The Impact of Criteria-based and Data-driven Sampling Approaches on the Heterogeneity and
Interpretability of Posttraumatic Stress Symptom Networks

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Abstract

Background. The application of psychopathological symptom networks requires reconciliation of the observed cross-sample heterogeneity. We leveraged the largest sample to be used in a PTSD network analysis ($N = 28,594$) to examine the impact of criteria-based and data-driven sampling approaches on the heterogeneity and interpretability of networks.

Methods. Severity and diagnostic criteria identified four overlapping subsamples and cluster analysis identified three distinct data-derived profiles. Networks estimated on each subsample were compared to a respective benchmark network at the symptom-relation level by calculating sensitivity, specificity, correlation, and density of the edges. Negative edges were assessed for Berkson's bias, a source of error that can be induced by threshold samples on severity.

Results. Criteria-based networks showed reduced sensitivity, specificity, and density but edges remained highly correlated and a meaningfully higher proportion of negative edges was not observed relative to the benchmark network of all cases. Among the data-derived profile networks, the Low Severity network had the highest proportion of negative edges not present in the benchmark network of symptomatic cases. The High Severity profile also showed a higher proportion of negative edges, whereas the Medium Severity profile did not.

Conclusion. Although networks showed differences, Berkson's bias did not appear to be a meaningful source of systematic error. These results can guide expectations about the generalizability of symptom networks across samples that vary in their ranges of severity. Future work should continue to explore whether network heterogeneity is reflective of meaningful and interpretable differences in the symptom relations from which they are composed.

Introduction

In the last decade, network analysis has been increasingly used to characterize psychological disorders in general (Robinaugh et al., 2020) and posttraumatic stress disorder (PTSD) in particular (Birkeland et al., 2020). The conceptualization of symptoms as causally interconnected nodes in a network provides a compelling alternative to the disease model, which frames symptoms as indicators of an underlying biological (brain) disorder (Borsboom et al., 2019). The former posits that posttraumatic stress symptoms activate and maintain each other, giving rise to “PTSD” (McNally, 2017; McNally et al., 2015), and the latter defines “PTSD” as the neurobiological illness that gives rise to posttraumatic stress symptoms (Insel & Cuthbert, 2015). The network approach can be used to empirically examine clinically intuitive feedback loops (e.g., nightmares impact cognition and mood through their effect on sleep), and to generate novel hypotheses through the identification of links that are imperceptible to even the most gifted clinicians (Borsboom et al., 2016; Eronen, 2019). However, this depends crucially on the ability of network models to capture accurate connections, and the application of findings requires reconciliation of the heterogeneity observed across psychopathological networks in general, and PTSD networks in particular (Armour et al., 2017; Fried & Cramer, 2017; Guloksuz et al., 2017).

Several recent works illustrate the diversity of network comparison methods and highlight the challenge of drawing firm conclusions from the extant PTSD network literature. A comparison of four treatment-seeking samples applying severity cutoffs or diagnostic criteria detected significant differences in the omnibus tests; however, the number of individual edges that were significantly different between networks ranged from 2-8 (out of 120 potential edges), and the correlation of edge weights ranged from .62 to .74 (Fried et al., 2018). A recent review of 20 PTSD network analyses found that only a few edges (e.g., startle - hypervigilance, anhedonia

- detachment, thought avoidance - cue avoidance, and sleep - nightmares) were consistently prominent across networks, defined as edges that were significantly different than two-thirds of the edges in the same network (Birkeland et al., 2020). A potential source of network heterogeneity that was not systematically analyzed in the abovementioned work was the variability in symptom-related inclusion criteria. Across PTSD networks, such criteria have ranged from the inclusion of all cases even if they reported no symptoms (e.g., Sullivan et al., 2018), to various severity thresholds such as symptomatic (i.e., a total severity greater than zero) (e.g., Benfer et al., 2018), a minimum total score for probable PTSD (e.g., Birkeland et al., 2017), DSM diagnostic criteria (e.g., Cao et al., 2019), or both severity and diagnostic requirements (e.g., McNally et al., 2015). Therefore, our overarching aim was to systematically investigate the impact of criteria-based and data-driven sampling approaches on the heterogeneity and interpretability of PTSD symptom networks.

