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Sampling Variability Is Not Nonreplication: A Bayesian Reanalysis of Forbes, Wright, Markon, and Krueger

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ABSTRACT

Forbes, Wright, Markon, and Krueger claim that psychopathology network characteristics have "limited" or "poor" replicability, supporting their argument primarily with data from two waves of an observational study on depression and anxiety. They developed "direct metrics" to gauge change across networks (e.g., change in edge sign), and used these results to support their conclusion. Three key flaws undermine their critique. First, nonreplication across empirical datasets does not provide evidence against a method; such evaluations of methods are possible only in controlled simulations when the data-generating model is known. Second, they assert that the removal of shared variance necessarily decreases reliability. This is not true. Depending on the causal model, it can either increase or decrease reliability. Third, their direct metrics do not account for normal sampling variability, leaving open the possibility that the direct differences between samples are due to normal, unproblematic fluctuations. As an alternative to their direct metrics, we provide a Bayesian re-analysis that quantifies uncertainty and compares relative evidence for replication (i.e., equivalence) versus nonreplication (i.e., nonequivalence) for each network edge. This approach provides a principled roadmap for future assessments of network replicability. Our analysis indicated substantial evidence for replication and scant evidence for nonreplication.

KEYWORDS

Network analysis; psychopathology; replication; reliability; Bayesian statistics

Introduction

The ability to replicate previous findings is a prerequisite for a self-correcting psychological science. Although not even the most careful and robust science can claim to be perfectly replicable, scientists should strive to improve replicability. Examples of helpful practices include providing publicly available de-identified data and code, shifting values toward high-quality research rather than merely surprising or novel findings, using robust statistical techniques that appropriately model uncertainty and reduce false-positives, and avoiding questionable practices such as "p-hacking" and "HARKing" (Munafò et al., 2017). Encouragingly, such practices seem to be spreading within psychology (Vazire, 2018).

During a similar timeframe, the network approach to mental disorders has emerged as a growing perspective in clinical psychology (for reviews see Contreras et al., 2019; Fried & Cramer, 2017; Robinaugh et al., 2020). The network approach views mental disorders as emergent phenomena arising from causal interactions among symptoms rather than as underlying latent categorical or dimensional entities functioning as the common causes of the symptoms signifying their presence (Borsboom, 2017). Proponents of network theory have commenced their investigation into complex systems of psychopathology by estimating cross-sectional dependence graphs among symptoms and other aspects of mental disorders (Contreras et al., 2019).

As in any area of research, psychological network analysts must develop best practices for producing replicable and reliable results. Indeed, this concern drove the introduction of network regularization (Epskamp & Fried, 2018), permutation testing for network differences (van Borkulo et al., 2017), network bootstrapping (Epskamp et al., 2018), and Bayesian network estimation (Williams et al., 2018). After

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analyzing two psychiatric epidemiology data sets, Forbes et al. concluded that "psychopathology networks have limited replicability" (Forbes et al., 2017a, p. 969; Forbes et al., 2017b). In a reanalysis of the data, Borsboom et al. (2017, p. 990) concluded that the data "supported the exact opposite of [Forbes et al.'s] conclusion: Psychopathology networks replicate very well."

Revisiting this controversy with new data and arguments, Forbes et al. (2019) repeat their claim that network characteristics in psychopathology have "poor replicability" (p. 1) or "limited replicability" (p. 4). In support of their conclusion, they performed network analysis on two waves of data from an observational study on depression and anxiety, as well as four data sets on posttraumatic stress disorder (PTSD). They first use extant network analytic methods for assessing replicability, showing that network characteristics are generally stable and robust. They then use their alternative "direct metrics" (p. 1) for assessing replicability to support their claim that psychopathology symptom network characteristics have limited replicability. The purpose of our commentary is to discuss three apparent flaws in Forbes et al.'s critique, and to offer a Bayesian alternative to the direct metrics devised by Forbes et al.

First, observed differences across empirical data sets cannot provide evidence for or against the use of a *method,* regardless of how rigorously conducted. Observed differences across data sets may not only signify meaningful differences between samples (i.e., nonreplication) but they also may signify random sampling variation, poor reliability in measurement, or a variety of other explanations. However, adjudicating among these possibilities is nontrivial and unaddressed by Forbes et al.

Second, they incorrectly assert that the removal of shared variance between variables via statistical control (e.g., use of partial correlations) inherently leads to reduced reliability. In fact, appropriate statistical control increases reliability. Statistical control only reduces reliability when used inappropriately vis-à-vis the underlying causal model, which remains unknown in this case.

