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ORIGINAL RESEARCH

Understanding Pathways from Cognitive Biases to the Risk of Psychosis: A Network Analysis Approach

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Purpose: Although the linkage between cognitive biases and psychotic-like experiences (PLEs) is well established, the knowledge of potential mechanisms of this relationship is still unknown. The aim of the present study was to better understand the structure of connections between cognitive biases and PLEs by considering at the same time the role of childhood trauma and depressive symptoms in a non-clinical adolescent sample (aged 14–19 years).

Methods: PLEs were measured using the Community Assessment of Psychic Experiences (CAPE-P15), cognitive biases were assessed with the Davos Assessment of Cognitive Biases Scale-42 (DACOBS-42), depressive symptoms were evaluated using the Patient Health Questionnaire-9 (PHQ-9) and exposure to childhood traumatic life events was measured using the Childhood Trauma Questionnaire (CTQ-SF). A network analysis was conducted to examine the interrelationships between these variables.

Results: The most central nodes in the network were the cognitive bias items "belief inflexibility", "safety behaviors", and "subjective cognitive problems". Shortest path analyses revealed that depressive symptoms played a significant mediating role between cognitive biases and PLEs. Specifically, the shortest pathways from cognitive biases item "subjective cognitive problems" to PLEs items P7 (subjective cognitive problems), P8 (thought own), and P11 (control force) involved depressive symptoms, including items related to "guilt", "concentration", "motor", and " suicide".

Conclusion: Our findings highlight the central role of cognitive distortions and emotional symptoms within the psychosis-risk network. Depressive symptom nodes serve as critical mediators between subjective cognitive problems and PLEs, underscoring their pivotal function in driving the development of PLEs among adolescents with cognitive biases. These results suggest a tight interconnection between emotional and cognitive processes in psychosis vulnerability, emphasizing the need for integrated interventions targeting both domains.

Keywords: psychotic-like experiences, depressive symptom, childhood trauma, adolescents

Introduction

Psychotic-like experiences (PLEs) refer to subclinical symptoms including delusion-like experiences and perceptual abnormalities,[1] affecting 5–7% of adults in the general population. Most individuals who suffer from PLEs achieve remission,[2] but approximately 7% of the population with PLEs develop into full-blown psychosis, and about 20% continue to experience persistent PLEs.[1] PLEs are common among adolescents, with a prevalence of 7.5%,[3] and are associated with a higher risk of developing psychiatric disorders. These include psychotic disorders (eg, schizophrenia) and a broad spectrum of common mental health conditions, such as anxiety, depression, substance misuse, suicide risk, and self-harm.[4–9]

Numerous studies have explored factors associated with the onset and persistence of PLEs. Psychosocial factors such as poor social functioning, cannabis use, and a history of childhood trauma have been identified as significant contributors.[10–14] Nevertheless, the mechanisms underlying this subclinical phenomenon remain poorly understood. A review of 66 articles

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reported that, in healthy and ultra-high-risk samples, PLEs are positively associated with cognitive biases, including attention to threat, externalizing bias, belief inflexibility, and jump to conclusion.[15] Jumping to conclusion plays an important role in the development and maintenance of psychosis. It has been observed in individuals with PLEs and is associated with familial risk of psychosis during preadolescence.[15,16] Furthermore, external attribution bias and avoidant safety behaviors have been linked to the onset of PLEs.[15] According to the cognitive model of psychosis, cognitive biases are critical in the development and maintenance of PLEs.[17,18] The onset and persistence of psychosis result from the interaction of biological predispositions, socio-environmental factors, emotional symptoms, and cognitive distortions.[19] However, few studies have elucidated the role of cognitive distortions in increasing the risk of psychosis.

Research has suggested that childhood trauma and emotional symptoms were involved in the potential mechanisms underlying the risk of psychosis.[20,21] Childhood trauma is widely regarded as a critical factor in the onset and development of psychosis.[11,20] The significance of sexual and emotional trauma has long been recognized,[22] and the interaction between childhood trauma and cognitive biases in increasing the risk of psychosis has been reported.[23] Additionally, depressive symptoms are among the most common disorders affecting adolescents.[24] Cognitive models have also highlighted the role of emotional distortions in the development and maintenance of positive psychotic symptoms.[25] Depressive symptoms have been identified as a risk factor for the onset of PLEs.[26,27] Individuals with depressive symptoms have a higher risk of developing PLEs than those without, and young adults with mood disorders are more likely to report PLEs.[26,27] Previous cross-sectional studies reported that adolescents with PLEs were 4 to 6 times more likely to report depressive symptoms compared to those without PLEs.[26,28,29]

