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Autistic Symptoms and Social Functioning in Psychosis: A Network Approach

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Psychotic and autistic symptoms are related to social functioning in individuals with psychotic disorders (PD). The present study used a network approach to (1) evaluate the interactions between autistic symptoms, psychotic symptoms, and social functioning, and (2) investigate whether relations are similar in individuals with and without PD. We estimated an undirected network model in a sample of 504 PD, 572 familial risk for psychosis (FR), and 337 typical comparisons (TC), with a mean age of 34.9 years. Symptoms were assessed with the Autism Spectrum Quotient (AQ; 5 nodes) and the Community Assessment of Psychic Experiences (CAPE; 9 nodes). Social functioning was measured with the Social Functioning Scale (SFS; 7 nodes). We identified statistically significant differences between the FR and PD samples in global strength ($P < .001$) and network structure ($P < .001$). Our results show autistic symptoms (social interaction nodes) are negatively and more closely related to social functioning (withdrawal, interpersonal behavior) than psychotic symptoms. More and stronger connections between nodes were observed for the PD network than for FR and TC networks, while the latter 2 were similar in density ($P = .11$) and network structure ($P = .19$). The most central items in *strength* for PD were *bizarre experiences*, *social skills*, and *paranoia*. In conclusion, specific autistic symptoms are negatively associated with social functioning across the psychosis spectrum, but in the PD network symptoms may reinforce each other more easily. These findings emphasize the need for increased clinical awareness of comorbid autistic symptoms in psychotic individuals.

Key words: autism/functional outcome/psychosis/schizophrenia/network analysis/network models

Introduction

Psychotic disorders (PD) are typically accompanied by impairments in social functioning, which are often persistent and resistant to therapeutic interventions.[1–3] For many people with a PD diagnosis and their relatives, improvement in the social domain is one of the most preferred outcomes of treatment.[4] One factor that may contribute to poor social functioning in those with psychosis is the presence of autism spectrum conditions, which are characterized by social impairments.[5–9] Symptoms within the psychosis and autism spectra commonly co-occur, and recent evidence suggests comorbid autistic symptoms may negatively impact social cognition and functioning in people with psychosis.[4,10–16] Here, we argue that a better understanding of relations between psychosis, autism, and social functioning is essential to determine appropriate intervention targets.

Network models are relatively novel statistical tools in psychiatric research, which grew popular in the past decade, as they allow zooming into interactions at the symptom level (for clarity, the term "symptom(s)" is used here regardless of whether the underlying assessments are perceived as more indicative of traits or state-like symptoms). The appeal of the network approach is that it can overcome limitations of more traditional research strategies by conceptualizing psychopathology as mutually interacting elements of a complex network (eg, symptoms, biological components, environmental risk factors[17–22]). Network analysis can provide information on symptom relations, such as visual representation of the overall network structure, clustering, as well as the relative centrality of specific symptom clusters or "nodes" (ie, which nodes are connected to most other nodes, as well as which nodes connect specific clusters). In addition, network models

have been argued to be especially useful when investigating links between different domains of interest (eg, comorbid features, risk factors, and symptomatology),[19,23,24] as they may allow pinpointing specific bridging components (eg, nodes, links), which may provide useful information in identifying suitable treatment targets.

Although recent studies investigated symptom network structures in PD populations,[25] as well as the impact of potential etiological[20,26] and comorbid features,[27] none included the relative impact of comorbid autistic features. Zhou et al[28] conducted a meta-analysis and network analysis (n = 2469 college students) of relations between schizotypal and autistic symptoms. The outcome showed that negative symptoms were strongly correlated with autistic social/communicative symptoms, whereas positive symptoms were inversely correlated with autistic symptoms. However, it is unclear to what extent these findings in student populations translate to more heterogeneous nonclinical or clinical populations and whether the co-occurrence of symptoms can have an incremental impact on outcome.