We based our approach on a recent simulation study that found depression symptom networks were prone to Berkson's bias, or the induction of spurious negative relations between variables in samples that have applied cutoff scores derived from those variables; simulated networks that applied higher severity thresholds had more negative edges and were less sensitive and specific in their overall detection of edges compared to the "true" network estimated on an extant dataset of participants in a large clinical trial (De Ron et al., 2019). The authors stated Berkson's bias posed a major concern for network analyses of clinical samples selected on severity thresholds calculated from the symptoms comprising the network and proposed several alternatives, including data-driven identification of distinct symptom profiles (De Ron et al., 2019). Although data-driven PTSD profiles have typically reflected differences in overall severity and provided evidence for a dissociative subtype (Hansen et al., 2017), this approach

may overcome the problem of Berkson's bias because participants are clustered on multivariate patterns of symptom relations, as opposed to sharp boundaries of severity. We leveraged the largest sample to be used in a PTSD network analysis ($N = 28,594$) to systematically test the impact of criteria-based and data-driven sampling strategies on PTSD symptom networks and examine whether any resulting network heterogeneity was primarily a product of Berkson's bias.

Methods

Reproducibility and Ethics

The Institutional Review Board of the University of Texas at Austin determined that this secondary analysis was exempt from IRB oversight. All analyses described below were conducted in R, a free open-source programming language (R Core Team, 2013). Analytic scripts and detailed descriptions of the analysis environment (e.g., package versions) are provided in the Open Science Foundation website for this project (osf.io/7nucs/). The full dataset, codebook, description of procedures, and summary statistics of all collected data have been made publicly available on the United Nations's Redatam server (www.redatam.org).

Participants and Measures

On February 27, 2010, an 8.8 magnitude earthquake and tsunami impacted individuals living in Chile (Lorito et al., 2011). Between May and June 2010, Chile's Ministry of Planning and Cooperation conducted fully-structured face-to-face interviews with a total of 28,594 individuals in the affected areas. As part of the post-earthquake interview, 28,121 individuals completed the Davidson Trauma Scale (DTS) which assessed the 17 DSM-IV PTSD symptoms on a frequency scale ranging from "not at all" to "every day" and a severity scale ranging from "not at all distressing" to "extremely distressing" (Davidson et al., 1997). The DTS demonstrated excellent internal consistency ($\alpha = .93$) in a previously published psychometric analysis of this dataset

(Leiva-Bianchi & Araneda, 2013). The structure and scoring of the DTS parallels the Clinician Administered PTSD Scale (CAPS) (Blake et al., 1995), and the two have shown high convergent validity (Davidson et al., 1997; Weathers et al., 2001).

Statistical analyses

Criteria-based networks

We applied criteria used in previous network analyses of PTSD symptoms to identify four subsamples and compare their network structures to a benchmark network estimated on the complete sample. The symptomatic subsample excluded participants with zero total scores on the DTS, and three subsamples with probable PTSD were identified by applying either a total severity cutoff score of 40, DSM-IV criteria, or both. Probable PTSD based on DSM-IV criteria (i.e., at least one re-experiencing symptom, three avoidance or numbing symptoms, and two hyperarousal symptoms) was determined by applying common scoring convention: a symptom was considered “present” if participants reported a frequency of at least “once or twice” and a severity of at least “moderate” (Davidson et al., 1997; Weathers et al., 2001) .

Data-derived profile networks

To identify distinct profiles based on multivariate patterns of individual symptom severity, we performed a cluster analysis, which estimates finite mixture models using continuous variables, compares various fit criteria between models with differing number of cluster solutions (i.e., profiles) and assigns profile membership to each case once the best-fit model is identified (Scrucca et al., 2016). Once the profiles were identified, the distributions of individual- and overall-symptom severity were compared to assess differences between profiles. Participants that did not report any symptoms ($n = 11,090$, 38.78%) were excluded from the clustering analysis.

