


Methodology and Research Practice

Evidence for the Network Theory of Mental Disorders in People at Ultra High Risk of and Diagnosed With Schizophrenia

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Keywords: symptom networks, latent variable model, structural equation model, Bayesian networks, schizophrenia

<https://doi.org/10.1525/collabra.158304>

Collabra: Psychology

Vol. 12, Issue 1, 2026

The network theory of mental disorders proposes that symptoms cause the expression of other symptoms. Research on the network theory is increasing, but empirical support is lacking. We aim to assess the viability of an integrated latent variable model and network model of psychopathology. We sourced 795 ultra-high-risk participants from the North American Prodromal Longitudinal Three Study and 1,446 participants with schizophrenia from the Clinical Antipsychotic Trials of Intervention Effectiveness study. We reconstructed a Bayesian network on the Scale of Psychosis Risk Symptoms and Positive and Negative Syndrome Scale on five training samples and then estimated the parameters on five test samples from each study, respectively. We compared the three models (Network model, latent variable model, and integrated model) on the five test samples from each assessment (30 models). The integrated model had a significantly superior fit than the LVM and had a better fit than the network model in all test samples. This novel finding provides partial support that items may interact and that networks with latent variables may be used to model the structure of an assessment if there is a poor fit to the latent variable model structure.

Introduction

The network theory of mental disorders is a recent paradigm that attempts to model an underlying psychological disorder or trait based on the complex system of interactions among its symptoms Borsboom (2017). This theory posits that each symptom can jointly influence, cause, or exacerbate the expression of other symptoms. The network theory proposed by Borsboom (2008) and expanded by Borsboom (2017), Bringmann et al. (2022), and Cramer et al. (2010) has been the subject of considerable research and debate (Abplanalp & Green, 2022; Guyon et al., 2017; Jones et al., 2017; Oude Maatman, 2020) and has been adopted in progressively more publications since its inception. One principle proposed under this theory is the complexity principle, which identifies that mental disorders are best characterized in terms of the interaction between different components in a psychopathology network (Borsboom, 2017, p.7). The interaction between components identifies that: Symptoms or observed behavioural variables are associated with the expression of other symptoms or observed behavioral variables in a network. Borsboom (2017) considered that systems of interacting components

may be the most plausible explanation for the phenomena of mental disorders. Symptom networks as a theory provide a fresh perspective on modeling mental disorders, and the method may have strong explanatory value, like other modeling techniques, such as the latent variable model.

Over many decades, scientists have developed network statistical methods to represent complex systems of interacting variables. Network methods have been recently applied to explain the characteristics of psychological disorders, including schizophrenia (Borsboom, 2008; Contreras et al., 2019; Isvoranu et al., 2017), depression (Fried et al., 2016; van Borkulo et al., 2015), and post-traumatic stress disorder (Benfer et al., 2018; Fried et al., 2018), among others. The body of research on networks and mental disorders in recent years has marked the beginning of a paradigm in the field of mental health: Symptom networks.

The network approach to psychological disorders takes a complex systems approach to study symptoms, latent traits, observed behavior, and historical factors of an individual. These variables are the *nodes* in the network, and relationships between variables are encoded in the network's edges. The network model can be directed, with arrows pointing from the parent node (predictor) to the child node

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(response), partially directed or undirected. Bayesian networks were developed in the 1970s to graphically represent and probabilistically model the components of a complex system for top-down and bottom-up perceptual processes (Pearl, 2011). Bayesian networks are one statistical method that graphically represents the relationships between variables in a network and encodes conditional probability relationships into a directed acyclic graph. We refer to 'network model' to describe a Bayesian network for the remainder of this work.

Networks may have different uses than those of other models of psychopathology. Child nodes are dependent on parent nodes in directed symptom networks. Therefore, relationships between variables may be used to identify nodes to target for clinical interventions (Briganti et al., 2022). They can also identify associations between symptoms and node centrality statistics, as a measure of symptom importance (Fried et al., 2017). Some researchers posit that developing an intervention based on the properties of a single node or edge is not feasible, as most psychosocial therapeutic interventions often target an array of variables (Bringmann et al., 2019). However, theoretical implications may be obtained from a reconstructed network of symptoms of one or more conditions. For example, symptoms, such as those in the Diagnostic and Statistical Manual of Mental Disorders fifth edition (DSM-5; American Psychiatric Association, 2013), can be represented in terms of a network, and clusters of symptoms could represent conditions (Nuijten et al., 2016).

Another paradigm of psychopathology, the latent variable model (LVM), proposes that observed behaviors, symptoms, or test scores are caused by underlying latent variables (hidden factors) that exist on a continuum of intensity or severity. Latent variables are inferred to cause the observed responses to items of a psychometric test with a degree of measurement error. LVMs assume that items of a test have local independence, given the relationship with the latent variable (Borsboom et al., 2003a). The local independence assumption states that a latent variable underlies a set of observed variables, and conditioning on the latent variables renders the observed variables independent (Borsboom et al., 2003a). Here, all observed variables should be independent, as in principal components analysis (Borsboom et al., 2003a). This assumption has been at the forefront of psychometric theory and can be tested using networks and SEMs.

A structural equation model (SEM) is often used as the statistical method to test LVMs using confirmatory factor analysis. A particular type of SEM, a structural causal model or Bayesian network, may be a valuable method for symptom networks as they encode directed relationships between symptoms and have functionality similar to other SEM methods. Therefore, a data-driven method like Bayesian networks may identify a locally optimal set of directed relationships between symptoms to test in an SEM. These directed relationships can help identify predictor and response relationships in an SEM. Furthermore, the structure of the SEM is often hypothesized a priori, based on evidence from previous literature. However, given the re-

search on methods in Bayesian networks in the last 30 years, methods have been developed to learn the networks using data-driven approaches (Scutari & Denis, 2021a).

Bayesian networks are not the only method to test using SEMs. Other methods, such as exploratory factor analysis, are data-driven and have been used to build a structure to test in an SEM (Liew, Hartvigsen, et al., 2023; Marcot & Penman, 2019; Pearl, 2009). In contrast to a Bayesian network, which encodes conditional probability relationships between random variables in a directed acyclic graph, an exploratory factor analysis is a dimension reduction technique to identify latent (hidden) variables from a set of observed variables (Kline, 2023). An exploratory factor analysis structure may be tested in later studies using confirmatory factor analysis, but not on the same data (Kline, 2023). With factor analysis, there may be departures from the local independence assumption. Generally, symptom networks encode dependencies between observed variables and exploratory factor analysis attempts to find latent variables that force items to be locally or conditionally independent, given the latent variables (Borsboom, 2017; Borsboom et al., 2003b).

The complexity principle of the network theory, according to Borsboom (2017), may be assessed empirically by comparing an LVM to a network model with latent variables using items of an assessment as nodes in the network. If the latent variable model does not fulfil the local independence assumption, associations or dependencies between items may be included in the model in addition to latent variables. This would be an integration of the two approaches, similar to that proposed by Epskamp et al. (2017), except that we use a structural causal model using a Bayesian network and an LVM in place of latent network modelling or generalization residual network modeling. Although most research on symptom networks employs Gaussian graphical models (Borsboom & Cramer, 2013; Epskamp et al., 2017), Bayesian networks can be used as a model to test within a structural causal model. Additionally, within a structural causal model, an LVM can be modelled with and without directed item associations to test the complexity principle in the network theory of psychological disorders (Borsboom, 2017).

Structural causal models that include associations between items and latent variables have been used in other disciplines and is an established method (Kline, 2023), but we are not aware of any research that uses this technique in symptom networks to test the complexity principle in the network theory of psychological disorders (Borsboom, 2017). Structural causal models of latent variables, with and without associations between observed variables, are nested and can be tested using a likelihood ratio test. Structural causal models and likelihood ratio tests are key advantages of using Bayesian networks to test the complexity principle of the network theory of psychological disorders. This integration may have several benefits as network methods generally do not include latent variables and a latent variable model violates the local independence assumption (Epskamp et al., 2017). Here we take the associations between latent variables and items, and the directed

associations between items, and include these in the model in lavaan. We postulate that network methods are adequate to generate an interpretable model with a good fit to the data when combined with an a priori LVM based on the literature.

Given the increasing literature on symptom networks and the arguments made by symptom network theorists, efforts have been made to compare the model fit of LVM and network models in the study of psychometric data (Kan et al., 2020). Kan et al. (2020) proposed a method to directly compare partial correlation networks to the structures hypothesized in LVMs. In addition to Kan et al. (2020) and McFarland (2020) researchers have compared the fit statistics of network models and LVM (Christensen et al., 2019; Kan et al., 2020; Moriana et al., 2022; Moriconi et al., 2025; Schmank et al., 2019). Arguably, fit statistics, while informative as to which model fits the data best, do not lead to a rejection of models and are not hypothesis tests of model fit (Barrett, 2007). Fit statistics are not significance tests because as there is no inherent binary decision to retain or reject a null hypothesis, and there is no generally demarcation of the limits of sampling error (Kline, 2023). A likelihood ratio test, however, can be used to empirically test nested models for whether a theoretical proposition is correct, such as the complexity principle proposed by Borsboom (2017). An integrated network model with latent variables, and a latent variable model without associations between items, then the difference in the likelihood of both models can be assessed using a likelihood ratio test, because they are nested (Kline, 2023). Model fit statistics can also be estimated for nested models to make comparisons on model fit. Research needs to be undertaken to examine whether the model should include dependency between items of an assessment, as network theory proposes. Although Kan et al. (2020) and McFarland (2020), aim to provide evidence in favor of network models, likelihood ratio tests are more suited to test this hypothesis formally. Hence, we can directly compare whether the proposed LVM has a poorer fit than an integration between the two models using a hypothesis test. While networks and SEMs are used together in other disciplines, including educational assessment (Almond et al., 2007), transportation (Díez-Mesa et al., 2018), and ecology and climate change (Li et al., 2018), we are not aware of any research with people diagnosed with schizophrenia (PDS) that has subjected the complexity principle of the network theory of mental disorders to a hypothesis test, using SEM and likelihood ratio tests (Buchwald et al., 2024). The network model with latent variables represents an integration of both the network and latent variable models. To the best of the authors' knowledge, there has been no empirical comparison to date between a network model, an LVM model, and an integration of these two models in PDS (Buchwald et al., 2024). We therefore aim to assess the validity of the complexity principle proposed by Borsboom (2017) by identifying whether associations between observed variables, as identified in a symptom network, should be included within an LVM. We aim to provide an application of an integrated model as theorised by Epskamp et al. (2017). If we reject an LVM without

associations between variables and accept an LVM with associations between variables, this will provide partial support for the complexity principle (Borsboom, 2017). Additionally, we hypothesize that a network model with latent variables has a better fit than a network model without latent variables.

