

On Formalizing Theoretical Expectations: Bayesian Testing of Central Structures in Psychological Networks

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Network theory has emerged as a popular framework for conceptualizing psychological constructs and mental disorders. Initially, network analysis was motivated in part by the thought that it can be used for hypothesis generation. Although the customary approach for network modeling is inherently exploratory, we argue that there is untapped potential for confirmatory hypothesis testing. In this work, we bring to fruition the potential of Gaussian graphical models for generating testable hypotheses. This is accomplished by merging exploratory and confirmatory analyses into a cohesive framework built around Bayesian hypothesis testing of partial correlations. We first present a motivating example based on a customary, exploratory analysis, where it is made clear how information encoded by the conditional (in)dependence structure can be used to formulate hypotheses. Building upon this foundation, we then provide several empirical examples that unify exploratory and confirmatory testing in psychopathology symptom networks. In particular, we (1) estimate exploratory graphs; (2) derive hypotheses based on the most central structures; and (3) test those hypotheses in a confirmatory setting. Our confirmatory results uncovered an intricate web of relations, including an order to edge weights within comorbidity networks. This illuminates the rich and informative inferences that can be drawn with the proposed approach. We conclude with recommendations for applied researchers, in addition to discussing how our methodology answers recent calls to begin developing formal models related to the conditional (in)dependence structure of psychological networks.

Keywords: Gaussian graphical model, Bayesian, hypothesis testing, bridge symptoms, exploratory research, confirmatory research

Introduction

Network theory has emerged as a popular framework in the social-behavioral sciences for analyzing psychological constructs (Cramer et al., 2012; Dalege, Borsboom, van Harreveld, & van der Maas, 2019; Epskamp, Maris, Waldorp, & Borsboom, 2018; McNally, 2016). The underlying rationale is that a group of observed variables, say, self-reported symptoms, form a dynamic system wherein they mutually influence and interact with one another (Borsboom, 2017; McNally et al., 2015). In networks, observed variables are called “nodes” and the featured connections between them are called “edges”. This work will focus on psychological networks in which the edges are undirected and represent conditional dependence between nodes representing symptoms of mental disorders, that is, pairwise relations between symptoms after controlling for all other symptoms.

This approach has led to powerful new insights into a range of mental disorders including obsessive compulsive disorder (OCD; McNally, Mair, Mugno, & Riemann, 2017), depression (Boschloo, van Borkulo, Borsboom, & Schoevers, 2016; Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016; Hoorelbeke, Marchetti, De Schryver, & Koster, 2016), anxiety (Beard et al., 2016), and posttraumatic stress disorder (PTSD; Afzali et al., 2017; Armour, Fried, Deserno, Tsai, & Pietrzak, 2017; Fried et al., 2018; McNally et al., 2015).

This surge of research stems from a shift away from the “common cause” perspective to the “network” perspective of mental disorders (Cramer, Waldorp, van der Maas, & Borsboom, 2010b; McNally, 2016). The key distinction lies in the assumptions of their respective statistical models. The latter uses network models that account for the mutual interactions between psychopathological symptoms (Borsboom, 2017; Borsboom & Cramer, 2013), whereas the former uses latent variable models that fail to capture mutual relationships between symptoms due to the assumption of “local independence” (Cramer, Waldorp, van der Maas, & Borsboom, 2010a, but see Bringmann & Eronen, 2018). There are also notable differences relating to their perceived pur-

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pose. For example, undirected networks are customarily estimated with a data-driven approach thought to be ideal for hypothesis generation (Epskamp & Fried, 2018; Epskamp, van Borkulo, et al., 2018). On the other hand, latent variable models have a long tradition of confirmatory hypothesis testing (e.g., Bentler, 1980). Although this distinction is commonplace, network modeling has untapped potential for confirmatory testing of conditional (in)dependencies.

Confirmatory testing with networks remains uncommon in part because edges are often thought to *merely* represent a causal skeleton (Borsboom, 2017; Borsboom & Cramer, 2013; Epskamp, van Borkulo, et al., 2018). Typically causality is associated with directionality, that is, say, $A \rightarrow B$, which implies that A causes B (e.g., Pearl, 2009). Estimating such a graph would require abandoning a partial correlation network. This is because the relations are inherently *undirected*. Hence, the notion of using networks to generate *causal* hypotheses perhaps implies that an alternative model is needed for confirmatory testing. This is not the case. Networks are an effective method to study pairwise relationships and can be used for confirmatory hypothesis testing (Epskamp, Rhemtulla, & Borsboom, 2017; Ryan, Bringmann, & Schuurman, 2019). In fact, approaches for estimating directed graphs of conditional dependencies (e.g., DAGs) are also inherently data-driven (e.g., Kalisch & Bühlmann, 2007). This is distinct from confirmatory testing, where the focus is on *a priori* expectations that allow for rich inference. Testing expectations is analogous to predicting the observed data — an important signature of a theory’s explanatory power.

A prerequisite for using networks in a confirmatory setting is having hypotheses to test. Here there is also untapped potential. For example, although hypothesis generation is commonly proposed as an advantage of network models (Cramer et al., 2012; Epskamp & Fried, 2018; Epskamp, Waldorp, Möttus, & Borsboom, 2018; Ryan et al., 2019), we are not aware of any examples in psychology that have actually formulated hypotheses to then test. The unrealized idea is to use a network estimated in an exploratory setting to generate hypotheses regarding, say, which nodes are most central in a system (Epskamp, van Borkulo, et al., 2018; Jones, Ma, & McNally, 2019; Robinaugh, Millner, & McNally, 2016). The main contribution of this work is bringing to fruition the idea of using networks to generate hypotheses for testing in a confirmatory setting.

In this work, we focus on testing hypotheses related to central nodes and the conditional (in)dependence structure therein. In psychopathology, special attention has been drawn to central nodes due to the idea that intervening on them would affect the rest of the network (Beard et al., 2016; McNally et al., 2015; Robinaugh et al., 2016). This idea implies the notion of causality, but in fact, centrality measures have been critiqued as poor indicators of causal influ-

ence (Dablander & Hinne, 2019). However, because centrality scores are summary statistics that describe an exploratory analysis they can be used to formulate confirmatory hypotheses. In particular, strength-based metrics (Jones et al., 2019; Newman, 2010) are useful for developing hypotheses related to the edge weights of central nodes. This is perhaps unsurprising, given that they can be calculated using a population parameter with a known distribution (e.g., a partial correlation, Fisher, 1924; Yule, 1897). Together, centrality indices provide untapped sources of information that can be used to narrow the focus on to particular aspects of an estimated network.

To test hypotheses related to the edge weights, we use recently proposed Bayesian methodology that readily allows for exploratory and confirmatory testing in partial correlation networks, or Gaussian graphical models (GGMs; Williams & Mulder, 2019). This approach facilitates a workflow wherein central nodes can be identified in an exploratory stage and hypotheses related to these nodes can then be tested in a confirmatory setting. A particular advantage of the confirmatory aspect is that hypotheses are expressed using (in)equality constraints on the parameters of interest and tested against competing theoretical expectations. For instance, one could test $\mathcal{H}_1 : \rho_{12} > \rho_{13} > \rho_{14} > 0$ against $\mathcal{H}_2 : \rho_{12} = \rho_{13} = \rho_{14} = 0$ (Hojtink, 2001; Hoijtink, Mulder, van Lissa, & Gu, 2019; Mulder, 2016). This provides a formal comparison between \mathcal{H}_1 , which states that there is an order to the edges weights, or effect sizes, and they are all positive, versus \mathcal{H}_2 , which expresses that they are all equal to zero. A major contribution of this work is to extend the idea of informative Bayesian testing to psychological networks, in addition to providing a comprehensive framework that can propel the field toward developing formal models (e.g. Borsboom, van der Maas, Dalege, Kievit, & Haig, 2020; Haslbeck, Ryan, Robinaugh, Waldorp, & Borsboom, 2019).

This work is organized as follows. We first give a concise overview of Gaussian graphical models. We then proceed to illustrate how hypotheses can be derived based on an exploratory network analysis. Here we show that the information encoded by the conditional (in)dependence structures can be used to formalize theoretical expectations. Next we provide an overview of the confirmatory strategy in this work, where the advantage of adopting a Bayesian approach for confirmatory testing is made clear. Specifically, the ability to directly compare theoretical models formulated through an exploratory analysis. We then discuss in detail how the proposed testing framework can be used in an applied setting. We conclude with a discussion on the proposed methods including limitations and recommendations.

