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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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## **Key Points**

**Question** Does exposure to trauma during childhood and adolescence increase the risk of developing psychotic experiences?

**Findings** In a cohort study of 4,433 adolescents, we find strong evidence that all types of trauma, at any time from early childhood through adolescence, are associated with subsequent psychotic experiences after adjusting for a number of plausible confounders. Effect-sizes were larger for repeated exposure, exposure to multiple types of trauma, and for more proximal exposure to trauma.

**Meaning** These findings are consistent with the thesis that trauma has a causal effect on psychotic experiences, and highlights the need to identify modifiable mediators in this relationship to inform prevention strategies.

## **Abstract**

**Importance** Cross-sectional and longitudinal studies have consistently reported associations between childhood trauma and psychotic experiences and disorders. However, few studies have been able to examine whether timing of exposure or specific trauma-types have differential effects on risk.

**Objectives** To examine whether exposure to trauma, assessed at multiple time-points between 0 and 17 years of age, is associated with increased risk of psychotic experiences by age 18 years, and whether this association varies according to type, timing and frequency of exposure

**Design** Birth cohort study using The Avon Longitudinal Study of Parents and Children, with participants recruited between April 1, 1991, and December 31, 1992. Analyses were carried out from January to November 2017.

**Setting** Population-based

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

**Exposure** Exposure to 6 different types of trauma (covering inter-personal violence and neglect), measured contemporaneously during three age-periods (early childhood, mid-childhood, adolescence)

**Main Outcome** Suspected or definite psychotic experiences (9.3%) assessed using the semi-structured PLIKSi interview at age 18 years.

**Results** We analysed data from 4,433 participants (56.5% female). All trauma-types across ages 0-17 years were associated with an increased odds of psychotic experiences, with little attenuation when adjusting for confounding (OR<sub>crude</sub> for exposure to any trauma 3.13; 95% CI 2.32, 4.22; OR<sub>adj</sub> 2.91, 95% CI 2.15, 3.93). Assuming this estimate is accurate and causal, the population attributable fraction for childhood and adolescent trauma on psychotic experiences was 45% (95% CI 25%, 60%). Effect sizes for most trauma-types were greater for exposure that was more proximal to the outcome, though confidence intervals overlapped with those for more distal trauma. There was strong evidence to support dose-response associations for exposure to multiple trauma-types and for exposure at multiple

timepoints. In an analysis aimed at minimising reverse causality, adolescent trauma was also associated with past-year incident psychotic experiences at age 18 years.

**Conclusions and Relevance** Our findings are consistent with the thesis that trauma has a causal effect on psychotic experiences, and highlight the need to identify modifiable mediators of this relationship to inform prevention strategies for psychotic experiences and related adverse mental health outcomes.

## **Introduction**

Meta-analyses show that exposure to childhood trauma is associated with a 2-3 fold increase in risk of psychotic outcomes<sup>1-4</sup>. 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<sup>5</sup>, further elevates this risk<sup>6-10</sup>.

However, there is substantial heterogeneity in effect sizes across studies<sup>11-13</sup>, 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<sup>14-16</sup>. 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<sup>9,15,17,18</sup>, although confidence intervals often overlap with those for other types of trauma exposure<sup>19</sup>.

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<sup>14</sup>, another found no evidence of difference for exposure pre- and post-13 years<sup>16</sup>, and another<sup>20</sup> 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-

characterised UK birth cohort we examine: i) whether a comprehensive measure of trauma exposure, using both child and parent-reported data during childhood and adolescence, is associated with PEs at age 18 and if this is attenuated after adjusting for a comprehensive range of potential confounders, or explained by reverse causation ii) whether there is evidence to support a 'dose-response' association with exposure to multiple types of trauma, iii) whether specific types of trauma are more strongly associated with risk of PEs than others, and iv) whether sensitive or critical periods of exposure to trauma exist between 0-17 years of age.

## Methods

### Sample

We used data from a prospective cohort study, the Avon Longitudinal Study of Parents and Children (ALSPAC). The initial cohort consisted of 14,062 children born to women residing in the former Avon Health Authority area with expected delivery dates between April 1991-December 1992. The total sample, including later enrolment phases, is 14,775 live births<sup>21</sup>. All participants provided written informed consent. A fully searchable data dictionary is available:

<http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary/>.

Ethical approval for this study was obtained from the ALSPAC Law and Ethics Committee and the Local Research Ethics Committees.