Method

Participant Characteristics

The first dataset considered is from the North American Prodromal Longitudinal Study (NAPLS3) study conducted by the University of California San Francisco (Addington et al., 2022), where we obtained the data from the National Institute of Health data repository (National Institute of Mental Health, 2024a). The NAPLS3 study (Addington et al., 2022) is a consortium of nine programs that follow people ultra high risk of schizophrenia over a two-year period and assesses predictors of and transition to schizophrenia. This dataset was retrieved from the National Institute of Mental Health (2024b). The sample included participants from health care providers, educators, social services, and self-referrals. In addition to the exclusion criteria documented in Addington et al. (2022), participants who did not complete all items on the Scale of Psychosis Risk Symptoms (SOPS) were excluded. A total of 795 participants fully completed the SOPS, and when participants had undertaken repeated measures on the SOPS during the longitudinal study, we selected the latest observation. Table 1 provides the descriptive statistics for people who participated in the NAPLS3 study. Most participants were Male (427; 53.7%) and identified as White (European American; 479; 60.3%). Other ethnic groups in this study include participants who identify as Asian (99; 12.5%), Black (African American; 95; 12.0%), or American Indian and Alaska Native (17, 2.1%). This study contained a cohort with a mean age of less than 20 years ($M = 18.7$; $SD = 4.1$). Only a few individuals had a diagnosis of a psychotic disorder at the end of the study (64; 8.1%).

Table 2 presents the descriptive statistics of people who participated in the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) study between 2001 and 2004 and were included in the present study. People aged 18 to 65, who met the DSM-IV criteria for schizophrenia, were amenable to oral medication, and had adequate decision-making capacity were included in the CATIE study if they had chronic or recurrent schizophrenia (Stroup et al., 2003). First episode and wholly treatment-refractory patients were excluded in the original CATIE study, and people diagnosed with schizoaffective disorder, intellectual disability, pervasive developmental disorder, delirium, dementia, amnesia, or other cognitive disorders were excluded (Stroup et al., 2003). Other exclusion criteria are reported in Stroup et al. (2003). Our only additional selection criterion is that the participants must have completed the PANSS assessment at baseline ($N = 1,446$). Multiple assessments of the PANSS were collected during the trial, and we used the pretreatment assessment for this study. In terms of the participants from the CATIE study, the large majority

Table 1. Demographics for the NAPLS3 study

Demographic	N (M)	% (SD)	Missing
Age	(18.7)	(4.1)	
Sex			
Male	427	53.7	
Female	368	46.3	
Ethnicity			1
White or European American	479	60.3	
Asian	99	12.5	
Black or African American	95	12.0	
American Indian/Alaska Native	17	2.1	
Other	104	13.1	
Immigration status			4
Born in USA or Canada (not 1st or 2nd generation)	602	76.1	
2nd generation migrant (born in USA or Canada)	99	12.5	
Subject and parents born abroad	90	11.4	
Diagnosis (SCID-5, latest assessment)			
Schizophrenia	30	3.8	
Schizoaffective disorder	12	1.5	
Schizophreniform	4	0.5	
Other psychotic disorder	18	2.3	

Note: N = Sample size; M = Mean; SD = Standard deviation; SCID-5 = Structured clinical interview for the DSM-5; SOPS = Scale for the assessment of prodromal symptoms; SOFA = Social and Occupational Functional Assessment Scale of the DSM-5

Table 2. Demographics for the CATIE study

Demographic	N (M)	% (SD)	Missing
Age	(40.5)	(11.1)	2
Sex			2
Male	1070	74.1	
Female	374	25.9	
Ethnicity			4
White or European American	869	60.3	
Black or African American	503	34.9	
Asian	33	2.3	
American Indian/Alaska Native	8	0.6	
Other	29	2.0	
Diagnosis (SCID-5, Past five years)			
Schizophrenia	1,413	97.7	0

Note: N = Sample size; M = Mean; SD = Standard deviation; Proportions do not include missing values; SCID-5 = Structured clinical interview for the DSM-5

of the participants from the CATIE study sample were Males (1070; 74.1%) who were aged on average 40.5 years old, and identified as White or European American (869; 60.3%). Fewer compared to Black or African American (503; 34.9%) or Asian (33; 2.3%) people participated. Some individuals reported multiple ethnicities or other minority ethnic groups, and these were categorised, along with other minority ethnic groups, as other. Most (1,413; 97.7%) participants had received their diagnosis of schizophrenia within the past five years.

Measures and Covariates

The SOPS (Miller et al., 2002) is a 19-item assessment used to quantify severe schizophrenia-like symptoms and to identify if a person has brief intermittent psychotic syndrome, attenuated positive symptom syndrome, and genetic risk and functional decline psychosis risk syndrome. The SOPS is a clinician-rated assessment which is part of a broader structured diagnostic interview called the Structured Interview for Psychosis-Risk Syndromes (Miller et al., 1999). The SOPS has four subscales: Positive, negative, dis-

organized, and general (Miller et al., 1999). The items are ranked on a scale from zero to six, and a score of six on some items of the SOPS means the participant met one criterion for current psychosis. Miller et al. (2002) found interrater reliability for diagnoses derived from the SOPS was excellent ($Kappa=.81$). Woods et al. (2019a) reviewed the SOPS and reported excellent interrater reliability estimates across studies and strong predictive, convergent, and discriminant validity across studies.

The PANSS (Kay et al., 1987) was developed to assess the severity of positive and negative symptoms of schizophrenia and their contribution to global psychopathology. The PANSS has three subscales: positive, negative, and general (Kay et al., 1987). Kay et al. (1987) reported the PANSS has acceptable to good reliability across the positive and negative subscales of the PANSS positively correlated to the Brief Psychiatric Rating Scale and Manifest Affect Rating Scale subscales. Since then, it has been the subject of many systematic reviews and meta-analyses that identify its utility for use with PDS (Geck et al., 2025; Lim et al., 2021; Obermeier et al., 2011; Shafer & Dazzi, 2019). However, there has been some concern about the factor structure of the PANSS (Geck et al., 2025). For negative symptoms in particular, there are newer dedicated tools to assess negative symptoms that may be preferred over the PANSS (Kumari et al., 2017; Weigel et al., 2023). Research has also identified differential responses on the PANSS across cultures, which may be due to differing interpretations (Khan et al., 2013).

Data Diagnosis

The SOPS and PANSS items were left unstandardized. We assumed that the responses to the SOPS and PANSS were continuous to implement a maximum likelihood SEM in the R package lavaan (Rosseel, 2012) and because there were many seemingly equidistant levels in the SOPS and PANSS items. To account for any non-normality of the error terms, we used robust standard errors using the “MLR” option in the R package lavaan (version 0.6-16; Rosseel, 2012).

Analysis

Analysis was completed in R version 4.3.2 (R Core Team, 2024). Given a heuristic based on a sample size to parameter ratio of 20 to 1 (Kline, 2023), the subscales *general* and *disorganized* from the SOPS in the analysis of the NAPLS3 study were excluded to simplify the model further. The general subscale from the PANSS was also not included. We attempted to run the analysis with all dimensions of the SOPS and PANSS; however, the model was over-parameterised for the sample size, and a solution could not be found. One sample size heuristic is to have 20 participants per parameter (Kline, 2023), and this would have been exceeded when including all subscales of the PANSS and SOPS. Our proposed latent variable structure aligns with the assessment manuals of these assessments for positive and negative symptom dimensions (Kay et al., 1987; Miller et al., 2002). An illustration of the various models used in this study is provided in Figure 1.

We used Bayesian networks in place of Gaussian graphical models so the model can be included in a structural causal model to test the local independence assumption, and the complexity principle of the network theory of psychological disorders, by comparing a network model, a LVM, and a network model with latent variables (integrated model), using likelihood ratio tests. A significant difference between an LVM and an integrated model would indicate that the model needs to include directed associations between items, as the network theory of psychological disorders prescribes, and therefore provide partial support of the network theory of psychological disorders.

The latent variable model in Figure 1 model 1, is estimated using confirmatory factor analysis on the structure of the PANSS and SOPS, utilising items from the subscales and a latent variable that represents the subscales. For example, using the PANSS, a latent variable named positive was a parent of the five items measuring positive symptoms, and a latent variable named negative was a parent of the five items measuring negative symptoms. Items here represent the manifest variables or observed variables. The confirmatory factor analysis was estimated using the *cfa* function with maximum likelihood with Huber-White robust standard error estimators in lavaan (Rosseel, 2012). A covariance term was added for the latent variables, as this fit the data better. The key assumptions in this model is that the items have local independence, that changes in the latent variable causes changes in the items, that the items have a rationale or conceptual unity, that the levels rise or fall according to the underlying latent variable, are internally consistent, can be substituted with one another without changing the concept, and contribute only what they share to the approximation of the concept (Kline, 2023).

The Bayesian network in Figure 1, Model 2, represents conditional probability relationships between observed variables in a directed acyclic graph. The Bayesian network was first reconstructed in R using the package bnlearn, using the hill climbing algorithms and BIC as the score criterion (Scutari, 2009). In the hill-climbing algorithms, we started with an empty network, and then edges were either added, deleted, or the direction of the edge was reversed until no improvement in the score could be made. A Bayesian network has several assumptions, including: That all edges are directed and acyclical, that each node is independent of its non-descendants given its parents, that the data is iid, and that latent variables are explicitly modelled when they affect the network (Scutari & Denis, 2021a).

