Adult ADHD and Depression –

a Reliable Network Analysis

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Abstract

Objective. This work aims to enhance understanding of connections between ADHD and depression on a bridge symptom level to aid detection of undiagnosed ADHD in patients seeking therapeutic help because of onset depression.

Method. SCL-90R data of a transdiagnostic sample of adult psychotherapy patients in outpatient CBT (n = 1772) were analyzed by employing the network model approach with the advancement of psychopathological noise reduction. Bridge expected influence was measured to identify bridge symptoms that link depression and ADHD symptom clusters.

Results. Three bridge symptoms were found. “Feeling blocked in getting things done” was the strongest, followed by “feeling low in energy or slowed down” and “trouble concentrating”.

Conclusion. ADHD diagnostics should be considered for patients presenting with depression and suffering from one or more bridge symptoms. Additionally, exhaustion is a potential marker for ADHD in depressed patients.

Adult ADHD and Depression – a Reliable Network Analysis

Attention-deficit/hyperactivity disorder (ADHD) is a severe, common, and heterogeneous neurodevelopmental disorder. It is highly heritable and typically begins in childhood (Biederman et al., 1992; Biederman et al., 1990; Faraone, 2000; Faraone & Doyle, 2001; Frodl & Skokauskas, 2012; Mahone & Denckla, 2017; Tripp & Wickens, 2009). ADHD symptomatology is tied to executive functioning deficits (Silverstein et al., 2020) that entail, e.g., poor working memory (Thorell et al., 2019) and impaired self-regulation skills (Christiansen et al., 2019). Correspondingly, inattention, impulsivity, and hyperactivity are core symptoms (American Psychiatric Association, 2013). Douglas (1972) observed hyperactivity getting less relevant when patients grew older. Even though Lis et al. (2010) observed an increased motor activity in adult patients, Weyandt et al. (2003) showed that subjective feelings of internal restlessness sometimes fully replace hyperactivity symptoms.

While it is established that the disorder persists into adulthood (Arnold, 1972; Douglas, 1972), the extent to which it does is still controversial (Barkley et al., 2002). The reported persistence rates vary from 15 to 86.5 % in longitudinal studies (Barkley et al., 2002; Biederman et al., 2010; Cheung et al., 2015; Faraone et al., 2006; Hechtman et al., 2016; Van Lieshout et al., 2016), depending on the definition of remission (Biederman, 2004; Faraone et al., 2006) and methodology (Barkley et al., 2002). Correspondingly, the prevalence rate in adults has a broad range of 2.9 to 16.4 %, with screening studies identifying approximately 4 % of the adult population affected in the U.S. (Faraone & Biederman, 2005; Kessler, 2006; Kessler et al., 2006; Kessler et al., 2005). According to the current World Health Organization World Mental Health Surveys in which the Composite International Diagnostic Interview (CIDI) was administered to 26744 respondents worldwide, the prevalence of adult

ADHD is 2.8 % overall with a 57 % persistence rate in childhood-affected patients (Fayyad et al., 2017).

Adult ADHD causes severe impairments in various areas of patients' lives. It not only has a negative impact on educational and work performance (Hechtman et al., 2016; Shifrin et al., 2010) but correlates with interpersonal problems (Harpin et al., 2016; Sodano et al., 2021), low self-esteem (Cook et al., 2014; Harpin et al., 2016), higher risk for accidents (Aduen et al., 2015) and Covid-19 (Merzon et al., 2020), lower socioeconomic status (Pelham et al., 2020), as well as an reduced quality of life (Quintero et al., 2019; Thorell et al., 2019).

The risk for comorbid disorders increases with the number of ADHD symptoms (Vogel et al., 2018). Recent studies indicate that up to 87 % of ADHD patients develop one or more other psychiatric disorders throughout their lives (Sobanski et al., 2007; Torgersen et al., 2006). Affective disorders involving depressed mood are among the most common comorbidities with cumulative rates of up to 55 % (Biederman et al., 2008; Sobanski et al., 2007; Torgersen et al., 2006). According to Fayyad et al. (2017), ADHD precedes mood disorders in 85.6 % of the cases. This rate aligns with findings that support that ADHD and subthreshold ADHD increase the risk for depression (Biederman et al., 2008; Chronis-Tuscano et al., 2010; Monuteaux et al., 2007; Roy et al., 2014), dysthymia, and suicide (Chronis-Tuscano et al., 2010). Conversely, ADHD-specific treatment increases resilience for depression (Oddo et al., 2018).

Some symptoms are linked to depression and ADHD likewise. Concentration difficulty and restlessness are criteria of both diagnostic categories (American Psychiatric Association, 2013). Accordingly, Lundervold et al. (2016) found that cognitive function limitations and restlessness are two of the main co-occurring ADHD symptoms in depressed adolescents. These findings align with Lundervold et al.'s (2013) analysis of data from the Mood and Feelings Questionnaire, filled in by 9702 adolescents, which indicated that

restlessness and concentration difficulties are independent of, as well as part of depression symptomatology. Furthermore, emotional dysregulation is a symptom of depression (Beauregard et al., 2006) and ADHD (Corbisiero et al., 2012; Retz et al., 2012). In a longitudinal study from Seymour et al. (2012), emotional regulation was identified as a potential mechanism linking the two disorders in children.

Lundervold et al. (2016) argue that, if reported by depressed patients, concentration difficulties and restlessness may disguise a broader spectrum of problems from the inattention and hyperactivity/impulsivity domains of ADHD. This problem aligns with the findings from a survey among clinicians conducted by Schneider et al. (2019), in which overlapping symptoms were identified as a major challenge in diagnosing ADHD. Accordingly, it is no surprise that ADHD is frequently overlooked (Fayyad et al., 2017). Studies showed that 5.4 % to 22 % of outpatients with major depression (Alpert et al., 1996; Pehlivanidis et al., 2014; Rao et al., 2011) and 42 % diagnosed with recurrent brief depression (Hesslinger et al., 2003) met the criteria for ADHD diagnosis. As Barkley and Brown (2008) point out, the estimation of how many patients with a major depressive disorder also have undiagnosed ADHD varies widely, depending on methodology and sampling.

By diagnosing the neurological disorder more regularly, the life of many formerly overlooked patients can dramatically change for the better. Fleischman and Miller (2013, p. 47) state: “Once diagnosed with ADHD, these adults were able to construct a more coherent view of their life and of their difficulties, move beyond guilt, and understand that they could overcome their challenges.”. With a corresponding diagnosis, symptoms can be improved by providing adequate psychopharmaceutical (Elliott et al., 2020) and psychotherapeutic treatment (Knouse et al., 2017). Therefore, the primary goal of this work is to enhance understanding of the connections between ADHD and depression to improve the detection of ADHD in patients seeking help for depression.

Network modeling approach of psychopathology. Preszler et al. (2020) and Preszler and Burns (2019) demonstrate that the *network analysis framework* can generate additional insights into comorbidity within ADHD research compared to the standard *latent variable model*. Therefore, the network analysis framework was used for the work presented here. This approach is still a relatively novel method in psychopathological research, so it is briefly explained and contrasted to the classical theoretical framework in the following.

Within the latent variable approach, as Cramer et al. (2010) elaborate, symptoms are seen as markers of an underlying mental disease, much like in physical illnesses. Since the latent disease factor is treated as cause of covariance in symptoms, the symptoms are theorized to be conditionally independent, both within and between disorders. Cramer et al. (2010, p. 138) state that in this framework, comorbidity is "conceptualized as a (bi)directional relationship between two latent variables (i.e., disorders) that underlie a set of symptoms.". Most researchers in the field of psychopathology and ADHD base their work on the *latent variable model*. In this line of research, Roy et al. (2017) found that cognitive functioning is impaired stronger in adolescent ADHD patients with comorbid depression than without, and Bron et al. (2016) have shown that ADHD patients are prone to sleep disturbances beyond depression and anxiety.

In contrast, the network modeling approach of psychopathology explains the covariance of symptoms by defining the relationship of psychological symptoms and disorders in a mereological way. Guloksuz et al. (2017, p. 1) summarize the central axiom of network analysis as follows: "Mental disorders […] emerge from a dynamic interplay between symptoms, and therefore, signs and symptoms are not mere reflections of a discrete entity but a causal particle – a building brick – of the extended network of symptoms.". For instance, a patient that will be diagnosed with depression may first experience a stressful event that causes a depressed mood, which results in insomnia, which in turn leads to fatigue,

causing difficulties with concentration, which leads to a depressed mood because of induced feelings of failure and worthlessness. This harmful and self-reinforcing network of symptoms is understood as not caused by, but itself constituting a depression. A psychological disorder is therefore hypothesized to be a causal system consisting of interdependent symptoms. This system is represented by a statistical network comprised of *nodes* referring to symptoms and *edges* referring to their association with each other, with *weighted edges* representing proportional associations such as partial correlations. It is possible to identify which symptoms are closely related within this framework by looking for corresponding clusters. (Borsboom & Cramer, 2013; Cramer et al., 2010; Epskamp, 2017; Fried et al., 2016; Jones et al., 2019; McNally, 2016; Nuijten et al., 2016)

When it comes to comorbidity, Cramer et al. (2010, p. 138) argue that the network approach presents a "radically different" conceptualization because it "nullifies the need to invoke latent variables." The authors describe symptoms no longer serving as mere measurements of an underlying disorder, which is bi-directionally connected with a comorbid condition, which itself can only be measured by its predefined symptoms. By changing the methodological focus to direct connections between symptoms of comorbid disorders, former methodological problems disappear. For instance, the problematic assumption of all symptoms being equally influential can be dropped, as it arose from unweighted summation of symptom scores as standard procedure for measuring disorders. (Cramer et al., 2010)

One network analysis method to explore direct connections between symptoms of comorbid disorders is identifying the most influential nodes between disorder-specific node clusters within a network. These so-called *bridge symptoms* can foster knowledge about possible links of both disorders. Cramer et al. (2010) define bridge symptoms as either overlapping symptoms of disorders or symptoms of one disorder that increase the risk of containing the other disorder. Jones et al. (2019) point out that considering bridge symptoms

is highly relevant when clinical research aims to find the most suitable symptoms to be the target of deactivation in therapy. By employing a simulation of the contagion of a mental disorder, Robinaugh et al. (2016) demonstrated how eliminating bridge symptoms was especially successful in preventing the development of comorbidity.

Identifying bridge symptoms is the primary statistical goal of this work for the following two reasons. Firstly, finding bridge symptoms would aid detection of unrecognized ADHD in depressed patients, as they may serve as a marker for clinicians to investigate the possibility of comorbid ADHD. Secondly, enhanced understanding of the links between both disorders may aid in preventing the development of depression as a secondary disorder as it would enable clinicians to target bridge symptoms for deactivation.

Hypothesis. No work published to date investigates the relationship between adult ADHD and depression in a network analysis framework. Few studies have employed network analysis to deepen understanding of ADHD in children (cf. Preszler et al., 2020; Silk et al., 2019) or utilize network analysis to explore ADHD under the aspect of its comorbidity with other disorders than depression (cf. Goh et al., 2020; Preszler & Burns, 2019). Therefore, the network analysis presented here was conducted exploratively, and no a priori hypothesis was specified. However, as inattention and restlessness are overlapping criteria according to DSM-5 (American Psychiatric Association, 2013), and both turned out to be overlapping according to Lundervold et al. (2016), they are expected to be identified as bridge symptoms.

Method

Sample. Analyses are based on a sample of 1772 adults who received cognitive behavioral therapy (CBT) treatment at the Therapy and Counselling Center at the University of Göttingen, Germany, from 2007 to 2017. The center offers outpatient psychotherapeutic individual and group treatments for adults, children, and adolescents. The presented sample consists of adults who gave their informed consent to their data being used anonymously for

research purposes. The sample has a mean age of 38.20 (*SD* = 13.05), and 58.75 % of patients were female.

