

**Distress, impairment and the extended psychosis phenotype: A network analysis of psychotic experiences in a US general population sample**

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## Abstract

It has been proposed that subclinical psychotic experiences (PEs) may causally impact on each other over time and engage with one another in patterns of mutual reinforcement and feedback. This subclinical *network* of experiences in turn may facilitate the onset of psychotic disorder. PEs however are not inherently distressing, nor do they inevitably lead to impairment. The question arises therefore, whether non-distressing PEs, distressing PEs, or both, meaningfully inform an *extended psychosis phenotype*. The current study first aimed to exploit valuable ordinal data that captured the absence, occurrence and associated impairment of PEs in the general population to construct a general population based *severity network* of PEs. The study then aimed to partition the available ordinal data into two sets of binary data to test whether an *occurrence network* comprised of PE data denoting absence (coded 0) and occurrence/impairment (coded 1) was comparable to an *impairment network* comprised of binary PE data denoting absence/occurrence (coded 0) and impairment (coded 1). Networks were constructed using state-of-the-art regularized pairwise Markov Random Fields (PMRF). The *severity network* revealed strong interconnectivity between PEs and nodes denoting paranoia were among the most central in the network. The binary PMRF *impairment network* structure was similar to the *occurrence network*, however the *impairment network* was characterised by significantly stronger PE interconnectivity. The findings may help researchers and clinicians to consider and determine how, when and why an individual might transition from experiences that are non-distressing to experiences that are more commonly characteristic of psychosis symptomology in clinical settings.

**Key words:** psychotic experiences, psychosis phenotype, psychosis continuum, network analysis, epidemiology; schizotypy

## Introduction

Evidence that variation in the psychosis phenotype can be better represented by the concept of a continuum stems from decades of research indicating that schizotypal traits are commonly identifiable in ‘healthy’ individuals,<sup>1,2</sup> and by more recent discoveries indicating that large numbers of individuals in the population report subclinical psychotic experiences (PEs) without seeking psychiatric treatment<sup>3</sup> (although they may seek help in other ways<sup>4</sup>). Evidence has also shown however that those who experience PEs are often at higher risk of *transitioning* to psychotic disorder.<sup>5,6</sup>

Moreover, while PEs are transitory in about 80% of individuals, around 20% go on to develop persistent PEs and 7% go on to develop a psychotic disorder<sup>6-8</sup> In most cases however it seems PEs are not associated with distress, and do not lead to a malign outcome.<sup>9</sup> Some authors<sup>10,11</sup> therefore have argued that PEs in the general population are distinct from *true* symptoms of psychosis, as they are often too mild and transient to be clinically meaningful,<sup>12</sup> and are not specific to psychotic disorder.<sup>13,14</sup> An important question arises therefore regarding the nature of PEs i.e. whether non-distressing experiences, distressing experiences, or both, should meaningfully inform a continuum.

### *The extended psychosis phenotype*

Offering a unique and eloquent perspective from which to consider the possible ‘evolution’ of the psychosis phenotype from schizotypal traits and PEs at one end of the proposed continuum to clinically relevant symptom expression at the other, van Os and Linscott proposed that the onset of psychotic disorder may be explained in part by “subclinical experiences causally impacting on each other over time” [p.227].<sup>15</sup> Promoting an *extended psychosis phenotype* and advocating a *network* perspective, these authors proposed that the onset of psychotic disorder may be preceded and explained by nuanced and complex

interactions between individual PEs in the general population. However, given that PEs often do not negatively affect individuals in terms of functioning and well-being and given that PEs are often experienced positively<sup>16-20</sup> it remains to be qualified whether the extended psychosis phenotype makes reference only to experiences that result in impairment or distress or whether it is inclusive of non-distressing PEs also. A number of studies that have compared PEs in individuals with and without a need for care,<sup>14, 21-23</sup> seem to suggest that the extended phenotype is likely to be inclusive of PEs that may be considered to be ‘non-distressing’.

For example, Peters et al.<sup>14</sup> compared people with persistent PEs and no "need for care" with patients diagnosed with a psychotic disorder and controls without PEs, in terms of their phenomenological, socio-demographic and psychological features. Their results showed that non-clinical individuals experienced hallucinations in all modalities as well as first-rank symptoms, with an earlier age of onset than those in the clinical group. Moreover, somatic/tactile hallucinations were more frequent in the non-clinical group also, while commenting and conversing voices were rare. Participants in the non-clinical group were differentiated from their clinical counterparts by being less paranoid and deluded, apart from ideas of reference, and having fewer cognitive difficulties and negative symptoms. Importantly, unlike the clinical group, those in the non-clinical group were characterized neither by low psychosocial functioning nor by social adversity.

In a review of auditory verbal hallucination (AVH) research findings Johns et al.<sup>21</sup> showed that cross-sectional comparisons of individuals with AVHs with and without need for care revealed similarities in phenomenology and some underlying mechanisms but also highlighted key differences in emotional valence of AVHs, appraisals, and behavioural responses. Longitudinal studies suggested that AVHs were an antecedent of clinical disorders when combined with negative emotional states, specific cognitive difficulties and poor

coping, plus family history of psychosis, and environmental exposures such as childhood adversity. A more recent review of this literature<sup>22</sup> also suggests continuity in AVH experience between clinical and ‘healthy’ voice hearers. In this review both groups seem similar in relation to e.g. subjective, perceptual experiences of voices and brain activity during hallucinatory experiences. Risk factors such as childhood and familial trauma also appear similar between groups. Groups differ significantly however in e.g. beliefs about voices, control over voices, voice related distress and affective difficulties.

In addition to this, Brett et al.<sup>23</sup> compared PEs among patients diagnosed with a psychotic disorder, with help-seeking ultra-high risk (UHR) individuals and non-clinical individuals presenting with enduring PEs. All groups reported "positive" experiences, such as ideas of reference and hallucinations, with the non-clinical group displaying more PEs in the paranormal/hallucinatory component than both clinical groups. These researchers concluded that help-seeking and need-for-care were associated with the presence of subjective cognitive disturbances and that anomalies of cognition and attention may have been more relevant to poorer outcomes than the presence of anomalous experiences. Collectively, these studies seem to suggest that PEs can commonly emerge in both clinical and non-clinical settings but that they are ultimately differentiated from one another by a range of other explanatory variables such as e.g. compromised functioning, adversity, negative emotional states, environmental exposures, and/or family history of psychotic disorder etc.

An exploration of this extended phenotype, where subclinical experiences are assumed to causally impact upon each other, would seem to require an analytic framework that is capable of statistically modelling the potential contribution of each symptom/experience in a psychosis taxonomy to all other symptoms/experiences, i.e. a *network model*. Moreover, to adequately test whether non-distressing PEs meaningfully inform this *extended phenotype* this analytic framework would seem also to require data that

captures not only the occurrence of PEs but the associated impairment/distress of the experiences also.

