IMPACT OF ADVERSE CHILDHOOD EXPERIENCES AND TRAUMA EXPOSURE ACROSS THE LIFESPAN ON SOCIAL COGNITIVE FUNCTIONING AND AMYGDALA VOLUME IN A SAMPLE OF PRECARIOUSLY HOUSED ADULTS

FARZANEH MASHHADI

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Abstract

Precariously housed individuals are exposed to a multitude of adverse factors, such as elevated rates of childhood adversity, lifespan exposure to trauma, psychiatric illnesses, and substance use, which significantly increase their vulnerability to negative outcomes. The main objective of the current study was to investigate the impact of adverse childhood experience (ACEs) and lifespan trauma exposure on social cognitive functioning, including affective decision-making and theory of mind (ToM) abilities in a sample of precariously housed adults. Furthermore, the study aimed to explore the association between ACEs and lifespan trauma on the amygdala volume. Participants included 481 precariously housed adults living in an impoverished neighbourhood of Vancouver, Canada. Affective decision-making was measured using the Iowa Gambling Task (IGT), and ToM ability was assessed with the Reading the Mind in the Eyes Test (RMET). The results revealed that a greater number of traumatic exposures throughout the lifespan were associated with enhanced affective decision-making and ToM abilities. However, it was found that ACEs did not significantly impact social cognitive performances. Furthermore, no significant relationships were observed between ACEs, lifespan trauma, and the volume of the amygdala. Moreover, the study found no significant moderation effect of psychiatric illnesses and stimulant dependence on the relationship between ACEs, lifespan trauma, and social cognitive functioning. These findings shed light on the existence of inherent resilience factors within adverse experiences that can potentially mitigate negative outcomes. This underscores the need for future research to identify and delve into similar protective factors, especially within vulnerable populations such as precariously housed individuals who encounter significant barriers in accessing mental health services.

Keywords: Childhood adversity; lifespan trauma exposure; social cognition; affective decision-making; theory-of-mind; amygdala volume; precarious housing

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List of Acronyms

ACC	Anterior cingulate cortex
BECED	Best Estimate Clinical Evaluation and Diagnosis
CAN	Childhood Abuse and Neglect Questionnaire
DLPFC	Dorsolateral Prefrontal Cortex
DSM-IV	Diagnostic and Statistical Manual for Mental Disorders Fourth Edition
FFE	Fast Field Echo
IFG	Inferior Frontal Gyrus
IGT	Iowa Gambling Task
LHPA	Limbic-hypothalamic-pituitary-adrenal
MRI	Magnetic Resonance Imaging
MICE	Multivariate Imputation by Chained Equations
THQ	Trauma History Questionnaire
RMET	Reading the Mind in the Eyes Test
OFC	Orbitofrontal Cortex
PANSS	Positive and Negative Syndrome Scale
PFC	Prefrontal Cortex
ROI	Region of interest
SRO	Single-room occupancy

Introduction

An adverse childhood environment and trauma exposure across one's life are increasingly recognized for their substantial impact on physical and mental well-being across the lifespan (Hart & Rubia, 2012; Shonkoff et al., 2012). Epidemiological studies report a high prevalence of such adverse experiences in the general population (50%–70% estimate) and have recognized them as one of the major public health concerns of our time (Qureshi et al., 2011). Adverse childhood experiences (ACEs) encompass an array of highly negative early experiences, including emotional and physical abuse and neglect (together referred to as maltreatment), as well as negative household factors such as parental mental illness or suicide attempts, parental criminal activity and substance use, parental separation or death, prolonged separation from the caregiver, witnessing domestic violence, lack of education, and extreme poverty (Afifi et al., 2009; Xiang et al., 2022). Maltreatment and adversity are sometimes both referred to as trauma by the literature; however, there is still no clear definition of what constitutes a traumatizing experience (Kalmakis & Chandler, 2014). For clarity, in this study, the term adversity will be used to refer to negative childhood environment factors. Furthermore, the term trauma will signify a wide range of traumatic experiences, including physical and sexual abuse, major car accident or physical injury, serious or life-threatening illness, witnessing a natural disaster, death of a loved one, and crime-related events (e.g., being robbed or having experienced a break-in), which may have happened at any time during a person's life.

Part of the profound public health burden of these stressful experiences is due to the fact that the affected individuals can develop persistent challenges across a wide range of domains over their lives (Harms et al., 2016). Research has documented higher risks of homelessness (Liu et al., 2021), psychopathology (Evans et al., 2008; Copeland et al., 2018;), substance use

(Hughes et al., 2017; Dass-Brailsford & Myrick, 2010), suicidality (Afifi et al., 2016; Ásgeirsdóttir et al., 2018), cardiovascular disease, cancer, chronic lung disease, and drug overdose (Felitti et al., 1998; Dube et al., 2003; Danese et al., 2009; Wegman & Stetler, 2009; Bellis et al., 2019; Jones et al., 2020), and, more generally, lower quality of life (David et al., 2022) in individuals with ACEs and lifespan trauma exposure. Included in the extensive list of negative outcomes of ACEs and lifespan trauma is neurocognitive dysfunction (Masson et al., 2015; Pechtel & Pizzagalli, 2011; Teicher et al., 2016; Lynch & Lachman, 2020). Moderate to large effects have been reported across multiple cognitive domains, most notably in executive functioning (Kelder et al., 2018; Vasilevski & Tucker, 2016), processing speed (Malarbia et al., 2017; Masson et al., 2016), working memory (Blanchette & Caparos, 2016; Goodman et al., 2019), reward processing (Oltean et al., 2022), and intellectual functioning (Masson et al., 2015; Pechtel & Pizzagalli, 2011). Furthermore, the evidence from several meta-analyses and systematic reviews demonstrates that the detrimental impacts of ACEs on cognitive functioning appear to persist into adulthood, underscoring the enduring effect of childhood adversity on cognitive development (e.g., Lund et al., 2022; Masson et al., 2015; Pechtel & Pizzagalli, 2011). Evidently, an adverse childhood environment has been recognized as one of the earliest social determinants of health, which may create subsequent vulnerabilities to other risk factors (e.g., substance use and homelessness) that have an additive impact on cognition.

People who experience precarious housing, including accommodations that are unstable or in substandard conditions and provisional in nature, such as shelters and transitional housing, are disproportionally affected by ACEs and lifespan trauma exposure (Pluck et al., 2011). According to a meta-analysis conducted by Sundin and Baguley (2015), the prevalence of sexual and physical abuse in this population was estimated to be as high as 32% and 37%, respectively.

Previous studies have consistently reported elevated rates of childhood adversity among the precariously housed population. Moreover, individuals who have experienced ACEs, such as childhood abuse, neglect, household dysfunction, or parental substance abuse, are more likely to face housing instability during their early years, which in turn leads to an earlier age at entry into homelessness (Mar et al., 2014). The precariously housed population represents an exceptionally vulnerable segment of society, which is at higher risk of numerous adverse mental and physical health outcomes and premature mortality (Fazel et al., 2014). Alcohol and substance dependence affect a strikingly high proportion of the population, and evidence suggests that nearly half of the people who experience precarious housing are diagnosed with some form of psychotic illness (Fazel et al., 2008). Furthermore, emerging evidence suggests that cognitive impairment is an additional adverse outcome that is associated with unstable housing (Danese et al., 2017; Depp et al., 2015; Fry et al., 2017; Stone et al., 2019). The most prominently affected cognitive domains include verbal memory, sustained attention, and working memory, with additional but less pronounced deficits observed in executive functions and processing speed (Waclawik et al., 2019). Cognitive impairments in homeless and precariously housed persons are thought to reflect exposure to multiple risk factors (e.g., traumatic brain injury, substance use, and psychiatric illnesses; Gicas et al., 2014). However, the contribution of ACEs and lifespan trauma exposure has not been well researched; this topic is mostly limited to studies of attention, memory, and executive functioning.

This study extends this work by examining the effects of ACEs and lifespan trauma on social cognitive processes, including affective decision-making and theory-of-mind (ToM) —an individual's ability to understand and make accurate inferences about the intentions and thoughts of others—in a sample of homeless and precariously housed adults. Social cognitive processes

may be uniquely vulnerable to the impacts of trauma, given how frequently these adverse experiences, especially childhood maltreatment, occur in relational and interpersonal contexts. Young children depend on their caregivers to provide love and safety, forming the foundation of expected social relationships. However, when caregivers deviate from fulfilling these crucial roles, it can have an impact on the development of neural structures involved in social processing within the brain (Hein & Monk, 2017). Evidence suggests that maltreated children have notable disruptions in their interpersonal relationships and social functioning in adulthood (Rokita et al., 2018). Suggested pathways to social functioning difficulties in persons who have experienced ACEs or lifespan trauma include difficulties with emotion recognition in self and others (Wagner et al., 2015), mentalizing abilities (ToM; Quide et al., 2017; Rokita et al., 2021), social perception (Rokita et al., 2018), emotion regulation (Green et al., 2015; Kilian et al., 2017), and heightened emotional reactivity in interpersonal contexts (Cassiers et al., 2018). Dysfunctions in social cognition are highly predictive of negative outcomes in daily functioning, including occupational functioning (Green, 2016), sense of autonomy and independent living (Islam et al., 2020), maintenance of interpersonal relationships (Fett et al., 2011), and community integration and functioning (Horan et al., 2013). These negative outcomes are especially relevant to homeless and precariously housed persons, given their challenging living environments and the barriers they face in accessing mental health care (Argintaru et al., 2013; Honer et al., 2017). These individuals tend to have small and defective social support networks and are prone to loneliness and social alienation (Hawkins & Abrams, 2007), which may add an additional layer of complexity to their social cognitive functioning. Additionally, emerging evidence has documented the influence of ACEs on other aspects of social cognitive functioning, including reward processing, which is closely associated with emotion-based decision-making. Essentially,

rewards and punishments are less predictable and may be qualitatively different in adverse and abusive early environments from those experienced in more normative and typical settings (Armbruster-Genc et al., 2022). For instance, these experiences may be characterized by a greater emphasis on punishment and a lack of reward (Cyr et al., 2010). Studies of individuals with a history of ACEs who have experienced high levels of stress during their early years consistently report significant alterations in behavioural responses in fundamental aspects of reward processing (Armbruster-Genc et al., 2022). Evidence supports that these individuals seem to have difficulties in effectively using environmental cues that signal rewards and losses, which can have negative implications for their decision-making abilities (Birn et al., 2017). Findings from neuroimaging studies lend further support to the evidence of the association between ACEs and impaired reward processing. Particularly, conserving findings consistently demonstrated decreased neural activation associated with reward anticipation in the brain's reward circuitry in maltreated individuals relative to those with no history of childhood maltreatment (Dillon et al., 2009; Gerin et al., 2017; Hanson et al., 2015; Mehta et al., 2010). This evidence offers insights into the prevalent patterns of poor decision-making and maladaptive risk-taking behaviours among individuals with ACEs, which disproportionately increases their risk for manifold of negative physical and mental health outcomes (Birn et al., 2017).