Network analyses

Gaussian graphical models (GGM) were estimated from symptom scores (the sum of frequency and severity) with the graphical lasso approach, which applies least absolute shrinkage and selection operator regularization with Extended Bayesian Information Criterion to reduce weak (potentially spurious) edges to zero (Epskamp et al., 2018). Edge weights from the network analysis quantify the strength of the partial correlation between each node-pair in the network. In this PTSD symptom network, there are 136 potential edges corresponding to the number of unique connections between symptom pairs.

Network Comparisons

We based our approach on a previous examination of the impact of Berkson's bias on depression symptom networks (De Ron et al., 2019): the authors used a "true network" of depression symptoms from an extant dataset of real participants to simulate new datasets, applied different severity thresholds to subset the simulated datasets, and compared each corresponding network to the true network. For our analyses, we examined how symptom-related inclusion criteria impacted networks estimated on subsamples taken from the real dataset (as opposed to simulating data). First, a benchmark network was estimated: for the criteria-based analyses the benchmark network was estimated on the complete sample, and for the data-driven profile analyses the benchmark network was estimated on the complete symptomatic sample from which the profiles were identified. For each of the criteria-based and data-derived samples we estimated 1,000 networks in a bootstrapping procedure that compared each estimated network to its respective benchmark model on sensitivity (proportion of edges present that are also present in the benchmark network), specificity (proportion of edges absent that are also absent in the benchmark network), Pearson correlation of edge weights, Spearman correlation of edges ranked by their strength, density (the number of edges in the comparison network divided by the number

of edges in the benchmark network), and the proportion of negative edges that were not present in the benchmark model relative to the number of potential edges. The distribution of each metric calculated on each of the 1,000 network comparisons was represented in boxplots (median and quantiles).

Results

Criteria-based networks

Among the 28,594 respondents in the complete sample, 17,504 (61.22%) were symptomatic, 3,474 (12.15%) had probable PTSD based on severity criteria, 2,090 (7.31%) had probable PTSD based on DSM-IV diagnostic criteria, and 1,982 (6.93%) had probable PTSD based on both severity and diagnostic criteria. Figure 1 shows results of the comparisons between the full-sample network and each of the networks estimated on subsamples varying in their diagnostic inclusion criteria. The symptomatic network (i.e., cases with a total severity greater than zero) had significantly higher sensitivity, specificity, correlation, and density, than the three networks that applied criteria for probable PTSD. However, each of these metrics remained relatively high, indicating a high degree of similarity across networks regardless of the symptom criteria applied. Moreover, the proportion of negative edges not present in the benchmark model was low in each of the subsample networks.

Data-derived profile networks

The clustering algorithm identified three distinct symptom profiles in the sample. The severity distribution of individual symptoms (Fig. 2a) and total PTSD severity (Fig. 2b) suggested that among the 17,504 symptomatic respondents, 3,514 (20.08%) had a Low Severity profile, 7,893 (45.09%) had a Medium Severity profile, and 6,097 (34.83%) had a High Severity profile. Figure 3 shows the network structure of the symptomatic sample and each of the data-derived symptom

profiles. While several symptoms are linked consistently across all four networks (e.g., cue avoidance - thought avoidance, anger - concentration, and hyperarousal - startle), the Low Severity profile network exhibits higher overall density and more negative connections. Figure 4 shows comparisons between the symptomatic sample network and each of the data-derived profiles. All three profiles showed high sensitivity, specificity, and density, suggesting comparable detection of edges across these networks compared to the symptomatic sample network. However, only edges in the Medium- and High Severity networks were strongly correlated with edges in the symptomatic sample network. Consistent with the network plots in Figure 3, the Low Severity network had a significantly higher density and higher proportion of negative edges, followed by the Medium, and High severity network.

