

# Severe childhood trauma and clinical and neurocognitive features in schizotypal personality disorder


Velikonja T, Velthorst E, McClure MM, Rutter S, Calabrese WR, Rosell D, Koenigsberg HW, Goodman M, New AS, Hazlett EA, Perez-Rodriguez MM. Severe childhood trauma and clinical and neurocognitive features in schizotypal personality disorder.

**Objective:** Literature suggests that childhood trauma increases vulnerability for schizophrenia-spectrum disorders, including schizotypal personality disorder (SPD). Yet, it remains unexplored whether childhood trauma predicts symptom load and the level of neurocognitive functioning in SPD.

**Method:** We included 225 individuals with SPD and 127 healthy controls. Childhood trauma was evaluated using the Childhood Trauma Questionnaire, and schizotypal traits were assessed using the Schizotypal Personality Questionnaire. Standard neurocognitive assessments covered six cognitive domains.

**Results:** All types of reported childhood trauma were significantly associated with SPD, in a linear fashion. Severe sexual abuse showed the greatest magnitude of association with higher cognitive–perceptual load (e.g., ideas of reference, odd belief or magical thinking); severe emotional neglect was associated with interpersonal scores (e.g., excessive social anxiety, constricted affect) within the SPD group. SPD individuals who reported severe trauma showed worse cognitive functioning (i.e., working memory, verbal/visual learning and memory, as well as verbal fluency).

**Conclusions:** Particular severe childhood trauma types were associated with higher cognitive–perceptual and interpersonal symptoms in SPD, along with worse cognitive functioning. These findings highlight the need for clinicians to enquire about childhood trauma in SPD patients, since unaddressed early adverse experiences may carry long-term negative consequences.

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Key words: childhood adversity; abuse; neglect; schizophrenia spectrum; psychosis; cognition

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## Significant outcomes

- Sexual abuse and emotional abuse were the types of childhood trauma most strongly associated with a SPD diagnosis, especially in severe forms.
- Severe forms of sexual abuse showed the strongest magnitude of association with cognitive–perceptual traits, while severe emotional neglect showed the strongest magnitude of association with interpersonal symptoms (e.g., excessive social anxiety, constricted affect) among SPD.
- Severe childhood trauma was associated with worse overall cognitive functioning among SPD.

## Limitations

- The evaluation of childhood trauma was based on self-report measures.
- A cross-sectional study design does not allow any conclusions about directionality of the associations between childhood trauma types and increased schizotypal personality disorder traits. Causality is compromised by the assumption that childhood trauma preceded the development of SPD.
- The neurocognitive assessments were only completed for a subset of our sample, precluding more detailed analysis (e.g., exploring the associations between cognition and different trauma types, sex differences).

## Introduction

Schizotypal personality disorder (SPD) is a milder, non-psychotic schizophrenia-spectrum disorder (1). There is robust evidence highlighting genetic (2), neurobiological (3), and etiological overlap between SPD and schizophrenia. As such, studying pathways to SPD might provide essential clues for early identification, treatment, and possible prevention of full-blown psychosis, without potential confounding effects of the illness (e.g., medication, hospitalization). Given that heritability estimates of SPD usually fall between 50% and 80% (4, 5), it is likely that the etiology of SPD symptoms involves both genetic and environmental factors. Significant associations between childhood traumatic experiences and the development of broader schizotypy (6–9) and SPD (10–13) have been consistently documented, suggesting that childhood victimization may increase vulnerability for the disorder. Furthermore, evidence supports a dose–response relationship between trauma and disorders along schizophrenia spectrum (9, 14, 15). While the strongest associations are found for emotional abuse (13) and neglect (16), in more severe/chronic forms (15), some fundamental questions have remained unexplored.

In particular, it is unclear whether childhood trauma is associated with higher SPD rates in general, or whether there are specific associations between particular trauma types and symptoms/traits within the SPD population. There are differences in genetic/familial effects across dimensions of schizotypal personality (e.g., small genetic effect for cognitive–perceptual domain, medium for interpersonal, and large for disorganized domain (5)). Additionally, different trauma types may underlie different symptomatology (9, 13) and, therefore, warrant a different therapeutic approach. Overall, childhood trauma has been shown to be associated with an increased likelihood of experiencing cognitive–perceptual (also referred to as ‘positive’, given their similarity to the positive symptoms of schizophrenia) schizotypal symptoms/traits (9), but not exclusively so (13, 17). Similarly, while some symptom-specific effects of individual trauma types have been proposed [e.g., physical abuse and unusual perceptions (13), sexual abuse and paranoia (7), or eccentric behaviour (13)], others have suggested no significant difference across maltreatment types (18). The present study begins to address these questions in a large well-characterized sample of individuals with SPD.

Studies of the association between childhood trauma experiences and SPD have largely focused

on positive symptomatology (8, 19) but little is known about negative symptoms or cognitive impairment, which are among the most prominent characteristics of SPD (20). No studies to date have explored the relationship between childhood trauma and neurocognition in this population, despite evidence underlining marked cognitive deficits in SPD (21, 22). Some indirect evidence linking trauma to cognitive difficulties comes from general population studies, showing impairments in victimized groups on executive function (23), working memory (24), and attention (25). For example, in a twin study, children who were exposed to domestic violence showed on average, eight points lower IQ compared to children who did not experience abuse, independent of confounding genetic effects (26). These adverse outcomes of childhood victimization might not be limited to childhood, but have effects on cognition that persist into adulthood (27). In studies of adult clinical groups, both in first-episode psychosis (28) and in more chronic schizophrenia patients (29), childhood trauma was shown to contribute to some of the neuropsychological impairments, such as processing speed (29), working memory (30), verbal fluency (31), and learning and visual context processing (30). Examining the association between childhood victimization and cognition in SPD may help elucidate this relationship in the schizophrenia spectrum. Studying SPD has many advantages including eliminating confounds such as psychoactive medication and hospitalization that plague schizophrenia research. Cognitive deficits observed in SPD individuals who experienced childhood trauma (and not in those without such experiences), could imply that impaired cognitive functioning in schizophrenia patients might be the effect of early victimization rather than treatment (e.g., antipsychotic medication) or illness itself. There is very little empirical support trying to explain the possible mechanism underlying the relationship between neurocognitive impairments in adults with psychosis (or SPD) who are also survivors of childhood trauma. Primarily, the traumagenic neurodevelopmental (TN) model (32) postulates that sufficiently severe trauma can contribute to pathological alternation of neurodevelopmental processes, such as changes to hypothalamic–pituitary–adrenal (HPA) axis (33). Other hypotheses imply more indirect effects of childhood trauma on neurocognition; for example, cognitive impairments due to lack of stimuli or under-nutrition, frequently found in families where childhood maltreatment is happening (34), or genetic load that carries both a higher risk of childhood trauma and more impaired cognitive abilities (28).