Third, their direct metrics presuppose that any variation in parameter estimates across samples signifies nonreplication. Yet sampling inevitably results in departures from invariance; even if two samples are derived from the same population, one cannot expect them to be equivalent. Explicitly modeling the amount of expected variation is necessary for any meaningful interpretation of such "direct metrics." Without this context, it is impossible to tell whether a difference in a parameter estimate in a second data set (i.e., a "direct metric") provides information inconsistent with its estimate in the first data set (i.e., nonreplication). Accordingly, we provide a Bayesian analysis that quantifies this uncertainty, providing a statistically sound roadmap for future researchers. Our reanalysis of Forbes et al.'s (2019) data revealed substantial evidence for replication for network edges and very little evidence for nonreplication.

The problem with evaluating statistical methods with empirical data

Forbes et al. use both established and novel methods to evaluate the replicability of several data sets, concluding that network analysis is not a replicable method. Unfortunately, regardless of whether Forbes et al. use the existing suite of methods or alternative metrics of replication, the very premise of evaluating a method by using empirical data is problematic.

Consider the following thought experiment: Researcher A measures two psychological variables in a given sample. Upon performing a multiple linear regression controlling for several other key variables, he concludes that the two variables are significantly related. Researcher B then measures the same variables in the same group of individuals sometime later. After repeating the identical analysis, she finds that the two variables are not significantly related. Researcher B will likely consider multiple hypotheses that could explain the discrepant results (e.g., true differences between timepoints, random sampling variability, unreliable measurement), but she will not conclude that multiple linear regression *per se* is an unreliable statistical method with limited replicability.

Methods can best be evaluated via systematic simulations when investigators can directly control the model generating the simulated data. Importantly, the generating properties of simulations are known to investigators, whereas those of empirical data are not. Accordingly, simulations can establish the statistical confidence associated with a parameter given certain assumptions. Forbes et al. criticize previous simulation studies for bearing "little resemblance to … real world psychopathology data" (p. 16) and suggest that the performance of network methods should be evaluated via simulations based on real-world psychopathology network structures. Such inquiry would usefully add to the growing body of network simulation studies (e.g., Epskamp et al., 2018; Williams et al., 2019). In contrast, further arguments about network methods

based on empirical data alone are unlikely to be productive.

Does statistical control reduce reliability?

Forbes et al. claim that statistical control via removal of shared variance inherently diminishes reliability. This claim is incorrect. For example, imagine that we are interested in assessing an individual's basal blood pressure. If we control for relevant covariates, such as recent caffeine consumption and physical activity, we will *increase* the reliability of our assessment over repeated measurements, not decrease it. These causal covariates affect momentary blood pressure measurements, and removing shared variance increases the reliability of assessment of basal blood pressure. Moreover, adjusting for these causal covariates will increase the reliability of predicted outcomes (e.g., high basal blood pressure predicting heart attacks).

On the other hand, statistical control can indeed lead to unreliable results in other causal models. For example, statistically controlling for the presence of thunder would lead to an unreliable assessment of lightning (i.e., leaving only measurement error or lightning seen by the deaf). Forbes et al. conclude that statistical control leads to unreliability, but this is only true given certain assumptions regarding the underlying causal model. Statistical control can lead to either increased or decreased reliability depending on the true causal structure among variables.

Interpreting all variability as nonreplication

When estimating parameters that pertain to a sample (or samples), statisticians must carefully correct for random sampling variability before making assertions about the population. A familiar example is a null hypothesis significance test—if the p-value falls below a certain threshold, researchers have minimum justification for making an inference about the population; otherwise they cannot.

Forbes et al. overlooked this difference when making claims about the nonreplication of networks in their samples. Their direct metrics conflate sampling variability and true variability, making them uninterpretable. They regard any difference in the presence or direction of an edge between the two networks as a genuine difference between them. Ironically, this means that the direct metrics presented by Forbes et al. are themselves not statistically replicable. As shown by the permutation test employed in their own analysis (p. 14), none of the direct metrics of differences between the two networks met a minimum threshold of statistical significance. In other words, Forbes et al. make claims about the overinterpretation of network parameters by interpreting parameters that are themselves statistically nonsignificant.

We suspect that most of these "changes" between networks arose from ordinary fluctuation between the samples. For instance, imagine that the true value of a given edge in a generating model is 0.005. Even if this edge were evaluated across several very large samples, it would fluctuate between a negative and positive value (or fluctuate between a zero and nonzero value in a regularized network). In this scenario, the variability is entirely due to expected sampling error, rather than to any inherent unreliability of partial correlations in psychological data.[1] Unfortunately, their direct metrics of replication conflate these sources of variability. We incorporate this key point into the following analysis to perform a statistically principled test of replication between the networks.

