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The effects of developmental trauma on theory of mind and its relationship to psychotic experiences: A behavioural study

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ABSTRACT

Background: Developmental psychological trauma induces vulnerability to psychosis. However, the mechanisms underlying this association are poorly understood. Impairments in Theory of Mind (ToM) have been observed in adult survivors of developmental trauma and individuals with psychosis. ToM is therefore a candidate mechanism underlying the association between developmental trauma and psychosis.

Methods: We used a computerised version of the Director task - where a participant is instructed by a confederate to move an object around a 4 × 4 grid, whilst taking account of whether these objects are visible to a confederate who instructs the participant - to investigate impairments in ToM in 209 participants (age: $M = 37.8$, SD=13.6; 56% female). Participants were divided into a) developmental trauma-positive (DT+) and control groups (DT-) based on their history of developmental trauma and b) then further into subclinical (S) and healthy groups (H) as based on psychotic experiences indexed by the CAPE-P15. After exclusion, the numbers in each group were: DT+*H* (47), DT+*S* (84), DT-H (54), DT-S (12). (Total: 197).

Results: Developmental trauma exposure was associated with psychotic experiences (OR: 7.89, $p < .001$), which remained significant after controlling for demographic and clinical confounds (adjusted $R^2 = 0.452$, R^2 change = 0.0184, $p = .009$). Participants with developmental trauma ($F_{1,\ 194}$) = 5.46, $p = .020$, $\eta p^2 = 0.027$) and participants more prone to psychotic experiences ($F_{1,\ 194}$) = 4.71, $p = .031$, $\eta p^2 = 0.024$) demonstrated significantly lower accuracy on the Director task relative to their respective control, after controlling for the effects of age.

Conclusions: ToM deficits are associated with self-reported developmental trauma and psychotic experiences. Further work is needed to explore these relationships further and whether they represent generalised or specific effect effects on developmental trauma and psychopathological domains.

Abbreviation:
ToM, Theory of Mind.

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1. Introduction

Psychologically traumatic experiences during childhood and adolescence, hereon referred to as developmental trauma, have been extensively recognized as a risk factor of adult psychopathology including psychosis (Schäfer and Fisher, 2011; Aas et al., 2014; Varese et al., 2012), with approximately a third of cases of psychosis in adulthood attributable to early adverse experiences (Varese et al., 2012). The role of developmental trauma in worsening the clinical severity and course of psychosis is well-recognised, with a growing body of evidence indicating developmental trauma predicts increased symptom severity, treatment resistance, and hospitalisation rates (Gibson et al., 2016), as well as poorer treatment outcomes (Thomas et al., 2019), in psychotic populations. However, while recent evidence suggests that developmental trauma is associated with cognitive impairment, which in turn precedes the onset of psychosis (Begemann et al., 2016), research exploring the associations between developmental trauma and neurocognition has yielded mixed findings. A recent meta-analysis found evidence of mediating roles of dissociation and emotional dysregulation between developmental trauma and hallucinations (Bloomfield et al., 2021). However, there is yet to be converging evidence on the role of social cognition in the association between developmental trauma and psychotic symptoms. The meta-analysis also highlighted the paucity of experimental research into the underlying neurocognitive mechanisms (Bloomfield et al., 2021). Our limited understanding of the specific neurocognitive mechanisms through which developmental trauma induces vulnerability to adulthood psychosis is concerning, as this significantly limits our current evidence base for trauma-informed care (Bloomfield et al., 2020).

Theory of Mind (ToM) may represent one vulnerability mechanism, and therefore interventional target, for adult survivors of developmental trauma. ToM is part of the process of mentalization (Nijhof et al., 2016) and broadly refers to the ones ability to attribute knowledge, emotions, thoughts, and beliefs to oneself and others (Premack and Woodruff, 1978; Sabbagh, 2004). This allows us to predict how the behaviours, intentions, and desires of others may differ from our own (Baron-Cohen, 1997) and, in doing so, respond with appropriate, relevant social actions. ToM is, therefore, a crucial domain of social cognition (Bell et al., 2017; Wimmer and Perner, 1983). Examples of ToM in action, may be receiving a pass on a football field (Santiesteban et al., 2015) or in identifying an individual's emotional state based on their facial expression.

Developmental trauma has been associated with schizotypy (Velikonja et al., 2014; Sheinbaum et al., 2014), both in psychiatrically healthy people, patients with (psychotic) bipolar disorder or schizophrenia/schizoaffective disorder (Quidé et al., 2018). Additionally, this same group reported that the severity of childhood trauma exposure was associated with aberrant patterns of activation in key regions for ToM in patients with schizophrenia, while performing an affective ToM task (Quidé et al., 2017). This association between developmental trauma and impairments in ToM (Quidé et al., 2017; Benarous et al., 2015) has been shown to alter brain areas involved in the neural processing of ToM and perspective-taking during adulthood (Cracco et al., 2020). Moreover, schizotypy and childhood trauma are independently associated with changes of grey matter in brain regions critical for cognition and social cognition (Quidé et al., 2021). Since childhood and adolescence represent critical periods for brain maturation (Goddings et al., 2014; Bloomfield et al., 2019), involving processes such as myelination and synaptogenesis (Miller et al., 2012) developmental trauma can lead to the alterations seen in brain structure and function (Teicher et al., 2016). Therefore, it is likely that impairments in ToM arise secondary to developmental-trauma-induced alterations to ToM- localised regions (Quidé et al., 2020; Sayar-Akalsan et al., 2021; Raucher-Chéné et al., 2020; Maat et al., 2015).