The present study aims to explore the associations between different types and degrees of severity of childhood trauma and SPD. Furthermore, the study aims to evaluate clinical and neurocognitive features among individuals with SPD, with and without experiences of severe childhood victimization. The analysis of neurocognitive data needs to be considered as exploratory, given this information was only available for a subset of the sample. Advancing knowledge on the relationship between childhood trauma and SPD may provide important clues for the etiology of schizophrenia-spectrum disorders and carries important implications for clinical practice.

## Methods

### Participants

As part of an ongoing examination of the neurobiology and cognitive impairment of individuals with schizotypal personality disorder, we enrolled 225 individuals with DSM-IV schizotypal personality disorder (SPD) and 127 healthy controls (HC). Participants were recruited over the course of 20 years (1998–2018). All participants (SPD and HC) were aged between 18 and 64 (mean = 36.6, SD = 11.7), either non-treatment seeking individuals recruited from the community through online and print advertisements, or referred from the out-patient psychiatry clinics at the Icahn School of Medicine at Mt. Sinai and the James J. Peters VA Medical Center. Diagnostic interviews were conducted by masters- or doctoral-level psychologists supervised by a clinical psychologist with expertise in the diagnosis of personality disorders (MMM), using the Structured Clinical Interview for DSM-IV Axis I Disorders [SCID-IV (35)] and Structured Clinical Interview for DSM-IV Personality Disorders [SIDP-IV (36)]. More recently, studies transitioned to the use of DSM-5 (37) and SCID-5 (38) (nb. criteria for the SPD between the DSM-IV and DSM-5 editions have remained unchanged). Over the course of recruitment, there had been approximately 10 SIDP raters, with one rater performing all diagnostic interviews during a given time (supervised by MMM). The interrater reliability for a SPD diagnosis in our research group was 0.73 (*Kappa coefficient*), which reflects good strength of agreement (39). Potential participants were excluded if they met criteria for bipolar I disorder, schizophrenia, schizoaffective disorder or other psychotic disorder, seizure disorder, had a lifetime history of significant head trauma, had met criteria for

substance abuse or dependence within the last three months, or were currently taking a psychotropic medication. The sociodemographic characteristics of the sample are presented in Table 2. Data on neurocognitive measures were only available for a subset of SPD ( $N = 32$ ) and HC ( $N = 23$ ) individuals, who participated in study protocols that involved cognitive testing. For a full description of recruitment and inclusion/exclusion criteria for this subset of individuals, see previous publications (40–42).

All studies were approved by the Mount Sinai and James J. Peters VA Medical Center (JJPVAMC) Institutional Review Boards. Written consent was obtained in accordance with IRB and institutional guidelines.

### Measures

**Childhood trauma.** Childhood trauma was evaluated using the Childhood Trauma Questionnaire (CTQ (43)). This is a self-report tool, containing 28 items rated on a 5-point scale; ranging from 1 (never true) to 5 (very often true). The measure assesses five types of childhood trauma up to the age of 18: Emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. The total CTQ score falls between 25 and 125 (5–25 scores per subscale/childhood trauma subtype). According to the original CTQ manual (44), the sum of the subscales is categorized into four levels of severity: No trauma, low trauma, moderate trauma, and severe trauma. Studies suggest that CTQ is a valid and reliable instrument (internal consistency of 0.95) to assess childhood trauma in non-clinical (45) and clinical (43, 46) samples. For the purpose of analysis and consistent with previous studies on childhood trauma and SPD (13), dichotomized CTQ scores were used to categorize childhood trauma ‘Absence’ and ‘Presence’. First, all trauma severities (low/moderate/severe) were considered as ‘Trauma Present’ and No trauma as ‘Trauma Absence’. Second, the scores were dichotomized into ‘Severe’ type of trauma vs. ‘No trauma or Non-severe trauma’ (low/moderate trauma), using the following cutoff points (*see CTQ manual*) (44): 16 or higher for emotional abuse, 13 or higher for physical abuse, 13 or higher for sexual abuse, 18 or higher for emotional neglect, and 13 or higher for physical neglect.

**Schizotypal symptom severity.** Schizotypal personality traits were evaluated using the Schizotypal Personality Questionnaire [SPQ (47)]. The measure was originally developed to assess schizotypal personality disorder according to the DSM-III-R (48).

The SPQ is a self-report measure, covering 74 items with a Yes/No format. The items are grouped into nine subscales, which can be further grouped into second-order factors. There is a general agreement that although SPQ measures a multidimensional construct, it is best conceptualized with a three-factor structure (49, 50), including (i) cognitive-perceptual factor (ideas of reference, odd beliefs or magical thinking, unusual perceptual experiences, and suspiciousness subscales); (ii) interpersonal factor (excessive social anxiety, no close friends, constricted affect, and suspiciousness subscales), and (iii) disorganized factor (odd or eccentric behaviour and odd speech subscales). The total SPQ score ranges between 0 and 74. The measure has a high internal reliability (0.91; test-retest = 0.82) and discriminant and criterion validity (0.62 and 0.68 respectively) (47). In this study, the total scores on each of the three factors were used as continuous variables. All mean scores of the nine subscales across severe trauma types are presented in Fig. 1.