The Gaussian Graphical Model

The Gaussian graphical model (GGM) encapsulates conditional relations among multivariate normal data. These

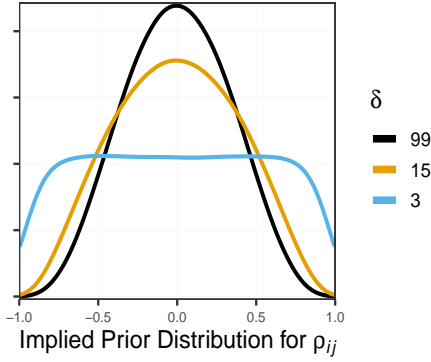


Figure 1. The implied marginal distribution for the encompassing prior on the partial correlations. The prior hyperparameter, δ , controls the standard deviation. Values of 99, 15, and 3 correspond to standard deviations of 0.10, 0.25, and 0.50, respectively.

relations are customarily visualized to infer the underlying dependence structure (i.e., the partial correlation “network”; Højsgaard, Edwards, & Lauritzen, 2012; Lauritzen, 1996). A GGM is an undirected graph that can be denoted by $G = (V, E)$, where $V = \{1, \dots, p\}$ is a vertex set and $E \subseteq V \times V$ is an edge set. V refers to the p “nodes” in the network, say, items on a depression scale, and E defines the estimated network structure. Let $\mathbf{y} = (y_1, \dots, y_p)^T$ be a vector of observed random variables that index the vertices in G , and assume it to be multivariate normal, $\mathbf{y} \sim \mathcal{N}_p(\boldsymbol{\mu}, \boldsymbol{\Sigma})$. Here $\boldsymbol{\mu}$ is a $p \times 1$ mean vector and $\boldsymbol{\Sigma}$ is a $p \times p$ positive definite covariance matrix.

Throughout the rest of this paper we will use \mathbf{Y} to denote the $n \times p$ data matrix, where each row corresponds to observations from an individuals. Without loss of information, we assume the data to be mean centered, that is, $\boldsymbol{\mu} = \mathbf{0}$. The undirected graph G is obtained by establishing the non-zero off-diagonal elements in the precision matrix, $\boldsymbol{\Theta} = \boldsymbol{\Sigma}^{-1}$. That is, $(i, j) \in E$ when nodes i and j are determined to be conditionally dependent and set to zero otherwise. The edges in a GGM correspond to partial correlations, ρ_{ij} , that is, the correlation between variables i and j after controlling for all other variables. These can be computed directly from the elements in $\boldsymbol{\Theta}$,

$$\rho_{ij} = \frac{-\theta_{ij}}{\sqrt{\theta_{ii}\theta_{jj}}}, \quad (1)$$

Thus, partial correlation networks can be estimated by testing each relation in (1) (Drton & Perlman, 2004; Williams & Mulder, 2019). The resulting elements can then be used to construct the $p \times p$ adjacency matrix, \mathbf{A}^{CD} , that is,

$$\mathbf{A}_{ij}^{CD} = \begin{cases} 1 & \text{if } \rho_{ij} \neq 0 \\ 0 & \text{otherwise} \end{cases}, \quad (2)$$

where $\text{diag}(\mathbf{A}^{CD}) = 0$ and CD denotes conditional dependence. The position of 1’s for a given node j correspond to its “neighborhood” (Meinshausen & Bühlmann, 2006) and encodes the conditional dependence structure.

Note that this formulation for GGMs does not typically allow for statements with respect to conditional independence, that is, one cannot ascertain whether two nodes have *no* relationship conditional on all other nodes. This can be seen in (2), where those partial correlations not detected are *merely* set to 0. However, directly testing the null hypothesis of conditional independence is possible by employing Bayesian methodology (Mulder, 2016; Williams & Mulder, 2019). This is especially relevant for this work, because conditional independence is a hypothesis that can be formally evaluated in a confirmatory setting. For example, it is possible to determine if a set of symptoms in a network are independent. To this end, we follow Williams and Mulder (2019) and define an additional $p \times p$ adjacency matrix, \mathbf{A}^{CI} ,

$$\mathbf{A}_{ij}^{CI} = \begin{cases} 1 & \text{if } \rho_{ij} = 0 \\ 0 & \text{otherwise} \end{cases}, \quad (3)$$

where $\text{diag}(\mathbf{A}^{CI}) = 0$ and CI denotes conditional independence. The position of 1’s encode the conditional independence structure. Note that both (2) and (3) are determined with Bayesian hypothesis testing. The key insight here is that there are two adjacency matrices that can be used to generate and then test hypotheses.

Formalizing Theoretical Models

Psychological theories can be expressed as hypotheses with constraints on the parameters of interest (Hoijtink, 2011). This translates into thinking of theories in terms of constraints among conditional (in)dependencies. In a GGM, for example, it may be expected that a set of partial correlations are approximately equal to each other, larger or smaller than another set of partial correlations, or larger or smaller than a constant (typically zero). These kinds of hypotheses can be derived from theory or an exploratory analysis. This work focuses on the latter. Here the goal is not to determine the graph (e.g., McNally et al., 2017), but rather the structure of interrelations among partial correlations.

A major hurdle to confirmatory testing in networks has been the lack of available methods. Recently, however, a Bayes factor approach was introduced specifically for this purpose (Williams & Mulder, 2019; Williams, Rast, Pericchi, & Mulder, 2020). This opens the door for testing hypotheses

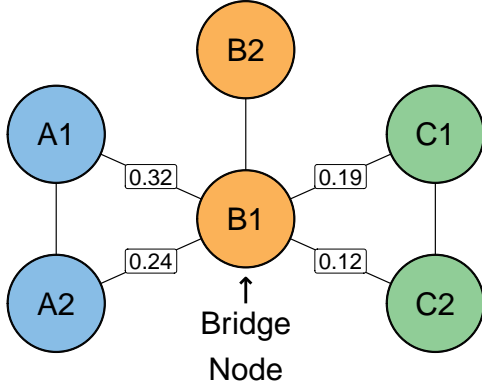


Figure 2. Example network with three communities: A, B, and C. Lines between two nodes indicates conditional dependence. It can be seen that B1 is a central node which bridges community B to communities A and C. Hypotheses can then be gleaned from the graph. For example, $\mathcal{H}_1 : \rho_{B1-A1} > \rho_{B1-A2} > \rho_{B1-C1} > \rho_{B1-C2} > 0$ or $\mathcal{H}_2 : (\rho_{B1-A1}, \rho_{B1-A2}, \rho_{B1-C1}, \rho_{B1-C2}) = 0$. The former tests the order of bridge edges and constrains them to be positive. The latter tests whether B1 is conditionally independent of nodes outside the B community. This captures how network structures encode information that can be used to formalize and test a model.

not currently possible with classical statistics (i.e., p -values). For instance, there is theoretical interest in characterizing central structures involving bridge nodes, or nodes that connect to multiple communities (i.e., clusters) within a network (c.f., [Castro et al., 2019](#); [Cramer et al., 2010a, 2010b](#); [Jones et al., 2019](#)). These nodes can be identified through visualizing a network (e.g., [Beard et al., 2016](#)) or bridge centrality metrics ([Jones et al., 2019](#)). For example, by inspecting Figure 2, it is possible to formulate hypotheses relating to the order of edges or effect sizes within (or between) clusters, that is,

$$\mathcal{H}_1 : \rho_{B1-A1} > \rho_{B1-A2} > \rho_{B1-C1} > \rho_{B1-C2} > 0 \quad (4)$$

$$\mathcal{H}_2 : (\rho_{B1-A1}, \rho_{B1-A2}, \rho_{B1-C1}, \rho_{B1-C2}) = 0$$

$$\mathcal{H}_3 : \text{"not } \mathcal{H}_1 \text{ or } \mathcal{H}_2."$$

In (4), \mathcal{H}_1 captures the order of edges in Figure 2. Substantively, this hypothesis can be interpreted as capturing the order of importance (defined by effect size) for the bridging relations that connect clusters of nodes in a network. Furthermore, there is an additional constraint that all of the edges are positive. This reflects the expectation of a positive manifold that has a central role in network theory ([Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011](#)). \mathcal{H}_2 then tests whether all the nodes are actually conditionally independent,

which also implies that there is no inherent ordering. Finally, \mathcal{H}_3 captures some yet to be hypothesized structure of relations. These hypotheses are formal models that can be evaluated. That is, one can directly quantify support for \mathcal{H}_1 versus \mathcal{H}_2 with a Bayes factor, a measure of relative support between competing hypotheses ([Kass & Raftery, 1995](#)). This demonstrative example captures the guiding idea of this work: network structures (e.g., Figure 2) encode information that can be used to formalize and test models.

