

RESEARCH ARTICLE

Associations among psychosis, mood, anxiety, and posttraumatic stress symptoms: A network analysis

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Abstract

The associations among psychotic experiences (i.e., hallucinations and delusions), trauma exposure, and posttraumatic stress symptoms are complex and multidirectional. Using network analysis to understand how psychotic experiences and symptoms of posttraumatic stress disorder (PTSD) relate to one another may identify new interventional targets to treat comorbidity and its underlying pathological processes. This study aimed to use network analysis to examine the associations among psychotic experiences; negative symptoms of psychosis; and symptoms of PTSD, anxiety, and depression. In this population-based cohort study, 4,472 participants (36.7% male) were assessed for psychotic experiences, negative symptoms of psychosis, PTSD, anxiety, and depression at age 23 ($M = 23.86$ years, $SD = 0.520$) or 24 years ($M = 24.03$, $SD = 0.848$). Associations among symptoms were assessed via network analysis. Exploratory graph analysis identified three clusters of densely connected symptoms within the overall network: psychotic experiences; PTSD symptoms; and depressive and anxiety symptoms and negative symptoms of psychosis. Psychotic experiences had the strongest associations with other symptoms in the network, and symptoms of anxiety played a key role in bridging psychotic experiences, symptoms of PTSD, and depressive symptoms. Consistent with the stress reactivity and affective models for psychotic experiences, the results suggest that symptoms of anxiety and emotional distress (e.g., hyperarousal, panic) may have a key role in the development and maintenance of psychotic experiences and symptoms of PTSD. Targeting these symptoms may ameliorate symptom burden transdiagnostically.

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Childhood adversity and trauma are common (Bellis et al., 2014), and both are associated with an increased risk of psychotic experiences (PEs; i.e., hallucinations and delusions; Croft et al., 2019; Trotta et al., 2015; Varese et al., 2012), which persists even after adjusting for lifetime comorbid mental disorders, genetic risks, and socioeconomic adversity (Croft et al., 2019; McGrath, McLaughlin, et al., 2017). Psychotic experiences are necessary but not sufficient for a psychotic disorder diagnosis, and most individuals who report PEs do not have psychotic disorder (Croft et al., 2019). Estimates suggest that the population-attributable fraction for childhood and adolescent trauma on psychotic experiences is 45% (Croft et al., 2019; Varese et al., 2012). Many individuals who experience adverse childhood experiences and traumatic events also develop posttraumatic stress symptoms (PTSS), some of whom will meet the criteria for posttraumatic stress disorder (PTSD; Karatzias et al., 2019; Kessler et al., 2017). Per the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; American Psychiatric Association [APA], 2013), symptoms of PTSD include avoidance, reexperiencing, negative changes in thoughts and mood, and hyperarousal. The associations among trauma exposure, psychotic experiences, and PTSD are complex and likely multidirectional. Although evidence is consistent with a causal effect of trauma exposure on psychotic experiences, the latter also increases the likelihood an individual will be involved in further traumatic events (McGrath, Saha, et al., 2017). There appears to be a dose–response relationship between exposure to childhood adversity and increased PEs (Scott et al., 2007) suggestive of a causal pathway (Croft et al., 2019; McGrath et al., 2016). More severe and chronic trauma exposure is more likely to be associated with PTSD (Brewin et al., 2000) and psychotic outcomes (Croft et al., 2019) than other factors, such as parental loss, unintentional injury, or economic adversity (Arseneault et al., 2011; McGrath, McLaughlin, et al., 2017). A better understanding of the complex interplay between symptoms of PTSD and psychosis might provide insights into the pathological processes underlying these disorders and their comorbidity and offer new possibilities for prevention and treatment.

As the association between PTSD and psychotic outcomes is multifactorial and bidirectional, we used network analysis to examine this relationship at a symptom level using a transdiagnostic approach (Fried et al., 2018; Hardy et al., 2021; Isvoranu et al., 2017). Network theory is well placed to address transdiagnostic complexity, as it assumes that psychopathology arises from dynamic feedback loops between symptoms, or “nodes,” rather than a common latent variable (Armour et al., 2017; Hardy et al., 2021). Certain nodes play a more fundamental role within the network, with some connecting across diagnostic boundaries (i.e., “central” and “bridge” symptoms; Armour et al.,

2017; Hardy et al., 2021). These central and bridge symptoms are important in linking densely connected clusters of nodes nested within broader networks. Thus, network analysis suggests these central and bridge symptoms may represent promising treatment targets, as they expedite the deactivation of the network of interactions between symptoms of psychopathology (Castro et al., 2019). This study examined whether bridge symptoms exist between PTSS, positive and negative symptoms of psychosis, and mood and anxiety symptoms, which may provide insight into potential mechanisms that give rise to and/or maintain comorbidity and suggest useful targets for interventions to prevent the development of comorbidity (Afzali et al., 2017).

