

On Accepting the Null Hypothesis of Conditional Independence in Partial Correlation  
Networks: A Bayesian Analysis

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## Abstract

Partial correlation networks have emerged as an increasingly popular model for studying mental disorders. Although conditional independence is a fundamental concept in network analysis, which corresponds to the null hypothesis, the focus is typically to detect and then visualize non-zero partial correlations (i.e., the “edges” connecting nodes) in a graph. As a result, it may be tempting to interpret a missing edge as providing evidence for its absence—analogously to misinterpreting a non-significant  $p$ -value. In this work, we first establish that a missing edge is incorrectly interpreted as providing evidence for conditional independence, with examples spanning from substantive applications to tutorials that instruct researchers to misinterpret their networks. We then go beyond misguided “inferences” and establish that null associations are interesting in their own right. In the following section, three illustrative examples are provided that employ Bayesian hypothesis testing to formally evaluate the null hypothesis, including a reanalysis of two psychopathology networks, confirmatory testing to determine whether a particular post-traumatic stress disorder symptom is disconnected from the network, and attenuation due to correcting for covariates. Our results shed light upon conditionally independent symptoms and demonstrate that a missing edge does not necessarily correspond to evidence for the null hypothesis. These findings are accompanied with a simulation study that provides insights into the sample size needed to accurately detect null relations. We conclude with implications for both clinical to theoretical inquiries.

*Keywords:* partial correlation network, conditional independence, null hypothesis, Bayesian inference, Bayes factor

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In the social-behavioral sciences, network theory has emerged as an increasingly popular framework for understanding psychological constructs and mental disorders (Borsboom, 2017; Jones, Heeren, & McNally, 2017). The underlying rationale is that a group of variables are a dynamic system that mutually influence and interact with one another (Borsboom & Cramer, 2013). This stands in contrast to the customary approach, where it is assumed that there is an unobserved, or latent, common cause. This perspective has been criticized by network proponents because it ignores the possibility of functional associations among observed variables (Borsboom & Cramer, 2013; Cramer, Waldorp, van der Maas, & Borsboom, 2010). This important and novel theoretical distinction has resulted in an explosion of research, for example, the network approach has been used to explore a variety of constructs including depression (Mullarkey, Marchetti, & Beevers, 2018), post-traumatic stress disorder (McNally et al., 2015), personality (Costantini et al., 2015), and narcissism (Briganti & Linkowski, 2019; Di Pierro, Costantini, Benzi, Madeddu, & Preti, 2019).

This work focuses on psychological networks that are estimated with Gaussian graphical models (Epskamp & Fried, 2018a; Epskamp, Waldorp, Mottus, & Borsboom, 2018). The basic idea is to characterize multivariate relations by learning the conditional dependence structure, that is, the partial correlation “network” (Epskamp & Fried, 2018a; Epskamp, Waldorp, et al., 2018). In a psychopathology network, for example, symptoms are “nodes” and the featured connections linking nodes are “edges” that graphically represent the conditional dependence structure. This relates to the idea that networks represent a causal skeleton (but see Ryan, Bringmann, & Schuurman, 2019), because, in observational data, an important ingredient of causality is conditional independence (Epskamp, van Borkulo, et al., 2018; Pearl, 2009). In networks, this corresponds to a partial correlation of zero—after controlling for all other observed variables, there is *no*

effect. However, we are not aware of many examples that have formally evaluated the null hypothesis of conditional independence. This is a striking oversight.

To understand the idea behind conditional relations, and thus network modeling more generally, it is informative to think of a simple model that includes only three nodes. In Figure 1 (panel A), the *true* network structure does not have a connection between  $A$  and  $C$ , which indicates that conditional on  $B$  they are not correlated (i.e., conditionally independent). Although these examples are commonplace in the literature, they can also be misleading, in that, while panel A is indeed the ground truth, in practice we merely have an *estimate* of the network structure (some possibilities are illustrated in Figure 1, panel B). Those nodes not connected *could* be conditionally independent but another possibility is a non-zero relation that was not detected and incorrectly set to zero (a false negative). This insight is not new, that is, “Inferring the dependence structure of such a Gaussian graphical model is thus the same as estimating which [partial correlations] are nonzero,” (p.494, Xie, Liu, & Valdar, 2016). But it could be underappreciated, because suggesting there is necessarily no effect would be analogous to misinterpreting a non-significant  $p$ -value (Altman & Bland, 1995; Goodman, 2008).

It is well-known that non-significance is often mistaken as providing evidence for the null hypothesis (see references in Greenland et al., 2016). The vast majority of networks, however, have been estimated with  $\ell_1$ -regularization that can push estimates to exactly zero (Epskamp & Fried, 2018b; Friedman, Hastie, & Tibshirani, 2008). The same idea applies, in that the zeroes do not provide evidence for the null hypothesis. This is because automated model selection is used to detect non-zero relations. What is more, the focus is typically on a conservative network, which necessarily increases the false negative rate (e.g., pg. 143 in Borsboom, Robinaugh, Rhemtulla, & Cramer, 2018). From surveying the applied network literature, we encountered several instances of interpreting no connection without the necessary nuance, for example, “An absent edge means that two nodes are conditionally independent given all other nodes in the network” (p. 308, Hevey, 2018).

Furthermore, in the many introductions to network modeling, it is often suggested that no connection (a missing edge) in a graph as indicates a lack of correlation or conditional independence (for but some examples see [Boschloo, Schoevers, Borkulo, Borsboom, & Oldehinkel, 2016](#); [Epskamp, van Borkulo, et al., 2018](#); [Fried & Cramer, 2017](#); [Kossakowski et al., 2016](#); [Papachristou et al., 2019](#)). But, again, while this is the case for the *true* generating structure, in practice the estimated network structure is visualized that merely contains the detected edges (e.g., [Figure 1](#), panel B).

### Is No Connection Substantively Meaningful?

Although misinterpretations abound, it is also important to consider whether the null hypothesis is of substantive and theoretical interest to network researchers. This would suggest that there is a need to formally evaluate the null hypothesis, as opposed to more carefully interpreting the estimated conditional dependence structure. The following three examples indicate that null associations are meaningful.

First, in [McNally et al. \(2015\)](#), network models were introduced for studying PTSD, with a focus on juxtaposing association networks, that consist of zero-order (or bivariate) correlations, versus partial correlations networks. Here it was noted that

Clusters of associations remain [after controlling for all other symptoms] among anger, sleep problems, and concentration impairment, and among intrusive thoughts, dreams, and flashbacks. However, the other two reexperiencing symptoms, physiological reactivity in response to reminders of the trauma and feeling upset upon encountering these reminders, have essentially no connection to the other reexperiencing symptoms... (p. 842).

This example is relevant for two reasons. First, the clusters of nodes were obtained from the DSM-IV manual and thus establishing conditional independence would indicate that the clusters are in need of revision. This is because a prerequisite of clustering is shared connections (section 4.1 in [Armour, Fried, Deserno, Tsai, & Pietrzak, 2017](#)). Second,

psychometric scales are commonly used in network analysis that, by construction, will have strong zero-order correlations, resulting in a fully connected association network. Thus, formally establishing no connection or evidence for the null hypothesis is a unique inference provided by partial correlation networks. This was done explicitly in [Wasil, Venturo-Conerly, Shinde, Patel, and Jones \(2020\)](#), where null associations that arose in an exploratory setting were interpreted in light of the broader literature (p. 284).<sup>1</sup>

Second, relations between depression and makers of inflammation were examined in [Fried et al. \(2019\)](#), including networks corrected for covariates (e.g., demographic variables). This was accomplished by including covariates into the model and then noting whether the “links [edges or connections] survived covariate adjustment” (p. 16). For example, “When corrected for covariates (1b), the relationship between IDS total score and IL-6 was attenuated, and the link with CRP disappeared” and “when corrected for covariates, no single edge emerged between markers and depressive symptoms.” It was concluded that “symptoms and markers were unrelated” (p. 1), although the null hypothesis was not formally evaluated. This example demonstrates that directly testing the null hypothesis would be quite useful for understanding the extent to which covariates attenuate edges (i.e., partially or completely).

