SUPPLEMENTARY MATERIAL

Unravelling the contribution of complex trauma to psychopathology and cognitive deficits: a cohort study

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SUPPLEMENTARY METHODS

Statistical analysis

To investigate psychopathology and cognitive deficits linked with complex and non-complex trauma in young people, we compared participants exposed to complex trauma or non-complex trauma with trauma-unexposed participants respectively. Additionally, to investigate psychopathology and cognitive deficits uniquely linked with complex trauma within trauma-exposed individuals, we compared participants exposed to complex trauma with those exposed to non-complex trauma.

First, we compared these groups to assess whether complex trauma exposure is associated with more severe psychopathology and poorer cognitive function. We described psychopathology and cognitive function in the three groups, including means and prevalences, with 95% confidence intervals based on robust standard errors accounting for clustering within families (prevalence confidence intervals were calculated using the Agresti-Coull method[1]). Then we tested associations between trauma exposure (independent variable) and psychopathology and cognitive function (dependent variables) using univariable (unadjusted) generalized estimating equation (GEE) regression models with an exchangeable correlation structure and robust standard errors, accounting for clustering within families. 'p' was tested using linear regression; count of psychiatric disorders was tested using negative binomial regression; psychiatric disorders were tested using logistic regression; and IQ, executive function, and processing speed were tested using linear regression. Where data were missing (0-28/2,058 participants with trauma data), for each model we included participants with non-missing data for variables in that model so that all available data were included.

Next, we assessed whether greater early childhood vulnerability is associated with higher risk of complex trauma exposure. We tested associations between early childhood vulnerabilities (independent variables) and trauma exposure (dependent variable) using univariable (unadjusted) GEE multinomial regression models. If early childhood vulnerabilities are associated with complex or non-complex trauma exposure, these vulnerabilities (known to be associated with psychopathology and cognitive deficits based on previous research[2,3]) might have confounding effects, a plausible non-causal mechanism that could explain trauma-related psychopathology and cognitive deficits.

Then, to examine this possible underlying mechanism of confounding, we assessed whether early childhood vulnerabilities account for psychopathology and cognitive deficits linked to complex trauma. We tested associations between trauma exposure (independent variable) and psychopathology and cognitive function (dependent variables) in multivariable models which included covariates for early childhood vulnerabilities, and compared the size and statistical significance of coefficients for trauma exposure from these models with coefficients from corresponding univariable models. Where data were missing (68-93/2,058 participants with trauma data), for each multivariable model we included data from participants with non-missing values for all variables in that model. These results were compared with corresponding univariable models based on data from the same participants.