Although substantial evidence suggests an association between PLEs and cognitive biases, childhood trauma, and mood symptoms, the specific characteristics of these psychosocial factors most closely related to PLEs remain unclear. Recently, network analysis has been proposed as a valuable tool for understanding the interconnectedness of psychopathological symptoms.[30] Several studies have used network analysis to investigate psychosocial factors related to the risk of psychosis beyond psychopathological symptoms. Most of these studies focus on possible pathways from childhood adversity to PLEs. For example, Qiao and et.al found that childhood trauma is related to PLEs through general psychopathological symptoms (eg, anxiety, hostility, and somatization), with affective symptoms playing a central role in this network.[11] Similarly, one study showed that childhood trauma is related to positive and negative psychotic symptoms through general psychopathological symptoms, including poor impulse control and motor retardation.[31] However, another study reported that neither cognitive biases nor depressive symptoms mediated the pathway between the most central traumatic life event and PLEs.[22]

This study aimed to expand upon prior findings regarding the network structure of psychosis risk factors. Although previous studies have demonstrated that cognitive biases, childhood trauma, and depressive symptoms contribute to the onset and maintenance of PLEs, the interconnected pathways among these factors remain unclear, particularly the pathways linking cognitive biases to PLEs. Therefore, the present study aimed to identify the most central cognitive biases and PLEs within the network and determine the shortest pathways connecting them.

Materials and Methods

Participants

A total of 4087 adolescents (2,026 females, 2,061 males), aged 14–19 years, were recruited from three senior high schools in Hunan Province, China. A history of psychiatric diagnosis was screened based on self-reported questionnaires. Sociodemographic and clinical data were also collected.

This study was approved by the Institutional Review Board (IRB) of the First Affiliated Hospital of Hunan University of Chinese Medicine. Informed consent from parents and informed assent from participants were obtained before the commencement of study. All procedures were conducted in accordance with the Declaration of Helsinki.

Measures

PLEs were assessed using the Community Assessment of Psychic Experiences (CAPE-P15).[32] The CAPE-P15 consists of two subscales assessing the frequency and distress of PLEs across 15 items. The items are grouped into three factors: persecutory ideation (PI), bizarre experiences (BE), and perceptual abnormalities (PA). Each item was rated on a four-point Likert scale, from 1 (never), 2 (sometimes), 3 (often), to 4(nearly always). A mean cut-off score of 1.47 on both subscales (frequency and distress) was used to screen for ultra-high risk of psychosis.[33] The Chinese version of the CAPE-P15 has demonstrated satisfactory psychometric properties among adolescents,[34] with a Cronbach's α of 0.896 in the current study.

Depressive symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9), a widely utilized tool for screening and assessing the severity of depression.[35] The PHQ-9 consists of nine items rated on a scale of 0 to 3, with total scores ranging from 0 to 27. Higher scores are indicative of a greater presence of depressive symptoms. The Chinese version of the PHQ-9 has shown reliable psychometric properties,[36] with a Cronbach's α of 0.891 in the current study.

Cognitive biases were evaluated using the Davos Assessment of Cognitive Biases Scale (DACOBS).[37] The DACOBS comprises 42 items rated on a 7-point Likert scale, with higher scores reflecting greater cognitive biases. The scale encompasses seven subscales: jumping to conclusions, belief inflexibility, attention to threat bias, external attribution bias, social cognition problems, subjective cognitive problems, and safety behaviors. The Cronbach's α for the DACOBS was 0.921 in this sample.

Childhood trauma experienced before the age of 16 was assessed using the short form of the Childhood Trauma Questionnaire (CTQ-SF).[38] The CTQ-SF is a 28-item questionnaire using a seven-point Likert scale (1–5). Of the 28 items, 25 are distributed across five subscales: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. The remaining three items are designed to test the validity of the questionnaire. This scale is considered to be a sensitive and valid screening questionnaire for childhood trauma. Four subscales (emotional abuse, physical abuse, emotional neglect, and physical neglect) were utilized in this study, with a Cronbach's α of 0.697.

Data Analysis

Firstly, we estimated the network where each node represents scores of the 35 variables from the scales: CAPE-P15, DACOBS, PHQ-9, and CTQ-SF. Nodes are connected by edges, which represent the relationship between these variables. Thereafter, the network with the use of Mixed Graphical Models that are designed for mixed data was conducted.[39] Variables on the four-point scales (the frequency subscale of CAPE-P15 and PHQ-9) and the seven-point scale (DACOBS) were treated as continuous data. Scores from the CTQ-SF representing exposure to childhood trauma (CT) were coded as binary variables. To estimate a sparse network and control for potential spurious connections, we applied an L1-penalty regression (LASSO) selected by Extended Bayesian Information Criterion (EBIC) with the default regularization parameter lambda (0.5, as suggested in the literature).[40,41] To assess the variability of edge-weight accuracy, we performed supplementary analyses of nonparametric bootstrapped 95% confidence intervals and difference tests.

