



## Research Paper

# The roles of neuroticism and schizotypy in emotional abuse and mental health association: a replication and extension of Alnassar et al. (2024)

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## ABSTRACT

**Background:** Childhood emotional abuse (EA) has been consistently linked to adverse mental health outcomes. Recent data suggest that neuroticism may partially mediate this association but the role of schizotypy which overlaps with neuroticism and also predicts negative emotional experiences remains unknown. This study examined the roles of both neuroticism and schizotypy in the association of EA with poor mental health and sleep quality.

**Methods:** Data were collected from 478 healthy adults (179 males, 299 females) who completed self-report measures of childhood trauma (emotional abuse and neglect, physical abuse and neglect, sexual abuse), mental health (depression, anxiety, stress), sleep quality, and personality traits (neuroticism, schizotypy) in a single session. Structural equation modelling was used to test neuroticism and schizotypy as potential mediators of EA association with poor mental health and sleep quality.

**Results:** All forms of childhood trauma were associated with poor mental health and sleep quality, with EA showing these associations most strongly ( $r: 0.30-0.42$ ). Neuroticism and schizotypy were significantly correlated ( $\beta=0.52$ ) and independently mediated the relationship between EA and poor mental health (neuroticism:  $\beta=0.12$ ; schizotypy:  $\beta=0.11$ ), while only neuroticism mediated the relationship between EA and poor sleep quality ( $\beta=0.12$ ).

**Conclusions:** Both neuroticism and schizotypy mediate EA-mental health association possibly due to poor cognitive control and heightened stress sensitivity, exaggerating maladaptive emotion regulation. Further research should aim to examine underlying cognitive mechanisms (e.g., selective negative recall bias) through which neuroticism and/or schizotypy exert their influence in these associations and develop suitable psychological interventions to target them.

## 1. Introduction

Childhood maltreatment is typically defined as sexual, emotional, or physical abuse and emotional and physical neglect from a caregiver or an unrelated adult (Barnett et al., 1993). Growing evidence suggests the role of childhood maltreatment in the pathogenesis and maintenance of various mental health illnesses, including depression, anxiety, psychosis, post-traumatic stress disorder, and sleep-related disturbances (Aas et al., 2016; Brindle et al., 2018; Dunn et al., 2017; Kuzminskaite et al., 2021; Pandey et al., 2020). While all forms of abuse and neglect are known to cause psychological and biological changes that increase the risk of mental disorders and relapse vulnerability (Norman et al., 2012),

emotional abuse (EA) shows the strongest association with poor mental health (Alnassar et al., 2024; Norman et al., 2012; Pandey et al., 2020).

Recently, Alnassar and colleagues (2024) reported that higher levels of neuroticism may explain the relationship of EA with depression as well as anxiety symptomatology in young healthy adults in a UK sample ( $N = 1116$ ). There is also a considerable body of evidence showing that neuroticism, a personality trait associated with a tendency to emotionally react, worry, and increased likelihood of experiencing negative mood and psychopathology (Claridge and Davis, 2001), is found to be positively correlated with self-reported history of EA across cultures (Hengartner et al., 2015; Hovens et al., 2016; Hu et al., 2025; Li et al., 2014; Schwandt et al., 2013; Wang et al., 2019). Individuals with a

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history of EA may employ psychological defence mechanisms to deal with the incongruence between their internal needs and reality, potentially leading to atypical personality development and higher levels of neuroticism (Li et al., 2014; Wang et al., 2019). This also aligns with the attachment theory (Bowlby, 1969), according to which secure bonding with at least one caregiver is integral for normal personality development. Thus, early life trauma may contribute to maladaptive personality traits, such as neuroticism (Mc Elroy and Hevey, 2014). Early life trauma or maltreatment may also strengthen existing neurotic predispositions (Ogle et al., 2014), and neuroticism in itself is consistently found to be an independent predictor and correlate of psychological distress, various internalising (e.g., depression, anxiety) and externalising disorders (e.g., impulsive control), and psychosis (Krabbendam et al., 2002; Nilsen et al., 2024).

Another personality trait that may be relevant in this context is schizotypy. Schizotypy is phenotypically similar to psychosis (Meehl, 1990), and shows a substantial positive correlation with neuroticism (e.g.,  $r = 0.48$ ; Ettinger et al., 2005). It also has known links with negative emotional experiences (Cohen et al., 2015), for example experiencing rejection sensitivity (i.e., a type of social anxiety marked by fear of rejection in ambiguous interpersonal relationships; Downey and Feldman, 1996; Premkumar et al., 2012, 2015). Similar to neuroticism, the relationship between EA and schizotypy is well-established (Dizinger et al., 2022; Goodall et al., 2015; Toutountzidis et al., 2022). Velikonja and colleagues (2015) reported that EA independently predicted positive and negative symptoms of schizotypy, even after controlling for other forms of childhood trauma, suggesting that EA may be an important environmental contributor to the development of schizotypy (Velikonja et al., 2015). However, the extent to which schizotypy may play a unique role in the EA-mental health relationship is not known.