Third, connectivity (and lack thereof) is an important concept in network theory. In psychopathology networks ([Borsboom, 2017](#); [Borsboom & Cramer, 2013](#)), for example, an underling idea is that vulnerability is related to network connectivity:

Analogous to a “domino effect,” among individuals with networks of densely connected symptoms, the activation of one symptom by another symptom or external stressor is more likely to lead to widespread activation of closely related symptoms. In contrast, loosely connected (i.e., low density) networks are more flexible and can adjust more adaptively to symptom activations and

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<sup>1</sup> To our knowledge, this is the only example in which the null hypothesis was formally evaluated and interpreted.

external stress (p. 217, [Smith et al., 2018](#))

This quote makes clear that “symptoms can cause other symptoms,” which is a central tenet of network theory, as opposed to a latent variable (common cause) model. As a result, in clinical applications, the most central symptoms are thought to be promising targets for intervention, that is, “the alleviation of a highly connected symptom/s may ultimately break down the overall PTSD network and possibly help facilitate treatment gains” (p. 57 [Armour et al., 2017](#)). Here the hope is to induce a “cascade of symptom deactivation” (p. 6 in [Fried et al., 2017](#)). Hence, *formally* establishing evidence for “deactivation” or no connection should have an important place on the mantel of network theory.

### Testing the Null Hypothesis

Recently, Bayesian hypothesis testing was introduced for psychological networks ([Williams, 2019](#)), which can be used to formally test of the null hypothesis of conditional independence. This can ensure that no connection is interpreted correctly and to answer questions for which null associations are of interest. For each partial correlation,  $\rho$ , this is accomplished by comparing competing hypotheses, that is,

$$\begin{aligned}\mathcal{H}_0 : \rho &= 0 \quad (\text{conditional independence}) \\ \mathcal{H}_1 : \rho &\neq 0 \quad (\text{conditional dependence}),\end{aligned}\tag{1}$$

to determine which best predicts the data at hand. This does have similarities to the so-called “conditional independence test” used extensively in the DAG literature ([Kalisch, Mächler, Colombo, Maathuis, & Bühlmann, 2012](#)). In that case, however, a classical null hypothesis significance test is used. Thus statements such as, “testing whether a partial correlation is zero or not” (p. 618, [Bühlmann, Kalisch, & Meier, 2014](#)), are incorrect—the

null hypothesis can only be rejected. On the other hand, the Bayes factor quantifies relative evidence for or against  $\mathcal{H}_0$ . This is a defining feature of Bayesian inference, in that, instead of assuming conditional independence is *true* (as in null hypothesis significance testing), theoretical models are formally compared to one another.

Additionally, this approach is important even when only the conditional dependence structure is of interest. This is because, if neither hypothesis in Equation (1) is supported, the evidence is ambiguous which indicates there could still be a connection between nodes. As a result, there are three estimated structures, including for conditional independence ( $\mathcal{H}_0$ ), conditional dependence ( $\mathcal{H}_1$ ), and an ambiguous network that includes relations for which the evidence was inconclusive. By formally comparing hypotheses, our hope is that this will not only provide network researchers with a tool for testing the null hypothesis, but also a more nuanced view of the underlying structure of conditional relations.

## Overview

In what follows, we tackle the important topic of conditional independence using Bayesian methodology. We first describe Gaussian graphical models in the context of a motivating example, that is meant to highlight underappreciated aspects of the estimated dependence structure. We then proceed to reanalyze two psychopathology networks with Bayesian hypothesis testing. Although this places our work within the tradition of using Bayesian methods to gain a better understanding of null effects (Brydges & Bielak, 2020; Brydges & Gaeta, 2019; Hoekstra, Monden, van Ravenzwaaij, & Wagenmakers, 2018), we take it one step further by then providing examples where gathering evidence for a null association is the question of interest. Next, a brief simulation is included to provide insights into the sample size needed to detect conditionally independent relations.

## The Gaussian Graphical Model

For multivariate normal data (Baba, Shibata, & Sibuya, 2004; Baba & Sibuya, 2005), a GGM captures conditional relations that are typically visualized to infer the underlying

dependence structure (i.e., the partial correlation “network”; Højsgaard, Edwards, & Lauritzen, 2012; Lauritzen, 1996). There is an undirected graph that is denoted  $G = (V, E)$ , consists of a vertex  $V = \{1, \dots, p\}$  and an edge set  $E \subset V \times V$ . The former refers to “nodes” that are, say, symptoms, whereas  $E$  is the estimated dependence structure. Let  $\mathbf{y} = (y_1, \dots, y_p)'$  be a random vector indexed by the graph’s vertices that is assumed to follow a multivariate normal distribution,  $\mathbf{y} \sim \mathcal{N}_p(\boldsymbol{\mu}, \boldsymbol{\Sigma})$ , with the mean vector  $\boldsymbol{\mu} = (0_1, \dots, 0_p)'$  and a  $p \times p$  positive definite covariance matrix  $\boldsymbol{\Sigma}$ .

The undirected graph is obtained by determining which off-diagonal elements in the precision matrix,  $\boldsymbol{\Theta} = \boldsymbol{\Sigma}^{-1}$ , are non-zero. That is,  $(i, j) \in E$  when nodes  $i$  and  $j$  are determined to be conditionally dependent and set to zero otherwise. Note that standardizing  $\boldsymbol{\Theta}$  and reversing the sign yields partial correlations, that is,

$$\rho_{ij \cdot \mathbf{z}} = \frac{-\theta_{ij}}{\sqrt{\theta_{ii}\theta_{jj}}}, \quad (2)$$

where  $\mathbf{z}$  contains the nodes conditioned on (i.e.,  $p - 2$ ). Hence, it is possible to determine  $E$ , that includes the conditionally dependent relations (i.e.,  $\rho_{ij \cdot \mathbf{z}} \neq 0$ ), with Bayesian hypothesis testing for each partial correlation (Williams & Mulder, 2020a). Conversely, the null hypothesis of conditional *independence* (i.e.,  $\rho_{ij \cdot \mathbf{z}} = 0$ ) is paradoxically absent from this formulation. This is echoed in Jeanmougin, Charbonnier, Guedj, and Chiquet (2014):

Therefore, recovering non-zero entries of  $\boldsymbol{\Theta}$  is equivalent to inferring the graph of dependencies, and the correct selection of non-zero entries is the main issue in this framework (p. 124).

Note that a zero in the precision corresponds to a partial correlation of zero. Hence, when estimating a GGM, typically an attempt is made to merely detect conditionally dependent relations and there is no consideration of conditional independence. This also applies to the psychological network literature.

By happenstance, we came across some confusion surrounding conditional relations.

For example, in [Forbes, Wright, Markon, and Krueger \(2017\)](#), conditional dependence was confused with a zero-order correlation:

However, association networks do not account for the fact that the correlation between a pair of nodes might be due to their shared relationships with other symptoms (i.e., they may only be conditionally dependent) (p. 970).

In fact, a conditionally dependent effect refers to a partial correlation, that, by definition, has controlled for “shared relationships.” Furthermore, it has also been suggested that conditional independence refers to a non-zero effect. This was also stated in [Forbes et al. \(2017\)](#), “we can see the conditionally independent relationship between A and B (i.e., the relationship that is shared between A and B, but unshared with any other symptoms) ” (p. 970). This unknowingly provides the definition of a conditionally dependent effect ( $\rho_{ij \cdot z} \neq 0$ ). These misunderstandings share a common thread that directly relates to this work: they obscure the correspondence between the null hypothesis and conditional independence.

### Motivating Example

With conditional relations clearly defined, we now highlight what is perhaps an underappreciated aspect of network modeling; namely, that visualizing only conditional dependencies can obscure the inherent uncertainty surrounding what may be the *true* network structure and even the important relations. For example, in addition not being able to establish evidence for conditional independence, it is not necessarily the case that a detected relation is more important (i.e., larger) than those set to zero.

