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Obsessive-Compulsive Symptoms and Other Symptoms of the At-risk Mental State for Psychosis: A Network Perspective

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Background: **The high prevalence of obsessive-compulsive symptoms (OCS) among subjects at Ultra-High Risk (UHR) for psychosis is well documented. However, the network structure spanning the relations between OCS and symptoms of the at risk mental state for psychosis as assessed with the Comprehensive Assessment of At Risk Mental States (CAARMS) has not yet been investigated. This article aimed to use a network approach to investigate the associations between OCS and CAARMS symptoms in a large sample of individuals with different levels of risk for psychosis.** ***Method:*** **Three hundred and forty-one UHR and 66 healthy participants were included, who participated**

in the EU-GEI study. Data analysis consisted of constructing a network of CAARMS symptoms, investigating central items in the network, and identifying the shortest pathways between OCS and positive symptoms. *Results:* Strong associations between OCS and anxiety, social isolation and blunted affect were identified. Depression was the most central symptom in terms of the number of connections, and anxiety was a key item in bridging OCS to other symptoms. Shortest paths between OCS and positive symptoms revealed that unusual thought content and perceptual abnormalities were connected mainly via anxiety, while disorganized speech was connected via blunted affect and cognitive change. *Conclusions:* Findings provide valuable insight into the central role of depression and the potential connective component of anxiety between OCS and other symptoms of the network. Interventions specifically aimed to reduce affective symptoms might be crucial for the development and prospective course of symptom co-occurrence.

Key words: network analysis/clinical high risk/psychosis/obsessive-compulsive/anxiety/depression/ultra-high risk

Introduction

Obsessive-compulsive symptoms (OCS) have a prevalence rate of 30.7% and comorbid obsessive-compulsive disorders (OCD) of 12.3% in patients with a psychotic disorder.[1] Accordingly, a review of the literature reported increased prevalence rates of OCS (13.7%) and OCD (5.5%) in subjects at Ultra-High Risk (UHR) for psychosis[2,3] compared to the general population. Recent studies reported even higher rates in individuals meeting UHR criteria.[4] Subjects at UHR are defined by either experiencing brief intermittent psychotic symptoms (BLIPS), or sub-threshold attenuated psychotic symptoms (APS), or having a genetic risk for psychosis in combination with a significant decline in functioning.[5] Of those individuals meeting UHR criteria, 22% develop a psychotic disorder within 3 years after presentation to clinical services.[6] The high OCS prevalence rates in UHR subjects have resulted in increased research interest in investigating the effects of OCS on clinical outcomes, such as psychosocial functioning and transition rates.[2,4,7] The results have been inconclusive. Some authors have found higher impairment of functioning and positive symptoms in UHR subjects with co-occurring OCS compared to those without[7] whereas others have reported no associations or even better functioning in the group with OCS.[2,4]

Up-to-date, the association between OCS/OCD and subthreshold symptoms of psychosis has solely been reported as co-occurring prevalence rates or associations on the syndrome level in the general population[8] in healthy relatives of patients[9,10] and in UHR subjects.[2,7,11] The investigation of the effects of OCS/OCD in UHR subjects assumes a clear distinction between these constructs. However, phenomenologically distinguishing OCS from APSs, eg, intrusive thoughts from overvalued ideas is challenging.[12] In accordance, the Comprehensive Assessment of At-Risk Mental State (CAARMS) for psychosis, the standard instrument for assessing prodromal psychopathology and identification of the UHR status, assesses OCS as part of psychopathology dimensions, thought to be associated with an imminent psychotic disorder.[13] Hence, exploring associations between OCS and symptoms of the at-risk mental state for psychosis may uncover novel perspectives on the patterns and interrelations between them.

The network approach to psychopathology[14,15] allows for the investigation of such direct relations and interactions on the symptom level, without a priori assumptions regarding the adequacy of the diagnostic categories constructed out of these symptoms. Classical psychometric practices are typically based on latent variable theory, where psychopathological symptoms are caused by a hypothetical underlying mental disorder (eg, OCD)—implying that symptoms cannot be directly related to each other if the disorder is absent (ie, local independence).[14,15] Within a network model, however, symptoms are viewed as active causal agents that can influence one another. Hence, a disorder can be seen as emerging from a system of interacting components. As a result, within a network structure, we can investigate associations between individual symptoms, as well as potential bridging elements (eg, symptoms or other factors) connecting clusters of (other) interacting symptoms.[15]

To this end, the purpose of the current study was to explore the interplay between OCS and other symptoms of the CAARMS using a network model, in a sample with different levels of risk for psychosis, by including individuals meeting UHR criteria as well as healthy controls. Including healthy control subjects helped to broaden the focus to subclinical symptoms in a sample of non-help seeking individuals and also increased power of the conducted analyses. We aimed to identify aspects of symptom co-occurrence, such as symptoms that may play an important role in facilitating the co-occurrence of OCS and psychotic symptoms.