The sample is transdiagnostic. No inclusion or exclusion criteria based on diagnosis were defined. In network analysis, restricting the sample to patients with relevant diagnoses may result in a sampling bias. Patients who do not suffer from a symptom classified as core symptom in the chosen classification system (e.g., ICD-10 or DSM-5) would be excluded a priori, even if they suffered from all other symptoms. As a consequence, core symptoms may turn out more influential than they are. If, however, the classification system is accurate, there should be no difference between the network of a sample restricted to relevant diagnoses and a non-restricted sample. To test this, two subsamples were derived based on disorders. (Guloksuz et al., 2017)

The subsamples were based on the diagnostic categories of depressive disorders (F.32 – F.34; n = 877) and ADHD (F.90; n = 5) from the International Classification for Diseases (ICD-10; World Health Organization, 1992; WHO), as assigned by a therapist, as well as on a possible diagnosis of ADHD according to a specific pattern of answers on the ADHS-Selbstbeurteilungsskala (self-assessment scale; ADHS-SB; Rösler et al., 2004).

The ADHS-SB, a German questionnaire for self-assessment of ADHD symptoms, was filled in by 739 patients that received treatment from 2011 to 2016. Data of patients who filled in at least 75% of the questionnaire were included (n = 712). To be categorized as probably having ADHD, all diagnostic criteria according to DSM-5 had to be present according to the ADHS-SB except for the criterion of symptoms being better explained by another disorder (American Psychological Association, 2013). This item is not part of the ADHS-SB because it is a self-assessment, and patients do not have the expertise to make a respective judgment. In total, 207 patients have been classified as possibly having ADHD, which amounts to 13.73 % of patients overall and 29.07 % of patients to whom the self-

assessment was administered. While these numbers may appear high, the criterion was even stricter than the recommended ADHS-SB criterion of a cut-off at 18 points, at which it has a sensitivity of 65 % with a specificity of 92 % (Rösler et al., 2004). If this cut-off had served as the criterion, 41 % of patients would have been categorized as probably having ADHD.

The first subsample includes all patients with an ICD-10 diagnosis of ADHD (F.90; n = 5), every patient identified as probably having ADHD according to their ADHS-SB answer pattern (n = 207), as well as patients who were assigned the diagnosis of a disorder involving depressed mood according to ICD-10 categories F.32 to F.34 (n = 877) (WHO, 1992). This subsample is called the DA subset and includes 946 patients, some of which fulfill several of the criteria mentioned above.

The second subsample contains patients who fulfill neither of the criteria above, including patients who did not fill out the ADHS-SB questionnaire and those who did not qualify by assigned diagnosis (NoDA subset; n = 561).

Measures. Data from the German Symptom Checklist 90 Revised (Franke, 2002; SCL-90R) were analyzed. The questionnaire provides information about the amount of distress either of 90 psychopathological and physiological symptoms, like “Headache” or “Feeling blue”, cause to a patient. It includes a relevant set of depression and ADHD symptoms. Answers refer to the distress-related question “In the previous week, how much were you bothered by”, which is rated on a 5-point scale from “Not at all” (0) to “Extremely” (4). In this paper, items are described with the letter I for “item” followed by its assigned number, which also is the position within the SCL-90R (e.g., item number one is called "I1”).

The standard procedure would have been to exclusively collect data about symptoms of ADHD and depression via specific questionnaires and disregard the symptoms of every other disorder (cf. Heeren et al., 2018). With the current data, the equivalent would have been to only include items relevant for ADHD and depression while excluding all other items a

priori. However, this method is problematic because various interconnected symptoms outside considered disorders may produce considerable noise. For example, in this analysis, a highly relevant amount of noise could be caused by general anxiety disorder (GAD) symptoms that are significantly connected to depression symptoms (cf. Beard et al., 2016). When calculating partial correlations between ADHD and depression symptoms, excluding GAD symptoms may lead to overestimating or falsely detecting edges. Figure 1 shows how the connection between two nodes can be falsely positive because a GAD symptom fully mediates it or a GAD symptom equally causes both nodes. Therefore, all items of the SCL-90R were considered in the network model estimation to improve the specificity of the procedure.

For further analyses, SCL-90R items that correspond with symptoms of the two disorders of interest were picked. Depression symptoms were chosen to align to DSM-5 diagnostic criteria (American Psychiatric Association, 2013). Symptoms relevant to ADHD correspond to the items Eich et al. (2012) identified for their rating scale of adult ADHD based on the SCL-90R. The authors selected items in coherence with the German short form of the Wender-Utah Rating Scale (WURS-k), a retrospective assessment of childhood ADHD in adulthood (Retz-Junginger et al., 2002), and on clinical experience. Table 1 lists all items selected as relevant for depression. Table 2 gives an overview of relevant items for ADHD.

Jones et al. (2019) developed *bridge centrality metrics* to identify bridge symptoms. They have an overall sensitivity of 92.7 % and a specificity of 84.9 %. Of all metrics, *bridge expected influence* is the most robust. It measures the sum of all weighted edges that connect a specific node of a predefined cluster with all nodes of another predefined cluster. Therefore, it represents the sum connectivity of a symptom from a specific disorder with all symptoms of one or more other disorders. The higher the metric, the more likely the symptom is overlapping and the more influential it may be on nodes of the other disorders.

Data analytic plan

Pre-existing SCL-90R data were analyzed in a novel and exploratory manner. Statistical analyses were conducted using *R* (Version 4.0.3, R Core Team, 2020). The complete reproducible code is available in the supplementary materials.

Predictive mean matching (Van Buuren, 2018) allowed the imputation of missing data points based on the SCL-90R subscales. The function *aregImpute* of the *Hmisc* package (Harrell, 2020) was used accordingly. The *Wilcoxon rank sum test* (Bauer, 1972) was executed via the *wilcox.test* function (*stats* package; R Core Team, 2020) to ensure datasets did not differ before and after imputation. The *describe* function of the *psych* package (Revelle, 2020) returned descriptive statistics of the imputed dataset.

The paper by Epskamp et al. (2017a) served as a guideline for setting up the network models. It is recommended to investigate it for information about the underlying statistical method. Two types of network analyses were performed and compared. The first type was based on the maximum number of available SCL-90R symptoms and therefore corresponded to the advanced method developed in this work. According to the standard approach described by Epskamp et al. (2017a), the second type only considered symptoms of ADHD and depression. Partial correlations were defined as edge weights for all networks, which means that node associations were undirected, and the influence of all other nodes was controlled for. Partial correlation networks are also called *Gaussian graphical models* (GGM; Epskamp, 2017). All network model estimations were executed with the *estimateNetwork* function from the *bootnet* package (Epskamp et al., 2017a).

For the calculation of edges, the graphical *Least Absolute Shrinkage and Selection Operator* (LASSO; Friedman et al., 2008) was used within the *estimateNetwork* function. When executing this function, the *bootnet* package (Epskamp et al., 2017a) utilizes the *glasso* package (Friedman et al., 2014) as well as the *qgraph* (Epskamp et al., 2012) package, which

provides the *Extended Bayesian Information Criteria* for model selection (EBIC, Foygel & Drton, 2010). The LASSO was developed by Tibshirani (1996) to deal with the problem of relatively small psychological datasets being insufficient for calculating the many parameters a network has (e.g., for 50 nodes, there are 1275 parameters; Epskamp et al., 2017a). The operator employs bootstrapping to eliminate spurious associations attributed to influences of other nodes and to remove very small associations to avoid false positive edges, thus returning reasonably sparse networks (Epskamp, 2017). The *graphical LASSO*, or glasso, is an established algorithm for estimating the LASSO regularization (Epskamp et al., 2017a). With EBIC, model selection is automized. The hyperparameter γ (gamma), which is manually defined, controls the degree to which simpler models will be preferred. It ranges between 0 and 0.5 and was set to 0.5, according to the recommendation by Foygel and Drton (2010). With a high gamma like this, more sparse networks were preferred. The *EBICglasso* function, which is also an attribute of the *estimate network* function, is a combination of EBIC and glasso. It allows the calculation of sparse GGMs out of covariance or correlation matrices, where partial correlation coefficients may directly be used as edge weights (Epskamp, 2017).

For the ordinal data originating from the SCL-90R, Spearman's and Polychoric partial correlations would have been possible. As the Polychoric method is based on a complex estimation and prone to instability and error, Epskamp et al.'s (2017a) recommendation was followed in choosing the Spearman method. This decision was confirmed by personal correspondence with the author.

However, even the more stable Spearman's correlations cause difficulties under certain circumstances. One common problem is non-positive definite correlation matrices, which can cause GGMs to become unstable or impossible to calculate. As Lorenzo-Seva and Ferrando (2021) describe, a correlation matrix is positive definite when it has no negative eigenvalues. Eigenvalues of an inter-item correlation matrix reflect the amounts of variance

explained by the principal components of the items (Hoff, 2018). To test the required partial correlation matrix for negative eigenvalues, the function *eigen* of the *eigenmodel* package (Hoff, 2019) was applied to the correlation matrix. This kind of matrix can be calculated with the *cor* function (*base* package; R Core Team, 2020) for Spearman correlations or *cor_auto* function from the *graph* package (Epskamp et al., 2012), which utilizes the *lavaan* package (Rosseel, 2012) for other correlations. Lorenzo-Seva and Ferrando (2021) discuss several conditions that produce negative eigenvalues. The first one concerns items all participants answer alike, which leads to a skewed distribution. Redundant items with high correlation and items correlating close to one are described as troublesome, too. As non-positive correlation matrices occurred, strategic troubleshooting was applied by deleting error-causing items. This procedure enabled the continuation of analyses with the highest possible amount of information.

As described earlier, the first type of network analysis used all available symptoms as nodes to account for the highest possible amount of noise. In this manner, a network was estimated for the transdiagnostic sample, the DA subset, and the NoDA subset. To ensure sufficient stability of these large networks, *case-drop bootstrapping* was employed with the *bootnet* function of the *bootnet* package (Epskamp et al., 2017a). In this way, the maximum proportion of cases that can be dropped while retaining a correlation of .7 in at least 75% of the samples is calculated. According to Epskamp et al. (2017a), the resulting *correlation stability coefficient* (CS-Coefficient) should be at least above .25, preferably above .5. After stability was ensured, the networks were compared concerning structure, overall connectedness of nodes (*global strength*), and edge weight differences with the *NCT* function of the *NetworkComparisonTest* package (Van Borkulo, 2017) with Bonferroni correction for multiple testing.

The next step of the procedure provided a matrix that enabled a focus on the relevant items in the graphical display of the model, as well as in bridge metric estimation, without restricting the data considered in edge weight estimation beforehand. First, regular edge weight matrices were extracted from networks using the *getWmat* function of the *qgraph* package (Epskamp et al., 2012). Secondly, novel edge weights matrices were derived from the first ones by generating subsets containing rows and columns corresponding to all predefined ADHD and depression items. The *subset* function (*base* package; R Core Team, 2020) and the regular syntax of R were used to do so.

The novel edge weight matrices served as input for the *qgraph* function (*qgraph* package; Epskamp et al., 2012), which was used for network visualization. *Multidimensional scaling of networks* (MDS) was employed to generate a layout that delivers information upon sight. As Jones et al. (2018a, p. 3) explain, "MDS is particularly useful for understanding networks because the distances between plotted nodes are interpretable as Euclidean distances. That is, highly related nodes will appear close together, whereas weakly related ones will appear far apart.". The required *smacof* package (Leeuw & Mair, 2008) needs dissimilarities as input, so the partial correlation matrix was converted into a dissimilarity matrix with the packages function *sim2diss.* Further, using this package's *mds* and *head* functions, the required informational input for scaling was derived. Different scaling options can be compared with the normalized stress value (Mair et al., 2016), *stress-1*. The lower the *stress-1* value, as shown by the *plot* function, the more accurately the specific MDS layout represents the data (Jones et al., 2018a). The best fit MDS layout is used in the *qgraph* layout specification.