The main aim of the present study was therefore to use a network approach to explore interactions between autistic symptoms, psychotic symptoms, and social functioning. In addition, we aimed to identify specific nodes within the autism and psychosis spectra that may play a pivotal role in determining social functioning in PD. As both psychosis and autism may represent broad phenotypes that can affect individuals at a subclinical level,[29] we compared network characteristics between individuals with different levels of risk for psychosis: individuals with a familial risk for psychosis (FR), who share a genetic and environmental liability to psychosis due to their relatedness and physical proximity to an individual with a PD, and typical comparisons (TC) drawn from the general population with a default risk for psychosis. Building on the theory-driven work of Ziermans et al,[5] who analyzed aggregated sumscores, we applied a data-driven network approach to establish which autistic symptom clusters would be more closely related to social functioning clusters than psychotic symptom clusters. Furthermore, aligned with previous research showing more densely connected networks for clinical populations[27,30,31] we expected that relations within the PD network would be stronger than for FR and TC networks.

Methods

Sample Characteristics

We used data from the Genetic Risk and Outcome of Psychosis (GROUP) study, data release 6.0. GROUP is a multisite longitudinal observational study carried out between April 2004 and December 2013, investigating vulnerability and protective factors that may influence the onset and course of PD. The study was approved by the Medical Ethics Committee of the University Medical Center Utrecht and subsequently by local review boards of each participating institute.[32]

Current analyses included a subset of the full data, from the third data collection time point (ie, 6-year follow-up) and from individuals who completed the measures described below: 504 PD, 572 FR (siblings only), and 337 TC participants, of which 53.9% were male (365, 254, and 153, respectively), with an age range between 21 and 63 years, a mean age of 34.9 years (SD = 8.65), and a mean IQ of 108 (abbreviated IQ based on Arithmetic, Block Design, Digit Symbol-Coding, and Information of the Dutch version of the Wechsler Adult Intelligence Scale—Third Edition[33]). The majority of participants were Caucasian (1244; 88.04%), followed by Mixed ethnicity (94; 6.65%), Surinamese (21; 1.49%), Moroccan (15; 1.06%), Turkish (10; 0.71%), Other (9; 0.64%), Asian (2; 0.14%), Antillean (1; 0.07%), while for 5 individuals (0.35% of the sample) it was unknown. For additional group characteristics see sTable 3 and table 1 in Ziermans et al.[5]

Materials

All individuals completed 3 measures: (1) the Autism Spectrum Quotient (AQ), (2) the Community Assessment of Psychic Experiences (CAPE), and (3) the Social Functioning Scale (SFS), as well as demographic attributes (IQ and age), resulting in 23 variables included in the network. IQ was entered in the networks because it differentiated substantially between groups (see sTable3). All questionnaires are widely used, well-validated instruments.[34–38]

Autistic Symptoms The AQ[39] is a measure of autistic features in individuals. It is a brief, self-administered questionnaire consisting of 50 items (4-point scale: 1 = definitely agree to 4 = definitely disagree). These are divided into five 10-item subcategories: social skills, attention switching, attention to detail, communication, and imagination, scored in the traditional manner, ie, recoded into dichotomous scores with a range of 0–50 for the total score. A higher score indicates a higher level of autistic features in the specified area.

Psychotic Symptoms The CAPE[35] is a measure aimed at detecting psychotic-like experiences. It consists of 42 items (4-point scale: 1 = never to 4 = nearly always), with the reference period for reporting experiences being the past 3 years. The CAPE items correspond to 3 different symptom dimensions: (1) positive, (2) negative, and (3) depression, further subdivided into 9 clusters.[40] These are positive symptoms: bizarre experiences (7 items), hallucinations (4 items), paranoia (5 items), grandiosity (2 items), and magical thinking (2 items); negative symptoms: social withdrawal (4 items), affective flattening (3 items), avolition (7 items), and a single cluster for depression (8 items). A higher score on any dimension indicates higher symptomatology.

Social Functioning The SFS[38] is an assessment of social functioning within the past 3 months. The SFS consists of 7 different dimensions: withdrawal (5 items), interaction (4 items), prosocial (22 items), independence performance (13 items), independence competence (13 items), recreation (15 items), and occupation (max. 8 [conditional] items). A score key is used to calculate raw scores, and a higher score indicates higher level of social functioning.