To this end, we reanalyzed 20 post-traumatic stress disorder symptoms ( $N = 221$  [Armour et al., 2017](#)). The node descriptions are provided in [A1](#). Because these data were measured on the 5-point Likert scale, we estimated an ordinal GGM with R package **BGGM** ([Williams & Mulder, 2020b](#)). In this example, we do not employ Bayesian hypothesis testing, but instead use Bayesian estimation to make frequentist inference, given

that the necessary long-run properties are not compromised with diffuse prior distributions (Little, 2006). In **BGGM**, the prior follows a beta distribution that is symmetric and centered at zero, with a hyperparameter,  $\delta$ , governing the scale (Equation 17 in Williams & Mulder, 2020a). The prior scale was set to 0.50 (Figure 2) and 5,000 samples were drawn from the posterior distribution (excluding a burn-in of 500 iterations). Due to using the maximum likelihood estimate for the initial value of  $\Theta$ , this number of posterior samples was adequate, as indicated by inspecting trace plots (Figure B1).

**Results.** Figure 3 (panel A) includes the estimated conditional dependence structure. An edge (partial correlation) was included in the graph if the 95% credible interval excluded zero, which translates into a significance test with  $\alpha = 0.05$ . The graph appears very sparse and perhaps thought to be an accurate depiction of the underlying structure. This is not necessarily the case, however, as the connections merely reflect those edges that were detected and set to zero otherwise. Two nodes not sharing a connection could reflect conditional independence or conditional dependencies that escaped detection (e.g., Figure 1).

Panel B includes 95% credible intervals (CrI) for each partial correlation. In frequentist inference, the intervals can be interpreted as containing those values that would *not* be rejected at  $\alpha = 0.05$  and rejecting those that are not within. This is the logic behind an equivalence test (Lakens, 2017), in that, if an interval is contained within some null region, say, spanning from  $\pm 0.10$  (the shaded region in panel B), then values larger than a small effect size can be rejected. In this case, because none of the intervals are contained within the null region, all relations set to zero could still be an edge larger than 0.10. This suggests that the possibility of a fully connected network with edges that are at least small in effect size cannot be ruled out.

One thought could be that the estimated dependence structure includes the most important relations still yet. Panel C includes differences between each edge (a connection in panel A) compared to those set to zero (no connection in panel A). Those difference for

which the 99% CrI excluded zero are highlighted in black. The results reveal that the majority of partial correlations in panel A are not more important (i.e., larger) than those set to zero.

## Summary

This example highlighted challenges that arise when the focus is on detecting edges that are then visualized in a graph. Although Figure 3 (panel A) appears to provide a clean picture of the network structure, our analysis indicated that not much was learned from the data, over and above what are likely non-zero relations. In other words, it would be a mistake to infer that “an absent edge means that two nodes are conditionally independent.” It also would be a misguided to conclude that Figure 3 (panel A) visualizes the most important relations, given that there is not necessarily a difference between a connection and no connection. Together, rather than providing rich inference, the graph is a mere visual representation of detected effects. A more informative understanding of the network structure, as demonstrated below, can be obtained by employing Bayesian hypothesis testing.

## Illustrative Examples

In psychological science, there are now several papers describing the various benefits of Bayesian inference (Kruschke & Liddell, 2015; Wagenmakers et al., 2018). As noted in Rouder, Morey, Verhagen, Province, and Wagenmakers (2016), the above frequentist equivalence test (e.g., Kruschke, 2013) “has a free lunch property where researchers need not make detailed assumptions about the alternative to test the null hypothesis” (p. 250), although it should be noted that our goal was rhetorical in nature. To fully reap the benefits of Bayesian hypothesis testing, an alternative hypothesis in the form of a prior distribution must be specified. This captures the hypothesized edge size and it is compared to the null hypothesis. Fortunately, Wysocki and Rhemtulla (2019) recently summarized edge sizes from 37 psychological networks, where it was shown that they are rarely larger

than 0.50 and typically small to medium in effect size. This information is utilized in the following examples.

### Exploratory Reanalysis

In this first illustration, we use Bayesian hypothesis testing to shed new light upon results originally obtained with frequentist methods. Although using Bayesian methods for this purpose is commonplace in psychological science (see for example [Brydges & Bielak, 2020](#); [Brydges & Gaeta, 2019](#)), this has yet to be not done in the network literature. We follow the recommended approach described in [Williams and Mulder](#) (see Figure 2 therein, [2020a](#)), and plot three graphs, including the conditional dependence and independence structures, as well as an ambiguous “network” for which the evidence was inconclusive (i.e., neither  $\mathcal{H}_0$  or  $\mathcal{H}_1$  was supported).

**Dataset 1.** These data come from a study using the General Anxiety Disorder scale (GAD-7) to measure anxiety symptoms with 7 items ( $N = 403$ ). The items are scored from 0 (“not at all”) to 3 (“nearly everyday”). The node descriptions are provided in [A2](#). Data were gathered at two waves and the first is used in this reanalysis. Further details can be found in [Forbes, Wright, Markon, and Krueger \(2019\)](#).

**Dataset 2.** These data come from a study using the 11-item version of the Center for Epidemiologic Studies Depression Scale (CES-D) to measure depressive symptoms ( $N = 515$ ). The items are scored from 1 (“hardly ever”) to 3 (“most of the time”). The node descriptions are provided in [A3](#). Further details can be found in ([Citation Fried et al., 2015](#)).

**Model Fitting.** Ordinal GGMs were fitted with the R package **BGGM** ([Williams & Mulder, 2019](#)). 5,000 samples were drawn from the posterior distribution (excluding a burn-in of 500 iterations). Due to using the maximum likelihood estimate for the initial value of  $\Theta$ , this number of posterior samples was adequate, as indicated by inspecting trace plots (e.g., Figure [B1](#)). For each partial correlation,  $\mathcal{H}_0 : \rho = 0$  versus  $\mathcal{H}_0 : \rho \neq 0$  was tested

with the prior scale set to 0.25. This prior was informed by [Wysocki and Rhemtulla \(Table 2 therein, 2019\)](#). Note that the Bayes factor is a continuous measure of evidence, but including relations in the network requires a decision rule. We used the (mostly arbitrary) value of three that is considered positive evidence ([Kass & Raftery, 1995](#)), which is typically more conservative than setting  $\alpha = 0.05$ .

**Results.** Figure 4 includes the estimated structures. A point of emphasis is that typically the conditional dependence structure is visualized in network analysis, which in this case appears rather sparse for both datasets. However, “an absent edge” did not necessarily translate into evidence for the null hypothesis. This was especially the case for the depression dataset, where the *majority* of relations were included in the ambiguous graph.

Several interesting findings emerged when focusing on the conditional independence structure. For example, in the anxiety dataset (panel A), node 5 (“being so restless that it’s hard to sit still”) was independent of node 2 (“not being able to stop or control worrying”) and 3 (“trouble falling or staying asleep, or sleeping too much”). That is, after controlling for the other symptoms, those symptoms were no longer correlated. In the depression dataset (panel B), node 7 (“I enjoyed life”) was independent of three nodes (2 = “I felt that everything I did was an effort”; 3 = “my sleep was restless”; 11 = “I could not get going”). This finding is intriguing, because the zero-order correlations were all negative (inversely related) and medium to large in size, yet they were independent when controlling for the other symptoms in the network.

Together, this reanalysis demonstrated that a “missing edge” can hardly be interpreted as implying that two nodes are conditionally independent, the ambiguous network lends itself to more nuanced inferences, and formally evaluating null associations provides unique insights not possible when focusing on conditional dependencies.

**Sensitivity Analysis.** When using Bayesian hypothesis testing, the results typically depend heavily on the assumed prior distribution (or alternative hypothesis),

regardless of the sample size. This is different than Bayesian estimation, where, with more data, the credible interval will often coincide with the confidence interval. It is thus commonplace to perform sensitivity analyses that investigate robustness to the assumed prior distribution. We follow this tradition here, although it should be noted that sensitivity analyses are most advantageous when there is prior uncertainty. This is not the case in our analyses, given our prior was informed by a comprehensive review in [Wysocki and Rhemtulla \(2019\)](#).

The sensitivity analysis is provided in Figure C1. Recall that the narrower priors most accurately capture an expected edge size, which also had the most relations in the ambiguous network. In both datasets, as the prior widened, such that more probability mass was on large edges, the number of ambiguous relations decreased. This was a result of more associations being included in the conditional independence network. When using too wide of a prior, this can result in too often accepting  $\mathcal{H}_0$ , even when there is a non-zero effect. This is also shown below in the simulation study (Figure 5). For the priors spanning a reasonable range, however, the results were consistent in so far as there being inconclusive evidence for a large proportion of the relations.