**Neurocognitive assessments.** Ten assessments of neurocognitive functioning were included (listed in Table 1) for the purpose of this analysis covering six cognitive domains identified in the MATRICS Consensus Cognitive Battery (51).

In addition, a sociodemographic questionnaire was used to determine the participant's age, gender, race (subsumed under four categories: White (1), Black (2), Asian (3), and Other (4)), years of

education, and county of birth (United States (1), Europe (2), Asia (3), Caribbean (4), Mexico and South America (5), and Other (6)).

**Statistical analysis.** Logistic regressions were carried out to explore the main associations between all five childhood trauma subtypes measured and SPD diagnosis. Logistic regressions were then repeated to explore the associations between SPD and moderate/severe childhood trauma, or between SPD and severe childhood trauma alone. All analyses were adjusted for age (continuous variable), gender and race (categories listed above), years of education (continuous variable), and country of birth. To reduce the risk of type I errors because of multiple testing, a  $P$  value  $< 0.01$  was deemed significant (Bonferroni correction) (52).

Linear regressions were run to investigate the associations between severe childhood trauma types in the SPD group and each of the three factors of the SPQ (cognitive-perceptual, interpersonal, and disorganized). Analysis of covariance (ANCOVA) was used to compare performance across neurocognitive domains in SPD individuals with and without reports of severe childhood trauma. Analyses were covaried for age, gender, and race. Due to the number of tests for cognitive functioning, a statistical significance of  $P < 0.01$  was considered (52). Cohen's  $d$  effect sizes were computed based on differences in mean scores. Effect sizes of 0.2, 0.5, and 0.8 reflect 'small', 'medium', and 'large' effects respectively (53). We

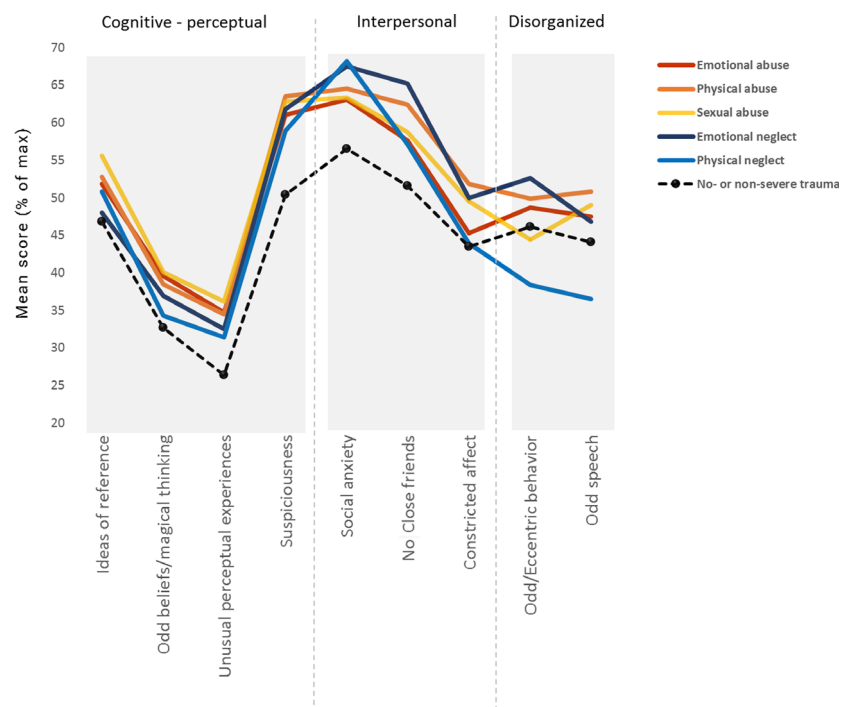


Fig. 1. Mean scores (in percentage of maximum score) on 9 subscales of schizotypal personality questionnaire across severe trauma types among SPD. The maximum score for odd beliefs/magical thinking and odd/eccentric behavior is 7; for suspiciousness, social anxiety, and constricted affect is 8; and for ideas of reference, unusual perceptual experiences, no close friends, and odd speech is 9. [Colour figure can be viewed at wileyonlinelibrary.com]



Table 1. List of assessment measures used (by domain)

Domain	N. of measures	Measures/description
Processing speed	2	The Trail Making Test (TMT), Part A (122); the participant is asked to connect numbers presented on a standard sheet of paper in ascending order (1,2). The dependent variable is the amount of time required to complete the task
Working memory	4	The Symbol Coding subtest of Brief Assessment of Cognition in Schizophrenia (123); the subject is asked to fill in the corresponding numbers beneath each mark. The dependent variable is the sum of correct numbers filled in within 90 s Spatial Span subtest of the Wechsler Memory Scale, Third Edition (WMS3) (124); during which the participant's nonverbal working memory is assessed. The dependent variables are the total scores for spatial span forward and backward During the Letter Number Span (125) task, the participant is asked to recall and rearrange in numerical and alphabetical order varying length strings of letters or numbers. The dependent variables are the length of the longest span and the sum of all correct responses The DOT (126) test measures visuospatial working memory; during this assessments, the participant is presented with a dot at a specific position on a standard size paper and then asked to reproduce it at the same location on a separate sheet after either no delay or after a 30-s delay. For each trial, the distance in cm between the stimulus and the dot drawn by the participant is calculated (distance error). The dependent variable is the difference between the average distance error during no delay trials and the average distance error during 30-s delay trials.
Verbal learning and memory	1	The Paced Auditory Serial Addition Test (PASAT) (127) assesses verbal/auditory working memory. The participant listens to a tape-recorded voice presenting a series of numbers and is asked to add each adjacent pair of numbers and respond by verbalizing the sum. The total number of correct responses is the dependent variable
Visuospatial learning and memory	1	Hopkins Verbal Learning Test—Revised (HVL-R) (128); the participant is asked to repeated a list of 12 words in 3 taxonomic categories over three trials. The dependent variable is the total number of correct recalls across three trials
Verbal fluency	1	Brief Visuospatial Memory Test—Revised (BVM-R) (129); the participant is asked to reproduce 6 geometric figures from memory, assessing visual learning. Scores are based on the accuracy of the drawings and the location of the figures
Reasoning and problem solving/executive functioning	1	Category Fluency: Animal Naming (130); during which the participant is asked to name as many animals as possible in 60 s, assessing semantic processing. The dependent variable is the number of accepted words The Mazes subtest of Neuropsychological Assessment Battery (NAB) (131); the participant is asked to trace a route through mazes of increasing difficulty. The dependent variable is the total score, based on correctly completed mazes and the time required to complete them

also explored the comorbidity of SPD with traits from all other personality disorders (based on DSM criteria). The highest frequencies (between 41% and 68%) were observed for traits reflecting paranoid personality disorder (PPD); with 36.9% of SPD individuals meeting threshold criteria for PPD. When we accounted for PPD in our analyses, there was no impact on our outcomes. All data were analyzed using STATA 15 (54).

