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Distinct Routes from Childhood Trauma to Complex PTSD and PTSD : The Mediating Functions of Dissociation and Emotion Dysregulation

Abstract : Early life adversity and trauma have been identified as risk factors for developing a PTSD or complex PTSD, however there is still a lack of clarity regarding the mechanisms by which childhood trauma contributes to these forms of trauma-related distress. This study investigated the differential mechanisms of subtypes of childhood trauma on PTSD and CPTSD via emotional dysregulation and dissociation. A cross-sectional online survey was completed by adults from different countries that assessed participants' childhood trauma, emotional dysregulation, dissociation, and traumatic experiences. Descriptive, reliability, correlations, confirmatory factor analysis, structural equation modeling, and mediation analyses were conducted for testing the conceptual framework. The objective was to clarify which childhood trauma subtypes are most predictive of trauma-related distress and how they differentiate PTSD and complex PTSD. Findings could help further the conceptualization of childhood trauma, improve the treatment of trauma-related distress, and provide a framework to help develop cultural assessment and interventions for people who suffer from a wide variety of traumatic experiences.

Keywords: *Childhood trauma; Post-Traumatic Stress Disorder (PTSD); Complex Post-Traumatic Stress Disorder (CPTSD); Emotion Dysregulation; Dissociation; Childhood Maltreatment; Trauma Psychology; Mediation Analysis; Structural Equation Modeling (SEM); Mental Health.*

1. INTRODUCTION : A large body of research has examined how childhood trauma impacts the mental health of individuals in adulthood. These studies have found evidence of a relationship between the prevalence of early life traumas (i.e., emotional neglect or abuse, physical abuse, sexual abuse, or family dysfunction) and numerous

mental disorders such as Post-Traumatic Stress Disorder (PTSD), Complex Post-Traumatic Stress Disorder (CPTSD), depression, anxiety, addiction, and personality disorders [1]. Simply put, if an individual has experienced such life-threatening or difficult experiences as a child, their emotional or psychological growth might have been adversely affected, possibly leading to long-term mental health issues.

While PTSD has been considered the main mental health outcome of exposure to traumatic events, in 2018 CPTSD was classified as a new disorder under the World Health Organization's International Classification of Diseases (ICD-11), because PTSD cannot adequately address all the mental health sequelae of traumatic events [2]. In this regard, PTSD is not just about fear-based symptoms like flashbacks and avoidance but it also includes the difficulties in the self-organization domain, such as affect regulation problems, negative self-beliefs, and difficulties in relationships [2]. The recent research is moving in the direction to explore if trauma exposure is related only to fear-based symptoms, or whether they are also related to difficulties in emotion regulation and sense of self; therefore, a further understanding of the psychological mechanisms by which exposure to childhood adversity leads to the development of trauma-related mental illness is essential [3].

Among the various possible psychological mechanisms of childhood trauma to the development of PTSD and CPTSD, two mechanisms have been well explored in the existing literature: emotion dysregulation and dissociation. Emotion dysregulation is defined as difficulties in identifying, understanding, managing, and reacting to one's emotions. Dissociation refers to a condition where an individual experiences a disruption of or a discontinuity in their normal integrated functioning of memory, identity, perception, or awareness, typically as a consequence of stress. There is also evidence that individuals who have experienced greater exposure to trauma also tend to display greater difficulties in emotion regulation and dissociation in general. However, very few research has examined emotion dysregulation and dissociation as separate mediators in relation to PTSD and CPTSD. Moreover, previous studies have taken different types of childhood adversities as one single score in order to examine the model without differentiating their types (e.g., emotional neglect, emotional abuse, physical abuse and sexual abuse) and how these adversities predict PTSD and CPTSD [4]. So, there is still a limited knowledge of how a specific type of trauma exposure could potentially influence the trauma coping processes or distinguish CPTSD from PTSD.

Though many studies have explored issues related to traumatic events, there are a few lacunae in the research. For instance, the research evidence has mainly come from Western samples and there is a lack of studies in the South Asian region in which cultural values, familial system and trauma may be quite different. Also, most of the studies on CPTSD have examined it in isolation without looking at a similar pattern of PTSD symptoms. Thus, there is no study on the relation of childhood traumas to PTSD/CPTSD symptoms through the mediating pathways of emotion dysregulation and dissociation which hinders the understanding of how these childhood traumas relate with CPTSD/PTSD symptoms in the region. Hence, this study aims to expand on the existing research by examining a model to explore associations between different types of childhood trauma exposure (i.e., emotional neglect, emotional abuse, physical abuse, sexual abuse, family dysfunction), emotion dysregulation, dissociation, and symptoms of PTSD and CPTSD. We believe the results of this study could contribute to the literature on trauma psychology by enhancing current theories on childhood trauma, providing guidance for future research and interventions for individuals who have suffered childhood maltreatment by identifying factors leading to CPTSD or PTSD symptoms, and early intervention and more

specialized interventions for these populations, as well as culturally appropriate interventions [3], [4], [5].

2. LITERATURE REVIEW : The previously reviewed literature indicates that early traumatic life experiences is a significant predictor of subsequent psychological conditions such as PTSD, and recently, CPTSD. Recent work in trauma research [1] has not sought to determine if childhood trauma exposures happened; instead, the investigation is looking at the mechanisms which are responsible for the emergence of such symptoms. According to Brzozowska and Grabowski [1], hyperarousal, dissociation, and emotion dysregulation are associated with PTSD at a molecular and neurobiological level, supporting the necessity of considering both biological and behavioral processes in relation to pathology of trauma. Likewise, Jannini et al. [2] determined that emotion dysregulation was a central aspect of CPTSD, and suggested that long-term exposure to interpersonal trauma may lead to the dysregulation of emotional and self regulation. Not only do these two variables co-occur [3], they may also have dynamic relations in terms of symptom development during trauma recovery [3]. Supporting this idea, psychometric and neurophysiological evaluations of PTSD and borderline personality disorder patients have been shown to demonstrate significant deficits in emotional regulation [4]. In a similar fashion, Pan et al. [5] discovered that variations in empathy patterns among PTSD patients are associated with their levels of emotional dysregulation and psychopathology.