Previous network analyses have used small samples sizes and have not investigated subthreshold symptoms, focusing on individuals with psychotic disorder (Isvoranu et al., 2017) or psychotic disorder and PTSD (Hardy et al., 2021; Isvoranu et al., 2017). This study aimed to investigate which symptoms are most influential in a network of PTSS, mood symptoms, anxiety symptoms, psychotic experiences, and negative symptoms of psychosis within a general population sample. This network analysis of subthreshold and threshold symptoms was undertaken on an exploratory basis with no firm a priori hypothesis specified with regard to bridging symptoms between comorbidities. We did, however, expect that symptoms related to emotional reactivity, affective dysregulation, and markers of generalized distress would feature prominently, as in previous work, possibly suggesting an affective pathway to psychosis (Hardy et al., 2021; Isvoranu et al., 2017).

METHOD

Participants and procedure

This network analysis used data from the Avon Longitudinal Study of Parents and Children (ALSPAC), a large, well-defined birth cohort in the United Kingdom (Fraser, Boyd, et al., 2013; Harris et al., 2009; Northstone et al., 2019). ALSPAC recruited women in the Avon Health Authority area who were expected to give birth (i.e., expected delivery date) between April 1, 1991, and December 31, 1992, resulting in an initial cohort of 14,062 children. Later enrolment expanded this sample to 15,645 individuals (Fraser, Boyd, et al., 2013; Fraser, Macdonald-Wallis, et al., 2013). A total of 4,472 individuals were included in this study, as not all recruited individuals completed the full breadth of measures (Supplementary Figure S1). Ethical approval for the ALSPAC study was obtained from local research ethics committees and the ALSPAC Law and Ethics Committee. Research questions were approved by ALSPAC, and all participants provided written consent

for the collection and use of these data. This network analysis did not use clinical or administrative records and used fully anonymized ALSPAC data.

Measures

PEs

PEs at 24 years of age ($M = 24.03$ years) were assessed using the Psychosis-Like Symptoms Semi-Structured Interview (PLIKSi; Sullivan et al., 2020). The PLIKSi is used to assess the presence of 12 psychotic experiences, including hallucinations, delusions, and thought interference (Zammit et al., 2008). We assessed the presence of any psychotic experience without requiring a threshold for psychotic disorder, allowing us to examine associations with subthreshold symptoms in addition to symptoms of a higher severity that might meet the criteria for a psychotic disorder diagnosis. Psychotic symptoms were coded as present if a respondent endorsed one or more experiences as “suspected” or “definitely present” over the previous 6 months. PLIKSi interviews were carried out by trained psychologists and rated following the Schedules for Clinical Assessment in Neuropsychiatry guidelines (World Health Organization [WHO], 1994). When administered at 24 years of age, the PLIKSi demonstrated good interrater reliability, intraclass correlation coefficient ($ICC = 0.81$, 95% [CI 0.68, 0.89]), and test–retest reliability, $ICC = 0.9$, 95% confidence interval (CI) [0.83, 0.95] (Lewis et al., 1992). In the present sample, Cronbach’s alpha for PEs was .53.

Negative symptoms of psychosis

Negative symptoms at age 24 years ($M = 24.03$ years) were measured using 10 items from the self-report Community Assessment of Psychic Experiences (CAPE; Mark & Toulopoulou, 2016). Responses were scored on a 4-point Likert scale ranging from 0 (*never*) to 3 (*always*). Questions assess behavior such as a lack of sociability (e.g., “Have you felt that you have no interest to be with other people?”) and apathy (e.g., “Have you felt that you are spending all your days doing nothing?”). In the present sample, Cronbach’s alpha was .65.

Depressive and anxiety symptoms

Past-month symptoms of depression and anxiety at age 24 years ($M = 24.03$ years) were measured using 19 items related to well-being, depression, and anxiety from the self-report Clinical Interview Schedule–Revised (CIS-R; Lewis

et al., 1992). The CIS-R assesses a range of symptoms and can be used as a diagnostic tool for depression and anxiety disorders, per criteria outlined in the *International Statistical Classification of Diseases and Related Health Problems* (10th ed.; *ICD-10*; WHO, 2016). In the present sample, Cronbach’s alpha was .59.

PTSD symptoms

Past-month PTSD symptoms at age 23 years ($M = 23.86$), as were assessed using the self-report PTSD Checklist for *DSM-5* (PCL-5; Weathers, Litz, et al., 2013; Bovin et al., 2016). Respondents were asked to rate 20 items on a 5-point Likert scale ranging from 0 (*not at all*) to 4 (*extremely*), with higher scores indicating more severe symptoms. PCL-5 scores were only included for individuals who endorsed a *DSM-5* Criterion A event (APA, 2013). In the present sample, Cronbach’s alpha was .95.