There are reports of individuals with a history of childhood maltreatment, including EA, experiencing sleep-related disturbances, including shorter sleep duration, issues with maintaining sleep, and delayed sleep onset (Brindle et al., 2018; McWhorter et al., 2019). Poor sleep quality has also found to be associated with neuroticism both in cross-sectional (e.g. Catherman et al., 2023) and longitudinal studies (Stephan et al., 2018), while atypical sleep disturbances have been reported in association with a high level of schizotypy (Koffel and Watson, 2009). The extent to which neuroticism and/or schizotypy might mediate EA-poor sleep quality association is not known.

This study, therefore, aimed first to replicate the findings of Alnassar and colleagues (2024) which showed that neuroticism mediates the relationship between EA and mental health, and furthermore to examine the independent mediating roles of neuroticism and schizotypy in the relationships of EA with mental health and sleep quality. Based on previous findings in clinical and healthy adults (Alnassar et al., 2024; Martins et al., 2014; Norman et al., 2012; Pandey et al., 2020) and a meta-analysis by Gardner and colleagues (2019) demonstrating that EA has the strongest effect (odds ratio 2.35, CI: 2.14–2.87) of all childhood trauma types on mental health, we hypothesised that EA would have the strongest association, of all forms of childhood trauma, with poor mental health (depression, anxiety, stress) and sleep outcomes, and that these relationships would be mediated, at least partially, via neuroticism. Additionally, based on previous findings of Velikonja et al. (2015) showing that EA independently predicts both positive and negative symptoms of schizotypy, and given that schizotypy, in turn, is associated with increased risk of psychopathology and poor mental health (Hodgekins, 2015; Ödén and Goulding, 2018), we further expected schizotypy to partially mediate (independent of neuroticism) the EA-poor mental health (and sleep) association.

## 2. Methods

The study sample consisted of 490 healthy adults aged between 18 and 50 years who had participated in our previous studies (Chauhan et al., 2024a, 2024b). Participants were required to meet the following

inclusion/exclusion criteria: (i) aged 18 years or older, (ii) native or proficient fluency in English, (iii) no regular use of medication except for contraceptives or multivitamins, and (iv) no current or past diagnosis (as self-reported) of a mental disorder or substance abuse. From the initial sample of 490 participants, 12 individuals were excluded due to incomplete data. Consequently, the final sample consisted of 478 adults (179 males, 299 females).

The study procedures were approved by the College of Health, Medicine, and Life Science Research Ethics Committee, Brunel University of London. All participants provided written informed consent to their participation.

### 2.1. Self-report assessments

All participants completed (online) self-report measures of childhood trauma, mental health, sleep, and personality traits of interest via Qualtrics (<https://www.qualtrics.com/>), taking approximately 30–35 min in total.

Childhood trauma was assessed using a 28-item short version of the *Childhood Trauma Questionnaire* (Bernstein et al., 2003). It has five subscales: emotional abuse, physical abuse, sexual abuse, physical neglect, emotional neglect, and denial/minimisation. Each of these subscales, except denial (3 items), has five items that are scored on a five-point Likert scale. Higher scores indicate a history of neglect and abuse. The internal consistency of the CTQ subscales in the current sample was acceptable-to-excellent [emotional abuse ( $\alpha=0.81$ ), physical abuse ( $\alpha=0.86$ ), sexual abuse ( $\alpha=0.91$ ), emotional neglect ( $\alpha=0.83$ ), and physical neglect ( $\alpha=0.60$ )].

Mental health was assessed using a 21-item *Depression, Anxiety and Stress Scale-21* (Lovibond and Lovibond, 1995). It has subscales for measuring depression, anxiety, and stress, with each subscale consisting of seven items that are scored on a four-point Likert scale. Higher scores indicate higher symptom severity. The internal consistency of the scale in the current sample was good [depression ( $\alpha=0.89$ ), anxiety ( $\alpha=0.84$ ), and stress ( $\alpha=0.82$ )]. In addition, sleep quality of an individual over the past month was assessed using the 19-item *Pittsburgh Sleep Quality Index* (PSQI; Buysse et al., 1989). Higher PSQI scores indicate poor sleep quality. The internal consistency of the scale in the current sample was acceptable ( $\alpha = 0.74$ ).

Personality traits were measured using a 48-item short version of the *Eysenck Personality Questionnaire* (EPQ-SF; Eysenck and Eysenck, 1992) and a 43-item short version of the *Oxford-Liverpool Inventory of Feelings and Emotions* (s-OLIFE; Mason et al., 2005). The EPQ-SF has three subscales: extraversion, neuroticism, and psychoticism, and a lie scale, each consisting of 12 items answered yes or no. Higher scores on the first three subscales indicate higher levels of extraversion, neuroticism, and psychoticism, respectively, while higher scores on the lie scale reflect a tendency to provide misleading or socially desirable responses. The internal consistency of the scale in the current sample was poor-to-good [extraversion ( $\alpha=0.83$ ), neuroticism ( $\alpha=0.82$ ), and psychoticism ( $\alpha=0.35$ )]. The psychoticism subscale was removed from all analyses because it had low reliability in our sample as also seen in other samples (Eysenck, 1993). The s-OLIFE has four subscales: unusual experiences (12 items), cognitive disorganisation (11 items), introvertive anhedonia (10 items), and impulsive non-conformity (10 items). Higher scores indicate higher levels of schizotypy. The internal consistency of the OLIFE subscales in the current sample was acceptable-to-good for unusual experiences ( $\alpha=0.77$ ) and cognitive disorganisation ( $\alpha=0.79$ ) subscales, though relatively poor for introvertive anhedonia ( $\alpha = 0.46$ ), and impulsive non-conformity ( $\alpha=0.55$ ) subscales.