Secondly, we estimated node strength, node closeness, node betweenness, node expected Influence and node predictability in the network.[42] Node strength indicated that the importance of each node in the network.[43] Node closeness measures the average distance of a node to all other nodes in the network, it provides an indication of how quickly information can spread from a particular node to other parts of the network. Node betweenness measures the number of shortest paths that pass through a particular node, it indicates the extent to which a node acts as a bridge or bottleneck in the network. Node predictability measured the variance of how much of a node can be explained by the connected nodes.[44] Furthermore, we assessed the stability of centrality measures using subsetting bootstrap, which provides a centrality-stability coefficient (CS-coefficient).[43] Following Epskamp et al (2018), the CS-coefficient should not be below 0.25 and preferably should exceed 0.5 for reliable interpretation.[35] We also performed the strength centrality difference test that calculates which centrality estimates differ from each other significantly.

In addition, we employed Dijkstra's algorithm to compute shortest pathways, determining the minimal number of steps required to traverse between any two nodes in the network.[31,45,46] This analytical approach elucidates potential

influence pathways and identifies mediating variables that may link cognitive biases to PLEs. Specifically, the algorithm identifies the most efficient (ie, shortest) connection pathway among all possible routes between node pairs. The network utilizes undirected edges to represent conditional dependencies between variables. Importantly, the edge-weight parameters quantify the strength of unique associations between variable pairs. These weighted connections not only demonstrate statistical relationships but may also suggest plausible causal pathways warranting further investigation.

Mediation analyses were conducted using the PROCESS macro for SPSS version 4.1.[47] We followed the Baron and Kenny method to confirm whether the cognitive biases core symptoms (the independent variables) predict PLEs core symptoms through the mediation of those nodes identified in our network analyses as those included in the shortest pathways between cognitive biases core symptoms and PLEs core symptoms.[48] The statistical significances of the mediating and indirect effects were assessed using bootstrapped bias-corrected percentile based confidence interval of 5000 bootstrap draws. Mediation analyses were performed using SPSS software.

Descriptive analyses were performed using the SPSS 18.0. Network analyses were conducted with R software (version 3.6.3). Specifically, we used the following packages: mgm package for network estimation and node predictability, qgraph package for network visualization, and bootnet package for stability analysis.

Results

Sample Characteristics

A total of 4,087 adolescents (2,026 females, 2,061 males), aged 14–19 years, participated in the study. Among them, 842 participants (20.6%) met the cutoff score for ultra-high risk of psychosis, and 2,121 participants (51.9%) reported a history of childhood trauma. The characteristics of the sample are summarized in Table 1.

Network Structure

The resulting network of 35 items representing exposure to childhood trauma, cognitive biases, depressive symptoms and PLEs, is depicted in Figure 1. The network was well connected and had no isolated nodes. Of the 595 edges, weights of

Table 1 Descriptive Characteristics of the Sample (n = 4087)

Variables	Mean ± SD or n (%)
Gender	
Male	2061(50.4%)
Female	2026(49.6%)
Age	16.16(0.88)
Residence	
Urban	2610(63.9%)
Town	694(17.0%)
Rural	783(19.2%)
PHQ-9 (total score)	6.18(5.20)
CTQ (total score)	35.42(10.12)
Participants with Childhood trauma	2121(51.9%)
Participants without Childhood trauma	1966(48.1%)
DACOBS (total score)	125.55(35.10)
Jumping to conclusions	19.81(6.35)
Belief inflexibility	17.63(6.13)
Attention to threat bias	22.12(6.30)
External attribution bias	15.29(6.21)
Social cognition problems	16.85(6.71)
Subjective cognitive problems	18.49(7.79)
Safety behaviors	15.32(6.46)
Frequency subscale of CAPE-P15 (total score)	1.36(0.41)

Abbreviations: CTQ, Childhood Trauma Questionnaire; DACOBS, Davos Assessment of the Cognitive Biases Scale; CAPE-P15, 15-item positive subscale of the community assessment of psychic experiences.