Operating within a Bayesian framework, Williams et al. (2020) have devised an alternative direct test of replicability across samples that incorporates uncertainty. This method directly accounts for normal variations in sampling and provides a Bayes Factor assessing the degree of evidence for either equivalence or nonequivalence (Williams et al., 2020). It resembles the Network Comparison Test permutation method that generates a p-value for each edge comparison, noting when edges significantly differ between networks. However, this method can assess *relative* evidence between competing models which allows for richer inference than merely rejecting or failing to reject the null hypothesis. This is accomplished by viewing replication in terms of predictions. On the one hand, there is a restricted model (H_1) that predicts replication (equivalence), whereas on the other hand, there is an unrestricted model (H_2) interpretable as "not H_1" (nonequivalence/nonreplication).

Using the BGGM R package (Williams & Mulder, 2019a), we computed Bayes Factors (H_1 = equivalence, H_2 = nonequivalence) for each pairwise partial correlation in the depression and anxiety samples furnished by Forbes et al.[2] These methods are introduced

[1]As a side note, the expected sampling variability of estimates can be quantified using *expected network replicability*, which calculates the expected replicability assuming the data indeed arises from a network structure (i.e., when assumptions are *not* violated; see Williams, 2020). In a preliminary analysis, we computed thesample size needed to achieve an expected network replicabilityof 80% given the present network structure. We estimated thata sample size of roughly 1200 would be needed (or 2800 ifBonferroni corrections were used).

[2]We focused on the depression and anxiety sample data because it was indeed sampled from the same population, albeit at different time points.

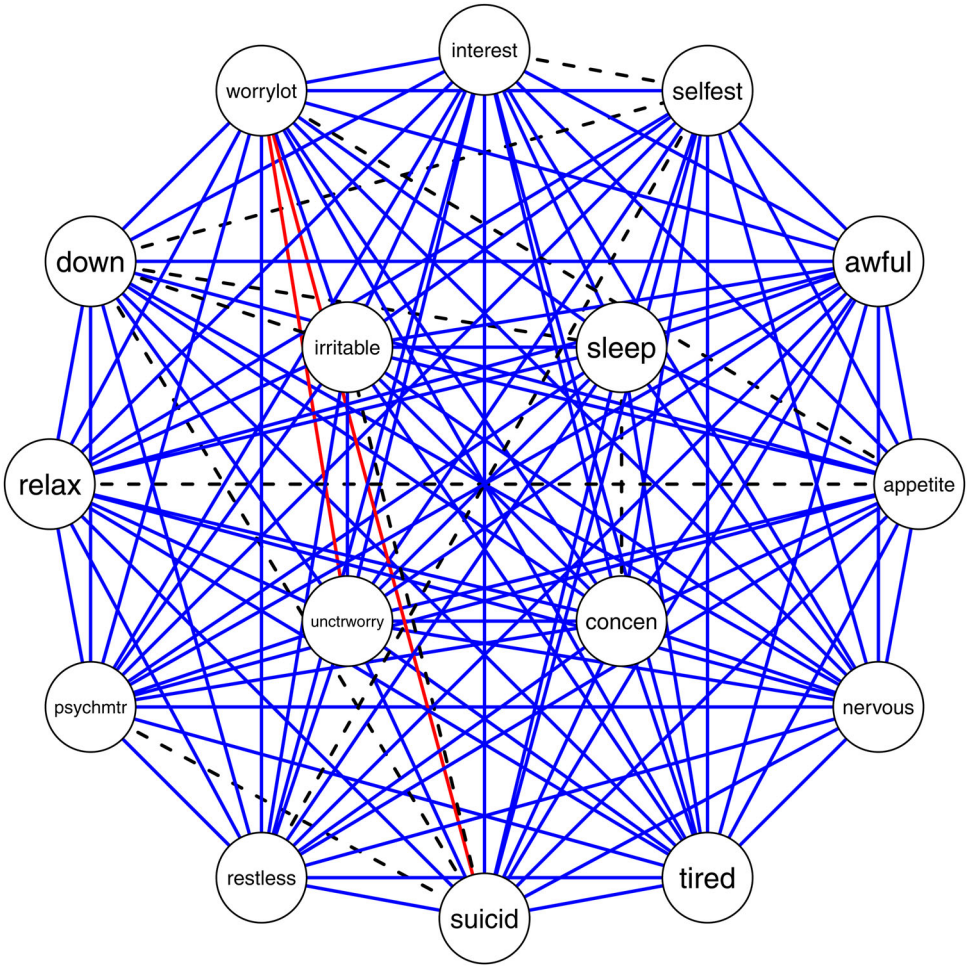

Figure 1. Relative evidence for replication or nonreplication using Bayes Factors. Replicated edges appear in solid blue and non-replicated edges appear in solid red. Edges that did not reach substantial evidence for either hypothesis are in dotted black.