### 2.2. Statistical analysis

All self-report data were checked for suitability for parametric statistical testing (skewness and kurtosis  $< \pm 2$ ). Prior to probing our hypotheses, we investigated potential sex differences in all self-report

measures using independent *t*-test, followed by Pearson's *r* to examine the relationship of childhood trauma dimensions (emotional abuse, sexual abuse, physical abuse, emotional neglect, and physical neglect) with mental health (depression, anxiety, stress), and personality traits of interest (neuroticism, schizotypy, and extraversion). Fisher's Exact *z*-test was used to examine any sex-related differences in the magnitude of these correlations. No sex differences were found, thus sex was not considered further. As in our previous studies (Chauhan et al., 2024a; 2024b), effect sizes for correlation coefficients were interpreted following Cohen's guidelines:  $\pm 0.1$  to  $\pm 0.29$  as small,  $\pm 0.3$  to  $\pm 0.49$  as medium, and  $\pm 0.5$  to  $\pm 1$  as large (Cohen, 1988).

Given that EA had the strongest association of all trauma dimensions with mental health, both in males and females (and no significant difference between males and females; see Results), and most associations between other trauma dimensions (sexual abuse, physical abuse, emotional neglect, physical neglect) and mental health variables became non-significant when we controlled for EA (Supplementary Table 1), all further analyses focused on EA, mental health, sleep quality and personality variables of interest.

We used structural equation modelling to first examine both direct and indirect effects of EA (predictor) via neuroticism (mediator) on mental health (outcome) (Fig. 1). The maximum likelihood method was employed to evaluate the model fit and estimate the path coefficients. The following indices were used to evaluate the model fit: the comparative fit index (CFI > 0.95), root mean square error of approximation (RMSEA < 0.08), the ratio of the maximum-likelihood chi-square to degrees of freedom ( $\chi^2/df < 5$ ), goodness of fit index (GFI > 0.95), Tucker-Lewis index (TLI > 0.95), and adjusted goodness of fit index (AGFI > 0.90) (Hooper et al., 2008). To evaluate the statistical significance of both direct and indirect paths, we used a bias-corrected 95 % bootstrap confidence interval and corresponding *p*-values. Similar procedures were applied while examining the mediating and direct effects of schizotypy (alongside neuroticism) on the relationship of EA with mental health and sleep quality (Fig. 2).

### 3. Results

The sample was young (mean age  $\pm$  SD = 24.6  $\pm$  4.56 years) and the majority self-reported being South Asian (72.8 %), followed by White European (12.1 %), East Asian (3.6 %), Mixed (3.1 %), Black/Caribbean (2.9 %), any other ethnicity (3.8 %), and 1.7 % chose preferred not to say. Overall, females were younger than males [ $t(476) = 4.31, p < .001$ ] and scored higher on depression [ $t(476) = 2.64, p = .004$ ], anxiety [ $t(476) = 3.45, p < .001$ ], stress [ $t(476) = 4.92, p < .001$ ], neuroticism [ $t(476) = 7.73, p < .001$ ], schizotypy [ $t(476) = 3.16, p < .001$ ], EA [ $t(476) = 3.82, p < .001$ ] and sexual abuse [ $t(476) = 2.47, p = .007$ ]. All other sex differences were non-significant  $p > .05$  (Table 1).

#### 3.1. Childhood trauma, mental health, and personality traits

EA was positively associated with poor sleep quality ( $r = 0.376, p < .001$ ), depression ( $r = 0.428, p < .001$ ), anxiety ( $r = 0.399, p < .001$ ),

stress ( $r = 0.411, p < .001$ ), neuroticism ( $r = 0.304, p < .001$ ) and schizotypy ( $r = 0.385, p < .001$ ). Other forms of childhood trauma also showed associations with mental health, sleep quality, and personality traits in the same direction, but their associations were weaker than those observed for EA. Specifically, physical abuse was associated with poor sleep quality ( $r = 0.134, p = .003$ ), depression ( $r = 0.224, p < .001$ ), anxiety ( $r = 0.267, p < .001$ ), stress ( $r = 0.189, p < .001$ ), neuroticism ( $r = 0.114, p < .001$ ), and schizotypy ( $r = 0.277, p < .001$ ). Sexual abuse was also found to be associated with poor sleep quality ( $r = 0.118, p = .010$ ), depression ( $r = 0.170, p < .001$ ), anxiety ( $r = 0.240, p < .001$ ), stress ( $r = 0.230, p < .001$ ), neuroticism ( $r = 0.145, p = .002$ ), extraversion ( $r = 0.096, p = .036$ ), and schizotypy ( $r = 0.234, p < .001$ ). Emotional neglect was associated with poor sleep quality ( $r = 0.209, p < .001$ ), depression ( $r = 0.313, p < .001$ ), anxiety ( $r = 0.247, p < .001$ ), stress ( $r = 0.231, p < .001$ ), neuroticism ( $r = 0.253, p < .001$ ), extraversion ( $r = -0.151, p < .001$ ), and schizotypy ( $r = 0.391, p < .001$ ). Lastly, physical neglect was also associated with depression ( $r = 0.235, p < .001$ ), anxiety ( $r = 0.257, p < .001$ ), stress ( $r = 0.160, p < .001$ ), neuroticism ( $r = 0.129, p = .005$ ), and schizotypy ( $r = 0.285, p < .001$ ). After controlling for EA, the majority of significant correlations between other forms of childhood trauma and mental health outcomes became non-significant (Supplementary Table 1).