## Results

The sample consisted of 225 (63.9%) individuals with DSM-IV schizotypal personality disorder (SPD) and 127 (36.1%) healthy controls (HC); 54.1% of the total samples were males and 45.9% were females. The sociodemographic characteristics of the SPD and HC groups are presented in Table 2.

### Trauma prevalence

Childhood trauma (including all severity levels) was reported by 45.7% individuals in the HC group and by 91.6% individuals in the SPD group; severe trauma by 7.9% of HC and 56.4% of SPD individuals. The most frequently reported childhood trauma types in both HC and SPD groups were emotional neglect (29.1% and 74.7%, respectively) and emotional abuse (22.8% and 68%

respectively). Two or more types of severe childhood trauma were reported by 2.4% of HC and 29.8% of SPD. The difference in reported trauma in HC and SPD participants was most pronounced for physical neglect (14.2% and 54.7% respectively). All trauma frequencies are presented in Table 2.

### Associations between childhood trauma types and SPD

All subtypes of reported childhood trauma were associated with SPD (ORs between 4.18 and 8.74; all  $P$ 's < 0.001; see Table 2). The strongest associations were observed for reported emotional abuse (OR = 8.74, 95% CI = 4.63–16.47) and emotional neglect (OR = 7.33, 95% CI = 4.01–13.23). When looking at severe types of trauma only, the magnitude of associations increased, and all reported subtypes of trauma were associated with SPD (ORs = 9.35–17.75;  $P$ 's = <0.001–0.015). Sexual abuse (OR = 17.75, 95% CI = 3.69–85.46) and emotional abuse (OR = 16.51, 95% CI = 4.78–57.05) showed the strongest associations with SPD. If only one type of severe childhood trauma was reported, the OR was 9.27 (95% CI = 3.60–23.89,  $P$  < 0.001); if two or more types of severe trauma were reported, the OR was 35.83 (95% CI = 8.05–159.53,  $P$  < 0.001). The overall model statistics are presented in Supplemental Material.

## Severe childhood trauma and SPD

Table 2. Trauma frequencies (and % within the group) reported by the healthy control group and the SPD group and the associations (OR) between the different childhood trauma types and SPD

	HC	SPD	Total
<i>N</i> (%)	127 (36.1)	225 (63.9)	352 (100.0)
Gender* (%)			
Female	56.9	40.0	45.9
Male	43.1	60.0	54.1
Age† (Mean, SD)	32.8 (11.2)	38.6 (11.5)	36.6 (11.7)
Race‡ (%)			
White	49.1	37.1	41.3
Black	22.4	46.5	38.0
Asian	22.4	8.4	13.4
Other	6.1	8.0	7.3
Years of education§ (Mean, SD)	16.1 (2.9)	14.1 (2.8)	14.8 (2.9)
Country of birth¶			
United States	69.0	75.4	73.1
Mexico and S. America	4.3	4.7	4.6
Asia	14.7	6.2	9.2
Caribbean	3.4	7.6	6.1
Europe	6.0	2.8	4.0
Other	2.6	3.3	3.0

	<i>N</i> (%)	<i>N</i> (%)	<i>N</i> (%)	OR	95% CI	<i>P</i> -value
Any trauma						
Low/Moderate/Severe	58 (45.7)	206 (91.6)	264 (75.0)	10.45	5.22–20.93	<0.001
Moderate/Severe	32 (25.2)	173 (76.9)	205 (58.2)	8.35	4.55–15.30	<0.001
Severe	10 (7.9)	127 (56.4)	137 (38.9)	15.68	6.88–35.74	<0.001
Distinct trauma types						
Emotional abuse						
Low/Moderate/Severe	29 (22.8)	153 (68.0)	182 (51.7)	8.74	4.63–16.47	<0.001
Moderate/Severe	10 (7.9)	108 (48.0)	118 (33.5)	13.45	5.69–32.02	<0.001
Severe	3 (2.4)	67 (29.8)	70 (19.9)	16.51	4.78–57.05	<0.001
Physical abuse						
Low/Moderate/Severe	23 (18.1)	129 (57.3)	152 (43.2)	4.18	2.26–7.72	<0.001
Moderate/Severe	11 (8.7)	94 (41.8)	105 (29.8)	6.22	2.86–13.52	<0.001
Severe	3 (2.4)	47 (20.9)	50 (14.2)	9.35	2.65–32.99	0.001
Sexual abuse						
Low/Moderate/Severe	16 (12.6)	97 (43.1)	113 (32.1)	5.52	2.69–11.38	<0.001
Moderate/Severe	12 (9.5)	76 (33.8)	88 (25.0)	4.60	2.15–9.85	<0.001
Severe	3 (2.4)	46 (20.4)	49 (13.9)	17.75	3.69–85.46	<0.001
Emotional neglect						
Low/Moderate/Severe	37 (29.1)	168 (74.7)	205 (58.2)	7.33	4.01–13.23	<0.001
Moderate/Severe	10 (7.8)	97 (43.1)	107 (30.4)	11.38	4.77–27.18	<0.001
Severe	5 (3.9)	53 (23.6)	58 (16.5)	10.62	3.05–36.65	<0.001
Physical neglect						
Low/Moderate/Severe	18 (14.2)	123 (54.7)	141 (40.1)	5.67	2.69–10.86	<0.001
Moderate/Severe	6 (4.7)	76 (33.8)	82 (23.3)	8.07	3.02–21.60	<0.001
Severe	1 (0.8)	35 (15.6)	36 (10.2)	12.70	1.63–98.69	0.015

HC, healthy controls; SPD, schizotypal personality disorder; SD, standard deviation; OR, odds ratio.