### Measures

#### *Psychotic Experiences*

PEs were assessed using the Psychosis-like Symptoms semi-structured interview (PLIKSi) at age 12<sup>22</sup> and 18 years<sup>23</sup>. The assessment at age 12 years rated PEs present in the previous six months. The assessment at age 18 years rated PEs occurring since age 12 (outcome used for primary analyses), and PEs that were incident in the previous 12 months (outcome used for sensitivity analysis addressing potential reverse causation effects; see below). The interviews were carried out by trained psychologists and rated following SCAN guidelines.

The questions assessed the presence of 12 PEs including hallucinations, delusions and experiences of thought interference. PEs were coded as present if one or more experiences were rated as “suspected” or “definitely present” (see eMethods).



### *Trauma*

Trauma variables were derived from 121 questions relating to traumatic events from 49 assessments completed by the parents or self-reported by the participants. 48 of these assessments assessed data contemporaneously from participant ages 0 to 17 years. However, as there was no participant self-reported assessment of sexual abuse during adolescence, and limited self-report information on emotional neglect and physical abuse at this age, data were supplemented with information from a questionnaire completed at age 22 years, where participants were asked about these experiences, and the age period during which these had occurred (see below for sensitivity analyses omitting data from this assessment). Selection of questions used to inform each trauma type (physical abuse, sexual abuse, emotional abuse, emotional neglect, domestic violence, bullying), and responses relating to severity and frequency, were carefully considered to ensure that a coding of 'exposed' reflected experiences that would likely be highly upsetting to anyone who experienced them.

Variables were derived to represent i) exposure to any trauma type between ages 0 and 17, ii) exposure to any trauma type within distinct age periods: early childhood (0-4.9 years), middle childhood (5-10.9 years), and adolescence (11-17 years), iii) exposure to specific trauma types between ages 0 and 17, and iv) exposure to specific trauma types within distinct age periods: early childhood, middle childhood and adolescence. All trauma variables were coded as binary measures. Variables reflecting the number of trauma-types exposed to during the different age periods were also derived, each ranging from 0 to 6 (see eMethods).

### *Confounding variables*

A range of variables were examined as potential confounders, based on the literature in this field, and included: parental information (psychiatric history, genetic risk for schizophrenia, drug use, criminal history, income, smoking during pregnancy, marital status, living conditions; all assessed around the participants' birth), and participant information (sex, ethnicity, genetic risk for different mental health

disorders, temperament (at 6 months), developmental delay (at 18 months), and IQ (at 8 years; though this could also be a potential mediator of early trauma). Only confounders that changed unadjusted estimates by  $\geq 5\%$  were included in the final model (see eMethods).

## Statistical Analysis

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

Logistic regression was used to calculate odds ratios (ORs) and 95% confidence intervals (95%CI) for PEs in relation to exposure to trauma both before, and after, adjusting for confounding. We examined the independent association of specific trauma types by additionally adding all trauma types to the confounder-adjusted model, and dose-response associations by comparing categorical variables modelled as dummy variables to modelling them as linear terms.

We conducted a series of sensitivity analyses to examine the robustness of our findings. To minimise reverse causation, whereby associations between trauma and PEs might arise from childhood PEs leading to trauma, we examined the association between: i) pre-adolescent trauma (0-10.9 years) and PEs by age 18 years in a subgroup of individuals who did not report PEs at age 12, and ii) adolescent trauma and past-year incident PEs at age 18 years. To address possible lack of measurement invariance across rater-types we conducted separate analyses of parent-reported and child-reported trauma. To examine the association between trauma and more severe PEs we used a narrower outcome of 'definite' vs 'suspected or no' PEs at 18 years old. To further examine proximal versus distal trauma exposure we compared the association between trauma in early childhood and PEs at 12 years old with that for trauma in mid-childhood. Finally, to rule out potential recall bias in the measures of trauma that included data from the age 22 questionnaire we repeated the analyses after omitting this data.

## Study Sample

The complete sample with data on exposure, outcomes and confounders was 3,758 (Supplementary Figure 1). We conducted multiple imputation for the sample that had completed the PLIKSi at age 18 (n=4,433) by creating 50 imputed datasets (see eMethods). Our primary results are presented using the sample with imputed confounder and exposure data (n=4,433). Results of analyses using non-imputed data were similar to those using imputed data (eTables 3-4, 6 and 10).