While exploring sex differences, the relationship of emotional neglect with sleep quality (Fisher's *z* test = -1.70,  $p = .044$ ), depression (Fisher's *z* test = -2.41,  $p = .008$ ) as well as stress (Fisher's *z* test = -2.37,  $p = .009$ ) was stronger in females than males. The relationship between physical abuse and schizotypy (Fisher's *z* test = 2.51,  $p = .006$ ) was stronger in males than females (Supplementary Table 2). However, none of these sex differences survived Bonferroni correction for multiple comparisons ( $p > .0038$ ).

#### 3.2. Mediating role of neuroticism in EA and mental health relationship

The model employed (Fig. 1) to replicate the findings of Alnassar and colleagues' (2024) had a very good fit [ $(\chi^2/df = 2.17, p = .069)$ , GFI (0.99), AGFI (0.97), CFI (0.99), TLI (0.99), RMSEA (0.05)] and confirmed a direct significant association between EA and poor mental health ( $\beta = 0.32, p < .001$ ) and also that this association was partially mediated by neuroticism ( $\beta = 0.29, p < .001$ ) (Fig. 3).

#### 3.3. Mediating role of neuroticism and schizotypy in EA, mental health, and sleep quality relationship

The model (Fig. 2) we employed to extend the findings of Alnassar and colleagues (2024) and examine the influence of neuroticism along with schizotypy in mental health and sleep quality had an acceptable fit [ $(\chi^2/df = 4.39, p < .001)$ , GFI (0.92), AGFI (0.88), CFI (0.90), TLI (0.87), RMSEA (0.08)]. When we nested schizotypy in the original model, EA's direct significant effect on mental health was reduced from  $\beta = 0.32$  to  $\beta = 0.26$  ( $p < .001$ ) and schizotypy explained 23 % of the shared variance in mental health (Supplementary Fig. 1). Interestingly, when we added sleep quality as an outcome variable (see Fig. 4), sleep quality explained

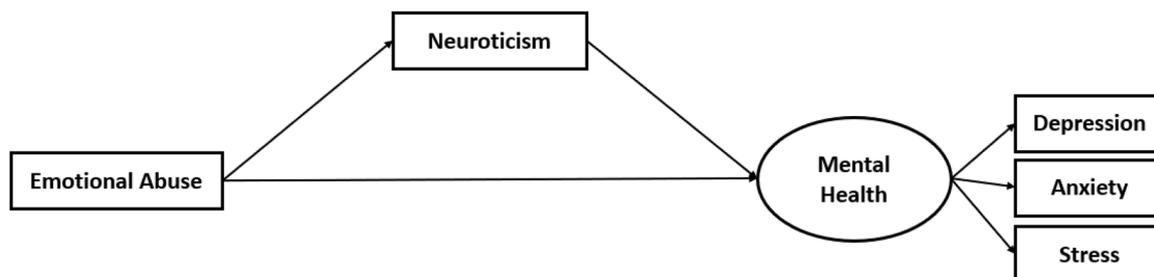


Fig. 1. Structural equation model with EA as the predictor, neuroticism as the mediator, and mental health as the outcome used to replicate Alnassar et al. (2024) findings.