Method

Participants and Procedure

Data were collected in the European network of national schizophrenia networks studying Gene-Environment Interactions (EU-GEI study), which was conducted between May 2010 and May 2015. The EU-GEI study was designed to identify the interactive genetic, clinical, and environmental determinants involved in the development, severity, and outcome of psychosis. Participants were part of the High Risk Study of the EU-GEI cohort. A sample of UHR individuals were recruited from 11

health care institutions (Amsterdam, Barcelona, Basel, Cologne, Copenhagen, London, Melbourne, Paris, Sao Paulo, The Hague, and Vienna). Controls were recruited from the same geographical catchment area as the UHR group, but only from 4 centers.[16] Inclusion and exclusion criteria for UHR and control participants are detailed in Appendix 1 in the supplementary material. The protocol of the EU-GEI study was approved by the Medical Ethics Committees of all participating study sites. In the current study, we included UHR individuals and healthy subjects for whom CAARMS measures were available.

Materials

All participants completed a detailed sociodemographic questionnaire which included questions on their age, gender, and ethnicity. Furthermore, participants completed the CAARMS, a 28-item semi-structured interview developed to assess UHR criteria that is intensity, frequency, duration, and recency of subthreshold psychotic symptoms, as well as other dimensions of psychopathology thought to be indicative of or associated with an imminent psychotic disorder.[17,18] The CAARMS can be clustered into seven domains: positive symptoms, cognitive symptoms, emotional disturbance, negative symptoms, behavioral change, motor changes, and general psychopathology. As part of general psychopathology, one CAARMS item assesses symptoms of OCD by asking participants whether they repeatedly experience upsetting thoughts that they cannot stop, whether they repeat actions they feel they should do, whether they have to do things in a specific way to prevent feeling extremely anxious, whether they need to check things repeatedly like light switches/gas/electrical appliances, or doors or whether they are doing something to prevent unpleasant things from happening (rituals/superstitions). The intensity of reported OCS is measured on a 7-point Likert scale from 0: never present to 6: extreme, with a higher score reflecting higher symptom severity. In addition, the frequency of symptoms is assessed also on a 7-point Likert scale from 0: absent to 6: continuously present and a question assessing whether OCS were manifested by substance abuse. In the current study, CAARMS intensity scores were included.

Data Analysis

In the current analysis, we estimated a network model of OCS and ARMS symptoms, using the *R* statistical software version 3.6.3.,[19] the R package *bootnet* version 1.3.,[20] and we visualized the resulting network structure using the R package *qgraph* 1.6.5.[21] Due to the relatively low sample size in relation to a large number of nodes, and in an attempt to ensure more stable network estimates, we chose to reduce the number of nodes, prior to running the analyses. We did so by excluding low variability items (ie, items with more than 75% response of 0: never), which would have had the potential to bias results (eg, due to severe violations of the normality assumption).[22] Low variability items were selected based on the full sample, but these were identical when investigated in the UHR only sample. Each remaining CAARMS item was represented as a node in the network. The regularized partial correlation between two nodes, after controlling for all other nodes in the network, was represented as an edge.

First, we constructed Pairwise Markov Random Fields (PMRFs) to estimate an undirected network structure (ie, a Gaussian Graphical Model),[23] which allows us to identify the conditional associations between variables.[24] To prevent spurious connections and to estimate a sparse model, we adopted the least absolute shrinkage and selection operator (LASSO) statistical regularisation technique. We chose the graphical LASSO, which uses the Extended Bayesian Information Criterion (EBIC) model selection with a tuning hyperparameter set to 0.5, as commonly employed in previous research.[25–27] To account for the non-normal distribution of some items and missing data, we based our analyses on Spearman correlations using pairwise complete observations.[28]

Second, we assessed the importance of each node in the resulting network by computing centrality indices of the network structure, using the R package *qgraph.*[21] Specifically, we computed strength centrality for each node in the network (ie, the sum of the weights of the connections, in absolute value), as well as the node-specific predictive betweenness (ie, how often a node lies on the pathways between two other nodes, of which one is always the OCS node).[29–31] We chose to investigate only these two centrality measures because (1) previous research has reported that strength centrality is the most stable and interpretable centrality measure,[28] and (2) our interest was in bridging nodes that may play a role in linking OCS to other symptom clusters, and may thus pave the way to co-occurrence.

Third, we used the *pathways* function from the R package qgraph,[21] as used in previous research,[26] to examine the shortest pathways between the OCS node and each of the positive psychotic symptoms. This examination allows us to identify the shortest path from point A to point B for specific symptoms of interest (eg, from OCS to each positive symptom) and is computed using Dijkstra's algorithm.[32]

Finally, we used the R package *bootnet* 1.3. to conduct robustness analyses to ensure stability and accuracy of results.[20,28] Appendix 2 in supplementary material details on these analyses.