Such regions are collectively referred to as the 'mentalising network' and this has been well characterised in neuropsychological and neuroimaging research (including regions such as the medial prefrontal cortex (mPFC), the anterior (ACC) and posterior cingulate cortex (PCC), the bilateral temporoparietal junction (lTPJ and rTPJ) and the superior temporal sulcus (STS) (Mar, 2011)). Indeed, there is evidence of altered connectivity between key regions in the mentalization network in survivors of developmental trauma (Teicher et al., 2016). Importantly, beyond the neurocognitive effects of developmental trauma, the psychological experience of developmental trauma can have the potential to result in deficits in mentalization capacity at imaginative levels of social cognitive understanding (Fonagy & Allison, 2011; Luyten, Campbell, Allison and Fonagy, 2020).

In parallel, psychosis has also been associated with impairments in social cognition (Bell et al., 2017), which may be partially accounted for by deficits in ToM (Yang et al., 2017; Zhang et al., 2016; Stanford et al., 2011; Ohmuro et al., 2016). There exists a strong body of evidence in support of alterations in brain structure and function associated with psychosis. There is evidence that the mentalization network is affected in people with psychosis. For example, a neuroimaging meta-analysis found significantly decreased activation in brain regions such as the mPFC, PCC, ACC and insula in schizophrenia patients compared to controls when performing facial emotion recognition and ToM tasks (Jáni and Kašpárek, 2018). Patients with psychosis also display reduced grey matter volume in multiple ToM-related regions, including the prefrontal cortex and cingulate cortex (Read et al., 2014). This suggests that deficits in ToM represent one candidate vulnerability mechanism through which developmental trauma elevates the risk of psychotic disorders (McCrory and Viding, 2015; Teicher et al., 2016).

We therefore sought to investigate the relationship between developmental trauma, ToM and proneness to psychotic experiences by testing the following hypotheses. First, that there is an association between developmental trauma, indexed using the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003), and psychotic experiences measured with the Community Assessment of Psychic Experiences-Positive Scale (CAPE-P15; Capra et al., 2013), after accounting for demographic and clinical variables. Second, that there is an association between developmental trauma and ToM, measured through performance on a computerised version Director Task. Third, that there is an association between psychotic experiences and ToM. Finally, we explored the hypothesis that impaired mentalising ability mediates the relationship between developmental trauma and psychotic experiences.

2. Material and methods

2.1. Ethics

This study was approved by the UCL Research Ethics Committee (reference number: 14,317/001) and was conducted in accordance with the Declaration of Helsinki and Good Clinical Practice.

2.2. Participants

The participants ($n = 209$) were from a non-clinical population and recruited through social media and Facebook advertisements. Participants that were currently taking medication for their mental health were excluded from the study. We did not exclude participants with a history of psychiatric disorders. All participants completed informed consent forms. Participants scoring moderate-severe or severe-extreme in one or more CTQ subscales were allocated to the 'DT+' group; participants scoring less than moderate-severe in all subscales were allocated to the control group.

The participants were also split into two psychotic experiences groups based on their responses on the CAPE-P15. Using the CAPE-P15 cut-off score of 1.47 for ultra-high risk for psychosis status (Bukenaite et al., 2017), participants were divided into 'subclinical levels of psychotic experiences (subclinical) and 'healthy/low levels of psychotic experiences' (healthy). [DT+*H* (47), DT+*S* (84), DT-H (54), DT-S (12)].

2.3. Demographic data

Demographic data on the participant's assigned sex at birth, age, ethnicity, educational attainment, childhood socioeconomic status (SES) (indexed by parental occupation at 18 years of age and prior eligibility for free school meals), past psychiatric medication and tobacco smoking were collected. Participants also completed the Drug Abuse Screening Test (DAST-10; Skinner, 1982) and the Short Michigan Alcoholism Screening Test (SMAST; Selzer et al., 1975).

The Drug Abuse Screening Test (DAST-10; Skinner, 1982) is a self-report measure that investigates problems related to drug misuse over the past 12 months – the total DAST score yields a quantitative index of this. The DAST has long shown diagnostic validity in the assessment of drug disorders – showing 85% overall accuracy in classifying patients according to DSM-III diagnosis. (Gavin et al., 1989).

The Short Michigan Alcoholism Screening Test (SMAST) is a thirteen-question questionnaire that concerns one's involvement with alcohol during the past twelve months - it only takes a few minutes to complete (Selzer et al., 1975). Previous data shows it to be an effective diagnostic instrument (Connor et al., 2007). It is also strongly recommended to be used alongside the DAST-10 unless there is a clear indication that the client uses alcohol but does not use any other drug at all – hence our use of the DAST-10. A score of 0–2 indicates that no problems have been reported and that no further action should be taken at that time; a score of 3 indicates a borderline alcohol problem reported and that further investigation is required; a score of 4 or more indicates that potential alcohol abuse has been reported and that a full assessment is required.

2.4. Developmental trauma

2.4.1. Childhood trauma questionnaire (CTQ)

The Childhood Trauma Questionnaire (CTQ) is a retrospective measure of child abuse and neglect, both of which have been associated with long-term psychological consequences. (Aloba et al., 2020) The original CTQ - developed by Bernstein et al., 1994 - was a 70-item version, which took around 10–15 min to give. Due to its length and the possible time constraints that it may present, a shorter version of the CTQ was developed that would take no longer than 5 min to administer. This version, called the Childhood Trauma Questionnaire Short Form (CTQ-SF), is a 28-item retrospective measure for assessing childhood trauma (Bernstein et al., 2003). It measures childhood trauma in five domains: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. As the psychometric properties of the CTQ-SF have been corroborated in both community and clinical samples (Bernstein et al., 2003), we used the CTQ-SF as a retrospective measure for assessing childhood trauma in our study.