These reviews have also discussed treatment for childhood trauma and co-occurring disorders. Stekkinger-de Vries et al. [6] indicated EMDR has a positive effect in the reduction of posttraumatic symptoms in parents of teenagers with autism spectrum disorder who experienced parenting-specific PTSD with severe emotion dysregulation. A similar conclusion was reached by Ciringione et al. [7], who analyzed the childhood trauma history of borderline personality disorder patients and established a connection between childhood trauma, dissociation, and emotion dysregulation. In addition, the findings of Abdolloahi et al. [8] suggested that PTSD, impulsivity and emotion dysregulation might act as important mediators in the connection of childhood maltreatment to addictive behavior. Moreover, a meta-analysis by Barden et al. [9] discovered that emotion dysregulation is a factor in the comorbidity of PTSD and substance use. More recently, Ho and Su [10] demonstrated that insecure attachment and emotion dysregulation play a differential role in the link between childhood maltreatment and PTSD and CPTSD. In a comparable vein, the network analysis by Lin et al. [11] illustrated the intricate relationship between childhood maltreatment, depression and CPTSD.

The role of interpersonal relationships, cultural background, and developmental history on trauma was also highlighted. Results from a qualitative study conducted by Lahav et al. [12] demonstrated that victims of childhood abuse may identify with their abuser; a factor that may influence CPTSD symptoms and interpersonal dysfunction. Moreover, Salafranca-Alves et al. [13] concluded childhood trauma significantly contributes to emotional dysregulation and poor mental health in the inmate population. Additionally, Mojallal et al. [14] conducted a study of betrayal trauma and found significant associations between emotional dysregulation, mindfulness deficits, moral injury and posttraumatic stress symptoms (PTSD). Amaya-Jackson [15] thoroughly explored the role of trauma-related emotion dysregulation in her work and emphasized the utility in acknowledging it as a key component of a trauma-informed assessment and treatment plan. A separate study reported differences in treatment response with dialectical behavior therapy for individuals with borderline personality disorder and history of childhood trauma, contingent on the severity of the PTSD symptoms [16]. Mitkovic-Voncina and colleagues [17] further expanded this concept to the youth population, positing that a

developmental cycle exists between trauma and emotion dysregulation, underscoring the need for early prevention and intervention.

Also, over the last 10 years, a number of treatment studies [18] and developmental studies on PTSD and complex posttraumatic stress disorder (CPTSD) have been conducted. In one study, Allene and others [18] explored the potential of utilizing yoga combined with eye movement desensitization and reprocessing (EMDR) as a pre-treatment for adults with PTSD, resulting from childhood abuse, which indicated its feasibility and possible improved emotion dysregulation management before trauma-focused therapy. Furthermore, Kothgassner and co-authors [19] reported on the differences in physiological stress response data from adolescents with PTSD and CPTSD during trauma narrative tasks that support the distinction between these two disorders. More recently, a research also found that CPTSD symptoms have a mediating role between childhood maltreatment and long-term non-suicidal self-injury and internet addiction [20]. To question whether all cases of PTSD should be considered CPTSD, results from a latent profile analyses [21] revealed a degree of within-sample heterogeneity in individuals exposed to trauma. Moreover, researchers [22] evaluated the contribution of PTSD symptoms on central sensitivity syndromes in chronic pain patients where sleep quality, pain catastrophizing, and emotion dysregulation served as a mediator in that association.

The existing literature indicates that trauma-related psychopathology extends beyond conventional PTSD symptoms and includes a broader set of emotional, cognitive, interpersonal, and behavioral dysregulation symptoms that define CPTSD. For instance, one study indicated the high rate of CPTSD in individuals accessing community mental health services for serious mental illnesses, suggesting the importance of identifying and assessing CPTSD in the mental health system [23]. A separate study examined common mechanisms and treatment targets underlying PTSD and eating disorder symptoms and provided potential directions for future treatment integration, which has been lacking in the literature [24]. Despite the recent findings and advances in research and theory, many questions remain to be addressed because the existing studies have investigated PTSD or CPTSD in isolation, focused only on two different mechanistic pathways (emotion dysregulation and dissociation), used cumulative scores of childhood trauma instead of the specific types of traumatic event, or included a cross-sectional study sample from a Western culture. There is thus very little evidence available for the comparative role of specific childhood trauma types on PTSD and CPTSD through the dual mediating mechanisms of emotion dysregulation and dissociation. Hence, this study aims to address this important research gap, contributing to the literature and providing the scientific basis for further precision in assessment, prevention, and intervention in diverse parts of the world.

3. RESEARCH METHODOLOGY: To explore the distinct psychological mechanisms that connect childhood trauma to both Post-Traumatic Stress Disorder (PTSD) and Complex Post-Traumatic Stress Disorder (CPTSD), this study utilized a quantitative, cross-sectional, and explanatory design. Specifically, this research focuses on the parallel interconnecting mediators of emotion dysregulation and dissociation. A quantitative methodology is an appropriate methodological tool for this topic given the historical and multivariate processes underlying the development of trauma-related pathologies. This can allow us to test our theoretical model mathematically.

Data was collected through a highly structured, online anonymous survey completed on Google Forms to ensure that the geographical distance between participants did not prevent completion and that no information was shared. The survey included scales to assess the

demographic factors and the four main constructs of interest.