### *Network Analysis*

Network Analysis, now commonly employed by researchers in various fields, (e.g. clinical psychology,<sup>24-27</sup> psychiatry,<sup>28, 29</sup> personality research<sup>30, 31</sup> and social psychology<sup>32</sup>) is an analytic framework where correlations between symptoms are no longer explained by a common latent factor, but instead are conceptualized as complex systems, where individual symptoms have autonomous causal power to influence one another (see review<sup>33</sup>).<sup>34-36</sup> To date in the psychosis literature Network Analysis has been employed to investigate potential pathways between psychosis symptoms in clinical data,<sup>28,37</sup> transdiagnostic experiences surrounding auditory verbal hallucinations (AVHs),<sup>38</sup> and the interplay between environmental risk factors, expression of psychosis, and symptoms of general psychopathology in prospective general population cohort data.<sup>39</sup> While these studies have certainly illustrated the potential value of Network Analysis to elucidate psychosis symptom/disorder variation in a clinical context and in the context of recognised risk, no known study as yet has exploited the technique to explore the proposed continuum of psychosis independently of risk.

Network Analysis may afford a novel and valuable opportunity therefore to explore the extended psychosis phenotype by modeling PE interplay in the general population. Moreover, it may afford an opportunity to evaluate whether a network that does not discriminate between PE occurrence and impairment, is comparable in *form and function* to one where PEs are discretely characterised by personal and social impairment only.

The current study sought to model these alternative perspectives by estimating three network models using valuable ordinal data that captured the absence, occurrence and

associated impairment of PEs in the general population. The first research aim involved estimating a *PE severity network* using the data in its entirety. The second research aim partitioned the ordinal data into two sets of binary data to test whether a *PE occurrence network* (i.e. PE not experienced versus any PE experienced regardless of distress/impairment) mirrored a *PE impairment network* (i.e. PE not experienced or experienced without distress/impairment versus PE experienced with distress/impairment). Given the strength of associations between positive PEs (and symptoms and dimensions) evidenced in the factor analysis literature,<sup>40 - 43</sup> it was hypothesized that a strongly connected network would emerge in the *severity network*. Moreover, given the extant literature regarding potential positive psychosis symptom *interplay*, particularly that featuring persecutory/referential delusions and hallucinations,<sup>44 - 47</sup> it was anticipated that either paranoia or hallucinatory experiences (or both) would occupy central positions within the network. Finally, in light of available evidence where PEs have been shown to be phenomenologically similar between those with and without a need for care<sup>14, 21-23</sup> it was predicted that a *PE occurrence network* would be comparable to a *PE impairment network* and that the pattern of associations between PEs in each would be consistent. More specifically it was predicted that a PE network that was inclusive of non-distressing PEs would mirror a network where PEs reflected distressing experiences only.

Testing these hypotheses may not only advance our understanding of the potential interplay between subclinical psychotic phenomena but may also help researchers and clinicians alike to consider and, in time, determine how, when and why an individual might transition from experiences that are non-distressing to experiences that are more commonly characteristic of psychosis symptomology in clinical settings.

## Method

### *Sample*

Analysis was conducted on the second wave of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC).<sup>48</sup> The NESARC is a longitudinal survey that was designed to be representative of the civilian, non-institutionalized adult population of the United States, including residents of the District of Columbia, Alaska, and Hawaii.<sup>48</sup> Descriptions of the survey design, and data collection processes, available in greater detail elsewhere,<sup>49–52</sup> are also summarized in the supplementary materials.

### *Measures*

The NESARC made use of the Alcohol Use Disorder and Associated Disabilities Interview Schedule—DSM-IV version (AUDADIS-IV).<sup>52</sup> The AUDADIS-IV is a fully-structured, self-report, diagnostic interview designed to be administered by clinicians or trained laypersons.<sup>52</sup> The AUDADIS-IV assesses both past year and lifetime occurrence of a variety of psychiatric disorders, including psychosis.<sup>51</sup> The AUDADIS-IV measures of psychiatric disorders have been shown to demonstrate high reliability in general population samples.<sup>51, 53</sup>

### *Psychotic Experiences*

Sixteen PEs were drawn from Section 10 of the AUDADIS-IV - “Usual Feelings and Actions”. Each PE was associated with one of three distinct schizotypal dimensions; ‘Social/Interpersonal’ e.g. ‘Have you felt suspicious of people, even if you have known them for a while?’; ‘Disorganization’ e.g. ‘Have people thought you are odd, eccentric or strange?’; Cognitive/Perceptual e.g. ‘Have you often thought that objects or shadows are really people or animals, or that noises are actually people’s voices?’. Respondents were asked if they had ever experienced a PE (Yes/No response option). Each specific PE item also had a follow-up question that enquired about any distress or impaired functionality that



may have been associated with that PE (i.e. “Did this [experience] ever trouble you or cause problems at work or school, or with your family or other people”).

### *Missing data*

In total, 182 (0.5% of the sample) individuals had complete missing data (i.e. across all 16 PEs). These cases were excluded from the analysis. An additional 929 adults (2.7% of the sample) had missing data on one or more PE however these were coded as missing (*NA*) and were retained in the analysis, resulting in analytic sample of 34,471.

### *Data analysis*

The network analysis was conducted in a number of stages. Details of the analyses, associated output and the R-code used to conduct the modelling is available in the supplementary material.

### *Network estimation*

A popular network model to use in estimating psychological networks is the state-of-the-art Pairwise Markov Random Field (PMRF).<sup>54-56</sup> A PMRF is a network in which nodes represent variables (in this case PEs), connected by undirected *edges*, which in turn indicate conditional dependence between two variables (PEs).<sup>54</sup> For the purposes of this study, three PE networks were estimated, using both ordinal (i.e. the *severity network*) and binary data (i.e. the *occurrence network* and *impairment network*).

### *Centrality estimation*

Quantifying the importance of each PE to each network is achieved by estimating three indices of node centrality: (a) strength, (b) closeness, and (c) betweenness.<sup>56, 57</sup> Node strength is a measure of the sum of the weights of the edges (i.e. correlation magnitudes) attached to that node. It is the most important centrality estimate for psychopathological research,<sup>58</sup>

given that high strength nodes indicate the increased likelihood that (in this instance) the activation of a PE will be followed by the activation of other PEs.

Node closeness represents the average distance between a given node and the remaining nodes in the network. In the current study, PEs with high closeness estimates may reflect those experiences that are likely to be quickly affected by changes in other PEs either directly or through changes between other PEs.