2.5. Psychotic experiences

2.5.1. Community assessment of psychotic experiences (CAPE-P15)

The Community Assessment of Psychic Experiences-Positive Scale (CAPE-P) is a 20- item measure of lifetime psychotic-like experiences, it was developed to measure their lifetime prevalence across the general population. (Steffanis et al., 2002; Konings et al., 2006); It is a self-report questionnaire modified after the Peters et al. Delusions Inventory [PDI 21], (Peters et al., 2004; van Os and Delespaul, 2003) used as a screening-instrument in the detection of individuals at ultra-high risk for psychosis. The original CAPE-P consisted of five subscales: persecutory ideation, bizarre experiences, perceptual abnormalities, magical thinking, and grandiosity. However, a 15-item revision of the scale was made which omitted the latter two subscales due to their low concurrent validity with other indicators of mental health (Capra et al., 2013; Armando et al., 2013; Wigman et al., 2011). The 15-item, self-report scale therefore assesses the frequency of, and distress associated with, lifetime psychotic experiences via the subscales of persecutory ideation, bizarre experiences, and perceptual abnormalities. (Capra et al., 2017). Each item uses a 4-point Likert scale form 0, 'never', through 'sometimes' and 'often', to 3, 'nearly always'. If the participants endorsed a psychotic-like experience (at least 'sometimes'), they were also asked how distressed they were about the experience using a 4- point Likert scale from 0, 'not distressed', through 'a bit distressed' and 'quite distressed', to 3, 'very distressed', which produced a total score of 0–45 (Capra et al., 2017). Ultra-high risk for psychosis status was assessed using a pre-defined cut-off score of 1.47. (Knight et al., 2017). Higher scores are indicative of a higher frequency of, and distress associated with, psychotic experiences. The 1.47 is a weighted, mean score based upon a 4 point Likert scale for the CAPE positive items.

2.5.2. Oxford-liverpool inventory of feelings and experiences short version (sO-LIFE)

The Oxford-Liverpool Inventory of Feeling and Experiences short version (sO-LIFE) is a scale for assessing schizotypal traits. This 43-item scale consists of four sub-scales: unusual experiences, cognitive disorganisation, introvertive anhedonia and impulsive non-conformity (Mason et al., 2005). In the context of our study, the sO-LIFE was used to assess proneness to psychotic experiences. In previous studies, the sO-LIFE has demonstrated good psychometric validity and internal consistency in non-clinical populations (Fonseca-Pedrero et al., 2015, Lin et al., 2013), as well as adequate reliability and convergent validity (Foneseca-Pedrero et al., 2015, Mason et al., 2005).

2.6. Director task

We implemented the computerised Director Task, developed by Dumontheil and colleagues (2010), on the Gorilla Experiment Builder (Anwyl-Irvine et al., 2018) to assess mentalising ability. Each visual stimulus involved a 4 × 4 array of slots, with each slot containing a single miscellaneous object e.g., a large jar. In the 'director condition', participants were instructed by a figure (i.e., the 'director') to move objects from one slot to another. Importantly, although all objects are visible to the participant, some are occluded to the director. The participant was therefore required to consider the limits of the director's perspective i.e., what objects were occluded to the director and what objects were not. In the no-director condition, the participant heard audio instructions to move one of the objects from one slot to another in the absence of a director. In this condition, participants were required to instead account for the colour of the slot, wherein grey slots represented those occluded to the director.

The study used two within-group variables, these being condition (director or no-director) and trial type (experimental, control or filler), replicating the original experimental design by Dumontheil and colleagues (2010). In both the director and no-director conditions, each participant completed 8 control, 8 experimental, and 32 filler trials with trial order counterbalanced between subjects. In the filler trials, the objects that the participants were asked to move were placed in slots that were not occluded. These filler trials were discarded from analyses. In both control and experimental trials, objects were arranged identically (Fig. 1). In the experimental trials, a similar object to that instructed by the director was placed on the slots as a distractor – this was replaced with an irrelevant object in the control trials. This ensured the participants not only inhibited prepotent responses (i.e., moving the object solely based on whether it fits director's instructions), but also fulfilled the task's demands on executive functions, such as working memory (Dumontheil et al., 2010).

We used accuracy and reaction time to assess performance on the director task and therefore ToM ability. We calculated accuracy as the number of correctly completed experimental trials divided by the total number of experimental trials. This was the same for the control trials and this was extended across both director and no-director conditions. We used the average reaction time for both control and experimental trial types in both director and no-director conditions. These measures of