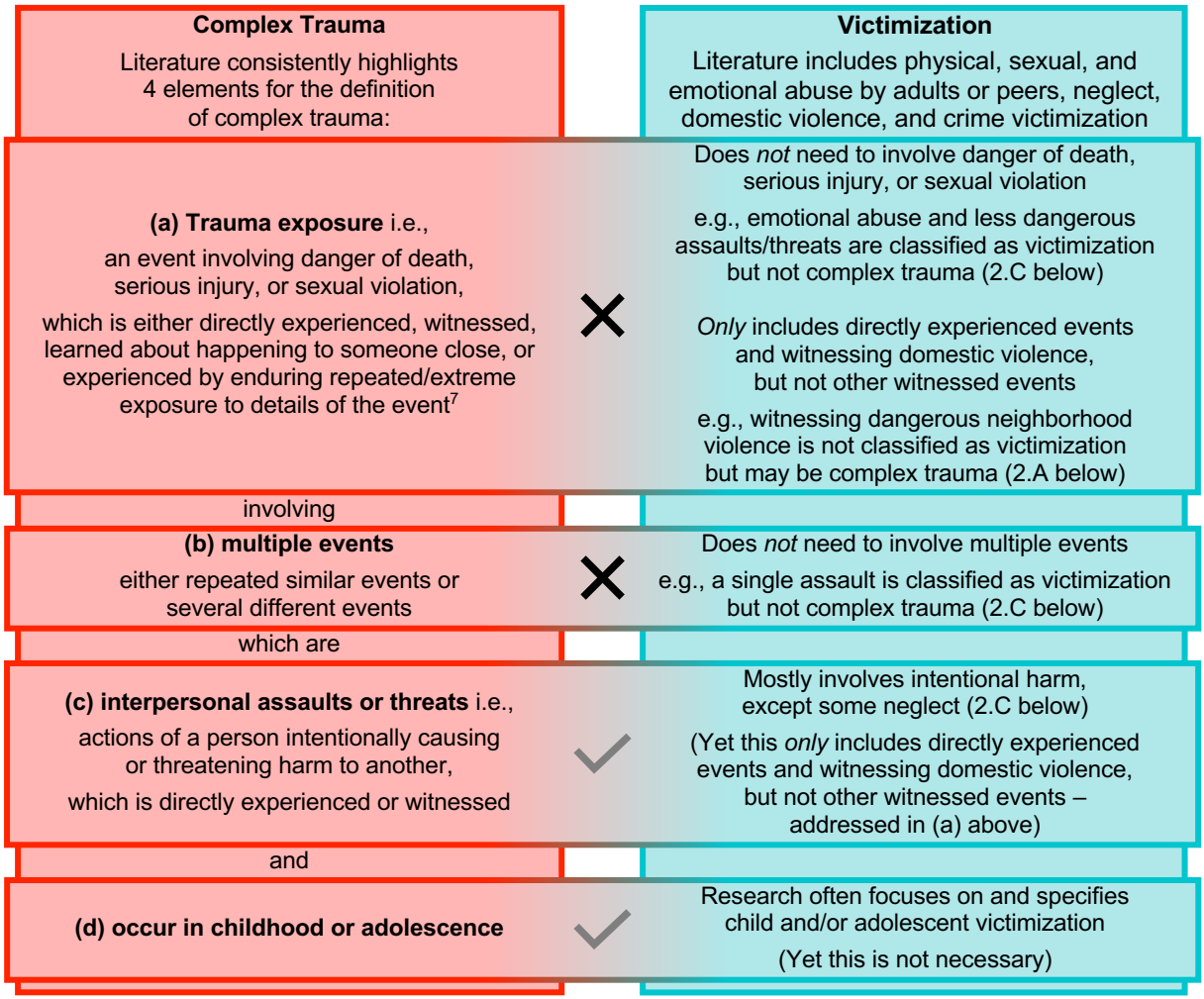
Additionally, we tested whether unobserved genetic or family-wide environmental factors accounted for psychopathology and cognitive deficits linked to complex or non-complex trauma by examining whether differences in trauma exposure were correlated with differences in 'p' or IQ within twin pairs. Because twins share their family environment and half (dizygotic [DZ] twins) or all (monozygotic [MZ] twins) their segregating genes, significant within-pair correlations would indicate that trauma is associated with 'p' or IQ independent of the family environment and genetic influences (specifically, half the genetic influences in analyses of DZ twins or all genetic influences in analyses of MZ twins).

The premise and analysis plan for this project was pre-registered on https://sites.google.com/site/dunedineriskconceptpapers/e-risk-approved/lewis-s. Analyses reported here were checked for reproducibility by an independent data-analyst, who recreated the code by working from the manuscript and applied it to a fresh dataset. We undertook most analyses using Stata version 15,[4] except for multinomial models which were conducted using R version 3.6.1[5] and the multgee package.[6]