Network Analyses

We carried out all analyses in the *R* statistical software,[41] using the packages *bootnet*[42] v1.4.3, *qgraph*[43] v1.6.9, and *networktools*[44] v1.2.3. Missing data for individual items (under 1% for the 3 scales) were imputed using the package *mice*[45] v3.11.0. The dimensions of the 3 scales, and *age* and estimated *IQ* as covariates, were included as nodes. An edge between 2 nodes represents a partial correlation between the nodes, while controlling for all other nodes in the network.[46] Blue (red) edges were used to illustrate positive (negative) associations, and the wider and more saturated the edge, the stronger the association.

Network Estimation To estimate the network models, we fitted Gaussian graphical models (undirected network structures) in each group separately. To ensure sparse and interpretable models, we used the least absolute shrinkage and selection operator (LASSO)[47] statistical regularization technique. Specifically, we employed the graphical LASSO algorithm[48] in combination with Extended Bayesian Information Criterion (EBIC) model selection, with a tuning hyperparameter set to .5, which has now been widely used when estimating network structures.[26,49,50] Missing data for individual items were handled using pairwise estimation and Spearman correlations were used when estimating the networks, as recommended for nonnormality distributed data.[51] The network layout was matched using the average of the Fruchterman and Reingold algorithm[52] (ie, each network was first plotted individually and then the layout was averaged), which places the nodes with stronger connections closer to the center of the network.

Network Comparison Network structures were constructed and compared using control, family, and PD data. To assess the differences between samples we used the Network Comparison Test (NCT).[53] The NCT is a permutation test that investigates differences in the global strength of the networks, their structure, as well as differences in individual edges and centrality measures. We only investigated differences in edges when either the structure or global strength was identified as significantly different. Due to the conservative nature of the NCT, we report results both with and without a Bonferroni correction.

Centrality Estimation Centrality indices[54] were computed to investigate which nodes display the highest connectivity and play a central role in bridging different dimensions. Specifically, we computed strength (ie, a measure of how well connected a node is) and bridge strength (ie, a measure of how well a node connects to other clusters). These measures were chosen as they were well aligned with our research aims, while still expected to be robust.[42] Predictability and expected influence centrality estimates were further computed and included in Appendix 1 in the Supplementary Online Content, for the interested reader. Of note, expected influence disregards the presence of negative edges. As for the SFS assessment a higher score indicates better social functioning, we considered negative edges to be of interest here and thus focused our main analysis on strength and bridge strength centrality metrics.

Robustness Analysis Finally, we conducted a robustness analysis to assess the stability and accuracy of our results,[42] available in Supplement, Appendix 2.

Results

Network Analysis and Comparison

sTable 1 in the Supplementary Online Content presents the means and SDs of all individual items. No statistically significant differences were identified between the FR and the TC sample in global strength (P = .11) and

Table 1. Edge Differences Between Family and PD Samples

Variable 1	Variable 2	*P* Value
Social skills	Attention switching	.007
IQ	Imagination	.044
Social skills	Imagination	.047
Bizarre experiences	Hallucinations	<.001*
Attention switching	Grandiosity	.011
Imagination	Grandiosity	.008
Paranoia	Grandiosity	.006
Bizarre experiences	Magical thinking	.001
Grandiosity	Magical thinking	.001
Bizarre experiences	Social withdrawal	.026
Communication skills	Affective flattening	.039
Affective flattening	Avolition	.011
IQ	Depression	.008
Attention to detail	Depression	.048
Avolition	Prosocial activities	.007
Withdrawal	Prosocial activities	.013
Imagination	Independence performance	.036
IQ	Independence competence	.049
Independence performance	Independence competence	<.001*
Paranoia	Recreational activities	.003
Prosocial activities	Recreational activities	.008
Imagination	Occupation employment	.002*
Withdrawal	Occupation employment	.002
Prosocial activities	Occupation employment	.024
Independence competence	Occupation employment	<.001*

Note: PD, psychotic disorders.
*Remained significant after Bonferroni correction.

network structure (P = .19), and therefore we focus our results on the comparison between the FR and the PD sample, of which sample sizes are higher and more comparable.