A developing brain is experience-dependent and requires consistent stimulation from the environment to promote neural growth and connectivity (Vasilevski & Tucker, 2016). Thus, adverse childhood experiences can impact the brain's developmental trajectory and create varied functional difficulties (Vasilevski & Tucker, 2016). Neuroimaging research has demonstrated further impacts of trauma exposure and acute stress on brain morphology, predominantly in neocortical structures and the amygdala (Luo et al., 2022). The amygdala, a vital component of

the limbic system, is primarily recognized for its role in processing fear-related and emotionally salient stimuli (Gothard & Fuglevand, 2022). More recently, observations from focal brain lesions in humans and experimental lesion studies in animals have revealed the amygdala's contributions to higher-order social information processes (Han et al., 2021), including social decision-making (Gangopadhyay et al., 2020), social judgment, and social interaction (Adolphs, 2010). Upon birth, the amygdala is present in its basic cytoarchitecture and functional capacities; it develops rapidly thereafter, reaching its maturation as early as age 4 in females (Shaw et al., 2008). This rapid maturation indicates a very early sensitive period during which stressors in the child's environment can disrupt the amygdala's normal development (Tottenham & Sheridan, 2010). A comprehensive meta-analysis conducted by Paquola et al. (2016) revealed a significant association between childhood trauma and structural abnormalities in the amygdala, specifically bilateral volume reduction. Furthermore, one of the most extensively documented functional alterations associated with childhood adversity (Hein & Monk, 2017; Kraaijenvanger et al., 2020; Teicher & Samson, 2016) and traumatic experience across one's life (Patel et al., 2012) is the heightened amygdala hyperactivity in response to emotional stimuli. Additionally, accumulating neuroimaging findings have demonstrated alterations in functional connectivity patterns among individuals with a history of ACEs (Cassiers et al., 2018; Lim et al., 2020; Tian et al., 2021). These alterations often involve disrupted connectivity within brain networks associated with emotion regulation, stress response, cognitive control, and reward processing. Studies have consistently documented connectivity alterations within the prefrontal-limbic circuitry, characterized by amygdala hyperconnectivity with medial and dorsolateral prefrontal cortices associated with ACEs (Cisler, 2017; Luo et al., 2020; Peverill et al., 2019). Together these findings suggest that the observed changes in the amygdala's structure and functioning

likely contribute to the complex relationship between ACEs, lifespan trauma, and social cognitive outcomes.

Despite the extensive evidence supporting the impact of childhood adversity and lifespan trauma exposure on cognitive functioning and the brain, significant gaps in the literature still exist. Specifically, certain potentially key moderators of this effect have received limited research attention. In particular, the increased prevalence of psychiatric diagnoses and substance use within the trauma-exposed population (Kessler et al., 2010; McLaughlin, 2016) necessitates further investigation into the potential moderating role of these risk factors in adverse outcomes (Schalinski et al., 2018). Cognitive impairment is a hallmark of nearly all primary psychotic disorders, with a strikingly high percentage of the affected individuals demonstrating markedly impaired performance across a wide range of cognitive domains (McCleery & Nuechterlein, 2019). Research has revealed an additive effect of childhood maltreatment and psychotic illness, whereby individuals who have experienced both risk factors exhibit more pronounced neurocognitive deficits compared to individuals with psychosis alone and no history of childhood maltreatment (Dauvermann & Donohoe, 2019). Furthermore, according to research by Fernández-Serrano et al. (2011), stimulant use, particularly compared to other illicit substances, is more likely to result in persistent neurocognitive deficits. Research has consistently shown that prolonged and excessive stimulant use is associated with a significant impact on cognitive functioning across multiple domains, including attention, memory, executive functions, and decision-making abilities (Verdejo-Garcia & Rubenis, 2020; Wood et al., 2013). In spite of this evidence, there is a lack of comprehensive studies exploring the potential moderating effects of substance use and psychiatric illness on the association between childhood adversity and cognitive deficits in adulthood (Edalati & Krank, 2016). To fill this gap in the literature, the

current study aims to investigate the influence of psychotic illness and stimulant dependence on the relationship between ACEs, lifetime trauma exposure, and social cognitive outcomes.

Current Study

Objectives

The overall objective of this study was to investigate the impacts of ACEs and lifespan trauma exposure on cognition and the regional brain volume in a sample of precariously housed adults. Specifically, the study aimed to examine the effect of ACEs and lifetime trauma on social cognitive functioning in the domains of affective decision-making and ToM abilities, given the potential unique vulnerability of these cognitive processes to adverse experiences. Additionally, amygdala volume was examined as the key neural correlate that may subserve the above association. Secondary analyses were conducted to investigate the contribution of key moderators (psychotic illnesses and stimulant use) on social cognitive outcomes associated with trauma and childhood adversity.

Hypotheses

1) Higher rates of ACEs and lifespan trauma exposure will be associated with poorer performance on measures of affective decision-making and ToM abilities.
2) Higher rates of ACEs and lifespan trauma exposure will be associated with smaller total amygdala volume.
3) Total amygdala volume will mediate the association between ACEs, lifespan trauma exposure and social cognitive performance.
4) Diagnosis of psychotic illness and stimulant dependence will moderate the association between ACEs, lifespan trauma exposure, and social cognitive performance. Specifically, it

is expected that these risk factors will strengthen the association between ACEs, lifespan trauma, and social cognitive performance.

Methods

Participants

Participants were recruited as part of an ongoing longitudinal study of the health of precariously housed persons (the Hotel study; Honer et al., 2017; Vila-Rodriguez et al., 2013). Four hundred eighty-one (N = 481) participants were recruited via staggered enrollment between 2008 and 2017 from single-room occupancy (SRO) hotels and the community courthouse located in Vancouver's impoverished Downtown East Side neighbourhood. Enrollment eligibility included being 18 years of age or older, having fluency in English, and having the ability to provide written informed consent. All the participants received a monetary honorarium for their involvement in the study. Ethics approval for data collection was obtained from the Clinical Research Ethics Board of the University of British Columbia and Simon Fraser University. Ethics approval to conduct secondary analyses of the data was obtained from York University's Office of Research Ethics.

Materials and Procedures

Clinical and Risk Factor Assessment

Clinical assessments were conducted for all participants at study entry by trained research assistants, psychiatrists, and neurologists. The assessment sessions were scheduled at a different time from the neuropsychological test sessions. Demographic variables, including age, total years of education, gender, and ethnicity, were obtained through self-report during the baseline interview. Psychiatric and substance dependence diagnoses were determined by a psychiatrist using the Best Estimate Clinical Evaluation and Diagnosis (BECED) method (Endicott, 1988),

including the Mini-International Neuropsychiatric Interview (Sheehan et al., 1998), and rendered by consensus according to the Diagnostic and Statistical Manual for Mental Disorders fourth edition (DSM-IV; APA, 2013). History of regular use and the age of first exposure to alcohol and major classes of drugs (e.g., cocaine, methamphetamine, heroin, cannabis) were obtained by self-reports. Vila-Rodriguez et al. (2013) present a comprehensive overview of the assessments in the Hotel study. Details related to the current study are presented below.

Childhood Adversity and Lifespan Trauma History

A total number of lifespan traumatic experiences, including physical and sexual abuse, crime-related events, and natural disasters, were obtained with the Trauma History Questionnaire (THQ; Green, 1996), which is a commonly used measure of life history of traumatic events that has demonstrated good psychometric properties (Hooper et al., 2011). In the THQ, for each question, participants report whether they have ever experienced that type of trauma, and a total score is calculated as the sum of all the occurrences.

An assessment of adverse childhood home environment was obtained with the Childhood Abuse and Neglect (CAN) questionnaire (Wilson et al., 2006). The CAN questionnaire consists of five subscales assessing emotional neglect, family turmoil, financial need, parental intimidation, and parental violence. All the questions address experiences during the first 18 years of life. A composite score combining information from all five subscales can also be produced; hereafter, this will be defined as the study's index of ACEs.

Neurocognitive Assessment

Affective decision-making performance was measured with the Iowa Gambling Task (IGT). The IGT is a standardized computerized measure that assesses decision-making under risk and uncertainty (Bechara et al., 1994). In the IGT, participants are instructed to maximize

monetary gain while repeatedly choosing from four decks of cards (labelled A, B, C, and D), which yield wins and losses variably across 100 trials. Choices from decks C and D lead to moderate gains and moderate losses, resulting in a net gain, while choices from decks A and B result in high immediate gains and high long-term losses resulting in a net loss. Accordingly, decks C and D are deemed "advantageous," while decks A and B are deemed "disadvantageous" (Mukherjee & Kable, 2014). Participants are unaware of these facts and must learn to maximize their financial gain based on the win and loss feedback they receive after each selection. Typical performance evolves as the task progresses, as most healthy participants begin to learn the implicit properties of each deck and make more choices from the advantageous decks by the end of the task (Brand et al., 2007). Performance is depicted in terms of net score, calculated as the number of cards selected from the advantages decks minus the number of cards selected from the disadvantages decks (i.e., [Deck C + Deck D] - [Deck A + Deck B]). Therefore, higher net scores signify better performance in the task. In the current study, the IGT net score was used as a global index of affective decision-making performance. The IGT taps into different aspects of value-based decision-making, including one's affective reaction to reward and punishment (De Vries et al., 2008), one's ability to learn based on positive and negative feedback (i.e., reinforcement learning; O'Leary et al., 2013), and one's cognitive flexibility (i.e., the ability to respond flexibly to changing contingencies; Barry & Petry, 2008).

Theory of mind ability was assessed with the Reading the Mind in the Eyes Test (RMET; Baron-Cohen et al., 2001). The RMET is a 36-item computerized measure in which participants view photographs of the eye regions of a person depicting an emotional state. Each photograph is accompanied by four alternative labels proposing what the person may be thinking or feeling. Participants are then asked to decide which of the four labels accurately describes the person's

mental state (Megías-Robles et al., 2020). In this study, a modified version of. The RMET was utilized. This modified version included the provision of word definitions to participants when needed, with the aim of compensating for any potential language-related difficulties. Performance on the RMET is reported based on the number of corrected responses. The RMET was developed under the premise that social emotion perception is heavily based on the understanding of eye gaze in others (Baron-Cohen et al., 2001; Olderbak et al., 2015).

Following completion of the neurocognitive assessment, the examiner subjectively rated the validity of the measures on a scale ranging from 1 = clearly invalid to 5 = clearly valid. Invalid ratings were provided for several reasons, such as participant intoxication, extreme fatigue, inability to adequately comply with test instructions, frustration, or equipment failure.

Neuroimaging Acquisition and Processing

Structural magnetic resonance imaging (MRI) scans were acquired close to the neurocognitive testing session. Whole-brain MRI scans were obtained using a Philips Achieva 3T scanner equipped with an 8-channel SENSE head coil. Visual inspection of the images was performed to detect significant motion artifacts. A 3D Fast Field Echo (FFE) T1-weighted sequence was obtained in the sagittal plane. One hundred and ninety slices, each 1 mm thick, were obtained with the following parameters: acquisition matrix = 256 × 250, field of view = 256 × 256 × 190 mm^3, reconstruction matrix 256 × 256 × 190, voxel size = 1 × 1 × 1 mm^3, flip angle = 8◦, TR = 7.6 ms, and TE = 3.5 ms. Automatic cortical parcellation was conducted using the publicly available FreeSurfer 6.0 software (available for download at https://www.nmr.mgh.harvard.edu/) according to the Desikan-Killiany atlas (Desikan et al., 2006). The Desikan-Killiany atlas is a commonly used scheme to parcellate brain volume regions of interest (ROIs; Alexander et al., 2019). An additional automated protocol from FreeSurfer 6.0

was used to measure the bilateral whole amygdala volume in cubic millimetres (mm^3). To account for variations in head size, the adjusted amygdala volume was calculated using the residuals from a linear regression based on the least-squares between the raw volumes and intracranial volumes (ICV).