Testing Strategy

We use the testing strategy in [Williams and Mulder \(2019\)](#) for confirmatory analyses. In this approach, hypotheses are expressed as (in)equality constraints on partial correlations. For example, \mathcal{H}_1 and \mathcal{H}_2 in (4) were expressed using inequality and equality constraints, respectively, on the edges in Figure 2. The evidence for such hypotheses can be quantified with the Bayes factor — a measure of relative support between competing hypotheses or models. In matrix notation, order constrained hypotheses can be written as

$$\mathcal{H}_t : \mathbf{R}_t \boldsymbol{\rho} > \mathbf{r}_t, \quad (5)$$

where $t = 1, \dots, T$ denotes the competing hypotheses. In (5), $[\mathbf{R}_t | \mathbf{r}_t]$ is an augmented matrix that specifies the constraints under \mathcal{H}_t . In reference to (4), the system of inequalities under, say, \mathcal{H}_1 , are formulated as

$$\mathbf{R}_{\mathcal{H}_1} \boldsymbol{\rho} = \begin{bmatrix} 1 & -1 & 0 & 0 \\ 0 & 1 & -1 & 0 \\ 0 & 0 & 1 & -1 \end{bmatrix} \begin{bmatrix} \rho_{B1-A1} \\ \rho_{B1-A2} \\ \rho_{B1-C1} \\ \rho_{B1-C2} \end{bmatrix} > \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix}, \quad (6)$$

where $\mathbf{R}_{\mathcal{H}_1}$ denotes a matrix containing the coefficients for the contrasts of interest. Bayes factors can then be computed using the encompassing prior approach ([Klugkist, Kato, & Hoijtink, 2005](#)). The idea is to first specify an unconstrained (or encompassing) distribution for a hypothesis, \mathcal{H}_u , that does not place constraints on the partial correlations. This corresponds to an unconstrained network where theoretical expectations are not incorporated. The encompassing prior for GGMs is specified for the precision matrix, $\boldsymbol{\Theta}$, as a matrix- F distribution ([Mulder & Pericchi, 2018](#)). The implied marginal prior for the partial correlations is then

$$\rho_{ij} \sim \text{beta}\left(\frac{\delta}{2}, \frac{\delta}{2}\right) \text{ on } (-1, 1), \quad (7)$$

where δ is a prior hyperparameter that controls the standard deviation. The prior distribution for different values of δ can be seen in Figure 1. Note that it is not possible to place a beta prior on each ρ_{ij} directly because the resulting joint prior distribution for the partial correlation matrix would not be

positive definite (for technical details see [Mulder & Pericchi, 2018](#); [Williams & Mulder, 2019](#)).

The prior distributions under the constrained hypotheses are then obtained by truncating the encompassing prior according to the imposed constraints. Thus, instead of having to formulate T separate priors, only the unconstrained prior needs to be formulated. Furthermore, due to the encompassing prior approach, the Bayes factor of each constrained hypothesis against the unconstrained hypothesis \mathcal{H}_u is straight forward to obtain, that is,

$$\text{BF}_{tu} = \frac{\Pr(\boldsymbol{\rho} \in \Omega_t | \mathbf{Y}, \mathcal{H}_u)}{\Pr(\boldsymbol{\rho} \in \Omega_t | \mathcal{H}_u)}, \quad (8)$$

where Ω_t denotes the subspace under a constrained hypothesis \mathcal{H}_t that satisfies the constraints on $\boldsymbol{\rho}$. In (8), the posterior probability in the numerator and the prior probability in the denominator can be understood as measures of ‘relative fit’ and ‘relative complexity’ of \mathcal{H}_t relative to \mathcal{H}_u , respectively ([Mulder, 2014](#)). Once (8) has been obtained for all constrained hypotheses of interest, the Bayes factors between them can be computed using the transitivity property of the Bayes factor. For example, $\text{BF}_{12} = \frac{\text{BF}_{1u}}{\text{BF}_{2u}}$ provides the relative evidence in favor of \mathcal{H}_1 . If we had, say, $\text{BF}_{12} = 5$, this would indicate the observed partial correlations are five times more likely under \mathcal{H}_1 than \mathcal{H}_2 . Importantly, Bayes factors can also be viewed as measuring the relative success at predicting the observed data ([Kass & Raftery, 1995](#)). Once computed, Bayes factors can be used to obtain posterior model probabilities, that is, the probability that a hypothesis t is true given the data ([Mulder, 2016](#)). Assuming that all models have equal prior probabilities (i.e., $\frac{1}{T}$), the posterior probabilities for the $t = 1, \dots, T$ hypotheses under consideration are given by

$$\Pr(\mathcal{H}_t | \mathbf{Y}) = \frac{\text{BF}_{tu}}{\sum_{t'=1}^T \text{BF}_{t'u}}. \quad (9)$$

Whereas Bayes factors reflect the relative probability of the data under two hypotheses, posterior probabilities reflect relative support for a set of hypotheses given the data.

Summary

In this section we described a framework wherein Gaussian graphical models are used for both exploratory and confirmatory analyses. There are two aspects of this approach worth emphasizing. First, it allows for flexible testing of constraints in psychological networks. This readily allows for comparing theoretical models. For example, even for the relatively simple hypothesis in (4), testing whether the partial correlations are all greater than zero formally expressed the theoretical expectation of a positive manifold. Second, we demonstrated that the underlying network structure from

an exploratory analysis encodes the necessary ingredients to generate hypotheses (e.g., Figure 2). This is a central idea of network analysis. The critical distinction is that we are presenting a comprehensive approach for formalizing and testing hypotheses generated from an exploratory analysis.

Empirical Applications

We now discuss in further detail how exploratory and confirmatory approaches can work in tandem to test hypotheses related to central structures. Recall that one motivation for network analysis was to generate hypotheses in an exploratory setting, and, in turn, a primary goal of this work is to bring this idea to fruition. To this end, we take on the perspective of a network researcher that formulates hypotheses based on an initial exploratory analysis and then tests them in a confirmatory setting. Note that Bayesian testing provides both the conditional dependence structure, \mathbf{A}^{CD} , and the conditional independence structure, \mathbf{A}^{CI} , which further opens the door for novel insights.

As mentioned above, there is theoretical and clinical interest in characterizing central structures involving bridge nodes ([Castro et al., 2019](#); [Cramer et al., 2010a, 2010b](#); [Jones et al., 2019](#)). This is because bridge symptoms are thought to drive the co-occurrence of symptoms between communities and serve as targets for intervention ([Beard et al., 2016](#); [McNally et al., 2017](#)). Thus, we focus on testing hypotheses related to bridge symptoms. To identify bridge symptoms, we rely on bridge strength rather than visual inspection. This is for two reasons. First, a node’s bridge strength is defined as the absolute sum of its inter-community edges, and therefore, highlights larger effects ([Jones et al., 2019](#)). This brings in to focus central structures (i.e., a subsystem) on which hypotheses can be formulated. Second, the Bayesian framework we use places prior distributions on the partial correlations (Equation 7). With the exception of bridge expected influence, bridge strength is the only bridge statistic that accounts for these parameters¹.

Single Disorder

In the following, we estimate a GGM of PTSD symptoms and demonstrate how bridge strength can be used to identify central structures for which hypotheses can be formulated. In several examples, we discuss how to test these hypotheses in an independent dataset. We use data reported in [Fried et al. \(2018\)](#). Specifically, we use two samples of patients receiving treatment for PTSD ($n = 926$ and $n = 956$; Samples 3 and 4 in Table 1 in [Fried et al. \(2018\)](#)). The presence and severity of PTSD symptoms were assessed using the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders*

¹Bridge expected influence is identical to bridge strength but does not take the absolute value of edges before summing them ([Jones et al., 2019](#))

(*DSM-IV*, American Psychiatric Association, 1994). Each of the 16 symptoms ($p = 16$) belonged to one of three communities (Re-experiencing, Avoidance, and Arousal).

Exploratory Analysis. We began by estimating the network structures \mathbf{A}^{CD} and \mathbf{A}^{CI} with the R package **BGGM** (Williams & Mulder, 2019) (panels A and B in Figure 3). Recall that there is strong theory in the network literature that expects all relations to be positive (i.e., a positive manifold, Borsboom et al., 2011; Horn & Cattell, 1966). This was formally incorporated into the analysis with a one-sided hypothesis test, $\mathcal{H}_0 : \rho_{ij} = 0$ versus $\mathcal{H}_1 : \rho_{ij} > 0$, for each partial correlation in the network (see The Gaussian Graphical Model). Hence, \mathbf{A}^{CD} includes only positive relations. Because this analysis was used to formulate confirmatory hypotheses, we “erred on the side of discovery” (Bem, 2004) and used a Bayes factor threshold of three (this is considered “moderate” evidence, Lee & Wagenmakers, 2013) to determine the network structures.

With the network structures in hand, we proceeded to identify central nodes as indicated by bridge strength with the R package **networktools** (Jones et al., 2019). This is also the customary approach in network analysis, where, for example, the most central nodes are identified after estimating the structure (e.g., Beard et al., 2016; McNally et al., 2017). The results indicated that D1 (“sleep problems”, bridge strength = 0.65) and B4 (“disinterest in activities”, bridge strength = 0.53) were the most central nodes (see Table A1 for full definitions). The neighborhood of bridge relations for both nodes can be seen in Figure 3 (panel A).