Results

Sample Characteristics

Of the 344 UHR subjects and 67 healthy controls originally included in the EU-GEI study, 341 UHR and 66

healthy participants provided comprehensive data on the CAARMS and were included in the current study. Slightly more than half of the sample (52.7%) were male, and the mean age was 22.5 years (SD = 4.79). Twenty-nine (8.2%) participants fulfilled the diagnostic criteria for an obsessive-compulsive disorder (OCD), 53 (21.0%) reported moderate to severe obsessions and 54 (14.4%) moderate to severe compulsions, respectively. Table 1 shows sociodemographic and clinical characteristics for the overall sample and the UHR and healthy controls subgroup separately. In addition, supplementary table S1 presents the means and standard deviations of individual CAARMS items.

General Network Structure

We identified and excluded the following seven symptoms with low variability (ie, items with more than 75% response of 0: never): GP3 (mania), E3 (observed inappropriate affect), B4 (disorganizing, odd, stigmatizing behavior), M1 (subjective motor change), M2 (observed motor change), M3 (impaired bodily sensation), and M4 (impaired autonomic functioning) from the network structure. Figure 1 presents the network constellation of the 21 remaining symptoms from the CAARMS. All symptoms were connected, either directly or indirectly, with other symptoms. Overall, there were stronger connections with items within the same domain than across domains, as defined in table 2. While robustness analyses indicate some caution is needed when interpreting the strength and presence of weaker edges, our results here focus on highlighting the stronger and thus likely robust edges in the network structure. For instance, strong connections were found between GP1 and GP2 (depression and suicidality), P1 and P2 (unusual thought and nonbizarre ideas), N1 and N2 (anhedonia and avolition), and B2 and B3 (social isolation and impaired role function). Across domains, a strong connection was found between GP1 and N1 (depression and anhedonia). Interestingly, GP6 (OCS) was indirectly connected to other CAARMS items mainly via GP5 (anxiety), B2 (social isolation), and E2 (observed blunted affect).

Strength and Node-specific Predictive Betweenness Measures

Figure 2 (left) illustrates the magnitude of the node-strength for each symptom. In the entire network, GP1 (depression) was the most central symptom, significantly stronger than most other nodes in the network. GP6 (OCS) was the least central symptom, indicating that, as compared to other CAARMS symptoms, GP6 (OCS) was not a well-connected node in the network. Figure 3 illustrates the *node-specific predictive betweenness* values for each node in the network—ie, how often a node lies on the pathways between two other nodes, of which one is always the OCS. The white dots represent the node-specific predictive betweenness in the current sample, while the black lines represent the variability of the measure across 1000 nonparametric bootstrap iterations. Item GP5 (anxiety) had the highest node-specific predictive betweenness score, followed by item E2 (observed blunted affect). This

Table 1. Sociodemographic and Clinical Characteristics for the Overall Sample, as well as Subjects at Ultra-High Risk (UHR) and Healthy Controls (HC) Apart

	Overall Sample N = 407	UHR N = 341	HC N = 66	Comparison between UHR and HC
Age	22.52 (4.79)	22.42 (4.91)	22.91 (4.09)	T = −0.760, P = .447
Gender (male %)	53.3	53.4	53.0	X = 0.003, P = .959
Ethnicity (Caucasian %)	69.7	71.2	62.1	X = 15.856, P = .007
Years of education	14.68 (3.08)	14.37 (3.07)	16.15 (2.72)	T = −4.346, P < .001
Current employment (yes %)	65.6	60.5	90.7	X = 36.8091, P < .001
Current living (alone %)	13.3	14.8	6.1	X = 25.171, P < .001
DSM comorbid diagnosis (yes %)				
Depression	27.8	33.0	0	X = 28.815, P < .001
OCD	8.2	9.7	0	X = 5.582, P = .018
Anxiety disorder	38.8	45.1	6.3	X = 34.085, P < .001
CAARMS				
Positive	8.63 (5.01)	10.09 (3.96)	1.12 (2.54)	T = 17.692, P < .001
Negative	6.02 (3.87)	6.94 (3.41)	1.35 (2.47)	T = 15.685, P < .001
Cognitive	2.70 (1.83)	3.14 (1.65)	0.48 (.91)	T = 17.920, P < .001
Emotional	2.71 (2.45)	3.16 (2.40)	0.39 (.95)	T = 15.616, P < .001
Social	6.92 (4.51)	8.02 (3.96)	1.39 (2.70)	T = 16.580, P < .001
Motor	1.90 (2.46)	2.24 (2.55)	0.25 (.89)	T = 10.671, P < .001
General	12.83 (7.59)	14.96 (6.33)	2.52 (3.71)	T = 21.556, P < .001
Medication use				
Antidepressant (yes %)		29.8		
Antipsychotics (yes %)		9.6		
Anxiolytics (yes %)		9.2		