Statistical Analyses

All analyses were conducted using R software (R Core Team, 2020). Data has been screened for missing items and invalid entries. As a sensitivity analysis to explore the impact of missing data, all primary analyses were reconducted using an imputed dataset generated with a multivariate imputation by chained equations (MICE) procedure (see Azur et al., 2011 for further details). The results based on the imputed dataset are outlined in Appendix B. A comparable set of findings was observed, and the results showed that the missingness in the data did not have a substantial effect or influence on the results. All results presented herein are based on the original (not imputed) dataset.

Hypothesis 1: Higher rates of ACEs and lifespan trauma exposure will be associated with poorer social cognitive performance.

A series of multiple regressions was conducted to test the first hypothesis. Separate models were fitted for each neurocognitive outcome measure (IGT, RMET). The models were examined and the residual plots were visually inspected to assess for assumptions of linearity, normality of residuals, homogeneity of residuals, independence of residual error terms, and multivariate outliers. None of the assumptions were violated for any of the models. Each model contained a key predictor variable, the CAN composite score or THQ total score, and an outcome variable, the IGT or RMET scores. All four models included demographic variables

(age, gender, total years of education) as covariates. These covariates were selected based on their known associations with cognitive functioning (Brewster et al., 2014; Qin et al., 2020).

Hypothesis 2: Higher rates of ACEs and lifespan trauma exposure will be associated with smaller total amygdala volume.

Two multiple regression analyses were conducted to assess the second hypothesis. The models were examined, and the residual plots were visually inspected to assess if all the regression assumptions were met. All assumptions of normality, independence of residual error terms, multivariate outliers, and homoscedasticity were deemed met. The models included the CAN composite score or the THQ total score as predictor variables and an index of amygdala volume measured in cubic millimetres (mm^3) as the outcome. For each participant, the amygdala volume is divided by intracranial volume to adjust for head size. Age and gender were included in the model as covariates, given their associations with structural differences and changes in the brain (Lee & Kim, 2022; Zhang et al., 2020).

Hypothesis 3: Total amygdala volume will mediate the association between ACEs and trauma exposure and social cognitive performance.

A series of mediation analyses were conducted to determine if the pathway from ACEs and lifespan trauma exposure to social cognitive functioning will operate indirectly via amygdala volume (see Figure 1. For the conceptual diagram of the statistical model). Separate models were fitted for each neurocognitive measure (IGT, RMET). Total amygdala volume was entered into the models as the mediator, and age and gender were included as the covariates.

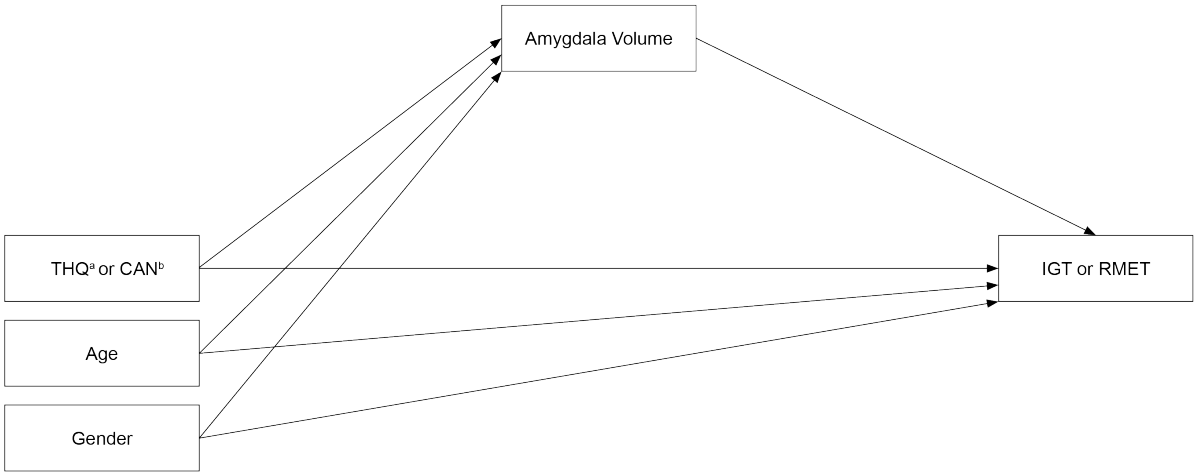


Figure 1. Conceptual Diagram of the Mediation Model Examining the Mediating Effect of Amygdala Volume on the Association Between ACEs, Lifespan Trauma Exposure, and Social Cognitive Performances.

Note. THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = IOWA Gambling Task; RMET = Reading the Mind in the Eyes Test.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Hypothesis 4: Diagnosis of psychotic illness and stimulant dependence will moderate the association between ACEs, lifespan trauma exposure, and social cognitive performance. Specifically, it is expected that these risk factors will strengthen the association between ACEs, lifespan trauma, and social cognitive performance.

A series of moderation analyses were conducted to examine whether a diagnosis of psychotic illness (schizophrenia, schizoaffective disorder, mood disorders with psychosis, substance-induced psychosis) and stimulant dependence will moderate the association between ACEs, lifespan trauma exposure, and social cognitive performance (see Figure 2. for the conceptual diagram of the statistical model). Separate models were fitted for each neurocognitive measure (the IGT and RMET), and each model contained the CAN composite score or the THQ total score as the predictor variable. Diagnoses of psychotic illness and stimulant dependence were separately entered as moderators in the models. The models included demographic variables (age, gender, total years of education) as covariates.

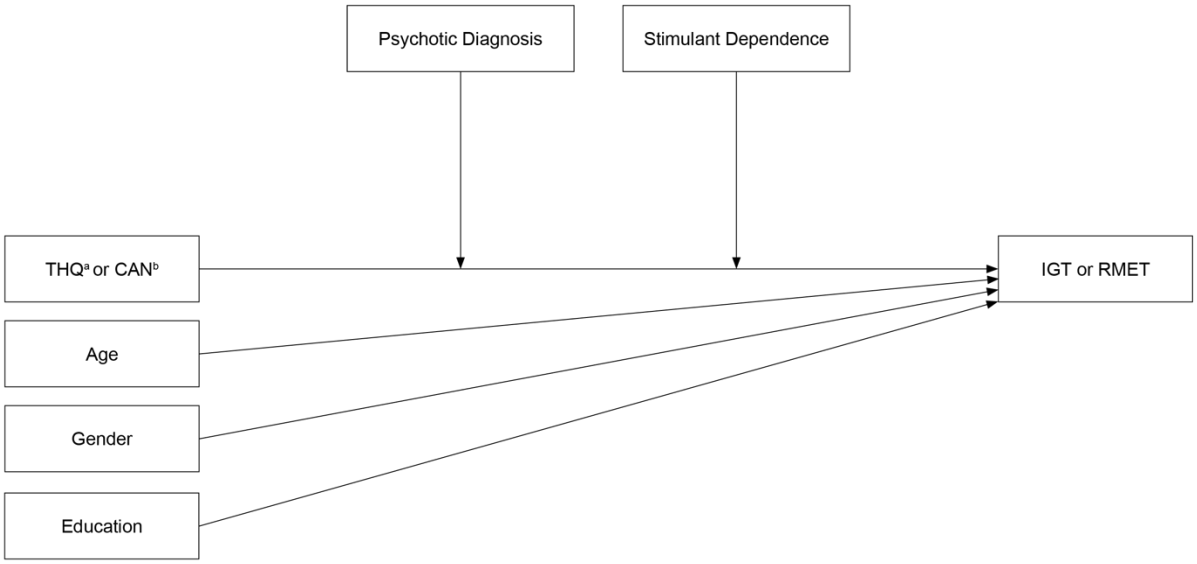


Figure 2. Conceptual Diagram of the Moderation Analyses Examining the Impact of Psychotic Illnesses and Stimulant Dependence on the Association Between ACEs, Lifespan Trauma Exposure, and Social Cognitive Performance.

Note. THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = IOWA Gambling Task; RMET = Reading the Mind in the Eyes Test.

c. Total number of lifespan traumatic experiences obtained from the THQ.
d. A composite score obtained from the CAN questionnaire subscale.

Results

Sample Characteristics

Descriptive information for the sample is presented in Table 1. To summarize, the average age of the participants was 42.59 (*SD* = 12.10, range = 20-75); 75% were male and had approximately 10.5 years of education (*SD* = 2.38, range = 2-16). Sixty percent of the participants self-identified as being of European descent, 34% as Indigenous or Indigenous mix, and 6% as other ethnicity. There was a high rate of major mental illnesses among the participants. The most prevalent psychiatric illnesses included schizophrenia and schizoaffective disorder (20%), substance-induced psychosis (16%), mood disorders, including bipolar I or II (11%), and major depression (13%). The sample also had high rates of substance use disorders, including stimulant dependence (78%), opioid dependence (49%), cannabis dependence (37%),

and alcohol dependence (20%). The mean scores for the PANSS included 37.53 (*SD* = 8.70) for the general subscale, 16.57 (*SD* = 5.59) for the negative subscale, and 16.33 (*SD* = 6.14) for the positive subscale. These mean scores are consistent with persons with serious mental illness who are living in the community and have stable psychotic symptoms, suggesting that participants were mostly not acutely psychotic at the time of testing (Whelton et al., 1999).

A summary of the descriptive statistics for the risk factor assessment, neurocognitive tasks, and brain measures is depicted in Table 2. Of the total participants (N = 481), different measures had different sample sizes due to invalid and/or missing data, as outlined in Table 2. The percentage of missing data included 11% for the IGT, 60% RMET, 2% for the THQ, 22% for the CAN questionnaire, and 19% for MRI brain scans.

Table 1. Sample Characteristics (N = 481).

Characteristic	N (%)	Mean (SD)	Median	Range
Age (years)	481	42.59 (12.10)	44	20–75
Education (years)	481	10.49 (2.38)	10	2–16
Symptoms of Psychosis (PANSS)	459 (95.43)			
Positive		16.33 (6.14)	15	7–36
Negative		16.57 (5.59)	16	7–39
General		37.53 (8.70)	37	19–63
Total		70.42 (17.18)	68	11–132
Gender				
Male	362 (75.26)			
Female	119 (24.74)			
Ethnicity				
White	291 (60.50)			
Indigenous	120 (24.95)			
Indigenous Mix	43 (8.94)			
Other/Mixed/Unknown	27 (5.61)			
Psychiatric Diagnosis				
Psychotic illnesses, any	250 (52)			
Schizophrenia	50 (10.40)			
Schizoaffective	46 (9.56)			
SIP	77 (16.01)			
Mood disorders, any	119 (24.74)			
Depression	64 (13.31)			
Bipolar I	35 (7.28)			
Bipolar II	21 (4.37)			

Substance Dependence Disorder	
Alcohol	97 (20.17)
Cannabis	176 (36.59)
Opioid	235 (48.86)
Stimulant	377 (78.38)

Note. N = 481; PANSS = Positive and Negative Syndrome Scale; SIP = substance-induced psychosis.