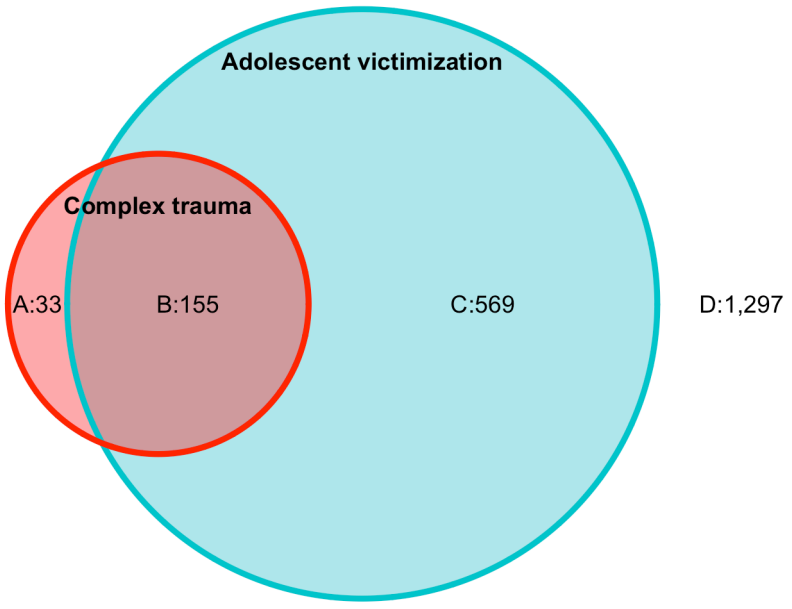
SUPPLEMENTARY FIGURES AND TABLES

Figure S1: Comparison of complex trauma and victimization

1: Comparison of definitions



2: Comparison of exposure rates in the E-Risk Study

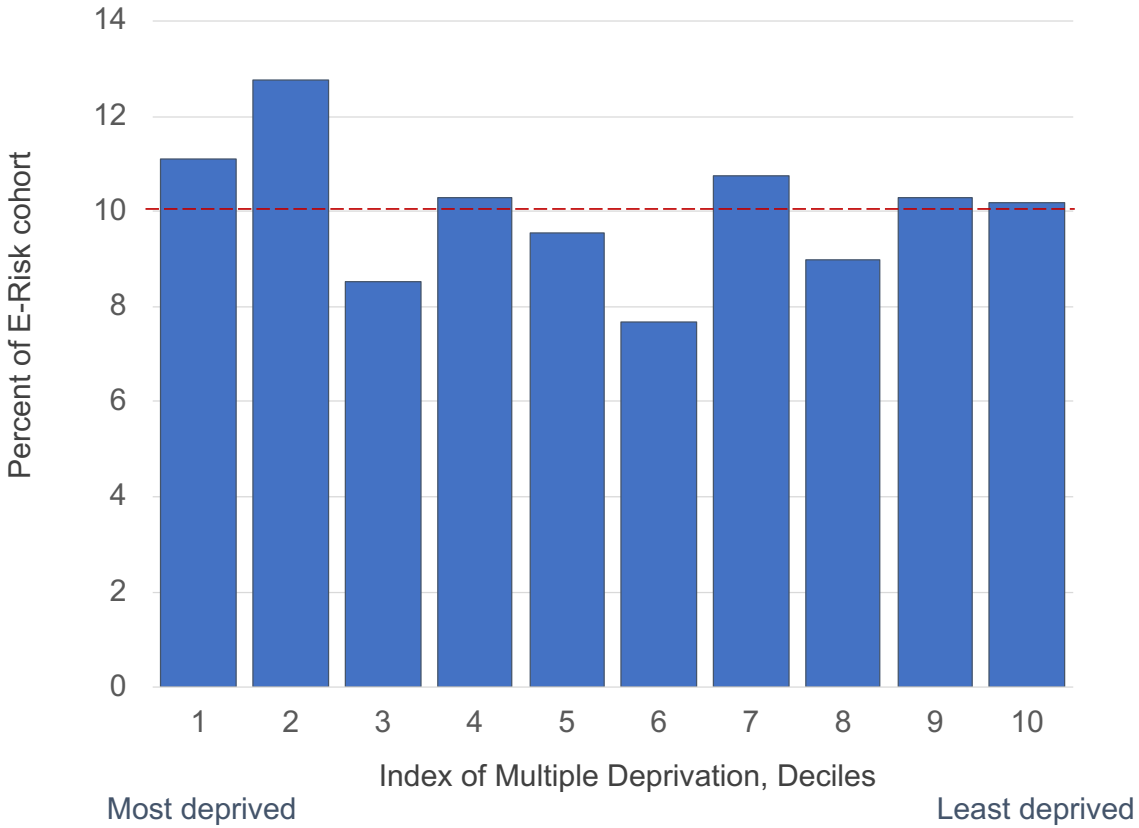


The figure presents a comparison of complex trauma and victimization definitions (Panel 1). There is no formal definition of complex trauma, though previous literature consistently highlights four important elements outlined in Panel 1, first column.[8–11] Victimization is typically defined in terms of a set of experiences listed in Panel 1, second column.[12–14] In the figure we show that there are differences in the complex trauma and victimization definitions: victimization does not need to meet criteria for complex trauma (in particular trauma exposure or multiple events), and some complex traumas may not be classified as experiences typically measured in victimization research.

To consider whether these theoretical differences confer practical differences in the young people identified by measures of complex trauma and victimization, we compared exposure rates in the E-Risk Study (Panel 2). Both complex trauma and adolescent victimization were measured at age 18 years during private interviews with participants. To assess complex trauma, we first asked participants whether they had been exposed to trauma during their lifetime, according to DSM-5 PTSD criterion A.[7] Participants' descriptions of their traumas were then used to identify those exposed to complex trauma, i.e., traumas that involved multiple events which were interpersonal assaults or threats and occurred in childhood or adolescence (see Table S1). To assess adolescent victimization, we used the Juvenile Victimization Questionnaire 2nd Revision adapted as a clinical interview to enquire about participants' exposure to different forms of victimization since age 12 years, grouped into seven categories: crime victimization, peer/sibling victimization, Internet/mobile phone victimization, sexual victimization, family violence, maltreatment, and neglect. Participants' descriptions of these events were used to identify those with severe exposure to one or more of these categories of victimization.[12,13] Childhood victimization was assessed repeatedly when participants were 5, 7, 10, and 12 years of age, based on interviews with mothers, some interviews with participants, and researchers' impressions. Dossiers with this cumulative information were used to identify participants exposed before age 12 years to domestic violence between the mother and her partner, frequent bullying by peers, physical maltreatment by an adult, sexual abuse, emotional abuse/neglect, or physical neglect.[12–14] We found that, although there is some overlap between complex trauma and adolescent victimization (Panel 2.B), most participants who reported adolescent victimization did not describe complex trauma (Panel 2.C), and a small number of participants who described complex trauma did not report adolescent victimization (Panel 2.A). Theoretical examples of why this may have occurred are described in Panel 1, second column. (Participants who did not report complex trauma or adolescent victimization are shown in Panel 2 as D.) We found similar patterns of overlap when comparing complex trauma with childhood victimization, and with childhood or adolescent victimization.