The integrated model in Figure 1, Model 3, is a combination of a confirmatory factor analysis model and a Bayesian network model. This model has been proposed in Epskamp et al. (2017). Epskamp et al. (2017) identifies that the assumptions of an integrated model carry over the assumptions of a network model and LVM except that there is no longer the assumption of local independence, and no longer the assumption that the covariance structure of items in the network is not due to latent variables. In latent variable models, it is assumed that latent variables cause observed variables; however, with the introduction of associations between observed variables, both latent variables

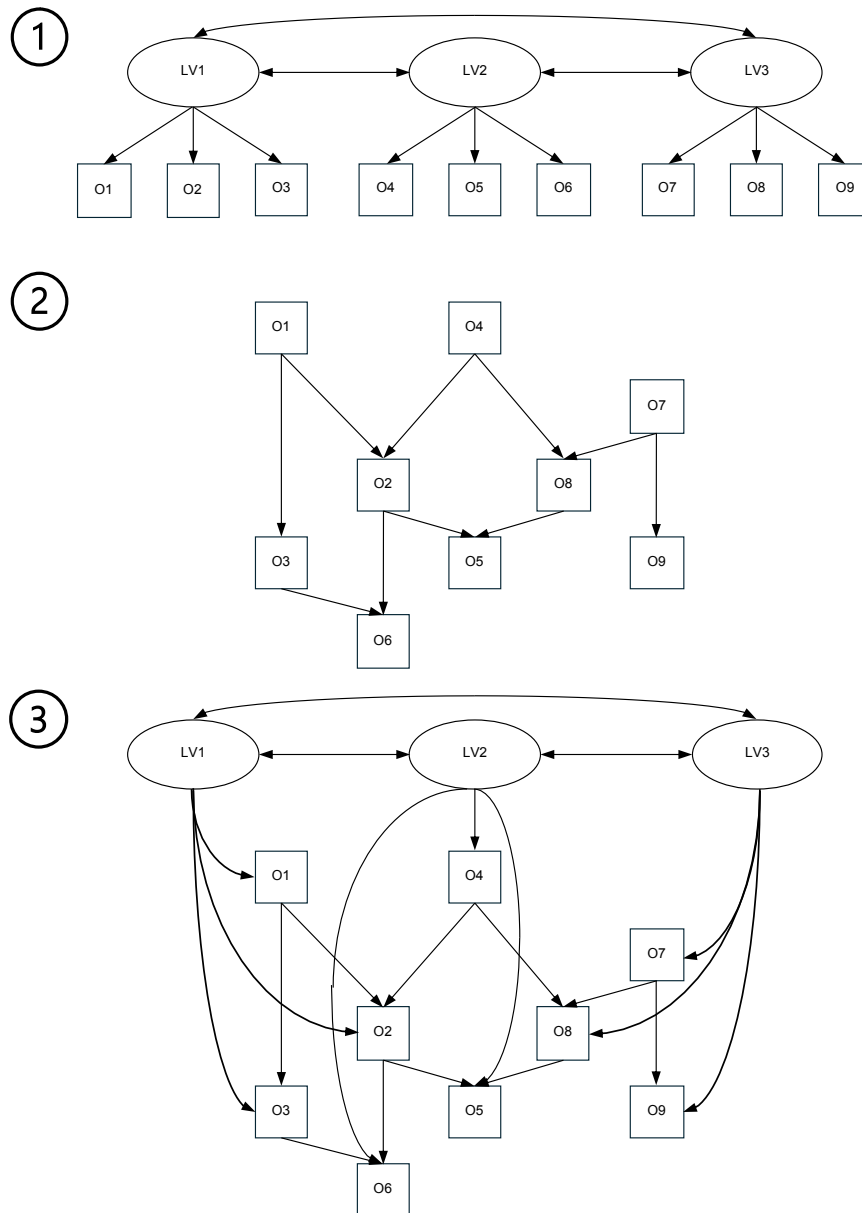


Figure 1. Illustration of Latent Variable Model, Network Model, and Integrated Model

Note. 1 = A latent variable model; 2 = A directed network model; 3 = An integrated model; LV = Latent variables; O = Observed variables; Single-headed arrows represent a directed relationship; Double-headed arrows represent undirected covariance; Arrows representing measurement error are omitted from this chart.

or observed variables may cause responses to other observed variables (Borsboom et al., 2003b; Epskamp et al., 2017). Latent variables may also be essential in Bayesian networks to assume causality (Scutari & Denis, 2021a). An integrated model may offer more flexibility than an LVM, which can be explicitly compared using likelihood ratio tests. We evaluated this multiple times using various data samples.

We used a similar concept to cross-validation to avoid unfairly comparing the model generated on the data (network model) to a model not generated on the data (LVM). We used a random training sample of size 100 for the NAPLS3 study and one of size 180 for the CATIE study (approximately 12.5%) to generate the network model struc-

ture. This sample size was chosen to ensure the structural equation model have a sufficient sample size to fit the required number of parameters. We then implemented this structure in an SEM on the remaining data to estimate the parameters. This split represents one-eighth of the data and allows for the parameters to be estimated in the SEM when using the test sample. We use a small sample to estimate the Bayesian network and a large sample to fit the three models in lavaan using an SEM: An integrated model, LVM, and network model. Portioning the data allows for the test sample not to contain the data on which the network was generated, to reduce the risk of overfitting. This does mean the results can vary in the test sample versus the training sample, so we repeated this five times

to ensure generalisability. According to Kuhn and Johnson (2013) five-fold or ten-fold cross-validation is a common choice, but there is no formal rule, although this is not cross-validation in the usual sense, as we are not predicting values in the test dataset. We could not use split half or k-fold cross-validation as the sample size in the test sample was insufficient to estimate the parameters of an SEM. This strategy allows the network structure to be generated on an out-of-bag sample to make a fair comparison to the LVM approach, which also was not generated on the data. We repeated this five times on different training and test datasets for both the NAPLS-3 and CATIE studies to ensure the results can be replicated. Using multiple training and test datasets is useful to avoid the training sample being unusual with respect to the test dataset. No participant was included more than once across training samples.

Regarding the NAPLS3 dataset, the network model was a reconstructed network model on 11 items of the SOPS, representing items from the positive and negative subscales (network model). We added a covariance term between latent variable models, which was significant for each LVM in the test datasets. We also combined the edges found in the network model (between items) and LVM (between items and latent variables) and named this the integrated model. We repeated this five times for five training and test samples for the NAPLS3 study. Given this, we constructed 15 SEMs on five test datasets for each model (network model, LVM, and integrated model) for the NAPLS3 study. Similarly, for the CATIE dataset, the network model is a reconstructed a network model of 14 items of the PANSS representing the positive and negative symptom subscales. Again, we added a covariance term between latent variables as this was significant for each LVM in the test datasets for the CATIE dataset. Lastly, the integrated model combines the LVM and network model, where relationships between latent variables and items in the LVM and the relationships between items in the network model were included in the integrated model. We repeated this process for five training and test datasets. Given this, we constructed 15 SEMs representing five test samples for each model for the CATIE study.

All Bayesian network models were implemented using the hill climbing algorithm in the package bnlearn version 4.9.4 (Scutari & Denis, 2021b). We also used the package lavaan version 0.6-17 (Rosseel, 2012) to implement the SEMs. We selected the hill climbing algorithm in the package bnlearn (Scutari & Denis, 2021b) for the Bayesian network, as it can use BIC scores to optimize the network. We did not specify priors or hyperparameters. We used maximum likelihood estimation of the parameters to estimate the model fit. We obtained the comparative fit index (CFI), Tucker-Lewis Index (TLI), and root mean square error of approximation (RMSEA). We also evaluated the fit of the model-implied variance-covariance matrix to the observed variance-covariance matrix using a chi-square test of model fit. Additionally, we extracted the Akaike information criterion (AIC), BIC, and the log-likelihood for each model from fitted SEMs using the lavaan package (Rosseel, 2012). For CFI and TLI, scores range between zero and one, where

higher scores indicate that the model fits the data better, with scores above .95 being acceptable (Hu & Bentler, 1999). Accordingly, RMSEA has values between zero and one, where lower values indicate a better model. RMSEA values should be below approximately .06 to suggest a satisfactory model fit (Hu & Bentler, 1999). We then compared the LVM and integrated model for both the NAPLS3 and CATIE data, using likelihood ratio tests as the LVM is included within the integrated model. Because this study aimed to assess the complexity principle, given our a priori hypotheses, we did not conduct likelihood ratio tests of the integrated model versus the network model. We did, however, include the fit statistics to compare the integrative model to the network model. The summary statistics for SOPS and the PANSS can be found in Table S1 and Table S2 in the Supplementary Materials Summary Scores for the NAPLS3 and CATIE study section, respectively. This study was not preregistered and all code used to produce the results of this data can be found in https://github.com/Khan-Buchwald/Evidence_for_the_Network_Theory_of_Mental_Disorders.

Results

Table 3 provides the model fit statistics for the 30 models mentioned in the Analysis subsection. The results in Table 3 suggest that the integrated models better fit the data than the LVMs in the NAPLS3 and CATIE studies when comparing CFI, TLI, and RMSEA. The integrated models fit the data adequately, even when obtaining the structure of the BN on a small sample size. The LVM had a relatively poor fit to the NAPLS3 and CATIE study data for the CFI, TLI, and RMSEA indices. The network models had better BIC, AIC, and log-likelihood scores than the LVMs and integrated models in all samples across the NAPLS3 and CATIE studies. In a chi-square test of model fit, the model implied variance-covariance matrix and the observed variance-covariance matrix were significantly different from each other in every network model ($p = .001$), LVM model ($p < .001$), and integrated model ($p < .001$) in the NAPLS3 study and the CATIE study. The statistical significance suggests that the models may not fully account for the observed covariance structure in the data. Two integrated models had estimated negative variance in the model, suggesting the model is underpowered or mis-specified.

Table 4 presents the results of the likelihood ratio tests between the integrated models and the LVMs. The degrees of freedom in Table 4 represent the difference between the number of unique pieces of information and the number of estimated parameters. Hence, the model with fewer degrees of freedom is more complex. The results suggest that for each random sample, the integrated model had a significantly better fit than the latent variable model ($p < .001$) in every sample and for both datasets. The statistical significance indicates that the model fit is significantly better in the integrated model after considering the difference in the number of parameters of the two models.

The structural equation model in Figure 2 presents the best-fitting model according to CFI, TLI, and RMSEA and represents an integrated model from the NAPLS3 dataset.