\*Pearson  $\chi^2 = 8.662$ ,  $P = 0.003$ .

† $t$ -test =  $-4.453$ ,  $P < 0.001$ .

‡Pearson  $\chi^2 = 25.423$ ,  $P < 0.001$ .

§ $t$ -test =  $5.925$ ,  $P < 0.001$ .

¶Fisher's exact test,  $P = 0.066$ .

||Adjusted for age, gender, race, years of education, and country of birth.

Associations between severe childhood trauma types and three-factor structure of SPD

Severe forms of reported sexual abuse ( $\beta = 2.30$ , 95% CI =  $-0.21$ – $4.82$ ,  $P = 0.072$ ) showed the greatest magnitude of association with higher levels of cognitive–perceptual symptoms/traits in the SPD group, but this fell short of statistical

significance (see Table 3). Severe emotional neglect ( $\beta = 3.82$ , 95% CI =  $0.29$ – $7.36$ ,  $P = 0.034$ ) showed the strongest association with higher interpersonal symptoms (with a trend observed for severe physical abuse:  $\beta = 3.38$ , 95% CI =  $-0.22$ – $6.98$ ,  $P = 0.065$ ). No such relationship was observed for the disorganized domain.

The overall model statistics are presented in Supplemental Material.

#### Severe childhood trauma and neurocognitive functioning in SPD

The subsample for which the neurocognitive information was available consisted of 60.0% ( $N = 33$ ) females and 40.0% ( $N = 22$ ) males; the mean age was 41.8 ( $SD = 10.5$ ), which is slightly older than the mean age of the total sample. All mean raw cognition scores split by severe trauma or no-/non-severe childhood trauma are shown in Table 4. SPD individuals who reported experiences of severe trauma performed significantly worse on all (but one) measure of working memory ( $P$  values  $< 0.001$ – $0.009$ ), verbal learning and memory ( $P < 0.001$ ), visual learning and memory ( $P < 0.001$ ), and verbal fluency ( $P = 0.002$ ), than individuals with SPD who reported no/non-severe childhood trauma. No significant between-group difference was found for processing speed. The standardized  $z$ -scores across cognitive domains for SPD (severe trauma and no-/non-severe trauma) relative to the HC group from this study are presented in Fig. 2.

#### Discussion

The results of the present study demonstrate that all types of self-reported childhood abuse and neglect were higher in SPD compared to the healthy control group. The types of trauma more

strongly associated with SPD were sexual abuse and emotional abuse, especially for severe forms, suggestive of a dose–response relationship. This is consistent with previous reports, implying that childhood trauma may increase vulnerability for the disorders along the schizophrenia spectrum (schizotypy (55), SPD (11, 56), and psychotic disorders (14, 57)). Previous research also observed that it is especially more frequent/chronic types of abuse that predict both non-clinical (9) and clinical psychotic symptoms (15).

The prevalence of childhood abuse and neglect for HC and SPD was consistent with reports from USA representative population-based studies (11). In addition, previous findings suggest that a large proportion of individuals with a personality disorder (PD) report exposure to abuse (73.0%) (58) and neglect (82.0%) (58) when growing up, similar to current findings. Also in parallel with our study, the most prevalent types of trauma among a PD sample are emotional abuse and emotional neglect (59).

The findings suggest that self-reported severe childhood trauma is associated with more severe clinical profiles in SPD. In particular, individuals with SPD who reported experiences of severe sexual abuse also reported higher levels of cognitive–perceptual symptoms/traits (ideas of reference, odd beliefs or magical thinking, unusual perceptual experiences, and suspiciousness subscales) compared to those without these childhood experiences. In addition, physical abuse, sexual abuse, and, in particular, emotional neglect were

Table 3. Associations between severe types of childhood trauma and cognitive–perceptual (Min 0, Max 33), interpersonal (Min 0, Max 33), and disorganized (Min 0, Max 16) domains in the SPD group

	Cognitive–perceptual			Interpersonal			Disorganized		
	Mean	SE		Mean	SE		Mean	SE	
No- or non-severe trauma	13.88	0.75		15.61	1.45		7.21	0.44	
Severe trauma	15.32	0.68		18.18	0.72		7.39	0.39	
	$\beta^*$	95% CI	$P$ -value	$\beta^*$	95% CI	$P$ -value	$\beta^*$	95% CI	$P$ -value
Any trauma									
Severe	1.38	−0.76–3.52	0.204	2.94	−0.17–6.04	0.064	0.16	−1.08–1.40	0.797
Distinct trauma types									
Emotional abuse									
Severe	1.39	−0.91–3.70	0.236	1.45	−1.93–4.82	0.399	0.01	−1.33–1.37	0.982
Physical abuse									
Severe	1.67	−0.78–4.16	0.180	3.38	−0.22–6.98	0.065	0.81	−0.63–2.24	0.268
Sexual abuse									
Severe	2.30	−0.21–4.82	0.073	3.16	−0.52–6.84	0.092	0.46	−1.00–1.93	0.533
Emotional neglect									
Severe	1.22	−1.22–3.66	0.327	<b>3.82</b>	0.29–7.36	<b>0.034</b>	0.32	−1.09–1.74	0.652
Physical neglect									
Severe	0.10	−2.70–2.89	0.948	1.79	−2.29–5.87	0.388	−1.58	−3.18–0.03	0.054

Significant results are presented in bold.

$\beta$ , beta coefficient; SE, standard error.

\*Adjusted for age, gender, race, and years of education.