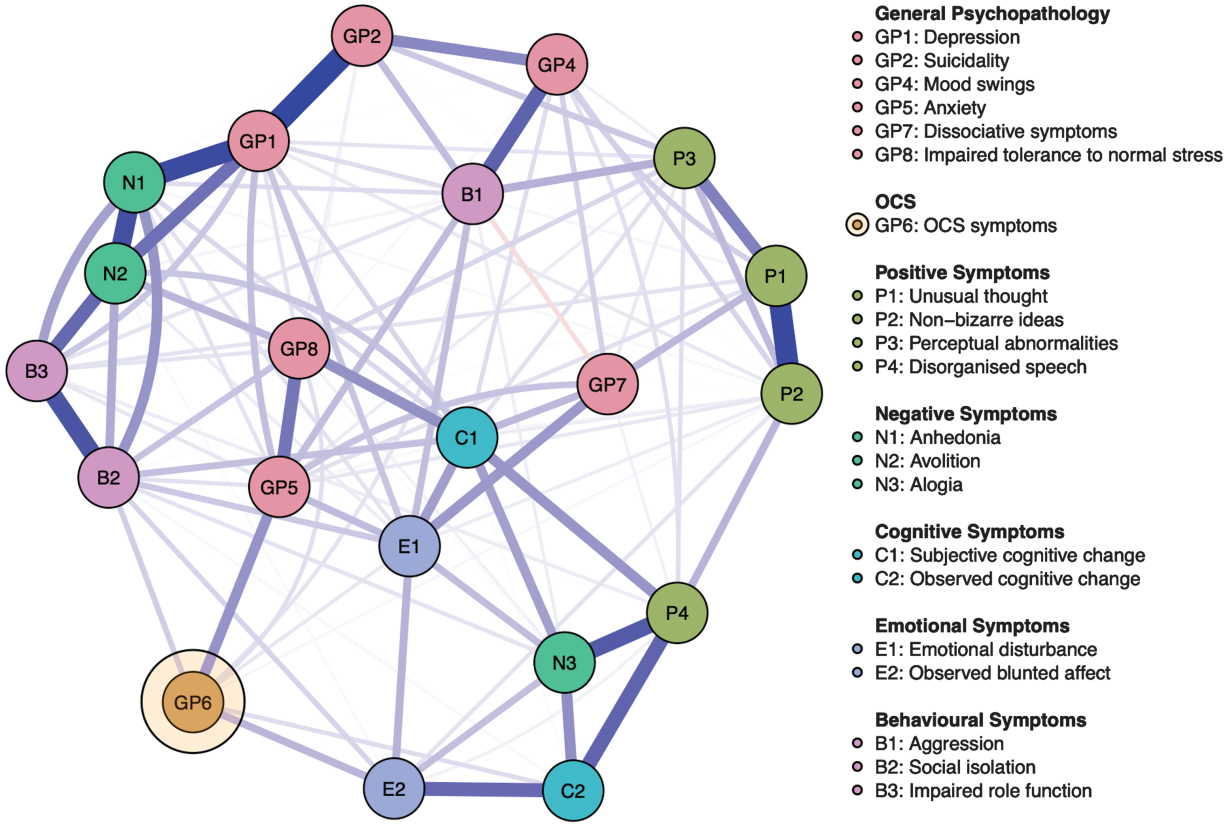

Fig. 1. Network structure of 21 CAARMS symptoms. Item groups are differentiated by color. The color of the edge indicates the size of the association (blue for positive associations; red for negative associations). For a color version, see this figure online.

finding suggests that anxiety may be the main connector between OCS and other ARMS symptoms.

Robustness Analysis

Robustness analyses (see supplementary appendix 2) revealed some relatively wide bootstrapped confidence intervals (CIs). We, therefore, recommend caution when interpreting the network structure, especially when interpreting the strength and presence of weaker edges. Nonetheless, the bootstrap mean was generally close to the sample mean, indicating interpretable results (see supplementary figure S1).

The strength centrality stability plot is shown in figure 2 (right), while figure 3 includes the variability of the node-specific predictive betweenness across 1000 nonparametric bootstrap iterations (ie, black lines). The CS coefficient obtained for strength was CS = 0.59. This is above the preferred 0.5 cut-off, indicating stable results. For significance difference testing, the node with the highest strength centrality, GP1 (depression), showed significantly higher node strength than most of the other nodes (refer to supplementary figure S3 for detail). Of note, correcting for multiple testing when carrying out significance difference testing is not feasible, as elaborately explained in the cited paper.[28] While the functionality for calculating the CS coefficient and running significance difference testing for node-specific predictive betweenness is not yet implemented in the *bootnet R* package,[20] the bootstrap results show node GP5 (anxiety) was consistently identified as the most central bridging node, followed by node E2 (observed blunted affect). Furthermore, in the additional case-drop bootstrap analysis carried out (see supplementary figure S4), node GP5 remains visibly the main and most stable bridging node in the network. Markedly, in general and aligned with previous research,[28] the node-specific predictive betweenness is unstable and shows high variability; despite this, node GP5 (anxiety) stands out as a stable bridging node both in the nonparametric and case-drop bootstraps.