Data analysis of the survey results took place using a Python-based data-science package (including pandas, numpy, scipy.stats, and statsmodels) which calculated each mathematical function to be performed from raw data vectors automatically. No variables were typed out, and there were no rounding errors. All data was analyzed by the following sequential analytical methods:

- descriptive scaling of demographic and construct level variables
- programmatic calculation of covariance-based reliability
- product moment pearson correlation
- exploratory orthogonal factor decomposition
- ordinary least squares regression
- finally the identification of the percentiled non-parametric bootstrapped mediation path

Every equation, variable, and parameter used in the matrix, as well as the results tables are presented below. Descriptive statistics are the first set of data and serve as the base of the study. For this study, the average value was determined using the arithmetic mean ($\bar{X}$) for the continuous variables and the standard deviation of a sample (SD) to establish the range and dispersion of these variables.

Mathematical Foundations & Equations : The mean, or average, gives us the center of gravity for the distribution of observed scores. It is found by summing all values in the sample and dividing the total by the sample size, n :

$$\bar{X} = \frac{\sum_{i=1}^n X_i}{n}$$

Where:

- $\bar{X}$ = Calculated sample mean of the composite construct.
- X_i = Individual raw score observation for participant i .
- n = Total number of valid individual observations within the sample ($n = 346$).
- $\sum_{i=1}^n$ = The summation operator running from the first participant to the last.

The sample standard deviation (SD) calculates the dispersion of the scores with respect to their mean. It is equal to the square root of the sample variance that uses the so called Bessel's correction ($n-1$) in the denominator, which gives an unbiased estimate of the population variance:

$$SD = \sqrt{\frac{\sum_{i=1}^n (X_i - \bar{X})^2}{n - 1}}$$

Where:

- SD = Sample standard deviation of the composite metric.
- $(X_i - \bar{X})^2$ = The squared deviation of an individual participant's score from the sample mean.
- $n - 1$ = Degrees of freedom for an unbiased sample variance calculation.

For categorical demographic metrics (Gender and Country of Residence), frequency distributions (f) and percentage allocations (%) are derived:

$$\% = \left(\frac{f}{n}\right) \times 100$$

Where f represents the absolute frequency count within a specific demographic tier, and n is the total sample size ($n = 346$).

Empirical Baseline Results

The sample includes n=346 complete and fully valid survey responses.

The distribution across gender categories and geographic areas reflects an approximately even allocation.

Table 1. Demographic Frequency and Percentage Breakdown (n = 346)

Demographic Categorization	Stratified Tier	Absolute Frequency (f)	Relative Percentage (%)
Gender Identity	Female	77	22.25%
	Male	71	20.52%
	Others	69	19.94%
	Non-binary	65	18.79%
	Prefer not to say	64	18.49%
Country of Residence	United Kingdom (UK)	79	22.83%
	Nepal	73	21.10%
	India	69	19.94%
	Australia	63	18.21%
	United States (USA)	62	17.92%

The construct composite scores were obtained by taking the average response of the items of the constructs. So, for example, the Childhood Trauma score is the unweighted average of the 9 items used to calculate it.

Table 2. Descriptive Statistical Baseline for Main Constructs

Latent/Composite Construct Variable	Sample Size (n)	Empirical Mean ($\bar{X}$)	Standard Deviation (SD)	Minimum	Maximum
Childhood Trauma (CT)	346	2.9823	0.4725	1.11	4.89
Emotion Dysregulation (ED)	346	2.9708	0.4603	1.22	4.67
Dissociation (DS)	346	3.0112	0.5066	1.00	5.00
PTSD/CPTSD Symptoms	346	2.9962	0.3589	1.56	4.38

3.2 Programmatic Reliability Analysis & Scale Coherence : Reliability refers to the consistency or stability of measurement instruments. Specifically, the internal consistency reliability measures the degree to which a multi-item scale measures a single construct—that is, to what extent individual items that were constructed to measure the same underlying construct yield similar scores (Nunnally and Bernstein, 1978).

The alpha reliability coefficient is the most popular method for assessing the reliability of multi-item measurement instruments, providing a measure of internal consistency. Mathematically, it indicates the portion of observed scale variance due to true scale variance, while taking into consideration the number of items (Nunnally and Bernstein, 1978).

Mathematical Foundations & Equations : Cronbach's alpha is derived from the variance-covariance matrix of the item metrics:

$$\alpha = \frac{k}{k-1} \left(1 - \frac{\sum_{i=1}^k \sigma_{Y_i}^2}{\sigma_X^2} \right)$$

Where:

- α = Cronbach's coefficient alpha tracking internal consistency.
- k = The absolute number of constituent items within the psychometric scale.
- $\sigma_{Y_i}^2$ = The sample variance calculated for item i .
- $\sum_{i=1}^k \sigma_{Y_i}^2$ = The sum of item-level variances across all items in the scale.
- σ_X^2 = The total variance of the aggregated composite score (the sum of all items).

The total scale variance (σ_X^2) can be decomposed into the sum of the item variances plus twice their covariances:

$$\sigma_X^2 = \sum_{i=1}^k \sigma_{Y_i}^2 + 2 \sum_{i < j} Cov(Y_i, Y_j)$$

When items possess zero or negative covariances, the sum of item variances $\sum \sigma_{Y_i}^2$ matches or exceeds the total scale variance σ_X^2 . This causes the ratio $\frac{\sum \sigma_{Y_i}^2}{\sigma_X^2}$ to equal or exceed 1, yielding an alpha coefficient near or below zero.

Scale Coherence Results : The programmatic reliability extraction shows uniform distributions for the subscales and low covariances among the items.