## Confirmatory Testing

Network modeling is customarily geared towards data-driven exploration. By this we mean that data are typically fed into some algorithm and what happens to emerge is interpreted. A more rigorous approach, that is commonly used in genetic network modeling, is to first mine the data and then confirm substantively interesting findings in either unseen data or with controlled experiments. The former is most applicable to the social-behavioral sciences, due the data typically being observational, and it is pursued in this example.

In [Armour et al. \(2017\)](#), it was found that the PTSD symptom, amnesia, lacked connections with other symptoms belonging to the same cluster per the DSM IV manual. It was further noted that “From a network perspective, the absence of a connection

between two symptoms implies that they are conditionally independent of each other given the other symptoms in the network,” which is not the case. This result can be seen in Figure 3 (panel A), where D6 (amnesia) does not have any connections with the other nodes in the same cluster (e.g., D2 - D7). If these null associations were to be confirmed, it would suggest that amnesia is actually conditionally independent of other symptoms thought to belong to a distinct cluster.

**Dataset.** To test this hypothesis, we used data from (Fried et al., 2018) that included four samples of those with PTSD. To ensure the most power, the two largest samples are used in this example ( $N = 926$  and  $N = 965$ ). There are 16 items in total. Because only the correlation matrices were available, we tested the hypotheses assuming that the data were Gaussian (further details are provided in Model Fitting).

**Hypothesis.** Amnesia (C1) is thought to belong to the negative alterations in cognition and mood cluster with four other symptoms (denoted C2 - C5). We compared the following hypotheses to see which the data were more likely under

$$\mathcal{H}_0 : (\rho_{C_1C_2}, \rho_{C_1C_3}, \rho_{C_1C_4}, \rho_{C_1C_5}) = 0 \quad (3)$$

$$\mathcal{H}_1 : (\rho_{C_1C_2}, \rho_{C_1C_3}, \rho_{C_1C_4}, \rho_{C_1C_5}) > 0$$

$$\mathcal{H}_2 : \text{“not } \mathcal{H}_0 \text{ or } \mathcal{H}_1\text{”}$$

$\mathcal{H}_0$  is the conditional independence hypothesis, which predicts that amnesia is disconnected from the other symptoms in the same cluster (i.e., all are equal to zero).  $\mathcal{H}_1$  can be understood as the positive manifold hypothesis, that is, the symptoms are positively related to one another and all relations are greater than zero (Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011). Furthermore,  $\mathcal{H}_1$  seems to be the expectation implied by the the DSM-IV.  $\mathcal{H}_3$  is the complement that provides the relative evidence for neither  $\mathcal{H}_0$  or  $\mathcal{H}_1$ .

**Results.** Table 1 includes the posterior hypothesis probabilities. For  $\mathcal{H}_0$ , they were 0.04 ( $N = 926$ ) and basically zero ( $N = 965$ ), respectively, which indicates little support for the idea that the amnesia is disconnected from its cluster. Furthermore, the most probable hypothesis in both data sets was  $\mathcal{H}_1$ , although, relative to  $\mathcal{H}_2$ , the evidence was not strong in sample 1. In sample 2, however, there was overwhelming support for the positive manifold hypothesis,  $\mathcal{H}_1$ , relative to the conditional independence hypothesis,  $\mathcal{H}_0$ , and moderate evidence for  $\mathcal{H}_1$  relative to  $\mathcal{H}_2$  ( $\text{BF}_{12} \approx 3$ ).

Although the confirmatory test was not supported, this example highlights the utility of formally evaluating the null hypothesis, as opposed to inferring conditional independence from “an absence of a connection.” Of note, the sample size in (Armour et al., 2017) was only 221, a situation in which many edges will be incorrectly set to zero (e.g., Figure 3).

### Correcting for Covariates

An interesting question in network analysis is how the underlying structure is impacted by covariates. The customary approach for answering this question is a two-step procedure, that consists of estimating the graph without and with covariates included, and noting which relations are no longer connected (or detected) after adjusting for the covariates and attenuation by inspecting mere point estimates (Fried et al., 2019). This is problematic for a couple reasons. First, because the difference between connected and not connected is not necessarily meaningful (see Figure 3, panel C). Second, because the sampling variability of a partial correlation increases with each additional node (Williams, 2020), there is reduced chance of detecting the relation. A naive approach could be to directly compare the adjusted and unadjusted, given this would ignore their interdependence, for which we are not aware of approaches for partial correlations. A step in the right direction is to evaluate the null hypothesis in the model including the covariates, resulting in a test for complete attenuation.

**Dataset.** We estimated a personality network with a dataset from the R package `psych` (Revelle, 2019). There are 25 self-reported items ( $p = 25$ ) that measured personality in 2236 subjects. The items (scored from one to six) were taken from the international personality item pool. The majority of subjects were between 20 and 60 years old ( $M = 29.5$  years,  $SD = 10.6$  years). 68% were females. We assumed the data were Gaussian, due to the number of categories, and fitted one model with only the 25 items and then another including the covariate age (further details are provided in [Model Fitting](#)).

**Results.** We first noted the proportion of the corrected relations that were smaller than the uncorrected relations (based on the posterior mean). Although nearly 44% were smaller, these differences were extremely small and ranged between basically zero to at most 0.013.

Table 2 includes the summarized posterior distributions for relations that were connected in the uncorrected network but not so in the corrected network. Recall that a vanishing connection has been suggested to reflect complete attenuation, which implies the null hypothesis has been accepted. The results reveal that the relations are very similar to one another, especially when considering the entire distribution. It appears the primary reason the corrected relations were not detected was because the effect was very small in the first place, such that even a natural fluctuation in the estimate can result in not being detected. This is reflected when looking at the Bayes factor in favor of the alternative hypothesis, which is more than one, thereby suggesting the evidence tended towards there being an edge (but it did not reach the threshold of 3). These findings highlight the importance of going beyond noting the absence of edges when correcting for covariates.

### Planning for Conditional Independence

In the exploratory reanalysis, the results revealed that the *true* structure is rather uncertain, even though it should be noted that the sample sizes were representative of the literature (Wysocki & Rhemtulla, 2019). In both datasets, a large portion of the partial

correlations were in the ambiguous network (Figure X). Consequently, it is important to investigate the sample size needed for detecting conditionally independent nodes—that is, planning for null associations.

To this end, we conducted a brief simulation. We first estimated the empirical partial correlations from the PTSD data used in the motivating example and set absolute values less than 0.05 to zero (Epskamp, 2016; Williams, Rhemtulla, Wysocki, & Rast, 2019). This served as the *true* network structure. Note that the generated data were multivariate normal. As pointed out in Williams (2020), there can be more uncertainty in polychoric partial correlations estimated from ordinal data with few categories. This indicates these simulation results will be overly optimistic for those kinds of data. We computed and the false positive rate for detecting non-zero and null associations across a range of sample sizes. We used scales for the prior distributions (0.25 and 0.50) and the a Bayes factor cut-off of three. Recall that 0.25 reflects the review of edge sizes provided in Wysocki and Rhemtulla (2019). The results were averaged across 100 simulation trials.

## Results

Figure 5 includes the simulation results. Here a key aspect of Bayesian methodology was revealed. That is, with more observations, model selection with the Bayes factor will converge on the *true* model (e.g., Casella, Girón, Martínez, & Moreno, 2009). This can be inferred by noting that the error rate goes to (essentially) zero and power goes to (nearly) 1. This was the case for both the conditional dependence and independence structures. The results also illustrate how “absence of evidence does not provide evidence for absence.” For example, when focusing on a sample size of 1,000 and conditionally dependence relations, power did not exceed 0.75. Consequently, this corresponds to over 25% of the *true* edges being incorrectly set to zero. Furthermore, the results were sensitive to the prior distribution. The wider prior had a much higher error rate for detecting null effects. This is because too much density was placed on improbable values. This conflicts with review in

Wysocki and Rhemtulla (2019), and, as a result, the data were better predicted by  $\mathcal{H}_0$ .