Shortest Pathways: OCS to Positive Symptoms

Overall, all positive symptoms of psychosis (P1, P2, P3, and P4) were indirectly connected to GP6 (OCS) via other symptoms (figure 4, left). Results of shortest pathways analyses showed that the main connective pathways were through node GP5 (anxiety) and through nodes E2 (observed blunted affect) and C2 (observed cognitive change), directly linked to P4 (disorganized speech). Our robustness analysis (figure 4, right) showed that these were the most stable pathways. The pathways from GP5 (anxiety) to the remaining positive symptoms P1, P2, and P3 were less stable and should thus be interpreted with caution, with multiple potential connective components, most often

Table 2. Node Labels and Corresponding Symptom Names of the 28-CAARMS Symptoms

Item Label	Item Description	Domains
P1	Unusual thought	Positive
P2	Non-bizarre ideas	Positive
P3	Perceptual abnormalities	Positive
P4	Disorganized speech	Positive
N1	Anhedonia	Negative
N2	Avolition	Negative
N3	Alogia	Negative
C1	Subjective cognitive change	Cognitive
C2	Observed cognitive change	Cognitive
E1	Emotional disturbance	Emotional
E2	Observed blunted affect	Emotional
E3*	Observed inappropriate affect	Emotional
B1	Aggression	Behavioral
B2	Social isolation	Behavioral
B3	Impaired role function	Behavioral
B4*	Disorganizing, odd, stigmatizing behavior	Behavioral
M1*	Subjective motor change	Motor
M2*	Observed motor change	Motor
M3*	Impaired bodily sensation	Motor
M4*	Impaired autonomic functioning	Motor
GP1	Depression	General Psychopathology
GP2	Suicidality	General Psychopathology
GP3*	Mania	General Psychopathology
GP4	Mood swings	General Psychopathology
GP5	Anxiety	General Psychopathology
GP6	OCD symptoms	General Psychopathology
GP7	Dissociative symptoms	General Psychopathology
GP8	Impaired tolerance to normal stress	General Psychopathology

Note: *Seven symptoms were excluded from the network analysis due to low variability.

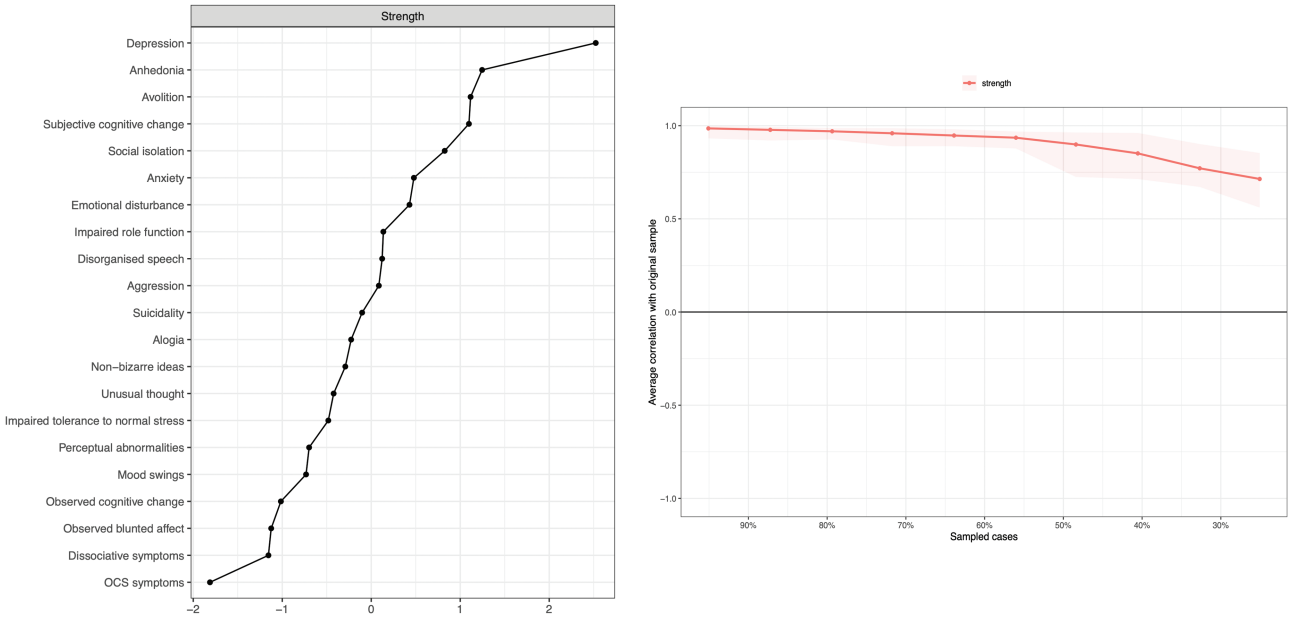

Fig. 2. (Left) Node-strength centrality for each CAARMS symptom, and (right) stability of centrality indices. Centrality measures are shown as standardized *z*-scores. The right panel indicates the average correlation with the original sample after reducing the sample size through case-dropping bootstrapping. For a color version, see this figure online.

including GP8 (impaired tolerance to normal stress) and B1 (aggression) to P3 (perceptual abnormalities), and direct pathways from GP5 (anxiety) to P1 (unusual thought). Other potential pathways could also be identified, but since these were very small and, in most cases, present in less than 20% of the bootstraps, we do not highlight these.