Confirmatory Analysis. We emphasize that centrality indices summarize an inherently exploratory analysis and only provide information from afar. For example, it is possible to have a top ranking bridge symptom emerge in two datasets with completely different bridging structures. Our approach extends the utility of bridge centrality metrics to confirmatory testing by using them to formulate hypotheses on the most central symptoms in the network. We thus focus on node D1 (“sleep problems”) and node B4 (“disinterest in activities”). Figure 3 (panel C and D) zooms in on these top ranking bridge symptoms and their respective neighborhoods of bridge relations. The key idea is that honing into central symptoms allows researchers to easily formulate hypotheses of substantive or theoretical importance.

Varying degrees of replication. The topic of replicability has recently captivated the network literature (Forbes, Wright, Markon, & Krueger, 2017; Fried et al., 2018; Williams, 2020). To assess replicability, it is common to focus on individual edges with either classical (van Borkulo et al., 2016) or Bayesian testing (Williams et al., 2020). Although the latter has the advantage of directly providing evidence for equality of partial correlations, it is possible to ask even more fine-grained questions about replication. For example, to what degree do central structures replicate? This

can be expressed with formal models.

We first focused on node B4 (Figure 3, panel D) and tested the following hypotheses

$$\mathcal{H}_1 : (\rho_{B4-C1}, \rho_{B4-C7}, \rho_{B4-D3}, \rho_{B4-D4}) > 0 \quad (10)$$

$$\mathcal{H}_2 : \rho_{B4-C1} > (\rho_{B4-C7}, \rho_{B4-D3}, \rho_{B4-D4}) > 0$$

$$\mathcal{H}_3 : \text{“not } \mathcal{H}_1 \text{ or } \mathcal{H}_2\text{.”}$$

In (10), \mathcal{H}_1 is testing for replication of all edges but is otherwise agnostic towards the interplay among bridge relations. \mathcal{H}_2 then provides a refined view into the bridge neighborhood by testing an additional constraint that the strongest edge replicated. That is, all of the bridge relations *and* the strongest edge re-emerged in an independent dataset. Furthermore, \mathcal{H}_1 and \mathcal{H}_2 both reflect a positive manifold. We also included \mathcal{H}_3 which accounts for structures that are not \mathcal{H}_1 or \mathcal{H}_2 . We compared the first two hypotheses against \mathcal{H}_3 , where there was strong evidence for both \mathcal{H}_1 ($\text{BF}_{13} = 33.4$) and \mathcal{H}_2 ($\text{BF}_{23} = 120.4$). Hence, the data were more likely under the replication models than a model that did not include replication-based constraints. We then compared \mathcal{H}_1 to \mathcal{H}_2 . Although the evidence was not strong, the data were more likely under \mathcal{H}_2 ($\text{BF}_{21} = 3.6$). This analysis indicates that (1) the bridge relations replicated in an independent dataset; and (2) the relation between “sleep problems” (node B4) and “avoidance of thoughts” (node C1) *could* be the strongest bridge between the Re-experiencing and Avoidance communities.

We then focused on node D1 (Figure 3, panel D) and tested the following hypotheses

$$\mathcal{H}_1 : (\rho_{D1-C2}, \rho_{D1-C6}) > 0 \quad (11)$$

$$\mathcal{H}_2 : (\rho_{D1-C2}, \rho_{D1-C6}) < 0$$

$$\mathcal{H}_3 : (\rho_{D1-C2}, \rho_{D1-C6}) = 0.$$

In (11), the hypotheses are in relation to the Avoidance community and they again reflect network replication. For example, \mathcal{H}_1 expresses that both relations are positive, but does not impose an order restriction among bridge edges, whereas \mathcal{H}_2 expresses that both relations are negative and similarly does not impose an order restriction. Alternatively, \mathcal{H}_3 then captures the importance of ruling out conditional independence or that the effects are actually zero. Although the data were more likely under \mathcal{H}_1 than \mathcal{H}_2 ($\text{BF}_{12} = 12.3$), the evidence favored \mathcal{H}_3 over \mathcal{H}_1 ($\text{BF}_{31} = 11$). In other words, of the formal, replication models, there was evidence for null associations, or that these might *not* be bridge relations after all.

The same hypotheses in (11) were tested in relation to the Re-experiencing community. In this case, there was overwhelming evidence for \mathcal{H}_1 . The posterior hypothesis probability was essentially 1, which translates into an infinite

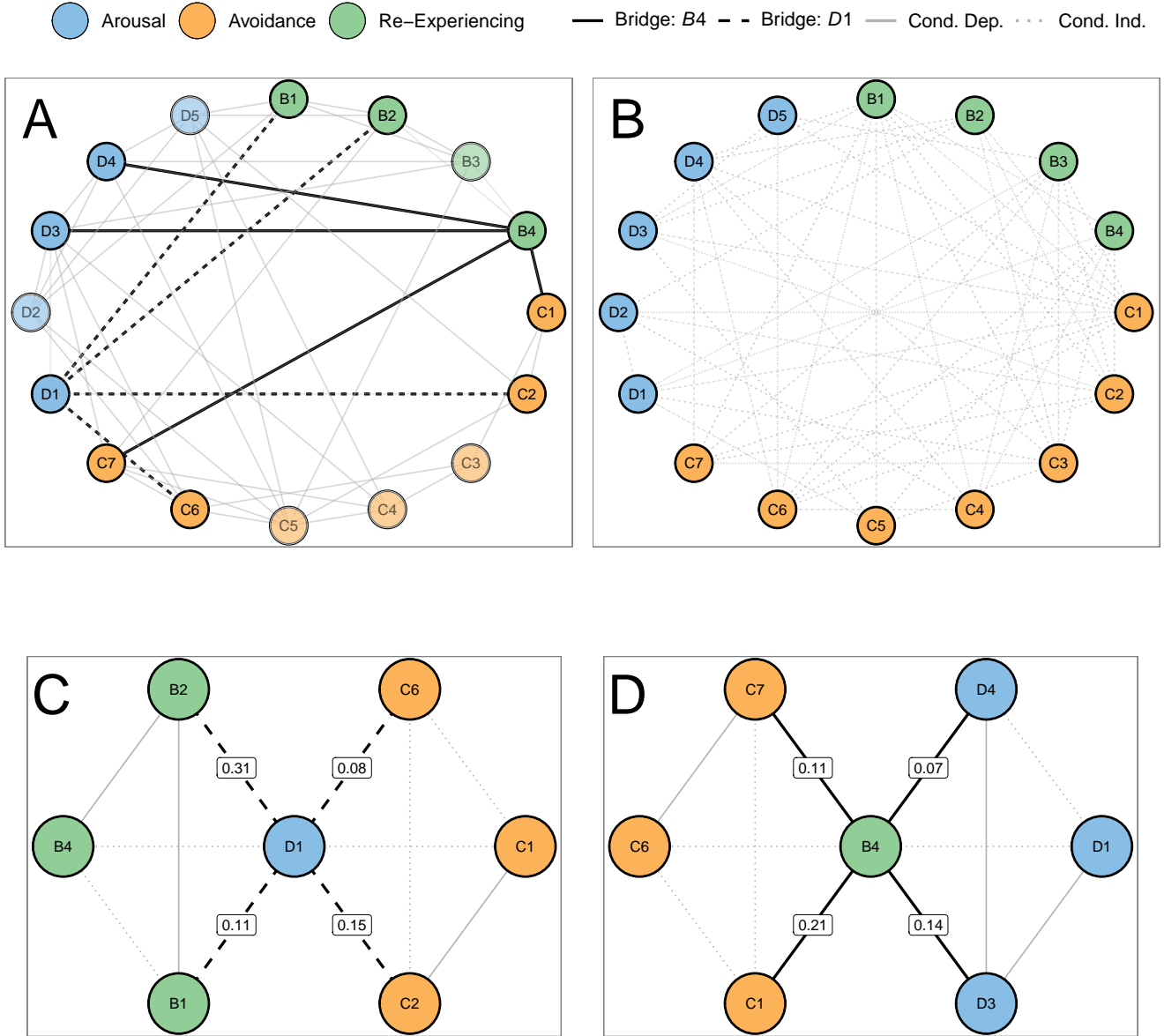


Figure 3. Exploratory network structure of PTSD symptoms. D1 (“sleep problems” and B4 (“disinterest in activities”) emerged as bridge nodes. **(A)** The conditional dependence structure. Lines between two nodes indicate an association between them after controlling for all other nodes. This structure encodes information for testing associations such as $\mathcal{H}_1 : (\rho_{D1-C2}, \rho_{D1-C6}) > 0$. **(B)** The conditional independence structure. Dotted lines indicate that there is no association between two nodes after controlling for all other nodes. This structure encodes information for testing null associations such as $\mathcal{H}_1 : (\rho_{B4-C6}, \rho_{D1-B4}, \rho_{D1-C1}) = 0$. **(C & D)**. A magnified look at the neighborhood of bridge relations for D1 and B4. We extracted the information encoded in these structures to formulate and test hypotheses (see Equations 10, 11, and 12).