Table 3. Fit Statistics for each SEM and BN

Study	Model	Sample	CFI	TLI	RMSEA	Chi Sq.	df	p	BIC	AIC	Log Likelihood
NAPLS3	Network model	1	0.925	0.883	0.090	230.5	35	<.001	23668	23532	-11736
NAPLS3	Network model	2	0.862	0.795	0.118	396.5	37	<.001	23815	23687	-11816
NAPLS3	Network model	3	0.839	0.773	0.127	473.3	39	<.001	24062	23944	-11946
NAPLS3	Network model	4	0.888	0.829	0.108	329.5	36	<.001	23721	23589	-11766
NAPLS3	Network model	5	0.929	0.843	0.103	208.7	25	<.001	23732	23550	-11735
NAPLS3	LVM model	1	0.884	0.852	0.101	345.5	43	<.001	26460	26356	-13155
NAPLS3	LVM model	2	0.872	0.837	0.106	376.3	43	<.001	26496	26392	-13173
NAPLS3	LVM model	3	0.878	0.844	0.105	372.4	43	<.001	26414	26310	-13132
NAPLS3	LVM model	4	0.877	0.843	0.104	364.6	43	<.001	26411	26306	-13130
NAPLS3	LVM model	5	0.871	0.834	0.106	376.6	43	<.001	26483	26379	-13166
NAPLS3	Integrated model	1	0.987	0.970	0.045	58.5	24	<.001	26298	26107	-13011
NAPLS3	Integrated model	2	0.974	0.944	0.062	95.1	26	<.001	26326	26145	-13032
NAPLS3	Integrated model	3	0.975	0.951	0.059	95.2	28	<.001	26235	26062	-12993
NAPLS3	Integrated model	4	0.975	0.950	0.059	91.8	27	<.001	26242	26065	-12994
NAPLS3	Integrated model	5*	0.956	0.914	0.076	140.9	28	<.001	26346	26173	-13049
CATIE	Network model	1	0.885	0.822	0.092	693.0	59	<.001	50921	50690	-25300
CATIE	Network model	2	0.904	0.854	0.084	587.5	59	<.001	46240	46019	-22967
CATIE	Network model	3	0.909	0.866	0.079	551.1	62	<.001	50613	50397	-25156
CATIE	Network model	4	0.892	0.839	0.087	649.3	61	<.001	51062	50841	-25378
CATIE	Network model	5	0.910	0.870	0.079	556.7	63	<.001	50452	50241	-25079
CATIE	LVM model	1	0.730	0.677	0.124	1557.6	76	<.001	55840	55690	-27816
CATIE	LVM model	2	0.743	0.692	0.122	1496.5	76	<.001	55775	55626	-27784
CATIE	LVM model	3	0.732	0.680	0.122	1510.1	76	<.001	55918	55769	-27855
CATIE	LVM model	4	0.737	0.685	0.122	1511.7	76	<.001	56032	55883	-27912
CATIE	LVM model	5	0.723	0.669	0.125	1590.7	76	<.001	55922	55773	-27858
CATIE	Integrated model	1	0.950	0.916	0.063	328.6	54	<.001	54768	54506	-27202
CATIE	Integrated model	2	0.978	0.955	0.047	168.3	45	<.001	54668	54360	-27120
CATIE	Integrated model	3*	0.948	0.905	0.066	329.6	50	<.001	54923	54640	-27265
CATIE	Integrated model	4	0.963	0.936	0.055	251.9	52	<.001	54944	54671	-27283
CATIE	Integrated model	5	0.946	0.908	0.066	351.7	54	<.001	54841	54578	-27238

Note. LVM = Latent variable model; N = Sample size; CFI = Comparative fit index; TLI = Tucker-Lewis index; RMSEA = Root mean square error of approximation; Chi sq. = Chi-square test statistic; df = Degrees of freedom; p = p-value; BIC = Bayesian information criterion; AIC = Akaike information criterion;*. This model had estimated negative variances; Bold statistics indicate which model has the best fit for each fit statistic for both the NAPLS3 and the CATIE study.

Table 4. Likelihood Ratio Test of LVM and Integrated Model for Each Sample

Study	Model one	Model two	Sample	df Model 1	df Model 2	Chi Sq. diff	df diff	p ¹
NAPLS3	LVM model	Integrated model	1	43	24	221.1	19	<.001
NAPLS3	LVM model	Integrated model	2	43	26	217.8	17	<.001
NAPLS3	LVM model	Integrated model	3	43	28	216.1	15	<.001
NAPLS3	LVM model	Integrated model	4	43	27	199.5	16	<.001
NAPLS3	LVM model	Integrated model	5*	43	28	155.2	15	<.001
CATIE	LVM model	Integrated model	1	76	54	950.9	22	<.001
CATIE	LVM model	Integrated model	2	76	45	1151.1	31	<.001
CATIE	LVM model	Integrated model	3*	76	50	865.7	26	<.001
CATIE	LVM model	Integrated model	4	76	52	1042.4	24	<.001
CATIE	LVM model	Integrated model	5	76	54	956.7	22	<.001

Note. LVM = Latent variable model; df = Degrees of freedom; p = p-value; 1: Using the Satorra and Bentler approximation; *: This model had estimated negative variances; degrees of freedom represent the difference between the number of unique pieces of information minus the number of parameters, as more complex models have fewer degrees of freedom.

The model includes the relationships between items and the latent variables and the relationships between items as identified by the network model and is presented to give a visual view of the integrated models reconstructed in this study. Significance values between latent variables and items have been omitted from this Figure. The results suggest that social anhedonia is not a child node of any other item, meaning it is a predictor variable in this study. Perceptual abnormalities/hallucination and expressions of emotion are child nodes only, indicating that they are response variables only. In addition to this, the strongest significant item-item relationship was between avolition and occupational functioning, with higher scores on the avolition items predicting higher scores on the occupational functioning item. Similarly, there was a moderate positive relationship between disorganized communication and ideational richness, and a moderate positive relationship between the predictor disorganized communication and response unusual thought content/delusional ideas. Items suspicious/persecutory ideas and perceptual abnormalities/hallucinations had associations with other variables, but these were non-significant. All other figures for the integrated models are provided in the Supplementary Materials Model Structures section.

Figure 3 presents the best-fitting model for the PANSS from the CATIE study. The results suggest that the items delusions and poor rapport were not child nodes of other items, and stereotyped thinking was not a parent of other items, a child node only. The strongest relationship was between emotional withdrawal and passive/apathetic social withdrawal. Poor rapport was also highly associated with lack of spontaneity and flow of conversation, and delusions

was also highly associated with grandiosity, hallucinatory behaviour, and suspiciousness/persecution. These had strong or close to strong effect sizes. Delusions was also conditionally independent of conceptual disorganisation given scores on grandiosity, excitement given scores on grandiosity, and hostility given scores on suspiciousness/persecution. Hallucinations was also conditionally independent of suspiciousness/persecution and grandiosity, given scores on delusions. Emotional withdrawal had the highest degree, the most edges. In terms of items, emotional withdrawal had the highest number of outgoing edges, and stereotyped thinking had the most incoming edges.

Discussion

We aimed to assess the complexity principle of Borsboom (2017) and identify if we could integrate the latent variable model and network model to obtain a better-fitting model. This study compared models of the theorized relationships between items of the SOPS in people at ultra-high risk of schizophrenia based on models from the network theory of mental disorders and the LVM approach. This is replicated for items of the PANSS with participants with chronic schizophrenia. Our research provides empirical support for directed acyclic graphs with latent variables as adequate models of the underlying model of the responses to items of the SOPS and PANSS, given the LVM structure. Adding associations between items found in a network model and the associations between the latent variables and the items found in the LVM model significantly improved the fit over the LVM model alone in the

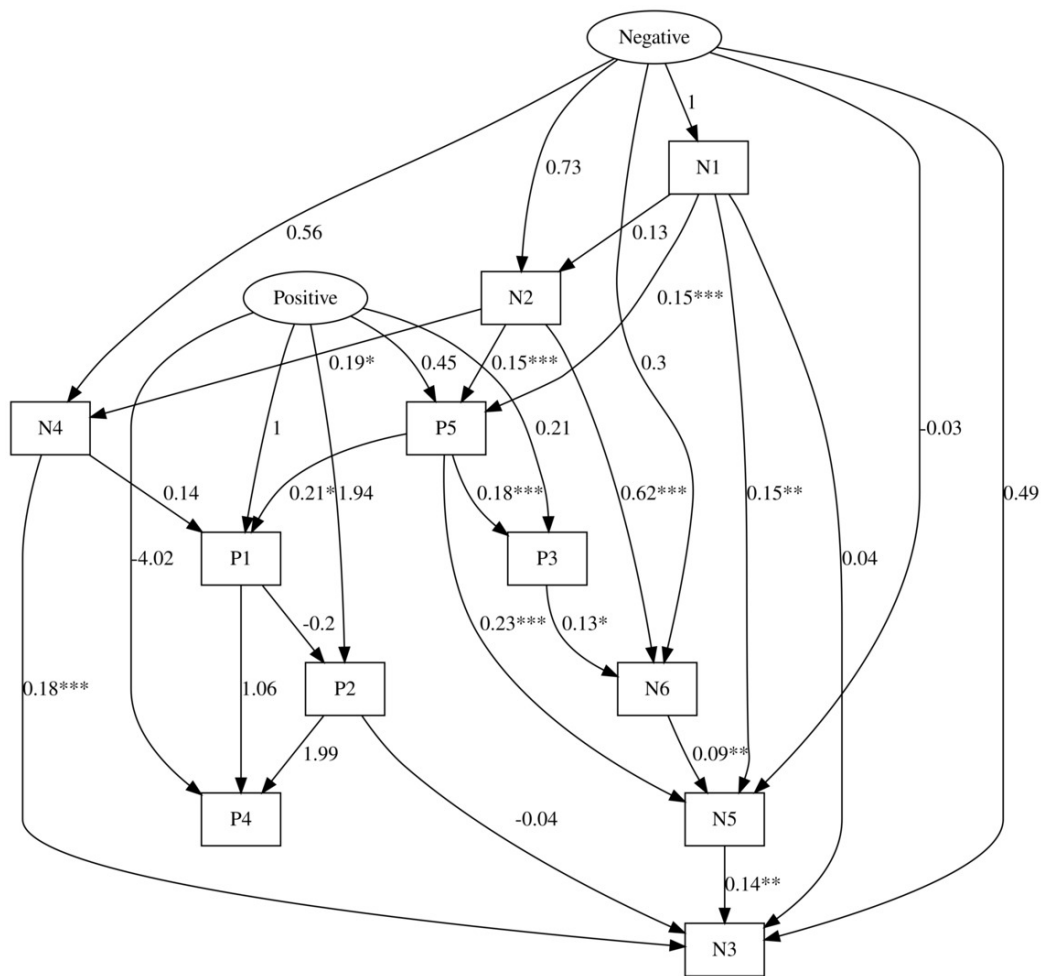


Figure 2. Structural Equation Model Diagram of Best-Fitting Model for the SOPS

Note. Positive = Latent variable for the positive symptoms subscale; negative = Latent variable for the negative symptoms subscale; P1 = Unusual thought content/delusional ideas; P2 = Suspicious/persecutory ideas; P3 = Grandiose ideas; P4 = Perceptual abnormalities/hallucinations; P5 = Disorganized communication; N1 = Social anhedonia; N2 = Avolition; N3 = Expression of emotion; N4 = Experience of emotions and self; N5 = Ideational richness; N6 = Occupational functioning; Edge weights = Unstandardized regression coefficients; * = Significant; ** = Moderately significant; *** = Highly significant.