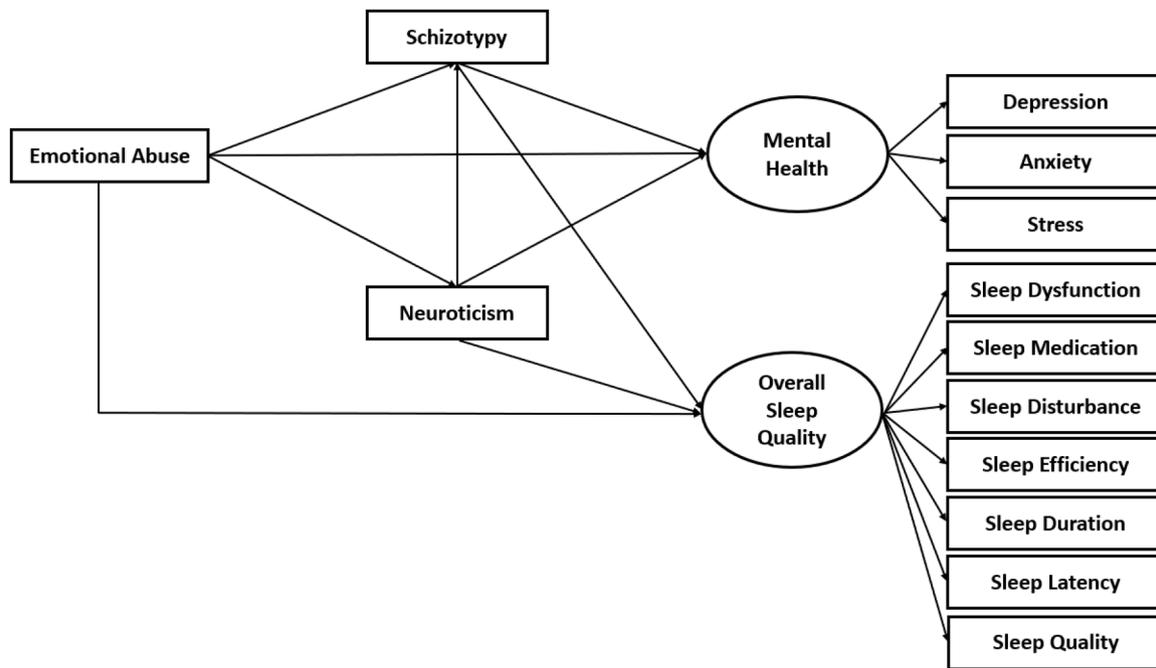


Fig. 2. Proposed model (nested in model 1) with EA as predictor, neuroticism and schizotypy as mediators, and mental health and sleep quality as outcomes.

**Table 1**  
Descriptive statistics for trauma, mental health, sleep and personality traits.

Variables		Males (N = 179)		Females (N = 299)		Overall (N = 478)	
		Mean±SD	Range	Mean±SD	Range	Mean±SD	Range
<b>Age</b>		25.74±5.03	18–40	23.92±4.11	18–46	24.6 ± 4.56	18–46
<b>CTQ-SF</b>	Emotional Abuse	9.35±4.06	5–25	11.04±5.01	5–25	10.4 ± 4.74	5–25
	Physical Abuse	8.12±3.96	5–25	8 ± 4.45	5–25	8.04±4.27	5–25
	Sexual Abuse	7.63±4.36	5–21	8.8 ± 5.38	5–25	8.36±5.05	5–25
	Emotional Neglect	11.2 ± 4.36	5–25	11.84±4.86	5–25	11.60±4.69	5–25
	Physical Neglect	10.57±3.58	5–18	10.44±3.32	5–21	10.49±3.42	5–21
	CTQ Total	46.85±15.60	25–100	50.11±17.27	25–110	48.89±16.72	25–110
<b>DASS-21</b>	Depression	11.35±10.12	0–42	14.07±11.28	0–42	13.05±10.93	0–42
	Anxiety	10.36±8.94	0–42	13.53±10.13	0–42	12.34±9.81	0–42
	Stress	11.64±8.42	0–42	15.83±9.32	0–42	14.26±9.21	0–42
<b>PSQI</b>	Sleep Quality	4.89±2.34	0–13	6.01±2.59	0–15	5.59±2.56	0–15
<b>EPQ-SF</b>	Neuroticism	5.55±3.35	0–12	7.89±3.10	0–12	7.01±3.39	0–12
	Extraversion	7.06±3.24	0–12	6.87±3.50	0–12	6.94±3.40	0–12
<b>S-OLIFE</b>	Schizotypy	16.66±8.06	1–36	19.03±7.84	3–36	18.14±8	1–36

**Abbreviations:** CTQ-SF, Childhood Trauma Questionnaire-short form; DASS-21, Depression, Anxiety and Stress Scale-21; PSQI, Pittsburgh Sleep Quality Index; EPQ-SF, Eysenck Personality Questionnaire - Short Form; S-OLIFE, Short Form of Oxford-Liverpool Inventory of Feeling and Experiences.

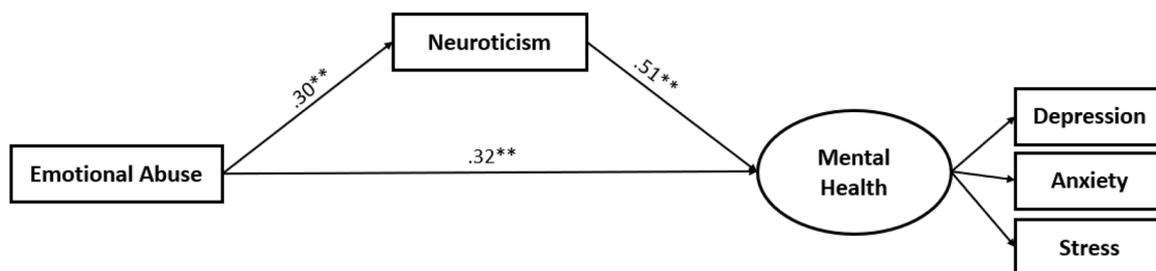


Fig. 3. Results of the SEM analysis aimed at replicating the findings of Alnassar et al. (2024) (\*\* $p < .001$ ).