Finally, node betweenness equals the number of times that a node lies on the shortest path between two other nodes.<sup>59</sup> The importance of nodes with high betweenness estimates relates to their removal from the network; if this were to occur, the distance between other paths would generally increase.<sup>55</sup> For all measures of centrality, higher values reflect a nodes greater centrality to the network.<sup>58</sup>

### *Visualisation*

The nature of an edge is indicated by both colour (green and red lines represent positive and negative connections, respectively) and thickness (thicker lines represent stronger connections; thinner lines represent weaker connections).<sup>60</sup> The R package *qgraph*<sup>60</sup> implements the Fruchterman and Reingold algorithm,<sup>61</sup> which graphically positions strongly correlated nodes together.

## **Results**

Table 1 here

### *PE Severity network*

A description of the node labels can be seen in Table 1. Here, the resulting network (Fig. 1) was well connected, with no isolated nodes. Especially strong connections emerged between e.g. nodes 4 (supernatural) and 6 (force); nodes 11 (emotion) and 12 (express); and between

nodes 15 (act strange), 14 (ideas) and 8 (odd). Other connections were absent, for instance between Node 5 (sixth sense) and Node 9 (close to); this implied that these symptoms were statistically independent when conditioning on all other symptoms (i.e. their regularized partial correlation was zero).

Figure 1 here

Edge thickness suggested a *corridor* of nodes e.g. running from the *top* of the network (nodes 11 & 12) along the *perimeter* (via nodes 9, 10, 13, 2, 1, 3) to the *bottom* of the network (to nodes 5, 7, 6, 4; *implied direction for descriptive purposes only*; see Figure 2 and discussion).

Figure 2 here

#### *Centrality estimates*

Fig 3 displays the centrality estimates from the *severity* network. Node 15 (act strange) had the highest *strength* estimate, followed by Nodes 2 (being watched), 4 (supernatural), 5 (sixth sense), 8 (odd) and 13 (suspicious). Node 13 (suspicious) and Node 2 (being watched) had the highest *closeness* estimates in the network, meaning that these experiences were likely to be quickly affected by changes in other PEs. Thus, Nodes 13 and 2 had strong influence in the network due to the short paths that connected them to other PEs. In relation to high *betweenness*, Node 10 (feel nervous) and Node 2 (being watched) were central, which indicated that if these PEs were removed from the network, the distance between other paths would generally increase. The centrality indices were substantially related; for the 16-item PE, correlations were 0.63 (B~C), 0.70 (B~N), and 0.60 (C~N).

Figure 3 here

#### *Network accuracy & stability*

Figures 1 - 3 in the supplementary material show the results from the bootstrapping procedure of the centrality estimates from the severity network. As expected due to the large sample,

the stability of all estimates perform very well. The stability of centrality estimates can be quantified using the correlation stability (CS)-coefficient.<sup>54</sup> The results revealed that although the betweenness estimate was not stable (CS-coefficient=0.43), both closeness and node strength were stable (CS-coefficients of 0.59 and 0.75, respectively) and therefore can be interpreted with confidence. The node with the largest strength, Node 15 (actstrange), was significantly larger than all other nodes.

#### *PE occurrence network versus PE impairment network*

Panels A and B in Figure 4 display the networks for the PE occurrence and the PE impairment networks respectively. The test statistic for the difference in global strength (i.e. connectivity; weighted sum of absolute connections) between the PE impairment and PE occurrence network was statistically significant (17.549;  $p = <0.001$ ), meaning that the PE impairment network was more densely connected than that of the PE occurrence network (see supplementary materials). The network structure comparison test was also statistically significant (1.1272;  $p = <0.0001$ ), which means that the network structures (the topology) differed from each other. As a follow-up to this omnibus test, we therefore investigated which particular edges differed across the two networks (i.e. we compared all individual edges).

Results showed that there was no statistical difference between 73% of the edges in the occurrence and impairment networks. Both networks generally possessed the same edge structure, in that edges within the occurrence network were also evident in the impairment network.

However, a number of edges were statistically stronger in the impairment network compared to the occurrence network e.g. edges between Nodes 9 (closeto) and 10 (nervous); Nodes 16 (shadows) and 2 (watched); Nodes 13 (Suspicious) and 12 (express); Nodes 11 (emotion) and 12 (express); and between Nodes 13 (suspicious) and 1 (meaning) were

significantly stronger in Panel B than in Panel A. In total 37 edges statistically differed in strength between networks (see Table 1a supplementary materials).

Figure 4 here

## Discussion

Using the available data that denoted PE absence, occurrence and impairment, the ordinal PMRF model returned a well-connected network with visibly stronger connections between specific *clusters* of experiences.

### *The network of PEs*

Specifically, four distinct but strongly connected clusters of PEs seemed to scaffold the network. First, disorganization PEs (nodes 8, 14 and 15) seemed to congregate and occupy a distinct and separate space. Characterised notably by the *attributional* nature of the PEs ('have people thought you...') nodes 8, 14 and 15 suggested that disorganized experiences/symptoms may be a distinct set of reinforcing experiences in the general population that may be less influenced by other PEs. Notably, these PEs had some of the lowest *closeness* estimates indicating that they were some of the least likely to be affected by changes in other PEs.

Second, and occupying the lower left quadrant of the network, a constellation of strongly connected cognitive/perceptual PEs (nodes 3, 4, 5, 6, and 7) seemed to reflect discrete Schneiderian-like beliefs/feelings/experiences. These nodes however were seemingly much more widely connected to the remaining PEs in the network than those within the disorganization PE cluster. Third a group of referential-delusion/paranoia PEs (nodes 1, 2, 10\* and 13) seemed to occupy the lower right quadrant of the network while lastly, PEs denoting social/interpersonal impairment/difficulty (nodes 9, 10\*, 11 and 12) occupied the top right quadrant. Notably node 10 ('often felt nervous when with other people...') seemed

to constitute a *bridging* node between these latter two clusters. It was noted that node 10 could conceivably be conceptually anchored to either cluster, in that it potentially captured both paranoia and social/interpersonal difficulties.

Somewhat independently, node 16 (hallucinatory item) seemed to straddle each of the four PE clusters. Strong connections were evident between node 16 and nodes denoting e.g. disorganized PEs (node 15), cognitive/perceptual PEs (nodes 5, 6, 7) and referential/paranoia PEs (nodes 2, 13). Subclinical hallucinatory experience therefore seemed to potentially influence and be influenced by many other experiences in the network. Furthermore, the centrality statistics from the current analysis suggested that specific PEs relating most notably to paranoia (specifically the feeling of being watched or stared at) appeared to be most central to the extended phenotype in this sample. Both of these findings seemed to be consistent with evidence from other studies regarding the role of individual PEs e.g. hallucinations have been shown to give rise to delusions,<sup>44, 45</sup> and paranoia has been shown to underpin other delusional experiences and hallucinations.<sup>46, 47</sup>

Overall, the general position and alignment of the PEs in the network seemed to suggest two potential *pathways* of influence beginning with (i) social and interpersonal difficulties, or conversely (ii) cognitive/perceptual experiences (see Figure 2). Each of these proposed *pathways* can be tentatively evidenced from the research literature. For example researchers have previously proposed separate cognitive and affective pathways for psychosis symptom expression<sup>62</sup> while others have noted specific gender differences in symptom aetiology; females for instance typically seem to have more of a *social etiology* whereas males seem to have more of a *cognitive etiology*.<sup>63</sup> Moreover, social deafferentation<sup>64</sup> and defeat<sup>65</sup> literatures might both explain the suggested pathway denoted by Panel-A where social and socialising difficulties create the necessary conditions for distorted perceptions and beliefs. Conversely hallucinatory and delusional experiences, specifically via paranoia and

persecutory beliefs, are known to compromise social perceptions, behaviour and relations.<sup>66-</sup>

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### *PE occurrence versus PE impairment*

A second aim of the study was to explore alternative formulations of the proposed extended phenotype based on PE impairment status. It was predicted that a PE network that was inclusive of non-distressing PEs would mirror a network where PEs reflected distressing experiences only.