in greater detail in Williams and Mulder (2019b) and Williams et al. (2020). To test differences between partial correlations, the method uses the novel matrix-F prior distribution which is a generalization of the customary Wishart prior distribution for the inverse of the covariance matrix (X). The prior distribution for a given partial correlation is approximately Beta($\frac{\delta}{2}$, $\frac{\delta}{2}$), where δ governs the width of the prior distribution. The idea is to first estimate the partial correlations for each group, compute the posterior distribution for the partial correlation difference, and then use the implied prior distribution of that difference to test the exact equality constraint of group equality. For each model, we drew 5,000 samples from the posterior distribution with a Gibbs sampler implemented in BGGM. The chains converged, as indicated by scale reduction factors below 1.10 (Brooks & Gelman, 1998). Convergence was also determined by visually inspecting the trace plots, available in the Supporting Information.

We first considered an unrestricted model that was essentially agnostic to the size of the partial correlations—i.e., a nearly uniform distribution between —1 and 1 (Marsman & Wagenmakers, 2017). This was accomplished by setting $\delta = 3$ which results in a Beta prior distribution that is zero centered with a standard deviation of 0.50. For convenience of interpretation, we used a cutoff (*Bayes Factor* = 3) to indicate evidence in favor (or opposed) to the edges being equivalent. These results appear in Figure 1. For 89% of edges, Bayes Factors indicated evidence in favor of the hypothesis that the edges were equivalent between the two samples. For 9% of the edges, the Bayes Factors indicated that there was insufficient information to conclude in favor of either equivalence or non-equivalence. We found evidence in favor of

On the other hand, the PTSD data differ in numerous non-trivial ways, including country of origin, trauma type, and gender composition.

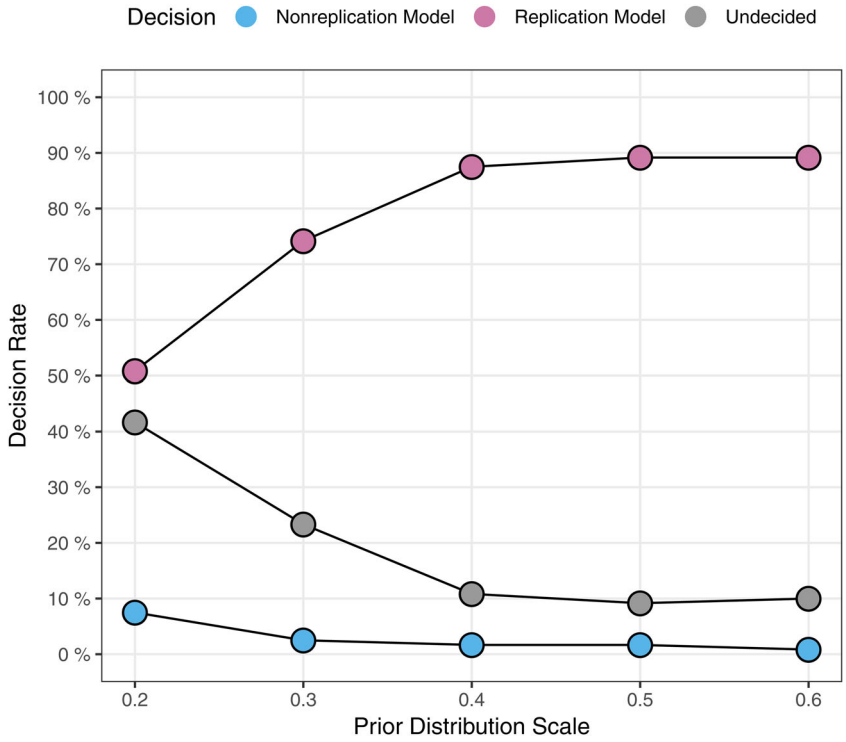

Figure 2. Sensitivity analysis for the Bayesian analysis. The decision rate (*y*-axis) is the proportion of edges (out of 120) that supported either the replication model, the nonreplication model, or neither (Undecided). The width of the unrestricted model (*x*-axis) is the standard deviation of a beta distribution between ± 1. Thus, larger values approach a uniform distribution.

nonequivalence for only two edges (i.e., less than 2% of the total edges). The exact values of Bayes Factors are tabulated in the Supporting Information. We also conducted a sensitivity analysis to assess the influence of modifying the Bayes Factor threshold, indicating results consistent with our interpretation that evidence for nonequivalent edges was rare. A figure displaying this sensitivity analysis is available in the Supporting Information.