Figure 1 presents the network structures (1) for the FR sample, (2) for the PD sample, (3) highlighting edge (presence and strength) and centrality differences between the 2 networks as identified by the NCT. Figure 1c includes 2 network structures, as to allow pinpointing where specifically the differences lie (eg, in the presence/absence of edges, as would be the case with the edge between IQ and D, which is present in the PD sample only, or in the strength of the edges, as would be the case with for instance the edge between N2 and N3, which is present in both networks). Overall, within both networks, negative symptoms and depression were more strongly linked to positive psychotic symptoms than autistic symptoms, while autistic symptoms were more closely related to social functioning than psychotic symptoms. The items *social skills* (A1), *attention switching* (A2), and

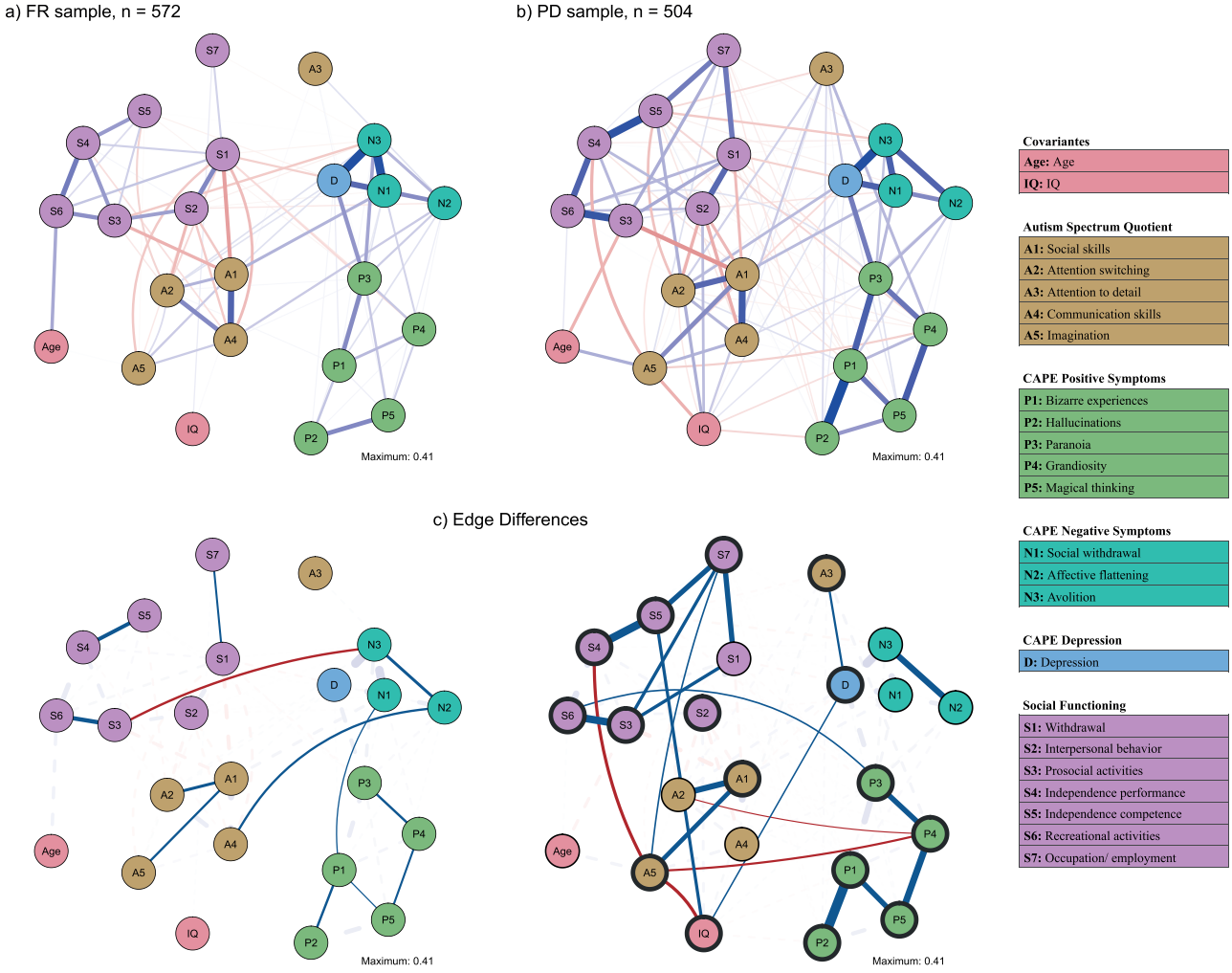

Fig. 1. Network structure (a) for the family sample (left panel); (b) for the PD sample (right panel); (c) highlighting significant edge differences between the family sample and PD sample (bottom panel). A gray and wider border around a node (bottom panel) indicates a significant difference in strength centrality for that node between the 2 groups (ie, a higher value in the group where the border is present). The nodes represent the different dimensions of the Community Assessment of Psychic Experiences, Autism Spectrum Quotient, Social Functioning, and covariates. Item groups are differentiated by color. The dashed edges in the background (bottom panel) indicate the presence of other edges in the network, which were not identified as significantly different between the networks. 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. *Note*: FR, familial risk; PD, psychotic disorders.

Table 2. Strength Centrality Differences Between Family and PD Samples

Variable	*P* Value
IQ	<.001*
Social skills	.002*
Attention detail	<.001*
Imagination	<.001*
Bizarre experiences	<.001*
Hallucinations	<.001*
Paranoia	<.001*
Grandiosity	<.001*
Magical thinking	<.001*
Depression	.033
Interpersonal behavior	.008
Prosocial activities	.028
Independence performance	<.001*
Independence competence	<.001*
Recreational activities	.049
Occupation employment	<.001*

Note: PD, psychotic disorders.
*Remained significant after Bonferroni correction.

communication skills (A4) were negatively linked to *withdrawal* (S1) and *interpersonal behavior* (S2). For both networks, the item *attention to detail* (A3) did not cluster well with the remaining autism nodes, and instead was more closely related to the CAPE positive psychotic symptoms and depression. Within both networks, *communication skills* (A4) were linked to *paranoia* (P3) (this link is less visible for the FR network, but nonetheless present and not identified as different in edge strength according to the NCT), while *attention switching* (A2) was linked to *bizarre experiences* (P1) and *depression* (D).