The binary PMRF *occurrence* network structure was indeed similar to the *impairment* network structure, in that most edges within the occurrence network were also evident in the impairment network. These findings seemed to suggest that the pathways between individual PEs and the overall network structure underpinning the extended psychosis phenotype were *stable* irrespective of the level at which PEs were measured. Notably however, the *impairment* network displayed significantly stronger interconnectivity between many PEs i.e. edges between nodes were statistically stronger when PEs denoted distress/impairment only. According to van Borkulo et al.<sup>29</sup> more densely connected networks should feature stronger feedback among the symptoms modelled (in this case PEs) and may suggest a higher level of vulnerability. Given that the psychosis phenotype is likely to *evolve* from less severe levels to levels of greater severity, before disorder onset occurs, these networks seemed to reflect the underlying variation in PE severity within the general population. Notably, at more severe levels PEs seemed to reinforce one another more strongly. Several studies have suggested that variation in PE severity (i.e. distress) between individuals with and without a need for care can be explained by the presence/absence of paranoid beliefs.<sup>69-71</sup> Given (i) the centrality estimates for the paranoia items in the severity network (ii) the greater connectivity of Node 13 to other nodes in the impairment network and (iii) the edge thickness between

Node 2 and Node 16 in the impairment network, paranoia certainly seemed to play an important role within the present networks.

### *Limitations*

While the current analyses were successful in providing a cross-sectional map of a PE network and suggesting possible symptom pathways within this network, the study fell short of fully testing van Os and Linscott's hypotheses,<sup>15</sup> specifically regarding time. For example the current data did not afford an opportunity to (i) assess PEs prospectively (ii) assess individual PE duration or (iii) temporally order PE data to more accurately infer causal process.

Also, the current networks were based on positive PEs only. Evidence would suggest that subclinical negative symptoms may be as prevalent as subclinical positive symptoms in the general population.<sup>71, 72</sup> Moreover, subclinical negative symptoms have been found to be predictive of, and co-occur with, subclinical positive symptoms, and co-occurrence of subclinical positive and negative symptoms seem to predict later functional impairment and help-seeking behaviour.<sup>71, 73</sup> Depression and anxiety symptomology have also been shown to be important when modelling psychosis from a network perspective.<sup>39</sup> Incorporation of these other psychopathological/symptom experiences within future networks will be necessary to fully map and illustrate the interplay between PEs along the extended phenotype.

The data for the current study was also derived from a schizotypal personality measure. While this measure was a trait based assessment it still captured experiential accounts pertaining to both thoughts and perceptions. Moreover, use of a schizotypal personality scale as a proxy for experiential assessment is consistent with many other studies. For example, in a recent systematic review on definitions and assessments of psychotic-like experiences (PLEs), Lee et al<sup>74</sup> showed that a significant proportion of reviewed studies used



schizotypal personality measures to investigate PLEs. Furthermore studies have shown that measures of schizotypal personality provide non-clinical analogues of the heterogeneous symptomatology found in schizophrenia.<sup>75</sup> However, as Pedero et al.<sup>76</sup> point out, “while recent conceptualizations of the schizotypy framework indicate that it provides a unifying construct that efficiently links a broad continuum of clinical and subclinical psychosis manifestations (e.g., schizotypal traits, PLEs, attenuated psychotic symptoms, basic symptoms), as well as “normal” personality variation<sup>77</sup>,....schizotypal traits usually are stable in time (trait-like approach), whereas PLEs are unstable or a state in nature (symptom approach)”<sup>78</sup> [p.6 & 7]. This is an important distinction that must be acknowledged in the context of the current findings.

Finally, the authors are mindful of the subjective nature of network interpretation and accept that the networks produced in the current study are likely to evoke alternative/competing interpretations. Although it was not the focus of the current set of analyses, community detection techniques can facilitate the identification of statistical communities among items in networks.

### *Conclusions*

Individual experiences/symptoms in a psychosis context have been repeatedly evidenced to predict, impact or influence other experiences/symptoms. If we assume therefore that associations observed between components of psychological constructs such as psychosis (i.e. PEs/symptoms) are potentially *causal*,<sup>79</sup> then psychosis may best be construed as a causal system, embodied in a network of functionally interconnected symptoms/experiences.<sup>80, 81</sup> In the current findings the multiple connections of varying strength between specific PEs and others in the network seemed to offer a unique and valuable opportunity to visually

represent, and in turn speculate about, the *role/importance* of individual experiences in the context of the broader psychosis phenotype.

## References

1. Chapman LJ, Chapman JP. Scales for rating psychotic and psychotic-like experiences as continua. *Schizophr Bull.* 1980; 6: 477–489.
2. Claridge GS. Can a disease model of schizophrenia survive? In: Bentall RP ed. *Reconstructing Schizophrenia*. London, UK: Routledge; 1990:157–183.
3. van Os J, Hanssen M, Bijl RV, Ravelli A. Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophr Res.* 2000;45:11–20.
4. Murphy J, Shevlin M, Houston J, Adamson G. A population based analysis of subclinical psychosis and help-seeking behavior. *Schizophr Bull.* 2010; 38: 360–367.
5. Chapman LJ, Chapman JP, Kwapil TR, Eckblad M, Zinser MC. Putatively psychosis-prone subjects 10 years later. *J Abnorm Psychol.* 1994;103:171–183. 36.
6. Linscott RJ, van Os J. An updated and conservative systematic review and meta-analysis of epidemiological evidence on psychotic experiences in children and adults: on the pathway from proneness to persistence to dimensional expression across mental disorders. *Psychol Med.* 2013;43:1133–1149. 37.
7. Kaymaz N, Drukker M, Lieb R et al. Do subthreshold psychotic experiences predict clinical outcomes in unselected non-help-seeking population-based samples? A systematic review and meta-analysis, enriched with new results. *Psychol Med.* 2012;42:2239–53.
8. Zammit S, Kounali D, Cannon M et al. Psychotic experiences and psychotic disorders at age 18 in relation to psychotic experiences at age 12 in a longitudinal population-based cohort study. *Am J Psychiatry.* 2013;170:742–50.
9. van Os J, Linscott RJ, Myin-Germeys I et al. A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. *Psychol Med.* 2009;39:179–95.
10. David AS. Why we need more debate on whether psychotic symptoms lie on a continuum with normality. *Psychol Med.* 2010;40:1935–42.
11. Lawrie SM, Hall J, Owens DGC, Johnstone EC. The ‘continuum of psychosis’: scientifically unproven and clinically impractical. *Br J Psychiatry.* 2010;197:423–425.
12. Stranghellini G, Langer AI, Ambrosini A et al. Quality of hallucinatory experiences: differences between a clinical and a non-clinical sample. *World Psychiatry* 2012;11:110–3.