Table 2. Descriptive Statistics for Brain Imaging and Neurocognitive Measures.

Variable	N (%)	Mean (*SD*)	Median	Range
IGT	424 (88.14)	-2.10 (33.83)	-2	-96–100
RMET	191 (39.71)	21.10 (5.82)	22	7–34
THQ[a,e]		8.21 (4.31)	8	0–19
Physical	463 (96.26)	1.20 (1.03)	1	0–3
Sexual	466 (96.88)	0.71 (1.03)	0	0–10
Crime		1.85 (1.28)	2	0–4
General		4.53 (2.59)	4	0–12
CAN[b,f]	374 (77.75)	21.30 (12.47)	20	0–51
Neglect		7.37 (5.51)	8	0–18
Family Turmoil		3.36 (2.06)	3	0–8
Parental Intimidation		6.05 (4.51)	6	0–16
Parental Violence		2.69 (2.34)	2	0–8
Poverty		1.83 (2.09)	1	0–8
MRI brain scan[c]	391 (81.29)			
Intracranial volume × 10^{-3}		1555.17 (138.48)	1558.87	1059.86–1910.95
Total amygdala volume (mm^3)[d]		3423.81 (691.09)	3386.76	1478.36–5489.46

Note. N = 481; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = IOWA Gambling Task; RMET = Reading the Mind in the Eyes Test.

a. THQ total and subscales, n (%) = 473 (98.34) unless otherwise specified.
b. CAN total and subscales, n (%) = 374 (77.75).
c. MRI brain scans, n (%) = 391 (81.29).
d. Amygdala volume reflects adjusted data.
e. Total number of lifespan traumatic experiences obtained from the THQ.
f. A composite score obtained from the CAN questionnaire subscale.

Regression Analyses

Iowa Gambling Task (IGT)

Two separate models were fitted to examine the impact of ACEs and lifespan trauma exposure on IGT performance. A Pearson correlation coefficient estimate revealed a moderate positive correlation (r = .32) between the two predictor variables (THQ and CAN), indicating a

strong likelihood that they capture distinct constructs. A summary of the regression coefficients is provided in Table 3, and the scatterplots of the correlations are shown in Figure 3.

Regression analyses revealed that lifespan trauma exposure but not ACEs is significantly associated with performance on the IGT. Specifically, in a regression model adjusted for age, gender, and education, a greater total number of lifespan traumatic experiences was associated with better IGT performance (B = 1.07, $p = 0.005$). Although this was in line with the study's first hypothesis, the positive direction of the association was contrary to expectation. A similar positive association was also observed for the relationship between ACEs and IGT, but the results were not statistically significant. In both models, education was significantly associated with the IGT performance. No significant associations were found between other covariates (age and gender) and the outcome measure. Secondary analyses revealed a similar pattern of results for the association between trauma subtypes as indexed by THQ and CAN subscale scores and the performance on the IGT (a summary of the regression coefficients is reported in Appendix A). The analysis of the THQ subscales showed that crime (B = 2.94, $p = 0.02$) and general trauma (B = 1.79, $p = 0.006$) subscale scores were significantly associated with better IGT performance, whereas the physical and sexual subscales coefficient estimates were not significant. The results revealed no significant association between any of the five CAN subscales and the IGT scores, consistent with the finding that there is no significant correlation between ACEs and performance on the IGT.

Reading the Mind in the Eyes Test (RMET)

Two individual models were fitted to examine the association between ACEs, lifespan traumatic experiences, and RMET performance. Regression analyses showed that lifespan trauma exposure is significantly correlated with performance on the RMET. When age, gender, and education were held constant, a greater number of lifespan traumatic experiences were

associated with better performance on the RMET (B = 0.29, p = 0.003). Although this was consistent with what was hypothesized, the positive direction of this effect was unexpected. No significant association was found between ACEs and the RMET. In both models, education was positively correlated with the performance on the RMET. No other significant associations were found for the other two covariates (age and gender). Table 4 provides a summary of the regression coefficients, and the scatterplots of the relationships are presented in Figure 4. Results of the secondary analyses revealed consistent findings regarding the association between THQ and CAN subscale scores and the RMET scores (Appendix A provides a summary of the regression coefficients). The results showed no significant association between the CAN subscales and the RMET scores, similar to the finding that there is no significant association between ACEs and the performance on the RMET. With regards to the THQ subscales, physical trauma was the only subscale with no significant association with the RMET score. Other subscales, including sexual (B = 1.06, p = 0.03), crime (B = 0.99, p = 0.003), and general (β = 0.35, p = 0.02) trauma subscales, were all positively associated with performance on the RMET.

Table 3. Model Coefficients for the Association Between the Adverse Childhood Experiences, Lifespan Trauma Exposure, and the Iowa Gambling Task (IGT).

	B (*SE*)	*t*-value	*p*-value	95% CI
Lifespan trauma exposure				
Intercept	-21.13 (9.92)	-2.13	0.03 *	[-40.63—1.62]
THQ[a]	1.07 (0.39)	2.73	0.005 **	[0.30—1.84]
Age	-0.09 (0.14)	-0.63	0.53	[-0.36—0.19]
Gender	-6.67 (3.97)	-1.68	0.09	[-14.47—1.14]
Education	1.47 (0.71)	2.07	0.04 *	[0.08-2.87]
ACEs				
Intercept	-19.51 (12.21)	-1.60	0.11	[-43.53—4.50]
CAN	0.07 (0.16)	0.43	0.67	[-0.24—0.40]
Age	-0.01 (0.16)	-0.04	0.97	[-0.13—0.3]
Gender	-7.18 (4.57)	-1.57	0.12	[-16.17—1.81]
Education	1.75 (0.83)	2.10	0.04 *	[0.11—3.39]

Note. THQ x IGT, n = 418; CAN x IGT n = 308; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = IOWA Gambling Task; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Table 4. Model Coefficients for the Association Between Adverse Childhood Experiences (ACEs), Lifespan Trauma Exposure, and Reading the Mind in the Eyes Test (RMET).

	B (*SE*)	*t*-value	*p*-value	95% CI
Lifespan trauma exposure				
Intercept	13.69 (2.91)	4.70	<.001 ***	[7.94—19.43]
THQ[a]	0.29 (0.96)	3.07	0.003 ***	[0.10—0.48]
Age	-0.01 (0.04)	-0.27	0.78	[-0.10—0.07]
Gender	0.60 (1.02)	0.59	0.56	[-1.42—2.61]
Education	0.50 (0.19)	2.65	0.01 **	[0.13—0.87]
ACEs				
Intercept	13.26 (3.3)	3.29	<.001 ***	[6.76—19.76]
CAN[b]	0.03 (0.04)	0.04	0.33	[-0.03—0.10]
Age	0.04 (0.04)	0.05	0.45	[-0.06—0.13]
Gender	0.16 (1.09)	1.09	0.88	[-1.99—2.32]
Education	0.57 (0.19)	2.92	0.004 ***	[0.18—0.95]

Note. THQ × RMET, $n = 190$; CAN × RMET $n = 160$; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; RMET = Reading the Mind in the Eyes Test; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Amygdala volume

In two regression models adjusted for age and gender, neither the number of lifespan traumatic experiences nor ACEs were associated with total amygdala volume. A summary of the regression coefficients is depicted in Table 5, and the scatterplots of the correlations are shown in Figure 5. In both models, age and gender were negatively correlated with amygdala volume. The overall models were both statistically significant; however, this is likely due to the substantial effects of the two covariates (age and gender) included in the models. Therefore, in contrast to the second hypothesis, a higher rate of lifespan traumatic experiences and ACEs were not associated with total amygdala volume. In a secondary set of analyses, the results showed no significant association between any of the THQ and CAN subscales and the total amygdala volume (see Appendix A for a summary of the regression coefficients).

Table 5. Model Coefficients for the Association Between Adverse Childhood Experiences, Lifespan Trauma Exposure, and Total Amygdala Volume.

	B (*SE*)	*t*-value	*p*-value	95% CI
Lifespan trauma exposure				
Intercept	4135.16 (1344.2)	30.82	<.001 ***	[3871.4—44399]
THQ[a]	6.89 (7.76)	0.89	0.38	[-8.37—22.15]
Age	-15.82 (2.75)	-5.75	<.001 ***	[-21.23—-10.41]
Gender	-371.90 (78.61)	-4.73	<.001 ***	[-526.5—-217.33]
ACEs				
Intercept	4119.57 (169.42)	24.32	<.001 ***	[3786.10—4453.04]
CAN[b]	1.30 (3.22)	0.40	0.69	[-5.04—7.65]
Age	-14.46 (3.25)	-4.46	<.001 ***	[-20.85—-8.07]
Gender	-358.84 (92.2)	-3.89	<.001 ***	[-540.27—-177.41]

Note. Exposure to trauma model, *n* = 384; Adverse childhood experiences model, n = 290; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Mediation Analyses

A series of mediation analyses were conducted to examine whether amygdala volume will mediate the relationship between ACEs, lifespan trauma and social cognitive performance. A summary of the regression coefficients is depicted in Tables 8 and 9. The results revealed that the estimated direct effect of lifespan trauma on IGT (B = 1.19, $p = 0.001$) and RMET (B = 0.38, $p = 0.23$) performance was significant. However, similar to the primary multiple regression analyses, the results showed no significant direct effect of ACEs on the IGT and RMET. Furthermore, none of the indirect effect coefficients were statistically significant. Thus, contrary to what was hypothesized, amygdala volume did not mediate the association between ACEs, lifespan trauma exposure, and social cognitive performance.

Table 6. Model Coefficients for the Association Between Adverse Childhood Experiences, Lifespan Trauma Exposure, and the Iowa Gambling Task (IGT) as Mediated by Total Amygdala Volume.