## Severe childhood trauma and SPD

Table 4. Neurocognitive domains and measures (mean scores + standard errors) for the SPD group ( $N = 32$ ) across severe childhood trauma types

Cognitive domains	No- or non-severe trauma $N = 16$			Severe trauma $N = 16$			Group difference		
	Mean	SE	95% CI	Mean	SE	95% CI	ES Cohen's $d$	$F$	$P$ -value
Processing speed									
Trail Making Test A [time in s]	34.2	2.8	28.5–39.0	36.2	4.2	27.7–44.7	0.14*	0.48	0.494
BASC Symbol Coding [total correct]	40.6	2.8	34.9–46.3	40.6	1.7	37.1–44.1	0	1.31	0.264
Working memory									
WMS Spatial span, Forward [total score] Min 0, Max 16	8.0	0.3	7.3–8.7	6.6	0.7	5.1–8.0	0.66	<b>8.31</b>	<b>0.009</b>
WMS Spatial span, Backward [total score] Min 0, Max 16	6.7	0.4	6.0–7.5	5.2	0.5	4.3–6.2	0.90	<b>24.78</b>	<b>&lt;0.001</b>
Letter number span [total correct] Min 0, Max 24	16.0	0.7	14.6–17.4	10.4	1.2	8.0–12.8	1.45	<b>31.89</b>	<b>&lt;0.001</b>
Letter number span [longest span] Min 0, Max 7	5.8	0.2	5.5–6.3	4.5	0.3	3.9–5.1	1.27	<b>35.88</b>	<b>&lt;0.001</b>
PASAT [total score] Min 0, Max 49	38.1	2.7	32.5–43.7	25.9	4.0	17.7–34.1	0.88	<b>23.46</b>	<b>&lt;0.001</b>
DOT [distance error]	1.2	0.2	0.8–1.6	2.1	0.2	1.6–2.6	0.84*	6.06	0.021
Verbal learning and memory									
HVLT [total correct] Min 0, Max 36	25.6	1.3	23.0–28.2	22.6	1.1	20.4–24.9	0.63	<b>20.67</b>	<b>&lt;0.001</b>
Visual learning and memory									
BVMT [total correct] Min 0, Max 36	21.6	1.5	18.5–24.7	14.5	1.5	11.5–14.5	1.20	<b>89.84</b>	<b>&lt;0.001</b>
Verbal fluency									
Category fluency [total correct]	21.1	1.1	18.9–23.3	18.2	0.7	16.8–19.7	0.79	<b>12.47</b>	<b>0.002</b>
Reasoning and problem solving/executive functioning									
NAB Mazes [total score] Min 0, Max 26	17.6	1.1	15.2–20.0	8.4	1.6	5.0–11.7	1.62	<b>60.93</b>	<b>&lt;0.001</b>

SE, standard error; BASC, Brief Assessment of Cognition in Schizophrenia; WMS, Wechsler Memory Scale-III; NAB, Neuropsychological Assessment Battery; PASAT, Paced Auditory Serial Addition Test; HVLT, Hopkins Verbal Learning Test—Revised (HVLT-R); BVMT, Brief Visuospatial Memory Test; ES, effect size. Min and Max scores refer to a range of possible scores (if not provided, Min = 0, Max = unlimited).

Significant results are presented in bold.

\*For ease of interpretation, the direction of ES has been changed. Higher scores on these measures represent better (not worse) functioning, and ES favors no- or non-severe trauma group.

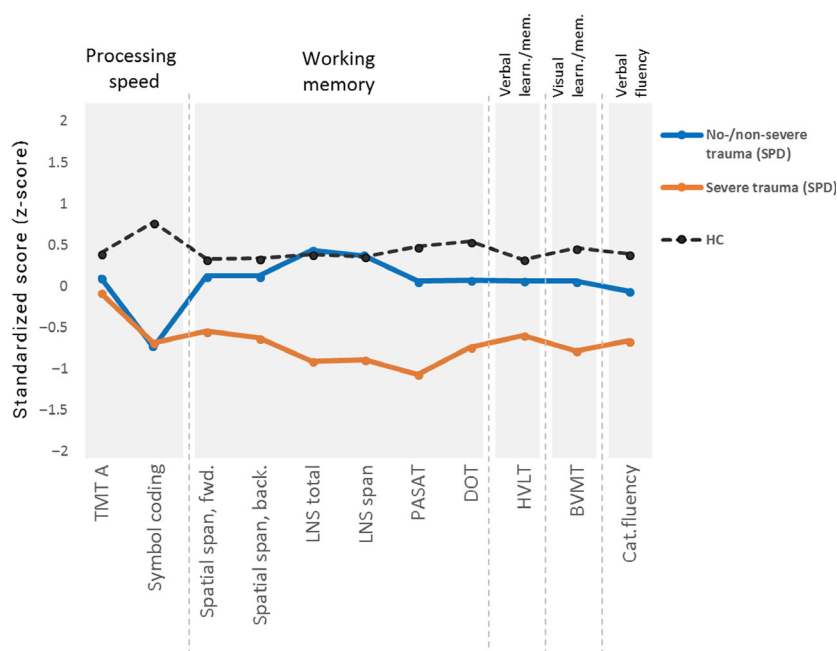


Fig. 2. Standardized z-scores for SPD ( $N = 32$ ) divided by Severe trauma ( $N = 16$ ) and no-/non-severe trauma ( $N = 16$ ), and HC ( $N = 23$ ). Due to a small sample size, HC sample could not be divided by severe trauma reports vs. no-/non-severe trauma. No data were available for reasoning and problem solving/executive functioning domain for a healthy control group. [Colour figure can be viewed at wileyonlinelibrary.com]



associated with more pronounced interpersonal symptoms/traits in SPD (excessive social anxiety, no close friends, constricted affect, and suspiciousness subscales). This parallels findings from the schizophrenia literature, suggesting that childhood trauma is associated with more severe positive symptomatology (hallucinations and delusions) in individuals with psychotic disorders (57). Moreover, the associations between childhood trauma and positive (9) and interpersonal (17) symptoms/traits have been consistently reported for adults in the general population with high schizotypy scores, as has the association between trauma and the disorganized schizotypy domain (13). No such associations were found for the disorganized domain in this study, similar to some previous observations (60). However, this might also be explained by the smaller range of scores for this domain, characterized by two items only.