Trauma exposure

At 23 years of age ($M = 23.86$), respondents completed a questionnaire assessing trauma exposure. The instrument included questions on childhood abuse and neglect, selected from the Childhood Trauma Questionnaire (Bernstein & Fink, 1998), as used in the U.K. E-risk child/adolescent cohort (Matthews et al., 2015) and U.K. Biobank (Sudlow et al., 2015); questions on relationship abuse since age 16 years, based on questions used in the British Crime Survey on Domestic Abuse; and questions on lifetime exposure to selected traumatic events included in the Life Events Checklist for *DSM-5* (Gray et al., 2004; Weathers, Blake, et al., 2013).

Data analysis

Before estimating a symptom network, we considered whether certain items shared overlapping content or similar wording, rendering them redundant (e.g., negative symptoms and symptoms of mood disorders; Boks et al., 2007; Fried & Cramer, 2017; Purnine et al., 2000). Including redundant nodes may exaggerate the importance of some symptoms within the overall network (Fried & Cramer, 2017). Two authors (Laurence Astill Wright, Eoin McElroy) independently screened the items for content overlap, and where there was consensus that two or more items contained similar content, only one item was retained. Subsequently, the remaining items were analyzed using the Goldbricker function in the R package *networktools* (Jones, 2017.). The Goldbricker function assesses whether

correlations between a common variable and two separate unique variables are significantly different from one another, using a p value threshold of .05; this is repeated for every combination of correlations within the network. Following this method, we included 45 symptoms in the network (Supplementary Table S1).

Symptoms in the network are represented as nodes, with the connecting lines between the nodes, known as edges, representing the conditional associations between symptoms. Edges within these networks can be interpreted as partial correlations and control for all other variables in the network. Thicker edges represent stronger associations between nodes. The Fruchterman–Reingold graphical algorithm arranges nodes according to how strongly they are associated with one another. Nodes with weaker correlations are placed peripherally in the network (Epskamp et al., 2018). We used the R package *qgraph* (Epskamp et al., 2012) to estimate a network using the PLIKSi, PCL-5, CAPE, and CIS-R. Using *qgraph*, we computed the appropriate correlation matrix to estimate a regularized partial correlation network. We estimated networks using Spearman correlations and the graphical Bayesian information criterion graphical least absolute shrinkage and selection operator (BICglasso; i.e., a method for scoring and selecting a conservative and interpretable model) threshold method to shrink edges and set very small edges to 0, using the tuning parameter of 0 (Epskamp et al., 2018). This avoids problems associated with multiple testing and produces a sparse network that balances fit with explanatory power. We tested for modularity using the *walktrap* algorithm in the exploratory graph analysis (EGA) R package (Golino & Epskamp, 2017). This function detects clusters of densely connected nodes that are nested within the broader network (Figure 1) to estimate the best model.

We used node centrality to estimate each symptom's importance within the network (Opsahl & Skvoretz, 2010). Centrality is a measure of the connectivity of symptoms within a network (i.e., the number and strength of connections a node has with other nodes) and is proposed to denote the importance and clinical relevance of symptoms within a given network. We used strength, indicating the absolute sum of edge weights, as the only measure of node centrality, as it is stable across network analysis studies (Birkeland et al., 2020). Expected influence (EI) indicates the sum of edge weights, thus accounting for negative edges. The sum of the edge weights directly between a node and all other nodes is known as 1-step EI, whereas 2-step EI accounts for the indirect effect that a node may have on other nodes (Epskamp et al., 2018). To determine which nodes were influential in bridging symptom clusters during EGA, we calculated bridge expected influence (BEI), bridge strength, and bridge betweenness. These

metrics focus solely on connections between symptom clusters. Bridge centrality indices were calculated using the R package *networktools* (Jones, 2017.) All centrality and bridge centrality indices were presented as standardized Z scores, with higher scores reflecting higher levels of importance. We assessed the stability of individual networks using the R package *bootnet* (Epskamp et al., 2018). Case-dropping bootstrapping was used to estimate a correlation stability coefficient for centrality metrics, with values above 0.5 implying strong stability, and nonparametric bootstrapping (1,000 iterations) was used for edge weight differences and centrality differences, with 95% confidence intervals calculated around the network edge weights.

We estimated the network using a pairwise present approach to missing data. To assess whether the missing data could affect the estimation of the network, we imputed missing PLIKSi, PCL-5, CAPE, and CIS-R values using the R package *missforest*, which uses a random forest trained on the observed values of the data to predict missing values.

