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The transdiagnostic network of childhood trauma, anxiety, and depression in college students: centrality of emotional neglect and the bridging function of emotional abuse

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Abstract

Background College students are highly susceptible to anxiety and depression, for which childhood trauma (CT) is a major risk factor. However, the fine-grained pathways linking specific CT subtypes to these disorders remain unclear, limiting targeted intervention. This study aimed to use network analysis to investigate the associations between CT subtypes and symptoms of anxiety, depression, and their comorbidities among college students.

Methods A cross-sectional online survey was conducted among 2,864 students from nine Chinese universities. Participants completed the Childhood Trauma Questionnaire-Short Form (CTQ-SF), Generalized Anxiety Disorder-7 (GAD-7), and Patient Health Questionnaire-9 (PHQ-9). Three regularized partial correlation networks (CT-anxiety, CT-depression, and CT-comorbidity) were constructed using the R package. Expected influence (EI) and bridge expected influence (BEI) indices were used to identify central and bridge nodes. Network stability was assessed via case-dropping bootstrapping.

Results Emotional neglect consistently emerged as the most central node across all three networks. In the CT-anxiety network, emotional abuse and “afraid something will happen” were key bridging nodes. In the CT-depression network, emotional abuse and “thoughts of death” possessed the greatest degree of bridge centrality. Critically, in the CT-comorbidity network, this same pairing again exhibited the strongest bridge centrality, demonstrating its stable role as a transdiagnostic bridge linking CT to both anxiety and depression, even within comorbid presentations.

Conclusions Emotional neglect, emotional abuse, and the key symptoms “afraid something will happen” and “thoughts of death” emerge as the central and bridge nodes linking CT to anxiety, depression, and their comorbidity. These nodes represent precise targets for early intervention to reduce the mental health burden among college students.

Trial registration Not applicable.

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Keywords Childhood trauma, Anxiety, Depression, Comorbidity, Network analysis, College students

Introduction

Anxiety and depressive disorders are among the most prevalent and debilitating internalizing disorders and constitute a substantial portion of the global psychiatric burden [1]. These conditions are of particular concern during late adolescence and early adulthood, with college students representing a highly vulnerable population [2]. This critical developmental period, marked by academic pressure, identity formation, and evolving interpersonal relationships, can heighten susceptibility to internalizing symptoms [3, 4]. A meta-analysis revealed alarmingly high prevalence rates—36% for anxiety and 39% for depression—among college students globally [5]. The ramifications extend beyond emotional distress, frequently manifesting as declining academic performance, sleep disturbances, and an increased risk of suicidal behaviors [6–8]. Furthermore, the co-occurrence of anxiety and depression is exceptionally common and is associated with greater clinical complexity, poorer treatment response, and heightened functional impairment [9–11]. Notably, a study focusing on college students revealed that the comorbidity rate for anxiety and depression exceeds 32%, approximately four times the prevalence of either disorder alone [12]. The etiology of these conditions in this population is multifaceted and involves a complex interplay of biological, psychological, and environmental factors [13]. Therefore, identifying key risk factors that contribute to the onset of anxiety, depression, and their comorbidity in this population is critical for developing effective interventions.

In this context, childhood trauma (CT) has emerged as a robust transdiagnostic risk factor for internalizing disorders [14]. Defined as harmful acts of commission or omission by caregivers, CT is commonly categorized into emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect [15]. Exposure to CT is highly prevalent among college students [16]. A comprehensive review indicated that individuals with a history of CT are significantly more likely to develop anxiety and depression [17], a correlation supported by longitudinal research [18]. Critically, a recent meta-analysis of prospective longitudinal studies confirmed that various forms of CT, including emotional abuse, significantly increase the risk for subsequent anxiety disorders [19]. Notably, the severity of CT often has a dose–response relationship with symptom severity, an association most pronounced in individuals with comorbid conditions [20]. Given this established link, elucidating the specific symptom-level pathways linking CT to anxiety, depression, and their comorbidity is essential for understanding

the precise mechanisms underlying this heterogeneous psychopathology.

However, the fine-grained links between specific CT subtypes and individual symptoms remain poorly understood. A substantial body of research has established that early exposure to CT is significantly associated with adult psychopathology, including depression and anxiety [21–23]. Meta-analyses have consistently demonstrated that emotional abuse and emotional neglect exhibit the strongest associations with depression [21, 24, 25], and that emotional abuse significantly increases the risk for subsequent anxiety disorders [19, 26]. Empirical studies further indicate that emotional abuse is a particularly deleterious form of maltreatment, associated with more severe depressive and anxiety symptoms relative to other subtypes [27, 28]. Despite these well-documented differential associations at the subtype level, the overwhelming majority of these studies have relied on total scores or latent variable models of anxiety and depressive symptoms. As Fried and Nesse argued, depression sum scores do not simply add up, because individual depressive symptoms are distinct phenomena that differ in their underlying biology, functional impairment, and risk factors [29]. Indeed, anxiety and depression are heterogeneous syndromes comprising symptoms with differing clinical importance [29], and CT is a multidimensional construct whose subtypes may be differentially linked to specific symptomatic outcomes [30]. From a dimensional perspective, CT subtypes can be meaningfully organized along the dimensions of threat and deprivation [31]. Emotional abuse and physical abuse represent experiences of threat, which primarily disrupt emotional processing and social information biases, thereby heightening vulnerability to anxiety and fear-related symptoms [32]. In contrast, emotional neglect and physical neglect constitute deprivation, which predominantly impairs cognitive development and executive functions, contributing to anhedonia and depressive symptoms [32, 33]. This theoretical framework supports the idea that distinct CT subtypes may influence psychopathology through dissociable pathways, reinforcing the need to examine symptom-level associations rather than relying on total scores. For instance, prior evidence suggests distinct associations, such as between emotional neglect and anhedonic depression [34], and anhedonia has been shown to mediate the relationship between specific CT subtypes and the severity of depressive symptoms [35]. Neglecting this symptomatic heterogeneity may mask the differential relationships between CT subtypes and individual symptoms, thereby hindering progress in

identifying precise pathogenic pathways and intervention targets [29].