An additional GGM, in which only nodes relevant for ADHD and depression were considered, was estimated from the transdiagnostic sample to compare the advanced method to the standard procedure (cf. Epskamp et al., 2017a). This network and the advanced version

were visually compared using the *averageLayout* function (*qgraph* package, Epskamp et al., 2012). The function creates a *layout* attribute for *qgraph* to align the node positions of both networks. Statistical comparison with the *NCT* function (*NetworkComparisonTest* package; Van Borkulo, 2017) would require both networks to be created by the *estimateNetwork* function (*bootnet* package; Epskamp et al., 2017a) with the same number of nodes, which was not the case here.

To find out which symptoms connect ADHD and depression, bridge centrality measures were computed. To obtain bridge centrality metrics, the *bridge* function of the *networktools* package (Jones et al., 2020) was used. As required, communities representing the two relevant disorders were defined. Nodes were grouped according to the categorization of items as ADHD or depression symptoms. Restlessness and concentration difficulties, which are overlapping criteria according to DSM-5 (American Psychological Association, 2013), are core symptoms in ADHD while not being central for depression. For this reason, they were defined as belonging to the ADHD community. *Bridge expected influence (1-step)* is focused upon as it is the most robust bridge metric, according to Jones et al. (2019). The authors define it as the sum of the value (positive or negative) of all weighted edges between node A and nodes in a designated community of which A is not part. As 2-step bridge expected influence is not relevant here, bridge expected influence (1-step) is further referred to as bridge expected influence (*eI1*).

Bootstrapping was employed via *bootnet* and *corStability* functions of the *bootnet* package (Epskamp et al., 2017a) to ensure the stability of bridge centrality metrics. This function estimates the average correlation of the bridge centrality metrics with the corresponding values of drawn subsamples with a varied amount of dropped cases. By this, the method delivers information about the general stability of a particular bridge metric. Furthermore, the function enables generating a graph showing confidence intervals for bridge

metrics of all nodes. The broader these intervals are, the less accurate the estimated value is. This method was introduced and explained by Jones (2019) on his blog. The procedure used here, which involved using the *estimateNetwork* (*bootnet* package; Epskamp et al., 2017a) output and categorizing all irrelevant nodes to an "other" category, was determined based on personal correspondence with Dr. Jones.

Results

Data of patients who answered less than 75 % of the SCL-90R were dropped (n = 265). Of the remaining patients (n = 1507), 658 missing responses were imputed. There was no significant difference between the datasets before and after imputation ($w = 3942$, $p = .76$). All analyses were executed with imputed data. Descriptive statistics for relevant items are shown in Table 3. As expected from psychometric data measured with a Likert scale, the distribution of some items was skewed.

The attempt to calculate a GGM for all 90 items with Spearman's correlations for the transdiagnostic sample returned the error of the correlation matrix not being positive definite. Analysis of the correlation matrix revealed negative eigenvalues, which caused the error (Lorenzo-Seva & Ferrando, 2021). Troubleshooting was performed accordingly. Nearly all participants answered I16 ("Hearing voices that other people do not hear") negatively, which resulted in a trimmed mean of zero with low variance and nearly no distribution (Table 3). Hence, I16 was excluded from further analysis. In total, six item pairs correlated very highly ($\rho > .99$), and four pairs correlated highly ($\rho \geq .75$) while containing items with redundant content. One item of each pair was removed from the analysis, and the corresponding items were renamed as shown in Table 4. 79 items remained as nodes in all networks.

Casedrop-bootstrapping showed high network stability with a CS-Coefficient of .75 for the transdiagnostic sample. The network of the NoDA subset had good stability (CS-Coefficient = .59). With a CS-Coefficient of .44, the network based on depressed patients

and/or patients with ADHD fulfilled the requirement of being over .25 and was considered moderately stable (Epskamp et al., 2017a).

There was no difference in structure (p = 1) or global strength (p = .83) for the networks derived from all patients and the subgroup of patients with depression and/or ADHD. There was no significant difference between edge weights (*ew*) except between the edges connecting I9 (“Trouble remembering things“) and I17 (“Trembling”; p = .04). Furthermore, the network of patients without ADHD and Depression diagnosis did not differ in global strength (p = .52) or structure (p = .99) from the network including all patients. These networks also did not differ significantly in edges (p = 1). The networks of the DA subset and the NoDA subset did not differ significantly in global strength (p = .26) and structure (p = .26). Despite the conservative Bonferroni approach, eight edge weights differed between networks, but none of them connected symptoms between or within relevant disorder clusters.

According to its lowest *stress-1* value, the ordinal MDS layout was used for all network graphs. The GGM for relevant symptoms of the transdiagnostic sample created with the advanced procedure is depicted in Figure 2. Figure 3 enables visual comparison of the standard procedure network with the advanced one for the transdiagnostic sample. In both, ADHD symptoms are displayed as orange and depression symptoms as blue, overlapping symptoms I55 (“Trouble concentrating”) and I78 (“Feeling so restless you couldn’t sit still”) as yellow nodes. Green edges show positive correlations, and red edges would have shown negative ones. Thickness and saturation of edges increase with correlation level. In every network graph, ADHD and depression symptoms clustered together, with I55 and I78 located between clusters. However, the assigned ADHD symptom I28 (“Feeling blocked in getting things done”) was part of the depression cluster in every comorbidity network. The clustering of the network model estimated with the standard procedure was similar to the

methodologically advanced networks, but the standard network showed more and thicker edges.

For relevant items and the transdiagnostic sample, all bridge metric values are listed in Table 5. Furthermore, *z*-values of all bridge metrics are displayed in Figure 4. Both shall enable future comparison with studies that may focus on different bridge metrics than bridge expected influence. Classifying an overlapping node as depression instead of ADHD symptom did not change the ranking of symptoms in bridge expected influence. I28 and I14 ("Feeling low in energy or slowed down") stood out as only nodes approximately 2 or more standard deviations above the mean (Figure 4). Fittingly, I28 had the highest bridge expected influence in the transdiagnostic sample, followed by I14, then I55 (Table 5). As shown in Figure 5, the rank wase similar for the DA but different for the NoDA subset. The first two items ranked the same for people without either diagnosis, but I9 ("Trouble remembering things") was the third strongest.

Bridge expected influence refers to the sum of all partial correlations connecting a node with all nodes of the opposing disorder. Therefore, it was determined that an *eI1* above .1 equates a small, above .3 a moderate, and above .5 a strong correlation with the opposing disorder. Within the sample of patients with or with a likely diagnosis of ADHD and/or depression, node I28 correlated strongly, while I14 and I55 correlated moderately with nodes of the opposite disorder. All other nodes had a small or no corresponding correlation in this subgroup. Table 6 provides corresponding *eI1* values for every sample within the advanced procedure.

Figure 6 shows the bridge expected influence for the network calculated according to the standard procedure compared to the advanced procedure based on the transdiagnostic population. Even though there were differences in the metric values, the rank of the first two nodes was similar. I28 (eI1 = .85) was the most influential bridge symptom in the standard

procedure, followed by I14 (eI1 = .61), then I46 (“Difficulty making decisions”; *eI1* = .39). The standard procedure resulted in higher bridge metric values for several nodes that scored low according to the advanced method.

For the transdiagnostic sample, Figure 7 depicts the results of bootstrapping, which show that the average correlation of the bridge expected influence with the corresponding values of a drawn sub-sample stayed nearly one, even with 70% dropped cases. The metric had high accuracy, as the line was close to linear. Confidence intervals of bridge expected influence values were moderate to narrow, as displayed in Figure 8. Both were also true for the DA subset.

Discussion

This work aimed to identify bridge symptoms of ADHD and depression in adults. These symptoms are the most influential nodes between disorder clusters within a psychopathological network (Cramer et al., 2010; Jones et al., 2019; Robinaugh et al., 2016). Since bridge symptoms can be a marker to investigate the possibility of a specific comorbid disorder, their identification serves the primary goal of this work, which is to propagate the detection of undiagnosed ADHD in patients presenting with depression. Furthermore, the identification of bridge symptoms may aid in the prevention of depression as a secondary disorder as they can be targeted in ADHD therapy (Jones et al., 2019; Robinaugh et al., 2016). Additionally, an advanced method was developed to reduce noise and enhance the reliability of results within the applied network analysis framework.

Bridge expected influence was calculated to identify bridge symptoms of ADHD and depression. There is no established rule about how high a bridge metric needs to be for a node to qualify as a bridge symptom. In this work, all symptoms partially correlating at least moderately with the symptoms of the other disorder are classified as bridge symptoms. When this rule was applied, the nodes “feeling blocked in getting things done”, “feeling low in

energy or slowed down" and "trouble concentrating" qualified as bridge symptoms in the subsample which includes patients with or with probable ADHD and/or depression. As this work aimed to enhance ADHD diagnosis of affected patients presenting with depression, this sample is considered most important for the classification of bridge symptoms. Fittingly, the three symptoms also scored highest in bridge expected influence for the transdiagnostic sample. For both samples, "Feeling blocked in getting things done" was the strongest, followed by "feeling low in energy or slowed down", then "trouble concentrating". The first two symptoms also scored highest in the subset of patients without either diagnosis and in the standard method network of the transdiagnostic sample. Accordingly, "feeling blocked in getting things done", "feeling low in energy or slowed down", and "trouble concentrating" are defined as representing bridge symptoms of ADHD and depression.

Difficulties to concentrate being a bridge symptom aligns with the results of Lundervold et al. (2016), who found that cognitive function limitation was one of the main co-occurring ADHD symptoms in depressed adolescents. Furthermore, it aligns with the expectation evoked by the overlap of the corresponding DSM-5 criteria (American Psychological Association, 2013). Because of the same overlap, concentration difficulties are classified as a bridge symptom of depression and GAD in the network perspective paper by Borsboom and Cramer (2013). Additionally, disrupted ability to concentrate was found to be linking depression and obsessive-compulsive disorder (OCD) in a network analysis by Jones et al. (2018b). Difficulty concentrating seems to be a symptom that is strongly connected to all these disorders. It may be worthwhile to investigate if the symptom also qualifies as a bridge symptom of ADHD and GAD, as well as ADHD and OCD in future research.

The node representing restlessness scored low in bridge expected influence, even though the symptom is an overlapping DSM-5 criterion of ADHD and depression, as well (American Psychological Association, 2013). This result also contradicts the findings of

Lundervold et al. (2016), who identified restlessness as another main co-occurring ADHD symptom in depressed adolescents. Correspondingly, results only partly align with Lundervold et al. (2013), who found that concentration difficulties, tiredness, and restlessness are both independent of and part of depression. As bridge symptoms, the nodes of "trouble concentrating" and "feeling low in energy or slowed down" are connected to depression, which may account for dependence, but also to ADHD, which may account for independence from depression. However, this is not the case for the node "feeling so restless you couldn't sit still", which did not turn out to be among the bridge symptoms in this work. This result may be interpreted as evidence against the importance of the agitation symptom for depression.

Exhaustion may be an underlying link between the bridge symptoms. Feeling low in energy due to sleep deprivation is established as a disrupter of attention, resulting in difficulties to perform ongoing goal-directed behavior (Krause et al., 2017). Fittingly, ADHD and depression were identified as predictors for nurses' exhaustion in a study by Kim et al. (2019). Moreover, this aligns with Brattberg's (2006) findings of 56 % of patients on long-term sick leave due to burnout having undiagnosed ADHD or possible ADHD, compared to none of the patients without burnout. In this study, all patients except five are undiagnosed with ADHD as well. Still, it needs to be noted that while exhaustion probably is the main symptom of burnout, there still are no consensual diagnostic criteria, and the overlap between depression and burnout remains unclear (Bianchi et al., 2015).