Bayes factor. This indicates that the bridge relations replicated for the Arousal and Re-experiencing communities, and demonstrates the utility of comparing formal models for clinical applications in particular. Although network analyses are often thought to identify target symptoms for interventions (e.g., [Beard et al., 2016](#)), we are not aware of any work that has followed up an initial, exploratory analysis, with the goal of confirming hypotheses related to the potential targets. In this case, the results suggest that the symptom “sleep problems” may be useful in guiding interventions.

Ruling Out Bridges. It is important to rule out bridge relations in establishing the structure of inter-community relations. Here, the question of replication is concerned with null associations re-emerging in an independent dataset. To show this, we included two null associations in both panel C and D of Figure 3 (the dotted lines). We thus formulated the following hypotheses

$$\begin{aligned}\mathcal{H}_1 : (\rho_{B4-C6}, \rho_{D1-B4}, \rho_{D1-C1}) &= 0 \\ \mathcal{H}_2 : (\rho_{B4-C6}, \rho_{D1-B4}, \rho_{D1-C1}) &> 0 \\ \mathcal{H}_3 : \text{“not } \mathcal{H}_1 \text{ or } \mathcal{H}_2\text{.”}\end{aligned}\quad (12)$$

which represent a null model (\mathcal{H}_1), a positive manifold model (\mathcal{H}_2), and a model accounting for alternative structures (\mathcal{H}_3). The positive manifold model had a posterior hypothesis probability of essentially zero, indicating that positive associations can be ruled out. Further, the data were more likely under the conditional independence model, \mathcal{H}_1 , than under \mathcal{H}_3 ($\text{BF}_{13} = 4.7$)². This is striking because these very same relations have large bivariate correlations, yet, after controlling for the other symptoms in the network, there was evidence for conditional independence.

Multiple Disorders

Here we provide further examples our proposed framework using a comorbidity network. We estimate a GGM containing anxiety and depression symptoms and use bridge strength to identify central structures for which hypotheses can be formulated. In several examples, we discuss how to test these hypotheses in an independent dataset. We use data from [Beard et al. \(2016\)](#) that includes 16 symptoms gathered from 1029 patients receiving treatment for depression and anxiety. Symptoms were assessed using the Patient Health Questionnaire-9 ([Kroenke, Spitzer, & Williams, 2001](#)) and the 7-item Generalized Anxiety Disorder Scale ([Spitzer, Kroenke, Williams, & Löwe, 2006](#)). Nine symptoms were in the “depression” community and seven symptoms were in the “anxiety” community. Because only one dataset was available, we performed exploratory analyses on one half of the data, and used the other half for confirmatory testing (i.e., “data splitting”; [Dahl, Grotle, Šaltytė Benth, & Natvig, 2008; Faraway, 1995](#)).

Exploratory Analysis. We followed the same procedure as above: (1) estimate the conditional dependence and independence structures; (2) identify the top scoring bridge symptoms; and (3) formulate hypotheses based on the results. The results indicated that node D8 (“motor”, bridge strength = 0.40)³ and node D6 (“guilt”, bridge strength = 0.27) were the most central according to bridge strength (see Table A2 for full definitions). Figure 4 displays the resulting (in)dependence structures (panels A and B) and the magnified neighborhood of bridge relations for nodes D8 and D6 (panel C).

Confirmatory Analysis. We reiterate that our confirmatory testing approach builds upon identifying bridge symptoms in an exploratory analysis to gain insights regarding central structures of a network. This can be done by developing hypotheses targeting the most central nodes as determined by centrality statistics (in this case bridge strength). Accordingly, we have magnified the neighborhood of bridge relations for nodes D8 and D6 (panel C Figure 4). This readily allows for devising hypotheses with substantive and theoretical relevance. Of course, an important first step is to investigate the extent to which the relations replicate. Note that we are not referring to simply detecting the effect, but testing the constrained models implied from the exploratory analysis.

Intra- and Inter-Bridge Sets. In addition to testing whether the edges for a bridge symptom simply re-emerge in a new dataset, it may be useful to test whether their exact order replicates. If the order of edges is known, and assuming a useful focal point is the strongest relation, this would imply an ordering among possible intervention targets. This notion is encoded in the exploratory analysis (panel C in Figure 4) which leads to the following hypotheses

$$\begin{aligned}\mathcal{H}_1 : \rho_{D8-A5} = \rho_{D8-A7} &> (\rho_{D6-A3}, \rho_{D6-A6}) > 0 \\ \mathcal{H}_2 : \rho_{D8-A5} > \rho_{D8-A7} &> \rho_{D6-A3} > \rho_{D6-A6} > 0 \\ \mathcal{H}_3 : \text{“not } \mathcal{H}_1 \text{ or } \mathcal{H}_2\text{.”}\end{aligned}\quad (13)$$

In (13), the hypotheses focus on characterizing bridge sets, or the set of bridge edges belonging to a given symptom. For example, \mathcal{H}_1 posits that the bridge set for node D8 (“motor”) is collectively greater than the set for node D6 (“guilt”), with constraint that the edges for node D8 are equal to each other. This effectively corresponds to testing whether node D8 has greater bridge strength than node D6. \mathcal{H}_2 then refines \mathcal{H}_1 by testing an exact order both between and within bridge sets. The data were more likely under both \mathcal{H}_1 ($\text{BF}_{13} = 4.4$) and

²Changing the prior distribution resulted in *more* support for \mathcal{H}_1 . This suggests $\text{BF}_{13} = 4.7$ is a lower bound for the evidence in favor of the null model.

³“motor” refers to physical lethargy or restlessness ([Kroenke et al., 2001](#))

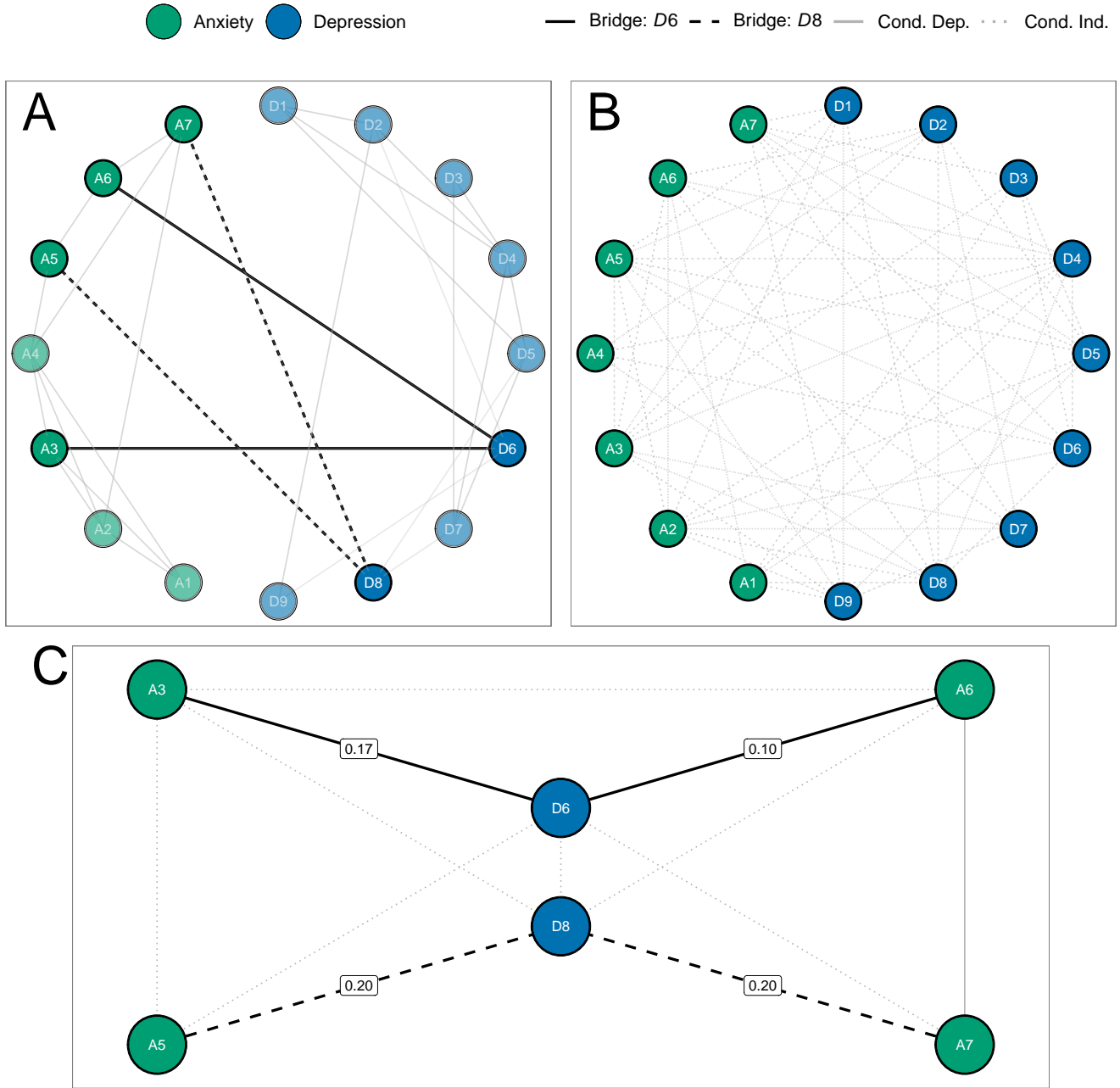


Figure 4. Exploratory network structure of depression and anxiety symptoms. D6 (“guilt”) and D8 (“motor”) emerged as bridge nodes. **(A)** The conditional dependence structure. Lines between two nodes indicate an association between them after controlling for all other nodes. **(B)** The conditional independence structure. Dotted lines indicate that there is no association between two nodes after controlling for all other nodes. **(C)**. A magnified look at the neighborhood of bridge relations for D6 and D8. We extracted the information encoded in these structures to formulate and test hypotheses (see Equations 13, 14, and 15).