## Results

### Study Sample

As summarised in Table 1, those included in the analytic sample were more likely to be female, come from a higher socio-economic position and less likely to report parental history of drug use or mental health problems. Trauma in early childhood was associated with non-completion of the PLIKSi at 18 years old.

Of the sample of 4,433 participants, 410 (9.3%) were rated as having had suspected or definite PEs at the age 18 year assessment. The frequency of specific trauma-types within each age period were higher in the imputed compared to the complete case data (eTable 1); 64.5% of the imputed sample reporting exposure to trauma between 0 to age 17 years. Correlations between trauma types at each time-point ranged from 0.01 to 0.72 (eTable 2). Of the candidate confounding variables examined, sex, parental drug use, crowded living conditions, income, and maternal education were included in the final adjusted model. Individuals exposed to different types of trauma were, in general, more likely to report more adverse family characteristics, though sex showed differential patterns of association with different trauma types (Table 1).

### Is trauma exposure associated with psychotic experiences?

In those with PEs at aged 18, 83.8% reported exposure to trauma, compared to 62.6% without PEs (imputed data). Exposure to any trauma experienced up to age 17 years was associated with increased odds of PEs at age 18 years (OR 3.13; 95%CI 2.32, 4.22;  $p < 0.001$ ; Table 3). Adjusting for confounders attenuated the OR by approximately 10% (adjusted OR 2.91; 95%CI 2.15, 3.93;  $p < .001$ ). The population attributable fraction for any trauma experienced up to age 17 on PEs at age 18 was 45% (95%CI 25%, 60%).

Is there a dose-response relationship?

We observed an increase in effect size with exposure to a greater number of trauma types between ages 0 to 17 years (linear trend; adjusted OR 1.70; 95% CI 1.54, 1.87;  $p < .001$ ; Table 3). Reporting more than 3 types of trauma exposure between 0 to 17 years was associated with a 4.7-fold increase in odds of PEs (95% CI 3.40, 6.59;  $p < .001$ ).

There was also clear evidence that exposure to trauma in all 3 age periods was associated with higher risk of developing PEs than exposure within only 1 or 2 timepoints (linear trend: OR<sub>adj</sub> 1.51; 95% CI 1.36, 1.68) (eTables 5 & 6).

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

There was strong evidence to support increased odds of PEs for all trauma types exposed to between ages 0-17 years of age (adjusted ORs 1.69 to 2.50; all  $p < .001$ ; Table 3).

The confidence intervals for associations between specific trauma types and PEs all overlapped substantially. In the multivariable model adjusting for all trauma types, strong evidence of association with PEs persisted for physical abuse, sexual abuse, bullying, emotional neglect; associations for exposure to domestic violence and emotional abuse were substantially attenuated.

Are there sensitive or critical periods of risk?

Exposure to trauma during any of the age periods we examined was associated with increased odds of PEs (Table 4). Adjusting for confounding had slightly stronger attenuating effect on the estimate for trauma exposure during early childhood than on trauma exposure during adolescence (approximately 20% and 10% attenuation respectively). Effects sizes were greater for exposure to trauma that was more proximal to the outcome, although confidence intervals overlapped with more distal exposure.

## Sensitivity analyses

Results of association between exposure to both pre-adolescent and adolescent trauma and subsequent PEs were substantively the same when excluding participants that reported PEs at age 12 years (eTable 7), or only examined PEs at age 18 years incident in the last year (eTable 8). Estimations of effect sizes were similar when using a narrower definition of PEs at age 18 years (eTable 9) and comparing effect sizes in mid-childhood and adolescence between trauma reported by parents and children (eTable 10). Similarly to our main analysis, exposure to trauma in mid-childhood was more strongly associated with PEs at age 12 years than exposure in early childhood ( $OR_{adj}$  1.80; 95%CI 1.45, 2.16; and  $OR_{adj}$  1.33; 95%CI 1.08, 1.65 respectively), although confidence intervals overlapped. Finally, when excluding trauma data collected at 22 years, effect sizes were smaller, though the strength of evidence remained similar, for most trauma variables (e.g.  $OR_{adj}$  for any trauma age 0-17 years = 2.62; 95%CI 2.02, 3.41;  $p < 0.001$ ; eTable 11).

## 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<sup>9,24,25,14</sup>.

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<sup>26</sup>.

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<sup>24,15</sup>; 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<sup>27-30</sup>; 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<sup>20</sup>, but not all<sup>14,16,20</sup> studies that have examined differential effects of age of trauma exposure on PEs.

