error resulting from loss of

322   any self-reported information on some trauma-types during adolescence.

323

324   Implications of findings

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326   Our study indicates that, assuming the effect is accurate and causal, a substantial proportion (25%-

327   60%) of individuals would not have developed PEs if they had not been exposed to traumatic

328   experiences during childhood, consistent with previous estimates[4].

329   PEs are associated with the presence of, and with increased risk of developing, a wide range of

330   adverse mental health outcomes apart from psychotic disorders[,38,39], and also occur outside of the

331   context of mental illness. Whilst they may be a non-specific marker of severity of general

332   psychopathology[40], PEs are associated with substantial levels of distress and impairment at a

333   population-health level[23]. Novel interventions that aim to address the effects of trauma on mechanisms

334   leading to the development of PEs could improve mental health outcomes in population-based and

335   clinical contexts.

336

337   Conclusion

338

339 Our findings, of consistent associations between different trauma types and PEs, not explained by a

340 broad range of confounders, of dose-response relationships, and with strongest effects observed for

341 more proximal traumas, support the thesis that traumatic experiences have a causal effect on PEs. The

342 results do not suggest that there is a sensitive period of risk associated with a greater risk of PEs..

343 Longitudinal studies that examine potentially modifiable mediators in the relationship between trauma

344 and psychosis are required to inform prevention strategies and could improve outcomes for a range of

345 mental health disorders.

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Author Contributions

Ms Croft and Prof Zammit had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Concept and design: Croft, Heron, Zammit

Acquisition, analysis, or interpretation of data: All authors

Drafting of the manuscript: Croft, Zammit

Critical revision of the manuscript for important intellectual content: All Authors

Statistical analysis: Croft, Heron, Zammit.

Obtained funding: Thompson, Cannon, Wolke, Heron, Zammit

Supervision: Heron, Zammit, Teufel

Conflict of Interest Disclosures: None reported.

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473

474   Tables

475   Table 1: Sample Characteristics for Participants Who Completed the Psychotic Experiences

476   Assessment

	Analytic sample availability		OR (95% CI)	*p*
	Included[a]	**Excluded**		
Reference category	**(n=3,758)**	**(n=10,196)**		
	N (%)	**N (%)**		
Female sex	2,111 (56.17)	4,636 (45.48)	1.54 (1.43, 1.67)	<.001
Parental Drug Use	329 (8.75)	978 (10.12)	0.85, (0.75, 0.97)	.017
Living 1+ per room	123 (3.27)	755 (8.37)	0.37 (0.31, 0.45)	<.001
Lowest Income	492 (13.09)	1,497 (24.28)	0.38 (0.33, 0.43)	<.001
Maternal education <O-level	639 (17.00)	3,084 (35.71)	0.29 (0.26, 0.32)	<.001
Parental psychiatric history	617 (16.43)	1,781 (19.02)	0.84 (.76, .93)	.001

477   Abbreviation: OR, odds ratio. [a]Participants included in analytic sample were those who had completed the
478   assessment of psychotic experiences at age 18 years

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Table 2: Summary statistics of confounders in relation to trauma exposure (0-17 years)

		N(%) of confounding variable reported in exposed/unexposed trauma groups				
		Sex (Female)	Parental drug use	Living in crowded conditions	Low Income	Maternal Education (<O level)
Physical Abuse	Yes	470 (56.29)	86 (10.39)	43 (5.36)	120 (15.94)	158 (19.55)
	No	2,027 (56.48)	307 (8.63)	118 (3.41)	421 (13.31)	647 (18.59)
Emotional Abuse	Yes	513 (59.24)	109 (12.66)	49 (5.89)	143 (17.99)	163 (19.22)
	No	1,979 (55.72)	284 (8.06)	110 (3.21)	398 (12.75)	640 (18.62)
Bullying	Yes	597 (49.01)	102 (8.42)	53 (4.51)	151 (13.78)	242 (20.30)
	No	1,859 (59.22)	279 (8.96)	102 (3.37)	386 (13.73)	534 (17.51)
Sexual Abuse	Yes	303 (87.07)	33 (9.54)	16 (4.82)	58 (18.30)	166 (48.54)
	No	2,159 (53.77)	355 (8.91)	136 (3.51)	483 (13.43)	1,850 (47.36)
Domestic Violence	Yes	465 (42.66)	123 (15.34)	63 (8.15)	167 (22.94)	167 (21.36)
	No	2,011 (56.24)	264 (7.43)	93 (2.69)	374 (11.73)	626 (17.95)
Emotional Neglect	Yes	151 (50.00)	28 (9.33)	12 (4.17)	45 (16.48)	57 (19.39)
	No	2,291 (56.95)	848 (8.72)	141 (3.63)	483 (13.43)	716 (18.3)