One approach to overcome these limitations is network analysis. Network theory conceptualizes that mental disorders arise from the active interactions among their symptomatic elements and related factors, which provides a new perspective for understanding psychopathology [36]. In the network, symptoms or related factors are represented by nodes, and their relationships with one another are signified by edges, allowing for the examination of direct symptom-level relationships [36]. Centrality indices, such as expected influence, can identify the most influential nodes in the network [37]. Crucially, bridge centrality metrics, such as bridge expected influence, can identify nodes that connect different clusters, providing unique insights into the mechanisms underlying comorbidity [38]. Several recent studies have applied network analysis to examine the associations between childhood trauma and psychopathology. Tao et al. compared anxiety-depression symptom networks between college students with and without childhood trauma and identified anhedonia and tension as central symptoms in the trauma-exposed group [39]. Tang et al. examined symptom-level relationships among depression, anxiety, stress, and problematic smartphone use in individuals with childhood trauma [40]. Huang et al. incorporated childhood abuse subtypes into a network with depression and anxiety symptoms among depressed adolescents and identified emotional abuse as a bridge node connecting maltreatment to psychopathology [41]. Guo et al. investigated the intergenerational network of parental CT, depression, anxiety, and adolescent maladaptive traits [42]. Li et al. analyzed the network of abuse types and depressive symptoms in Chinese adolescents [43].

Notably, despite these contributions, prior network studies have key limitations that preclude the identification of fine-grained differential associations between specific CT subtypes and individual symptoms. Specifically, some studies treated childhood trauma as a binary grouping variable, which aggregates all CT subtypes into a single category and cannot differentiate their distinct roles [39, 40]. Others incorporated multiple CT subtypes but estimated a single integrated network that combines all CT subtypes and psychopathology symptoms together, which cannot determine whether the association patterns of specific CT subtypes differ qualitatively across anxiety, depression, and comorbid symptom profiles [41–43]. More critically, no study to date has simultaneously constructed and compared three separate networks to delineate the distinct symptom-level associations between specific CT subtypes and the individual symptom clusters of anxiety, depression, and their comorbidity, respectively. Such separate network comparisons are essential to test whether a given CT subtype exhibits stronger and

qualitatively different associations with depressive symptoms than with anxiety symptoms, and whether these patterns shift in the context of comorbidity.

To fill this gap, the present study employed network analysis to examine the symptom-level relationships among the five CT subtypes and individual symptoms of anxiety, depression, and their comorbidity in a large sample of Chinese college students. We constructed three regularized partial correlation networks using Gaussian graphical models based on cross-sectional data. Prior meta-analytic evidence indicates that emotional abuse and emotional neglect show the strongest associations with depression and anxiety [21, 24], and recent network findings identify emotional abuse as a bridge node connecting childhood trauma to psychopathology [41]. Drawing on this evidence, we hypothesized that specific CT subtypes, particularly emotional abuse and emotional neglect, would exhibit distinct and robust direct associations with individual symptoms of anxiety and depression, beyond what total symptom scores can capture. We further hypothesized that these CT subtypes would show substantially higher expected influence, acting as the most influential hubs in their respective networks, and higher bridge expected influence, serving as transdiagnostic bridges connecting the trauma cluster to the symptom clusters of anxiety, depression, and their comorbidity.

Accordingly, the study had three primary aims: (a) to identify the most robust symptom-level associations between CT subtypes and individual symptoms of anxiety and depression; (b) to identify the most influential central and bridge nodes within the networks using centrality and bridge centrality indices, respectively; and (c) on the basis of the bridge centrality findings, to determine which CT subtypes act as transdiagnostic factors by serving as key bridges that connect the trauma cluster to the anxiety, depression, and comorbidity symptom clusters. This is the first study to simultaneously construct and compare three separate CT-symptom networks. Therefore, our work is largely exploratory. This study aims to enhance the transdiagnostic understanding of internalizing disorders and identify potential precision intervention targets within university settings.

Methods

Participants

This cross-sectional study was conducted online between January and May 2025 with a convenience sample of college students in China. The cross-sectional design precludes causal inferences, and all reported associations should be interpreted accordingly. A convenience sample was recruited from nine universities across three provinces. Eligibility criteria included being a full-time undergraduate aged 18–24 and providing informed consent.

It should be noted that this was a convenience sample, which may limit the generalizability of the findings to the broader population of Chinese college students.

Among the 3,405 initial submissions, 541 were excluded because of rapid completion (<2 minutes), failed attention-check items, or inconsistent responses on validity items, resulting in a final sample of 2,864 participants (valid response rate: 84.1%). The sample had a mean age of 20.6 years ($SD = 1.5$); 39.5% were male, 33.1% were only children, 38.8% were from rural areas, and 31.9% were from single-parent families.

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board (Approval No. 2025–005). Data were collected anonymously via an online survey platform. All participants provided electronic informed consent voluntarily and without compensation.

Measures

Childhood trauma

CT was assessed using the Childhood Trauma Questionnaire-Short Form (CTQ-SF), a well-validated 28-item self-report measure [15]. It retrospectively evaluates the following five subtypes: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Each item is rated on a 5-point Likert scale from 1 (never true) to 5 (very often true), with total scores ranging from 25 to 125. In accordance with the established thresholds, participants were classified as having experienced a specific CT subtype if their subscale score met the following cutoff criteria: emotional neglect ≥ 15 , physical neglect ≥ 10 , sexual abuse ≥ 8 , emotional abuse ≥ 13 , or physical abuse ≥ 10 [15]. The Chinese version of the CTQ-SF has been shown to have good reliability and validity among college students [44]. In this study, the internal consistency of the scale was good (Cronbach's $\alpha = 0.803$). It should be noted that retrospective self-report measures of childhood trauma are subject to recall bias, and current emotional states may influence how participants recall and report early adverse experiences.

Anxiety symptoms

Anxiety symptoms were assessed with the 7-item Generalized Anxiety Disorder Scale (GAD-7) [45]. The scale measures symptom severity over the past two weeks. Each item is rated on a 4-point scale from 0 ("not at all") to 3 ("nearly every day"), with total scores ranging from 0 to 21. A cutoff score of 5 was used to indicate the presence of mild anxiety symptoms, which is consistent with practices in nonclinical populations [45]. The Chinese version of the GAD-7 has shown satisfactory psychometric properties among Chinese college students [46]. In this study, its internal consistency was good (Cronbach's $\alpha = 0.905$).