Because the constructs of complex trauma and victimization only partially overlap, it should not be assumed that findings from victimization research necessarily apply to complex trauma. This conclusion is also likely in relation to other similar constructs, such as adverse childhood experiences. Therefore, adequate investigation of complex trauma is needed to accurately understand this construct, which is particularly important because it has become popular and influential in clinical practice.

Figure S2: Population representativeness of the E-Risk Study



The histogram shows that E-Risk families' addresses at age 18 years are a near-perfect match to the deciles of England's Lower-layer Super Output Area (LSOA) Index of Multiple Deprivation 2015 (IMD) which averages 1,500 residents; approximately 10% of the cohort fills each of IMD's 10% bands for England.

Table S1: Assessment of trauma exposure, psychopathology, cognitive function, and early childhood vulnerabilities

Measure	Age, years	Informant	Description	Reporting period	Reference
Trauma exposure					
Complex and non-complex trauma exposure	18	Participant	Trauma exposure was assessed during private interviews. Participants were asked whether they had been exposed to trauma during their lifetime, according to DSM-5 PTSD criterion A; i.e., an event involving actual or threatened death, serious injury, or sexual violation, which was either directly experienced, witnessed, learned about happening to a close member of the person's social network, or experienced by enduring repeated or extreme exposure to details of the event.[7] Participants who reported trauma exposure were then asked to describe the traumas they had experienced. We reviewed these descriptions, alongside information gathered in the Juvenile Victimization Questionnaire 2nd Revision (JVQ-R2)[12] adapted as a clinical interview,[13] to identify participants who had been exposed to complex trauma during their lifetime. **We defined complex trauma exposure as having experienced:** **(a) trauma** (according to DSM-5, noted above), **that involved** **(b) multiple events** (either repeated similar events, or several different events), **which were** **(c) interpersonal assaults or threats** (actions of a person intentionally causing or threatening harm to another; directly experienced or witnessed by the participant), **and** **(d) occurred in childhood or adolescence** (applies to all E-Risk participants in this study, as they were assessed at age 18). These criteria were selected because they have been consistently highlighted as key elements for the definition of complex trauma in previous literature, supported by evidence suggesting that each criterion considered alone is associated with psychopathology or cognitive deficits.[8–11] Examples of complex traumas included repeated child abuse, severe bullying, and witnessing neighborhood violence. Participants were classed as having experienced non-complex trauma if they were exposed to trauma that involved a single event or non-interpersonal events only, i.e., their experience met criterion (a) but not all other criteria needed to indicate complex trauma exposure in their lifetime. Examples of non-complex traumas included a one-off assault, a road-traffic accident, and learning about the sudden death of a parent. Therefore, two mutually exclusive groups of trauma-exposed participants were formed: those exposed to complex trauma (whether or not they were also exposed to non-complex trauma) and those exposed to non-complex trauma (and not complex trauma). Trauma dossiers for each trauma-exposed participant were coded to indicate complex or non-complex trauma exposure by two independent psychiatrists who were blind to any other information about participants (inter-rater reliability of coding for trauma-exposed participants: kappa=0.86). Of all participants with available data, 9.1% (188/2,064) reported complex trauma, 21.7% (448/2,064) reported non-complex trauma, and 0.3% (6/2,064) reported trauma but declined to provide a description (classed as missing and excluded from these analyses).	Lifetime	7–11