It should be noted that the cross-sectional and correlational nature of our study precludes any inference of causality from our findings. This limitation is shared by most studies studying the effect of childhood trauma on adult outcomes. Studies with extremely long follow-up periods are required to study the consequences of prospectively assessed childhood trauma. Literature proposes various theoretical models on mechanisms linking childhood trauma and schizophrenia-spectrum symptomatology. The affective pathway (61) to psychosis postulates that childhood trauma elicits negative emotions (anxiety, depression) (62) and negative beliefs about self and others (63), predisposing feelings of threat and suspiciousness (64). Therefore, it may not be surprising that emotional abuse (at any severity) showed the strongest associations with SPD in our study, as these types of abuse in particular have been linked to feelings of degradation, humiliation, and defeat (65, 66). Certain schizotypal traits like paranormal beliefs might even be the results of individual attempting to alleviate powerlessness and hopelessness (67).

Positive symptoms on the other hand have previously been explained as the effect of underlying biological vulnerability caused by early trauma (68) through dysregulation of hypothalamic–pituitary–adrenal axis (HPA) (69) which plays a crucial role in hormonal responses to stress. Additionally, some studies proposed positive symptoms as an object of intrusive memories of traumatic events (70). Research also shows that individuals scoring high on schizotypy might suffer from trauma-related intrusions (71), possibly resulting in ‘source monitoring’ errors (i.e., difficulties distinguishing between internally sourced events and those

created externally) (72). Although this could raise concerns about the validity of their trauma reporting, a comparison between low and high schizotypy groups found no difference in frequencies of deliberately retrieved memories (73). Furthermore, those with elevated schizotypal traits even displayed improved capacity to imagine the autobiographical past (i.e., mental time travel) (74). Some observed that sexual abuse in particular leads to impaired source monitoring processing (75). This might help to explain why sexual abuse in our study showed the strongest magnitude of association with higher levels of cognitive–perceptual SPD traits. Others have suggested that this type of childhood abuse may lead to disruption of ‘internal anchors’ (e.g., sense of being connected to one’s body, sense of self/identity), possibly as a result of dissociative detachment (76, 77), further impacting mechanisms underlying psychosis formation. Furthermore, sexual abuse has also been linked to paranoia and suspiciousness in non-clinical samples with high schizotypal traits (7).

Childhood traumatic experiences can also contribute to the appraisals of psychological, perceptual, or bodily experiences as being more powerful (78) or outside the individual’s control (79). Given the autobiographical nature of cognitive schemas, reflecting individual’s past and current experiences, this is not unexpected. The associations between childhood trauma and interpersonal (negative) symptoms/traits are, however, more difficult to understand. It is largely believed that the interpersonal/negative symptom domain is underpinned by biological factors, linked to genetic risk of psychosis (80) and alterations in brain development (81). Some researchers have hypothesized that symptoms such as reduced responsiveness and emotional numbing only mimic negative symptoms but are actually reactions to the childhood traumatic experiences (6). Emotional neglect in our study showed the strongest magnitude of association with interpersonal SPD traits, possibly the indirect result of early developed fear and avoidance of social situations following trauma (82) also mediated by poor early attachment (83–85). However, this might not be specific to emotional neglect and associations with interpersonal SPD traits (although reduced) were observed across trauma types. Because childhood trauma occurs during a critical time for the development of social functioning skills, traumatic experiences can disrupt the development of these skills, resulting in lifelong difficulties in interpersonal functioning. For example, childhood trauma can disrupt the normative development of self-awareness, social cognitive skills (86), and the ability to develop secure

attachments to others (87), which have been linked to the development of negative symptoms (88). Consistent with this notion, in longitudinal studies of early psychosis, childhood trauma predicts social functioning difficulties in adulthood (89).

According to the 'continuum of psychosis' model (90, 91), clinical phenotypes have some degree of continuity below the clinical threshold and schizotypal- or psychosis-like experiences are observed in the general population. The expression of subclinical and clinical schizotypal traits is influenced by both genetic/familial (92) and non-genetic factors (69). Yet, it might be that severe and repeated environmental stressors (interacting with genetic risk) influence the level of severity and persistence of symptoms/traits and predispose an individual to transition to clinical symptomatology, via psychological and biological sensitization (93). Although this hypothesis has not yet been examined in a sufficiently well-powered sample, the current evidence suggests that both polygenic risk for schizophrenia/schizotypy and childhood adversity increase risk for psychosis *independently* from each other, without evidence of an interaction. This is consistent with a multifactorial threshold model. Moreover, it appears that polygenic risk for psychosis does not increase the likelihood of exposure to or reporting of childhood trauma (94). Studies showed that shifts from non-clinical to clinical outcomes are associated with the number and severity of symptoms (95, 96), not only positive symptoms (most consistently associated with childhood trauma) but also negative and disorganized symptoms (97).

The current study addresses the gap in the literature concerning childhood trauma and neurocognitive functioning in SPD, although given the sample size, these findings need to be interpreted as preliminary. Individuals who reported severe traumatic experiences in their childhood showed worse performance on all (but one) cognitive domains measured. Specifically, severe childhood trauma was associated with impairments in working memory, visual and verbal learning and memory, and verbal fluency, compared with individuals with SPD who did not report childhood trauma. In fact, the profiles of individuals with SPD who did not report childhood trauma were similar to those observed in the healthy control group. These findings are consistent with studies using full-blown psychosis samples where worse neurocognitive performance was observed in individuals who reported childhood trauma (compared to those who did not report it) (28, 98). Although no such effect was found for processing speed in the current

study, this domain was impaired across the SPD sample to a similar degree regardless of the presence of reported childhood trauma. This is in contrast with findings by Lysaker et al. (29), showing that individuals with schizophrenia who also reported sexual abuse displayed impairments in processing speed compared to those without such trauma. This could also imply that there are differential effects of trauma types on neurocognition, which could not be explored in this study. Nevertheless, processing speed has been proposed as a core cognitive impairment in individuals with schizophrenia (99, 100) and high schizotypy (101), so perhaps it is not surprising that the deficits are evident across the SPD population and could not be explained by childhood traumatic experiences alone. On the other hand, it is possible that group differences between high and low/no trauma reporters were not observed due to a floor effect in TMT-A scoring (time to complete the task). Also worth noting, the deficit in processing speed was observed during symbol coding assessment much more than during the TMT-A.