Of note, we identified statistically significant differences between the FR and PD samples both in global strength ($P < .001$) and in network structure ($P < .001$). Furthermore, we identified several statistically significant differences between edges (see table 1) and centrality measures of the 2 networks (see table 2). Especially within the same domain, relations were more connected for the PD network, as more and stronger connections between nodes were observed than for the FR network. All links between autism spectrum items were stronger for the PD network, as well as between the social functioning items and psychotic experiences items. Within the PD network, the item *paranoia* (P3) was positively linked to *recreational activities* (S6), while the item *grandiosity* (P4) was negatively linked to *attention switching* (A2) and *imagination* (A5).

Centrality Analysis

Figure 2 illustrates the node strength for each node in the network, both for the FR and the PD samples. The 3 most central items in terms of strength for the FR sample were *avolition* (N3), *social withdrawal* (N1), and *depression* (D), while for the PD sample these were *bizarre experiences* (P1), *social skills* (A1), and *paranoia* (P3) (see sFigures 11–14 for bootstrapped difference tests).

Figure 3 illustrates the bridge strength for each node included in the network, both for the FR and the PD samples. The 3 most central items in terms of bridge strength for the FR sample were *withdrawal* (S1), *social skills* (A1), and *attention switching* (A2), while for the PD sample these were *IQ*, *imagination* (A5), and *social skills* (A1).

Robustness Analysis

The results of the robustness analyses are presented and described in Supplementary Online Content, Appendix 2. Generally, the results show high stability both for the FR and the PD network structures Of note, the CS coefficient for bridge strength for the PD network was below the preferred 0.5 cutoff score, but above the recommended 0.25. We therefore advise caution when interpreting this measure.

Discussion

The current study presents a bird's-eye view of relations between autistic and psychotic symptom clusters, and social functioning in individuals with PD, FR, as well as TC. Our network analyses revealed the following: (1) across all group networks, all autistic nodes, except attention to detail, were more closely related to nodes reflecting frequency of social interactions than psychotic symptom nodes; (2) the FR an TC networks were highly similar in terms of global strength and network structure, prompting further focus on PD and FR network comparisons; (3) the PD network was generally characterized by stronger and more connections than the FR network, though only few individual edges differed significantly after Bonferroni correction; and (4) centrality measures indicated that the relative importance of single nodes may differ between the PD and FR networks. The implications of these findings are discussed further in the paragraphs below.