Table S1 continued

Measure	Age, years	Informant	Description	Reporting period	Reference
Psychopathology					
Psychopathology dimensions	18	Participant	Derived by fitting a bi-factor model to data collected during interviews about psychopathology. The observed data consisted of 11 symptom scales: post-traumatic stress disorder, major depressive disorder, generalized anxiety disorder, disordered eating, attention-deficit hyperactivity disorder, conduct disorder, alcohol dependence, cannabis dependence, nicotine dependence, psychotic symptoms, and prodromal symptoms. The bi-factor model included a general psychopathology factor 'p', and residual internalizing, externalizing, and thought disorder factors. We focused on 'p' because previous research has found that 'p' captures much of the propensity to psychopathology and the shared variation between victimization and psychopathology.[2,15] Scores were scaled to a mean of 100 and standard deviation of 15.	12 months	15
Post-traumatic stress disorder (PTSD)	18	Participant	Measured using structured interviews to assess DSM-5 diagnostic criteria.	12 months	7,16
Major depressive disorder	18	Participant	Measured using the Diagnostic Interview Schedule for DSM-IV.	12 months	17,18
Generalized anxiety disorder	18	Participant	Measured using the Diagnostic Interview Schedule for DSM-IV.	12 months	17,18
Attention-deficit hyperactivity disorder (ADHD)	18	Participant	Measured using structured interviews to assess DSM-5 diagnostic criteria.	12 months	7,19
Conduct disorder	18	Participant	Measured using self-completed computer-based surveys to assess DSM-IV diagnostic criteria.	12 months	20
Alcohol dependence	18	Participant	Measured using the Diagnostic Interview Schedule for DSM-IV.	12 months	17,18
Cannabis dependence	18	Participant	Measured using the Diagnostic Interview Schedule for DSM-IV.	12 months	17,18
Nicotine dependence	18	Participant	Measured using the Fagerström Test of Nicotine Dependence (FTND). Nicotine dependence was defined as a FTND score ≥4.	12 months	21,22
Psychotic symptoms	18	Participant	Measured using structured interviews to assess for seven psychotic symptoms, validated by experts. Participants who had experienced one or more of these symptoms were classed as having experienced psychotic symptoms.	12 months	23,24
Cognitive function					
IQ	18	Participant	Measured using a short form of the Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV). Using two subtests (Matrix Reasoning and Information), participant's IQs were prorated according to the method recommended by Sattler. Scores were scaled to a mean of 100 and standard deviation of 15.	At time of assessment	25,26

Table S1 continued

Measure	Age, years	Informant	Description	Reporting period	Reference
Cognitive function continued					
Executive function	18	Participant	Tested using subtests of the Cambridge Neuropsychological Test Automated Battery (CANTAB). The measures that tap executive function are described below. – *Rapid visual information processing (RVP) A' (A-prime)* is a signal detection measure that taps sustained attention, often called attentional vigilance. The participant scans for a three-digit target sequence in a digit stream that is ongoing for seven minutes, and responds whenever a target sequence is spotted. At the most difficult level, the participant scans simultaneously for two target sequences. Higher scores are better. – *RVP total false alarms* records impulsive jumping to respond too soon before the correct target digit sequence is complete. Because relatively few participants made numerous false alarms, this measure is categorical, coded 0=none, 1=one false alarm, 2=two or more false alarms. Lower scores are better. – *Spatial working memory (SWM) total errors* assesses capacity to hold information about spatial location in active memory while searching for information. At the most difficult level, participants memorize ten locations in one problem. Lower scores are better. – *SWM strategy* records trials on which the participant applied a problem-solving strategy by opening boxes in a systematic sequence. Lower scores are better (fewer non-strategic trials). – *Spatial span* is the visual non-verbal equivalent of the oral-auditory test Digit Span forward, and measures working memory. White squares are shown, some of which briefly change color in a variable sequence. The participant must then touch the boxes which changed color in the same order that they were displayed. At the most difficult level, participants memorize a sequence of nine colored stimuli. Higher scores are better. – *Spatial span reversed* is the visual non-verbal equivalent of the oral-auditory test Digit Span backward, and is a more difficult measure of working memory. White squares are shown, some of which briefly change color in a variable sequence. The participant must then touch the boxes which changed color in the reverse order that they were displayed. Higher scores are better. Scores were scaled to a mean of 100 and standard deviation of 15. Where lower scores indicated better cognitive functioning, scores were reverse coded so that for all measures of cognitive function lower scores indicated poorer functioning.	At time of assessment	27–29