	B (*SE*)	*z*-value	*p*-value	95% CI
Lifespan trauma exposure				
Amygdala.volume × THQ[a]	6.92 (7.4)	0.94	0.35	[-9.64—21.68]
Amygdala.volume × IGT	-0.003 (0.003)	-1.30	0.19	[-0.008—0.003]
Amygdala.volume × Age	-15.79 (2.66)	-5.93	<.001 ***	[-21.09—-9.83]
Amygdala.volume × Gender	-374.50 (81.33)	-4.61	<.001 ***	[-556.07— -234.42]

THQ.net × IGT (Direct effect)	1.19 (0.36)	3.28	0.001 ***	[0.30—1.84]
IGT × Age	-0.2 (0.13)	-1.21	0.23	[-0.39—0.10]
IGT × Gender	-8.63 (3.67)	-2.35	0.02	[-18.54—-2.63]
Indirect effect	-0.02 (0.04)	-0.62	0.54	[-0.12—0.03]
Total effect (Direct + Indirect)	1.16 (0.36)	3.21	0.001	[0.40—1.92]
Mediated proportion (Indirect/Total)	-0.02 (0.05)	-0.45	0.65	[-0.15—0.03]
ACEs				
Amygdala.volume x CAN[b]	1.32 (3.26)	0.41	0.69	[-4.69—7.68]
Amygdala.volume x IGT	-0.003 (0.003)	-1.02	0.31	[-0.01—0.001]
Amygdala.volume × Age	-14.42 (3.18)	-4.54	<.001 ***	[-21.21—8.26]
Amygdala.volume × Gender	-360 (100.83)	-3.56	<.001 ***	[-591.12— -173]
CAN.net × IGT (Direct effect)	0.06 (0.15)	0.38	0.70	[-0.28—0.30]
IGT × Age	-0.06 (0.14)	-0.46	0.64	[-0.39—0.14]
IGT × Gender	-9.31 (4.31)	-2.16	0.03	[-18.39—0.22]
Indirect effect	-0.004 (0.013)	-0.28	0.78	[-0.05—0.01]
Total effect (Direct + Indirect)	0.05 (0.15)	0.36	0.72	[-0.31—0.30]
Mediated proportion (Indirect/Total)	-0.07 (0.83)	-0.08	0.94	[-9.72—0.01]

Note. THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = Iowa Gambling Task; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Table 7. Model Coefficients for the Association Between Adverse Childhood Experiences, Lifespan Trauma Exposure, and the Reading the Mind in the Eyes Test (RMET) as Mediated by Total Amygdala Volume.

	B (*SE*)	***z*-value**	***p*-value**	**95% CI**
Lifespan trauma exposure				
Amygdala.volume × THQ[a]	6.76 (7.38)	0.92	0.36	[-8.86—23.58]
Amygdala.volume × RMET	0.001 (0.001)	2.07	0.04 *	[0.00—0.003]
Amygdala.volume × Age	-15.95 (2.64)	-6.04	<.001 ***	[-22.08— -11.21]
Amygdala.volume × Gender	-369.63 (83.92)	-4.41	<.001 ***	[-514.83— -158.1]
THQ.net × RMET (Direct effect)	0.31 (0.11)	2.86	0.004 ***	[0.10—0.53]
RMET × Age	0.01 (0.05)	0.25	0.81	[-0.07—0.11]
RMET × Gender	0.58 (1.10)	0.53	0.60	[-1.86—2.51]
Indirect effect	0.01 (0.01)	0.72	0.47	[-0.006—0.06]
Total effect (Direct + Indirect)	0.32 (0.11)	2.86	0.004 ***	[0.10—0.54]
Mediated proportion (Indirect/Total)	0.03 (0.05)	0.57	0.57	[-0.03—0.22]
ACEs				
Amygdala.volume x CAN[b]	1.31 (3.22)	0.41	0.68	[-5.08—7.49]
Amygdala.volume x RMET	0.001 (0.001)	1.25	0.21	[-0.001—0.002]
Amygdala.volume × Age	-14.58 (3.12)	-4.68	<.001 ***	[-19.71— -7.10]
Amygdala.volume × Gender	-358.21 (92.73)	-3.86	<.001 ***	[-504.42— -118]
CAN.net × IGT (Direct effect)	0.02 (0.04)	0.55	0.59	[-0.05—0.10]
RMET × Age	0.05 (0.05)	1.003	0.32	[-0.05—0.16]
RMET × Gender	0.06 (1.09)	0.06	1.00	[-2.03—2.10]
Indirect effect	0.001 (0.004)	0.34	0.73	[-0.004—0.01]
Total effect (Direct + Indirect)	0.02 (0.04)	0.58	0.56	[-0.05—0.10]
Mediated proportion (Indirect/Total)	0.05 (1.00)	0.06	1.00	[-0.08—3.04]

Note. THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = Iowa Gambling Task; *p < .05, **p < .01, ***p < .005.

c. Total number of lifespan traumatic experiences obtained from the THQ.
d. A composite score obtained from the CAN questionnaire subscale.

Moderation Analyses

The primary multiple linear regression models were extended by including interaction terms of the independent variables and the moderator variables for investigating whether stimulant dependence and diagnosis of psychotic illness will moderate the association between ACEs or lifespan trauma and social cognitive performance. A summary of the results is outlined below in Tables 8 and 9. The coefficient estimates were not statistically significant for any of the interaction terms, including exposure to trauma and stimulant dependence, exposure to trauma and diagnosis of psychotic illness, ACEs and stimulant dependence, as well as ACEs and psychotic diagnosis. Therefore, the association between social cognition and both exposure to trauma and ACEs is not significantly moderated by stimulant dependence or diagnosis of psychotic illness. In all four models, education was the only estimated coefficient that was statistically significant.

Table 8. Coefficients for the Moderation Models Between the Adverse Childhood Experiences, Lifespan Trauma Exposure, and the Iowa Gambling Task (IGT).

	B (*SE*)	*t*-value	*p*-value	95% CI
Lifespan trauma exposure				
Intercept	-12.06 (12.32)	-0.98	0.33	[-36.28—12.16]
THQ[a]	0.93 (0.88)	1.06	0.29	[-0.79—2.65]
Age	-0.12 (0.14)	-0.81	0.42	[-0.40—0.17]
Gender	-6.88 (4.00)	-1.72	0.09	[-14.74—0.98]
Education	1.43 (0.71)	2.01	0.05 *	[0.03—2.83]
Stimulant Dependence	-4.39 (8.62)	-0.51	0.61	[-21.34—12.56]
Psychotic Diagnosis	-7.60 (7.45)	-1.02	0.31	[-22.24—7.04]
THQ.net × Stimulant Dependence	0.00 (0.92)	0.00	1.00	[-1.81—1.82]
THQ.net × Psychotic Diagnosis	0.27 (0.79)	0.35	0.73	[-1.27—1.82]
ACEs				
Intercept	-6.77 (15.21)	-0.44	0.66	[-36.71—23.16]
CAN[b]	-0.24 (0.35)	-0.67	0.50	[-0.93—0.46]
Age	-0.02 (0.16)	-0.14	0.89	[-0.34—0.29]
Gender	-8.17 (4.65)	-1.76	0.08	[-17.33—0.98]
Education	1.58 (0.85)	1.87	0.05 *	[-0.09—3.25]

Stimulant Dependence	-5.86 (9.31)	-0.63	0.53	[-24.18—12.46]
Psychotic Diagnosis	-11.33 (7.75)	-1.46	0.14	[-26.57—3.91]
CAN.net × Stimulant Dependence	0.15 (0.36)	0.42	0.67	[-0.56—0.87]
CAN.net × Psychotic Diagnosis	0.38 (0.32)	1.20	0.23	[-0.24—1.00]

Note. THQ x IGT, $n = 418$; CAN x IGT $n = 308$; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = IOWA Gambling Task; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Table 9. Coefficients for the Moderation Models Between Adverse Childhood Experiences, Lifespan Exposure to Trauma, and the Reading the Mind in the Eyes Test (RMET).

	B (*SE*)	***t*-value**	***p*-value**	**95% CI**
Lifespan trauma exposure				
Intercept	16.44 (3.62)	4.54	<.001 ***	[9.29—23.58]
THQ[b]	0.12 (0.21)	0.58	0.56	[-0.30—0.55]
Age	-0.02 (0.04)	-0.53	0.60	[-0.11—0.06]
Gender	0.5 (1.04)	0.47	0.64	[-1.56—2.54]
Education	0.5 (0.19)	2.48	0.01 **	[0.10—0.84]
Stimulant Dependence	-0.64 (2.48)	-0.26	0.80	[-5.54—4.26]
Psychotic Diagnosis	-2.8 (1.93)	-1.44	0.15	[-6.58—1.03]
THQ.net × Stimulant Dependence	0.11 (0.24)	0.44	0.66	[-0.37—0.59]
THQ.net × Psychotic Diagnosis	0.16 (0.20)	0.80	0.43	[-0.23—0.54]
ACEs				
Intercept	11.30 (3.92)	2.88	0.005 **	[3.56—19.04]
CAN[b]	0.15 (0.09)	1.74	0.08	[-0.02—0.32]
Age	0.02 (0.05)	0.43	0.67	[-0.07—0.11]
Gender	0.19 (1.11)	0.17	0.86	[-2.00—2.38]
Education	0.57 (0.20)	2.91	0.004 **	[0.18—0.96]
Stimulant Dependence	3.19 (2.32)	1.38	0.17	[-1.39—7.77]
Psychotic Diagnosis	-0.09 (1.82)	-0.05	0.96	[-3.69—3.51]
CAN.net × Stimulant Dependence	-0.12 (0.09)	-1.38	0.17	[-0.30—0.05]
CAN.net × Psychotic Diagnosis	-0.04 (0.07)	-0.54	0.59	[-0.18—0.10]

Note. THQ × RMET, $n = 190$; CAN × RMET, $n = 160$; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; RMET = Reading the Mind in the Eyes Test; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Figure 3. Scatterplot of the Relationship Between Adverse Childhood Experiences (ACEs), Lifespan Trauma Exposure, and the Iowa Gambling Task (IGT).

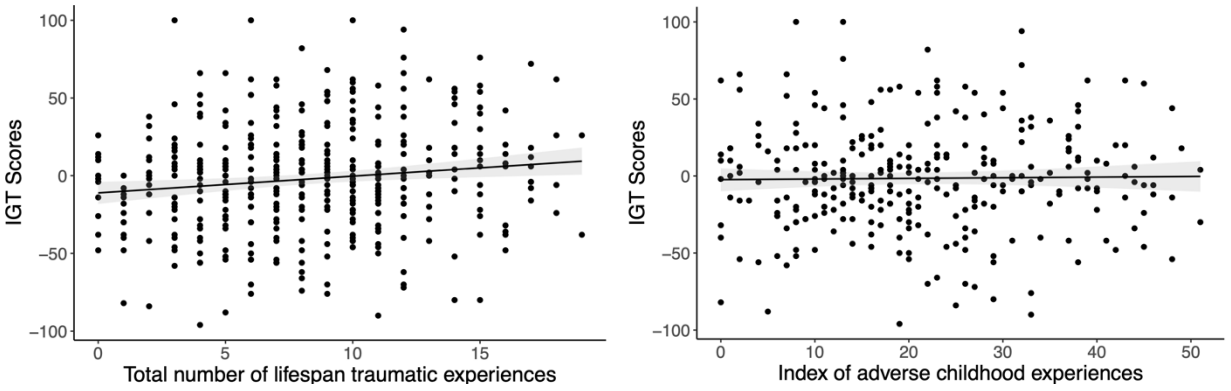


Figure 4. Scatterplot of the Relationship Between Adverse Childhood Experiences (ACEs), Lifespan Trauma Exposure, and the Reading the Mind in the Eyes (RMET) Test.