Importantly, network analysis as a statistical technique is best thought of as an exploratory technique that identifies patterns of conditional independence that may be interpreted as empirical phenomena. These phenomena can hold clues that facilitate a better understanding of the causal structure and dynamics that characterizes disorders.[55,56] By using a purely data-driven approach that creates an actual image of how symptom clusters may interact with each other, we were able to show that for both the FR and PD networks, autistic symptom clusters were more directly related to social functioning nodes than psychotic symptom clusters. In particular, the autistic nodes "Social skills," "Attention switching," and

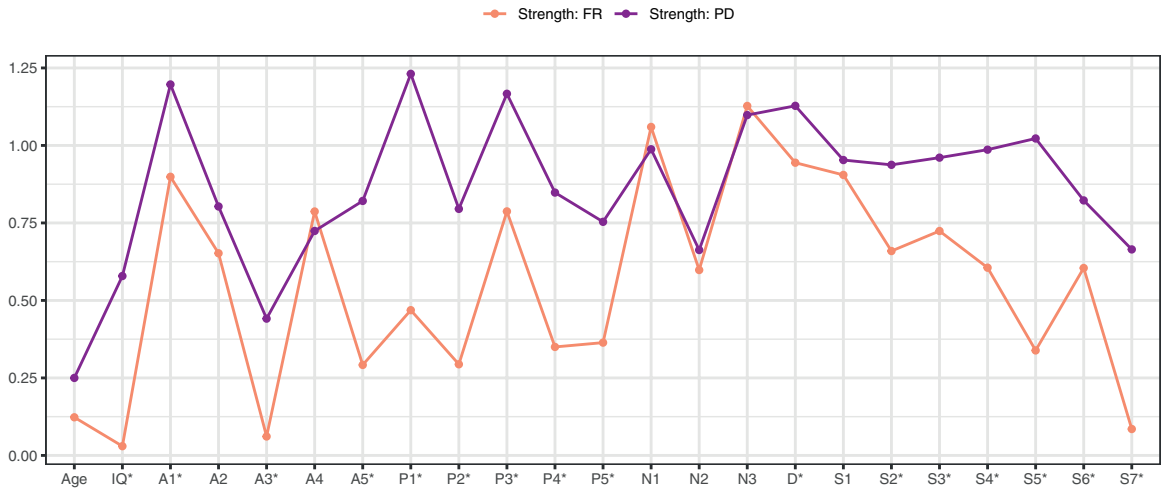

Fig. 2. Strength centrality for each node included in the network. The orange line indicates strength centrality for the familiar risk (FR) sample, while the purple line indicates strength centrality for the psychotic disorder (PD) sample. For a color version, see this figure online. *Remained significant after Bonferroni correction.