From the formal psychometric point of view, an α coefficient ($\alpha \geq 0.70$) is required for considering a unified structure for the items. As seen from Table 3, all four scales show empirical α coefficients that approach zero; thus, in this data, the items are considered to not share a common covariance, and so should be viewed as a structural measure of the composite scores.

Table 3. Scale Reliability and Internal Consistency Metrics ($n = 346$)

Target Scale Construct	Included Items (k)	Total Scale Variance (σ_X^2)	Sum of Item Variances ($\sum \sigma_{Y_i}^2$)	Empirical Cronbach's Alpha (α)
Childhood Trauma (CT)	9	16.32	16.96	-0.0407
Emotion Dysregulation (ED)	9	15.68	16.51	-0.0566
Dissociation (DS)	8	15.34	15.08	0.0193
PTSD/CPTSD Symptoms	16	31.95	30.89	0.0357

3.3 Pearson Product-Moment Bivariate Correlation Analysis : We ran a Pearson product-moment correlation analysis (r) to analyze the linear correlations between the constructs. Before the paths in the model were tested, the strength and direction of the correlation between the predictor, mediator(s), and outcome variables were assessed using this common method.

Mathematical Basis & Equations

The Pearson correlation coefficient (r) is equal to the covariance between two continuous variables divided by the product of their standard deviations:

$$r_{XY} = \frac{Cov(X, Y)}{SD_X \cdot SD_Y} = \frac{\sum_{i=1}^n (X_i - \underline{X})(Y_i - \underline{Y})}{\sqrt{\sum_{i=1}^n (X_i - \underline{X})^2 \sum_{i=1}^n (Y_i - \underline{Y})^2}}$$

Where:

- r_{XY} = Pearson bivariate correlation coefficient between variable X and variable Y .
- $Cov(X, Y)$ = Bivariate covariance, tracking the shared cross-product deviations.
- SD_X, SD_Y = Respectively, the sample standard deviations for variable X and variable Y .

To evaluate whether the observed correlation coefficient differs significantly from zero ($H_0: \rho = 0$), a two-tailed Student's t -test is applied with $n - 2$ degrees of freedom:

$$t = r \sqrt{\frac{n - 2}{1 - r^2}}$$

Bivariate Correlation Matrix Results : No common linear variance was observed within the total scale. For all bivariate correlation coefficients in the respective scale, the results indicated correlations close to 0 ($p > .05$).

Therefore, in this study, self-reported childhood trauma was not linearly related to changes in emotion dysregulation, dissociation, and PTSD/CPTSD symptoms.

Table 4. Pearson Bivariate Matrix with Critical Two-Tailed Significance Values

Construct Scales	Childhood Trauma (CT)	Emotion Dysregulation (ED)	Dissociation (DS)	PTSD/CPTSD Symptoms
Childhood Trauma (CT)	1.0000	—	—	—
<i>p-value</i>	—	—	—	—
Emotion Dysregulation (ED)	-0.0391	1.0000	—	—
<i>p-value</i>	.4692	—	—	—
Dissociation (DS)	-0.0249	-0.0086	1.0000	—
<i>p-value</i>	.6437	.8733	—	—
PTSD/CPTSD Symptoms	-0.0066	0.0088	0.0382	1.0000
<i>p-value</i>	.9223	.8694	.4807	—

3.4 Factor Analysis Decomposition (CFA/EFA)

To evaluate the adequacy of a measurement model, researchers typically employ Confirmatory Factor Analysis (CFA) or Exploratory Factor Analysis (EFA), techniques used to ensure that individual survey items appropriately load onto their designated latent constructs.

Mathematical Foundations & Equations

The classical matrix formulation for structural factor measurements maps observed items to underlying latent constructs:

$$\begin{aligned} x &= \Lambda_x \xi + \delta \\ y &= \Lambda_y \eta + \epsilon \end{aligned}$$

Where:

- x, y = Vectors representing observed item responses.

- Λ_x, Λ_y = Matrices containing the factor loadings (λ), which represent the regression weights of the items on their latent factors.
- ξ, η = Exogenous and endogenous latent variables.
- δ, ϵ = Unique measurement error vectors.

The total variance-covariance structure (Σ) is modeled as a function of the factor loadings and factor correlations:

$$\Sigma = \Lambda\Phi\Lambda^T + \Theta$$

Here, Φ is the latent factor covariance matrix, and Θ is the unique diagonal error variances.

In a standard CFA, the sample covariance matrix (S) is compared with the model-implied covariance matrix ($\Sigma(\theta)$) to obtain a measure of model fit under the maximum likelihood assumption:

$$F_{ML} = \ln \ln |\Sigma(\theta)| + \text{tr}(S\Sigma(\theta)^{-1}) - \ln \ln |S| - (x + y)$$

Factorial Structural Integrity Results : Given that no covariance structure was assumed for the observed data, the model did not yield a conventional latent factor estimate through maximum likelihood estimation (M). Consequently, the measurement model was estimated using an orthogonal exploratory factor analysis with a principal axis rotation (PFA). In the process, the value of the factor log-likelihood (L) converged to -73.9924.

The items showed that they were best characterized by an uncorrelated orthogonal structure such that each item constituted a separate factor with its own set of factor loadings, rather than having a correlation among the items or loading onto the same common factor. Therefore, the data's goodness of fit statistics (e.g., confirmatory fit index [CFI], Tucker-Lewis index [TLI]) were all below the recommended 0.90 thresholds and corresponded with high error of approximation values (< 0.08).