Several hypotheses have been suggested in trying to understand the mechanisms supporting the relationship between deficits observed in neurocognitive functioning of individuals with childhood trauma among psychosis patients. The aforementioned traumatogenic neurodevelopmental (TN) model (32) assumes the critical impact of HPA axis dysregulation (33), dopamine irregularities (102), and structural brain abnormalities (103), leading to vulnerability/oversensitivity to stresses later in life (104). This is not surprising since early childhood is a period of immense neuroplasticity of the brain and the traumatic experiences can cause the disruption in neuronal growth (105). But it could also be that individuals with SPD might be more susceptible to the effects of childhood trauma due to increased levels of stress biomarkers (106). On the other hand, children with cognitive impairments may be at greater risk of being maltreated compared to high functioning children, implying reverse causality. Moreover, one could argue that it is deficits in working memory (as observed in our study) that predict higher childhood trauma reports. Although the working memory capacity has been cited as necessary component for autobiographical recall (107), there is little reason to believe that childhood trauma reports would be inflated in individuals with working memory deficits.

More research is required to unravel the mechanisms underlying the associations between childhood trauma and cognitive functioning in SPD. Also, there is emerging evidence suggesting

differential illness trajectories to schizophrenia-spectrum disorders. For example, one study observed that individuals with first-episode psychosis who reported childhood trauma had an average premorbid IQ and showed significant cognitive decline whereas individuals without childhood trauma had lower IQ from the onset and did not show a significant change after the illness (98). Moreover, comorbidity among childhood adversities has been widely cited (108) and observed in our study as well, possibly implying that the differences in trauma types are due to particular trauma clustering (109) and not purely a distinct effect of certain traumas. More exploration is needed to unravel whether it is the particular traumatic event or rather a combination of traumas that has the highest impact on symptoms and neurocognition in SPD population.

#### Limitations

The findings of the study need to be considered in light of several limitations. First, the Childhood Trauma Questionnaire is a self-report measure, fully based on retrospective recall. Several reporting biases have been proposed, including childhood amnesia (110), forgetting (111), mood-congruent recall (112), and reconstructive nature of memory (113). Also, a recent study by Newbury et al. (114) compared retrospective reports and prospective observations of childhood trauma and found the agreement between two methods was only slight to fair. Nevertheless, both methods of childhood trauma assessment (prospective and retrospective) showed an equal strength of association between trauma and adult psychopathology (115); and retrospective reporting has been consistently shown to be reliable over time, including in clinical groups (116). The frequency of traumatic experiences in the present study was also consistent with previous general population studies and PD samples in the United States (11, 58). Also, the profiles/distribution of childhood trauma experiences reported by SPD were similar to those of healthy controls (e.g., the highest frequencies for both were for emotional neglect and emotional abuse). Furthermore, even healthy adults who report childhood trauma experience an increase in subclinical symptoms/traits of personality disorders (117).

An additional limitation is that neurocognitive assessments were only completed for a subset of our sample (limiting sufficient power on particular measures, see Supplemental Material). Also, a larger sample of individuals with SPD would be needed to investigate the relationship between

childhood trauma types and neurocognitive functioning and explore the potential sex differences, as evidence suggests that stress impacts cognition differently in males and females (118). This was also reported by Aas et al. (98) using a sample of first-episode psychosis participants, where males were especially vulnerable to the effects of trauma on cognition. Third, we had no information available on parental psychopathology. Parental history of mental illness not only stands as a proxy for genetic risk for the disorder but can also partly reflect individual's early environment (119). Fourth, a cross-sectional study design does not allow any conclusions about directionality of the associations between childhood trauma types and increased schizotypal personality disorder traits. Causality is compromised by the assumption that childhood trauma preceded the development of SPD. But, it is possible that individuals who exhibit particular schizotypal symptoms/traits (e.g., odd behaviour, social anxiety) (120) might be more at risk of being maltreated compared to those without such traits (reverse causality). It can also be hypothesized that individuals with SPD either grew up in circumstances where childhood maltreatment was more likely to occur or have different interpretation of such traumatic experiences. For example, childhood trauma could be a proxy for poor parenting, low socio-economic status, or family dysfunction among others (121). Nevertheless, this study observed a dose-response association between trauma severity and SPD along with differential effects of childhood trauma types on SPD symptoms/traits, which would be more difficult to explain if the SPD symptomatology preceded early adversity. Fifth, we did not have any available data to explore how the severity of SPD symptomatology relates to everyday functioning. More research is therefore needed to be able to identify the effective ways to improve the real-world outcomes for this population.

#### Conclusion

The results of this study indicate that all types of reported childhood trauma are associated with a SPD diagnosis, in a dose-response fashion. Additionally, some severe types of childhood trauma predicted higher cognitive-perceptual and interpersonal symptoms/traits among the SPD group. Individuals with SPD who reported severe trauma also showed worse overall cognitive performance compared to individuals who did not report such experiences. Further understanding of the mechanisms underlying these associations



is required to allow more tailored intervention approaches.

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### Declaration of interest

None.

### Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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## Supporting Information

Additional Supporting Information may be found in the online version of this article:

**Figure S1.** Standardized z-scores for SPD ( $N = 32$ ) divided by No-/Non-severe trauma ( $N = 16$ ), 1 Type of severe trauma ( $N = 10$ ) and 2 or more types of severe trauma ( $N = 6$ ); and HC ( $N = 23$ ).

**Table S1.** Overall model statistics – For logistic regressions

**Table S2.** Overall model statistics – For linear regressions.