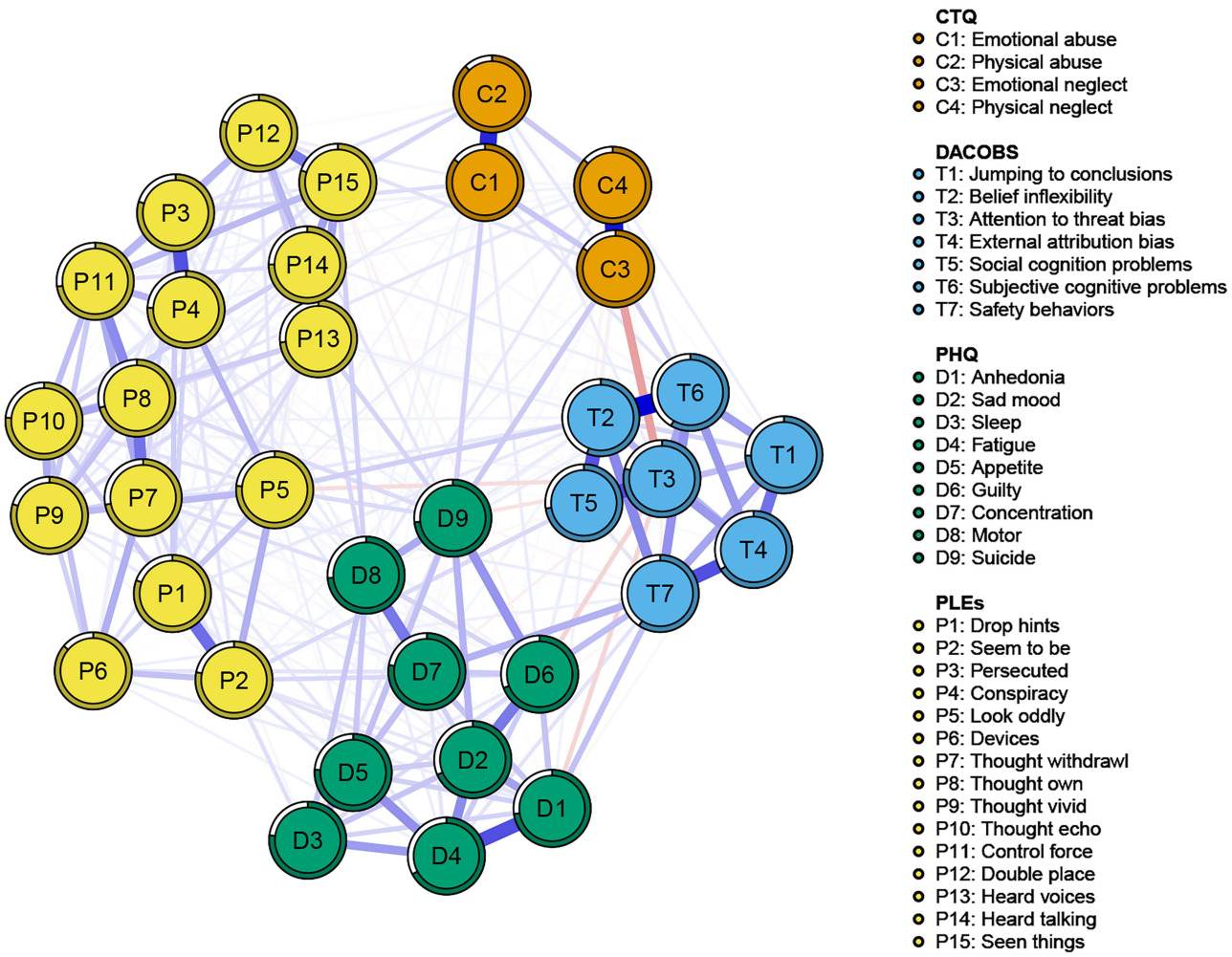

Figure 1 The network analyzed in the present study. The filled part of the ring around each node represents predictability of each node, ie, the proportion of variance of specific node explained by the nodes directly connected to it.

282 edges (47.39%) were non-zero and the mean weight of edges was 0.03. The edge weights are presented in Table S1. Among these connections, the most robust relationships across different domains were identified as follows: between cognitive bias item T3 (attention to threat bias) and childhood trauma item C3 (emotional neglect) (Edge weight= −0.14); cognitive bias item T7 (safety behaviors) and depressive symptom item D7 (concentration) (Edge weight=0.12); T7 (safety behaviors) and D6 (Guilty) (Edge weight=0.10); T7 (safety behaviors) and D1 (anhedonia) (Edge weight=0.09). Notably, T3 had a negative association with C3, while T7 was positively associated with D1, D7, and D6. Results of the analysis testing for the weights of these edges were illustrated in Figure S1. The correlation structure among the nodes is presented in Figure S2.