Table 3: Associations Between Exposure to Trauma and Subsequent Psychotic Experiences According to Type and Frequency[a]

		Unadjusted			Adjusted[a]			Adjusted[a,b]		
	% Exposed	OR	95% CI	*p*	OR	95% CI	*p*	OR	95% CI	*p*
Any Trauma	64.5	3.13	2.32, 4.22	<.001	2.91	2.15, 3.93	<.001			
Physical Abuse	23.1	2.36	1.85, 3.02	<.001	1.69	1.27, 2.23	<.001	2.24	1.75, 2.87	<.001
Emotional Abuse	23.7	1.94	1.53, 2.46	<.001	1.81	1.42, 2.31	<.001	1.25	0.94, 1.65	.125
Bullying	32.9	2.07	1.66, 2.57	<.001	2.05	1.65, 2.57	<.001	1.80	1.43, 2.26	<.001
Sexual abuse	11.0	2.75	2.00, 3.79	<.001	2.50	1.79, 3.51	<.001	2.04	1.42, 2.91	<.001
Domestic Violence	21.9	2.02	1.59, 2.56	<.001	1.79	1.40, 2.29	<.001	1.48	1.13, 1.94	.004
Emotional Neglect	7.8	2.41	1.75, 3.30	<.001	1.89	1.35, 2.65	<.001	2.33	1.70, 3.21	<.001

		OR	95% CI	p	OR	95% CI	p
Number of trauma types (%)	1- 26.7	1.94	1.33, 2.81	.001	1.89	1.30, 2.74	.001
	2 – 16.4	2.67	1.81, 3.91	<.001	2.54	1.72, 3.75	<.001
	3+ - 21.3	5.19	3.76 7.16	<.001	4.74	3.40, 6.59	<.001
	Linear Trend	1.70	1.54, 1.87	<.001	1.65	1.48, 1.82	<.001

[a]Imputed dataset, n=4,433 Abbreviation: OR, odds ratio [b]Adjusted for confounders: sex, parental income, parental drug use, maternal education, crowded living conditions
[c]Adjusted for other trauma exposures

Table **4**: Associations Between Exposure to Trauma and Psychotic Experiences at 18 years According to Timing and Type[a]

	% exposed	Unadjusted			Adjusted[b]			Adjusted[b,c]		
		OR	95% CI	*p*	OR	95% CI	*p*	OR	95% CI	*p*
Any trauma (age-period)										
Any Trauma (0-4.9 years)	22.5	1.88	1.49, 2.38	<.001	1.70	1.33, 2.17	<.001			
Any Trauma (5-10.9 years)	43.6	2.27	1.81, 2.84	<.001	2.16	1.71, 2.71	<.001			
Any Trauma (11-17 years)	40.1	2.92	2.29, 3.71	<.001	2.72	2.13, 3.47	<.001			
Trauma Types (0-4.9 years)										
Physical Abuse	4.7	1.32	0.83, 2.09	.244	1.30	0.82, 2.08	.264	.93	0.56, 1.55	.781

Emotional Abuse	11.2	1.64	1.21, 2.23	.002	1.52	1.11, 2.07	.009	1.31	0.83, 1.86	.125
Bullying	1.7	1.81	0.90, 3.66	.095	1.71	0.84, 3.48	.137	1.68	0.82, 3.43	.158
Sexual abuse	0.2	3.52	0.69, 17.85	.129	2.42	0.46, 12.84	.299	2.47	0.46, 13.26	.292
Domestic Violence	13.2	2.08	1.60, 2.71	<.001	1.83	1.39, 2.40	<.001	1.71	1.27, 2.29	<.001
Emotional Neglect	3.5	-	-	-	-	-	-	-	-	-