13. Kounali D, Zammit S, Wiles N et al. Common versus psychopathology-specific risk factors for psychotic experiences and depression during adolescence. *Psychol Med*. 2014;44:2557-66.
14. Peters E, Ward T, Jackson M, Morgan C, Charalambides M, McGuire P, ... Garety PA. Clinical, socio-demographic and psychological characteristics in individuals with persistent psychotic experiences with and without a "need for care". *World Psychiatry*. 2016;15: 41-52.
15. Van Os J, Linscott RJ. The Extended Psychosis Phenotype—Relationship With Schizophrenia and With Ultrahigh Risk Status for Psychosis. *Schizophr Bull*. 2012; 38: 227-230.
16. Bak M, Myin-Germeys I, Delespaul P, Vollebergh W, de Graaf R, & van Os J. Do different psychotic experiences differentially predict need for care in the general population? *Comprehensive Psychiatry*. 2005; 46: 192–199.
17. Brett CMC. Transformative crises. In I. Clarke (Ed.), *Psychosis and spirituality: Consolidating the new paradigm* (2nd ed., pp. 155–174). 2010; Chichester, UK: Wiley.
18. Heriot-Maitland C, Knight M, Peters E. A qualitative comparison of psychotic-like phenomena in clinical and non-clinical populations. *Brit J Clin Psych*. 2012; 51: 37–53.
19. Jenner JA, Rutten S, Beuckens J, Boonstra N, Sytema S. Positive and useful auditory vocal hallucinations: Prevalence, characteristics, attributions, and implications for treatment. *Acta Psychiatr Scand*. 2008; 118: 238–245.
20. Lovatt A, Mason O, Brett C, Peters E. Psychotic-like experiences, appraisals, and trauma. *J Nerv Ment Dis*. 2010; 198: 813–819.
21. Johns LC, Kompus K, Connell M, Humpston C, Lincoln TM, Longden E, ... Fernyhough C. Auditory verbal hallucinations in persons with and without a need for care. *Schizophr Bull*. 2014; 40: S255-S264.
22. Baumeister D, Sedgwick O, Howes O, Peters E. Auditory verbal hallucinations and continuum models of psychosis: A systematic review of the healthy voice-hearer literature. *Clin Psychol Rev*, 2017; 551: 125-141.
23. Brett CMC, Peters ER, McGuire PK. Which psychotic experiences are associated with a need for clinical care? *Euro Psychiatr*. 2015; 30: 648-654.
24. McNally RJ, Robinaugh DJ, Wu GW, Wang L, Deserno MK, Borsboom D. Mental Disorders as Causal Systems A Network Approach to Posttraumatic Stress Disorder. *Clin Psychol Sci*. 2015; 3:836–849.
25. Boschloo L, Van Borkulo CD, Rhemtulla M, Keyes KM, Borsboom D, Schoevers RA. The network structure of symptoms of the diagnostic and statistical manual of mental disorders. *PLoS One*. 2015; 10:e0137621.
26. Fried EI, Epskamp S, Nesse RM, Tuerlinckx F, Borsboom D. What are 'good' depression symptoms? Comparing the centrality of DSM and non-DSM symptoms of depression in a network analysis. *J Affect Disorders*. 2016; 189: 314-320.

27. Goekoop R, Goekoop JG. A network view on psychiatric disorders: network clusters of symptoms as elementary syndromes of psychopathology. *PloS One*. 2014; 9(11): e112734.
28. Isvoranu AM, Borsboom D, van Os J, Guloksuz S. A Network Approach to Environmental Impact in Psychotic Disorders: Brief Theoretical Framework. *Schizophr Bull*. 2016; 42:870–873.
29. van Borkulo CD, Boschloo L, Borsboom D, Penninx BWJH, Waldorp LJ, Schoevers RA. Association of Symptom Network Structure With the Course of Depression. *JAMA*. 2015; 72:1219–1226.
30. Costantini G, Richetin J, Borsboom D, Fried EI, Rhemtulla M, Perugini M. Development of indirect measures of conscientiousness: combining a facets approach and network analysis. *Eur J Personality*. 2015; 29:548–567.
31. Cramer AO, Sluis S, Noordhof A, Wichers M, Geschwind N, Aggen SH, . . . Borsboom D. Dimensions of normal personality as networks in search of equilibrium: You can't like parties if you don't like people. *Eur J Personality*. 2012; 26: 414-431.
32. Dalege J, Borsboom D, van Harreveld F, van den Berg H, Conner M, van der Maas HLJ. Toward a formalized account of attitudes: The Causal Attitude Network (CAN) model. *Psychol Rev*. 2016; 123:2–22.
33. Fried EI, van Borkulo CD, Cramer AOJ, Lynn B, Schoevers RA, Borsboom D. Mental disorders as networks of problems: a review of recent insights. *Soc Psych Psych Epid*. 2016; DOI 10.1007/s00127-016-1319-z
34. Borsboom D, Cramer AOJ. Network analysis: an integrative approach to the structure of psychopathology. *Annu Rev Clin Psychol*. 2013;9:91–121. 12.
35. Cramer AOJ, Waldorp LJ, van der Maas HLJ, Borsboom D. Comorbidity: a network perspective. *Behav Brain Sci*. 2010;33:137–150; discussion 150–193. 13.
36. Kendler KS, Zachar P, Craver C. What kinds of things are psychiatric disorders? *Psychol Med*. 2011;41:1143–1150.
37. van Rooijen G, Isvoranu AM, Meijer CJ, van Borkulo CD, Ruhé HG, & de Haan L. A symptom network structure of the psychosis spectrum. *Schizophr Res*. 2017; doi.org/10.1016/j.schres.2017.02.018
38. Wigman JT, de Vos S, Wichers M, van Os J, Bartels-Velthuis AA. A transdiagnostic network approach to psychosis. *Schizophr Bull*; 2017; 43: 122-132.
39. Isvoranu AM, van Borkulo, Boyette L, Wigman JTW, Vinkers CH, Borsboom D, GROUP Investigators. A Network Approach to Psychosis: Pathways between Childhood Trauma and Psychotic Symptoms. *Schiz Bull*. 2016; Advance Access published May 10, 2016.
40. Shevlin M, McElroy E, Bentall RP, Reininghaus U, Murphy J. The psychosis continuum: testing a bifactor model of psychosis in a general population sample. *Schizophr Bull*. 2016; 43: 133-141.