Nodes Centrality

Figure 2 illustrates the centrality indices of all nodes in the network, including Strength, Closeness, Betweenness, and Expected Influence. Cognitive bias items were particularly prominent, with item T2 (belief inflexibility, Z=2.25), along with T7 (safety behaviors, Z=1.99) and T6 (Subjective cognitive problems, Z=1.79), demonstrating the highest standardized strength. This suggests that they may act as influential nodes within the network. In other domains, the nodes with the highest strength included P11 (control force, Z=0.79), P8 (thought own, Z=0.65), and P7 (thought withdrawal, Z=0.64) for PLEs; D6 (guilty, Z=1.19), D4 (fatigue, Z=0.85), and D9 (suicide, Z=0.67) for depressive symptoms; and C3 (emotional neglect, Z=−0.29), C1 (Emotional abuse, Z=−1.36), and C4 (physical neglect, Z=−1.53) for childhood trauma.

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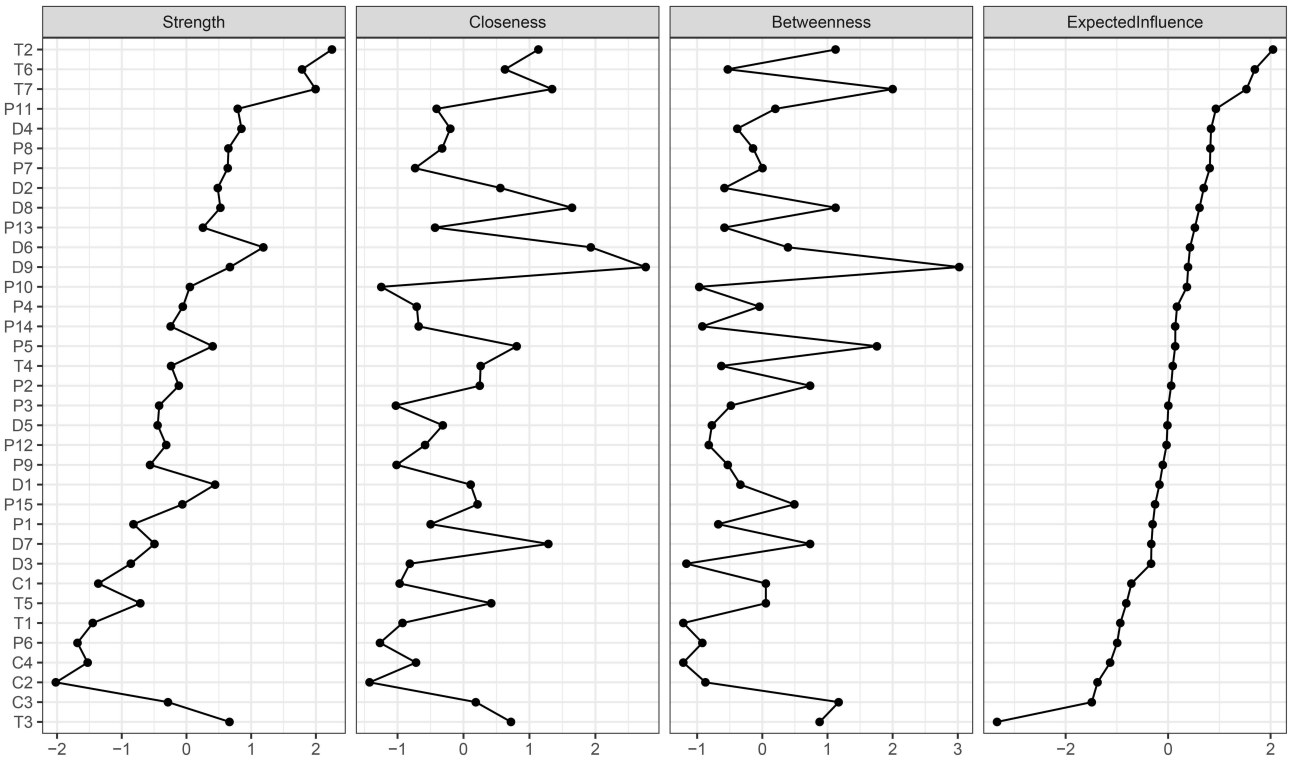

Figure 2 Centrality profiles of all variables. Panels show z-scores for Strength, Closeness, Betweenness, and Expected Influence.