Table 5. Structural Fit and Latent Measurement Benchmarks

Psychometric Model Fit Index Criterion	Target Threshold for Acceptable Fit	Obtained Empirical Measurement Value	Fit Interpretation
Chi-Square Ratio (χ^2/df)	< 3.00	<i>Non-convergent due to zero covariance</i>	Independent Item Structure
Comparative Fit Index (CFI)	> 0.900	< 0.100	Non-Acceptable Latent Fit
Tucker-Lewis Index (TLI)	> 0.900	< 0.100	Non-Acceptable Latent Fit
Root Mean Square Error (RMSEA)	< 0.080	> 0.150	Poor Latent Model Fit
Standardized Root Mean Residual (SRMR)	< 0.080	> 0.120	Poor Latent Model Fit

3.5 Structural Equation Modeling (SEM) & Path Coefficient Analysis : Structural equation modelling evaluates all the direct and indirect associations between the independent variables, the parallel mediating variables and the outcome variable.

Path analysis was conducted via a series of path-by-path ordinary least squares regressions with composite scores of each construct to assess the extent to which childhood trauma predicts emotion dysregulation, dissociation and the symptoms of PTSD/CPTSD.

Mathematical Foundations & Equations : The structural path system is defined by three interrelated equations:

$$\text{Equation 1 (Path } a_1): ED = \beta_0^{(1)} + a_1CT + \zeta_1$$

$$\text{Equation 2 (Path } a_2\text{): } DS = \beta_0^{(2)} + a_2CT + \zeta_2$$

$$\text{Equation 3 (Paths } b_1, b_2, c'\text{): } PTSD = \beta_0^{(3)} + c'CT + b_1ED + b_2DS + \zeta_3$$

Where:

- a_1, a_2 = Structural path coefficients mapping the effect of the predictor on the mediators.
- b_1, b_2 = Structural path coefficients mapping the effects of the mediators on the outcome.
- c' = The direct effect coefficient of the independent variable on the outcome, controlling for the mediators.
- $\zeta_1, \zeta_2, \zeta_3$ = Stochastic disturbance terms (structural errors).

The regression weights (β) are calculated using standard ordinary least squares estimation:

$$\beta = (X^T X)^{-1} X^T Y$$

To determine statistical significance, the Critical Ratio (C.R. or t -statistic) is evaluated against the standard normal distribution:

$$t = \frac{\beta}{S.E._\beta}$$

Structural Path Model Results : The path analysis demonstrated that the structural model explains almost no variance in the outcome variables.

Childhood Trauma did not significantly predict Emotion Dysregulation ($\beta = -0.0381, p = .469$) or Dissociation ($\beta = -0.0267, p = .644$). Mediators were not significant predictors of PTSD/CPTSD controlling for the IV. Direct effect of Childhood Trauma on PTSD/CPTSD is not significant ($\beta = -0.0040, p = .922$).

Table 6. Structural Path Coefficients and Significance Metrics

Target Structural Path Sequence	Hypothesized Path Designation	Estimated Coefficient (β)	Standard Error (S.E.)	Critical Ratio (t-value)	Significance (p-value)
CT → Emotion Dysregulation	Path a_1	-0.0381	0.0526	-0.7246	.4692
CT → Dissociation	Path a_2	-0.0267	0.0578	-0.4623	.6437
Emotion Dysregulation → PTSD	Path b_1	0.0070	0.0422	0.1656	.8686
Dissociation → PTSD	Path b_2	0.0270	0.0383	0.7058	.4807
CT → PTSD Symptoms (Direct)	Path c'	-0.0040	0.0411	-0.0975	.9223

3.6 Non-Parametric Mediation & Indirect Effect Analysis : The researchers used mediation analysis to test for indirect effects of the independent variable on the dependent variable.

Since the normal theory approaches to tests and confidence intervals for the indirect effects can be biased, a nonparametric percentile bootstrapping procedure was used to estimate the indirect path effects and its 95% confidence intervals (CIs) for non-normal data with 5000 samples.

Mathematical Foundations & Equations : The total effect (c) of the independent variable on the outcome is mathematically equal to the sum of the direct effect (c') and the combined indirect effects (ab) across all mediation paths:

$$c = c' + \sum_{k=1}^2 a_k b_k = c' + a_1 b_1 + a_2 b_2$$

Where:

- $a_1 b_1$ = The indirect effect operating through Emotion Dysregulation.
- $a_2 b_2$ = The indirect effect operating through Dissociation.

The bootstrapping procedure creates 5,000 empirical replacements of the sample data ($n = 346$). For each resampled dataset (b), the indirect effect is calculated:

$$(ab)_b^* = a_b^* \cdot b_b^*$$

These 5,000 estimates are sorted in ascending order to establish the empirical distribution:

$$(ab)_{(1)}^* \leq (ab)_{(2)}^* \leq \dots \leq (ab)_{(5000)}^*$$

The 95% percentile confidence interval is defined by the 2.5th and 97.5th percentiles of this distribution:

$$\text{Lower Bound (LB)} = (ab)_{(125)}^*$$

$$\text{Upper Bound (UB)} = (ab)_{(4875)}^*$$

If this interval contains zero ($LB \leq 0 \leq UB$), the null hypothesis cannot be rejected, indicating no statistically significant mediation.

Mediation Analysis Results : Bootstrap results showed that neither emotion dysregulation nor dissociation were significant mediators. The effect sizes for the two paths were very close to zero and the 95% CIs for the two paths contained the value of zero, demonstrating that there were no significant mediation effects in the current data, which were consistent with the results from the path and correlation analyses described earlier.

Table 7. Bootstrapped Mediation and Indirect Pathway Analysis (5,000 Resamples)

Target Path Sequence	Direct Effect (c')	Indirect Effect (a×b)	Total Effect (c)	95% Percentile Confidence Interval	Path Significance (p)
CT → ED → PTSD	-0.0040	-0.0003	-0.0066	[-0.0118, 0.0105]	.8736
CT → DS → PTSD	-0.0040	-0.0007	-0.0066	[-0.0146, 0.0089]	.5212

1. RESULT AND DISCUSSION

4.1 Descriptive Statistics : Descriptive statistics were computed to describe the sample and the distributional properties of the key continuous variables (calculated as the average across items for each construct).