These results can inform sample size planning for network analysis. To detect null associations with a low error rate, a sample size of at least 1,000 may be required. Of course, even with that many observations, power could still be rather low. This can also be seen in Figure 4, where the sample sizes in both data sets were less than 1,000 and the conditional independence network contained few relations. The results also reveal that samples sizes greater than 5,000 could be required to make inference about the *true* network structure (i.e., few relations included in the ambiguous network). This is striking, given that samples sizes are typically smaller than 1,000 (Table 1 in Wysocki & Rhemtulla, 2019).

## Discussion

This work focused on the important topic of conditional independence in psychological networks. We highlighted that merely noting the absence of an edge in a graph does not necessarily correspond to evidence for the null hypothesis, although there are numerous examples in the literature suggesting otherwise, including high cited tutorials introducing network analysis. We did more than bring attention to this issue, however, as Bayesian hypothesis testing was employed to characterize the conditional independence structure of psychopathology symptoms. Hence, this work provides the first glimpse into accepting the null hypothesis in network analysis.

A unique aspect of this work is that we went beyond traditional Bayesian reanalyses, that typically focus on misinterpretations, and discussed the importance of null associations in network psychometrics. Several illustrative examples then demonstrated how to answer questions wherein the null hypothesis was of substantive interest. These spanned from determining whether a specific PTSD symptom was disconnected from the network to assessing attenuation after correcting for covariates. In each case, the research question was inspired from the extant literature, and in particular, where no connection

was misinterpreted (e.g., assuming nodes are unrelated due to a missing edge). For example, although findings in [Armour et al. \(2017\)](#) seemed to call into question symptom clusters, our confirmatory tests revealed little support for the null hypothesis. These conflicting findings could be due to the small sample size in [Armour et al. \(2017\)](#) (i.e.,  $N = 221$ ), which likely translated into many false negatives (e.g., Figure 5). Together, these examples highlighted the importance of formally evaluating null associations, rather than making “inference” from missing edges.

The simulation study provided context for interpreting the illustrative examples and provided some insight into planning for conditional independence. We found that accurately detecting null associations is not a bridge too far, but it will require larger sample sizes than typically used in network analysis. In a recent review of 37 psychological networks, for example, 81% had sample sizes smaller than 1,000 ([Wysocki & Rhemtulla, 2019](#)). Based on the simulation, even with  $N = 1,000$ , many relations will likely be included in the ambiguous network. This also applies to detecting non-zero effects, in that the false negative rate was over 0.25 with  $N = 1,000$ . To make sense of this, consider that edges are typically small in size and 50% power (half of the time) to detect just one edge (or partial correlations) requires a sample size of nearly 400. As pointed out in [Williams \(2020\)](#), the typical goal in network analysis is to *simultaneously* detect many small edges which requires an even larger sample sizes. Hence the importance of visualizing more than the edges that happened to be detected (e.g., Figure 3).

Of course, we are not the first to caution that the absence of an edge does not provide evidence for its absence. Here to, however, we worry there is much to be desired. For example, [Blanken et al. \(2019\)](#) stated “The absence of a direct edge should not be interpreted as an absence of any effect, but rather as an indication that an indirect effect is more likely given the available data” (p. 16). We are not entirely sure what this means, as even informally ruling out an association requires more forethought. Furthermore, the sample size in [Blanken et al. \(2019\)](#) was merely 52, a situation in which even strong

association (in the *true* structure) can be incorrectly set to zero. One possibility would be to define a substantively meaningful edge size and then computing the require sample size to have, say, 80% statistical power. Then if it did go undetected, perhaps the effect might as well be considered null. Unfortunately, sample size planning is mostly missing from network psychometrics (but see [Williams, 2020](#)). We thus caution researchers from interpreting a “missing edge” at all, unless, of course, the null hypothesis is formally testing (best case scenario) or some thought is given to statistical power to detect the smallest edge size of interest (at minimum).

### Approximately Null Associations

We focused on a precise null hypothesis, whereas it may be desirable to consider approximate conditional independence. For example, in observational data, perhaps it is more realistic to assume the null hypothesis always false ((see discussion in X) [Marsman & Wagenmakers, 2017](#)). This does make sense for significance testing, given the underlying assumption is that the null hypothesis is true.<sup>2</sup> However, in Bayesian hypothesis testing, this is not required:

The logarithm of the marginal probability of the data may also be viewed as a predictive score...[This] leads to the interpretation of the Bayes factor that does not depend on viewing one of the models as “true” (p. 777 [Kass & Raftery, 1995](#))

Hence, the more pressing question is whether considering “the relative predictive accuracy of one model over another” is meaningful (p. 106 [Rouder, Haaf, & Vandekerckhove, 2018](#)), given the Bayes factor is always interpretable as such. In other words, it is always possible to make inference as to whether the conditional independence model better predicts the data than an alternative (without assuming either is true), which we argue is useful

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<sup>2</sup> Note that in equivalence testing (Figure 3, panel B) a null region is considered. A common approach is two one one-sided tests, which assumes each null value (at the lower and upper limits of the region) is true.

information to behold. Furthermore, a Bayes factor can be obtained for a null region (e.g., [Morey & Rouder, 2011](#)), if desired, although the results will be quite similar to testing a precise null hypothesis.

## Implications

This work has both theoretical and clinical implications. For the latter, since the emergence of the network approach to psychopathology, it has been pointed out that perceiving mental disorders as a network of mutually influencing symptoms could lead to choosing specific symptoms as a target for clinical intervention ([Fried et al., 2018](#)). In this framework, network intervention analysis has been introduced to monitor how a treatment changes specific symptoms, and how such changes may affect the network structure of a mental disorder (e.g., [Blanken et al., 2019](#)). From a clinical point of view, and because of the growing interest in the clinical applications of the network approach, focusing on the formal evaluation of conditional independence relationships (or conditional dependence relationships) and the amount of evidence supporting the null hypothesis is of great importance, as it will allow for a more straightforward interpretation of the absence (or presence) of a connection between two symptoms in a network structure of a mental disorder or psychological construct.

Formally testing the null hypothesis of conditional independence can be woven into the fabric of network theory ([Borsboom, 2017](#); [Cramer et al., 2010](#); [Jones et al., 2017](#)). A central idea of network modeling is to generate testable hypotheses from exploratory analyses. There is now a wealth of networks in the extant literature, and synthesizing this information into formal theories is a pressing challenge ([Haslbeck, Ryan, Robinaugh, Waldorp, & Borsboom, 2019](#)). In our opinion, considering null associations provides a rich source of information that can be used to build anew or enhance existing theory, say, by directly testing for disconnection. Furthermore, as revealed in [Figure 4](#), there was evidence for conditionally independent symptoms that have large zero-order correlations. Yet,

because the focus is typically on conditional dependence, explaining findings of this nature will remain elusive so long as the emphasis does not expand to formally consider conditional independence.

## Recommendations

The most obvious recommendation is to adopt a Bayesian approach for network analysis. Unfortunately, a tutorial is beyond the scope of this work, but we encourage researchers to become familiar with Bayesian methodology before employing it practice. We recommend supplementing the ideas in this work with, to get started [Dienes \(2019\)](#); [Morey, Romeijn, and Rouder \(2016\)](#); [Wagenmakers et al. \(2018\)](#), which are excellent introductions to Bayesian inference that are geared specifically towards research psychologists.

We offer a general suggestion that can reduce ambiguity, over and above having a large sample size. First note that natural sampling variability will increase with more variables in the network and sampling variability will be relatively large for polychoric partial correlations estimated from ordinal data with few categories. Both of these factors can reduce “power” to detect both conditionally dependent and independent relations ([Williams, 2020](#)). Accordingly, if there are a variety of scales that purport to measure the same thing, then choosing that with the fewest items and the most ordinal categories can reduce the number of relations in the ambiguous network.

## A Note On $\ell_1$ -Regularization

It would remiss of us to write a paper about network analysis without discussing  $\ell_1$ -regularization. Because it is still the most commonly used estimator ([Williams & Rast, 2019](#); [Williams et al., 2019](#)), it would be convenient if it could be used to accept the null hypothesis. With a non-regularized estimator (e.g., maximum likelihood), the bootstrap could be employed to conduct an equivalence test based on a confidence interval. However, due to the nature of the  $\ell_1$ -penalty, bootstrapped “confidence” intervals are invalid, that is,

The (limiting) distribution of such a sparse estimator is non-Gaussian with point mass at zero, and this is the reason why standard bootstrap or subsampling techniques do not provide valid confidence regions or  $p$ -values. Thus, we have to use other approaches to quantify uncertainty (p. 7 [Bühlmann et al., 2014](#)).