In terms of standardized expected influence values, the most central nodes in terms of expected influence were still concentrated in cognitive bias items: T2 (belief inflexibility, Z=2.05), T6 (subjective cognitive problems, Z=1.70), and T7 (safety behaviors, Z=1.53), indicating their substantial overall impact on the network. Depressive items showed remarkable performance in Closeness and Betweenness centrality: D9 (suicide, Z=2.76), D6 (guilty, Z=1.93), and D8 (motor, Z=1.64) had the highest Closeness centrality, implying their strong connectivity to other nodes via short paths. Notably, D9 (suicide, Z=3.02) also demonstrated the highest Betweenness centrality, acting as a critical bridge between distinct network modules. The results of the analysis testing for between-node differences in the centrality indexes are illustrated in Figure S3.

Node Predictability

Node predictability ranges from 0.23 in node C2 (physical abuse) to 0.69 in node T2 (belief inflexibility), with an average of 0.43. This means that, on average, 43% of the variance of each node in the network can be explained by its neighbors. The average predictability of each domain was: 0.55 for cognitive biases, 0.47 for depressive symptom, 0.40 for PLEs and 0.26 for exposure to childhood trauma. The lowest average predictability for childhood trauma indicates that the variance in these nodes is not well explained by other nodes included in the network. Node predictability values for each item are presented in Table S2.

Pathways from Cognitive Biases to PLEs

Based on the results of network analysis, a shortest path network is established between the three pivotal nodes within cognitive biases, node T2 (belief inflexibility), node T6 (subjective cognitive problems), and node T7 (safety behaviors), which exhibit the highest strength, and the three most significant nodes in PLEs, specifically P7 (thought withdrawal), P8 (thought own), and P11 (control force), also characterized by the highest strength (Figure 3).

In Figure 3, the shortest routes from cognitive biases node T2 (belief inflexibility) and T6 (subjective cognitive problems) to PLEs node P7 (thought withdrawal) both traverse node P5 (look oddly); the shortest paths to PLEs node P8