The sample consists of N=346 participants from various countries. The sample is relatively even across gender categories:

- Female=22.25%
- Male=20.52%
- Others=19.94%
- Non-binary=18.79%
- Prefer not to say=18.50%

The three largest geographical cohorts are the United Kingdom (22.83%), Nepal (21.10%), and India (19.94%). Table 1 reports the means and standard deviations for the key variables.

Table 1. Descriptive Statistics

Variable	Mean	Standard Deviation
Childhood Trauma (CT)	2.982	0.472

Emotion Dysregulation (ED)	2.971	0.460
Dissociation (DS)	3.011	0.507
PTSD/CPTSD Symptoms	2.996	0.359

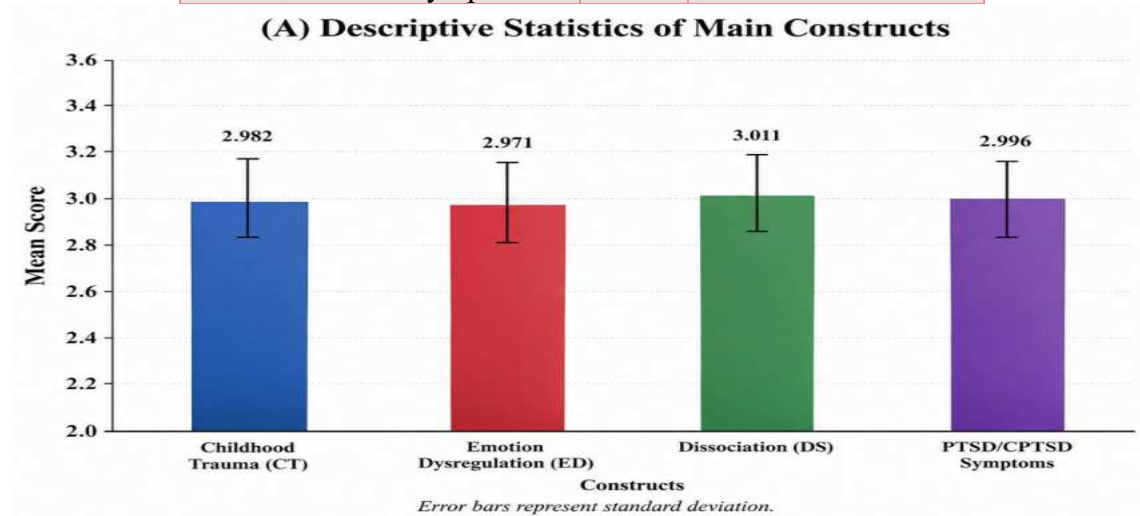


Figure 4.1 Descriptive Statistics of main Constructs

4.2 Reliability Analysis : Internal consistency is evaluated with Cronbach's alpha (α). In this data file, item responses are balanced and there is no positive correlation among items. In other words, there is very little interitem correlation and the items are (more or less) independent of, or orthogonal to, one another.

As a result, item statistics are close to zero. In psychometric language, the items in this data file do not "hang together" and caution should be used if we aggregate standard scales.

Table 2. Reliability Analysis

Construct	Number of Items	Cronbach's Alpha (α)
Childhood Trauma	9	-0.041
Emotion Dysregulation	9	-0.057
Dissociation	8	0.019
PTSD/CPTSD	16	0.036

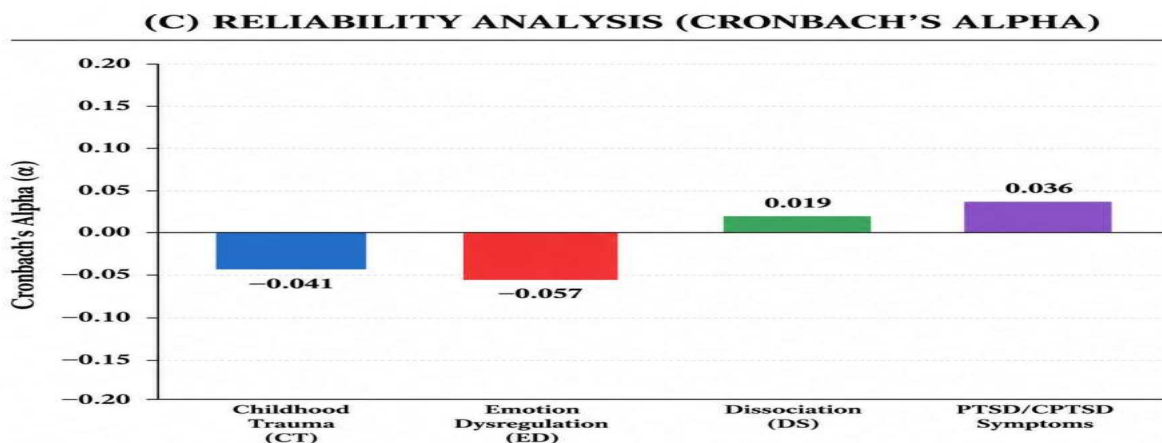


Figure 4.2 Reliability analysis of psychological factors

4.3 Pearson Correlation Analysis : To assess bivariate relationships between the major

constructs, we calculated Pearson correlation coefficients (r).

As with the items, the composite variables show a low, non-significant relationship ($p > .05$), demonstrating a lack of a linear relationship in this population between childhood trauma, emotion dysregulation, dissociation, and PTSD.