An alternative explanation for the pattern may be that the identified bridge symptoms are strongly connected to DSM-5 criteria of both disorders. As already established, "trouble concentrating" serves as a diagnostic criterion for depression ("Diminished ability to think or concentrate"), while "Inattention" is a category for ADHD criteria in DSM-5. The criterion closely related to "feeling blocked in getting things done" is called "Often avoids, dislikes, or

is reluctant to tasks that require mental effort over a long period of time" for ADHD and "Markedly diminished interest […] in all, or almost all, activities most of the day, nearly every day" for depression. Fatigue, however, is solely a criterion of depression, called "Fatigue or loss of energy nearly every day", so the exhaustion hypothesis is maintained as a probable explanation. (American Psychological Association, 2013)

Past studies found evidence of emotional dysregulation, which presents itself as poor temper control, emotional over-reactivity, and affect lability, being linked to both: depression and ADHD (Beauregard et al., 2006; Corbisiero et al., 2012; Retz et al., 2012; Seymour et al., 2012). However, none of the matching symptoms analyzed in this study ("Trembling, impulsive temper outbursts", "Arguments, racing heart", "Crying easily", "Blank mind with rage impulses") qualified as bridge symptoms. Still, with a nearly moderate bridge expected influence, "Blank mind with rage impulses" was ranked fourth within the subgroup of patients struggling with or with probable ADHD and/or depression. This symptom ranking high and nearly qualifying as bridge symptom in the most important subgroup is the only further evidence for emotional dysregulation being present in both disorders here.

One argument in favor of network models is that current classification systems were incapable of capturing the complexity of psychopathology (cf. Boschloo et al., 2015; Guloksuz et al., 2017; Fried et al., 2016). This is opposed here, as there was no difference found in global strength and structure between networks of the transdiagnostic sample and diagnose specific subsamples. Additionally, network models showed a straightforward clustering of nearly all depression and ADHD symptoms according to DSM-5 disorder categories, regardless of method and sample. This finding supports the accuracy of the classification system, as does the result of "trouble concentrating" qualifying as bridge symptom, while also being an overlapping criterion according to DSM-5. However, restlessness did not qualify as bridge symptom despite the overlap of criteria in DSM-5,

which may support the argument against its accuracy. Additionally, the node of “feeling blocked in getting things done” clusters with depression symptoms, even though it is categorized as ADHD symptom. But, as explained before, this item may simply be more directly connected to the loss of interest in activities associated with depression than the corresponding ADHD symptom, which only refers to certain kinds of tasks. For this reason, the clustering with depression symptoms is comprehensible and not seen as evidence for questioning the DSM-5. (American Psychological Association, 2013)

Apart from reaching the substantial goals of this work, the methodological approach to network analysis in comorbidity research was improved. Following the standard procedure, previous studies measured symptoms with disorder-specific questionnaires. For example, Heeren et al. (2018) used the Beck Depression Inventory and Liebowitz Social Anxiety Scale to create a comorbidity network for depression and social anxiety. For this analysis, data from a questionnaire covering a broad range of diverse symptoms were used to account for other disorders' influence. The main achievement of this procedure was an improvement of specificity due to the successful elimination of false positive edges and shrinkage of edge weights which would have been overestimated otherwise. It must be noted that this improvement only holds if the sparsity assumption concerning the true network is correct. Following this assumption, there has already been a methodological effort in this area of research to enhance specificity (cf. Epskamp, 2017; Epskamp et al., 2017a). However, Epskamp et al. (2017b) point out how this increased the risk of overestimating the sparsity of a network. Their simulation showed that to correctly estimate a dense undirected network while using the LASSO parameter (which was designed as a regulation method to improve specificity), a sample size of more than 5000 participants is required. Nonetheless, when the true network was sparse, their simulation demonstrated how using the LASSO parameter aids in estimating the most accurate model. When calculating a sparse network with nine nodes,

all edges were correctly identified with 500 participants when the LASSO parameter was employed. In contrast, no regulation led to false positive edges, even with a large sample size of 5000. Therefore, the method introduced here may further decrease the sample size needed in relation to the number of nodes to estimate a sparse network accurately. Negative eigenvalues, which are the most common problem with the necessary employment of a high number of nodes within the established procedure, can be avoided by systematically eliminating troublesome items and joining strongly correlating ones. The advancement enabled a stable network by employing a broad clinical questionnaire with 79 nodes with a transdiagnostic sample of about 1500 participants.

A second achievement of the methodological advancement concerned bridge symptom identification. While Jones et al.'s (2019) simulation showed a low negative impact of noise on sensitivity and specificity of all bridge metrics, this analysis demonstrated quite notable differences between methods in bridge expected influence scores for several nodes. Ten symptoms in total qualified as bridge symptoms according to the standard procedure only. Additionally, the ranking was altered when irrelevant symptoms were not excluded a priori. The impact of noise was more severe than predicted by Jones et al. (2019).

Limitations

It remains unclear which theoretical framework works best for describing the relationship between depression and ADHD. An essential disease factor that would favor the factor analysis is not easy to pinpoint for depression. Borsboom and Cramer (2013) argue:

> […] although in the past decades much has been made of the suggestion that symptoms in psychopathology do have […] root causes (variously suggested to have a basis in repressed desires, learned helplessness, hormonal imbalances, neural abnormalities, or genetic defects), it has so far been impossible to identify these empirically. (p. 94)

Their argument is fit for depression. Nevertheless, it is well established that ADHD comes with, for example, typical neurological impairments that can be observed via brain imaging (cf. Cortese et al., 2012; Frodl & Skokauskas, 2012). For example, Tripp and Wickens (2009) point out that the brains of patients with ADHD differ in the dimensions of the frontal lobe, caudate nucleus, and cerebellar vermis. It is relatively safe to assume that at least some covariance of the core symptoms of ADHD arises from a joint latent variable that is a neurobiological deviation from the norm. Therefore, the network model seems to be a good theoretical fit for depression while not ideal for describing ADHD. The inclusion of biological root causes of ADHD as latent factors would have been necessary to estimate networks that model reality more accurately. Unfortunately, no such method is established so far, so that source of covariance is unaccounted for in this analysis. Likely, this is not the only one. While a broad range of psychopathological noise was controlled for, the SCL-90R may not cover all critical confounding symptoms. Furthermore, there was no control for factors outside of psychological symptoms, like important events, relationships, and physical illness, which may significantly impact mental health. Guloksuz et al. (2017) theorized about an approach that may solve this problem. They suggest a multi-layered model containing symptom networks at the outermost layer and neurological and genetic factors at the most central layers. A method that follows this approach would enable a more valid investigation of the relationship between neurological disorders like ADHD and mental disorders with no clear biological cause like depression.

Another limitation arises from the data being cross-sectional. One central assumption of psychopathological network theory is that symptoms of one disorder can cause an activation cascade in a nearby symptom cluster, leading to enough symptoms being present to constitute a comorbid disorder (cf. Jones et al. 2019; Robinaugh et al., 2016). Biological root causes being present in ADHD while not yet discovered in depression lead to the hypothesis

that depression can be ignited by bridge symptoms of other disorders, while ADHD cannot be caused in this way. This assumption is supported by Fayyad et al. (2017), who found that ADHD preceded mood disorders in more than 85 % of the cases. However, it is essential to note that this work is unsuitable for supporting or dismissing such a hypothesis. Undirected network models cannot deliver any information about causal coherences and sequences, which limits the depth of information supplied by this study (Borsboom & Cramer, 2013). Therefore, it remains unclear if the bridge symptoms of either of the two disorders are causally involved in the development of the other. Because of this, it is not secured that targeting the identified bridge symptoms would prevent the development of a depressive episode in adult ADHD patients, as the simulation of Robinaugh et al. (2016) suggests. Longitudinal studies and with it directed networks are required to test this assumption.

Two limitations arise from the use of the SCL-90R (Franke, 2002). Firstly, the questionnaire only refers to symptoms being present one week before the assessment. Both ADHD and depression symptoms must be present for a more extended period to justify a diagnosis. Secondly, a self-assessment may be problematic because patients tend to underestimate the severity and implications of their symptoms (Du Rietz et al., 2016; Manor et al., 2012). For this reason, multi-informant assessment is recommended concerning ADHD symptomatology in general (Nelson, 2013). It may be possible that symptoms correlate differently if based on the assessment of others, which may result in an alteration of network models depending on the informational source. While there have already been ADHD network studies involving external assessments with children (Goh et al., 2020; Silk et al., 2019), no such study has been published involving adults. Additionally, no network study to date compared networks based on external or internal assessment of ADHD. However, Preszler and Burns (2019) found differences in ADHD symptom networks, depending on the

external information source. Further research based on multi-informant assessment may be essential to secure the findings.

Conclusion

This study reached its goal of providing insights into the connection between ADHD and depression on a symptom level. Three bridge symptoms linking both disorders were identified: feeling blocked in getting things done, being low in energy or slowed down, and concentration impairment. It is recommended that clinicians consider ADHD whenever patients present with depression symptoms combined with one or more bridge symptoms. The hypothesis of exhaustion being a mediator between ADHD and depression, with the first causing the latter, is still to be confirmed by longitudinal studies. However, expanding diagnostic efforts for ADHD in patients presenting with depressive symptoms, especially when they are exhausted, is worthwhile at this point already. All bridge symptoms can be caused by fatigue (Krause et al., 2017), and the frequent co-occurrence of depression and ADHD is already secured (cf. Alpert et al., 1996; Biederman et al., 2008; Chronis-Tuscano et al., 2010; Hesslinger, 2003; Monuteaux et al., 2007; Pehlivanidis et al., 2014; Rao et al., 2011; Roy et al., 2014). Furthermore, it is established that ADHD is drastically underdiagnosed to date in general (Brattberg, 2006; Fayyad et al., 2017). Therefore, this work advocates for higher awareness of ADHD in the diagnostics of mental disorders.

Apart from the substantial findings summarized above, the aspired methodological improvement in psychopathological network analysis for sparse networks was successful. Psychopathological noise caused by symptoms not belonging to the investigated disorders was accounted for to a high degree. In this work, symptoms of irrelevant disorders were not excluded a priori, as would have been the established standard procedure, by solely using disorder-specific questionnaires. Instead, they were accounted for by using data from a broad clinical questionnaire. Reducing graphs to relevant nodes after estimation rendered the results

of the standard and improved method comparable. The advanced procedure generated more specific networks by eliminating false positives and reducing overestimated edge weights. The advancements also impacted bridge symptom identification by altering bridge metric values and the corresponding rank of nodes in bridge expected influence. Furthermore, the novel approach made modeling an extensive, stable network of 79 nodes with about 1500 participants possible. It is therefore recommended for future research in this area.

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Table 1

Selected SCL-90R items for ADHD aligned to corresponding DSM-5 criteria

SCL-90R items		DSM-5 criteria[a]
I2	“Nervousness or shakiness inside“	Hyperactivity ("adolescents and adults may be limited to feeling restlessness")
I9	“Trouble remembering things“	Inattention ("Is often forgetful in daily activities")
I11	“Feeling easily annoyed or irritated”	"There is clear evidence that the symptoms interfere with, or reduce the quality of, social […] functioning."
I24	“Temper outbursts that you could not control”	Impulsivity ("often interrupts or intrudes on others")
I28	“Feeling blocked in getting things done”	Inattention ("Often avoids, dislikes, or is reluctant to tasks that require mental effort over a long period of time")
I55	“Trouble concentrating“ [b]	Inattention (e.g., "Often has trouble holding attention on tasks")
I57	“Feeling tense or keyed up”	Hyperactivity ("adolescents and adults may be limited to feeling restlessness")
I74	“Getting into frequent arguments“	"There is clear evidence that the symptoms interfere with, or reduce the quality of, social […] functioning."
I78	“Feeling so restless you couldn’t sit still” [b]	Hyperactivity ("Often leaves seat when remaining seated is expected")

Note. Selection based on SCL-90R ADHD-Screening developed by Eich et al. (2012).
[a]American Psychiatric Association (2013). [b]Overlapping criterion.