Depressive symptoms

Depressive symptoms were assessed with the 9-item Patient Health Questionnaire (PHQ-9) [47]. The scale evaluates the severity of depressive symptoms over the past two weeks. Each item is scored from 0 ("not at all") to 3 ("nearly every day"), yielding total scores between 0 and 27. A cutoff score of 5 was applied to denote mild depression, following established conventions [47]. The reliability and validity of the Chinese PHQ-9 have been confirmed in college student populations [48]. In this analysis, the internal consistency of the scale was good (Cronbach's $\alpha = 0.831$).

Data analysis

Descriptive statistics and reliability assessments (Cronbach's α coefficients) were performed using IBM SPSS Statistics (Version 25.0) (Armonk, NY: IBM Corp). All network analyses were conducted in R (Version 4.1.2).

Network estimation and centrality

We employed Gaussian graphical models (GGMs) to estimate three distinct undirected networks: (1) a network of CT subtypes and anxiety symptoms (CT-anxiety network), (2) a network of CT subtypes and depression symptoms (CT-depression network), and (3) a network of CT subtypes combined with all anxiety and depression symptoms (CT-comorbidity network) [49, 50]. All three networks were based on Spearman correlation matrices.

Within each network, the nodes represented the five CT subtypes (emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect) and individual symptoms of anxiety and/or depression, while the edges represented regularized partial correlations, which were estimated using the graphical least absolute shrinkage and selection operator (LASSO) [50, 51]. This regularization technique, in which the tuning parameter was selected via the extended Bayesian information criterion (EBIC; $\gamma = 0.5$), shrinks trivially small edge weights to zero, thereby removing spurious connections and yielding sparse, interpretable networks [50]. The network layout was determined using the Fruchterman–Reingold algorithm [52]. All estimations and visualizations were performed using the qgraph package in R [53].

To identify the most influential nodes within each overall network, we calculated the EI centrality, defined as the sum of a node's edge weights to all other nodes [37]. EI was computed using the qgraph package.

For the bridge centrality analysis, two communities were predefined in each network: the CT community and the symptom community. The symptom community in the CT-anxiety network consisted solely of anxiety symptoms, while that of the CT-depression network consisted solely of depression symptoms. The symptom community in the CT-comorbidity network included both anxiety

symptoms and depression symptoms. To identify nodes that specifically connect these communities, we calculated the BEI, defined as the sum of a node's edge weights connecting it to all nodes in the other community [38]. A higher BEI value indicates a greater likelihood of activating the opposite community. The BEI value was computed using the networktools package in R [38].

Network stability and accuracy estimation

The robustness of the network estimates was assessed using the R package bootnet [54]. Because network structures and centrality indices are estimated from sample data, they are subject to sampling variation. Bootstrapping procedures are therefore necessary to evaluate the accuracy and stability of the estimated parameters before substantive interpretation [54].

First, the edge weight accuracy was evaluated by calculating 95% confidence intervals (CIs) for each edge using nonparametric bootstrapping (with 1,000 bootstrap samples). A narrow 95% CI denotes good accuracy [54]. Second, the stability of centrality indices (EI and BEI) was assessed using a case-dropping subset bootstrap approach (with 1,000 bootstrap samples). Stability was quantified using the correlation stability (CS) coefficient.

Table 1 The means, SDs, and abbreviations for each node of the CTQ-SF, GAD-7 and PHQ-9

Variables	Abb	M	SD
Childhood trauma (CTQ-SF)			
Emotional neglect	C1	7.12	2.72
Physical neglect	C2	5.83	1.80
Sexual abuse	C3	5.41	1.50
Emotional abuse	C4	7.50	3.42
Physical abuse	C5	6.14	2.01
Anxiety symptoms (GAD-7)			
Nervousness or anxiety	A1	0.41	0.60
Uncontrollable worry	A2	0.34	0.60
Worry too much	A3	0.42	0.61
Trouble relaxing	A4	0.33	0.56
Restlessness	A5	0.22	0.48
Irritability	A6	0.34	0.56
Afraid something will happen	A7	0.19	0.45
Depressive symptoms (PHQ-9)			
Anhedonia	D1	0.64	0.64
Depressed or sad mood	D2	0.57	0.61
Sleep difficulties	D3	0.90	0.93
Fatigue	D4	0.78	0.63
Appetite changes	D5	0.54	0.63
Feeling of worthlessness	D6	0.35	0.59
Concentration difficulties	D7	0.44	0.61
Psychomotor agitation	D8	0.26	0.52
Thoughts of death	D9	0.10	0.35

Note: Abb, abbreviation; M, mean; SD, standard deviation; CTQ-SF, Childhood Trauma Questionnaire-Short Form; GAD-7, Generalized anxiety disorder 7-item scale; PHQ-9, Patient health questionnaire-9

The CS coefficient represents the maximum proportion of cases that can be dropped while maintaining a correlation of at least 0.7 with the original centrality order in 95% of the samples. Based on the simulation results reported by Epskamp et al. [54], the CS-coefficient should not be below 0.25, and preferably above 0.50, to indicate that the centrality indices are sufficiently stable. Finally, bootstrapped difference tests ($\alpha = 0.05$, with 1,000 bootstrap samples) were conducted to compare the strength of specific edge weights, EI values, and BEI values within and between the estimated networks.

Results

Descriptive statistics

The final sample included 2,864 Chinese college students. Among them, 594 (20.74%) reported a history of CT. The prevalence of clinically significant anxiety symptoms (GAD-7 score ≥ 5) was 19.55% ($n = 560$), and that of depressive symptoms (PHQ-9 score ≥ 5) was 42.28% ($n = 1,211$). The comorbidity rate, defined as meeting the threshold for both anxiety and depressive symptoms, was 21.09% ($n = 604$). Table 1 presents the mean scores for all the items.

The CT-Anxiety network

The regularized partial correlation network of CT subtypes and anxiety symptoms is shown in Fig. 1A. The network contained 19 cross-community edges. Several bridging connections were observed, including C3–A7, C4–A7, C4–A6, and C1–A2. Among these connections, the C3–A7 edge was the strongest (see Supplementary Material 1). In terms of node influence, C1 had the highest EI (Fig. 1B). According to the bridge centrality analysis, C4 and A7 were identified as the key bridge nodes (highest BEI; Fig. 1C).