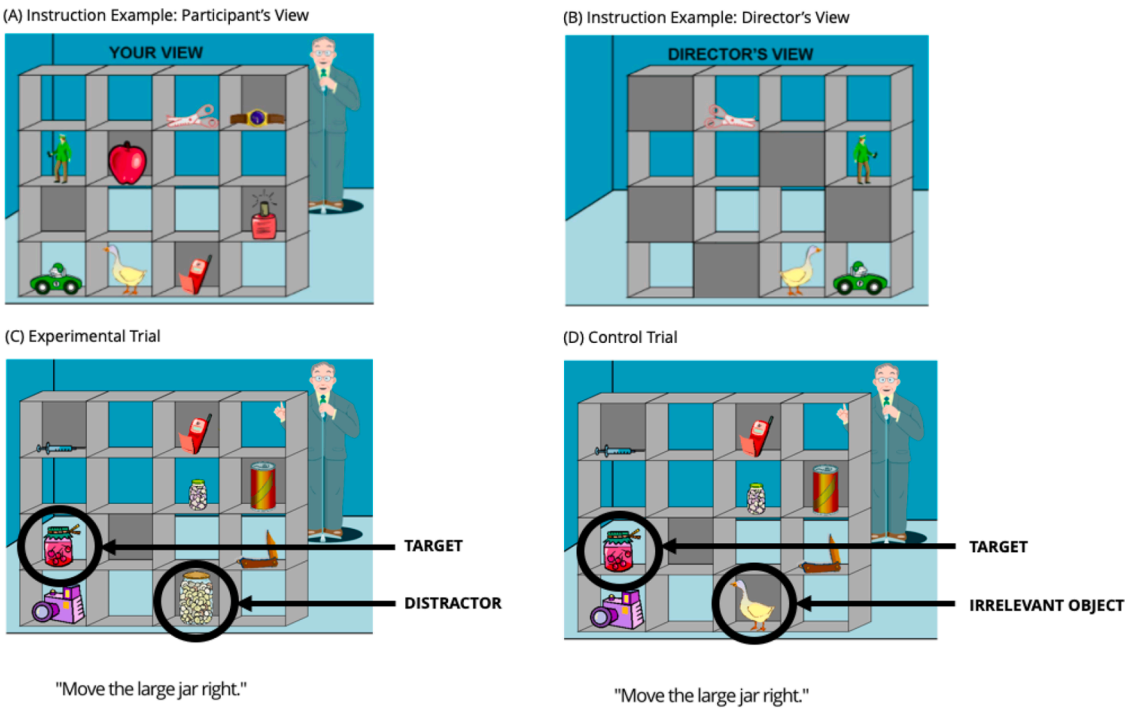

Fig. 1. Computerized Director Task. The stimuli were replicated from Dumontheil et al. (2010) on the Gorilla platform. (A) and (B) depict stimuli presented to participants on instruction screens. The former (A) shows their view, the latter (B) indicates that of the director with four occluded slots. (C) and (D) depict stimuli presented to participants as part of the Control Trial and the Experimental Trial coupled with audio and written instructions. In the Experimental Trial (D), participants would be expected to move the target object if they take the director's perspective into account and move the distractor object if they do not do so. In the Control Trial (C), there is no distractor object.

task performance have previously been found to be robust in measuring mentalising ability (Pile et al., 2017).

2.7. Mental health

Depressive symptom severity was measured by the 16-item Quick Inventory of Depressive Symptomatology (QIDS-SR, Rush et al., 2003). This scale was developed from the 30-item Inventory of Depressive Symptomatology (IDS), the 30 items include all DSM-IV diagnostic criterion items for major depressive disorder (MDD), as well as commonly associated symptoms, such as anxiety, irritability, and melancholic and atypical symptom features. However, the 16-item Quick Inventory of Depressive Symptomatology Self-Report Version (QIDS-SR_{16}) contains only items from the 30-item scales that assessed DSM-IV criterion diagnostic symptoms (Rush et al., 2003). The scoring system here converts responses to 16 separate items into the nine DSM-IV symptom criterion domains. [1) sad mood; 2) concentration; 3) self-criticism; 4) suicidal ideation; 5) interest; 6) energy/fatigue; 7) sleep disturbance (initial, middle, and late insomnia or hypersomnia); 8) decrease/increase in appetite/weight; and 9) psychomotor agitation/retardation]. The total score ranges from 0 to 27.

The presence and severity of state and trait anxiety were measured using the State-Trait Anxiety Inventory (STAI; Spielberger C., 2010). Its most popular version, Form Y, has 20 items for assessing trait anxiety and 20 for state anxiety and it is used to diagnose anxiety and distinguish it from depressive syndromes. All items are rated on a 4-point Likert scale (from 'almost never' to 'almost always'); for State-Anxiety the scale assesses symptom intensity and for Trait-Anxiety the scale assesses symptom frequency - higher scores indicate greater anxiety. A great deal of evidence has confirmed the construct and concurrent validity of the scale (Spielberger, 1989).

2.8. Procedure

The data collected for this experiment were part of a larger study on developmental trauma and cognition. Participants first completed a series of online questionnaires on the web-based experimental platform Gorilla. Questions include those about demographic information (e.g., age, sex), smoking and alcohol history, current serious medical conditions, usage of mental health services, DAST-10, SMAST, QIDS, CTQ, CAPE-P15 and sO-LIFE. The participants then completed the Director Task, along with other computerized tasks to be reported elsewhere.

2.9. Statistical analysis

ToM performance was quantified as accuracy and reaction time (for correct trials only) on the Director Task. Participants with missing reaction time data, due to a failure to respond in time on a particular trial, were excluded from the relevant analyses but were included in accuracy analyses for maximum power (34 from the Control Director Condition (DC), 33 from the Control Non- Director Condition (NC), 50 from the Experimental Director Condition (DE), 52 from the Experimental Non-Director Condition (NE). Group differences amongst demographic and clinical variables were subject to Shapiro-Wilk's test for normality before examined through Welch's t-tests (two-tailed) and chi- square tests of association, with Cohen's d and Cramer's V measures of effect size respectively. A 3-step hierarchical multiple regression analysis was conducted to examine whether developmental trauma significantly predicted psychotic experiences after adjusting for candidate confounds (demographic variables in step 1 and clinical variables in step 2).

ToM performance was further analysed with a mixed $2 \times 2 \times 2$ ANCOVA, using developmental trauma group (DT+ v.s. DT-) as the between-subjects factor, and Condition (director v.s. no-director) and Trial Type (Experimental v.s. Control) as within-subjects factors, and with age as a covariate. The same mixed ANCOVA was performed, except with psychotic experiences groups as the between-subjects factor.

The mediation effect of ToM performance on the relationship between self-reported developmental trauma and psychotic experiences is explored with bootstrapping mediation analysis recommended by Preacher and Hayes (2004) with psychotic experiences as the dependant variable, CTQ score as the predictor, ToM performance as the mediator. 95% confidence intervals were computed using 10,000 bootstraps.

Task understanding was investigated by examining whether participants were performing at floor in the director–experimental trials (Dumontheil et al., 2010), as participants that were unable to understand the director's perspective would make a similar number of errors in the control trials as correct responses in the experimental trials. Paired t-tests were conducted to compare the percentage of correct responses in experimental trials and the percentage of errors in control trials.