41. Murphy J, Shevlin M, Adamson G, Houston JE. (2010). Positive psychosis symptom structure in the general population: Assessing dimensional consistency and continuity from “pathology” to “normality”. *Psychosis*. 2010; 2: 199-209.
42. Shevlin M, Murphy J, Dorahy MJ, Adamson G. The distribution of positive psychosis-like symptoms in the population: a latent class analysis of the National Comorbidity Survey. *Schizophr Res*. 2007; 89: 101-109.
43. Fleming S, Shevlin M, Murphy J, Joseph S. Psychosis within dimensional and categorical models of mental illness. *Psychosis*. 2014; 6: 4-15.
44. Smeets F, Lataster T, Dominguez MD, et al. Evidence that onset of psychosis in the population reflects early hallucinatory experiences that through environmental risks and affective dysregulation become complicated by delusions. *Schizophr Bull*. 2012; 38: 531-542.
45. Krabbendam L, Myin-Germeys I, Hanssen M, et al. Hallucinatory experiences and onset of psychotic disorder: evidence that the risk is mediated by delusion formation. *Acta Psychiatr Scand*. 2004;110:264–272.
46. Simons CJ, Wichers M, Derom C, et al. Subtle gene-environment interactions driving paranoia in daily life. *Genes Brain Behav*. 2009;8:5–12.
47. Myin-Germeys I, Marcelis M, Krabbendam L, Delespaul P, van Os J. Subtle fluctuations in psychotic phenomena as functional states of abnormal dopamine reactivity in individuals at risk. *Biol Psychiatry*. 2005;58:105–110.
48. Grant BF, Dawson DA. Introduction to the national epidemiologic survey on alcohol and related conditions. *Alcohol Health Res World*. 2006;29:74. 50.
49. Grant B, Kaplan K. Source and Accuracy Statement for the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism; 2005. 51.
50. Grant B, Kaplan K, Shepard J, Moore T. Source and Accuracy Statement for Wave 1 of the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions. Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism; 2003. 52.
51. Grant BF, Dawson DA, Stinson FS, Chou PS, Kay W, Pickering R. The Alcohol Use Disorder and Associated Disabilities Interview Schedule-IV (AUDADIS-IV): reliability of alcohol consumption, tobacco use, family history of depression and psychiatric diagnostic modules in a general population sample. *Drug Alcohol Depend*. 2003;71:7–16.
52. Grant BF, Dawson D. The Alcohol Use Disorder and Associated Disabilities Interview Schedule-IV (AUDADIS-IV). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism; 2000. 54.
53. Grant BF, Harford TC, Dawson DA, Chou PS, Pickering RP. The Alcohol Use Disorder and Associated Disabilities Interview schedule (AUDADIS): reliability of alcohol and drug modules in a general population sample. *Drug Alcohol Depend*. 1995;39:37–44.

54. Epskamp S, Borsboom D, Fried EI. (2016). Estimating psychological networks and their accuracy: a tutorial paper. *ArXiv Preprint*, 501, 1-25.
55. Costantini G, Epskamp S, Borsboom D, Perugini M, Mttus R, Waldorp LJ, Cramer AO. (2015). State of the aRt personality research: A tutorial on network analysis of personality data in R. *J Res Personality*, 54, 13-29.
56. Van Borkulo CD, Borsboom D, Epskamp S, Blanken TF, Boschloo L, Schoevers RA, Waldorp LJ. A new method for constructing networks from binary data. *Sci Reports*. 2014; 4: 1–10.
57. Borsboom D, Cramer AO. Network analysis: an integrative approach to the structure of psychopathology. *Annu Rev Clin Psychol*. 2013; 9: 91-121.
58. McNally RJ. Can network analysis transform psychopathology? *Behav Res Ther*. 2016; 86: 95-104.
59. Opsahl T, Agneessens F, Skvoretz J. Node centrality in weighted networks: Generalizing degree and shortest paths. *Soc Networks*. 2010; 32: 245-251.
60. Epskamp S, Cramer AO, Waldorp LJ, Schmittmann VD, Borsboom D. qgraph: Network visualizations of relationships in psychometric data. *J Stat Software*. 2012; 48: 1-18.
61. Fruchterman TM, Reingold EM. Graph drawing by force-directed placement. *Software Pract Exper*. 1991; 21: 1129-1164.
62. Garety PA, Kuipers E, Fowler D, Freeman D, Bebbington PE. A cognitive model of the positive symptoms of psychosis. *Psychol Med*. 2001; 31: 189-195.
63. Myin-Germeys I, van Os J. Stress-reactivity in psychosis: evidence for an affective pathway to psychosis. *Clinical Psychol Rev*. 2007; 27: 409-424.
64. Hoffman RE. A social deafferentation hypothesis for induction of active schizophrenia. *Schizophren Bull*. 2007; 33: 1066-1070.
65. Selten JP, van der Ven E, Rutten BP, Cantor-Graae E. The social defeat hypothesis of schizophrenia: an update. *Schizophren Bull*. 2013; 39: 1180-1186.
66. Combs DR, Penn DL. The role of subclinical paranoia on social perception and behavior. *Schizophren Res*. 2004; 69: 93-104.
67. Combs DR, Michael CO, Penn DL. Paranoia and emotion perception across the continuum. *Brit J Clin Psychol*. 2006; 45: 19-31.
68. Combs DR, Finn JA, Wohlfahrt W, Penn DL, Basso MR. Social cognition and social functioning in nonclinical paranoia. *Cognitive Neuropsychiat*. 2013; 18: 531-548.
69. Brett CMC, Peters EP, Johns LC, Tabraham P, Valmaggia LR, McGuire PK. Appraisals of Anomalous Experiences Interview (AANEX): a multidimensional measure of psychological responses to anomalies associated with psychosis. *Brit J Psychiat*. 2007; 191: s23-s30.