The CT-Depression Network

The final network of correlations between CT subtypes and depression symptoms is shown in Fig. 2A. This network comprises 26 cross-community edges. The strongest cross-community edges included C4–D9, C5–D8, C4–D6, C4–D8, and C1–D7. Among these edges, the C4–D9 edge was the strongest (see Supplementary Material 1). C1 again had the highest EI (Fig. 2B). According to the bridge analysis, C4 and D9 were identified as the key bridge nodes (highest BEI; Fig. 2C).

The CT-comorbidity network

The network linking CT subtypes to cooccurring anxiety and depression symptoms is shown in Fig. 3A. The network contained 31 cross-community edges. Notably, the strongest bridging edges (C3–A7 and C4–D9) were identical to the strongest links found in the separate CT-anxiety and CT-depression networks, respectively. C1

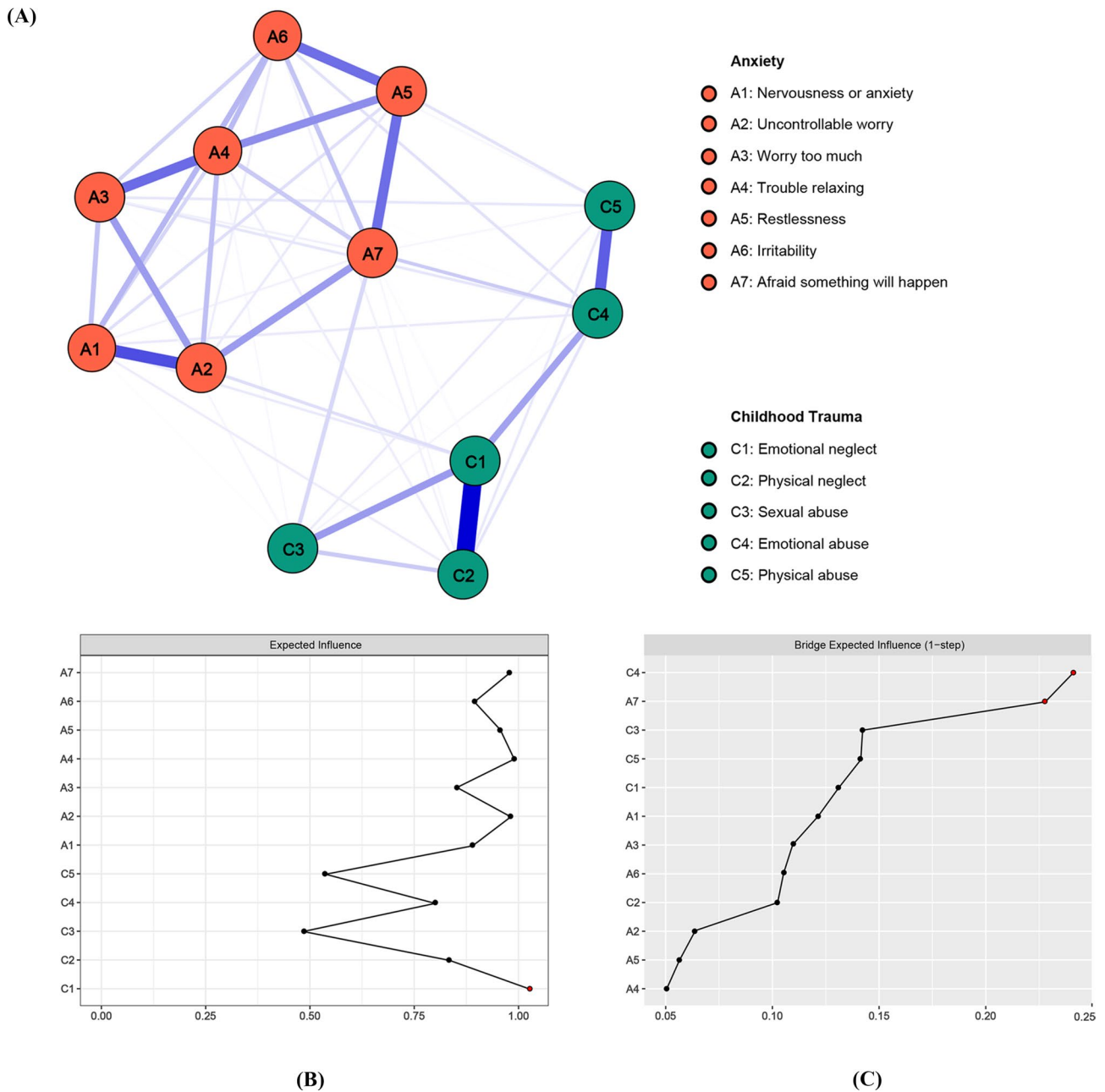


Fig. 1 Network model of CT-anxiety and the EIs and BEIs of the nodes in the network. (A) Network model of CT-anxiety. The blue line represents a positive partial correlation, while the red line represents a negative partial correlation. A thicker line and a more saturated color represent a larger partial correlation coefficient. (B) The EI indices of the nodes in the network (raw values). (C) The BEI indices of the nodes in the network (raw values)

maintained the highest EI (Fig. 3B). Bridge analysis confirmed C4 and D9 as the key bridge nodes (highest BEI; Fig. 3C), demonstrating that the C4–D9 pathway serves as the principal transdiagnostic bridge within the comorbid symptom network.

Network accuracy and stability

Network robustness tests indicated that the estimated CT-anxiety, CT-depression, and CT-comorbidity networks were reliable. The bootstrapped 95% CIs for edge

weights were narrow across all the networks (see Supplementary Material 1), indicating that the edge weights were estimated with good accuracy. The CS coefficient was 0.75 for both edge weights and the EI. For the BEI, the CS coefficients were 0.52 (CT-anxiety), 0.67 (CT-depression), and 0.75 (CT-comorbidity). All CS-coefficients exceeded the recommended threshold of 0.50, indicating that the centrality indices were sufficiently stable. Collectively, these results validate the reliability of the study outcomes.