NAPLS3 study. This significance adds partial empirical support to the complexity principle of the theory by Borsboom (2017), as items representing symptoms or observations should have associations between them. This finding, however, rests on the assumption that the local independence assumption was not met with the a priori latent variable model. We also found that including latent variables (as prescribed under the LVM) improved the fit statistics CFI, TLI, and RMSEA over the network model without latent variables. This finding suggests that network models with latent variables can improve the fit of a latent variable model and network models without latent variables, which may be used in future research where these models have suboptimal fit.

The network model with latent variables integrates the two modeling approaches and fits better than the network model and the latent variable model alone. The notion that items interact, as Borsboom (2017) proposed, is supported by these findings. We also confirmed these results in a second empirical assessment of the PANSS data from the CATIE pharmaceutical trial. Hence, we provide empirical evidence to support the validity of the complexity prin-

ciple of the network theory of mental disorders as proposed by Borsboom (2017), when the latent variable model is suboptimal in PDS with a chronic condition and in people at ultra-high risk of schizophrenia on the PANSS and SOPS assessments, respectively.

Borsboom and Cramer (2013), Borsboom (2017), Bringmann et al. (2022), and Cramer et al. (2010) advocated for the advancement of the theory that psychological disorders are embedded in a network of symptoms. Additionally, Epskamp and Borsboom published previously on network models with latent variables. Adding latent variables to networks is not a novel method used in our study (Epskamp et al., 2017; Pearl, 1995). Other research comparing the LVM and network model tends to identify that the network models generally have a better fit to the data (Christensen et al., 2019; Kan et al., 2020; Moriana et al., 2022; Moriconi et al., 2025). We found a good fit even when generating the network on a small sample size of 100, potentially because networks are data-driven models compared to our LVMs (Liew, Palacios-Ceña, et al., 2023). Network models maybe useful even when the underlying factor structure has been well-researched (Schmank et al., 2019). This may par-

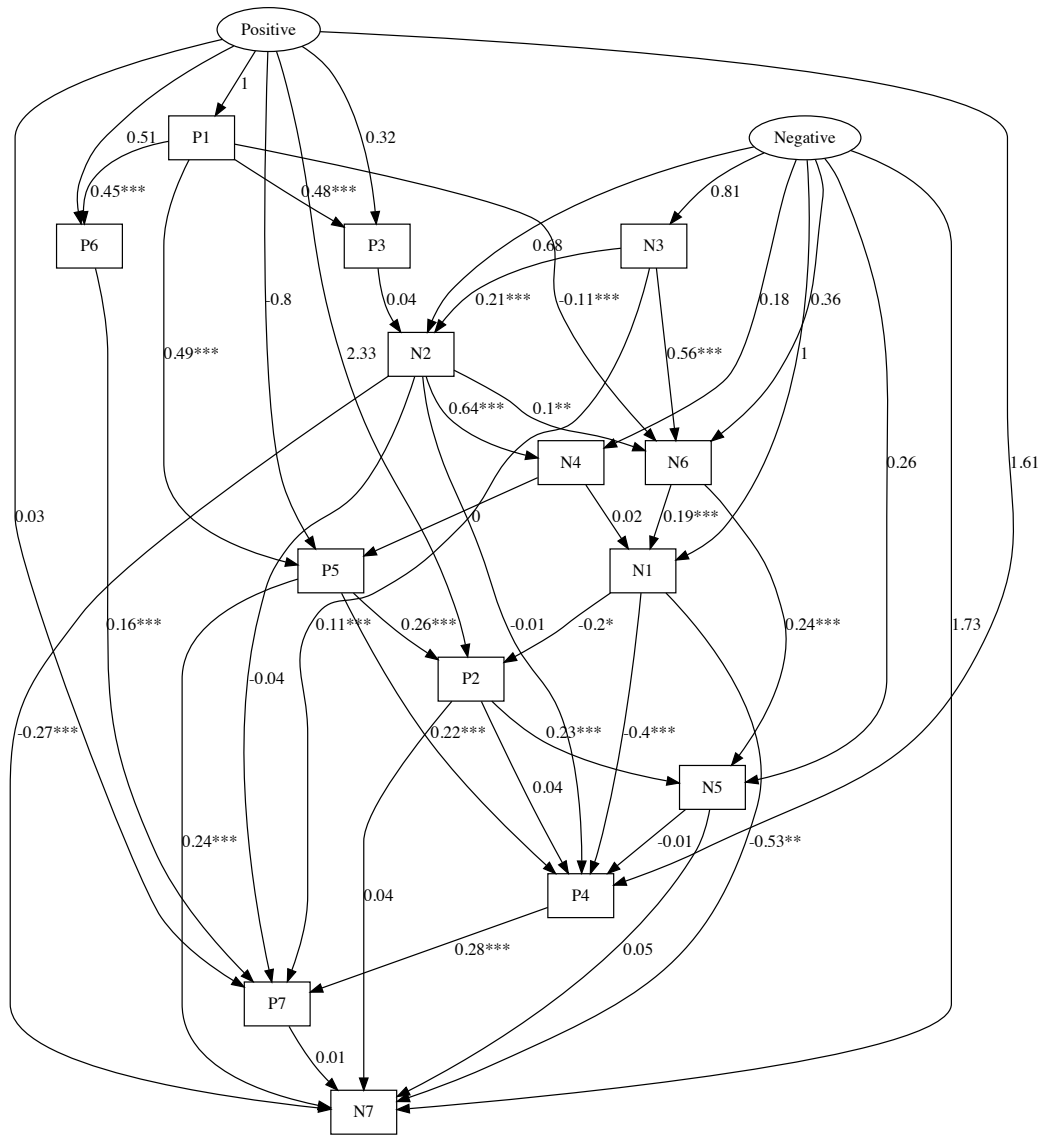


Figure 3. Structural Equation Model Diagram of Best-Fitting Model for the PANSS

Note. P1 = Delusions; P2 = Conceptual disorganisation; P3 = Hallucinatory behaviour; P4 = Excitement; P5 = Grandiosity; P6 = Suspiciousness/persecution; P7 = Hostility; N1 = Blunted affect; N2 = Emotional withdrawal; N3 = Poor rapport; N4 = Passive/apathetic social withdrawal; N5 = Difficulty in abstract thinking; N6 = Lack of spontaneity/flow of conversation; N7 = Stereotyped thinking; Positive = Latent variable for the positive symptoms subscale; negative = Latent variable for the negative symptoms subscale; * = Significant; ** = Moderately significant; *** = Highly significant.

tially be due to the use of sum scores to measure latent variables, as opposed to using the factors from the principal components analysis, which could be problematic (McNeish & Wolf, 2020). In a proposed LVM using exploratory factor analysis, the observed variables are forced and assumed to be conditionally independent given the latent variable. Although conditional (local) independence is guaranteed using principal components analysis (Yong & Pearce, 2013), as previously discussed, using sum scores to represent latent variables may introduce unmodelled residual variation. This unmodelled residual variation allows correlations or dependancies between observed variables to occur, enabling relationships found in a network to be modelled (Epskamp et al., 2017).

Other researchers have identified that although these two approaches are different conceptually, and many arguments have been debated on both sides (Bringmann &

Eronen, 2018), these models can be complementary. Bringmann and Eronen (2018) identify that if variable A and Variable B have a common cause (latent) variable C, then this common cause may explain the covariance of variable A and variable B, but not all of it. This is essentially what is found in our empirical study, where the latent variable model does not explain all the covariance structure found in the observed responses to the items of the SOPS and PANSS. This may be inherent in the calculation of subscores to represent latent variables. It is unclear the extent to which latent variable models proposed in the wider psychometric literature have a suboptimal fit, where relationships between observed values can be included to improve the model, as identified by the network theory of mental disorders.

Although we found a significantly better fit for a network model with latent variables, the LVM may still be superior

when modeling some phenomena, or another latent variable structure may be better for the PANSS and SOPS (Kay et al., 1987; Miller et al., 2002). We used latent variables as documented by Kay et al. (1987) and Miller et al. (2002), although this does not appear to be the optimal latent variable model for these data or these assessments. The latent variables were proposed by these authors a priori instead of being conducted using methods such as principal components analysis. Hence, although we found the integrated model fit better, and in this case, items can interact, this does not refute other latent variable models that could fit these data.

In terms of outcomes for schizophrenia and people ultra-high risk of schizophrenia, although the PANSS and SOPS are well documented as measures of the severity of symptoms of schizophrenia, with several meta-analyses, reviews, and publications of this measure to date (Geck et al., 2025; Lim et al., 2021; Miller et al., 1999; Obermeier et al., 2011; Shafer & Dazzi, 2019; Woods et al., 2019b), we found that the factor structure of positive and negative latent variables was a poor fit to the data for both the PANSS and the SOPS. This evidence has been reported in a previous meta-analysis for the PANSS (Geck et al., 2025). A lack of fit would indicate that the model of the PANSS for positive and negative symptoms needs to be revised, either by introducing additional factors (Shafer & Dazzi, 2019), re-specifying associations between items and factors (Kline, 2023), or as found in this research, introduce associations between items as local independence may be violated. Our integrated models show excellent fit, better than an LVM without edges and a network model without latent variables. This means that it may be necessary to model dependency between items in this measure for PDS and people at ultra-high risk of schizophrenia.