41 % of the shared variance in mental health and EA’s direct significant effect on mental health was further reduced from  $\beta=0.26$  to  $\beta=0.12$  ( $p=.002$ ) (Fig. 4). EA also had a significant direct effect on sleep quality ( $\beta=0.33$ ,  $p<.001$ ). In this model (Fig. 4), we found a reduced partial effect of neuroticism (reduced from  $\beta=0.29$  to  $\beta=0.12$ ,  $p<.001$ ) in

mediating the EA-mental health relationship. Neuroticism, but not schizotypy, also partially mediated the EA-sleep quality relationship ( $\beta=0.12$ ,  $p<.01$ ). Schizotypy partially mediated the relationship between EA-mental health only ( $\beta=0.11$ ,  $p<.001$ ).

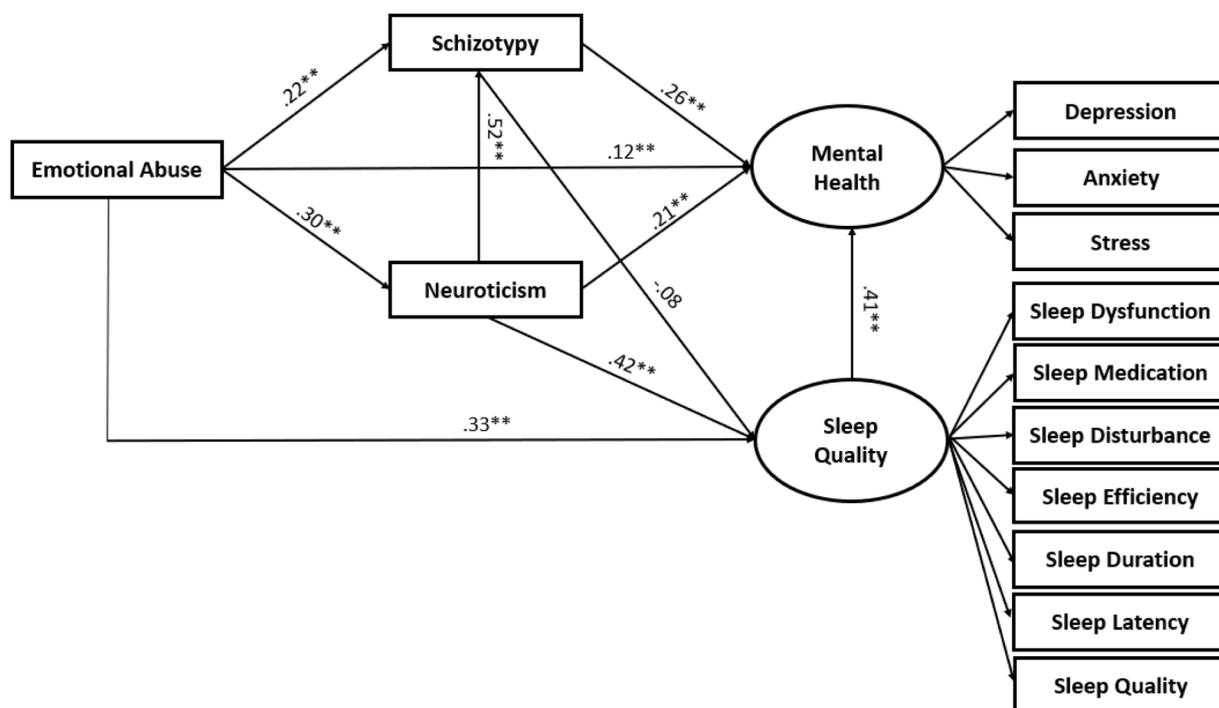


Fig. 4. Results of the SEM analysis with EA as predictor, neuroticism and schizotypy as mediators, and mental health and sleep quality as outcomes (\*\* $p < .001$ ). All the indicators of latent variables (i.e., mental health and sleep quality) were allowed to covary.

#### 4. Discussion

This study aimed to replicate the findings of [Alnassar and colleagues \(2024\)](#) on the mediating role of neuroticism in the relationship between EA and mental health and further extend the investigation to examine the independent mediating roles of neuroticism and schizotypy in the relationship between EA and mental health in healthy adults (18–50 years). The main findings were: (i) EA had medium-sized correlations ( $r$ : 0.30–0.42) while other forms of childhood maltreatment had small-to-medium-sized correlations ( $r$ : 0.09–0.39); only a few significant after controlling for EA) with mental health outcomes, sleep quality, and various personality traits (ii) neuroticism and schizotypy independently mediated the relationship between EA and poor mental health, but only neuroticism mediated the relationship between EA and poor sleep quality. While females (on average) scored higher on various self-report measures and had numerically stronger correlations between childhood trauma and mental health, sex did not significantly influence the EA-mental health relationship.