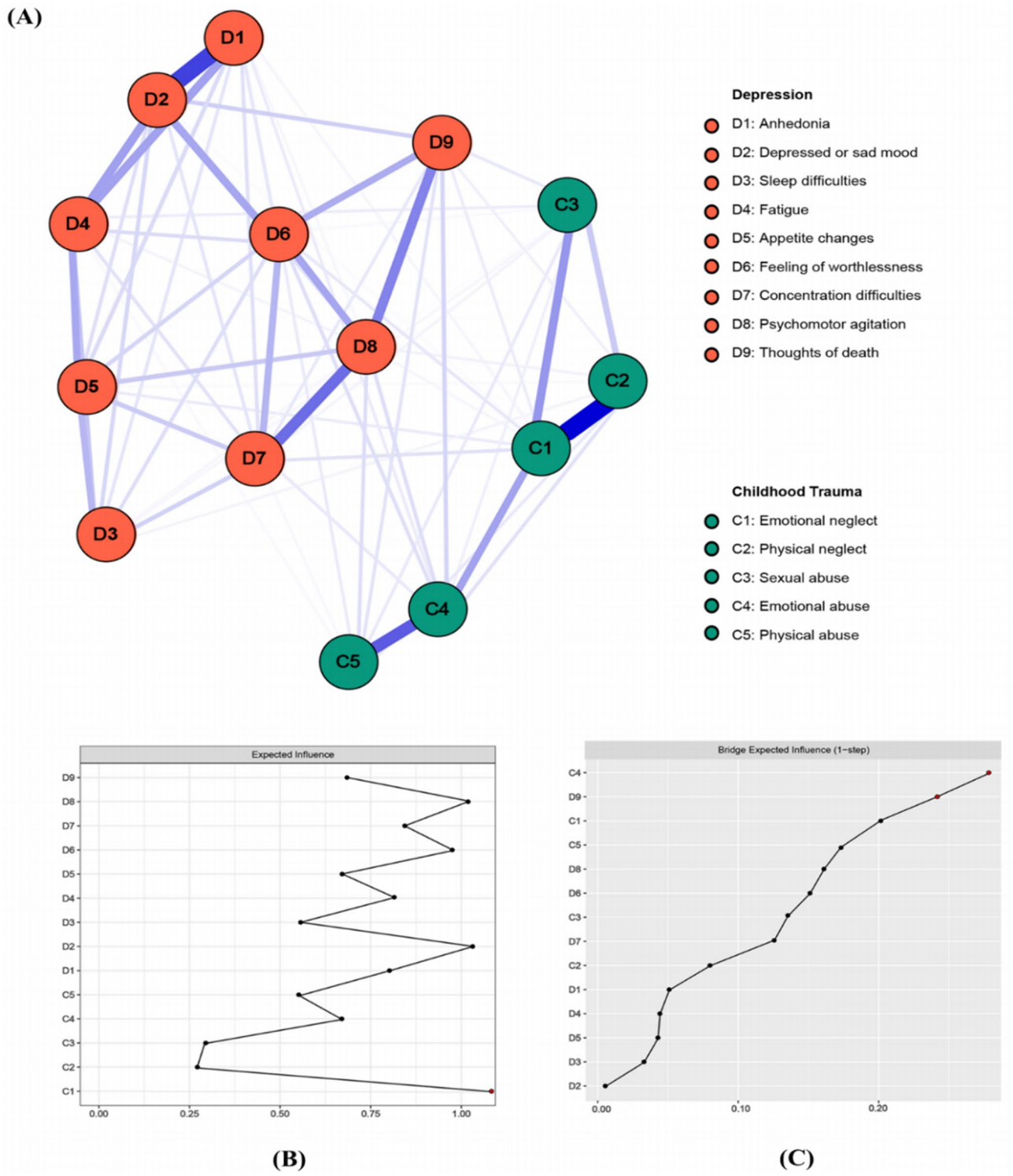


Fig. 2 Network model of CT-depression and the EIs and BEIs of the nodes in the network. **(A)** Network model of CT-depression. The blue line represents a positive partial correlation, while the red line represents a negative partial correlation. A thicker line and a more saturated color represent a larger partial correlation coefficient. **(B)** The EI indices of the nodes in the network (raw values). **(C)** The BEI indices of the nodes in the network (raw values)