## Possible Mechanisms

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<sup>31</sup> that are the most widely-supported aetiological models of psychosis<sup>32</sup>. Cognitive and perceptual biases that can arise post exposure to trauma<sup>33</sup>, that are observed more frequently in people with psychosis<sup>34,35</sup>, and that have been associated with dopaminergic and glutamatergic dysfunction<sup>36</sup> are strong candidates as mediators of the trauma-PE relationship and, whilst further evidence of this is required<sup>37</sup>, might be potential target for interventions.

## Strengths and Limitations

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.

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



exposure and with missingness, to make the missing-at-random assumption more plausible and thus minimise potential attrition bias.

Second, whilst the majority of our exposure data was collected prior to age 18 years, we had no such data on sexual abuse in adolescence, whilst we also lacked self-report measures of physical abuse and emotional neglect during this developmental period. This information was therefore obtained from an assessment at age 22 years, and hence may have been subject to recall bias. Our sensitivity analyses omitting data from this questionnaire led, in the main, to smaller effect sizes in the association between exposure to trauma and PEs, which could either support the influence of recall bias leading to an over-estimation in our main reported analyses, or greater measurement error resulting from loss of any self-reported information on some trauma-types during adolescence.

### Implications of findings

Our study indicates that, assuming the effect is accurate and causal, a substantial proportion (25%-60%) of individuals would not have developed PEs if they had not been exposed to traumatic experiences during childhood, consistent with previous estimates<sup>4</sup>.

PEs are associated with the presence of, and with increased risk of developing, a wide range of adverse mental health outcomes apart from psychotic disorders<sup>38,39</sup>, and also occur outside of the context of mental illness. Whilst they may be a non-specific marker of severity of general psychopathology<sup>40</sup>, PEs are associated with substantial levels of distress and impairment at a population-health level<sup>23</sup>. Novel interventions that aim to address the effects of trauma on mechanisms leading to the development of PEs could improve mental health outcomes in population-based and clinical contexts.

### Conclusion

Our findings, of consistent associations between different trauma types and PEs, not explained by a broad range of confounders, of dose-response relationships, and with strongest effects observed for more proximal traumas, support the thesis that traumatic experiences have a causal effect on PEs. The results do not suggest that there is a sensitive period of risk associated with a greater risk of PEs..

Longitudinal studies that examine potentially modifiable mediators in the relationship between trauma and psychosis are required to inform prevention strategies and could improve outcomes for a range of mental health disorders.

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The funding sources had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

## **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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## Tables

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

Assessment

Reference category	Analytic sample availability		OR (95% CI)	<i>p</i>
	Included <sup>a</sup> (n=3,758) N (%)	Excluded (n=10,196) 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

Abbreviation: OR, odds ratio. <sup>a</sup>Participants included in analytic sample were those who had completed the assessment of psychotic experiences at age 18 years



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

		<b>N(%) of confounding variable reported in exposed/unexposed trauma groups</b>				
		<b>Sex (Female)</b>	<b>Parental drug use</b>	<b>Living in crowded conditions</b>	<b>Low Income</b>	<b>Maternal Education (&lt;O level)</b>
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<sup>a</sup>

	% Exposed	Unadjusted			Adjusted <sup>a</sup>			Adjusted <sup>a,b</sup>		
		OR	95% CI	<i>p</i>	OR	95% CI	<i>p</i>	OR	95% CI	<i>p</i>
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

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

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

**Table 4: Associations Between Exposure to Trauma and Psychotic Experiences at 18 years According to Timing and Type<sup>a</sup>**

	%	Unadjusted			Adjusted <sup>b</sup>			Adjusted <sup>b,c</sup>		
		OR	95% CI	<i>p</i>	OR	95% CI	<i>p</i>	OR	95% CI	<i>p</i>
<b>exposed</b>										

**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
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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
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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)**

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

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

Table 5: Associations Between Exposure to Trauma According to Frequency of Types and Psychotic Experiences at 18 Years Old<sup>a</sup>

Time Point	N types of trauma (%)	Unadjusted			Adjusted <sup>b</sup>		
		OR	95% CI	<i>p</i>	OR	95% CI	<i>p</i>
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
	<b>Linear Trend</b>	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
	<b>Linear Trend</b>	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
	<b>Linear Trend</b>	1.94	1.72, 2.18	<.001	1.86	1.64, 2.10	<.001

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