Concerning our first hypothesis, we found small-to-large-sized associations between childhood maltreatment and mental health, sleep quality, and psychopathology-related personality traits, as also seen in previous studies ([Alnassar et al., 2024](#); [Brindle et al., 2018](#); [Chauhan et al., 2024a, 2024b](#); [Gama et al., 2021](#); [Shi et al., 2022](#); [Toutontzidis et al., 2022](#); [Velikonja et al., 2015](#)). EA showed the strongest association with poor mental health and sleep quality, and personality traits of interest. Notably, after controlling for EA, the associations of other forms of childhood trauma with mental health became non-significant, highlighting its significant impact on mental health ([Kumari, 2020](#)). Previous research indicates a higher prevalence of EA (approximately 36.3 %; [Stoltenborgh et al., 2015](#)) and has consistently found it to be more strongly associated with the risk of developing and maintaining affective disorders, post-traumatic stress disorder and suicidal ideation than physical and sexual abuse as well as emotional or physical neglect ([Radell et al., 2021](#)). This is perhaps not surprising as EA, a less visible form of psychological and emotional distress, may persist for longer and go unnoticed ([Kumari, 2020](#)). Chronic emotional distress has been

closely linked with an altered hypothalamic-pituitary-adrenal (HPA) axis function ([Karin et al., 2020](#)), resulting in aberrant cortisol levels and impaired feedback inhibition of HPA mechanism ([George et al., 2025](#); [Holsboer and Ising, 2008](#)). This mechanism comprises of glucocorticoids receptors in the limbic system (e.g., hippocampus, hypothalamus) and suppresses corticotropin releasing hormone (CRH) and adrenocorticotropic hormone, thereby regulating the cortisol production ([George et al., 2025](#)). Chronic stress reduces the glucocorticoids receptors sensitivity resulting in persistently increased cortisol levels, and this in turn has been associated with structural and functional changes in the limbic system, and aberrant emotional regulation and fear response ([George et al., 2025](#)). History of childhood EA can further exacerbate these impairments making individuals more susceptible to developing and maintaining mental health issues (e.g., depression). Additionally, sleep onset is typically preceded by reduced cortisol secretion, whereas awakening is marked by increased cortisol secretion ([Meerlo et al., 2008](#)). As can be expected, cortisol disruption triggers increased night awakening, shorter sleep durations, and sleep fragmentation (review, [Buckley and Schatzberg, 2005](#)). These sleep-related disturbances can, in turn, influence the HPA regulation by elevating CRH secretion (review, [Buckley and Schatzberg, 2005](#)) and atypical melatonin secretion ([Meerlo et al., 2008](#)), further increasing the prevalence of sleep problems, behavioural dysfunction, affective disorders, and psychosis ([Chauhan et al., 2023](#); [Ferracioli-Oda et al., 2013](#); [Sanchez-Barcelo et al., 2017](#)).

We also replicated previous findings of [Alnassar and colleagues \(2024\)](#) of the mediating role of neuroticism in EA-depression and anxiety symptomatology relationship in healthy adults, further providing evidence of the predisposing role of neuroticism in mediating the childhood trauma and mental health relationship ([Roy, 2002](#)). While there is consistent evidence of a relationship between neuroticism and poor sleep ([Wang et al., 2025](#)), we report that neuroticism also plays a mediating role in the relationship between EA and sleep quality. Neuroticism, a relatively stable and inherited trait, negatively impacts cognitive and affective functioning (review, [Mitchell and Kumari, 2016](#); [Munoz et al., 2020](#)) and is associated with altered brain structures and

functions (e.g., reduced activation of the thalamus, hippocampus, and middle temporal gyrus; Kumari et al., 2007; Siragusa et al., 2021), which are important for intact social cognition and emotion processing (Lin et al., 2023). These alterations are linked to both acute and chronic emotional distress which further impair normal sleep functioning (e.g., sleep deprivation, delayed sleep onset; Kalmbach et al., 2018), significantly contributing to the severity and persistence of various psychopathologies including depression and schizophrenia (Kumari and Ettinger, 2020; Scott et al., 2021). Individuals with high levels of neuroticism often exhibit dysfunctional serotonin and dopaminergic pathways, along with abnormal cortisol levels (Canli, 2008; Karin et al., 2020; Takano et al., 2007). These abnormal physiological changes, independently or in interaction with EA of varying intensity and severity, can reinforce stress sensitivity, sleep disruption, and negative emotions, all of which contribute to the development and maintenance of psychopathologies (Alnassar et al., 2024; Roy, 2002). Positive association of neuroticism with self-reported EA and poor mental health may also be related to its known link with selective or preferential recall of negatively valenced autobiographical memories (Denkova et al., 2012; Ruiz-Caballero and Bermúdez, 1995).