Table 2

Selected SCL-90R items for depression aligned to corresponding DSM-5 criteria

SCL-90R items		DSM-5 criteria[a]
I5	“Loss of sexual interest or pleasure”	"Markedly diminished interest"
I14	“Feeling low in energy or slowed down”	"Fatigue or loss of energy nearly every day"
I15	“Thoughts of ending your life”	"recurrent suicidal ideation"
I19	“Poor appetite”	"decrease or increase in appetite”
I20	“Crying easily”	"appears tearful"
I26	“Blaming yourself for things”	"inappropriate guilt"
I30	“Feeling blue”	"Depressed indicated by subjective report"
I32	“Feeling no interest in things”	"Markedly diminished interest"
I41	“Feeling inferior to others”	"Feelings of worthlessness"
I44	“Trouble falling asleep”	“Insomnia or hypersomnia nearly every day”
I46	“Difficulty making decisions”	“indecisiveness”
I51	“Mind is going blank”	“Diminished ability to think or concentrate”
I55	“Trouble concentrating” [b]	“Diminished ability to think or concentrate”
I59	“Thoughts of death or dying”	“Recurrent thoughts of death”
I66	“Sleep that is restless or disturbed”	“Insomnia or hypersomnia nearly every day”
I78	“Feeling so restless you couldn’t sit still” [b]	“Psychomotor agitation”
I79	“Feelings of worthlessness”	“Feelings of worthlessness”
I89	“Feelings of guilt”	“Feelings of […] guilt”

Note. Selection based on DSM-5 Symptoms. [a]American Psychiatric Association (2013). [b]Overlapping criterion.

Table 3

Descriptive statistics for relevant SCL-90R items in transdiagnostic sample (n = 1507)

SCL-90R item	*M*	*M* (trimmed)	*SD*	*SE*	*Skew*	*Kurtosis*
I2	1.58	1.52	1.18	.03	.33	-.83
I5	1.32	1.15	1.36	.03	.68	- .81
I9	1.37	1.25	1.21	.03	.67	-.46
I14	1.67	1.61	1.23	.03	.27	-.93
I15	.44	.21	.89	.02	2.34	5.28
I19	.52	.31	.90	.02	1.96	3.54
I20	1.27	1.10	1.30	.03	.75	-.58
I26	1.47	1.35	1.27	.03	.51	-.79
I28	1.82	1.77	1.27	.03	.19	-1.01
I30	1.36	1.24	1.24	.03	.60	-.69
I32	1.16	1.01	1.24	.03	.80	-.48
I41	1.43	1.31	1.31	.03	.51	-.91
I44	1.43	1.29	1.39	.04	.58	-.97
I46	1.46	1.33	1.29	.03	.54	-.80
I51	.94	.75	1.16	.03	1.08	.16
I55	1.78	1.72	1.25	.03	.27	-.96
I57	1.65	1.59	1.21	.03	.29	-.86
I59	.92	.71	1.20	.03	1.19	.35
I66	1.66	1.58	1.37	.04	.36	-1.10
I74	.84	.64	1.08	.03	1.23	.69
I78	.88	.67	1.13	.03	1.24	.66
I79	1.35	1.19	1.35	.03	.67	-.80
I89	1.28	1.13	1.30	.03	.68	-.73

Note. Selection of items based on relevance for ADHD and depression (Table 1 and 2). Additionally, items that correlated highly with a relevant symptom (Table 4) are listed.

Table 4

Artificial creation of items out of highly correlating pairs to correct for negative eigenvalues

ρ	Included	Excluded	New Name
.99	Your mind going blank (I51)	Shouting or throwing things (I81)	I51 – 81 Blank mind with rage impulses
.99	Nervousness or shakiness inside (I2)	Feeling easily annoyed or irritated (I11)	I2 – 11 Feeling restless, nervous, irritable
.99	Trembling (I17)	Temper outbursts […] (I24)	I17 – 24 Trembling, temper outbursts
.99	Suddenly scared for no reason (I23)	[…] urges to beat, injure, or harm […] (I63)	I23 – 63 Startled easily, urge to hurt
.99	Feeling fearful (I33)	[…] urges to break or smash things (I67)	I33 – 67 Fearful, urge to destroy
.99	Getting into frequent arguments (I74)	Heart pounding or racing (I39)	I74 – 39 Arguments, racing heart
.85	Feeling […] talked about […] (I43)	Feeling uneasy […] talking about you (I61)	I43 – 61 Subject of conversation
.80	Feeling weak in parts of your body (I56)	Heavy feelings in your arms or legs (I58)	I56 – 58 Weak and heavy body parts
.75	Feeling lonely (I29)	Feeling lonely with people (I77)	I29 – 77 Loneliness
.75	Sleep that is restless or disturbed (I66)	Trouble falling asleep (I44)	I66 – 44 Insomnia

Note. Troubleshooting of negative eigenvalues (cf. Lorenzo-Seva & Ferrando, 2021).

Table 5

Bridge metrics for all relevant items for the transdiagnostic sample (n = 1507)

	eI1[a]	Bridge Betweenness[b]	Bridge Closeness[c]	Bridge Strength[d]
I28 – Feeling blocked in getting things done	.64	34	.07	.64
1I4 – Feeling low in energy or slowed down	.41	9	.07	.41
I55 – Trouble concentrating	.24	41	.04	.24
I46 – Difficulty making decisions	.22	3	.04	.22
I51 – 81 Blank mind with rage impulses	.20	1	.05	.20
I9 – Trouble remembering things	.18	0	.04	.18
I19 – Poor appetite	.13	26	.04	.13
I66 – 44 – Insomnia	.13	0	.03	.13
I78 – Feeling so restless you couldn't sit still	.13	6	.03	.13
I32 – Feeling no interest in things	.12	1	.04	.12
I17 – 24 – Impulsive	.10	32	.03	.10
I30 – Feeling blue	.10	4	.04	.10
I2 – 11 Feeling restless, nervous, irritable	.06	25	.03	.06
I26 – Blaming yourself for things	.06	7	.03	.06
I57 – Feeling tense or keyed up	.04	0	.03	.04
I20 – Crying easily	.02	0	.03	.02
I5 – Loss of sexual interest or pleasure	.01	0	.02	.01
I74 - 39 – Arguments, racing heart	.00	0	.02	.00
I79 – Feelings of worthlessness	.00	7	.03	.00
I15 – Thoughts of ending your life	.00	17	.03	.00
I59 – Thoughts of death or dying	.00	0	.02	.00
I89 – Feelings of guilt	.00	1	.03	.00
I41 – Feeling inferior to others	.00	0	.02	.00

Note. [a]Bridge expected influence (1– step) is defined as the sum of the value (+ or –) of all edges that exist between a node A, and all nodes that are in a designated community node A is not a part of. [b]Bridge Betweenness measures the cumulative number of times a node lies on the shortest path between nodes i and j, where nodes i and j come from different communities. [c]Bridge Closeness is defined as the inverse of the average length of the path from a node A to all nodes that are in a different designated community as node A. [d]Bridge Strength is defined as the sum of the absolute value of all edges that exist between a node A and all nodes that are in another designated community as node A. (Jones et al., 2019)

Table 6

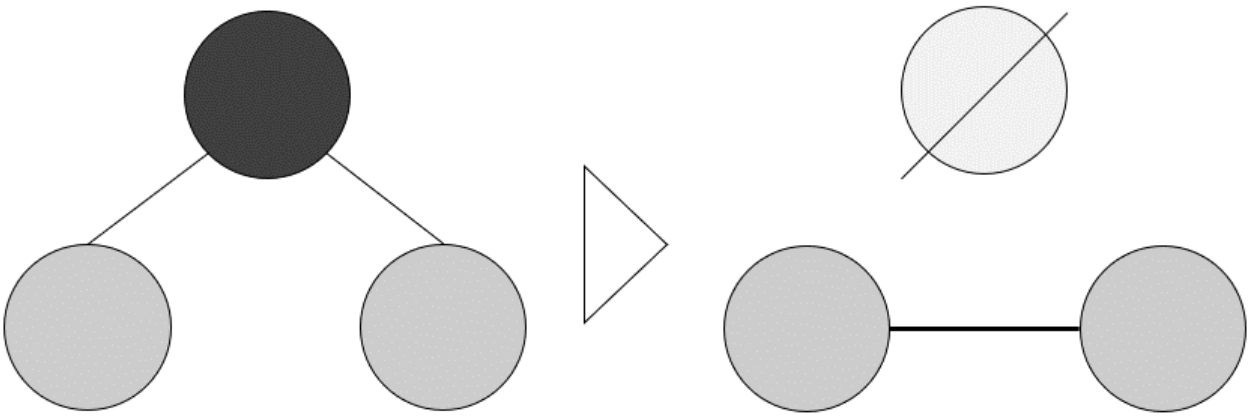
Bridge expected influence (1-step) for all samples according to advanced method

Community	Node	Transdiagnostic	DA	NoDA
2	I28 – Feeling blocked in getting things done	.64	.66	.55
1	I14 – Feeling low in energy or slowed down	.41	.39	.37
2	I55 – Trouble concentrating	.24	.34	.12
1	I46 – Difficulty making decisions	.22	.25	.17
1	I51 – 81 Blank mind with rage impulses	.20	.27	.06
2	I9 – Trouble remembering things	.18	.12	.23
1	I19 – Poor appetite	.13	.09	.18
1	I66 – 44 – Insomnia	.13	.12	.17
2	I78 – Feeling so restless you couldn't sit still	.13	.09	.20
1	I32 – Feeling no interest in things	.12	.11	.12
2	I17–24 – Impulsive	.10	.12	.10
1	I30 – Feeling blue	.10	.09	.11
2	I2 – 11 Feeling restless, nervous, irritable	.06	.10	.03
1	I26 – Blaming yourself for things	.06	.03	.10
2	I57 – Feeling tense or keyed up	.04	.03	.08
1	I20 – Crying easily	.02	.04	.02
1	I5 – Loss of sexual interest or pleasure	.01	.02	.01
2	I74 – 39 – Arguments, racing heart	.00	.00	.00
1	I79 – Feelings of worthlessness	.00	.01	.00
1	I15 – Thoughts of ending your life	.00	.02	.00
1	I59 – Thoughts of death or dying	.00	.00	.00
1	I41 – Feeling inferior to others	.00	.00	.01
1	I89 – Feelings of guilt	.00	.01	.00

Note. Values show bridge expected influence (1-step), which describes the sum of edge weights (partial correlations) of all edges that exist between the node and all nodes that are in a designated community node A is not a part of. Community 1 represents depression symptoms, community 2 represents ADHD symptoms.