Table S1 continued

Measure	Age, years	Informant	Description	Reporting period	Reference
Cognitive function continued					
Processing speed	18	Participant	Tested using subtests of the CANTAB. The measures that tap visual-motor processing speed are described below. – *RVP mean latency* measures the latency of response across target signals on the RVP vigilance task, and reflects reaction time to the visual targets. Lower scores are better (faster). – *SWM mean time* measures the time to last response across trials, and reflects how rapidly participants solved visual spatial working memory problems. Lower scores are better (faster). Scores were scaled to a mean of 100 and standard deviation of 15. Scores were reverse coded so that for all measures of cognitive function lower scores indicated poorer functioning.	At time of assessment	27–29
Early childhood vulnerabilities					
Internalizing symptoms	5	Mother, teacher	Measured using Child Behavior Checklist (CBCL) completed by mother and Teacher Report Form (TRF) completed by teacher. Withdrawn, anxious/depressed, and somatic scales ratings were combined and standardized to a mean of 0 and SD of 1.	6 months	30,31
Externalizing symptoms	5	Mother, teacher	Measured using Child Behavior Checklist (CBCL) completed by mother and Teacher Report Form (TRF) completed by teacher. Delinquent behavior and aggressive behavior scales ratings were combined and standardized to a mean of 0 and SD of 1.	6 months	30,31
IQ	5	Participant	Measured using a short form of the Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R). Using two subtests (Vocabulary and Block Design), children's IQs were prorated following procedures described by Sattler. Scores were standardized to a mean of 0 and SD of 1.	At time of assessment	32,33
Proportion of family members with a history of mental illness	12	Mother	Mothers reported on their own mental health history and the mental health history of their biological mother, biological father, biological sisters, biological brothers, as well as the twins' biological father. Mothers were asked to report if anyone on the aforementioned list experienced difficulties with substance use problems, alcohol problems, depression, psychosis, or suicide attempts. Proportions were standardized to a mean of 0 and SD of 1.	Lifetime	34
Lower family socioeconomic status	5	Mother	Family socioeconomic status was defined through a standardized composite of parental income, education, and occupation. The three socioeconomic status indicators were highly correlated (r=0.57-0.67) and loaded significantly onto one latent factor. The population-wide distribution of the resulting factor was divided in tertiles, and reverse coded so that the highest tertile indicates lowest family socioeconomic status.	At time of assessment	35
Female sex	5	Mother	Reported by mothers.	At time of assessment	

Table S2: Associations between trauma exposure and psychopathology, including controlling for early childhood vulnerabilities

	In all participants		In trauma-exposed participants	
	Univariable models	**Multivariable models**	**Univariable models**	**Multivariable models**
A: Psychopathology dimensions	b (95% CI)	b (95% CI)	b (95% CI)	b (95% CI)
p, general psychopathology				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	**8.57 (7.12, 10.02)**	**8.32 (6.91, 9.74)**	0.00 [base]	0.00 [base]
Complex trauma	**16.69 (14.18, 19.21)**	**15.53 (13.07, 18.00)**	**9.14 (6.29, 11.99)**	**7.72 (4.88, 10.56)**
B: Psychiatric disorders count	IRR (95% CI)	IRR (95% CI)	IRR (95% CI)	IRR (95% CI)
Count of below disorders				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	**1.58 (1.38, 1.83)**	**1.54 (1.34, 1.78)**	1.00 [base]	1.00 [base]
Complex trauma	**2.57 (2.15, 3.07)**	**2.44 (2.02, 2.94)**	**1.70 (1.39, 2.08)**	**1.64 (1.33, 2.03)**
C: Psychiatric disorders	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
PTSD				
No trauma	-	-	-	-
Non-complex trauma	-	-	1.00 [base]	1.00 [base]
Complex trauma	-	-	**4.42 (2.71, 7.21)**	**4.07 (2.43, 6.82)**
Major depressive disorder				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	**1.74 (1.35, 2.25)**	**1.70 (1.31, 2.21)**	1.00 [base]	1.00 [base]
Complex trauma	**3.05 (2.15, 4.32)**	**2.54 (1.76, 3.66)**	**1.90 (1.30, 2.77)**	**1.62 (1.08, 2.42)**
Generalized anxiety disorder				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	**1.68 (1.12, 2.50)**	**1.65 (1.10, 2.47)**	1.00 [base]	1.00 [base]
Complex trauma	**3.67 (2.32, 5.82)**	**3.27 (2.02, 5.30)**	**2.27 (1.37, 3.79)**	**2.12 (1.22, 3.67)**
ADHD				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	**1.78 (1.23, 2.56)**	**1.84 (1.26, 2.69)**	1.00 [base]	1.00 [base]
Complex trauma	**2.17 (1.37, 3.43)**	**1.80 (1.10, 2.96)**	1.21 (0.72, 2.04)	0.90 (0.49, 1.63)
Conduct disorder				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	**1.91 (1.46, 2.50)**	**2.02 (1.51, 2.68)**	1.00 [base]	1.00 [base]
Complex trauma	**2.86 (1.99, 4.13)**	**3.33 (2.22, 5.01)**	**1.54 (1.02, 2.34)**	1.61 (1.00, 2.59)
Alcohol dependence				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	**1.46 (1.07, 1.99)**	**1.43 (1.04, 1.94)**	1.00 [base]	1.00 [base]
Complex trauma	**1.75 (1.14, 2.67)**	**1.79 (1.16, 2.76)**	1.20 (0.76, 1.89)	1.32 (0.82, 2.13)
Cannabis dependence				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	1.47 (0.85, 2.55)	1.42 (0.80, 2.52)	1.00 [base]	1.00 [base]
Complex trauma	**3.14 (1.72, 5.71)**	**2.83 (1.50, 5.35)**	**2.28 (1.15, 4.50)**	**2.07 (1.00, 4.27)**
Nicotine dependence				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	0.96 (0.65, 1.43)	0.96 (0.64, 1.45)	1.00 [base]	1.00 [base]
Complex trauma	**2.65 (1.70, 4.15)**	**2.34 (1.45, 3.78)**	**2.87 (1.74, 4.75)**	**2.48 (1.44, 4.27)**
Psychotic symptoms				
No trauma	1.00 [base]	1.00 [base]	-	-
Non-complex trauma	1.13 (0.40, 3.18)	1.09 (0.40, 2.97)	1.00 [base]	1.00 [base]
Complex trauma	**5.83 (2.71, 12.54)**	**4.80 (2.19, 10.50)**	**5.95 (2.30, 15.38)**	**4.43 (1.46, 13.49)**