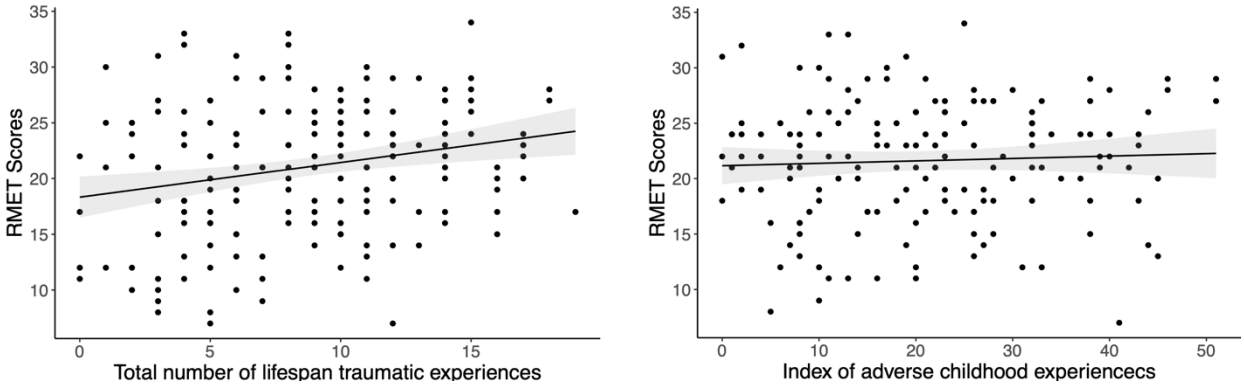
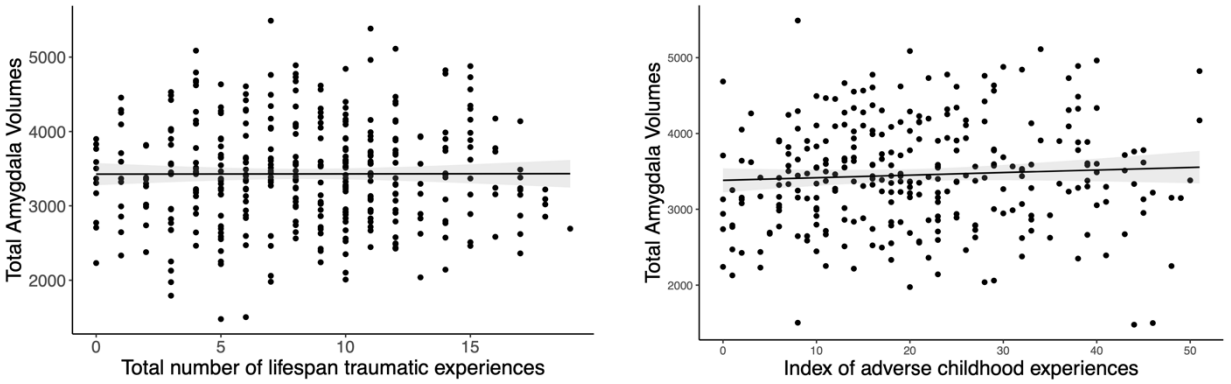


Figure 5. Scatterplot of the Relationship Between Adverse Childhood Experiences (ACEs), Lifespan Trauma Exposure, and Total Amygdala Volume.



Discussion

The primary goal of this study was to examine the impact of adverse childhood experiences (ACEs) and lifespan trauma exposure on social cognitive functioning in the domains of affective decision-making and theory-of-mind (ToM) in a sample of precariously housed adults. Although most of the existing evidence suggests that childhood adversity and trauma exposure throughout life are associated with poorer performance in a wide range of cognitive domains (Honzel et al., 2014; Lynch & Lachman, 2020; Polak et al., 2012), the results of this study do not align with this finding. In contrast to what was hypothesized, the results revealed that a greater total number of traumatic experiences throughout one's life was associated with better performances on affective decision-making and ToM tasks. Furthermore, the findings demonstrated that ACEs were not significantly associated with social cognitive abilities in adulthood. Contrary to what was predicted, ACEs and lifespan trauma were not associated with amygdala volume. Moreover, amygdala volume did not mediate the relationship between early adversity and lifespan trauma and performances on the social cognitive measures as expected. Finally, the predicted moderating effect of psychotic illness and stimulant dependence on the relationship between ACEs, lifespan trauma, and social cognitive abilities was also not detected.

One explanation for the unexpected finding of the positive association between lifespan trauma exposure and affective decision-making performance in the context of risk and reward could be attributed to the connection between traumatic experiences and risk-taking behaviours. Emerging evidence suggests that individuals who have experienced traumatic events or adverse life circumstances may tend to engage in fewer risky behaviours over the long term (Augsburger & Elbert, 2017). For instance, the Uncertainty Management Model proposed by Amir et al. (2018) suggests that adverse life experiences, such as experiencing low socioeconomic status,

can shape individuals' inclination to adopt a specific set of preferences aimed at minimizing the potential negative consequences associated with uncertainty. According to this model, individuals who have encountered such adversity often display a risk-averse attitude when confronted with uncertainty, opting for a more cautious and conservative decision-making style. Moreover, studies have indicated that individuals who have undergone traumatic experiences tend to display heightened fear responses characterized by increased sensitivity to perceived threats and elevated physiological arousal (Glover et al., 2011; Gonzalez & Martinez, 2014). This heightened fear response is often viewed as an adaptive mechanism that emerges following trauma, aiming to protect an individual's well-being in uncertain and risky situations. This adaptive response can manifest as heightened risk aversion, leading individuals to exhibit increased caution and a tendency to avoid uncertain or potentially harmful choices and situations (Huh et al., 2016). Therefore, it can be theorized that prior trauma exposure may lead to risk-averse behaviours, which result in enhanced decision-making in situations involving risk and uncertainty, such as those encountered in the IGT.

A potential explanation as to why trauma exposure might be associated with better ToM abilities is that individuals who have experienced trauma may develop a heightened sensitivity to social cues, characterized by a state of hypervigilance (Sadeghi et al., 2022; Weston, 2014). This adaptive hypersensitivity can potentially enable the trauma-exposed individuals to be more perceptive of subtle social signals and nonverbal communication, thereby improving their capacity to interpret other people's facial expressions and emotional states (Kraaijenvanger et al., 2020). Accumulating evidence from neuroimaging studies offers additional support for the hypervigilance associated with ACEs and lifespan exposure to trauma (McCrory et al., 2011; Suzuki et al., 2014; Tiecher & Samson, 2016). Studies have consistently reported functional

alterations in the amygdala, a key brain region involved in emotion and threat processing, in response to fear-related stimuli in individuals with a history of childhood adversity and lifespan trauma (Hein and Monk, 2017; Teicher and Samson, 2016). Given the significance of facial expressions in non-verbal communication during social interactions, as well as the crucial role of the amygdala in emotion and threat processing, it has been suggested that the observed amygdala hyperactivity may signify an increased sensitivity and awareness toward potential social threats (Kraaijenvanger et al., 2020). Indeed, a recent review conducted by Stevens and Jovanovic (2020) revealed that the widely observed trauma-related amygdala hyperactivity is mainly reported with regard to facial cues as opposed to other types of stimuli. Therefore, it is reasonable to speculate that the amygdala's adaptive hyperresponsiveness to emotionally-valenced facial expressions could potentially contribute to the enhanced ToM abilities observed in trauma-exposed individuals.

An alternative explanation for the observed positive association between lifespan trauma exposure and outcomes related to social cognitive functioning could be related to the unique characteristics of the sample used in this study. It is plausible to consider that the highly challenging living conditions faced by the precariously housed individuals have provided unique opportunities for the cultivation of resilience, which, in turn, may alleviate the adverse effects of traumatic experiences. Indeed, studies have shown that people who are exposed to stressful conditions are more likely to develop adaptive coping mechanisms (Ellis et al., 2017). These coping skills encompass a range of cognitive and behavioural strategies that individuals employ to manage stressors and mitigate associated emotional distress effectively (Holahan et al., 2017). Thus, through repeated exposure to stressors, individuals can acquire new problem-solving strategies, cultivate resilience, and construct a repertoire of adaptive skills that enable them to

effectively navigate and thrive in their challenging environments (Ellis et al., 2017). Therefore, within this context, traumatic experiences can be theorized to have multifaceted effects, functioning both as a risk factor and a potential protective factor that shapes the development of adaptive skills and traits (Dunn, 2018). Ellis et al. (2020) describe these skills as "hidden talents" since they have been largely overlooked within the traditional deficit-based frameworks utilized to study the effects of adverse experiences. These hidden and often untapped talents hold significant potential for understanding and promoting positive outcomes and the resilience that can emerge in individuals who face adverse experiences (Ellis et al., 2020).

Another major finding of this study was the lack of association between ACEs and social cognitive performance within the precariously housed sample. This does not align with a bulk of previous research which has consistently documented that childhood adversity is associated with deficits in social cognitive domains (e.g., Bora et al., 2015; Germine et al., 2015; Luke et al., 2013; Mrizak et al., 2016; Nazarov et al., 2014; Rokita et al., 2021; Seitz et al., 2022; Stevens & Jovanovic, 2018). A potential explanation for the non-significance finding could be attributed to the complex nature of childhood adversity. Numerous factors contribute to the long-lasting and pervasive effects of ACEs on individuals' life trajectories. The multifaceted nature of ACEs encompasses various dimensions, such as the type and severity of the experiences, the duration and frequency of exposure, the age at which they occur, and the presence of protective factors or support systems. These factors interact in complex ways, shaping the developmental pathways and outcomes for those who have experienced ACEs in distinct ways. For instance, some researchers have put forward a theoretical proposition suggesting the existence of sensitive periods during which the impacts of childhood adversity on cognition are especially influential. This concept suggests that there may be differential effects of ACEs based on the age at which

the person has experienced adversity (Dunn et al., 2016; Hambrick et al., 2019). Others have suggested the importance of the onset and the duration, or the chronicity, of adverse experiences in shaping the outcomes (Cowell et al., 2016). Taken together, this evidence may help explain the lack of association between ACEs and social cognitive performance found in this study. Given the inherent complexities and intricacies associated with adverse experiences, such as the multitude of factors influencing their manifestation and the subjective nature of their impact, it may be difficult to detect a combined effect, particularly within the context of the precariously housed sample characterized by heterogeneous histories of adversity. Future research should seek to clarify the differential effect of the individual variations in the onset, duration, and severity of ACEs on social cognitive outcomes.