This means that none of the inferences included in this work, over and above the estimated edge set, are possible with  $\ell_1$ -regularization, such as accepting the null hypothesis, comparing relations to see which are most important, and confirmatory hypothesis testing.

Furthermore, because the  $\ell_1$ -penalty can provide estimates of exactly zero, we worry this makes it especially susceptible to misinterpreting a missing edge. As with maximum likelihood and Bayesian methodology, there is always the danger of a false negative. However, because it is not possible to obtain a valid sampling distribution, there is no way to quantify uncertainty to, say, determine which values are compatible with the data. For example, we used the same data as [Armour et al. \(2017\)](#) in the motivating example, but there was a valid measure of uncertainty.<sup>3</sup> When simply looking at the credible intervals, it was apparent that not much was learned from the data (i.e., the intervals spanned a wide range of values). Furthermore, attenuation due to correcting for covariates has been inferred from an  $\ell_1$  estimate of zero. In our example, however, the measure of uncertainty indicated that it was not even clear there was a notable difference between the corrected and uncorrected association, let alone complete attenuation. Thus, as it relates to conditional independence, our illustrative examples highlighted what may be an underappreciated aspect of sparse estimators.

## Conclusion

This work described a tool for testing the null hypothesis and provided the insight that in common situations absence of evidence does not imply evidence of absence. This

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<sup>3</sup> [Armour et al. \(2017\)](#) computed  $\ell_1$ -regularized “confidence” intervals.

was accomplished by employing recently introduced Bayesian methodology that allows for estimating the conditional independence structure of a psychological network. To ensure the methods can readily be adopted, the analyses have been implemented in a tutorial that is freely available.

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Table 1  
*Hypothesis probabilities (Equation 3)*

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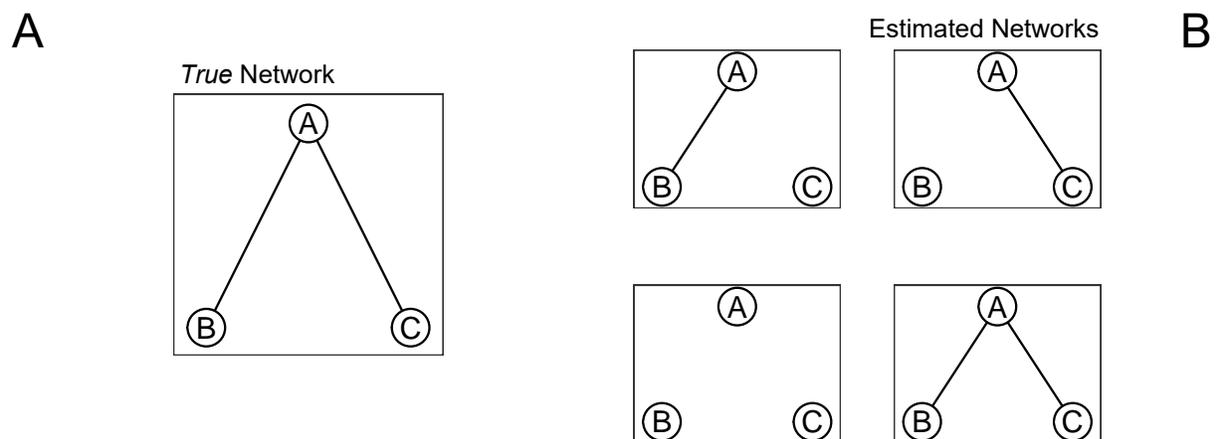
|          | $p(\mathcal{H}_0 \mathbf{Y})$ | $p(\mathcal{H}_1 \mathbf{Y})$ | $p(\mathcal{H}_2 \mathbf{Y})$ |
|----------|-------------------------------|-------------------------------|-------------------------------|
| Sample 1 | 0.04                          | 0.58                          | 0.38                          |
| Sample 2 | 0                             | 0.80                          | 0.20                          |

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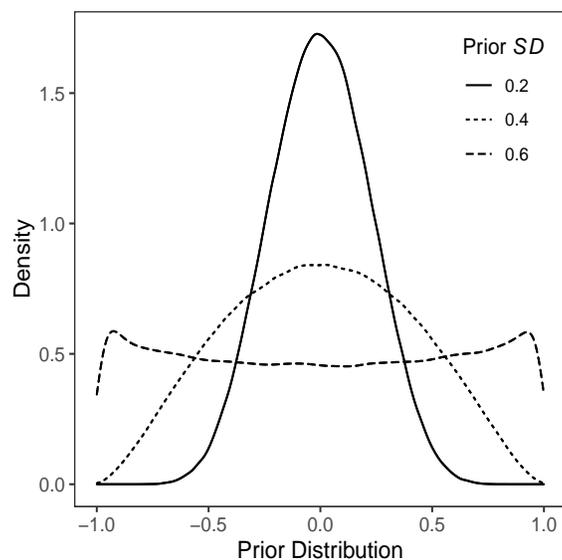
Table 2  
*Correcting for covariates*

| Relation                     | M     | SD    | 95% CrI        | BF <sub>10</sub> |
|------------------------------|-------|-------|----------------|------------------|
| A4–A5 <sub>uncorrected</sub> | 0.068 | 0.02  | [0.028, 0.108] | 17.071           |
| A4–A5 <sub>corrected</sub>   | 0.055 | 0.02  | [0.014, 0.095] | 2.560            |
| E3–N3 <sub>uncorrected</sub> | 0.061 | 0.020 | [0.021, 0.100] | 6.062            |
| E3–N3 <sub>corrected</sub>   | 0.055 | 0.021 | [0.015, 0.095] | 2.441            |

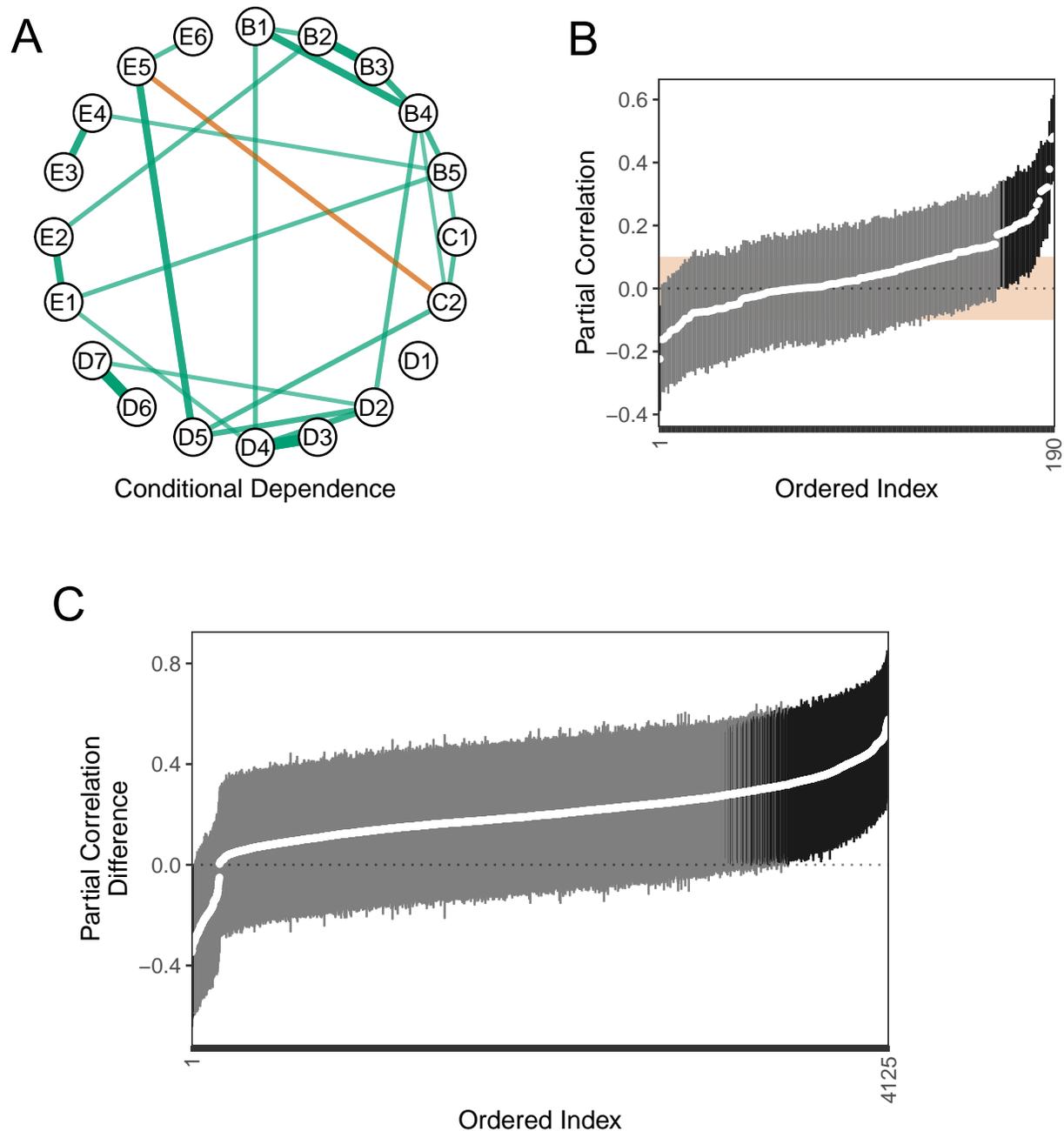
*Note.* Posterior mean (M), standard deviation (SD), and 95% credible interval (CrI). BF<sub>10</sub> is the Bayes factor in favor of the alternative hypothesis, relative to the null hypothesis. A4 and A5 are items measuring agreeableness, E3 extroversion, and N3 neuroticism.