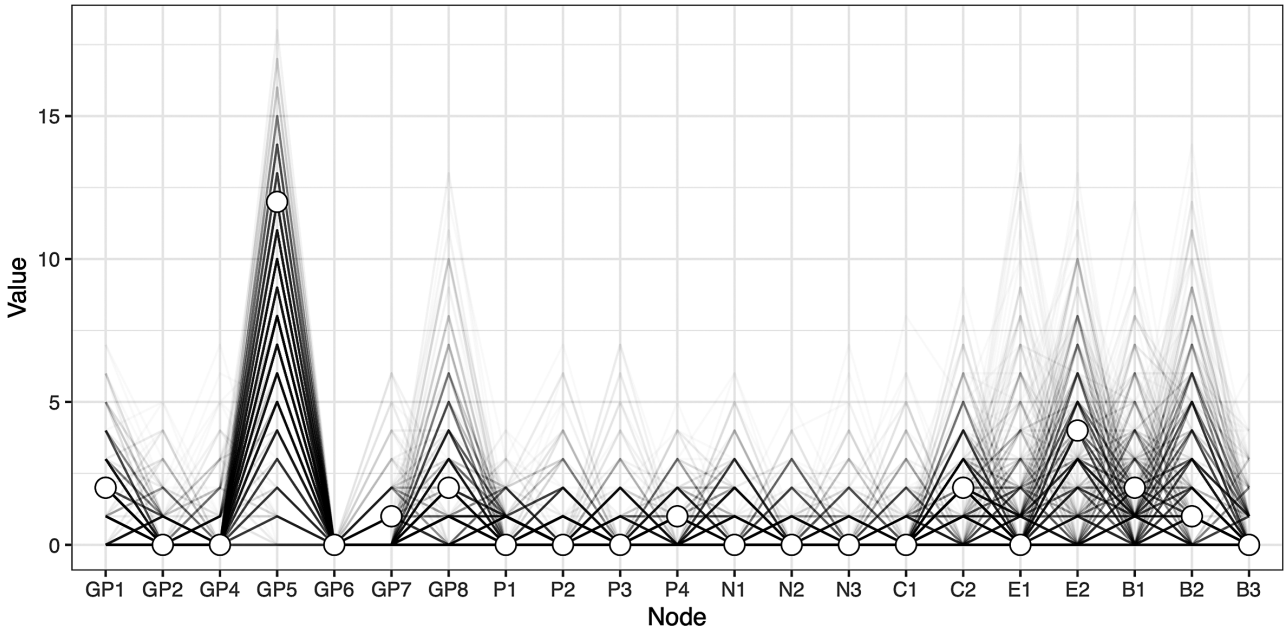

Fig. 3. Node-specific predictive betweenness. The white dots represent the node-specific predictive betweenness in the current sample, while the black lines represent the variability of node-specific betweenness across 1,000 nonparametric bootstrap iterations.