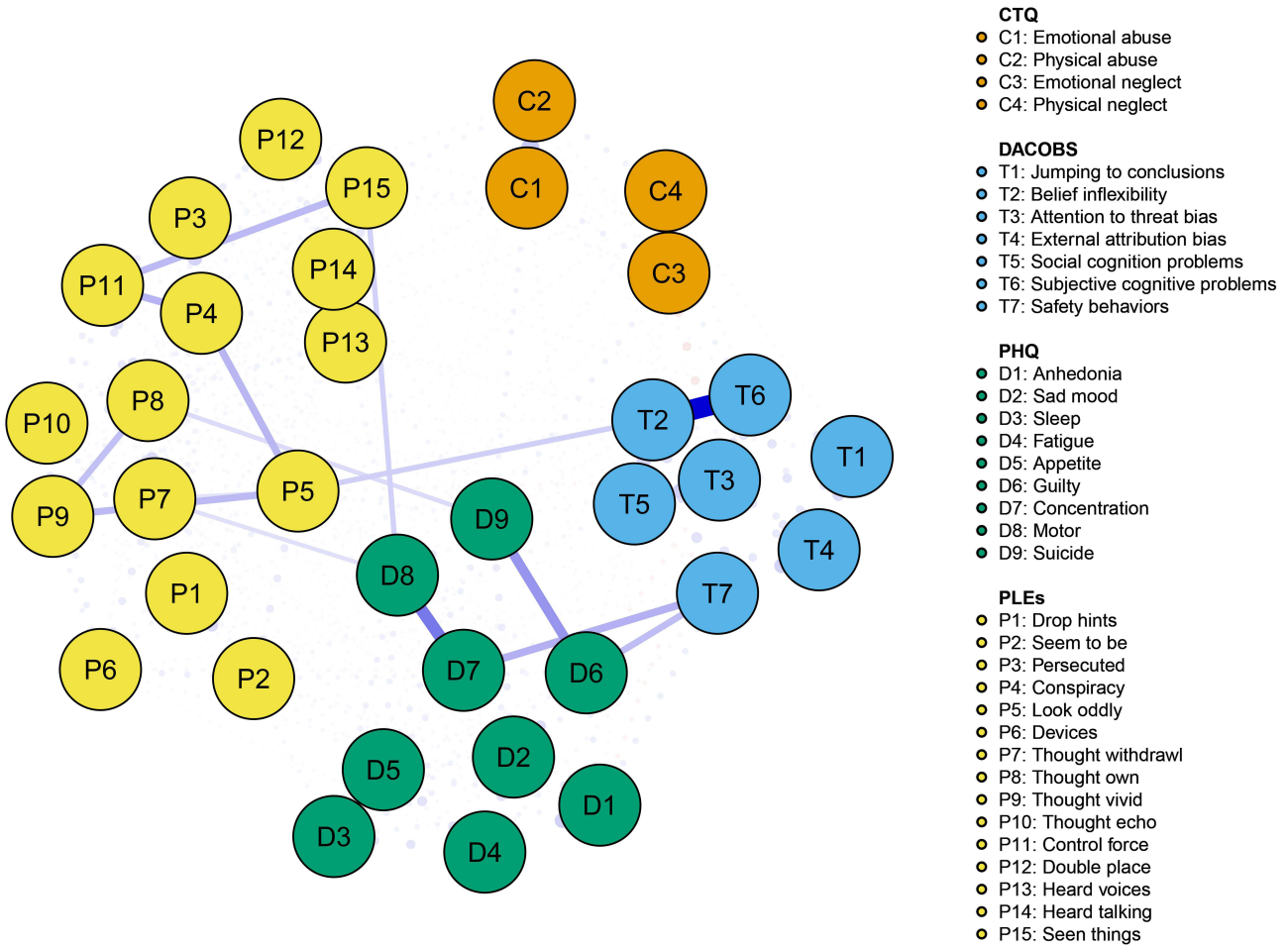

Figure 3 Networks displaying shortest routes from cognitive biases node T2 (belief inflexibility) and T6 (subjective cognitive problems) to PLEs node P7 (thought withdrawal), P8 (thought own), and P11 (control force). Dashed lines indicate connections existing within the network framework, but are less relevant when investigating shortest paths.

(thought own) involve sequential nodes P5 (look oddly), P7 (thought withdrawal), and P9 (thought vivid); and the shortest pathways to PLEs node P11 (control force) pass through nodes P5 (look oddly) and P4 (Conspiracy). Notably, the connections from node T2 (belief inflexibility) and T6 (subjective cognitive problems) to the three pivotal nodes of PLEs do not include any nodes of childhood abuse or depressive symptoms. For node T7 (safety behaviors), three distinct pathways link it to the three PLEs pivotal nodes: (1) T7 (safety behaviors) connects to node P7 (thought withdrawal) via nodes D7 (concentration) and D8 (motor); (2) T7 (safety behaviors) connects to node P8 (thought own) via nodes D6 (guilty) and D9 (suicide); (3) T7 (safety behaviors) connects to node P11 (control force) via nodes D7 (concentration), D8 (motor), and P15 (seen things). All pathways from node T7 (safety behaviors) to the three PLEs pivotal nodes pass through nodes of depressive symptoms.

Mediation Analyses

The results of the shortest path network analysis identify nodes D6, D7, D8, and D9 of depressive symptoms as critical intermediaries through which T7 (safety behaviors) can evoke PLEs symptoms P7 (thought withdrawal), P8 (thought ownership), and P11 (control force). To determine whether the association between cognitive biases (T7) and PLEs symptoms (P7, P8, P11) is mediated by these depressive symptoms, three separate mediation models are constructed and tested.

For P7 (thought withdrawal), the shortest pathway between T7 (safety behaviors) and P7 (thought withdrawal) involves D7 (concentration difficulties) and D8 (motor symptoms). Specifically, the mediation analysis reveals that D7

and D8 partially mediated the relationship between T7 and P7 (Indirect Effect: b = 0.0482, 95% CI [0.0391, 0.0582]). Regarding P8 (thought ownership), both D6 (guilt) and D9 (suicidal ideation) are identified in the shortest path from T7 (safety behaviors) to P8 (thought own). The second mediation model shows that T7 predicted P8 partially through D6 and D9 (Indirect Effect: b = 0.0046, 95% CI [0.0037, 0.0056]). And for P11 (control force), the shortest path includes D7 (concentration difficulties), D8 (motor symptoms), and P15 (perceptual disturbances). The third mediation model demonstrates that these variables collectively partially mediated the relationship of T7 and P11 (Indirect Effect: b = 0.0013, 95% CI [0.0009, 0.0017]). These findings collectively indicate that depressive symptoms (D6, D7, D8, D9) and P15 serve as partial mediators in the association between safety behaviors (T7) and nodes of PLEs (P7, P8, P11), highlighting their critical role in the cognitive-affective pathway underlying PLEs.

Network Accuracy and Stability

The CS coefficients were 0.75 for strength, 0.59 for closeness, 0.59 for betweenness, and 0.75 for expected influence, indicating that the network model was robust (Figure S4). The bootstrapped 95% CI ranges of edge weights were relatively narrow suggesting sufficient accuracy (Figure S5).