All statistical analyses were conducted using R (R Core Team, 2020). Factorial ANCOVA was analysed using the afex (Singmann, 2018) and emmeans (Lenth, 2018) packages, descriptive statistics using the compareGroups (Subirana et al., 2014) package, regression using the olsrr package (Hebbali, 2020) and mediation using the mediation package (Schoemann et al., 2017). A significance level of 0.05 was used. Bonferroni corrections were used where appropriate.

3. Results

3.1. Demographic and clinical data

209 participants between the ages of 18 and 65 ($M = 37.5$, SD=13.7) were recruited. Data from 12 participants that attempted less than 25% of trials (24 out of 96) in the computerized Director Task were excluded, as they were unable to successfully complete the task. Demographics and scores on clinical measurements are reported by developmental trauma group in Table 1. There were no significant differences of age distribution, sex, ethnicity, tobacco smoking and drug use between developmental trauma exposed and controls. However, educational

Table 1
Demographics, clinical data and ToM performance by developmental trauma group.

	All (n = 197)	Developmental Trauma (DT)	Control (n = 66)	Statistic	p-value	Effect Size
Demographic Data						
Age	37.5 (13.7)	37.8 (13.7)	36.9 (13.7)	0.425	0.672	0.064
Assigned sex:				0.002	1.000	0.003
Female	113 (57.4%)	75 (57.3%)	38 (57.6%)			
Male	84 (42.6%)	56 (42.7%)	28 (42.4%)			
Ethnicity:				2.756	0.249	0.118
White	159 (80.7%)	110 (84.0%)	49 (74.2%)			
Asian	12 (6.09%)	7 (5.34%)	5 (7.58%)			
Other	26 (13.2%)	14 (10.7%)	12 (18.2%)			
Educational Attainment:				12.633	0.001	0.253
Lower	76 (38.6%)	62 (47.3%)	14 (21.2%)			
Higher	121 (61.4%)	69 (52.7%)	52 (78.8%)			
Childhood Socioeconomic Status (SES):				14.566	0.001	0.272
Low	63 (32.0%)	53 (40.5%)	10 (15.2%)			
Intermediate	31 (15.7%)	21 (16.0%)	10 (15.2%)			
High	103 (52.3%)	57 (43.5%)	46 (69.7%)			
Eligibility for free school meals	63 (32.0%)	53 (40.5%)	10 (15.2%)	12.921	0.001	0.256
Tobacco Smokers	73 (37.1%)	54 (41.2%)	19 (28.8%)	2.909	0.121	0.122
Prior access of mental health services	138 (70.1%)	108 (82.4%)	30 (45.5%)	28.620	<0.001	0.381
Past psychiatric medication use	85 (43.1%)	67 (51.1%)	18 (27.3%)	10.196	0.002	0.228
DAST (Drug Abuse Screening Test)	1.04 (1.67)	1.16 (1.91)	0.79 (1.02)	1.787	0.076	0.244
SMAST (Short Michigan Alcohol Screening Test)	1.14 (2.00)	1.41 (2.29)	0.61 (1.07)	3.367	0.001	0.451
Clinical Variables						
CAPE-p15 (Community Assessment of Psychotic Experiences Positive	23.5 (5.98)	25.3 (6.23)	20.0 (3.36)	7.751	<0.001	1.059
Item Scale)	10.6 (3.08)	11.6 (2.99)	8.61 (2.09)	8.294	<0.001	1.178
Persecutory Ideation	9.24 (2.86)	9.82 (3.18)	8.11 (1.55)	5.071	<0.001	0.683
Bizarre Experiences	3.66 (1.19)	3.85 (1.37)	3.30 (0.55)	3.958	<0.001	0.522
Perceptual Abnormalities						
O-LIFE	19.9 (7.41)	22.1 (6.93)	15.5 (6.33)	6.729	<0.001	1.000
Unusual Experiences	3.83 (3.04)	4.59 (3.13)	2.32 (2.22)	5.875	<0.001	0.837
Cognitive Disorganisation	6.07 (3.61)	7.12 (3.26)	3.98 (3.38)	6.226	<0.001	0.945
Introvertive Anhedonia	4.93 (1.40)	5.05 (1.40)	4.70 (1.39)	1.693	0.093	0.255
Impulsive Nonconformity	5.07 (1.93)	5.36 (1.92)	4.48 (1.83)	3.107	0.002	0.465
QIDS (Quick Inventory of Depressive Symptomatology)	10.9 (6.37)	13.0 (6.02)	6.70 (4.84)	7.887	<0.001	1.148
STAI-S (State-Trait Anxiety Inventory – State)	43.9 (14.0)	48.1 (13.3)	35.7 (11.7)	6.712	<0.001	0.992
STAI-T ((State-Trait Anxiety Inventory – Trait)	51.0 (14.0)	55.8 (12.1)	41.7 (12.7)	7.480	<0.001	1.139
CTQ (Childhood Trauma Questionnaire)	54.1 (22.3)	65.4 (18.8)	31.7 (5.04)	19.226	<0.001	2.453
ToM (Theory of Mind) Performance						
DC Accuracy Director Control Accuracy)	0.54 (0.34)	0.52 (0.35)	0.58 (0.31)	−1.135	0.258	−0.169
NC Accuracy (No Director Control Accuracy)	0.67 (0.36)	0.64 (0.38)	0.74 (0.33)	−1.852	0.066	−0.273
DE Accuracy (Director Experiment Accuracy)	0.29 (0.29)	0.25 (0.27)	0.38 (0.30)	−2.918	0.004	−0.450
NE Accuracy (No Director Experiment Accuracy)	0.43 (0.39)	0.39 (0.38)	0.51 (0.39)	−2.122	0.036	−0.321
DC RT (Director Control Reaction Time)	2816 (539)	2845 (551)	2763 (515)	0.958	0.340	0.155
NC RT (No Director Control Reaction Time)	2691 (502)	2697 (547)	2679 (410)	0.236	0.814	0.037
DE RT (Director Experiment Reaction Time)	2711 (683)	2662 (743)	2794 (567)	−1.210	0.228	−0.199
NE RT (No Director Experiment Reaction Time)	2740 (579)	2777 (591)	2676 (557)	1.026	0.307	0.176

attainment, childhood SES, access to psychological therapies, prior psychiatric medication and alcohol use was significantly higher for the developmental trauma group. The developmental trauma exposed group showed significantly higher levels of depression, state and trait anxiety than controls.