RESULTS

Sample demographic and symptom descriptive characteristics are shown in Table 1 (current sample: $N = 4,472$, 36.7% male; total ALSPAC sample: $N = 15,645$, 49.2% male). Trauma exposure is shown in Supplementary Table S2. Descriptive characteristics of the primary network measures are shown in Supplementary Table S3, and Cronbach's alpha values for internal consistency are given in the Measures section and Supplementary Table S4.

EGA clustering

The EGA produced a three-cluster solution including (a) symptoms of PTSD, (b) psychotic experiences, and (c) anxiety symptoms, depressive symptoms, and negative symptoms of psychosis. These represent three broad interrelated categories of symptoms.

Network structure and centrality analysis

Strength centrality and bridge centrality metrics are presented in Figure 2 and Figure 3. Psychotic experiences were the symptoms with the highest strength centrality, also displaying high bridge centrality. Symptoms of generalized anxiety, panic, tiredness, and risk-taking also displayed high bridge centrality. Among PTSD symptoms, the reexperiencing phenomena of disturbing dreams and

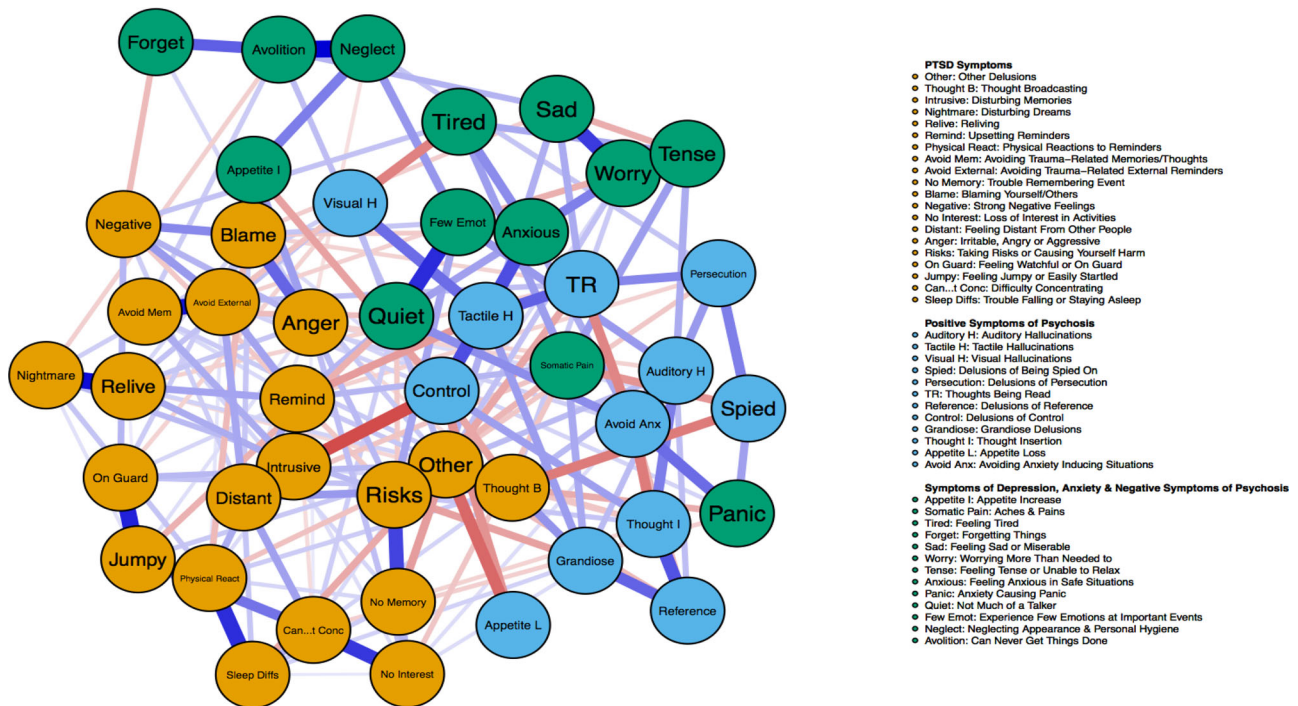


FIGURE 1 Network of psychotic experiences (positive symptoms of psychosis) and negative symptoms of psychosis, posttraumatic stress, anxiety, and depression

Note: Blue lines represent positive associations, with the thickness of the line indicating association strength. The color of the node represents to which of the three clusters the symptom belongs, as estimated using exploratory graph analysis.

reliving the traumatic event did not have high bridge centrality, but disturbing memories, blame, anger, feeling distant, difficulty concentrating, and sleep disturbances were associated with higher bridging metrics.