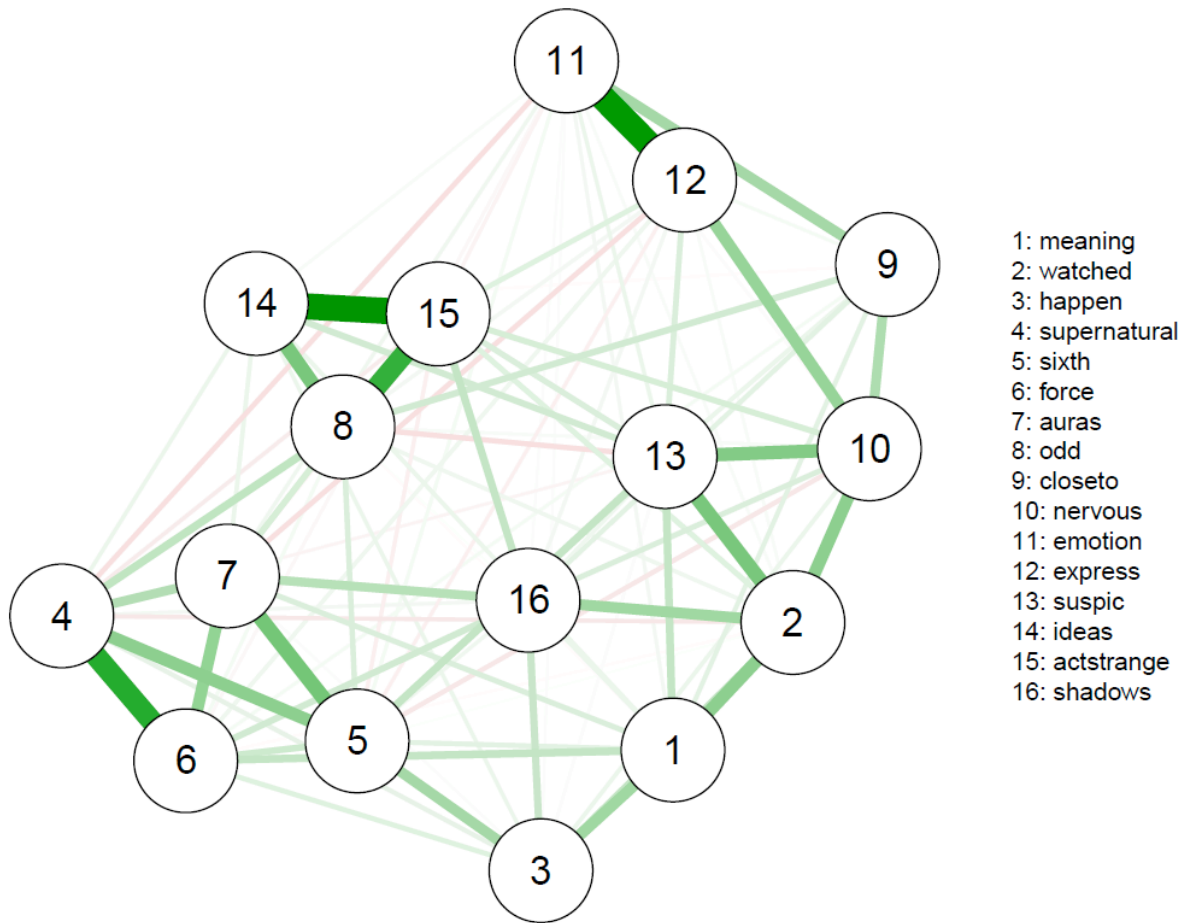
70. Ward TA, Gaynor KJ, Hunter MD, Woodruff PW, Garety PA, Peters ER. Appraisals and responses to experimental symptom analogues in clinical and nonclinical individuals with psychotic experiences. *Schizophren Bull.* 2014; 40: 845-855
71. Dominguez MD, Saka MC, Lieb R et al. Early expression of negative/disorganized symptoms predicting psychotic experiences and subsequent clinical psychosis: a 10-year study. *Am J Psychiatry.* 2010;167:1075-82.
72. Werbeloff N, Dohrenwend BP, Yoffe R et al. The association between negative symptoms, psychotic experiences and later schizophrenia: a population-based longitudinal study. *PLoS One* 2015;10:e0119852.
73. van Os J, Reininghaus U. Psychosis as a transdiagnostic and extended phenotype in the general population. *World Psychiatry.* 2016; 15: 118-124.
74. Lee, K. W., Chan, K. W., Chang, W. C., Lee, E. H. M., Hui, C. L. M., & Chen, E. Y. H. (2016). A systematic review on definitions and assessments of psychotic-like experiences. *Early intervention in psychiatry*, 10(1), 3-16.
75. Cochrane, M., Petch, I., & Pickering, A. D. (2010). Do measures of schizotypal personality provide non-clinical analogues of schizophrenic symptomatology?. *Psychiat Res*, 176(2), 150-154.
76. Pedrero, E. F., & Debbané, M. (2017). Schizotypal traits and psychotic-like experiences during adolescence: An update. *Psicothema*, 29(1), 5-17.
77. Kwapil, T. R., & Barrantes-Vidal, N. (2015). Schizotypy: Looking back and moving forward. *Schizophren Bull*, 41, S366-373.
78. Debbané, M., & Barrantes-Vidal, N. (2015). Schizotypy from a developmental perspective. *Schizophren Bull*, 41, S386-395.
79. Cramer AO, Sluis S, Noordhof A, Wichers M, Geschwind N, Aggen SH, . . . Borsboom D. Dimensions of normal personality as networks in search of equilibrium: You can't like parties if you don't like people. *Eur J Personality.* 2012; 26: 414-431.
80. Eaton NR. Latent variable and network models of comorbidity: toward an empirically derived nosology. *Soc Psych Psych Epid.* 2015;50: 845-849.
81. Fried EI, Nesse RM. The impact of individual depressive symptoms on impairment of psychosocial functioning. *PloS One.* 2014; 9: e90311.