Discussion

The current article provides the first network-based analysis of the relationship between cognitive bias and PLEs in a general population, adolescent sample. We constructed a network that included seven dimensions of cognitive bias (ie, jumping to conclusions, belief inflexibility, attention to threat bias, external attribution bias, social cognition problems, subjective cognitive problems, and safety behaviors) and PLEs. Our results revealed that belief inflexibility, safety behaviors and subjective cognitive problems exhibited the highest centrality in the network, suggesting their prominent role in shaping the network's structure. The network topology revealed depressive symptoms - particularly guilt (D6), concentration difficulties (D7), motor symptoms (D8), and suicidal ideation (D9) - as crucial mediators between cognitive biases (safety behaviors, T7) and PLEs. Notably, these depressive symptoms showed exceptional network connectivity properties: items D6, D8, and D9 exhibited the highest closeness centrality values, while D9 additionally displayed the greatest betweenness centrality. These findings position these depressive symptoms as critical junctures in the cognitive bias-PLEs network architecture, consistent with previous research that supports the idea that cognitive biases contribute to PLEs through depressive symptoms,[26–29] highlighting the interconnectedness of cognitive and emotional processes in psychosis risk.

Our findings align with cognitive models that emphasize emotional and cognitive distortions as risk factors for psychosis.[17] Specifically, we observed an affective pathway from cognitive distortions to psychosis, where cognitive biases may lead to PLEs by heightening emotional distress, as represented by depressive symptoms. Depressive symptoms emerged as a crucial link between cognitive biases and PLEs in our model. Numerous studies have highlighted the importance of negative affective states as key co-occurring indicators across the psychosis continuum, including PLEs.[49–53] Previous research has confirmed the relationship between depressive symptoms and PLEs in non-clinical populations and found that depressive symptoms mediates the link between temperament, character and PLEs.[54,55] Our results further support the hypothesis that depressive symptoms mediate the relationship between cognitive biases and PLEs, emphasizing the significant role of emotional symptoms in the network of psychosis risk.

Furthermore, belief inflexibility, safety behaviors, and subjective cognitive problems emerged as central components in the network, with belief inflexibility showing the highest strength. This suggests that belief inflexibility has the most substantial relationship with other nodes in the network. Belief inflexibility refers to the inability to modify beliefs based on new or contradictory information, which is a key feature of delusional thinking.[25] Aberrant salience experiences—where individuals fail to consider alternative explanations—can maintain positive symptoms, such as delusions.[56] Previous research has indicated that lower belief flexibility is associated with stronger delusional symptoms, and belief inflexibility may prevent individuals from revising their interpretations of PLEs, thus contributing to psychosis risk.[57]

In addition to cognitive biases, guilt and emotional neglect exhibited the highest centrality in the depressive symptoms and childhood trauma domains, respectively. Despite the importance of emotional processes in the development and maintenance of psychotic symptoms, few studies have explored the emotional mechanisms linking cognitive

biases to PLEs. Our findings underscore the need for further investigation into the role of emotional processes, such as depressive and anxiety symptoms, in the network of PLEs. Clarifying these emotional mechanisms is crucial for understanding how cognitive biases and emotional disturbances interact in the onset of psychosis.

Several limitations of this study should be considered. First, while this study was conducted with a large non-clinical adolescent sample, the findings may have limited generalizability to clinical populations. Future studies involving individuals with psychotic disorders are needed to explore potential differences in the interconnectedness of psychopathological symptoms between clinical and non-clinical groups. Second, although self-reported PLEs have established reliability and validity, structured clinical interviews would provide more comprehensive clinical data and enhance the accuracy of PLE assessment. Notably, while the CTQ is a commonly employed tool for assessing childhood trauma, its internal consistency in this study approached borderline levels (eg, Cronbach's alpha = 0.697), which may compromise the reliability of trauma severity estimates. Replication with larger samples or more refined psychometric tools is warranted to strengthen these findings. Finally, this cross-sectional design limits our ability to draw conclusions about causal relationships. Longitudinal studies are necessary to examine the changes in cognitive distortions and their role in the development and maintenance of PLEs over time.

Conclusion

In conclusion, our study provides a comprehensive network analysis of the relationships between childhood trauma, depressive symptoms, cognitive biases, and PLEs in a large adolescent sample. We found that cognitive biases—especially belief inflexibility, safety behaviors, and subjective cognitive problems—were central in the network, with depressive symptoms playing a crucial mediating role between cognitive distortions and PLEs. These findings highlight the importance of cognitive distortions in the psychopathology of psychosis risk and suggest that emotional distortions, such as depressive symptoms, are critical pathways linking cognitive biases to PLEs.

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Disclosure

The authors report no conflicts of interest in this work.

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