3.2. Relation between developmental trauma and psychotic experiences

Participants with a history of developmental trauma reported significantly higher CAPE and O-LIFE scores than controls (Table 1). Developmental trauma exposure was associated with an increased ultra-high risk for psychosis (DT+: 84 (64.1%), Control: 12 (18.2%), OR: 7.89 [CI: 3.94 - 16.9], $p < .001$). The association between developmental trauma as measured by the CTQ and psychotic experiences as measured by the CAPE-P15 remained significant (adjusted R2 = 0.487, R2 change = 0.041, F change(13,195) = 16.205, $p < .001$) after adjusting for demographic variables (age, sex, ethnicity, educational attainment, childhood SES, smoking, prior access to mental health services, prior psychiatric medication), and clinical variables (DAST-10, SMAST, QIDS, STAI). The details of the regression model and results are presented in Supplementary Table 2.

3.3. Task understanding

The percentage of correct responses in experimental trials ($M = 0.29$, SD = 0.29) significantly differed from the percentage of errors in control trials ($M = 0.46$, SD = 0.34; $t_{196} = 4.34$, $p < .001$, $d = 0.31$). The results suggest that the participants were able to take the director's perspective into consideration when completing the experimental trials.

3.4. Relation between developmental trauma and task performance

Performance in the Director Task in terms of accuracy and reaction time is reported in Table 1. Reaction in the experimental trials did not differ between developmental trauma groups (director: $t_{136} = -1.210$, $p = .228$; no-director: $t_{114} = 1.026$, $p = .307$), hence subsequent analyses regarding performance in the Director Task performance focused on accuracy.

The 2 × 2 × 2 ANCOVA (Fig. 2) revealed a significant main effect of developmental trauma group ($F_{1, 194} = 5.46$, $p = .020$, $\eta p^2 = 0.027$), with the DT+ group performing significantly worse in the Director Task. Both groups performed better in control trials than in experimental trials ($F_{1, 194} = 129.23$, $p < .001$, $\eta p^2 = 0.068$), and in the no-director condition than in the director condition ($F_{1, 194} = 73.91$, $p < .001$, $\eta p^2 = 0.059$), which is visualized in Fig. 2. Age as the mean- centred covariate was also a significant predictor of accuracy ($F_{1, 194} = 10.34$, $p = .002$, $\eta p^2 = 0.05$. There were no significant interactions between factors (developmental trauma group, condition, trial type, age).

3.5. Relation between psychotic experiences and task performance

The 2 × 2 × 2 ANCOVA (Fig. 3) revealed a significant main effect of psychotic experiences group ($F_{1, 194} = 4.71$, $p = .031$, $\eta p^2 = 0.024$), with the subclinical group performing significantly worse than the healthy control group. Again, both groups performed better in control trials than in experimental trials ($F_{1, 194} = 155.36$, $p < .001$, $\eta p^2 = 0.071$), and in the no-director condition than in the director condition ($F_{1, 194} = 79.12$, $p < .001$, $\eta p^2 = 0.059$), visualized in Fig. 3. Age as the mean-centred covariate was also a significant predictor of accuracy ($F_{1, 194} = 11.66$, $p < .001$, $\eta p^2 = 0.057$). There were no significant interactions between factors (psychotic experiences group, condition, trial type, age).

3.6. Mediating effect of ToM on the relation between developmental trauma and psychotic experiences

The effect of developmental trauma on psychotic experiences was not significantly mediated by ToM performance (Fig. 4). The regression coefficient between CTQ score and ToM performance was significant (estimate = −0.0028, $p = .0022$). The regression coefficient between ToM performance and CAPE-P15 score (estimate = −0.8221, $p = .5377$) was not significant. The indirect effect was insignificant (estimate = 0.0023 [bootstrapped 95% CI: −0.0032- 0.0096]) The total effect (estimate = 0.1310, $p < .001$) and direct effect (estimate = 0.1333, $p < .001$) was significant.

Post-hoc Monte Carlo simulation based statistical power analysis show that the mediation analysis has adequate power. The significance of the mediation effect is evaluated using the percentile bootstrap confidence interval with 1000 bootstraps and 1000 replications as implemented in the R package *bmem* (Zhang, 2014). The power to detect the mediation effect of ToM performance on the relationship between CTQ and CAPE scores with the current sample ($n = 197$) is about 73.9%. The power analysis suggests that future replications of the current study with a sample size of at least 275 will reach 81.3% power to detect the hypothesized mediation effect.