**Table 1.** Node names, labels and PE response frequencies

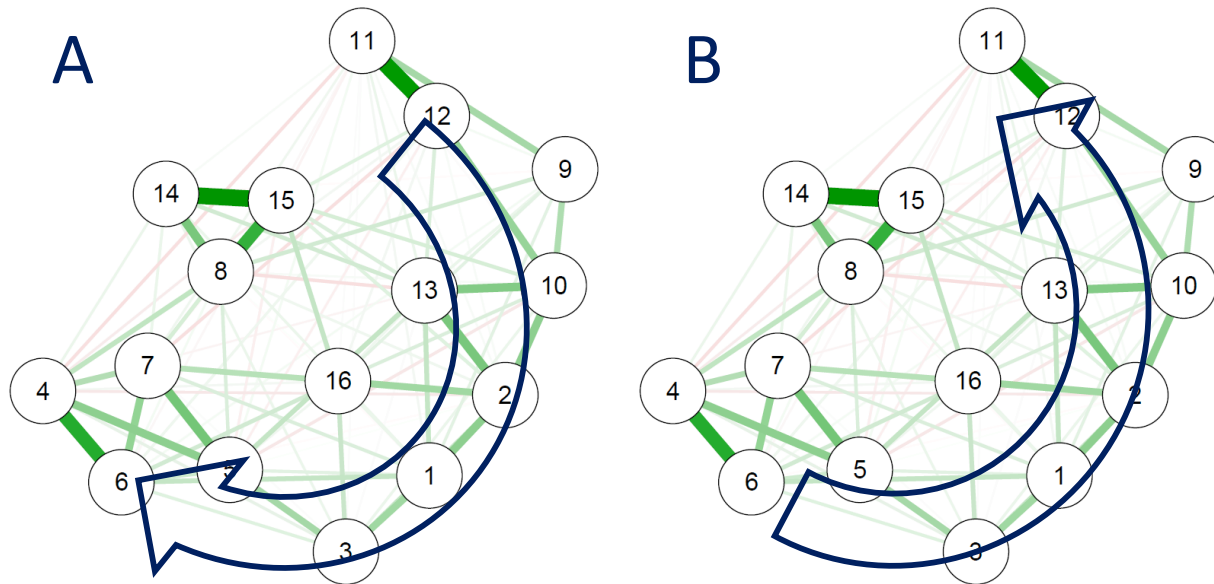
Node	Node label	N (%)			
		No	Yes	Impair	Miss
1 Have you often had the feeling that things that have no special meaning to most people are really meant to give you a message?	meaning	30853 (89.5)	2951 (8.6)	397 (1.2)	270 (0.8)
2 Have you often had the feeling of being watched or stared at, when around people?	watched	31098 (90.2)	2648 (7.7)	682 (2.0)	43 (0.1)
3 Have you ever felt that you could make things happen just by making a wish or thinking?	happen	31956 (92.7)	2296 (6.7)	166 (0.5)	53 (0.2)
4 Have you had personal experiences with the supernatural?	supernatural	31275 (90.7)	2888 (8.4)	210 (0.6)	98 (0.3)
5 Have you believed that you have a “sixth sense” that allows you to know and predict things that others can’t?	sixth	31213 (90.5)	2970 (8.6)	222 (0.6)	66 (0.2)
6 Have you had the sense that some force is around you, even though you cannot see anyone?	force	27923 (81.0)	6186 (17.9)	268 (0.8)	94 (0.3)
7 Have you often seen auras or energy fields around people?	auras	33453 (97.0)	895 (2.6)	68 (0.2)	55 (0.2)
8 Have people thought you are odd, eccentric or strange?	odd	30591 (88.7)	3220 (9.3)	438 (1.3)	222 (0.6)
9 Have there been very few people that you’re really close to outside of your immediate family?	close to	23271 (67.5)	10638 (30.9)	492 (1.4)	70 (0.2)
10 Often you felt nervous when with other people even whom you have known for a while?	nervous	32190 (93.4)	1762 (5.1)	491 (1.4)	28 (0.1)
11 Have you rarely shown emotion?	emotion	28646 (83.1)	4971 (14.4)	749 (2.2)	105 (0.3)
12 Have you had trouble expressing your emotions and feelings?	express	29720 (86.2)	2932 (8.5)	1762 (5.1)	57 (0.2)
13 Have felt suspicious of people, even if you have known them for a while?	suspicious	30000 (87.0)	3379 (9.8)	1033 (3.0)	59 (0.2)
14 Have people thought you have strange ideas?	ideas	29897 (86.7)	3819 (11.1)	524 (1.5)	231 (0.7)
15 Have people thought you act strangely?	act strange	31457 (91.3)	2355 (6.8)	455 (1.3)	204 (0.6)
16 Have you often thought that objects or shadows are really people or animals, or that noises are actually people’s voices?	shadows	33802 (98.1)	484 (1.4)	124 (0.4)	61 (0.2)



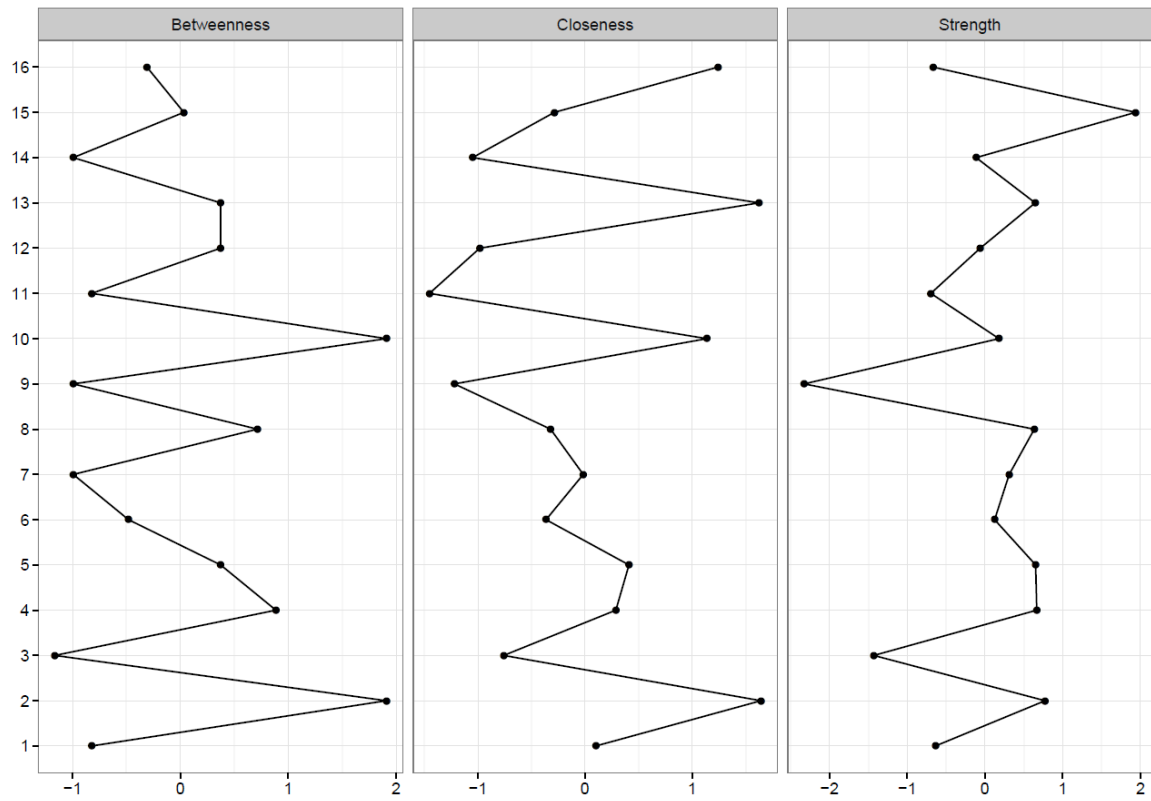
**Figure 1.** Estimated network structure of 16 PEs. The network structure is a Gaussian graphical model, which is a network of partial correlation coefficients.



**Figure 2.** Potential PE causal pathways



**Figure 3.** Centrality indices for the Gaussian graphical model (bottom panel). Centrality indices are shown as standardized z-scores.



**Figure 4.** Panel A: Occurrence network (PE with/without distress); Panel B: Impairment network (Distressing PEs only)