While previous research has highlighted the importance of adverse early experiences (e.g., childhood maltreatment) and their link with marked alterations in the developing brain (Lim et al., 2020; Paquola et al., 2016; Teicher et al., 2016), the present study, on the contrary, did not find any significant association between ACEs and lifespan trauma exposure with the amygdala volume. Additional findings also indicated that the volume of the amygdala does not mediate the relationship between ACEs, lifespan trauma exposure, and social cognitive outcomes. Given its central role in the processing of emotional and fear-related stimuli, as well as its involvement in the modulation of physiological responses related to stress and emotional arousal, the amygdala has received considerable attention in trauma-related research. However, In addition to the amygdala, other essential components of the fronto-limbic neural system, including the hippocampus, anterior cingulate cortex (ACC), and certain regions of the prefrontal cortex (PFC), also play a significant role in emotion processing (Braun, 2011). Numerous studies have documented diffused structural anomalies related to lifespan trauma and childhood

maltreatment across several brain regions, including the hippocampus, ACC, and regions of the PFC (Ahmaed-Leito et al., 2016; Cassiers et al., 2018; Gorka et al., 2014; Kühn & Gallinat, 2013; Li et al., 2020; Lim et al., 2014; Pollok et al., 2022). These observations could potentially help elucidate the lack of significant findings concerning the association between ACEs, lifetime trauma, and the volume of the amygdala. It is plausible that the complex interplay of risk factors and multiple comorbidities within the precariously housed population could contribute to more diffuse and extensive brain atrophy rather than distinct and localized alterations in isolated brain regions. Furthermore, it is also probable that the effects of trauma and maltreatment on the brain can manifest in diverse ways beyond simple localized or global reductions in grey matter. Research has documented alterations in white matter integrity and structural connectivity (Sieh et al., 2018; Goetschius et al., 2020; Lim et al., 2020), disruptions in functional connectivity, particularly within the frontal-limbic circuitry (Teicher & Khan, 2019; Ross et al., 2021; Luo et al., 2022), heightened neural reactivity in response to emotional stimuli (Stark et al., 2015; Scot et al., 2015; Teicher et al., 2016; Kraaijenvanger et al., 2020), as well as hypoactivity in regions of the medial prefrontal cortex (Patel et al.., 2012; Heany et al., 2018; Henigsberg et al., 2019) associated with ACEs and lifespan trauma exposure. In addition, research conducted in both animal and human studies has revealed alterations in neurotransmitter activities, specifically within the dopaminergic system (Aliev et al., 2020; Bloomfield et al., 2019; Egerton et al., 2016; Sherin & Nemeroff, 2011). Thus, it is reasonable to propose that the impact of ACEs and lifetime trauma on the brain may manifest in various ways that extend beyond the scope of the neuroimaging techniques employed in this study. Exploring these additional dimensions can help elucidate the full extent of the impact of adverse experiences on the brain's structure and function.

The examination of the influence of psychotic illnesses and stimulant dependence on the association between childhood adversity, lifespan trauma, and social cognition yielded no significant moderating effects. This suggests that the relationship between childhood adversity, lifespan trauma, and social cognitive functioning may operate independently of the presence of psychotic illnesses and stimulant dependence. This finding is especially intriguing, considering the extensive body of research that has consistently established a robust association between psychotic diagnoses (Fioravanti et al., 2012; McCleery & Nuechterlein, 2019; Schaefer et al., 2013; Sheffield et al., 2018; Van Rheenen et al., 2019), substance use (Pottvin et al., 2014; Ramey & Regier 2019; Zhong et al., 2016), and cognitive dysfunction across a wide array of domains. This holds particular relevance for precariously housed persons, given the strikingly high prevalence of substance dependence and psychotic illness among this population. Studies have estimated that nearly 95% of people living in precarious housing use some form of substance, and nearly half are affected by psychotic disorders (e.g., Edidin et al., 2012; Gutwinski et al., 2021; Hossain. Et al., 2020).

It is crucial to recognize that, despite the positive correlation found between the number of lifespan traumatic experiences and social cognitive performances in this study, individuals who have experienced trauma continue to face a multitude of cognitive and psychosocial challenges in their daily lives. Previous research has consistently shown that trauma can have enduring impacts on various aspects of functioning, including cognitive abilities, emotional regulation, interpersonal relationships, and overall well-being. Thus, the observed positive correlation does not negate the significant difficulties these individuals continue to face, underscoring the need for comprehensive support and interventions to address the multifaceted impact of trauma on their well-being. This is especially significant for the precariously housed

population, given their highly challenging living environment. The experience of precarious housing introduces additional stressors, such as financial instability, limited access to healthcare and support services (Argintaru et al., 2013), social isolation, and increased exposure to violence and victimization (Turner et al., 2018; Perron et al., 2008). These cumulative risk factors can magnify the effects of trauma, heightening the cognitive, emotional, and social challenges faced by individuals within this population.

Limitations and Future Directions

The present study should be interpreted considering certain limitations. Firstly, the lack of a healthy comparison group is a notable limitation in the present study, which makes it challenging to determine the exact impact of ACEs and lifetime trauma on social cognitive functioning and neuroanatomical alterations. Unfortunately, the recruitment of a well-matched control group that accurately represents middle-aged individuals with low socioeconomic status and limited formal education poses significant challenges. Furthermore, the absence of well-established normative databases makes it difficult to assess the extent to which the observed findings deviate from the norm. Without reliable normative data for comparison, it becomes challenging to determine whether the observed social cognitive functioning and neuroanatomical alterations are within the range of typical variation or if they represent significant deviations. Lastly, it is essential to acknowledge that due to the observational nature of the current study, we are unable to draw any definitive conclusions about casual associations. Future evidence from multiple lines of research, such as prospective studies, randomized controlled trials, longitudinal studies, as well as experimental studies conducted with animal models, will enable a more comprehensive understanding of the complex mechanisms and causal pathways in the link between trauma, social cognition, and neuroanatomical outcomes.

Secondly, the indices of adverse childhood experiences and lifetime trauma exposure were captured using retrospective self-report measures. Despite the wealth of information they provide, self-reporting methods may be susceptible to biases that could affect the validity and reliability of the data. There is a potential for social desirability bias, memory recall limitations and errors, lack of objectivity, and limited verifiability of the reporting (Althubaiti, 2016). This is particularly pertinent when collecting data from adults regarding events from their childhood, as it introduces the potential for increased recall biases. Furthermore, research has demonstrated that the experience of a highly traumatic event like sexual assault can profoundly impact an individual's memory processes, often resulting in alterations in how memories are encoded, stored, and retrieved (Greenhoot & Sun, 2014). Thus, while the self-report measures such as the THQ and CAN questionnaires utilized in this study are often the best available approach, future studies could overcome the limitation of this study by incorporating information from multi-informant sources (e.g., caregiver accounts, government's child protection services documentation, as well as police report for official records of significant events or involvement).

Thirdly, it is essential to acknowledge potential limitations related to the outcome measures employed in this study. The Iowa Gambling Task (IGT) is one of the most widely used paradigms in the study of affective decision-making in the context of risk and reward. However, poor psychometric characteristics have always posed a barrier to using traditional analyses of behavioural data, such as those provided by the IGT (Schmitz et al., 2020). In the case of the IGT, one of the major challenges is the lack of specificity. Although the IGT is highly sensitive for detecting decision-making difficulties, due to the complexity of the task, the behavioural performance data alone does not provide sufficient information to identify the specific underlying neurocognitive processes responsible for the observed decision-making deficits

(Haines et al., 2018). To address the lack of specificity of the net-score analysis of the IGT, several computational models have been proposed that aim to isolate and identify the distinct psychological processes that drive performance on the task (Ahn et al., 2008; Busemeyer & Stout, 2002; d'Acremont et al., 2009; Steingroever et al., 2013; Worthy et al., 2013). An example of a more novel and improved model is the Outcome-Representation Learning (ORL) model proposed by Haines et al. (2018). The ORL is a reinforcement learning model that aims to break down IGT behavioural data into four distinct cognitive strategies that are consistently observed. Future research could incorporate these models into their analyses to overcome the limitations associated with relying solely on routine total score metrics. This integration would result in a deeper understanding of the specific cognitive processes underlying decision-making deficits, particularly in situations involving risk and uncertainty, and may have a clearer association with underlying neuroanatomical volumes, including the amygdala. Furthermore, an important limitation that needs to be considered regarding the Reading the Mind in the Eyes Test (RMET) is its high reliance on language abilities. The RMET assesses individuals' ability to infer emotions and mental states from photographs of the eye region, primarily through the interpretation of subtle facial cues. However, since the test heavily relies on verbal understanding and interpretation, individuals with language difficulties or limited proficiency may encounter challenges in accurately comprehending and responding to the test items. It is important to highlight that in order to address this limitation to some extent, a modified version of the RMET was utilized in this study. In this modified version, participants were provided with word definitions if needed, aiming to compensate for any potential language-related difficulties and limited vocabulary. However, it is crucial to acknowledge that this modified version of the test should be considered as a mitigating factor rather than a complete resolution to the language-

related limitation. Therefore, it is important to interpret the findings of the study with caution, recognizing the potential influence of language abilities on performance on the test. Future investigations could address this limitation by exploring alternative measures that are less dependent on language abilities and would allow more inclusive assessments for individuals with limited proficiency. Additionally, researchers could investigate the use of technology-based assessments, such as eye-tracking or reaction time measures, to analyze the processing of social cues and facial expressions without the need for explicit language interpretations.

Finally, limitations related to invalid and missing data should also be considered when interpreting the findings of this study. Despite efforts to collect comprehensive data, it is important to acknowledge there was a higher proportion of missing data for the CAN questionnaire assessing adverse home environments, as well as the RMET used to measure ToM abilities. The presence of missing data introduces the possibility of bias and may limit the generalizability of the study's results. Nevertheless, it is worth highlighting that the results obtained from analyses incorporating imputed missing data and those conducted solely on the original data were found to be comparable. This suggests that the imputation of missing data did not substantially influence the overall findings and conclusions of the study.

Implications

The outcomes of this study have several important real-world implications. The findings can provide valuable guidance for the development of targeted interventions and the improvement of health service delivery in the vulnerable precariously housed population, who face numerous impediments in accessing and utilizing such services (Argintaru et al., 2013). Firstly, the study underscores the limitations of a deficit-based approach when addressing risk factors and highlights the importance of adopting a strengths-based perspective. The

conventional deficit-based approach often focuses solely on identifying weaknesses or shortcomings and inadvertently overlooks the remarkable resilience and coping capacities of individuals who have endured adversity. Another significant takeaway from our research challenges the prevailing narrative surrounding trauma and contributes to mitigating the stigma associated with trauma survivors. While trauma is often linked solely to negative consequences, our findings serve to highlight a more nuanced perspective that recognizes the multifaceted nature of trauma's impact, suggesting that its effects can encompass a wide spectrum of outcomes, some of which may be unexpectedly positive or growth-oriented. Lastly, raising public awareness about the potential for positive outcomes can serve as a wellspring of inspiration, motivating individuals to actively seek support and uncover untapped reservoirs of strength within their own resilience.

Conclusion

In summary, this study has shed light on the complex and multifaceted associations between adverse childhood experiences (ACEs), lifespan trauma, and their impact on social cognitive functioning and amygdala volume in a sample of precariously housed adults. The results revealed that early childhood environments did not show a significant association with social cognitive functioning during adulthood; however, lifespan traumatic exposure demonstrated a positive impact on ToM and affective decision-making abilities. Importantly, these associations were not mediated by amygdala volume and did not vary in the context of psychosis or stimulant use. These findings underscore the significance of considering the role of the protective and resilience factors, in addition to the risk exposures in shaping individual trajectories. Future research is warranted to explore the underlying mechanisms through which negative experiences could contribute to developing resilience and serve as a protective buffer

against adverse outcomes. This line of inquiry will contribute to the development of targeted interventions aimed at promoting positive adaptation and well-being in the face of challenging life circumstances, especially for vulnerable populations such as precariously housed individuals who face a multitude of challenges and risk exposures.