There are some applied implications for our integrated model of people at ultra-high risk of schizophrenia. As identified in the results of the SOPS, occupational functioning is highly dependent on the predictor avolition, although this may not be a causal relationship. Another study, but on PDS, found that avolition was highly associated with employment as a functional outcome, but also recreational activities, potentially because people with schizophrenia have difficulty with estimating the amount of effort required (Okada et al., 2021). We also found an association between disorganized symptoms and delusional ideas. A longitudinal study on psychopathology identified that disorganized symptoms and negative symptoms predict later scores on symptoms of hallucination and delusions (Dominguez et al., 2010). Hence, it may be possible that disorganised thinking is in the pathway to experiencing delusions, although we cannot test this hypothesis directly in this study. Our study departs from the findings from Dominguez et al. (2010) as our study identified that perceptual abnormalities/hallucinations were also conditionally independent of disorganized communication, given unusual thought content/delusional ideas. Thought content/delusional ideas, therefore, acted as a mediating variable in the pathway. Our study was also cross-sectional and evaluated at one time period, unlike the study by Dominguez et al. (2010). These

inferences arise out of the added benefits of integrating a network model with latent variables, adding inferential capability provided by both approaches.

In terms of the applied implications of scores on the PANSS assessment for people with schizophrenia, delusions and hallucinations were highly associated. Both delusions and hallucinations frequently load onto the positive dimension of the PANSS across studies, according to one meta-analysis (Shafer & Dazzi, 2019). However, in these studies, it is assumed that hallucinations and delusions are conditionally independent given latent variables. In terms of other research on symptom networks of schizophrenia, two studies specifically examined relationships between items in the PANSS using a network approach (Demyttenaere et al., 2022; van Rooijen et al., 2018). They found that hallucinations and delusions are associated but tend not to be strongly associated, as reported in our study (Demyttenaere et al., 2022; van Rooijen et al., 2018). Interestingly, the item hallucinations is conditionally independent of grandiosity and suspiciousness/persecution given delusions, and this was also found in the partial correlation networks by Demyttenaere et al. (2022) and van Rooijen et al. (2018). This may indicate that themes of persecution and grandiosity are more associated with delusions, as opposed to hallucinations, especially given the wording of the items (Kay et al., 1987). Unlike Demyttenaere et al. (2022) and van Rooijen et al. (2018), the item emotional withdrawal tends to have many associations with other items. However, the interconnectedness of emotional withdrawal was more pronounced for acute populations as identified by Demyttenaere et al. (2022) and van Rooijen et al. (2018). Delusions and poor rapport were predictor variables (parent variables) only in the network, and stereotyped thinking was a child node (response variable) only in the network. More research is needed to identify whether these nodes have the potential to have cause-and-effect relationships, particularly using Markov equivalence classes (Scutari & Denis, 2021a). Our network may be suboptimal, and other networks, with different predictor and response relationships, may fit the data better. Hence, it is not possible, in our study, to identify the causal relationships using these orientations of predictor and response variables.

Network models and LVM are useful to explore statistics applicable in clinical contexts. The benefits of both models can be used to make clinical inferences for the care, well-being, or to improve outcomes for schizophrenia or other psychological disorders. The proposed LVM has the ability to be used to identify severity scores based on latent variables or cut-off values as a measure of severity or as a diagnostic tool, depending on the assessment's psychometric properties. Additionally, network statistics can be conducted on the network model itself. According to Borsboom (2017), mental disorders are best represented by interactions between symptoms as components of a network. Given this, the results of networks may have clinical utility for understanding or treating schizophrenia. Network statistics have been explored in depth by other researchers, including introducing probability queries to the network by treating it as an expert system, centrality statistics, and

edge statistics (Briganti et al., 2022; Bringmann et al., 2019). Treating networks as an expert system enables the user to query the network to determine the probability of an event given some evidence. For example, what is the probability that occupational functioning is in the 90th percentile when avolition is above the median? In the future, this may be utilized in clinical practice when formulating treatment plans. Recent research suggests that networks have the potential to be used to guide treatment using single-case longitudinal data, although considerable work is needed to validate this approach (Bringmann, 2021). In terms of centrality and edge statistics, symptom networks enable users to identify which nodes or edges are important, thereby characterizing the condition (Bringmann et al., 2019). Although intervening on one node may not be realistic in therapeutic settings (Bringmann et al., 2019), especially given the complex systems approach. However, our systematic review identified that cognition is central to schizophrenia across various studies (Buchwald et al., 2024). This may indicate that research on improving cognition in schizophrenia is necessary. Hence, both the LVM and symptom network model offer different benefits to the understanding and treatment of schizophrenia and psychological disorders, and the integrative approach combines the utility of LVM and the network approach to psychopathology.

Limitations

Our study has several limitations. First, we used ordinal data when obtaining the multivariate normal likelihood estimates. We used robust standard errors to reduce the effect of potential violations of test assumptions (Williams, 2021). Secondly, the implied variance-covariance structure was significantly different from the observed variance-covariance structure for all models, indicating that our identified models were not entirely faithful to the data. This suggests that although we found that the network models have a better fit, other models may better represent the responses to the SOPS and PANSS items. Some of the models did not converge and may suggest the model is over-parameterized, and a simpler model, such as an LVM or network model, may be better for simplicity. Lastly, some integrative models' coefficients have estimated negative variance despite having a better fit than the LVM and network model. Both of these considerations may be due to a lack of power in estimating many coefficients in the model.

Conclusions

The network models in the present study that included latent variables derived from the literature showed adequate fit, superior to the LVM that did not allow interactions between items. The finding that responses to items of the SOPS and PANSS interact is notable in our study as this is prohibited in LVMs due to all information already being explained by the latent variables (Fabrigar et al., 1999). However, for the latent variable model selected based on the authors of the SOPS and PANSS (Kay et al., 1987; Miller et al., 2002), we found that integrating the network model

and latent variable model fit the data the best. Hence, in a sample of people at ultra-high risk of schizophrenia and PDS, responses to items interact or are associated with the responses on other items. This provides partial empirical support for principle 1 of the network theory of mental disorders (Borsboom, 2017), when the assumption of local independence is violated. The integrated model can be used if the LVM has a poor fit to the data, to improve the model fit, and can be used to generate statistics from both the LVM and network model.

Contributions

Conceptualization: Khan Buchwald (Lead), Matthieu Vignes (Supporting), Margaret Sandham (Supporting), Ajit Narayanan (Supporting). Data curation: Khan Buchwald (Lead), Margaret Sandham (Supporting), Ajit Narayanan (Supporting), Richard Siegert (Supporting). Formal Analysis: Khan Buchwald (Lead), Matthieu Vignes (Supporting). Investigation: Khan Buchwald (Lead), Matthieu Vignes (Supporting), Ajit Narayanan (Supporting). Methodology: Khan Buchwald (Lead), Matthieu Vignes (Supporting), Ajit Narayanan (Supporting), Matt Williams (Supporting), Richard Siegert (Supporting). Project administration: Khan Buchwald (Lead). Software: Khan Buchwald (Lead). Validation: Khan Buchwald (Supporting), Matthieu Vignes (Lead), Margaret Sandham (Supporting), Ajit Narayanan (Supporting). Visualization: Khan Buchwald (Lead). Writing – original draft: Khan Buchwald (Lead), Margaret Sandham (Supporting). Writing – review & editing: Khan Buchwald (Supporting), Matthieu Vignes (Supporting), Margaret Sandham (Supporting), Ajit Narayanan (Supporting), Matt Williams (Supporting), Richard Siegert (Lead). Supervision: Matthieu Vignes (Supporting), Margaret Sandham (Supporting), Ajit Narayanan (Supporting), Richard Siegert (Lead).

Acknowledgements

Data and/or research tools used in the preparation of this manuscript were obtained from the National Institute of Mental Health (NIMH) Data Archive (NDA). NDA is a collaborative informatics system created by the National Institutes of Health to provide a national resource to support and accelerate research in mental health. Dataset identifier(s): NIMH Study DOI: 10.15154/8r2r-w424 This manuscript reflects the views of the authors and may not reflect the opinions or views of the NIH or of the Submitters submitting original data to NDA.

Data Accessibility

The raw data can be accessed from the National Institute of Mental Health (NIMH)'s website [NIMH Data Archive - NDA](#). The NIMH data archive is a national data repository that grants access through a standardized process to ensure ethical and secure data use. Researchers can independently gain access to the dataset by fulfilling the NIMH's standard

requirements. The following resource identifies the process of accessing the data: [Getting Access to Shared Data - NDA](#). Two datasets to be downloaded to reproduce our results are: Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) for Schizophrenia (Collection ID: 2081), and the 1/9 to 9/9 Predictors and Mechanisms of Conversion to Psychosis (NAPLS3) (Collection ID: 2275) datasets. To support the reproducibility of this research, the full analysis code—from raw data processing to the final results—is publicly available at:

https://github.com/KhanBuchwald/Evidence_for_the_Network_Theory_of_Mental_Disorders

Additionally, the analysis was conducted using the R programming language (v4.4.1), available at [R: The R Project for Statistical Computing](#)

All the analyses and the results in our paper can be fully reproduced using this data, code, and software.

Funding Information

The authors declare no funding was received for this research.

Ethics Statement

Ethics approval for research on each dataset was obtained (22/387 Static Symptom Networks of Schizophrenia; 22/388 Dynamic Symptom Networks of Schizophrenia) by the Auckland University of Technology Ethics Committee.

Competing Interests

The authors report no actual or perceived competing interests.

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Editors: Ginette Lafti (Associate Editor)

Submitted: March 03, 2025 PDT. Accepted: February 04, 2026 PDT. Published: March 16, 2026 PDT.