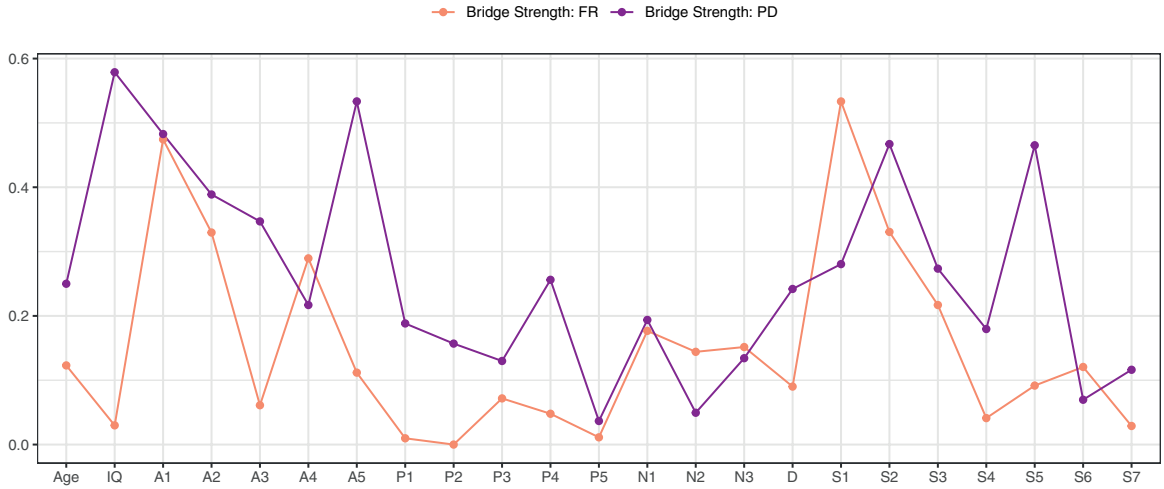

Fig. 3. Bridge strength centrality for each node included in the network. The orange line indicates bridge strength centrality for the familial risk (FR) sample, while the purple line indicates bridge strength centrality for the psychotic disorder (PD) sample. For a color version, see this figure online.

"Communication skills" were negatively linked to SFS "Withdrawal" and "Interpersonal behavior." While commonly clumped together into a single "social interaction" factor,[36] our results indicate that using standardized assessment of specific autistic symptoms instead—which tend to be relatively stable phenotype—may help better identify social vulnerabilities and resilience in PD, in line with previous findings.[8,57,58] Further, our results show both "Paranoia" and "Social skills" to have a negative edge with "Withdrawal." Therefore, this could be an area within the network where one might expect an interaction effect. Specifically, experiencing both increased paranoia and poor (autistic-like) social skills could be related to severe social withdrawal. In addition, focusing on withdrawal (which was also identified to have high bridging properties in both populations) may in turn facilitate better outcomes.

One of the general objectives of the GROUP project[32] is to investigate vulnerability factors contributing to the expression of psychosis. However, the current FR network structure was highly similar to the TC network. This could suggest that interrelations between autistic, psychotic, and social functioning features are either a poor indicator of latent manifestations of PD, or they may only become clinically relevant for psychosis onset in high-risk individuals if other (mediating) factors are entered into the equation. There is some evidence in favor of the latter, as a recent network study based on data collected with the experience sampling method showed that inclusion of a "stress" node resulted in a higher number

of network connections, with stress having a central position in the network structure across TC, FR, and PD groups, and also showing direct connections with subsequent psychotic experiences.[59] Future studies including high-risk samples should aim to replicate current findings and are encouraged to incorporate known risk factors, such as distress and childhood trauma[26,60] into network structures.

Given its global similarity with the TC network and the larger and more comparable sample size, the FR network was subsequently used for network comparisons with PD. In line with our expectation, the PD network was denser than the FR network, with more connections identified especially between nodes of the same domain. This included the connection between "Hallucinations" and "Bizarre experiences," one of the main links surviving Bonferroni correction (table 1) and indicating the strong presence of feedback loops between the psychotic symptoms. Denser network structures have been previously reported for PD,[59] and more so for nonremission compared to remission,[27] but also for other clinical populations.[30,31] Presumably, and in line with network theory,[18] this could reflect the capacity of symptoms to reinforce each other much more steadily, resulting in a higher level of vulnerability,[30] eg, the occurrence of high frequent hallucinations may more easily trigger an increase of other positive symptoms such as thought interference (as indicated by the "Bizarre experiences" node, in some studies referred to as "Delusions"[61]). However, denser networks may also reflect a greater potential for more (transfer) effect of treatments. In line with this, it was found that treatment-responsive individuals diagnosed with PD had more densely connected symptom networks after antipsychotic treatment than did treatment-responsive individuals at baseline,[62] which could indicate that in the presence of a more densely connected network structure (ie, even under greater severity), treatment may be more efficient. Identifying how these specific mechanisms act may be an important next step in the field of network modeling. Of note, research in other domains—especially depression—also identified less dense network structures in clinical populations.[63,64] These findings could indicate that symptom reactivity may be different across various mental disorders, and raises the question of whether network topology differs across distinct mental health conditions.