Linear regression coefficients (b), incidence rate ratios (IRR), and odds ratios (OR) with 95% confidence intervals (95% CI) are presented for associations between trauma exposure and psychopathology in all participants (n=1,965-1,990) and in trauma-exposed participants (n=613-620). Univariable (unadjusted) model results are presented. Additionally, we present results from multivariable models adjusted for the effects of early childhood vulnerabilities (internalizing symptoms at age 5, externalizing symptoms at age 5, IQ at age 5, proportion of family members with a history of mental illness, lower family socioeconomic status at age 5, and female sex). Where data were missing, for each multivariable model we included data from participants with non-missing values for all variables in that model. The corresponding univariable model included data from the same participants as the multivariable model. Bold text signifies $p<0.05$. These data are shown graphically in Figure 3.

Table S3: Correlations between differences in trauma exposure and differences in 'p' within twin pairs

	DZ and MZ twin pairs		DZ twin pairs only		MZ twin pairs only	
	r	p	r	p	r	p
p, general psychopathology	**0.3066**	**<0.0001**	**0.3070**	**<0.0001**	**0.3072**	**<0.0001**

It is not possible to carry out these analyses with an exposure variable that has more than two categories and is not ordered. Therefore, for this analysis we ordered the trauma variable (0=no trauma, 1=non-complex trauma, and 2=complex trauma). This seems reasonable given the observation that, compared to participants exposed to no trauma, those exposed to non-complex trauma had higher scores of 'p', and those exposed to complex trauma had even higher scores of 'p' (see Figure 1). This variable was correlated with 'p' in all participants (n=2,058, r=0.3984, p<0.0001; and in those with non-missing co-twin data included in this twin analysis n=2,034, r=0.3952, p<0.0001).

Pearson correlation coefficients (r) and their p-values (p) are presented for correlations between differences in trauma exposure and differences in 'p' within twin pairs (DZ n_{pairs}=442, MZ n_{pairs}=575).

The results show that twins exposed to higher scores of trauma (when coded 0=no trauma, 1=non-complex trauma, and 2=complex trauma) had greater risk of general psychopathology compared to their co-twin with lower scores of trauma, indicating that trauma is associated with general psychopathology independent of family environment and genetic risk.

Table S4: Associations between trauma exposure and cognitive function, including controlling for early childhood vulnerabilities