Network stability

The confidence intervals for edge weights and, thus, the stability of the network and sampling variability of our findings are presented in Supplementary Figure S2 (Epskamp et al., 2018; Fried et al., 2018). The confidence intervals were moderately narrow, thus indicating a fair precision of the network estimation. Bootstrapping procedures supported the robustness of centrality measures, graph structure, and edge weights (Supplementary Figures S2–S4). The correlation stability coefficient for the strength centrality metric was .283, which is above the cutoff score of .25 but below the recommended score of .50 as a reliable indicator of stability (Epskamp et al., 2018), which was not ideal. The Goldbricker function did not highlight any overlapping symptoms. The pairwise present approach to missing data (Figure 1) and the imputation of missing data produced very similar networks (Supplementary Figure 5).

DISCUSSION

These analyses demonstrate the central role of psychotic experiences and generalized symptoms of anxiety (e.g., panic, avoidance) in the association between symptoms of PTSD, negative symptoms of psychosis, depressive symptoms, and other symptoms of anxiety. Our results are consistent with the thesis that generalized anxiety plays a central role, via stress reactivity mechanisms, in the development and maintenance of psychosis and PTSD symptoms. PEs were central to the network—more so than negative symptoms of psychosis—and expressed high strength centrality, possibly highlighting the particularly debilitating nature of positive symptoms of psychosis (Harvey & Strassnig, 2012). The analyses demonstrated three broad interrelated categories: PTSD symptoms; psychotic experiences; and negative symptoms of psychosis, depressive symptoms, and anxiety symptoms.

The identification of anxiety symptoms as a possible affective pathway to psychotic outcomes (Myin-Germeys & van Os, 2007) echoes the findings of other network analyses in the field (Hardy et al., 2021; Isvoranu et al., 2017). The results highlight the possible role of heightened emotional distress (e.g., anxiety, hyperarousal) as a bridge between PEs and PTSS, similar to Isvoranu et al.'s

TABLE 1 Sample demographic and symptom characteristics

Variable	Current sample (<i>n</i> = 4,472)		Full ALSPAC sample (<i>n</i> = 15,645)	
	<i>n</i>	%	<i>n</i>	%
Race/ethnicity				
White	3,903	97.9	12,064	96.0
Non-White	83	1.8	481	3.8
Missing	486	10.9	3,100	19.8
Employment status				
Unemployed	316	7.1	316	2.0
Employed/in education/training scheme	3,569	79.8	3,569	22.8
Missing	587	13.1	11,760	75.2
Living arrangements				
Living on own	225	5.0	225	1.4
Living with partner	1,202	26.9	1,202	7.7
Living with others in shared accommodation	840	18.8	840	5.4
Living with parents	1,543	34.5	1,543	9.9
Other	76	1.7	76	0.5
Missing	586	13.1	11,759	75.2
Outcome completion				
PLIKSi completers	3,874	86.6	3,874	24.8
CAPE completers	3,892	87.0	3,892	24.9
CIS-R completers	3,968	88.7	3,968	25.4
PCL-5 completers	1,690	37.8	1,690	10.8
Mental health symptoms				
Current PTSD at age 24 years	273	16.2	273	16.2
Current definite psychotic experience at age 24 years	136	3.5	136	3.5
GAD at age 23 years	327	13.0	327	13.0

Note: ALSPAC = Avon Longitudinal Study of Parents and Children; PLIKSi = Psychosis-Like Symptoms Interview; CAPE = Community Assessment of Psychic Experiences; CIS-R = Clinical Interview Schedule-Revised; PTSD = posttraumatic stress disorder; PCL-5 = PTSD Checklist for DSM-5; GAD = generalized anxiety disorder.

(2017) observation that anxiety acts as the key link between emotional abuse and PEs. This is consistent with the stress vulnerability model of psychotic outcomes, which states that symptoms emerge when an individual's ability to cope is exceeded by stressors, such as traumatic events (Myin-Germeys & van Os, 2007). Dysregulated emotional reactivity to daily stressors may create vulnerability to psychotic outcomes through both psychological (i.e., cognitive and perceptual biases; Hardy, 2017) and biological mechanisms (i.e., hypothalamic-pituitary-adrenal axis dysregulation and the disruption of the glutamatergic and dopaminergic systems; Fletcher & Frith, 2009; Howes & Kapur, 2009). Researchers have suggested that this stress reactivity pathway predominantly underlies positive rather than negative symptoms of psychosis (van Winkel et al., 2008).