\mathcal{H}_2 ($\text{BF}_{23} = 107$) than \mathcal{H}_3 . Furthermore, there was more evidence supporting the hypothesis testing solely inequality constraints, \mathcal{H}_2 , than the one including an equality constraint ($\text{BF}_{21} = 24.1$). This provides a clear characterization of the the bridge relations at hand — not only did the order of bridge strength replicate, but so did the order of the edges within the neighborhood of each bridge symptom. In this confirmatory test, the relationship between the depression symptom “motor” and anxiety symptom “restless” emerged as the top relation. This characterizes a central structure between anxiety and depression and can inform theory development with respect to these disorders.

Bridge Set Separation. It may further be of interest to identify whether bridge sets include common elements. That is, whether bridge symptoms connect to the same or different nodes. This may be useful in understanding whether bridge symptoms represent distinct central structures. As can be seen in panel C of Figure 4, the bridge sets for nodes D8 and D6 are mutually exclusive. This implies that there are two subsystems. Keeping this in mind, we formulated the following set of hypotheses

$$\begin{aligned}\mathcal{H}_1 : (\rho_{D8-A3}, \rho_{D8-A6}) &= 0 \\ \mathcal{H}_2 : (\rho_{D8-A3}, \rho_{D8-A6}) &> 0 \\ \mathcal{H}_3 : \text{“not } \mathcal{H}_1 \text{ or } \mathcal{H}_2\text{.”}\end{aligned}\quad (14)$$

In (14), \mathcal{H}_1 tests conditionally independent associations between the symptom “motor” and the bridge set for “guilt” (i.e., nodes A3 and A6) versus \mathcal{H}_2 , a positive manifold model, and \mathcal{H}_3 , a model accounting for alternative structures. Although the data were more likely under \mathcal{H}_1 than \mathcal{H}_3 ($\text{BF}_{13} = 3.7$), there was support in favor of \mathcal{H}_2 compared to \mathcal{H}_1 ($\text{BF}_{21} = 2$). This analysis suggests there is a small amount of evidence that “motor” has conditional dependent relations with the same nodes as “guilt”.

We repeated the hypothesis tests in (14) for “guilt”. Here, the findings differed slightly — the data were almost equally likely under \mathcal{H}_1 compared to \mathcal{H}_3 ($\text{BF}_{13} = 1.6$). Like above, however, the data were more likely under \mathcal{H}_2 than either \mathcal{H}_1 ($\text{BF}_{21} = 6.3$) or \mathcal{H}_3 ($\text{BF}_{23} = 10$). These analyses indicate that the null associations did not replicate, and instead support the idea that “motor” and “guilt” connect to the same symptoms. This information suggests, for example, that the nodes in panel C of Figure 4 make up a single central structure instead of two.

Bridge Node Separation. Thus far we have focused on testing multiple relationships simultaneously. While testing joint hypotheses is a key feature to our proposed testing strategy, it may be that a single parameter is of particular interest, say, due to theoretical importance. In this case, it is highly informative to test hypotheses focused on a single parameter. For example, panel C (Figure 4) indicates that nodes D8 and D6 are conditionally independent. However, this is in

contrast to what might be expected from two symptoms in the same community. Accordingly, one can test a hypothesis focused solely on this relationship, for example

$$\begin{aligned}\mathcal{H}_1 : \rho_{D8-D6} &= 0 \\ \mathcal{H}_2 : \rho_{D8-D6} &> 0 \\ \mathcal{H}_3 : \text{“not } \mathcal{H}_1 \text{ or } \mathcal{H}_2\text{.”}\end{aligned}\quad (15)$$

In (15), \mathcal{H}_1 tests a null association and \mathcal{H}_2 expresses a positive relationship. Though the data were more likely under \mathcal{H}_3 than \mathcal{H}_2 ($\text{BF}_{32} = 5.4$), there was more evidence in favor of \mathcal{H}_1 than \mathcal{H}_3 ($\text{BF}_{13} = 2.2$). Hence, there is some evidence in this confirmatory test that “motor” and “guilt” are conditionally independent symptoms. Importantly, focused hypotheses, such as in (15), can be used to draw powerful inferences with respect to relationships of particular interest.

Simulation Studies

Thus far we have provided a comprehensive framework for exploratory and confirmatory testing of central structures in partial correlation networks. Our hope is that researchers will integrate the proposed methods into their own work. Therefore, it is important to understand how these methods behave under certain conditions. To this end, we emphasize a few important points:

1. Bayes factors tend to infinity and posterior model probabilities tend to one in favor of the correct model with increasing sample size (O’Hagan, 1995). Although this property assumes the *true* model is being considered, recall that Bayes factors can also be interpreted as a measure for the relative success of predicting the observed data (Kass & Raftery, 1995). This perspective does not rely on the existence of a true model and is our preferred interpretation.
2. More specific hypotheses result in higher degrees of evidence, given that they are supported by the data. This is due to being relatively less ‘complex’ and having relatively better ‘fit’ (e.g., Klugkist et al., 2005; Mulder, 2014). This was observed in (10), where the data were most likely under two replication-based hypotheses, but of these two, the more specific one yielded stronger evidence.
3. The scale, or standard deviation, of the encompassing prior distribution influences the outcome when testing equality constrained hypotheses, but not when testing inequality constraints (i.e., the Jeffreys-Lindley paradox is not an issue, Mulder, 2014). This was seen in (12), where changing the prior distribution changed the evidence in support for \mathcal{H}_1 , an equality constrained hypothesis. This is important to consider when testing

confirmatory analyses because the resulting evidence can be considered objective for inequality constrained hypotheses⁴. That is, the Bayes factor is robust to the prior distribution.

We further examine these properties in two simulation studies. In each, data were generated with four variables and the true precision matrix

$$\Theta = \begin{bmatrix} 1 & -0.10 & -0.14 & -0.18 \\ -0.10 & 1 & -0.22 & -0.26 \\ -0.14 & -0.22 & 1 & -0.3 \\ -0.18 & -0.26 & -0.30 & 1 \end{bmatrix}. \quad (16)$$

The partial correlations then correspond to the off-diagonal elements of Θ with the sign reversed. These values were based on the most common partial correlations we observed in our analyses. We then tested hypotheses based Θ and computed posterior model probabilities. The first simulation study examines the advantage of testing precise hypotheses and the second evaluates the influence of the prior scale when testing inequality constrained hypotheses.

Study 1: Specific Hypotheses

In this study, we investigate posterior model probabilities for true hypotheses that have a different number of constraints. The idea is that more specific hypotheses (i.e., more constraints are placed) have smaller prior probabilities than less specific ones under the encompassing prior (see Equation 8). If a more specific model is supported by the data, this will result in a larger Bayes factor, and accordingly, a larger posterior probability. Thus, there is a greater “reward” for formulating and testing specific hypotheses on central structures. We formulated $s = 1, 2, 3$ sets of hypotheses with $t = 1, 2, 3$ hypotheses in each. The *true* hypotheses corresponded to

$$\begin{aligned} \mathcal{H}_1^1 &: \rho_{23} > \rho_{14} > \rho_{13} > \rho_{12} \\ \mathcal{H}_1^2 &: \rho_{24} > \rho_{23} > \rho_{14} > \rho_{13} > \rho_{12} \\ \mathcal{H}_1^3 &: \rho_{34} > \rho_{24} > \rho_{23} > \rho_{14} > \rho_{13} > \rho_{12}. \end{aligned} \quad (17)$$

In (17), \mathcal{H}_t^s denotes hypothesis t in set s . Each ρ_{ij} is the partial correlation corresponding to the element in the i th row and j th column of Θ in (16). The number of constraints for the true hypotheses imply different prior proportions in agreement with the unconstrained parameter space, Ω_t , such that more constraints result in smaller prior proportions. For the hypotheses in (17), the proportions in agreement with Ω_t are .04, .008, and .001, respectively. Each \mathcal{H}_t^s was compared to a null and complement hypothesis. For example, \mathcal{H}_1^1 was compared to

$$\begin{aligned} \mathcal{H}_2^1 &: (\rho_{23}, \rho_{14}, \rho_{13}, \rho_{12}) = 0 \\ \mathcal{H}_3^1 &: \text{“Not } \mathcal{H}_1^1 \text{ or } \mathcal{H}_2^1 \text{.”} \end{aligned} \quad (18)$$

Within each set, we assumed equal prior probabilities (i.e., $\frac{1}{3}$). Each hypothesis was first compared to the unconstrained model, \mathcal{H}_u , which resulted in BF_{tu}^s for all hypotheses. These Bayes factors are not of substantive interest, but they are needed for then computing BF_{12}^s , BF_{13}^s , and the posterior model probabilities. We considered sample sizes ranging from 100 to 1,500 (in increments of 100), and two values for the scale, $\delta \in \{3, 15\}$. The latter values correspond to prior standard deviations of 0.5 and 0.25, respectively, and were chosen because we view them as the most likely to be used in practice. The posterior hypothesis probabilities, $p(\mathcal{H}_1|\mathbf{Y})$, were averaged across 500 simulation trials.