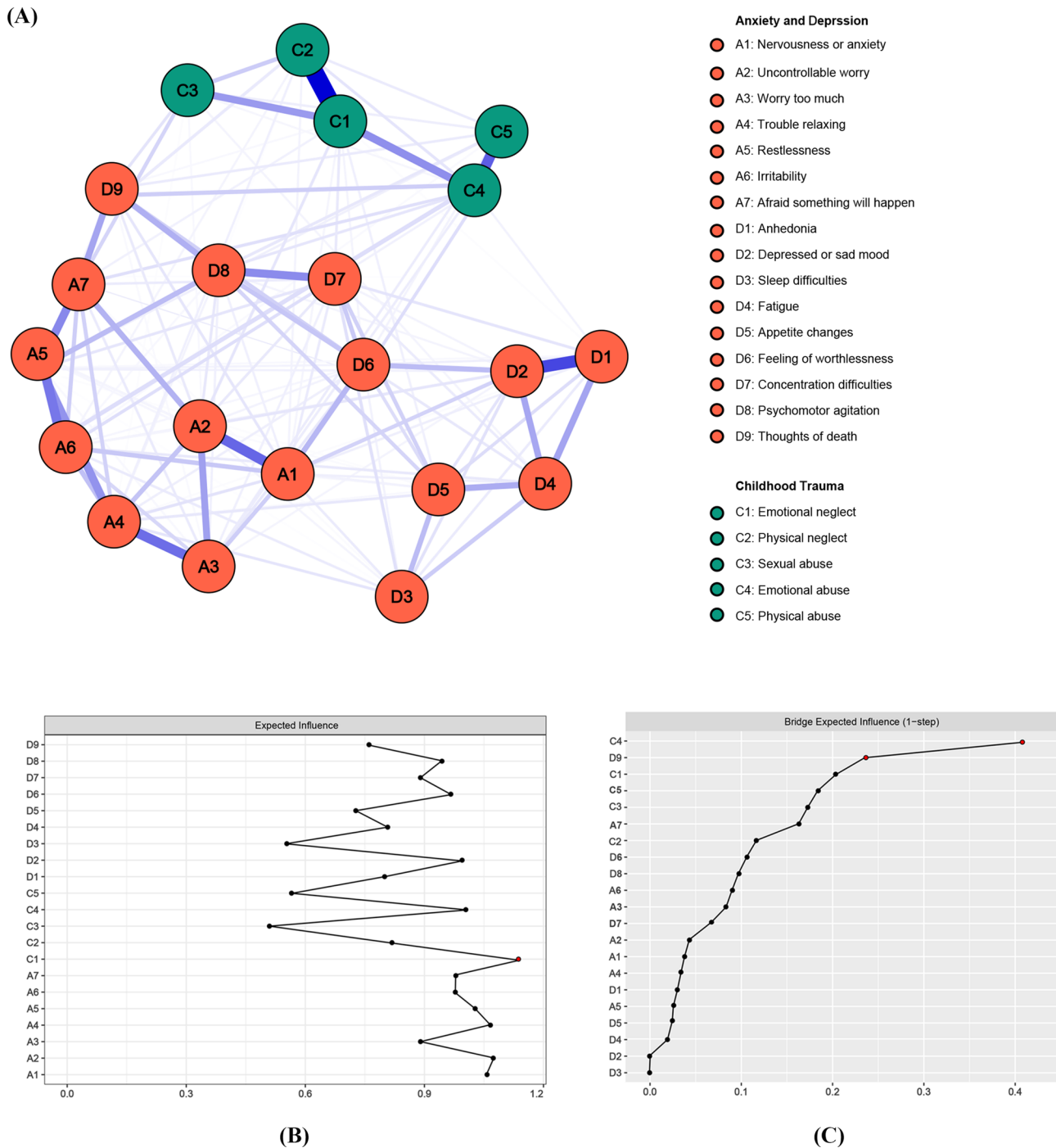


Fig. 3 Network model of CT comorbidity and the EIs and BEIs of the nodes in the network. (A) Network model of CT comorbidity. The blue line represents a positive partial correlation, while the red line represents a negative partial correlation. A thicker line and a more saturated color represent a larger partial correlation coefficient. (B) The EI indices of the nodes in the network (raw values). (C) The BEI indices of the nodes in the network (raw values)

Discussion

This study employed a network analysis approach to delineate, at the symptom level, the psychopathological pathways linking specific subtypes of CT to anxiety, depression, and their comorbidity among Chinese college students. By constructing three distinct regularized

partial correlation networks, we aimed to (a) identify robust symptom-level associations between the five CT subtypes and symptoms of anxiety, depression, and their comorbidity; (b) determine the central and bridge nodes within these networks; and (c) elucidate potential trans-diagnostic mechanisms. Our principal findings revealed

stable and specific associations: sexual abuse (C3) was strongly linked to the anxiety symptom “afraid something will happen” (A7), whereas emotional abuse (C4) exhibited a strong connection with the depressive symptom “thoughts of death” (D9). According to centrality analyses, emotional neglect (C1) was found to be the most central node across all the networks. Bridge centrality analyses further highlighted emotional abuse (C4), “afraid something will happen” (A7), and “thoughts of death” (D9) as key bridge nodes connecting the CT cluster to anxiety, depression, and comorbidity symptom clusters. These findings are largely consistent with our hypotheses: as expected, emotional neglect emerged as the most central hub across networks, whereas emotional abuse functioned as a transdiagnostic bridge node. Importantly, network robustness tests confirmed the stability of both the network structures and centrality indices, supporting the reliability and repeatability of these findings.

Symptom-level pathways in the CT-Anxiety, CT-Depression, and CT-Comorbidity networks

Within the CT-anxiety network, sexual abuse (C3) exhibited the strongest positive correlation with “afraid something will happen” (A7), thus suggesting a core pathological pathway from a specific CT type to anxiety symptoms. These findings align with previous research indicating that childhood sexual abuse is a significant risk factor for internalizing disorders, with anxiety and depression being especially prominent among adolescents [55, 56]. As one of the most severe forms of child maltreatment, sexual abuse fundamentally involves the loss of individual autonomy and the systematic erosion of security, often accompanied by profound feelings of helplessness, shame, and loss of control [57–59]. This disruption of fundamental security not only may impair the development of secure attachment, potentially fostering insecure attachment styles [60], but also may contribute to the formation of maladaptive early schemas such as “the world is dangerous” [61]. This process can be understood through the lens of attachment theory [62, 63], which posits that when a caregiver becomes a source of threat rather than safety, the child’s internal working models of self and others are fundamentally altered, leading to chronic expectations of harm and betrayal. Empirical research further indicates that individuals with a history of childhood maltreatment exhibit heightened threat-related attention biases, reflecting a cognitive processing pattern that prioritizes the detection of potential danger in the environment [64]. In addition, neurobiological evidence suggests that childhood sexual abuse is associated with structural and functional alterations in the amygdala and prefrontal cortex, regions central to fear conditioning and emotion regulation, and their disruption may contribute to the chronic hypervigilance

observed in survivors [65, 66]. Accordingly, individuals may remain in a state of hypervigilance toward potential threats [67, 68]. While prior research has examined these mechanisms primarily at theoretical and neurobiological levels, the present study extends this work to the symptom level by demonstrating that this heightened vigilance may manifest specifically as the anxiety symptom “afraid something will happen” (A7), which may serve as a defensive strategy that preempts anxiety to counter uncontrollable risks [69]. Notably, this specific pathway (C3→A7) remained equally robust in the CT-comorbidity network, underscoring its stability and suggesting that hypervigilance rooted in sexual abuse may be a pervasive factor contributing to anxiety symptoms even within comorbid presentations.

With respect to the CT-depression network, emotional abuse (C4) exhibited the strongest connection with “thoughts of death” (D9) and served as a pivotal bridge symptom. This core pathway (C4→D9) was replicated with comparable strength in the CT-comorbidity network. Our findings align with and extend recent network analyses, which have consistently identified emotional abuse as the childhood maltreatment subtype most robustly linked to suicidal ideation in adolescent and young adult samples [41, 70]. This consistency highlights emotional abuse as a stable, transdiagnostic factor that may be involved in suicidal thinking.