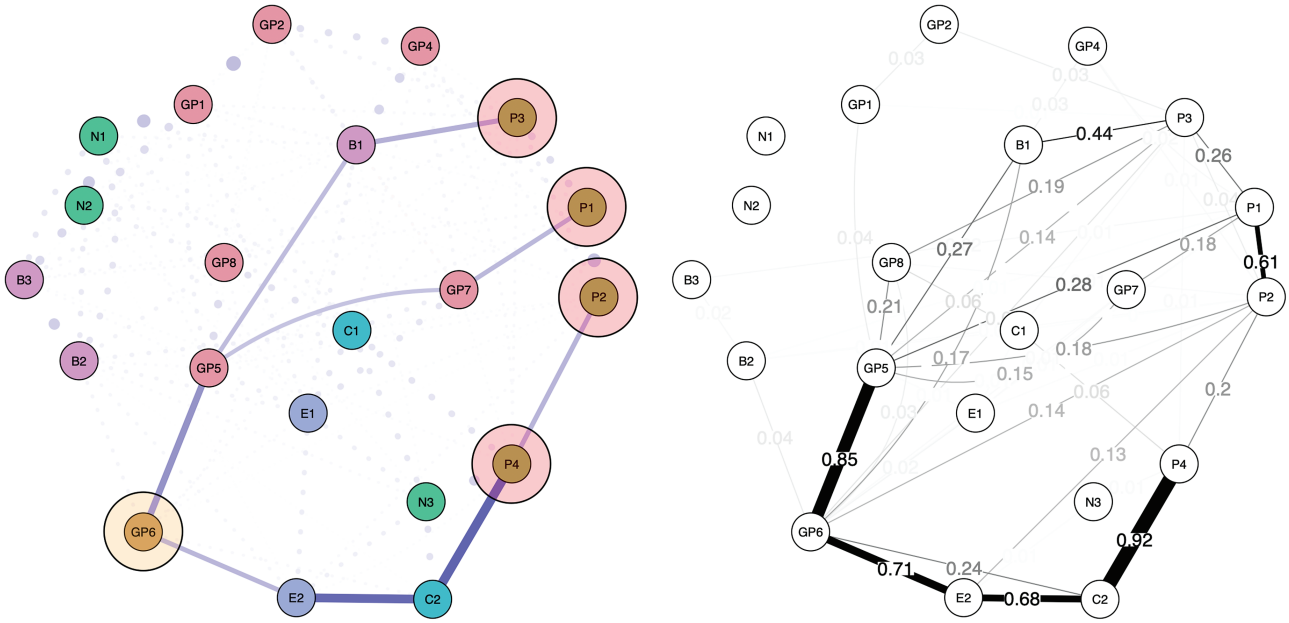

Fig. 4. (Left) Network showing the shortest paths between the OCS and the positive symptoms of the CAARMS scale (P1, P2, P3, and P4) and stability analyses (right). The right panel displays the extent to which the pathways were identified across 1,000 non-parametric bootstraps (eg, the 0.85 edge between *GP6* and *GP5* indicated the edges was identified in 85% of the cases). For a color version, see this figure online.

Discussion

The current study was the first to examine symptom-level associations of OCS and other symptoms of the ARMS in individuals with different levels of risk for psychosis, and the first to evaluate the centrality measures and bridging characteristics of symptoms. Overall, depression was found to be the most central symptom in terms of direct connectivity, while anxiety was the main node linking OCS to other symptom clusters. While OCS did not occupy a central position in the network, they were directly connected to social isolation, anxiety, and blunted affect, and via anxiety and blunted affect to positive CAARMS symptoms. In general, connections between symptoms within each domain were stronger than connections between domains.

In the current study, comorbid depression (27.8%) and anxiety disorders (38.8%) frequently occurred. The

high centrality of depression and bridging characteristic of anxiety suggests that these variables might be essential in their influence on other symptoms of the network. The role of affective symptoms in UHR subjects has been of increased research interest. Meta-analyses reported that over 50% of UHR individuals fulfilled the criteria for a co-occurring depression or anxiety disorder.[33,34] Both depression and anxiety have been found associated with higher symptom severity of UHR symptoms and poorer global functioning.[35,36] Fusar-Poli and colleagues concluded that affective disorders are likely to impact the overall longitudinal outcome of UHR subjects albeit no associations have been found with a transition to psychosis.[34,37] Co-occurring OCD in 9.7% of the current UHR subjects exceed previously reported mean estimates of 5.5% in subjects at increased risk for psychosis,[2] but high variability between studies has been reported.[34] Some previous studies reported associations between OCS and severity of psychotic symptoms on the syndrome level in UHR subjects[7] and psychotic experiences in individuals from the general population.[8] However, the majority of studies do not find associations with positive symptom severity[2,4,38] and a review of the literature does not show an increased or decreased risk for transition to psychosis in UHR subjects with OCS/OCD.[34] In the current CAARMS network, OCS were not identified as central symptoms, nor directly linked to positive symptoms. Low centrality might be explained by the content of the item, which is a more standalone item, compared to most other items that are part of clearly structured subdomains and show stronger within-domain associations. These associations might thus drive other items to be more central, including other general psychopathology items (eg, depression and suicidality are very strongly related to each other). In addition, while other items are associated with a wide array of items from multiple subdomains, the OCS node has less and more well-defined associations, resulting in lower centrality, but suggesting more clear pathways. Direct associations with social isolation can be explained by often observed avoidance and social withdrawal in individuals with OCD and social isolation has also been identified as a predictor for later development of OCD.[39] Shortest paths analyses furthermore showed that OCS were rather indirectly related to symptoms of the positive cluster, via anxiety and blunted affect coupled with observed cognitive change.

One possible interpretation of these interrelations suggests that neurobiological alterations—such as aberrant activation of the dopamine system and associated aberrant salience assignment to irrelevant stimuli causes uncertainty and consequently anxiety and emotional distress.[40] In an attempt to make sense of these experiences and in combination with cognitive alterations such as a tendency to jump to conclusion, psychotic symptoms might develop. Similarly, Szechtman and Woody emphasized feelings of uncertainty accompanied with feelings of insecurity (disturbances in the so-called security motivation system) as important underlying mechanisms of OCS and OCD as an attempt to gain control and reduce distress.[41] Another aspect of the aberrant salience model namely failing to appropriately respond to meaningful reward cues and diminished reward processing has been directly linked to anhedonia and blunted affect.[42] These negative symptoms are closely associated with cognitive deficits,[43,44] which again are conceptually related to disorganization in psychotic disorders.[45] More recently studies suggest that reward dysfunctions and associated blunted affect might also constitute a neuropathological mechanism in OCD.[46] These findings might explain the connecting role of blunted affect in the network and account for the frequent co-occurrence of OCS and psychotic symptoms.[47]