Results. The results are shown in panel A Figure 5. The lines capture the posterior probability for the true hypotheses in (17) and each color denotes their respective prior complexity (i.e., the prior proportion in agreement with Ω_t). Across all conditions, the posterior probabilities tended towards one as sample size increased. Importantly, the most specific hypothesis (light blue line) received more support across all conditions. In fact, the posterior probability for \mathcal{H}_1^3 was over 0.9 with just 500 observations for both values of δ . However, note that the posterior probabilities differ according to the prior scale. This is due to the inclusion of each \mathcal{H}_2^s , an *equality* constrained hypothesis. This is a natural property of the methodology because the prior reflects the expected magnitude of the partial correlations when the equality does not hold. Together, these results show that when conducting confirmatory analyses, more specific hypotheses are preferred, and indeed result in greater posterior probabilities, given that they are supported by the data.

Study 2: Prior Specification

The use of Bayes factors has been critiqued for being overly sensitive to the choice of prior distribution (e.g., Liu & Aitkin, 2008, but see Rouder, Morey, & Wagenmakers, 2016). However, a proposed advantage of the encompassing prior approach is that the resulting Bayes factors are robust to the prior variance when testing inequality constrained hypotheses (Hojtink, 2011; Klugkist et al., 2005). This is an important consideration in confirmatory testing because the Bayes factor would be consistent regardless of how the scale for the encompassing prior is specified. Thus, we investigated the extent to which the prior hyperparameter δ influences posterior probabilities for inequality constrained hypotheses. We first formulated a single hypothesis set

⁴In the case of equality constrained hypotheses, sensitivity analyses can be performed to determine the influence of the prior on the resulting Bayes factors (Hojtink et al., 2019).

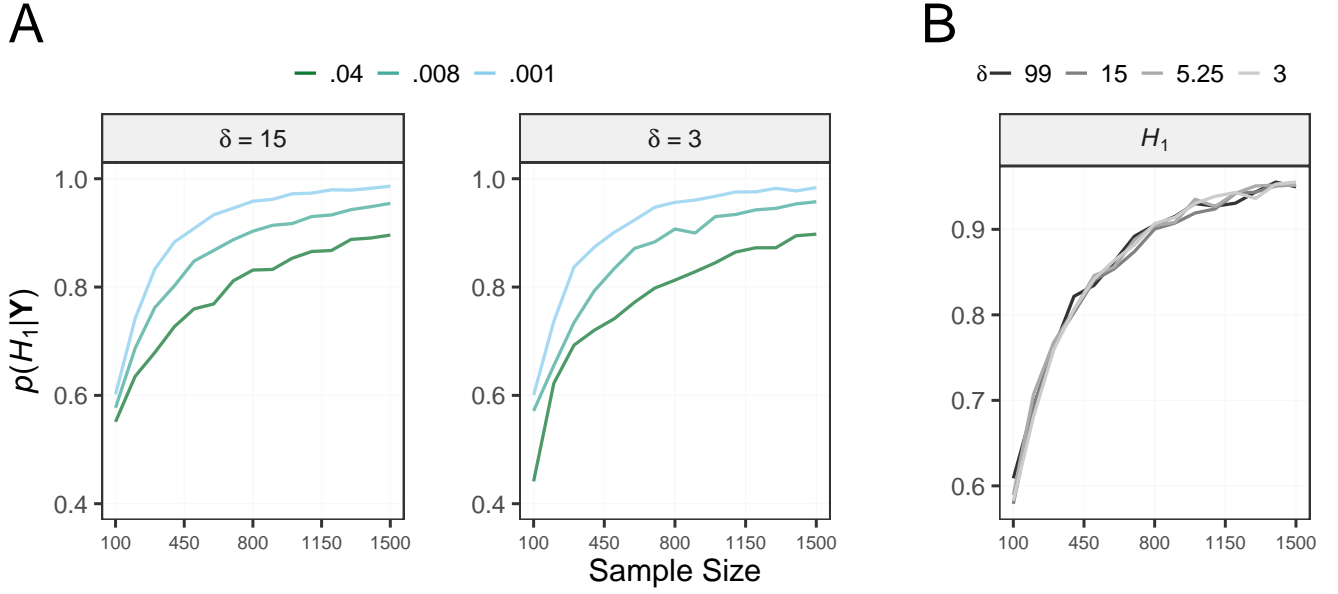


Figure 5. Results from the [Simulation Studies](#). The y-axes denote posterior model probabilities (PMPs). (A) In [Study 1: Specific Hypotheses](#), three true and increasingly specific hypotheses were tested against competing hypotheses, across two values for the prior variance (δ). Each line captures the PMP for a true hypothesis and each color denotes its respective prior proportion in agreement with the unconstrained hypothesis. Though all PMPs tended to one with increasing sample size, more specific hypotheses needed less samples to receive strong evidence. The results support the notion that more specific hypotheses are preferred in the encompassing prior approach given that they are supported by the data. (B) In [Study 2: Prior Specification](#), one true inequality constrained hypothesis was specified against a competing hypothesis across five values of δ . All PMPs tended to one with increasing sample size, and notably, overlap almost perfectly. This supports the notion that the evidence when testing inequality constrained hypotheses is robust to the prior specification.

Discussion

In this paper, we presented an innovative strategy for integrating exploratory analyses with confirmatory hypothesis testing in partial correlation networks. In doing so, one of the primary motivations for network analysis — hypothesis generation — has been fully realized. We began with an illustrative example based on a customary exploratory approach wherein, by simply plotting the network structure, we formulated several hypotheses regarding the most central node. This highlighted how information encoded by the partial correlations and the conditional (in)dependence structures can be employed to formalize clinical and theoretical expectations, that in turn, can be tested in a confirmatory setting.

The core contribution of this work demonstrated how centrality metrics can be used to guide hypothesis generation in exploratory network analyses. In extensive examples, bridge strength, a measure of inter-community connectivity, served to identify central bridge symptoms in two networks. Once identified, we formulated hypotheses with the goal of understanding various aspects of the bridging structures. For instance, whether the set of edges for one bridge symptom overlap with the set of another or whether two bridge symptoms are conditionally independent (see [Empirical Applica-](#)

$$\begin{aligned} \mathcal{H}_1 : \rho_{34} > \rho_{24} > \rho_{23} > \rho_{13} > \rho_{12} \\ \mathcal{H}_2 : \text{"not } \mathcal{H}_1." \end{aligned} \quad (19)$$

with \mathcal{H}_1 as the true hypothesis. We then varied the prior hyperparameter $\delta \in \{99, 15, 5.25, 3\}$. These values correspond to prior standard deviations of 0.1, 0.25, 0.4, and 0.5, respectively. Prior probabilities for each hypothesis were assumed to be equal, and BF_{12} was computed. We considered sample sizes ranging from 100 to 1,500 (in increments of 100), and posterior hypothesis probabilities, $p(\mathcal{H}_1|Y)$, were averaged across 500 simulation trials.

Results. The results are shown in Panel B Figure 5. Each line corresponds to the posterior probability of \mathcal{H}_1 versus \mathcal{H}_2 in (19) for a given value of δ . Importantly, the posterior hypothesis probabilities for each prior overlap almost perfectly across all sample sizes, and tended towards one as sample size increased. Together, the results indicate that the resulting Bayes factor is robust to different priors testing inequality constrained hypotheses.

tions). Together, these examples highlighted how inherently exploratory metrics can inform hypotheses aimed at replicating aspects of the network structure.

Testing specific structures can shed a new light upon the issue of replicability in networks. Indeed, formulating fine-grained hypotheses focused on characterizing central nodes is an active issue in the network literature (Epskamp, Borsboom, & Fried, 2018; Forbes et al., 2017; Forbes, Wright, Markon, & Krueger, 2019). In this work, however, we successfully replicated multiple central structures across distinct datasets. Note that we chose to focus on aspects we deemed most important. Namely, edge weights for the most central structures. Hence, it seems that network structures of interest can indeed be replicated.