The potential mechanisms underlying the EA-mental health relationship are likely to be complex. Therefore, we also explored the mediating roles of both schizotypy and neuroticism in EA-mental health relationship. Our findings revealed that schizotypy partially mediated ( $\beta=0.11$ ) the EA-mental health relationship. Neuroticism and schizotypy tend to be moderately correlated (e.g., Ettinger et al., 2005), as also seen in our study ( $\beta=0.52$ ), and play a role in the aetiology of various affective, psychiatric, and sleep-related problems (O'Hare et al., 2024; Sun et al., 2022; Watson, 2001). Individuals with schizotypy exhibit altered brain structure and function (e.g., reduced hippocampus, amygdala, and striatum volume; Ettinger et al., 2014) and report a history of abuse and neglect (Quide et al., 2021). Individuals with schizotypy also have dysfunctional dopaminergic and gamma-aminobutyric acid pathways (Ettinger et al., 2014; Mohr and Ettinger, 2014), and HPA axis, which independently (Mohr and Ettinger, 2014; Montgomery et al., 2007) or via interacting with an environmental stressor, for example EA, might elevate stress sensitivity (Murphy et al., 2022) which is known to be associated with poor coping, emotion dysregulation (Meerlo et al., 2008) and heightened risk of developing psychopathologies (Schneider et al., 2021). Another plausible explanation might be that individuals with a history of EA develop a negative self-schema, increased stress reactivity, rumination, and poor sleep (Brindle et al., 2018; McWhorter et al., 2019; Raes and Hermans, 2008; Shapero et al., 2014), further elevating their negative affect and perceived external threat leading to difficulties in emotion regulation (Bradley et al., 2011; Krause et al., 2017). An environmental trigger like EA may exacerbate underlying negative affect and/or maladaptive beliefs, either independently or via interaction with certain personality traits (as shown in our results) and thereby triggering poor mental health. In this context, schizotypy might be important in the EA-poor mental health association because schizotypy has been associated with poor cognitive control, over and above that seen for neuroticism (Völter et al., 2012), and this exacerbates maladaptive emotion regulation strategies in the context of childhood EA experience. Lastly, while the moderating effects of schizotypy have been previously reported on the relationship between sleep quality and social cognition (Hammer et al., 2024), schizotypy, unlike neuroticism, did not significantly mediate the relationship between EA and sleep quality in this study.

Our study has a few limitations. First, the retrospective investigation of childhood trauma might introduce recall or information bias, as also highlighted in previous research (Frissa et al., 2016). Second, we studied a general population sample. Although the mean CTQ-SF scores observed in our sample overlapped with those reported for healthy and psychiatric outpatient samples (e.g., Devi et al., 2019), it would be valuable to replicate our findings in a clinical or high-risk group. Third, we did not collect any information on menstrual cycle, sexual

orientation, or socio-economic status, which are reported to influence mental health conditions and sleep-related disturbances (Ibrahim et al., 2013; Papadopoulos and Sosos, 2023). Fourth, we employed a cross-sectional study design; therefore, our findings cannot speak of causation. Lastly, we used CTQ which records whether an adverse event occurred without seeking information about the frequency of such traumatic events. This may potentially reduce the sensitivity to inter-individual differences emerging from prolonged exposure of a traumatic event. Our findings underscore the need for future research to employ a longitudinal design to better understand psychological mechanisms through which neuroticism and schizotypy might mediate the association between EA and poor mental health.

To conclude, the findings of this study confirmed stronger association of EA with poor mental health than other forms of childhood maltreatment and replicated the findings of Alnassar and colleagues (2024) concerning the mediating role of neuroticism in the EA-mental health relationship. Our study further adds to the existing literature by examining the mediating role of schizotypy and finding it to be a potent mediator, in addition to neuroticism, of the EA-mental health relationship. This finding underscores the importance of identifying and addressing positive and/or negative symptoms of schizotypy in individuals with a history of EA, especially when these symptoms contribute to increased distress, fear, and/or social withdrawal. Our findings not only demonstrate that EA is an important *risk factor* for poor mental health which is worthy of societal and clinical attention in its own right, but also indicate that tailored interventions for individuals with a history of EA should prioritise targeting negative affectivity, stress reactivity, and rumination (for example, via cognitive behavioural approaches that target threat appraisals and repetitive negative thinking in such individuals). Further studies, preferably using longitudinal design, are needed to probe specific psychological mechanisms, for example, exaggerated or selective recall of negative information from childhood (Denkova et al., 2012; Ruiz-Caballero and Bermúdez, 1995) or greater inter-personal sensitivity (Hall et al., 2009; Premkumar et al., 2012), through which neuroticism and schizotypy might mediate the association between EA and poor mental health. This knowledge would be helpful for developing psychological interventions for counteracting adverse impact of EA on mental health outcomes.

#### Author statement

All authors have reviewed and approved the final manuscript, confirming this is their original work and has not been submitted or published elsewhere.

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**Satyam Chauhan:** Writing – review & editing, Writing – original draft, Visualization, Software, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Holly Cooper:** Writing – review & editing, Writing – original draft, Conceptualization. **Rakesh Pandey:** Writing – review & editing, Data curation. **Veena Kumari:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Resources, Methodology, Formal analysis, Data curation, Conceptualization.

#### Declaration of competing interest

Authors declare no competing interests.

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## Supplementary materials

Supplementary material associated with this article can be found, in the online version, at [doi:10.1016/j.jadr.2026.101051](https://doi.org/10.1016/j.jadr.2026.101051).

## Data availability

The data are publicly available at [doi:10.17633/rd.brunel.31446223](https://doi.org/10.17633/rd.brunel.31446223).

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