The stability of this symptom-level pathway can be parsimoniously explained by the interpersonal theory of suicide (IPTS [71, 72]). The IPTS posits that suicidal ideation emerges from the confluence of “thwarted belongingness” and “perceived burdensomeness.” A growing number of studies have shown that prolonged emotional abuse is a potent distal risk factor for the development of thwarted belongingness and perceived burdensomeness [73–75]. This association is theoretically coherent. As defined in the foundational literature, emotional abuse involves acts that communicate to the child that they are worthless, flawed, unloved, unwanted, or of value only in meeting another’s needs [76]. Meta-analytic evidence suggests that such emotional abuse is particularly damaging to the development of self-worth and emotion regulation capacities [21, 23, 27]. According to the hopelessness theory of depression, children who experience such emotional abuse may internalize the negative causal attributions communicated by the abuser, forming a stable depressogenic cognitive style that persists into adulthood [77, 78]. Notably, a prior network analysis in a clinical sample found that emotional abuse was centrally linked to suicidality specifically via perceived burdensomeness [79], directly aligning with our identified C4→D9 bridge pathway—a link that remains robust even in the presence of comorbid anxiety symptoms.

Thus, our network provides a precise, symptom-level pattern that is consistent with this mechanism. Emotional abuse may cultivate perceived burdensomeness by severely compromising the ability to regulate emotions, potentially fostering helplessness and a self-schema of being a burden [80, 81], a process documented in research on childhood maltreatment and emotional dysregulation [82]. The robust and replicable C4→D9 link suggests that this pathway may play an important role in suicidal ideation within the symptom architecture of both pure depression and its comorbidity with anxiety. However, as with all mechanistic interpretations in this study, these pathways remain preliminary and require longitudinal validation before causal conclusions can be drawn.

Central symptoms in the CT-Anxiety, CT-Depression, and CT-Comorbidity networks

Beyond mapping specific symptom pathways, we examined the core architecture of the three networks using EI metrics. The results indicated that emotional neglect (C1) consistently exhibited the highest EI across all the networks, underscoring its paramount connectivity and central position [37]. From a network perspective, this finding identifies emotional neglect as a pivotal hub within the internalizing symptom network of college students, suggesting that it plays a critical role in network dynamics and represents a prime target for monitoring and intervention [83].

The centrality of emotional neglect can be understood through its underlying mechanism. Unlike the “active” harm of emotional abuse, emotional neglect constitutes “passive” deprivation [84], characterized by a chronic failure to meet core needs for emotional security and belonging [85]. Whereas emotional abuse may directly instill negative self-beliefs through explicit criticism and hostility, emotional neglect may exert its effects through the absence of necessary emotional input [86]. This systemic deprivation may foster maladaptive schemas (e.g., “I am unloved/unworthy”) [61, 86], which may impair emotion regulation and undermine self-worth [87, 88], thereby potentially contributing to a broad vulnerability to anxiety and depression [89]. These emotion regulation deficits, in turn, have been suggested to mediate the pathway from early neglect to psychopathology [90]. Specifically, maladaptive cognitive emotion regulation strategies, particularly rumination and catastrophizing, have been shown to mediate the association between emotional neglect and both anxiety and depression in Chinese adolescents [91]. Notably, compared to sexual and physical abuse, emotional neglect has been found to be uniquely associated with anhedonic symptoms of depression [34]. Moreover, in college students, such deprivation may erode perceived control and regulatory

emotional self-efficacy, forming a key pathway to depression [92]. Crucially, the unique significance of emotional neglect is supported by evidence that controls for the co-occurrence of multiple adversities. In a large-scale study of adults with affective disorders, Spinhoven et al. found that emotional neglect demonstrated particularly specific associations with depressive disorders, dysthymia, and social phobia, even after accounting for comorbidity and the clustering of other childhood adversities [93]. Longitudinal evidence further corroborates this independent status: Hovens et al. followed adults with depressive and/or anxiety disorders over two years and reported that emotional neglect was specifically associated with a chronic course of depression and comorbid anxiety, an effect mediated primarily through higher baseline depressive symptom severity [94].

Particularly noteworthy is the developmental nuance revealed by our findings. While our study identified emotional neglect as the central node in college students, recent network analyses conducted exclusively with middle school student samples have consistently pointed to emotional abuse as the core node [95, 96]. This pattern of divergence raises the possibility of a meaningful developmental shift: during early and middle adolescence, the overt “threat” of abuse may function as the most salient driver of internalizing symptom networks, whereas by the transition to young adulthood, the chronic “deprivation” inherent in emotional neglect may become a more prominent factor. The consistent centrality of emotional neglect across our anxiety, depression, and comorbidity networks suggests that it may play an important role in maintaining internalizing psychopathology in this population. However, as these findings are preliminary and largely exploratory, further research is needed to validate and clarify the nature of this observed pattern.

Bridge symptoms in the CT-Anxiety, CT-Depression, and CT-Comorbidity networks

To determine whether the roles played by specific CT subtypes in connecting to depression, anxiety, and their comorbidity are coherent, we calculated the BEI index [38]. The results show that emotional abuse (C4) has the highest BEI across all the networks, indicating that it is a critical bridge node. These findings suggest that emotional abuse may function as a transdiagnostic influencing factor, thus propagating the adverse effects of CT across anxiety, depression, and their comorbidity. This aligns with latent variable studies documenting strong associations between emotional abuse and both disorders [97, 98]. It is also consistent with recent network analyses identifying emotional abuse as a key bridge node in both clinical and community adolescent populations [70, 71].

The transdiagnostic bridging role of emotional abuse may be explained through its distinct psychological

impact. Unlike emotional neglect, which operates through passive deprivation [84], emotional abuse is an active threat involving direct verbal hostility, humiliation, and criticism [76]. Such experiences directly instill negative self-schemas, specifically beliefs of being worthless and being in constant danger [86, 89]. These schemas may simultaneously fuel the hypervigilance and catastrophic worry central to anxiety and the hopelessness and self-blame central to depression [99, 100]. Long-term exposure to emotional abuse often engenders pervasive hopelessness [101], which, in turn, may sustain both catastrophic anticipation of threat and profound feelings of despair [102]. These cognitive-affective states are further reinforced by maladaptive cognitive emotion regulation strategies: catastrophizing serves as a more effective predictor of anxiety, whereas self-blame plays a prominent role in predicting depression [103–105]. Through these dual cognitive pathways, emotional abuse may concurrently activate both anxiety- and depression-spectrum symptoms, providing a plausible mechanistic account of why it emerged as the strongest bridge node.