Implications for Building Formal Models

The proposed testing strategy has several desirable qualities for building formal models. There is now a wealth of network analyses for several mental disorders (e.g., PTSD) and synthesizing this information to develop formal theories is a pressing challenge (for detailed discussions see Haslbeck et al., 2019 and Borsboom et al., 2020). In order to move towards formal theories, researchers must move away from the traditional exploratory approach and begin testing confirmatory hypotheses. We argue that this is not only necessary for building formal models, but also thinking about clinical interventions. This important step is absent from the current literature, in that results from exploratory analyses are never confirmed. This is in contrast to other scientific disciplines that also use partial correlation networks. In biological fields, for instance, exploratory results are actually used to generate hypotheses that are then tested (Kelder, Conklin, Evelo, & Pico, 2010; Krumsiek et al., 2012). These fields often conduct controlled experiments — perhaps a bridge too far for the most common applications in the social-behavioral sciences. However, as we demonstrated, it is certainly possible and quite useful to confirm findings that emerged in an exploratory context.

Furthermore, the Bayesian aspect of our approach is well-suited for constructing theories. Because we use the Bayes factor for confirmatory testing, we are quantifying the relative success of hypotheses at predicting the observed data (Kass & Raftery, 1995) — an important measure of explanatory power. Moreover, developing formal theory is an iterative process which requires updating as more data becomes available. Bayesian analyses are naturally lend themselves to this because prior information can be incorporated. Specifically, the results of a Bayesian analysis (i.e., posterior odds) can be formally incorporated into subsequent analyses as prior odds. This allows for monitoring the evidence a given theory has amassed.

Finally, our approach facilitates testing “risky predictions” (Mayo, 1991; Meehl, 1967). That is, a prediction that ex-

tends beyond refuting a null hypothesis or simply testing a direction (e.g., the effect is positive). The idea behind our approach is that hypotheses can express precise expectations through (in)equality constraints. This was demonstrated in this work, for example, by testing whether an exact ordering of effects replicated in a new dataset. This is useful in developing theories. Further, our approach can be used for testing theories by allowing researchers to be even more explicit about what should be observed. For instance, one could extend an exact ordering of edges by stipulating an additional constraint that they are all bounded between two values, say 0.10 and 0.20. This is a key aspect of theory building, that is, formulating and testing theoretical expectations.

Embracing the Gaussian Graphical Model

We urge researchers to embrace Gaussian graphical modeling. In our opinion, the focus on causality in psychological networks has led to an underappreciation of undirected networks as valuable tools for more than just exploratory data analysis. As we demonstrated, formalizing theoretical models can be accomplished by thinking in terms of constraints, on, say, the interactions between clinical symptoms. This allows researchers to establish, describe, and characterize important relations. This can be accomplished by adopting the powerful framework described in this work for exploratory and confirmatory testing. This is an important first step towards moving beyond the notion that GGMs are *merely* a stepping stone to directed networks.

Limitations

There are some notable limitations in this work. First, we only considered bridge strength as a metric to identify the most central structures in a network. We viewed this as a sensible choice because both the prior distributions for the confirmatory testing strategy and bridge strength focus on the partial correlations. Though this choice made it straightforward to formulate hypotheses, it may not always be so clear what parameters to focus on when using alternative exploratory metrics (Bringmann et al., 2019; Jones et al., 2019). Second, because only covariance matrices were available we assumed multivariate normality when generating data. However, the data were collected as ordinal. In practice, ordinal data results in more sampling variability, and thus less statistical power to replicate effects⁵ (Williams, 2020). Third, Bayes factor estimates may be unstable when testing overly specific hypotheses. This is because the prior probability that the constraints are in agreement with the unconstrained parameter subspace becomes quite small, and thus, a prohibitively large number of samples are needed for accurate

⁵Methods for dealing with ordinal data in GGMs are implemented in the R package **BGGM**

estimates (Mulder, 2016). In our experience, this issue typically arises when overly specific hypotheses are specified in conjunction with unordered groupings (e.g., (ρ_{12}, ρ_{13})). Fourth, when comparing nested hypotheses such as \mathcal{H}_1 and \mathcal{H}_2 in (10), the Bayes factor for the more specific hypothesis (e.g., BF_{21}) is bounded. As a result, the scale of the Bayes factor is difficult to interpret and the evidence for the true hypothesis does not tend to infinity with increasing sample size (if the more specific hypothesis is true, Mulder, Hoijtink, & Klugkist, 2010). Nested hypotheses can still be tested if there is a reason to do so, say, based on theoretical reasoning, but this caveat should be kept in mind when interpreting the evidence. Lastly, we did not conduct sensitivity analyses for any of our confirmatory hypothesis tests so it is uncertain to what extent the prior distribution influenced our results. While sensitivity analyses should be conducted in practice, we avoid doing so due to the demonstrative nature of this work.

Recommendations

We recommend that researchers make several considerations when using the exploratory and confirmatory strategies described in this paper. To start, researchers should carefully think about how exploratory metrics relate to the scientific question at hand when using them to guide the formulation of hypotheses. In particular it is not clear what centrality indices measure in psychological networks (Bringmann et al., 2019). For example, metrics using measures of “betweenness” and “closeness” assume the existence of a shortest path. Because shortest paths do not account for edge weight they may contradict how psychological variables are thought to interact. As such, it is important that researchers determine how exploratory metrics relate to their research prior to their use.

If independent samples are not available or if hypotheses cannot be derived from previous research, we recommend researchers take advantage of data splitting methods (Anderson & Magruder, 2017; Dahl et al., 2008; Faraway, 1995). We believe data splitting is underutilized in psychological research, and provides an accessible method for obtaining independent data on which to formulate and then test hypotheses. Indeed, data splitting could be called “one of the most seriously neglected ideas in statistics” (comment in Stone, 1974). Although this procedure results in a loss of statistical power, there are several ways to mitigate this. For example, power can be increased by focusing on inequality constrained hypotheses (e.g., Mulder & Raftery, 2019), or by focusing on the strongest edges. Additionally, a lower Bayes factor threshold can be used in determining the graph. Though lowering the threshold results in greater power, it also increases the rate of false positives. Thus, we recommend researchers focus on testing inequality constraints and large effects when splitting their data.

In fact, it may be useful to prioritize large effects in gen-

eral. This is because there is an inherent limit to what can and cannot be confirmed in a data set. Characterizing large effects first then and smaller effects second can be thought of as a *top-down* approach that can guide exploratory and confirmatory analyses over time. This idea is not new. In the genetics literature, it has been suggested that focusing on large effects is a useful way to begin understanding a system. For example, Altay and Emmert-Streib (2010) state that

“However, practically, no method can guarantee to [infer an entire network] for a given data set, not even for simulated data when a very large number of samples is available...For this reason, we lower the bar from the beginning by not aiming to infer the entire network, instead, [inferring] the strongest interactions among covariates only.” (p. 2)

In fact, we attribute part of our success in replicating bridge relations to focusing on the strongest edges. Hence, we recommend that researchers focus on large effects when transitioning from exploratory to confirmatory analyses.

Conclusion

This work demonstrated that confirmatory testing can be woven into the very fabric of network analysis and theory. The ideas presented in this paper provide the foundation from which to begin comparing formalized expectations related to the (in)dependence structure of psychological constructs and mental disorders. We hope this bridges the gap between between hypothesis generation and testing in psychological networks. The testing strategy is implemented in the R package **BGGM**. A detailed tutorial is available on the [Open Science Framework](#).

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Appendix Node Definitions

Table A1. *Definitions for nodes in Figure 3.*

Node	Symptom	Community
B1	Intrusive thoughts	Re-Experiencing
B2	Nightmares	Re-Experiencing
B3	Flashbacks	Re-Experiencing
B4	Physiological/psychological reactivity	Re-experiencing
C1	Avoidance of thoughts	Avoidance
C2	Avoidance of situations	Avoidance
C3	Amnesia	Avoidance
C4	Disinterest in activities	Avoidance
C5	Feeling detached	Avoidance
C6	Emotional numbing	Avoidance
C7	Foreshortened future	Avoidance
D1	Sleep problems	Arousal
D2	Irritability	Arousal
D3	Concentration problems	Arousal
D4	Hypervigilance	Arousal
D5	Startle response	Arousal

Table A2. *Node definitions for Figure 4.*

Node	Symptom	Community
D1	Lower interest or pleasure	Depression
D2	Feeling down, hopeless	Depression
D3	Trouble sleeping	Depression
D4	Tired or little energy	Depression
D5	Poor appetite/overeating	Depression
D6	Guilt	Depression
D7	Trouble concentrating	Depression
D8	Moving slowly/restless	Depression
D9	Suicidal thoughts	Depression
A1	Nervous, anxious, on edge	Anxiety
A2	Uncontrollable worry	Anxiety
A3	Worry about different things	Anxiety
A4	Trouble relaxing	Anxiety
A5	Restless	Anxiety
A6	Irritable	Anxiety
A7	Afraid something awful might happen	Anxiety