We were not able to explore causal relationships in the analyses, and it is possible that both psychotic experiences and symptoms of PTSD led to anxiety in the

sample. However, if generalized anxiety symptoms and stress reactivity underlie the PTSD-psychosis symptom overlap, as described previously, targeting these specific factors and emotional distress more broadly could ameliorate symptoms of both disorders. The authors of previous network analyses have proposed that broad psychological intervention, such as creating a sense of control, safety, and self-worth could reduce the widespread influence of trauma-related beliefs on symptoms (Hardy et al., 2021). Trauma-focused interventions are effective and safe in treating PTSS in individuals with psychotic disorder (de Bont et al., 2013; van den Berg et al., 2015) and may have some benefits with regard to PEs among individuals without PTSD (Brand et al., 2018). Treating PTSS, if present, may represent an opportunity to decrease anxiety symptoms, stress reactivity, and emotional distress more broadly in individuals with PEs (van den Berg et al., 2015).

The associations between positive and negative symptoms of psychosis, anxiety symptoms, and symptoms of

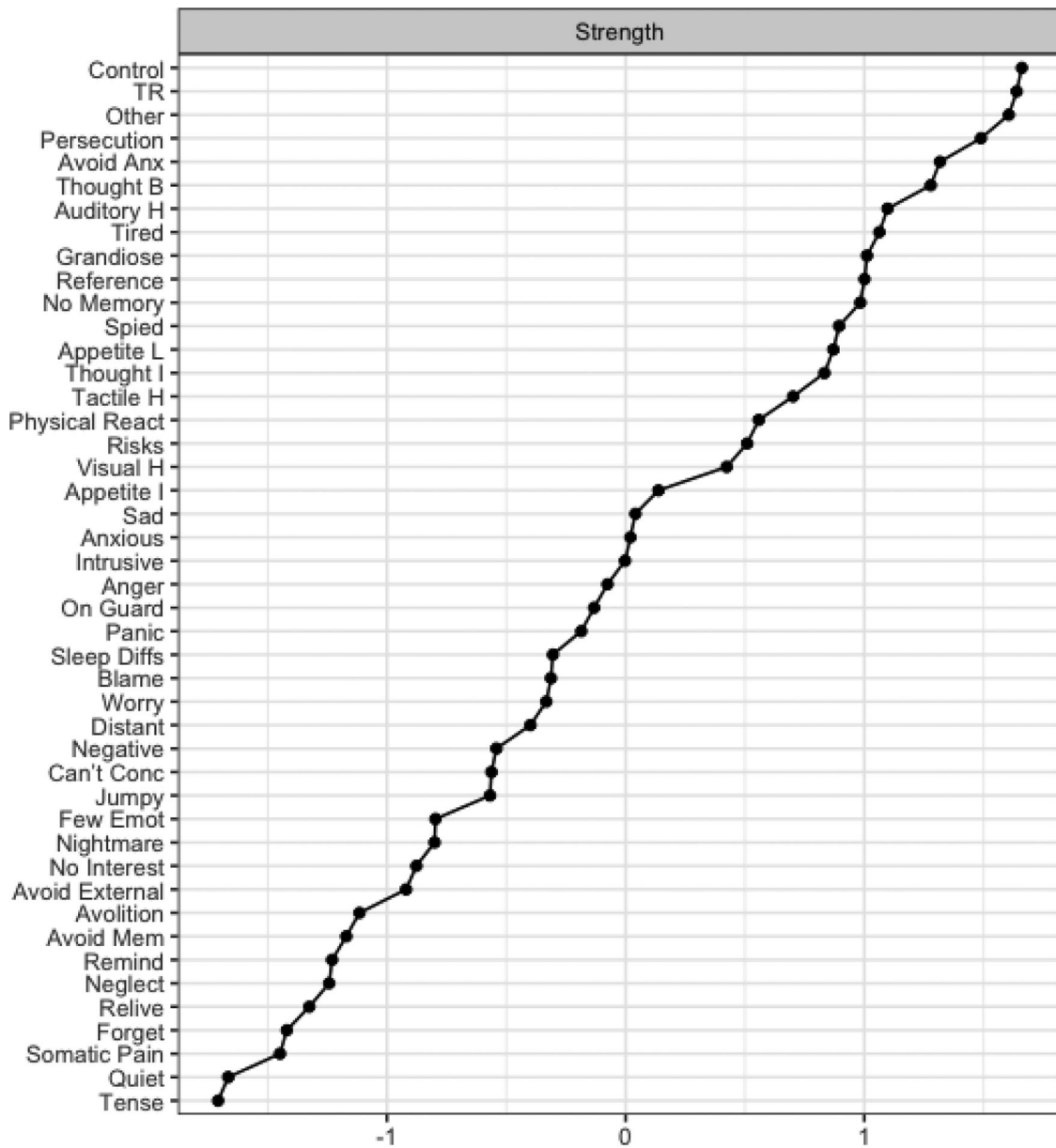


FIGURE 2 Centrality analysis using the strength of each node

PTSD in the network could also be due to confounding by factors like substance misuse or dissociation such that neither positive symptoms of psychosis, negative symptoms of psychosis, anxiety symptoms, nor symptoms of PTSD have a causal effect on the other symptoms. Furthermore, the PLIKSi, PCL5, and CIS-R may overlap in the measurement of anxiety, thus inflating its importance in the network. This, however, is unlikely, as the Goldbricker analysis suggested minimal symptom overlap between nodes following a qualitative assessment of node overlap and item removal before network estimation.