Figure 1

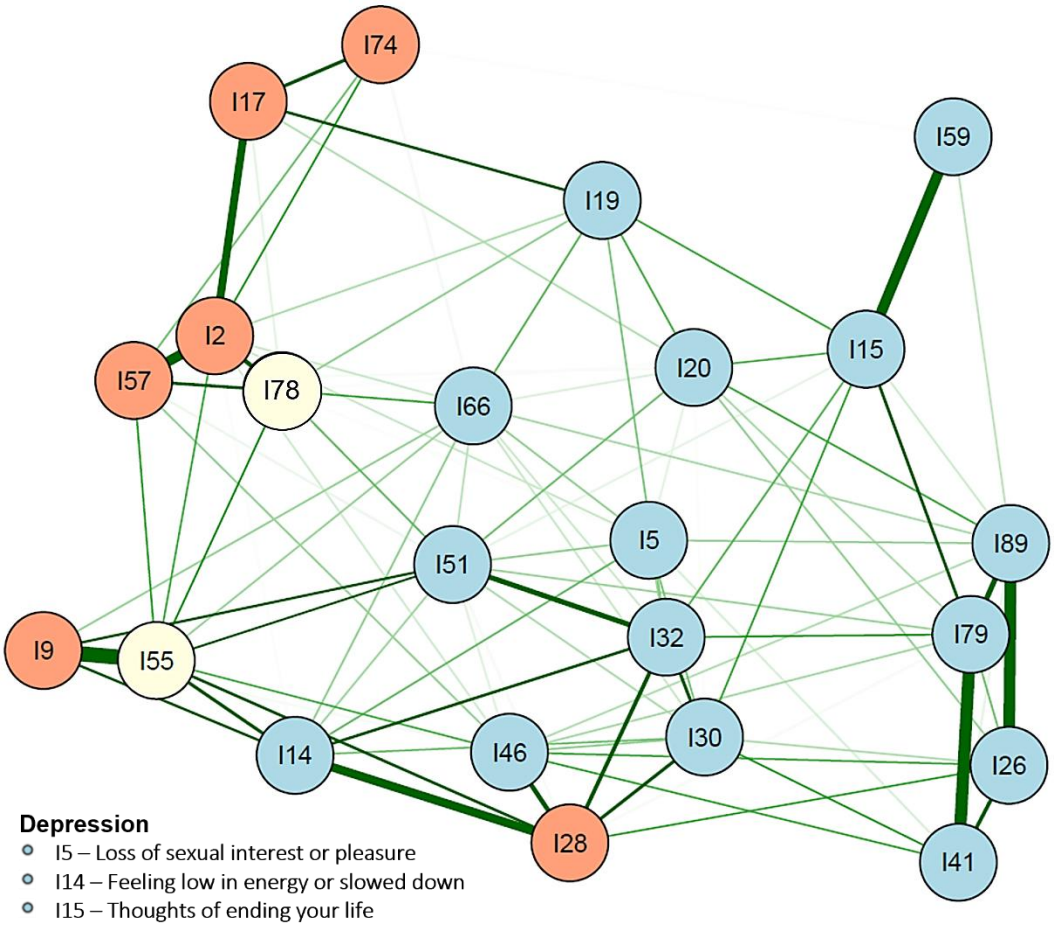
Alteration of edges due to the elimination of a node in an unweighted network



Note. Illustration of one possible consequence of a priori node elimination in an unweighted network. The dark node represents a GAD symptom. The removal of the GAD node makes the two lighter nodes, representing depression symptoms, appear correlated, even though they are not.

Figure 2

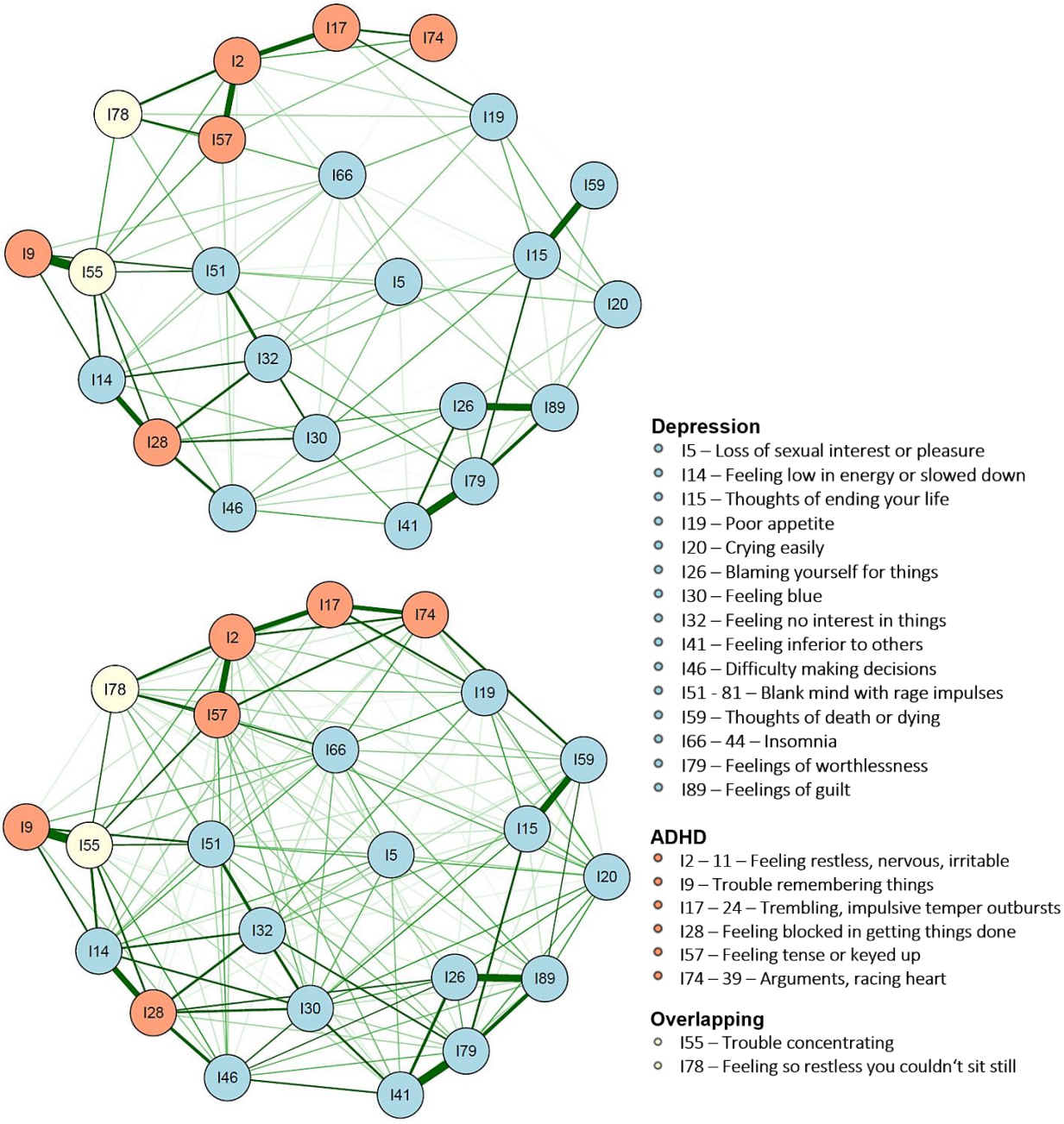
Gaussian graphical model of depression (blue), ADHD (red), and overlapping symptoms (yellow), based on the transdiagnostic sample (n = 1507) according to the advanced method



Note. Undirected partial correlation network with relevant SCL– 90R items as nodes and partial Spearman's correlations as weighted edges. The underlying network was calculated using 79 nodes with diverse psychopathological symptoms to enhance specificity. Green edges show positive correlations. The thickness of edges and saturation increase with correlation. The position of nodes is according to the MDS-Layout, with highly related nodes close together and weakly related ones far apart (Jones et al., 2018a).

Figure 3

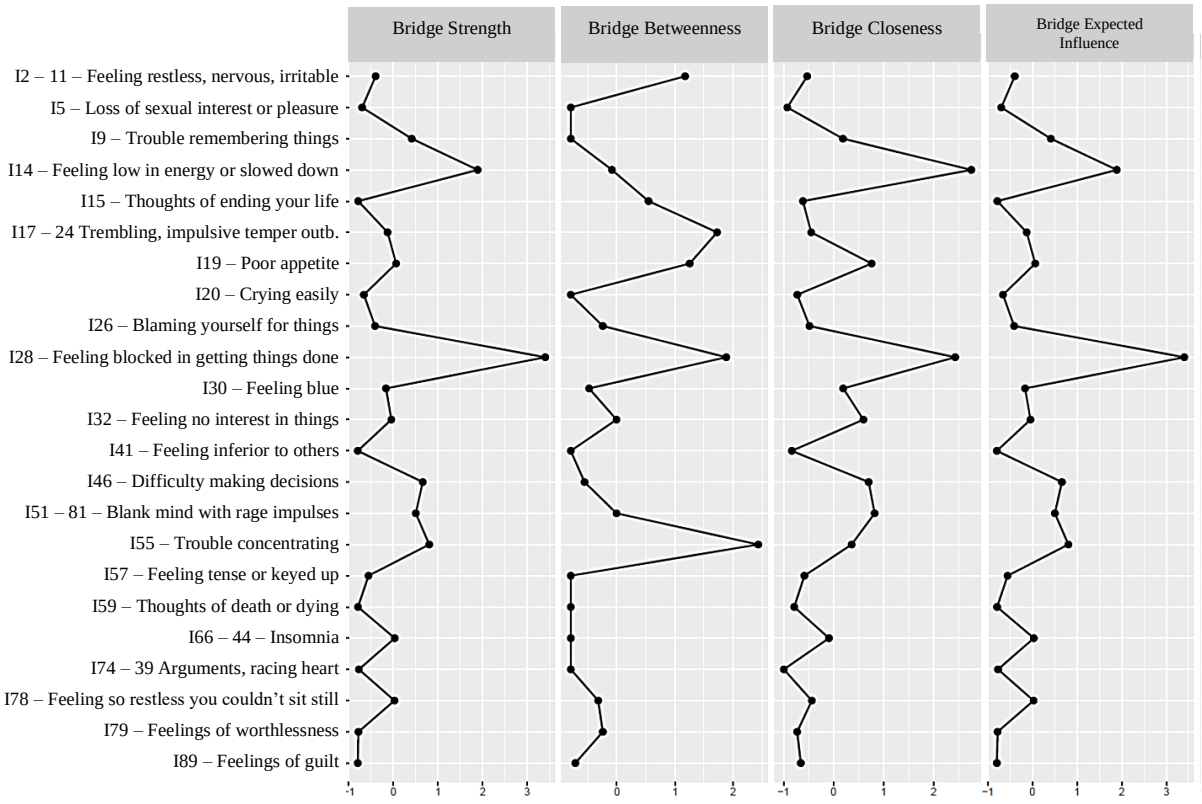
Gaussian graphical model of depression (blue), ADHD (red), and overlapping symptoms (yellow), based on the transdiagnostic sample (n = 1507) with and without advanced method



Note. Undirected partial correlation network with SCL– 90R items as nodes and partial Spearman's correlations as weighted edges. Green edges show positive correlations. The thickness of edges and the saturation increase with correlation. The network at the top was calculated with the advanced method, the network below with the standard procedure. Position of nodes is based on the MDS-Layout, with highly related nodes close together and weakly related ones far apart (Jones et al., 2018a), adjusted for comparison.

Figure 4

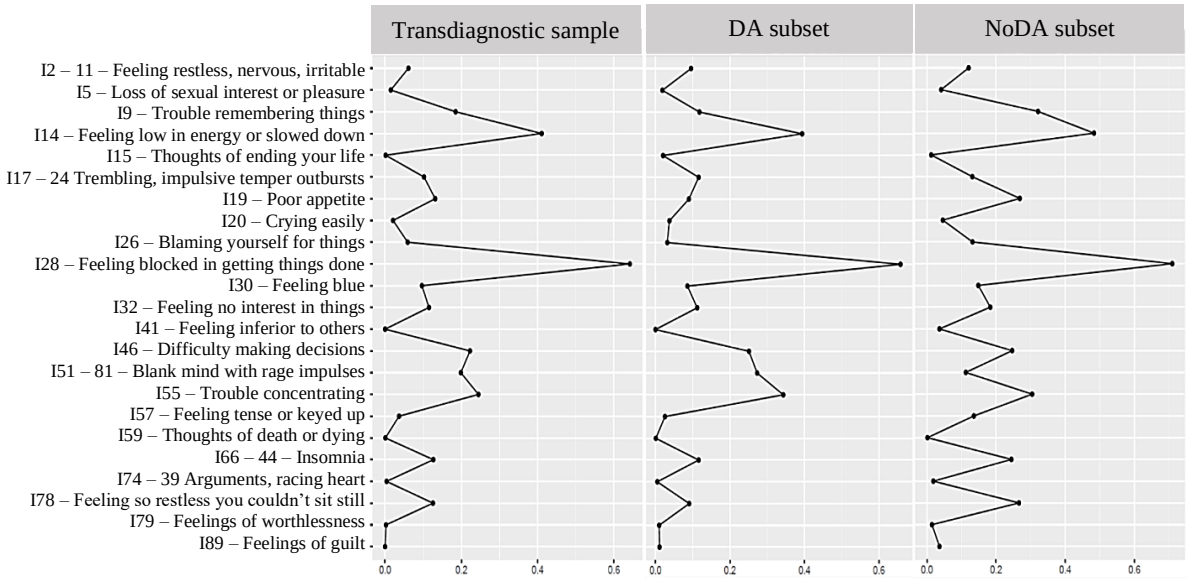
Bridge metric z-values for all relevant items, from the transdiagnostic sample (n = 1507)



Note. There was no difference when overlapping symptoms were classified as depression instead of ADHD symptoms.