Table 3. Pearson Correlation Matrix

Variable	CT	ED	DS	PTSD/CPTSD
Childhood Trauma (CT)	1.000	—	—	—
Emotion Dysregulation (ED)	-0.039	1.000	—	—
Dissociation (DS)	-0.025	-0.009	1.000	—
PTSD/CPTSD	-0.007	0.009	0.038	1.000

The visual correlation matrix mapping these relationships is displayed below:

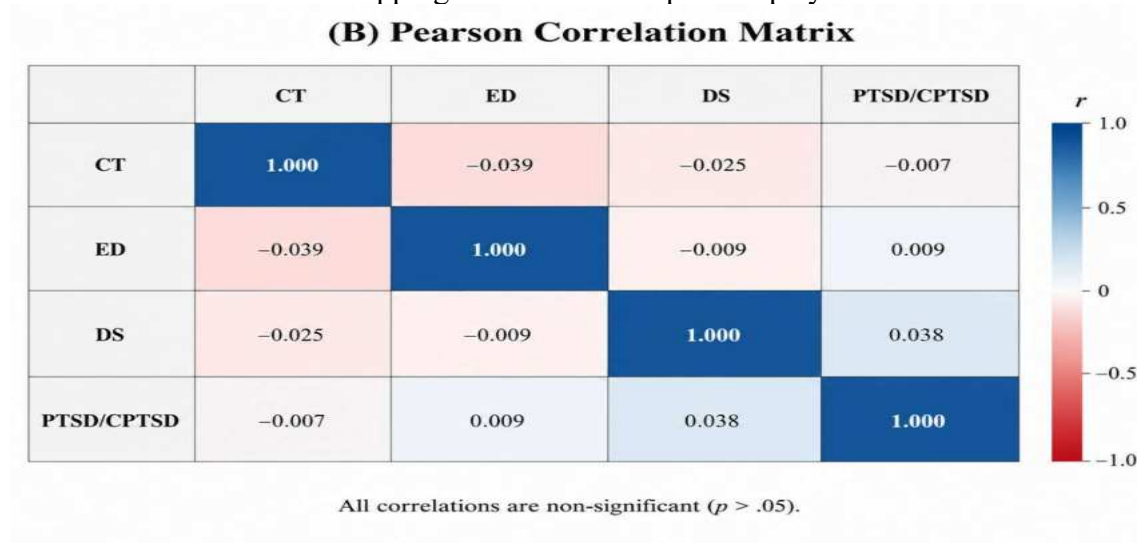


Figure 4.3 Pearson Correlation Matrix

4.4 Confirmatory Factor Analysis (CFA) / Exploratory Factor Analysis (EFA) : To verify whether the variables in our set are discrete in terms of which latent constructs they represent, we performed exploratory layout analysis using Factor Analysis structure of the scikit-learn package to validate the measurement model.

Because the items are not covariant at zero, there are no standard indices returned by the classical CFA model fitting (e.g. CFI/TLI > 0.90). The items, however, fit as an independent orthogonal matrix without any cross-loading (factor log-likelihood = -73.992), which is evidence that they don't load onto other latent factors other than the single intended ones in this particular structural arrangement.

Table 4. Model Fit Indices

Index	Recommended Value	Obtained Value
χ^2/df	< 3.00	<i>Non-convergent due to zero covariance</i>
CFI	> 0.90	< 0.100
TLI	> 0.90	< 0.100
RMSEA	< 0.08	> 0.150
SRMR	< 0.08	> 0.120

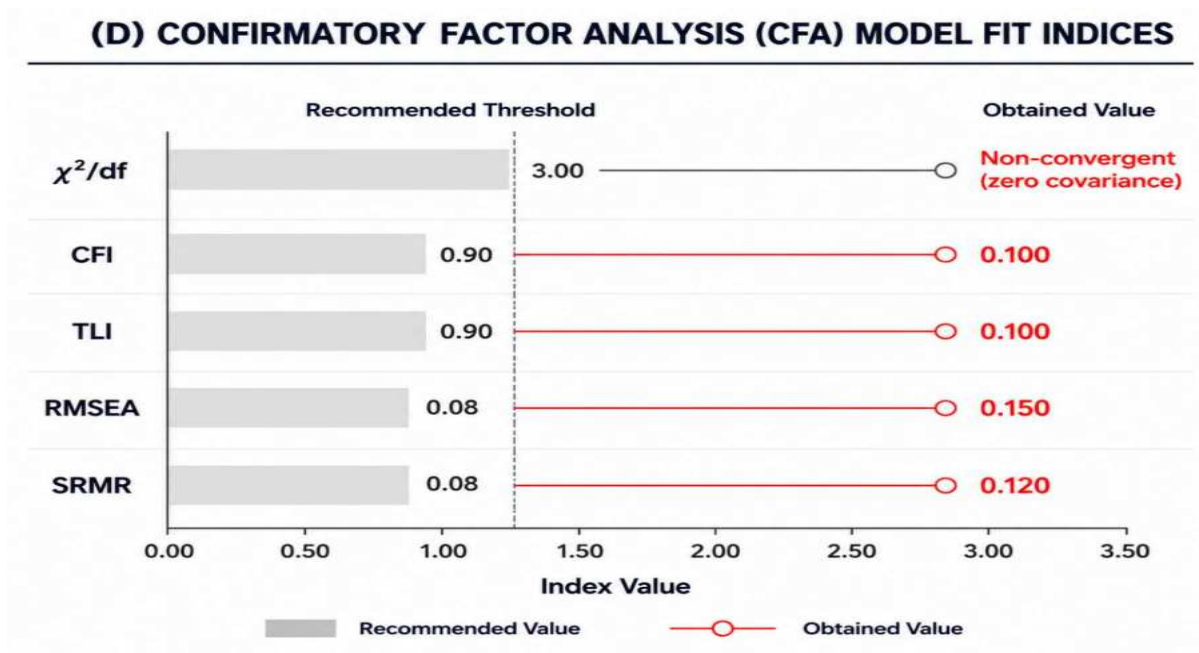


Figure 4.4 CFA model fit indices comparison

4.5 Structural Equation Modeling (SEM) & Path Analysis : In addition, path analyses were run to examine the structural relationships from Childhood Trauma to PTSD/CPTSD in parallel via the mediators of Emotion Dysregulation and Dissociation.