The network identified feeling distant, blame, risk-taking, and anger as bridging symptoms, similar to what was observed by Hardy et al. (2021), who identified hyper-

arousal and trauma-related beliefs, such as blame and negative perceptions of the world and the self, as key bridges within their network. These posttraumatic cognitions may exacerbate interpersonal difficulties, cognitive biases, and sensory-perceptual experiences and contribute to the development and maintenance of psychotic outcomes and PTSD (Freeman & Garety, 2014). Furthermore, the prominence of risk-taking as a bridging symptom may highlight the role of emotional dysregulation and impulse control, factors also identified as connecting symptoms between childhood trauma and psychosis in previous networks (Isvoranu et al., 2017).

Risk-taking and poor impulse control (Reddy et al., 2014) are common in individuals with both psychosis

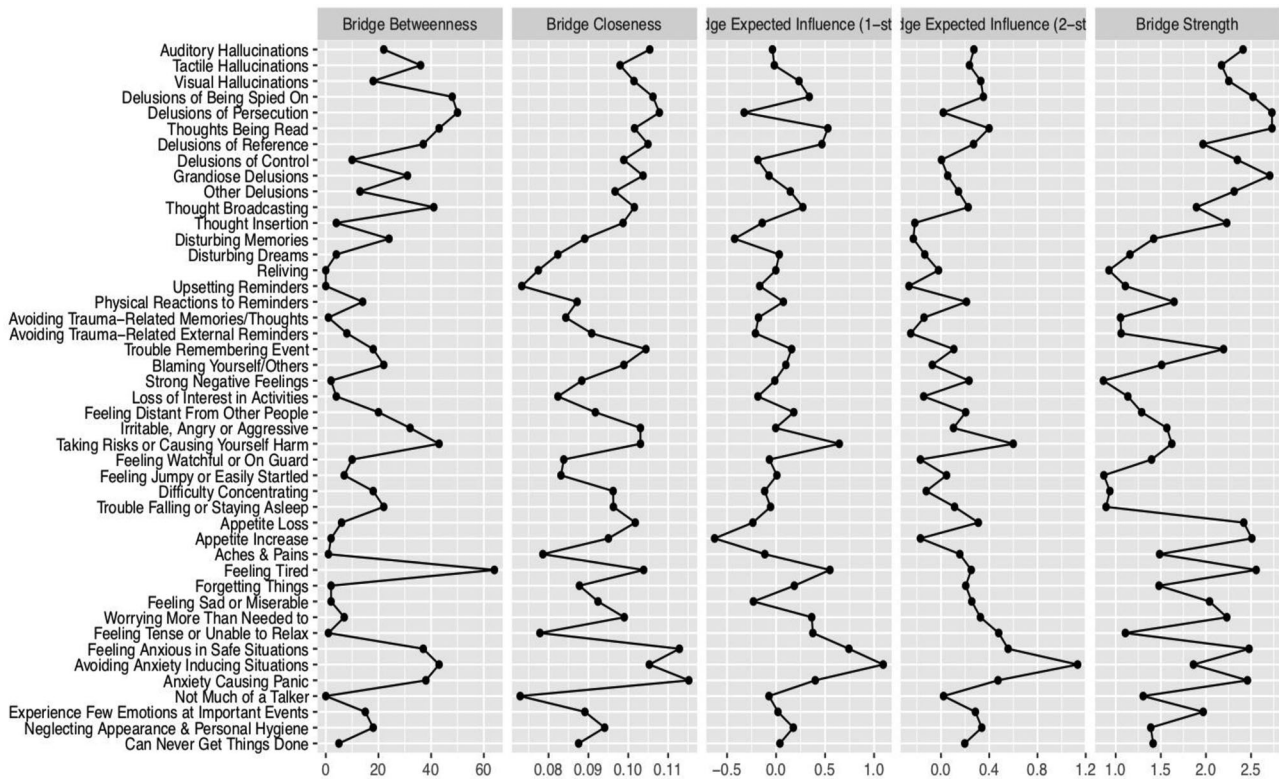


FIGURE 3 Bridge centrality strength network statistics

and symptoms of PTSD (Martin et al., 2016). Possible mechanisms underlying risk-taking in psychosis include cognitive deficits and reward learning dysfunction (Reddy et al., 2014), whereas risk-taking in PTSD has been associated with emotional dysregulation (Martin et al., 2016). Risk-taking and impulsivity may also further increase the risk of traumatic event exposure, and this may negatively impact stress reactivity in psychosis or represent an entirely separate pathway to psychotic outcomes.