Figure 5

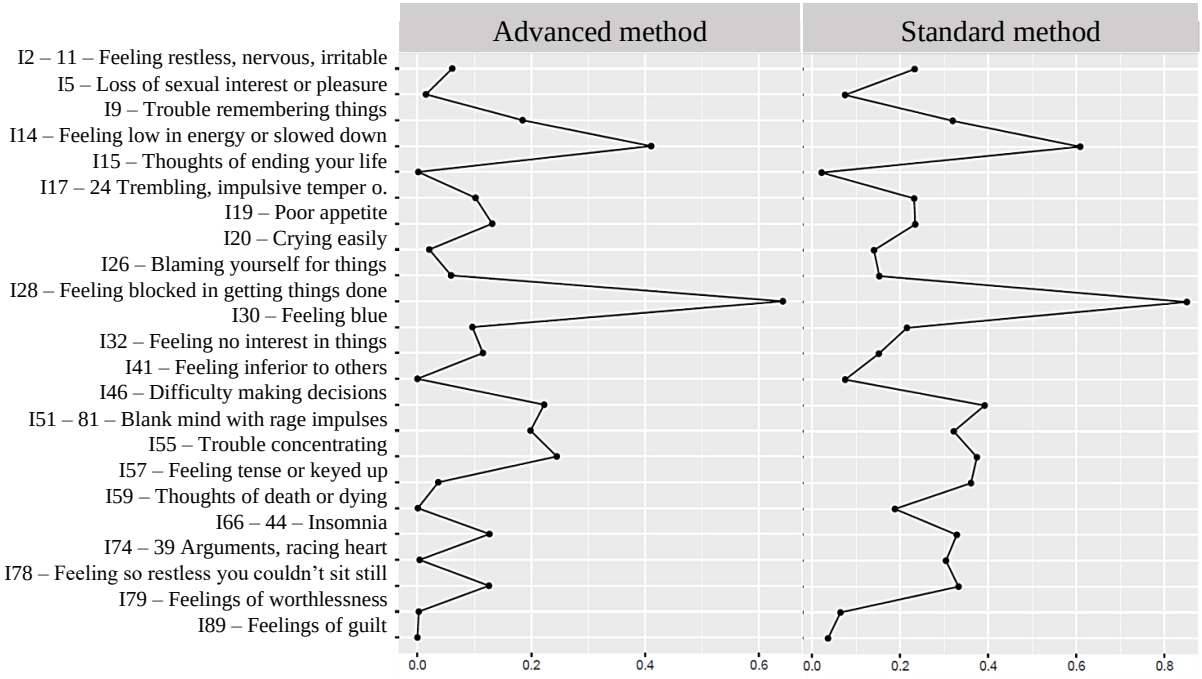
Bridge expected influence for all populations based on advanced method



Note. There was no difference when overlapping symptoms were classified as depression instead of ADHD symptoms.

Figure 6

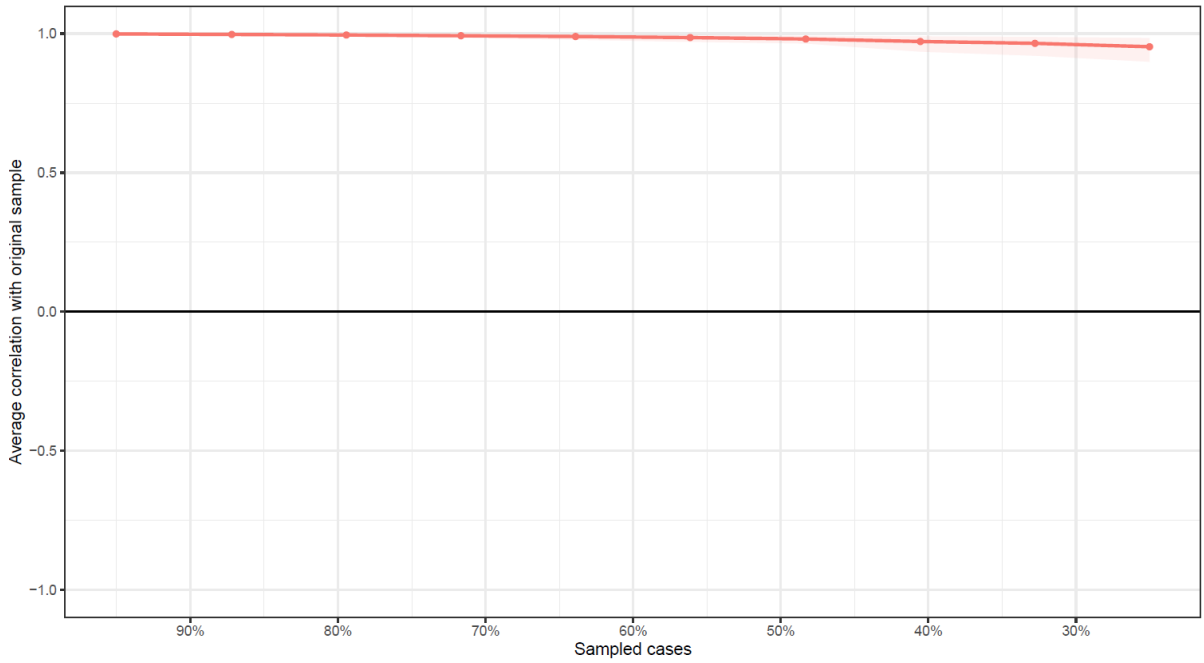
Bridge expected influence comparison between procedures



Note. There was no difference when overlapping symptoms were classified as depression instead of ADHD symptoms.

Figure 7

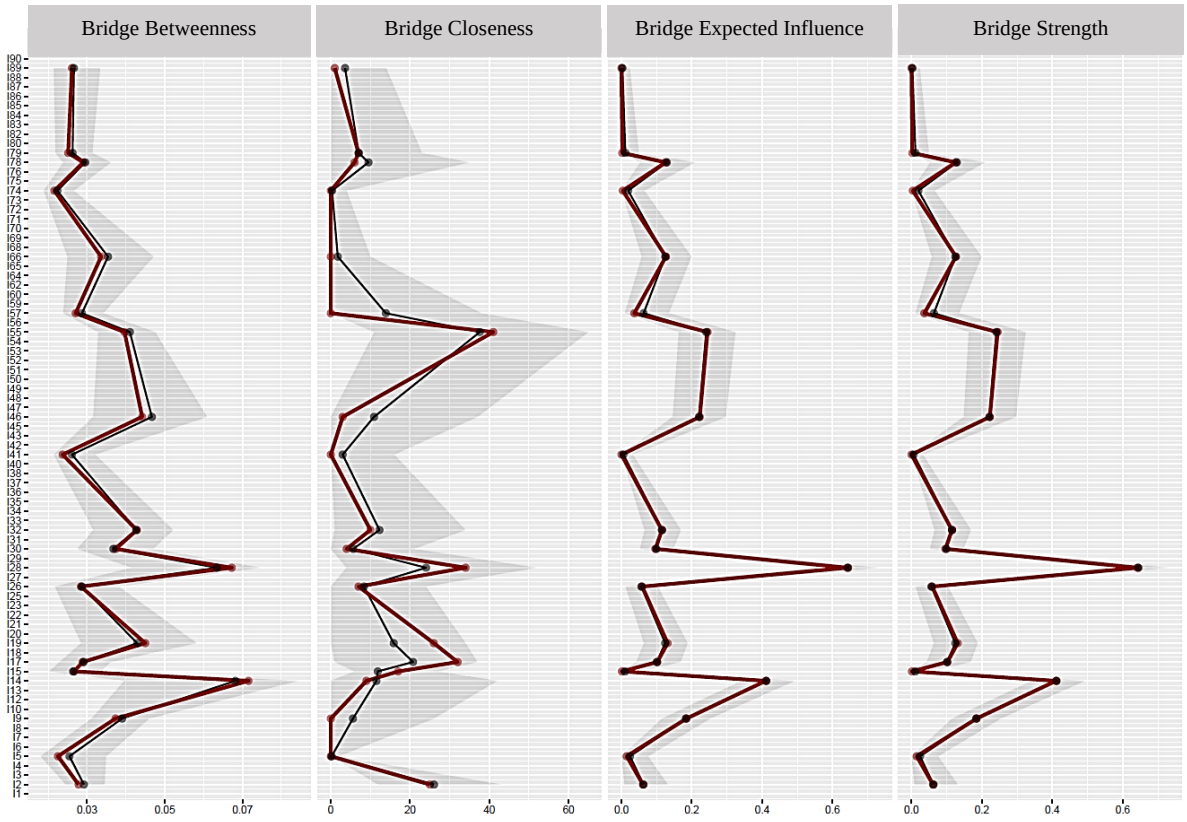
Bootstrapping result for bridge expected influence of the transdiagnostic sample (n = 1507)



Note. The average correlation of bridge expected influence with the corresponding values of drawn subsamples with a varied amount of dropped cases (Jones, 2019). The flat curve indicates a stable metric.

Figure 8

Bridge metric bootstrap for the transdiagnostic sample (n = 1507)



Note. The wider the grey confidence intervals, the more unstable the results. The black line shows the bootstrap mean, red the sample values.

Appendix – R Code

The following packages are needed, as explained in the paper:

```
base, psych, stats, Hmisc, bootnet, eigenmodel, qgraph, smacof,
corStability, NetworkComparisonTest, networktools, lavaan, glasso
```

The following basic packages have not been discussed in the paper but are needed:

```
ggplot2 (for plotting), tidyr (counting missing values)

#Install and load
      install.packages("packagename")
      library("packagename")
```

1. Data is loaded. It contains missing values, dropouts are already excluded. Subsets are created according to subscales of the questionnaire. Multiple Imputation is used.

```
#Load dataset
      data <-  read.csv("D:/Users/Public/R/data.csv", header=TRUE, sep=";")
      data <-  subset(data, select = c(I1:I90, ICD_depressiondiagnosis,
      DSM_ ADHDdiagnosis, PatNr))

#Create subsets (for all subscales)
      SCL_som <-  subset(data, select = c(I1, I4, I12, I27, I40, I42, I48,
      I49, I52, I53, I56, I58, PatNr, ICD_Depression, DSM_ADHS_Diagnose))
      …

#Impute (for all scales) like this
      som_impute <-  aregImpute(formula= ~ I1 + I4 + I12 + I27 + I40 + I42
      + I48 + I49 + I52 + I53 + I56 + I58, data = SCL_som, n.impute = 10)
      som_data <-  SCL_som
      imputed_som <-  impute.transcan(som_impute, imputation
      =1,data=SCL_som, list.out = TRUE, pr=FALSE, check=FALSE)
      som_data[names(imputed_som)] <-  imputed_som
      …

#Rejoin imputed subscale data for an imputed final data set
      finaldata <-  dplyr::full_join(som_data,soc_data, by ="PatNr")
      finaldata <-  dplyr::full_join(finaldata,dep_data, by ="PatNr")
      finaldata <-  dplyr::full_join(finaldata,angst_data, by ="PatNr")
      finaldata <-  dplyr::full_join(finaldata,agg_data, by ="PatNr")
      …

#Ask: How many NA's are left? Did it work?
      missing=which(is.na(finaldata), arr.ind = T)

#Note: When you integrate several questionnaires with different scales,
invert them (if necessary) and compute z-values
      recode(testdata, "1='4';2='3' ;3='2'; 4='1'")
      z_data <-  sapply(testdata, function(x){x- mean(x)/sd(x)})
```