Discussion

We leveraged the largest sample to be used in a PTSD network analysis to examine the impact of criteria-based and a data-driven sampling approaches on symptom networks. A few key findings emerged. First, the application of severity criteria commonly used to limit samples to probable PTSD cases reduced the sensitivity, specificity, and density of networks compared to a benchmark model that included all cases, but edges remained highly correlated across networks, and thresholding samples on severity did not induce a meaningfully higher proportion of negative edges (i.e., Berkson's bias). Several factors may have contributed to the notable difference between our results and the simulation study of Berkson's bias in depression networks (De Ron et al., 2019), including the disorder under examination (PTSD vs. depression), the way networks were estimated (real data vs. simulated data), and the type of severity criteria applied (cutoffs and/or diagnostic criteria for probable PTSD vs. cutoffs for mild, moderate, severe, and very severe depression). In the simulated depression networks (De Ron et al., 2019), Berkson's

bias had a meaningful impact on negative edges when the “severe” or “very severe” cutoffs were applied, which correspond to 38% and 56% of the maximum score, whereas our study applied a diagnostic cutoff score that corresponded to approximately 29% of the maximum score. While it is possible that applying a higher threshold in our analysis may have induced Berkson’s bias, we used threshold criteria from the literature and there are no PTSD symptom network studies that applied severe or very severe cutoffs. Our results do not provide strong evidence of spurious negative edges in networks estimated on clinical PTSD samples.

The data-derived subsamples were similar in density to the benchmark model of symptomatic cases, but each had lower sensitivity and specificity, indicating a difference in the specific edges that were (or were not) detected. Consistent with the observable differences in the network graphs, edge correlation analyses demonstrated that the Low Severity network was the most divergent, driven by the high proportion of negative edges that were not present in the benchmark network. The High Severity profile also showed a higher proportion of negative edges, whereas the Medium Severity profile did not. While these data-derived profiles were not based on cutoff scores, all cases in the Low Severity profile had a total score less than 11; therefore, cases with high severity on some symptoms would need to have low scores on other symptoms. If this pattern was consistent across specific symptom pairs, then it would yield negative associations. On the other hand, the High Severity profile had a median total severity of 44 and a range of 4-136, indicating participants in this profile were approximately equally distributed below or above the diagnostic threshold of 40 used in the criteria-based analyses. Therefore, it seems less plausible that the negative edges in the High Severity profile were artifacts of the total severity characteristics.

Berkson's bias is a problem because it induces spurious negative links between variables that would otherwise have no association (or even a positive association). Across our analyses, networks showed differences including the presence of negative edges that did not exist in the benchmark models, but Berkson's bias did not appear to be a meaningful source of systematic error. How, then, should negative edges be interpreted in these PTSD symptom networks, and more broadly speaking, which of the sampling approaches is optimal for uncovering the "true" PTSD network? If network models accurately capture, then one way to make sense of a nonspurious negative association is to consider whether the activation of one symptom can inhibit the activation of another. While it is unclear how emotional, cognitive, or physiological symptoms of PTSD can have inhibitory links, avoidance symptoms are plausible avenues for negative associations in the network given that the explicit goal of these behaviors is to inhibit the traumatic memory. We see an example of this in the Low Severity network, where a strong negative link was detected between *avoidance of thoughts* and *intrusive thoughts*, yet in the High Severity profile, these two symptoms are positively associated. Individuals in the Low Severity profile may be able to successfully reduce the frequency or severity of intrusive thoughts through avoidance, whereas in the High Severity profile, avoidance and intrusions form a pathological feedback loop, which represents a key theoretical feature of the PTSD model, and one of the explicit targets of exposure therapy, the first line treatment for PTSD. While these are *post hoc* interpretations of our results, the adaptive value of avoidance has been examined in the context of naturalistic research on recovery from natural disasters or the loss of a loved one (Bonanno & Kaltman, 2001; Many et al., 2012), experimental psychopathological research on trauma memory (James et al., 2016), and treatment formulation (Hofmann & Hay, 2018). A challenge for future research is to test *a priori* hypotheses about changes in the direction of association

between specific symptoms, which may be relevant in the transition between low and high severity (or subclinical to clinical) states - a topic that has seen renewed interest (Nelson et al., 2017; Wichers et al., 2019)