In addition to shared underlying neurobiological alterations, causal interactions on the symptom level might also be assumed. Psychotic-like experiences could lead to increased anxiety, rumination, worries, and doubt, which are strongly related to obsessive thoughts and might be associated with a subsequent attempt to reduce the resulting anxiety through compulsive behavior. In this line, early psychopathological concepts speculated that sometimes OCS might be seen as an attempt to control for the feeling of self-disintegration.[48] In support of this hypothesis, one study found inverse associations between OCS and disorganization.[49] However, more recent research associated co-occurring OCS with dysfunctional coping tendencies,[50] increased general psychopathology and does not support direct associations with increased or decreased severity of positive symptoms, including disorganization.[51] The link between increased anxiety, worry, and fear and the co-occurrence of OCS and psychosis has also been reported on the trait level. Schreuders and colleagues found increased neuroticism in patients with psychosis who reported comorbid OCS compared to those without OCS. Exploratory analyses suggested a mediating effect of neuroticism on the course of OCS severity over time.[52] To disentangle hypothesized causal pathways, analyses of prospective data are necessary.

Taken together, current findings indicate the importance of affective symptoms and their direct relations and interactions with other symptoms of the CAARMS. Noteworthy, in a previous network study, anxiety was found to be the main connective component between experienced traumatic events and psychotic symptoms.[26] Based on these findings, it seems advisable to not only focus treatment on psychotic experiences in UHR subjects but extend the focus to emotional disturbances. Interventions aimed to reduce experienced anxiety and distress could be important treatment targets as they seem to play a central role in the network and might be crucial for the development of symptoms.

Limitations and Future Directions

The current findings should be interpreted considering some limitations. First, while our robustness analysis indicates that the resulting network and centrality indices are interpretable, this should be done with some caution, especially for weaker links in the network and centrality measures.

Second, the current study is exploratory, and interpretation of results is hypothetical in need of further investigations. Moreover, the current analysis was based on cross-sectional data; thus, it is not possible to infer causality, such as the direction of edges. Future research should consider longitudinal data to specify symptoms that actively trigger other symptoms as indicators of the prospective development of the at-risk mental state for psychosis. Unfortunately, due to missing data and the relatively small sample size, we were not able to explore prospective symptom interaction. Along this line, we also chose to only include CAARMS intensity scores in the current study. Future studies in a larger sample should integrate information on frequencies of occurring symptoms.

Third, we included both healthy and UHR subjects in our analysis to evaluate subclinical symptom networks across the risk spectrum for psychosis. This approach led to increased power and accounted for a broad distribution of symptomatic expression at the subclinical level. Nonetheless, we included within supplementary appendix 3 a network structure constructed in the UHR sample only, which shows comparable results, with the note that many of the connections were weaker or missing, indicating a significant loss in power. The edges in the UHR sample only network were mainly—and as expected—a subset of the edges in the overall network structure. Sample sizes were too small to incorporate network comparison tests, investigating whether network structure or centrality measures would be different between groups. Analyses with large enough samples of non-help-seeking controls, UHR subjects, as well as first-episode patients would be desirable.

Fourth, findings are limited by OCS assessment with one item of the CAARMS. Future studies should include a state-of-the-art instrument to assess OCS such as the Yale Brown Obsessive and Compulsive Scale.

Fifth, the majority of individuals included in the current study is Caucasian/White. Replication in a more racially diverse sample is necessary to investigate to which extent findings generalize to non-Caucasian populations.

Finally, in an attempt to ensure more stable network estimates and avoid potential biased results (eg, due to severe violations of normality assumption), we reduced the number of nodes in the network structure by excluding low variability items from the network structures. This led to the exclusion of clinically interesting dimensions such as mania or motor symptoms, which could have contributed to knowledge on the interdependence of mixed affective symptoms and motor symptoms with symptoms of obsession, compulsion, anxiety, and psychosis. For the interested reader, we included an extended network structure with all 28 CAARMS items in supplementary appendix 4. Of note, the network structure is for reference only and should be interpreted with the utmost caution and replication in larger samples is needed. All the main links identified and discussed in the current manuscript are retained in the extended network structure, including the associations between OCS and other symptoms.

Conclusions

We aimed to expand on our understanding of the symptom-level association between OCS and other symptoms of the ARMS, in a large sample of individuals with different levels of risk for psychosis, using novel network models. Our results did not reveal a central role of OCS in the network, nor direct associations with positive symptoms, but indirect associations mainly via anxiety. In addition, results suggested a central role of depressed mood in the symptom network. In sum, these findings point to the critical role of affective symptoms in the network of the ARMS in individuals with different levels of risk for psychosis.

Supplementary Material

Supplementary material is available at https://academic.oup.com/schizophreniabulletin/.

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Conflict of interest

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

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