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References

- Abplanalp, S. J., & Green, M. F. (2022). Symptom structure in schizophrenia: Implications of latent variable modeling vs network analysis. *Schizophrenia Bulletin*, 48(3), 538–543. <https://doi.org/10.1093/schbul/sbac020>
- Addington, J., Liu, L., Brummitt, K., Bearden, C. E., Cadenhead, K. S., Cornblatt, B. A., Keshavan, M., Mathalon, D. H., McGlashan, T. H., & Perkins, D. O. (2022). North American prodrome longitudinal study (NAPLS 3): Methods and baseline description. *Schizophrenia Research*, 243, 262–267. <https://doi.org/10.1016/j.schres.2020.04.010>
- Almond, R. G., DiBello, L. V., Moulder, B., & Zapata-Rivera, J.-D. (2007). Modeling diagnostic assessments with Bayesian networks. *Journal of Educational Measurement*, 44(4), 341–359. <https://doi.org/10.1111/j.1745-3984.2007.00043.x>
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596>
- Barrett, P. (2007). Structural equation modelling: Adjudging model fit. *Personality and Individual Differences*, 42(5), 815–824. <https://doi.org/10.1016/j.paid.2006.09.018>
- Benfer, N., Bardeen, J. R., Cero, I., Kramer, L. B., Whiteman, S. E., Rogers, T. A., Silverstein, M. W., & Weathers, F. W. (2018). Network models of posttraumatic stress symptoms across trauma types. *Journal of Anxiety Disorders*, 58, 70–77. <https://doi.org/10.1016/j.janxdis.2018.07.004>
- Borsboom, D. (2008). Psychometric perspectives on diagnostic systems. *Journal of Clinical Psychology*, 64(9), 1089–1108. <https://doi.org/10.1002/jclp.20503>
- Borsboom, D. (2017). A network theory of mental disorders. *World Psychiatry*, 16(1), 5–13. <https://doi.org/10.1002/wps.20375>
- Borsboom, D., & Cramer, A. O. (2013). Network analysis: An integrative approach to the structure of psychopathology. *Annual Review of Clinical Psychology*, 9(1), 91–121. <https://doi.org/10.1146/annurev-clinpsy-050212-185608>
- Borsboom, D., Mellenbergh, G. J., & Van Heerden, J. (2003a). The theoretical status of latent variables. *Psychological Review*, 110(2), 203. <https://doi.org/10.1037/0033-295X.110.2.203>
- Borsboom, D., Mellenbergh, G. J., & Van Heerden, J. (2003b). The theoretical status of latent variables. *Psychological Review*, 110(2), 203–219. <https://doi.org/10.1037/0033-295X.110.2.203>
- Briganti, G., Scutari, M., & McNally, R. J. (2022). A tutorial on Bayesian networks for psychopathology researchers. *Psychological Methods*. <https://doi.org/10.1037/met0000479>
- Bringmann, L. F. (2021). Person-specific networks in psychopathology: Past, present, and future. *Current Opinion in Psychology*, 41, 59–64. <https://doi.org/10.1016/j.copsyc.2021.03.004>
- Bringmann, L. F., Albers, C., Bockting, C., Borsboom, D., Ceulemans, E., Cramer, A., Epskamp, S., Eronen, M. I., Hamaker, E., & Kuppens, P. (2022). Psychopathological networks: Theory, methods and practice. *Behaviour Research and Therapy*, 149, 1–15. <https://doi.org/10.1016/j.brat.2021.104011>
- Bringmann, L. F., Elmer, T., Epskamp, S., Krause, R. W., Schoch, D., Wichers, M., Wigman, J. T., & Snippe, E. (2019). What do centrality measures measure in psychological networks? *Journal of Abnormal Psychology*, 128(8), 892–903. <https://doi.org/10.1037/abn0000446>
- Bringmann, L. F., & Eronen, M. I. (2018). Don't blame the model: Reconsidering the network approach to psychopathology. *Psychological Review*, 125(4), 606–615. <https://doi.org/10.1037/rev0000108>
- Buchwald, K., Narayanan, A., Siegert, R. J., Vignes, M., Arrowsmith, K., & Sandham, M. (2024). Centrality statistics of symptom networks of schizophrenia: A systematic review. *Psychological Medicine*, 54(6), 1061–1073. <https://doi.org/10.1017/S003329172300363X>
- Christensen, A. P., Gross, G. M., Golino, H. F., Silvia, P. J., & Kwapił, T. R. (2019). Exploratory graph analysis of the Multidimensional Schizotypy Scale. *Schizophrenia Research*, 206, 43–51. <https://doi.org/10.1016/j.schres.2018.12.018>
- Contreras, A., Nieto, I., Valiente, C., Espinosa, R., & Vazquez, C. (2019). The study of psychopathology from the network analysis perspective: A systematic review. *Psychotherapy and Psychosomatics*, 88(2), 71–83. <https://doi.org/10.1159/000497425>
- Cramer, A. O., Waldorp, L. J., Van Der Maas, H. L., & Borsboom, D. (2010). Comorbidity: A network perspective. *Behavioral and Brain Sciences*, 33(2–3), 137–150. <https://doi.org/10.1017/S0140525X09991567>
- Demyttenaere, K., Leenaerts, N., Acsai, K., Sebe, B., Laszlovszky, I., Barabácssy, Á., Fonticoli, L., Szatmári, B., Earley, W., Németh, G., & Correll, C. U. (2022). Disentangling the symptoms of schizophrenia: Network analysis in acute phase patients and in patients with predominant negative symptoms. *European Psychiatry*, 65(1), Article e18. <https://doi.org/10.1192/j.eurpsy.2021.2241>
- Díez-Mesa, F., de Oña, R., & de Oña, J. (2018). Bayesian networks and structural equation modelling to develop service quality models: Metro of Seville case study. *Transportation Research Part A: Policy and Practice*, 118, 1–13. <https://doi.org/10.1016/j.tra.2018.08.012>
- Dominguez, M. -d.-G., Saka, M. C., Lieb, R., Wittchen, H.-U., & Os, J. v. (2010). Early expression of negative/disorganized symptoms predicting psychotic experiences and subsequent clinical psychosis: A 10-year study. *American Journal of Psychiatry*, 167(9), 1075–1082. <https://doi.org/10.1176/appi.ajp.2010.09060883>

- Epskamp, S., Rhemtulla, M., & Borsboom, D. (2017). Generalized network psychometrics: Combining network and latent variable models. *Psychometrika*, 82(4), 904–927. <https://doi.org/10.1007/s11336-017-9557-x>
- Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods*, 4(3), 272–299. <https://doi.org/10.1037/1082-989X.4.3.272>
- Fried, E. I., Eidhof, M. B., Palic, S., Costantini, G., Huisman-van Dijk, H. M., Bockting, C. L. H., Engelhard, I., Armour, C., Nielsen, A. B. S., & Karstoft, K.-I. (2018). Replicability and generalizability of posttraumatic stress disorder (PTSD) networks: A cross-cultural multisite study of PTSD symptoms in four trauma patient samples. *Clinical Psychological Science*, 6(3), 335–351. <https://doi.org/10.1177/2167702617745092>
- Fried, E. I., Epskamp, S., Nesse, R. M., Tuerlinckx, F., & Borsboom, D. (2016). What are “good” depression symptoms? Comparing the centrality of DSM and non-DSM symptoms of depression in a network analysis. *Journal of Affective Disorders*, 189, 314–320. <https://doi.org/10.1016/j.jad.2015.09.005>
- Fried, E. I., van Borkulo, C. D., Cramer, A. O., Boschloo, L., Schoevers, R. A., & Borsboom, D. (2017). Mental disorders as networks of problems: A review of recent insights. *Social Psychiatry and Psychiatric Epidemiology*, 52(1), 1–10. <https://doi.org/10.1007/s00127-016-1319-z>
- Geck, S., Roithmeier, M., Bühner, M., Wehr, S., Weigel, L., Priller, J., Davis, J. M., & Leucht, S. (2025). COSMIN systematic review and meta-analysis of the measurement properties of the Positive and Negative Syndrome Scale (PANSS). *eClinicalMedicine*, 82, 41–50. <https://doi.org/10.1016/j.eclinm.2025.103155>
- Guyon, H., Falissard, B., & Kop, J.-L. (2017). Modeling psychological attributes in psychology—an epistemological discussion: Network analysis vs. latent variables. *Frontiers in Psychology*, 8, 1–10. <https://doi.org/10.3389/fpsyg.2017.00798>
- Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, 6(1), 1–55. <https://doi.org/10.1080/10705519909540118>
- Isvoranu, A.-M., Boyette, L.-L., Guloksuz, S., & Borsboom, D. (2017). Symptom network models of psychosis. In C. A. Tamminga, E. I. Ivleva, U. Reininghaus, & J. van Os (Eds.), *Psychotic disorders: Comprehensive conceptualization and treatments* (pp. 70–78). Oxford University Press.
- Jones, P. J., Heeren, A., & McNally, R. J. (2017). Commentary: A network theory of mental disorders. *Frontiers in Psychology*, 8, 1–3. <https://doi.org/10.1002/wps.20375>
- Kan, K.-J., de Jonge, H., van der Maas, H. L. J., Levine, S. Z., & Epskamp, S. (2020). How to compare psychometric factor and network models. *Journal of Intelligence*, 8(4), 1–10. <https://doi.org/10.3390/jintelligence8040035>
- Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The Positive and Negative Syndrome Scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, 13(2), 261–276. <https://doi.org/10.1093/schbul/13.2.261>
- Khan, A., Yavorsky, C., Liechti, S., Opler, M., Rothman, B., DiClemente, G., Lucic, L., Jovic, S., Inada, T., & Yang, L. (2013). A rasch model to test the cross-cultural validity in the Positive and Negative Syndrome Scale (PANSS) across six geo-cultural groups. *BMC Psychology*, 1(1), 1–18. <https://doi.org/10.1186/2050-7283-1-5>
- Kline, R. B. (2023). *Principles and practice of structural equation modeling* (4th ed.). Guilford Press. <https://www.guilford.com/books/Principles-and-Practice-of-Structural-Equation-Modeling/Rex-Kline/9781462551910>
- Kuhn, M., & Johnson, K. (2013). *Applied predictive modeling*. Springer. <https://doi.org/10.1007/978-1-4614-6849-3>
- Kumari, S., Malik, M., Florival, C., Manalai, P., & Sonje, S. (2017). An assessment of five (PANSS, SAPS, SANS, NSA-16, CGI-SCH) commonly used symptoms rating scales in schizophrenia and comparison to newer scales (CAINS, BNSS). *Journal of Addiction Research & Therapy*, 8(3), 1–14. <https://doi.org/10.4172/2155-6105.1000324>
- Li, X., Zhang, Y., Guo, F., Gao, X., & Wang, Y. (2018). Predicting the effect of land use and climate change on stream macroinvertebrates based on the linkage between structural equation modeling and Bayesian network. *Ecological Indicators*, 85, 820–831. <https://doi.org/10.1016/j.ecolind.2017.11.044>
- Liew, B. X. W., Hartvigsen, J., Scutari, M., & Kongsted, A. (2023). Data-driven pathway analysis of physical and psychological factors in low back pain. *Journal of Clinical Epidemiology*, 153, 55–77. <https://doi.org/10.1016/j.jclinepi.2022.11.010>
- Liew, B. X. W., Palacios-Ceña, M., Scutari, M., Fuensalida-Novo, S., Guerrero-Peral, A., Ordás-Bandera, C., Pareja, J. A., & Fernández-de-las-Peñas, C. (2023). Path analysis models integrating psychological, psycho-physical and clinical variables in individuals with tension-type headache. *The Journal of Pain*, 24(3), 426–436. <https://doi.org/10.1016/j.jpain.2022.10.003>
- Lim, K., Peh, O.-H., Yang, Z., Rekhi, G., Rapisarda, A., See, Y.-M., Rashid, N. A. A., Ang, M.-S., Lee, S.-A., Sim, K., Huang, H., Lencz, T., Lee, J., & Lam, M. (2021). Large-scale evaluation of the Positive and Negative Syndrome Scale (PANSS) symptom architecture in schizophrenia. *Asian Journal of Psychiatry*, 62, 1–8. <https://doi.org/10.1016/j.ajp.2021.102732>
- Marcot, B. G., & Penman, T. D. (2019). Advances in Bayesian network modelling: Integration of modelling technologies. *Environmental Modelling & Software*, 111, 386–393. <https://doi.org/10.1016/j.envsoft.2018.09.016>
- McFarland, D. (2020). The effects of using partial or uncorrected correlation matrices when comparing network and latent variable models. *Journal of Intelligence*, 8(1), 1–6. <https://doi.org/10.3390/jintelligence8010007>