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Appendix A. THQ and CAN subscale multiple regression coefficients.

Regression models were fitted for each subscale individually. Based on invalid and/or missing data for different subscales, different models may have had distinct sample sizes, as noted in the tables. In the social cognitive performance models with the IGT and RMET as the outcome variables, age, gender, and education were included as the covariates. In the brain measure model with the amygdala volume as the outcome variable, only age and gender were included as the covariates.

Table A1. Model Coefficients for the Association Between the CAN Questionnaire and THQ Subscales and the Iowa Gambling Task (IGT).

	N	B (SE)	t-value	p-value	95% CI
THQ					
Physical	411	2.38 (1.66)	1.44	0.15	[-0.88—5.64]
Sexual	414	0.37 (1.67)	0.22	0.82	[-2.90—3.65]
Crime		2.94 (1.29)	2.28	0.02 *	[0.40—5.49]
General		1.79 (0.65)	2.74	0.006 **	[0.51—3.08]
CAN					
Neglect		0.01 (0.34)	0.04	1.00	[-0.67—0.70]
Family Turmoil		-0.17 (1.00)	-0.18	0.86	[-2.06—1.72]
Parental Intimidation		0.48 (0.43)	1.13	0.26	[-0.36—1.32]
Parental Violence		0.25 (0.82)	0.31	0.76	[-1.36—1.87]
Poverty		-0.16 (0.93)	-0.18	0.86	[-20—1.66]

Note. THQ subscales, $n = 418$ unless otherwise specified; CAN subscales, $n = 308$; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = IOWA Gambling Task; *p < .05, **p < .01, ***p < .005.

Table A2. Model Coefficients for the Association Between the CAN Questionnaire and THQ Subscales and the Reading the Mind in the Eyes Test (RMET).

	N	B (SE)	t-value	p-value	95% CI
THQ					
Physical	188	0.46 (0.43)	1.08	0.28	[-0.38—1.30]
Sexual	190	1.06 (0.48)	2.20	0.03 *	[0.11—2.01]
Crime		0.99 (0.33)	3.04	0.003 **	[0.35—1.64]
General		0.35 (0.15)	2.29	0.02 *	[0.05—0.64]
CAN					
Neglect		0.02 (0.08)	0.22	0.83	[-0.14—0.17]
Family Turmoil		0.28 (0.22)	1.31	0.19	[-0.14—0.71]
Parental Intimidation		0.16 (0.10)	1.69	0.09	[-0.03—0.35]
Parental Violence		0.18 (0.19)	0.95	0.34	[-0.19—0.55]
Poverty		-0.15 (0.21)	-0.72	0.47	[-0.57—0.26]

Note. THQ subscales, *n* = 190 unless otherwise specified; CAN subscales, *n* = 160; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; RMET = Reading the Mind in the Eyes Test; *p < .05, **p < .01, ***p < .005.

Table A3. Model Coefficients for the Association Between the CAN Questionnaire and THQ Subscales and the Total Amygdala Volume.

	N	B (SE)	t-value	p-value	95% CI
THQ					
Physical	379	7.74 (32.41)	0.24	0.81	[-56—71.46]
Sexual	380	8.42 (33.24)	0.25	0.80	[-56.94—73.78]
Crime		20.00 (25.91)	0.77	0.44	[-31—70.91]
General		11.24 (12.88)	0.87	0.38	[-14.08—36.56]
CAN					
Neglect		-2.28 (7.26)	-0.32	0.75	[-16.57—12.002]
Family Turmoil		16.63 (19.86)	0.84	0.40	[-22.46—55.73]
Parental Intimidation		10.40 (8.89)	1.17	0.24	[-7.10—27.91]
Parental Violence		11.69 (17.24)	0.68	0.50	[-22.25—45.62]
Poverty		-15.95 (19.11)	-0.84	0.40	[-53.55—21.66]

Note. THQ subscales, *n* = 384 unless otherwise specified; CAN subscales, *n* = 290; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; *p < .05, **p < .01, ***p < .005.

Appendix B. Multiple regression coefficients of the primary analyses conducted using the imputed data.

Table B1. Model Coefficients for the Association Between the Adverse Childhood Experience, Lifespan Trauma Exposure, and the Iowa Gambling Task (IGT) Using the Imputed Dataset.

	B (SE)	t-value	p-value	95% CI
Lifespan trauma exposure				
Intercept	-16.37 (9.07)	-1.81	0.07	[-0.06—1.45]
THQ[a]	0.65 (0.36)	1.80	0.07	[-0.06—1.35]
Age	-0.06 (0.13)	-0.47	0.64	[-0.31—0.19]
Gender	-2.01 (3.55)	-0.57	0.57	[-8.98—5]
Education	1.16 (0.64)	1.80	0.07	[-0.10—2.43]
ACEs				
Intercept	-11.9 (9.60)	-1.24	0.22	[-30.71—7]
CAN[b]	-0.05 (0.13)	-0.37	0.71	[-0.31—0.21]
Age	-0.04 (0.13)	-0.28	0.78	[-0.29—0.21]
Gender	-1.82 (3.56)	-0.51	0.61	[-8.82—5.18]
Education	1.24 (0.64)	1.92	0.06	[-0.03—2.50]

Note. N = 481; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = Iowa Gambling Task; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Table B2. Model Coefficients for the Association Between the Adverse Childhood Experience, Lifespan Trauma Exposure, and the Reading the Mind in the Eyes Test (RMET) Using the Imputed Dataset.

	B (SE)	t-value	p-value	95% CI
Exposure to trauma				
Intercept	13.15 (1.6)	8.42	<.001 ***	[10.08—16.21]
THQ[a]	0.37 (0.06)	5.96	<.001 ***	[0.25—0.49]
Age	-0.02 (0.02)	-0.71	0.48	[-0.06—0.03]
Gender	1.26 (0.61)	2.05	0.04 *	[0.05—2.5]
Education	0.51 (0.11)	4.56	<.001 ***	[0.29—0.72]
ACEs				
Intercept	15.36 (1.71)	9.00	<.001 ***	[12.01—18.71]
CAN[b]	-0.01 (0.024)	-0.61	0.54	[-0.06—0.03]
Age	-0.0006 (0.02)	-0.03	0.98	[-0.05—0.04]
Gender	1.35 (0.63)	2.13	0.03 *	[0.10—2.59]
Education	0.55 (0.11)	4.81	<.001 ***	[0.33—0.78]

Note. N = 481; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; RMET = Reading the Mind in the Eyes Test; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Table 3B. Model Coefficients for the Association Between the Adverse Childhood Experience (ACEs), Lifespan Trauma Exposure, and the Total Amygdala Volume Using the Imputed Dataset.

	B (SE)	t-value	p-value	95% CI
Exposure to trauma				
Intercept	4127.55 (123.57)	33.40	<.001 ***	[3884.75—4370.35]
THQ[a]	4.23 (7.17)	0.59	0.56	[-9.9—18.31]
Age	-13.23 (2.55)	-5.18	<.001 ***	[-18.25—-8.22]
Gender	-365.73 (70.94)	-5.16	<.001 ***	[-505.12—-226.35]
ACEs				
Intercept	4103.60 (133.27)	30.79	<.001 ***	[3841.69—4365.43]
CAN.net[b]	2.01 (2.68)	0.75	0.45	[-3.25—7.28]
Age	-12.86 (2.54)	-5.06	<.001 ***	[-17.86— -7.86]
Gender	-367.26 (70.97)	-5.17	<.001 ***	[-506.72— -227.80]

Note. $N = 481$; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; *p < .05, **p < .01, ***p < .005.

a. Total number of lifespan traumatic experiences obtained from the THQ.
b. A composite score obtained from the CAN questionnaire subscale.

Table B4. Model Coefficients for the Association Between the CAN Questionnaire and THQ Subscales and the Iowa Gambling Task (IGT) Using the Imputed Dataset.

	B (SE)	t-value	p-value	95% CI
THQ				
Physical	1.68 (1.49)	1.13	0.26	[-1.25—4.62]
Sexual	-0.42 (1.56)	-0.27	0.79	[-3.48—2.64]
Crime	2.07 (1.20)	1.72	0.09	[-0.30—4.44]
General	1.108 (0.60)	1.80	0.07	[-0.10—2.26]
CAN				
Neglect	-0.24 (0.3)	-0.84	0.40	[-0.79—0.31]
Family Turmoil	0.09 (0.78)	0.11	0.91	[-1.44—1.61]
Parental Intimidation	0.13 (0.34)	0.38	0.71	[-0.54—0.79]
Parental Violence	-0.53 (0.65)	-0.81	0.42	[-1.82—0.75]
Poverty	0.11 (0.71)	0.16	0.88	[-1.28—1.50]

Note. $N = 481$; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; IGT = IOWA Gambling Task; *p < .05, **p < .01, ***p < .005.

Table B5. Model Coefficients for the Association Between the CAN Questionnaire and THQ Subscales and the Reading the Mind in the Eyes Test (RMET) Using the Imputed Dataset.

	B (SE)	t-value	p-value	95% CI
THQ				
Physical	0.48 (0.27)	1.78	0.07	[-0.04—1]
Sexual	0.71 (0.28)	2.57	0.01 *	[-0.17—1.25]
Crime	1.18 (0.21)	5.69	<.001 ***	[0.77—1.59]
General	0.55 (0.10)	5.30	<.001 ***	[0.35—0.75]
CAN				
Neglect	-7.9×10^{-2} (5×10^{-2})	-1.42	0.16	[-0.17—0.03]
Family Turmoil	0.10 (0.14)	0.71	0.48	[-0.17—0.37]
Parental Intimidation	-1.7×10^{-5} (6.01×10^{-2})	0.00	1.00	[-0.12—0.12]
Parental Violence	0.18 (0.12)	1.55	0.12	[-0.05—0.41]
Poverty	-0.25 (0.13)	-1.96	0.05	[-0.49—8.91×10^{-5}]

Note. *N* = 481; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; RMET = Reading the Mind in the Eye. Test; *p < .05, **p < .01, ***p < .005.

Table B6. Model Coefficients for the Association Between the CAN Questionnaire and THQ Subscales and the Total Amygdala Volume Using the Imputed Dataset.

	B (SE)	t-value	p-value	95% CI
THQ				
Physical	7.96 (29.82)	0.27	0.79	[-50.64—66.55]
Sexual	09.52 (31.07)	-0.31	0.76	[-70.58—51.53]
Crime	19.58 (24.00)	0.82	0.41	[-27.57—66.72]
General	7.003 (11.94)	0.59	0.56	[-16.45—30.45]
CAN				
Neglect	-5.91 (5.58)	-1.06	0.29	[-16.88—5.07]
Family Turmoil	37.008 (15.39)	2.40	0.02 *	[6.77—67.25]
Parental Intimidation	9.64 (6.73)	1.43	0.15	[-3.59—22.86]
Parental Violence	17.89 (13.02)	1.37	0.17	[-7.70—43.47]
Poverty	-0.48 (14.14)	-0.03	0.97	[-28.26—27.31]

Note. *N* = 481; THQ = Trauma History Questionnaire; CAN = Childhood Abuse and Neglect Questionnaire; *p < .05, **p < .01, ***p < .005.