2. Wilcoxon Rank Sum Test is performed to ensure that the imputation did not alter the dataset fundamentally and it can be used in further analysis.

```
#Generate subsets for comparison
      data_raw <-  subset(data, select = c(I1:I90))
      finaldata_nI <-  subset(finaldata, select = c(I1:I90))

#Tests for significance
      wilcox.test(colMeans(data_raw, na.rm = TRUE), colMeans(finaldata_nI,
      na.rm = TRUE))
      wilcox.test(apply(data_raw, 2, sd, na.rm = TRUE),apply(finaldata_nI,
      2, sd, na.rm = TRUE))
```

3. The network is estimated with Spearman's partial correlations for the transdiagnostic sample. The error "correlation matrix is not positive definite" can be expected with many nodes and a relatively small population. If this is not the case, continue with point number six.

```
      Network_all_sp <-  estimateNetwork(finaldata_nI,
      default="EBICglasso", corMethod = "Spearman")
```

4. Computing eigenvalues of the correlation matrix.

```
#Calculate Spearman's correlation matrix
      All_Matrix_sp <-  cor(finaldata_nI, method = c("Spearman"))

#Calculate the partial correlation matrix. 1507 is the number of cases, and
gamma, the EBIC tuning parameter, is set to 0.5 to return a simple model
      All_Matrix_pcor <-  EBICglasso(All_Matrix_sp, gamma = 0.5, 1507)

#Calculate eigenvalues
      Eigen_all_sp <-  eigen(All_Matrix_sp)
```

5. Procedure to avoid negative eigenvalues
    a. Find items with high kurtosis

```
#Show statistical values
      stats_SCL <-  as.matrix(psych::describe(finaldata_nI))
```

    b. Identify item pairs that correlate nearly fully by viewing the correlation matrix
    c. Identify item pairs with high correlation (over .75) and very similar content
    d. Delete all troublesome items according to a., then delete one item of each pair according to b. and c.

```
#Listing all items to be included under select by their column-names

      finaldata2 <-  subset(finaldata, select =
      c(I1,I2,I3,I4,I5,I6,I7,I8,I9,I10,I12,I13,I14,I15,I17,I18,I19,I20,I21,
      I22,I23,I25,I26,I27,I28,I29,I30,I31,I32,I33,I34,I35,I36,I37,I38,I40,I
      41,I42,I43,I45,I46,I47,I48,I49,I50,I51,I52,I53,I54,I55,I56,I57,I59,I6
      0,I62,I64,I65,I66,I68,I69,I70,I71,I72,I73,I74,I75,I76,I78,I79,I80,I82
      ,I83,I84,I85,I86,I87,I88,I89,I90))

#Base network for all nodes – repeat with new dataset
      Network_all_sp <-  estimateNetwork(finaldata2,  default="EBICglasso",
      corMethod = "Spearman")
```

6. Testing network accuracy with bootstrapping.

```
#Case-bootstrap the first network
      All_boot_casedrop <-  bootnet(Network_all_sp, nBoots = 1000, nCores =
      16, type = "case", statistics = c("edge"))

#Plot average correlations between centrality indices of sampled networks
with a variable number of persons dropped and the original sample. Lines
indicate the means and areas indicate the range from the 2:5th quantile to
the 97:5th quantile. (Epskamp et al., 2017a) – The straighter the plotted
line, the better
      plot(All_boot_casedrop, statistics = "all")

#Execute Correlation Stability Analysis – extract and save CS-Coefficient.
According to Epskamp et al. (2017a) the CS-Coefficient should be at least
above 0.25 and preferably above 0.5
   All_corStability_casedrop <- corStability(All_boot_casedrop)
```

7. Extracting the edge weights matrix from the network model.

```
   all_bootnet_Wmat_sp <-  getWmat(Network_all_sp)
   all_bootnet_Wmat_sp <-  as.data.frame(all_bootnet_Wmat_sp)
```

8. Deleting all irrelevant items. Numbers indicate positions (2 is the second row and column).

```
   All_Subset_relevant <-  subset(all_bootnet_Wmat_sp, select= c(2, 5, 9,
   13, 14,15,17,18,23,25,27,29,37,41,46,50,52,53,58,65,68,69,78))

   All_Subset_relevant <-  All_Subset_relevant[c(2, 5, 9, 13,
   14,15,17,18,23,25,27,29,37,41,46,50,52,53,58,65,68,69,78), ]
```

9. Creating MDS–Layout input. For further information see Jones et al. (2018a).

```
   All_MDS_dis_matrix_rel <-  sim2diss(All_Subset_relevant)
   All_MDSr <-  mds(All_MDS_dis_matrix_rel)
   head(round(All_MDSr$conf, 2))
```

```
#Identify the best transformation function according to the lowest stress-1
value. For ordinal data, "ordinal" and "polychoric" are relevant. For other
types of data, see Jones et al. (2018a).

#Ordinal
      All_MDS_ordinal <-  mds(All_MDS_dis_matrix_rel, type ="ordinal")
      plot(All_MDS_ordinal, plot.type = "Shepard", main="Ordinal")
      round(All_MDS_ordinal$stress,2)

#Polychoric
      All_MDS_mspline <-  mds(All_MDS_dis_matrix_rel, type ="mspline")
      plot(All_MDS_mspline, plot.type = "Shepard", main="Mspline")
      round(All_MDS_mspline$stress,2)
```

10. Visualizing the network of relevant items.

```
#Import a legend. Use the first column of the CSV and list the item or node
titles in the same order as they are listed in the weights matrix
      Names <-  scan("D:/Users/Public/R/Legend.csv", what = "character",
      sep = "\n")

#Plot the network with two groups for the different diagnoses. The vector
c() refers to the position of the nodes within the columns
      All_MDS_ordinal <-  qgraph(All_Subset_relevant, layout =
      All_MDS_ordinal$conf, esize = 7, labels =
      colnames(All_Subset_relevant), width = 1500, vsize=5, nodeNames =
      Names, groups = list(Depression =
      c(2,4,5,7,8,9,11,12,13,14,15,18,19,22,23), ADHS = c(1,3,6,10,17,20),
      Both = c(16, 21)), color = c("lightblue", "lightsalmon",
      "lightyellow"), legend.cex = 0.37, title = "Advanced network of
      transdiagnostic sample")

      plot(All_MDS_ordinal_rel_graph)
```

11. Bridge metric calculation.

```
#Categorize all nodes according to their designated community (e.g.,
disorder). Inside the community vector, 1 and 2 represent the two different
communities, the position of the indexing number is according to the node
position inside the input matrix columns
      communities_all <-  c('2', '1', '2','1','1','2','1','1',
      '1','2','1','1','1','1','1','2','2','1','1','2','2','1','1')

#Calculate bridge metrics
      All_bridge <-  bridge(All_MDS_ordinal_rel_graph, communities =
      communities_all)

#Create the bridgeplot
      All_bridge_plot <-  plot(All_bridge_rel_2)
```

12. Measurement of the accuracy and stability of the bridge centrality values with bootstrapping.

```
#Define communities. As above, inside the community vector, 1 and 2
represent the two different communities, the position of the indexing
number is according to the node position inside the input matrix columns.
Because it is only possible to bootstrap the original bootnet network,
community 3 is added to create an "other" category
      communities_boot <-
      c('3','2','3','3','1','3','3','3','2','3','3','3','1','1','2','3','1'
      ,'1','3','3','3','3','1','3','2','3','1','3','1','3','3','3','3','3',
      '3','3','1','3','3','3','1','3','3','3','3','1','3','3','3','2','3','
      2','1','3','3','3','3','1','3','3','3','3','3','3','2','3','3','2','1
      ','3','3','3','3','3','3','3','3','3','1','3')

#Case-bootstrap, tell the function which communities to use
      All_boot_bridge_rel <-  bootnet(Network_all_sp, nBoots = 1000,
      type="case", statistics = c("all") , nCores = 20, communities =
      communities_boot, useCommunities = c('1', '2'))

#Execute correlation stability analysis to access CS-Coefficient. Again, a
CS-Coefficient over .25 is the minimum, and one above .5 is good. If the
output is faulty and showing 0 as CS-Coefficient for bridge values, the
plot can be used for judgment of accuracy (linear is good)
      cor_stab_all <-  corStability(All_boot_bridge_rel)

#Plot average correlations between centrality indices of sampled networks
with a variable number of persons dropped and the original sample. Lines
indicate the means, and areas indicate the range from the 2:5th quantile to
the 97:5th quantile(Epskamp et al., 2017a)
      plot(All_boot_bridge_rel, statistics = c("bridgeBetweenness",
      "bridgeCloseness", "bridgeExpectedInfluence", "bridgeStrength"))
```

13. Plotting of the confidence intervals for bridge metrics to access accuracy

```
#Nonparametric bootstrapping
      All_boot_bridge_rel_np <-  bootnet(Network_all_sp, nBoots = 1000,
      type="nonparametric", statistics ="all", nCores = 20, communities =
      communities_boot, useCommunities = c('1', '2'))

#Plot confidence intervals. Subset limits the graph to the relevant items
      All_boot_bridge_central_plot <-  plot(All_boot_bridge_rel_np,
      statistics = c("bridgeBetweenness", "bridgeCloseness",
      "bridgeExpectedInfluence", "bridgeStrength") , bootlwd = 0.2, subset
      =
      c("I5","I9","I14","I15","I19","I26","I30","I32","I41","I46","I66","I7
      9","I89","I2","I17", "I28","I55","I57","I74","I78"))
```

14. If necessary: Create the same qgraph layout input from two networks for comparison

```
      layout <-  averageLayout(Networkgraph1, Networkgraph2)
```

The standard procedure (for comparison)

```
standard_finaldata <-  subset(finaldata, select =
c(I2,I5,I9,I14,I15,I17,I19,I20,I26,I28,I30,I32,I41,I46,I51,I55,I57,I59,I66,
I74,I78,I79,I89))

standard_network <-  estimateNetwork(standard_finaldata,
default="EBICglasso", corMethod = "Spearman")
standard_boot_casedrop <-  bootnet(standard_network, nBoots = 1000, nCores
= 16, type = "case", statistics = c("edge", "strength", "betweenness",
"closeness", "distance", "expectedInfluence", "length"))
plot(All_standard_boot_casedrop, statistics = "all")

standard_corStability_casedrop <- corStability(standard_boot_casedrop)

matrix_standard <-  cor(standard_finaldata, method = c("Spearman"))
matrix_standard_pcor <-  EBICglasso(matrix_standard, gamma = 0.5, 1507)

standard_dis_matrix <-  sim2diss(matrix_standard_pcor)
standard_MDSr <-  mds(standard_dis_matrix)
head(round(standard_MDSr$conf, 2))

standard_MDS_ordinal <-  mds(standard_dis_matrix, type ="ordinal")
plot(standard_MDS_ordinal, plot.type = "Shepard", main=”ordinal")
round(standard_MDS_ordinal$stress,2)

standard_MDS_mspline <-  mds(standard_dis_matrix, type ="mspline")
plot(standard_MDS_mspline, plot.type = "Shepard", main="mspline")
round(standard_MDS_mspline$stress,2)

standard_MDS_ordinal_graph <-  qgraph(matrix_standard_pcor, layout =
standard_MDS_ordinal$conf, esize = 7, width = 1500, labels =
colnames(matrix_standard_pcor), vsize=5, nodeNames = Names, groups =
list(Depression = c(2,4,5,7,8,9,11,12,13,14,15,18,19,22,23), ADHS =
c(1,3,6,10,17,20), Both = c(16, 21)), color = c("lightblue", "lightsalmon",
"lightyellow"), legend.cex = 0.37, title = "Standard network of
transdiagnostic sample")
plot(standard_MDS_ordinal_graph)

standard_bridge <-  bridge(standard_MDS_ordinal_graph, communities = c('2',
'1', '2','1','1','2','1','1',
'1','2','1','1','1','1','1','2','2','1','1','2','2','1','1'))

standard_bridge_plot <-  plot(standard_bridge)

standard_bridge_table <-  as.data.frame(standard_bridge[c("Bridge Expected
Influence (1- step)")])
```