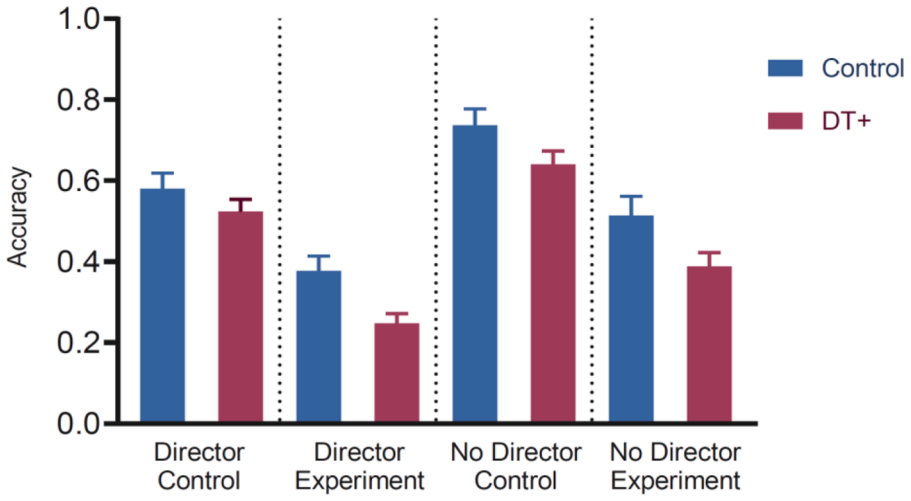

Fig. 2. Director task accuracy across trial types between developmental trauma groups. Mean accuracy in the Director Task was significantly lower for the developmental trauma exposed group than controls, including after controlling for age ($F_{1, 194} = 5.46$, $p = .020$, $\eta p^2 = 0.027$). Both groups performed worse in experimental trials and the director condition. Error bars are the standard error of the mean (SEM).

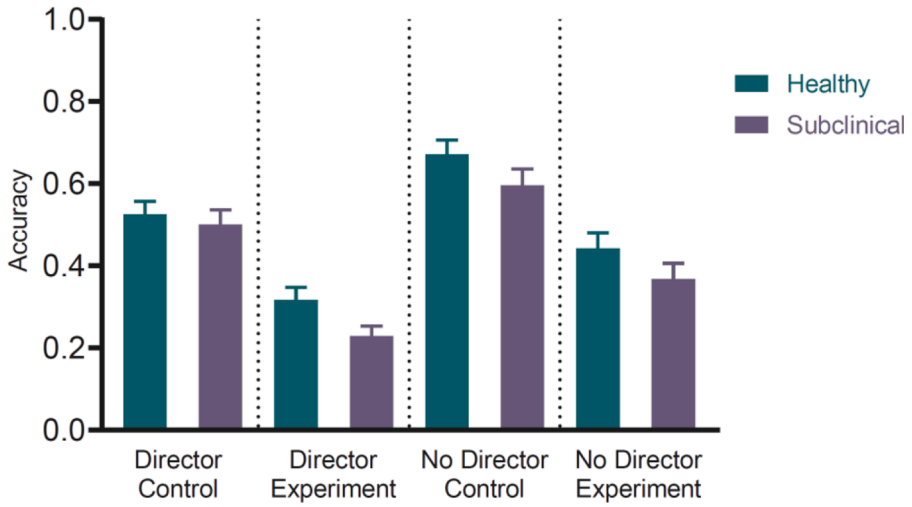

Fig. 3. Director task accuracy across trial types between psychotic-like experiences groups. Mean accuracy in the Director Task was significantly lower for participants with subclinical levels of psychotic experiences than healthy controls, including after controlling for age ($F_{1, 194} = 4.71$, $p = .031$, $\eta p^2 = 0.024$). Both groups performed worse in experimental trials and the director condition. Error bars are the standard error of the mean (SEM).

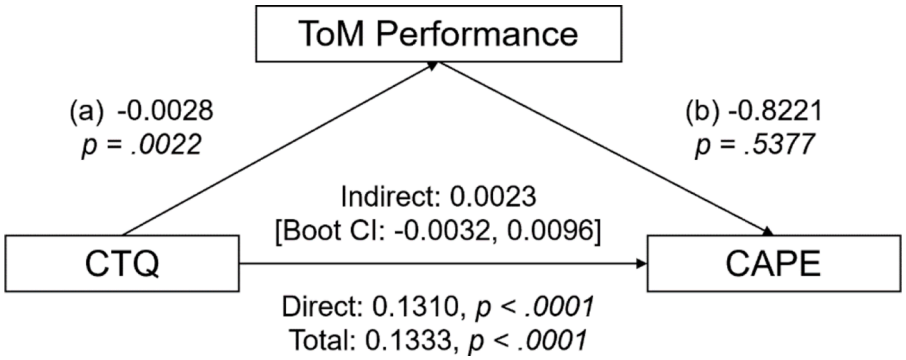

Fig. 4. Path diagram of mediation analysis. ToM performance did not mediate the association between CTQ and CAPE-P15 scores. Unstandardized beta regression coefficients are shown.

4. Discussion

To our knowledge, this is the first study in a non-clinical sample investigating the relationship between developmental trauma, ToM and psychotic experiences. Our findings suggest that participants who were exposed to developmental trauma and those with subclinical psychotic experiences made less accurate decisions when instructed to consider the Director's perspective. These results contribute to current literature on possible cognitive vulnerability mechanisms underlying the relationship between developmental trauma and psychotic experiences, implying that perspective-taking is impaired in people with a history of developmental trauma and/or psychotic experiences.

4.1. Interpretation of findings

Consistent with existing findings, we found elevated psychotic experiences in participants with a history of developmental trauma compared with controls. This association remained significant after adjusting for demographic and comorbid psychopathology related to trauma (Velikonja et al., 2015; Gibson et al., 2016). In particular, developmental trauma remained the greatest predictor of psychotic experiences after controlling for socioeconomic and clinical variables. Our findings support previous interpretations (Varese et al., 2012; Matheson et al., 2013) whereby exposure to traumatic life experiences during development impacts the pathogenesis of psychotic experiences in a dose- response manner.

In support of the hypothesis of the association between developmental trauma and ToM performance, the DT+ group demonstrated lower accuracy than the control group in experimental trials, demonstrating developmental trauma-related deficits in cognitive ToM. However, there was no difference in reaction time observed between the two groups, which could be attributed to unmeasured confounds. The hypothesis of the association between psychotic experiences and ToM performance was also supported, given that participants with psychotic experiences were less likely to apply information about the Director's perspective, even when given explicit instructions to do so. In contrast with our exploratory hypothesis, we did not find evidence of a mediating effect of ToM, as the effect of developmental trauma was not significantly mediated by ToM performance in the Director Task. Together, these results suggest that while developmental trauma and psychosis are associated with impaired ToM, these impairments may reflect general, non-specific cognitive effects of developmental trauma and/or psychosis, and further research is needed into whether ToM may represent a diagnosis-specific candidate vulnerability mechanism in trauma-induced psychosis. (Caspi A et al. 2014).