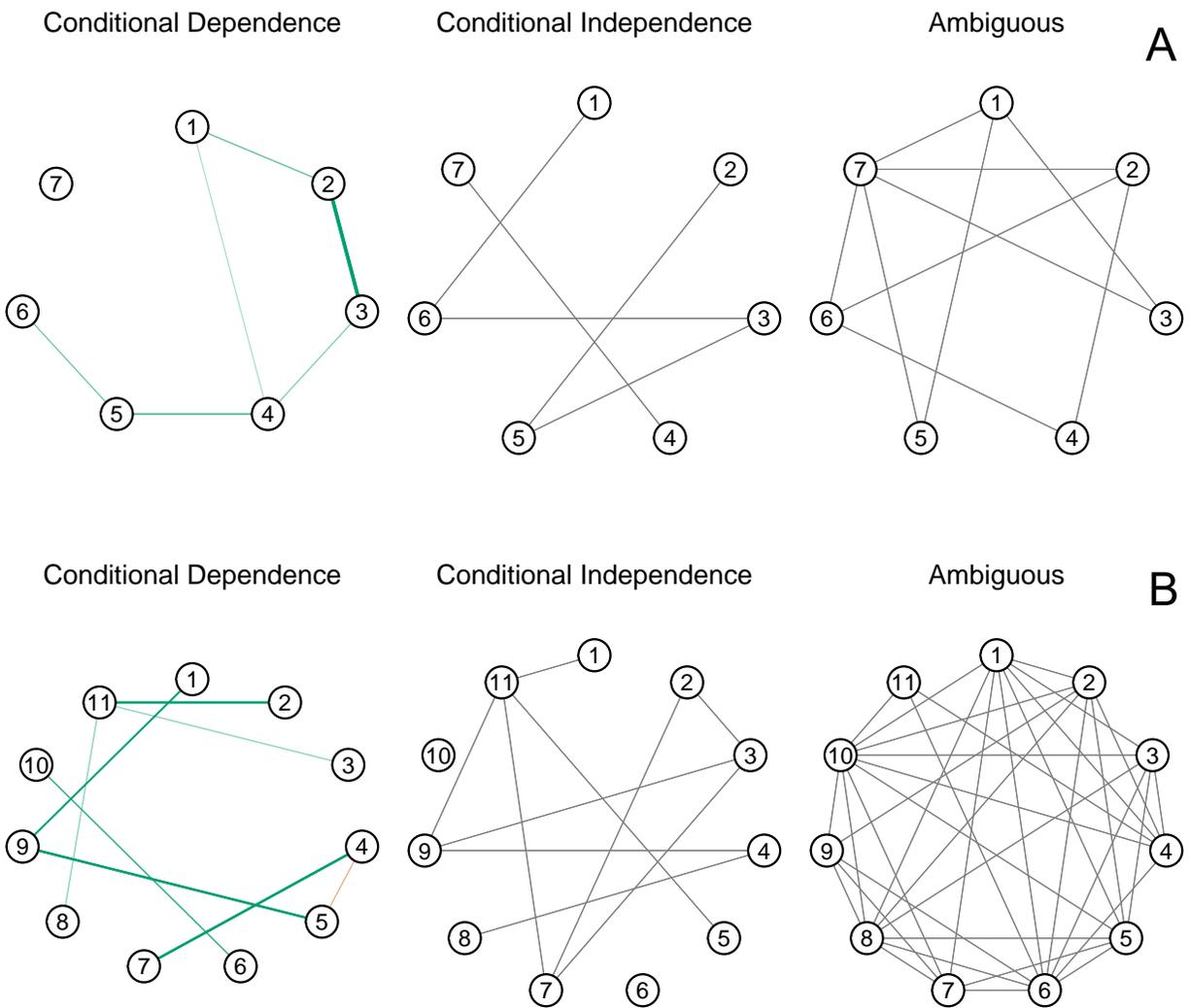
*Figure 1.* A) An illustrative network with three nodes. The connected nodes are conditionally dependent (e.g., A and B), whereas those nodes not sharing a connection are conditionally independent (B and C). In the network literature, it is often suggested to interpret a missing connection in a graph as providing evidence for the null hypothesis of conditional independence. While this is the case for the *true* network structure, in practice network graphs depict the *estimated* conditional dependence structure that includes those edges that happened to be detected: a missing edge could correspond to either conditional independence or an edge that was incorrectly set to zero (a false negative). B) Illustrative network structures that could be estimated, given the true network in panel A. In all but one graph, inferring conditional independence from a missing connection would be incorrect.



*Figure 2.* Illustrative prior distributions. According to [Wysocki and Rhemtulla](#) (see Table 2 therein, [2019](#)), edges are unlikely to be greater than 0.50 in partial correlation networks. This suggests that a small prior standard deviation most accurately captures a priori beliefs about an edge size. Said another way, the wider distributions place an unreasonable amount of prior mass on large edges.



*Figure 3.* A) The estimated conditional dependence structure of 20 PTSD symptoms (see Table X). Green lines represent positive associations, orange lines negative, and thickness the edge size. B) 95% credible intervals for each partial correlation, where those excluding zero correspond to the relations visualized in panel A. The shaded area is a region of practical equivalence ( $\pm 0.10$ ). Notice that none of the intervals are completely contained within that region, which indicates the values set to zero in panel A cannot be interpreted as practically null associations. C) 99% credible intervals for partial correlation differences between each edge in panel A and all relations set to zero. There was not often a difference between a detected and an undetected relation. The take-home message of this figure is that merely visualizing detected effects is a limited source of information, given that those relations set to zero are not reasonably null and those edges included in the network are not necessarily more important (i.e., larger) than the missing edges.



*Figure 4.* A) The estimated networks of seven anxiety symptoms (see Table A2). B) The estimated networks of 11 depression symptoms (see Table A3). Green lines represent positive associations, orange lines negative associations, and the thickness indicates the edges size. The conditional dependence and independence structures include relations determined to be non-zero and zero, respectively, whereas the ambiguous “network” includes relations for which a decision could not be reached (based on a Bayes factor of 3).