Several things need to be taken into account in the interpretation of these results. An uncommon feature of this PTSD dataset, in addition to the large sample size, is that symptoms were assessed around the same time among individuals living in the same region in reference to the same index trauma; this may have reduced factors that typically contribute to network heterogeneity in other research, including differences in trauma type and time since trauma. Therefore, it is not clear how these and other additional sources of sample heterogeneity may interact with symptom-related sampling criteria to impact PTSD network structures. Although similar methods (e.g., between-networks edge correlations) have been used to compare networks in other disorders (e.g., De Ron et al., 2019), this metric is sensitive to the number of edges in a given network, which is a direct product of the number of symptoms. The number and complexity of symptoms involved in the diagnostic criteria varies across disorders, the number of symptoms in a network can vary for the same disorder depending on the instrument used, and even the suggested severity cutoffs can vary for the same instrument. Therefore, it is likely that the impact of criteria-based and data-driven sampling on symptom networks varies across disorders. Moreover, data-driven sampling may not yield similar profiles across samples, and large datasets may be necessary to identify profiles with sufficient cases for robust network estimation.

In conclusion, we examined PTSD symptom networks in subsamples based on either symptom-related criteria applied in previous research or data-derived symptom profiles. Networks showed differences across these analyses but Berkson's bias did not appear to be a

meaningful source of systematic error. These results can guide expectations about the generalizability of symptom networks across samples that vary in their ranges of severity. Future work should continue to explore whether network heterogeneity is reflective of meaningful and interpretable differences in the symptom relations from which they are composed (Borsboom et al., 2018).

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Conflicts of interest

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Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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Fig. 1. Network comparisons between the complete sample and criteria-based subsamples. The complete sample ($N = 28,594$) network served as the benchmark for each comparison.

Bootstrapped analyses (1,000 replications) were performed on subsamples applying the following criteria: severity > 0 (symptomatic, $n = 17,504$), severity > 40 (probable PTSD, $n = 3,474$), DSM-IV symptom criteria (probable PTSD, $n = 2,090$), severity > 40 and DSM-IV symptom criteria (probable PTSD, $n = 1,982$). Networks from each bootstrap replication were compared to the benchmark model on sensitivity, specificity, correlation, density, and proportion of negative edges. The boxplots represent the distribution of results from those comparisons.

A) Symptom-Level Severity Distributions for Each Data-Derived Profile

B) Total Severity Distributions for Each Data-Derived Profile

Fig. 2. The boxplots (a) show the distribution of individual symptom scores and the histogram (b) shows the distribution of total severity scores for each data-derived profile.

A) Symptomatic Sample (N = 17,504)

B) Low Severity (N = 3514)

C) Medium Severity (N = 7893)

D) High Severity (N = 6097)

Fig. 3. Symptom networks for the symptomatic sample (a), Low Severity profile (b), Medium Severity profile (c), and High Severity profile (d). Each symptom is represented by a node in the network, and the edge connecting nodes represents their correlation after controlling for all other nodes. Edge thickness corresponds to the weight of the partial correlation, and edge color represents the direction of the correlation (blue = positive, red = negative).

Fig. 4. Network comparisons between the symptomatic sample and data-derived profiles. The symptomatic sample's network ($n = 17,504$) served as the benchmark for comparisons with the Low Severity profile ($n = 3,514$), Medium Severity profile ($n = 7,893$), and High Severity profile ($n = 6,097$). Bootstrapped analyses (1,000 replications) were performed on each data-derived profile, and networks from each bootstrap replication were compared to the benchmark model on sensitivity, specificity, correlation, density, and proportion of negative edges. The boxplots represent the distribution of results from those comparisons.