Centrality analyses revealed many statistically significant differences between the FR and PD sample (see figure 2 and table 2), with most surviving the Bonferroni correction. The strongest nodes for FR were negative and depressive symptoms and for PD positive and autistic symptoms, suggesting that the relative importance of single nodes differs between populations. For the PD group, such findings emphasize that an increase in positive and autistic symptoms may quickly impact neighboring nodes and thus lead the network structure faster into a disorder state, while for the FR group a focus on affective symptoms may be warranted. While at this stage this remains hypothesis generating, longitudinal research can further investigate these timescales in future studies, preferably with adjusted questionnaires that allow for more variation to capture differences in trait and state-like symptoms.

In terms of bridge centrality (figure 3), ie, how well individual nodes connect to other node clusters, the overall profile was rather different for FR and PD. For brevity, we highlight only 2 individual nodes here, fully acknowledging that other findings might merit further discussion as well. First, although IQ was virtually irrelevant within the FR network, and showing marginal strength in the PD network, it does show widespread connections to individual nodes in the PD network structure. This illustrates that lower IQ is as much a part of the clinical phenotype of PD as any other node in the network and should not automatically be "controlled" for in experimental group comparisons. Second, "Attention to detail," unlike any other node, does not cluster within its expected domain (ie, AQ node cluster) and is relatively unrelated to other nodes. This is strikingly similar to the network results in a nonclinical student population,[28] as well as with previous research that identified, in 2 clinical samples, attention to detail to be the least central node and only weakly connected to other nodes.[65] This finding further fits with different factor models of the AQ, in which attention to detail comprises its own separate factor. We therefore suggest attention to detail is subject to further scrutiny with regard to its construct validity.

The current study should be considered in light of several limitations. First, our analyses are exploratory in nature and our findings are hypothesis generating. In light of this, we chose to use the *EBICglasso* algorithm in the estimation of the network, erring on the side of discovery. Of note, however, is that this may be more prone to specificity concerns[51,66] and as such we recommend caution in interpreting smaller edges and replication of results. Second, current results are based on cross-sectional data and thus causality cannot be inferred. Third, our study did not identify statistically significant differences between the TC and FR sample in global strength and structure. This may be a real effect, or may result from limited power of the NCT to identify effects (ie, the control sample is considered small for network analysis). Further, scientific conclusions and policy decisions should not be based only on whether a *P* value passes a specific threshold.[67] It is thus essential for future research with larger sample sizes to attempt to replicate current results. Given these findings, as well as our aim to retain comparable sample sizes while comparing the networks using the NCT (ie, as to avoid effects due to sample size), we chose not to collapse TC and FR samples, but keep these independent. Of note, while means (and thus severity) should not play a role in the network structure and the NCT, the variance could affect the results if floor

or ceiling effects are present. Here, we did not identify such pronounced effects. Fourth, despite their psychometric strengths and common use, self-report questionnaires have inherent limitations to capture psychological phenomena (eg, cultural bias, debatable factor structures). In addition, some of the subdomains of AQ and SFS could be argued to be conceptually related constructs. Nonetheless, overlap in measurement can usually be seen through very strong connections between 2 items in a network, which was not the case here (ie, not even for the 2 withdrawal items). Correlations of AQ and CAPE were modest and response types for all questionnaires were quite different and as such we do not expect current results and conclusions to be explained away by measurement overlap. Finally, denser network structures could also be an indication of the presence of a common cause (eg, a central dysfunction in the brain), but this seems unlikely as even constructs within the same domain (eg, positive vs negative psychotic symptoms) are generally considered to derive from different etiological pathways.

Conclusions

To conclude, the present study was the first to use a data-driven network approach in an aim to explore interactions between autistic symptoms, psychotic symptoms, and social functioning across different levels of risk for psychosis. While the presence of autistic symptoms has a general negative effect on social functioning, in the PD network autistic and psychotic symptoms may reinforce each other more strongly than for unaffected individuals, and thereby affect social functioning more steadily. On the one hand, this suggests an increased vulnerability for bispectral symptomatology, on the other hand a greater potential for more (transfer) effect of treatments. Consequently, these findings emphasize the need for increased clinical awareness and default assessment of comorbid autistic symptoms in individuals with a PD to help enrich their daily social environments.

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Supplementary Material

Supplementary material is available at *Schizophrenia Bulletin* online.

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