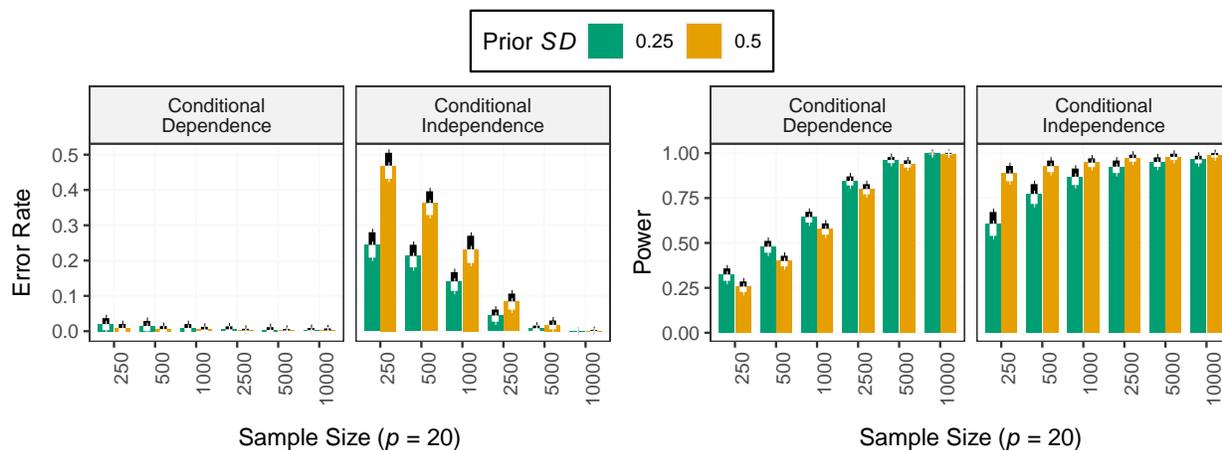


Figure 5. Simulation results averaged across 100 trials. The error bars denote one standard deviation. Conditional independence corresponds to  $\mathcal{H}_0$ , whereas conditional dependence corresponds to  $\mathcal{H}_1$  (Equation 1). The reported error rate is analogous to “specificity” (SPC) which is commonly used to assess performance in simulation studies. It has a direct relationship with the false positive rate (FPR), that is,  $1 - \text{SPC} = \text{FPR}$ . In this case, because the errors refer to incorrectly concluding there is either conditional independence or dependence, we used the generic term “error rate.” Power is analogous to “sensitivity” (SN) that is also used as a performance measure. It has a direct correspondence to the false negative rate (FNR), that is,  $1 - \text{SN} = \text{FNR}$ . Hence it is possible to infer the proportion of edges incorrectly set to zero for a given sample size. Prior  $SD$  is the standard deviation of a beta distribution (Figure 2). Note that the green bar corresponds to the informed prior distribution used in the illustrative examples.

Appendix A  
Node Descriptions

Table A1  
*Post-traumatic Stress Disorder*

| Node | Symptom                            |
|------|------------------------------------|
| B1   | Intrusive Thoughts                 |
| B2   | Nightmares                         |
| B3   | Flashbacks                         |
| B4   | Emotional cue reactivity           |
| B5   | Psychological cue reactivity       |
| C1   | Avoidance of thoughts              |
| C2   | Avoidance of reminders             |
| D1   | Trauma-related amnesia             |
| D2   | Negative beliefs                   |
| D3   | Blame of self or others            |
| D4   | Negative trauma-related emotions   |
| D5   | Loss of interest                   |
| D6   | Detachment                         |
| D7   | Restricted affect                  |
| E1   | Irritability/anger                 |
| E2   | Self-destructive/reckless behavior |
| E3   | Hypervigilance                     |
| E4   | Exaggerated startle response       |
| E5   | Difficulty concentrating           |
| E6   | Sleep disturbance                  |

Table A2  
*General Anxiety Disorder*

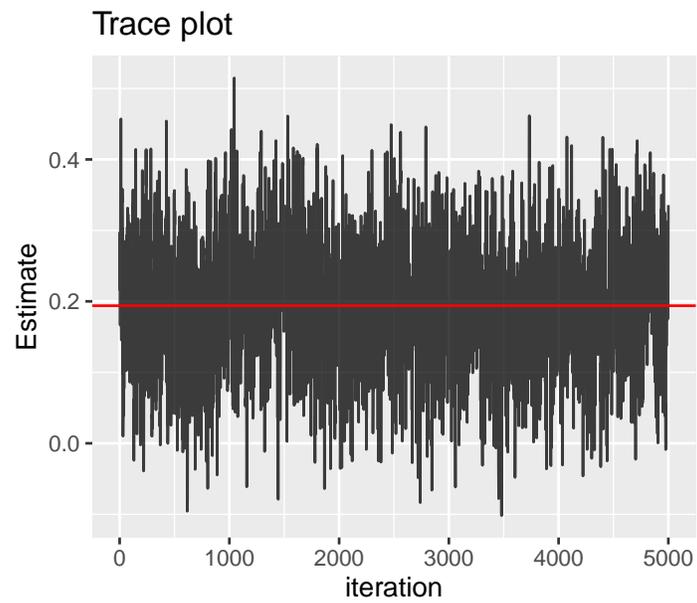
| Node | Symptom   |
|------|---|
| 1    | Feeling nervous, anxious, or on edge              |
| 2    | Not being able to stop or control worrying        |
| 3    | Worrying too much about different things          |
| 4    | Trouble relaxing                                  |
| 5    | Being so restless that it's hard to sit still     |
| 6    | Becoming easily annoyed or irritable              |
| 7    | Feeling afraid as if something awful might happen |

Table A3  
*Depression*

| Node | Symptom                                    |
|------|--|
| 1    | I felt depressed                           |
| 2    | I felt that everything I did was an effort |
| 3    | My sleep was restless                      |
| 4    | I was happy                                |
| 5    | I felt lonely                              |
| 6    | People were unfriendly                     |
| 7    | I enjoyed life                             |
| 8    | My appetite was poor                       |
| 9    | I felt sad                                 |
| 10   | I felt that people disliked me             |
| 11   | I could not get going”                     |

## Appendix B

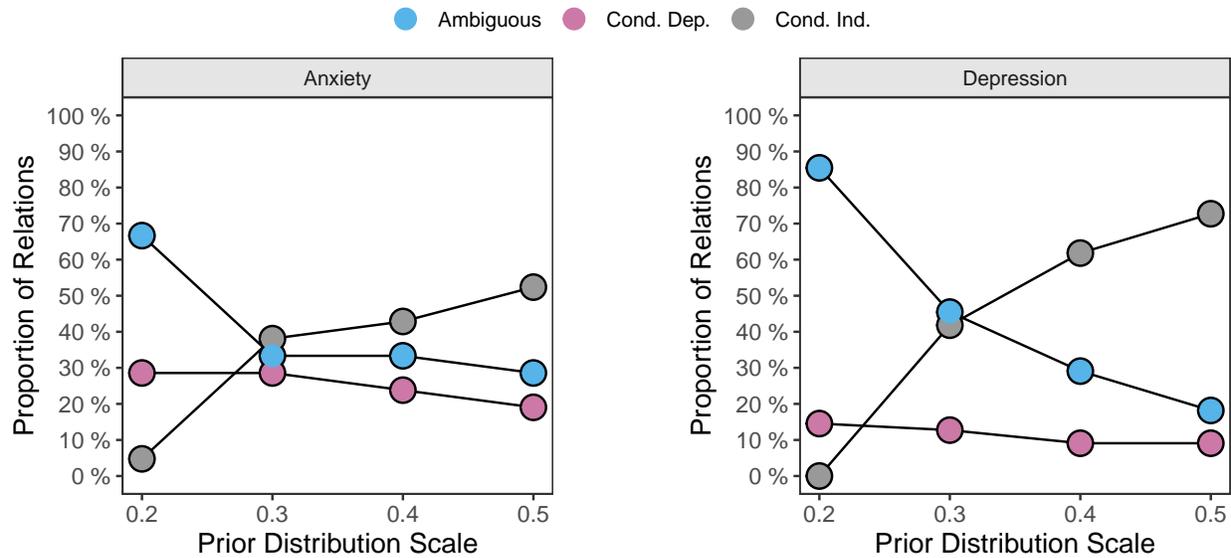
## Model Convergence



*Figure B1.* Illustrative trace plot of Markov Chain Monte Carlo (MCMC) draws for a polychoric partial correlation coefficient.

## Appendix C

## Sensitivity Analysis



*Figure C1.* The proportion of relations included in the ambiguous, conditional dependence, and conditional independence networks, as a function of the prior distribution scale. The Bayes factor cut-off for inclusion in either the conditional independence or dependence networks was three.