Performance in the director condition was poorer than that in the no-director condition across both DT+ and control groups. This may be because the director condition demanded greater utility of explicit ToM and executive function. In the no-director condition, participants solely needed to follow a simple memorisable rule (i.e., only move objects that are not placed in slots with grey backgrounds). On the contrary, this was absent in the director condition, and participants were expected to rely

on executive cognitive processes, such as visuospatial working memory and inhibitory control — all relevant processes in ToM and making social judgements — to deduce which items were visible to the director, allowing them to infer which object was being referred to. This difference in task accuracy is consistent with previous findings, reporting higher accuracy in the no-director condition, as opposed to the director condition irrespective of control or experimental trial types (Symeonidou et al., 2016; Pile et al., 2017; Dumontheil et al., 2010). We observed that reaction time did not differ between the developmental trauma and control groups. Previous findings with this outcome measure have been mixed (Dumontheil et al., 2010; (Pile et al., 2017) which may be due to small samples in previous research. However, our study was conducted in a larger sample.

Our findings suggest that deficits in explicit ToM are associated with a history of developmental trauma and psychotic experiences in adulthood, this remaining significant despite matching our sample for general demographic and clinical confounds such as age, sex, ethnicity and substance use. Such findings extend previous research reporting deficits in ToM at early stages of psychosis, including individuals with at-risk mental states (Ohmuro et al., 2016), clinical high risk (Stanford et al., 2011; Thompson et al., 2012; Zhang et al., 2016), as well as in first-episode psychosis (Langdon et al., 2014; Ohmuro et al., 2016; Sullivan et al., 2014; Thompson et al., 2012; Yang et al., 2017). Our findings in our non-clinical community sample are consistent with those reported by Bora and Pantelis (2013), wherein ToM was not only impaired in individuals with first-episode psychosis and individuals at an ultra-high risk for psychosis, but also in their unaffected relatives.

The lack of a mediating effect of ToM performance on developmental trauma and psychotic experiences suggests that ToM deficits may be non-specific to psychosis and may instead be common to disorders associated with developmental trauma. Impairments in affective ToM in people with borderline personality disorder (BPD) have been reported in a meta-analysis (Németh et al. 2018). Likewise, these impairments have been observed in people with post- traumatic stress disorder (PTSD) (Nazarov et al., 2014). This, in combination with our findings, suggests that ToM impairment may not be a diagnosis-specific mechanism underlying the development of psychotic experiences alone, but instead may reflect impairment in social cognition being involved in a shared, transdiagnostic traumatogenic increased risk of psychopathology (McLaughlin, K. A., 2016). Further research is needed to investigate these possibilities.

4.2. Research and clinical implications

Our findings on behavioural differences in ToM performance highlights the need for future research on the neurocognitive effects of developmental trauma on mentalization and its relationship to psychotic symptoms. It has been long understood that childhood encompasses a critical developmental period for explicit ToM, with traditional false-belief tasks typically completed by the age of 4 or 5 (Wellman et al., 2001; Scott and Baillargeon, 2017). However, maturation of ToM has been more recently reported to continue into adolescence (Dumontheil et al., 2010). While the developmental trajectory of ToM has been extensively researched, it is of importance for future research to explore the effects of early vs. late developmental trauma on ToM.

Deficits in ToM implicate difficulties with executive functioning. (Németh et al., 2020; Zelazo, 2020) This may therefore support the idea that executive processing difficulties are a consequence of atypical development and can be considered a transdiagnostic indicator of such development. This is important because executive processing difficulties indicate an increased risk for general features of psychopathology i.e., they may serve as a diagnostic tool for both atypical development and increased risk of general psychopathology. Moreover, such difficulties provide a target for therapeutic intervention; Executive processing skills can be improved through training and interventions (Diamond and Lee, 2011) and the efficacy of such training can be enhanced by mitigating disruptive influences such as stress (Zelazo, 2020). Future research therefore needs to investigate whether executive processing training can therefore offset psychosis risk progression in those that have experienced developmental trauma.

Our findings have several implications for adult survivors of developmental trauma with psychosis. In particular, the ToM deficits observed in undiagnosed individuals with psychotic experiences stresses the importance of early screening for psychotic symptoms in survivors of developmental trauma with difficulties in social cognition, as well as in people with a history of adverse childhood events. Growing evidence suggests ToM to be a risk factor for at- risk individuals transitioning to psychosis (Healey et al., 2013; Mayo et al., 2017). Monitoring mentalising ability in early screening may serve as a valuable measure of psychosis risk progression and therefore represents a possible biomarker.

5. Conclusion

In conclusion, our findings indicate that ToM is associated with developmental trauma and psychotic experiences. Our finding of a relationship between ToM performance and psychotic experiences supports the view that ToM may be involved in this association. However, it is possible that our finding may also indicate that impaired ToM may reflect a more generalised neurocognitive impairment in the clinical groups. To demonstrate whether or not this is so, further research could include a secondary behavioural task whereby the absence of a global neurocognitive impairment would be indicative of a specific neurocognitive impairment involved in the association. Further research investigating not only the late developmental trajectory of ToM, but also whether alterations in mentalization processing are specific to psychosis or represent a generalised increase vulnerability to psychopathology would shed light on the specific processes precipitating and exacerbating psychosis outcomes.

Declaration of Competing Interest

The authors declare no conflicts of interest.

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.psychres.2022.114544.

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