Consistent with this account, emotional abuse was identified as a critical bridge node alongside “afraid something will happen” (A7) in the CT–anxiety network. It also consistently formed the strongest bridge connection with “thoughts of death” (D9) in both the CT–depression and comorbidity networks. These connections suggest two complementary symptom-level pathways. First, emotional abuse may sensitize individuals to uncertain threat, activating the catastrophic anticipatory worry captured by A7. Second, it may erode perceived value and safety in the world, thereby facilitating the hopelessness and despair that culminate in suicidal ideation captured by D9 [71]. Although the temporal ordering of these pathways remains speculative without longitudinal evidence, they align with the network-theoretic premise that high-BEI nodes function similarly to shared risk factors capable of activating symptom clusters across disorders [38].

Given that comorbidity often arises from common underlying risk factors [106], the present findings provide network-level evidence that emotional abuse is a stable transdiagnostic bridge factor. Accordingly, the identified bridge nodes represent potential priority targets for prevention and intervention [38]. An integrated approach that addresses emotional abuse (C4) alongside the key symptoms it may activate—specifically A7 and D9—could theoretically enhance intervention effectiveness for anxiety, depression, and their comorbidity in this population. For example, trauma-focused work can modify maladaptive schemas originating from emotional abuse [86]. Exposure-based techniques can reduce catastrophic worry [107], cognitive restructuring can target hopelessness and self-blame [108], and safety planning can directly address suicidal ideation [109]. Such an approach

aligns with transdiagnostic treatments that integrate trauma processing with emotion regulation training [110]. It must be stressed, however, that this proposed intervention framework is derived from cross-sectional data and requires rigorous validation through longitudinal research and controlled clinical trials before any clinical recommendations can be made.

Theoretical and clinical implications

The findings of the present study have important theoretical and clinical implications. Theoretically, this study provides a symptom-level map of the pathways linking specific CT subtypes to anxiety, depression, and their comorbidity. Moving beyond sum-score approaches reveals distinct associations that were previously overlooked, such as between sexual abuse and the anxiety symptom “afraid something will happen” (A7) and between emotional abuse and the depressive symptom “thoughts of death” (D9). According to the centrality and bridge centrality analyses, emotional neglect is the most central node and emotional abuse, A7, and D9 are key bridge symptoms. These findings position CT (and particularly its emotional subtypes) as a critical transdiagnostic risk factor and help to clarify the precise symptom-level mechanisms underlying internalizing disorders.

Clinically, the identified central and bridge nodes represent prime targets for precision intervention grounded in network theory. For assessment, systematic screening for CT, with particular attention to emotional neglect and abuse, should be integrated into routine mental health evaluations for college students. For intervention, strategies should aim to modify these key network nodes. This involves addressing emotion regulation deficits stemming from emotional neglect, restructuring maladaptive self-schemas related to emotional abuse, and directly targeting CT-driven symptoms such as catastrophic worry (A7) and suicidal ideation (D9). Focusing on these core mechanisms is likely to increase the efficacy of interventions aimed at reducing anxiety, depression, and their comorbidity in this population.

Limitations and directions for future research

This study has several limitations. First, the cross-sectional design precludes causal inference regarding the symptom-level pathways identified. Future longitudinal or experience sampling studies are needed to establish their temporal dynamics and causality. Second, our assessment captured broad CT subtypes but not nuanced characteristics such as severity, timing, or frequency. Integrating these multidimensional indicators in future work will provide a more detailed understanding. Third, reliance on retrospective self-report data may introduce biases such as memory distortion. Given that

participants' current depressive or anxious states may influence how they recall and report childhood experiences, the observed associations may be partially inflated by mood congruent recall. Multimethod designs incorporating clinical interviews or prospective measures would be valuable for future replication and would help disentangle the effects of actual trauma exposure from recall biases. Finally, the generalizability of the findings may be limited by the nonclinical student sample, and the model did not include other influential factors, such as genetics or social support. Direct clinical application therefore requires caution, and testing this model in broader clinical and community populations is an essential next step.

Despite these limitations, the primary aim of this study, which was to map the symptom-level architecture linking CT to internalizing symptoms and to identify central and bridge nodes as high-priority candidates for intervention, was successfully achieved. The stable, specific, and theory-consistent patterns observed across three distinct networks provide a valid and robust foundational blueprint for generating causal hypotheses in future research.

Conclusion

This study is the first to use network analysis to simultaneously examine the symptom-level interactions among CT subtypes, anxiety, depression, and their comorbidity. Our findings elucidate the specific pathways linking distinct CT subtypes to individual anxiety and depressive symptoms. Centrality analyses were used to identify emotional neglect as the most central node across networks. According to bridge centrality analyses, emotional abuse, "afraid something will happen," and "thoughts of death" were identified as key bridge symptoms, delineating precise transdiagnostic pathways that connect CT to the symptom clusters of anxiety, depression, and their comorbidity. These findings offer specific targets for refined screening and intervention in campus mental health practice.

Abbreviations

CT	Childhood trauma
CTQ-SF	Childhood trauma questionnaire-short form
GAD-7	Generalized anxiety disorder 7-Item scale
PHQ-9	Patient health questionnaire-9
LASSO	The least absolute shrinkage and selection operator
EBIC	Extended Bayesian information criterion
CI	Confidence interval
CS	Correlation stability
EI	Expected influence
BEI	Bridge expected influence

Supplementary Information

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Supplementary Material 1

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Author contributions

Concept and design: Y.H, N.C, W.L; Acquisition of the data: Y.H, J.Y; Analysis and interpretation of the data: Y.H, Y.D, Y.S; Drafting of the manuscript: Y.H. Critical revision of the manuscript: Y.H, N.C, W.L.

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Data availability

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

Declarations

Ethical approval

This study was approved by the Ethics Committee of Shanghai Normal University (No 2025–005) and conducted in accordance with the Declaration of Helsinki. All participants have given their informed consent, and all methods were carried out in accordance with relevant guidelines and regulations.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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