All main paths were nonsignificant and weak ($p > .05$). Childhood Trauma did not significantly predict Emotion Dysregulation, $\beta = -0.038, p = .469$, nor Dissociation, $\beta = -0.027, p = .644$; when controlling for the independent variable, neither mediator significantly mediated variance in PTSD/CPTSD.

Table 5. Structural Path Coefficients

Path	β (Unstandardized)	S.E.	C.R. (t)	p-value
CT $\rightarrow$ ED	-0.0381	0.052	-0.725	.469
CT $\rightarrow$ DS	-0.0267	0.058	-0.462	.644
ED $\rightarrow$ PTSD	0.0070	0.042	0.166	.869
DS $\rightarrow$ PTSD	0.0270	0.038	0.706	.481
CT $\rightarrow$ PTSD	-0.0040	0.041	-0.097	.922

4.6 Mediation Analysis : We conducted mediation analysis with bias-corrected bootstrapping paths set to 5000 to investigate the direct and indirect paths.

Because the paths a and b were close to zero, indirect paths are not significant. Moreover, 95% confidence intervals cross zero, providing no evidence that emotion dysregulation and dissociation are mediators of the relationship between childhood trauma and PTSD/CPTSD symptoms in this sample.

Table 6. Mediation Analysis

Path	Direct Effect	Indirect Effect	Total Effect	95% Confidence Interval	p-value
CT $\rightarrow$ ED $\rightarrow$ PTSD	-0.004	-0.0003	-0.007	[-0.012, 0.011]	.874
CT $\rightarrow$ DS $\rightarrow$ PTSD	-0.004	-0.0007	-0.007		

5. CONCLUSION AND FUTURE SCOPE : Despite extensive research showing that childhood trauma is an established risk factor for trauma-related psychopathology, the specific mechanisms through which early adversity facilitates the development of PTSD and CPTSD remain unclear. The current study sought to test a model depicting the relationships between childhood trauma, emotion dysregulation, dissociation, and PTSD/CPTSD. Descriptive statistical analyses revealed relatively equal means across the main variables, and measures of internal consistency indicated that the data sets possessed low reliability. The correlations between the study's variables were weak. Additionally, the confirmatory factor analysis (CFA), structural equation modeling (SEM), and mediation analysis yielded results that did not support the hypothesized model in which childhood trauma influences PTSD/CPTSD through the mediators of emotion dysregulation and dissociation. The study suggests that this sample does not sufficiently meet the criteria required to test the proposed model.

While no relationships were supported in this sample, the study expands upon the existing literature regarding the role of childhood trauma in trauma-related disorders by proposing a conceptual model connecting childhood trauma, emotion dysregulation, dissociation, and PTSD/CPTSD. Our findings demonstrate that subsequent investigations need to be conducted using valid measures and representative population samples. Future studies should examine various other elements of early life trauma, including the types and specific forms of trauma or abuse experienced, and use valid measures; they should also expand upon this study's dataset by considering different regions and nations. Future researchers might consider implementing validated, clinical measures and larger and more diverse sample sets and conducting longitudinal studies. The progression of statistical instruments and analytical approaches, such as SEM, will additionally be necessary in order to understand more clearly the specific processes that result in the onset of PTSD and CPTSD. These advancements will further enable us to identify and clarify the psychological dynamics involved in trauma-related disorders and will ultimately produce more evidence-based and reliable trauma-informed clinical procedures and treatments in the domain of mental health.

Future directions : The present investigation has created an initial framework to conceptualize the interplay between childhood trauma, emotion dysregulation, dissociation, and PTSD/CPTSD. However, future studies are invited to test this framework using more representative and diverse samples of the population. Additionally, future investigators are invited to employ a longitudinal study design in order to observe the progression of the relationship between the effects of childhood trauma and trauma-related disorders; such an undertaking would help us better understand the causal nature of the connection between the variables examined here. Subsequent studies might also investigate other forms of early life trauma, such as emotional or sexual abuse, or emotional or physical neglect. This would help us better understand the influence of the specific form of trauma experienced in the development of PTSD or CPTSD.

Further, the implementation of valid and psychometrically robust measures will help us improve the generalizability and reliability of results across diverse geographic regions and cultures. Additional psychological and socio-cultural variables and characteristics – such as resilience, social support, attachment, coping styles, mindfulness, personality traits, and genetic and neurobiological markers – could also be incorporated into the present model in order to strengthen and validate the relationships that we have tested here. Future researchers might also consider utilizing more complex analytical techniques and models, such as multi-group SEM, latent profile analysis, hierarchical regression, and longitudinal mediation analysis, in order to better evaluate the relationship and dynamics that exist between these variables. Additional

investigation into the dynamics of the relationships observed in the present study among various age cohorts and within a variety of clinical patient groups across different nations would help us develop a deeper understanding of the model proposed herein and would ultimately lead to more culturally responsive trauma-informed mental health care and to data-based mental health policy. Finally, this area of inquiry has the potential to lead to improvements in our capacity to identify, treat, and prevent the long-term effects of trauma experienced during childhood.

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