- McNeish, D., & Wolf, M. G. (2020). Thinking twice about sum scores. *Behavior Research Methods*, 52, 2287–2305. <https://doi.org/10.3758/s13428-020-01398-0>
- Miller, T. J., McGlashan, T. H., Rosen, J. L., Somjee, L., Markovich, P. J., Stein, K., & Woods, S. W. (2002). Prospective diagnosis of the initial prodrome for schizophrenia based on the Structured Interview for Prodromal Syndromes: preliminary evidence of interrater reliability and predictive validity. *American Journal of Psychiatry*, 159(5), 863–865. <https://doi.org/10.1176/appi.ajp.159.5.863>
- Miller, T. J., McGlashan, T. H., Woods, S. W., Stein, K., Driesen, N., Corcoran, C. M., Hoffman, R., & Davidson, L. (1999). Symptom assessment in schizophrenic prodromal states. *Psychiatric Quarterly*, 70(4), 273–287. <https://doi.org/10.1023/A:1022034115078>
- Moriana, J. A., Jurado-González, F. J., García-Torres, F., Contreras, A., Muñoz-Navarro, R., González-Blanch, C., Medrano, L. A., Ruiz-Rodríguez, P., & Cano-Vindel, A. (2022). Exploring the structure of the GAD-7 scale in primary care patients with emotional disorders: A network analysis approach. *Journal of Clinical Psychology*, 78(2), 283–297. <https://doi.org/10.1002/jclp.23217>
- Moriconi, V., Menéndez, A., Neimeyer, R. A., & Moggia, D. (2025). Adaptation of the Spanish Grief and Meaning Reconstruction Inventory: An initial validation and network analysis. *OMEGA - Journal of Death and Dying*, 90(3), 1229–1249. <https://doi.org/10.1177/00302228221118169>
- National Institute of Mental Health. (2024a). Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) [Dataset]. In *National Institute of Mental Health Data Archive*. https://nda.nih.gov/edit_collection.html?id=2081
- National Institute of Mental Health. (2024b). North American Prodromal Longitudinal Study 3 (NAPLS3) [Dataset]. In *National Institute of Mental Health Data Archive*. https://nda.nih.gov/edit_collection.html?id=2275
- Nuijten, M., Deserno, M., Cramer, A., & Borsboom, D. (2016). Mental disorders as complex networks: An introduction and overview of a network approach to psychopathology. *Clinical Neuropsychiatry*, 13(4/5), 68–76.
- Obermeier, M., Schennach-Wolff, R., Meyer, S., Möller, H.-J., Riedel, M., Krause, D., & Seemüller, F. (2011). Is the PANSS used correctly? A systematic review. *BMC Psychiatry*, 11(1), 1–5. <https://doi.org/10.1186/1471-244X-11-113>
- Okada, H., Hirano, D., & Taniguchi, T. (2021). Impact of negative symptom domains and other clinical characteristics on functional outcomes in patients with schizophrenia. *Schizophrenia Research and Treatment*, 2021(1), 1–8. <https://doi.org/10.1155/2021/8864352>
- Oude Maatman, F. (2020). Reformulating the network theory of mental disorders: Folk psychology as a factor, not a fact. *Theory & Psychology*, 30(5), 703–722. <https://doi.org/10.1177/0959354320921464>
- Pearl, J. (1995). Causal diagrams for empirical research. *Biometrika*, 82(4), 669–688. <https://doi.org/10.1093/biomet/82.4.669>
- Pearl, J. (2009). *Causality*. Cambridge university press. <https://doi.org/10.1017/CBO9780511803161>
- Pearl, J. (2011). *Bayesian networks*.
- R Core Team. (2024). *R: A language and environment for statistical computing* (4.3.2) [Computer software]. R Foundation for Statistical Computing. <https://r-project.org/>
- Rosseel, Y. (2012). lavaan: An R package for structural equation modeling. *Journal of Statistical Software*, 48, 1–36. <https://doi.org/10.18637/jss.v048.i02>
- Schmank, C. J., Goring, S. A., Kovacs, K., & Conway, A. R. A. (2019). Psychometric network analysis of the Hungarian WAIS. *Journal of Intelligence*, 7(3), 1–13. <https://doi.org/10.3390/jintelligence7030021>
- Scutari, M. (2009). Learning Bayesian networks with the bnlearn R package. *arXiv*. <https://doi.org/10.48550/arXiv.0908.3817>
- Scutari, M., & Denis, J.-B. (2021a). *Bayesian networks: with examples in R*. CRC press. <https://doi.org/10.1201/9780429347436>
- Scutari, M., & Denis, J.-B. (2021b). <https://doi.org/10.1201/9780429347436>. CRC press. <https://doi.org/10.1201/9780429347436>
- Shafer, A., & Dazzi, F. (2019). Meta-analysis of the Positive and Negative Syndrome Scale (PANSS) factor structure. *Journal of Psychiatric Research*, 115, 113–120. <https://doi.org/10.1016/j.jpsychores.2019.05.008>
- Stroup, T. S., McEvoy, J. P., Swartz, M. S., Byerly, M. J., Glick, I. D., Canive, J. M., McGee, M. F., Simpson, G. M., Stevens, M. C., & Lieberman, J. A. (2003). The National Institute of Mental Health Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) project: Schizophrenia trial design and protocol development. *Schizophrenia Bulletin*, 29(1), 15–31. <https://doi.org/10.1093/oxfordjournals.schbul.a006986>
- van Borkulo, C., Boschloo, L., Borsboom, D., Penninx, B. W. J. H., Waldorp, L. J., & Schoevers, R. A. (2015). Association of symptom network structure with the course of depression. *JAMA Psychiatry*, 72(12), 1219–1226. <https://doi.org/10.1001/jamapsychiatry.2015.2079>
- van Rooijen, G., Isvoranu, A.-M., Kruijt, O. H., van Borkulo, C. D., Meijer, C. J., Wigman, J. T., Ruhe, H. G., & de Haan, L. (2018). A state-independent network of depressive, negative and positive symptoms in male patients with schizophrenia spectrum disorders. *Schizophrenia Research*, 193, 232–239. <https://doi.org/10.1016/j.schres.2017.07.035>
- Weigel, L., Wehr, S., Galderisi, S., Mucci, A., Davis, J., Giordano, G. M., & Leucht, S. (2023). The Brief Negative Symptom Scale (BNSS): A systematic review of measurement properties. *Schizophrenia*, 9(1), 45. <https://doi.org/10.1038/s41537-023-00380-x>
- Williams, M. N. (2021). Levels of measurement and statistical analyses. *Meta-Psychology*, 5, 11–14. <https://doi.org/10.15626/MP.2019.1916>

Woods, S. W., Walsh, B. C., Powers, A. R., & McGlashan, T. H. (2019a). Reliability, validity, epidemiology, and cultural variation of the Structured Interview for Psychosis-risk Syndromes (SIPS) and the Scale Of Psychosis-risk Symptoms (SOPS). In H. Li, D. I. Shapiro, & L. J. Seidman (Eds.), *Handbook of attenuated psychosis syndrome across cultures: International perspectives on early identification and intervention* (pp. 85–113). https://doi.org/10.1007/978-3-030-17336-4_5

Woods, S. W., Walsh, B. C., Powers, A. R., & McGlashan, T. H. (2019b). Reliability, validity, epidemiology, and cultural variation of the Structured Interview for Psychosis-Risk Syndromes (SIPS) and the Scale of Psychosis-Risk Symptoms (SOPS). In H. Li, D. I. Shapiro, & L. J. Seidman (Eds.), *Handbook of attenuated psychosis syndrome across cultures: International perspectives on early identification and intervention* (pp. 85–113). Springer International Publishing. https://doi.org/10.1007/978-3-030-17336-4_5

Yong, A. G., & Pearce, S. (2013). A beginner's guide to factor analysis: Focusing on exploratory factor analysis. *Tutorials in Quantitative Methods for Psychology*, 9(2), 79–94. <https://doi.org/10.20982/tqmp.09.2.p079>