These results are influenced by assumptions made regarding the unrestricted model[3] (Carlsson et al., 2017). This is an advantage, not a limitation. That is, in this case, we can rigorously evaluate the competing replication and nonreplication models across a range of assumptions. Results across a broader range of assumptions appear in Figure 2.[4] The nonreplication model indicates robustness, in that, at *most*, nonreplication was supported for 7% of the edges. On the other hand, the replication model was more sensitive to the choice of prior distribution, with the support ranging from approximately 50 to 90%. In other words, varying the assumptions seemed to change whether sufficient evidence emerged for edge replication (versus insufficient evidence, "undecided"). Finding insufficient evidence for some edges is unsurprising given the limited power of the data sets. In addition, we tested each partial correlation individually, which presents the issue of multiple comparisons. However, applying a correction would simply result in more inconclusive evidence (i.e., "undecided"), further suggesting that the sample is insufficient to make strong claims about network replicability. In summary, across the various choices in assumptions, we found little evidence indicating nonreplication in the depression and anxiety samples.[5]

Together, these results complement key aspects of this work. First, in direct opposition to Forbes et al.'s claim that "key differences … indicated limited

[3]Note that nonreplication will also be influenced by the chosen alpha level, and in the case of regularized estimation, there are many factors that influence performance (Williams et al., 2019).

[4]We analyzed the data assuming both continuous and ordinal data. The presented results were robust to this choice, and as such, we presented those from assuming continuous data.

[5]We did not have access to the original data for the PTSD networks and were therefore unable to conduct tests directly on these data. Simulating data based on sample size and correlation matrices allowed for an approximate test, in which we tested relative evidence for two hypotheses: (1) that the edges were equivalent across all four networks [omnibus equality] or (2) that the edges differed in at least one network. The test yielded substantial evidence for replication for 90 edges and substantial evidence for heterogeneity (i.e., omnibus test across all four networks) for 19 edges. Results of the robustness analysis for the PTSD networks appear in the Supporting Information.

reliability and replicability in the networks" (p. 13), we found very little evidence for the nonreplication model in these data. The replication model fared much better. At best, there was overwhelming support for replication between the two networks overall, consistent with the conclusion of Borsboom et al. (2017). At worst, the results point toward either the replication model or neither model ("undecided", i.e., an insufficient sample size to determine replication or nonreplication). This again stands in direct contrast to the claims of Forbes et al.

Conclusion

Although it makes sense to ask whether network analytic methods are suitable for psychopathology data, the analysis by Forbes et al. is uninformative for several reasons. First, empirical results cannot directly inform statistical practice, even in the best of scenarios. Carefully controlled simulations are necessary. Second, the impact of statistical control on reliability depends on the causal structure of the data. If psychopathology symptoms arise from a common source, the statistical control employed in network analysis would indeed be problematic. However, if psychopathology symptoms instead influence one another in a causal system, as hypothesized in the network theory of mental disorders, appropriate statistical control could increase reliability. Third, the data presented by Forbes et al. do not show evidence for nonreplication in the first place. Their direct metrics overestimate differences across samples by counting *any* change in sign or regularization as evidence for nonreplication, conflating nonreplication with normal sampling variability. Such changes are expected due to normal variation across samples, especially for edges that have a true value close to zero. When taking normal sampling variation into account, Bayesian hypothesis tests indicated substantial evidence for replication and very little evidence for nonreplication in the primary analysis.

Researchers have a powerful suite of methods to perform tests on the stability and replicability of network analyses. These methods continue to evolve as they are vetted in various simulated scenarios. We expect that significant heterogeneity exists within psychopathological systems; identifying and studying it is a major goal of network analysis. Network researchers should continue to calculate and explicitly report stability metrics, confidence intervals, and other validated measures of reliability. Moreover, they should judiciously select nodes and interpret parameter estimates carefully. In conclusion, although psychological network analysis faces many challenges, we find no evidence that limited replicability is among them.

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