	In all participants		In trauma-exposed participants	
	Univariable models	**Multivariable models**	**Univariable models**	**Multivariable models**
A: IQ	b (95% CI)	b (95% CI)	b (95% CI)	b (95% CI)
WAIS-IV				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	0.88 (-0.46, 2.22)	0.61 (-0.62, 1.83)	0.00 [base]	0.00 [base]
Complex trauma	**-2.37 (-4.42, -0.33)**	-0.86 (-2.71, 0.99)	**-4.38 (-6.75, -2.02)**	-2.01 (-4.19, 0.16)
B: Executive function	b (95% CI)	b (95% CI)	b (95% CI)	b (95% CI)
RVP A'				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	-0.07 (-1.58, 1.44)	-0.10 (-1.54, 1.34)	0.00 [base]	0.00 [base]
Complex trauma	**-3.03 (-5.47, -0.60)**	-1.90 (-4.33, 0.52)	**-3.42 (-6.07, -0.77)**	-1.77 (-4.42, 0.88)
RVP false alarms (reverse coded)				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	-0.95 (-2.58, 0.68)	-0.90 (-2.51, 0.72)	0.00 [base]	0.00 [base]
Complex trauma	-1.83 (-4.30, 0.64)	-1.17 (-3.60, 1.27)	-1.16 (-3.92, 1.59)	-0.55 (-3.30, 2.20)
SWM errors (reverse coded)				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	0.04 (-1.42, 1.49)	-0.14 (-1.56, 1.28)	0.00 [base]	0.00 [base]
Complex trauma	**-2.83 (-5.11, -0.55)**	-2.22 (-4.49, 0.05)	**-3.62 (-6.27, -0.97)**	-2.62 (-5.35, 0.10)
SWM strategy (reverse coded)				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	0.59 (-0.96, 2.13)	0.38 (-1.12, 1.88)	0.00 [base]	0.00 [base]
Complex trauma	-0.97 (-3.12, 1.19)	-0.09 (-2.20, 2.03)	-2.09 (-4.61, 0.42)	-0.78 (-3.32, 1.76)
Spatial span				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	-0.13 (-1.63, 1.37)	-0.31 (-1.76, 1.13)	0.00 [base]	0.00 [base]
Complex trauma	**-3.67 (-5.89, -1.44)**	**-2.63 (-4.78, -0.48)**	**-3.82 (-6.27, -1.37)**	**-2.57 (-5.01, -0.14)**
Spatial span reversed				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	-0.71 (-2.16, 0.75)	-0.86 (-2.28, 0.56)	0.00 [base]	0.00 [base]
Complex trauma	**-2.74 (-4.90, -0.58)**	-1.54 (-3.69, 0.62)	-2.42 (-4.85, 0.01)	-1.21 (-3.58, 1.15)
C: Processing speed	b (95% CI)	b (95% CI)	b (95% CI)	b (95% CI)
RVP mean latency (reverse coded)				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	-0.31 (-1.90, 1.28)	-0.20 (-1.80, 1.40)	0.00 [base]	0.00 [base]
Complex trauma	**-6.04 (-8.68, -3.41)**	**-5.18 (-7.83, -2.53)**	**-5.59 (-8.47, -2.70)**	**-4.66 (-7.50, -1.81)**
SWM mean time (reverse coded)				
No trauma	0.00 [base]	0.00 [base]	-	-
Non-complex trauma	0.33 (-0.91, 1.58)	0.14 (-1.08, 1.36)	0.00 [base]	0.00 [base]
Complex trauma	**-3.35 (-5.77, -0.92)**	**-3.03 (-5.39, -0.67)**	**-4.48 (-7.22, -1.73)**	**-3.88 (-6.51, -1.24)**

Linear regression coefficients (b) with 95% confidence intervals (95% CI) are presented for associations between trauma exposure and cognitive function in all participants (n=1,973-1,983) and in trauma-exposed participants (n=-614-618). Univariable (unadjusted) model results are presented. Additionally, we present results from multivariable models adjusted for the effects of early childhood vulnerabilities (internalizing symptoms at age 5, externalizing symptoms at age 5, IQ at age 5, proportion of family members with a history of mental illness, lower family socioeconomic status at age 5, and female sex). Where data were missing, for each multivariable model we included data from participants with non-missing values for all variables in that model. The corresponding univariable model included data from the same participants as the multivariable model. Bold text signifies $p<0.05$. These data are shown graphically in Figure 4.

Table S5: Correlations between differences in trauma exposure and differences in IQ within twin pairs

	DZ and MZ twin pairs		DZ twin pairs only		MZ twin pairs only	
	r	p	r	p	r	p
IQ: WAIS-IV	-0.0470	0.1355	-0.0834	0.0814	-0.0106	0.8000

It is not possible to carry out these analyses with an exposure variable that has more than two categories and is not ordered. The ordered trauma variable (0=no trauma, 1=non-complex trauma, and 2=complex trauma) was not correlated with IQ in all participants (n=2,048, r=-0.0394, p=0.0748). Therefore, for this analysis we compared participants exposed to complex trauma with those exposed to no trauma or non-complex trauma (0=no trauma/non-complex trauma, and 1=complex trauma). This seems reasonable given the observation that, compared to participants exposed to no trauma, those exposed to non-complex trauma had similar scores of IQ, but those exposed to complex trauma had lower scores of IQ (see Figure 2). This variable was correlated with IQ in all participants (n=2,048, r=-0.0688, p=0.0018; and in those with non-missing co-twin data included in this twin analysis n=2,018, r=-0.0662, p=0.0029).

Pearson correlation coefficients (r) and their p-values (p) are presented for correlations between differences in trauma exposure and differences in IQ within twin pairs (DZ n_{pairs}=438, MZ n_{pairs}=571).

The results show that complex trauma was not correlated with IQ within twin pairs, suggesting that associations observed at the individual level were likely explained by family environment and genetic factors.

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