Trauma Types (5-10.9 years)

Physical Abuse	10.3	2.07	1.52, 2.84	<.001	1.98	1.45, 2.72	<.001	1.58	1.10, 2.26	.013
Emotional Abuse	12.9	1.86	1.41, 2.45	<.001	1.77	1.34, 2.35	<.001	1.37	0.98, 1.91	.062
Bullying	21.6	1.89	1.46, 2.37	<.001	1.91	1.48, 2.44	<.001	1.74	1.34, 2.25	<.001
Sexual abuse	2.8	1.87	1.07, 3.28	.028	1.50	0.84, 2.67	.172	1.18	0.64, 2.17	.589
Domestic Violence	13.1	1.99	1.46, 2.72	<.001	1.75	1.26, 2.43	.001	1.47	1.04, 2.08	.029
Emotional Neglect	3.5	2.45	1.58, 3.18	<.001	2.32	1.49, 3.63	<.001	1.95	1.23, 3.09	.004

Trauma Types (11-17 years)

		OR[a]	95% CI	p	OR[b]	95% CI	p	OR[c]	95% CI	p
Physical Abuse	15.6	2.63	2.02, 3.42	<.001	2.43	1.86, 3.18	<.001	1.83	1.36, 2.47	<.001
Emotional Abuse	7.3	2.42	1.75, 3.35	<.001	2.23	1.60, 3.10	<.001	1.40	0.95, 2.06	.094
Bullying	14.4	2.17	1.69, 2.78	<.001	2.10	1.64, 2.70	<.001	1.87	1.45, 2.42	<.001
Sexual abuse	9.4	3.21	2.31, 4.46	<.001	3.00	2.12, 4.21	<.001	2.34	1.62, 3.37	<.001
Domestic Violence	5.0	1.99	1.22, 3.23	.006	1.70	1.03, 2.81	.036	1.37	0.80, 2.33	.246
Emotional Neglect	3.5	2.33	1.56, 3.74	<.001	2.29	1.52, 3.44	<.001	1.96	1.28, 3.00	.002

[a]Imputed dataset, n=4,433 Abbreviation: OR, odds ratio [b]Adjusted for confounders: sex, parental income, parental drug use, maternal education, crowded living conditions [c]Adjusted for other trauma exposures

Table **5**: Associations Between Exposure to Trauma According to Frequency of Types and Psychotic Experiences at 18 Years Old[a]

Time Point	N types of trauma (%)	Unadjusted			Adjusted[b]		
		OR	95% CI	*p*	OR	95% CI	*p*
0-4.9 years	1 – 15.4	1.74	1.31, 2.31	<.001	1.56	1.79, 2.10	.002
	2 – 5.4	2.27	1.54, 3.36	<.001	2.03	1.36, 3.02	<.001
	3+ - 1.5	1.93	.93, 4.02	.078	1.82	.87, 3.80	.114
	Linear Trend	1.45	1.26, 1.67	<.001	1.38	1.19, 1.59	<.001
5 – 10.9 years	1 – 28.7	1.80	1.39, 2.34	<.001	1.75	1.34, 2.28	<.001
	2- 10.7	2.80	2.01, 3.91	<.001	2.65	1.88, 3.73	<.001
	3+ - 4.3	4.33	2.85, 6.57	<.001	3.88	2.53, 5.94	<.001
	Linear Trend	1.65	1.47, 1.85	<.001	1.60	1.42, 1.80	<.001
11 – 17 years	1 – 25.9	2.20	1.66, 2.91	<.001	2.09	1.57, 2.78	<.001
	2 – 8.9	3.47	2.43, 4.94	<.001	3.20	2.23, 4.58	<.001
	3+ – 3.8	7.73	5.12, 11.67	<.001	6.75	4.42, 10.31	<.001
	Linear Trend	1.94	1.72, 2.18	<.001	1.86	1.64, 2.10	<.001

[a]Imputed dataset, n=4,433 Abbreviation: OR, odds ratio [b]Adjusted for confounders: sex, parental income, parental drug use, maternal education, crowded living conditions