The present results challenge the key role of reexperiencing symptoms as an explanation for the association between psychological trauma and psychosis (Strelchuk et al., 2022). We identified a comparatively minor role of intrusive memories as a bridging symptom relative to anxiety symptoms, and this is consistent with interventional evidence suggesting that reducing reexperiencing phenomena via trauma-focused therapies does not reduce the severity of auditory hallucinations (van den Berg et al., 2015, 2018). These results also replicate previous network analyses (Hardy et al., 2021). The minor bridging role of intrusive memories within the network may represent another minor connecting pathway between psychosis and PTSD. Despite the suggested role of reexperiencing and dissociative detachment in the development of auditory hallucinations (Berry et al., 2017), we were unable to directly assess the role of dissociation; however, pre-

vious work has not highlighted emotional numbing as a key symptom within networks (Hardy et al., 2021). Indeed, it is likely that there are multiple affective pathways in the highly complex association between PTSD and psychosis (Luhmann et al., 2019), and there may be pathways through symptoms that were not assessed in this study (e.g., disturbances in self-organization in complex PTSD; Karatzias et al., 2019).

To our knowledge, this study was the first network analysis to assess symptoms of psychosis, posttraumatic stress, anxiety, and depression. We used a large, well-characterized sample (Fraser et al., 2013) to assess subthreshold symptoms transdiagnostically using well-validated, psychologist-administered and self-report measures. The PLIKSi, CIS-R, and CAPE were administered 2–3 months apart, on average. Comparatively few individuals completed the PCL-5 at age 23 ($n = 1,690$) relative to completion of PLIKSi ($n = 3,874$), CAPE ($n = 3,892$), and CIS-R ($n = 3,968$), but the random-forest imputation of missing data produced a similar network to the pairwise present approach to missing data (Supplementary Figure S5; Hong & Lynn, 2020; Stekhoven & Bühlmann, 2011). The PCL-5, a self-report measure that is only able to provide a provisional PTSD diagnosis, does not distinguish between childhood trauma and more recent traumatic experiences; thus, this lack of longitudinal investigation limits our

ability to comment on whether bridging symptoms are likely to play a causal role in the development of new symptoms. Fundamentally, the cross-sectional nature of the sample precludes us from making temporal inferences. Furthermore, a minority of individuals reported trauma exposure (Supplementary Table S2), so the lack of associations among reexperiencing symptoms in the network may be due to a lack of reexperiencing symptoms more broadly in the sample, and many of the symptoms measured by the PCL-5 in these analyses might be indicative of generalized distress rather than related specifically to PTSD.

Although the ALSPAC cohort is probably the largest study available worldwide with such detailed information collected, it is still relatively small for examining uncommon outcomes, such as PEs. This inevitably caused imprecision in the present analyses. Furthermore, the majority of the sample was female, and although women generally experience higher rates of certain psychological trauma and PTSS than men (McGrath, McLaughlin, et al., 2017; Olf et al., 2019), sample attrition may have caused selection bias and led to errors in network estimation. Furthermore, although we assumed the network pattern was the same in men and women, there could be sex-related psychosis symptom profiles that may have created a different array of symptom presentations had the sample been gender-balanced (Read, 2013). The sample was also nonclinical, and different associations may have emerged in a clinical sample. In addition, although we assessed trauma exposure (Supplementary Table S2), some participants may have been rating symptoms that were not related to the DSM-5 Criterion A event they endorsed. Some measures, such as the measure of psychotic experiences, demonstrated low internal consistency (Supplementary Table S4). Future research could compare sex-related differences and explore the role of dissociative symptoms and emotional dysregulation.

In support of the stress reactivity and affective pathways to psychosis, the results of this study suggest that symptoms of anxiety and emotional distress have a key role in the development and maintenance of psychotic experiences and PTSS (Brand et al., 2018). In addition, the findings indicate that posttraumatic cognitions, emotional dysregulation, and interpersonal problems, rather than reexperiencing phenomena, may also play a key role in traumatic stress and psychosis comorbidity. Targeting symptoms of anxiety, psychological distress, and PTSD-related emotional and behavioral processes may ameliorate symptom burden transdiagnostically.

OPEN PRACTICES STATEMENT

The fully searchable data dictionary of ALSPAC is available at www.bristol.ac.uk/alspac/researchers/our-data/. The data that support the findings of this study are available on request from the corresponding author. The

data are not publicly available due to privacy or ethical restrictions.

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SUPPORTING INFORMATION

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